Review Article

Nutritional Management in Patients with Chronic Liver Disease: A Comprehensive Review

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Abstract

Chronic liver disease (CLD) is a progressive disorder marked by deteriorating liver function resulting from persistent inflammation, fibrosis, and eventually cirrhosis. It affects more than 844 million people worldwide and is responsible for approximately 2 million deaths each year. CLD arises from a variety of causes, including excessive alcohol consumption, non-alcoholic fatty liver disease (NAFLD), viral hepatitis, autoimmune disorders, and inherited conditions.

Malnutrition is a frequent complication in CLD, primarily due to disruptions in fat absorption, metabolic alterations, and overall nutritional deficiencies. Protein-calorie malnutrition (PCM) is particularly prevalent, occurring in up to 90% of individuals with advanced liver disease. This nutritional deficit contributes to worsening outcomes, increasing the risk of complications such as hepatic encephalopathy (HE), hepatorenal syndrome, and diminished liver regeneration.

Management of nutrition in CLD focuses on preserving energy and protein balance through appropriate feeding strategies. Current guidelines recommend an energy intake of 35–40 kcal/kg/day and a protein intake of 1.2–1.5 g/kg/day. Enteral nutrition, which includes both oral intake and feeding via tubes, is typically preferred due to its alignment with natural digestive processes and reduced complication rates. Oral feeding strategies emphasize small, frequent meals that are rich in complex carbohydrates, high-quality protein, and branched-chain amino acids (BCAAs). Tube feeding becomes necessary when oral intake fails to meet nutritional needs, with formulas customized to the patient's condition. In situations where enteral feeding is not possible, parenteral nutrition may be used to provide precise amounts of essential nutrients intravenously.

Although nutritional support has advanced significantly, both enteral and parenteral methods carry risks such as infection, refeeding syndrome, and gastrointestinal side effects. Therefore, successful nutritional management in CLD requires a multidisciplinary approach, tailored to the individual, to reduce complications, enhance nutritional status, and support liver function recovery.

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1. INTRODUCTION

Chronic liver disease (CLD) refers to the progressive decline in liver function lasting over six months, impairing vital processes such as the synthesis of clotting factors and proteins, detoxification of metabolic byproducts, and bile excretion. It involves ongoing inflammation, damage, and regeneration of liver tissue, ultimately leading to fibrosis and cirrhosis. CLD has a wide range of causes, including prolonged alcohol abuse, toxins, infections, autoimmune diseases, and genetic or metabolic disorders. Cirrhosis, the advanced stage of CLD, is characterized by disrupted liver structure, widespread nodule formation, vascular reorganization, new blood vessel growth, and extracellular matrix deposition. At the cellular level, fibrosis results from the activation of stellate cells and fibroblasts, while liver tissue regeneration depends on hepatic stem cells. CLD is a prevalent clinical condition, with emphasis placed on its common causes, symptoms, and management strategies [1].

Over 844 million people globally are estimated to be affected by chronic liver disease, which accounts for around 2 million deaths annually [2]. Non-alcoholic fatty liver disease (NAFLD) is the leading cause of chronic liver disease, affecting an estimated 25% of the global population and up to 90% of individuals with obesity. In comparison, its more severe form, non-alcoholic steatohepatitis (NASH), has a prevalence of approximately 3–5% [3]. The liver plays a central role in various metabolic processes involving proteins, carbohydrates, and fats, making it essential for maintaining proper nutritional balance. In advanced stages of liver disease, fat malabsorption occurs, along with deficiencies in fat-soluble vitamins, reduced levels of water-soluble vitamins, and altered micronutrient metabolism. Consequently, liver dysfunction can lead to malnutrition and sarcopenia [4]. Diet and nutritional status play a crucial role as both therapeutic and prognostic factors in patients with liver disease [5]. A healthy, balanced diet with a variety of foods is typically recommended for patients with chronic liver disease, along with the avoidance of ultra-processed industrial foods, sugar-sweetened beverages, and high-fat foods [6]. Recommendations for the intake of macro- and micronutrients are based on addressing deficiencies caused by the specific nature of the disease (such as absorption issues or metabolic disorders affecting carbohydrate, protein, and lipid metabolism) or to support liver function, such as improving liver enzyme levels [5]. Alcohol should be completely avoided, as ethanol and its metabolites induce metabolic, biochemical, and molecular disruptions not only in the liver but also in skeletal muscles, ultimately leading to impaired proteostasis [7]. The primary aim of treatment is to halt disease progression and prevent complications, necessitating a multidisciplinary approach that includes nutritional support [1]. Protein-calorie malnutrition (PCM) is a common condition across all stages of chronic liver disease (CLD) and affects 65–90% of patients with advanced stages of the disease [8-10]. Malnutrition emerges in the early stages of liver disease, with a nearly direct correlation between the severity of the disease and the extent of malnutrition [11, 12]. Protein-calorie malnutrition (PCM) is linked to a higher risk of complications, including esophageal varices, hepatic encephalopathy (HE), hepatorenal syndrome, reduced liver function and regenerative capacity, as well as increased surgical morbidity and mortality. Additionally, malnutrition is an independent predictor of mortality in patients with chronic liver disease (CLD) [13].

