

Nutritional Status Assessment in Chronic Liver Disease Patients According to Severity and Etiology

Research Article

Anamitra Hait*

Physician and ICU in charge, KG Hospital, Chittaranjan, West Bengal, India

***Corresponding author:** Anamitra Hait, Physician and ICU in Charge, KG Hospital, Chittaranjan, West Bengal, India, E-mail Id: anamitrahait241@gmail.com

Article Information: Submission: 21/12/2024; Accepted: 10/01/2025; Published: 15/01/2025

Copyright: © 2025 Hait A. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Introduction

Chronic liver disease (CLD) is a progressive condition marked by the gradual deterioration of liver functions over a period exceeding six months. The liver's ability to synthesize clotting factors and proteins, detoxify harmful metabolic byproducts, and excrete bile is significantly impaired in CLD [1,2]. This progressive damage often arises from continuous inflammation, destruction, and regeneration of liver tissue, ultimately resulting in fibrosis and cirrhosis. Cirrhosis, the terminal stage of CLD, is characterized by diffuse hepatic fibrosis, nodular regeneration, disruption of liver architecture, vascular reorganization, and extracellular matrix deposits [3,4]. The etiological spectrum of CLD is diverse, encompassing prolonged alcohol abuse, toxins, infections, autoimmune diseases, genetic predispositions, and metabolic disorders. In the Indian context, liver diseases are increasingly recognized as significant public health concerns. Notably, India accounted for 18.3% of the two million global liver disease-related deaths in 2015. Since 1980, the mortality attributable to cirrhosis and related complications has shown an upward trajectory in India, contrasting with declining trends in other Asian countries [5-7]. This rise can be attributed to a cultural and lifestyle shift, including greater adoption of Western dietary habits, sedentary lifestyles, and diminishing societal taboos surrounding alcohol consumption. Consequently, alcohol-related liver disease and metabolic-associated fatty liver disease (MAFLD) have emerged as prominent contributors to CLD, surpassing viral causes. Cirrhosis and CLD collectively accounted for 2.1% of all deaths in India in 2016, underscoring the urgency of addressing this escalating healthcare burden. A significant and potentially reversible complication of

cirrhosis is malnutrition, which adversely affects disease progression and patient outcomes. Malnutrition in cirrhosis has been identified as an independent predictor of mortality, with malnourished patients exhibiting a significantly higher incidence of complications, including sepsis, uncontrolled ascites, hepatic encephalopathy, spontaneous bacterial peritonitis, and hepatorenal syndrome, compared to well-nourished individuals [9,10]. Studies, including those by Biyyani et al. and Alberino et al., have reinforced this association, emphasizing the critical role of nutritional status in determining survival outcomes. Sarcopenia, characterized by skeletal muscle loss, is a common manifestation of malnutrition in cirrhosis, observed in up to 60% of patients. Its prevalence correlates with the severity of liver disease, as measured by the Child-Turcotte-Pugh (CTP) score, and contributes significantly to complications such as hepatic encephalopathy [11,12].

The pathophysiology of malnutrition in CLD is multifaceted. Altered metabolic processes in cirrhosis lead to disruptions in protein, fat, and carbohydrate metabolism. For instance, decreased hepatic and muscle glycogen reserves force the body to rely on fats and proteins as alternative energy sources, often exacerbating protein-calorie malnutrition. Additionally, increased resting energy expenditure (REE) and hypermetabolism, prevalent in a subset of cirrhotic patients, further contribute to nutritional depletion. Conditions such as ascites exacerbate these metabolic alterations, increasing the risk of sarcopenia and malnutrition [13,14]. Sarcopenia in CLD is compounded by factors such as impaired amino acid metabolism, increased muscle protein breakdown, and reduced protein synthesis. The decline in the ratio of branched-chain amino acids (BCAAs) to aromatic amino acids (AAAs) not only

contributes to muscle wasting but also predisposes patients to hepatic encephalopathy. Overnight fasting, often resulting in a starvation-like state, accelerates gluconeogenesis and lipolysis, further depleting energy reserves and exacerbating protein deficiency. These metabolic derangements underline the critical role of nutritional interventions in mitigating complications and improving outcomes in CLD [15,16].

Given these complexities, nutritional management in CLD patients has gained increasing importance, particularly with the rise in hepatic transplantation as a definitive treatment for end-stage liver disease. Malnutrition significantly impacts transplantation outcomes, with pre-transplant nutritional deficits linked to increased operative complications, prolonged hospital stays, and higher postoperative mortality rates. Thus, addressing malnutrition is not only essential for enhancing survival rates but also for optimizing transplantation success [17,18].

This study aims to provide a comprehensive assessment of nutritional status in CLD patients using an array of methodologies, including anthropometric measurements, functional assessments, and biochemical evaluations. It further seeks to elucidate the relationship between malnutrition and the severity of CLD, offering valuable insights into targeted interventions. By addressing these critical aspects, this research endeavors to contribute to improved clinical management and outcomes for patients suffering from chronic liver disease.

Materials and Methods

The study was conducted in the Department of General Medicine at B.R. Singh Hospital and Centre for Medical Education and Research, Eastern Railway, Sealdah, Kolkata. It included both outpatient department (OPD) patients and indoor admissions. The study employed a single-center, cross-sectional observational design and was carried out from January 2021 to June 2022. Cohort flowchart figure 1 representing the study process:

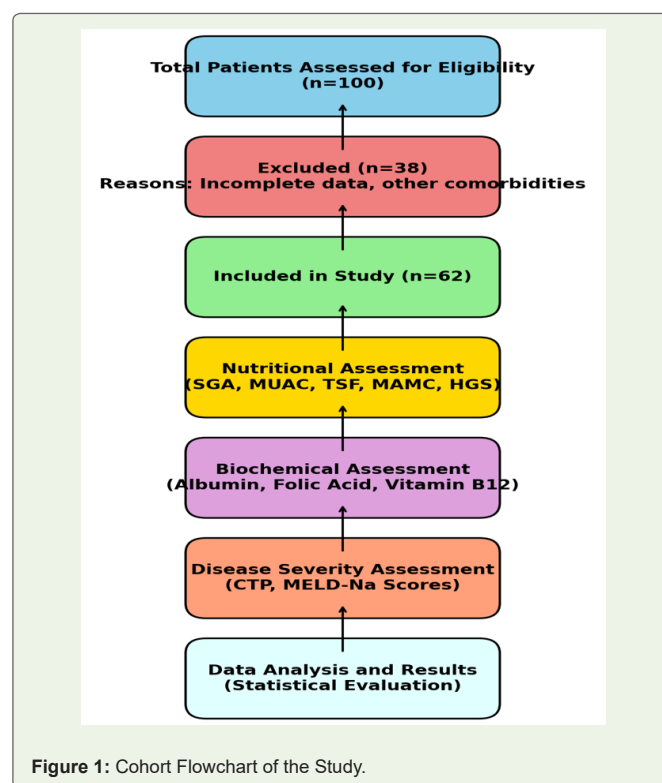
This flowchart illustrates the inclusion process of patients in the study, starting with 100 assessed patients, exclusions due to incomplete data or comorbidities, and the final cohort of 62 patients undergoing nutritional, biochemical, and disease severity assessments.

