



Original Article

Association of Serum Sodium Levels with Disease Severity, Clinical Complications, and Prognostic Scores in Patients with Chronic Liver Disease: A Hospital-Based Cross-Sectional Study

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ABSTRACT

Background: Chronic liver disease (CLD) is a major global health problem and a leading cause of morbidity and mortality, particularly in low- and middle-income countries such as India. Progressive hepatic fibrosis and cirrhosis result in portal hypertension, impaired hepatic synthetic function, and multiple systemic complications. Hyponatremia, defined as a serum sodium concentration of ≤ 135 mEq/L, is one of the most common electrolyte abnormalities encountered in patients with advanced CLD. It develops primarily due to non-osmotic release of arginine vasopressin, leading to impaired renal free water excretion and dilutional hyponatremia. Low serum sodium has been associated with severe complications including ascites, hepatic encephalopathy, spontaneous bacterial peritonitis, hepatorenal syndrome, and increased mortality. Although serum sodium has been incorporated into prognostic models such as the MELD-Na score, regional data evaluating its association with disease severity remain limited. Therefore, the present study was undertaken to assess the relationship between serum sodium levels and the severity of chronic liver disease in patients attending a tertiary care centre.

Aim: To evaluate the association between serum sodium levels and the severity of chronic liver disease and its related complications in patients attending a tertiary care hospital.

Materials and Methods: A hospital-based cross-sectional observational study was conducted in the Department of General Medicine, Gandhi Medical College and Hospital, Secunderabad, Telangana, over a period of 13 months from September 2022 to October 2023. A total of 100 patients aged 30–50 years with clinically, biochemically, and radiologically confirmed chronic liver disease were enrolled after obtaining informed consent. Patients with hepatocellular carcinoma, heart failure, hepatorenal syndrome at presentation, other major organ failure, and those receiving drugs known to affect serum sodium were excluded. Detailed demographic and clinical data were recorded. Laboratory investigations included liver function tests, renal function tests, coagulation profile, complete blood count, viral markers, and serum sodium estimation. Disease severity was assessed using Child–Pugh classification and MELD score. Statistical analysis was performed using SPSS version 26.0. A p-value < 0.05 was considered statistically significant.

Results: Among the 100 study participants, the mean age was 40.01 ± 6.01 years, with a male predominance (70%). Alcohol-related liver disease was the most common etiology (62%), followed by hepatitis B virus infection (22%). Hyponatremia (serum sodium ≤ 135 mEq/L) was observed in 46% of patients, while 54% had normal serum sodium levels. A significant inverse association was

observed between serum sodium levels and disease severity. Patients with advanced Child–Pugh class and higher MELD scores had significantly lower serum sodium concentrations ($p<0.001$). Hyponatremia was also significantly associated with complications such as ascites, hepatic encephalopathy, esophageal varices, spontaneous bacterial peritonitis, and hepatorenal syndrome. Patients with severe hyponatremia demonstrated substantially higher in-hospital mortality than those with normal serum sodium levels.

Conclusion: Serum sodium is an important, inexpensive, and readily available biochemical marker that correlates closely with the severity and prognosis of chronic liver disease. Lower serum sodium levels are associated with advanced cirrhosis, increased frequency of complications, and poorer clinical outcomes. Routine assessment of serum sodium should be incorporated into the evaluation and prognostic stratification of all patients with chronic liver disease, alongside established scoring systems such as Child–Pugh and MELD, to facilitate early identification of high-risk patients and improve clinical management.

