Special Article

Nutritional Assessment in Cirrhosis

Chonmaitree P, MD[1], Roongsangmanoon W, MD[1]

[1] Department of Medicine, HRH Princess Maha Chakri Sirindhorn Medical Center, Faculty of Medicine, Srinakharinwirot University, Nakhon Nayok, Thailand

Malnutrition is common in cirrhosis. It is associated with mortality and morbidity. Pathogenesis of malnutrition in cirrhotic patients is multifactorial. Nutritional assessment is crucial but often ignored. No gold standard of nutritional assessment tools is established. Many assessment tools have limitations for using in cirrhosis with fluid retention. CT scan to assess sarcopenia is promising. Royal Free Hospital-nutritional prioritizing tool (RFH-NPT) and Liver disease undernutrition screening tool (LDUST) are proposed as screening tools. Complete nutritional assessment should be performed in high risk patients. Vitamin and trace element levels should be checked. Adequate energy (at least 35 kcal/kg body weight) and protein (1.2 to 1.5 g/kg body weight) are recommended. Repeat assessment is advocated. In this review, pathogenesis, nutritional assessment tools and treatment of malnutrition in cirrhotic patients are presented.

Keywords: Nutritional assessment, Cirrhosis, Malnutrition, Sarcopenia

J Med Assoc Thai 2019;102(Suppl.6): 119-26
Website: http://www.jmatonline.com

Malnutrition is one of the common complications in patients with cirrhosis. Malnutrition is associated with mortality and various complications including ascites, hepatic encephalopathy, spontaneous bacterial peritonitis, variceal hemorrhage, and hepatorenal syndrome[1-4]. Malnourished cirrhotic patients had longer hospitalization, ICU stay, higher in-hospital mortality and cost[4,5]. Perioperative morbidity and mortality increase in malnourished patients who undergo surgery[6]. Cirrhotic patients with preoperative malnutrition have higher rate of mortality, transfusion requirement, length of stay, and complications following liver transplantation[7]. Malnutrition is related to poor quality of life[8]. Malnutrition is a predictor of poor clinical outcome even in early cirrhosis[9]. Nutritional status can assess prognosis in cirrhosis when using with Child-Pugh score and Model for End Stage Liver Disease scores[10]. Muscle has important role in energy and protein metabolism. Sarcopenia is associated with mortality and morbidity[11,12].The correlation of malnutrition and cirrhosis is quite complicated.

Prevalence of malnutrition in cirrhosis

Prevalence of malnutrition varies, ranging from 5 to 99% depending on assessment tools[13]. In Thailand, prevalence of malnutrition in cirrhotic patients ranges from 18 to 92%[14]. Severity of cirrhosis is also affected by malnutrition[14,15]. Malnutrition is reported at least 20% in compensated cirrhosis and 50 to 100% in decompensated cirrhosis[1]. Prevalence of malnutrition is 44.5, 75.3 and 94.4% in Child-Pugh A, B and C respectively[15]. Malnutrition can be found in all types of cirrhosis particularly in alcoholic cirrhosis[16,17].

Correspondence to:

Chonmaitree P.

Department of Medicine, HRH Princess Maha Chakri Sirindhorn Medical Center, Faculty of Medicine, Srinakharinwirot University, 62 Moo 7, Rangsit-Nakhon Nayok Road, Ongkharak, Nakhon Nayok 26120, Thailand.

Phone : +66-37-095085

E-mail: piyanant_n@yahoo.co.th

Etiology of malnutrition in cirrhosis

Pathogenesis of malnutrition in cirrhosis is multifactorial including decreased dietary intake, impaired digestion and absorption, and altered metabolism[18-20].

Decreased dietary intake can occur from many reasons. Increase in inflammatory cytokines, particularly tumor necrosis factor-α, increase in leptin, and alcohol consumption are leading to anorexia[21-23]. Early satiety, nausea and vomiting may occur in cirrhosis with tense ascites[24]. Diuretic can cause dysgeusia (impairment of taste sensation) from zinc and magnesium deficiency[25]. Salt-restricted diet will make food unpalatable. Acute illness from hepatic encephalopathy, sepsis, and gastrointestinal bleeding may be the cause of inadequate intake. The process of investigation and endoscopy can even make cirrhotic patients malnourished due to fasting before those procedures.

Impaired digestion and absorption involve multiple processes and are poorly defined. Fat malabsorption may develop as a result of chronic alcoholic pancreatitis, small bowel bacterial overgrowth, portal hypertensive enteropathy, and decreased bile acid production from cholestasis[19]. Altered intestinal motility, change of pH in intestine and increase in intestinal permeability may also lead to malabsorption.

Metabolism in cirrhosis has changed. Increase in insulin resistance, hyperinsulinemia, and impairment of hepatic glycogen storage lead to gluconeogenesis[19]. Lipid and protein catabolism are accelerated. Amino acid from muscle is used. Protein synthesis is impaired in late stage of cirrhosis[20]. Muscle wasting and hyperammonemia develop as a consequence of abnormal amino acid metabolism. Resting

How to cite this article: Chonmaitree P, Roongsangmanoon W. Nutritional Assessment in Cirrhosis. J Med Assoc Thai 2019;102(Suppl.6): 119-26.

energy expenditure (REE) is increased. From the study, 33.8% of cirrhosis were in the hypercatabolic state and 41% of hypermetabolic patients had REE more than 30% above the predicted value[26]. Endotoxinemia, inflammatory cytokines and/or the beta-adrenergic system may be the cause of hypermetabolism[23]. Ascites may increase REE. Cirrhosis is in the accelerated state of starvation. After overnight fasting, carbohydrate metabolism in cirrhotic patients shifts to fat metabolism and derives energy from protein catabolism higher than healthy subjects[27]. Fifty-eight to seventy-five percent of energy received from fat oxidation after overnight fasting[28]. After a 12-hour fasting, cirrhosis derived energy from fat, carbohydrate and protein comparable to 3 days of starvation in the healthy[29].

