Nutrition in Patients with Diseases of the Liver and Pancreas

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Keywords

Cirrhosis · Liver disease · High-protein diet · Acute pancreatitis · Chronic pancreatitis · Obesity-related liver disease

Key Points

- Patients with liver disease are at high risk for malnutrition and should be screened at regular intervals as malnutrition is associated with higher mortality.
- A high-protein diet of 1.2–1.5 g/kg/day is recommended for all patients with cirrhosis. Sodium restriction is reserved for those with ascites or other clinical signs of volume overload.
- Patients with cirrhosis should be screened for vitamin and mineral deficiencies and offered supplementation if necessary. The most common deficiencies are in fat-soluble vitamins.
- Patients with acute pancreatitis should be offered nutrition as soon as tolerable. Those with severe acute pancreatitis are at higher risk for malnutrition and should be offered early enteral feeding if needed.
- Chronic pancreatitis can result in exocrine insufficiency which may be ameliorated with pancreatic enzyme replacement therapy.

Introduction

Diseases of the liver and the pancreas are vast in number and varied in physiology. However, cirrhosis and pancreatitis (both acute and chronic) are two of the most common diseases encountered in general practice. These diseases are highly morbid and often lead to frequent contact with the healthcare system and poor quality of life. One aspect of these diseases that is often overlooked is that of nutrition. Specific evidence-based strategies exist that, when utilized, can meaningfully impact not only the disease morbidity but also patient quality of life.

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Patients with Liver Disease

Cirrhosis is a severe disease with a high mortality rate with as many as two million global deaths each year. Complications of cirrhosis include ascites, gastrointestinal hemorrhage, hepatic encephalopathy, and infection, all of which also confer a high degree of morbidity [1]. While advances in the treatment of hepatitis C virus have reduced mortality due to viral liver disease, the mortality rates associated with alcoholic liver disease and non-alcoholic fatty liver disease (NAFLD) continue to increase [2]. The management of cirrhosis is challenging and requires optimization of many factors, including nutritional status.

Patients with cirrhosis are at high risk for protein-calorie malnutrition (PCM) and its associated complications. Up to 90% of patients with cirrhosis have findings of PCM, and the prevalence of PCM increases with disease severity [3]. Patients with alcoholic liver disease have the highest rates of PCM when compared to patients with cirrhosis due to other etiologies, and PCM is an independent predictor of morbidity and mortality [4]. For this reason, those caring for cirrhotic patients should understand the underlying mechanism of and treatment strategies for PCM in liver disease.

Malnutrition in liver disease arises for several reasons. Patients with ascites and gastrointestinal edema experience nausea and early satiety which leads to poor intake. Those with hepatic encephalopathy may struggle with the mechanics of eating and have little desire to eat. Impairment in bile acid metabolism may result in small bowel maldigestion. Additionally, the liver is a major site of protein catabolism and glycogen storage; disruption of those processes results in malnutrition [3, 5]. Ultimately, malnutrition in patients with cirrhosis results in sarcopenia, an independent predictor of increased mortality.

Patients with liver disease should be screened for PCM through history, physical examination, and objective data. Physical examination identifies temporal and proximal muscle wasting and decreased muscle strength, which indicate severe malnutrition and are less sensitive for those in early stages of malnutrition. As obesity rates increase, it has become increasingly difficult to identify those with significant sarcopenia as BMI does not distinguish between adipose and non-adipose mass, an important distinction when assessing nutritional status [6].

Laboratory measurements including serum albumin, transthyretin (prealbumin), and international normalized ratio (INR) have traditionally been used to estimate body protein stores, and therefore PCM, but they are imperfect measures and susceptible to confounding by concomitant hepatic dysfunction and inflammation. Assessment of skeletal muscle mass on imaging (based on abdominal muscle mass or psoas muscle diameter) is a useful metric, but specialized software is required and is not widely available [5, 7]. These limitations in identification of PCM require clinicians to pay close attention to subtle signs of malnutrition in this patient population.

