International Journal of Medicine & Health Research
www.internationalmedicalpublishing.com

Research Article

Section: General Medicine

The Association Between the Serum Sodium Level & Severity of Complications of Chronic Liver Disease - A Hospital-Based Observational Study

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HIGHLIGHTS

- Hyponatremia predicts prognosis
- Lower sodium worsens complications
- Correlates with cirrhosis severity
- Increases short-term mortality
- Simple prognostic marker

Key Words:
Chronic liver disease
Hyponatremia
Cirrhosis, Prognosis
MELD score
Child-Pugh score

ABSTRACT

Introduction: Chronic liver disease (CLD) leads to cirrhosis and complications like ascites and encephalopathy. Hyponatremia, often resulting from impaired solute-free water clearance, is common in these patients. This study evaluates the association between serum sodium levels and the severity of complications in CLD. **Aims & Objective:** To evaluate the association between serum sodium levels and the severity of complications in CLD, establish hyponatremia as a prognostic marker, identify complications at different sodium levels, and estimate short-term survival rates. **Materials & Methods:** This hospital-based observational study was conducted at Tezpur Medical College and Hospital over one year (2024-2025) on 278 patients with liver cirrhosis. Diagnosis was confirmed by ultrasonography. Serum sodium levels were correlated with complications, severity scores (Child-Pugh, MELD), 28-day morbidity, and mortality using appropriate statistical tests. **Results:** A strong, statistically significant association was observed between lower serum sodium levels and increased severity of complications ($p<0.001$). Patients with sodium <130 mEq/L had the highest rates of severe complications (43%), Child-Pugh Class C (100%), and higher mean MELD scores (26.72±4.38). Hyponatremia was significantly associated with ascites, hepatorenal syndrome, variceal bleeding, SBP, and higher grades of hepatic encephalopathy ($p<0.001$ for all). Strong negative correlations were found between sodium and both the Child-Pugh ($r = -0.84$) and MELD ($r = -0.81$) scores. Twenty-eight-day morbidity (100%) and mortality (24%) were highest in the <130 mEq/L group. ROC analysis identified a serum sodium cut-off of 131.5 mEq/L for predicting 28-day mortality (AUC 0.67). **Conclusion:** Serum sodium is a simple, valuable prognostic marker in CLD. Hyponatremia is strongly associated with more severe complications, advanced liver disease, and higher short-term morbidity and mortality.

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Article History: Received 13 May 2026; Received in Revised form 02 June 2026; Accepted 09 July 2026
How To Cite: Pritam Barai & Rupam Pegu. The Association Between the Serum Sodium Level & Severity of Complications of Chronic Liver Disease - A Hospital-Based Observational Study. *International Journal of Medicine & Health Research.* 2026;14(2):1-9. DOI: https://doi.org/10.71393/x9m5yy29

INTRODUCTION

Chronic liver disease (CLD) is a progressive condition characterized by persistent inflammation and gradual destruction of liver tissue lasting more than six months. Continuous hepatic injury caused by viral hepatitis, alcohol abuse, metabolic disorders, autoimmune diseases, or genetic factors leads to fibrosis and eventually cirrhosis, which represents the irreversible stage of liver damage. In cirrhosis, normal hepatic architecture is replaced by fibrous tissue and regenerative nodules, resulting in distortion of intrahepatic blood flow and impaired liver function. Since the damaged hepatic structure cannot regenerate completely, cirrhosis is considered permanent and is associated with substantial morbidity and mortality [1]. The progression of cirrhosis is closely related to repeated cycles of hepatocellular injury and incomplete repair, which gradually increase resistance within the portal venous system and impair the metabolic and synthetic functions of the liver. These pathological changes contribute to severe complications such as portal hypertension, ascites, variceal bleeding, hepatic encephalopathy, and hepatorenal syndrome, significantly reducing survival and quality of life [2].

Globally, chronic liver disease and cirrhosis remain major public health concerns, contributing to more than one million deaths annually. Although viral hepatitis and alcohol-related liver disease continue to be important etiological factors, non-alcoholic fatty liver disease has emerged rapidly due to increasing obesity, diabetes, and sedentary lifestyles worldwide.

In India, the burden of liver disease is particularly high, accounting for a significant proportion of global liver-related mortality. Rapid urbanization, unhealthy dietary habits, increased alcohol consumption, and metabolic disorders have further accelerated the prevalence of cirrhosis and its complications in recent decades. Consequently, CLD imposes substantial economic and healthcare burdens through repeated hospitalizations, disability, and premature deaths. Effective prevention, early diagnosis, and timely management are therefore essential to reduce disease progression and improve outcomes [3,4].