Sample Size and Justification

The sample size was calculated using the Epi Info software (version 7.2.2.2), a tool developed by the Centers for Disease Control and Prevention (CDC). Based on findings by Sherpa et al., the prevalence of malnutrition in chronic liver disease (CLD), determined by mid-arm muscle circumference (MAMC) below the 5th percentile, was estimated to be 74% ($p = 0.74$). Using a significance level of 5%, a power of 85%, and accounting for a 15% loss of data, the required sample size was determined to be 62 participants. The calculation followed the formula

$$n = \frac{4pq}{L^2}$$

where $q=1-p$ and L represents the loss percentage. Accordingly, a sample size of 62 participants was selected.



Study Population

Patients were selected randomly from the gastroenterology outpatient department and inpatients admitted to the general medicine ward. Randomization was performed using a random number table. Data were systematically recorded in a pre-designed proforma.

Inclusion Criteria

- Adult patients (both male and female) diagnosed with CLD attending the gastroenterology OPD or admitted to the general medicine department.
- Diagnosis was based on clinical symptoms (e.g., jaundice, ascites, gastrointestinal bleeding), laboratory results (e.g., liver function tests, HBsAg, Anti-HCV, autoimmune profiles), and imaging findings (e.g., coarse echotexture, nodule formation, portal vein dilatation).

Exclusion Criteria

1. Patients with other causes of malnutrition.
2. Those with hepatic encephalopathy or in a comatose state.
3. Patients on corticosteroid therapy.
4. Individuals with specific endocrinopathies, such as Grave's disease or Cushing's syndrome.
5. Patients with sepsis, suspected hepatocellular carcinoma, or other malignancies.
6. Those with chronic diarrhea or renal failure.

7. Patients suffering from chronic debilitating diseases, such as tuberculosis or diabetes mellitus.

Data Collection and Methodology

Hospital ethical committee approval was obtained prior to study initiation, and informed consent was secured from all participants. Detailed medical histories, physical examinations, and relevant investigations were conducted for all patients.

1. Anthropometric Measurements:
 - Measurements followed the guidelines of the International Society for the Advancement of Kinanthropometry (ISAK).
 - BMI: Calculated as weight (kg)/height (m²).
 - MUAC: Measured at the midpoint between the olecranon and acromion process.
 - TSF: Measured using a skinfold caliper on the posterior aspect of the arm.
- MAMC was derived using the formula: $MAMC\ (cm) = MUAC\ (cm) - [TSF\ (mm) \times 0.314]$.
2. Functional Assessment:
 - Handgrip Strength (HGS): Measured using a hydraulic dynamometer. Sarcopenia was defined as HGS <28 kg for men and <18 kg for women.
3. Nutritional Assessment:
 - Subjective Global Assessment (SGA): Patients were categorized into well-nourished (SGA-A), moderately malnourished (SGA-B), or severely malnourished (SGA-C).
 - Royal Free Hospital Nutrition Prioritizing Tool (RFH-NPT): Classified patients as malnourished (scores 2-7) or not at risk (score 1).
 - Mini-Nutritional Assessment (MNA): Evaluated changes in dietary intake, weight loss, psychological stress, and neuropsychological status to classify patients as normal, at risk, or malnourished.
4. Liver Disease Severity:
 - Severity was assessed using the Child-Turcotte-Pugh (CTP) score and MELD score, incorporating clinical and laboratory parameters.
5. Laboratory Investigations:
 - Complete blood count (CBC), bilirubin, albumin, prothrombin time (PT), INR, serum urea, and creatinine.
 - Vitamin B12 and folic acid levels were measured using standard laboratory methods.
6. Radiological and Endoscopic Evaluations:
 - Ultrasound and endoscopy were used to identify signs of portal hypertension and varices.

Statistical Analysis

Data analysis was conducted using Epi Info (version 7.2.2.2). Descriptive statistics calculated means and standard deviations. The chi-square test was used for categorical variables, while t-tests compared means between groups. Statistical significance was set at $p<0.05$. Receiver operating characteristic (ROC) curves were generated to assess the predictive power of nutritional assessment tools based on the area under the curve (AUC).

Result

(Table1) Demographic details, etiological factors, alcohol consumption, and disease severity scores for chronic liver disease patients in the study. This table1 summarizes the demographic and clinical characteristics of the study population (n=62). Age distribution shows the majority of participants were aged 51–60 years (37.1%), with a smaller representation in the younger (20–30 years, 8.1%) and older (71–80 years, 1.6%) age groups. Males predominated, accounting for 75.8% of the population. Alcohol was the most common etiology of chronic liver disease (51.6%), followed by non-alcoholic fatty liver disease (16.1%) and viral hepatitis. Notably, 58.1% of patients reported alcohol consumption. Based on the Child-Turcotte-Pugh (CPT) scoring system, 29% of patients were classified as grade A, while 35.5% each were in grades B and C, indicating significant disease severity in a substantial portion of the cohort.

Table 2 presents the clinical and nutritional assessment of the study population (n=62). The majority of patients had a MELD-Na score in the range of 10–19 (50.0%), followed by 20–29 (41.9%), with a smaller proportion scoring 30–39 (8.1%), indicating varying

Table 1: Demographic and Clinical Characteristics of CLD Patients

Characteristic	Category	Frequency (n)	Percentage (%)
Age	20-30	5	8.1
	31-40	12	19.4
	41-50	11	17.7
	51-60	23	37.1
	61-70	10	16.1
	71-80	1	1.6
Gender	Male	47	75.8
	Female	15	24.2
Etiology of CLD	Alcohol	32	51.6
	NAFLD	10	16.1
	Hepatitis B	5	8.1
	Hepatitis C	2	3.2
	Autoimmune Hepatitis (AIH)	1	1.6
	AIH + Primary Sclerosing Cholangitis (PSC)	1	1.6
	Hepatitis E + Hepatitis B	1	1.6
	Wilson's Disease	3	4.8
Alcohol Consumption	Cryptogenic	3	4.8
	Yes	36	58.1
	No	26	41.9
CPT Score	A	18	29.0
	B	22	35.5
	C	22	35.5

Table 2: Clinical and Nutritional Assessment Based on MELD-Na Score, BMI, and MUAC

Characteristic	Category	Frequency (n)	Percentage (%)
MELD-Na Score	10-19	31	50.0
	20-29	26	41.9
	30-39	5	8.1
BMI	Underweight	8	12.9
	Normal	26	41.9
	Overweight at Risk	17	27.4
	Obese I	9	14.5
	Obese II	2	3.2
MUAC (Mid-Upper Arm Circumference)	Malnourished	32	51.6
	Normal	20	32.3
	Overweight	3	4.8
	Obese	7	11.3

severities of liver dysfunction. Nutritional assessment using BMI showed that 12.9% were underweight, 41.9% had a normal BMI, while 27.4% were classified as overweight at risk, and 17.7% were obese (Obese I and II combined). MUAC assessment revealed malnutrition in 51.6% of patients, normal nutritional status in 32.3%, and obesity in 11.3%, highlighting a significant prevalence of malnutrition among the cohort.

