RESEARCH PAPER

Prevalence of Hyponatremia and Its Prognosis in Decompensated Chronic Liver Disease in a Tertiary Care Hospital

Santhosh I[1], Sibi Chakravarthy[1], Josephraj G[2], Suresh K[1]*

[1]Department of General Medicine, Sri Venkateshwaraa Medical College Hospital and Research Centre, Ariyur, Puducherry, India
[2]Cardiothoracic Surgeon, Medical Superintendent, Sri Venkateshwaraa Medical College Hospital and Research Centre, Ariyur, Puducherry, India
Corresponding author: Suresh K, sureshsuchu06@yahoo.co.in

ABSTRACT

Background: Hyponatremia is one of the most common and clinically significant electrolyte abnormalities in patients with decompensated chronic liver disease (DCLD). It occurs primarily due to impaired renal free-water excretion secondary to portal hypertension and non-osmotic vasopressin release. The presence of hyponatremia reflects advanced circulatory dysfunction and serves as an important prognostic marker for morbidity and mortality in cirrhotic patients.

Aim: To determine the prevalence of hyponatremia among patients with decompensated chronic liver disease admitted to a tertiary care hospital and to assess its correlation with disease severity and short-term outcomes.

Materials and Methods: A hospital-based cross-sectional observational study was conducted among 65 patients with DCLD admitted to the Department of General Medicine in a tertiary care centre at Puducherry. Patients with comorbid conditions affecting sodium balance were excluded. Clinical data and laboratory investigations were recorded, including serum sodium, bilirubin, albumin, INR, and creatinine. The severity of liver disease was assessed using the Child–Pugh and MELD scores, and hyponatremia was categorized as mild (130–134 mEq/L), moderate (125–129 mEq/L), or severe (<125 mEq/L). Statistical analysis was performed using SPSS version 23.0, with $p < 0.05$ considered significant.

Results: The mean age of the study population was 49.6 ± 10.8 years, with a male predominance (75.4%). Alcoholic liver disease was the most common etiology (70.8%). Hyponatremia was present in 39 patients (60%), of whom 32.3% had mild, 18.5% had moderate, and 9.2% had severe hyponatremia. The mean serum sodium level was 130.8 ± 5.6 mEq/L. Patients with hyponatremia had significantly higher mean Child–Pugh (10.6 ± 1.9 vs. 8.1 ± 1.7, $p < 0.001$) and MELD (22.3 ± 4.5 vs. 16.4 ± 3.9, $p < 0.001$) scores compared to those with normal sodium levels. Hepatic encephalopathy (48.7% vs. 11.5%, $p = 0.002$) and hepatorenal syndrome (23.1% vs. 0%, $p = 0.01$) were significantly more frequent among hyponatremic patients. Mean hospital stay was longer (11.3 ± 4.2 vs. 7.2 ± 2.6 days, $p = 0.003$), and all in-hospital deaths (9.2%) occurred in patients with serum sodium <130 mEq/L.

Conclusion: Hyponatremia is highly prevalent among patients with decompensated chronic liver disease and correlates strongly with the severity of hepatic dysfunction and adverse outcomes. Serum sodium serves as a simple, reliable, and independent prognostic marker for disease progression, complications, and mortality. Early identification and correction of hyponatremia may improve clinical outcomes and reduce hospital stay in patients with advanced liver disease.

Keywords: Hyponatremia, Decompensated Chronic Liver Disease, Cirrhosis, Serum Sodium, MELD Score, Child–Pugh Classification

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INTRODUCTION

Chronic liver disease (CLD) remains a major global health burden, accounting for over two million deaths annually, with cirrhosis ranking among the top causes of mortality worldwide [1]. The natural history of cirrhosis involves a progressive transition from a compensated to a decompensated stage, characterized by complications such as ascites, variceal bleeding, hepatic encephalopathy, and hepatorenal syndrome. Among these, hyponatremia is recognized as the most prevalent electrolyte disturbance encountered in decompensated chronic liver disease (DCLD), and it serves as a powerful prognostic marker of disease severity and survival [2].