Keywords: *Chronic liver disease, Cirrhosis, Hyponatremia, Serum sodium, Child–Pugh score, MELD score, Hepatic encephalopathy, Ascites, Prognosis, Liver cirrhosis.*

INTRODUCTION

Chronic liver disease (CLD) is a major global health problem and represents one of the leading causes of morbidity and mortality worldwide. It encompasses a broad spectrum of progressive liver disorders characterized by persistent hepatic inflammation, fibrosis, distortion of normal liver architecture, and ultimately cirrhosis with its associated complications. CLD results from chronic injury to hepatocytes caused by viral infections, excessive alcohol consumption, non-alcoholic fatty liver disease (NAFLD), autoimmune liver diseases, inherited metabolic disorders, and chronic exposure to hepatotoxic agents. Progressive fibrosis eventually culminates in cirrhosis, portal hypertension, hepatic insufficiency, and liver failure if left untreated. The burden of CLD has increased substantially over the last two decades, largely due to the rising prevalence of metabolic syndrome and alcohol-related liver disease worldwide. [1,2]

According to the Global Burden of Disease Study, chronic liver disease accounts for more than two million deaths annually worldwide, representing approximately 4% of all global deaths. Liver cirrhosis is among the top ten causes of mortality in adults and contributes significantly to disability-adjusted life years (DALYs), particularly in low- and middle-income countries. India bears a substantial burden of chronic liver disease owing to the high prevalence of hepatitis B virus infection, hepatitis C virus infection, alcohol abuse, and the rapidly increasing incidence of metabolic dysfunction-associated steatotic liver disease (MASLD). [3–5]

The liver performs numerous essential physiological functions, including carbohydrate, protein and lipid metabolism, synthesis of plasma proteins and coagulation factors, detoxification of endogenous and exogenous substances, immune regulation, and maintenance of electrolyte and fluid homeostasis. Progressive hepatic dysfunction profoundly affects these physiological processes and predisposes patients to multisystem complications. As liver disease advances, portal hypertension develops due to increased intrahepatic vascular resistance and increased portal blood flow. Portal hypertension is responsible for many of the clinical manifestations of cirrhosis, including ascites, esophageal varices, splenomegaly, spontaneous bacterial peritonitis, hepatic encephalopathy, and hepatorenal syndrome. [6,7]

One of the most common biochemical abnormalities observed in patients with advanced cirrhosis is hyponatremia. Hyponatremia is generally defined as a serum sodium concentration of ≤ 135 mEq/L and represents dilutional rather than depletion hyponatremia in the majority of cirrhotic patients. The development of hyponatremia reflects severe circulatory dysfunction and impaired renal water excretion resulting from progressive portal hypertension and systemic vasodilatation. Numerous studies have demonstrated that hyponatremia is associated with poor prognosis, prolonged hospitalization, increased incidence of complications, and higher mortality among patients with chronic liver disease. [8–10]

The pathophysiology of hyponatremia in cirrhosis is complex and multifactorial. Progressive portal hypertension causes marked splanchnic arterial vasodilatation mediated primarily by increased nitric oxide production. Despite increased total body fluid volume, the effective arterial blood volume decreases significantly. This reduction activates several neurohumoral compensatory mechanisms, including the renin–angiotensin–aldosterone system (RAAS), sympathetic nervous system, and non-osmotic secretion of arginine vasopressin (antidiuretic hormone). Excessive vasopressin promotes water reabsorption through aquaporin-2 channels in the renal collecting ducts, leading to disproportionate water retention relative to sodium retention and ultimately resulting in dilutional hyponatremia. [11–13]

Hyponatremia is not merely a laboratory abnormality but a clinically significant predictor of adverse outcomes in chronic liver disease. Reduced serum sodium levels are strongly associated with refractory ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, hepatorenal syndrome, acute-on-chronic liver failure, increased intensive care admission, prolonged hospital stay, and poor transplant-free survival. Furthermore, severe hyponatremia has been shown to increase neurological complications during liver transplantation due to rapid osmotic shifts. Consequently, serum sodium has emerged as an important prognostic biomarker in cirrhosis. [14–16]