Figure 2 compares MUAC classifications with BMI categories. Patients with underweight BMI were predominantly malnourished by MUAC, while variability was observed in normal BMI patients. Overweight and obese BMI categories also showed notable malnutrition, emphasizing the need for multimodal nutritional assessment in chronic liver disease patients.

Figure 3 compares Triceps Skinfold Thickness (TSF), Mid-Arm Muscle Circumference (MAMC), and Handgrip Strength (HGS) across normal and malnourished patients. While TSF and MAMC values are higher in the normal group, HGS shows a significant drop in malnourished patients, indicating its reliability in detecting malnutrition. These findings emphasize the utility of multiple parameters in nutritional assessment.

Figure 4 shows the distribution of patients based on their folic acid levels. A larger proportion of patients (n=36) had low folic acid levels compared to those with normal levels (n=26), highlighting a significant prevalence of folic acid deficiency in the study population.

Table 3 summarizes the prevalence and distribution of sarcopenia, nutritional assessments, and biochemical parameters among chronic liver disease (CLD) patients.

- Sarcopenia: Present in 72.6% of patients, with significantly lower Handgrip Strength (HGS: 17.3 ± 5.1 kg) compared to non-sarcopenic patients (30.6 ± 6.2 kg; $p < 0.0001$).
- Subjective Global Assessment (SGA): Most patients were moderately malnourished (SGA B, 67.7%), with a mean albumin level of 3.05 ± 0.58 g/dL. Severely malnourished patients (SGA C, 14.5%) had the lowest albumin levels (2.40 ± 0.36 g/dL), showing significant malnutrition severity ($p < 0.0001$).

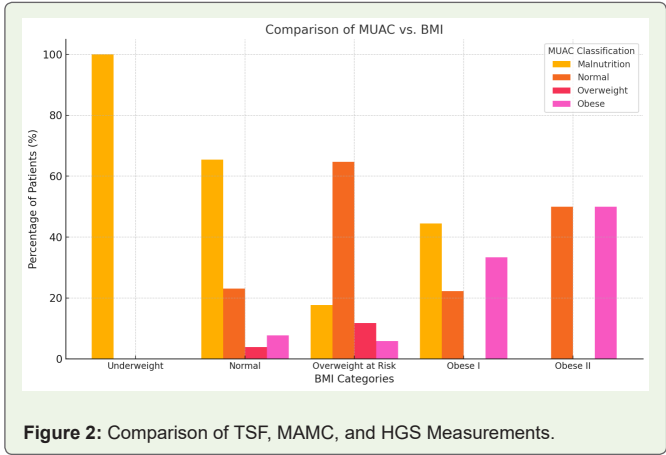


Figure 2: Comparison of TSF, MAMC, and HGS Measurements.

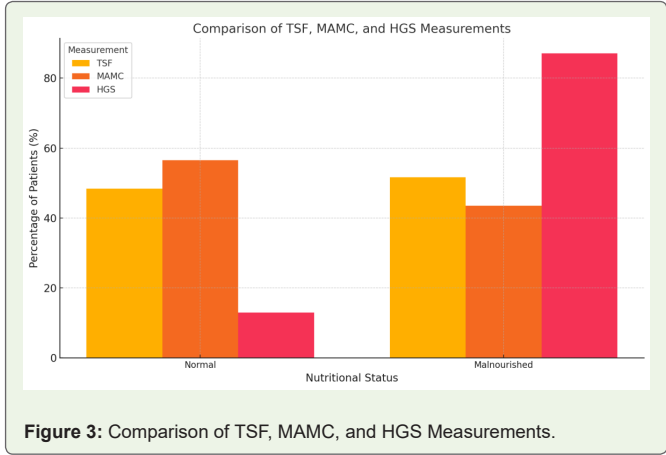


Figure 3: Comparison of TSF, MAMC, and HGS Measurements.

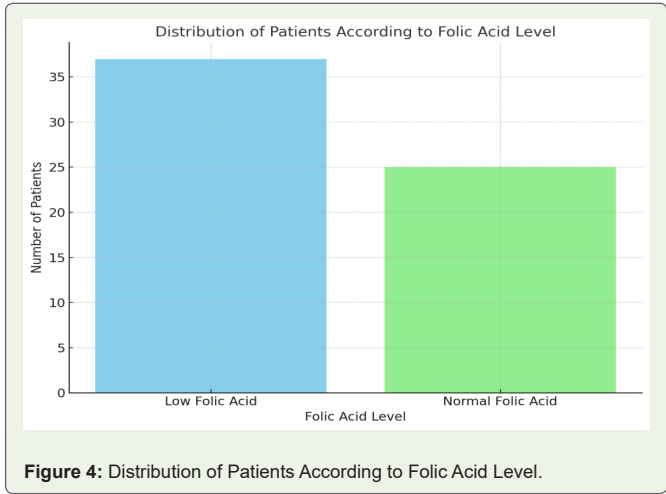


Figure 4: Distribution of Patients According to Folic Acid Level.

- RFH-NPT (Royal Free Hospital Nutritional Prioritizing Tool): High nutritional risk was observed in 72.6% of patients, with a mean BMI of 21.1 ± 2.9 kg/m², while patients with no risk had the highest BMI (26.9 ± 3.1 kg/m²; $p < 0.0001$).
- Mini-Nutritional Assessment (MNA): Nearly half the patients (48.4%) were malnourished with a mean albumin

Table 3: Nutritional and Biochemical Parameters of Chronic Liver Disease Patients