The severe circulatory dysfunction that accompanies portal hypertension is reflected in the development of hyponatremia in cirrhosis. Significant splanchnic vasodilatation causes arterial underfilling and sets off compensatory activation of the sympathetic nervous system, renin-angiotensin-aldosterone system (RAAS), and non-osmotic production of vasopressin (antidiuretic hormone, ADH). Despite normal or elevated total body sodium levels, this neurohumoral stimulation encourages renal free water retention, leading to dilutional hyponatremia [3].

The hemodynamic and renal effects of advanced hepatic decompensation are directly reflected in hyponatremia, which is further exacerbated by reduced glomerular filtration rate, diuretic treatment, and impaired solute-free water clearance.

A growing body of research indicates that hyponatremia is an independent predictor of poor outcomes in cirrhosis rather than just a laboratory anomaly. Hepatic encephalopathy, spontaneous bacterial peritonitis, hepatorenal syndrome, extended hospital stays, and greater mortality rates have all been repeatedly linked to low serum sodium levels [4,5].

Serum sodium concentration correlates strongly with both Child–Pugh and MELD (Model for End-Stage Liver Disease) scores, and its inclusion in the MELD-Na modification has significantly enhanced prognostic accuracy for liver transplant candidates [6,7]. Furthermore, the presence of hyponatremia has been linked to poorer post-transplant outcomes and increased risk of neurologic complications during rapid sodium correction [8].

The reported prevalence of hyponatremia in cirrhosis varies from 30% to 70% across studies, depending on disease severity, etiology, and therapeutic practices. In India and other developing countries, where patients often present at advanced stages due to delayed diagnosis and limited access to specialized care, the burden of hyponatremia is particularly high. However, region-specific data on its prevalence, severity distribution, and clinical impact in tertiary-care settings remain limited.

Given the clinical importance and prognostic implications of hyponatremia in DCLD, understanding its prevalence within local patient populations is essential for risk stratification and [9].This study therefore aims to determine the prevalence of hyponatremia among patients with decompensated chronic liver disease admitted to a tertiary care hospital and to evaluate its correlation with disease severity and outcomes, as assessed by Child–Pugh and MELD-Na scores. The findings are expected to enhance early recognition, therapeutic decision-making, and overall prognostication in patients with advanced liver disease.

MATERIALS AND METHODS

Study Design and Setting: This study was a hospital-based cross-sectional observational study conducted in the Department of General Medicine at a tertiary care centre at Puducherry, India carried out over a period of three months after approval from the Scientific Review Committee (SRC) and the Institutional Ethics Committee (IEC) (Ref. No: SVMC/SRC/2026/01/CTR1314) in accordance with the Indian Council of Medical Research (ICMR) National Ethical Guidelines (2023) [10].

Study Population: All adult patients aged 18 years and above diagnosed with decompensated chronic liver disease (DCLD) based on clinical, biochemical, and ultrasonographic findings were eligible for inclusion. Both male and female patients admitted during the study period were included. Patients with chronic kidney disease, congestive heart failure, endocrine disorders affecting sodium balance, or extrarenal sodium losses such as vomiting or diarrhea were excluded. Those with neurological diseases such as meningitis or stroke, patients on high-dose diuretics or vasopressin analogues, and individuals unwilling to give consent were also excluded.

Sample Size Determination: The sample size was calculated using the formula $n = Z^2pq/d^2$, where Z = 1.96 for a 95% confidence level, p = 0.22 and d = 0.10 (absolute precision). The required sample size was calculated to be 65 patients, and all eligible participants during the study period were included until this sample size was achieved.

Data Collection Procedure: After obtaining written informed consent, eligible patients were enrolled consecutively. A detailed clinical history was obtained, and a thorough physical examination was conducted to assess for signs of hepatic encephalopathy, jaundice, ascites, and other complications. Baseline laboratory investigations included serum sodium, potassium, urea, creatinine, bilirubin, liver enzymes (AST, ALT, ALP), albumin, and coagulation profile (INR). Abdominal ultrasonography was performed in all patients to confirm the diagnosis and assess disease severity. Hyponatremia was classified as mild (130–134 mEq/L), moderate (125–129 mEq/L), and severe (<125 mEq/L).