Micronutrient deficiency is common in cirrhosis, particularly vitamin A, D, B12, folate, thiamine, and pyridoxine. Vitamin deficiency develops as a result of cholestasis and portal hypertensive enteropathy. Diuretic use leads to zinc and magnesium deficiency.

Nutritional assessment in cirrhosis

Nutritional assessment in cirrhosis is essential. Goal of nutritional assessment is to identify patients at risk for morbidity and mortality and quantify severity. Ideally, nutritional assessment tools should be simple, easy to use by non-expert personnel, less time consuming, reasonable sensitivity and specificity, and reproducible. Although multiple methods are proposed, gold standard of nutritional assessment in cirrhosis is absent. In clinical practice, nutritional assessment is ignored. Several problems are mentioned. Definition of malnutrition in cirrhosis is not well-defined. Most of nutritional assessment tools are complicated, time consuming, require experts and special equipments, lack of objectivity, reproducibility and prognosis discrimination. Many tools have limitation in decompensated cirrhosis. Different methods have diverse results. Detailed nutritional assessment in all cirrhosis is not practical. Nutritional status in compensated cirrhosis is similar to the healthy. Experts advise rapid nutritional screening in all cirrhosis and further complete nutritional assessment in high risk patients including Child-Pugh C cirrhosis[18]. Screening tool in cirrhosis is inconclusive. Nutritional assessment should be repeated but proper interval is not defined.

Nutritional assessment comprises of anthropometry, body composition assessment, functional assessment, dietary assessment and global assessment tools.

Anthropometry

Anthropometry includes measurement of body weight, height, body mass index (BMI), triceps skinfold (TSF), mid-arm circumference (MAC), mid-arm muscle circumference (MAMC), and mid-arm muscle area (MAMA).

Body mass index (BMI) is defined as body weight divided by the square of the height. Adjusting BMI for fluid retention is not defined. Postparacentesis body weight or scale weight minus ascites weight (mild 5%; moderate 10%; severe 15%; additional 5% if bilateral pedal edema) was proposed[30]. BMI can detect malnutrition in cirrhosis. BMI of 22, 23 and 25 kg/m^2 in cirrhosis with no ascites, mild ascites and tense ascites, respectively, were the optimal cutoff values for detecting malnutrition. Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) were 86.2, 87.7, 92.7 and 92.3%, respectively. Malnutrition in this study was defined by MAC and TSF[31]. BMI was less accurate than other anthropometry[32]. BMI is often overestimated in cirrhosis with ascites or edema.

Skinfold measurement is the method to evaluate fat reserve and can be performed at many sites in the body. Triceps skinfold (TSF) is usually used in cirrhosis because this site is less influenced by fluid retention. TSF is measured by caliper at midpoint between acromion and olecranon. TSF was more sensitive than BMI for detecting malnutrition in cirrhosis. Malnutrition was detected by TSF in 72% of patients while BMI detected only 8% of patients[33]. While comparing with others anthropometry, TSF was the most efficient and associated with mortality. Two thirds of patients who had severe malnutrition and died were detected by TSF[33]. In cirrhosis without fluid retention, skinfold measurement estimated body fat comparable to dual-energy x-ray absorptiometry (20.3 ± 7.7 vs. 20.3 ± 7.7 kg, P = NS)[34]. The study of TSF in evaluating malnutrition showed conflicting results, interobserver variability, and did not correlate with Child-Pugh score[35].

Mid-arm circumference (MAC) is measured at midpoint between acromion and olecranon in nondominant arm[28]. Mid-arm muscle circumference (MAMC) is derived from TSF and MAC. MAMC is the marker of lean tissue stores and not affected by fluid retention[36]. MAMC below 5th percentile means severe malnutrition[31]. Malnutrition evaluated by MAMC or TSF was the independent predictor for survival[37]. MAMC and TSF were correlated with Child-Pugh score[28,38]. MAMC and TSF combining with Child-Pugh score improved accuracy of prognosis[37]. MAMC predicted prognosis better than TSF (HR of survival 6.9, 95% CI 2.7 to 18 vs. 5.7, 95% CI 2.3 to 13.7)[37]. Mid-arm muscle area (MAMA) is derived from MAMC. In patients undergoing liver transplantation, malnutrition assessed by MAMA and TSF had higher bacterial infection and mortality[39].

Body composition

Body composition can be measured by bioelectrical impedance analysis (BIA), dual energy x-ray absorptiometry (DXA), magenetic resonance spectroscopy, Deuterium oxide dilution, and air displacement plethysmography. Body cell mass (BCM) is the metabolically active compartment of lean tissue. BCM is the method to evaluate protein calorie malnutrition. Reduction of BCM and body fat was demonstrated in cirrhosis even in mild disease[40]. Loss of fat was found in the initial stage and loss of body mass was found in the late stage of cirrhosis[40]. MAMC less than 23 cm. combined with HGS less than 30 kg was sensitive for detecting BCM by isotope dilution[41].

