Nutritional Considerations in Liver Disease

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KEYWORDS

• Malnutrition • Liver disease • Transplantation

KEY POINTS

- Nutrition is very important in liver disease.
- When it comes to the pathophysiology of malnutrition in liver diseases, malnutrition is multifactorial.
- Cirrhotic patients should avoid fasting for long periods of time.
- Liver transplantation is the only true "cure" of malnutrition in patients with end-stage liver disease.

Malnutrition occurs in up to 80% of patients with cirrhosis and is associated with higher rates of morbidity and mortality.[1] It is often underrecognized and undertreated despite improved outcomes with appropriate management. In this review, the authors discuss the pathophysiology of malnutrition and methods to optimize nutrition status in liver disease and include a brief section on perioperative and postoperative nutrition.

PATHOPHYSIOLOGY OF MALNUTRITION IN LIVER DISEASE

Malnutrition in chronic liver disease (CLD) is multifactorial (**Box 1**).

Decreased Intake of Nutrients

Anorexia and early satiety are well-known causes of decreased nutrient intake in patients with CLD. Anorexia could be explained by the fact that cirrhotic patients tend to have elevated circulating levels of certain proinflammatory cytokines, with known anorexic properties, for example, tumor necrosis factor-α, interleukin (IL)-1B, IL-6, and Leptin.[2–7] Similarly, alcohol-induced anorexia is another important reason for diminished nutrient intake in patients with alcoholic liver disease (ALD). Certain conditions, like esophagitis, gastritis, and pancreatitis, can further contribute to poor dietary

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Box 1
Factors associated with malnutrition in patients with chronic liver disease

Factors

Anorexia

Ascites

Altered taste perception

Metabolic and inflammatory derangements

Inadequate diet restrictions

Decreased social status

Polypharmacy

Multiple paracentesis

Variceal bleeding

Long fasting periods for laboratory tests and diagnostic procedures

intake.[8] Anorexia can also lead to zinc deficiency and hyperglycemia, whereas iatrogenic factors also contribute to poor oral intake. For example, most patients with CLD are advised to consume a low-salt diet in an effort to avoid fluid retention.[9] Consequently, this modified diet is generally unpalatable, leading to a decrease in caloric intake.[9] Many studies recommend against protein restriction given the higher degree of sarcopenia in these patients; however, many providers do not follow those recommendations, which further leads to malnutrition.[10] Furthermore, patients with decompensated liver disease suffer from complications that are generally associated with increased readmissions rates and are frequently kept nil per oral pending examinations and procedures.[8]

Early satiety can occur as a result of impaired gastric accommodation of the stomach likely because of the presence of ascites.[2,11,12] Studies have shown that these patients are also at high risk for the development of functional dyspepsia, secondary to autonomic neuropathy, further causing nausea and early satiety.[5,13]

Decreased Digestion and Absorption of Nutrients

Intestinal digestion and absorption can be impaired by portal hypertension because of congestion by intestinal mucosa.[13] Similarly, patients with cholestatic liver diseases (eg, primary biliary cirrhosis, primary sclerosing cholangitis) have decreased intraluminal bile salt concentration, which leads to fat malabsorption and deficiency of fat-soluble vitamins, such as vitamin A, D, E, and K.[14] Fat malabsorption is also common in patients with ALD, especially with concomitant pancreatic damage. Alcohol itself causes direct toxicity to small intestinal mucosa and brush border enzymes, leading to increased mucosal permeability, impaired salt and water absorption, and rapid intestinal transit.[14,15]

Altered Metabolism

Altered protein metabolism is the most significant metabolic disturbance in patients with CLD. These patients primarily are hypoalbuminemic as a result of decreased hepatic functional capacity and increased amino acid demands.[16] Cirrhotic patients also have diminished hepatic glycogen reserves due to impaired synthetic capacity of hepatocytes.[15] To compensate for this unavailable source of glucose, there is a higher rate of gluconeogenesis, causing mobilization of amino acids from skeletal muscles