The pathophysiology of cirrhosis involves progressive fibrosis and vascular distortion, leading to increased resistance to portal blood flow and development of portal hypertension. Elevated portal pressure causes the formation of collateral circulation and contributes to complications such as variceal bleeding and splenomegaly. Simultaneously, systemic and splanchnic vasodilation produce a hyperdynamic circulatory state, reducing effective arterial blood volume despite fluid overload. This perceived circulatory underfilling activates neurohormonal systems including the renin-angiotensin-aldosterone system and sympathetic nervous system, causing sodium and water retention. These hemodynamic disturbances lead to accumulation of ascitic fluid, worsening portal hypertension, & impairment of renal perfusion. Persistent portal hypertension also predisposes patients to spontaneous bacterial peritonitis & hypersplenism-associated cytopenias [5,6].

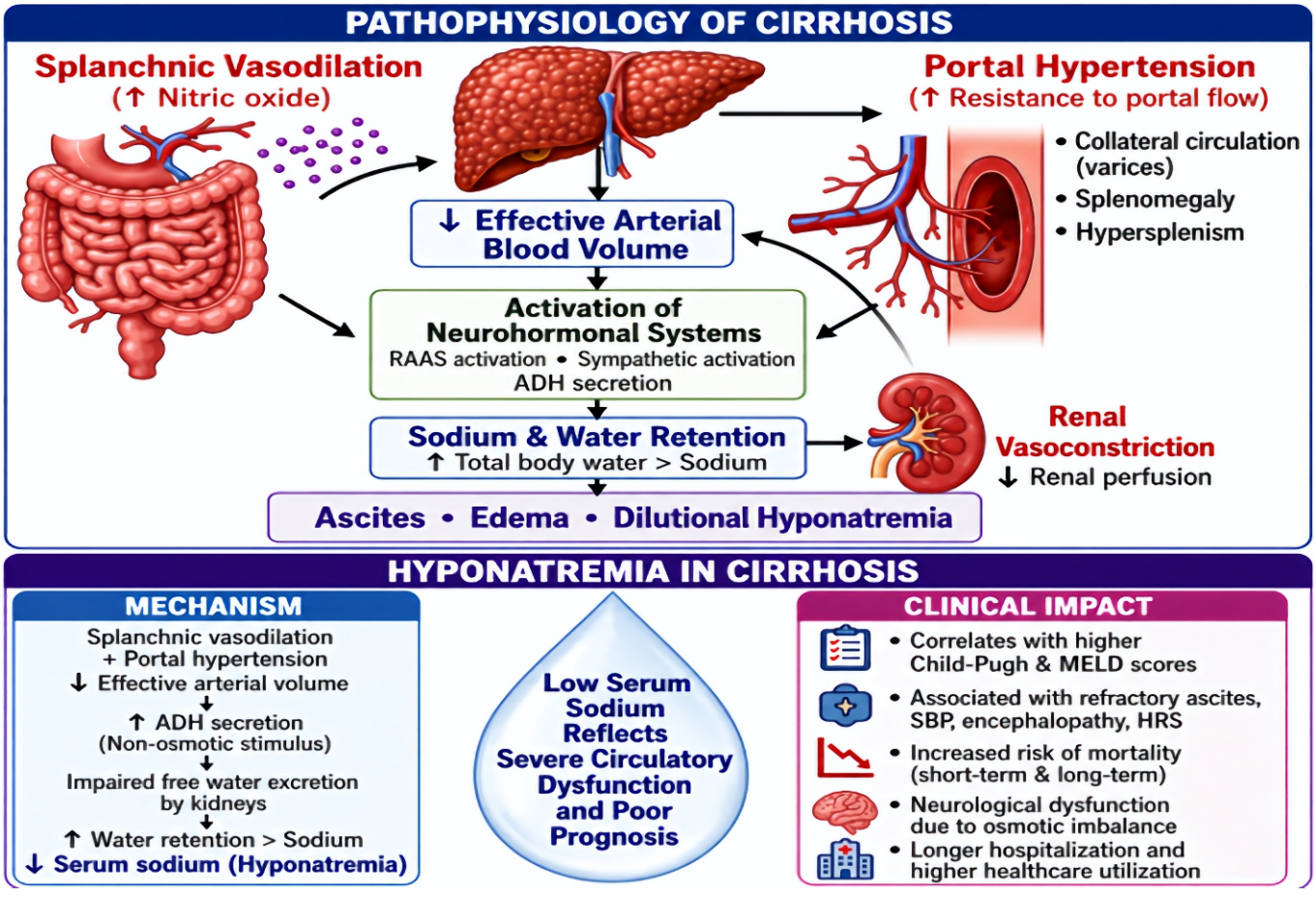

Figure 1: Pathophysiology of Cirrhosis and Mechanisms of Hyponatremia.

