

**Bones in Patients With IBD**

**Gary R. Lichtenstein, MD**  
Professor of Medicine

University of Pennsylvania School of Medicine  
Director, Center for IBD,  
Hospital of the University of Pennsylvania  
Philadelphia, PA

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**Aims of This Lecture**

1. Establish the scope of the problem of decreased bone mineral density (BMD) in the general population and in patients with inflammatory bowel disease (IBD)
2. Establish the degree of increased risk of BMD loss in patients with Crohn's disease (CD)
3. Differentiate the risks associated with conventional corticosteroids from those associated with EC Budesonide
4. Review established guidelines for the screening, prevention, and treatment of bone loss in patients with IBD
5. Review evidence-based treatments for BMD loss in patients with IBD
6. Draw conclusions applicable to clinical practice

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**Osteoporosis in the General Population**

	BMD (g/cm <sup>2</sup> )			
	Lumbar	Neck	Trochanter	Total Body
Age (yr)	-0.491 ( <i>P</i> < 0.001)	-0.535 ( <i>P</i> < 0.001)	-0.391 ( <i>P</i> < 0.01)	-0.632 ( <i>P</i> < 0.001)
Weight (kg)	0.142 (NS)	0.238 ( <i>P</i> = 0.05)	0.240 ( <i>P</i> = 0.05)	0.119 ( <i>P</i> = NS)
Hormonal state*	-0.461 ( <i>P</i> < 0.001)	-0.432 ( <i>P</i> < 0.001)	-0.311 ( <i>P</i> < 0.05)	-0.601 ( <i>P</i> < 0.001)

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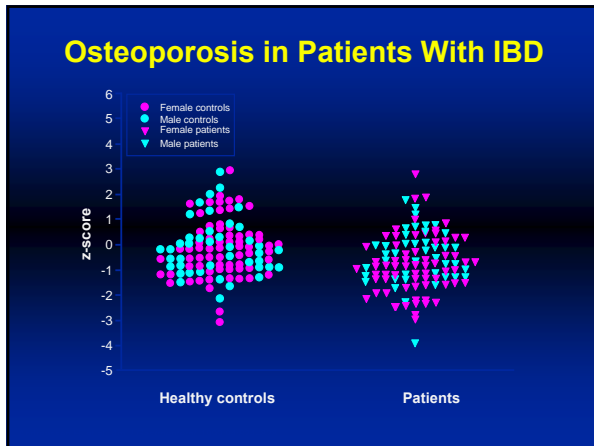
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### Risk of Fracture in Patients With IBD

Fracture Type and Age Group	Incidence Rate Ratio (95% CI)		
	Patients With CD	Patients With UC	All Patients With IBD
<b>Hip*</b>			
0-39 yr	0.0 (0.0-4.86)	1.90 (0.04-17.0)	0.68 (0.02-4.49)
40-59 yr	0.51 (0.01-3.18)	2.92 (0.96-7.47)	1.74 (0.66-3.91)
≥60 yr	1.63 (1.10-2.35)	1.60 (1.13-2.21)	1.61 (1.25-2.05)
<b>Total</b>	<b>1.47 (1.03-2.10)</b>	<b>1.69 (1.26-2.28)</b>	<b>1.59 (1.27-2.00)</b>
<b>Spine†</b>			
0-39 yr	1.60 (0.73-3.15)	1.69 (0.80-3.24)	1.65 (0.98-2.63)
40-59 yr	1.10 (0.34-2.74)	1.33 (0.46-3.13)	1.21 (0.58-2.26)
≥60 yr	1.78 (0.91-3.23)	2.26 (1.39-3.54)	2.06 (1.41-2.95)
<b>Total</b>	<b>1.54 (1.04-2.30)</b>	<b>1.90 (1.36-2.65)</b>	<b>1.74 (1.34-2.24)</b>

\*Defined as a hospital discharge abstract diagnosis of fracture of the femoral neck (International Classification of Diseases, Ninth Revision, Clinical Modification) ICD-9-CM code 820.xx.  
†Defined as diagnosis recorded on a physician billing claim or on a hospital discharge abstract using the following ICD-9-CM codes: spine (806.xx), wrist forearm (813.xx), or rib (807.xx).  
Bernstein CN, et al. *Ann Intern Med*. 2000;133:795-799.

