BILIARY/PANCREAS

S1 Outstanding Research Award in The Biliary/Pancreas Category (Trainee)

Results of First Round of Enrollment from the Screening for Pancreatic Cancer in High-Risk Individuals (Pancreas Scan Study): A Prospective Multi-Center Study

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Introduction: Multiple guidelines endorse pancreas cancer screening in genetically susceptible individuals. However, data on outcomes of screening are limited. Our goal was to conduct a prospective, multicenter study to evaluate yield, harms, and outcomes of pancreas cancer screening.
Methods: All high-risk individuals undergoing EUS or MRCP for pancreas cancer screening at 5 institutions in the US from 2020 to 2022 were prospectively enrolled. Pancreas pathology was designated as low-risk (fatty pancreas or chronic pancreatitis-like changes), intermediate-risk (neuroendocrine tumor (NET) <2 cm or branch duct IPMN) and high-risk (High grade PanIN/dysplasia, main duct IPMN, NET >2 cm, or pancreatic cancer). Harms from screening included: (i) adverse events during screening EUS or MRI, or (ii) undergoing low-yield pancreatic surgery (where pathology did not show high-grade dysplasia or cancer) (ClinicalTrials.gov: NCT05006131).
Results: During study period, 252 patients undergoing pancreas cancer screening (EUS in 208 and MRCP in 44 patients) were enrolled, of which 97 underwent their first screening. Mean age was 59.9 years, 69% were female and 79.4% were White. Indication was familial pancreatic cancer syndrome (31.7%), BRCA2 (29%), BRCA1 (7.5%), ATM (3.5%), Lynch syndrome (6.7%), Peutz-Jeghers (4.3%), and FAMMM (3.5%). Remaining 13% did not meet current guideline screening criteria. Low-risk pancreas pathology was noted in 23.4%, intermediate risk in 31.7%, and high risk in 0.8% of patients (Table 1). In all, tissue sampling was performed for 5 solid pancreatic lesions, 12 pancreatic cystic lesions, 1 abnormal duct, and 1 lymph node. Two patients had pancreatic cancer, including 1 patient with T2N1M0 (stg IIB) cancer, who underwent neoadjuvant treatment and pancreatic resection. The second patient had T2N1M1 (stg IV) cancer and underwent palliative chemotherapy. Twelve (4.8%) patients underwent further pancreatic evaluation due to screening findings (EUS in 8 patients and an MRCP in 4 patients). None of the patients underwent low-yield pancreatic surgery. There were no adverse events from screening tests or from further interventions.
Conclusion: Pancreas cancer screening in high-risk individuals detected intermediate-risk lesions in 32% and high-risk lesions in 0.8% patients. Screening did not lead to any harms. Resection with curative intent was performed in 1 of 2 patients with pancreatic cancer.

Table 1. Prevalence of Pancreatic Pathology Among High-Risk Patients Undergoing Pancreatic Cancer Screening

Low-risk lesions	
Fatty Pancreas	15 (5.95%)
Chronic pancreatitis-like changes	44 (17.5%)
Definitive CP meeting Rosemont criteria	0 (0%)
Intermediate-risk lesions	
BD-IPMN	79 (31.3%)
Mean size of largest BD-IPMN	8.17 mm
Patients with multiple BD-IPMN	35 (44.3%)
NET< 1 cm	1 (0.4%)
High-risk lesions	
MD-IPMN	0 (0%)
PanIN	0 (0%)
NET >2 cm	0 (0%)
Adenocarcinoma	2 (0.8%)

S2

Recurrent Episode of Acute Pancreatitis in Patients With Idiopathic Acute Pancreatitis and Fatty Pancreas on Endoscopic Ultrasound

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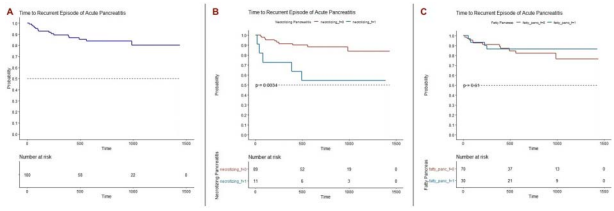
Introduction: Idiopathic acute pancreatitis (IAP) is diagnosed after a negative evaluation. It remains unclear if fatty pancreas is a cause of acute pancreatitis (AP) or a risk factor for recurrent AP. We aimed to determine if fatty pancreas was associated with a recurrent episode of AP in patients with IAP.
Methods: Patients with IAP were identified across three academic centers. Patients were included if they underwent an endoscopic ultrasound (EUS) at one of our centers. Presumed IAP prior to EUS was confirmed through retrospective review of clinical data. Patients who had an etiology discovered on EUS were excluded, leaving a cohort of "true" IAP. Presence of fatty pancreas on EUS was noted. Recurrent episode of AP, emergence of diabetes, chronic pancreatitis, and follow up cholecystectomy were recorded. Univariable analysis was performed using Wilcoxon Rank Sum or Fisher Exact tests. A Cox Proportional Hazard Regression Analysis was performed for a recurrent episode of AP.
Results: 105 patients with "true" IAP were identified. Fatty pancreas was noted in 31 patients on EUS. Older age, male gender, white race, higher BMI, obesity, presence of fatty liver, and type 2 diabetes mellitus were associated with fatty pancreas on univariable analysis (Table 1). Five patients had no follow up data after the EUS. Median time of follow up was 687 days (IQR 464–1026 days). During the follow up period, 16 patients had a recurrent episode of AP (16/100, 16%; median time to recurrence 216 days, IQR 76–420 days), 8 were diagnosed with new diabetes (8/48, 16.7%), 5 with chronic pancreatitis (5/38, 13.2%), and 10 underwent cholecystectomy (10/56, 17.9%). On unadjusted Cox regression analysis, being evaluated as a referral ($P = 0.0417$), a history of necrotizing pancreatitis ($P = 0.0073$), number of attacks within the preceding year before EUS ($P = 0.0012$), and total bilirubin at the most recent episode of AP ($P = 0.0217$) were associated with a recurrent episode of AP (Figure 1). Neither the presence of fatty pancreas nor fatty liver were significant.
Conclusion: The incidence of a recurrent episode of AP was 16% over approximately 2 years in patients with IAP. The presence of fatty pancreas was not associated with a recurrent episode. Further studies on fatty pancreas in IAP are recommended.

Table 1. Patient characteristics

Baseline Characteristics Median (IQR) or Fraction (%)		Unadjusted Univariable Analysis Median (IQR) or Fraction (%)			Unadjusted Cox Regression For Recurrent Episode AP	
	All Patients N=105	No fatty pancreas N=74	Fatty pancreas N=31	p-value	HR (95% CI)	p-value
Age at EUS, per 5 years	53.8 (40.4-64.3)	47.6 (37.9-60.4)	63.7 (53.9-68.2)	< 0.001[1]	0.92 (0.79-1.08)	0.3203
Male gender	45 (42.9%)	27 (36.5%)	18 (58.1%)	0.053[2]	1.56 (0.57-4.30)	0.3901
BMI, per 5 kg/m2	28.2 (24.2-33.7)	26.6 (22.9-29.5)	33.7 (30.4-38.8)	< 0.001[1]	1.00 (0.73-1.38)	0.9769
Obesity	43 (41.0%)	17 (23.0%)	26 (83.9%)	< 0.001[2]	1.14 (0.41-3.15)	0.7984
White Race	96 (91.4%)	65 (87.8%)	31 (100.0%)	0.055[2]	0.55 (0.12-2.46)	0.4361
Hispanic Ethnicity	3 (2.9%)	2 (2.7%)	1 (3.2%)	1.000[2]	2.46 (0.32-18.82)	0.3859
Never Smoker	63 (60.0%)	47 (63.5%)	16 (51.6%)	0.281[2]	0.45 (0.16-1.27)	0.1324
Diabetes Mellitus, type 2	35 (33.3%)	20 (27.0%)	15 (48.4%)	0.043[2]	1.23 (0.44-3.47)	0.6890
Cholecystectomy	47 (44.8%)	34 (45.9%)	13 (41.9%)	0.830[2]	0.81 (0.29-2.28)	0.6947
Fatty Liver	47 (44.8%)	24 (32.4%)	23 (74.2%)	< 0.001[2]	0.69 (0.25-1.94)	0.4848
Evaluated as referral	74 (70.5%)	54 (73.0%)	20 (64.3%)	0.482[2]	0.35 (0.13-0.96)	0.0417
Necrotizing pancreatitis	11 (10.5%)	8 (10.8%)	3 (9.7%)	1.000[2]	4.36 (1.49-12.78)	0.0073
Single episode of AP	43 (41.0%)	30 (40.5%)	13 (41.9%)	1.000[2]	0.92 (0.33-2.58)	0.8712
Total attacks of AP	2 (1-3)	2 (1-3)	2 (1-3)	0.933[1]	1.16 (0.96-1.40)	0.1299
Attacks within year of EUS, per 1 attack	1 (1-2)	1 (1-2)	1 (1-2)	0.349[1]	1.85 (1.28-2.70)	0.0012
Fatty pancreas on EUS	31 (29.5%)	NA	NA	NA	0.74 (0.24-2.34)	0.6618
At most recent episode of AP						
Lipase, per 500 U/L	920 (275-2530)	1000 (413-2500)	359 (190-3000)	0.152[1]	1.01 (0.97-1.06)	0.5787
Total bilirubin, per 1 mg/dL	0.4 (0.3-0.6)	0.4 (0.3-0.6)	0.5 (0.4-0.6)	0.595[1]	5.52 (1.28-23.71)	0.0217
Direct bilirubin, per 1 mg/dL	0.2 (0.2-0.2)	0.2 (0.2-0.2)	0.2 (0.2-0.2)	0.070	23.55 (0.3-1835.4)	0.1552
ALT, per 10 U/L	24 (17-35)	21 (16-33)	30 (24-39)	0.009[1]	0.96 (0.79-1.17)	0.7021
Calcium, per 1 mg/dL	9.4 (9.1-9.6)	9.4 (9.1-9.7)	9.3 (9.1-9.5)	0.238[1]	0.25 (0.06-1.08)	0.0626
Triglycerides, per 50 mg/dL	103 (75-173)	103 (74-158)	104 (85-177)	0.604[1]	1.07 (0.83-1.36)	0.6142
IgG4 level, per 10 mg/dL	23.9 (11.1-41.0)	25.2 (11.9-40.1)	22.5 (11.3-39.7)	0.887[1]	0.96 (0.76-1.21)	0.7190
CA 19-9, per 10 U/mL	8.5 (5-13)	8.5 (5.8-13.0)	7.5 (5-15)	0.712[1]	0.87 (0.49-1.53)	0.6281

IQR, interquartile range; AP, acute pancreatitis; HR, hazard ratio; 95% CI, 95% confidence interval; EUS, endoscopic ultrasound; BMI, body mass index; NA, not applicable; ALT, alanine transaminase; IgG4, immunoglobulin G type 4; CA 19-9, carbohydrate antigen 19-9 Reference ranges: Lipase 13 – 60 U/L, Total Bilirubin ≤ 1.2 mg/dL, Direct Bilirubin 0.0 – 0.3 mg/dL, ALT 7 – 55 U/L, Triglycerides < 150 mg/dL, Calcium 8.8 – 10.2 mg/dL, CA 19-9 < 35 U/mL, IgG4 2.4 – 121.0 mg/dL 1. Wilcoxon Rank Sum Test 2. Fisher Exact Test



[0002] **Figure 1.** Kaplan-Meier Estimates for Time to Recurrent Episode of Acute Pancreatitis. A) Entire Cohort of Patients with IAP. B) By History of Necrotizing Pancreatitis. C) By Presence of Fatty Pancreas on EUS. Legend B: Blue = History of Necrotizing Pancreatitis, Red = No History of Necrotizing Pancreatitis. Legend C: Blue = Fatty Pancreas on EUS, Red = Non-Fatty Pancreas on EUS.

S3

EUS-Guided Fine-Needle Aspiration (FNA) vs Core-Needle Biopsy (CNB) for Diagnostic Yield and Tissue Sampling Adequacy for Molecular Testing in Pancreatic Ductal Adenocarcinoma

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Introduction: Pancreatic ductal adenocarcinoma (PDAC) is the third most common cause of cancer death in the US, with a 5-year survival rate of around 9%. Endoscopic ultrasound (EUS) has become the mainstay of diagnosis of pancreatic cancer, with tissue acquisition either through fine-needle aspiration (FNA) or core-needle biopsy (CNB). There is limited literature on the adequacy of samples for molecular testing obtained via FNA versus CNB. Our study aims at comparing these two modalities, regarding sample adequacy for genomic sequencing.
Methods: A retrospective chart review was conducted among all patients who underwent EUS, at Saint Luke's Hospital (SLH), KC, MO starting January 1, 2018, till December 31, 2021, for PDAC. Patients were stratified based on the mode of tissue acquisition (FNA vs CNB). Age, gender, tumor size and location, needle gauge size, number of passes, CA 19-9 level, and CEA level, were recorded. Rapid on-site evaluation was performed in all cases by cytotechnologists. Slides review of FNA and FNBs was performed by board-certified cytopathologists for the final pathological diagnosis, tumor surface area, tumor cellularity, and specimen adequacy for molecular testing.
Results: A total of 132 patients underwent EUS guided biopsies. 76 of them were FNA, 48 CNB, and 8 were combined. 94.6% (125/132) of the lesions were greater than 2 cm^2 in size. 56.8% (75/132) of them involved the head of the pancreas. The mean number of passes needed for CNB and FNA were 2.58 ± 1.06 and 2.49 ± 1.07 respectively. 71.4% (35) of CNB obtained samples were adequate for molecular testing compared to 32.1% (26) of FNA. Genomic testing was performed in 46.4% (26) of CNB samples and in only 23.8% (20) of FNA samples.
Conclusion: Although there was no significant difference in the number of passes needed to establish a diagnosis. EUS-CNB under the guidance of rapid on-site specimen evaluation demonstrated a higher value in obtaining adequate samples for molecular testing. Tumor surface area and tumor cellularity play a key role, regardless of tissue acquisition modality.

Table 1. EUS Guided Fine-Needle Aspiration (FNA) Vs Core-Needle Biopsy (CNB) for Diagnostic Yield and Tissue Sampling Adequacy for Molecular Testing in Pancreatic Ductal Adenocarcinoma

Baseline patient and procedure characteristics			
Characteristic	**CNB (n=48)**	**FNA (n=76)**	**FNA & CNB (n=8)**
Mean age, years (±SD)	69.88 ± 10.38	72.11 ± 10.86	67.25 ± 9.44
Male, n (%)	22 (45.8%)	36 (47.4%)	6 (75.0%)
CEA level, n Median (Q1, Q3)	15 5.40 (2.70, 47.90)	27 5.10 (1.70, 16.90)	2 8.70 (6.60, 10.80)
CA 19-9 level, n Median (Q1, Q3)	35 952.00 (129.00, 4483.00)	53 312.00 (43.00, 1661.00)	5 195.00 (174.00, 223.00)
Complications, n (%)	0	0	0
Characteristic	**CNB (n=56)**	**FNA (n=84)**	**P value**
Pancreatic lesion size in cm2 < 2 cm2 ≥ 2 cm2	 4 (7.1%) 52 (92.9%)	 4 (4.8%) 80 (95.2%)	0.713
Pancreatic lesion location, n (%) Body Body and tail Head / uncinate process Head and neck Neck and body Neck/genu Tail	 7 (12.5%) 7 (12.5%) 28 (50.0%) 5 (8.9%) 1 (1.8%) 2 (3.6%) 6 (10.7%)	 11 (13.1%) 5 (6.0%) 50 (59.5%) 4 (4.8%) 3 (3.6%) 2 (2.4%) 9 (10.7%)	0.719
Needle gauge, n (%)* 20 G 22 G 25 G	 22 (40.0%) 32 (58.2%) 1 (1.8%)	 10 (11.9%) 69 (82.1%) 5 (6.0%)	< 0.001
Categorical variables were compared using chi-square or Fisher's exact test. * There is 1 missing in the CNB group			

Comparison of pathological characteristics			
Characteristic	**CNB (n=56)**	**FNA (n=84)**	**P value**
Tumor Surface Area in mm2, n Median (Q1, Q3)	49 25.00 (4.00, 100.00)	81 4.00 (1.00, 25.00)	< 0.001
Tumor cellularity* < 20% 20%-49% >49%	 9 (18.4%) 25 (51.0%) 15 (30.6%)	 30 (37.0%) 32 (39.5%) 19 (23.5%)	0.079
Mean number of smear slides (±SD)	4.30 ± 2.82	4.49 ± 2.80	0.704
Continuous variables compared using Student's T-test or Wilcoxon rank-sum test. Categorical variables compared using chi-square or Fisher's exact test. * There are 7 missing in CNB group and 3 missing in FNA group.			

Comparison of outcomes for FNA and CNB			
Outcome measure	**CNB (n=56)**	**FNA (n=84)**	**P value**
Mean pass counts (±SD)*	2.58 ± 1.06	2.49 ± 1.07	0.5096
Sample adequacy for molecular testing, n (%)	35 (71.4%)	26 (32.1%)	< 0.001
Genomic testing Performed, n (%)	26 (46.4%)	20 (23.8%)	0.0050
Continuous variables were compared using Student's T-test. Categorical variables were compared using chi-square or Fisher's exact test.*Outcomes were statistically analyzed after adjusting for age, sex, Pancreatic lesion location, and Pancreatic lesion size. There are 3 missing in the CNB group.			

S4 ACG Governors Award for Excellence in Clinical Research

Autoimmune Pancreatitis Secondary to Immune Checkpoint Inhibitor Therapy (Type 3 AIP): Insights into a New Disease from Clinical Review and Serial Pancreatic Imaging

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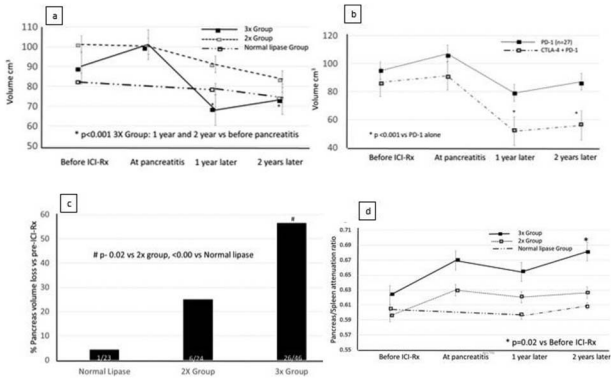
Introduction: Autoimmune pancreatitis (AIP), types 1 and 2, are well described. While type 1 AIP is a pancreatic manifestation of immunoglobulin G4-related disease (IgG4-RD) and type 2 AIP is a duct-centric pancreatic injury sometimes associated with inflammatory bowel disease. A third form of AIP (type 3) is caused by increased immune activity by immune checkpoint inhibitor therapy (ICI-R) for advanced malignancies. We describe the clinico-radiological spectrum and short-term natural history of type 3 AIP.
Methods: We performed a detailed clinical record review of the 248/11,165 (2.2%) adult patients receiving ICI-R who developed type 3 AIP (>3-fold serum lipase elevation + pain, absent other etiologies). A radiologist reviewed 379 abdominal computerized tomography scans (CT) and measured pancreas volume in a subset of CTs done before, during and after pancreatitis.
Results: At onset, type 3 AIP was painful in 96 (38%) and asymptomatic in 154 (62%); none had jaundice or local/systemic complications. CT showed normal pancreas (43%), peri-pancreatic edema (27%), loss of feathery pattern (21%) or diffuse/focal pancreatic enlargement (10%). A minority achieved complete and persistent biochemical resolution, more often when ICI-R was held (32/89) vs no intervention (13/84, $P = 0.003$) or vs steroid treatment alone (2/23, $P = 0.01$). Early or late relapses occurred equally frequently regardless of intervention. Compared to pancreas volume before ICI-R, >20% volume loss occurred in 55% 1 year post-pancreatitis, more so with dual ICI-Rx, regardless of pain or steroid therapy at presentation. 18% had new diabetes within 2 years of pancreatitis. No patient developed calcifications or duct dilation.
Conclusion: Type 3 AIP is a novel drug-induced chronic inflammatory disease of the pancreas. Predominantly asymptomatic and mild in severity, it causes rapid pancreatic atrophy and functional endocrine failure despite steroid therapy or ICI-R withdrawal.

Table 1. Clinical profile of all type 3 and subjects in imaging cohorts

	Type 3 AIP* (n=248)		Imaging cohort (n=93)		
	Symptomatic (n=94)	Asymptomatic (n=154)	≥3x lipase elevation cohort (n=48)	2x lipase elevation cohort (n=24)	Normal lipase cohort (n=21)
Median Age in years (IQR)	61 (50-68)	62 (54-69)	58 (49-67)	63 (54-69)	59 (55-67)
Male N (%)	37 (39%)	72 (47%)	17 (35%)	18 (75%)	13 (62%)
Cancer type (%)					
Genitourinary	27 (29%)	63 (41%)	20 (42%)	14 (58%)	10 (48%)
Melanoma	26 (28%)	28 (18%)	16 (33%)	3 (13%)	5 (24%)
Other	41 (44%)	63 (41%)	12 (25%)	7 (29%)	6 (29%)
ICI Agent (%)					
CTLA-4	8 (9%)	10 (6%)	5 (10%)	2 (8%)	4 (19%)
PD-1	62 (66%)	95 (62%)	28 (58%)	14 (58%)	10 (48%)
Combined	24 (26%)	49 (32%)	15 (31%)	8 (33%)	7 (33%)
ICI doses prior to elevated lipase (IQR)	4 (2-6)	3 (2-8)	5.5 (3-10)	4.5 (3-13)	3 (2-5, to normal lipase)
Median days from ICI to pancreatitis (IQR)	114 (69-264)	113 (52-230)	124 (83-261)	175 (63-348)	95 (47-167, to normal lipase)
ICI discontinued (%)	67 (71%)	74 (48%)	28 (58%)	14 (58%)	11 (52%)
Risk factors (%)					
Alcohol consumption	40 (43%)	69 (45%)	22 (46%)	13 (54%)	12 (57%)
Smoking history	52 (55%)	77 (50%)	24 (50%)	17 (71%)	7 (33%)
Diabetes prior to pancreatitis	16 (17%)	44 (29%)	15 (31%)	6 (25%)	4 (19%)
Drug allergy	56 (60%)	81 (53%)	22 (46%)	10 (42%)	10 (48%)
Prior history of pancreatitis	5 (5%)	6 (4%)	2 (4%)	1 (4%)	0

*Autoimmune pancreatitis, IQR= interquartile range, ICI= immune checkpoint inhibitor, CTLA-4 (cytotoxic T lymphocyte-associated protein 4), PD-1 (programmed cell death receptor-1) and PD-L1 (programmed cell death ligand-1)



[0004] **Figure 1.** a: Serial pancreatic volume in normal lipase, 2x and 3x groups (median, cm3), b: Serial Pancreatic Volume: Single Agent vs Combination in 3x group (median, cm3), c: Percentage of patients with >20% pancreas volume loss in each group.

S5

Predictors of Diabetes Mellitus Following Admission for Acute Pancreatitis: Analysis From a Prospective Observational Cohort

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Introduction: Diabetes mellitus (DM) has become increasingly recognized as a potential sequela of acute pancreatitis (AP). However, specific risk factors for the development of DM after an episode of AP are incompletely defined. We aim to characterize the clinical and biochemical factors associated with the subsequent development of DM.
Methods: We identified patients hospitalized for acute pancreatitis between January 2015 and March 2021 as part of an ongoing prospective observational cohort study. We included patients who had an episode of acute pancreatitis without pre-existing DM to evaluate for our primary outcome defined as the subsequent development of DM (using A1c ≥6.5) following index admission for acute pancreatitis. At least 9 months of follow up were available following hospitalization. Information on demographics, medical history, biochemical data, severity of the pancreatitis episode (Revised Atlanta Classification), and imaging were obtained for analysis. Logistic regression was used for comparative analysis.
Results: A total of 723 unique patients who were hospitalized for acute pancreatitis without pre-existing DM were analyzed. Within the cohort, 360 (50%) were female with a mean age of 43 (±14) years old. A majority of this pancreatitis population was Hispanic (n = 578, 80%), consistent with the overall population demographics of our hospital (63.4% of all admitted patients in our hospital were Hispanic between 2019 and 2020). Most (n = 610, 84.4%) of the cases of pancreatitis were mild. The development of DM was identified in 33 (4.6%) patients within the cohort. Strong predictors for subsequent DM included episodes of moderate to severe pancreatitis [OR 2.94 (1.34-6.42)], history of smoking [2.17 (1.02-4.63)] and cirrhosis [3.23 (1.02-4.63)]. Etiology of pancreatitis was not shown to be a significant predictor in multivariate analysis.
Conclusion: Moderate to severe pancreatitis, smoking, and cirrhosis were found to predict the subsequent development of DM after AP. This suggests that a combination of local injury, toxin exposure, and systemic disease may contribute to the endocrine dysfunction acute pancreatitis patients may develop.

Table 1. Predictors of the development of subsequent diabetes following index admission for acute pancreatitis

	Total Cohort (n=723); n (%)	Subsequent DM Cohort (n = 33); n (%)	Univariate Analysis Odds Ratio (95% CI)	Multivariate Analysis Odds Ratio (95% CI)
Female, n (%)	360 (50.0)	10 (30.3)	0.42 (0.20-0.90)	
Age > 65 years old	47 (6.5)	3 (9.1)	1.47 (0.43-5.00)	
Mean, years (SD)	43 (14)	43 (14)		
Hispanic Ethnicity	578 (80.0)	27 (81.8)	1.14 (0.46-2.80)	
Severity of Pancreatitis				
Mild	610 (84.4)	22 (66.7)	0.35 (0.16-0.74)	
Mod/Severe	113 (15.6)	11 (33.3)	2.88 (1.36-6.12)	2.94 (1.34-6.42)
Local Complications	48 (12.5)	6 (18.2)	2.32 (0.81-6.67)	
Etiology of Pancreatitis				
Alcohol	175 (24.2)	12 (36.4)	2.29 (1.01-5.22)	1.39 (0.56-3.45)
Gallstone	386 (53.4)	12 (36.4)	1	
Other	162 (22.4)	9 (27.2)	1.83 (0.75-4.43)	
Smoking	186 (26.7)	15 (45.5)	2.53 (1.24-5.17)	2.17 (1.02-4.63)
Cirrhosis	41 (5.7)	6 (18.2)	4.16 (1.61-10.73)	3.23 (1.02-4.63)
CHF	18 (2.5)	1 (3.0)	1.24 (0.16-9.59)	
CKD3	27 (3.7)	1 (3.0)	0.80 (0.10-6.07)	

*Variables included in the multivariate model: mod/severe pancreatitis, etiology of pancreatitis, cirrhosis, smoking

S6

Short and Long-Term Outcomes of Pancreatic Duct Disruption and Disconnection in Necrotizing Pancreatitis

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Introduction: Pancreatic duct (PD) disruption and disconnection are less common but important complication of necrotizing pancreatitis (NP). We investigated the short and long-term outcomes of patients with NP complicated by PD disruption and disconnection.
Methods: All index admissions of patients with NP managed between 2009-2019 at our large tertiary academic center were identified. PD disruption was defined as partial interruption of PD; PD disconnection was defined as complete interruption of PD. Diagnosis of PD disruption or disconnection was made by CT, MRI, or EUS during index admission without a previously known diagnosis. Patients with NP and PD disruption or disconnection were compared to control patients with NP and intact PD. Logistic regression models were constructed.
Results: A total of 613 patients were included: 476 patients (78%) with intact PD, 94 patients (15%) had disrupted PD, and 43 patients (7%) had disconnected PD (Table 1A). Patients presenting with a recurrent episode of NP had higher occurrences of disrupted [67 (71%)] and disconnected PD [28 (65%)] than patients with intact PD [263 (55%)] ($P = 0.01$). NP involvement of the pancreatic body was associated with increased occurrence of both PD disruption [27 (29%)] and disconnection [10 (23%)] compared to intact PD [83 (17%)] ($P = 0.03$). Both disrupted and disconnected PD patients had increased development of pancreatic fistulas [15 (16%) and 7 (16%), $P < 0.01$] and distal biliary strictures [29 (31%) and 11 (26%), $P < 0.01$] compared to intact PD patients. Pancreatic fistula formation was 4x more likely in disrupted and disconnected PD patients than in intact PD patients ($P < 0.01$). Additionally, disrupted and disconnected PD patients developed duodenal ulcers 3-6x more frequently than intact PD patients ($P = 0.02$, 0.04) (Table 1B). Patients with disconnected PD had the highest occurrence of acute kidney injury (AKI) [11 (31%), $P < 0.01$], altered mental status (AMS) [15 (35%), $P = 0.04$], readmission [20 (47%), $P < 0.01$], and duodenal ulceration [5 (12%), $P = 0.03$] compared to both intact and disrupted PD patients. Disconnected PD patients had a nearly three-fold increased risk of developing chronic abdominal pain compared to intact and disrupted PD patients (OR 2.91, $P < 0.01$).
Conclusion: Our study characterized the increased short and long-term complications associated with PD disruption and disconnection in NP. Additionally, this study is one of the first to distinguish differences in clinical outcomes between PD disruption and disconnection.

Table 1A. Clinical Characteristics and Short/Long-Term Outcomes of Pancreatic Duct Complications

Factor	Normal (N=476)	Disrupted (N=94)	Disconnected (N=43)	p-value
Clinical Characteristics				
Pancreatitis etiology				
Alcohol	177 (37%)	40 (43%)	12 (28%)	0.26
Galstones	125 (26%)	28 (30%)	15 (35%)	0.41
Hypertriglyceridemia	22 (5%)	0 (0%)	0 (0%)	**0.04**
Idiopathic	124 (26%)	24 (26%)	10 (23%)	0.92
Pancreatic severity[1]				
Moderate	325 (68%)	62 (66%)	28 (65%)	0.85
Severe	151 (32%)	32 (34%)	15 (35%)	0.85
Pancreatic occurrence				
First episode	207 (43%)	27 (29%)	15 (35%)	**0.02**
Recurrence	263 (55%)	67 (71%)	28 (65%)	**0.01**
Chronic	78 (16%)	17 (18%)	7 (16%)	0.92
Location of PFC[2]				
Head	88 (18%)	22 (23%)	12 (28%)	0.22
Body	83 (17%)	27 (29%)	10 (23%)	**0.03**
Tail	86 (18%)	21 (22%)	10 (23%)	0.48
Infected PFC[2]	145 (31%)	32 (34%)	14 (33%)	0.78

Table 1A. (*continued*)

Factor	Normal (N=476)	Disrupted (N=94)	Disconnected (N=43)	p-value
Balthazar score	5.4+2.4	6.2+2.6	6.3+2.5	<**0.01**
SIRS[3]	195(44%)	32 (36%)	18 (53%)	0.19
Acute kidney injury	96 (22%)	7 (8%)	11 (31%)	<**0.01**
Altered mental status	89 (19%)	17 (18%)	15 (35%)	**0.04**
Blood transfused	91 (19%)	29 (31%)	12 (28%)	**0.03**
Gastrointestinal blood	24 (5%)	9 (10%)	4 (9%)	0.16
Length of hospital stay (days)	10 (5-20)	12.5 (6-30)	10 (6.5-18.5)	**0.04**
Clinical outcomes				
Mortality	80 (17%)	16 (17%)	5 (12%)	0.67
Readmission	97 (20%)	23 (24%)	20 (47%)	<**0.01**
Pancreatic fistula	21 (4%)	15 (16%)	7 (16%)	<**0.01**
Internal	16 (3%)	13 (4%)	6 (14%)	<**0.01**
External	8 (2%)	2 (2%)	1 (2%)	0.92
Chronic abdominal pain	27 (6%)	14 (15%)	3 (7%)	**0.01**
Diabetes	62 (13%)	14 (15%)	6 (14%)	0.87
Exocrine insufficiency	18 (4%)	5 (5%)	5 (12%)	0.06
Recurrent acute pancreatitis	144 (30%)	33 (35%)	10 (24%)	0.40
Gastric outlet obstruction	25 (5%)	8 (9%)	7 (16%)	**0.01**
Doudenal ulceration	18 (4%)	2 (2%)	5 (12%)	**0.03**
Biliary tract stricture	44 (9%)	29 (31%)	11 (26%)	<**0.01**
Abdominal thrombosis	105 (22%)	26 (28%)	13 (30%)	0.28
Abdominal pseudoaneurysm	24 (5%)	6 (6%)	1 (2%)	0.60
Follow up duration (days)	612 (107-1583)	574 (243-1586)	481 (180-1388)	0.58

[1]Pancreatitis severity per Atlanta classification;
[2]Pancreatic fluid collection (PFC);
[3]Systemic inflammatory response syndrome (SIRS)

Table 1B. **Prediction of Long-Term Complications**

Complication	Odds Ratio	95% Confidence Interval	p-value
Pancreatic fistula			
Disconnected	4.11	2.03 - 8.32	< **0.01**
Disrupted	4.21	1.68 - 10.57	< **0.01**
Chronic abdominal pain			
Disconnected	2.91	1.46 - 5.79	< **0.01**
Disrupted	1.25	0.36 - 4.29	0.73
Diabetes			
Disconnected	1.18	0.63 - 2.22	0.60
Disrupted	1.08	0.44 - 2.67	0.86
Exocrine insufficiency			
Disconnected	1.43	0.52 - 3.95	0.49
Disrupted	3.35	1.18 - 9.52	**0.02**
Recurrent acute pancreatitis			
Disconnected	1.25	0.78 - 1.99	0.35
Disrupted	0.72	0.34 - 1.50	0.38
Gastric outlet obstruction			
Disconnected	1.68	0.73 - 3.84	0.22
Disrupted	3.51	1.42 - 8.66	**0.01**
Duodenal ulceration			
Disconnected	3.35	1.18 - 9.52	**0.02**
Disrupted	6.05	1.12 - 32.57	**0.04**
Abdominal thrombosis			
Disconnected	1.35	0.82 - 2.23	0.24
Disrupted	1.53	0.77 - 3.04	0.22

S7 **Presidential Poster Award**

Endoscopic Ultrasound-Guided Pancreatic Duct Drainage: A 10-Year Single-Center Experience

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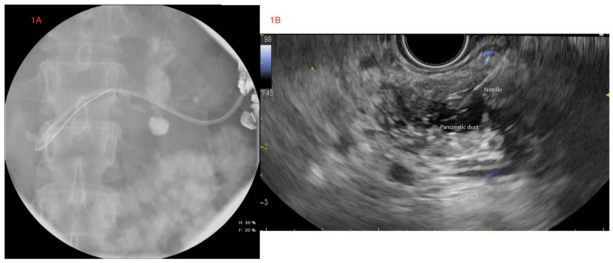
Introduction: EUS-guided pancreatic duct drainage (EUS-PDD) has been recognized as an alternative to surgery for those in whom transpapillary drainage is unfeasible. Previous studies have outlined hurdles that may be encountered with performance of this challenging procedure. These can include lack of stability of the echoendoscope, difficulty with moving a guidewire through the needle for creation of a fistulous tract, or dilation of the fistulous tract.
Methods: We performed a retrospective chart review of cases performed in a tertiary academic center of those in whom EUS-PDD was attempted from January 2010 to November 2021. Documentation and imaging were reviewed up to 12 months after the incident event. Figure demonstrates an example of the performance of the procedure.
Results: 27 patients were identified for whom baseline characteristics are listed in Table: The pancreatic duct was accessed through the stomach in 26 of 27 cases. Technical success was achieved in 22 of these. Access through the duodenum was attempted in a single case but was unsuccessful due to a discontinuous duct. Clinical success was defined in terms of improvement of upstream dilation of the pancreatic duct on imaging and of symptoms, and was achieved in all 22 patients. However, 5 patients had persistent albeit improved abdominal pain after the procedure. One had bleeding that resulted in a self-limited hematoma, which developed an infection with Escherichia coli that resolved after conservative management with antibiotics. Another patient developed a pancreatic duct leak that was managed with cystgastrostomy with eventual resolution. Stent migration occurred in two of which one was monitored conservatively without laboratory or imaging findings of recurrence of pancreatic duct obstruction. In the second case, the patient had recurrence of pain and was found to have migration of the distal end of the stent on endoscopic evaluation. The stent was nonetheless left in place to allow the fistulous tract to remain patent, with eventual removal 16 months later with resolution of stricture (Figure 1).
Conclusion: EUS-PDD is a technically challenging procedure that may nonetheless be the only nonsurgical option in patients in whom endoscopic retrograde pancreatography is infeasible. Technical and clinical success in this cohort is comparable with previous studies. By describing our experience with EUS-PDD, we hope to inform endoscopists of potential hurdles and adverse events that may occur with performance of the procedure (Table 1).



[0007] **Figure 1.** Sample images from one of the cases in this review. 1A shows the fluoroscopic image of the stent placed during EUS-PDD. 1B demonstrates cannulation of the PD through the stomach under ultrasound guidance.

Table 1. Baseline characteristics of patient cohort and post-procedural follow-up

Characteristic	n (%)
Age, mean (SD)	54.6 (12.8)
Gender	
Male	12 (44.4)
Female	15 (55.6)
Indication for EUS-PDD	
Stricture	9 (33.3)
Stone	8 (29.6)
Mixed (stone + stricture)	2 (7.4)
Pancreas divisum	5 (18.5)
Discontinuous duct	3 (11.1)
Approach	
Antegrade	22 (81.5)
Retrograde	5 (18.5)
Technical success	22 (81.5)
Type of stent	
Straight	12 (54.5)
Pigtail	10 (45.5)
Adverse events	
Pancreatitis	3 (11.1)
Stent migration	2 (7.4)
Bleeding	1 (3.7)
Pancreatic duct leak	1 (3.7)
Clinical success	22 (100)
Number of readmissions, mean (SD)	1.7 (SD 2.2)
Re-intervention required	2 (9.1)

S8 Presidential Poster Award

Pilot Trial of Endoscopic Ultrasound-Guided Fiducial Marker Placement to Facilitate Intraoperative Management of Pancreas Tumors

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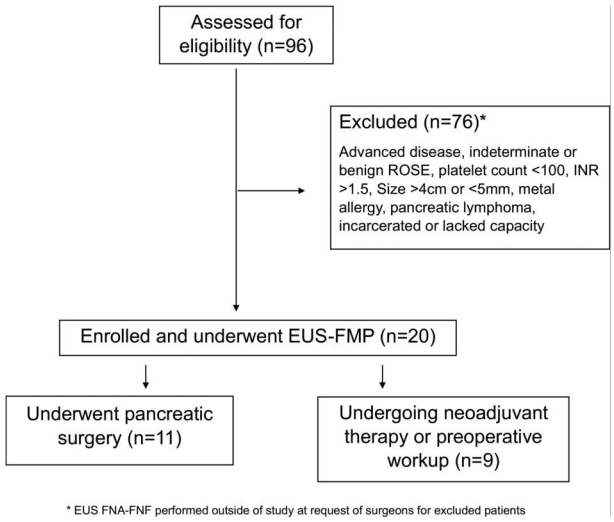
Introduction: Localization of small pancreatic tumors is challenging due to minimally invasive approach pancreatic resection. EUS-guided fiducial marker placement (EUS-FMP) to aid intraoperative identification has been proposed, but its feasibility has not been explored in a controlled prospective manner. The objective of this study is to determine the technical feasibility of placement of EUS guided fiducial placement to mark pancreas tumors in 20 patients and to assess fiducial utility during surgery (ClinicalTrials.gov Identifier: NCT02863783) (Figure 1).
Methods: In this prospective study, we enrolled 20 consecutive patients in two tertiary-care referral medical centers undergoing consideration for pancreatic masses. These patients underwent successful Endoscopic Ultrasound-Fiducial Marker Placement (EUS-FMP) at time of initial confirmation by EUS-Fine needle aspiration (FNA) and Rapid OnSite Evaluation (ROSE). The co-primary outcomes assessed are 1) the successful placement by EUS-FMP, and 2) relative ease of detection of tumor with fiducial during pancreatic surgery based on Likert Scale from 1 to 10. Secondary outcomes include post-EUS-FMP complications including pancreatitis, abdominal pain, infection and bleeding up to 3 months following placement.
Results: Between January 2017 and March 2022, a total of twenty patients, twelve female and eight male, underwent EUS for confirmation of pancreatic tumor and simultaneous EUS-FMP prior to a possible surgery (Table). A total of thirteen patients had pancreatic adenocarcinoma, six had pancreatic neuroendocrine tumors, and one patient had pancreatic solid pseudopapillary neoplasm. The average tumor size was 2.2 ± 1.4 cm. Eleven patients underwent surgery and all patients had negative surgical margins on subsequent pathology. For patients that underwent surgical excision, average tumor size on EUS was 1.7 ± 0.9 cm (range: 0.5–3.6 cm). Average operation time was 429 ± 170 minutes (range: 144–690 minutes). The mean ease of fiducial placement was 9.1 ± 1.3 and mean ease of detection of lesion during surgery was 7.8 ± 2.2. One of twenty patients had pancreatitis.
Conclusion: EUS-FMP is feasible at initial diagnosis of EUS-FNA during pancreatic lesions. Placement of fiducials may improve clinical detection during surgical management. Gold coil fiducial markers are safe prior to surgery with minimal adverse three month follow up outcomes (Table 1).

Table 1. Patient and tumor characteristics of patients undergoing EUS-FMP

Patient and Tumor Characteristics	
Patient characteristics (Mean ± SD)	
Age (y)	58 ± 14
Body Mass Index	25 ± 4
Weight Loss (lb)	16 ± 16
Demographics and comorbidities N (%)	
Female Gender	12 (60)
Hispanic Ethnicity	11 (55)
Diabetes	6 (30)
Endoscopic findings (Mean ± SD)	
Tumor Size (cm)	2.2 ± 1.4
Pancreatic duct diameter (mm)	3.7 ± 1.6
Bile duct diameter (mm)	9.8 ± 6.2
Tumor type N (%)	
Adenocarcinoma	13 (65)
Neuroendocrine	6 (30)
Other	1 (5)



[0008] **Figure 1.** Schematic for screening, enrollment and surgery for EUS-FMP.

S9 Presidential Poster Award

Development of a Clinical Screening Tool for Exocrine Pancreatic Insufficiency in Patients With Chronic Pancreatitis

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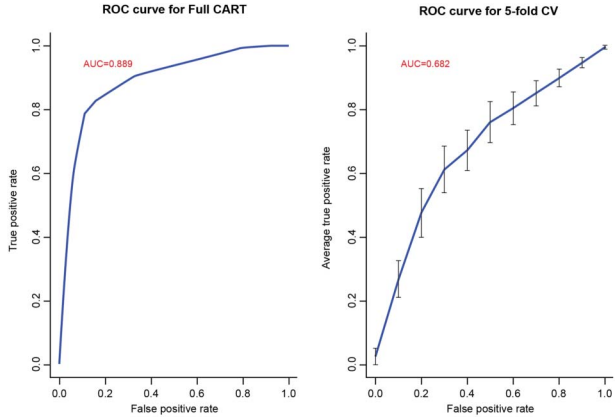
Introduction: No accurate diagnostic tests exist for exocrine pancreatic insufficiency (EPI). As a result, EPI is misdiagnosed, including in chronic pancreatitis (CP), and there is an unmet need for clinical tools to help clinicians evaluate patients with CP at risk for EPI. The aim of this study was to generate a simple clinical tool to predict EPI in patients with CP.
Methods: 49 variables from medical records of CP patients with or without EPI were entered into a Classification and Regression Tree (CART) model using the unimputed full analysis set and 4 other models (Logistic Regression with Least Absolute Shrinkage and Selection Operator regularization, Support Vector Machine, Random Forest, and Gradient Boosting Machine) using imputed full analysis set. EPI misclassification rate (mRate) served as primary metric to train the 5 prediction models. Each model's generalizability was assessed using 5-fold cross-validation and the model that was best suited for clinician use based on mRate and other characteristics was selected.
Results: Records of 274 patients with CP from 6 pancreatitis centers across the US were included. Among the 160 (58%)/114 (42%) patients with EPI/without EPI, 48%/55% were female, mean age was 55/52 years, 76%/69% were white, 29%/52% were never smokers, and 39%/53% were never alcohol users. All 5 prediction models demonstrated similar EPI mRates. The CART model was selected. The final CART decision tree includes 10 variables (in order of importance): smoking, pancreatic atrophy, steatorrhea, alcohol use, hemoglobin, serum vitamin D, pancreatic duct contour change, serum albumin, pancreatic parenchymal calcifications, and use of opioid analgesics. The mRate without and with 5-fold cross-validation of the CART was 0.153 (training error) and 0.314 (prediction error), and the area under the receiver operating characteristic curve was 0.889 and 0.682, respectively (Figure 1). Sensitivity/specificity without/with 5-fold cross validation was 0.888/0.789 and 0.794/0.535, respectively.
Conclusion: A parsimonious set of 10 variables associated with EPI in CP was identified. The CART decision tree demonstrated good prediction performance for EPI and was selected as the most practical screening tool to aid clinicians in the assessment of EPI in patients with CP. Further external validation of the EPI screening tool is planned.



[0009] **Figure 1.** AUC-ROC of the CART model trained on the full analysis set and after 5-fold cross-validation. AUC, area under the curve; CART, classification and regression tree; CV, cross-validation; ROC, receiver operating characteristic.

S10 Presidential Poster Award

Mortality in Acute Necrotizing Pancreatitis at Baseline-B Scoring for the Inpatient Mortality of Acute Necrotizing Pancreatitis

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Introduction: Acute necrotizing pancreatitis (ANP) can result in significant healthcare burden. It is essential to accurately identify patients with a high likelihood of mortality promptly to determine the need for aggressive measures. The present study aimed to develop a new scoring system based on data from the United States population.
Methods: We retrospectively analyzed 22,980 patients diagnosed with acute necrotizing pancreatitis (ANP) using the National Inpatient Sample (NIS) in the validation cohort. The mortality in acute necrotizing pancreatitis at baseline (MANP)-B scoring system was derived using multivariable cox regression analysis and validated using receiver operating characteristic curves in a validation cohort.
Results: Six variables were selected for incorporation into the MANP-B score, including age ≥60 years (aHR 2.8 [95% CI 2.03–3.8, $P < 0.001$), Peripheral vascular disease (aHR 1.79 [95% CI 1.1–2.8, $P < 0.001$), Chronic kidney disease or ESRD (aHR 1.54 [95% CI 1.09–2.2, $P < 0.001$), Chronic liver disease (aHR 1.60 [95% CI 1.17–2.17, $P < 0.001$), Disorders of coagulation (aHR 1.97 [95% CI 1.34–3.24, $P < 0.001$) and fluid or electrolyte imbalance (aHR 2.1 [95% CI 1.34–3.24, $P < 0.001$). Each variable was allotted one point except age and fluid/electrolyte imbalance which were allotted two points due to higher hazard ratios. The new scoring system yields a total maximum score of 8 points. Based on the calculated highest sensitivity and specificity values from the receiver operating characteristic (ROC)curves, the determined cut-off values for predicting ANP inpatient mortality at 30-day periods was 4 points using Liu index (Sensitivity 70.14%, Specificity 75.82%) and 3 points for mortality at 7-day periods was 4 points using Liu index (Sensitivity 69.70%, Specificity 78.60%). The area under the curve (AUC) using the ROC curve of derivation cohort was 0.7965 (95% CI 0.74766–0.84526, $P < 0.01$) for 7-day periods. The AUC of the validation cohort 0.8005 (95% CI 0.74612–0.85479, $P < 0.01$) for 7-day periods. The area under the curve (AUC) using the ROC curve of derivation cohort was 0.7905 (95% CI 0.7905–0.81896, $P < 0.01$) for 30-day periods. The AUC of the validation cohort 0.8190 (95% CI 0.78883–0.84910, $P < 0.01$) for 30-day periods.
Conclusion: This system can be used as an objective method for predicting seven and 30 day all cause mortality for ANP hospitalizations on admission.

Table 1. Biodemographic Characteristics of Hospitalizations for Acute Necrotizing Pancreatitis (ANP) for the Derivation Cohort

Patient characteristics	ANP-associated survivor cohort	ANP -associated mortality cohort	P value
Total hospitalizations	21875	1105	
Gender			P = 0.5
Male	13825 (63%)	720 (65%)	
Female	8050 (37%)	385 (35%)	
Mean Age (years) ± SE	51.69 ± 0.27	61.40 ± 1.1	P < 0.001
Race/ethnicity			P = 0.06
White	14165 (67%)	785 (73%)	
Black	2660 (13%)	100 (9%)	

Table 1. (*continued*)

Patient characteristics	ANP-associated survivor cohort	ANP -associated mortality cohort	P value
Hispanic	2825 (13%)	90 (8%)	
Asian or Pacific Islander	750 (4%)	60 (6%)	
Native American	255 (1%)	5 (< 1%)	
Other	600 (3%)	30 (3%)	
Elixhauser Comorbidity Index score			P < 0.001
0	720 (3%)	0 (0%)	
1	2070 (9%)	10 (1%)	
2	3350 (15%)	30 (3%)	
≥3	15735 (72%)	1065 (96%)	
Median annual income in patient's zip code, US$			P = 0.2
$1-24,999	5970 (28%)	355 (33%)	
$25,000-34,999	5395 (25%)	295 (27%)	
$35,000-44,999	5615 (26%)	245 (23%)	
45,000 or more	4560 (21%)	190 (18%)	
Insurance type			P < 0.001
Medicare	6200 (30%)	550 (51%)	
Medicaid	5080 (24%)	135 (13%)	
Private	8065 (39%)	325 (30%)	
Uninsured	1595 (8%)	60 (6%)	
Hospital characteristics			
Hospital region			P = 0.8
Northeast	3690 (17%)	175 (16%)	
Midwest	5300 (24%)	290 (26%)	
South	7830 (36%)	370 (33%)	
West	5055 (23%)	270 (24%)	
Hospital status			P = 0.3
Rural	910 (4%)	25 (2%)	
Urban non-teaching	2880 (13%)	135 (12%)	
Urban teaching	18085 (83%)	945 (86%)	

S11 Presidential Poster Award

Short- and Long-Term Outcomes of Necrotizing Pancreatitis After Endoscopic, Percutaneous and Surgical Management

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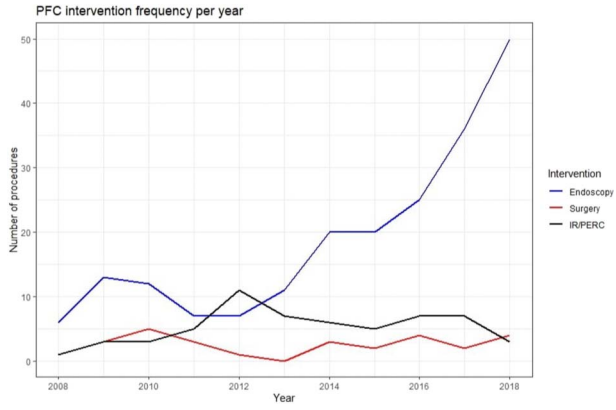
Introduction: Pancreatic fluid collections (PFCs) are a common complication of necrotizing acute pancreatitis (NAP) and are a significant cause of morbidity and mortality. Multiple intervention approaches are utilized in a step-up fashion for the management of symptomatic PFC in patients with NAP. The aim of this study is to analyze the short- and long-term clinical outcomes in these patients treated endoscopically, percutaneously, or surgically.
Methods: This retrospective cohort study was conducted in a large tertiary center. Patients with NAP complicated by PFC were selected for possible inclusion. Short-term (<90 days) and long-term (>90 days) clinical outcomes including mortality were compared between three groups; the first group was defined as patients who underwent endoscopic ultrasound-guided drainage alone or in combination with percutaneous drainage, and the second group are patients who underwent surgery only and the third group is patients who underwent percutaneous drainage alone.
Results: A total of 637 patients with NAP complicated by PFC on imaging were included, of those; 318 patients underwent an intervention for management of PFC from 2009 to 2019 with a median follow-up time of 17 months. Group 1 had 229 patients, group 2 had 30 patients and group 3 had 59 patients. Patients in groups 2 and 3 had longer hospital stays (18 and 19 days, respectively, $P < 0.05$), were more likely to be transferred to ICU (33% and 24%, respectively, $P < 0.05$) and had severe pancreatitis at the time of presentation (57% and 59%, respectively, $P < 0.05$). Patients in group 1 were more likely to present with recurrent episodes of AP (74%, $P < 0.05$). Alcoholic pancreatitis was the most common etiology overall and in group 1 39%, $P < 0.05$. The infected collection was the most common indication for drainage overall, and in group 3 (64%, $P < 0.05$). Transfer to ICU was lower in group 1 (14%, $P < 0.05$). 90-day mortality was lower in group 1 with 5.2% ($P < 0.05$). No difference was noted in long-term complications between the different groups.
Conclusion: Endoscopic management alone or in combination with percutaneous drainage of symptomatic PFC in patients with necrotizing acute pancreatitis leads to a shorter hospital stay, and lower mortality when compared to percutaneous drainage alone or surgery. There was no difference in the long-term complications including chronic pancreatitis, EPI, new-onset DM, and chronic abdominal pain across these groups. A multidisciplinary approach is recommended in NAP.

Table 1. Patient Characteristics, Short- and Long-Term Complications

Characteristics	Overall (N=318)	Intervention Modality			p-value
		Endoscopy Alone or Endoscopy+IR/PERC (N=229)	Surgery Alone (N=30)	IR/PERC Alone (N=59)	
Body Mass Index, Median (IQR)	26 (23-32)	26 (23-30)	27 (23-32)	32 (26-36)	< 0.001
Severe Acute Pancreatitis on Presentation, n (%)	103 (32)	51 (22)	17 (57)	35 (59)	< 0.001
Occurrence, n (%)					< 0.001
First	98 (31)	58 (25)	12 (40)	28 (47)	
Recurrent	216 (68)	170 (74)	17 (57)	29 (49)	
Chronic	4 (1.3)	1 (0.4)	1 (3.3)	2 (3.4)	
Alcoholic Pancreatitis, n (%)	104 (33)	90 (39)	4 (13)	10 (17)	< 0.001
Multiple Pancreatic and Peripancreatic Fluid Collections, n (%)	180 (57)	129 (57)	10 (33)	41 (69)	0.005
PFC Size on CT Scan, Median (IQR)	89 (56-128)	78 (50-112)	120 (89-138)	126 (82-158)	< 0.001

Table 1. (*continued*)

Characteristics	Overall (N=318)	Intervention Modality: Endoscopy Alone or Endoscopy+IR/PERC (N=229)	Intervention Modality: Surgery Alone (N=30)	Intervention Modality: IR/PERC Alone (N=59)	p-value
Retroperitoneal PFC with Pelvic Extension, n (%)	55 (17)	29 (13)	6 (20)	20 (34)	< 0.001
Drain Indication					
Abdominal Pain, n (%)	89 (28)	71 (31)	13 (43)	5 (8.5)	< 0.001
Infected Collection, n (%)	102 (32)	50 (22)	14 (47)	38 (64)	< 0.001
Gastric Outlet Obstruction, n (%)	35 (11)	32 (14)	1 (3.3)	2 (3.4)	0.023
Length of Hospital Stay, Median (IQR)	10 (6-22)	8 (5-18)	18 (10-29)	19 (11-38)	< 0.001
Transferred to ICU, n (%)	65 (20)	32 (14)	11 (37)	22 (37)	< 0.001
90-Day Mortality, n (%)	26 (8.2)	12 (5.2)	3 (10)	11 (19)	0.004
Long-Term Complications					
Pancreatic Fistula, n (%) Internal Fistula External Fistula	 31 (86) 5 (41)	 20 (83) 4(17)	 1 (50) 1 (50)	 10 (100) 0 (0)	0.10
New Chronic Pancreatitis, n (%)	36 (12)	28 (12)	2 (6.9)	6 (11)	0.8
Chronic Abdominal Pain, n (%)	25 (7.9)	19 (8.3)	2 (6.7)	4 (6.8)	>0.9
New-Onset Diabetes, n (%)	50 (16)	32 (14)	7 (23)	11 (19)	0.3
Exocrine Insufficiency, n (%)	17 (5.3)	11 (4.8)	2 (6.7)	4 (6.8)	0.6
Pancreatic Cancer, n (%)	5 (1.6)	4 (1.7)	0 (0)	1 (1.7)	>0.9
Duodenal Ulceration, n (%)	16 (5.0)	14 (6.1)	1 (3.3)	1 (1.7)	0.5
Abdominal Thrombosis, n (%)	78 (25)	63 (28)	3 (10)	12 (20)	0.079
Abdominal Pseudoaneurysm, n (%)	21 (6.6)	18 (7.9)	0 (0)	3 (5.1)	0.3
Biliary Tract Stricture, n (%)	67 (21)	53 (23)	4 (13)	10 (17)	0.3



[0011] **Figure 1.** PFC Interventions Frequency Per Year

S12 Outstanding Research Award in the Biliary/Pancreas Category
Presidential Poster Award

Impact of Pancrelipase on Stool Frequency and Consistency in Patients With Exocrine Pancreatic Insufficiency Due to Chronic Pancreatitis or Pancreatic Surgery: Analysis of Randomized Trial Patient-Reported Daily Symptoms

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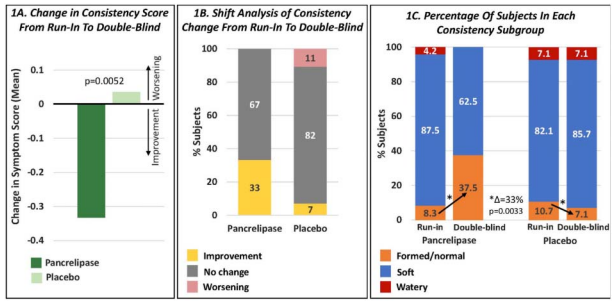
Introduction: Patients (pts) with exocrine pancreatic insufficiency (EPI) suffer from maldigestive symptoms that negatively impact quality of life. Pancreatic enzyme replacement therapy (PERT) is the mainstay of EPI treatment. This study assessed the impact of PERT on EPI symptoms.
Methods: Post-hoc analysis of a double-blind, randomized phase 3 trial in pts with EPI due to chronic pancreatitis (CP) or pancreatic surgery (PS). After a 5-day placebo (PBO) run-in period, pts were randomized to pancrelipase (PL, 72,000 LU/meal; 36,000 LU/snack) or PBO for a 7-day double-blind period. Pts completed a daily diary, reporting stool frequency (numbers of stools), stool consistency (–1 = hard; 0 = formed/normal; 1 = soft; 2 = watery), flatulence, and abdominal pain (0 = none; 1 = mild; 2 = moderate; 3 = severe). Average of daily reported symptoms was calculated in each period and mean results are presented for each treatment group. Two-sample *t*-tests compared mean change from run-in to double-blind between PL and PBO. Population level marginal difference-in-difference models were used to assess differences in the change in the % of pts in symptom subgroups from run-in to double-blind period between PL and PBO.
Results: 52 pts (24 PL; 28 PBO) were included (75% CP; 25% PS). Mean age was 51.7 yrs for PL and 50.4 yrs for PBO, 75% and 68% were male, respectively. A mean reduction of 1.2 stools/day (95% CI –2.284, –0.128) with PL vs PBO ($P = 0.0296$) was observed (Table). A mean change in stool consistency score of –0.369 (95% CI –0.623, –0.115) with PL vs PBO ($P = 0.0052$) was observed (Figure 1A). A shift analysis of change in stool consistency from run-in to double-blind period for PL/PBO demonstrated improvement in 33%/7% pts, no change in 67%/82%, and worsening in 0%/11%, respectively (Figure 1B). In the formed/normal stools subgroups, absolute % change of pts from run-in to double-blind was 33% greater for PL vs PBO ($P = 0.0033$), and PL eliminated watery stools (Figure 1C). There was a trend towards improvement in flatulence and abdominal pain for PL vs PBO.
Conclusion: Stool frequency and consistency significantly improved in pts with EPI due to CP or PS during 1-week treatment with PL vs PBO. PL reduced mean number of stools by 1.2/day, eliminated watery stools, and increased number of pts with formed stools by 33% vs PBO. Patient-reported stool frequency and consistency could be utilized with nutritional markers to assess clinical efficacy of PERT for treatment of EPI due to CP or PS.

Table 1. Change in Stool Frequency

	Pancrelipase (n=24)	Placebo (n=27)
Number of stools/day, mean (SD)		
Run-in placebo period	3.7 (2.41)	3.5 (1.40)
Double-blind treatment period	2.1 (0.85)	3.1 (1.33)
Change in number of stools/day, mean (SD)		
From run-in to double-blind period	–1.6 (2.39)	–0.4 (1.05)
Treatment difference (95% CI)	–1.206 (–2.284, –0.128) p=0.0296	



[0012] **Figure 1.** Change in Stool Consistency

S13 Presidential Poster Award

Early Feeding Rates in Acute Pancreatitis Is Associated With Decreased Length of Hospitalization

Melanie Pascal, MD[1], Samantha Magier, MD, ME[2], Ahmad Nawaz, MD[2], Thiruvengadam Muniraj, MD, PhD[2], Kenneth W. Hung, MD, MS[2].
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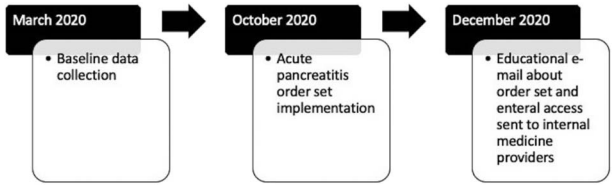
Introduction: Guidelines recommend that patients with acute pancreatitis (AP) receive early oral or enteral feeding within 24 hours of diagnosis rather than keeping patients nil per os. Early feeding reduces rates of multiorgan failure and necrotizing pancreatitis. We sought to understand whether early feeding affected length of hospitalization for AP, identify factors associated with early feeding, and determine whether an order set for acute pancreatitis would improve early feeding rates.
Methods: We identified adult patients (age ≥18 years) hospitalized at Yale New Haven Hospital, a tertiary academic medical center with a diagnosis of AP by ICD-10 code and/or serum lipase ≥3 times the upper limit of normal from April 2019-January 2021 and reviewed their charts to confirm the diagnosis of AP and the timing of diet order entry. We created and implemented an electronic medical record order set for AP including guidelines and diet, imaging, and consult orders in October 2020 (Figure 1). E-mails were sent to notify hospitalists and medicine residents of the order set. Primary outcome was the proportion of patients who received a diet order within 24 hours following diagnosis of AP. Logistic regression was performed to evaluate the relationship between early feeds within 24 hours of AP diagnosis and patient factors.
Results: A total of 707 patients were hospitalized with AP, and 496 patients (70.2%) had enteral feeds within 24 hours of AP diagnosis. Patients receiving early feeds had shorter hospitalizations compared to patients receiving delayed feeds (mean [SD] 8.4 days [12.8] versus 5.4 days [8.2]) ($P < 0.01$) and were less likely to be hospitalized in the intensive care unit (ICU) (odds ratio 0.45, 95% confidence interval 0.32–0.63) (Table 1). There was no difference in gender, race, ethnicity between patients who received feeds within 24 hours of AP diagnosis and those who received feeds >24 hours after AP diagnosis. Implementation of an order set containing guidelines and orders for diet, imaging, and consults did not significantly improve early feeding within 24 hours of AP diagnosis (70.5% versus 68.9%) likely due to limited usage by providers.
Conclusion: Early enteral nutrition is associated with shorter hospitalizations among patients with AP. Patients receiving oral or enteral feeds within 24 hours of AP diagnosis were less likely to be in the ICU. Durable and effective quality improvement initiatives are needed to improve rates of early enteral feeding in patients with AP.

Table 1. Factors associated with early enteral feeding in acute pancreatitis

Variable	Odds Ratio	95% Confidence Interval	p-value
Intensive Care Unit Hospitalization	0.45	0.32-0.63	< 0.01
Age	1.00	0.99-1.01	0.99
Race			0.92
White/Caucasian	reference	reference	
Black/African-American	0.92	0.63-1.36	
Other/Not Listed	0.98	0.63-1.52	
Ethnicity			0.33
Non-Hispanic	Reference	Reference	
Hispanic	1.16	0.75-1.79	
Other/Not Listed	0.22	0.02-2.40	



[0013] **Figure 1** Timeline of Interventions

S14 Presidential Poster Award

Incidence and Risk Factors for Deep Vein Thrombosis and Pulmonary Embolism in Necrotizing Pancreatitis: An Underappreciated Sequalae

David Jonason, MD[1], Satish Munigala, MBBS, MPH[2], Gaurav Suryawanshi, MD[1], Amanda Hjeltness, PA-C[1], Stuart K. Amateau, MD, PhD[1], Nabeel Azeem, MD[1], Shawn Mallery, MD[1], Martin L. Freeman, MD[1], Guru Trikudanathan, MD[1].
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Introduction: Inflammatory dysregulation of the coagulation cascade and vascular stasis in hospitalized necrotizing pancreatitis (NP) patients serve as a milieu for venous thromboembolism which is often underrecognized. We aimed to identify the incidence and independent risk factors for deep vein thrombosis (DVT) and pulmonary embolism (PE) in our NP cohort.
Methods: All adult NP patients hospitalized at our center between 2009 and 2022 were identified from a prospective database and categorized into two groups based on development of DVT or PE (cases) or not (controls), within 6 months after NP hospitalization. Baseline data included demographics, ASA score, SIRS and organ failure on admission and at 48 hours, interventions (endoscopic, percutaneous or surgical), length of stay, transfer status, need for ICU, clinical and imaging characteristics and anticoagulation during admission. Univariable and multivariable analysis identified independent predictors for DVT and PE. $P < 0.05$ was considered significant.
Results: Among 641 NP patients, 510 patients [males 349 (68%), median age 52 years (IQR 38–64)] were eligible for inclusion. DVT/PE developed in 62 (12%) patients; 26 DVT (5%), 22 PE (4%) and 14 with both (3%) after a median 17 (IQR 7–34) days from NP. Demographics were similar between groups though cases were older with more comorbidities (higher ASA) including a personal history of cancer. Nearly all patients [n = 506, (99%)] including all cases were on DVT prophylaxis (pharmacologic or mechanical) during hospitalization. Significant clinical and imaging predictors for DVT/PE on multivariable analysis were age (>50 years) [OR 2.1 (1.05–4.13)], personal history of cancer [OR 5.02 (1.30–19.35)], peripancreatic extent [OR 7.15 (3.7–13.81)], infected necrosis [OR 2.00 (1.01–3.96)] and increased length of stay (LOS) [OR 1.01 (1.00–1.02)].
Conclusion: Incidence of DVT/PE in our NP cohort was 12% (comparable to incidence rates in cancer and IBD), usually diagnosed within one month of NP hospitalization. Age >50, peripancreatic necrosis extent, personal history of cancer, infected necrosis, and prolonged hospitalization were independent risk factors. This high-risk group of patients may benefit from intensified DVT prophylaxis during hospitalization and closer follow up after discharge (Table 1).

Table 1. Univariable and Multivariable Analysis of Clinical and Imaging Predictors for DVT and PE in NP

Characteristic	Univariable OR (95% CI)	P-Value	Multivariable OR (95% CI)	P-Value
Age (>50 years)	2.26 (1.26-4.02)	0.006	2.1 (1.05-4.13)	**0.035**
Personal h/o cancer	3.89 (1.41-10.78)	0.009	5.02 (1.30-19.35)	**0.019**
ASA score	2.21 (1.46-3.34)	0.0002	1.29 (0.72-2.32)	0.397
Persistent multiorgan failure	3.04 (1.59-5.80)	< 0.0001	2.05 (0.81-5.17)	0.128
Peripancreatic involvement	6.94 (3.94-12.21)	< 0.0001	7.15 (3.7-13.81)	< **0.0001**
Infected necrosis	3.07 (1.74-5.44)	0.0001	2.00 (1.01-3.96)	**0.046**
TLOS	1.03 (1.02-1.03)	< 0.0001	1.01 (1.00-1.02)	**0.016**
Collection Size	1.07 (1.02-1.12)	0.005	1.01 (0.95-1.08)	0.663

ASA- American society of anesthesiologists; TLOS- total length of stay

S15 Presidential Poster Award

Index Admission Cholecystectomy in Uncomplicated Acute Biliary Pancreatitis

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[1]NYU Grossman School of Medicine, Brooklyn, NY; [2]One Brooklyn Health Interfaith Medical Center, Brooklyn, NY; [3]Rochester General Hospital, Rochester, NY; [4]One Brooklyn Health System, Brooklyn, NY; [5]The Research Institute at Nationwide Children's Hospital, Columbus, OH; [6]West Virginia University, Charleston, WV; [7]Banner MD Anderson Cancer Center at Banner University Medical Center, Phoenix, AZ.

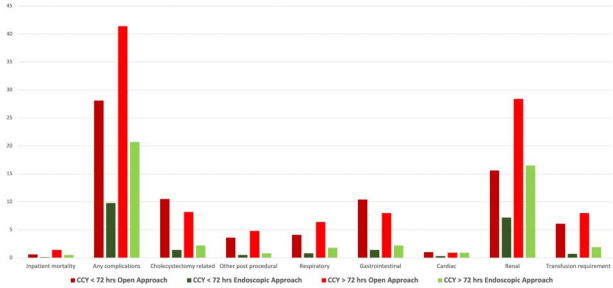
Introduction: Despite an increase in acute biliary pancreatitis hospitalizations (ABPH) index admission cholecystectomy (I-CCY) has remained sub-optimally performed nationwide as low as 50-55%.
Methods: Nationwide Inpatient Sample (NIS) 2016-2019 yy was analyzed for adult ABPH and associated I-CCY. ICD-10 diagnosis, procedure, and complication codes were used and separate sociodemographic, and outcomes analyses performed for open (O-CCY) and laparoscopic CCY (L-CCY). I-CCY timing was further divided into early <72 hrs and delayed >72 hrs groups. The primary outcome was defined as the percentage of I-CCY performed; Mortality, length of stay (LOS), mean charges and complications were analyzed.
Results: Out of total 274,580 ABPH, 162,590 patients (59.2%) had I-CCY, 72.5% (117,945) performed in <72 hrs and 25% (40,040) >72 hrs. 156,125 patients (96%) had L-CCY, and 6,465 patients (4%) had O-CCY; 64.4% and 75.1% performed in <72 hrs, while 35.5% and 25% >72 hrs for O-CCY and L-CCY respectively. The highest number of L-CCY was performed on day 2 (28.1%) and >5 days (22.7%) for O-CCY. Mortality was 0.1% and 0.5% for the early and delayed group. Overall and all types of complications were higher in the delayed CCY group 10.4% vs. 21.8%, with renal complications being significantly higher at 7.5% vs. 17% (Table 1). Both O-CCY and L-CCY had higher complications when performed >72hrs: 28% vs. 41% for open CCY and 10% vs. 21% for L-CCY. Sociodemographic analysis revealed that males were more likely to receive O-CCY and females were significantly higher proportionately to receive L-CCY. (O-CCY: M:F 54% vs. 46% L-CCY: 37% vs. 63%). Asian, Native Americans, and Black patients were least likely to receive L-CCY, and patients with public insurance and living in the lowest income areas were more likely to receive O-CCY (Figure 1).
Conclusion: Our analysis has demonstrated an increased total number of I-CCY in uncomplicated ABP across the U.S. but only up to 59% which remains suboptimal. Mortality and all-cause complications are higher in patients with ABP who undergo delayed I-CCY. The optimal timing for CCY during hospital admission for ABP is < 72hrs from admission. Racial, gender and economic disparities exist in the type of I-CCY performed. I-CCY decreases the recurrence of ABP, improves outcomes and survival, and its increased utilization should be strongly encouraged (Table 1).

Table 1. Timing of Index Cholecystectomy by days from Admission in Uncomplicated Acute Biliary Pancreatitis Hospitalizations, NIS 2016-2019

		Open Cholecystectomy	%	95% CI	Laparoscopic Cholecystectomy	%	95% CI
Cholecystectomy (CCY)	Total	6,465	100.0	100.0	156,125	100.0	100.0
Days of CCY from admission	CCY 0 day	920	14.8	12.9-16.8	10,875	7.2	6.8-7.5
	CCY 1 day	780	12.6	10.7-14.4	27,380	18.0	17.6-18.5
	CCY 2 day	1,150	18.5	16.3-20.7	42,645	28.1	27.6-28.6
	CCY 3 day	1,150	18.5	16.4-20.7	33,045	21.8	21.3-22.3

Table 1. (*continued*)

	Open Cholecystectomy	%	95% CI	Laparoscopic Cholecystectomy	%	95% CI
CCY 4 day	795	12.8	11.0-14.7	17,950	11.8	11.4-12.2
CCY 5+ days	1,405	22.7	20.4-25.0	19,890	13.1	12.7-13.5



[0015] **Figure 1.** Mortality and Complications by Timing of Index Admission Cholecystectomy, NIS 2016-2019

S16 Presidential Poster Award

Partially-Covered versus Uncovered: Self-Expandable Metal Stents in the Palliative Treatment of Malignant Distal Biliary Obstruction

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Introduction: Self-expandable metal stents (SEMSs) are commonly used to relieve malignant biliary obstruction. Covered self-expandable metal stents (CSEMSs) were introduced to improve the patency of SEMSs by preventing tissue ingrowth. Unlike uncovered self-expandable metal stents (USEMSs), which are integrated into the tumor or duct wall, CSEMSs do not embed, but has an increased risk of migration. Although several retrospective studies comparing these two types of stents for malignant biliary obstruction have shown longer patency with CSEMSs, prospective, randomized trials have not been able to confirm these results. Partially covered self-expandable metal stents (PCSEMSs) has been introduced to reduce migration with advantage of CSEMSs preventing tumor ingrowth. The aim of this study was to compare stent patency and clinical outcomes between PCSEMSs and USEMSs in malignant biliary obstruction.
Methods: A total of 91 patients with malignant biliary obstruction were randomly assigned to either PCSEMSs (n = 43) or USEMSs (n = 48) for palliation of biliary obstruction. Duration of stent patency, procedure-related adverse events and intervention for stent occlusion were evaluated.
Results: The mean period of follow-up was 132.7 ± 114.3 days for the PCSEMSs group and 193.9 ± 136.6 days for the USEMSs group ($P = 0.02$). Stent occlusion occurred in 5 patients after 97.2 ± 30.3 days in the PCSEMSs group and in 15 patients after 146.0 ± 67.8 days in the USEMSs group ($P = 0.141$). Cumulative stent patency estimated by the Kaplan-Meier method showed no significant difference between PCSEMSs group and USEMSs group ($P = 0.15$). There was no procedure-related adverse event in both groups. Additionally, migration of the stent occurred in two patients of PCSEMSs group.
Conclusion: There was no significant difference in stent patency time between PCSEMSs and USEMSs in the palliative treatment of malignant distal biliary obstruction, although there was the trend that more stent occlusion occurred in USEMSs group than PCSEMSs group. Migration of PCSEMS occurred in two patients. Further studies with large population are warranted to validate these results.

S17

Radiofrequency Ablation With Stent versus Stent-Only for Biliary Tree Drainage in Patients With Malignant Biliary Strictures: A Systemic Review and Meta-Analysis

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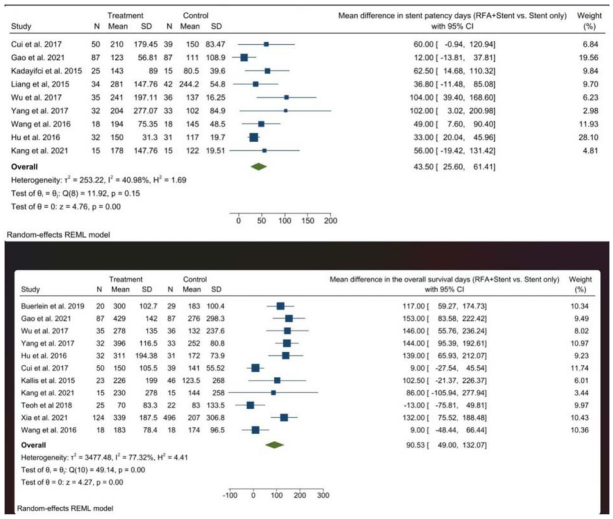
[1]University of Kansas, Wichita, KS; [2]Essen Healthcare System, New York, NY; [3]Beth Israel Deaconess Medical Center, Boston, MA.

Introduction: Cholangiocarcinoma and adenocarcinoma are major causes of malignant biliary obstruction. Palliative stent placement is the general management strategy for biliary drainage in unresecTable malignant biliary strictures. Studies have shown that Endoscopic Radiofrequency Ablation (RFA) can improve the overall stent patency and survival time among patients with malignant biliary tumors. Therefore, our study aimed to assess the efficacy and safety of biliary stenting with RFA compared with stents alone to treat malignant biliary strictures.
Methods: A systemic search of major databases through April 2022 was done. All original studies comparing radiofrequency ablation along with stenting versus stenting alone for treating malignant biliary tumors were included. The primary outcomes of interest were the difference in the mean stent patency and overall survival days between the two groups. Secondary outcome of interest was adverse events between the two groups. The mean difference in the stent patency and overall survival days was pooled by using a random-effect model. We calculated the odds ratio to compare the adverse events between the two groups. All analyses were done by using STATA 17 software.
Results: A total of 13 studies with 1339 patients were identified, reporting follow-up data of the original research. The pooled weighted mean difference in stent patency was 43.50 days (95% confidence interval [CI], 25.60–61.41), favoring the RFA plus stenting. Moreover, the pooled weighted mean difference in overall survival was 90.53 days (95% CI, 49.00–132.07), showing improved survival in RFA group. Our analysis showed no statistically significant difference OR 1.07 (95% CI, 0.80–1.34) between the RFA and stent-only group regarding adverse events such as post-procedural abdominal pain, and cholangitis, pancreatitis, and acute cholecystitis.
Conclusion: Our systematic review shows that RFA along with stent is safe and is associated with improved stent patency and overall patient survival in malignant biliary strictures. More robust prospective studies should assess this association further (Figure 1).



[0017] **Figure 1.** Forest Plots of the Mean Difference between RFA with stent and stent only (Stent Patency and Overall Survival)

S18

Overall Survival Trends of Pancreatic Cancer, a Surveillance, Epidemiology and End Results Population-Based Data (2002–2016)

Dina Elantably, MD[1], Abdul Rahman Al Armashi, MD[2], Faris Hammad, MD[3], Anas Al Zubaidi, MD[4], Akram Alkrekshi, MD[1].
[1]MetroHealth Medical Center/Case Western Reserve University, Cleveland, OH; [2]University Hospitals Case Western Reserve University, Cleveland, OH; [3]St. Vincent Charity Medical Center, Cleveland, OH; [4]Johns Hopkins University, Baltimore, MD.

Introduction: Pancreatic cancer (PC) is the third leading cause of cancer-related deaths in the United States. The general 5-year survival rate for people with pancreatic cancer in the US is 11%. Survival rates and outcomes are based on many factors, including the specific stage of the disease when it is diagnosed. We conducted an evaluation of survival trends after PC diagnosis overall and by stage of the disease.
Methods: We performed a database query into the Surveillance, Epidemiology, and End Results (SEER) Program 17 registry. Between 2002 and 2016, we included individuals diagnosed with pancreatic cancer to evaluate 5-year survival trends. The results were observed using the Actuarial–Ederer II method for cumulative expected survival (absence of other causes of death).
Results: Cancer stage was an important factor in explaining variability in 5-year survival. The SEER localized stage of pancreatic cancer showed an improvement of the overall 5-year survival between 2012 and 2016 (38.8%) compared to those between 2002 and 2006 (21%). The Regional stage also showed an improvement of the overall 5-year survival between 2012 and 2016 (12.9%) compared to those between 2002 and 2006 (8.3%). However, in the distant stage, the 5-year overall survival showed a minimal improvement from 2 % to 2.8%.
Conclusion: From 2002 to 2016, the overall survival of patients with PC has improved across all the SEER stages, with a remarkable improvement by 17.8% in the localized stage and 4.6% in the regional stage. This improvement may reflect the enhanced diagnostic and treatment modalites. The negligible improvement in the distant stage is partly explained by the poor overall prognosis and the use of only supportive care (Table 1).

Table 1. Cumulative Summary of 5 Year Overall Survival Based on Staging

Cumulative Summary of 5 Year Overall Survival Based on Staging	N	Observed Survival
Localized 2012-2016		
60 months	5,130	38.80%
Localized 2007-2011		
60 months	3,674	26.00%
Localized 2002-2006		
60 months	1,848	21.00%
Regional 2016-2012		
60 months	13,430	12.90%
Regional 2007-2011		
60 months	11,360	10.00%
Regional 2002-2006		
60 months	5,609	8.30%
Distant 2012-2016		
60 months	23,601	2.80%
Distant 2007-2011		
60 months	20,578	2.40%
Distant 2002-2006		
60 months	11,314	2.00%

S19

Investigation of Linerixibat 40 Mg BID for Cholestatic Pruritus of Primary Biliary Cholangitis; Further Data From the Phase 2b GLIMMER Study to Support the Phase 3 GLISTEN Study

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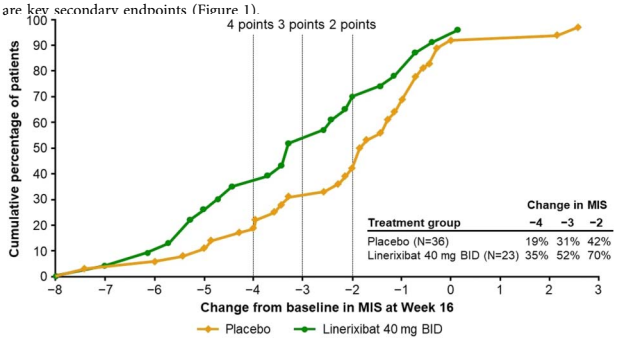
[1]GSK, London, United Kingdom; [2]GSK, Research Triangle Park, NC; [3]GSK, Collegeville, PA.

Introduction: Reduction of pruritic bile acids (BA) in circulation is under investigation for the treatment of cholestatic pruritus in PBC. Due to lack of previously approved therapies, what constitutes a meaningful within-patient change in itch has not been determined. GLIMMER was a Phase 2b randomized, double-blind, placebo-controlled, dose response study of linerixibat, a minimally absorbed ileal BA transporter inhibitor, in PBC patients with moderate to severe pruritus. Data from GLIMMER were reanalyzed to look at multiple itch responder definitions in the linerixibat 40mg twice daily (BID) group, the Phase 3 dose. Changes in total serum BA (TSBA) are also reported.
Methods: Patients (N = 147) assessed itch daily on a 0–10 numeric rating scale. Proportion of responders was assessed using reductions in monthly itch score (MIS) at Week 16 compared to baseline (BL). An empirical cumulative distribution function (eCDF) graph was generated for the percentage of patients with change from BL in MIS for the linerixibat 40 mg BID (N = 23) and placebo (N = 36) groups. BA samples were reanalyzed using an enzymatic assay that quantifies TSBA consistent with the Phase 2a study method. Changes from BL in TSBA were analyzed using a mixed model repeated measures (MMRM) analysis.
Results: The eCDF curves showed clear separation of linerixibat 40 mg BID and placebo groups. The percentages of patients with an improvement from BL in MIS at Week 16 were greater in the linerixibat group than the placebo group for a wide range of responder threshold values. The largest differences were observed between thresholds of -3 to -2, where the cumulative percentages were >20% greater in the linerixibat group versus placebo. Linerixibat 40mg BID (n = 22) reduced mean (SD) TSBA from a BL of 18.6 μM (21.8) by -6.94 μM (17.5) after 12-weeks of treatment. A MMRM analysis showed a significant decrease of 39% (P = 0.0001) from BL and 37% (P = 0.0030) from placebo (n = 36) over the 12-week double-blind treatment period.
Conclusion: Consistent with inhibition of reuptake of BA, 40 mg BID linerixibat resulted in significant reductions in TSBA. The proportion of patients with change from BL in MIS at Week 16 was greater in the linerixibat 40 mg BID group than placebo over a range of responder threshold values. Linerixibat 40mg BID is being studied in the ongoing Phase 3 GLISTEN study (NCT04950127). Responder thresholds of 2-, 3- and 4-point improvements in MIS compared to BL are key secondary endpoints (Figure 1).



[0019] **Figure 1.** Cumulative percentage of patients with specified change from baseline in monthly itch score (MIS) at Week 16

S20

Prognostic Value of Systemic Inflammatory Markers in Patients With Pancreatic Adenocarcinoma

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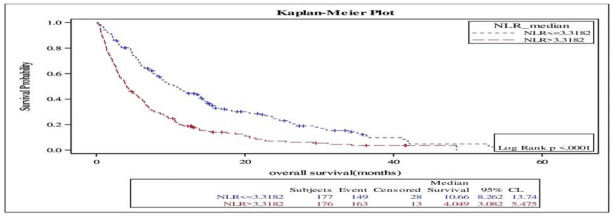
Introduction: Several studies have shown the role of inflammatory markers, especially the neutrophil-to-lymphocyte ratio (NLR), as indicators of poor prognosis in various gastrointestinal malignancies. We aimed to examine the prognostic value of NLR, among other markers, and their relationship with the presence of baseline distant metastasis in patients with pancreatic adenocarcinoma.
Methods: We retrospectively reviewed the charts of 355 patients with pancreatic cancer treated at a tertiary cancer center from 2013 to 2018. We examined the relationship between absolute eosinophilic count (AEC), absolute lymphocyte count (ALC), absolute monocytic count (AMC), absolute neutrophil count (ANC), monocyte to lymphocyte ratio (MLR), NLR, and platelet to lymphocyte ratio (PLR) with the presence distant metastases, and overall survival (OS). We used multivariable logistic regression analyses to test the association between the variables and the presence of baseline distant metastases.
Results: The median age was 60 years, and males comprised 59% of the patients. The ROC value of 3.3 was determined as the cutoff value for NLR. High NLR (NLR >3.3 μL) was significantly associated with the presence of distant metastasis at diagnosis (P-value < 0.0001, Odds Ratio (OR): 1.7, CI: 2.6–4.0). High baseline ANC (≥5500/μL), high AMC (≥600/μL), and high MLR (≥0.3) were also associated with baseline distant metastases (P-value: 0.02, 0.001, and <0.0001 respectively). Multivariable analysis showed that high NLR (P-value, 0.0003, OR 2.5 95% CI 1.5–4.1) was an independent risk factor for distant metastasis at presentation. High ANC, NLR, MLR, and PLR and low ALC were associated with poor OS (P-value: <0.0001, <0.0001, <0.0001, 0.04, and 0.01, respectively) (Figure 1).
Conclusion: High systemic inflammatory markers are associated with poor prognosis (the presence of distant metastasis) and poor OS in patients with pancreatic cancer. Simple laboratory tests such as complete blood counts can be used as markers of poor prognosis and poor OS in patients with pancreatic cancer (Table 1).

Table 1. The association between systemic inflammatory markers with the presence of distant metastases

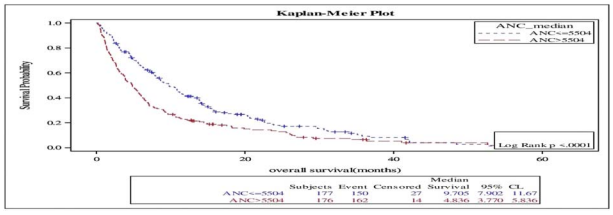
	Baseline distant metastases				
	Present	Absent	p- value	Odds Ratio	95% Confidence Interval
ANC≥5500	113 (64%)	63 (36%)	0.024	1.65	(1.0-2.5)
ANC< 5500	92 (52%)	85 (48%)			
ALC≥1680	99 (57%)	75 (43%)	0.65	0.9	(0.6-1.4)
ALC< 1680	106 (59%)	73 (41%)			
AMC≥ 600	117 (66%)	59 (34%)	0.001	2.0	(1.3-3.0)
AMC< 600	88 (50%)	89 (50%)			
AEC≥ 143	83 (62%)	51 (38%)	0.38	1.2	(0.7-2.0)
AEC< 143	76(57%)	58 (43%)			
NLR≥3.3	126 (69%)	56 (31%)	< .0001	2.6	(1.7-4.0)
NLR< 3.3	79 (46%)	92 (54%)			
MLR≥0.3	130 (68%)	62 (32%)	< .0001	2.4	(1.5-3.7)
MLR< 0.3	75 (46%)	86 (54%)			
PLR≥0.15	108 (61%)	68 (39%)	0.2	1.3	(0.85-2.0)
PLR< 0.15	97 (55%)	80 (45%)			

Figure 1



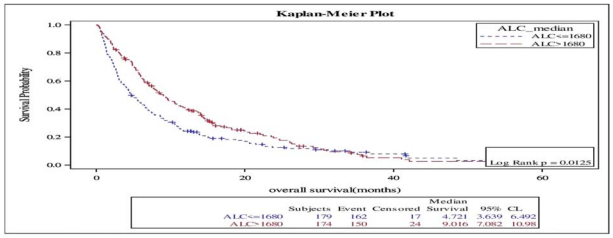
[0020] **Figure 1.** Kaplan Meier curve for overall survival with NLR

Figure 2



[0020] **Figure 2.** Kaplan Meier curve for overall survival with ANC

Figure 3



[0020] **Figure 3.** Kaplan Meier curve for overall survival with ALC

S21

Fluid Administration and Severity of Acute Pancreatitis Stratified by BISAP Score: Retrospective Cohort Study

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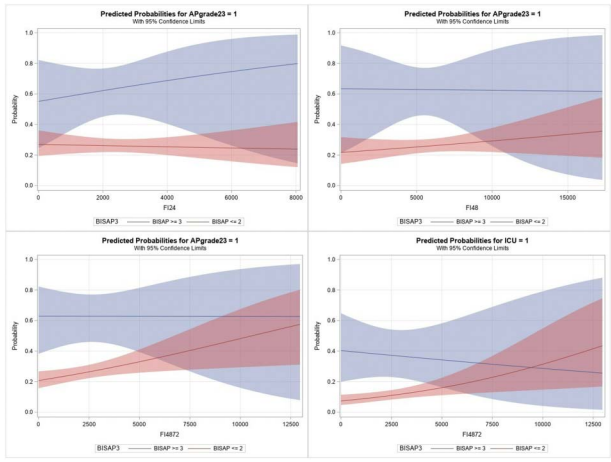
Introduction: Fluid administration is a standard of care in managing patients with acute pancreatitis (AP). There are many gaps in our knowledge. There is no study has been conducted to assess fluid administration in patients with predicted AP severity on presentation and it is effect on AP severity. In this study we aimed to evaluate the association between the fluid administration, and it is effect on AP severity defined according to Modified Atlanta Criteria in patients with AP stratified by Bedside Index for Severity in Acute Pancreatitis (BISAP) score in the first 24-48 h.
Methods: We included adult patients with AP admitted to our tertiary center between 2017 and 2019. Patients were stratified based on their BISAP scores in to low and high score groups ≤2 and ≥3, respectively. Volumes of fluid received in the first 24, 48, and 72 hours from admission were measured. Outcomes included severity of AP as defined by the modified Atlanta and ICU.
Results: 444 patients were included. 409 with BISAP of ≤2 and 35 with BISAP of ≥3. 25.9% of patients with BISAP score ≤2 and 62.9% with BISAP score ≥3 developed Moderate-Severe (M-S) AP ($P < 0.001$). The probability of M-S AP by the volume of fluid intake in the first 24 h was not different when patients were stratified by BISAP score ($P = 0.548$). A clear trend noted of lower probability of M-S AP by fluid intake given in the first 48 h ($P = 0.069$). When stratified by BISAP scores, there was no difference in the probability of developing M-S AP and fluid intake in the first 48. The probability of M-S AP by fluid intake from 48 to 72 h was higher in all patients regardless of their BISAP score ($P = 0.044$). This probability was higher in patients with lower BISAP scores ($P = 0.403$). The probability of being admitted to the ICU by fluid intake from 48 to 72 h was higher in patients with low BISAP score ($P = 0.157$) (Figure 1).
Conclusion: Higher fluid intake in the first 48h is associated with lower probability of developing moderate-severe pancreatitis (M-S AP). The overall trend of better outcomes is likely driven by patients with a higher BISAP scores. Additional fluid volume given between 48 and 72 h revealed statistically significant worse outcomes for all patients with AP. This was likely driven by patients with BISAP scores ≤2. Increased probability of being admitted to the ICU with higher fluid intake from 48 to 72 h was likely related to increased rates of fluid overload and respiratory failure in these patients with lower BISAP (Table 1).



[0021] **Figure 1.** Probability of Moderate/Severe acute pancreatitis by fluid intake adjusted by time of administration and stratified by BISAP score (interaction p = .548, p = .787, p = .403 respectively). Probability of being in the ICU by fluid intake from 48-to-72 hours stratified by BISAP score (interaction p = .157).

Table 1. AP severity by BISAP score

	AP Severity by BISAP score		
	Modified Atlanta Criteria		
BISAP score	Mild (n= 316)	Moderate/sever (n= 128)	P value
≤2 (n= 409)	74.1%	25.9%	< 0.001
≥3 (n= 35)	37.1%	62.9%	

S22

Impact of Smoking on Clinical Outcomes in Patients with Chronic Pancreatitis: A National Database Study

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[1]Case Western Reserve University, Cleveland, OH; [2]Oschner LSU Health Shreveport, Shreveport, LA; [3]University of Mississippi Medical Center, Jackson, MS; [4]University of Nebraska Medical Center, Omaha, NE; [5]University of Utah, Salt Lake City, UT; [6]University Hospitals Cleveland Medical Center, Cleveland, OH; [7]University Hospitals Clinical Research Center, Cleveland, OH; [8]McLeod Digestive Health Center, Florence, SC.

Introduction: Smoking has been shown to have a dose-dependent, adverse association with chronic pancreatitis with several, smaller studies showing worsening of disease activity, increased opioid analgesic use, and recurrent hospital admissions. We aimed to compare clinical outcomes in chronic pancreatitis patients with and without a history of smoking.
Methods: We queried clinical data from TriNetX, a large national database which aggregates healthcare data from 59 healthcare organizations in the USA comprising over 70 million patients. TriNetX uses ICD-10 codes as well as CPT codes to identify diagnoses and procedures. All patients were adults ≥18 years with chronic pancreatitis (due to any cause). Cases had a history of smoking whereas controls were never smokers. Greedy 1:1 nearest neighbor propensity score matching was utilized to match cases and controls for age, gender, race, BMI ≥25, type 2 diabetes, hypertension, chronic kidney disease, COPD, alcohol dependence, hyperlipidemia, cholelithiasis and family history of GI malignancies. Odds ratios (OR) and 95% confidence intervals (CI) were calculated for the outcomes of interest.
Results: In the unmatched group, there were 41,432 chronic pancreatitis patients who had a smoking history (cases), in comparison to 66,852 adults with chronic pancreatitis who had no prior smoking history (controls). After propensity matching, cases and controls were evenly matched in terms of age, BMI, alcohol use, and other comorbidities, though a higher number of cases were long term users of opiate analgesics (18.8% vs. 8.4%, $P < 0.001$). After propensity matching, there was a higher incidence of acute pancreatitis flare (12% vs 8%, OR = 1.52, 95% CI = 1.43–1.62). Similarly, the incidence of walled-off pancreatic necrosis, inpatient admissions, ICU admissions and adjusted 5-year all-cause mortality were all higher among cases when compared to controls (Table 1). We did not find a significant difference in the incident risk of diabetes, however the risk of pancreatic cancer was surprisingly lower among cases versus controls (Table 1).
Conclusion: Patients with chronic pancreatitis who are smokers have worse outcomes when compared to chronic pancreatitis patients who are not smokers. The reason for the lower incidence of pancreatic cancer among smokers with chronic pancreatitis is unclear and is in contrast with existing literature. Larger long term prospective studies would be necessary to clarify these associations.

Table 1. Outcomes of chronic pancreatitis in smokers vs. non-smokers in the propensity matches cohorts

Outcomes	Risk (n/N and %) or mean (SD) in Cases	Risk (n/N and %) or mean (SD) in Controls	Odds Ratio (OR) or p-value	95% Confidence Interval (CI)
Acute pancreatitis flare	2,815/23,468 (12%)	2,068/25,162 (8%)	1.52	1.43, 1.62
Walled-off pancreatic necrosis	621/30,318 (2%)	501/30,318 (1.7%)	1.24	1.10, 1.40
ICU admissions (all-cause)	3,452/30,318 (11.4%)	2,516/30,318 (8.3%)	1.37	1.30, 1.44
Mean # of ICU admissions	1.90 (3.55)	2.27 (4.54)	< 0.001	
Hospital admissions (all-cause)	16,574/30,318 (54.7%)	13,211/30,318 (43.6%)	1.56	1.51, 1.61
Mean # of inpatient admissions	6.16 (14.46)	7.16 (15.43)	< 0.001	
Diabetes mellitus (new onset)	2,184/19,404 (11.3%)	2,237/18,964 (11.8%)	0.95	0.89, 1.01
Pancreatic cancer	848/28,651 (3%)	1,144/28,717 (4%)	0.73	0.67, 0.80
5-year survival	4,275/30,184 (14%)	3,930/30,145 (13%)	1.10	1.05, 1.15
Overall mortality	4,921/30,318 (16.2%)	4,665/30,318 (15.4%)	1.06	1.02, 1.11
Median survival (days)	5006	6500	< 0.001	

S23

Long-Term Risk of Pancreatic Cancer in Patients With Prediabetes: A Systematic Review and Meta-Analysis of Prospective Studies

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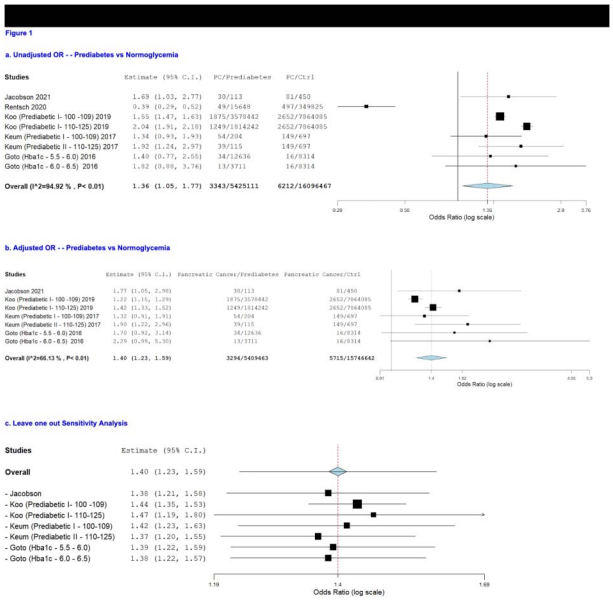
[1]Staten Island University Hospital, Staten Island, NY; [2]New York Medical College-Saint Michael's Medical Center, Newark, NJ; [3]The Brooklyn Hospital Center, Brooklyn, NY; [4]Kasturba Medical College, Manipal, Karnataka, India; [5]Kamineni Academy of Medical Sciences and Research Centre, Hyderabad, Telangana, India; [6]University of Texas at Dallas, Plano, TX; [7]Apollo Institute of Medical Sciences and Research, Hyderabad, Telangana, India; [8]University of South Dakota Sanford School of Medicine, Vermillion, SD; [9]Saint Michael's Medical Center, Newark, NJ; [10]People University of Medical and Health Sciences, Nawabshah, Sindh, Pakistan; [11]KMC Manipal, Manipal, Karnataka, India; [12]Independent Researcher, Atlanta, GA.

Introduction: Hyperglycemic states are known to have a bidirectional relationship with pancreatic disorders. The literature remains limited in discussing the role of prediabetic states on intermediate or long-term risk of having pancreatic cancer. Therefore, we conducted this updated meta-analysis to evaluate the risk of Pancreatic cancer among individuals with Prediabetes or impaired fasting glucose.
Methods: We systematically searched PubMed/Medline, EMBASE, Scopus, and Google Scholar to ascertain prospective studies describing pancreatic cancer in prediabetes between May 1, 2012 to May 1, 2022. Random-effects models were used to perform meta-analysis and subgroup analyses. I2 statistics was used to assess heterogeneity. Sensitivity analysis was done using the leave-one-out method.
Results: Five studies (1 USA, 1 UK, 1 Sweden, 1 Korea, 1 Japan) comprising 5,425,111 Prediabetics (mean age 59.3 years) and 16,096,467 normoglycemic patients with a median follow-up of 8.5 years were included in this study. A total of 3343 (0.06%) pancreatic cancer events were noted in the prediabetic group whereas 6212 (0.04%) pancreatic cancer events took place in the normoglycemic group. The unadjusted OR is 1.36 (95% CI 1.05–1.77, $P = 0.02$) (Figure 1a) and when adjusted for confounders like age, sex, etc, the overall estimated OR reported was 1.40 (1.23–1.59, $P < 0.01$) (Figure 1b). Sensitivity analysis using the leave one out method did confirm equivalent results (Figure 1c). Heterogeneity analysis for unadjusted OR had considerable heterogeneity with an overall I^2 of 94.92% with a P value <0.01 and for adjusted OR had moderate heterogeneity with an overall I^2 of 66.13% with a P value <0.01. Subgroup analysis by age showed that studies with older participants of mean/median ages 60 and above had higher odds of 1.83 (95% CI 1.28–2.62, $P< 0.01$) when compared to studies with relatively younger participants with mean/median ages <60 years which reported odds of 1.35 (95% CI 1.18–1.55, $P < 0.01$). The risk of pancreatic cancer among pre diabetics was higher in studies from Japan (OR 1.89, 95% CI 1.15–3.10, $P < 0.01$) as compared to USA (OR 1.32; 95% CI 1.13–1.53, $P < 0.01$).
Conclusion: This meta-analysis showed a 40% higher risk of pancreatic cancer in patients with prediabetes over a long-term median follow-up of over 8 years. A special screening protocol is warranted for pancreatic cancer screening which could lessen the disease burden including morbidity and mortality in high-risk patients.



[0023] **Figure 1.** Probability of Moderate/Severe acute pancreatitis by fluid intake adjusted by time of administration and stratified by BISAP score (interaction p = .548, p = .787, p = .403 respectively). Probability of being in the ICU by fluid intake from 48-to-72 hours stratified by BISAP score (interaction p = .157).

S24

Outcomes of Percutaneous vs Endoscopic Biliary Drainage in Malignant Hilar Obstruction: Analysis of a Large Electronic Health Record Dataset

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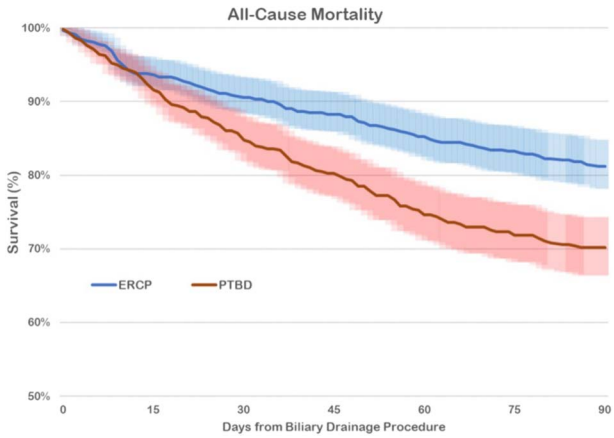
Introduction: Most patients with malignant hilar obstruction (MHO) will require biliary drainage with percutaneous transhepatic biliary drainage (PTBD) or endoscopic retrograde cholangiopancreatography (ERCP). Two recent randomized control trials (RCTs) attempted to directly compare these modalities; however, both were terminated prematurely. Post-hoc analyses for both studies revealed higher short-term all-cause mortality in the PTBD group, only one of which reached statistical significance with a wide confidence interval. It is important to better define this mortality signal to further the understanding of an area in which there remains clinical equipoise regarding the optimal biliary drainage strategy. This study aimed to evaluate 90-day all-cause mortality and other outcomes in patients undergoing ERCP and PTBD for MHO.
Methods: Data from TriNetX, an electronic health record-derived national dataset was used. Current procedural terminology (CPT) and international classification of disease (ICD) codes were used to establish a cohort of adult patients with MHO that underwent ERCP or PTBD as initial biliary drainage strategy from 2010-2020. Patients who underwent ERCP or PTBD in the preceding year before diagnosis and index drainage procedure were excluded. Propensity matching was performed based on age, sex, race, ethnicity, serum bilirubin, carcinoembryonic antigen (CEA), and carbohydrate antigen (CA) 19-9 at the time of the index drainage procedure. After matching, univariate and Kaplan-Meier analyses were performed (Figure 1).
Results: A total of 4,061 patients were identified. After matching, both cohorts contained 587 patients. All covariates were well-matched aside from higher bilirubin and CA 19-9 levels in the PTBD group. All cause 90-day mortality was lower for the ERCP cohort compared with the PTBD cohort (17.2% vs 26.7%, OR 0.6, 95% CI 0.43–0.75). At 6 months, there was no significant difference between the cohorts in rates of curative surgical resection or liver transplant (3.4% vs 5.1%, OR 0.72, 95% CI 0.41–1.25) or chemotherapy utilization (19.6% vs 16.7%, OR 1.2, 95% CI 0.87–1.70) between the PTBD and ERCP groups, respectively (Table 1).
Conclusion: Patients undergoing ERCP for MHO have significantly lower 90-day all-cause mortality compared with those undergoing PTBD. While the possibility of selection bias due to unidentified confounding variables exists, these findings are consistent with those from prematurely terminated RCTs and may help inform the design of future RCTs in this patient population.

Table 1. Post Propensity-Matching Characteristics

Covariate	PTBD	ERCP	SMD	P-Value
Age (mean ± SD)	65.4 ± 11.8	65.4 ± 11.6	0.0039	0.9463
Female sex	256 (43.6%)	273 (46.5%)	0.0582	0.3187
Race N (%)				
White	422 (71.89%)	421 (71.72%)	0.0038	0.9483
African American	85 (14.48%)	83 (14.14%)	0.0097	0.8676
Asian	24 (4.09%)	25 (4.26%)	0.0085	0.884
American Indian or Alaska Native	10 (1.70%)	10 (1.70%)	1	< 0.0001
Native Hawaiian or Other Pacific Islander	10 (1.70%)	10 (1.70%)	1	< 0.0001
Unknown Race	53 (9.03%)	52 (8.86%)	0.006	0.9185
Bilirubin (mean ± SD)	8.97 ± 8.69	6.2 ± 7.01	0.3505	< 0.0001
CA 19-9 (mean ± SD)	961 ± 1,941	658 ± 1,438	0.1774	0.0354
CEA (mean ± SD)	55.5 ± 270	34.8 ± 173	0.091	0.3518



[0024] **Figure 1.** 90-day Kaplan-Meier Mortality Curve

S25

Deep Learning for the Automatic Identification of Neoplastic Biliary Nodules in Patients With Indeterminate Biliary Stenosis During Digital Cholangioscopy

Tiago Ribeiro, MD[1], Miguel Mascarenhas, MD[1], Joao Afonso, MD[1], Filipe Vilas-Boas[1], João Ferreira, PhD[2], Pedro Pereira[1], Guilherme Macedo, MD, PhD[1].
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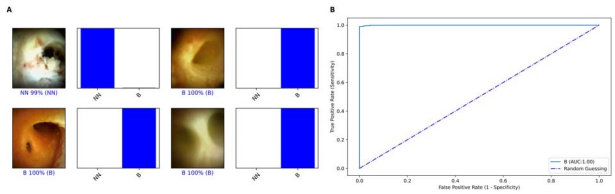
Introduction: Digital single-operator cholangioscopy (DSOC) provides direct visual exploration of the biliary tract. This tool has become essential for distinguishing benign from malignant biliary strictures. The visual aspect of these lesions is highly sensitive for the diagnosis of malignancy. Macroscopic characteristic such as tumor vessels, papillary projections, masses and nodules are common findings in patients with malignant biliary strictures. Recent endoscopic literature has demonstrated large interest in artificial intelligence (AI) systems for detection of endoscopic lesions. However, the application of these systems for automatic characterization of biliary lesions has not been explored. This study aimed to develop a convolutional neural network for automatic detection of biliary nodules in D-SOC images.
Methods: A convolutional neural network (CNN) was designed for automatic identification of biliary nodules using DSOC images. A total of 16150 frames were extracted from a pool of 85 patients undergoing DSOC (Spyglass DS II system, Boston Scientific, Marlborough, MA, USA). Each frame was classified by two endoscopists with experience in D-SOC regarding the presence or absence of nodules. Two image datasets were built for training and validation of the CNN (80% and 20% of the full image dataset, respectively). The performance of the model was measured by calculating the area under the receiving operating characteristic curve (AUC), sensitivity, specificity, positive and negative predictive values (PPV and NPV, respectively).
Results: The architecture of the network was optimized for the detection of malignant biliary nodules. The CNN reached a sensitivity of 94.0%, a specificity of 99.9%, a PPV of 98.8% and a NPV of 99.7%. The overall accuracy of the *deep learning* system was 99.6%. The AUC was 1.00.
Conclusion: The development of *deep learning* algorithms for application to DSOC may further potentiate the diagnostic capabilities of this modality. The application of these AI systems on real-time may help to guide biopsies and, thus, mitigate the current limitations of DSOC-guided biopsies, to achieve a more accurate diagnosis and timelier treatments. Our deep learning algorithm has demonstrated high performance levels for the detection of malignant nodules of the biliary tract, with high sensitivity, specificity and overall accuracy (Figure 1).



[0025] **Figure 1.** 1A - Output obtained from the application of the CNN. A blue bar represents a correct prediction. B – benign/normal findings; NN – nodules. 1B - ROC analyses of the network's performance. AUC: area under the curve; CNN: convolutional neural network; B: benign/normal findings; NN: nodules.

BILIARY/PANCREAS

S26

Comparison of Machine and Deep Learning Methods With the Mayo Clinical Risk Score in Predicting 1-Year Mortality in Primary Biliary Cholangitis Patients

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Introduction: To compare machine learning (ML) and deep learning (DL) approaches to predict survival outcome in patients at 1- year with Primary Biliary Cholangitis (PBC) and to compare with the Mayo Clinical risk score
Methods: The data is taken from a randomized trial in *PBC* patients conducted between 1974 and 1984 by the Mayo Clinic. The first 312 cases in the dataset were included out of a total of 424 PBC patients referred. The rest that contained subjects with >30% missing data, were excluded. We evaluated the following models: 1) The 5-covariate Mayo model: log(bilirubin), albumin, log(prothrombin time), edema, and age, 2) 17-covariate Cox proportional hazards model by componentwise likelihood based boosting with stepnumber 10 and penalty number 100, 3) 17-covariate Deephit: DeepHit: trains a neural network to learn the estimated joint distribution of survival time and event, while capturing the right-censored nature inherent in survival data. Analysis was done with frac 0.3, relu activation, 0.1 dropout, 100L epochs and a batch size of 32L, 4) 17-covariate Multitask logistic regression model with ranking based feature selection to predict survival using a logistic regression model and the parameters from each model are estimated simultaneously in the maximization of the joint likelihood function, 5) random survival forest with 1000 trees and 6) Support vector machine. For the ML & DL models all 17-covariates were include in the analysis. Analyses were done in RStudio, and missing values were imputed using *missRanger* package.[1] The data is split into 80 percent training and 20 precent validation. All models were 5-fold cross validated. Prediciton statistics was calculated for each model developed. Finally, the ML & DL models were compared with the 5-covariate model of the Mayo Clinic Risk score.
Results: Multitask logistic regression model showed the highest AUC (1.00) with a Harrell's C-statistic of 0.90. Cox boost and random survival forest had equal AUCs (0.87) and C-indices (0.84). But the 5-covariate model of Mayo clinic Risk score had a higher AUC when compared to cox boost, random survival forest, support vector machine or even Deep hit.
Conclusion: Machine learning methods offered limited improvement over the Mayo Clinical risk score except for the Multitask logistic regression model in predicting PBC survival outcome at 1-year (Table).

Table 1. Performance of Risk Scores from the 5-Covariate Model in comparison to the machine learning and deep learning models at 1-year

S.No.	Model specification	AUC	95% CI	Harrell's C-statistic
1.	5-covariate model of Mayo clinic Risk score	0.88	0.85-0.91	0.79
2.	Cox boost	0.87	0.84-0.90	0.84
3.	Deep hit	0.70	0.66-0.74	0.30
4.	Multitask logistic regression	1.00	1-1	0.90
5.	Random survival forest	0.87	0.84-0.90	0.84
6.	Support Vector Machine	0.56	0.52-0.61	0.43

S27

Impact of an Exocrine Pancreatic Insufficiency Order Set on Adequacy of Management of Exocrine Pancreatic Insufficiency and Related Complications

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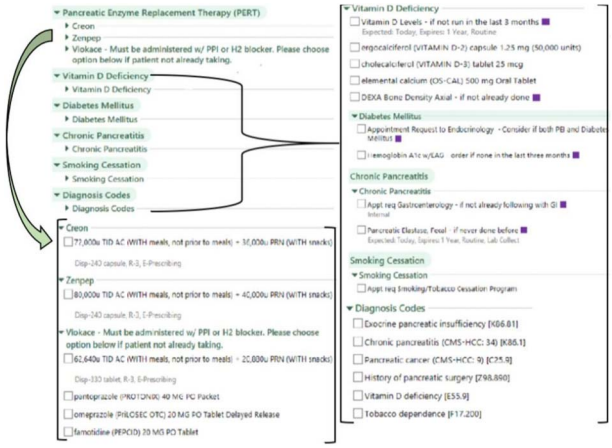
Introduction: Exocrine pancreatic insufficiency (EPI) is common in chronic pancreatitis (CP), pancreatic cancer (PDAC), and post pancreatic resection. 40-50% of CP patients, and 70-80% of PDAC patients develop EPI. Only about 1/3 of these patients are prescribed pancreatic enzyme replacement therapy (PERT), often at an inadequate dose, with evidence that this leads to increased morbidity and mortality. The aim of this study was to develop and implement an EPIC-based best practice alert (BPA) and smart set with goal of improving management of EPI.
Methods: All patients with ICD-10 codes of EPI, CP, and PDAC or CPT code for pancreatic resection, who were seen in either an outpatient or inpatient setting at a tertiary care center from Feb-2018 to Feb-2021 were included. Data was extracted from the institution's integrated electronic data repository. This study was approved by the institutional review board. Appropriate use of PERT was defined as ≥ 40,000 USP units of lipase with each meal. The initial retrospective analysis was conducted prior to implementation of the BPA and smart set from feb-2018 to feb-2020. The BPA and smart set were implemented on Feb-2020 and another retrospective analysis was done on patients from feb-2020 to feb-2021. The BPA fired if patient were already on PERT or if provider placed an order for PERT. The BPA would provide link to smart set which had appropriate PERT doses prefilled.
Results: A statistically significant increase in the proportion of patients on minimum therapeutic dose of PERT from 61.9 to 72.9% (P=< 0.001) was observed. Ordering of pancreatic elastase, A1c, vitamin D, and DEXA all increased from 20.4 to 29.9% (P< 0.001), 54.7 to 62.1% (P=0.001), 30.9 to 48.1% (P< 0.001) and 10 to 18% (P< 0.001), respectively after initiation of BPA and smart set. An increase in vitamin D supplementation from 25.5 to 33.4% (P< 0.001) and a decrease in the proportion of patients with unknown metabolic bone disease status from 86.8 to 76.8% (P< 0.001) was observed. (Figure)
Conclusion: EPI smart set usage was associated with an increase in ordering of pancreatic elastase, A1c, vitamin D, DEXA as well as an increase in the proportion of patients on minimum therapeutic dose of enzyme despite no change in the proportion of patients following with a gastroenterologist. There was no change in the proportion of patients on PERT, which was likely explained by the constraints of BPA since it did not fire if the patient was not already on PERT or if the physician was not ordering PERT (Table).



[0027] **Figure 1.** Pancreatic exocrine insufficiency smart set

Table 1. Characteristics prior to and after best practice alert and smart set implementation

Characteristic	Pre order set (n=1,464)	Post order set (n=679)	Odds Ratio	*P* value
Prescribed PERT (%)	837 (57.2)	387 (56.8)		0.925
Minimum therapeutic dose (%)	518/837 (61.9)	282/387 (72.9)	1.64	< .001
Vitamin D checked (%)	453 (30.9)	326 (48.0)	2.06	< .001
On Vitamin D supplementation (%)	373 (25.5)	225 (33.1)	1.45	< .001
Hgb A1c checked (%)	801 (54.7)	422 (62.1)	1.36	.001
DEXA ordered (%)	156 (10)	122 (18)	1.96	< .001
Metabolic bone disease status known (%)	193 (13.2)	157 (23.1)	1.99	< .001
Gastroenterologist following (%)	470 (32.1)	226 (33.3)		0.564
Pancreatic elastase checked (%)	299 (20.4)	203 (29.9)	1.67	< .001

PERT = Pancreatic enzyme replacement therapy DEXA = Dual-energy X-ray absorptiometry Hgb A1c = Glycated hemoglobin.

S28

Evaluation of a Novel EUS-Compatible Cryoablation Device for the *in situ* Destruction of Pancreatic Cancer

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Introduction: There is a pressing need for the development of new devices facilitating advanced minimally invasive approaches for the *in situ* treatment of pancreatic cancer (PaCa). To this end, a new endoscopic ultrasound (EUS) compatible cryocatheter (Frostbite) has been developed. When paired with the novel Pressurized Subcooled Nitrogen (PSN) cryoconsole, a cryogen is circulated within EUS cryocatheter delivering ultracold ablative temperatures to a targeted tissue in a closed loop manner. In this study, we evaluated cryocatheter performance for its potential use in transesophageal in situ ablation of PaCa and liver cancer.
Methods: A ~1m cryocatheter with a 13 cm long 17 gauge needle with a 3cm ablation tip, was connected to PSN and then passed through the working channel of a EUS endoscope. Performance evaluations included a 37°C ultrasound gel model, ex vivo tissue engineered PaCa model and an acute porcine study wherein 6 lesions were created within the liver (under IACUC Approval). Performance assessment included measurement of ice ball size, isotherm profile in real time and destruction area created. A single 5-minute freeze protocol was employed for all evaluations.
Results: Bench studies demonstrated the generation of a 2.4cm diameter iceball with a tip temperature of < -170°C and penetration of the -40 and -20°C isotherms to 1.5cm and 2.1cm (respectively) following 5mins. Analysis of tissue destruction using PaCa tumor model revealed the creation of a 2.1cm ablation area 1 day post freeze. The porcine study demonstrated the consistent generation of a 2cm x 3.1cm (diameter x length) ablation zone following a single 5 minute freeze protocol. The porcine study also demonstrated the ability to deliver targeted destruction of tissue in close proximity to major vasculature without damaging the blood vessel.
Conclusion: The results of this study demonstrated that the cryocatheter was able to rapidly and effectively freeze targeted tissue via a EUS approach. The results showed the device was able to consistently ablate a 2cm x 3cm area using a single 5 minute freeze protocol across all models. Analysis of the ablation efficacy revealed ~70% destruction within the overall frozen mass compared to < 40% attained with current percutaneous based cryodevices. Although further testing and refinement are needed, these studies demonstrated the potential of this new approach to provide a next-generation strategy for the treatment of PaCa.

S29

Dilated Main Pancreatic Duct Without a Visible Pancreatic Mass: Long-Term Follow-Up and Predictors of Future Pancreatic Ductal Adenocarcinoma

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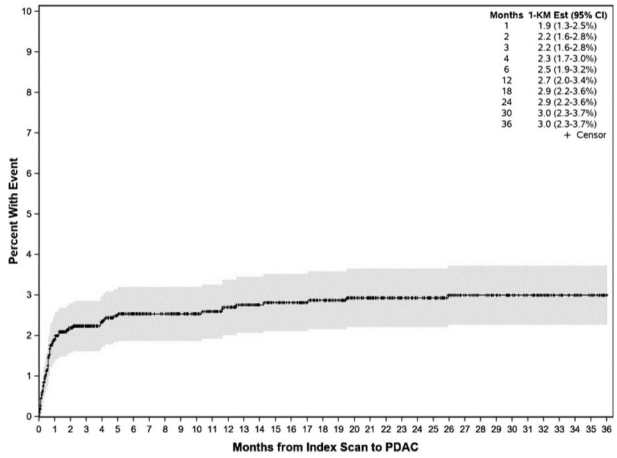
Introduction: Pancreatic ductal adenocarcinoma (PDAC) is a lethal disease frequently diagnosed at an advanced stage when therapeutic options are limited. Pancreatic imaging abnormalities that predate diagnosis of PDAC may serve as an early detection tool. Dilation of the main pancreatic duct (D-MPD) has been shown to occur up to a year before PDAC diagnosis. However, while D-MPD in the absence of a visible pancreatic mass raises concern for occult neoplasm, the natural history of D-MPD remains poorly understood. From a large cohort of patients with D-MPD and no pancreatic mass, we aimed to assess long-term outcomes and compare patients who were subsequently diagnosed with PDAC to those who did not develop PDAC.
Methods: We identified all subjects ≥18 years old at our institution with D-MPD (MPD >3mm) on either abdomen/pelvis CT or MRI scans between 1/1/2012 and 12/31/2017. Patients with prior PDAC, pancreatic surgery, definite pancreas mass or other visible cause of D-MPD were excluded after expert review by study radiologist. Pertinent clinical, demographic, and imaging data were summarized. A stratified univariate Cox proportional hazards model was utilized to evaluate risk factors for PDAC development.

Results: Among 2307 patients (baseline CT:1615, MRI: 692) who met our study criteria, 63.7% were female, median age was 71 (IQR 59.5 -80.1), and median follow-up was 1466 days (IQR 1426-1509). The 1-year and 3-year event rates for PDAC were 2.70 and 2.99 per 100-patient years respectively. Out of sixty-three (2.7%) patients who developed PDAC within 3 years of the baseline scan, the majority (58/63) were within 1 year (Figure). Factors associated with a significantly increased 3-year risk of PDAC included male gender, BMI >25, diabetes mellitus, non-O blood type, elevated serum CA 19-9, focal narrowing of MPD, equivocal pancreatic mass and suboptimal baseline scan quality (Table).
Conclusion: D-MPD may predate the diagnosis PDAC and raises concern for neoplasm. However, in our study only a small subset of D-MPD patients without an overt pancreatic mass developed PDAC on follow-up. The risk is highest in the first year with rare events after one year of PDAC-free follow-up. Risk factors identified in this study may enrich assessment and guide surveillance of patients with D-MPD. Future studies exploring machine learning tools can further enhance the identification of those at risk of future PDAC and facilitate early detection.



[0029] **Figure 1.** Kaplan-Meier curve estimate of PDAC event rates over the 3-year followup duration

Table 1. Univariate Survival Analysis (w/ strata) assessing the risk of developing PDAC within 3 years from baseline CT/MRI in 2307 patients with D-MPD

Characteristics		Reference	Hazard Ratio	95% HR CI		*p*-value
Age (years)	≥50	< 50	7.109	0.987	51.192	0.0515
Body Mass Index (kg/m^2)	≥25	< 25	1.972	1.172	3.319	0.0105
Maximum MPD diameter (mm)	≥5-< 10 ≥10	≥3-< 5 ≥3-< 5	3.773 20.081	1.987 8.455	7.167 47.691	< .0001 < .0001
CA19-9	≥35	< 35	4.819	2.678	8.670	< .0001
Gender	Male	Female	1.937	1.181	3.176	0.0088
ABO Blood type	Type Non- O	Type O	2.238	1.162	4.310	0.0161
Alcohol	Ever Use	Never Use	1.029	0.612	1.731	0.9137
Aspirin	Ever Use	Never Use	0.936	0.534	1.640	0.8177
Diabetes mellitus	Yes	No	2.596	1.554	4.338	0.0003
Chronic Pancreatitis	Yes	No	0.386	0.094	1.576	0.1847
Acute Pancreatitis	Yes	No	1.116	0.551	2.260	0.7609
Family History of PDAC	Yes	No	1.952	0.930	4.097	0.0772
Pancreas Cyst on imaging	Yes	No	1.444	0.856	2.439	0.1688
Equivocal Mass on imaging	Yes	No	12.524	7.248	21.640	< .0001
Image Quality	Suboptimal but diagnostic	Optimal	2.241	1.309	3.839	0.0033
Dilation Pattern	Segmental/Multifocal	Diffuse	1.175	0.717	1.926	0.5214
Focal Narrowing of the MPD	Present	Absent	40.800	9.732	171.038	< .0001

S30

Serial Lipase Measurement Prolongs Hospitalization in Patients With Acute Pancreatitis

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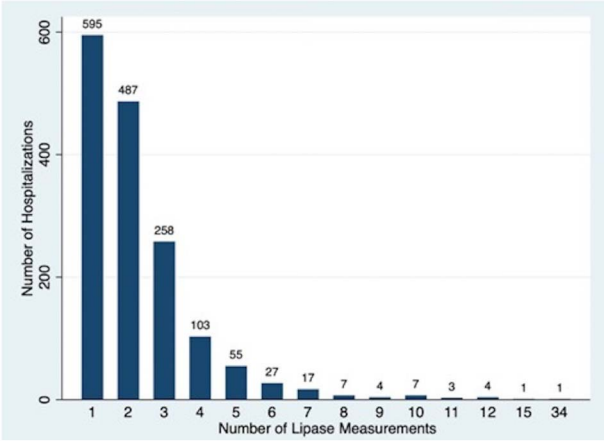
Introduction: Acute pancreatitis (AP) is one of the most common gastrointestinal indications for inpatient admission. Serial serum lipase measurement has negligible value in gauging clinical course or prognosis in AP. The American Society for Clinical Pathology recommends against routine repeat lipase measurement after an initial diagnosis of AP. However, repeat serum lipase testing is routinely performed with combined direct and indirect costs attributed to testing alone exceeding $30 million nationally. We sought to understand the frequency of serial lipase measurement, assess the impact of repeated lipase measurement on length of hospitalization, and identify factors associated with repeat lipase measurement.
Methods: We retrospectively studied adults ≥ 18 years of age with a diagnosis of AP based on ICD-10 code and/or serum lipase greater than three times the upper limit of normal at Yale New Haven Hospital from March 2019-April 2021. Logistic regression was used to assess factors associated with multiple lipase measurements during the hospitalization. A linear regression model was built to identify the effect of repeat lipase on duration of hospitalization.
Results: A total of 1,569 hospitalizations with AP ICD-10 codes were identified, and 974 of these hospitalizations (62.1%) had more than one lipase measurement (Figure). Female patients are more likely to have repeated lipase measurements during the hospitalization for AP. (P< 0.03). Patient age, race, and ethnicity were not associated with multiple lipase measurements during a hospitalization. Duration of hospitalization for AP increased with advanced age. The length of stay (LOS) was significantly longer among patients who stayed in ICU (mean 11.5 days, 95% CI 10.1-12.9) vs. non-ICU (mean 4.9 days, 95% CI 4.5-5.3). Even after controlling for ICU stay and age, the LOS was significantly longer among the patients who received multiple vs. single lipase measurements. (p < 0.01) (Table).
Conclusion: More than 2/3 of the patients hospitalized with AP had repeat lipase measurements. Serial lipase measurement in AP is associated with significantly prolonged LOS. Future studies should evaluate provider and hospitalization factors associated with serial lipase measurement, and quality improvement initiatives are needed to reduce unnecessary repeat lipase measurements.



[0030] **Figure 1.** Distribution of Lipase Measurements per Hospitalization

Table 1. Factors associated with multiple lipase measurements

Variable	Single Lipase Measurement, n = 595 Mean (Standard Deviation) or n (%)	Multiple Lipase Measurements, n = 974 Mean (Standard Deviation) or n (%)	P-value
Length of hospitalization	5.76 (6.3)	8.1 (13.4)	< **0.01**
Female gender	240 (40.3)	449 (46.1)	**0.03**
Intensive care unit hospitalization	193 (32.4)	355 (36.5)	0.11
Race			0.40
African-American	158 (26.5)	246 (25.3)	
White	357 (60.0)	573 (58.8)	
Other/Not Listed	80 (13.5)	155 (15.9)	
Ethnicity			0.14
Hispanic	95 (16.0)	165 (16.9)	
Non-Hispanic	496 (83.3)	808 (83.0)	
Not Listed	4 (0.7)	1 (0.1)	

S31

Cole Relaxation Frequency: A Parameter for Pancreatic Cancer Detection

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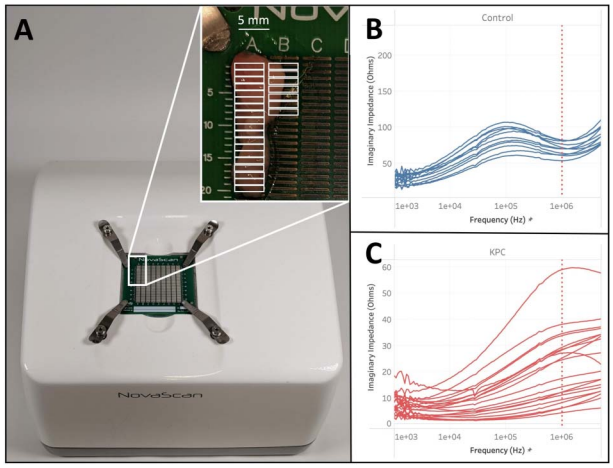
Introduction: Pancreatic cancer (PC) accounts for half a million new cases and 4.7% of the world's cancer-related deaths in 2020. It is considered one of the most lethal malignancies with a global survival rate of only 5%. Innovative imaging and endoscopic techniques are being studied to improve the accuracy of PC detection. Endoscopic ultrasound (EUS) guided tissue acquisition is currently the gold standard for sampling pancreatic masses. EUS guided fine needle biopsy has been considered superior to fine needle aspiration, but both are limited by the subjectivity of endoscopists and invasiveness of the procedure. Nonetheless, at present, there is no standard procedure for PC detection. The Cole Relaxation Frequency (CRF) – a derived electrical bioimpedance signature for cancer detection proposed by NovaScan – has been proven to quantitatively detect cancer in breast, skin, and lung tissues. The aim of this pilot study was to determine if the CRF based NovaScan technology can detect cancer in pancreatic tissues.
Methods: CRF was measured with NovaScan technology in multiple locations (Figure A) on the pancreas samples in a cohort of n=26 mice, of which 15 were KPC (model of pancreatic ductal adenocarcinoma), 2 were KC (model of pancreatic neoplasia), and 9 were wild type controls. NovaScan determined cancer presence when the CRF parameter was measured above 1 MHz (Figure:B,C). Outcomes were compared to histopathology results for each of these samples to determine the specificity and sensitivity of the NovaScan device.
Results: From histology, 12 KPC pancreases were confirmed as cancerous, 9 controls were confirmed as noncancerous, while 5 pancreases (3 KPC and 2 KC) presented with pancreatic intraepithelial neoplasia (PanIN), a precancerous condition. NovaScan technology identified 4 out of 5 PanIN samples as cancerous. Considering the entire cohort (n=26), specificity and sensitivity were 100% and 94%, respectively. If PanIN samples were excluded, specificity and sensitivity were both 100% (n=21).
Conclusion: Future development of NovaScan CRF based technology into a rapid onsite evaluation or an endoscopic device would provide an affordable means for accurate and quantitative PC detection that guides or substitutes pancreatic biopsy acquisition. Identification of malignant precursors indicates the technological capability of NovaScan's device to detect early stage PCs. Further technology development could allow for the distinction between cancerous and precancerous lesions.



[0031] **Figure 1.** (A) KPC pancreas sample placed on Novascan's electrode array for a series of spectral bioimpedance measurements. A zoom in of the electrode with a pancreas sample is also shown. White rectangles indicate multiple locations measured across the sample. Spectral impedance scans for a control mouse (B) and a KPC mouse (C). The examination of CRF peak properties was used for cancer identification in pancreas samples. For the control mouse the CRF peaks fall below the threshold of 1 MHz, determining no cancer. For the KPC mouse several scans have CRF peaks above the threshold of 1 MHz, determining cancer presence.

S32

Females Develop Less Severe Acute Pancreatitis: A Multivariate Analysis Controlled for Etiology and Core Factors

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Introduction: In gastrointestinal diseases such as alcohol-associated liver disease, gender has been shown to be an independent risk factor for severity due to differences in first-pass metabolism of alcohol, Kupffer cell activation, and hormones. We aim to describe the impact of gender on the clinical course of acute pancreatitis (AP).
Methods: We prospectively characterized a cohort of unique patients presenting between January 2015 to March 2021 with AP. Our primary outcome was the development of moderately severe or severe pancreatitis (as defined by the Revised Atlanta Classification) and our primary predictor was gender. Linear and multivariate logistic regression analyses were performed, controlling for both patient (age, ethnicity, BMI) and clinical (etiology of AP, comorbidities, fluids administered in first 48 hours) factors.
Results: 1473 patients presented with AP. Of this cohort, 45% were female, the mean (±SD) age was 45 (±16), and 82% were Hispanic. 1176 (79.8%) patients presented with mild AP and 297 (20.2%) patients presented with moderately severe or severe AP. Age and amount of fluids given in the initial 48 hours did not differ between genders, however, females had a higher mean BMI (29.43 vs 28.22; P=0.003). Males presented with alcohol AP more frequently (40.7% vs 6.1%; P < 0.001), whereas females presented with gallstone AP more frequently (59.2% vs 28.6%; P < 0.001). Other causes of AP were equally distributed between genders (P=0.120). When controlled for etiology as well as age, ethnicity, BMI, comorbidities, and fluid administration, females were less likely to present with moderately severe or severe pancreatitis (OR 0.606; 95% CI 0.435-0.844). They were also less likely to develop necrotizing AP, pancreatic pseudocysts, organ failure, and require ICU level of care (Table).
Conclusion: Females are less likely to present with moderately severe or severe AP compared to males when controlled for etiology and core factors. This may be due to sex-specific steroid stress responses or the effects of estrogen on the pancreas. Further studies are needed to confirm these findings and elucidate the mechanism.

Table 1. Covariates and outcomes

	All patients N=1473	Females N=669	Males N=804	
Covariates				
Age, mean (±SD)	45 (±16)	45 (±17)	44 (±14)	P=0.308
BMI, mean (±SD)	28.76 (±7.14)	29.43 (±7.45)	28.22 (±6.84)	P=0.003
≥ 6L Fluids in 48H, n (%)	724 (49.2%)	320 (47.8%)	404 (50.2%)	P=0.356
Etiology, n (%)				
Alcohol	368 (25.0%)	41 (6.1%)	327 (40.7%)	P < 0.001
Gallstone	626 (42.5%)	396 (59.2%)	230 (28.6%)	P < 0.001
Other	479 (32.5%)	232 (34.7%)	247 (30.7%)	P=0.120
Outcomes	N (%)	N (%)	N (%)	OR (95% CI)
Mild Pancreatitis	1176 (79.8%)	568 (84.9%)	608 (76.6%)	1.650 (1.185-2.298)
Moderately Severe or Severe Pancreatitis	297 (20.2%)	101 (15.1%)	196 (24.4%)	0.606 (0.435-0.844)
Organ Failure	159 (10.8%)	56 (8.4%)	103 (12.8%)	0.615 (0.397-0.954)
Pancreatic Pseudocyst	35 (2.4%)	8 (1.2%)	27 (3.4%)	0.382 (0.155-0.940)
Necrotizing Pancreatitis	59 (4.0%)	9 (1.3%)	50 (6.2%)	0.244 (0.112-0.531)
Length of Stay < 48 Hours	416 (28.2%)	153 (22.9%)	263 (32.7%)	0.713 (0.507-1.002)
ICU Stay	254 (17.2%)	79 (11.8%)	175 (21.8%)	0.415 (0.290-0.594)
30-Day Readmission	198 (13.4%)	81 (12.1%)	117 (14.6%)	0.994 (0.692-1.440)
Mortality	27 (1.8%)	11 (1.6%)	16 (2.0%)	1.067 (0.405-2.81048

S33

Endoscopic Ultrasound Biopsy for Molecular Analysis in Pancreatic Cancer: Findings From a Large Academic Medical Center

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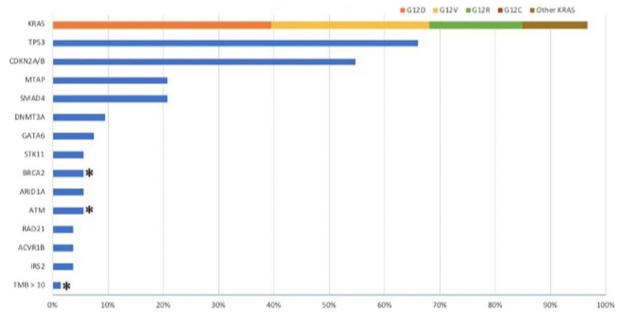
Introduction: Pancreatic cancer continues to carry a dismal prognosis due to the high failure rates of conventional first line treatments. There is growing interest in the molecular profiling of tumors to guide early initiation of targeted therapies. Nearly all patients undergo endoscopic ultrasound (EUS) fine needle aspiration or biopsy as the initial diagnostic procedure. Therefore, we sought to assess the yield of EUS biopsies in obtaining samples for molecular profiling of pancreatic tumors and investigated the endoscopic factors associated with successful EUS sampling.
Methods: We performed a search for all EUS-guided needle biopsies done for the indication of suspected pancreatic mass on imaging between January 2017 and January 2022. We then limited our cases to those diagnosed with pancreatic adenocarcinoma and had EUS samples sent for molecular profiling. Molecular profiling was done with next-generation sequencing with either a targeted panel of 648 genes or 324 genes. Differences in tumor size, number of needle passes, and needle gauge size between the successful sampling and non-successful sampling groups were determined by Mann-Whitney U Test using SPSS Statistics.
Results: We identified 309 consecutive cases where the diagnosis of pancreatic adenocarcinoma was established by EUS. Fifty-nine EUS biopsies were sent for molecular profiling and of these, fifty-three were sufficient for molecular testing (89.5% success rate). No procedural factors were significantly associated with successful sampling though we observed larger mean tumor sizes (31.3 vs 28 mm) and greater mean number of needle passes (3.4 vs 2.7 mean passes) in the successful sampling group. In Figure, we show the most commonly identified mutations and identify those that at the time had potential clinical impact on therapies. The yield of actionable mutations was 14% in the 53 patients who were successfully tested. (Figure)
Conclusion: Our results support that yield of somatic mutation testing is high from standard of care EUS biopsies and no obvious procedural factors were associated with failure of testing. We found that 14% of patients had actionable mutations. As the number of available targeted therapies improve, we expect the impact of this highly technically successful approach to grow (Table).



[0033] **Figure 1.** Percentage frequency the most common genes were mutated or tumor mutation burden was greater than 10 muts/mb in study cohort. Asterix identifies actionable mutations.

Table 1. Interventions by gene mutation derived from molecular testing of endoscopic ultrasound samples

Gene	Actionable Interventions	Potential Interventions	No Current Interventions	Frequency (%)
KRAS		MEK/autophagy inhibitor combination		51 (96%)
G12D				21
G12V				15
G12R				9
G12C	Sotorasib	SHP2 inhibitors		0
TP53		WEE1 inhibitors		35 (66%)
CDKN2A/B		CDK4/6 inhibitors		29 (55%)
MTAP		MAT2 inhibitors		11 (21%)
SMAD4			x	11 (21%)
DNMT3A			x	5 (9%)
GATA6			x	4 (8%)
STK11	UGH	mTOR inhibitors		3 (6%)
ATM	Platinum chemotherapy; PARP inhibitors			3 (6%)
ARID1A		ATR inhibitors		3 (6%)
BRCA2	Platinum chemotherapy; PARP inhibitors			3 (6%)
RAD21			x	2 (4%)
ACVR1B			x	2 (4%)
IRS2		IGF1R inhibitors		2 (4%)
TMB > 10	PD-1/PDL-1/CTLA-4 inhibitors			1 (2%)

S34

Impact of Smoking Cessation Attempts in Reducing Low Bone Density Disease in Chronic Pancreatitis

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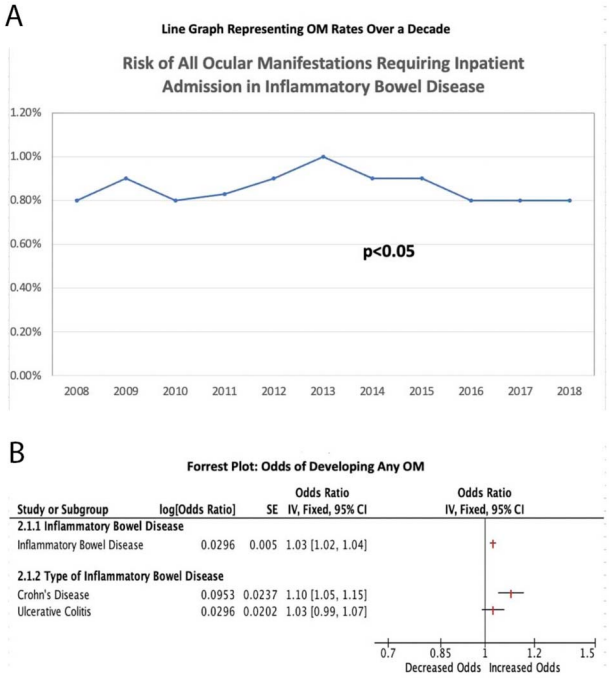
Introduction: Patients with chronic pancreatitis are at increased risk of low bone density, and tobacco smoking is an independent risk factor for the development of low bone density in this patient population. We aimed to determine if smoking cessation attempts would affect the risk of low bone density in chronic pancreatitis patients.
Methods: We conducted a retrospective cohort study utilizing TriNetX, which is a global federated health research network providing access to electronic medical records across 59 large healthcare organizations. Utilizing ICD-10 codes, we identified chronic pancreatitis patients with tobacco smoking history and divided them into two cohorts of smoking cessation and no smoking cessation. Smoking cessation attempt was defined as a prescription for varenicline/bupropion or a smoking cessation counseling visit. Propensity score matching was performed for demographics and medications (pancreatic

enzyme replacement, calcium, and Vitamin D). Outcomes included osteoporosis, malnutrition, calcium deficiency, Vitamin D deficiency, and mortality. For continuous data, we performed independent t-tests. For categorical data, we performed chi-square tests.

Results: Of chronic pancreatitis patients with tobacco smoking history, we identified 9056 patients with smoking cessation attempts (Cohort 1) and 56,662 patients with no smoking cessation attempts (Cohort 2). After propensity score matching, 9,000 patients were accounted for in each group (Figure). Reviewing one-year outcomes of these patients after propensity score matching, no difference in osteoporosis (OR 1.031, 95% CI 0.906-1.173) and calcium deficiency (OR 1, 95% CI 0.416-2.404) was noted between each cohort. 1,149 patients in Cohort 1 Vitamin D deficiency compared to 849 patients in Cohort 2 (OR 1.405, 95% CI 1.279-1.543). Malnutrition was seen in 1,012 patients in Cohort 1 and 1,672 patients in Cohort 2 (OR 0.555, 95% CI 0.510-0.604). One-year mortality noted 486 deaths in Cohort 1 and 772 deaths in Cohort 2 (OR 0.608, 0.541-0.684) (Table 1).

Conclusion: Smoking cessation attempts in chronic pancreatitis patients with smoking histories are associated with one-year decreased risk of mortality and malnutrition. Further attempts to provide smoking cessation options to these patients for improved outcomes.



[0234] **Figure 1.** Propensity Score Matching for Smoking Cessation and No Smoking Cessation Cohorts.

Table 1. **One-Year Outcomes After Propensity Score Matching**

Outcome	Smoking Cessation (N=9,000)	No Smoking Cessation (N=9,000)	Odds Ratio	95% Confidence Interval
Osteoporosis	492 (5.5%)	478 (5.3%)	1.031	0.906-1.173
Calcium Deficiency	10 (0.1%)	10 (0.1%)	1	0.416-2.404
Vitamin D Deficiency	1149 (12.8%)	849 (9.4%)	**1.405**	**1.279-1.543**
Malnutrition	1012 (11.2%)	1672 (18.6%)	**0.555**	**0.510-0.604**
Mortality	486 (5.4%)	772 (8.6%)	**0.608**	**0.541-0.684**

S35

Outcomes of Endoscopic Stent vs Balloon Dilation in Primary Sclerosing Cholangitis-Related Dominant Strictures: A National Database Study

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Introduction: Primary sclerosing cholangitis (PSC) is a chronic cholestatic disorder characterized by multi-focal bile duct strictures. The presence of a dominant stricture in PSC is considered to be a poor prognostic factor. The optimal endoscopic strategy (stent versus balloon dilation) is unclear due to paucity of available data and small number of patients in existing studies.

Methods: We used a large national database (TriNetX, LLC.), which aggregates data from 59 healthcare organizations comprising over 70 million patients using ICD-10 and CPT codes to construct a case-control study. All patients were adults ≥ 18 years with PSC with a dominant stricture. Cases underwent endoscopic stent placement, whereas controls underwent endoscopic balloon dilation. We propensity matched the groups for age, male sex, white race, BMI ≥ 30, diabetes, ulcerative colitis, smoking, alcohol use, history of appendectomy and hypertriglyceridemia. Cases and controls were compared for 30-day risk of procedure related cholangitis, perforation, bleeding and 30-day hospital readmission rates. We also compared 14-day risk of post-ERCP pancreatitis, and risk of 1-year and 3-year mortality between cases and controls.

Results: In the matched cohort analysis, patients who received a stent had a higher 30-day risk of procedure related cholangitis when compared to those who only underwent balloon dilation (7.7% vs. 2.8%, OR = 2.8, 95% CI = 1.2, 6.9). There were no significant differences between the two groups with respect to 30-day hospital readmission rates (26% vs. 22%, OR = 1.2, 95% CI = 0.9, 1.8). Similarly, there were no differences between the stent and the balloon dilation groups in terms of 14-day post procedural pancreatitis (6.8% vs. 4.5%, OR = 1.5, 95% CI = 0.7, 3.1). Patients in the stent group had a higher risk of 1-year mortality (8% vs. 3%, OR = 2.8, 95% CI = 1.3, 5.9) and 3-year mortality (13% vs. 6%, OR = 2.3, 95% CI = 1.3, 4.0) as compared to balloon dilation (Table). Outcome data on post-procedural bleeding, perforation and 30-day mortality were too small to be able to detect differences between cases and controls.

Conclusion: Patients with PSC related dominant stricture who had stent placement seem to have a higher risk of immediate (within 30 days) procedure related acute cholangitis, as well as higher 1-year and 3-year mortality. Larger prospective randomized clinical trials are necessary to further clarify the differences in outcomes between these two procedures (Table).

Table 1. Outcomes of Endoscopic stent vs Balloon dilation in Primary Sclerosing Cholangitis with dominant strictures in the propensity matched cohorts

	Risk in cases (PSC + dominant stricture + stent)	Risk in controls (PSC + dominant stricture + balloon)	Odds Ratio (OR)	95% Confidence Interval (CI)
Cholangitis within 30 days	18/232 (7.7%)	7/247 (2.8%)	2.8	1.2, 6.9
All cause hospital re-admissions within 30 days	89/336 (26%)	74/336 (22%)	1.2	0.9, 1.8
Post-ERCP pancreatitis within 14 days	20/293 (6.8%)	14/313 (4.5%)	1.5	0.7, 3.1
1-year mortality	27/336 (8%)	10/336 (3%)	2.8	1.3, 5.9
3-year mortality	45/336 (13.4%)	21/336 (6.3%)	2.3	1.3, 4.0

S36

Early Cholecystectomy Improves Clinical Outcomes in Acute Biliary Pancreatitis Patients Across All Age Groups: A Multicenter Retrospective Study

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Introduction: Gall stone disease is the most common etiology of acute pancreatitis accounting for 40-70% of all cases. Cholecystectomy is recommended early in patients with acute biliary pancreatitis (ABP) to prevent a recurrence. Elderly patients have a higher incidence of the disease and are at risk of worse outcomes, however, this cohort has not been studied adequately. Thus, we aim to study the outcomes of patients who undergo early cholecystectomy versus those who do not.
Methods: This is a retrospective, multicenter cohort study utilizing TriNetX (Cambridge, MA), "a global federated health research network". Adult patients with ABP were identified using appropriate ICD-10 CM codes and divided into two groups, younger than 70 and those who are 70 and older. Within each group, a cohort of patients who had an early cholecystectomy (within 2 weeks) was compared with patients who did not have early cholecystectomy. The primary outcomes were 90-day mortality and 90-day re-hospitalization.
Results: A total of 30,062 adults (9,728 elderly) with ABP were identified and 13,574 (45%) of them underwent early cholecystectomy. Among younger patients (< 70 years), approximately 49% underwent early cholecystectomy, while 36% of the elderly patients underwent early cholecystectomy. After propensity score matching, younger patients (< 70 years) who had early cholecystectomy experienced lower 90-day (RR 0.16, p< 0.001) mortality and 90-day rehospitalization (18.5% Vs 34.2%, RR 0.54, p< 0.001) (Table). Patients who underwent early cholecystectomy were at a lower risk of acute cholangitis (RR 0.60, p< 0.001). The elderly subgroup had similar outcomes with improved 90-day mortality (1.3% Vs 4.9%, RR 0.26, p< 0.001) and 90-day rehospitalization (29% Vs 39%, RR 0.76, p< 0.001).
Conclusion: Early cholecystectomy significantly reduces short-term mortality and hospital readmission in younger and older patients with acute biliary pancreatitis. Our findings emphasize the importance of performing cholecystectomy during the same hospitalization for acute biliary pancreatitis in all patients, including older.

Table 1. Clinical Outcomes in Younger (< 70 years) Patients with Acute Biliary Pancreatitis

Outcomes	Early Cholecystectomy	No early Cholecystectomy	Risk ratio (Risk difference)	P value	Odds ratio	95% confidence interval
30-day mortality	0.12% (10)	1.12% (92)	0.11 (-1%)	< 0.001	0.11	0.06-0.21
90-day mortality	0.33% (27)	2.1% (173)	0.16 (-1.77	< 0.001	0.15	0.10-0.23
30-day readmission	15.88% (1,302)	28.30% (2,320)	0.56 (-12.4%)	< 0.001	0.48	0.44-0.52
90-day readmission	18.5% (1,530)	34.24% (2,831)	0.54 (-15.7%)	< 0.001	0.44	0.41-0.47
Acute kidney injury	1.56% (128)	3.67% (301)	0.42 (-2.1%)	< 0.001	0.41	0.34-0.51
Necrotizing pancreatitis	0.75% (62)	1.66% (136)	0.45 (-0.8%)	< 0.001	0.45	0.33-0.61
Acute cholangitis§	9.12% (748)	15.07% (1,236)	0.60 (-5.9%)	< 0.001	0.57	0.51-0.62
ERCP†	4.06% (353)	7.83% (712)	0.52 (-3.7%)	< 0.001	0.50	(0.44-0.57)
Endoscopic sphincterotomy¶	2.78% (230)	6.12% (506)	0.45 (-3.3%)	< 0.001	0.44	0.37-0.51
Biliary leak/perforation	0.13% (11)	0.12% (10)	1.10 (0.01%)	0.82	1.10	0.46-2.59

§Within 90 days of acute pancreatitis.
†All ERCP within a month of acute pancreatitis.
¶Within a week of acute pancreatitis. ERCP Endoscopic retrograde cholangiopancreatography.

S37

The Impact of Early Detection of Malignant Biliary Obstruction by Endoscopic Ultrasound on Clinical Outcomes After Surgical Resection

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Introduction: Computerized tomography (CT) scan and endoscopic ultrasound (EUS) are the main diagnostic tools for detecting periampullary cancers. EUS allows the detection of small tumors missed by CT scan in patients presenting with bile duct obstruction. However, the impact of EUS in diagnosing small tumors on clinical outcomes is yet to be explored. This study aimed to compare the tumor recurrence rate and survival benefit of patients following pancreaticoduodenectomy (PD) for periampullary cancers detected by abdominal CT vs. EUS after negative CT.
Methods: A retrospective review of the EUS and surgery database from 2009 to 2021. The patients who underwent PD for periampullary cancer were recruited. The patients were divided into CT-guided surgery group (CT group) and EUS-guided surgery group (EUS group). The clinical outcomes between the 2 groups were compared.
Results: 429 patients were enrolled. 372 patients were diagnosed by CT scan, and EUS diagnosed 57 patients after CT scans failed to detect the cause of bile duct obstruction. The mean age was 64.7+ 11.9 years. The common presentations included jaundice (79.0 %) and weight loss (66.9%). Pancreatic adenocarcinoma (PDAC) was the most common cancer (43.6%), followed by ampullary cancer (32.4%), distal cholangiocarcinoma (14.2%), and duodenal cancer (9.8%). There were no differences in patient demographics between the two groups regarding age, gender, and underlying diseases. Patients in the CT group had higher serum bilirubin (p=0.025), higher serum alkaline phosphatase (p=0.047), lower serum hematocrit (p=0.043), and larger CBD diameter (p=0.005) compared to the EUS group. The T and M staging were not different between the two groups; however, the percentage of patients with nodal metastasis was higher in the CT group (p=0.014). Multivariate analysis showed a trend towards higher recurrence and 5-year mortality rate in the CT group with odds ratios (OR) of 1.33 (95% CI, 0.82-2.13) and 1.43 (95% CI, 0.84-2.42), respectively. Subgroup analysis of PDAC and cholangiocarcinoma demonstrated a significantly higher mortality rate of patients in the CT group with an OR of 2.82 (95% CI, 1.14-6.99, p=0.026) and a trend toward a higher recurrence rate after surgery with the OR of 1.45 (95% CI, 0.80-2.66) compared to the EUS group.
Conclusion: EUS added a diagnostic value in the early detection of periampullary cancers, which may lead to a survival benefit, especially in PDAC and cholangiocarcinoma.

S38

Impact of Diabetes Mellitus on Acute Pancreatitis Outcomes in a Large Prospective Observational Cohort

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Introduction: Acute pancreatitis (AP) is a leading indication for hospital admission. The relationship between AP and diabetes mellitus (DM) is becoming increasingly recognized. Many patients with DM have comorbid conditions (ex. heart and renal disease) that may increase the risk of severe pancreatitis or pancreatitis outcomes. We aim to identify the impact of DM on acute pancreatitis hospital outcomes including organ failure, readmission, and death.
Methods: We identified patients hospitalized for acute pancreatitis between January 2015 and March 2021 using our prospective observational cohort. We included patients who had an episode of acute pancreatitis with or without pre-existing DM. Outcomes of interest included severity of pancreatitis, necessity of an intensive care unit (ICU) stay, organ failure, readmission, and death. Information on demographics, medical history, biochemical data, severity of the pancreatitis episode (Revised Atlanta Classification), and imaging were obtained for analysis. Logistic regression was used for analysis.
Results: A total of 1340 unique patients were included in the analysis. 313 (23.4%) of the patients had pre-existing DM while 1027 (76.6%) did not. The overall cohort was 46.8% female and 81.3% Hispanic. The mean age in the patients with pre-existing DM was 53 (± 14) years old, while the non-diabetic cohort was 43 (± 15) years old. Patients with diabetes mellitus were significantly more likely to have moderate-to-severe pancreatitis [OR 1.52 (1.11-2.09)]. With regards to hospital outcomes, the diabetes cohort were more likely to have an intensive care unit (ICU) stay [2.26 (1.65-3.11)], and necessity of ICU interventions such as vasopressors [5.06 (2.25-11.38)], intubation [2.21 (1.13-4.35)], and renal replacement therapy (RRT) [4.77 (1.92-11.88)]. No significant difference was seen in readmission within 30 days [0.79 (0.51-1.23)] but patients with diabetes were more likely to have hospitalization result in death [3.49 (1.41-8.60)].
Conclusion: Within our acute pancreatitis population, patients with diabetes mellitus were more likely to have both local and systemic complications as well as necessity of more invasive hospital interventions such as intubation and vasopressors compared to their non-diabetic counterparts. These results emphasize the importance of adequately controlling patients' underlying diabetes to minimize risk of hospital complications (Table).

Table 1. Comparison of hospitalization outcomes for acute pancreatitis in diabetics vs. non-diabetics

	No Diabetes n=1027, (76.6%)	Diabetes n=313, (23.4%)	P value	OR (95% CI)
Pancreatitis Severity				
Mild	857 (83.4%)	238 (76.0%)	0.003	
Mod/Severe	170 (16.6%)	75 (24.0%)	0.003	**1.52 (1.11-2.09)**
Organ Failure (any)	104 (10.1%)	54 (17.3%)	0.001	**1.69 (1.16-2.44)**
Respiratory Failure	81 (7.9%)	34 (10.9%)	0.100	
Circulatory Failure	20 (2.0%)	15 (4.8%)	0.006	
Renal Failure	37 (3.6%)	33 (10.5%)	< 0.0001	
ICU Stay (yes/no)	138 (13.4%)	82 (26.2%)	< 0.0001	**2.26 (1.65-3.11)**
ICU LOHS (days)	0.8	1.5	0.031	
Intubation	23 (2.2%)	16 (5.1%)	0.008	**2.21 (1.13-4.35)**
Vasopressors	11 (1.1%)	17 (5.4%)	< 0.0001	**5.06 (2.25-11.38)**
RRT	9 (0.9%)	12 (3.8%)	< 0.0001	**4.77 (1.92-11.88)**
Readmission within 30 days	116 (11.3%)	29 (9.3%)	0.312	0.79 (0.51-1.23)
Death	10 (1.0%)	12 (3.8%)	< 0.0001	**3.49 (1.41-8.60)**

Footnote: -OR=odds ratio, from multivariate analysis controlling for age, gender, and comorbidities such as heart failure, chronic kidney disease, and cirrhosis. ICU= intensive care unit; RRT = renal replacement therapy.

S39

A Systematic Review of Cannabis-Induced Acute Pancreatitis: Is "HASH"ing Out the New Increasingly Common Culprit of Pancreatitis?

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Introduction: Cannabis is the most frequently used illicit drug in the world. Grant et al. first reported cannabis-induced acute pancreatitis (AP) in 2004. However, after the legal or decriminalized status of cannabis in almost half of the United States, AP could be increasingly recognized in clinical practice. This systematic review aims to increase community awareness by summarizing available clinical evidence on cannabis-related AP.
Methods: A systematic search of MEDLINE, Embase, Scopus, and Cochrane was conducted for English-only articles, published between inception and June 15, 2022. Abstracts from major gastroenterology conferences and bibliography lists of relevant studies were also screened. Search terms were "cannabis" and "acute pancreatitis," with all available permutations. The diagnosis of AP was made by fulfilling 2 of the 3 criteria of the Revised Atlanta Classification. Cannabis-induced AP was designated by active cannabis use based on physician or patient reports, or urine drug testing, and the exclusion of alternative causes of AP. Three authors reviewed each paper for eligibility. The search resulted in 298 hits, but 34 articles fulfilled the inclusion criteria, dating from 2004 to 2022.
Results: A total of 37 patients with cannabis-induced AP were identified. The mean age of patients was 29.66 ± 10.24 years (range: 16-48 years), with 86% of patients being below age 35. In terms of gender distribution, 89% of cases were reported in males. A temporal relationship between cannabis use and AP onset or cannabis cessation and AP symptom resolution was noted in 64% of patients. Most patients had no comorbidities, and alternative causes were meticulously excluded. Cannabis relapse and recurrent AP were found in 36% of cases. In 32% of patients, cannabis cessation resulted in no recurrent AP.
Conclusion: We reiterate that the cannabis use can be an underdiagnosed etiology for AP. The pathogenesis of this causal relationship remains controversial. Notably, cannabis-related AP has often been encountered in young patients. This trend has clear clinical implications as cannabis status changes may exorbitantly increase the occurrence of AP in coming years. Therefore, clinicians should remain cognizant of AP in cannabis users, especially young patients presenting with abdominal pain. Toxicology screening may aid in early diagnosis. Naranjo score can help in causality assessment (Table). Permanent cannabis cessation is imperative in avoiding recurrent AP.

Table 1. Naranjo assessment scale depicting a score of 10 in a case of cannabis-induced pancreatitis (definitive)

Naranjo Adverse Drug Reaction Probability Scale				
Questions	**Yes**	**No**	**Do not know**	**Patient's score**
1. Are there previous *conclusive* reports on this reaction?	+1	0	0	+1
2. Did the adverse event appear after the suspected drug was administered?	+2	-1	0	+2
3. Did the adverse reaction improve when the drug was discontinued or a *specific* antagonist was administered?	+1	0	0	+1

Table 1. (*continued*)

Naranjo Adverse Drug Reaction Probability Scale				
Questions	**Yes**	**No**	**Do not know**	**Patient's score**
4. Did the adverse event reappear when the drug was re-administered?	+2	-1	0	+2
5. Are there alternative causes (other than the drug) that could on their own have caused the reaction?	-1	+2	0	+2
6. Did the reaction reappear when a placebo was given?	-1	+1	0	
7. Was the drug detected in blood (or other fluids) in concentrators known to be toxic?	+1	0	0	+1
8. Was the reaction more severe when the dose was increased or less severe when the dose was decreased?	+1	0	0	+1
9. Did the patient have a similar reaction to the same or similar drugs in *any* previous exposure?	+1	0	0	
10. Was the adverse event confirmed by any objective evidence?	+1	0	0	
Total score				10
Note: A score of < 1 is doubtful, 1–4 possible, 5–8 probable, and >9 is definitive for adverse drug reaction.				

S40

Presentation and Management of Fluid Status for Cirrhotic Patients With Acute Pancreatitis in a Prospective Cohort Study

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Introduction: Research regarding cirrhotic patients with acute pancreatitis has been limited and inconclusive. A vital topic is the fluid management volume of fluids given to this population given their sensitivity to hemodynamic changes.
Methods: We analyzed all unique patients presenting with acute pancreatitis to LAC+USC Medical Center captured in a prospective cohort. Our main predictor was the pre-existence of cirrhosis based on clinical exam, laboratory, diagnostic findings. Our primary outcome was development of moderately severe and severe pancreatitis, and our secondary outcomes were volume overload, organ failure, ICU admission, mortality, and new volume overload. Linear and logistic regression analyses were performed, controlling for both patient (age, sex, ethnicity) and clinical (etiology of pancreatitis, comorbidities) factors.
Results: Between January 2015 and March 2021, 1,433 patients presented to LAC+USC Medical Center with acute pancreatitis. Of those patients, 87 (6.1%) patients had a diagnosis of cirrhosis. Patients with cirrhosis received less fluids in the first 24 hours compared to patients without cirrhosis (mean volume 2351.02 (1947.77-1755.24) ml vs 3512.33 (3398.03-3626.63) ml, p< .01). In multivariate analysis, cirrhotic patients more likely presented with volume overload (OR 18.52 95% CI 9.17-37.0). There were similar rates of local complications of pancreatitis and ICU admission. However cirrhotic patients were more likely to have new acute kidney injury (OR 2.31 95% CI 1.34-3.98), severe pancreatitis (OR 2.75, 95% CI 1.37-5.56), and mortality (OR 8.33 95% CI (3.36-20.83) during inpatient stay.
Conclusion: Patients with cirrhosis were more likely to present volume overloaded and received less fluids than patients without. Despite the presentation of their volume status, patients with severe liver disease were more likely to have AKI, severe pancreatitis, and mortality. Our study highlights the importance of further research regarding fluid management in this hemodynamically complex population (Table).

Table 1.

Clinical Outcomes in Patients with Cirrhosis	
Outcome	**OR (95% CI)**
Volume Overload	18.52 (9.17-37)
New AKI	2.31 (1.34-3.98)
Severe Pancreatitis	2.75 (1.37-5.56)
Mortality	8.33 95(3.36-20.83)
Dialysis Initiation	2.46 (0.70-8.62)
ICU Admission	1.33 (0.74-2.38)
Readmission	1.30 (0.66-2.56)

S41

Serum Lactate Dehydrogenase Levels: The Grim Reaper Sign in Acute Pancreatitis?

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Introduction: Acute pancreatitis (AP) is one of the most common gastrointestinal presentations to the emergency department. Current scoring systems in place for predicting the severity and mortality of AP include Ranson criteria, the Acute Physiology And Chronic Health Evaluation II (APACHE II), and the Bedside Index of Severity in Acute Pancreatitis (BISAP) score. However, these scoring systems have their limitations and variable utility. Lactate dehydrogenase (LDH) is an enzyme found in almost all body tissues that is released during tissue damage and can be used as a marker of an organ injury. The purpose of this study was to investigate whether specific LDH values can predict mortality risk in patients with AP and it's correlation with hospital length of stay.
Methods: We conducted a retrospective cohort study of patients who had presented to HCA Healthcare facilities with the diagnosis of AP in the period of January 2011 - January 2021. A Total 514 patients that presented with AP and had a serum LDH data obtained on admission were identified and divided into three groups based on LDH cutoff levels. Length of hospital stay, length of intensive care unit stay (ICU), and mortality rate were compared among groups. Other variables included age, gender, race, BMI, and prior medical history of heart disease or cerebrovascular accident. The study was conducted using a Logistic Regression approach that used statistical analysis to predict the odds of a desired association.
Results: Total 514 eligible patients were identified and divided into three groups. Group 1 consisted of 301 patients with LDH level < 300 IU/L, Group 2 consisted of 158 patients with LDH level between 300-600 IU/L, and Group 3 consisted of 55 patients with LDH level >600. AP patients with an initial LDH value of more than 600 IU/L on admission were likely to have longer hospital length of stay, by 4.5 days on average, 3.2 times more likely to be admitted to ICU, and 12.1 time more likely to expire than patients with an initial LDH value of less than 300 IU/L.
Conclusion: This study has demonstrated that serum LDH is a cheap and a convenient test that can be obtained in AP patients to predict the length of hospital stay, ICU needs, and mortality rates. However, given our study is retrospective, additional randomized controlled studies are necessary to corroborate the beneficial effects of obtaining LDH in patients presenting with AP on admission (Table).

Table 1. The average length of hospital stay, percentage of patients required ICU care, and percentage of patients who has expired in each group along with the median and mode

Groups	Average Length of Hospital Stay	Percentage of Patients required ICU care	Percentage of Patients who has expired
Group 1	8.09	22.92%	3.32%
Group 2	10.03	33.54%	10.76%
Group 3	15.63	54.55%	23.64%

S42

Epidemiology and Outcomes of Sarcopenia in Patients With Chronic Pancreatitis: A Nationwide Population-Based Study

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Introduction: Patients with chronic pancreatitis (CP) frequently have more advanced malnutrition. Various factors such as pain, alcoholism, malabsorption, and maldigestion from pancreatic exocrine insufficiency render these patients at increased risk for sarcopenia. This work intends to assess the prevalence of sarcopenia in CP and its outcomes.
Methods: We reviewed data from a large multi-center database (Explorys IBM) aggregated from 26 large nationwide healthcare systems. Using systemized nomenclature of clinical medical terms (SNOMED CT), CP patients with and without sarcopenia were identified. Additionally, demographic data, including age, sex, race, ethnicity, smoking habits, and outcomes of CP, were obtained. We calculated the odds ratio (OR) using a univariate analysis model.
Results: Among 69 million adult patients in the database, 106,540 patients with CP were identified, and 31.3% of these patients were found to have sarcopenia. Patients with history of sarcopenia were more likely to be elderly (>65-years-old) (OR: 1.26; 95% confidence interval [CI] 1.23-1.29; P < 0.0001), female (OR, 1.11; 95% CI, 1.08–1.13; P < 0.0001), Caucasian (OR, 1.09; 95% CI, 1.06–1.11; P < 0.0001), and smokers (OR 1.53; 95% CI 1.49-1.57, P < 0.0001). CP patients with sarcopenia had increased prevalence of primary pancreatic malignancy (OR, 1.47; 95% CI, 1.4–1.55; P < 0.0001), pancreatic pseudocyst (OR, 1.41; 95% CI, 1.36–1.47; P < 0.0001), exocrine pancreatic insufficiency (OR, 1.93; 95% CI, 1.4–2.65; P = 0.0001), pancreatic malabsorption (OR, 1.72; 95 CI, 1.51-1.97; P < 0.0001), venous thrombosis (OR, 1.57; 95% CI, 1.53–1.62; P < 0.0001), and gastrointestinal hemorrhage (OR, 2.69; 95% CI, 2.63–2.75; P < 0.0001) (Table).
Conclusion: This is the largest study to characterize the epidemiology and outcomes of chronic pancreatitis patients with sarcopenia. Our data indicate that sarcopenia is associated with an increased risk of CP complications and may be an important prognostic factor in CP.

Table 1. Comparison of baseline characteristics and outcomes of CP with and without sarcopenia

	CP with sarcopenia (n=48,220)	CP without sarcopenia (n=106,050)	OR (CI)	P-value
Demographics				
>65	21,970 (46%)	42,250 (40%)	1.26 (1.23-1.29)	< 0.0001
Female	24,410 (51%)	50,970 (48%)	1.11 (1.08-1.13)	< 0.0001
Caucasian	33,810 (70%)	72,500 (68%)	1.09 (1.06-1.11)	< 0.0001
Smoking	31,120 (77%)	72,700 (69%)	1.53 (1.49-1.57)	< 0.0001
CP outcomes				
Exocrine pancreatic insufficiency	70 (0.2%)	80 (0.1%)	1.93 (1.4-2.65)	0.0001
Pancreatic malabsorption	390 (1%)	500 (0.5%)	1.72 (1.51-1.97)	< 0.0001
Pancreatic Pseudocyst	4,640 (10%)	7,440 (7%)	1.41 (1.36 -1.47)	< 0.0001
Primary pancreatic malignancy	2,400 (5%)	3,640 (3%)	1.47 (1.4-1.55)	< 0.0001
Gastrointestinal hemorrhage	15,540 (32%)	24,050 (23%)	2.69 (2.63-2.75)	< 0.0001
Venous thrombosis	10,350 (22%)	15,690 (15%)	1.57 (1.53-1.62)	< 0.0001

S43

Role of Residents' Didactics in Improving Management of Acute Pancreatitis: A Quality Improvement Project

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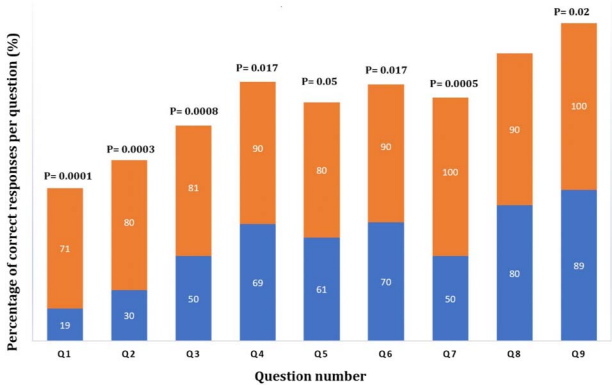
Introduction: Fluid resuscitation is the cornerstone of management of pancreatitis to prevent progression to pancreatic necrosis and multiorgan failure. Current ACG and AGA guidelines acknowledge the importance of intravenous fluids (IVFs) in the first 12-24 hours of presentation. Despite strong evidence for fluid resuscitation in decreasing pancreatitis complications and improving overall mortality, patients are often under resuscitated in the first 24 hours. Often the first providers who encounter patients with acute pancreatitis are residents. In this study we aim to assess the understanding of acute pancreatitis management among internal medicine residents in a teaching hospital and the role of didactics in the education of residents.
Methods: All the residents in the internal medicine residency program were included. Residents were sent an online survey of 9 questions related to management of acute pancreatitis to assess their understanding of the same 1 week prior to didactics. A 20 min lecture by gastroenterology fellow on management of acute pancreatitis was given and the residents were asked to take the same survey again. Responses were assessed before and after didactics. Questions of the survey are in (Table).
Results: A total of 46 residents took pre didactics survey and 41 took post didactics survey. Among the survey takers 30% are PGY1s, 30% are PGY2 and 40% are PGY3 residents. Residents' responses to individual questions are as in (Figure). An absolute increase of ≥50% correct responses were noted with questions related to bolus and maintenance IVFs post didactic session (OR: 9.9; CI: 3.7-26.7 and OR: 9.4; CI: 3.5-25.5 respectively). Post educational interventions there is a 100% relative increase in residents choosing enteral nutrition over parenteral nutrition (OR: 9.2; CI: 2.8-30.2) and are four-fold more likely to initiate diet within first 24 hours of hospitalization (CI: 1.2-13.5). Over all there is approximately 30% improvement in the residents understanding of acute pancreatitis management post educational intervention.
Conclusion: Our study demonstrates the importance of ongoing educational interventions to improve residents' knowledge on management of most common GI conditions. In our study a single 20 min educational intervention improved residents' management choices by 30%. The impact of repeated didactics sessions needs to be further studied. Gastroenterology fellows as teachers can achieve this goal.



[0043] **Figure 1.** Residents responses to online questionnaire before and after didactics Blue color: pre didactics survey, Orange color: post didactics survey

Table 1. Acute Pancreatitis Management Questionnaire

Question Number	Question	Options	Best Answer
1	What is the IVFs rate for INITIAL 30-60 min of acute pancreatitis management?	a. 15-20 cc/kg/hr b. 3-5 cc/kg/hr c. 20-30 cc/kg/hr d. 5-10 cc/kg/hr	A
2	What is the rate of MAINTAINANCE IVFs for 12-24 hours in patients with acute pancreatitis?	a. 3-5 cc/kg/hr b. 5-10 cc/kg/hr c. 10-15 cc/kg/hr	A
3	Based on current evidence-based medicine which of the following are appropriate in acute pancreatitis management?	a. Goal directed judicious IVFs b. Aggressive IV hydration	A
4	Based on current evidence, what type of fluids are ideal for patients with acute pancreatitis?	a. Ringer lactate b. Albumin c. Normal Saline d. Type of fluid doesn't matter as long as patient receives appropriate hydration in the first 24 hours	A
5	What parameters needs to be monitored to direct IVFs rate and predicts mortality?	a. Lactate b. BUN c. Serum creatinine d. White blood cell count	B
6	Based on current evidence-based medicine when can the oral intake be initiated in a patient with acute pancreatitis?	a. Within first 24 hours b. Bowel rest till the abdominal pain is completely better c. can wait up to 1 week	A
7	Based on current evidence-based medicine, in patients with acute necrotizing pancreatitis which of the following is true?	a. Total parenteral nutrition is equivalent to enteral nutrition b. Nasogastric or nasojejunal is not inferior to peripheral parenteral nutrition c. Oral feeding is superior to all other forms of nutrition	C
8	What is the appropriate time for cholecystectomy in patients with acute biliary pancreatitis?	a. Same hospital stay b. Within 6 weeks c. Within 1 week d. Timing doesn't matter	A
9	What type of diet can be initiated in acute pancreatitis?	a. Clear liquids b. Low fat c. Full liquids d. Type of diet doesn't matter as long as patient tolerates	D

S44

The Incidence of Cholangiocarcinoma Among Primary Sclerosing Cholangitis Patients Is Lower Than Previously Reported

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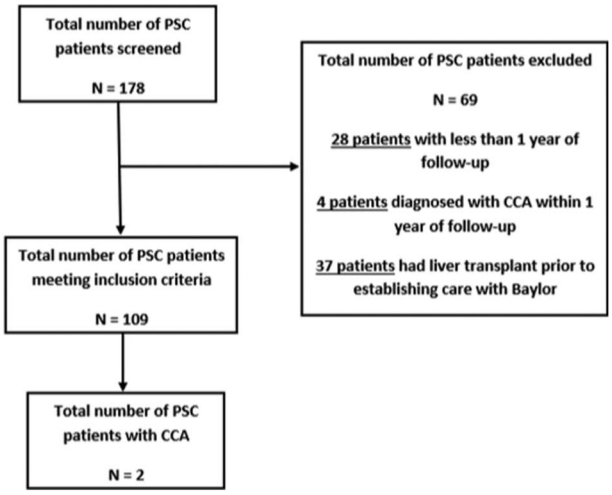
Introduction: Primary sclerosing cholangitis (PSC) is a chronic inflammatory disease involving the bile ducts with increased risk of cancer. The risk of cholangiocarcinoma (CCA) is of particular concern due to high mortality. The relative risk (RR) of CCA in PSC has been reported as high as 1,560 which may have been overestimated. The utility of current surveillance practice in PSC patients for CCA is unclear. We aimed to estimate the relative risk of CCA in PSC patients and effectiveness of surveillance for CCA in this
Methods: Data were retrospectively collected from electronic medical records of patients with a diagnosis of PSC at Baylor St. Luke's Medical Center, Texas from 1998 to 2021. Patients with less than 1 year of follow up were excluded. Demographic data, risk factors, and CA-19-9 levels and biliary imaging records (every 6-12 months) were collected. The incidence of CCA in the study population was compared to the general population using cancer data from the Surveillance, Epidemiology, and End Results (SEER) Program.
Results: A total of 109 patients with imaging or biopsy evidence of PSC were included (Figure). With 532 person-years of follow up, the mean follow-up time was 4.9 (range=1.04-22.9) years per patient. Two patients developed CCA at an annual incidence rate of 0.38% (95% confidence interval [CI]= 0.04%-1.4%). The annual incidence in the SEER registry from 1998 to 2018 was 1.6 per 100,000 person years. The relative risk of CCA in the PSC population was 234.9 (95% CI= 26.4-848.2). IBD, diabetes, alcohol use, and tobacco use were not associated with CCA ($p > 0.05$). The two patients with CCA passed away 165 days and 359 days following the cancer diagnosis. Median peak CA-19-9 level in PSC patients without CCA was 24 U/mL. The two CCA patients had CA-19-9 levels of 3,060 and 21,197 U/mL. Of the two patients with CCA, neither were diagnosed by surveillance but after presenting with obstructive symptoms prompting additional workup. (Figure)

Conclusion: our study shows the risk of CCA among PSC patients is higher than the general population but lower than previously reported. Routine surveillance failed to detect CCA in our patients, who subsequently passed away within 1 year of cancer diagnosis, suggesting current surveillance practice with imaging and CA-19-9 may not help diagnose CCA or improve mortality. This study questions the current practices of CCA surveillance, but further investigation is warranted (Table).



[0044] **Figure 1.** Flow diagram of patients included in the study

Table 1. Characteristics of PSC cohort with and without CCA

Variable		No CCA	CCA
Number of patients		107	2
Age (median [IQR])		48.00 [33.00, 64.50]	55.00 [51.50, 58.50]
Age at PSC diagnosis (median [IQR])		37.00 [24.00, 54.50]	41.50 [34.75, 48.25]
Sex (%)	Male	64 (59.8)	1 (50.0)
	Female	43 (40.2)	1 (50.0)
Race (%)	White	72 (67.3)	1 (50.0)
	Black	25 (23.4)	1 (50.0)
	Other	10 (9.3)	0 (0.0)
IBD (%)	No	41 (38.3)	1 (50.0)
	Yes	65 (60.7)	1 (50.0)
	Unknown	1 (0.9)	0 (0.0)
Cirrhosis (%)	No	35 (33.3)	2 (100.0)
	Yes	70 (66.7)	0 (0.0)
Diabetes mellitus (%)	No	94 (87.9)	2 (100.0)
	Yes	13 (12.1)	0 (0.0)
Alcohol use (%)	Never	57 (53.3)	2 (100.0)
	Previous but quit	33 (30.8)	0 (0.0)
	Current user	17 (15.9)	0 (0.0)
Tobacco use (%)	Never	91 (85.0)	2 (100.0)
	Previous but quit	12 (11.2)	0 (0.0)
	Current user	4 (3.7)	0 (0.0)

IQR = interquartile range.

S45

Trends in the Incidence of Intrahepatic Cholangiocarcinoma Between 1990 and 2019 Stratified by Race, Ethnicity and Gender Among Pennsylvania Residents

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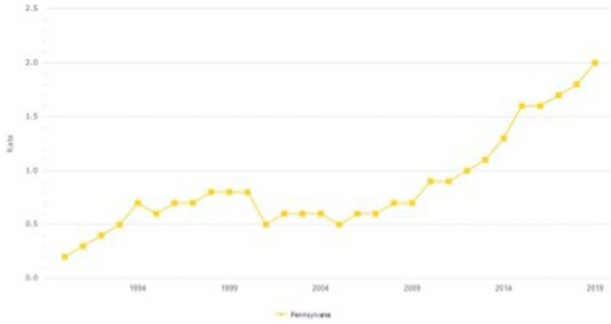
Introduction: Research reports a rising incidence of intrahepatic cholangiocarcinoma (ICC) but recent trends and differences by race/ethnicity and gender have not been explored. Epidemiologic evidence is needed to enable clinicians better understand evolving trends, to inform prevention strategies and screening guidelines. We examined trends in the incidence of ICC among Pennsylvania residents between 1990 and 2019, stratified by age, gender, race, and stage at presentation.
Methods: Repeated cross-sectional analyses were conducted among patients diagnosed with ICC between 1990-2019 in Pennsylvania using data from the Pennsylvania Department of Health Cancer Registry. Temporal trends with age adjusted incidence rates according to race/ethnicity, gender, age and stage at presentation were assessed using the Enterprise Data Dissemination Informatics Exchange (EDDIE).
Results: 4,051 cases of invasive ICC were diagnosed between 1990 and 2019 (Table). The age adjusted incidence rate of ICC increased by a factor of 10, from 0.2 per 100,000 population in 1990 to 2.0 per 100,000 population in 2019 (Figure). The incidence rate was higher among those aged more than 40 years compared to those less than 40 years old (2.24 vs 0.04 per 100,000 population) and higher among males relative to females (1.15 vs 1.03 per 100,000 population). Incidence rate was also highest amongst Whites (1.14 per 100,000 population), compared to Asian/Pacific Islander (0.91 per 100,000 population), Blacks (0.76 per 100,000 population) and Hispanics (0.53 per 100,000 population). 31% of patients presented with stage III/IV disease while 28% presented with stage II disease. The rates per decade increased slightly from the 1990's to the 2000's (0.65 vs 0.74 per 100,000 population) before increasing drastically in the 2010's to 1.83 per 100,000 population.