Dietary modifications are of paramount importance in the management of patients with advanced liver disease. A high-protein diet is an important recommendation that is made to all patients with cirrhosis, regardless of nutritional status. A goal of 1.2–1.5 g/kg/day is recommended to counteract the muscle breakdown that occurs in advanced liver disease. The presence of hepatic encephalopathy does not warrant protein restriction. Very-low protein intake (<0.8 g/kg/day) has been shown to independently affect mortality [8]. For those patients with decompensated cirrhosis or malnutrition, the total calorie goal should be set at 30–35 kcal/day, often divided over 4–6 small meals to combat symptoms of early satiety and anorexia. A protein-enriched bedtime snack can help patients increase their daily protein intake and is associated with improved clinical outcomes.

Sodium restriction is recommended in the presence of ascites or other signs of volume overload. Ascites is the most common complication of cirrhosis and occurs in 50–60% of patients within 10 years of diagnosis. Ascites develops due to portal hypertension, splanchnic vasodilation, and impaired renal sodium excretion. A diet that restricts sodium intake to 2000 mg/day is recommended for patients with ascites. Daily fluid restriction may be warranted in patients with significant dilutional hyponatremia [9].

Appropriate vitamin supplementation is an important consideration in patients with cirrhosis. These patients are often deficient in fat-soluble vitamins due to poor intake and malabsorption. Vitamin D deficiency results in osteopenia and osteoporosis. Repletion is inexpensive and well tolerated. Vitamin A deficiency can result in visual changes, and levels should be monitored periodically, with replacement provided if necessary. Over-repletion of vitamins A and D may cause adverse outcomes, so levels should be monitored during repletion. Patients may also have deficiencies in B vitamins and folate as well as in minerals such as copper and zinc [3]. B1 (thiamine) deficiency is common in those with alcoholic liver disease and may result in neurological dysfunction. Zinc deficiency can cause dysgeusia, and replacement may result in better dietary intake and improved nutritional status [10].

Deficiencies in the three essential branched-chain amino acids (BCAAs) valine, leucine, and isoleucine are common in patients with chronic liver disease. Supplementation with BCAAs may improve hepatic encephalopathy, reduce muscle catabolism, and improve quality of life. BCAAs may be most beneficial in patients with sarcopenia, and supplementation with BCAA is a reasonable choice especially in those patients requiring a supplemental protein source. These supplements are well tolerated but may be difficult to obtain due to poor insurance reimbursement [11].

The prevalence of obesity-related liver disease and cirrhosis is rising. This creates a unique situation in which the need for adequate nutrition is balanced with the need for weight loss. Patients with obesity-related liver disease should be encouraged to lose 7–10% of their body weight. Weight loss to this degree results in reduced hepatic steatosis and decreases the rate of liver disease progression. Strategies for weight loss should include caloric limitation. Recent literature points to the benefit of a Mediterranean-type diet in patients with cirrhosis. This diet, which is discussed more thoroughly in Chap. 15, encourages consumption of fruit, vegetables, and whole grains, while limiting processed sugars and unrefined carbohydrates has been shown to reduce hepatic steatosis and improve insulin resistance [12]. Finally, a high-protein diet, as detailed previously, should be recommended to this patient population, regardless of BMI.

Often, despite diligent effort, patients with chronic liver disease are unable to maintain oral intake due to factors such as hepatic encephalopathy, early satiety, and frailty. In these patients, alternative methods of nutrition are considered. Enteral feeding remains the preferred method for nutrition whenever possible including via feeding tube [13]. Enteral nutritional supplementation should not be initiated without first discussing goals of care and anticipated disease course. Enteral nutritional supplementation may be a necessary choice for those awaiting liver transplantation to decrease the severity of malnutrition and improve surgical outcomes. Total parenteral nutrition is a well-tolerated means of nutritional support but can lead to parenteral nutrition-associated liver disease and bloodstream infections [14, 15].