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- ### IBD-Specific Risk Factors
- Chronic inflammation leading to
    - Elevation of cytokines (eg, TNF-alpha), which adversely affects the balance between bone formation and bone resorption
    - Excessive loss or insufficient absorption of calcium
  - Low body mass index (BMI) with lessening of weight-bearing forces on bone
  - Conventional corticosteroid exposure

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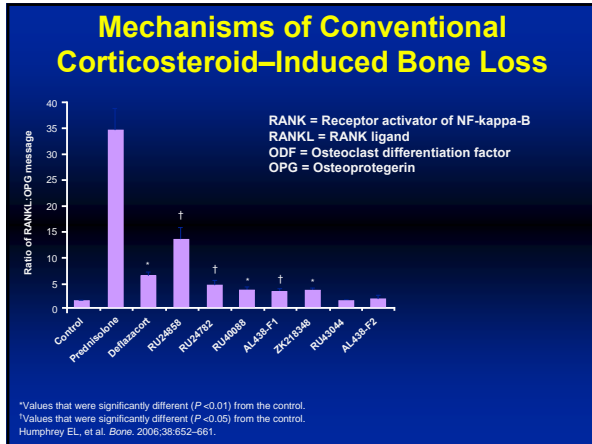
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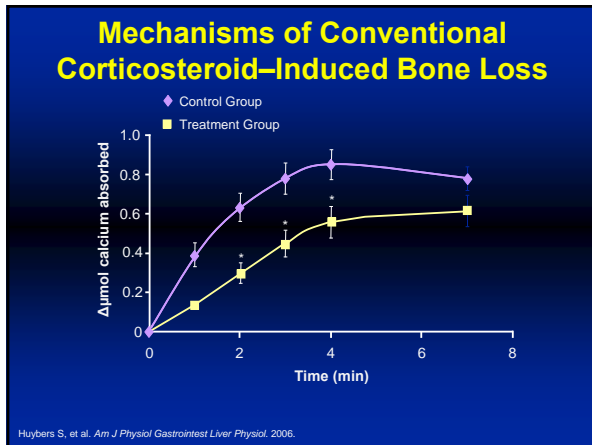
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### Risk of Osteoporosis in IBD Patients

Incidence of Osteoporosis by Gender

	Male	Female	Total, %	P
Number of patients	62	38	100	
% with vertebral osteoporosis (n)	3 (2/62)	11 (4/38)	6	0.18
% with hip osteoporosis (n)	2 (1/62)	18 (7/38)	8	0.004
% with any osteoporosis (n)	5 (3/62)	24 (9/38)	12	0.008

Incidence of Osteoporosis by Disease Type

	UC	CD	Total, %	P
Number of patients	53	47	100	
% with vertebral osteoporosis (n)	4 (2/53)	9 (4/47)	6	0.42, NS
% with hip osteoporosis (n)	2 (1/53)	15 (7/47)	8	0.023, NS
% with any osteoporosis (n)	6 (3/53)	19 (9/47)	12	0.06, NS

Kornbluth A, et al. *Am J Gastroenterol*. 2006;101:1546-1550.