In advanced cirrhosis, impaired hepatic detoxification and portosystemic shunting result in accumulation of neurotoxins such as ammonia, leading to hepatic encephalopathy characterized by altered cognition and consciousness. Renal dysfunction may progress to hepatorenal syndrome due to severe renal vasoconstriction secondary to systemic vasodilation and neurohormonal activation. Cirrhosis can additionally produce cardiopulmonary complications such as hepatopulmonary syndrome and cirrhotic cardiomyopathy, reflecting the multisystem nature of advanced liver disease. Among the various metabolic abnormalities seen in cirrhosis, disturbances in sodium and water balance are particularly important because they indicate severe circulatory dysfunction and poor prognosis. Splanchnic vasodilation and activation of antidiuretic hormone secretion impair renal free water excretion, resulting in dilutional hyponatremia, where total body water increases disproportionately compared to sodium levels [7,8].

Hyponatremia is one of the most common electrolyte abnormalities in cirrhosis and is frequently associated with advanced disease and ascites. Reduced serum sodium levels correlate strongly with higher Child-Pugh and MELD scores, indicating worsening hepatic dysfunction and circulatory impairment. Clinically, hyponatremia is associated with increased risks of refractory ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, hepatorenal syndrome, prolonged hospitalization, and mortality. Studies have shown that low serum sodium levels are independent predictors of poor short-term and long-term outcomes in cirrhotic patients. Severe hyponatremia increases susceptibility to neurological dysfunction due to osmotic imbalance and worsens renal hypoperfusion, thereby contributing to hepatorenal syndrome and multiorgan dysfunction [9,10].

Because of its strong prognostic significance, serum sodium has been incorporated into prognostic models such as the MELD-Na score used in liver transplantation prioritization. Hyponatremia not only reflects disease severity but also serves as an important marker for monitoring disease progression and guiding clinical management. Lower sodium levels have consistently been associated with increased morbidity, mortality, longer hospital stays, and greater healthcare utilization among patients with cirrhosis. Therefore, routine assessment of serum sodium plays a critical role in evaluating prognosis, identifying high-risk patients, and improving management strategies in chronic liver disease [11,12]. **Figure 1.** Pathophysiology of Hyponatremia in Cirrhosis. Splanchnic vasodilation and portal hypertension activate neurohormonal pathways, causing sodium and water retention, dilutional hyponatremia, and poor clinical outcomes.

The aim of this study is to evaluate the association between serum sodium levels and the severity of complications in patients with chronic liver disease. The objectives are to assess the prevalence of hyponatremia in cirrhosis, analyze its relationship with major cirrhosis related complications, and determine its prognostic significance with respect to disease severity, morbidity, and clinical outcomes in a hospital based observational setting in northeastern states of India.

MATERIALS & METHODS

This hospital-based observational study was conducted at the Department of General Medicine, Tezpur Medical College & Hospital, Tezpur, Assam, from June 2024 to May 2025. Ethical approval has been obtained from the Ethical Approval Committee of Tezpur Medical College & Hospital, Tezpur, Assam.

Study Population

The study population included hospitalized patients with complications of liver cirrhosis aged 29–82 years, having Child-Pugh scores between 5–15, MELD scores between 5–36, and serum sodium concentrations ranging from 115 to 147. Patients were excluded if hepatocellular carcinoma was present at admission or during follow-up, if they had used antiviral drugs during the survival period, or if they had chronic kidney disease or cardiac failure.

Data Analysis

Data analysis was conducted using SPSS software after data entry and verification in Microsoft Excel for completeness and errors. Baseline characteristics were summarized using descriptive statistics including mean, standard deviation, frequency, and percentage. Inferential statistics involved Chi-square tests for categorical variables & ANOVA for comparison of means. Pearson correlation analysis assessed associations between serum sodium and severity scores, while relationships between sodium levels and complications were evaluated. A p-value below 0.05 was considered statistically significant.