Characteristic	Category	Frequency (n)	Percentage (%)	Mean ± SD	p-Value
Sarcopenia	Yes	45	72.6	HGS: 17.3 ± 5.1 kg	<0.0001
	No	17	27.4	HGS: 30.6 ± 6.2 kg	
SGA (Subjective Global Assessment)	A (Well-Nourished)	11	17.7	Albumin: 3.37 ± 0.69 g/dL	<0.0001
	B (Moderately Malnourished)	42	67.7	Albumin: 3.05 ± 0.58 g/dL	
	C (Severely Malnourished)	9	14.5	Albumin: 2.40 ± 0.36 g/dL	
RFH-NPT (Nutritional Prioritizing Tool)	High Risk	45	72.6	BMI: 21.1 ± 2.9 kg/m²	<0.0001
	Moderate Risk	13	21.0	BMI: 23.7 ± 3.2 kg/m²	
	No Risk	4	6.5	BMI: 26.9 ± 3.1 kg/m²	
MNA (Mini-Nutritional Assessment)	Malnutrition	30	48.4	Albumin: 3.0 ± 0.5 g/dL	0.57
	Risk of Malnutrition	32	51.6	Albumin: 3.1 ± 0.6 g/dL	
Albumin Levels	Low	47	75.8	Albumin: 2.9 ± 0.4 g/dL	<0.0001
	Normal	15	24.2	Albumin: 3.7 ± 0.2 g/dL	
Hemoglobin (Hb) Levels	Low	62	100.0	Hb: 8.7 ± 1.2 g/dL	<0.0001
	Normal	0	0.0	Not applicable	
Vitamin B12 Levels	Low	42	67.7	B12: 155 ± 38 pg/mL	<0.0001
	Normal	20	32.3	B12: 288 ± 32 pg/mL	
Folic Acid Levels	Low	37	59.7	Folic Acid: 3.5 ± 1.1 ng/mL	0.0046
	Normal	25	40.3	Folic Acid: 7.2 ± 0.9 ng/mL	

level of 3.0 ± 0.5 g/dL. No significant difference in albumin levels was observed between malnourished patients and those at risk (p=0.57).

- Biochemical Parameters:
 - Albumin Levels: Low in 75.8% of patients (mean: 2.9 ± 0.4 g/dL), significantly lower than normal albumin levels (3.7 ± 0.2 g/dL; p<0.0001).
 - Hemoglobin Levels: All patients had low hemoglobin (mean: 8.7 ± 1.2 g/dL; p<0.0001).
 - Vitamin B12 Levels: Deficiency was noted in 67.7% of patients, with significantly lower levels (155 ± 38 pg/mL) compared to those with normal levels (288 ± 32 pg/mL; p<0.0001).
 - Folic Acid Levels: Low in 59.7% of patients, with a mean level of 3.5 ± 1.1 ng/mL, significantly lower than the normal range (7.2 ± 0.9 ng/mL; p=0.0046).

Table 4 Shows, MELD-Na: Significantly higher in patients with advanced disease (CPT C). BMI and MUAC: Decrease as CPT scores increase, indicating worsening nutritional status.TSF: Lower in CPT C, reflecting higher fat depletion in advanced disease.Albumin and Hb: Lower in CPT C, showing more severe hypoalbuminemia and anemia in advanced liver disease.MAMC, HGS, and MNA: Show no significant difference but trend lower in advanced stages.

Table 5 Compares clinical, nutritional, and biochemical parameters across CPT score categories (A, B, and C).

1. Significant Findings: MELD-Na, BMI, MUAC, TSF, albumin, hemoglobin, folic acid, and vitamin B12 levels decline significantly with worsening CPT scores, indicating progressive liver dysfunction and nutritional deterioration.

2. Non-Significant Findings: Age, MAMC, HGS, MNA, and RFH-NPT show no significant differences but suggest worsening trends in advanced disease stages.

The Table 6 analysis of nutritional and functional assessments across Subjective Global Assessment (SGA) categories (A: Well-nourished, B: Moderately malnourished, and C: Severely malnourished) highlights significant trends in malnutrition and sarcopenia. Malnutrition, as assessed by Mid-Upper Arm Circumference (MUAC), was significantly higher in SGA B (68.75%) and SGA C (28.13%) compared to SGA A (3.13%), reflecting greater muscle wasting in patients with advanced SGA severity (p<0.001). Triceps Skinfold Thickness (TSF) values showed higher rates of adipopenia in SGA B (78.13%) and SGA C (18.75%), while SGA A had predominantly normal TSF values (33.33%), confirming significant fat loss with advancing malnutrition (p=0.007). Similarly, Mid-Arm Muscle Circumference (MAMC) revealed severe muscle depletion in SGA C (29.63% malnourished) and SGA B (66.67% malnourished), with only 3.7% of malnourished cases in SGA A, indicating progressive muscle wasting (p=0.002). Sarcopenia, assessed separately, was observed in 93.33% of SGA B and 100% of SGA C patients, while only 6.67% of SGA A patients exhibited muscle loss, further highlighting the correlation between SGA severity and sarcopenia (p<0.001). Handgrip Strength (HGS), a functional marker, demonstrated significant declines with increasing SGA severity. Normal HGS was primarily seen in SGA A (50%) and B (50%), but none of the SGA C patients had normal functional strength. Conversely, malnourishment by HGS was highest in SGA B (70.37%) and SGA C (16.67%), with fewer cases in SGA A (12.96%), reflecting a significant loss of functional capacity as malnutrition worsened (p=0.028). The Royal Free Hospital Nutritional Prioritizing Tool (RFH-NPT) further identified high nutritional risk in 77.78% of SGA B and 20% of SGA C patients, with all SGA A patients classified as either low or moderate risk. High nutritional risk in SGA B and

Table 4: Multivariate Analysis of CPT Score and Nutritional Assessments

Parameter	CPT A (Mean ± SD)	CPT B (Mean ± SD)	CPT C (Mean ± SD)	p-Value
Age (years)	47.06 ± 13.48	49.45 ± 14.16	50.68 ± 10.68	0.648
MELD-Na	16.28 ± 3.54	18.91 ± 2.51	25.91 ± 4.23	<0.001
BMI (kg/m²)	22.66 ± 4.13	24.21 ± 3.36	20.20 ± 2.40	<0.001
MUAC (cm)	23.51 ± 4.56	23.85 ± 3.55	20.98 ± 3.39	0.017
TSF (mm)	9.22 ± 2.84	10.57 ± 2.75	7.77 ± 2.18	0.003
MAMC (cm)	20.55 ± 4.01	20.56 ± 3.18	18.49 ± 3.18	0.077
HGS (kg)	22.59 ± 8.21	21.90 ± 8.14	18.90 ± 6.56	0.294
MNA	16.18 ± 3.38	16.48 ± 4.97	15.86 ± 2.82	0.567
Albumin (g/dL)	3.27 ± 0.66	3.12 ± 0.65	2.70 ± 0.47	0.014
Hemoglobin (g/dL)	8.65 ± 1.20	9.93 ± 1.16	8.37 ± 1.47	<0.001
RFH-NPT	1.89 ± 0.90	1.77 ± 1.11	2.41 ± 0.67	0.107

Table 5: Clinical, Nutritional, and Biochemical Parameters Across Child-Pugh-Turcotte (CPT) Scores