Bioelectrical impedance analysis (BIA) uses electrical conduction through the human body and measures conductivity and resistance. BIA is easy, noninvasive, and reliable methods for assessment of muscle mass and adipose tissue. BCM had negative correlation with MELD score in cirrhotic patients without ascites and may be used as nutritional assessment tool[42]. Cirrhosis with BCM of less than 35% of body weight was associated with post liver transplantation mortality[43]. In one study, BIA could evaluate BCM in cirrhosis with or without ascites[44]. Study comparing BIA with TSF showed that BIA should not be used for assessing fat stores[45]. Single-frequency BIA is not recommended for evaluating malnutrition in cirrhosis because it has low accuracy in cirrhosis with fluid retention[41,45]. Multi-frequency BIA is less interfered by fluid retention and further study in cirrhosis is required[46]. Phase angle (PA) obtained from BIA. PA is the relationship between fluid (resistance) and cellular membranes (capacitance) of the human body[46]. PA was not affected by fluid retention[47]. PA of less than or equal to 4.9° correlated with mortality (HR 2.15, 95% CI 1.18 to 3.92)[48], anyway a reference value for diagnosis of malnutrition in cirrhosis is not well defined.

Dual-energy X-ray absorptiometry (DXA) measures fat and lean mass by comparing absorption of two different energies of X-ray. Anthropometry and subjective global assessment (SGA) underestimated malnutrition in Child-Pugh A and B cirrhosis when comparing with DXA[49]. DXA is high-cost, requiring expertise, not widely available, cannot be performed at bedside, and less accurate in cirrhosis with fluid retention[50].

Muscle involves in ammonia metabolism and it is used as energy source in fasting state. Malnutrition in cirrhosis predominantly occurs in lean body mass (protein) and adipose tissue[20,46]. Muscle mass depletion is the central core of nutritional assessment in cirrhotic patients[18]. Sarcopenia means low in muscle mass and function (strength or performance)[51]. It is the objective assessment and not interfered by fluid retention. Sarcopenia can be found in normal or overweight cirrhotic patients. If muscle mass depletion occurs with increase adipose tissue, it is called sarcopenic obesity. Skeletal muscle index less than 50 cm^2/m^2 in male and 39 cm^2/m^2 in female were the optimal cut-offs to define sarcopenia in end-stage liver disease awaiting liver transplantation[54]. Inclusion of sarcopenia within MELD (MELD-sarcopenia) improved prediction of mortality (C-statistics 0.73 , 95% CI 0.70 to 0.77)[55]. Cirrhotic patients with sarcopenia had a higher mortality and complication, including hepatic encephalopathy and ascites[11,16,18]. Impaired quality of life and response to stress was shown in sarcopenic patients[35]. Cirrhotic patients with sarcopenia had poorer outcome after liver transplantation (HR = 3.7 per 1,000 mm^2 decrease in psoas area; $p = 0.0001$)[56]. Sarcopenia did not correlate with Child-Pugh score or MELD[11]. Thigh muscle thickness (measured by ultrasound) combining with BMI had similar accuracy with CT or MRI[30]. However, its use is limited in obesity and requires study to demonstrate correlation with clinical outcome. Anthropometry usually did not correlate with sarcopenia. Limitation of evaluation of sarcopenia by CT or MRI is that it is high-cost, less availability, have risk of radiation and contrast exposure. Moreover, muscle depletion is different between genders causing limitation of muscle assessment[46].

Functional assessment

Muscle strength is one of the predictor of malnutrition. It deteriorates in cirrhosis with decompensation and malnutrition. Muscle strength declines faster than muscle mass[18]. Functional assessment in cirrhosis usually evaluated by handgrip strength (HGS) and 6 minutes walk test (6MWT).

Handgrip strength (HGS) is measured by hand dynamometer. It is easy to perform and can predict sarcopenia. Patients should squeeze the dynamometer with maximal strength three times and the maximal value is chosen. The value is compared with age, gender-adjusted reference. Malnutrition is defined as HGS of less than 2 SD from the mean of age and sex groups[57]. HGS had been compared with Subjective global assessment and Prognostic nutritional index, HGS was the only predictor of 1-year major complications (uncontrolled ascites, hepatic encephalopathy, spontaneous bacterial peritonitis and hepatorenal syndrome)[57]. This study had some limitations because it used SGA as gold standard and most of patients were Child-Pugh A cirrhosis. HGS is simple, rapid, low-cost and not interfered by fluid retention. Its use is limited in uncooperative patients.

Six minute walk test (6MWT) predicted mortality in cirrhotic patients ($p = 0.024$)[58]. Severity of cirrhosis assessed by Child-Pugh score had negative correlation with six minute walk distance (6MWD, r = -0.328, $p<0.01$)[58]. 6MWT is limitedly used in cirrhosis with hepatic encephalopathy.

Global assessment

Subjective Global Assessment (SGA) comprises of 5 clinical parameters and 3 physical examinations. SGA classifies patients into 3 groups: nourished (A), moderate malnourished (B) and severe malnourished (C). SGA had a strong association with Child-Pugh score[14]. SGA can be used as a tool to assess nutritional status after protein supplement[59]. SGA may be used to identify sarcopenia in men but not in women[33]. SGA underestimated malnutrition in cirrhotic patients comparing with DXA[49]. SGA predicted in-hospital mortality[14]. SGA was less predictive of 1-year major complications than HGS[57]. In cirrhosis undergoing liver transplantation, malnutrition diagnosed by SGA had more transfusion requirement, ventilatory support and length of stay[39,60]. SGA is relatively simple, low-cost and easy to perform. Its use is limited in uncooperative patients.