Etiology

Chronic liver disease (CLD) arises from various causes, with some of the most common being alcoholic liver disease, non-alcoholic fatty liver disease (NAFLD), chronic viral hepatitis, genetic conditions, autoimmune disorders, and other less common factors. Alcoholic liver disease encompasses a spectrum ranging from alcoholic fatty liver (with or without hepatitis) to alcoholic hepatitis (reversible with acute ingestion) and cirrhosis (irreversible). It is most frequently seen in patients with severe alcohol use disorder, making it a leading cause of CLD. NAFLD is strongly linked to metabolic syndrome, including obesity, hyperlipidemia, and diabetes mellitus, and may progress to non-alcoholic steatohepatitis (NASH), which leads to liver fibrosis. Metabolic syndrome risk factors aggravate this condition. Chronic viral hepatitis, particularly hepatitis B, C, and D, is a significant cause of CLD, particularly in East Asia and Sub-Saharan Africa. Hepatitis C genotypes vary geographically: genotypes 1a and 1b are more common in Europe and North America, genotype 3 in Southeast Asia, and genotype 4 (subtype 4a) in Egypt. Chronic hepatitis C, if untreated, can lead to hepatocellular carcinoma. Genetic conditions also contribute to CLD. Alpha-1 antitrypsin deficiency is the most common genetic cause in children, while hereditary hemochromatosis, an autosomal recessive disorder caused by HFE gene mutation, leads to excessive iron absorption and subsequent organ fibrosis. Wilson disease, another autosomal recessive disorder, results in copper accumulation. Autoimmune causes of CLD include autoimmune hepatitis, which involves liver parenchyma destruction by autoantibodies and predominantly affects females, often presenting with cirrhosis. Primary biliary cirrhosis (PBC), an autoimmune condition leading to the destruction of intrahepatic bile ducts and liver fibrosis, is more common in middle-aged women and associated with elevated alkaline phosphatase levels. Primary sclerosing cholangitis (PSC), often linked with ulcerative colitis, is characterized by inflammation and fibrosis, causing narrowing of bile ducts. Autoimmune hepatitis (AIH), a chronic inflammatory condition, is more prevalent in women and involves elevated levels of autoantibodies like antinuclear antibodies and anti-smooth muscle antibodies. Other less common causes of CLD include drug-induced liver damage from agents such as amiodarone, isoniazid, methotrexate, phenytoin, and nitrofurantoin; vascular conditions like Budd-Chiari syndrome; and idiopathic or cryptogenic origins, which account for approximately 15% of cases [1].

Epidemiology

Chronic liver disease is a leading cause of mortality, particularly in developing countries, and its prevalence has been increasing in recent years. In developed nations, the most common causes of chronic liver disease include alcoholic liver disease, chronic

viral hepatitis (hepatitis B and C), non-alcoholic fatty liver disease (NAFLD), and hemochromatosis. According to the National Vital Statistics Report (2017) from the Centers for Disease Control and Prevention, approximately 4.5 million adults in the United States, or 1.8% of the adult population, were affected by chronic liver disease and cirrhosis. That year, chronic liver disease and cirrhosis accounted for 41,473 deaths, translating to a mortality rate of 12.8 deaths per 100,000 people [1, 14].