Parameter	CPT A (Mean ± SD)	CPT B (Mean ± SD)	CPT C (Mean ± SD)	p-Value
Age (years)	47.06 ± 13.48	49.45 ± 14.16	50.68 ± 10.68	0.648
MELD-Na	16.28 ± 3.54	18.91 ± 2.51	25.91 ± 4.23	<0.001
BMI (kg/m²)	22.66 ± 4.13	24.21 ± 3.36	20.20 ± 2.40	<0.001
MUAC (cm)	23.51 ± 4.56	23.85 ± 3.55	20.98 ± 3.39	0.017
TSF (mm)	9.22 ± 2.84	10.57 ± 2.75	7.77 ± 2.18	0.003
MAMC (cm)	20.55 ± 4.01	20.56 ± 3.18	18.49 ± 3.18	0.077
HGS (kg)	22.59 ± 8.21	21.90 ± 8.14	18.90 ± 6.56	0.294
MNA	16.18 ± 3.38	16.48 ± 4.97	15.86 ± 2.82	0.567
Albumin (g/dL)	3.27 ± 0.66	3.12 ± 0.65	2.70 ± 0.47	0.014
Hemoglobin (g/dL)	8.65 ± 1.20	9.93 ± 1.16	8.37 ± 1.47	<0.001
Folic Acid (ng/mL)	7.25 ± 1.32	6.80 ± 1.15	4.89 ± 1.05	<0.001
Vitamin B12 (pg/mL)	230.5 ± 45.6	180.3 ± 32.4	140.7 ± 27.9	<0.001
RFH-NPT	1.89 ± 0.90	1.77 ± 1.11	2.41 ± 0.67	0.107

Table 6: Nutritional and Functional Assessments by SGA

Parameter	SGA A (n = 11)	SGA B (n = 42)	SGA C (n = 9)	Total (n = 62)	p-Value	Significance
MUAC	Malnutrition: 3.13%	Malnutrition: 68.75%	Malnutrition: 28.13%	Malnutrition: 51.6%	<0.001	Significant
	Normal: 20%	Normal: 80%	Normal: 0%	Normal: 32.3%		
	Overweight: 0%	Overweight: 100%	Overweight: 0%	Overweight: 4.8%		
	Obese: 85.71%	Obese: 14.29%	Obese: 0%	Obese: 11.3%		
TSF	Normal: 33.33%	Normal: 56.67%	Normal: 10%	Normal: 48.4%	0.007	Significant
	Malnourished: 3.13%	Malnourished: 78.13%	Malnourished: 18.75%	Malnourished: 51.6%		
MAMC	Normal: 28.57%	Normal: 68.57%	Normal: 2.86%	Normal: 56.5%	0.002	Significant
	Malnourished: 3.7%	Malnourished: 66.67%	Malnourished: 29.63%	Malnourished: 43.5%		
Sarcopenia	No: 47.06%	No: 52.94%	No: 0%	No: 27.4%	<0.001	Significant
	Yes: 6.67%	Yes: 93.33%	Yes: 100%	Yes: 72.6%		
HGS	Normal: 50%	Normal: 50%	Normal: 0%	Normal: 12.9%	0.028	Significant
	Malnourished: 12.96%	Malnourished: 70.37%	Malnourished: 16.67%	Malnourished: 87.1%		
RFH-NPT	No risk: 100%	No risk: 0%	No risk: 0%	No risk: 6.5%	<0.001	Significant
	Moderate risk: 46.15%	Moderate risk: 53.85%	Moderate risk: 0%	Moderate risk: 21%		
	High risk: 2.22%	High risk: 77.78%	High risk: 20%	High risk: 72.6%		

C groups underscores the escalating burden of malnutrition and associated complications in advanced SGA categories (p<0.001).

Table 7 Highlights the significant differences in clinical, nutritional, and functional parameters across SGA categories. The progression from SGA A to SGA B&C shows a clear deterioration in liver function, body composition, and overall nutritional status, underscoring the impact of advanced malnutrition and liver disease.

These findings emphasize the need for early nutritional interventions to improve outcomes in patients with moderate to severe malnutrition. Let me know if you need further clarifications!

1. Significant Parameters:
- o MELD-Na: Increased significantly from SGA A (16.64) to SGA B&C (21.49), indicating worsening liver disease severity.

Table 7: Multivariate Analysis of SGA and Study Components

Parameter	SGA A (Mean \pm SD)	SGA B (Mean \pm SD)	SGA C (Mean \pm SD)	SGA B&C (Mean \pm SD)	p-Value
Age (years)	44.09 \pm 14.21	50.83 \pm 12.41	47.78 \pm 11.65	50.29 \pm 12.22	0.313
MELD-Na	16.64 \pm 2.62	21.02 \pm 5.57	23.67 \pm 4.12	21.49 \pm 5.40	0.007
BMI (kg/m²)	25.01 \pm 3.69	22.01 \pm 3.22	20.60 \pm 4.43	21.76 \pm 3.46	0.014
MUAC (cm)	27.27 \pm 3.36	22.44 \pm 3.18	18.56 \pm 2.49	21.75 \pm 3.39	<0.001
TSF (mm)	11.23 \pm 1.57	9.26 \pm 2.59	6.33 \pm 2.83	8.75 \pm 2.83	0.005
MAMC (cm)	23.82 \pm 3.35	19.48 \pm 2.84	16.54 \pm 2.18	18.96 \pm 2.94	<0.001
HGS (kg)	30.56 \pm 6.17	19.94 \pm 6.17	14.51 \pm 5.35	18.98 \pm 6.34	<0.001
MNA	16.73 \pm 6.37	16.13 \pm 2.66	15.67 \pm 4.44	16.04 \pm 3.02	0.324
Albumin (g/dL)	3.37 \pm 0.69	3.05 \pm 0.58	2.40 \pm 0.36	2.94 \pm 0.60	0.045
Hemoglobin (g/dL)	9.77 \pm 1.41	8.95 \pm 1.51	8.34 \pm 0.72	8.84 \pm 1.42	0.070
RFH-NPT	0.73 \pm 0.65	2.19 \pm 0.71	2.89 \pm 0.60	2.31 \pm 0.73	<0.001

- o BMI, MUAC, TSF, MAMC: All showed significant declines from SGA A to SGA B&C, reflecting progressive malnutrition and body composition changes.
- o HGS: A marked reduction in handgrip strength was noted in SGA B&C (18.98) compared to SGA A (30.56), indicating reduced functional capacity.
- o RFH-NPT: Higher scores in SGA B&C indicate a greater risk of nutritional complications.
- o Albumin: Levels decreased significantly from SGA A (3.37) to SGA B&C (2.94), reflecting worsening liver synthetic function and malnutrition.

2. Non-Significant Parameters:

- o Age: No significant difference was observed among the groups.
- o MNA and Hemoglobin: Despite a trend toward worsening nutritional scores and anemia in SGA B&C, the differences were not statistically significant.

