EVIDENCE-BASED GI AN ACG PUBLICATION



Fueling the Failing Liver—ACG Clinical Guideline: Malnutrition and Nutritional Recommendations in Liver Disease





Leandro Sierra, MD Contributing Writer

Nikki Duong, MD Associate Editor

Leandro Sierra, MD¹ and Nikki Duong, MD²

¹Internal Medicine Resident, Department of Internal Medicine, Cleveland Clinic, Cleveland, OH

²Clinical Assistant Professor of Medicine, Gastroenterology & Hepatology, Stanford University School of Medicine, Stanford, CA

This summary reviews Singal AK, Wong RJ, Dasarathy S, Abdelmalek MF, Neuschwander-Tetri BA, Limketkai BN, Petrey J, McClain CJ. ACG Clinical Guideline: Malnutrition and Nutritional Recommendations in Liver Disease. Am J Gastroenterol 2025;120:950–972.

Correspondence to Nikki Duong, MD. Associate Editor. Email: EBGI@gi.org

Keywords: guideline, liver disease, nutrition

STRUCTURED ABSTRACT

Question: What are the appropriate methods for assessing malnutrition in patients with chronic liver disease, and what nutritional therapies are recommended across different stages of chronic liver disease?

Design: The guideline was developed by hepatology experts under the ACG Practice Parameters Committee using PICO questions and the Grading of Recommendations, Assessment, Development and Evaluation (GRADE) methodology to assess evidence strength and develop recommendations.¹ Both recommendation statements and expert key concepts are included.

Patients: Adults with chronic liver disease, including those with cirrhosis, alcohol-associated liver disease, and metabolic dysfunction-associated steato-hepatitis (MASH).

Exposure or Interventions: The guideline recommends adequate caloric and protein intake, late-evening carbohydrate snacks, plant-based or branched chain amino acids (BCAA)-enriched protein sources, and avoidance of unnecessary protein restriction. Micronutrient repletion (zinc, vitamin D), vitamin E for MASH, and regular coffee intake are also suggested. Enteral nutrition is preferred over parenteral when oral intake is insufficient.

Outcomes: Through individualized nutritional interventions and disease-specific dietary strategies, patients with chronic liver disease can improve their nutritional status, reduce their risk of complications (e.g., hepatic encephalopathy, infections, and ascites), enhance their muscle mass, improve their quality of life, and reduce their short—and long-term mortality.

Data Analysis: Recommendations were developed through a structured literature review of studies assessing nutritional interventions in liver disease. The GRADE framework was used to assess the quality of evidence and strength of recommendations. Where evidence was insufficient for formal grading, expert consensus informed clinical guidance. Recommendations are categorized as strong or conditional based on benefit-risk assessment, certainty of evidence, feasibility, and patient-centered values.

Funding: This guideline was funded by the ACG. No external commercial support was provided, and all guideline panel members completed conflict of interest disclosures in accordance with ACG policy. The development process was overseen by the ACG Practice Parameters Committee to ensure methodological rigor and independence.

Results: The 2025 ACG Clinical Guideline on Malnutrition and Nutritional Recommendations in Liver Disease provides evidence-based guidance for optimizing nutritional assessment and therapy across a spectrum of chronic liver diseases. Recommendations and selected key supporting evidence are summarized in **Table 1**. The guideline emphasizes the importance of early and routine nutritional screening in patients with chronic liver disease, including cirrhosis, highlighting that malnutrition and sarcopenia are prevalent and worsen with increasing liver disease severity. In hospitalized patients with cirrhosis, early oral or enteral nutrition (EN)