Patients with Pancreatic Disease

Acute pancreatitis is an inflammatory disease of the pancreas that results in nausea, vomiting, and abdominal pain that may be worsened by oral food intake. The diagnosis of acute pancreatitis is based on the presence of typical epigastric abdominal pain, elevations in serum amylase and lipase, and often cross-sectional imaging showing inflammation of the pancreas. Treatment strategies for acute pancreatitis include early and aggressive intravenous fluid resuscitation coupled with analgesics. Previously, bowel rest was included in this strategy; however, starvation has been associated with increased intestinal inflammation and atrophy. Oral intake should be resumed as soon as clinically tolerated and often can be reinitiated within a few days of presentation [16]. A solid diet is preferred as studies have shown no increased length of hospitalization with initiation of a low-fat solid diet when compared to a clear liquid diet [17].

Severe acute pancreatitis is characterized by a systemic inflammatory response syndrome and can be accompanied by multiorgan failure associated with high morbidity and mortality. Patients are often

unable to tolerate oral intake for an extended period of time and risk malnutrition due to the catabolic nature of the disease process. They should be evaluated for enteral nutrition once the severity of their disease is appreciated. Studies show benefit in starting enteral nutrition within 24–72 h of admission via either nasogastric or nasojejunal tube with no clear preference for formulation of feeds. If patients have difficulty tolerating enteral feeds, strategies such as decreasing the rate of infusion or changing to a different formulation may improve tolerability. Because parenteral nutrition has been associated with higher risk of infection and other complications, its use should be avoided unless absolutely necessary [18, 19].

Chronic pancreatitis is a disease caused by progressive inflammation of the pancreas that leads to fibrosis and eventual loss of both exocrine and endocrine function. The most common etiology of chronic pancreatitis is alcohol use disorder, but it may also result from genetic and autoimmune disorders. Symptoms may be similar to those of acute pancreatitis and include abdominal pain, nausea, and anorexia, all leading to malnutrition. The pancreas acts as an exocrine gland by producing enzymes such as lipase and trypsin that aid in fat digestion. In chronic pancreatitis, acinar cell destruction leads to impaired enzyme production, and overt steatorrhea occurs once enzymatic function falls to <10%. A diagnosis of pancreatic exocrine insufficiency can be confirmed by measuring fecal elastase-1, with low levels confirming the diagnosis [20].

Pancreatic enzyme replacement therapy (PERT) should be initiated in patients with pancreatic exocrine insufficiency but should not be routinely offered in those without exocrine dysfunction. PERT may be started at 20,000–50,000 IU (lipase) with every meal (half dose with snacks) and uptitrated based on response. Some patients may require up to 90,000 IU. Patients should ingest PERT along with meals and snacks and not before or after. Proton pump inhibitors may improve the effectiveness of pancreatic enzyme replacement. PERT is typically effective in relieving steatorrhea and allows patients to eat a normal fat diet. Low-fat diets are only recommended in patients who have steatorrhea refractory to PERT supplementation that can lead to malabsorption of fat-soluble vitamins. Patients with chronic pancreatitis should be screened for deficiencies in these vitamins at least annually [18, 21]. Patients should also be screened for type 3c diabetes mellitus (T3cDM) (pancreatogenic diabetes mellitus). T3cDM may be difficult to manage with oral diabetes agents and often requires insulin therapy. These patients should be counseled on the nutritional goals of a diabetic diet [22].

Even in patients without overt exocrine insufficiency, malnutrition is common. This is due in part to the symptoms of chronic pancreatitis which can be difficult to control. Pain in particular leads to frequent hospitalizations and poor quality of life. Analgesia with non-opiate medications is preferred but is often ineffective. Endoscopic and surgical options to relieve pain are available but must be carefully considered on a case-by-case basis. Patients who are malnourished secondary to pain should be advised to eat small, frequent meals to improve tolerance. Enteral nutrition is sometimes necessary in severe cases, but parenteral nutrition is rarely indicated due to high risk for complications [20].

Conclusion

The management of cirrhosis and pancreatitis is complex and treatment requires manipulation of many variables. Optimal treatment of these diseases should involve early and careful assessment of nutritional status to improve disease outcomes. By working to educate and empower patients to improve nutritional status, quality of life and overall outcomes can be improved. With the information presented, clinicians should be able to identify and treat these patients who are at high risk for malnourishment. Additionally, a multidisciplinary approach involving colleagues who specialize in nutrition should also be considered for all patients.

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Suggested Further Readings

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