

Factors Associated Towards Malnutrition in Liver Cirrhosis Patient: A Literature Review

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Abstract

Malnutrition is a common condition in patients with acute or chronic diseases in the community, and it is usually an under-recognized and undertreated problem facing patients and clinicians. It is frequent in liver cirrhosis patients, and its presence has significant prognostic implications, which worsen as the severity of the disease increases. The clinical factors and pathophysiology mechanisms that trigger malnutrition in liver cirrhosis are varied and interconnected. This literature review aimed to see from the previous study regarding factor associated towards malnutrition in liver cirrhosis patient. It was found that liver plays vital role in controlling the nutritional status and energy balance, and patients with hepatic diseases are particularly susceptible to developing malnutrition. As a result, numerous factors contribute to malnutrition in liver cirrhosis patient.

Keywords : Malnutrition, Liver, Cirrhosis, Nutrition, Complications

1. Introduction

Malnutrition is a common condition in patients with acute or chronic diseases in the community, and it is usually an under-recognized and undertreated problem facing patients and clinicians (1). There is no agreed-upon definition of the term "malnutrition", it has been used to characterize nutrient deficiencies, excesses, and imbalances that negatively impact body composition, function, and clinical outcomes (2). As a fundamental human necessity, nutrition is essential for health promotion and disease prevention. Dietary intake and its regulating mechanisms (such as hunger and satiety) are incredibly complex physiological processes. These processes significantly impact nutritional status, determined by dietary intake, its balanced supply of macro- and micronutrients, and fluid consumption (3). The underlying medical condition may directly impair nutritional intake and generate metabolic or psychological issues that raise nutritional demands or decrease food intake (4). Malnutrition is frequent in liver cirrhosis patients, and its presence has significant prognostic implications, which worsen as the severity of the disease increases. The clinical factors and pathophysiology mechanisms that trigger malnutrition in liver cirrhosis are varied and interconnected (5). A recorded incidence of malnutrition in cirrhosis is widely heterogeneous, extending from 5% to 92%, implying either a knowledge gap, diagnostic issues, or both. A study revealed that only 20% of gastroenterologists correctly identified the prevalence of malnutrition in patients with liver cirrhosis, indicating the knowledge gap (6). Due to the decline in health caused by cirrhosis, an estimated one million people die from cirrhosis worldwide, making it a significant disease burden that must be addressed (7). Malnutrition is aligned with life-threatening complications like refractory ascites, spontaneous bacterial peritonitis, hepatorenal syndrome, and variceal bleeding. Malnutrition also has

substantial implications in liver transplantation, and it has been proven that patients with a poorer nutritional state prior to the transplant have a greater risk of postoperative complications and mortality (8). Although malnutrition is recognized to have adverse effects, few effective therapies are available to counteract such effects in cirrhosis. In theory, malnutrition is a controllable element that can be targeted to alter the course of liver disease and patient outcomes, but this is rarely done in practice.

2. Malnutrition in Cirrhotic Patients

The concept of malnutrition in cirrhosis is quite heterogeneous, primarily because adult malnutrition needs to be clearly defined. Malnutrition in children is defined as mainly protein malnutrition, also known as kwashiorkor, and combined protein and calorie malnutrition, also known as marasmus (9). Most human proteins are found in skeletal muscle; hence, we have characterized clinical adult protein malnutrition as mainly skeletal muscle loss. Although it is more difficult to define, adult fat malnutrition can be described as a decrease in total body fat mass since adipose tissue is the largest storehouse of energy (10). The emergence of malnutrition in cirrhosis is complex, with low nutritional intake, impaired metabolism, and malabsorption being the primary contributors. Patients may be affected by one or more of these etiological causes, each presenting unique obstacles to nutritional assistance and management (11). According to various studies, malnutrition, which manifests as a loss of lean body mass and a reduction in the weight of the skeletal muscles, affects 20% to 60% of patients with cirrhosis. Most studies have concentrated on measuring lean body mass with various tools, yet skeletal muscle makes up between 40% and 50% of lean body mass. Direct measurements made possible by imaging are now considered more accurate indicators of skeletal muscle mass. With increasing liver disease severity as indicated by the Child's score and the emergence of portosystemic shunting, skeletal muscle atrophy in cirrhosis worsens (12,13). Patients with cirrhosis face particular difficulties getting a comprehensive and reliable nutritional assessment. Because of decreased protein synthesis (albumin) and volume overload, which affect body weight, specific nutrition biomarkers are altered in cirrhosis. There are currently few validated screening methods available, and there is no universal agreement on what constitutes malnutrition in this patient population. To identify patients at risk of malnutrition, assessments frequently begin with a nutritional screening questionnaire. A trained dietitian with experience in liver disease should do a more thorough nutritional assessment if this first screening raises any concerns (14).