3 Sierra and Duong

HEPATOLOGY

Statement	Quality of Evidence/Strength of recommendation	Key Supporting Evidence
1. Suggest initiating early oral or enteral nutrition in hospitalized pa- tients with cirrhosis	Low/Conditional	Three RCTs and 2 cohort studies ²⁻⁶ showed that starting oral/enteral feeds within 48 h increased calorie/protein delivery, shortened hospital/ICU stay, and reduced in-hospital mortality by $\approx 30\%$
2. Suggest implementation of nutri- tional supplementation therapy in patients with cirrhosis or alcohol-as- sociated hepatitis	Very Low/ Conditional	In AH, daily 1-month adequate caloric intake in VA tri- als correlated with 6-month survival; EN improved bili- rubin, antipyrine clearance, and reduced 1-year mortali- ty vs prednisone; intake <21.5 kcal/kg/day or <77.6 g protein/day predicted worse outcomes ⁷⁻⁹
3. Suggest use of natural vitamin E (800 IU/day) in patients with MASH without cirrhosis	Low/Conditional	PIVENS (adults) and TONIC (children) RCTs showed 800 IU/day d- α -tocopherol for 96 wk improved steatosis, ballooning, and inflammation in 43%-58 % vs 19%-28% on placebo, with ALT normalization in >50% ¹⁰⁻¹²
4. Suggest intake of ≥2 cups of cof- fee/day in chronic liver disease to reduce fibrosis risk progression and HCC development	Low/Conditional	Large prospective cohorts (>200,000 subjects) demonstrated dose-responsive 25%-40% reductions in advanced fibrosis and 50% lower HCC incidence among drinkers of \geq 2 cups/day, independent of alcohol and obesity ¹³⁻¹⁵
5. No recommendation for or against strict sodium restriction in patients with cirrhosis and ascites managed with diuretic therapies	Insufficient	Two small RCTs (≤ 60 patients) comparing 2g vs unre- stricted sodium showed inconsistent effects on ascites control, hyponatremia, and QoL; both with high bias risk ^{16, 17}
6. Recommend against protein re- striction in decompensated cirrhosis with HE	Very Low/ Conditional	Four crossover studies showed ≤ 0.6 g/kg protein wors- ened nitrogen balance and muscle loss without HE im- provement; reintroduction of 1.2 g/kg improved cogni- tion ^{18,19}
7. Suggest vegetarian protein sources in cirrhosis with HE when supplementation is needed	Low/Conditional	Plant-based protein lowers ammonia via higher ar- ginine/fiber content. Four studies have shown clinical improvement with vegetarian protein. A RCT (120 pa- tients) found vegetable protein improved minimal HE (71% vs 23%) and reduced HE risk (10% vs 22%) ^{20,21}
8. Recommend adding branched-chain amino acids to standard therapy in patients with cirrhosis and HE	Moderate/Strong	BCAA deficiency impairs ammonia detoxification via muscle. BCAA supplementation improves muscle mass, reduces ammonia, and enhances glutamine synthesis. A Cochrane meta-analysis of 16 RCTs (n=827) found oral BCAAs improved HE (RR 0.67); after excluding lactu- lose/neomycin controls, reduced mortality (RR 0.76); IV BCAAs showed no benefit ^{22,23}
9. Recommend incorporating a late-evening snack in cirrhosis to improve BMI, lean mass, and reduce ascites/HE	Moderate/Strong	Outpatient nutrition therapy improves survival and re- duces hospitalizations in cirrhosis. Late evening snacks (\approx 710 kcal) reduce protein catabolism and improve ni- trogen balance, with meta-analyses showing improved liver function and fewer complications (ascites, HE); effects on muscle mass and survival remain incon- sistent ^{24, 25}

Table 1. Summary of recommendations with supporting evidence.

AH, alcohol associated hepatitis; BCAA, branched chain amino acids; BMI, body mass index; EN, enteral nutrition; HCC, hepatocellular carcinoma; HE, hepatic encephalopathy; ICU, intensive care unit; IV, intravenous; MASH, metabolic dys-function-associated steatohepatitis; RCT, randomized controlled trial; RR, relative risk; VA, Veterans Administration.

HEPATOLOGY

within 48 hours is associated with improved protein-calorie delivery, reduced hospital length of stay, and lower in-hospital mortality (approximately 30% reduction). In patients with alcohol associated hepatitis (AH), nutritional support, particularly through enteral supplementation, has been shown to improve liver function markers and reduce 1-year mortality compared to corticosteroid therapy, mainly by lowering infection rates. Furthermore, lower caloric and protein intake thresholds (less than 21.5 kcal/kg/day and less than 77.6 g/day) independently predict worse outcomes in this population.

In the outpatient setting, structured nutrition counseling and frequent small meals or late evening snacks (high-complex carbohydrate ~200 kcal) are associated with improved nitrogen balance, reduced sarcopenia, and a decreased incidence of hepatic encephalopathy (HE) and ascites. These benefits are demonstrated in multiple studies and meta-analyses, although evidence on survival impact remains limited.

