e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2025; 17(10); 574-580

Original Research Article

Serum Sodium Level in Decompensated Chronic Liver Disease and Its Relationship with Outcome: A Cross-Sectional Study

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Received: 01-07-2025 Revised: 15-08-2025 / Accepted: 21-09-2025
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Conflict of interest: Nil

Abstract

Background: Chronic liver disease (CLD) remains a major cause of morbidity and mortality worldwide, particularly in developing countries. Among the many biochemical derangements associated with decompensated chronic liver disease (DCLD), hyponatraemia is a frequent electrolyte disturbance and has been associated with poor prognosis and increased mortality.

Objectives: To estimate the proportion of deranged serum sodium levels in patients with DCLD and to determine the relationship between serum sodium and clinical outcomes among hospitalised patients.

Methods: A descriptive, longitudinal study was conducted among 100 consecutive patients with DCLD admitted to the Department of General Medicine, Agartala Government Medical College and GBP Hospital, Tripura. Patients above 18 years were included after informed consent. Serum sodium levels, biochemical parameters, and disease severity scores (Child–Pugh and MELD) were assessed. Patients were categorised as having hyponatraemia (<130 mEq/L), normonatraemia (131–135 mEq/L), or hypernatraemia (≥136 mEq/L). Data were analysed using SPSS version 22; $p < 0.05$ was considered statistically significant.

Results: Of 100 patients, 83% were male. The mean age was 51.1 ± 9.5 years. Hyponatraemia was observed in 29%, normonatraemia in 44%, and hypernatraemia in 27%. Alcoholic liver disease was the most common aetiology (47%). Hyponatraemia was significantly associated with higher Child–Pugh and MELD scores, ascites, coagulopathy, hepatic encephalopathy, hepatorenal syndrome (HRS), and increased mortality ($p < 0.05$).

Conclusion: Hyponatraemia is common among patients with DCLD and is strongly associated with disease severity and mortality. Serum sodium measurement serves as a simple, cost-effective prognostic marker for clinical outcome in DCLD patients.

Keywords: Hyponatraemia, Chronic Liver Disease, Decompensated Cirrhosis, Serum Sodium, MELD Score, Child–Pugh Score.

Introduction

Chronic liver disease (CLD) is defined as the progressive and irreversible destruction of hepatic parenchyma lasting for more than six months, culminating in fibrosis and cirrhosis of the liver [1]. Cirrhosis represents the final common pathway of a variety of chronic hepatic insults and is characterised by distortion of hepatic architecture, nodule formation, and portal hypertension [2].

The disease burden of CLD in India is rising, with an estimated 18.3% contribution to global liver-related deaths [3]. Decompensated chronic liver disease (DCLD) manifests with ascites, hepatic encephalopathy, jaundice, upper gastrointestinal (UGI) bleeding, hepatorenal syndrome, and coagulopathy [4]. Among the complications, hyponatraemia — defined as a serum sodium concentration below 135 mmol/L — is one of the most frequent electrolyte abnormalities observed in cirrhotic patients [5].

The pathophysiology of hyponatraemia in cirrhosis involves splanchnic vasodilatation, arterial under filling, and non-osmotic release of arginine vasopressin (ADH), leading to impaired renal water excretion [6,7]. Chronic hyponatraemia may contribute to worsening hepatic encephalopathy, ascites, and renal dysfunction [8,9]. Recent studies

suggest that serum sodium level is an independent predictor of mortality in cirrhosis and its inclusion in the MELD-Na score improves prognostic accuracy [10,11]. Despite increasing evidence, data from north-eastern India remain limited. This study was therefore undertaken to assess serum sodium levels in patients with DCLD and examine their association with clinical outcomes.

Review of Literature: The prevalence of chronic liver disease in India has been steadily increasing due to rising alcohol consumption, non-alcoholic fatty liver disease (NAFLD), and viral hepatitis [3,12]. Cirrhosis is the end stage of most chronic liver disorders and is characterised by fibrotic replacement of normal hepatic tissue leading to portal hypertension [13].

