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Original Research Article

ASSOCIATION OF SERUM ALBUMIN LEVELS WITH DISEASE SEVERITY AMONG PATIENTS WITH CHRONIC LIVER DISEASE: A CROSS-SECTIONAL OBSERVATIONAL STUDY

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ABSTRACT

Background: Chronic liver disease (CLD) is associated with progressive hepatic dysfunction and various systemic complications. Serum albumin is an important biochemical indicator of hepatic synthetic function and may reflect the severity of liver disease. Assessment of its association with clinical manifestations and established severity scores may facilitate the identification of patients with advanced hepatic dysfunction. The aim is to evaluate the association between serum albumin levels and disease severity among patients with chronic liver disease.

Materials and Methods: The present hospital-based, cross-sectional observational study included 104 patients diagnosed with chronic liver disease at a tertiary care hospital. Demographic characteristics, clinical manifestations, serum albumin levels, biochemical parameters, Child–Pugh classification, and MELD-Na scores were evaluated. Statistical analysis was performed using SPSS version 27.0, and a p-value <0.05 was considered statistically significant.

Results: The majority of patients were males (66.35%), and alcohol-associated liver disease was the most common etiology (36.54%). The mean serum albumin concentration was 2.93 ± 0.62 g/dL, with hypoalbuminemia observed in 82.69% of patients. Moderate and severe hypoalbuminemia were present in 30.77% and 26.92% of patients, respectively. Among patients with severe hypoalbuminemia, 85.71% belonged to Child–Pugh class C (p<0.001). Hepatic decompensation, ascites, encephalopathy, jaundice, and peripheral edema were significantly associated with albumin categories. Mean MELD-Na scores increased from 11.09 ± 2.66 in patients with normal albumin levels to 26.37 ± 3.64 in those with severe hypoalbuminemia (p<0.001).

Conclusion: Reduced serum albumin levels were significantly associated with advanced liver disease, higher MELD-Na scores, and increased clinical complications. Serum albumin may serve as a simple, accessible biochemical marker for evaluating disease severity when interpreted alongside clinical findings and established scoring systems.

Keywords: Chronic liver disease, Serum albumin, Hypoalbuminemia, Child–Pugh classification, MELD-Na score.

INTRODUCTION

Chronic liver disease (CLD) is a progressive disorder characterized by persistent hepatic injury, inflammation, fibrosis, and gradual deterioration of liver structure and function. Its major etiological factors include harmful alcohol consumption, chronic hepatitis B and C infections, metabolic dysfunction-associated steatotic liver disease, autoimmune disorders, and inherited metabolic conditions. Progression to cirrhosis produces widespread architectural distortion, regenerative nodules, and altered hepatic blood flow. Clinical presentation varies from asymptomatic biochemical abnormalities

