SSR Institute of International Journal of Life Sciences
ISSN (O): 2581-8740 | ISSN (P): 2581-8732
Singh, 2025
doi: 10.21276/SSR-IIJLS.2025.11.5.44

Original Article

open access

Nutritional Status and Its Association with Severity in Patients with Alcoholic Liver Disease: A Cross-sectional Study from Northern India

Kuldeep Singh*

Consultant Gastroenterologist, Hepatologist and Endoscopist, Department of Gastroenterology, Apex Hospital, Malviya Nagar, Jaipur, India

***Address for Correspondence:** Dr. Kuldeep Singh, Consultant Gastroenterologist, Hepatologist and Endoscopist, Department of Gastroenterology, Apex Hospital, Malviya Nagar, Jaipur, India
E-mail: kuldeepsingh122@gmail.com **& ORCID ID:** https://orcid.org/0000-0001-9163-0996

Received: 02 Jun 2025/ Revised: 24 Jul 2025/ Accepted: 27 Aug 2025

ABSTRACT

Background: Alcoholic liver disease (ALD) is a major cause of chronic liver disease worldwide. Malnutrition is common in cirrhosis and worsens clinical outcomes. This study aimed to assess nutritional status in ALD patients and its association with disease severity.

Methods: A cross-sectional study was conducted at the Department of Gastroenterology, Apex Hospital, Malviya Nagar, Jaipur, from June 2024 to May 2025. A total of 320 male patients with ALD were enrolled. Nutritional status was assessed using anthropometry, body composition (Tanita BC-601 analyzer), dietary recall, and biochemical parameters. Disease severity was graded using the Child-Pugh score. Statistical analysis was performed using SPSS v21.

Results: The mean age was 53.8 ± 8.9 years. Based on BMI, 43.1% were normal, 7.5% underweight, 31.3% overweight, and 18.2% obese. However, 85.6% had reduced mid-arm circumference and 87.5% had decreased muscle mass. Total body water increased in 85.6%, while 86.3% revealed a reduction in fat mass. Anemia was present in 86.3%, hypoalbuminemia in 93%, and lymphopenia in 48.8%. Malnutrition prevalence rose with longer alcohol intake and an advanced Child-Pugh stage. Severe malnutrition was most common in Child-Pugh C (30.6%), compared to none in Child-Pugh A ($p < 0.001$).

Conclusion: Malnutrition is highly prevalent among ALD patients in Jaipur. BMI underestimates nutritional deficits due to fluid retention; thus, anthropometric and body composition measures are more reliable. Early nutritional assessment and intervention should be integrated into the management of ALD to improve outcomes.

Key-words: Alcoholic liver disease, Malnutrition, Cirrhosis, Nutritional assessment, Child-Pugh score, Body composition

INTRODUCTION

Alcoholic liver disease (ALD) is a major public health challenge worldwide [1,2], contributing significantly to morbidity and mortality. According to the World Health Organization, alcohol consumption accounts for nearly 3 million deaths each year [3], with liver disease being one of the primary causes. In India, alcohol consumption has risen steadily over the past two decades [4], paralleling an increase in the burden of ALD.

How to cite this article
Singh K. Nutritional Status and Its Association with Severity in Patients with Alcoholic Liver Disease: A Cross-sectional Study from Northern India. SSR Inst Int J Life Sci., 2025; 11(5): 8503-8508.

Access this article online
https://iijls.com/

Pathophysiologically, ALD results from chronic excessive alcohol intake leading to a spectrum of hepatic injuries [1,2] ranging from fatty liver to alcoholic hepatitis, fibrosis, and cirrhosis. Ethanol metabolism generates toxic metabolites, particularly acetaldehyde and reactive oxygen species [5], which induce oxidative stress, lipid peroxidation, mitochondrial injury, and activation of proinflammatory cytokines. These mechanisms promote hepatocellular damage and the progression of fibrosis.

