

# Nutrition and chronic liver disease

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**W Siriboonkoom, L Gramlich. Nutrition and chronic liver disease. *Can J Gastroenterol* 1998;12(3):201-207.** Malnutrition frequently occurs in patients with chronic liver disease, and may influence both short and long term clinical outcome in these patients. Therefore, nutritional intervention may play an important role in the management of chronic liver disease patients. Recent progress in nutritional assessment, principles of nutrition management and the impact of malnutrition upon the clinical outcome of chronic liver disease are reviewed and summarized. Recommendations on how to manage/prevent malnutrition in these patients will be presented.

**Key Words:** *Chronic liver disease, Nutrition*

## Nutrition et maladie hépatique chronique

**RÉSUMÉ :** La malnutrition s'observe fréquemment chez les patients atteints d'une maladie hépatique chronique et peut influencer sur le pronostic de la maladie, à court terme, comme à long terme. C'est pourquoi une intervention nutritionnelle peut jouer un rôle important dans le traitement de ces patients. Ce article passe en revue et résume les récents progrès enregistrés au chapitre de l'évaluation nutritionnelle, des principes de gestion nutritionnelle et de l'impact de la malnutrition sur l'issue clinique de la maladie hépatique chronique. On y présente en outre des recommandations sur la façon de traiter et de prévenir la malnutrition chez ces patients.

Chronic liver disease is common. It was the ninth leading reported cause of death in the United States between 1980 and 1989 (1). In developing countries chronic liver disease is predominantly caused by viral hepatitis; in more developed countries it is largely related to alcoholic liver disease.

The relationship between nutritional status and chronic liver disease is complex. At a physiological and biochemical level, the liver plays a pivotal role in carbohydrate, protein and lipid metabolism. End-stage liver disease and its complications affect both energy consumption and energy expenditure, which in turn influence nutritional status. Nutritional status itself may affect the outcome of chronic liver disease. This paper explores the relationship between nutrition and chronic liver disease.

### PREVALENCE

The prevalence of malnutrition in chronic liver disease varies according to the severity of hepatic damage and progression of liver disease. It has ranged from 10% to 100% in

different studies, largely depending on the method of nutritional assessment performed and the population studied (2). Although many of the available data on nutritional status in chronic liver disease come from the study of alcoholic liver disease, emerging data indicate that malnutrition is a common finding in both alcoholic and nonalcoholic cirrhosis (3-5).

Caregaro et al (3) evaluated 120 hospitalized patients with liver cirrhosis – 77 with alcoholic and 43 with virus-related cirrhosis. Nutritional assessment included anthropometric measurements, creatinine-height index (CHI), visceral protein status and assessment of immunological competence. Energy malnutrition, defined as triceps skinfold thickness (TSF) and/or midarm muscle circumference (MAMC) below the fifth percentile of standard values, was found in 34% of the study population. Protein malnutrition, defined as low serum albumin, prealbumin, transferrin and retinol binding protein concentration, was found in 81% of the study population.

Immunoincompetence was found in 59% of the study par-

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**TABLE 1**  
**Nutritional assessment in chronic liver disease**

Method	Comments
Anthropometry	
Total body weight	Insensitive, affected by total body salt and water retention
Triceps skin-fold thickness	Provides body fat reserves, less likely affected by salt and water retention; reliability increased when performed by a unique observer
Midarm muscle circumference	Provides muscle protein reserves, less likely affected by salt and water retention; reliability increased when performed by a unique observer
Plasma proteins	
Albumin, prealbumin, transferrin, retinol binding protein	Unreliable, low levels reflect a decrease in liver synthetic function, increased catabolic rate or increased volume of distribution
Immune competence	
Total lymphocyte count, delayed hypersensitivity skin test	Unreliable, affected by many non-nutritional factors such as cirrhosis, drugs (corticosteroids, immunosuppressants)
Creatinine height index	A good predictor of lean body mass, affected by certain diets, trauma, infection and end-stage renal disease

ticipants. The prevalence, characteristics and severity of protein caloric malnutrition (PCM) were comparable in both alcoholic cirrhosis and virus-related cirrhosis patients. The incidence of PCM progressively increases from the Child-Pugh class A to C. Caregaro et al (3) support the argument that malnutrition is related to the consequences of liver injury rather than the etiology of the liver disease.

