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Original Research Article

Association of Serum Sodium Levels with Severity, Complications, and Outcomes in Liver Cirrhosis

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Abstract

Background: Hyponatremia is a frequent electrolyte abnormality in liver cirrhosis and reflects advanced hepatic dysfunction, circulatory derangement, and poor prognosis. The present study aimed to evaluate the clinical significance of serum sodium levels in patients with liver cirrhosis and their correlation with disease severity, complications, and short-term outcomes.

Methods: This cross-sectional observational study included 108 patients with clinically, biochemically, and ultrasonographically confirmed liver cirrhosis admitted to a tertiary care teaching hospital. Clinical data, liver function parameters, and serum sodium levels were recorded. The severity of liver disease was assessed using Child–Turcotte–Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores. Patients were categorized into normal sodium (≥135 mmol/L), mild hyponatremia (130–134 mmol/L), and moderate-to-severe hyponatremia (<130 mmol/L) groups. Correlation and comparative analyses were performed using ANOVA, Chi-square, and Pearson's correlation tests, with $p < 0.05$ considered statistically significant.

Results: The mean age of patients was 51.8 ± 10.6 years, with 75.9% males and 57.4% alcoholic etiology. Hyponatremia was observed in 61.1% of cases. Mean serum sodium levels declined progressively from 136.2 ± 3.1 mmol/L (CTP A) to 128.8 ± 4.1 mmol/L (CTP C) ($p < 0.001$). Patients with severe hyponatremia had significantly higher CTP (10.4 ± 1.9) and MELD (24.6 ± 4.2) scores compared to those with normal sodium ($p < 0.001$). Hyponatremia was significantly associated with ascites ($p < 0.001$), hepatic encephalopathy ($p = 0.005$), spontaneous bacterial peritonitis ($p = 0.011$), and hepatorenal syndrome ($p = 0.007$). Serum sodium correlated negatively with CTP ($r = –0.592$) and MELD ($r = –0.645$) scores. The mean hospital stay increased from 6.8 to 11.2 days with decreasing sodium levels ($p < 0.001$), and mortality rose from 2.4% to 23.1% ($p = 0.008$).

Conclusion: Hyponatremia is highly prevalent in liver cirrhosis and serves as an independent marker of disease severity, complications, and short-term mortality. Routine assessment of serum sodium is a simple, cost-effective prognostic tool that can aid in risk stratification and timely management of cirrhotic patients.

Keywords: Liver cirrhosis; Hyponatremia; Serum sodium; Child–Turcotte–Pugh score; MELD score.

Introduction

Liver cirrhosis is the end stage of chronic liver disease, characterized by progressive fibrosis, architectural distortion, and regenerative nodule formation, leading to hepatic insufficiency and portal hypertension. Globally, cirrhosis accounts for a significant health burden, ranking as the 11th leading cause of death and the 15th leading cause of morbidity worldwide [1]. Cirrhosis contributes to over 1.3 million deaths annually, with viral hepatitis, alcohol-related liver disease, and non-alcoholic fatty liver disease being the predominant etiologies [2]. In India, the prevalence of cirrhosis is estimated at around 1% of the population, contributing to approximately 170,000 deaths each year [3].

As the disease progresses, complications such as ascites, hepatic encephalopathy, variceal bleeding, and hepatorenal syndrome mark the transition from compensated to decompensated cirrhosis, substantially worsening prognosis [2]. Accurate and easily measurable biomarkers that reflect disease severity are therefore critical for timely intervention and prognostication [3]. Among biochemical parameters, serum sodium concentration has emerged as an important indicator of circulatory and renal dysfunction in cirrhosis. Hyponatremia, commonly defined as serum sodium <135 mmol/L, is observed in 20–50% of hospitalized cirrhotic patients, particularly those with ascites and advanced disease [4]. It

predominantly results from impaired renal free water excretion secondary to marked splanchnic vasodilation, activation of the renin–angiotensin–aldosterone system (RAAS), elevated sympathetic tone, and non-osmotic secretion of arginine vasopressin (AVP). These mechanisms cause water retention and dilutional hyponatremia, reflecting the severity of the underlying hemodynamic derangement [5].