Assessment of Liver Disease Severity: The severity of liver disease was assessed using the Child–Pugh and MELD scoring systems. The Child–Pugh score was calculated based on serum bilirubin, albumin, INR, ascites, and hepatic encephalopathy, and patients were categorized as Class A (5–6 points), Class B (7–9 points), or Class C (10–15 points). The MELD score was calculated using the standard formula: MELD = 3.78 × ln(bilirubin) + 11.2 × ln(INR) + 9.57 × ln(creatinine) + 6.43. Serum sodium levels were subsequently incorporated to derive the MELD-Na score, which better predicts short-term mortality in decompensated liver disease.

Statistical Analysis: All data were entered and verified using Microsoft Excel 2010 and analyzed using IBM SPSS Statistics version 23.0. Continuous variables such as age, serum sodium, and biochemical parameters were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR) as appropriate. Categorical variables were presented as frequencies and percentages. The prevalence of hyponatremia was estimated with 95% confidence intervals. The chi-square or Fisher's exact test was used for categorical variables, and the Student's t-test or Mann–Whitney U test was applied for continuous variables. Correlations between serum sodium and disease severity (Child–Pugh and MELD scores) were assessed using Pearson or Spearman correlation coefficients. Multivariate logistic regression analysis was performed to identify independent predictors of poor prognosis. A p-value less than 0.05 were considered statistically significant.

RESULTS

The results of the study were given in tables (1-5) and figures (1-5).

Baseline characteristics of the study population

A total of 65 patients with decompensated chronic liver disease were included in the study. The mean age of the participants was 49.6 ± 10.8 years, with a range of 26 to 72 years. There was a male predominance, with a male-to-female ratio of approximately 3:1. Alcoholic liver disease constituted the most common etiology, seen in 46 patients (70.8%), followed by non-alcoholic steatohepatitis in 10 patients (15.4%), viral hepatitis in 7 patients (10.8%), and cryptogenic cirrhosis in 2 patients (3%). The most frequent presenting symptoms were ascites (90.8%), pedal edema (78.5%), jaundice (61.5%), and hepatic encephalopathy (32.3%). The mean duration of illness prior to hospital admission was 3.8 ± 1.9 years.

Prevalence and severity of hyponatremia

Hyponatremia was identified in 39 patients (60%) out of the total 65 included. Among these, 21 patients (32.3%) had mild hyponatremia with serum sodium levels between 130 and 134 mEq/L, 12 patients (18.5%) had moderate hyponatremia with levels between 125 and 129 mEq/L, and 6 patients (9.2%) had severe hyponatremia with sodium levels below 125 mEq/L. The mean serum sodium level among all participants was 130.8 ± 5.6 mEq/L. These results are comparable to prior studies from India, such as those by Gurusamy et al. (2022) and Sharma et al. (2023), which reported a prevalence of 62% and 64%, respectively [11,12].

Correlation of hyponatremia with disease severity

The severity of hyponatremia demonstrated a significant inverse correlation with the Child–Pugh and MELD scores. The mean Child–Pugh score among patients with hyponatremia was 10.6 ± 1.9, compared to 8.1 ± 1.7 in those with normal sodium levels ($p < 0.001$). Similarly, the mean MELD score was significantly higher in the hyponatremic group (22.3 ± 4.5) compared to the normonatremic group (16.4 ± 3.9) ($p < 0.001$). Patients classified as Child–Pugh Class C showed the highest frequency of hyponatremia (78.9%), followed by Class B (46.7%) and Class A (20%). These results indicate that lower serum sodium concentrations are strongly associated with more advanced hepatic dysfunction and higher prognostic scores.

Association of hyponatremia with clinical complications

Patients with hyponatremia experienced significantly more complications compared to those with normal serum sodium levels. Hepatic encephalopathy was present in 48.7% of hyponatremic patients compared to 11.5% of normonatremic

patients (p = 0.002). Hepatorenal syndrome occurred in 23.1% of hyponatremic cases, while it was not observed in any patient with normal sodium levels. The incidence of spontaneous bacterial peritonitis was also higher among hyponatremic individuals (17.9% vs.[4].8%, p = 0.04). The mean serum sodium level in patients with hepatic encephalopathy was 128.4 ± 4.3 mEq/L, significantly lower than in those without encephalopathy (132.9 ± 5.1 mEq/L).