The Italian Multicentre Cooperative Project in Liver Cirrhosis evaluated the nutritional status of 1402 cirrhotic patients, 37% of whom had alcoholic cirrhosis. Anthropometric measurements and clinical evaluation, such as history of weight loss and body mass index, were employed as nutritional assessment tools. In addition, Child-Pugh criteria were used to evaluate the severity of liver disease. PCM was observed in 30% of patients with cirrhosis. The prevalence of malnutrition increased as liver function deteriorated. Furthermore, patients with alcoholic cirrhosis had a significantly higher prevalence of malnutrition than patients with nonalcoholic cirrhosis; the alcoholic cirrhotics also had more frequent severe liver deterioration (Child-Pugh classes B and C) (5).

The correlation between the nutritional status and the severity of chronic liver disease is somewhat controversial. Although no correlation between nutritional status and severity of chronic liver disease was found by Mills et al (6) or by Thuluvath and Triger (4), larger studies indicated that malnutrition correlated with severity and progression of liver disease in both alcoholic and nonalcoholic cirrhosis patients (3,5,7).

## PATHOGENESIS

The pathogenesis of PCM in cirrhotic patients is multifactorial. Poor dietary intake is common in both ambulatory and hospitalized patients with chronic liver disease. It is due mainly to anorexia, nausea and vomiting, and is compounded by unpalatable low sodium, low protein diets. Other factors – such as impaired nutrient digestion and absorption, either due to bile acid insufficiency, the presence of bacterial overgrowth, associated pancreatic insufficiency or splenic lymphatic hypertension – clarify possible mechanisms leading to PCM (8).

Liver cirrhosis is associated with abnormal fuel metabolism. Accelerated protein breakdown and inefficient protein synthesis play a role in the development of malnutrition (8,9). In cirrhotics, hepatic glycogen stores are depleted (10), the rate of lipid oxidation is increased and the rate of glucose oxidation is decreased (11-13). This metabolic pattern characterizes accelerated starvation, with early recruitment of alternative fuels, and may contribute to malnutrition.

The impact of alteration in resting energy expenditure (REE) in clinically stable cirrhotic patients is ill-defined. Some studies demonstrate an increase in energy expenditure in cirrhotic patients when caloric consumption is expressed per unit of lean body mass (12,14). However, in a larger study carried out in more than 120 patients being evaluated for liver transplantation, only 18% of patients were hypermetabolic, whereas 31% of patients were hypometabolic and 51% were normometabolic. The authors indicated that hypermetabolism is not a constant feature of cirrhosis and results more from extrahepatic factors than from hepatic factors (15).

The measurement of REE in end-stage liver disease may be difficult, and measured REE may differ from predicted values in up to 70% of patients (15). Therefore, it appears that there is a need for further study for precise evaluation of REE in cirrhotics to optimize the ratio of caloric intake to energy expenditure in order to promote the optimization of nutritional status.

## NUTRITIONAL ASSESSMENT

The goal of nutritional assessment is to identify the presence and degree of malnutrition so that nutritional intervention can be pursued. Unfortunately, nutritional assessment in cirrhotics is difficult. Table 1 reveals the methods commonly employed for assessing nutritional status.