RESULTS

This study was conducted at Tezpur Medical College and Hospital from June 2024 to May 2025 over a duration of one year. A total of 278 patients with chronic liver disease fulfilling the inclusion criteria were enrolled after obtaining informed consent. A statistically significant association was observed between serum sodium levels and severity of complications ($\chi^2 = 52.84$, $p < 0.001$). Patients with serum sodium levels below 130 mmol/L had the highest proportion of severe complications (43%), whereas patients with sodium levels above 135 mmol/L predominantly had mild complications (51%), indicating that worsening hyponatremia was strongly associated with increased clinical severity in liver cirrhosis. Regarding age distribution, the majority of patients belonged to the 51–60 years age group (32.73%), followed by 61–70 years (24.46%) and 41–50 years (18.35%). Patients aged 71–80 years accounted for 12.23%, while 8.27% were between 29–40 years, and only 3.96% were older than 80 years. These findings suggest that chronic liver

disease and cirrhosis were most prevalent among middle-aged and elderly individuals, particularly those between 51 and 70 years of age. Ascites was the most common complication among liver cirrhosis patients (65.47%), followed by hepatic encephalopathy (38.85%) and coagulopathy (26.62%), while hepatorenal syndrome was the least frequent (14.39%). Overall, fluid-related and neurological complications were more prevalent than renal and infective complications **(Table 1)**. Alcoholic liver disease was the leading cause of liver cirrhosis (44.96%), followed by Hepatitis B (18.35%) and NAFLD (16.19%), while autoimmune hepatitis and cryptogenic cirrhosis were least common. Overall, alcohol-related liver disease remained the predominant etiological factor in the study population **(Table 2)**. Patients with serum sodium <130 mmol/L showed the highest frequency of severe complications, whereas those with sodium >135 mmol/L predominantly had mild complications. Overall, decreasing serum sodium levels were strongly associated with increasing severity of complications, highlighting hyponatremia as an indicator of advanced liver disease severity **(Figure 2)**. A highly statistically significant association was observed between serum sodium levels and Child-Pugh class ($\chi^2 = 242.61$, $p < 0.001$). All patients with serum sodium levels below 130 mmol/L belonged to Child-Pugh Class C, indicating severe hepatic dysfunction among hyponatremic patients. In the 131–135 mmol/L category, most patients were classified as Child-Pugh Class B, while patients with sodium levels above 135 mmol/L were predominantly distributed in Child-Pugh Classes A and B, with none in Class C. These findings clearly demonstrate that decreasing serum sodium levels are strongly associated with worsening liver disease severity. A significant association was also found between serum sodium levels and the presence of ascites ($\chi^2 = 61.84$, $p < 0.001$). Ascites was present in 87% of patients with sodium levels below 130 mmol/L, compared to 55% in the 131–135 mmol/L group and only 23 patients in the >135 mmol/L category, indicating that hyponatremia is closely associated with fluid accumulation and advanced portal hypertension in liver cirrhosis. Similarly, hepatorenal syndrome showed a significant association with serum sodium levels ($\chi^2 = 32.75$, $p < 0.001$), with the highest frequency observed among patients with sodium <130 mmol/L (36%), while only 4% of patients with sodium >135 mmol/L had hepatorenal syndrome, suggesting that worsening hyponatremia parallels deterioration in renal function and circulatory status. Variceal bleeding was also significantly associated with serum sodium levels ($\chi^2 = 14.92$, $p = 0.001$). The prevalence of variceal bleeding progressively increased as serum sodium levels declined, being highest among patients with sodium <130 mmol/L and lowest among those with sodium >135 mmol/L, demonstrating that hyponatremia is linked with more severe portal hypertensive complications. Infection rates were significantly higher in patients with lower sodium levels ($\chi^2 = 29.64$, $p < 0.001$), with half of the patients having sodium <130 mmol/L developing infections compared to only 12 patients in the >135 mmol/L category, indicating that hyponatremia is associated with increased susceptibility to infective complications in cirrhosis. Serum sodium levels also showed a highly significant association with 28-day morbidity ($\chi^2 = 125.47$, $p < 0.001$), as all patients with sodium <130 mmol/L experienced morbidity, whereas only 21% of patients with sodium >135 mmol/L developed complications during follow-up. Likewise, a statistically significant association was observed between serum sodium levels and 28-day mortality ($\chi^2 = 13.87$, $p = 0.001$), with mortality rates being highest among severely hyponatremic patients and progressively decreasing with rising sodium levels, confirming that low serum sodium is an important predictor of poor short-term outcomes in liver cirrhosis. In contrast, no statistically significant association was observed between serum sodium levels and age distribution ($\chi^2 = 16.17$, $p = 0.09$), although most patients across all sodium categories belonged to the 41–60 years age group. Similarly, gender distribution did not differ significantly across sodium categories ($\chi^2 = 3.10$, $p = 0.21$), despite a predominance of male patients in all groups. These findings suggest that hyponatremia is more closely related to disease severity and complications rather than demographic characteristics such as age or gender. No significant association was found between serum sodium levels and diabetes mellitus, hypertension, or dyslipidemia ($p > 0.05$), as their distribution remained comparable across sodium categories. However, serum sodium showed a significant association with spontaneous bacterial peritonitis (SBP) ($\chi^2 = 24.08$, $p < 0.001$), with SBP occurring more frequently in patients with lower sodium levels, indicating a strong link between hyponatremia and SBP in liver cirrhosis patients **(Table 3)**.