Conclusion: These results demonstrate significant increase in incidence rates of ICC in recent years. Five-year survival rates of ICC regardless of stage have been reported in other studies at 9%. Given this high mortality and rising incidence rates, clinicians may need to start considering possible screening strategies. Furthermore, other prevention and treatment approaches need to be investigated.



[0045] **Figure 1.** Total Age Adjusted Incidence Rates per 100,000 of Invasive Intrahepatic Cholangiocarcinoma

Table 1. Descriptive statistics and age-adjusted incidence rates for intrahepatic cholangiocarcinoma

	Frequency (%)	Age Adjusted/100,000
Total	4051 (100)	
Age, years		
< 40	74 (2)	0.04
40-54	416 (10)	0.55
55-64	816 (20)	1.93
65-74	1157 (29)	3.65
75-84	1123 (28)	5.65
85+	465 (11)	5.77
By Decade		
1990-1999	786 (19)	0.65
2000-2009	921 (23)	0.74
2010-2019	2344 (58)	1.83
Age group, years		
< 40	74 (2)	0.04
40+	3977 (98)	2.24
Gender		
Male	2083 (51)	1.15
Female	1968 (49)	1.03
Race		
Whites	3626 (89)	1.14
Blacks	301 (7)	0.76
AAPI (2000-2019)	63 (2)	0.91
Hispanics (2002-2019)	68 (2)	0.53
Stage		
I	851 (21)	0.23
II	1134 (28)	0.3
III/IV	1255 (31)	0.34
Unknown	811 (20)	0.22

S46

EUS-Guided Through the Needle Biopsy vs Fine Needle Aspiration for Pancreatic Cystic Lesions: A Systematic Review and Meta-Analysis

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Introduction: Endoscopic Ultrasound guided fine needle aspiration (EUS-FNA) has been widely used to collect samples from pancreatic cystic lesions (PCLs) for cytology and fluid analysis. However, EUS guided FNA has relatively lower sensitivity in discriminating the types of lesions as well as detection of malignancy. Recent studies have investigated the EUS guided through the needle biopsy (EUS-TTNB) as an

alternative method for sample collections in PCLs. We conducted a systemic review and meta-analysis on the studies that compared EUS-FNA and EUS-TTNB for adequate sampling and diagnostic accuracy in patients with PCLs.

Methods: We performed a comprehensive search of the databases: PubMed/MEDLINE, Embase, and the Cochrane Central Register of Controlled Trials from inception through May 10th, 2022. We considered randomized controlled trials, cohort studies, and case-control studies. We excluded abstracts, animal studies, case reports, reviews, editorials, and letters to editors. The primary outcome was sample adequacy which is defined as the presence of enough sample for histopathological evaluation. The secondary outcome was sample accuracy which is defined as the ability to have a definite diagnosis. The random-effects model was used to calculate the risk ratios (RR) and confidence intervals (CI). A p value < 0.05 was considered statistically significant. Heterogeneity was assessed using the Higgins I^2 index.

Results: Nine observational studies involving 520 patients were included in the meta-analysis. The rate of sample adequacy was significantly higher in the EUS-TTNB group compared to the EUS-FNA group (RR 1.64, 95% CI 1.19-2.26, p =0.003, I^2 = 95%) (Figure 1A). Only four studies compared the accuracy rate between the EUS-TTNB method and the EUS-FNA group. The diagnostic accuracy was significantly higher in the EUS-TTNB group compared to the EUS-FNA group (RR 2.03, 95% CI 1.13-3.65, p = 0.02, I^2 = 87%) (Figure 1B).

Conclusion: Our meta-analysis demonstrated that the rates of both sample adequacy and accuracy were higher in the EUS-TTNB group compared to the EUS-FNA group. EUS-TTNB should be considered where applicable clinically for improving the diagnostic yield in patients undergoing evaluation of PCLs. Further randomized controlled trials are needed to confirm our findings.

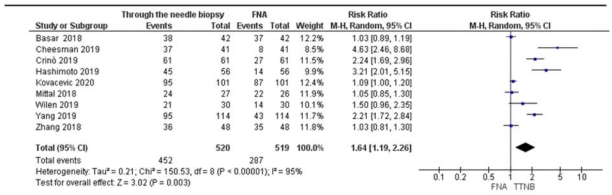

Figure 1 a

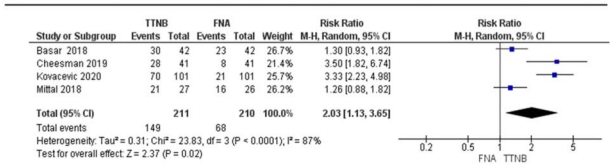

Figure 1 b

[0046] **Figure 1.** a) sample adequacy rate b) diagnostic accuracy rate

S47

Presentation, Management, and Outcomes in Patients With Concomitant Acute Pancreatitis and Acute Kidney Injury

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Introduction: Patients with acute pancreatitis(AP) as well as acute kidney injury(AKI) have been shown to have increased mortality. Although aggressive fluid resuscitation is the mainstay in management for acute pancreatitis, the optimal fluid management strategy in patients with concomitant AKI remain uncertain. We aim to characterize their presentation, management, and clinical outcomes.

Methods: As part of an ongoing prospective study of patients presenting with AP between 1/2015 and 3/2021 to a safety-net tertiary care hospital, we evaluated patients who presented with AKI, defined as a creatinine(Cr) increase of ≥0.3mg/dL from baseline or increase in serum Cr to 1.5 times baseline. Our primary outcome was the development of volume overload as evidenced by granular data such as pleural effusions or peripheral edema. Secondary outcomes were mortality, ICU admission, development of severe pancreatitis, and length of stay. Categorical variables were compared using chi square analysis and continuous variables were compared using Student's t-test. Logistic and linear multivariate regression models were performed controlling for baseline characteristics (age, sex, race) and clinical features such as etiology of pancreatitis and co-morbidities.

Results: A total of 1494 patients who presented with AP were included in the analysis; among those, 214 also had an AKI(14%). The mean admission Cr in the AKI cohort was 2.07 mg/dL. Baseline characteristics are described in Table: Patients with co-morbidities such as DM, CHF, and cirrhosis were more likely to present with AKI. Patients with an AKI were more likely to have SIRS on presentation and require ICU on admission. They also received more fluids, 3,974mL on average in the first 24 hours, with a mean difference of 590mL above the non-AKI cohort (p = .002). In a multivariate analysis, patients with concomitant AKI and AP were more likely to develop signs of volume overload including new pleural effusions (OR 2.29, 95% CI 1.26 – 4.15) and peripheral edema (OR 2.52, 95% CI 1.02 – 6.21). They were also more likely to develop severe pancreatitis (OR 8.7 95% CI 4.8 – 15.6) and had an increased length of stay of 4.7 days over the average 5.7 days (95% CI 1.67 – 7.8).

Conclusion: Patients with concurrent AP and AKI are at risk for both more severe pancreatitis and adverse events related to volume overload. This highlights the challenges with defining a fluid management strategy in this population. More studies are needed to optimize the care of these patients.

Table 1. Acute Kidney Injury in Patients with Acute Pancreatitis: Baseline characteristics, Fluid Management, Clinical Outcomes Footnotes: p = <0.05 for all comparisons in this Table

	No AKI N = 1280	*AKI N = 214*
Age Mean± SD (Mean difference 10.1)	43.5± 14.9	53.6 ± 16.6
Sex	667 males (53%)	140 Males (65%)
Diabetes	287 (22.9%)	97 (45.3%)
CHF	29 (2.3%)	14 (6.6%)
CKD	36 (2.9%)	34 (15.9%)
Cirrhosis	55 (4.4%)	28 (13.1%)
SIRS on presentation	331 (26.4%)	106 (49.5%)
ICU on admission	127 (10.1%)	71 (33.2%)
Fluid Management		
Fluids given in first 24H Mean± SD (Mean Difference 590 mL)	3384 mL ± 2058	3974 mL ± 2687
Fluids given in second 24H Mean± SD (Mean difference 443mL)	2538 mL ± 2017	2981 mL ± 2361
Clinical Outcomes in patients with AKI		

Table 1. (*continued*)

	No AKI N = 1280	*AKI N = 214*
New pleural effusion OR (95% CI)		2.29 (1.26 – 4.15)
New peripheral edema OR (95% CI)		2.52 (1.02 – 6.21)
Severe Pancreatitis OR (95% CI)		8.7 (4.8 – 15.6)
Length of stay Additional days from mean 5.7 days		4.74 days, p = .003

S48

Rates of Retained Biliary Stents Could Be Affected by Socioeconomic Status and Language Barrier

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Introduction: Endoscopically placed common bile duct stents are used for biliary decompression. Studies have reported median patency of plastic stents between 77 to 126 days. It is advisable to remove or exchange these stents within three months of the index procedure to prevent complications such as stent occlusion, dysfunction, migration, and cholangitis. We hypothesized that the risk of retained biliary stents might be high in the underserved population owing to low socio-economic status, language and intellectual barriers, and demographical distribution.
Methods: A retrospective study was conducted amongst all patients who underwent ERCP-guided plastic biliary stent placement between January 2016 and December 2021 at our community-based institution. Charts were reviewed to collect demographics, index ERCP, removal/exchange procedure, complications, and follow-up office visits. Retained biliary stents were defined as patients who did not show up for their follow-up ERCP for stent removal (missed stent group) or those who presented for stent removal later than the recommended time frame of 3 months (Delayed stent removal group). Descriptive analysis was performed. Chi-square and Fisher exact tests were used to compare categorical variables and t-tests for continuous variables.
Results: A total of 431 ERCPs were performed, out of which 46 (10.7%) patients had retained stents. Fifty percent of the cohort (n=23) were white and 63% (n=29) were females. 10 patients (21.8%) were non-English speakers, and 10 patients (21.8%) were non-insured. 32 (69.6%) of the index ERCPs performed were done in the outpatient setting. Our ERCP reports stated the recommended follow-up time for stent removal for all patients whether performed in the inpatient or outpatient setting. However, 8/14 (57%) of the inpatients did not have these instructions included in their discharge summaries. No statistical significance was seen when comparing missed versus delayed stent removal groups, except for the ERCP location (Table).
Conclusion: Socio-economic and demographic factors, including the language barrier and lack of insurance as well as the absence of specific and clear follow-up dates on the discharge instructions from the hospital, could be associated with retained biliary stents.

Table 1. **Factors contributing to a higher risk of retained biliary stents**

		Missed n=17	Delayed n=29	P-value	Total n=46
Race	Non- White	11 (64.7%)	12 (41.4%)	0.127	23 (50%)
Sex	Female	12 (70.6%)	17 (58.6%)	0.416	29 (63%)
Language	Non-English	4 (23.5%)	6 (20.7%)	0.821	10 (21.8%)
Insurance	Non-insured	3 (17.6%)	7 (24.1%)	0.606	10 (21.8%)
ERCP location	Inpatient Outpatient	10 (58.8%) 3 (17.6%)	4 (13.8%) 29 (100%)	0.001 < 0.0001	14 (30.4%) 32 (69.6%)

S49

Differential Diagnosis and Management of Subcentimeter Solid Pancreatic Lesions

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Introduction: Improved imaging techniques allow the detection of small pancreatic solid lesions, with lesions as small as 4 mm can be detected by EUS. Given the risk of malignancy of these lesions, evaluation by EUS-guided fine needle aspiration (EUS-FNA) is commonly warranted. The differential diagnosis of lesions ≤ 1 cm detected on EUS has not been thoroughly elucidated, and guidance on the appropriate management strategy remains limited. The aim of this study is to determine the etiology of small pancreatic solid lesions ≤ 1 cm in maximal diameter to optimize clinical management.
Methods: Over a period of 10 years, patients with pancreatic solid lesions ≤ 1 cm in size detected on EUS with a definite histologic or cytologic diagnosis were retrospectively evaluated. Cystic or semisolid lesions were excluded. Indication, EUS characteristics, and further management was evaluated.
Results: A total of 118 patients with solid pancreatic lesions ≤ 1 cm on EUS were included in our analysis. The median age was 59 years (48-66) and 55.1% were females. 79.6% of patients were referred for an EUS for an incidental nodule detected on cross-sectional imaging. The median lesion size was 8 mm (range 3.8-10 mm), and most common location was pancreatic tail (34.7%). Endosonographically, most lesions were described as hypoechoic, and 66.9% as regular. Nearly half of the patients had more than one pancreatic lesion on EUS. On cytology obtained by FNA, 65.3% were neuroendocrine tumors, 11.9% were metastatic, and 4.2% showed pancreatic adenocarcinoma (PDAC). Renal cell carcinoma was the most common metastatic lesion in 64.3% of the cases. The median number of passes obtained by EUS-FNA was 2. Most patients were managed conservatively with repeat cross-sectional imaging 49.2% of the cases and repeat EUS 13.6% of the time. 18.6% underwent surgical resection while 7.6% had systemic chemotherapy or radiation. 1 and 2-year survival were 94.9% and 86% respectively. There was no significant difference in 2-year survival according to the etiology.
Conclusion: This retrospective study shows the etiologic variability of subcentimeter pancreatic solid lesions discovered on EUS. Only a minority of patients were diagnosed with pancreatic adenocarcinoma. EUS-FNA can provide useful adjunctive information to optimize management strategies. Accurate characterization of small solid pancreatic lesions without delay is crucial and could avoid morbidity associated with pancreatic surgery by reliable diagnosis of non-pancreatic adenocarcinoma (Table).

Table 1. **Differential Diagnosis and Management of Subcentimeter Solid Pancreatic Lesions.**

Variable (N=118)	**Value**
Female, N (%)	65 (55.1)
Age (years), median (IQR)	59 (48-66)
EUS Indication	
Incidental finding on cross-sectional imaging	94 (79.6)
Pancreatic cancer screening	11 (9.3)

Table 1. (*continued*)

Variable (N=118)	Value
Follow-up of known pancreatic cystic lesion	6 (5.1)
Evaluation of subepithelial lesion	1 (0.8)
Esophageal cancer surveillance	1 (0.8)
Characteristics on EUS	
Lesion size (mm), median (IQR)	8 (6-9.2)
Head/uncinate process nodule, N (%)	38 (32.2)
Pancreatic body/neck nodule, N (%)	39 (33.05)
Pancreatic tail nodule, N (%)	41 (34.75)
Hypoechoic, N (%)	111 (94.1)
Hyperechoic, N (%)	2 (1.7)
Isoechoic, N (%)	5 (4.2)
Irregular nodule, N (%)	29 (24.6)
Regular Nodule, N (%)	79 (66.9)
Additional solid lesion on EUS, N (%)	58 (49.2)
Additional cystic lesion on EUS, N (%)	25 (21.2)
Evidence of chronic pancreatitis, N (%)	2 (1.7)
Pancreatic duct dilation (>3 mm), N (%)	12 (10.2)
Number of Passes on EUS-FNA, median (IQR)	2 (2-3)
Procedure time (minutes), median (IQR)	66.5 (53-85.5)
Management	
Repeat EUS, N (%)	16 (13.6)
Surgical resection, N (%)	22 (18.6)
Systemic chemotherapy or radiation, N (%)	9 (7.6)
Palliative therapy, N (%)	2 (1.7)
Follow-up with cross-sectional imaging, N (%)	58 (49.2)
Diagnosis	
Neuroendocrine tumor, N (%)	77 (65.3)
Metastatic lesion, N (%)	14 (11.9)
Benign lesion, N (%)	14 (11.9)
Indeterminate, N (%)	8 (6.8)
Pancreatic adenocarcinoma, N (%)	5 (4.2)

S50

Insulin Therapy vs Plasmapheresis in Patients With Hypertriglyceridemia-Associated Pancreatitis: A Systematic Review and Meta-Analysis

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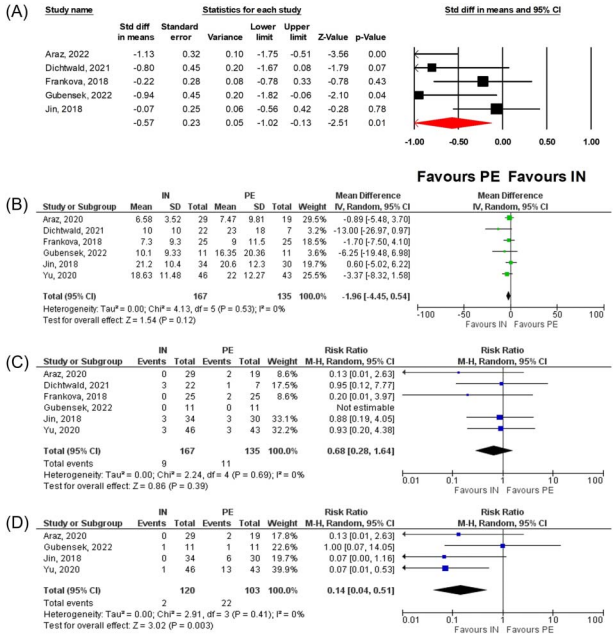
Introduction: Insulin therapy (IT) and plasmapheresis are used to treat hypertriglyceridemia-associated pancreatitis (HTAP). However, the optimal treatment modality for lowering the triglyceride level in patients with HTAP remains unclear. Therefore, we evaluated the efficacy and safety of IT and plasmapheresis in managing HTAP.
Methods: We performed a comprehensive literature search using PubMed, Embase, and Web of Science databases through May 30, 2022, for all studies that compared IT vs. plasmapheresis in patients with HTAP. The primary outcomes were effectiveness (reduction in triglycerides within 24-hours of admission) and clinical outcomes, including hospital length-of-stay (LOS), mortality, acute renal failure (ARF), hypotension, and need for invasive mechanical ventilation (IMV). The secondary outcome was the overall treatment-related adverse events (AEs). Random-effects meta-analysis was conducted, and risk ratio (RR) and mean difference (MD) or standardized mean difference (SMD) for proportional and continuous variables were computed, respectively. For each outcome, forest plot, 95% confidence interval (CI), P-value (< 0.05 considered statistically significant), and I^2 statistic (> 50% considered as significant heterogeneity) were generated.
Results: Six studies (1 randomized controlled trial [RCT] and 5 cohort studies) with 302 patients with HTAP (167 on IT vs. 135 on plasmapheresis) were included. Plasmapheresis was more effective than IT in reduction of triglycerides within 24-hours (SMD -0.57; 95% CI -1.02, -0.13; P=0.01, I^2=56.8%, Figure 1A). However, LOS (MD -1.96; 95% CI -4.45, 0.54; P=0.12; I^2=0%, Figure 1B), mortality (RR 0.68, 95% CI 0.28-1.64, P=0.39, I^2=0%, Figure 1C), ARF (RR 0.44, 95% CI 0.06-3.05, P=0.41, I^2=84%), hypotension (RR 0.63, 95% CI 0.16-2.52, P=0.51, I^2=79%), and need for IMV (RR 0.52, 95% CI 0.12-12.35, P=0.40, I^2=80%) were similar between two groups. The treatment-related AEs were significantly lower in IT than plasmapheresis (RR 0.14, 95% CI 0.04-0.51, P=0.003, I^2=0%, Figure 1D).
Conclusion: Our meta-analysis demonstrated that despite the greater reduction of triglycerides with plasmapheresis compared to insulin therapy, the clinical outcomes, including LOS, mortality, ARF, hypotension, and need for IMV, were comparable with lower treatment-related adverse events with insulin therapy. Future large-scale RCTs are necessary to validate our findings.



[0050] **Figure 1.** Forest plots comparing between insulin therapy and plasmapheresis regarding: (A) the reduction of triglyceride within 24 hours, (B) length of hospital stay, (C) mortality, and (D) treatment-related adverse events.

S51

Trends in Acute Pancreatitis With Cannabis Use Disorder and Subsequent Acute Kidney Injury in Young Adults: Evidence From Nationwide Cohort

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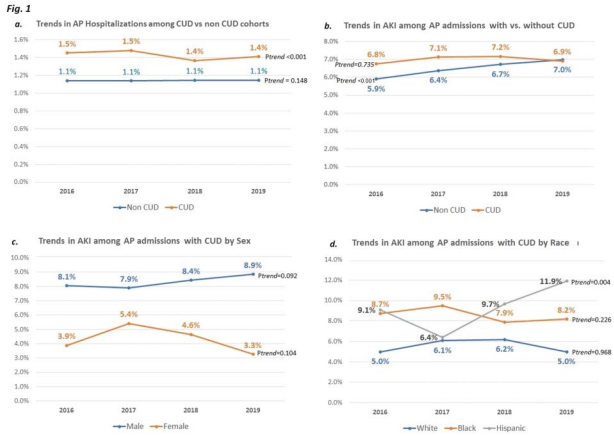
Introduction: Cannabis has been a well-recognized etiology of acute pancreatitis (AP) over last decade with some controversies. With increased availability of cannabis for recreational and medicinal purposes in managing chronic pain, it is essential to evaluate potential risk to multi-organ system. However, there is scarcity of large-scale data on the burden and risk of AP and subsequent acute kidney injury (AKI) among cannabis users.

Methods: Using weighted discharges from the National Inpatient Sample (2016-2019), young (18-44 years) patients primarily admitted for AP with concomitant cannabis use disorder (CUD) were identified. Primary endpoints included odds and trends in AP with vs. without CUD, subsequent trends in AKI with associated sex/racial disparities and all-cause mortality. Secondary endpoints included patient disposition, hospital stay, and cost.

Results: Of total 395,215 AP admissions, 29,815 (7.5%) were among patients with CUD. Crude rate of AP in CUD cohort was slightly higher vs. non-CUD admissions (n=29,815, 1.4 % vs. n=365,400, 1.1%), however, with slightly lower odds when adjusted for confounders (aOR 0.94, 95%CI:0.90-0.97, p< 0.001). The AP-CUD cohort often consisted of younger (median 33 vs 35 years), male (67.5% vs. 53.9%), blacks (28.9% vs 17.6%), patients from lowest income quartile, Medicaid enrollees vs. non-CUD cohort. Alcohol abuse, smoking, and drug abuse were significant higher in the AP-CUD cohort. Rate of AKI in AP-CUD cohort was 7% vs 6.5 % in non-CUD. Interestingly, among AP-CUD cohort, the rate of AKI was higher in males (8.3%, p_{trend} =0.092) vs females (4.3%, p_{trend} =0.104) with steady trends. The burden and trend of AKI in AP-CUD cohort was higher in Hispanics (Hispanic 9.3%, black 8.6%, white 5.6%; H: 9.1% in 2016 to 11.9% in 2017 p_{trend}< 0.004) (Figure). There was no significant difference in all-cause mortality between CUD and non-CUD cohorts (aOR 0.40; 95%CI:0.16-1.03, p=0.058). Routine discharges and median length of stay were comparable between cohorts (3 days) with an average cost was $25,724 in the AP-CUD cohort.

Conclusion: There was a higher crude rate of AP admissions in the young CUD cohort, however, when adjusted for confounders, CUD was not independently associated with higher risk of AP, subsequent inpatient mortality, or rising trends in AKI. Hispanics demonstrated concerning trends in AKI. Future studies are warranted assessing long-term impact of polysubstance use on AP admissions and outcomes.



[0051] **Figure 1.** Trends in Acute Kidney Injury among Acute Pancreatitis admissions with Cannabis users by sex and race.

S52

African Americans With Pancreatic Adenocarcinoma Have Higher Inpatient Mortality at a Younger Age: A Nationwide Inpatient Sample Study

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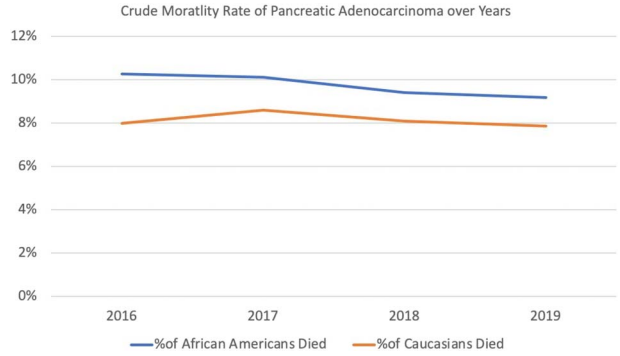
Introduction: Pancreatic Adenocarcinoma (PA) is currently the 4th leading cause of cancer related death worldwide and its prevalence has increased over the last decade. PA has a high mortality rate because symptoms of PA does not become apparent until later stages. Risk factors for PA include nicotine, alcohol abuse, obesity, genetic predispositions, and chronic pancreatitis. Until today, no specific screening test for PA has been identified for early diagnosis of PA.
Methods: We utilized the Nationwide Inpatient Sample (NIS) database from 2016 to 2019. Adult hospitalizations due to PA were identified by previously validated ICD-10-CM codes. We subsequently divided admissions from PA into two groups based on race (Caucasians (CA) and African Americans (AA). Univariate and multivariate logistic regression for categorical variables and linear regression for continuous variables were carried out to identify independent associations at $p < 0.05$. Statistical Analysis was performed using R studio.
Results: 513,285 patients had admissions related to PA with 83,655 (16%) were AA. AA patients were younger as compared to CA (65 vs 69 years, p value < 0.01). AA females had higher incidence of PA as compared to CA females (55% vs 48%, p-value < 0.01). 50% of AA patients were from lower household income group of $1-24,999. 17% of AA patients were on medicaid as compared to 6.6% amongst CA patients. AA patients had a higher prevalence of obesity, diabetes, smoking, and chronic pancreatitis. AA patients have statistically significantly higher rates of crude mortality rate, and increased complications as reported in Table 1. On multivariate analysis, AA patients had a higher risk of inpatient mortality compared to CA patients (OR 1.21, 95% CI: 1.14–1.28); greater risk of sepsis compared to CA (OR 1.35, 95% CI: 1.17-1.55); higher risk of requiring ICU level of care (Mechanical Ventilation and Vasopressor use) and increased length of stay (LOS) (Table 2).
Conclusion: AA patients were younger, belonged to lower income group, were on medicaid, had higher prevalence of risk factors for PA, had higher inpatient mortality and complications during inpatient admissions eventually increasing LOS as compared to CA patients. Further studies are needed to evaluate if being AA is an inherent risk factor for PA given the higher incidence in this population. Furthermore, healthcare utilization should be more focused on AA patients given the higher mortality rate and complications during inpatient admissions.



[0052] **Figure 1.** Trend of Crude Mortality Rate AA vs CA Over Years

Table 1.

Table:	Demographics	Caucasians, N = 429,630	African American, N = 83,655	p-value
	Age (median, IQR)#	69 (61, 77)	65 (57, 73)	< 0.001
	Gender			
	Male	224,055 (52%)	37,600 (45%)	< 0.001
	Female	205,365 (48%)	46,025 (55%)	
	Bed size of hospital			< 0.001
	Small	71,650 (17%)	12,645 (15%)	
	Medium	114,000 (27%)	22,825 (27%)	
	Large	243,980 (57%)	48,185 (58%)	
	Location/teaching status of hospital			< 0.001
	Rural	32,705 (7.6%)	3,130 (3.7%)	
	Urban nonteaching	78,820 (18%)	11,105 (13%)	
	Urban Teaching	318,105 (74%)	69,420 (83%)	
	Region of hospital			< 0.001
	North East	99,065 (23%)	15,995 (19%)	
	Mid West	106,150 (25%)	16,730 (20%)	
	South	149,825 (35%)	43,785 (52%)	
	West	74,590 (17%)	7,145 (8.5%)	
	Primary expected payer			< 0.001
	Medicare	272,095 (63%)	44,435 (53%)	

Table 1. (*continued*)

Table:	Demographics	Caucasians, N = 429,630	African American, N = 83,655	p-value
	Medicaid	28,140 (6.6%)	14,355 (17%)	
	Private Insurance	111,945 (26%)	19,445 (23%)	
	Self Pay	6,095 (1.4%)	2,475 (3.0%)	
	No charge	460 (0.1%)	185 (0.2%)	
	Other	10,440 (2.4%)	2,655 (3.2%)	
	Year			0.1
	2016	104,140 (24%)	19,640 (23%)	
	2017	105,230 (24%)	21,055 (25%)	
	2018	107,615 (25%)	21,115 (25%)	
	2019	112,645 (26%)	21,845 (26%)	
	Income			< 0.001
	$1-24,999	89,490 (21%)	41,545 (50%)	
	$25,000-34,999	110,250 (26%)	17,365 (21%)	
	$35,000-44,999	113,410 (27%)	14,030 (17%)	
	$45,000 or more	110,175 (26%)	9,375 (11%)	
	Smoking	3,350 (0.8%)	960 (1.1%)	< 0.001
	Alcohol	1,650 (0.4%)	305 (0.4%)	0.7
	Age Group			< 0.001
	Less than 50 Years	26,965 (6.3%)	8,710 (10%)	
	Greater than 50 years	402,665 (94%)	74,945 (90%)	
	Diabetes	87,750 (20%)	20,015 (24%)	< 0.001
	Hyperlipidemia	154,550 (36%)	25,120 (30%)	< 0.001
	Hypertension	69,070 (16%)	19,040 (23%)	< 0.001
	Acute pancreatitis	76,280 (18%)	14,620 (17%)	0.4
	Chronic pancreatitis	12,085 (2.8%)	2,665 (3.2%)	0.008
	Obesity	34,950 (8.1%)	7,375 (8.8%)	0.003
Table 2:	Outcomes (Univariate Analysis)			
	Died during hospitalization	34,945 (8.1%)	8,135 (9.7%)	< 0.001
	Vasopressor_Use	4,915 (1.1%)	1,260 (1.5%)	< 0.001
	Mechanical_Ventilation	10,545 (2.5%)	2,945 (3.5%)	< 0.001
	Sepsis	5,460 (1.3%)	1,455 (1.7%)	< 0.001
	Shock	23,630 (5.5%)	5,525 (6.6%)	< 0.001
	AKI*	85,350 (20%)	22,750 (27%)	< 0.001
	Length of stay (Median, IQR)#	4.0 (3.0, 7.0)	5.0 (3.0, 9.0)	< 0.001
	Total Charges in Dollars	45,284 (24,252, 85,750)	48,805 (25,838, 91,718)	< 0.001
Table 3:	Outcomes (Multivariate analysis)	Reference: Caucasians		
		aOR	Range	p-value
	Inpatient Mortality	1.21	1.14-1.28	< 0.01
	Sepsis	1.35	1.17-1.55	< 0.01
	Shock	1.2	1.12-1.29	< 0.01
	AKI*	1.49	1.43-1.56	< 0.01
	Mechanical Ventilation	1.35	1.23-1.49	< 0.01
	Vasopressor Use	1.33	1.15-1.55	< 0.01

#IQR: Interquartile Range
*AKI: Acute Kidney Injury

S53

Disparities in the Readmission Rates After Hospitalization for Acute Pancreatitis

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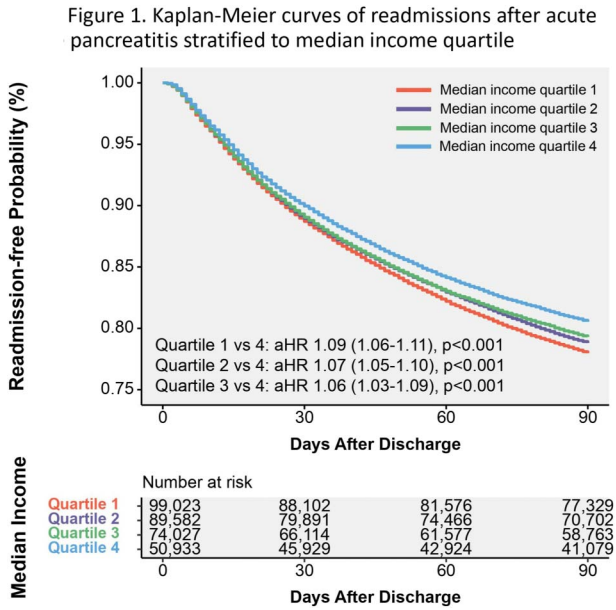
Introduction: Acute pancreatitis (AP) is one of the leading causes of hospitalization in the United States. Patients with AP frequently get readmitted and that proves to be a great burden on our healthcare system. Age, male sex, tobacco use, alcohol use, and necrotizing pancreatitis have been linked to a higher readmission rate in AP patients. However, studies on the impact of socioeconomic status are lacking. Our study attempts to find the impact of income on the readmission rates in patients admitted for AP.
Methods: We queried the National Readmission Database (NRD) from 2017 to 2018 to identify patients with a primary diagnosis of AP. Those who died during the index hospitalization were excluded when examining readmission rates. Multivariable Cox proportional-hazards regression was used to study the impact of income on 90-day readmission adjusted for age, sex, comorbidities, hospital characteristics, and primary payer.
Results: A total of 313,565 AP patients were identified, 99,023 of whom belonged to median income quartile 1, 89,582 to quartile 2, 74,027 to quartile 3, and 50,933 to quartile 4 with quartile 4 having the highest income. Slightly more than half of each quartile group were men (53.1%, 52.8%, 53.0%, 53.5%, respectively). Patients with lower income had higher prevalence of comorbidities. Among those who survived the index hospitalization, 21,694 (21.9%), 18,880 (21.1%), 15,264 (20.6%), and 9,854 (19.3%) patients in quartiles 1, 2, 3, and 4 were readmitted within 90 days, respectively. When compared with the highest income quartile (quartile 4), patients in quartiles 1, 2, and 3 were associated with an increasingly higher hazard of readmission (quartile 1, aHR 1.09, p< 0.001; quartile 2, aHR 1.07, p< 0.001; quartile 3, aHR 1.06, p< 0.001). (Figure)
Conclusion: Our study finds a positive correlation between poverty and the 90-day readmission rate in AP patients. Alcohol use is one of the top causes of pancreatitis in the US. It would be interesting to investigate if there is a difference in the prevalence of substance use in patients of various economic strata that could be impacting the readmission. Furthermore, it is imperative that we bring reforms in our financial and health policy to make it more equiTable in order to alleviate some of the disparities we see.



[0053] **Figure 1.** Kaplan-Meier curves of readmissions after acute pancreatitis stratified to median income quartile

S54

Trends in Race-Specific Mortality From Pancreatic Cancer in the United States Between 2000 and 2020

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Introduction: Pancreatic cancer is the 3rd leading cause of cancer-related death in the United States after lung and colon cancers. In this study, we sought to identify the trends in race-specific mortality from pancreatic cancer in the United States
Methods: Using the multiple cause of death database (ICD-10 revision codes), we identified all patients who died of pancreatic cancer (C25.x registered as the underlying cause of death) in all races (White, Black, Asian or Pacific Islander, and American Indian or Alaska Native) between 2000 and 2020 in the United States. Age-adjusted mortality rates were calculated per 1000,000 persons (PMP), standardized to the US census data from 2000, and stratified by race
Results: Between 2000 and 2020, a total of 780,134 pancreatic cancer deaths were identified in all races, with an overall age-adjusted mortality of 108.8 PMP. We identified a total of 662,076 deaths, 93,111 deaths, 21,478 deaths, and 3,469 deaths in the White, Black, Asian or Pacific Islander, and American Indian or Alaska Native populations respectively. The overall age adjusted mortality were 134 PMP, 107.8 PMP, 75.2 PMP and 65 in the Black, White, Asian or Pacific Islander, and American Indian or Alaska Native populations respectively. Over the 20 years, the age-adjusted mortality decreased by 8% in Black (from 140.1 PMP in 2000 to 129.5 PMP in 2020), increased by 7% in White (from 103.6 PMP in 2000 to 110.6 PMP in 2020), 1% in Asian or Pacific Islander (from 73.8 PMP in 2000 to 74.7 PMP in 2020), and16% in American Indian or Alaska Native (from 58.3 PMP in 2000 to 67.9 PMP in 2020)
Conclusion: This study concludes that between 2000 and 2020, pancreatic cancer mortality is highest in Black and lowest in Asian or Pacific Islander. In Black, it is twice that in Asian or Pacific Islander. Interestingly, over the twenty years, the mortality has decreased in Black but it increased in all other races, particularly in American Indian or Alaska Native.

S55

Racial Disparities and Social Determinants in Pancreatic Transplantation

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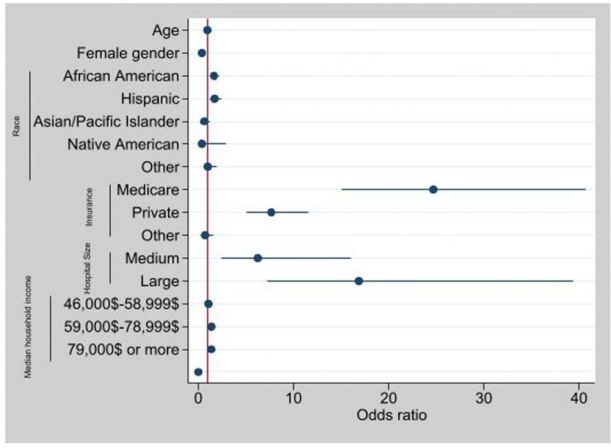
Introduction: Pancreatic transplantation is still the only treatment that can restore normoglycemic and complete insulin independence. Potentially life-threatening complications of diabetes, hypoglycemia unawareness, and intolerable problems with insulin therapy can all be indications of pancreatic transplantation. In this study, we aimed to investigate the healthcare determinants and racial disparities in pancreatic transplantation.
Methods: A retrospective study was conducted utilizing the Nationwide Inpatient Sample database (NIS) for the years 2016 to 2018. Patients who underwent pancreatic transplantation were identified using ICD-10 diagnosis codes from all listed discharge diagnoses. Patients younger than 18 years of age, and missing information for age, gender, or race were excluded. Multivariate logistic regression analysis was performed to compare different predictors of pancreatic transplantation in the included population.
Results: A total of 2530 patients underwent pancreatic transplantation were identified during the study period. Compared to White patients, African American and Hispanic patients had increased transplant rates (OR 1.64, 95% CI 1.26-2.14, P-value < 0.00) and (OR 1.72, 95% CI 1.23-2.42, P-value 0.02), respectively. While Native American and Asian/Pacific Islander patients had statistically insignificant results. Female gender was associated with decreased transplant rates (OR 0.93, 95% CI 0.925-0.936, P-value < 0.00). Patients with Medicare and private insurances had considerably higher transplant rates compared to those with Medicaid (OR 24.7, 95% CI 14.9-40.7, P-value < 0.00) and (OR 7.66, 95% CI 5.07-11.57, P-value < 0.00), respectively. In addition, the 59,000$-78,999$ and 79,000$ or more income quartile were associated with higher transplant rates (OR 24.7, 95% CI 14.9-40.7, P-value < 0.00) and (OR 7.66, 95% CI 5.07-11.57, P-value < 0.00), respectively.
Conclusion: This study demonstrated that race and social determinants of health, including gender, income, and insurance status, are associated with the likelihood of receiving pancreatic transplantation in the United States. Poverty and unequal access to health care can contribute to health care disparities. National-level initiatives raising public and provider awareness of racial and social disparities in care are needed in addition to expanding health insurance coverage. (Figure)



[0055] **Figure 1.** Multivariate logistic regression analysis investigates different variables in predicting the likelihood of pancreatic transplantation in the United States.

S56

Perfusing the Pancreas: A Quality Improvement Project to Optimize Pancreatitis Management in a Tertiary Academic Center

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Introduction: Acute pancreatitis (AP) is one of the leading gastrointestinal (GI) causes of hospitalization. Numerous studies have addressed the role of early fluid resuscitation and nutrition initiation in reducing the morbidity and length of stay associated with AP. Multiple GI societies have developed guidelines on the optimal management of AP. At our institution, we observed variations in the management of AP from current guidelines. We aim to identify the trends in the management of AP and the barriers to guideline adherence. We plan to use this data to enact systematic interventions that improve adherence to guideline recommendations.
Methods: We evaluated all patients hospitalized with a diagnosis of acute or acute-on-chronic pancreatitis (ACP) from September to December 2021. Pre-intervention data on fluid resuscitation and initiation of oral diet/enteral feeding were collected. A survey was also developed and sent to hospital medicine and teaching teams to identify potential barriers. A Pareto chart showed that knowledge was not the primary barrier. Rather, there were clinical concerns that aggressive fluid resuscitation and early nutrition initiation could lead to potential complications of volume overload and worsening pancreatitis severity.
Results: We reviewed data for 74 patients admitted with a diagnosis of AP or ACP. 93% of patients received intravenous fluids (IVF) within the first 24 hours, but only 8.5% received the recommended resuscitation rate of at least 250 cc/hr. With regards to nutrition, 46% of patients admitted with AP received oral/enteral nutrition within the first 24 hours. A Pareto chart of these responses identified that the biggest barriers to aggressive IVF resuscitation were concern for volume overload and patient co-morbidities, such as congestive heart failure and chronic kidney disease. A subsequent secondary analysis showed that the concern for CHF and CKD may not be valid, as 81% of patients admitted for AP did not have these co-morbidities. For early nutrition, the biggest barriers were concern for clinical worsening and PO intolerance.
Conclusion: Our QI project aims to optimize pancreatitis management at our institution by improving adherence to early fluid resuscitation and oral/enteral nutrition initiation. A pro forma is in development to standardize management and will be distributed to clinical staff. Post-intervention data will be collected to assess for improvement.

S57

Opioid Use Patterns in Patients With Chronic Pancreatitis and Concomitant Mental Illness: A Propensity-Matched Cohort Analysis

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Introduction: Chronic pancreatitis (CP) is commonly associated with pain that can be difficult to manage. This can result in high rates of healthcare utilization and chronic opioid use. Many patients with CP have concomitant mental health disorders (MHDs) which may be associated with greater disease burden. Genetic links between CP-associated pain and MHDs have been described. Given the current opioid epidemic trend, it is critical to assess whether concomitant MHDs affect opioid usage patterns in patients with CP. The aim of this study was to identify whether patients with coexisting CP and mental illness have higher opioid usage compared to their counterparts without any diagnosed mental illness.
Methods: This was a large cohort study using TriNetX, which links anonymized inpatient and outpatient electronic health record data from more than 80,000,000 patients. We created two cohorts: patients with chronic pancreatitis from 2010-2020 with and without co-existing mental health disorders. The cohorts underwent 1:1 propensity matching based on age, sex, race, ethnicity, and alcohol and nicotine dependence. The primary outcome was a new diagnosis of opioid use disorder at 3 years after the first diagnosis of CP. Secondary outcomes at 3 years included any opioid use, long-term opioid use, opioid overdose, and all-cause mortality.
Results: After matching, two cohorts of 48,960 patients remained for analysis. All covariates were well-matched. In the MHD cohort, 51.6% had mood /affective disorders (F30-39), 49.5% had anxiety (F40-48), and 5.9% had non-mood psychotic (F20-29). At 3 years, the MHD cohort had a near 5-fold increase in the rate of opioid use disorder (7.1% vs 1.6%, OR =4.8, 95% CI 4.4-5.2). The MHD cohort also had significantly higher rates of any opiate use,(OR 1.5, 95% CI 1.4-1.5), long-term opioid use (OR 2.2, 95% CI 2.1-2.3), opioid overdose (OR 4.2, 95% CI 3.7-4.6), and all-cause mortality (OR 1.3, 95% CI 1.2-1.3) (Table).
Conclusion: In propensity-matched cohorts, patients with CP and concomitant MHDs had higher rates of opioid use disorder, long-term use, overdose, and all-cause mortality compared with those without MHDs. As CP-related pain has been shown to be augmented in patients with MHDs – specifically depression – identifying this opioid risk is critical in the management of these patients.

Table 1. Measures of Association. CP w/ MHD: chronic pancreatitis with mental health disorder cohort, CP w/o MHD: chronic pancreatitis without mental health disorder cohort

Outcome	Patients with outcome (% risk), CP w/ MHD	Patients with outcome (% risk), CP w/o MHD	Odds Ratio (OR)	95% Confidence Interval (CI)	p-value
Opioid use disorder (dependence or abuse)	3,467 (7.1%)	767 (1.6%)	4.8	4.4-5.2	< 0.0001
Opiate use (ever)	13,846 (28.3%)	10,310 (21.1%)	1.5	1.4-1.5	< 0.0001
Adverse opiate event	592 (1.2%)	158 (0.3%)	3.8	1.4-1.5	< 0.0001
Opiate overdose	1,809 (3.7%)	448 (0.9%)	4.2	3.7-4.6	< 0.0001
Long-term opiate analgesic use	9,896 (20.2%)	5,046 (10.3%)	2.2	2.1-2.3	< 0.0001
All-cause mortality	6,958 (14.4%)	5,702 (11.8%)	1.3	1.2-1.3	< 0.0001

S58

Endoscopic Retrograde Cholangiopancreatography After Pancreaticoduodenectomy and the Utility of Rigidizing Overtube in Improving Procedural Success – A Tertiary Cancer Center Experience

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Introduction: Endoscopic retrograde cholangiopancreatography (ERCP) remains a challenge in patients with a pancreaticoduodenectomy (PD). Advances in endoscopic technology may improve success of the ERCP after PD. Herein we report outcomes of ERCP after PD at a tertiary cancer center. We also summarize our experience with the use of a novel rigidizing overtube (RO) for ERCP post- PD.
Methods: We performed retrospective review of patients with prior classic or pylorus-preserving PD who underwent ERCP with Pathfinder use at MD Anderson Cancer Center from 2006 to 2021. Outcomes included technical success (TS) rate, which was defined as successful cannulation of biliary tree and treatment of stricture with dilation or stent placement when applicable, and clinical success (CS) rate, defined as improvement in patient symptomatology with or without normalization of bilirubin level.
Results: A total of 48 patients underwent 102 ERCPs; RO was used in 11 procedures (**Table**). ERCP was most commonly done for cholangitis in index and repeat cases. In cases where RO was used, 63.6% had biliary obstruction without cholangitis. TS was achieved in 81% of cases without RO and 91% cases with RO. Stent placement was done in 60% and 64% of cases without and with RO, respectively. The CS rate was 67%% and 100% of cases without and with RO, respectively. The overall adverse event rate was 5%. All adverse events occurred in cases where no RO was used, with an adverse event rate of 6% in cases without RO used; no complications were noted in the RO cases.
Conclusion: ERCP after PD, despite being technically challenging, carries a favorable success rate. TS of ERCP after PD may be improved with use of RO based on our limited experience. There is a need of randomized controlled trials to further ascertain the utility of RO in improving TS and CS of ERCP in surgically altered anatomy. (**Figure**)

Endoscopic Retrograde Cholangiopancreatography after Pancreaticoduodenectomy; Patient Characteristics – 48 Patients; 102 Procedures	
Age in years, median (IQR)	68.0 (50.5-81.6)
Male	31 (65%)
Race, n (%)	
White	37 (77%)
Hispanic	8 (17%)
Black or African American	2 (4%)
Asian	1 (2%)
Indication for Pancreaticoduodenectomy, n (%)	
Pancreatic Adenocarcinoma	27 (56%)
Pancreatic Neuroendocrine Carcinoma	6 (13%)
Intraductal Papillary Mucinous Neoplasms	3 (6%)
Ampullary Carcinoma	4 (8%)
Duodenal Carcinoma	5 (10%)
Cholangiocarcinoma	1 (2%)
Neoadjuvant Therapy, n (%)	
Chemoradiation	14* (29%)
Chemotherapy	8 (17%)
Immunotherapy	2 (4%)
*1 patient received only radiotherapy	

Indication for Initial ERCP (N=48)	*Number of patients, n (%)*
Cholangitis	22 (46%)
Biliary Obstruction Without Cholangitis	16 (33%)
Pancreatitis	3 (6%)
Choledochojejunostomy Stricture	3 (6%)
Elective Stent Removal/Exchange	3 (6%)
Other	1 (2%)
Indication for repeat ERCP (N=54)	*Number of ERCPs, n (%)*
Cholangitis	20 (39%)
Choledochojejunostomy Stricture	12 (23%)
Elective Stent Placement/Exchange/Removal	10 (19%)
Biliary Obstruction Without Cholangitis	11 (21%)
Biliary calculi	1 (2%)
Total	54
1 patient with abdominal pain and jaundice was categorized to have biliary obstruction without cholangitis	

[0058] **Figure 1.** ERCP After Pancreaticoduodenectomy: Patient Characteristics and Indication for ERCP

Table 1. ERCP Results

	ERCP without Rigidizing Overtube	ERCP with Rigidizing Overtube
Technical Success, *n (%)*	74/91 (81.0%)	10/11 (90.9%)
Clinical Success, *n (%)*	57/85 (76.2%)	10/10** (100.0%)
Adverse Events, *n (%)*	5/91 (6.0%)	0/11 (0.0%)
Post-procedure Fever, *n (%)*	4/5 (80.0%)	-
Cholangitis, *n (%)*	1/5 (20.0%)	-

**No follow-up data available for 1 case.

S59

EUS-Guided Portal Pressure Gradient Measurements to Diagnose Cirrhosis: A Systematic Review and Meta-Analysis

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Introduction: Measuring portal pressure gradient helps to assess the severity of complications in patients with chronic liver disease. EUS guided portal pressure gradient (PPG) measurement is a novel technique to assess portal hypertension. This is a systematic review and meta-analysis to assess the safety and efficacy of this novel method to assess portal pressure gradient.
Methods: Selection criteria included studies with EUS guided PPG measurement. Data was collected and extracted from medline, pubmed, and Ovid journals. Statistical analysis was done using fixed and random effects models to calculate the pooled proportions.
Results: On initial search 136 articles were found, out of which 51 were selected and data was extracted from 4 studies (n=128) that looked at EUS-guided PPG measurements. The pooled proportions of patients with successful portal pressure measurement was 91.61% (95% CI = 86.25 to 95.74). Patients with failed portal pressure measurement had a pooled proportion of 2.22% (95% CI = 0.40 to 5.45). The pooled analysis of patients with PPG >5 mmHg was 53.06% (95% CI = 44.48 to 61.55) and patients with clinically significant PPG >10 mmHg was 30.51% (95% CI = 22.92 to 38.67). We assessed all patients with clinically significant PPG for esophago-gastric varices and the pooled data of patients with varices were 31.65% (95% CI = 23.96 to 39.87). Post-procedural complications included post-procedure bleeding, perforation, and infection with a pooled proportion of 0% (95% CI = 0 to 2.85). The pooled analysis for post-procedure abdominal pain was 6.15% (95% CI = 2.68 to 10.91), emergency department visits was 3.11% (95% CI = 0.83 to 6.77), and post-procedural sore throat was 2.82% (95% CI = 0.68 to 6.35). Publication bias calculated by using the Harbord-Egger bias indicator gave a value of 1.48 (95% CI = -1.03 to 4.00, p = 0.12). The Begg-Mazumdar indicator gave Kendall's tau b value of 1 (p = 0.08).
Conclusion: EUS guided PPG measurement is a novel method to assess portal hypertension and can be used as an alternative for IR guided portal pressure measurement. It has technical success and minimal post procedural complications. There is good correlation with clinical portal hypertension and portal pressure gradients. This can be used as a one stop shop to assess varices, portal pressure measurement, and liver biopsy under one anesthetic procedure which makes it a more efficient and cost effective alternative for an IR procedure.

S60

Cannabis Use in Patients With Chronic Pancreatitis Improves in Hospital Outcomes

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Introduction: Background: Chronic pancreatitis is a debilitating, progressive, and irreversible disorder characterized by a cycle of inflammation and fibrosis. The etiology of chronic pancreatitis is broad and ranges from genetic to anatomic factors. Chronic pancreatitis is also a painful disorder with numerous patients using narcotics for relief. We aimed to study the clinical outcomes of patients with chronic pancreatitis with a concomitant diagnosis of cannabis use in comparison to patients with chronic pancreatitis who do not use cannabis.
Methods: The NIS database was queried for the years 2015-2019. Adult patients (>age 18) with a diagnosis of cannabis use and chronic pancreatitis versus those with chronic pancreatitis only as a principal discharge diagnosis were identified using ICD-10 codes. The primary outcome was inpatient mortality. Secondary outcomes were hospital length of stay (LOS) and total hospital charges (TOTHC). Statistical analysis was performed using STATA.
Results: We identified 153,407 patients who had chronic pancreatitis, of which 8,985 patients had a concomitant diagnosis of cannabis use. After propensity score matching, patients with a diagnosis of chronic pancreatitis and cannabis use had decreased mortality (OR 0.34, p< 0.0001, CI:0.27-0.45), decreased LOS (-0.98 days, p< 0.0001, CI: 1.12 to -0.83) and decreased (TOTHC -$13,845, p< 0.0001, CI: -$15,954 to -11,736) compared to patients with only a diagnosis of chronic pancreatitis.
Conclusion: Patients with chronic pancreatitis who use cannabis interestingly had lower mortality, LOS, and TOTHC compared to patients with chronic pancreatitis who do not use cannabis. This is an important study that demonstrates that cannabis use is not detrimental and can be effective in controlling symptoms and improving outcomes in certain patient populations. Further randomized controlled trials are necessary to further illustrate our results.

S61

Opiate Use Is Associated With Increased Risk of Post ERCP Pancreatitis: A National Cohort Study

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Introduction: Endoscopic retrograde cholangiopancreatography (ERCP) has evolved as a valuable tool in therapeutic management of pancreatobiliary disorders but is associated with a range of significant complications, the most common being post-ERCP pancreatitis (PEP). The aim of this study was to elucidate the impact of opiate use on the development and severity of post-endoscopic retrograde cholangiopancreatography. Pancreatitis (PEP).
Methods: We retrospectively analyzed patients who were hospitalized in 2017 using the national inpatient sample (NIS) database who underwent endoscopic retrograde cholangiopancreatography (ERCP) (via ICD-10 procedure codes). Our study group consisted of patients who had a secondary diagnosis of opiate use, and our control group consisted of patients who did not have a secondary diagnosis of opiate use. Our primary end point was to elucidate if the presence of opiate use increased the risk of developing post-ercp pancreatitis. Additionally, we assessed if opiate use worsened outcomes in patients who underwent ERCP. We accomplished this by extracting variables such as length of hospital stay (LOS), total hospital charges and in hospital mortality rates. Categorical variables were compared using the chi-square test, and continuous variables were compared using t-test. Multivariate regressions models and all statistical analysis were performed using STATA 16 software.
Results: A total of 172,255 patients who underwent ERCP were identified, of which 2,120 were opiate users. Opiate users were hospitalized at a younger age when compared to opiate non-users (53.7 years vs 60.5 years; p-value: < 0.01), had a longer length of hospital stay (8.3 days vs 6.0 days; p-value: < 0.01), total hospital charges ($102,469 vs $82,191; p-value: < 0.01) and higher incidence of PEP (4% vs 2.7%; p-value: < 0.01).
Conclusion: Opiate use is associated with younger age of presentation, increased LOS, total hospital charges, and increased frequency of PEP amongst patients undergoing ERCP. Further investigation is needed to assess the extent to which chronic opiate use may be a risk factor for post-ERCP pancreatitis and worse outcomes. If so, discontinuation of opiates before ERCP may be reasonable when possible. (Table)

Table 1. Hospital outcomes in opiate users vs non-users undergoing ERCP

	Opiate Use (N=2,120)	No Opiate Use (N=170,135)	P-value
Age of presentation	53.7	60.5	< 0.01
LOS (days)	8.3	6.0	< 0.01
Total charges (USD)	$102,469	$82,191	0.087
Died	35 (1.6%)	2,520 (1.4%)	0.525
PEP	85 (4%)	4,740 (2.7%)	< 0.01

LOS: Length of hospital stay; USD: United States Dollar; PEP: Post-ERCP Pancreatitis.

S62

Benefits of Using Pancreatic Cyst Fluid Glucose in Community-Based Setting to Evaluate Pancreatic Cystic Lesions

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Introduction: In the evaluation of incidental asymptomatic pancreatic cystic lesions (PCLs), it is necessary to identify mucinous cysts due to their risk for malignant transformation.[1] Current guidelines recommend obtaining cyst fluid carcinoembryonic antigen (f-CEA), but this test has limitations.[2] Recently, a low cyst fluid glucose level (f-Glu) < 50 mg/dl has emerged as an alternative to f-CEA.[3]
Methods: Patients from 11/2021-4/2022 with a PCL were retrospectively analyzed. Demographics, radiology & EUS findings, & test results were collected. f-CEA and f-Glu were ordered on each patient, with priority placed on f-CEA, followed by f-Glu.
Results: 13 patients (7 M:6 F, median age 74) with PCLs were analyzed. The median cyst size was 27 mm (16.8-77 mm), and the median fluid volume obtained was 1.5 mL (0-170 mL). In our patients, both f-CEA and f-Glu were available in 46% (6/13), only f-CEA in 2/13 (15%), and only f-Glu in 3/13 (23%). In 2 patients, f-CEA/f-Glu could not be obtained. In 6 patients, when both f-CEA and f-Glu were available, there was 100% agreement between the two test results. In 5/13 cases, f-CEA could not be obtained. In 2 cases, f-Glu was the only test result that was obtained.
Conclusion: Since pancreatic f-Glu has emerged as an alternative to f-CEA,[3] we aimed to incorporate f-Glu in evaluating PCLs in our community-based EUS program. In our study, f-Glu did correlate with f-CEA results. Unfortunately, in a community hospital, f-CEA requires >3 mL for analysis. In contrast, f-Glu requires >1 mL, and is readily available, making it an ideal alternative to f-CEA. In our series, the median fluid volume aspirated was 1.5 mL, and in 38% of cases, f-CEA could not be obtained. In 69% of cases, f-Glu was measured despite low fluid volume (< 3 mL), making it advantageous in the evaluation of a PCL. In this limited series, f-Glu was a more favorable alternative to f-CEA in a community-based EUS program. (Table).

Table 1. Summary of Patient Characteristics and Test Results

Patient	Age	Sex	f-CEA	f-Glu	Cyst Size (mm)	Amt of Cyst Fluid (mL)	Cytology
1	78	M	N/A	127	27 × 21	N/A	No malignancy
2	82	M	N/A	< 10	70 × 50	N/A	Degenerated cells
3	74	F	15.5	N/A	19 × 9	1	N/A
4	79	M	N/A	N/A	38 × 32	N/A	Mucin and cell atypia
5	58	M	107	20	26 × 25	4.5	Inflammatory cells
6	81	F	N/A	N/A	33 × 23	N/A	Mucin and papillary cells w/ LGD
7	74	F	789	N/A	19 × 11	1.5	N/A
8	66	M	216	< 10	21.6 × 16.8	3	No malignancy
9	55	F	29	19	58 × 65	5	No malignancy
10	84	F	N/A	< 10	14.8 × 16.8	1	No malignancy
11	64	M	457	< 10	22.5 × 20.4	2	Fibrovascular tissue w/ scant fragments of reactive glandular tissue
12	65	F	88,809	< 10	77 × 44	170	No malignancy
13	80	M	532	< 10	50	7	No malignancy

N/A, not available.

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S63

Prevalence of Depression in Patients With Chronic Pancreatitis: A Systematic Review and Meta-Analysis

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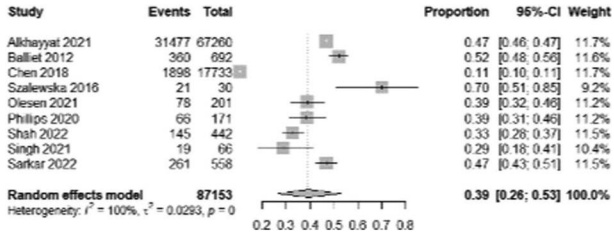
Introduction: Chronic Pancreatitis (CP) is a fibroinflammatory condition with debilitating symptoms affecting 35-50% of individuals worldwide. Quality of life is severely affected in patient with CP and they are likely to suffer from mental health disorders, including depression. We conducted a systematic review and meta-analysis assessing the prevalence of Depression in patients with CP.
Methods: Multiple electronic databases including PubMed, MEDLINE (OVID), PsycINFO, Cochrane Library, Embase, Scopus and Web of Science were searched from inception to June 2022 to identify manuscripts reporting the prevalence of Depression (diagnosed clinically or with a validated scale without language restriction) among patients with Chronic Pancreatitis of any etiology. Case series, reports, editorials, and reviews were excluded. Two independent reviewers extracted data. Disagreements were resolved by a third author. The pooled prevalence was calculated using a random effects model. Heterogeneity was assessed by the inconsistency index (I2).
Results: Among 3647 articles identified, 58 studies were identified for full text review, and ultimately nine studies were deemed eligible for inclusion with perfect agreement (kappa=1.0). Five of them were cross-sectional studies, whereas the rest were cohorts. A total of 87136 patients were included in the studies (**Table**). CP was determined with previous historical diagnosis, the M-ANNHEIM criteria and the presence of a clinical, radiologic and/or endosonographic features. Depression was diagnosed clinically and using Center for Epidemiological Studies 10-

item Depression Scale (CESD), Beck Depression Inventory (BDI) and Hospital Anxiety and Depression Scale (HADS). Depression prevalence in patients with CP varied from 11%-70% among the studies. The pooled prevalence of Depression in CP patients was 39% (95% CI 0.26-0.53) **(Figure)**. Four studies included healthy controls with lower prevalences of Depression.
Conclusion: Depression among patients with CP deserves a call-to-action due to its medical implications and worsening quality of life. Chronic pain and malabsorption symptoms could predispose to the development of Depression. Our findings raise awareness of the importance of screening patients with chronic pancreatitis for mental health disorders and provide support. Further well-designed clinical trials would be necessary to test tentative therapeutic measures seeking the reduction of symptoms for this specific group of patients.



[0063] **Figure 1.** Forest plot of the prevalence of Depression in patients with Chronic Pancreatitis

Table 1. Baseline characteristics of included studies

Study	Design	Country	Duration of follow up or data collection	Study Population	Chronic Pancreatitis (N)	Etiology of Chronic Pancreatitis	Diagnosis of Chronic Pancreatitis	Depression (N)	Depression Diagnosis	Cutoff for Depression	Controls (N)	Type of Controls	Depression in Chronic Pancreatitis (%)	Depression in Controls (%)
Alkhayyat (2021)	Retrospective cohort analysis	USA	2014 -2019	Patients with diagnosis of CP	67,260	Any etiology	Previous diagnosis (chart review)	31477	Clinical	NA	30,209,550	Matched healthy controls	47%	14.7%
Balliet (2012)	Cross-sectional	USA	NA	Patients with nonalcohol-related CP	692	Nonalcohol-related	Previous diagnosis (chart review)	360	Center for Epidemiological Studies Depression Scale	scores ≥ 10	NA	NA	52%	NA
Chen (2018)	Retrospective cohort	Taiwan	2000-2010	Patients with newly diagnosed CP	17,733	Any etiology	Previous diagnosis (chart review)	1, 898	Clinical	NA	35,466	Matched healthy controls	11%	0.9%
Szalewska (2016)	Cross-sectional	Poland	NA	Patients with CP exacerbation	30	Any etiology	Previous diagnosis (chart review)	21	Beck Depression Inventory	scores >10	NA	NA	70%	NA
Olesen (2021)	Cross-sectional	USA and Denmark	2016-2021	Patients with diagnosis of CP	184	Any etiology	M-ANNHEIM criteria: (Cambridge III or IV or pancreatic calcifications on cross-sectional imaging)	71	Hospital Anxiety and Depression Scale	scores >7	NA	NA	39%	NA
Phillips (2020)	Cross-sectional	USA and Denmark	NA	Patients with diagnosis of CP	171	Any etiology	M-ANNHEIM criteria: (Cambridge III or IV or pancreatic calcifications on cross-sectional imaging)	66	Hospital Anxiety and Depression Scale	scores >7	NA	NA	39%	NA
Shah (2022)	Cross-sectional	USA	2016- 2021	Patients with CP with and without chronic opioid use	442	Any etiology	Presence of a clinical and radiologic (CT or MRCP) and/ or endosonographic features	145	Clinical	NA	NA	NA	33%	NA
Singh (2021)	Prospective cohort study	India	2013-2014	Patients with Idiopathic CP, with/without diabetes, and >18 years of age	66	Idiopathic CP with/without Diabetes	Previous diagnosis based on United European Gastroenterology Guidelines	19	Hospital Anxiety and Depression Scale (HADS)	scores >7	152	Matched healthy controls	29%	1%
Sarkar (2022)	Prospective observational study	India	NA	Patients between 18-60yrs with documented CP who visited the Pancreas Clinic	558	Any etiology	1. Pancreatic calcifications on imaging. 2. Pancreatic ductal changes on MRCP or ERCP per Cambridge criteria 3. Ductal and parenchymal changes on EUS per Rosemont criteria	261	Beck Depression Inventory II	scores >13	67	Matched healthy relatives or caregivers	47%	7.5%

S64

A Case Series to Assess Effectiveness and Complications of Percutaneous Transhepatic Cholangioscopy With Spyglass™ Direct Visualization System in Individuals With Altered Gut Anatomy

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Introduction: Endoscopic retrograde cholangiopancreatography (ERCP) can be challenging or impossible for management of obstructive bile duct pathology in individuals with altered gut anatomy. Percutaneous transhepatic cholangiography (PTC) with drain placement usually achieves adequate drainage but is limited with regards to therapeutic options. Percutaneous cholangioscopy (PC) using Spyglass™ via PTC route can allow for meaningful interventions like those performed during ERCP.
Methods: We describe a case series of fourteen patients who underwent PTC drain placement followed by PC performed in collaboration between gastroenterology and interventional radiology between January 2015 to May 2022 at Henry Ford Hospital. Cases were identified by searching for relevant billing codes on ProVation databases. Each chart was then individually reviewed to extract relevant information such as indication, procedural details and complications.
Results: Fourteen patients underwent PTC drain placement with IR followed by subsequent PC at a later date. Most patients (92.9%) had choledocholithiasis on imaging and (57.1%) had cholangitis on initial presentation. Mean age of the population was 66.4 years and majority were female 64.3%. All individuals had altered anatomy, with Roux-en-Y gastric bypass in 71.4%, duodenal switch anatomy in 14.2%, Billroth II and Roux-en-Y hepaticojejunostomy in 7.1% each. Three had prior failed ERCP attempts, and one had a prior failed EDGE procedure attempt. Pre-procedural labs showed elevated alanine amino transferase with mean of 89.9 IU/L, alkaline phosphatase 295.4 IU/L and total bilirubin at 1.8 mg/dL. Multiple stones were discovered in 71.4% of patients, single stone 14.2%, stricture 21.3% and no stone seen in 7.1%. Three patients had benign biliary strictures evaluated by spybites. Electrohydraulic Lithotripsy (EHL) was utilized in 57.1%, retrieval balloons in 35.7% and basket in 14.3% of cases to achieve duct clearance. Median one session was required for duct clearance and procedure was described as successful in all cases of choledocholithiasis (92.9%). No complications were seen with any of the fourteen cases, specifically pancreatitis, infection or thirty-day mortality.
Conclusion: In cases with altered anatomy making ERCP challenging or impossible, cholangioscopy via PTC route is a viable therapeutic option with low risks and high success rate. Further research is needed to compare this approach to other options, including an EDGE procedure where anatomy allows. (Table).

Table 1. Patient and procedural characteristics	
Age, Mean ± SD	66.4 (12.0)
Gender, Male N (%)	5 (35.7)
Indication, N (%)	
Stone Removal/Decompression	13 (92.9)
Stricture Diagnosis	1 (7.1)
Altered anatomy. N (%)	
Roux-en-Y Gastric Bypass	10 (71.4)
Billroth II	1 (7.1)
Duodenal Switch	2 (14.2)
Hepaticojejunostomy	1 (7.1)
Prior ERCP Attempt, N (%)	3 (21.4)
Prior EDGE Attempt, N (%)	1 (7.1)
Preprocedural Labs, Mean ± SD	
ALT (IU/L)	89.9 ± 116.1
ALP (IU/L)	295.4 ± 210.6
Total Bilirubin (mg/dL)	1.8 ± 1.9
Findings N (%)	
Multiple Stones	10 (71.4)
Single Stone	2 (14.2)
Stricture	3 (21.3)
No Stone	1 (7.1)
Number of Sessions, Median (IQR)	1 (1-2)
Therapeutic Device, N (%)	
EHL	8 (57.1)
Balloon	5 (35.7)
Basket	2 (14.3)
Spybites, N (%)	3 (21.4)
Success, N (%)	13 (92.9)
Complication, N (%)	0 (0)
Follow up Months, Median (IQR)	28.5 (17-36)

ERCP: Endoscopic Retrograde Cholangio Pancreatography, EDGE: Endoscopic ultrasound Directed transGastric ERCP, ALT: Alanine aminotransferase, ALP: Alkaline Phosphatase.

S65

Distinct Imaging Patterns of Immune Checkpoint Inhibitor-Induced Acute Pancreatitis

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Introduction: The presentation of immune checkpoint inhibitor (ICI)-induced pancreatic injury ranges from asymptomatic hyperlipasemia to acute pancreatitis. Imaging features of ICI-induced acute pancreatitis (ICI-AP) remain poorly described. We evaluated radiographic patterns of pancreatic inflammation in patients with ICI-AP.
Methods: We assessed a retrospective cohort of patients diagnosed with ICI-AP after initiation of ICI therapy between 2011 and 2019. Abdominal imaging findings with computed tomography (CT), magnetic resonance imaging (MRI), and 18-Fluorine-flurodeoxyglucose positron emission tomography (^{18}F-FDG PET/CT) were reviewed by an independent radiologist. We performed univariate analyses to evaluate associations between clinical characteristics and radiographic patterns of ICI-AP.
Results: Among 6,450 cancer patients treated with ICI, 27 (0.4%) developed ICI-AP. All three Atlanta criteria for AP (typical symptoms, imaging, and lipase ≥3 upper limit of normal) were satisfied in 13 (48%). Abdominal pain was present in 23 (85%), and 4 (15%) presented asymptomatically with hyperlipasemia and pancreatic inflammation on imaging. Diagnostic abdominal imaging was performed in 26/27 patients (mean age 62.4 ± 11.3 years, 46% female, 92% White) via CT in 22 (85%), MRI in 3 (12%), and ^{18}F-FDG PET/CT in 1 (4%). The most frequent radiologic appearance was a diffuse interstitial pattern with diffuse edematous pancreas and peripancreatic stranding, present in 10 (38%) patients, followed by a focal interstitial pattern with focal pancreatic edema and peripancreatic stranding in 7 (26%), and pancreatic enlargement without peripancreatic inflammation in 3 (12%). Six patients (23%) had normal imaging. Individuals with diffuse interstitial inflammation and normal imaging were more likely to be female (p=0.02). Those with diffuse inflammation or pancreatic enlargement without inflammation were older than those with focal inflammation and normal pancreatic imaging (p=0.046).
Conclusion: We propose three distinct radiologic patterns of ICI-AP. The notable subset of patients who were asymptomatic but had imaging evidence of ICI-AP supports the role of abdominal imaging in patients on ICIs with hyperlipasemia. The presence of pancreatic enlargement without peripancreatic inflammation in a small but notable proportion of patients highlights the importance of comparing baseline pancreatic imaging when interpreting imaging for ICI-AP. (Table)

Table 1. Title: Imaging Characteristics in Patients with Immune Checkpoint Inhibitor-Induced Acute Pancreatitis

Characteristics	Overall (N = 26)	Diffuse interstitial pattern (N = 10)	Focal interstitial pattern (N = 7)	Pancreatic enlargement alone (N = 3)	Normal (N = 6)	*P*-value
Age (years), mean (SD)	62.4 (11.3)	65.6 (11.9)	57.4 (14.6)	70.0 (3.6)	59.2 (4.0)	**0.046**
Female, n (%)	12 (46.2%)	6 (60.0%)	1 (14.3%)	-	5 (83.3%)	**0.02**
Race (White) n (%)	24 (92.3%)	9 (90.0%)	7 (100%)	2 (66.7%)	6 (100%)	0.27
Cancer type, n (%)						0.40
Melanoma	11 (42.3%)	3 (30.0%)	2 (28.6%)	2 (66.7%)	4 (66.7%)	

Table 1. (*continued*)

Characteristics	Overall (N = 26)	Diffuse interstitial pattern (N = 10)	Focal interstitial pattern (N = 7)	Pancreatic enlargement alone (N = 3)	Normal (N = 6)	*P*-value
Non-melanoma	15 (57.8%)	7 (70.0%)	5 (71.4%)	1 (33.3%)	2 (33.3%)	
ICI class, n (%)						0.77
CTLA4	2 (7.7%)	-	1 (14.3%)	-	1 (16.7%)	
PD1/PDL1	18 (69.2%)	8 (80.0%)	5 (71.4%)	2 (66.7%)	3 (50.0%)	
CTLA4 + PD1/PDL1	6 (23.1%)	2 (20.0%)	1 (14.3%)	1 (33.3%)	2 (33.3%)	
Pancreatitis severity by revised Atlanta criteria, n (%)						0.38
Mild	23 (88.5%)	9 (90.0%)	7 (100%)	3 (100%)	4 (66.7%)	
Moderately severe	3 (11.5%)	1 (10.0%)	-	-	2 (33.3%)	
Lipase severity by CTCAE v.5						0.99
3	4 (15.4%)	2 (20.0%)	1 (14.3%)	-	1 (16.7%)	
4	22 (84.6%)	8 (80.0%)	6 (85.7%)	3 (100%)	5 (83.3%)	
Days from ICI initiation to pancreatitis, mean (SD)	161.8 (242.5)	134.0 (148.7)	156.1 (158.8)	451.0 (633.4)	70.2 (40.7)	0.52
Duration of lipase elevation (days), mean (SD)	65.4 (81.1)	54.2 (62.3)	41.0 (37.0)	32.3 (43.4)	129.0 (130.0)	0.66
Managed with steroids, n (%)	21 (80.1%)	8 (80.0%)	5 (71.4%)	2 (66.7%)	6 (100%)	0.53
ICI discontinued due to ICI-induced pancreatic injury, n (%)	19 (73.1%)	7 (70.0%)	5 (71.4%)	1 (33.3%)	6 (100%)	0.23
Response to steroids, n (%)	10 (38.5%)	3 (30.0%)	4 (57.1%)	1 (33.3%)	2 (33.3%)	0.76

Abbreviations: Common Terminology Criteria for Adverse Events version 5.0 (CTCAE v.5); cytotoxic T-lymphocyte-antigen protein-4 (CTLA-4); Immune checkpoint inhibitor (ICI); programmed cell death-1 (PD-1) / PD-1 ligand (PD-L1).

S66

Aggressive Intravenous Hydration in Patients With Acute Pancreatitis Is Beneficial Only During the First 24 Hours: A Meta-Analysis

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Introduction: Early aggressive intravenous hydration (EAIH) remains the only initial treatment specifically for patients with acute pancreatitis. The timing, type, and amount of fluid remains the subject of study, debate, and controversy. In order to define our current state of knowledge of the clinical evidence behind the recommendation for EAIH, we performed a meta-analysis of the published literature.
Methods: A Pubmed search to identify all clinical trials evaluating the role of EAIH in patients with acute pancreatitis resulted acute pancreatitis 25 studies, 17 prospective, 18 retrospective, 13 randomized controlled clinical trials (RCT). Studies that evaluated NS to LR were not included. In order to be included in the final analysis, the study needed to have two or more groups defined by different rates of hydration. One group must have included EAIH and define the outcome(s) studied. Outcomes used in our analysis included SIRS, pancreatic necrosis, persistent organ failure, length of stay, ICU admission, and mortality. Any decrease in these outcomes compared to the "control" groups was considered a "benefit". The weighted mean for each study was defined by the sample size.
Results: In a pooled analysis of all studies (n=1865), there was no difference in morbidity or mortality between EAIH and the "control group" (RR 1.1, 95% CI 0.66-1.8). When including only RCT (n=987), there was also no difference in morbidity or mortality (RR 0.88, 95% CI 0.78-1.4). However, when including studies and RCT that focused only on EAIH within the first 24 hours, excluding all studies that defined EAIH as the amount of fluid provided at 48 hours, there was a "benefit" to EAIH (RR 0.62, 95% CI 0.44-0.84).
Conclusion: Our results show that the benefit observed in EAIH occurs only within 24 hours of enrollment. While guidelines on the management of acute pancreatitis have generally recommended EAIH, some have suggested that "goal directed therapy" is preferred due to complications. Our study demonstrates that it is important for clinicians caring for patients with acute pancreatitis not miss the "goal" within the first day of managing patients with acute pancreatitis. Our meta-analysis shows aggressive intravenous hydration after the first 24 hours is not beneficial and may be harmful. Based on this analysis, we suggest EAIH be applied to all patients during the first 24 hours and "goal directed" hydration after the first 24 hours.

S67

Overutilization of Amylase and Lipase Testing in Acute Pancreatitis

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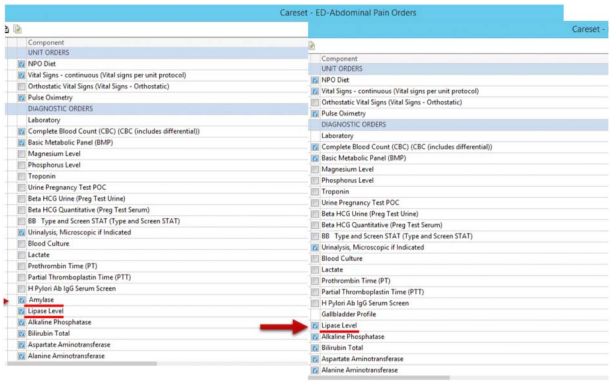
Introduction: Acute pancreatitis (AP) is a common cause of hospitalization and is a substantial financial burden to the healthcare system. The American College of Gastroenterology (ACG) guidelines recommend using serum lipase over amylase to diagnose AP, as it has superior specificity and sensitivity. However, based on our institution's observation, patients diagnosed with AP had both amylase and lipase ordered and had follow-up measurements after diagnosis. We aim to decrease co-testing of amylase and lipase during pancreatitis workup by 40% in 6 months at our community hospital.
Methods: The Institute of Healthcare Improvement model was used for this quality improvement project. A multidisciplinary team approach was utilized. The Plan, Do, Study, Act (PDSA) cycle was used to format this project and test change. Root cause analysis was done using the fishbone diagram.
Results: Pre-intervention, 98% of admissions for AP had serum amylase and lipase ordered at the time of diagnosis, and 82% of patients had repeat testing. In PDSA cycle 1, there was no significant decrease in ordering both amylase and lipase during initial diagnosis, but the intervention decreased repeat testing to 73%. PDSA 2 involved educating residents and the emergency department (ED) about the guidelines. After two cycles failed to result in change, a root cause analysis was done and showed an EMR system order set for abdominal pain with pre-selected amylase and lipase. All patients with abdominal pain had both the tests ordered unless the ordering provider de-selects. In PDSA 3, we used a multidisciplinary approach and worked to remove the pre-selected amylase from the order set. Post EMR change, we reviewed 98 patients over a six-month period who had abdominal pain and acute pancreatitis workup. Results showed a 48% reduction in amylase orders. (Figure)
Conclusion: PDSA 3 cycle involved removing the pre-selected amylase from the ED order set. The average cost of amylase testing is $35. Post-intervention, six-month data revealed a 48% reduction in amylase co-ordering. This result showed a $1645 direct cost saving. At this pace, yearly cost savings of $3290 would be observed. Performing a root cause analysis allows discovering key stakeholder and system components contributing to outcomes. We used a process flow map to identify a system problem, including pre-selecting unnecessary labs during patient admission. Targeting a system change and de-selecting amylase from the order set allowed us to reduce unnecessary testing and hospital costs.



[0067] **Figure 1.** We were successful in removing amylase from the order set. Providers could still select the test, but with no pre-selection.

S68

Alpha-Gal Syndrome Complicating the Management of Suspected Pancreatic Exocrine Insufficiency

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Introduction: IgE antibodies to the oligosaccharide galactose-α-1,3-galactose (α-gal) are an important cause of allergic reactions to mammalian meat and other mammal-derived products. The symptoms of α-gal syndrome (AGS) can involve urticaria or anaphylaxis, but increasingly we are aware that GI tract symptoms, including diarrhea, are also a major feature of AGS. Pancreatic exocrine insufficiency (PEI) is a common cause of diarrhea and treatment involves the use of pancreatic replacement enzymes (PRE). PRE are porcine derived and contain α-gal. Patients receiving PRE who are α-gal IgE positive are at risk for allergic reactions and GI symptoms due to α-gal sensitivity. Here we reviewed patients with suspected PEI and concomitant α-gal IgE sensitization in the practice of one gastroenterologist in Virginia.
Methods: Retrospective chart review was carried out using inclusion criteria of i) diarrhea, ii) low fecal elastase (< 200 **μg/g** feces), and iii) α-gal IgE sensitization (>0.10 kU/L).
Results: 15 patients were identified with mean fecal elastase of 123 and median IgE α-gal level 0.96 (Table). 9 improved off of mammalian-containing food products and 3 of these did not require PRE. 11 patients received PRE. Of 5 patients with pre-existing systemic allergy symptoms to mammalian meat, 1 improved off of mammalian products and did not require PRE, 1 had increased diarrhea with Creon and was lost to follow up, 1 tolerated Creon with pruritus, 1 experienced hives from Creon but successfully underwent office-based desensitization, and 1 patient avoided PRE due to the severity of allergy symptoms. 6 patients without the classic cutaneous allergy symptoms of AGS tolerated PRE, though 1 developed some urticaria.
Conclusion: In this series of 15 patients with suspected PEI who had concomitant α-gal IgE >0.1 kU/L, 60% improved with removal of α-gal containing products from the diet and 20% did not require PRE. Of the 11 patients who were treated with ongoing PRE, 3 experienced urticaria and 1 had increased diarrhea, but none had severe allergic symptoms. In our experience, patients who are sensitized to α-gal can usually tolerate PRE. Practitioners should also be aware that worsening diarrhea during PRE treatment could be a consequence of α-gal-related hypersensitivity, rather than medication non-compliance. Recognition of AGS superimposed upon PEI will allow improve management in this complex patient population.

Table 1. Clinical data from 15 patients with suspected pancreatic exocrine insufficiency diagnosed with concomitant α-gal syndrome

Characteristics	Total Cohort (n=15)
Age, mean years (range)	59.5 (19-80)
Sex, female, n (%)	9 (60%)
Race, Caucasian, n (%)	15 (100%)
Fecal Elastase, mean μg/g fecal material (range)	123 (49-183)
IgE to α-gal, median kU/L (range)	0.96 (0.41-26.1)
Diarrhea Severity recorded, n (%)	13 (87%)
Mild, 0-4 stools per day, n (%)	4 (31%)
Moderate, 5-8 stools per day, n (%)	6 (46%)
Severe, >8 stools per day, n (%)	3 (23%)
Improvement with mammalian avoidance, n (%)	9 (60%)
Treated with PRE, n (%)	11 (73%)
Creon, n (%)	10 (91%)
Zenpep, n (%)	1 (9%)
Allergy Symptoms attributed to PRE, n (%)	4 (36%)
Urticaria, n (%)	3 (27%)
Diarrhea, n (%)	1 (9%)

S69

Two Decades of Trends in Acute Biliary Pancreatitis in Women and the Impact of Management Guidelines

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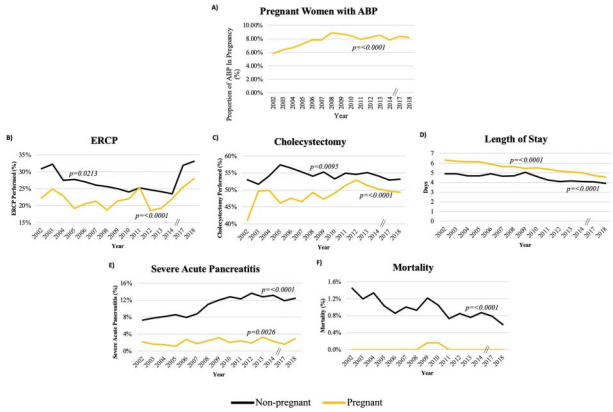
Introduction: An estimated 12% of women will develop cholelithiasis during pregnancy. Nearly 70% of cases of acute pancreatitis in pregnancy are attribuTable to gallstone disease. Acute biliary pancreatitis (ABP) during pregnancy can cause serious morbidity, but there is a paucity of literature on how outcomes and management of ABP in pregnancy have changed with time. Hence, we sought to study trends of ABP and its management in pregnant women over the last two decades at a national level.
Methods: We used the National (Nationwide) Inpatient Sample to identify all women (≥18 years) with an inpatient admission for ABP in the United States from 2002 to 2018. We excluded data from 2015-2016 due to less than expected incidence attribuTable to a transition from the ICD-9 to ICD-10 coding system. Baseline patient characteristics were compared between pregnant and non-pregnant women with ABP utilizing Chi-square and two sample t-tests. Trends in outcomes and management were analyzed using Cochran-Armitage and F-Tests.

Results: Among 40,359 pregnant patients and 476,965 non-pregnant women hospitalized for ABP, a higher proportion of pregnant women with ABP were Hispanic ($p< 0.001$) and used Medicaid ($p< 0.001$) than non-pregnant women on univariate analysis (Table). Trend analyses from 2002-2018 revealed an increasing proportion ($p< 0.001$) of ABP admissions in pregnant females (Figure). An increasing trend was observed for ERCP ($p< 0.001$) and cholecystectomy ($p< 0.001$) in pregnant women admitted for ABP (Figure). Severe acute pancreatitis increased in both pregnant and non-pregnant patients across time, but a lower increase was seen in the pregnant cohort. Overall mortality in the pregnant cohort remained low (< 0.1%), ICU admission was less than 0.6%, and mean length of stay decreased from 5 days to 4 days ($p< 0.001$) for pregnant women with ABP.

Conclusion: Over the last two decades, an increasing proportion of pregnant women are admitted with ABP, and therapeutic interventions (ERCP and cholecystectomy) are occurring more frequently with favorable hospital outcomes. These national-level data strongly support current surgical and endoscopic management guidelines of ABP in pregnancy.



[0069] **Figure 1.** Trends in Acute Biliary Pancreatitis in Non-Pregnant and Pregnant Women

Table 1. Baseline Characteristics for Females Admitted with Acute Biliary Pancreatitis

Variable	Non-Pregnant (n=476,965)		Pregnant (n=40,359)		
	n	%	n	%	p-value
Age (mean, SE)	55.97	0.14	26.12	0.07	< 0.0001
Race					
White	251,213	52.67	14,326	35.5	< 0.0001
Black	45,633	9.57	3,668	9.09	
Hispanic	81,010	16.98	13,645	33.81	
Other/Missing	99,110	20.78	8,719	21.6	
Median Income					
Low (0-25th percentile)	129,345	29.66	12,303	32.63	< 0.0001
Moderate (26th to 50th percentile)	115,539	26.5	10,597	28.11	
High (51st to 75th percentile)	104,546	23.97	9,168	24.32	
Very High (76th to 100th percentile)	86,648	19.87	5,633	14.94	
Type of Insurance					
Medicare	183,908	38.62	358	0.89	< 0.0001
Medicaid	74,005	15.54	21,620	53.68	
Private	156,631	32.89	14,291	35.48	
Other, self-pay, no charge	61,623	12.94	4,009	9.95	
Type of Hospital					
Rural	61,686	12.97	3,608	8.98	< 0.0001
Urban non-teaching	203,194	42.74	15,545	38.7	
Urban teaching	210,542	44.29	21,014	52.32	
Hospital bed size					
Small	70,512	14.83	4,421	11.01	< 0.0001
Medium	132,502	27.87	10,778	26.83	
Large	272,408	57.3	24,966	62.16	
Hospital region					
Northeast	83,151	17.43	5,440	13.48	< 0.0001
Midwest or North Central	100,177	21	7,850	19.45	
South	182,145	38.19	15,535	38.49	
West	111,491	23.38	11,533	28.58	
AHRQ-Elixhauser Index					
< 3	317,136	66.49	37,201	92.18	< 0.0001
≥3	159,828	33.51	3,157	7.82	
Admission Day					
Weekday (Mon-Fri)	347,061	72.77	29,322	72.65	0.8329
Weekend (Sat-Sun)	129,899	27.23	11,037	27.35	

S70

Impact of Frailty on Readmissions and Inpatient Outcomes of Older Adults (≥65 Years) With Acute Pancreatitis

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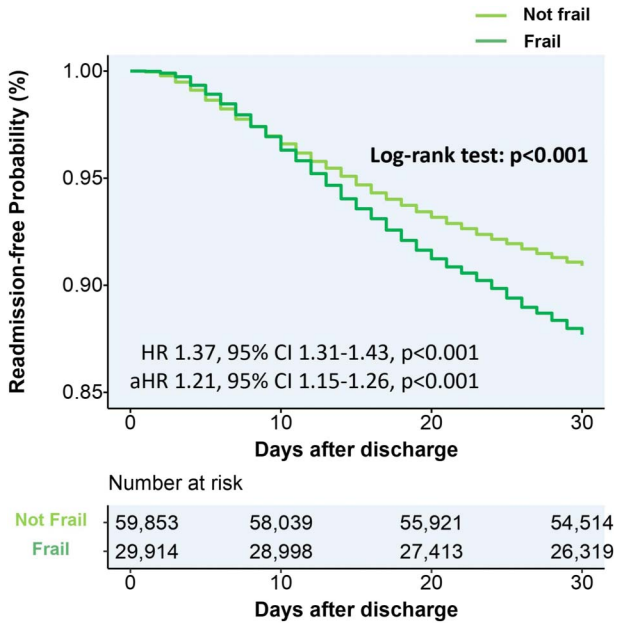
Introduction: Acute pancreatitis (AP) is a major cause of adult hospitalizations in the United States. More than 10 billion US dollars are spent each year on treatment of patients admitted for AP. While data on hospitalizations for AP is widely analyzed, there are limited studies regarding the role that frailty has on this group of patients. The goal of our study is to determine what impact patient frailty has on readmission and on inpatient outcomes.
Methods: The National Readmission Database (NRD) 2017-18 was queried. AP patients age >= 65 were selected based on appropriate ICD-10 diagnostic codes. Hospital frailty risk score (HFRS) was calculated for all the AP patients such that patients with HFRS score < 5 were considered to be not frail (NF) and patients with HFRS score >= 5 were considered frail based on the prevalence of comorbidities in each individual. Differences in mean length of stay (LOS) and in-hospital mortality were analyzed using multivariate logistic regression. Cox proportional-hazards regression analysis was used to determine the difference in 30-day readmission between the two groups.
Results: A total of 91,031 patients aged 65 years and older with AP were identified in our search. Of these, 31,012 (mean age 76.5) were considered frail (average HFRS score 8.2) and the remaining 60,020 (mean age 74.1) were deemed to be not frail (average HFRS score 2.1). The rate of in-patient mortality was significantly higher in the frail group compared to the not frail group (3.53% vs 0.27%) with an adjusted odds ratio of 10.95 ($p < 0.001$). Frail AP patients had a significantly longer LOS when compared to NF (6.8 vs 3.6 days; adjusted mean difference 2.6 days, $p < 0.001$). The difference in 30-day readmission rates between the frail and NF group was also highly statistically significant; (12.0% vs 8.9%, respectively; adjusted hazard ratio 1.2%, 95% CI 1.15-1.26%; $p < 0.001$).
Conclusion: AP patients consume a great deal of healthcare resources and the burden is increasing due to the aging population. Our study finds that the elderly frail group of AP patients is more fragile and has a higher 30-day readmission rate. It would be prudent to risk stratify patients admitted with AP and devise a more customized treatment approach for the more vulnerable frail group, in hopes of reducing inpatient mortality and lessening the burden on our healthcare system that results from longer stays and readmissions. (Figure)



[0070] **Figure 1.** Kaplan-Meier curves of readmission after acute pancreatitis comparing the frail and not frail (NF) groups

S71

The Natural History of Pancreatic Cystic Lesions in Liver Transplant Recipients: A Systematic Review and Meta-Analysis

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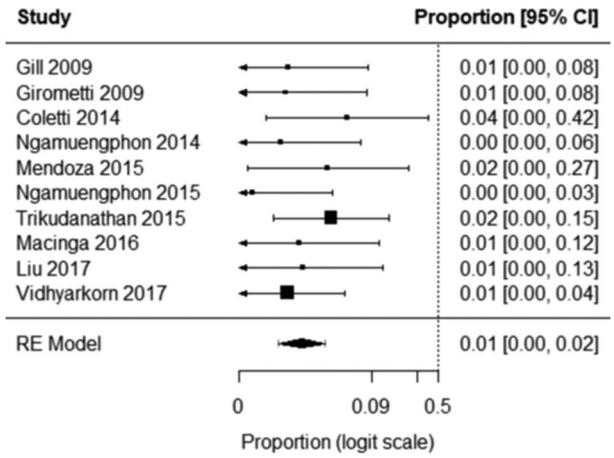
Introduction: Pancreatic cystic lesions (PCLs) exhibit a wide array of clinicopathologic behavior. As such, risk stratifying these is important to prevent the progression to malignancy. Theoretically PCLs in liver transplant (LT) recipients are at increased risk of accelerated carcinogenesis in the setting of lifelong immunosuppressive medications. With improvements in surgical outcomes, LT patients are living longer and understanding the incidence and natural course of these lesions is paramount. As such we conducted a systematic review and meta-analysis to investigate the risk of malignant progression and outcomes of PCLs in LT recipients.
Methods: Multiple databases (PUBMED, Embase, the Cochrane Library) were searched for studies looking at PCLs in post-LT patients from inception until February 2022. Data was extracted to calculate pooled estimates and risk ratios using a random effects model. Primary outcomes were the incidence of PCLs in transplant recipients and progression to malignancy. Secondary outcomes included outcomes of those undergoing surgical resection for progression and change in size over time. There was moderate heterogeneity of our sample.
Results: 12 studies met inclusion criteria (17,862 patients with 1,411 PCLs). The pooled proportion of new PCL development in post-LT patients was 68% (95% CI, 42 – 86, I^2 94%) over a follow up of 3.7±1.5 years. The relative risk of developing PCL in LT recipients compared to non-LT recipients was associated with a significant 67% reduction (RR 0.33; 95% CI, 0.14 – 0.79, I^2 93.91, $p = 0.01$). Among 4158 patients receiving LT, 295 developed PCL and 4 of them progressed to malignancy; the pooled progression to malignancy was 1% (95% CI, 0 – 2, I2 0%) (Figure). LT was associated with a significant 71% reduction in relative risk of undergoing surgical resection (RR 0.29; 95% CI, 0.09 - 0.93, I2 0%, $p = 0.04$).
Conclusion: Compared to non-transplant patients, incidental PCLs in LT patients do not carry a higher risk of malignant transformation. In the setting of immunosuppressive medication, the risk of malignant progression appears to be negligible, and these patients can be followed like non-LT patients based on guidelines. Furthermore, our findings emphasize that incidental PCLs should not preclude a LT evaluation.



[0071] **Figure 1.** Individual estimates and pooled proportions of progression of pancreatic cystic lesions to malignancy in post liver transplant patients

S72

Frailty Is Independently Associated With Higher Mortality and Readmissions in Hospitalized Patients With Acute Biliary Pancreatitis

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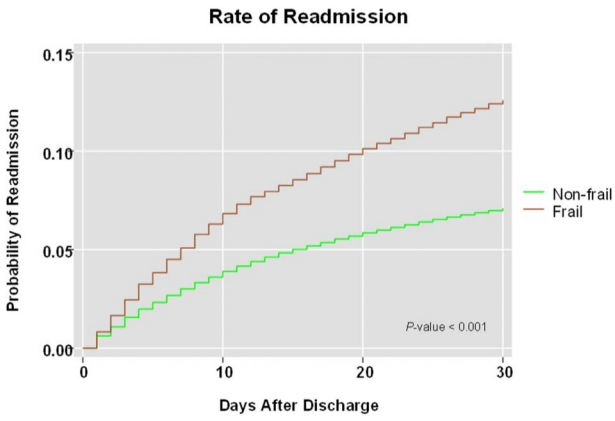
Introduction: Acute pancreatitis is the most common gastrointestinal cause of hospital admissions in the United States of which biliary or gallstone disease is the most common inciting factor. Its costs has been estimated up to $2.6 billion per year. To this end, predictive models aimed at identifying patients at risk for worse outcomes and higher healthcare utilization is important. We conducted a nationally representative cohort study to estimate the independent effects of frailty on burden, costs, and causes for hospitalization in patients with acute biliary pancreatitis.

Methods: We analysed the Healthcare Cost and Utilization Project's (HCUP) Nationwide Readmission Database from 2016 to 2019. Patients admitted with acute biliary pancreatitis were identified; those with concurrent cholangitis were excluded. Patients were categorized into two groups, frail and non-frail, based on Gilbert's Hospital Frailty Risk Score. Categorical variables were described using proportions and compared with Chi-Squared Test. Continuous variables were presented as means and compared using the adjusted Wald test. Logistic and Cox regression models were used to predict the impact of frailty on 30-day readmission, length of stay, mortality, and total costs of hospitalization, respectively.

Results: 162,202 index hospitalizations with acute biliary pancreatitis without cholangitis were identified, of whom 59.2% (n=96,045) were female and 22.49% (n=36,475) were classified as frail. 39.17% (n=14,287) of frail patients were 75 years old or older. Readmissions within 30 days were higher among frail patients (12.58% vs 7.09%, P < 0.001) compared to non-frail patients respectively. Regression modelling showed that frail patients had higher odds of readmission (OR: 1.32; 95% CI 1.24-1.42, P < 0.001), longer lengths of stay (8.18 days vs 4.11 days), and higher average costs of hospitalization ($21,511 vs $12,261) compared to non-frail patients respectively. Cox regression analysis showed that frail patients had a higher risk of mortality (HR 5.43; 95% CI 4.06 – 7.29, P< 0.001) compared to non-frail patients, respectively. Sepsis was the leading cause of readmission in frail patients. (Figure)

Conclusion: Frailty is independently associated with higher mortality and burden of healthcare utilization in patients with acute biliary pancreatitis without cholangitis; sepsis was the leading cause of readmission. We suggest using the Hospital Frailty Risk Score as part of the treatment algorithm in patients with acute biliary pancreatitis. (Table)



[0072] **Figure 1.** Rates of readmission in frail and non-frail patients

Table 1. Study Characteristics

	Gilbert Frailty Risk Category							
	Non-frail		Frail		Total			
	%	CI	%	CI	%	CI	Obs	P-value
Age 75 and over								
Under 75 Years Old	84.45	[84.13-84.77]	60.83	[60.04-61.61]	79.14	[78.81-79.47]	128,367	0.000
75 or Older	15.55	[15.23-15.87]	39.17	[38.39-39.96]	20.86	[20.53-21.19]	33,835	
Total	100.00		100.00		100.00		162,202	

Table 1. (*continued*)

	Gilbert Frailty Risk Category							
	Non-frail		Frail		Total			
	%	CI	%	CI	%	CI	Obs	P-value
Indicator of sex								
Male	39.73	[39.31-40.14]	44.44	[43.67-45.21]	40.79	[40.42-41.16]	66,157	0.000
Female	60.27	[59.86-60.69]	55.56	[54.79-56.33]	59.21	[58.84-59.58]	96,045	
Total	100.00		100.00		100.00		162,202	
Died during hospitalization								
Did not die	99.89	[99.86-99.91]	97.08	[96.80-97.34]	99.26	[99.19-99.32]	160,960	0.000
Died	0.11	[0.09-0.14]	2.92	[2.66-3.20]	0.74	[0.68-0.81]	1,205	
Total	100.00		100.00		100.00		162,166	
Calendar year								
2016	25.44	[24.57-26.33]	24.29	[23.26-25.36]	25.18	[24.35-26.03]	40,847	0.011
2017	25.12	[24.29-25.97]	24.97	[24.00-25.97]	25.09	[24.30-25.89]	40,693	
2018	25.02	[24.20-25.86]	25.07	[24.08-26.09]	25.03	[24.24-25.84]	40,601	
2019	24.42	[23.57-25.29]	25.66	[24.64-26.71]	24.70	[23.88-25.53]	40,061	
Total	100.00		100.00		100.00		162,202	
Insurance Carrier, cleaned								
Medicare	35.77	[35.30-36.24]	66.96	[66.15-67.76]	42.83	[42.38-43.28]	67,222	0.000
Medicaid	18.60	[18.12-19.09]	10.46	[9.97-10.98]	16.76	[16.33-17.19]	26,300	
Private insurance	39.75	[39.23-40.27]	19.76	[19.09-20.45]	35.22	[34.77-35.68]	55,280	
Self-pay	5.89	[5.64-6.15]	2.82	[2.56-3.10]	5.19	[4.98-5.42]	8,148	
Total	100.00		100.00		100.00		156,950	
Median household income								
0-25th percentile	27.23	[26.45-28.03]	27.69	[26.71-28.69]	27.33	[26.58-28.10]	43,754	0.658
26th to 50th percentile (median)	27.76	[27.13-28.40]	27.45	[26.60-28.33]	27.69	[27.09-28.30]	44,323	
51st to 75th percentile	25.54	[24.94-26.15]	25.30	[24.48-26.15]	25.49	[24.91-26.07]	40,799	
76th to 100th percentile	19.47	[18.73-20.23]	19.55	[18.65-20.49]	19.49	[18.76-20.24]	31,196	
Total	100.00		100.00		100.00		160,072	
Bed size of hospital								
Small	19.32	[18.61-20.05]	17.96	[17.06-18.89]	19.02	[18.33-19.73]	30,844	0.001
Medium	28.84	[28.01-29.69]	28.71	[27.70-29.74]	28.81	[28.01-29.63]	46,736	
Large	51.83	[50.86-52.81]	53.33	[52.16-54.50]	52.17	[51.23-53.11]	84,622	
Total	100.00		100.00		100.00		162,202	
Hospital urban-rural designation								
Large metropolitan areas with at least 1 million residents	53.85	[52.62-55.09]	54.10	[52.62-55.58]	53.91	[52.68-55.13]	87,443	0.000
Small metropolitan areas with less than 1 million residents	37.67	[36.45-38.90]	38.66	[37.20-40.13]	37.89	[36.68-39.11]	61,456	
Micropolitan areas	6.53	[6.16-6.92]	5.75	[5.28-6.26]	6.35	[6.00-6.73]	10,308	
Not metropolitan or micropolitan (non-urban residual)	1.95	[1.77-2.15]	1.49	[1.27-1.75]	1.85	[1.68-2.03]	2,996	
Total	100.00		100.00		100.00		162,202	
Teaching status of urban hospitals								
Metropolitan non-teaching	24.78	[24.04-25.54]	24.24	[23.30-25.20]	24.66	[23.94-25.39]	39,993	0.000
Metropolitan teaching	66.74	[65.90-67.58]	68.52	[67.48-69.55]	67.14	[66.33-67.94]	108,906	
Non-metropolitan hospital	8.48	[8.07-8.91]	7.24	[6.72-7.80]	8.20	[7.81-8.61]	13,304	
Total	100.00		100.00		100.00		162,202	
Patient Location: NCHS Urban-Rural Code								
Central counties of metro areas of >=1 million population	25.56	[24.50-26.64]	23.61	[22.46-24.80]	25.12	[24.09-26.17]	40,654	0.000
Fringe counties of metro areas of >=1 million population	25.99	[24.95-27.05]	26.86	[25.63-28.12]	26.18	[25.16-27.23]	42,374	
Counties in metro areas of 250,000-999,999 population	22.51	[21.50-23.56]	22.75	[21.61-23.93]	22.57	[21.57-23.59]	36,522	
Counties in metro areas of 50,000-249,999 population	9.55	[8.97-10.16]	10.37	[9.66-11.12]	9.73	[9.16-10.33]	15,749	
Micropolitan counties	9.12	[8.72-9.53]	9.05	[8.47-9.66]	9.10	[8.72-9.50]	14,731	
Not metropolitan or micropolitan counties	7.28	[6.85-7.74]	7.37	[6.83-7.96]	7.30	[6.89-7.73]	11,817	
Total	100.00		100.00		100.00		161,848	
Admission day is a weekend								
Admitted Monday-Friday	71.95	[71.56-72.33]	72.07	[71.38-72.75]	71.98	[71.65-72.30]	116,746	0.771
Admitted Saturday-Sunday	28.05	[27.67-28.44]	27.93	[27.25-28.62]	28.02	[27.70-28.35]	45,456	
Total	100.00		100.00		100.00		162,202	

S73

Effect of Substance Abuse on Cholangiocarcinoma Outcomes and Screening in a Large Urban Safety-Net Hospital

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Introduction: Patients with substance use disorders are at an increased risk of viral hepatitis and alcohol-related liver disease and, consequently, at higher risk of developing cholangiocarcinoma (CCA). Further, patients with substance abuse are often less compliant with healthcare screening strategies. We performed a retrospective study to evaluate the association of substance use disorders with CCA screening and outcomes.
Methods: A manual chart review was performed for all patients diagnosed with CCA at a large urban safety-net hospital from 2010-2019. Patients were divided into two cohorts, CCA patients with and without a history of substance abuse. Patient characteristics, liver disease etiology, and radiologic screening intervals were recorded. CCA outcomes such as mortality rate, disease metastasis, and treatment decision at tumor board were noted. Statistical analyses were performed using Statistical Product and Service Solutions version 26.9 (SPSS, Inc., Chicago, Illinois). Qualitative and quantitative differences between groups were analyzed by Chi-squared or Fisher's exact tests for categorical variables and t-test for continuous variables.
Results: A total of 46 patients were identified with CCA. Among them, 11 patients (23.9%) had a history of substance abuse, while 35 patients (76.1%) did not have a history of substance abuse. CCA patients with a history of substance abuse were significantly younger (mean age 60 vs. 65) at diagnosis (Table). There were no significant differences in the adequacy of screening in CCA patients with or without a history of substance abuse. While not statistically significant, compared to those without substance abuse, patients with CCA and substance abuse trended toward a higher mortality rate (72.7% vs. 54.3%) and were more likely to be recommended a palliative approach at tumor board (72.7% vs. 57.1%) (Figure).
Conclusion: Notably, those patients with substance abuse were diagnosed with CCA at a significantly younger age. Therefore, cirrhotic patients with substance abuse may benefit from earlier referral to specialist care with hopes of more adherence to screening exams. There is suggestion of worse outcomes in those with substance abuse, as they had higher deaths in follow up and higher likelihood of referral to palliative care upon diagnosis. Overall, this study emphasizes the need for more multidisciplinary linkage to care for this vulnerable patient population.

	CCA Total (n=46)	Substance Abuse (n=11)	No substance abuse (n=35)	P-value
Screening (cross-sectional imaging or US) interval prior to CCA diagnosis				
< 6 months	31 (67.4%)	9 (81.8%)	22 (62.9%)	0.1587
>= 6 months	15 (32.6%)	2 (18.2%)	13 (37.1%)	
Seen by Gastroenterology				
Yes	19 (41.3%)	7 (63.6%)	12 (34.3%)	0.0663
No	27 (58.7%)	4 (36.4%)	23 (73.7%)	
Dead?				
Yes	27 (58.7%)	8 (72.7%)	19 (54.3%)	0.2492
No	10 (21.7%)	2 (18.2%)	7 (20.0%)	
Disease metastasis				
Yes	32 (69.6%)	7 (63.6%)	25 (71.4%)	0.2172
No	12 (26.1%)	4 (36.4%)	8 (22.9%)	
MELD at dx (mean, SD)	10.9, 9.1	11.8, 5.3	10.4, 10.5	0.8377
Treatment decision at tumor board				
Therapeutic[1]	14 (30.4%)	1 (9.1%)	13 (37.1%)	0.0976
Palliative[2]	28 (82.3%)	8 (72.7%)	20 (57.1%)	

[0073] **Figure 1.** Screening adequacy and disease outcome in Cholangiocarcinoma patients with and without substance abuse. Legend: 1Therapeutic treatment options include resection, transplantation, ablation, or Transarterial chemoembolization. 2 Palliative treatment options include chemotherapy and hospice.

Table 1. Baseline characteristics of Cholangiocarcinoma patients with and without substance abuse

	CCA Total (n=46)	Substance Abuse (n=11)	No substance abuse (n=35)	P-value
Age (mean +/- sd)	64.4 +/- 11.1	60.9 +/- 5.4	65.5 +/- 12.2	**0.0278**
Gender				
Male	22 (47.8%)	8 (72.7%)	14 (40.0%)	**0.0485**
Female	24 (52.2%)	3 (27.7%)	21 (60.0%)	
Race				
African Americans	14 (30.4%)	2 (18.2%)	12 (34.3%)	0.2064
White	25 (54.3%)	7 (63.6%)	18 (51.4%)	
Asian	1 (2.2%)	0 (0%)	1 (2.9%)	
Ethnicity				
Hispanic	5 (10.9%)	2 (18.2%)	3 (8.6%)	0.2626
Non-Hispanic	41 (89.1%)	9 (81.8%)	32 (91.4%)	
Insurance				
Medicaid	14 (30.4%)	4 (36.4%)	10 (28.6%)	0.8376
Medicare	15 (32.6%)	4 (36.4%)	11 (31.4%)	
Financial Assistance	1 (2.2%)	0 (0%)	1 (2.9%)	
Private	9 (19.6%)	1 (9.1%)	8 (22.9%)	
No insurance	7 (15.2%)	2 (18.2%)	5 (14.3%)	
Liver disease				
HCV	11 (23.9%)	6 (54.5%)	5 (14.3%)	0.3049
Alcohol	7 (15.2%)	5 (45.5%)	2 (5.7%)	
NASH	3 (6.5%)	0 (0%)	3 (8.6%)	
HCV treatment				
HCV treated	1 (9.1%)	1 (16.7%)	0 (0%)	0.5455
HCV not treated	10 (90.9%)	5 (83.3%)	5 (100%)	

Abbreviations: HCV hepatitis C virus, NASH = Non-alcoholic steatohepatitis

S74

Demographic Analysis of Pancreatic Cancer Incidence Rates by Race and Gender

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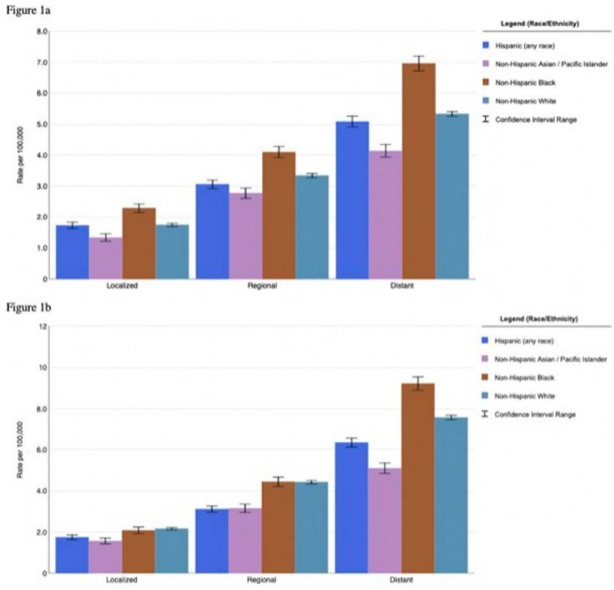
Introduction: Pancreatic cancer (PC) is the third-leading cause of cancer related-deaths in the US as of 2020. In recent years, there has been an increasing focus on the disparities of PC incidence. African Americans have worse overall outcomes compared to other racial groups. The stage of PC at time of diagnosis may be utilized to address this disparity.
Methods: This study analyzed the stage of PC at diagnosis in males by race/ethnicity from 2015-2019 using data extracted from the National Cancer Institute Surveillance, Epidemiology, and End Results Cancer (SEER) Statistics Explorer Network. Age-adjusted incidence rates per 100,000 are categorized into localized, regional, and distant stages of cancer diagnosis and stratified by race/ethnicity.
Results: For female patients, the rate of diagnosis was significantly higher for African Americans (AA) compared to other races across all stages of PC. AA females had higher incidence rates of localized, regional, and distant disease at 2.29%, 4.11%, and 6.96%, respectively [Table, Figure 1a]. In comparison, Asian/Pacific Islander (API) females had the lowest incidence rate at 1.34%, 2.78%, and 4.15%, respectively. The incidence rate per 100,000 for localized and regional PC was not significantly different between AA and Caucasian (CN) males at 2.10 and 2.18, respectively, for localized PC, at 4.44 and 4.45, respectively for regional PC [Figure1b]. Hispanic (HP) and API males had significantly lower incidence compared to AA and CN males at 1.76 and 1.58, respectively, for localized disease, and 3.13 and 3.17, respectively, for regional. Distant PC at time of diagnosis was significantly greater among AA males at 9.23%, compared to rates of 7.59% in CN males, 6.36% in HP males, and 5.11% in API males. All results $p < 0.01$.
Conclusion: The rate of incidence for AA females is greater across all stages of PC, suggesting a genetic or socioeconomic predisposition, although further investigation is necessary. In males with PC, there is a disproportionately greater incidence of stage IV PC. With well-established increased mortality rates among AA, it is important to consider the role of access to healthcare. While multifactorial in etiology, highlighting the racial disparity in stage of PC at diagnosis emphasizes the importance of clinical suspicion. Earlier recognition should be prioritized with interventions to improve outcomes of PC in AA males.



[0074] **Figure 1.** SEER Age-Adjusted Incidence Rates of PC, 2015-2019 by Stage at Diagnosis and Race/Ethnicity, Female (1a) and Male (1b)

Table 1. SEER Age-Adjusted Incidence Rates (with 95% CI) of PC, 2015-2019 By Stage at Diagnosis and Race/Ethnicity, Female and Male

	Localized	Regional	Distant
Female			
Black	2.29 (2.16-2.43)	4.11 (3.93-4.29)	6.96 (6.73-7.20)
White	1.76 (1.71-1.80)	3.35 (3.28-3.41)	5.34 (5.26-5.42)
Hispanic (any race)	1.74 (1.64-1.84)	3.06 (2.93-3.20)	5.09 (4.92-5.27)
Asian/Pacific Islander	1.34 (1.23-1.46)	2.78 (2.61-2.95)	4.15 (3.95-4.36)
Male			
Black	2.10 (1.94-2.26)	4.45 (4.23-4.68)	9.23 (8.91-9.56)
White	2.18 (2.12-2.24)	4.44 (4.36-4.52)	7.59 (7.48-7.70)
Hispanic (any race)	1.76 (1.64-1.88)	3.13 (2.98-3.28)	6.36 (6.14-6.58)
Asian/Pacific Islander	1.58 (1.44-1.73)	3.17 (2.97-3.37)	5.11 (4.86-5.37)

S75

Safety and Efficacy of Endoscopic Drainage of Pancreatic Fluid Collections Performed by Early Career Advanced Endoscopists: A Multicenter Experience

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Introduction: Over the past decade, endoscopic ultrasound (EUS) guided drainage has become the gold standard in management of symptomatic pancreatic fluid collections (PFCs). While there are ample data showing the safety and efficacy of endoscopic drainage of PFCs, these data are largely derived from expert advanced endoscopists. Comparatively, there is little published on outcomes of endoscopic drainage of PFCs performed by early career advanced endoscopists. We aimed to evaluate the safety and efficacy of endoscopic drainage of PFCs performed by early career advanced endoscopists.

Methods: This was a multicenter, retrospective analysis of all patients who underwent EUS-guided drainage of PFCs, performed by 6 early career advanced endoscopists (defined as within 2 years of graduating advanced endoscopy fellowship). Patient and procedure characteristics were recorded. Procedure characteristics included type and location of collection, type and size of stent placed and location of stent placement. Primary outcomes were technical and clinical success. Other outcomes included adverse events and procedure-related mortality.

Results: A total of 24 patients underwent endoscopic drainage of PFCs. The mean age was 53 years and 21% were female [Table]. Median duration of follow-up was 90 days. Three patients (12.5%) were anticoagulated. The average Charlson Comorbidity Index was 2.3. The collections included pseudocysts (37.5%%), walled-off necrosis (45.8%) and post-surgical collections (16.7%). The location was peripancreatic in 10 cases (41.7%) and pancreatic in 14 cases (58.3%). The PFC drainage approach was transgastric in 22 cases (91.7%) and transduodenal in 2 cases (8.3%). The primary drainage modality was lumen-apposing metal stents (LAMS) in 21 cases (87.5%) and plastic stents in 3 cases (12.5%). Clinical success was achieved in 22 cases (91.7%) and technical success in 23 cases (95.8%). The only adverse event was LAMS maldeployment in 1 case (8%), which was immediately removed and the defect closed with an over-the-scope clip without immediate or delayed bleeding or perforation. One patient (8%) had delayed bleeding during subsequent necrosectomy and underwent angioembolization by interventional radiology. There was no procedure-related mortality during the study period.

Conclusion: We found that endoscopic drainage of pancreatic fluid collections is safe and effective in the hands of early career advanced endoscopists, with outcomes comparable to those reported by later career endoscopists.

Table 1. Patient and procedure characteristics

	n = 24
Patient characteristics	
Age, years	53 (SD 14)
Female	5 (21%)
Inpatient	9 (56%)
Platelets (K/cmm)	298
INR	1.13
Any anticoagulation	3 (12.5%)
Argatroban	1 (4.2%)
Apixaban	1 (4.2%)
Warfarin	1 (4.2%)
Charlson Comorbidity Index, average	2.6
Collection Type	
Pseudocyst	9 (37.5%)
Walled-off necrosis	11 (45.8%)
Post-surgical collection	4 (16.7%)
Collection Location	
Peripancreatic	10 (41.7%)
Pancreatic	14 (58.3%)
Head	2
Body/Tail	9
Entire pancreas	3
Procedure characteristics	
Site of EUS Pancreatic Drainage	
Stomach	22 (91.7%)
Duodenum	2 (8.3%)
Native Anatomy	23 (95.8%)
Stent used	
LAMS	21 (87.5%)
Plastic	3 (12.5%)
LAMS Size	
10 mm x 10 mm	2 (9.5%)
15 mm x 10 mm	14 (66.7%)
20 mm x 10 mm	5 (23.8%)
Clinical Success	22 (91.7%)
Technical Success	23 (95.8%)
Adverse events	
LAMS maldeployment	1 (4.2%)
Bleeding	0
Stent migration	0
Post-procedural pain within 30-days	3 (12.5%)
Need for admission within 30-days	7 (29.2%)
Death within 30-days	0
Stent removed	19 (79.2%)
Mean time until stent removed, days, SD	27 (SD 22)
Mean number of GI interventions	1.6
Duration of follow-up median, days	90

S76

Incidence, Co-Morbidities and Outcomes of Necrotising Pancreatitis in a National Inpatient Sample Database

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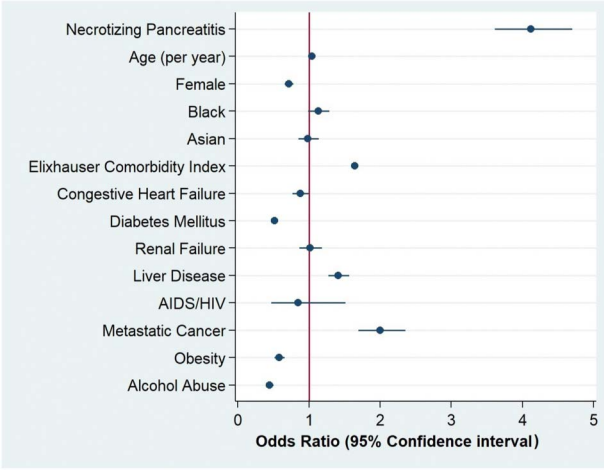
Introduction: Acute pancreatitis (AP) is the most common cause of gastrointestinal hospitalization in the United States and poses a significant financial burden to the healthcare system with estimated cost over 2.2 billion annually. Pancreatic necrosis develops in 10-20% of all patients with AP and is associated with increased complications, longer hospitalization with multiple readmissions and higher mortality. Aim of the study was to determine the financial burden and demographics of patients with necrotising pancreatitis.
Methods: A National representative cohort of AP patients was included between the years 2016-2019 from the National Inpatient Sample (NIS) database. Patients between ages of 18 and 90 with diagnosis AP were included in the study with stratification based on presence of necrotising pancreatitis (NP) via ICD 10 codes. Statistical analysis was done using STATA 17.0. Baseline characteristics and inpatient complications were compared between groups. Outcomes were compared after adjusting for other medical comorbidities using Elixhauser scoring. Secondary outcomes including length of stay, mortality, hospital characteristics and financial measures were compared between groups.
Results: Of the total, 54940 (3.5%) patients (mean age 50.4 ± 17.0 years, 35.9% females, 66.8% whites) were included in the analysis. There were no racial and gender-based differences. Mortality rates were higher among AP with necrosis than without necrosis (3.0% vs. 0.7%). AP with necrosis was independently associated with risk of mortality (OR 4.13, 95% CI 3.63 - 4.72, $p < 0.001$). Alcohol abuse was more common as an etiology of AP with necrosis than others (35.3% vs. 30.5%, $p < 0.001$). The length of stay was longer in necrosis vs. without necrosis (median LOS for AP with necrosis vs. without necrosis; 6 days vs. 3 days, $p < 0.001$). The cost for hospitalization was higher for NP vs. AP (median cost for AP with necrosis vs. without necrosis; $53737 vs $28531, $p < 0.001$). There was also a trend in the increase in the diagnosis of AP with necrosis from 2016 to 2019. Risk factors for mortality are shown in the Figure:
Conclusion: NP has significantly higher morbidity and mortality in individuals with AP. NP is also associated with high hospital charges and non-elective admissions.



[0076] **Figure 1.** Baseline Characteristics for Necrotising pancreatitis

S77

Dynamic Pancreatic Parenchymal Changes in the Setting of Fatty Pancreas: A Short-Term Follow-Up Study

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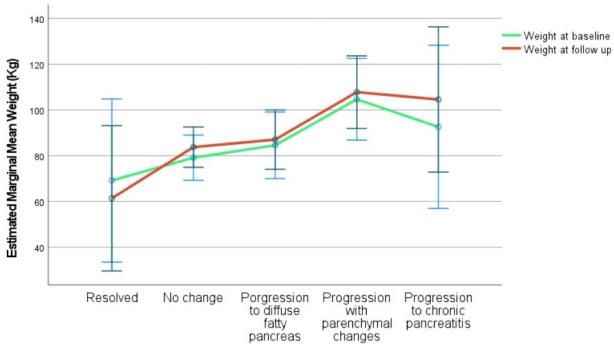
Introduction: Clinical significance and long-term impacts of Fatty pancreas (FP) on pancreatic parenchyma are not well-recognized. The aim of this study is to assess parenchymal alterations over time in patients with FP.
Methods: This is a retrospective study (2014-2021) of patients with diffuse echogenicity of the pancreas, suggestive of FP, on endoscopic ultrasound (EUS). Subjects with subsequent EUS, Magnetic Resonance Imaging (MRI), or Computed Tomography (CT) scan at least two years after the initial EUS were included. Incidence of parenchymal changes and development of chronic pancreatitis (CP) overtime were recorded.
Results: A total of 39 patients with a mean age of 51.24 ± 12.31 years were enrolled. Mean initial weight was 80.17±17.75kg. Diabetes mellitus (DM), fatty liver, and exocrine pancreatic insufficiency (EPI) were present in 15%, 46% and 33% of the patients at baseline, respectively. Patients were followed by EUS (n=25), CT scan (n=9), and MRI (n=5) over an average follow up period of 2.38 ± 0.94 years. In 25 patients with available follow up EUS, 16% (n=4) progressed to CP and 24% (n=6) had additional parenchymal changes without meeting the criteria for CP. Only one patient from the latter group developed new onset DM during the follow up period. No major parenchymal changes were noted in 52% (n=13). Of the two remaining patients, one had progressed to diffuse echogenicity of the entire pancreas rather than the body alone, while the other patient was noted to have resolution of FP with minimal hyperechoic strands after weight loss. Average weight was statistically higher at baseline and follow-up in patients with progressive parenchymal changes (92.6 ± 5.2 kg[baseline] and 96.2± 6.09 kg [follow-up]) in comparison to those with parenchymal appearance (78.43 ± 4.6 kg [baseline] and 82.17 ± 4.4 kg [follow-up]); p-value 0.032. In multivariate analysis, progressive parenchymal changes on EUS were associated with an increase in weight over time, independent of the effects of gender, alcohol, or tobacco (p-value = 0.04). (Table) (Figure)
Conclusion: Progressive parenchymal changes was noted in 44%. FP is a potential precursor for chronic pancreatitis and further parenchymal changes. Weight gain may be an independent contributor to the development of further parenchymal changes in patients with FP. Our results suggest that FP is a dynamic process with the possibility of progression or regression over time.



[0077] **Figure 1.** Trend of weight overtime according to parenchymal changes

Table 1.

Characteristics	Total	With progressive changes (n=11)	Without progressive changes (n=14)	p-value
Age (Mean ± SD) years	51.21 ± 12.34	44.7 ± 13.68	54.67 ± 10.6	0.99
Gender (female)	64% (n=25)	72.7% (n=8)	64.3% (n=9)	1.00
Race (White)	97%			
Ethnicity (Non-Hispanic Latino)	74.4%			
History of alcohol use	51.3% (n=20)**			
Tobacco use				0.42
Never smoker		63.6% (n=7)	57.1% (n=8)	
Current smoker	12.8% (n=5)***	36.4% (n=4)	28.6% (n=4)	
Former smoker	28.2% (n=11)	0%	14.3% (n=2)	
Baseline weight (Kg)	80.17±17.75	92.6 ± 5.2	78.43 ± 4.6	0.032
		96.2± 6.09	82.17 ± 4.4	
Baseline BMI (kg/m^2)	29.59 ± 6.45			
Location of Fatty Infiltration				
Diffuse	92.3% (n=36)			
Body and tail	7.7% (n=3)			
Parenchymal Changes at baseline				
Hyperechoic strands	64.1% (n=25)			
Hyperechoic foci	38.5% (n=15)			
Visible side branches	17.9% (n=7)			
Dilated pancreatic duct	15.4% (n=6)			
Lobularity	10.3% (n=4)			
Atrophy	5.1% (n=2)			
Cyst	5.1% (n=2)			
Irregular duct wall	2.6% (n=1)			
Hyperechoic duct wall	2.6% (n=1)			
Fatty liver	46.15% (n=18)			
Baseline		72.7% (n=8)	28.6% (n=4)	0.047
Follow up		72.7% (n=8)	21.4% (n=3)	0.015
Diabetes mellitus	15.38% (n=6)			
Baseline		36.4% (n=4)	0%	0.026
Follow up		45.5% (n=5)	0%	0.009
Exocrine pancreatic insufficiency	33.33% (n=13)	71.4% (n=5 out 7 with available results)	71.4% (n=5 out 7 with available results)	

**Of the nineteen patients without history of alcohol use at the time on index EUS, three patients reported remote history of ethanol consumption.
***All five patients successfully quit tobacco consumption by the time of the follow up imaging modality.

Outcomes of Acute Pancreatitis in Pregnancy: A National Study

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Introduction: Acute pancreatitis (AP) during pregnancy is a rare but severe disease with a high maternal fetal mortality. The most common causes for AP in pregnancy are gallstone pancreatitis which can be attributed to hormonal changes in pregnancy and familial hypertriglyceridemia. Management strategy for gallstone pancreatitis in pregnancy is with laparoscopic cholecystectomy in the second trimester. However, management of AP of any other etiology is conservative like in non-pregnant individuals.

Methods: De-identified patient data was used in this study using the National Inpatient Sample (NIS) database. The data covered the years 2002-2016 and ICD-9 and ICD-10 codes were used for identification of the different variables being studies and the study population which was stratified in two groups: pregnants with AP and the control group. The data was analyzed using SPSS 10.
Results: A total of 7,886,986 pregnant women in the NIS database were studied with 3,295 of this study population were diagnosed with AP during pregnancy. 48.9% of AP occurred in the third trimester with 40.4% occurring during 30-39 weeks of gestation, 23.4% occurred in the first trimester, 27.8% in the second trimester. More cases occurred in White race than in African American and Hispanic races (37.5% vs 17.8% vs 35.1% respectively, P< 0.001). Fetal mortality with AP was 79.5% compared to 6.4% in the control group (P< 0.001). Spontaneous abortion was noted to be higher with AP compared to controls (0.5% vs 0.2%, P =0.009). Comorbidities that were associated with AP to a greater extent than in controls were overweight/obesity (14.9% vs 8.1%, P< 0.001) and hypertension (1.2% vs 0.2%, P < 0.001). (Table)
Conclusion: AP in pregnancy is uncommon with an incidence of 0.082%. However, it is a serious disease that is associated with higher incidence of spontaneous abortion (0.5%) and higher fetal mortality (79.5%) as well as a higher rate of pregnancy complications such as hypertension. AP presents mainly during the third trimester of pregnancy and was found to be more common in women of the white race. It was also noted that obesity and overweight were significantly associated with AP during pregnancy which can contribute to the formation of cholelithiasis which is known to be one of the most common causes for AP in pregnancy along with familial hypertriglyceridemia.

Table 1. Comparison of fetal and maternal outcomes between pregnant females with and without acute pancreatitis

	Pregnant with Acute Pancreatitis (N=3,295)	Control group (N=7,867,126)	P-value
Race			< 0.001
White	37.5%	51.9%	
African American	17.8%	15.6%	
Hispanic	35.1%	20.8%	
Others	9.6%	11.7%	
Trimester	23.4%	1.2%	< 0.001
First	27.8%	2.8%	
Second	48.9%	96.0%	
Third			
Liveborn	20.5%	0.1%	< 0.001
Spontaneous abortion	0.5%	0.2%	0.009
Hypertension	0.2%	0.2%	< 0 .001
Obesity	14.9%	8.1%	< 0 .001
Eclampsia	0.3%	0.1%	0.062

S79

EUS-Guided FNA Cytology and EUS-Guided FNB Histology Comparison for Solid Pancreatic Lesions: Single Tertiary Care Center Retrospective Study

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Introduction: Endoscopic Ultrasound (EUS)-guided fine needle aspiration (FNA) and fine needle biopsy (FNB) are the current standard of care for making a diagnosis in solid pancreatic lesions (SPLs). There is conflicting data on the diagnostic utility of cytology and histology for diagnosing SPLs. The aim of this study was to evaluate the diagnostic utility of cytology and histology for diagnosing SPLs.
Methods: A retrospective chart review was performed and all patients greater than 18 years with solid pancreatic lesions who underwent EUS between October 2019 and June 2021 were included. All the procedures were performed by a single advanced endoscopist and same needle (Acquire 22G, Boston Scientific) was used for all the procedures. Primary outcomes assessed were diagnostic advantage of cytology and histology in terms of providing a final diagnosis and diagnostic accuracy of cytology and histology with surgery specimen being the gold standard. Secondary outcomes assessed were relation of diagnostic accuracy to specimen length, no. of passes and size of lesion using t-test.
Results: A total of 59 patients were included in our study with majority of them being males (52.4%). Mean needle passes per patient were 4.25 and average size of lesion on EUS was 3.7 cm. Turnaround time of histology was superior (2.7 days) as compared to cytology (3.3 days). In 39 cases, there was agreement between histology and cytology and 36/39 (92%) of those were malignant. Of the remaining 20 patients, histology was found to be more advantageous for 17 (85%) patients while cytology was more advantageous for the other 3 patients (15%). All 3 cases where cytology was advantageous were malignant. Of the 17 histologically advantageous cases, 15 (88%) were malignant. The histological diagnosis and cytological diagnosis were accurate in 7/8 (88%) and 3/8 (38%) respectively. T-test did not show any significant difference between two groups in relation to diagnostic accuracy to specimen length, no. of passes and size of lesion using t-test.
Conclusion: Histology provided more accurate diagnosis for discordant cases and had faster turnaround times. However, there were malignant cases diagnosed on cytology which could have been missed if only histology was performed. Histology and cytology are important in conjunction with each other for maximizing the diagnostic accuracy for SPLs.

S80

Safety of Cholangioscopy Using Spyglass in Individuals With Primary Sclerosing Cholangitis

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Introduction: Primary sclerosing cholangitis (PSC) is associated with hepatobiliary complications including increased risk of cholangiocarcinoma and bile duct stones that require endoscopic therapy. Use of per-oral-cholangioscopy (POC) during ERCP can provide direct intraductal visualization and aid in diagnosis and treatment of PSC related biliary complications. However, there is little data regarding safety of this device in PSC patients.
Methods: We aimed to compare rates of complications such as infection, bleeding, pancreatitis, perforation and thirty-day mortality following ERCP with POC compared to ERCP without POC. Approval for the study obtained from Henry Ford Hospital institutional review board. Patients who were diagnosed with PSC and underwent ERCP with or without POC between 2013 and 2021 were identified using relevant diagnostic and billing codes from ProVation software. Each chart was then individually reviewed to gather data about demographics, procedural details and rate of complications.
Results: Twenty patients underwent ERCP with POC during this time periods and were compared to sixty-one patients who underwent ERCP without POC. There was no major difference in age and gender between the two groups. Similar proportion of individuals had cirrhosis and cholangiocarcinoma between the two groups. Main ERCP findings were dominant stricture in 65% of POC group compared to 55.7% without POC, stone disease in 30% POC group compared to 18% without POC, and cholangitis in 5% POC group compared to 9.8% in the other group. Overall complication rate was 10% in the POC group compared to 13.1% in the group without POC. In the POC group, one patient (5.0%) had perforation during the procedure, and another had post procedural bacteremia (5.0%). In the standard ERCP group, there were 7 (11.5%) patients who had infections, 2 (3.3%) had bleeding episodes and 1 (1.6%) patient had pancreatitis post procedure. There was no patient death within thirty days of the procedure in either group. Median total procedure time was longer in the POC group at 90 minutes, compared to 70 minutes for standard ERCP group. Total fluoroscopy time was similar between the two groups; 10.4 minutes in the POC group compared to 11.5 minutes in the standard ERCP group.
Conclusion: ERCP with cholangioscopy has a similar rate of complications compared to ERCP without cholangioscopy in patients with PSC.

Table 1. Comparison of ERCP with cholangioscopy versus standard ERCP without cholangioscopy groups with regards to demographics, procedural details and complications

	ERCP + Cholangioscopy	ERCP	P value
Age, Median (IQR)	50.5 (38.5-57.0)	42 (33.0-57.0)	0.38
Gender, Female, N (%)	6 (30.0)	29 (47.5)	0.17
Underlying Cirrhosis	10 (50.0)	33 (54.1)	0.75
Cholangiocarcinoma	2 (10.0)	4 (6.6)	0.63
ERCP Findings			
Stone	6 (30.0)	11 (18.0)	0.25
Dominant Stricture	12 (65.0)	34 (55.7)	0.47
Cholangitis	1 (5.0)	6 (9.8)	0.68
Diagnostic Modality			
Brush	13 (65.0)	35 (57.4)	
Forceps	2 (10.0)	4 (6.6)	
Spybites	8 (40.0)	0 (0)	
Complications	2 (10.0)	8 (13.1)	0.67
Infection/Cholangitis	1 (5.0)	7 (11.5)	
Pancreatitis	0 (0)	1 (1.6)	
Bleeding	0	2 (3.3)	
Perforation	1 (5.0)	0 (0)	
Total Procedure Time (mins), Median (IQR)	90.0 (50 – 99)	70.0 (48 – 81)	0.22
Flouroscopy Time (mins), Median (IQR)	10.4 (6.7 – 14.2)	11.5 (7.0 – 18.4)	0.59

ERCP: Endoscopic retrograde cholangiopancreatography, IQR: Inter-quartile range, mins: minutes.

S81

Veterans With Pancreatic Cysts, on the Road to Cancer? A 22-Year Retrospective Analysis

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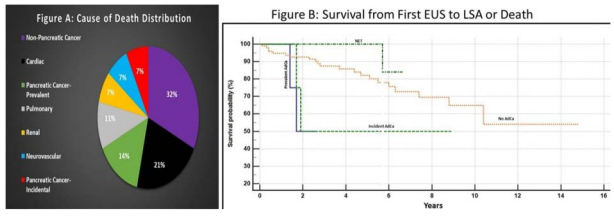
Introduction: Increased use of CT and MRI has increased detection rates of pancreatic cysts up to 15%.. Currently there is a lack of agreement on how best to survey incidental pancreatic cysts. We aim to evaluate the natural history of pancreatic cyst amongst veterans with the goal of ultimately reducing the risk of pancreatic cancer. This is one of the first analyses of the natural history of pancreatic cyst exploring the long term data, EUS findings, and surgical versus non-surgical outcomes.
Methods: A retrospective chart review from 2000 - 2022 is being conducted on Hines VA Veterans with pancreatic cyst utilizing Computerized Patient Record System. CPT codes retrieved EUS and ERCP data. Veterans not diagnosed with incidental pancreatic cyst were excluded from the study. SPSS software used to conduct Chi-square analysis, ANOVA test and log rank test to compare survival curves amongst different causes of death. Prevalent cancer is presence of cancer at the time of cyst diagnosis.
Results: This is an ongoing study of 1905 veterans that underwent EUS, the total number with pancreatic cysts is yet to be determined. Preliminary analysis and demographics of initial charts are presented in Table: The causes of mortality for veterans with pancreatic cyst are shown in the pie diagram in Figure 1A. Important to note that only 3% of veterans with pancreatic cysts developed incidental pancreatic cancer and only 1.5% died of pancreatic cancer. Kaplan-Myer Survival Curve shown in Figure 1B of 113 patients.
Conclusion: Our study population is geriatric with a high comorbidity index, high rates of smoking, alcoholism, and high rates of non-pancreatic cancers. Despite these poor prognostic factors, our preliminary data of incidental cancer developing from cysts was 3%. Non-pancreatic cancer and cardiovascular causes are seen as the two most common causes of death. Most incidental cancers were detected within two years of cyst presentation, reinforcing the need for initial surveillance. Since no cumulative increase of pancreatic cancers were observed long-term, surveillance may need to be adjusted accordingly. The findings of this study could lead to guideline enhancement, cost-effective surveillance and surgical resection strategies for pancreatic cysts. As it is an ongoing study additional charts were reviewed and demographics, patient results and causes of mortality pie diagrams were generated on 133 patients. Complete results and analysis will be presented in October 2022.



[0081] **Figure 1.** Cause of Death Distribution; Figure 1B: Survival from First EUS to LSA or Death

Table 1. Patient Demographics and Results

Gender Distribution	100% Male
Age Range	35 years to 89 years
Average Age at Diagnosis	68
Average Age at Death	76
Smoking Rates	58%
Alcohol Rates	55%
Percentage of Cysts that developed into Incidental Pancreatic Cancer	3.03%
Percentage of Cysts that led to Death from Incidental Pancreatic Cancer	1.52%

S82

Antibiotics Use in Acute Pancreatitis: An Ongoing Problem

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Introduction: Antibiotics use in acute pancreatitis remains a controversial practice. Antibiotic use in acute pancreatitis is indicated in cases of infected pancreatic necrosis, ideally after a culture is obtained. In clinical practice however, antibiotic use is common even in cases without obvious or documented infection.
Methods: This retrospective cohort study included adult patients diagnosed with acute pancreatitis in our hospital between 01/2016- 12/2019. Pancreatitis was confirmed by using the revised Atlanta criteria. Antibiotic use, pancreatitis type (IEP: Idiopathic Edematous pancreatitis or NP: Necrotizing Pancreatitis), etiology, concerns for pancreatic or extra-pancreatic infection and length of stay (LoS) were also collected. Statistical analysis performed by Minitab.
Results: 810 patients were included. 727 (90%) and 83 (10%) had IEP and NP respectively. The most common etiologies were alcohol (25%) and gallstones (28%). 324 (40%) patients received antibiotics. When cases with extra-pancreatic infection concerns were excluded, antibiotics were used in 25% of patients. When cases with any infection concerns were excluded, the antibiotic utilization rate was 19%. NP cases were more likely to receive antibiotics. When patients with any infection concerns were excluded, the administration rate was 62% while in IEP cases was 17%($p< 0.001$). Some etiologies were more likely to get antibiotics. Antibiotics were used in 50% of ERCP-induced pancreatitis and 39% of gallstone-induced pancreatitis. NP cases had a significant LoS increase relative to IEP of 5.5 days. An LoS increase of 3 and 6 days was noted with extra-pancreatic and pancreatic infection concerns respectively. The etiology did not affect LoS. The median LoS, with no antibiotic use was 4 days while with antibiotic use, was 6 days. The LoS was significantly shorter in those who received no antibiotics, even when cases with infection concerns were excluded. (Table)
Conclusion: Despite clear guidelines, antibiotics are inappropriately used in acute pancreatitis management. Our study indicated antibiotic use in up to 40% of acute pancreatitis cases, while in 19% no clear indication was observed. Antibiotic use was much more common in NP cases. LoS was significantly longer when antibiotics were used, even when no infection suspected. To improve this problem, an acute pancreatitis order set will be included in our hospital's electronic health records and the antibiotic utilization rate will be reassessed.

Table 1. **Antibiotics & Length of stay in days (LoS) in patients without infection concerns**

Antibiotics	N	Mean LoS	Median LoS
No	469	4.7	4
Yes	119	8.1	6

S83

Fully Covered vs Uncovered Self-Expanding Metal Stents for the Treatment of Distal Malignant Biliary Obstruction in the Setting of Locally Advanced Pancreatic Cancer

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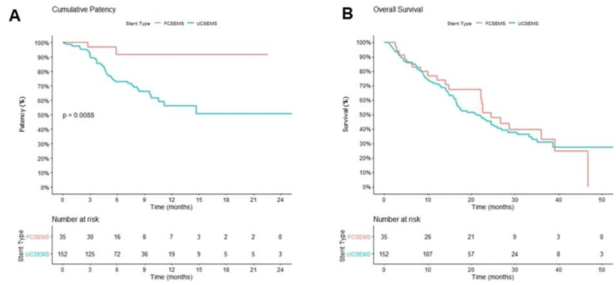
Introduction: Self-expanding metal stents (SEMS) are preferred for the treatment of distal biliary obstruction (dMBO) caused by pancreatic cancer. Optimizing biliary drainage in the neoadjuvant period while patients await definitive surgical treatment is essential. The aim of this study is to compare clinical outcomes of patients who underwent fully covered (FCSEMS) versus uncovered (UCSEMS) placement for the treatment of dMBO due to locally advanced pancreatic cancer (LAPC).
Methods: Consecutive patients who underwent biliary SEMS placement for treatment of dMBO in the setting of LAPC were identified from a single, tertiary care center between May 2017 and May 2021. Patients were categorized into FCSEMS or UCSEMS cohorts based on the type of stent placed during index ERCP. UCSEMS included WallFlex (Boston Scientific,Boston,MA) and Flexxus (ConMed,Utica,NY). Primary outcomes were clinical success, overall incidence of adverse events (AEs), and need for unplanned endoscopic reintervention. Secondary outcomes included stent patency, type of AEs, and overall survival.
Results: 817 cases of ERCP with SEMS placement were reviewed to identify 187 patients with dMBO due to LAPC. 35 (18.7%) patients underwent FCSEMS placement and 152 (81.3%) UCSEMS (49 WallFlex, 103 Flexxus). High rates of clinical success were seen in both cohorts (92.1% UCSEMS vs. 97.1% FCSEMS, $p = 0.29$). (Table) Stent patency was significantly longer in patients who underwent FCSEMS placement (Figure 1A). There was no difference in rates of occlusion or time to occlusion between the two UCSEMS models. AE rates were significantly higher after UCSEMS placement (32.9% vs. 14.3%, $p = 0.03$) as was need for unplanned reintervention (29.0% vs. 5.7%, $p = 0.004$), largely driven by SEMS occlusion (28.9% vs. 2.9%, $P = 0.001$) in the setting of tissue ingrowth (42/44 cases). A similar number of patients underwent surgical resection (UCSEMS 55.9% vs. FCSEMS 65.7%, $p = 0.29$) at similar interval after stent placement (UCSEMS 7.0 vs. FCSEMS 6.0 months, $p=0.57$). All-cause mortality and duration of survival was not different between the two groups (Figure 1B).
Conclusion: FCSEMS were associated with longer patency times and fewer interventions in a cohort of patients with LAPC that otherwise exhibited similar survival and rates of surgical intervention. Additional studies are needed to evaluate cost implications, but this data suggests placement of a FCSEMS may be preferred to UCSEMS in the palliation of dMBO in anatomically amenable patients with LAPC.



[0083] **Figure 1.** Cumulative patency time. Stent patency was defined as the interval between the time of stent insertion and the time of biliary reintervention because of stent dysfunction or the end of the follow-up. Patients without stent obstruction were censored at the time of surgery, last follow-up or death. 1B: Overall survival

Table 1. **Clinical outcomes of patients who underwent metal biliary stent placement for the management of locally advanced pancreatic cancer**

	UCSEMS n=152	FCSEMS n=35	P Value
Clinical Success	140 (92.1)	34 (97.1)	0.29
Adverse Events	50 (32.9)	5 (14.3)	**0.03**
Stent Occlusion	44 (28.9)	1 (2.9)	**0.001**
PEP	8 (5.3)	2 (5.7)	1.00
Stent Migration	3 (2.0)	2 (5.7)	0.24

Table 1. (*continued*)

	UCSEMS n=152	FCSEMS n=35	P Value
Cholecystitis	1 (0.7)	0 (0.0)	1.00
Unplanned reintervention	44 (29.0)	2 (5.7)	**0.004**
Median time to unplanned reintervention (months)	4.4 (3.0-5.6)	4.3 (3.6-5.1)	0.81
Median time to stent occlusion (months)	4.4 (3.0-5.6)	5.9 (5.9-5.9)	0.73
Median stent dwell time (months)	7.0 (5.0-8.7)	6.0 (3.5-8.5)	0.16
Median follow up time (months)	15.6 (8.8-24.6)	22.4 (8.3-27.9)	0.23
Surgical resection	85 (55.9)	23 (65.7)	0.29
Median time to surgical resection (months)	7.0 (5.0-8.9)	6.0 (4.4-8.2)	0.57
Death	84 (55.3)	22 (62.9)	0.41

Values are presented as median (IQR) or n (%). UCSEMS uncovered self-expandable metal stent FCSEMS fully covered self-expandable metal stent PEP post-ERCP pancreatitis.

S84

Chronic Pancreatitis and Risk of Atherosclerotic Cardiovascular Disease (ASCVD): A U.S. Cohort Propensity Matched Study

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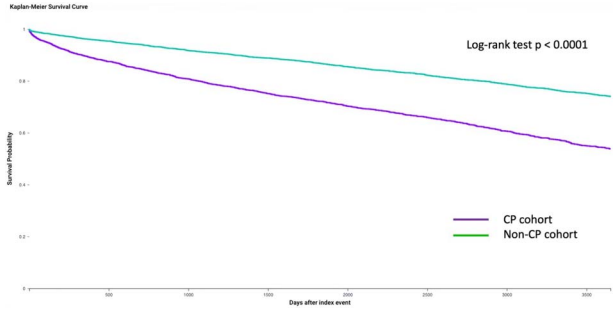
Introduction: The worldwide prevalence of chronic pancreatitis (CP) has continued to rise in recent years, with data suggesting increased risk of atherosclerotic cardiovascular disease (ASCVD) in these patients. The primary aim of our study was to assess the risk of ASCVD and outcomes of ischemic heart disease (IHD) in patients with CP.
Methods: We compared the risk of ischemic heart disease (IHD), cerebrovascular accident (CVA) and peripheral arterial disease (PAD) between CP and non-CP cohort after propensity matching of known risk factors of ASCVD using TriNetX, a multi-institutional database. We also evaluated the risk of outcomes of IHD including acute coronary syndrome (ACS), heart failure (HF), cardiac arrest and all-cause mortality between CP and non-CP cohort. Sub-group analysis was performed based on use of aspirin (ASA) or statin and severity of diabetes mellitus (DM) and chronic pancreatitis prior to the development of IHD.
Results: A total of 28,290 patients (mean age 65.7 +/- 17.3) were in the CP cohort. Cumulative incidence of IHD, CVA and PAD was 25.7%, 6.2% and 6.2% respectively. CP cohort was also found to have an increased risk for IHD (adjusted OR [aOR] 1.08, 95% CI 1.03 – 1.12), CVA (aOR 1.12, 1.05 – 1.20) and PAD (aOR 1.17, 1.1 – 1.24) compared to non-CP cohort after propensity-score matching. CP cohort with IHD was also found to have an increased risk for ACS (aOR 1.16, 1.04 – 1.30), cardiac arrest (aOR 1.24, 1.01 – 1.53) and mortality (aOR 1.60, 1.45 – 1.77) compared to non-CP cohort. Kaplan-Meier analysis revealed that patients in the CP cohort with IHD had poor survival probability in terms of all-cause mortality (log-rank test $p < 0.0001$) compared to non-CP cohort with IHD. CP cohort were still at an increased risk for mortality in spite of ASA (aOR 1.74, 1.50 – 2.01) or statin (aOR 1.47, 1.14 – 1.89) use before IHD. Severe CP cohort were at an increased risk for mortality (aOR 1.55, 1.18 – 2.04) compared to non-severe CP cohort. (Figure) (Table)
Conclusion: CP patients are at a higher risk for ASCVD when compared to the general population, matched for confounding etiological, pharmacological and comorbid variables. Routine healthcare maintenance should focus on cardiovascular risk assessment as well as timely diagnostic and therapeutic interventions.



[0084] **Figure 1.** Kaplan-Meier survival curves for all-cause mortality between CP and non-CP cohorts with IHD.

Table 1. **Outcomes of IHD between CP and non-CP cohort based on different medications prior to the development of IHD**

Medication	Outcome	Cohort	N (%)	OR (95% CI)
Aspirin	ACS	CP	455 (20.4)	1.31 (1.12 – 1.53)
		Non-CP	364 (16.3)	
	HF	CP	766 (34.4)	1.01 (0.89 – 1.15)
		Non-CP	757 (34.0)	
	Cardiac arrest	CP	92 (4.1)	1.45 (1.05 – 2.01)
		Non-CP	64 (2.8)	
	Mortality	CP	578 (26.0)	1.74 (1.50 – 2.01)
		Non-CP	373 (16.7)	
Statin	ACS	CP	158 (20)	1.24 (0.96 – 1.60)
		Non-CP	132 (16.7)	
	HF	CP	264 (33.4)	1.07 (0.86 – 1.32)
		Non-CP	252 (31.8)	
	Cardiac arrest	CP	30 (3.7)	1.07 (0.63 – 1.81)
		Non-CP	28 (3.5)	

Table 1. (*continued*)

Medication	Outcome	Cohort	N (%)	OR (95% CI)
	Mortality	CP	178 (22.5)	1.47 (1.14 – 1.89)
		Non-CP	130 (16.4)	
Insulin	ACS	CP	1094 (19.2)	0.99 (0.90 – 1.09)
		Non-CP	1097 (19.3)	
	HF	CP	1893 (33.3)	0.83 (0.77 – 0.89)
		Non-CP	2133 (37.5)	
	Cardiac arrest	CP	310 (5.4)	1.23 (1.04 – 1.46)
		Non-CP	254 (4.4)	
	Mortality	CP	1547 (27.2)	1.40 (1.29 – 1.53)
		Non-CP	1194 (21.0)	

S85

Early vs Delayed Minimally Invasive Intervention for Infected Pancreatic Necrosis – A Systematic Review and Meta-Analysis

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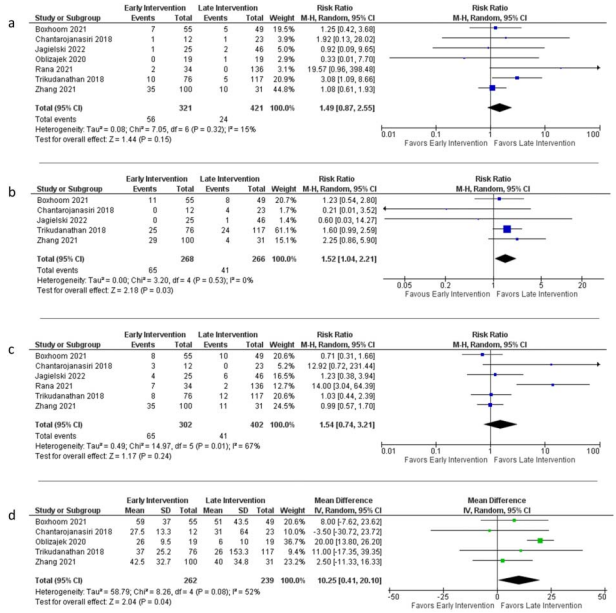
Introduction: Pancreatic necrosis complicates about 20% of acute pancreatitis cases, and 30-40% of those become infected. Current guidelines recommend that invasive intervention for pancreatic necrosis should be delayed to 4 or more weeks from disease onset. However, recent studies have challenged the optimal timing of intervention, especially with the advent of minimally invasive interventions.
Methods: We conducted a systematic review and meta-analysis of studies that early and delayed minimally invasive intervention for infected pancreatic necrosis. We performed a comprehensive search in the databases of PubMed/MEDLINE, Embase, and the Cochrane Central Register of Controlled Trials from inception through April 11, 2022. We considered randomized controlled trials, cohort studies, case-control studies, and case series. We excluded abstracts, animal studies, case reports, reviews, editorials, and letters to editors. From each study, we collected the number of patients who underwent early and late interventions for infected pancreatic necrosis. Early intervention was defined as an intervention that was carried out within 4 weeks of acute pancreatitis onset, while delayed intervention was carried out after 4 weeks of acute pancreatitis onset. Outcomes were mortality, gastrointestinal fistula or perforation, bleeding, and length of hospital stay. The random-effects model was used to calculate the risk ratios (RR), mean differences (MD), and confidence intervals (CI). A p value < 0.05 was considered statistically significant. Heterogeneity was assessed using the Higgins I^2 index. (Figure)
Results: Seven studies involving 742 patients were included in the meta-analysis. Timing of intervention had no statistically significant effect on mortality (RR 1.49, 95% CI 0.87 – 2.55, p = 0.15, I2 = 15%) or bleeding (RR 1.54, 95% CI 0.74 – 3.21, p = 0.24, I2 = 67%). However, early intervention was associated with a statistically significant higher risk of gastrointestinal fistula or perforation (RR 1.52, 95% CI 1.04 – 2.21, p = 0.03, I2 = 0%) and a longer hospital length of stay (MD 10.25 days, 95% CI 0.41 – 20.10, p = 0.04, I2 = 52%).
Conclusion: Our meta-analysis demonstrated that the timing of intervention had no effect on mortality or bleeding in infected pancreatic necrosis. Early intervention resulted in a higher risk of gastrointestinal fistula or perforation and a longer length of hospital stay. Further randomized controlled trials are needed to confirm our findings.



[0085] **Figure 1.** Forest plots comparing early and late intervention in terms of (a) mortality, (b) gastrointestinal fistula or perforation, (c) bleeding, and (d) length of hospital stay

S86

Acute Pancreatitis Is Associated With Increased Risk of In-Hospital Mortality and Healthcare Utilization Among Patients With Hematopoietic Stem Cell Transplantation

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Introduction: Acute pancreatitis (AP) carries a significantly increased morbidity and mortality worldwide. AP is a potential complication of hematopoietic stem cell transplantation (HSCT) although its incidence remains unclear. HSCT recipients are at increased risk of AP due to various factors but the effect of AP on mortality and resource utilization in the adult population has not been studied. We investigated the impact of AP on hospitalization outcomes among patients following HSCT.

Methods: We queried the National Inpatient Sample (NIS) database using the ICD-10 codes. All adult patients with a diagnosis or procedure code of HSCT were included in the study. Patients were divided into those with a diagnosis of AP and those without. Sensitivity analysis was performed for patients with length of stay greater than 28 days. The relationship between AP and mortality, length of stay, total hospitalization cost and charge was assessed using multivariate analysis. Adjustments were made for patient demographics, hospital characteristics, etiology of pancreatitis, common indications for stem cell transplant and common biliary interventions.

Results: Of the 140,130 adult patients with HSCT, 855 (0.61%) patients developed acute pancreatitis. A complete list of demographics is presented in Table: There was a 2.2 times higher risk of mortality in patients with AP as compared to controls (aOR: 2.2, p-0.012). There was no statistically significant difference in the length of stay, hospitalization charge or cost before sensitivity analysis. After sensitivity analysis, 13,240 patients were included, from which 125 (0.94%) had AP. There was 4.25 times higher risk of mortality in patients who developed AP as compared to controls (aOR: 4.25, p-0.002). There was a statistically significant increase noted in the length of stay (adj coeff: 19.96 days, p-0.02), hospital charge (+$354,527.3, p-0.014) and cost (+$119,822, p-0.001) in patients with AP as compared to those who did not develop AP.

Conclusion: Recipients of HSCT who develop AP have shown to have higher mortality in both initial analysis as well as sensitivity analysis. This study highlights that acute pancreatitis in HSCT patients is associated with worse outcomes and higher resource utilization. Physicians should be aware about this association as presence of pancreatitis portends poor prognosis.

Table 1. Characteristics of patients with hematopoietic stem cell transplant (HSCT) stratified by presence of acute pancreatitis

	Without pancreatitis n (%)	With pancreatitis n (%)	p-value
Age category			< 0.001
18-45	30,045 (21.57)	360 (42.11)	
45-65	63,845 (45.84)	330 (38.6)	
>65	45,385 (32.59)	165 (19.30)	
Sex			0.26
Male	79,465 (57.06)	450 (52.60)	
Female	59,810 (42.94)	405 (47.40)	
Race			0.38
White	97,105 (69.72)	540 (63.16)	
African American	17,020 (12.22)	140 (16.37)	
Hispanic	14,695 (10.55)	110 (12.87)	
Asian/Pacific islander	4,405 (3.16)	35 (4.09)	
Native American	475 (0.34)	0 (0)	
Other	5,575 (4.00)	30 (3.51)	
Insurance			0.05
Medicare	53,505 (38.42)	260 (30.41)	
Medicaid	15,735 (11.3)	180 (21.05)	
Private	64,080 (46.01)	365 (42.69)	
Uninsured	1,485 (1.07)	25 (2.92)	
Income			0.27
Lowest quartile	28,760 (20.65)	190 (22.22)	
Second quartile	32,890 (23.62)	235 (27.49)	
Third quartile	37,755 (27.11)	175 (20.47)	
Highest quartile	39,870 (28.63)	255 (29.82)	
Indications of HSCT			
Myelodysplastic	9,375 (6.73)	50 (5.85)	0.64
Lymphoid leukemia	11,595 (8.33)	120 (14.04)	0.009
Myeloid leukemia	26,095 (18.74)	155 (18.13)	0.84
Hodgkin lymphoma	4,430 (3.18)	20 (2.34)	0.53
Non-Hodgkin lymphoma	3,505 (2.52)	15 (1.75)	0.52
Bone marrow failure syndromes	30 (0.02)	0 (0)	0.85
HLH	290 (0.21)	0 (0)	0.57
Thalassemia	290 (0.21)	0 (0)	0.57
Sickle cell disease	1,590 (1.14)	25 (2.92)	0.06
Metabolism error diseases	210 (0.15)	0 (0)	0.64
Primary immunodeficiency	5,710 (4.10)	35 (4.09)	0.99
GVHD	19,390 (13.92)	185 (21.64)	0.005
Hemolytic uremic	130 (0.09)	0 (0)	0.73
Conditions related to pancreatitis			
Cholangitis	660 (0.47)	55 (6.43)	< 0.001
Choledocholithiasis	1,680 (1.21)	135 (15.79)	< 0.001
Other biliary conditions	1,680 (1.21)	55 (6.43)	< 0.001

Table 1. (*continued*)

	Without pancreatitis n (%)	With pancreatitis n (%)	p-value
Hypertriglyceridemia	1,025 (0.74)	90 (10.53)	< 0.001
SLE	455 (0.33)	10 (1.17)	0.06
Diabetic ketoacidosis	255 (0.18)	25 (2.92)	< 0.001
Alcohol-related pancreatitis	1,805 (1.30)	50 (5.85)	< 0.001

HLH: hemophagocytic lymphohistiocytosis, GVHD: graft versus host disease, SLE: systemic lupus erythematosus.

S87

Trends in Prescription and Non-Prescription Digestive Enzyme Use in the United States From 2009 to 2020

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Introduction: The use of digestive enzymes like amylase, lipase, and related compounds has not been well-characterized. There are both prescription medications and nonprescription supplements that contain a range of digestive enzymes, which may impact the gut microbiome in diverse ways or have other effects. Our aim was to describe the use of prescription and non-prescription digestive enzymes by US adults and children and reported reasons.
Methods: We used data from the Centers for Disease Control and Prevention's National Health and Nutrition Examination Survey (NHANES) from 2009 to 2020 to assess prescription and nonprescription supplement use in the prior 30 days. We evaluated ingredient lists to identify those including digestive enzymes. We then compared trends in use over time and by sex, age, race/ethnicity, education, income, and alcohol use using quasipoisson models.
Results: Prescription and nonprescription digestive enzyme use was stable from 1999 to 2020. Prevalence of use of non-prescription digestive enzyme-containing products was 1.6% for all age groups (95% confidence interval (CI) 1.4-1.8, Table). Prevalence of prescription digestive enzymes was 0.05% overall (95% CI 0.03-0.07, Table). Rates of non-prescription digestive enzymes were significantly higher in older age groups (Table). Individuals aged 60 years and older were the most likely to take prescription digestive enzymes (0.2% prevalence, p=0.006 compared to < 60 years). There were no significant gender-related differences in digestive enzyme use. Non-Hispanic white individuals were significantly more likely to use non-prescription digestive enzymes than other racial/ethnic groups (p=0.003). There was significantly more non-prescription digestive enzyme use by individuals who had ever used alcohol (p=0.01), had higher household incomes (p< 0.001), and had some college education or more (p=0.005 compared to less than a 9th grade education). The most common reasons for non-prescription digestive enzyme use were to improve and maintain overall health and to get more energy. Only 13% of people on non-prescription digestive enzyme supplements reported that the product was recommended by a healthcare provider.
Conclusion: Around 5.3 million people in the United States take non-prescription digestive enzymes. Higher income, older, more educated, non-Hispanic white people who have ever used alcohol are more likely to take non-prescription enzymes. They are most often used without the recommendation of a healthcare provider.

Table 1. **Characteristics of non-prescription and prescription digestive enzyme use in the United States. Weighted estimates as percent prevalence in the United States based off a sample of n=55,969 individuals. P-values from unadjusted quasipoisson models of enzyme use and each characteristic. Data from CDC NHANES 2009-2020**

Characteristics	Non-prescription digestive enzymes			Prescription digestive enzymes		
	Estimate (% prev)	SE	p-value	Estimate (% prev)	SE	p-value
Overall	1.64	0.10		0.05	0.01	
Age						
Under 20 years	0.55	0.08	Ref.	0.01	0.01	Ref.
20-39 years	1.71	0.19	< 0.001	0.03	0.02	0.43
40-59 years	1.93	0.18	< 0.001	0.02	0.01	0.50
60 and over	2.60	0.31	< 0.001	0.16	0.04	< 0.001
Gender						
Men	1.49	0.12	Ref.	0.05	0.01	Ref.
Women	1.78	0.15	0.12	0.05	0.01	0.66
Race/Ethnicity						
Hispanic	1.17	0.15	Ref.	0.04	0.02	Ref.
Non-Hispanic Asian	1.15	0.17	0.94	0.05	0.04	0.62
Non-Hispanic Black	1.06	0.12	0.54	0.08	0.03	0.17
Non-Hispanic White	1.96	0.18	< 0.01	0.05	0.01	0.48
Other	1.84	0.55	0.14	0.07	0.07	0.54
Education level (adults)						
Less than 9th grade	0.98	0.27	Ref.	0.07	0.04	Ref.
9th-11th grade, no HS diploma	1.15	0.27	0.66	0.11	0.05	0.53
High school graduate/GED	1.19	0.16	0.52	0.04	0.02	0.37
Some college or AA degree	2.21	0.20	< 0.01	0.09	0.02	0.52
College graduate or above	2.91	0.31	< 0.001	0.04	0.01	0.74
Household Percent income to poverty ratio						
PIR≤130%	0.92	0.12	Ref.	0.06	0.02	Ref.
PIR >130%	1.93	0.13	< 0.001	0.05	0.01	0.50
Alcohol use						
Ever	2.38	0.28	Ref.	0.08	0.03	Ref.
Never	1.59	0.34	0.04	0.05	0.03	0.56

S88

Immune Checkpoint-Inhibitors Are Associated With a Higher Risk of Cholangitis: A Nationwide Population-Wide Study

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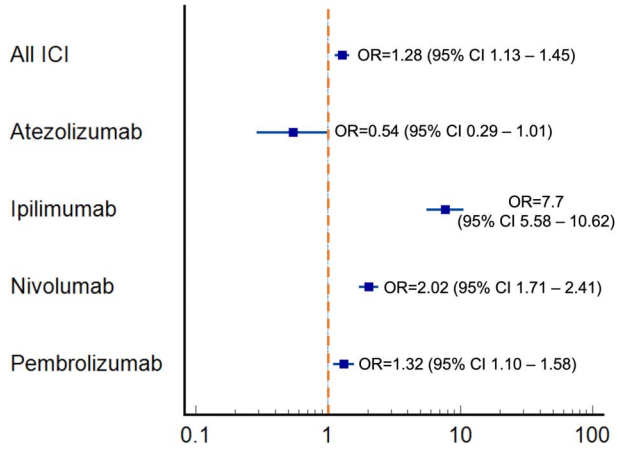
Introduction: Immune checkpoint-inhibitors (ICIs) improve the survival in certain cancers. However, side effects of ICIs might limit its use. Little is known about their biliary toxicity. Accordingly, we used a large database to investigate the epidemiology of ICI-induced cholangitis and describe underlying associations.
Methods: A multi-institutional database (Explorys Inc, Cleveland, OH, USA), an aggregate of electronic health record data from 26 US healthcare systems was surveyed. A cohort of patients who were on ICIs (nivolumab, pembrolizumab, ipilimumab and atezolizumab) between 2011 and 2022 was identified. Subsequently, patients who developed new Systematized Nomenclature of Medicine-Clinical Terms diagnosis of cholangitis after taking ICIs were selected. The prevalence of ICI-induced cholangitis as calculated, and underlying associations were described.
Results: There were 70,398,640 in the database of which 417,390 patients had a diagnosis of cholangitis. Among these patients, 260 patients had a prior history of ICI use. Compared to patients with history of ICI use who didn't develop cholangitis (n=19,940), patients with ICI and cholangitis were more likely to be female [OR: 1.38; 95% CI 1.08–1.76]. There were no statistically significant age or race-based differences. Overall, patients who received any ICI had a significantly higher risk of cholangitis compared to the general population [OR: 1.28; 95% CI 1.13–1.45]. Patients who received Ipilimumab had the highest odds of developing ICI-induced cholangitis (Figure).
Conclusion: In this large retrospective study, we found that patients taking ICI have a higher risk of cholangitis compared to the general population. Ipilimumab poses the greatest risk for ICI-induced for cholangitis. The risk of cholangitis should be discussed with all patients prior to initiating an ICI, as it may be a factor in choosing among ICIs.



[0088] **Figure 1.** Odds Ratio with 95% Confidence Interval of Immune Checkpoint Inhibitor-Induced Cholangitis

Table 1. Baseline Characteristics of Patients Receiving ICIs

Variable	Patients who received ICI		Patients with Cholangitis who did not receive ICI
	ICI without Cholangitis	ICI-Induced Cholangitis	
Age			
18 -65	7190 (37%)	90 (35%)	417,140 (59%)
>65	12,250 (63%)	160 (62%)	288,960 (41%)
Female	8,130 (42%)	130 (50%)	475,350 (67%)
Race			
Caucasian	16,150 (84%)	220 (85%)	
Others	3180 (16%)	40 (15%)	
Co-morbidities			
Hypertension	19,330 (71%)	200 (77%)	382,890 (54%)
T2DM	5,560 (29%)	90 (35%)	176,910 (25%)
Hyperlipidemia	11,830 (62%)	160 (62%)	329,520 (47%)
Obesity	4,510 (23%)	90 (35%)	236,120 (33%)
CAD	5,720 (30%)	90 (35%)	130,060 (18%)
Choledocholithiasis	1,570 (8%)	170 (65%)	532,590 (75%)

S89

Analyzing Trends in the Incidence and Mortality of Pancreatic Cancer and Cholangiocarcinoma in the United States: A 15-Year Population-Based Study

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Introduction: Pancreatic cancer and cholangiocarcinoma have historically been associated with particularly poor survival rates. The aim of this study was to identify temporal trends for these two malignancies in a national population cohort admitted to U.S. hospitals from the years 2005-2019.
Methods: The National Inpatient Sample (NIS) database for the years 2005-2019 was queried to identify adult (age >18 years) patients admitted with the principal procedural codes for pancreatic cancer and cholangiocarcinoma. Data was obtained from US states. We estimated trends in the total number of patients yearly, prevalence, mortality, and mortality rate for patients admitted for pancreatic cancer and cholangiocarcinoma. Weighted analysis utilizing Stata 17 MP was performed.
Results: A total of 1,406,778 patients had pancreatic cancer, of which 119,176 died. A total of 204,347 patients were found to have cholangiocarcinoma, of which 15,863 died. Throughout the years, there was an increase in the prevalence in pancreatic cancer from 0.19% to 0.33%, mortality rates decreased from 12% to 7.1% ($p< 0.01$), hospital length of stay decreased from 7.7 to 6.1 days ($p< 0.01$), total hospital charges increased from $37575 in 2005 to $73564 in 2019, Mean age remained the same 67.8 years in 2005 and 67 years in 2009 ($p >0.05$) (Table 1,2). Over the same timeframe, there was an increase in the prevalence of cholangiocarcinoma from 0.02% to 0.07%, mortality rates decreased from 11.4% to 7.4% ($p< 0.01$), hospital length of stay decreased from 8.8 days in 2005 to 6.6 days in 2019 ($p< 0.01$), total hospital charges increased from $35951 in 2005 to $83729 in 2019 ($p< 0.01$), mean age remained the same 67.9 years in 2005 and 68.3 years in 2019 ($p< 0.01$).

Conclusion: Pancreatic cancer and cholangiocarcinoma continue to be among the deadliest malignancies in terms of mortality rate. However, over the past 15 years advancements have been made in ERCP and EUS which have allowed for an increased rate of diagnosis. Furthermore, mortality rates have significantly decreased in both diseases, which further highlights the importance and efficacy of these diagnostic interventions in treating these malignancies. This is the first NIS study to analyze trends in these two disease entities over this specific period (2005-2019). Our study also reflects that the advancements in our diagnostic modalities have likely significantly improved mortality rates and demonstrates the importance of further development of these modalities in the future.

Table 1. Prevalence and mortality rates of Pancreatic Cancer from 2005-2019

Year	Total	Prevalence	Mortality (n)	Mortality rate (%)	P value (Moratlity rate)
2005	73,323	0.19%	8,929	12%	p<0.05
2006	74,048	0.19%	8,179	11%	p<0.05
2007	82,385	0.21%	8,041	9.8%	p<0.05
2008	89,468	0.22%	8,757	9.8%	p<0.05
2009	86,829	0.22%	8,162	9.4%	p<0.05
2010	95,014	0.24%	7,894	8.3%	p<0.05
2011	96,743	0.25%	7,785	8.0%	p<0.05
2012	90,504	0.25%	7,105	7.8%	p<0.05
2013	91.095	0.26%	7,015	7.7%	p<0.05
2014	95,130	0.27%	7,360	7.7%	p<0.05
2015	97,924	0.27%	3,757	7.7%	p<0.05
2016	102,250	0.29%	7,615	7.4%	p<0.05
2017	105,775	0.30%	8,320	7.8%	p<0.05
2018	110,220	0.31%	8,220	7.4%	p<0.05
2019	116,070	0.33%	8,280	7.1%	p<0.05

Table 2. Prevalence and Mortality Rate of Cholangiocarcinoma from 2005-2019.

Year	Total	Prevalence (%)	Mortality (n)	(Mortality rate)	P value (Mortality rate)
2005	7,205	0.02%	825	11.4%	$p < 0.05$
2006	7,405	0.02%	687	9.2%	$p < 0.05$
2007	8,849	0.02%	875	9.9%	$p < 0.05$
2008	9,737	0.02%	908	9.3%	$p < 0.05$
2009	10,069	0.03%	919	9.1%	$p < 0.05$
2010	11,864	0.03%	931	7.9%	$p < 0.05$
2011	11,468	0.03%	878	7.7%	$p < 0.05$
2012	11,945	0.03%	800	6.7%	$p < 0.05$
2013	12,750	0.04%	970	7.6%	$p < 0.05$
2014	13,970	0.04%	1,075	7.6%	$p < 0.05$
2015	11,060	0.04%	620	8.1%	$p < 0.05$
2016	18,990	0.05%	1,410	7.4%	$p < 0.05$
2017	20,745	0.06%	1,470	7.1%	$p < 0.05$
2018	22,815	0.06%	1,600	7.0%	$p < 0.05$
2019	25,475	0.07%	1,895	7.4%	$p < 0.05$

S90

Adherence to Most Recent Guidelines on Pancreatic Cysts Management

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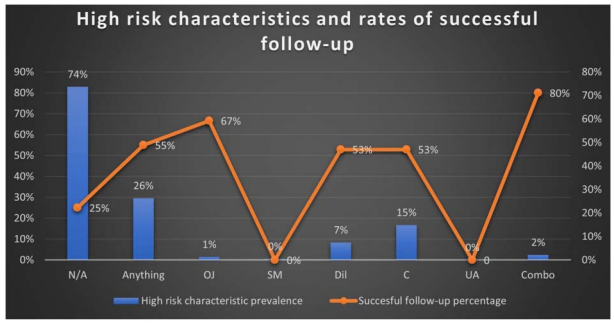
Introduction: Intraductal Papillary Mucinous Neoplasms (IPMN), pseudocysts, mucinous cystic neoplasms (MCN), serous cystadenomas (SCA), other non-cancerous cysts (NoNC) are all terms describing pancreatic cysts. Some of those have been identified as pre-malignant. IPMN malignant transformation risk greatly varies depending on location, while MCN also represent potentially pre-malignant lesions, with studies showing malignancy in up to 17.5%. Pancreatic cancer is a lethal, rapidly progressive disease with low 5-year survival rate. Clear guidelines have been released to monitor pancreatic cysts, discovered incidentally on imaging, in order to identify and excise high-risk lesions prior to malignant transformation.
Methods: This is a retrospective observational cohort study, consisting of adult patients incidentally diagnosed with pancreatic cysts on imaging at our hospital from January 2016 to December 2019. Any follow-up 6 months earlier or later than the suggested time and utilization of screening methods other than the proposed were considered unsuccessful.
Results: Of the 228 patients included only 33% received appropriate follow-up. Patients who had a new cyst and at least one high risk characteristic on initial presentation were followed correctly in 55% of cases. Patients with more than one risk factor had even higher rates of appropriate follow-up. *(Figure)* On further analysis, the current study identifies IPMN lesions measuring < 1cm, 1-2 cm, and 2-3 cm having succesful follow-up in 16%, 35% and 53% of cases, respectively, demonstrating a clear association between the cyst size and the success of surveillance.
Conclusion: Our study shows that only 33% of patients with incidentally discovered pancreatic cysts were successfully followed up based on current guidelines. Studies in the past also revealed that only around 1/3 of patients who had pancreatic cysts met minimum surveillance criteria, while presence of main pancreatic duct dilatation and absence of multiple pancreatic cysts were both associated with higher success rates. Our study confirmed that patients with any high risk characteristic had higher rates of appropriate follow-up, while the rates of successful management were directly proportional to the cyst size. Our study highlights again the gap between published guidelines and their implementation in daily practice. Additional interventions and methods for improvement should be further studied.



[0090] **Figure 1.** (N/A: No high-risk characteristic, Anything: Any high-risk characteristic, OJ: Obstructive Jaundice, SM: Associated Solid Mass presence, Dil: Main pancreatic duct diameter>5mm, C: Cyst size>3cm, UA: Change in main duct caliber with upstream atrophy, Combo: Combination of one or more high-risk characteristics)

S91

Management of Biliary Complications in Liver Transplant Recipients Using a Fully Covered Self-expandable Metal Stent With Antimigration Features

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Introduction: Following liver transplant (LT) with duct-to-duct anastomosis, biliary strictures and leaks are typically managed with endoscopic retrograde cholangiopancreatography (ERCP) and stenting. While multiple plastic stents are typically used for strictures, self-expandable metal stents (SEMS) can be used to decrease the number of ERCPS with longer periods of stent patency. However, their use is limited by stent migration. The use of fully covered SEMS (FCSEMS), with antimigration fins to manage benign biliary complications following LT may provide stricture resolution with limited adverse events. As such we aimed to describe clinical outcomes of FCSEMS in LT recipients using one type of FCSEMS.
Methods: This was a single center retrospective study of consecutive adult LT patients undergoing FCSEMS from January 2014 to April 2022. Primary outcomes were stricture resolution and recurrence. Secondary outcomes included rates of stent migration, stent occlusion, and number of ERCPs required.
Results: 46 LT patients with anastomotic strictures (n=40), bile leaks (n=4) or both (n=2) (average age 55.8 years, 33% women) were treated with a FCSEMS with antimigration features (Viabil, W.L. Gore, Flagstaff, AZ) approved for malignant strictures. The median time from LT to FCSEMS placement was 132.5 days. Within one year of LT, 32 patients (69.5%) required intervention; early intervention at less than 30 and 90 days was needed in 7 and 20 patients, respectively. At the time of study conclusion there were 3 patients with stents in situ. Stricture resolution was seen in 34 patients (74%) after an average stent dwell time of 134.5 ± 71 days; recurrence was observed in 8 patients (17%). There were three instances of partial proximal stent migration that did not require reintervention or interfere with removability. Procedural details are listed in Table: The total mean number of ERCPS required was 2.48 (range 1-5).
Conclusion: The use of a FCSEMS with antimigration features offers an opportunity to enhance stricture resolution with longer stent dwell times and fewer ERCPs. In our cohort there were no instances of clinically significant stent migration, dysfunction, or procedure related adverse events. Furthermore, these stents provide an opportunity reduce procedure related costs. Additional studies are needed to identify suitable patients for FCSEMS placement.

Table 1. Procedure related details and stent outcomes

Stent Dwell Time +/- Standard Deviation (days)	134.5 +/- 71.8
Stent Placement transpapillary vs intraductally, n (%)	36 (78.3%) vs 10 (21.7%)
Prior Sphincterotomy, n (%)	17 (40%)
Stricture Recurrence, n (%)	8 (17%)
Average Number ERCPs, n (range)	2.48 (1-5)
Stent Migration, n (%)	3 (6.5 %)
Stent Occlusion, n (%)	8 (17%)

S92

Racial and Ethnic Disparities in Opioid Prescriptions in Benign and Malignant Pancreatic Disease in the United States, 2006-2015

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Introduction: Racial and ethnic disparities in pain management are well established, but not for patients with pancreatic disease. We sought to evaluate racial-ethnic disparities in opioid prescriptions for patients with acute and chronic pancreatitis and pancreatic cancer.
Methods: Data from the National Ambulatory Medical Care Survey (NAMCS) were used to examine opioid prescriptions recorded during ambulatory visits by adult pancreatic disease patients between 2006 and 2015. Diagnoses were determined by ICD-9 codes 557.0 and 577.1 for acute and chronic pancreatitis and 157 for pancreatic cancer. Visits by patients with other painful conditions associated with opioid use were excluded. Bivariate analysis was used to detect differences in opioid prescriptions by patient race, ethnicity, and sex. Data weights for national-level estimates were not applied in accordance with NAMCS guidelines requiring a relative standard error of 30% or less.
Results: We identified 421 outpatient visits by adults with pancreatic disease. Eighteen patients with comorbid painful conditions (lumbago, myalgia, generalized pain, ankylosing spondylitis, osteoarthritis, multiple sclerosis, and mononeuritis) were excluded. A total of 207 pancreatitis and 196 pancreatic cancer patient visits were identified, representing 9.8 million visits, but weights were repealed for analysis. No sex differences in opioid prescriptions were found among pancreatitis (p=0.78) or pancreatic cancer patient visits (p=0.57). Opioids were prescribed at 58% of non-Hispanic Black, 37% non-Hispanic White, and 19% Hispanic pancreatitis (P=0.05) patient visits (Table). Opioid prescriptions were less common in visits by Hispanic compared to non-Hispanic pancreatitis patients (OR 0.35, 95% CI 0.14-0.91, p=0.03) and there was a trend toward higher rates in non-Hispanic Black versus non-Hispanic White patient visits. No racial-ethnic differences in opioid prescriptions were seen among pancreatic cancer patient visits.
Conclusion: While racial-ethnic disparities in opioid prescriptions were observed in pancreatitis patient visits, none were seen in pancreatic cancer patient visits. These findings suggest racial and ethnic bias in opioid prescription practices for patients with painful but benign pancreatic disease, but there may be a lower threshold for opioid provision in the treatment of malignant and terminal pancreatic disease.

