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Original Article

Nutrition evaluation and metabolic profiling analysis of liver cirrhosis patients: a cross-sectional study

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Abstract: Malnutrition is the most common complication found in cirrhosis patients. It has been associated with development of severe complications and poor prognosis. However, the nutrition status of cirrhosis patients is often underestimated. Analyzing nutrition risk, malnutrition, body composition, and metabolic profiling of liver cirrhosis patients, the current study examined the mutual correlation of these indexes. The current study enrolled 120 liver cirrhosis patients and 20 concurrent healthy controls. Nutrition risk screening was conducted with NRS-2002. Nutrition status was evaluated by subjective global assessment (SGA). Body mass index (BMI) and body composition levels were measured by multi-frequency bioelectrical impedance analysis. Metabolic profiling was detected by tandem mass spectrometry. This study further explored the mutual correlation between these indexes. There were no significant differences in prevalence of nutritional risk and malnutrition between patients with different causes of liver cirrhosis (X^2=0.845, P=0.358; X^2=1.386, P=0.239; X^2=0.702, P=0.402). There were significant differences in malnutrition, nutrition risk, and body composition of different CTP score liver cirrhosis patients (X^2=15.609, P<0.001; X^2=27.799, P=0.037; X^2=6.599, P<0.001; T=34.179, P<0.001). Homocysteine and phenylalanine in the nutrition risk group was significant higher than that in the non-nutrition risk group (T=2.106, P=0.037; T=3.019, P=0.004), while alanine and citrulline/arginine ratios were significantly lower than those in the non-nutrition risk group (*T*=-4.508, *P*<0.001; *T=-3.552, P*=0.001). Nutrition risk and malnutrition commonly exist in liver cirrhosis patients. It presents with body composition alterations. Amino acids and other substances lead to metabolic disorders, further leading poor prognosis. These may be improved and prevented through active nutrition support treatment.

Keywords: Liver cirrhosis, nutrition risk, malnutrition, body composition analysis, metabolic profiling

Introduction

The liver is a core metabolic organ, playing a pivotal role in sustaining nutrition states and homeostasis. With the formation of pseudolobules, anatomic configuration and biochemical function disorders may occur. If they cause liver cirrhosis, further nutrient metabolism disorders and protein-energy malnutrition (PEM) follow [1]. Several studies have shown that malnutrition is the most common complication of cirrhosis patients. It has been associated with condition aggravation, occurrence of other associated complications, and short-term mortality [2-4]. Although the importance of nutrition status has been recognized in patients with liver cirrhosis, there remains no gold standard for nutritional assessment [5, 6].

For liver cirrhosis patients complicated with malnutrition, timely nutrition support treatment can effectively improve nutrition status, reduce treatment-associated complications, increase physical capacity, improve living quality, and reduce repeated admission rates and mortality risks [7, 8]. Hence, it is very important to accurately evaluate nutrition risk and malnutrition in liver cirrhosis patients. In recent years, malnutrition statuses in liver cirrhosis patients have been paid increasing attention. The European Society for Clinical Nutrition and Metabolism (ESPEN) announced a definition of nutrition risk and introduced a screening tool, NRS-2002 (Nutrition risk screening-2002) in 2002. They also published guidelines of enteral and parenteral nutrition support in 2009, recommending that NRS-2002 be used to perform nutrition risk screening. Patients complicated with nutrition risks should be given nutrition support/treatment [9, 10]. Hanai et al. [11] and Anand et al. [12] have indicated that decreased lean mass, especially decreased skeletal muscle,