A highly significant association was observed between serum sodium levels and hepatic encephalopathy grade ($\chi^2 = 378.13$, $p < 0.001$), with lower sodium levels linked to more severe encephalopathy. Patients with sodium <130 mmol/L predominantly had Grade 3–4 HE, whereas all patients with sodium >135 mmol/L were in Grade s0, indicating a strong relationship between hyponatremia and worsening hepatic encephalopathy severity **(Table 4)**. A significant association was found between serum sodium levels and hepatic hydrothorax ($\chi^2 = 33.52$, $p < 0.001$), with hydrothorax occurring more frequently in patients with lower sodium levels. Patients with sodium <130 mmol/L had the highest prevalence of hepatic hydrothorax, while those with sodium >135 mmol/L showed the lowest prevalence, indicating a strong association between hyponatremia and hepatic hydrothorax in liver cirrhosis patients **(Figure 3)**.

A highly significant difference in both Child-Pugh and MELD scores was observed across serum sodium categories ($p < 0.001$), with patients having sodium <130 mmol/L showing the highest mean scores and those with sodium >135 mmol/L showing the

lowest. These findings indicate a strong inverse relationship between serum sodium levels and liver disease severity, highlighting hyponatremia as a marker of advanced hepatic dysfunction **(Table 5)**.

A strong negative correlation was observed between serum sodium levels and liver disease severity scores. Serum sodium demonstrated a significant inverse relationship with both Child-Pugh score ($r = -0.84$, $p < 0.001$) and MELD score ($r = -0.81$, $p < 0.001$), indicating that lower sodium levels were associated with progressively worsening liver dysfunction and advanced cirrhosis severity. As serum sodium decreased, both Child-Pugh & MELD scores increased significantly, confirming hyponatremia as an important marker of hepatic decompensation. Receiver operating characteristic (ROC) analysis further evaluated the predictive value of serum sodium for 28-day mortality and demonstrated moderate discriminatory ability, with an area under the curve (AUC) of 0.67. The optimal serum sodium cut-off value for predicting short-term mortality was 131.50 mmol/L, which provided a sensitivity of 78% and specificity of 58%. These findings suggest that serum sodium has acceptable predictive performance for short-term mortality in patients with liver cirrhosis, with relatively higher sensitivity compared to specificity at the selected threshold.

Table 1: Patients with Complications

Complication	Number	%
Ascites	182	65.47
Hepatic Encephalopathy	108	38.85
Variceal Bleeding	62	22.3
SBP	45	16.19
Hepatorenal Syndrome	40	14.39
Coagulopathy	74	26.62

Table 2: Causative Factors of Liver Cirrhosis

Factor	Number	%
Alcoholic Liver Disease	125	44.96
Hepatitis B	51	18.35
Hepatitis C	34	12.23
NAFLD	45	16.19
Autoimmune Hepatitis	11	3.96
Cryptogenic	12	4.31
Total	278	100