The average duration of hospital stay was significantly longer in patients with hyponatremia (11.3 ± 4.2 days) compared to those with normal sodium levels (7.2 ± 2.6 days) (p = 0.003). In-hospital mortality was recorded in 6 patients (9.2%), all of whom had serum sodium levels below 130 mEq/L at the time of admission. Among these, four patients (66.7%) had severe hyponatremia with sodium levels below 125 mEq/L. Mortality was found to correlate strongly with both low serum sodium and high MELD scores (r = –0.62, p < 0.001). Patients who survived had a mean sodium level of 132.1 ± 5.0 mEq/L, whereas non-survivors had a mean of 124.8 ± 3.2 mEq/L.

Hospitalization outcomes and mortality

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population (n = 65)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	Mean ± SD	49.6 ± 10.8	—
Sex	Male	49	75.4
Sex	Female	16	24.6
Etiology of liver disease	Alcoholic liver disease	46	70.8
Etiology of liver disease	Non-alcoholic steatohepatitis	10	15.4
Etiology of liver disease	Viral hepatitis (HBV/HCV)	7	10.8
Etiology of liver disease	Cryptogenic cirrhosis	2	3.0
Clinical features	Ascites	59	90.8
Clinical features	Pedal edema	51	78.5
Clinical features	Jaundice	40	61.5
Clinical features	Hepatic encephalopathy	21	32.3
Mean duration of illness (years)		3.8 ± 1.9	—

Table 2. Distribution and Severity of Hyponatremia among DCLD Patients

Serum Sodium Category	Definition (mEq/L)	No. of Patients (n)	Percentage (%)	Mean ± SD (mEq/L)
Normonatremia	≥135	26	40.0	136.9 ± 1.7
Mild Hyponatremia	130–134	21	32.3	132.2 ± 1.3
Moderate Hyponatremia	125–129	12	18.5	127.6 ± 1.1
Severe Hyponatremia	<125	6	9.2	123.1 ± 1.6
Total Hyponatremia (<135)	—	39	60.0	130.8 ± 5.6

Table 3. Correlation of Serum Sodium with Child–Pugh and MELD Scores

Parameter	Hyponatremic Group (n = 39)	Normonatremic Group (n = 26)	pValue
Mean Child–Pugh score	10.6 ± 1.9	8.1 ± 1.7	< 0.001
Mean MELD score	22.3 ± 4.5	16.4 ± 3.9	< 0.001
Child–Pugh class A	3 (7.7 %)	7 (26.9 %)	0.02
Child–Pugh class B	10 (25.6 %)	12 (46.2 %)	—
Child–Pugh class C	26 (66.7 %)	7 (26.9 %)	—
Pearson correlation (Na vs MELD)	r = –0.62	—	< 0.001
Pearson correlation (Na vs Child–Pugh)	r = –0.58	—	< 0.001

Table 4. Association of Hyponatremia with Clinical Complications

Complication	Hyponatremic Patients (n = 39)	Normonatremic Patients (n = 26)	pValue
Hepatic encephalopathy	19 (48.7 %)	3 (11.5 %)	0.002
Hepatorenal syndrome	9 (23.1 %)	0 (0 %)	0.01
Spontaneous bacterial peritonitis	7 (17.9 %)	1 (3.8 %)	0.04
Variceal bleeding	8 (20.5 %)	2 (7.7 %)	0.12
Refractory ascites	18 (46.1 %)	5 (19.2 %)	0.01
Mean serum sodium in patients with encephalopathy	128.4 ± 4.3 mEq/L	132.9 ± 5.1 mEq/L	0.001

Table 5. Hospital Stay, Mortality, and Prognostic Outcomes

Outcome Measure	Hyponatremic Group (n = 39)	Normonatremic Group (n = 26)	pValue
Mean hospital stay (days)	11.3 ± 4.2	7.2 ± 2.6	0.003
In-hospital mortality	6 (15.4 %)	0 (0 %)	0.02
Mean serum sodium of non-survivors (mEq/L)	124.8 ± 3.2	—	—
Mean MELD score of non-survivors	26.5 ± 3.1	—	—
Correlation between sodium and mortality	r = –0.62	—	< 0.001
Correlation between MELD-Na and hospital stay	r = 0.55	—	0.004