Table 1. Baseline characteristics of the study participants

Measurements	Cirrhosis patients	Healthy controls
Age (y)	64.2±13.3	52.6±12.5
Male/Female n (%)	78/79 (65.0/35.0)	12/8 (40.0/60.0)
Etiology induced cirrhosis (%)		
HBV	52 (36.6)	
HCV	8 (5.6)	
ALD	36 (25.4)	
NASH	8 (5.6)	
PBC	16 (11.3)	
Score Child-Turcotte-Pugh	7.7±2.8	
Laboratory tests		
Prothrombin time (second)	17.5±9.0	12.5±0.9
Lymphocyte count (10^9/L)	1.4±0.7	2.0±0.6
Alanine aminotransferase (IU/L)	40.4±26.1	21.9±10.7
Aspartate aminotransferase (IU/L)	55.3±41.4	19.9±5.6
Total bilirubin (umol/L)	43.4±46.2	16.5±7.7
Total protein (g/L)	69.6±8.2	67.7±8.5
Albumin (g/L)	34.8±6.6	42.3±4.3
Cholinesterase (U/L)	4626.2±2712.8	8185.8±1265.5
Triglyceride (mmol/L)	1.2±0.8	1.3±0.6
Total cholesterol (mmol/L)	4.0±1.1	5.0±1.3

could be a predictive factor of mortality risk. However, skeletal muscle management, including the addition of branch chain amino acids, could improve patient survival rates. Currently, there are no rapid, accurate, and unified nutrition screening and evaluation tools.

Metabolomics analysis, a rapidly expanding tool used to study small molecules, has been applied extensively for liver disease diagnosis, helping to better understand the pathophysiology [13, 14]. However, there remains a lack of relevant research on the relationship between nutrition statuses of liver cirrhosis patients and metabolomics variation. The aim of the current study, therefore, was to analyze nutrition risks, malnutrition, body composition, and metabolic profiling of liver cirrhosis patients, examining correlation levels between these indexes.

Materials and methods

Subjects

The current study enrolled 120 liver cirrhosis patients and 29 concurrent healthy controls, between October 2015 and September 2017. Inclusion criteria: 1) Patients 18 ≤ age ≤ 65 years old; 2) Sane and able to cooperate; 3) Diagnosis based on [15-19]; and 4) Checkup results of healthy controls within the normal limits (**Table 1**). Exclusion criteria: 1) Patients combined with heart, lung, kidney, cerebral, or hematological system diseases, as well as malignant tumors and abnormal thyroid function; 2) Pregnant or breastfeeding women; 3) Recent surgical operations; and 4) Patients that refused to cooperate with this research. This research was approved by the hospital Ethics Committee and all subjects provided written informed consent. Regarding the metabolomics study, the cirrhosis participants were subdivided into two groups, including NRS-2002 scores below 3 (<3, n=67) and scores 3 and over (≥3, n=53).

Metabolism profiling analysis

Consecutive fixed-point sampling was used to collect fasting venous blood on the second admission day. Routine blood examinations and biochemical testing were carried out. Capillary blood was collected and sent to the Inspection Center. Agilent 1200 high efficiency liquid chromatography (Agilent Technologies Co. Ltd, Palo Alto, America) was used to perform metabolism profiling analysis.

Nutrition risk screening and evaluation

On the first admission day, NRS-2002 was used to screen nutrition risks regarding age, nutrition levels, and disease severity. Scores ≥3 indicate a nutrition risk. SGA was used to evaluate malnutrition degrees. Concerning the 8 items, ≥5 items with SGA Grading C indicated severe malnutrition. Moreover, ≥5 items with SGA Grade B indicated mild to moderate malnutrition. On the second day after admission, heights, weights, and BMIs during fasting and after emptying the bladder and bowels were measured.