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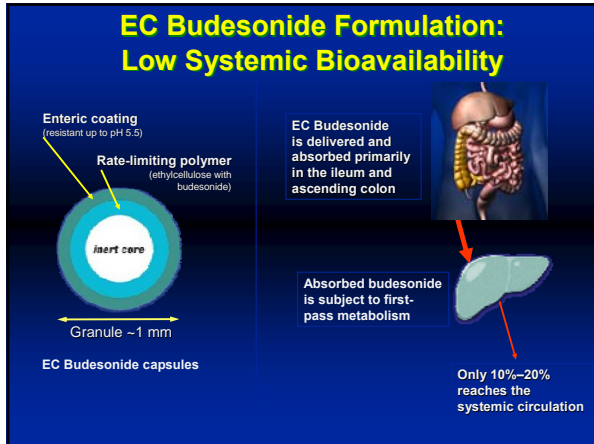
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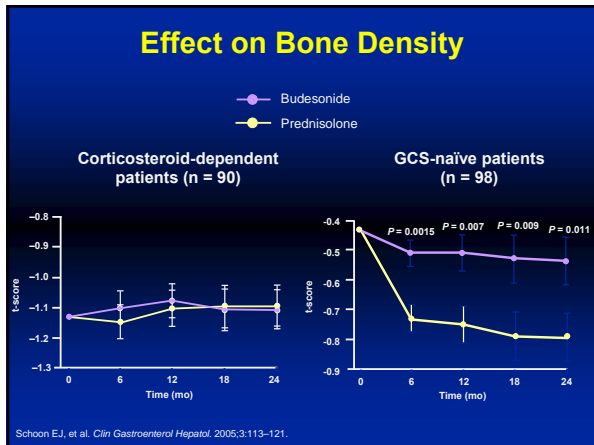
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### Summary of Schoon Study

1. The greatest loss of BMD occurs with the first course of corticosteroids, particularly during the first 6 months
2. Less bone loss occurs with subsequent courses of corticosteroids, but baseline BMD is also lower
3. Over 24 months, patients who had never been exposed to corticosteroids lost very little bone when treated with controlled-release budesonide
4. Practice Point: Based upon efficacy evidence combined with the results of the Schoon study, "CR Budesonide should be considered as the first-line therapy for mild to moderate ileal or ileocecal Crohn's disease in corticosteroid-naïve patients."

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### Evidence-Based Prevention of Osteoporosis: General Measures

- Avoid smoking
- Avoid excessive alcohol consumption
- Engage in regular weight-bearing exercise
- Maintain adequate calcium intake
- In young patients, emphasize importance of maximizing bone density by age 30

Lichtenstein GR, et al. *Inflamm Bowel Dis* 2006;12:797-813.

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### DXA: The Standard Measure of BMD

Ideal Attributes of BMD Test	Dual-Energy X-ray Absorptiometry (DXA, DEXA)
Rapidly performed	Yes
Inexpensive	Yes
Reproducible	Yes
Safe	Yes
Accurate	Yes, but variable

Lichtenstein GR, et al. *Inflamm Bowel Dis* 2006;12:797-813.

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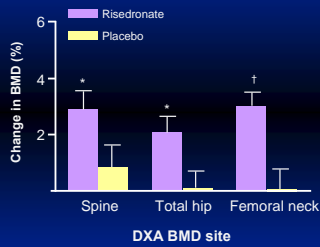
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### The Role of Bisphosphonates



\*P < 0.05.  
†P < 0.005 after adjustment for significant covariates.  
Henderson S, et al. *Am J Gastroenterol* 2006;101:119-123.

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### Summary

- Assess risk for osteoporosis in all patients
- Monitor BMD periodically in select high-risk patients
- Encourage general preventive bone health practices, as all are associated with multiple health benefits, especially smoking cessation in CD patients
- Practice IBD-specific primary, secondary, and tertiary prevention of bone loss
  - Avoid conventional corticosteroids, especially if patient has never received them
  - Discontinue conventional steroids in patients when possible
  - Use of EC Budesonide according to the product label is not deleterious to bone health
  - Use bisphosphonates or alternatives in selected patients, according to guideline recommendations

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### Kidney Stone Disease In IBD

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### Etiology of Stone Disease

- Supersaturation of urine is the key to stone formation
- Intermittent supersaturation - Dehydration
- Crystal aggregation
- Anatomic Abnormalities –
  - PyeloUreteral Junction
  - Medullary Sponge Kidney
- Bacterial Infection
  - E.Coli infection increases matrix content in urine
  - Proteus makes urine alkaline
- Defects in transport of Calcium and Oxalate by Renal epithelia