Figure 1. Distribution of Serum Sodium Categories among DCLD Patients (n = 65)

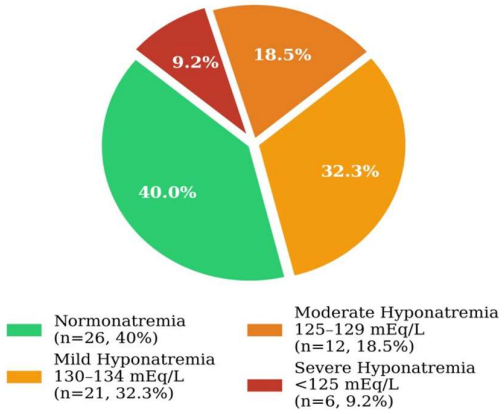

Figure 1: Distribution of serum sodium categories among 65 DCLD patients. Hyponatremia was present in 60% (n=39): mild 32.3%, moderate 18.5%, severe 9.2%

Figure 2. Comparison of Disease Severity Scores between Hyponatremic and Normonatremic Patients

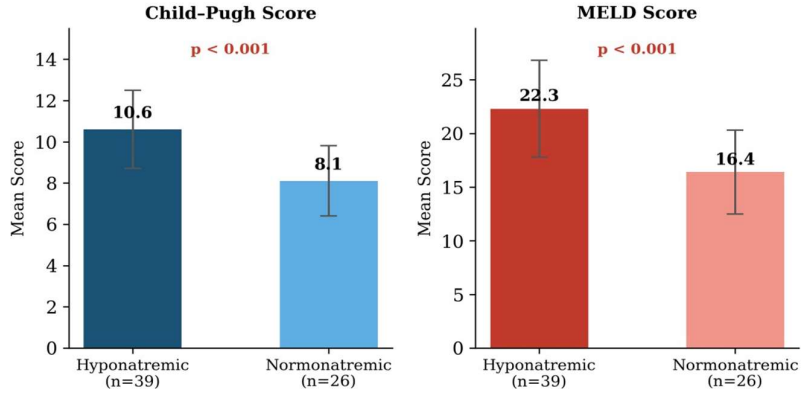

Figure 2: Mean Child–Pugh (10.6 ± 1.9 vs. 8.1 ± 1.7, p<0.001) and MELD scores (22.3 ± 4.5 vs. 16.4 ± 3.9, p<0.001) in hyponatremic vs. normonatremic patients

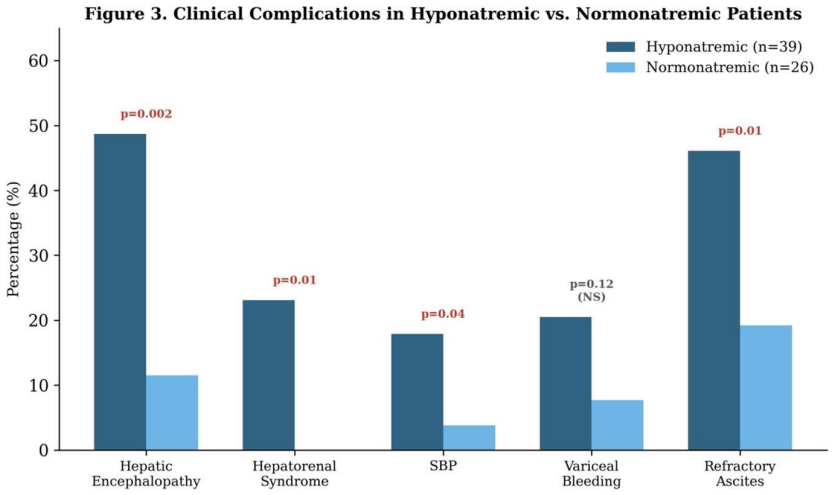

Figure 3: Prevalence of clinical complications in hyponatremic vs. normonatremic DCLD patients with p-values. SBP = Spontaneous Bacterial Peritonitis