to advanced hepatic insufficiency with multiple systemic complications. Early recognition of disease severity is essential for guiding clinical evaluation, treatment planning, monitoring, and timely referral for specialized care. These changes may evolve gradually, making recognition of deteriorating hepatic reserve an important clinical priority.[1] The course of chronic liver disease is closely associated with progressive portal hypertension and declining functional hepatic reserve. Patients with compensated cirrhosis may remain relatively stable, whereas decompensation is characterized by complications such as ascites, variceal hemorrhage, and hepatic encephalopathy. These events reflect interactions between increased intrahepatic vascular resistance, splanchnic vasodilatation, neurohormonal activation, and disturbed systemic circulation. Clinical examination alone may not adequately represent the degree of hepatic dysfunction, particularly during earlier stages. Consequently, accessible biochemical indicators and validated severity scoring systems are important components of comprehensive disease assessment.[2] Serum albumin is a major plasma protein synthesized predominantly by hepatocytes and serves as an important indicator of hepatic synthetic capacity. It maintains colloid oncotic pressure, contributes to intravascular fluid balance, and transports numerous endogenous and exogenous substances, including bilirubin, hormones, fatty acids, and medications. Albumin also participates in antioxidant defense, immune modulation, and preservation of vascular endothelial function. In chronic liver disease, impaired hepatocellular synthesis may reduce circulating albumin concentrations. Systemic inflammation, altered vascular permeability, increased distribution into extravascular compartments, and nutritional deficiencies may further contribute to hypoalbuminemia, making its interpretation clinically relevant but multifactorial.[3] Beyond its circulating concentration, the biological activity of albumin deserves consideration in advanced liver disease. Oxidative stress, inflammation, and other metabolic disturbances can alter the structural integrity of the albumin molecule and compromise its transport, detoxification, and antioxidant functions. Consequently, a laboratory measurement of total serum albumin does not necessarily describe the entire functional albumin pool. Nevertheless, routine albumin testing is inexpensive, widely available, and readily repeated during hospital evaluation. Its potential relationship with clinical decompensation and established severity indices therefore remains especially relevant in settings where specialized functional albumin assays are unavailable.[4] Assessment of chronic liver disease severity requires integration of clinical manifestations and biochemical parameters rather than reliance on any isolated investigation. Serum bilirubin reflects aspects of hepatic excretory function, while prothrombin time and international normalized ratio provide information about coagulation-related synthetic function. Renal impairment and hyponatremia may accompany advanced portal hypertension and circulatory dysfunction. The Child–Turcotte–Pugh classification combines albumin, bilirubin, coagulation status, ascites, and encephalopathy, whereas MELD-Na incorporates bilirubin, creatinine, INR, and sodium. Importantly, albumin forms part of the Child–Pugh score but not MELD-Na; this distinction is necessary when interpreting associations between albumin and disease severity.[5] Hypoalbuminemia may coexist with clinically important complications such as ascites, peripheral edema, kidney dysfunction, and infection-related deterioration. Reduced hepatic albumin synthesis and changes in vascular physiology can jointly influence fluid distribution; however, ascites develops through mechanisms extending beyond low oncotic pressure alone. Albumin measurements must therefore be assessed alongside physical findings, renal function, electrolyte concentrations, and evidence of portal hypertension. A comprehensive approach can help differentiate an isolated biochemical abnormality from a wider pattern of hepatic decompensation and may assist clinicians in identifying patients who require more intensive evaluation and coordinated management. Assessment at a single visit is nevertheless unable to establish temporal or causal relationships.[6]

MATERIALS AND METHODS

The present study was designed as a hospital-based, cross-sectional observational study conducted at a tertiary care hospital to evaluate the association between serum albumin levels and disease severity among patients with chronic liver disease (CLD). The study assessed serum albumin concentrations in relation to clinical manifestations, biochemical abnormalities, hepatic functional impairment, and established disease severity scoring systems. A total of 104 patients diagnosed with chronic liver disease were included in the study. Patients were enrolled through consecutive sampling according to predefined eligibility criteria. The study included adult patients aged 18 years and above with an established diagnosis of chronic liver disease based on clinical history, biochemical investigations, and radiological findings. The diagnosis was supported by evidence of persistent hepatic dysfunction, chronic viral hepatitis, alcohol-associated liver disease, metabolic dysfunction-associated steatotic liver disease, autoimmune liver disease, or other recognized chronic hepatic disorders. Patients with compensated and decompensated chronic liver disease were considered eligible to assess variations in serum albumin levels across different stages of disease severity.

Inclusion Criteria

Patients aged 18 years and above with clinically, biochemically, or radiologically confirmed chronic

liver disease were included. Patients with available serum albumin measurements and relevant liver function parameters were considered eligible. Participants who provided written informed consent and underwent the required clinical and laboratory assessments were included in the study.

Exclusion Criteria

Patients with acute liver failure, acute-on-chronic liver failure, active sepsis, nephrotic syndrome, severe protein-losing enteropathy, or other conditions independently affecting serum albumin concentrations were excluded. Patients receiving recent intravenous albumin supplementation, those with advanced malignancy, and patients with incomplete essential clinical or laboratory information were also excluded.