Table 1. Demographic characteristics and likelihood of opioid use among visits by adult pancreatitis and pancreatic cancer patients in NAMCS, 2006-2015

		No opioid	Opioid Prescription	OR (95% CI)	p-value
Pancreatitis		N=132 (64%)	N=75 (36%)		
Race/Ethnicity, %					
A)	Non-Hispanic	106 (61)	69 (39)	Reference	Reference
	Hispanic	26 (81)	6 (19)	**0.35 (0.14-0.91)**	**.03**
B)	White non-Hispanic	87 (63)	52 (37)	Reference	Reference
	Black non-Hispanic	10 (42)	14 (58)	2.34 (0.97-5.65)	.06
	Hispanic	26 (81)	6 (19)	**0.39 (0.15-1.00)**	**.05**
	Other non-Hispanic	9 (75)	3 (25)	0.56 (0.42-0.84)	.40
Sex, %					
	Female	59 (65)	32 (35)	Reference	Reference
	Male	73 (63)	43 (37)	1.09 (0.61-1.92)	.78
Pancreatic Cancer		N=126 (64%)	N=70 (36%)		
Race/Ethnicity, %					
	White non-Hispanic	97(64)	54(36)	Reference	Reference
	Black non-Hispanic	17(65)	9(35)	0.95 (0.40-2.28)	.91
	Hispanic	8(62)	5(38)	1.12 (0.35-3.60)	.85
	Other non-Hispanic	4(67)	2(33)	0.90 (0.16-5.06)	.90
Sex, %					
	Female	63(62)	38(38)	Reference	Reference
	Male	63(66)	32(34)	0.84 (0.47-1.51)	.57

S93

Native vs Non-Native Papilla: Defining and Mitigating the Post-ERCP Pancreatitis Risk

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Introduction: Pancreatitis occurs in approximately 5-10% of patients undergoing endoscopic retrograde cholangiopancreatography (ERCP) making it the most common adverse event of an endoscopic procedure. While it has been proposed that native papilla increases the risk of post-ERCP pancreatitis (PEP), this risk has not been quantified and strategies to prevent PEP in this subset of patients is undefined.
Methods: As part of an ongoing randomized clinical trial at Los Angeles County Hospital (Clinicaltrials.gov: NCT03087656), we captured patient characteristics and procedural factors including the anatomy, procedural complexity score, use of rectal indomethacin, and volume of fluids administered for patients both included and excluded from the trial. The primary predictor was the presence of a native or non-native papilla. The primary outcome was the development of PEP. We used a bivariate regression model to determine whether prophylactic rectal indomethacin and aggressive hydration (>3.5L of fluids/24 hours) mitigated the risk of PEP.
Results: From October 2019 to December 2021, we evaluated 416 cases of ERCP. PEP developed following 25 procedures (overall incidence 6.0%), of which 22 had a native papilla (unadjusted OR 4.1; 95% CI 1.3-12.8). On multivariate analysis adjusting for the ERCP complexity score, patients with a native papilla were more likely to develop PEP (OR 5.4; 95% CI 1.6-17.9). Rectal indomethacin was used in 268 procedures, aggressive hydration in 283 procedures, and both maneuvers in 211 procedures (Table). Native papilla remained a compelling risk factor in patients who received rectal indomethacin (adjusted OR 6.0; 95% CI 1.2-30.6), aggressive hydration (adjusted OR 7.8; 95% CI 1.6-39.0), and the combination (adjusted OR 9.0; 95% CI 1.1-77.3).
Conclusion: Patients with a native papilla are significantly more likely to develop PEP regardless of procedural complexity. This association remained in the setting of rectal indomethacin use and aggressive hydration. This study emphasizes the need to develop and study preventative measures particularly in patients undergoing their first ERCP.

Table 1. Post-ERCP Pancreatitis in Native versus Non-Native Papilla; Overall Cohort and Subset Receiving Prophylactic Measures

	N	PEP in Native Papilla (n=262)	PEP in Non-Native Papilla (n=154)	Univariate OR (95% CI)	Multivariate OR (95% CI)
Total Cohort	416	22	3	**4.1 (1.3-12.8)**	**5.4 (1.6-17.9**
Rectal Indomethacin	268	18	2	3.8 (0.9-16.7)	**6.0 (1.2-30.6)**
Aggressive Hydration	283	19	1	**6.0 (1.2-29.4)**	**7.8 (1.6-39.0)**
Indomethacin + Hydration	211	16	1	5.7 (0.7-44.1)	**9.0 (1.1-77.3)**

S94

Pre-Existing, Early Onset and Late Onset Diabetes in Chronic Pancreatitis: Do Outcomes Differ?

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Introduction: Diabetes secondary to endocrine insufficiency in chronic pancreatitis (CP) may develop at any time during the course of the disease. Not much is known about the clinical characteristics and outcomes in these patients. Hence we sought to evaluate differences in pre-existing diabetes, early-onset diabetes, and late-onset diabetes in CP patients.
Methods: We retrospectively reviewed CP patients seen at our Pancreas Center between 2016 and 2021. We divided the patients into four groups based on a co-diagnosis of diabetes: those without diabetes, those with pre-existing diabetes (more than 2 years before CP diagnosis), those with early-onset diabetes (diagnosed within 2 years before or 2 years after CP diagnosis), and those with late-onset diabetes (diagnosed 2 years after CP diagnosis). Patients with diabetes after surgery were excluded. We then compared the clinical characteristics, parameters of glucose control, and outcomes.
Results: We identified 450 patients with CP: 271 without diabetes, 99 with pre-existing diabetes, 51 with early-onset diabetes and 29 with late-onset diabetes. Early-onset diabetes patients were younger (54.1 vs 57.3 vs 62.5 vs 61.9 years) and more likely to have alcohol-related CP (45.1% vs 31.7% vs 32.3% vs 31%) compared to the other three groups ($p< 0.05$). Early-onset diabetics had a higher mean HbA1C level (8.02% vs 5.11% vs 7.71% vs 7.66%) and were more likely to be on insulin (78.4% vs 0% vs 48.4% vs 65.5%, $p< 0.05$). Early-onset diabetics used more opioids (64.7% vs 43.9% vs 55.1% vs 44.8%) and gabapentinoids (66.7% vs 43.5% vs 48% vs 60.7%) compared to other groups ($p< 0.05$). Patients who developed diabetes after the diagnosis of CP had significantly higher rates of exocrine insufficiency,

anatomical complications and interventions for pain control. Other demographics and common complications are outlined in Table: There was no significant difference in pancreatic cancer in the four groups (p=0.7).

Conclusion: Our study suggests that CP patients who are younger and use alcohol, are at higher risk of having early-onset diabetes after CP diagnosis. This population should be screened earlier as they have poorer glucose control and higher insulin requirement compared those with pre-existing and late-onset diabetes. Also, patients who develop diabetes after CP diagnosis have worse outcomes and use more resources. Long-term prospective studies are needed to generate more robust data.

Table 1. Demographics, clinical characteristics, and resource utilization of CP patients with and without diabetes

	No diabetes N=271	Pre-existing diabetes N=99	Early-onset diabetes (< 2 years) N=51	Late-onset diabetes (>2years) N=29	P-value
Mean age in years (SD)	57.3 (16.0)	62.5 (12.8)	54.1 (13.5)	61.9 (11.5)	**0.002**
Mean age at diagnosis of CP (SD)	45.0 (16.2)	53.0 (14.5)	41.2 (15.5)	47.8 (13.4)	**<0.001**
Female sex (%)	133 (49.6%)	43 (43.4%)	22 (43.1%)	13 (44.8%)	0.659
White race (%)	201 (75.6%)	71 (71.7%)	34 (66.7%)	24 (82.8%)	0.34
Mean number of follow up years in pancreas center	7.84 (5.36)	7.86 (6.68)	8.74 (5.49)	12.1 (5.05)	**0.004**
Etiology of CP					**<0.001**
Alcohol	86 (31.7%)	32 (32.3%)	23 (45.1%)	9 (31.0%)	
Genetic	8 (2.95%)	2 (2.02%)	3 (5.88%)	1 (3.45%)	
Idiopathic	88 (32.5%)	21 (21.2%)	8 (15.7%)	6 (20.7%)	
Smoking	69 (25.5%)	36 (36.4%)	15 (29.4%)	11 (37.9%)	
Other	20 (7.38%)	8 (8.08%)	2 (3.92%)	2 (6.90%)	
History of recurrent acute pancreatitis (%)	214 (78%)	69 (69.6%)	35 (68.6%)	21 (72.4%)	0.943
Chronic abdominal pain (%)	205 (79.2%)	80 (80.8%)	48 (94.1%)	23 (79.3%)	0.094
Splanchnic vein thrombosis(%)	22 (8.43%)	15 (15.3%)	7 (13.7%)	8 (27.6%)	**0.013**
Pseudocyst (%)	81 (30.9%)	39 (39.8%)	25 (49.0%)	14 (48.3%)	**0.027**
Biliary obstruction (%)	30 (11.7%)	22 (22.7%)	9 (17.6%)	7 (24.1%)	**0.031**
Vitamin D deficiency (%)	78 (32.1%)	45 (51.7%)	23 (48.9%)	20 (83.3%)	**<0.001**
Mean Hba1c Level (SD)	5.11 (1.15)	7.71 (1.38)	8.02 (1.61)	7.66 (0.98)	**<0.001**
On insulin (%)	0	48 (48.4%)	40 (78.4%)	19 (65.5%)	**<0.001**
Pancreatic malignancy (%)	10 (3.88%)	4 (4.08%)	3 (5.88%)	0 (0.00%)	0.709
Exocrine pancreatic insufficiency (%)	144 (53.1%)	65 (65.7%)	36 (70.6%)	21 (72.4%)	**0.014**
Opioid use (%)	116 (43.9%)	54 (55.1%)	33 (64.7%)	13 (44.8%)	**0.024**
Non-opioid controlled medications (%)	63 (24.0%)	32 (32.7%)	17 (33.3%)	10 (34.5%)	0.220
Gabapentinoid use (%)	114 (43.5%)	47 (48.0%)	34 (66.7%)	17 (60.7%)	**0.012**
Medical marijuana use (%)	19 (7.66%)	8 (8.60%)	9 (18.8%)	3 (10.7%)	0.120
Mean number of flares requiring hospitalization (SD)	2.02 (3.42)	2.55 (3.18)	3.06 (3.44)	2.41 (3.39)	0.202
Recurrent ED visits (%)	15 (5.75%)	9 (9.18%)	4 (8.00%)	2 (6.90%)	0.617
Mean total abdominal imaging since diagnosis (SD)	4.28 (3.60)	5.64 (4.53)	7.49 (6.43)	7.28 (4.52)	**< 0.001**
Celiac block for pain (%)	27 (10.3%)	12 (12.2%)	17 (33.3%)	6 (21.4%)	**< 0.001**
Pancreatic surgery for pain (%)	22 (8.43%)	9 (9.18%)	15 (29.4%)	13 (46.4%)	**< 0.001**

S95

Increased Risk of Pancreatitis, Pancreatic Cancer, and Mortality in Patients With Celiac Disease

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Introduction: Celiac disease (CD) is an immune-mediated enteropathy associated with inflammation precipitated by dietary gluten in genetically predisposed individuals. Few studies reported a higher incidence of pancreatitis in the CD population. It is unclear whether this risk has changed in the era of more comprehensive diagnosis rates and less severe clinical disease. Using a large database, we sought to evaluate the association between CD and acute pancreatitis (AP), chronic pancreatitis (CP), and pancreatic cancer(PC) risk at a population level.

Methods: This multicenter, retrospective cohort study was conducted using the TriNetX platform. All adult patients (>18 years) diagnosed with CD were identified based on ICD-10 codes between March 2005 and April 2022. The primary outcome was the occurrence of AP and CP in CD patients. Secondary outcomes were the incidence of PC and mortality. Any patients with a history of pancreatic disease events before March 2005 were excluded. Data were matched with individuals without CD during the same period. We performed a 1:1 propensity score matching (PSM) for demographics, smoking, alcohol-related disease, diabetes, hyperlipidemia, and comorbid conditions. The risk ratio (RR) was calculated to compare the association between CD and the outcome

Results: A total of 479,020 patients were analyzed, and there were 162,362 patients with CD. After PSM, there were 142,902 patients with CD or without CD. The mean age of patients was 41.7 years, with 70.7% women and 78% White. Patients in the CD group had a higher prevalence of comorbidities such as diabetes and hypercholesterolemia than those without CD. The incidence of AP and CP after 3 years of CD diagnosis was 0.19% and 0.39%, respectively; PC and mortality were 0.04% and 1.16% compared to non-CD patients. Individuals with CD had a higher risk of AP (RR 1.30; 95% CI 1.08-1.56), CP(1.57; 95% CI 1.37-1.80), PC(1.48; 1.01-2.16) and mortality (RR 1.36; 95% CI 1.26-1.46) than non-CD patients (Table)

Conclusion: This population-based multicenter study suggests that individuals with CD are at increased risk of pancreatitis, PC, and mortality.

Table 1. Incidence of Pancreatitis, Pancreatic Cancer, and Mortality in patients with and without Celiac Disease after Propensity Matching at 3 years

	With CD		Without CD		Outcomes
Outcomes	Number of patients	Patients with the outcome, n(%)	Number of patients	Patients with outcome, n(%)	Risk ratio (95% CI)
Primary outcomes					
Acute Pancreatitis	140528	268(0.19)	138467	202(0.14)	1.30(1.08-1.56)
Chronic Pancreatitis	136924	546(0.39)	136212	345(0.25)	1.57(1.37-1.80)
Etiology of Pancreatitis					
Biliary Pancreatitis	142670	48(0.03)	142599	25(0.01)	1.91(1.18-3.11)
Idiopathic Pancreatitis	142744	45(0.03)	142651	28(0.01)	1.60(1.00-2.57)
Alcohol Pancreatitis	142752	28(0.02)	142724	10(0.01)	2.79(1.36-5.76)
Secondary outcomes					
Pancreatic cancer	142583	67(0.04)	142148	45(0.03)	1.48(1.01-2.16)
Mortality	137778	1611(1.16)	136892	1194(0.85)	1.36(1.26-5.76)

Abbreviations: CD, celiac disease; CI, confidence interval.

S96

Overutilization of Contrast-enhanced CT (CECT) Abdomen in the Diagnosis of Acute Pancreatitis (AP) in the Emergency Department (ED)

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Introduction: AP is an inflammatory disorder of the pancreas which is one of the leading causes of gastrointestinal disorder admissions in the United States (US). As incidence of AP increases, so does the demand for effective means of diagnosis. According to revised Atlanta classification of 2012, typical abdominal pain in the epigastrium or left upper quadrant along with an elevated lipase level greater than 3-5 times the upper limit of normal is enough to diagnose AP. Contrast-enhanced CT (CECT) should only be obtained in cases of atypical presentation or failure to improve in 48-72 hours. However, availability of CECT has led to overutilization of its use in the diagnosis of AP, which can lead to delay in management, unnecessary radiation, and increased cost burden. Here we report a one-year data from a single medical center to assess utilization of CECT in the emergency department (ED) in the diagnosis of AP and if it changed management, length of stay (LOS), or outcomes.
Methods: Data from 2018 to 2019 for all patients diagnosed with AP in the ED were analyzed. In total, 140 patients, 68 males and 72 females were analyzed. We evaluated lipase levels, CECT findings, LOS, and outcome. We used t-test to compare average means.
Results: The most common etiology causing AP was alcohol abuse (37%), idiopathic (28%), gallstones (22%), followed by 8 cases due to drugs, 6 due to hypertriglyceridemia, 2 due to hypercalcemia, and 1 due to a pancreatic mass. All patients had presented with epigastric pain. Average lipase among all patients was 1727 U/L with a median of 530 U/L. CECT imaging was performed in 105 patients (75%). Among those who underwent CECT abdomen, 80 cases (76%) had positive finding for AP, while remaining 25 (24%) did not. Average lipase values were significantly elevated (2290 U/L vs 1732 U/L) among those who did not undergo CECT abdomen compared to those who did (P-value < 0.02). There were no statistical significance in both groups as far as management, LOS, hospitalization course, or outcomes.
Conclusion: The purpose of this study was to assess how frequently was CECT used in the initial diagnosis in the ED setting and if it was helpful in changing management. Our results indicated that routine use of imaging in the diagnosis of AP is not necessary and has no impact on management and outcome. However, it can lead to increased radiation exposure and cost burden. Therefore, we recommend judicious use of CECT and recommend against routine utilization in the initial diagnosis of AP.

S97

Outcomes of Long-Term Aspirin Use on Patients With Ascending Cholangitis Undergoing ERCP: A Nationwide Analysis

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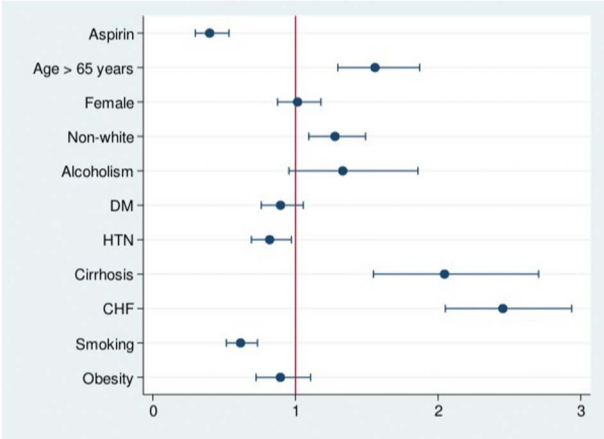
Introduction: Endoscopic retrograde cholangiopancreatography (ERCP) has been shown to improve mortality and outcomes in patients with ascending cholangitis. However, it is associated with various complications, with hemorrhage being one of the most common. Management of antiplatelet agents in patients undergoing ERCP is challenging because interrupting these agents increases the risk of cardiovascular and cerebrovascular events, whereas it increases bleeding risk if continued. Various society guidelines recommend continuing aspirin use in patients undergoing ERCP. However, there remains a paucity of data and wide variation in clinical practice. We conducted the study to evaluate the effects of aspirin use in patients with ascending cholangitis undergoing ERCP.
Methods: Data were extracted from the National Inpatient Sample (NIS) database from 2016 to 2019. The CD-10-CM codes were used to obtain baseline demographic data, in-hospital mortality, hospital charges, and hospital length of stay (LOS). Statistical analyses were completed using t-test and Chi-squared analysis. Multivariate analysis for the mortality odds ratio (OR) was calculated after adjusting for possible confounders.
Results: A total of 105,840 patients with ascending cholangitis underwent ERCP, out of which 17,445 patients were on long-term aspirin. The mean age of the aspirin group was 74.5 years vs. 67 years in the non-aspirin group (p-value < 0.001). The majority of the aspirin group population were males and whites. Long-term aspirin use was associated with lower odds (OR 0.38; $P< 0.001$) and a lower incidence of mortality (1.38% vs. 3.38%, $P< 0.001$) compared to non-aspirin users. Patients on aspirin were also more likely to have a lower total hospital charge ($73,507 vs. $93,071; $P< 0.001$) and a shorter LOS (5.66 vs. 6.8 days; $P< 0.001$). Age greater than 65 years, non-white race, cirrhosis, and congestive heart failure were independently associated with higher mortality. Long-term aspirin use was also associated with lower odds of mechanical ventilation (OR 0.51), vasopressor use (OR 0.65), and ICU admission (0.53), all with a statistical significance of p-value < 0.001. (Figure)
Conclusion: Aspirin has been shown to have other potential benefits as it may reduce the risk of certain malignancies and inflammatory diseases. Our study concluded that long-term Aspirin use is associated with a reduction in mortality, hospitalization complications, and length of stay in patients who are admitted with ascending cholangitis and undergoing ERCP. (Table)



[0097] **Figure 1.** Multivariate logistic regression for the effect of aspirin on ascending cholangitis undergoing ERCP

Table 1. Clinical characteristics and demographics of the study population

Variable	No Aspirin	Aspirin	P-value
Age (mean, yr)	67	74	< 0.001
Gender (%)			< 0.001
Male	50.8%	58.5%	
Female	49.2%	41.5%	
Race (%)			< 0.001
White	65%	75%	
Black	7%	6%	
Hispanic	13%	8%	
Others	15%	11%	
Comorbidities (%)			
Alcoholism	4.5%	2.9%	< 0.001
DM	29.8%	41.4%	< 0.001
HTN	62.2%	84.8%	< 0.001
CHF	12.4%	18.4%	< 0.001
Cirrhosis	4.7%	2.64%	< 0.001
Smoking	31.8%	42.88	< 0.001
Obesity	15.8%	16.2%	0.58

S98

Exocrine and Endocrine Pancreatic Insufficiency Induced by Immune Checkpoint Inhibitors: A Case-Control Study

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Introduction: The widespread use of immune checkpoint inhibitors (ICI) have shed light on a variety of ICI-induced pancreatic injuries. Of these, ICI-related diabetes (DM) has been recognized in multiple cases in literature, while ICI-related exocrine pancreatic insufficiency (EPI) has been recognized much less. We describe the first case-control study describing an association between EPI and worsening of glycemic control.
Methods: A single center, retrospective case-control study was performed of all patients treated with ICIs between January 2011 and July 2020. Patients with pancreas cancer, pancreas metastasis, or prior pancreatic surgery were excluded. Cases were patients with ICI-related EPI, defined as patients who started new pancrelipase after receiving ICI therapy, had clearly documented symptoms of EPI, including steatorrhea, abdominal discomfort, or weight loss, and demonstrated symptomatic improvement with pancrelipase. Controls were matched 2:1 by age, cancer type, gender, year of the first dose of ICI, and race. ICI-related hyperglycemia was defined as a new decline in glycemic control after the first dose of ICI, determined by a new HgbA1c $\geq$ 6.5 after the start of ICI or by initiation of new diabetes medications after ICI use.
Results: 23 patients developed ICI-related EPI and were matched to 46 controls. 9 (39.1%) patients with EPI developed new hyperglycemia after ICI use vs 3 (6.5%) controls ($p < 0.01$). Cases and controls had similar proportions of pre-existing diabetes prior to ICI use (2 (8.7%) vs 6 (13.0%), $p = 0.92$). The median time to onset of EPI was 390 days (IQR 252-578), while the median time to onset of hyperglycemia post ICI was 518 days (IQR 178-595). Notably, 7 of the 9 patients (77%) developed EPI and hyperglycemia within 10 weeks of the other. Of the EPI group, 2 patients developed new onset type 1 DM with autoantibodies, 3 patients developed acute hyperglycemia after steroid use, 2 of whom required ongoing antidiabetic agents, and 2 patients had decompensation of their pre-existing DM.
Conclusion: There are multiple phenotypes of hyperglycemia that can manifest after ICI therapy. The majority of these patients displayed loss of glycemic control within weeks of exhibiting symptoms of EPI. This is highly suggestive that there can be concurrent loss of endocrine and exocrine pancreatic function secondary to ICI use.

Table 1. Characteristics of Exocrine Pancreatic Insufficiency Patients with Hyperglycemia

Patient	Cancer Type	ICI	DM prior to ICI	Time to Onset of EPI (weeks)	Time to Onset of Hyperglycemia (weeks)	Clinical Presentation	Presenting Glucose (mmol/L)	Presenting A1c (%)	Presenting Fructosamine (mcmol/L)	C peptide	Auto-antibodies	Recent steroid use	DM agent	Type of Hyperglycemia
55M	Melanoma	CTLA-4/ PD-1	No	81	178	DKA	307	8.9	NR	0.1	IA-2	No	Insulin	Type 1 DM
73F	Lymphoma	PD-1	No	86	76	Asymptomatic	129	6.5	288	NR	NR	No	none	Unclear
62M	Genitourinary	PD-1	No	78	74	Asymptomatic	172	6.4	NR	NR	NR	No	PO	Type 2 DM
59M	GI/HPB	PD-L1	Yes	70	NA	NA	NA	NA	NA	0.41	NR	No	Insulin	Type 2 DM
67M	Melanoma	CTLA-4/ PD-1	No	15	11	Acute Hyperglycemia	404	9.3	346	2.94	NR	Yes	PO	Type 2 DM
61F	Melanoma	CTLA-4/ PD-1	No	79	413	Asymptomatic	NR	NR	NR	NR	NR	No	PO	Type 2 DM
56F	Genitourinary	CTLA-4/ PD-1	No	81	85	Asymptomatic	123	7.1	274	2.11	NR	No	none	Type 2 DM
50F	Lung	CTLA-4/ PD-1	Yes	39	NA	NA	NA	NA	NA	3.06	NR	No	Insulin + PO	Type 2 DM
57M	Lung	PD-1	No	21	25	Acute Hyperglycemia	594	9.8	NR	1.19	None	Yes	PO	Steroid Induced Hyperglycemia
47M	Melanoma	CTLA-4/ PD-1	No	2	11	Acute Hyperglycemia	470	8	NR	<0.2	GAD	Yes	Insulin	Type 1 DM
68M	Sarcoma	CTLA-4/ PD-1	No	49	55	Asymptomatic	131	6.5	NR	NR	NR	No	PO	Type 2 DM

Footnote: ICI, immune checkpoint inhibitor; EPI, exocrine pancreatic insufficiency; GI, gastrointestinal; HPB, Hepato-Pancreato-Biliary; DM, diabetes; NA, not applicable due to presence of diabetes before ICI administration; NR, not reported; PO, oral; DKA, diabetic ketoacidosis

S99

Patients With Cannabis Use and Cholangiocarcinoma Have Decreased Inpatient Mortality: A Population-Based Study

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Introduction: Cancer has numerous side effects, including pain, nausea, and emesis. Cholangiocarcinoma is cancer involving the biliary tree. It is a deadly disease entity with high morbidity and mortality. Furthermore, cancer-related pain is extremely common, especially in patients with advanced disease. The aim of our study was to investigate the outcomes of hospitalized patients who use non-traditional pain modalities such as cannabis.
Methods: All patients aged 18 years and above with a diagnosis of cholangiocarcinoma with and without cannabis use from 2015-to 2019 were identified from the US Nationwide Inpatient Sample (NIS), a large publicly available all-payer inpatient care database. ICD-10 codes were utilized. The primary outcome was inpatient mortality. Secondary outcomes were hospital length of stay (LOS) and total hospital charges (TOTHC). Statistical analysis was performed using STATA.
Results: We identified 92,940 patients who had cholangiocarcinoma, of which 815 patients were using Cannabis. After propensity score matching, patients with cannabis use and cholangiocarcinoma had decreased mortality (OR 0.40, p< 0.04, CI: 0.16-0.97). There was decreased LOS (-0.52 days, p=0.33, CI: -1.55-0.52) and decreased TOTHC ($-7,139, p=0.40, CI: $ -23,725-$9,447) compared to patients with only cholangiocarcinoma. However, LOS and TOTHC were not statistically significant.
Conclusion: Cholangiocarcinoma is a deadly disease entity that portends extremely high mortality and poor prognosis due to the diagnosis of the disease in a late/advanced stage. Cannabis historically has been considered an illicit substance, however, it has recently been used as a palliative treatment modality for cancer patients. Recently studies have shown that it is a safe, effective, and well-tolerated treatment that helps improve the quality of life for many patient populations. Our study is important because it is the first NIS study to investigate this patient population and proves that cannabis use is not always detrimental, especially since there is less mortality noted in patients with cholangiocarcinoma who use cannabis with no change in TOTHC or LOS.

S100

Predictors of Need of Surgery in Patients With Acute Necrotic Collections Treated With Percutaneous Drainage

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Introduction: Percutaneous drainage (PCD) is an important initial component of the "step-up" treatment strategy for the management of infected acute necrotic collections consequent to acute necrotizing pancreatitis (ANP). However, PCD alone has a high failure rate and these patients require additional necrosectomy. Delaying necrosectomy can increase morbidity and mortality and therefore, timely intervention is important. Identifying predictors of failure of percutaneous therapy can help in making timely management decisions. In this prospective single-center study, we attempted to identify predictors of failure of PCD in patients with infected ANC.
Methods: Twenty-one patients (males:13, mean age:36.05years) (etiology: ethanol-related 47.6% (10/21), GSD related 28.6% (6/21) unknown19% (4/21) PEP 4.8% (1/21)) admitted with infected ANC who were treated with step-up strategy of initial PCD followed by endoscopic or surgical necrosectomy in non-responders were enrolled and studied prospectively. We studied the association between the success of PCD (survival without necrosectomy) & baseline clinical and investigational parameters including etiology, severity scores, C-reactive protein (CRP), computed tomography severity indices (CTSI), and PCD parameters (size of catheter & timing).
Results: Nine (42.8%) patients with infected ANC were successfully treated with PCD alone whereas 12 (57.2%) patients either underwent necrosectomy or succumbed to the illness. The frequency of males was significantly higher (83.3% vs 33.33%) in the PCD failure group($p=0.03$). The PCD was inserted later in the failure group as compared to the successful group (mean day of insertion of PCD being 20.5 days vs 15.1 days respectively; $p=0.023$). The mean SIRS score at admission (2.92 vs 2.33; $p=0.102$) & at 48 hours (3.5 vs 2.89; $p=0.12$), mean APACHE-II score (7.8 vs 7.0; $p=0.508$), mean ferritin levels (2916.9 vs 1033.4ng/mL; $p= 0.394$), and mean CRP levels (249.5 vs 209.6ng/mL; $p=0.508$.) were higher in the PCD failure group but did not reach statistical significance. The corrected calcium levels were lower(8.3 vs 9.0gm/dL p=0.069) in the PCD failure group. Also, the mean diameter of PCD catheters was more in the success group but the difference was not statistically significant (16.2 vs 14.7F; $p=0.277$). (Table)
Conclusion: Male gender & delayed insertion of PCD seem to be associated with failure of PCD alone in patients with infected necrotic collections.

Table 1. Comparison of parameters between PCD Success and PCD Failure Group

Parameter	PCD Success group n=9	PCD failure group n=12	P Value
Age	37.3±7.5yrs	36.8±13.4yrs	0.65
Males	3(33.33%)	10(83.33%)	**0.03**
BMI	24.5±4.3Kg/m2	22.4±3.8kg/m^2	0.22
TLC	13871±6616.1/mm^3	15014.2±7818.6/mm^3	0.862
Platelet Count	321666.7±187813.7/mm^3	304350.0±155671.1/mm	0.972
Ferritin Level	1033.4±628.2ng/mL	2916.9±2961.8ng/mL	0.394
Corrected Calcium	9.0±0.3gm/dL	8.3±1.2gm/dL	**0.069**
qCRP Levels	209.6±95.9mg/L	249±107.1mg/L	0.508
SIRS			
At admission	2.33±0.5	2.92±1.0	0.10
At 48hrs	2.89±0.93	3.50±0.52	0.12
APACHE-II	7.0±3.0	7.8±8.0	0.51
MMS	1.4±0.9	1.7±1.8	0.70
BISAP	2.1±0.3	2.3±0.8	0.55
Size of largest collection on CT	10.3±4.34cm	11.15±3.42cm	0.81
CTSI Score	7.3±2.5	7.8±2.5	0.65
mCTSI Score	8.7±1.7	9.2±1.3	0.60
Day of PCD placement	15.1±4.9	20.5±3.9	**0.02**
Size of largest PCD in the French scale	16.2±3.1	14.7±4.2	0.28

S101

Immediate Etiology of Incidental Finding of Dilated Common Bile Duct on Abdominal Images—A Preliminary Study

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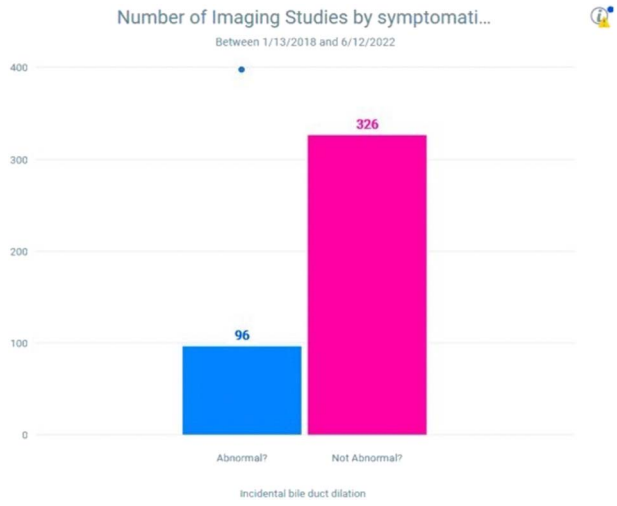
Introduction: Dilation of the common bile duct is frequently noted on computed tomography, ultrasound, & magnetic resonance imaging scans; however, this abnormality is incidentally found in patients without any biliary symptomatology or clinical concern for an obstructing process. The etiology of this abnormality in these patients is not well established.
Methods: In this retrospective analysis, consecutive patients who underwent a CT abdomen, Ultrasound of the abdomen, MRCP, & MRI of the abdomen at a tertiary care health care center from 2018 to 2022 with incidental finding of biliary duct dilation on imaging were retrieved in our radiology department records. We evaluated the immediate etiology which could explain bile duct dilation in patients without an obstructing process versus this abnormality in patients with an obstructing process. (Figure)
Results: In total, 422 patients were identified. Patients with an immediate explanation of bile duct dilation (i.e., prior Cholecystectomy, opioid use, or potential obstructing lesions by elevated total bilirubin) were found in 22.7% (96) of 422. Alternatively, patients without an immediate etiology to explain the bile duct dilation were 77.25% (326) of 422, which makes the investigation statistically significant ($P < 0.005$). Those patients had repeat abdominal images in 3 to 6 months to further identify non-immediate etiology, we will report those findings separately.
Conclusion: Dilation of CBD on computed tomography, ultrasound, & magnetic resonance imaging is suggestive that the etiology of these findings is clinically significant with about 1/4 of patients with an immediate etiology including prior cholecystectomy, nacortic medication use, etc. Most patients do not have an immediate etiology and merit further abdominal images for follow up.



[0101] **Figure 1.** Bile duct dilation in patients without an obstructing process versus this abnormality in patients with an obstructing process.

S102

Percutaneous Cholangioscopy With the Novel Short Cholangioscope for the Management of Biliary Disease

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Introduction: Cholangioscopy is indicated for the diagnosis and treatment of biliary strictures and complex choledocholithiasis but is often precluded in patients with altered anatomy. Percutaneous cholangioscopy with the novel short cholangioscope (SpyGlass Discover, Boston Scientific, Natick, MA) can circumvent this limitation. Its performance has not been systematically evaluated.
Methods: We performed a retrospective review of percutaneous cholangioscopies performed between January 2021 and February 2022 at UCDMC and Sutter Health Medical Center. The primary endpoint was procedural success defined as differentiation of the etiology of biliary stricture, and clearance of choledocholithiasis. We collected the following data - age, sex, BMI, procedure indication, reason for preclusion or failure of ERCP, the number of percutaneous biliary drains and cholangioscopes used, procedure duration, complications, and overall clinical impact.
Results: Thirteen patients were studied. Average age: 59.7 years. Eight were male. Primary indications: proximal stent migration (1), acute calculous cholecystitis (1), choledocholithiasis (5), extrahepatic biliary stricture (2), observed jaundice (1), cholelithiasis (3). ERCP was precluded in 3 patients due to perforated cholecystitis (with choledocholithiasis), necrotizing pancreatitis with a large paraesophageal hernia (with extrahepatic biliary stricture), and duodenal switch bariatric surgery (with biliary stricture). ERCP failed in 4 patients due to stent migration proximal to an iatrogenic biliary stricture, and roux-en-Y gastric bypass surgery. One patient needed 3 cholangioscopies, 4 patients needed two, and 8 needed one. Average number of drains: 4.2 (1 pre- and 3 post-procedure). Average procedure duration: 52.6 minutes (range 19.5 - 120 min) Stone clearance achieved: 2/3 cases with cholelithiasis, 4/5 with choledocholithiasis. Etiology of biliary stricture established: 2/2 cases (1 malignant, 1 benign). Post-op complications: post procedure leak (2), recurrent cholangitis (1), and skin site infection (1), without procedure related mortality.
Conclusion: Percutaneous cholangioscopy using the novel short cholangioscope successfully and safely managed choledocholithiasis and biliary strictures in patients with surgically altered anatomy that precluded ERCP or rendered it unsuccessful, and helped avoid long-term percutaneous cholecystostomy drains by treating cholelithiasis in patients unable to undergo cholecystectomy. Larger multi-center studies are awaited.

S103

Choleperitonitis: A Relatively Rare but Potentially Fatal Entity Encountered in the U.S. Hospitals

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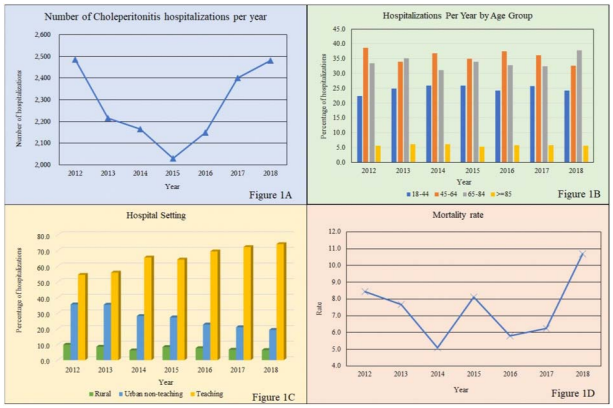
Introduction: Choleperitonitis is defined as inflammation of the peritoneum caused by the escape of bile into the peritoneal cavity. This may be secondary to pathologic or iatrogenic rupture of the gallbladder and/or the biliary system. Limited epidemiological data exist on the number of hospitalizations, demographic variation, cost of care, comorbidity measures, and outcomes for Choleperitonitis.
Methods: We analyzed the National Inpatient Sample (NIS) database for all hospitalizations with Choleperitonitis (ICD-9 code 567.81 and ICD-10 code K65.3 as applicable) as primary or secondary diagnosis during the period from 2012-2018. NIS is the largest all-payer inpatient care database in the United States. Statistical significance of variation in the number of hospitalizations, demographic disparity, cost of care, comorbidity measures, and outcomes during the study period were determined using Cochran-Armitage trend test.
Results: Between 2012 and 2018, the number of hospitalizations for Choleperitonitis ranged from from 2,030 to 2,485 (p=0.04, Figure 1A). Hospitalizations with Choleperitonitis were found to be more common in women (p=0.03) and Caucasians (p=0.001). Although age group 45-64 remained the most affected, there was an overall proportional decrease from 38.6% to 32.5% (p=0.0006, Figure 1B). Generally, South remained the most affected region (p=0.03) throughout the study period. There was a significant rise in the West from 24.1% to 29.2% (p=0.0002) with a concurrent decrease in the South from 37.2% to 31.3% (p=0.03). A proportional decrease in the number of hospitalizations was seen at both, urban non-teaching (35.6% to 19.4%, p< 0.0001, Figure 1C) and rural hospitals (9.9% to 6.5%, p< 0.0001, Figure 1C), while the number increased at urban teaching hospitals (54.5% to 74.2%, p< 0.0001, Figure 1C). Mean length of hospital stay for Choleperitonitis ranged from 12.3 ± 0.6 to 14.3 ± 0.9 days (p=0.07). Overall mortality ranged from 5.1% to 10.7% (p=0.05, Figure 1D). Some of the most associated comorbid conditions with Choleperitonitis were fluid and electrolyte disorders, hypertension, weight loss, deficiency anemias, and obesity.
Conclusion: During the study period, the annual number of hospitalizations with Choleperitonitis largely remained stable with interesting demographic variations and association with comorbidities. Further studies are needed to identify factors responsible for such trends to better elucidate our findings.



[0103] **Figure 1.** A) Number of Choleperitonitis hospitalizations per year, B) Hospitalizations per year by age group, C) Hospital Setting, D)Mortality Rate

S104

Outcomes After Anticoagulation for Patients With Splanchnic Vein Thrombosis Due to Acute Pancreatitis

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Introduction: Acute pancreatitis (AP) is an inflammatory process with a wide variety of clinical manifestations and sequelae. Splanchnic vein thrombosis (SVT), defined as thrombosis of the portal vein, mesenteric veins, or splenic vein, can be seen in a range of intra-abdominal inflammatory processes including AP, as well as in malignancies, inherited hypercoagulable states, and most commonly cirrhosis. There is a paucity of data regarding outcomes of SVT due to AP. The aim of this study is to review clinical outcomes of patients with mesenteric or splenic vein thrombosis due to AP who received therapeutic anticoagulation compared to those who did not receive anticoagulation.
Methods: We performed a retrospective chart review of patients who were diagnosed with splenic or mesenteric vein thrombosis in the setting of AP at our center from 2008-2021. A total of 395 patients were identified. 255 patients with portal vein thrombosis were excluded. A further 19 patients with other indications for anticoagulation (e.g., atrial fibrillation, mechanical valve, DVT/PE) were excluded. Age, sex, location of thrombosis, and initiation of anticoagulation were recorded. Categorical variables of anticoagulation and no anticoagulation were compared with incidence of death, blood transfusion, EGD and colonoscopy in one year using the Chi-Square test.
Results: A total of 121 patients identified as having splenic or mesenteric vein thrombosis in setting of AP without any other indication for anticoagulation. 68 were placed on anticoagulation while 53 were not. There was statistically significant association for incidence of EGD and colonoscopy in one year in the anticoagulation group compared to the group on no anticoagulation. While the anticoagulation group was associated with increased rate of need for transfusion (35% vs 26%) and death (10% vs 4%) compared to the group on no anticoagulation, these findings were not statistically significant. (Table)
Conclusion: Use of anticoagulation solely for splenic or mesenteric vein thrombosis in setting of AP was associated with increased use of endoscopy in one year compared to those not started on anticoagulation. While increased mortality at one year and need for blood transfusion was seen in those started on anticoagulation, this was not statically significant. Further investigation including larger sample size and

prospective data collection to evaluate is needed.

Table 1.

	EGD in one year	Colonoscopy in one year	Transfusion in one year	Mortality in one year
Anticoagulation (N=68)	44	17	24	7
No anticoagulation (N=53)	23	5	14	2
	p=0.0193	**p=0.0276**	p=0.296	p=0.175

S105

Acute Pancreatitis in a Los Angeles Safety Net Hospital System: Patient Characteristics and Predictors of Readmission

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Introduction: Acute pancreatitis (AP) is the most common gastrointestinal cause for hospital admission in the United States and may disproportionately affect minority populations. We aimed to characterize patients with AP and identify predictors of readmission in hospitals belonging to the Los Angeles County Department of Health Services, a large safety net hospital system.
Methods: A database of patient encounters at Olive View-UCLA, Harbor-UCLA, and LAC+USC Medical Centers was queried for patients seen in 2017 with an elevated lipase or diagnosis matching ICD-10-AM codes K85.0, K85.1, K85.2, K85.3, K85.8, or K85.9. Patients were included in the study if they met at least 2 of the following 3 criteria: 1) lipase >3x the upper limit of normal, 2) radiographic findings suggestive of AP, or 3) abdominal pain. Patients were excluded if radiographic evidence of chronic pancreatitis was present. Outcomes included overall length of stay (LOS), mortality due to AP during the index admission, and readmission for AP or AP-related complications. A multiple logistic regression model was used to identify predictors of readmission for AP.
Results: Six hundred and twenty-three patients with median age 45 years, 83% Hispanic, and 51% male met inclusion criteria for the study. The most common AP etiology was gallstones, seen in 251 patients (40%), followed by alcohol, seen in 144 (23%). Radiographic evidence of acute interstitial edematous (IEP) and necrotizing pancreatitis (NP) was seen in 61% and 7% of patients, respectively. Greater than half of all patients with IEP developed an acute peripancreatic fluid collection, while 48% and 39% of patients with NP developed an acute necrotic collection or walled-off necrosis, respectively. The median LOS was 4 days (IQR 2 – 6). Five patients (1%) died during the index admission due to an AP-related complication, while 48 (8%) had a readmission for AP at a median of 91 days (IQR 15 – 178) from discharge. Increasing age (OR = 0.97, P = 0.038) was associated with decreased odds of readmission for AP when controlling for sex, BMI, and presence of any local complication on imaging. (Table)
Conclusion: In our safety net patient population, gallstones and alcohol were the most common AP etiologies and increasing age at admission was associated with decreased odds of readmission for AP, mirroring findings reported in the literature. Our mortality rate was comparatively low at just 1%, however this may be because we did not limit our study cohort to patients with a primary diagnosis of AP.

Table 1. Predictors of readmission for AP modeled using multiple logistic regression

Predictor	Odds Ratio	Std. Err.	P-Value	95% Confidence Interval
Male	1.578	0.634	0.256	0.718 – 3.470
Age	0.972	0.013	0.038	0.947 – 0.998
BMI	1.009	0.021	0.678	0.968 – 1.050
Presence of local complications	1.560	0.618	0.261	0.718 – 3.391

S106

Management of Post-Cholecystectomy Bile Leaks: Predictors for Persistent Leak After Initial ERCP

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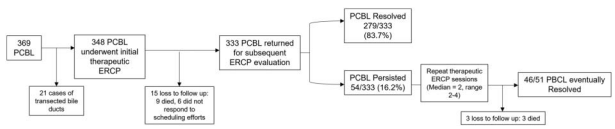
Introduction: Endoscopic retrograde cholangiopancreatography (ERCP) is a 1st-line treatment for post-cholecystectomy bile leaks (PCBL). Despite appropriate initial ERCP interventions that optimize transpapillary bile flow, PCBL can persist. We aim to evaluate baseline clinical factors associated with persistent PCBL after initial ERCP.
Methods: We created a retrospective database of patients with PCBL referred for ERCP at Indiana University Health (IUH) University Hospital. Data collected included endoscopic reports, fluoroscopic imaging, patient demographics, type of PCBL, ERCP timing and interventions, technical success, and treatment outcomes. A high-grade bile leak (HG-BL) was defined as visualization of contrast extravasation from the bile duct before filling of intrahepatic biliary branches with contrast. The PCBL was deemed to be persistent if > 1 interventional ERCP was required for the leak to resolve on cholangiogram. (Figure)
Results: From 2011 to 2021, 369 cases of PCBL were referred for ERCP. After excluding cases with transected bile ducts (n=21) and patients lost to follow up (n=15), 333 cases were included in data analysis (Image 1). 21 patients received their initial ERCP at an outside hospital. All patients received biliary sphincterotomy with stenting unless there was coagulopathy or Roux-en-Y gastric bypass (n=12). Univariate logistic regression analysis identified male gender, steroids use at time of ERCP, presence of duodenal stricture, a HG-BL, Strasberg class D PCBL, presence of biloma, presence of abdominal drain, presence of biliary stricture, and initial ERCP performed at outside hospital to be significant variables for persistent PCBL (Table). On multivariate analysis, presence of a HG-BL (OR 7.08, CI 1.96 – 25.58, p = 0.003) and initial ERCP performed at an outside facility (OR 29.14, CI 3.35 – 253.63, p=0.002) remained significantly associated with persistent PCBL. Being a female gender (OR 0.30, CI 0.11 – 0.85, P = 0.022) remained significant for higher odds of PCBL resolution after initial ERCP.
Conclusion: Our study suggests that a HG-BL in a male patient may justify more aggressive ERCP interventions or more prolonged stent indwell time. As a tertiary referral hospital, we had a significant referral bias in our cohort, which could have acted as a confounder causing statistical significance for lower initial ERCP success rates at outside medical facilities. The relatively high volume of ERCP cases at our center may also have contributed to better ERCP outcomes.



[0106] **Figure 1.** Flowchart of the Study

Table 1. Univariate analysis of clinical variables for persistent PCBL after initial ERCP treatment

Variables	Initial ERCP failure	OR (95% CI)	P value
Patient Characteristics			
Female	25/204 (12.25%)	0.49 (0.27 – 0.86)	**0.013**
Age 55 years old or more	34/175 (19.43%)	1.60 (0.90-2.86)	0.112
Median BMI (IQR)	32.0 (26.0, 36.0)	1.00 (0.98 – 1.03)	0.874
On diabetic treatment	13/58 (22.41%)	1.81 (0.93 – 3.53)	0.083
On steroids	4/6 (66.67%)	10.18 (1.82 – 57.00)	**0.008**
Major papilla within duodenal diverticulum	11/57 (19.30%)	1.46 (0.73 – 2.93)	0.282
Duodenal stricture/stenosis	5/10 (50.00%)	5.17 (1.45 – 18.48)	**0.011**
Location and timing of ERCP Procedure			
ERCP performed > 3 days	48/273 (17.58%)	1.00 (0.47 – 2.12)	0.994
Initial ERCP performed at outside facility	17/21 (80.95%)	30.94 (9.96 -96.06)	< **0.001**
Bile leak characteristics			
Strasberg Class A bile leaks	44/302 (14.57%)	0.26 (0.12 – 0.54)	< **0.001**
Strasberg Class D bile leaks	10/31 (32.26%)	2.70 (1.19 – 6.12)	**0.018**
Bile leak from cystic duct*	34/192 (17.71%)	1.25 (0.70 – 2.24)	0.446
Bile leak from duct of Luschka	4/104 (3.85%)	0.21 (0.09 – 0.50)	< **0.001**
Bile leak from common bile duct/common hepatic duct	7/25 (28.00%)	2.66 (1.13 – 6.27)	**0.025**
High-grade bile leak	25/63 (39.68%)	4.80 (2.58 – 8.95)	< **0.001**
Concomitant biloma present	27/115 (23.48%)	2.06 (1.13 – 3.76)	**0.018**
Percutaneous abdominal drain present	42/204 (20.59%)	2.02 (1.07 – 3.82)	**0.030**
Presence of biliary stones	5/67 (7.46%)	0.34 (0.13 – 0.88)	**0.026**
Presence of biliary stricture	7/21 (33.33%)	2.63 (1.01 – 6.84)	**0.048**
ERCP interventions			
Biliary sphincterotomy performed	56/325 (17.23%)	1.04 (0.22 – 4.88)	0.960
Biliary sphincterotomy alone	2/6 (33.33%)	2.46 (0.44 – 13.73)	0.306
Biliary stenting alone	2/12 (16.67%)	0.96 (0.21 – 4.51)	0.960
Bridging biliary stents	20/134 (14.93%)	0.78 (0.43 – 1.43)	0.426
Multiple biliary plastic stents	6/55 (10.91%)	0.55 (0.22 – 1.36)	0.198
Self-expandable metal stent	2/11 (18.18%)	1.09 (0.23 – 5.21)	0.910

*Reference group. IQR, interquartile range; IUH, Indiana University Health.

S107

More Than Just an Itch: Impact of Cholestatic Pruritus in Primary Biliary Cholangitis (PBC) on Health-Related Quality of Life (HRQoL)

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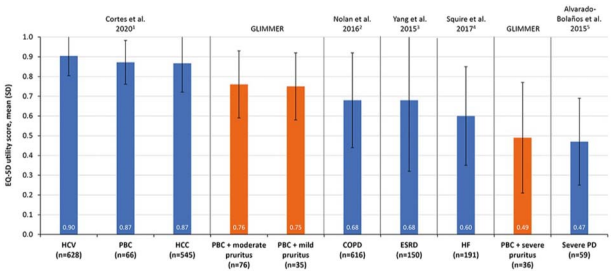
Introduction: Pruritus associated with PBC affects sleep and social and emotional wellbeing. Limited data exist on the impact of pruritus on health utility (a value between 0 [death] and 1 [perfect health]), commonly used in health technology assessments to calculate quality-adjusted life years and compare different conditions. A recent UK study explored EQ-5D utilities in a broad PBC population.[1] Here, using data from the Phase 2b GLIMMER study (post hoc) investigating linerixibat for the treatment of pruritus in PBC (NCT02966834), the impact of itch severity on health utility in PBC is explored and quantified for the first time.
Methods: Patients in GLIMMER recorded itch twice daily on a 0–10 numeric rating scale (NRS) and completed the EQ-5D-5L at study entry, baseline (BL) and end of treatment. BL followed a 4-week single-blind placebo run-in. Patients were classed as having mild (< 4), moderate (≥4 to < 7) or severe pruritus (≥7 to 10) based on mean Worst Daily Itch NRS score in the 7 days prior to BL.
Results: The GLIMMER population (N=147) was 94% female with a mean (SD) age at BL of 55.8 (11.04) years. Most patients had moderate pruritus (n=76), with similar numbers with mild (n=35) and severe (n=36) pruritus. At BL, alkaline phosphatase levels were higher with greater itch severity: mean (SD) 177 (115.4) and 249 (190.8) IU/L in patients with mild and severe itch, respectively. Overall, mean (SD) BL utility was 0.69 (0.23), lower than the general PBC population (**Figure**) and with a clear and notable impact of pruritus severity on health utility. Thus, patients with mild or moderate pruritus at BL had similar utilities (0.75 [0.17] and 0.76 [0.17], respectively), marginally lower than the general UK population (mean at age 55–64 years: 0.804). Patients with severe pruritus at BL had notably worse utility (0.49 [0.28]), similar to patients with severe Parkinson's disease (0.47 [0.22]; **Figure**). Over the course of the study health utility declined in the placebo group (–0.01) and increased across all linerixibat arms (0.04–0.05). Although improvements were small (confidence intervals crossed zero), the directional change supports a treatment effect of linerixibat.
Conclusion: Pruritus (particularly severe pruritus) has a significant negative impact on HRQoL and health utility. Presence and severity of itch should be evaluated in PBC and prioritized in treatment plans.



[0107] **Figure 1.** Baseline utility scores in PBS patients with cholestatic pruritis (GLIMMER) compared with those seen in other chronic conditions. 1. Cortesi PA, et al. Liver International 2020;40: 2630–42; 2. Nolan CM, et al. Thorax 2016;71:493–500; 3. Yang, F et al. Eur J Health Econ 2015;16:1019–26; 4. Squire I, et al. Br J Cardiol 2017;24:30–4; 5. Alvarado-Bolaños A, et al., J Neurol Sci

2015;358:53–7. COPD, chronic obstructive pulmonary disease; ESRD, end-stage renal disease; HCC, hepatocellular carcinoma; HCV, hepatitis C; HF, heart failure; PBC, primary biliary cholangitis; PD, Parkinson's disease.

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S108

Benefits of Early Cholecystectomy in Patients Presenting With Acute Gallstone Cholangitis Following ERCP: A Systematic Review and Meta-Analysis

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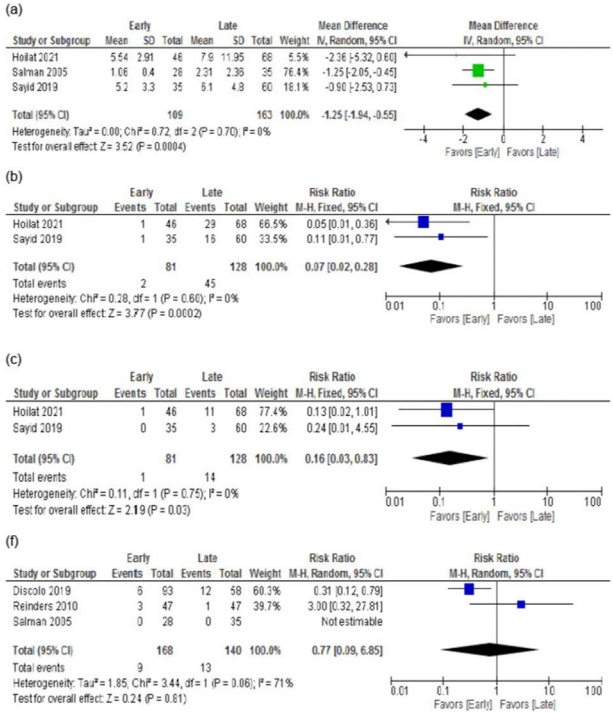
Introduction: The mainstay of management of acute gallstone cholangitis includes intravenous fluid resuscitation, appropriate antibiotic coverage, biliary drainage, and subsequent cholecystectomy. Even though cholecystectomy following an episode of gallstone associated cholangitis is strongly supported in the literature, the exact timing of cholecystectomy remains uncertain. Delay in cholecystectomy after the diagnosis of acute cholangitis may increase the risk of recurrent emergency department visits and hospital admissions. This systematic review aims to evaluate and compare the advantages, safety and efficacy of early cholecystectomy in patients with acute cholangitis.
Methods: A systematic review and meta-analysis was carried out according to the PRISMA and Cochrane Handbook for Systematic Review of Interventions. Six databases (PubMed, Scopus, Embase, Google Scholar, Web of Science, and Cochrane Central Register of Controlled Trials) were screened from inception until 01-May-2022. The inclusion criteria comprised all randomized controlled trials (RCTs) and nonrandomized comparative trials (NCTs) that evaluated early versus late cholecystectomy among patients with acute cholangitis. The efficacy outcomes were summarized as mean difference (MD) or risk ratio (RR) with 95% confidence interval (CI).
Results: Six studies met the inclusion criteria, comprising a total of 604 patients (289 and 315 patients were allocated to the early and late cholecystectomy groups, respectively). The mean length of hospital stay (MD=-1.25 d, 95% CI [-1.94, -0.55], p< 0.001), rate of readmission within 30 days (RR=0.07, 95% CI [0.02, 0.28], p< 0.001), rate of readmission due to a biliary cause (RR=0.06, 95% CI [0.02, 0.18], p< 0.001), and rate of death within 30 days (RR=0.16, 95% CI [0.03, 0.83], p=0.03) were significantly reduced in favor of the early cholecystectomy group compared with the late cholecystectomy group. (Figure)
Conclusion: Performing cholecystectomy during the same admission for acute gallstone cholangitis was associated with a lower 30-day readmission rate and 30-day mortality rate. Additionally, there was no increase in operative time or the incidence of post-operative complications.



[0108] **Figure 1.** Meta-analysis of the efficacy endpoints: (a) mean length of hospital stay, (b) rate of readmission within 30 days, (c) rate of death within 30 days, (d) rate of postoperative complications.

S109

Clinical Predictors of Ileus in Hospitalized Patients With Acute Pancreatitis: A Nationwide Analysis

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Introduction: Ileus is a well-known complication of acute pancreatitis (AP). Limited data exists on incidence or factors associated with ileus in patients with AP. We aimed to investigate the incidence and clinical predictors of ileus in hospitalized patients with AP.
Methods: We queried the 2016-2019 National Inpatient Sample (NIS) database using the ICD-10 codes. All adult patients with a diagnosis of AP (ICD-10 K85) were included and those with chronic pancreatitis were excluded from the analysis. We studied demographics, comorbidities, complications, and interventions in patients with AP and stratified the results by the presence of ileus. Multivariate analysis was conducted to elucidate clinical factors associated with ileus formation in patients with acute pancreatitis. We adjusted for patient demographics, hospital characteristics, elixhauser comorbidities and common complications of pancreatitis.
Results: Of the 1,386,389 adult patients admitted with AP, 50,170 (3.6%) developed ileus. Female gender was associated with a 44% lower risk of ileus (aOR- 0.56, 95% CI- 0.53-0.58, p< 0.001). Hispanic patients had the lowest risk of ileus (aOR- 0.82, 95% CI- 0.76-0.88, p< 0.001) while White patients had the highest risk. Patients between the age of 18-44 had a lower risk of ileus as compared to patients in the 45-64 (aOR-1.11, 95% CI- 1.05-1.17, p< 0.001) and >65 age group (aOR-1.14, 95% CI- 1.06-1.24, p< 0.001). Other factors associated with ileus include the presence of pseudocyst (aOR- 1.52, p< 0.001), sepsis (aOR-1.72, p< 0.001), and portal vein thrombosis (aOR-1.36, p< 0.001). ERCP was not associated with ileus development, however, pancreatic drainage was associated with a higher risk of ileus (aOR-1.21, p< 0.007).

The results of the multivariate analysis are depicted in Table: Patients with ileus were also noted to have a statistically significant higher mortality (a0R-1.58, 95% CI- 1.43-1.75, p< 0.001), length of stay (+4.9 days, 95% CI- 4.63-5.12, p< 0.001), total hospitalization cost (+67,855.91, p< 0.001) and charges ($16,252.6, p< 0.001).

Conclusion: This study highlights age, gender, and racial disparities in the development of ileus in patients with AP. It also reveals a significant association of ileus with pseudocyst, portal vein thrombosis, and pancreatic drainage. Timing of initiation of oral feeds is essential in patients with acute pancreatitis and ileus. Physicians should be aware of this high-risk group and consider early enteral feeding to prevent disease progression.

Table 1. Predictors of ileus in patients with acute pancreatitis. In this Table, we provide the results of multivariate logistic regression with odds ratio, p-value and 95% confidence interval

Ileus	Odds ratio	p-value	95% Confidence interval
Age category			
18-44	Reference		
45-64	1.11	< 0.001	1.05-1.17
>65	1.14	< 0.001	1.06-1.24
Sex			
Male	Reference		
Female	0.56	< 0.001	0.54-0.59
Race			
White	Reference		
African American	0.95	0.149	0.89-1.02
Hispanic	0.82	< 0.001	0.76-0.88
Asian/Pacific Islander	1.01	0.846	0.89-1.15
Native American	0.72	0.01	0.56-0.93
Primary expected payer			
Medicare	Reference		
Medicaid	0.83	< 0.001	0.77-0.9
Private	1.11	< 0.001	1.04-1.18
Uninsured	0.8	< 0.001	0.72-0.88
Income quartile			
Lowest quartile	Reference		
Second quartile	1.04	0.227	0.98-1.1
Third quartile	1.11	< 0.001	1.04-1.17
Highest quartile	1.14	< 0.001	1.07-1.22
Bed size of Hospital			
Small	Reference		
Medium	1.04	0.224	0.98-1.11
Large	1.11	< 0.001	1.04-1.17
Region of hospital			
Northeast	Reference		
Midwest	1.31	< 0.001	1.21-1.42
South	1.16	< 0.001	1.08-1.25
West	1.17	< 0.001	1.08-1.26
Location of hospital			
Rural hospitals	Reference		
Urban hospitals	1.03	0.502	0.94-1.13
Teaching status of hospitals			
Non-teaching hospitals	Reference		
Teaching Hospitals	1.05	0.084	0.99-1.1
Etiology of pancreatitis			
Biliary pancreatitis	Reference		
Alcohol pancreatits	0.98	0.583	0.89-1.17
Biliary + alcohol pancreatitis	1.81	< 0.001	1.32-2.47
Other causes	0.88	< 0.001	0.84-0.93
Number of elixhauser comorbidities			
0	Reference		
1	1.26	< 0.001	1.11-1.43
2	1.41	< 0.001	1.24-1.6
3 or more	1.54	< 0.001	1.35-1.76
Comorbidities			
Cardiac arrhythmias	0.86	< 0.001	0.8-0.93
Congestive heart failure	1.26	< 0.001	1.19-1.33
Valvular disease	0.97	0.587	0.86-1.09
Pulmonary circulation disorders	0.96	0.558	0.85-1.09

Table 1. (*continued*)

Ileus	Odds ratio	p-value	95% Confidence interval
Peripheral vascular disorders	0.95	0.279	0.87-1.04
Paralysis	1.32	0.009	1.07-1.62
Other neurological disorders	1.01	0.83	0.94-1.08
Chronic pulmonary disease	1.01	0.704	0.95-1.07
Chronic kidney disease	0.83	< 0.001	0.78-0.89
Liver disease	1.01	0.637	0.96-1.07
Metastatic solid tumor	0.84	0.036	0.72-0.99
Non-metastatic solid tumor	1.05	0.407	0.93-1.19
Rheumatoid arthritis/collagen vascular disorder	0.87	0.054	0.76-1
Coagulopathy	0.98	0.647	0.92-1.05
Malnutrition	1.32	< 0.001	1.24-1.41
Fluid/electrolyte disorders	1.59	< 0.001	1.51-1.67
Blood loss anemia	1.17	0.146	0.95-1.45
Deficiency Anemia	0.93	0.152	0.84-1.03
Alcohol abuse	0.73	< 0.001	0.68-0.78
Drug use	0.88	0.003	0.81-0.96
Psychosis	0.97	0.721	0.8-1.17
Depression	0.87	< 0.001	0.81-0.92
Diabetes	0.71	< 0.001	0.68-0.75
Hypertension	0.98	0.348	0.93-1.03
Complications			
Pneumonia	1.63	< 0.001	1.52-1.76
Bacteremia	0.97	0.787	0.76-1.23
Acute kidney injury	1.38	< 0.001	1.31-1.46
Portal vein thrombosis	1.41	< 0.001	1.2-1.66
Pseudocyst	1.57	< 0.001	1.44-1.72
Sepsis	1.75	< 0.001	1.63-1.89
Shock	1.18	< 0.001	1.08-1.29
ICU admission	1.99	< 0.001	1.83-2.17
Interventions			
TPN	4.22	< 0.001	3.84-4.64
ERCP	1.05	0.203	0.97-1.13
Pancreatic drainage	1.25	0.009	1.06-1.48

S110

Racial Differences in the Performance of Endoscopic Retrograde Cholangiopancreatography (ERCP) in Acute Cholangitis: A Nationwide Time Trend Analysis, 2008-2018

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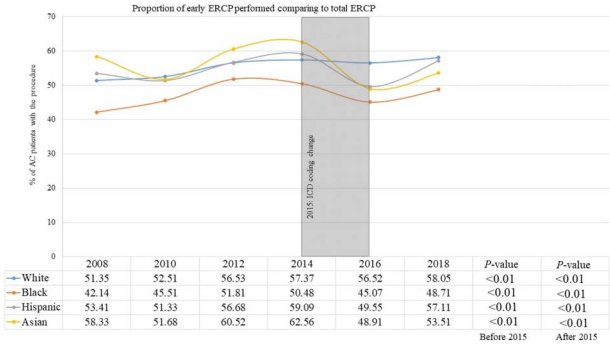
Introduction: Black race was shown to have high mortality from acute cholangitis (AC) previously (1998-2009) and to undergo less endoscopic retrograde cholangiopancreatography (ERCP) (2009-2012). We conducted a longitudinal racial breakdown of ERCP performed in AC in the USA over 11 years (2008-2018).
Methods: This is a retrospective longitudinal trend analysis using National Inpatient Sample. Adult patients (≥18 years old) with AC were identified. Multivariate linear or logistic regression was performed when appropriate. To control for the severity of AC, we also included severe sepsis, septic shock, systemic inflammatory response syndrome with acute organ dysfunction, acute renal failure, acute respiratory failure, thrombocytopenia, altered mental status, and abnormal coagulation in the regression model. Stata (v.14.2) was used to perform analyses considering a 2-sided *P*-value< 0.05 as statistically significant.
Results: A total of 312,849 patients with AC were included in the analysis. Before 2015, the longitudinal trend for the overall total and early ERCPs performed in Whites and Hispanics was increasing ($P< 0.01$) (Table). The trend was stable in Asians but in Blacks, even though the trend for early ERCP was increasing ($P< 0.01$) but the overall ERCPs performed for AC remained the same ($P=0.07$). After 2015, the trends for both early and total ERCPs remained stable for all races ($P >0.05$). However, upon examining the proportion of early ERCPs among total ERCPs performed, Blacks represented the racial category with the lowest numbers (Figure). Even though the trend of proportions for all racial categories was increasing ($P< 0.01$) however, the rate of increase was lowest for Blacks (per year increase for Whites was 1.37%, Blacks 1.18%, Hispanics 1.47%, and 1.37 for Asians). Racial mortality comparison showed that compared to Whites, Blacks had the highest odds of mortality (for Blacks, adjusted odds ratio (aOR) 1.86, $P< 0.01$, Hispanics aOR 1.29, $P< 0.01$, Asians aOR 1.30, $P=0.01$).
Conclusion: The modification in International Classification of Diseases (ICD) coding in 2015 resulted in an apparent sharp change in the proportion of ERCPs performed due to coding change. Even though the trend for the performance of early ERCP for AC is on the rise in all races, there still exists a racial disparity in the use of early ERCP. The black population was at risk of receiving lower rates of early ERCP (calculated as a fraction of total ERCP), which may impart higher mortality.



	2008	2010	2012	2014	2016	2018	*P*-value	*P*-value
White	51.35	52.51	56.53	57.37	56.52	58.05	<0.01	<0.01
Black	42.14	45.51	51.81	50.48	45.07	48.71	<0.01	<0.01
Hispanic	53.41	51.33	56.68	59.09	49.55	57.11	<0.01	<0.01
Asian	58.33	51.68	60.52	62.56	48.91	53.51	<0.01	<0.01

[0110] **Figure 1.** Trend for the proportion of early ERCP comparing to total ERCP (ERCP: endoscopic retrograde cholangiopancreatography, AC: acute cholangitis, %: percentage, ICD: International Classification of Diseases)

Table 1. Trends for early and total ERCP performance in acute cholangitis

		Year							
		2008	2010	2012	2014	*P* value[a,b]	2016	2018	*P* value[a,b]
White	Total ERCP, %	47.58	48.75	50.21	50.55	< 0.01	33.42	32.55	0.37
	Early ERCP, %	24.43	25.60	28.38	29.01	< 0.01	18.89	18.90	0.99
Black	Total ERCP, %	41.20	39.75	43.59	43.45	0.07	30.97	30.21	0.72
	Early ERCP, %	17.34	18.09	22.58	21.93	< 0.01	13.96	14.72	0.66
Hispanic	Total ERCP, %	53.87	53.85	56.36	58.14	< 0.01	35.61	35.51	0.96
	Early ERCP, %	28.70	27.64	31.94	34.36	< 0.01	17.64	20.28	0.13
Asians	Total ERCP, %	54.34	52.86	55.28	53.04	0.84	35.08	37.58	0.39
	Early ERCP, %	31.69	27.32	33.45	33.18	0.31	17.16	22.33	0.27

ERCP: endoscopic retrograde cholangiopancreatography.
[a]Linear *P* trend values.
[b]Time-interrupted trends (before and after 2015) were obtained due to International Classification of Diseases (ICD) coding change from ICD-9 to ICD-10 in 2015.

S111

Outcomes of Endoscopic Retrograde Cholangiopancreatography in Patients With Situs Inversus Viscerum

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Introduction: Situs inversus (SIV) is a rare congenital condition defined by the left to right transposition of all viscera as a mirror image. This uncommon anatomic variant has been reported to cause technical challenges while performing endoscopic retrograde cholangiopancreatography (ERCP). Data of patients with situs inversus undergoing ERCP is limited to case reports and the clinical and technical success rate of ERCP in these patients is unknown. The goal of our study is to evaluate the clinical and technical success rates and procedural characteristics of patients with situs inversus undergoing ERCP.
Methods: A retrospective review of data from consecutive patients with SIV undergoing ERCP. Data was collected by querying the nationwide Veterans Affairs Health System database for patients with a diagnosis of situs inversus (ICD Q89.3) who underwent ERCP between January 1, 2010, to March 31, 2022. Patient demographic and procedure characteristics were collected (Table).
Results: A total of 654 patients with situs inversus were identified. Eight of these patients had undergone ERCP. The median age was age of 68. The most common indication for ERCP was choledocholithiasis (5 patients, 62.5%). The technical success rate (defined as ability to achieve successful biliary cannulation) was 63%. Subsequent ERCP with interventional radiology assisted rendezvous increased the technical success rate to 88%. Clinical success (defined as successful management of choledocholithiasis, cholangitis, stricture, or obtaining specimens for the diagnosis of malignancy) was achieved in 63% of cases. When considering subsequent rendezvous ERCP if conventional ERCP failed, clinical success was achieved in 88% of cases.
Conclusion: In our limited series, the clinical and technical success rate of ERCP for patients with SIV is higher than previously reported. In patients with SIV where conventional ERCP biliary cannulation fails, IR assisted rendezvous ERCP can be considered.

Table 1. Patient demographic and procedure characteristics

Gender	Race	Age	Indication	Technical Success	Clinical Success	Reason for failure	Rendezvous ERCP attempted	Rendezvous ERCP technically successful	Rendezvous ERCP clinically successful
Male	White	72	Choledocholithiasis	Yes	Yes				
Male	African American	74	Pancreatic adenocarcinoma	Yes	Yes				
Male	White	88	Choledocholithiasis with cholangitis	No	No	Unable to cannulate the ampulla	Yes	Yes	Yes
Male	White	62	Periampullary Adenocarcinoma	Yes	Yes				
Male	White	59	Choledocholithiasis	No	No	Unable to cannulate the ampulla	No		
Male	White	88	Choledocholithiasis	Yes	Yes				
Female	Native American	63	Benign biliary stricture	Yes	Yes				
Male	Hispanic	63	Choledocholithiasis with cholangitis	No	No	Unable to visualize the ampulla	Yes	Yes	Yes

S112

Implications of Butyrate Kinase and Intestinal Microbiota on Diabetes Risk in a Community Enriched With Native Hawaiians

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Introduction: The incidence of type 2 diabetes mellitus (T2DM) within the Native Hawaiian population among the highest of any major ethnic group within the state of Hawaii. Modern research is beginning to mechanistically understand ancient Hawaiian concepts and practices demonstrating the importance of environment to human health. Environmental factors, such as socioeconomic status, toxicants, diet/nutrition, *etc*, contribute to individual risk of T2DM, where impacts on the gut microbiome may play key roles in inflammatory and metabolic dysregulation characteristic of diabetes. In this study, we focused on understanding the relationship between the gut microbiome and T2DM risk in the Native Hawaiian population.
Methods: The study enrolled a cohort of Hawaii residents (16 years or older) in a community enriched with Native Hawaiians. Microbial DNA was isolated from self-collected stool samples of all participants and used for 16S amplicon-based sequencing for metagenomic analyses and for quantifying microbial-specific butyrate kinase (BUK) gene expression levels using qPCR approaches. From each participant, anthropometric measures and blood levels of HbA1c, a diagnostic indicator of T2DM, were taken. Blood was collected by venous puncture from which plasma was isolated to measure inflammatory and metabolic biomarkers. Herein, we identified and describe significant differences between diabetic and non-diabetic microbiome composition and diversity.
Results: Consistent with other studies, we observed increased levels of CRP ($p< 0.01$) and decreased levels of leptin ($p=0.04$) in the plasma of diabetic compared to non-diabetic participants. Additionally, we observed that BUK levels were negatively correlated with that of HbA1c (R=-0.18, $p=0.04$), and positively correlated with that of metabolic hormones PYY (R=0.30; $p=0.007$) and GLP-1 (R=0.19; $p=0.04$), which were largely attributed to diabetes status. Higher plasma levels of PYY and GLP-1 have previously been associated with reduced T2DM risk.
Conclusion: Altogether, our data suggest a role for butyrate-producing gut bacteria in the maintenance of the homeostatic inflammatory and metabolic states that are dysregulated in the progression of T2DM and implicates novel targets for reducing T2DM risk among health disparate populations.

S113

Incidence and Clinical Predictors of Portal Vein Thrombosis (PVT) in Acute Pancreatitis: A Nationwide Analysis

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Introduction: Portal vein thrombosis (PVT) is a well-known complication in patients with acute pancreatitis (AP). Limited data exist on the incidence and factors associated with PVT in patients with AP. We investigate the incidence and clinical predictors of PVT in hospitalized patients with AP between 2016-2019.
Methods: We queried the 2016-2019 National Inpatient Sample (NIS) database using ICD-10 codes. All adult patients with a diagnosis of AP (ICD10 K85) were included, while patients with chronic pancreatitis were excluded from the analysis. We studied demographics, comorbidities, complications, and interventions in patients with acute pancreatitis and stratified the results by the presence of PVT. Multivariate analysis was conducted to elucidate factors associated with PVT in patients with AP. We adjusted for patient demographics, hospital characteristics, elixhauser comorbidities, and common complications of pancreatitis. We also studied the effect of PVT on length of stay, total hospitalization charge and mortality.
Results: Of the 1,386,389 adult patients admitted with AP, 11,135 (0.8%) patients had PVT. A complete list of patient characteristics is presented in Table: Female gender was associated with a 15.3% lower risk of developing PVT (aOR-0.847, p-< 0.001). There was no significant difference between the age groups (18-44, 45-64, and >65 years) on the risk of developing PVT. Hispanic patients had the lowest risk of PVT (aOR-0.74, p< 0.001) while White patients had the highest risk. PVT was associated with pancreatic pseudocyst (aOR-4.15, p< 0.001), bacteremia (aOR-2.66, p< 0.001), sepsis (aOR-1.55, p< 0.001), shock (aOR-1.69, p < 0.001) and ileus (aOR-1.38, p< 0.001). The presence of PVT was associated with a higher risk of mortality (aOR-1.35, 95% CI-1.09-1.66, p< 0.001), length of stay (+4.02 days, p< 0.001), and total hospitalization charge ($43,592.1, p< 0.001) as compared to patients without PVT.
Conclusion: Our retrospective analysis demonstrated that a significant association exists between portal vein thrombosis and factors such as pancreatic pseudocyst, bacteremia, and ileus in patients with AP. Thrombotic events are a known complication of AP and should be evaluated and managed diligently. Physicians should be aware of the factors associated with portal vein thrombosis as the presence of PVT portends to poor prognosis.wrap>

Table 1. Demographics and clinical predictors of portal vein thrombosis in patients with acute pancreatitis

Demographics	Absence of PVT	Presence of PVT	p-value
Age Category			< **0.001**
18-44	437,375 (31.8)	3,315 (29.77)	
45-64	531,215 (38.63)	4,980 (44.72)	
>65	406,665 (29.57)	2,840 (25.51)	
Sex			< **0.001**
Male	696,515 (50.65)	6,710 (60.26)	
Female	678,740 (49.35)	4,425 (39.74)	
Race			< **0.001**
White	878,314 (63.87)	7,755 (69.65)	
Black	204,035 (14.84)	1,375 (12.35)	
Hispanic	201,540 (14.65)	1,235 (11.09)	
Asian/Pacific Islander	36,030 (2.62)	290 (2.6)	
Primary Insurance			< **0.001**
Medicare	481,070 (34.98)	3,390 (30.44)	
Medicaid	303,850 (22.09)	2,400 (21.55)	
Private	425,575 (30.95)	4,015 (36.06)	
Uninsured	115,795 (8.42)	850 (7.63)	
Median Household Income			< **0.001**
Lowest quartile	438,215 (31.86)	2,895 (26)	
Second quartile	364,870 (26.53)	3,205 (28.78)	
Third quartile	325,255 (23.65)	2,710 (24.34)	
Highest quartile	246,915 (17.95)	2,325 (20.88)	
Hospital Location			< **0.001**
Rural	141,405 (10.28)	545 (4.89)	

Table 1. (*continued*)

Demographics	Absence of PVT	Presence of PVT	p-value
Urban	1,233,850 (89.72)	10,590 (95.11)	
Teaching Status			< **0.001**
Non-teaching hospitals	496,854 (36.13)	2,430 (21.82)	
Teaching hospitals	878,400 (63.87)	8,705 (78.18)	
Elixhauser Comorbidities			< **0.001**
0	103,800 (7.55)	335 (3.01)	
1	185,245 (13.47)	860 (7.72)	
2	242,405 (17.63)	1,530 (13.74)	
>3	843,805 (61.36)	8,410 (75.53)	
Etiology of Pancreatitis			< **0.001**
Biliary pancreatitis	319,730 (23.25)	1,755 (15.76)	
Alcohol related pancreatitis	263,505 (19.16)	2,465 (22.14)	
Biliary and alcohol related pancreatitis	3,190 (0.23)	45 (0.4)	
Other causes	788,830 (57.36)	6,870 (61.7)	
Complications			
Pneumonia	64,965 (4.72)	945 (8.49)	< **0.001**
UTI	113,240 (8.23)	830 (7.45)	**0.181**
Pseudocyst	46,030 (3.35)	2,125 (19.08)	< **0.001**
Acute kidney injury	257,985 (18.76)	3,130 (28.11)	< **0.001**
Bacteremia	8,310 (0.6)	230 (2.1)	< **0.001**
Sepsis	59,965 (4.36)	970 (8.71)	**0.031**
Interventions			
Pancreatic drainage	9,710 (0.71)	685 (6.15)	< **0.001**
Total parenteral nutrition	17,220 (1.25)	760 (6.83)	< **0.001**
ERCP	127,670 (9.28)	1,110 (9.97)	0.2648

S114

Patient Experience Analysis of an Online Community of Patients With Exocrine Pancreatic Insufficiency due to Chronic Pancreatitis Using a Patient-Centered Observational Approach

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Introduction: Exocrine pancreatic insufficiency (EPI) occurs in patients (pts) with a relative deficit of functional pancreatic enzymes, commonly secondary to chronic pancreatitis (CP). Pts with EPI typically experience maldigestion/malnutrition, negatively affecting quality of life (QoL). A two-pronged approach using a US-based registry for pts with EPI taking pancreatic enzyme replacement therapy and an online pt community with structured/unstructured data collection activities is being implemented to capture data/experiences to better appreciate the pt's lived experience, pt needs, and burden of illness. The goal of this study was to understand the pt's experience of CP with EPI and identify common themes discussed by members of the community.
Methods: A thematic analysis approach, utilizing HealthUnlocked CP community posts from 13JAN2021 to 02FEB2022, was used to identify/analyze common themes discussed by members.
Results: In total, 386 community posts were analyzed. Key topics were pain, diagnosis, treatment, and diet. Members frequently discussed CP-related pain impacting QoL. Members reported difficulty finding the source of and adequate treatment options for pain, often leading to prolonged pain. Sharing diagnosis experiences, members often found specialists the most helpful, actively seeking providers working to alleviate symptoms and not dismissive of concerns; some were frustrated with misconceptions from providers. Generally, members tried many treatment options before finding the right combination. Common treatment discussions included opioids, enzyme treatments, alternative medicine, other medications, diabetes management, and dietary changes; some found nontraditional treatments effective. Many members found CP-related pain and symptoms were greatly influenced by diet; changes often caused painful flare-ups. Members described CP symptoms were influenced by low fat diet, limiting alcohol consumption, vegan/vegetarian diets, herbal teas, and bland food.
Conclusion: This observational analysis of pt experiences of CP with EPI using a pt-driven online community determined key areas of focus including pain, diagnosis, treatment, and diet. Results highlight areas for practitioners to focus pt education to optimize experiences/care and QoL of pts with CP with EPI. Ongoing analysis of community discussions and comparisons of findings to registry data is practical/novel and will help develop a comprehensive understanding of the pt's lived experience.

S115

Clinical Characterization of the Silent Chronic Pancreatitis Patient

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Introduction: "Silent," or painless, chronic pancreatitis (CP) exists when patients with diagnostic features of CP describe no abdominal pain. It is a poorly understood phenomenon but it is important as it may go unnoticed until serious complications arise, including pancreatic insufficiency, diabetes, and even cancer. The aim of this study was to better characterize the silent CP patient and investigate potential risk factors associated with this condition.
Methods: We performed a retrospective analysis using data from a single tertiary care center between 2018-2021. 286 patients were identified using ICD-10 codes ("other chronic pancreatitis"-K86.1 and "exocrine pancreatic insufficiency"-K86.81). Two expert reviewers assessed each patient. Patients were included if they demonstrated feature(s) of CP including parenchymal (atrophy, calcifications +/- scarring) and/or ductal (stricture +/- dilation) changes on CT, MRI, or EUS imaging. Patients were excluded if their pain was attributed to a cause other than CP or if they underwent prior pancreaticobiliary surgery. Patients were categorized as having painful or silent CP, based upon documentation from at least two clinic visits. Patient demographics, CP etiology, presence of pancreatic duct stent & diabetes, substance use, and pain medication usage were abstracted. Descriptive statistics, bivariate and logistic regression analyses (utilizing variables with a $p < 0.1$ on bivariate analysis) were performed to characterize the study cohort and evaluate for independent associations with silent CP.
Results: 117 patients with CP were included in this study (mean age 57.2 years, 61.5% male). 23 patients (19.7%) had silent CP (Table). Patients with silent CP were older, more likely male, more likely diabetic, and less likely to use opioids or any pain medications. On multivariate analysis, older age (OR 1.06, 95% CI 1.01-1.11, p=0.03), male sex (OR 5.38, 95% CI 1.38-20.96, p=0.02), and opioid use (OR 0.18, 95% CI 0.03-0.96, p=0.04) were associated with silent CP.

Conclusion: Silent CP is relatively common and affected patients are older, more likely male, and less likely to use opioids. This suggests there may be inherent differences in visceral pain perception contributing to this clinical phenotype. Future studies should focus on evaluating larger cohorts longitudinally to confirm our findings and to more carefully investigate the underlying pathophysiology of silent CP, as well as to evaluate clinical outcomes of this condition.

Table 1. Multivariate Analysis for Association Between Clinical Characteristics and Silent vs Painful Chronic Pancreatitis

Clinical Characteristic	Odds Ratio	95% Confidence Interval		P Value
Age	1.060	1.010	1.110	0.027
Male Sex	5.376	1.383	20.964	0.015
Diabetes	2.430	0.774	7.610	0.128
Current Opioid Use	0.176	0.032	0.956	0.044
Any Current Pain Medication Use	0.860	0.241	23.070	0.816

S116

Early EUS in Choledocholithiasis: A Prospective Study

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Introduction: Choledocholithiasis (CDL) is a common gastrointestinal problem that can lead to life-threatening illness if left untreated. The American Society for Gastrointestinal Endoscopy (ASGE) revised its risk stratification tool for suspected CDL in 2019, in which endoscopic ultrasound (EUS) is reserved for intermediate risk patients. This study aims to evaluate the performance of an early EUS strategy in the diagnosis and management of CDL.
Methods: This is a prospective, single center study of 51 patients admitted with suspicion for CDL. Patients with upper abdominal pain, acute elevation in LFTs (>2x upper limit of normal), or transabdominal ultrasound or abdominal computed tomography (CT) were considered for the study. Exclusion criteria included clinical evidence of ascending cholangitis, history of post-surgical gastric or biliary anatomy, prior ERCP, or history of hepatobiliary malignancy. All suspected patients were initially triaged with EUS. Those with a dilated CBD (>6mm) or stone visualized on EUS then underwent same-session ERCP. Those without EUS findings of choledocholithiasis were triaged according to the 2019 ASGE risk stratification guidelines for CDL. Patients in the high risk category underwent ERCP, and those in the intermediate risk category were scheduled for elective cholecystectomy. Only patients with confirmatory testing with ERCP were included in this study.
Results: Of the 51 patients, the average age was 40, BMI of 32, and the majority (78%) were female. 39 (76%) had stone visualized on EUS, 28 (55%) had a dilated CBD, and 24 (47%) had both visualized stone and dilated CBD. Stone visualized on EUS had a sensitivity of 95% and specificity of 83%. The combination of visualized stone and dilated CBD and a sensitivity of 62% and specificity of 100%. Of the 51 patients, 13 (25%) met criteria for high risk categorization, and 38 (51%) met criteria for intermediate risk categorization. The high risk categorization had a sensitivity of 26% and specificity of 75% for diagnosis of choledocholithiasis. Of the patients in the high risk category, EUS had a sensitivity and specificity of 100% for choledocholithiasis. (Table)
Conclusion: EUS findings had a higher sensitivity and specificity for CDL than the high risk category per risk stratification. In the high risk category, EUS was effective in determining which patients would benefit from ERCP. Early EUS serves as a more effective triage than the 2019 risk stratification guidelines for CDL.

Table 1. Performance of select criteria in the diagnosis of choledocholithiasis

Diagnostic Accuracy of Triage Criteria					
	N (%)	Sensitivity (CI)	Specificity (CI)	PPV (CI)	NPV (CI)
		ASGE Risk Stratification[a]			
High Risk group	13 (25%)	0.26 (0.13, 0.42)	0.75 (0.43, 0.95)	0.77 (0.46,0.95)	0.24 (0.11,0.40)
Intermediate Risk group	58 (75%)	0.74 (0.58, 0.87)	0.25 (0.05, 0.57)	0.76 (0.60,0.89)	0.23 (0.05, 0.54)
		EUS findings			
Visualization of CBD stone	39 (76%)	0.95 (0.83, 0.99)	0.83 (0.52, 0.98)	0.95 (0.83, 0.99)	0.83 (0.52, 0.98)
Both Visualization of stone and dilated CBD[b]	24 (47%)	0.62 (0.45, 0.77)	1.00 (0.74,1.00)	1.00 (0.86,1.00)	0.44 (0.25, 0.65)

[a]Patients were risk stratified per the 2019 ASGE guidelines for choledocholithiasis
[b]Dilated CBD was defined as CBD measuring >6 mm in diameter
CBD = Common bile duct; EUS = Endoscopic Ultrasound; PPV = positive predictive value; NPV = Negative predictive value; N = number of patients; CI = Confidence interval

S117

Endoscopic Ampullectomy - Where Do We Stand Today?

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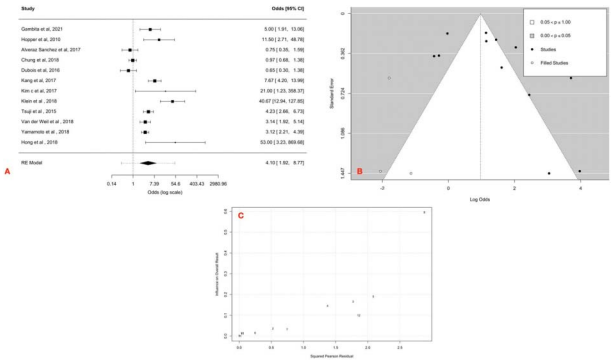
Introduction: The first endoscopic ampullectomy was done in 1983 by Suzuki et al. Since then the technique has been questioned regarding its place in management of ampullary lesions due concerns linked with oncological clearance. The European society of Gastrointestinal endoscopy(ESGE) and the Japanese Gastroenterological Endoscopy society(JGES) have recommendations on endoscopic ampullectomy but American Society of Gastrointestinal endoscopy does not have similar recommendations. Fritzsche et al used Delphi method to draft recommendations but no consensus was reached on 51% of questions. This study aims to investigate the ability of endoscopic ampullectomy to achieve complete resection of the ampullary lesions.
Methods: We reviewed the literature from 2010 to 2022 to evaluate endoscopic ampullectomy for its ability to resect the ampullary lesions completely. "Endoscopic ampullecomy", "endoscopic papillectomy" were used as search words in Pubmed.A pooled odd's ratio was then computed. Statistical software "R" version 4.2.0 was used for data analysis.
Results: We identified 21 studies, two of these studies provided information on successful resection and one was a meta analysis comparing endoscopic ampullectomy with surgical ampullectomy/ pancreatic duodenctomy. Ten more articles were identified from this meta analysis and included in our study. A total of 877 patients were included from a total of 12 studies. 674 patients had complete resection and did not require any additional treatment.A pooled Odd's ratio[95%CI] of 4.10[1.92-8.77] was computed Figure(A). No significant publication bias was noted Figure(B).One of the study by Kim et al was identified as an outlier and an influencer Figure(C).
Conclusion: Endoscopic ampullectomy is effective in complete resection of ampullary lesions with average odds of successful resection of 4. The limitations of this study stems of missing information on the nature of lesions and the size of lesions resected. Also the duration of follow up was not included in most studies. Unlike the JGES, the definition of complete resection was not defined in any study. However the study provides supportive evidence that resection of ampullary lesions is possible endoscopically, without the morbidity and mortality associated with Whipple procedure/ Surgical ampullectomy.



[0117] **Figure 1.** A) Shows the forest plot with odds ratio from the individual studies and pooled Odd's ratio, The results have been subjected to exponential axis transformation. B) Funnel plot showing the publication bias. C) Buajat plot

S118

The Impact of Frailty on Outcomes Among Hospitalized Patients With Chronic Pancreatitis

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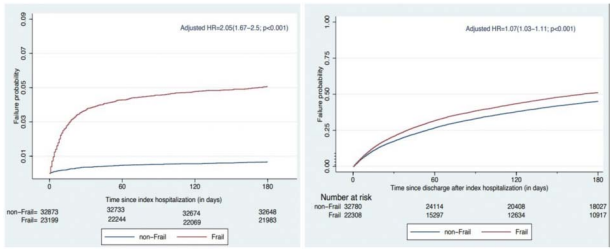
Introduction: The impact of frailty on patients with chronic pancreatitis has not been studied previously. This study aimed to assess the prevalence and impact of frailty on mortality, readmission rates, and healthcare utilization among hospitalized chronic pancreatitis patients in the United States.
Methods: A retrospective cohort study using Nationwide Readmission Database (NRD) on chronic pancreatitis patients with index admissions from Jan 1st, 2019, to June 30th, 2019. These patients were followed till Dec 31st, 2019, in the database. We applied a previously validated hospital frailty risk scoring system to classify chronic pancreatitis patients into frail (score of ≥ 5) and non-frail (score of < 5) on index hospitalization and compared the characteristics of frail and non-frail patients. The primary outcomes of this study were mortality and readmission rates at 6 months. The secondary outcomes included (1) time to death and readmission from discharge after index hospitalization (2) hospitalization characteristics (3) causes of readmission (4) healthcare utilization.
Results: Of 56,072 eligible patients, 41.37% were classified as frail. A total of 65.8% of frail patients were of age less than 65 years, and 32.7% had none or only one comorbidity. Frail patients experienced a higher rate of unplanned and preventable hospitalizations (Table). On multivariate analysis, frailty was independently associated with higher mortality risk (adjusted hazard ratio [aHR], 2.05; 95% CI, 1.7–2.5) (fig 1a). Frailty was also associated with a higher risk of all-cause readmission with an aHR of 1.07; (95% CI, 1.03–1.1) (fig 1b). The mean time to death (28.3 days (24.5-32) vs 48 (37-59)) and to readmission (56.7 days (55.4-58; *p< 0.001*) vs 59.3 (58-61; *p< 0.001*) was significantly shorter among frail patients. The infectious causes were the most common cause of readmission among frail patients (16.8%) compared to acute pancreatitis among non-frail patients (32.3%) (Figure). Frail patients experienced a longer length of stay (7.21 days vs 4.11 days), higher hospitalization costs ($18,705 vs $9918), and hospitalization charges ($78,527 vs $41,927).
Conclusion: Among hospitalized chronic pancreatitis patients, frailty was independently associated with a higher risk of readmissions and mortality at 6 months. Frail patients also accounted for significantly higher health resource utilization in the United States.



[0118] **Figure 1.** Kaplan-Meier curve showing (a) 6-month mortality (b) 6-month readmission among chronic pancreatitis patients stratified by frailty

Table 1. Primary outcomes, hospitalization characteristics, and causes of readmissions among non-frail and frail patients with chronic pancreatitis

Outcome	Frail (%, 95%CI)	non-Frail (%, 95%CI)	p-value
Primary Outcomes			
Readmission rates at 6 months	51.06(50.15-52.12)	45(44.32-45.92)	< **0.001**
Mortality at 6 months	5.24(5.13-5.69)	0.68(0.56-0.81)	< **0.001**
Hospitalization characteristics			
Hospitalizations	96.1(95.7-96.5)	92(91.2-92.7)	< **0.001**
Preventable Hospitalizations	12.72(12.21-13.24)	7.6(7.21-8)	< **0.001**
Severe hospitalizations (Grade 3 and 4)	62.45(61.56-63.32)	24.1(23.5-24.8)	< **0.001**
Severe AP related hospitalizations	5.67(5.37-5.99)	3.99(3.75-4.25)	< **0.001**
Chronic Pancreatitis related procedures	3.59(3.19-4.04)	8.8(8.1-9.54)	< **0.001**
Causes of readmission			
Acute pancreatitis	13.43	32.3	< **0.001**

Table 1. (*continued*)

Outcome	Frail (%, 95%CI)	non-Frail (%, 95%CI)	p-value
Infectious causes	16.68	7.38	< **0.001**
Gastrointestinal (except pancreatitis)	14.01	15.25	< **0.001**

S119

Prognostic Value of Elevated Lipase in Pancreatitis versus Non Pancreatitis Hyperlipasemia (NPHL) due to Non-Malignant Causes

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Introduction: While elevated lipase is typically used to diagnose acute pancreatitis, it has also been associated with other critical disease states including sepsis, COVID-19, bowel obstruction, and trauma. In this study we compared outcomes of patients with elevated lipase who had pancreatitis and NPHL.
Methods: Retrospective analysis was performed on all patients who presented to the Emergency Department between February 2016 and August 2020 with lipase ≥ 3x the upper limit of normal. Patient demographics and past medical history, including active cancer, were noted. Patient outcomes were followed through November 2021. If applicable, dates of death were also documented.
Results: 414 total patients were included in this study. Upon initial evaluation, 305/414 (74%) were diagnosed with acute pancreatitis (AP) and 109/414 had NPHL. The age (54 ±17 vs. 58 ±18, p=0.0220), Sex (male 164/305 vs. 49/109, p=0.1194), and BMI (28.9 ±7.4 vs. 25.8 ±4.6, p=0.0066) were compared between the AP and NPHL groups. The serum lipase in the AP and NPHL group were respectively 1471 ±1070 vs. 605 ±555 (p< 0.0001). The most common causes of NPHL were sepsis (10/109; 9%) renal failure (7/109; 6%), GI bleed (5/109; 4%), and bowel obstruction (5/109; 4%). The NPHL group had higher rate of malignancy (29/105; 28%) compared to those with AP (35/305; 11%, p< 0.0001). NPHL patients without malignancy had a higher mortality rate (63/80; 80%) compared to those without malignancy in the AP group (17/270; 6.3%, p< 0.0001). The most common malignancy in patients with AP was breast (6/35; 17%, vs. 3/29; 10%, p=0.4943). In NPHL, the most common malignancies were pancreatic (4/29; 14%, vs. 3/35; 9%, p=0.6920) and bowel malignancies (4/29; 14%, vs. 4/35, 11% p=1.0000).
Conclusion: Patients with NPHL without malignancy have higher mortality than those with pancreatitis despite lower serum lipase levels. A limitation of our study is the difference between age and BMI of AP versus NPHL patients. Whether this impacts the prognostic relevance of NPHL on survival need to be explored in future studies.

S120

Acute Cholangitis in the Geriatric Population: Interventions and Outcomes From a Nationwide Analysis (2016-2018)

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Introduction: Acute cholangitis (AC) is a medical emergency resulting from biliary obstruction and infection of the biliary tract. ERCP is the treatment of choice but percutaneous drainage by Interventional Radiology (IR) or surgical drainage are also performed. Geriatric AC patients are high-risk for severe morbidity/mortality. We studied the utilization of these interventions and outcomes among these patients.
Methods: We used National Inpatient Sample for 2016 to 2018. All diagnoses and procedures were identified using ICD 10 codes. We identified geriatric (age ≥65) patients admitted with AC. We stratified data into 2 groups: age 65–79 (G1) and age ≥80 (G2). We identified ERCP, IR and surgical procedures for treatment of AC using ICD-10 PCS codes. Inpatient mortality and length-of-stay (LOS) were calculated. Logistic regression was used to adjust for age, sex, race, comorbidities (using Elixhauser Index), and interventions (ERCP, IR, surgery or any combination of these) to calculate adjusted odd ratio (OR) for inpatient mortality.
Results: 87,950 geriatric patients were admitted from 2016 to 2018 with AC. 55,570 (63.2%) belonged to G1 and 32,380 (36.8%) to G2. There was a significantly higher proportion of females in G2 (50.9% vs 42.9%, P-value < 0.01). Overall mortality was 6.8%; higher in G2 (7.3% vs 6.6%, P-value< 0.01). The median LOS was 5 days (G1 and G2). ERCP only was performed with less frequency in G1 (38.9% vs 43.1%). IR only was performed more in G1 (5.9% vs 3.8%) and surgery only was done rarely in each group (0.3% vs 0.3%). Combinations of ERCP, IR and surgery were rarely performed (Table). Mortality without procedures was 8.7% overall, but lower in G1 (7.9% vs 10.0%, P< 0.05). ERCP had the lowest overall mortality of 3.9% compared to 9.5% with IR and 7% with surgery. Mortality was lower in G1 with ERCP vs IR (4% vs 10.0%, P< 0.001) and similarly lower in G2 with ERCP vs IR (3.7% vs 8.2%, P< 0.001). Mortality was similar in G1 with ERCP vs surgery (4.0% vs 5.4%, P=0.343) but lower in G2 with ERCP vs surgery (3.7% vs 10.0%, P=0.004). LOS data is summarized in Table: Adjusted OR for mortality was higher for female gender (OR 1.33, 95% CI 1.17–1.51), lower for ERCP vs no intervention (OR 0.49, 95% CI 0.42–0.57) and higher for G2 (vs G1) (OR 1.27, 95% CI 1.11–1.44).
Conclusion: In AC patients, ERCP is associated with lower mortality and decreased length of stay compared to IR or surgery. ERCP can be safely considered as the preferred intervention in geriatric patients with AC.

Table 1. **Interventions performed for Acute Cholangitis in the two geriatric age groups.**

Intervention	Age Group	Utilization	Mortality	Length of Stay
None	G1	53.0% *	7.9% *	4
	G2	51.2% *	10.0% *	5
ERCP only	G1	38.9% *	4.0%	5
	G2	43.1% *	3.7%	5
IR only	G1	5.9% *	10.0% *	8
	G2	3.8% *	8.2% *	8
Surgery only	G1	0.3%	5.4%	8
	G2	0.3%	10.0%	9
ERCP and IR	G1	1.6%	10.5%	10
	G2	1.4%	11.4%	11
ERCP and Surgery	G1	0.3%	10.0%	12
	G2	0.2%	16.7%	10
IR and Surgery	G1	< 0.1%	n/a	12
	G2	< 0.1%	n/a	10
ERCP, IR and Surgery	G1	< 0.1%	n/a	12
	G2	< 0.1%	n/a	10

G1 = Age 65 to 79 years; G2 = Age 80 and above.
*P<0.05 for comparison with other age group within same intervention.

S121

A Comparison of Acute Pancreatitis Severity Scores in Predicting Patient Outcomes

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Introduction: Acute pancreatitis is commonly seen in the inpatient setting, and its presentation can vary widely, ranging from mild disease to a more severe presentation, which is commonly associated with a high morbidity and mortality. With the advent of pancreatitis scoring systems, however, physicians can predict certain outcomes for patients with more reliability. The primary objective of this study is to compare two new pancreatitis scoring systems in determining illness severity, complications, and need for ICU level care. The secondary objective is to determine if there is a correlation with severity and age, gender, race, etiology of pancreatitis, and insurance.
Methods: We performed a retrospective chart review of adult patients admitted with acute pancreatitis to a community hospital from 2018 to 2021. We utilized two novel scoring systems: the Chinese simple scoring system (CSSS) and the Pancreatic activity scoring system (PASS) and compared them with pre-existing and validated scoring systems: the APACHE-II scoring system, Ranson scoring system, BISAP scoring system, and Glasgow-Imrie scoring system by calculating each score. We included other variables such as length of stay, disposition (general ward, telemetry, ICU), readmission, complications, etiology of pancreatitis, presence of necrotizing pancreatitis, calcium level on Day 2, and C-reactive protein levels.
Results: The main result of this study was to determine which score for both the CSSS and PAAA risk assessments was most predictive of a need for ICU level care. Overall, a total of 16/88 of patients admitted for pancreatitis (18%) were critical enough to require ICU monitoring. Of these patient's triaged to the ICU for treatment of pancreatitis, the average CSSS score was 253. The average PASS score of those admitted to the ICU for pancreatitis was 3.88. Patients with a length of stay greater than 7 days, were re-admitted within 30 days 55% of the time and had a CSSS and PASSS score of 243 and 3.55, respectively.
Conclusion: It is important as clinicians to evaluate severity of pancreatitis when triaging patients as predicting complications can be challenging. These scoring systems have been invented and validated to help determine the level of care needed for the patient and to foresee potential complications. Our data suggests the PASS and CSSS scoring systems can be essential triage tools to better predict patient outcomes, complications, mortality, and possible need for escalation of care.

S122

Endoscopic Ultrasound Evaluation of Primary Pancreatic Lymphoma Compared to Adenocarcinoma: A Case-Control Study

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Introduction: Primary pancreatic lymphoma (PPL) is a rare extranodal lymphoma representing 0.5% of all pancreatic neoplasms. Prognosis and treatment strategies of PPL vastly differ from pancreatic ductal adenocarcinoma (PDAC), therefore making an accurate diagnosis is vital. Our aim is to describe the presentation, endoscopic ultrasound (EUS) features, and the role of fine needle aspiration (FNA) in the diagnosis of PPL compared with PDAC.
Methods: Out of 1,946 patients who underwent EUS-FNA of solid pancreatic lesions, 11 were diagnosed with PPL. Patients who had peripancreatic lesions, lymph nodes, or bile duct masses were excluded. Age and gender- matched controls with a diagnosis of PDAC were identified in a 1:3 ratio. Presenting symptoms, demographics, and EUS characteristics were evaluated.
Results: There were 11 patients with PPL and 33 age and gender-matched controls with PDAC. The median age was 65 years (60-76) and 63.6% were males. There was no difference between two groups in smoking, alcohol consumption, or history of a second malignancy. PPL patients were more likely to present with abdominal pain (81.8% vs 26.4%, p=0.01). Pancreatic head location was most common in both groups. Both PPL and PDAC tended to be hypoechoic and poorly defined on EUS, while PPL were more commonly described as heterogeneous (36.4% vs 6.1%, p=0.01). PPL were significantly larger on EUS (45.8 mm vs 31 mm, p< 0.01) and less likely associated with pancreatic duct dilation. A higher number of FNA passes was required for the diagnosis of PPL compared to PDAC (5 vs 3, p< 0.01).
Conclusion: While PPL remains rare, making an accurate distinction with PDAC is critical as treatment and prognosis radically differ. On presentation, PPL is more likely to cause abdominal pain. Certain EUS features were found to be beneficial in differentiating PPL from PDAC in our study, as PPL tend to be larger, more likely heterogeneous, and are less likely associated with pancreatic duct dilation compared to PDAC. Tissue diagnosis is more challenging in PPL as reflected by a significantly higher number of passes obtained on EUS- FNA.

Table 1.

	Adenocarcinoma (n=33)	Primary Pancreatic Lymphoma (n=11)	P value
Ethnicity, white n (%)	21 (63.6)	8 (72.7)	0.58
Smoking history (>15 pack-years), n (%)	9 (27.3)	5 (45.5)	0.26
Alcohol history (>1 drink/day), n (%)	7 (21.2)	2 (18.2)	0.83
History of cancer diagnosis, n (%)	10 (30.3)	3 (27.3)	0.85
Multiple pancreatic lesions, n (%)	1 (3)	0	0.56
Presenting symptoms			
Abdominal pain, n (%)	12 (36.4)	9 (81.8)	**0.01**
Jaundice, n (%)	11 (33.3)	6 (54.5)	0.21
Weight loss, n (%)	8 (24.2)	3 (27.3)	0.84
Incidental, n (%)	5 (15.2)	1 (9.1)	0.61
Located in the head of the pancreas, n (%)	23 (69.7)	9 (81.8)	0.43
Regional lymphadenopathy on imaging, n (%)	20 (60.6)	10 (90.9)	0.06
EUS features			
Hypoechoic, n (%)	31 (93.9)	9 (81.8)	0.23
Heterogeneous, n (%)	2 (6.1)	4 (36.4)	**0.01**
Poorly defined, n (%)	23 (69.7)	8 (72.7)	0.85
Common bile duct dilation, n (%)	14 (42.4)	5 (45.5)	0.86
Pancreatic duct dilation, n (%)	23 (69.7)	3 (27.3)	**0.01**
Number of passes on FNA, mean (IQR)	3 (1-3)	5 (2-7)	**0.008**
Largest diameter on EUS (mm), median (IQR)	31 (25-38.8)	45.8 (35.5-56)	**0.001**
Treatment			
Chemotherapy, n (%)	29 (87.9)	10 (90.9)	0.78
Radiation therapy, n (%)	17 (51.5)	2 (18.2)	0.053
Surgery, n (%)	7 (21.2)	1 (9.1)	0.37

S123

A Prospective Cohort Study Evaluating Pan-Promise: A Patient-Reported Outcome Measure to Detect Post-ERCP Pancreatitis Symptoms

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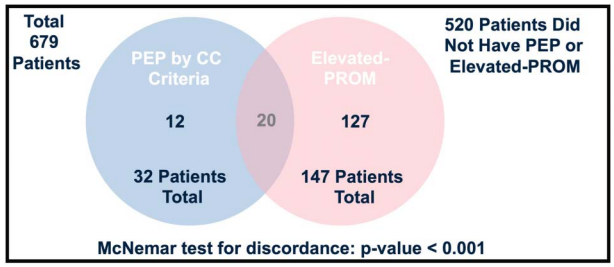
Introduction: The Cotton Consensus (CC) criteria for post-ERCP pancreatitis (PEP) may not capture post-ERCP morbidity and uses length of stay to define severity. PAN-PROMISE, a patient-reported outcome measure, was developed to quantify AP-related morbidity. This study aims to determine the value of PAN-PROMISE in independently defining ERCP-related morbidity.
Methods: We conducted a prospective cohort study of patients undergoing ERCP at two academic centers from 09/2021 to 08/2022. We administered PAN-PROMISE and assessed QOL and work-productivity at baseline, 48-72 hours, seven days, and 30 days following ERCP. Patients also received a standardized telephone call to assess for the CC criteria. CC-PEP was determined by a blinded, three-physician adjudication committee. The McNemar's test assessed discordance between matched pairs of CC-PEP and changes in PAN-PROMISE > 7 at 7 days. After enrollment of 150 patients, an interim analysis using ROC analysis identified that the optimal cutpoint in a change of PAN-PROMISE was an increase of >7 points (sensitivity 83%, specificity 78%, AUC 0.85) (Figure)
Results: 679 patients were enrolled (Median age 63, 55% male, 55% had a history of cancer). The indications for ERCP were choledocholithiasis (30%), malignant biliary obstruction (29%), and liver transplant (14%). 95% of patients received rectal indomethacin. 32 (4.7%) patients developed PEP. 147 (21.6%) patients had an elevated-PROM, while only 20 (13.4%) of them had PEP by the CC criteria ($p < 0.001$ for discordance). An elevated-PROM strongly correlated with lower physical QOL and increased direct and indirect healthcare costs ($80 and $25 per point increase in PAN-PROMISE, respectively). Patients with pancreatic cancer (OR 4.52 95%-CI 1.68-10.74) and PSC (OR 1.79 95%-CI 1.29-2.45) had the highest odds of elevated-PROM. (Table)
Conclusion: A substantial number of patients experience significant morbidity after ERCP despite not developing PEP or other adverse events. Future studies are needed to characterize better the reasons behind this increase in symptoms and potential interventions to reduce the symptom burden post-ERCP.



[0123] **Figure 1.** Venn Diagram comparing discordance between PEP and an elevated-PROM.

Table 1. Risk Factors for an Elevated-PROM Using Multivariable Logistic Regression.

	Multivariable Analysis	
Variable	**aOR (95% CI)**	**P-Value**
Clinical Characteristics		
Age	0.99 (0.98 - 1.01)	0.75
Female Gender	1.07 (0.75 - 1.55)	0.69
Pre-Procedure Total Bilirubin	1.05 (1.02 - 1.09)	**0.001**
Chronic Opiate Usage	2.74 (1.82 - 3.9)	**< 0.001**
History of Chronic Pancreatitis	1.05 (0.57 - 1.93)	0.45
Indication (compared to Choledocholihtiasis)	–	–
Pancreatic Cancer	4.25 (1.68 - 10.74)	**0.002**
Primary Sclerosing Cholangitis	1.79 (1.29 - 2.45)	**0.005**
OLT	0.69 (0.21 - 0.89)	**0.01**
Procedural Characteristics		
Native Papilla	1.12 (0.77 - 1.66)	0.55
SEMS Placement	2.27 (1.25 - 4.17)	**0.007**
PD Manipulation	1.52 (1.01 -2.30)	**0.04**
Peri-Procedural Hydration with LR	0.51 (0.31 - 0.83)	**0.007**
PD Stent Placement	0.56 (0.29 - 0.96)	**0.03**

S124

The Voice of the Patient With Exocrine Pancreatic Insufficiency Secondary to Chronic Pancreatitis: Preliminary Findings From a Patient-Driven Registry

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Introduction: Exocrine pancreatic insufficiency (EPI) remains underdiagnosed and undertreated despite its impact on quality of life (QoL) and increased morbidity/mortality. Chronic pancreatitis (CP) is the most common disease of the pancreas associated with EPI. The North American Pancreatitis Study 2 explored the natural history (including exocrine insufficiency) of recurrent acute pancreatitis (RAP) and CP; however, more data are needed on this subgroup. To better understand EPI, its impact on QoL/healthcare resources, and treatment effects, an innovative dual approach using a patient online community and patient-driven traditional registry was used to collect data from patients and their gastroenterologists on real-world experiences with EPI and pancreatic enzyme replacement therapy (PERT). Preliminary findings from the prospective registry are reported.
Methods: This is a longitudinal, noninterventional study in the US with a planned enrollment of 400 patients, spanning ~5 years. Adults with suspected/confirmed EPI, receiving PERT, and diagnosed with CP/RAP at enrollment are eligible. Assessments are collected at prespecified intervals. To understand impact of clinical practices and EPI clinical course, multivariate modeling will be used to analyze effects of medical history, comorbidities, and treatments on EPI history/progression, QoL, and healthcare resource use.

Results: As of February 22, 2022, 35 patients are enrolled. Median age is 62 years, and most patients are White (Table). Alcohol (susceptibility/progression) and diabetes mellitus were common etiologies. Abnormal fecal elastase-1 test, vitamin deficiency, and weight loss were frequently used evidence for EPI diagnosis. Steatorrhea was present some of the time in 14 patients; 12 never had steatorrhea. Patients reported PERT greatly (n=12) and somewhat (n=11) improved symptoms. Over the past 3 months, 10 patients reported ER visits; 7 needed hospitalizations.

Conclusion: Enrolled patients with EPI are diverse in terms of demographics, etiologies, and clinical presentations. Initial findings include heterogeneous diagnostic methodologies, substantial but not overwhelming presence of steatorrhea, and improvement of symptoms with PERT in most patients. Early experience confirms feasibility of this data collection modality in those with CP; future analyses will help clarify areas of unmet needs in EPI management, patient experience, and patient/physician factors that may affect treatment success.

Table 1. Patient Demographics and Disease Characteristics at Baseline

Characteristic	Total (N=35)
Patient demographics	
Age (years), median (range)	62 (31–81)
Sex, n (%)	
Male	16 (45.7)
Female	14 (40.0)
Race, n (%)	
White	23 (65.7)
Black or African American	3 (8.6)
Asian	2 (5.7)
Other	2 (5.7)
Ethnicity, n (%)	
Hispanic or Latino	6 (17.1)
Not Hispanic or Latino	24 (68.6)
Chronic pancreatic etiologyb,c, n (%)	
Toxic-metabolic	
Alcohol (susceptibility/progression)	13 (37.1)
Tobacco smoking	7 (20.0)
Hyperlipidemia (fasting >300 mg/dL, nonfasting >500 mg/dL)	5 (14.3)
Medications	3 (8.6)
Toxins, other	3 (8.6)
Toxins, other, not otherwise specified	3 (8.6)
Hypercalcemia (total calcium levels >12.0 mg/dL or 3 mmol/L)	2 (5.7)
Toxins, chronic kidney disease (stage 5, end-stage renal disease)	1 (2.9)
Not applicable	16 (45.7)
Metabolic, other	
Diabetes mellitus	12 (34.3)
Not applicable	23 (65.7)
Idiopathic	
Late (>35 years of age)	12 (34.3)
Early (< 35 years of age)	2 (5.7)
Not applicable	21 (60.0)
Evidence of suspected/confirmed EPI diagnosisc, n (%)	
Abnormal fecal elastase-1 test	10 (28.6)
Vitamin deficiency	9 (25.7)
Weight loss	9 (25.7)
Clinical steatorrhea	7 (20.0)
Pancreatic function testing	3 (8.6)
Other	7 (20.0)
Not applicable	6 (17.1)
Clinical manifestations of EPIc, n (%)	
Gastrointestinal symptoms	
Diarrhea/loose stool	22 (62.9)
Abdominal pain	22 (62.9)
Bloating	17 (48.6)
Flatulence	8 (22.9)
Steatorrhea	8 (22.9)
Not applicable	3 (8.6)
Nutritional	
Vitamin D deficiency	15 (42.9)
Other vitamin/mineral deficiencies	8 (22.9)
Unintentional weight loss/difficulty gaining weight	8 (22.9)
Malnutrition	6 (17.1)

Table 1. (*continued*)

Characteristic	Total (N=35)
Other	1 (2.9)
Not applicable	17 (48.6)

[a]Data were unavailable for 5 patients at the time of data analysis.
[b]Selected chronic pancreatic etiology; other categories not shown include genetic, autoimmune, recurrent and severe acute pancreatitis, obstructive, and other.
[c]Multiple options may be selected for individual participants.
Abbreviation: EPI, exocrine pancreatic insufficiency.

S125

Predictors and Causes of 30-Day Readmissions in Primary Biliary Cholangitis: Analysis of the Nationwide Readmission Database

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Introduction: Primary biliary cholangitis (PBC) is an autoimmune disorder that most commonly affects middle-aged women. Cirrhosis, hepatocellular carcinoma, metabolic bone disease, and malabsorption can complicate PBC. In this study we aimed to describe the causes and predictors of 30-day readmissions in patients with PBC.
Methods: This was an observational retrospective study involving adult patients hospitalized with PBC in the US. We utilized the Nationwide Readmission Databases 2016 to 2019. The first hospitalization for PBC within the year was marked as the index admission. We identified one subsequent hospitalization within 30 days, this was marked as a readmission. We analyzed the rates, causes, and outcomes of the readmitted cohort. We used multivariable cox regression analysis to identify independent predictors of readmissions.
Results: We included a total of 3954 hospitalizations with PBC as the primary diagnosis for admission. 30.5% of the discharged cohort were readmitted within 30 days. Excluding PBC, the most common reasons for readmission were hepatic failure (9.6%) and sepsis (6.7%). Fluid and electrolyte disorders were the most common comorbidities in both the index hospitalization and readmission cohorts. Mortality was higher in the readmitted patients compared to index hospitalizations (10.1% and 4.3%, respectively, $p < 0.001$). Predictors of 30-day readmissions were peptic ulcer disease (aHR 1.64, $p = 0.040$), renal failure (aHR 1.29, $p = 0.038$), weight loss (aHR 1.28, $p = 0.024$), fluid and electrolyte disorders (aHR 1.28, $p = 0.011$), and coagulopathy (aHR 1.22, $p = 0.029$).
Conclusion: Patients with PBC have high readmission rates, these readmissions are associated with high inpatient mortality. PBC patients are at risk of liver cirrhosis and malabsorption which explains the high rates of sepsis and electrolyte abnormalities observed in our study. Coagulopathy and weight loss might be reflective of the severity of liver dysfunction and the malabsorptive state, hence, there were associated higher readmission risk. Excellent short and long-term survival have been described following liver transplantation for PBC patients. However, recent data demonstrated that they have higher wait-list mortality among patients listed for liver transplantation.

S126

Comorbid Diabetes on Hospitalization Outcomes in Acute Pancreatitis

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Introduction: Data on the association between comorbid diabetes mellitus (DM) and acute pancreatitis (AP) remain limited. Utilizing a large, nationwide database, we aimed to examine the impact of comorbid diabetes mellitus on patients admitted for acute pancreatitis.
Methods: This was a retrospective case-control study of adult patients with AP utilizing the National Inpatient Sample from 2015-2018, using ICD-10 codes. Hospitalization outcomes of patients admitted for AP with comorbid DM were compared to those without comorbid DM at the time of admission. The primary outcome was a mortality difference between the cohorts. Multivariate regression analysis was performed.
Results: 940,789 adult patients with AP were included, of which 256,330 (27.3%) had comorbid DM. Comorbid DM was associated with a 31% increased risk of inpatient mortality (aOR: 1.31; $p=0.004$), a 53% increased risk of developing sepsis (aOR: 1.53; $p=0.002$), increased hospital length of stay (LOS) (4.5 days vs. 3.7 days; $p < 0.001$), and hospital costs ($9934 vs. $8486; $p < 0.001$). Whites admitted for AP with comorbid DM were at a 49% increased risk of mortality as compared to Hispanics (aOR: 1.49; $p < 0.0001$).
Conclusion: Comorbid DM appears to be a risk factor for adverse hospitalization outcomes in patients admitted for AP with male sex and race as additional risk factors. Future prospective studies are warranted to confirm these findings to better risk stratify this patient population.

S127

Adverse Events Following ERCP Are Higher in Patients With Chronic Pancreatitis

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Introduction: Endoscopy retrograde cholangiopancreatography (ERCP) is frequently used to manage complications of chronic pancreatitis (CP) including pancreatic duct stones, strictures, and leaks. Previous studies have indicated a protective effect of chronic pancreatitis on rates and severity of post-ERCP pancreatitis. The aim of this study was to evaluate the risk of adverse events (AEs) in a large national cohort of patients with chronic pancreatitis who underwent ERCP.
Methods: We queried the Explorys database (Cleveland, OH), comprised of electronic medical record data from 26 major U.S. healthcare systems. Adult patients, 18 years of age and older, with and without CP who underwent ERCP between 1999 and 2021 were identified based on systematized nomenclature of medicine-Clinical Terms (SNOMED-CT). Differences in baseline characteristics and demographics were analyzed using chi-square tests. Odds ratio analyses were performed between CP and non-CP patients for AEs within 30 days of ERCP. We considered P-values less than 0.05 to be statistically significant.
Results: Out of total of 147,360 patients who underwent ERCP, we identified 13,270 (9%) patients with CP. Patients with CP who underwent ERCP were more likely male (47.9% vs. 39.3%, $P< 0.0001$) and African American (16.3% vs. 9.8%, $P< 0.0001$). CP patients had higher rates of chronic obstructive pulmonary disease (27.5% vs. 16.7%, $P< 0.0001$), diabetes mellitus (46.0% vs. 29.2%, $P< 0.0001$), alcohol abuse (19.2% vs. 4.0%, $p< 0.0001$), and tobacco abuse (35.9% vs. 16.2%). CP patients compared to non-CP patients had higher odds of adverse events within 30 days of ERCP including mortality (OR 5.06, $P< 0.0001$), sepsis (OR 1.14, $P< 0.0001$), AKI (1.20, $P< 0.0001$), gastrointestinal bleeding (OR 1.35, $P< 0.0001$), and myocardial infarction (OR 1.36, $P< 0.0001$). The odds of acute pancreatitis within 7 days were also higher (OR 2.32, $P< 0.0001$). The odds of cholangitis within 30 days were lower in the CP group (0.48, $P< 0.0001$). (Table)
Conclusion: Compared to non-CP patients, CP patients had higher odds of post-ERCP AEs. Contrary to prior data, rates of acute pancreatitis after ERCP were higher in the CP group. Differences in AEs may be related to altered biliary and pancreatic ductal anatomy, pancreatic reserve, and comorbid conditions stemming from higher rates of tobacco and alcohol use in CP patients. Further studies are warranted to help clarify these associations and stratify the safety of ERCP in CP patients.

Table 1.

Outcome	ERCP + Chronic Pancreatitis (n=13,270), n	ERCP + CP, %	ERCP + No CP (n=134,090), n	ERCP + No CP, %	Odds Ratio	CI (95%)	P-Value
Mortality*	10	0.1%	20	0.0%	5.0562	2.3662, 10.8041	< 0.0001
Sepsis*	1,040	7.8%	8,970	6.7%	1.1441	1.0702, 1.2230	< 0.0001
Cholangitis*	1,780	13.4%	31,760	23.7%	0.4803	0.4563, 0.5056	< 0.0001
AKI*	1,040	7.8%	8,610	6.4%	1.1953	1.1180, 1.2780	< 0.0001
GI bleed*	590	4.4%	4,310	3.2%	1.3531	1.2392, 1.4774	< 0.0001
Acute pancreatitis (1-7 days)	890	6.7%	3,900	2.9%	2.3157	2.1483, 2.4961	< 0.0001
MI*	560	4.2%	4,070	3.0%	1.3594	1.2422, 1.4876	< 0.0001
Gender							
Female	6,910	52.1%	81,370	60.7%			< 0.0001
Male	6,350	47.9%	52,690	39.3%			< 0.0001
Race							
White	10,110	76.2%	106,210	79.2%			< 0.0001
African American	2,160	16.3%	13,160	9.8%			< 0.0001
Asian	140	1.1%	2,040	1.5%			0.7041
Hispanic/Latino	120	0.9%	1,650	1.2%			0.7689
Age							
Adults (18-65y)	7,110	53.6%	77,960	58.1%			0.0001
Seniors (>65y)	6,090	45.9%	54,340	40.5%			0.0001
Comorbidities							
Stroke	2,300	17.3%	19,310	14.4%			0.0002
CAD	3,490	26.3%	30,290	22.6%			< 0.0001
Cardiomyopathy	910	6.9%	6,920	5.2%			0.0329
CHF	1,990	15.0%	18,150	13.5%			0.0643
COPD	3,410	25.7%	22,350	16.7%			< 0.0001
PAD/PVD	5,060	38.1%	40,560	30.2%			< 0.0001
HTN	9,350	70.5%	80,980	60.4%			< 0.0001
HLD	7,480	56.4%	66,430	49.5%			< 0.0001
DM	6,100	46.0%	39,170	29.2%			< 0.0001
CKD	2,920	22.0%	23,140	17.3%			< 0.0001
ESRD	740	5.6%	4,040	3.0%			0.0003
Cirrhosis	1,540	11.6%	8,660	6.5%			< 0.0001
Alcohol abuse	2,550	19.2%	5,330	4.0%			< 0.0001
Tobacco abuse	4,760	35.9%	21,660	16.2%			< 0.0001
Obesity	3,360	25.3%	34,510	25.7%			0.612

*Within 30 days of ERCP.

S128

Mortality Associated With Pancreatic Malignant Tumors Is Improving but Incidence Continues to Rise

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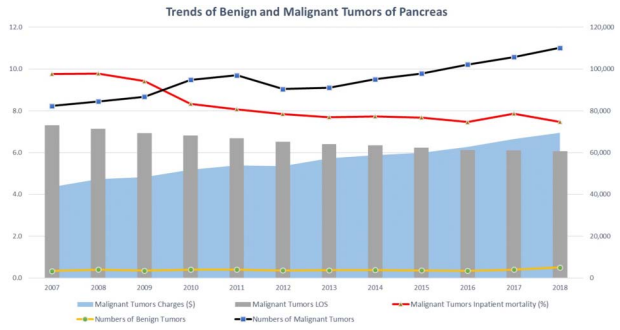
Introduction: Multiple guidelines have been proposed since 2006 for screening and surveillance of pancreatic neoplasms. However, effective screening strategies for pancreatic malignancies remain elusive. In this study we sought to identify any changes in incidence and mortality trends of pancreatic neoplasms after the introduction of pancreatic cancer screening guidelines.
Methods: Adult patients with a principal or secondary diagnosis of benign or malignant pancreatic neoplasm were selected from the National Inpatient Sample (NIS) database for the years 2007 to 2018. Yearly incidence of pancreatic benign and malignant tumors was calculated as well as trends of inpatient mortality for these patients. Secondary variables of interest included healthcare resource utilization [length of stay (LOS) and total hospitalization costs and charges]; as well as demographic variables and risk factors associated with pancreatic neoplasms.
Results: From 2007 to 2018, a total of 4616 patients were diagnosed with benign pancreatic tumors and 1,143,034 had malignant pancreatic tumors. There was an increase in the incidence of malignant pancreatic tumors by approximately 2.4% from 2007 to 2018; however, the annual mortality improved from 9.7% to 8.2% [$p < 0.0001$]. There was no significant variation in the incidence of benign pancreatic tumors or associated mortality [$p < 0.31$]. Mortality and healthcare utilization trends are summarized in Figure: The mean age for patients with malignant tumors was 67.9 years with no significant change in trends [$p < 0.2$]. From 2007 to 2018 the female preponderance for benign and malignant tumors decreased from 50.23% to 49.34% [$p < 0.0001$]. There was a significant increase in Medicare (56.4% to 58.3%) and Medicaid (6.2% to 7.9%) share as primary payer [$p < 0.0001$]. Risk factors associated with pancreatic malignancy also had a significantly rising trend.

Conclusion: The incidence of malignant pancreatic tumors continues to rise. However, the associated mortality has been decreasing. Despite improving implementation of screening guidelines, there was no change in number of benign pancreatic lesions being diagnosed from 2007 to 2018. Risk factors associated with pancreatic malignancy continue to rise. These trends indicate the need for continued efforts for improved screening for early-stage pancreatic malignancies as well as stronger efforts to reduce risk exposure in high-risk patients (Table).

Table 1. Demographics and Risk factors

Year	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	2017	2018	Total	p-value
Female (%)	50.23	50.93	49.74	49.09	51	48.91	48.77	49.29	48.69	48.25	48.4	49.18	49.34	*< 0.0001*
Race (%)														*0.35*
White	74.78	75.22	74.2	73.61	73.19	72.55	71.72	72.69	71.66	72.03	70.78	70.86	72.6	
Black	11.82	11.29	11.65	14.38	13.32	13.53	14.11	13.48	14.13	13.64	14.17	13.76	13.38	
Hispanic	6.98	7.02	7.33	6.98	7.97	7.68	7.91	7.8	7.91	7.72	7.92	8.69	7.72	
Asian/Pacific islander	3.3	3.06	3.46	2.4	2.5	2.82	3.18	3.08	3.19	3.16	3.32	3.38	3.07	
Native American	0.59	0.29	0.59	0.4	0.29	0.46	0.36	0.35	0.35	0.38	0.33	0.37	0.39	
Other	2.53	3.12	2.77	2.22	2.73	2.96	2.72	2.6	2.77	3.08	3.47	2.94	2.84	
Primary Expected Payer (%)														< 0.0001
Medicare	56.43	56.26	56.53	55.94	58.46	58.08	58.37	58.81	58.96	59.3	60.59	61.52	58.39	
Medicaid	6.25	6.92	6.57	8.15	7.28	7.9	8.12	8.51	9.13	8.75	8.69	8.53	7.96	
Private insurance	32.88	31.95	31.58	30.79	29.05	28.51	28.12	28.06	27.43	27.4	26.42	25.67	28.84	
Self-pay	1.91	1.85	2.55	2.77	2.06	2.7	2.43	1.99	1.84	1.83	1.66	1.69	2.09	
No charge	0.25	0.22	0.19	0.31	0.33	0.18	0.41	0.25	0.16	0.14	0.16	0.11	0.22	
Other	2.27	2.81	2.57	2.04	2.81	2.63	2.54	2.38	2.47	2.58	2.48	2.49	2.51	
Region of Hospital (%)														0.99
Northeast	25.1	23.41	21.7	24.15	21.26	22.51	22.69	22.67	21.77	22.52	22.61	21.4	22.6	
Midwest	21.95	21.48	24.87	23.6	22.76	22.96	22.33	23.06	22.75	22.23	22.8	22.91	22.81	
South	34.24	34.15	33.6	34.65	38.5	36.03	36.49	35.76	36.86	36.66	35.26	36.16	35.75	
West	18.71	20.96	19.83	17.6	17.47	18.5	18.5	18.51	18.61	18.59	19.33	19.52	18.84	
Type of hospital (%)														< 0.0001
Rural	9.88	9	12.41	11.74	10.83	12.03	11.65	15.22	14.03	14.9	14.83	15.91	12.84	
Non-teaching urban	20.23	19.44	20.99	18.17	20.59	22.91	23.07	25.84	26.17	25.09	25.55	26.74	23.06	
Teaching urban	69.89	71.56	66.6	70.09	68.58	65.06	65.28	58.94	59.79	60.01	59.62	57.36	64.1	
Median Household income quartile (%)														0.11
$1-24,999	25.04	22.76	22.97	25.61	26.11	25.93	24.64	24.09	26.35	25.78	25.19	24.28	24.92	
$25,000-34,999	22.49	26.41	25.76	23.77	23.11	23.54	25.04	27.03	23.46	24.43	24.94	25.54	24.65	
$35,000-44,999	24.18	23.78	24.89	23.94	26.52	24.86	24.63	23.87	24.8	24.86	25.11	25.83	24.81	
$45,000 or more	28.28	27.04	26.38	26.68	24.27	25.68	25.69	25.01	25.39	24.93	24.75	24.34	25.63	
Alcohol Use disorder (%)	1.17	1.15	1.38	1.42	1.59	1.55	1.6	1.58	4.22	11.94	12.23	12.08	4.63	< 0.0001
Smoking Use disorder (%)	16.35	17.22	20.83	23.26	24.21	26.53	28.94	32.17	35.43	37.05	37.75	37.91	28.66	< 0.0001
Obesity (%)	2.54	3.15	4.1	4.36	4.98	5.89	6.05	7.21	7.45	6.6	7.4	7.86	5.74	< 0.0001
Chronic Pancreatitis (%)	2.2	2.39	2.09	2.77	2.21	2.54	2.63	2.49	2.65	2.37	2.63	2.93	2.5	0.17
Diabetes Mellitus (%)	31.49	32.05	35.3	34.95	36.43	37.11	37.16	38.01	38.25	39.43	40.58	40.42	36.96	< 0..0001



[0128] **Figure 1.** Incidence, Mortality and Healthcare Utilization trends.

S129

Characteristics of Patients With Ampullary vs. Other Periampullary Carcinoma

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Introduction: Ampullary carcinoma (AC) are a rare neoplasia comprising 0.2% of all gastrointestinal cancers. Despite this, they are responsible for up to 20% of all tumor related obstructions of the common bile duct (CBD), and their rates are increasing. These neoplasia are distinct but a part of a group of cancers labeled periampullary carcinoma (PC), which consist of primary duodenal, distal bile duct, pancreatic and ACs anatomically centered around the Ampulla of Vater. Here we describe individual patient data as well as trends in comorbidities, outcomes, and socioeconomic factors in patients with AC vs. PC in a large system-wide database.
Methods: Deidentified patient information was obtained via data gathering software based on CPT codes. Of these, 16075 patients were found to have a diagnosis of one periampullary carcinoma. 5599 were removed due to lack of data, leaving us with 10476 patients. Statistical analysis was done on commercially available software.
Results: There were 245 patients with AC and 10231 with other PC. Other PC included 9199 (89.91%) pancreatic, 620 (6.06%) bile duct, and 412 (4.03%) duodenal. AC was more common in Hispanic patients (19.59% vs. 13.08%, $P< 0.001$). More patients with AC were discharged home than those with periampullary (74.29% vs. 57.79%, $P< 0.001$). ICU admission was more common among patients with AC (37.55% vs. 28.92%, $P=.004$). Patients with other PC were more likely to be admitted from the ED (77.19% vs. 58.37%, $P< 0.001$). The length of stay of patients with AC was higher (8.45±8.59 vs. 6.17±6.41 days, $P< 0.001$). Patients with AC were more likely to present with cholelithiasis (15.51% vs. 6.16%, $P< 0.001$), primary sclerosing cholangitis (PSC) (46.12% vs. 28.25%, $P< 0.001$), and to seek surgical treatment (79.18% vs. 49.94%, $P< 0.001$). They also presented with higher ALP (4.11.42±365.51 vs. 339.12±378.55, $P=0.004$) and total bilirubin (4.69±6.21 vs. 3.40±4.50, $P=0.002$). Compared to AC patients with the lowest income ($19,000-$50,000), the patients in the income group $70,000 to $100,000 had a length of stay 10.328 days shorter ($P=0.044$)
Conclusion: In our large retrospective study we show that patients with AC differ in both demographics and clinical characteristics compared to patients with other types of PC. These findings will help clinicians tailor their evaluation and allow more individualized management. We also show that socioeconomic level plays a substantial part in the outcomes of these patients, a topic which has yet to be previously studied.

BILIARY/PANCREAS

S1662 Presidential Poster Award

A Rare Case of Intraductal Papillary Mucinous Neoplasm of the Biliary Ducts With Malignant Degeneration into Cholangiocarcinoma

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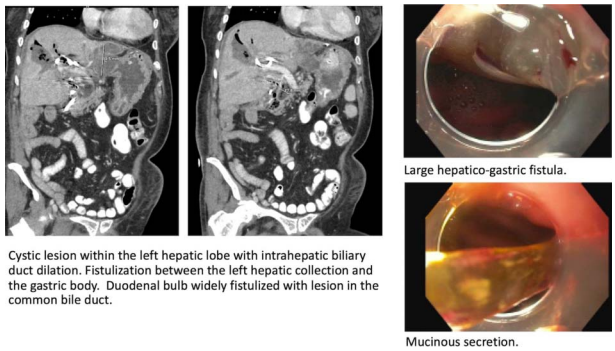
Introduction: Intraductal papillary mucinous neoplasm (IPMN) is a mucin-producing papillary epithelial neoplasm arising from the pancreaticobiliary system. IPMN of the pancreas (P-IPMN) are common and widely recognized. Biliary tract IPMNs (BT-IPMN) are much rarer and have higher malignant transformation potential. We present a case of BT-IPMN with biliary-gastric and biliary-duodenal fistulas and malignant degeneration.
Case Description/Methods: A 56-year-old Asian female with history of cholecystectomy and hyperlipidemia presented with abdominal pain and fever to an outside hospital, where she was diagnosed with cholangitis, and a large hepatic cystic lesion. A percutaneous biliary drain was placed and drained 500 cc of bilious fluid. The patient was transferred to us for higher level of care. Her cholestasis markers were elevated with a total bilirubin of 7.4 and alkaline phosphatase of 1195. CT showed intrahepatic ductal dilatation, a large fistula from the common bile duct to the duodenum, and a large (15.3 x 4.1 x 5 cm) intrahepatic cystic lesion with wide mouthed fistula tracts between the left hepatic biliary system and the stomach (Figure). ERCP showed copious amount of viscous mucin in the stomach originating from the large hepatico-gastric fistula. A second fistula extending from the duodenal bulb to the mid-proximal bile duct was seen and the same mucinous secretion was present. Stenting of the biliary tree was not performed due to the drainage of copious amount of highly viscous mucin. A regular upper endoscope was introduced through the duodenal and gastric fistulas to the extrahepatic bile duct and the left intrahepatic biliary ducts respectively. The biliary epithelium was abnormal with villous projections. Biopsies were taken. Pathology showed papillary projections lined by intestinal-type epithelium with low grade dysplasia, compatible with an intraductal papillary neoplasm. CT-guided biopsy of the liver cystic lesion showed adenocarcinoma of pancreaticobiliary origin. She was diagnosed with BT-IPMN with malignant degeneration into cholangiocarcinoma involving much of the central liver that was not amenable for surgical resection. The patient was started on chemotherapy.
Discussion: BT-IPMNs are rare and not well understood. There is no clear guideline available. Surgical resection is the treatment of choice when discovered early. Our case highlights the high malignancy potential of BT-IPMNs and a need for their better understanding.



[1662] **Figure 1.** CT and ERCP findings.

S1663 Presidential Poster Award

Diagnosis of Biliary Varices Using Digital Cholangioscopy: Uncommon Cause of Biliary Obstruction and Bleeding

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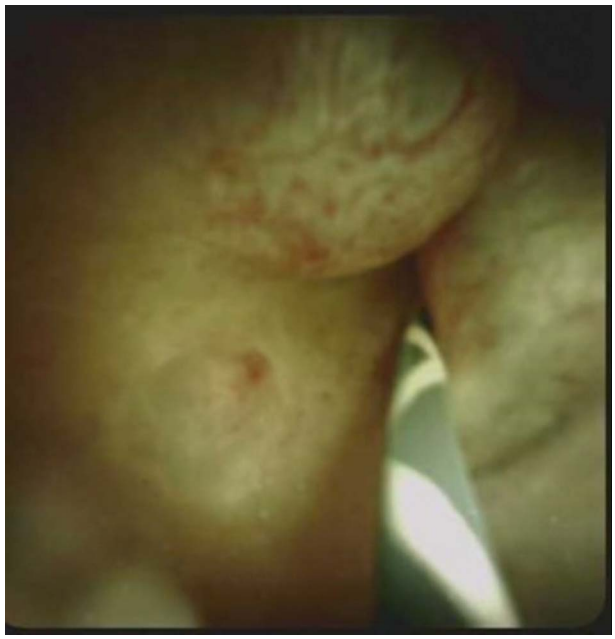
Introduction: Portal biliopathy is one of the rare complications of cirrhotic portal hypertension. We present a case of a patient with underlying cirrhosis presenting with biliary obstruction and bleeding in setting of biliary varices.
Case Description/Methods: A 60-year-old male with history of decompensated cirrhosis and heavy alcohol use was admitted for inpatient evaluation for liver transplant. His labs were remarkable for total bilirubin of 13.7, AST 66 of, ALT of 52 and alkaline phosphatase of 102. Patient reported several days of melena prior to admission but hemoglobin remained stable and there was no evidence of bleeding after admission. MRI abdomen completed as part of his pre-transplant workup showed severe intrahepatic biliary duct dilation with multifocal stricturing and beaded appearance of the bile ducts. Patient underwent endoscopic retrograde cholangiopancreatography (ERCP) for further evaluation. ERCP showed multiple filling defects on the cholangiogram that disappeared with contrast injection. Biliary sphincterotomy and balloon sweep of the biliary duct was negative other than blood clots. The filling defects remained in place despite multiple sweeps. Digital cholangioscopy (Spyglass) was introduced into bile duct that showed mucosal changes consistent with biliary varices (Figure). Four days post-procedure the patient developed melena with acute anemia. Repeat ERCP showed evidence of bleeding from sphincterotomy site and again biliary tree sweep revealed intraductal blood clots. Spyglass was introduced again with evidence of biliary varices without active bleeding. A covered metal stent was placed to help control post-sphincterotomy bleeding. The bleeding stabilized and the patient's total bilirubin dropped from 14.2 to 8.9 over the following 3 days. CT A/P showed patent vessels with no evidence of portal vein thrombosis.
Discussion: Portal biliopathy is a biliary abnormality caused by portal cavernous malformation and biliary varices in patients with portal hypertension[1]. Biliary varices have been reported to be more common in patients with portal vein thrombosis rather than cirrhotic portal hypertension[2,3]. Majority of patients are asymptomatic, but a smaller subset of patients can present with jaundice, fever, or strictures. This diagnosis can be frequently overlooked which highlights the need to keep a high index of suspicion for this rare diagnosis when the cause for biliary obstruction or bleeding in patients with cirrhosis is unclear.



[1663] **Figure 1.** Image of biliary varices noted during digital cholangioscopy procedure.

S1664 Presidential Poster Award

Icteric Leptospirosis Leading to Multiorgan Failure and Concomitant Pancreatitis

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Introduction: Leptospirosis is a zoonotic disease caused by *Leptospira interrogans* with relatively higher prevalence in tropical regions.[1] The clinical presentation of leptospirosis ranges from asymptomatic to multiorgan failure with complications including aseptic meningitis, renal failure, liver failure, pulmonary hemorrhage, acute respiratory distress syndrome, and dysrhythmias.[2] Cases of pancreatitis are rare.[3] We present a case of leptospirosis causing acute kidney and liver injury, dysrhythmias, and pancreatitis.
Case Description/Methods: A 36-year-old male with a history of schizoaffective disorder and polysubstance misuse presented with bilateral calf pain and pruritis for one week. He denied recent alcohol use or travel. Vitals were normal except for heart rate of 118 beats per minute. Exam revealed scleral icterus and calf tenderness. Laboratory findings showed leukocytosis, thrombocytopenia, elevated creatinine, transaminase elevation, hyperbilirubinemia, and elevated lipase (Table). Lower extremity ultrasounds were normal. Magnetic resonance cholangiopancreatography showed mild fatty infiltration without bile duct dilatation and edema around the pancreatic head and neck consistent with pancreatitis. The patient developed pleuritic chest pain with mild troponin elevation, B-type natriuretic peptide elevation, and ST segment elevations in the lateral leads (Table). He then developed anuria and hypotension requiring vasopressors, steroids, and hemodialysis. His bilirubin peaked at 40 milligrams/deciliter. Leptospirosis antigen and polymerase chain reaction (PCR) were obtained, and ceftriaxone was empirically started. He improved with normalization in hemodynamics, urine output, bilirubin, transaminases, and renal function. Leptospirosis antigen and PCR resulted positive.
Discussion: In reflection, our patient's chief complaint was bilateral calf tenderness which is a distinguishing physical finding.[2] He had signs of severe leptospirosis including renal, hepatic, and cardiac dysfunction. Interestingly, our patient also had pancreatitis. From 2002 to 2019, only 17 cases of leptospirosis-related pancreatitis have been published and were largely localized to Central Europe and Sri Lanka with just one case coming from North America.[3,4] This patient's life-threatening illness underscores the importance of recognizing the variable clinical presentation of leptospirosis, particularly in temperate climates with lower prevalence.

Table 1. Pertinent lab values

White blood cells	12,750 per microliter thousand per /ml
Platelets	57,000 per milliliter (ml)
Blood urea nitrogen (BUN)	91 milligrams/deciliter (mg/dl)
Creatinine	7.8 mg/dl
Aspartate aminotransferase (AST)	392 units/liter (U/L)
Alanine aminotransferase (ALT)	134 U/L
Albumin	3.1 grams/dl
Alkaline phosphatase	105 U/L
Total bilirubin	14.6 mg/dl
Direct bilirubin	9.1 mg/dl
International normalized ratio (INR)	1.15
Lipase	912 U/L
Creatinine kinase	7000 U/L
Acetaminophen level	undetec
Viral hepatitis panel	negative
Troponin	0.059 nanograms (ng)/ml
B-type natriuretic peptide (BNP)	38,400 picograms (pg)/ml

S1665 Presidential Poster Award

Hypertriglyceridemia Independent Propofol-Induced Acute Pancreatitis—A Rare and Unusual Complication

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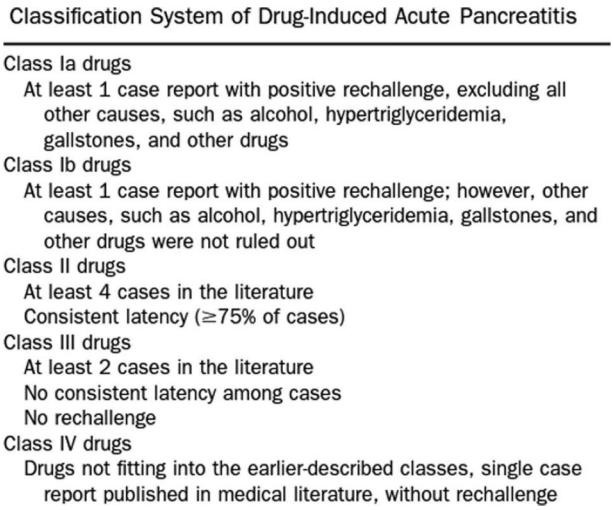
Introduction: Propofol is a widely utilized lipophilic sedative anesthetic. Common side effects include hypotension, apnea, and rash. While rare, acute pancreatitis (AP) is a documented complication of propofol administration. Though the exact mechanism has yet to be established, current theories include propofol-induced hypertriglyceridemia (HTG) or idiosyncratic hypersensitivity reaction. We present a rare case of HTG-independent propofol-induced AP after an elective rhytidectomy.
Case Description/Methods: A 75-year-old woman with hypertension and hypothyroidism, presented with 4 days of worsening mentation, abdominal distention and pain after an outpatient rhytidectomy. She was a former smoker, but denied alcohol or illicit drug use. On arrival, vital signs were significant for fever of 100.4F and tachycardia to 106 beats/min. Physical exam was significant for abdominal distension and epigastric tenderness. Laboratory studies revealed leukocytosis of 12,200 mm^3 with 10.4% bandemia, alkaline phosphatase of 143 U/L, serum calcium of 8.9 mg/dL, lipase of 222 U/L, and triglycerides of 111 mg/dL. Abdominal ultrasound was unremarkable without evidence of gallstones or biliary dilatation. A computed tomography of the abdomen revealed extensive peripancreatic fat stranding and fluid with homogenous parenchymal enhancement consistent with severe interstitial edematous AP. An infectious workup was unremarkable for a concomitant source. Medication reconciliation revealed no confounding medications known to cause pancreatitis. She received intravenous fluids, proton pump inhibitor and pain regimen. Her diet was gradually advanced and she clinically improved over the next 48 hours with mentation returning to baseline. She was subsequently discharged home.
Discussion: AP is responsible for over 230,000 hospitalizations annually in the US, with drug-induced AP comprising 0.5-2% of all cases. Propofol-induced AP has been sparsely reported in the setting of HTG, though exceedingly rare without this lab abnormality. The Badalov classification system differentiates association and causality of drug-induced AP by evaluating latency, rechallenge, and published evidence (Figure). Within this system, propofol has traditionally been characterized as a class II drug. However, recent published evidence has shown recurrence of AP upon propofol rechallenge, suggesting a more causal relation and a reclassification to class Ib. We present this case to raise awareness of the possible complications of propofol administration.



[1665] **Figure 1.** The Badalov Classification System of Drug-Induced Acute Pancreatitis.

S1666 Presidential Poster Award

Hepatolithiasis Caused by Right Hepatic Artery Branches Forming an Arterial Ring Compressing the Common Hepatic Duct

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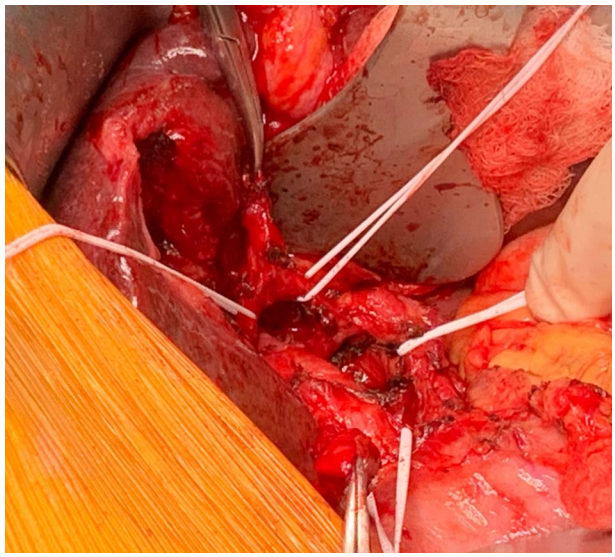
Introduction: Anatomic variants of the hepatic artery are common, but are generally of no physiologic significance. We present a patient with an early branching of the right hepatic artery (RHA), wherein the right anterior and right posterior sectoral arteries encircled and compressed the CHD, resulting in proximal hepatolithiasis.
Case Description/Methods: A healthy 51-year-old male presented with abdominal pain, jaundice and fever. Computed tomography of the abdomen showed bilateral intrahepatic duct stones and a focal CHD stricture near its bifurcation. At endoscopic retrograde cholangiopancreatography, the stricture was dilated and a plastic biliary stent was inserted. Since the hepatolithiasis could not be cleared endoscopically, surgical common bile duct exploration was performed (Figure). At laparotomy, we identified an early bifurcation of the RHA. The right anterior artery crossed anterior to the CHD, whereas the right posterior artery coursed posterior to the CHD. These arterial branches were densely adherent to and circumferentially constricting the CHD. We performed an arterial divestment to release the anterior and posterior RHA branches from the CHD. We transected the CHD 1cm distal to its bifurcation and transposed the anterior RHA branch posterior to the CHD. We used Spyglass cholangioscopy to guide clearance of the hepatolithiasis and to evaluate the biliary epithelium. The CHD was reconstructed in an end-to-end fashion over plastic biliary stents. The patient had an uneventful postoperative course. Post operative ERCP demonstrated no evidence of stones or biliary stricture. The patient remains asymptomatic to date.
Discussion: This case illustrates a rare but clinically important phenomenon of CHD compression within an arterial ring formed by the anterior and posterior sectoral branches of an early branching RHA. Tsuchiya *et al.* reported the first case of CHD compression caused by an anterior-crossing RHA, and coined this entity the "right hepatic artery syndrome". Since then, 10 additional cases of CHD compression caused by topographical variants of the hepatic artery have been described. Most patients underwent surgery for bile duct exploration and bilioenteric drainage, or to release the artery from the CHD. Both surgical approaches provide a high rate of durable symptom resolution. In recent years, there have been reports describing purely endoscopic management of this condition using ERCP or direct cholangioscopy to clear intrahepatic stones.



[1666] **Figure 1.** Relationship between the common hepatic duct (asterisk) and the mobilized anterior (solid arrow) and posterior (dotted arrow) right hepatic artery branches.

S1667 Presidential Poster Award

Isolated Intramural Gastric Metastasis of Pancreatic Ductal Adenocarcinoma (PDAC) Detected on Surveillance Esophagogastroduodenoscopy (EGD) Before Endoscopic Ultrasound (EUS) Guided Biopsy

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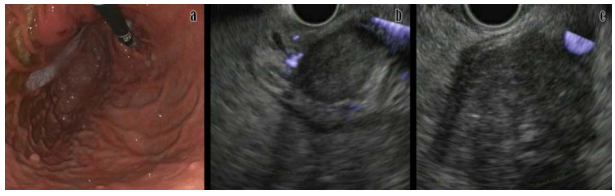
Introduction: PDAC is an aggressive malignancy that requires prompt diagnosis and treatment to provide the patient with the best chance of long-term survival. Patients who have an imaging-confirmed solitary pancreatic mass generally undergo a EUS-guided biopsy for histological confirmation of the diagnosis. We describe a rare case of solitary intramural gastric metastases discovered on surveillance EGD prior to EUS-guided biopsy.
Case Description/Methods: 63-year-old male with a past medical history of hypertension, chronic pancreatitis, and tobacco use presented with 2 months history of epigastric pain, and 5lbs weight loss. He denied jaundice, loss of appetite, pale-colored stools, or generalized itching. Initial CT abdomen showed a 2.7cm x2.6cm mass in the pancreas body with associated proximal pancreatic duct dilatation and atrophy, suspicious of pancreatic neoplasm. Ca19-9 was 111, and CEA was 1.1. No evidence of metastasis was found on the CT chest/abdomen. EUS-guided biopsy of the mass was scheduled. At the beginning of the procedure, a surveillance EGD was performed which showed a 2cm submucosal, non-circumferential mass in the gastric fundus (Figure a). EUS was performed next which confirmed a 2.1 cm intramural mass in the gastric fundus originating from the muscularis propria (Figure b), and a 3.9cm x 3.8cm mass in the pancreatic body with invasion into superior mesenteric vein, splenic vein, and splenoportal confluence with no noted arterial involvement (Figure c). EUS-guided fine needle biopsy of both lesions was obtained using separate needles. Histopathology from both biopsies demonstrated invasive adenocarcinoma, histomorphologically similar and suggestive of a pancreatic primary. This changed the staging of the patient from borderline resectable to metastatic and he was referred for palliative chemotherapy.
Discussion: Isolated gastric metastasis of PDAC is extremely rare, and documented cases in the literature are from a prior diagnostic biopsy that created a seeding tract. Our case highlights 2 major points: 1) Though rare, isolated intramural gastric metastasis can occur and can be missed on cross-sectional imaging. 2) Currently there is no consensus or guideline that mandates an EGD prior to EUS. In this case, this significant finding which changed the staging, treatment, and prognosis of the patient could have been missed with an oblique viewing EUS scope. At our center, we routinely perform EGD prior to EUS. Prospective studies are needed to confirm the utility of this practice.



[1667] **Figure 1.** a: EGD demonstrating 2cm submucosal, non-circumferential mass in the gastric fundus. b: EUS demonstrating a 2.1 cm intramural mass in the gastric fundus originating from the muscularis propria. c: EUS demonstrating 3.9-cm x 3.8-cm mass in the pancreatic body with invasion into the superior mesenteric vein, splenic vein, and splenoportal confluence with no noted arterial involvement.

S1668 Presidential Poster Award

Intrahepatic Biliary Colloid Carcinoma: A Rare Site for Recurrence

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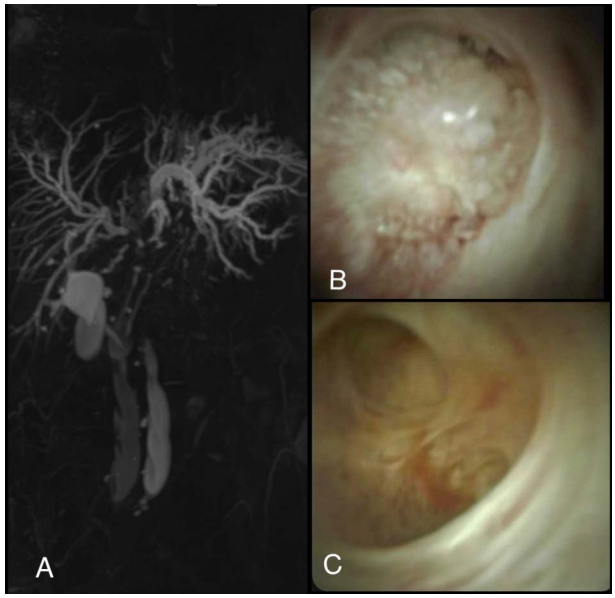
Introduction: Colloid carcinoma (CC) of the pancreas is a rare variant of ductal adenocarcinoma (DAC) and represents 1% of pancreatic carcinomas. Nearly all CC cases develop from intrapapillary mucinous neoplasm (IPMN). CC is also recognized as a histological subtype of invasive IPMN and its differentiation from IPMN can be challenging. CC has been shown to have better overall survival than DAC but its recurrence rate has not been established. Also, the liver is the most common site of distant metastases in pancreatic invasive IPMN but its frequency has not been described in CC metastases. To our knowledge, no other case of pancreatic CC with multiple liver metastatic lesions and intrahepatic biliary duct (BD) recurrence has been reported.
Case Description/Methods: A 72-year-old female with history of stage IV pancreatic CC with metastases to liver, spleen, and stomach presented with dark urine and jaundice. Diagnosed 5 years earlier, requiring distal pancreatectomy, splenectomy, and partial gastrectomy. At that time, pathology showed poorly differentiated CC of the pancreas invading into the stomach with negative surgical margins. Unfortunately, one month later, metastatic liver lesions were found. She failed Gemzar, Abraxane, liver CT-guided microwave ablation and FOLFOX therapy. Pembrolizumab was started for disease progression and showed stable disease for 2 years but it was stopped due to neutropenia. Two years after the last dose of immunotherapy, MRCP showed new moderate left intrahepatic duct dilation with left hepatic duct narrowing. She underwent ERCP twice, requiring biliary stent placement and brush cytology with benign results. Discussion at the multidisciplinary tumor board led to a consensus opinion of IPMN-B. During

the most recent hospitalization, labs showed elevated liver function studies with a total bilirubin of 4 mg/dl. ERCP was done with cholangioscopy. The left main hepatic duct was partially obstructed by a large frondlike mass. It was biopsied and pathology reported as CC (Figure).

Discussion: Our case highlights the difficulties of distinguishing IPMN-B from CC of the BD. A new BD stricture raised questions about the presence of malignancy, especially with the history of metastatic pancreatic cancer. The cholangioscopy findings and the pathology report should make us investigate the best strategy for cancer surveillance since brush cytology can often be negative. Further studies will be needed to address the recurrence and metastatic rates in pancreatic CC.



[1668] **Figure 1.** A) MRCP with left intrahepatic BD dilation. B) Frondlike mass in the left hepatic main duct and C) normal right hepatic main duct under cholangioscopy.

S1669 Presidential Poster Award

Pancreatitis-Related Purtscher's-Like Retinopathy Presenting With Visual Hallucinations

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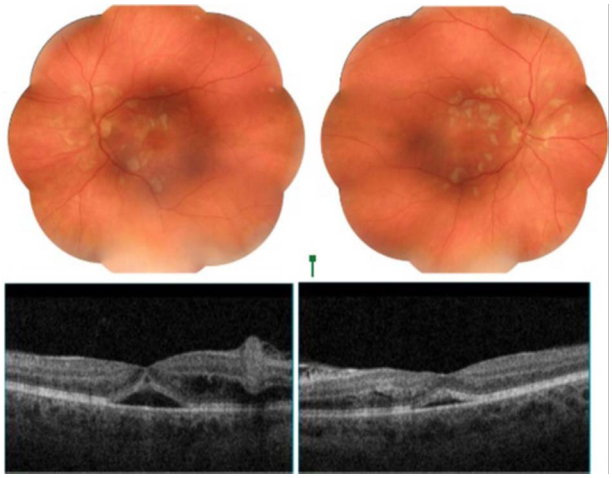
Introduction: One of the most undiagnosed complications of pancreatitis is the development of Purtscher's like retinopathy. As a result of diffuse complement activation and leuko-embolization patients will experience blurry vision which can oftentimes be permanent. This usually manifests with sudden onset painless vision loss however, in rare instances, visual hallucinations may be the initial sign. We present a case of a young woman with Purtscher's like retinopathy from pancreatitis who's initial symptom was visual hallucinations. Recognizing the signs, causes and the overall course of this disease are integral in preventing permanent vision loss.

Case Description/Methods: A 38-year female with a PMH of alcoholic pancreatitis presented with 2 days of severe persistent epigastric pain. Vital signs and laboratory tests were normal excluding a lipase of 5000 U/L. CT scan showed acute interstitial pancreatitis. Supportive care was employed. The abdominal pain resolved on the second day. On the 3rd day, the patient experienced acute onset visual hallucinations. She reported seeing "imaginary people", "chairs are moving across the room", and "1 person appears as 3 separate people". There was no evidence of alcohol withdrawal. Later that evening, the patient reported sudden onset bilateral vision loss. CT scan of the head was unremarkable. Ophthalmology evaluation showed severely diminished visual acuity. Dilated fundoscopic exam revealed bilateral multiple peripapillary cotton wool spots (CWS) and polygonal shaped areas of retinal whitening (Purtscher flecken), making a diagnosis of Purtscher's like retinopathy. Optical coherence tomography (OCT) scan was performed 2 weeks later revealing CWS and bilateral macular edema (Figure). No treatment was given. The visual hallucinations and blurry vision gradually improved over 1 month. 2 months after the diagnosis, her visual acuity had returned to baseline.

Discussion: Purtscher's like retinopathy is a severe vaso-occlusive vasculopathy. Severe systemic illnesses or traumas trigger complement activation and leukocyte embolization causing arteriolar occlusions in the retina. This has been associated with numerous gastrointestinal related illnesses such as: acute pancreatitis, pancreatic adenocarcinoma, cryoglobulinemia, and hemolytic uremic syndrome. It is imperative to be aware of the symptoms and to treat the underlying condition in Purtscher-like retinopathy to prevent further damage to the retina. At this time, no ocular treatment is recommended.



[1669] **Figure 1.** Optical coherence tomography showing bilateral cotton wool spots (Top) and bilateral macular edema (Bottom) 2 weeks after initial diagnosis.

S1670 WITHDRAWN

S1671 Presidential Poster Award

Sarcoidosis Masquerading as Pancreatic Adenocarcinoma: A Case Report

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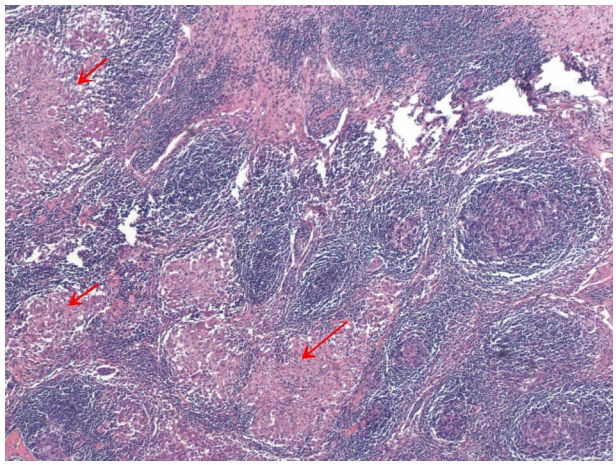
Introduction: Sarcoidosis is a multisystem, idiopathic disease characterized by granulomatous inflammation. Symptomatic involvement of the GI tract occurs in less than 1% of cases, and of those, the stomach is most involved. Pancreatic involvement is exceedingly rare. The diagnosis of pancreatic sarcoidosis remains difficult, as pancreatic involvement may manifest as direct invasion of the organ, obstruction of the pancreatic or biliary ducts, or as a mimicker of pancreatic adenocarcinoma. We present a rare case of suspected pancreatic malignancy revealed as pancreatic sarcoid after surgical resection, in a patient without known history of sarcoidosis.
Case Description/Methods: A 50-year-old male presented to the GI clinic reporting 3-4 years of epigastric pain with 30 pound weight loss. CT demonstrated a 2.6 cm mass-like lesion with infiltrative margins in the pancreatic head and uncinate process with mild biliary and pancreatic ductal dilation, highly suspicious for pancreatic adenocarcinoma. He twice underwent EUS which each revealed biliary dilatation with a mass in the head of the pancreas, with pathology showing chronic inflammation. Follow up PET scan, however, revealed a lesion in the pancreatic head that had mild FDG uptake suspicious for malignancy. Patient underwent Whipple without complication. Pathology was fortunately negative for malignancy, however revealed granulomatous inflammation in the pancreas (Figure). Findings suggestive of sarcoidosis. At follow up, patient had no evident additional manifestations of sarcoidosis.
Discussion: Pancreatic involvement in sarcoidosis can manifest in many ways, including mimicking pancreatic adenocarcinoma. Given that pancreatic cancer typically portends a poor prognosis, there is greater urgency in diagnosis and treatment for suspicious pancreatic masses. A high degree of clinical suspicion is necessary to consider sarcoidosis in this setting. Our case was particularly interesting because this patient had no personal or family history of sarcoidosis and experienced no other signs or symptoms of sarcoidosis after his Whipple procedure. This patient had 2 separate EUS procedures with no evidence of malignancy, however a PET scan was concerning for pancreatic malignancy. This provided a difficult clinical circumstance, as this patient suffered from debilitating abdominal pain with no definitive diagnosis and thus no definitive treatment option. Ultimately, patient underwent Whipple procedure with relief of symptoms, and a situationally fortunate diagnosis of sarcoidosis.



[1671] **Figure 1.** Pathology slide revealing non-necrotizing granulomas within a peripancreatic lymph node.

S1672 Presidential Poster Award

A Novel Approach to Obtaining Tissue in a Difficult to Access Indeterminate Biliary Stricture: Percutaneous Cholangioscopy and Biopsy

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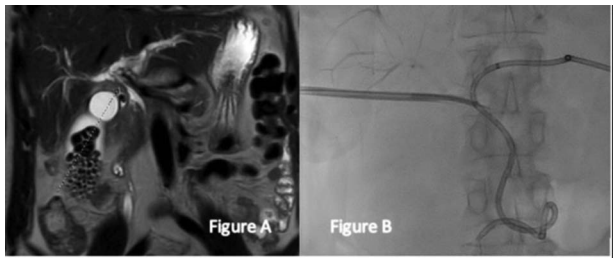
University of Rochester Medical Center, Rochester, NY.

Introduction: When evaluating biliary strictures, establishing a diagnosis can present challenges. We present a case of percutaneous transhepatic cholangioscopy (PTCS) using the Spyglass (SG) DS cholangioscope, resulting in a definitive diagnosis of a cholangiocarcinoma after previous failed attempts by several standard methods.
Case Description/Methods: A 59-year-old female presented with one week of epigastric pain, jaundice and pruritus. Alkaline phosphatase, direct bilirubin and CA 19-9 were elevated. Magnetic resonance imaging (MRI) showed intrahepatic biliary ductal dilation with cutoff near the hepatic hilum, concerning for cholangiocarcinoma (Figure A). Endoscopic retrograde cholangiopancreatography (ERCP) showed luminal narrowing in the duodenal sweep with edema and ulcerations, and biopsies from stricture showed small bowel inflammation. Interventional radiology (IR) performed direct biliary duct biopsy that showed atypical epithelial cells. Endoscopic ultrasound (EUS) with fine needle aspiration (FNA) of a hypoechoic soft tissue area in the gallbladder neck showed acute inflammation. To ultimately reach a diagnosis, percutaneous access was obtained by IR and a SG DS cholangioscope was advanced percutaneously through the IR placed sheath. The common hepatic duct stricture was accessed from above to obtain biopsies, which were positive for adenocarcinoma (Figure B). The patient was then started on chemotherapy.
Discussion: While ERCP with brushings is the first line method to evaluate biliary strictures, tissue yield has low sensitivity (41.6%). Tortuous duodenal or cholangial anatomy can also prevent access to the target area. EUS-FNA has the highest tissue sensitivity at 93.8% but can also present anatomic restrictions. Traditionally, PTCS has been the answer for biopsies unable to be obtained with the modalities above but requires large tract dilation and days of sinus tract maturation to allow for a scope. SG assisted PCTS has been reported to have superior sensitivity, smaller dilation requirement, and less adverse events when compared to traditional PCTS. However, reports of SG with a transcutaneous approach are extremely limited. Our case highlights a multi-disciplinary approach with SG assisted PTCS in diagnosing malignancy when standard methods were unsuccessful, positioning SG as an important tool for future evaluation of indeterminate biliary stricture.



[1672] **Figure 1.** (A) MRI showing intrahepatic ductal dilation with cutoff at the hilum and prominent perihilar soft tissue. (B) showing Spyglass DS cholangioscope advancing through PTC to obtain biopsies from hilar stricture.

S1673 Presidential Poster Award

Acute Pancreatitis Secondary to Pembrolizumab-Induced Hypertriglyceridemia: First Clinical Experience

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[1]Allama Iqbal Medical College, Lahore, Punjab, Pakistan; [2]Wexham Park Hospital, Slough, England, United Kingdom; [3]Woodhull Medical Center, Brooklyn, NY; [4]Hackensack Meridian Raritan Bay Medical Center, Perth Amboy, NJ; [5]Marshfield Medical Center, Marshfield, WI; [6]Woodhull Medical Center/NYC Health + Hospitals, Brooklyn, NY; [7]Orlando Health Cancer Institute, Orlando, FL.

Introduction: Pembrolizumab is a programmed cell death receptor-1 (PD-1) inhibitor that has revolutionized therapeutics in several advanced cancers. A plethora of immune-related adverse events have also been reported, including pneumonitis, colitis, hepatitis, and autoimmune diabetes. Pembrolizumab-induced hypertriglyceridemia (HTG) has also been reported, but acute pancreatitis secondary to this complication remains an unusual entity. To our knowledge, our patient is the first case of acute pancreatitis with pembrolizumab-related severe HTG as the probable etiological factor.
Case Description/Methods: A 39-year-old woman with stage IVB non-small-cell lung cancer presented with progressive abdominal pain and nausea, 14 days after receiving a pembrolizumab-based chemotherapy cycle. Her lipid profile was normal before pembrolizumab initiation 4 months ago. Laboratory studies revealed serum lipase 12562 IU/L and triglyceride 16901 mg/dL, with significantly elevated HbA1c and deranged liver and renal function test results (Table). As per the Revised Atlanta Classification, the patient was diagnosed with acute pancreatitis as she fulfilled all 3 criteria. After careful exclusion of alternative etiologies, pembrolizumab-induced HTG was considered as the probable cause. She was then transferred to the ICU for treatment with fluid replacement and insulin infusion. However, HTG did not respond to conservative treatment with insulin infusion. Subsequently, she received 2 cycles of therapeutic plasma exchange. The patient recovered well with no complications.
Discussion: The exact pathogenesis of pembrolizumab-induced HTG remains unclear. However, the deficiency or autoantibodies targeting GP1HBP1 may have a role by halting lipoprotein lipase (LPL) to reach capillary lumen. It causes low LPL levels and deranged intravascular degradation of triglycerides, culminating in HTG. This article has pertinent clinical implications due to the widespread use of immune checkpoint inhibitors. Community awareness regarding different presentations of such rare adverse events is imperative for clinical management in order to avoid morbidity and mortality. HTG can be treated with insulin infusion or therapeutic plasma exchange. Monitoring of serum triglyceride levels at baseline, during, and after pembrolizumab therapy can be considered, particularly in patients with diabetes and lipid disorders. Therefore, clinicians should remain vigilant for acute pancreatitis as it can be a life-threatening adverse event of immune-checkpoint inhibitors.

Table 1. Laboratory data of the patient at the initial presentation showing remarkably deranged results

Laboratory parameter	Patient value	Reference range
Lipase	12562	23-300 IU/L
Triglycerides	16901	10-150 mg/dL
Total cholesterol	1387	< 200 mg/dL
Low-density lipoprotein	1016	50-100 mg/dL
Alanine aminotransferase	385	0-34 IU/L
Aspartate aminotransferase	243	15-46 IU/L
Alkaline phosphatase	618	45-140 mg/dL
Total bilirubin	2.8	< 1.2 mg/dL
Sodium	118	136-145 mmol/L
Chloride	86	98-107 mmol/L
Bicorbonate	16.8	23-29 mmol/L
Creatinine	1.4	0.6-1.3 mg/dL
Hemoglobin A1c	11.4	≤6.5%

S1674 Presidential Poster Award

An Unusual Cause of Septic Shock Secondary to Lemmel Syndrome

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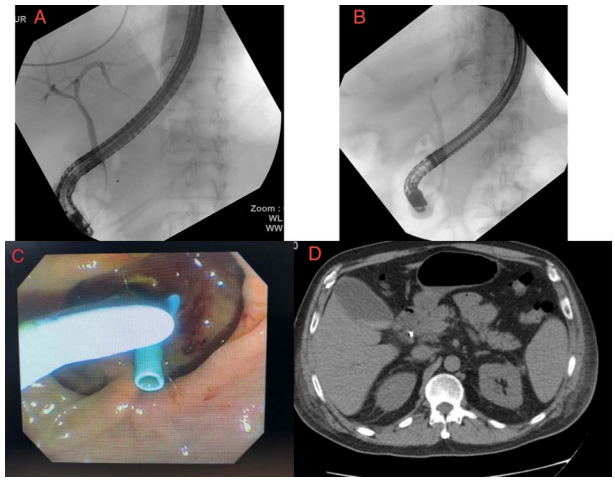
[1]Saint Michael's Medical Center, New York Medical College, Newark, NJ; [2]St. Georges University, Linden, NJ.

Introduction: Lemmel syndrome (LS) is a rare cause of obstructive jaundice occurring in the absence of pancreaticobiliary tumors or choledocolithiasis and commonly caused by periampullary diverticulum (PAD) with an incidence of 1-27%. They are typically asymptomatic and underreported leading to complications in ~5% of cases. Herein, we report a case of septic shock due to acute cholangitis secondary to Lemmel Syndrome.
Case Description/Methods: A 58-year-old male with a medical history significant for hypertension who presented to the ER with right upper quadrant abdominal pain, jaundice, and multiple episodes of nonbloody-nonbilious emesis. On admission, the patient was febrile, tachycardic and hypotensive. Initial laboratory tests are shown in the Table. CT of the abdomen showed cholelithiasis and diffuse gallbladder thickening with no intra or extrahepatic biliary duct dilation. He developed septic shock requiring vasopressors. Blood cultures were positive for *Escherichia coli*, and antibiotics were administered. Emergent ERCP was performed due to the presence of acute cholangitis. He was found to have a large diverticulum located around the major ampulla. Double wire technique was performed to access the ventral pancreatic duct (VPD) and common bile duct (CBD). Fluoroscopic images revealed biliary tapering in the distal third but no visualized stones (Figure). Plastic stents were placed into the VPD and CBD and biliary sphincterotomy was performed. He also underwent laparoscopic cholecystectomy. The patient was discharged with resolution of symptoms.
Discussion: The pathophysiology of LS is due to direct mechanical irritation of the diverticulum causing papillary fibrosis. Secondly, the diverticulum may cause sphincter of Oddi dysfunction. Thirdly, the distal CBD may be directly compressed by the periampullary diverticulum. The imaging modalities of choice to confirm the diagnosis include enhanced contrast CT and MRCP. ERCP allows for direct visualization of the duodenal diverticulum. Treatment of these entities is required if the patient becomes symptomatic. The mainstay of treatment is endoscopic sphincterotomy with stent placement or papillary balloon dilation. Due to the high recurrence rate, if endoscopic treatment fails then surgical approach via diverticulectomy is performed. It is important physicians remain vigilant regarding this condition since misdiagnosis leads to delay in therapy. The incidence of septic shock in LS is unknown and to our knowledge, there has only been one other reported case in literature.



[1674] **Figure 1.** A: Cholangiogram of biliary tree with no stones B: Cholangiogram with diverticulum, stent in place and no obvious stones C: Periampullary diverticulum after placement of CBD and pancreatic stent D: CT abdomen showing CBD stent in place with gallbladder sludge.

Table 1. Initial Laboratory Values

Blood Chemistry		Reference Range
Sodium	137 mmol/L	136 - 145 mmol/L
Potassium	3.8 mmol/L	3.5 - 5.3 mmol/L
Blood urea nitrogen (BUN)	10.0 mg/dL	6 - 24 mg/dL
Creatinine	1.0 mg/dL	0.6 - 1.2 mg/dL
Lactic acid	5.3 mmol/L	0 – 2.0 mmol/L
C-reactive protein	21.8 mg/dL	0.0-0.8 mg/dL
TSH	1.1180 μIU/mL	0.400 - 4.500 μIU/mL
Total bilirubin	3.3 mg/dL	0.2–1.2 mg/dL
Direct bilirubin	1.94 mg/dL	0.0-0.3 mg/dL
AST	487 U/L	10-36 U/L
ALT	365 U/L	9-46 U/L
Alkaline phosphatase	155 U/L	40-115 U/L
Complete blood count		
White blood cell (WBC)	4.3x10 3/ uL	4.4 - 11 × 103/μL
Hemoglobin	12.2 g/dL	13.5 - 17.5 g/dL
Platelet	107 x103 /uL	150 - 450 × 103/μL

S1675

Blame It on the Bamboo Stump: A Pancreatic Ductal Disruption Successfully Managed With Two-Step Endoscopic Therapy

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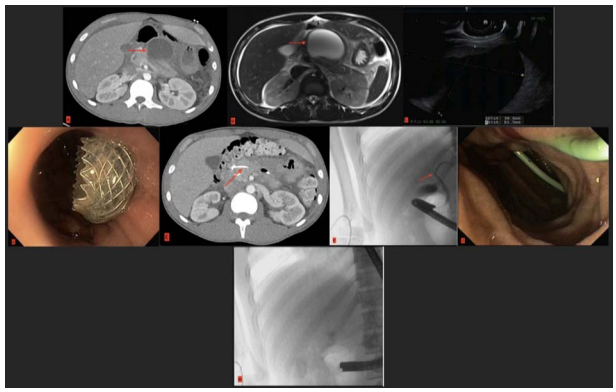
Introduction: Non-penetrating, isolated pancreatic injuries are rare, and the management is not well-established and challenging. Most injuries are managed by surgery, but recently several case reports discuss endoscopic therapy for treatment of main pancreatic duct disruption. We report a case of pancreatic injury from falling on a bamboo stump with complete pancreatic ductal disruption and complete pancreas divisum with a dominant dorsal duct that was successfully nonoperatively managed through endoscopic therapy.
Case Description/Methods: An 18-year-old male transferred for worsening abdominal pain from a grade IV pancreatic injury complicated by pancreatic ascites and massive pancreatic fluid collections (PFCs) sustained a month prior after a non-penetrating fall onto a bamboo rod in the Caribbean. He had failed conservative therapy and 2 attempts with ERCP for pancreatic duct (PD) stent placement. CT A/P and MRCP showed a 13cm cyst consistent with hemorrhagic pancreatic pseudocyst (Figure A-B). Labs showed leukocytosis, elevated lipase and LFTs, but he was hemodynamically stable. US-guided paracentesis drained 250mL of fluid consistent with pancreatic ascites. To allow for pancreatic edema reduction, we elected for a 2-step procedure with separate EUS and ERCP. EUS showed multiple PFCs s/*P* endoscopic cyst-gastrostomy with Axios stent (Figure C-D). Repeat CT A/P showed decrease in size of the PFCs (Figure E). He was discharged and returned in one week for ERCP that revealed a surprising complete pancreas divisum and total disrupted main dorsal PD, which was endoscopically treated with sphincterotomy of the minor papilla and a long PD stent across the disruption (Figure F-H). The Axios stent was removed a week later, and 4 weeks later we successfully removed the PD stents from both the major and minor papillae. He made an uneventful recovery and remains well.
Discussion: Pancreatic disruption syndrome or Disconnected Pancreatic Duct Syndrome (DPDS) is an interruption of pancreatic duct continuity, usually from necrotizing pancreatitis, but can occur with blunt or penetrating abdominal injury, as in our patient. Endoscopic management is a minimally invasive option for DPDS and pancreatic ascites. Our patient had severe pancreatic injury managed using 2-stage endoscopic therapy, demonstrating a successful treatment strategy for pancreatic injury with total main DPDS. Further studies are required regarding the selection of patients, safety, and long-term outcomes.



[1675] **Figure 1.** Image A: CT A/P (coronal) demonstrating edematous pancreas with significant peripancreatic fat stranding, with a peripherally enhancing hypodense fluid collection near the body and tail of the pancreas measuring approximately 4.0 x 4.8 x 13 cm in the maximum AP, RL and SI dimensions (red arrow). Image B: MRCP (coronal) Abutting the pancreatic body is a multilobulated rim-enhancing complex fluid collection that extends from the level of the diaphragmatic hiatus to just above the level of the aortoiliac bifurcation and measures 6.8 x 9.7 x 19.1 cm (AP by TR by SI, red arrow). Image C-D: EUS demonstrating large pancreatic fluid collection (PFC, left) and s/*P* Axios cyst-gastrostomy (right). Image E: CT A/P (coronal) demonstrating cyst-gastrostomy placement with interval decrease in PFC. Image F-H: Pancreatogram showed extravastion of contrast material at the junction of proximal 2/3 and distal1/3 of the dorsal PD c/w disrupted dorsal PD (Complete Pancreas divisum, left). Placement of a 5 Fr 9 cm Zimmon PD stent with pigtail crossing the disruption at the minor papilla (center). Pancreatogram showing no extravasation of contrast material s/*P* PD stent placement.

