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ORIGINAL ARTICLE

Effects of an appropriate oral diet on the nutritional status of patients with HCV-related liver cirrhosis: A prospective study

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KEYWORDS
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Summary
Background & Aims: In patients with liver cirrhosis, protein-energy malnutrition is a frequent finding and a risk factor influencing survival. The aim was to estimate the effects of an adequate diet on malnutrition and clinical outcome in patients with Child A or B HCV-related liver cirrhosis.
Methods: We enrolled 90 consecutive outpatients ($M/F = 52/38$) with liver cirrhosis, 30 in Child class A and 60 in class B. Patients were evaluated by anamnesis, clinical examination, estimation of daily caloric intake and measurement of anthropometrical and biochemical indexes. Patients were randomized into two groups: group 1 with a 3-month oral controlled diet started one week after the first examination and this was followed by a 3-month of spontaneous dietary intake, and group 2 which started a 3-month spontaneous dietary intake followed by a 3-month of controlled diet. The follow-up was performed every month.
Results: During the period of controlled diet in patients of both groups, protein malnutrition assessed by midarm muscle circumference, creatinine-height index and serum albumin significantly improved independently of the Child class. Lipid malnutrition, assessed by triceps skin fold thickness values, did not improve during the course of the study. The compliance to the prescribed diet was very high in both groups, and no carry over effect of the previous dietary intake was observed during the follow-up period.
Conclusions: The results emphasize the importance of both nutritional status evaluation and improvement in the Child A and B cirrhotic patients with HCV-related disease. The proposed nutritional approach was able to influence their protein malnutrition positively.

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Introduction

Protein-energy malnutrition is a frequent finding in cirrhotic patients, irrespective of the origin of cirrhosis. Malnutrition is a risk factor influencing survival in patients with this disease and can modify their prognosis.[1–3] It occurs in all clinical stages, but it becomes more severe in the advanced stages as it is related to the degree of the liver injury rather than to its etiology.[2] Abnormalities in nutritional status are associated with a higher risk of clinical complications and a higher mortality rate, and complicate transplantation and other abdominal surgery outcomes by increasing perioperative mortality and morbidity.[4–7]

The pathogenesis of protein-energy malnutrition in cirrhosis involves many factors, including poor oral intake, malabsorption, metabolic abnormalities, increased energy requirements, alterations of substrate oxidation (reduced glucose oxidation and increased lipid oxidation), accelerated protein break-down and inefficient protein synthesis.[8,9] Several studies indicate that cirrhotic patients belonging to class B or C of Child classification, corresponding to those with moderate or severe liver disease, are hypercatabolic, having a deficient whole-body protein turn-over.[10] Furthermore, anorexia too, can play a role, since the liver is an organ primarily involved in the metabolic control of eating, because of the activation of hepatic metabolic signals to the brain stem.[11]

Cirrhosis complications, such as ascites and hepatic encephalopathy, make malnutrition worse. Nevertheless, the therapy for these complications, such as a reduced nitrogen intake for encephalopathy, could potentially represent another cause of malnutrition. Furthermore, micronutrient deficiencies are frequently reported.[12]

Unfortunately, in patients with chronic liver disease, the assessment of the nutritional status is very inaccurate, and most of the traditional indexes used to estimate nutritional state have considerable limitations[13]: body weight and BMI are insensitive indicators because of salt and water retention, and abnormalities of lymphocyte count and plasma protein depletion are produced by liver function impairment, irrespective of malnutrition. A generally proposed index of protein malnutrition is the creatinine-height index (CHI),[14] but in patients with liver disease it seems influenced by the reduced liver synthesis of creatine,[15] while the bioelectrical impedance analysis (BIA)[16] presents certain limitations, since malnutrition assessment is hampered by body water retention.[17]

For these reasons, nutritional status in patients with liver disease is generally better assessed by anthropometric measurements[18,19] such as triceps skinfold thickness (TSF), midupper arm circumference (MAC), and midarm muscle circumference (MAMC).

The aim of the present study was to estimate the effects of an appropriate oral diet on the nutritional status of patients with cirrhosis related to hepatitis C virus (HCV) in Child-Pugh A or B class during an observation period of 6 months.

Patients and methods

This is a prospective, randomized, double period cross-over study. We enrolled outpatients with histologically diagnosed liver cirrhosis of viral origin (HCV), in Child A or B class, consecutively admitted to the Hepato-gastroenterology Unit from January to December 2003.

Patients with HBV infection, autoimmune liver diseases, drug or alcohol abuse, hepatocellular carcinoma, HIV infection, orthotopic liver transplantation, impaired renal function, sepsis or thyroid dysfunction, following specific dietary regimens, ascites and in current or previous treatment with albumin were excluded from the study. Patients that required albumin treatment or that developed ascites during the course of the study were also excluded from the final analysis because it is well known that ascites negatively affects nutritional status. Moreover, the control of ascites[20] and the use of albumin may induce improvement in nutritional status. Finally, we did not include in the study patients in Child C class because of their unstable clinical conditions, due to the occurrence of major complications, and short life expectancy.

The viral etiology of the cirrhosis was defined by routine tests for HCV (third-generation enzyme immunoassay for anti-HCV and RT-PCR for HCV-RNA); the diagnosis of liver cirrhosis was determined on the basis of history, clinical and laboratory data, as well as endoscopic and ultrasonographic criteria and confirmed by liver biopsy.

Patients were evaluated at study entry by dietary anamnesis, clinical examination, estimation of daily caloric intake and measurement of anthropometrical and biochemical indexes. Child-Pugh score was used for assessing liver disease severity.[7]

All patients were interviewed by an experienced clinical dietitian, and their usual food consumption of the previous month was assessed using the cross-check dietary history.[21] This dietary history was considered as representative of the patients' usual dietary habits for a period of at least 3 months.