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Perspective

Optimizing Diet to Support Liver Function and Recovery in Chronic Liver Disease

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DESCRIPTION

Chronic Liver Disease (CLD) represents a growing global health challenge, encompassing conditions such as Non-Alcoholic Fatty Liver Disease (NAFLD), alcoholic liver disease, hepatitis-induced cirrhosis and other forms of long-term hepatic injury. In clinical practice, the role of nutrition in the management of CLD is often underestimated, despite being a cornerstone of both preventive and therapeutic strategies. In this perspective, I argue that nutrition should be regarded not only as supportive care but as an active intervention capable of modulating disease progression, managing symptoms and improving quality of life in patients with CLD.

Malnutrition is a prevalent yet underdiagnosed complication of chronic liver disease. It manifests through muscle wasting, vitamin and mineral deficiencies and impaired metabolic function, contributing to poor clinical outcomes. Particularly in cirrhosis, protein-energy malnutrition is observed in nearly 50%-90% of patients, affecting both compensated and decompensated stages. Despite this high prevalence, nutritional screening is not routinely implemented in many care settings, highlighting a gap between evidence and practice. I believe that integrating systematic nutritional assessments into hepatology care could significantly alter the course of disease and improve patient prognosis.

A key consideration in nutritional management is the preservation of lean body mass. Sarcopenia loss of skeletal muscle is a major predictor of mortality in CLD patients and is frequently compounded by dietary protein restriction, which was historically recommended in cases of hepatic encephalopathy. However, recent evidence has shifted this paradigm, showing that adequate if not slightly increased protein intake is essential for maintaining nitrogen balance and muscle function. Patients with CLD should be encouraged to consume 1.2–1.5 grams of protein per kilogram of body weight per day, with special attention to Branched-Chain Amino Acids (BCAAs), which may support hepatic and muscle metabolism.

Energy requirements in CLD patients are also complex. While some patients may exhibit hyper-metabolism and increased energy expenditure, others particularly those with NAFLD may be overweight or obese with underlying insulin resistance. Thus, personalized energy prescriptions are critical. For malnourished individuals, energy-dense meals and oral nutritional supplements may be necessary. In contrast, those with metabolic syndrome-related liver disease may benefit from caloric restriction and structured weight loss programs, emphasizing whole foods, plant-based diets and reduction of saturated fats and refined sugars.

Micronutrient deficiencies are common in CLD and must be corrected proactively. Fat-soluble vitamins (A, D, E and K) are often deficient due to impaired bile secretion, while minerals like zinc, magnesium and selenium are frequently depleted due to altered absorption or urinary losses. These deficiencies can exacerbate complications such as bone demineralization, immune dysfunction and hepatic encephalopathy. For instance, zinc supplementation has shown promise in improving ammonia metabolism and cognitive function in encephalopathic patients.

In the context of NAFLD and Non-Alcoholic Steatohepatitis (NASH), lifestyle intervention remains the most effective treatment strategy. Dietary modification, particularly the adoption of Mediterranean-style diets rich in antioxidants, monounsaturated fats and fiber, has been associated with reduced hepatic steatosis and improved insulin sensitivity. Weight loss of 7%-10% of body weight has been shown to significantly improve liver histology, even reversing fibrosis in some cases. However, maintaining long-term adherence remains a challenge, underscoring the need for ongoing nutritional counseling and behavioral support.

Another area of growing interest is the gut-liver axis and how dietary components influence liver disease through modulation of the gut microbiota. Diets high in fiber and polyphenols may promote a more favorable gut environment, reducing systemic inflammation and endotoxemia that can worsen liver injury.

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Probiotics and prebiotics are being actively investigated as adjunct therapies for this reason, although more research is needed before they can be universally recommended.

In conclusion, the integration of nutrition into the management of chronic liver disease must move from the periphery to the center of clinical care. It is not merely about preventing malnutrition but about actively using diet as a tool to modulate disease mechanisms, improve functional status and enhance patient outcomes. With the rising burden of liver disease worldwide, there has never been a more urgent time to reframe nutrition as a therapeutic pillar in hepatology.