Several prognostic scoring systems are currently used to assess disease severity and predict mortality in patients with chronic liver disease. The Child–Pugh classification incorporates serum bilirubin, serum albumin, prothrombin time/INR, ascites, and hepatic encephalopathy to categorize patients into Child–Pugh Classes A, B, and C. The Model for End-stage Liver Disease (MELD) score, which includes serum bilirubin, creatinine, and INR, has become the standard prognostic tool for liver transplant allocation. Subsequently, incorporation of serum sodium into the MELD score resulted in the MELD-Na score, which provides superior prediction of short-term mortality compared with MELD alone and is currently recommended by major international hepatology societies. [17–19]

Measurement of serum sodium is inexpensive, readily available, reproducible, and routinely performed in all tertiary healthcare settings. Despite its simplicity, serum sodium provides valuable prognostic information regarding disease severity and impending clinical deterioration. Early identification of hyponatremia enables clinicians to optimize fluid management, avoid inappropriate diuretic therapy, identify patients requiring closer monitoring, and prioritize liver transplantation when indicated. In resource-limited settings such as India, where advanced diagnostic modalities may not always be available, serum sodium estimation represents a practical and cost-effective biomarker for risk stratification.

However, regional data evaluating the association between serum sodium levels and severity of chronic liver disease remain limited. Therefore, the present study was undertaken to evaluate the association between serum sodium concentration and disease severity, prognostic scores, and major complications among patients with chronic liver disease attending a tertiary care teaching hospital. [20]

MATERIALS AND METHODS

Study Design

The present study was a **hospital-based observational cross-sectional study** conducted to evaluate the association between serum sodium levels and the severity of chronic liver disease (CLD). The study was designed to determine the relationship between serum sodium concentration, disease severity, clinical complications, and prognostic scoring systems in patients diagnosed with chronic liver disease.

Study Setting

The study was carried out in the **Department of General Medicine, Gandhi Medical College and Hospital, Secunderabad, Telangana, India**, a tertiary care teaching hospital catering to patients from urban and rural areas of Telangana and neighboring states. The hospital provides specialized diagnostic and therapeutic facilities for patients with chronic liver disease and its complications.

Study Period

The study was conducted over a period of **13 months, from September 2022 to October 2023**.

Study Population

The study population comprised adult patients diagnosed with chronic liver disease attending the outpatient department or admitted to the medical wards with complications of liver cirrhosis during the study period. Diagnosis was established on the basis of clinical history, physical examination, biochemical investigations, and radiological findings.

Sample Size

A total of **100 patients** fulfilling the eligibility criteria were included in the study using a consecutive sampling technique.

Sampling Technique

A **consecutive non-probability sampling method** was employed. All eligible patients presenting during the study period were enrolled after obtaining written informed consent until the required sample size was achieved.

Inclusion Criteria

The following patients were included in the study:

1. Patients aged **30–50 years** of either gender.
2. Patients diagnosed with chronic liver disease or liver cirrhosis based on clinical, biochemical, and radiological findings.

3. Patients attending the outpatient department or admitted with complications of liver cirrhosis.
4. Patients with hepatitis B virus-associated chronic liver disease.
5. Patients willing to participate and providing written informed consent.

Exclusion Criteria

The following patients were excluded:

1. Patients with hepatocellular carcinoma.
2. Patients with any other malignancy.
3. Patients with severe cardiac failure.
4. Patients with other major organ failure.
5. Patients with hepatorenal syndrome at presentation.
6. Patients receiving diuretics, selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants (TCAs), monoamine oxidase inhibitors (MAO inhibitors), cytotoxic drugs, or other medications known to alter serum sodium concentration.
7. Pregnant women.
8. Patients unwilling to participate in the study.

Data Collection

After obtaining approval from the Institutional Ethics Committee and written informed consent, detailed demographic and clinical information was collected using a predesigned case record form. Information regarding age, gender, alcohol consumption, viral hepatitis status, presenting symptoms, duration of illness, previous hospitalization, and associated comorbidities was recorded.

A comprehensive clinical examination was performed, including assessment for jaundice, pallor, pedal edema, ascites, splenomegaly, hepatomegaly, hepatic encephalopathy, gastrointestinal bleeding, and other complications of chronic liver disease.