Royal Free Hospital Subjective Global Assessment (RFH-SGA) combines subjective assessment of nutritional status with BMI, MAMC, and dietary intake[61]. It is simple, inexpensive, takes short time to assess. RFH-SGA was associated with mortality in cirrhotic patients[62]. In another

Table 1. Nutritional assessment tools

Method	How to assess	Advantage	Disadvantage
BMI	Body weight/height^2	-Simple, short time to assess	Interfere by fluid retention
TSF	Use caliper to measure midpoint between acromion and olecranon	-Simple, short time to assess, low cost -Less influenced by fluid retention -Sensitive than BMI -Predict mortality -Correlated with severity	Interobserver variability
MAC	Measure at midpoint between acromion and olecranon in nondominant arm	-Simple, short time to assess, low cost -less influenced by fluid retention -Correlated with severity	
MAMC	MAC-(TSFx0.314)	-Simple, short time to assess, low cost -Less influenced by fluid retention -Predict mortality -Correlated with severity -Predict prognosis better than TSF	
MAMA	(MAMC)^2/4x0.314	-Simple, short time to assess, low cost -Less influenced by fluid retention -Predict post LT mortality	
BIA	Electrical conduction through the human body	-Simple, noninvasive, low cost, portable -Less interfered by fluid retention -Predict post LT mortality -PA correlated with severity	-No cutoff value
DXA	2 different energies of x-ray		-High cost -Radiation exposure -Require expertise -Not widely available -Cannot perform bedside -Interfere by fluid retention
Sarcopenia	CT scan	-Less interfered by fluid retention -Predict mortality and complication -Predict post LT outcome	-No cutoff value -High cost -Less availability -Risk of radiation and contrast exposure
HGS	Squeeze hand dynamometer 3 times	-Simple, low cost -Less interfered by fluid retention -Predict sarcopenia -Predict complication	
6MWT		-Predict mortality -Correlated with severity	
SGA	5 clinical parameter and 3 physical examination	-Simple, low cost -Fair to good inter-observer reproducibility -Predict inhospital mortality -Correlate with severity -Predict post LT complication	-Less predictive of complication than HGS -Underestimate comparing with HGS -Low agreement with other methods
RFH-SGA	Subjective assessment, BMI, MAMC, dietary intake	-Simple, inexpensive, short time to assess, reproducible -Predict mortality -Predict post LT complication	
RFH-NPT	Subjective and objective assessment (alcoholic hepatitis, fluid over load, BMI, weight loss, acute illness, dietary intake)	-Simple, short time to assess -Excellent intraobserver and interobserver reproducibility -Good external validity -Predict clinical deterioration and mortality -Correlated with severity and complication	
LDUST	6 questionaire	-Simple, short time to assess -Can be use in outpatients -High PPV -Better than MUST	Low NPV

BMI = body mass index; TSF = triceps skinfold; MAC = midarm circumference; MAMC = midarm muscle circumference; MAMA = midarm muscle area; BIA = bioelectrical impedance; PA = phase; DXA = dual-energy x-ray absortiometry; CT = computer tomography; HGS = handgrip strength; 6MWT = 6 minutes walk test; SGA = subjective global assessment; RFH-SGA = Royal Free Hospital subjective global assessment; RFH-NPT = Royal Free Hospital-nutritional prioritizing tool; LDUST = Liver disease undernutrition screening tool

study, malnutrition assessed by RFH-SGA had shorter survival (χ^2 15.04; df = 2; p = 0.0005)[61].

Royal Free Hospital-Nutritional Prioritizing tool (RFH-NPT) comprises of alcoholic hepatitis, fluid overload, BMI, weight loss, acute illness, dietary intake and classified patients into 3 groups (low, moderate and high risk)[13,63]. It is easy and less time-consuming. Comparing with RFH-SGA, sensitivity of RFH-NPT for detecting patients with high risk for malnutrition was 100%[64]. RFH-NPT was proposed to be a screen nutritional tool in cirrhosis[16,63]. RFH-NPT predicted clinical deterioration (HR 1.814, 95% CI 1.314 to 2.505) and transplant-free survival (HR 1.589, 95% CI 1.26 to 2.164)[65]. It correlated with severity of disease and complications[65].

Liver Disease Undernutrition Screening Tool (LDUST) is the rapid, easy to perform and can be used in outpatient setting. It comprises of six questions. LDUST had high positive predictive value (93%) but low negative predictive value (37.5%)[18]. LDUST was better than Malnutrition Universal Screening Tool (MUST) in detecting malnutrition in cirrhosis[66].

Other tools

Prothrombin time, albumin, prealbumin, creatinine height index, delayed-type hypersensitivity reaction are less reliable for assessing malnutrition in cirrhosis[35]. Serum albumin and prealbumin was correlated with severity of liver disease and degree of inflammation rather than nutritional status[67]. Albumin had low sensitivity and positive predictive value for detecting malnutrition in cirrhosis[36].

ESPEN guideline recommended nutritional assessment using SGA or anthropometry (TSF and MAC). Phase angle and body cell mass can be used[6]. Vitamin level should be checked in patients with symptoms and cholestatic liver disease. Check for trace elements every 6 months is adviced[6]. EASL proposed nutritional assessment algorithm in cirrhosis[63]. Nutritional screening should be done in all cirrhotic patients. Child-Pugh C cirrhosis or BMI of less than 18 kg/M^2 was stratified in high risk group. In patients with BMI 18.5 to 29.9 kg/M^2, nutritional screening by RFH-NPT or LDUST should be performed. High risk patients should undergo detailed nutritional assessment and CT scan to assess sarcopenia. Detailed nutritional assessment comprised of global assessment tools (SGA, RFH-SGA) and detailed dietary intake assessment. Medium risk patients should undergo detailed nutritional assessment. CT scan should be performed in malnutrition patients stratified by detailed nutritional assessment. DXA and BIA can be used in patients with no fluid retention. Outpatients should be reassessed every 1 to 6 months. Inpatients should be assessed at admission and reassessed periodically during hospital stay.