In chronic liver disease, in particular end-stage liver disease, total body weight is an insensitive marker of nutritional status due to increase in total body salt and water. On the other hand, TSF provides an estimate of the energy reserves, stored in the form of fat, and MAMC reflects the reserves of muscle protein. TSF and MAMC can be measured fairly accurately in patients with advanced cirrhosis, and are not likely to be affected by salt and water retention because edema accumulates to a lesser extent in the upper extremities (5,16). Interobserver variability is the main limitation of this method of nutritional assessment. Hall and co-workers (17) studied the variance of measurements of 21 patients by

three different observers; the coefficient of variation was 4.7% for arm circumference and 22.6% for TSF. However, when patients with anasarca and/or edema extending to the upper extremities are excluded, and measurements are performed by a unique observer, skinfold anthropometry is considered a reliable method to assess body composition in patients with chronic liver disease. A TSF/MAMC measurement that is in less than the fifth percentile is considered diagnostic of malnutrition (4).

In patients with chronic liver disease, the ability to interpret the nutritional significance of serum proteins is limited by impaired liver function and is considered unreliable. In malnutrition, decreased serum albumin and serum protein levels (such as prealbumin, retinol binding protein and transferrin in part) reflect a decrease in liver protein synthesis, which is due to inadequate synthetic reserve, inadequate protein intake or both. Other causes of hypoalbuminemia and hypoproteinemia in patients with chronic liver disease include an increased volume of distribution and an increased catabolic rate (16).

Although immune competence, as measured by delayed hypersensitivity skin test or total lymphocyte count, is reduced in malnutrition, it is affected by non-nutritional factors such as cirrhosis and drugs (eg, corticosteroids, immunosuppressants) and is considered an unreliable indicator of nutritional status in chronic liver disease (16,18).

CHI is an important laboratory test for detecting PCM. Daily urine creatinine excretion (a breakdown product of lean tissue) is compared with an ideal control value. CHI has been shown to be a good predictor of lean body mass (16). Certain diets can affect creatinine excretion, and trauma and infection can increase creatinine excretion (16). The validity of CHI in cirrhosis is uncertain because creatinine is synthesized in the liver. It has been suggested that impaired liver function rather than reduced muscle mass may cause the low level of urinary creatinine excretion frequently observed in cirrhotics. However, renal insufficiency, which is frequently associated with advanced cirrhosis, may also contribute significantly to the low level of urinary creatinine excretion. A recent study reveals that renal dysfunction, but not impaired liver function, systematically affects the urinary creatinine method for the estimation of muscle mass in cirrhosis (19).

Subjective global assessment (SGA), a clinical assessment of nutritional status done at bedside, takes into account six features of the patient's condition: weight change, dietary intake, gastrointestinal symptoms for more than two weeks, functional capacity, degree of stress and various physical signs. The patients are then scored A, B or C. SGA provides a reproducible assessment of nutritional status and can predict the likelihood of complications related to malnutrition (18). There are limited studies of the validity of SGA in chronic liver disease. In a study of liver transplant candidates, SGA has an overall fair to good interobserver reproducibility rate (20).

More sophisticated body composition analysis also provides evidence for PCM in both alcoholic and nonalcoholic

**TABLE 2**  
**Recommendations for nutritional management in chronic liver disease**

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1. Energy requirement = resting energy expenditure x 1.2 to 1.4  
or  
25 to 30 kcal/kg/day
  2. Protein = 0.8 to 1 g/kg/day in well-compensated nonencephalopathic cirrhotics. May increase to 1.5 to 2.0 g/kg/day in malnourished cirrhotics, especially while enduring major complications of liver disease
  3. Carbohydrate = 50% to 55% of total caloric intake
  4. Fat = 30% to 35% of total caloric intake
  5. Provide appropriate amounts of multivitamins and minerals to correct and prevent deficiency
  6. Provide appropriate amount of salt and water in accordance with patient's volume status
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cirrhosis patients (21,22). Based on the four-compartment model of body composition, Prijatmoko et al (21) assessed body composition by using anthropometry and bioelectrical impedance anthropometry for the determination of fat-free mass; *in vivo* neutron activation analysis (IVNAA) for the determination of total body nitrogen; deuterium oxide dilution for the assessment of total body water; and dual-energy x-ray absorptiometry for the assessment of bone mineral content. These researchers demonstrated an alteration in body composition in alcoholic cirrhotics. In addition, their study revealed a significant and progressive reduction in body protein, expressed as nitrogen index (NI) derived from IVNAA measurement in patients with Child's A, B and C cirrhosis. Furthermore, six of the patients with Child's A cirrhosis had low NI but a normal serum albumin level. This finding indicates that generalized muscle wasting occurs in early cirrhosis, and NI derived from IVNAA measurement is a sensitive method in the early detection of protein depletion in cirrhotics (21).