S1676

Things Are Not Always What They Seem: A Rare Case of Pancreatic Carcinoma Manifesting Through Cutaneous Metastasis

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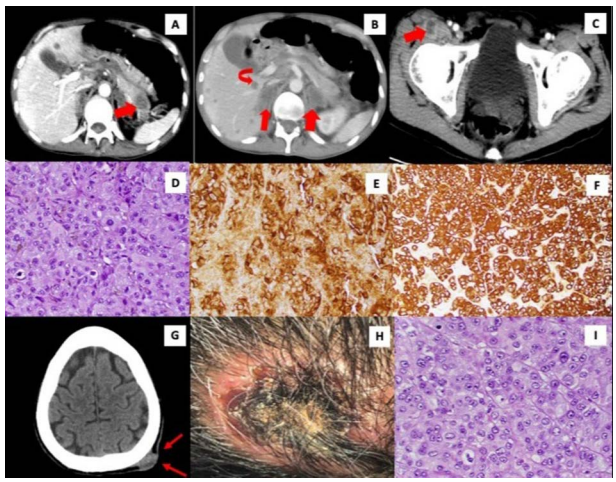
Introduction: Pancreatic cancer is an aggressive disease that commonly metastasizes to the liver, lung, and peritoneum. Cutaneous metastasis is a rare occurrence with an incidence of only 2.0%. We present a case of metastatic pancreatic adenocarcinoma primarily presenting as a cutaneous nodule.
Case Description/Methods: A 50-year-old male was undergoing outpatient evaluation for a raised, left scalp lesion along with neck nodules. Associated symptoms included an unintentional 30lb weight loss. Outpatient CT soft tissue neck revealed numerous necrotic lateral neck lymph nodes, lytic lesion with epidural soft tissue extension at C2-4, and occlusion/thrombosis of the left subclavian, brachiocephalic, and left internal jugular veins (Figure). Given these findings, the patient was referred to the Emergency Department (ED). At time of ED arrival, vital signs were stable along with initial laboratory values. CTA PE study showed no pulmonary embolism but revealed numerous pulmonary and bilateral adrenal nodules, multifocal liver and osseous lesions, and widespread lymphadenopathy. Additionally, CT head demonstrated a left scalp soft tissue lesion. Follow up CT abdomen/pelvis revealed a 2.8 x 2.2 x 1.8 cm ill-defined pancreatic lesion and confirmed widespread metastasis including to the muscles. Additional testing revealed elevated cancer markers with Cancer antigen 19-9 (CA 19-9) of 5418 U/mL and carcinoembryonic antigen (CEA) of 63.8 ng/mL. Pathology from initial scalp lesion biopsy showed fragments of necrotic dermis with small foci of poorly differentiated carcinoma. Biopsies of the right and left neck masses revealed poorly differentiated adenocarcinoma of uncertain primary. Therefore, liver biopsy was performed and confirmed non-small cell carcinoma of pancreatobiliary origin. Further imaging to determine treatment course discovered intracranial metastasis and cervical spine disease resulting in cord compression. Ultimately, the patient underwent 4 out of 10 fractions of palliative radiation to the cervical spine but passed away after transitioning to comfort care one month after diagnosis.
Discussion: There are less than 25 documented cases of pancreatic cancer with cutaneous involvement; however, it is even more rare that cutaneous involvement prompts initial diagnosis. The umbilicus is the most common site of cutaneous involvement and is referred to as the Sister Mary Joseph Nodule. Involvement of the soft tissue of the scalp and neck is much less common and poorly documented making this case extremely unique.



[1676] **Figure 1.** Axial contrast enhanced CT of the abdomen shows a 2.8 cm ill-defined hypoattenuating mass in the pancreatic tail (A) with multifocal metastatic liver lesions and bilateral adrenal metastasis (B). Axial CT image of the pelvis shows a metastatic lesion in the right rectus femoris muscle (C). On liver biopsy, H&E, 40X,(S21-41666) revealed high grade carcinoma with vesiculated nuclei, prominent nucleoli (D), CA 19-9 IHC 20X diffuse positivity in tumor cells (E) and CK AE1/3 IHC, 20X, diffusely positivee for pancytokeratin in tumor cells confirming carcinoma (F). Axial CT of the head shows a 2.4 cm nodular left parietal scalp soft tissue lesion (G) which was also seen on clinical examination (H). Biopsy of the scalp lesion (H&E, 40X) demonstrated high grade carcinoma with vesiculated nuclei, prominent nucleoli and mitoses (I), histologically similar to liver biopsy findings.

S1677

A Case of Bilioptysis After Transarterial Radioembolization of Primary Cholangiocarcinoma

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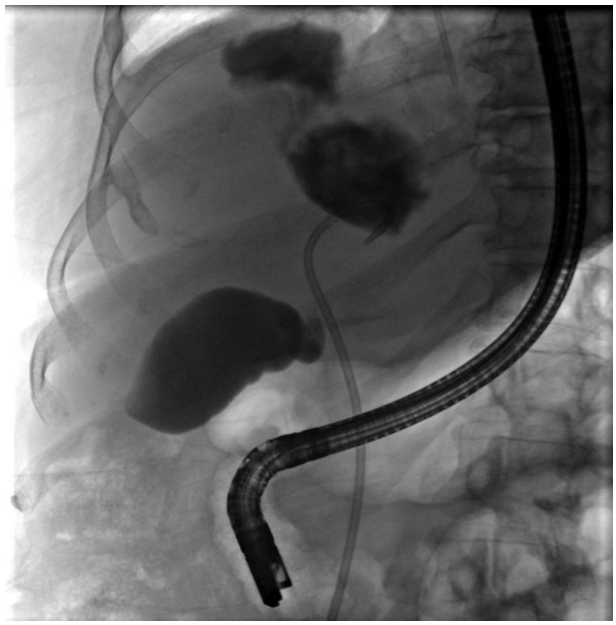
Introduction: Bilioptysis is a rare clinical finding that can be alarming to patients. It is commonly associated with hydatid cysts, trauma, or malignancy. Bilioptysis is typically caused by a bronchobiliary fistula (BBF), but can be secondary to any manipulation of the normal liver architecture adjacent to the diaphragm. We present a rare case of bilioptysis caused by a BBF secondary to transarterial radioembolization (TARE).
Case Description/Methods: A 73-year-old female with a history of metastatic cholangiocarcinoma presented to the ED with symptoms of progressive productive cough, with sputum described as thin, bright greenish-yellow, and right upper quadrant abdominal pain over a 5-week period. About 18 months prior to presentation, she underwent treatment of the cholangiocarcinoma with systemic chemotherapy and local TARE. Physical exam was notable for productive cough with rhonchi of the right lower lobe. Labs were notable for alkaline phosphatase of 138 IU/L (normal 25-125), and normal AST (13), ALT (10), total bilirubin (0.5). A chest x-ray found no acute abnormality, but a subsequent CT of the chest with contrast revealed hypodense areas around the previous TARE treatment zone. The patient had an endoscopic retrograde cholangiopancreatography performed with a double pigtail stent placement into biloma and BBF (Figure). Due to continued bilioptysis, interventional radiology performed a percutaneous transhepatic embolization of the bronchobiliary fistula with coils and an external drain. The patient required 2 additional embolizations before resolution of the BBF.
Discussion: A BBF is a rare, and potentially fatal finding. It is generally associated with liver abscesses, hydatid cysts, malignancies, or trauma. Our patient had development of a BBF approximately 18 months after a TARE procedure for treatment of her cholangiocarcinoma. BBF secondary to TACE has been previously described; however, to our knowledge, this is the first case of BBF secondary to TARE. Treatment options include biliary decompression, such as ERCP and biliary stenting, obliteration of the fistulous tract via embolization, or surgery. Our patient's treatment consisted of stenting via ERCP and percutaneous transhepatic embolization of biliary bronchial fistula on 3 separate occasions. After her first embolization, she achieved significant relief for about 3 weeks. Unfortunately, she developed recurrent bilioptysis and required repeat embolization.



[1677] **Figure 1.** ERCP with stent placement into biloma with bronchobiliary fistula.

S1678

Gastric Ectopic Pancreas With Pseudocyst Formation Causing Gastric Outlet Obstruction

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Introduction: Ectopic pancreas (EP) is a rare entity referring to the presence of pancreatic tissue at an anatomic location distinct from the pancreas which is commonly asymptomatic and found incidentally. EP can become symptomatic due to similar pathologic changes found in the anatomic pancreas including pancreatitis, bleeding, or pseudocyst formation. Gastric EP lesions present a diagnostic challenge as they lack distinguishing imaging and endoscopic features from other types of gastric submucosal tumors. This case report describes a rare case of EP presenting as a gastric antral mass with a unique combination of complications: chronic pancreatitis and pseudocyst formation causing gastric outlet obstruction.
Case Description/Methods: A 59-year-old man presented to the hospital with nausea, vomiting, weight loss, and epigastric pain for 5 days. Exam was notable for epigastric tenderness, and labs were unremarkable. Computed tomography (CT) and index upper endoscopy showed a 4.4 x 3.5 cm multiloculated cystic mass in the gastric antrum (Figure). Endoscopic ultrasound (EUS) showed that the lesion originated from the submucosa, and fine needle aspiration (FNA) demonstrated histiocytes and epithelial cells with a high amylase level. On repeat endoscopy, interval growth of the cystic gastric mass was observed, and repeat CT abdomen/pelvis again showed the mass now with multiple cystic collections causing mass effect on a distended stomach suggesting gastric outlet obstruction. The lesion was surgically resected and determined to be EP with histologic changes of chronic pancreatitis and multiple pseudocysts. Patient did well after surgical resection and had no recurrence on repeat upper endoscopy.
Discussion: EP can occur anywhere in the GI tract but most commonly are found in the duodenum or stomach. Symptoms are associated with the location, size, and pathology of the tissue. Lesions greater than 1.5 cm are more likely to be symptomatic with non-specific symptoms such as epigastric pain, abdominal fullness, nausea, diarrhea or melena. Diagnosis can be challenging, and in this case, multiple diagnostic modalities (imaging, endoscopy, EUS with FNA biopsy and surgical resection) were needed to reach a definitive diagnosis. EP is often found incidentally, and the combination of pseudocyst formation and gastric outlet obstruction in this case has been rarely reported in the literature. Prompt recognition of ectopic pancreas can facilitate the management of these complications.

[1678] **Figure 1.** A: Computed tomography of abdomen and pelvis from initial presentation, showing a multiloculated cystic mass (noted by *) and thickening of the gastric antral wall. B: Esophagogastroduodenoscopy (EGD) image showing a submucosal mass in the gastric antrum. C: Endoscopic ultrasound of gastric antrum with multicystic lesion appearing to originate from the submucosa.

S1679

A Case of Polyarthritis Panniculitis and Pancreatitis (PPP) due to Recurrent Alcoholic Pancreatitis

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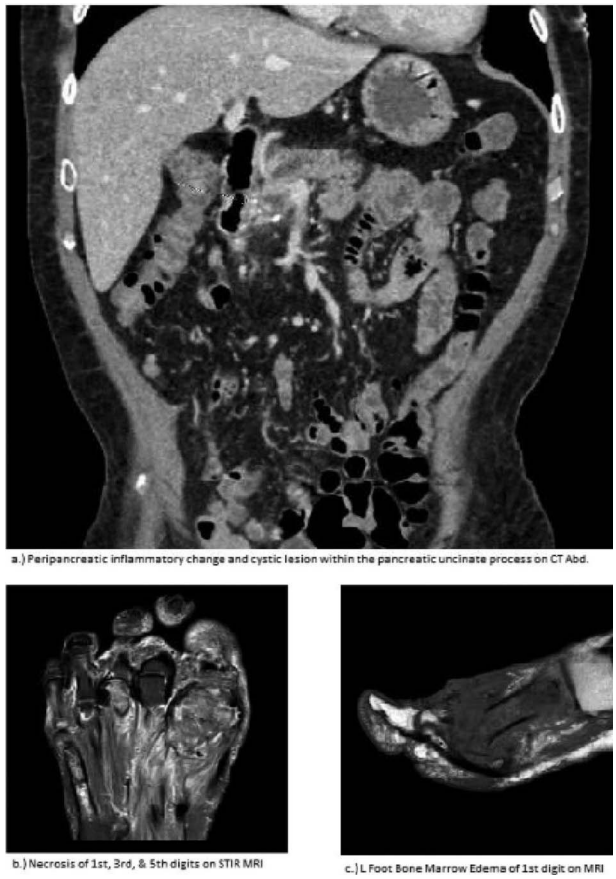
Introduction: Polyarthritis Panniculitis and Pancreatitis (PPP) is a rarely seen extra pancreatic morbidity hallmarked by the triad of joint pain (polyarthritis), tender skin lesions (panniculitis), and pancreatic inflammation (pancreatitis). The pathogenesis is mediated by pancreatic enzyme lipolysis of lipid rich skin and joint sites. Unfortunately, PPP is an elusive diagnosis given the minimal intrabdominal symptoms and a delayed diagnosis may worsen prognosis by as much as 24%. As such, we aim to present a case of this rare diagnosis to familiarize clinicians with the diagnosis of PPP.
Case Description/Methods: 67 M with prior alcohol use disorder, recurrent pancreatitis, and complex pancreatic cyst (2.1 x 1.4 cm) status-post fine needle aspiration presented with fever, malaise, diffuse joint pain, and rash. Exam was notable for diffuse visible synovitis, tender joints, and subcutaneous nodules. Patient denied abdominal pain and was hemodynamically stable. Laboratory findings are shown in the Table. Work-up including tickborne panel, Hepatitis B & C serology, Blood cultures, Urinalysis, Chlamydia, Gonorrhea, ASO, CCP, ACE, ANA, ANCA, IgG4, SSA, and SSB were all within normal limits. Right knee aspiration revealed straw colored fluid with 8552 WBC, 86 Neutrophils, no crystals, no growth on culture, and < 3000 RBCs. CT of the Abdomen & Pelvis revealed complex cystic lesion of the pancreatic head measuring 2.2 cm, punctate foci of calcification, no pancreatic ductal dilatation, distal common bile duct within normal limits (Figure A). MRI of the left foot revealed multifocal intramedullary osteonecrosis with bone marrow edema, multifocal synovitis, and prominent intermetatarsal bursitis (Figure B and C). Skin biopsy of the right thumb revealed lobar panniculitis with necrosis of adipocytes and residual "ghost cells". Patient was managed with IV solumedrol 80 mg, and prednisone taper. On follow-up one month later, patient's symptoms completely resolved. Follow-up MRI of the abdomen showed stable 2 cm pancreatic cyst which was not consistent with underlying malignancy.
Discussion: PPP is a potentially devastating pathology most commonly observed in males with prior alcohol use. The diagnosis often proves elusive given the lack of abdominal symptoms, but early intervention is crucial in reducing mortality and morbidities as seen in our case.



[1679] **Figure 1.** CT of abdomen (A) and MRI of left foot (B, C).

Table 1. Laboratory Findings

Hematology	Value
Hemoglobin	12.2 g/dL
Hematocrit	37 %
White Blood Cells	12.4 K/uL
Platelets	304 K/uL
Chemistry	**Value**
Sodium	139 mmol/L
Potassium	3.8 mmol/L
Chloride	104 mmol/L
Bicarbonate	26 mmol/L
Blood Urea Nitrogen	18 mg/dL
Creatinine	0.87 mg/dL
Glucose	94 mg/dL
Total Bilirubin	0.68 mg/dL
Alkaline Phosphatase	69 IU/L
AST	27 IU/L
ALT	34 IU/L
Lipase	20,521 IU/L
C-Reactive Protein	14 mg/L
Uric Acid	3.6 mg/dL

S1680

Successful Treatment of a Complex Bile Leak With Endoclips During Percutaneously Necrosectomy

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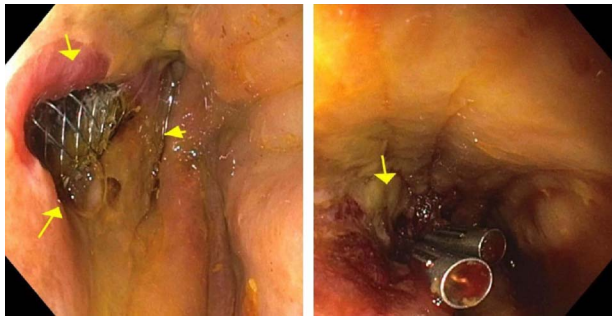
Introduction: Biliary injuries, such as bile leaks and fistulas, although rare, carry an increased risk of morbidity and mortality for patients. There are variable methods on how to proceed when one occurs, including stent placement and sphincterotomy via ERCP or percutaneous drainage. If these methods don't succeed due to complexity or other factors, unconventional methods may be employed.
Case Description/Methods: A 36-year-old male with a symptomatic cholelithiasis and choledocholithiasis underwent a laparoscopic cholecystectomy and ERCP with stone removal at an outside hospital. Patient continued to have smoldering abdominal pain after the procedure and EUS revealed a large peripancreatic necrotic collection with lipase >4000 and bilirubin of 14.5. ERCP showed a large caliber common hepatic duct leak and an 8mm x 8cm covered metal biliary stent was placed within the common bile duct, as well as an 18mm x 10cm covered esophageal stent was placed in the right lower quadrant, across the cystostomy, for percutaneous endoscopic necrosectomy. HIDA scan showed a persistent active bile leak near the location of the peripancreatic fluid collection. Necrosectomy was performed with successful clearance of the cavity. A concomitant 2cm bile duct wall defect was noted with exposure of the metal biliary stent (Figure). A dual approach with 2 endoclips were used to approximate the wall defect and a fully covered metal stent was left in the common bile duct to seal the defect from the inside and direct bile into the duodenum. The location of the defect on the common bile duct, not the cystic duct remnant, as well as the lack of visualization of leak on initial ERCP support that this was a fistula between the common bile duct and the peripancreatic fluid collection. Repeat ERCP at 3 and 5 months showed no further contrast extravasation and no bile duct stricture, and the metal stent was removed at 5 months.
Discussion: This case illustrates a novel approach that successfully treated a fistula between the bile duct and a pseudocyst with endoclips during a percutaneous necrosectomy. Although undocumented in the literature, complex, refractory biliary injuries require unconventional methods at times. This case proved successful in a young but very complicated patient and should be explored.



[1680] **Figure 1.** Endoscopic view of the 2 cm bile duct wall defect via a percutaneous approach (Left) vs the repaired defect with endoclips (Right).

S1681

Pancreatitis Secondary to Paxlovid-Induced Tacrolimus Toxicity

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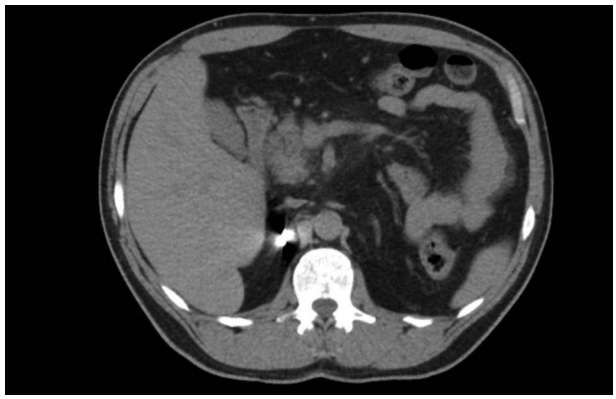
Introduction: Pancreatitis is a very common gastrointestinal disease that results in hospital admission. Early detection and treatment leads to better outcomes. This is the first reported case of pancreatitis secondary to elevated tacrolimus in a patient with prior renal transplantation after receiving Paxlovid for a COVID-19 infection.
Case Description/Methods: A 57-year-old male with past medical history of 4 renal transplants secondary to posterior urethral valves who presented to the emergency room with acute onset epigastric pain for 24 hours. He was on tacrolimus 5 mg every 48 hours monotherapy for his immunosuppression. 10 days prior to his presentation he had developed chills and anxiety. He tested positive for COVID-19 at that time on a home rapid test. His symptoms had not significantly improved and given his immunosuppressed state he was given Paxlovid (Nirmatrelvir/ritonavir). He took 2 days of Paxlovid, however after his

second day of treatment he developed severe epigastric pain requiring him to go to the emergency room. On admission his labs were notable for a lipase of 150 U/L (ULN 63 U/L). He underwent a CT scan was notable for an enlarged pancreatic head and neck with peripancreatic fat stranding (**Figure**). He also had a right upper quadrant ultrasound without any cholelithiasis and only trace sludge noted. His creatinine was noted to be 1.81 mg/dl which was above his baseline of 1.2 mg/dl. His tacrolimus trough level resulted at a level 45.6 ng/ml and later peaked at 82.2 ng/ml. His liver enzymes were normal. He was treated as acute pancreatitis with hydration and his tacrolimus was held with overall clinical improvement.

Discussion: Tacrolimus is one of the most common medications used in solid organ transplantation. It is a calcineurin inhibitor that inhibits both T-lymphocyte signal transduction and IL-2 transcription. It is metabolized by the protein CYP3A and levels are monitored closely. Paxlovid is currently prescribed as an antiviral therapy for COVID-19 infection. The ritonavir compound in Paxlovid is potent inhibitor of CYP3A. Currently the guidelines do not recommend Paxlvoid as a therapeutic in patients taking tacrolimus as there is concern about increased drug levels. There have been several case reports of pancreatitis in setting of tacrolimus. This case report helps to demonstrate the need for close monitoring of therapeutics levels, especially in medications with high risk of drug to drug interaction to help prevent serious side effects such as tacrolimus induced pancreatitis.



[1681] **Figure 1.** Axial image of acute pancreatitis. Noted enlarged pancreatic head and neck with peripancreatic fat stranding

S1682

Endoscopic Management of Migrated Pancreatic Stent into Hepaticojejunostomy Causing Acute Pancreatitis

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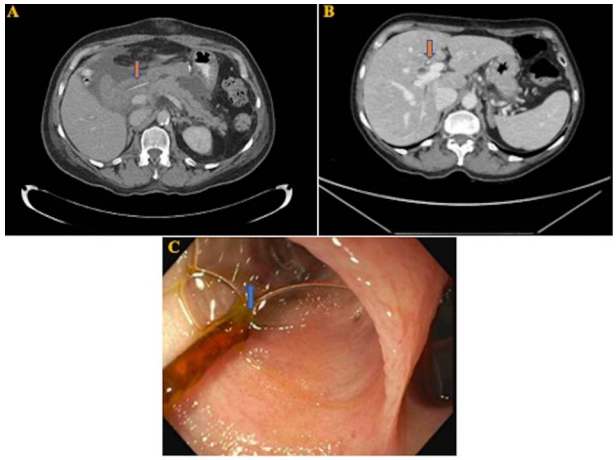
Introduction: Trans-anastomotic pancreatic stents after the pancreatoduodenectomy are placed to decrease the complications, maintain ductal patency and support healing of the anastomosis. However, complications of migration of trans-anastomotic stent from the pancreatojejunostomy (PJ) through the hepaticojejunostomy (HJ) is rare. Here, we present a case of acute pancreatitis in the setting of pancreatic duct (PD) stent migration into the biliary tree in a patient who had previously undergone pancreaticoduodenectomy.

Case Description/Methods: A 65-year-old female with a past medical history of duodenal adenocarcinoma status post pancreaticoduodenectomy with cholecystectomy and partial antrectomy with placement of a 5-French (Fr) PD stent across the PJ presented with acute abdominal pain, nausea, and vomiting. Significant labs showed lipase > 1200 and normal liver function tests. Computer tomography (CT) demonstrated a dilated PD up to 6 mm with no PD stent, and interval placement of a biliary stent terminating in the left intrahepatic duct (Figure A, B). The patient denied an interval endoscopic intervention since her pancreaticoduodenectomy. It was determined that the current biliary stent was likely the migrated PD stent. Endoscopic Retrograde Cholangiopancreatophraphy (ERCP) demonstrated one plastic stent into the biliary tree emerging from the widely patent HJ which was removed endoscopically (Figure C). The PJ was severely scarred with a pinpoint opening and moderate diffuse dilation of the PD in the body and tail of the pancreas. A 2 mm ventral pancreatic sphincterotomy and dilation of the PD orifice with a 4 mm balloon dilator was performed. A 7 Fr by 5 cm plastic PD stent with a full external pigtail and a single internal flap was placed 5 cm into the dorsal PD. The patient improved clinically with rapid resolution of pain on the first post-procedure day.

Discussion: PD stent migration into the biliary tree is a rare complication. However, it is postulated to be related to the proximity and angulation of the PJ and HJ. Complications of stent migration into the biliary tree include both pancreatic, as reported in this case and biliary complication.[3] Retrieval of migrated stent in biliary system is challenging due to altered anatomy after pancreaticoduodenectomy, yet is a standard procedure. The current case demonstrates the importance of consideration of PD stent migration as a cause of pancreatitis albeit uncommon.



[1682] **Figure 1.** A) Computed Tomography scan of the abdomen showing stent in the pancreatic duct, B) migrated stent in common bile duct with no stents in pancreatic duct after 4 months. C) Endoscopic image showing choledocojejunostomy anastomosis with plastic stent, Orange and blue arrows showing the stent.

S1683

A Rare Case of SARS-CoV-2-Induced Cholangiopathy

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Introduction: Severe acute respiratory syndrome coronavirus, a novel coronavirus that was declared a pandemic in 2019 is now known to affect multiple organ systems. While the primary organ affected has been the lungs, as time elapses, this virus finds victims in multiple organ systems. This case describes the clinical course and histopathological findings of a rare SARS-CoV-2 induced cholangiopathy.
Case Description/Methods: A 37-year-old male presented with 5 days of sharp, 10/10, periumbilical pain radiating to the RUQ with no exacerbating or alleviating factors. He had RUQ, and epigastric tenderness to palpation, jaundice and scleral icterus. Labs showed lipase >3000, total bilirubin 12.5, direct bilirubin 9.6, ALP 281, ALT 144, AST 70, and positive SARS-CoV- 2 PCR. Previous liver chemistries were normal. Autoimmune and viral hepatitis work-up was negative. US was concerning for acute cholecystitis with CBD 9mm. Antibiotics, IVF and morphine were initiated for presumed gallstone pancreatitis/ acute cholecystitis/choledocholithiasis. ERCP was pursued due to worsening biliary chemistry despite medical management. It revealed non-dilated CBD, but Mirizzi Syndrome was suspected due to a notably distended gallbladder compressing the common hepatic duct during cholangiogram. Sphincterotomy, sludge removal and biliary stent placement were done. Post-ERCP, ALT, AST, and ALP improved but total bilirubin remained elevated. MRCP failed to reveal intraductal filling defects or abnormalities to suggest sclerosing cholangitis. Cholecystectomy and liver biopsy were done which showed hepatocellular and canalicular cholestasis, lymphocytic inflammation, arterialization of central venules, hypocellular bridging fibrous septa connecting adjacent central areas. Patient was discharged with outpatient follow up after clinical improvement.
Discussion: Hepato-biliary complications from COVID-19 remain an area of research. Some of the complications reported include cholangiopathy, acalculous cholecystitis and secondary sclerosing cholangitis. Cases of cholangiopathy are described as a late complication of COVID-19 with diagnosis up to 118 days post infection. Though the time of COVID -19 infection is unclear, intrahepatic cholestasis due to pancreatitis/medication and COVID -19 are all contributing factors in our patients' disease progression. Making an accurate diagnosis of COVID-19 cholangiopathy is prudent as it may progress to cirrhosis, especially if a patient has underlying liver pathology.

S1684

Are They Related? A Case of Elevated IgG4 Autoimmune Pancreatitis and Pancreatic Cancer

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Introduction: IgG4 level has been recognized as a useful tool to differentiate between pancreatic cancer (PC) and autoimmune pancreatitis (AIP). However, there is lacking evidence regarding the risk of both of these diseases occurring concomitantly. We present a case of a patient with an elevated IgG4 level who was diagnosed with invasive pancreatic adenocarcinoma.
Case Description/Methods: A 65-year-old male with a past medical history significant for liver abscess, pulmonary embolism s/*P* IVC, hypertension, and GERD presents to the ED for 1 week of abdominal pain. On initial workup, findings were significant for elevated bilirubin (T bili: 5.3mg/dl; D: 3.0 mg/dl; I: 2.3mg/dl) and elevated liver enzymes (alk phos: 196; ALT: 412; AST: 237). CT abdomen showed a 2.9 cm ill-defined lesion of the pancreatic head. MRCP showed a dilated common bile duct (12.7mm) with a distal stricture and a short segment occlusion of the splenic and portal vein junction. Initial workup showed a IgG4 level of 287 mg/dl (normal low: 2-96mg/dl) and elevated Ca19-9 at 468. He was started on steroid therapy due to suspected AIP and instructed to follow up outpatient. A week later, was readmitted for jaundice where he underwent an EUS with pancreatic biopsy followed by ERCP and biliary stent placement. Pancreatic biopsies showed evidence of invasive adenocarcinoma with surrounding desmoplastic fibrotic response.
Discussion: AIP and PC may be difficult to differentiate from many mimicking entities which present with painless jaundice, new-onset diabetes mellitus, and elevation of tumor markers. IgG4 has been found to be elevated in PC where levels are usually less than a 2-fold increase from baseline. In small studies, a threshold of 280mg/dl has been used to favor a diagnosis of AIP over PC. Nonetheless, elevated IgG4 levels alone cannot be used to exclude a diagnosis of PC. Elevated tumor markers specifically CA19-9 level > 150 U/mL and imaging may also aid in the differentiation of AIP from PC. Biopsy remains the gold standard for definitive diagnosis. In cases where biopsies may be of high risk a short trial of glucocorticosteroids with clinically significant clinical and radiologic response favors a diagnosis of AIP. There are few cases reported of concomitant AIP and PC, such as in our patients, but there is a lack of clinically significant evidence to support this association. We look forward to further studies to assess the risk of PC in AIP and development of future PC screening guidelines in AIP.

S1685

COVID-19-Associated Sclerosing Cholangitis in Critically Ill Patients: A Case Report

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Introduction: COVID-19 was declared a world pandemic in March 2020. The etiology identified as SARS-CoV-2 is implicated in developing multisystemic manifestations, including liver injury. The liver damage in COVID-19 is represented as a hepatocellular and cholestatic pattern. Sclerosing cholangitis (SC) diagnosis in a critically ill patient (SC-CIP) with severe COVID-19 infection can mimic other biliary pathologies on imaging; the clinical history is decisive in guiding the diagnosis.
Case Description/Methods: A 52-year-old male with no prior history of liver disease is evaluated in a Hepatology clinic after presenting persistent liver enzyme elevation posterior to contracting COVID19. The patient was hospitalized for dyspnea and hypoxemia and intubated for 28 days. Since then, he has had itching, mainly at night, which interferes with his sleep. Laboratory data revealed a total bilirubin 3.2, direct bilirubin 2.4, ALP 4099, ALT 399, AST 135. Negative viral hepatitis panel and ANA. The rest of the workup was unremarkable. MRCP protocol revealed mild to moderate intrahepatic ductal dilation to the central ductal confluence and beading of the intrahepatic biliary ducts. ERCP demonstrated a normal-appearing common duct with evidence of stricturing areas within the intrahepatic biliary radicals, and a stent was placed in the right biliary system. The cytology was negative for malignancy, and colonoscopy showed no IBD. The study demonstrates SC after severe COVID-19 infection with negative smooth muscle and mitochondrial antibodies. He was treated with ursodiol, which improved symptoms and liver enzymes. A repeat MRI scan after treatment showed improvement in biliary ductal dilatation.
Discussion: COVID-19-associated SC-CIP occurs after long-term intensive care treatment. These patients with severe COVID-19 infection admitted to the ICU, have no evidence of prior hepatobiliary disease. The biliary epithelium is more susceptible to arterial blood flow disturbances than the hepatic parenchyma. Hence, prolonged hypotension, vasopressors, and mechanical ventilation are associated with ischemic injury of intrahepatic bile ducts. COVID-19-associated factors promote the development of SC-CIP, such as virus-associated vascular damage to the peribiliary plexus or direct damage to cholangiocytes through ACE-2 receptors. Therefore, diagnosis of COVID-19-associated SC-CIP is multidisciplinary and includes typical biliary imaging findings, critically ill patients with coexistence of COVID-19, and negative autoimmune serologies.

S1686

Primary Pancreatic Diffuse Large B Cell Lymphoma: A Rare and TreaTable Malignancy

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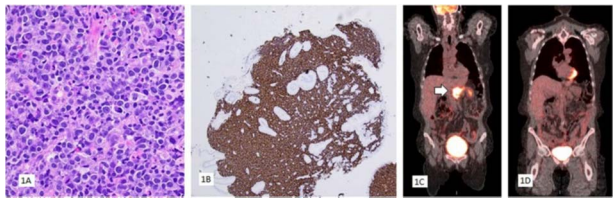
Introduction: Pancreatic lymphoma comprise less than 1 % of pancreatic malignancies. Most common location of diffuse large B cell lymphoma (DLBCL) are lymph nodes. Clinical symptoms and imaging studies have significant similarity resulting in delayed diagnosis or early initiation of comfort care measures. It is important to determine the histology of pancreatic tumor given the differences in management and prognosis. Here we report a rare case of primary DLBCL arising from pancreas.
Case Description/Methods: 62-year-old female with history of HTN and DM presented with 30 lbs unintentional weight loss epigastric abdominal pain and early satiety. CT abdomen with IV and oral contrast demonstrated pancreatic mass. Core biopsy of pancreatic head mass demonstrated infiltration by large atypical lymphoid cells with high N/C ratio, round nuclear contour, fine chromatin and variably prominent nucleoli (Figure A). Atypical mitosis and apoptosis seen in background. Lymphoma cells positive for CD 19, CD 20 (Figure B). Final diagnosis Large B cell lymphoma germinal center type. FISH for BCL 2, C MYC were normal ruling out possibility of double or triple hit lymphoma. Cancer antigen 19-9 at the time of diagnosis was within normal limits. FDG PET/CT prior to initiation of treatment showed main foci of increased tracer activity appears to be in the proximal body of the pancreas just medial to the stomach, measures greater than 4 cm in diameter, and has a maximum SUV of 11.86 (Figure C). Rituximab-Cyclophosphamide HydrOxyadriamycin Prednisone (R-CHOP) every 3 weeks initiated. After one cycle of R-CHOP reported significant improvement in epigastric pain and started gaining weight. She completed 6 cycles of R-CHOP. Post treatment FDG PET/CT complete resolution of upper abdominal lymph node mass (Figure D). She remains in remission for the past 1 year.

Discussion: Our case is an example of successful biopsy and treatment with aim to cure pancreatic malignancy. Despite radiological differences between pancreas adenocarcinoma and lymphoma, it is crucial to obtain adequate tissue sampling during from pancreas to establish histological diagnosis prior to discussion of prognosis and treatment plan. In general, DLBCL germinal center type has a 5-year survival of 70% with R-CHOP. Differential diagnosis of pancreatic mass that confer better prognosis than adenocarcinoma include neuroendocrine tumor, solid pseudopapillary tumor and lymphoma. Therefore, it is important to obtain biopsy and direct therapy based on histology.



[1686] **Figure 1. A**: Core biopsy of pancreatic head mass demonstrated infiltration by large atypical lymphoid cells with high N/C ratio, round nuclear contour, fine chromatin and variably prominent nucleoli. **B**: Lymphoma cells positive for CD 19, CD 20. **C**: FDG PET/CT prior to initiation of treatment showed main foci of increased tracer activity appears to be in the proximal body of the pancreas just medial to the stomach, measures greater than 4 cm in diameter, and has a maximum SUV of 11.86. **D**: Post treatment FDG PET/CT complete resolution of upper abdominal lymph node mass.

S1687

A Case of Primary Biliary Cirrhosis With New Onset Hyperthyroidism and Supratherapeutic INR

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Introduction: Hyperthyroidism is known to increase catabolism of vitamin-K-dependent clotting factors (II, VII, IX, X) and increase the response of vitamin K antagonists, usually warfarin. Primary biliary cirrhosis (PBC) has been associated with thyroid dysfunction (TD), especially with autoimmune thyroid disease. In the below case, a patient with known PBC on warfarin is found to have severely elevated INR related to new-onset hyperthyroidism with clinical consequences of hemorrhage including upper GI bleed.
Case Description/Methods: A 64-year-old female with PBC and antiphospholipid antibody syndrome on warfarin was admitted for hemorrhagic epiglottitis requiring emergency intubation and supra-therapeutic INR. Her PBC was diagnosed as stage II on biopsy 23 years ago and has remained clinically stable on ursodiol therapy. On presentation, the patient was tachycardic, tachypneic, and had O_2 saturations $<$ 90% on HFNC prior to intubation. Physical exam significant for larger goiter with diffuse upper airway swelling. She was admitted and found to have COVID-19 infection, INR $>$16.0 and PT $>$200.0 (limit of lab), WBC of 22.8, and lactate of 2.5. LFTs WNL aside from albumin of 2.0. TSH was $<$ 0.0017 (limit of lab) and free T4 of 3.4, free T3 of 5.3. TSH receptor antibody (TRAB) and thyroid stimulating immunoglobulin (TSI) levels were normal. Her last TSH was normal a year ago. CTA chest found a 5.7cm heterogeneous, partially calcified superior mediastinal mass consistent with multinodular thyroid goiter. Patient was initially given prothrombin complex concentrate and vitamin K with correction of INR over the following few days. She was extubated and started on methimazole. During the hospital course, she was found to have coffee ground emesis for which an EGD was done with findings of non-bleeding gastric ulcer (Forrest Class IIc) and LA Grade D esophagitis with adherent clot and bleeding for which hemostatic spray was applied. Patient was discharged a few days later following resumption of warfarin and on pantoprazole and methimazole.
Discussion: The above case demonstrates a rare case of PBC and new-onset hyperthyroidism due to multinodular thyroid goiter causing significantly elevated INR in the setting of warfarin use with hospital course complicated by GI bleed. PBC is associated with TD - hyperthyroidism, hypothyroidism, and thyroid cancer. Hyperthyroidism is less commonly associated with PBC compared to other TDs but should be considered especially with a finding of elevated INR.

S1688

A Rare Case of Bee Sting Associated With Pancreatic Necrosis

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Introduction: Hymenoptera insect sting bite was known to cause toxic reactions leading to hemolysis, serum sickness, rhabdomyolysis, vasculitis, renal failure, myocarditis, and neuritis. It is extremely rare to present with pancreatic involvement, as noticed in our case. Here we report an 82-year-old with severe necrotizing pancreatitis and multi-organ failure secondary to a bee sting.
Case Description/Methods: An 82-year-old male presented with complaints of nausea, non-bilious, non-bloody vomiting, and abdominal pain within an hour following a bee sting. He has a past medical history of colon cancer s/*P* cecal resection with side-to-side anastomosis, hypertension, and hyperlipidemia. His Initial blood workup was significant for elevated BUN/Creatinine- 60/3.1 mg/dl, elevated lipase –6800 u/l, elevated AST/ALT- 161/116 u/l, with normal ALP and T Bil-1.4 mg/dl. CT abdomen with IV contrast showed peripancreatic inflammatory changes with fluid extending into the perirenal and paracolic gutters, hypoenhancement of the pancreatic body and tail concerning pancreatic necrosis. He was started on intravenous fluids and a broad-spectrum antibiotic, Meropenem. During the hospital course, his clinical condition deteriorated with the development of sepsis, and he had an episode of PEA arrest, which he got through with resuscitation in 4 minutes. After 9 days in the hospital with continuous pressor requirements and worsening mental status, the family decided to make him comfortable care.
Discussion: Pancreatic necrosis is associated with substantial morbidity and mortality. Optimal management requires a multidisciplinary approach, IV antibiotics penetrating the pancreas, drainage, or debridement in persistent cases. Given the rarity of the bee sting-associated pancreatitis without a specific anti-toxin, it would be clinically challenging to manage. We noticed that this could also progress rapidly and require immediate transfer to a tertiary care center, which was unsuccessful in our case due to an unstable clinical condition.

S1689

Isolated Thrombosis Within a Superior Mesenteric Vein Aneurysm in the Setting of Acute Pancreatitis

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Introduction: Without involving the splenic or portal vein, isolated thrombosis of the superior mesenteric vein is very rare. However, untreated disease progression can lead to high morbidity or mortality from mesenteric ischemia or bowel infarction. We present a case of isolated thrombosis with a superior mesenteric vein aneurysm in the setting of acute alcoholic pancreatitis.
Case Description/Methods: A 55-year-old female with a medical history of pancreatitis and alcohol abuse presented to the emergency department for acute nausea with diffuse abdominal pain. Laboratory testing demonstrated lipase elevation of 7130 units/L. Initial computed tomography (CT) imaging demonstrated acute interstitial pancreatitis with peripancreatic inflammation and no evidence of necrosis. Patient was treated with conservative measures. However, after 5 days, patient developed new fevers and worsening abdominal pain prompting repeat imaging. CT abdomen demonstrated a new superior mesenteric venous aneurysm measuring 2.7 x 1.7 cm with a thrombosis within the aneurysm measuring 1.6 x 1.9 cm. Through a multi-disciplinary team approach, a decision was made to initiate anticoagulation with apixaban. With continued conservative management, patient continued to improve with no progression of mesenteric ischemia. Upon discharge, patient continued apixaban for 3 months. Subsequent imaging 6 months later demonstrating complete resolution of the venous thrombosis and without recurrence.
Discussion: Primary venous aneurysms (PVA) are significantly less common compared to arterial aneurysms with only about 200 total reported cases in the literature. PVAs have a low risk of complications with 88% of patients showing no clinical progression of aneurysm size, or subsequent complications including: thrombosis, biliary tract obstruction, or duodenal compression. Our patient developed an isolated thrombosis within her PVA. Superior mesenteric vein (SMV) thrombosis is uncommon accounting for 5% of all mesenteric vessel occlusive disease. In our case, the thrombosis likely occurred due to an imbalance between fibrinolysis and coagulation due to local inflammatory processes. Delayed diagnosis and treatment of SMV thrombosis can lead to fatal complications. Acute treatment is consistent of systemic anticoagulation and close clinical monitoring. Surgical exploration remains for patients developing progressive ischemia and bowel infarctions. Due to the lack of specificity of clinical symptoms, diagnostic accuracy of SMV thrombosis can be challenging.

S1690

A Slow Gut Can Hold up Recovery for Cholecystectomy-Induced Bile Leak

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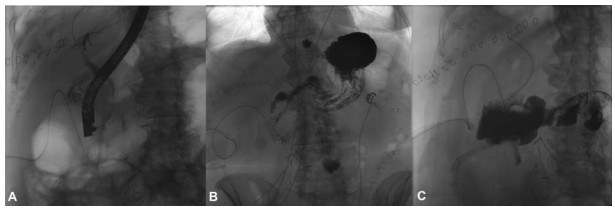
Introduction: Bile leak (BL) can occur in up to 0.5% of open cholecystectomies (CCY) and up to 2.7% of laparoscopic CCY. Strasberg types A and D post-CCY BL can be treated with endoscopic retrograde cholangiopancreatography (ERCP) with excellent success rates > 90%. ERCP interventions can decrease the pressure gradient between the bile duct and duodenum and facilitate transpapillary flow of bile.
Case Description/Methods: An 81-year-old woman with dementia, recurrent deep vein thrombosis and coronary artery disease presented to an outside hospital with severe abdominal pain and was diagnosed with acute cholecystitis. CCY was delayed by 6 days because she was on ticagrelor and rivaroxaban. Intraoperatively, a gangrenous perforated gallbladder was found, and a surgical drain (SD) was left in-situ. Post CCY she had persistent output from her SD and was transferred to our center for ERCP evaluation of suspected BL. On initial ERCP we found a high-grade Strasberg type A BL from the cystic duct (**Figure** A). Her major papilla was entirely located within a large duodenal diverticulum. Due to difficulty identifying safe cutting margins within the diverticulum, biliary sphincterotomy (Bsc) was not performed, and a 7 Fr plastic biliary stent was placed which resulted in good flow of bile into the duodenum. Her SD output decreased, and she was discharged the next day. Unfortunately, she was re-admitted to another hospital 3 days later with bile leakage around her SD, vomiting and constipation. An upper gastrointestinal (GI) series excluded a gastric outlet obstruction (**Figure** B). She was transferred back to our center for ERCP. The scout film showed that oral contrast that was ingested 3 days prior was still present within the proximal jejunum (**Figure** C). The biliary stent was patent and in good position, but a persistent high-grade BL was seen. Bsc was performed, and an 8mm by 6 cm fully covered self-expanding metal stent (FCSEMS) was placed. The patient was started on an aggressive bowel regimen with resolution of ileus. The SD output ceased, and the SD was removed 3 days later. Unfortunately, patient passed away in hospice before her follow-up ERCP from other causes.
Discussion: Despite successful diversion of bile to the duodenum via a biliary stent, small bowel ileus can increase the pressure gradient across the major papilla, resulting in a persistent BL. More aggressive ERCP interventions using FCSEMS, along with aggressive medical treatment of ileus, allowed the BL to heal expeditiously.



[1690] **Figure 1.** A: High-grade cystic duct bile leak on initial ERCP evaluation. B: Contrast flowing from stomach to duodenum in upper gastrointestinal series. The plastic biliary stent is in-situ. C: the oral contrast ingested approximately 3 days prior is still visible within the proximal jejunum on ERCP scout film.

S1691

No Way to Rest Easy With Acute Pancreatitis of a Pancreas Rest

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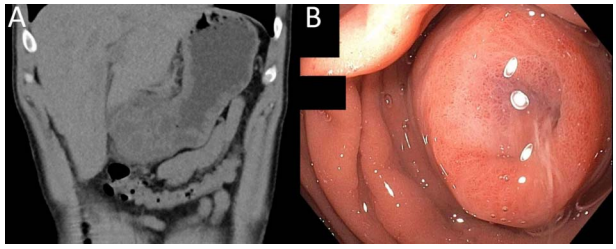
Warren Alpert Medical School of Brown University, Providence, RI.

Introduction: Ectopic pancreas tissue (also known as pancreas rest or heterotopic pancreas) is congenital pancreas tissue occurring outside of the orthotopic pancreas, most commonly in the stomach, duodenum, or jejunum. Ectopic pancreas tissue is generally asymptomatic, and usually discovered incidentally. Acute ectopic pancreatitis occurs uncommonly. Acute ectopic pancreatitis without lipase elevation is an even rarer phenomenon. We report a case of acute pancreatitis of a gastric pancreas rest with a normal lipase requiring esophagogastroduodenoscopy (EGD) for diagnosis.
Case Description/Methods: A 42-year-old man with a history of alcohol use disorder presented to the emergency department with a 3-day history of severe, sharp, nonradiating epigastric abdominal pain, nausea, and vomiting. His last alcoholic beverage was one week prior to arrival. The patient was mildly tachycardic. Vitals were otherwise stable and he was afebrile. Physical exam was notable for epigastric tenderness. His labs including lipase were unremarkable. A CT scan of the abdomen and pelvis revealed marked gastric antral thickening and mild associated inflammatory stranding (**Figure A**). The appearance was concerning for gastritis or peptic ulcer disease; however, an underlying mass could not be excluded. The pancreas was normal in appearance. The patient then underwent an EGD which demonstrated a single swollen/edematous appearing 25 mm submucosal papule (nodule) with central umbilication in the gastric antrum endoscopically consistent with a pancreas rest (Figure B). The patient was diagnosed with acute pancreatitis of a gastric pancreas rest and managed with aggressive intravenous hydration and analgesia. By the third day of hospitalization, his diet was advanced to solid foods and his pain had improved. His labs remained unremarkable, and he was discharged home.
Discussion: Acute pancreatitis of a pancreas rest without an elevation in serum lipase level is an exceedingly rare phenomenon. Acute gastric ectopic pancreatitis must be considered in the differential for antral thickening and can be mistaken on CT imaging for gastritis, peptic ulcer disease, or a mass. This case highlights the classic endoscopic appearance of an inflamed gastric pancreas rest. Early EGD is important in the diagnosis of acute ectopic pancreatitis, especially when encountering a patient with antral thickening and associated fat stranding of unclear etiology on CT.



[1691] **Figure 1.** A: CT scan of the abdomen without intravenous contrast demonstrating gastric antral thickening and mild associated inflammatory stranding. B: EGD demonstrating a single swollen/edematous appearing 25 mm submucosal papule (nodule) with central umbilication in the gastric antrum endoscopically consistent with a pancreas rest.

S1692

Bilothorax: Uncommon Accumulation in the Pleural Space

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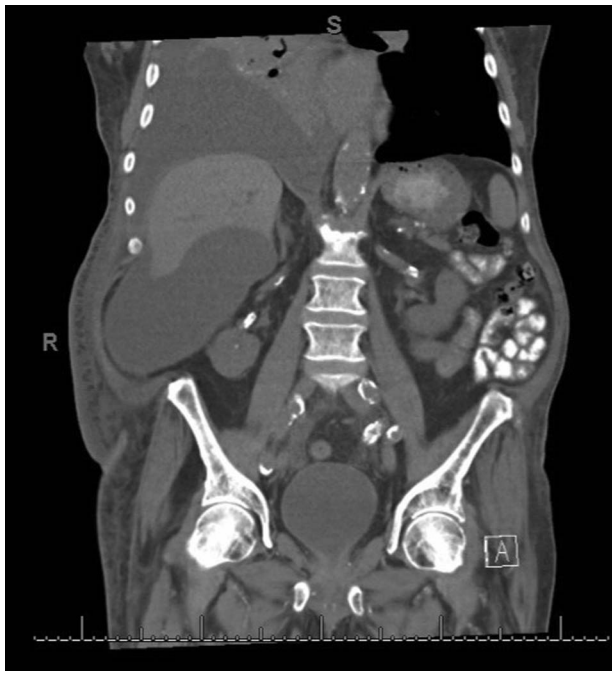
Introduction: In rare circumstances, pleural fluid can accumulate from outside of the blood vessels. This disease state has been termed pleural effusion of extra-vascular origin (PEEVO) and can include urine, CSF, bile, dialysate or chyle. We present a case of spontaneous translocation of bile into the thorax in a patient with a hepatic duct stricture.
Case Description/Methods: An 88-year-old male with history of cholecystitis status post cholecystectomy complicated by common hepatic duct stricture with indwelling T-tube presented with 2 days of fatigue, abdominal pain and shortness of breath. Prior to presentation, the T-tube was functional for several months and then was removed inadvertently while at home. CT of the abdomen showed intracapsular fluid collection along the posteroinferior liver and right sided pleural effusion (**Figure**). MRCP showed CBD stricture with intrahepatic ductal dilatation. ERCP was performed and the proximal biliary tree was unable to be opacified with contrast. A percutaneous trans-biliary drain was placed with fluid samples positive for staph simulans. Repeat ERCP was performed, and 2 stents were placed in the common bile duct. A

repeat CT scan showed an interval increase in both the size of the hepatic subcapsular fluid collection and the pleural effusion. A chest tube was placed and 1.5L of green fluid was removed. Fluid studies showed WBC 925, glucose 54, LDH 1831k, protein 2.9, Bilirubin 7.5 (serum bilirubin 0.8), and pH 7.21. The diagnosis of bilothorax was made. Repeat imaging demonstrated improvement in fluid collection and the chest tube was removed after 5 days.

Discussion: PEEVO in which bile is the accumulated fluid is termed as "bilothorax" and is often the result of injury to the biliary tree leading to a fistula between the tree and pleural space. In this case, there was spontaneous translocation of bile which is quite uncommon. Imaging will demonstrate isolated right-sided pleural effusion. Diagnosis is confirmed with fluid studies, the effusion is often exudative, and the distinguishing feature is the presence of bilirubin in the fluid. If the bilirubin concentration in the pleural fluid is greater than that of the serum bilirubin, the diagnosis of bilothorax is made. Coordination between pulmonology and gastroenterology specialists is essential for prompt drainage of the accumulation and decompression of the biliary system. The presence of a fistula would additionally warrant surgical correction.



[1692] **Figure 1.** CT Abdomen demonstrating sub-capsular fluid collection with expansion into right pleural space

S1693

Incidental Pancreatic Pseudoaneurysm Following ERCP

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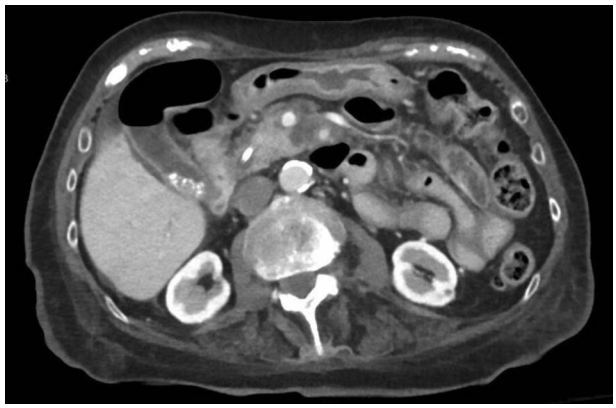
Introduction: ERCP is a commonly performed procedure, with up to 500,000 ERCPs performed annually. Commonly known complications include infection, pancreatitis, hemorrhage and perforation. Here we describe a case of pancreatic pseudoaneurysm (PDA) which resulted from Post-ERCP pancreatitis.

Case Description/Methods: An 84-year-old female presented to hospital with vomiting, epigastric pain and increased weakness for one week after undergoing ERCP with stone removal and stent placement. On presentation, she was hemodynamically stable. Hepatic and pancreatic enzymes were within range, hemoglobin and hematocrit were stable, 12.6g/dL and 37.1%, respectively, and near patient's baseline (13g/dL). CT Abdomen with IV contrast showed a 9mm hyperattenuating PDA in/adjacent to pancreatic head (which was new from recent MRCP). For better characterization, a CT angiogram with pancreatic protocol was performed, which redemonstrated stable 9mm PDA (**Figure**). Given no evidence of bleeding and improved clinical condition of the patient, no intervention was indicated, and the patient was discharged home.

Discussion: Post-ERCP pancreatitis has an annual incidence of around 14%. Post-procedure pancreatic PDA is rarer still, seen in around 10% of the patients with post-ERCP pancreatitis. Irritation of arterial and ductal walls by indwelling catheter can lead to the development of both pancreatitis and PDA. Most of the previously reported cases of post-ERCP PDA were seen in sickle cell disease patients [1, 2]. Our patient is unique as she did not have sickle cell disease and did not have pancreatitis on arrival as per revised Atlanta criteria. We suspect patient developed pancreatitis prior to arrival to ED as she was symptomatic for one week after ERCP. A bleeding PDA is an emergency with high morbidity and mortality, so prompt diagnosis and treatment is imperative to prevent further complications.



[1693] **Figure 1.** Small 9 mm focus of enhancement at the pancreatic head concerning for pseudoaneurysm

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S1694

Atypical Presentation of Pancreatic Serous Cystadenoma

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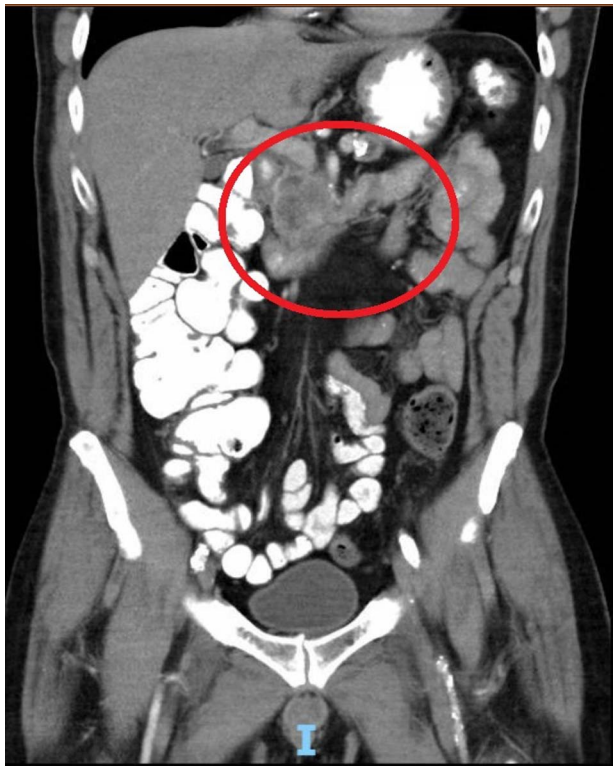
Introduction: Pancreatic serous cystadenomas (PSCA) which represent one-third of pancreatic cystic neoplasms are typically benign asymptomatic lesions usually found incidentally on imaging studies at the body or tail of the pancreas. When symptomatic, presents typically with abdominal pain and a palpable mass. We report a case with PSCA presented as chronic diarrhea due to pancreatic duct (PD) obstruction with further enlargement over the years leading to biliary and partial duodenal obstruction.
Case Description/Methods: A 65-year-old man with chronic diarrhea and a remote history of Hodgkin's Lymphoma treated with Mantle and pelvic radiation. Initially diagnosed as radiation enteritis, presented to our clinic with worsening diarrhea, flatulence, bloating, and weight loss. Endoscopic evaluation was negative for celiac disease or microscopic colitis. Diarrhea responded to empiric trial of pancreatic enzymes and he regained weight. CT scan of abdomen done to determine the etiology of exocrine pancreatic insufficiency (EPI) revealed a 3.7 cm complex cystic mass in the in the head of the pancreas with PD dilation and atrophy of the body and tail (**Figure**). Endoscopic ultrasound showed pancreatic mass with involvement of SMA and portal vein. Fine needle aspiration was not diagnostic. CT-guided biopsy was consistent with PSCA. 4 years later he complained of pale stools and dark urine; labs revealed ALT 495, alkaline phosphatase 463, bilirubin 4.5. MRI showed cyst enlargement to 5.7 cm with biliary dilation. Surgical consultation deemed him not to be a surgical candidate because of radiation induced vascular disease. ERCP with fully covered stent placement led to resolution of symptoms and normalization of liver tests. A year later ERCP with stent exchange showed compression of the duodenum distal to the stent leading to partial obstruction.
Discussion: PSCA is usually an incidental finding on imaging studies. EPI is most commonly a result of chronic pancreatitis. Our patient presented with chronic diarrhea due to EPI as a result of PD obstruction by a PSCA. This case underscores the importance of imaging the pancreas in all patients with EPI to evaluate for potential underlying neoplasm causing obstruction of the PD. Our case also highlights that PSCA although benign if in the head of the pancreas can grow in size leading to gastric outlet obstruction and obstructive jaundice which can be associated with significant morbidity if the patient is not a surgical candidate.



[1694] **Figure 1.** There is an approximately 4.0 cm x 3.7 cm ill-defined complex mass in the head of the pancreas.

S1695

Application of Endoscopic Powered Resection (EPR) in Pancreatic Necrosectomy Post Cystogastrostomy

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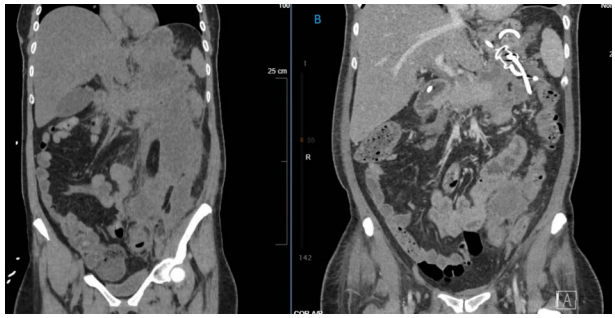
Introduction: Patients with severe pancreatitis routinely develop walled off necrotic collections that require a procedure known as a Cystogastrostomy. This procedure creates an opening between the necrotic collection and the stomach to help facilitate removal of necrotic tissue. Serial endoscopic necrosectomies are often required to remove all the necrotic debris. Endoscopic powered resection (EPR) is a procedure used to perform mechanical mucosectomies of polyps within the gastrointestinal tract. This method of mechanical resection has recently been applied to endoscopic pancreatic necrosectomy and debridement. The following case highlights a novel application of EPR in performing pancreatic necrosectomies following cystogastrostomy to help decrease the number of repeat procedures.
Case Description/Methods: A 47-year-old patient with past medical history of asthma, hyperlipidemia, breast cancer, recent hypertriglyceridemia-induced acute pancreatitis initially presented with fevers, chills and abdominal pain. Initial labs remarkable for leukocytosis WBC 19.7 K/UL. CT scan showed concern for new infected peripancreatic necrotic fluid collection measuring 9.5 x 6.0 cm extending inferiorly along the left paracolic gutter (**Figure**). An EUS with cystogastrostomy and endoluminal stent placement was performed to facilitate drainage of the necrotic collection. Repeat EGD with both EPR and snare necrosectomy was performed the following week. CT scan completed 2 days later showed marked interval decrease in the multiloculated peripherally enhancing peripancreatic air and fluid collection now measuring 7.3 x 4.0 cm. The patient underwent second necrosectomy one week later with EPR and snare mechanical debridement which revealed an 18 cm cyst cavity. Significant amount of necrotic tissue removed at that time and patient scheduled for repeat EGD in 3 weeks with endoluminal stent retreatment.

Discussion: Endoscopic pancreatic necrosectomy is often performed 4 weeks after the initial episode of pancreatitis to allow formation of a true, walled off necrotic collection. When performing endoscopic necrosectomy of large collections, at least 5 procedures are often required to completely removal all necrotic tissue. As shown in this case, implementation of EPR with traditional endoscopic snare necrosectomy can facilitate efficient removal of debris and help decrease the number of repeat necrosectomies. This could ultimately improve resource utilization and have major implications on overall patient morbidity and mortality.



[1695] **Figure 1.** A. Large (9.5 x 6.0 cm) peripancreatic necrotic fluid collection with inferior extension into left paracolic gutter. B. Marked interval decrease in multiloculated necrotic collection following second EPR necrosectomy with endoluminal stent visualized.

S1696

Biliary Sump Syndrome Causing Cholangitis From Displaced EUS-choledochoduodenostomy Stent Migration, Salvaged With Cholangioscopy and Endoscopic Therapy

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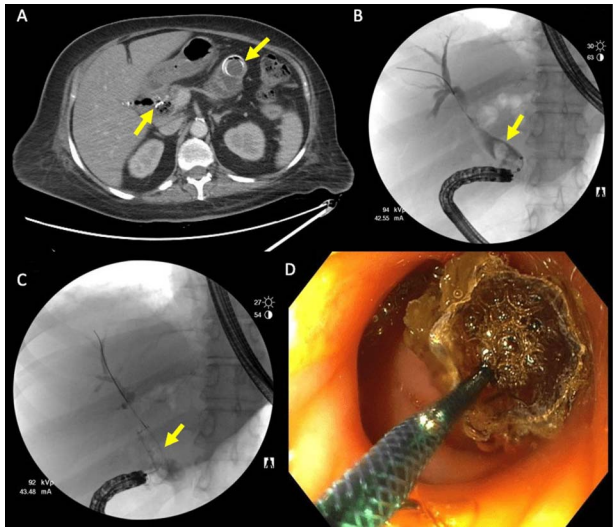
Introduction: EUS-guided choledochoduodenostomy (EUS-CDD) is a great salvage procedure for relieving biliary obstruction from failed transpapillary biliary access, however, we describe a patient with EUS-CDD who developed a rare complication of choledochoduodenostomy stent migration causing recurrent biliary obstruction and cholangitis, salvaged with endoscopic intervention with repeat stenting.
Case Description/Methods: A 58-year-old woman with pancreas head adenocarcinoma presented with gastric outlet and biliary obstruction. Both these conditions were successfully treated with same session EUS-CDD with a fully covered metal biliary stent, along with EUS-guided gastroenterostomy (EUS-GE) with 20 mm Axios stent. She now presented 5 months later with signs of recurrent biliary obstruction and cholangitis. Labs revealed elevated alkaline phosphatase 1986 U/L (normal 35-130), and total bilirubin 3.6 mg/dL (normal ≤ 1.2). Repeat CT scan showed a patent EUS-GE Axios stent, but outward migration of the EUS-CDD stent (Figure A), causing recurrent biliary obstruction from food debris impaction in the dependent bile duct reservoir causing Sump Syndrome seen on repeat endoscopy and ERCP (Figure B). We were able to salvage the EUS-CDD site with cholangioscopy and food disimpaction along with repeat Viabil fully covered metal biliary stent with anti-migration flaps placement via the existing EUS-CDD site (Figure C and D).
Discussion: Biliary sump syndrome is a rare long-term complication previously seen in surgical CDD patients due to food impaction in the distal CBD from the new anastomosis to act as a "sump," a poorly drained reservoir making this prone to accumulation of debris, increasing chances of complications, such as biliary obstruction, abscess, and cholangitis. Our case highlights the importance of this rare phenomenon even in the new era of therapeutic EUS procedures, but with the caveat that it can be successfully treated with repeat endoscopic therapy, and thereby avoiding the need for surgery or percutaneous intervention.



[1696] **Figure 1.** A. CT abdomen with left arrow showing displaced CDD stent and right showing patent Axios EUS-GE stent. B. ERCP showing debris obstructing distal CBD causing reservoir "Sump Syndrome" C. ERCP with placement of new Viabil stent with antimigration flaps for salvage of EUS-CDD. D. Endoscopic image of new Viabil stent with antimigration flaps.

S1697

Biliary Schwannoma in a Post-Liver Transplant Patient Presenting as Biliary Obstruction

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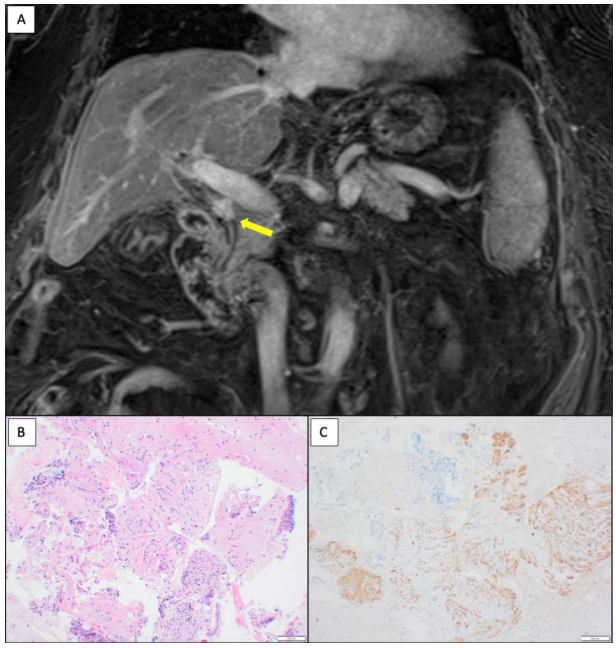
Introduction: Schwannomas are benign, mesenchymal-derived neoplasms which present as solid, slow growing tumors. They can rarely be observed in the gastrointestinal (GI) tract, with very limited reports of them occurring in the biliary system. In this case, we describe a bile duct schwannoma in a post-liver transplant patient.
Case Description/Methods: A 77-year-old man with a history of alcohol-related cirrhosis with hepatocellular carcinoma treated with orthotopic liver transplantation 16 years prior, and end stage renal disease on hemodialysis presented with jaundice. Labs demonstrated new elevations in his liver enzymes: AST 181 U/L, ALT 189 U/L, alkaline phosphatase 625 U/L, total bilirubin (TB) 7.9 mg/dL, and direct bilirubin

5.0 mg/dL. A magnetic resonance cholangiopancreatography was performed and demonstrated a 16 x 15 x 11 mm enhancing mass causing abrupt narrowing of the mid common bile duct (CBD) with upstream duct dilation to 10 mm (Figure A) without other discrete mass identified. A follow-up endoscopic ultrasound confirmed a hypoechoic mass in the mid CBD and fine needle biopsy was obtained. An endoscopic retrograde cholangiopancreatography was subsequently performed, which demonstrated a severe biliary stricture at the level of the prior biliary anastomosis which was treated with balloon dilation and placement of a covered metal stent. The procedure was complicated by post-sphincterotomy bleed and mild post-ERCP pancreatitis. His liver enzymes failed to improve, and his TB progressively worsened to 23.3 mg/dL. Pathology returned showing a low-grade spindle cell tumor (**Figure** B), with staining positive for S100 (**Figure** C) and negative for CD117 and desmin, consistent with a diagnosis of schwannoma. Surgical intervention was not pursued, in line with his goals of care, and he was ultimately transitioned to comfort-oriented care and passed 5 days after admission.

Discussion: Schwannomas are rare neoplasms which can arise from the nonepithelial wall of the GI tract containing nerve fibers. They are composed of highly cellular spindle cells which stain positive for vimentin (marker of mesenchymal origin) and S100 protein (marker of nerve sheath origin), and negative for CD117 (marker of GI stromal tumors) and desmin (muscle cell marker). Neoplasms can often be suspected in late presentations of post-transplant biliary obstruction. To our knowledge, this is the first case report of a biliary schwannoma occurring at the site of a biliary anastomosis in a liver transplant patient.



[1697] **Figure 1.** A) MRCP T1-weighted coronal image demonstrating a 16 x 15 x 11 mm enhancing mass with diffusion restriction (yellow arrow) at the mid common bile duct causing an abrupt biliary stricture with upstream dilation, B) 10x magnification H&E showing spindled, wavy nuclei in a fibroblastic stroma consistent with a spindle cell neoplasm in a background of unremarkable columnar biliary epithelium, C) 10x magnification S100 immunohistochemical stain showing diffuse nuclear and granular cytoplasmic positivity consistent with nerve sheath origin.

S1698

Beware of the Hemosuccus Pancreaticus

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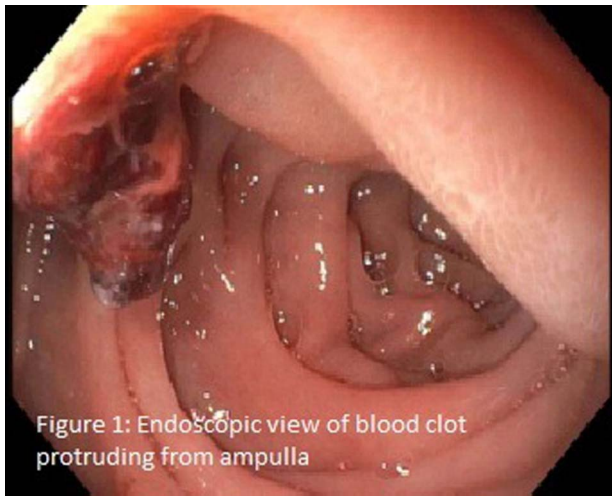
Introduction: While most cases of acute pancreatitis resolve without issue, roughly 20% are considered moderately severe and associated with local complications (e.g. pseudocysts, necrotic collections), organ failure, or systemic complications such as exacerbation of comorbidities. We present an interesting case of acute pancreatitis complicated by necrosis, portal vein thrombosis, and hemosuccus pancreaticus.
Case Description/Methods: A 55-year-old man with chronic alcohol use presented to the emergency room with epigastric pain radiating to the back, vomiting, and decreased colostomy output for one day. CT abdomen revealed pancreatic fat stranding, peripancreatic head fluid collection, and a portal vein thrombus. Heparin drip was initiated. On hospital day 4, while awaiting MRI abdomen to further evaluate the fluid collection, the patient developed maroon stools and frank clots in the colostomy bag, eventually requiring transfusion of 2 units of RBCs. The MRI clarified the fluid collection as a 3.1 cm necrotic fluid collection filled with debris, fluid, and hemorrhage. An emergent EGD revealed a blood clot protruding from the ampulla (Figure). Imaging and endoscopic findings supported the diagnosis of hemosuccus pancreaticus. On visceral angiography, interventional radiology reported an eroded gastroduodenal artery which was embolized with coils. The patient required no further intervention to address the GI bleeding.
Discussion: Hemosuccus pancreaticus is the least common cause of upper GI bleed, estimated at 1/1500 cases, which translates to difficult or delayed diagnosis, and high mortality rates (overall estimated at 9.6%; 90% if untreated), owing to its risk of exsanguination. It is defined as arterial bleeding into the pancreatic duct. Diagnostic work-up with imaging such as CT angiography or MRCP should be pursued in patients with pancreatitis, pancreatic pseudocysts or tumors, who present with gastrointestinal bleeding. Endoscopy may reveal bleeding or clots at the ampulla, and should be done to rule out other causes. Treatment often involves visceral angiography, though may require pancreaticoduodenectomy in severe cases.



[1698] **Figure 1.** Endoscopic view of blood clot protruding from ampulla

S1699

Biliary Fascioliasis Presented With Recurrent Pruritus

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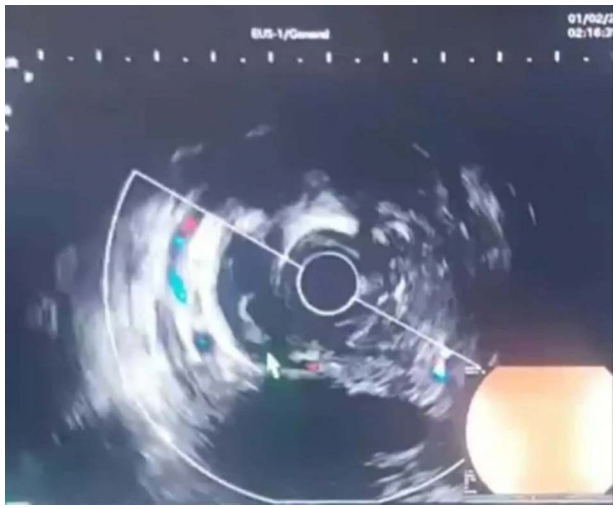
Introduction: Fascioliasis is a waterborne disease caused by *Fasciola Hepatica* (FH), mainly involves the hepatobiliary and has 2 phases of manifestation. The hepatic phase typically occurs 6-12 weeks after ingestion, characterized by right upper quadrant abdominal pain, elevated liver enzyme, and eosinophilia. Adult flukes migrate to biliary tracts during the biliary phase and can be asymptomatic or present with biliary obstruction, cholangitis, and pancreatitis. In the endemic areas, FH should be considered for idiopathic biliary obstruction with atypical symptoms. Here, we report a 36 y/o male from Iron who presented with recurrent pruritus and was found to have FH which was removed by ERCP.
Case Description/Methods: A 36-year-old man from Iran was initially admitted due to 2 years history of occasional vague abdominal pain. The patient also had recurrent non-rash pruritus not responding to medication. Past medical history was unremarkable. The laboratory test were all normal except for elevated CRP and eosinophil percentage (3%). Upper and lower endoscopy were all negative. Abdominal US showed dilated CBD without any significant findings. Follow-up EUS showed dilated CBD of 16mm with some filling defects and 2 live parasites (Figure). EUS-guided ERCP was done with CBD cannulation and biliary stent placement. 10mg/kg of triclabendazole was administered as a single dose 4 days after the procedure. Another ERCP was performed later for biliary stent removal and live trematode extraction without any complication. During one month follow up, the patient was free of symptoms with back to normal CRP and eosinophil percentage. A 1-year EUS follow-up showed normal CBD without any stone, sludge, or lesion.
Discussion: Humans can be accidental hosts for FH with variable symptoms, such as urticaria, cough, dyspnea, cholangitis, biliary obstruction, and cholecystitis. Stool microscopy in addition to imaging studies is always used for diagnosing. The first-line treatment is 10mg/kg triclabendazole every 12 hours for 2 doses. However, due to the high risk of biliary obstruction and related complications, endoscopic clearance is mandatory in cases like this. Biliary sphincterotomy and extraction of flukes by balloon extraction or basket are very effective and safe interventions. In a patient suffering from recurrent pruritus not responding to medications and eosinophilia, abdominal US should be considered for FH, especially in an endemic region of the world.



[1699] **Figure 1.** EUS Showing the Parasite with the arrow pointing to the location.

S1700

Barking up the Wrong Hepatobiliary Tree: A Case of Elevated CA19-9

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Introduction: Incidental elevations in Carbohydrate Antigen 19-9 (CA19-9) can trigger extensive medical evaluations for malignancy. Though classically associated with pancreatic cancer, CA19-9 is a non-specific manifestation of multiple benign and malignant disease processes.
Case Description/Methods: An asymptomatic, healthy 50-year-old female presented to primary care for an elevated CA19-9 level obtained for pancreatic cancer screening in Asia in 2019. Her evaluation in 2019 included abdominopelvic CT and magnetic retrograde cholangiopancreatography, which were normal. She was offered endoscopic ultrasonography to further evaluate pancreaticobiliary etiologies but was lost to follow-up amid the COVID-19 pandemic. She returned to the US in 2021, and basic laboratory testing and routine cervical cancer screening were performed. She was referred to Gastroenterology (GI) for further evaluation. Cervical cytology revealed atypical endometrial cells, and endometrial biopsy by gynecology was concerning for gastric-type endocervical adenocarcinoma. Transvaginal ultrasound revealed a thickened endometrial stripe, and pan CT revealed duodenal thickening, for which GI performed bidirectional endoscopy without significant abnormalities and no pancreatic or metastatic disease. Repeat CA19-9 increased. She was referred to gynecologic oncology, where cervical biopsy and pelvic MRI confirmed an endocervical mass. She was diagnosed with Stage IIB gastric-type endocervical adenocarcinoma and underwent hysterectomy and left salpingectomy with adjuvant chemoradiation.
Discussion: CA19-9 is synthesized in multiple organ systems. Elevations in asymptomatic patients are rarely predictive of pancreatic cancer but may expose patients to unnecessary testing and inadvertent harms without identifying malignancy. Thus, CA19-9 is not recommended for pancreatic cancer screening. Incidental elevations do warrant repeat testing. Benign processes will yield stable or decreasing levels, while rising levels suggest progressive or malignant processes. If concern for pancreatic malignancy is low, a reasonable investigation includes chest X-ray or CT, metabolic studies, hemoglobin A1c, liver and thyroid function panels, abdominopelvic CT or gynecologic cancer evaluation, and any other age-indicated cancer screening. In this case, prior imaging studies suggested low concern for pancreatic cancer. Her subsequent evaluation aligned with this suggested work-up and revealed gynecologic cancer as the ultimate etiology for her elevated CA19-9.

S1701

Bile Leak: A Rare Complication of Percutaneous Liver Biopsy

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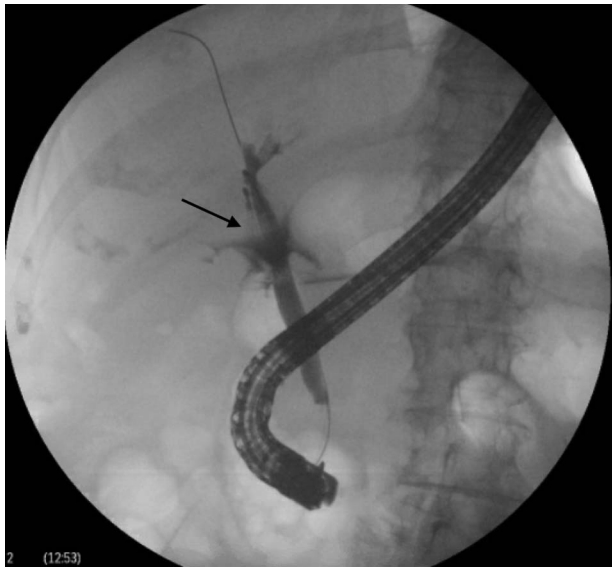
Introduction: Percutaneous liver biopsy is a cornerstone in diagnosing various hepatobiliary disorders. It is generally a safe procedure. Bile leak is a very rare complication, with rates less than 0.001%.
Case Description/Methods: A 79-year-old woman presented to the emergency department with severe right upper quadrant abdominal pain along with nausea and vomiting after undergoing an ultrasound-guided percutaneous liver biopsy for elevated liver enzymes. On examination, right upper quadrant tenderness was appreciated. Computed tomography (CT) and Magnetic resonance imaging (MRI) of the abdomen were suggestive of blood products within the gallbladder. She underwent Endoscopic retrograde cholangiopancreatography (ERCP) for increased bilirubin (Table). On Fluoroscopy, she was found to have a bile leak at the level of the common hepatic duct (Figure). A temporary plastic biliary stent was placed into the common bile duct (CBD).
Discussion: There are guidelines by the American Association for the Study of Liver and the Society of Interventional Radiology to prevent complications after percutaneous needle biopsies and liver biopsies. Utilizing ultrasound can help avoid injury to the gall bladder and large bile ducts. A biopsy of at least 2-3 cm length and 16-gauge caliber is considered sufficient. Patients should be closely observed for at least 2-4 hours after the procedure. Keeping a low threshold for prompt evaluation with appropriate imaging modalities and timely intervention can help overcome this challenge and reduce morbidity and mortality.



[1701] **Figure 1.** Fluoroscopy demonstrates extravasation of contrast at the level of the common hepatic duct, suggestive of bile leak

Table 1. Trend of total bilirubin through the hospital course. Notice the 50% decrease after biliary stent placement

Total Bilirubin (mg/dL)	0.8	3.6	6.2	6.7	7.4	3.1	2.7	2.2	0.9

S1702

Coil-Angitis: A Rare Case of Coil Migration into the Biliary Tree

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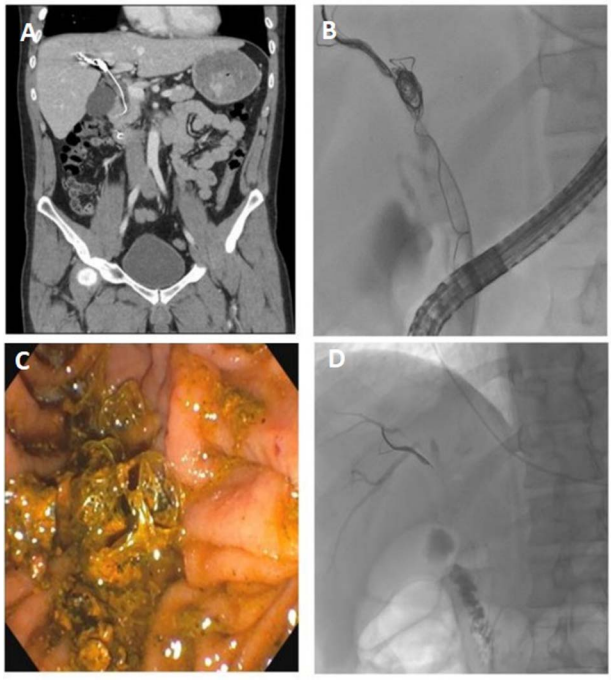
Introduction: Embolization coils are commonly used to manage hemorrhage after liver injury; however, migration into the biliary tree is uncommon. When this occurs, symptoms of biliary obstruction might develop such as jaundice, cholangitis, stone formation and pancreatitis. Physicians may not be aware of this potential risk, resulting in a delay in diagnosis and an increased morbidity and mortality risk. Herein, we present the case of a patient with history of liver laceration requiring bile duct embolization, and concurrent bile leak, presenting >3 years later with ascending cholangitis.
Case Description/Methods: A 27-year-old male with remote history of motor vehicle accident complicated by a liver laceration presented to the ED with one-week onset of right upper quadrant (RUQ) pain, vomiting, and fever. Evaluation at admission was notable for fever of 100.3F, RUQ tenderness, White blood cell count of 13.000/mL. Liver chemistries with bilirubin of 5.0mg/dL, ALT 405U/L, AST 217U/L, ALP 200U/L, consistent with obstructive pattern. Computed tomography showed sequela of liver laceration and bile duct embolization in segment 8, with an unfolded portion of the embolization coil extending inferiorly to the common bile duct, and a dilated bile duct to 15mm (Figure A). The patient was started on antibiotics and underwent urgent ERCP with findings of a coil bundle in the common bile duct with adherent stone and cast material surrounding the coil cast (Figure B). The wire bundle was successfully removed using the extraction balloon and an endoscopic forceps. A significant amount of stone, sludge and pus was also removed from the duct (Figure C). The final cholangiogram demonstrated no further evidence of intraductal embolization coils or other filling defects (Figure D). His pain was relieved, and he was discharged with a common bile duct stent and outpatient follow-up.

Discussion: Cholangitis due to biliary coil migration represents an unusual, delayed complication from endovascular embolization. The migrated coil serves as a nidus for stone formation, leading to choledocholithiasis and cholangitis. Our patient was managed successfully with ERCP allowing coil extraction with a favorable outcome. Similar cases of this possible complication have been rarely described in the literature. Our case highlights an uncommon cause of life-threatening cholangitis, warranting a high degree of suspicion and need for detailed documentation of prior interventions.



[1702] **Figure 1.** A. CT showing an unfolded portion of the embolization coil extending inferiorly to the common bile duct. B. Cholangiogram showing a coil bundle in the common bile duct with multiple filling defects suggestive of stones. C. Significant amount of stone, sludge, and coils were removed from the biliary tree. D. Cholangiogram with complete removal of the coil bundle and no further filling defects. The second coil seen in theright upper quartrant were in the liver parenchyma or intra-vascular as confirmed by fluoroscopy.

S1703

Circumferential Pancreatic Pseudocyst Encasing the Duodenum Causing Gastric Outlet Obstruction

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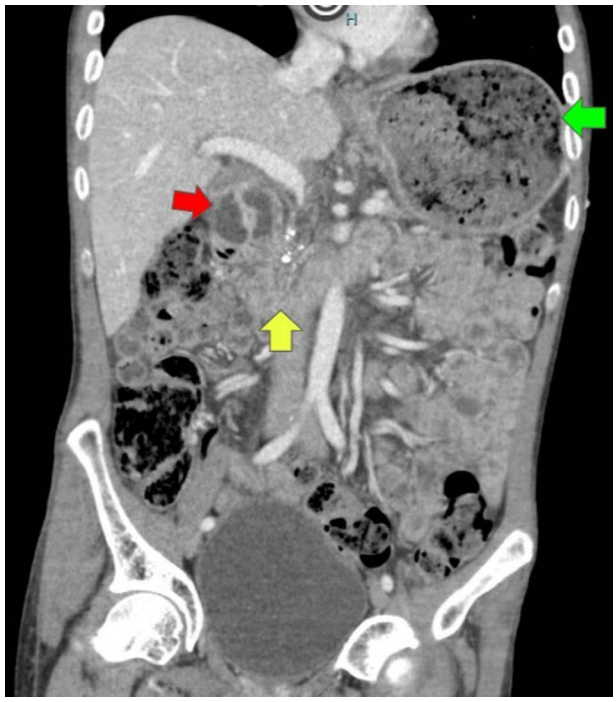
Introduction: Pancreatic pseudocysts are common findings that form from inflammation and fluid accumulation. Paraduodenal Pancreatitis is a form of chronic pancreatitis where inflammation is limited to the pancreatic head for unknown reasons, though chronic alcohol abuse and strictures of the distal common bile duct and pancreatic duct are commonly reported. Approximately 40% of these patients will have a pseudocyst in the pancreatic head and the presence of cysts in adjacent structures may occur in up to half of patients. Pseudocysts may cause obstructive symptoms in both the biliary and GI tracts, usually due to mass effect and not encasement. We present the case of a patient with a pseudocyst circumferentially enveloping the duodenum causing gastric outlet obstruction (GOO).
Case Description/Methods: A 35-year-old man with chronic pancreatitis and alcohol abuse presented after syncopizing. Upon arrival he was unresponsive, tachycardic, and had a distended abdomen with a left upper quadrant mass. The patient was profoundly hypoglycemic which was treated with continuous dextrose-containing fluids. A CT scan identified a donut shaped torus, with communication to a pancreatic duct, causing duodenal and GOO (Figure). Attempts to relieve this obstruction via nasogastric tube placement were unsuccessful. Endoscopy with intraoperative ultrasound (EUS) was performed, biopsies were taken, and 10 milliliters of fluid were aspirated. The biopsy was benign and the fluid had elevated concentrations of amylase and lipase confirming the diagnosis of pancreatic pseudocyst and a cystoduodenostomy was performed on a second EUS. Despite initial improvement the patient had continued difficulty eating with a follow up endoscopy revealing severe esophagitis and persistent GOO. Laparoscopic gastrojejunostomy with conversion to an open procedure due to adhesions was performed to bypass the lesion. His postoperative period was uneventful and had an uncomplicated discharge.
Discussion: The anatomy of this pseudocyst is rare and no guidelines exist regarding management. Prior to EUS many patients underwent invasive procedures, and patients have benefited from the availability of less invasive options. The anatomic characteristics of our patient's pseudocyst are rare and may represent a type of pseudocyst that requires more invasive management. Anatomic features of pseudocysts may be a reasonable way to stratify patients into noninvasive or invasive management options.



[1703] **Figure 1.** Abdominal CT of the patient with key findings. Red arrow- "Donut-Shaped" pancreatic pseudocyst enveloping the duodenum. Green arrow- Patient's stomach which is markedly distended and full of gastric contents. Yellow arrow- Head of the pancreas.

S1704

Casting a Wider Net: Rare Presentation of Biliary Cast Syndrome in a Non-Liver Transplant Patient

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[1]Franciscan Health Olympia Fields, Olympia Fields, IL; [2]Michigan State University College of Human Medicine, Chicago, IL; [3]St. Matthews University of Medicine, Chicago, IL; [4]St. Joseph Amita Health, Joliet, IL.

Introduction: Biliary cast syndrome (BCS) has been described in literature as the presence of casts within the confines of the biliary system, in extra or intrahepatic regions. A rare presentation, most cases of BCS have been described in patients who have undergone liver transplantation. The exact mechanism of BCS has yet to be clearly extrapolated but point to inflammation of the bile duct or ischemic injury of biliary endothelium. Here, we describe the incidental finding of BCS mimicking biliary parasitosis in a patient without history of liver transplantation and subsequent retrieval and pathological analysis.
Case Description/Methods: The patient is a 57-year-old male with past medical history of diabetes mellitus and prior venous thromboembolism on warfarin who presented with infected decubitus ulcers. His initial laboratory evaluation was significant for an incidental finding of elevated alkaline phosphatase level of 713 but total bilirubin level of 0.7 and AST/ALT levels of 17 and 11 respectively. He had a subsequent CT abdomen and pelvis as part of his workup which revealed an incidental finding of a 1.4cm x 1.1 cm lesion of the pancreatic head that was described as a likely pseudocyst. The patient was evaluated by gastroenterology and underwent EGD with EUS and FNA which revealed no cytological evidence of malignancy but did reveal a lesion in the common bile duct suspicious for polyp. A subsequent ERCP was performed which revealed a blockage in the bifurcation of the right and left hepatic ducts. There was initial concern for biliary parasite infestation, however final pathology reports demonstrated paucicellular proteinaceous fluid containing degenerated pancreatic tissue and chronic inflammatory cells suggestive of biliary casts.
Discussion: As cases of BCS are rare in transplant patients and exceedingly rare in those without transplant, the discussion focuses on the composition of biliary casts, potential causes of development and consensus means for treatment. Based on our knowledge and existing literature, only 6 commonly cited cases exist of BCS in non-transplant settings. No consensus currently exists on the management and evaluation of BCS, with most non-transplant patients undergoing ERCP for evaluation and removal of biliary casts. With this case, we contribute to the small body of literature on non-liver transplant regarding the clinical presentation of patients with BCS, the laboratories associated with BCS, as well as treatment options.

S1705

Celiac Disease and Chronic Pancreatitis

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Introduction: Celiac disease involves a T-cell-mediated immune response to dietary gluten. Celiac disease has 3-fold increased risk for developing chronic pancreatitis but only has a reported incidence of 0.52%. We present a case of a 61-year-old male with newly diagnosed celiac disease who was incidentally found to have associated chronic pancreatitis.
Case Description/Methods: A 61-year-old male with a past medical history of diabetes, hypertension, coronary artery disease, and a remote history of hepatitis presented to our clinic for his first screening colonoscopy and recent anemia. Prior to colonoscopy, the patient noted a 45-pound unintentional weight loss over the past few years, and with recent lab studies revealing a hemoglobin level of 11.7 g/dL. The patient then underwent colonoscopy, esophagogastroduodenoscopy (EGD), and an extensive laboratory work-up that was significant for iron deficiency anemia, anti-gliadin (AGA) IgA >150 U/mL, AGA-IgG 134 U/mL, endomysial (EMA)-IgA positive, tissue transglutaminase (tTG)-IgA >100 U/mL, total IgA 280 mg/dL, fecal elastase 31 mcg/g and was positive for anti-smooth muscle antibodies (1:160). Subsequent

results were negative for viral hepatitis panel, antinuclear antibodies, anti-mitochondrial antibodies, alpha-1 antitrypsin levels, and normal ceruloplasmin levels. Colonoscopy was unrevealing except for a diminutive tubular adenoma. EGD later revealed blunted villi with flat mucosa in the first and second portion of the duodenum. Biopsy results later confirmed pathology results compatible with celiac disease. During this course of workup, the patient developed nephrolithiasis and with incidental findings of pancreatic atrophy and pancreatic head calcifications detected on CT urogram. Chronic pancreatitis was later confirmed on endoscopic ultrasound with evidence of multiple intraductal stones. Patient did not have a history of alcohol use or pancreatic problems in the family. After being diagnosed with Celiac disease and secondary chronic pancreatitis with exocrine pancreatic insufficiency, he was started on a gluten-free diet and pancrelipase. Patient was noted to have 9-pound weight gain during a 4 month follow-up appointment.

Discussion: Chronic pancreatitis is a rare extraintestinal manifestation of celiac disease, reported to have a threefold increased risk but only with a reported incidence of 0.52% at least 30 days after diagnosis of celiac disease compared to 0.13% in non-celiac disease patients.

S1706

Chylous Ascites (CA) From Necrotizing Pancreatitis (NP): A Case Series

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Introduction: Chylous ascites (CA) occurs from obstruction or perforation of the peritoneal and retroperitoneal lymphatic system. Common etiologies include malignancy, cirrhosis and infections. On rare occasion, CA may be a consequence of necrotizing pancreatitis (NP). We report a case series of 5 NP patients who developed CA as a late sequalae.

Case Description/Methods: Five patients (males-2, mean age- 65 years) hospitalized at our institution between 2019-2022 with necrotizing pancreatitis developed CA after a median 85 (IQR 80-169) days from NP. NP etiologies varied (**Table**). All patients were endoscopically treated for infected walled off necrosis (WON) and 2 required percutaneous and surgical intervention a median 55 days (IQR 17-108) prior to CA development. CA presentation was variable. Patient A noted weight gain and painless abdominal distention with incidental new ascites on CT scan prior to scheduled endoscopic necrosectomy. Patient B reported fevers and abdominal pain and was found to have CA in the setting of suppurative cholecystitis and WON. Patient C had clinical concerns for secondary bacterial peritonitis (SBP) from a retroperitoneal abscess that initially improved with antibiotics but subsequently developed into CA. Patient D acquired CA during a prolonged NP hospitalization requiring abdominal washout and drain placement for loculated pancreatic ascites. Lastly, patient E developed CA while treated in the ICU for E.coli bacteremia and WON. The mean ascites triglyceride level was 644 mg/dL (IQR 463-750). No malignant cells were found on cytology. One patient had a SAAG >1.1. Lymphoscintigraphy failed to show lymphatic leaking in cases used. All patients were treated with a high protein and low fat diet with medium chain triglyceride (MCT) supplementation. One patient received octreotide and one was placed on TPN. Two patients had persistent CA at 3 month follow up, which resolved by 6 months. Three patients ultimately died over the next year due to complications of sepsis (n=2) and respiratory failure (n=1). One patient is still being followed.

Discussion: Chylous ascites is a rare complication of NP with an unknown natural history. The need for intervention for NP and development of systemic infection may increase this risk, as was common among our patients. Dietary modifications yielded variable outcomes in our group. Providers should be conscious of this potentially fatal complication.

Table 1. Characteristics, Management and Outcomes of Patients with Chylous Ascites

Parameter	Patient A	Patient B	Patient C	Patient D	Patient E
Gender	Male	Male	Female	Female	Female
Age, years	64	39	43	66	68
Etiology of NP	Idiopathic	HTG	HTG	Biliary	Biliary
NP interventions	Endoscopic	Endoscopic	Endoscopic, percutaneous, surgical	Endoscopic, percutaneous, surgical	Endoscopic
Indication for intervention	Infected WON	Infected WON	Infected WON	Infected WON	Infected WON, GOO
Days from NP to Chylous Ascites	169	80	1757	15	85
Days from last mechanical intervention to CA	143	55	108	14	17
Positive lymphoscintigraphy	No	No	No	No	No
Ascites triglyceride, mg/dL (IQR)	714	835	463	750	458
SBP	No	No	Yes	Yes	Yes
Malignancy	No	No	No	No	No
SAAG >1.1	No	Yes	No	No	No
MCT supplementation	Yes	Yes	Yes	Yes	Yes
Octreotide	No	No	No	No	Yes
TPN	No	No	No	Yes	No
Persistent CA (3 months)	n/a	Yes	No	No	Yes
Duration of CA, days	n/a	180	7	7	180
Cause of death	None	Sepsis	Sepsis	Sepsis, Respiratory failure	None

NP, necrotizing pancreatitis; HTG, hypertriglyceridemia; WON, walled off necrosis; GOO, gastric outlet obstruction; CA, chylous ascites; SBP, spontaneous bacterial peritonitis; SAAG, serum albumin-ascites gradient; MCT, medium chain triglycerides; TPN, total parenteral nutrition.

S1707

Case Report: Sepsis Due to *Klebsiella oxytoca* Bacteremia and Jaundice Originating From Von Meyenburg Complex Superinfection

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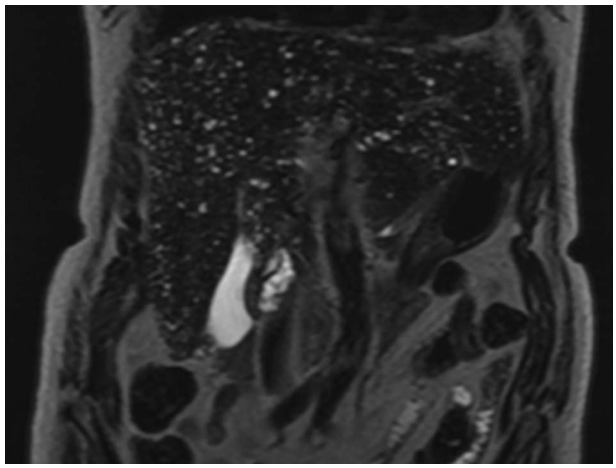
Introduction: Multiple biliary hamartomas (A.K.A. von Meyenburg Complexes, VMCs) are benign lesions found incidentally on hepatic imaging. Scarce reports describe complications in patients with VMCs, including abdominal pain, infection, portal hypertension, and cholangiocarcinoma.[2–8] We present a case of Klebsiella oxytoca bacteremia leading to sepsis and jaundice, originating from a hamartoma superinfection.

Case Description/Methods: A 75-year-old woman presented with malaise, jaundice and fever. She denied abdominal pain, vomiting or diarrhea. Past medical history was notable for alcoholic liver disease (non-cirrhotic), hepatic VMCs, and recently diagnosed lung adenocarcinoma. She was not taking any medications, supplements or vitamins and had not been abusing alcohol prior to her presentation. On arrival, she was tachycardic, hypotensive, but afebrile. Laboratories were noted for WBC 15.9/mm^3, ALP 260 IU/L, AST 88 IU/L, ALT 52 IU/L, total bilirubin 4.9mg/dL, direct 4.4mg/dL, creatinine 5.99mg/dL. Urinalysis ruled out pyuria and chest CT was without infection. Both abdominal US and CT were without biliary dilation or cholecystitis. Biliary hamartomas (VMCs) were unchanged from prior MRI (**Figure**). Blood cultures grew Klebsiella oxytoca. Clinical improvement, including improvement of serum bilirubin, was noted with 48 hours of cefepime. She was discharged in stable condition after 4 days of hospitalization.

Discussion: This case presents a gram-negative bacteremia of a known gastrointestinal tract/biliary pathogen, resulting in sepsis in a patient with VMCs. Potential sources of infection – including respiratory, urinary, hepatitis, cholangitis, and colitis – were excluded. Thus, superinfection of biliary hamartoma was the most likely cause for this patient's presentation. In the medical literature, there have been 7 reports describing VMC superinfection as a cause of sepsis. This case adds to that body of literature. The diagnosis of VMC induced sepsis is one of exclusion after other possibilities have been investigated and is a rare entity that should be recognized.



[1707] **Figure 1.** Image from patient's prior MRI abdomen demonstrating innumerable biliary hamartomas consistent with von Meyenburg complex.

S1708

Combination of Pancreatic and Hepatic Pseudocyst in a Patient With Pancreatitis: A Case Report

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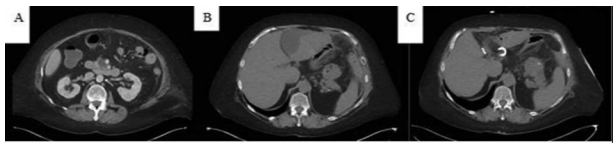
Introduction: Pancreatic pseudocysts are common occurrences after episodes of acute pancreatitis. Up to 20% of pseudocysts are extra-pancreatic throughout the abdomen including intrahepatic. There are only a few cases of hepatic pseudocysts reported in the literature. We report this unique case with both pancreatic and intrahepatic pseudocysts occurring at the same time in a patient with pancreatitis.
Case Description/Methods: A 56-year-old White woman with history of alcoholism and chronic pancreatitis, presented with epigastric and left upper quadrant abdominal pain radiating to the left shoulder. Examination revealed tenderness in the epigastric and left upper quadrant of the abdomen. Labs showed an elevated lipase of 217 IU/L (13-60), normal total bilirubin and transaminases, hemoglobin of 9.1 g/dL(11.5-15), and slight leukocytosis 12.6 K/uL (3.7-10.4). She was admitted 3 times in the last 4 months. Prior computerized tomography (CT) scan showed pseudocysts in the head and tail of the pancreas. CT scan this admission showed a decreased size of pseudocyst in the pancreatic head from 2.5 cm to 2.2 cm and pancreatic tail from 4.2 cm to 3.7 cm (Figure). It also showed 2 subcapsular fluid collections in the left lobe of the liver measuring 9X5X2cm and 9X7X3 cm. Percutaneous drainage of the hepatic cysts with the placement of a drainage catheter was performed by interventional radiology. 50 mL of bilious appearing fluid was drained with negative gram stain and cultures with an elevated fluid amylase level of 1,765 U/L. The patient's pain improved and a subsequent CT a few weeks after discharge showed a decrease in the size of the hepatic cysts.
Discussion: This is the first documented case of combined pancreatic and intrahepatic pseudocyst formation. Elevated amylase level in the hepatic fluid analysis confirmed the diagnosis of a hepatic pseudocyst. The interesting fact is that the size of the pancreatic pseudocyst decreased as the hepatic pseudocyst increased because of fluid tracking likely in the pre-renal space or along the hepato-gastric ligament. The diagnosis of hepatic pseudocyst needs to be considered in a patient with new hepatic fluid collection with a recent history of pancreatitis and pancreatic pseudocyst.



[1708] **Figure 1.** A – Axial CT showing previous pancreatic pseudocyst prior to admission B – Axial CT showing new hepatic pseudocyst adjacent to falciform ligament during admission C – Axial CT showing drain in hepatic pseudocyst.

S1709

Cholecystitis: A Rare Presentation for Diffuse Eosinophilic Granulomatosis With Polyangiitis

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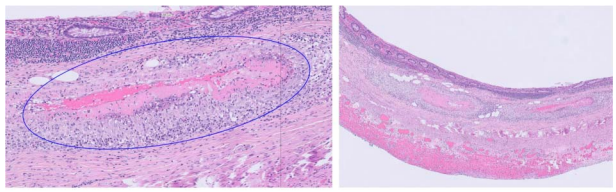
Introduction: Eosinophilic granulomatosis with polyangiitis (EGPA) is a rare disease with an incidence of 1-3 cases per 100,000. It results from small to medium vessel vasculitis driven by eosinophil rich inflammation and anti-neutrophil cytoplasmic antibodies. It usually manifests with airway inflammation, followed by tissue damage in the lungs and digestive tract. Gastrointestinal involvement has been seen in roughly 50% of patients, but involvement of the gallbladder is very rare. Our case highlights a unique scenario of cholecystitis leading to an official pathological diagnosis of EGPA.
Case Description/Methods: A 17-year-old woman with a history of asthma presented for 2 weeks of nausea, vomiting, abdominal pain, decreased urinary output, and 20 pound weight loss. Labs were remarkable for a WBC of 33.3, granulocytes of 10.46, eosinophils of 17.44, ESR 78, CRP 65.6, and a urinalysis showing 20-50 rbcs. Renal ultrasound was done to evaluate for pyelonephritis, but incidentally found slight thickening of the gallbladder wall and biliary sludge. She was started on ceftriaxone, and due to her clinical picture, a cholecystectomy was performed. Despite elevated WBC and eosinophils, she was discharged as symptoms improved. Gallbladder pathology returned and showed necrotizing vasculitis with fibrinoid necrosis and marked eosinophilia concerning for EGPA (Figure). New work up noted positivity for ANCA. At follow up, she acutely developed profound weakness in right upper and bilateral lower extremities, so she was re-admitted for EGPA related peripheral vasculitis neuropathy. Despite being started on steroids, she had a long hospitalization including bowel perforation, respiratory failure, seizures and glomerulonephritis. Ultimately, she stabilized on antibiotics, high dose steroids, and cytoxan and was discharged to follow up.
Discussion: EGPA involving the gallbladder is extremely uncommon. As blood vessel inflammation and eosinophilic proliferation are the foundation of illness, any organ system can be involved. Lung involvement with an asthma equivalence is usually the initial presentation, but in patients with intestinal involvement, nausea, vomiting, and intestinal perforation can be seen. Biliary involvement can present as a cholecystitis picture. This case provides a degree of educational value for everyone. Early recognition is important as a multi-specialty approach is crucial, and early immunosuppressive therapy can prevent escalation of disease.



[1709] **Figure 1.** Eosinophilic Vasculitis of Gallbladder and Appendix.

S1710

Cholelithiasis Causing Asymptomatic Cholecystoenteric Fistula

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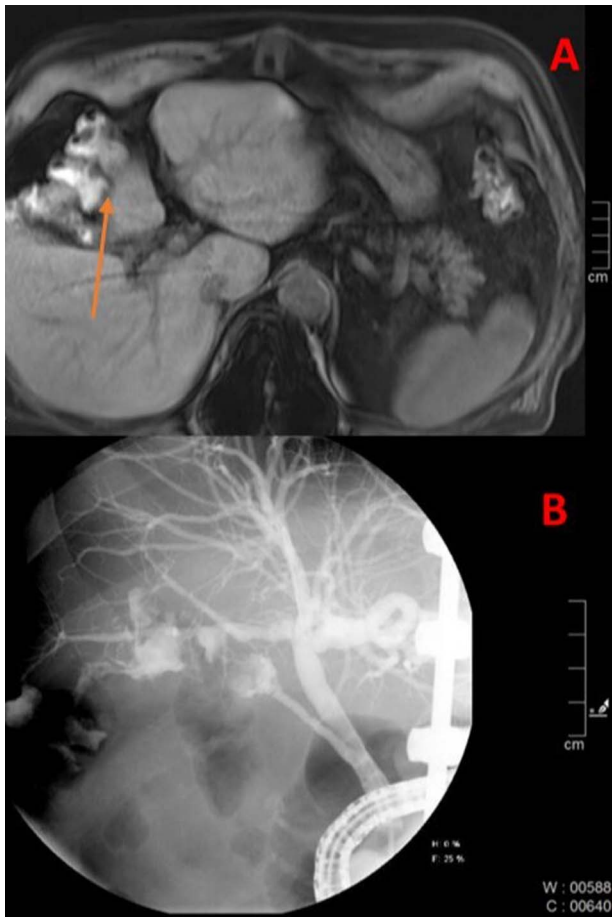
Introduction: Biliary fistulas are a rare complication of gallstones. Fistula formation can occur in a number of adjacent sites; even more rare complication is the formation of a cholecystocolonic fistula.
Case Description/Methods: A 74-year-old man who had recently undergone an extensive hospitalization secondary to inflammatory demyelinating polyneuropathy (IDP) and COVID-19 infection. During his hospitalization, he required ICU admission and mechanical ventilation with subsequent PEG tube placement. He was discharged to an inpatient rehabilitation facility when he developed worsening respiratory distress. Laboratory examinations were pertinent for ALT of 252, AST of 140 and ALP of 401 without hyperbilirubinemia. Blood cultures revealed Escherichia coli bacteremia. Given transaminitis and bacteremia, an MRCP was performed which demonstrated evidence absent space between gallbladder and hepatic flexure of the colon suggesting a CCF (Figure A). An ERCP with sphincterotomy was performed which showed extravasation of contrast from the gallbladder into the colon at the hepatic flexure (Figure B). He underwent cholecystectomy and fistula repair without any complications and gradual improvement in liver function test. He was discharged to a rehabilitation facility.
Discussion: Complications of gallstones are well established, which include the common bile duct obstruction, but also include the rare occurrences of acute cholangitis, malignancy, and fistula formation. CCF is a rare complication of gallstones which can occur in the stomach, duodenum, or colon with a variable clinical presentation. Complications from an undiagnosed fistula can be life threatening including colon perforation and fecal peritonitis. This case highlights the diagnostic challenge and the high degree of clinical suspicion involved in establishing the diagnosis of CCF in patient without abdominal symptoms suggestive of gallbladder disease. We hypothesize that stone formation resulting in the development of the fistula may be secondary to the underlying history of IDP and subsequent immobility. Although rare, CCF should be considered in patients presenting with unexplained pneumobilia and bacteremia. A timely diagnosis should be made to proceed with immediate treatment including cholecystectomy and fistula closure to prevent fatal complications.



[1710] **Figure 1.** MRCP imaging of colon abutment with gallbladder (A) and ERCP cholangiogram showing extravasation of contrast into the hepatic flexure of the colon (B).

S1711

Cerebral Infarction as a Rare Downstream Complication of Pancreatitis

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Introduction: Pancreatitis is characterized by progressive and irreversible loss of function. Long term complications of pancreatitis include refractory abdominal pain, malabsorption, and diabetes. The downstream complications may also be systemic. In the case below we suggest that the acute release of pancreatic enzymes led to a hypercoagulable state and severe inflammatory reaction contributing to an acute cerebrovascular stroke presentation.
Case Description/Methods: 55-year-old right-handed woman with a past medical history significant for chronic idiopathic pancreatitis, HTN, chronic thrombocytosis, and HLD presented to the hospital with complaints of nausea and vomiting. Initial work-up was significant for a lipase of 1,178 U/L and subsequent imaging showed peritoneal thickening and nodularity extended from the pancreatic head / neck junction most likely due to ongoing pancreatitis. Patient was admitted to hospital medicine but unfortunately that night the patient became acutely aphasic and confused. She developed right-sided neglect, profound aphasia, and left gaze preference. She was immediately evaluated by neurology and a stat CT revealed acute ischemic changes but no large vessel occlusion. She received intravenous tPA and was transferred to the critical care unit. CT angiogram revealed thrombus within the aortic arch at the origins of the right brachiocephalic arteries and follow-up MRI brain revealed scattered infracts within the left MCA territory. It was suspected that her stroke was likely precipitated by artery-to-artery embolus worsened in the setting of her acute on chronic pancreatitis attack. Patient worked extensively with rehabilitation services for weeks before eventually being discharged home with residual weaknesses.
Discussion: Acute on chronic pancreatitis is an inflammatory process that can produce complications through both loss of endocrine and exocrine function. Additionally, the thrombotic tendency in pancreatitis may be related to systemic hypercoagulability resulting from inflammatory cytokines, endothelial dysfunction due to release of proteases and lytic enzymes, and compression of vessels from surrounding inflammation. These factors could contribute to the development of acute cerebrovascular stroke as seen in our patient. Furthermore, this case serves to highlight the need for further study into the systemic effects of acute on chronic pancreatitis and to serve as a reminder that though rare cerebral infarction can be a rare complication of pancreatitis.

S1712

Choledochal Cyst Mimicking the Gallbladder With a Surprising Twist

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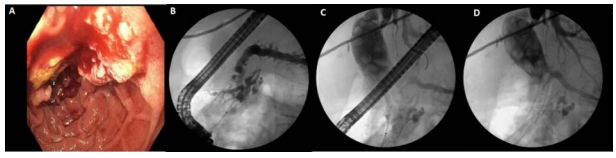
Introduction: Choledochal cysts are congenital rarities and often a pediatric diagnosis but 20% go undetected until adulthood. Etiology remains unclear but an obstructed common bile duct (CBD) has been identified as a pathogenic feature. We present a rare case of choledochal cyst initially mimicking the gallbladder in an elderly female.
Case Description/Methods: A 90-year-old woman presented to the hospital for abdominal pain and unintentional weight loss. Past medical history was notable for breast cancer and colorectal cancer status post resection. Liver function tests and pancreatic markers were normal with mildly elevated alkaline phosphatase. CT abdomen and pelvis revealed a 2.8 cm dilated common bile duct (CBD), choledocholithiasis, pancreatic duct dilation and absent gallbladder. Patient however denied undergoing a cholecystectomy. Given the discrepancy of imaging and history, an abdominal ultrasound was performed demonstrating large gallstones in the gallbladder with normal CBD size. Endoscopic ultrasound (EUS) revealed a 28x26 mm mass in the ampulla (Figure). FNB was performed. The distal CBD measured 7 mm with a 35 mm proximal CBD consistent with a choledochal cyst full of large stones. Pancreatic duct measured up to 8 mm. Gallbladder was confirmed to be surgically absent. ERCP was performed which revealed an infiltrative 3-4 cm mass at the major papilla. Pancreatic and biliary sphincterotomies were performed and metal stents were placed in the ventral pancreatic duct and CBD. Biopsies taken revealed pancreatic adenocarcinoma. The following month, hepatobiliary surgery performed a Whipple procedure (pancreaticoduodenectomy). Pathology findings confirmed pancreatic adenocarcinoma with metastasis to one of twelve lymph nodes. Patient was arranged to follow up with outpatient oncology.
Discussion: Choledochal cysts can be an elusive diagnosis in elderly adults given its rarity, prevalence in children, and common presentation of vague abdominal pain. Choledochal cyst should be part of the differential diagnosis regardless of patient age especially in cases of accompanying weight loss due to its increased risk of biliary malignancy. In our case, the patient was found to have choledochal cyst misrepresented as the gallbladder and incidentally found to have pancreatic adenocarcinoma. Additionally, patients may be unreliable historians leading to misinterpretation of imaging results. Further investigation is therefore warranted in these perplexing cases for an accurate diagnosis and appropriate management.



[1712] **Figure 1.** A. Ampullary mass B. Pancreatic duct dilation C. Choledochal cyst. D Post ERCP.

S1713

Direct Bilirubinemia and Arthralgia as Presenting Features of Anaplasmosis

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Introduction: Human granulocytic anaplasmosis (HGA) is a tick-borne illness caused by Anaplasma phagocytophilum, and is transmitted by Ixodes scapularis tick. Hyperbilirubinemia is an unusual feature of HGA, and if present, is usually indirect. We present a case of HGA who presented with jaundice and a non-obstructive direct hyperbilirubinemia.
Case Description/Methods: A 60-year-old man with a history of hypogonadism and no known history of chronic liver disease presented with a 2-day history of yellowish eye discoloration and dark urine. These symptoms were associated with fever, chills, and arthralgias in bilateral hips and elbows. The patient resides in the New England area and spends significant time outdoors. He denied abdominal pain, nausea, vomiting, diarrhea, dizziness, joint swelling, skin rashes, tick bites, or antibiotic use. Vital signs were normal, and he was afebrile. Physical examination was remarkable for jaundice and mild tenderness at the elbows with no redness or swelling. The abdominal exam was normal. Laboratory tests revealed an AST of 93 IU/L, ALT of 151 IU/L, ALP of 310 IU/L, Total bilirubin of 7.5 mg/dl (direct bilirubin 4.8 mg/dl), Hgb 16.1 g/dl, platelets 41 x10exp9/L, reticulocyte index 0.82, LDH 256 IU/L, haptoglobin 197 mg/dl (Table), and INR of 1.6. CT scan of the abdomen demonstrated normal liver and biliary tree morphology without evidence of biliary dilation. Given the relatively high prevalence of tick-borne illnesses, the patient was empirically placed on doxycycline. The anaplasma Phagocytophilium IgG ab titer was strongly positive (1:320), and acute infection was confirmed with PCR. Ehrlichia PCR and Lyme serology were negative; babesiosis was also ruled out. 48 hours after starting therapy, the patient's jaundice and all other symptoms resolved.
Discussion: We present an unusual case of HGA presenting with direct hyperbilirubinemia and mild transaminitis in the absence of biliary obstruction. The pathophysiology of direct hyperbilirubinemia in HGA in not known. HGA is prevalent in the Northeast and Upper Midwest of the United States, and it is a common cause of undifferentiated fever in these regions. Infection, in most cases is mild, but rarely lead to life-threatening complications including demyelinating disorders, secondary infections, renal failure, and seizures. Hence, recognition and early therapy are essential. Although rare, direct hyperbilirubinemia can be a manifestation of HGA.

Table 1. Summary of initial laboratory tests' results

Laboratory test	Reference Range & Units	Value
WBC	3.5 - 11.0 x10exp9/L	8.4
Hemoglobin	13.5 - 16.0 G/DL	14.8

Table 1. (*continued*)

Laboratory test	Reference Range & Units	Value
Platelets	150 - 400 x10exp9/L	43
Reticulocyte Index		0.82
Haptoglobin	40 - 268 MG/DL	197
LDH	100 - 220 IU/L	256
ALT	6 - 45 IU/L	151
AST	10 - 42 IU/L	93
Alkaline Phosphatase	34 - 104 IU/L	310
Bilirubin, Total	0.2 - 1.3 MG/DL	7.5
Bilirubin, Direct	0.0 - 0.3 MG/DL	4.8
Anaplasma Phagocytophilium PCR		Detected
A. Phagocytophilum Ab, IgG	< 1:80	1:320

S1714

Diagnosis and Management of Intraductal Papillary Neoplasm of the Bile Duct via Endoscopic Ultrasound Evaluation

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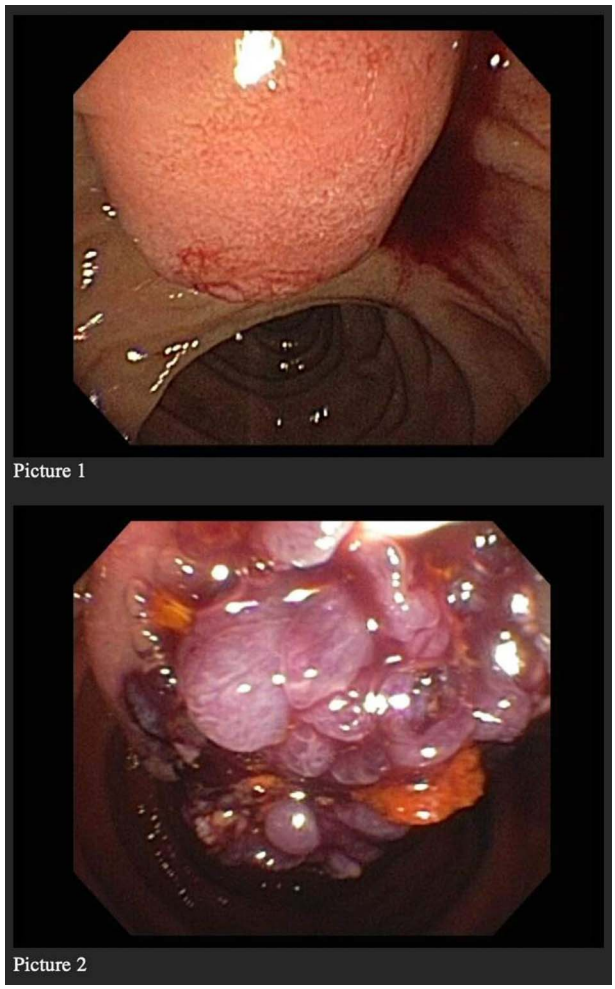
Introduction: Intrampullary papillary neoplasm (IAPN) is a rare tumor seen in women in their 60s, with an incidence of 0.025 per 100,000. The 5-year survival rate is 33%. It is distinct from an ampullary adenoma, which arises from the duodenal mucosa overlying the ampulla. It is unlikely to be cured by ampullectomy.
Case Description/Methods: A 63-year-old woman with prior cholecystectomy was found to have abnormal liver tests (AST 104, AST 233, alkaline phosphatase 290) and mild hyperbilirubinemia (total bilirubin 1.7). Abdominal ultrasound showed diffuse biliary dilatation without obvious stones or stricture. MRCP showed multiple filling defects in the distal common bile duct (CBD). ERCP revealed a prominent ampulla. Digital cholangioscopy done for further investigation showed an exophytic papillary tumor in the distal CBD (Figure) along with excess luminal mucous (Figure). The endoscopic and cholangioscopic findings of a papillary intraluminal mass and mucobilia was suggestive of intraductal papillary neoplasm of the bile duct (IPNB). A 69-year-old woman with a complicated biliary history was seen by her PCP for jaundice and R shoulder pain and was found to have cholelithiasis and choledocholithiasis. She underwent laparoscopic cholecystectomy after MRI showed CBD stones. An ampullary prominence of 1 cm was concerning for an invasive carcinoma. ERCP showed multifocal frond like papillary projections at the left hilum and distal CBD and copious thick mucin through the examined extrahepatic duct and hilum; concerning for IPNB with progression to cholangiocarcinoma. Both patients underwent surgical evaluation.
Discussion: IAPN is a rare tumor with poor prognosis. It is unclear if it is a variant of IPNB that also includes the ampulla or if it primarily arises within the ampullary channel and spreads proximally into the bile duct and the pancreatic duct. Diagnosis is established by cholangioscopy with biopsies. Endoscopic ultrasound of the ampulla and pancreas is essential. Cholangioscopy should be undertaken for tumor mapping. Pancreaticoduodenectomy should be treatment of choice for IAPN or IAPN with adenocarcinoma without distant metastases in order to achieve tumor free margins, as seen via pathology tissue. IAPN should be suspected based on endoscopic ampullary features such as a prominent ampulla, prolapse of papillary fronds on sphincterotomy, lack of resolution of cholestasis despite duct clearance. Cholangioscopy should be undertaken establish an early diagnosis.



[1714] **Figure 1.** Photo 1 (top): exophytic papillary tumor in the distal common bile duct; Photo 2 (bottom): excess luminal mucous in common bile duct.

S1715

Cystic Artery Aneurysm Mimicking Mirizzi Syndrome: A Rare Case Complicated by Severe Gangrenous Cholecystitis

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Introduction: Mirizzi syndrome (MS) is a rare condition caused by an impacted gallstone at the gallbladder (gb) infundibulum causing obstructive jaundice, cholecystitis and visceral inflammation. Most reported cases of visceral aneurysms reference cystic artery pseudoaneurysms (CAP) caused by inflammation. Here we present a case of cystic artery aneurysm (CAA) mimicking neoplasm on imaging and presented as painful jaundice complicated by gangrenous cholecystitis (GC).
Case Description/Methods: 69-year-old-man with alcohol abuse presented with 2 weeks of worsening RUQ abdominal pain, vomiting, jaundice and 15 kg unintentional weight loss. Vitals were stable and he had mild RUQ tenderness with negative murphy's sign. Pertinent labs are listed in the **Table**. CT was significant for hepatic mass infiltrating the gb with intrahepatic biliary duct dilation. MRCP revealed possible hepatic or celiac artery aneurysm abutting the gb causing biliary obstruction resulting in ERCP and biliary stent placement. CTA abdomen, doppler US liver and IR angiogram confirmed large cystic artery aneurysm adherent to gb wall. Despite antibiotics, he was clinically deteriorating with worsening leukocytosis, fever and Klebsiella pneumoniae bacteremia. With suspicion of possible fistula between gb and CAA, and with multidisciplinary recommendations, he underwent exploratory laparotomy with cholecystectomy. Intraoperative findings revealed CAA fistulizing into gb with haemobilia and gangrenous cholecystitis with gallstone. Pathology and serum markers were negative for neoplasm. He survived this complicated case without complication.
Discussion: Similar to MS, CAA can cause mass effect leading to obstructive jaundice, cholangitis and in rare cases haemobilia and life-threatening hemorrhage. Like CAP, the etiology of CAA is postulated to be an inflammation from cholecystitis, pancreatitis, intraabdominal trauma or iatrogenic injury. Our patient had gallstone, hence MS complicated by cholangitis and gangrenous cholecystitis is likely etiology for CAA. Angiographic embolization or open surgical repair are reported modalities of its management with mortality around 2%. GC alone carries mortality as high as 22% and concurrent CAA with cholangitis definitely makes prognosis guarded. Initial CT abdomen was suspicious for a neoplasm hence in a resource limited country and without multidisciplinary evaluation with appropriate imaging modalities, it could easily be mismanaged as malignancy, which could lead to an unfavorable prognosis for the patient.

Table 1. Pertinent Labs

Labs: Liver Function Test	AST (U/L)	ALT (U/L)	ALP (U/L)	Total Bilirubin (mg/dl)	Direct Bilirubin (mg/dl)
On admission	594	539	430	19.2	>13
Day 2	124	254	1003	17.3	12.1
Labs: CBC	WBC (k/ul)	Hgb	PLT (k/ul)	Differentials	
	12.8	9.2	103	No bands	
Labs: Others	INR	CEA	AFP	Ca 19-9	Hepatitis Profile
	2.2	Normal	Normal	Mild elevation	Negative

S1716

Diagnostic Challenges of an Insulinoma in a Healthcare Worker

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Introduction: Insulinomas are the most common cause of hypoglycemia related to endogenous hyperinsulinism. It occurs in 1-4 people per million in the general population. Symptoms include diaphoresis, palpitations, tremors and even confusion or behavioral/personality changes.1 The small size of insulinomas presents a challenge in diagnosis via standard imaging techniques.2
Case Description/Methods: A 47-year-old healthcare worker with a history of goiter presented with 6 months of memory problems and associated lightheadedness, tremors, and blurry vision. She endorsed a 15-pound weight gain in the past month. She also recalled lapses in memory such as forgetting where she parked her car at the grocery store and more dangerously occurring during work when she was not able to remember if she administered medications to patients appropriately. Historical episodes of hypoglycemia had been ongoing for the past 2 years. Baseline labs included a mildy elevated c-peptide, normal IGF-2, TSH, and cortisol, and negative insulin antibody and sulfonyurea screen. Physical examination was normal. MRI showed no pancreatic abnormalities and she was subsequently admitted to our hospital for 72-hour inpatient fast. The fast ended at 22 hours due symptomatic hypoglycemia that resolved after administration of glucagon. Labs revealed blood glucose of 42, insulin 9 mU/L, beta-hydroxybutyrate 0.4 mg/dL, C-peptide 1.4 ng/L , proinsulin 40.5 pmol/L which were all diagnostic for insulinoma. CT abdomen showed no enhancing pancreatic masses or metastatic lesions. Thus, the patient underwent endoscopic ultrasound (EUS) fine-needle aspiration which identified an 8x5 mm lesion in the tail of the pancreas. Pathology showed a well-differentiated neuroendocrine tumor, and the patient underwent radiofrequency ablation (RFA). Four months later, the patient was no longer symptomatic and no biochemical evidence of hyperinsulinemia remained on laboratory evaluation.
Discussion: Insulinomas remain a diagnostic challenge, due to their rare presentation, nonspecific symptoms and small size. Although most insulinomas are benign with survival rate of 95% they require surgical intervention or radiofrequency ablation to improve survival. Diagnosis of a suspected insulinoma can be made via labs during a supervised, prolonged fast and accurate localization of smaller tumors may require minimally invasive procedures, like EUS.[2]

S1717

Direct Bilirubinemia 15 Years After Cholecystectomy: A Case of Post-Cholecystectomy Syndrome

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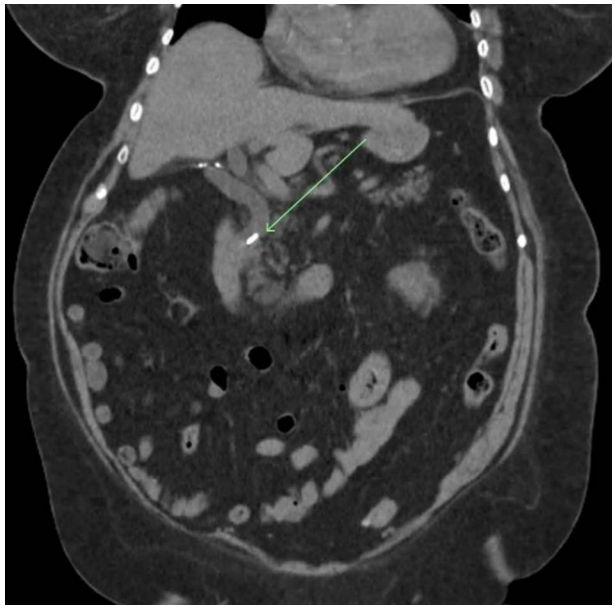
Introduction: Post cholecystectomy syndrome constitutes a variety of clinical signs and symptoms of the GI tract after cholecystectomy which can occur from days to years after the procedure. One of the well-known serious complications is biliary obstruction. However, rarely are there complications related to migration of the surgical clip. Clip migration as a late complication of cholecystectomy in the setting of this syndrome can at times be overlooked resulting in a delay of care and worsened prognosis.
Case Description/Methods: We present a 66-year-old female patient with a past medical history of diabetes, hypertension, and choledocholithiasis s/*P* cholecystectomy after ERCP with sphincterotomy 15 years prior who presented to the emergency department with acute and progressive right upper quadrant abdominal pain for 2 days. Initial physical exam revealed RUQ abdominal tenderness without Murphy's sign. Patient was afebrile and tachycardic. Laboratory studies revealed elevated AST, ALT, alkaline phosphatase 170 U/L, 254 U/L, and 227 IU/L respectively. Direct and total bilirubin were elevated at 2.1 mg/dL and 4.4 mg/dL respectively. CBC was unremarkable. CT abdomen/pelvis without IV contrast showed CBD of 1.2cm, surgical clip in the CBD as well as pneumobilia. The patient underwent EUS/ERCP with findings of surgical clip in the CBD, 1 cm dilation of the CBD, prior evidence of sphincterotomy, and new narrowing of the ampullar opening suggestive of ampullar stenosis. Biliary sphincterotomy and occlusion cholangiogram was performed and multiple pigmented stones, pus, as well as the metallic clip were extracted from the distal aspect of the CBD. Finally, CBD was stented with good post-procedure bile flow. Patient was discharged to complete 10 days of antibiotics.
Discussion: One of the interesting and unusual aspects of this case was her late presentation with classic features of choledocholithiasis and "cholecystitis" with prior history of cholecystectomy. Additionally, multiple factors contributed to her clinical picture, including biliary stones, surgical clip, as well as ampullary stenosis even with history of sphincterotomy. The above findings could represent multiple etiologies for patients signs and symptoms in this rare case of post cholecystectomy syndrome (Figure).



[1717] **Figure 1.** CT scan of abdomen and pelvis without IV or PO contrast demonstrating prior cholecystectomy with a cholecystectomy clip within the common bile duct causing a 1.2cm duct dilation.

S1718

Delayed Gastrointestinal Hemorrhage From Visceral Artery Pseudoaneurysm in Necrotizing Pancreatitis After LAMS Removal

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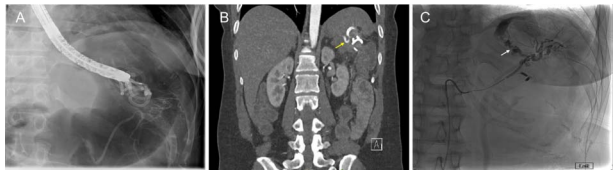
Introduction: Delayed bleeding from visceral artery pseudoaneurysm (PA) formation is an uncommon complication of lumen-apposing metal stent (LAMS) placement. Risk factors for gastrointestinal bleeding (GIB) in patients with LAMS remain largely undefined. We report a case of delayed GIB due to rupture of a splenic artery PA 16 months after LAMS removal.
Case Description/Methods: A 34-year-old female with a history of severe necrotizing pancreatitis presented to our hospital with massive hematemesis. 23 months prior to presentation, she underwent uncomplicated placement of a 15x10 mm LAMS and 2 7 Fr x 4 cm double pigtail plastic stents for drainage of walled-off pancreatic necrosis. She was briefly lost to follow-up and returned for LAMS removal 5

months later. Contrast was injected into the residual cyst cavity and contrast filling was noted of what appeared to be a splenic vessel without active bleeding (Figure A). CT angiogram showed no PA but proximity of the splenic hilar vasculature to the LAMS (Figure B). The LAMS was successfully removed 2 months later without complication. On presentation, she was tachycardic but normotensive. Laboratory studies were notable for hemoglobin 8.8 g/dL (baseline 14 g/dL). Upper endoscopy showed brisk bleeding in the proximal gastric body near the area of prior LAMS. Her bleeding was refractory to endoscopic hemostatic maneuvers, and she underwent emergent mesenteric angiography. Angiography revealed active arterial extravasation arising from the inferior segmental branch of the splenic artery (Figure C) which was successfully embolized with no further bleeding.

Discussion: Visceral artery PA development after LAMS placement is thought to occur due to friction of the inner flange against regional vasculature surrounding the necrotic cavity as it collapses. Close anatomical proximity of LAMS to regional vasculature increases the risk of PA development. In our case, the LAMS was placed near the rich vascular supply of the splenic hilum. Most reported bleeding events related to LAMS have occurred within 8 weeks of placement. Our case is notable for the prolonged delay between LAMS removal and bleeding presentation. Adherent scar tissue between the gastric wall and vasculature could potentially cause delayed PA development after LAMS removal. This case reinforces that high clinical suspicion for PA bleeding is necessary for patients who have previously undergone distant LAMS removal who present with massive GIB.



[1718] **Figure 1.** (A) Fluoroscopic image of contrast injection into residual cyst cavity demonstrating opacification of likely splenic vasculature; (B) Coronal images demonstrating the proximity of the LAMS to the vasculature of the splenic hilum (yellow arrow); (C) Super-selective angiography demonstrating active extravasation into the lateral aspect of the stomach from a pseudoaneurysm (White arrow) involving an inferior segmental branch of the splenic artery.

S1719

Digesting a Differential of Zebras in a Unique Case of Pancreatitis

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Introduction: While acute pancreatitis is often gallstone or alcohol related, initial workup usually includes consideration of medications, triglyceride level, malignancy, and autoimmune pancreatitis. There remains however a list of rare causes that is often overlooked and misdiagnosed as idiopathic. This is unfortunate and may delay potentially life-saving therapy. Here, we present a unique case of acute pancreatitis seropositive for lupus and mumps that spiraled into lupus cerebritis.

Case Description/Methods: A 42-year old male (PMHx. Hypertension) presented with a 6-week history of abdominal pain, weight loss, fever, parotitis and odynophagia. History was significant for close-contact confinement and negative for alcohol use. He was febrile and tachycardic. US was negative for gallstones. CTA showed acute pancreatitis. Labs were significant for WBC 3.21 (abs lymph 0.29), AST 128/ALT 75. Lipase, TSH, lipid panel, and IgG panel was unremarkable. An extensive viral panel was ordered and positive for mumps IgM. He was aggressively fluid resuscitated and developed clinical improvement throughout the week before an unexpected relapse of his symptoms. Over the next few weeks, he developed erythematous and vasculitic appearing rashes, arthralgias, oral ulcers, pericarditis, and eventually acute encephalopathy that required intubation. Skin biopsy showed leukocytoclastic vasculitis and connective tissue disease panel was positive for anti-dsDNA and ANA. He was started on high-dose pulse steroids for lupus cerebritis with remarkable improvement.

Discussion: We present a unique case of lupus-pancreatitis in a patient without any prior history. He was seropositive for mumps, which we ordered due to his history. However, mumps-pancreatitis is rare and usually mild. Interestingly, his clinical deterioration coincided with clinical features and serology consistent with lupus. Lupus can falsely elevate mumps IgM and his pancreatitis was likely from lupus-related vasculitis. Lupus-pancreatitis is uncommon and typically presents during lupus flares. To our knowledge, this is the first report of acute pancreatitis as an initial presentation of lupus cerebritis. Lupus-pancreatitis responds to prompt administration of IV steroids. Unfortunately, our patient's late lupus presentation delayed immunotherapy. Therefore, we recommend consideration of connective tissue diseases in the autoimmune workup of persistent or relapsing pancreatitis.

S1720

Diabetic Ketoacidosis as a Rare Complication of Alcoholic Pancreatitis in a Previously Euglycemic Patient

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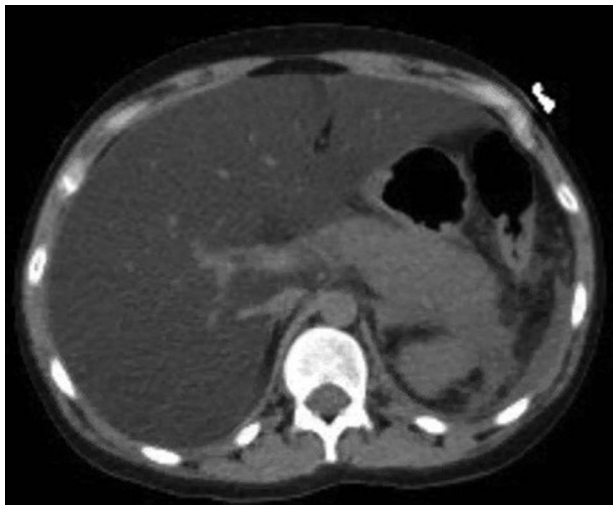
Introduction: Acute alcoholic pancreatitis is an acute inflammation of the pancreas secondary to alcohol intake due to autodigestion from an increase in pancreatic enzyme release. Most commonly due to gallstones, alcohol intake, and hypertriglyceridemia, pancreatitis commonly presents with severe epigastric abdominal pain, nausea and vomiting and is managed with aggressive intravenous (IV) fluid resuscitation. Some complications include acute respiratory distress syndrome and compartment syndrome. A very rare complication of acute pancreatitis is diabetic ketoacidosis (DKA), especially in patients without a history of diabetes mellitus.

Case Description/Methods: A 25-year-old female with a medical history significant for alcohol abuse presented with nausea, vomiting, and abdominal pain for 2 days. The patient reported drinking one pint of liquor per day. She was tachycardic with a heart rate of 115 and other vitals were normal. Significant laboratory studies showed a lipase of 1715, aspartate transaminase of 292, alanine transaminase of 155, glucose of 540, venous pH of 7.277, bicarbonate of 9, anion gap of 21, and beta-hydroxybutyrate of 5.1. A computed tomography scan of her abdomen and pelvis without IV contrast showed edematous and inflammatory changes in the tail of the pancreas and severe hepatic steatosis (**Figure**). The patient was managed with aggressive IV fluids and IV insulin infusion. Her abdominal pain and anion gap resolved on day 2 of admission and she was able to tolerate a diet. Glycated hemoglobin (HbA1c) returned a value of 4.7 and C-peptide was low at < 0.10; the subsequent pancreatic antibody panel was negative. The patient was seen in the clinic 6 months later and HbA1c at that time was 4.8.

Discussion: This case illustrates DKA as a rare complication of acute pancreatitis. It provides an example of why a high level of suspicion for DKA should be present in patients presenting with alcoholic pancreatitis even in the absence of any diabetic history. Though there is no definitive mechanism of action for this presentation, it is likely from a combination of pancreatic beta-cell destruction and counter-regulatory hormone effects. Pancreatic beta-cell destruction causes impaired insulin production, leading to acute hyperglycemia. Furthermore, as acute pancreatitis can lead to severe intravascular depletion, increased secretion of counter-regulatory hormones such as cortisol and glucagon, in combination with impaired insulin secretion, can precipitate DKA in such a scenario.



[1720] **Figure 1.** CT abdomen without contrast demonstrating edematous and inflammatory changes in the tail of the pancreas.

S1721

Doxycycline-Induced Pancreatitis: An Uncommon Complication of a Common Drug

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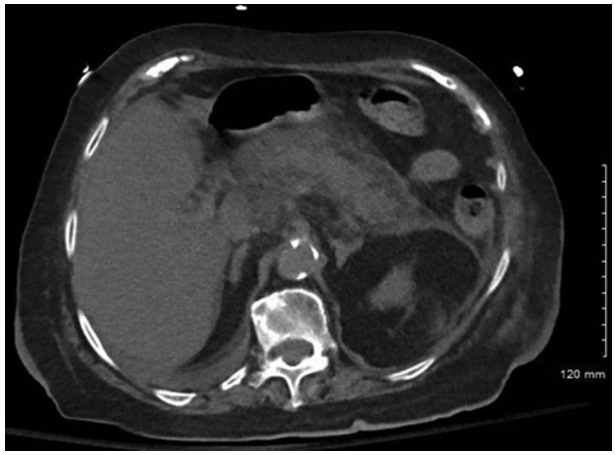
Introduction: Drug induced pancreatitis (DIP) is rare but potential cause of acute pancreatitis and accounts for 0.1-5.3% of all cases. In this case series, we report 2 cases of patients with doxycycline induced pancreatitis.
Case Description/Methods: A 60-year-old woman with cervical osteomyelitis on iv ceftriaxone and doxycycline presented with 2-day history of severe nausea, vomiting, and epigastric pain. On exam, she had epigastric and right upper quadrant tenderness. Laboratory evaluation was significant for serum lipase 6,699 u/l , creatinine of 1.94 mg/dl. Liver function tests and lipid panel were within normal limits. Computerized tomography (CT) of the abdomen and pelvis confirmed acute interstitial pancreatitis (**Figure**). Triglyceride, IgG subclasses and calcium levels were normal. Thorough review of her medications revealed doxycycline induced pancreatitis was suspected. Her symptoms gradually improved and lipase returned to 85 u/l with discontinuation of doxycycline. A 91year-old woman with recent history of left elbow fracture complicated by wound dehiscence on doxycycline therapy, presented for hospital admission with several days of increased confusion, malaise, and generalized, severe abdominal pain. She appeared ill, with dry mucous membranes and diffuse abdominal tenderness. Laboratory test results were notable for blood urea nitrogen (BUN) 56 mg/dl, creatinine 2.35 mg/dl, ALP 223 u/l, total bilirubin 1.1 mg/dl, wbc count 26.7 k/ul, calcium 6.4 mg/dl, lipase 301 u/l, AST, ALT, triglycerides and IgG subclass levels were within normal limits. CT abdomen showed extensive intrapancreatic and peripancreatic edema, the patient was diagnosed with severe pancreatitis. With careful exclusion of other etiologies, she was diagnosed with doxycycline induced pancreatitis. Her symptoms improved with aggressive hydration and discontinuation of doxycycline.
Discussion: Onset of symptoms with relation to starting the offending drug is key in identifying the causative agent of DIP. The severity, onset of symptoms, dosage of doxycycline and latency have been reported as variable as in our cases. Symptom onset was at 14 days of doxycycline therapy in the first vs 22 in the second case. Treatment includes cessation of doxycycline and aggressive IV fluid resuscitation. This case series emphasizes the importance of considering doxycycline as a cause of acute pancreatitis especially with its increased use in recent years.



[1721] **Figure 1.** CT abdomen with extensive intrapancreatic and peripancreatic edema (second case presented).

S1722

Double Trouble: A Rare Anatomical Variant of the Major Papilla

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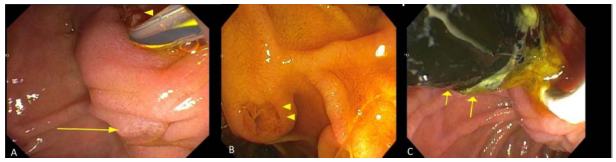
Introduction: The common bile duct (CBD) and pancreatic duct (PD) join together at the level of the ampulla before opening into the duodenum via a single orifice. Biliary tree anatomic abnormalities stem from failure of rotation or recanalization during embryo development. We present a case of choledocholithaisis explored via endoscopic retrograde cholangiopancreatography (ERCP), revealing a double major papilla.

Case Description/Methods: A 61-year-old female with a history of cholecystectomy for symptomatic cholelithiasis presented to the emergency department with 4 weeks of epigastric pain and associated nausea and vomiting. Vitals were within normal limits and her exam was notable for epigastric tenderness. Laboratory investigation revealed WBC 11.2 x 106, AST 104 IU/L, ALT 196 IU/L, total bilirubin 1.3 mg/dL, and alkaline phosphatase 433 IU/L. Abdominal ultrasound showed dilatation of the CBD to 10 mm and MRCP demonstrated a 2.9 cm filling defect in the CBD with intrahepatic and extrahepatic biliary dilation indicative of obstructing choledocholithiasis. The patient underwent ERCP which found a bulging major papilla consisting of 2 separate openings, both draining bile (Figures A,B). The proximal duct was deeply cannulated and contrast extended throughout the entire biliary tree. Removal of an obstructing, 23mm biliary stone was achieved by sphincterotomy and balloon extraction (Figure C). The patient was pain free and tolerated oral intake at discharge. At one week follow-up, liver function tests were improved.
Discussion: The major duodenal papilla is the terminal portion of the hepatopancreatic drainage system that develops from the ventral wall of the midgut during the fourth week of embryogenesis. By week 8-12 the hepatopancreatic ducts have a superior and inferior orifice; the inferior is usually later suppressed. Non-union of the CBD and duct of Wirsung can lead to a double major duodenal papilla. The cranial orifice opens to the bile duct and the caudal orifice opens to the PD. Only 10 other similar cases have been reported. In order to avoid unnecessary instrumentation or damage, cannulation of the caudal orifice was deferred in our case. This nonunion of the ducts has been shown to increase risk for choledocholithiasis. Recognition of this anatomic anomaly by endoscopists may reduce procedural complications such as post ERCP pancreatitis and failure of bile duct cannulation.



[1722] **Figure 1.** A: Two apertures of the Major Papilla: the proximal (short arrow) and distal opening to the common bile duct (long arrow) separated by 2 to 3 cm of mucosa B: Distal opening to pancreatic duct (2 short arrows) C: Stone removal.

S1723

Cystic Disguise, Cancerous Surprise: A Rare Case of Pancreatic Lymphoma

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Introduction: Pancreatic cancer is an extraordinarily aggressive and devastating form of malignancy serving as the fourth leading cause of cancer related deaths in the United States. The most common subtype is adenocarcinoma; however, other subtypes of pancreatic neoplasms exist, including primary pancreatic lymphoma (PPL). Here we present a rare case of PPL masquerading as a pancreatic cyst.
Case Description/Methods: A 49-year-old female with a history of MALT lymphoma of the right eye presented with upper epigastric abdominal pain ongoing for the past 3 months. The pain was burning in nature that would worsen with eating. Associated symptoms included intermittent fevers and unintentional weight loss of 10 pounds since the onset of symptoms. Labs were significant for abnormal liver function tests, including AST 72 U/L, ALT 116 U/L, ALP 836 U/L, total bilirubin 0.7 mg/dL. CT abdomen/pelvis revealed a 5.7 x 5.4 cm cystic mass of the pancreatic head and duodenum with nearby lymphadenopathy. Subsequent endoscopic ultrasound confirmed the cystic lesion with evidence of hemorrhagic and solid components. Pathology of FNA obtained specimen was suggestive of a pseudocyst. Given persistent pain, patient underwent exploratory laparotomy, revealing a necrotic portal lymph node with cystic degeneration adjacent to the duodenum, which was subsequently excised. A Whipple procedure was not performed. Biopsy results showed large sized abnormal lymphoid cells displaying irregular nuclear contours consistent with diffuse B-cell lymphoma. PET scan showed stage 1 disease with Ki-67 proliferation index of 90%. Patient was referred to oncology and started on R-CHOP therapy with curative intent.
Discussion: PPL is a rare malignancy, accounting for less than 0.5% of pancreatic cancer. Early diagnosis is challenging given that clinical presentations and imaging findings are nonspecific and commonly mimicking adenocarcinoma. As such, histopathological findings are crucial for differentiation. PPL responds well to chemotherapeutic regimens and portends a much more favorable prognosis with a 5-year survival rate of around 50% as compared to 11% for pancreatic adenocarcinoma. Therefore, it is important for clinicians to consider PPL as part of their differential for pancreatic lesions given the differences in management and outcomes.

S1724

Eosinophilic Cholangitis: A Great Mimic

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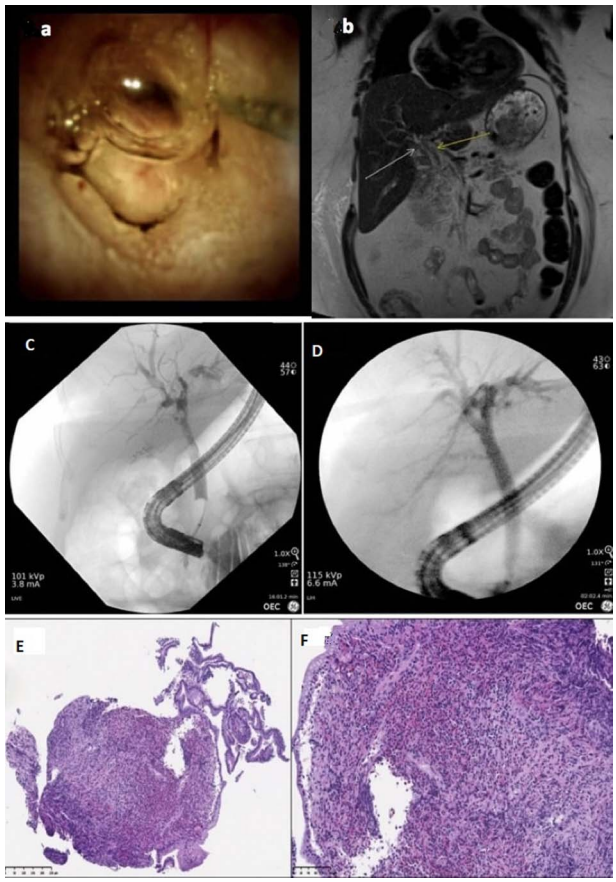
Introduction: Eosinophilic Cholangitis (EC) is a rare infiltrative disorder of the biliary tree and a diagnostic dilemma. The initial presentation mimics malignancy and is distinguished by histopathology. Steroid response supports the diagnosis. This case elucidates the diagnostic and therapeutic approach to EC in a patient with acute jaundice.
Case Description/Methods: A 44-year-old female with a history of SLE and marginal cell lymphoma in remission presented with fevers, leukocytosis, abdominal pain, and hyperbilirubinemia. She underwent an ERCP that showed a common bile duct (CBD) stricture with stent placement that was removed 6 weeks later (Figure). She then underwent a cholecystectomy for chronic cholecystitis with an intraoperative liver biopsy for persistently elevated LFTs (AST 512 U/L, ALT 500 U/L). Surgical specimens showed dense eosinophils at the cystic duct margin with mild non-specific lobular inflammation involving 15% of liver parenchyma. Six months later, she presented to our hospital with worsening abdominal pain and jaundice. Pertinent labs included a total bilirubin of 16.9 mg/dL, AST 321 U/L, ALT 335 U/L, and alkaline phosphatase 2700 U/L. MRCP showed CBD narrowing at the hilum with intrahepatic ductal dilation She underwent an ERCP which identified a Bismuth IV hilar stricture and a ductal polypoid mass on cholangioscopy. Two biliary stents were placed across the stricture into the right and left intrahepatic ducts. A direct biopsy, while negative for malignancy, revealed marked eosinophilia. Labs included peripheral eosinophilia of 0.78 x10^9/L. IgG4 and Anti-mitochondrial antibody were normal. A month later, repeat ERCP with stents exchange and ductal mass re-biopsy was performed given high concern for malignancy, however again demonstrated sheets of eosinophils. After multidisciplinary discussion, she was presumptively diagnosed with EC and started on 40mg of prednisone. Seven weeks later, repeat ERCP showed complete resolution of the stricture. LFTs and bilirubin normalized. Her symptoms resolved and she was transitioned to azathioprine for maintenance.
Discussion: EC is a rare, benign process affecting the biliary system and poses a significant diagnostic challenge. Our case supports proposed diagnostic criteria for EC, including biliary stricture, eosinophilic infiltration on histopathology, and steroid responsiveness. Our case is unique in our demonstration of the direct cholangioscopy view of this benign entity and the utility of cholangioscopy in achieving the diagnosis.



[1724] **Figure 1.** a) Ductal polypoid mass near common hepatic duct identified via endoscopic retrograde cholangiopancreatography with cholangioscopy. b) Intrahepatic biliary ductal dilatation seen on magnetic resonance cholangiopancreatography. c) Occlusion cholangiogram during endoscopic retrograde cholangiopancreatography showing a Bismuth Type IV common bile duct stricture. d) Occlusion cholangiogram during endoscopic retrograde cholangiopancreatography showing resolution of the common bile duct stricture after steroid treatment. e) Biliary stricture biopsies showing fragments of bile duct mucosa with acute and chronic inflammation with marked eosinophilia. f) Ductal mass/biliary stricture biopsy demonstrating inflammation and eosinophilia on Hematoxylin-eosin stain.

S1725

EBV-Positive Fibroblastic Reticular Cell Tumor of the Spleen With Pancreatic Invasion

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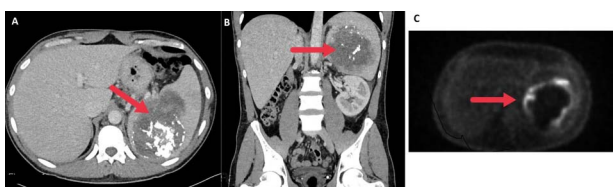
Introduction: Fibroblastic reticulum cells (FBRCs) are a subtype of dendritic cells, which are stromal cells found in structures such as the spleen and tonsils. They are accessory cells of the immune system with structural and functional roles. Other subtypes of dendritic cells include follicular dendritic cells (FDC) and interdigitating dendritic cells (IDC). Tumors arising from FDCs and IDCs are common, whereas tumors arising from FBRCs are extremely rare. To our knowledge, this is the only documented case of Epstein-Barr virus (EBV) positive FBRC tumor of splenic origin.
Case Description/Methods: A 37-year-old male presented with a 1-week history of worsening left upper quadrant abdominal pain. The pain was associated with night sweats and was worse with inspiration and palpation. He also reported increased weakness, fatigue, and loss of appetite. A contrast CT of the abdomen and pelvis revealed a large, calcified heterogeneous splenic mass with lobulated contours and ill-defined margins inferiorly, concerning for neoplastic process with metastases (Figure A, B). PET scan demonstrated 2 additional splenic lesions, one at the apical posterior aspect and another at the inferior margin (Figure C). The patient underwent splenectomy and open distal pancreatectomy as the splenic mass was found to be invading the distal pancreas. Histopathological analysis demonstrated a well circumscribed tumor within splenic parenchyma composed of spindle cells with atypical, vesicular ovoid nuclei with pale eosinophilic cytoplasm arranged in a fascicular fashion with a background of chronic inflammatory infiltrate. Immunohistochemistry analysis showed these cells were positive for SMA and negative for pan-keratin, CAM5.2, desmin, S100 protein, CD21, CD34, and CD35. The spindle cells displayed nuclear positivity for EBV-encoded RNA by in-situ hybridization. A diagnosis of EBV positive FBRC tumor was established.
Discussion: According to our review of the literature, there are only 2 documented cases of FBRC tumors arising from the spleen, neither of which was EBV positive. Therefore, our case is the first documented case of EBV positive FBRC of splenic origin. They are often mistaken as metastatic lesions and are grossly indistinguishable from other splenic malignancies. Microscopic analysis is critical for accurately diagnosing this entity. It is thought to be an indolent and benign lesion, but the malignant potential is not clear given lack of literature. Further research is needed to investigate the role of EBV in development of this disease.



[1725] **Figure 1.** A and B: CT abdomen showing a large heterogenous splenic mass with lobulated contours and irregular calcifications concerning for neoplastic process. C: PET scan showing a large heterogeneous splenic mass with rim of severe hypermetabolism consistent with a malignancy.

S1726

Eosinophilic Infiltration Causing Biliary Tree Obstruction Without Stricture

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Introduction: We present an exceedingly rare case of eosinophilic cholangitis (EC) without stricture. Of the 40 cases of EC reported in the literature, fewer than 10 cases did not involve a stricture.
Case Description/Methods: A 60-year-old female with no significant past medical history presented with symptoms of biliary colic for 2 days. Laboratory results were significant for elevated hepatobiliary enzymes including alkaline phosphatase of 300 IU/L and total bilirubin of 3.8 mg/dL. A hepatobiliary iminodiacetic acid scan was performed with an absence of radiotracer excretion into the biliary tract. However, a magnetic resonance cholangiopancreatography showed no obstructing stone, stricture, or lesion. A broad workup including hepatitis and autoimmune serologies was negative. The patient underwent an uncomplicated laparoscopic cholecystectomy, and direct inspection of the gallbladder confirmed significant edema. Histopathology of the gallbladder showed acute and chronic acalculous cholecystitis with eosinophilic rich infiltrates. Notably, the patient had an absolute eosinophil count of 1.2 cells/μL. She was diagnosed with eosinophilic cholangitis and treated with corticosteroids. At her one-month follow-up, her symptoms had resolved.
Discussion: Eosinophilic cholangitis is a rare, self-limiting condition featuring eosinophilic invasion of the biliary tree. Laboratory values in EC reflect an obstructive process, but diagnosis is frequently delayed as both symptoms and imaging mimic multiple other biliary conditions including malignancy, primary strictures, primary sclerosing cholangitis, amongst others. Endoscopic dilation of strictures often provides a surprising diagnosis when pathology demonstrates dense infiltration of eosinophils. Although a majority of EC is associated with stricture, EC may also present with obstructive features in the absence of stricture. Likewise, peripheral eosinophilia may or may not be present. Risk factors for EC have not been established. Literature suggests that patients with pre-existing eosinophilic processes such as eosinophilic esophagitis and asthma are more likely to develop EC than the general population. There is no standard treatment, but corticosteroids have demonstrated empiric efficacy, similar to the other eosinophilic processes mentioned. Increased physician awareness of eosinophilic cholangitis will help prevent delays in diagnosis and initiation of effective medical therapy.

S1727

Endoscopic Ultrasound-Guided Transgastric Drainage of an Infected Biloma After Cholecystectomy: A Case Report

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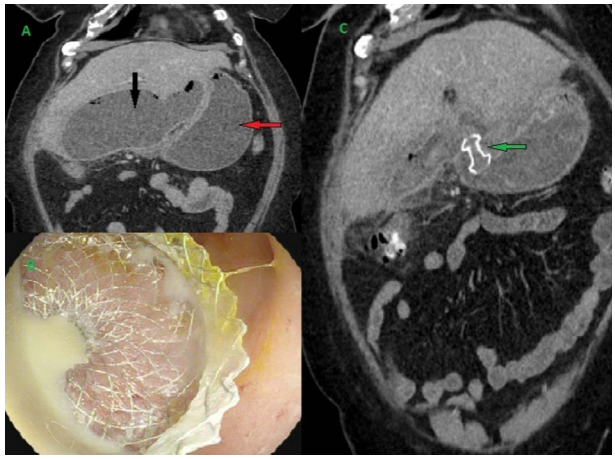
Introduction: A biloma is a well-defined collection of bile outside the biliary tree typically caused by iatrogenic disruption of a bile duct. Percutaneous drainage is the traditional treatment modality with surgery reserved for refractory cases. Endoscopic ultrasound (EUS) guided transenteric drainage has emerged as a suitable alternative. We present a case of an infected post-cholecystectomy biloma treated using EUS-guided drainage with a transgastric LAMS.
Case Description/Methods: A 57-year-old female presented with fevers, abdominal pain, nausea, and vomiting following a laparoscopic cholecystectomy for acute cholecystitis 12 days prior. Labs noted a leukocytosis with normal liver chemistries. CT scan of the abdomen showed a peripherally enhancing gas and fluid collection in the subhepatic space measuring 14.5 x 7 x 8 cm, resulting in a mass effect on the antrum and proximal duodenum. Gastroenterology was consulted for the management of suspected bile leak and infected biloma. Endoscopic retrograde cholangiography (ERC) revealed a bile leak with contrast extravasation from the cystic duct stump. This was treated with biliary sphincterotomy and placement of a 10 Fr x 7 cm plastic biliary stent. Same session EUS demonstrated a large fluid collection abutting the antrum with a well-defined wall. EUS-guided drainage of the biloma was performed using a 15 x 10 mm electrocautery enhanced LAMS via a transgastric approach. Approximately 1 L of purulent fluid was evacuated from the cavity. A subsequent CT scan confirmed the correct positioning of the LAMS with marked cavity decompression (Figure). The patient's symptoms resolved. Endoscopy with LAMS removal and placement of 2 10 Fr x 4 cm double pigtail stents (DPT) was performed 1 week later. CT 1 week after DPT placement noted near-complete resolution of the fluid collection. Six weeks after the index procedure, ERC showed resolution of the cystic duct leak and the biliary stent was removed. Both DPTs were removed 2 weeks later. The patient has remained well clinically.
Discussion: EUS-guided transenteric biloma drainage is a safe and effective alternative to percutaneous drainage. Initial cases were performed using plastic stents but more recently, transduodenal drainage with a LAMS was reported. Endoscopic drainage also enables same session identification and treatment of the culprit bile leak which is essential to prevent biloma recurrence. Our case is the first reported EUS-guided transgastric drainage of an infected biloma using a LAMS.



[1727] **Figure 1.** A. CT scan illustrating a large subhepatic fluid and gas-containing collection (Black arrow) with resultant gastric outlet obstruction (red arrow). B. Successful placement of a transgastric lumen apposing metal stent (LAMS) into the biloma cavity with immediate drainage of purulent fluid. C. Post-drainage CT scan demonstrating biloma cavity decompression and appropriate positioning of the LAMS (green arrow).

S1728

Emergent Decompressive ERCP or Not; Go With Your Gut

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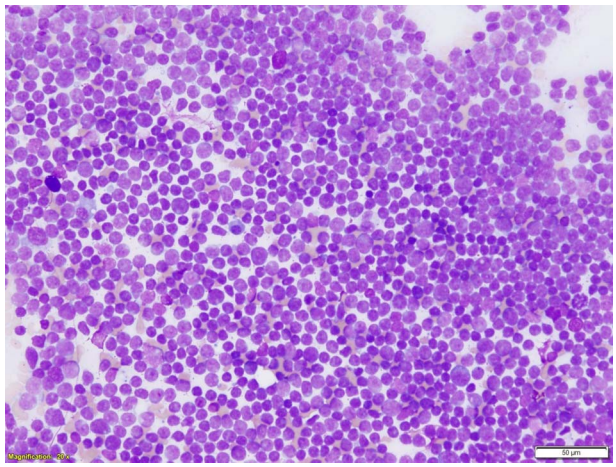
Introduction: Acute Cholangitis is a biliary emergency that requires prompt diagnosis & appropriate management in a timely manner. Guidelines and best practices dictate that if the Charcot's triad and Tokyo guidelines are indicating moderate to severe cholangitis, ERCP should be performed urgently. In certain situations however, clinical decision making is not as straight forward. We present a case in which a number of confounding factors caused hesitancy in making that decision.
Case Description/Methods: 38-year woman with PMH of Crohn's colitis on adalimumab presented with 2 weeks of progressively worsening upper abdominal pain associated with decreased appetite, nausea, vomiting, change in urine color and chills but no fevers. She completed a course of Augmentin 20 days back for a URI. She was also recovering from alcohol use disorder. She was afebrile and hemodynamically stable. On exam, had marked scleral icterus, palpable liver, spleen with upper abdominal tenderness. Lab were remarkable for lymphocytic leukocytosis, thrombocytopenia and Alk phos 316 U/L, T and DBilirubin-7/6.3 mg/dL, AST/ALT-72/131 U/L, respectively. CT abdomen pelvis with contrast showed no intra-or extrahepatic biliary ductal dilation and moderate hepatosplenomegaly. Ultrasound liver with

Doppler confirmed above. GI team was called to evaluate for possible ERCP for cholangitis. Later Peripheral smear which was initially ordered to evaluate low platelets, showed blasts cells following which flow cytometry and bone marrow biopsy confirmed B-lymphoblastic leukemia with BCR-ABL1 fusion 97% of cellularity by differential count (**Figure**). Liver enzymes normalized after initiation of R-HyperCVAD.
Discussion: Intrahepatic cholestasis is a known phenomenon in ALL, however initial presentation with intrahepatic cholestasis is rare except for few reported cases in pediatric population. Leukemic infiltration causing intrahepatic cholestasis causes an important diagnostic dilemma and establishing correct diagnosis has important implications especially since the treatment of choice, ERCP, for the alternate diagnosis is not only invasive but the risk of adverse events is high. Keeping this in mind hematologic malignancies should always be considered as a differential in patients with constitutional symptoms and hepatosplenomegaly. The diagnosis can be confirmed by immunohistochemical stains showing immature B cell lineage from a Liver biopsy; however, it is not necessary as in above case. Systemic chemotherapy is the main treatment for B-ALL/LBL.



[1728] **Figure 1.** Bone Marrow Smear

S1729

Drug-Induced Pancreatitis: Meth, Cannabis, or Meth-Laced Cannabis?,

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Introduction: Drug-induced pancreatitis (DIP) is a rare entity with a 0.1 to 2% incidence of acute pancreatitis cases. Methamphetamine is a serotonergic drug used as a stimulant. It has infrequently been observed to cause pancreatic and hepatic damage due to its keen vasoconstrictive properties. Cannabis in its recreational form has also been linked with drug-induced pancreatitis. We report a 36-year-old female who presented with findings of acute pancreatitis revealing a drug-induced etiology.
Case Description/Methods: The patient is a 36-year-old previously healthy female who presented with diffuse, sudden in onset abdominal pain, radiating to the back, aggravated with food without alleviating factors of 3 days duration. She reported nausea on the first day of her pain along with a single episode of nonbilious vomiting that had self-subsided. Social history was remarkable for smoking marijuana since her teenage years unremarkable for alcohol and tobacco use. She reported resorting to street, methamphetamine laced marijuana, the past month. The patient was vitally stable with a physical exam remarkable for epigastric and left lower quadrant tenderness with negative peritoneal signs. Laboratory tests showed elevated lipase, amylase, and CRP with normal liver function tests, hematocrit, BUN, bilirubin, triglycerides, and phosphocalcium balances. Ultrasound was done corroborating the absence of obstruction. Computed tomography (CT) abdomen was subsequently performed showing irregular contour of the pancreatic margins, blurring of peripancreatic fat planes, soft-tissue stranding, and trace-free fluid at the pancreatic body and tail without bile duct dilatation. Serological survey for mumps virus, cytomegalovirus (CMV), epstein barr virus (EBV), and human immunodeficiency virus (HIV) was negative. Anti-nuclear antibodies (ANA), neutrophil anti-cytoplasmic antibody (ANCA), and serum Ig G4 were also negative. She was diagnosed with mild acute pancreatitis with the culprit agent being methamphetamine-laced cannabis. The patient was followed up 3 months after agent cessation with marked symptomatic improvement and unremarkable lab work.
Discussion: This case highlights that methamphetamine might also be the precipitating cause of pancreatitis in street marijuana-associated pancreatitis cases. Drug-induced pancreatitis is often challenging because there are no unique clinical characteristics to distinguish drugs from other etiologies, let alone the possibility of identifying one potential drug versus another.

S1730

ERCP in a Patient With Situs Inversus Totalis

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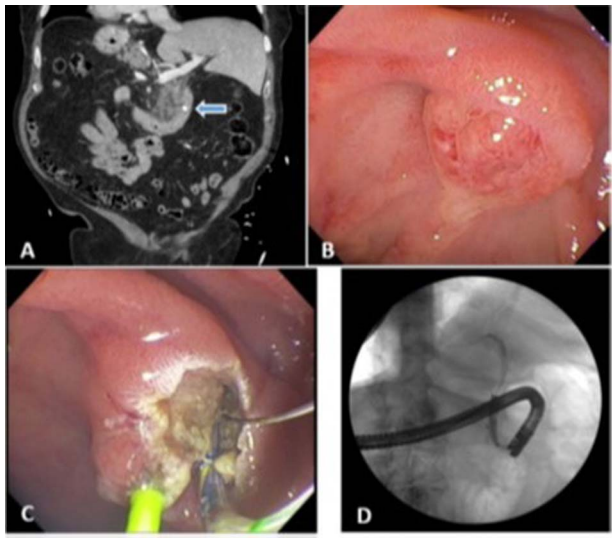
Introduction: Situs inversus (SI) is a congenital anomaly resulting in transposition of thoracic and abdominal organs. SI is often found incidentally. We present a case of choledocholithiasis in a patient with situs inversus totalis (SIT). We highlight some technical considerations in performing ERCP for these patients which may increase procedural success. In addition, we included images showing the unique orientation of bile duct to the pancreatic duct.
Case Description/Methods: An 89-year-old man presented to the emergency department with progressive epigastric abdominal pain for one month associated with nausea and non-bloody, non-bilious vomiting. In the emergency department, the patient was found to have elevated lipase 1903 U/L, total bilirubin 1.7 mg/dL, and a CT abdomen showed changes of acute pancreatitis with a 7 mm obstructing stone at the ampulla (Figure A). The CT also incidentally detected situs inversus totalis. After treatment of acute pancreatitis, patient underwent ERCP for removal of the common bile duct (CBD) stone. The patient was positioned in right semiprone position with the endoscopist positioned on the right side of the table. The side-viewing duodenoscope was advanced into the stomach, the gastric folds were followed with slight clockwise rotation of the scope until reaching the antrum. Long scope position was used to visualize and access the papilla. The major papilla was visualized in the upper right quadrant of the screen (Figure B). Deep biliary cannulation was challenging so a pancreatic duct stent was placed first to aid in biliary cannulation. Subsequently, biliary cannulation was achieved with biliary sphincterotomy and balloon sweep performed (Figure C shows the orientation of CBD to the right of the pancreatic duct). Clearance of the common bile duct was achieved (Figure D). The patient tolerated the procedure well and underwent laparoscopic cholecystectomy during the same hospitalization.
Discussion: SI is found in approximately 1 in 10,000 which can obscure the diagnosis of abdominal pathology. Often imaging is necessary to fully determine organ position and existing anatomy. In our case SIT was noted on CT along with the culprit CBD stone that caused acute pancreatitis. Performing ERCP in this setting can be very challenging. In such patients, careful planning for ERCP to minimize adverse events and maximize success is essential.



[1730] **Figure 1.** CT scan demonstrating situs inversus with pancreatitis and visible stone (A), major papilla (B), CBD to the right of the pancreatic duct (C), confirmed clearance of common bile duct (D).

S1731

Fibrosing Pancreatitis Initially Diagnosed as Ductal Adenocarcinoma by Endoscopic Ultrasound of a Pancreatic Mass in a Pediatric Patient

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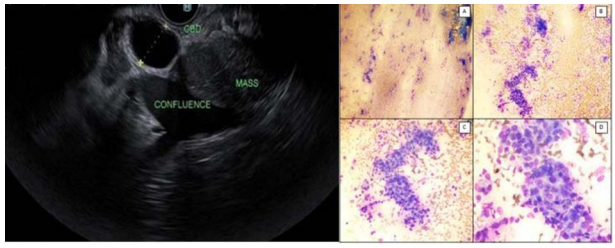
Introduction: Pancreatic cancer is very rare in the pediatric population. Incidence data is scarce, and few case reports have been published. Pancreatic ductal adenocarcinoma (PDA) is the most common subtype in adults, but is rare in children. We report a case of a benign pancreatic head mass in a pediatric patient initially diagnosed as PDA on endoscopic ultrasound (EUS) guided biopsy.
Case Description/Methods: A 13-year-old male with past medical history of treated *Helicobacter pylori* infection 6 months prior was admitted for severe acute intermittent epigastric pain. Labs showed elevated transaminases and gamma-glutamyl transferase. Abdominal ultrasound demonstrated an indeterminate solid mass-like lesion of 3 cm at the pancreatic head, and the common bile duct (CBD) was dilated to 0.9 cm. Magnetic resonance cholangiopancreatography showed a 2.2 cm indeterminate lesion along the pancreatic head region with dilated CBD of 1.4 cm, tapering at the level of the lesion. On hospital day 3, the patient underwent esophagogastroduodenoscopy and EUS. A parenchymal mass of 2.2 cm was visualized in the head of pancreas, and fine needle biopsy was performed. Cytology diagnosis was well differentiated ductal adenocarcinoma. Serum carcinoembryonic antigen, carbohydrate antigen 19-9, and alpha-fetoprotein were all normal. The patient underwent elective Whipple procedure. Surprisingly, no malignancy was identified on the surgical specimen. Instead the findings supported a diagnosis of localized fibrosing pancreatitis with high background of immunoglobulin G4 with interlobular pattern. Patient was discharged home on post-operation day 13. EUS cytology was reviewed at a tertiary cancer center and later reported as acute on chronic inflammation without malignancy (**Figure**).
Discussion: Despite increased use of EUS for diagnosis of pancreatic masses, 5-10% of patients who undergo Whipple procedure for presumed malignancy will have benign pathology: most commonly chronic fibrosing pancreatitis, chronic pancreatitis, or focal active pancreatitis. The false positive rate for diagnosing malignancy using EUS-fine needle aspiration (FNA) of solid pancreatic lesions is less than 1%. However, it may be higher in pediatrics as the prevalence of PDA is much lower in adults. For suspected cases of PDA in pediatric patients, additional review of EUS cytology at an experienced and dedicated cancer institution is advisable before further intervention to prevent the medical and psychosocial ramifications of a false positive cancer diagnosis.



[1731] **Figure 1.** (Left) A parenchymal mass (2.2 x 1.8 cm) in the head of the pancreas by endoscopic ultrasound (EUS) adjacent to the portal confluence, with the common bile duct dilated to 12.8 mm (marked by yellow cross signs). Initial cytopathology from EUS-FNA of pancreatic head mass A. Diff-Quik staining at low magnification, 4x. B. Smear is cellular and well preserved with sheets, clusters and single malignant cells, 10x. C. Cells have crowded nuclei, 20x. D. Malignant cells exhibit loss of polarity, irregular nuclear contours, finely granular chromatin and some with prominent nucleoli, 40x . Based on cytomorphological features, the mass is a well differentiated ductal adenocarcinoma of pancreatic head. Note: The review of this cytopathology by a tertiary cancer institution was inconsistent with malignancy. The glandular cells had mild cytologic atypia, and the acinar cells remained lobular in configuration. Although there was some higher nuclear to cytoplasmic ratio, no absolute nuclear enlargement was noticed.

S1732

ERCP With Per-Oral Pancreatoscopy-Guided Laser Lithotripsy for Difficult Pancreatic Duct Stones

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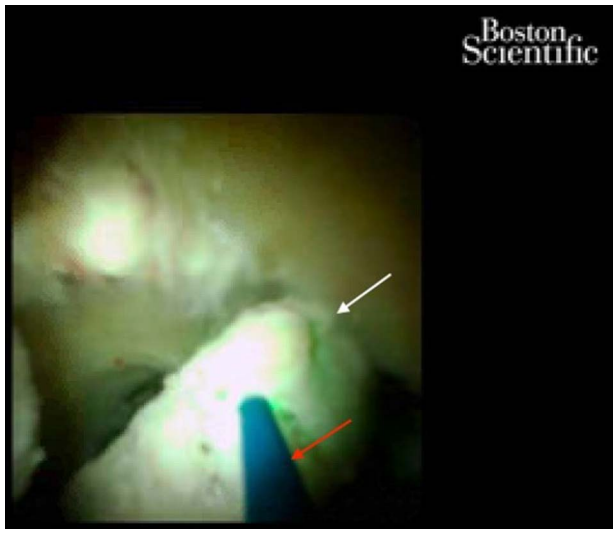
Introduction: Endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy and balloon/basket extraction is the first line management for symptomatic pancreatic duct (PD) stones. The risk of stone extraction failure via ERCP increases with PD stones that are >10mm, impacted, multiple stones or in a complicated location [1]. With difficult PD stones, fragmentation prior to extraction may be required. Stone fragmentation can be accomplished through extracorporeal shock wave lithotripsy (ESWL), per-oral pancreatoscopy (POP) laser lithotripsy (LL) and POP- electrohydraulic lithotripsy (EHL). We report a case of difficult PD stones successfully managed by POP-LL holmium laser.
Case Description/Methods: A 48-year-old man with advanced primary sclerosing cholangitis with percutaneous biliary drainage and chronic pancreatitis presented for recurrent jaundice, right upper quadrant and midepigastric abdominal pain and pruritus. ERCP revealed the ventral PD filled with numerous stones and was swept with a balloon and 1 stone was removed. Repeat ERCP was performed 2 weeks later with balloon extraction of a few small stones and debris from the PD. The ventral duct was then explored under pancreatoscopy using Spyglass, revealing multiple stones in the head and neck of the pancreas. Lithotripsy was then accomplished using a holmium laser. No immediate nor distant post-ERCP complications were encountered. A 1-week follow up revealed resolution of abdominal pain.

Discussion: The patient in our case had recurrent abdominal pain caused by pancreatic duct stones resulting in pancreatic outflow obstruction. Both attempts using ERCP with balloon sweeping were unsuccessful in complete stone extraction leading to the use of POP Spyglass with laser lithotripsy for the management of the multiple remaining stones. Direct visualization of the PD with technology like Spyglass, has shown to reduce the risk of duct injury, allows the visualization of stones that may have been missed previously, and permits confirmation of clearance of the PD (Figure). As seen in this case, POP allowed for visualization of PD stones that could not be accessed through ERCP management and used in conjunction with laser lithotripsy, the PD stones were successfully and safely fragmented. POP-LL has been shown to successfully fragment difficult to manage PD stones, as evidenced in this case and other studies, and should be considered as a useful alternative in the management of numerous PD stones and/or multiple unsuccessful ERCP attempts.



[1732] **Figure 1.** Laser lithotripsy with holmium laser being applied through the catheter (red arrow) to the PD stone (White arrow).

S1733

Exceedingly Rare Coexistence of Metamorphosis of Tubulovillous Adenoma of the Ampulla to Adenocarcinoma with Small Bowel Gastrointestinal Stromal Tumor - Case Report and Review of the Literature

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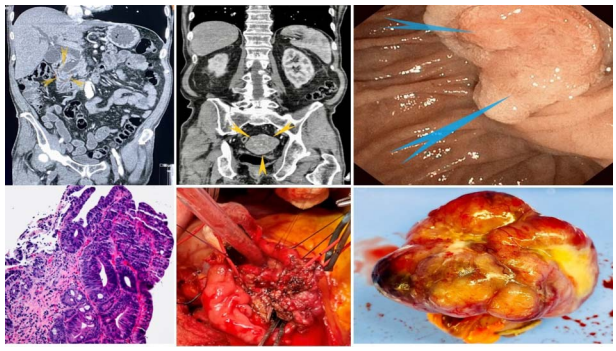
Introduction: This is a unique case of a patient with 3 tumors, which include one benign tumor, tubulo-villous adenoma of the ampulla of Vater (TVAoA), and 2 extremely rare malignant tumors, adenocarcinoma of the ampulla of Vater arising from TVAoA and gastrointestinal stromal tumor (GIST) involving the jejunum. Based on literature review, this is the first case of ampullary adenocarcinoma coexisting with GIST. Distal duodenal polyps are uncommon and have preponderance to occur in and around the ampulla of Vater. We report a case of a 77-year-old male who was admitted for painless obstructive jaundice with a 40-pound weight loss over a 2-month period, and who was subsequently diagnosed with 3 tumors.
Case Description/Methods: A 77-year-old man was admitted for generalized weakness with associated weight loss of 40 pounds in the previous 2 months and was noted to have painless obstructive jaundice. Computed tomography (CT) of the abdomen and pelvis and magnetic resonance cholangiopancreatography were consistent with a polypoid mass at the level of the common bile duct (CBD) and ampulla of Vater with CBD dilatation. The same lesions were visualized by endoscopic retrograde cholangiopancreatography. Histopathology of endoscopic forceps biopsy showed tubulo-villous AoA (TVAoA). Histopathology of the surgical specimen of the resected ampulla showed adenocarcinoma arising from the TVAoA. Abdominal and pelvic CT also showed a coexisting heterogeneously enhancing, lobulated mass in the posterior pelvis originating from the jejunum (Figure). The patient underwent resection of the mass and jejunojejunal anastomosis. The histopathology of the resected mass confirmed it as a high-grade GIST.
Discussion: AoA can occur sporadically and in a familial inheritance pattern in the setting of FAPS. We emphasize screening and surveillance colonoscopy when one encounters AoA in upper endoscopy to check for FAPS. AoA is a premalignant lesion, particularly in the setting of FAPS, that carries a high risk of metamorphism to ampullary adenocarcinoma. Final diagnosis should be based on a histopathologic study of the surgically resected ampullary specimen, and not on endoscopic forceps biopsy.This case is unique because he had 3 tumors and rare coexistence, which included one premalignant TVAoA and 2 malignant tumors: ampullary adenocarcinoma arising from the TVAoA and coexisting jejunal GIST.