Table 8 discuss following

1. **Age:** Alcoholic patients were slightly older on average (51.56 \pm 9.09 years) compared to non-alcoholic patients (45.92 \pm 16.07 years), but this difference was not statistically significant (p=0.121).
2. **MELD-Na:** Although alcoholic patients had a higher mean MELD-Na score (21.78 \pm 5.34) compared to non-alcoholic patients (19.04 \pm 5.02), this difference was not statistically significant (p=0.058). However, the trend suggests that alcoholic patients may have more severe liver dysfunction.
3. **BMI (Body Mass Index):** Alcoholic patients had a lower BMI (21.69 \pm 3.57 kg/m²) compared to non-alcoholic patients (23.24 \pm 3.72 kg/m²), indicating greater nutritional compromise in alcoholics. However, this difference was not statistically significant (p=0.113).
4. **MUAC (Mid-Upper Arm Circumference):** MUAC values were slightly lower in alcoholic patients (22.54 \pm 3.84 cm) than in non-alcoholic patients (23.00 \pm 4.22 cm), but the difference was not significant (p=0.338).

Table 8: Multivariate Analysis of Alcohol Abuse and Nutritional Components

Parameter	Non-Alcoholic (Mean \pm SD)	Alcoholic (Mean \pm SD)	p-Value
Age (years)	45.92 \pm 16.07	51.56 \pm 9.09	0.121
MELD-Na	19.04 \pm 5.02	21.78 \pm 5.34	0.058
BMI (kg/m²)	23.24 \pm 3.72	21.69 \pm 3.57	0.113
MUAC (cm)	23.00 \pm 4.22	22.54 \pm 3.84	0.338
TSF (mm)	9.90 \pm 2.79	8.67 \pm 2.75	0.056
MAMC (cm)	19.89 \pm 3.75	19.78 \pm 3.41	0.568
HGS (kg)	20.82 \pm 7.31	21.19 \pm 8.04	0.926
MNA	17.39 \pm 3.41	15.39 \pm 3.88	0.176
Albumin (g/dL)	3.32 \pm 0.64	2.79 \pm 0.53	0.002
Hemoglobin (g/dL)	8.84 \pm 1.65	9.13 \pm 1.30	0.668
RFH-NPT	1.92 \pm 0.93	2.11 \pm 0.95	0.404

5. **TSF (Triceps Skinfold Thickness):** Alcoholic patients showed a trend toward lower TSF (8.67 \pm 2.75 mm) compared to non-alcoholic patients (9.90 \pm 2.79 mm), reflecting possible adipose tissue loss. However, the difference did not reach statistical significance (p=0.056).
6. **MAMC (Mid-Arm Muscle Circumference):** MAMC values were almost identical between alcoholic (19.78 \pm 3.41 cm) and non-alcoholic (19.89 \pm 3.75 cm) patients, with no significant difference (p=0.568).
7. **HGS (Handgrip Strength):** Handgrip strength was slightly higher in alcoholic patients (21.19 \pm 8.04 kg) compared to non-alcoholic patients (20.82 \pm 7.31 kg), but this difference was not statistically significant (p=0.926).
8. **MNA (Mini-Nutritional Assessment):** MNA scores were lower in alcoholic patients (15.39 \pm 3.88) compared to non-alcoholic patients (17.39 \pm 3.41), suggesting worse nutritional status in alcoholics, but the difference was not statistically significant (p=0.176).
9. **Albumin:** Alcoholic patients had significantly lower albumin levels (2.79 \pm 0.53 g/dL) compared to non-alcoholic patients (3.32 \pm 0.64 g/dL), with this difference being statistically significant (p=0.002). This indicates poorer liver synthetic function and more severe malnutrition in alcoholic patients.

10. **Hemoglobin:** Mean hemoglobin levels were slightly higher in alcoholic patients (9.13 ± 1.30 g/dL) than in non-alcoholic patients (8.84 ± 1.65 g/dL), but this difference was not statistically significant ($p=0.668$).

11. **RFH-NPT (Royal Free Hospital Nutritional Prioritizing Tool):** Alcoholic patients had slightly higher RFH-NPT scores (2.11 ± 0.95) than non-alcoholic patients (1.92 ± 0.93), but the difference was not significant ($p=0.404$).

(a) Sarcopenia Prevalence in Alcoholic vs. Non-Alcoholic CLD Patients: This graph illustrates the higher prevalence of sarcopenia in alcoholic patients compared to non-alcoholic patients. Among alcoholic patients, 83.3% had sarcopenia, whereas only 53.8% of non-alcoholic patients exhibited sarcopenia. The results highlight the significant impact of alcohol consumption on muscle wasting in chronic liver disease (CLD) patients.

(b) Folic Acid Levels in Alcoholic vs. Non-Alcoholic CLD

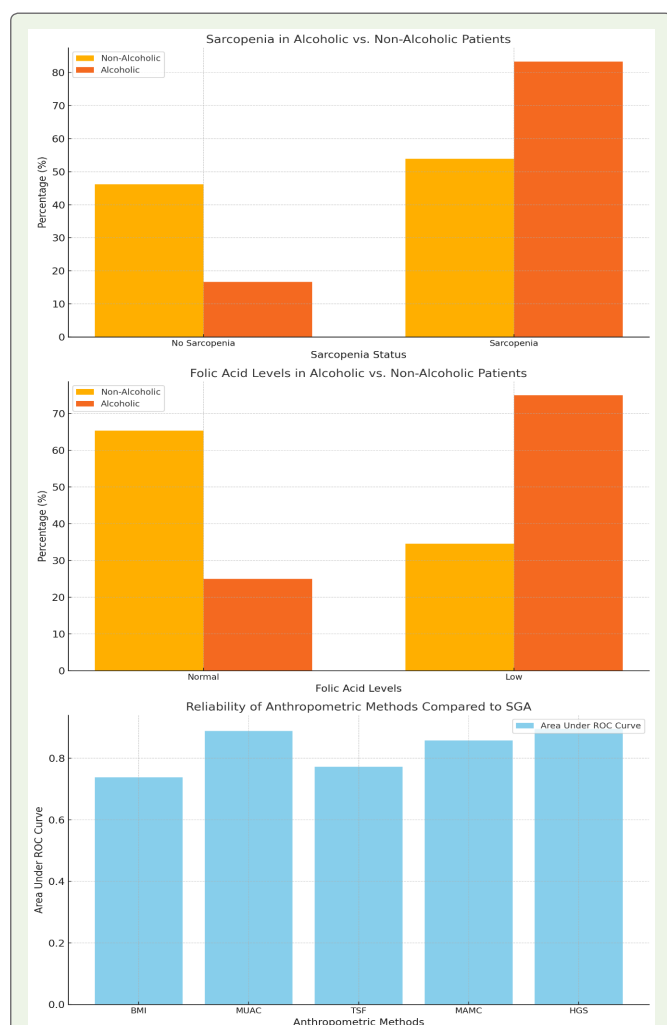


Figure 5: Nutritional and Diagnostic Comparisons in Chronic Liver Disease Patients.