Another study by Crawford and colleagues (22) in 57 nonalcoholic cirrhotics assessed body cell mass by total body potassium measurement and total body water by deuterium oxide dilution, and then calculated body fat. They demonstrated a reduction of the body cell mass and body fat in cirrhotics. More importantly, they demonstrated that 71% of patients with Child's A cirrhosis had a significant reduction in body cell mass, body fat or both (22).

In summary, the available data indicate that muscle mass, fat stores and body cell mass are decreased in cirrhosis patients. All the methods commonly used for nutritional assessment are influenced by chronic liver disease, independent of PCM, and no single measurement is highly sensitive and/or specific for identifying malnutrition. Therefore, nutritional assessment in cirrhotic patients should be carefully performed and interpreted. Because of the high prevalence of malnutrition in a population of patients with chronic liver disease, there should be a high index of suspicion. The more sophisticated, expensive and difficult technologies, such as body composition analysis, appear to be more sensitive in detecting PCM in early cirrhosis.

## NUTRITIONAL MANAGEMENT

Recommendations for nutritional management in chronic liver disease are outlined in Table 2.

**Energy requirement:** As mentioned earlier, hypermetabolism is not a constant feature of cirrhosis. In general, REE in cirrhotic patients is considered to be similar to that in patients without cirrhosis. In most patients, the daily energy requirement equals  $REE \times 1.2$  to  $1.4$ , which is generally equivalent to 25 to 30 kcal/kg/day (16). However, REE appears to be increased when it is related to lean body mass (2,16). It seems reasonable to calculate energy requirement on the basis of lean body mass by measuring urinary creatinine excretion (2) or by using indirect calorimetry in some patients with severe liver disease (16). Under conditions of metabolic stress, such as the postoperative period, sepsis, gastrointestinal bleeding or fulminant hepatic failure, the energy requirement may be higher (23).

It is crucial to provide adequate calories to sustain protein synthesis. If suboptimal energy is provided, amino acids will be preferentially used for energy generation via gluconeogenesis, and decreased incorporation of amino acids into nascent proteins will occur (23).

**Protein, carbohydrate and fat:** Protein requirement in well-compensated nonencephalopathic cirrhotics does not appear to be increased. Protein intake of 0.8 to 1.0 g/kg/day can enable a positive nitrogen balance. However, malnourished patients with cirrhosis may be considered to have increased protein requirement, especially while enduring major complications of liver disease (23). These requirements may increase to 1.5 to 2.0 g/kg/day (2). During episodes of hepatic encephalopathy, and in individuals who become encephalopathic while receiving larger amounts of protein (protein intolerance), dietary protein restriction is required. Unfortunately this restriction results in a negative nitrogen balance and may lead to a reduction in lean body mass. It has been noted that even modest protein intake (20 to 40 g/day) is preferable to the traditional absolute protein restriction. Several approaches have been tried to deal with the protein-intolerant cirrhotic patients, including the use of standard protein formulas, branched-chain amino acids (BCAAs), vegetable protein diets and nitrogen-free ketoanalogues of the BCAAs. The primary goal is to supply the patients with adequate protein without inducing encephalopathy (23).

Carbohydrate is an important energy source in patients with chronic liver disease. Considerations that must be taken into account with carbohydrate delivery include hyperglycemia in the setting of insulin resistance, which is often seen in chronic liver disease, promotion of hepatic lipogenesis and an increase in carbon dioxide production at high levels of carbohydrate provision, which may lead to increased work of breathing. Carbohydrate delivery should be limited to less than 5 mg/kg/min (24).