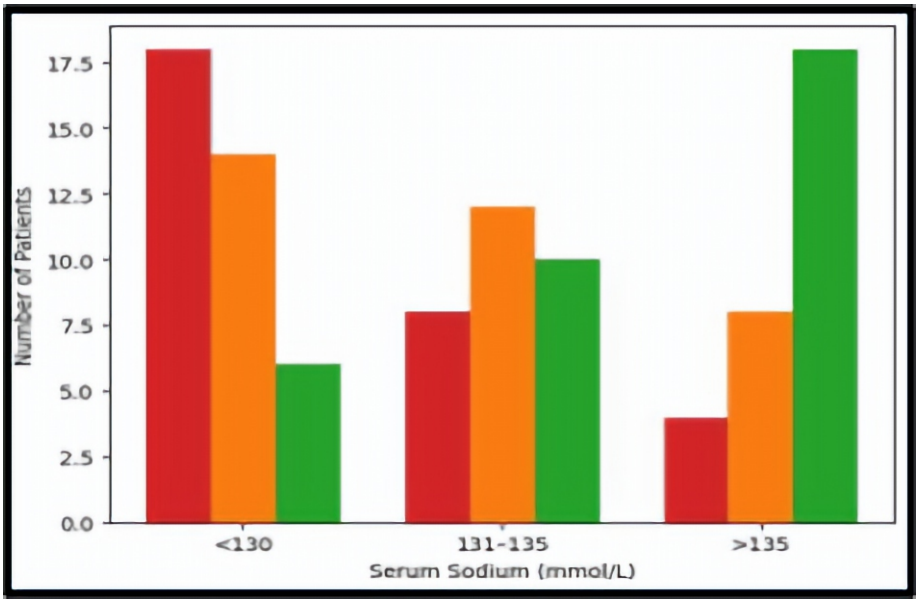

Figure 2: Frequency of Complications by Serum Sodium Level

Table 3: Association between Serum Sodium Levels and Clinical Comorbidities/Complications

Variable	Sodium (mmol/L)	No n (%)	Yes n (%)	Total	χ^2	p-value
	<130	60 (60%)	40 (40%)	100	rowspan="3"	rowspan="3"
Diabetes Mellitus	131–135	64 (64%)	36 (36%)	100	1.88	0.39
	>135	42 (53.8%)	36 (46.2%)	78		
	<130	50 (50%)	50 (50%)	100	rowspan="3"	rowspan="3"
Hypertension	131–135	55 (55%)	45 (45%)	100	5.09	0.08
	>135	52 (66.7%)	26 (33.3%)	78		
	<130	64 (64%)	36 (36%)	100	rowspan="3"	rowspan="3"
Dyslipidemia	131–135	67 (67%)	33 (33%)	100	0.23	0.89
	>135	52 (66.7%)	26 (33.3%)	78		
Spontaneous	<130	57 (57%)	43 (43%)	100	rowspan="3"	rowspan="3"
Bacterial	131–135	75 (75%)	25 (25%)	100	24.08	<0.001*
Peritonitis (SBP)	>135	70 (89.7%)	8 (10.3%)	78		

Table 4: Association: Sodium vs Hepatic Encephalopathy Grade

Sodium	Grade 0	Grade 1	Grade 2	Grade 3	Grade 4	Total	χ^2	p
<130	0	0	30	44	26	100		
131–135	0	37	41	22	0	100		
>135	78	0	0	0	0	78	378.13	<0.001*

Table 5: Mean Child-Pugh and MELD Scores Across Serum Sodium Categories

Parameter	Sodium (mmol/L)	N	Mean	SD	F-value	p-value
	<130	100	11.84	1.32	rowspan="3"	rowspan="3"
Child-Pugh Score	131–135	100	9.62	1.41	412.76	<0.001*
	>135	78	6.48	0.92		
	<130	100	26.72	4.38	rowspan="3"	rowspan="3"
MELD Score	131–135	100	19.94	3.86	356.41	<0.001*
	>135	78	12.83	2.91		