Treatment of malnutrition in cirrhosis

ESPEN guideline recommended energy of 35 to 40 kcal/kg dry body weight and protein of 1.2 to 1.5 g/kg body weight (maximum 100 g/day) in cirrhotic patients[6]. ASPEN guideline recommended energy of 25 to 35 kcal/kg body weight in cirrhosis without hepatic encephalopathy, 35 kcal/kg body weight in cirrhosis with hepatic encephalopathy, and 30 to 40 kcal/kg body weight in stable cirrhosis with malnourished[19]. EASL recommended energy should not be lower than 35 kcal/kg body weight and protein of 1.2 to 1.5 g/kg body weight[63]. In patients with ascites, dry weight should be used for calculation. Carbohydrate of 45 to 65% of daily caloric intake is recommended[19].Cirrhotic patients who receive protein 1.2 g/kg body weight had lower ammonia, Child-Pugh score, and encephalopathy than patients who receive protein 0.8 g/kg body weight[20]. Protein from vegetable is advised. It contains high quality amino acid which less converts to ammonia. Fiber in vegetable slows transit time and decreases ammonia absorption[20]. Clinical course of hepatic encephalopathy in patients receiving protein restriction was not different from patients receiving normal protein[68].Whole protein formula is recommended. Standard and branched chain amino acid (BCAA)-enriched food had similar benefit[69]. BCAA is effective in treatment of hepatic encephalopathy. BCAA has time to recovery from hepatic encephalopathy similar to lactulose[69]. Cirrhotic patients who develop hepatic encephalopathy during enteral nutrition, BCAA should be given[6]. One year nutritional supplement with BCAA significantly reduced combine event rate (death, need of hospitalization and duration of hospitalization) in advanced cirrhosis[71]. Two years of BCAA improved event free survival, quality of life, and liver function in decompensated cirrhosis[72]. Intravenous BCAA did not improve cerebral function and mortality[73]. Limitation of oral BCAA is less palatable and high-cost.

Late evening snack with complex carbohydrate of at least 50 g is recommended[13]. One year nighttime (9 PM to 7 AM) supplement nutrition containing 710 kcal increased total body protein[74]. BCAA-enriched late evening snacks improved serum albumin, nitrogen balance, respiratory quotient, protein stores, decreased proteolysis, and impaired quality of life[74-76]. Snack should be put at bedside[77]. If oral normal food is not adequate, supplement enteral nutrition or tube feeding should be considered. Percutaneous endoscopic gastrostomy (PEG) is not recommended because high risk of complication. Prolong fasting should be avoided. Small meals 4 to 6 times/day are suggested in cirrhosis with ascites. Salt restriction of less than or equal to 2 g/day should be advised in cirrhosis with ascites or edema[20]. Salt restriction may cause less palatable and hard to be sustained. Diuretic may be used.

Vitamin A replacement of 10,000 to 200,000 IU every 4 weeks in patients with vitamin A deficiency is advised. Parenteral thiamine should be administered in alcoholism. In vitamin D deficiency, calcium of 1,200 to 1,500 mg and vitamin D of 400 to 800 IU are recommended in cirrhosis with vitamin D deficiency or osteoporosis. Folic and thiamine supplement should be given in chronic alcoholism. Some experts supplement elemental zinc routinely[20].

Conclusion

Malnutrition is common in patients with cirrhosis and associated with worse prognosis. Nutritional assessment and treatment is important. Body mass index and body composition analysis have limitations to be used in cirrhosis with fluid retention. CT scan to assess sarcopenia is promising. Nutritional screening should be performed in all cirrhotic patients. High risk patients should undergo complete nutritional assessment. Adequate energy and protein supplement is crucial.

What is already known on this topic?

Malnourished cirrhotic patients had higher mortality and morbidity.

Nutritional assessment tools are inconclusive.

What this study adds?

Most of nutritional assessment tools have limitations. CT scan to assess sarcopenia is promising. Further study is required.

Nutritional screening should be performed in all cirrhotic patients. RFH-NPT and LDUST may be used. Complete nutritional assessment should be done in high risk patients. Repeat assessment should be performed.

Energy and protein supplement is important. Branched chain amino acid may be use. Multiple small meals with late evening snack are advised.

Potential conflicts of interest

The authors declare no conflicts of interest.