Laboratory Investigations

The following investigations were performed for all enrolled patients:

- Complete blood count (CBC)
- Liver function tests (bilirubin, AST, ALT, ALP, serum albumin)
- Renal function tests (blood urea and serum creatinine)
- Serum electrolytes (sodium, potassium, chloride)
- Prothrombin time and International Normalized Ratio (INR)
- Viral markers (HBsAg and anti-HCV)
- Random blood glucose
- Ultrasonography of the abdomen
- Upper gastrointestinal endoscopy where indicated for esophageal varices

Serum sodium concentration was measured using an automated electrolyte analyzer based on the ion-selective electrode method.

Assessment of Disease Severity

Disease severity was assessed using validated prognostic scoring systems:

- **Child–Pugh Classification:** Patients were categorized into Child–Pugh Class A, B, or C based on serum bilirubin, serum albumin, INR/prothrombin time, ascites, and hepatic encephalopathy.
- **Model for End-stage Liver Disease (MELD) Score:** MELD score was calculated using serum bilirubin, serum creatinine, and INR to estimate disease severity and prognosis.

Outcome Measures

The primary outcome was the association between serum sodium levels and the severity of chronic liver disease.

Secondary outcomes included:

- Association between serum sodium and Child–Pugh class.
- Association between serum sodium and MELD score.
- Association between serum sodium and complications such as ascites, esophageal varices, hepatic encephalopathy, spontaneous bacterial peritonitis, and hepatorenal syndrome.
- Association between serum sodium levels and in-hospital mortality.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using **Statistical Package for the Social Sciences (SPSS) version 26.0**. Continuous variables were expressed as **mean $\pm$ standard deviation (SD)**, while categorical variables were presented as frequencies and percentages.

Comparisons between groups were performed using the **Student's t-test** or **one-way analysis of variance (ANOVA)** for continuous variables and the **Chi-square test** or **Fisher's exact test** for categorical variables, wherever appropriate. Correlation between serum sodium levels and disease severity scores was assessed using **Pearson's correlation coefficient**. A **p-value <0.05** was considered statistically significant.

Ethical Considerations

The study protocol was approved by the **Institutional Ethics Committee of Gandhi Medical College and Hospital, Secunderabad** before commencement of the study. Written informed consent was obtained from all participants. Confidentiality of patient information was maintained throughout the study, and all procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki.

RESULTS

Table 1: Age-wise Distribution of Study Participants

Table 1 depicts the age distribution of the study participants diagnosed with chronic liver disease. A total of 100 patients were enrolled in the study. The majority of the patients belonged to the **41–50 years age group**, followed by the **30–40 years** age group. The mean age of the study population was **40.01 $\pm$ 6.01 years**, indicating that chronic liver disease predominantly affected middle-aged adults. This observation suggests that prolonged exposure to risk factors such as chronic alcohol consumption, viral hepatitis, metabolic syndrome, and unhealthy lifestyle habits contributes significantly to the development of chronic liver disease during the most productive years of life. The relatively younger age of presentation observed in the present study reflects the increasing burden of alcohol-related liver disease and non-alcoholic fatty liver disease in developing countries. Early diagnosis and timely intervention in this age group may reduce disease progression and improve long-term outcomes.