[1733] **Figure 1.** Figure1A: CT abdomen and pelvis with contrast. Obstructed distal common bile duct due to a 9 mm polypoid intraluminal (pointed yellow arrows) lesion in the distal CBD. Figure1B: CT of the abdomen and pelvis with contrast. Heterogeneously enhanced lobulated mass with punctate calcifications (pointed yellow arrows) in the posterior pelvis originating from the serosal surface of the pelvic small bowel. Figure1C: Endoscopic retrograde cholangiopancreatography (ERCP) showed abnormal papilla with polypoid mass. Sphincterotomy and deep cannulation procedures were performed and confirmed by fluoroscopy. It showed common bile duct dilatation, and there was an abrupt cutoff at the distal aspect. Figure1D: High-power photomicrograph showing high-grade glandular dysplasia (hematoxylin and eosin stain, 200 X original magnification) Figure1E: A broad base ampullary mass with red fogerty cath was advanced from the cystic duct down through the ampulla to the duodenum. Figure1F: Pedunculated GIST arising from the small bowel approximately 150 cm proximal to the ileocaecal valve.

S1734

Etanercept: A Rare Cause of Drug-Induced Pancreatitis

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Introduction: Despite causing as low as 0.1% of cases of acute pancreatitis (AP), drug-induced pancreatitis(DIP) is a growing and notable cause of AP. Prior data comes from case reports and series, with definitive studies and trials lacking. To definitively diagnose DIP, a latency period with reintroduction of the medication associated with a return of symptoms is required. This is often not feasible due to risk of disastrous complications. DIP presents an uncommon cause of AP, with no established diagnostic algorithm. Amongst these cases, etanercept-induced pancreatitis presents a rare subset rarely reported in the literature.
Case Description/Methods: Our patient is a 65-year-old female with a history of rheumatoid arthritis (RA) who presented endorsing 3 days of epigastric abdominal pain. Labs indicated a normal liver function tests, calcium of 10.1mg/dl, lipase of 1012U/L, and triglycerides of 187 mg/dl. Abdominal imaging indicated hepatic steatosis, with no findings of AP nor notable biliary/gallbladder pathology. Patient was started on fluids with clinical improvement noted. Antinuclear antibody(ANA) and immunoglobulin G4(IgG4) would be negative. The patient was advised to stop etanercept therapy and follow up with gastroenterology and rheumatology outpatient. She has since been discharged and is doing well off etanercept outpatient.
Discussion: Since being first reported in the 1950s, DIP has been associated with over 500 medications per the World Health Organization. It is a diagnosis of exclusion, with common etiologies of alcohol abuse, gallstone disease, and other metabolic derangements having to be ruled out first. TNF-*a* inhibitors are involved in the treatment of a variety of autoimmune conditions. AP is not a commonly reported effect of these medications. Limited prior case reports attribute pancreatitis to infliximab and adalimumab use. To our knowledge, there are only 2 prior case reports of AP in the setting of recent etanercept initiation. After excluding more common causes of AP via imaging and labs, medication review was only notable for etanercept, which was started recently. A variety of mechanisms for DIP have been proposed. These include pancreatic/biliary duct constriction, direct cytotoxic effects, metabolic effects, and accumulation of potentially toxic metabolites. We hope to bring greater awareness to etanercept as a cause of drug-induced pancreatitis. Further studies are needed to elucidate its association with pancreatitis.

S1735

Epigastric Pain With Significant Elevation of Serum Lipase but Not Pancreatitis? A Unique Case Emphasizing Early Scanning When There Is Diagnostic Uncertainty

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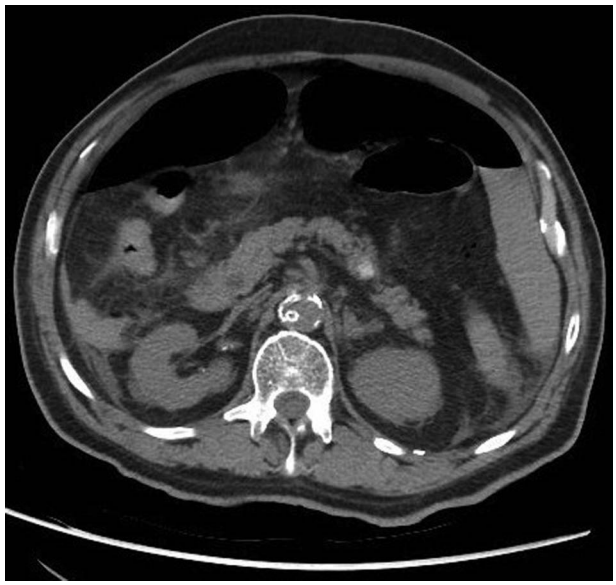
Introduction: Serum lipase has high sensitivity and specificity for the diagnosis of acute pancreatitis (AP) when it is above 3 or more times the upper limit of normal (ULN). We present a unique case of a patient who presented with typical abdominal pain and significantly elevated serum lipase and was initially diagnosed with acute pancreatitis but later found to have a perforated gastric antrum in the absence of AP.
Case Description/Methods: A 66-year-old man with alcohol use disorder presented with severe epigastric pain radiating to the back with associated non-bilious non-bloody emesis for 2 days. Physical exam was significant for mild abdominal tenderness, without guarding or rebound. His vitals were normal and lab results were unremarkable except for lipase of 444 U/L, creatinine of 1.9, and anion gap of 11. A diagnosis of AP was made and the patient was managed per guidelines. Ultrasound abdomen showed no evidence of cholelithiasis or acute cholecystitis. After 24hrs of treatment, the patient developed increasing abdominal pain and tenderness with guarding, the anion gap increased to 15 and lactic acid obtained was elevated at 4.5 mmol/L. This led the team to obtain a CT with oral contrast which showed pneumoperitoneum secondary to perforation of the gastric antrum but no evidence of AP (**Figure**). IV contrast could not be administered due to AKI at the time. The patient underwent exploratory laparotomy with a graham patch repair. Two days after the repair the lipase levels had completely normalized to a level of 40 U/L, despite worsening AKI, with a creatinine level increasing to 2.4.
Discussion: Lipase is normally produced by pancreatic acinar cells but mild elevations may be seen in a wide range of conditions. However, lipase levels 3 or more times the ULN is highly sensitive and specific for AP, and there are limited cases that describe such significant lipase elevation in the absence of AP. In our case, lipase was significantly elevated despite no evidence of AP on the CT scan, and gastric antrum perforation was confirmed on exploratory laparotomy. AKI can potentially explain the elevated lipase levels in our case, however, the lipase level normalized after the procedure despite worsening AKI. Our case report emphasizes the importance of considering alternative causes of significant lipase elevation in order to facilitate more accurate interpretation and improve patient care. Clinicians should utilize early imaging when in doubt to reduce morbidity and mortality associated with delayed diagnosis.



[1735] **Figure 1.** CT abdomen shows contrast extravasation and a large amount of pneumoperitoneum.

S1736

Gallbladder Adenocarcinoma Ascites Masquerading as Intrahepatic Portal Hypertension

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Introduction: Gallbladder cancer has 2 patterns of growth. The more common type is infiltrative growth, and the less common type is exophytic growth. Infiltrative growth can cause deep ulcerations that lead to fistula formation with adjacent structures, such as the liver. Exophytic growth displays a pathognomonic cauliflower appearance invading the gallbladder wall. Risk factors include chronic inflammation, usually secondary to gallstones or infection.
Case Description/Methods: Here we present a case of a 57-year-old male with gallbladder adenocarcinoma s/*P* laparoscopic cholecystectomy and section 4b and 5 hepatectomy, cirrhosis most likely secondary to HCV with ascites of unknown origin complicated by recurrent SBP refractory to antibiotic therapy. On initial presentation, vital signs were largely unremarkable with physical examination remarkable for

markedly distended, non-tender abdomen, with a positive fluid wave and shifting dullness. Laboratory findings revealed body fluid studies with a SAAG ratio less than 1.1 indicating malignancy, but a high hepatic venous pressure gradient pointing to portal hypertension, with multiple cytology findings negative for malignant cells, however with good synthetic liver function. CT abdomen pelvis upon presentation was unremarkable, and consistent with previous imaging findings of known hepatectomy. After repeated paracentesis and multiple courses of antibiotic therapies for SBP, with recurrent ascites, complicated by hyponatremia with no mental status changes, further management of the case involved interventional radiology placing a Denver shunt to control the ascites. After a couple of months, the patient was followed up and found to have progression of disease with cancer seeding into the peritoneum with omental caking.
Discussion: We strongly believe the patient's recurrent ascites of unknown origin was most likely a case of recurrent malignancy in ascitic fluid, masquerading as intrahepatic portal hypertension. We hope that this case report highlights that although in the setting of poorly differentiated gallbladder adenocarcinoma with local metastasis and negative margin resections, malignant ascites can occur and must not be mistaken for portal hypertension given the conflicting SAAG ratio and HPVG.

S1737

Germ Cell Tumor of Testis With Metastasis to the Common Bile Duct: A Rare Case Report

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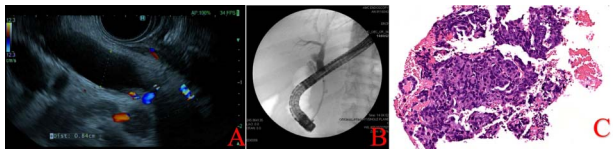
Introduction: Testicular cancer is an uncommon malignancy, but it is the most common cancer amongst males between ages 15 and 35. Germ cell tumors (GCTs) account for up to 95% of testicular cancers with retroperitoneal lymph nodes (LNs) as the most common site of metastasis. We present a case of GCT of the testis with metastasis to the retroperitoneal lymph nodes and common bile duct (CBD) causing biliary stricture and CBD dilation. To the best of our knowledge, this would be one of 2 case reports of biliary stricture caused by such metastatic cancer found in literature.
Case Description/Methods: A 35-year-old male presented to the hospital for one month of right upper quadrant abdominal pain and unintentional weight loss. Outpatient imaging showed biliary duct dilation and several areas of lymphadenopathy. Endoscopic ultrasound (EUS) and endoscopic retrograde cholangiopancreatography (ERCP) were performed and showed abnormal porta hepatic lymphadenopathy as well as stricture at the middle and lower third of the CBD. Brushings from the CBD and fine needle aspirations from the porta hepatic LNs were taken for cytology and temporary stent was placed for symptomatic relief of biliary stricture. Metastatic cancer work-up with scrotal ultrasound showed a mass in the right testes. Serum quantitative hCG, AFP, and LD were elevated (1184.9, 39.6, and 1360 respectively). Patient was diagnosed with stage III non-seminomatous testicular cancer. Both CBD brushings and LN biopsy of the porta hepatis resulted as embryonal carcinoma GCT originating from the testis (Figure). Patient's hospital course was complicated by continually rising bilirubin from the malignant intraabdominal lymphadenopathy causing extrinsic compression on the CBD resulting in multiple ERCP interventions. Moderate biliary stricture was found in the lower and middle third of the CBD which was stented. Bilirubin eventually normalized.
Discussion: This is a rare case of obstructive jaundice and biliary stricture in the setting of stage III non-seminomatous GCT that metastasized to the retroperitoneal LNs and CBD from the right testis. With EUS and ERCP, CBD stricture was found to be due to both extrinsic retroperitoneal LN enlargement and bile duct mucosa involvement. Metastatic GCT of the testis should be in the differential diagnosis amongst the young male patient population who present with obstructive jaundice and unintentional weight loss. EUS with ERCP are essential in diagnosis and treating obstructive jaundice due to metastatic GCT to CBD.



[1737] **Figure 1.** (A) Dilation of the common hepatic duct due to stricture in the CBD due to lymph nodes visualized on EUS. (B) Irregular narrowing of the mid and distal bile duct with dilated common hepatic duct and intrahepatic branches on ERCP. (C) 20x magnification of an H&E section of a lymph node biopsy at the porta hepatis showing pleomorphic tumor cells with prominent nucleoli and moderate amount of cytoplasm. Frequent mitosis is also identified. The tumor cells are positive for PLAP, CD30, SALL-4, AE1/AE3, OCT3/4 and Glypican 3 by immunohistochemistry (not shown). Morphology and immunoprofile is consistent with embryonal carcinoma.

S1738

Hemorrhagic Cholecystitis: A Rare Cause of Brisk Upper Gastrointestinal Bleeding

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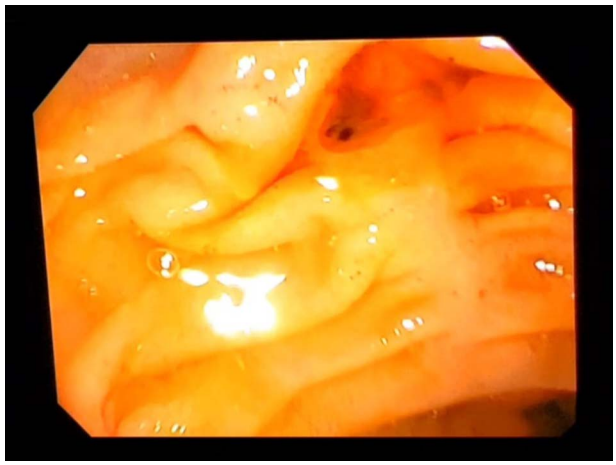
Introduction: Gastrointestinal bleeding is a commonly seen problem in the hospital and timely identification of the source is important in brisk bleeds to allow for prompt interventions. Unfortunately, the cause of the bleeding can sometimes be difficult to identify and can require thinking outside the box. We present a rare case of hemorrhagic cholecystitis manifesting as hematochezia and hypotension in the setting of chronic liver disease.
Case Description/Methods: A 55-year-old man with history of alcoholic cirrhosis (Meld-Na 24, Child-Pugh C) initially presented to an outside hospital with one week of nausea, vomiting, abdominal pain and watery diarrhea in the setting of alcohol withdrawal. He underwent an esophagogastroduodenoscopy (EGD) revealing a single gastric varix with red nipple without active bleeding and no esophageal varices. His liver function began to worsen and improved with supportive care. His improvement interestingly correlated with acute onset of hematochezia. A repeat EGD and colonoscopy revealed numerous blood clots in the colon without an active source of bleeding. He was transferred to our academic medical center for higher level of care. Upon arrival, he continued to have hematemesis and hematochezia. Repeat EGD/colonoscopy showed small non-bleeding gastric varices as well as massive maroon-colored clots throughout the colon and terminal ileum but no signs of active bleeding. CT angiography (CTA) revealed hyperdense material within the gallbladder lumen and extrahepatic biliary ductal systems suspicious for hemobilia and acute hemorrhagic cholecystitis. Pt underwent capsule endoscopy revealing an irregular oozing of blood in the proximal small intestine. A push enteroscopy then revealed actively oozing blood from the ampulla of Vater (Figure). Patient subsequently underwent successful cholecystostomy tube placement and cystic artery embolization with resolution of his bleeding.
Discussion: Hemorrhagic cholecystitis is an infrequent complication of cholecystitis with few published case reports. Due to the rarity of hemorrhagic cholecystitis causing brisk upper gastrointestinal bleeding with hematochezia, the cause of the bleeding was overlooked despite numerous endoscopies and not found until a capsule endoscopy was performed. This case teaches us to keep our differentials broad when the cause of the gastrointestinal bleeding is unclear. Prompt recognition can lead to earlier diagnosis and intervention to improve outcomes in this patient population.



[1738] **Figure 1.** Enteroscopy demonstrating bleeding at the Ampulla of Vater.

S1739

Giant Mucinous Cystic Neoplasm of the Pancreas in a 17-Year-Old: A Case Report

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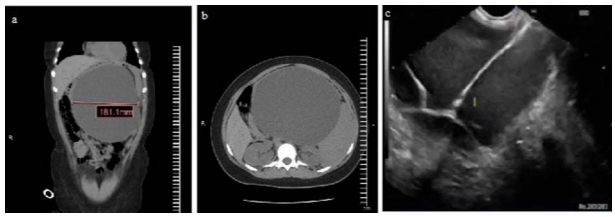
Introduction: Mucinous cystic neoplasms (MCNs) of the pancreas represent one of the most common primary pancreatic cystic neoplasms, accounting for approximately half of all cases. The probability of pancreatic cystic neoplasms being detected is raising year by year, although they are usually detected between ages 40-60, affecting women more than men. We present an unusual case of a gigantic MCN occurring in a 17-year-old patient.
Case Description/Methods: A 17-year-old female with no past medical history presented to the ER with 3-month history of progressive abdominal distention, pain and an unintentional 9lb weight loss for a month. She was hemodynamically compensated, with physical exam significant for epigastric tenderness. Hemoglobin/hematocrit was noted at 6.2 g/dL and 24%, lipase 221 U/L. CT abdomen and pelvis without contrast revealed a large cystic mass in the pancreas, with marked splenomegaly (**Figure**). MRI of the abdomen w/wo contrast confirmed the presence of a large, complex, cystic structure with septations measuring 18x17cm. EUS with FNA of the mass resulted in aspiration of 100 cc of clear, mucoid fluid. Analysis of the aspirate revealed amylase of 8 U/L, glucose 27 mg/dL, and a CA19-9 level of 25,960 IU, raising concern for mucinous cystic neoplasm. She successfully underwent open resection of the mass which measured 20cm, with distal pancreatectomy and splenectomy. Pathology of the mass revealed a mucinous cystic neoplasm with low-grade dysplasia. The patient recovered and was discharged home. She reported significant improvement at a follow up in clinic 2 months later.
Discussion: Giant MCN of the pancreas is described in the literature but has not been observed at such a young age. The incidence of detection of pancreatic cystic lesions increases year by year and is thought to be due to better imaging modalities detecting incidental lesions. To our knowledge, this is the youngest reported patient with symptomatic MCN.



[1739] **Figure 1.** a) coronal view on CT scan of the mass, b) axial view on CT scan of the mass, c) EUS demonstrating multiple septations of the giant mass.

S1740

Hematochezia Due to Cholecystocolonic Fistula and Gallstone in the Transverse Colon

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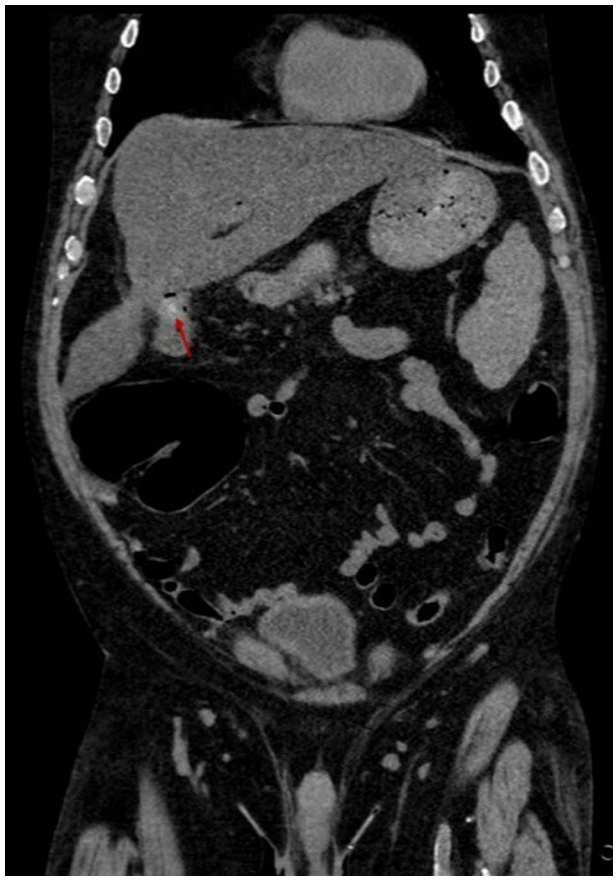
Introduction: We present this case, because cholecystocolonic fistulas are rare, accounting for less than 30% of all cholecystoenteric fistulas. This case emphasizes the unique finding of a rare fistula and the importance of sound clinical judgement for timely diagnosis and follow-up, despite the atypical presentation of the patient.
Case Description/Methods: The patient is a 65-year-old man, with a history of hypertension, hereditary spherocytosis, alcohol-related cirrhosis (MELD-Na of 20) with prior non-bleeding esophageal varices, who presented with hematochezia. He reported 3 episodes of gross bloody stools for one day, and denied hematemesis and abdominal pain. Seven years prior to this admission, he had a history of choledocholithiasis and presented with painless jaundice. At that time, he underwent an endoscopic retrograde cholangiopancreatography with sphincterotomy and biliary stent placement. On admission, he was vitally stable. His labs were the following: Hgb 8.3 g/dL (baseline of 12.7 g/dL a year prior to presentation), BUN 6 mg/dL, Cr 0.7 mg/dL, INR 1.4, ALP 108 IU/L, ALT 19 IU/L, AST 41 IU/L, total bilirubin 3.3 mg/dL, and direct bilirubin 1.7 mg/dL. Due to concern for bleeding, both an esophagoduodenoscopy (EGD) and colonoscopy were obtained. The EGD showed portal hypertensive gastropathy without significant source of bleeding. The colonoscopy revealed a 2 cm gallstone surrounded by friable mucosa at the proximal transverse colon. Further work-up with a CT abdomen showed a fistula connecting the gallbladder and transverse colon, presence of a stone in the gallbladder, and pneumobilia, with no intrahepatic or extrahepatic bile duct dilation (Figure). The gallstone lead to mucosal irritation and hematochezia. With the image confirmed cholecystocolonic fistula as the cause of the gallstone in the transverse colon, general surgery recommended conservative management given the resolution of the patient's hematochezia and his poor surgical candidacy. On follow-up, patient had no further bleeding.
Discussion: Cholecystocolonic fistulas are typically a rare complication of cholelithiasis with cholecystitis, are commonly reported in the sigmoid colon, and commonly lead to gallstone ileus and bleeding. Although these fistulas are typically challenging to diagnose given non-specific symptoms and although our patient presented with a fistula in the transverse colon and no cholecystitis, sound correlation of reported history with labs, procedures, and imaging lead to timely diagnosis and appropriate follow-up.



[1740] **Figure 1.** The CT abdomen and pelvis shows the area of the cholecystocolonic fistula with gallstone in the gallbladder (both marked with red arrow), and pneumobilia.

S1741

Gelatinous Ascites and a Double Duct Sign: A Case of Pancreatic Adenocarcinoma

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Introduction: Pseudomyxoma peritonei (PMP) is a rare disorder characterized by the accumulation of gelatinous material within the abdomen and pelvis. While usually appendiceal in origin, it can be seen with mucinous tumors arising from the ovary, GI tract, urachus, and pancreas. We describe a case of pancreatic adenocarcinoma diagnosed due to the clinical syndrome precipitated by pseudomyxoma peritonei.
Case Description/Methods: A 59-year-old Hispanic male presented to the hospital for evaluation due to a 2 month history of poor oral intake and worsening abdominal fullness, distension, discomfort, and intermittent pain. The patient reported one prior hospitalization for pancreatitis of unclear etiology, 3 years ago. A CT abdomen & pelvis was obtained; findings reported include multiple hepatic subcapsular cystic liver lesions, abdominal omental caking, ascites, and extrahepatic biliary and pancreatic ductal dilatation without definitive evidence of a mass. CEA and CA 19-9 were found to be elevated at 49.4 and 416 respectively. CT chest revealed a left-sided pleural effusion. Upper endoscopy and colonoscopy were negative. Paracentesis yielded gelatinous ascites. Clinical suspicion for appendiceal malignancy was raised which required surgical exploration and biopsy to confirm. An MRI of the abdomen was obtained to assess for pancreatic mass given presence of double duct sign. MRI revealed 2.2 x 2.7 x 5 cm lesion in the body of the pancreas, pseudomyxoma peritonei with scalloping of the liver and extension into the right posterior mediastinum via the esophageal hiatus, metastatic deposits in the porta hepatis and peritoneal carcinomatosis. No apparent abnormality in the appendix was observed. EUS FNA was performed; splenic vessel invasion by the mass was noted. Cytopathology supported a diagnosis of pancreatic adenocarcinoma. The patient was initiated on palliative chemotherapy with gemcitabine and nab-paclitaxel which led to a significant reduction in ascites and pleural effusion and improvement in symptoms.
Discussion: This case highlights the potential of malignancies such as pancreatic adenocarcinoma to cause pseudomyxoma peritonei despite it more commonly being associated with appendiceal origin. Knowledge of this can help guide diagnostic and therapeutic plans.

S1742

Gastric Outlet Obstruction Caused by Groove Pancreatitis

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Introduction: Paraduodenal or groove pancreatitis is a rare form of chronic pancreatitis. Anatomically it involves the area between the pancreatic head, duodenal wall, and common bile duct, also known as the pancreatic-duodenal groove. Symptoms include abdominal pain, nausea, vomiting, and weight loss. We present a case of a middle-aged man diagnosed with groove pancreatitis complicated by gastric outlet obstruction (GOO).
Case Description/Methods: 62-year-old male with PMH of HTN, HLD, peripheral vascular disease, COPD, chronic alcohol and tobacco use disorder presented with 4-week history of burning epigastric pain, nausea, and vomiting. In addition, he had a 20-pound weight loss over the past few months and a history of recurrent alcoholic pancreatitis. Lipase on admission was 695. CT abdomen/pelvis showed a dilated stomach consistent with GOO to the level of the pyloric channel, cystic area along the first portion of the duodenal wall and mild fat stranding around pancreatic head. Initial differential included perforation secondary to peptic ulcer disease vs malignancy as the underlying cause of obstruction. GI was consulted given concern for duodenal malignancy, but further review of imaging correlated with groove pancreatitis resulting in GOO. Patient was managed conservatively and obstruction was treated with a PEG-J tube.
Discussion: Groove pancreatitis was first described in 1970s as an uncommon form of chronic pancreatitis localized to the pancreaticoduodenal groove. It is commonly seen in middle aged men and is strongly associated with alcohol and tobacco use. The underlying etiology is not well understood but is thought to involve anatomical or structural obstruction. One theory suggests alcohol and tobacco increases the viscosity of the pancreatic juice leading to impaired outflow. CT with contrast and MRI is used primarily for diagnosis. Characteristic imaging findings include cystic lesions in the duodenal wall, dilation of Santorini's duct, or hyperplasia of Brunner's gland. Treatment involves conservative management with pain control and nutritional support. Pancreatoduodenectomy is an option if symptoms do not resolve.

Complications such as GOO can be treated initially with decompression via nasogastric tube followed by PEG-J placement for venting and post pancreatic enteral feeds. Although groove pancreatitis is rare, timely diagnosis based on imaging is essential to ensure appropriate management and to avoid unnecessary workup of other, more serious causes of GOO such as malignancy.

S1743

Hemosuccus Pancreaticus or Just a Bunch of Hocus Pocus

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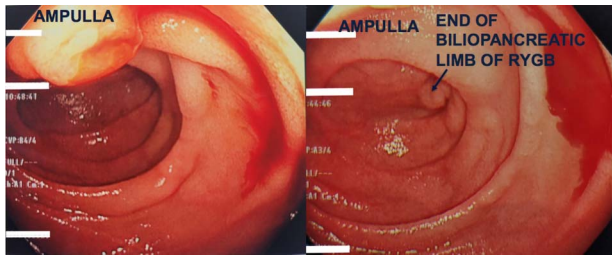
[1]Largo Medical Center - HCA Healthcare/USF Morsani College of Medicine, Largo, FL; [2]Largo Medical Center, Largo, FL.

Introduction: Hemosuccus pancreaticus (HP) is a rare phenomenon defined as bleeding from the pancreatic duct into the gastrointestinal tract via the ampulla of Vater. Many potential causes for HP exist, including pancreatic inflammation, arterial aneurysms, and bariatric surgery. A high degree of clinical suspicion and a multidisciplinary approach is key for early diagnosis and treatment.
Case Description/Methods: 50-year-old female with a history of RYGB, cholecystectomy, pancreatitis, and iron deficiency anemia, was admitted to the intensive care unit after presenting with maroon stools, abdominal pain, and fatigue with a hemoglobin of 4.8. Within the prior 5 months, she had multiple admissions for abdominal pain and GIB and was diagnosed with alcohol-induced pancreatitis, diverticular bleed, anastomotic erosions, peptic ulcer disease, and a Dieulafoy lesion on numerous EGD/colonoscopies. EGD/enteroscopy performed during this admission revealed old blood in the stomach, without active or old blood in the roux or biliopancreatic limbs. Her colonoscopy showed dark tarry stool throughout. Over the subsequent few days, she required a total of 11 units of packed RBCs. Multiple imaging modalities including computed tomography angiography (CTA) and Meckel's diverticulum scan were negative. She experienced worsening abdominal pain, hematochezia and hematemesis, and required emergent intubation. EGD/enteroscopy was performed and revealed fresh blood at the jejuno-jejunal anastomosis, the roux and biliopancreatic limbs, and active bleeding at the ampulla, consistent with HP (Figure). Interventional Radiology was contacted immediately, and repeat angiography revealed a 6mm splenic artery pseudoaneurysm. A covered stent was placed, and our patient's hemoglobin remained stable during the rest of her hospitalization, without further episodes of bleeding or abdominal pain.
Discussion: HP is an obscure, and potentially life-threatening cause of GIB that is important to include in the differential diagnosis. The classic triad includes sporadic bleeding (upper and lower GIB), intermittent abdominal pain, and hyperamylasemia. There appears to be a significant association between HP and pancreatic inflammation, which our patient had. Additionally, our patient's RYGB anatomy made the ampulla more challenging to reach, delaying the diagnosis. In addition to IR and surgery, advanced endoscopy has presented novel therapeutic options with endoscopic ultrasound, lumen apposing metal stents and fibrin glue/histoacryl adhesives.



[1743] **Figure 1.** Enteroscopy imaging showing evidence of brisk bleeding from the ampulla of Vater, consistent with hemosuccus pancreaticus.

S1744

Idiopathic Non-Caseating Pancreatic Granuloma Mimicking Neuroendocrine Tumor: A Challenging Diagnostic Dilemma

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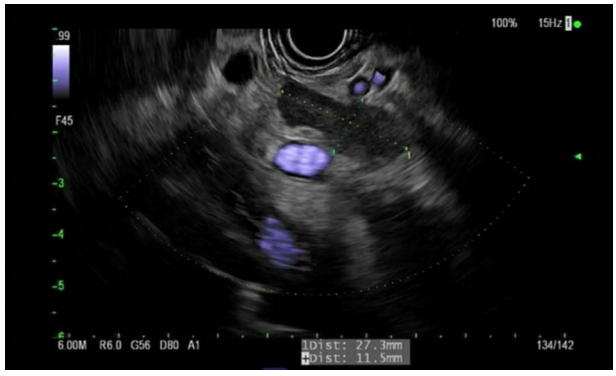
Introduction: Pancreatic granuloma is rare. Caseating granuloma is usually caused by tuberculosis, while non-caseating granuloma is mainly secondary to systemic diseases such as sarcoidosis, rheumatoid arthritis, fungal infection, and Crohn's disease. Idiopathic etiology can present a diagnostic challenge, requiring further investigation to differentiate it from malignancy. We present an interesting case of a patient who presented with a concern for a neuroendocrine tumor before diagnosing idiopathic non-caseating pancreatic granuloma.
Case Description/Methods: A 75-year-old female presented with left upper quadrant pain, chronic diarrhea, and palpitations. The physical exam was unremarkable. Workup was significant for elevated serum chromogranin 3,123 ng/ml, with mildly elevated urine metanephrines. Magnetic resonance imaging showed a stable 2.5 cm cystic lesion in the tail of the pancreas communicating with the main pancreatic duct and hypointense area in the tail. 68Ga DOTATATE scan was negative for neuroendocrine tumor. Endoscopic ultrasound showed a multi-cystic lesion in the tail of the pancreas with thick septations consistent with branch-duct intrapapillary mucinous neoplasm (BD-IPMN), and a 2.7 cm homogenous well-defined hypoechoic area in the tail of pancreas along with multiple enlarged peripancreatic lymph nodes (**Figure**). Fine needle biopsy showed multiple non-caseating granulomas with benign pancreatic and lymphoid tissue. There was no evidence of malignancy, and stains for acid-fast bacilli, FITE stain, and fungal cultures were negative. A benign idiopathic etiology was concluded. At the 6-months follow-up, the patient was doing well.
Discussion: Non-caseating pancreatic granuloma has been described in a few case reports. Sarcoidosis was the most common cause, followed by rheumatoid arthritis and Crohn's disease. Other cases were associated with granulomatosis with polyangiitis (formerly Wegener's granulomatosis), xanthogranulomatous disease, insulin-dependent diabetes mellitus, or foreign body (Talc). Patients may present with epigastric pain, sometimes associated with weight loss, jaundice, fever, or nausea. Further workup and sampling, or even surgery, might be necessary before reaching a solid diagnosis and ruling out malignancy. Therefore, it is essential to rule out other etiologies before confirming the diagnosis of idiopathic pancreatic non-caseating granuloma.



[1744] **Figure 1.** Endoscopic ultrasound (EUS) showing a homogenous & well-defined hypoechoic mass in the tail of the pancreas, measuring 11 x 27 mm.

S1745

IgG4-Seronegative Autoimmune Cholangiopathy With Pancreatic and Hepatic Involvement Mimicking as Primary Sclerosing Cholangitis

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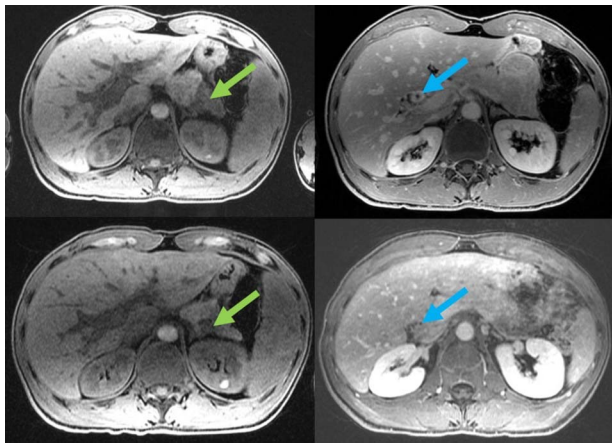
[1]Stanford University, Redwood City, CA; [2]Stanford University School of Medicine, Palo Alto, CA; [3]Stanford, Palo Alto, CA; [4]Stanford University University School of Medicine, Redwood City, CA.

Introduction: IgG4-seronegative autoimmune cholangiopathy is a rare cause of biliary strictures.
Case Description/Methods: 27-year-old male presented in 2011 with 4 months of abdominal bloating and cholestatic elevation of liver enzymes. MRI showed attenuated intra/extrahepatic ducts with beading, consistent with PSC. He was lost to follow up until 2017, when he presented with fever concerning for cholangitis. All autoimmune serologies were negative (ANA, SMA, AMA, pANCA, ASLA, LKM), and Total IgG and IgG4 were normal. ERCP showed a diffusely diseased biliary system characterized by thin, irregular, beaded ducts without dominant stricture, consistent with PSC. A liver biopsy was negative for features of autoimmune hepatitis and IgG4-related disease with negative IgG4 staining. He subsequently had multiple hospitalizations for cholangitis with bacteremia. MRCP at that time showed new focal stricture/intrahepatic biliary dilatation, and ERCP showed a common hepatic duct stricture extending into the left main hepatic duct that required stenting with multiple exchanges. Due to recurrent bouts of cholangitis with bacteremia, he was listed for liver transplantation for PSC in 2020 with intent to undergo live donor transplant. In 2021, MRCP showed a new T2 moderately hyperintense, T1 hypointense appearance of the distal 4 cm of the pancreatic tail with early hypoenhancement and delayed hyperenhancement suggestive of inflammation and fibrosis with marked periductal enhancement of the common hepatic duct with stenosis definitive for sclerosing cholangitis. Endoscopic ultrasound with fine needle biopsy failed to obtain sufficient tissue sample. These diagnostic studies suggested IgG4-seronegative autoimmune cholangiopathy with pancreatic involvement rather than PSC. Three weeks after initiation of prednisone, his liver tests showed substantial improvement in alkaline phosphatase and ALT, MRI showed improvement of biliary wall thickening, and follow-up ERCP showed improvement in biliary strictures (Figure); all stents were removed. He is no longer pursing live donor transplantation and he remains stable on azathioprine and low dose prednisone.
Discussion: IgG4-seronegative autoimmune cholangiopathy is a treatable though rare cause of biliary strictures that should be considered in those who present with morphologic changes of PSC. The diagnosis was considered when MRI revealed pancreatic inflammatory changes in addition to the biliary strictures.



[1745] **Figure 1.** Left, top: Axial T1 weighted pre-contrast fat-suppressed image reveals sharply demarcated abnormal hypointense T1 signal in the pancreatic tail (green arrow). Right, top: Delayed post-contrast T1 weighted fat-suppressed image demonstrates marked peribiliary thickening and enhancement around the common hepatic duct (blue arrow). Left, bottom: Axial T1 weighted pre-contrast fat-suppressed image obtained 6 months after initiation of steroids shows normalization of T1 signal in the pancreatic tail (green arrow). Right, bottom: Delayed post-contrast T1 weighted fat-suppressed image obtained 6 months after initiation of steroids demonstrates resolution of peribiliary thickening and enhancement around the common hepatic duct (blue arrow).

S1746

Hemosuccus Pancreaticus: More Than at First Blush

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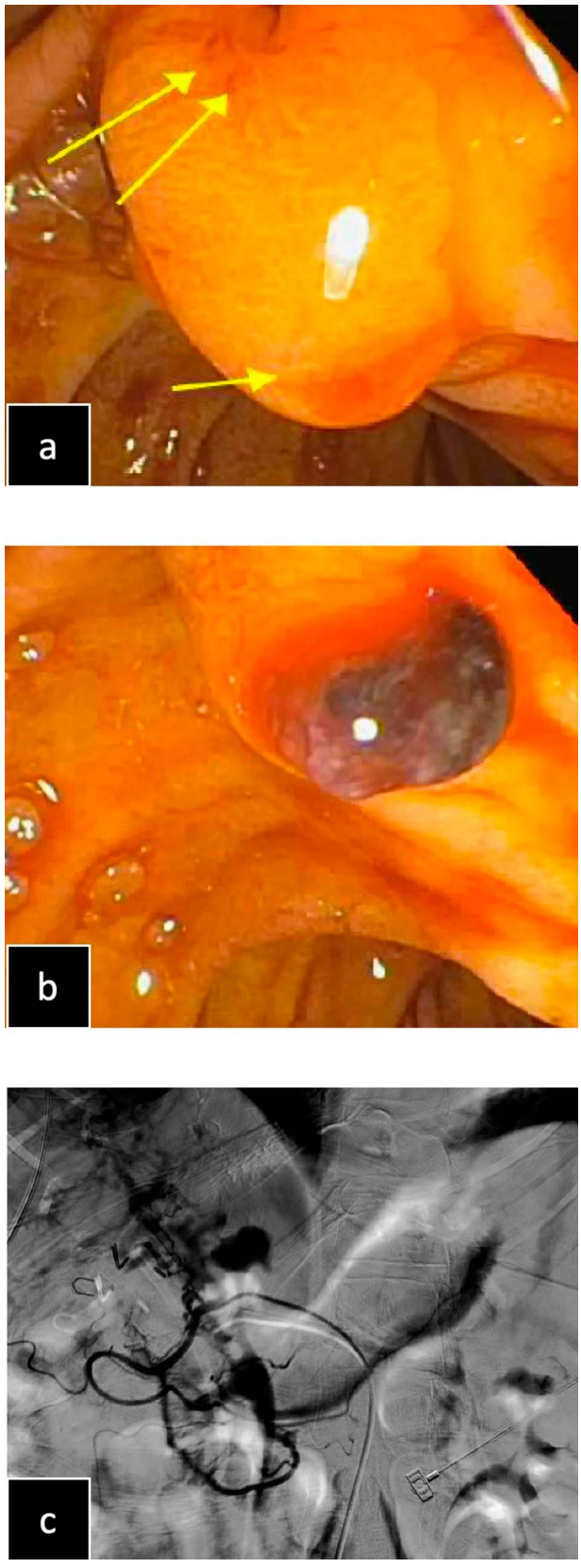
Introduction: Hemosuccus pancreaticus describes hemorrhage from the ampulla of Vater via the pancreatic duct, and is an infrequent but potentially life-threatening cause of upper gastrointestinal (GI) bleeding. Diagnosis can be elusive and treatment challenging. Here we report a case of hemosuccus pancreaticus in the setting of acute pancreatitis identified on upper endoscopy (EGD) and endoscopic retrograde cholangiopancreatography (ERCP), treated successfully with gastroduodenal artery (GDA) embolization by interventional radiology.
Case Description/Methods: A 52-year-old female with compensated alcoholic cirrhosis and cholecystitis status post cholecystectomy presented with epigastric pain, coffee ground emesis, and bright red blood per rectum. Vitals were stable, hemoglobin 5.9 g/dL, lipase 1570 IU/L, and total bilirubin 10.4 mg/dL. Computed tomography (CT) showed acute pancreatitis with peripancreatic inflammatory changes. After initial resuscitation, EGD revealed active bleeding at the ampulla but no blood in the stomach. Immediate ERCP confirmed hemosuccus pancreaticus (Figure A, B) and an increased rate of hemorrhage, with bright red blood now pooled in the stomach and duodenum. Patient underwent transcatheter angiography with empiric coil embolization of the GDA, achieving hemostasis (Figure C).
Discussion: Hemosuccus pancreaticus accounts for less than 1% of upper GI bleeds. As clots form and dissolve in the pancreatic duct, subsequent elevations in intraductal pressure lead to waxing and waning abdominal pain, with hemorrhage in the form of melena, hematemesis, or hematochezia. Primary diagnosis relies on direct visualization of the bleed. EGD, an imperative part of the initial workup, rarely reveals active bleeding from the ampulla and is only diagnostic in 30% of cases. More sensitive diagnostic tests include abdominal CT angiography, magnetic resonance cholangiopancreatography (MRCP), or catheter-based mesenteric angiography. Alternately, both diagnosis and treatment can be accomplished via ERCP, with the side-viewing duodenoscope allowing for a full assessment for pathology of the ampulla, bile duct, and pancreatic duct. Both its intermittent symptoms and anatomic location present significant diagnostic challenges and require early consideration of hemosuccus pancreaticus in the evaluation of obscure GI bleeds. Though it can be evasive, early diagnosis is imperative given the often rapid progression of these bleeds, as displayed in our patient above, and the up to 90% mortality in untreated cases.



[1746] **Figure 1.** (a) Biliary orifice (double arrow), pancreatic orifice (single arrow) with active bleeding, seen on ERCP. (b) Blood clot at the ampulla, seen on ERCP. (c) IR transcatheter embolization of the omental branch of the GDA.

S1747

Iatrogenic Cushing's From Celiac Plexus Blocks for Chronic Pancreatitis

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Introduction: Celiac plexus blocks (CPB) and celiac plexus neurolysis (CPN) have been implemented to decrease opiate dependency and treat chronic pancreatitis and/or pain resulting from pancreatic malignancy. There are various approaches to facilitate CBP/CPN including percutaneous, surgical and endoscopic, guided as CT, fluoroscopy, US or EUS techniques. EUS is the latest development in CPB/CPN and the least commonly utilized method, however recent studies have shown high efficacy and minimal complications or risks. Despite various complications associated with different techniques, no case report or current literature has documented the development of Iatrogenic Cushing's disease from use of steroids during CPB via any approach. Herein we report the first case of Iatrogenic Cushing's disease from CPB in the treatment of chronic pancreatitis.
Case Description/Methods: A 27 YO F presented to the ED with severe epigastric pain associated with nausea. She was diagnosed with chronic pancreatitis a year and a half prior. At that time, she underwent an EUS showing severe chronic pancreatitis with biopsies positive for benign pancreatic tissue, fibrotic stromal tissue and scattered eosinophilic infiltrates. Due to recurring pain a subsequent nerve block was repeated. She was prescribed hydromorphone 4 mg q3h as needed, methadone 30 mg BID and Pancrealipase, however a celiac plexus block was repeated due to refractory pain. Upon follow up, she endorsed a 10 lb weight gain, fatigue, acne, facial hair and fullness, dark abdominal striae and insomnia. She was suspected to have iatrogenic cushingoid features due to multiple celiac nerve blocks. A morning cortisol level was 0.3 ug/dL but a salivary cortisol level with within normal limits (< 0.010 ug/dL). She was referred to Endocrinology, who recommended withholding steroids for 3 to 6 months. Repeat testing with ACTH and DEXA suppression confirmed adrenal insufficiency. Five months later, the patient's pain returned, and her methadone was increased. Repeat EGD/EUS showed pancreatic parenchymal abnormalities consisting of hyperechoic strands, hyperechoic foci and lobularity throughout the entire pancreas. Due to refractory pain, she was referred for a pancreatic transplant program.
Discussion: EUS-guided CPB is an effective, minimally invasive modality in treating pain from abdominal malignancy and/or pancreatitis, however future studies are warranted to evaluate the agents used for chemical destruction, to avoid complications such as Iatrogenic Cushing's disease.

S1748

Hepatobiliary Ascaris: A Curious Wanderer

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Introduction: *Ascaris lumbricoides* is a nematode. Hepatobiliary and pancreatic ascariasis (HPA) encompasses a wide spectrum of clinical diseases involving the helminth, *Ascaris lumbricoides*, ranging from biliary colic to acute cholangitis to acute pancreatitis.
Case Description/Methods: A 53-year-old male with past medical history of cholelithiasis status post cholecystectomy presented to the emergency department (ED) with intermittent severe postprandial epigastric pain associated with nausea and one episode of non-bloody, non-bilious emesis. He was a long-time resident of the Northeastern US with no history of travel. Initial vital signs were normal and physical examination revealed mild epigastric tenderness. He had an alanine transaminase (ALT) 146 U/L, aspartate transaminase (AST) 170 U/L, lipase 65 U/L, total bilirubin 0.8 mg/dL, and alkaline phosphatase (ALP) 89 U/L. CT scan of the abdomen showed no acute intra-abdominal or pelvic process. Subsequently was discharged home from the ED. 8 days later, he returned complaining of sharp epigastric pain. Examination revealed anicteric sclera and right upper quadrant tenderness. Bloodwork now showed ALT of 638 U/L, AST of 500 U/L, lipase of 64 U/L, total bilirubin 1.1 mg/dL, and ALP 126 U/L. White blood cell count was 7,100/L. An abdominal ultrasound was normal. An MRI abdomen revealed multiple filling defects within the common bile duct. He was admitted due to concerns for choledocholithiasis. Repeat blood work showed ALT 1,040 U/L, AST 446 U/L, lipase 72 U/L, total bilirubin 1.4 mg/dL, and ALP 158 U/L. He had a fever of 100.4 F. WBC count was 5,700/L with 7.2% eosinophils. He received a dose of ceftriaxone and metronidazole and underwent ERCP. Fluoroscopy revealed filling defects in the common bile duct. A large worm originating in the biliary tree was seen emerging from the major papilla. The worm was removed with forceps and was 10 cm in length. Pathology revealed it to be Ascaris lumbricoides. He received a one-time dose of albendazole 400 mg. He was asymptomatic without any recurrent symptoms, was able to tolerate a diet and was discharged.
Discussion: *A. lumbricoides* is the most common human parasitic infection in the world, although it is not common in North America where it is considered non-endemic. Therefore, HPA is a relatively rare cause of biliary colic in the US. There are about 4 million people infected in US, most of who are immigrants. Our patient does not fit this demographic, as he is a native of the northeastern US.

S1749

Hypertriglyceridemia-Induced Pancreatitis in the Setting of Acute COVID-19 Infection

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Introduction: Profound hypertriglyceridemia has been documented in cases of acute and prior COVID-19 infections. A common cause of acute pancreatitis is hypertriglyceridemia. In some cases, this can require intensive care unit (ICU) admission for management.
Case Description/Methods: The patient is a 49-year-old male with a history of recent COVID-19 infection who initially presented for epigastric pain with intractable nausea and vomiting. He was found to have acute pancreatitis on imaging and lab work. Incidentally, he was found to be COVID-19 positive. He had no respiratory or infectious symptoms from this infection. The patient was initially admitted to ICU for his triglyceride level reading greater than 5680. He was managed with an insulin drip for resolution of his hypertriglyceridemia. The patient required an extended ICU stay as his triglyceride level remained difficult to decrease while on the insulin drip. Eventually, he was able to be downgraded to the hospital floor after a 6-day ICU stay requiring an insulin drip to decrease his profound hypertriglyceridemia.
Discussion: Interestingly, the patient's refractory hypertriglyceridemia remained difficult to treat. It has been previously documented that COVID-19 infections can lead to hypertriglyceridemia. This can lead to an increase of acute pancreatitis admissions secondary to COVID-19 infections. In conclusion, more research is warranted into the association between hypertriglyceride-induced pancreatitis and COVID-19 infection.

S1750

Intraductal Papillary Neoplasm of the Bile Duct Diagnosed Post Liver Transplant in Patient With Recurrent Hepatocellular Carcinoma

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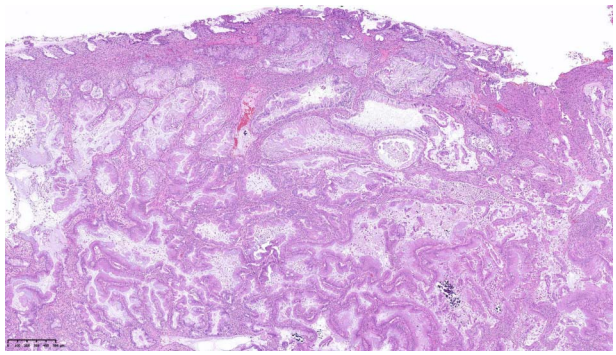
Introduction: This case describes a patient diagnosed with metastatic intraductal papillary neoplasm of the bile duct (IPNB) during his post liver transplant course. IPNB is a rare biliary tumor characterized by papillary proliferation in the bile duct lumen. IPNBs have been mainly described in countries such as Japan, China, Korea, and Taiwan, with only 11% of cases coming from the United States. Though more favorable in prognosis than infiltrating bile duct cancer, this case demonstrates the challenge in diagnosing IPNB and its metastatic potential.
Case Description/Methods: A 67-year-old Middle Eastern male with recurrent hepatocellular carcinoma (HCC) and decompensated hepatitis C cirrhosis was referred for liver transplant evaluation. Prior HCC treatments included hepatic resection and transarterial chemoembolization and radioembolization (TACE, TARE) which were complicated by the development of biliary strictures requiring percutaneous drainage. His course was further complicated by multiple episodes of cholangitis with drug-resistant organisms. At that time, endoscopic retrograde cholangiopancreatography revealed biliary strictures that were negative for dysplasia or malignancy. The patient ultimately underwent orthotropic liver transplant with Roux-en-Y biliary reconstruction, with intraoperative findings notable for chronic inflammation and intra-abdominal abscesses. Pathology from his native bile duct and liver revealed IPNB with mucin production, not detected on prior specimens from cholangiography (Figure). Three months later, he underwent Whipple procedure which revealed widely metastatic IPNB. Chemotherapy with gemcitabine and cisplatin was initiated.
Discussion: Though rare, IPNB has been increasingly reported in medical literature and is now recognized as its own entity with 4 histological subtypes. Excessive mucin production and friability of the tumor are thought to cause intermittent biliary obstruction and are responsible for its symptoms, including jaundice, abdominal pain, and cholangitis. In our case, locally invasive IPNB was found only after bile duct explant requiring the patient to undergo a Whipple procedure following liver transplant. It is questionable whether his recurrent HCC or prior TACE and TARE procedures are linked to such tumor development. This further highlights the need to have a high index of suspicion and awareness for the elusive presentation of IPNB. It should also be considered as a potential diagnosis in patients presenting with recurrent cholangitis.



[1750] **Figure 1.** Native bile duct collection demonstrating multiple fragments of intraductal papillary neoplasm of the bile duct (IPNB) with mucin production and high-grade dysplasia (hematoxylin and eosin stain, low-power field).

S1751

Isolated IgG4-Related Sclerosing Cholangitis a Rare Mimicker of Malignant Strictures

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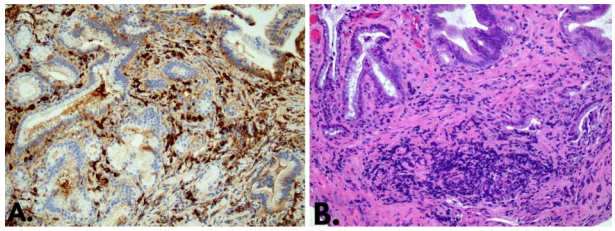
Introduction: IgG4 related sclerosis cholangitis (IgG4-RSC) is the biliary presentation of a systemic condition known as IgG4-related disease (IgG4-RD). It is a rare cause of cholangitis characterized by dense lymphoplasmacytic infiltrate rich in IgG4-positive plasma cells. It is a multi organ disease with male predominance which is most commonly associated with autoimmune pancreatitis. Isolated IgG4-RSC is exceedingly rare form of this disease often misdiagnosed as cholangiocarcinoma or pancreatic cancer.
Case Description/Methods: A 74-year-old diabetic man presented to the emergency department with complaints of a 44 pound weight loss within the last 2 months prior to presentation and a 1 month history of generalized pruritus, skin discoloration and pale stool. Exam was notable for scleral icterus, jaundice and no abdominal pain. Laboratory evaluation showed total bilirubin of 14.2 mg/dl, direct bilirubin > 10 mg/dl, AST 61 and ALT 60 with alkaline phosphatase of 328, lipase was normal. CA 19-9 antigen was 212, CEA and AFP were normal. A computed tomography (CT) of abdomen was obtained which showed no pancreatic mass however noted intra and extra-biliary ductal dilation with common bile duct measuring up to 1.8 cm. Magnetic resonance cholangiopancreatography (MRCP) revealed a focal wall thickening involving mid and lower portion CBD causing severe stenosis concerning for malignancy. Endoscopic retrograde cholangiopancreatography (ERCP) with endoscopic ultrasound (EUS) was planned. ERCP demonstrated a proximal dilatation with 2 cm long stricture located at the distal end of bile duct. A sphincterectomy was performed, biliary tree was swept with 12-15 mm ballon, multiple biopsies were obtained and CBD stent was replaced. Via EUS, a biopsy of CBD was obtained using a trans-duodenal approach. Pathology ultimately revealed plasma cells with IgG4 positive forms (Figure). Serum IgG4 was 246 mg/dl. Patient was diagnosed with IgG4 sclerosis cholangitis and he was started on systemic steroids with an adequate response.
Discussion: IgG4-RSC often initially presents as painless jaundice, weight loss or abdominal pain. Our case illustrates challenges in diagnosis given initial strong suspicion for malignancy. A combination of imaging, serological studies and biopsy are needed for diagnosis. Serum IgG4 is a non-specific test however concentration >135 mg/dl is often considered suspicious. Timely diagnosis and treatment with systemic steroids is essential in order to prevent secondary infection or permanent fibrosis.



[1751] **Figure 1.** A. Magnification 200x. IGG4 immunohistochemical staining with surrounding bile duct mucosa. B. Magnification 200x. Bile duct mucosa with extensive inflammation and lymphoid aggregates in the bottom of image.

S1752

Infiltration of the Biliary Tract With Cytomegalovirus

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Introduction: Cytomegalovirus (CMV) is known to affect immunosuppressed patients. CMV causes infections in post-transplant and HIV/AIDS patients but also those with advanced age or hematologic malignancies. This case presents CMV infiltration of the biliary tract in an elderly patient with 2 hematologic malignancies.
Case Description/Methods: A 74-year-old male with tobacco use and chronic alcohol abuse was undergoing evaluation for anemia and thrombocytopenia. EGD and colonoscopy were unremarkable. Bone marrow biopsy revealed myelodysplastic syndrome (MDS) and B cell lymphoma. He subsequently developed jaundice. Labs revealed a total bilirubin of 10.4, ALP 1077, AST 107 and ALT 131. Viral hepatitis panel and HIV were negative. MRCP showed a dilated CBD and a filling defect within the CHD extending to the left hepatic duct with contour irregularity and narrowing in the left hepatic duct giving a beaded appearance. ERCP demonstrated a dilated CBD and CHD in addition to mild left and right hepatic duct dilatation with no definitive strictures or filling defects. A plastic bile duct stent was placed due to poor drainage of contrast. His LFTs continued to remain elevated. Liver biopsy revealed moderate cholestasis, sinusoidal dilatation, mild portal inflammation, biliary metaplasia, and stage 2 fibrosis. Repeat MRCP showed worsening intrahepatic and extrahepatic biliary dilatation with no further filling defect. ERCP with cholangioscopy was then performed. Bile duct mucosa had diffuse superficial granular like appearance in the common duct and main hepatic branches, which did not resemble typical previous biliary stent inflammatory changes. Biopsies of the bile duct were obtained. A fully covered metal stent was placed for further biliary drainage due to persistent poor contrast drainage. Bile duct pathology showed chronic active inflammation with CMV viral inclusions in the hepatic hilum and left hepatic takeoff. CMV PCR from the biliary specimen was positive. Blood CMV IgG was positive. Blood CMV PCR and CMV IgM were negative. His liver enzymes showed improvement after a fully covered stent was placed. He was started on a 2 week course of valganciclovir, with serial blood CMV PCR monitoring while on Rituximab for his B cell lymphoma.
Discussion: This patient's immunosuppression secondary to MDS, B cell lymphoma and advanced age increased his risk of a CMV infection with bile duct involvement. Cholangioscopy can aid in viewing subtle bile duct irregularities with biopsies to assess for CMV involvement.

S1753

Kaposi Sarcoma: An Unusual Cause of Biliary Obstruction

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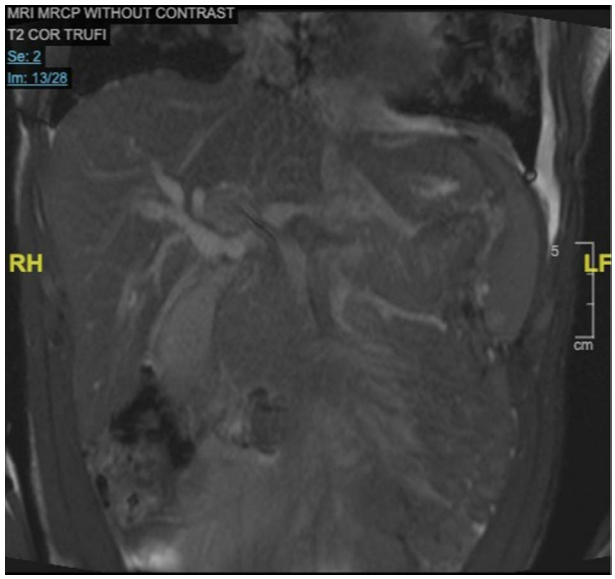
Introduction: Kaposi sarcoma (KS) is an AIDs defining disease caused by human herpesvirus-8 occurring in setting of severe immunosuppression. AIDS-associated KS affects primarily the skin and the lungs, but biliary tract involvement has been reported. We present an uncommon presentation of disseminated Kaposi sarcoma causing intrabiliary stricturing that resolved after biliary stent placement.
Case Description/Methods: 29-year-old African-American male with past medical history of HIV/AIDS with CD4 count 15 recently started on HAART, Kaposi sarcoma, who initially presented to our institution for elective bronchoscopy. During hospital stay, patient developed elevated alkaline phosphatase 1196, AST 180, ALT 150. He denied any abdominal symptoms. Ultrasound abdomen noted a distended gallbladder with markedly hyperechoic shadowing, dilated common bile duct to 1.5 cm. Of note, patient was previously admitted to a sister hospital with abnormal liver enzymes. MRCP at that time showed intra and extrahepatic biliary duct dilation with abrupt tapering of the portal vein and dilated CBD at the portal hepatis just above the pancreatic head (**Figure**). Patient underwent ERCP with sphincterotomy and cholangioscopy with biopsy, which showed irregular-appearing common bile duct mucosa characterized as circumferential scallop and friable appearing mucosa extending approximately 1 cm in length located near the mid common bile duct. Biopsies resulted in bile duct epithelium. He had a repeat MRI abdomen this admission that showed a possible filling defect or polypoid mass in proximal CBD resulting in biliary dilation Given this, he underwent EUS that noted a transition point in mid CBD with severely dilated duct to 14mm. FNA was performed. There was also a filling defect within the bile duct but no mass seen. A 3cm lymph node was identified in the periportal area and also sampled. ERCP with biopsy of structured area performed and 10Fx12 cm stent was deployed. The FNA of both the lymph node and bile duct returned Kaposi sarcoma. He was evaluated by oncology who planned for outpatient chemotherapy.
Discussion: AIDS-associated KS is known to primarily affect the skin; however, it can extend to internal organs, with the GI tract being the most common extracutaneous site. This may present with non-specific symptoms or as in this case elevated LFTs in a cholestatic pattern. Other times, patients may present with jaundice, cholangitis. Chemotherapy with antiretroviral therapy has been demonstrated to reduce disease progression.



[1753] **Figure 1.** MRCP with possible filling defect/polypoid mass in proximal CBD resulting in biliary dilation.

S1754

Late Metastatic Recurrence of Renal Cell Cancer to the Pancreas Presenting With Occult Gastrointestinal Bleeding and Symptomatic Anemia

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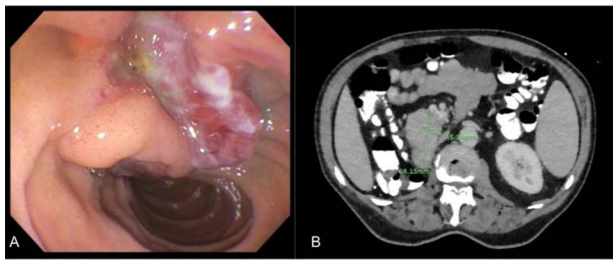
Introduction: Metastatic disease to the pancreas accounts for 2-5% of pancreatic malignancies. Renal cell cancer (RCC) is the most common primary malignancy which metastasizes to the pancreas. RCC is unique among cancers that metastasize to the pancreas in that that metastatic disease may not appear until over a decade after nephrectomy with curative intent. Guidelines recommend surveillance for recurrent disease with cross-sectional imaging for 5 years with further surveillance at the discretion of the clinician. We present a case of very late metastatic recurrence of RCC to the pancreas.
Case Description/Methods: A 68-year-old man with extensive tobacco use, RCC status post nephrectomy 21 years prior, and lung adenocarcinoma status post wedge resection 8 years prior presented with one month of shortness of breath. His physical exam was notable for pallor and small external hemorrhoids. Initial labs were notable for new iron deficiency anemia with hemoglobin 7.5 g/dL (MCV 76.8) and ferritin 5.4 UG/L. On EGD he was found to have an ulcerated, friable mass surrounding the ampulla which was better visualized with a side-viewing scope; biopsies were taken. A CTAP was obtained with IV contrast which demonstrated 2 masses in the pancreas, one invading into the second part of the duodenum, without evidence of any other sites of disease (Figure). Pathology returned consistent with metastatic clear cell RCC. He underwent total pancreatectomy, hepaticojejunostomy, gastrojejunostomy, cholecystectomy, and splenectomy with curative intent. Eight weeks later he was started on pembrolizumab with a plan for one year of treatment.
Discussion: Late metastatic recurrence of RCC after curative resection is a common phenomenon but usually occurs within 5 years of nephrectomy. Here we present a case of a very late recurrence, 21 years after nephrectomy, presenting as metastatic disease to the pancreas invading the duodenum leading to GI bleeding and symptomatic anemia. This case highlights the potential for late metastatic RCC to present as pancreatic malignancy over 2 decades after nephrectomy. The clinician must maintain a high index of suspicion in all patients with a history of RCC, no matter how remote, who present with new pancreatic masses. This case also demonstrates the wide array of presentations of metastatic disease to the pancreas with the presenting symptom in our patient being symptomatic anemia secondary to occult GI bleeding from duodenal invasion and ulceration.



[1754] **Figure 1.** A. Endoscopic appearance of ulcerated duodenal mass. B. CT abdomen with IV contrast demonstrating pancreatic mass invading into the duodenum.

S1755

Intrahepatic Cholangiocarcinoma in a Patient With Alcoholic Liver Cirrhosis and Markedly Elevated Serum Alpha-Fetoprotein

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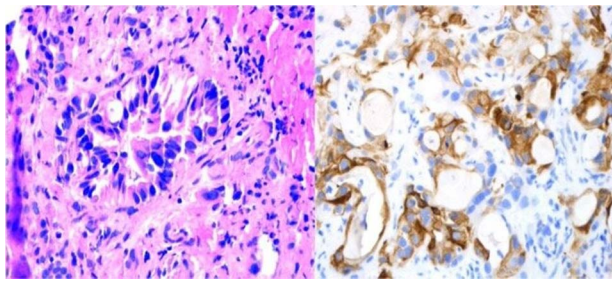
[1]RWJ Barnabas Health/Trinitas Regional Medical Center, Elizabeth, NJ; [2]Rutgers Health/Trinitas Regional Medical Center, Elizabeth, NJ.

Introduction: Intrahepatic cholangiocarcinoma (ICC) represents 5% of all primary liver malignancies, second only to hepatocellular carcinoma (HCC). Although risk factors such as primary sclerosing cholangitis, hepatolithiasis, and asbestos have been reported, the etiology of ICC is not well understood. An elevated serum AFP accompanied by a space-occupying solid lesion in a cirrhotic patient is typically indicative of HCC. In high-risk patients, such as those with chronic liver disease, AFP > 400 ng/mL has a high positive predictive value for HCC and a specificity of >95%. An elevated serum AFP greater than 20 ng/mL is seen in < 25% of ICC.
Case Description/Methods: We present a patient with a past medical history of alcoholic liver cirrhosis Child-Pugh Class C presenting with acute change in mental status. On arrival, he was hemodynamically stable with classic stigmata of chronic liver disease on the exam. Non-contrast CT Head was non-revealing. Laboratory investigations revealed an obstructive pattern of transaminitis with a normal ammonia level, negative hepatitis panel, and serum AFP of 1955.8 ng/mL. EGD demonstrated portal hypertensive gastropathy. CT Abdomen/Pelvis demonstrated a large infiltrative heterogeneous mass involving the entire right hepatic lobe with a non-occlusive thrombus extending to the portal vein. Ultrasound-guided liver biopsy and immunohistochemical analysis demonstrated positive expression for cytokeratin 20, and CA 19-9. Neoplastic cells were negative for Hep-Par1, Glypican 3, and Arginase 1. The histological pattern (**Figure)** was consistent with poorly differentiated adenocarcinoma, most likely pancreaticobiliary in origin consistent with intrahepatic cholangiocarcinoma (ICC).
Discussion: Elevated serum AFP accompanied by a space-occupying solid lesion in a cirrhotic patient is typically indicative of HCC. However, Xiong et al reported an increased risk by 3.92-fold of cholangiocarcinoma in patients with alcoholic liver disease. Even though, an increase in AFP has been reported in patients with ICC, an elevation higher than 20 ng/mL was seen in approximately 20% of the patients according to Wang et al. In our patient, surprisingly, the histopathological report showed findings consistent with ICC and negative hepatic markers, hence a high suspicious index should be held by clinicians for this entity upon approaching a patient with a solid liver mass associated with significantly elevated AFP levels.



[1755] **Figure 1.** On the left, histology of poorly differentiated adenocarcinoma composed of duct-like structures with high pleomorphism. H&E 400X. On the right, immunohistochemical study shows positive CA 19-9 stain of adenocarcinoma.

S1756

Intrapancreatic Accessory Spleen, an Important Consideration That Can Reduce Invasive Interventions in the Evaluation of Pancreatic Lesions

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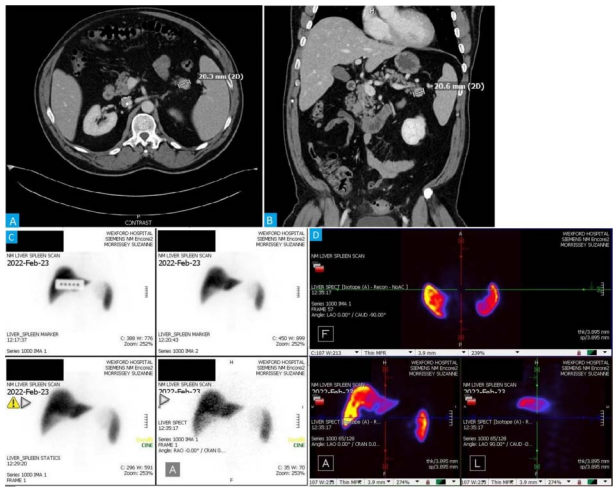
Introduction: The evaluation, diagnosis and management of pancreatic lesions is determined by radiographic imaging and Endoscopic Ultrasound with biopsy. We present a case of a pancreatic tail lesion that highlights the importance of considering an intrapancreatic accessory spleen within a differential diagnosis.
Case Description/Methods: A 48 year old White male with a past medical history significant for recurrent sigmoidal diverticulitis (complicated by a para-colonic abscess) and Clostridium difficile colitis who presented to an outpatient appointment for the evaluation of a pancreatic tail lesion that was incidentally identified on a CT abdomen/pelvis with contrast which was present on prior imaging dating back to 3 years time. On presentation, the patient endorsed that he was asymptomatic and specifically denied abdominal pain, nausea, vomiting, diarrhea, early satiety, weight loss, jaundice, flushing or hypoglycemia. The patient denied a personal or family history of pancreatitis or pancreatic cancer. Physical examination revealed that the patient was without scleral icterus, jaundice and had a benign abdominal exam. Lab findings revealed a normal complete blood count, complete metabolic panel and lipase level. CT abdomen/pelvis with contrast was reviewed which revealed a 2.1 cm hyperdense, hypervascular lesion localized within the pancreatic tail which was concerning for a pancreatic neuroendocrine tumor (Figure). Upon detailed review, the patient was determined a suitable candidate for EUS with biopsy. However, the patient was concerned regarding the risk of malignant seeding with transgastric biopsy. Prior to proceeding with biopsy Gastroenterology recommended a Nuclear Medicine liver spleen scan to rule out the presence of an intrapancreatic accessory spleen, as its presence would eliminate the need for invasive interventions. A NM scan was performed which revealed a 2.1 cm contrast-enhancing soft tissue density in the pancreatic tail, which was most compatible with intrapancreatic accessory spleen. No further diagnostic or therapeutic interventions were required given the benign nature of the patient's diagnosis.
Discussion: This case highlights the importance of the consideration of an intrapancreatic accessory spleen during the evaluation of pancreatic body and tail lesions, as clinicians can prevent unnecessary invasive diagnostic and surgical procedures ultimately reducing patient morbidity and mortality.



[1756] **Figure 1.** CT scan remarkable for Pancreatic tail lesion and NM scan showing 2.1 cm contrast-enhancing soft tissue density in the pancreatic tail, compatible with intrapancreatic accessory spleen.

S1757

Infected Biloma Secondary to Laparoscopic Cholecystectomy

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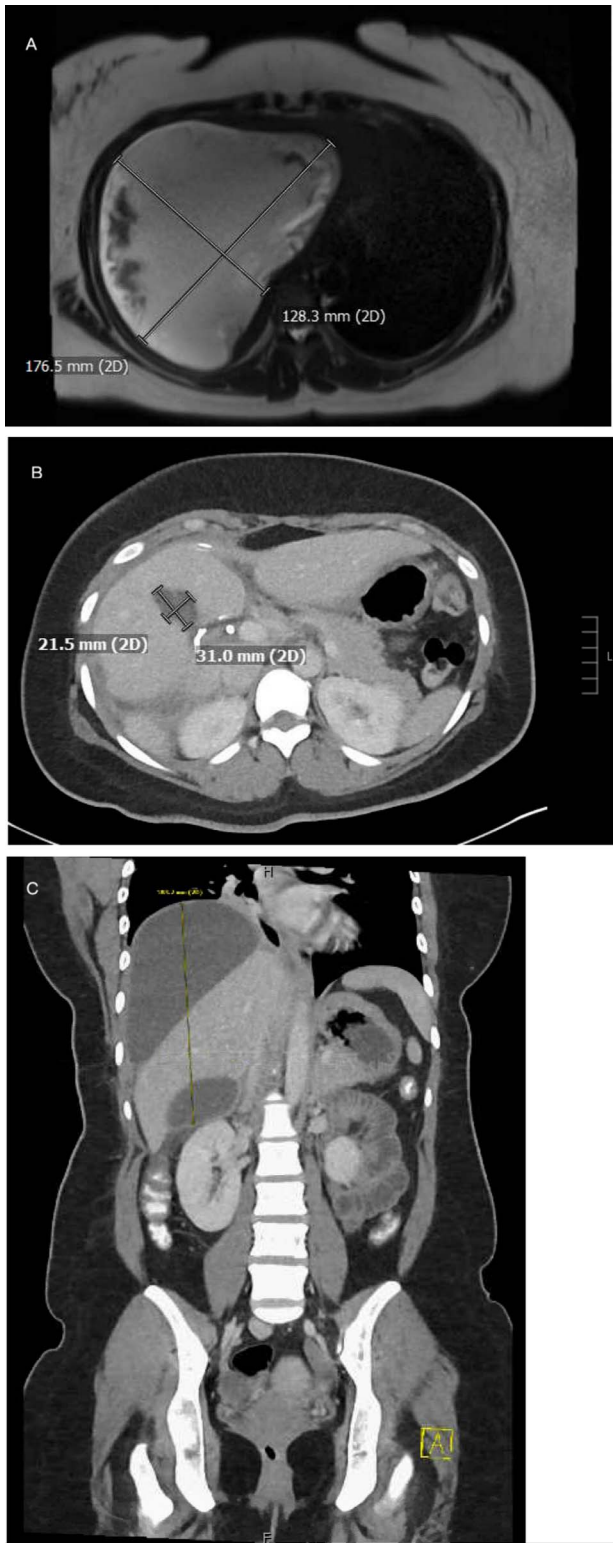
Introduction: Biloma is an extrahepatic bile collection secondary to iatrogenic or traumatic biliary tree disruption. It is a rare complication of laparoscopy cholecystectomy with an incidence rate of approximately 2.5%. Without proper management, biloma can become infected and cause life-threatening complications such as peritonitis, biliary fistula, bilhemia and hemobilia. Here we described a case of complicated biloma after laparoscopic cholecystectomy.
Case Description/Methods: The patient was a 24-year-old female with a past medical history of hypertension, obesity, and recent laparoscopic cholecystectomy complicated by hepatic subcapsular biloma. It was managed by biliary stent placement via endoscopic retrograde cholangiopancreatography (ERCP) and percutaneous drainage during the previous hospitalization. However, 6 days later, she presented with fever, chills, nausea, and right upper quadrant pain. Vital signs were fever 102.3 F and tachycardia 110 to 120 per min. The CT abdomen revealed decreased size in perihepatic fluid collection with air bubbles (14 x 11 x 18 cm; Figure). It also showed a common bile duct stent in place and a percutaneous drainage catheter tip in the inferior aspect of the collection. Lab results showed leukocytosis to 10.3, normal AST/ALT, total/direct bilirubin 2.1/12 mg/dL, and GGT 152 U/L. Broad-spectrum antibiotics were given in ED. The surgery team performed a laparoscopic lavage and discovered that the drain was not connected with the biloma. Two new drains were placed during the operation. She was discharged with PO antibiotics, and an outpatient follow-up was scheduled for drain removal.
Discussion: The management of biloma depends on the severity of the disease. Endoscopic therapy, such as a transpapillary biliary stent placement, can decrease the transpapillary pressure gradient, thus allowing preferential transpapillary bile flow rather than accumulation at the leaking site. However, given that stent placement does not reabsorb formed collection, patients failing ERCP should undergo percutaneous drainage or bile duct repair.Iatrogenic biloma can be detected by post-operational physical exams and image studies. Laparoscopic lavage with drainage should be considered in unresolved or infected biloma due to the high risk of peritonitis.



[1757] **Figure 1.** A. superior dominant of subcapsular periherpatic biloma B. collection in the gallbladder fossa C. coronal view of the biloma.

S1758

Mesenteric Vasculitis Presenting as a Pancreatic Pseudoaneurysm Originating From the Superior Mesenteric Artery

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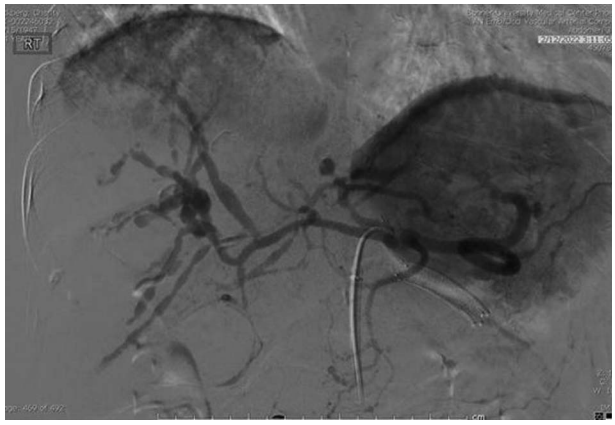
Introduction: Pseudoaneurysms form at the site of an arterial injury due to blood extravasating into the surrounding tissues and have a propensity to rupture as they do not involve any layer of the vessel wall. In the pancreaticobiliary system, they almost always occur in conjunction with pancreatitis, surgery, or trauma. Other causes, like vasculitis, are extremely rare. Approximately 60-65% of cases of pancreatic pseudoaneurysms originate from the splenic artery, 20-25% from the gastroduodenal artery, 5-10% from the hepatic artery, and less than 5% from the left gastric arteries. After diagnosis on CT, pseudoaneurysms necessitate treatment, most typically with coil embolization or stenting by interventional radiology.
Case Description/Methods: A 74-year-old woman female with a past medical history of hypertension, Parkinson's disease, and chronic pain presented with a 3-day history of jaundice, with subsequent lab workup revealing bilirubin 12.3 mg/dL, AST 238, ALT 18, alkaline phosphatase 760, and CA19-9 4087 u/mL. CT abdomen revealed a 9.5 x 5.5 x 6.5 cm mesenteric area pseudoaneurysm with 2.1 cm central arterial extravasation with a thick thrombus which surrounded the pancreatic head and neck and compressed the common bile duct, resulting in distal biliary obstruction (Figure). CT angiography was performed and showed innumerable mesenteric, intrahepatic, and intrasplenic pseudoaneurysms. Interventional radiology was consulted for fluoroscopic-guided embolization of the pseudoaneurysms and the 6 largest aneurysms involving the superior mesenteric artery were both stented and coil-embolized. The patient was treated with adjuvant prednisone and a repeat CT one month later showed resolution of the hematoma.
Discussion: This case illustrates a rare entity: a pancreatic pseudoaneurysm originating from the superior mesenteric artery that developed without typical predisposing factors, but due to an underlying systemic vasculitis. This case enforces that the gold standard for diagnosis of both visceral pseudoaneurysms and vasculitis is CT angiography, as the diffuse nature of the mesenteric pseudoaneurysms was not evident until angiography was utilized. This case challenges typical treatments for pseudoaneurysms, as an aggressive approach of both stenting and transcatheter coil embolization of large vascular territories was used and resulted in resolution of the pancreatic pseudocyst with no evidence of bowel infarction, despite the aggressive therapy.



[1758] **Figure 1.** CT angiogram showing diffuse mesenteric pseudoaneurysms.

S1759

Management of Bouveret Syndrome: A Case Report

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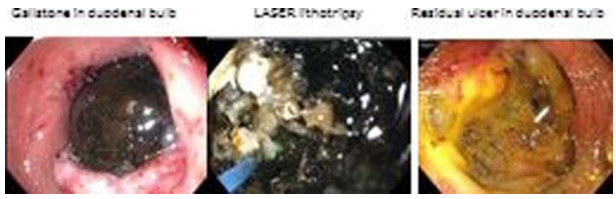
Introduction: Bouveret syndrome is an unusual complication of gallstone disease, causing less than 0.2% of small bowel obstructions. It often requires multidisciplinary approaches for treatment. We present here a unique case report of Bouveret syndrome, managed successfully using Holmium LASER alone.
Case Description/Methods: A 74-year-old man presented to our hospital with vomiting, decreased appetite, periumbilical pain and nausea. He denied heartburn, fever, chills or jaundice. His past medical history was significant for cardiomyopathy (EF 35%), atrial fibrillation and ventricular tachycardia with AICD placement. He had been admitted 3 months prior with acute cholecystitis and elevated liver function tests. Due to cardiac comorbidities, he was treated with IV Zosyn, cholecystostomy tube and ERCP with sphincterotomy with improvement and he was discharged. 6 weeks later, a tube check confirmed correct placement of the tube and decompression of the gall bladder. This time, his pain differed from the right upper abdominal pain of prior admission. His tube drainage had reduced considerably in the previous day. Physical examination revealed epigastric and supraumbilical tenderness with no guarding or rebound. The cholecystostomy bag had purulent drainage. WBC count was 11.95 x 10,000/cu.mm; liver enzymes and S. Lipase were normal. CT scan showed a change in the position of a previously seen large gallstone, measuring 3.7 cm in diameter. During the previous admission, it was in the gall bladder neck. At this time, it was occupying the duodenal bulb lumen, indicating development of a cholecystoduodenal fistula, and causing complete obstruction. The surgical team recommended endoscopic treatment due to anticipated friability of the tissue (Figure). Endoscopic evaluation showed complete obstruction of the duodenal bulb by a large smooth stone, not allowing passage of even a guidewire beyond the stone. Lithotripsy was chosen to help relieve the obstruction. 410 micron and 990 micron Holmium LASER probes were used with assistance from Urology, via an antegrade approach from the pylorus through a gastroscope. After 2 sessions of lithotripsy 4 days apart, each breaking through pigmented and calcified stone, the stone passed through the small bowel, relieving the obstruction.
Discussion: The patient did not require surgical intervention and has recovered well. This case illustrates the feasibility of Holmium LASER to treat heavily calcified and pigmented gallstones. Holmium LASER should be developed for use in GI Endoscopy.



[1759] **Figure 1.** Gallstone obstructing duodenal bulb; LASER lithotripsy of calcified and pigmented stone; residual ulcer in duodenal bulb after stone passed.

S1760

Metastatic Cervical Squamous Cell Carcinoma of the Distal Bile Duct

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Introduction: Squamous cell carcinoma (SCC) of the bile duct is sporadic. Most cases are primary. Only 2 cases of cervical SCC metastatic to the bile ducts have been reported. Consideration of this diagnosis is essential in patients who have a history of malignancy and present with symptoms of biliary obstruction.
Case Description/Methods: A 63-year-old female presented with persistent abdominal pain, nausea, and vomiting. Mild abdominal tenderness and scleral icterus were noted on physical examination. Bilirubin, AST, and ALT were elevated. Magnetic resonance cholangiopancreatography (MRCP) revealed dilated intra- and extra-hepatic bile ducts; however, there was no evidence of an apparent mass or gallstones. A lesion was observed at the ampullary level measuring around 1 cm that was brushed and biopsied on endoscopic cholangiopancreatography (ERCP). A metal stent was placed for decompression in the common

bile duct. Pathology revealed poorly differentiated SCC with an immune profile favoring metastasis from the gynecologic tract. The patient was diagnosed with SCC of the cervix 15 months prior to presentation. A biopsy of the cervical tissue showed poorly differentiated cervical SCC with positivity in CK7 and HMWK, scattered positive staining of vimentin. Chemo-radiation was initiated with Cisplatin, Paclitaxel, and Bevacizumab. Next-generation sequencing (NGS) showed an immunohistochemical profile consistent with gynecological cancers, namely, MLH1, PMS2, MSH2, and MSH6. Pembrolizumab was also started due to positive PDL-1 expression and mutations in ATM and KMT2D. These agents were continued after she underwent the biliary decompression.
Discussion: Metastatic SCC of the biliary tract is rare. Due to its low incidence, there is no standardized treatment strategy; however, there are reported cases of surgical resection in addition to chemoradiotherapy. Very few cases of cervical SCC have been reported to spread to gastrointestinal organs. NGS is a modality that has been widely implemented. The combination of immunohistology staining and NGS can aid in the diagnosis and help guide chemotherapy. The genetic variations present a valuable tool to aim the treatment of cervical cancer. While survival rates for patients with cervical carcinoma have improved, treatment remains difficult as not enough cases exist to compare treatment modalities. Limited knowledge is present regarding managing extra-hepatic duct squamous cell carcinoma since it is rare.

S1761

Lemmel's Syndrome, a Rare Complication of Periampullary Diverticula

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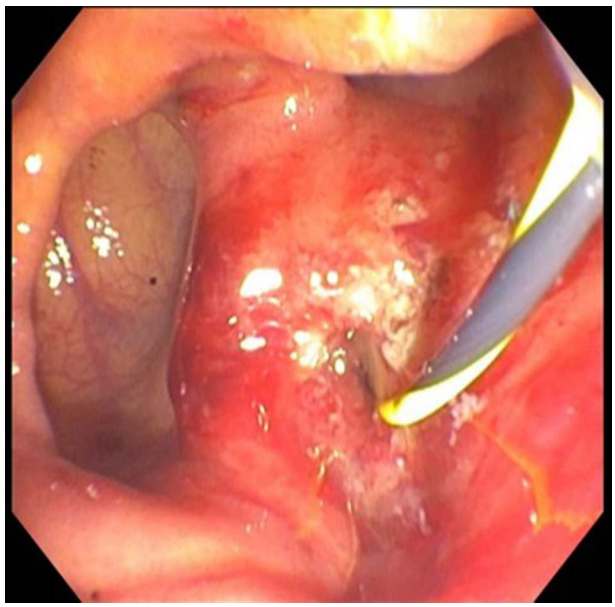
Introduction: Lemmel's Syndrome (LS) is obstructive jaundice that occurs concurrently with the periampullary diverticula. Obstructive jaundice can cause a build-up of pancreatic enzymes and bilirubin similar to choledocholithiasis. Typically there will be extrahepatic and intrahepatic dilation of the bile ducts.
Case Description/Methods: A 58-year-old gentelman presents to the emergency department with abdominal pain, nausea, and vomiting. Upon admission, he developed a fever of 101.4 F, and tachycardia (Pulse = 131 BPM). On physical exam, he had a tender mid-epigastrium with no rebound, distention, guarding, or rigidity. Liver function tests were elevated: AST = 487U/L (normal range: 10-36 U/L), ALT = 365 U/L (normal range: 9-46 U/L), ALP = 155 U/L (40-115 U/L), and total bilirubin= 3.9 mg/dl (normal range: 0.6-1.2 mg/dl) with direct bilirubin of: 3.2 mg/dl (normal range: 0.0-0.3 mg/dl). An US of the right upper quadrant revealed normal size biliary ducts. CT scan revealed a diverticulum in the second part of the duodenum, with no dilation in the extra/intrahepatic ducts. He was started on antibiotics and fluids as per sepsis protocol and then underwent ERCP (Figure). The upper GI tract was normal, however, the normal major papilla in the descending duodenum was located entirely within a large diverticulum (image-1). The pancreatic duct was deeply cannulated with the sphinctertomy and a stent was placed. In the following 2 days, he improved dramatically.
Discussion: Periampullary diverticula (PAD) are the most common type of the duodenal diverticula with 70-75% of all duodenal diverticula. They consist of outpouching in the extraluminal part of the duodenum at 2-3 cm surrounding the ampulla of vater. Most of the cases are asymptomatic and diagnosed incidentally during endoscopies, though there is a wide range of complications. LS is defined as hepatocholeangiopancreatic disease leading to obstructive jaundice in the absence of choledocholithiasis. The diagnosis of the LS is challenging. Many modalities can be used including CT, MRI, and upper gastrointestinal endoscopy. Though, a side-viewing endoscope during ERCP is the gold standard in diagnosing PAD. On imaging, it will appear as thin-walled cavitary lesions located on the medial wall of the second portion of the duodenum with extrahepatic and intrahepatic biliary ducts dilatation. In our case, the patient presented with obstructive jaundice with cholangitis without dilatation of the biliary ducts which makes the case more unique.



[1761] **Figure 1.** ERCP revealed: ERCP normal upper GI tract, however the normal major papilla in the descending duodenum was located entirely within a large diverticulum.

S1762

Loop Diuretic-Induced Pancreatitis

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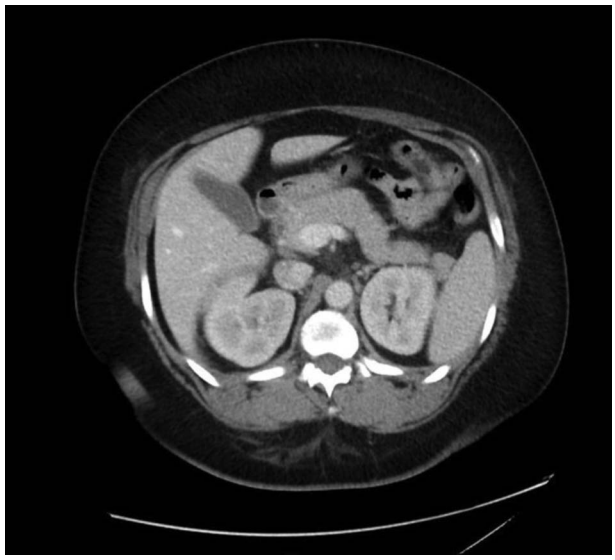
Introduction: Acute Pancreatitis (AP) has become the leading cause of hospitalization related to Gastrointestinal diseases in the US in recent years. More often than not, AP is usually associated with gall stones and alcohol abuse disorder. Drug-Induced Pancreatitis (DIP), once considered a rare cause (0.1 – 2%) of AP, is seen more clinically and is reported more frequently in the literature lately. Here, we describe a patient who developed acute pancreatitis after being on loop diuretic for a short duration.
Case Description/Methods: A 36-year-old woman with past medical history of nephrotic syndrome of unknown etiology, mixed connective tissue disease, and pulmonary embolism presented to the ED with acute epigastric pain, nausea and vomiting. Her medications included Prednisone, Plaquenil, and Eliquis. Three weeks prior to this presentation, she was started on furosemide for anasarca. In the ED labs, her lipase was 122, BUN 20, WBC 8.8, and CT abdomen revealed acute interstitial edematous pancreatitis (Figure). She was treated with IV fluids and pain control. On workup, She denied alcohol and illicit drug usage, triglycerides were not significantly elevated (252), no hypercalcemia, and abdominal ultrasound showed no evidence of gallstones. IgG4 was negative, denied hereditary pancreatitis and sulfa drug allergy. Furosemide was held as it was a possible etiologic agent, and she improved. A month later, she was switched to bumetanide as an outpatient, but within 10 days of starting bumetanide, she was readmitted with epigastric pain and Lipase 907 and was diagnosed with AP. She had an Endoscopic ultrasound (EUS) that showed no evidence of pancreatic mass, cysts, common bile duct stones, or evidence of chronic pancreatitis. Bumetanide was held and later switched to ethacrynic acid with the resolution of symptoms.
Discussion: Though perceived as a rare cause, the true incidence of DIP is much higher, as they are predominantly underreported and underdiagnosed. Based on Naranjo et al. Naranjo adverse drug reaction probability scale, our patient scored 7 (yes to questions 1,2,3,4,10), making loop diuretic the most probable cause of AP. Literature suggests loop diuretic exerts direct toxicity on pancreas and a concurrent

decrease in extracellular fluid volume leads to pancreatic ischemia and inflammation. Nevertheless, our patient developed AP only with furosemide and bumetanide, indicating an added immune-mediated pathway involving sulfa metabolites likely contributing to AP, which also has been reported in the literature.



[1762] **Figure 1.** CT abdomen from the time of initial ED presentation demonstrating edematous pancreas with minimal surrounding fat stranding and fluid in the region of the head and uncinate process, consistent with acute interstitial pancreatitis.

S1763

Metastatic Ampullary Lesion as a Rare Cause of Acute Blood Loss Anemia

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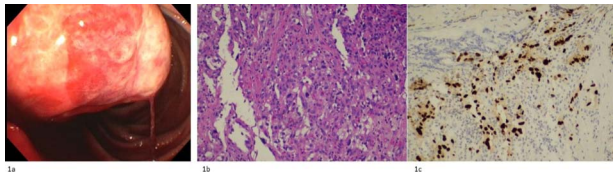
Introduction: Metastatic spread to the upper gastrointestinal tract is rare. Typically, the stomach is more often affected than the duodenum. The most common primary cancers found in the upper gastrointestinal tract include lung cancer, breast cancer, and melanoma. Metastasis to the ampulla of Vater (ampulla) is rare with only 33 cases reported. Affected patients commonly present with abdominal pain, jaundice, pruritus and upper GI bleeding. We present a case of acute blood loss anemia and melena from metastatic renal cell carcinoma to ampulla.
Case Description/Methods: A 77-year-old female with history of stage IV, clear-cell renal cell carcinoma (RCC) status post right nephrectomy and partial left nephrectomy 3 years prior, complicated by stable metastatic spread to the spine and lungs, actively on pembrolizumab, presented with a week of shortness of breath, fatigue, and dark stools. Her hemoglobin was 5.1mg/dl, down from a baseline of 11mg/dl. CT scan of the abdomen revealed stable metastatic disease. Esophagogastroduodenoscopy was significant for hematin in the stomach and a large fungating mass with bleeding was found at the ampulla (Figure A). Biopsy results returned positive for renal cell carcinoma (Figure B and C)
Discussion: We present a case of acute anemia due to metastatic renal cell carcinoma that had spread to the ampulla, 4 years after initial nephrectomy. Of the 33 cases of metastatic spread to the ampulla reported in the literature, 39% were renal carcinoma, 30% melanoma, and 12% breast cancer. Bone, ovary, uterus, bladder, and larynx primary cancers have also been reported. The time interval between the diagnosis of RCC and ampullary metastasis can be as high as 10 years. In our case, the patient had active disease in lungs, and given all her comorbidities, resection of all lesions was not possible. As the treatment of cancer rapidly progresses with advances in genetically guided therapeutics as well as immunotherapy, patients presenting with what today is considered a rare complication as a site of metastatic disease will become more common and recognizing such rare complications can aid in a faster diagnosis and therapy.



[1763] **Figure 1.** Figure A: ampullary mass oozing actively as visualized on Endoscopy ; Figure B: H&E staining of biopsied tissue. Ulcerated tissue with loose, infiltrative cells within the edematous stroma and enlarged nuclei noted; Figure C: Vimentin positive staining.

S1764

Localized Truncal Xanthoderma Secondary to Bile Leakage Post Laparoscopic Cholecystectomy

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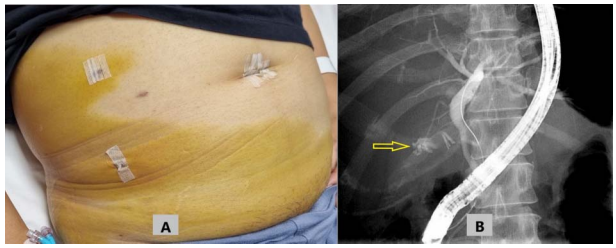
Introduction: Xanthoderma is a yellow to yellow-orange discoloration of the skin. The 2 main pathologies leading to xanthoderma are jaundice and carotenoderma. Although the most common mechanism for jaundice is the elevation of serum bilirubin, localized jaundice can rarely occur in the settings of bile leakage into the retroperitoneal space.
Case Description/Methods: A 40-year-old female patient presented with biliary pain. Examination showed right upper quadrant tenderness. Initial labs revealed total bilirubin of 49 μmol/L, with a direct bilirubin of 47μmol/L (Table). The US abdomen showed cholelithiasis. MRCP showed a small distal CBD stone; CBD diameter was 9 mm. She underwent laparoscopic cholecystectomy with an intraoperative cholangiogram. The small distal CBD stone was pushed to the duodenum. Repeated IOC showed a smooth passage of contrast. Her clinical status didn't improve postoperatively, and her bilirubin was persistently elevated. Localized yellowish discoloration of the right side of the abdominal wall and the suprapubic area was observed 5 days after the surgery (Figure A). Repeated MRCP showed suspicious distal CBD stone with gall bladder bed collection and diffuse inflammatory change of the right abdominal wall. Endoscopic retrograde cholangiopancreatography (ERCP) revealed a bile leak at the site of the cystic stump (Figure A) and a small filling defect in the distal CBD. Sphincterotomy and CBD stenting were performed. The bilirubin improved gradually after ERCP, but the abdominal pain persisted. CT abdomen showed large perihepatic and perisplenic fluid collection measuring 10x 17.6x 20 cm, necessitating US-guided percutaneous drainage. Turbid yellow fluid was drained, confirming that the collection was a biloma related to the bile leak. Four weeks later, the percutaneous drain was removed after the collection resolved. Repeated ERCP showed no biliary leak.

Discussion: In our patient, the localized xanthoderma post laparoscopic cholecystectomy was secondary to bile leakage from the cystic duct stump. In a similar case report, the authors explained that the localized xanthoderma occurred as a result of bile staining of the tissues deep to the membranous layer of the superficial fascia (fascia of Scarpa) after the bile has leaked from the cystic duct through the hepatoduodenal ligament. The management of our patient involved sphincterotomy and bile duct stenting combined with percutaneous drainage of the collections.



[1764] **Figure 1.** A: Localized yellowish discoloration of the anterior abdominal wall. B: An ERCP fluoroscopic image showing contrast leakage near the cystic stump.

Table 1. Laboratory investigations

Laboratory test	Patient's value	Normal range
Total bilirubin	49 μmol/L	0-21
Direct bilirubin	47 μmol/L	0-9
AST	287 U/L	0-32
ALT	249 U/L	0-33
Alkaline phosphatase	264 U/L	35-104

S1765

Management of Young Patients With Recurrent Episodes of Acute Pancreatitis

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Introduction: Hereditary pancreatitis (HP) is a rare genetic condition, the evolution of which is marked by recurrent episodes of acute pancreatitis that begin in childhood or adolescence and lead to the early inception of chronic pancreatitis and a significant increase in the risk of pancreatic cancer in young adults.
Case Description/Methods: We present the case of a 22-year-old patient, with repeated appearances in the gastroenterology department for episodes of acute pancreatitis (11 episodes), started at the age of 15, for which recent computed tomography scan (2022) showed changes suggestive for chronic pancreatitis. Anamnestic and laboratory tests have ruled out alcohol use, medications, dysmetabolic syndrome, as the most common causative factors involved in triggering episodes of acute pancreatitis. The echo-endoscopic investigation with biopsy was negative for specific markers of autoimmune pancreatitis. Given the positive history of acute pancreatitis (multiple episodes) of relatives (father and uncle), the presence of inherited genetic mutations was taken into account. The result of the genetic panel revealed the presence of a pathogenic variant probably heterozygous in the PRSS1 gene associated with hereditary pancreatitis (Figure). The case was included in an individualized management program with multidisciplinary involvement, with a focus on pain management, medical therapy for endocrine and exocrine insufficiency, and surveillance of the sequelae of chronic pancreatitis and pancreatic adenocarcinoma.
Discussion: The recurrence of acute pancreatitis episodes at a young age, with negative results for the causes frequently involved in the onset of acute episodes (toxic, metabolic, autoimmune), but with a positive family history of acute pancreatitis, justifies the extension of genetic testing investigations to establish an early diagnosis on hereditary pancreatitis. Given that there are no clearly established methods for preventing the development or progression of the disease in the context of the presence of a genetic mutation associated with HP, the emphasis in case management will focus on avoiding the triggers that can exacerbate and aggravate pancreatitis and monitoring the progression toward adenocarcinoma pancreatic.

Clinical Information: Patient with recurrent episodes of acute pancreatitis. The etiological autoimune cause was excluded, metabolic or toxic. Relevant family history: father and uncle have history of recurrent episodes of acute pancreatitis.

RESULT AND INTERPRETATION

The presence of a heterozygous likely pathogenic variant in the *PRSS1* gene associated with hereditary pancreatitis has been identified. (See Recommendations)

Gene	Variant*	Zygosity	Inheritance pattern	Classification^
PRSS1	NM_002769.5:**c.311T>C p.(Leu104Pro)**	Heterozygosis	Autosomal dominant	**Likely Pathogenic**

[1765] **Figure 1.** The result of the genetic panel.

S1766

Metastatic Renal Cell Cancer with Pancreatic Mass

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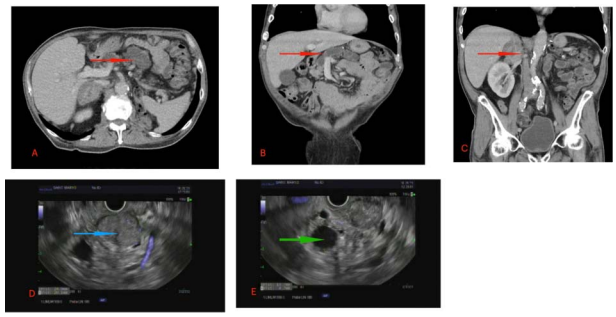
Introduction: Pancreas is an uncommon site of metastatic disease. Almost 63% of pancreatic metastasis arise from renal cell cancer. We report a case of metastatic renal cell cancer to pancreas, with contralateral adrenal metastasis 15 years after a radical nephrectomy.
Case Description/Methods: 61 years old man with past medical history of renal cell cancer, status post left radical nephrectomy fifteen years ago, presented with dull pain in right lower abdominal for one day. He denied any other abdominal symptoms or weight loss. He smokes up to half a pack of cigarettes daily. His examination was unremarkable but he was noted to have elevated lipase (213U/L) and a bilirubin of 1.1 mg%. On computer tomography abdomen he had bulky, enhancing, soft tissue mass (~6.7 cm), with dilated pancreatic duct of 1.5 cm with right adrenal mass (Figure). On EUS, the lesion appeared well circumscribed and previously known pancreatic duct dilation was seen. Fine needle biopsy of the mass, which showed numerous atypical cells with round irregular nuclei with prominent nucleoli with clear cytoplasm suggestive of clear cell renal cell carcinoma (Figure). On immune -histochemistry, the tumor was positive for CD10, renal cell carcinoma antigen and PAX 8(Figure)
Discussion: Metachronous renal cell cancer to pancreas fifteen years after radial nephrectomy is rare. Our patient had pancreatic metastasis with adrenal involvement and presented with vague abdominal symptoms. .He was noted to have elevated lipase and dilation of main pancreatic duct. Due to a remote history of nephrectomy and the presenting complaints, pancreatic primary was suspected. However, on endoscopic ultrasound the patient was noted to have a well circumscribed lesion in the head of pancreas with dilated pancreatic duct(Figure). Regular borders and absence of pancreatic duct dilation suggested

metastatic disease. Our patient had well circumscribed lesion but with a dilated duct. Most pancreatic metastasis are reported to occur within 10 years after treatment of the primary disease but our patient had metastasis fifteen years later without any loco-regional recurrence. Endoscopic ultrasound is extremely important and allows to evaluate the extent of the disease, involvement of portal vein/ superior mesenteric artery, presence of enlarged lymph nodes, which are a must for evaluating the surgical candidature besides providing tissue diagnosis which helped us establish the diagnosis of metastasis.



[1766] **Figure 1.** CT(A,B,C) and EUS(D,E) findings. Image A shows the mass in the Pancreatic head(arrow with red head) with a dilated pancreatic duct seen in imageB(arrow with red head). Adrenal metastasis is seen in image C marked with a red arrow. On EUS, the mass appears well circumscribed(blue arrow, image D) with dilated pancreatic duct seen (green arrow) in the head of pancreas in E.

S1767

Metastatic Solid Pseudopapillary Neoplasm of the Pancreas, Rare Presentation of an Uncommon Tumor

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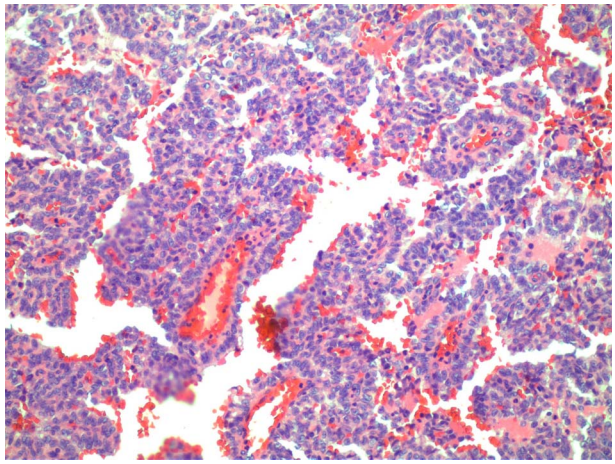
Introduction: Solid pseudopapillary neoplasms (SPN) make up less than 3% of all diagnosed pancreatic neoplasms. Abdominal CT or MRI are the imaging modalities most utilized, and biopsy is necessary to confirm the diagnosis. Resection of the mass is standard of care and the prognosis is quite good, with 5-year survival rates of over 95%. Both recurrence and metastasis of the tumor are exceedingly rare, occurring in only 1-4% of patients.
Case Description/Methods: A 26-year-old female presented with several years of abdominal pain. CT scan revealed a 10cm mass in the neck and body of the pancreas and patient underwent a hybrid resection including subtotal pancreatectomy, splenectomy, and cholecystectomy. Pathology showed a neoplasm extending to the inked margins with positive beta-catenin and CD10 nuclear staining with no evidence of metastasis. At the age of 32, the patient presented again with abdominal pain. A CT and PET scan showed numerous LUQ, perirenal, pancreatic, and periadrenal soft tissue densities. Laparoscopic resection of a 3.5x3 cm soft tissue mass near the remnant pancreas was performed, showing fibrous stroma with angulated islands of epithelioid cells with cystic degeneration and positive staining for vimentin, CD56, and beta-catenin. Resection of multiple abdominal soft tissue tumors, in bulk measuring more than 10 cm, as well as complete omentectomy, left adrenalectomy, bilateral salpingo-oophorectomy, appendectomy, and hyperthermic intraperitoneal chemotherapy with cisplatin was performed. Pathology showed multiple metastatic tumors all consistent with SPN. The appendix, left adrenal gland, bilateral ovaries, and all lymph nodes were negative for any tumor. Two years later, MRI revealed 3.0 x 2.1 cm hyperintense lesion in the caudate lobe of the liver. EUS with biopsy was performed, revealing clusters of uniform, bland cells with ovoid nuclei and associated fibrillary stroma circumscribing vessels with a background of hemorrhagic debris (Figure). Immunohistochemistry showed cells positive for vimentin, LEF1, and beta-catenin, again consistent with SPN.
Discussion: This is one of the few cases of multiple instances of both recurrence and metastasis of SPN documented in the literature. This case report shows that serial imaging studies and close follow-up are needed even years after resection and that, although rare, recurrence and metastasis can occur more than one time after the initial diagnosis.



[1767] **Figure 1.** Biopsy of the caudate liver lesion showing clusters of uniform, bland cells with ovoid nuclei and associated fibrillary stroma and hemorrhagic debris, consistent with SPN.

S1768

Missing the Viewing Angle on Obstructive Jaundice: Lemmel's Syndrome

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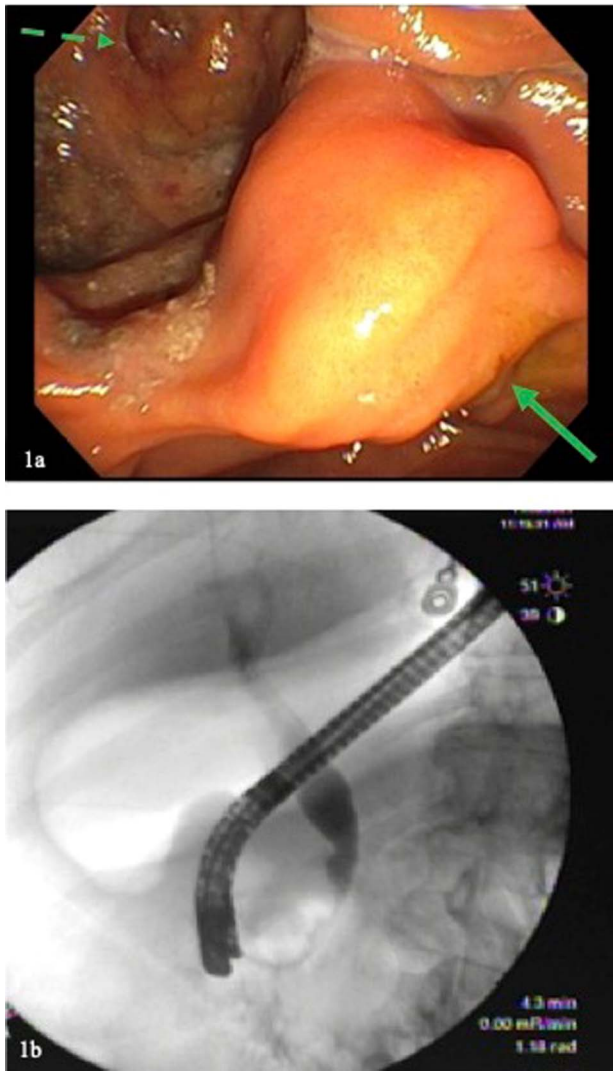
Introduction: Lemmel's syndrome (LS) is a rare form of obstructive jaundice that is often misdiagnosed. We report a case of elusive jaundice masquerading as DILI until endoscopy revealed the presence of a periampullary diverticulum.
Case Description/Methods: A 79-year-old man presented with fatigue and pruritus. Medical history was notable for recent E. coli bacteremia treated with cefdinir. Upon presentation, vital signs were normal. Laboratory studies revealed total bilirubin 8.6 mg/dL, direct bilirubin 6.1 mg/dL, AST 106 U/L, ALT 236 U/L, alkaline phosphatase 854 U/L, and GGT 1120 U/L. CT of the abdomen/pelvis showed dilation of the pancreatic duct and intra/extrahepatic bile ducts. MRCP had similar findings but no biliary stones or masses. Dedicated CT of the pancreas was also negative for pancreatic or biliary lesions. EGD with EUS showed ductal dilation but was otherwise unremarkable. Liver biopsy revealed evidence of cholestasis. Steroid therapy was started for possible DILI however cholestasis continued to worsen and abdominal pain

ensued. Repeat EGD with a duodenoscope revealed a patulous ampulla and large periampullary diverticulum (Figure A). ERCP was performed revealing no choledocholithiasis or stricture; however, an irregularity was found in the distal CBD due to compression from the diverticulum (Figure B). Cytology was obtained from the CBD irregularity which showed benign biliary epithelium. The diagnosis of LS was made and a 10Fr 7cm plastic biliary stent was placed. The patient's bilirubin and alkaline phosphatase quickly improved from a peak of 17.8 mg/dL and 1036 U/L, respectively, to 8.5 mg/dL and 719 U/L within 24 hours of stent placement. Due to recurrent cholangitis requiring multiple ERCPs the patient is awaiting diverticulectomy and cholecystectomy for definitive management.

Discussion: LS is characterized by obstructive jaundice due to a periampullary diverticulum. In many patients the diagnosis can be challenging. In this case the presumption of DILI led to a delay in diagnosis. Only in the setting of persistent jaundice and worsening right upper quadrant abdominal pain was the possibility of LS entertained. In hindsight, the patient's initial bacteremia was likely due to cholangitis due to LS. EGD with duodenoscope helped to confirm the diagnosis. Visualization of a periampullary diverticulum via duodenoscope is considered the gold-standard diagnostic modality for LS. Its use should be considered in cases where suspicion for LS is raised.



[1768] **Figure 1.** (1a) Periampullary diverticulum, with solid arrow demarcating the papilla and dashed arrow revealing the large periampullary diverticulum containing smaller internal diverticuli. (1b) ERCP with cholangiogram showing external compression of CBD by the periampullary diverticulum with upstream ductal dilation.

S1769

Nirmatrelvir/ritonavir - A New Culprit of Acute Pancreatitis

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Introduction: Nirmatrelvir/ritonavir is a new medication approved for the treatment of COVID-19 infection. It prevents viral replication by inhibiting the SARS-CoV-2 main protease. While mild adverse effects were described, including dysgeusia, diarrhea, hypertension and myalgia1, there were no reported cases of pancreatitis.

Case Description/Methods: An 81-year-old female with a past medical history of hypertension and COPD presented to the hospital complaining of abdominal pain and nausea for one day. She had no history of alcohol, tobacco or marijuana use, recent travel, or trauma. Her medications included lisinopril and prednisone, and she had completed a 5-day course of nirmatrelvir/ritonavir for the treatment of COVID-19 infection 2 days prior to presentation. On abdominal exam, she had left upper and lower quadrant tenderness. Blood tests revealed an amylase of 1333 U/L, lipase of 3779 U/L, triglycerides of 297 mg/dL and calcium of 8.7 mg/dL. CT scan revealed an indurated pancreatic body and tail with peripancreatic fluid along the paracolic gutter. Ultrasound of the abdomen and MRCP did not reveal any acute findings. IgG subclasses 1-4 were normal.

Discussion: According to the revised Atlanta criteria, the patient had clinical findings consistent with acute pancreatitis. Common causes such as gallstone, alcohol, autoimmune and hypertriglyceridemia-induced pancreatitis were ruled out. There were no masses or structural abnormalities on imaging that might have explained her diagnosis. There have been at least 2 reported cases of lisinopril and prednisone induced pancreatitis, however according to Badalov et al.2 both of these medications are class III drugs that lack any rechallenge in the literature. Moreover, the patient had been taking these medications for many years, making them an unlikely cause of the presenting diagnosis. There are no reports of nirmatrelvir/ritonavir associated pancreatitis or known pharmacologic interaction with her home medications, and a meta-analysis conducted by Babajide et al. revealed no association between acute pancreatitis and COVID-19 infection (3). Given the negative findings stated above and the recent initiation of a new medication, nirmatrelvir/ritonavir was the likely cause of acute pancreatitis.

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S1770

Neuroendocrine Tumor in the Biliary Tract Presenting With Cholangitis

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Introduction: Neuroendocrine tumors (NETs) of the biliary tract account for less than 2% of all gastrointestinal cancers with an incidence of 0.32%.[1,2] Extrahepatic biliary NETs are mostly found incidentally in the distal common bile duct (CBD) but in rare cases they may present with obstructive jaundice. We present a case of an elderly male with symptoms of biliary obstruction and acute cholangitis secondary to a NET in the proximal bile duct.
Case Description/Methods: A 64-year-old male with hypertension presented with epigastric abdominal pain, vomiting and fever. Patient reported an unintentional 50-pound weight loss over the past year. Vital signs were remarkable for heart rate of 118 beats/min. Labs revealed White blood cell count of 21,000 uL, AST 303 U/L, ALT 563 U/L, alkaline phosphatase 300 U/L, total bilirubin 1.9 mg/dL. CT abdomen showed intrahepatic ductal dilatation to 1.1 cm, and a 1.2 x 1.1 cm low attenuating lesion within the left lobe of the liver and wall thickening of the proximal jejunal loops. Piperacillin-tazobactam was started for acute cholangitis. Endoscopic ultrasound showed duct dilation in the left hepatic lobe and ERCP showed ulceration of major papillae. Following biliary sphincterotomy, exploration revealed severe stenosis of the main bile duct and a 12 mm polypoid lesion in the upper third of the CBD. Histology showed well differentiated NET with trabecular and organoid pattern, positive chromogranin and synaptophysin with Ki-67 index of 3%. Special staining was also positive for reticulin and trichrome. A 10fr x 9cm stent was placed. Following a 7-day course of antibiotics and symptom resolution, the patient was discharged with a plan for elective surgery. Left hepatectomy, CBD resection, and Roux-en-Y hepaticojejunostomy were performed 6 months after hospitalization. At 2 months follow up in clinic, the patient was asymptomatic.
Discussion: Biliary NETs are found in the common hepatic duct, CBD and the cystic duct.[3] Biliary NETs are twice more common in females, with a mean age of 47 years old. Although most cases are incidental, presenting symptoms are jaundice and pruritis. Diagnosis requires histologic evidence and positive expression of chromogranin A, synaptophysin, CD56, or neural cell adhesion molecule.[4] Treatment is surgical excision with Roux-en-Y hepaticojejunostomy. Since biliary NETs uncommonly present with obstructive jaundice, this case presentation highlights the need for a broad differential diagnosis in evaluating patients with obstructive jaundice.

S1771

Non Obstructive Cholangitis with EBV (Epstein Barr Virus) Hepatitis

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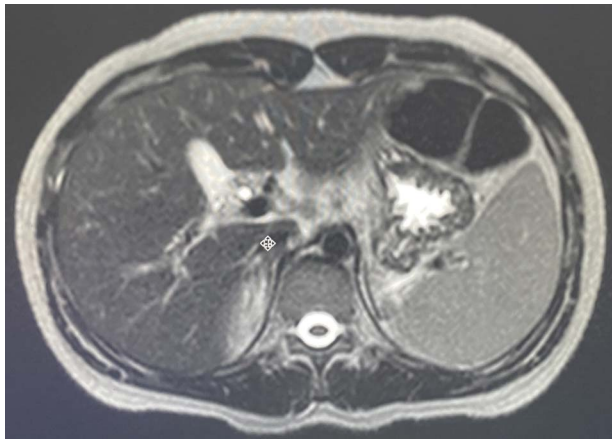
Introduction: The incidence of EBV causing scleral icterus and cholestasis is less than 5%. In literature, the presence of EBV cholangitis in the absence of underlying cause is unknown. We report a case of EBV associated nonobstructive acute cholangitis.
Case Description/Methods: A 21 year old female with history of endometriosis and spina bifida occulta presented with right upper quadrant and epigastric abdominal pain for 4 days. It was non-radiating, colicky with nausea and vomiting. She had fevers and chills with yellow discoloration of her skin, eyes and urine. Vitals were stable. The physical exam was significant for jaundiced skin and scleral icterus with right upper quadrant tenderness and negative Murphy's sign. On admission, lab results showed hemoglobin of 14.1 g/dl, White blood cell count of 8.5 K cells/ml, and hyperbilirubinemia (5.8). Liver enzymes including aspartate transaminase, alanine transaminase and alkaline phosphatase were elevated at 630, 823 and 355 respectively. Lipase was normal. Abdominal ultrasound showed a severely contracted gallbladder with a thickened wall, and small pericholecystic fluid. MRI revealed inflammation in periportal/peribiliary area, no gallstones and with gallbladder hydrops (Figure). The CBD was 5 mm with no intra or extrahepatic biliary dilation. Lab values were only positive for mononucleosis and EBV DNA PCR which showed 2800 copies/mL. No surgical intervention was performed. The patient was treated supportively with analgesics and anti-emetics.
Discussion: We report the first case of primary EBV infection complicated by acute cholangitis. The pathogenesis of EBV associated cholangitis still remains unclear. Our patient imaging showed no stricture or dilatation in the CBD. In 2020, Colbran & Ng reported a case of EBV hepatitis presenting as ascending cholangitis in an immunocompetent patient. Similar to our case, their patient presented with Charcot's triad; however, our patient had peribiliary inflammatory changes which was not seen in their patient. EBV is capable of decreasing the production of pro-inflammatory cytokines which can interfere with the activity of the bile transport systems and cholestasis could be due to abdominal lymphadenopathy. Thus, for a young patient with acute cholangitis, in the absence of a clear etiology for jaundice, physicians need to be vigilant in ordering EBV workup in order to avoid the overuse of antibiotics, decrease the rate of antibiotic resistance and avoid unnecessary invasive procedures.



[1771] **Figure 1.** MRI showing Peribiliary/periportal inflammation.

S1772

Neurofibromatosis-Related Biliary Obstruction: Is There a Role in Screening for Biliary Malignancy?

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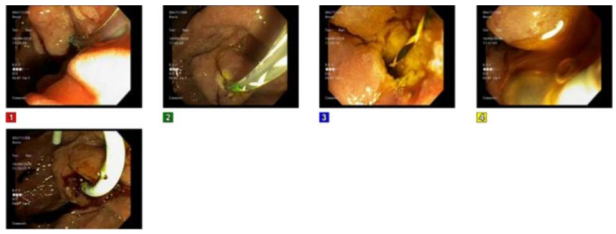
Introduction: Neurofibromatosis-1 (NF1) has known associations with numerous malignancies including breast, bone, thyroid, liver, lung, colon, ovary, esophagus, non-Hodgkins lymphoma, rhabdomyosarcoma, leukemia, and CNS tumors. Other than early initiation of breast cancer screening, there are no other specific cancer screening guidelines for these patients compared to the general population, despite the known high-prevalence of concomitant tumors in these patients. Up to 25% of NF1 patients can have GI tract involvement, but only 5% are symptomatic. Biliary involvement has been seen from periampullary neuroendocrine tumors and gastrointestinal stromal tumors. Biliary obstruction and biliary strictures from ganglioneuromas are rare.

Case Description/Methods: A 63-year-old female with NF-1 who presented with a 5-month history of abdominal pain, nausea, and 40-lb weight loss was found to have ileocecal thickening and intra/extrahepatic biliary dilation on imaging. CEA & CA19-9 were normal. EGD showed multiple large polyps in the second portion of the duodenum. ERCP, as shown in Figure, was notable for a periampullary mass causing biliary obstruction and a mid-CBD stricture that was biopsied and stented across. Pathology revealed ganglioneuroma, and was negative for malignancy. After ileocecal thickening was found to be colon adenocarcinoma, she underwent right hemicolectomy and chemotherapy. Subsequently, she has had recurrent episodes of cholangitis related to periampullary obstruction and numerous biliary stents. EGD/ERCP at 1 year showed villous appearance of the ampulla with surrounding congestion. An irregular biliary stricture was again appreciated with pathology showing ganglioneuroma.
Discussion: NF-1 patients with periampullary or biliary tract masses have the potential for malignant transformation. There are no screening guidelines to facilitate early detection of tumor progression in this population. Patients with neurofibromatosis-like findings should also be considered to have other potential underlying genetic mutations, including constitutional mismatch-repair deficiency, which increases the risk of malignant transformation ~80%. Our patient had numerous ERCPs over one year. Although last ERCP had a villous appearance of her periampullary mass, biopsy remained negative for malignancy. She will undergo repeat sampling and stent removal in a few months, where biopsy will be repeated. We suggest the need for biliary imaging/sampling to screen for malignant transformation in similar patients.



[1772] **Figure 1.** ERCP images demonstrating periampullary mass, and CBD stricture.

S1773

Mind the Gap: A Case of Autoimmune Pancreatitis

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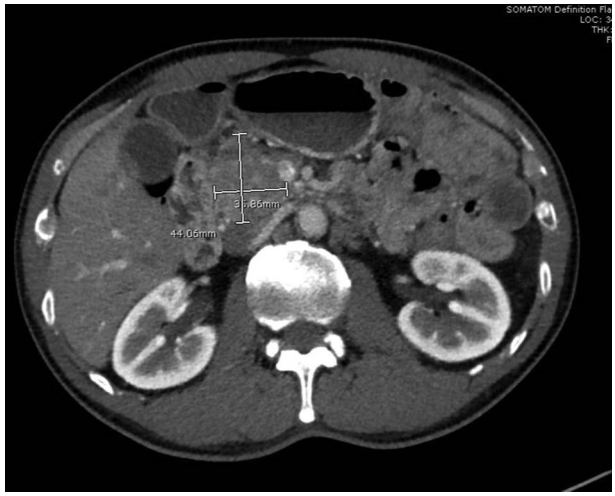
Introduction: Autoimmune pancreatitis (AIP) classically presents as painless jaundice, similar to its mimicker pancreatic cancer, but it can also present in other manifestations to include abdominal pain, weight loss, steatorrhea or even cholangitis. The diagnosis and distinction between AIP and pancreatic cancer is often very difficult even beyond clinical presentation and despite utilization of laboratory analysis and imaging, it is estimated that around 30% of AIP cases require core biopsy, steroid trial, or surgery to make definitive diagnosis. Additionally, the high mortality associated with pancreatic cancer without treatment can spear-head more aggressive treatment.
Case Description/Methods: We present a case of a 63-year-old male who underwent workup for postprandial abdominal pain and mild elevation of liver associated enzymes with normal bilirubin. He then had an MRI, which showed an irregular pancreatic head mass suspicious for malignancy that was further analyzed via EUS and biopsy showing atypia but no definite evidence of adenocarcinoma (Figure). Tumor markers showed elevation of CA 19-9. He was referred to an oncologist and the decision was made that this was most likely pancreatic cancer. He was initiated on modified folfirinox and radiation therapy which led to reduction in size of the mass on repeat imaging. The patient eventually underwent pancreaticoduodenectomy. Labs after his surgery did not improve and actually worsened in both cholestatic and hepatocellular patterns. The specimen demonstrated storiform fibrosis and lymphoplasmacytic infiltrate consistent with HISORt type 1 AIP as well as positive IgG4 staining. After referral to tertiary care center GI, an IgG4 level was found significantly elevated, consistent with AIP in addition to autoimmune cholangiopathy. He had a robust response to steroids and eventually transitioned to rituximab with significant improvement in his clinical and biochemical markers.
Discussion: The persistent elevation in this patient's protein gap likely represented his elevated IgG4 level and may have been a clue to the diagnosis well before this patient's interventions (Table). Ultimately, diagnosis is made with the HISORt criteria. It has been proposed utilizing a combination of IgG4 and CA 19-9 to distinguish AIP from pancreatic carcinoma with CA19-9 < 74 and IgG4 >1.0 showing a 100% PPV and a 96% NPV. This case demonstrates the importance of considering AIP during evaluation of pancreatic head mass to avoid delayed diagnosis, unnecessary treatment and patient harm.



[1773] **Figure 1.** Pancreatic head mass demonstrated on CT abdomen.

Table 1. Labs at time of discovery of pancreatic mass on imaging

Total Protein	8.5
Albumin	4.4
Alkaline Phosphatase	434
Aspartate Aminotransferase	1052
Alanine Aminotransferase	972
Total Bilirubin	1.8

Table 1. (*continued*)

Direct Bilirubin	0.7
IgG Subclass 4 (obtained post Whipple)	688
CA 19-9 (obtained pre Whipple)	81

S1774

Not All Gastroparesis Is Gastroparesis

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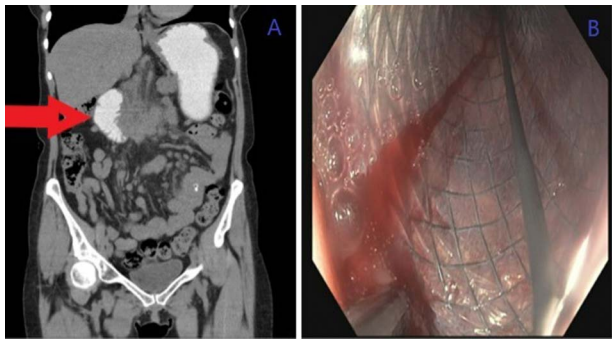
Introduction: Gastroparesis is delayed gastric emptying in the absence of mechanical obstruction. Over 4 million people in the US suffer from the disease with the most common etiology being idiopathic followed by diabetes. Mimickers of gastroparesis need to be ruled to appropriately treat the patient. We present one such case of what was originally thought to be gastroparesis but found to have pancreatic adenocarcinoma causing small bowel obstruction.
Case Description/Methods: A 62-year-old female with a past medical history of recurrent gallstone pancreatitis status post cholecystectomy presented with symptoms of nausea, vomiting, bloating, reflux, and 22lb weight loss. A gastric emptying study done at a local hospital showed 95% retention after 4 hours. She underwent a feeding jejunostomy with venting. Because of presumed persistent gastroparesis, and repeated hospitalization she was scheduled for gastric pacemaker placement at outside facility. Patient sought a second opinion in our motility clinic. Physical exam was significant for a succussion splash. A CT abdomen showed an ill-defined soft tissue structure centered at the pancreaticoduodenal groove with associated duodenal stenosis (Figure A) – indicating groove pancreatitis. EGD showed complete obstruction of the second portion of the duodenum. Initial stricture biopsies were benign. Following this, balloon dilation was performed and a 20 mm X 12cm fully covered metal stent (Figure B) was placed. Patient however continued to report symptoms of nausea and vomiting with new onset of jaundice. Repeat CT abdomen demonstrated distal CBD obstruction with upstream dilation. Patient underwent PTC drain placement, and CBD brush biopsy obtained demonstrated non-viable cells without evidence of malignancy. Labs revealed normal lipase and IgG4 levels. Given non-resolution of pancreatic inflammation, to rule out malignancy, it was decided to perform EUS with FNA which showed pancreatic head adenocarcinoma with duodenal infiltration.
Discussion: Groove pancreatitis affects the anatomical area between the head of the pancreas, duodenum, and common bile duct. Common complications include biliary stricture with obstructive jaundice and duodenal stricture. In our case, the duodenal stricture caused secondary gastroparesis. Groove pancreatitis and pancreatic adenocarcinoma are challenging to differentiate with similar presentation. It is important to differentiate a benign from a malignant etiology as treatment and prognosis varies significantly.



[1774] **Figure 1.** (A) – Red arrow shows the area of abrupt duodenal narrowing with proximal duodenum and stomach showing opacification with oral contrast. (B) - 20 mm wide X 12 cm long fully covered metal stent at the site of duodenal stricture.

S1775

Lower Gastrointestinal Bleeding From a Cystic Artery Pseudoaneurysm in the Setting of Cholecystocolonic Fistula and Gangrenous Cholecystitis

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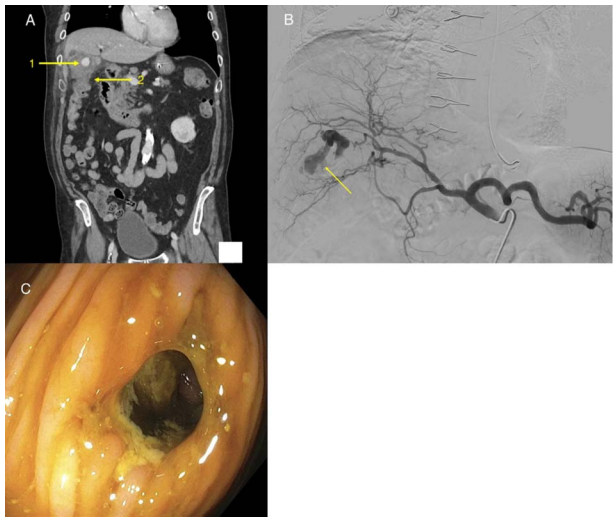
Introduction: Cholecystocolonic fistula (CCF) is an uncommon complication of cholecystitis in which a tract is formed between the inflamed gallbladder and the colon. CCF are usually asymptomatic and are discovered intraoperatively in < 1% of cholecystectomies.[1-2] Herein we discuss an atypical presentation of a CCF.
Case Description/Methods: A 78-year-old male with type II diabetes, coronary artery disease, and a 20 pack-year smoking history presented with 2 days of progressive hematochezia, dyspnea, and weakness following 6 hours of periumbilical pain. On admission, he was hemodynamically unstable with a blood pressure of 66/34, pulse of 95 BPM, and hemoglobin of 6.2 g/dL. CT abdomen/pelvis revealed an inflamed gallbladder with a hematoma, a pseudoaneurysm, and a CCF to the hepatic flexure (Figure A). Mesenteric angiogram confirmed a pseudoaneurysm of the cystic artery with active bleeding (Figure B). Colonoscopy confirmed an 8mm circular tract at the hepatic flexure (Figure C). Upper endoscopy was normal. The patient was diagnosed with gangrenous cholecystitis with CCF and was treated with massive transfusion protocol followed by cystic artery embolization, which successfully halted the hematochezia. The patient was eventually discharged with plans for outpatient cholecystectomy and fistula resection.
Discussion: CCFs are most often found in the setting of biliary disease. The tracts may be formed secondary to stone impaction and subsequent pressure necrosis of the gallbladder lumen in uncomplicated cholecystitis or due to gallbladder wall distension and ischemic necrosis in gangrenous cholecystitis. 85% of these fistulas are found between the gallbladder and duodenum, and only 15% involve the colon.[3] CCF is treated with cholecystectomy and fistula resection. While once considered a contraindication to laparoscopic surgery, laparoscopic techniques are now commonly used to treat CCFs with similar success rates and decreased rates of postoperative complications and hospital stays when compared to open procedures.[4]



[1775] **Figure 1.** Contrast enhanced coronal CT scan of the abdomen and pelvis revealed an inflamed gallbladder with a hematoma and pseudoaneurysm (arrow 1) and a fistula from the gallbladder to the hepatic flexure (arrow 2) (a). Mesenteric angiogram confirmed a pseudoaneurysm of the cystic artery with active bleeding (b). Colonoscopy confirmed an 8mm circular tract at the hepatic flexure (c).

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S1776

Pancreatic Stones, Abdominal Groans, and Encephalopathic Overtones Presented by a Type 3c Diabetes and Familial Hypocalciuric Hypercalcemia Duet

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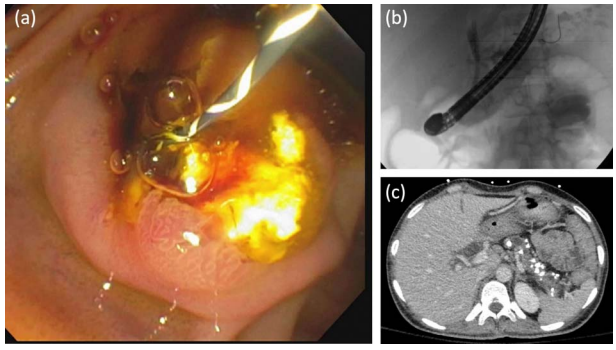
Introduction: Chronic pancreatitis related to hypercalcemia may occur in genetic conditions such as Familial Hypocalciuric Hypercalcemia (FHH). Recurrent episodes of hypercalcemia-induced acute pancreatitis result in chronic pancreatitis, marked by pancreatic edema, necrosis, and fibrinogenesis. Progression of pancreatic dysfunction to endocrine insufficiency results in pancreatogenic (type 3c) diabetes, present in 0.5-1% of all patients with diabetes. We present a rare case of FHH induced chronic pancreatitis, complicated by type 3c diabetes.
Case Description/Methods: A 24-year-old female with history of insulin-dependent diabetes mellitus (IDDM) presented with acute encephalopathy, right upper quadrant abdominal pain, and oliguria. Patient was diagnosed with IDDM at a young age after several episodes of recurrent acute pancreatitis of unknown etiology. Her mother and maternal grandmother had history of recurrent acute pancreatitis. Patient was diagnosed with sepsis secondary to gram negative bacteremia suggestive of cholangitis, with elevated ionized calcium of 1.83. Hypercalcemia evaluation revealed fractional excretion of calcium of $< 1\%$, diagnostic of FHH. CT scan revealed a dilated common bile duct (CBD) up to 10 mm without stones, absence of cholelithiasis, diffuse pancreatic calcifications, and a dilated main pancreatic duct (PD) up to 12 mm, consistent with chronic pancreatitis. Endoscopic retrograde cholangiopancreatography revealed a large pancreatic duct stone lodged at the ampulla (Figure). Cholangiogram showed a dilated biliary tree without filling defects. The PD was severely dilated and contained sludge and stones. Dual sphincterotomy was performed with retrieval of pancreatic duct stones only. Patient met criteria for type 3c diabetes when further workup revealed negative anti-glutamic acid decarboxylase antibodies.
Discussion: FHH is a rare, benign cause of hypercalcemia, characterized by inactivating mutations of the calcium-sensing receptor (CASR). Via CASR, sustained hypercalcemia prematurely activates trypsinogen and depletes ATP, leading to pancreatic inflammation and necrosis. The role of CASR in pathogenesis of recurrent acute and chronic pancreatitis has been debated in several clinical reviews. Type 3c diabetes is a consequence of chronic pancreatitis and presents at higher rates in patients with early onset calcific pancreatic disease. Our case highlights a rare etiology of recurrent acute and chronic pancreatitis.



[1776] **Figure 1.** Hypercalcemia-induced chronic pancreatitis evidenced by (a) ERCP with evidence of pancreatic duct stone lodged in ampulla, (b) cholangiogram with biliary tree dilation, and (c) CT with diffuse pancreatic calcifications.

S1777

Impacted Pancreatic Stone Resulting in Biliary Obstruction, Choledochal Duodenal Fistula Development, and Ascending Cholangitis

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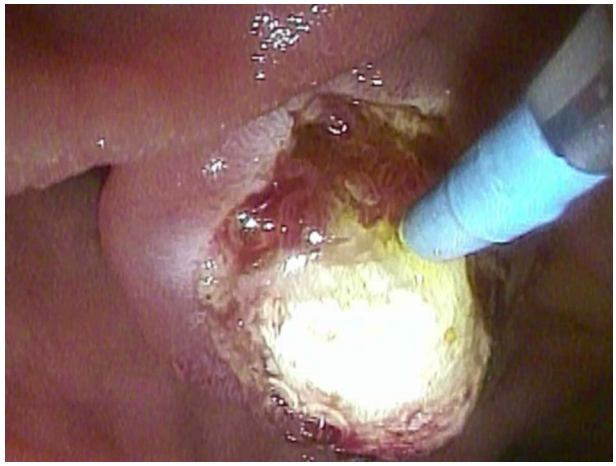
Introduction: Pancreatic stones are present in about one-half of patients with chronic pancreatitis (CP). Pancreatic stones are associated with alcohol, smoking, metabolic and hereditary factors. The most common complication of pancreatic stones is acute pancreatitis. Pancreatic stones can lead to pancreatic fistulas and/or biliary obstruction when associated with bile duct strictures. Stones are typically managed by surgery, endoscopy, and extracorporeal shock wave lithotripsy. We present a case of an obstructive pancreatic stone complicated by ascending cholangitis and a choledochal duodenal fistula (CDF).
Case Description/Methods: A 69-year-old male with a history of a remote cholecystectomy, alcohol abuse, and CP presented with abdominal pain and vomiting. On arrival he was hypotensive and febrile. Blood work was significant for leukocytosis, elevated liver enzymes, and Klebsiella bacteremia. Computed tomography demonstrating intra and extrahepatic bile duct (BD) dilatation, diffuse pancreatic calcifications, and a dilated pancreatic duct. Given concern for ascending cholangitis the patient was started on antibiotics and underwent urgent endoscopic retrograde cholangiopancreatography (ERCP). Endoscopy revealed a bulging major papilla consistent with and an impacted stone and obvious biliary drainage from a fistula proximal to the papillary orifice. Precut biliary sphincterotomy was performed revealing a 12mm impacted pancreatic stone (Figure). A pancreatic septotomy was then performed resulting in spontaneous stone passage. Plastic stents were placed across both ducts to ensure drainage. Post procedure the patient improved with resolution of jaundice and abdominal pain.
Discussion: Obstructive jaundice and enteric fistulas are known complications of bile duct stones. However, there are limited reports describing obstructive jaundice secondary to pancreatic stones. To our knowledge, this is the first report of a chronic pancreatitis complication whereby pancreatic stone impaction led to chronic biliary obstruction but also with ascending cholangitis and fistula development. This case highlights a rare, but potentially dangerous complication of a large pancreatic stone that led to biliary obstruction and cholangitis that was successfully managed with ERCP.



[1777] **Figure 1.** Impacted Pancreatic Stone Exposed After Biliary Sphincterotomy.

S1778

A Striking Case of Mucinous Gallbladder Adenocarcinoma Mimicking Mirizzi's Syndrome

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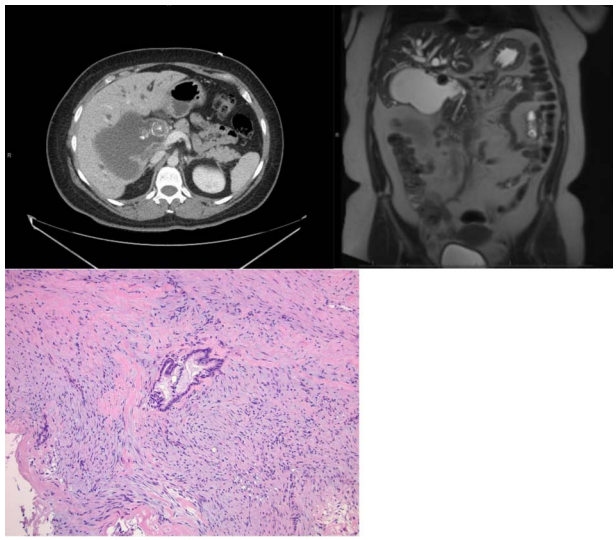
Introduction: Gallbladder carcinoma is the most common biliary malignancy in the United States. Here we describe a presentation of a mucinous adenocarcinoma of the gallbladder resulting in prominent gallbladder distension and biliary obstruction, which clinically imitated Mirizzi's syndrome, a common hepatic duct obstruction caused by an extrinsic compression from an impacted stone in the cystic duct or gallbladder neck.
Case Description/Methods: A 49-year-old woman with no significant past medical history presented with 10 days of jaundice and a sharp, intermittent right upper quadrant pain. Vitals were within normal limits. Labs were significant for elevated aminotransferases, alkaline phosphatase, and bilirubin (Table). CT and MRCP [Figure [top]) revealed gallbladder distension with 2 large gallstones at the gallbladder neck, as well as significant dilatation of the intra and extrahepatic ducts with abrupt cut off of the common hepatic duct at the level of the adjacent gallstones. She underwent endoscopic retrograde cholangiopancreatography (ERCP), during which sphincterotomy revealed thick mucus, multiple calculi (up to 1 cm), and sludge. Biliary stents were placed in the CBD. Due to persistently elevated liver chemistries, she underwent cholecystectomy a week later where an inflamed gallbladder was encountered. Total cholecystectomy was unsuccessful due to significant scarring. Retrieved surgical specimen revealed a 1cm gallbladder wall tumor and pathology demonstrated a well-differentiated invasive mucinous adenocarcinoma, stage pT1b Figure [bottom]). Chemotherapy was initiated. Twelve months later, she passed due to complications from abdominal compartment syndrome due to large volume malignant ascites from omental metastases.
Discussion: Given the paucity of sensitive signs and testing, diagnosis of gallbladder adenocarcinoma is usually incidental or at an advanced stage. In this case, the presence of stones confounded the diagnosis as brushings were negative and the patient met clinical criteria for Mirizzi's syndrome: right upper quadrant pain with jaundice, dilatation of the biliary system above the gallbladder neck, and a stone in the gallbladder neck. The treatment of choice in respectable cases of mucinous adenocarcinoma of the gallbladder is radical cholecystectomy. Overall, the prognosis is poor as is seen in this case. In the largest series documenting outcomes of 15 cases, the 3-year survival rate was 1% versus 39% seen in the comparator group with conventional gallbladder adenocarcinoma.



[1778] **Figure 1.** Top: CT and MRCP showing gallbladder distension. Bottom: Pathology showing invasive mucinous adenocarcinoma.

Table 1. WBC = White blood cells Hgb = hemoglobin BUN = blood urea nitrogen AST = aspartate transaminase ALT = alanine transaminase INR = international normalized ratio

	Admission	Normal Range
WBC	7.1 K/mcL	3.4 - 11 K/mcL
Hgb	12.6 g/dL	11.9 - 15.3 g/dL
Platelets	182 K/mcL	150 - 425 K/mcL
Sodium	142 mmol/L	137 - 145 mmol/L
Potassium	3.9 mmol/L	3.5 - 5.1 mmol/L
Chloride	104 mmol/L	98 - 107 mmol/L
CO2	25 mmol/L	22 - 30 mmol/L
BUN	13 mg/dL	7 - 17 mg/dL
Creatinine	0.6 mg/dL	0.5 - 1.0 mg/dL
Glucose	113 mg/dL	70 - 125 mg/dL
Albumin	4.2 g/dL	3.5 - 5.0 g/dL
Total protein	**8.6 g/dL**	6.3 - 8.2 g/dL
Total bilirubin	**9.0 mg/dL**	0.2 - 1.3 mg/dL
Direct bilirubin	**7/5 mg/dL**	0.0 - 0.4 mg/dL
AST	**106 U/L**	14 - 36 U/L
ALT	**246 U/L**	< 34 U/L
Alkaline phosphatase	**394 U/L**	38 - 126 U/L
INR	1.0 seconds	0.9 - 1.1 seconds
Lactate	1.0 mmol/L	0.7 - 2.1 mmol/L

S1779

A Peculiar Case of Jaundice and Renal Failure After Hurricane Ida

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Introduction: Leptospirosis is a zoonotic disease seen in tropical and temperate regions, with roughly 1.03 million cases worldwide and 58,900 deaths annually. Humans are infected when exposed to the tissue or urine of colonized rodents, cattle, swine, dogs, or sheep. The symptoms are nonspecific but include fever, myalgia, and rigors. Laboratory findings in severe cases include hyponatremia, elevated hepatic enzymes, renal failure, and thrombocytopenia. Transaminases are typically mildly elevated, but total bilirubin can increase to as high as 80 mg/dL. The associated jaundice is thought to be due to hepatocellular necrosis, intrahepatic cholestasis, and absorption of tissue hemorrhage. In severe cases of jaundice and renal failure, "Weil's Disease" is diagnosed.
Case Description/Methods: Hurricane Ida produced record rainfall in the northeastern US over an 18 hour period. Five days following the storm, a 52 year old male with no significant past medical history, presented to our ER complaining of fever, night sweats, chills, fatigue, and generalized weakness. For work, he had been tasked to clear pools of stagnant water from normally dry areas of urban parks. On presentation, his blood pressure was 136/68 mmHg, heart rate 159 bpm, temperature 36.4 degrees C, and oxygen saturation of 95% on room air. His exam was notable for scleral icterus, jaundice, and tachycardia. The abdomen was soft, non-tender and non-distended, and without hepatomegaly. Laboratory values are given in the Table. The patient's renal failure subsequently worsened with BUN/creatinine reaching 114/7.48 mg/dL. Abdominal ultrasound and MRCP demonstrated no abnormalities. On day 4 urine DNA for *Leptospira* was positive, though serum *Leptospira* PCR was negative. He was started on doxycycline and ceftriaxone with improvement in his symptoms, jaundice and renal failure.
Discussion: *Leptospira* is endemic in mostly southern and Pacific coastal areas of the US, as well as Hawaii. In developed countries, leptospirosis is commonly seen after heavy rainfall and floods. Over the past 5 years, locally acquired cases of leptospirosis have surpassed travel associated infection in our local jurisdiction. Climate studies have pointed to a positive correlation between cases of leptospirosis and significant changes in climate and associated flooding. Given the potential severity of leptospirosis, community education to avoid stagnant water and barrier coverage in emerging Leptospirosis hotspots could be beneficial in the prevention of disease.

Table 1. Relevant Labs from Admission

Hemoglobin	14.3 g/dL
Platelets	68,000 u/L
MCV	84 fL
ESR	58 ml/Hr
BUN / Cr	54 mg/dL / 2.9 mg/dL
Sodium	129 mmol/L
Total Bilirubin	6.7 mg/dL
Direct Bilirubin	5.08 mg/dl
ALT	78 unit/L
AST	191 unit/L
Alkaline Phosphatase	102 unit/L
CPK	3,600 unit/L
Lipase	2.3 unit/L
Hepatitis A,B,C serology	evidence of prior HBV vaccination
Ds-DNA	within normal limits
Immunoglobin A,G,E,M	within normal limits

S1780

Immunoglobulin G4-Associated Cholangitis Mimicking Cholangiocarcinoma in Elderly

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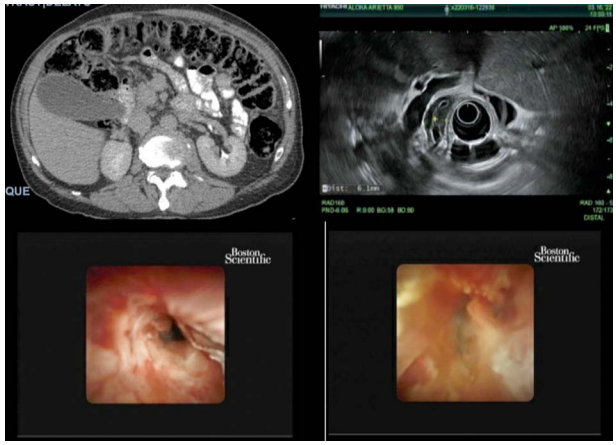
Introduction: Sclerosing cholangitis is a diffuse inflammation and fibrosis that progressively leads to stenosis and destruction of the bile ducts. It includes 3 types: primary sclerosing cholangitis (PSC), secondary cholangitis, and IgG4 sclerosing cholangitis (IgG4-SC). Usually they all present with cholestatic symptoms like pruritus and abdominal pain along with elevated ALP and GGT as well as bilirubin, however IgG4-SC has better outcomes, treatment response to steroids and less recurrence. We hereby presenting a case of IgG4-SC presenting as cholangiocarcinoma.
Case Description/Methods: An 80 year-old male with a past medical history of bladder cancer was referred to the gastroenterology for EUS after blood work showed (ALT 320, AST 297, Bilirubin 1.4, GGT 2032), and CT abdomen with IV contrast revealed intra and extrahepatic biliary dilatation with an abrupt cutoff in mid CBD, suspicious for either a primary CBD mass or pancreatic mass (Figure). EUS showed an irregular mass in the CBD wall, suspicious for cholangiocarcinoma (Figure). The ERCP showed malignant appearing stricture of the extrahepatic bile duct consistent with cholangiocarcinoma, Bismuth 2 vs early 3B (Figure). Exploratory laparotomy was done and portal node biopsy came back benign; for which a repeat ERCP/Spyglass with direct visualization cholangioscopy, was done and showed diffusely dilated main bile duct with sludge and pus, single diffuse stenosis in right hepatic duct sludge with lining malignant appearing abnormal mucosa (nodularity and dilated vessels). IgG4 was elevated (931) consistent with IgG4 cholangitis (Figure). Biopsy and cytology brush showed no signs of dysplasia or neoplasia. Patient was then started on prednisone for a month and scheduled to repeat ERCP/Spyglass after.
Discussion: IgG4-RD is a fibrous-inflammatory process related to immunomodulation, therefore IgG4-SC often co-presents with auto-immune pancreatitis, dacryoadenitis and sialadenitis. IgG4-SC is extremely challenging, as it's frequently misdiagnosed on imaging as a pseudotumor, and can progress to liver cirrhosis if left untreated, however it comes with great response to steroids. In 2021, Mendoza criteria (tortuous vessels, irregular nodulations, raised intraductal lesions, irregular surface, and friability) was published and found to be accurate to differentiate malignant and benign causes of biliary stricture, with overall diagnostic accuracy of 77%. Our case didn't meet the 5 criterias of Mendoza, which is consistent with IgG4-SC.



[1780] **Figure 1.** Top: CT of abdomen and ERCP. Bottom: Exploratory laparotomy.

S1781

Caseating Granulomas Caused by Dropped Gallstones Post Cholecystectomy

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Introduction: We present an interesting case of a patient with a history of cholelithiasis with cholecystectomy found to have caseating granulomatous lesions in the liver and peritoneal cavity, ultimately found to have "drop gallstone." With laparoscopic cholecystectomy becoming the standard of treatment, this case illustrates the importance of having high suspicion for this diagnosis and its complications, as it can lead to unnecessary diagnostic testing to rule out autoimmune or malignant mimics.

Case Description/Methods: A 74-year-old male with remote history of metabolic co-morbidities, cholelithiasis status post cholecystectomy, and spinal cord injury complicated by neurogenic bladder who presented with generalized weakness, found to have leukocytosis and electrolyte abnormalities on admission at outside hospital. Computed tomography of abdomen and pelvis scan showed several low-density lesions in the liver and scattered indistinct nodules throughout the peritoneal cavity and abdominal wall musculature. Biopsy results showed caseating granulomatous inflammation and fibrinoid necrosis concerning for an autoimmune or malignant process. However, autoimmune and oncologic work-up were overall unremarkable. Upon transfer to our hospital, secondary pathology review of the biopsies additionally noted bile acid casts and refractile material consistent with gallstones, diagnosing him with "dropped gallstone."
Discussion: Stones or biliary leakage into the abdominal cavity at the time of a laparoscopic cholecystectomy can lead to complications including chronic inflammation that can mimic malignancy or autoimmune disease. Thorough irrigation and evacuation of the gallbladder fossa and abdominal cavity during the procedure can help minimize this complication. High clinical suspicion for "drop gallstone" can minimize unnecessary diagnostic testing for the patient.

S1782

Tamoxifen-Induced Hypertriglyceridemia Causing Acute Pancreatitis

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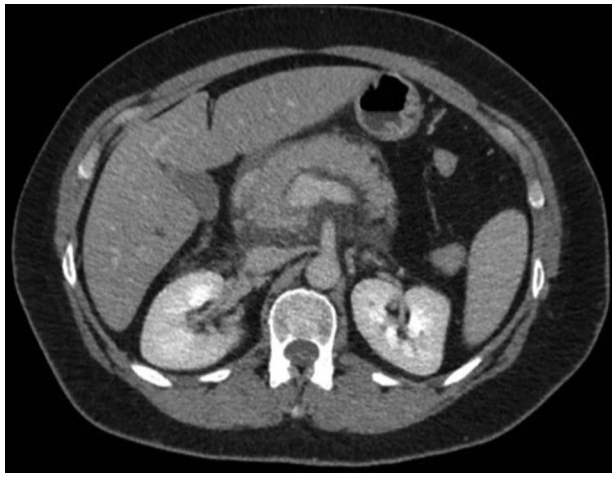
Introduction: Acute pancreatitis is an inflammatory disease that can be associated with significant morbidity and mortality. Common causes include gallstones, significant alcohol use, and hypertriglyceridemia. Many medications have been known to cause hypertriglyceridemia including tamoxifen, a selective estrogen receptor modulator. We present a rare case of acute pancreatitis caused by tamoxifen-induced hypertriglyceridemia.
Case Description/Methods: A 53-year-old female presented to the ED with sharp abdominal pain with radiation to her chest. Lab workup revealed an elevated lipase at 169 U/L. CT of the abdomen and pelvis showed acute inflammatory changes at the head of the pancreas (Figure). A triglyceride level was obtained showing a level >1000 mg/dL. The patient was then started on an insulin drip and admitted to the ICU for further monitoring. The patient had been diagnosed with breast cancer about 5 years prior to admission and had been taking tamoxifen during that time. Chart review revealed a triglyceride level performed in the outpatient setting a year prior with a result greater than 4000 mg/dL. The patient reported that this was determined to be a lab error and was left untreated. Her pain and triglycerides decreased after IV fluids and insulin drip. Tamoxifen was discontinued and the patient was started on fenofibrate and atorvastatin.
Discussion: Diagnosis of acute pancreatitis is based on the Revised Atlanta Criteria which should include 2 or more of the following symptoms: epigastric pain, serum lipase >3 times the upper limit of normal, and/or characteristic findings on abdominal imaging. Further testing should be done if no clear etiology. Treatment includes aggressive IV fluids, analgesia, and directed therapy based on etiology. Hypertriglyceridemia causing acute pancreatitis accounts for about 1%-4% of cases. Elevation in triglycerides can cause sludging of pancreatic vasculature and increasing activity of pancreas lipases leading to cytotoxic injuries. Tamoxifen is a selective estrogen receptor modulator and is thought to increase the synthesis of triglycerides and very low-density lipoproteins. It is also proposed to decrease the activity of lipoprotein and triglyceride lipase. Treatment of hypertriglyceridemia includes discontinuing the offending agent with insulin drip or plasmapheresis. This case suggests that those on tamoxifen should have regular monitoring of serum lipids and treat any abnormal lab value as significant until proven otherwise.



[1782] **Figure 1.** Inflammatory change in the pancreatic head region is suggestive of acute pancreatitis.

S1783

Noncirrhotic Portal Hypertension and Pancreaticogastric Fistula Complicating Acute Pancreatitis With Fluid Collections

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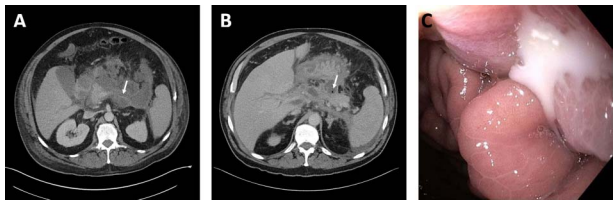
Introduction: Noncirrhotic portal hypertension constitutes an increase in pressure of the portal venous system in the absence of cirrhosis. This is a rare condition; however, it is insufficiently acknowledged given its decreased prevalence in North America and Europe compared to other nations. We present a rare case of noncirrhotic portal hypertension and pancreatico-gastric fistula complicating acute pancreatitis with an acute fluid collection and resultant portal vein narrowing.
Case Description/Methods: A 55-year-old man with history of alcohol use disorder presented to the emergency department with worsening abdominal pain 2 weeks after discharge for uncomplicated acute pancreatitis. Lipase was 48 IU/L (normal 11-82 IU/L). CT abdomen showed increasing peripancreatic fluid collection with marked narrowing of the main portal vein, which appeared near threadlike, and narrowing of the bifurcation of the portal vein extending into the left portal vein. The splenic vein was also severely narrowed (Figure A). He was discharged home with outpatient follow-up, however, returned to the ED for intractable pain one month later. A CT abdomen showed florid changes of pancreatitis with pseudocyst formation with new foci of air within the collection concerning for fistulous tract formation with the gastric lumen (Figure B). The portal vein and splenic vein narrowing improved, but the narrowing of the right and left portal veins worsened. He underwent upper endoscopy which showed a small area of White "milky" base in the posterior wall of the incisura angularis which was suggestive of fistulous opening in the area (Figure C). Four columns of small distal esophageal varices were also noted. During admission, paracentesis was performed for ascites. Serum ascites albumin gradient was over 1.1 consistent with ascites due to portal hypertension – likely due to the portal vein narrowing. Patient was discharged in stable, improved condition.
Discussion: Although portal hypertension normally presents in cirrhotic patients, it may rarely present in the absence of cirrhosis by conditions such as portal vein thrombosis or portosinusoidal vascular disease. Compression of the portal vein from an acute pancreatic fluid collection is an extremely rare cause of noncirrhotic portal hypertension. Clinicians should have a high index of suspicion for complications related to portal hypertension such as ascites or varices in patients with a prolonged course of acute pancreatitis without cirrhosis.



[1783] **Figure 1.** (A) Contrast CT abdomen showing marked narrowing of the main portal vein, which appears near threadlike. (B) One of 2 foci of air in the collection at the anterior superior aspect of the pancreatic body. Appears inseparable from the posterior wall of the gastric body, concerning for fistulous involvement. (C) Area of White "milky" base in the posterior wall of the incisura angularis. This area was rinsed, however re-accumulated despite no obvious opening seen.

S1784

IgG4-Related Autoimmune Pancreatitis Following RNA-Based COVID-19 Vaccination

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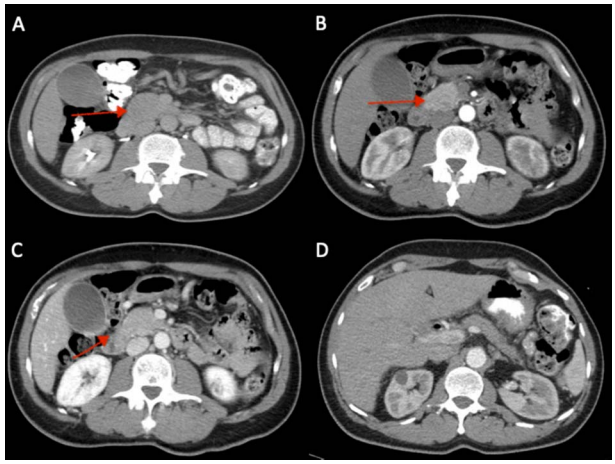
Introduction: Autoimmune pancreatitis type 1 is a manifestation of IgG4-related disease and can often mimic tumor-like masses. Currently, there are no cases in which IgG4-RD involving the hepatobiliary system has been reported following the COVID-19 vaccination. We present the first case of AIP type 1 to be associated with the mRNA-based COVID-19 vaccination.
Case Description/Methods: A 63-year-old African-American male was in his normal state until 2021. He received 2 doses of the mRNA-based COVID-19 vaccine in March/April 2021. In June, he presented to his PCP with complaints of fatigue and a rapid 20lb weight loss. He was diagnosed with diabetes mellitus. He denied any significant alcohol intake or history of illicit drugs, hepatotoxic medications, or liver disease. Physical exam was unremarkable. He was started on metformin and repaglinide but his blood sugar remained uncontrolled. In September, he was found to have liver enzyme abnormalities so repaglinide was stopped. However, he had worsening jaundice and developed pruritus. He was then referred to gastroenterology. Routine blood tests revealed mild anemia and elevated transaminases, alkaline phosphatase, total and direct bilirubin. Hepatitis B and C were negative. Ca 19-9 and CEA were normal. IgG and IgG4 levels were elevated to 1703 mg/dL and 679.9 mg/dL, respectively. Contrast enhanced CT abdomen/pelvis revealed a hypervascular arterially enhancing pancreatic head mass measuring up to 5.5cm with moderate intrahepatic biliary dilatation and mild common bile duct dilatation to the pancreatic head (Figure). He underwent an ERCP with biliary stent placement and an endoscopic ultrasound-guided pancreas biopsy. The biopsy specimen was diffusely positive for IgG4 but was "over-stained" and felt to be non-diagnostic. Liver biopsy was not obtained. AIP was suspected given the absence of malignancy on the biopsy, normal Ca 19-9 and CEA levels, and the very elevated IgG4 level. He was started on prednisone 40mg daily with a tapering schedule. Within 2 weeks, his liver enzymes were normal. Imaging obtained at 6 weeks demonstrated resolution of the pancreatic mass with continued normal liver transaminases.
Discussion: Autoimmune phenomena following COVID-19 mRNA vaccination are being increasingly reported. We report the first case of IgG4-RD and AIP type 1 to be associated with the COVID-19 vaccine. Patients with new onset DM or cholestasis with or without signs of obstructive jaundice in the era of COVID-19 vaccination should be screened for IgG4-RD.



[1784] **Figure 1.** (A) Increased fullness to the pancreatic head with a hypoenhancing mass-like lesion measuring up to 3 cm. (B, C) Hypervascular arterial enhancing pancreatic head mass measuring up to 3.6 x 2.6 x 5.5 cm in the largest dimensions (D) Compared to prior studies (previous images), the pancreas is no longer swollen and edematous but appears somewhat atrophic. No definite enhancing masses seen in the head of the pancreas.

S1785

Recurrent Type 2 Autoimmune Pancreatitis and Ulcerative Proctitis

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Introduction: Autoimmune pancreatitis (AIP) is most commonly an IgG4-related disease (type 1 AIP). Type 2 AIP is rare, more common in younger adults, does not typically recur, and is associated with inflammatory bowel disease (IBD). There are no diagnostic serum tests for type 2 AIP and the gold standard is tissue pathology demonstrating neutrophilic infiltration in the pancreatic ducts.
Case Description/Methods: A 33-year-old female presented with recurrent episodes of pancreatitis. She did not have a clear etiology for pancreatitis including no alcohol use, no gallstones or hepatobiliary abnormalities on repeated imaging, normal levels of triglycerides, calcium, and IgG4, and she was not on any offending medications. MRI pancreas had shown an area of abnormal diffusion and delayed relative increased enhancement in the pancreatic tail. Due to recurrent attacks of unclear etiology, she underwent a distal pancreatectomy with splenectomy. Pathology from the distal pancreas showed periductal lymphoplasmacytic inflammatory infiltrates with neutrophilic inflammation in the ductal epithelium and acinar atrophy in majority of large and medium sized pancreatic ducts, and a negative IgG4 stain. These changes were most consistent with type 2 AIP. During this time, she started having bloody diarrhea and colonoscopy showed proctitis with the rest of the colon and terminal ileum appearing normal. Rectal biopsy showed chronic active proctitis. These symptoms improved with topical mesalamine. Despite the pancreatectomy, she continued to have recurrent episodes of pancreatitis with lipase elevations to 300-800, and CT findings of peripancreatic stranding and interstitial edema. She could not tolerate prednisone (side effects of confusion and hallucinations) or azathioprine (developed chest and abdominal pain). In order to manage her abdominal pain, she underwent celiac plexus blocks with only minimal relief. Currently, she is undergoing screening for initiation mycophenolate mofetil or rituximab.
Discussion: Type 2 AIP is a rare and challenging diagnosis, requiring histological confirmation and a multidisciplinary team approach. There is a significant overlap with IBD, predominantly ulcerative colitis, prevalent in up to 15-30% of patients. Concomitant AIP and IBD does not appear to impact disease severity of the other. Relapse is not typical and treatment strategies commonly target both AIP and IBD including steroids, azathioprine, and TNF alpha inhibitors.

S1786

Pancreatic Metastasis Secondary to the Masters of Costumes, Renal Cell Carcinoma

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Introduction: Renal cell carcinoma (RCC) has been one of the most studied cancers, and its metastatic capabilities are well documented in literature. RCC accounts for 80%-85% of primary renal neoplasm. Approximately 25% of individuals have distant metastases at the time of diagnosis. Its metastatic capabilities continue to be one of the most challenging aspects of the disease. Metastasis to the pancreas has been described in about 2%-5% of all malignant pancreatic tumors.
Case Description/Methods: Case of an 80-year-old man with medical history of Stage IV RCC, treated with immunotherapy. Treatment was discontinued due to adverse effects. Patient presents to the ER with a complaint of intractable pruritus. On laboratory values, found with elevated liver enzymes and total bilirubin, with a mixed hepatocellular and cholestatic pattern, reason why gastroenterology service was consulted. Physical examination showed generalized jaundice. Abdominal CT scan showed multiple intrabdominal lesions, including in the pancreas, causing intra and extrahepatic ductal dilation. MRCP showed multiple intrabdominal masses encasing the common bile duct, causing intra and extrahepatic biliary tree and distal pancreatic duct dilation. Concern of a primary pancreaticobiliary malignancy arose. On EGD, multiple gastric and duodenal clean base ulcers and 2 large pedunculated polyps in the second portion of the duodenum removed using hot snare polypectomy technique, and random biopsies on gastric and duodenal ulcers taken. On EUS, a large hyperechoic vascular lesion was observed in the pancreatic head, infiltrating the intrapancreatic bile duct. Another large 3.3 cm x 3.0 cm vascular mass at the gastro-hepatic ligament was observed. Biopsies from duodenal ulcer, duodenal polyp, and pancreatic mass were consistent with metastatic clear cell neoplasm as seen in RCC. ERCP was made to place a fully covered metallic stent at CBD stricture caused by extrinsic compression.
Discussion: RCC is the most common renal tumor. Up to 25% of cases present with advanced disease at diagnosis. Our patient presents with multiple intrabdominal masses causing biliary tree and pancreatic duct obstruction. Advanced endoscopic techniques and cross-sectional images play an essential role in the diagnosis and management of pancreaticobiliary pathologies. Other etiologies such as metastatic RCC may mimic primary pancreaticobiliary malignancies. In this case, advanced endoscopic modalities were used for tissue diagnosis and management of metastatic disease.

S1787

Steroid-refractory IgG4-Related Disease Presenting as Sclerosing Cholangitis

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Introduction: IgG4-related disease (IgG4-RD) is characterized by tumefactive infiltration of IgG4-bearing plasmablasts and other lymphocytes, along with storiform fibrosis, into one or multiple organs, with resultant organ enlargement, fibrosis, and dysfunction. Although initial reports of IgG4-RD concerned involvement of the pancreas, other organs such as the biliary tract can be involved. We present a case of steroid refractory IgG4- RD sclerosing cholangitis (IgG4-SC).
Case Description/Methods: A 67-year-old man with hypertension presented with a 2-week history of unintentional weight loss, painless jaundice, and pruritus. Exam showed jaundice and scleral icterus. Laboratory revealed elevated liver tests with an obstructive pattern, with ALP, GGT, & Bilirubin of 431 U/L, 438 IU/L, 17.6mg/dL, respectively. MRCP, ERCP, and subsequent EUS demonstrated diffuse pancreatic parenchymal hypo-echogenicity, long segment strictures at the distal common bile duct with proximal duct dilation, dilated common hepatic duct, and multiple segmental intrahepatic biliary duct strictures. Biliary sphincterotomy and stent placement resolved symptoms. Biopsy of the Ampulla of Vater revealed chronic inflammation with ulceration. Further work-up uncovered an elevated serum IgG4 of 266 mg/dL with a negative ANA, SMA, AMA, and tumor markers. He was then diagnosed with IgG4-SC and completed a course of prednisone. Relapse of symptoms prompted a repeat ERCP which uncovered biliary tree pruning and irregularity, necessitating biliary stent replacement. Due to poor tolerance with rituximab infusion, he was treated with Mycophenolate Mofetil which accomplished a year of disease quiescence.
Discussion: The prime gastrointestinal manifestations of IgG4-RD include type 1 Autoimmune Pancreatitis and less commonly, IgG4-SC. Management involves high dose steroids for 4 weeks with a slow taper over years to prevent permanent fibrosis. Patients are at highest risk of relapse during steroid taper or within 6 months of completion, with a relapse rate of 15-60%. Although our patient did not have the classic predictive factors of relapse, such as proximal strictures or serum IgG4 >280 mg/dL, he had a suboptimal response to steroids which prolonged his clinical course and exposed him to repeated procedures. There is still a need for further studies regarding the role for possible combination therapy (steroids & immunosuppressive therapy) to prevent relapse in high-risk individuals.

S1788

Pancreatic Schwannoma: A Rare Pancreatic Tumor

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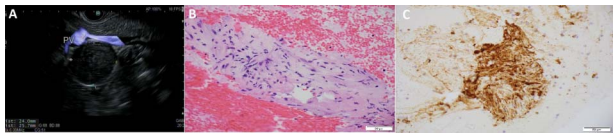
Introduction: Schwannomas or Neurilemmomas are encapsulated tumors of the peripheral nerve sheath. Most schwannomas are sporadic but about 10% are associated with familial neurofibromatosis type 2. Schwannoma may occur in spinal nerve roots, internal organs, or soft tissues. The extrapancreatic nerve plexus may give rise to pancreatic schwannomas. Pancreatic schwannoma is a rare tumor that can mimic other pathologic entities with a more aggressive course such as neuroendocrine tumor or gastrointestinal stromal tumor making preoperative diagnosis critical.
Case Description/Methods: A 21-year-old otherwise healthy female with a history of smoking presented with abdominal pain to the emergency department. She had no nausea, vomiting or fever and urine pregnancy test was negative. The abdomen was soft and non-tender with no masses or bruit and bowel sounds were normal. Given the unexplained pain, abdominopelvic computed tomography (CT) scan was performed. A 2.7 x 2.5 cm mass inferior to the pancreatic tail. The patient was referred for Endoscopic Ultrasonography (EUS). On EUS a round hypoechoic extraluminal lesion was visualized from the second portion of the duodenum. The lesion had a well-defined outer margin, with intact interface seen between the lesion and the pancreas. The lesion was abutting the portal vein with intact interface between the lesion and the vein. The rest of the pancreas showed no significant endosonographic abnormalities with normal pancreatic duct. Endoscopic ultrasound-guided fine needle biopsy (EUS-FNB) was performed using a 25 gauge biopsy needle. Rapid on-site evaluation (ROSE) was suggestive of a stromal cell neoplasm. Final pathological examination showed groups of bland spindle cells with no nuclear atypia, mitotic activity or necrosis and immunohistochemistry revealed diffuse immunoreactivity for S100 and focal reactivity for SOX10, CD117, and SMA consistent with the diagnosis of schwannoma (Figure).
Discussion: Pancreatic Schwannoma is rare, with less than 100 reported cases in the literature. Pancreatic Schwannoma most commonly presents with abdominal pain. EUS with tissue acquisition allows for a definitive diagnosis of solid pancreatic lesions. Preoperative diagnosis of pancreatic masses is critical for appropriate surgical planning and follow-up. This case showcases a typical clinical course for pancreatic schwannoma and serves as a reminder to keep a broad differential diagnosis and appropriate workup, particularly with incidental or unexpected findings on imaging.



[1788] **Figure 1.** A: EUS, B: H&E, C: S100.

S1789

Pancreatic Sarcoidosis Masquerading as Pancreatic Adenocarcinoma

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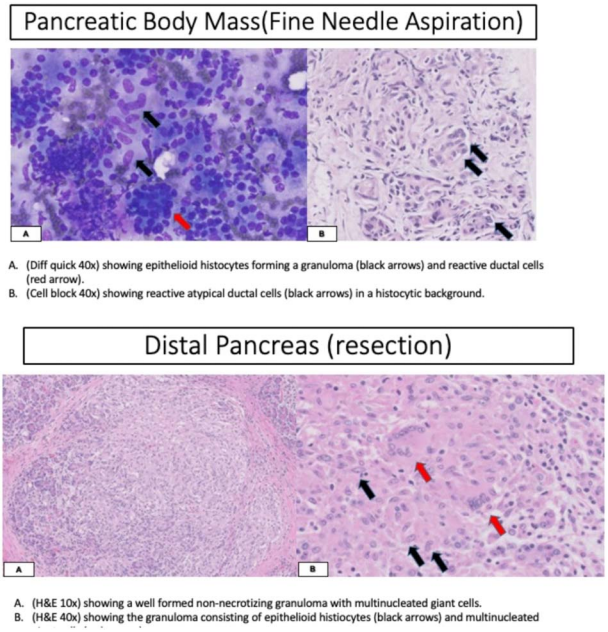
Introduction: Sarcoidosis is a rare, systemic disease that can affect any organ but is most associated with pulmonary manifestations. Excluding the liver, gastrointestinal involvement of sarcoidosis is rare. PS can masquerade as underlying malignancy which poses a diagnostic and therapeutic challenge. Clinical presentation includes symptoms secondary to pancreatic duct obstruction or pancreatic parenchymal

infiltration from enlarged lymph nodes that can result in abdominal pain, jaundice, weight loss, and nausea or emesis. However, these non-specific symptoms can be present in patients with pancreatitis or malignancy. This case highlights the rare finding of pancreatic sarcoidosis masquerading as a pancreatic adenocarcinoma

Case Description/Methods: A 52-year-old African American female with a past medical history of hypertension and pulmonary sarcoidosis presented with one month of epigastric pain. She denied any fatigue, early satiety, diarrhea, nausea, vomiting, obstructive symptoms, or unexpected weight loss. Computed tomography (CT) of the abdomen demonstrated an approximately 2.0 cm low-density lesion within the body of the pancreas with mild pancreatic ductal dilatation in the more distal body and tail of the pancreas concerning a pancreatic neoplasm. She underwent additional imaging for staging including a CT chest that demonstrated cervical lymphadenopathy and progressive hilar adenopathy consistent with her history of pulmonary sarcoidosis. She underwent endoscopic ultrasound which demonstrated a poorly defined 23 mm x 18 mm mass within the pancreatic body with many enlarged lymph nodes in the upper abdomen without any pancreatic duct abnormalities. Fine needle aspiration (FNA) demonstrated epithelioid cells with evidence of atypia and minimal mitotic activity; however, concerning for an adenocarcinoma (Figure). Given the concern of an underlying neoplastic process secondary to distal pancreatic duct changes and the presence of atypical epithelioid cells, the patient underwent a distal pancreatectomy and splenectomy. Pathology demonstrated one large, primary mass measuring 2.4 cm and multiple additional masses forming areas of sarcoidosis. There was no evidence of malignancy. Her postoperative course was uncomplicated with plans for surveillance imaging in 1 year.

Discussion: Although most cases of PS are asymptomatic, the risk of an underlying malignancy needs to be weighed against the risk of PS. Sharing this knowledge with patients can help guide decision-making.



[1789] **Figure 1.** Histologic analysis of the distal pancreatectomy and splenectomy specimen demonstrating no evidence of malignancy.

S1790

Portal Biliopathy in a Patient Who Underwent a Whipple Procedure With Portal Vein Reconstruction

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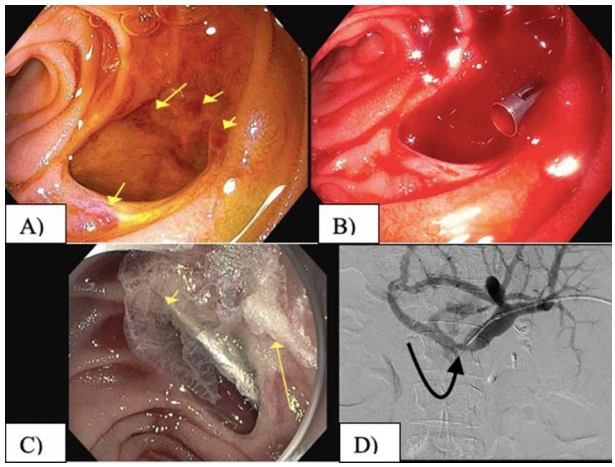
Introduction: Portal biliopathy is a condition that is defined as abnormalities in the biliary tract due to portal hypertension. Some patients may present with abdominal pain, fever, jaundice, or pruritis. Rarely, portal hypertension secondary to portal vein abnormalities cause peri-biliary varices. We report a case of a patient with a peri-biliary variceal bleed due to portal hypertension from acquired portal vein stenosis.

Case Description/Methods: A 59-year-old female with a past medical history of pancreatic ductal adenocarcinoma presented to the hospital with 5 days of Black tarry stools. Two years prior, she was diagnosed with pancreatic head adenocarcinoma and underwent a pancreaticoduodenectomy with portal vein reconstruction. Subsequent CT scans demonstrated post surgical portal vein stenosis. On admission, she denied fevers, abdominal pain, or hematemesis. Labs were notable for a hemoglobin of 5.7 g/dl, INR of 1.6, and bilirubin of 1.0 mg/dl. Physical exam demonstrated an abdominal fluid wave and melena on digital rectal exam. CT abdomen showed post Whipple anatomy, severe portal vein stenosis, large volume ascites, and extensive upper abdominal collaterals including peri-portal and peri-biliary varices. Upper endoscopy revealed blood in the afferent jejunal limb. Bleeding was found to be originating from the choledochojejunostomy. Multiple large visible vessels with stigmata of recent bleeding were visualized within the common bile duct. A single hemostatic clip was placed on one of the vessels, resulting in increased bleeding. Hemostasis was achieved with cyanoacrylate injection. The patient was transferred to the interventional radiology suite, where she underwent portal venography confirming severe stenosis of the porto-mesenteric confluence. Interventional radiology performed portal vein balloon dilation and stent placement. The patient had no further bleeding following the procedure (Figure).

Discussion: Bleeding from peri-biliary varices is a rare cause of upper GI bleeding. In this case, non cirrhotic portal hypertension from acquired portal vein stenosis after pancreaticoduodenectomy led to clinically significant gastrointestinal bleeding. Portal biliopathy is an uncommon and late presentation in patients with extrahepatic portal vein obstruction. In many cases, patients present with signs and symptoms of biliary obstruction. Treatment of PB should involve a multidisciplinary discussion among various subspecialties including gastroenterology, interventional radiology, and hepatobiliary surgery.



[1790] **Figure 1.** A. Choledochojejunostomy with visible varices. B. Hemostatic clip placement with subsequent bleeding. C. Injection of cyanoacrylate glue with hemostasis. D. Portal venography demonstrating portal vein stenosis at the porto-mesenteric confluence.

S1791

Pancreatic Adenocarcinoma With Metachronous Proximal and Distal Colon Metastasis

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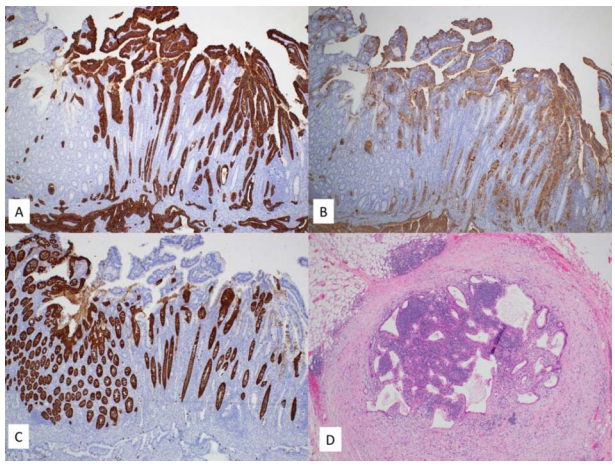
Introduction: Pancreatic Adenocarcinoma (PA) is one of the most common malignancies that carries high morbidity and mortality. PA very infrequently metastasizes to the colon because of its retroperitoneal anatomy. PA with metachronous metastasis to the colon is extremely rare and must be considered when colonic lesions are discovered.
Case Description/Methods: A 67-year-old White man with history of tobacco abuse presented with chief complaint of painless jaundice. Labs showed elevated CA 19-9. Computed Tomography (CT) & MRCP showed biliary and pancreatic ductal dilatation. An endoscopic ultrasound revealed a 2.8 cm pancreatic head mass. Pathology revealed PA. The patient elected to have a pancreaticoduodenectomy, which showed invasive adenocarcinoma, pancreaticobiliary type. The patient tolerated surgery well. He elected to forgo adjuvant chemotherapy.
One year later, he presented with abdominal pain. A CT showed wall thickening of the right colon. CA 19-9 was again elevated. He underwent an exploratory laparotomy with right hemicolectomy for a right colon mass. Surgical pathology showed metastatic PA confirmed with Cytokeratin 7 (CK7) and CA 19-9. He recovered from surgery and received adjuvant chemotherapy with Gemcitabine. 6 months later he presented again with abdominal pain and constipation. A CT showed diffuse sigmoid colitis. He underwent exploratory laparotomy with resection of a sigmoid mass. Surgical pathology showed metastatic PA with CK7 and CA 19-9 expression (Figure). Chemotherapy was stopped and the patient was transitioned to hospice.
Discussion: PA is one of the most common and deadly cancers affecting both men and women. It ranks as the fourth most deadly cancer in the US. Early stage PA is asymptomatic, and many patients are diagnosed with advanced disease. Common areas of metastasis include lymph nodes, liver, and peritoneum; with the lung, pleura, and bone less commonly involved. However, the pancreas is located deep within the retroperitoneum and PA has the ability to infiltrate neurovasculature that supply the GI tract. Cytokeratin 7 is a protein expressed in epithelial cells of the pancreas but not the colon. Therefore, its positivity was key to confirming a diagnosis of PA metastasis. Since 1979, there have been 10 case reports of PA with metastasis to the colon reported in the literature, 3 of which had metachronous colonic disease and the remaining having synchronous lesions. This is the first case of multiple metachronous colon lesions from PA.