Table 2. Nutrition statuses of different etiology-induced liver cirrhosis patients (cases, %)

Nutrition status	Viral hepatitis induced liver cirrhosis group (*n*=60)	Non-viral hepatitis induced liver cirrhosis group (*n*=60)	Total	X^2	*P*
Nutrition risk (NRS-2002)	24 (40.0)	29 (48.3)	53 (44.2)	0.845	0.358
Malnutrition (SGA)	16 (26.7)	22 (36.7)	38 (31.7)	1.386	0.239
Malnutrition (BMI)	2 (3.3)	4 (6.7)	6 (5)	0.702	0.402

Body composition analysis

Inbody 770 type multi-frequency bioelectrical impedance analysis tomography (InBody Co. Ltd, Seoul, Korea) was used to detect phase angle (PA), body cell mass (BCM), total body water (TBW), extracellular water (EC), edema index (ECW/TBW), fat mass (FM), and skeletal muscle mass (SMM).

Examination methods

In a fasting state, the research subjects were required to empty their bladders and stools and remove metal and electronic materials. They stood on the tomography machine, with both arms bilaterally opened at 15 degrees, avoiding touching their trunks. After measuring body weights, the basic information was entered. Measurements were performed after keeping still for 2 minutes.

Statistical analysis

Data was analyzed using SPSS 20.0 software. Measurement variables are expressed as mean ± substandard deviation. They were compared using *t*-tests and analysis of variance (ANOVA). Chi-square testing was used to compare groups with categorical variables (rates). LSD was used for inter-group multiple comparisons. Pearson's correlation analysis was used to assess correlation levels in NRS-2002, body composition, and Child-Turcotte-Pugh (CTP) scores. With each metabolic profiling index as an independent variable and nutrition risk as a dependent variable, the logistics model was used to analyze correlation levels. *P*-values less than 0.05 indicate statistical significance.

Results

Baseline characteristics

This study included liver cirrhosis patients, aged 41 to 88 years old, with a mean age of (64.2±13.3) years old. Regarding gender composition, males accounted for 65.0% (78 cases), while females accounted for 35.0% (42 cases). There were 52 cases of hepatitis B cirrhosis, 8 cases of hepatitis C-induced liver cirrhosis, 36 cases of alcoholic liver cirrhosis, 16 cases of primary biliary cirrhosis, and 8 cases of nonalcoholic fatty liver disease-induced liver cirrhosis. The 20 healthy controls were aged between 47-81 years old, with a mean age of (61.1±12.6) years old. Males accounted for 60.0% (12 cases), while females accounted for 40.0% (8 cases). Age and gender composition showed no significant differences between liver cirrhosis patients and the healthy controls.

Nutrition risk and malnutrition in different etiologies-induced liver cirrhosis

The 120 cases of liver cirrhosis patients were divided into the viral hepatitis-induced liver cirrhosis group and non-viral hepatitis induced liver cirrhosis group. After NSR-2002 screening, the incidence rate of nutrition risk was 40% in the viral hepatitis-induced liver cirrhosis group and 48.3% in the non-viral hepatitis induced liver cirrhosis group. There were no significant differences between the two groups (*P*=0.358). After SGA evaluation, 26.7% of viral hepatitis induced liver cirrhosis patients were complicated with malnutrition, while 36.7% of non-viral hepatitis induced liver cirrhosis patients were complicated with malnutrition. There were no significant differences between the two groups (*P*=0.239). After BMI evaluation, 3.3% of viral hepatitis induced liver cirrhosis patients were complicated with malnutrition, while 6.7% of non-viral hepatitis induced liver cirrhosis patients were complicated with malnutrition. There were no significant differences between the two groups (*P*=0.402) (**Table 2**).

Nutrition risk and malnutrition in different child-turcotte-pugh score classified liver cirrhosis patients

According to Child-Turcotte-Pugh scores, the liver cirrhosis patients were classified into A, B,

Table 3. Nutrition statuses of different CTP scores in liver cirrhosis patients (cases, %)

Evaluation method	A group (*n*=58)	B group (*n*=28)	C group (*n*=34)	Total (*n*=120)	X^2	*P*
Nutrition risk (NRS-2002)	15 (25.9)	16 (57.1)	22 (64.7)	53 (44.2)	15.609	<0.001
Malnutrition (SGA)	5 (9.6)	14 (50.0)	19 (55.9)	38 (31.7)	27.799	<0.001
Malnutrition (BMI)	0 (0)	2 (7.1)	4 (11.8)	6 (5)	6.599	0.037