TABLE 1: AGE DISTRIBUTION OF STUDY PARTICIPANTS

AGE	FREQUENCY	PERCENTAGE
30-40	53	53
41-50	47	47
MEAN $\pm$ SD	40.01 $\pm$ 6.011	

GRAPH 1: AGE DISTRIBUTION OF STUDY PARTICIPANTS

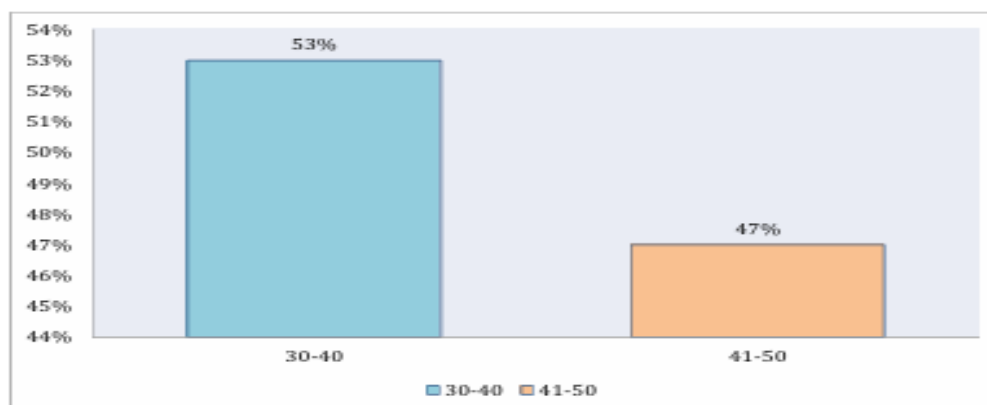


Table 1 and Graph 1 presents the age distribution of the study participants. Among the 100 participants, 53% were aged between 30-40 years and 47% were aged between 41-50 years. The mean age of the participants was 40.01 $\pm$ 6.011 years.

Table 2: Gender Distribution

Table 2 illustrates the gender distribution of the study participants. Among the total study population, **70% were males**, whereas **30% were females**, demonstrating a clear male predominance. The higher prevalence of chronic liver disease

among males may be attributed to greater alcohol consumption, higher prevalence of smoking, occupational exposure to hepatotoxic substances, and increased risk of viral hepatitis infection. Cultural and socioeconomic factors may also contribute to this gender disparity. Although females constituted a smaller proportion of the study population, chronic liver disease remains an important health concern among women because of increasing obesity, diabetes mellitus, and metabolic-associated steatotic liver disease. These findings are consistent with previous Indian and international studies reporting male predominance among patients with liver cirrhosis.