Literature shows that lower serum sodium levels correlate with increased disease severity and poor outcomes [6,7]. Hypo-natremia has been associated with higher Child–Turcotte–Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores, as well as increased risk of complications such as hepatic encephalopathy, spontaneous bacterial peritonitis, and hepatorenal syndrome. Importantly, incorporation of serum sodium into the MELD scoring system (MELD-Na) has significantly enhanced its predictive accuracy for short-term mortality and liver transplantation outcomes [7]. For instance, literature have shown that each 1 mmol/L decrease in serum sodium below 135 mmol/L increases mortality risk by nearly 5–10%, underscoring its prognostic relevance [8,9]. Despite these findings, in many clinical settings—especially in resource-limited regions—routine evaluation of serum sodium is often underutilized for disease staging or prognostication [10]. Given that it is an inexpensive and easily measurable biochemical parameter, its role in risk stratification and management of cirrhotic patients deserves greater emphasis. Hence, the present study aimed to assess the clinical and prognostic significance of serum sodium levels in patients with liver cirrhosis, and to analyze their correlation with disease severity and complications. Understanding this relationship could facilitate early identification of high-risk patients and guide more appropriate therapeutic interventions.

Materials and Methods

Study Design and Setting: The present study was designed as a hospital-based cross-sectional observational study conducted in the Department of Medicine, of a tertiary care teaching hospital located in North India. The study was carried out over a period of 2 years, from January 2023 to December 2024.

Study Population: A total of 108 adult patients diagnosed with liver cirrhosis of various etiologies were included in the study. The diagnosis of cirrhosis was based on a combination of clinical features, biochemical derangements, and radiological findings consistent with chronic liver disease. Patients presenting with ascites, jaundice, splenomegaly, palmar erythema, spider angiomas, or features suggestive of portal hypertension were screened. Ultrasonographic evidence of a coarse hepatic echotexture with surface nodularity, altered hepatic architecture, and presence of ascites was considered confirmatory.

Inclusion and Exclusion Criteria: Patients aged 18 years and above with a confirmed diagnosis of liver cirrhosis, either compensated or decompensated, were eligible for inclusion.

Exclusion criteria were strictly applied to eliminate confounding factors. Patients with acute liver failure, chronic kidney disease (serum creatinine >1.5 mg/dL), congestive cardiac failure, or sepsis were excluded.

Individuals who had received diuretic therapy or vasopressin analogues within 48 hours prior to sample collection were also excluded, as these could influence serum sodium levels. Pregnant and lactating women, as well as those unwilling to provide consent, were not included in the study.

Data Collection and Clinical Assessment: Upon admission, a detailed clinical history was obtained, including age, sex, duration of illness, probable etiology (alcoholic, viral, or cryptogenic), and presence of prior complications such as ascites, hepatic encephalopathy, or variceal bleeding.

A comprehensive physical examination was conducted, with emphasis on signs of chronic liver disease and its sequelae—such as jaundice, pedal edema, ascites, flapping tremors, and hepatosplenomegaly. Each patient underwent systematic assessment for clinical staging as either compensated or decompensated cirrhosis based on the presence of complications.

Laboratory Investigations: Venous blood samples were collected under aseptic precautions after an overnight fast. Serum sodium concentration was determined immediately using an ion-selective electrode method on a fully automated electrolyte analyzer. Other routine biochemical investigations included liver function tests (serum bilirubin, alanine aminotransferase [ALT], aspartate aminotransferase [AST], serum albumin, and alkaline phosphatase), renal function tests (blood urea and serum creatinine), and prothrombin time with INR. Complete blood count and viral markers for hepatitis B surface antigen (HBsAg) and anti-HCV antibody were also assessed.

Abdominal ultrasonography was performed in all cases to evaluate liver morphology, splenic size, ascites, and portal venous system. Serum sodium values were categorized as follows: Normal sodium: ≥135 mmol/L; Mild hyponatremia: 130–134 mmol/L; and Moderate to severe hyponatremia: <130 mmol/L

Assessment of Disease Severity: The Child–Turcotte–Pugh (CTP) score was calculated for each patient using five clinical and laboratory

parameters—serum bilirubin, serum albumin, prothrombin time (INR), ascites, and hepatic encephalopathy. Based on the total score, patients were classified as Class A (5–6 points), Class B (7–9 points), or Class C (10–15 points).

Additionally, disease severity was quantified using the Model for End-Stage Liver Disease (MELD) scoring system, calculated by the standard formula:

MELD=3.78 × ln (serum bilirubin) +11.2 × ln (INR) + 9.57 × ln (serum creatinine) + 6.43

For improved prognostic accuracy, serum sodium was incorporated into the modified MELD-Na formula. The correlation between serum sodium levels and both the CTP and MELD scores was analyzed to assess the relationship between hyponatremia and the severity of liver dysfunction.