Patients: This graph compares folic acid levels between alcoholic and non-alcoholic CLD patients. A higher proportion of alcoholic patients (75%) had low folic acid levels compared to non-alcoholic patients (34.6%). The findings underscore the adverse effects of alcohol on micronutrient deficiencies, particularly folic acid, in CLD patients.

(c) Reliability of Anthropometric Methods Compared to SGA: This graph compares the reliability of various anthropometric methods in diagnosing malnutrition, using the Subjective Global Assessment (SGA) as the reference standard. Handgrip Strength (HGS) showed the highest Area Under the Curve ($AUC = 0.895$), followed by Mid-Upper Arm Circumference (MUAC), Mid-Arm Muscle Circumference (MAMC), and Triceps Skinfold Thickness (TSF). Body Mass Index (BMI) demonstrated the lowest reliability ($AUC = 0.738$). These results establish HGS as the most effective anthropometric tool for nutritional assessment in CLD patients.

Discussion

This hospital-based prospective observational study evaluated the nutritional status of chronic liver disease (CLD) patients, focusing on its severity and etiology. A total of 62 patients were included, with the majority (54.8%) aged between 40 and 60 years, a demographic pattern aligning with prior studies, such as that by Mukherjee et al., which also reported a median age of 43 years. The current study observed a notable male predominance (75.8%), with a male-to-female ratio of 3.1:1, comparable to earlier findings in India. However, a significant proportion (19.4%) of the patients were in the 30-40 age group, suggesting an alarming trend of earlier disease onset and increased healthcare burden [19,20].

Etiological Patterns and Disease Severity

Alcoholic liver disease (ALD) was identified as the leading cause of CLD (51.6%), followed by non-alcoholic fatty liver disease (NAFLD, 16.1%), while hepatitis B and C together accounted for only 12.9%. These findings are consistent with a nationwide transition in CLD etiology, where alcohol abuse and NAFLD are replacing viral causes, as highlighted in studies by Mukherjee et al., Sarin et al., and Anand et al. The rising incidence of NAFLD in the current study aligns with increasing BMI and diabetes prevalence, reflecting a broader epidemiological shift in India [21,22,23].

In alignment with findings by Nunes et al., this study demonstrated that alcoholic patients exhibited greater disease severity. A significantly higher proportion of alcoholic patients fell into Child-Turcotte-Pugh (CTP) grade B or C categories, with correspondingly elevated MELD-Na scores, underscoring their worsened liver function. Furthermore, the study by Ciocirlan et al. corroborated the distribution of CTP scores observed here (A: 29%, B: 35.5%, C: 35.5%), indicating a high burden of decompensated CLD in both cohorts [24,25].

Malnutrition and Sarcopenia in CLD

The prevalence of malnutrition in CLD patients was assessed using various tools. Subjective Global Assessment (SGA) identified high risk (67.7%) and severe malnutrition (14.5%), findings consistent with Aguila et al., who reported similar associations between malnutrition and higher CTP and MELD-Na scores. Notably, malnutrition severity significantly correlated with SGA categories, with higher SGA scores reflecting worse nutritional outcomes [26,27].

Anthropometric measurements revealed malnutrition in a significant proportion of patients: 51.6% by TSF and 43.5% by MAMC. These results closely align with Sherpa et al. and Campillo et al., who also reported high malnutrition rates in advanced CLD patients[28,29,30]. Interestingly, the current study found BMI less predictive of nutritional status due to confounding factors such as ascites and fluid retention, echoing findings by De Mattos et al.

Sarcopenia, a critical manifestation of malnutrition in CLD, was identified in 72.6% of patients, significantly higher in alcoholic patients (83.33%) compared to non-alcoholic patients (53.85%). These findings support prior studies by Dasarathy et al. and Kumar et al., which highlighted alcohol's deleterious effects on muscle mass and metabolism. The strong association between SGA categories and sarcopenia further validates the reliability of SGA as a malnutrition assessment tool[31].

Folic Acid and Nutritional Assessment Reliability

Alcoholic patients had significantly lower folic acid levels (75% deficient) compared to non-alcoholic patients (34.62%), corroborating studies by Nunes et al. This deficiency reflects a higher malnutrition risk in ALD. Among the anthropometric methods evaluated, handgrip strength (HGS) emerged as the most reliable tool for detecting malnutrition (AUC: 0.895), outperforming MUAC, MAMC, and TSF. This finding aligns with studies by Magdy et al. and Johnson et al., which also endorsed HGS for its predictive value in cirrhosis complications[34,35].

Strengths of the Study

1. One of the few Indian studies addressing malnutrition in CLD comprehensively.
2. Adequate sample size ensuring robust statistical conclusions.
3. Conducted in a tertiary referral hospital, reflecting the broader Eastern Indian population.
4. Adherence to ESPEN 2006 guidelines with standardized anthropometric and biochemical assessments.

Limitations of the Study

1. A larger sample size might yield more definitive results.
2. The cross-sectional design precluded longitudinal analysis of treatment outcomes.
3. Advanced diagnostic tools like CT SMI, DEXA, and BIA scans were not employed to detect sarcopenia.

Conclusion

Malnutrition is a pervasive complication in chronic liver disease (CLD) and worsens with disease severity. CLD patients commonly experience not only protein-calorie malnutrition but also adipopenia, sarcopenia, muscle wasting, frailty, and micronutrient deficiencies. Malnutrition serves as an independent predictor of mortality and complications in CLD, highlighting the critical need for comprehensive nutritional screening, even in early stages of the disease.

Anthropometric and questionnaire-based assessment methods reliably estimate the severity of malnutrition. In this study, Subjective Global Assessment (SGA), Royal Free Hospital Nutritional Prioritizing Tool (RFH-NPT), Handgrip Strength (HGS), and Mid-Arm Muscle Circumference (MAMC) were found to be reliable, reproducible, and effective tools for assessing malnutrition. The findings also underscore the detrimental role of alcohol as an etiological factor, significantly exacerbating malnutrition. Even obese alcoholic patients, as classified by BMI, were found to have underlying sarcopenia, contributing to increased complications and mortality.

The present study emphasizes the urgent need to prioritize nutritional assessment in CLD patients. Early detection and management of malnutrition can significantly improve prognosis and clinical outcomes. Increased awareness among clinicians and the integration of nutritional assessments into routine CLD management protocols will pave the way for better care and outcomes in this vulnerable patient population.

