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A comprehensive review on influence of protein and sodium on liver cirrhosis

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***Abstract*---**Cirrhosis of the liver is linked to considerable nutritional concerns, leading to severe hepatic consequences and low survivorship. Nutrition is a vital role yet underappreciated component of cirrhosis treatment. As a result, the goals of this study are to determine the associated dietary risks with its pathophysiology to support dietary guidelines for this population of patients. Even in the phase of the disease, alterations in substrate utilization for the production of energy are a common hallmark of liver cirrhosis, resulting in increased breakdown of protein reserves and a tendency to protein-energy malnutrition. Taking supplements with branched-chain amino acid residues has shown promising results in lowering cirrhosis-related complications, but it currently limits gastrointestinal disorders and poor palatability. Furthermore, more significant research into dietary modulation of branched-chain amino acids is needed. Finally, a vital result of this study is the need to enhance the type and effectiveness of diet intervention programs for patients with cirrhosis, precisely when partial or entire food sources are used. To summarize, nutritional treatment of cirrhosis is not a one-size-fits-all solution, but it should be incorporated early in the therapy algorithms to enhance clinical cirrhosis prognosis.

***Keywords*---**liver cirrhosis, protein, sodium, nutrition.

Introduction

Asia has one of the highest rates of liver disease of any continent. Cirrhosis of the liver is the leading cause of liver-related death and morbidity, with yearly mortality rates surpassing 1 million and rising in some countries [1, 2]. Cirrhosis refers to a stage of liver deterioration in which substantial fibrosis changes the

lobular structure of the liver into proliferative nodules, and functional capacities are severely reduced. Complications range from no symptoms in the well-compensated condition to excessive lack of energy, decreased appetite, vomiting, and abdominal distension in mild cirrhosis, itching, loss of muscle mass, brain damage, intraventricular bleeding, abdominal distension, bacteremia peritonitis, and kidney failure in later stages [3]. Poor nutrition (malnutrition), described as a process of perpetual and insufficient oral ingestion that results in a nutritionally deficient state, significant weight loss, and loss of lean muscle mass, is also a severe and common complication among these individuals. As a result, the extent of liver failure is believed to impact patient's quality of life [4–6]. Nutrition treatment is a practical component of multimodal cirrhosis treatment [7] Dietary intervention in the treatment of liver cirrhosis focuses on avoiding and treating malnutrition, preventing liver failure, and controlling disease-related comorbidities. On the other hand, Dietary assessment management is frequently disregarded in these patients [8]. There are currently several guidelines [9-11] for individuals with liver cirrhosis and disease-related comorbidities that include nutritional or dietary suggestions.

Metabolism of protein and role of liver

Proteins, as well as carbs and fats, the other two macronutrients, are all metabolised by the liver. In protein metabolism, the liver has four main activities [12,13]. The first is the production of blood proteins, which are generated in the liver and secreted into the bloodstream to perform a variety of activities [12]. Blood proteins include clotting factors, carrier and transport proteins, hormones, and other related proteins in homeostasis and oncotic pressure management, like as albumin. The liver's second essential function, amino acid interconversion, is also involved. Essential amino acids are those that our organs cannot generate and must be taken from food, whereas non-essential amino acids are those that the body can create. To synthesize the amino acids required by the body, the liver can change the structure of amino acid residues and transfer amino radicals to a keto acid [12]. Many biological functions, including gluconeogenesis, rely on this mechanism [12].

Amino acid breakdown is the liver's third job in protein metabolism, and the byproducts can be used to generate energy (ATP). On the other hand, proteins are not a preferred energy source, although they will be utilised in times of starvation. Urea synthesis is the final of the four primary functions. Because ammonia, one of the consequences of protein breakdown, is hazardous to the body, the liver eliminates it by converting it to urea, which is then expelled by the kidneys [12]. Aside from these four activities, many other hormones in the body, including insulin, glucagon, epinephrine, and steroids, affect protein metabolism [13] with the effects magnified in liver disease. Because proteins play such an essential part in the body, it's easy to see how alterations in protein metabolism caused by the liver disease can induce a slew of physiologic and chemical changes in the body, disrupting homeostasis.

Nutritional intervention-protein needs of cirrhotic patients

Following a thorough assessment of the patient's nutritional state, the most appropriate intervention for each patient should be carried out. Protein limitations were once considered a mainstay of liver disease treatment [14, 15] because of their role in ammonia generation and hepatic encephalopathy (HE) formation. Researchers have looked into several elements of protein consumption, including the amount and type of protein consumed. Many studies have been carried out to find a standard gold treatment; At the same time, they used varied procedures and outcome indicators to analyze their findings; the majority of researchers agree that the earlier recommendations of protein limitations should no longer be followed. In fact, not only are cirrhotic patients' protein requirements higher than healthy patients' due to the changes in protein metabolism and PCM described earlier, and there is some clinical evidence with cirrhosis may also have protein-losing enteropathy, in which portal hypertension causes excessive intestinal protein losses, necessitating even more protein intake [12]. However, many studies have been conducted to indicate no proven link between protein intake and HE, and that patients who restrict their protein intake typically have worse HE and outcomes [15]. This is because, despite consuming less protein, the patients' blood can still have high ammonia levels. The only difference is that this ammonia comes from body protein breakdown and amino acid release from skeletal muscles rather than from food protein metabolism [15].