Decompensated cirrhosis is associated with complications such as ascites, spontaneous bacterial peritonitis (SBP), variceal haemorrhage, hepatic encephalopathy, and hepatorenal syndrome [14]. Each of these complications significantly worsens survival and quality of life [15].

Hyponatraemia in cirrhosis can be classified as hypovolaemic, hypervolaemic, or dilutional [16]. Dilutional hyponatraemia is most common, occurring due to impaired water excretion rather than sodium loss [17]. Splanchnic vasodilatation leads to activation of the renin–angiotensin–aldosterone system and non-osmotic release of ADH, causing disproportionate water retention [18].

Numerous studies have established the prognostic importance of serum sodium levels in cirrhosis. Biggins et al [19] demonstrated that inclusion of serum sodium in the MELD score improves prediction of short-term mortality in transplant candidates. Similarly, Kim et al [20] found that patients with serum sodium ≤130 mEq/L had higher mortality and more frequent complications.

Hyponatraemia has been linked to increased risks of hepatic encephalopathy, ascites refractory to diuretics, hepatorenal syndrome, and prolonged hospitalisation [21,22]. Hence, serum sodium measurement is now recommended as a routine prognostic marker in DCLD management.

Materials and Methods

Study Design and Setting: A descriptive, longitudinal study was conducted at the Department of General Medicine, Agartala Government Medical College and Govind Ballabh Pant (GBP) Hospital, Tripura, over a period of 18 months.

Study Population: All adult patients (≥18 years) admitted with decompensated chronic liver disease were enrolled consecutively after obtaining informed consent.

Inclusion Criteria

- Patients diagnosed with DCLD based on clinical, biochemical, and radiological findings.

Exclusion Criteria

- Patients on medications affecting sodium balance (SSRIs, TCAs, MAO inhibitors, cytotoxic drugs).
- Those unwilling to participate.

Sample Size: Sample size was calculated using the formula for single proportion:

$N = Z^2 \times P(1–P)/L^2$

Using prevalence (P) = 45.5%, allowable error (L) = 10%, and Z = 1.96, the sample size was 95, rounded to 100.

Data Collection: A predesigned proforma was used to record demographic data, clinical features, laboratory findings, aetiology, and outcome parameters.

Laboratory investigations included complete blood count, liver and renal function tests, coagulation profile, and serum sodium. Disease severity was assessed using Child–Pugh and MELD scores.

Serum sodium levels were categorised as follows:

- **Hyponatraemia:** <130 mEq/L
- **Normonatraemia:** 131–135 mEq/L
- **Hypernatraemia:** ≥136 mEq/L

Outcomes assessed included recovery or death during hospitalisation.

Ethical Considerations: Ethical approval was obtained from the Institutional Ethics Committee of AGMC and GBP Hospital. Written informed consent was taken from all participants or their guardians.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using IBM SPSS version 22. Quantitative data were summarised as mean ± SD, while qualitative data were expressed as proportions. Associations were assessed using chi-square and ANOVA tests. A p-value <0.05 was considered statistically significant.

Results

Demographic Characteristics

Table 1: Distribution of patients by age group (n = 100)

Age group (years)	Frequency	Percentage (%)
30–39	11	11
40–49	37	37
50–59	31	31
60–69	21	21
Total	**100**	**100**