Malnutrition is a frequent and severe complication of ALD, with prevalence estimates ranging from 20% in compensated cirrhosis to as high as 80–100% [6-9] in patients with decompensated cirrhosis. Mechanisms of malnutrition include reduced dietary intake due to anorexia, early satiety from ascites, altered taste perception, and psychosocial neglect. In addition, ALD is

doi: 10.21276/SSR-IIJLS.2025.11.5.44

associated with malabsorption, increased protein catabolism, and hypermetabolic states, which accelerate muscle wasting and sarcopenia. Protein-energy malnutrition is a key determinant of prognosis [10-12], associated with higher rates of infection, hepatic encephalopathy, ascites, poor quality of life, and increased mortality. Importantly, malnutrition before liver transplantation significantly worsens post-transplant outcomes.

Despite its clinical importance, nutritional assessment is often overlooked in ALD management. Conventional prognostic scores, such as the Child-Pugh classification and the Model for End-Stage Liver Disease (MELD) [13], emphasise biochemical and clinical parameters but fail to account for nutritional status. Recent studies have emphasizeed the importance of integrating nutritional assessment into prognostic models [14-16]. Anthropometric measures such as mid-arm circumference and triceps skinfold thickness [6,7,15], as well as advanced modalities like dual-energy X-ray absorptiometry and CT-based skeletal muscle assessment [17-19], have been used to quantify malnutrition in cirrhosis.

In India, studies evaluating the nutritional status of ALD patients remain limited, with most conducted in tertiary government hospitals. There is a paucity of data from private tertiary care centers such as Apex Hospital, Jaipur [4,20]. This study aimed to assess the prevalence and patterns of malnutrition in ALD patients at our center and correlate these findings with alcohol intake duration and Child-Pugh class, thereby contributing to the regional epidemiological evidence and emphasizing the role of nutritional assessment in ALD management.

MATERIALS AND METHODS

Study Design and Setting- A cross-sectional study was conducted at the Department of Gastroenterology, Apex Hospital, Malviya Nagar, Jaipur, Rajasthan, India. The study period was from June 2024 to May 2025.

Study Population- A total of 320 male patients aged above 18 years with a confirmed diagnosis of alcoholic liver disease (ALD) were included in the study.

Inclusion Criteria- Patients with a history of significant alcohol intake, clinical features suggestive of ALD, and abnormal liver function tests consistent with alcoholic liver injury were enrolled.

Exclusion Criteria- Patients with viral hepatitis, autoimmune liver disease, metabolic liver disorders, or those on hepatotoxic drugs were excluded from the study.

Data Collection- Each participant underwent a detailed clinical evaluation, which included demographic information, a history of alcohol consumption, and disease duration. The diagnosis of ALD was established based on clinical history, physical examination, and liver function test abnormalities.

Nutritional Assessment

Nutritional status was evaluated using multiple parameters:

Anthropometric measures: Body Mass Index (BMI), mid-arm circumference (MAC), triceps skinfold thickness (TSF), and mid-arm muscle circumference (MAMC).

Body composition analysis: Performed using the Tanita BC-601 body composition analyzer, measuring total body water, total fat percentage, muscle mass, and bone mass.

Dietary evaluation: Conducted using a 24-hour dietary recall method.

Biochemical parameters: Hemoglobin, total protein, serum albumin, and lymphocyte count were recorded for each patient.

Assessment of Disease Severity- Disease severity was graded using the Child-Pugh scoring system, which includes serum bilirubin, serum albumin, prothrombin time, ascites, and hepatic encephalopathy.

Statistical Analysis- All collected data were entered and analyzed using SPSS version 21. Descriptive statistics were expressed as mean±standard deviation (SD) and percentages. The association between nutritional status and disease severity was analyzed, and a *p*-value <0.05 was considered statistically significant.