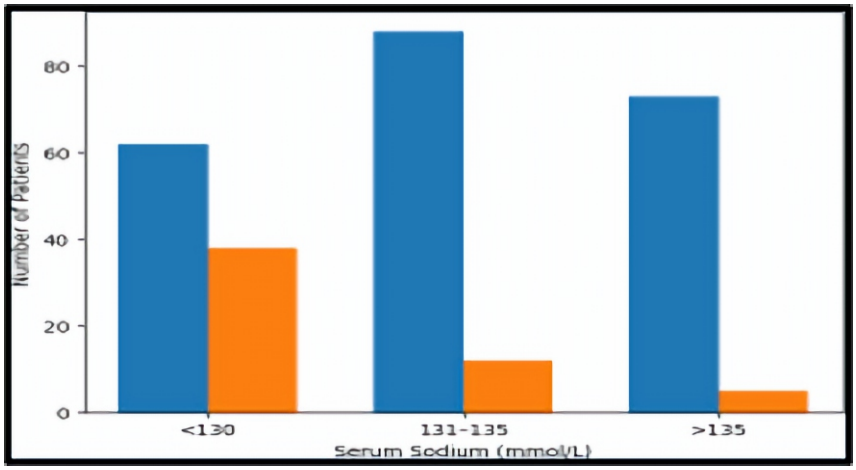

Figure 3: Association between Serum Sodium and Hepatic Hydrothorax

DISCUSSION

Chronic liver disease (CLD) is a progressive disorder characterized by continuous hepatic injury leading to fibrosis, cirrhosis, portal hypertension, and multisystem complications. As liver disease advances, disturbances in fluid and electrolyte balance become increasingly prominent, with hyponatremia emerging as the most common electrolyte abnormality in cirrhotic patients. Hyponatremia in cirrhosis is predominantly dilutional and results from impaired renal free water excretion due to systemic vasodilation, circulatory dysfunction, & persistent non-osmotic release of vasopressin.

These changes reflect advanced hepatic decompensation and are strongly associated with worsening clinical status and poor prognosis. The prevalence of hyponatremia in cirrhosis ranges from 20% to 60%, depending on patient population and diagnostic criteria, and is particularly common in patients with ascites and portal hypertension. Even mild reductions in serum sodium are clinically significant because they indicate early circulatory impairment and altered renal handling of water [10,13].

Ibrahim AA, et. al; 2017 and **Umemura T, et. al; 2015,** demonstrated a strong inverse relationship between serum

sodium levels and the severity of complications in chronic liver disease. Lower sodium concentrations are closely associated with major complications such as ascites, hepatic encephalopathy, spontaneous bacterial peritonitis (SBP), hepatorenal syndrome (HRS), and variceal bleeding. Hyponatremic patients generally exhibit higher Child-Pugh and MELD scores, confirming that serum sodium is an important marker of advanced hepatic dysfunction and systemic circulatory failure. Furthermore, hyponatremia contributes to renal hypoperfusion, impaired cerebral function, and increased susceptibility to infections, thereby worsening morbidity and mortality. Due to its strong prognostic significance, serum sodium has been incorporated into the MELD-Na scoring system used for risk stratification and liver transplant prioritization. Lower sodium levels are also associated with prolonged hospitalization, increased healthcare burden, and poorer survival outcomes, emphasized its importance in routine assessment and management of cirrhotic patients [14,15].

The present study demonstrated a highly significant inverse relationship between serum sodium levels and the severity of complications in chronic liver disease. Patients with severe hyponatremia (<130 mEq/L) had a substantially higher burden of severe complications compared to those with normal sodium levels, indicating that progressive decline in sodium parallels worsening hepatic decompensation. **Younas A, et. al; 2021,** studied 260 cirrhotic patients and observed a strong association between hyponatremia and hepatic encephalopathy severity, with most hyponatremic patients demonstrating advanced neurological dysfunction. **Li X-J & Meng H-H. 2024,** reported that cirrhotic patients with hyponatremia had significantly higher frequencies of hepatic encephalopathy, renal impairment, and infections, confirmed that low serum sodium identifies patients with advanced systemic decompensation and poor prognosis [16,17]. Ascites was the most common complication observed in the present study, followed by hepatic encephalopathy, coagulopathy, variceal bleeding, spontaneous bacterial peritonitis, and hepatorenal syndrome. Alcoholic liver disease emerged as the predominant etiology, followed by hepatitis B and non-alcoholic fatty liver disease, findings that closely resemble the Indian meta-analysis by **Swaroop S, et. al; 2024,** which identified alcohol as the leading cause of cirrhosis in India. Patients with serum sodium <130 mEq/L showed significantly higher rates of ascites and hepatorenal syndrome, supporting the concept that worsening hyponatremia reflects progressive circulatory dysfunction and renal compromise. **NAREDDY SR, et. al; 2020,** found significant associations between hyponatremia, severe Child-Pugh class, spontaneous bacterial peritonitis, and hepatorenal syndrome in cirrhotic patients [18,12]. The present study also demonstrated significant associations between hyponatremia and variceal bleeding, infection, hepatic encephalopathy, and hepatic hydrothorax. Infection rates increased markedly as serum sodium declined, indicating that hyponatremia is linked to severe infective decompensation and immune dysfunction.