[1791] **Figure 1.** A) Cytokeratin 7 is expressed in the neoplastic cells (40X). B) CA19-9 is expressed in the neoplastic cells (40X). C) Villin is expressed in the background benign colonic mucosa (40X). D) A lymph node with metastatic adenocarcinoma.

S1792

Painless Obstructive Jaundice: Pancreatic Adenocarcinoma Cannot Be Excluded

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Introduction: Autoimmune pancreatitis (AIP) is a chronic inflammatory condition typically presenting with abdominal pain, jaundice and weight loss. AIP is an uncommon cause of pancreatitis and diagnosis may be challenging, but it is highly responsive to corticosteroids. We present a case of painless obstructive jaundice complicated by a mass on the pancreatic head.

Case Description/Methods: A 57-year-old Indian male with no past medical history presented with vague abdominal pain, pruritus, and clay colored stools. Vital signs were unremarkable and laboratory investigations revealed alkaline phosphatase 515 U/L, alanine transaminase 732 IU/L, and total bilirubin 7.6 mg/dL. The patient underwent a right upper quadrant ultrasound which showed an enlarged gallbladder and dilated common bile duct (1.9 cm) down to the level of the pancreatic head. An endoscopic ultrasound (EUS) revealed a 3.2 x 3.7 cm hypoechoic mass in the head of the pancreas obstructing the bile duct, 2 enlarged lymph nodes in the area near the bile duct, marked bile duct dilation, and an enlarged sludge-filled gallbladder. Of note, unlike most ductal tumors the pancreatic duct upstream of the mass was not dilated. Fine needle aspiration (FNA) was unsuccessful due to the obstruction by the lymph nodes. Endoscopic retrograde cholangiopancreatography (ERCP) showed stricture of bile duct consistent with a pancreatic head mass, and was stented. Abdominal and pelvic computed tomography (CT) confirmed a 3 cm pancreatic head mass. Pathology result from the attempted FNA was inconclusive. Further labs revealed normal IgG4 and CA 19-9 level of 194; indicative of possible pancreatic malignancy. The patient developed jaundice, scleral icterus and rapid weight loss. EUS with FNA with repeat biopsies revealed rare degenerated epithelial cells. The patient was treated with high dose steroids, with planned pancreaticoduodenectomy. Follow-up CT revealed resolution of the pancreatic mass and was confirmed by repeat EUS/ERCP and normal CA 19-9 level.
Discussion: Although painless obstructive jaundice with a pancreatic mass is a typical presentation for a pancreatic neoplasm; it is not the only possible diagnosis. Despite biochemical and radiographic evidence suggesting a malignancy; if AIP remains in the differential, a trial of steroids may be warranted to avoid unnecessary surgical intervention and associated morbidity.

S1793

Obstructive Jaundice From Adenosquamous Carcinoma of the Ampulla of Vater

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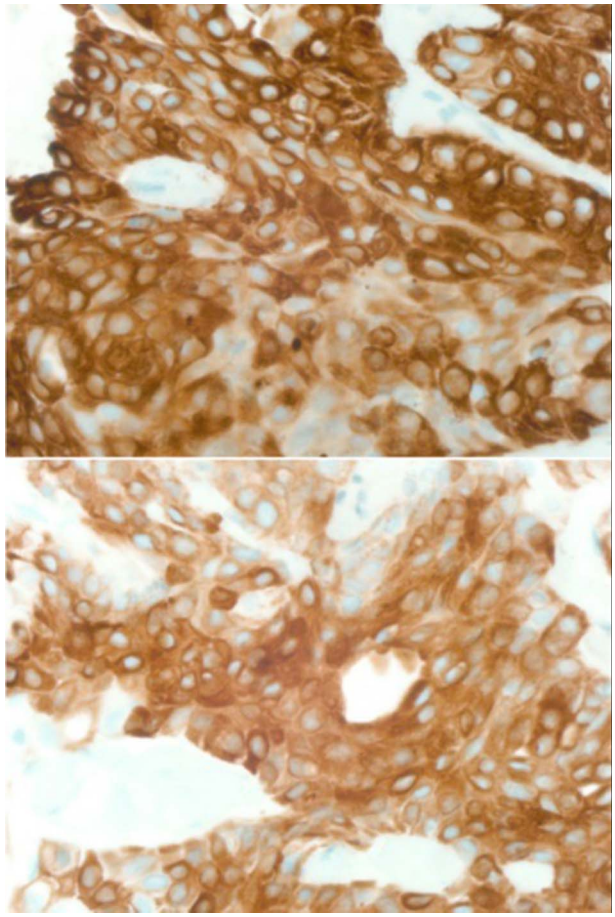
Introduction: Adenosquamous carcinoma of the pancreatobiliary system is a rare malignancy of the gastrointestinal tract, and uniquely unusual due to its composition of both glandular and squamous components. While it carries a worse prognosis compared to more common adenocarcinomas, adenosquamous carcinoma of the ampulla of Vater is reported to have superior postoperative mortality compared to that of the pancreas. Thus, accurate diagnosis is essential for guiding management and prognosis for the patient. Here we report a case of ampullary adenosquamous carcinoma presenting as obstructive jaundice, with imaging suggestive of a pancreatic mass but later identified as ampullary by advanced endoscopy.
Case Description/Methods: A 64-year-old male with GERD presented with 3 weeks of jaundice, pale stools, and dark urine. He was noted to have a 30-pound weight loss over 3 months. He denied a family history of cancer or gastrointestinal disease. The following labs were noted: total bilirubin 22.4 mg/dL, ALP 653 U/L, AST 131 IU/L, ALT 103 U/L, lipase 642 U/L, CEA 5.4 ng/mL, and CA 19-9 4,880 U/mL. CT imaging revealed biliary and pancreatic duct dilation, as well as an ill-defined 7mm pancreatic head mass. EUS revealed a severely dilated pancreatic duct with significant atrophic pancreatic parenchyma and hyperechoic ampullary and periampullary areas. A choledochoduodenostomy was performed with biopsies of the ampulla revealing adenosquamous carcinoma, whereas biopsies of the pancreas showed only inflammatory cells. Histopathology was positive for cytokeratin 5 and 6, focally positive for cytokeratin 7, and positive for p63, which are similar to stains utilized in adenosquamous carcinoma of the pancreas (Figure).
Discussion: While conventional imaging initially suggested a pancreatic malignancy for our patient, advanced endoscopy revealed a rare ampullary malignancy. The histopathology of this patient's adenosquamous carcinoma of the ampulla of Vater resembled that of adenosquamous carcinoma of the pancreas. Postoperative mortality for the former, however, can be more favorable than the latter. This case highlights the importance of accurate distinction of pancreatobiliary adenosquamous carcinoma, and underscores advanced endoscopy as a valuable tool for achieving the correct diagnosis.



[1793] **Figure 1.** (Top) Biopsy of the ampullary mass was positive for immunohistochemical stains CK5/6, mostly seen in squamous cells. (Bottom) Biopsy of the ampullary mass was also positive for immunohistochemical stain CK7, mostly seen in glandular epithelium.

S1794

Pleomorphic Sarcoma Presenting as Pancreatitis, Panniculitis and Polyarthritis Syndrome

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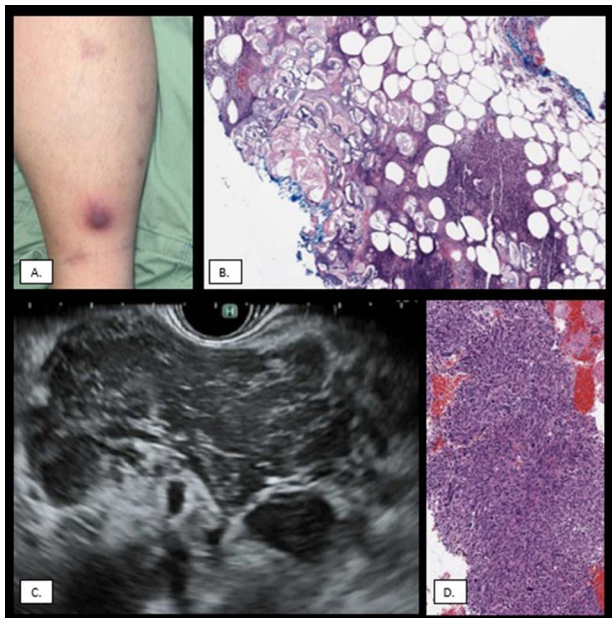
Introduction: Pancreatitis, Panniculitis and Polyarthritis Syndrome (PPP) is a rare triad that develops with chronic or acute pancreatitis and pancreatic cancer. Roughly 50% of PPP patients deny abdominal pain, delaying identification of a pancreatic etiology. Treatment includes symptomatic control and resolving the pancreatic disease. In a review of 59 PPP cases, 11.9% were attributed to pancreatic cancer. None were due to pancreatic sarcomas which account for 1% of pancreatic cancer. To our knowledge, we report the first case of pleomorphic sarcoma presenting as PPP.
Case Description/Methods: An 18-year-old male with no medical history presented for fever and progressive joint pain. He denied respiratory symptoms, abdominal pain, dysuria, or rash. Physical exam revealed erythematous, tender nodules on his shins with ankle, foot and hand swelling. Labs were notable for elevated AST 47, ALT 83, ALP 136 and lipase 9754. Rheumatologic and infectious evaluation was unremarkable. CT imaging revealed a 5.9 x 5 cm solid pancreatic mass with intracystic component. EUS and FNA was completed with histology notable for marked cellular atypia, patchy positivity for CD34, and negative cytokeratin, c-kit, SMA and desmin (Figure). While staining was positive for S100, the retained expression of H3K27me was consistent with pleomorphic sarcoma. Skin biopsy showed panniculitis. Patient underwent chemotherapy with plans for surgical resection.
Discussion: PPP is an extra-pancreatic manifestation of pancreatic cancer. While the pathogenesis is unknown, it is thought to be due to circulating pancreatic enzymes causing lipolysis and can present weeks before abdominal pain. Elevated lipase in the serum or synovial fluid increases suspicion for PPP. Despite pancreatic cancer rarely causing PPP, it should not be discounted in patients without abdominal pain or risk factors for pancreatic cancer. Pleomorphic sarcoma was previously referred to as malignant fibrous histiocytoma due to the spindle cells and significant pleomorphism. Pleomorphic sarcoma is a diagnosis of exclusion and requires careful evaluation to detect cell differentiation. The lack of identifying characteristics on imaging poses a diagnostic challenge. In patients with PPP, symptomatic control is with NSAIDs and steroids; however, resolution depends on definitive treatment of the underlying pancreatic disease. The mainstay of pleomorphic sarcoma remains surgery although chemotherapy and radiation can be used as adjuncts.



[1794] **Figure 1.** A) Erythematous, tender nodules on lower extremity at time of presentation. B) Skin biopsy of the left shin. H&E Stain. High power view of the subcutaneous tissue demonstrates fat necrosis with necrotic adipocyte ghosts and basophilic material, consistent with pancreatic panniculitis. C) Endoscopic ultrasound identification of pancreatic head mass measuring 50mm x 60mm. D) Pancreatic mass aspirate. Cell block, H&E Stain. The pancreatic mass aspirate demonstrates a highly cellular pleomorphic sarcoma with frequent bizarre mitotic figures and anaplastic cytology, consistent with an undifferentiated pleomorphic sarcoma.

S1795

Portobiliary Fistula on Single Operator Cholangioscopy

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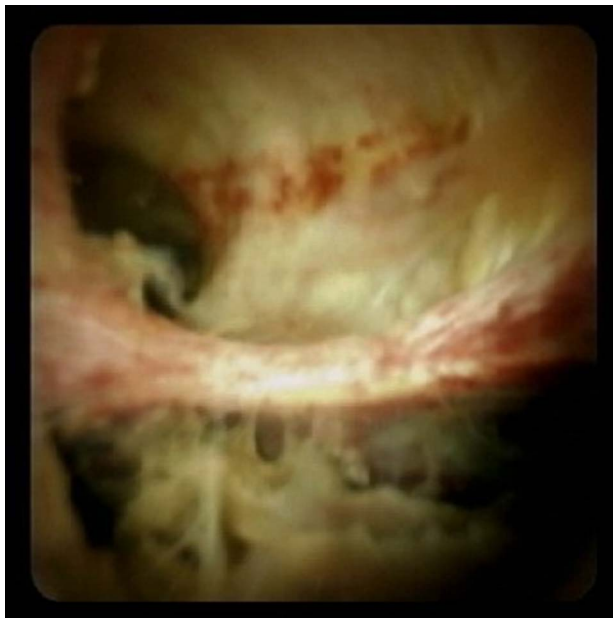
Introduction: Portobiliary fistula formation is an exceedingly rare complication following endoscopic retrograde cholangiopancreatography (ERCP), with a scarcity of cases reported in scientific literature. Patients may present with ensuing—often fatal—gastrointestinal hemorrhage with challenges in diagnosis and management as a result.
Case Description/Methods: We present a case of 78-year-old male who was found to have opacification of vascular structures on intraoperative cholangiography during laparoscopic cholecystectomy. This was concerning for the development of a biliary-vascular fistula. The patient had recently undergone ERCP with biliary stent placement at an outside facility for choledocholithiasis. Gastroenterology was consulted for endoscopic evaluation. An ERCP was performed. Selective biliary cannulation was achieved, and contrast was injected to identify an irregularity in the distal common bile duct with upstream dilatation up to 13 mm. No clear contrast extravasation was noted in the biliary tree. The biliary tree was then swept using a 15 mm extraction balloon with successful removal of gallstones and sludge. Despite multiple contrast injections, extravasation was not seen, as previously noted on intraoperative cholangiography. Therefore, single operator cholangioscopy was introduced and advanced to the hilum of the common hepatic duct. Examination revealed a small defect in the common bile duct communicating with a thrombosed vascular structure, likely a branch of the portal vein (Figure). After a multidisciplinary discussion with surgery service, a fully covered 10 mm by 8 cm metallic stent was placed across the defect, anchored by a second 7 French by 10 cm double-pigtail plastic stent, with trans-papillary drainage.
Discussion: We present a novel approach for the identification and management of portobiliary fistulas. Our case demonstrates the utility of biliary stents in the management of this disease. Our patient tolerated the procedure well with no post-operative complications.



[1795] **Figure 1.** Portobiliary Fistula on Single Operator Cholangioscopy.

S1796

Pancreatitis, Panniculitis and Polyarthralgia Syndrome: A Rare Complication of Pancreatic Pathology

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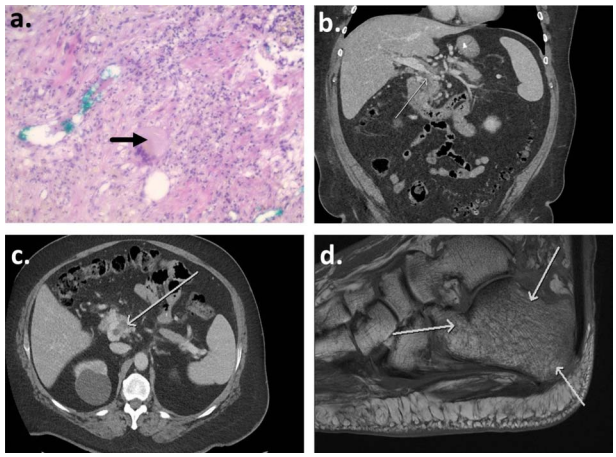
Henry Ford Jackson, Jackson, MI.

Introduction: Pancreatitis, panniculitis, and polyarthritis (PPP) syndrome is a rare syndrome consisting of multiorgan extra-pancreatic manifestations of pancreatitis due to high serum lipase levels. Patients can often have little to no abdominal symptoms. The syndrome can also mimic infectious etiologies leading to misdiagnosis, mistreatment, and the possibility of deterioration.
Case Description/Methods: A 64-year-old male with a history of alcohol abuse presented with painful nodular purulent lesions in his legs and bilateral joint pain in his hips and shoulders. NSAIDs and glucocorticoids did not provide relief. He was initially diagnosed with erythema nodosum. The patient had an elevated Erythrocyte Sedimentation Rate of 75 mm/h, C-reactive protein of 5.7 mg/dL, slightly elevated transaminases, and elevated Lipase of 2328 U/L. A basic metabolic profile and complete blood count were unremarkable. A computed tomography scan of the abdomen showed acute pancreatitis with peripancreatic inflammatory changes. It also showed partial occlusion thrombosis of the portal and superior mesenteric veins. Notably, the patient did not have any abdominal pain on presentation. MRI of the left calcaneus showed bone edema suggesting radiographic evidence of osteomyelitis. The patient was treated with a prolonged course of broad-spectrum antibiotics without improvement. A biopsy of the panniculitis lesions showed lobular panniculitis and ghost cells which were pathognomonic for pancreatic panniculitis. (Figure) Hence, the cause of panniculitis and bone necrosis was thought to be due to the intravascular leakage of lipase due to PPP syndrome. The patient's polyarthralgia was described due to the deposition of free fatty acids in joints following fat autodigestion.
Discussion: After the diagnosis of PPP syndrome, the patient was largely asymptomatic. This case highlights the importance of bone biopsy in diagnosing osteomyelitis. Reactive changes in bone on imaging could be seen in other non-infectious pathologies such as PPP syndrome, as highlighted in this case. The delayed diagnosis led to poor antibiotic stewardship and delay in treatment. This case also demonstrates the importance of a multidisciplinary team in identifying and managing a rare disease.



[1796] **Figure 1.** a. H&E stain of panniculitis lesion biopsy showing ghost cell (arrow) with a background of inflammation suggesting pancreatic panniculitis b. Partial thrombosis of portal vein (arrow) on CT of abdomen (coronal view) c. Partial thrombosis of superior mesenteric vein (arrow) in transverse view of CT abdomen d. Reactive bone changes on MRI of left foot (arrows).

S1797

Pancreatic Hepatoid Carcinoma: A Very Rare and Intriguing Entity

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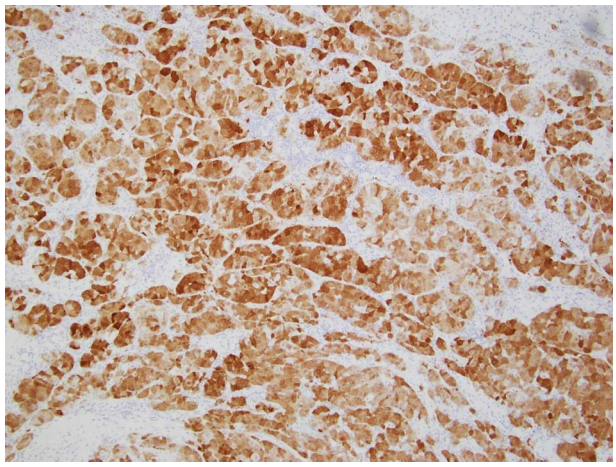
Introduction: Hepatoid carcinoma (HC) is a rare tumor with features morphologically and immunohistochemically like focal hepatocellular carcinoma (HCC). HC is extremely rare in pancreas compared to other organs with less than 50 cases reported in the literature so far.
Case Description/Methods: 67-year-old male with compensated cirrhosis due to hepatitis C, was evaluated for gradual weight loss and poor appetite. Abdominal ultrasound (US) showed a pancreatic head mass only and a cirrhotic liver. Computed tomography scan of the abdomen & pelvis with contrast revealed a 10.1 cm pancreatic head mass and another mixed attenuation 4.18 cm mass in lateral right hepatic lobe suggesting a metastatic neoplastic disease. CA 19-9 levels were normal, and Alfa Fetoprotein (AFP) levels were significantly elevated. Fine needle aspiration (FNA) of the mass via endoscopic US was initially reported as pancreatic adenocarcinoma. An excisional supraclavicular lymph node biopsy had features consistent with metastatic hepatoid (hepatocellular) carcinoma. Additional immunohistochemical staining and evaluation by pathologists of the pancreatic FNA sample confirmed a hepatoid (hepatocellular) carcinoma (Figure). The neoplastic cells were strongly positive for Cam 5.2, Hep par-1, arginase-1, glypican-3, villin, beta-catenin and SMAD-4. The patient was started on atezolizumab and bevacizumab therapy.
Discussion: The theories proposed to explain pathogenesis of pancreatic hepatoid carcinoma (PHS) are a) pancreas has ectopic liver tissue where an HC originates b) the pancreatic cells transdifferentiate into hepatocytes and c) there may be activation of the genes controlling hepatic differentiation of pancreatic cells during carcinogenesis, which are normally suppressed. Four histological subtypes are noticed on a review of 41 cases: purely HCC-like morphology, with neuroendocrine differentiation, and with acinar or glandular differentiation. Serum AFP levels are mostly elevated and used as a marker to determine the success of therapy. Hep Par-1 is thought to be the most sensitive among all the markers. Prognosis is difficult to predict and there is no consensus on preferred chemotherapy due to limited data. Surgical resection is the preferred treatment option. PHC should be considered in the differential diagnosis pancreatic tumors. This case will add valuable information to the limited literature on PHC.



[1797] **Figure 1.** Arginase-1 stain positive.

S1798

Pancreatic Duct Injury Secondary to Pancreatic Trauma Successfully Treated With Transgastric Pancreatic Drainage

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Introduction: Disconnected pancreatic duct syndrome (DPDS) is an underrecognized complication of pancreatic trauma and acute pancreatitis. DPDS is characterized by a disruption of the main pancreatic duct. Its diagnosis is confirmed by either MR cholangiopancreatography (MRCP) or retrograde cholangiopancreatography (ERCP). In this case, we present a patient who developed DPDS secondary to blunt abdominal trauma, successfully treated with a transgastric pancreatic drainage.
Case Description/Methods: A 36-year-old female with a medical history of renal dysplasia and chronic kidney disease stage V arrived at our institution after presenting with epigastric abdominal pain, nausea, and early satiety of a week of evolution. The patient was recently discharged from a trauma center after being admitted for 2 weeks after a domestic violence incident involving blunt abdominal trauma. Upon initial evaluation, the abdominal exam was remarkable for tenderness in the epigastric area upon deep palpation. Laboratory were remarkable for elevated renal parameters and severely elevated pancreatic enzymes. Initial Abdominopelvic CT showed pancreatic transection across the head and neck pancreatic junction, with an associated fluid collection consistent pancreatic fluid extravasation. MRCP was performed, which confirmed the pancreatic transection with an associated pseudocyst at the pancreatic genu consistent with disconnect pancreatic duct syndrome. After interdisciplinary evaluation, an endoscopic ultrasound-guided cystgastrostomy stent was placed. After the procedure, the patient significantly improved symptoms and was discharged 2 days later. The patient was re-evaluated 2 weeks later with a CT scan, which showed transgastric pancreatic drainage with complete resolution of peripancreatic fluid collection. Transgastric pancreatic drainage was removed 2 months later. Follow up Ct scan done 2 months after stent removal showed normal anatomy of the pancreas with no distinct inflammatory tissue reaction and no evidence of peripancreatic collection.
Discussion: Pancreatic ductal injury after trauma is mainly managed surgically but may result in high morbidity and mortality. Advances in endoscopy have allowed minimally invasive interventions as an alternative that can be performed in dstan effective and safe manner.

S1799

Now You See Me and Now You Don't: A Rare Case of Post Cholecystectomy Clip Migration

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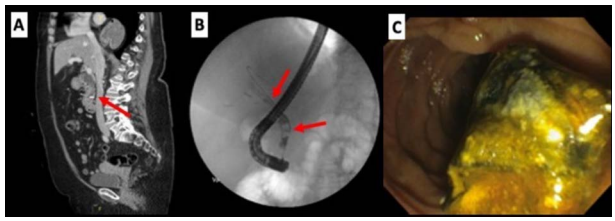
Introduction: Laparoscopic cholecystectomies (LC) have grown to become the gold standard treatment for symptomatic biliary disease, such as cholelithiasis. Each year, over 13 million LC are performed globally. Post Cholecystectomy Clip Migration (PCCM) is a known but rare complication of LC that occurs in less than 5% of cases. We present a case of PCCM presenting as choledocholithiasis that was successfully treated with Endoscopic Retrograde Cholangiopancreatography (ERCP).
Case Description/Methods: A 68-year-old female with a history of laparoscopic cholecystectomy 7 years ago presented with the complaint of right lower quadrant abdominal pain and associated nausea with vomiting. On arrival, temperature was 97.6°, heart rate 86 beats per minute, and blood pressure 162/106 mmHg. Laboratory values were significant for an aspartate transaminase (AST) 192 U/L, alanine transaminase (ALT) 141 U/L, alkaline phosphatase 199 U/L, and total bilirubin 0.68 mg/dL. Follow up Computed Tomography (CT) abdomen/pelvis was completed and revealed a migrated cholecystectomy clip within the common bile duct (CBD) which measured 2.1 cm (Figure A). Moderate intrahepatic biliary dilation was also noted. On the second day of admission, laboratory values peaked at AST 520 U/L, ALT 593 U/L, alkaline phosphatase 217 U/L, and total bilirubin 2.32 mg/dL. Patient was therefore taken for ERCP and guidewire canulation was completed without difficulty. Contrast was injected and the CBD was found to be dilated to a maximum diameter of 14 mm. Two floating filling defects were noted through use of fluoroscopy (Figure B). One defect had a hyperdense focus suggesting the presence of the migrated clip. A major papilla sphincterotomy was performed, and 2 large stones were able to be successfully extracted. One of the 2 stones was found to have the migrated surgical clip embedded within it (Figure C).
Discussion: During cholecystectomy, surgical clips are placed prior to transection of the cystic duct to allow for gallbladder removal. These clips remain in place for years without the need for removal but can act as a potential nidus for stone formation. Problems arise when the surgical clips migrate into the CBD, such as in this case, creating an obstructing pattern and requiring advanced intervention. Although rare, with the increasing number of LC being performed annually, the number of PCCM cases is expected to steadily rise making it imperative that physicians understand and recognize this potential complication.



[1799] **Figure 1.** Image 1: (A) CT abdomen/pelvis with contrast revealing a migrated cholecystectomy clip within the distal common bile duct which measured 2.1 cm. Moderate intrahepatic biliary dilation also noted. (B) ERCP with fluoroscopy demonstrating a floating filling defect due to stones in the distal and mid common bile duct. One clip noted to have a hyperdense focus consistent with migrated cholecystectomy clip. (C) Extraction of the stone with embedded cholecystectomy clip.

S1800

Pancreatic Rosai-Dorfman Disease Diagnosed Without Surgery

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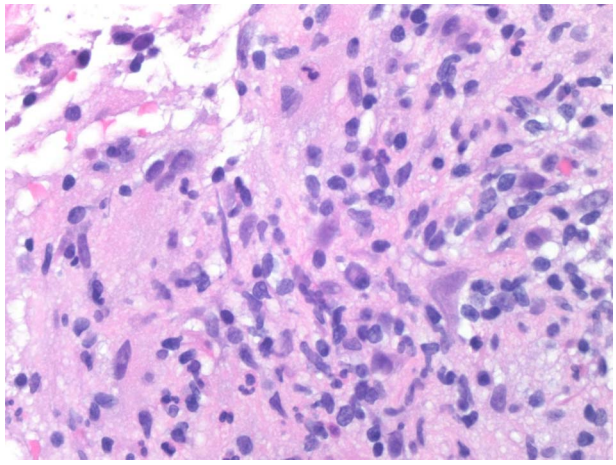
Introduction: Rosai-Dorfman disease (RDD) is a benign non-Langerhans cell histiocytosis that rarely involves the pancreas, of which only eighteen cases have been published to our knowledge. At least fourteen of these cases required surgical intervention. Here we present a case of pancreatic RDD diagnosed by endoscopic ultrasound with fine needle aspiration (EUS-FNA) only, thus avoiding surgical intervention for the patient.
Case Description/Methods: A 49-year-old female with diabetes and hypertension presented with intermittent abdominal pain. CT scan of the abdomen showed a 4cm pancreatic body mass and a 2.3cm retroperitoneal mass. Pathology from EUS-FNA biopsies of both masses showed findings consistent with RDD. This included histiocytosis with emperipolesis, stains positive for S100, CD68, and CD163, and negative for CD1a (Figure). There were no signs of carcinoma or IgG4-related disease. Repeat CT imaging 2 years later demonstrated progressive growth of the masses. The pancreatic mass grew to 4.4cm and the retroperitoneal mass grew to 3.3cm. Repeat EUS-FNA biopsies from both sites once again demonstrated pathologic findings consistent with RDD. The patient agreed to continue routine surveillance without surgical intervention given her stable symptoms.
Discussion: Most cases of pancreatic RDD have required surgical intervention for diagnosis due to initially nonspecific EUS or CT-guided biopsies. Some of these initially nonspecific biopsies were shown to have findings of RDD on re-review of pathology. While there are currently no consensus guidelines for RDD treatment, a wide array of nonsurgical options exists such as observation (as in our case), immunomodulatory agents, and chemotherapy. (Table) Increased awareness of RDD can potentially avoid unnecessary surgical intervention, thus affecting patient mortality and morbidity. We propose that pancreatic RDD does not need surgical intervention for diagnosis, and in cases of nonspecific inflammation on pathology without obvious evidence of malignancy, re-review of the pathology with a focus on RDD should be considered before surgical intervention.



[1800] **Figure 1.** Atypical histiocytic proliferation consistent with Rosal-Dorfman disease.

Table 1. List of all published cases of pancreatic Rosai-Dorfman Disease, including year of publication, patient age, gender, race, pancreatic location, size, and whether surgical intervention was pursued

Year	Age	Gender	Race	Pancreatic location	Size	Surgery
1990	N/A	F	Black	N/A	N/A	N/A
1999	48	F	Black	Body and tail	4cm	Yes
2009	63	F	Black	Head	2.6cm	Yes
2010	35	F	Hispanic	Tail	10.2cm	Yes
2012	74	F	Black	Head	2cm	Yes
2015	59	F	N/A	Head	N/A	Yes
2016	55	F	N/A	Body and tail	3.5cm	Yes
2016	65	F	N/A	Head, body and tail	1.5cm	No
2017	75	F	Black	Head	4.5cm	No
2019	71	F	Asian	Tail	3.5cm	Yes
2019	65	F	Black	Tail	1.9cm	Yes
2019	65	F	Black	Tail	2.1cm	Yes

Table 1. (*continued*)

Year	Age	Gender	Race	Pancreatic location	Size	Surgery
2019	51	F	Black	Tail	2.9cm	Yes
2019	47	M	N/A	Body	4.2cm	Yes
2019	69	M	Black	Tail	2.3cm	Yes
2020	40	M	Black	Tail	1.6cm	Yes
2021	70	M	White	Head	4.8cm	Yes
2022	78	M	White	Head	N/A	No
	49	F	Black	Body	4.4cm	No

The patient of this case report is listed in the bottom row.

BILIARY/PANCREAS

S1801

Pancreatic Plasmacytoma Presenting as Jaundice in Patient With Relapsed Multiple Myeloma

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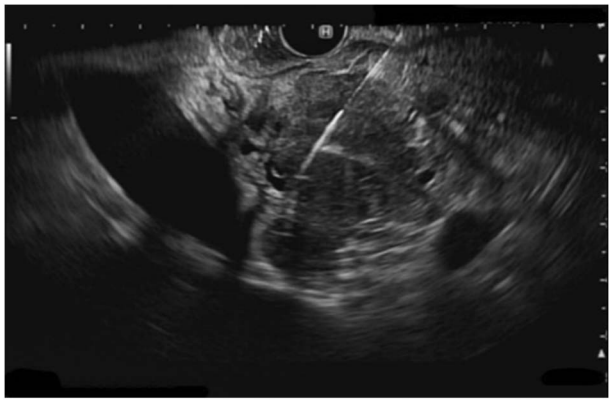
Introduction: Multiple myeloma (MM) is characterized by the neoplastic proliferation of plasma cells producing a monoclonal immunoglobulin. Extramedullary plasmacytomas (EP) are seen in 7% of patients with MM at the time of diagnosis, with an additional 6% developing as the disease progresses. The development of extramedullary disease (EMD) is associated with adverse prognosis and is difficult to treat. The most common sites of EMD include the chest wall, liver, lymph nodes, skin/soft tissue, and paraspinal area. EMD to the pancreas is among the most uncommon areas of involvement and is often diagnosed postmortem due to the indolent and generally asymptomatic progression. We present a 63-year-old female with relapsed MM manifesting as jaundice and biliary obstruction secondary to a pancreatic plasmacytoma. Her diagnosis was made using endoscopic ultrasound with fine-needle aspiration biopsy (EUS-FNA).
Case Description/Methods: 63-year-old female with history of hypertension, anxiety, hypothyroidism, presented with back pain. Imaging showed widespread osteolytic lesions throughout the axial and appendicular skeleton. Lab workup revealed anemia, renal insufficiency, hypercalcemia, and a free kappa/lambda ratio of < 0.01. Bone marrow biopsy demonstrated sheets of large, atypical plasma cells compatible with MM. A repeat biopsy after 6 cycles of treatment showed 5-10% of residual plasma cells. An auto stem cell transplant was performed with minimal residual neoplasm on follow up. She presented again a few months later with rib pain. Imaging showed extensive multifocal osseous activity and a cutaneous plasmacytoma was found. Therapy was initiated, and she subsequently developed jaundice. Imaging demonstrated a 3.1x3.0cm lesion within the head of the pancreas, dilation of the intra and extrahepatic biliary ducts, and metastatic lesions involving the liver. EUS-FNA of liver and pancreatic lesions was performed and revealed malignant CD138+ cells with plasmablastic features (Figure).
Discussion: The development of EMD in multiple myeloma is uncommon and associated with poor prognosis. The patient in our case developed multiple EPs including paraspinal, skin, liver, and pancreas. The pancreas is one of the least common areas of EMD development. It typically does not present until later in the disease course due to the indolent nature and is often diagnosed postmortem. Plasmacytoma can appear similar to other primary pancreatic diseases on imaging, which gives EUS-FNA an important diagnostic role.



[1801] **Figure 1.** Endosonographic image of a fine-needle biopsy targeting a hypoechoic and well-defined lesion within the pancreatic head.

S1802

Obstructive Jaundice Secondary to Hepatic Hilar Tuberculosis

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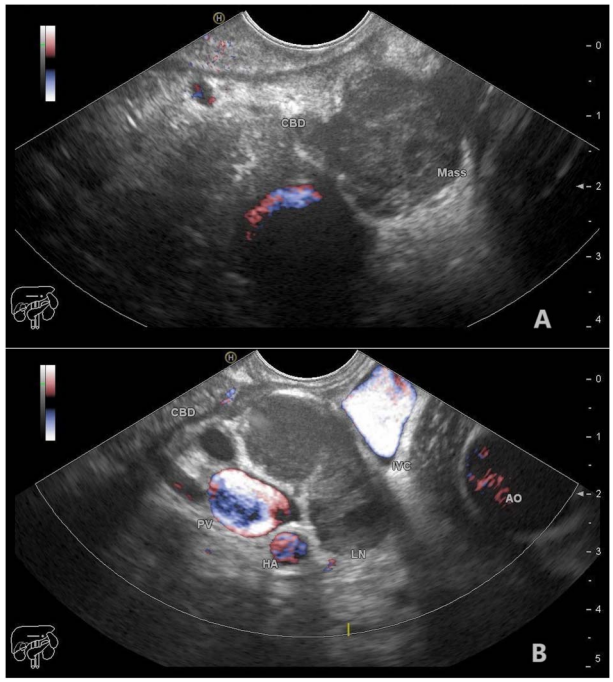
Introduction: Tuberculosis is an infectious disease caused by mycobacterium tuberculosis. It usually involves the lung, but extrapulmonary involvement is not uncommon. Hepatobiliary tuberculosis is a rare form of extrapulmonary tuberculosis that can be mistaken for hepatic hilar malignant tumors.
Case Description/Methods: A 38-year-old gentleman, originally from India, presented to the hospital with right upper quadrant pain for 10 days that was associated with itching, and dark urine; he had no history of fever or weight loss. He was a cigarette smoker but has no history of alcohol drinking; he was not taking any medications. On examination, he was vitally stable. His sclera was icteric, and his abdomen was soft with no tenderness. His laboratory investigations were remarkable for direct hyperbilirubinemia with elevated AST, ALT, and ALP (Table). His ultrasound abdomen showed mildly prominent central intrahepatic biliary duct and prominent upper CBD (measuring 9 mm) with no obvious cholelithiasis. Magnetic resonance cholangiopancreatography (MRCP) showed narrowing in the CBD with a cluster of necrotic conglomerate peripancreatic lymph nodes. Endoscopic ultrasound (EUS) demonstrated a 24x19 mm lesion that is adjacent to the pancreatic head and neck, at the liver hilum, compressing the proximal CBD (Figure). EUS-guided fine-needle biopsy showed necrotizing granulomatous inflammation. TB PCR and culture from the same lesion were positive, and the patient was started on an anti-TB medication regimen.
Discussion: Extrapulmonary tuberculosis can be associated with pulmonary tuberculosis or occur in an isolated form. The absence of constitutional and pulmonary symptoms makes the diagnosis more challenging. Few cases reported compression of the common bile duct due to tubercular hilar adenopathy leading to obstructive jaundice. In such cases, the endoscopic ultrasound plays an important role in obtaining a tissue biopsy to establish the diagnosis and to exclude malignant hepatic hilar tumors that can present in a similar fashion.



[1802] **Figure 1.** Endoscopic ultrasound images showing the tuberculous lymphadenopathy in close proximity to the common bile duct.

Table 1. Laboratory investigations

Laboratory test	Patient's value	Normal range
WBC	6.50 × 10^9/L	4.5 - 11
Hgb	15.3 gm/dL	13.0 - 17.0
Platelet	298 × 10^9/L	150 - 400
Creatinine	1 mg/dL	0.7 to 1.3
Total bilirubin	79.70 μmol/L	3.50 - 24
Direct	46.30 μmol/L	0.0 - 5.10
ALT	478.20 U/L	0.0 - 40.0
AST	270 U/L	0 - 37
Alkaline phosphatase	413 U/L	40.0 - 129.0
CRP	11.20 mg/L	0.0 - 5.0
Hepatitis C Ab	Non-reactive	Non-reactive
Hepatitis B Surface Ag	Non-reactive	Non-reactive

S1803

Pneumobilia: A Feature of Ascending Cholangitis Secondary to Periampullary Diverticulum

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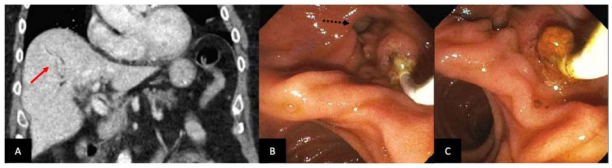
Introduction: Duodenal diverticula (DD) are found in up to 27% of patients who undergo upper endoscopy, with periampullary duodenal diverticula (PAD) being the most common. While largely asymptomatic, PAD can cause biliary obstruction and related complications. We present a case of ascending cholangitis secondary to extrinsic common bile duct (CBD) compression by a PAD.
Case Description/Methods: An 82-year-old man with remote cholecystectomy and ulcerative colitis in remission presented with one day of vomiting and abdominal pain. He had no recent endoscopic or surgical interventions. Initial HR was 120 bpm. Vital signs and physical exam were otherwise unremarkable. Labs were notable for WBC 10.7, AST 279, ALT 104, Alk Phos 205 and total bilirubin of 2.9. Abdominal CT revealed pneumobilia of uncertain etiology (Figure). Due to concern for ascending cholangitis, antibiotics were initiated. MRCP showed mild central intrahepatic biliary ductal dilation and 2 CBD filling defects. Blood cultures grew Klebsiella pneumoniae, Escherichia coli, and Streptococcus gallolyticus. In addition to confirming the MRCP findings, ERCP found a large 2 cm PAD. Sphincterotomy and balloon sweeps removed 2 brown pigmented CBD stones. Occlusion cholangiogram then confirmed successful clearance of filling defects but found a persistent smooth distal CBD narrowing at the area of the PAD, suggestive of extrinsic CBD compression due to the large PAD. Therapeutic decompression with plastic CBD stent placement resulted in immediate clinical and LFT recovery. Final diagnosis was early ascending cholangitis secondary to extrinsic compression by PAD leading to primary choledocholithiasis and bacteremia.
Discussion: While PAD are common, symptomatic presentations are rare and can manifest with CBD dilation, choledocholithiasis, and ascending cholangitis. Lemmel Syndrome is defined by obstructive jaundice caused by a PAD resulting in CBD compression and upstream biliary dilation. We illustrate a case where obstructive jaundice is absent early in the disease course. Proposed mechanisms include stasis-induced choledocholithiasis secondary to mechanical extrinsic CBD compression and bacterial overgrowth of beta-glucuronidase producing bacteria within the PAD and biliary duct, resulting in deconjugation of bilirubin glucuronides and precipitation of calcium bilirubinate stones. Endoscopic interventions are often successful and sufficient, especially in older patients who may be poor surgical candidates.



[1803] **Figure 1.** A. Abdominal CT revealed pneumobila, air within the biliary tree (red arrow). Pneumobilia is most often found after iatrogenic biliary tract manipulation and rarely in emphysematous cholecystitis and biliary-enteric fistulas. However, gas-forming bacteria in cholangitis can also cause pneumobilia, as in our case. B. ERCP revealed large periampullary duodenal diverticulum (dotted Black arrow). C. Removal of brown pigmented stone from ampulla of Vater.

S1804

Poorly Cohesive Carcinoma of Gallbladder Presenting as Metastatic Gastric Outlet Obstruction

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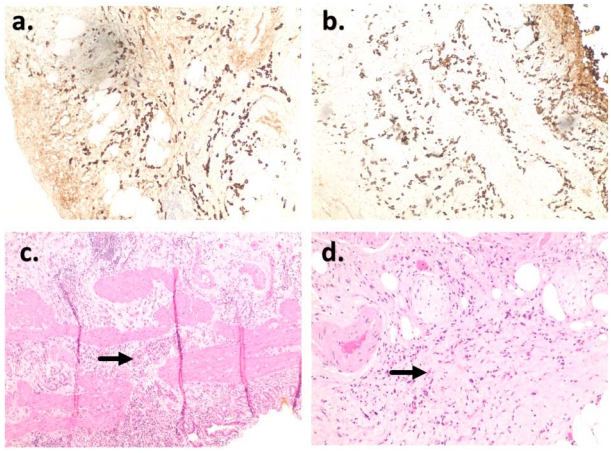
Henry Ford Jackson, Jackson, MI.

Introduction: Gallbladder carcinoma (GBCA) is the most common malignancy of the biliary tract and the fifth most common gastrointestinal malignancy. Gallbladder adenocarcinoma is the most common histological subtype, accounting for about 90-95% of all GBCA. The World Health Organization coined the term 'poorly cohesive carcinoma' (PCC) for a subset of GBCA that was previously known as "signet ring cell" or "diffuse infiltrative" type tumors. PCC is an extremely rare subset of GBCA that is highly aggressive.
Case Description/Methods: The patient is a 77-year-old male with a medical history significant for stage 1 lung adenocarcinoma s/*P* lobectomy and gallbladder carcinoma s/*P* resection and chemotherapy who presented to the outpatient GI clinic for postprandial pain and abdominal fullness. An upper GI series showed a high-grade gastric outlet obstruction with retained gastric contents and minimal passage of barium. An EGD with biopsy was then completed, which showed a pyloric stricture (Figure). Gastric biopsies revealed metastatic poorly cohesive carcinoma of gallbladder origin. The patient underwent an EGD with the placement of a metal duodenal stent to relieve the metastatic gastric outlet obstruction.
Discussion: Although gallbladder carcinoma is a common GI malignancy, the PCC subtype of GBCA is a rare entity. It accounts for less than 5% of all GBCAs. PCC are aggressive tumors with a median survival of 4 months. PCC type of GBCA has been associated with metastasis to various locations including meninges, skin, bone and pulmonary vasculature. However, gastric outlet obstruction secondary to the PCC is yet to be reported. Histopathologically, PCCs from the gallbladder have classically demonstrated sheets of signet ring cells, which were not present in our case. Untreated chronic symptomatic cholelithiasis plays a major role in developing GBCA. Pathological examination of our patient's gallbladder specimen showed evidence of chronic cholecystitis with cholelithiasis. Given the rarity, the pathogenesis behind the development of PCC is unclear. One of the proposed mechanisms involves the presence of pluripotent cells that can differentiate into different types of metaplastic cells leading to metaplasia-dysplasia-carcinoma pathway. Due to the aggressive nature of this tumor, traditional treatments including cholecystectomy have failed to improve the outcome. This warrants further large-scale studies to evaluate the pathogenesis and treatment options for PCC.



[1804] **Figure 1.** a. and b. Pan Cytokeratin AE1/AE3 stain showing poorly cohesive carcinoma. c. and d. H&E staining of the gallbladder biopsy showing tumor cells (arrows).

S1805

Obstructive Jaundice From Portal Biliopathy Following EUS-Guided Glue/Coil Embolization of Duodenal Varices

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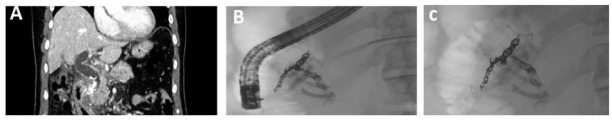
Introduction: Portal biliopathy refers to abnormalities in intra or extrahepatic bile ducts and gallbladder associated with extrahepatic portal vein obstruction and consequently elevated portal pressures. Distended venous collaterals from longstanding portal venous obstruction can lead to compression of the bile duct and thereby portal biliopathy. We hereby describe an unusual presentation of portal biliopathy following EUS guided glue and coil embolization of duodenal varices.
Case Description/Methods: 57-year-old male with a history of duodenal resection with duodenojejunal anastomosis due to a bleeding arteriovenous malformation presented with hematemesis to an outside hospital. The source was unclear on EGD and empiric esophageal variceal banding followed by gastroduodenal artery embolization was performed. Due to continued bleeding patient was transferred and repeat EGD showed actively bleeding varices at the duodenojejunal anastomosis for which variceal banding was performed. CT imaging showed portal vein thrombosis with numerous collateral vessels at the anastomosis and extrahepatic bile duct. Due to continued bleeding and inability to recanalize the portal vein, EUS guided coiling and embolization of ectopic varices was performed. This was done carefully to avoid extravasation into the bile duct Bleeding had stopped and patient did well for several weeks before presenting with epigastric pain, nausea, vomiting, acholic stool and jaundice. Labs were significant for elevated LFTs and lipase levels (Table). CT imaging showed embolization material protruding common bile duct with marked biliary dilation. (Figure A). ERCP showed stenosis at the lower third of the bile duct (Figure B). Biliary sphincterotomy performed and bile duct swept with a balloon resulting in removal of debris and glue material from the duct. Finally, a covered metal stent was placed across the distal common bile duct (Figure C). Post procedurally, abdominal pain and liver tests improved. Patient was discharged with plans for repeat EUS and ERCP in 6-8 weeks for variceal surveillance and biliary stent removal.
Discussion: EUS guided glue injection/coil embolization of pericholedochal varices should be avoided. When inevitable as above, endoscopists should know that, even though there may not be any visible extravasation of glue/coil at the time of procedure, subsequent reorganization, fibrosis and glue/coil extrusion can lead to bile duct obstruction. Patients should be warned and assiduously followed.



[1805] **Figure 1.** Figure1A: Coronal CT imaging showing evidence of extravasation of glue/coil material in the lower part of the bile duct with dilation of portion of bile duct upstream to this Figure1B: Cholangiogram during ERCP showing evidence of extravasated material in the lower part of the common bile duct with non-opacification of upper part of bile duct indicating obstruction Figure1C: Fluoroscopy image after bile duct sweep and placement of covered metal stent.

Table 1. Relevant Laboratory Values

Lab variable	Value
Total Bilirubin	3.2 mg/dL
Alanine Aminotransferase (ALT)	372 IU/L
Aspartate Aminotransferase (AST)	451 IU/L
Alkaline Phosphatase (ALK)	360 IU/L
Lipase	19,590 IU/L

S1806

Pancreatic Somatostatinoma Associated With Neurofibromatosis Type 1

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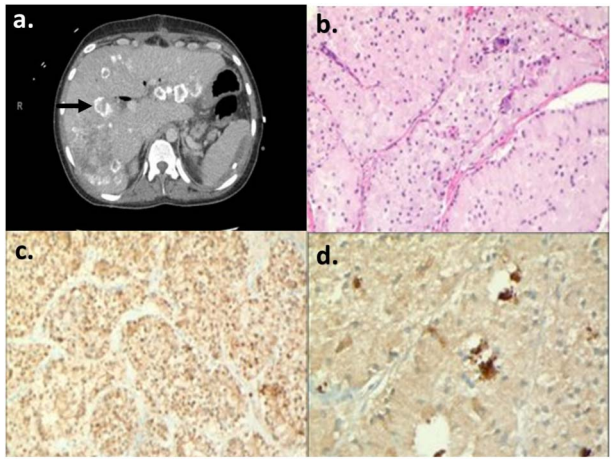
Henry Ford Jackson, Jackson, MI.

Introduction: Somatostatinoma is a rare neuroendocrine tumor often associated with neurofibromatosis type 1, Von Hippel Lindau syndrome, and tuberous sclerosis. The incidence of somatostatinoma is estimated to be 1 in 40 million. Somatostatinomas have an insidious growth, and as a result, it presents in later stages as a malignant disease. Somatostatinoma presenting with "somatostatinoma syndrome," which consists of diarrhea, diabetes, and gallstones, is uncommon.
Case Description/Methods: We present the case of a 24-year-old female who presents with newly diagnosed diabetes, diarrhea, and abdominal pain. The patient's family history consists of MEN (type unknown) syndrome in the patient's father. Physical examination revealed extensive skin nodules (neurofibromas) and hyperpigmented patches (Café au lait spots), raising concerns for neurofibromatosis. Imaging demonstrated innumerable heterogeneous enhancing masses throughout the hepatic parenchyma and a hypodense lesion in the uncinate process of the pancreas, a stent in the biliary duct with a dilated pancreatic duct, enlarged retroperitoneal and mesenteric lymph nodes, and intraabdominal free-air suggestive of bowel perforation. Biopsy of the liver mass demonstrated neoplastic cells. The immunohistochemical evaluation showed cells that stained positive for AE1/AE3, KI67, Chromogranin, CD56, and CK7. The clinical presentation, radiological appearance, and immunohistochemical staining pattern support a diagnosis of somatostatinoma (Figure).
Discussion: Although somatostatinoma presents at a later age, given the insidious growth, it should not be missed in younger individuals with typical symptoms of somatostatinoma syndrome. Given its association with genetic disorders, an effort should be made to distinguish sporadic tumors from those associated with genetic conditions.



[1806] **Figure 1.** a. Transverse view of a CT of the abdomen showing metastatic lesions of the liver (arrow). b-d. Remining images show Immunohistochemistry analysis of liver biopsy using Mouse Monoclonal anti-beta-NGF Antibody IHC stain (b.), AE1/AE3 IHC stain (c.), and Chromogranin IHC stain (d.).

S1807

Pancreatic Neoplasm Presenting as Afferent Limb Syndrome in a Patient With a Remote History of Roux-en-Y Gastric Bypass

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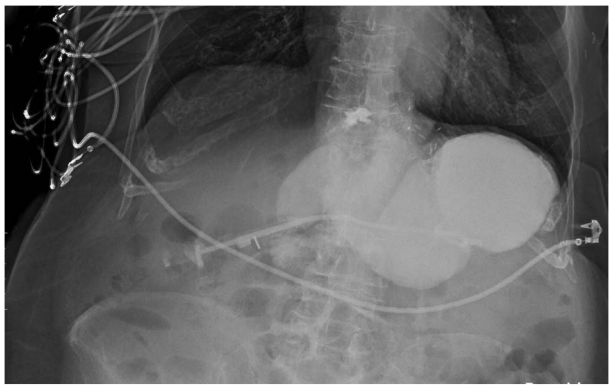
Introduction: Afferent limb syndrome (ALS) is a rare complication of gastrojejunostomy, where the excluded segment carrying digestive juices termed the afferent limb is obstructed distally from varying etiologies. We present a case of pancreatic mass which presented as ALS in a female with a history of Roux-en-Y gastric bypass (RYGB) managed via percutaneous gastrostomy tube (G-Tube) decompression.
Case Description/Methods: A 75-year-old female with a remote history of RYGB presented to the emergency department (ED) due to few days of nausea and dry heaves. Evaluation revealed a hemodynamically stable patient with abdominal distention, and grossly benign laboratory values. Computed tomography (CT) abdomen indicated obstruction with fluid distention of the gastric remnant to the level of the third portion of the duodenum. Supportive care was initiated. General surgery assessed this to be ALS, and recommended medical admission, bowel rest, and G-Tube decompression. Ultrasound-guided percutaneous G-Tube was inserted at the dilated limb, which instantly drained a liter of bilious fluid, and continued for a few more days with exceptional clinical improvement and return of bowel function. Upper GI series was obtained using G-Tube catheter as insertion site for contrast which showed it persisting in the afferent limb for almost 6 hours as in Figure. CT abdomen with intravenous contrast confirmed 2.1x1.6 cm soft tissue lesion involving the uncinate process. Arrangements were made for transfer to tertiary center for surgical oncology evaluation.

Discussion: ALS is a rare complication of gastrojejunostomy due to distal obstruction of the afferent limb leading to accumulation of digestive secretions here while the alimentary limb remains patent. Etiology includes adhesions, stenosis from marginal ulceration, cancer recurrence, and enteroliths. CT finding of dilated afferent limb is diagnostic. Management involves consideration of etiology, site, and functional status. In malignant ALS, management targets primarily palliative treatment and secondarily surgery with curative intent. Key points from the case are: While ALS most commonly follows Billroth II, it can occur in RYGB; Decompression via G-tube placed at the dilated limb proximal to obstruction serves as a palliative measure prior to definitive surgery; While most malignant ALS cases involve recurrence, this mass was uncovered via ALS; While conventional management of malignant ALS was surgical, percutaneous and endoscopic methods are increasingly being employed.



[1807] **Figure 1.** Upper GI series with contrast injected via the catheter of the percutaneous gastrostomy tube demonstrates persistent contrast in the excluded portion of the reconstruction including the gastric remnant and proximal duodenum at 5 hours and 40 minutes.

S1808

Post-Covid Cholangiopathy: An Emerging Complication of COVID-19 Infection

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Introduction: Post-COVID-19 Cholangiopathy is an emerging sequela of the coronavirus infection. Cases have been reported of a secondary sclerosing cholangitis (SSC) type picture presenting insidiously in severe COVID patients.
Case Description/Methods: A 45-year-old female, with recent COVID infection complicated by acute respiratory distress syndrome requiring intubation and tracheostomy, along with ECMO support and VRE and E. coli bacteremia, presented with abnormal liver function tests (LFTs). MRCP noted mild biliary dilation and intrahepatic biliary strictures. ERCP showed diffuse rarefaction of the left and right intrahepatic biliary branches. Acute hepatitis panel, HIV, and autoimmune work up including ANA, anti-smooth muscle antibody, and anti-mitochondrial antibody were negative. Subsequent liver biopsy showed minimal portal hepatitis with prominent bile duct injury and cholestasis with a few bile infarcts. Findings were consistent with SSC likely due to COVID. She was treated with Ursodiol with normalization of LFTs in 6 weeks. MRCP 10 months later demonstrated significant improvement in intrahepatic biliary strictures.
Discussion: Although the respiratory system is the primary target of COVID, involvement of the gastrointestinal system became evident with report of abdominal pain, nausea, vomiting, diarrhea and abnormal LFTs. Post-COVID cholangiopathy has emerged as a complication of the infection and has been characterized as a variant of secondary sclerosing cholangitis in critically ill patients (SSC-CIP). The persistence of cholestasis after recovery of other organ systems in SSC-CIP discriminates it from other hepatobiliary diseases. In the setting of COVID-19, these symptoms persist after recovery from the infection itself indicating the irreversible nature of the damage. A case has been reported of end stage liver disease requiring liver transplantation after severe COVID. Direct viral damage to the biliary epithelium through ACE2 receptors, inflammatory mediators, pharmacologic therapy (antivirals, antibiotics, and glucocorticoids) used in COVID-19, direct cytotoxic effects, focal hypoxia, and hypercoagulable state in COVID infection have been implicated as pathways for hepatobiliary damage with predisposition to SSC-CIP. This case serves to highlight post-COVID cholangiopathy as a rare complication of severe infection and to emphasize the importance of work up of abnormal LFTs in the setting of COVID-19 infection.

S1809

Portal Cavernoma Cholangiopathy Presenting With Acute Cholangitis

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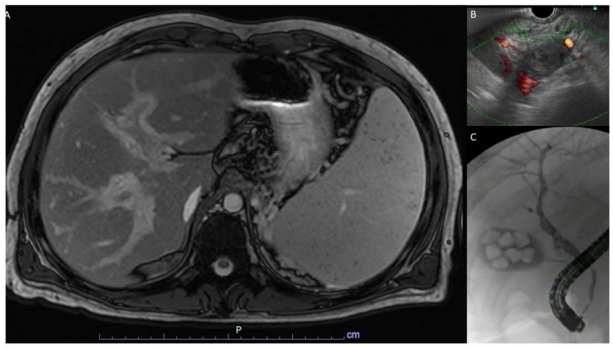
Introduction: Portal cavernoma cholangiopathy (PCC) is defined as abnormalities in the biliary tree due to a portal cavernoma. Most cases occur in non-cirrhotic patients who remain asymptomatic. 5-30% of patients develop symptomatic disease and only 8% of symptomatic cases present with acute cholangitis. This case describes PCC as an uncommon cause of acute cholangitis.
Case Description/Methods: A 26-year-old male with a medical history of acute lymphocytic leukemia in remission, cryptogenic cirrhosis, and portal vein thrombosis (PVT) on warfarin presented with 2-weeks of worsening abdominal pain, darkening of the urine, and weight loss. On examination, he was febrile to 101°F, normotensive, and had both scleral and palatal icterus. Initial labs revealed White blood count: 13.5 x10(3)/mcL, total bilirubin 7.5mg/dL, & ALP 398U/L. MRCP showed biliary dilation related to extensive portal vein cavernous transformation and narrowing in the common bile duct (CBD) due to compression by cavernous vessels in porta hepatis (Figure). Endoscopic ultrasound (EUS) showed PVT and collateral flow. Endoscopic retrograde cholangiopancreatography (ERCP) revealed biliary narrowing and beading. These findings were diagnostic of cavernous transformation with cholangiopathy. The patient improved after biliary stent placement with repeat stenting 6 months later. There was a discussion about TIPS and Portal vein reconstruction. However, the patient did not follow through.
Discussion: PCC develops through PVT-related ischemia that underlies the development of collateral flow, biliary stricture, and dysmotility predisposing patients to cholangitis. Management is only indicated with symptomatic disease with endoscopic and surgical intervention. We report a rare presentation of PCC successfully managed with ERCP.



[1809] **Figure 1.** A) MRCP: Cavernous transformation of the portal vein. B) EUS: A portal vein thrombus involving area of portal vein confluence and collateral flow consistent with cavernous transformation. C) ERCP: Abnormal areas of narrowing and beading throughout the biliary system with extrinsic stenosis.

S1810

Pancreaticobiliary Limb Stenosis in Billroth II Reconstruction as a Cause for Recurrent Acute Pancreatitis

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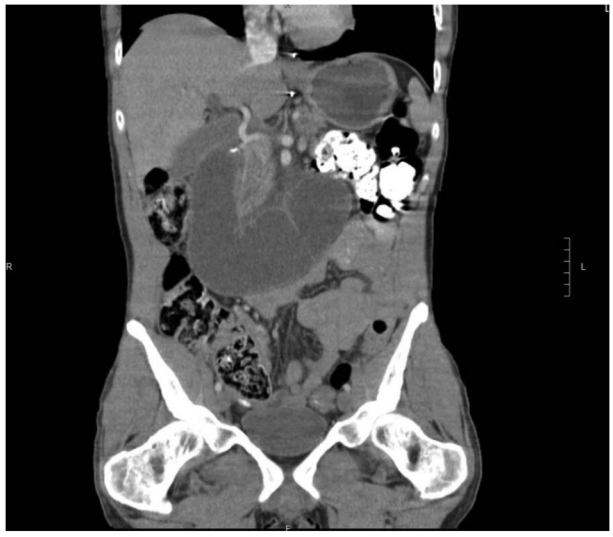
Introduction: Afferent loop syndrome (ALS) has been described in patients with altered gastric anatomy, especially after gastrojejunostomy with Billroth II or Roux-en-Y reconstruction. It has been suggested that a distal obstruction causes increased intraluminal pressure within the afferent small bowel limb leading to accumulation of bile, pancreatic fluids, and small intestinal secretions. We present a case of afferent loop syndrome presenting as acute pancreatitis.
Case Description/Methods: A 38-year-old male with a history of pre and post pyloric strictures status post Billroth II reconstruction who was admitted to our hospital with abdominal pain, nausea and vomiting for 2 days. He was found to have a lipase of 2660 U/l. CT scan of the abdomen showed a dilated duodenal C-loop and changes consistent with acute pancreatitis. He initially underwent an esophagogastroduodenoscopy (EGD) which revealed Billroth II anatomy with surgical staples at the anastomosis, surrounding ulceration and friable mucosa. The entrance to the afferent small bowl limb was severely stenosed and could not be traversed with the ultrathin gastroscope. The patient's clinical status improved with supportive management and he left against medical advice the following day. He again presented 9 days later with epigastric pain radiating to the back, a lipase of 1927 U/l and CT imaging again showing acute pancreatitis and a dilated duodenal C- loop up to 6.5 cm (Figure). He underwent EGD with fluoroscopy; severe stenosis was again found at the afferent jejunal limb orifice. A wire was passed through the stenosed limb, and a Lumen Apposing Metal Stent (LAMS) was deployed through a therapeutic gastroscope. The stenosis was then dilated up to 10 mm through the stent. The patient's symptoms improved and he was discharged with plans for repeat endoscopy in 6-8 weeks.
Discussion: Pancreaticobiliary limb stenosis at the anastomosis site should be considered as one of the rare causes of recurrent acute pancreatitis, especially in patients with previous gastrectomy Billroth II reconstruction. Prompt identification and treatment of any possible stenosis is essential. The use of LAMS may have the potential advantage of reduced stent migration risk and improved likelihood of stenosis resolution.



[1810] **Figure 1.** Dilated duodenal C- loop.

S1811

Pancreaticopleural Fistula: A Rare Presentation of Recurrent Bilateral Effusions in an Elderly Male

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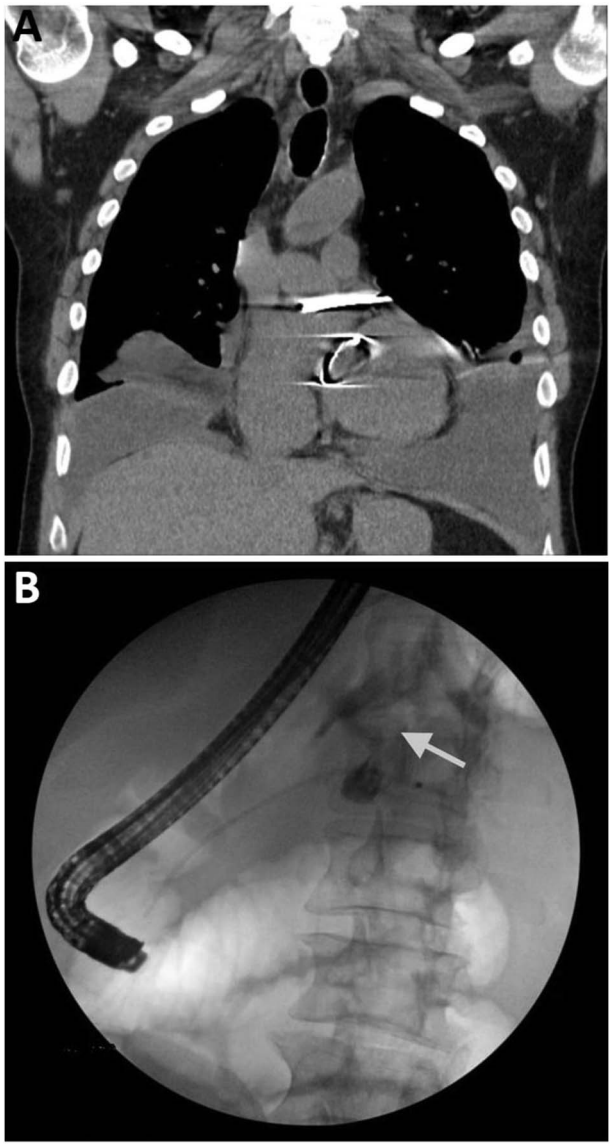
Introduction: Pleural effusions due to pancreaticopleural fistulas (PPFs) are exceptionally rare, often presenting as recurrent, left-sided effusions in middle-aged men with a chronic history of alcohol use and pancreatitis.[1-3] Rarely, PPFs may occur in elderly patients and present as bilateral pleural effusions, as illustrated in this case.
Case Description/Methods: Our patient is a 68-year-old male with a recent episode of acute alcoholic pancreatitis who had since developed bilateral pleural effusions with progressively worsening dyspnea. His recent echocardiogram had resulted as normal and he had been started on oral diuretics. He had also undergone a thoracentesis with ~1.6 L serosanguinous fluid removed, with only tentative improvement in his symptoms; fluid analysis had not shown a clear etiology. He presented to ED with persistent dyspnea and was admitted with recurrence of bilateral pleural effusions (Figure A). He received IV diuresis and a repeat thoracentesis was performed, significant for an amylase level above the assay limit (32,770 U/L). Gastroenterology was consulted for further evaluation and performed endoscopic retrograde cholangiopancreatography (ERCP) which showed pancreatic ductal disruption with contrast extravasation towards the pleural space – consistent with pancreaticopleural fistula – warranting stent placement (Figure B). Following his procedure, the patient had difficulty with extubation and continued to require high-flow oxygen therapy. CT imaging demonstrated recurrence of bilateral pleural effusions with evidence

of a persistent fistula. Surgery was consulted and recommended bilateral chest tube placement along with continuing conservative management of his pancreatic leak (eg, octreotide infusion, TPN). Over time, the patient's symptoms resolved as chest tube output steadily decreased. In the following months, the patient was able to resume PO intake and remove his chest tubes without recurrence of large, symptomatic pleural effusions. A repeat ERCP, conducted several months after discharge, confirmed resolution of the pancreaticopleural fistula.
Discussion: Pancreaticopleural fistulas should be considered in patients with a prior history of alcoholic pancreatitis who present with recurrent pleural effusions, which may rarely occur bilaterally. Although typically occurring in middle-aged male patients, PPFs may less commonly present in the elderly. Lastly, failure of endoscopic therapy may warrant prolonged conservative management or surgical intervention.



[1811] **Figure 1.** A: CT chest with contrast illustrating bilateral pleural effusions. B: ERCP demonstrating extravasation of contrast towards pleural space.

S1812

Pancreatic Adenocarcinoma With Duodenal Metastasis Presenting as Acute Pancreatitis

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Introduction: Pancreatic carcinomas account for 3% of all cancers in the United States and are usually adenocarcinomas. Patients commonly present with fatigue, weight loss, and abdominal pain. We present a rare case of pancreatic adenocarcinoma with duodenal metastasis presenting as acute pancreatitis.
Case Description/Methods: A 65-year-old female with PMHx of hypertension and alcohol use presented to the ED with acute epigastric pain. Lab work revealed a lipase level of 5,800. CT scan revealed findings consistent with acute pancreatitis and inflammation in the duodenum. She was started on intravenous fluids and had improvement in symptoms. She then had sudden onset of abdominal pain, rising liver function tests, and fever after a few days. HIDA scan was negative for acute pathology. An EUS and ERCP revealed a lesion in the ampulla of vater and a 1 x 1.1 cm mass in the uncinate process of the pancreas. Biopsy of the duodenal and pancreatic masses revealed poorly differentiated adenocarcinoma with signet-ring features. Patient was discharged to home and followed up outpatient with gastroenterology.
Discussion: Pancreatic carcinomas (PC) can be composed of exocrine or endocrine cells. Over 95% of PC are exocrine and, of these, 95% are adenocarcinomas. The only potentially curative treatment is surgical resection. Due to late presentation, only 15-20% of patients are surgical candidates. The 5-year survival rate after complete resection is 30% for node-negative disease and 10% for node-positive disease. PC usually metastasizes to the liver, peritoneum, and lungs, and rarely metastasizes to the duodenum. Patients commonly present with insidious epigastric abdominal pain radiating to the back. PC can uncommonly present as an episode of acute pancreatitis and studies have found that patients with this presentation may have a survival advantage due to an earlier stage at diagnosis [1]. As such, pancreatic adenocarcinoma should be considered in patients presenting with idiopathic acute pancreatitis, and duodenal metastasis should be considered in patients with pancreatic adenocarcinoma.

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S1813

Pancreaticopleural Fistula: A Unique Manifestation of the Sequelae of Pancreatitis

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Introduction: Acute pancreatitis is a common diagnosis with established guidelines of treatment and patient monitoring. Further known are the potential for common sequelae such as transition to chronic pancreatitis, calcification, and nutritional deficiencies. Lesser known and published in literature are the pleural sequelae of pancreatitis, namely the potential for pancreaticopleural fistulization. Here, we present a case of pancreatitis with pleural fistula formation to shed light on the common presentation, evaluation and treatment methods of this less common diagnosis.
Case Description/Methods: The patient is a 47-year-old male with a history of alcoholism with prior episodes of pancreatitis, presenting with a complaint of dyspnea. The patient had abdominal pain, as well as elevation of lipase concerning for pancreatitis. Initial imaging demonstrated bilateral pleural effusions for which the patient underwent right sided thoracentesis with fluid studies demonstrating elevated lipase, amylase, and protein. Initially, conversative management was pursued for pancreatitis with NPO status and fluid resuscitation. Without noted clinical improvement, MRCP was performed which demonstrated a concern for fistulization from the pancreatic duct to the lesser sac of the stomach and potential pancreatic pleural fistula formation. With gastroenterology consultation, the patient underwent ERCP with placement of a pancreatic duct stent. With continued clinical decline, CT surgery consultation was requested for recurrence of the pleural effusions. The patient underwent right sided decortication with chest tube placement and bronchoscopy further confirming the diagnosis of pancreatic pleural fistulization.
Discussion: With this case, we establish the pattern used for treatment of fistulization. Literature purports initial management to be conservative with NG or parenteral nutrition. Due to our patient's clinical decline, second line treatment was initiated with endoscopic evaluation. As the patient continued to have pleural effusion formation bilaterally, which while only seen in 15% of cases, prompted tertiary treatment modalities with surgical decortication. This pattern also suggests that should treatment continue to fail, next steps would justify pancreatic drainage or pancreatic resection (Bak et al). Our case provides insight into the step-wise approach to pancreaticopleural fistula management with unique imaging findings, adding to medical literature the management of a lesser known complication of pancreatitis.

S1814

Plasmapheresis in the Treatment of Severe Hypertriglyceridemia-Induced Pancreatitis: A Case Report

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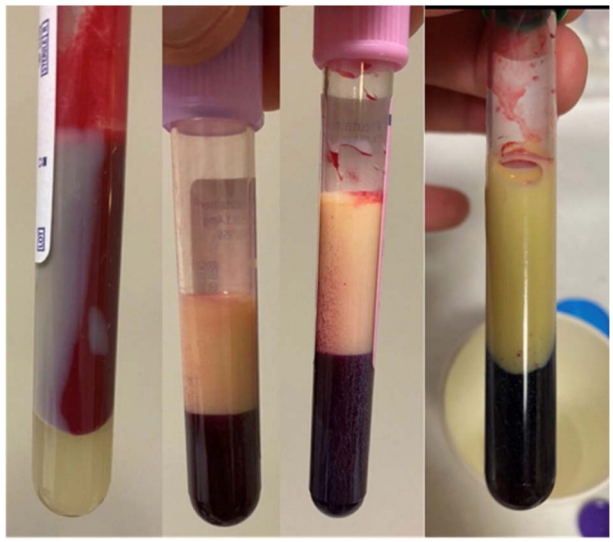
Introduction: Hypertriglyceridemia (HTG) is the third most common cause of acute pancreatitis in the United States after gallstones and alcohol abuse.
Case Description/Methods: Our patient was a 34-year-old male with a past medical history of intermittent binge drinking who presented to the emergency department with a one-day history of left-sided, cramping abdominal pain radiating to the back. He denied similar previous pain or any inciting incidents. As well as any known history of diabetes, medication use, or family history of genetic dyslipidemias and was found to have a normal body mass index. At bedside the patient was found to be normotensive, tachycardic, afebrile, and had a normal respiratory rate. Physical exam revealed moderate tenderness to palpation in all quadrants. The laboratory analysis was initially delayed as the patient's blood sample was too viscous to be analyzed using traditional techniques. After 32 dilutions of the blood sample, laboratory workup revealed calcium 6.8 mg/dL, alanine aminotransferase 73 U/L, aspartate aminotransferase 80 U/L, triglycerides 9708 mg/dL, low-density lipoprotein 373 mg/dL, and lipase 245 U/L (Figure). A CT scan of the abdomen revealed acute interstitial pancreatitis with peripancreatic fluid surrounding the pancreatic tail within the anterior pararenal space. The patient was admitted to the medical ICU for HTG-induced pancreatitis (HTGP). He was started on an insulin drip with a dextrose 10% in water drip titrated to maintain euglycemia, high-volume IV fluids, electrolyte replenishment, and analgesia. The patient's serum TG level was initially measured to be 4025 mg/dL. Given the patient's severe clinical condition, emergency plasmapheresis was initiated with a volume replacement of 1250 cc of fresh frozen plasma and 1250 cc of 5% albumin. After one cycle of plasmapheresis, the TG level was found to be 603 mg/dL. The insulin drip and other treatments were continued during and after the plasmapheresis. After one day, the TG level decreased further to 304 mg/dL. The patient was later transferred to medical floors for continued management.
Discussion: Numerous case reviews and case series have studied the use of plasmapheresis in HGTP. However, clinical evidence remains lacking, and no guidelines have been established regarding morbidity or mortality benefits. We present a unique case of a patient with severe HTGP who was successfully treated with plasmapheresis and experienced a quick and significant reduction in triglyceride levels.



[1814] **Figure 1.** Blood samples from patient.

S1815

Purtscher Retinopathy: A Rare Clinical Manifestation of Acute Pancreatitis

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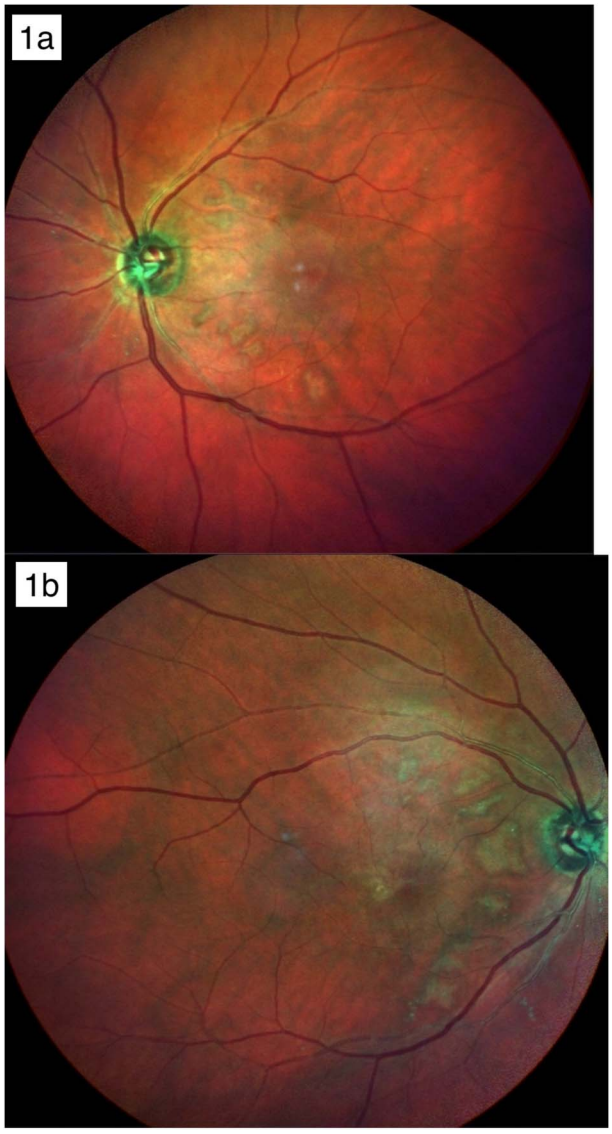
Introduction: Purtscher retinopathy was first described in patients with severe head trauma. Since its discovery it has since been associated with multiple clinical entities, including but not limited to acute pancreatitis and pancreatic adenocarcinoma. Purtscher and Purtscher- like retinopathy share similar pathophysiology and treatment and are collectively referred to as "Purtscher retinopathy", the most commonly associated etiology includes trauma followed by acute pancreatitis. Purtscher retinopathy is extremely rare with a combined estimated incidence of 0.24 cases per million, thus lending to the utility of reporting such cases.
Case Description/Methods: A 64-year-old woman presents with mid-epigastric abdominal pain that alleviates with sitting forward, patient rates the pain 9/10 in severity with non-bilious emesis, diarrhea and poor oral intake. Initial lipase was 3230 U/L and CT of abdomen pelvis was remarkable for diffuse pancreatic edema with infiltration of peripancreatic fat. On Day 7 of hospitalization, she complained of

decreased visual acuity with an inability to see color and count fingers. MRI revealed extensive white matter signal in bilateral occipital lobes suggestive of Posterior Reversible Encephalopathy Syndrome (Figure). Fundoscopic examination revealed cotton wool spots with intraretinal hemorrhages, suggestive of Purtscher-like retinopathy. Patient's vision improved throughout her hospital course with self-resolution of her acute pancreatitis.

Discussion: Pathogenesis of Purtscher retinopathy involves leucocyte aggregation induced by complement C5a activation with proteases in the setting of pancreatic injury. Leukoembolization results in arteriolar precapillary occlusion and ischemia of the microvascular bed in retinal vasculature. Fundoscopy is remarkable for cotton-wool spots with diffuse retinal whitening (Figure A, Figure B), hemorrhage and Purtscher flecken (capillary bed infarcts) which is pathognomonic. Systemic review identified 68 total cases of those 13 occurred secondary to acute pancreatitis. Prognosis for visual recovery is variable and currently there is no evidence-based guidelines regarding definitive treatment. Retinopathy associated with systemic vasculitis is controversially treated with high-dose corticosteroids, which failed to demonstrate statistically significant improvement in outcomes. Observation and treatment of underlying disease etiology demonstrates a judicious therapeutic option.



[1815] **Figure 1.** A. Residual patchy retinal whitening in nasal macula both eyes. B. Residual patchy retinal whitening in nasal macula both eyes.

S1816

Primary Pancreatic Undifferentiated Pleomorphic Sarcoma

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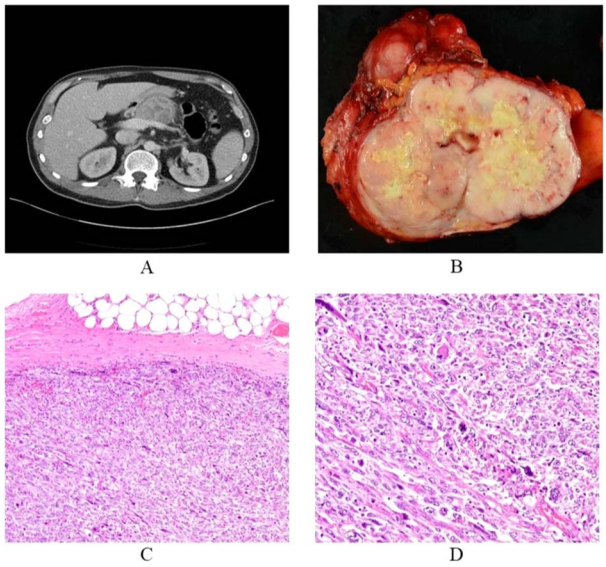
Introduction: Primary pancreatic sarcomas are rare malignancies with an incidence of 0.1%[1, 2]. Sarcomas are frequently seen in the surrounding pancreatic tissue, hence it's important to differentiate primary pancreatic sarcomas from other peripancreatic tumors[3]. We report a case of high-grade primary undifferentiated pleomorphic sarcoma (UPS) of pancreas.

Case Description/Methods: A 48-year-old man presented with acute lower abdominal pain and weight loss. He had a family history of colorectal cancer. He was tachycardic on presentation and physical examination revealed tenderness in LLQ. Laboratory studies showed a mild anemia and a white blood cell count of 13.3/mm^3. Liver panel and CA 19-9 level were normal. Abdominal CT scan revealed a 6.8 cm complex solid-cystic pancreatic lesion (Figure A). Abdominal MRI showed a 9.2 cm macrolobulated mass involving the pancreas without local extension or metastasis. PET CT showed large fluorodeoxyglucose (FDG) avid centrally necrotic pancreatic mass without metastasis. Subsequent endoscopic ultrasound revealed a 6.5 cm solid-cystic mass in the pancreatic body with no vascular involvement and fine needle biopsies revealed malignant spindle cells. Immunohistochemistry (IHC) failed to characterize lineage and differential included carcinoma or sarcoma. He underwent distal pancreatectomy and splenectomy (Figure B). Intraoperative frozen section showed epithelioid and spindled malignancy with IHC negative for EMA, broad spectrum keratins (AE1/AE3 and OSCAR), and high molecular weight keratins. S-100 and other tumor markers were negative. Final pathology revealed high grade undifferentiated pleomorphic sarcoma measuring 10.5 cm with negative margins (Figure C, D). He underwent adjuvant chemotherapy with 4 cycles of doxorubicin/ifosfamide. Surveillance with CT chest/abdomen/pelvis every 3 months was recommended. Follow-up imaging was unremarkable.

Discussion: This case highlights a rare case of primary pancreatic UPS in a middle-aged man. A search of literature revealed 16 cases of UPS with a similar clinical presentation (Table). A step-wise approach with cross-sectional imaging and biopsy is important to appropriately manage these rare malignancies.



[1816] **Figure 1.** A: CT Scan B: Post pancreatectomy and splenectomy C: Undifferentiated pleomorphic sarcoma (UPS) of the pancreas, showing extension into peripancreatic adipose tissue (H+E, x100) D: Higher power view, showing highly malignant-appearing, pleomorphic spindled cells and an atypical mitotic figure (center) (H+E, x200).

Table 1. Summary of all Undifferentiated Pleomorphic Sarcoma of Pancreas report to date (references of the table will be displayed in the poster due to the abstract character count limit)

Author	Age	Sex	Histologic type	Location	Treatment	Preoperative diagnosis	Postoperative therapy	Follow-up (months)
Ishiguchi, et al.	44	M	Pleomorphic	Body-tail	Left pancreatectomy, splenectomy	Pancreatic neoplasms	NA	15, NED
Garvey, et al.	77	M	Storiform-pleomorphic	Uncinate lobe	Enucleation	Pancreatic head mass	NA	48, NED
Pascal, et al.	39	M	Storiform-pleomorphic	Head	Pancreaticoduodenectomy	Mesenchymal tumor	NA	0, DOC
Allen, et al.	46	M	Storiform-pleomorphic	Body-tail, local invasion	Pancreatectomy, splenectomy, subtotal gastrectomy	NA	Chemotherapy	5, DOD
Tsujimura, et al.	43	F	Storiform-pleomorphic	Tail	Pancreatectomy, splenectomy	Pancreatic cystadenoma	Chemotherapy	5, NED
Ben Jilani, et al.	72	M	Storiform-pleomorphic	Body-tail	Left pancreatectomy, splenectomy	Pancreatic mass	NA	12, DOD
Balen, et al.	37	M	Pleomorphic	Body-tail	Extended left pancreatectomy	Pancreatic mass	Radiotherapy and chemotherapy	7, DOD
Haba, et al.	70	M	Storiform-pleomorphic	Head	Pancreaticoduodenectomy	Tumor of pancreatic head	Chemotherapy	22, NED
Bastian, et al.	67	M	Storiform-pleomorphic	Body	Left pancreatectomy, splenectomy, transverse colectomy, subtotal gastrectomy	Pancreatic cancer	NA	34, NED
Darvishian, et al.	74	M	Storiform-pleomorphic	Head	Pancreaticoduodenectomy	Pancreatic head cancer	NA	4, NED
Akatsu, et al.	67	M	Storiform-pleomorphic	Body-tail	Left pancreatectomy, splenectomy, transverse colectomy, total gastrectomy	Pancreatic cancer	NA	35, NED
Mizukami, et al.	44	F	Pleomorphic	Body-tail	Total gastrectomy, left pancreatectomy	Pancreatic tumor	NA	20, NED
Yu, et al.	67	M	Storiform-pleomorphic	Head	Pancreaticoduodenectomy	Pancreatic head cyst	NA	11, DOD
Jarry, et al.	45	M	Storiform-pleomorphic	Head	Pancreaticoduodenectomy	Pancreatic cancer	Radiotherapy and chemotherapy	36, NED
Sanei, et al.	72	F	Pleomorphic	Head and neck	Pancreaticoduodenectomy	Pancreatic head cancer	NA	22, NED
Liang, et al.	37	F	Pleomorphic	Body-tail	Distal pancreatectomy	Pancreatic mass	NA	12, DOD
Own case	48	M	Pleomorphic	Body	Pancreatectomy with splenectomy	Pancreatic mass	Chemotherapy	12, NED

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S1817

Primary Squamous Cell Biliary Carcinoma With Liver Metastasis Is Rare but Malicious

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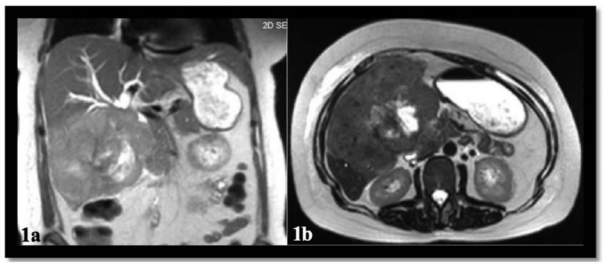
Introduction: Primary squamous cell biliary carcinoma (SCBC) with liver metastasis is quite rare, and to our knowledge, very few cases have been reported in the literature. The exact pathogenesis of the disease is unestablished; however, it's believed that chronic inflammation predisposes to malignant transformation of squamous metaplasia in biliary glandular epithelium. We report a case of a middle-aged woman who was initially diagnosed with adenocarcinoma of the biliary tree that later transformed into SCBC.
Case Description/Methods: A 50-year-old woman with no prior medical history initially presented with postprandial epigastric & right upper quadrant pain. Her symptoms continued to worsen and were associated with early satiety, nausea, and weight loss of 25 pounds over two months, which prompted further evaluation by her primary care physician. CT and MRI (Figure) examination a month later revealed a large heterogeneous area measuring 8.5 x 2.4 x 7.4 cm in the inferior right hepatic lobe with heterogeneous enhancement and involvement of the gallbladder concerning cholangiocarcinoma. Given radiographic findings, she underwent a CT-guided core biopsy of the liver, which showed a necrotic malignant tumor, favoring adenocarcinoma, and was also found to have germline BRCA mutated. PET scan revealed a large partially necrotic FDG avid mass possibly arising from the gallbladder fossa with an invasion of both lobes of the liver and probable involvement of a portion of the ascending colon. There was no gross evidence of distant metastatic disease. The patient underwent staging laparoscopy before initiating chemotherapy, and another biopsy was done, which returned in favor of squamous cell carcinoma, with immuno-histochemical stains being positive for CK19, Ber-EP4, & P40; while negative for CK7, CK20, CDX-2, PAX-8, & Mucicarmine. The patient was started on platinum-based chemotherapy due to germline BRCA mutation. However, due to her poor performance status and recurrent cholangitis related to her cancer and chemotherapy, she could not stay on treatment for an adequate period of time to assess for a response.
Discussion: Primary SCBC remains an unexplored aggressive malignancy with a poor prognosis. Diagnosis can be challenging and requires high clinical suspicion due to the scarcity of specific laboratory workup. Pathological diagnosis remains the gold standard; however, it also carries its own challenges. Treatment is usually case-oriented, and definitive protocols have yet to be established.



[1817] **Figure 1.** a: MRCP showing 12.9 cm heterogeneous mass with a large necrotic center involving the right and left hepatic lobes, common hepatic duct, and proximal common bile duct as well as the ascending colon, especially near the hepatic flexure. b: MRCP showing Intrahepatic (left greater than right) and extrahepatic biliary ductal dilatation due to invasion and/or extrinsic compression in the region of the porta hepatis and proximal extrahepatic biliary tree.

S1818

Rare Sequelae of Obstructive Jaundice: Hyperferritinemia and Bile Cast Nephropathy

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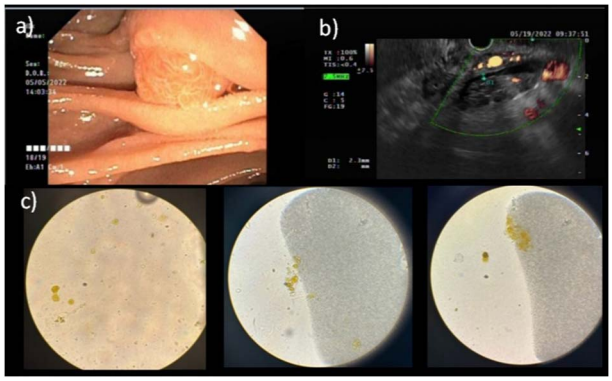
Introduction: Obstructive jaundice occurs due to a physical blockage in the biliary outflow tract or external compression. While jaundice and scleral icterus are common symptoms initially, long term sequelae include severe systemic manifestations from the buildup of various compounds normally excreted in bile such as bilirubin.
Case Description/Methods: A 67-year-old male smoker presented with 3 weeks of pruritus, acholic stools, dark urine, nausea, vomiting, and 20-pound weight loss. Physical exam was notable for jaundice and scleral icterus, LUQ and epigastric tenderness to palpation, but lacked hepatosplenomegaly, ascites or other stigmata of cirrhosis. Liver function tests showed AST 486 IU/L, Alk Phos 1419 IU/L, Alt 600 IU/L, and total bilirubin >30.0 mg/dL with direct bilirubin >10.0 mg/dL. Ferritin was >7500 ng/mL with decreased transferrin of 159 mcg/dL. Hepatitis panel and malignancy/autoimmune markers were negative. CMP showed BUN 74 mg/dL, and creatinine 5.1 mg/dL with 2+bilirubin on urinalysis and microscopic analysis showing pigmented renal tubular cells, bilirubin crystals, and waxy casts suggestive of ATN due to bilirubin damage and possible CKD given waxy casts (Table). CT abdomen reported severe intra and extra hepatic biliary duct dilation with CBD of 17 mm and abrupt tapering with pancreatic head fullness confirmed with MRCP. On day 2, ERCP showed a tight 15mm distal biliary stricture. A small sphincterotomy followed by balloon dilation allowed for placement of a CBD stent with excellent drainage of dark green bile. Following ERCP, patient had a drop of liver function tests and ferritin levels, gradual improvement in BUN and creatinine, and was discharged in stable condition on hospital day 6. On day 16, EUS revealed a hypoechoic area within the periampullary area with atypical cells in brushings collected during repeat ERCP (Figure).
Discussion: Iron and iron binding proteins have been shown to be excreted in bile, primarily in iron excess. Our patient showed a large increase in ferritin signifying the potential role of biliary excretion of excess ferritin, with unknown ramifications. Additionally, severe biliary tree obstruction led to markedly elevated bilirubin, overwhelming renal clearance leading to severe tubular damage. With alleviation of obstruction, there was a steady decrease in ferritin and return to normal baseline renal function. Further research is needed to clarify both the consequences of excess ferritin in biliary obstruction and the significance of excess bilirubin in renal injury.



[1818] **Figure 1.** a) Ampulla prior to ERCP on day 2 shows no periampullary abnormality. b) Day 16 - Endoscopic Ultrasound showing narrowing of distal common bile duct with hypoechoic intraductal area. c) Bile casts and bilirubin crystals seen on microscopy.

Table 1. Patient's Laboratory test results on and post admission

Laboratory Values	Reference Range	Day 1	Day 5 (3 days post-ERCP)	Day 21
AST	13 – 39 IU/L	486	187	18
ALT	7 – 52 IU/L	600	315	27
Alkaline Phosphatase	34 – 104 IU/L	1419	886	160
Total Bilirubin	0.3 - 1.2 mg/dL	>30.0	13.0	4.4
Albumin	3.5 - 5.2 g/	3.0	2.9	3.4
BUN	7 – 25 mg/dL	74	64	18
Creatinine	0.5 - 1.2 mg/dL	5.1	2.90	1.01
WBC	4 – 11 K/cmm	13.8	17.7	10.1
Hgb	13.7 - 17.5 g/dL	11.9	9.4	8.5
Ferritin	24 – 336	>7500	4677	976
Transferrin	300 to 360 mcg/dL	159		233
Iron	50 - 182 ug/dL	126		61
Iron Saturation		57%		19%
TIBC	250 – 450 ug/dl	233		326
Urine Bilirubin	Negative	2+		Negative
Urobilinogen	0.0 - 2.0 EU/dL	4.0		< 2.0
PT	9.1 - 13.2 sec	26.6	12.8	
PTT	23.3 - 36.6 SEC	42.0	33.0	
INR	0.8 - 1.2	2.3	1.1	
Alpha-1-Antitrypsin (ATT)	83 – 199 mg/dL	256		
Ceruloplasmin	18– 36 mg/dL	51		
Carcinoembryonic Antigen (CEA)	0.0 - 3.0 ng/mL	3.9		

S1819

Reactive Lymphoid Hyperplasia of the Pancreas

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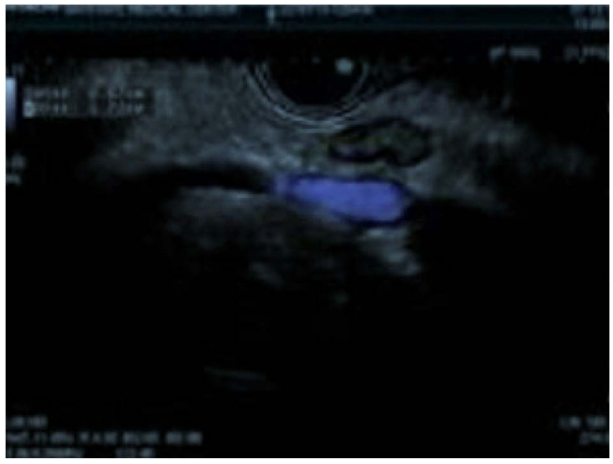
Introduction: Reactive lymphoid hyperplasia (RLH) refers to a benign, nodular mass that is characterized histologically as proliferation of non-neoplastic lymphocytes forming follicles and germinal centers. It can be found in the skin, orbit, lung, stomach, and liver. RLH of the pancreas is extremely rare with only a few cases reported in the literature.
Case Description/Methods: A 72-year-old woman with history of tobacco use and pulmonary nodules underwent a computed tomography (CT) chest for routine nodule surveillance. It revealed interval enlargement of the nodules, suspicious for malignant transformation. A follow up positron emission tomography (PET) scan revealed focal increased Fludeoxyglucose F18 (FDG) uptake within not only the pulmonary nodules but also a small nodular lesion in the pancreatic neck. Magnetic resonance imaging (MRI) of the abdomen subsequently showed a 2.8 x 1.0 cm mass at the level of the pancreatic neck concerning for primary pancreatic neoplasm. Endoscopic ultrasound (EUS) with needle biopsy was requested. EUS revealed an oblong, hypoechoic, slightly heterogeneous, well-circumscribed mass which measured 1.2 x 0.8 cm sonographically in the neck of the pancreas (Figure). There was no nearby adenopathy or celiac plexus adenopathy. The overall appearance was not felt to be typical of pancreatic adenocarcinoma but more consistent with intrapancreatic lymphadenopathy or possibly a neuroendocrine tumor. A transgastric needle biopsy was performed with a good tissue specimen retrieved. The specimen consisted primarily of small lymphocytes and immunohistochemical studies were consistent with lymphoid cells with the majority being T cells with a smaller subset of B cells, favoring a reactive lymph node. The patient was informed that his pancreatic lesion was a benign lymph node in the pancreas.
Discussion: The pathophysiology of RLH is not well studied. An autoimmune or immune reaction to gastrointestinal malignancy has been suggested. RLH is largely considered a benign condition. Spontaneous regression in the liver and lungs has been observed. Fine needle aspiration-guided biopsy with immunophenotyping is helpful in distinguishing RLH from other pancreatic neoplasms, particularly primary pancreatic lymphoma given their shared radiologic features. RLH of the pancreas is generally managed conservatively given its benign nature. Clinicians should consider RLH in the differential diagnosis of hypoechoic lesions located in the pancreas.



[1819] **Figure 1.** Endoscopic ultrasound revealed an oblong, hypoechoic, slightly heterogeneous, well-circumscribed mass which measured 1.2 x 0.8 cm sonographically in the neck of the pancreas.

S1820

Rare Presentations of Metastatic Urothelial Carcinoma to GI Tract: A Case Series Highlighting Key Principles of Endoscopy in the Cancer Patient

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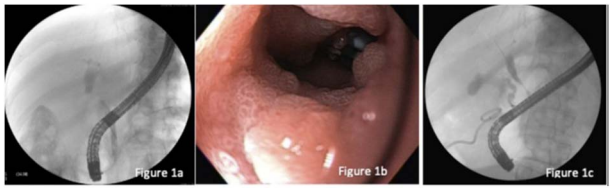
Introduction: In the US, ~80,000 new cases of bladder cancer are diagnosed yearly, 4% of which present with metastatic disease (lymph nodes, liver, peritoneum, lung). Most bladder cancers are pure urothelial carcinoma (Uca). Metastatic spread to the biliary system and small bowel has been rarely described. We present a report of 3 patients with known urothelial carcinoma who presented with gastrointestinal (GI) metastasis.
Case Description/Methods: Case 1: A 71-year-old male with Uca presented with cholangitis. Endoscopic retrograde cholangiopancreatography (ERCP) was done, subhilar biliary stricture found, brush cytology performed, biliary stent placed (Figure A). Endoscopic ultrasonography (EUS) revealed an ill-defined hypoechoic peribiliary mass. Fine-needle aspiration (FNA) cytology confirmed malignant cells from metastatic Uca. Case 2: A 63-year-old male with UCa presented with gastric outlet obstruction (GOO). Imaging revealed a possible pancreatic head mass. Biopsies of the duodenum were non-diagnostic initially. EUS-FNA of the pancreatic head mass revealed benign pancreatic cells. Endoscopy was repeated with extensive biopsies taken of a lumen obstructing externally infiltrating duodenal mass/stricture (Figure B) which confirmed poorly differentiated carcinoma consistent with metastatic Uca. A duodenal stent was placed. Case 3: A 68-year-old male with Uca with metastases to the liver and omentum presented with abdominal pain and biliary obstruction. ERCP was performed with biliary stent placement across the biliary stricture. Brush cytology revealed malignant cells of urothelial origin, representative of metastasis to the CBD (Figure C).
Discussion: Metastatic Uca to the biliary tree and duodenum has been rarely reported. GOO from Uca is also unusual but may occur due to retroperitoneal, hepatic flexure or duodenal masses. Endoscopic evaluation can be challenging due to altered anatomy. This case series emphasizes several important points: *i.* In the appropriate clinical context, non-GI malignancies should be in the differential diagnosis when managing biliary obstruction and GOO. *ii.* Role of repeat endoluminal biopsy if initial effort is non-diagnostic. *iii.* Role of EUS-FNA for tissue sampling. *iv.* Role of ERCP and biliary stenting for palliation of biliary obstruction. *v.* Importance of an accurate pathologic diagnosis in a patient with suspected metastatic malignancy, given management and prognostic implications.



[1820] **Figure 1.** Figure A: Cholangiogram from ERCP for Case 1 demonstrating a high-grade subhilar biliary stricture Figure B: Infiltrating, obstructing duodenal mass with stricture secondary to urothelial carcinoma metastases in Case 2 Figure C: Cholangiogram from ERCP for Case 3 demonstrating a long stricture of the distal CBD and the mid CBD.

S1821

Rare Case of Recurrent Liver Abscess Caused by Choledocodoudenal Fistula in Ampullary Diverticulum

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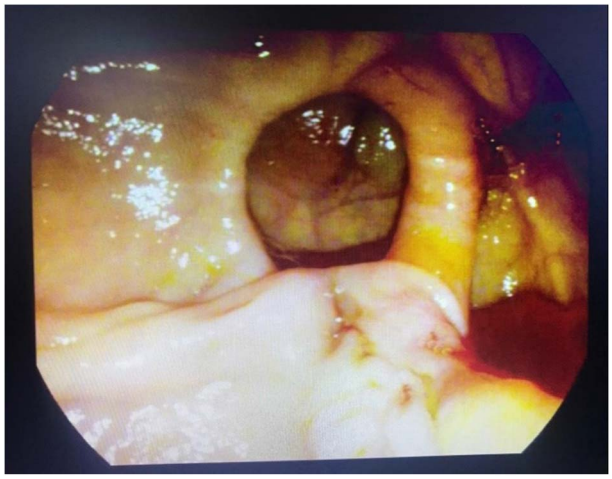
Introduction: Choledocodoudenal Fistula is a rare condition that may be caused by Choledocholithiasis, surgical or laparoscopic cholecystectomy, duodenal ulcer and tumor invasion. Choledocodoudenal fistula has no specific symptoms and may be accidentally discovered during upper GI endoscopy; but in some cases, it may lead to recurrent cholangitis and liver abscess. In this article, a case of recurrent liver abscess caused by Choledocodoudenal fistula in ampullary diverticulum is reported.
Case Description/Methods: A 68-year-old male was admitted to outside facility because of abdominal pain and liver lesion on ultrasound. The abdominal CT showed large heterogeneous mass measuring more than 16*11 cm with single porta hepatis enlarged lymph node measuring about 2*1cm. Hepatocellular Carcinoma was suspected, but liver biopsy revealed active hepatitis with areas of necrosis and supportive inflammation. He was Referred to ALAhli Hospital for evaluation of liver abscess (**Table**). ERCP showed Tiny Ampullary orifice in large diverticulum (**Figure**). The injection of the contrast revealed a fistula at the apex of diverticulum with good drainage through it. In cases of liver abscess secondary to fistula complication, surgical or medical management may be needed. The liver abscess in this case study was treated medically.
Discussion: A management strategy for CDF depends on correcting the underlying cause. As this condition is most commonly stone induced Endoscopic sphincterotomy is the gold standard for CBD stone extraction, however this approach has been found to be successful in only 12% of cases of large stone extraction, usually requiring additional therapies. Endoscopic papillary balloon dilation is an alternative approach to maintain the function of the sphincter and to reduce the morbidity associated with traditional endoscopic sphincterotomy. Currently, the approach of sphincterotomy followed by balloon dilation has provided the best outcomes. Patients with multiple abscesses should receive antibiotics for 4–6 weeks. Percutaneous drainage is the treatment of choice for liver abscesses. Operative drainage is indicated for patients with an identified intra-abdominal focus of infection and for those in whom percutaneous drainage is not feasible or has failed. Anatomic liver resection is rarely required and is withheld for cases of underlying surgical hepatobiliary pathology. In our patient we choose the medical treatment with antibiotics in combination with image-guided percutaneous drainage with excellent outcome.



[1821] **Figure 1.** Ampulla in large Diverticulum.

Table 1. Laboratory results of the patient upon admission to Al-Ahli Hospital

WBC	7.8K/ul	S. Albumin	2.1g/dl	Sodium	133mEq/L
HCT	50%	SGPT	6mg/dl	Potassium	3.7 mEq/L
Hemoglobin	16.8%	SGOT	15mg/dl	Cholride	107mmol/l
Platelets	173K/ul	Bilirubin(T)	0.6mg/dl	S. Creatinine	0.85mg/dl
ESR	75mm/hour	Bilirubin(D)	0.4mg/dl	BUN	17mg/dl
INR	1.3	ALK.PHOS	171 U/L	CRP	190mg/l

S1822

Recurrent Pancreatitis Secondary to Common Channel Volvulus Through Petersen's Space Defect in a Patient With Roux-en-Y Bypass

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Introduction: Petersen's space hernia is an internal hernia that can occur after Roux-en-Y gastrojejunostomy. The intestinal loops herniate through a defect between the retroperitoneum, the transverse mesocolon and the small bowel limbs. We present a case of recurrent pancreatitis in a patient with Roux-En-Y bypass found to have common channel hernia through a Petersen's space defect.
Case Description/Methods: We present the case of a 34-year-old female with a history of Roux-en-Y surgery in 2018 and subsequent recurrent pancreatitis who presented to the emergency department with a chief complaint of severe epigastric and left lower quadrant abdominal pain associated with hematemesis. Patient reported 3 episodes of pancreatitis within 1 year previously. CT abdomen and pelvis showed mildly dilated common bile duct and intrahepatic biliary dilatation with no evidence of pancreatitis. Significant lab work included elevated lipase at 184 U/L. Patient was admitted to the medical service. Gallbladder ultrasound revealed no evidence of cholelithiasis, a prominent CBD of 9 mm and redemonstrated mild intrahepatic biliary dilatation. MRCP revealed a mesenteric swirl in the mid abdomen which was suspicious for an internal hernia in the setting of antecolic Roux-en-Y gastric bypass. It also showed focally dilated intrahepatic with underlying segmental atrophy. General surgery consultation was sought, with eventual plans for diagnostic laparoscopy after ruling out marginal ulcer via EGD. An EGD was performed which did not show evidence of marginal ulcer. Patient then underwent diagnostic laparoscopy which revealed a 360-degree volvulus of the common channel through a Petersen's space defect; this was carefully reduced, and the Petersen's space defect was closed. Patient also underwent laparoscopic cholecystectomy. Patient did not have any further episodes of pancreatitis after surgery.
Discussion: This case demonstrates recurrent pancreatitis in a patient with a history of Roux-En-Y bypass found to have a common channel volvulus through a Petersen's space defect. It is our understanding that the volvulus likely caused compression of the pancreaticobiliary system, thus causing recurrent pancreatitis. Reduction of the volvulus and closing of the Petersen's defect resulted in complete resolution of recurrent pancreatitis in the patient.

S1823

Recurrent Gallstones in a Patient With a Congenitally Absent Gallbladder and Cholangiocarcinoma

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Introduction: Congenital Gallbladder Agenesis (CGA) is a rare anomaly of the biliary tract with an annual incidence of 0.007%. Most patients are asymptomatic but can present with biliary colic and are subsequently misdiagnosed with gallstones. Cholangiocarcinoma (CCA) is a malignant tumor arising from the biliary ducts in patients with underlying chronic biliary tract inflammation, PSC or other diseases. Few studies have reported recurrent cholelithiasis in CGA, but there is only one known case of concomitant CCA and CGA. Herein we present a 79-year-old ma who presented with recurrent gallstones and was diagnosed with gallbladder agenesis intraoperatively with pathology consistent with cholangiocarcinoma.
Case Description/Methods: A 79-year-old male PMH presented to the ED with generalized abdominal pain for 6 weeks prior to admission. Pain was associated with nausea, diarrhea, anorexia and unintentional weight loss of 18 lbs. A gallbladder was not identified on CT A/P and an US was performed, which showed shadowing and contracted gallbladder concerning for obstructive choledocholithiasis. Labs were remarkable for elevated LFT's in a mixed hepatocellular-cholestatic pattern with conjugated hyperbilirubinemia. MRCP showed loss of signal in the mid-CBD and small distal filling defects suggestive of small stones. ERCP identified 2 stones in the lower third of the main bile duct and a sphincterotomy with plastic biliary stenting was performed. A laparoscopic cholecystectomy was performed however aborted as no gallbladder was found. Specimens obtained intraoperatively from a perihilar hepatic mass and CBD resulted positive for cholangiocarcinoma. He underwent partial liver resection and gastrojejunostomy and was discharged under medical oncology for further management.
Discussion: Despite advances in modern technology the preoperative diagnosis of CGA is low. False positive sonographic results may show a shrunken, sclerotic gallbladder, likely artifact from periportal tissue interpreted as hyperechogenic shadows with cholelithiasis and possible CBD dilatation, as such patients are taken for surgical exploration. Surgeons should thoroughly investigate the intrahepatic, retro-hepatic, retro-duodenal, retro-pancreatic and retroperitoneal regions intraoperatively and send specimens for pathology. The relationship between CGA and CCA is unclear, but chronic inflammation of the hepatobiliary tract in the setting of chronic bile stasis due to biliary dyskinesia likely predisposed our patient to CCA.

S1824

Recurrent Shock in a 31-Year-Old Woman With Chronic Pancreatitis and Cirrhosis

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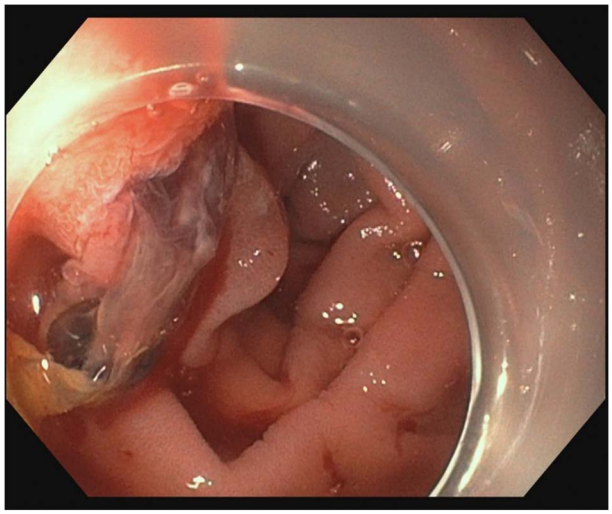
Introduction: Below is the case of a 31-year-old woman with recurrent shock in the setting of chronic pancreatitis and cirrhosis.
Case Description/Methods: A 31-year-old woman with decompensated alcoholic cirrhosis and chronic pancreatitis presented with shock in the setting of epistaxis and tertiary adrenal insufficiency. Endoscopy was initially deferred as evaluation a month prior upon similar presentation with epistaxis and hematochezia showed no luminal evidence or sources of bleeding. During her repeat admission, she required pressor support and stress dose steroids with spontaneous cessation of epistaxis and resolution of shock. She subsequently developed hematochezia. Repeat EGD demonstrated fresh blood and clots emancipating from the ampulla. CT angiography revealed active arterial bleeding within the pancreatic head, consistent with hemosuccus pancreaticus (HP), as well as acute on chronic pancreatitis with peripancreatic fluid collections in the head and tail. Hemostasis was achieved via IR guided coil embolization of the right gastroepiploic artery and gel foam embolization of the gastroduodenal artery and pancreaticoduodenal arcade (**Figure**).
Discussion: HP is a rare cause of gastrointestinal hemorrhage which describes bleeding from the duodenal papilla via the pancreatic duct. The intermittent nature of the bleeding likely explains the frequency of negative endoscopy and delays in diagnosis. Endovascular and surgical management are effective in controlling hemorrhage and reducing mortality, which may exceed 90% without adequate intervention. Chronic pancreatitis is present in 76% of cases of HP and acute pancreatitis is present in 13%. Other, rare etiologies of HP include neoplasm and trauma. Pancreatitis induced inflammation and erosion likely promote vessel degradation and pseudoaneurysm formation. Pseudoaneurysm rupture is the most common cause of HP and as many as 98% of patients with HP have >1 pseudoaneurysm. A minority of patients with HP have no identifiable pseudoaneurysm. The case herein is notable for a number of atypical features, including the infrequent co-occurrence of alcohol induced cirrhosis and pancreatitis with such an early age of onset, as well as involvement of atypical vasculature. The case underscores the importance of a low threshold for repeat endoscopic evaluation of hemorrhagic shock, particularly in patients with high-risk comorbidities.



[1824] **Figure 1.** Fresh blood and clots protruding for the ampulla.

S1825

Secondary Atypical Hemolytic Uremic Syndrome Triggered by Acute Pancreatitis

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Introduction: Hemolytic uremic syndrome (HUS) is a thrombotic microangiopathy defined by hemolytic anemia, thrombocytopenia, and acute kidney injury. HUS can be categorized as typical in the presence of Shiga toxin-producing Escherichia coli, or atypical (aHUS). aHUS is caused by dysregulation of complement pathways from inherited or acquired abnormalities in complement proteins, or secondary to systemic disease. aHUS is exceptionally rare, with an incidence of 1 in 1,000,000. aHUS secondary to pancreatitis has been reported in few cases in literature, and the underlying pathogenic mechanism has yet to be elucidated. Pancreatitis has also been described as a rare extrarenal manifestation of aHUS. It is difficult to differentiate between the two, but genetic evaluation can provide some insight. We present a rare case of secondary aHUS from pancreatitis with negative genetic workup.
Case Description/Methods: A 35-year-old male with no medical history was brought in by ambulance after being found unresponsive. Initial labs revealed a glucose of 1375mg/dL, Creatinine 2.25mg/dL, lipase 1636U/L, hemoglobin 19.8g/dL, sodium 162mmol/L, and platelets 388x10^9/L. CT abdomen showed signs of pancreatitis. Two days later, his platelets dropped to 65x10^9/L. Further lab workup included low haptoglobin, elevated bilirubin, and schistocytes on peripheral smear. Coombs' test was negative. He developed anasarca and oliguria with creatinine increase to 11.7mg/dL and hemodialysis was initiated. Treatment with pulse dose steroids and plasmapheresis was started. ADAMTS13 levels returned normal, and Shiga toxin was negative. He was then started on eculizumab and evaluated for genetic complement mutations which returned negative, supporting secondary aHUS. Eculizumab was continued with return of renal function.
Discussion: Direct damage on the vascular endothelium from drugs, autoimmune diseases, infections, and cancer are well-known causes of secondary aHUS. aHUS secondary to pancreatitis has been described few times in literature. The underlying mechanism in the setting of acute pancreatitis remains elusive but may be related to release of activated enzymes causing endothelial damage and subsequent activation of proinflammatory cascades, leading to complement dysregulation and end-organ damage. The introduction of eculizumab, a C5 complement inhibitor, is now considered first line therapy and can prevent multi-organ damage. This case aims to provide more information in the diagnosis and management of this rare disease.

S1826

Severe Steatorrhea in the Setting of Chronic Pancreatitis Leading to Hyperoxaluria and Subsequent Oxalate Nephropathy Necessitating Dialysis

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Introduction: Steatorrhea is a common clinical entity encountered by gastroenterologists spanning a wide array of patient populations. In addition to causing disruptive symptoms which negative impact quality of life, steatorrhea leads to fat-soluble vitamin deficiency and the corresponding adverse affects including mineral and bone disease, clotting factor deficiency, and delayed wound healing. While multiple disorders can lead to steatorrhea, this report will focus on chronic pancreatitis. Regardless of the etiology, some patients may ultimately develop hyperoxaluria due to the interaction between non absorbed fat in the gut lumen, calcium, and oxalate. This has the potential to lead to oxalate nephropathy, and in extreme cases, acute renal failure. We herein present the case of an 89-year-old man receiving treatment for chronic pancreatitis who developed acute renal failure requiring dialysis secondary to oxalate nephropathy.
Case Description/Methods: Our case involves an 89-year-old man with a past medical history significant for chronic pancreatitis on enzymes replacement, IPMN, and COPD who presented to the hospital at the request of his primary physician for abnormal blood work. On initial evaluation, he was found to be in acute renal failure with a BUN of 104 and creatinine 9.34. Gastroenterology and nephrology teams were consulted. During the course of his stay, our patient was started on dialysis and subsequently underwent kidney biopsy, which revealed oxalate nephropathy. He did not undergo invasive testing such as EGD with secretin administration and subsequent duodenal bicarbonate aspiration to confirm pancreatic insufficiency, as this diagnosis was assumed from prior gastroenterology evaluation and appropriate clinical findings. He was discharged home after kidney function was stabilized and continues on dialysis at this time.
Discussion: This case presentation highlights a potential adverse outcome associated with severe steatorrhea in the setting of chronic pancreatitis, and is a useful lesson for the gastroenterologist so that similar events might be detected at an early stage in future patients. Recognizing acute kidney injury in this setting is of the utmost importance, so that progression to ESRD requiring dialysis may be avoided. Management of steatorrhea in this setting may require aggressive pancreatic enzyme replacement and dietary therapy. Fecal fat quantity may be monitored with stool sudan stain as well as fecal elastase, in an effort to avoid invasive testing.

S1827

Recurrent Acute Pancreatitis in the Setting of Abnormal Pancreaticobiliary Junction

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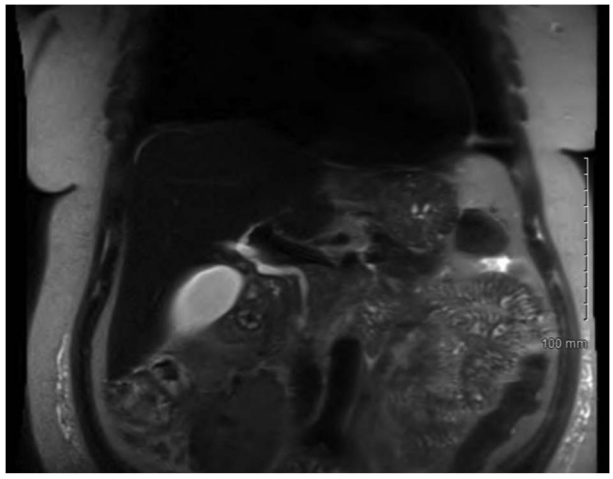
Introduction: Abnormal pancreaticobiliary junction (APBJ) is a condition that can pose a diagnostic challenge. Certain types can cause recurrent acute pancreatitis (RAP). Normally, the major pancreatic duct and the common bile duct open into the second part of the duodenum alone or after joining as a common channel. APBJ happens when these ducts join outside the wall of the duodenum and form a long common channel (>8 mm). We present a case of RAP where the common channel was approximately 24mm long.
Case Description/Methods: A 51-year-old female with a history of RAP. She presented with epigastric pain radiating to her back. Lipase level of 5131 U/L (Reference range 11-82 U/L). Upon review of her history, this was the 6th documented episode of acute pancreatitis in the past 6 years. She denied any significant alcohol use. Multiple abdominal ultrasounds and images did not show signs of cholelithiasis or

choledocholithiasis. MRCP on admission demonstrated aberrant pancreatic duct anatomy with a long common channel of the distal bile duct and the main pancreatic duct measuring approximately 24mm in length (**Figure**).

Discussion: APBJ is an important, albeit rare cause of recurrent acute pancreatitis (RAP). The frequency of APBJ ranged from 1.5%-3.2% in different ethnic populations. The recurrence of acute pancreatitis is at its highest among these patients. Diagnosis is typically made with either an MRCP or ERCP. The clinical features of APBJ vary between patients. Whilst some experience RAP, others can be asymptomatic. Certain features that were associated with a higher incidence of acute pancreatitis include a long (>21mm) and wide (>5mm) common channel, a wide diameter of the proximal pancreatic duct (>2.5mm), the presence of a filling defect in the common channel, and the presence of a pancreatic duct anomaly. In addition to the RAP, APBJ has been associated with chronic pancreatitis, pancreatic carcinoma, and gallbladder carcinoma as the reflux of pancreatic juice into the gallbladder may cause increased bile pressure, and result in epithelial hyperplasia. Most treatment options for APBJ have been surgical, including cholecystectomy and hepaticojejunostomy depending on the presence or absence of an accompanying choledochal cyst. Total resection of the extrahepatic bile duct and hepaticojejunostomy is recommended in children diagnosed with APBJ. Early diagnosis and early surgical treatment provide a good prognosis with a reduction in complications.



[1827] **Figure 1.** Aberrant pancreatic duct anatomy with a long common channel of the distal bile duct and the main pancreatic duct measuring approximately 24mm in length.

S1828

Recurrent Pancreatitis Secondary to Adderall Use With Pancreatic Ascites: A Sight Typically Unseen

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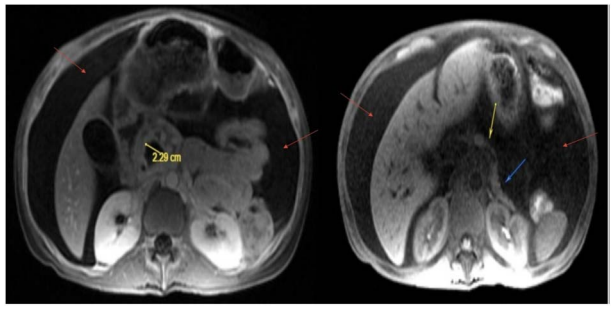
Introduction: Pancreatic ascites is a rare disease with a prevalence of 3.5% resulting from pancreatic duct injury. While small ascites resolves spontaneously, large volume, persistent ascites can cause significant morbidity and mortality. We present a case of pancreatic ascites in a patient with recurrent pancreatitis from Adderall use.

Case Description/Methods: 38-year-old female with attention deficit hyperactive disorder (ADHD), remote alcohol use, and recurrent pancreatitis complicated by pseudocyst and ascites presented with acute on chronic epigastric pain. Her last reported alcohol use was 2 years ago. Home medications included Adderall (amphetamine and dextroamphetamine) and benzodiazepines. Physical examination revealed normal vital signs, severe cachexia and peripheral edema. Abdomen was distended with epigastric tenderness. Pertinent labs included hemoglobin of 10.8g/dl, calcium 7.6mg/dl, albumin 1.7g/dl and a lipase of 462U/l. Bilirubin, transaminases, alkaline phosphatase and INR were normal. Quantitative immunoglobulins, IgG4, triglycerides and thyroid stimulating hormone were within normal limits. Blood alcohol and phosphatidylethanol (PETH) levels were negative. Previous CT scan demonstrated pancreatic head cyst (4.6cm), stable pseudocysts in the head and body, peripancreatic inflammation and large abdominal ascites. MRCP this admission revealed pancreatic head fluid collection decreased in size (2.3cm), consistent with walled off necrosis (**Figure**). Diagnostic paracentesis was significant for: WBC 93 cells/mm3, PMN 25%, RBC 2050 cells/mm3, protein 1.9g/dl, lactate dehydrogenase 102U/L, amylase 1496U/L and a serum ascites-albumin gradient (SAAG) < 1.1. An endoscopic retrograde cholangiopancreatography (ERCP) was pursued due to suspicion for pancreatic ascites which showed abrupt cut off of the pancreatic duct in the head, without filling of the pancreatic duct in the body or tail likely due to stricture. A pancreatic stent was placed into the ventral pancreatic duct to maintain patency with plan for endoscopic ultrasound examination (EUS) to exclude obstructive pathology.

Discussion: Pancreatic ascites is often seen with recurrent pancreatitis commonly of alcoholic etiology. Our patient had recurrent pancreatitis and ascites due to chronic adderall use. While pancreatitis from adderall use is rare, presentation with ascites can pose a diagnostic challenge. Hence, clinicians should familiarize themselves with early diagnosis and endoscopic management which improves prognosis in this rare disease.



[1828] **Figure 1.** MRCP images revealing large volume ascites (red arrow), walled off necrosis in the pancreatic head (yellow arrow) and peripancreatic inflammation (blue arrow).

S1829

Synchronous Pancreatic Masses

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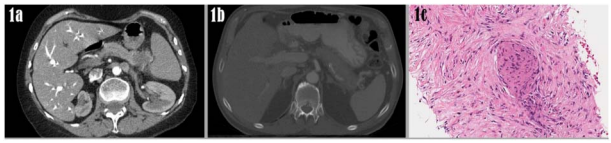
Introduction: Any mass lesion in the pancreas typically raises concern of undiagnosed pancreatic malignancy. Presence of synchronous multiple pancreatic masses is a rare finding. In this case series, patients presented with two or more synchronous solid masses as a result of pancreatic cancer (PC), autoimmune pancreatitis (AIP), and sarcoidosis.
Case Description/Methods: Case1:65-year-old female presented with abdominal pain and 20lbs unintentional weight loss over 4 months. CT scan revealed two suspicious solid masses in the body/tail of the pancreas (**Figure A**). IgG4 level was normal, but CA19-9 was elevated at 75u/mL. EUS with individual fine needle biopsies (FNB) of both masses confirmed infiltrative PC. Due to the significant cardiac history, the patient was deemed not a surgical candidate and was referred to oncology for chemoradiation/palliative therapy. Case2:76-year-old male presented to the hospital with postprandial abdominal discomfort and unintentional weight loss. CT Abdomen demonstrated localized inflammation in the pancreatic tail (**Figure B**). EUS showed mass-like lesions in the pancreatic head and tail. Immunohistochemistry was positive for IgG4-positive plasma cells. He was diagnosed with AIP and was started on steroids. Case3:54-year-old male with complicated sarcoidosis (pulmonary/extrapulmonary involvement), presented with an abnormal PET scan showing focal increased uptake in the head/tail of the pancreas. His CT scan did not show any mass or duct dilation. EUS demonstrated ill-defined, infiltrative masses involving the pancreatic head and the tail. FNB showed scattered non-necrotizing granulomas (**Figure C**). After excluding other causes of granulomatous diseases, he was diagnosed with pancreatic sarcoidosis.
Discussion: Only a few examples of synchronous pancreatic masses have been recorded in the medical literature. Our case series includes three distinct pancreatic diseases that result in multiple mass lesions with similar appearance on imaging (**Table**). The clinical course for all of the patients differed greatly depending on the pathology. The plurality of solid masses and comparable imaging features of each with PC, which is the 4th highest cause of cancer-related deaths in the United States is the highlight of this series. When encountering such individuals, a broad differential should be examined, as the clinical history of the illness varies. The whole pancreas should be investigated with multimodal imaging and EUS-guided acquisition histopathology to reach a clear diagnosis.



[1829] **Figure 1. A**: Abdominal CT showing two solid masses in the body and tail of the pancreas. B: CT Abdomen demonstrated localized inflammation in the pancreatic tail.C: High power view of a sarcoidal granuloma. (Hematoxylin and eosin, 200× magnification).

Table 1. **Summary Table**

Case #	Past Medical History	Presentation	Pancreatic lesions	Pathology	Management
1	Coronary artery bypass Heart failure Mitral and tricuspid regurgitation Atrial fibrillation Pulmonary hypertension Renal Thrombosis	Abdominal pain, unintentional weight loss, fatigue	Two (Pancreatic body and tail)	Adenocarcinoma	Follow up with hematology/ oncology, radiation oncology, palliative care
2	Coronary artery disease Uncontrolled diabetes Previous smoker (80+ pack-years) Former alcoholic	Postprandial gastric discomfort, unintentional weight loss	Two (Pancreatic head, and tail)	Areas of fibrosis and edema with lymphoplasmacytic infiltration and immunohistochemistry was positive for IgG4 positive plasma cells consistent with Type 1 Autoimmune Pancreatitis	Responding to steroids
3	Complicated sarcoidosis diagnosed in 2015 with pulmonary and extrapulmonary involvement including bones, spleen on prednisone taper History of Waldenstrom's, marginal zone lymphoma status post RCVP (Rituximab, Cyclophosphamide, Vincristine, Prednisolone) therapy in 2015 followed by Rituximab maintenance till 2017	Incidental findings on imaging	Three (Pancreatic head, uncinate process, and tail)	Non-caseating granulomas consistent with sarcoidosis	Off plaquenil, methotrexate, infliximab due to side effects, not on any sarcoidosis medication since 2019. Clinical monitoring and cardiac MRI

S1830

Spontaneous Fistulization of Walled-Off Necrosis Into Duodenum Requiring Video-Assisted Retroperitoneal Debridement: A Rare Complication of Acute Necrotizing Pancreatitis

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Introduction: Gastrointestinal fistulas are an uncommon complication of acute or chronic pancreatitis, with colonic and duodenal fistula occurring in 60.5% and 26% of cases, respectively. We present a case of a spontaneous fistulization between the walled-off pancreatic necrosis (WOPN) and the duodenal bulb.
Case Description/Methods: A 35-year-old male with a history of peptic ulcer disease was transferred to our institution after developing abdominal pain, distension, and decreased urinary output for several days. Upon arrival, he was found with tachycardia, fever, and respiratory failure requiring mechanical ventilation. Laboratories were remarkable for leukocytosis, transaminitis, and hyperbilirubinemia with normal pancreatic enzymes. Images showed necrotizing pancreatitis, with more than 90% of devitalized pancreatic parenchyma and multiple homogenous peripancreatic non-enhancing fluid collections, the largest one measuring 10.9cm x 11.5cm x 4.8cm, with associated bilateral paracolic gutters with abundant free fluid. Supportive therapy with bowel rest, hydration, and antibiotics were started. Bilateral pararenal space percutaneous drainage was placed with subsequent upscaling of French catheter diameter without improvement. Follow-up images were remarkable for a larger retroperitoneal walled-off necrosis with fistulous communication between the walled-off necrosis and the first segment of the duodenum. Despite initial efforts, the patient remained septic with symptoms concerning gastric outlet obstruction. Therefore, a decision was made to perform a video-assisted laparoscopic retroperitoneal pancreatic debridement of pancreatic walled-off necrosis. After two weeks, the patient improved clinically, and repeated imaging revealed no drainable fluid collections.
Discussion: Duodenal fistulization of WOPN is extremely rare, and most occur due to superimposed infection of necrotic pancreatic material. Sterile and asymptomatic WOPNs can be treated conservatively. Drainage endoscopically, percutaneously, or surgically is best for infected WOPN; however, these have higher morbidity and mortality rates. Our case depicts a spontaneous fistulization of WOPN into the duodenum that failed a "step-up" approach of percutaneous drainage requiring video-assisted retroperitoneal debridement with complete resolution of symptoms. This emphasizes the importance of infection source control to prevent further clinical deterioration and a prompt evaluation of acute abdominal pain.

S1831

Solid Pseudo-Papillary Pancreatic Tumor With a Rare Advanced Metastatic Presentation

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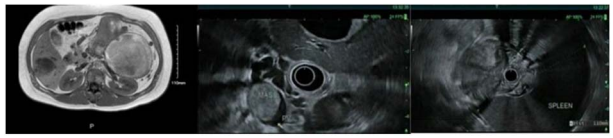
Introduction: Solid pseudopapillary tumor (SPT) of the pancreas is a rare neoplasm, usually characterized by a well encapsulated mass, with low malignant potential. It occurs predominantly in young females, in the third decade of life, accounting for about 1% of all tumors of the pancreas mainly located in the body and tail. SPT usually exhibits benign behavior but we present a case of a young female with advanced metastatic disease on presentation.

Case Description/Methods: A 32-year-old female with history of gastritis presents with stabbing, intermittent epigastric pain radiating to the back. In the past, she had presented to the emergency department for gastritis. Vitals notable for low-grade fever and tachycardia. She had no prior colonoscopy/EGD. Family history significant for colon cancer in dad at age of 70. Physical exam was remarkable for epigastric tenderness. Labs showed normal complete blood count, chemistry, liver function tests, lipase. Liver function test were mildly elevated with AST 40 and alkaline phosphatase 131. CT followed by MRI revealed multiple bilobar hepatic lesions, filling defects in the portal veins with periportal and perisplenic collateral, complex multiple masses from the pancreatic tail, the largest measuring 14.9cm (**Figure**). CA19-9, CEA, and AFP were negative. Esophagogastroduodenoscopy (EGD) and endoscopic ultrasound was done with fine-needle aspiration of the pancreatic mass. Histopathology was consistent with SPT.
Discussion: The solid pseudopapillary tumor of the pancreas is an uncommon exocrine pancreatic tumor that accounts for less than 1% of all pancreatic tumors. Malignancy can arise in roughly 15% of instances, presenting as metastases or invasion of surrounding structures, despite the fact that majority show benign behavior. The most common metastatic sites are the liver and the omentum. They may present as an abdominal mass, jaundice, or abdominal pain as described in this case. The vast majority are found in the pancreatic body and tail. Ultrasound or CT scans are commonly used to diagnose them. In addition to a pre-operative pathology diagnosis, endoscopic US allows for further FNA biopsies. Histopathology of SPT reveals solid nests of poorly cohesive cells resulting in a pseudopapillary architecture. Alpha1-antitrypsin, CD56, CD10, and Vimentin are characteristically found in them. They are usually highly responsive to chemotherapy. In conclusion, pancreatic pseudopapillary tumors are rare neoplasms with malignant potential.



[1831] **Figure 1.** MRI showing multiple hepatic lesions as well as pancreatic mass and endoscopic ultrasound illustrating heterogenous mass within the pancreatic tail with portal vein invasion.

S1832

Spontaneous Pancreatic Pseudocyst-Superior Mesenteric Vein Fistula: A Case Report

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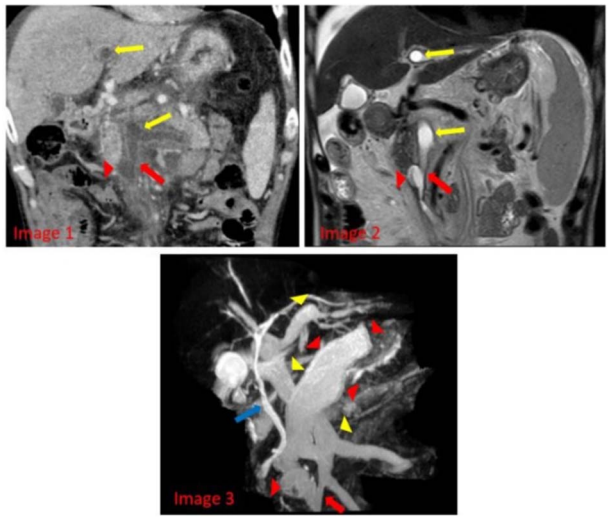
Introduction: Fistula development between pancreatic pseudocysts and the portal venous system is a rare and life-threatening complication of acute pancreatitis that is challenging to diagnose and treat. We present this case to highlight certain aspects of diagnosis, management, and potential complications.
Case Description/Methods: A 50-year-old man with alcohol use disorder presented with 6 months of fatigue, unintentional weight loss, and worsening intermittent abdominal pain. He was diagnosed with acute on chronic pancreatitis based on presentation, labs, and imaging. Contrast-enhanced CT demonstrated diffuse portal venous thrombosis with a questionable fistulous connection between a pancreatic pseudocyst and the portal venous system. A follow-up MRCP revealed a retroperitoneal pseudocyst with an obvious communication with the superior mesenteric and the left portal vein. An ERCP showed a pancreatic duct leak with secondary pancreatic ascites that was managed by pancreatic ductal stenting. Post ERCP course was complicated by septic shock with bacteremia that swiftly resolved with antibiotics. Anticoagulation was deemed inappropriate given the high risk to benefit ratio in the setting of the existing fistula. His course was further complicated by recurrent secondary bacterial and fungal ascites with significant neutrophilia that resolved with antibiotics, pleural effusion requiring brief chest tube placement, and malnutrition that was managed by early enteral nutrition. He gradually improved and was discharged to a rehabilitation facility with stable interval abdominal imaging while on suppressive oral antibiotics (**Figure**).
Discussion: While initial CT raised suspicion for the fistula, MRCP was necessary to make a definitive diagnosis. This is consistent with existing literature that has shown MRCP to be considerably more sensitive than CT. Prompt diagnosis with MRCP and follow up ERCP lead to early pancreatic ductal stenting for management of a pancreatic duct leak with secondary pancreatic ascites, and anticipation of other potential complications (bleeding, infection, and malnutrition) without any need for a surgical intervention. Considerable complications were seen in this clinical scenario similar to the previously reported literature. Aggressive treatment and anticipation of these complications with accurate imaging, early enteral feeding, pancreatic ductal evaluation, and targeted suppressive antibiotic therapy contributed to the positive outcome on follow up.



[1832] **Figure 1.** Images 1 and 2: Coronal CT and T2 weighted MR images, respectively, of the the upper abdomen demonstrate the inferior component of the partially visualized large pseudocyst (red wedge) fistulizing a branch of the SMV near the pancreatic head (red arrow). Fluid density and correlating MR T2 bright fluid signal is noted in the partially visualized proximal SMV and the left portal vein (yellow arrow). Image 3: MRCP MIP reconstructions are used to emphasize fluid in a 3D image. The large pseudocyst is noted (red wedge) with cystic component fistulizing a branch of the SMV (red arrow). Extension of the pseudocyst conforms to the extrahepatic and intrahepatic portal venous system (yellow wedge). Normal appearing common bile duct also noted (blue arrow).

S1833

Single Operator Cholangioscopy in a Patient With Pancreaticoduodenectomy

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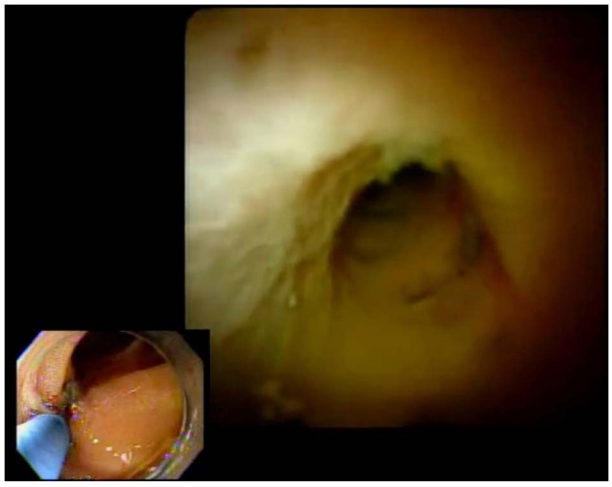
Introduction: Cholangioscopy in patients with surgically altered anatomy can be technically challenging. In most patients with pancreaticoduodenectomy undergoing single operator cholangioscopy, the most common approach is through a percutaneous biliary drain placed by interventional radiology. We present a case in which a modified therapeutic upper endoscope (1T scope) was used for single operator cholangioscopy in a patient with pancreaticoduodenectomy.

Case Description/Methods: A 64-year-old female with Gardner syndrome requiring colectomy and classic pancreaticoduodenectomy for an ampullary adenoma 10 years prior presented for surveillance endoscopy. She previously underwent endoscopic mucosal resection and intraductal radiofrequency ablation (RFA) of adenomatous tissue at the hepaticojejunal (HJ) anastomosis. She subsequently developed a stricture at the HJ anastomosis needing sequential dilations. A 1T scope was used and advanced to the HJ anastomosis. After balloon dilation of the HJ anastomotic stricture, cholangioscopy was performed and revealed abnormal biliary mucosa (**Figure**) approximately 2 cm below the hilum raising concern for intraductal extension of adenomatous tissue. Cholangioscopy directed biopsies were obtained and pathology revealed tubular adenoma without high-grade dysplasia. Intraductal RFA is planned in the future.
Discussion: ERCP in patients with pancreaticoduodenectomy can be challenging. Different endoscopes have been used to overcome some challenges of ERCP in altered anatomy, each offering certain advantages and limitations. Commonly used endoscopes for ERCP in patients with pancreaticoduodenectomy include the adult and pediatric colonoscopes and single balloon enteroscope. These endoscopes do not allow for performing single operator cholangioscopy due to the length of the scope or the width of the accessory channel. Our report highlights that the 1T endoscope allows for performing single operator cholangioscopy in patients with prior pancreaticoduodenectomy when the HJ anastomosis can be reached.



[1833] **Figure 1.** Intraductal extension of adenomatous tissue seen on cholangioscopy.

S1834

Spontaneous Bacterial Peritonitis in a Postpartum Female Secondary to Necrotizing Pancreatitis

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Introduction: SBP is an infection of the fluid that accumulates in the abdomen caused by translocation of bacteria without an obvious source, such as bowel perforation. Typically, this translocation represents a failure in defensive factors to contain pathogens to the bowel and can lead to seeding other extra-intestinal sites. Our patient is unique in that her SBP was characterized in the setting of a recent pregnancy. Her course was complicated by pre-eclampsia resulting in emergent c-section, further complicated by hypertriglyceridemia-induced pancreatitis. After significant intervention, necrotizing pancreatitis leading to retroperitoneal fat saponification and abscess formation was the likely etiology for SBP.
Case Description/Methods: A 27-year-old female presented with diffuse abdominal pain, nausea, vomiting, and diarrhea. Past medical history is significant for recent emergency c-section due to pre-eclampsia complicated by hypertriglyceridemia-induced pancreatitis. On admission, she was hypotensive, tachycardic, tachypneic and febrile, with significant bilious emesis. CT revealed large volume ascites, peri-pancreatic fluid, small volume fluid and gas within the endometrial cavity. Peritoneal fluid analysis presented an SBP picture. Abdominal drains were placed to mitigate peritoneal fluid collection, which settled in the retro-peritoneal space. Cultures resulted in pan-sensitive E.coli growth and the patient was started on appropriate therapy. However, the patient continued to have persistent fevers and severe abdominal pain while on medical management. At this point, exploratory abdominal surgery was indicated revealing significant amounts of retroperitoneal saponified fat secondary to necrotizing pancreatitis, and extensive communicating abscesses which were subsequently drained.
Discussion: SBP is an infection of fluid that accumulates within the abdomen, typically seen in patient's with chronic liver disease and can be managed with medical therapy. In our patient's case, there was no know history of liver disease. Our patient presented febrile, with diffuse abdominal pain. Imaging revealed significant ascites and cultures resulted in pan-sensitive E.coli. Multiple differentials were assessed including recurrent pancreatitis, C.difficile infection leading to bowel perforation and/or protein-losing enteropathy, endometritis given recent c-section amongst others. After surgical assessment, our likely diagnosis was necrotizing pancreatitis seeding pancreatic enzymes into the retroperitoneal cavity.

S1835

Silent Hemoperitoneum and MRSA Bacterascites: An Unusual Presentation of Gallbladder Perforation

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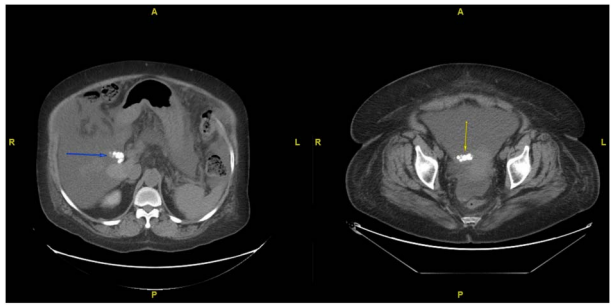
Introduction: Gallbladder perforation (GBP) is an uncommon, life-threatening event, most often seen as a complication of acute cholecystitis. Occurrence of GBP in the absence of cholecystitis is exceedingly rare, but may occur in those with cholelithiasis. We report a case of spontaneous GBP without cholecystitis presenting as painless hemoperitoneum and methicillin-resistant *Staphylococcal aureus* (MRSA) bacterascites in a patient with decompensated cirrhosis.
Case Description/Methods: A 63 year old female with past history of NASH cirrhosis (MELD 27, Child-Pugh C), esophageal varices, and ascites requiring weekly therapeutic paracentesis presented with encephalopathy. A paracentesis on admission was negative for spontaneous bacterial peritonitis by cell count and culture. CT imaging on admission revealed a partial small bowel obstruction (pSBO) and cholelithiasis with an otherwise normal gallbladder and biliary tree. The pSBO resolved with conservative management. One week into the hospitalization, her ascites worsened. A second paracentesis showed 253,290 RBC/mm^3, with a corrected ANC of -54 cells/mm^3. A 3 gram serum hemoglobin drop was also noted. A repeat abdominal CT scan showed a non-inflamed gallbladder with her known gallstones now layering in the pelvis, suggestive of GBP (**Figure**). She was initially started on piperacillin-tazobactam, but her ascitic fluid culture later grew MRSA. Based on her surgical risk and overall clinical stability, she was managed non-operatively. She was transitioned to indefinite therapy with amoxicillin-clavulanate and doxycycline, and later discharged home.
Discussion: Most GBPs are due to severe inflammation or trauma. Risk factors for spontaneous GBP include cholelithiasis, congenital obstruction, and anticoagulant therapy. Although rare, patients with cirrhosis may have a higher risk for GBP due to venous congestion from portal hypertension and their propensity for cholelithiasis. Her MRSA bacterascites may have been from iatrogenic seeding during the first paracentesis, but the timeline to discovery raised concerns that it originated from the GBP. MRSA is rarely a biliary pathogen, but may result from bacteremia that seeds the gallbladder. Regardless, the patient began indefinite antibiotic therapy, as her gallstones posed as persistent nidi for infection. This case highlights the rare possibility of silent GBP as a cause for worsening ascites, as well as the therapeutic dilemma of retained peritoneal gallstones in a poor surgical candidate.



[1835] **Figure 1.** Gallstones residing in intact gallbladder on left, compared to subsequent scan on the right demonstrating gallstones in patient's pelvis.

S1836

Six Years of Evolution With Portal Cavernomatosis and Portal Biliopathy

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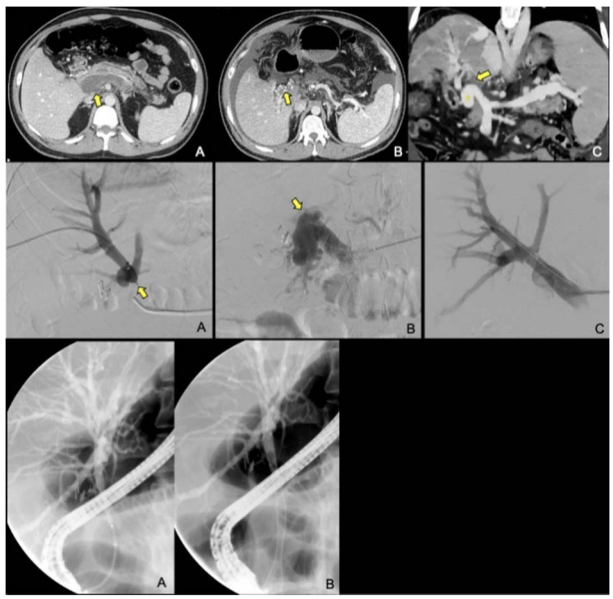
Introduction: In patients with Portal Biliopathy (PB), It is paramount to engage several branches of gastroenterology to interject the aggressive evolution of this condition.
Case Description/Methods: Our patient is a Man of 45 years old. Medical history of pancreatic divisum, chronic pancreatitis, biliary litho extraction, cholecystectomy, use of endoprosthesis, and pancreatic conduct stenosis. On CT scans in February 2015 he presented a pancreatitis Baltazar E, generating a perfusion defect, and multiple collaterals, in Abril 2015 portal cavernomatosis, and by January 2018 the collateral veins provoked biliary ectasis. During the next 2 years, he presented two events of mild cholangitis. To solve the choledochal impingement, on October 8, 2019, it took place a portal vein angioplasty with a balloon. The direct extrahepatic portal vein pressure was 30 mmHg pre-plasty, diminishing to 22 mmHg post-plasty. Posterior to the intervention, he had a new severe healthcare-associated colangitis plus acute pancreatitis, with a 7 mm dilatation only in the intrahepatic biliary pathway. To improve the biliary stasis, by CPRE it was placed a metallic stent covered with a Wallflex 10 mm x 8 cm. By February 2020, a CT scan still showed a permeable porta in the confluence, yet the portal prosthesis was not visible. Finally in September 2021, without clinic traits of portal hypertension, events of cholestasis, and improvement in hepatic function. (**Figure)**
Discussion: In the biliary tract, venous drainage passes through two venous plexus named, the epicholedochal venous plexus of Saint and the paracholedochal venous plexus of Petren. In portal biliopathy, changes are a result of dilatation of veins of the plexus of Saint, causing bile duct impingement. It has been hypothesized that this lead to fibrous scarring of porta hepatis, and ischemic injury to the bile ducts resulting in stricture formation and caliber irregularity with upstream dilatation. Several studies have shown that bile duct changes occur in 81–100% of patients with extrahepatic portal vein obstruction, but only 5–30% of patients present symptoms of biliary obstruction. In adults conditions such as thrombophilia, intra-abdominal infection, malignancy, myeloproliferative disorders and cirrhosis have been implicated PB is mainly a feature of extrahepatic portal venous obstruction, patients presenting with symptoms require an integrated management addressing biliary obstruction and portal hypertension in tandem. However, the relief of both conditions is only temporary.



[1836] **Figure 1. Top**. Initial axial CT scan following the known event of acute pancreatitis, showing acute splenic-portal venous thrombosis demonstrated by unenhanced and distended portal vein (yellow arrow in a). The middle image three months later shows the formation of small collateral vessels in the hepatic hilium, (yellow arrow in b), and also notes new-onset ascites due to impaired liver function. Coronal reformat image two years later, points to the focal area of chronic portal thrombosis, yellow arrow in (c) with more prominent appearance collateral vessels "cavernomatosis" (asterisk), several perfusion defects in the liver parenchyma, and marked splenomegaly. **Middle**. DSA images, in (a) transhepatic percutaneous venography with opacification of intrahepatic portal vasculature with a sudden interruption in the site of thrombosis (yellow arrow in a). (b) splenic approach with opacification of the extrahepatic portal vein and aneurismatic collateral vessels due to cavernomatosis and upstream thrombotic interruption (yellow arrow in b). (c) final imagen after portal vein angioplasty demonstrates adequate and complete opacification of the portal vein and its branches. **Bottom**. ERCP in (a) shows intrahepatic bile duct dilatation and impingement and (b) after stent placement with diminished dilatation.

S1837

Successful Use of Minor Papillotomy in Pancreas Divisum With Walled-Off Necrosis: Ready for Prime Time?

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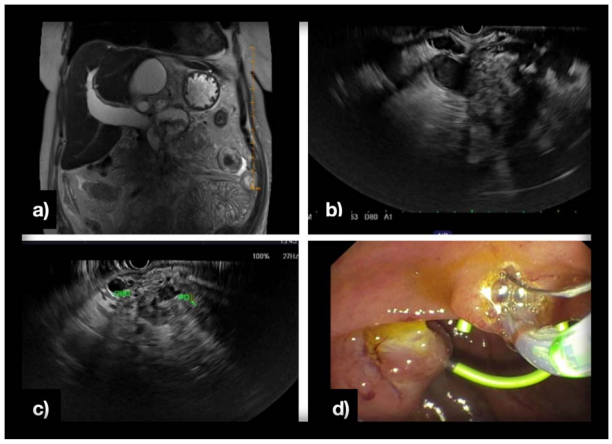
Introduction: Despite the relative rarity of congenital anomalies of the pancreas, they can result in some clinical symptoms, such as recurrent abdominal pain, recurrent acute pancreatitis (RAP), or chronic pancreatitis. Nearly 8-10% of the population experience pancreatic congenital anomalies. The most common is the pancreas divisum (PD). To successfully treat a symptomatic PD, minor papillotomy (MiP) is the mainstay of endoscopic therapy, especially when access is difficult via the major papilla. Here we present a case of PD complicated with necrotizing pancreatitis successfully treated with MiP and pancreatic stenting.
Case Description/Methods: A 56-year-old female with prior history of recurrent acute pancreatitis (RAP) presented with intermittent epigastric pain, nausea, and vomiting for one month. The patient admitted to the social use of alcohol only and no tobacco use. A physical exam showed stable vital signs and mild epigastric tenderness. Laboratory work showed mild elevation of WBC (12 x 10^9/L) and serum lipase 5 x of ULN. Liver & kidney functions were within normal limits. Magnetic resonance imaging (MRI) showed multiple pancreatic fluid collections (PFC) (**Figure A**) and evidence of incomplete pancreatic divisum (PD). Endosonography showed a large pseudocyst between the liver and stomach and walled-off necrosis around the pancreatic head, invading a thrombosed main portal vein (PV) (**Figure** B). Also, EUS confirmed incomplete PD (**Figure** C). Transluminal drainage of the WON was not attempted because of the PV invasion. Endoscopic retrograde cholangiopancreatography (ERCP) was successfully performed, with major & minor pancreatic papillotomy & plastic stenting (**Figure** D). Biliary cannulation showed a dilated bile duct but was otherwise unremarkable. The patient was continued on supportive therapy and later required percutaneous drainage of the pseudocyst between the liver and stomach. A subsequent MRI showed significant WON resolution three months later, and the patient was convalescing well.
Discussion: Minor papillotomy (MiP) is the cornerstone of endoscopic therapy for symptomatic PD (RAP, chronic pancreatitis, or chronic abdominal pain). MiP as an adjunct treatment for walled-off necrosis (WON) has not been described in the few studies that assessed endoscopic therapy for symptomatic PD. MiP and endoscopic therapy use in PD with WON might benefit this subset of patients, but that requires further investigation.



[1837] **Figure 1.** a) Magnetic resonance T2-coronal image showing walled-off necrosis (WON) invading a thrombosed portal vein and a pseudocyst between the liver and stomach. b) Endosonography image showing the walled-off necrosis (WON) invading the portal vein. c) Incomplete divisum with the pancreatic duct coursing across the bile duct toward the minor papilla on EUS. d) Endoscopic treatment with major papillotomy (below) is done, and minor papillotomy MiP (above) is undertaken with a pull-type sphincterotome.

S1838

Spontaneous Gastro-pancreatic Fistula: A Rare Complication of Acute Pancreatitis

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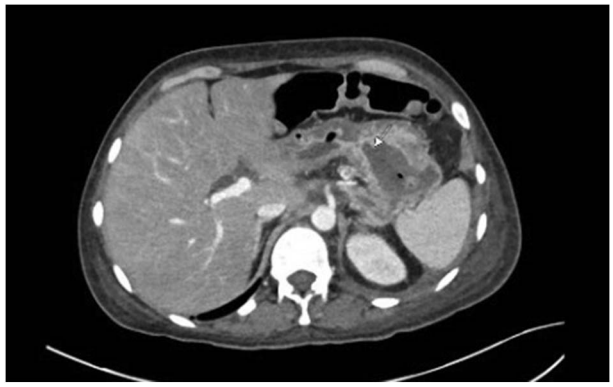
Introduction: Although Gastrointestinal fistula is a well-recognized complication of acute pancreatitis, it has been rarely reported. Here we present a rare case of spontaneous gastro-pancreatic fistula following acute pancreatitis.
Case Description/Methods: 42 y/o female with PMH of SLE with a recent prolonged hospitalization for acute drug-induced pancreatitis with pseudocyst came to ED with fever, abdominal pain, nausea, and vomiting. She was tachycardic, had leukocytosis, and was positive for COVID-19. CT Scan A/P showed multiple infected peripancreatic collections with communication of the left upper quadrant collection with the gastric lumen (**Figure)**. The patient was hospitalized, Kept NPO, and started on fluids and antibiotics. IR evaluated and put 2 pigtail catheters for drainage of peripancreatic collections. The tip of the pigtail catheter in the left peripancreatic/retroperitoneal collection was in the gastric lumen. The surgery team recommended continuing with conservative treatment with parenteral nutrition, and IV antibiotics as the patient were nontoxic with no signs of free perforation, and pancreatitis would more likely erode a staple or suture line and would put the patient at further risk of free perforation if repair attempted. IR was successful in pulling the drain out of the gastric lumen on the second attempt to allow gastric perforation to heal. Antibiotics were upgraded as per the culture and sensitivity results of the drain fluid. Repeated multiple bedside leak tests and CT scans with oral contrast continue to be positive for patent gastro-pancreatic fistula. Pigtails catheter continues to drain significant necrotic collection. The patient continues to be hospitalized and is being managed conservatively with Parenteral nutrition, and IV antibiotics.
Discussion: Fistula of the GI tract following acute pancreatitis can be caused by multiple reasons. Necrosis of the bowel may occur concomitantly with the pancreatic or peripancreatic tissue. Furthermore, enzyme-rich fluid and necrosis can lead to vascular thrombosis, which compromises the blood supply of the segmental GI tract, eventually leading to bowel necrosis. GI fistulas are more common in patients with necrotizing pancreatitis with infected pancreatic necrosis. Despite pharmacologic suppression of pancreatic exocrine secretion and advances in endoscopic and percutaneous therapeutic techniques, pancreatic fistula continues to be a source of morbidity and mortality following pancreatitis and requires multidisciplinary treatment.



[1838] **Figure 1.** Peripherally enhancing fluid collection in the left upper quadrant/gastric wall. There is a communication between this collection with the gastric lumen.

S1839

Superior Mesenteric Vein Aneurysm Mimicking Pancreatic Head Mass

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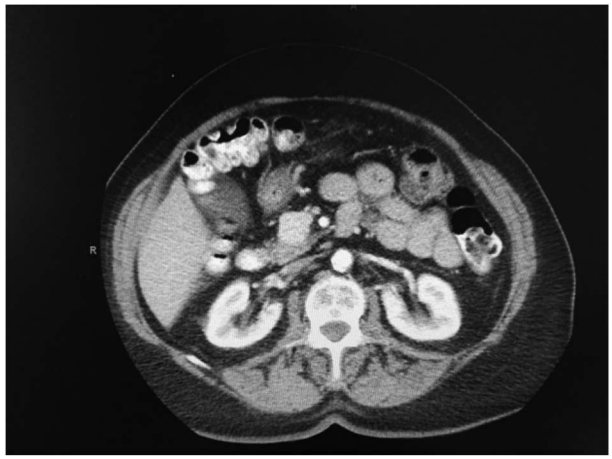
Introduction: Superior mesenteric vein (SMV) aneurysms are a rare entity which was first described in the literature in 1982. Aneurysms in the portal venous system, which include extrahepatic portal, splenic, and superior mesenteric veins, represent approximately 3% of all venous aneurysms with a reported prevalence of 0.43%. With the advancement of abdominal imaging, SMV aneurysms are more frequently being identified. We report a case with a patient who was found to have to have a SMV aneurysm mimicking as a pancreatic head mass for the workup of abdominal pain.
Case Description/Methods: Our patient is a 73-year-old female who was found to have a pancreatic head mass (2x2.4x1.9cm) that was found on imaging for workup of acute abdominal pain (**Figure**). Patient denied any weight loss and LFT's were found to be unremarkable. For further workup, patient had EUS performed which showed evidence of a superior mesenteric aneurysm measuring 2.4 x 2.1 cm that was mimicking as a pancreatic head mass. No further intervention performed and patient's abdominal pain resolved.
Discussion: Aneurysms in the portal venous system, which include extrahepatic portal, splenic, and superior mesenteric veins, are rare as they represent approximately 3% of all venous aneurysms with a reported prevalence of 0.43%. Etiologies for superior mesenteric aneurysms are not completely understood. A congenital etiology includes an incomplete regression of the caudal part of the right vitelline vein. Acquired causes of SMV aneurysms reported in the literature include portal hypertension secondary to chronic liver disease, pancreatitis, trauma and previous surgical intervention. Of these, portal hypertension is considered the most common cause of SMV aneurysms. This is due to intimal thickening with compensatory medial hypertrophy with fibrous tissue leading to weakening of the venous wall making it more susceptible to aneurysmal dilatation. Complications of SMV aneurysm includes thrombosis, biliary tract obstruction, inferior vena cava obstruction, duodenal compression, and rarely aneurysm rupture (up to 2.2% of cases). In asymptomatic patient's, conservative management with follow-up imaging to assess aneurysm size is recommended. If the patient is symptomatic or has complications of a SMV aneurysm, surgical or interventional radiology intervention may be needed which may include portal venous shunt, aneurysmorrhaphy, stent graft placement and coil embolization.



[1839] **Figure 1.** EUS showing evidence of a superior mesenteric aneurysm.

S1840

The Importance of Cholangioscopic Biopsies: An IgG4-Related Sclerosing Cholangitis Story

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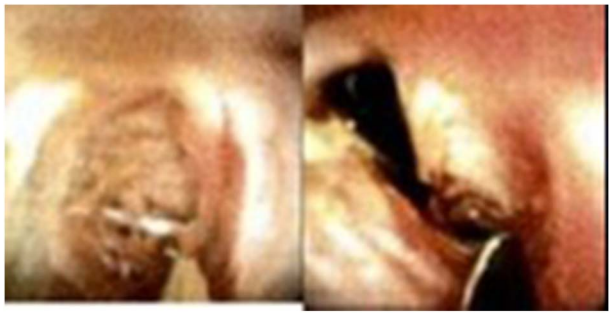
Introduction: IgG4 related sclerosing cholangitis (IgG4-SC) has generally been associated with autoimmune pancreatitis, retroperitoneal fibrosis, and sialadenitis. However, there are rare cases involving only the biliary tree. In addition, IgG4-SC can be easily confused with cholangiocarcinoma and primary sclerosing cholangitis (PSC). IgG4 serum levels more than 135 suggest IgG4-SC, but lower levels do not rule out the disease. Here, we present an atypical case of IgG4-SC.
Case Description/Methods: 68-year-old male with past medical history of alcohol misuse disorder presented for pruritis, scleral icterus, and 40 pounds of weight loss in three weeks. On admission, notable labs were AST 158, ALT 81, ALP 713, total bilirubin 5.8, and direct bilirubin 4.9. IgG was 3203 and IgG4 was 113. Remainder of the work up was negative. Magnetic resonance cholangiopancreatography showed isolated left intrahepatic biliary dilation. Endoscopic retrograde cholangiopancreatography (ERCP) showed segmental strictures at the common hepatic duct and left intrahepatic duct. Cholangioscopy showed a fibrotic intraluminal growth in the upper third of the common bile duct, which was unable to be traversed. Biliary brushings showed only acute inflammation (**Figure**). A plastic biliary stent was placed for decompression and a percutaneous internal-external biliary drain was performed to facilitate subsequent rendez-vous ERCP. Repeat cholangioscopy showed scarring and fibrosis of the entire biliary tree and

segmental stricturing at the hilum and left intrahepatic duct. Cholangioscopy-guided forceps biopsies showed more than ten IgG4 cells per high powered field indicating IgG4-SC. Patient was started on high dose corticosteroids with improvement of his symptoms and lab abnormalities.
Discussion: Awareness of IgG4-SC is important as it can mimic cholangiocarcinoma and PSC. Our patient presented with obstructive jaundice and isolated dilatation of the biliary tree with a normal serum IgG4 suggestive of underlying malignancy. However, he also had elevation of total serum IgG, which was suspicious for an underlying autoimmune disorder. Biliary brushings can exclude malignancy but have a poor sensitivity for the diagnosis of IgG4-SC (36%). The most useful modality of diagnosis is cholangioscopy-guided forceps biopsy as this is positive in about 88% of specimens. Thus, IgG4-SC should be considered in patients with an atypical presentation of obstructive jaundice with incongruent lab and imaging findings, as this condition is treatable.



[1840] **Figure 1.** Left picture shows common hepatic duct stricturing and mass on cholangioscopy. Right picture shows left intrahepatic duct stone in light of stricturing on cholangioscopy.

S1841

The Curious Case of CA 19-9 in Mirizzi Syndrome

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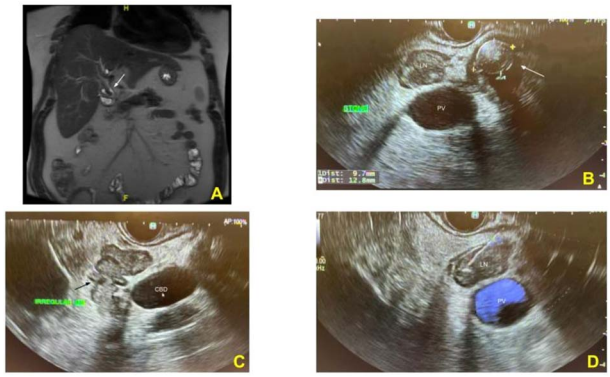
Introduction: Mirizzi Syndrome (MS) is a rare complication of gallstone disease, caused by an obstructing stone in the cystic duct or the gallbladder neck, leading to obstruction of the common hepatic duct. This case discusses the utility of EUS in improving diagnostic yield of MS and the importance of cancer screening in these patients.
Case Description/Methods: A 44-year-old male with a history of gastritis presented with right upper quadrant (RUQ) abdominal pain, jaundice, and scleral icterus for one week. On exam, the patient was afebrile with mild RUQ abdominal tenderness. Labs showed a total bilirubin of 5.4 mg/dL, AST of 66 U/L, ALT of 228 U/L, and a total alkaline phosphatase of 159 U/L. Ultrasound demonstrated intra- and extrahepatic biliary dilation with suggestion of a shadowing stone within the common bile duct (CBD). The gallbladder wall was thickened with trace pericholecystic fluid. MRCP showed a 15 mm calculus presumed to be in the CBD, with a 16 mm CBD dilation. ERCP showed a filling defect in the midportion of the CBD, and a CBD stent was placed. Due to these findings, a CA 19-9 antigen was drawn, measuring 3038 U/mL. EUS was then performed, which visualized a cystic duct stone partially extending into the CBD– a finding consistent with MS. An enlarged lymph node was visualized in the porta hepatis region. FNA of the lymph node as well as CBD brushings from the ERCP were both negative for malignant cells. The patient was discharged and underwent a cholecystectomy two weeks later. Surgical findings were consistent with MS and a 20 mm stone was removed with the gallbladder. The intraoperative frozen section of the gallbladder was negative for malignant cells. (**Figure)**
Discussion: Initial evaluation for MS should include an ultrasound or CT scan, followed by MRCP. The pooled sensitivity of MRCP for detecting CBD stones is around 87%. While MRCP is often used to diagnose MS, the anatomy may not be well visualized due to the proximity of the cystic duct to the CBD. EUS has a pooled sensitivity of 97% in detecting a CBD stone and can better visualize the biliary tract anatomy, aiding in diagnosis. Diagnosis of MS prior to surgery can be helpful in avoiding biliary tract injuries. MS evaluation should also include screening for malignancy, as MS has an association with gallbladder cancer. While elevated CA 19-9 levels are concerning for malignancy, they can also be seen in chronic benign biliary obstruction. It is important to obtain a CA 19-9 level when suspecting MS, both before and after stone removal.



[1841] **Figure 1.** A. MRI: A T2-weighted image showing a 15 mm filling defect (white arrow) within the cystic duct, partially extending into the common bile duct, consistent with an obstructing stone. Intrahepatic and extrahepatic biliary dilation is also seen. B. Endoscopic ultrasound: An ovoid, hyperechoic structure, measuring 11 mm x 15 mm, with shadowing suggestive of a gallstone at the level of the cystic duct and compression of the common bile duct. C. Endoscopic ultrasound: An irregularly contracted gallbladder (black arrow) with adjacent dilated common bile duct. D. Endoscopic ultrasound: Fine needle aspiration of an enlarged lymph node with well defined margins in the porta hepatis region, measuring 20 mm x 15 mm in maximal cross-sectional diameter. Color Doppler imaging was utilized prior to needle puncture to confirm a lack of significant vascular structures within the needle path.

S1842

Tertiary Lymphoid Structure Mimicking Pancreatic Mass

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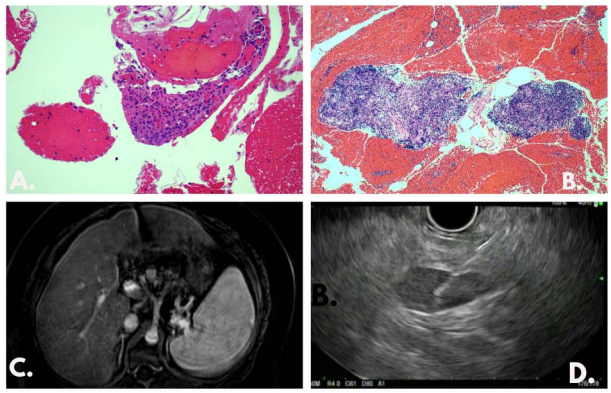
Introduction: Tertiary lymphoid structures (TLS) are defined as non-congenital ectopic lymphoid tissues that can be found in non-lymphoid organs and form in response to chronic inflammation. TLS's etiology as well as it's physiological and pathological roles remain a highly debated topic.
Case Description/Methods: An asymptomatic 50-year-old obese diabetic female with a history of biopsy proven cirrhosis secondary to non-alcoholic fatty liver disease (NAFLD) presented to the clinic after a routine hepatocellular carcinoma (HCC) surveillance MRI. A 1.3 cm enhancing nodule at the neck of pancreas was noted. Due to concern for pancreatic cancer an endoscopic ultrasound with fine needle biopsy (EUS-FNB) was planned. During EUS an 11.4 mm by 8.5mm oval mass with irregular margins was identified in the pancreatic body. Fine needle biopsy with 22-gauge needle was performed using trans gastric

approach. An enlarged lymph node was identified in peripancreatic region measuring 24.2 mm by 10.9 mm, an additional fine needle biopsy was obtained. Pathology of pancreatic mass and lymph node later revealed fragments of lymphoid tissue consistent with reactive lymph node with single cluster of cytologically mildly atypical cells however no evidence of metastatic malignancy was appreciated. Immunohistochemical staining was positive for CD3 and CD20. CD56, chromogranin, synaptophysin, CD10, e-cadherin and AMACR stains were negative. Flow cytometry analysis was ordered. It demonstrated no evidence of a lymphoproliferative disorder. A three-month post procedure MRI was obtained which demonstrated a 1.6 cm nodule at neck of the pancreas increased in size when compared to prior image. **(Figure)**

Discussion: In immunology, TLS are a highly debated topic as to whether they serve as mediators of protective or pathologic immune responses in certain chronic inflammatory diseases and in the regulation of immune responses. When associated with both primary and or metastatic tumors they're known as tumor-associated TLS (TA-TLS). In cancer their presence is associated with prolonged increased rates of disease-free survival. When identified flow cytometry should always be ordered in order to rule out a lymphoproliferative disorder. TLS and its implications are still an area of active study, clear guidelines in terms of management are still being developed. Furthermore as to whether they represent premalignant lesion is still unknown. Our patient remains asymptomatic and continues to be monitored with serial imaging.



[1842] **Figure 1.** A. Magnification 200x. Small fragment of benign pancreatic acinar cells near lymphoid cluster. B. Magnification 100x. Aggregates of lymphoid tissue with intervening capillaries representing lymph node like structure within pancreas. C. MRI Abdomen with contrast showing 1.3 cm enhancing nodule at neck of pancreas. D. EUS. Fine needle aspiration of 11.4 mm by 8.5 mm intrapancreatic mass.

S1843

The Path of Least Resistance: A Case of the Lemmel

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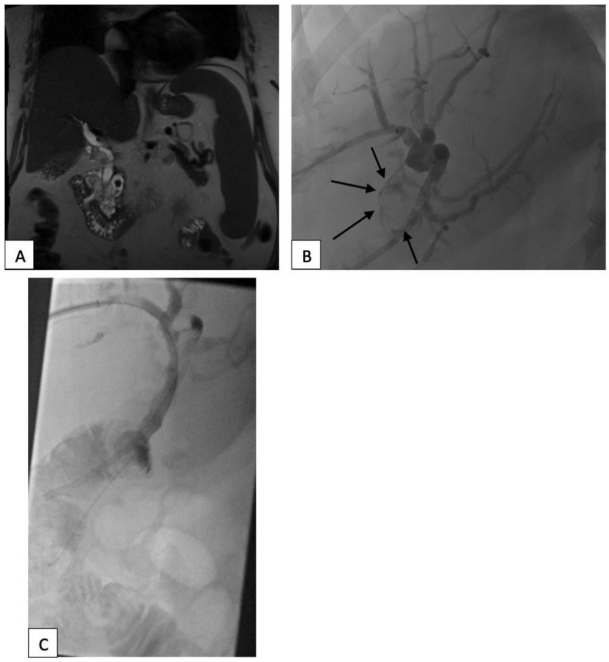
Introduction: Lemmel Syndrome is defined as obstructive jaundice caused by a periampullary duodenal diverticulum (PAD) compressing the intrapancreatic common bile duct (CBD) with resultant bile duct dilatation and obstruction. Management is based on symptomatology, ranging from conservative management to endoscopic extraction of retained food debris, or surgery. We present a unique case of Lemmel Syndrome in a patient who suffered from recurrent obstructive jaundice and underwent multiple endoscopic and ultimately, surgical interventions

Case Description/Methods: A 56-year-old obese female with a history of alcohol use disorder initially presented with right upper quadrant abdominal pain for 2 days. Physical exam was remarkable for right upper quadrant tenderness. Labs revealed total bilirubin 3 mg/dL. Complete blood count was within normal limits. Viral hepatitis panel and CA 19-9 were unremarkable. Ultrasound gallbladder showed cholelithiasis without cholecystitis with mild CBD dilatation. MRCP revealed a large 6 cm multiloculated mass centered near the pancreatic head/ampulla of Vater, with internal heterogeneity and multiple air-fluid levels and internally contiguous with duodenal lumen. She was given broad spectrum antibiotics and an upper endoscopy with endoscopic ultrasound was performed revealing a large, non-bleeding periampullary diverticulum impacted with food debris. The food debris was extracted. Multiple such procedures were needed in a short period of time. She continued to suffer from recurrent obstructive jaundice for several months, requiring a cholecystectomy, Roux-En Y hepaticojejunostomy, and gastric antrum resection with Roux-En Y BillRoth II gastrojejunostomy in an attempt to divert food debris away from the diverticulum. She has now developed stenosis at the hepaticojejunostomy requiring ERCP with dilation of the anastomosis, and stenting, as well as percutaneous transhepatic cholangiography, internal external biliary drain placement, lithotripsy of hepatic duct stones, and covered metal stent placement. **(Figure)**

Discussion: Treatment of Lemmel Syndrome incorporates a variety of modalities. A conservative approach is usually preferred, but invasive intervention may sometimes be required. The most definitive approach would be a Whipple procedure. However, in an obese patient, this would be associated with significant morbidity and mortality risk. Our case portrays an unusual approach which offered the patient a less invasive combined endoscopic, interventional radiology and surgical treatment



[1843] **Figure 1.** A: Initial MRI which shows duodenal diverticulum with biliary dilation consistent with Lemmel Syndrome B: Initial Cholangiogram: Stent within left hepatic duct as well as central right hepatic duct appearing goblet shaped with stones (as shown with arrows) C: Subsequent imaging reveals right hepatic duct status post stone extraction, appearing relatively normal (compare with Image B).

S1844

The Gallbladder Houdini: A Rare Presentation of Eosinophilic Cholecystitis and Gastroenteritis

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Introduction: Eosinophilic gastroenteritis (EG) is an inflammatory condition with eosinophilic infiltration of the GI tract, but biliary tract involvement is rare. We report a rare case of EG with concurrent eosinophilic cholecystitis.
Case Description/Methods: A 24 y/o WF with a history of Samter's triad (nasal polyposis, asthma, aspirin allergy) managed with dupilumab, presented to the ER with chronic LUQ abdominal pain, N/V, dysphagia, diarrhea, and weight loss. Lab revealed normal LFTs and an elevated WBC to 13.6k with 9.2% eosinophils (eos). RUQ US showed fatty liver and cholelithiasis with normal caliber CBD. She underwent laparoscopic cholecystectomy with surgical pathology revealing early acute on chronic cholecystitis and cholelithiasis with extensive eosinophilic inflammation. She noted persistent symptoms with follow up labs showing increasing WBC to 26k with 67% eos. Repeat CT scan was unremarkable. Initial EGD showed gastritis with distal esophageal biopsies showing 100 eos/hpf. She was started on PPI. Repeat EGD showed gastritis and duodenitis with biopsies remarkable for eosinophilia of the stomach and proximal small bowel. Colonoscopy with TI intubation was endoscopically normal with normal biopsies of the TI and colon. She was started on PPI for eosinophilic esophagitis and a prednisone taper for eosinophilic gastroenteritis with symptom resolution.
Discussion: The prevalence of EG is highest in children but can occur in adults often with concomitant atopic disease in the 3rd-5th decades. The clinical presentation varies, depending on the depth of inflammation (mucosal, muscular, or serosa) and location of involvement and may include abdominal pain, N/V, diarrhea, and weight loss. Diagnosis is based on pathology obtained during endoscopy or surgery. It is prudent to obtain multiple biopsies given patchy disease and consider transmural biopsies to identify processes limited to the muscular layer. Imaging can show non-specific inflammation. Peripheral eosinophilia is present in ~80% of cases. The mainstay of therapy is corticosteroids, with a 90% symptomatic response. Azathioprine can be considered as a steroid sparing agent. Eosinophilic cholecystitis has rarely been reported in the literature but often in association with other illnesses. We present a unique case of eosinophilic cholecystitis in a patient with concurrent eosinophilic gastroenteritis. Upon review of the literature only 5 other similar cases have been described.

S1845

The Undercover Underdog of Pancreatic Cysts: Benign Lymphoepithelial Cyst

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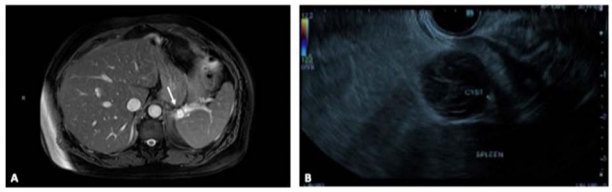
Introduction: Pancreatic lymphoepithelial cyst (LEC) is a rare non-neoplastic, true pancreatic cyst accounting for only 0.5% of all pancreatic cysts. We present a case of a pancreatic LEC in which confounding variables and its nonspecific features led to its challenging diagnosis.
Case Description/Methods: A 57-year-old male presented with epigastric pain, nausea, fever and general malaise. Laboratory work-up was remarkable for anemia and thrombocytopenia. Liver enzymes and pancreatic markers were normal. Tick panel was pursued which came back positive for anaplasmosis phagocytophilum. Patient was treated with doxycycline and discharged. He presented a few days later reporting the recurrence of his epigastric pain, nausea and vomiting. He was evaluated with a computed tomography (CT) scan of the abdomen and pelvis which revealed a pancreatic tail cyst. This was further investigated with magnetic resonance cholangiopancreatography (MRCP) which confirmed an 18 x 12.9 mm thin wall cystic lesion overlying the posterior margin of the pancreatic tail medial to the spleen (**Figure** 1A). Endoscopic ultrasound (EUS)-guided fine needle biopsy (FNB) was performed of the pancreatic lesion (**Figure** 1B). Pathology findings were consistent with a benign LEC. The patient is currently being managed conservatively while surgical resection of the cyst is under discussion.
Discussion: Pancreatic LECs commonly present in middle aged males with abdominal pain but also with nonspecific complaints such as nausea and malaise. Therefore, when pancreatic cysts are identified on imaging with such complaints, pancreatic LECs should be part of the differential diagnosis. Further investigation with EUS and tissue sampling is essential due to the prevalence of pancreatic cysts with premalignant and malignant potential. EUS-guided fine needle aspiration (FNA) is the diagnostic choice but FNB may be preferred depending on the fluid viscosity for adequate tissue sampling. Symptomatic patients with uncomplicated cysts can undergo cyst enucleation but resection is preferred when its malignant potential is uncertain. In asymptomatic patients, observation is optional once the diagnosis of pancreatic LEC is confirmed.



[1845] **Figure 1.** (A) Magnetic resonance cholangiopancreatography (MRCP) of the pancreatic tail cyst. (B) Endoscopic ultrasound (EUS) of the pancreatic tail cyst.

S1846

The GIST of Abdominal Pain With Jaundice

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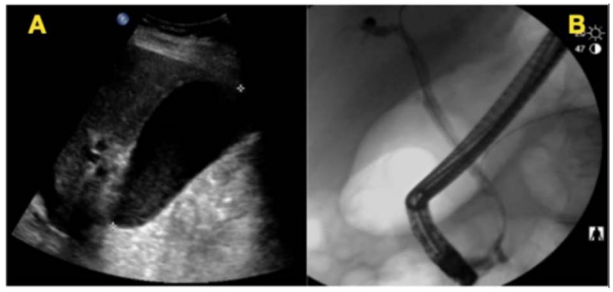
Stony Brook Southampton Hospital, Southampton, NY.