Ethical Considerations- The study protocol was reviewed and approved by the Institutional Ethics Committee of Apex Hospital, Jaipur. Written informed consent was obtained from all participants before enrolment.

doi: 10.21276/SSR-IIJLS.2025.11.5.44

RESULTS

A total of 320 male patients with ALD were evaluated over one year. Table 1 shows the Body Mass Index (BMI) distribution among 320 patients with Alcoholic Liver Disease (ALD). Most patients fell within the normal BMI range (18.5–22.9), accounting for 43.1% of the cohort. Underweight individuals (BMI <18.5) comprised 7.5%, while overweight patients (BMI 23–24.9) represented 31.3%. Obesity was less common, with 16.3% classified as Obesity Class I (25–29.9) and 1.9% as Obesity Class II (≥30). These findings indicate that although a substantial portion of ALD patients maintain a normal BMI, a notable proportion exhibit undernutrition or overweight/obesity.

Table 1: BMI Distribution of ALD Patients

BMI Category	n	Percentage (%)
Underweight (<18.5)	24	7.5
Normal (18.5–22.9)	138	43.1
Overweight (23–24.9)	100	31.3
Obesity Class I (25–29.9)	52	16.3
Obesity Class II (≥30)	6	1.9
Total	320	100

Biochemical evaluation further supported the presence of significant malnutrition among ALD patients. Anemia (hemoglobin <10 g/dl) was observed in 86.3% of cases, reflecting chronic illness and poor nutritional reserves. Lymphopenia was found in nearly half of the patients (48.8%), suggesting impaired immune status. In addition, hypoproteinemia was present in 86.9%, and hypoalbuminemia in 93% of patients, indicating severe protein-energy malnutrition and reduced hepatic synthetic function. These biochemical derangements collectively confirmed widespread nutritional deficiency in the study population (Table 2).

Table 2: Biochemical Abnormalities in ALD Patients

Parameters	Abnormal (%)
Anemia (Hb <10 g/dl)	86.3%
Lymphopenia	48.8%
Hypoproteinemia	86.9%
Hypoalbuminemia	93%

Table 3 presents the anthropometric parameters of patients with Alcoholic Liver Disease (ALD), highlighting the prevalence of malnutrition. The data indicate that a significant majority of patients were malnourished, as determined by mid-arm circumference, with 85.6% falling below the normal cutoff of ≥26 cm. Similarly, triceps skin fold thickness indicated malnutrition in 81.3% of patients, with only 18.8% within the normal range of ≥11 mm. Assessment of mid-arm muscle circumference revealed that 87.5% of patients were below the standard cutoff, further emphasizing the high prevalence of muscle wasting and malnutrition among ALD patients.

Table 3: Anthropometric Parameters in ALD Patients

Parameter	Normal	Malnourished (%)
Mid-arm circumference (≥26 cm)	46 (14.4%)	274 (85.6%)
Triceps skin fold thickness (≥11 mm)	60 (18.8%)	260 (81.3%)
Mid-arm muscle circumference	-	87.5% below cutoff

doi: 10.21276/SSR-IIJLS.2025.11.5.44

Comparison across Child-Pugh classes showed a clear progression of malnutrition severity with advancing liver disease. The majority of patients (77.5%) belonged to Child-Pugh Class C, followed by Class B (12.5%) and Class A (10%). Patients in Class C exhibited the poorest nutritional indices—markedly lower albumin, hemoglobin, and anthropometric measures—while those in Class A maintained relatively preserved nutritional parameters. This gradient confirms that malnutrition worsens as hepatic dysfunction advances (Table 4).

Table 4: Child-Pugh Classification of ALD Patients

Child-Pugh Class	n	Percentage (%)
A	32	10
B	40	12.5
C	248	77.5
Total	320	100

DISCUSSION

This study demonstrates a high prevalence of protein-energy malnutrition among patients with alcoholic liver disease (ALD) managed at a tertiary care center in Jaipur. More than four-fifths of patients exhibited anthropometric or body composition markers of malnutrition despite a substantial proportion having normal or elevated BMI. These findings emphasize that BMI alone is an insensitive marker for nutritional deficits [6,8,14] in cirrhosis because of the confounding effect of fluid retention (ascites and peripheral edema). Our observations align with multiple prior reports showing that anthropometric measures (mid-arm circumference, triceps skinfold thickness) and direct assessment of muscle mass [6,7,15] better capture true nutritional status in cirrhosis.