Methodology: Detailed demographic and clinical information was recorded using a structured study proforma. Parameters included age, gender, body mass index (BMI), residence, etiology of chronic liver disease, history of alcohol consumption, smoking status, diabetes mellitus, hypertension, and associated comorbidities. Clinical evaluation included assessment of jaundice, pallor, peripheral edema, ascites, splenomegaly, gastrointestinal bleeding, and hepatic encephalopathy. Previous episodes of hepatic decompensation, hospital admissions, and relevant treatment history were documented. Patients were categorized as having compensated or decompensated cirrhosis based on the presence of ascites, hepatic encephalopathy, or variceal hemorrhage.

Laboratory Investigations: Venous blood samples were collected under standard aseptic precautions for biochemical and hematological investigations. Serum albumin concentration was measured in grams per deciliter (g/dL) using an automated biochemical analyzer. Additional investigations included total serum bilirubin, direct bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), total serum protein, serum globulin, prothrombin time (PT), international normalized ratio (INR), serum creatinine, serum sodium, hemoglobin concentration, and platelet count. Relevant hepatitis B and hepatitis C serological markers were recorded where available. Serum albumin levels were categorized as normal (≥3.5 g/dL), mildly reduced (3.0–3.49 g/dL), moderately reduced (2.5–2.99 g/dL), and severely reduced (0.05** was considered statistically significant.

Statistical Analysis: The collected data were entered into Microsoft Excel and analyzed using SPSS version 27.0. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The Chi-square test, independent t-test, and one-way ANOVA were applied as appropriate, while Pearson's or Spearman's correlation was used to assess the association between serum albumin levels and disease severity scores. A p-value <0.05 was considered statistically significant.

RESULTS

[Table 1] Distribution of Patients According to Demographic Characteristics, Etiology, and Comorbidities (N=104)

[Table 1] shows the demographic characteristics, etiological distribution, and associated comorbidities among 104 patients with chronic liver disease. The majority of patients belonged to the 46–60 years age group (41.35%), followed by those aged above 60 years (25.00%), 31–45 years (24.04%), and 18–30 years (9.62%). Male patients constituted 66.35% of the study population, whereas females accounted for 33.65%. A higher proportion of patients belonged to rural areas (59.62%) compared with urban areas (40.38%). Alcohol-associated liver disease was the most common etiology (36.54%), followed by metabolic dysfunction-associated steatotic liver disease (27.88%), hepatitis B (16.35%), hepatitis C (11.54%), and other causes (7.69%). Diabetes mellitus was present in 29.81% of patients, while 70.19% were non-diabetic. Similarly, hypertension was observed in 25.96% of patients, whereas 74.04% had no history of hypertension.

[Table 2] Distribution of Patients According to Serum Albumin Levels (N=104)

[Table 2] demonstrates the distribution of serum albumin concentrations among patients with chronic liver disease. The mean serum albumin level was 2.93 ± 0.62 g/dL, indicating reduced albumin concentrations in the study population. Among the 104 patients, only 18 (17.31%) had normal serum albumin levels of ≥3.5 g/dL. Mild hypoalbuminemia, defined as serum albumin levels between 3.0 and 3.49 g/dL, was observed in 26 patients (25.00%). Moderate hypoalbuminemia, with serum albumin levels between 2.5 and 2.99 g/dL, was the most frequently observed category, affecting 32 patients (30.77%). Severe hypoalbuminemia, characterized by serum albumin levels below 2.5 g/dL, was identified in 28 patients (26.92%). Overall, 86 patients (82.69%) demonstrated hypoalbuminemia of varying severity, indicating a high prevalence of reduced serum albumin concentrations among the included patients.

[Table 3] Association Between Serum Albumin Levels and Child–Pugh Classification (N=104)

[Table 3] illustrates the association between serum albumin concentrations and disease severity according to the Child–Pugh classification. Among patients with normal serum albumin levels, 88.89% belonged to Child–Pugh class A, while 11.11% belonged to class B, with no patients classified as class C. In the mild hypoalbuminemia group, 30.77% were classified as Child–Pugh A and 69.23% as Child–Pugh B. Among patients with moderate hypoalbuminemia, 3.12% belonged to class A, 37.50% to class B, and 59.38% to class C. In contrast, among patients with severe hypoalbuminemia, 85.71% belonged to Child–Pugh class C and 14.29% to class B, with no patients classified as class A.