Mean (±SD) age: 51.1 ± 9.5 years

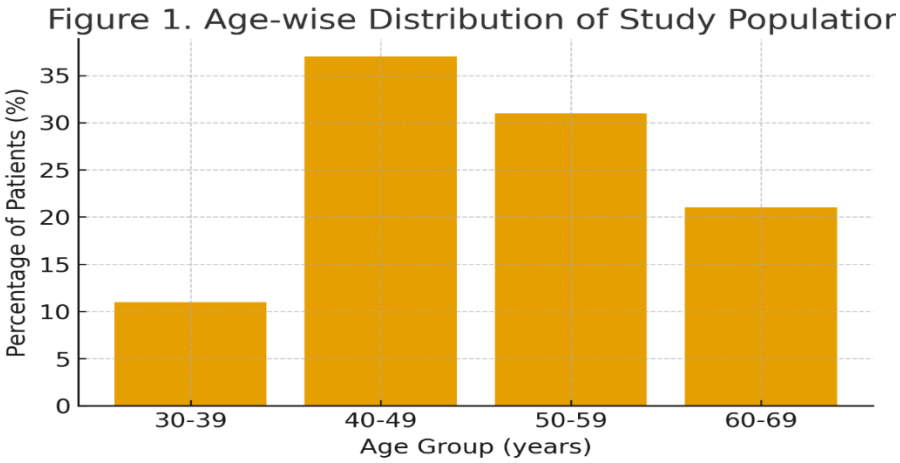

Figure 1: Bar chart showing age-wise distribution of study population

Males predominated (83%), giving a male-to-female ratio of approximately 5:1.

Clinical Profile

Table 2: Distribution of symptoms and signs (n = 100)

Clinical Feature	Present (%)
Abdominal distension	100
Lower limb swelling	95
Jaundice	55
Bleeding (haematemesis/melaena)	24
Altered sensorium	26
Ascites (mild/tense)	82/18
Hepatic encephalopathy (Grades 1–2 / 3–4)	23 / 3
Hepatorenal syndrome	13

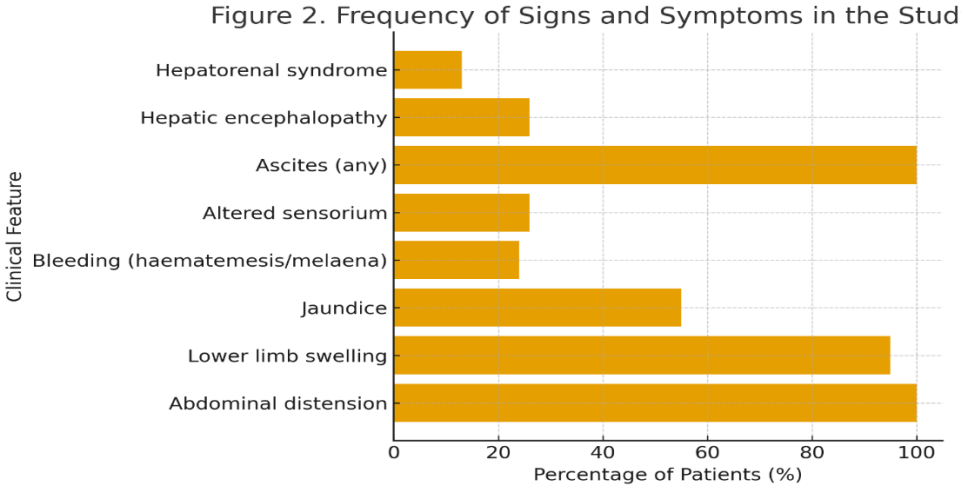

Figure 2: Frequency of signs and symptoms

Laboratory Findings

Table 3: Mean laboratory parameters (n = 100)

Parameter	Mean	SD
Urea (mg/dL)	39.7	15.4
Creatinine (mg/dL)	1.2	0.6
Total bilirubin (mg/dL)	3.8	2.4
Serum albumin (g/dL)	2.9	0.5
ALT (IU/L)	66.2	32.0
AST (IU/L)	72.1	30.2
INR	1.4	0.3

Severity Scores: The mean MELD score was 15.1 ± 5.9, while the mean Child–Pugh score was 9.4 ± 1.9.