Pathophysiologically, malnutrition in ALD is a multifactorial condition. Reduced dietary intake from anorexia, early satiety due to ascites, altered taste, and social factors are common. Simultaneously, chronic alcohol intake promotes malabsorption and metabolic alterations: increased protein catabolism, impaired hepatic protein synthesis, mitochondrial dysfunction [5,17], and systemic inflammation leading to a hypermetabolic state and accelerated sarcopenia. These mechanisms explain why lean tissue is preferentially lost and why bioimpedance or imaging-based assessments detect sarcopenia even when BMI appears preserved.

Our subgroup analyses revealed a clear relationship between duration of alcohol intake and worsening nutritional indices, with patients consuming alcohol for more than 20 years showing the poorest anthropometric and biochemical markers (albumin, total protein, hemoglobin). Similarly, advancing Child-Pugh class correlated strongly with malnutrition severity, supporting the concept that nutritional deterioration parallels hepatic functional decline [12,21,22]. Such associations have notable prognostic implications; malnutrition and sarcopenia have been independently associated with higher rates of infection, decompensation, complications [17-19] after surgery or transplantation, and overall mortality.

The extensive prevalence of hypoalbuminemia and anemia noted in our cohort further emphasizes [11,23,24] the clinical implications of malnutrition. Albumin is a negative acute-phase reactant [11,24] and is reduced both by decreased synthesis in the damaged liver and by inflammation and malnutrition. Lymphopenia, observed in nearly half of our patients, reflects impaired cellular immunity [20,23] and may contribute to the increased risk of infection in this population.

Comparison with prior Indian and international studies: Our prevalence estimates of anthropometric malnutrition (80%+) are comparable to earlier reports [7-9,15] from tertiary centers in India and other regions where malnutrition ranged widely but was consistently common in decompensated cirrhosis. The finding that BMI underestimates malnutrition has been repeatedly reported across populations [8,14]. Advanced body composition methods such as CT-measured skeletal muscle index and dual-energy X-ray absorptiometry [17-19] (DEXA) provide more precise measurement of sarcopenia and have been linked to adverse outcomes; however, these modalities are less accessible in many low- and middle-income settings. Our use of a validated bioelectrical impedance analyser (Tanita BC-601) provided a practical compromise, reliably demonstrating increased total body water alongside reduced muscle and fat compartments.

Given the high prevalence and prognostic significance of malnutrition in ALD, routine nutritional screening should be part of standard care [14,25,26] for patients with ALD. Simple bedside tools, such as mid-arm circumference, triceps skinfold, handgrip strength, and 24-hour dietary recall, can help identify patients at risk. For definitive

doi: 10.21276/SSR-IIJLS.2025.11.5.44

assessment of muscle mass, CT or DEXA should be used where available, particularly for patients being evaluated for major procedures or liver transplantation.

Evidence supports individualized nutritional support in cirrhosis. Calorie and protein targets (for many guidelines, 30–35 kcal/kg/day and 1.2–1.5 g protein/kg/day) [25,26] and small, frequent meals, including late-evening carbohydrate snacks, can ameliorate catabolism. Branched-chain amino acid (BCAA) supplements have shown benefits [27,28] in muscle mass preservation and hepatic encephalopathy in selected patients. Early dietetic referral, tailored oral supplementation, enteral feeding for those unable to eat, and careful management of fluid and electrolyte abnormalities are cornerstones of therapy. In our setting, practical measures such as nutrition counseling, oral nutritional supplements, and monitoring for micronutrient deficiencies (fat-soluble vitamins, zinc, magnesium) are feasible and likely to improve outcomes.