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### Inhibitors & Promoters of Stone Formation in Urine

#### INHIBITORS

- Citrate – complexes with Ca
- Magnesium – complexes with oxalates
- Pyrophosphate - complexes with Ca
- Zinc- Inhibits crystal Aggregation
- Glycosaminoglycans
- Nephrocalcin
- Tamm- Horsfall Protein

#### PROMOTERS

- Bacterial Infection
- Matrix
- Anatomic Abnormalities-
  - Medullary Sponge Kidney
  - PyeloUreteral Junction obstruction
- Altered Ca and oxalate transport in renal epithelia
- Prolonged immobilization
- Increased uric acid levels i.e taking increased purine subs- promotes crystallization of Ca and oxalate
- ?? Nanobacteria – seen in 97% of renal stones

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### Nephrolithiasis - Types

- 1.) Calcium (Ca-oxalate, Ca-phosphate, or both) – 85%. Radioopaque. Associated with hypercalcemia and hypercalciuria (70%).
- 2.) Magnesium Ammonium Phosphate (NH<sub>4</sub>-Mg-Phos – O) Radiolucent. Formed in alkaline urine by urease positive bugs such as Proteus or Staph. Can form large struvite calculi.
- 3.) Uric acid – Strong association with hyperuricemia (gout). Seen with diseases that have increased cell proliferation (leukemia, myeloproliferative dx).
- 4.) Cystine – secondary to cystinuria.

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### DISEASES ASSOCIATED WITH HYPERCALCEMIA & HYPERCALCURIA

- Hyperparathyroidism
- Sarcoidosis
- Multiple myeloma
- Hyperthyroidism
- Metastatic Malignant Neoplasm
- Leukemia
- Lymphoma
- Myxedema
- Adrenal Insufficiency
- Vitamin D Intoxication

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### HYPERCALCURIA

- 70% of calcium-containing stones are caused by *hypercalciuria*. A number of conditions may produce hypercalciuria. Many are due to genetic factors, but most cases are *idiopathic*.
- The following are mechanisms that can lead to hypercalciuria and calcium stones:
  - Overly efficient intestinal absorption of calcium. In many cases, the source of calcium overload in urine is the intestine, not the kidney. In most of these conditions, genetic factors conspire to increase calcium absorption in the intestine. Researchers are investigating a number of suspects, including a possible defective gene that regulates calcitriol, a form of vitamin D, which, in excess levels, may increase intestinal absorption of calcium. (This is the situation in which restricting calcium in the diet can help prevent stones.)

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### HYPERCALCURIA

- Excessive sodium absorption. Calcium absorption in the renal tubules follows the absorption of sodium and water. High urinary levels of sodium result in increased levels of urinary calcium. Defects in the kidney tubules transport system can cause imbalances in sodium and phosphate that result in elevated calcium in the urine. A high salt (sodium) diet can also produce this effect.
- Excessive chloride absorption. Chloride has a negative charge and calcium a positive one, so they are often used by the body to balance each other. Excess chloride, then, may lead to excess calcium. A gene known as CLCN5, which regulates chloride in the urine, is defective in many patients with calcium stones.
- Renal calcium leak. This is a condition in which the filtering processes in the kidney fail, causing an increase of calcium in the urine.

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### HYPEROXALURIA

- Primary hyperoxaluria is an inherited disorder in which excess oxalate in the urine is the primary problem.
- Secondary hyperoxaluria is caused by specific conditions that result in excess urinary oxalate.
  - Secondary hyperoxaluria is usually caused by excessive intake of dietary oxalates (found in a number of common vegetables, fruits, and grains) or by abnormalities in the metabolism of oxalates. Such defects may be due to various factors, such as the following:
    - Deficiencies of pyridoxine (vitamin B6). Severe vitamin B6 deficiencies (usually due to genetic disorders) can result in overproduction of oxalic acid.
    - Deficiencies in *Oxalobacter formigene*. Deficiency in an intestinal bacterium called *Oxalobacter formigenes* is now a suspect in some cases. This bacterium degrades oxalate and low levels in the intestine increase the risk for oxalate absorption and stone formation.