Excessive dietary fat can also cause adverse effects. Patients who received 50% of energy from intravenous fat had a higher postoperative mortality rate than patients who received glucose as the only nonprotein calorie source (16). Furthermore, excessive fat intake has been associated with

undesirable alteration of acute phase proteins, hypoxemia and bacteremia. The provision of fat at less than 1 g/kg/day should prevent these complications (24).

On the basis of this observation, Nompleggi and Bonkovsky (16) recommended that 30% to 35% of total caloric intake should be given as fat and 50% to 55% should be given as carbohydrate.

When nutritional therapy is considered, the preferred route of administration of supplementation is oral. Multivitamins and minerals should be given in appropriate amounts to correct and prevent deficiency (16).

If the patient cannot achieve adequate intake by mouth, either due to anorexia or encephalopathy, enteral tube feeding is a safe alternative route of nutrition support and is a more physiological route compared with parenteral feeding. Enteral feeding also has known beneficial trophic effects on the gut and the liver resulting from the first-pass effects of nutrients. Factors that regulate hepatic metabolism and growth include administered nutrients and various hormones, such as insulin and glucagon, which are elaborated in response to these enterally delivered nutrients. The presence of nutrients in the gut lumen is also important to maintain the structure and function of the gastrointestinal tract (16). The presence of esophageal varices is not a contraindication to the placement of feeding tubes. No complications were reported with a fine pore polyvinyl chloride feeding tube and the incidence of variceal bleeding is similar in tube-fed cirrhotics and those who received oral diet (8).

Several enteral feeding formulas are commercially available. Because sodium and water retention are frequently associated with advanced cirrhosis, requiring sodium and fluid restriction, high caloric density, low sodium enteral formulas should be used. Several controlled and uncontrolled trials on enteral nutrition in stable cirrhotics indicate that enteral feeding is well tolerated with a low rate of complications (8). Moreover, there is an improvement in nitrogen balance and in other measures of nutritional status. These supplements have not been shown to exacerbate encephalopathy, azotemia, edema or ascites (16).

Total parenteral nutrition (TPN) should be reserved for patients without a useable gastrointestinal tract or those who cannot tolerate enteral feeding. However, the use of TPN in cirrhotic patients has two potential complications. First, mechanical and infectious complications of central venous lines are high in advanced cirrhosis. Second, the metabolic milieu favouring salt and water retention in patients with chronic liver disease mandates meticulous attention to laboratory data to anticipate potential metabolic complications (8).

## ROLE OF SOME SPECIAL NUTRIENTS

**BCAAs:** In cirrhosis, the alteration in amino acid metabolism is characterized by decreased BCAAs and increased aromatic amino acid. This imbalance has been implicated in the pathogenesis of hepatic encephalopathy and PCM in cirrhotics.

Patients with stable cirrhosis can tolerate higher levels of both standard and BCAA-enriched formula, achieving posi-

tive nitrogen balance with both formulas. Standard amino acid formula supplements also result in an improvement in plasma aminoglycans, and potassium and phosphorus balance. Provision of BCAAs without other essential amino acid does not produce improvement in nitrogen balance (16).

The role of intravenous BCAA in the treatment of acute hepatic encephalopathy is controversial: both positive and negative randomized controlled trials have been published. However, a meta-analysis of this issue shows that intravenous BCAA is effective in the treatment of acute hepatic encephalopathy, but the rate of recovery is not better than conventional treatment with lactulose (8).

The role of oral BCAA supplements in chronic hepatic encephalopathy is not yet established. A recent study attempting meta-analysis of this subject was carried by Fabbri et al (25). Nine studies were selected from papers published from January 1976 to December 1992 on oral BCAA treatment in chronic hepatic encephalopathy. Criteria in the selection of studies included random allocation of treatment regimens; patients with hepatic encephalopathy of the chronic recurrent or chronic permanent type; evaluation of hepatic encephalopathy according to clearly outline criteria, ie, portal-systemic encephalopathy (PSE) index; and measurement of at least three of five variables considered in the calculation of the PSE index (mental state, asterixis, Reitan test, ammonia and electroencephalogram). Because of incomplete reporting of results, Fabbri et al (22) asked the authors of these nine studies to provide individual data. Unfortunately, they received the individual data from only the two largest studies, thus precluding any meta-analysis.