Table 4. Multiple frequency biochemical impedance of body composition analysis ($\bar{x} \pm s$)

Groups	Healthy control group (n=20)	Liver cirrhosis group (n=120)			F	*P*
		A group (n=58)	B group (n=28)	C group (n=34)		
PA (°)	5.6±0.8	5.0±0.2	4.8±0.2	4.5±0.4	34.179	<0.001
BCM (kg)	33.2±6.4	30.0±4.7	29.2±4.6	26.5±4.6	8.246	<0. 001
FM (kg)	18.0±6.6	12.6±2.4	10.9±1.8	10.3±2.8	25.449	<0.001
SCM (kg)	29.1±5.7	27.3±7.2	24.7±4.2	22.7±3.7	7.095	<0.001
TBW (L)	36.8±6.9	36.5±5.5	37.2±6.5	38.9±5.0	1.216	0.306
ECW/TBW	0.374±0.009	0.396±0.005	0.402±0.003	0.412±0.005	239.550	<0.001

and C groups. After NSR-2002 screening, nutrition risks of the A group, B group, and C group were, respectively, 25.9%, 50.0%, and 64.7%, with no significant differences between the three groups (*P*<0.001). Nutrition risks of group B and group C were higher than that of group A, with significant differences in comparison (*P*= 0.004, P<0.001). After SGA evaluation, complicated malnutrition rates of the three groups were, respectively, 9.6%, 50.0%, and 55.9%. There were significant differences between the three groups. Complicated malnutrition rates of group B and group C were higher than that of group A, with significant differences in comparison (*P*<0.001). The complicated malnutrition rate of group C was higher than that of group B, with no significant differences between the two groups (*P*=0.578). After BMI evaluation, malnutrition percentages of the three groups were, respectively, 0%, 7.1%, and 11.8%. There were no significant differences between the three groups (*P*=0.037). The malnutrition percentage of group C was higher than that of group B, with no significant differences (*P*=0.012). The malnutrition percentage of group B was compared with that of group A and group C, showing significant differences (*P*=0.151, *P*=0.400) (**Table 3**).

Body composition analysis results (multi-frequency bioelectrical impedance analysis)

There were significant differences, according to inter-groups comparisons of PA, BCM, FM, SMM, and ECW/TBW, between the liver cirrhosis patient group and healthy control group (*P*<0.001). There were no significant differences in TBW between the liver cirrhosis patient group and healthy control group (*P*=0.337) (**Table 4**). Indexes with significant differences underwent inter-group comparisons. The PA of group A, B, and C was, respectively, compared with that of the healthy control group, showing significant differences (P<0.001). There were also significant differences in PA between group A and the group B, between group A and group C, and between group B and group C (respectively, *P*=0.020, *P*<0.001, *P*=0.005). BCM of group A, B, and C was, respectively, compared with that of the healthy control group. There were significant differences (*P*=0.015, *P*=0.006, *P*<0.001). There were significant differences in BCM between group A and group C and between group B and group C (*P*=0.001, *P*=0.033), but there were no significant differences in BCM between group A and group B (*P*=0.487). Furthermore, FM of group A, B, and C was, respectively, compared with that of the healthy control group. There were significant differences (*P*<0.001). There were significant differences in FM between group A and group B and between group A and group C (*P*=0.029, *P*=0.002), but there were no significant differences in FM between group B and group C (*P*=0.487). There were significant differences in SMM between the healthy control group and group B and between the healthy control group and group C (*P*=0.011, *P*<0.001). There were significant differences in SMM between group A and group B and between

Table 5. Correlation of CTP scores with NRS-2002, SGA, PA, and ECW/TBW

Observation indexes	Pearson's coefficient	*P*
NRS-2002	0.713	<0.001
SGA	0.510	<0.001
PA	-0.647	<0.001
ECW/TBW	0.888	<0.001

group B and group C (P=0.049, P<0.001). There were no significant differences in SMM between the healthy control group and group A and between group B and group C (P=0.255, P=0.176). There were significant differences in ECW/TBW between the healthy control group and groups A, B, and C (P<0.001). There were significant differences in ECW/TBW, the edema index, between group B and group A, between group C and group A, and between group B and group C (P<0.001).