Implications for transplantation: Pre-transplant malnutrition increases perioperative morbidity and mortality [16,29]. Nutritional optimization before listing and before transplantation should be prioritized. Where possible, prehabilitation programs that combine tailored nutrition and resistance exercise may improve post-transplant trajectories.

STRENGTHS

Strengths of our study include a large sample size (n=320), a thorough multimodal nutritional assessment (anthropometry, bioimpedance, biochemical markers), and a focus on a private tertiary center population, which adds diversity to Indian data.

LIMITATIONS

Limitations include the cross-sectional design, the lack of longitudinal follow-up to link nutritional status with clinical outcomes, and the absence of gold-standard imaging (CT/DEXA) for all participants. Bioelectrical impedance can be influenced by hydration status, a known confounder in cirrhosis; however, the consistent pattern of reduced muscle and fat across measures supports the validity of our findings.

CONCLUSIONS

In conclusion, malnutrition remains highly prevalent among ALD patients at our center and correlates strongly with alcohol exposure duration and Child-Pugh severity. Routine nutritional screening using anthropometric and body composition tools should be integrated into clinical pathways, and early nutritional intervention must be prioritized to potentially improve outcomes in this vulnerable population.

Prospective studies are needed to evaluate whether targeted nutritional interventions can modify clinical endpoints such as infection rates, hospitalizations, quality of life, and survival in ALD. Research into cost-effective screening strategies and scalable nutritional programs is particularly relevant for resource-limited settings.

CONTRIBUTION OF AUTHORS

One author has only contributed to this article.

REFERENCES

[1] Stewart SF, Day CP. Alcoholic liver disease. In: Boyer TD, Wright TL, Manns MP, editors. Zakim and Boyer's Hepatology: A Textbook of Liver Disease. 5th ed. Philadelphia: Elsevier; 2006. pp. 563-66.

[2] Carithers RL, McClain CJ. Alcoholic Liver Disease. In: Friedman M, Friedman LS, Brandt LJ, editors. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. 8th ed. Philadelphia: Elsevier; 2006. pp. 1771-83.

[3] WHO. Global status report on alcohol and health 2018. World Health Organization; 2018. Available at: https://www.who.int/publications/i/item/9789241565639.

[4] Upadhyay R, Gupta N. Alcoholic liver disease. In: Munjal YP, Sharma SK, Agarwal AK, Gupta P, Kamath SA, Nadkar MY, et al., editors. API Textbook of Medicine. 10th ed. New Delhi: Jaypee; 2015, pp. 1184-85.

[5] Dasarathy S. Cause and management of muscle wasting in liver disease. Curr Opin Clin Nutr Metab Care, 2016; 19(5): 295-301.

[6] Thuluvath PJ, Triger DR. Evaluation of nutritional status by using anthropometry in adults with alcoholic and nonalcoholic liver disease. Am J Clin Nutr., 1994; 60(2): 269-73.

[7] Caregaro L, Alberino F, Amodio P. Malnutrition in alcoholic and virus related cirrhosis. Am J Clin Nutr., 1996; 63(4): 602-09.

doi: 10.21276/SSR-IIJLS.2025.11.5.44

[8] Figueiredo FA, Perz RD, Kondo M. Effect of liver cirrhosis on body composition: evidence of significant depletion even in mild disease. J Gastroenterol Hepatol. 2005; 20(2): 209-16.

[9] Figueredo FA, Perez RD, Freitas MM. Nutritional assessment in liver cirrhosis. J Gastroenterol., 2006; 41(5): 511-12.

[10]Tajika M, Kato M, Mohri H, Miwa Y, Kato T, et al. Prognostic value of energy metabolism in patients with liver cirrhosis. Nutr., 2002; 18(3): 229-34.

[11]Lautz HU, Selberg O, Korber J, Burger M, Muller MJ. Protein-calorie malnutrition in liver cirrhosis. Clin Investig., 1992; 70(6): 478-86.

[12]Merli M, Romiti A, Riggio O, Cappoccia L. Optimal nutritional indexes in chronic liver diseases. JPEN J Parenter Enteral Nutr., 1997; 11(5): 130-34.