The results of these two well-designed studies, which were the largest of the nine studies, were in favour of oral BCAA supplements. Of the nine studies, these two studies accounted for over 60% of total enrolled patients (165 patients), and calculated quality score of 0.84 and 0.69 (mean  $0.47 \pm 0.22$ ) and carried study periods of three months and three weeks, respectively. On the other hand, the remaining seven studies (64 patients), which had a poor average quality score of  $0.39 \pm 0.15$ , were against oral BCAA supplements (25). Large, multicentre long term studies are needed to provide evidence of definite benefit of oral BCAA supplement in chronic hepatic encephalopathy.

**Glutamine:** Glutamine, a nonessential amino acid, is the most abundant amino acid in humans; 61% of the skeletal muscle free amino acid pool is glutamine. In catabolic states, there is an accelerated synthesis of glutamine, leading to the depletion of intracellular glutamine in skeletal muscle and associated skeletal muscle protein degradation and muscle wasting. Most glutamine released is taken up by intestinal cells as an energy source (16). In this setting, glutamine becomes conditionally essential.

Cirrhotic patients frequently have endotoxemia; one proposed mechanism for this finding is abnormal permeability of intestinal mucosa. Endotoxemia was proposed to stimulate tumour necrosis factor (TNF) and interleukin (IL)-1 production, the latter further stimulating IL-6 production. IL-6 suppresses hepatic albumin synthesis and TNF causes

**TABLE 3**  
**Protein caloric malnutrition as a prognostic factor**

Studies	Patient profile	Outcome
Muller et al (15)	123 cirrhotics undergoing liver transplantation	Improved survival in patients having better nutritional status (BCM >30% BW)
Caregaro et al (3)	120 alcoholic and virus-related cirrhotics	Higher survival rate in patients with MAMC and/or TSF >5th percentile
Merli et al (28)	1053 cirrhotics (alcoholic, viral and cryptogenic cirrhosis)	Higher cumulative survival in patients with MAMA and/or MFA >5th percentile

BCM Body cell mass; BW Body weight; MFA Midarm fat area; MAMA Midarm muscle area; MAMC Midarm muscle circumference; TSF Triceps skin-fold thickness

skeletal muscle protein degradation, contributing to PCM in cirrhosis. Endotoxin also inhibits glutamine uptake and oxidation by the intestinal cells, an effect that parallels abnormal intestinal permeability. Glutamine supplementation increases glutaminase activity, glutamine oxidation and glutamine transport into intestinal cell, and thereby can prevent intestinal injury (26).

According to these mechanisms Teran and co-workers (26) postulated that glutamine supplementation in cirrhosis will preserve the gut barrier and reduce endotoxemia (leading to reduction of protein catabolism), and may directly stimulate skeletal muscle protein synthesis. There is no available study of benefit of long term glutamine supplementation on outcome in cirrhotic patients.

### PROGNOSIS

Malnutrition is an independent risk factor for predicting clinical outcome in patients with chronic liver disease. It is interesting that nutritional status is one of the parameters of the Child-Turcotte classification for predicting mortality in patients undergoing portacaval shunt surgery (2).

PCM has prognostic value in patients undergoing liver transplantation. Muller et al (15), reviewing overall mortality of cirrhotics undergoing liver transplantation, found a striking improvement in survival in patients who had better nutritional status. Other studies have also demonstrated that pretransplantation nutritional status correlates with post-transplantation survival (27,28).