Sodium and its role in liver health

Volume of blood, heart rate, osmotic balance, and pH of blood are all regulated by sodium. It's another nutrient that can contribute to malnutrition in some people. Due to its impact on water holding capacity and, as a result, the development of edema and ascites, or the build-up of fluid in the abdominal cavity, sodium restriction is frequently the very first diet intervention given to a liver patient. Excess sodium and liquid generate ascites for various reasons, but the most prevalent cause is portal hypertension, which is a typical symptom of liver illness. Portal hypertension, caused by increasing liver fibrosis, is initially partially balanced by splanchnic blood vessel dilatation. However, as liver disease progresses, this compensatory mechanism fails, resulting in a drop in arterial pressure and, as a result, baroreceptor stimulation, which leads to an increased in the renin-angiotensin framework, circulating catecholamines (vasopressin), and, finally, sodium and water holding capacity in the kidneys [16,17]. Fluid backs up in the interstitial tissue when renal sodium and fluid excretion decline, creating edema and ascites as fluid leakage into the abdominal wall cavity [17,18].

Ascites is one of the three primary cirrhosis complications [19] and a significant milestone in the course of chronic liver disease. Ascites can lead to secondary issues such as abdominal pain, discomfort, and difficulty breathing because the fluid inside the abdomen presses against the diaphragm, lungs, and stomach, causing early satiation reflux symptoms. Infection of the ascitic fluid can lead to bacterial peritonitis, which produces discomfort, abdominal soreness, and nausea [18]. Ascites increase the chance of other significant problems, including renal failure, hepatic hydrothorax, or variceal haemorrhage, among other issues that might develop due to paracentesis or fluid removal [20]. All of which support the

necessity for sodium restriction. On the other hand, sodium restriction will only eliminate ascites in about 10% to 15% of individuals.

As a result, different therapeutic choices are required [18,21]. Diuretics are used to promote sodium excretion and fluid elimination through the urine. As previously stated, paracentesis is also done to remove substantial amounts of ascites from the abdomen [18,19]. Patients' desire, enjoyment, and their need to consume a suitable amount of food may be harmed by salt limits since low-sodium foods are unpleasant, resulting in lower consumption of protein and calories in general, which contributes to PCM [21]. As a result, several academics question the necessity sodium limitation.

Although ascites is not a desirable indication of liver illness, they often indicate a patient's transition from balanced to decompensated liver cirrhosis. However, rigorous sodium restriction contributes to and may worsen Protein calorie malnutrition (PCM) in cirrhotic patients [19,21]. Hypernatremia and diuretic-induced renal impairment are potentially possible side effects [22]. As a result, it's critical to properly assess patients and provide them with the medication that will help them the most, based on their signs, symptoms, and severity of the liver disease. According to the American Association for the Study of Liver Diseases (AASLD) position document on ascites management [19], a dietary sodium limit of 2000 mg/day is appropriate for ascites management. Water follows sodium passively. Therefore, fluid restriction is typically unnecessary [19]. Patients with persistent hypertension may benefit from eating about 1500 mg of sodium per day, as recommended by the American Heart Association [23]. Patients on a sodium-restricted diet should get a comprehensive nutrition education on the reasons for the restriction. Even though some cultures adapt to sodium restriction more readily than others [20], many patients still refuse to follow this diet due to its unpalatability. As a result, a dietitian must provide patients alternatives to using salt to flavor meals to improve food intake and compliance. Patients should be aware that craving for salty is an acquired taste that evolves with time.

Conclusion

The liver is an essential organ for keeping a healthy nutritional condition. Nutritional counselling of liver cirrhosis is complicated, and it may necessitate a multifaceted strategy to address various concerns as well as other symptoms that affect nutritional intake. The lack of controlled dietary intervention trials to enhance clinical and nutritional outcomes for individuals with liver cirrhosis is problematic, given the enormous impact of liver cirrhosis on liver-related mortality. This involves implementing a protein-restricted diet in hepatic encephalopathy. There is increasing evidence for manipulating the types of protein types and possibly adding more fermentable fibre to act as "natural" lactulose. Furthermore, involving a dietician earlier in the treatment process is critical for providing aggressive nutritional management and alleviating the high incidence of malnutrition in this patient population. Cirrhosis has resulted in dietary shortages with systemic consequences as irreversible functional impairments have worsened. As a result, the current study was designed to evaluate the nutritional benefit of protein as well as sodium in the context of liver

cirrhosis. As a result, this study is important for doctors, researchers, nutraceutical companies, and anybody involved in nutrition and dietetics to understand the dietary treatment in liver cirrhosis.

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