Correlation of NRS-2002, SGA, PA, and ECW/TBW with CTP scores

Pearson's correlation analysis was used to evaluate correlation levels of NR-S2002, SGA, PA, and ECW/TBW with CTP scores. The correlation coefficient of ECW/TB and Child-Pugh score was 0.888 (P<0.001), showing a high correlation between ECW/TB and Child-Pugh scores. The correlation coefficient of NRS2002, SGA, and PA with Child-Pugh scores was, respectively, 0.713, 0.510, and -0.647 (P< 0.001), which showed a moderate correlation between the three indexes and CTP scores (**Table 5**).

Metabolic profiling and nutrition risks

Based on MS-MS metabolic analysis, 29 kinds of amino acids and its metabolites, along with the 9 correlation co-efficient metabolic profiling results, were obtained. According to NRS-2002 scores, the research subjects were divided into the nutrition risk group and non-nutrition risk group. Two independent samples *t*-tests showed significant differences in 8 kinds of amino acids and its metabolites and 6 correlation coefficients (P<0.05), which were respectively Ala (alanine), Arg (arginine), Hcy (homocysteine), His (histidine), Orn (ornithine), Phe (phenylalanine), Pip (piperine), Val (valine), Cit (citrulline)/Arg, Gly (glycine)/Ala, Met (methionine)/Phe, Orn/Cit, Tyr (tyrosine)/Cit, and Val/Phe (**Table 6**). Logistic multiple regression analysis was performed on these indexes and the following regression equation was obtained.

ROC curves were used to test the determinant power of the regress equation. As shown in **Figure 1**, in determining whether there was nutrition risk, sensitivity was 69.8%, specificity was 88.5%, AUC was 0.851, the substandard error of AUC was 0.0332, and the 95% confidence interval was 0.78-0.906. In summary, it is reasonable to believe the accuracy and reliability of the regression equation. Upregulation of Hcy and Phe and downregulation of Ala and Cit/Arg were found in the blood of patients with nutrition risks.

Discussion

Malnutrition is one of the most common complications of liver cirrhosis, negatively impacting prognosis. Malnutrition is reversible. Thus, early detection and treatment is of critical clinical importance. The current study showed that body composition analysis and metabolic profiling, as nutritional assessment tools, offer several advantages over traditional methods in patients with liver cirrhosis. Traditional methods, such as BMI, have underestimated the prevalence of malnutrition. This illusion is more notable in the early stages of cirrhosis. In clinical practice, it is important to avoid these traditional methods when advanced alternatives are available.

In the current study, SGA and BMI were used to evaluate malnutrition in liver cirrhosis patients. Results suggest that nutrition risks and protein-energy malnutrition commonly exist in liver cirrhosis patients. Although there were significant differences in malnutrition ratios in liver cirrhosis patients with different CTP scores, the diagnostic efficiency of BMI in diagnosis of malnutrition was far lower than that of SGA. It was also far lower than incidence rates of nutrition risk, consistent with Sorrentino and Morgan et al. [20, 21]. Due to abnormal liver function, water-sodium retention and complicated ascites might hamper the reliability of BMI and other traditional anthropometry indexes applied in the liver functional decompensatory period [5, 22]. This can cause malnutrition in liver cirrhosis patients, losing the significance of guiding clinical nutrition treatment.