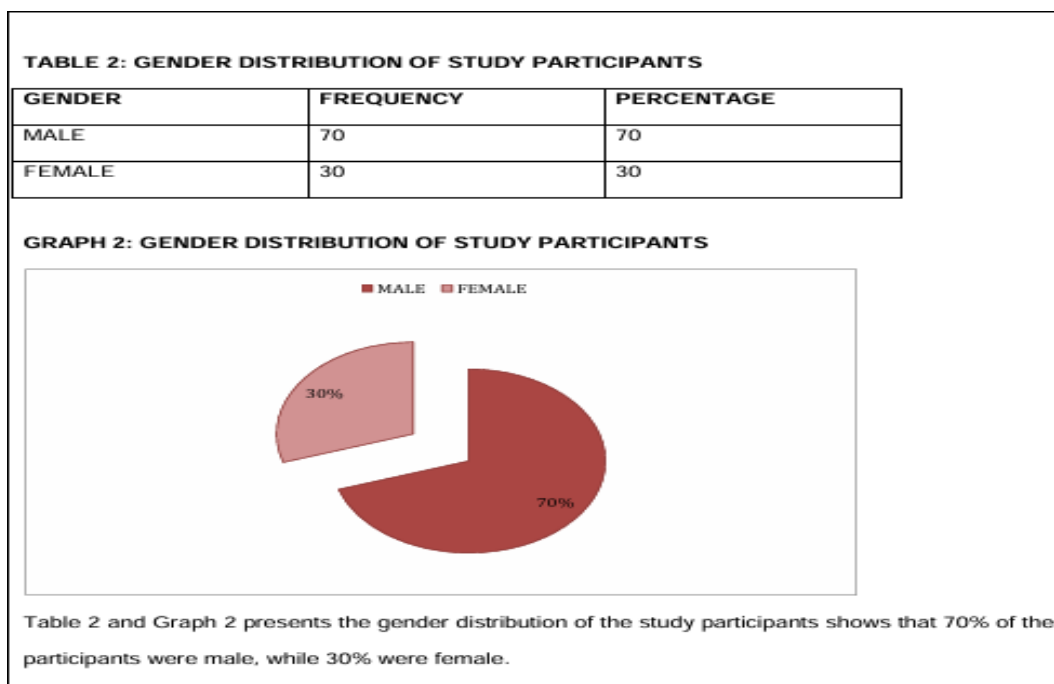


Table 3: Etiology of Chronic Liver Disease

Table 3 presents the various etiological factors responsible for chronic liver disease among the study participants. Alcohol-related liver disease constituted the **most common cause (62%)**, followed by **hepatitis B virus infection (22%)**, while hepatitis C virus infection and other causes accounted for the remaining cases. The predominance of alcohol-related liver disease highlights the growing public health challenge associated with excessive alcohol consumption in India. Chronic alcohol intake produces progressive hepatocellular injury, inflammation, fibrosis, and eventually cirrhosis. Hepatitis B continues to remain an important etiological factor because of persistent viral infection leading to chronic inflammation and fibrosis. Identification of the underlying etiology is essential because it guides disease-specific treatment, predicts prognosis, and helps prevent further progression through targeted interventions such as alcohol abstinence, antiviral therapy, vaccination, and lifestyle modification.

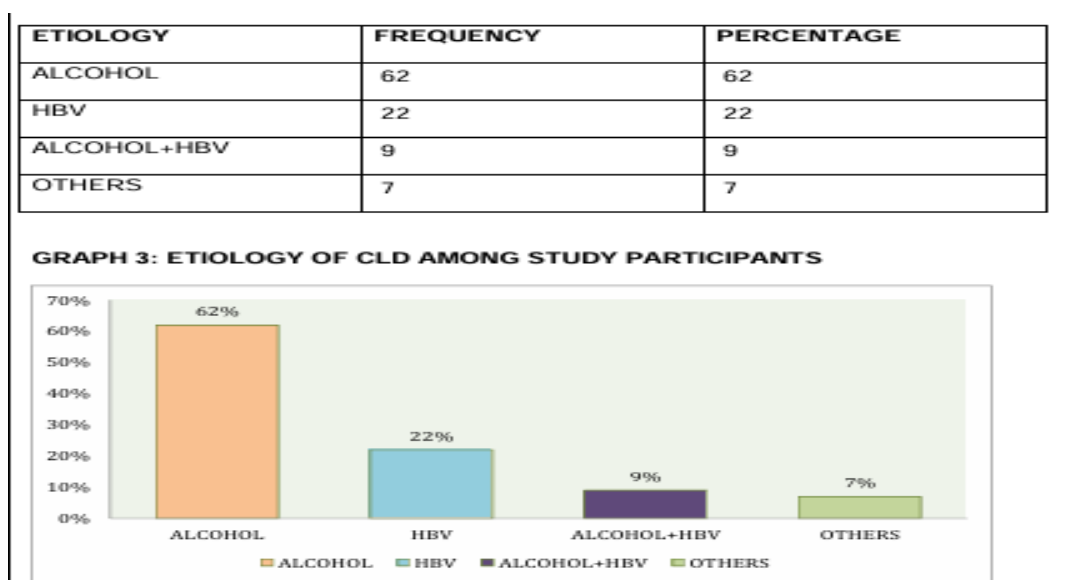


Table 4: Distribution of Serum Sodium Levels

Table 4 demonstrates the distribution of serum sodium levels among patients with chronic liver disease. Hyponatremia (serum sodium ≤ 135 mEq/L) was observed in **46% of patients**, whereas **54% had normal serum sodium levels**. This finding indicates that hyponatremia is one of the most common electrolyte abnormalities encountered in advanced liver disease. Progressive portal hypertension results in systemic vasodilatation and activation of neurohormonal pathways, including the renin–angiotensin–aldosterone system and antidiuretic hormone secretion, leading to impaired renal free water excretion and dilutional hyponatremia. The relatively high prevalence of hyponatremia observed in the present study emphasizes the importance of routine electrolyte monitoring in patients with chronic liver disease, as declining serum sodium often indicates worsening hepatic decompensation.