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### HYPEROXALURIA

- **Short bowel syndrome or Crohn's disease (ileal).** A functional or actual Short bowel syndrome, which may result as a consequence of ileal Crohn's disease or surgery on the small intestine. This is marked by the inability of the intestines to absorb fat and nutrients properly (malabsorption). In such cases, calcium may bind to unabsorbed fat instead of to oxalates. This leaves excess oxalate, which is absorbed by the intestine and excreted into the kidney.
- **Hormones.** Some studies have suggested that androgens are associated with a higher risk for the formation of calcium oxalate crystals while estrogens are linked to a lower risk. Estrogen may help prevent the formation of calcium stones by keeping urine alkaline and raising protective citrate levels.

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### HYPERCALCEMIA

- Hypercalcemia may result as a consequence of the following conditions:
  - Hyperparathyroidism- accounts for approximately 5% of calcium stones. Patients with this disorder have at least a 20% chance of developing kidney stones. Women are more likely to have this disorder than men.
  - Immobilization.
  - Renal tubular acidosis, a disorder that causes acid and alkaline imbalance. It not only increases calcium levels in the bloodstream, it also reduces citrate levels.

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### TYPES OF KIDNEY / URETER STONES

- CALCIUM (OXALATE and /or PHOSPHATE)
- PHOSPHATE (MAGNESIUM AMMONIUM PHOSPHATE)
- URIC ACID
- CYSTINE

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### Uncommon Stones

**XANTHINE STONES**

– (Autosomal Recessive Deficiency of Xanthine Oxidase leading to Xanthinuria)

**DIHYDROXYADENINE STONE**

– ( Deficiency of enzyme adenine phospho ribosyl transferase )

**SILICATE STONES**

– Rare in humans ( excess intake of Antacid with Mg Trisilicate. Mostly in cattle due to ingestion of Sand )

**MATRIX**

- Infection by Proteus - Radiolucent ( all calculi have some amt ( 3% ) of matrix but matrix calculus has 65% Matrix content in calculi)

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### Uncommon Stones

**TRIAMTERENE**

– Anti-hypertensive used with hydrochlorothiazide – spare Potassium. Mostly found as a nucleus in Ca oxalate or uric acid calculus

**Indinavir Stones**

- Drug to treat AIDS (4 to 13%)

**Ephedrine or Guaifenesin**

– Cough medicine - Radiolucent

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### Stones – Chemical Constituents

- **Whewellite** – Calcium Oxalate Monohydrate –  $\text{CaC}_2\text{O}_4 \cdot \text{H}_2\text{O}$
- **Weddelite** - Calcium Oxalate dihydrate –  $\text{CaC}_2\text{O}_4 \cdot 2\text{H}_2\text{O}$
- **Brushite** – Calcium Hydrogen phosphate dihydrate –  $\text{CaHPO}_4 \cdot 2\text{H}_2\text{O}$
- **Whitlockite** - TriCalcium Phosphate –  $\text{Ca}_3(\text{PO}_4)_2$
- **Struvite** – Magnesium Ammonium hexahydrate –  $\text{MgNH}_4\text{PO}_4 \cdot 6\text{H}_2\text{O}$

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### Differential Diagnosis of Radiolucent filling defect on IVU in Ureter or Kidney

#### Must Know

- Uric Acid Calculus
- Matrix Calculus
- Sloughed Papilla
- Blood Clots
- Transitional Cell Carcinoma
- Renal Cysts
- Vascular Lesions

#### Know For Brownie Points

- Xanthine Calculus
- Hydroxyadenine Calculus
- Ephedrine Calculus
- Infection due to gas forming Organism
- Fungal Ball
- Tuberculoma
- Malacoplakia
- Hypertrophied Papilla
- Renal pseudo-tumor