Table 6. Metabolic profiling alteration of nutrition risk of nutrition risk groups compared with that of the non-nutrition risk group

Metabolites (umol/l) or its ratio	Nutrition risk group	Non-nutrition risk group	t	*P*
Arg	8.01±6.37	4.89±3.42	3.304	0.002
Hcy	9.53±1.20	9.13±1.02	2.106	0.037
Orn	65.76±56.62	45.60±37.15	2.312	0.023
Phe	60.23±26.57	48.48±12.80	3.019	0.004
Pip	273.44±209.67	209.33±87.64	2.118	0.038
Ala	102.29±36.76	140.65±55.04	-4.508	<0.001
His	42.99±26.61	67.80±50.33	-3.836	<0.001
Val	108.99±29.29	127.14±36.48	-3.078	0.003
Gly/Ala	2.23±1.06	1.58±0.78	3.938	<0.001
Tyr/Cit	2.24±1.44	1.78±0.91	2.083	0.041
Orn/Cit	2.33±1.38	1.75±1.51	2.272	0.025
Cit/Arg	4.91±3.56	8.96±9.72	-3.552	0.001
Met/Phe	0.47±0.21	0.54±0.20	-2.039	0.043
Val/Phe	2.12±0.74	2.85±0.90	-5.006	<0.001

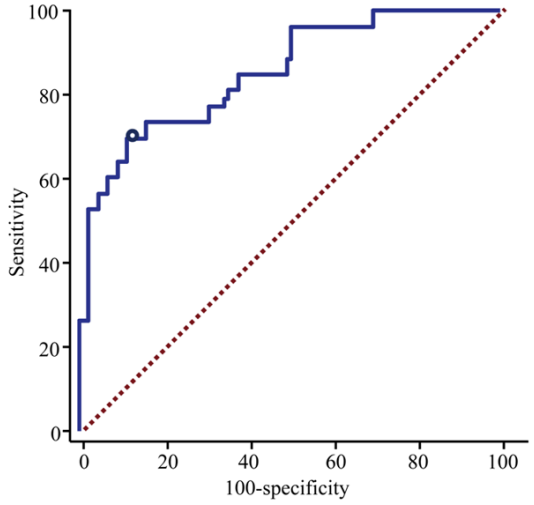

Figure 1. ROC curve of the nutritional risk and metabolic profiling logistic regressioin equation.

According to present results, NRS-2002 nutrition risk screening showed that nutrition risk ratios of liver cirrhosis patients had no significant correlation with etiology. The supposed nutrition risk showed a positive correlation with disease severity. The nutrition risk ratio reached 64.7% in CTP grading C liver cirrhosis patients. Even in decompensated cirrhosis patients, if the patients did not receive appropriate nutrition support treatment, the mortality risk would be 4 times that of patients receiving nutrition support treatment [23]. This suggests that liver cirrhosis patients should be given nutrition risk screening. Patients complicated with nutrition risks should be given active intervention, aiming to improve adverse clinical outcome of cirrhosis.

Multiple frequency bioelectrical impedance analysis has gradually gained support for clinical nutrition evaluation. It has the advantages of non-invasiveness, easy operation, short measure times, and relatively accurate measurement data. It is suited for dynamic monitoring body composition of liver cirrhosis patients. In these indexes, the phase angle is calculated by a fixed equation using primary data [24, 25]. The reactance and resistance, which are seldomly influenced by distribution of body fluids, can objectively reflect the function and integrity of the cytomembrane. Belarmino et al. [26] and Ruiz-Margain et al. [27] showed that PA is an independent factor in predicting short-term mortality and incidence of complications in liver cirrhosis patients. In the current study, PA showed significant differences in each group. Pearson's correlation analysis showed that PA had a negative correlation with CTP scores, indicating that reduction of PA correlated with poor prognosis. This is consistent with the results of Ruiz-Margain et al. [27]. Belarmino et al. [26] study results implied that a PA ≤ 4.9° cutoff was associated, independently, with mortality and identified patients with worse metabolic, nutritional, and disease progression profiles.