Introduction: Gastrointestinal Stromal Tumors (GISTs) are subepithelial neoplasms characterized by the expression of KIT protein (CD117 antigen). They are the most common non-epithelial neoplasms of the GI tract, often found in the stomach and distal small intestine, with small GISTs often undiagnosed. GISTs have malignant potential, but nodal involvement is rare, explaining why surgical resection is usually first line treatment. Adjuvant treatment following resection or neoadjuvant treatment in the setting of unresectable/metastatic GISTs may produce an improved median survival. We present a patient with painful jaundice found to have GIST.
Case Description/Methods: A 70-year-old man with a history of lung cancer, coronary artery disease, and nephrolithiasis presented with waxing and waning diffuse abdominal pain occasionally radiating to the back for three weeks. Associated symptoms included increased eructation, decreased appetite, generalized weakness, steatorrhea, rust-colored urine, and jaundice. The pain worsened with fatty meals and improved with defecation. The patient appeared jaundiced with conjunctival icterus. Abdomen was soft, non-distended with right upper quadrant tenderness, voluntary guarding and hypoactive bowel sounds. Labs revealed a cholestatic pattern of liver enzymes indicating obstruction. Of note, the CA 19-9 level was 142. Biliary imaging was performed (**Figure** A), which showed peripancreatic mass and severe biliary stricture in the lower third of main bile duct (**Figure** B). Pathology results of fine needle biopsy indicated low-grade spindle cell neoplasm consistent with GIST, CD117, CD34 and smooth muscle actin positive, S-100 and desmin negative. Given the location of the tumor, elective robotic pancreaticoduodenectomy was performed two months later with nodal resection.
Discussion: Historically, the presentation of painful jaundice is associated with extrahepatic cholestasis including intrinsic and extrinsic tumors (cholangiocarcinoma, pancreatic cancer), choledocholithiasis, primary sclerosing cholangitis, acute pancreatitis, sphincter of Oddi dysfunction or biliary tract strictures following invasive procedures. The unusual location of this patient's GIST led to differential diagnoses that did not include his ultimate diagnosis. This case report highlights the presentation of an often undiagnosed cancer, GIST, especially given its unique location in the GI tract. It is vital that as clinicians we keep a broad differential while performing workups of abdominal pain with jaundice.



[1846] **Figure 1.** Biliary Imaging. A: RUQ abdominal ultrasound demonstrating hydropic gallbladder 11.5 cm in length with sludge. B: ERCP fluoroscopy showing evidence of biliary duct stricture.

S1847

The Enigmatic Triangle of Hypertriglyceridemia-Induced Pancreatitis and Its Association With Diabetic Ketoacidosis and Resulting Pancreatic Pseudocyst: A Case Report

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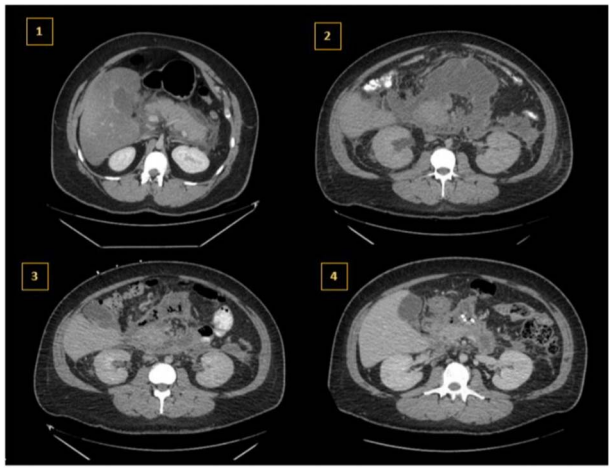
Introduction: Acute pancreatitis (AP) presents with acute epigastric pain radiating to the back and elevated pancreatic enzymes. The most common causes are gallstones and alcohol. Hypertriglyceridemia-induced pancreatitis (HTGP), causes 14% of all AP cases, rising to 56% in pregnancy. We present a unique case of HTGP complicated by diabetic ketoacidosis (DKA) and pancreatic pseudocyst.
Case Description/Methods: A 27-year-old male with type 1 diabetes mellitus, hyperlipidemia, ADHD, and depression presented with sudden onset constant, sharp, diffuse abdominal pain with radiation to the back. He reported nausea, dry heaving, increased thirst and urinary frequency. Relevant family history includes familial hypertriglyceridemia. The patient was found to have DKA, lactic acidosis, leukocytosis, and hypocalcemia. Triglycerides (TG) and lipase/amylase were elevated. Computed tomography (CT) of the abdomen showed AP with fat stranding (**Figure**). He received fluids, an insulin drip, and was transferred to the intensive care unit. Patient met criteria for plasmapheresis and was transferred to a tertiary care center. On follow up, repeat CT three weeks post-presentation indicated a developing pseudocyst. CT four weeks later showed anterior pancreatic pseudocyst with a decrease of peripancreatic fluid collection and CT seven weeks later showed decreased peripancreatic and retroperitoneal fluid with 2 stents extending from stomach to collection.
Discussion: TG > 1000 is required for diagnosis of HTGP. TG 1000-1999 carries a 5% risk of developing HTGP, increasing to 20% in TG >2000. Patients are typically young, male, obese, and diabetic. AP may precipitate or complicate DKA. DKA can mask AP, occurring in approximately 10-15% of cases. In 100 consecutive cases of DKA, 11 had a CT confirmed AP with most caused by hypertriglyceridemia and alcohol. Hypocalcemia, lactic acidosis or sepsis necessitate intensive therapy including therapeutic plasma exchange (TPE), which replaces plasma with colloid solution. HTGP is managed with supportive care and insulin/TPE until TG < 500. TG< 200 prevents recurrence and is managed with pharmacotherapy and lifestyle modifications. HTGP can cause AP and should be considered in the differential for acute abdominal pain, especially in younger males. It can be complicated by DKA and pancreatic pseudocyst, leading to a more severe presentation and necessitates the addition of TPE to insulin. Prompt recognition and management of HTGP is imperative in preventing complications and expediting recovery.



[1847] **Figure 1.** Progression of AP to development of anterior pancreatic pseudocyst and stent placement. 1: AP with fat stranding. 2: Developing anterior pancreatic pseudocyst. 3: Full development of pancreatic pseudocyst with ring enhancing. 4. Decreased peripancreatic and retroperitoneal fluid with 2 stents.

S1848

Testosterone Supplement-Induced Pancreatitis

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Introduction: Common causes of acute pancreatitis (AP) in the United States are gallstone pancreatitis and alcohol use. However, about 2% of AP cases are attributed to Drug-induced pancreatitis (DIP). Though infrequent, the pathology is associated with substantial morbidity and mortality, which makes timely identification of the inflicting agent important. Supplements that enhance athletic performance and libido are commonplace in today's community and remain largely unregulated. The lack of understanding and awareness of the detrimental adverse effects of such drugs can affect physical and mental health.
Case Description/Methods: A 51-year-old gentleman with a history of chronic kidney disease Stage II, hyperlipidemia on statin, diverticulosis, and GERD on omeprazole presented to the ER with acute abdominal pain associated with nausea and vomiting for a day. The epigastric pain was described as 10/10 in intensity, sharp with radiation to the back and associated with 3 episodes of non-projectile, bilious, non-bloody vomiting. He is a non-smoker and drinks alcohol occasionally, his last drink being a can of beer 1 week prior. He denied recent infections, abdominal procedures, or trauma. On examination, his vitals were normal, and was in minimal distress. Tenderness was elicited in the epigastric region. Initial labs showed leukocytosis with left shift, elevated AST of 225, ALT of 202, LDH of 274, and a significant increase in levels of lipase >3000. A CT scan of the abdomen showed findings consistent with acute interstitial pancreatitis. Imaging and laboratory investigations were negative for biliary disease and IgG4 disease. On asking further history, he reported taking over the counter testosterone supplements to enhance his libido for 2 weeks. A diagnosis of DIP was made and was treated appropriately with aggressive hydration and pain management.
Discussion: DIP is attributed to four classes of drugs (I to IV) and steroids like testosterone are placed under Class I. The possible mechanism for causing AP include pancreatic duct constriction, cytotoxic and metabolic effects, accumulation of a toxic metabolite or intermediary, and hypersensitivity reactions. Prevalent use of testosterone in the male community for bodybuilding and increasing libido warrant the need to create awareness. Additionally, these drugs are difficult to study due to the varied consumption patterns, unknown origin and ingestion of high doses.

S1849

Watch out for Semaglutide: Potential Cause of Pancreatitis?

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Introduction: Recent clinical trials (STEP program) have shown Semaglutide to be the cornerstone in type 2 diabetes and weight management. Here, we present a case of acute pancreatitis in a patient who was recently started on semaglutide for diabetes mellitus.
Case Description/Methods: The 61-year-old female with the medical history of diabetes Mellitus type 2, hypertension, depression, and obesity (BMI 48.87) presented with one day of 10/10 upper quadrant abdominal pain radiating to her back associated with nausea. The patient denied any alcohol and recreational drug use. The patient had a cholecystectomy 5 years ago. On arrival, the patient's blood pressure was 178/80. Lab work was unremarkable except Liver function noted elevated: AST 324, ALT 140. Lipase level 4986. Her calcium level was normal, and her lipid panel was unremarkable. The patient was diagnosed with acute pancreatitis (AP) based on two out of three AP diagnostic criteria: characteristic abdominal pain, amylase and/or lipase ≥ 3ULN, and/or characteristic findings on pancreas imaging. GI was consulted. MRCP did not show any abnormalities or stones. She was continued on IVF and had a resolution of all symptoms the following day. She was also able to tolerate a solid diet well with no symptoms. Of note, the Patient was recently started on Ozempic 2 months ago. She was advised to discontinue Ozempic on discharge and discuss with PCP regarding other diabetes medications options
Discussion: GLP 1 agonists are becoming more favored among diabetes mellitus type 2 patients due to weight loss, less risk of hypoglycemia, and better compliance. GLP-1 agonists directly stimulate GLP-1 receptors in pancreatic islet beta cells and exocrine duct cells which may cause an overgrowth of the cells that cover the smaller ducts, thereby resulting in hyperplasia, increased pancreatic weight, duct occlusion, back pressure, and subsequent acute or chronic pancreatic inflammation. Acute pancreatitis was observed with semaglutide at rates similar to placebo during the SUSTAIN-6 trial. However Pancreatitis associated with GLP 1 agonists might present with atypical presentation, therefore the prevalence might be underreported, and other database studies have also shown conflicting results. Given the nature of observational studies, data could have been confounded, since patients with diabetes who have an indication for GLP-1RA therapy often have concomitant risk factors for pancreatitis (obesity, longer diabetes duration, and co-medication).

S1850

Vasoactive Intestinal Peptide Tumors (VIPoma) Missed on CT but EUS to the Rescue

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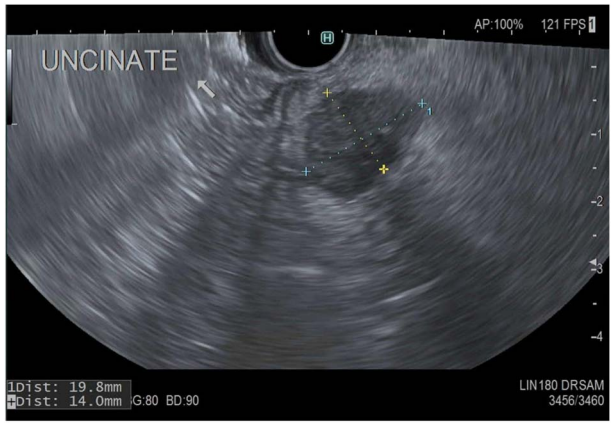
Introduction: Pancreatic neuroendocrine tumors (pNETs) are rare with >85% being non-functional. Amongst the functioning pNETs, vasoactive intestinal peptide tumors (VIPoma) account for < 2% pNETs. VIPoma secretes unregulated vasoactive intestinal peptide (VIP) hormones that can cause secretory diarrhea with electrolyte abnormalities with incidence rate of 0.05% to 2.0%. VIPoma are usually diagnosed with elevated serum VIP level. Imaging modalities are utilized to localize the tumor, usually with CT scans. Here we present a case of a diagnosed VIPoma patient with negative initial imaging, but lesions found with endoscopic ultrasound (EUS).
Case Description/Methods: 74-year-old female who has had recurrent urgent care visits for chronic diarrhea with dehydration for the past year. She was found to have elevated VIP (in the 900s) with hypokalemia and hypercalcemia, but CT abdomen and pelvis with IV contrast was negative for any lesions in the pancreas. Given the concern for a VIPoma, an EUS was ordered. EUS demonstrated a 19.8mm x 14.0mm hypoechoic mass in the uncinate process. Fine needle biopsy (FNB) was performed and revealed a well-differentiated neuroendocrine tumor that was WHO grade 1. Following diagnosis, the patient was

started on octreotide for symptomatic management with further workup of MEN1. The patient showed hepatic lesion concerning metastasis on follow up MRI of abdomen. Repeat EUS with FNA of hepatic lesion showed pathology result suggestive of metastasis.**(Figure)**

Discussion: CT is used as primary method to localize the lesion, given that it is readily available and takes shorter time to obtain imaging. Though one study reported sensitivity of CT being >80%, another study reported CT sensitivity of 58%. As also demonstrated in this case, it can be a challenge to localize VIPoma with a single imaging test, and may require multi-modality approach, including CT, MRI, PET, and EUS. Modality used in our study, EUS, has shown detection rate of approximately 90%. To demonstrate this, EUS was able to detect lesions in 33% of the patients that tested negative on initial CT. It is important to keep in mind that VIP levels may be false negative given that VIPoma may secrete VIP intermittently. This case and literature review implore that in the setting of clinical suspicion with or without VIP elevation, even if CT is negative, it does not necessarily rule out VIPoma and further imaging should still be considered.



[1850] **Figure 1.** EUS showing uncinate mass.

S1851

Trapped Lung From Pancreatico Pleural Fistula: A Rare Complication of Acute Pancreatitis

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Introduction: Pancreaticopleural fistula (PPF) is a rare complication which occurs in approximately 0.4% of patients after pancreatitis [1]. Pancreatic fluid can fistulalize through the esophageal or aortic hiatus or directly through the diaphragm resulting in a unilateral or bilateral pleural effusion [1,4]. Management of PPF is based on clinical manifestations.

Case Description/Methods: A 46-year-old woman presented to the emergency department three weeks after hospitalization for gallstone pancreatitis with severe abdominal pain and shortness of breath. Physical exam was pertinent for tachycardia, diminished breath sounds over the left lung, and epigastric tenderness. Labs revealed normal lipase of 55 and elevated serum amylase to 238. Computed tomography (CT) scan of the chest, abdomen, and pelvis showed multiple pancreatic and peripancreatic fluid collections with the largest walled off necrosis in the pancreatic body measuring 12x6 cm and a large, loculated, left pleural effusion with collapsed left lung. Direct endoscopic necrosectomy with lumen-apposing metal stents (LAMS) was performed. Thoracentesis showed amylase >2960 units/L in the pleural fluid suggesting the presence of a PPF. Four days following placement of a 24 French chest tube, the effusion persisted and the lung remained collapsed. The administration of intrapleural tissue Plasminogen Activator (tPA) (10mg) and dornase (5mg) through a 10 French pigtail drain for three days resulted in lung expansion one day after the last dose. Chest tubes were removed. Necrosectomy was repeated a week later and LAMS were removed. There was no recurrence of pleural effusion or pancreatitis after 6 month follow-up.

Discussion: PPF is a rare complication of pancreatitis diagnosed by high amylase in the pleural fluid [2]. PPF has traditionally been treated by transpapillary drainage of the pancreatic duct with pancreatic duct stents [3] and therapeutic thoracentesis. In this case, thoracentesis with chest tube drainage was not successful in lung reexpansion, presumably due to trapped lung from pleural fibrosis. The next step is usually surgical decortication, but we then took a novel approach of co-administering ribonuclease and protease into the pleural space. Cystgastrostomy had already been performed to prevent any reaccumulation of fluid. This approach allowed resolution of the PPF while avoiding operative procedures.

S1852

When Life Gives You Lemmel's, Call Your Gastroenterologist

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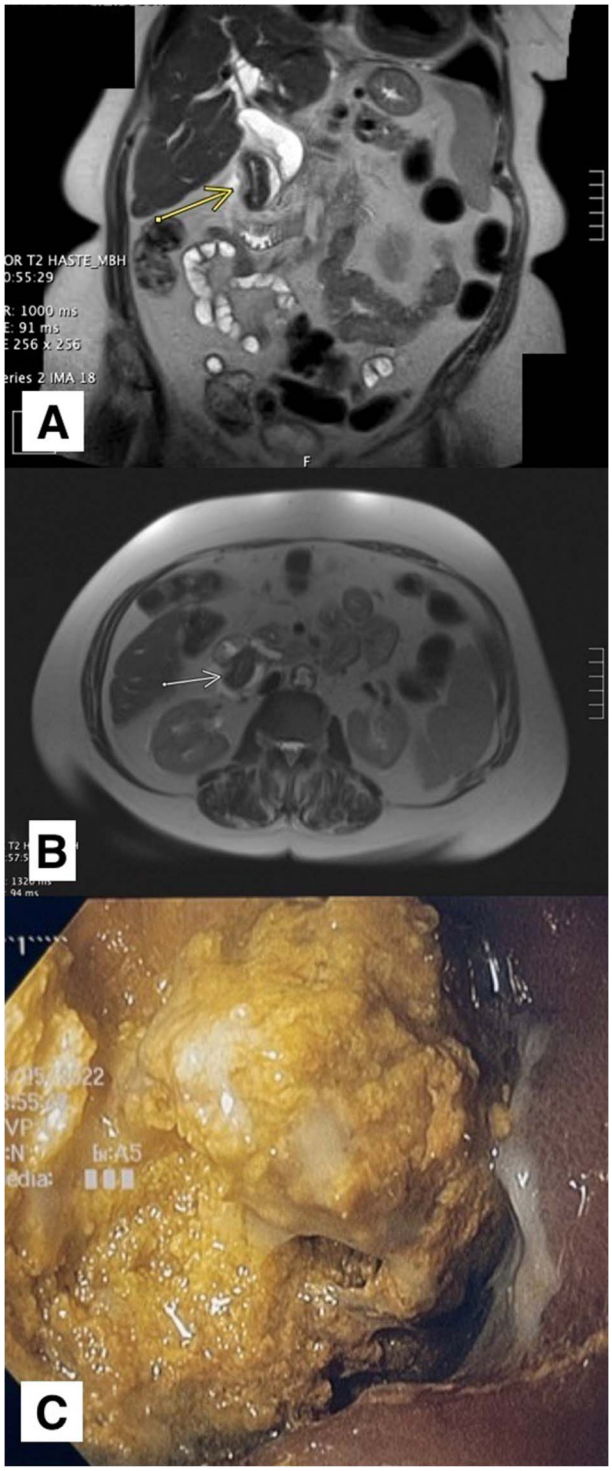
Introduction: Lemmel's syndrome is defined as obstructive jaundice caused by periampullary duodenal diverticulum (PAD), leading to bile duct compression and dilation. PAD leading to the obstruction of the ampulla of Vater is the most common, with an incidence as high as 27%. Most PAD are found incidentally and are suspected to be found in up to 22% of the population, however, some patients can have life-threatening pathology, necessitating quick intervention. This case outlines a patient with this rare pathology and discusses when to choose specific therapeutic options.

Case Description/Methods: A 77-year-old Caucasian female with a history of cholecystectomy 20 years ago presented to the emergency department with a 1-day history of epigastric abdominal pain, mild fever, jaundice, and non-bloody diarrhea. Laboratory evaluation was notable for a lipase of 3326 U/L and transaminitis with ALP 908 IU/L, AST 234 U/L, ALT 153 U/L, and total bilirubin 7.2 mg/dL. CT abdomen with IV contrast revealed moderate-severe pancreatitis and intra/extrahepatic biliary ductal dilation up to 19 mm in diameter with additional dilation of the pancreatic duct. A subsequent MRCP revealed a large periampullary duodenal diverticulum with obstruction of the common bile duct, consistent with Lemmel's syndrome, and possible duodenal diverticulitis (**Figure** A, B). Piperacillin-tazobactam was initiated due to concern for ascending cholangitis and diverticulitis. ERCP was performed the following morning, which revealed a large duodenal diverticulum in the second portion of the duodenum with fecalized material impacted within the diverticulum (**Figure** C). The entire biliary ampulla was obscured by obstructing material and surrounding ulceration was noted. Several attempts to remove the impaction were unsuccessful. A temporary PTC drain was placed with interval improvement in her pain and lab abnormalities. The patient was transferred to another facility for advanced endoscopy, where she had successful removal of obstruction and intra/extrahepatic stenting.

Discussion: Therapeutic options vary, but ERCP is considered the initial treatment of choice because it allows for the placement of a biliary stent and a sphincterotomy. If Lemmel's is due to chronic papillary fibrosis or sphincter of Oddi dysfunction, then an endoscopic sphincterotomy is the preferred therapy. If the disease is severe or complicated, a diverticulectomy is recommended in patients who are at lower risk for adverse complications.



[1852] **Figure 1.** A) Coronal slice from MRI abdomen and pelvis demonstrating a large periampullary duodenal diverticulum with obstruction of the common bile duct. B) Transverse slice from MRI abdomen and pelvis demonstrating a large periampullary duodenal diverticulum with obstruction of the common bile duct. C) Image obtained during ERCP revealing impacted fecalized material within a large duodenal diverticulum in the second portion of the duodenum with surrounding ulceration.

S1853

Type 1 Autoimmune Pancreatitis Unmasked by COVID-19 Vaccine

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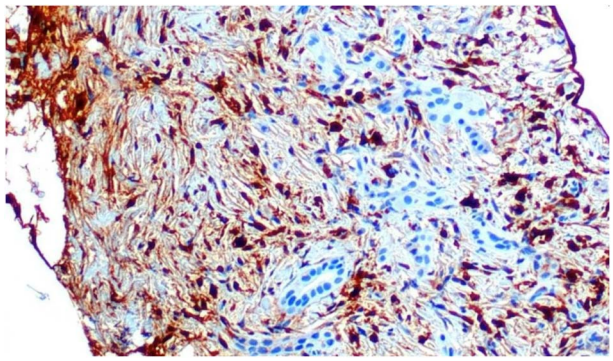
Introduction: Type 1 AIP is a rare form of idiopathic chronic pancreatitis associated with IgG4-related systemic disease. It predominantly affects male adults and has multi-organ extra-pancreatic manifestations. It is much less associated with inflammatory bowel disease compared to type 2 AIP, which is reported to have a least a 15% predilection for ulcerative colitis. To date there are no reports of vaccine-induced type 1 AIP. We present a case of newly diagnosed type 1 AIP in a patient one month after receiving COVID-19 vaccination.
Case Description/Methods: 54-year-old male with history of UC presented with decreased appetite, abdominal pain, fatigue, and 25 pound unintentional weight loss. He had received the second dose of Pfizer/BioNTech COVID-19 mRNA vaccine about 1 month prior. Laboratory tests were significant for hypovolemia, hyperglycemia, and elevated liver enzymes. IgG4 level was 287.0 mg/dL. CT scan showed distended pancreatic parenchyma with peri-pancreatic stranding with segments of biliary ductal dilation. MRCP showed dilated bile ducts to the level of the head of the pancreas where there was an abrupt truncation of the extrahepatic common duct. The pancreas was diffusely enlarged with some irregular beading and narrowing of the main pancreatic duct. On ERCP the lower third of the main bile duct contained a single severe stenosis 20-25 mm in length and a biliary stent was placed. EUS was performed and pancreatic parenchyma was diffusely abnormal with lobularity, generalized hypoechoic gland, hyperechoic foci without shadowing. The pancreatic duct was irregular in contour. Immunohistochemical tests showed IgG and IgG4 positive plasma cells. The patient was started on a three-week prednisone taper. On 9-month follow-up clinical symptoms, laboratory work and repeat imaging showed improvement. (**Figure)**
Discussion: Both vaccine-induced acute hepatitis and pancreatitis have been reported occurring 2-3 days after receiving Pfizer/BioNTech COVID-19 mRNA vaccine. Our case suggests a temporal association between COVID-19 vaccination and AIP although a cause effect relation cannot be definitely established. Our patient was the appropriate age and had ulcerative colitis; thus, the correct demographic for developing AIP. He may have been genetically predisposed and the vaccination triggered the immune system. Like other cases of AIH, no other confounding risk factors were identified. It is important to consider that the COVID-19 vaccine could be the inciting factor for Type 1 AIP.



[1853] **Figure 1.** Medium Power (10x) Immunohistochemical positivity for IgG on plasma cells.

S1854

Two Cases of Post-COVID Cholangiopathy: A Rare Complication of SARS-CoV-2 Infection

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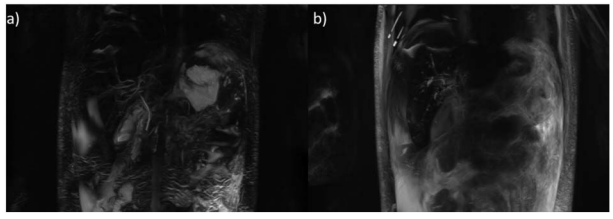
Introduction: Hepatobiliary manifestations are seen in most SARS-CoV-2 infections as mild hepatocellular elevations. A rare complication of severe SARS-CoV-2 infection, termed post-COVID cholangiopathy, has been reported a handful of times. Herein, we present two unique cases of post-COVID cholangiopathy to offer further insight into this unique entity.
Case Description/Methods: Patient 1 is a 69-year-old Hispanic female with a past medical history of prior drug-induced liver injury admitted in August 2020 for acute hypoxic respiratory failure secondary to SARS-CoV-2 pneumonia requiring mechanical ventilation and vasopressor support. 390 days following initial admission, the patient was found to have an elevated alkaline phosphatase (ALP,985 U/L), gamma-glutamyl transferase (GGT, >1600 units), and normal total bilirubin (TBILI, 0.4 mg/dl). Magnetic resonance cholangiopancreatography (MRCP) showed intrahepatic biliary ductal dilatations. The patient continues to follow with hepatology with a most recent MELD of 6. Patient 2 is a 27-year-old Hispanic male with chronic kidney disease admitted in August 2021 for provoked seizures in the setting of active SARS-CoV-2 infection. The patient rapidly deteriorated requiring mechanical ventilation, vasopressors, and continuous renal replacement therapy. On day 49 of admission laboratory values revealed an elevated TBILI (31.6 mg/dl), GGT (1569 U/L), and ALP (5000 U/L) (**Figure**). On day 79, a liver biopsy showed signs of large duct obstruction. MRCP on day 139 showed intrahepatic biliary strictures and dilatation not seen on prior MRCP. The patient was unable to undergo liver transplant evaluation despite a MELD of 36.
Discussion: The most striking features of post-COVID cholangiopathy are the disproportionate elevation in ALP followed by delayed intrahepatic biliary strictures and dilatations. Post-COVID cholangiopathy has exclusively been reported in patients requiring ventilation and vasopressor support. As such, it is postulated that this disease is a result of biliary ischemia induced by COVID hypoxemia and vasopressor-induced splanchnic hypoperfusion. Although rare, we believe the high mortality rate of severe SARS-CoV-2 infection and the delayed histological and anatomical findings needed for diagnosis have contributed to an underestimation of the prevalence of post-COVID cholangiopathy. Further studies are needed to establish the true prevalence as well as definitive diagnostic and treatment criteria for this novel disease.



[1854] **Figure 1.** Patient 2 MRCP day 49 (A) vs day 139 (B).

S1855

Unusual Picture of Pancreatic Abscess in a Patient With a Near-Total Distal Pancreatectomy

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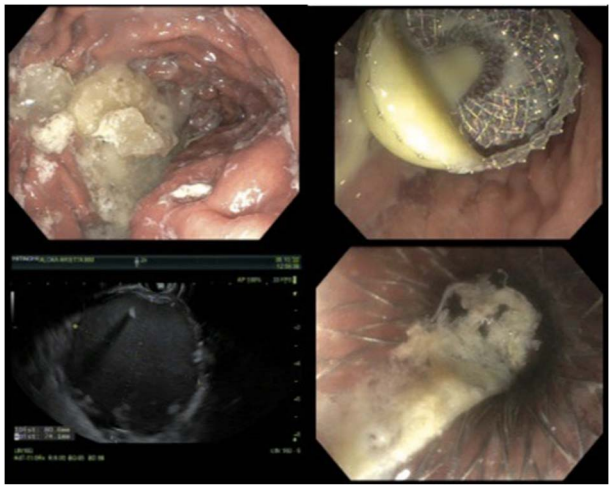
Introduction: Pancreatic abscess is an infection of the pancreatic pseudocyst which usually occurs 4 weeks after the onset of acute pancreatitis, meanwhile other causes include but are not limited to chronic pancreatitis, iatrogenic intra-abdominal procedures, and seeding from distant sites. We hereby report a case of an unusual occurrence of pancreatic abscess in a patient with a near-total distal pancreatectomy.
Case Description/Methods: Patient is a 56-year old male with past medical history of diabetes, pancreatic adenocarcinoma, status post near-total distal pancreatectomy 6 months earlier, pancreatic pseudocyst found 3 months ago and splenectomy presented with persistent progressive worsening left upper abdominal non-radiating pain, associated with nausea, eight episodes of bilious vomiting, abdominal distension and chills. Patient denied fever. Vitals and labs were insignificant. CT scan of the abdomen showed significant increase in the size of the pancreatic cyst by the head of the pancreas with gastric outlet obstruction. Patient was started on Unasyn and the GI team proceeded to EGD/EUS and cystogastrostomy on the same day. A stent was placed [with drainage into the stomach], the cyst was aspirated, the collected purulent

fluid (**Figure)** was sent for culture which came back positive for klebsiella pneumoniae. Patient reported relief after the procedure and was discharged on ciprofloxacin for 2 weeks. After 3 weeks, a follow-up EGD was done for necrosectomy with removal of the stent.

Discussion: Pancreatic abscess is most commonly caused iatrogenically during management of necrotizing pancreatitis, however it can happen in absence of pancreatitis, secondary to biliary tract disease or duodenal disease. A high index of suspicion should be maintained in patients with acute pancreatitis, who don't improve after initial management especially with high RANSON score, and chronic pancreatitis with persistent pain which can lead to systemic inflammatory response syndrome and death. CT is the gold standard in diagnosis with a higher sensitivity than ultrasonography, however a following fine needle aspiration, has a sensitivity of almost 100%, and is crucial to distinguish sterile inflammation from infection. Gram negative bacteria is most commonly found in aspirated fluids, however gram positive can be seen. Invasive surgical cystogastrostomy or cystoduodenostomy, depending on the location of the abscess, should not be delayed and have better outcomes than solo antibiotic therapy or endoscopic intervention.



[1855] **Figure 1.** Stent placement.

S1856

Diffuse-Type Pancreatic Ductal Adenocarcinoma Mimicking Autoimmune Pancreatitis

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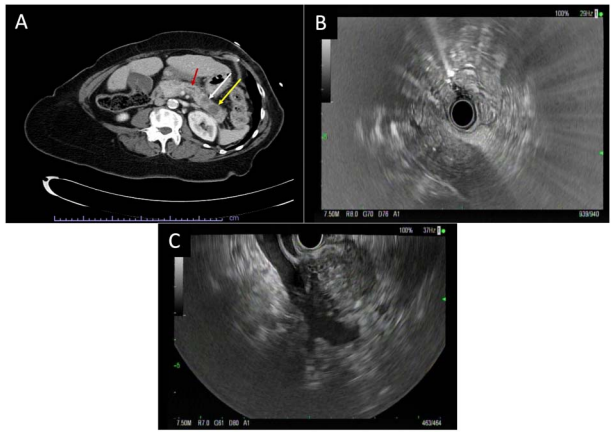
Introduction: Pancreatic ductal adenocarcinoma (PDAC) classically presents as a solitary pancreatic mass on cross-sectional imaging. Diffuse-type PDAC (DTP) is an unusual variant that accounts for 1-5% of PDAC. Due to its rarity, there are no established radiographic or endoscopic definitions of DTP. Previous case reports describe presentations of DTP either as a solitary pancreatic mass or diffuse pancreatic enlargement. We report a unique case of DTP presenting with CT findings of two distinct masses in the pancreatic head and tail, and with EUS findings of diffuse gland enlargement mimicking autoimmune pancreatitis.

Case Description/Methods: A 70-year-old female presented with two weeks of severe epigastric pain and unintentional weight loss. A CT scan showed two distinct pancreatic masses measuring 2.3 x 1.6 cm and 1.6 x 1.0 cm in the head and tail respectively (**Figure** A). EUS demonstrated a "sausage-shaped" diffusely enlarged pancreas with duct dilation of 2.5 mm in the body; no discrete lesions were seen (**Figure** B, C). Workup for autoimmune pancreatitis was negative with normal IGG4 levels. EUS-FNB of the pancreatic head revealed infiltrating adenocarcinoma. A repeat CT scan showed interval enlargement of the tail mass and subsequent EUS-FNB of the pancreas tail returned with moderately differentiated invasive adenocarcinoma. ERCP performed for worsened liver function tests demonstrated a 3 cm stricture of the distal common bile duct, which was managed with a fully-covered metal stent. A multi-disciplinary tumor board deemed the neoplasm borderline-resectable diffuse-type PDAC, and the patient was started on a neoadjuvant chemotherapy regimen of gemcitabine and abraxane in preparation for total pancreatectomy.

Discussion: The differential for multiple pancreatic masses is broad and includes autoimmune pancreatitis, secondary metastases, and pancreatic neuroendocrine neoplasms. DTP is a rare manifestation of PDAC and little is known about its etiology and endoscopic findings. DTP could stem from progression of a focal PDAC or could result from synchronous multifocal tumor development. To our knowledge, this is the first documented case of DTP presenting with multiple pancreatic masses and EUS findings mimicking autoimmune pancreatitis. The case illustrates the importance of sampling several areas of the pancreas when diffuse enlargement is present on EUS and multiple pancreatic masses are seen on cross-sectional imaging.



[1856] **Figure 1.** A. Axial-oblique CT demonstrates a low-density lesion in the pancreatic tail (yellow arrow) measuring 2.3 x 1.6 cm. An adjacent ill-defined low-density lesion (white arrow) measures 1.6 x 1.0 cm. The pancreas is borderline in size and the pancreatic duct is prominent (red arrow). B. EUS demonstrates a diffusely enlarged pancreas with the head, body, and tail displaying a diffusely hypoechoic "sausage-like" appearance. C. EUS demonstrates focal prominence of the pancreatic head with loss of interface with the portal vein-superior mesenteric vein confluence.

S1857

Disappearing Act: A Case of Kikuchi-Fujimoto Disease

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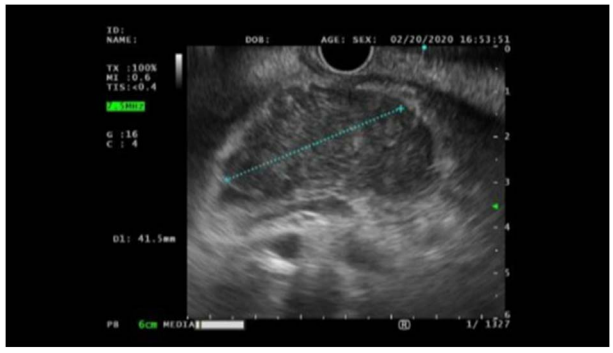
Introduction: Kikuchi-Fujimoto disease (KFD), also known as histiocytic necrotizing lymphadenitis, is a rare, self-limited disorder that most frequently presents with tender lymphadenopathy, rash, fever, and night sweats. We present a case of a patient with diffuse lymphadenopathy and fever diagnosed with KFD.
Case Description/Methods: A 31-year-old female without significant past medical history presented for evaluation for three weeks of dull abdominal pain, fevers, nausea, non-bloody emesis, and 14lb weight loss. Outpatient workup of diffuse lymphadenopathy by her primary care physician with CT and subsequent MRI of abdomen and pelvis had demonstrated a bilobed retroperitoneal mass adjacent to the pancreatic head process concerning for necrotic lymph nodes versus malignancy. Autoimmune serologies were negative, and culture data and viral serologies were negative. She underwent endoscopic ultrasound and fine needle aspiration of the mass (**Figure**). Cytology demonstrated benign-appearing ductal epithelial cells, necrotic debris, and spindle cells with fibro-inflammatory stroma, and was negative for malignancy. Flow cytometry was also negative for malignancy. She continued to remain febrile and was placed on antibiotics following consultation with the infectious disease service. Repeat biopsy was discussed with interventional radiology and general surgery services; however, there lacked an adequate window for percutaneous or laparoscopic approaches. The patient's symptoms were controlled with antipyretics and antiemetics and she was discharged on a course of oral antibiotics. Repeat endoscopic ultrasound-guided biopsy was performed two months later of an enlarged peripancreatic lymph node. Cytology revealed mixed T and B cells with zones of necrosis, but no granulomas. No features of malignancy were seen, and fungal and acid-fast staining were negative. The patient was closely monitored, and her adenopathy and symptoms completely resolved two months later.
Discussion: The diagnosis of KFD is based on histologic examination which demonstrates proliferative and necrotizing lymph nodes with abundant T cells in the lesion. The disease is benign and self-limited, with primary treatment being supportive care, and it has a low recurrence rate. Due to its rarity, the disease poses a diagnostic challenge to clinicians, as it often mimics systemic lupus erythematosus and non-Hodgkin's lymphoma. Our patient presented with fever, weight loss, and a retroperitoneal mass concerning for malignancy, that was found to be KFD.



[1857] **Figure 1.** Endoscopic ultrasound image demonstrating retroperitoneal mass measuring 41mm in maximal cross-sectional diameter.

S1858

Syphilis: The Rare Primary Biliary Cholangitis Mimic

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Introduction: Syphilis is a rare cause of acute hepatitis. This report shows a case of syphilitic hepatitis mimicking Primary Biliary Cholangitis (PBC). This case shows syphilitic hepatitis presenting with an elevated alkaline phosphatase, and a positive Anti-Mitochondrial Antibody M2 subtype (AMA-M2). Treatment of the underlying syphilis with penicillin leads to resolution of the hepatitis and normalization of lab-work.
Case Description/Methods: A 53-year-old male was referred from his PCP for possible PBC. The patient was complaining of night sweats, and chills for a couple months. He was also complaining of diffuse joint pain, eye pain, and rash. He states his PCP started him on steroids which seemed to be resolving symptoms. The patient had A negative CT abdomen, CRP elevated at 81.5, ESR elevated at 61, ferritin elevated at 439, alkaline phosphatase elevated at 207, and had a positive AMA-M2 subtype count at 84.3. We started the patient on a course of Ursodiol 500mg, since he met current criteria for PBC with an elevated alkaline phosphatase >1.5 times upper limit and a positive AMA. Repeat labs 4 weeks later showed alkaline phosphatase of 99. Two months later the patient followed-up stating his rheumatologist did a lumbar puncture and found neurosyphilis. The patient was treated with penicillin and had improvement of symptoms. A literature review was conducted showing syphilis can cause an elevation of alkaline phosphatase and false positive AMA-M2. It was decided to stop Ursodiol for 2 months and repeat labs. Repeat laboratory showed alkaline phosphatase of 110 and negative AMA.

Discussion: A rare presentation of syphilis is with liver manifestations. Syphilis can easily be confused with PBC, with an isolated elevation of alkaline phosphatase, and AMA. This is an important consideration when making the diagnosis of PBC. Syphilis should be considered greater in patients with history of multiple sexual partners, patients with HIV, or other sexually transmitted diseases. The patient in this case had complained of joint pain and rash which were thought to be an unrelated rheumatologic condition given improvement with steroids. Alkaline phosphatase was presumed to be artificially decreased secondary to the steroids before starting Ursodiol. Treatment of the underlying syphilis normalized lab values. This case demonstrates the need to consider syphilis in the differential diagnosis of patients presenting with suspected PBC, especially male patients with no history of other autoimmune diseases.

S1859

A Case of IgG4-Related Sclerosing Cholangitis in a Patient With Chronic Pancreatitis

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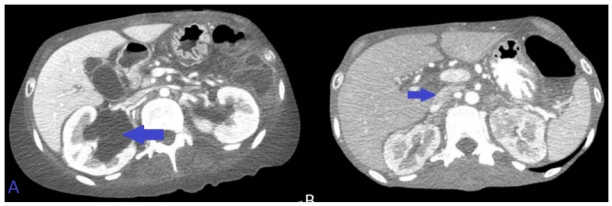
Introduction: Immunoglobulin G4-related disease (IgG4-RD) is an immune-mediated fibroinflammatory condition that is capable of affecting multiple organs. IgG4-related sclerosing cholangitis is a form of sclerosing cholangitis that is clinically different from primary sclerosing cholangitis (PSC) and occurs usually as a part of the IgG4-RD. Additionally, it is also the most common extrapancreatic manifestation of type 1 AIP (IgG4-related) and is present in over 70% of such patients. It rarely occurs in the absence of pancreatitis.
Case Description/Methods: We present a case of 43-year-old female patient with medical history of chronic pancreatitis and secondary bacterial peritonitis status post exploratory laparotomy and gastrostomy tube (G tube) insertion who presented to with the complaint of abdominal pain and leakage around the G tube site. Computed tomography abdomen and pelvis was suggestive of mild intrahepatic ductal dilatation (**Figure**). Laboratory results showed elevated ALP (>1200) and GGT levels. Ultrasound abdomen showed gallstones with sludge and bilateral hydronephrosis. Magnetic resonance cholangiopancreatography (MRCP) was unremarkable. Further workup showed elevated IgG4 levels (143 mg/dL). The autoimmune workup (ASMA, AMA, Anti-cardiolipin, LKMA) were negative. Patient had normal A1AT levels and viral hepatitis panel was negative. Patient underwent a liver biopsy which showed findings suggestive of chronic hepatitis with mild activity, secondary mild hemosiderosis and the presence of lymphoplasmacytic inflammation. IgG4 immunostain highlighted scattered lgG4 positive plasma cells in portal tracts
Discussion: Treatment is usually initiated with prednisone (0.6 mg/kg/day) and then tapered to discontinuation over a two-month period. Rituximab is generally considered in patients who do not respond to up to 40 mg/day of prednisone or cannot be tapered to < 5 mg daily as well as in patients who have strong relative contraindications to glucocorticoid therapy in these doses. Prognosis of this condition is not well established. In some cases, spontaneous improvement can be seen, however disease often recurs without the treatment. Most patients respond to initial therapy with glucocorticoids, but relapses are not uncommon once therapy is discontinued.



[1859] **Figure 1.** A CT Abdomen showing Bilateral Hydronephrosis (Right more than the left) B CT Abdomen showing Pancreatic Calcifications with findings suggestive of Chronic Pancreatitis.

S1860

Diffuse Large B Cell Lymphoma Presenting as Acute Recurrent Pancreatitis

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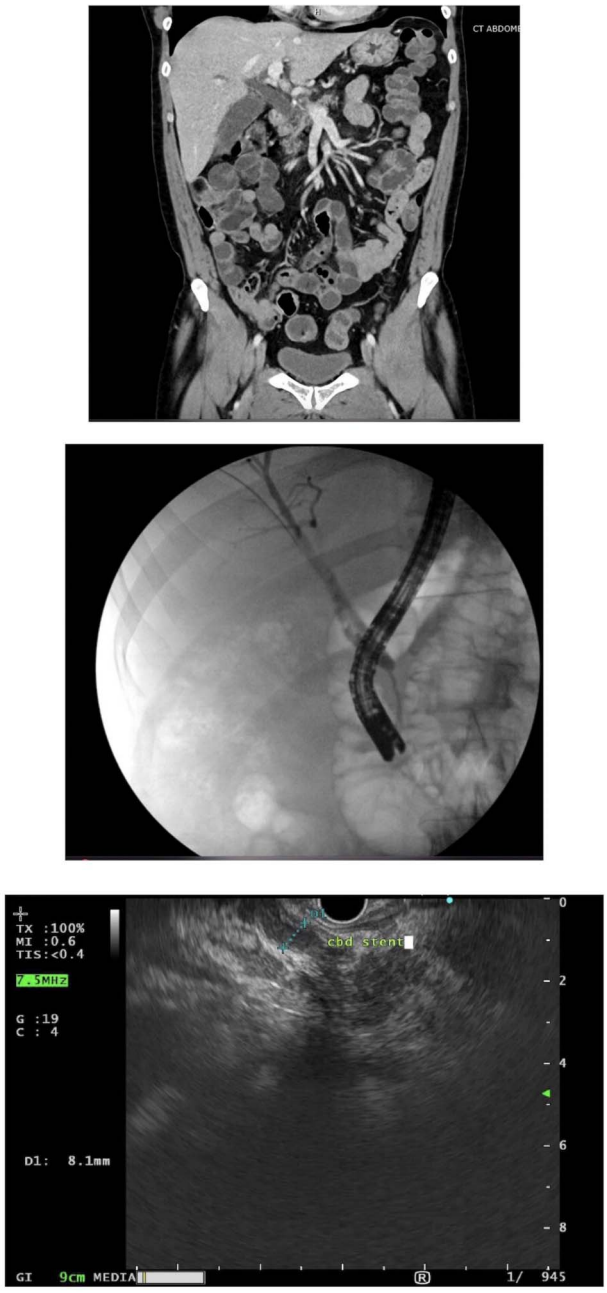
Introduction: Extra-nodal Non-Hodgkin's lymphomas are mostly made of gastrointestinal sites[1]. Primary pancreatic lymphomas account for < 1% of all extra-nodal lymphomas and are extremely rare. Diagnosis is often missed or delayed due to the prevalence of chronic pancreatitis or pancreatic adenocarcinoma as thought to be the main causes of any pancreatic stricture or mass seen when diagnosing a patient. We present a case of pancreatic lymphoma presenting as acute recurrent pancreatitis.
Case Description/Methods: A 51-year-old male with past medical history of rheumatoid arthritis and nicotine use presented with jaundice, abdominal pain, and fatigue and 30-pound weight loss. MRCP showed intra- and extra-hepatic biliary dilation, showing double duct sign. CT suggested acute pancreatitis. ERCP was done showing a single localized biliary malignant appearing stricture in the lower third of the main bile duct with upstream dilation. EUS showed a 19 mm by 19 mm mass in the head of pancreas and 8 mm dilation of the common bile duct. EUS fine needle biopsy showed chronic pancreatitis with dense fibrosis and benign peri-pancreatic lymph nodes. PET CT showed activity in the pancreas and retroperitoneal lymph nodes. Tumor markers (AFP, CEA, CA 19-9) were within normal limits. (**Figure**) Patient had no known risk factors for chronic pancreatitis therefore, the acute presentation and biopsy results in congruent with the clinical history led us to repeat EUS one month later. This showed a 20 mm by 15 mm irregular mass in the head of the pancreas with upstream pancreatic duct dilation. This time, pathology was sent for flow cytometry and came back positive for lymphoid infiltrate with a preponderance of B-cells surrounded by dense fibrosis and was positive for CD 19, 20, 22 and 10. By this point, malignancy other than adenocarcinoma was suspected and IR guided biopsy of retroperitoneal lymph nodes confirmed a diagnosis of diffuse large B cell lymphoma. Patient started chemotherapy with a 70% chance of being cured. ERCP one month later showed the stricture had resolved and the stent was removed.
Discussion: Keeping a broad differential in mind for pancreatic masses is essential to prevent delayed diagnoses. With that in mind, although rare, pancreatic lymphoma should always be in the differential and physicians should consider sending for flow cytometry to avoid missing this diagnosis. When patients present with recurrent pancreatitis and no risk factors, lymphoma should be kept in the differential.



[1860] **Figure 1.** a) CT showing inflammatory reaction of the pancreas with bile duct dilation; b) Bile duct stricture on ERCP; c) EUS image showing common bile duct stent.

S1861

Blame It on the Drug: A Rare Case of Doxycycline-Induced Pancreatitis

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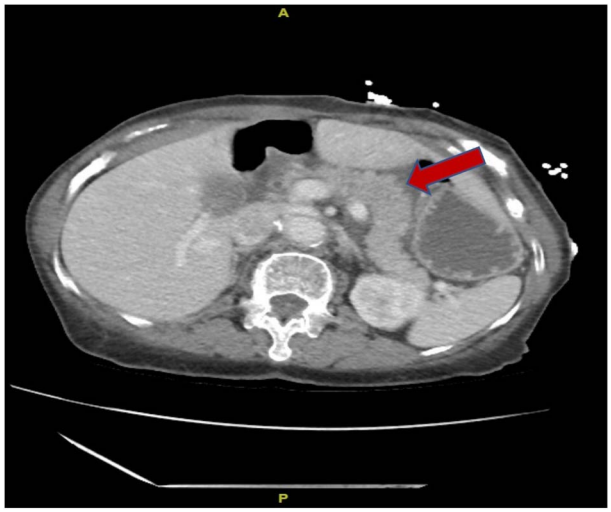
Introduction: Doxycycline is a broad-spectrum bacteriostatic antibiotic that belongs to the tetracycline class. It is a relatively safe medication with reported side effects being gastrointestinal symptoms, bone and teeth discoloration, photosensitivity, and renal toxicity. Acute pancreatitis is an uncommon adverse effect with only a few reported cases in literature. The World Health Organization reports 525 drugs that are linked to acute pancreatitis with a direct causality in 31 drugs. Despite tetracyclines being labeled as a probable causative agent of drug-induced pancreatitis (DIP), doxycycline has been rarely implicated.
Case Description/Methods: A 65-year-old woman with a past medical history of pancreatitis, and community-acquired pneumonia (CAP), presented with nausea and epigastric pain radiating to her back for the past two days. She was recently started on doxycycline three days ago for the treatment of CAP. On further questioning, her symptoms were similar to her previous episode of acute pancreatitis which occurred after starting doxycycline. The patient denied smoking, alcohol, or recent endoscopic retrograde cholangiograms. On arrival, she was febrile and tachycardic. Physical exam was significant for epigastric tenderness. Labs were notable for a leukocytosis of 33, 900, lipase of 3431 IU/L with normal calcium levels, liver enzymes, and lipid panel. A CT of the abdomen and pelvis revealed peripancreatic fluid and fat stranding consistent with acute pancreatitis ((**Figure**). A right upper quadrant ultrasound did not reveal any gallstones or biliary distention. She was started on aggressive intravenous fluids and pain control. Doxycycline was also stopped and her lipase trended down to 573 IU/L on day 2 and 530 IU/L on day 3. She reported symptomatic relief and was discharged home.
Discussion: DIP is a rare phenomenon with an estimated overall incidence rate of less than 2% of all acute pancreatitis cases. It is challenging to diagnose due to the lack of specific clinical symptoms. It is diagnosed by excluding other common causes of pancreatitis, resolution with discontinuation of the drug, and reappearance of symptoms with the same drug as seen in the case above. Our patient scored 8 on

the Naranjo Adverse Drug Reaction meaning that there was a "probable" adverse reaction to doxycycline. Doxycycline is a commonly prescribed antibiotic and it is crucial to report these cases for early identification and cessation in the treatment of acute pancreatitis.



[1861] **Figure 1.** Contrast tomography (CT) of the abdomen and pelvis showing an edematous pancreas with peripancreatic fluid and fat stranding.

S1862

A Rare Pancreatic Tail Schwannoma in an Asymptomatic 58-Year-Old Female

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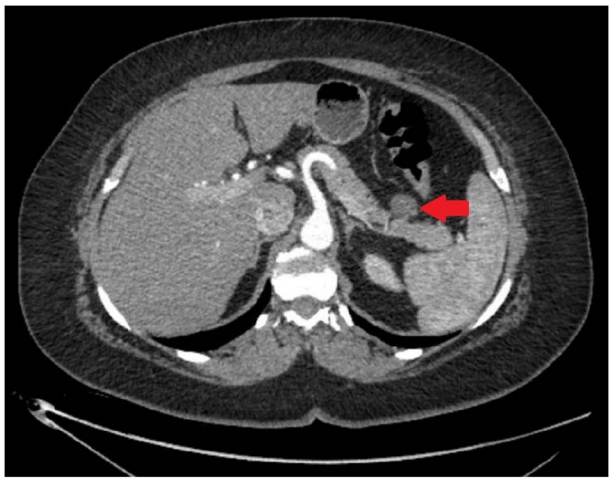
Introduction: Schwannomas are tumors which originate from Schwann cells responsible for fabricating myelin. Although Schwannomas are the most common benign peripheral nerve tumor in adults, there are several variants which are remarkably less common. Pancreatic schwannomas are an exceedingly rare type of nerve sheath tumor which arise from either sympathetic or parasympathetic vagal nerve fibers within the pancreas. In 2017, only 68 cases of pancreatic schwannoma had been reported in the preceding forty years with most occurring in the pancreatic head and body. In this case, we discuss an extraordinarily uncommon presentation of a pancreatic tail schwannoma in an asymptomatic 58-year-old female.
Case Description/Methods: A 58-year-old female with a past medical history of hypertension and hypothyroidism presented with findings of a 2 cm exophytic pancreatic tail lesion seen on prior CT imaging (**Figure**). The patient reportedly had a strong family history of aortic aneurysms and was found to have a right renal lesion on screening CT. She subsequently underwent CT abdomen and pelvis which revealed a lesion concerning for pancreatic tail malignancy. Endoscopic ultrasound (EUS) was performed which showed a 17 x 20 mm isoechoic peripheral pancreatic tail lesion. Fine needle aspiration (FNA) was performed which revealed spindle cells concerning for malignancy. Pathology and immunohistochemistry were inconclusive due to scant FNA aspirate obtained during EUS. Patient was taken to the operating room for exploratory laparotomy with distal pancreatectomy and splenectomy. Pathology of resected pancreatic mass showed typical histology with nuclear palisading and thick-walled vessels. Immunohistochemical staining supported the diagnosis of Schwannoma with diffuse, strong positivity for S-100 and SOX10 as well as negative staining for desmin, smooth muscle actin, CD34, pancytokeratin, CD117 and DOG1.
Discussion: Pancreatic schwannoma most commonly presents with abdominal pain although 30% of cases are found in asymptomatic patients with lesions discovered incidentally on screening CT scans. Although these lesions rarely display malignant transformation, they pose a significant diagnostic dilemma despite advances in radiographic imaging modalities. Endoscopic ultrasound is often limited by insufficient specimen collection and the preoperative diagnosis often becomes quite difficult. Enucleation of tumor is typically a sufficient therapeutic modality however radical resection is often required to establish the definitive diagnosis.



[1862] **Figure 1.** CT scan showing 2 cm exophytic pancreatic tail lesion (red arrow).

S1863

A Case of Adenomyomatosis of the Gallbladder: Evaluating Malignant Potential

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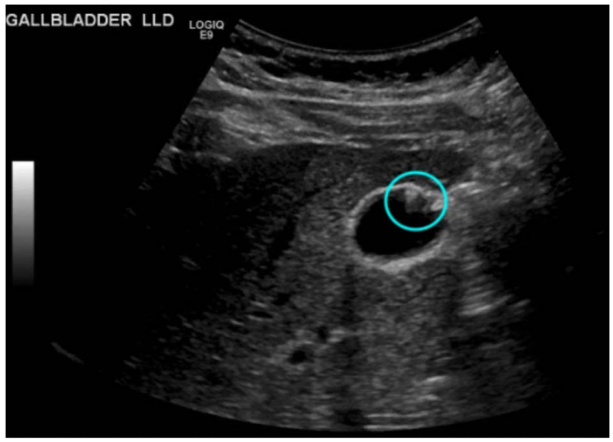
Introduction: Adenomyomatosis of the gallbladder(GA) is a rare pathology involving muscularis growth in the gallbladder(GB). It is associated with an increased risk of gallbladder malignancy. While usually incidentally found post-resection or on abdominal imaging. We present a case of a 42-year-old female who was found to have GA on right upper quadrant ultrasound, in the hopes of encouraging prompt prophylactic removal in such cases due to a risk of progression to malignancy.
Case Description/Methods: A 42-year-old female presented with right upper quadrant pain of 1-year duration. The pain was intermittent, associated with nausea and some degree of constipation, which resolved with fiber supplements. She had no change in the nature of her stool nor any bloating or excessive belching. She also reported no correlation of her pain with food intake, although did report a decreased appetite during this time. Laboratory data was unremarkable. Due to persistent, yet intermittent pain, a right upper quadrant ultrasound was obtained, which revealed focal adenomyomatosis involving the anterior gallbladder wall (**Figure**). The patient was subsequently referred for laparoscopic cholecystectomy with the general surgery team and on follow-up, has been symptom-free since her procedure.
Discussion: GA is a benign incidental finding in as many as 9% of cholecystectomy samples. GA is thought to develop due to persistently elevated intrabiliary pressures, ultimately resulting in a hyperplastic muscularis layer and polyps [4]. Though there is no consensus on the progression of GA to adenocarcinoma of the GB, GA is associated with a less than 5% five-year mortality if left untreated[3]. A study by Ootani et al also described a 6% incidence of GB adenocarcinoma in patients with segmental type GA as opposed to only 3% in patients without GA[3]. Given the association of malignancies with chronic inflammation, GA may render an environment favorable for cancerous growth. With this case study we hope to advocate for the need for more research on the possible association of GB adenocarcinoma in patients with GA, particularly the segmental type and if there are any benefits from prophylactic cholecystectomy in patients with GA found incidentally.



[1863] **Figure 1.** Right Upper Quadrant Ultrasound showing likely adenomyomatosis of the Gallbladder wall.

S1864

A Rare Case of Symptomatic Lymphoepithelial Cyst of the Pancreas

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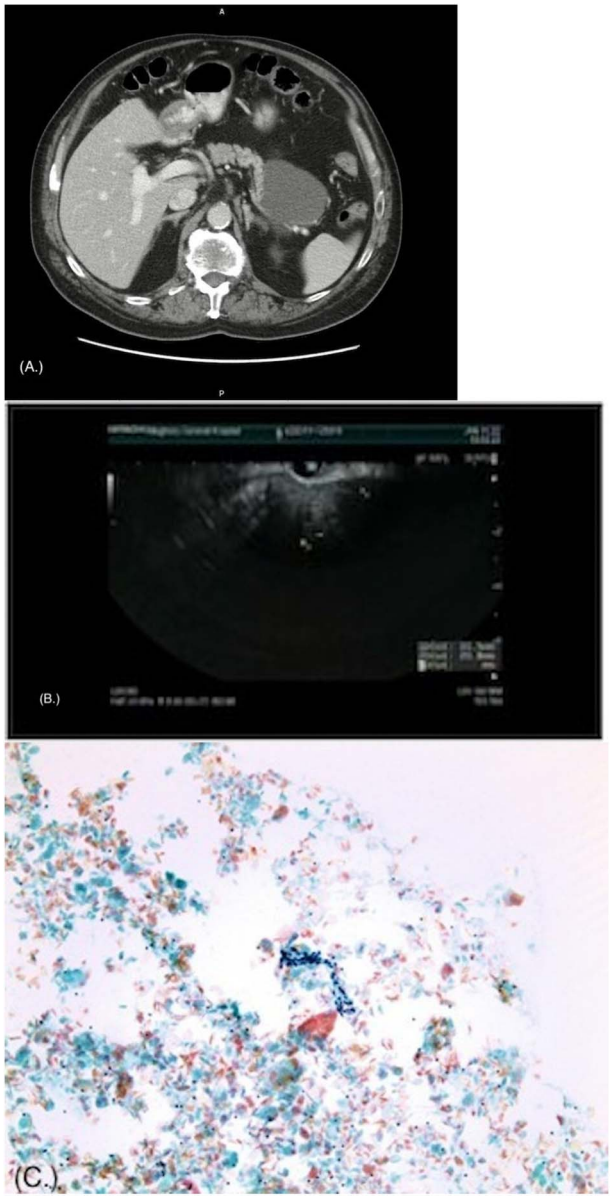
Introduction: Lymphoproliferative cysts (LEC) of the pancreas are rare, nonmalignant cysts. LECs of the pancreas are true pancreatic cysts as defined by their squamous epithelium lining, while being surrounded by mature lymphoid tissue. These cysts can be very difficult to differentiate from other pancreatic cysts or neoplasms on imaging. Patients with LEC cysts will typically present with nausea, abdominal pain, unintentional weight loss, vomiting, and diarrhea as the cyst grows. In 2013, there were 109 documented cases of LEC cysts in the literature (1). Here, we describe a patient presenting with a LEC of the pancreas.
Case Description/Methods: A 60-year-old male with past medical history of hyperlipidemia and type 2 diabetes mellitus presented with a chief complaint of left sided abdominal pain for three months and a 30-pound unintentional weight loss (**Figure A).** Computed tomography revealed a cystic pancreatic tail mass 8.2 cm by 6.0 cm by 4.9 cm. No biliary dilation was noted. Magnetic resonance imaging nine days later revealed a 7.1 cm by 6.7 cm by 5.3 cm heterogenous pancreatic tail mass (**Figure B).** On endoscopic ultrasound, there was an irregular, hypoechoic, heterogenous mass identified in the pancreatic tail which was biopsied using a trans-gastric approach (**Figure C).** Biopsy of the mass showed keratinous debris, fibrotic tissue, and focal mucin. There were noted strips of epithelial squamous cells with lymphocytes. This lesion was diagnosed as a lymphoepithelial cyst of the pancreas.
Discussion: This case was discussed at a multi-disciplinary pancreas conference and surgical resection was advised for symptomatic management. LEC's are extremely rare pancreatic lesions which predominantly occur in men. Imaging alone is not adequate for a diagnosis. In this patient, diagnosis was confirmed with endoscopic ultrasound guided biopsy.



[1864] **Figure 1.** A. CT Scan revealing pancreatic tail mass 8.2 cm by 6.0 cm by 4.9 cm. B. EU: Hypoechoic, heterogenous mass identified in the pancreatic tail, which was biopsied using a trans-gastric approach. C. Pap stain, 10X: Pap stain demonstrating squamous epithelium (center), frequent lymphocytes (small, dark, round cells throughout the field) and dead, sloughed off squamous epithelium (orange and blue background debris). The presence of squamous epithelium and lymphocytes on a fine-needle biopsy of the pancreas supports the diagnosis of pancreatic lymphoepithelial cyst.

S1865

Pancreatic Adenocarcinoma in the Setting of Autoimmune Pancreatitis

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Introduction: Autoimmune pancreatitis (AIP) accounts for 2% of pancreatitis cases and is characterized by chronic pancreas inflammation of autoimmune etiology. Two types of AIP are recognized. Type 1 AIP is an IgG4-related disease which often affects multiple organs. Type 2 AIP is IgG4-negative and affects mainly the pancreas with a third of patients exhibiting additional manifestations. The chronic inflammatory nature of AIP may be associated with higher rates of pancreatic cancer as compared to chronic pancreatitis of other etiologies. This case demonstrates a new diagnosis of pancreatic cancer thought to be secondary to Type 2 AIP.
Case Description/Methods: A 35-year-old female with a past medical history of diverticulitis and chronic pancreatitis of suspected autoimmune etiology presented to the hospital with persistent abdominal pain. An abdominal ultrasound demonstrated a hypoechoic pancreas with focal dilation of the main pancreatic duct (PD). IgG4 was within normal limits. An endoscopic ultrasound (EUS)/endoscopic retrograde cholangiopancreatography (ERCP) with fine needle biopsy (FNB) demonstrated signs of pancreatic inflammation and PD dilation. Biopsy was negative for malignancy. She was discharged on oxycodone and gabapentin and was readmitted without resolution of abdominal pain. On readmission, a CT abdomen/pelvis demonstrated a hypo-enhancing infiltrating lesion in the pancreatic head and uncinate process measuring 2.8 x 2.7 cm with vascular involvement. Ca-19.9 was within normal limits. EUS/ERCP with FNB demonstrated hypoechoic expansion of the pancreas head with PD dilation. Biopsy was positive for pancreatic ductal adenocarcinoma determined to be stage III and unresectable. Treatment with chemotherapy was initiated and currently ongoing at three month follow up.
Discussion: AIP and pancreatic cancer have similar presentations and must be clinically distinguished for appropriate treatment. There are many case reports of misdiagnosed pancreatic cancer later determined to be AIP by negative biopsy, however, there are few reported cases of AIP associated pancreatic cancer with positive biopsy. AIP is a chronic state of pancreas inflammation which may represent a pre-malignant process. This case demonstrates a patient with Type 2 AIP and negative biopsy who subsequently developed biopsy proven pancreatic cancer. Patients with Type 2 AIP may require close follow up for early detection of AIP induced pancreatic cancer. Further investigation is needed to determine the pre-malignant potential of AIP.

S1866

Panniculitis and Pancreatitis: Inflammation and Necrotic Mechanisms in a Patient With Alcohol Use Disorder and Chronic Pancreatitis

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Introduction: Panniculitis is an inflammatory process localized to subcutaneous tissue, with etiologies including infection, malignancy, external insults, enzymatic destructive processes, and inflammatory disorders. Here, we present a rare case of a patient with chronic pancreatitis with features concerning for myeloma given lytic lesions, who was found to have incidental panniculitis.
Case Description/Methods: A 53-year-old male with a history of T2DM, HTN, DLD, hypothyroidism, MDD, alcohol use disorder, and chronic pancreatitis presented with lower abdominal pain and was found to have acute descending colon diverticulitis with an abscess, with an incidental finding of lytic pelvic lesions. Within the past 2 years, the patient had 2 prior episodes of pancreatitis and endorsed daily alcohol consumption. CT of the abdomen and pelvis demonstrated acute diverticulitis of the descending colon with associated abscess, liver parenchymal disease, chronic pancreatitis, and an incidental finding of lytic pelvic lesions and a posterior L iliac bone/L acetabular cyst sclerotic lesion. Workup for multiple myeloma was negative. A skeletal survey demonstrated lucent lesions within bilateral pelvic bones versus overlying bowel gas and a cortically based sclerotic lesion along the distal femur, with chronic rib fracture of the right 11th posterior rib. Given his findings, the patient's osteolytic pelvic lesions were considered to be panniculitis secondary to pancreatitis. The patient was treated with antibiotics and intravenous fluids for diverticular abscess and pancreatitis and he was discharged home due to clinical improvement and resolution of his diverticular abscess on repeat imaging.
Discussion: The incidence of panniculitis with subcutaneous fat necrosis with underlying pancreatic diseases is 2-3%, which may encompass associated periarthritis with bone necrosis and panniculitis (PPP syndrome). PPP syndrome results due to systemic activity of pancreatic enzymes, leading to disturbances within the microcirculatory system, and fat necrosis of medullary bone marrow; yet, exact pathophysiology remains unknown. Our patient provided an interesting clinical picture given his alcohol use disorder, and lytic lesions which initially lead the team towards a malignancy workup such as myeloma; however, given his negative studies, his panniculitis was considered to be derived from his chronic pancreatitis. Additional literature is warranted regarding the relationship between panniculitis in patients with chronic pancreatitis.

S1867

Crohn's Disease of Duodenum and Ampulla as Cause of Biliary Sepsis

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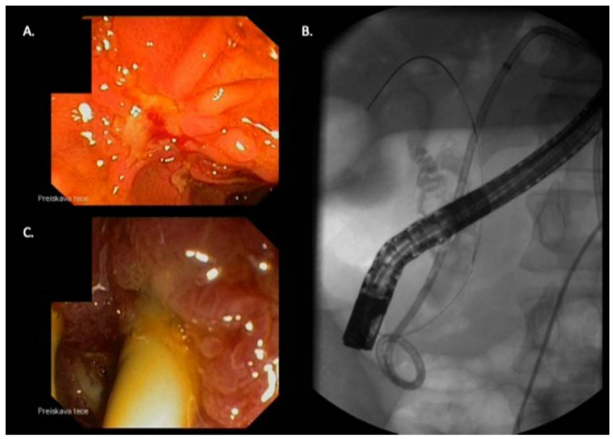
Introduction: We present a case of a 25-year-old man with large and small bowel Crohn's disease (CD) that was admitted for further evaluation of acute right upper quadrant pain, elevated liver function tests (AST 157 IU/L, ALT 184 IU/L, ALP 117 IU/L, Total bilirubin 1.58 mg/dL, GamaGT 267 IU/L, WBC 18.3 x 1000/uL) and fever. Blood cultures were positive for Klebsiella spp.
Case Description/Methods: He was recently switched from infliximab to adalimumab due to secondary treatment failure. Magnetic resonance cholangiopancreatography showed choledocholithiasis with dilatated common bile duct (CBD) at 20 mm. Endoscopic retrograde cholangiopancreatography (ERCP) failed due to extensive Crohn's disease in the duodenum that caused ulceration with subsequent fibrosis of the ampulla (**Figure** A). An external-internal drainage was inserted with fluoroscopic guided percutaneous transhepatic biliary access (PTD). Follow-up ERCP was performed with cannulation of the CBD beside the PTD catheter. Multiple stones were removed, and the PTD catheter was exchanged for biliary stent (**Figure** B and C). After discharge, he was scheduled for a repeat ERCP with stent exchange.
Discussion: CD of the duodenum is rare; it occurs in less than 5% of adult patients with CD. Manifestations of duodenal CD are nonspecific. While CBD obstruction as a complication of CD is reported in the literature, this is the first case that describes a fibrosis as a consequence of CD as cause of biliary sepsis. This case highlights a rare but important complication of duodenal CD. While pediatric guidelines recommend upper gastrointestinal endoscopy in all children with CD, the adult guidelines recommend it only be done in patients with upper gastrointestinal symptoms. Our case highlights the need for a case by case approach.



[1867] **Figure 1.** A) Crohn's disease ulceration with subsequent fibrosis of the ampulla. B) Fluoroscopic image of percutaneously placed biliary stents. C) Endoscopic image of percutaneously placed biliary stents.

S1868

Mirizzi Syndrome: A Real Pain in the "neck"

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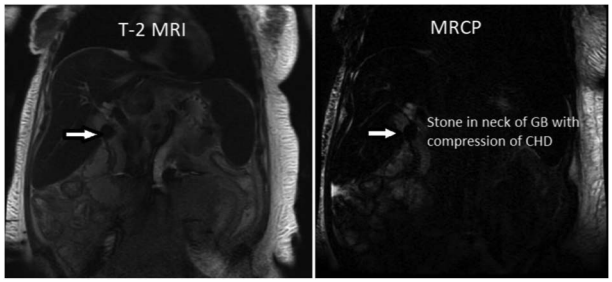
Introduction: Mirizzi syndrome is a rare biliary phenomenon whereby the hepatic duct becomes obstructed via extrinsic compression from an impacted stone in the neck of the gallbladder. Mirizzi syndrome has been associated with an increased risk of gallbladder cancer and occurs in 0.05 to 4 percent of patients undergoing surgery for cholelithiasis. In this case, we describe a patient who presented with cholangitic symptoms and MRCP findings concerning for Mirizzi Syndrome type 1a.
Case Description/Methods: A 49-year-old female with a past medical history of colorectal carcinoma status post chemotherapy and radiation, HTN, HLD and obesity presented to the ED with complaints of fever, jaundice and right upper quadrant pain. Of note, patient was admitted two months prior for similar presentation and was found to have an obstructing common bile duct stone treated with ERCP, balloon extraction and sphincterotomy. She was discharged in stable condition but was soon readmitted due to intractable nausea, vomiting, and was found to have a mechanical large bowel obstruction secondary to known colonic mass. She underwent diverting loop colostomy, was subsequently cleared by surgery, and discharged in stable condition. The patient now presents with fever, jaundice, and RUQ pain. Physical exam revealed profoundly icteric conjunctiva, diffuse jaundice of face and trunk, diminished bowel sounds, and right upper quadrant abdominal tenderness. Labs were remarkable for TBili 6.7, DBili 5.3, ALP 1,553, and leukocytosis. RUQ ultrasound showed a contracted gallbladder with CBD measuring 14.5mm. MRCP showed a large stone impacted in the gallbladder neck with external compression of the common hepatic duct (**Figure**). Patient was seen by Surgery and Gastroenterology who advised against surgery or endoscopic intervention given the patient's poor oncologic prognosis. The decision was made to undergo percutaneous cholecystostomy for biliary decompression. Given the severity of symptoms and overall poor prognosis, the patient was ultimately readmitted to inpatient hospice and passed away soon thereafter.

Discussion: Given the degree of obstruction and likelihood of cholangitis progression, Mirizzi syndrome must be treated as a surgical emergency. The mainstay of treatment involves surgical resection which permits direct removal of causal factors: both the gallbladder and impacted stone. Mirizzi syndrome carries a high mortality rate and should always be suspected in patients presenting with fever, jaundice, and right upper quadrant pain.



[1868] **Figure 1.** MRI and MRCP showing stone in Gallbladder neck with compression of Common Hepatic Duct.

S1869

A Case of Biliary Intraepithelial Neoplasia of the Extrahepatic Bile Duct

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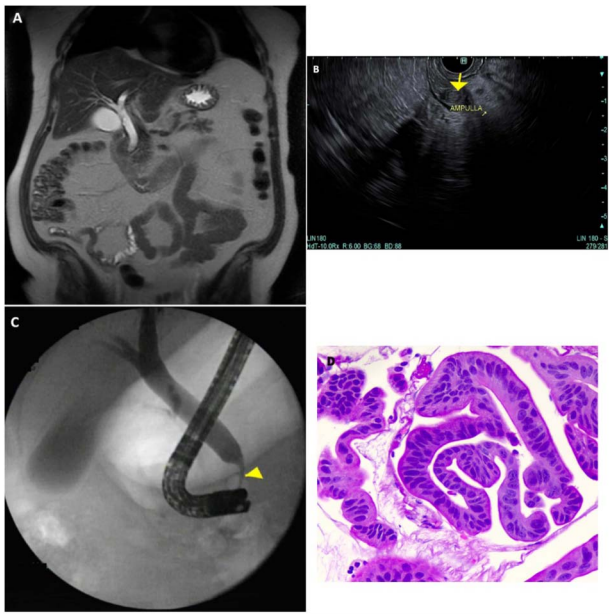
Introduction: Biliary intraepithelial neoplasia (BilIN) is a relatively new diagnosis that refers to flat or micropapillary precursor lesions of the bile duct which can develop into adenocarcinomas. Herein, we present a case of an extrahepatic biliary intraepithelial neoplasia that was found during endoscopic retrograde cholangiopancreatography (ERCP).
Case Description/Methods: 54-year-old male with a history of hypertension and tobacco abuse presented with sharp, right upper quadrant abdominal pain with intermittent nausea and vomiting of one month duration. On admission, he was hemodynamically stable, with normal labs including liver function tests. MRI/MRCP revealed intra- and extra-hepatic duct dilation up to 12 mm with no intraductal filling defect or stricture and a normal gallbladder. (**Figure** A).The patient underwent an EUS which revealed common bile duct dilation up to 12 mm with a hypoechoic lesion in the distal bile duct, just proximal to the ampulla (**Figure B**). Given the EUS findings, an ERCP was performed. Cholangiogram revealed diffuse dilation of the biliary tree with no obvious stricture or filling defect (**Figure C).** The biliary tree was swept, and nothing was found. Due to the suspicious lesion seen on the EUS we performed distal common bile duct brushing and biopsies. Histopathology revealed biliary intraepithelial neoplasia, grade 2 (**Figure D).** His pain was controlled and was discharged with plans for pancreaticoduodenectomy (Whipple's) procedure.
Discussion: BilIN refers to flat or micropapillary precursor lesions of the bile duct which can develop into adenocarcinomas. They are commonly found in liver samples of chronic biliary and liver diseases such as hepatolithiasis, primary sclerosing cholangitis, choledochal cyst, chronic hepatitis C, and alcoholic cirrhosis. However, they are rarely found in clinical practice since they are not easily accessible, nor do they usually cause bile duct obstruction. The natural course of BillN is not well understood and in 2017, diagnostic criteria were created for lesions found in the intrahepatic duct based on their cellular and structural atypia. Lesions in the extrahepatic duct do occur, however, very rarely. This case is unusual in that the BilIN caused symptoms suggestive of biliary obstruction with a dilated bile duct and because the BillN was found in the extrahepatic duct. The case highlights the importance of considering BilIN in biliary obstruction and perform biopsies to identify the lesions prior to development of malignancy.



[1869] **Figure 1.** (A) MRI T2 flare reveals intra- and extra-hepatic duct dilation up to 12 mm with no intraductal filling defect or stricture. (B) EUS reveals hypoechoic lesion in the distal bile duct, just proximal to the ampulla. (C) ERCP cholangiogram reveals diffuse dilation of the biliary tree with no obvious stricture or filling defect. (D) Hematoxylin and eosin stain reveals dysplastic biliary epithelium (flat architecture; enlarged, hyperchromatic nuclei with nucleoli and some pseudostratification, rare mitotic figures) consistent with BilIN grade 2.

S1870

A Case of Biliary Cast Syndrome After Liver Transplantation

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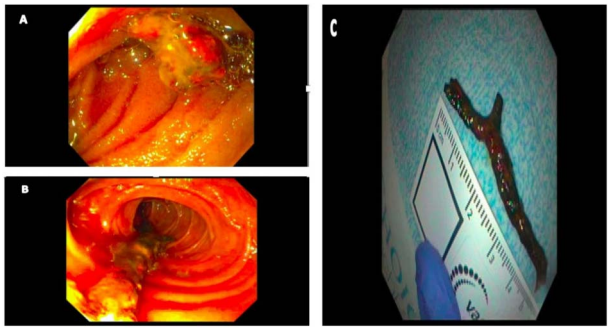
Introduction: Biliary Cast Syndrome (BCS) is characterized by biliary cast and debris resulting in biliary obstruction. It is a complication of orthotopic liver transplant (OLT) population occurring in 4-18% of recipients. Patients can present with cholangitis and graft damage or loss. Symptoms include fever, jaundice, and cholestatic liver enzyme elevation. Twenty-two percent of patients with BCS require repeat OLT. We present a case of BCS after OLT.
Case Description/Methods: A 67-year-old man with history of hypertension, alcoholic cirrhosis status post orthotic liver transplant three months ago with no acute complaints was admitted to the hospital for evaluation of elevated liver associated enzymes (LAEs). His liver transplant biliary anastomosis was a Roux-en-Y choledochojejunostomy due to poor quality of the recipient hepatic duct. The patient was compliant with his transplant clinic visits and immunosuppression medications. Initial vital signs and physical exam were unremarkable. The alkaline phosphatase was elevated to 790, alanine aminotransferase was elevated to 86, aspartate aminotransferase was elevated to 199, total bilirubin was 0.9, and gamma-glutamyl transferase was elevated to 1478. Liver biopsy showed mild portal edema with ductal proliferation and associated neutrophilic inflammation without evidence of rejection or infection. He underwent endoscopic retrograde cholangiopancreatography (ERCP). A balloon enteroscope was used to reach the choledochojejunal anastomosis where a biliary stone cast was found protruding into the lumen of the jejunum. A biopsy forceps was used to remove the 5.5 cm biliary cast. A cholangiogram was performed with contrast into the right and left intrahepatic ducts without any remaining stones. The patient LAEs improved post-procedurally and he was treated with Ciprofloxacin for five days. (**Figure)**
Discussion: Biliary complications after OLT occur in 10-25% of cases after liver transplantation. While biliary strictures, bile leaks and bile duct stones account for a majority of these complications, it is important to keep BCS on the differential diagnosis as well. The mechanism of cell injury can be a result of ischemia, acute cellular rejection, chronic rejection, infection or bile stasis resulting in desquamated epithelial cells forming hard casts with bile components. Treatment can include ERCP with sphincterotomy with removal of casts, lithotripsy with stent placement, and percutaneous drainage.



[1870] **Figure 1.** A.) Biliary cast on endoscopy B.) Biliary cast removal on endoscopy C.) Biliary cast specimen.

S1871

A Case of Ketamine-Induced Cholangiopathy: A Novel Diagnosis Associated With Chronic Recreational Ketamine Use

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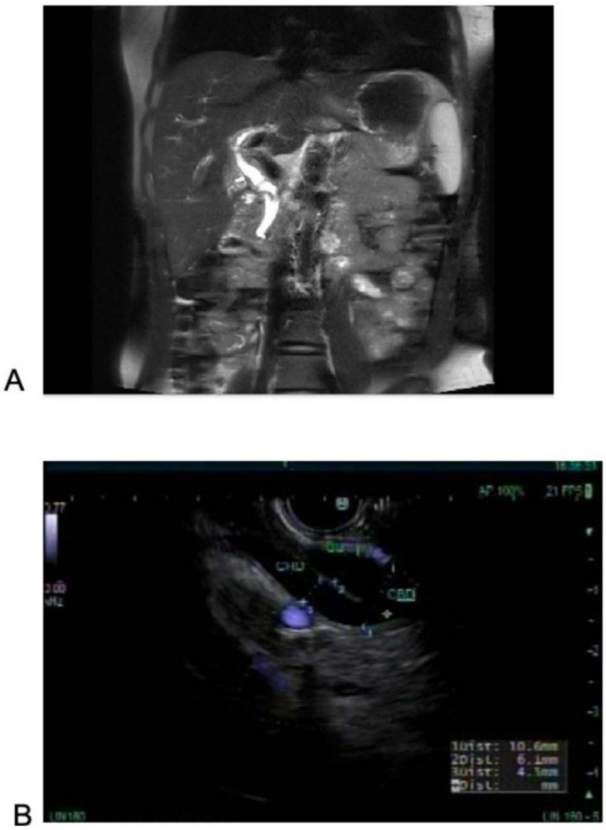
Introduction: Ketamine, an analgesic commonly used for procedural sedation, has become a popular agent for recreational use among young adults. The diagnosis of ketamine-induced cholangiopathy is uncommon. We report a case of a young adult with clinical and radiological features consistent with cholangiopathy induced by chronic ketamine use.
Case Description/Methods: A 19-year-old female with past medical history of peptic ulcer disease presented to emergency department (ED) with one year history of severe epigastric abdominal pain. On further questioning, patient reported chronic recreational ketamine use. Physical exam revealed mild epigastric and RUQ tenderness. Liver function test demonstrated elevated alkaline phosphatase of 174 U/L, AST of 237 U/L and ALT of 224 U/L. All other hematology and chemistry results were within normal limits. RUQ ultrasound showed minimally dilated common bile duct (CBD). An MRCP was obtained, showing mild-to-moderate dilatation of intra-/extra-hepatic bile ducts and dilated cystic duct (CD) without obvious intraductal calculi (**Figure A)**. Endoscopic ultrasonography (EUS) was performed showing dilatation of CBD, minimal dilatation of common hepatic duct, and minimal dilatation of CD, once again without obvious calculi (**Figure B)**. These imaging findings are compatible with ketamine-induced cholangiopathy. After being admitted for 6 days, the patient demonstrated significant improvement in her abdominal pain, with gradually improving liver test values. She was discharged with the recommendation to abstain from ketamine use and outpatient gastroenterology follow-up.
Discussion: Chronic exposure to ketamine has been known to cause urologic injury, referred to as ketamine-induced ulcerative cystitis. In comparison to the urologic complications of chronic ketamine use, its effects on the biliary system have been less commonly reported. Existing reports have mostly come from regions of Asia where recreational ketamine use is more prevalent. The nonspecific gastrointestinal symptoms that are typically reported by patients with ketamine-induced cholangiopathy may make the diagnosis more challenging to recognize. Due to increasing recreational ketamine use among young adults in recent years, cases of ketamine-induced cholangiopathy are likely to rise. Therefore, a better understanding of the clinical and radiological features of this condition are needed to allow for prompt recognition of ketamine-induced cholangiopathy.



[1871] **Figure 1.** A. MRCP image showing mild-to-moderate dilatation of the intrahepatic and extrahepatic bile ducts and dilatation of the cystic duct. There are no visible stones or evidence of other etiology for the dilatation. B. EUS image displaying CBD dilatation measuring up to 10.6 mm in the head region, with smooth tapering to the ampulla where it measured 2.7 mm.

S1872

A Case of IgG4 Sclerosing Cholangiopathy After Cholecystectomy

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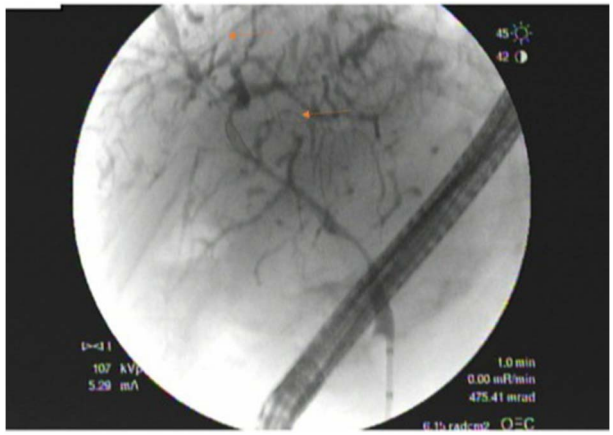
Introduction: Immunoglobulin (Ig) G4 (IgG4)-sclerosing cholangiopathy (IgG4-SC) is a hepato-biliary inflammatory condition that can affect any level of the biliary tree. The diagnosis remains challenging as it shares similar findings in other conditions.
Case Description/Methods: An 83-year-old Caucasian male presented to clinic for anorexia, abdominal pain, and elevated liver function tests (LFTs). The patient had a history of cholelithiasis and was status-post cholecystectomy eight months prior, but otherwise had no history of elevated liver enzymes. Upon presentation to clinic, his AST was 205, ALT 143, and alkaline phosphatase 253, with normal bilirubin. Autoimmune work-up as well as hepatic serologies for Hepatitis A, B, and C were negative. Serum alpha fetoprotein, alpha-1-antitrypsin, ceruloplasmin and ferritin levels were in the normal range. Magnetic resonance cholangiopancreatography (MRCP) revealed intrahepatic bile duct dilation, but normal common bile duct. Endoscopic retrograde cholangiopancreatography (ERCP) revealed intrahepatic dilation to 5 mm with a sclerosing cholangitis (**Figure**). Initially, there was a concern for an ischemic cholangiopathy secondary to the prior cholecystectomy, however his IgG4 returned at 1140 mg/dl, specific for IgG4-SC. The patient was started on high-dose prednisone. Repeat MRCP showed improvement of biliary ductal dilation and chronic IgG mediated pancreatic inflammation. Patient had resolution of anorexia, abdominal pain, and his liver function tests and IgG4 downtrended.
Discussion: IgG4-SC is a chronic inflammatory disease of the biliary system that typically occurs in association with other manifestations of IgG4-related disease. Most patients clinically present in their 7th or 8th decade of life and the disease has a male predominance. The presenting symptoms include jaundice, pruritis, abdominal pain, and weight loss. IgG4-SC is diagnosed via a combination of imaging, laboratory, serological, and histopathological findings. When IgG4 levels are greater than 250 mg/dl, the specificity of IgG4-SC is 90%. IgG4-SC can be differentiated from primary sclerosing cholangitis with elevated serum levels and steroid responsiveness. Rarely is a tissue diagnosis necessary. The mainstay of treatment is corticosteroids with patient response shown by normalization of LFT's, reduction in serum IgG4 levels and improvement in imaging. Relapse can occur and is typically treated with immunomodulators.



[1872] **Figure 1.** Intrahepatic duct dilation with strictures proximally (red arrows).

S1873

A Case of Gastrointestinal Lymphoma Presented as Obstructive Jaundice

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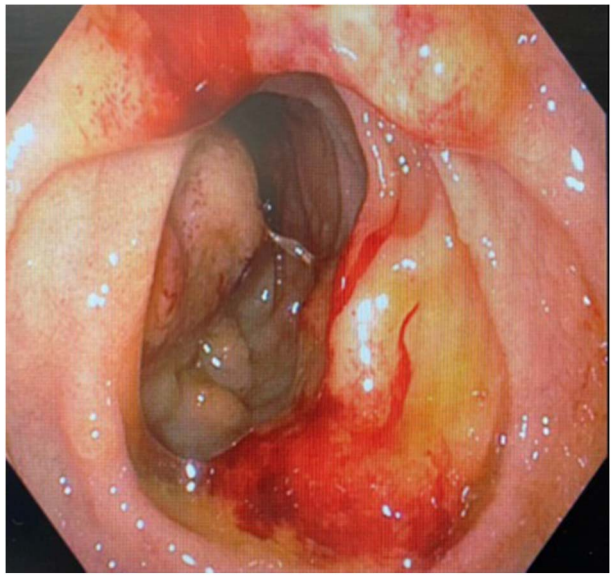
Introduction: The incidence of extra-nodal NHL is 25%. The Gastrointestinal (GI) tract is the most common extra-nodal site accounting for 30-40 % of extra-nodal NHL. GI lymphomas can be in the stomach (65%), small intestine (20%–30%), colon (10%–20%), and esophagus (< 1%). Here we are presenting a case of intestinal lymphoma presented with obstructive Jaundice.
Case Description/Methods: A 79-year-old woman with a past medical history of HIV on treatment presented with abdominal pain, loss of appetite, and weight loss of 15 kg over a period of one year. Labs showed total bilirubin of 5.6 mg/dl and direct bilirubin of 3.12 mg/dl. Imaging showed dilated CBD measuring 1.8 cm, neoplastic mass in the cecum. MRI showed dilated common bile duct and abnormal ampullary area (**Figure).** The patient underwent upper Endoscopy which showed a mass at the major papilla. Biopsy of the mass is positive for CD45 with Ki-67 80% consistent with Diffuse large B cell Lymphoma (DLBCL). Colonoscopy revealed multiple malignant polypoid lesions in the cecum, ascending colon, hepatic flexure, transverse colon, and rectum with biopsy of all lesions consistent with DLBCL. Immunohistochemical analysis confirms the presence of a malignant B-cell lymphoma diffusely positive for CD20, CD79a, BCL-2, BCL6, MUM-1, and c-MYC with a high Ki and positive for cyclin D1. Bone marrow biopsy showed no evidence of lymphoma. Biliary decompression with a metallic stent was done under CT guidance. Dose adjusted mini-CHOP treatment was administered under close monitoring. Eventually, the patient deceased due to non-response and hemodynamic instability.
Discussion: GI lymphomas can present with many manifestations but, obstructive jaundice is rare. We noticed the fungating mass around the ampulla our suspicion was a periampullary carcinoma. Colonoscopy revealed multiple masses and various locations, which lead us to consider a differential of carcinoid tumors. However, to our surprise biopsy revealed diffuse large B cell lymphoma as it accounts for only 1-2% of all GI malignancies. Obstructive jaundice occurs in 1-2 % of the cases of non-Hodgkin's lymphoma, which is mostly due to peri-hilar and peri-portal lymphadenopathy. This case is a rare finding due to intraluminal lymphoma causing distal biliary obstruction. Our patient is a rare finding as lymphoma has occurred even after risk mitigation for HIV. Standard treatment is a combination of chemo-immunotherapy (RCHOP)with excellent survival rates. Without therapy, the survival times are limited to a few months.



[1873] **Figure 1.** Fungating mass around the Ampulla.

S1874

A Bloody Obstruction: Obstructive Jaundice From a Gastroduodenal Artery Pseudoaneurysm

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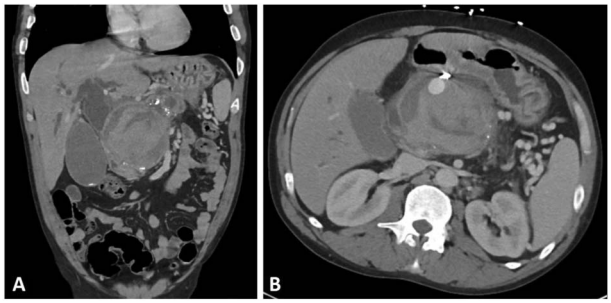
Introduction: Visceral artery aneurysms and pseudoaneurysm are uncommon vascular phenomena, with hepatic and splenic artery origins representing the most common sites, that have a high mortality if ruptured. Typically, these are asymptomatic incidental findings discovered on imaging. Gastroduodenal artery (GDA) pseudoaneurysms may present prior to rupture as obstructive jaundice.
Case Description/Methods: A 40-year-old male with a history of chronic alcohol pancreatitis, hypertension, remote history of abdominal procedure who presented to an outside hospital with two-week history of right upper quadrant abdominal pain with radiation to the back, jaundice, nausea, poor oral intake, and weakness. Upper endoscopy showed erosive esophagitis with no varices and a small hiatal hernia. On examination he was noted to be encephalopathic with pallor and scleral icterus. Total bilirubin was 20.2mg/dL, direct 14.2, hemoglobin 6.8, WBC 18k, CT abdomen/pelvis revealed a 9.5 x 8.9 cm GDA pseudoaneurysm with contained hemorrhage causing gastric outlet obstruction and marked intra- and extrahepatic ductal dilation, at which point he was transferred to our tertiary level care center (**Figure**). He underwent GDA coil embolization and placement of percutaneous transhepatic cholangiography (PTC) drain with control of hemorrhage, progressive resolution of hyperbilirubinemia and encephalopathy.
Discussion: GDA pseudoaneurysms are extremely rare forms of visceral aneurysms, often found incidentally or following rupture which worsens prognosis. Occasionally, GDA pseudoaneurysms will present as obstructive jaundice as in the case of this patient. Management of these rare vascular phenomena require a multidisciplinary team, with endovascular techniques as the preferred first line approach, to achieve favorable clinical outcomes.



[1874] **Figure 1.** CT Abdomen and Pelvis with IV contrast demonstrating gastroduodenal (GDA) pseudoaneurysm with active hemorrhage causing mass effect and intra- and extrahepatic ductal dilation.

S1875

A Case for Therapeutic Plasma Exchange in a Patient With Severe Hypertriglyceridemia-Induced Pancreatitis

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Introduction: Hypertriglyceridemia-induced acute pancreatitis (HTG-AP) is the third most common cause of acute pancreatitis after alcohol and gallstones. It is associated with severe outcomes, including pancreatic necrosis, multiorgan failure, and death. We present an extraordinary case where a patient with HTG-AP rapidly improved after initiating therapeutic plasma exchange (TPE).
Case Description/Methods: A 38-year-old female with a history of cholecystectomy presented to a hospital for acute abdominal pain, nausea, and vomiting. On presentation, she was afebrile and hemodynamically stable. Her exam was significant for generalized abdominal tenderness. Her labs were notable for a WBC of 23,000 cells per cubic millimeter and lipase of 5,131 units per liter. The CT of her abdomen showed inflammatory stranding involving the proximal pancreas consistent with acute pancreatitis. She was admitted to a telemetry floor for IV fluids and pain management. The following day she developed acute hypoxic respiratory failure and acute renal failure. She was found to have a triglyceride (TG) level of 2,751 mg per deciliter (mg/dL). She was transferred to the intensive care unit for an insulin drip and heparin drip for demand ischemia. The patient developed distributive shock, requiring pressors and broad-spectrum antibiotics, as well as intubation and mechanical ventilation for acute respiratory distress syndrome. Upon transfer to our hospital, the patient's TG level was 2,171 mg/dL. The patient received continuous renal replacement therapy and TPE. After two sessions of TPE, her TG levels decreased to 714 mg/dL. On day one post TPE, the patient was no longer in shock, and vasopressors were discontinued. On day five, the patient was successfully extubated, and antibiotics were discontinued with a negative infectious workup. After day nine, she had recovered renal function and was discharged on atorvastatin 40 mg qHS, fenofibrate 48 mg QD, and omega-3 acid ethyl esters capsules 2 g BID.
Discussion: TPE, on average, reduces TG levels by more than seventy percent after one session. TPE functions by removing plasma in blood and substituting it with colloids or crystalloid solutions. In this case, the goal is to remove very-low-density lipoprotein and chylomicrons, which are rich in triglycerides. Unfortunately, due to accessibility and cost, there is no clear consensus on when to initiate TPE for patients with HTG-AP. We postulate that patients with HTG-AP and multiorgan system failure should be considered for TPE.

S1876

A Case of Annular Pancreas With Malignant IPMN

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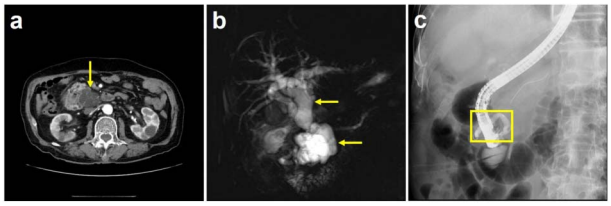
Introduction: Annular pancreas is an uncommon congenital malformation. Pancreatic tissue encircles the second portion of duodenum in which the unusual rotation of the pancreatic ventral bud occurs during embryonic development. Moreover, the combination of an annular pancreas with a pancreatic malignancy is extremely rare. Here, we report a rare case of annular pancreas with malignant intraductal papillary mucinous neoplasm (IPMN).
Case Description/Methods: A 82-year-old man was admitted to our hospital with one-month history of jaundice. The patient had a history of hypertension and diabetes. He had icteric sclera and right upper quadrant area pain with Murphy's sign. In laboratory test, total bilirubin was 24.84 mg/dl, direct bilirubin 19.44 mg/dl, aspartate aminotransferase 45 IU/L, alanine aminotransferase 76 IU/L, alkaline phosphatase 878 U/L, gamma-glutamic transpeptidase 387 U/L, amylase 105 U/L and lipase 109.33 IU/L. Abdominal CT and MRI with MRCP showed a huge IPMN (6 cm sized multi-locular mass) with high risk stigmata in pancreas head, causing extra hepatic biliary obstruction and coincidentally suspicious annular pancreas (**Figure**). We examined the duodenum using upper endoscopy first. It showed multiple irregular active ulcers from bulb to just above the second portion of duodenum with luminal narrowing. Then, ERCP (endoscopic retrograde cholangiopancreatography) revealed that widened ampullary orifice was discharging much mucin and confirmed that encircling ventral annular pancreatic duct (diameter 7mm) was connected to doral pancreatic duct using guidewire and contrast dyes. It showed long-segmental stricture (length 42mm) at the distal bile duct and a biliary plastic stent was inserted after minimal endoscopic sphincterotomy and biopsies at the bile duct stricture. The biopsy result was negative for malignancy, and the brush cytology was performed in the next ERCP session. The brush cytology demonstrated a cluster of malignant glandular cells with nuclear overlapping, nuclear membrane irregularity and loss of polarity. Because of both old age and poor general condition, the patient did not want any treatment. We just treated him conservatively. Although the total bilirubin was improved to 6.1 mg/dl about 20 days later, he died 2 months after with pregression of cancer, renal failure and sepsis.
Discussion: In conclusion, the possibility of coexisting pancreatobiliary disease or malignancy should be considered in adult patients with an annular pancreas although it is extremly rare.



[1876] **Figure 1.** (a) Abdominal CT The arrow indicates a huge IPMN (6 cm sized multi-locular mass) at pancreatic head. (b) Abdominal MRI with MRCP (Magnetic resonance cholangiopancreatography) The upper arrow indicates a diffuse common bile duct dilatation. And the lower arrow indicates a suspicious annular pancreas. (c) ERCP (Endoscopic retrograde cholangiopancreatography) Encircling ventral annular pancreatic duct (diameter 7mm) was connected to dorsal pancreatic duct using guidewire and contrast dyes.

S1877

A Misleading Presentation of Gallstone Pancreatitis Caused by Leptospirosis

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Introduction: Leptospirosis is a common zoonotic infection with presentation ranging from mild influenza-like symptoms to deadly multi-organ failure. Pancreatic involvement, including isolated hyperlipasemia, is rarely seen in Leptospirosis and is mainly caused by vascular damage. We describe an unusual case of Leptospirosis mimicking gallstone pancreatitis presenting with jaundice, hyperlipasemia, and typical pancreatitis-like clinical features.
Case Description/Methods: A 45 year old male with a past medical history of hypertension presented with a one week evolution of nausea, non-bloody/non-bilious emesis, fever, epigastric pain, pale diarrhea, and anorexia. Vital signs were remarkable for tachycardia. Physical examination revealed bilateral scleral icterus, dry oral mucosa and severe epigastric tenderness. Laboratory workup disclosed stable platelets and hemoglobin, WBC of 24,000/mm^3, creatinine of 2.77 mg/dL, with associated azotemia. Liver function enzymes revealed elevated total/direct bilirubin (12.62/11.84 mg/dL), GGT (372 U/L), alkaline phosphatase (143 U/L), and mildly elevated AST with negative viral hepatitis markers. Lipase levels showed increasing trend from 375 to 467 (n= 0-160 U/L). Patient was subsequently admitted under the diagnosis of gallstone pancreatitis for which aggressive IV hydration, IV antibiotics and symptomatic treatment was initiated with little improvement. Abdominal ultrasound revealed hepatomegaly with no biliary ductal dilatation, visible gallstones, or pancreatic abnormalities. Magnetic resonance cholangiopancreatography showed no evidence of cholelithiasis, choledocholithiasis, cholecystitis or pancreatitis. Abdominopelvic computed tomography disclosed no evidence of intra-abdominal pathologies. On day #4 patient presented with bilateral conjunctival suffusion which raised concern for Leptospirosis for which serology was ordered. Patient was initiated on oral Doxycycline therapy with subsequent improvement of symptoms and laboratory parameters. IgM Leptospira antibody test was positive. On day #6 patient left against medical advice.
Discussion: Rare cases have described pancreatitis caused by Leptospirosis without imaging confirmed structural changes of the pancreas which creates a diagnostic challenge for physicians. This case demarcates the importance of Leptospirosis awareness and high clinical suspicion warranted in order to allow for early diagnosis, prevent treatment delay and avoid unnecessary imaging or interventions.

S1878

A Case of Type II Autoimmune Pancreatitis in a Patient Treated With Pembrolizumab Adjuvant Therapy

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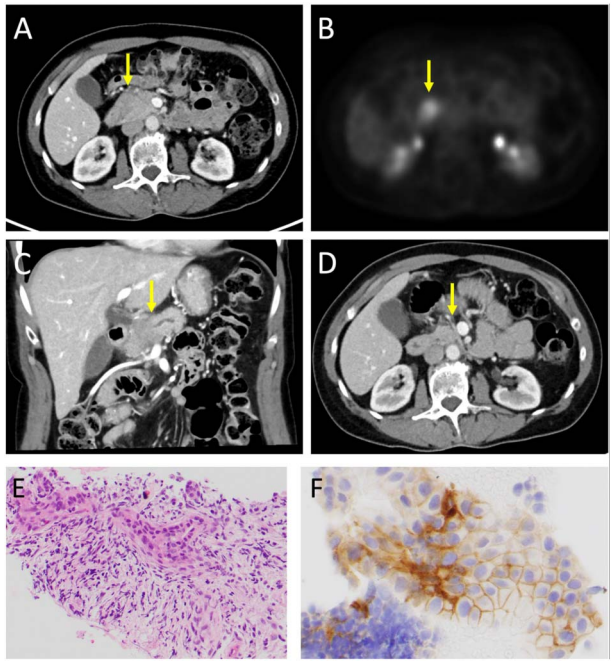
Introduction: Immune checkpoint inhibitor (ICI)-therapy causes GI toxicity but its association with mass-forming Type II AIP has not been described. While pancreatic manifestations of ICI-toxicity are known, it does not include mass-forming AIP. This case demonstrates development of a worrisome pancreatic mass during ICI-therapy which was not amenable to steroid treatment due to potential adverse oncological outcomes. We describe the pathological and imaging features of this entity so that others may consider this diagnosis in the future.
Case Description/Methods: A 59-year-old female was diagnosed with stage IIIC clear cell ovarian cancer. Treatment included optimal cytoreduction to no evidence of disease, adjuvant carboplatin/paclitaxel and bevacizumab, followed by two year bevacizumab/pembrolizumab maintenance to prevent recurrence. After 15 months on pembrolizumab, patient had developed mild epigastric pain and diarrhea, and restaging positron emission tomography scan showed a FDG-avid lesion in the pancreatic head (**Figure**). MRI showed a 3.5cm hypoenhancing mass in this region without pancreatic duct dilation. Labs included lipase of 1014, normal liver function tests, tumor markers and IgG subclass levels. Endoscopic ultrasound (EUS) with fine needle aspiration demonstrated atypical cells and fine needle biopsy (FNB) showed features consistent with chronic pancreatitis with few plasma cells. A diagnosis of Type II AIP versus ICI-pancreatitis was made and patient was monitored with close surveillance imaging. Follow up CT showed increased size of the pancreatic mass to 4 cm and new biliary duct dilation. Repeat EUS-FNB showed pancreatic parenchyma with cellular fibrosis, prominent periductal lymphohistiocytic and eosinophilic inflammation, and ductal cells positive for PD-L1 consistent with Type II AIP (**Figure**). Steroids were not given due to concern for adverse oncological outcome. Repeat imaging following completion of her ICI-therapy showed complete resolution of the mass and her symptoms improved.
Discussion: To our knowledge, we describe the first case of mass-forming Type II AIP in the setting of ICI-therapy. While imaging and histology were consistent with Type II AIP, it is possible that this represents a new entity of ICI-mass forming pancreatitis. Here we demonstrate that she was able to complete her ICI therapy while managed with short-interval imaging studies and pancreatic function monitoring. Additional studies are needed to determine the role of ICI-therapy in mass-forming AIP.



[1878] **Figure 1.** Contrast enhanced axial CT image shows hypo enhancing pancreatic head mass (yellow arrow) (A) which shows intense FDG avidity on the corresponding PET image (B). Ten weeks later contrast enhanced coronal CT image shows interval enlargement of the hypoenhancing pancreatic head mass (yellow arrow) with pancreatic duct obstruction with dilatation of the main pancreatic duct in the pancreatic bud (C). After 4 months of close monitoring axial contrast enhanced CT images show resolution of the mass with mild atrophy of the pancreatic head (yellow arrow) (D). The core biopsy shows the characteristic granulocytic epithelial lesion or "GEL" consisting of neutrophils infiltrating and injuring a duct (or acinus) surrounded by an inflamed fibrotic stroma (E, hematoxylin and eosin). Immunohistochemical staining with PDL1 shows membranous staining of the ductal epithelium, supporting the diagnosis of autoimmune pancreatitis (F).

S1879

A Diagnostically Challenging Case of Primary Pancreatic Lymphoma

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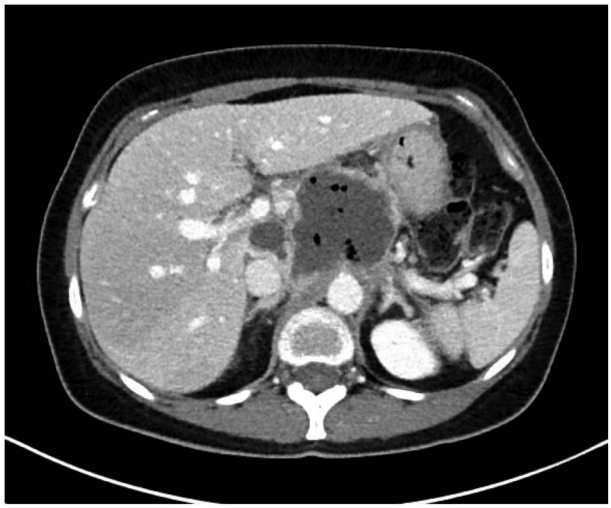
Introduction: Primary Pancreatic Lymphoma (PPL) is the exceedingly rare instance of extranodal Non-Hodgkin's Lymphoma developing mainly in the pancreas. We report a diagnostically challenging case of a patient presenting with a rapidly growing pancreatic mass, found to have PPL.
Case Description/Methods: A 48-year-old female with past history of tobacco use presented with several months of cramping abdominal pain following COVID-19 infection. She denied weight loss, fevers, or night sweats. Her physical exam, CBC, CMP, lipase, LDH, and CA 19-9 were unremarkable. An abdominal ultrasound revealed a 2.8 x 1.9 x 3 cm cystic mass of the pancreatic head, most congruent with a pseudocyst. Worsening abdominal pain prompted repeat ultrasound one month later, which showed a doubling in size. Endoscopic ultrasound (EUS) with fine needle aspiration of the cystic mass and surrounding lymph nodes yielded cystic contents and reactive lymphadenopathy. Two months later, her abdominal pain worsened and repeat imaging showed further doubling in size with encasement of the celiac plexus. A second FNA performed via EUS redemonstrated cystic contents. An ultrasound-guided core needle biopsy of the mass revealed necrotic CD30+ diffuse large B cell lymphoma (DLBCL). PET scan was suggestive of stage IV PPL (**Figure**). Imaging also identified an inguinal lymph node that returned as CD10+ BCL6+ high grade follicular lymphoma, which was thought to be a distinct lesion. She was started on R-CHOP. Her clinical course was complicated by the formation and subsequent rupture of a splenic artery pseudoaneurysm, gastrointestinal bleeding, anuric kidney injury, and intestinal ischemia. She ultimately transitioned to comfort care.
Discussion: Primary pancreatic lymphoma comprises 0.6% of extranodal lymphomas and 0.2% of primary pancreatic tumors. The clinical presentation is often vague and includes abdominal pain, B symptoms, jaundice, or bowel obstruction. The diagnostic criteria according to the WHO requires that the (1) majority of tumor burden be localized to the pancreas and (2) existing nearby and distant lymph node involvement should be secondary to pancreatic presentation. A biopsy is required to diagnose PPL, which is histologically most often DLBCL. Our case highlights the challenges associated with diagnosing PPL despite two EUS with FNA. Although rare, one should proceed with a high index of suspicion for PPL in any patient presenting with a rapidly enlarging pancreatic mass.



[1879] **Figure 1.** CT image of necrotic pancreatic mass.

S1880

A Delayed Diagnosis of a Functional Gastrinoma Mistaken for Atypical Crohn's Disease

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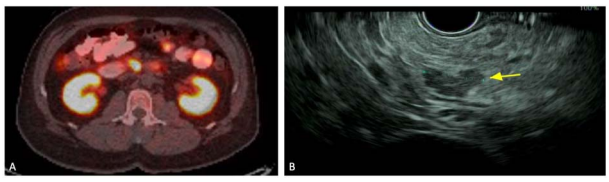
Introduction: Abdominal pain, vomiting, and diarrhea are cardinal symptoms of many GI diseases. A careful diagnostic workup is often necessary to find the etiology of the symptoms. Here, we present a patient with these symptoms whose diagnostic course was protracted but ultimately successful.
Case Description/Methods: A healthy 61yo man was admitted for 5 days of intractable diarrhea, vomiting and severe epigastric pain. Routine bloodwork was normal and CT showed nonspecific thickening in the stomach and small bowel without lesions. He was treated for viral gastroenteritis. However, his symptoms recurred. An MRI was normal without lesions. Colonoscopy was normal and push enteroscopy revealed esophagitis and multiple duodenal and jejunal ulcers. Biopsies showed focal acute jejunitis and erosion, suspicious for atypical Crohn's disease. CRP was 92 and fecal calprotectin was 409, further supporting this diagnosis. A gastrin level was 707, felt to be due to PPI use. He was planned for Crohn's treatment but did not start due to frequent ER visits for his symptoms that were treated conservatively. After 4 months, the patient was re-evaluated. He had an elevated chromogranin A of 2569. A DOTATATE PET was obtained given previous negative imaging and revealed increased uptake in the proximal pancreas and in the third portion of the duodenum (**Figure A**). EUS showed a 14mm pancreatic uncinate lesion (**Figure B**). FNB showed a low-grade neuroendocrine tumor (NET). He was diagnosed with a functional gastrinoma. He underwent a Whipple without complications and on follow up, his symptoms had completely resolved.
Discussion: This is a case of a delayed diagnosis of a functional gastrinoma causing persistent abdominal pain, vomiting and diarrhea. NETs are rare, found in 3/1,000,000 people, and only 10% are functional tumors. The diagnosis of a NET requires specific blood tests, visible lesions, and positive biopsies. Here, the patient's initial evaluation was indeterminant for NET and biopsies suggested Crohn's, delaying his correct diagnosis and treatment. CT and MRI have poor sensitivities (59% and 20%) for detection of gastrinomas without hepatic metastases. However, DOTATATE PETs have a detection rate of 92% in patients with equivocal CTs. EUS has a high sensitivity of 77% for pancreatic tumors. Thus, for patients with common and protracted symptoms, the differential diagnosis should remain broad, and further investigation with higher yield tests may be necessary to arrive at a timely diagnosis and treatment.



[1880] **Figure 1.** FIGURE A: DOTATATE PET Scan demonstrating increased FDG uptake at the 3rd portion of the duodenum. FIGURE B: Endoscopic ultrasound demonstrating a 14mm pancreatic uncinate lesion.

S1881

A Case of Non-Insulinoma Pancreatogenous Hypoglycemia Syndrome in a Cerebral Palsy Patient Where Diagnosis and Treatment Was Delayed Due to Continuous PEG-Tube Feeding

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Introduction: Non-insulinoma pancreatogenous hypoglycemia syndrome (NIPHS), a rare cause of post-prandial hypoglycemia, presents with neuroglycopenia and negative localization studies for insulinoma. We present a case of NIPHS in a female whose cerebral palsy and continuous tube feeds created difficulty in establishing her diagnosis.
Case Description/Methods: A 22-year-old female with cerebral palsy (baseline nonverbal status and quadriplegia) presented to the hospital with 5 days of lethargy. She had no fevers or seizures at home and had been on continuous enteral nutrition via PEG tube for two years. Vitals were normal. Physical exam showed decreased responsiveness and absent spontaneous eye opening on top of baseline posturing and muscle spasticity. Infectious workup was negative. Labs found a decreased blood glucose of 72mg/dl. As patient's mentation improved overnight, hospital tube feeds were restarted at a reduced rate. Patient's morning blood glucose was 66mg/dl so tube feeding rate was increased. Blood glucose further declined to 42mg/dl a few hours later. Continuous dextrose infusions, high carbohydrate tube feeds, and continuous glucagon drip were administered with minimal benefit. Insulin, proinsulin, and c-peptide were found to be within normal range. Insulin antibody test was also negative. Abdominal ultrasound and CT found no lesions in the pancreas or other acute pathology. Patient was deemed to have NIPHS. Acarbose was initiated with resolution of hypoglycemia and improvement in mentation and patient was discharged home with no further complications.
Discussion: The postprandial hypoglycemia differential in non-nondiabetic patients includes insulinoma, post bariatric surgery hypoglycemia, dumping syndrome, insulin autoimmunity, postprandial syndrome, and NIPHS. Our patient's cerebral palsy and nonverbal status masked the neuroglycopenic symptoms typical in NIPHS. Continuous home tube feeds via PEG initially prevented us from determining the postprandial timing of her lethargy. These clues could have pointed us in the direction of NIPHS sooner which would have allowed for more prompt utilization of acarbose, earlier discharge and avoidance of the use of dextrose infusions, glucagon, and high carbohydrate tube feeds. The high carbohydrate diet likely worsened our patient's condition as reports indicate free carbohydrate reduction improves the clinical status of NIPHS patients. Fortunately, we were able to administer acarbose to resolve our patient's condition without incident.

S1882

A Not So Typical Case of Abdominal Pain: A Case of Atypical Hemolytic Uremic Syndrome (aHUS) Induced by Recurrent Pancreatitis

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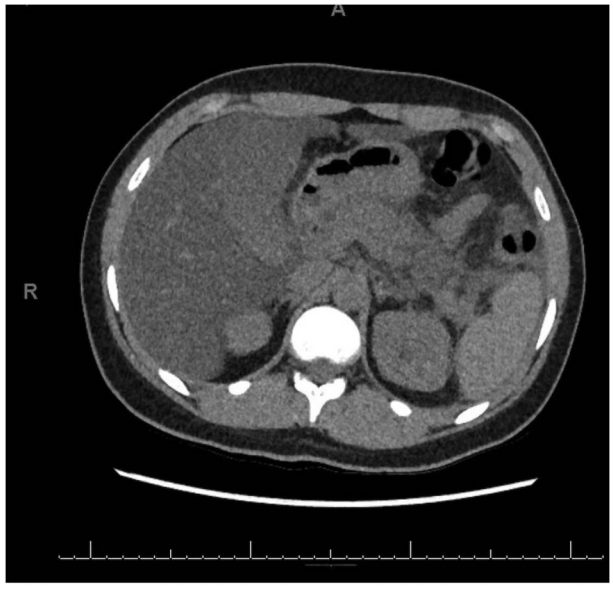
Introduction: Hemolytic uremic syndrome (HUS) is a thrombotic microangiopathy presenting with hemolytic anemia, thrombocytopenia, and kidney injury. Although typically caused by *E. coli* infection, atypical HUS (aHUS) occurs as a result of systemic disease. Causes include systemic lupus erythematous, complement disorders, and pre-eclampsia. Rare cases exist of aHUS induced by recurrent pancreatitis. In these situations, the cause of pancreatitis was alcohol-induced. We present a case of aHUS induced by non-alcoholic pancreatitis.
Case Description/Methods: The patient is a 20-year-old male with a history of autism spectrum disorder, seizures, and recurrent pancreatitis who presented due to 2 days of vomiting, fevers, and abdominal pain. He had acute pancreatitis 2 years prior, which was thought to be medication-induced. Lab work was remarkable for a creatinine of 4.18, a leukocytosis of 18.3, a hemoglobin of 14.8, a platelet count of 34, AST 111, ALT 51, total bilirubin of 4.5, and a lipase of 8771. CT imaging showed enlargement of the pancreas with diffuse pancreatic free fluid suggesting pancreatitis (**Figure**). He was admitted to the ICU. Acutely overnight the patient's hemoglobin decreased from 14.8 to 10.9, platelets to 9, and creatinine to 5.32. No signs of active bleed. Haptoglobin was decreased at 8. Indirect bilirubin and LDH were elevated. His hemoglobin and platelets continued to decrease, while his renal function worsened. DIC panel and Coombs test negative. His CH50 was decreased at 50. ADAMTS13 was 96%. The patient received two sessions of plasma exchange therapy. The leading differential diagnosis at the time was atypical HUS secondary to recurrent pancreatitis. On day 4, the patient received eculizumab and was started on a course of solumedrol. On day 5, the patient underwent hemodialysis. The patient's anemia and thrombocytopenia improved. His renal function returned to his baseline. He was subsequently transferred to the floors for continued management.
Discussion: Atypical HUS is not frequently associated as a sequela of pancreatitis. Most commonly associated are pseudocyst formation and biliary tree dilation. However, in this case, pancreatitis resulted in system-wide complement dysfunction. Through multi-specialty evaluation, the patient was able to receive appropriate care. Eculizumab has been shown to inhibit the thrombotic microangiopathy. As his pancreatitis improved with supportive care, the patient's consequences of this initial insult required much closer management.



[1882] **Figure 1.** Inflammation of the pancreas seen on CT imaging.

S1883

A Case of Pancreaticobiliary Actinomycosis Mimicking a Mass

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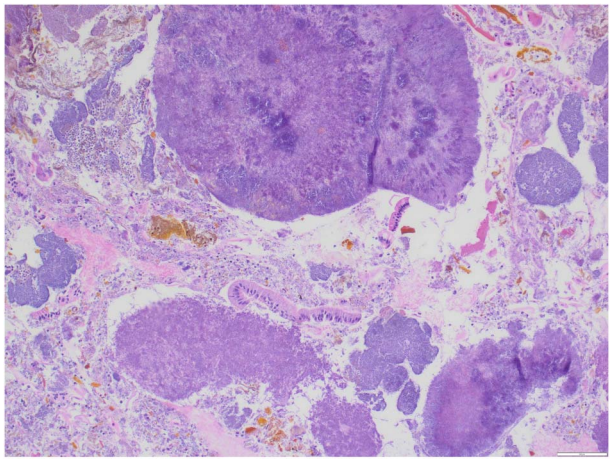
Introduction: Actinomyces are gram-positive anaerobic bacteria habitually found in the oral cavity, gastrointestinal and female genital tract. In rare cases, these bacteria may disrupt mucosal integrity and cause actinomycosis, a disease characterized by abscess and sinus tract formation, and fibrosis. We present a case of pancreaticobiliary actinomycosis.

Case Description/Methods: 50-year-old male with history of chronic pancreatitis complicated by biliary stricture requiring serial biliary stenting presented to the hospital with abdominal pain, decreased appetite, weight loss, and low-grade fevers. On exam the patient was hemodynamically stable and had mild epigastric abdominal tenderness. Labs revealed a lipase of 105 U/L, AST 20 U/L, ALT 18 U/L and total bilirubin of 0.7 mg/dL. Cross sectional imaging (CT scan) of the abdomen demonstrated a liver and pancreatic head mass at an outside institution two weeks prior to admission. Magnetic resonance cholangiopancreatography (MRCP) demonstrated dilated intrahepatic biliary ducts, bile duct of 11 mm with a stent in place, and intraluminal filling defects consistent of choledocholithiasis. Pancreatic head and body appeared bulky and heterogenous, whereas the pancreatic tail was atrophic. No focal liver lesions were seen. The patient underwent an endoscopic ultrasound which revealed calcific pancreatitis without solid masses. Endoscopic retrograde cholangiopancreatography (ERCP) was performed and the biliary stent was removed and sent for cytology. Cytology was negative for malignancy, however bacterial colonies consistent with actinomyces were found. Infectious disease service recommended the patient complete six weeks of ceftriaxone. After completion of treatment, his appetite returned, weight loss improved, and fevers resolved. Amoxicillin followed as maintenance therapy for approximately 12 months. **(Figure)**

Discussion: The symptoms of pancreaticobiliary actinomycosis are non-specific, making the diagnosis difficult. Actinomycosis should be included in the differential diagnosis when a pancreatic or liver mass is found, especially in a patient with a history of pancreatic or biliary stenting. In these patients, we suspect that the repeat cannulation of the bile duct during an ERCP may lead to mucosal disruption, allowing bacteria to infect the tissue. It is important for endoscopists to consider actinomycosis and pursue histopathological confirmation to prevent unnecessary surgical intervention.



[1883] **Figure 1.** Cell block preparation of bile duct brushing (H&E stain, 100x) showing benign-appearing columnar/ductal epithelium with filamentous bacterial colonies, consistent with Actinomyces.

S1884

A Case of Secondary Sclerosing Cholangitis Due to Polytrauma

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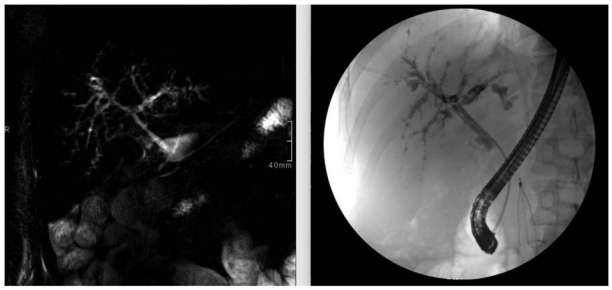
Introduction: Secondary sclerosing cholangitis (SSC) is a cholestatic biliary disease characterized by inflammation, fibrosis, stricture formation and ultimately destruction of the biliary tree leading to cirrhosis. SSC can appear morphologically similar to other biliary pathologies such as primary sclerosing cholangitis (PSC). However, unlike PSC, SSC is secondary to an underlying identifiable pathological process. We present a case of SSC occurring in the setting of a critically ill patient with polytrauma.

Case Description/Methods: A 48-year-old male with no past medical history presented to the emergency room after a motorcycle accident. The patient sustained many injuries including several orthopedic fractures and vascular injuries. He was admitted to the intensive care unit and underwent multiple surgeries. The patient's hospital course was complicated by several infections and septic shock. He also required ECMO and CRRT due to subsequent renal and cardiovascular failure. During his hospitalization, he developed persistent elevation of his liver enzymes, most notably his alkaline phosphatase (greater than 2,000U/L). An MRCP was performed and demonstrated intrahepatic biliary dilation with suspected multifocal areas of beading, which were not present on his initial abdominal imaging. A subsequent liver biopsy was obtained and showed prominent ductal proliferation with cholestasis. Gastroenterology was consulted and the patient underwent an ERCP which showed dilated intrahepatic ducts along with a beaded appearance (**Figure**). He was found to have an elevated IgG4 at 260mg/dL, raising the possibly of IgG4-mediated cholangiopathy. The patient's alkaline phosphatase began to downtrend and he was discharged home with close hepatology follow up.

Discussion: The most common causes of SSC include surgical or blunt biliary trauma, ischemic injury, intra-arterial chemotherapy, and recurrent pancreatitis. In order to diagnose a patient with PSC, one must first exclude secondary causes. Our patient's imaging demonstrated findings consistent with PSC; however, prior to his accident these findings were absent. He was found to have an elevated IgG4; however, the pathology from his liver biopsy did not demonstrate lymphoplasmacytic infiltration. Given the patient's history of polytrauma, septic shock, and overall critical illness, we suspect that he developed SSC. It is imperative for gastroenterologists to consider SSC on the differential of cholestasis as early diagnosis and treatment is associated with improved prognosis.



[1884] **Figure 1.** MRCP and ERCP showing dilated intrahepatic ducts with multifocal areas of beading.

S1885

A Diagnostic Conundrum: Pancreatic Retroperitoneal Fibrosis Masquerading as a Metastatic Pancreatic Tumor

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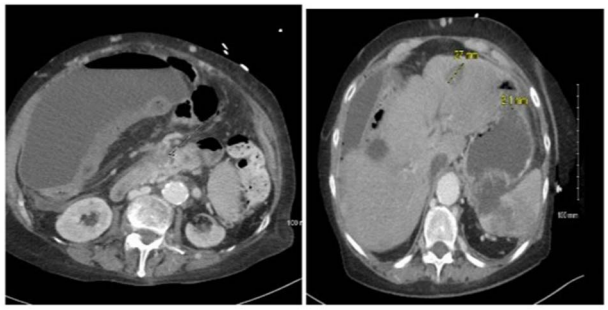
Introduction: Retroperitoneal fibrosis (RPF) is a rare condition, characterized by inflammation and progressive development of fibrotic mass in the retroperitoneal space. Due to the wide variety of presentations, diagnosis remains challenging, difficult, and often delayed. This case highlights an unusual presentation of this rare entity

Case Description/Methods: A 72-year-old female, with a history of hypothyroidism, presented with non-specific upper abdominal pain, abdominal distension, fatigue, and 24 pounds weight loss over 6 months. Physical examination was notable for ascites without stigmata of chronic liver disease. Blood work revealed normal CBC, CMP, and tumor markers. A CT scan of the abdomen revealed multiple hypodense ill-defined masses throughout the liver with soft tissues fullness of the pancreas. A mass in the root of the mesentery, which appeared to be originating from the pancreas, was seen on MRI, along with focal fatty infiltration of the liver. Steatohepatitis was seen in the liver biopsy, while pancreatic mass biopsy revealed acute inflammation with areas of fibrosis but no indication of malignancy (**Figure**). Repeat EUS-FNB returned with nonmalignant findings. Ascitic fluid cytology was negative for malignancy. she has had multiple hospitalizations for Spontaneous bacterial peritonitis, intra abdominal venous thrombosis, ileus etc. Repeated CT abdomen 4 months later, showed extensive desmoplastic reaction and fibrosis in the root of the mesentery, obliterating superior mesenteric vein and splenic vein producing cavernous transformation of the portal vein. ESR, CRP, and ANA were elevated. Serum IgG and IgG4 were elevated to 1723 and 156.9 respectively. ANA-specific antibodies, ANCA, and cardiolipin antibody were negative.IgG4 immunohistochemical analysis was performed however remained inconclusive with an average of 6 IgG4 and 22 IgG plasma cells per HPF due to insufficient sample tissue. The mass-forming lesion was diagnosed as IgG4-related retroperitoneal fibrosis. Treatment with prednisone was started with a very good clinical response. Follow-up CT a year later showed a stable mass. RPF-producing cavernous transformation of the portal vein, presenting as pancreatic mass, and portal hypertension have rarely been documented.

Discussion: RPF is a rare fibroinflammatory disorder affecting ~1.2 cases per 100,000 patients per year. CT, MRI, and PET are the mainstay of noninvasive diagnosis of the disease. Glucocorticoids are the mainstay of treatment.



[1885] **Figure 1.** A: Infiltrative soft tissue density mass above the pancreas concerning malignancy with occlusion of the portal vein and SMV. B: Indeterminate ill-defined hypodense hepatic lesions.

S1886

A Case Report of Iatrogenic Common Bile Duct Stricture

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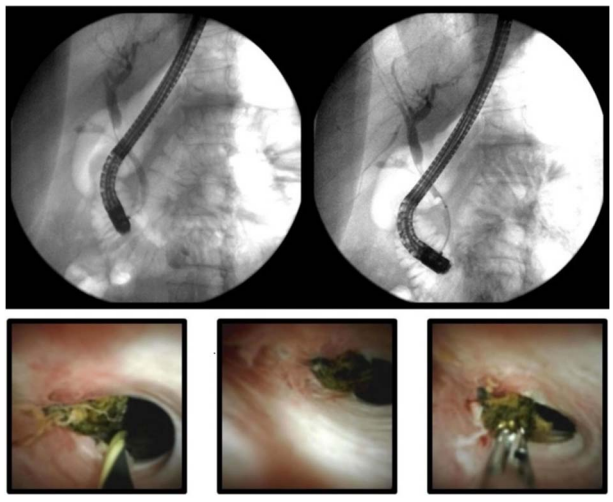
Introduction: Iatrogenic bile duct injuries (IBDIs) are common postsurgical complications following cholecystectomy. IBDI vary from a minor bile leakage to biliary stricture or a complete occlusion of the common bile duct. The authors report a case of a common bile duct stricture caused by suture material after an open cholecystectomy.

Case Description/Methods: A 57-year-old male with history of NAFLD presented with acute onset of severe epigastric abdominal pain, nausea and vomiting. Lipase was elevated. CT scan of the abdomen/pelvis revealed gallbladder wall thickening and dilated extrahepatic bile ducts consistent with cholecystitis and biliary obstruction. The patient underwent an open partial fenestrated cholecystectomy which was complicated by injury of the gastric antrum requiring primary two-layered repair and omental patching. ERCP was not performed due to gastric injury. His symptoms improved and he was discharged home. One month later he returned with severe epigastric pain. Initial work-up revealed elevated lipase and transaminases. CT of the abdomen/pelvis showed peripancreatic fluid and fat stranding with persistent mild biliary dilatation. MRCP identified three choledocholiths at the level of the ampulla. ERCP was performed with sphincterotomy, balloon extraction of several stones and sludge, and placement of a plastic biliary stent. There was an area of resistance felt during balloon sweep. He was discharged after symptom resolution with recommendations to repeat ERCP in 6 weeks for stent removal. A repeat ERCP revealed persistent area of resistance with filling defect on fluoroscopy. (**Figure**). A spyglass revealed a suture material and mild inflammation in the CBD at the cystic duct takeoff (**Figure**). Stone formation induced by suture material is thought to be the culprit of the recurrent acute pancreatitis.

Discussion: IBDIs account for the majority of postsurgical complications after cholecystectomy. The incidence of IBDIs is higher in laparoscopic cholecystectomy when compared to open cholecystectomy. IBDIs are associated with significant perioperative morbidity, mortality, and prolonged hospital stay. If left untreated, they can result in life-threatening complications such as cholangitis, secondary biliary cirrhosis, and portal hypertension. One of the uncommon postoperative complications is stone formation induced by suture material used in cystic duct ligature. Surgical sutures can function as a nidus for crystallization leading to obstruction and development of large choledocholiths.



[1886] **Figure 1.** Top. ERCP shows filling defect in the common bile duct. Bottom: Spyglass shows suture material and adjacent mild imflammation of the CBD at the systic duct takeoff.

S1887

A Case of PEG-Induced Pancreatitis

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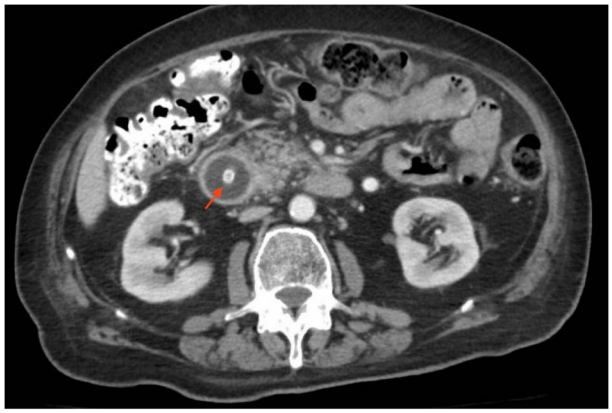
Introduction: PEG (percutaneous endoscopic gastrostomy) tube placement is a commonly performed procedure for patients in need of long-term enteral nutrition. It is a relatively safe procedure, with some complications including bleeding, infection, buried bumper syndrome, and dislodgement. Even rarer is the tube migration into the duodenum, obstructing the ampulla, causing biliary obstruction and pancreatitis. We present a case of pancreatitis secondary to migrated PEG tube bumper [1, 2].
Case Description/Methods: An 83-year-old male with a history of stroke and dysphagia, who one year prior had a PEG tube placed presenting with vomiting and epigastric pain and tenderness. Initial lipase was 1761 (reference 8-69 U/L), alkaline phosphatase was 145 (reference 36-112 IU/L), transaminases, bilirubin, and triglycerides were normal. Abdominal CT scan showed a migrated PEG tube balloon in second part of the duodenum at level of the ampulla (**Figure**). As it turned out patient was tolerating oral diet thus PEG was no longer needed. GI team deflated the balloon and removed the PEG without any further complications. With aggressive fluid resuscitation, patient's symptoms resolved after two days.
Discussion: Pancreatitis due to migrated PEG tube obstructing the ampulla is a rare entity, with only a few case reports published. Patient's can present with nonspecific symptoms with significantly elevated hepatic and pancreatic enzymes. CT findings can show pancreatic inflammation and biliary/pancreatic duct dilation with PEG bumper at level of ampulla [1, 2]. Physical exam findings would show very little external tubing indicating distal internal migration of PEG bumper. Interestingly this can also cause intermittent gastric outlet obstruction. Treatment is PEG tube removal/replacement, and IV fluids. Marking the tube at the insertion site can help in diagnosis of migration, if it happens. Patients and care givers should be educated about tube care, regular follow-ups to ensure positioning and to ascertain the timing of removal.



[1887] **Figure 1.** Migration of percutaneous gastrostomy tube balloon to second portion of duodenum at level of ampulla.

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S1888

A Curious Case of Recurrent Pancreatitis: A Case Report

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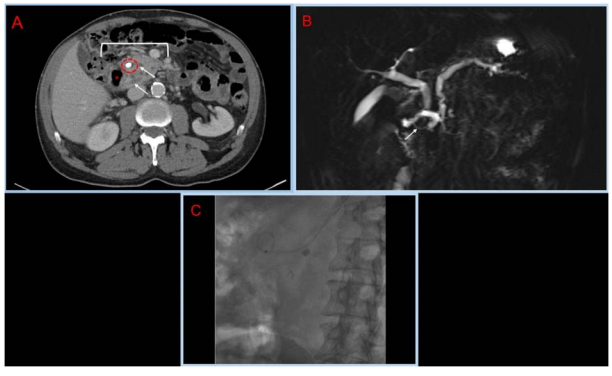
Introduction: Annular pancreas, a congenital anomaly characterized by incomplete rotation of the ventral pancreatic bud, causes a ring of pancreatic tissue to encircle the duodenum. It is commonly accompanied by pancreatic divisum, which occurs when the main pancreatic duct and accessory duct fail to fuse. Patients often present with recurrent pancreatitis due to partial obstruction of the pancreatic duct caused by fibrosis.
Case Description/Methods: A 72-year-old Caucasian male presented with recurrent episodes of acute pancreatitis. A CT abdomen (Figure A) revealed pancreatic tissue surrounding the second portion of the duodenum consistent with an annular pancreas. A pancreatic duct stone was also noted. An MRCP (Magnetic resonance cholangiopancreatography) was consistent with annular pancreas with persistent dilation of the main and accessory pancreatic ducts and a 6mm stone noted in the pancreatic duct (Figure B). On an initial ERCP (Endoscopic retrograde cholangiopancreatography), the pancreatic duct could not be cannulated through the major papilla and minor papilla could not be identified due to inflammation. An EUS (Endoscopic ultrasound) was performed, which was suggestive of pancreas divisum. The minor papilla was cannulated, and a stent was placed in the pancreatic duct. In a subsequent ERCP (Figure C), the patient had the pancreatic stone removed, with complete resolution of his symptoms.
Discussion: Typically, patients with annular pancreas present with symptoms during childhood. While infrequent, some patients remain asymptomatic until 20-50 years of age. This case demonstrates the potential to remain asymptomatic until even 72 years of age. Thus, it is important for physicians to be aware of the clinical manifestations of annular pancreas, as they could arise at any stage in life. By increasing awareness surrounding annular pancreas and management of its complications, we hope to increase the rate of early intervention.



[1888] **Figure 1.** Image 1A. Contrast-enhanced axial CT of the abdomen at the level of the pancreatic head. The pancreas (white bracket) is seen surrounding the 2nd part of the duodenum (white

asterisk), compatible with annular pancreas. A 6 mm calcification (circled) is present in the region of the minor papilla (arrows indicating pancreatic ducts). Image 1B. Coronal MRCP images. Annular pancreas, with only a thin rim of pancreatic tissue surrounding the duodenum. The dorsal duct is best shown to wrap completely around the duodenum on MRCP images (arrow). Image 1C. ERCP fluoro image showing the previously placed pancreatic duct stent (which does appear to have a loop-like configuration) and the pancreatic stone.

S1889

A Case of Ruptured Gastroduodenal Artery Pseudoaneurysms in a Patient With Chronic Pancreatitis

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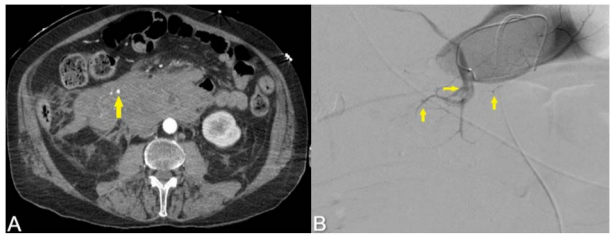
Introduction: Pancreatic pseudoaneurysm is a rare vascular complication of pancreatitis, with an estimated prevalence of 10%. Pseudoaneurysm can result from trauma, inflammation, infection, and iatrogenic causes. Due to its weak structural support, the risk of rupture is higher for pseudoaneurysm than that of a true aneurysm of similar size. Pancreatic pseudoaneurysms can lead to life-threatening complications such as rupture and bleeding, with increased morbidity and mortality up to 90% in untreated patients and 12.5% despite treatment. Due to its high mortality rate with rupture, pseudoaneurysms should be treated immediately once it is identified, regardless of their size or whether it is symptomatic or not
Case Description/Methods: We present a case of a 68-year-old male patient with a past medical history significant for chronic pancreatitis who presented with altered mental status and hypovolemic shock secondary to unexplained gastrointestinal and retroperitoneal hemorrhage (**Table**). Computed tomography (CT) Angiography was done which showed an enlarging retroperitoneal and right-sided intraperitoneal hematoma with suspicion for active arterial extravasation or punctate pseudoaneurysms in the pancreatic head (**Figure**). Celiac angiogram was done under fluoroscopic guidance, which showed gastroduodenal and superior pancreaticoduodenal artery pseudoaneurysms. He underwent successful endovascular coil embolization of the gastroduodenal artery. Post procedure he was monitored in intensive care unit. His clinical condition continued to improve and was discharged to a skilled nursing facility in stable condition.
Discussion: Physicians should be aware of pancreatic pseudoaneurysms as rare vascular complication associated with chronic pancreatitis that may lead to fatal gastrointestinal or retroperitoneal bleeding with a high mortality rate. Our case highlights the importance of early diagnosis and prompt treatment in these patients.



[1889] **Figure 1.** A: CT abdomen and pelvis without intravenous contrast showing peripancreatic inflammation surrounding the pancreatic body and head along with retroperitoneal bleed (red arrow). Pancreatic calcifications present (yellow arrow) signifying radiologic evidence of chronic pancreatitis B: CT Angiography showing retroperitoneal and right-sided intraperitoneal hematoma with active arterial extravasation or punctate pseudoaneurysms in the pancreatic head.

Table 1. Laboratory Investigation

Investigation	Day 1 of Hospitalization	Day 5	At Time of Discharge	Reference Range
White Blood Cell count	24.3	14.3 k/uL	7 k/uL	4.8-10.8 k/uL
Red Blood Cell Count	4.77	2.69 MIL/uL	3.19 MIL/uL	4.50-5.90 MIL/uL
Hemoglobin	15.8	8.5 mg/dl	10.2 mg/dl	12.0-16.0 g/dL
Hematocrit	47.6	25.7 %	30.5%	42-51 %
Platelet count	171	155 k/uL	215 k/uL	150-400 k/uL
Sodium, Serum	132	139 mEq/L	140 mEq/L	135-145 mEq/L
Potassium, Serum	3.0	3.2 mEq/L	3.9 mEq/L	3.5-5.0 mEq/L
Blood Urea Nitrogen, Serum	41	17 mg/ml	10 mg/ml	8-26 mg/dL
Creatinine, Serum	1.2	0.5 mg/dl	0.6 mg/dl	0.5-1.5 mg/dL
Bilirubin, Serum total	0.5	0.7 mg/dl	0.3 mg/dl	0.2-1.1 mg/dL
Serum Direct Bilirubin	0.3	0.3 mg/dl	< 0.2 mg/dl	0.0-0.3 mg/dL
Alkaline Phosphatase	51	48 units/L	73 units/L	56-155 unit/L
Aspartate Transaminase	125	21 units/L	18 units/L	9-48 unit/L
Alanine Aminotransferase	81	30 units /L	12 units /L	5-40 unit/L
Lactic acid Level	8.2	0.8 mmoles/L	1.1 mmoles/L	0.5-1.6 mmoles/L
Prothrombin Time	15.2	13.9 sec	10.5 sec	9.9-13.3 seconds
International Normalized Ratio	1.31	1.20	0.92	0.85-1.14
Serum Calcium	6.6mg/dl	7.6 mg/dl	9.2 3 mg/dl	8.5-10.5 mg/dl
Serum Lipase	24 U/L			< =61U/L
Serum Triglyceride	163mg/gl			55-150 mg/dl
Serum Ethanol	< 10			< = 10mg/dl
Urine Toxicology	Cocaine and methadone			Negative

S1890

A Disappearing Act: Spontaneously Resolving Pancreatic Pseudocyst Mimicking Duodenal Neoplasm

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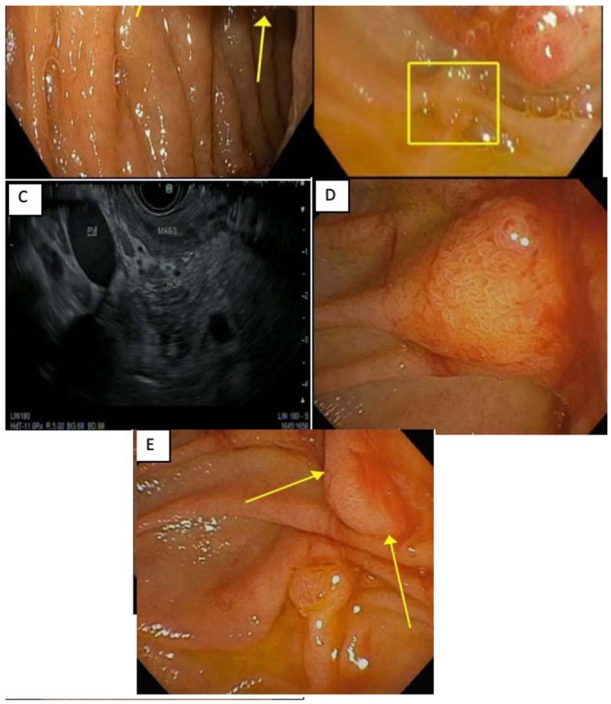
Introduction: Pancreatic head lesions have a broad differential ranging from inflammatory to malignant etiologies. Here we present a case of a pancreatic pseudocyst with surrounding inflammation mimicking a duodenal mass with neoplastic appearance on EGD/EUS only to spontaneously resolve weeks later.
Case Description/Methods: A 49-year-old female with history of decompensated liver cirrhosis secondary to alcohol use presented with persistent nausea, vomiting, abdominal pain, and 10-pound weight loss. A CT revealed a 5.4x4.1x4.1 cm "pancreatic head mass" with heterogeneous enhancement of the right hepatic lobe and mesentery concerning for metastasis. CA 19-9, CEA, and AFP were all normal. A repeat CT with pancreatic protocol was subsequently performed suggesting the mass may be a cystic lesion with mass effect over the proximal duodenum with heterogeneous contents more consistent with a pancreatic pseudocyst. Therefore, we performed an EGD/EUS for further delineation. (**Figure)** This procedure revealed a large ulcerated, fungating and infiltrative peri-ampullary duodenal mass at the 2nd portion of the duodenum, sparing the major papilla. On EUS, the "mass" measured 4.0x3.8 cm spanning the mucosa to the preduodenal region with close abutment to the pancreatic head. We collected biopsies of the duodenal mass and alerted the patient to her potential cancer diagnosis. She was discharged home with palliative support to follow up as an outpatient. Biopsy results returned as benign duodenal mucosa admixed with inflamed granulation tissue but no tumor. Given continued concern for a mass, a repeat CT was performed several weeks later with a shocking interval resolution of both the aforementioned pancreatic head lesion and the adjacent duodenal thickening. The next month an EGD was performed for a separate indication, in which the peri-ampullary duodenal lesion appeared smaller with significant improvement. Repeat biopsy again showed inflamed duodenal mucosa with no dysplasia. Our follow up MRI with pancreas protocol confirmed resolution of the lesion.
Discussion: With this patient's clinical and radiological signs of cancer, further diagnostic evaluation was required with EGD/EUS. However, here we saw an unusual presentation of a duodenal "mass" on endoscopy later revealed to be a pancreatic pseudocyst with adjacent organ involvement based on diagnostic sampling and spontaneous resolution on follow up imaging. It is important to be wary of non-neoplastic processes that may mimic neoplastic ones.



[1890] **Figure 1.** (A) Duodenal mass visualized from forwarding viewing scope in 2nd portion of duodenum. (B) Duodenal mass and major papilla (square) visualized with duodenoscope. (C) EUS imaging with the head of the pancreas, the duodenal mass, and portal vein. (D+E) Resolving peri-ampullary duodenal lesion on repeat EGD.

S1891

A Diagnostic Dilemma: Primary Sclerosing Cholangitis With High IgG4 vs IgG4-Related Sclerosing Cholangitis

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Introduction: Two etiologies of sclerosing cholangitis with overlapping features are Type 2 IgG4-Related Sclerosing Cholangitis (IgG4-SC) and Primary Sclerosing Cholangitis with high IgG4 (PSC). Here, we present a complex case of a young patient with features of both diseases.
Case Description/Methods: A 25-year-old Caucasian female with a medical history of diabetes and no previous pancreatic disease was incidentally found to have an AST and ALT in the low 200s, total bilirubin 2.6, and alkaline phosphatase of 1130 concerning for cholestatic obstruction. She was asymptomatic and not jaundiced at the time. Subsequent MRCP revealed a dilated common bile duct. The patient underwent ERCP/EUS demonstrating a common bile duct stricture. A plastic stent was placed, and cells for cytology/FISH analysis were unyielding for malignancy. Repeat ERCP/EUS was performed a few months later for stent exchange. Biopsies to evaluate the indeterminate stricture yielded IgG4+ plasma cells up to 14/HPF. Based on the findings suspicious for IgG4-SC, she started prednisone therapy for one month. Subsequent labs demonstrated mild improvement in alkaline phosphatase to 450, total bilirubin 0.2, and a normal IgG4 level but with persistent elevation of total IgG 1.5 times the upper limit of normal. Repeat ERCP demonstrated resolution of the distal biliary stricture. She never developed abdominal pain, jaundice, weight changes or loss of appetite. Six months later, LFTs began to uptrend and a third ERCP/EUS demonstrated new distal biliary stricture. Biliary biopsies showed dense lymphoplasmacytic infiltrates and fibrosis with an average of 10 IgG4 positive plasma cells/HPF. Liver biopsies showed periductal fibrosis and a fibro-obliterative duct lesion. Given the concern for PSC, colonoscopy was performed with biopsies showing mild crypt distortion, mild eosinophilia, and rare neutrophils in the lamina propria. The patient remained asymptomatic throughout the disease course but had persistent biochemical hepatitis despite being on steroid therapy. A shared provider-patient decision was made to withhold immunosuppressive or biologic therapy.
Discussion: In this case, the patient's epidemiologic, clinical, and histological factors were more consistent with PSC. Conversely, the mild biochemical and stricture steroid-responsiveness were initially more suggestive of IgG4-SC. It is imperative to delineate between the two, as prognosis, treatment-responsiveness, and anticipated comorbidities differ.

S1892

A Double Take on a 20-Year Double J-Stent

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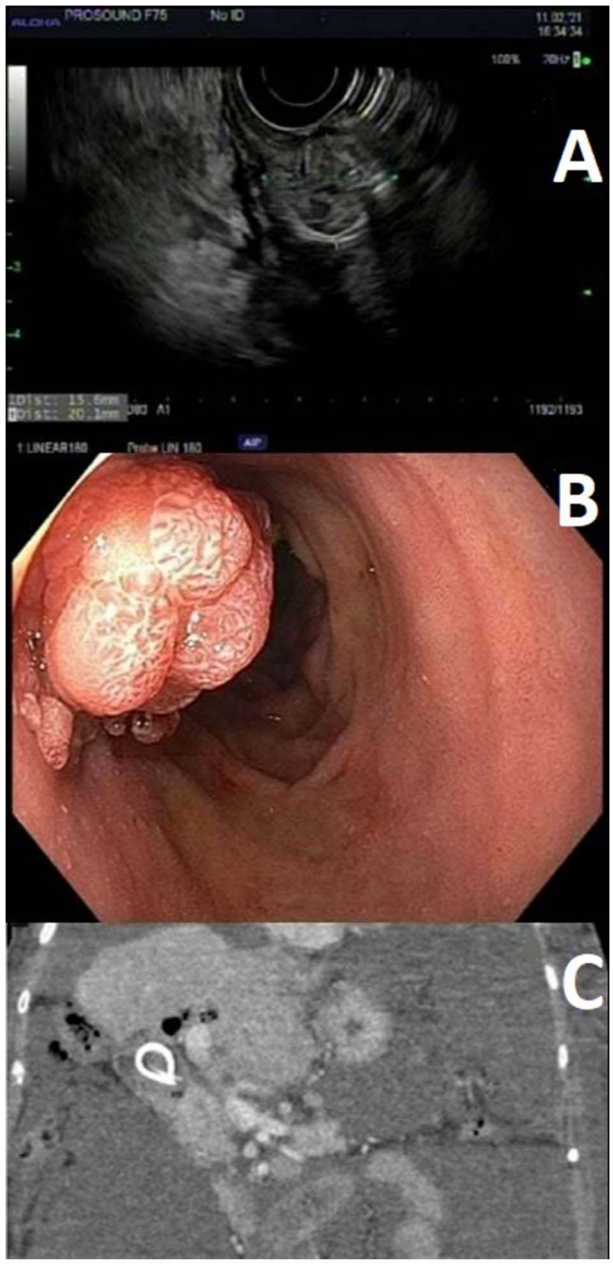
Introduction: The double J biliary stent is commonly used to relieve obstructions and aid in draining of pancreatic fluid. Indwelling or forgotten stents are rarely seen and their sequelae are unknown. We present the case of a patient who was lost to follow-up over twenty years ago following cholecystectomy and placement of double J stent. A routine esophagogastroduodenoscopy (EGD) for variceal surveillance was performed and discovered an ampullary fungating mass.

Case Description/Methods: A 79 y/o female with a history of hypertension presented to the ED with worsening abdominal distention. She was a poor historian but denied alcohol use, drug use, or any history of liver disease. Her workup included an abdominal paracentesis removing greater than 7 liters of fluid with a SAAG indicating portal hypertension. She had abnormal liver function tests (LFTs) and CT imaging was consistent with cirrhotic morphology of the liver and a double-J stent present in CBD (**Figure A**). An EGD for further evaluation and for variceal vein monitoring was done revealing grade 2 varices but more significantly, at the site of ampulla of vater a large fungating mass was noted. The mass was medium-sized and polypoid with no bleeding. The CBD stent was in place with noted hyperproliferation of the tissue of the ampulla vs a periampullar duodenal mass (**Figure** B). A repeat EDG with EUS was performed, finding the hypoechoic non-circumferential mass endosonographically within the ampulla. The mass measured 24 mm by 14 mm in maximal cross-sectional diameter (**Figure** C). The lesion extended from the mucosa to the muscularis mucosa. The endosonographic borders were well-defined. There was sonographic evidence suggesting invasion into the deep mucosa (Layer 2). Filamentous microorganisms were identified on HE and GMS stain. No individual atypical AE1/AE3 positive epithelial cells were identified.

Discussion: This case highlights the unusual sequelae of a retained double-J stent in a patient who was lost to follow up. It's unknown why the stent was placed, likely during an intraoperative cholangiogram, and the patient did not recall her follow-up instructions. Several questions arise surrounding the mass such as, did it have malignant potential and what role, if any, did the stent have in the formation of the mass? Did colonization represent an increased risk of ascending cholangitis? The stent was left in at the conclusion of the case and the patient was extensively educated about follow-up and maintenance endoscopy.



[1892] **Figure 1.** CT evidence of pneumobilia, ampullary fungating mass, and EUS imaging.

S1893

A Case of Silent Cancer: CT Negative Ampullary Adenocarcinoma

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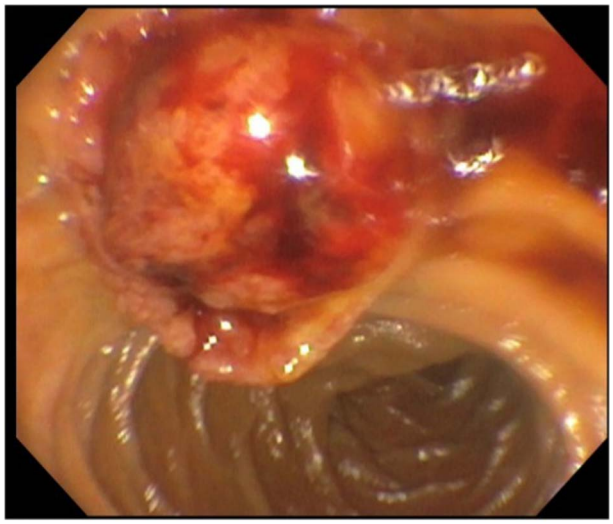
Introduction: Ampullary cancers are rare and account for approximately 0.2% of gastrointestinal cancers. These patients tend to have an improved prognosis as they typically present early and can often be diagnosed with imaging, leading to higher surgical resection rates than other pancreatobiliary malignancies. Early detection is crucial as surgical resection can be curative.
Case Description/Methods: A 59-year-old male with a history of hypertension and a prior cerebrovascular accident presented with 6 weeks of painless jaundice. He experienced itching, diarrhea, bloating, fatigue and 4–5-pound weight loss for the same duration of time. Initial labs showed a total bilirubin of 19.9, direct bilirubin of 14.7, alkaline phosphatase of 203, ALT of 112 and AST of 112. CT with IV contrast showed intra and extra-hepatic dilatation with the common bile duct greater than 20 mm. There was no evidence of biliary mass or pancreatic mass noted. The patient underwent ERCP where a large (5cm x 3cm) fungating ulcerated lesion was found (**Figure**). The mass was friable and appeared to extend into the biliary duct. Three 8 French by 9.5 cm plastic stents were placed into the common bile duct. Biopsies were obtained and the pathology resulted as adenocarcinoma.
Discussion: Ampullary adenocarcinoma is rare, with an incidence of 0.70 per 100,000 men and 0.45 per 100,000 women in the US. These patients present with symptoms of biliary obstruction and radiographic imaging demonstrating the double duct sign. Often imaging also demonstrates a discrete nodular mass that produces a filling defect at the pancreaticobiliary junction. In some cases, despite the presence of ductal dilation, there is no evidence of an obstructing mass, even on endoscopic evaluation, and a diagnosis is made solely through biopsy. Our patient presented with symptoms of obstructive jaundice with CT evidence of biliary and pancreatic duct dilation without radiographic evidence of a mass. Only through ERCP were we able to identify the large mass, which was diagnosed as adenocarcinoma. This case did not follow the typical presentation, as a mass was not seen on imaging and could have delayed diagnosis and treatment. We recommend the addition of diffusion weighted imaging to the diagnostic algorithm as it has been shown to improve the detection rate when compared to conventional imaging.



[1893] **Figure 1.** Endoscopic view of ampullary mass during ERCP.

S1894

A Case of Severe Acute Alcoholic Pancreatitis Complicated With Abdominal Compartment Syndrome

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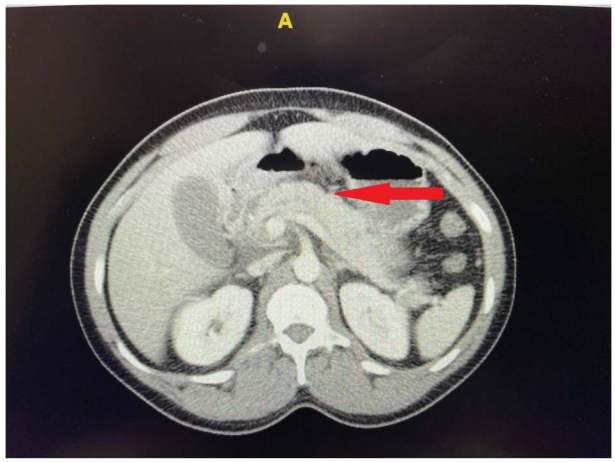
Introduction: Abdominal compartment syndrome (ACS) is a sustained intra-abdominal pressure (IAP) above 20mmHg with associated new-onset organ dysfunction. Even though it is rarely seen in clinical practice, up to 15% of patients with severe acute pancreatitis (SAP) develop ACS and have a mortality rate of up to 49%. Given that delayed treatment of ACS is associated with high mortality and adverse outcome, a higher clinical suspicion is needed to aid diagnosis. We present a case of severe pancreatitis complicated with ACS.
Case Description/Methods: A 29-year-old man with a history of alcohol abuse presented with five days of severe abdominal pain. He was diagnosed with SAP. About 8 hours into admission to the ICU and resuscitation, he began having worsening work of breathing and persistent severe abdominal pain despite being on adequate opioid analgesic. Physical examination revealed tachycardia, diffuse peritoneal signs, guarding, and shock. Repeat labs revealed worsening leukocytosis, thrombocytopenia, hyperkalemia, hypocalcemia, lactic acidosis, and elevated creatinine kinase. There was also evidence of renal failure. An edematous pancreas with some peripancreatic fluid collection was evident on the CT abdomen (**Figure**). The abdominal x-ray was unremarkable. Indirect intra-abdominal pressure (IAP) by intravesical catheter pressure measurement was 35mmHg. He commenced medical management with neuromuscular paralysis and mechanical ventilatory support. A repeat IAP was 24mmHg. He then got surgical decompression by full-thickness midline laparotomy with wound vac placement which further reduced the IAP to 10mmHg. He received serial dialysis, correction of abnormal electrolytes, and serial transfusion with blood products as required. He remained critically ill on a mechanical ventilator and vasopressors, with a poor prognosis at the time of this writing.
Discussion: The pathophysiology of elevated IAP in SAP is multifactorial. These include retroperitoneal inflammation, ascites, acute peripancreatic fluid collections, visceral edema, and aggressive fluid resuscitation. We believe some of the above listed had ensued in our patient for days before he presented to the hospital. Physical examination is inaccurate in diagnosing ACS. The first-line treatment is medical paralysis followed by surgical decompression if ineffective. We are writing this case to recommend early serial IAP measurements in patients with SAP.



[1894] **Figure 1.** CT abdomen showing edematous pancreas with some peripancreatic fluid collection.

S1895

A Case Review of Spontaneous Splenic Rupture Caused by Pancreatitis With Subsequent Peripancreatic Abscess

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Introduction: Spontaneous rupture of the spleen, a rare presentation, most commonly occurs secondary to a pathological inciting event. Most common causes include malignancy, infections, or inflammatory causes such as pancreatitis. Very few documented cases with imaging currently exist in literature, lending to call for more established treatment chronology and recommendations. Here, we present a case of non-traumatic spontaneous splenic rupture in the setting of sepsis with subsequent peri-pancreatic abscess.
Case Description/Methods: A 34-year-old male with a history of alcohol use disorder presented for two days of severe left upper quadrant abdominal pain with radiation to the left lateral side. His vitals included T 37.4 C, HR 140, BP 106/86, RR 20, O2 sat 95% on room air. The patient was diaphoretic, tender to palpation in the LUQ, with scleral icterus. Labs were significant for alk phos 125, AST 122, ALT 138, total bilirubin 3.1, lipase 107. CBC demonstrated WBC 18.1. CT abdomen/pelvis was remarkable for atraumatic splenic laceration with pleural effusion bilaterally with compressive atelectasis. CT imaging demonstrated an encapsulated fluid collection at the splenic hilum/ pancreatic tail with subtle peripheral wall enhancement. These findings may represent a walled off pancreatic pseudocyst, organized hematoma or possibly abscess. After interdisciplinary discussion, the patient was seen by both gastroenterology and interventional radiology with decision made to treat the patient with IV fluids, as well as antibiotic therapy and definitive management with percutaneous drain placement. It was also established that the finding of pseudocyst and presentation made it difficult to provide the radiographic diagnosis of pancreatitis. Through serology and further history, the spontaneous splenic rupture was considered to be most likely caused by an inflammatory process such as acute or chronic pancreatitis.
Discussion: With this case, we demonstrate the presentation of peri-pancreatic abscess, which is a diagnosis that is unique within itself. In consideration of its pathogenesis, we discuss both the rarity of this pathology as a sequelae of pancreatitis as well as an almost idiopathic splenic laceration. With our case, we call for further reading and literature on the diagnosis and management of atraumatic splenic rupture with subsequent abscess formation, and allow for unique imaging and our treatment methods to be presented amongst learners and practitioners.

S1896

A Rare Case of *Strongyloides* Cholangitis With Portal Vein Thrombus and Recurrent Intra-Abdominal Abscesses

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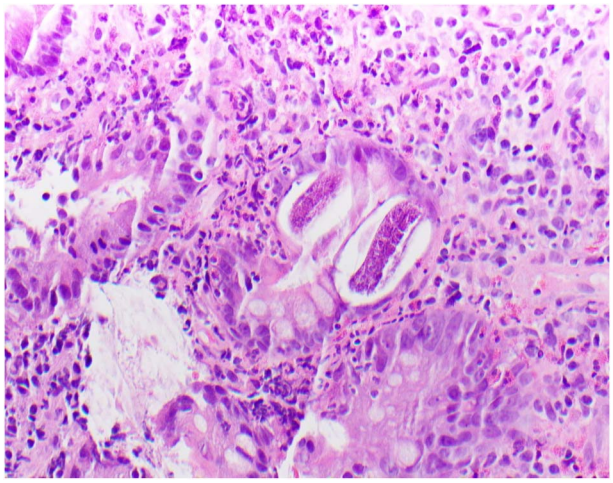
Introduction: *Strongyloides stercoralis* infection outside of the gastrointestinal tract is extremely uncommon, particularly in an immunocompetent host. We present a rare case of *S. stercoralis* cholangitis complicated by portal vein thrombus and recurrent intra-abdominal abscesses in an immunocompetent patient.
Case Description/Methods: A 65-year-old male who immigrated to the United States (US) over 40 years prior from Nigeria, presented with 4 days of epigastric pain, weakness, fevers and anorexia. His medical history was notable for COPD and an intra-abdominal abscess two years prior. His previous abscess was treated with percutaneous drainage and antibiotics. On arrival, he was afebrile and tachycardic to 110 bpm. He appeared jaundiced, and his abdomen was distended with right upper quadrant tenderness. Labs showed AST 121 IU/L, ALT 263 IU/L, ALP 267 IU/L, total bilirubin 8.3 mg/dL (direct 5.4 mg/dL), WBC 13.6 K/uL, eosinophils 0.28 K/uL. Non-contrast-enhanced abdominal computerized tomography (CT) scan showed an 8.4 cm subdiaphragmatic collection adjacent to the gastric fundus and intra- and extrahepatic ductal dilation. MRCP showed luminal irregularity of the common bile duct with multiple areas of stenosis of intra- and extrahepatic bile ducts and cavernous transformation of a chronic portal vein thrombus. Aspirates of the sub-diaphragmatic fluid collection and blood cultures grew *Pseudomonas aeruginosa*. Intravenous piperacillin-tazobactam was started. Upper endoscopy and endoscopic ultrasound of the stomach showed normal gastric and duodenal mucosa with multiple large periduodenal varices. Duodenal biopsies revealed *Strongyloides* species (**Figure**). *S. stercoralis* serologies were positive and HIV antibodies were negative. He underwent surgical washout of the abdominal abscess and was treated with 2 weeks of oral ivermectin and ciprofloxacin.
Discussion: *S.stercoralis* transmission occurs when human skin contacts the infective larvae. Carriers may remain asymptomatic for decades. Symptoms can include pruritus, diarrhea, weight loss and abdominal pain. Complications such as biliary tree invasion and portal vein thrombus are rare but have been reported. Deposition of eggs in the bowel wall can lead to bacterial gut translocation, gram negative sepsis and recurrent abscesses. Disseminated strongyloidiasis typically occurs in the setting of immunosuppression, HIV, or hematologic malignancies and rarely occurs in an immunocompetent host. Diagnosis can be challenging.



[1896] **Figure 1.** Strongyloides organism on duodenal biopsy.

S1897

A Rare Case of Hypercalcemia in the Setting of Relapsing Multiple Myeloma Presenting as Acute Pancreatitis

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Introduction: Hypercalcemia induced pancreatitis has a reported prevalence of 1.5-8%. Most reported cases have been secondary to hyperparathyroidism and few are secondary to malignancies or granulomatous diseases. Despite multiple myeloma (MM) commonly presenting with hypercalcemia, there are very few cases reported in literature of hypercalcemia induced pancreatitis in patients with MM.
Case Description/Methods: A 70-year-old male with past medical history significant for MM and chronic kidney disease presented to the hospital with recurrent, constant dull epigastric abdominal pain, non-radiating, 6/10 in intensity, worsened by movement and deep inspiration with no relieving factors. He denied any associated factors. He has undergone a bone marrow transplant in 2017 for his MM but relapsed in 2019. On initial presentation workup was significant for pancytopenia, an elevated lipase level of 1170 and an elevated corrected calcium level of 15. Computed tomography (CT) of the abdomen and pelvis revealed no abnormality of the pancreas. MRCP ruled out choledocholithiasis and gallbladder US was negative for cholelithiasis. He denied alcohol use and triglyceride levels were not significantly elevated. Medication overview did not show any relevant medications that may induce pancreatitis. It was determined that the etiology of his pancreatitis was hypercalcemia in the setting of MM.
Discussion: Hypercalcemia is present in 30% of patients with malignancy. MM commonly presents with hypercalcemia (19% of cases) but there are a handful of cases reported of calcium induced pancreatitis MM patients. Extensive workup is required to determine the etiology of acute pancreatitis starting from common to uncommon predisposing factors. In some cases it may remain cryptogenic. Multiple Myeloma patients may present with hypercalcemia in the setting of increased bone resorption caused by osteoclast activation. It is suggested that high serum calcium levels could be responsible for calcium deposit in the pancreatic ducts and activation of pancreatic enzymes, ultimately leading to pancreatitis. It is important as clinicians to keep in mind this rare presentation of hypercalcemia in MM patients in order to treat and manage the disease effectively and in a timely manner, improving patient outcomes. As calcium levels were normalized by hypercalcemia treatment, our patient's presenting symptoms significantly improved and later resolved.

S1898

A Rare Case of Delayed Bilioptysis Due to Radioemobilization of Intrahepatic Cholangiocarcinoma: Case Report and Review of Literature

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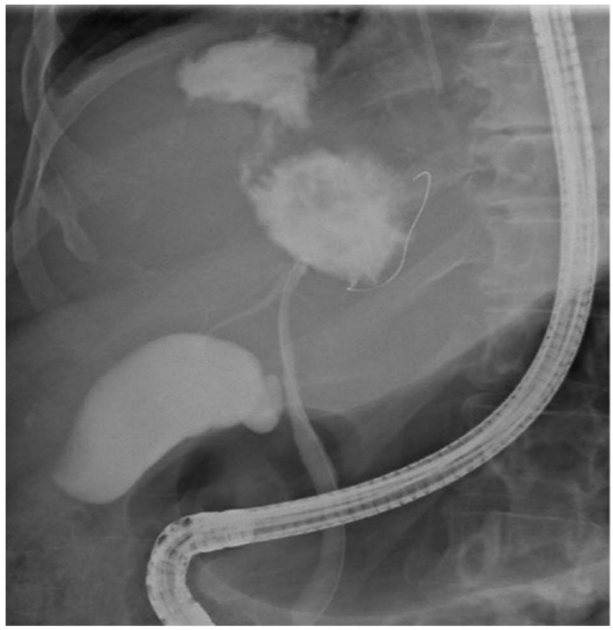
Introduction: Bronchobiliary fistula (BBF) is a rare condition that describes a pathologic communication between the biliary tree and bronchial airway. It can occur in the setting of trauma, infection, malignancy, or iatrogenesis. Bile in sputum (bilioptysis) is pathognomonic. We present a rare case of BBF managed with both ERCP and IR embolization.
Case Description/Methods: A 73-year-old female was diagnosed with large intrahepatic cholangiocarcinoma in segment 7/8 and was treated with systemic chemotherapy and radioembolization of the segment 6/7/8 branches of her right hepatic artery. Approximately 10 months after the initial radioembolization, residual viable disease in the right lobe of the liver was treated with repeat radioembolization combined with microwave ablation. Eighteen months after the initial radioembolization, she presented with progressive productive cough unresponsive to antibiotics and steroids. Her cough then turned bright yellow concerning for bilioptysis, and she was admitted for presumed BBF. CT thorax showed right lower lobe pneumonia, enlarging pulmonary nodules, and multiple peripherally enhancing liver lesions. ERCP was performed and established the diagnosis of BBF, via cholangiography (**Figure**), which was treated with biliary sphincterotomy and placement of a 10 Fr x 12 cm double pigtail stent into the biloma. Immediate reduction of bright yellow sputum was observed. Subsequent HIDA scan showed a large biloma overlying the right hepatic dome and persistent BBF. The patient underwent embolization of the BBF and placement of percutaneous drain into the biloma. The patient's bilioptysis has resolved for three weeks, and further embolization of the BBF has not been necessary. The patient has stabilized and is scheduled have a follow up ERCP.
Discussion: Intrahepatic cholangiocarcinoma treated with radioembolization is a rare cause of BBF. The pathophysiology includes chronic subdiaphragmatic inflammation with breakdown of the local diaphragm and lung tissue or frank invasion of the diaphragm and lung by an active liver process. Due to the infrequent presentation of this entity, there is no treatment protocol established for management of BBF. ERCP is a minimally invasive option that can be considered for both diagnosis and treatment of BBF, either alone or in conjunction with IR embolization.



[1898] **Figure 1.** Cholangiography demonstrating bronchobiliary fistula.

S1899

A Rare Case of Acute Necrotizing Pancreatitis Infected With *Prevotella* Species

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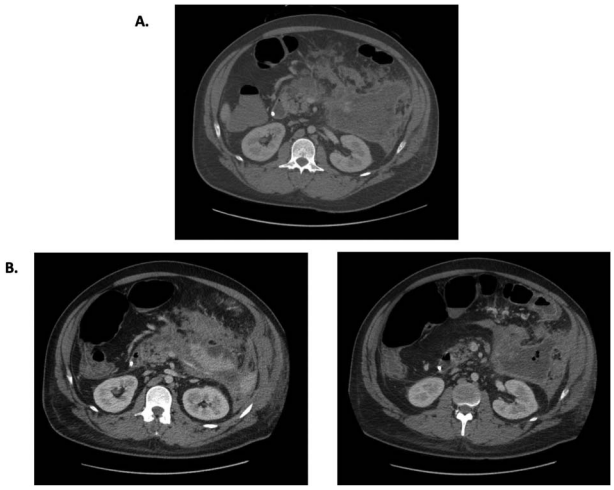
University of Pittsburgh Medical Center, Pittsburgh, PA.

Introduction: Acute pancreatitis is one of the leading causes of GI-related hospitalizations in the USA. Around 20% of these cases are characterized as severe, leading to complications including necrotizing pancreatitis.[1] Enterococci are the most-commonly isolated species in infected pancreatic necrosis followed by Escherichia coli, with anaerobic bacteria being more rarely involved.[2] We present a rare case of acute necrotizing pancreatitis infected with Prevotella species in a young patient.
Case Description/Methods: 28-year-old man with ADHD and hypertension presented with acute epigastric pain radiating to the left. He was found to have a lipase level of 3897U/L, a triglyceride level of 2716mg/dL, in addition to acute interstitial pancreatitis with extensive peri-pancreatic inflammation on CT scan. He was admitted for pain control, IV fluids and insulin. During his stay, he also received plasma exchange for the treatment of hypertriglyceridemia and was discharged upon improvement of symptoms. He represented with abdominal pain and was found to have pancreatic walled-off necrosis with gas concerning for superimposed infection with gastric outlet and colonic obstruction (**Figure**). A percutaneous drain was placed; his course was complicated by sepsis and broad spectrum antibiotics were initiated. Drain fluid cultures grew moderate Staphylococcus aureus and Streptococcus intermedius, heavy Prevotella buccae, Prevotella denticola and Fusobacterium. Hospital course was further complicated by worsening abdominal pain and distention. Repeat CT revealed increasing colonic and small bowel dilatation due to stricture in descending colon. Gastroview enema demonstrated probable fistula tract. He underwent percutaneous endoscopic gastrostomy tube placement and creation of a loop transverse colostomy. Post-operatively, patient was transitioned to oral antibiotics for additional four weeks with outpatient follow-up.
Discussion: Acute necrotizing pancreatitis, especially if infected, is associated with poor outcomes. We highlight a rare case of hypertriglyceridemia-induced pancreatitis complicated by necrotizing infection with heavy Prevotella species. We demonstrate the importance of early suspicion of complicated acute pancreatitis and the need for early intervention to prevent hospital re-admission and improve the morbidity and mortality associated with infected pancreatic necrosis.



[1899] **Figure 1.** A. Initial CT showing necrotizing pancreatitis with large evolving peri-pancreatic walled off necrosis. B. Follow up CT showing large evolving peri-pancreatic walled off necrosis with evidence of new gas locules within the collection.

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S1900

A Rare Case of Extra Pulmonary Tuberculosis Involving the Pancreas and Distal Esophagus

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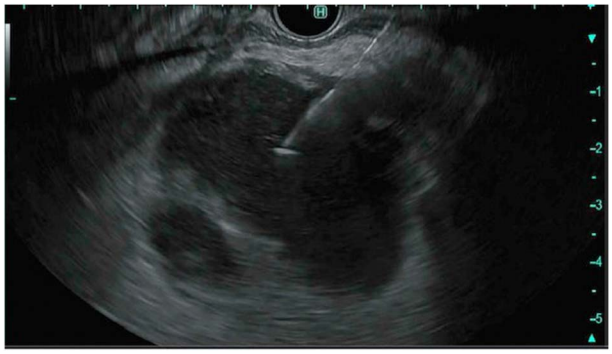
Introduction: We report an unusual case of extra-pulmonary tuberculosis (TB) involving the esophagus and pancreas. The case presented a diagnostic challenge with unrevealing original core biopsy and a battery of infectious, inflammatory, rheumatologic, and procedural testing.
Case Description/Methods: A 54-year-old female with a past medical history of renal transplant and latent TB treated with isoniazid presented from a regional hospital for further evaluation of epigastric pain associated with 15-pound weight loss. On admission, the patient was afebrile, hemodynamically stable, with mild epigastric abdominal tenderness without rebound or guarding. Her labs were significant for mild leukocytosis, normocytic anemia, and hyponatremia. Computerized tomography of the abdomen and pelvis was remarkable for extensive retroperitoneal adenopathy, circumferential esophageal wall thickening, and a peripancreatic mass. At the transferring hospital the patient underwent a core needle biopsy of the retroperitoneal mass with pathology showing granulomatous inflammation with necrosis and no evidence of malignancy. The patient was subsequently transferred to our hospital for further evaluation. After extensive infectious and rheumatologic workup, including bronchoalveolar lavage, the patient underwent endoscopic ultrasound-fine needle aspiration, which showed an irregular 5.6 cm by 5 cm mass in the peripancreatic region with mixed features near the body and tail **(Figure)**. A fine needle biopsy was performed, and 45 milliliters of cloudy, yellow, purulent fluid was sent for pathology and microbiology. Gastric and distal esophageal biopsies were also sent for pathology. The final report was significant for acid-fast bacilli in the esophagus and the pancreas. The fluid analysis of the pancreas with real-time polymerase chain reaction (PCR) was positive for *Mycobacterium tuberculosis*. The patient was subsequently treated with rifabutin, isoniazid, pyrazinamide, and ethambutol.
Discussion: Extra-pulmonary tuberculosis manifesting in the pancreas or esophagus is extraordinarily rare. Per the literature review, there are no reported synchronous pancreatic-esophageal tuberculosis cases. Esophageal tuberculosis mainly presents as an extension of pulmonary disease. Pancreatic tuberculosis generally presents as a pancreatic mass and is often misdiagnosed as a pancreatic adenocarcinoma. This case demonstrates the deceptive nature of tuberculosis and its capability to afflict the gastrointestinal system.



[1900] **Figure 1.** Endoscopic ultrasound-fine needle aspiration of pancreatic mass.

S1901

A Rare Case of Congenital Choledochal Cyst (CCC) Resulting in Recurrent Acute Pancreatitis in an Otherwise Healthy Young Woman

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Introduction: Congenital choledochal cyst (CCC) is a rare cystic dilatation of intrahepatic or extrahepatic biliary ducts. We present a case of a type IVb choledochal cyst presenting as recurrent acute pancreatitis in a young healthy female with initial negative screenings.
Case Description/Methods: An 18-year-old-female with a history of COVID-19 presented to the emergency department with one month of persistent abdominal pain, nausea, and vomiting. She was hospitalized once prior for similar symptoms and was diagnosed with acute pancreatitis. This admission, blood work showed elevated lipase, elevated liver enzymes, mild bilirubinemia with a normal lipid panel and urine was significant for infection. She received fluids, antiemetics and was started on prophylactic antibiotics for ascending cholangitis. A right upper quadrant ultrasound ruled out cholelithiasis or acute cholecystitis, but showed dilation of the common bile duct. MRCP confirmed dilation with bulbous termination in the periampullary region diagnosed as type IVb choledochal cyst.
Discussion: CCCs are rare in Western countries with an incidence between 1 in 100,000 to 150,000. 80% of these cysts are diagnosed in patients under the age of 10. They are difficult to diagnose due to variable clinical presentations. A study of 214 CCC patients demonstrated the most common symptom was abdominal pain, followed by jaundice and fever. When cysts are found in adults, symptoms resemble atypical acute biliary tract disease. Surgical cyst removal may be needed for patients with significant risk factors such as older age and age of symptom onset, due to increased risk of malignant transformation. Longer periods of observation have been documented to be associated with an increased chance of developing late complications, such as anastomotic stricture, biliary calculi and recurrent cholangitis. Type IVb CCCs, as seen in this case, consist of multiple extrahepatic cysts and hepaticojejunostomy is the treatment. This patient's young age and recurrent acute pancreatitis combined with her lab and imaging findings strongly suggest the diagnosis of CCC. The anatomical location of the CCC impeded flow of pancreatic enzymes through the ampulla of vater, leading to recurrent pancreatitis in an otherwise healthy young female. CCC, although very rare, should be considered in the differential of acute pancreatitis when other causes such as gallstones and heavy alcohol consumption cannot be identified, as prompt diagnosis and surgical removal is imperative.

S1902

A Rare Case of Lipomatous Pseudohypertrophy of the Pancreas Diagnosed With EUS-FNA

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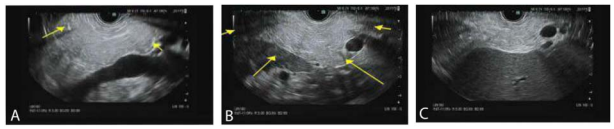
Introduction: Lipomatous pseudohypertrophy (LPH) of the pancreas is a rare benign entity of undetermined pathogenesis characterized by the enlargement of the pancreas due to replacement of exocrine parenchyma with adipose tissue in patients without obesity, diabetes, or pancreatitis. Adipose infiltration may be local or diffuse and forms a pseudotumor that mimics pancreatic malignancies, which has led to unnecessary resections. Endoscopic ultrasound with biopsy (EUS- FNA) has improved the ability to diagnose LPH and rule out malignancy. LPH is typically asymptomatic, however, abdominal pain may be a presenting symptom with steatorrhea due to exocrine insufficiency or jaundice due to mass effect seen in late stages.
Case Description/Methods: 48-year-old female with history of hypothyroidism and migraines presented with epigastric pain, poor oral intake due to postprandial vomiting, and non-bloody mucoid diarrhea. Prior to admission, she had been having chronic GI symptoms and recent MRI demonstrated near complete fatty replacement of an enlarged pancreas causing mass effect with only a small amount of normal parenchyma remaining. She had been diagnosed with exocrine pancreatic deficiency and started on replacement therapy. Surgery was consulted for potential resection of the body and tail of the pancreas due to compressive symptoms. Due to concern for other malignant process including liposarcoma, EUS-FNA was pursued **(Figure)**. The body and tail of the pancreas appeared diffusely hyperechoic and homogeneous, consistent with fatty replacement. Biopsies showed bland pancreatic islets in a background of mature adipose tissue consistent with fatty replacement of the pancreas. These pathology findings in conjunction with pancreatic hypertrophy and compression on surrounding organs without a history of obesity, diabetes, or pancreatitis, supported the diagnosis of LPH of the pancreas. She was ultimately treated conservatively with monitoring and pancreatic enzyme replacement therapy with improvement in symptoms.