Statistical Analysis: All collected data were entered into a Microsoft Excel spreadsheet and analyzed using Statistical Package for the Social Sciences (SPSS) version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), while categorical data were represented as frequency and percentage.

The comparison of mean serum sodium levels among different Child-Pugh classes and MELD categories was carried out using one-way analysis of variance (ANOVA). The Chi-square test was used for comparing categorical variables.

The Pearson's correlation coefficient (r) was applied to determine the relationship between serum sodium levels and severity indices (CTP and MELD scores). A p-value less than 0.05 was considered statistically significant for all analyses.

Ethical Considerations: Prior to commencement, the study protocol was reviewed and approved by the Institutional Ethics Committee. Written informed consent was obtained from each participant after explaining the study objectives, procedures, and potential risks. All data were anonymized to maintain confidentiality and used solely for academic research purposes.

Results

The mean age of the study population was 51.8 ± 10.6 years, with a predominance of male patients (75.9%), reflecting the higher prevalence of alcohol-related liver disease among men in India. The mean duration of liver disease was 29.4 ± 15.2 months. The commonest etiology was alcoholic liver disease (57.4%), followed by hepatitis B (16.7%), hepatitis C (9.3%), non-alcoholic steatohepatitis (8.3%), and cryptogenic/other causes (8.3%).

Among the cases, decompensated cirrhosis (64.8%) was more frequent than compensated disease (35.2%). The most prevalent clinical manifestations were ascites (70.4%), jaundice (57.4%), pedal edema (61.1%), splenomegaly (43.5%), and hepatic encephalopathy (18.5%), indicating that the majority of patients presented with advanced disease and portal hypertension-related complications (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (n=108)

Variables	Frequency (%)/Mean ± SD
Age (years)	51.8 ± 10.6
Gender	
Male	82 (75.9%)
Female	26 (24.1%)
Duration of liver disease (months)	29.4 ± 15.2
Etiology of cirrhosis	
Alcoholic	62 (57.4%)
Hepatitis B	18 (16.7%)
Hepatitis C	20 (9.3%)
Non-alcoholic steatohepatitis (NASH)	9 (8.3%)
Cryptogenic/others	9 (8.3%)
Type of cirrhosis	
Compensated	38 (35.2%)
Decompensated	70 (64.8%)
Major clinical features	
Ascites	76 (70.4%)
Jaundice	62 (57.4%)
Splenomegaly	47 (43.5%)
Hepatic encephalopathy	20 (18.5%)
Pedal edema	66 (61.1%)

SD – Standard Deviation.

The mean hemoglobin level was 10.6 ± 1.8 g/dL, indicating mild anemia as a common accompaniment in cirrhosis. The mean total bilirubin and serum albumin levels were 3.8 ± 1.6 mg/dL and 2.6 ± 0.4 g/dL, respectively, signifying impaired hepatic synthetic and excretory functions. The mean AST and ALT were 86.2 ± 34.8 U/L and 72.4 ± 29.5 U/L, respectively, while alkaline phosphatase averaged 172.8 ± 54.3 U/L. Coagulopathy was evident, with a prolonged prothrombin time (4.6 ± 2.3 seconds) and mean INR of 1.74 ± 0.48. The mean serum creatinine was 1.21 ± 0.43 mg/dL, reflecting preserved renal function in most patients. The mean serum sodium level across the cohort was 132.4 ± 5.9 mmol/L, suggesting that hyponatremia was a frequent finding in cirrhosis (Table 2).

Table 2: Laboratory Profile of Patients with Liver Cirrhosis (n=108)

Parameter	Mean ± SD
Hemoglobin (g/dL)	10.6 ± 1.8
Total bilirubin (mg/dL)	3.8 ± 1.6
Serum albumin (g/dL)	2.6 ± 0.4
AST (U/L)	86.2 ± 34.8
ALT (U/L)	72.4 ± 29.5
Alkaline phosphatase (U/L)	172.8 ± 54.3
Prothrombin time prolongation (sec)	4.6 ± 2.3
INR	1.74 ± 0.48
Serum creatinine (mg/dL)	1.21 ± 0.43
Serum sodium (mmol/L)	132.4 ± 5.9

AST – Aspartate Aminotransferase; ALT – Alanine Aminotransferase; INR – International Normalized Ratio.