Malnutrition

Malnutrition in patients with chronic liver disease (CLD) and cirrhosis is caused by multiple factors, including loss of appetite, malabsorption, and increased metabolic demands. Reduced dietary intake is commonly observed in cirrhotic patients. The liver plays a role in appetite regulation by clearing chemical mediators such as cholecystokinin, which induces satiety. Additionally, the liver contributes to the production of splanchnic cytokines that diminish hypothalamic-driven appetite signals. Ascites, a common complication of CLD, may also lead to a feeling of early fullness due to mechanical compression. Beyond reduced dietary intake, there are metabolic changes in energy utilization among CLD patients. These individuals show increased fat oxidation during fasting. A study revealed that during an overnight fast, 58% of energy in cirrhotic patients was derived from fat oxidation, compared to 55% from carbohydrates in healthy controls. This may be linked to the reduced hepatic glycogen stores seen in cirrhosis. Moreover, a study found that body size improved with refeeding therapy in cirrhotic patients, suggesting that insufficient dietary intake significantly contributes to body mass decline and malnutrition in these patients [15, 16]. Malabsorption is a significant factor contributing to malnutrition in patients with chronic liver disease (CLD). A reduction in the bile-salt pool caused by liver disease can result in fat malabsorption, particularly in individuals with coexisting biliary or pancreatic conditions, leading to decreased absorption of fat and fat-soluble vitamins. The extent of malabsorption in CLD patients without associated cholestatic disease remains debated. Some studies suggest that bacterial overgrowth due to small bowel hypomotility and portal hypertension in CLD patients contributes to malabsorption, regardless of the disease's underlying cause. However, other research indicates that fat and protein malabsorption is minimal unless biliary or pancreatic disease is also present [17, 18]. The role of increased metabolic demands in contributing to malnutrition in chronic liver disease (CLD) remains uncertain, with evidence varying on the extent of its impact. A study by Müller *et al*. involving 473 cirrhotic patients found that, on average, resting energy expenditure (REE) was normal. However, 34% of the patients exhibited hypermetabolism, with an REE exceeding 120% of the expected value. Proposed causes of hypermetabolism in CLD include infections, ascites, and portal hypertension, though the relationship between energy expenditure and malnutrition in cirrhosis remains unclear, warranting further investigation. In addition to metabolic changes, protein requirements are elevated in CLD patients.This increase is attributed to reduced protein synthesis and enhanced protein degradation. Low hepatic glycogen stores lead to increased gluconeogenesis from amino acids, derived from protein breakdown. This heightened protein demand in cirrhotic patients can exacerbate malnutrition [19, 20].

Management

Therapeutic strategies to maintain adequate nutrition in patients with chronic liver disease (CLD) can be categorized into enteral and parenteral approaches. The choice between these methods depends on individual patient factors and the severity of the disease. Generally, guidelines recommend an energy intake of 35–40 kcal/kg of body weight (BW) per day and a protein intake of 1.2–1.5 g/kg BW per day for cirrhotic patients [21, 22]. For an average 70 kg adult, these nutritional requirements can typically be met with a standard diet supplemented as necessary, often with protein supplements.