Discussion: LPH is a rare, benign disorder typically managed with enzyme therapy when symptoms develop. The mass formed by LPH mimics that of malignant disease which has led to unnecessary resections. The use of EUS-FNA to observe histological features and confirm diagnosis has led to a decrease in resections and differentiation from other causes of pancreatic fat infiltration. The pathogenesis remains unknown but is distinct from pancreatic steatosis commonly seen in metabolic syndromes or inflammation.



[1902] **Figure 1.** EUS showing pancreatic parenchymal abnormalities consisting of diffuse echogenicity with homogenous appearance in the Neck (A), Body (B), and Tail (C) of the pancreas. These findings consistent with fatty replacement of the pancreas. There is no focal mass noted, the pancreatic duct was not visualized.

S1903

A Rare Case of Metastatic Uterine Leiomyosarcoma to the Pancreas

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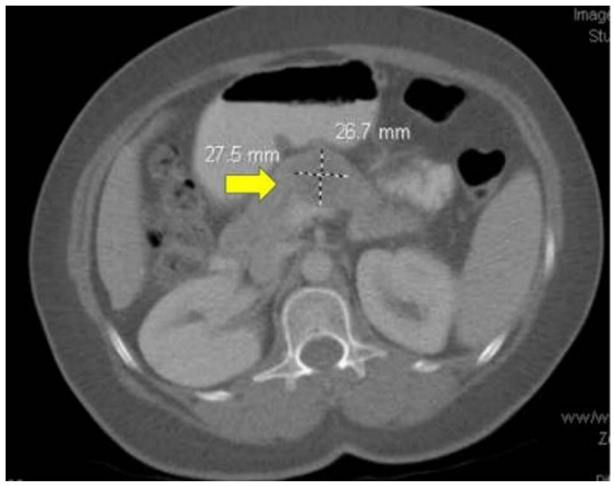
Introduction: Uterine leiomyosarcoma (ULMS) is a rare uterine malignancy accounting for 1.3% of all uterine malignancies and has a high risk of recurrence and death. UMLS is known to have hematogenous metastasis to the lungs, liver, abdomen, and pelvis, but metastasis to the pancreas is far less common.
Case Description/Methods: A 39-year-old female presented to the clinic with urinary frequency and left-sided pelvic pain. She was later diagnosed with a 10 x 8 x 12 cm solid pelvis mass, without signs of metastasis on CT and MRI and underwent a total abdominal hysterectomy with bilateral salpingo-oophorectomy. Pathology confirmed the diagnosis of ULMS without identifiable lymphovascular invasion. Estrogen receptor (ER) and progesterone receptor (PR) were positive 50% and 50%, respectively, with a negative Ki-67 marker. She subsequently received 6 cycles of Docetaxel and gemcitabine followed by pelvic radiation. She remained asymptomatic for 2 years but later had a recurrence with the right lower lung and liver on surveillance CT. Liver biopsy confirmed metastatic ULMS with ER 30% and PR 10%. She subsequently received 7 cycles of Doxorubicin, which showed a response with an interval decrease in mass size. She underwent liver nodule resection and stereotactic body radiation therapy to the right lung lobe. Follow-up surveillance CT chest, abdomen, & pelvis showed resolution of both lesions. However, a repeat scan 5 months later revealed a 2.8 x 2.7 cm hypoenhancing pancreatic body mass which was confirmed to be recurrent ULMS by the endoscopic ultrasound with a fine needle aspiration (EUS-FNA) despite being asymptomatic **(Figure)**. CEA, CA 15-3, chromogranin A, and CA 19-9 were within normal limits. She was started on Doxorubicin and is currently under evaluation for dacarbazine and the PTC596 trial.
Discussion: Pancreatic metastasis is extremely rare, and the most seen primary origins are lung, renal, and gastrointestinal tract malignancies. This case illustrates the aggressive nature of UMLS metastasis and how, pancreatic metastases are asymptomatic and incidentally found during the follow-up. When becoming symptomatic, the presentation such as obstructive jaundice, pain, and weight loss is similar to the primary pancreatic cancer. EUS-FNA biopsy is the key to diagnosing and differentiating metastatic from new primary lesions. Given the rarity, the treatment for metastatic ULMS to the pancreas is situationally determined and non-standardized, generally is still the combination of surgery, hormonal, and chemotherapy.



[1903] **Figure 1.** A 2.8 x 2.7 cm hypoenhancing pancreatic body mass which was confirmed to be recurrent uterine leiomyosarcoma by the endoscopic ultrasound with a fine needle aspiration.

S1904

A Rare Case of Necrotizing Pancreatitis in the Postpartum Period

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Introduction: Postpartum pancreatitis is a rare condition occurring in approximately 3 in 10,000 pregnancies. Progression to the necrotizing form of the disease leads to an increased mortality of up to 25%. High clinical suspicion is warranted in postpartum patients that present with abdominal pain and normal pancreatic enzymes in order to emergently diagnose and treat necrotizing pancreatitis.
Case Description/Methods: A 28 years old G2P2A0 female presented with diffuse abdominal pain, nausea, diarrhea, bilious emesis and poor oral intake two weeks after an uneventful cesarean delivery. Vital signs were remarkable for tachycardia and hypotension. Laboratories revealed leukocytosis, lactatemia and acute kidney injury. Lipid panel, pancreatic enzymes and liver function enzymes were within normal limits. Abdominopelvic computed tomography was remarkable for necrotizing pancreatitis with near complete devitalization of the pancreas and a partial rim enhancement in the lesser sac extending into the pericolic gutter, gastrosplenic recess and superior duodenal fossa. Abdominal ultrasound revealed dilatation of the common biliary duct with no visible intraluminal calculi and no intrahepatic biliary duct dilatation. Magnetic resonance cholangiopancreatography showed severe pancreatic inflammation with common biliary duct compression. Imaging guided percutaneous drainage was successfully performed with significant decrease in abscess size. Patient was placed on pancreatic enzyme replacement and broad spectrum intravenous antibiotics. Hospitalization course was complicated with pancreatogenic diabetes, hypernatremia with osmotic extrapontine myelinolysis, respiratory failure and septic shock. Work-up for causes of pancreatitis such as drug-induced, gallstone, autoimmune, alcohol-induced, infectious, among others, was negative. Follow up imaging showed sterile walled off pancreatic necrosis with marked clinical improvement for which patient was discharged with multidisciplinary care team follow up.
Discussion: This report contributes to a better understanding of an atypical cause of abdominal pain in the subacute postpartum period. Recognition of necrotizing pancreatitis in this population may be easily missed since clinical manifestations of pancreatitis tend to overlap with other diseases such as endometritis, septic pelvic thrombophlebitis, and wound infection. Idiopathic pancreatitis is seen in 10-15% of pancreatitis cases for which emergent clinical recognition and treatment are crucial to ensure survival.

S1905

A Rare Case of Acute Pancreatitis With Normal Lipase

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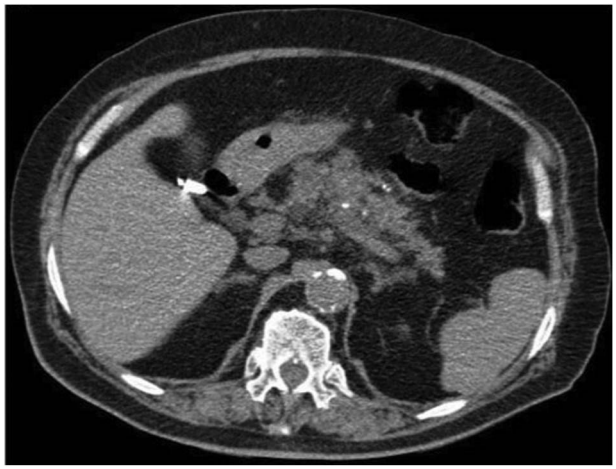
Introduction: Acute pancreatitis (AP) is an inflammatory disease of the pancreas that usually presents with epigastric pain radiating to the back. AP is a critical condition that can lead to multi-organ failure if not diagnosed early. It is diagnosed with 2 of the following: characteristic abdominal pain, imaging consistent with acute pancreatitis, and lipase levels greater than 3 times the upper limit of normal. The negative predictive value of lipase for diagnosing acute pancreatitis is between 94% and 100%. We present a rare case of acute pancreatitis with normal serum lipase levels. Our findings highlight the vital utility of imaging for diagnosing acute pancreatitis in the setting of normal lipase levels.
Case Description/Methods: An 80-year-old female presented with a 1-day history of epigastric pain radiating to her back associated with nausea, vomiting, and loss of appetite. Her past medical history was significant for hypertension, NIDDM II, and atrial fibrillation. She had no recent medication changes and took amlodipine, metformin, metoprolol, and apixaban. Previous surgeries included cholecystectomy and appendectomy. She abstained from alcohol, tobacco, or recreational drug use. She reported abdominal trauma from bicycle collision one week prior to arrival. Vital signs were remarkable for T 101.2 F. She did not appear in acute distress. Her physical exam was remarkable for epigastric tenderness on palpation. Murphy, Cullen, and Grey-Turner signs were absent. Laboratory evaluation revealed WBC 12.8 k/uL, lipase 29 U/L, amylase 52 U/L, AST 13 U/L, ALT 21 U/L, ALP 58 U/L (**Table**). Lipid profile and EtOH levels were within normal limits. CT of the abdomen and pelvis revealed interstitial edematous pancreatitis (**Figure**). She was started on aggressive intravenous fluid resuscitation, metoclopramide for nausea, and intravenous acetaminophen for pain. Her abdominal pain progressively resolved, and she tolerated a full diet by day 3.
Discussion: AP is a critical condition that requires early diagnosis. The negative predictive value of elevated lipase levels for diagnosing AP ranges from 94% to 100%. Our patient was diagnosed with acute pancreatitis due to trauma in the setting of normal lipase levels. We demonstrate the utility of imaging in diagnosing acute pancreatitis in the setting of normal serum lipase levels.



[1905] **Figure 1.** CT scan of the abdomen revealed peripancreatic fat stranding and interstitial edema consistent with findings of pancreatitis.

Table 1. Laboratory trends are displayed here. Lipase remained within normal limits through the 4 days measured.

	Admission	Day 1	Day 2	Day 3	Day 4	Normal Range
Hemoglobin	8.8	7.8	9.3	8.6	9.0	12.0 - 15.0 g/dL
WBC	12.8	7.4	5.9	5.7	6.2	3.4 - 11.0 10*3/uL
Amylase	52	39				29 - 103 U/L
Lipase	29	12				13 - 60 U/L
Calcium	8.9	8.1	8.9	8.8	8.4	8.4 - 10.2 mg/dL
Alkaline Phosphatase	58	44	46	59	54	40 - 129 U/L
ALT	21	16	29	17	11	10 - 50 U/L
AST	13	10	14	12	16	13 - 39 U/L

S1906

A Rare Cause of Delayed Life-Threatening Hemorrhage in a Patient With Pancreas Transplant

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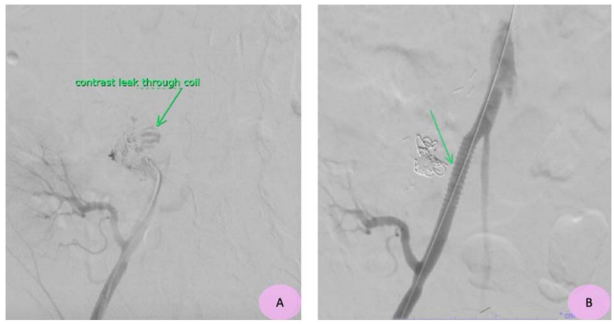
Introduction: Complications of vascular anastomosis usually occur early after pancreas transplant (PTx) and carry a substantial risk of graft loss and mortality. Hemorrhage may result from arterial anastomosis failure, but more commonly from superior mesenteric or splenic vessel ligation. We present a challenging case of GI hemorrhage from a pseudoaneurysm (PA) in the implanted vascular Y graft 7 years after PTx.
Case Description/Methods: A 45-year-old male 7 years post simultaneous pancreas-kidney transplant presented with painless rectal bleeding. EGD and Colonoscopy showed blood throughout the duodenum and colon, but the source could not be localized. Push enteroscopy showed blood extending into the proximal jejunum, and no active bleeding source could be identified. CT angiogram showed active extravasation into a small bowel loop near the PTx. Mesenteric angiography with IR revealed active extravasation into jejunum from a PA which had developed off the SMA Y graft anastomosis, eroding into the adjacent duodenal stump. Successful coil embolization was performed. 2 days later, he developed rectal bleeding requiring massive transfusion. Tagged RBC scan and CT angiography could not localize the bleeding source. Emergent mesenteric angiography showed contrast leakage into the anastomotic stump beyond the previously placed coil (**Figure** A). A covered stent was placed into the right external iliac artery arresting the bleeding into the SMA anastomotic stump (**Figure** B). Bleeding and shock resolved, and he was discharged with transplant surgery follow-up.
Discussion: Pseudoaneurysms, AV fistulae, arterial dissection and stenosis represent rare vascular complications of PTx. PAs, if early, may be the result of surgical technique, infection, severe pancreatitis, or allograft biopsy. Arterial anastomotic PAs can be asymptomatic or present with symptoms signaling impending rupture such as abdominal pain, pulsatile mass, or endocrine failure. Endoscopic localization and management of bleeding from Y graft PAs are challenging due to altered anatomy and brisk bleeding. Multidisciplinary management is essential and often involves endovascular hemostasis with IR, possibly with surgical exploration. Stent placement is usually preferred over embolization due to the ability to deploy with precision while maintaining end-organ perfusion. Although guidelines are lacking, it would be reasonable to consider periodic Doppler US to screen for post-operative PAs for early detection and preventative endovascular treatment.



[1906] **Figure 1.** A. Pre-stent mesenteric angiogram with contrast leaking through coil into SMA anastomotic stump; B. Post stent mesenteric angiogram with no contrast leaking into the SMA anastomotic stump.

S1907

A Rare Case of Type 1 Choledochal Cyst With Type C Anomalous Pancreaticobiliary Maljunction in a 52-Year-Old Patient

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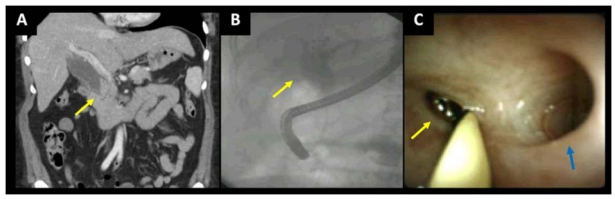
Introduction: Choledochal cysts are rare anomalies of the bile duct that generally present in childhood but can rarely be delayed until adulthood in 20% of cases. They are often diagnosed incidentally or present with acute pancreatitis or cholangitis. We report a case of a type 1 choledochal cyst (T1CC) and type C anomalous pancreaticobiliary maljunction (PBM), successfully managed by a multidisciplinary approach.
Case Description/Methods: A 52-year-old woman presented with right upper quadrant pain, jaundice and vomiting. Labs revealed elevated lipase, bilirubin, AST, ALT and alkaline phosphatase. A CT of the abdomen showed no gallstones but a marked dilation of extrahepatic bile duct with a transition point in the distal intrapancreatic portion (**Figure** a). An endoscopic retrograde cholangiopancreaticogram (ERCP) showed fusiform dilation of the CBD consistent with T1CC (**Figure** b). Spyglass cholangioscopy revealed focal stenosis in the distal CBD joining above the ventral pancreatic duct opening within the long common channel, suggestive of type C anomalous PBM (**Figure** c). A plastic biliary stent was placed into the CBD. EUS guided fine needle biopsy of the stricture showed no evidence of malignancy. Her clinical course was complicated by multiple admissions due to stent occlusion. After multidisciplinary discussion, the patient underwent choledochal cyst resection with hepaticojejunostomy, cholecystectomy and biliary reconstruction. Intraoperative gallbladder specimen revealed poorly differentiated adenocarcinoma with plan for chemotherapy.
Discussion: Choledochal cysts are a rare congenital anomaly characterized by enlargement of the CBD. There are five types of choledochal cysts (**Table**). Etiology remains unclear and while some believe these cysts are congenital in nature, others propose they arise from anomalous PBM. PBM is defined as congenital malformation in which the pancreatic and bile duct join outside the duodenal wall forming a long common channel. This leads to reflux of biliary secretions leading to chronic inflammation, increasing the risk of developing cholangiocarcinoma and gallbladder cancer, as seen in our patient. Placement of biliary stents can temporarily alleviate symptoms, but surgical excision is the definitive treatment. Our case highlights the importance of including type 1 choledochal cyst in the differential diagnosis even in adult patients with a significantly dilated CBD. A thorough endoscopic and radiological work up should be done to prevent progression to malignancy.



[1907] **Figure 1.** A. CT of the abdomen showing marked dilatation of extrahepatic bile duct with a transition point in the distal intrapancreatic portion. B. Cholangiogram showing Type 1 Choledochal Cyst and Type C Anomalous Pancreaticobiliary Maljunction. C. Spyglass cholangioscopy showing JagwireTM within the CBD (yellow arrow) revealing focal stenotic CBD joining the common channel just above the ventral pancreatic duct (blue arrow).

Table 1. Types of Choledochal Cysts

Type	Description
Type I	Fusiform dilation of the CBD
Type II	Bile duct diverticulum
Type III	Saccular dilation of the intraduodenal portion of the CBD
Type IVa	Multiple cysts at intra and extrahepatic ducts
Type IVb	Multiple cysts at extrahepatic ducts only
Type V	Fusiform or saccular cystic dilation of the intrahepatic bile duct

S1908

A Unique Case of Endoscopic Retrograde Cholangiopancreatography Associated *Eikenella corrodens* Liver Abscess

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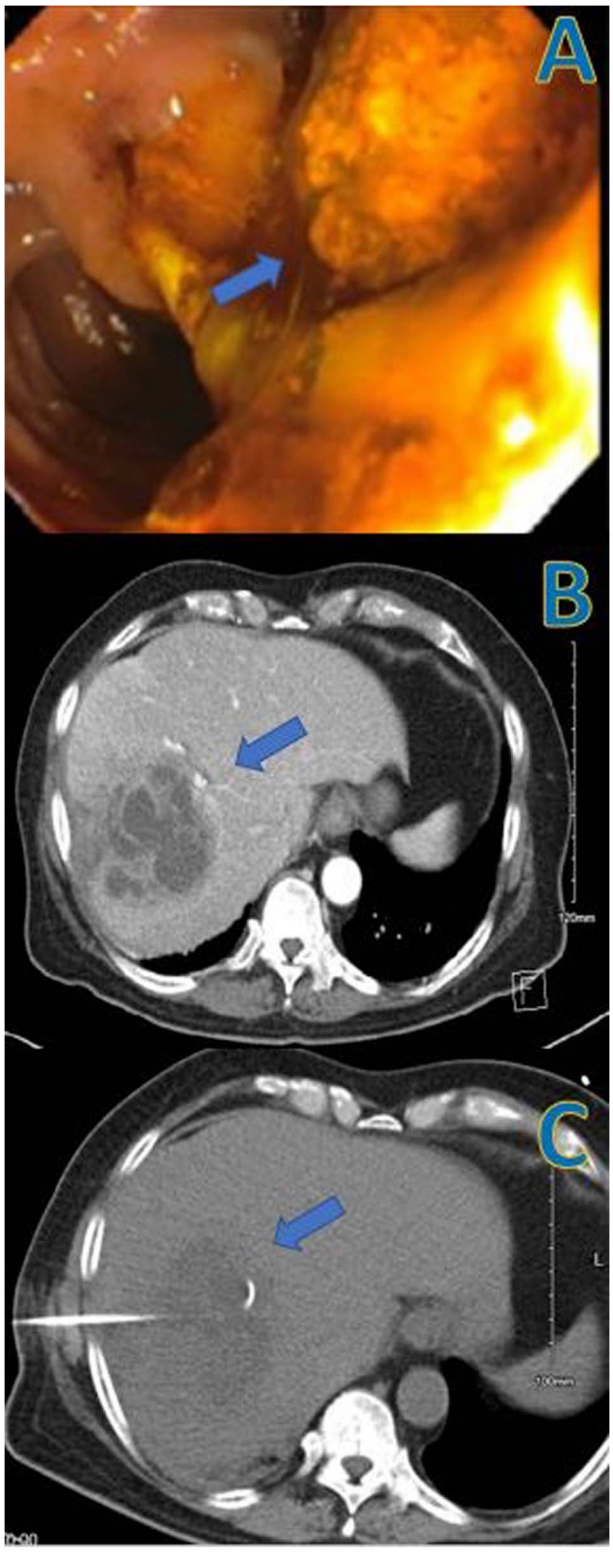
Introduction: The most common complications of endoscopic retrograde cholangiopancreatography (ERCP) are pancreatitis, hemorrhage, cholangitis, and duodenal perforation. Rarely, septic complications of ERCP involve hepatic abscess, with the most common organisms being E. coli and P. aeruginosa. We present a unique case of ERCP related Eikenella liver abscess.
Case Description/Methods: 68-year-old male with a history of cholelithiasis and choledocholithiasis who underwent an ERCP with sphincterotomy, CBD stent placement and cholecystectomy 3 months ago, and poor dentition, presented with abdominal pain and fever. 1 month prior, he had an ERCP with common bile duct (CBD) stent removal and balloon sweep removal of a large amount of sludge and small stones. Since the procedure, he reported fatigue with 1 week of worsening right sided abdominal pain, nausea, and fever. He was found to be tachycardic and febrile to 102°F. Labs showed leukocytosis, transaminitis, and bilirubin of 2.6 mg/dL. An abdominal CT scan showed a rim enhancing, multiloculated lesion in the liver and CBD dilation and wall enhancement. He had a repeat ERCP for suspected cholangitis which again showed thick sludge and small stones on balloon sweep. He underwent placement of a 12F percutaneous catheter to drain the liver abscess whose cultures grew Eikenella Corrodens. His endocarditis workup was negative, and he completed a course of ceftriaxone for his infection with repeat CT showing improvement of the abscess (**Figure**).

Discussion: The literature is limited in cases of post ERCP hepatic abscess complications. Generally, bacteria can enter the biliary tract by a hematogenous or retrograde route which are normally protected by anatomical barriers. Obstructed and immunocompromised patients can be more susceptible to complications as they have impaired bacterial defenses. In this case, the patient was found to have E. corrodens, a common oral flora organism. Infectious work up was otherwise negative, including an endocarditis work up. It has been suspected that contamination from the mouth flora, given his poor dentition, ultimately led to device related introduction into the biliary tree and subsequent development of this rare ERCP related abscess. Adhering to endoscopic equipment disinfection protocols and proper ERCP technique for duct drainage is of utmost importance for minimizing the risk of infection. This case also highlights the justification for recent increase in the use of disposable duodenoscopes for ERCP to decrease infectious complications.



[1908] **Figure 1.** A: large stone and sludge being removed during the patient's ERCP procedure prior to developing the abscess. B: CT finding of a liver abscess on presentation. C: CT showing improvement of the abscess post drainage from a percutaneous catheter.

S1909

A Treacherous Pain: IgG4-Related Disease Relapsing as Sclerosing Cholangitis

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Introduction: Immunoglobulin G4-related disease (IgG4-RD) is an immune-mediated fibroinflammatory condition resulting in tumor-like masses or organ enlargement from infiltration of IgG4-positive plasma cells. Elevated IgG4 serum levels can suggest disease, but tissue biopsy is gold standard for diagnosis. Systemic steroids are 1st line of treatment and are effective in inducing remission, but recurrent/refractory cases are common. IgG4-sclerosing cholangitis (IgG4-SC) involves intrahepatic and extrahepatic bile ducts, is more common in men, and can present with obstructive jaundice. Here we report a patient with prior history of IgG4-Ophthalmic-related disease who presented with a relapsing disease as IgG4-SC after steroid discontinuation.
Case Description/Methods: A 59-year-old male with history of retroocular follicular hyperplasia secondary to IgG-4 related disease, diagnosed by tissue biopsy and on long term steroid therapy until 3 months prior who visited the ER complaining of right upper quadrant abdominal pain of 5 days in evolution, associated with acholia, choluria, nauseas and vomiting. He denied new medications or supplements, use of NSAIDs, or travel. Physical exam was remarkable for palpable submandibular nodules and epigastric tenderness upon palpation. Liver chemistries were remarkable for an AST of 830, ALT of 380, total bilirubin of 3.3, and IgG4 serum levels at 1066 mg/dL. An abdominal US showed biliary tree dilatation with CBD >10 mm. An MRCP showed a dilated biliary tree, mucosal wall thickening at proximal to mid extrahepatic CBD with luminal narrowing, consistent with IgG4 related cholangitis. No choledocholithiasis evidenced. The patient was started on prednisone 40 mg daily during hospitalization. He was discharged home three days later to continue steroid therapy. A follow-up MRCP at one month showed a normal caliber common bile duct and interval resolution of mucosal thickening as well of the stenotic and dilated segments. Liver chemistries also normalized.
Discussion: Patients with IgG4-RD respond well to steroids, but relapse is common while tapering or after their withdrawal. Studies show that 50% of IgG4-RD patients present with new organ involvement on relapse. This case emphasizes the need to be mindful of IgG4-RD relapse to new organ involvement even if the primary site is controlled. An elevated serum IgG4 may aid in the rapid recognition of recurrent disease and prompt initiation of steroids can reduce organ inflammation, preserve function, and limit multiorgan involvement.

S1910

A Rare Metastatic Lesion in the Pancreas Presenting With Biliary Obstruction

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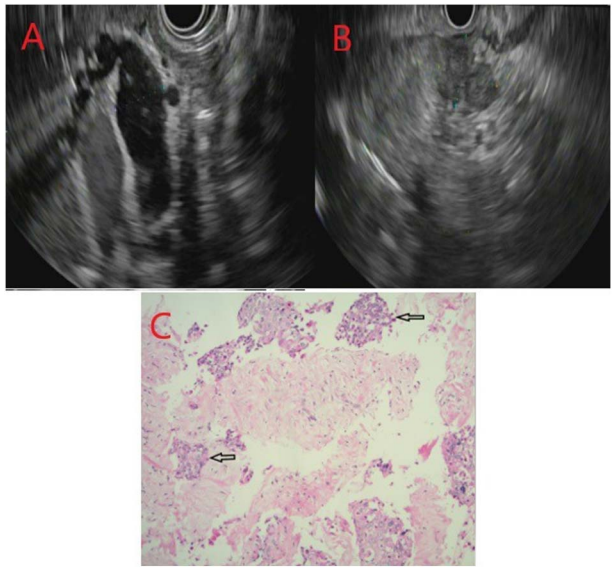
Introduction: Pancreatic malignancies can originate from either endocrine or non-endocrine cells of the pancreas. Ductal carcinoma is the most common type of non-endocrine tumor, of which adenocarcinomas constitute the most common pancreatic malignancy. Squamous cell carcinoma (SCC) is the other ductal tumor that is very rare as the pancreas is generally devoid of squamous cells. Primary SCC of the pancreas is thought to arise from the squamous metaplasia of the ductules secondary to chronic inflammatory conditions, however, metastasis remains the most common etiology. We present a patient with history of recurrent poorly differentiated SCC of the cervix complicated by pancreatic head metastasis and biliary obstruction.
Case Description/Methods: A 46-year-old female diagnosed with poorly differentiated SCC of the cervix stage IIB, status post multiple rounds of chemotherapy over a period of 4 years, was started on a new medication – Tisotumab. Four months following the initiation of this medication, patient experienced abdominal pain and was noted to have a lipase level of 1160 U/L. She was diagnosed with acute pancreatitis and treated accordingly. Subsequent labs showed continued up trending of her liver chemistry, withalkaline phosphatase of 520 U/L and total bilirubin of 9.7 mg/dl. A CT abdomen performed revealed dilation of both common bile duct (CBD) and pancreatic duct (PD) with subtle hypodense area in the head of pancreas. Endoscopic ultrasound (EUS) was performed which revealed a 3 cm x 2 cm ill-defined hypoechoic and heterogenous mass in the pancreas head (**Figure**). Fine needle biopsy of the lesion revealed SCC, most likely metastatic from the cervix. ERCP was performed and a 10mm x 8cm fully covered metal CBD stent was placed with normalization of liver enzymes. Palliative immunotherapy with Keytruda is currently being pursued.
Discussion: Metastasis to the pancreas is an uncommon clinical condition and accounts for 0.5-5% of all pancreatic malignancies. Because of the rarity, primary sources should always be excluded. Only a handful of reports of uterine and cervical cancer metastasis to pancreas have been reported, most of them being adenocarcinoma. We report a very rare case of SCC cervix metastasizing to the pancreas. Unfortunately, curative surgery is not an option with metastatic disease, and chemoradiation can be used palliatively. The median survival is generally poor with median rates of 4.8% and 1% at 1-yr and 5-yrs, respectively.



[1910] **Figure 1.** A - EUS image showing dilated CBD measuring up to x mm. B – EUS image demonstrating a 3 x 2 cm hypoechoic pancreatic head mass. C– H&E stain showing neoplastic clusters (black arrows) of metastatic SCC.

S1911

A Rare Presentation of Metastatic Melanoma With Obstructive Jaundice and Pancreatitis Due to Pancreatic Metastasis With Invasion into the Common Bile Duct

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Introduction: Malignant melanoma is a highly aggressive cancer with high rates of metastasis. Here we present a case of metastatic melanoma of the pancreas which invaded into the common bile duct leading to a unique presentation of obstructive jaundice and pancreatitis.

Case Description/Methods: An 84-year-old male presented with epigastric abdominal pain. 3-years prior he had wide local excision for cutaneous malignant melanoma of the upper back (R0 resection). His abdominal pain had worsened over the last 4-weeks and was preceded by jaundice and unintentional weight loss. Initial studies were significant for elevated alkaline phosphatase (1295), AST (270) ALT (212), total bilirubin (10.1) and lipase (12085). Computed tomography (CT) showed a 2.4 cm lesion in the pancreatic head along with dilated pancreatic duct and biliary dilation. Carbohydrate antigen 19-9 (CA19-9) was elevated (628). Cholangiogram showed a 15 mm distal biliary stricture with upstream dilation. Brushings of the biliary stricture were unsatisfactory due to insufficient cells. Subsequent endoscopic ultrasound (EUS) demonstrated a 25mm by 25mm mass in the pancreatic head invading the distal CBD. Fine-needle aspiration (FNA) showed a poorly differentiated malignancy with epithelioid and focal spindle cell features with positive S100 and SOX-10 immunophenotype. Findings consistent with melanoma. Additional imaging did not reveal other potential sites of metastasis. The patient has since been discharged and is undergoing further evaluation.
Discussion: Approximately one-third of patients with malignant melanoma develop metastases. However, metastatic melanoma of the gastrointestinal system is only seen in 2-4% of patients affected by cutaneous melanoma. This patient had a unique presentation with invasion of his pancreatic lesion into the distal common bile duct which lead to obstructive jaundice and pancreatitis. Pancreatitis as a result of metastatic melanoma is a rare presentation only described in a few case reports. Based on imaging it appears the patient initially had isolated metastatic disease of the pancreas which then invaded into the common bile duct. Isolated pancreatic metastasis represents less than 1% of all metastatic melanomas. It is difficult to differentiate a primary cancer from metastatic disease of the pancreas based on imaging alone. The use of EUS-FNA is essential for diagnosis. Though the exact prognosis of metastatic melanoma of the pancreas is unclear, generally metastatic melanoma carries a poor prognosis.

S1912

A Rare Case of Recurrent Hemosuccus Pancreaticus

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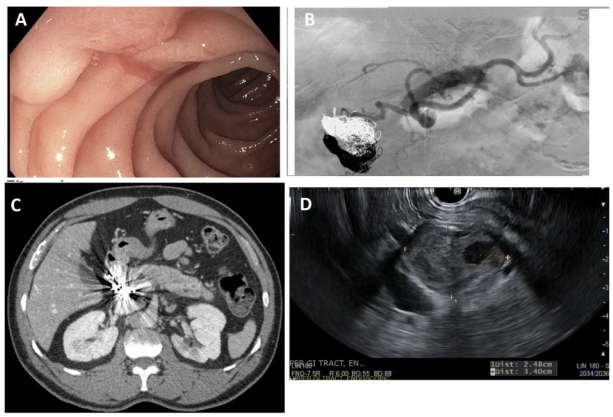
Introduction: Hemosuccus pancreaticus is a rare cause of GI bleeds characterized by intermittent bleeding from the major duodenal papilla. Chronic pancreatitis, via arterial wall necrosis, can form a visceral pseudoaneurysm in 10% of cases. Here is a rare case of recurrent hemosuccus pancreaticus caused by a gastroduodenal artery (GDA) pseudoaneurysm, despite repeated embolization.
Case Description/Methods: A 65-year-old man with alcoholic pancreatitis and pancreaticoduodenal pseudoaneurysm status post embolization in 2019 was being evaluated for hematochezia for 6 months. The patient underwent several negative EGDs and colonoscopies. Video capsule endoscopy showed scant blood streaking in proximal small bowel. Initial CT showed an enlarged pancreas with dilated pancreatic duct. EUS showed a 3x2.5cm well-defined cystic lesion at the pancreatic head with an anechoic center with vascular flow. Repeat CT showed a 1.6cm saccular aneurysmal dilation of the superior pancreaticoduodenal artery (SPDA). Prior to follow-up, he was admitted with hematochezia, with his hemoglobin down from 12 to 7.8. EGD showed blood in the second part of duodenum with fresh oozing from ampulla. An aortogram showed a non-bleeding pseudoaneurysm of the GDA, which was successfully embolized. One month later, he presented again with hematochezia. CTA showed a recurrent 1.5cm pseudoaneurysmal dilation of the SPDA and possible lower GI bleed. Colonoscopy showed dark red blood mixed with stool. NM scan showed a slow bleed from left common/internal iliac artery. An angiogram showed extravasation from the GDA, which was embolized along with the recurrent pseudoaneurysm. Seen 45 days after discharge, the patient was asymptomatic (**Figure**).
Discussion: Hemosuccus pancreaticus caused by GDA pseudoaneurysm is reported in less than 2% of population. Successful embolization in the first 6 months is estimated to be 67%, with rebleeding risk at 37%. Rebleeding is thought to be due to collateral vessels, but in this case, no extravasation was seen after each embolization. Our patient developed recurrent symptoms with development of an aneurysm involving the same artery 1 month later. Recurrence or formation of new pseudoaneurysms usually occur within the first 6 months after embolization. This case is unique in that the patient initially had a successful embolization in 2019 of an aneurysm involving the same artery without any bleeding for 2 years, showing that hemosuccus pancreaticus can recur even after the initial 6 month period after a successful embolization.



[1912] **Figure 1.** A: EGD showing bleeding from ampulla. B: Angiogram of celiac trunk with GDA pseudoaneurysm. C: CTA showed 1.5cm saccular pseudoaneurysmal dilatation of the superior pancreaticoduodenal artery. D: EUS showed a 3.4 x 2.5 cm cystic lesion with anechoic center.

S1913

A Rare Case of Spindle Cell Carcinoma of the Hepatobiliary System

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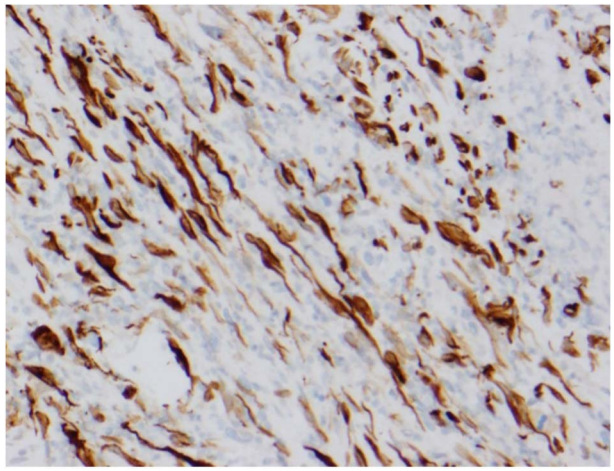
Introduction: Spindle cell also known as undifferentiated carcinomas of the pancreas (UCP) are rare anaplastic deviations of ductal adenocarcinomas of the pancreas. The general nature of these tumors is associated with destructive biological behavior and usually have a poor prognosis compared to most common variants of ductal adenocarcinoma. UCP include spindle cell carcinoma, sarcomatoid carcinoma and carcinosarcoma. In practice, it is hard to differentiate these subtypes since most of them possess a spindle element. UCPs are extremely rare tumors with a frequency of less than 2% of pancreatic exocrine tumors. Furthermore, UCP with focal osteochondroid differentiation are even rarer.
Case Description/Methods: A 50-year-old man with past medical history of cardiovascular disease, hypertension, hepatitis C & external hemorrhoids presented with worsening abdominal pain, nausea, vomiting. CT was notable for evidence of a hepatobiliary mass. Gastroenterology was consulted and performed an EGD. Upon initial evaluation of the stomach into the antrum, a large, perforated ulcer was identified with omental tissue. Additionally, liver parenchyma was visualized along with severe LA Grade D Esophagitis throughout the entire esophagus. Interventional radiology was consulted and subsequently performed a biopsy of the mass in which the pathological findings were suggestive of Spindle cell carcinoma/carcinosarcoma. CT chest was ordered for staging and showed several nodules of unclear origin. Oncology was consulted and patient was initiated on chemotherapy (Carboplatin/Gemcitabine). Once on chemotherapy, the patient grew reluctant about receiving medical management. Palliative care was consulted. Considering advanced stage spindle cell hepatopancreatic biliary carcinoma leading to multiple complications including duodenal perforation, peritonitis, sepsis, encephalopathy leading to terminal delirium, patient's prognosis was deemed very poor with less than 6 months requiring hospice care. Patient was then transferred to inpatient hospice service **(Figure)**.
Discussion: The reported prognosis of each of the histologic subtypes of UCP have overall poor prognosis. Surgery is the first choice for a resectable undifferentiated carcinoma. In a previous study it was identified that average post-operative survival interval was 6 months and the longest living patient survived for 15 months.



[1913] **Figure 1.** H&E immunochemistry stain of liver biopsy.

S1914

A Rare Incidence of Pancreatic Schwannoma

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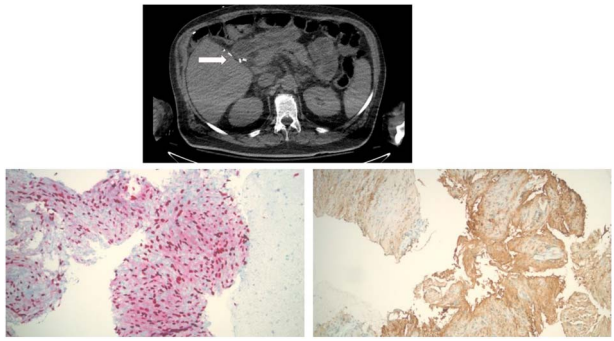
Introduction: Schwannomas are benign, slow-growing tumors that originate from Schwann cells. About 90% of schwannomas are sporadic, while the remaining 10% are associated with genetic disorders such as neurofibromatosis type 2. The majority of schwannomas occur in the head, neck, and distal extremities. Pancreatic schwannomas are exceptionally rare, with less than 70 reported cases in the literature in the past 30 years.
Case Description/Methods: A 64-year-old male with a history of intra-abdominal fibrosarcoma presented with acute encephalopathy from amphetamine use which resolved with supportive care. Patient was previously told that the fibrosarcoma was unresectable. He reported completing chemotherapy in 1991 and did not receive further treatment. Previous medical records were unavailable. A computerized tomography (CT) imaging of the abdomen revealed a 6.5x6.8x9.3 cm mass along the head of the pancreas with peripheral calcifications. Bilateral enlargement of inguinal, iliac, and retroperitoneal lymph nodes was also noted. Endoscopic ultrasound demonstrated a large heterogeneous and hypoechoic pancreatic head mass with septations and calcifications. Specimens attained with fine needle aspiration revealed numerous aggregates of spindle-shaped cells without tumor cell necrosis or mitotic figures. Immunohistochemical stains were positive for S100 and SOX-10, suggestive of a peripheral nerve sheath tumor; pankeratin staining was weakly positive. Immunostains such as smooth muscle actin, CD117, CD34, desmin, DOG-1, HMB-45, and Melan A were negative which excluded sarcoma, gastrointestinal stromal tumor (GIST), and melanoma. A diagnosis of pancreatic head schwannoma was made. Biopsy of the right inguinal lymph node was unremarkable for neoplasm. Due to the schwannoma encasing the superior mesenteric artery and vein, the patient was not a surgical candidate and was discharged for outpatient monitoring (**Figure**).
Discussion: Pancreatic schwannomas are an extremely rare clinical entity. Patients may display non-specific symptoms or remain asymptomatic. CT imaging commonly shows encapsulated, heterogeneous, and hypodense lesions as seen in our patient. S100 immunostaining is crucial for confirming the diagnosis. A specimen positive for S100 but negative for CD34, DOG-1, and HMB-45 as above is subtyped as a pancreatic schwannoma with ancient features. Conservative management is recommended; symptomatic patients may benefit from surgical resection.



[1914] **Figure 1.** Top: Computerized tomography of the abdominal cavity revealed a 6.5x6.8x9.3 cm mass at pancreatic head with inferior calcifications (white arrow) Bottom, Left: Biopsy of the pancreatic head revealed aggregates of spindle-shaped cells that stained positive for SOX10. Bottom, Right: Biopsy of the pancreatic head revealed aggregates of spindle-shaped cells that stained positive for S100.

S1915

A Rare Case of Pseudohyponatremia in an Elderly Male With Pancreatic Adenocarcinoma: Laboratory Artifact or Idiopathic?

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Introduction: Electrolyte imbalance in patients with obstructive jaundice is rare. Case reports of pseudohyponatremia in patients with obstructive jaundice have been previously described by Sivakumar et. al and Adashek et. al. They described the artifactual hyponatremia in the setting of hyperlipidemia and hyper lipoproteinemia. We describe a case of pseudohyponatremia with obstructive jaundice with modest elevation in total cholesterol and LDL
Case Description/Methods: A 73-year-old Asian patient with a history of type 2 diabetes mellitus presented with a 4 week history of generalized malaise, 2 week history of yellowish discoloration of both eyes, nightly upper abdominal pain, nausea and 1 episode of vomiting. He reported 10 kg weight loss in the last 6 weeks. He was noted to have hyponatremia and elevated liver enzymes by his primary doctor and was sent to the ER. His serum sodium on admission was 117. Corrected sodium to hyperglycemia was 122. Measured serum osmolality was 261 and calculated was 275. He was treated with NS 1L over 10 hours and

AM lipid panel was obtained (**Table**). A CT of the abdomen and pelvis showed a distended gallbladder with dilatation of the intrahepatic and common biliary ducts with the CBD measuring up to 11 mm near the head of the pancreas. The serum sodium did not improve after IV fluids which were stopped as he looked clinically euvolemic. AM lipid was obtained (**Figure**). ERCP revealed a 2cm biliary/pancreatic stricture compatible with pancreatic cancer and a 7 F stent was placed. CA 19-9 was 149.6. His serum sodium improved spontaneously without intervention. At discharge, his corrected serum sodium improved to 132. A EUS guided biopsy confirmed a pancreatic adenocarcinoma.

Discussion: Unmeasured proteins and/or lipids seen in intra-and extra hepatic cholestasis can falsely result in a low serum sodium. These spurious anomalies may impede diagnosis and initial management. A high degree of caution should be exercised when met with conflicting clinical and laboratory abnormalities. Clinicians may exercise inappropriate choice of fluids especially when met with those suffering from pancreatitis. This may further lead to complications of rapid correction of sodium and hypernatremia. A high clinical suspicion should be exercised when met with severe asymptomatic hyponatremia. Sodium should be monitored after relieving the obstruction. Measurement of serum lipids and evaluating for paraproteinemia can be helpful.

Lipid profile	Ref. Range	5/24/22 12:25
Cholesterol	Latest Ref Range: 0 - 200 mg/dL	349
Triglycerides	Latest Ref Range: 0 - 150 mg/dL	180
HDL	Latest Ref Range: 35 - 55 mg/dL	13
Non-HDL Cholesterol	Latest Units: mg/dL	336
Chol/HDL Ratio	Latest Ref Range: 2.00 - 5.50	26.8
VLDL	Latest Ref Range: 2.0 - 50.0 mg/dL	36
LDL Calculated	Latest Ref Range: <130 mg/dL	300

[1915] **Figure 1.** Lipid profile.

Table 1. Chemistry panel

Chem profile	Ref. Range	5/18/22 8:53	5/24/22 12:25	5/25/22 4:05	5/25/22 18:33	5/26/22 0:58	5/26/22 6:55	5/26/22 17:18	5/27/22 6:06	6/2/22 8:10
Total Protein	Latest Ref Range: 6.4 - 8.9 g/dL	7.7	6.6	6			6.8		6.8	7.2
Glucose	Latest Ref Range: 70 - 99 mg/dL	268	411	162	232	265	217	246	193	149
BUN	Latest Ref Range: 7 - 25 mg/dL	18	13	8	8	8	8	7	5	18
CREATININE	Latest Ref Range: 0.6 - 1.3 mg/dL	1.02	0.96	0.88	0.86	0.87	0.86	0.68	0.7	0.85
Sodium	Latest Ref Range: 133 - 144 mmol/L	124	117	119	118	119	121	121	125	128
Potassium	Latest Ref Range: 3.5 - 5.2 mmol/L	4.8	4.2	3.7	4.1	3.9	3.4	4.4	3.9	4.5
Chloride	Latest Ref Range: 98 - 107 mmol/L	88	81	87	86	89	89	91	92	93
CO2	Latest Ref Range: 21 - 31 mmol/L	27	23	25	20	23	23	21	24	27
Anion Gap	Latest Ref Range: 6 - 14 mmol/L	9	13	7	12	7	9	9	9	8
BUN/Creatinine Ratio	Latest Ref Range: 6.0 - 20.0	17.6	13.5	9.1	9.3	9.2	9.3	10.3	7.1	21.2
Calcium	Latest Ref Range: 8.6 - 10.3 mg/dL	9.7	9.1	8.6	8.7	8.4	8.6	8.4	8.7	9.3
Phosphorus	Latest Ref Range: 2.5 - 4.5 mg/dL						2.6			
Albumin	Latest Ref Range: 3.5 - 5.7 g/dL	4.2	3.8	3.4			3.7		3.8	3.8
AST	Latest Ref Range: 13 - 39 IU/L	150	168	167			88		73	104
ALT	Latest Ref Range: 7 - 52 IU/L	614	448	401			346		273	385
Alkaline Phosphatase	Latest Ref Range: 40 - 129 IU/L	363	589	480			514		424	524
Total Bilirubin	Latest Ref Range: 0.0 - 1.0 mg/dL	15.8	27.2	25.4			14.9		10.5	5.7

S1916

A Rare Case of Small Cell Cholangiocarcinoma

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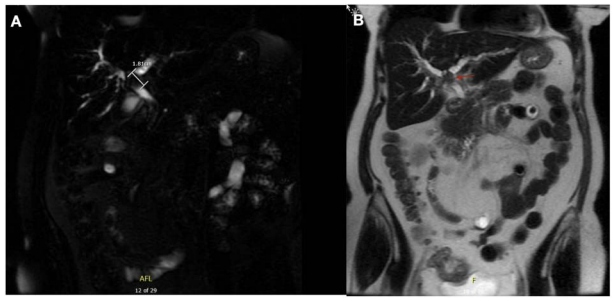
Introduction: Small cell cholangiocarcinoma is a rare entity and is encountered much less frequently than most other forms of cholangiocarcinoma. This case describes a 63-year-old female who presented with abdominal pain, fatigue, and nausea and was found to have a 1.4 cm mass at the confluence of the left and right intrahepatic bile ducts. This was deemed to be unresectable small cell cholangiocarcinoma, and she was ultimately provided palliative chemotherapy.

Case Description/Methods: A 63-year-old female with a history of chronic obstructive pulmonary disease presented to the hospital with right upper quadrant pain, fatigue, nausea, and bloating. Laboratory studies revealed an alkaline phosphatase (ALP) of 917 U/L, total bilirubin of 3.2 mg/dL, alanine aminotransferase (ALT) of 552 U/L, aspartate aminotransferase (AST) of 285 U/L, and a normal lipase. A magnetic resonance cholangiopancreatography showed a 1.4 cm mass at the confluence of the left and right intrahepatic bile ducts leading to biliary obstruction. Endoscopic retrograde cholangiopancreatography demonstrated a stricture of the common bile duct for which a stent was unable to be deployed. The patient underwent biliary decompression via percutaneous drain placement. A biopsy was obtained during an attempt at surgical resection, but the mass was unable to be removed due to the involvement of the main portal vein. The AFP level returned at 7 ng/mL, CA 19-9 was 6 U/mL, and CEA was 1.6 ng/mL. Biopsies showed positivity for CAM 5.2, keratin AE1/AE3, CD56, synaptophysin, and TTF1. Ki67 index was >95%. Chromogranin was negative. The patient was treated with etoposide, atezolizumab, and carboplatin, however, the disease course was complicated by refractory disease and brain metastasis (**Figure**).

Discussion: Small cell cholangiocarcinoma is a much less prevalent form than other cholangiocarcinomas. Symptoms and signs typically mimic the more common variants of cholangiocarcinoma and often include right upper quadrant discomfort, nausea, dyspepsia, jaundice, fatigue, and weight loss. Laboratory findings include elevations in ALP and bilirubin. ALT and AST may also be elevated, especially when the tumor is intrahepatic. Treatment depends on if the tumor is amenable to surgical resection. If it is unresectable, treatment with cisplatin/etoposide +/- immunotherapy is often used. It is the hope of this case to bring attention to this rare diagnosis and prompt further research into therapies for this malignancy.



[1916] **Figure 1.** MRCP showing an intrahepatic mass at the confluence of the right and left biliary ducts.

S1917

A Rare Case of Paraesophageal Hernia Repair Complicated by Pancreatic Injury

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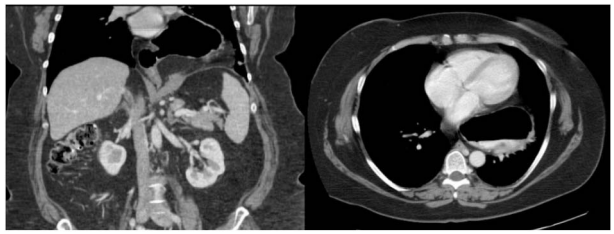
Introduction: Hiatal hernias (HH) affect anywhere from 10 to 50% of the adult population. Large and symptomatic hernias are often corrected surgically. Association of pancreatic injury with HH repair is rare. A PubMed search using the keywords "hiatal hernia + pancreas" yielded only 15 results. Here, we present a rare complication of hiatal hernia repair resulting in pancreatic injury and leak.
Case Description/Methods: A 58-year-old female withhypertension, acid reflux, , and a type III paraesophageal hernia presented to General Surgery clinic for evaluation of dysphagia, belching, and -bloating for several months. For a large symptomatic hiatal hernia, she underwent laparoscopic Nissen fundoplication. On post operative day (POD) 2, she developed hypoxia requiring intubation. CT chest demonstrated a large left pleural effusion extending across the midline of the abdomen with mass effect causing multifocal atelectasis (**Figure**). On POD 9, WBC count increased to 19.6 K/uL and she had intermittent fevers with a temperature of 38.8C. A left pigtail catheter was placed for the pleural effusion; amylase of the pleural fluid was elevated to 4,544 U/L. Repeat CT C/A/P showed peripancreatic stranding and fluid with a hypodense band within the pancreas concerning for focal injury and pancreatic leak. ERCP showed no brisk contrast extravasation, but due to a high clinical suspicion for pancreatic leak, sphincterotomy was performed and a flanged pancreatic duct stent was placed. Percutaneous drainage along with endoscopic pancreatic duct stent placement decreased the size of fluid collections and helped with the symptoms.
Discussion: Laparoscopic repair remains the treatment of choice for symptomatic hiatal hernias and is generally well-tolerated. Pancreatic injury and leak are rare complications associated with large hernia repairs. Gastroenterologists need to be aware of such presentations while following these patients during post operative period.



[1917] **Figure 1.** CT A/P demonstrating a large hiatal hernia.

S1918

A Rare Hepatobiliary Manifestation of a Common Opportunistic Gastrointestinal Infection

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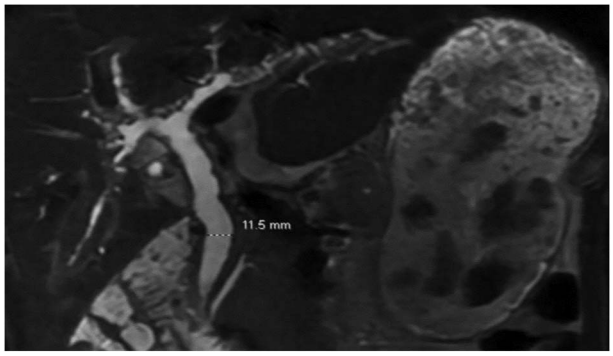
Introduction: AIDS cholangiopathy is a rare biliary tract syndrome seen in AIDS patients primarily with CD4 counts less than 100 cells/microliter. It commonly manifests as multiple biliary strictures secondary to chronic inflammation caused by one or more opportunistic infectious pathogens with Cryptosporidium parvum being isolated in most cases. The overall incidence is currently unknown but has decreased since initiation of potent ART in the mid-1990s. Here we describe a case of a 29-year-old male with newly diagnosed AIDS who presented with months of gradually worsening abdominal pain and diarrhea.
Case Description/Methods: A 29-year-old African American man with a past medical history of recently diagnosed HIV/AIDS and treated syphilis presented with 6 months of chronic diarrhea associated with persistent right upper quadrant abdominal pain. Physical examination on admission was significant for a cachectic male with severe tenderness to palpation in the right upper abdominal quadrant with a positive Murphy sign. Lab work revealed ALP 275 U/L, ALT 74, AST 93 with normal total bilirubin, CD4 count 68, fecal calprotectin 142, and lipase 467. Fecal pathogens were positive for Enteroaggregative Escherichia coli and Cryptosporidium parvum. Imaging revealed extensive hepatobiliary distension with gallbladder wall thickening. MRCP showed dilated intra- and extrahepatic biliary ducts with scattered microabscesses, multiple small filling defects within the CBD, and filling defects within the gallbladder lumen (**Figure**). AIDS-related cholangiopathy was diagnosed and treatment with nitazoxanide and ciprofloxacin was started following initiation of ART. ERCP was deferred given the patient's immunocompromised state and his improvement on medical therapy alone without evidence of biliary obstruction.
Discussion: The two most common pathogens associated with AIDS cholangiopathy are Cryptosporidium parvum and Cytomegalovirus. ERCP is often necessary to relieve any biliary strictures that predispose to obstruction but in this case, symptomatology improved with medical management of infection alone. This case highlights the importance of recognition of this rare AIDS-related syndrome and prompt GI service evaluation as this cholangiopathy is not seen in other immunocompromised cohorts such as transplant patients. Proper imaging with MRCP and consideration of ERCP to relieve strictures if obstruction is suspected is crucial.



[1918] **Figure 1.** MRCP findings.

S1919

A Rare Case of Signet Ring Cell Adenocarcinoma of the Gallbladder

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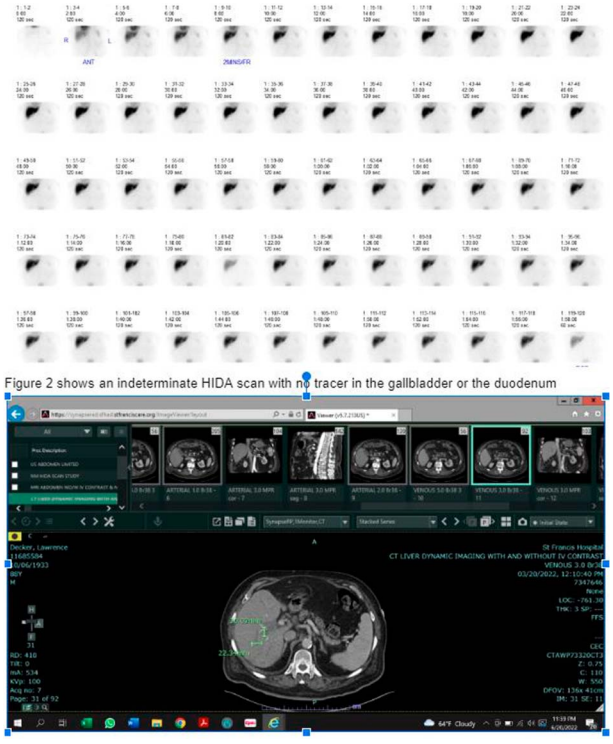
Introduction: Signet-ring cell adenocarcinoma (SCA) are extremely rare outside the stomach and colon. The majority of the literature consisted of case reports. The nonspecific presenting symptoms and the dismal prognosis associated with this disease, makes early detection crucial but very challenging. We report a case of SCA with rare presentation.
Case Description/Methods: An 88-year-old male with a history of diabetes and CAD, presented with a 3-week history of watery diarrhea. On presentation, he had signs of dehydration. His labs were significant for worsening cholestatic liver injury with uprising AST 179 (to 266) U/L, ALT 149 (to 192) U/L, ALP 844 (to 1,660) U/L, total bilirubin of 2.3 (to 16.5) mg/dL, but normal lipase. Gastroenteritis was initially suspected, however, infectious workup was negative. RUQ US ruled out CBD dilatation. Dynamic CT of the liver revealed soft tissue lesion in porta hepatis with periportal edema and mild biliary dilatation in the right hepatic lobe. MRI/MRCP revealed findings consistent with acute cholecystitis but no biliary dilatation. On HIDA scan, tracer was not found in gallbladder or intestines. Liver biopsy revealed SCA and CA 19-9 level was >20,000 U/mL. Patient passed away 6 weeks after presentation (**Figure**).
Discussion: Our SCA is an aggressive and rare variant of adenocarcinoma, that is mostly reported in gastric or colonic cancers, and is extremely rare outside these two locations. It has poor prognosis, owing to its late stage presentation with nonspecific symptoms. Our patient presented with symptoms mimicking gastroenteritis and imaging was suggestive of acute cholecystitis, with some indication of biliary obstruction. Liver biopsy revealed SCA which was otherwise an unexpected diagnosis. This highlights the importance of keeping rare cancers such as this in the differential, after the usual suspects have been ruled out. Heterogeneous attenuation of hepatic lobes on imaging, not correlating with clinical/symptom severity, warrants further investigation. With a median survival of only four months, early detection is crucial to reduce the morbidity and mortality associated with this condition. However, the diagnosis is usually delayed due to the nonspecific symptoms, which usually occur late in the course of the disease.



[1919] **Figure 1.** Pertinent imaging associated with the case.

S1920

A Rare Case of Small Cell Lung Cancer With Pancreatic Metastasis Presenting as Obstructive Jaundice

Tushar Khanna, MD[1], Suhind Kodali, MD[2], Christopher Hakim, MD[3], Stefan Odabasic, MD[1], Fadi Alali, MD[3], Serge Sorser, MD[3], Jay Levinson, MD[3].
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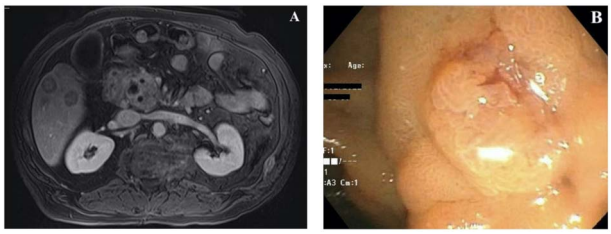
Introduction: Small cell lung cancer (SCLC) is an aggressive high grade neuroendocrine carcinoma that accounts for 15% of all lung cancers & usually presents with early distant metastatic spread typically to liver, bone, brain & lung on diagnosis. Pancreas is a very uncommon site of metastasis. We present a rare case of SCLC metastasis to the pancreas presenting as obstructive jaundice upon initial presentation.
Case Description/Methods: A 62-year-old male with a past medical history of recurrent pancreatitis presented to the emergency department with abdominal pain, nausea, vomiting, & jaundice. Routine blood work on admission were notable for elevated total bilirubin of 16.4 mg/dL along with elevation in alkaline phosphatase at 547 IU/L & elevation in AST & ALT to 120 U/L & 144 U/L, respectively. Imaging with CT followed by MRI abdomen showed focal enlargement of the pancreatic head with dilatation of the common bile duct & the main pancreatic duct along with innumerable rim-enhancing lesions within the liver consistent with metastasis. CT Chest was also done that showed a large right suprahilar mass with mass effect on the right main bronchus. CT guided biopsy of liver lesion was performed which confirmed small cell carcinoma on pathology. Worsening jaundice prompted ERCP with EUS which showed a large mass at the major papilla site. Pancreatic duct was cannulated & sphincterotomy was performed; However, biliary cannulation could not be completed due to the size of the mass & prior history of chronic pancreatitis cannulation. Patient subsequently underwent IR guided PTC drain placement with appropriate decrease in bilirubin. Chemotherapy was initiated with carboplatin/ etoposide for metastatic small cell cancer with spread to the pancreas & liver (**Figure**).
Discussion: SCLC metastasis to pancreas is rare & majority of patients with pancreatic metastasis are asymptomatic upon incidental diagnosis by abdominal CT. Pancreatitis & obstructive jaundice are seldom found on initial presentation, as seen in our patient. Jaundice can result from biliary ductal obstruction if SCLC spreads to lymph nodes in the porta hepatis or the pancreatic head. Endoscopic ultrasound (EUS) is more sensitive than CT or MRI in the diagnosis of pancreatic malignancy. Metastasis to pancreas typically indicates advanced disease & clinicians should consider palliative treatment with surgical biliary bypass & chemotherapy in select patient groups which has shown to lead to clinically significant increased mean survival time.



[1920] **Figure 1.** MRI Abdomen with contrast transverse image showing rim-enhancing lesions within the liver and focal enlargement of the pancreatic head with dilatation of the common bile duct and the main pancreatic duct (A). Endoscopic imaging showing a large fungating mass was found at the major papilla (B).

S1921

A Rare Cause of Small Bowel Obstruction

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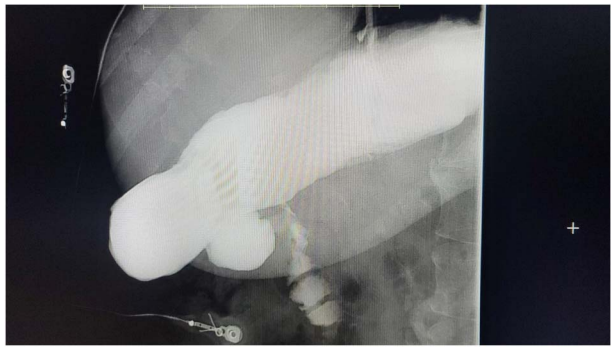
Introduction: We are presenting a rare case of small-bowel obstruction from gallstone. Gallstone ileus (GI) typically results from a cholecystoduodenal fistula. Incidence is less than 5% of patients who presents with small-bowel obstruction. Our case was unique presentation of mechanical small-bowel obstruction with a large 53 mm gallstone which passed through a 2 inch cholecystoduodenal fistula treated with 2 stage surgical procedure.
Case Description/Methods: A 62-year-old woman presented with diffuse abdominal pain, nausea and vomiting for 2 days. Her abdomen was soft, mildly distended and tender in epigastric region. A computed tomography of the abdomen showed distended small bowel with transition point in the right side of the abdomen suspicious for small bowel obstruction. Numerous small calcified densities in the lumen of small bowel noted just proximal to the obstruction point. A large ulceration extending from duodenal bulb into the gallbladder with inflammatory stranding noted. An upper GI series and small bowel follow through showed pocket of contrast projecting laterally from the proximal duodenum which persist on later images corresponding to fistulization (**Figure**). Patient was taken for exploratory laparotomy. During surgery, a large conglomerate of multiple gallstones was removed from proximal ileum. It was 53 mm in greatest dimension. Due to severity of inflammation and adhesions, fistula repair was not attempted. Patient did well after surgery. A second surgery was planned after 3 months. Patient underwent cholecystectomy, primary repair of fistula as well as lysis of adhesions.
Discussion: GI is a rare complication of cholelithiasis which presents as small-bowel obstruction. It was first described in 1654 by Dr Erasmus Bartholin. The pathogenesis involves adhesions formation between the inflamed gallbladder and adjacent part of the gastrointestinal tract. Subsequently, large stones within the gallbladder cause pressure necrosis, resulting in formation of a cholecystoenteric fistula, which allows gallstones direct access to the bowel. Most fistulas involve the duodenum, but fistulas to the stomach and colon have been described. This commonly effects elderly woman who usually presents with abdominal pain, abdominal distension nausea and vomiting. Patient may present with radiographic findings of Rigler's triad - air in bile duct, small intestinal obstruction and ectopic gallstone. Management is primarily surgical with 1 stage or 2 stage procedure depending on presence of severe inflammation and adhesions.



[1921] **Figure 1.** Upper GI series showing fistulous tract from proximal duodenum with opacification of gallbladder.

S1922

A Twisted Plot: Pancreas Divisum With Right-Sided Pancreaticopleural Fistula

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Introduction: We report on a 53-year-old male with a past medical history significant for chronic pancreatitis who presented with a right-sided pleural effusion secondary to pancreaticopleural fistula with pancreatic divisum. Pleural effusion secondary to pancreaticopleural fistula (PPF) is a rare complication of acute or chronic pancreatitis and usually presents with a left-sided pleural effusion. This case also highlights the difficulty and significant delay in diagnosing pancreas divisum on cross-sectional imaging.
Case Description/Methods: A 53-year-old male with a medical history significant for chronic pancreatitis complicated by pancreatic ascites, walled-off necrosis, splenic and portal vein thrombosis, alcoholic gastritis, and polysubstance use presented with five days of dyspnea with new and worsening oxygen requirements. Chest x-ray showed a new moderate right-sided pleural effusion. Pleural fluid analysis revealed elevated lipase of >3,000 U/L and amylase of 21,008 U/L concerning for a pancreaticopleural fistula. MRI of pancreas showed a disconnected duct at the tail of the pancreas with a fistula extending into the

mediastinum and peritoneum. During ERCP a bulging minor papilla was noted. After failed initial attempts at cannulating ventral duct, minor papilla was approached and dorsal duct was cannulated. Pancreatogram revealed a complete pancreatic divisum with a leak in the tail of the pancreas. A temporary plastic stent was placed in the dorsal duct after minor papilla sphincterotomy. The patient's symptoms resolved and he was discharged home with outpatient follow-up (**Figure**).

Discussion: PPF is a rare complication of pancreatitis that typically presents as large, recurrent, left-sided pleural effusion often refractory to thoracentesis management. Transpapillary stent placement in the pancreatic duct is widely used in the management of PPF. Our case shows a rare right-sided presentation of PPF in the setting of a newly diagnosed pancreas divisum missed on numerous previous cross-sectional imaging. High clinical suspicion of underlying divisum raised by endoscopic and imaging findings led to successful endoscopic management of this complex case.

[1922] **Figure 1.** MRCP Pancreas showing pancreas divisum and disconnected duct.

S1923

Abutting Percutaneous Drains Can Jeopardize Healing of Post-Cholecystectomy Bile Leaks

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Introduction: Endoscopic retrograde cholangiopancreatography (ERCP) is an effective treatment for post-cholecystectomy (CCY) biliary leaks (BL). However, the presence of a Jackson Pratt (JP) drain abutting on the site of BL may prevent ERCP interventions from being effective.

Case Description/Methods: In this case series, we present 5 cases of CCY induced BL with JP drains noted to be abutting the site of BL by the endoscopist during initial ERCP therapy. We reviewed the ERCP fluoroscopic images and confirmed that the tip of the JP drains was in proximity (within 1cm) or right against the site of contrast extravasation. The details of each case are summarized in Table 1 including the ERCP interventions and the intervals between ERCPs. All 5 cases had a persistent BL documented on repeat ERCP with their JP drains remaining in the same position on fluoroscopic images. For case 1, the JP drain was left in-situ after the first and second ERCPs and a persistent BL from the cystic duct stump can be seen flowing directly into the adjacent JP drain tip on the third ERCP (**Figure A**). In **Figure** B, the endoscopist withdrew the JP drain by about 3cm. In **Figure** C, the JP drain was already removed prior to the fourth ERCP, and the BL has resolved. Case 2 had ERCP earlier than scheduled due to cholangitis, while case no. 4 returned for an earlier ERCP due to persistently high JP drain output of 900-1000ml a day. For cases 2 to 4, when a persistent BL was found, the endoscopist communicated with the surgical team and the JP drain was either retracted by 1-3cm or removed on the same day. After JP drain adjustment, the patients did well, and their BL had resolved by the time of their subsequent ERCP 4 to 23 weeks later. For case no. 5, the JP drain was left in position for 14 weeks after intraoperative placement, and the drain tip was found to have eroded into the biliary duct during the third ERCP for the persistent BL. In this case, the surgeon on call was contacted who repositioned the drain in the endoscopy unit. The JP drain output decreased and was subsequently removed; the BL resolved on subsequent ERCP 22 weeks later (**Table**).

Discussion: From these cases, we observed that a JP drain abutting onto the site of BL can create a bigger pressure gradient between the bile duct defect and the drain, making ERCP interventions less effective for promoting transpapillary bile flow. This can be remedied by withdrawing the JP drain by 1-3 cm.

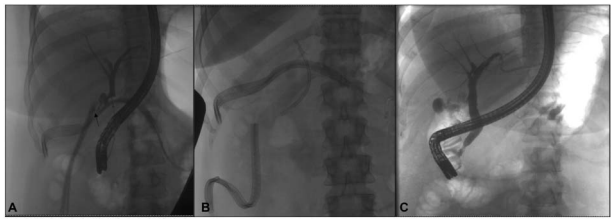
Table 1. Case Summaries

Case Index	Patient demographic	Type of Bile leak	1st ERCP Interventions	2nd ERCP Interventions	3rd ERCP Interventions	4th ERCP
1	42 YO female	Strasberg A BL from CD	BES + Single PS (JP drain left in place)	Persistent BL on ERCP 4 weeks later. SEMS placed. (JP drain left in place.)	Persistent BL on ERCP 4 weeks later. Single PS placed (JP drain retracted by endoscopist.)	*BL resolved on ERCP 4 weeks later
2	58 YO male	Strasberg A BL from CD	BES + Single PS (JP drain left in place)	Persistent BL on ERCP 1 week later. Two PS placed. (Surgical team consulted and JP drain retracted.)	*BL resolved on ERCP 4 weeks later .	N/A
3	75 YO female	Strasberg A BL from CD	BES + Single PS (JP drain left in place)	Persistent BL on ERCP 6 weeks later. SEMS placed. (Surgical team contacted and JP drain retracted.)	*BL resolved on ERCP 23 weeks later .	N/A
4	69 YO male	Strasberg A BL from CD	7Fr Single PS (JP drain left in place)	Persistent BL on ERCP 1 week later. BES+ 10 Fr Single PS placed. (JP drain removed by surgeon on same day post ERCP.)	*BL resolved on ERCP 5 weeks later.	N/A
5	72 YO male				Persistent BL on ERCP 8 weeks later. SEMS + two PS placed.	*BL resolved on ERCP 22 weeks later.

Table 1. (*continued*)

Case Index	Patient demographic	Type of Bile leak	1st ERCP Interventions	2nd ERCP Interventions	3rd ERCP Interventions	4th ERCP
		Strasberg D BL from CHD	BES + incomplete stone extraction + Single PS (JP drain left in place)	Persistent BL on ERCP 1 day later. Stone extraction with Spyglass with EHL + Two PS (JP drain left in place.)	(Spyglass cholangioscopy found abutting JP drain had eroded into CHD. Surgeon was contacted who repositioned the JP drain on same day.)	

Abbreviations: YO: Years old. CD: Cystic duct. CHD: Common hepatic duct. BES: Biliary endoscopic sphincterotomy. PS: Plastic stent. SEMS: Self-expandable metal stent. EHL: Electrohydraulic lithotripsy. JP: Jackson Pratt. BL: Bile leak. *Jackson Pratt drain was removed prior to time of ERCP.



[1923] **Figure 1.** A: A persistent bile leak is seen at the cystic duct stump (arrow) draining directly into the Jackson Pratt drain with its tip abutting the site of leakage during the patient's 3rd ERCP. The biliary metallic stent had been removed at time of this fluoroscopic image. B: A plastic biliary stent was placed. The Jackson Pratt drain was pulled back. C: The cystic duct bile leak resolved during the 4th ERCP. Prior to this ERCP, the patient had both abdominal drains removed by her surgical team due to low output.

S1924

AIDS Cholangiopathy Secondary to Kaposi Sarcoma: A Rare Presentation

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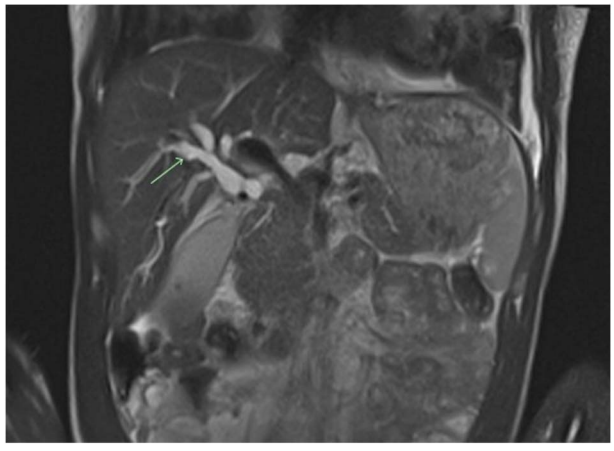
Introduction: AIDS cholangiopathy occurs in the setting of advanced immunosuppression in HIV patients and is caused by opportunistic pathogens such as Cryptosporidium and Cytomegalovirus (CMV). Although hepatic and GI tract involvement due to Kaposi sarcoma (KS) is a known entity, cholangiopathy involving the biliary tree is extremely rare and is sparsely reported in the literature. Here, we present a case of an immunocompromised patient with biopsy-proven KS of the bile duct.
Case Description/Methods: A 39-year-old male with a known history of HIV/AIDS presented to the ER with right upper quadrant abdominal pain ongoing for 2 weeks. He also reported significant weight loss and loss of appetite. Vital signs on presentation were unremarkable and examination revealed mild tenderness of the RUQ and enlarged right axillary lymph nodes. The pertinent laboratory panel included total bilirubin of 2.5 mg/dL, alkaline phosphatase of 1,883 U/L, AST 329 U/L, ALT 312 U/L, INR 1.0, and CD4 count of 15 cells/μL. Home medications included bictegravir/emtricitabine/tenofovir (Biktarvy) which was started approximately 6 weeks before hospitalization. MRCP showed dilation of intra-hepatic and hepatic ducts with possible filling defects in the common bile duct. ERCP with cholangiogram demonstrated severely dilated CBD measuring up to 14 mm and stricture of hepatic ducts which were cannulated using a 10 French stent. FNA of the biliary structures revealed Human Herpesvirus-8 (HHV-8) consistent with Kaposi Sarcoma (**Figure**).
Discussion: AIDS cholangiopathy is characterized by infection of the biliary tract by opportunistic organisms resulting in strictures and obstruction. The most frequent organisms implicated in HIV cholangiopathy include Cryptosporidium, CMV, Microsporidium, and Mycobacterium Avium. Cholangiopathy resulting from KS in the era of ART is an uncommon presentation and usually involves the liver, but very rarely the biliary tree. Cholangiopathy usually manifests as vague abdominal discomfort, markedly high levels of serum alkaline phosphatase (> 1000 U/L), and near-normal bilirubin levels. Four patterns of cholangiopathy have been described: 1) papillary stenosis, 2) sclerosing cholangitis, 3) combination of the 2 and 4) extra-hepatic strictures. Management involves imaging modalities such as MRCP and therapeutic interventions with ERCP to obtain bile duct brushing and possible sphincterotomy. It is also of paramount importance to initiate and maintain ART. Our patient most likely developed KS due to non-adherence to ART.



[1924] **Figure 1.** MRI abdomen with contrast showed dilation of intrahepatic bile ducts and possible filling defects in the common bile duct.

S1925

All Nodules Are Not Equal: A Rare Presentation of Pancreatic Cancer

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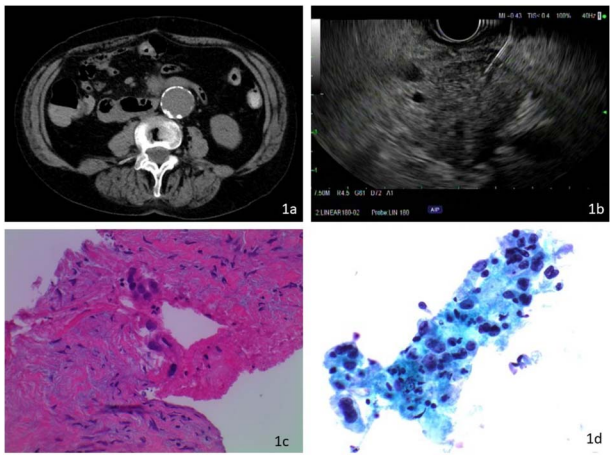
Introduction: Pancreatic cancer has a 5-year survival of about 11%. An atypical presentation can make early diagnosis a challenge. Standard diagnostic measures such as computed tomography (CT) and endoscopy (EGD/EUS) may not provide enough information for diagnosis of a pancreatic cancer, particularly if presence of unusual anatomy was not previously known. In this case, we present a patient with adenocarcinoma developing in an ectopic pancreas (EP) adjacent to the third part of duodenum (D3)
Case Description/Methods: A 71-year-old female with past medical history of an abdominal aortic aneurysm, coronary artery disease, end-stage lung disease on home oxygen, hypertension, and chronic abdominal pain presented with abdominal pain and nausea. She had a CT scan which revealed a 10mm mesenteric nodule adjacent to D3. A follow-up study six weeks later revealed enlargement of the node to 13mm x 11mm (**Figure A**), and she was referred to surgery for evaluation. Patient was then referred to the gastroenterology for an endoscopic evaluation of what was initially thought to be a lymph node. An EGD/EUS revealed normal endoscopic findings, with a heterogenous periduodenal lesion measuring 3cm x 2.1cm was identified (**Figure B**). This was separate from the remainder of the pancreas. A biopsy of this lesion was performed. Pathology revealed groups of crowded atypical glandular cells with anisonucleosis; consistent with a diagnosis of pancreatic tissue with well-differentiated adenocarcinoma (Figure C, D). The patient was deemed to not be a surgical candidate due to advanced COPD. The patient decided to pursue comfort and hospice care
Discussion: Prior research has shown that EP is uncommon, with discovery during surgery and autopsies estimated at 0.25% to 0.55-13.7% respectively. Still rare is the frequency of malignant transformation, estimated at 0.7-1.8% of all EP cases. Studies have shown that patients with a malignancy in EP have higher rates of symptomatic disease as compared to non-ectopic primary pancreatic cancer. A challenge in the diagnosis of malignant EP is the varying sites where ectopic tissue occurs. The most common location is the stomach (25-38%), followed by the duodenum (17-21%) and jejunum (15-21%), with other sites much less common. Our patients' presentation of malignancy in EP tissue adjacent to D3 was exceedingly rare. The prognosis of malignant EP remains unclear, primarily due to the rarity of this condition. Further research is needed to better evaluate and assess the natural course of the condition



[1925] **Figure 1.** a: CT scan showing the nodule; b: EUS image with nodule appearing as a hypoechoic lesion. Biopsy was performed. c: Pathologic staining, H&E (200x) show fibrotic / desmoplastic pancreatic tissue with irregular / abortive infiltrating glands with nuclear enlargement. d: Papanicolou staining at 400x magnification with similar findings.

S1926

A Young Female With Acute Cholangitis Due to Type IVa Choledochal Cyst With an Undifferentiated Spindle and Giant Cell Carcinoma Hiding Inside

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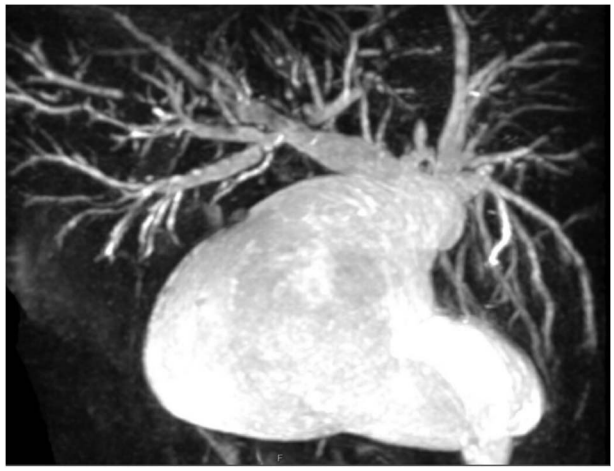
Introduction: This patient is being presented to demonstrate the rarity and severity of extrahepatic biliary cystic masses as the primary etiology of acute cholangitis in a young patient, after failing endoscopic stent placement.
Case Description/Methods: A 29-year-old female with no medical history presented with 2 days of sharp, epigastric pain radiating to the back associated with fevers and non-bilious vomiting. Patient's exam noted scleral icterus and abdominal exam showed tenderness to palpation in the right upper quadrant. Labs showed a WBC of 26,300, AST of 114, ALT of 289, ALP of 343 IU/L, and total bilirubin of 9.76 mg/dL. Computed tomography (CT) of the abdomen showed dilated proximal extrahepatic CBD with a large complex cystic mass in the porta hepatitis measuring 10 cm x 9 cm x 8 cm and this was contiguous with gallbladder and proximal common bile duct with mass effect on duodenum and pancreatic head concerning for Type I choledochal cyst. An ERCP showed a significantly dilated CBD with cystic dilation and balloon sweep with 9-10-12 mm balloon catheter was performed with passage of a small stone and pus. Two plastic stents 10 Fr x 7 cm and 10 Fr x 9 cm were placed with appropriate bile drainage. Fevers continued and total bilirubin increased to 14.2 mg/dL and ALP reached 535 IU/L, therefore patient was taken for laparoscopy which showed large choledochal cyst intimately involved with the head of the pancreas and duodenum, necessitating a Whipple procedure for distal margin. Proximal margin entered the left and the right hepatic ducts, consistent with a type IVa choledochal cyst. Anatomic pathology of the 7.0 cm x 6.0 cm x 3.0 cm mass in the cyst showed an undifferentiated spindle and giant cell carcinoma, confined to the bile duct histologically, with lymphovascular and perineural invasion present. There was no gallbladder involvement and 25 lymph nodes were negative for malignancy. The patient improved and was discharged to home after extended recovery (Figure).
Discussion: Undifferentiated carcinoma of the extrahepatic biliary system is extremely rare and has been previously found on middle-age to elderly patients. Diagnosis of undifferentiated carcinoma of the extrahepatic biliary system is difficult because the exact diagnosis cannot be determined prior to surgical resection. Patients with undifferentiated carcinoma of the extrahepatic bile duct may have a relatively better prognosis due to the early appearance of jaundice, compared to those with gallbladder involvement.



[1926] **Figure 1.** MRCP of Type IV choledochal cyst

S1927

A Unique Endoscopic Presentation of Intraductal Papillary Mucinous Neoplasms (IPMN) Presenting as a Hemorrhagic Mass

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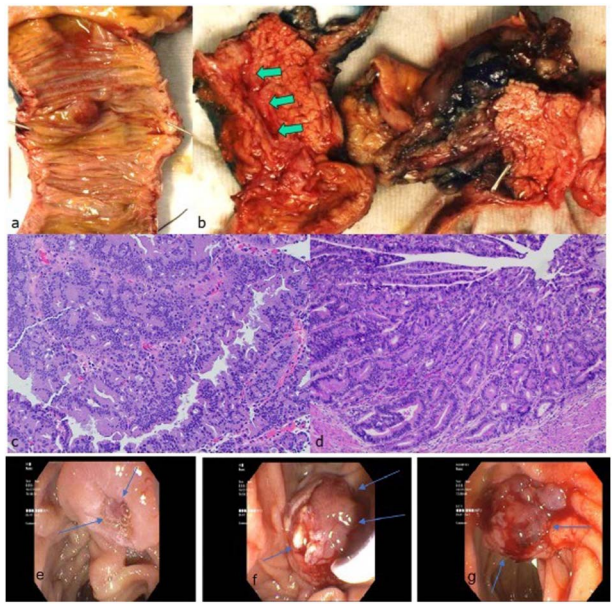
Introduction: Pancreatic Intraductal papillary mucinous neoplasms (IPMN) are mucinous lesions of the pancreatic duct, accounting for approximately 1% of all pancreatic neoplasms and one fourth of cystic neoplasms. Three types exist: main duct IPMN, branch duct IPMN, and mixed-type IPMN. IPMN is a histologically benign tumor with the potential to be precancerous, and subsequently progress to pancreatic ductal adenocarcinoma. Given the potential of progressing from low-grade dysplasia to invasive carcinoma in approximately 4-6 years, early detection can improve prognosis. They may be asymptomatic or cause abdominal pain, nausea, vomiting, weight loss, jaundice, and pancreatitis. Diagnosis is by CT, MRI, or, in some cases endoscopic ultrasound (EUS) with fine needle aspiration (FNA), followed by biochemical and histopathological analysis.
Case Description/Methods: We report a 45-year-old female with a history of hypertension, who presented to our hospital after outside imaging showed a pancreatic mass on CT abdomen and pelvis. EUS was performed, which showed an irregular, 10-mm mass with well-defined borders in the main pancreatic duct. There was no abnormality of the common bile duct and no stones identified. The patient subsequently underwent an ERCP one month later, which showed moderate dilation of both the ventral pancreatic duct in the head of pancreas and main pancreatic duct. A 12-15 mm hemorrhagic "cauliflower" mass was also found which was oozing and friable. Biopsy was consistent with intraductal papillary mucinous neoplasm. The patient subsequently underwent a successful Whipple procedure. Surgical pathology returned showing intra-ampullary papillary tubular neoplasms (IAPN) with high grade dysplasia, and resection margins negative for neoplasm (**Figure**).
Discussion: With technological advancements in radiographic and endoscopic imaging, the incidence of IPMN has increased. CT fish mouth ampulla sign (continuous water attenuation from the pancreatic duct to the duodenal lumen) and endoscopic fish mouth ampulla (E-FMA) signs are highly specific for main duct and mixed IPMN. Our patient's findings are unique in that her ERCP showed a hemorrhagic mass representing an unusual presentation of a common pancreatic lesion. Additionally, we aim to illustrate the importance of early endoscopic evaluation with endoscopy in patients with pancreatic changes on cross sectional imaging. The malignant potential for main duct IPMN is greater than for branch and mixed type IPMNs and therefore more commonly surgically resected.



[1927] **Figure 1.** (a) The mass is extensively involving the ampulla of vater (seen from the duodenum) bulging out into the duodenal lumen; (b) Whipple specimen showing dilated pancreatic duct. Microscopically, both EUS aspiration biopsy (c, cell block) and the whipple specimen (d) show mucinous adenocarcinoma with identical tumor morphology. [c and d, Hematoxylin and Eosin stain, 40x magnification]. Subsequent ERCP demonstrating fish mouth ampulla (e), cauliflower mass with stent placement (f), hemorrhagic mass with friability (g).

S1928

A Young Immunocompetent Patient Presenting With Extrapulmonary Tuberculosis Complicated by Pancreatic Involvement Mimicking a Malignant Tumor

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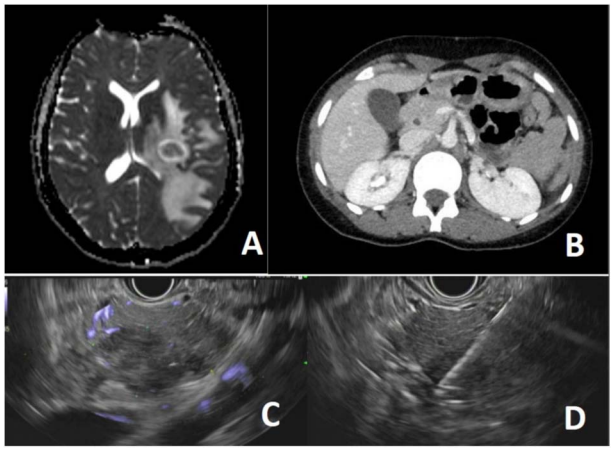
Introduction: Tuberculosis commonly affects the lungs and is communicable through respiratory droplets. Extrapulmonary Tuberculosis can involve almost any organ system but is much less common and often difficult to diagnose in the gastrointestinal tract.
Case Description/Methods: A 24-year-old Somali woman with no known past medical history experienced progressively worsening headaches associated with nausea and vomiting, as well as numerous small lesions on her left arm that eventually developed into large ulcerations. She subsequently had multiple falls associated with seizure-like movements and came to the United States to seek further care. MRI brain revealed rim-enhancing lesions with surrounding vasogenic edema (**Figure** A). CT abdomen and pelvis revealed an ill-defined 3.0 x 3.1 cm pancreatic head mass with mass effect on the portal vein and superior mesenteric vein, prominence of the common bile duct with abrupt cutoff (**Figure** B), and multiple enlarged thoracic lymph nodes. Endoscopic ultrasound (EUS) showed a poorly demarcated hypoechoic lesion with calcifications and focal necrosis in the pancreatic head (**Figure** C). Fine-needle biopsy was performed (**Figure** D). The remaining pancreatic parenchyma and duct appeared normal, and it was felt that the EUS findings were not typical malignant features. Cytopathology was negative for epithelial malignancy and revealed neutrophils with granulomatous inflammation. Further evaluation showed negative acid-fast bacilli (AFB) stains and Mycobacterium tuberculosis PCR, with an indeterminate non-TB Mycobacteria PCR. CA 19-9 and CEA were normal. Neuroradiology re-evaluated the MRI and felt atypical infection was a possible cause of the intracranial findings. Punch biopsy of her arm lesions revealed an ulcer with underlying necrotizing granulomatous inflammation. The multidisciplinary care team concluded her presentation was most consistent with extrapulmonary Tuberculosis given her necrotizing granulomas and prior residence in an endemic area. She was started on Rifampin, Isoniazid, Pyrazinamide, and Ethambutol.
Discussion: The diagnosis of extrapulmonary Tuberculosis can be challenging due to nonspecific symptoms and low sensitivity of the AFB stain. The most common sites include the bones, lymph nodes, and pleura, with the gastrointestinal organs much less frequently involved. Tissue biopsy of suspected lesions is helpful and can reveal granulomatous inflammation. Correlation with clinical, microbiological, laboratory, and radiologic findings is the key to diagnosis.



[1928] **Figure 1.** A) Rim-enhancing lesions with vasogenic edema B) Ill-defined pancreatic head mass with local mass effect C) Poorly demarcated hypoechoic pancreatic head lesion with calcifications and focal necrosis D) Fine-needle biopsy

S1929

Acinar Cell Cystadenoma With Pancreatic Intraepithelial Neoplasia: Is It Always Benign?

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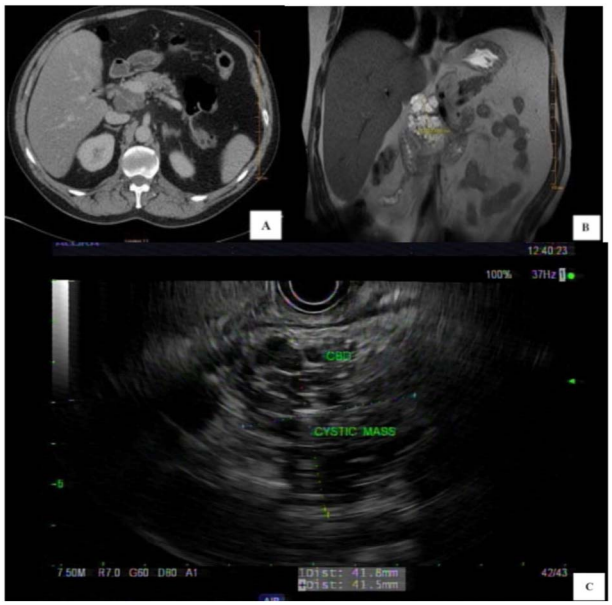
Introduction: Acinar cell neoplasms are rare, constituting < 2% of all pancreatic lesions. Acinar cell cystadenoma (ACC) was first described as a non-neoplastic entity in 2002. The exact etiology has been debated, primarily whether it originates from or carries a risk of underlying neoplasia. Pancreatic intraepithelial neoplasia (PanIN) is presumed to be one of the common noninvasive precursors of pancreatic ductal adenocarcinoma, intraductal papillary mucinous neoplasm, and mucinous cystic neoplasm. This report presents a rare case of an ACC with low-grade PanIN requiring surgical resection.
Case Description/Methods: A 60-year-old female with an unremarkable medical history presented with epigastric pain for two weeks. The physical exam was unremarkable. Her initial laboratory workup was notable for the mild isolated elevation of alkaline phosphatase but otherwise unremarkable. Computed tomography of the abdomen revealed a 5.0 x 4.0 cm cystic lesion in the pancreatic head with thick internal septations (**Figure** a). Magnetic resonance cholangiopancreatography showed a 5.2 x 4.5 x 6.8 cm lobulated cystic lesion in the pancreatic head with a microcystic configuration, multiple thin internal septations, and a hypointense central scar (**Figure** b). An endosonographic exam showed a large multi-cystic lesion in the pancreatic head region with normal ductal anatomy and the remainder of the pancreas (**Figure** c). Fine-needle aspiration showed a carcinoembryonic antigen level of 555 ng/ml & amylase of 13,593 U/L. No KRAS or GNAS mutations or loss of heterozygosity were detected. Because of the symptomatic nature of the large lesion, the patient underwent a Whipple procedure for resection. Pathology revealed a complex cystic lesion with well-differentiated acinar cells and patches of ductal epithelium compatible with ACC. Histology confirmed the presence of low-grade pancreatic intraepithelial neoplasia (PanIN) with no invasive carcinoma identified. Ten regional lymph nodes were negative for neoplasia. The patient recovered well from surgery, and repeat imaging two months later was unremarkable.
Discussion: Acinar cell cystadenoma (ACC) is a rare benign pancreatic lesion. High-grade pancreatic intraepithelial neoplasia (PanIN) may represent a precursor of pancreatic ductal adenocarcinoma, intraductal papillary mucinous neoplasm, and mucinous cystic neoplasm. Low-grade PanIN is usually found in more benign pancreatic lesions. Resection is recommended in symptomatic patients.



[1929] **Figure 1.** Radiographic findings in the patient diagnosed with acinar cell cystadenoma

S1930

Acute Acalculous Cholecystitis as an Unusual Extrapulmonary Manifestation of COVID-19: Do We Need to Think Twice in Patients Presenting With Abdominal Pain in COVID Times?

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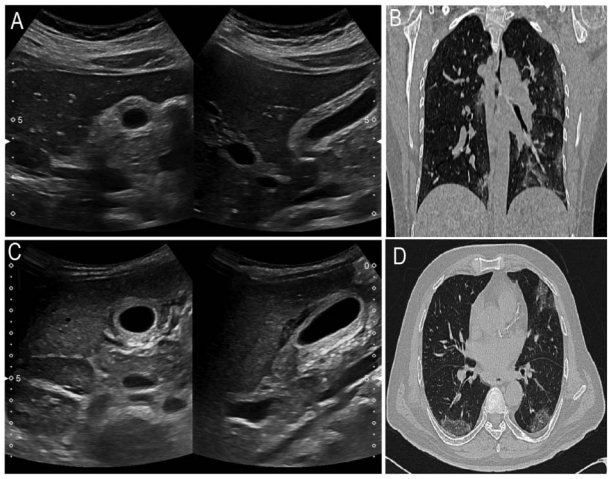
Introduction: The COVID-19 pandemic continues to pose a health challenge with emerging atypical presentations. It mainly affects the lungs, but patients may present with atypical GI symptoms. COVID-19-related acalculous cholecystitis (AAC) remains exceedingly rare. We hereby report 2 unique patients who were admitted with AAC-related abdominal pain as the only clinical presentation of COVID-19.
Case Description/Methods: Patient 1: A 41-year-old female presented to our hospital with severe, dull, right upper quadrant (RUQ) pain for 4 days. No other GI symptoms were noted. Clinical examination was positive for Murphy's sign. Laboratory studies revealed leukocytosis and elevated levels of inflammatory markers. Abdominal ultrasonography revealed gallbladder wall thickening and pericholecystic fluid, with no gallstones (**Figure** A). After one day in hospital, she also developed a low-grade fever, dry cough, and mild shortness of breath. Sars-CoV-2 testing using RT-PCR via nasopharyngeal swab was positive. CT scan also confirmed COVID pneumonia (**Figure** B). She achieved complete clinical recovery with piperacillin-tazobactam therapy, with no need for surgery. Patient 2: A 24-year-old previously healthy man came to our hospital for RUQ pain for 2 days. The dull, moderate pain radiated to his right shoulder. Physical examination showed RUQ tenderness. Murphy's sign was positive. Laboratory studies revealed an elevated white cell count and CRP level. Abdominal ultrasonography revealed marked gallbladder wall thickening and pericholecystic fluid collection, with no stones or sludge in the gallbladder fossa (**Figure** C). CT chest findings confirmed viral pneumonia (**Figure** D). Coronavirus testing with RT-PCR also came back positive. Treatment with meropenem and azithromycin achieved resolution of symptoms. The patient did not require surgery.
Discussion: The clinical association between AAC and COVID-19 remains fallible. We conducted a systematic review using terms: "acalculus cholecystitis" and "COVID-19," between inception and June 15, 2022. The search identified only 22 cases of COVID-19-related AAC. This article emphasizes that AAC can be the only clinical presentation of underlying COVID-19, with no pulmonary symptomology. SARS-CoV-2 should be excluded before admission using RT-PCR and CT chest in patients presenting with biliary symptoms related to cholecystitis. As in our cases, it is imperative to know that these patients may not require surgical intervention for AAC.



[1930] **Figure 1.** A: Abdominal ultrasound showing thickened gallbladder wall with pericholecystic fluid collection and no gallstones. B: CT chest revealing bilateral interstitial infiltrates. C: Ultrasonography abdomen showing gallbladder wall thickening, pericholecystic fluid collection, with no stones or sludge in the gallbladder fossa. D: CT chest showing bilateral, patchy, peripheral ground-glass opacities in the lungs.

S1931

Acellular Is Not Always Benign: A Case of Primary Pancreatic Sarcoma

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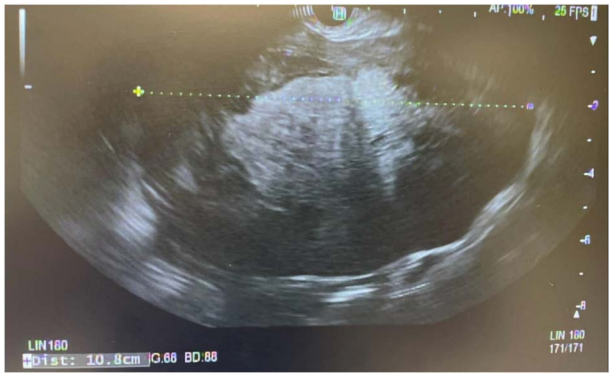
Introduction: Pancreatic Cystic Neoplasms (PCN) are encountered more frequently given the more widespread usage of cross sectional imaging. Primary pancreatic sarcomas represent 0.1% of all pancreatic malignancies and tend to be aggressive and have a poor prognosis.
Case Description/Methods: A 74-year-old woman with PMHx significant for HLD presented to ER with persistent nausea and vomiting of two days duration. She became concerned when she was unable to tolerate oral diet and decided to seek medical attention. There was no abdominal pain. Her only medication was atorvastatin. No family history of malignancy. Social history yielded occasional alcohol use (1-2/year) and no smoking. She was independent and ROS was unremarkable. Her PE was unremarkable with no abdominal tenderness. CXR and CBC were unremarkable. CMP was significant for a mildly elevated bilirubin 1.3. Given the fact that she was unable to tolerate PO she was admitted for further evaluation and supportive management. Due to unresolving symptoms she underwent a CT scan with IV contrast. It was significant for a 10.4 x 9.1 x 7.2cm cystic lesion centered in the root of the jejunal mesentery. The pancreatic body and tail were displaced and the main pancreatic duct appeared normal. The origin was unclear. MRI with and without contrast could not delineate the origin of the cyst either. AFP and CEA were unremarkable. EUS was undertaken and was significant for a large 10cmx8cm complex cyst in the pancreatic body (**Figure**). The cyst was heterogenous and had a solid component (90%). The bile ducts and pancreatic ducts were normal in caliber. There was no lymphadenopathy. A 22g needle was used to aspirate 1.5mL of fluid. Grossly the fluid was thin and serous appearing. Intraoperative microscopic review showed an acellular fluid with blood only. The patient improved with supportive management and was discharged home. Pathology subsequently showed high grade spindle cells positive for MDM2 with patchy SMA staining. They were negative for S100, SOX10, and CD117. This is consistent with a high-grade sarcoma however the specimen has been sent to a tertiary care center for a second opinion. The patient is currently awaiting oncology input but is systemically well
Discussion: It is of vital importance that large cystic lesions in close proximity to the pancreas be evaluated urgently with EUS. While intraoperative FNA results are helpful, what can appear benign can in fact be a highly aggressive tumor. Primary pancreatic sarcomas are a rare but important consideration of PCN.



[1931] **Figure 1.** EUS imaging of pancreatic complex cyst

S1932

Acute Pancreatitis After Liver Biopsy: Clots or Stones?

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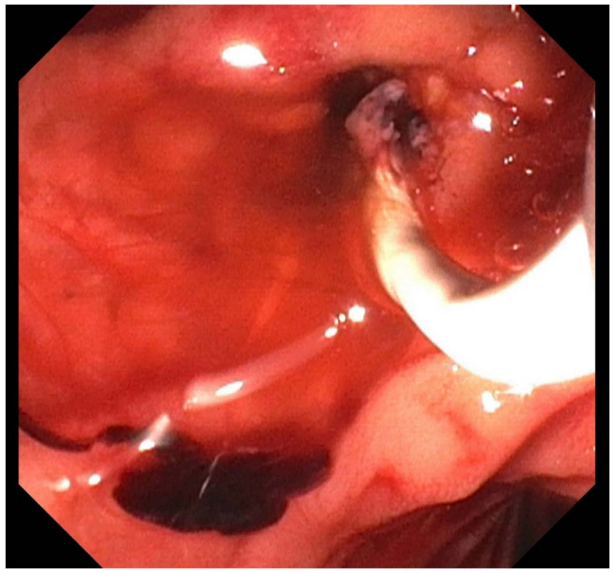
Introduction: Percutaneous liver biopsy is a frequently utilized tool in the diagnosis of many liver diseases including metastatic liver lesions. Bleeding complications occur in 1% of cases and hemobilia is rare. Hemobilia causing acute pancreatitis after percutaneous liver biopsy is extremely rare with approximately 20 reported cases in literature. Symptoms include right upper quadrant pain, nausea, jaundice, hematemesis, hematochezia or melena. We present an interesting case of a woman who developed acute pancreatitis secondary to hemobilia after liver biopsy.
Case Description/Methods: A 60-year-old female with recurrent high-grade serious peritoneal cancer and recent metastases to liver was admitted for acute pancreatitis. Patient presented with nausea and right upper quadrant pain that started after percutaneous liver biopsy one day prior. Labs notable for Hgb 10.4, lipase > 3000, amylase 3349, total bilirubin 3.6, direct 3.3, AST 631, ALT 482, alkaline phosphatase 240. CT A/P showed diffuse swelling of pancreas with fluid and mesenteric fat stranding suspicious for acute pancreatitis. Given liver enzyme elevation with mixed hepatocellular and cholestasis pattern, abdominal MRI was obtained to evaluate any biliary obstruction. MRI showed mildly prominent CBD at 0.8 cm with debris in distal CBD, cystic duct, and gallbladder indicative of hemorrhagic products. No hematoma noted in the liver parenchyma. Patient underwent ERCP with sphincterotomy and clots were cleared from the duct with balloon sweep (**Figure**).
Discussion: Hemobilia, described as upper GI bleeding within the biliary tree has a reported incidence of less than 0.059% after percutaneous liver biopsy. It can occur within a few hours or several days later, with an average interval of five days. Although rare, hemobilia can lead to acute pancreatitis in a mechanism similar to gallstone pancreatitis. Excessive bleeding and increased clot formation cause obstruction in the biliary tree and impedes drainage from the ampulla which in turn triggers pancreatitis. Diagnosis can be made by ultrasound, angiography, ERCP, or MRCP. Treatment depends on the extent of bleeding and includes conservative management to advanced interventions-hepatic angiography with arterial embolization, ERCP with sphincterotomy or stent placement. Thus, our case highlights importance of recognizing hemobilia as an etiology for acute pancreatitis after percutaneous liver biopsy.



[1932] **Figure 1.** Removal of blood clots from bile duct by balloon sweep via ERCP

S1933

Acute Pancreatitis Attributed to COVID-19: An Unusual Infectious Etiology

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Introduction: COVID-19 can affect multiple organs and has various presentations, including respiratory symptoms and fever being the most common. Gastrointestinal symptoms can be reported in up to 25% of patients, with diarrhea, nausea, and emesis the usual presentation. However, abdominal pain is less than 6.8% of cases, and of those, only 27% presented with Acute Pancreatitis (AP). Here we present a case of severe AP with multiorgan involvement as the initial presentation of COVID19.
Case Description/Methods: Case of a 19 y/o Female with a medical history of Obesity class I arrived at the ED with recurrent emesis and altered mental status. Symptoms began around four days prior with 6-7 episodes of non-bloody, non-bilious emesis, fatigue, subjective fever, and decreased oral intake, followed by acute onset of diffuse abdominal pain. Upon arrival, the patient was found encephalopathic with dry oral mucosa, tachycardic and tachypneic. Laboratories were remarkable for lipase 2483, amylase 382, LDH 315, ALP 219 U/L, sodium 155 mEq/L, creatinine 1.33 mg/dL, bicarbonate $<$ 9 mmol/L, pH 7.0, pO2Sat 96%, hematocrit 51.8%, WBC 14.9 cells/L, glucose 774 mg/dL, and COVID 19 PCR positive. Abdominal US and CT with no findings suggestive of gallstones or intraabdominal pathology. Aggressive intravenous fluid, insulin, broad-spectrum antibiotics, bicarbonate replacement, analgesia, and antiemetics were implemented. Hospitalization was complicated as the patient did not tolerate PO intake, and the high anion gap metabolic acidosis was difficult to close for several days. After almost two weeks in the ICU, the patient was discharged home without apparent pancreatitis or COVID-19 repercussions.
Discussion: Our case did not have any risk factors for AP, and extensive investigations did not reveal a clear etiology. Viral, bacterial, fungal, and parasitic infections are less common causes of AP. There are limited data on COVID-19 and pancreatitis; however, recent studies suggest a higher mortality rate with a 20% increased chance of dying and worse clinical outcomes. The mechanism of pancreatic injury is not well understood, but there is a link between COVID-19 and the precipitation of autoimmune and systemic diseases. Our case is an example of severe pancreatic injury with multiorgan involvement in mild symptomatic COVID 19.

S1934

Actinomycosis of the Common Bile Duct: A Rare Case Report

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Introduction: Actinomycosis is a rare, chronic disease caused by a group of anaerobic Gram-positive bacteria that normally colonize the respiratory, gastrointestinal and urogenital tract. Its incidence has diminished globally due to improved oral hygiene and the development of antibiotics. Multiple different clinical features of actinomycosis have been described, as various anatomical sites can be affected. Hepatobiliary actinomycosis is extremely rare and may present as biliary colic, acute or chronic cholecystitis or pancreatitis.
Case Description/Methods: A 75-year-old Caucasian woman performed a surgical ampullectomy due to ampullary adenoma with high-grade dysplasia in 2009. Nine years later, esophagogastroduodenoscopy revealed growth of adenomatous tissue in the same location. Mucosectomy was performed, without complications. Histological examination revealed a tubulovillous adenoma with low-grade dysplasia. In 2019, the patient presented to our institution with persistent right upper quadrant abdominal pain. The ultrasound revealed dilation of the common bile duct. Endoscopic retrograde cholangiopancreatography was performed and showed a deformed ampullary region, but without apparent residual lesion. Biopsies were performed, which revealed fragments of duodenal mucosa with abundant inflammatory infiltrate and identification of colonies of microorganisms with characteristics compatible with Actinomyces spp. The patient started treatment with intravenous penicillin for 2 weeks. Subsequently, she was medicated with amoxicillin 1g 8/8h for 12 months. She reported improvement in pain complaints. One year later, magnetic resonance cholangiopancreatography showed no bile duct changes. Duodenoscopy revealed the same endoscopic findings, however, the anatomopathological examination showed nonspecific chronic inflammatory changes without isolation of Actinomyces spp.
Discussion: The mechanisms of pathogenicity of Actinomyces species are not completely understood, but the invasion of breached or necrotic tissue has been proposed to be the initiating event for Actinomyces to penetrate and proliferate in deeper body structures. Although the prognosis of this infection normally is good with medical and surgical treatments, actinomycosis still can lead to the death of patients due to the difficulties of early diagnosis and the severe infection diffusion of the bacteria. Therefore, physicians must be aware of typical clinical presentations.

S1935

A Unique Case of Small Duct Primary Sclerosing Cholangitis Leading to Recurrent Cholestatic Hepatitis

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Introduction: Small Duct Primary Sclerosing Cholangitis (sdPSC) is the narrowing of intrahepatic bile ducts which often leads to cholestasis and cirrhosis. We present a patient diagnosed with sdPSC which is an uncommon form of PSC.
Case Description/Methods: A 42-year-old male patient, with a history of hypertension and gunshot wound with bowel resection presented for abdominal pain associated with recurrent cholangitis. Previous history also included bouts of abdominal pain, jaundice and hyperbilirubinemia that occurred roughly three times a year. A liver biopsy was taken during admission and revealed mild portal inflammation and cholestasis, bridging fibrosis, and intracytoplasmic iron granules. One month later a colonoscopy showed patchy erythema with biopsies demonstrating mild acute proctitis. Given cholestatic pattern, + ANA 1:

320, recurrent admissions with sepsis, and recent colonoscopy showing evidence of proctitis (biopsy proven), the patient was diagnosed with sdPSC with overlap of autoimmune cholangiopathy. The sdPSC was treated with a cadaveric liver transplant, and upon explant, the patient's liver showed chronic obstructive cholangiopathy. His postoperative course was notable for persistently elevated bilirubin of which half was indirect. Upon ERCP, the liver showed a mild anastomotic stricture and tortuous duct which was treated with a stent. However, the stent was removed because of lack of frank stricture. The patient was started on a prednisone taper with tacrolimus. The patient is currently doing well post-transplant on low dose tacrolimus and mycophenolate.
Discussion: Clinical presentation of sdPSC often consists of pruritis, jaundice, and acute cholangitis from cholestasis, and fatigue, liver failure and hepatosplenomegaly from liver cirrhosis. Notably, the patient presented with hepatomegaly, and jaundice. With this presentation, PSC, sdPSC, and benign recurrent intrahepatic cholestasis (BRIC) were all possible diagnoses. Unremarkable MRCP imaging highlighted the unlikeliness of PSC, and liver biopsy showed bridging fibrosis which excluded BRIC. Clinical diagnosis of sdPSC is difficult and a high degree of clinical suspicion is needed. The cause for Small Duct Primary Sclerosing Cholangitis is unknown, but there is an association with irritable bowel disease, presence of HLA-B8 and HLA-DR3 and autoimmune disease like hyper IgM.

S1936

Acute Pancreatitis Precipitated by Pancreatic Plasmacytoma

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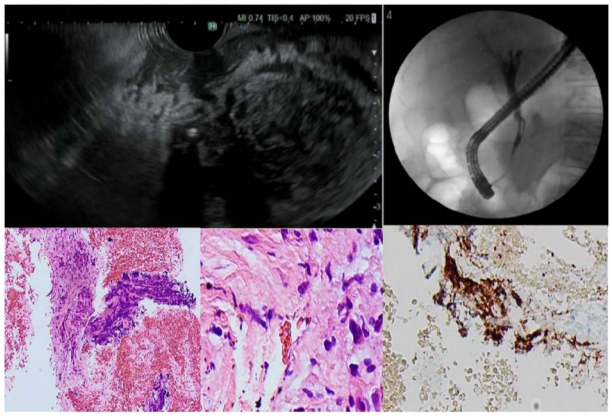
Introduction: Pancreatic plasmacytoma is rare entity of an extramedullary plasmacytoma defined as a plasma cell tumor occurring outside the bone marrow. It can present as a solitary plasma cell neoplasm but can also be associated with multiple myeloma. We present a case of pancreatic plasmacytoma in a patient with recurrent abdominal pain who initially presented with acute pancreatitis of unclear etiology.
Case Description/Methods: A 58-year-old female with a past medical history of multiple myeloma status post bone marrow transplant with relapse treated with pomalidomide presented with abdominal pain and failure to thrive. She was recently discharged from an outside facility treated for acute pancreatitis found to have an elevated lipase of 9675 U/L. Laboratory workup was remarkable for alkaline phosphatase (ALP) 532 IU/L, aspartate transaminase (AST) 475 IU/L, alanine transaminase (ALT) 422 IU/L, and bilirubin 1.4 mg/dl with concern of gallstone pancreatitis. Computed tomography (CT) scan of the abdomen and pelvis showed peripancreatic fluid collection which was further investigated with magnetic resonance cholangiopancreatography (MRCP) revealing a 6.6 x 5.1 x 7.0 cm large pancreatic head mass obstructing a 9.5 mm dilated common bile duct (CBD) and 8 mm dilated pancreatic duct. Due to concern for malignancy, endoscopic ultrasound (EUS) was performed and revealed a 6.0 x 5.2 cm irregular hypoechoic pancreatic lesion with fine needle biopsy (FNB) taken of the lesion. Endoscopic retrograde cholangiopancreatography (ERCP) demonstrated a distal CBD stricture and plastic stent was placed. Pathology results of the pancreatic mass revealed plasma cell neoplasm consistent with patient's history of multiple myeloma. The patient unfortunately passed away within 3 months of findings before further medical intervention (**Figure**).
Discussion: Acute pancreatitis is most commonly precipitated by alcohol use and gallstones but many cases have an unclear etiology. Literature suggests multiple myeloma is associated with acute pancreatitis but there is scarce data on pancreatic plasmacytoma precipitating acute pancreatitis. Typically, plasma cells cause microscopic infiltration of the pancreas and therefore, the presence of an obstructing pancreatic mass is unusual such as in this case. Our case highlights the importance of recognizing pancreatic plasmacytoma as a precipitant of acute pancreatitis due to its obstructive capability in patients with a long-standing history of relapsed multiple myeloma.



[1936] **Figure 1.** Top left: EUS showing pancreatic head mass. Top right: ERCP with distal CBD stricture Bottom row: H&E stain with neoplastic plasma cells and positive for CD 138.

S1937

Acute Pancreatitis Due to Vascular Insufficiency From Celiac Artery Stenosis

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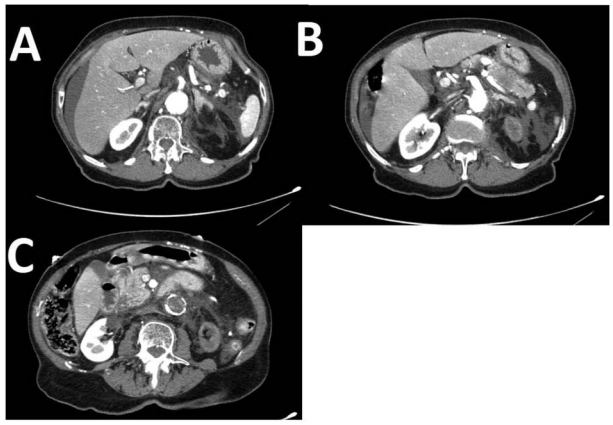
Introduction: Acute pancreatitis is a leading cause of hospitalizations in the United States. The pancreas relies on blood supply from the celiac and superior mesenteric arteries. Compromise to any part of the pancreatic circulation may lead to inflammation. Vascular insufficiencies are an uncommon cause of acute pancreatitis. We present a rare case of acute pancreatitis secondary to celiac stenosis with concomitant abdominal aortic aneurysm thrombosis.
Case Description/Methods: The patient is a 79-year-old female with history of tobacco use and peripheral vascular disease that presented for one day of abdominal pain. She noted nausea with non-bloody emesis during this time. Labs revealed a leukocytosis with WBC 17.1, lactate 2.7, AST 25, ALT 16, alkaline phosphatase 53, Total bilirubin 0.8, direct bilirubin 0.1, and a lipase of 976. A CTA of the abdomen was demonstrated inflammatory fluid with pancreatic tail necrosis, peripancreatic stranding, stenosis of the celiac origin and thrombosed aortic aneurysm measuring 6.1cm with distal reconstitution. She was placed on intravenous heparin and lactated ringer infusions. After the diagnosis of her acute pancreatitis, she was found to have a normal triglyceride level and IgG4 level. An abdominal ultrasound revealed gallbladder sludge without cholelithiasis or biliary dilation. There was no history of alcohol use preceding her hospitalization. Moreover, there were no new medications or over the counter supplements reported. It was felt the patient's pancreatitis was a result of arterial insufficiency from celiac artery stenosis. The patient was seen by vascular surgery and was treated medically. During her stay she developed worsening pleural effusion and hypoxic respiratory failure requiring oxygen. She was ultimately discharged to a skilled nursing facility when medically stable (**Figure**).
Discussion: Isolated case reports of acute pancreatitis due to vascular insufficiencies have been reported without defined incidence or prevalence. The head of the pancreas receives blood supply from the celiac trunk and superior mesenteric artery respectively. The body and tail of the pancreas are more vulnerable to ischemia, being perfused solely by the splenic artery. After ruling out common etiologies, compromise of the celiac artery was presumed to be the cause of this patient's disease process. Conservative treatment yielded satisfactory results in our case, however the standard of care regarding acute pancreatitis with vascular insufficiencies need additional elucidation.



[1937] **Figure 1.** A. Celiac artery stenosis. B. Pancreatitis with pancreatic tail necrosis and peripancreatic fluid and stranding. C. Thrombosis within distal aorta.

S1938

Acute Pancreatitis as a Consequence of COVID-19 Infection

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Introduction: Acute pancreatitis is a condition involving inflammation of the exocrine portion of pancreatic parenchyma. Although gallstones and alcoholism are the most common causes of acute pancreatitis, there have been a number of cases caused by infections. While the main manifestations of Covid-19 are respiratory, more and more systemic manifestations have come to light, including acute pancreatitis. While the exact mechanism of the relationship between the two conditions is unknown, one theory is that the virus attacks the angiotensin converting enzyme, which is heavily expressed on the pancreas.
Case Description/Methods: A 69-year-old female presents to the hospital after 1 week of epigastric abdominal pain. Patient described the pain as achy and radiating to the back. Patient also complained of nausea, loss of appetite, and dark stool. The patient was found to have a lipase of 668 units/L (reference range 14-63 unit/L), well above three times the upper limit of normal. She underwent CT of the abdomen, which showed peripancreatic stranding consistent with pancreatitis. After further inquiries, the patient denied any alcohol use and had a cholecystectomy in the past, the patient did however endorse having a viral illness 1 week prior to admission. Patient was tested for Covid-19 and was positive via PCR. Patient's triglycerides were within normal limits and IgG testing for autoimmune pancreatitis was negative. It was then surmised that the patient's recent Covid-19 infection was most likely the cause of the patient's acute pancreatitis. The patient received fluids, and was placed N.P.O. with instructions to advance the diet as tolerated. Unfortunately, the patient suffered a prolonged hospital course due to an acute right lower extremity DVT involving the right common femoral vein and saphenofemoral junction requiring mechanical thrombectomy due to continuing symptoms after medical therapy. The patient tolerated the procedure well, had significant improvement in nausea and abdominal pain, and was able to be discharged home.
Discussion: The true incidence of pancreatitis caused by Covid cannot fully be discerned as it often manifests with gastrointestinal symptoms which can disguise pancreatitis, and due to the highly transmissible nature of Covid leaving advanced imaging techniques which could reveal the cause of the pancreatitis rarely done. Nevertheless, when encountered with a Covid-19 patient presenting with abdominal pain, acute pancreatitis should always remain high on the differential.

S1939

Acute Necrotizing Pancreatitis as a Sequela of COVID-19 Infection

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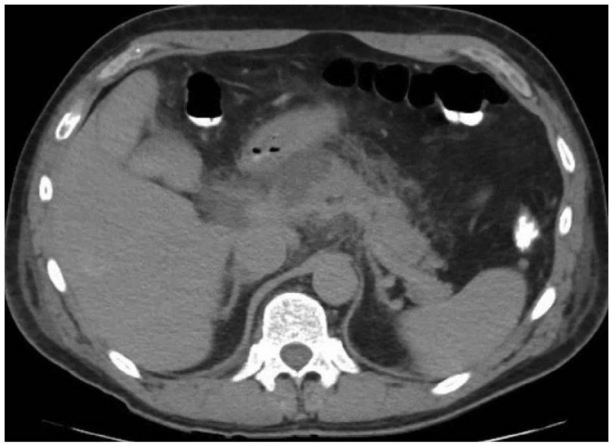
Introduction: The coronavirus disease 2019 (COVID-19) is an infectious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Although COVID-19 has been primarily affects the lungs, gastrointestinal (GI) involvement has also been reported. The GI manifestations of COVID-19 infection include anorexia, nausea, vomiting, abdominal pain, and diarrhea. We report a case of a 46-year-old male with no significant past medical history who developed acute necrotizing pancreatitis after the resolution of COVID-19 infection.
Case Description/Methods: A 46-year-old male with no past medical history presented to our hospital with epigastric abdominal pain. The patient denied any alcohol intake, smoking, or drug abuse history. The patient was discharged a week ago following the resolution of the COVID-19 infection. Vitals were stable on admission. Physical examination revealed severe epigastric tenderness and no signs of peritonitis. Clinical laboratory results were remarkable for elevated serum lipase levels >3000 U/L. Complete blood count, liver function, and lipid panel were within normal limits. Abdominal ultrasound showed no evidence of gallstones, cholecystitis, or intra or extrahepatic biliary dilation. Abdominal Computed Tomography (CT) with intravenous (IV) contrast showed peripancreatic fat stranding suggestive of acute pancreatitis (**Figure**). Magnetic resonance cholangiopancreatography was done, which confirmed the above findings. Autoimmune pancreatitis was ruled out with normal serum IgG-4 levels. Management included aggressive IV fluid therapy, antiemetics, and opioid analgesics. On day 3 of admission patient reported worsening abdominal pain. Labs showed worsening leukocytosis at 25,000. A repeat CT abdomen showed findings suggestive of acute necrotizing pancreatitis. The patient received one week of broad-spectrum antibiotics, reported resolution of symptoms, and was able to tolerate diet on the day of discharge.
Discussion: COVID-19 infection primarily affects the lungs; however, gastrointestinal involvement has also been reported. The mechanism of pancreatic injury in COVID-19 is due to the high expression of ACE2 receptors in the pancreatic cells. Glycosylated-spike protein of the virus binds to ACE2 receptor and mediates the host cell invasion causing cytopathic pancreatic harm. In conclusion, COVID-19-induced AP is possible and can present even after the resolution of viral infection, as seen in our case. COVID-19-induced AP is rare, and other common etiologies must be ruled out.



[1939] **Figure 1.** CT abdomen with contrast showing features suggestive of acute necrotizing pancreatitis

S1940

Acute Pancreatitis: Caused by Dislodged PEG Tube?

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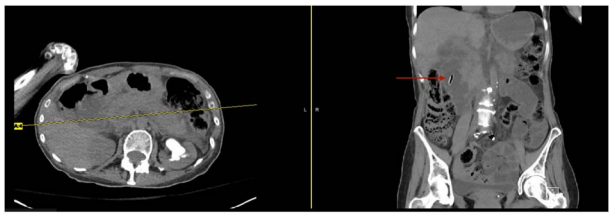
Introduction: Percutaneous endoscopic gastrostomy (PEG) is a common endoscopic procedure and a preferred route for long-term enteral nutrition for patients incapable of oral intake or for whom oral intake is insufficient. The insertion of PEG tubes is a relatively safe procedure with complications which include bleeding, aspiration, perforation, periostomal leaks, tube dislodgement and occlusion, gastric outlet obstruction due to tube migration and accidental removal. Acute pancreatitis secondary to PEG migration is rare. We present a patient with acute pancreatitis secondary to PEG tube migration.
Case Description/Methods: A 57-year-old woman known to have severe developmental delays, seizure disorder, bipolar 1 disorder presented with nausea, vomiting and abdominal pain. She is bedridden and nonverbal at baseline. She gets nutrition through a PEG tube for severe oropharyngeal dysphagia. Abdominal examination revealed significant biliary drainage from the gastrostomy site and tenderness present without rigidity or guarding. Labs showed WBC of 17.7 and lipase levels greater than 4,000 U/L. AST, ALT, alkaline phosphatase and total bilirubin were within normal range (31, 6, 94 and 0.9 respectively). CT abdomen without IV contrast revealed peripancreatic soft tissue stranding and pancreatic edema, the tip of the PEG tube in the second part of the duodenum (**Figure**). Her home medications are verified and are not known to cause acute pancreatitis (AP). So she was suspected to have AP from PEG tube migration. The tube was repositioned and she was treated with Ringer lactate. Four days later, her symptoms resolved and lipase level was within normal range (282 U/L).
Discussion: In comparison to other methods, enteral feeding has the benefit of maintaining gut mucosal integrity and decreasing the possibility for bacterial translocation. Migration of the PEG tube has been associated with gastric outlet obstruction and biliary obstruction. In rare cases, this can present as AP either through ampullary obstruction at the second part of the duodenum or through compression of the pancreatic head itself. This case highlights the inclusion of PEG tube induced pancreatitis as a differential for AP in patients receiving enteral feeding. Proper placement of the tube, regular examination of the site and marking the PEG position can help avoid migration and complications. The treatment for acute pancreatitis secondary to PEG tube migration is PEG tube removal followed by placement of a new PEG tube or repositioning.



[1940] **Figure 1.** Peripancreatic soft tissue stranding and pancreatic edema, the tip of the PEG tube in the second part of the duodenum.

S1941

Acute Pancreatitis Following a Lower Extremity Angioplasty

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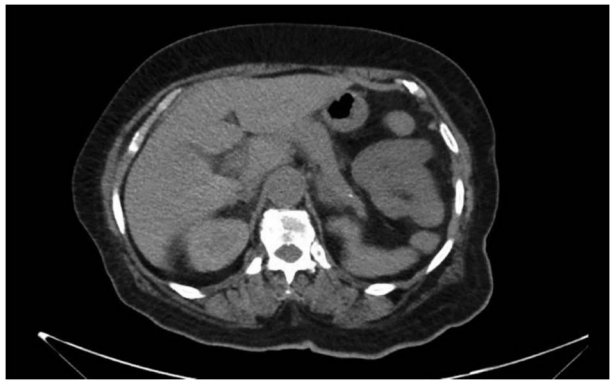
Introduction: Acute pancreatitis affects a significant population globally. Usual etiologies are gallstones, alcohol, hypertriglyceridemia, medications; less frequent are trauma, hypercalcemia, infections, toxins, ischemia, anatomic anomalies, vasculitis, and idiopathic. Pancreatitis post coronary intervention is an uncommon cause with only 19 published cases in the last two decades. Being cognizant of this etiology is important given the increasing number of patients undergoing angiography.
Case Description/Methods: An 81-year-old female with hypertension, diabetes, peripheral arterial disease, prior cholecystectomy underwent left lower extremity angioplasty at an outside center. Within a few hours, she started having severe epigastric pain radiating to her back, nausea, vomiting and loose bloody stool. She presented to the emergency department 24 hours after symptom onset. Epigastric tenderness was present on exam. Labs revealed leukocytosis (24,450/μL),elevated lipase (1410 U/L), elevated creatinine (1.3 mg/dL), lactate (3.1 mmol/L), calcium 9.4 mg/dL and triglycerides 161 mg/dL. Incidentally, found to be positive for COVID-19. Normal common bile duct diameter seen on sonogram. CT angiogram of the abdomen/pelvis showed acute pancreatitis, duodenal and central small bowel enteritis (**Figure**). She was not on any medications known to cause pancreatitis and denied alcohol use. Patient improved with analgesics and intravenous fluids. She had no recurrence of bloody stools and hemoglobin remained stable. On day 4, she was able to tolerate a regular diet, and leukocyte count and creatinine normalized. Patient did not have any COVID respiratory symptoms, and was discharged.
Discussion: Given the temporal association to angioplasty and no other identifiable cause, acute pancreatitis was presumed to be due to the contrast used during angioplasty. Other possibilities included cholesterol embolism but no peripheral signs of cholesterol embolism were seen. Patient was an asymptomatic COVID-19 case. Although, there are case series of pancreatitis due to COVID, those were found in very sick symptomatic patients. On review of literature, cholesterol embolism was identified as a definite cause only on autopsy or laparotomy (**Table**). Other possible mechanisms are: high viscosity of the contrast media leading to ischemia and necrosis, contrast causing NF-κB activation followed by epithelial damage, and vasospasm. Pancreatitis after coronary angiography is rare, nonetheless, an important differential especially if there is a temporal relationship.



[1941] **Figure 1.** CT scan (abdomen) of our patient demonstrating acute pancreatitis.

Table 1. Current literature reports on acute pancreatitis after coronary angiography/angioplasty

S. NO.	YEAR	AUTHOR	PROCEDURE	ETIOLOGY	TIME BETWEEN PROCEDURE AND SYMPTOM ONSET	CONTRAST USED	VOLUME OF CONTRAST(mL)
1	1994	Orvar et al[1]	Angiography	Cholesterol emboli detected on autopsy	14 hours	Not Mentioned	80-160
2	1994	Orvar et al[1]	Angiography	Cholesterol emboli detected on autopsy	Immediately	Not Mentioned	80-160
3	1994	Orvar et al[1]	Angiography	Contrast-Induced	Not Mentioned	Not Mentioned	80-160
4	1994	Orvar et al[1]	Angiography	Contrast-Induced	Not Mentioned	Not Mentioned	80-160
5	1994	Orvar et al[1]	Angiography	Contrast-Induced	Not Mentioned	Not Mentioned	80-160
6	1994	Orvar et al[1]	Angiography	Contrast-Induced	Not Mentioned	Not Mentioned	80-160
7	1994	Orvar et al[1]	Angiography	Contrast-Induced	Not Mentioned	Not Mentioned	80-160
8	1994	Orvar et al[1]	Angiography	Contrast-Induced	Not Mentioned	Not Mentioned	80-160
9	1994	Orvar et al[1]	Angiography	Contrast-Induced	Not Mentioned	Not Mentioned	80-160
10	1994	Orvar et al[1]	Angiography	Contrast-Induced	Not Mentioned	Not Mentioned	80-160
11	1994	Orvar et al[1]	Angiography	Cholesterol emboli detected on autopsy	Not Mentioned	Not Mentioned	80-160
12	1994	Orvar et al[1]	Angiography	Cholesterol emboli detected on autopsy	Not Mentioned	Not Mentioned	80-160
13	2013	Gorges et al[2]	Angiography	Contrast-Induced	1 hour	Iopamidol	120
14	2014	Abstract 343, Journal of Hospital Medicine[3]	Angioplasty	Contrast-Induced	Not Mentioned	Not Mentioned	Not Mentioned
15	2017	Hajimaghsoudi et al[4]	Angiography	Contrast-Induced	48 hours	Visipaque(Iodixanol)	100
16	2020	Rafiq et al[5]	Angioplasty	Contrast-Induced	Immediately	Ioversal	150
17	2020	Mui et al[6]	Angioplasty	Contrast-Induced	Immediately	Omnipaque(Iohexol)	120

A total of 19 cases were identified. However, only seventeen cases have been shown in the table. Due to inability to get permission for reuse, the remaining 2 cases have not been shown. 1 - Orvar K, Johlin FC. Atheromatous embolization resulting in acute pancreatitis after cardiac catheterization and angiographic studies. Arch Intern Med 1994;154:1755–61 2 - Gorges R, Ghalayini W, Zughaib M. A case of contrast-induced pancreatitis following cardiac catheterization. J Invasive Cardiol 2013;25:E203–4. 3 - An Unusual Source of Abdominal Pain.Abstract published at Hospital Medicine 2014, March 24-27, Las Vegas, Nev.. Abstract 343 Journal of Hospital Medicine, Volume 9, Suppl 2. https://shmabstracts.org/abstract/an-unusual-source-of-abdominal-pain/ 4 - Hajimaghsoudi M, Zeinali F, Mansouri M, et al. Acute necrotizing pancreatitis following coronary artery angiography: a case report. ARYA Atheroscler 2017;13:156–8. 5 - S1558 A Rare Case of Pancreatitis Secondary to Coronary Angiogram Dye. Rafiq, Rehan; Rafiq, Ahmad.The American Journal of Gastroenterology: October 2020 - Volume 115 - Issue - p S791doi: 10.14309/01.ajg.0000708280.94586.25 6 - Mui JJ, Shamavonian R, Thien KCP. Acute pancreatitis following coronary angiography: case report and review of contrast-induced pancreatitis. Int Surg J 2020; 7:870-2

S1942

Acetaminophen-Codeine-Induced Pancreatitis in a Young Male Patient

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Introduction: Drug-induced acute pancreatitis (DIAP) is a rare cause of acute pancreatitis. It is estimated to represent 0.1-2 % of all cases of acute pancreatitis and has been reported with various medications. However, DIAP secondary to acetaminophen-codeine is exceedingly rare.
Case Description/Methods: A 20-year-old male who underwent recent surgery for acetabular impingement syndrome presented to the ED with acute epigastric pain, nausea, and vomiting. He noted that 3 hours prior to the onset of symptoms, he had ingested one tablet of acetaminophen-codeine (300mg/30mg) for post-surgical pain. He reported prior use of the medication as needed for the past 7-days without adverse effects. He denied the use of alcohol. Initial exam was notable for significant epigastric tenderness. Laboratory analysis revealed leukocytosis of 17.9, lipase 1384 U/L, triglycerides 62 mg/dL, calcium 9.8 mg/dL, normal liver function enzymes, and undetectable serum alcohol level. Abdominal US was unremarkable for biliary and gallbladder pathology. MRCP was obtained and demonstrated pancreatic edema, a small amount of peripancreatic fluid, and a mildly prominent proximal common hepatic duct measuring 8mm. The patient was treated conservatively with IV fluids, bowel rest, morphine for analgesia, and was symptom-free within 24 hours.
Discussion: DIAP, also known as medication-induced acute pancreatitis (MIAP), represents an uncommon cause of acute pancreatitis. It should be considered after other common etiologies have been excluded. The mechanism of codeine-associated pancreatitis is thought to be due to transient sphincter of Oddi constriction with a predilection in patients who had previously undergone cholecystectomy. Symptoms often develop 2-3 hours after drug ingestion and have an overall milder course when compared to other etiologies of pancreatitis. Moreover, the mechanism of acetaminophen-induced pancreatitis is less clear. Although our patient used acetaminophen-codeine combination tablets, we speculate that codeine was the causative medication, as our patient previously used acetaminophen without any adverse symptoms. Furthermore, the time course of symptom onset and the rapid resolution of symptoms is more fitting with previously documented cases of codeine-associated pancreatitis. Acetaminophen-codeine is one of the most prescribed pain medications, and although rare, DIAP is a possible consequence of its use and should be known by its prescribers.

S1943

An Unusual Liver Tumor

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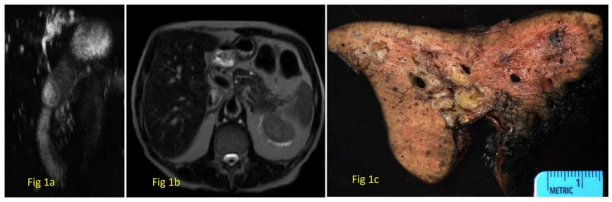
Introduction: Intraductal papillary neoplasm of bile duct (IPNB) is an uncommon premalignant biliary precursor lesion to intrahepatic cholangiocarcinoma (ICC). IPNB can be associated with intraductal papillary mucinous neoplasm (IPMN) of the pancreas. Von Meyenburg complexes (VMCs) have a strong association with hepatic fibro-polycystic liver disease. The association of all these rare entities has not been described, thus lending to the singularity of this case.
Case Description/Methods: A 69-year-old-male was referred for a 2-month history of right upper abdominal pain and 10-lb weight loss. CT showed left intrahepatic biliary ductal dilation and scattered small hepatic cysts. ERCP showed dilated left intrahepatic bile ducts with suboptimal brushings. MRI/CP (**Figure A**) revealed dilated left intrahepatic bile ducts with an abrupt cut off of the left hepatic duct and a subtle area of enhancement concerning for an obstructive malignancy. Multiple small side branch pancreatic IPMNs and many renal cysts were noted (**Figure B**). Due to concern for malignancy, a left hepatectomy was performed (**Figure C**). Intraoperatively, multiple cystic lesions were noted throughout the liver. Pathology revealed mucinous intestinal type IPNB with multiple VMCs. Tumor likely arose from a predisposing condition because of multiple VMCs, suggestive of fibro-polycystic liver disease in the non-involved liver. Patient did well post operatively.
Discussion: Four histologic types of IPNB have been described including pancreatobiliary, intestinal, gastric foveolar and oncocytic. There are 2 general categories of IPNB based on histologic similarities to their pancreatic counterpart. Type 1 shares many features with pancreatic IPMN. It develops in the intrahepatic bile ducts and is characterized by villous architecture, thin fibrovascular cores and frequent mucin production. Type 2 develops in the extrahepatic bile ducts and has complex papillary, polypoid, tubular, solid or cribriform intraductal structures. IPNB is considered premalignant. VMCs are thought to be a part of embryonic ductal plate malformation. Though largely innocuous, there has been a strong association with hepatic fibro-polycystic liver disease and in rare instances histological transformation to ICC. Prior case reports have tied IPNB to pancreatic IPMN, but this is the first case that associates IPNB, pancreatic IPMN, VMCs and cystic lesions of the liver and kidneys all together. We speculate there could be an underlying common pathological pathway that all these entities share.



[1943] **Figure 1.** a. Dilated left intrahepatic bile ducts with an abrupt cut off of the left hepatic duct. b. IPMN pancreas. c. Left hepatectomy showing tumor.

S1944

An Extraordinary Case of Gallbladder Small Cell Carcinoma

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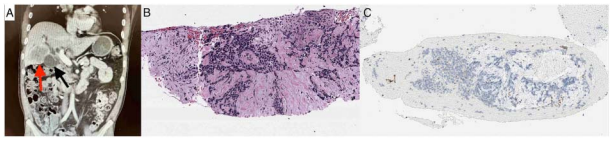
Introduction: We present a case of a patient diagnosed with Gallbladder Small Cell Carcinoma (SCC).
Case Description/Methods: This is a 58-year-old gentleman who presented to the emergency department complaining of pruritis. Associated with fatigue, appetite loss, and a 20-pound weight loss for three months. The physical exam was remarkable for scleral icterus and jaundice. Labs were remarkable for Total Bilirubin of 23.4 and Direct Bilirubin of 16.5. CT abdomen with IV contrast showed wall thickening of the gallbladder and infiltrates to his liver (**Figure** a). The patient had an Endoscopic retrograde cholangiography (ERCP) where the common hepatic duct contained single severe stenosis. One stent was placed into the common bile duct. A solid mass was found in the gallbladder, a biopsy was retrieved, and pathology confirmed Gallbladder SCC (**Figures** b and c). The patient was started on chemotherapy with carbo-etoposide. Another interval CT abdomen was obtained three months later which showed further invasion into segment four of the liver, new peritoneal metastasis, and increased biliary ductal dilation. The patient was subsequently started on FOLFIRINOX. Follow-up CT two months later after three rounds of chemotherapy showed stable hepatic lesions but increased intrahepatic ductal dilation. Interval CT abdomen two months later showed enlargement of hepatic segment four mass with involvement of segment five, peritoneal carcinomatosis, and new tumor implants along the anterior margin of the pancreas. The patient is currently on CAPTEM.
Discussion: Gallbladder SCC is an extraordinary finding as it has an incidence of .2% of all neuroendocrine tumors (1) with almost all cases either locally advanced or metastatic at the time of diagnosis. In Fuji et al's case series review, it was found that the one-and two-year survival rates of the 53 cases were 28% and 0% respectively (2). In patients with localized disease, surgical resection may result in prolonged survival. Chemotherapy is used to treat disseminated disease, although there are no clinical trials of a specific regimen given the scarcity of cases. Although incredibly rare, SCC of the gallbladder should be part of the differential diagnosis for gallbladder cancer.



[1944] **Figure 1.** A: CT abdomen with IV contrast showing gallbladder wall thickening (black arrow) and infiltrates into the liver (red arrow). B: Hematoxylin and eosin image of the gallbladder core needle biopsy showing high-grade neuroendocrine carcinoma involving fibrous stroma. Tumor cells are hyperchromatic with a high nucleus to cytoplasmic ratios and show evidence of molding and crush artifact. C: Core needle biopsy of the gallbladder immunohistochemical stain for RB1 shows loss of tumor cell nuclear expression of the Rb protein, characteristic for small cell carcinoma

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S1945

An Unusual Presentation of IgG4-Related Disease Mimicking Cholangiocarcinoma

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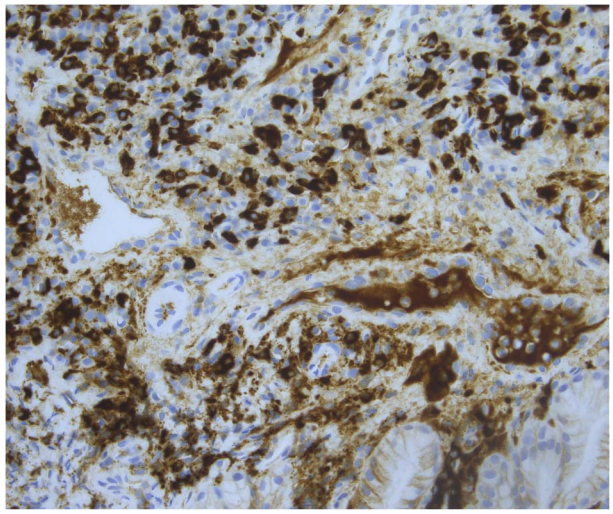
Introduction: IgG4-related disease (IgG4-RD) is a fibroinflammatory systemic autoimmune disease of unknown origin that can affect multiple organs. It affects the pancreatico-hepatobiliary system presenting either as sclerosing cholangitis, type 1 autoimmune pancreatitis (AIP), both, or rarely presents as IgG4-related hepatopathy. We present a challenging case of a patient with IgG4-RD.
Case Description/Methods: A 43-year-old man presented with painless jaundice and diarrhea for 1 month. Physical exam was relevant for scleral icterus with a negative Murphy's sign. Initial evaluation revealed alkaline phosphatase 237 and total bilirubin of 10.4. Diffuse intrahepatic biliary dilation with obstruction at the confluence of the common hepatic duct (CHD) was found on MRCP. Subsequent ERCP showed stenosis of the upper third of the right main bile duct. One 8.5 Fr by 15 cm transpapillary temporary stent was placed into the right hepatic duct. Biopsies showed normal ductal mucosa with no malignant cells. Within the next month, patient experienced worsening jaundice, elevated CA-19-9 (3995), serum IgG levels (2582), and total bilirubin (6.7). Repeat ERCP showed CHD stricture. CT abdomen showed increased

intrahepatic biliary ductal dilation and persistent soft tissue thickening at confluence of the right and left hepatic ducts. EUS showed normal pancreatic parenchyma and a 3cm mass at the peripheral area. Multiple biopsies were negative for cholangiocarcinoma (CCA). ERCP demonstrated nodular CHD, and biopsies of the biliary duct showed increased IgG4-positive plasma cells and an IgG4:IgG plasma cell ratio of 27%. After re-examination, it revealed an area with >20 plasma cells positive for IgG4, consistent with IgG4-related disease. The patient started on Prednisone 40mg once daily with a prolonged taper and improved significantly (**Figure**).

Discussion: IgG4 sclerosing cholangitis is the most common extrapancreatic manifestation of AIP type 1 presenting in >70% of patients. This case is suggestive of IgG4 sclerosing cholangitis without evidence of acute pancreatitis presenting as a hilar mass mimicking CCA. While biopsies failed to identify a neoplastic or infectious agent, it wasn't until biopsies were stained with IgG4 revealing the etiology of the disease. It is pertinent to be aware of the GI manifestations of IgG4-RD and to consider it in the differential when addressing unrelenting symptomatic jaundice. Early confirmation of >20 plasma cells HPF in biopsy avoids unnecessary procedures and allows for early treatment.



[1945] **Figure 1.** Immunohistochemical staining for IgG4 in a high power field showing markedly increased expression of IgG4 (up to 50 per high power field at 400X magnification). The upper portion of the image shows the positive plasma cells with cytoplasmic and membranous expression of IgG4

S1946

ANSA Pancreatica and Recurrent Acute Interstitial Pancreatitis: An Emerging Anatomic Variant

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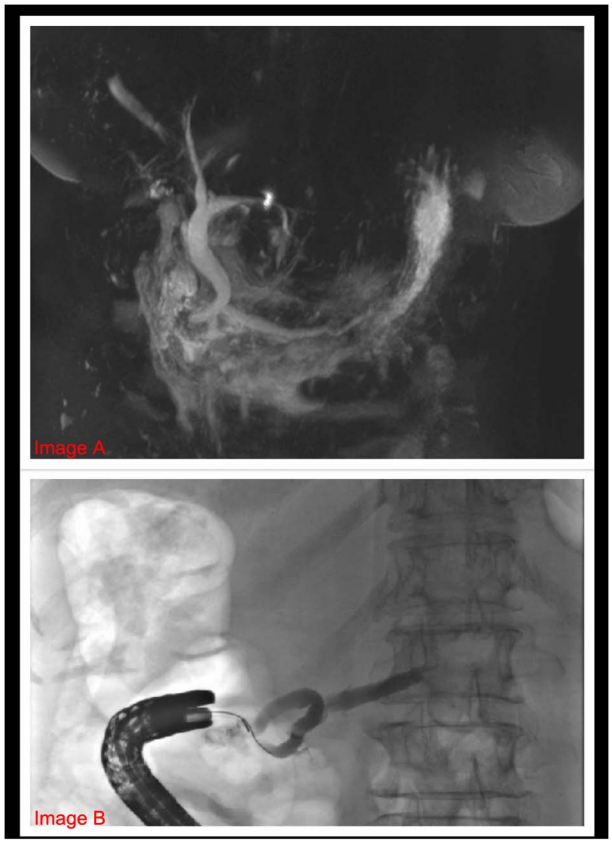
Introduction: Ansa pancreatica is a rare anatomic variant that has been associated with an increased risk of developing acute interstitial pancreatitis. We present a case of recurrent pancreatitis and ansa pancreatica diagnosed on endoscopic retrograde pancreatography (ERCP).

Case Description/Methods: Patient is a 76-year-old female with history of gallstone pancreatitis status-post cholecystectomy who presented with epigastric pain and associated nausea and vomiting. Patient denied alcohol use. Patient's lipase was 14,406 unit/L and triglyceride level was normal. A computerized tomography with contrast revealed evidence of acute interstitial pancreatitis, a dilated pancreatic duct of 7mm, concern for pancreatic duct stone, and intra- and extra-hepatic duct dilation. Patient underwent MRI of the abdomen with and without contrast which revealed pancreatic duct dilation and a pancreatic duct stone. There was irregular calcific appearing abnormality on the right lateral margin of the pancreatic head near the minor papilla. There were no imaging findings to suggest a mass lesion and no choledocholithiasis. The patient was medically treated with intravenous fluids, diet was advanced, and the patient was discharged with plans for outpatient ERCP. ERCP was performed 2 months after admission for acute pancreatitis. Pancreatic duct cannulation was achieved with a DASH spincterotome and revealed ansa pancreatica with a dominant draining dorsal duct. A filling defect was apparent in the duct of Santorini and a pancreatic sphincterotomy was performed at the major papilla. The minor papilla was cannulated and stone debris was removed following a minor papillotomy. Patient tolerated the procedure without incident. To date, the patient has not had a recurrence of their acute pancreatitis (**Figure**).

Discussion: We present a case of recurrent pancreatitis secondary to suspected ansa pancreatica. Ansa pancreatica is a rare anatomic variant, prevalence of 0.5-0.9%, described as an "S shaped loop" connecting the accessory pancreatic duct to the main pancreatic duct (1-6). There is an association between acute pancreatitis and ansa pancreatica, however, the mechanism is not well understood. This case highlights the anatomic variant specific to a dominant dorsal draining duct treated with a minor papillotomy and stone extraction. Further investigation is needed to address endoscopic cannulation of this anatomic variant and the association with acute pancreatitis.



[1946] **Figure 1.** Image A. MRCP demonstrating ansa pancreatica. Image B. ERCP demonstrating ansa pancreatica.

S1947

An Unusual Cause of Gastric Outlet Obstruction

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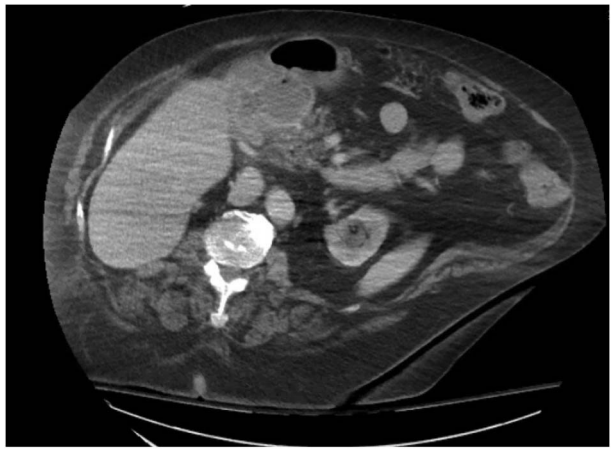
Introduction: Choledochoduodenal fistula (CDF) is a rare form of biliary tract fistula characterized by an abnormal connection between the common bile duct and duodenum. It is associated with duodenal ulcers, cholelithiasis, choledocholithiasis, and even malignancy. Given its low prevalence, understanding of CDF pathology remains limited.
Case Description/Methods: A 75-year-old woman with hypertension, diabetes mellitus, osteoarthritis (OA), and stage IIIb right-sided colon cancer (in remission after FOLFOX and right hemicolectomy 3 years prior) presented to the hospital with one day of nausea and vomiting. She reported 20 episodes of coffee-ground emesis and taking up to 8 200mg ibuprofen daily for months to treat her OA. Labs were notable for hemoglobin of 8.7 g/dL without known baseline. She was started on intravenous pantoprazole given concern for upper gastrointestinal bleed. Esophagogastroduodenoscopy revealed 1.5 liters of retained non-bloody fluid with obstructing objects in the duodenal bulb immediately distal to the pylorus that were unable to be removed and adjacent ulcerations not amenable to endoscopic therapy. A nasogastric tube was placed for decompression. The obstructing bodies were initially thought to be bezoars, but later identified as gallstones. Abdominal CT demonstrated a CDF with adjacent abscess (**Figure**). The patient underwent exploratory laparotomy, gastrotomy, gastroduodenoscopy with morcellation and removal of gallstones, and successful decompression of the duodenum. The CDF was visualized with a free-flowing stone in the lumen. Post-operatively, the patient had resolution of nausea and vomiting and was discharged tolerating an oral diet.
Discussion: CDF is usually an incidental and asymptomatic finding. When symptomatic, they commonly present with cholangitic symptoms such as fever, abdominal pain, and jaundice. Rarely, patients can present with Bouveret Syndrome, a clinical process describing gastric outlet obstruction in the setting of gallstone ileus and CDF. Nausea, vomiting, and abdominal pain are common presenting symptoms, but hematemesis and melena have also been described. There is no standardized management, but the treatment goal involves removing the culprit obstruction either via endoscopic or surgical retrieval. Symptomatic CDF patients are at risk for recurrence, although ongoing debate exists on risk-stratifying candidates for CDF repair based on location versus size of fistula. Proper management of symptomatic CDF is important for preventing further morbidity.



[1947] **Figure 1.** Abdominal CT demonstrating choledochoduodenal fistula and associated abscess

S1948

An Unusual Case of Jaundice Secondary to Hilar Lymphoma

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Introduction: We present a unique case of jaundice and biliary stricture with pathologic evaluation revealing high-grade B-cell non-Hodgkin's lymphoma.
Case Description/Methods: A 70-year-old female with a history of breast cancer presented with jaundice, abdominal pain and vomiting with a total bilirubin of 2.6 mg/dl in July of 2020. Serum testing revealed elevated CA 19-9 levels. Endoscopic retrograde cholangiopancreatography (ERCP) was performed and attempts at stent placement resulted in bile duct perforation. A stent was placed, and cholecystectomy was performed. Computed tomography (CT) imaging revealed soft tissue thickening surrounding the common bile duct and encasing the proper hepatic, right and left hepatic arteries, and the main portal vein. She was readmitted a month later after developing further jaundice. Bile duct brush cytology revealed atypical epithelial cells. Fine Needle Aspiration biopsy (FNAB) revealed no malignant cells. Endoscopic ultrasound revealed a mass measuring 39 mm by 28 mm. Repeat ERCP demonstrated persistent hilar stricture prompting stent exchange. Spyglass catheterization was unsuccessful due to ampullary stricture. Repeat FNAB of the common bile duct confirmed high-grade B-cell non-Hodgkin's lymphoma. The mass was diffusely positive for CD20 and PAX-5 with high Ki67/MIB-1 proliferation index. CT imaging demonstrated an infiltrating hypoenhancing mass within the hepatic hilum. In December of 2021, the patient presented with ascites. Peritoneal fluid cytology revealed malignant lymphoma with monoclonal B-cell proliferation with a positive antigen profile of CD19, CD45, and Lambda. She was treated with two cycles of R-CHOP (rituximab, cyclophosphamide, doxorubicin hydrochloride, vincristine, and prednisolone) followed by two cycles of Rituximab alone. She experienced life-threatening arterial hemobilia that was noted during an ECRP.
Discussion: Non-Hodgkin's Lymphoma represents only 1-2% of biliary obstruction in adults. It is rare for the bile duct to be involved in cases of non-Hodgkin's lymphoma and even more unusual to have lymphomatous obstruction of the biliary system. Although it is difficult to differentiate lymphomas from other common malignancies that impact the biliary system, it is imperative to make the distinction as lymphoma can be much more responsive to radiation and chemotherapy. Chemotherapy poses bleeding risks especially in lymphomas invading biliary trees.

S1949

An Unusual Case of Chronic Pancreatitis Presenting With Recurrent Obscure Gastrointestinal Bleeding

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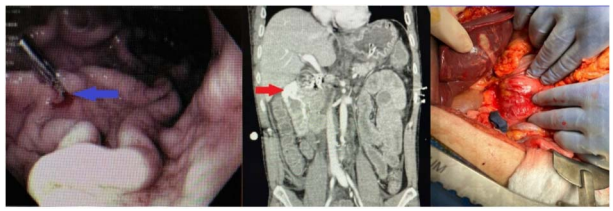
Introduction: Chronic pancreatitis (CP) is a sequela of repeated pancreatic injury resulting in loss of exocrine and endocrine function of the pancreas. In most cases, CP presents with chronic abdominal pain and patients usually have either clinical and radiological features of CP, with gastrointestinal bleeding (GIB) as a very rare occurrence. Herein is a presentation of recurrent obscure GIB caused by chronic pancreatitis.
Case Description/Methods: A 48-year-old male with a prior history of PUD and recurrent GIB episodes for several years presents with dark red bleeding per rectum for two days. Of note, the patient had undergone several endoscopies and the most recent endoscopies done six months prior were all unremarkable. He denied hematemesis, NSAIDs, or anticoagulant use. Denied liver or pancreatic disease but endorsed a prior history of moderate alcohol intake. Physical examination was unremarkable except for tachycardia and epigastric tenderness. He was mildly anemic with leukocytosis. The abdominal and pelvic CT scan was unremarkable. EGD revealed a small gastric oozing site (**Figure)** suspected to be an AVM or dieulafoy's lesion and clipped. He continued to have a frank bloody stool and developed hemorrhagic shock requiring multiple transfusions and vasopressors. Findings on two additional EGDs were unremarkable. He had mesenteric angiography performed with ligation of the gastroduodenal artery empirically. Despite the intervention, he continued to bleed. Abdominal CTA (**Figure)** noted active extravasation in the 2nd segment of the duodenum. Explorative laparotomy revealed an edematous head and uncinate portion of the pancreas with venous engorgement. There was erosion into the duodenum with acute hemorrhage (**Figure)**. A duodenotomy and Roux-en-y (duodenojejunostomy and jejunojejunostomy) were performed. On account of the above findings, a tissue biopsy was not obtained. Following surgery, he remained hemodynamically stable and got discharged.
Discussion: Massive obscure GIB due to complications of pancreatitis rarely does occur. A few cases reported are due to complications resulting from hemosuccus pancreaticus, pseudoaneurysm from a vascular supply, or erosion into adjacent viscus in the setting of known pancreatitis. Our patient represents an unusual presentation in which his recurrent obscure massive GI bleeding was the initial presentation of CP. We present this case to broaden our knowledge that obscure GI bleeding can be a complication of CP and the only manifestation of the disease.



[1949] **Figure 1.** The image on the left shows an EGD with the gastric oozing site with clips, and the middle image shows CTA with extravasation in the second segment of the duodenum. The image on the right shows the posterior aspect of the head of the pancreas, the uncinate, and venous engorgement.

S1950

An Unusual Case of Large Bowel Obstruction Secondary to Metastatic Pancreatic Adenocarcinoma

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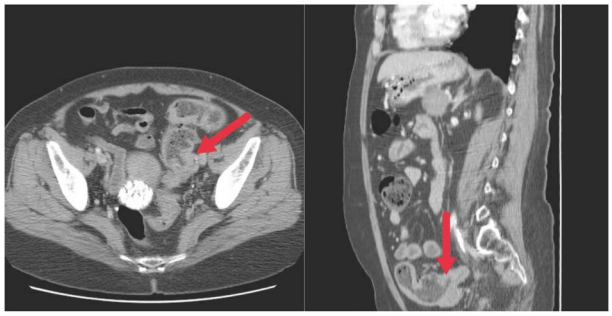
[1]Allama Iqbal Medical College, Sarnia, ON, Canada; [2]FMH College of Medicine & Dentistry, Lahore, Punjab, Pakistan; [3]AdventHealth Orlando, Orlando, FL; [4]Methodist Dallas Medical Center, Dallas, TX; [5]Allama Iqbal Medical College, Mississauga, ON, Canada.

Introduction: Pancreatic adenocarcinoma, though a relatively uncommon malignancy, is one of the leading causes of cancer mortality. Rarely it metastasizes to the large bowel to present as intestinal obstruction, complicating its diagnosis. Herein we report a case of pancreatic adenocarcinoma presenting as intestinal obstruction due to sigmoid metastasis in a female patient.
Case Description/Methods: A 60-year-old female with a past medical history of diabetes presented with constipation for 2 weeks and obstipation for 1 day associated with left lower quadrant abdominal pain. On admission, vital signs were stable and physical examination revealed normal bowel sounds and mild tenderness in the left lower quadrant. CT abdomen revealed an obstructing sigmoid tumor with concerning pancreatic and liver lesions. Subsequently, the patient underwent exploratory laparotomy with en bloc sigmoid colectomy with primary colorectal anastomosis. Pathology report revealed poorly differentiated adenocarcinoma involving the colonic muscularis propria, pericolonic adipose tissue, serosa, and involvement of 5 out of 14 lymph nodes. Since immunohistochemical markers were more consistent with metastatic disease from a pancreatobiliary primary rather than a colorectal primary, an EUS/FNA of the pancreatic lesion was performed, revealing pancreatic adenocarcinoma and confirming the pancreas as the site of primary malignancy. MRI abdomen further elucidated an infiltrative mass at the body and tail of the pancreas with adjacent vessel encasement. Due to the involvement of the celiac axis exceeding 180 degrees, the tumor was deemed unresectable, and patient was started on FOLFIRINOX chemotherapy (**Figure).**
Discussion: Pancreatic adenocarcinoma (PAC) is a rare but ominous diagnosis with less than 20% of patients presenting with an operable tumor and a 5-year survival approaching 5% [3]. PAC rarely presents as large bowel obstruction with 7 cases reported so far [4]. It can therefore be misdiagnosed as primary colorectal cancer. Knowing the true diagnosis beforehand in such a case can guide management, as palliative chemotherapy regimens, instead of local resection of colorectal disease, would be initiated if the pancreatic primary were first identified [5]. In our case, resection of the sigmoid mass was done to relieve symptoms of acute intestinal obstruction. Since patients with PAC are diagnosed after the disease has spread, palliative chemotherapy remains one of the few viable options for treatment.



[1950] **Figure 1.** Focal transmural thickening of the sigmoid colon wall with associated luminal narrowing (apple core lesion).

S1951

An Uncommon Case of Smoldering Pancreatitis

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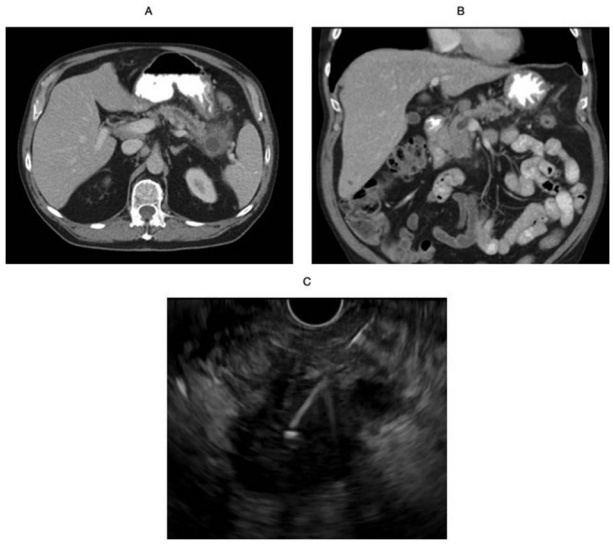
Introduction: We describe a rapid onset, poorly differentiated, invasive pancreatic carcinoma with histiocytoid features diagnosed post-pancreaticoduodenectomy, after endoscopic ultrasound (EUS) guided fine needle biopsy failed to identify it.
Case Description/Methods: A 69-year-old male with a history of alcohol abuse, and tobacco use, initially presented with epigastric pain and early satiety, diagnosed with acute pancreatitis. Computed tomography (CT) scan and Magnetic resonance imaging (MRI) of the abdomen/pelvis at this time were significant for a cystic lesion in the tail of the pancreas with fat stranding (**Figure** A). EUS was performed however had extremely limited visibility given inflammatory changes, main pancreatic ductal dilation was noted but no clear mass was identified. Fine needle aspiration (FNA) of the cystic structure was suggestive of a pseudocyst on cytology (CEA 25 ng/ml, amylase 3250 units/L). A portal lymph node was appreciated, and fine needle biopsies (FNBx) yielded only inflammatory cells. Clinical course continued to deteriorate over the next few months with non-resolving smoldering pancreatitis symptoms and >80 lb weight loss. Repeat CT abdomen/pelvis was significant for a 2.7 x 2.1 cm lesion of the head of the pancreas (**Figure** B). EUS was repeated and revealed a 32 x 29 mm hypoechoic mass in the head of the pancreas with upstream pancreatic ductal dilation to 13 mm with significant parenchymal atrophy as well as an enlarged porta hepatis lymph node. FNBx were obtained and were sent for analysis at a tertiary care center which revealed chronic inflammation with necrohistiocystic debris with atypical cells at both sites (**Figure** C). Due to clinical deterioration and after multidisciplinary discussion, patient underwent a pancreaticoduodenectomy for smoldering pancreatitis involving the head of pancreas and concerning atypical cells. The final pathology report revealed a poorly differentiated, invasive pancreatic carcinoma with histiocytoid features that extensively involved 6/16 regional lymph nodes after multiple reviews by multiple pathologists.
Discussion: To our knowledge, poorly differentiated, invasive pancreatic carcinoma with histiocytoid features has not been previously documented. This presentation with smoldering pancreatitis significantly delayed the diagnosis due to the marked inflammatory changes compromising initial imaging (CT, MRI, EUS). This case highlights the importance of a multidisciplinary approach in managing complicated non resolving pancreatitis cases.



[1951] **Figure 1.** A - Cystic lesion with inflammatory changes near the tail of the pancreas; B - Mass in the head of the pancreas with upstream pancreatic duct dilation; C - Endoscopic Ultrasound with fine needle biopsy of the mass in the head of pancreas

S1952

An Unexpected Presentation of Synchronous Malignancies

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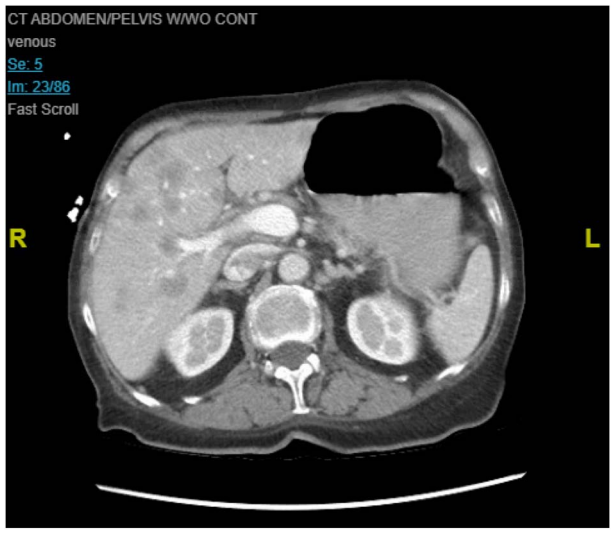
Introduction: Pancreatic neuroendocrine tumors (NETs) are rare malignancies, accounting for approximately 1-2% of all pancreatic cancers. There have been instances of synchronous carcinoid tumors or various other gastrointestinal NETs with concurrent secondary primary malignancies, however, there have not been previously documented cases of a pancreatic NET with concomitant colon adenocarcinoma presenting as acute pancreatitis.
Case Description/Methods: 73-year-old Caucasian female with no medical history, presented for 1 day of worsening right-sided abdominal pain. The pain began suddenly with no inciting event and she also reported an approximately 20-pound weight loss over the preceding months. Initial labs showed the patient had an elevated lipase, and computerized tomography (CT) of the abdomen/pelvis demonstrated extensive liver lesions consistent with metastatic disease (**Figure**). Additionally on the CT, the tail of the pancreas had a dilated pancreatic duct with a normal duct size in the proximal duct, indicating a pancreatic duct cut-off sign. Gastroenterology was consulted for further evaluation and an EGD with EUS was performed the following day revealing a 2cm x 3cm ill-defined mass in the head of the pancreas that was hypoechoic and heterogeneous in appearance. Multiple hypodense lesions were noted throughout the liver, and no pancreatic ductal dilatation was noted. Pathology revealed findings consistent with well differentiated grade 2 neuroendocrine tumor and was positive for CK-CAM5.2, CK7, CD56, chromogranin and synaptophysin, along with a KI-67 close to 20%. Tumor markers were also notable for a slightly elevated CA19-9 and a normal CEA. Colonoscopy was performed and notable for a 3cm descending colon polyp, which was later revealed to be well-differentiated adenocarcinoma with invasion into the submucosa. The patient was discharged with instructions to follow up outpatient with Hematology/Oncology, Surgical Oncology, and Gastroenterology, and is currently in the process of undergoing treatment.
Discussion: There are many causes for acute pancreatitis, with the most common being gallstones, alcohol, and medication-induced. A less frequently seen etiology is malignancy, and among the different types of pancreatic neoplasms, NETs are among the rarest. Although synchronous NETs and secondary primary malignancies have been documented, no cases have presented as acute pancreatitis. Prompt imaging, endoscopic procedures, and biopsies should be obtained in patients with potential NETs to expedite treatment.



[1952] **Figure 1.** CT abdomen/pelvis showing extensive liver lesions consistent with metastatic disease

S1953

An Unexpected Diagnosis in an Elderly Patient Presenting With Jaundice

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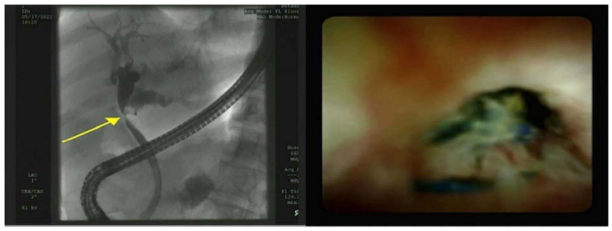
Introduction: Cholangioscopy is an important tool in the evaluation of indeterminate biliary strictures allowing for direct visualization of strictures and targeted biopsies. We present an unusual case of painless jaundice where cholangioscopy played a crucial role in making the diagnosis.
Case Description/Methods: A 76-year-old male with a past medical history of hypertension and diabetes mellitus, and a past surgical history of a laparoscopic cholecystectomy ten years prior to presentation was referred for one week of painless jaundice, darkening of urine, and pruritus. He denied any weight loss, abdominal pain, nausea, or vomiting. Laboratory evaluation was significant for a total bilirubin of 14.8, direct bilirubin of 7.6, AST 40, ALT 56, and alkaline phosphatase of 128. Magnetic resonance cholangiopancreatography was obtained, which revealed a poorly defined soft tissue lesion at the junction of the intrahepatic and extrahepatic common hepatic duct (CHD) with intrahepatic ductal dilation (IHD). Multiple small filling defects within the dilated ducts were also noted, suggestive of choleliths. The patient was referred for endoscopic ultrasound (EUS) and endoscopic retrograde cholangiopancreatography (ERCP). EUS revealed IHD and a benign appearing porta hepatis lymph node with no endosonographic evidence of pancreatic mass or cysts. The lymph node at the porta hepatis was sampled using fine needle aspiration (FNA). ERCP was then performed, revealing a single stenosis at the level of the CHD with IHD. Cholagioscopy was performed, which revealed an inflammatory-appearing stenosis of the CHD with embedded suture material and a cholelith proximal to the stenosis (**Figure**). The stenosis was biopsied and dilated with a 4 mm balloon dilator. An 8.5 x 10 cm plastic biliary stent was placed with subsequent flow of bile. FNA cytology and pathology of the CHD stricture were both negative for malignancy.
Discussion: Our patient likely sustained a bile duct injury during his prior cholecystectomy, which was repaired with suture, leading to an inflammatory stricture and obstructive jaundice. We had a high suspicion pre-procedure for malignancy given this patient's presentation, and as such cholangioscopy was crucial in determining the etiology of this patient's stricture. Our case highlights the importance of cholangioscopy in the evaluation of indeterminate biliary strictures, particularly in patients whom have undergone biliary surgery.



[1953] **Figure 1.** Left: Cholangiogram revealing hilar stricture. Right: Cholangioscopy revealing inflammatory stricture with embedded suture material.

S1954

An Unusual Location of Pseudoaneurysm in the Setting of Acute Severe Pancreatitis

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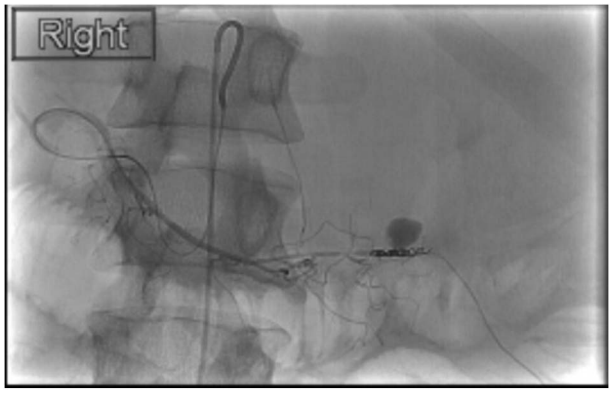
Introduction: Acute pancreatitis (AP) is one of the most common gastroenterological causes of hospital admissions. The mortality rate of AP is around 10% despite medical advancements. Around 4-17% of cases of acute pancreatitis are complicated by the formation of pseudoaneurysms. The most commonly involved arteries are splenic artery (60-65%), gastroduodenal (20-25%), pancreaticoduodenal (10-15%), hepatic (5-10%) and left gastric arteries (2-5%). We present a case of superior mesenteric artery (SMA) pseudoaneurysm secondary to acute severe pancreatitis.
Case Description/Methods: A 33-year-old male patient with a past medical history of alcohol abuse presented to the hospital for abdominal pain after an alcohol binge. He was found to have significant acute kidney injury, metabolic acidosis, and leukocytosis to 24 K/uL. CT showed acute pancreatitis and a large peripancreatic collection consistent with hemorrhage with posterolateral extension into the left iliopsoas space. He was found to have staphylococcus aureus bacteremia thus he was initially treated with fluid resuscitation and cefazolin. Several days into the admission he had an episode of hemodynamic instability with a CT showing a 7 mm pseudoaneurysm seen within the collection with active contrast extravasation. His hemoglobin dropped 2g during this episode requiring pRBC transfusion. He underwent IR coil embolization of the middle colic branch of the SMA (**Figure**). During a subsequent admission, he had worsening abdominal pain and distension with radiographic evidence of enlarging collections so a drain was placed into the peripancreatic collection by IR with improvement in symptoms.
Discussion: Pseudoaneurysm formation is an uncommon but life-threatening complication of pancreatitis. Pancreatic enzymes released in the setting of pancreatic inflammation leak into vessel walls causing destruction. The majority develop in the splenic artery given the proximity to the pancreas. Those in the SMA account for just 5% of cases. CT angiography is the best diagnostic modality for assessing pseudoaneurysm arterial bleeding. Treatment in the past was primarily surgical but with endovascular advancements, embolization with coils or glue is now first line. However, many patients may still require surgery if embolization fails or if hemodynamically unstable. Despite the rarity of pseudoaneurysm formation, it is important to have knowledge of the appropriate diagnostic workup and treatment given the potentially high mortality rate.



[1954] **Figure 1.** IR coil embolization of the middle colic branch of the SMA

S1955

An Atypical, Silent Spread of Gallbladder Cancer

Rami Abusaleh, MD[1], Chinonso Ilo, MD[1], Aida Rezaie, MD[1], Melinda Wang, MD[1], Indu Srinivasan, MD[2], Keng-Yu Chuang, MD[2].
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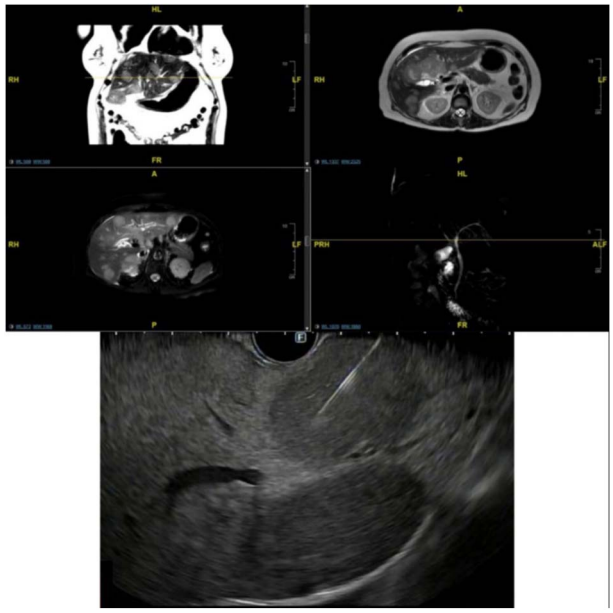
Introduction: Gallbladder cancer (GBC) though rare is the most common biliary cancer. It is associated with a poor prognosis due to delayed diagnosis and nonspecific clinical presentation. We present a case of a 62-year-old female that was incidentally found to have metastatic gallbladder cancer with no alarm symptoms on presentation.
Case Description/Methods: A 62-year-old female with diabetes mellitus II, obesity, and cholelithiasis presented to the emergency room with vague complaints of lower abdominal pain. Lab work was grossly normal except elevated white blood cell count of 16.1 K/uL. The pain resolved with bowel movement, however initial CT scan obtained was concerning for gall bladder mass. Tumor markers were obtained. Alpha-fetoprotein was 94.63, carcinoembryonic antigen was 13.17, and cancer antigen 19-9 was 24 within normal limits. Abdominal Magnetic resonance imaging (MRI) done for further evaluation showed an irregular mass from the gallbladder fundus measuring 5.0 x 8.8 x 5.9 cm along with numerous peripherally enhancing and diffusion restricting irregular lesions throughout the hepatic parenchyma consistent with metastatic disease. The patient underwent endoscopic ultrasound with fine-needle biopsy of the lesions and pathology was consistent with poorly differentiated adenocarcinoma of gallbladder primary (**Figure**).
Discussion: Gallbladder cancer (GBC) is an aggressive malignancy that is usually diagnosed at an advanced stage and thus carries a poor prognosis with a 5-year survival rate of less than 5% and with a median survival of less than 6 months. Surgical resection in early cancers can be potentially curative. Early diagnosis and treatment are essential however GBC presents a diagnostic and therapeutic challenge due to its vague, and in this case fleeting, symptom presentation. This case presentation shows that in patients with certain risk factors including cholelithiasis, obesity, and female gender consideration should be given to obtaining imaging studies to evaluate for atypical presentations of GBC.



[1955] **Figure 1.** Abdominal MRI demonstrating an irregular mass from the gallbladder and numerous peripherally enhancing lesions throughout the liver consistent with metastasis (top 4 panels). EUS-guided fine needle biopsy of the gallbladder mass suspicious for malignancy (bottom panel).

S1956

An Unsuspecting Mimicker: Pancreatic Metastases Imitating as an Autoimmune Pancreatitis

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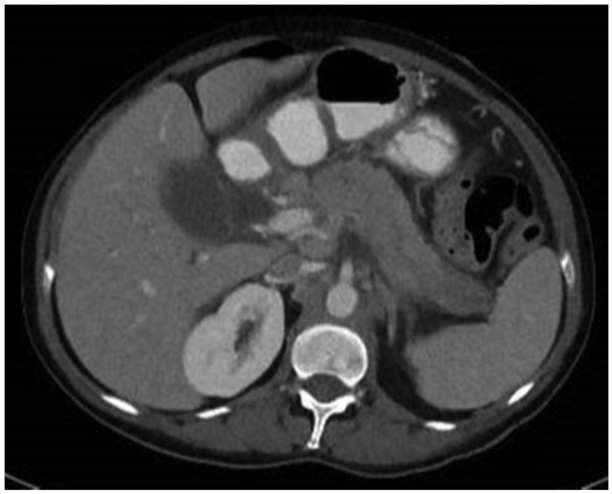
Introduction: Auto-immune pancreatitis (AIP) is an uncommon cause of recurrent pancreatitis characterized by chronic inflammation with lymphocytic infiltration on histology. It is further classified as type 1, IgG4 related and type 2, idiopathic duct-centric type, with a prevalence of 2% of chronic pancreatitis, affecting less than 1 per 100,000. We describe a case suspicious for AIP with pancreatic biopsy revealing metastatic lung adenocarcinoma.
Case Description/Methods: A 56-year-old female, former 10 pack year smoker with no medical history presented with complaints of worsening abdominal pain for one day. She notes a recent stay at an outside hospital for abdominal pain 5 days prior and was managed for gallstone pancreatitis. There, she underwent an ERCP with removal of biliary sludge and was planned for outpatient cholecystectomy. On this admission, a CT Abdomen/Pelvis revealed diffuse pancreatic enlargement with fullness most prominent at the head but no identifiable mass (**Figure**). Additionally, multiple enlarged retroperitoneal and mesenteric lymph nodes (LNs) were noted, measuring up to 2.5 cm. Radiographic findings were concerning for AIP versus malignancy. Labs were significant for a lipase 1078 and ANA 1:360 and subsequently managed for acute pancreatitis. IgG4 was at the upper limit of normal, 96 (2-96). On Day 5, she underwent an EGD/EUS, which revealed an enlarged pancreas, with no discrete mass as well as several mesenteric LNs, which were biopsied. Pathology revealed metastatic adenocarcinoma consistent with lung primary, PDL1 positive. An outpatient CT Chest revealed few pulmonary micronodules of unclear significance and borderline sized mediastinal LNs. She is currently following outpatient with thoracic oncology and undergoing further staging studies prior to initiation of treatment.
Discussion: Our patient presenting with clinical and radiographic features of AIP was diagnosed with metastatic lung cancer, a rare presentation of infiltrative metastasis mimicking autoimmune pancreatitis. While lung cancer is the 2nd leading cause of pancreatic metastasis, the literature has reported a few cases of lung cancer presenting as pancreatitis. Interestingly, 2 Japanese studies reported close temporality between AIP Type 1 diagnosis and cancer diagnosis, particularly with lung, stomach and prostate cancer, proposing the possibility that AIP can occur as an autoimmune paraneoplastic disease. Therefore, malignancy must be considered when evaluating for AIP and portends the necessity of endoscopic biopsy.



[1956] **Figure 1.** Enlarged heterogeneous pancreas, surrounding fat stranding and upper abdominal lymphadenopathy, suspicious for malignancy with co-existing pancreatitis.