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### OXALATE (CALCIUM OXALATE)

- ALSO CALLED "MULBERRY STONE"
- COVERED WITH SHARP PROJECTIONS
- SHARP → MAKES KIDNEY BLEED (HEMATURIA)
- VERY HARD
- RADIO - OPAQUE



Under microscope looks like

- Hourglass or Dumbbell shape if monohydrate
- Like an Envelope if Dihydrate

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### Radioopaque Kidney Stone



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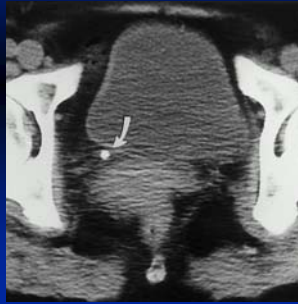
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### Phlebolith Versus Distal Ureteral Calculus

Plain Film (KUB)

NonContrast CT



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### Oxalate Crystals

The treatment of Hyperoxaluria involves the use of:

- Calcium citrate at a dose of 150 mg PO qd. This acts to bind oxalate in gut, preventing absorption; it raises urinary pH and urine citrate levels.
- Ingest low oxalate containing foods.
- It is recommended to add a thiazide diuretic if hypercalciuria develops

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### Food: Oxalate Contents

**Moderate Amounts :**

- Apple Juice
- Beer
- Coffee
- Cola

**High Amounts :**

- Cocoa
- Fresh Tea

**FOODS :**

- Almonds
- Asparagus
- Cashew
- Nuts

- Currants
- Greens
- Plums
- Raspberries
- Spinach

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### PHOSPHATE STONE

- USUALLY → CALCIUM PHOSPHATE
- SOMETIMES → CALCIUM MAGNESIUM AMMONIUM PHOSPHATE OR TRIPLE PHOSPHATE
- SMOOTH → MINIMUM SYMPTOMS
- DIRTY WHITE
- RADIO - OPAQUE



Calcium Phosphate also called 'Brushite' appears like Needle shape under microscope

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### PHOSPHATE STONES

IN ALKALINE URINE  
↓  
ENLARGES RAPIDLY  
↓  
TAKE SHAPE OF CALYCES  
↓  
STAGHORN →



Struvite can form Stag-horn and appear like coffin lid under microscope

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### CALCIUM PHOSPHATE STONES

- Hyperparathyroidism    ↑ Ca   ↓ P
- Renal Tubular Acidosis   ↓ K   ↓ CO<sub>2</sub>
- Medullary Sponge Kidney

**PTH Hormone**

- Promotes renal production of 1-25-dihydroxycholecalciferol (Active Vitamin D)
- Increases absorption of Calcium
- Decreases Phosphorus absorption from Kidneys

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### URIC ACID & URATE STONE

- HARD & SMOOTH
- MULTIPLE
- YELLOW OR RED-BROWN
- RADIO - LUCENT (USE ULTRASOUND)

Under microscope appear like irregular plates or rosettes

PKa of uric acid 5.75 – at this pH 50% of uric acid insoluble. If pH falls further - uric acid more insoluble



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### URIC ACID & URATE STONE - Rx

- Hyperuricosuria involves use of:
  - Dietary restriction (restriction of purines)
  - Use of allopurinol (Zyloprim) at a dose of 200 to 600 mg daily) or Potassium citrate (Urocit-K) at 30-90 mEq/d divided tid-qid with food. This agent Complexes calcium and inhibits urate-induced crystallization.
- For those individuals with a gouty diathesis the use of Potassium citrate 30-90 mEq/d divided tid-qid with food. This functions to increase urinary pH to >5.5. Caution should be exercised to avoid increasing urinary pH to >7.0 or risk calcium phosphate stones.