Overall, 25 patients (24.04%) belonged to class A, 36 (34.62%) to class B, and 43 (41.35%) to class C. The association between serum albumin levels and Child–Pugh classification was statistically significant ($p<0.001$), although this relationship was partly influenced by serum albumin being a component of the Child–Pugh scoring system.

[Table 4] Association of Serum Albumin Levels with Clinical Manifestations of Chronic Liver Disease (N=104)

[Table 4] presents the relationship between serum albumin categories and various clinical manifestations of chronic liver disease. Hepatic decompensation was observed in 11.11% of patients with normal albumin levels compared with 46.15%, 84.38%, and 82.14% of patients with mild, moderate, and severe hypoalbuminemia, respectively ($p<0.001$). Ascites was present in 11.11% of patients with normal albumin levels, increasing to 26.92% in mild, 71.88% in moderate, and 60.71% in severe hypoalbuminemia ($p<0.001$). Hepatic encephalopathy was absent in the normal and mild groups but was observed in 6.25% and 35.71% of patients with moderate and severe hypoalbuminemia, respectively ($p<0.001$). A history of variceal bleeding was reported in 19.23%, 34.38%, and 46.43% of patients with mild, moderate, and severe hypoalbuminemia, respectively, compared with none in the normal group ($p=0.004$). Jaundice increased from 11.11% in patients with normal albumin levels to 78.57% in those with severe hypoalbuminemia ($p<0.001$). Similarly, peripheral edema was observed in 22.22%, 23.08%, 59.38%, and 82.14% of patients across the four albumin categories, respectively ($p<0.001$), demonstrating significant associations between reduced serum albumin levels and several clinical manifestations of advanced liver disease.

[Table 5] Association of Serum Albumin Levels with Biochemical Parameters and MELD-Na Score (N=104)

[Table 5] demonstrates the relationship between serum albumin levels, biochemical abnormalities, and MELD-Na scores among patients with chronic liver disease. Mean total bilirubin concentrations increased progressively from 1.63 ± 0.87 mg/dL in patients with normal serum albumin levels to 4.64 ± 2.46 mg/dL in those with severe hypoalbuminemia ($p<0.001$). Similarly, the mean INR increased from 1.25 ± 0.14 in the normal albumin group to 2.06 ± 0.47 in the severe hypoalbuminemia group ($p<0.001$). Mean serum creatinine levels also increased across the albumin categories, from 0.93 ± 0.15 mg/dL to 1.47 ± 0.27 mg/dL ($p<0.001$). Conversely, mean serum sodium concentrations decreased from 137.57 ± 1.45 mEq/L in patients with normal albumin levels to 131.31 ± 3.48 mEq/L in patients with severe hypoalbuminemia ($p<0.001$). The mean MELD-Na score increased progressively from 11.09 ± 2.66 in the normal albumin group to 15.76 ± 2.67, 21.35 ± 4.05, and 26.37 ± 3.64 in the mild, moderate, and severe hypoalbuminemia groups, respectively ($p<0.001$). These findings demonstrated statistically significant differences in biochemical parameters and MELD-Na scores across serum albumin categories, with lower albumin concentrations corresponding to greater hepatic disease severity.

Table 1: Distribution of Patients According to Demographic Characteristics, Etiology, and Comorbidities (N=104)

Parameters	Categories	Number (%)
Age (years)	18–30	10 (9.62%)
	31–45	25 (24.04%)
	46–60	43 (41.35%)
	>60	26 (25.00%)
Gender	Male	69 (66.35%)
	Female	35 (33.65%)
Residence	Rural	62 (59.62%)
	Urban	42 (40.38%)
Etiology of CLD	Alcohol-associated liver disease	38 (36.54%)
	Metabolic dysfunction-associated steatotic liver disease	29 (27.88%)
	Hepatitis B	17 (16.35%)
	Hepatitis C	12 (11.54%)
	Other causes	8 (7.69%)
Diabetes mellitus	Present	31 (29.81%)
	Absent	73 (70.19%)
Hypertension	Present	27 (25.96%)
	Absent	77 (74.04%)

Table 2: Distribution of Patients According to Serum Albumin Levels (N=104)

Serum albumin category	Serum albumin (g/dL)	Number	Percentage (%)
Normal	≥3.5	18	17.31
Mild hypoalbuminemia	3.0–3.49	26	25.00
Moderate hypoalbuminemia	2.5–2.99	32	30.77
Severe hypoalbuminemia	Below 2.5	28	26.92
Total		104	100.00

Mean serum albumin: 2.93 ± 0.62 g/dL. Overall, 86 patients (82.69%) had hypoalbuminemia.