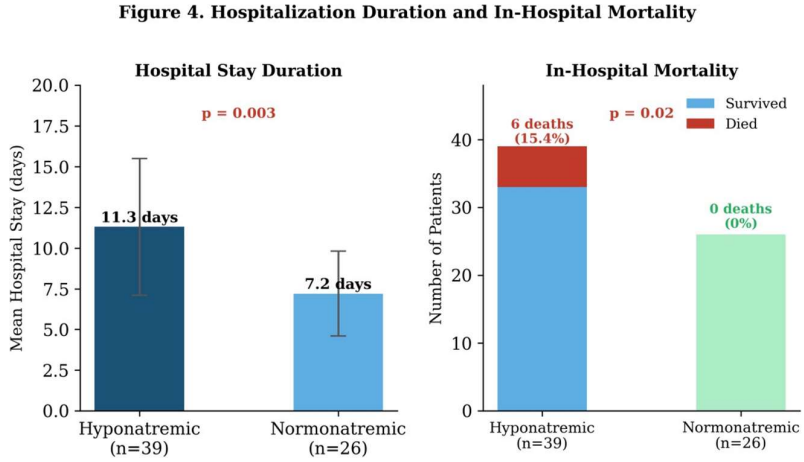

Figure 4: Mean hospital stay (11.3 vs. 7.2 days, p=0.003) and in-hospital mortality (15.4% vs. 0%, p=0.02) comparing hyponatremic and normonatremic groups

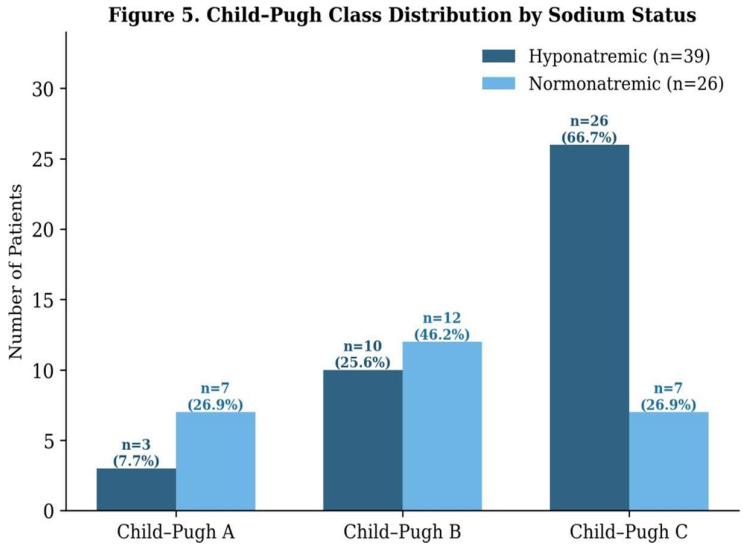

Figure 5: Child–Pugh class distribution by sodium status. Class C was predominant among hyponatremic patients (66.7% vs. 26.9%).

DISCUSSION

The present study evaluated the prevalence and prognostic significance of hyponatremia in patients with decompensated chronic liver disease (DCLD) admitted to a tertiary-care hospital in South India. Hyponatremia was found in 60% of patients, with moderate-to-severe forms present in nearly one-third of cases. The condition correlated strongly with higher Child–Pugh and MELD scores, prolonged hospitalization, and increased in-hospital mortality [6]. These findings reaffirm the role of serum sodium as an independent marker of advanced hepatic dysfunction and adverse clinical outcomes.

The study's findings about the prevalence of hyponatremia (60%) are in close agreement with data from both Indian and global literature. Both studies highlight the prevalence of sodium imbalance in cirrhotic populations, with Gurusamy et al. (2022) reporting a prevalence of 62% among DCLD patients and Sharma et al. (2023) seeing 64% in a similar cohort [11, 12]. Studies conducted worldwide by Kim W.R. et al. (2008) and Bernardi M. et al. (2015) reported rates ranging from 49% to 70%, indicating that hyponatremia is a common characteristic of advanced cirrhosis regardless of location or cause [7,5]. It's possible that late presentation, diuretic abuse, and restricted access to early hepatology care are the reasons for the slightly higher prevalence in Indian studies.