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### CYSTINE STONE

- AUTOSOMAL RECESSIVE DISORDER
- USUALLY IN YOUNG GIRLS
- DUE TO CYSTINURIA
- CYSTINE NOT ABSORBED BY TUBULES
- MULTIPLE
- SOFT OR HARD – can form staghorns
- PINK OR YELLOW
- RADIO-OPAQUE

Under microscope appears like hexagonal or benzene ring – ask for first morning sample



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### CYSTINE STONE - Management

- High Fluid Intake and Alkalanize Urine – dissolve most of the smaller cystine stones
- D-Penicillamine or MPG (Mercaptopropionylglycine) binds to cystine that is soluble in urine
- Side effects of Penicillamine restricts its use – Allergic rashes, GI problems- Nausea, Vomiting, Diarrhoea
- MPG better tolerated
- Large obstructive stones – Surgery required first

PKa of cystine is 8.3, hence alkalinization above pH7.5 helps to dissolve the stones

Cyanide Nitroprusside Colorimetric Test for detecting Cystinuria. If positive do amino acid chromatography

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### Surgical Conditions and Stone Disease

- Crohn's disease and Ileal Bypass Surgery for eg obesity can lead to increase oxalate absorption and stone disease.
- Ileostomies - In chronic diarrhea states with– bicarbonate loss – systemic acidosis and acidic urine – increases risk of uric acid stones

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### Initial Evaluation of Patients with Kidney Stone Disease

- Limited Evaluation
- Full workup is probably not indicated in patients with a single calcium stone. If there is evidence of active bone disease full work up is recommended. This is best done as outpatient, when patient is on his regular diet.
- Plasma Calcium concentration should be measured on 2 or 3 occasions as well as a 24 hours urine collections for calcium. Since calcium excretion parallels that of sodium, a low sodium intake may mask hypercalciuria. Urinary sodium should always be determined.
- Three 24 hours urine collections made and analyzed for uric acid, calcium, oxalate, and citrate content (citrate is more difficult to measure, may be only determined in patients who is negative for both hyperoxaluria and hyperuricosuria).
- Oxalates are usually measured if there is suspicion of hyperoxaluria secondary to intestinal causes or of primary type. Corresponding serum values obtained.
- Any stones passed should be analyzed.
- All patients should avoid dehydration
- No metabolic disturbance may be found in 10 to 15% of patients

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### Extensive Evaluation of Pts with Kidney Stone Disease

- Extensive evaluation
- Two 24 hour urine samples - (collected on a regular diet)
- One urine sample should be obtained after one week adherence on a diet restricted in calcium (400 mg/day), sodium (100 mEq/day) and oxalate.
- Samples are analyzed for
  - Calcium (Hypercalciuria is defined as calcium excretion greater than 300 mg per day in men and 250 mg per day in women)
  - Oxalate (greater than 40 mg per day of oxalate in the urine should prompt a search for enteric hyperoxaluria which can be treated with oral calcium supplements)
  - Phosphate
  - Uric acid
  - Citrate
  - pH
  - Total volume
  - Magnesium
- Fasting venous blood sample obtained for PTH
- Fasting urinary calcium
  - used to detect renal calcium leak and calciuric response to oral calcium load
- No metabolic disturbance may be found in 10 to 15% of patients

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### Prevention of Recurrence of Kidney Stone Disease

General measures: Goal is to prevent recurrence.

- Fluid intake sufficient to assure a minimum urine output of 3 L/day
- Dietary sodium restriction (100 mEq/d)
- A high sodium intake increases urinary calcium, lowers citrate and promotes sodium urate induced calcium oxalate crystallization and blunts the hypocalciuric response to thiazide
- Oxalate restriction in all patients with oxalate stones, also useful in patients with intestinal hyperabsorption of oxalate and in patients taking sodium cellulose phosphate
- Dietary calcium restriction recommended in patients with intestinal hypercalciuria
- Restriction of proteins useful in presence of hyperuricosuria or hypocitraturia

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### Prevention of Recurrence of Kidney Stone Disease