3. Associating Factor

Due to the vital role the liver plays in controlling the nutritional status and energy balance, patients with hepatic diseases are particularly susceptible to developing malnutrition. Additionally, a chronic liver condition may cause a decrease in appetite, affecting how much nutrition is consumed. As a result, numerous factors contribute to malnutrition in this patient population.

3.1 Decreased intake of nutrient

The pathophysiology of malnutrition in cirrhosis is heavily influenced by the fact that many individuals with the disease have decreased dietary intake (15). A comprehensive understanding of the factors contributing to a negative

energy balance, such as inflammation, early satiety from ascites, hepatic encephalopathy, negative gastrointestinal symptoms, taste alterations, and unappealing dietary restrictions, is necessary in identifying effective therapies to increase food intake (16–18). However, it still needs to be determined which of these elements contributes an essential role. As a result, it is essential to take a patient-specific approach to determine the causes of inadequate nutritional intake and provide effective treatment.

3.2 Metabolic derangements

Many people with cirrhosis also experience hypermetabolism, characterized as resting energy expenditure (REE) 20% higher than predicted. Compared to individuals with a normal metabolic rate, those with a hypermetabolic rate are more likely to be malnourished, have a lower lean body mass, and have a shorter survival time after a liver transplant. Cirrhosis patients already have difficulty meeting nutritional needs, and the condition's associated hypermetabolism further worsens matters (19). Cirrhosis also impairs the metabolism of macronutrients and is a major contributor to malnutrition in liver disease as glycogen storage capacity in the liver drops dramatically (20). Fasting can cause hypoglycemia because glucose utilization is impaired due to insulin resistance. Due to a lack of glycogen, the liver must start producing glucose immediately, which forces the body to rely more on other fuel sources such as muscle glycogen, amino acid deamination, free fatty acid oxidation, and hepatic synthesis of ketone bodies (21).

3.3 Malabsorption

Malabsorption is one of the most important factors leading to a negative energy balance and malnutrition in cirrhosis (22). Fat malabsorption can occur due to deconjugation of bile acids, which is caused by impaired bile acid metabolism, which inhibits the synthesis of micelles necessary for fat digestion and absorption of fat-soluble vitamins, and is frequent in individuals with liver cirrhosis (23). Fat malabsorption may also be caused by chronic pancreatitis, often seen in people with liver cirrhosis due to alcohol consumption (24). Macronutrients may be poorly digested due to the presence of pancreatic insufficiency. The number of calories the body can use is reduced because of the malabsorption of luminal nutrition brought on by medications that affect the gut flora or reduce the availability of bile acids (25). In addition to already poor nutrient absorption and utilization, portal hypertensive enteropathy and the resulting alterations in gut microbiota worsen malabsorption (26).

4. Conclusion

Malnutrition is widespread in people with liver cirrhosis, which worsens as the liver condition worsens. It is relatively straightforward to identify but is frequently disregarded. Malnutrition is a serious and frequently seen consequence of liver cirrhosis that is complicated by several interrelated factors. However, there is still a lack of evidence to guide action, making it difficult to use as a predictive marker in cirrhosis. It can be very challenging to meet the dietary needs of cirrhotic patients because of factors such as anorexia and other symptoms that cause poor oral intake, the catabolic nature of the disease process, and underlying metabolic abnormalities. Considering the complexity of the disease and the need for individualized management, it is clear that education on malnutrition in

cirrhosis for all healthcare professionals who treat patients with liver cirrhosis and the availability of trained dieticians will be crucial in the future management process of malnutrition in cirrhosis.