Of the 108 patients, 42 (38.9%) had normal serum sodium levels (≥135 mmol/L), 40 (37.0%) had mild hyponatremia (130–134 mmol/L), and 26 (24.1%) had moderate-to-severe hyponatremia (<130 mmol/L). Both CTP and MELD scores showed a significant inverse association with serum sodium levels ($p < 0.001$ for both comparisons). The mean CTP scores rose progressively from 6.9 ± 1.3 in the normonatremic group to 10.4 ± 1.9 in patients with severe hyponatremia. Similarly, the mean MELD scores increased from 12.8 ± 3.2 to 24.6 ± 4.2, underscoring that worsening hyponatremia correlated with advanced hepatic dysfunction (Table 3).

Table 3: Comparison of Serum Sodium Categories with Mean Child–Turcotte–Pugh (CTP) and MELD Scores

Serum Sodium Category	Frequency (%)	CTP Score	MELD Score
		Mean ± SD	
Normal (≥135 mmol/L)	42 (38.9%)	6.9 ± 1.3	12.8 ± 3.2
Mild hyponatremia (130–134 mmol/L)	40 (37.0%)	8.6 ± 1.7	18.2 ± 3.8
Moderate–severe hyponatremia (<130 mmol/L)	26 (24.1%)	10.4 ± 1.9	24.6 ± 4.2
p-value (ANOVA)	—	<0.001	<0.001

CTP – Child–Turcotte–Pugh; MELD – Model for End-Stage Liver Disease; ANOVA – Analysis of Variance.

A statistically significant decline in serum sodium concentration was observed with increasing CTP class ($p < 0.001$). Patients in Class A had a mean sodium level of 136.2 ± 3.1 mmol/L, while those in Class B and Class C demonstrated progressively lower means of 132.1 ± 4.5 mmol/L and 128.8 ± 4.1 mmol/L, respectively. This trend indicates that hyponatremia worsens with advancing liver failure, reflecting its pathophysiologic link to impaired free water excretion and portal hypertension-related circulatory dysfunction (Table 4).

Table 4: Relationship Between Child–Turcotte–Pugh (CTP) Class and Serum Sodium Levels

CTP Class	Frequency (%)	Serum Sodium (mmol/L)
		Mean ± SD
Class A (5–6)	26 (24.1%)	136.2 ± 3.1
Class B (7–9)	42 (38.9%)	132.1 ± 4.5
Class C (10–15)	40 (37.0%)	128.8 ± 4.1
p-value (ANOVA)	—	<0.001

CTP – Child–Turcotte–Pugh; SD – Standard Deviation; ANOVA – Analysis of Variance.

The frequency of cirrhosis-related complications showed a graded increase with severity of hyponatremia. The prevalence of ascites rose from 57.1% in normonatremic patients to 96.2% in those with severe hyponatremia ($p < 0.001$). Similarly, hepatic encephalopathy was more common in the severe hyponatremia group (38.5%) compared to the normal sodium group (7.1%; $p = 0.005$). SBP ($p = 0.011$) and hepatorenal syndrome ($p = 0.007$) were also significantly more frequent among those with sodium <130 mmol/L. The occurrence of upper gastrointestinal variceal bleeding was higher in hyponatremic patients (up to 34.6%) but did not reach statistical significance ($p = 0.112$). These findings emphasize the prognostic value of serum sodium in predicting complications in cirrhosis (Table 5).

Table 5: Association Between Serum Sodium Levels and Complications of Cirrhosis

Complication	Normal Na (≥135)	Mild Hyponatremia (130–134)	Severe Hyponatremia (<130)	p-value (χ^2)
	Frequency (%)			
Ascites	24 (57.1%)	34 (85.0%)	25 (96.2%)	<0.001
Hepatic encephalopathy	3 (7.1%)	7 (17.5%)	10 (38.5%)	0.005
Spontaneous bacterial peritonitis (SBP)	1 (2.4%)	3 (7.5%)	6 (23.1%)	0.011
Hepatorenal syndrome	0 (0%)	2 (5.0%)	5 (19.2%)	0.007
Upper GI variceal bleed	6 (14.3%)	10 (25.0%)	9 (34.6%)	0.112

Na – Sodium; SBP – Spontaneous Bacterial Peritonitis; χ^2 – Chi-square test.

Serum sodium demonstrated a strong negative correlation with both CTP score ($r = –0.592$, $p < 0.001$) and MELD score ($r = –0.645$, $p < 0.001$), indicating that sodium levels fall as hepatic dysfunction worsens. Significant negative correlations were also found between serum sodium and serum creatinine ($r = –0.424$, $p = 0.002$) as well as total bilirubin ($r = –0.359$, $p = 0.006$). These relationships highlight that hyponatremia reflects not only liver disease severity but also associated renal and metabolic derangements (Table 6).