Younas A, et. al; 2021, reported that lower sodium levels strongly correlated with higher grades of hepatic encephalopathy, while **Barakat AAE-K, et. al; 2015,** observed increased frequencies of pleural effusion, renal failure, and encephalopathy among hyponatremic cirrhotic patients. Both study further demonstrated that hepatic hydrothorax was associated with higher MELD-Na scores and significantly increased mortality, supported the role of hyponatremia as a marker of advanced disease [19,16].

A strong inverse relationship was also identified between serum sodium and both Child-Pugh and MELD scores. Patients with severe hyponatremia had significantly higher mean Child-Pugh and MELD scores, indicating advanced hepatic dysfunction. **Pawar T, et. al; 2025** and **Thuluvath PJ, et. al; 2022,** reported that low serum sodium was strongly associated with Child-Pugh class C disease, elevated MELD scores, and reduced survival (83,84). Correlation analysis in the present study further confirmed significant negative correlations between serum sodium and Child-Pugh as well as MELD scores, reinforcing the value of serum sodium as a reliable marker of disease severity. Additionally, serum sodium demonstrated significant predictive value for 28-day morbidity and mortality, with patients having sodium <130 mEq/L exhibiting markedly worse short-term outcomes. **Pawar T, et. al; 2025** and **Thuluvath PJ, et. al; 2022,** reported significantly higher mortality, renal failure, sepsis, and adverse clinical outcomes among hyponatremic cirrhotic patients [20,21].

Importantly, the study found no significant association between serum sodium and demographic factors such as age and gender or comorbid conditions including diabetes, hypertension, and dyslipidemia. This suggests that hyponatremia primarily reflects the severity of hepatic decompensation rather than background demographic or metabolic characteristics. **Barakat AAE-K, et. al; 2015**, **Naim H. 2021,** and **Islam S, et. al; 2022,** demonstrated that serum sodium is an independent prognostic and severity marker in chronic liver disease. Overall, the findings strongly supported serum sodium as a simple, cost-effective, and clinically valuable biomarker for assessing disease severity, predicting complications, and determining prognosis in patients with chronic liver disease [19,22,23].

CONCLUSION

The study population mainly comprised middle-aged and elderly males, with alcoholic liver disease identified as the most common cause. A significant inverse relationship was observed between serum sodium levels and the severity of major cirrhosis-related complications, including ascites, hepatic encephalopathy, hepatorenal syndrome, variceal bleeding, and spontaneous bacterial peritonitis. Lower sodium levels were associated with higher Child-Pugh and MELD scores, reflecting advanced liver dysfunction. Severe hyponatremia (serum sodium <130 mmol/L) was linked to markedly increased 28-day morbidity and mortality. Although no significant association was found with age, gender, diabetes, or hypertension, serum

sodium remained a strong prognostic marker for chronic liver disease severity and outcomes.

LIMITATIONS & FUTURE PERSPECTIVES

The study was limited by its single-centre design, relatively small sample size, and short duration, which may restrict generalizability. Future research could focus on multicenter studies with larger cohorts to validate findings, evaluate long-term outcomes, and explore innovative diagnostic and management strategies for appendicular perforation, improving patient prognosis and reducing complications.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical healthcare applications. It emphasizes the importance of translating scientific observations into meaningful improvements in patient care, diagnosis, and treatment outcomes. By highlighting realworld relevance, the study contributes to evidence-based medical practice and supports informed clinical decision-making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

CLD: Chronic Liver Disease
MELD: Model for End-Stage Liver Disease
SBP: Spontaneous Bacterial Peritonitis
ROC: Receiver Operating Characteristic
mEq/L: Milliequivalents per Liter

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AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of Medical Journal Editors (ICMJE) guidelines.

ACKNOWLEDGEMENT

The authors sincerely acknowledge the seniors of the Department of General Medicine, Tezpur Medical College & Hospital, Tezpur, Assam, India. We are grateful to our college for providing the necessary resources to carry out this work. We also extend our heartfelt thanks to our colleagues and technical staff for their valuable assistance during the study.

CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

FUNDING

None

ETHICAL APPROVAL & CONSENT TO PARTICIPATE

All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION

All necessary consent for publication was obtained by authors.

DATA AVAILABILITY

All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a reasonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

The authors confirm that no AI & LLM tools were used in the writing or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

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