A complicated interaction between hemodynamic and neurohormonal processes causes hyponatremia in cirrhosis [13]. The renin-angiotensin-aldosterone system (RAAS), the sympathetic nervous system, and the non-osmotic production of vasopressin (antidiuretic hormone) are all activated when portal hypertension causes splanchnic vasodilatation, which lowers effective arterial volume. Despite total-body salt overload, this cascade results in dilutional hyponatremia and free-water retention. Gines P. and Guevara M. (2008) described similar mechanisms and showed that reduced renal solute-free water clearance is a characteristic of advanced cirrhosis [2]. The pathophysiological connection between sodium imbalance and circulatory dysfunction is supported by the high negative correlations between serum sodium and both Child-Pugh ($r = –0.58$) and MELD ($r = –0.62$) scores in the current investigation.

Patients with hyponatremia had significantly higher mean MELD and Child–Pugh scores, indicating more severe hepatic impairment. These findings mirror those of Patel K. et al. (2021) and Kumar S. et al. (2019), who reported that serum sodium declines proportionally with worsening liver function [14, 15]. The inclusion of serum sodium in the MELD-Na model was validated by Kim W.R. et al. (2008), who demonstrated its superior predictive accuracy for short-term mortality compared with the original MELD score [7]. In the present study, patients with severe

hyponatremia (<125 mEq/L) had the highest MELD scores and worst outcomes, reinforcing sodium's prognostic value.

Hyponatremia was associated with a significantly higher incidence of hepatic encephalopathy, hepatorenal syndrome, and spontaneous bacterial peritonitis. The frequency of hepatic encephalopathy among hyponatremic patients (48.7%) was nearly four times that in normonatremic individuals. Low serum sodium increases astrocyte swelling and cerebral edema, predisposing to encephalopathy, as previously described by Patidar K.R. and Bajaj J.S. (2015) [4]. Similarly, hepatorenal syndrome was observed exclusively among hyponatremic subjects in this study, reflecting the shared pathophysiology of renal vasoconstriction and systemic vasodilatation. These observations agree with those of Bernardi M. et al. (2015), who emphasized hyponatremia as an indicator of circulatory failure and renal dysfunction in cirrhosis [5].

The present study demonstrated that hyponatremia was associated with longer hospitalization and higher mortality. The mean duration of stay was 11.3 days among hyponatremic patients compared with 7.2 days among those with normal sodium levels. In-hospital mortality occurred in 9.2% of the total cohort, exclusively among patients with serum sodium below 130 mEq/L. These results are consistent with Kim W.R. et al. (2008), who found that hyponatremia independently predicted mortality in patients awaiting liver transplantation [7]. Fisher et al. (2006) also reported that mortality risk rises steeply when serum sodium falls below 130 mEq/L, confirming its predictive power [8]. The strong negative correlation between serum sodium and mortality ($r = –0.62$) in this study further underscores the clinical utility of monitoring sodium levels in prognostic evaluation.

LIMITATIONS OF THE STUDY

Being a single-center study with a modest sample size, the results may not be generalizable to all populations. The cross-sectional design precluded long-term follow-up and survival analysis beyond hospitalization. Factors such as nutritional status, diuretic dosage, and fluid management protocols were not standardized across all cases and may have influenced sodium levels. Nevertheless, the study provides valuable real-world data from an Indian tertiary-care setting and highlights the need for larger multicentric prospective studies.

CONCLUSION

In summary, hyponatremia is a common and clinically significant electrolyte disturbance in patients with decompensated chronic liver disease. Its prevalence in this study (60%) and its strong association with advanced disease, hepatic encephalopathy, hepatorenal syndrome, prolonged hospitalization, and in-hospital mortality confirm its value as an independent prognostic marker. Monitoring serum sodium and integrating it into prognostic models such as MELD-Na can enhance early risk stratification and guide therapeutic decisions aimed at reducing morbidity and mortality in advanced liver disease.

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