DIAGNOSIS

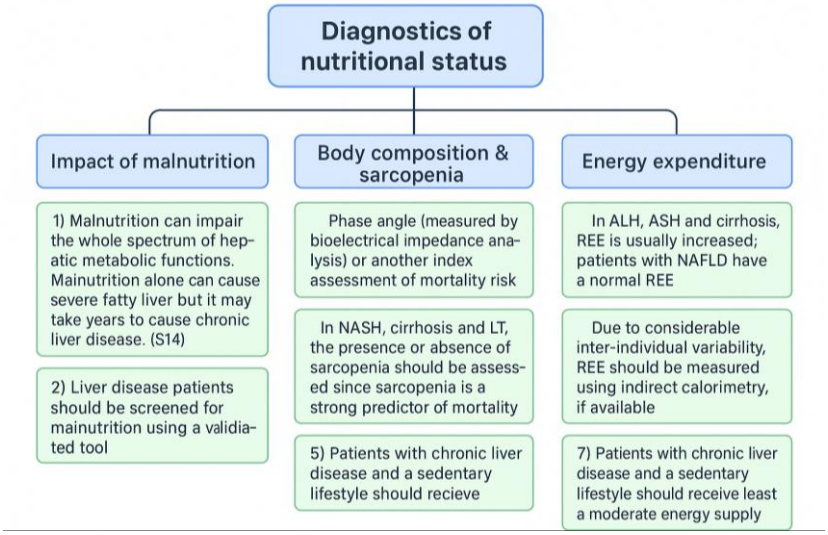

Fig 1: Diagnosis of nutritional status Adopted from Bischoff, Stephan C., *et al*. "ESPEN practical guideline: Clinical nutrition in liver disease." Clinical Nutrition 39.12 (2020): 3533-3562.

The dietary management of CLD patients aligns with a normal diet, provided caloric and protein needs are fulfilled. However, specific conditions, such as hepatic encephalopathy (HE) or hepatorenal syndrome (HRS), require additional considerations. In cases of HE, current recommendations advise continuing the usual diet while optimizing treatment with medications like lactulose or rifaximin. For HRS, there are no specific nutritional adjustments suggested in the literature; management focuses on treating the underlying causes, such as correcting hypovolemia with albumin infusions [23]. CLD can result from various pathologies, including non-alcoholic fatty liver disease (NAFLD), which is particularly associated with metabolic syndrome. Diet and lifestyle modifications play a critical role in managing NAFLD-related CLD. General dietary recommendations for these patients include reducing total fat, saturated fats, trans fats, and fructose, while increasing the intake of polyunsaturated and monounsaturated fats. Although these changes benefit all CLD patients, they are especially crucial for those with NAFLD [24]. Given the significant impact of nutrition on prognosis in CLD, the primary objective of any therapeutic intervention is to meet the recommended caloric and protein intake levels.

Enteral Nutrition

Enteral nutrition involves the administration of nutrients through the gastrointestinal (GI) tract, where absorption occurs via normal digestive processes. Nutritional intake can be achieved orally, directly into the stomach, or rectally. For patients unable to consume food orally due to comorbidities or other limitations, direct gastric administration through a nasogastric (NG) tube or percutaneous endoscopic gastrostomy (PEG) tube is an alternative.

Oral Nutrition: In patients with chronic liver disease (CLD) or cirrhosis, dietary content and distribution must be carefully managed to ensure adequate nutrition. Current recommendations suggest consuming 5–6 small meals daily, emphasizing complex carbohydrates. Lipids should make up 20%–30% of total caloric intake. While protein restriction was historically believed to reduce the risk of hepatic encephalopathy in end-stage liver disease, recent evidence indicates that protein intake should instead be increased to meet the higher nutritional demands of these patients. Diets low in protein are now discouraged [25]. One significant area of study in liver disease is the potential benefit of oral nutritional supplements, particularly branched-chain amino acids (BCAAs), which include leucine, isoleucine, and valine. These essential amino acids cannot be synthesized by the body and must be obtained through diet. Patients with CLD or cirrhosis often have reduced BCAA levels, impairing various functions such as ammonia detoxification. Low BCAA levels have been linked to worsening hepatic encephalopathy and poorer clinical outcomes. Research has shown that long-term oral BCAA supplementation can help prevent progressive hepatic failure and improve outcomes in patients with hepatic encephalopathy. In addition to BCAAs, controlled diets supplemented with casein-based protein mixtures have been associated with reduced bilirubin levels, improved prothrombin time, and lower infection rates. A review. found that oral nutritional supplements in liver disease patients were linked to decreased rates of ascites, infections, and hepatic encephalopathy. Multivitamins are also recommended for CLD patients, although evidence on their benefits remains limited [26].

Addressing Coexisting Conditions

An increasing number of CLD patients are also being diagnosed with celiac disease, creating additional challenges in achieving proper nutrition. Since the recommended diet for CLD emphasizes complex carbohydrates, it is essential that patients with celiac disease consume gluten-free carbohydrate sources. Many BCAA supplements are naturally gluten-free, and protein mixtures can also be sourced gluten-free to accommodate dietary restrictions. While celiac disease introduces added complexity, it can be managed with careful attention to food choices. These principles also apply to other methods of nutritional support discussed below [27-29].