Table 6: Correlation of Serum Sodium with CTP, MELD, Serum Creatinine, and Total Bilirubin

Variable	Pearson's Correlation Coefficient (r)	p-value
Serum Sodium vs CTP Score	–0.592	<0.001
Serum Sodium vs MELD-Score	–0.645	<0.001
Serum Sodium vs Serum Creatinine	–0.424	0.002
Serum Sodium vs Total Bilirubin	–0.359	0.006

r – Pearson's Correlation Coefficient; CTP – Child–Turcotte–Pugh; MELD – Model for End-Stage Liver Disease.

Patients with lower serum sodium experienced significantly worse short-term outcomes. The mean duration of hospital stay was 6.8 ± 2.1 days in normonatremic patients, increasing to 11.2 ± 3.6 days in those with severe hyponatremia ($p < 0.001$). In-hospital mortality rose sharply from 2.4% in the normal sodium group to 23.1% in the severely hyponatremic group ($p = 0.008$). These findings demonstrate that hyponatremia serves as a robust predictor of prolonged hospitalization and mortality among patients with liver cirrhosis (Table 7).

Table 7: Relationship of Serum Sodium Levels with Duration of Hospital Stay and Mortality

Serum Sodium Category	Duration of Hospital Stay (days)	Mortality
	Mean ± SD	Frequency (%)
Normal (≥135 mmol/L)	6.8 ± 2.1	1 (2.4%)
Mild Hyponatremia (130–134 mmol/L)	8.4 ± 2.7	3 (7.5%)
Severe Hyponatremia(<130 mmol/L)	11.2 ± 3.6	6 (23.1%)
p-value (ANOVA / χ^2)	<0.001	0.008

Na – Sodium; SD – Standard Deviation; ANOVA – Analysis of Variance; χ^2 – Chi-square test.

Discussion

The present study evaluated the clinical significance of serum sodium levels among 108 patients with liver cirrhosis and established a strong correlation between hyponatremia and disease severity, complications, and short-term outcomes.

Hyponatremia is increasingly recognized as a marker of advanced hepatic dysfunction and a

prognostic indicator in cirrhotic patients, particularly in decompensated stages [6].

The mean age of patients in this study was 51.8 years, with a clear male predominance (75.9%), reflecting the higher burden of alcohol-related cirrhosis among Indian men. Similar demographic trends have been reported by Swaroop et al., and Mukherjee et al., where alcohol remained the leading etiology of cirrhosis in over half of the cases [11,12]. In the present series, 57.4% of cases were due to alcohol, followed by hepatitis B (16.7%) and hepatitis C (9.3%), aligning with the epidemiological pattern of mixed etiologies in India [12]. The predominance of decompensated cirrhosis (64.8%) and the high frequency of ascites and jaundice suggest that most patients presented late in the course of illness, as commonly observed in resource-limited healthcare settings [13].

The biochemical parameters reflected significant hepatic dysfunction, as evidenced by elevated bilirubin (3.8 mg/dL), reduced albumin (2.6 g/dL), and prolonged INR (1.74). The mean serum sodium level was 132.4 ± 5.9 mmol/L, indicating that hyponatremia is highly prevalent among cirrhotic patients. Comparable mean sodium values have been reported in studies by Patel et al., and Choudhury et al., where hyponatremia was found in 55–65% of cirrhotic individuals [14,15]. The underlying mechanism involves impaired renal free water excretion secondary to non-osmotic vasopressin release, resulting from portal hypertension and systemic vasodilatation [16]. This pathophysiologic process leads to dilutional hyponatremia, which serves as a sensitive marker of circulatory dysfunction in advanced liver disease [17].

A clear stepwise relationship was noted between serum sodium categories and both Child–Turcotte–Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores. Patients with normal sodium had a mean CTP score of 6.9, which increased to 10.4 in those with moderate-to-severe hyponatremia ($p < 0.001$). Similarly, MELD scores rose from 12.8 to 24.6 across the same groups ($p < 0.001$). These findings underscore that declining serum sodium parallels worsening hepatic reserve and synthetic function [18,19].

This inverse association between serum sodium and liver severity indices corroborates findings by Manka et al., who demonstrated that incorporating sodium into the MELD formula (MELD-Na) improved predictive accuracy for survival in cirrhosis [20].

Mukherjee et al., and Naik et al., also observed that patients with serum sodium <130 mmol/L had significantly higher MELD and CTP scores, validating sodium as an independent marker of advanced disease [21,22].