References

1. Silva M, Gomes S, Peixoto A, Torres-Ramalho P, Cardoso H, Azevedo R, et al. Nutrition in Chronic Liver Disease. GE Port J Gastroenterol 2015;22:268-76.
2. Perez-Reyes E, Rivera-Sanchez J, Servin-Caamano AI, Perez-Torres E, Abdo-Francis JM, Higuera-de la Tijera F. Malnutrition is related to a higher frequency of serious complications in patients with cirrhosis. Rev Medica Hosp Gen Mexico 2016;79:11-6.
3. Raju V, Ganesh P. Assessment of Nutritional Status in Cirrhosis and its Impact on Complications. J Clin Exp Hepatol 2015;5:S47-8.
4. Bemeur C, Butterworth RF. Nutrition in the management of cirrhosis and its neurological complications. J Clin Exp Hepatol 2014;4:141-50.
5. Sam J, Nguyen GC. Protein-calorie malnutrition as a prognostic indicator of mortality among patients hospitalized with cirrhosis and portal hypertension. Liver Int 2009;29:1396-402.
6. Plauth M, Cabre E, Riggio O, Assis-Camilo M, Pirlich M, Kondrup J, et al. ESPEN Guidelines on Enteral Nutrition: Liver disease. Clin Nutr 2006;25:285-94.
7. Merli M, Giusto M, Gentili F, Novelli G, Ferretti G, Riggio O, et al. Nutritional status: its influence on the outcome of patients undergoing liver transplantation. Liver Int 2010 Feb;30(2):208-14.
8. Rojas-Loureiro G, Servin-Caamano A, Perez-Reyes E, Servin-Abad L, Higuera-de la Tijera F. Malnutrition negatively impacts the quality of life of patients with cirrhosis: An observational study. World J Hepatol 2017;9:263.
9. Gunsar F, Raimondo ML, Jones S, Terreni N, Wong C, Patch D, et al. Nutritional status and prognosis in cirrhotic patients. Aliment Pharmacol Ther 2006;24:563-72.
10. Montano-Loza AJ, Meza-Junco J, Prado CM, Lieffers JR, Baracos VE, Bain VG, et al. Muscle wasting is associated with mortality in patients with cirrhosis. Clin Gastroenterol Hepatol 2012;10:166-73, 173.e1.
11. Durand F, Buyse S, Francoz C, Laouenan C, Bruno O, Belghiti J, et al. Prognostic value of muscle atrophy in cirrhosis using psoas muscle thickness on computed tomography. J Hepatol 2014;60:1151-7.
12. Amodio P, Bemeur C, Butterworth R, Cordoba J, Kato A, Montagnese S, et al. The nutritional management of hepatic encephalopathy in patients with cirrhosis: International Society for Hepatic Encephalopathy and Nitrogen Metabolism Consensus. Hepatol Baltim Md 2013;58:325-36.
13. Bunchorntavakul C, Supanun R, Atsawarungruangkit A. Nutritional Status and its Impact on Clinical Outcomes for Patients Admitted to Hospital with Cirrhosis. J Med Assoc Thai 2016;99 Suppl 2:S47-55.
14. Maharshi S, Sharma BC, Srivastava S. Malnutrition in cirrhosis increases morbidity and mortality. J Gastroenterol Hepatol 2015;30:1507-13.
15. Roongpisuthipong C, Sobhonslidsuk A, Nantiruj K, Songchitsomboon S. Nutritional assessment in various stages of liver cirrhosis. Nutrition 2001;17:761-5.
16. Sobhonslidsuk A, Roongpisuthipong C, Nantiruj K, Kulapongse S, Songchitsomboon S, Sumalnop K, et al. Impact of liver cirrhosis on nutritional and immunological status. J Med Assoc Thai 2001;84:982-8.
17. Tandon P, Raman M, Mourtzakis M, Merli M. A practical approach to nutritional screening and assessment in cirrhosis. Hepatology 2017;65:1044-57.
18. Cheung K, Lee SS, Raman M. Prevalence and Mechanisms of Malnutrition in Patients With Advanced Liver Disease, and Nutrition Management Strategies. Clin Gastroenterol Hepatol 2012;10:117-25.
19. Moctezuma-Velazquez C, Garcia-Juarez I, Soto-Solis R, Hernandez-Cortes J, Torre A. Nutritional assessment and treatment of patients with liver cirrhosis. Nutrition 2013;29:1279-85.
20. Lin SY, Wang YY, Sheu WH. Increased serum leptin concentrations correlate with soluble tumour necrosis factor receptor levels in patients with cirrhosis. Clin Endocrinol (Oxf) 2002;57:805-11.
21. Kalaitzakis E, Bosaeus I, Ohman L, Bjornsson E. Altered postprandial glucose, insulin, leptin, and ghrelin in liver cirrhosis: correlations with energy intake and resting energy expenditure. Am J Clin Nutr 2007;85:808-15.
22. Plauth M, Schutz ET. Cachexia in liver cirrhosis.