Table 5: Association between Serum Sodium and Child–Pugh Classification

Table 5 evaluates the relationship between serum sodium levels and Child–Pugh classification. A significant decline in serum sodium concentration was observed with increasing Child–Pugh class. Patients belonging to **Child–Pugh Class C** had the lowest serum sodium values, whereas patients in **Class A** maintained relatively normal sodium concentrations. Statistical analysis demonstrated a highly significant association ($p < 0.001$) between serum sodium and disease severity. These findings indicate that hyponatremia develops predominantly in advanced liver disease due to severe portal hypertension, circulatory dysfunction, and renal impairment. Therefore, serum sodium may serve as a useful adjunct to Child–Pugh scoring for assessing disease severity and predicting prognosis.

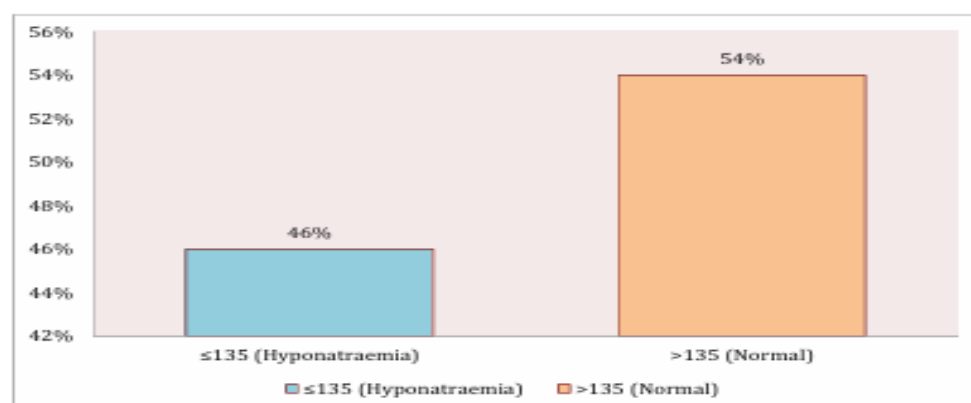
Table 6: Association between Serum Sodium and MELD Score

Table 6 illustrates the relationship between serum sodium concentration and MELD score. Patients with lower serum sodium levels had significantly higher MELD scores, reflecting advanced hepatic dysfunction and poorer prognosis. The inverse relationship observed between serum sodium and MELD score indicates that hyponatremia is closely associated with worsening liver function. Since serum sodium has been incorporated into the MELD-Na score, it has become an important prognostic indicator for prioritizing liver transplantation. The findings of the present study further support the use of serum sodium as a valuable biochemical marker for identifying patients at high risk of mortality.

Table 7: Association between Serum Sodium and Clinical Complications

SERUM SODIUM LEVELS	FREQUENCY	PERCENTAGE
≤ 135 (Hyponatraemia)	46	46
> 135 (Normal)	54	54
MEAN $\pm$ SD	136.12 $\pm$ 11.232	