Table 4: Child–Pugh classification (n = 100)

Class	Frequency	Percentage (%)
A	9	9
B	51	51
C	40	40

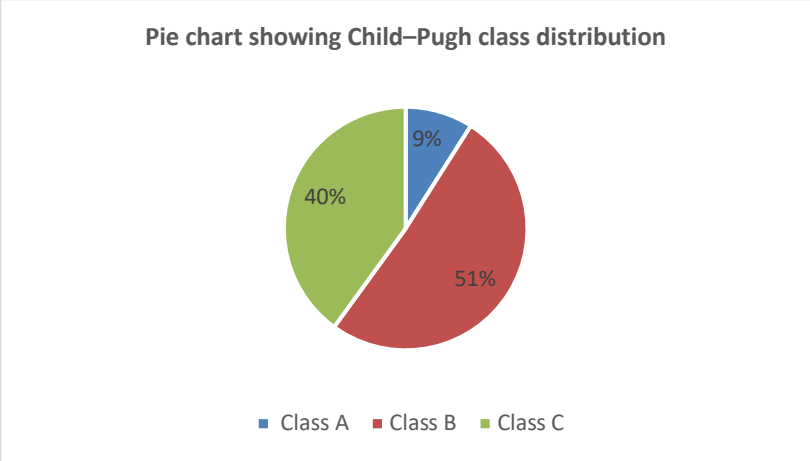

Figure 3. Pie chart showing Child–Pugh class distribution (placeholder)

Serum Sodium Status

Table 5: Serum sodium distribution (n = 100)

Serum Sodium (mEq/L)	Frequency	Percentage (%)
<130	29	29
131–135	44	44
≥136	27	27

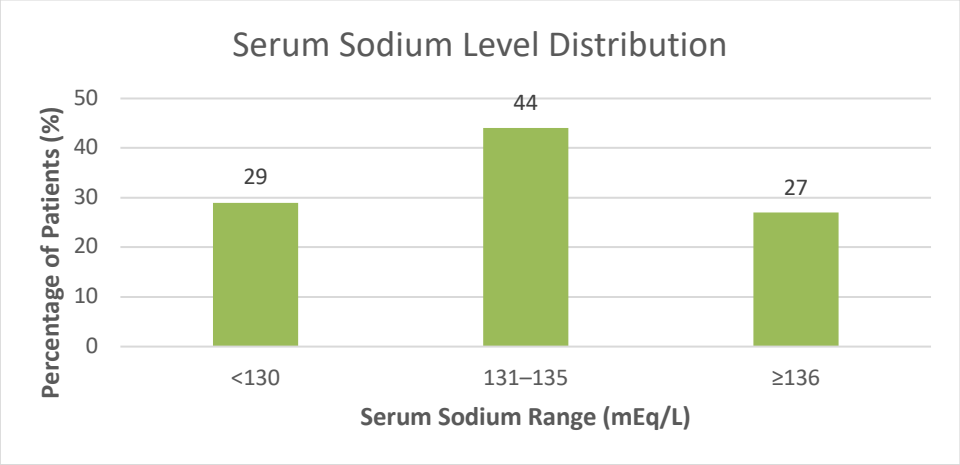

Figure 4: Serum sodium level distribution

Hyponatraemia was observed in nearly one-third of patients, while 27% exhibited hypernatraemia.

Aetiology of Liver Disease: Alcoholic liver disease was the predominant cause (47%), followed by NASH (24%), cryptogenic (9%), and viral hepatitis (8%). Other less frequent causes included autoimmune hepatitis, Budd–Chiari syndrome, and Wilson's disease.

Serum Sodium and Disease Severity

Table 6: Association between serum sodium and Child–Pugh class

Sodium (mEq/L)	Class A	Class B	Class C	p-value
<130	1 (3.4%)	11 (37.9%)	17 (58.6%)	0.001*
131–135	0	24 (54.5%)	20 (45.5%)	
≥136	8 (29.6%)	16 (59.3%)	3 (11.1%)	

***Statistically significant**

Patients with hyponatraemia were significantly more likely to belong to Child–Pugh class C.