Cachexia 2002;85:83-7.
23. Aqel BA, Scolapio JS, Dickson RC, Burton DD, Bouras EP. Contribution of Ascites to Impaired Gastric Function and Nutritional Intake in Patients With Cirrhosis and Ascites. Clin Gastroenterol Hepatol 2005;3:1095-100.
24. Madden AM, Bradbury W, Morgan MY. Taste perception in cirrhosis: Its relationship to circulating micronutrients and food preferences. Hepatology 1997;26:40-8.
25. Muller MJ, Bottcher J, Selberg O, Weselmann S, Boker KH, Schwarze M, et al. Hypermetabolism in clinically stable patients with liver cirrhosis. Am J Clin Nutr 1999;69:1194-201.
26. Sevastianos VA, Dourakis SP. Malnutrition and Sarcopenia in Advanced Liver Disease. J Nutr Food Sci 2016;6:1-8.
27. Italian Multicentre Cooperative Project on nutrition in liver cirrhosis. Nutritional status in cirrhosis. J Hepatol 1994;21:317-25.
28. Owen OE, Trapp VE, Reichard GA, Mozzoli MA, Moctezuma J, Paul P, et al. Nature and quantity of fuels consumed in patients with alcoholic cirrhosis. J Clin Invest 1983;72:1821-32.
29. Tandon P, Low G, Mourtzakis M, Zenith L, Myers RP, Abraldes JG, et al. A Model to Identify Sarcopenia in Patients With Cirrhosis. Clin Gastroenterol Hepatol 2016;14:1473-1480.e3.
30. Campillo B, Richardet JP, Bories PN. Validation of body mass index for the diagnosis of malnutrition in patients with liver cirrhosis. Gastroenterol Clin Biol 2006;1224: 1133.
31. Vulcano DS, Carvalhaes MA, Bakonyi Neto A. Evaluation of nutritional indicators and body composition in patients with advanced liver disease enrolled for liver transplantation. Acta Cir Bras 2013;28:733-9.
32. Nunes G, Santos CA, Barosa R, Fonseca C, Barata AT, Fonseca J. Outcome and nutritional assessment of chronic liver disease patients using anthropometry and subjective global assessment. Arq Gastroenterol 2017;54:225-31.
33. Fiore P, Merli M, Andreoli A, De Lorenzo A, Masini A, Ciuffa L, et al. A comparison of skinfold anthropometry and dual-energy X-ray absorptiometry for the evaluation of body fat in cirrhotic patients. Clin Nutr 1999;18:349-51.
34. Montano-Loza AJ. Clinical relevance of sarcopenia in patients with cirrhosis. World J Gastroenterol 2014;20:8061-71.
35. Piquet M-A, Ollivier I, Gloro R, Castel H, Tiengou L-E, Dao T. Nutritional indices in cirrhotic patients. Nutrition 2006;22:216-7.
36. Alberino F, Gatta A, Amodio P, Merkel C, Di Pascoli L, Boffo G, et al. Nutrition and survival in patients with liver cirrhosis. Nutrition 2001;17:445-50.
37. Teiusanu A, Andrei M, Arbanas T, Nicolaie T, Diculescu M. Nutritional Status in Cirrhotic Patients. Maedica 2012 Dec;7:284-9.
38. Harrison J, McKiernan J, Neuberger JM. A prospective study on the effect of recipient nutritional status on outcome in liver transplantation. Transpl Int 1997;10:369-74.
39. Figueiredo FA, De Mello Perez R, Kondo M. Effect of liver cirrhosis on body composition: evidence of significant depletion even in mild disease. J Gastroenterol Hepatol 2005;20:209-16.
40. Figueiredo FA, Dickson ER, Pasha TM, Porayko MK, Therneau TM, Malinchoc M, et al. Utility of standard nutritional parameters in detecting body cell mass depletion in patients with end-stage liver disease. Liver Transpl 2000;6:575-81.
41. Kawaguchi T, Taniguchi E, Itou M, Ibi R, Okada T, Mutou M, et al. Body cell mass is a useful parameter for assessing malnutrition and severity of disease in non-ascitic cirrhotic patients with hepatocellular carcinoma or esophageal varices. Int J Mol Med 2008; 22:589-94.
42. Selberg O, Bottcher J, Tusch G, Pichlmayr R, Henkel E, Muller MJ. Identification of high- and low-risk patients before liver transplantation: a prospective cohort study of nutritional and metabolic parameters in 150 patients. Hepatol Baltim Md 1997;25(3):652-7.
43. Pirlich M, Schutz T, Spachos T, Ertl S, Weiss ML, Lochs H, et al. Bioelectrical impedance analysis is a useful bedside technique to assess malnutrition in cirrhotic patients with and without ascites. Hepatol Baltim Md 2000;32:1208-15.
44. Madden AM, Morgan MY. A comparison of skinfold anthropometry and bioelectrical impedance analysis for measuring percentage body fat in patients with cirrhosis. J Hepatol 1994;21:878-83.
45. Cichoz-Lach H, Michalak A. A Comprehensive Review of Bioelectrical Impedance Analysis and Other Methods in the Assessment of Nutritional Status in Patients with Liver Cirrhosis. Gastroenterol Res Pract 2017;2017: 6765856.
46. Fernandes SA, de Mattos AA, Tovo CV, Marroni CA. Nutritional evaluation in cirrhosis: Emphasis on the phase angle. World J Hepatol 2016;8:1205-11.
47. Ruiz-Margain A, Macias-Rodriguez RU, Duarte-Rojo A, Rios-Torres SL, Espinosa-Cuevas A, Torre A. Malnutrition assessed through phase angle and its relation to prognosis in patients with compensated liver cirrhosis: a prospective cohort study. Dig Liver Dis 2015;47: 309-14.
48. Figueiredo FA, Perez RM, Freitas MM, Kondo M. Comparison of three methods of nutritional assessment in liver cirrhosis: subjective global assessment, traditional nutritional parameters, and body composition analysis. J Gastroenterol 2006;41:476-82.
49. Falck-Ytter Y, McCullough AJ. The effect of alcohol on body composition. Am J Gastroenterol 2000;95:2156-9.