The guideline advises against protein restriction in patients with HE. Instead, diets enriched in vegetarian protein sources may be beneficial due to their favorable effects on ammonia metabolism and neurotoxins. Supporting evidence from older but consistent RCTs indicates reduced ammonia levels and improved psychometric scores. Similarly, supplementation with BCAAs in HE improves the resolution of symptoms (relative risk [RR] 0.67 with oral BCAA) and reduces mortality when compared to standard therapy alone (RR 0.76 after exclusion of lactulose/ neomycin controls). BCAAs also enhance muscle mass and ammonia detoxification.

Vitamin and mineral supplementation is addressed with conditional recommendations. Vitamin D deficiency is common in cirrhosis and is associated with infections, spontaneous bacterial peritonitis, and mortality. While evidence is limited, supplementation improves biochemical profiles and Child-Pugh class. Zinc replacement is also recommended in individuals with low serum zinc levels or those with symptoms of deficiency. The guideline also supports ≥ 2 cups/day of coffee to reduce fibrosis progression and hepatocellular carcinoma risk in chronic liver disease.

5 Sierra and Duong

HEPATOLOGY

Lifestyle interventions, including diet and exercise, are endorsed for patients with MASH to reduce disease progression and improve metabolic outcomes, despite limited data on fibrosis reversal. Sodium restriction in cirrhosis with ascites remains inconclusive due to conflicting trial results. These findings support a paradigm shift toward early, individualized, and proactive nutritional management in liver disease to improve patient outcomes across inpatient and outpatient settings.



Figure 1. The impact of malnutrition, frailty, and sarcopenia in end-stage liver disease. LT, liver transplantation; ICU, intensive care unit. Adapted from Duong et al.²⁶

COMMENTARY

Why Is This Important?

Malnutrition and sarcopenia are highly prevalent and underrecognized, affecting 20% of patients with compensated cirrhosis and up to 60% in those with decompensated cirrhosis and are strongly linked to morbidity, mortality, and poor transplant outcomes (**Figure 1**). Malnutrition, sarcopenia, and frailty are not interchangeable terms, though they are linked. A review written by Duong et al highlights these concepts in detail.²⁶

This guideline reflects a growing understanding that structured nutritional assessment and individualized therapy are essential across the spectrum of liver disease. It marks the first ACG guideline dedicated to nutrition in liver disease, addressing gaps in both inpatient and outpatient management.

Key Study Findings

The guideline strongly recommends adding BCAAs to standard therapy in patients with cirrhosis and HE, given evidence supporting improved HE resolution and reduced mortality.

A late-evening snack is also strongly recommended to enhance body mass index, preserve lean muscle mass, and decrease the incidence of ascites and HE. Early oral or EN is advised for hospitalized cirrhosis patients to improve protein-calorie delivery and lower in-hospital mortality. Protein restriction is discouraged in decompensated cirrhosis with HE, and vegetarian protein sources may be preferred when supplementation is necessary due to their favorable effects on ammonia metabolism.

Additional conditional suggestions include nutritional supplementation in alcohol-associated liver disease, vitamin E supplementation in MASH without cirrhosis, and daily coffee intake to reduce fibrosis progression and HCC risk.

Caution

Some recommendations considered low or very low-quality evidence of older studies with small sample sizes. While findings support proactive nutrition management, the evidence base remains limited for some outcomes, particularly long-term survival.

My Practice

Vital signs are just that—vital. It would be unthinkable to see a patient without first reviewing their vital signs, whether in an outpatient or inpatient setting. From my perspective, assessing and understanding a patient's nutritional status should be viewed similarly, especially in those with chronic liver disease.

Evidence clearly shows that the severity of malnutrition correlates with the progression of liver disease. Sarcopenia, likewise, impacts outcomes throughout the transplant continuum. Yet, despite this well-established knowledge, I am often surprised when new patients express that no one has discussed fundamental nutrition goals with them—such as high protein intake, late-night protein snacks, coffee consumption, exercise, and limitations on salt intake.

While counseling patients takes time and deliberate effort, it is both essential and worthwhile, particularly during the initial visit. I approach nutrition counseling as an ongoing conversation rather than in isolation. I focus on small, achievable changes, explaining that progress—not perfection—is the goal. Encouraging even 1-3 changes between visits can lead to significant improvements over time.