Body composition analysis showed that the body composition of liver cirrhosis significantly lost. ECW/TBW was elevated with the severity of disease, but no obvious alterations of TBW were shown. This indicated that the total water content of liver cirrhosis patients should be not obviously altered. Main abnormal water metabolism disorders should be abnormal water distribution with increased extracellular fluid, decreased intracellular fluid, and reduced cell amounts and liver function. Compared with the healthy control group, the earliest alteration of the liver cirrhosis patient group was reduced FM and BCM, followed by reduced SCM and BCM in the latter period. Results were in accord

with Hiraoka et al. [28] and Fernandes et al. [29], indicating that, in early stages of liver cirrhosis, energy metabolism characteristics should be weakened glucose metabolism and active lipolysis. In late stages of liver cirrhosis, metabolism characteristics should be lipid depletion. If there is a lack of effective nutrition support treatment, the body may further catabolize the protein for energy, causing sacopenia. Sacopenia can seriously influence quality of life levels for liver cirrhosis patients. Moreover, it can cause high incidence of increased complications (infections, hyperammonemia, and hepatic encephalopathy) and high mortality risks [30-32].

The current study combined nutrition risk screening and metabolism profiling analysis. Results showed downregulation of Ala, His, and Val, the three kinds of glycogenic amino acids in liver cirrhosis, indicating that active gluconeogenesis and increased proteolysis in liver cirrhosis could cause lean mass reduction. This may further cause life quality reduction and poor prognosis, in accord with Preidis el al. [33]. Elevation of the aromatic amino acids Phe and Arg, the intermediate products of ornithine cycle, indicated ornithine cycle disturbance. This suggests that liver cirrhosis patients are prone to present hyperammonemia and further induce hepatic encephalopathy and other complications [34]. Reduction of Val, the branched-chain amino acid, could be caused by liver nutrition metabolism disturbances. These could cause branched-chain amino acid catabolism increases in skeletal muscle protein. Dillon et al. proved that through mTORC and MPS signal pathways, branched-chain amino acids could promote muscle content and improve nutrition status [35]. This indicated that appropriately supplying branched-chain amino acid as a nitrogen source, for liver cirrhosis patients, could reduce lean mass depletion, avoid activity reduction, and improve life quality. As changes of metabolic mass spectrometry emerge earlier than anthropometry measurements and biochemical tests, besides the advantages of precision and high speed, metabolic profiling is a prospective potential marker for nutritional assessment.

The single center nature and relatively small sample sizes were notable limitations in the current research. According to etiology, patients were simply divided into the viral hepatitis liver cirrhosis group and non-viral hepatitis liver cirrhosis group. This couldn't reflect differences in nutrition risks, malnutrition, alteration of body composition, and metabolism profiling in different sub-etiology groups. This limited its function in directing clinical individualized nutrition support for liver cirrhosis patients. In future studies, sample sizes should be expanded, performing multiple center randomized controlled trials, confirming present conclusions.

In conclusion, nutrition risks and malnutrition commonly exist in liver cirrhosis patients, presenting with body composition alterations. Amino acids and other substances may lead to metabolic disorders, which can cause poor clinical outcomes. These include complications occurrence, life quality reduction, and mortality risk elevation. Current results suggest that clinical physicians should routinely perform nutrition risk screening in liver cirrhosis patients and qualified hospitals should apply bioelectrical impedance analysis in evaluating body composition alteration and nutrition status. They should use metabolic profiling analysis of the metabolism variation, aiming to timely initiate targeted nutrition support treatment in liver cirrhosis patients complicated with nutrition risk and malnutrition. These measures may decrease complications, improve life quality, reduce mortality risks, and improve clinical outcomes.

Disclosure of conflict of interest

None.

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