50. Cruz-Jentoft AJ, Baeyens JP, Bauer JM, Boirie Y, Cederholm T, Landi F, et al. Sarcopenia: European consensus on definition and diagnosis: Report of the European Working Group on Sarcopenia in Older People. Age Ageing 2010;39:412-23.
51. Holt EW, Frederick RT, Verhille MS. Prognostic value of muscle wasting in cirrhotic patients. Clin Gastroenterol Hepatol 2012;10:1056; author reply 1056-1057.
52. Montano-Loza AJ, Meza-Junco J, Prado CMM, Tandon P, Bain VG, Ma M, et al. New cutoff values for sarcopenia for predictin 6-month mortality in cirrhotic patients. J Hepatol 2013;58:S95.
53. Carey EJ, Lai JC, Wang CW, Dasarathy S, Lobach I, Montano-Loza AJ, et al. A multicenter study to define sarcopenia in patients with end-stage liver disease. Liver Transpl 2017;23:625-33.
54. Montano-Loza AJ, Duarte-Rojo A, Meza-Junco J, Baracos VE, Sawyer MB, Pang JXQ, et al. Inclusion of Sarcopenia Within MELD (MELD-Sarcopenia) and the Prediction of Mortality in Patients With Cirrhosis. Clin Transl Gastroenterol 2015;6:e102.
55. Englesbe MJ, Patel SP, He K, Lynch RJ, Schaubel DE, Harbaugh C, et al. Sarcopenia and mortality after liver transplantation. J Am Coll Surg 2010;211:271-8.
56. Alvares-da-Silva MR, Reverbel da Silveira T. Comparison between handgrip strength, subjective global assessment, and prognostic nutritional index in assessing malnutrition and predicting clinical outcome in cirrhotic outpatients. Nutrition 2005;21:113-7.
57. Alameri HF, Sanai FM, Al Dukhayil M, Azzam NA, Al-Swat KA, Hersi AS, et al. Six Minute Walk Test to assess functional capacity in chronic liver disease patients. World J Gastroenterol 2007;13:3996-4001.
58. Putadechakum S, Klangjareonchai T, Soponsaritsuk A, Roongpisuthipong C. Nutritional Status Assessment in Cirrhotic Patients after Protein Supplementation [Internet]. International Scholarly Research Notices. 2012 [cited 2019 Jan 1]. Available from: https://www.hindawi.com/journals/isrn/2012/690402/cta/
59. Stephenson GR, Moretti EW, El-Moalem H, Clavien PA, Tuttle-Newhall JE. Malnutrition in liver transplant patients: preoperative subjective global assessment is predictive of outcome after liver transplantation. Transplantation 2001;72:666-70.
60. Morgan MY, Madden AM, Soulsby CT, Morris RW. Derivation and validation of a new global method for assessing nutritional status in patients with cirrhosis. Hepatol Baltim Md 2006;44:823-35.
61. Sasidharan M, Nistala S, Narendhran RT, Murugesh M, Bhatia SJ, Rathi PM. Nutritional status and prognosis in cirrhotic patients. Trop Gastroenterol Off J Dig Dis Found 2012;33:257-64.
62. Merli M, Berzigotti A, Zelber-Sagi S, Dasarathy S, Montagnese S, Genton L, et al. EASL Clinical Practice Guidelines on nutrition in chronic liver disease. J Hepatol 2019;70:172-93.
63. Arora S, Mattina C, McAnenny C, O'Sullivan N, McGeeney L, Calder N, et al. The development and validation of a nutritional prioritising tool for use in patients with chronic liver disease. J Hepatol 2012;56:S241.
64. Borhofen SM, Gerner C, Lehmann J, Fimmers R, Gortzen J, Hey B, et al. The Royal Free Hospital-Nutritional Prioritizing Tool Is an Independent Predictor of Deterioration of Liver Function and Survival in Cirrhosis. Dig Dis Sci 2016;61:1735-43.
65. McFarlane M, Hammond C, Roper T, Mukarati J, Ford R, Burrell J, et al. Comparing assessment tools for detecting undernutrition in patients with liver cirrhosis. Clin Nutr ESPEN 2018;23:156-61.
66. Cordoba J, Lopez-Hellin J, Planas M, Sabin P, Sanpedro F, Castro F, et al. Normal protein diet for episodic hepatic encephalopathy: results of a randomized study. J Hepatol 2004;41:38-43.
67. Koretz RL, Avenell A, Lipman TO. Nutritional support for liver disease. Cochrane Database Syst Rev 2012 May 16;(5):CD008344.
68. Rossi Fanelli F, Cangiano C, Capocaccia L, Cascino A, Ceci F, Muscaritoli M, et al. Use of branched chain amino acids for treating hepatic encephalopathy: clinical experiences. Gut. 1986;27 Suppl 1:111-5.
69. Marchesini G, Bianchi G, Merli M, Amodio P, Panella C, Loguercio C, et al. Nutritional supplementation with branched-chain amino acids in advanced cirrhosis: a double-blind, randomized trial. Gastroenterology 2003;124:1792-801.
70. Muto Y, Sato S, Watanabe A, Moriwaki H, Suzuki K, Kato A, et al. Effects of oral branched-chain amino acid granules on event-free survival in patients with liver cirrhosis. Clin Gastroenterol Hepatol 2005;3:705-13.
71. Wahren J, Denis J, Desurmont P, Eriksson LS, Escoffier JM, Gauthier AP, et al. Is intravenous administration of branched chain amino acids effective in the treatment of hepatic encephalopathy? A multicenter study. Hepatol Baltim Md 1983;3:475-80.
72. Plank LD, Gane EJ, Peng S, Muthu C, Mathur S, Gillanders L, et al. Nocturnal nutritional supplementation improves total body protein status of patients with liver cirrhosis: a randomized 12-month trial. Hepatol Baltim Md 2008 Aug;48:557-66.
73. Nakaya Y, Okita K, Suzuki K, Moriwaki H, Kato A, Miwa Y, et al. BCAA-enriched snack improves nutritional state of cirrhosis. Nutrition 2007;23:113-20.
74. Tsien CD, McCullough AJ, Dasarathy S. Late evening snack: exploiting a period of anabolic opportunity in cirrhosis. J Gastroenterol Hepatol 2012;27:430-41.
75. Lai JC, Tandon P. How I Approach It: Improving Nutritional Status in Patients With Cirrhosis. Am J Gastroenterol 2018;113:1574-6.