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Diet Therapy in Patients with Liver Disease and Liver Transplant

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Malnutrition is a common finding in patients with chronic liver diseases. It is described in all the stages of liver cirrhosis, its prevalence reaching as high as 65%–90% in patients with more advanced liver impairment.[1] A variety of mechanisms contribute to the development of malnutrition in cirrhosis; a crucial role is played by an inadequate dietary intake, which is due to multiple reasons such as nausea, loss of appetite, early satiety secondary to tense ascites, and altered gut motility. Recommended diets may be unpalatable because of required sodium restriction when treating ascites; moreover, dysgeusia associated with zinc or magnesium deficiency in cirrhotic patients has been reported. Fat malabsorption from reduced bile acid synthesis, increased intestinal protein losses, and relevant metabolic disturbances such as insulin resistance, increased fat turnover, and protein catabolism are also relevant factors contributing to the alteration of nutritional status in chronic liver disease.

Malnutrition, particularly when the protein compartment is involved, has been shown to increase the risk of complications and to adversely affect the outcome in cirrhotic patients.[2] Impaired nutritional status is an independent risk factor for infection and sepsis in cirrhotic patients; likewise, malnutrition is a recognized factor influencing survival when a cirrhotic patient is hospitalized.[3]

A complete nutritional status assessment is always required at the time of diagnosis in cirrhotic patients. Although accurate quantitative measurements may be hindered by fluid overload and impaired hepatic protein synthesis, simple bedside methods such as anthropometry and subjective global assessment are deemed adequate to identify patients at risk and have been demonstrated to correlate with clinical outcomes. Energy expenditure can be estimated by standard formulas and dietary intake should be carefully evaluated through a detailed interview. Most undernourished cirrhotic patients fail to intake an adequate amount of calories and proteins in their diet and consequently show a negative energy balance even when resting energy expenditure is normal (personal data). Since 1997, European guidelines suggest that an intake of 35–40 kcal/kg/day (dry body weight) and 1.2–1.5 g/kg/day of proteins is desirable in these patients.[4] Furthermore, several studies have shown that a modified eating pattern with 4–7 small daily meals, including at least one late evening snack to avoid prolonged fasting periods, improves the metabolic profile of cirrhotic patients. Although protein restriction was previously considered a therapeutic option for HE, nowadays it is recognized that a low protein diet exacerbates protein breakdown and should be avoided in patients with HE in whom protein requirements are even increased.[5] Branched chain amino acids supplementation may help reach the amount of protein intake needed in patients who are intolerant to dietary protein.[5] An adequate meal at breakfast time has been recently reported to improve cognitive function in patients with minimal HE.[6] Although sodium restriction is considered important to control water retention, a 2 g (88 mmol/L) sodium diet is acceptable to avoid

a too unpalatable diet. Micronutrient deficiencies also need to be taken into account; vitamin B complex is deficient in alcoholic cirrhosis, fat-soluble vitamins are low in cholestatic syndromes and trace elements such as zinc, selenium and magnesium are frequently decreased in chronic liver disease and supplementation may be required to comply with daily requirements. Polyunsaturated fatty acids deficiency is also common in cirrhosis because these are synthesized by the liver; however, the benefit of its supplementation is controversial. It has been suggested that increasing the omega-6/omega-3 ratio in liver diseases may favor a pro-inflammatory pattern. When a cirrhotic patient is severely malnourished and unable to maintain the prescribed calorie and protein intake by following diet recommendations, oral supplements should be added before artificial nutrition is finally commenced either via a nasogastric feeding tube (enteral nutrition) or intravenously (parenteral nutrition).

Malnutrition is known to be associated with a greater surgical risk in patients with liver disease,[7] a correlation that has been confirmed in cirrhotic patients undergoing liver transplantation;[8] recipients malnutrition was found to be associated with increased transoperative blood loss, higher mortality, and higher hospital costs. In our experience, nutritional status before transplantation is independently associated with infection episodes during hospitalization, length of stay in the intensive care unit and days spent in the hospital, suggesting that recipients' malnutrition should be taken into account as a factor increasing complications and transplantation costs.[8] As a result from restored liver function, normalization of energy metabolism, and improved dietary intake, malnourished patients are the ones showing the greater amelioration in nutritional status one year after successful liver transplantation. The development of metabolic syndrome following liver transplantation is still a matter of debate.

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