Adapting my approach to the patient's understanding is crucial. When a patient struggles to grasp the rationale, I simplify my explanations; when a patient is

7 Sierra and Duong

well-informed, I elevate the discussion. Cultural competence and flexibility are key to effective communication.

In the ideal situation, access to a strong multidisciplinary team that includes registered dieticians is instrumental to a patient-centered practice. Separate, dedicated teams for the general hepatology and transplant populations are also ideal, given the often varying needs of these unique cohorts.

Understandably, accessing a dietitian can often be cost-prohibitive as it is where I currently practice. Thus, I recently met with our dietitians to create a patient-friendly "Mediterranean diet" handout that is easily accessible in our clinic. I find that patients enjoy having a physical handout, allowing them to start making small dietary changes either before they meet with the dietitian or, if they cannot afford it, to utilize this handout as a starting point in their own journey to achieve their personal weight goals. Ensuring that such patient materials are available in multiple languages is key to providing culturally competent care. As a final point, in the Bay Area, where I live and practice, there is a fair number of patients with lean MASH. These patients are certainly some of the more challenging cases to care for due to the limited therapeutic options. In these instances, I have often recommended Vitamin E. I very seldom recommend BCAAs and acknowledge the variability in this practice among my colleagues.

In summary, we should continue to consider nutrition assessment as the fifth vital sign. To ensure equitable and inexpensive access to nutrition assessment, I suspect the future will leverage AI and serum biomarkers—or at least that is the hope!

For Future Research

According to the guideline, future research should focus on validating accessible biomarkers for sarcopenia using proteomic and metabolomic approaches and clarifying the therapeutic role of dysbiosis-targeted strategies beyond HCC.

Intervention trials are needed to assess whether reducing simple sugars benefits patients with cirrhosis and visceral adiposity. The optimal type, intensity, and impact of exercise before and after liver transplantation remains under investigation.

Additionally, the long-term effects of vitamin D supplementation on cirrhosis progression and HCC are still unknown. More evidence is also needed to confirm whether lifestyle modification can truly reverse hepatic fibrosis.

Conflict of Interest

The authors have no conflicts of interest to disclose.

HEPATOLOGY

Abbreviations

AH, alcohol associated hepatitis; AI, artificial intelligence; BCAA, branched chain amino acids; EN, enteral nutrition; HCC, hepatocellular carcinoma; HE, hepatic encephalopathy; ICU, intensive care unit; LT, liver transplantation; MASH, metabolic dysfunction-associated steatohepatitis; RCT, randomized controlled trial; RR, relative risk.

REFERENCES

- Singal AK, Wong RJ, Dasarathy S, Abdelmalek MF, Neuschwander-Tetri BA, Limketkai BN, Petrey J, McClain CJ. ACG Clinical Guideline: Malnutrition and Nutritional Recommendations in Liver Disease. *Am J Gastroenterol* 2025;120:950 –972.
- 2. Bunout D, Aicardi V, Hirsch S, et al. Nutritional support in hospitalized patients with alcoholic liver disease. *Eur J Clin Nutr* 1989;43(9):615–21.
- 3. Hirsch S, Bunout D, de la Maza P, et al. Controlled trial on nutrition supplementation in outpatients with symptomatic alcoholic cirrhosis. J Parenter Enteral Nutr 1993;17(2):119–24.
- 4. Bukharin VA, Bondarev II, Kagramanov II, et al. Surgical treatment of congenital mitral valve insufficiency [in Russian]. *Grud Serdechnososudistaia Khir* 1991 (7):13–20.
- 5. Dupont B, Dao T, Joubert C, et al. Randomised clinical trial: Enteral nutrition does not improve the long-term outcome of alcoholic cirrhotic patients with jaundice. *Aliment Pharmacol Ther* 2012;35(10):1166–74.