PCM also affects survival in cirrhotics. Caregaro et al (3) demonstrated that patients who had MAMC and/or TSF below the fifth percentile showed significantly low survival rate at three, six, 12 and 24 months. This finding is supported by one recent prospective study from Italy that included over 1000 cirrhotics (29). The presence of PCM was associated with a higher risk of mortality, especially in Child-Pugh class A and B, which may stress the importance of nutritional intervention in the early stages of liver cirrhosis.

Other factors associated with lower survival included Child-Pugh class B or C, presence of ascites, hepatic encephalopathy or esophageal varice, low serum albumin and

**TABLE 4**  
**Nutritional intervention in chronic liver disease**

Study	Patient profile	Therapy	Benefit
Cabre et al (30)	35 severely malnourished cirrhotics	Oral hospital diet in control group, enteral feeding 2115 kcal/day, including seven 1 g BCAA-enriched protein in study group	Improved serum albumin and Child's score, and lower hospital mortality in study group
Mendenhall et al (32)	57 alcoholic hepatitis	Oral hospital diet (2500 kcal/day) in control group Oral hospital diet plus Hepatic-aid (B Braun McGaw; San Juan, Puerto Rico) in study group	Improved nutritional parameters (albumin, retinol-binding protein, transferrin) in supplement group No difference in mortality
Kearns et al (33)	31 alcoholic liver disease	Standard oral diet in controls Casein-based tube-fed supplement (40 kcal/kg/day, and 1.5 g/kg/day protein) in study group	Decreased serum bilirubin and more rapid improvement in encephalopathy in study group No difference in mortality
Fan et al (31)	124 patients undergoing resection of HCC	Oral diet in controls Perioperative TPN (30 kcal/kg/day, protein 1.5 g/kg/day) in study group	Lower morbidity from septic complication in TPN group

BCAA Branched-chain amino acids; HCC Hepatocellular carcinoma; TPN Total parenteral nutrition

prothrombin activity, and high bilirubin level. Table 3 summarizes prognostic value of PCM in cirrhotics.

The benefit of nutritional intervention on survival in cirrhotics have not yet been established (Table 4). Cabre et al (30), studying severely malnourished cirrhotics, found that the hospital mortality was significantly lower in the enteral feeding group than in controls. Fan and colleagues (31) studied patients undergoing resection of hepatocellular carcinoma and noted a significant reduction in the overall postoperative morbidity in the perioperative TPN group because of fewer septic complications, predominantly in patients with underlying cirrhosis who underwent major hepatectomy. Other studies failed to demonstrate the benefit of nutritional intervention on survival (32,33).

Although the survival advantage of nutritional intervention has not been demonstrated, nutrition support has been shown to reduce the incidence of infectious complications (16,34), improve liver function and nutritional status (16), and reduce the frequency of hospitalization. Therefore, the goals of nutrition support in cirrhotics should be to prevent or correct PCM, prevent or correct hepatic encephalopathy and improve overall quality of life (16). In addition, nutritional intervention should be initiated in the early stage of liver disease (29).

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## CONCLUSIONS

PCM is a common feature in both alcoholic and nonalcoholic cirrhosis. Malnutrition likely correlates with severity and progression of liver disease. The pathogenesis of PCM in chronic liver disease is multifactorial and includes poor dietary intake, impaired nutrient digestion and absorption, and impaired protein synthesis.

Nutritional assessment in chronic liver disease is difficult. At present, no single measurement is highly sensitive and/or specific for identifying malnutrition. The more sophisticated body composition analysis appears to be more sensitive than nutritional assessment in detecting PCM in early cirrhosis, but is not readily available.

If formal nutrition support is required, the enteral route is preferred, because of both its positive physiological effects and the decreased potential for infectious and metabolic complications which may be seen with TPN.

The benefit of nutritional intervention on the long term survival in patients with cirrhosis has not yet been demonstrated. Large multicentre long term studies are needed to provide evidence of benefit of nutritional intervention on long term survival. In addition, future efforts should be directed at the role of nutrition support in altering the natural history of liver disease.

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