References

1. Vieira PM, De-Souza DA, Oliveira LC (2013) Nutritional assessment in hepatic cirrhosis; clinical, anthropometric, biochemical and hematological parameters. *Nutr Hosp* 28: 1615-21.
2. WHO (2020) World Health Organization. Newsroom Q & A Detail: Malnutrition. [Internet].
3. Kalaitzakis E, Simrén M, Olsson R, Henfridsson P, Hugosson I, et al. (2006) Gastrointestinal symptoms in patients with liver cirrhosis: associations with nutritional status and health-related quality of life. *Scand J Gastroenterol* 41: 1464-1472.
4. Huisman EJ, Trip EJ, Siersema PD, van Hoek B, van Erpecum KJ (2011) Protein energy malnutrition predicts complications in liver cirrhosis. *Eur J Gastroenterol Hepatol*. 23: 982-989.
5. Merli M, Lucidi C, Giannelli V, Giusto M, Riggio O, et al. (2010) Falcone M, Ridola L, Attili AF, Venditti M. Cirrhotic patients are at risk for health care-associated bacterial infections. *Clin Gastroenterol Hepatol* 8: 979-985.
6. Chadalavada R, Sappati Biyyani RS, Maxwell J, Mullen K (2010) Nutrition in hepatic encephalopathy. *Nutr Clin Pract* 25: 257-264.
7. Henkel AS, Buchman AL (2006) Nutritional support in patients with chronic liver disease. *Nat Clin Pract Gastroenterol Hepatol* 3: 202-209.
8. Silva M, Gomes S, Peixoto A, Torres-Ramvalho P, Cardoso H, et al. (2015) Nutrition in Chronic Liver Disease. *GE Port J Gastroenterol* 22: 268-276.
9. Rahimtoola SH (2014) Current Problems in Cardiology. Foreword. *Curr Probl Cardiol*. 39: 157.
10. Evans WJ, Morley JE, Argilés J, Bales C, Baracos V, et al. (2008) Cachexia: a new definition. *Clin Nutr* 27: 793-799.
11. Plauth M, Schütz ET (2002) Cachexia in liver cirrhosis. *Int J Cardiol* 85: 83-87.
12. Nunes G, Santos CA, Barosa R, Fonseca C, Barata AT, et al. (2017) OUTCOME AND NUTRITIONAL ASSESSMENT OF CHRONIC LIVER DISEASE PATIENTS USING ANTHROPOMETRY AND SUBJECTIVE GLOBAL ASSESSMENT. *Arq Gastroenterol* 54: 225-231.
13. Pimpin L, Cortez-Pinto H, Negro F, Corbould E, Lazarus JV, et al. (2018) EASL HEPALHEALTH Steering Committee. Burden of liver disease in Europe: Epidemiology and analysis of risk factors to identify prevention policies. *J Hepatol* 69: 718-735.
14. Rivera Irigoin R, Abilés J (2012) Soporte nutricional en el paciente con cirrosis hepática [Nutritional support in patients with liver cirrhosis]. *Gastroenterol Hepatol* 35: 594-601.

15. Plauth M, Cabré E, Riggio O, Assis-Camilo M, Pirlich M, et al. (2006) DGEM (German Society for Nutritional Medicine); Ferenci P, Holm E, Vom Dahl S, Müller MJ, Nolte W; ESPEN (European Society for Parenteral and Enteral Nutrition). ESPEN Guidelines on Enteral Nutrition: Liver disease. *Clin Nutr* 25: 285-94.
16. Adams DH (2007) Sleisenger and Fordtran's Gastrointestinal and Liver Disease. *Gut* 56: 1175.
17. Mondal D, Das K, Chowdhury A (2022) Epidemiology of Liver Diseases in India. *Clin Liver Dis (Hoboken)* 19: 114-117.
18. Mokdad AA, Lopez AD, Shahrz S, Lozano R, Mokdad AH, et al. (2014) Liver cirrhosis mortality in 187 countries between 1980 and 2010: a systematic analysis. *BMC Med* 12: 145.
19. Mukherjee PS, Vishnubhatla S, Amarapurkar DN, Das K, Sood A, et al. (2017) Chawla YK, Eapen CE, Boddu P, Thomas V, Varshney S, Hidangmayum DS, Bhaumik P, Thakur B, Acharya SK, Chowdhury A. Etiology and mode of presentation of chronic liver diseases in India: A multi centric study. *PLoS One* 12: e0187033.
20. Tessari P (2003) Protein metabolism in liver cirrhosis: from albumin to muscle myofibrils. *Curr Opin Clin Nutr Metab Care* 6: 79-85.
21. Bianchi G, Marzocchi R, Agostini F, Marchesini G (2005) Update on nutritional supplementation with branched-chain amino acids. *Curr Opin Clin Nutr Metab Care* 8: 83-87.
22. Bilbao I, Armadans L, Lazaro JL, Hidalgo E, Castells L, et al. (2003) Margarit C. Predictive factors for early mortality following liver transplantation. *Clin Transplant* 17: 401-411.
23. Alberino F, Gatta A, Amodio P, Merkel C, Di Pascoli L, et al. (2001) Nutrition and survival in patients with liver cirrhosis. *Nutrition* 17: 445-450.
24. Gunsar F, Raimondo ML, Jones S, Terreni N, Wong C, et al. (2006) Nutritional status and prognosis in cirrhotic patients. *Aliment Pharmacol Ther* 24: 563-752.
25. Lata J, Husová L, Juránková J, Senkyřík M, Díte P, et al. (2006) Factors participating in the development and mortality of variceal bleeding in portal hypertension—possible effects of the kidney damage and malnutrition. *Hepato gastroenterology* 53: 420-425.
26. Daphnee DK, John S, Vaidya A, et al. (2017) Handgrip strength: A reliable, reproducible, cost-effective tool to assess nutritional status and outcomes of cirrhotics awaiting liver transplant. *Clin Nutr ESPEN* 19: 49-53.
27. McClain CJ, Barve SS, Barve A, Marsano L (2011) Alcoholic liver disease and malnutrition. *Alcohol Clin Exp Res* 35: 815-820.
28. Kumar R (2018) Hepatogenous Diabetes: An Underestimated Problem of Liver Cirrhosis. *Indian J Endocrinol Metab* 22: 552-559.
29. Reccia I, Kumar J, Akladios C, Virdis F, Pai M, et al. (2017) Habib N, Spalding D. Non-alcoholic fatty liver disease: A sign of systemic disease. *Metabolism* 72: 94-108.
30. Sinclair M, Gow PJ, Grossmann M, Angus PW (2016) Review article: sarcopenia in cirrhosis—etiology, implications and potential therapeutic interventions. *Aliment Pharmacol Ther* 43: 765-77.
31. Haj Ali S, Abu Sneineh A, Hasweh R (2022) Nutritional assessment in patients with liver cirrhosis. *World J Hepatol* 14: 1694-1703.