- Low sodium intake and a diuretic may be the treatment of choice in patients with active stone disease. 90% reduction in risk of new stone formation was obtained by this modality of therapy versus 60% in a placebo treated group.
- Thiazides and/or potassium citrate is the treatment if hypercalciuria persist on this regimen.
- Amiloride can be added. Amiloride favorable effect is related to its correction of hypokalemia which can induce intracellular acidosis which can decrease citrate excretion and it also increase calcium reabsorption in the distal tubule. Citrate normally form a non dissociable but soluble compounds with calcium.
- Potassium citrate and not sodium citrate should be used since sodium will cause intravascular expansion and increase calcium excretion. Potassium citrate will have two indirect effects. most of the exogenous citrate is converted to bicarbonate thus correcting any degree of acidosis that may be contributing to stone formation and secondly the correction of hypokalemia.
- Avoidance of high protein diet is important to prevent recurrence of nephrolithiasis.
- This results in high content of undissociated uric acid. Once a uric acid stone is formed, it could induce formation of calcium oxalate stones by epitaxy, heterogeneous nucleation.

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## INVESTIGATIVE STUDIES

4. Plain KUB X-Ray of Abdomen : **Mandatory**

Effective for viewing radioopaque stones and evaluating stone size, stone shape, stone location, number of stones. Calcium containing kidney stones are radioopaque however, those stones that are uric acid, Indinavir-induced, and cystine calculi are relatively radiolucent

5. Intravenous Urogram (IVU) or Intravenous Pyelogram (IVP): **Not Mandatory**

- 1 in 40,000 patients die due to anaphylactic reaction to contrast
- Useful for radio-lucent stones & to detect Congenital Anomalies in Urinary tracts
- Is considered to be the standard study for assessing stone location and size as well as defining urinary tract anatomy. The drawback of the use of an IVP is that it mandates a bowel prep, uses iv contrast and is a labor intensive study.

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## INVESTIGATIVE STUDIES

6. Renal Ultrasound: **Mandatory**

- Will detect the presence of a stone and additionally will detect if hydronephrosis and hydroureter are present. A stone that is visible on ultrasound, but is not seen on plain abdominal radiograph, could be composed of uric acid or cystine.

7. Abdominal CT (Helical or Spiral): **Mandatory**

- Performed without the administration of iv contrast is the most sensitive diagnostic test for detection of kidney stones and currently the standard test used for evaluation of patients suspected of having nephrolithiasis! Stones that are not visualized on plain films (except indinavir-induced stones) are seen on a CT scan. A distinct advantages of using a CT scan is that other pathologies can also be seen with this study. In addition CT is a rapidly performed study, and it avoids administration of contrast. The disadvantage of using a CT scan is that it cannot evaluate renal function and that it is relatively more expensive than performing an IVP.

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## INVESTIGATIONS (Cont...)

8. DMSA OR DTPA OR MAG3 RENOGRAM –

To study the function of each kidney.

(NOTE: A renal tomogram is a study that is helpful to search for small stones in the kidneys, particularly in obese patients.)

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### Modern Management of Urolithiasis

- ESWL
- Ureterorenoscopy
- Percutaneous Nephrolithotomy
- Laparoscopic Approach to stones

Open Ureterolithotomy, Pyelolithotomy or Nephropyelolithotomy is required in less than 1% to 2% of modern stone management

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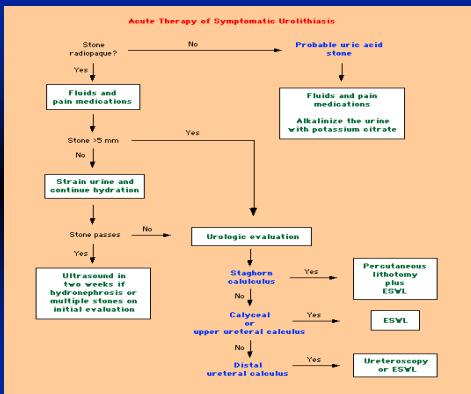
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### TREATMENT ALGORITHM



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