Table 3: Association Between Serum Albumin Levels and Child–Pugh Classification (N=104)

Serum albumin category	Child–Pugh A n (%)	Child–Pugh B n (%)	Child–Pugh C n (%)	Total
Normal (n=18)	16 (88.89)	2 (11.11)	0 (0.00)	18
Mild (n=26)	8 (30.77)	18 (69.23)	0 (0.00)	26
Moderate (n=32)	1 (3.12)	12 (37.50)	19 (59.38)	32
Severe (n=28)	0 (0.00)	4 (14.29)	24 (85.71)	28
Total	25 (24.04)	36 (34.62)	43 (41.35)	104

Overall p-value: p<0.001

Table 4: Association of Serum Albumin Levels with Clinical Manifestations of Chronic Liver Disease (N=104)

Clinical parameters	Normal (n=18) n (%)	Mild (n=26) n (%)	Moderate (n=32) n (%)	Severe (n=28) n (%)	p-value
Hepatic decompensation	2 (11.11)	12 (46.15)	27 (84.38)	23 (82.14)	<0.001
Ascites	2 (11.11)	7 (26.92)	23 (71.88)	17 (60.71)	<0.001
Hepatic encephalopathy	0 (0.00)	0 (0.00)	2 (6.25)	10 (35.71)	<0.001
History of variceal bleeding	0 (0.00)	5 (19.23)	11 (34.38)	13 (46.43)	0.004
Jaundice	2 (11.11)	7 (26.92)	18 (56.25)	22 (78.57)	<0.001
Peripheral edema	4 (22.22)	6 (23.08)	19 (59.38)	23 (82.14)	<0.001

Table 5: Association of Serum Albumin Levels with Biochemical Parameters and MELD-Na Score (N=104)

Parameters	Normal (n=18)	Mild (n=26)	Moderate (n=32)	Severe (n=28)	p-value
Total bilirubin (mg/dL)	1.63 ± 0.87	2.03 ± 1.04	3.22 ± 1.65	4.64 ± 2.46	<0.001
INR	1.25 ± 0.14	1.45 ± 0.20	1.81 ± 0.35	2.06 ± 0.47	<0.001
Serum creatinine (mg/dL)	0.93 ± 0.15	1.11 ± 0.23	1.24 ± 0.27	1.47 ± 0.27	<0.001
Serum sodium (mEq/L)	137.57 ± 1.45	135.35 ± 2.20	133.52 ± 3.07	131.31 ± 3.48	<0.001
MELD-Na score	11.09 ± 2.66	15.76 ± 2.67	21.35 ± 4.05	26.37 ± 3.64	<0.001