In the present study, mean serum sodium decreased progressively across CTP classes—from 136.2 mmol/L in Class A to 128.8 mmol/L in Class C ($p < 0.001$). This decline is pathophysiologically explained by increased portal hypertension, hypervolemic hyponatremia, and reduced effective arterial blood volume in advanced cirrhosis. Raj et al., reported a similar trend, noting that serum sodium ≤130 mmol/L was predominantly seen in Child C cirrhotics [23]. This highlights the utility of serum sodium as a simple biochemical marker that parallels the functional deterioration of hepatocytes and progression of portal-systemic circulatory failure [22].

Hyponatremia showed a strong association with the development of major complications such as ascites, hepatic encephalopathy, spontaneous bacterial peritonitis (SBP), and hepatorenal syndrome (HRS). In our cohort, the prevalence of ascites increased from 57.1% in normonatremic patients to 96.2% among those with severe hyponatremia ($p < 0.001$). Similarly, hepatic encephalopathy occurred in 38.5% of patients with sodium <130 mmol/L compared to only 7.1% in those with normal levels ($p = 0.005$). These findings are consistent with Mei et al., and Janičko et al., who reported that hyponatremia is an independent risk factor for hepatic encephalopathy and ascitic decompensation [24,25]. The occurrence of SBP ($p = 0.011$) and HRS ($p = 0.007$) also increased significantly with lower sodium levels, which may be attributed to renal vasoconstriction, impaired perfusion, and reduced glomerular filtration accompanying severe circulatory dysfunction [18]. Although upper gastrointestinal bleeding was more frequent in hyponatremic patients, the association was not statistically significant, possibly due to multifactorial influences on variceal rupture [19].

Correlation analysis further reinforced the inverse relationship of serum sodium with disease severity markers. Sodium correlated strongly and negatively with both CTP ($r = –0.592$, $p < 0.001$) and MELD ($r = –0.645$, $p < 0.001$) scores, as well as with serum creatinine ($r = –0.424$, $p = 0.002$) and total bilirubin ($r = –0.359$, $p = 0.006$). These findings are in agreement with those of Nareddy et al., who demonstrated that hyponatremia is significantly associated with renal dysfunction and hyperbilirubinemia, reflecting both hepatic and renal impairment in decompensated states [26].

The observed correlations affirm that serum sodium acts as an integrative indicator of hepatic, renal, and circulatory dysfunction in cirrhosis [19]. The prognostic significance of hyponatremia was evident inpatient outcomes. The mean hospital stay extended from 6.8 days in normonatremic patients to 11.2 days in severe hyponatremia ($p < 0.001$), and mortality increased markedly from 2.4% to

23.1% (p = 0.008) across the same groups. These findings highlight that hyponatremia is not merely a biochemical abnormality but a predictor of poor short-term prognosis.

Similar trends were reported by Acharya et al., and Goyal et al., who demonstrated that serum sodium ≤130 mmol/L is associated with higher in-hospital mortality and greater risk of organ failure in cirrhotics [27,28]. Singh et al., also found that hyponatremic cirrhotics had longer hospitalization and nearly threefold higher mortality rates compared to normonatremic patients [29].

Clinical Implications: From a clinical standpoint, the present findings underline the necessity of routine monitoring of serum sodium as part of the assessment of cirrhotic patients. Hyponatremia reflects not only advanced liver disease but also imminent complications and poorer outcomes. It should therefore be incorporated into risk stratification, prognostic scoring (MELD-Na), and treatment prioritization, including liver transplantation listing. Early identification and correction of sodium imbalance, through optimization of fluid status, discontinuation of diuretics, or use of vasopressin receptor antagonists, may improve morbidity and short-term prognosis.

Limitations: The study was limited by its single-center design and cross-sectional nature, which precludes establishment of long-term causal associations.

Moreover, serial sodium monitoring and longitudinal outcome tracking were not performed. Despite these limitations, the study provides robust evidence from an Indian tertiary care setting linking serum sodium to clinical severity and prognosis in cirrhosis.

Conclusion

Hyponatremia is a common and clinically significant finding in patients with liver cirrhosis. The present study establishes a strong inverse relationship between serum sodium levels and disease severity, as reflected by higher Child–Turcotte–Pugh and MELD scores, increased frequency of complications, prolonged hospital stay, and greater mortality. Routine monitoring of serum sodium can serve as a simple, cost-effective prognostic marker to guide early intervention and improve patient outcomes in cirrhosis.

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