Serum Sodium and Mortality

Table 7: Mortality across sodium categories

Sodium (mEq/L)	Survivors	Deaths	p-value
<130	21 (72.4%)	8 (27.6%)	0.001*
131–135	41 (93.2%)	3 (6.8%)	
≥136	27 (100%)	0	

***Statistically significant**

Mortality was highest among patients with serum sodium <130 mEq/L.

Association with Complications

Table 8: Complications according to serum sodium

Complication	<130 mEq/L (%)	131–135 mEq/L (%)	≥136 mEq/L (%)	p-value
Ascites (tense)	34.5	15.9	3.7	0.010*
Coagulopathy	44.8	20.5	7.4	0.004*
Hepatorenal Syndrome	41.1	2.3	0	0.000*
Hepatic Encephalopathy	31.0	29.5	14.8	0.388
SBP	10.3	13.6	14.8	0.871

***Statistically significant**

Hyponatraemia was associated with a significantly higher incidence of ascites, coagulopathy, and hepatorenal syndrome.

Discussion

Hyponatraemia is one of the most frequent electrolyte abnormalities in DCLD and reflects advanced portal hypertension and systemic vasodilatation [23,24]. In the present study, 29% of patients exhibited serum sodium <130 mEq/L, consistent with studies reporting prevalence rates between 25% and 40% [25,26]. The mean age of the study cohort (51.1 years) and the male predominance (83%) were comparable to findings by Senthilpriyan [27] and Devadas et al [28]. Alcohol was the leading cause of DCLD, similar to national trends showing alcohol-related cirrhosis as the most prevalent aetiology in India29.

In this study, patients with hyponatraemia had significantly higher MELD and Child–Pugh scores, suggesting a direct correlation between low sodium and advanced disease. This agrees with studies by Kim et al [20] and Moogambiga [30], who reported that patients with sodium <130 mEq/L were more likely to belong to Child–Pugh class C. Hyponatraemia exacerbates complications of cirrhosis. Dilutional hyponatraemia leads to cerebral oedema and may precipitate hepatic encephalopathy [31]. Although a higher proportion of encephalopathy was seen among hyponatraemic patients in the present study, the difference was not statistically significant, echoing observations by Qureshi et al [32].

A significant association was found between hyponatraemia and coagulopathy, ascites, and hepatorenal syndrome. Sodium imbalance increases neurohormonal activation, resulting in renal vasoconstriction and impaired perfusion, contributing to hepatorenal syndrome [33]. Angeli et al [9] and Shaikh et al [34] also reported that low sodium levels correlated with greater incidence of HRS and refractory ascites. Mortality was notably higher among hyponatraemic patients (27.6%) compared with those having normal or elevated

sodium (p = 0.001). This aligns with studies by Biggins et al [19] and Borroni et al [35] who demonstrated that serum sodium <130 mEq/L independently predicted mortality. Thus, serum sodium acts as a surrogate marker for disease severity and outcome in cirrhosis. Incorporation of sodium into MELD-Na improves its predictive value, particularly in patients awaiting transplantation [10,36].

Conclusion

Hyponatraemia is a common finding in decompensated chronic liver disease and is strongly associated with higher disease severity, complications, and in-hospital mortality. Measurement of serum sodium is simple, inexpensive, and provides valuable prognostic information.

Key findings:

- 29% of patients had hyponatraemia (<130 mEq/L).
- Hyponatraemia correlated significantly with ascites, coagulopathy, hepatorenal syndrome, and mortality.
- Serum sodium inversely correlated with MELD and Child–Pugh scores.

Routine monitoring of serum sodium in cirrhotic patients is recommended for timely identification of high-risk individuals and improved clinical management.

Limitations

1. The study was conducted at a single centre with a relatively small sample size.
2. Other potential causes of hyponatraemia (e.g., renal or cardiac dysfunction) may have influenced results.
3. The study did not assess long-term survival beyond hospital discharge.
4. Generalisability to other populations requires multicentric validation.

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