DISCUSSION

The present study included 104 patients with chronic liver disease, with the highest proportion belonging to the 46–60 years age group (41.35%), followed by patients aged above 60 years (25.00%). Male predominance was observed, with 66.35% males and 33.65% females. Alcohol-associated liver disease was the most common etiology (36.54%), followed by metabolic dysfunction-associated steatotic liver disease (27.88%), hepatitis B (16.35%), and hepatitis C (11.54%). Similar findings were reported by Rathi et al. (2019),[7] who conducted a multicentric Indian study involving 1,114 patients with liver cirrhosis. Their study reported a mean age of 49.52 years, with males constituting 80.90% of patients. Alcohol-associated liver disease was the leading etiology (43.27%), followed by viral hepatitis (21.45%). Compared with their findings, the present study demonstrated a lower proportion of male patients and alcohol-associated liver disease, although both studies identified alcohol consumption as an important contributor to chronic liver disease. These findings demonstrated similarities in the demographic and etiological profiles of cirrhosis patients across different Indian healthcare settings. The present study demonstrated that diabetes mellitus was present in 29.81% of patients, while hypertension was observed in 25.96%. Among the etiological factors, metabolic dysfunction-associated steatotic liver disease contributed 27.88% of cases, indicating the importance of metabolic abnormalities in patients with chronic liver disease. Maji et al,[8] (2024) conducted a prospective observational study involving 316 patients with liver cirrhosis and reported that 22.50% had hepatogenous diabetes, 26.30% had type 2 diabetes mellitus, and 51.30% were non-diabetic. Their study also demonstrated that 80.00% of patients were male, while alcohol-associated liver disease accounted for 29.70% of cases, followed by hepatitis B (26.60%) and non-alcoholic steatohepatitis (19.90%). Furthermore, 46.20% of their patients belonged to Child–Pugh class C, compared with 41.35% in the present study. Although the diabetes prevalence estimates were not directly comparable because Maji et al. separately identified hepatogenous diabetes through glucose tolerance testing, both studies demonstrated a substantial burden of metabolic abnormalities among patients with cirrhosis. The present study demonstrated that hypoalbuminemia was highly prevalent among patients with chronic liver disease, affecting 86 of 104 patients (82.69%), with a mean serum albumin concentration of 2.93 ± 0.62 g/dL. Moderate hypoalbuminemia was observed in 30.77% of patients, while severe hypoalbuminemia was present in 26.92%. A significant association was observed between serum albumin categories and Child–Pugh classification (p<0.001). Among patients with severe hypoalbuminemia, 85.71% belonged to Child–Pugh class C, compared with 88.89% of patients with normal albumin levels who belonged to class A. Janota et al,[9] (2023) evaluated 118 patients with liver cirrhosis and reported mean serum albumin concentrations of 4.181 g/dL, 3.281 g/dL, and 2.710 g/dL among Child–Pugh classes A, B, and C, respectively. They also observed reduced serum albumin concentrations in 7.60% of class A, 79.40% of class B, and 100.00% of class C patients. These findings were consistent with the pattern observed in the present study, demonstrating progressively lower serum albumin concentrations with increasing Child–Pugh severity. However, this relationship should be interpreted cautiously because serum albumin is included in the Child–Pugh scoring system.