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References

1. Norman K, Pichard C, Lochs H, Pirlich M. Prognostic impact of disease-related malnutrition. *Clinical Nutrition*. 2008 Feb;27(1):5–15.
2. Saunders J, Smith T. Malnutrition: causes and consequences. *Clinical Medicine*. 2010 Dec 1;10(6):624–7.
3. Reber E, Gomes F, Vasiloglou MF, Schuetz P, Stanga Z. Nutritional Risk Screening and Assessment. *J Clin Med*. 2019 Jul 20;8(7):1065.
4. Imoberdorf R, Meier R, Krebs P, Hangartner PJ, Hess B, Stäubli M, et al. Prevalence of undernutrition on admission to Swiss hospitals. *Clinical Nutrition*. 2010 Feb;29(1):38–41.
5. Chapman B, Sinclair M, Gow PJ, Testro AG. Malnutrition in cirrhosis: More food for thought. *World J Hepatol*. 2020 Nov 27;12(11):883–96.
6. Traub J, Reiss L, Aliwa B, Stadlbauer V. Malnutrition in Patients with Liver Cirrhosis. *Nutrients*. 2021 Feb 7;13(2):540.
7. Siddiqui A, tus S, Parkash O, Hashmi SA. Malnutrition and liver disease in a developing country. *World J Gastroenterol*. 2021 Aug 14;27(30):4985–98.
8. Sharma P, Rauf A, Matin A, Agarwal R, Tyagi P, Arora A. Handgrip Strength as an Important Bed Side Tool to Assess Malnutrition in Patient with Liver Disease. *J Clin Exp Hepatol*. 2017 Mar;7(1):16–22.
9. Periyalwar P, Dasarathy S. Malnutrition in Cirrhosis: Contribution and Consequences of Sarcopenia on Metabolic and Clinical Responses. *Clin Liver Dis*. 2012 Feb;16(1):95–131.
10. Argilés JM, Anker SD, Evans WJ, Morley JE, Fearon KCH, Strasser F, et al. Consensus on Cachexia Definitions. *J Am Med Dir Assoc*. 2010 May;11(4):229–30.
11. Saunders J, Brian A, Wright M, Stroud M. Malnutrition and nutrition support in patients with liver disease. *Frontline Gastroenterol*. 2010 Jul 1;1(2):105–11.
12. Tsien CD, McCullough AJ, Dasarathy S. Late evening snack: Exploiting a period of anabolic opportunity in cirrhosis. *J Gastroenterol Hepatol*. 2012 Mar;27(3):430–41.
13. Montano-Loza AJ, Meza-Junco J, Prado CMM, Loeffers JR, Baracos VE, Bain VG, et al. Muscle Wasting Is Associated With Mortality in Patients With Cirrhosis. *Clinical Gastroenterology and Hepatology*. 2012 Feb;10(2):166–173.e1.
14. Tandon P, Raman M, Mourtzakis M, Merli M. A practical approach to nutritional screening and assessment in cirrhosis. *Hepatology*. 2017 Mar;65(3):1044–57.
15. Ferreira LG, Ferreira Martins AI, Cunha CE, Anastácio LR, Lima AS, Correia MITD. Negative energy balance secondary to inadequate dietary intake of patients on the waiting list for liver transplantation. *Nutrition*. 2013 Oct;29(10):1252–8.
16. Bajaj JS, Lauridsen M, Tapper EB, Duarte-Rojo A, Rahimi RS, Tandon P, et al. Important Unresolved Questions in the Management of Hepatic Encephalopathy: An ISHEN Consensus. *American Journal of Gastroenterology*. 2020 Jul;115(7):989–1002.
17. FERNANDES SA, BONA S, CERSKI CTS, MARRONI NP, MARRONI CA. ALTERATION OF TASTE BUDS IN EXPERIMENTAL CIRRHOSIS. Is there correlation with human hypogeusia? *Arq Gastroenterol*. 2016 Dec;53(4):278–84.
18. Verne GN, Soldevia-Pico C, Robinson ME, Spicer KM, Reuben A. Autonomic Dysfunction and Gastroparesis in Cirrhosis. *J Clin Gastroenterol*. 2004 Jan;38(1):72–6.
19. Mathur S, Peng S, Gane EJ, McCall JL, Plank LD. Hypermetabolism predicts reduced transplant-free survival independent of MELD and Child-Pugh scores in liver cirrhosis. *Nutrition*. 2007 May;23(5):398–403.
20. Aql BA, Scolapio JS, Dickson RC, Burton DD, Bouras EP. Contribution of Ascites to Impaired Gastric Function and Nutritional Intake in Patients With Cirrhosis and Ascites. *Clinical Gastroenterology and Hepatology*. 2005 Nov;3(11):1095–100.
21. Norman K, Valentini L, Lochs H, Pirlich M. Proteinkatabolie und Mangelernährung bei Leberzirrhose – Möglichkeiten der oralen Ernährungstherapie. *Z Gastroenterol*. 2010 Jul 6;48(07):763–70.
22. Romiti A, Merli M, Martorano M, Parrilli G, Martino F, Riggio O, et al. Malabsorption and nutritional abnormalities in patients with liver cirrhosis. *Ital J Gastroenterol*. 1990 Jun;22(3):118–23.
23. Maslennikov R, Pavlov C, Ivashkin V. Small intestinal bacterial overgrowth in cirrhosis: systematic review and meta-analysis. *Hepatol Int*. 2018 Nov 4;12(6):567–76.
24. Li BR, Pan J, Du TT, Liao Z, Ye B, Zou WB, et al. Risk Factors for Steatorrhea in Chronic Pancreatitis: A Cohort of 2,153 Patients. *Sci Rep*. 2016 Aug 15;6(1):21381.
25. Vincent D, Myriam D, Frederic C, Jacques D. Fat Malabsorption Screening in Chronic Pancreatitis. *Am J Gastroenterol*. 2004 Jul;99(7):1350–4.
26. Dahlqvist GE, Jamar F, Zech F, Geubel AP. In-111 transferrin scintigraphy in cirrhosis with hypoalbuminemia: Evidence for protein-losing enteropathy in a small group of selected cases. *Scand J Gastroenterol*. 2012 Oct 2;47(10):1247–52.