Tube Feeding

When oral feeding fails to meet nutritional requirements, tube feeding becomes the next therapeutic option. Although clinicians may worry about the risk of gastrointestinal (GI) bleeding associated with nasogastric (NG) tubes, research indicates that this risk is minimal and should not discourage the use of NG tubes for enteral nutrition. According to the European Society for Parenteral and Enteral Nutrition (ESPEN) guidelines, tube feeding can be considered for patients unable to achieve adequate nutrition orally, even in the presence of esophageal varices. However, subsequent findings from a small randomized trial suggested that tube feeding could potentially trigger recurrent bleeding in such patients. As a result, guidelines now caution that tube feeding in the presence of esophageal varices requires close monitoring [30]. For long-term NG tube use, smaller diameter tubes are recommended to minimize nasal mucosal irritation. When extended enteral feeding is necessary, clinicians may consider using a percutaneous endoscopic gastrostomy (PEG) tube. However, PEG tubes are often contraindicated in patients with liver disease, particularly those with ascites, bleeding varices, coagulopathy, or other complications of decompensated cirrhosis, making them less commonly used in this population [31].

Comparative Outcomes: Oral vs. Tube Feeding

Research comparing oral diets and enteral tube feeding has yielded mixed results. Studies reported improvements in serum albumin, Child-Pugh scores, and mortality reduction in patients receiving tube feeding compared to those on oral diets [32, 33]. However, a multicenter trial of 99 cirrhotic patients found no significant difference in 1-year survival or liver parameters between those who received short-term tube feeding followed by oral supplementation and those who adhered strictly to oral diets. A review highlighted the variability in outcomes between oral and tube feeding for liver disease patients, concluding that the choice between the two methods depends largely on patient tolerance. Current guidelines recommend maximizing oral intake with a targeted diet and supplements as the initial approach. If oral feeding fails to meet caloric and nutrient needs, tube feeding

should begin within one week to prevent worsening nutritional status and delayed recovery. Among tube-feeding options, NG tubes are generally preferred, given the frequent contraindications of PEG tubes in CLD patients [34, 35].

Tailored Formulas for Tube Feeding

Tube-feeding formulas can be customized to meet the specific needs of individual patients. Standard formulas may be protein-rich or nutrient-dense, especially for patients with fluid restrictions. For those with impaired digestion, hydrolysed formulas may be used. Additional formulations may include branched-chain amino acids (BCAAs) or immune-enhancing components for immunocompromised patients. These options allow for significant flexibility in addressing the unique nutritional requirements of each patient.

Parenteral Nutrition: Parenteral nutrition is a method of delivering nutrients directly into the bloodstream via an intravenous line. It is typically used for patients with liver disease when oral or enteral feeding fails to provide adequate caloric and nutritional intake. It may also be employed in short-term scenarios, such as during prolonged fasting required for medical procedures, or in patients with conditions like compromised airways, encephalopathy, or impaired swallowing reflexes, which make oral and tube feeding challenging. Cirrhotic patients generally require a caloric intake approximately 1.2–1.3 times their resting energy expenditure (REE). Parenteral nutrition provides nutrients in specific proportions:

Carbohydrates: Supplied as glucose, contributing 50%–60% of non-protein energy requirements.

Lipids: Delivered as emulsions of unsaturated fatty acids, accounting for 40%–50% of non-protein energy requirements.

Proteins: Provided through amino acid infusions, at 1.2–1.5 g/kg/day depending on disease severity. These solutions typically have a higher proportion of branched-chain amino acids (BCAAs) and a reduced fraction of aromatic amino acids.

Micronutrients, including water- and fat-soluble vitamins and electrolytes, are often included in parenteral nutrition despite limited evidence of therapeutic benefit. Their inclusion is common because chronic liver disease (CLD), especially alcohol-related, is associated with significant micronutrient deficiencies. Parenteral nutrition is recommended only after oral and enteral feeding methods have been exhausted.

Complications of Parenteral Nutrition

Parenteral nutrition poses several risks, including: Infections and Clotting: Chronic vascular access increases the risk of catheter-related infections and thrombosis. TPN-Induced Liver Disease: Interactions between linoleic acid (a major lipid source) and liver tissue can lead to liver damage. Gastrointestinal Bypass Effects: Total parenteral nutrition (TPN) bypasses the GI system, which may cause severe hunger pains. Refeeding Syndrome: This condition can occur in malnourished patients with severe cirrhosis or CLD who have experienced prolonged nutrient deficiencies. Parenteral nutrition remains a valuable intervention for patients who cannot meet their nutritional needs through other means but requires careful management to minimize associated risks [31, 12].

