Nutritional assessment of cirrhotic patients: A new approach based on electrical bioimpedance

Avaliação nutricional do paciente cirrótico: Uma nova abordagem através da bioimpedância elétrica

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The functional integrity of the liver is essen tial for the utilization of nutrients. The liver influences the nutritional status by its production of bile acids and its role in the intermediate metabolism of proteins, carbohydrates, fats and vitamins.[1]

Both acute and chronic liver injuries frequently have nutritional consequences directly proportional to their severity which may progress to hepatic cirrhosis.[1] Hepatic cirrhosis is characterized by chronic and irreversible alteration of the liver parenchyma due to modifications of both hepatic structure and of the functional capacity of the hepatocytes and of the portal circulation.[1] Together, these alterations result in a progressive loss of liver shape and function, compromising to a varying extent the nutritional status and body homeostasis of patients affected by the disease.[1]

Hepatic cirrhosis is multifactorial and, according to its etiology, can be classified as metabolic, viral, alcoholic, drug-induced, autoimmune, biliary, and cryptogenic.[1] One of the methods used to assess the prognosis of hepatic disease is the Child-Pugh (CP) classification, table 1, which considers five factors that may affect prognosis and survival, i.e., ascites, hepatic encephalopathy, bilirubin and albumin levels, and prothrombin time.[2]

Unfortunately, this classification does not consider the nutritional status. Although albumin is considered to be a marker of nutritional status, in this case

Table 1
Child-Pugh classification[2]

POINTS	*1*	*2*	*3*
ENCEPHALOPATHY	ABSENT	1-2	3-4
Ascites	Absent	Mild or controlled with a diuretic	At least moderate despite diuretic therapy
Bilirubin	< 2	2 – 3	> 3
Albumiin (mg/DL)	> 3.5	2.8 - 3. 5	< 2.8
Prothrombin time INR	< 1.7	1.70 – 2.3	> 2.3

CHILD A: 5-6 points CHILD B: 7-9 points CHILD C: 10-15 points

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it may be altered due to changes inherent to the hepatic disease itself and not necessarily to malnutrition. According to the score obtained, patients can be classified as Child-Pugh class A (mild), B (moderate) and C (severe).[2]

Alberino et al[3] demonstrated that malnutrition is an independent predictor of survival and that the inclusion of arm muscle circumference (AMC) and tricipital skin fold (TSF) improves the prognostic precision of the CP classification. Thus, it is probable that nutritional status could be a useful addition to CP classification in the evaluation of the prognosis of cirrhotic patients.[3] The problem is that these anthropometric measures depend on the training of the anthropometrist and are not precise.

Although the CP classification has been the model most widely used to assess the prognosis of cirrhotic patients, its use is limited in individuals with closely similar laboratory markers and due to the subjective nature of measurements for the quantitation of ascites and encephalopathy. Thus, as an alternative for the assessment of prognosis, the Model End-Stage Liver Disease (MELD) has been used.[2] This model uses three laboratory indices: international normalized ratio (INR), bilirubin and serum creatinine and can discriminate in a more effective manner between patients who are likely to die and patients who will survive for at least three months.[4] Like the CP classification, the MELD does not incorporate measurements of nutritional status. The potential prognostic value of adding nutritional status to both the CP classification and the MELD is unknown.

Nutritional status is considered to be a predictor of morbidity and mortality in patients with advanced hepatic disease. Malnutrition also has important implications in liver transplantation and it has been demonstrated that patients with a worse nutritional status before the transplant have increased postoperative complications and higher mortality rates.[5]

In a study on 300 patients, Carvalho et al[6] showed that more than 55% of those with advanced hepatic disease had some degree of malnutrition, which was moderate to severe in 40%. In the same study, 95% of Child-Pugh class C patients were malnourished, as also were 74% of class B and 46% of class A patients.[6] The high prevalence of malnutrition even in patients in the early stages of the disease is a source of concern. Among the causes of malnutrition in these patients, particularly important are insufficient food intake, malabsorption and metabolic disorders.[5] Insufficient food intake may be caused by a series of factors. As is the case for other chronic diseases, anorexia makes a significant contribution to malnutrition, possibly being caused by physical symptoms of discomfort such as nauseas, swelling, fatigue and vomiting. Patients with ascites often have early satiety resulting from the mechanical effects of ascitic fluids that compress the stomach.[5]

In addition, the loss of appetite may be related to the increased regulation of inflammation and of appetite mediators.[5] Besides the hormonal influences and physical discomfort, the lack of interest in food may result from food restrictions and changes in taste.[5]

Dietary limitations such as sodium restriction for the control of ascites, preoperative fast, and limitation of protein intake due to severe hepatic encephalopathy may reduce the variety of foods and many patients do not accept the recommended foods. Although changes in taste may be commonly attributed to micronutrient deficiencies, several investigators have questioned whether they might be a consequence of cirrhosis itself.[5]

It is also important to consider the anorexia related to alcohol. According to the American Liver Foundation, 10% -20% of chronic alcohol users develop cirrhosis. A poor and irregular diet is common among patients with alcoholic cirrhosis.[5]