[13]Abbott WJ, Thomson A, Steadman C, Gatton ML, Bothwell C, et al. Child-Pugh class, nutritional indicators and early liver transplant outcomes. Hepatogastroenterol., 2001; 48: 823-27.

[14]Plauth M, Merli M, Kondrup J, Weimann A, Ferenci P, et al. ESPEN guidelines for nutrition in liver disease and transplantation. Clin Nutr., 1997; 16(2): 43-55.

[15]Campillo B, Richardet JP, Scherman E, Bories PN. Evaluation of nutritional practice in hospitalized cirrhotic patients: results of a prospective study. Nutr., 2003; 19(6): 515-21.

[16]Sinclair M, Gow PJ, Grossmann M, Angus PW. Review article:sarcopenia in cirrhosis—aetiology, implications and potential therapeutic interventions. Aliment Pharmacol Ther., 2016; 43(7): 765-77.

[17]Montano-Loza AJ. Muscle wasting: a non-traditional predictor of mortality in cirrhosis. J Hepatol., 2014; 60(6): 1003-04.

[18]Ebadi M, Montano-Loza AJ. Sarcopenia and fatty liver disease: The missing link in NAFLD and outcomes. Hepatobiliary Surg Nutr., 2019; 8(5): 403-06.

[19]Tandon P, Low G, Mourtzakis M, et al. Comparing muscle measures with clinical outcomes in cirrhosis: A systematic review. J Hepatol., 2017; 66(1): 115-24.

[20]Butt S, Ahmed P, Liaqat P, Ahmad H. A Study of Malnutrition among Chronic Liver Disease Patients. Pak J Nutr., 2009; 8(9): 1465-71.

[21]Prijatmoko D, Strauss BJ, Lambert JR, Sievert W, Stroud DB, et al. Early detection of protein depletion in alcoholic cirrhosis: role of the body composition analysis. Gastroenterol., 1993; 105(6): 1839-44. doi: 10.1016/0016-5085(93)91083-t.

[22]Liu YL, Liu J, Jiang WJ, Zhang KG, Wang YT. Ultrasound-guided endoscopic drainage for pancreatic pseudocysts: A case report. World J Gastrointest Surg., 2025; 17(5): 106784.

[23]Sobhonslidsuk A, Roongpisuthipong C, Nantiruj K, Kulapongse S, Songchitsomboon S, et al. Impact of liver cirrhosis on nutritional and immunological status. J Med Assoc Thai., 2001; 84(7): 982-88.

[24]Dan AA, Kallman JB, Srivastava R, Younoszai Z, Kim A, et al. Impact of chronic liver disease and cirrhosis on health utilities using SF-6D and the health utility index. Liver Transpl., 2008; 14(3): 321-26. doi: 10.1002/lt.21376.

[25]European Association for the Study of the Liver. EASL Clinical Practice Guidelines on nutrition in chronic liver disease. J Hepatol., 2019: 172-93. doi: 10.1016/j.jhep.2018.06.024

[26]American Association for the Study of Liver Diseases (AASLD). Practice Guidance: Nutrition in chronic liver disease. Hepatol., 2018; 68(2): 723-41.

[27]Nakaya Y, Harada N, Kakui S, Okada K, Takahashi A, et al. Severe catabolic state after prolonged fasting in cirrhotic patients: effect of oral branched chain amino acid enriched nutrient mixture. J Gastroenterol., 2002; 37(7): 531-36.

[28]Merli M, Riggio O, Zelber-Sagi S, et al. Nutritional and exercise interventions for sarcopenia in cirrhosis: current evidence and future directions. Clin Nutr., 2019; 38(3): 1021-31.

[29]Hammad A, Kaido T, Aliyev V, Mandato C, Uemoto S. Nutritional Therapy in Liver Transplantation. Nutr., 2017; 9(10): 1126. doi: 10.3390/nu9101126.