- 6. Fialla AD, Israelsen M, Hamberg O, et al. Nutritional therapy in cirrhosis or alcoholic hepatitis: A systematic review and meta -analysis. *Liver Int* 2015;35(9):2072–8.
- 7. Cabre E, Rodríguez-Iglesias P, Caballería J, et al. Short- and long-term outcome of severe alcohol-induced hepatitis treated with steroids or enteral nutrition: A multicenter randomized trial. *Hepatology* 2000; 32(1):36–42.
- 8. Moreno C, Deltenre P, Senterre C, et al. Intensive enteral nutrition is ineffective for patients with severe alcoholic hepatitis treated with corticosteroids. *Gastroenterology* 2016;150(4):903–10.e8.
- 9. Mohamed AA, Al-Karmalawy AA, El-Kholy AA, et al. Effect of vitamin D supplementation in patients with liver cirrhosis having spontaneous bacterial peritonitis: A randomized controlled study. *Eur Rev Med Pharmacol Sci* 2021;25 (22):6908–19.
- 10.Lavine JE, Schwimmer JB, Van Natta ML, et al. Effect of vitamin E or metformin for treatment of nonalcoholic fatty liver disease in children and adolescents: The TONIC randomized controlled trial. *JAMA* 2011; 305(16):1659–68.
- Sanyal AJ, Chalasani N, Kowdley KV, et al. Pioglitazone, vitamin E, or placebo for nonalcoholic steatohepatitis. N Engl J Med 2010;362(18): 1675–85.
- 12. Anty R, Marjoux S, Iannelli A, et al. Regular coffee but not espresso drinking is protective against fibrosis in a cohort mainly composed of morbidly obese European women with NAFLD undergoing bariatric surgery. J Hepatol 2012;57 (5):1090–6.
- 13. Chen CL, Chang WC, Yi CH, et al. Association of coffee consumption and liver fibrosis progression in patients with HBeAg-negative chronic hepatitis B: A 5year population-based cohort study. J Formos Med Assoc 2019;118(2):628–35.

- 9 Sierra and Duong
- 14. Hodge A, Lim S, Goh E, et al. Coffee intake is associated with a lower liver stiffness in patients with non-alcoholic fatty liver disease, hepatitis C, and hepatitis B. *Nutrients* 2017;9(1):56.
- 15. Sewter R, Heaney S, Patterson A. Coffee consumption and the progression of NAFLD: A systematic review. *Nutrients* 2021;13(7):2381.
- 16. Biggins SW, Angeli P, Garcia-Tsao G, et al. Diagnosis, evaluation, and management of ascites, spontaneous bacterial peritonitis and hepatorenal syndrome: 2021 practice guidance by the American Association for the Study of Liver Diseases. *Hepatology* 2021;74(2): 1014–48.
- 17.Gauthier A, Levy VG, Quinton A, et al. Salt or no salt in the treatment of cirrhotic ascites: A randomised study. *Gut* 1986;27 (6):705–9.
- 18.Cordoba J, Lo pez-Hellín J, Planas M, et al. Normal protein diet for episodic hepatic encephalopathy: Results of a randomized study. J *Hepatol* 2004;41(1):38–43.
- 19.Campollo O, Sprengers D, Dam G, et al. Protein tolerance to standard and high protein meals in patients with liver cirrhosis. *World J Hepatol* 2017;9(14):667 -76.
- 20.Iqbal U, Jadeja RN, Khara HS, et al. A comprehensive review evaluating the impact of protein source (vegetarian vs. meat based) in hepatic encephalopathy. *Nutrients* 2021;13(2):370.
- 21.Maharshi S, Sharma BC, Sachdeva S, et al. Efficacy of nutritional therapy for patients with cirrhosis and minimal hepatic encephalopathy in a randomized trial. *Clin Gastroenterol Hepatol* 2016;14(3):454-60.
- 22.Vilstrup H, Amodio P, Bajaj J, et al. Hepatic encephalopathy in chronic liver disease: 2014 practice guideline by the American Association for the Study of Liver Diseases and the European

Association for the Study of the Liver. *Hepatology* 2014;60(2):715–35.

- 23. Gluud LL, Dam G, Les I, et al. Branchedchain amino acids for people with hepatic encephalopathy. *Cochrane Database Syst Rev* 2017;5(5): CD001939.
- 24. Tsien CD, McCullough AJ, Dasarathy S. Late evening snack: Exploiting a period of anabolic opportunity in cirrhosis. *J Gastroenterol Hepatol* 2012;27(3):430–41.
- 25. Chen CJ, Wang LC, Kuo HT, et al. Significant effects of late evening snack on liver functions in patients with liver cirrhosis: A meta-analysis of randomized controlled trials. J *Gastroenterol Hepatol* 2019;34(7):1143–52.
- 26. Duong N, Sadowski B, Rangnekar AS. The impact of frailty, sarcopenia, and malnutrition on liver transplant outcomes. *Clin Liver Dis* (Hoboken) 2021;17(4):271 -6.