Malabsorption may be caused by pancreatic insufficiency and cholestasis and may be related to drugs that cause diarrhea (lactulose, antibiotics, diuretics, and cholestyramine).[5] Several mechanisms can lead to the malabsorption of nutrients, fats in particular, in cirrhotic patients. A complication that affects nutrient absorption is the portosystemic shunt. With the progression of cirrhosis, the nutrients bypass the liver through the portosystemic shunt without being processed metabolically.[5] In addition, many patients with cirrhosis due to alcohol abuse have chronic pancreatitis, which contributes to malabsorption.[5]

Another factor that leads to fat malabsorption in patients with cirrhosis is bile deficiency, which impairs the formation of micelles and the absorption of long-chain fatty acids through the usual lymphatic system. Portal absorption of these fatty acids may also occur in patients with cirrhosis.[5]

Among the metabolic disorders we may mention hypermetabolism during complications such as infections, hemorrhage, decompensation and ascites;

increased protein catabolism due to inflammation and impaired hepatic synthesis; reduced glucose homeostasis due to hepatic insulin resistance caused by changes in gluconeogenesis, low glycogen stores and impaired glycogenolysis; increased lipolysis and lipid oxidation, and proinflammatory cytokines (TNFα, interleukins and leptin).[5]

Malnutrition may also be related to iatrogenic causes involved in investigative procedures, to fasting periods, protein restriction during periods of encephalopathy, and to large volumes of paracentesis.[5]

Energy expenditure is also known to contribute to the decline of nutritional status.[5] While most cirrhotic patients have a resting energy expenditure (REE) similar to predicted values, 15% to 30% of them are hypermetabolic. Hypermetabolism may be defined as an REE of 120% compared to predicted values.[5]

The nutritional assessment of these patients is a challenge and should be performed with caution since changes inherent to the liver disease itself such as edemas, ascites and protein changes impair this task, preventing the use of the more traditional parameters for nutritional assessment. The 2006 guidelines of the European Society of Enteral and Parenteral Nutrition (ESPEN) recommend the use of subjective global assessment (SGA), anthropometric analysis and hand grip strength test (HGS) to identify patients with cirrhosis who are at risk for malnutrition.[5] SGA is a rapid tool for assessment used to collect information about food intake, changes in weight and gastrointestinal symptoms, including observations of loss of subcutaneous fat, loss of muscle mass, edema, and ascites. This tool is commonly used to assess patients with hepatic disease because it is simple and inespensive.[5]

Although the traditional anthropometric measurements such as weight, arm circumference and tricipital skin fold are considered to be adequate for the determination of the nutritional status of cirrhotic patients, this assessment should be careful.

Albumin is a poor nutritional marker because it is typically reduced in patients with advanced hepatic disease and fluctuates during periods of inflammation.[5]

The HGS measures the strength of hand and forearm muscles. It is a simple and rapid tool for the assessment of nutritional status, although its use as a single evaluation has not been well established.[5] The muscle strength test was compared to SGA in cirrhotic patients and proved to be a superior predictor of clinical complications such as decompensated ascites, hepatic and bacterial encephalopathy, peritonitis and hepatorenal syndrome. Malnutrition starts within the cells with a disequilibrium of the electrolytes, and muscle function can reflect this more rapidly.[5]

Thus, SGA, anthropometric measurements and the HGS are more commonly used in routine nutritional assessment. However, there is no gold standard method of easy application and low cost, without subjective data and not influenced by the professional who performs it.[5]

The analysis of bioelectrical impedance (BIA) is a sensitive, reproducible, safe and inexpensive method that can be used to determine nutritional status. This method is based on the property of the body of conducting an electric current, which has been known for hundreds of years. Tissues with a greater amount of water, due to the dissolution in electrolytes, are the major pathways of electric conduction, while body fat and bones are considered to be worse conductors.[7] A low alternating electrical current is conducted through a pair of electrodes while another pair, in which impedance is measured, measures the fall in tension. BIA measures parameters such as resistance (R) and capacitance (Xc), recording the fall in tension in the applied current. This change in tension is quantitated geometrically as the angular transformation of the proportion of capacitance to resistance, or phase angle (PA). The PA reflects the relative contribution of fluid (resistance) and cell membranes (capacitance) of the human body.[7]

Lean and fat body mass can be calculated. Lean mass is proportional to the amount of water, considering a 53.2% water constant in lean mass. When lean mass is subtracted from weight, fat mass is obtained. Since cirrhotic patients usually have fluid retention and ascites, i.e., they do not have a normal distribution of body water, this calculation of lean mass and fat mass is not indicated.

However, the PA has been used as a prognostic marker in various clinical conditions in which the integrity of the cell membrane is compromised and changes in fluid balance are observed, such as the malnutrition of advanced neoplastic diseases.[7] This measure has several advantages such as being independent of regression equations and the fact that it can be calculated even in situations in which it is not possible to estimate body composition.[7] In a recent study, Fernandes et al[7] found that the PA is the only parameter for nutritional assessment that is correlated with

the severity of hepatic disease assessed by the CP classification and emphasized the importance of establishing a cut-off point as a parameter for the classification of malnutrition in the population of cirrhotic patients.[7]

Thus, the interest in comparing the PA to other methods used for the nutritional assessment of cirrhotic patients is clearly justified, in order to obtain data about its performance as an indicator of the nutritional status of these patients. In addition, a precise, low-cost and reproducible nutritional parameter could be included in the Child-Pugh and MELD equations, contributing to a better prognosis for patients.

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