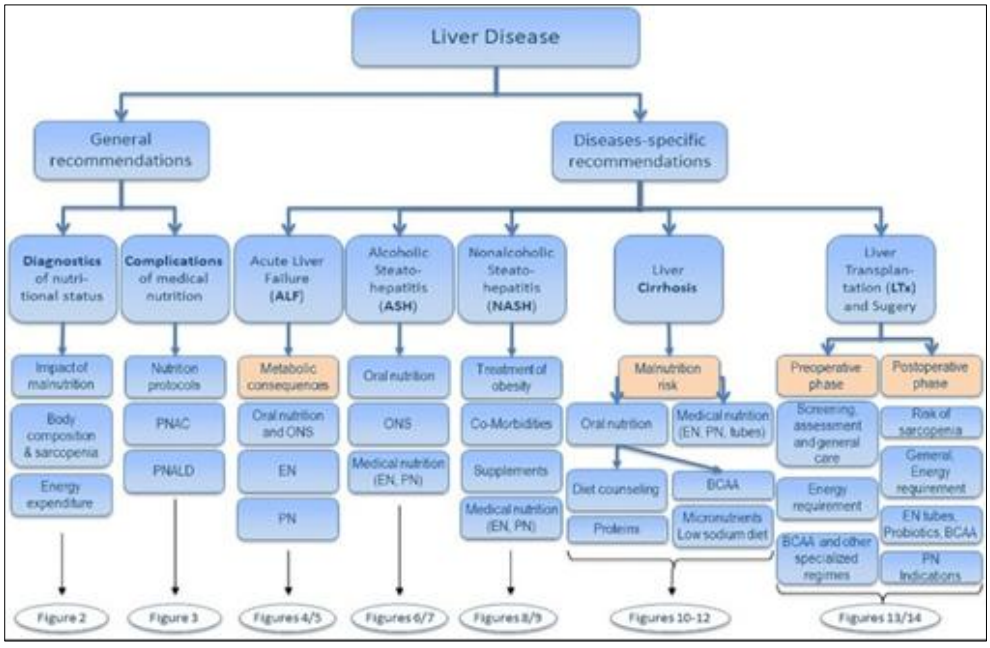

Figure 2: Nutritional guidelines in chronic liver disease Adopted from Bischoff, Stephan C., *et al*. "ESPEN practical guideline: Clinical nutrition in liver disease." Clinical Nutrition 39.12 (2020): 3533-3562.

CONCLUSION

Chronic liver disease (CLD) remains a global health concern due to its high prevalence, significant morbidity, and mortality. Nutritional deficiencies and malnutrition are key contributors to disease progression and adverse outcomes in CLD patients. Addressing these challenges necessitates tailored nutritional interventions that align with the patient's specific condition and nutritional requirements.

Nutritional management in CLD focuses on optimizing energy and protein intake, primarily through enteral feeding methods, which preserve gastrointestinal function and reduce complications. Oral nutrition, enriched with proteins, BCAAs, and micronutrients, serves as the foundation for dietary interventions. When oral intake is insufficient, tube feeding provides an effective alternative, with customized formulas to address individual needs. Parenteral nutrition, though invaluable in cases where enteral feeding is impractical, requires careful monitoring to prevent complications such as infections and total parenteral nutrition (TPN)-induced liver disease.

Emerging evidence underscores the importance of a multidisciplinary approach that integrates nutritional, pharmacological, and lifestyle interventions. Early identification and treatment of malnutrition are essential to improving clinical outcomes, including reduced complications, enhanced quality of life, and extended survival. Research into innovative nutritional strategies and personalized medicine will further enhance the efficacy of nutritional support in CLD patients. By addressing both the metabolic and nutritional aspects of the disease, healthcare providers can significantly influence the prognosis and overall management of chronic liver disease.

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CONFLICT OF INTEREST

The authors do not have any conflict of interest with any individual.

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