

THE IMPORTANCE OF ASSESSING NUTRITIONAL STATUS IN LIVER DISEASES

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Abstract

In recent years, increasing attention has been paid to liver diseases. One of the main reasons for this is the growing prevalence of these conditions worldwide every year, including in Uzbekistan, and the impact on the working-age population. Extensive prospective studies conducted in recent years in the United States have recorded new cases of chronic diffuse liver diseases at a rate of 63.9 per 100,000 people annually. Among these, two-thirds are diagnosed with hepatitis C virus (42%) or alcoholic liver disease (22%). Notably, in 20% of patients with chronic diffuse liver diseases, the first diagnosis of liver cirrhosis was made. All these facts highlight the importance of prevention, early detection, and treatment measures for liver diseases, which are not only medical but also of significant social importance.

Keywords: Chronic liver diseases, nutritional status, liver failure, diet therapy.

Introduction

Recent research has shown a correlation between hepatic failure and nutritional deficiency [1]. According to data from Compaillo Hammual, among 396 patients with liver cirrhosis examined, protein-energy malnutrition was identified in 48% of patients in Child-Pugh class A, 51.7% in class B, and 80.3% in class C [2].

Liver nutrients play a crucial role in modulating the physical state of nutritional components, which is vital for digestive processes. In this process, fats are emulsified with the help of fatty acids and phospholipids. The absorption and assimilation of cholesterol and fat-soluble vitamins are directly linked to the liver's functional state. The physiological significance of these processes is supported by the relationship between cholestasis, steatorrhea, and deficiencies of fat-soluble vitamins. These conditions in liver diseases often result from improper nutrition. For example, in liver failure, defects occur in the transport of fatty acids, lipoprotein structure, and clearance of fatty acids in peripheral tissues. In early stages of fasting, glucose levels are maintained by gluconeogenesis. In well-nourished patients, glycogen is stored for approximately 24 to 48 hours; however, due to deficiency of essential nutrients, after this period, glucose is produced solely through gluconeogenesis. As a result of gluconeogenesis, nitrogenous waste products are detoxified in the liver, leading to urea formation. In liver failure, nitrogen residues from amino acids are not properly detoxified, causing an increase in blood glutamine, ammonia, and aromatic amino acids. These changes are characteristic of liver failure and underscore the importance of protein and amino acid requirements in nutrition. Studies on aromatic amino acid clearance in patients with liver cirrhosis have confirmed a link between hepatic encephalopathy and nitrogen-reducing nutrition [3,4]. The





accumulation of aromatic amino acids in blood is primarily related to protein breakdown. Therefore, nutrition should primarily aim to slow down protein catabolism. In patients with liver failure, the ratio of amino acids in blood deviates from normal: aromatic amino acids increase, while the levels of "open chain" amino acids decrease. The use of "open chain" amino acids—such as tryptophan, threonine, leucine, lysine, phenylalanine, methionine, isoleucine, valine, arginine, histidine, proline, and alanine—adequately supplies these amino acids and reduces the risk of hepatic encephalopathy [5,6]. In patients with liver cirrhosis, resting energy expenditure can be high [7], normal [8], or decreased [9]. Resting hypermetabolism is associated with muscle, cellular, and extracellular tissue consumption. Therefore, early detection of hypermetabolism, monitoring of nutrition, and correction of nutritional status are critically important in patients with liver cirrhosis.

Laboratory methods essential for assessing nutritional status include total serum protein, plasma albumin, blood glucose levels, lymphocyte count, total cholesterol, potassium, sodium, serum creatinine, and urea. Additional tests include transferrin, lactate, triglycerides, magnesium, calcium, phosphorus, and iron.

Currently, several methods are available for assessing nutritional status. One of these is the Nutritional Risk Index (NRI), calculated as: $NRI = 1.519 \times \text{plasma albumin (g/l)} + 0.417 \times (\text{current body weight} / \text{usual body weight}) \times 100$, where current body weight is the weight at the time of examination and usual body weight is the patient's normal weight. Based on NRI values, nutritional status is classified as: no malnutrition ($NRI > 97.5$), moderate malnutrition ($97.5 > NRI > 83.5$), and severe malnutrition ($NRI < 83.5$). Other nutritional indices include Subjective Global Assessment (SGA), Nutritional Risk Screening (NRS), and Malnutrition Universal Screening Tool (MUST).

In conclusion, chronic liver diseases are often associated with nutritional deficiencies. Malnutrition negatively affects immune function, particularly cellular immunity. Identifying nutritional deficiencies requires careful treatment, as these conditions can lead to further complications in patients.

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