The association between reduced serum albumin levels and advanced hepatic dysfunction was further demonstrated in the present study, where Child–Pugh class C accounted for 41.35% of patients. Among those with moderate hypoalbuminemia, 59.38% belonged to Child–Pugh class C, whereas this proportion increased to 85.71% among patients with severe hypoalbuminemia. Amouzandeh et al,[10] (2023) investigated albumin and fibrinogen synthesis rates in 28 patients with advanced chronic liver disease. Their study demonstrated that mean plasma albumin concentration was significantly lower in Child–Pugh class C patients than in class B patients (2.31 ± 0.38 g/dL versus 2.73 ± 0.43 g/dL; p=0.033). The median absolute albumin synthesis rate was also significantly reduced in class C compared with class B (44 versus 66 mg/kg/day; p=0.0255). Furthermore, albumin synthesis demonstrated a significant negative correlation with MELD score (r=−0.617; p=0.0005). These findings supported the association between worsening hepatic function and reduced albumin availability. The differences observed in the present study were consistent with impaired hepatic synthetic function in advanced cirrhosis, although serum albumin concentrations may also be affected by inflammation, nutritional status, and fluid distribution. The present study demonstrated significant associations between serum albumin levels and clinical manifestations of chronic liver disease. Hepatic decompensation was observed in 11.11% of patients with normal serum albumin levels compared with 82.14% of those with severe hypoalbuminemia (p<0.001). Ascites was present in 11.11%, 26.92%, 71.88%, and 60.71% of patients with normal, mild, moderate, and severe hypoalbuminemia, respectively (p<0.001). A history of variceal bleeding also increased from 0.00% in the normal albumin group to 46.43% in the severe hypoalbuminemia group (p=0.004). Li et al,[11] (2024) conducted a retrospective observational study involving 265 patients with cirrhosis and ascites in China. Their study reported a 90-day hospital readmission rate of 43.00%. Multivariable analysis demonstrated that lower serum albumin concentrations were independently associated with hospital readmission (OR=0.88; 95% CI: 0.83–0.93), while higher MELD-Na scores were associated with increased readmission risk (OR=1.04; 95% CI: 1.01–1.07). Although their study evaluated readmission rather than the prevalence of decompensation, the findings were compatible with the association between reduced albumin levels and greater clinical disease burden observed in the present study. The present study also demonstrated an association between reduced serum albumin levels and hepatic encephalopathy. Hepatic encephalopathy was absent among patients with normal serum albumin levels and mild hypoalbuminemia but was observed in 6.25% of patients with moderate hypoalbuminemia and 35.71% of those with severe hypoalbuminemia (p<0.001). Additionally, jaundice was present in 78.57% of patients with severe hypoalbuminemia compared with 11.11% of those with normal serum albumin levels. Peripheral edema was also substantially more frequent in patients with severe hypoalbuminemia (82.14%) than in those with normal albumin levels (22.22%). Bai et al,[12] (2019) investigated the association of serum albumin concentrations with the development and mortality of overt hepatic encephalopathy among hospitalized patients with cirrhosis. Among 2,376 patients without hepatic encephalopathy at admission, 113 (4.80%) subsequently developed overt hepatic encephalopathy. Lower serum albumin was independently associated with encephalopathy development (OR=0.878; 95% CI: 0.834–0.924), with an optimal predictive cutoff of ≤3.16 g/dL and an area under the ROC curve of 0.770. Among 183 patients admitted with overt hepatic encephalopathy, 20 (10.90%) died during hospitalization, and lower albumin was also associated with mortality. These findings were consistent with the greater frequency of encephalopathy in patients with lower serum albumin levels in the present study, although the outcome definitions and study populations differed. The present study demonstrated significant differences in biochemical parameters and MELD-Na scores across serum albumin categories. Mean serum bilirubin increased from 1.63 ± 0.87 mg/dL in patients with normal albumin concentrations to 4.64 ± 2.46 mg/dL in those with severe hypoalbuminemia (p<0.001). Similarly, INR increased from 1.25 ± 0.14 to 2.06 ± 0.47, while serum creatinine increased from 0.93 ± 0.15 mg/dL to 1.47 ± 0.27 mg/dL. Conversely, mean serum sodium decreased from 137.57 ± 1.45 mEq/L to 131.31 ± 3.48 mEq/L. The mean MELD-Na score increased substantially from 11.09 ± 2.66 to 26.37 ± 3.64 across the same groups (p<0.001). Nguyen et al,[13] (2025) conducted a prospective study involving 279 patients with acute decompensated cirrhosis and demonstrated similar biochemical patterns across increasing bilirubin-to-albumin ratio categories. Median albumin decreased from 3.22 g/dL in the lowest ratio group to 2.50 g/dL in the highest ratio group, while median INR increased from 1.28 to 1.96 and serum sodium decreased from 138 to 133 mEq/L. Median MELD-Na increased from 9.57 to 27.38 (p<0.001). Their study also reported a reduction in 180-day survival from 95.70% in the lowest ratio quartile to 56.50% in the highest. Although the group definitions and summary measures differed, these findings supported the clinical relevance of reduced albumin concentrations alongside worsening liver function parameters. The present study was cross-sectional and therefore could not establish mortality prediction or longitudinal outcomes.

CONCLUSION

The present study demonstrated a significant association between reduced serum albumin levels and increased disease severity among patients with

chronic liver disease. Hypoalbuminemia was associated with advanced Child–Pugh classification, higher MELD-Na scores, and increased frequency of hepatic complications, including ascites, hepatic encephalopathy, and peripheral edema. Serum albumin emerged as a simple, inexpensive, and readily available biochemical marker for assessing hepatic dysfunction. Its evaluation alongside clinical findings and established severity scoring systems may facilitate early identification of patients with advanced liver disease and support appropriate clinical management.

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