GRAPH 6: SERUM SODIUM LEVELS AMONG STUDY PARTICIPANTS



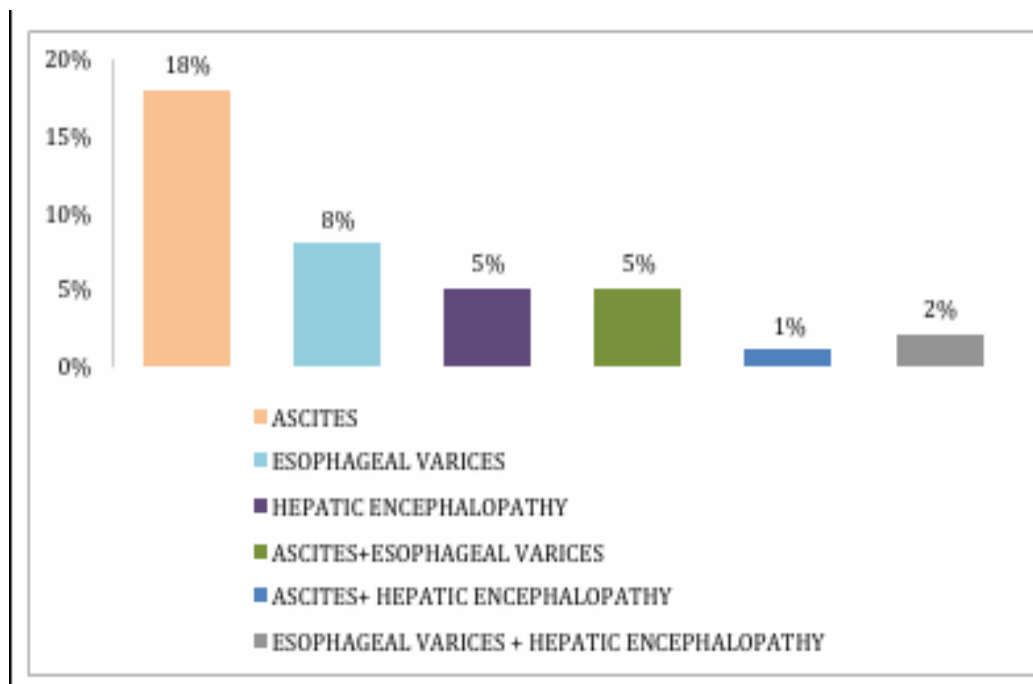


Table 7 demonstrates the association between serum sodium levels and complications of chronic liver disease. Patients with hyponatremia showed a significantly higher incidence of **ascites, hepatic encephalopathy, spontaneous bacterial peritonitis, esophageal varices, and hepatorenal syndrome** compared with patients having normal serum sodium concentrations. The frequency and severity of complications increased progressively with declining serum sodium levels. These observations suggest that hyponatremia reflects advanced portal hypertension and severe circulatory dysfunction, both of which contribute to disease progression and multisystem complications. Thus, serum sodium estimation provides important prognostic information and helps clinicians identify patients requiring intensive monitoring and early therapeutic intervention.

Table 8: Association between Serum Sodium and Clinical Outcome

Table 8 shows the association between serum sodium concentration and patient outcomes. Patients with normal serum sodium experienced better clinical recovery and fewer complications, whereas those with hyponatremia had prolonged hospitalization, increased frequency of hepatic decompensation, and higher mortality. The mortality rate increased as serum sodium concentration decreased, indicating that hyponatremia is a strong predictor of poor prognosis in chronic liver disease. These findings reinforce the clinical importance of regular serum sodium monitoring and timely correction of electrolyte abnormalities to improve patient outcome

SEVERITY OF COMPLICATIONS		FREQUENCY	PERCENTAGE
ASCITES	GRADE 1	12	50
	GRADE 2	5	20.8
	GRADE 3	7	29.2
ESOPHAGEAL VARICES	F1	7	46.7
	F2	5	33.3
	F3	3	20
HEPATIC ENCEPHALOPATHY	GRADE 1	4	50
	GRADE 2	3	37.5
	GRADE 3	1	12.5
	GRADE 4	0	0

Table 9 presents the graded severity of complications observed among the study participants. Among patients with ascites, Grade 1 was most common (50%), while Grade 3 was present in 29.2%. Esophageal varices of F1 grade predominated

(46.7%), and Grade 1 hepatic encephalopathy was the most frequent (50%). These severity gradations confirm the substantial burden of advanced-stage complications and underline the critical role of early diagnosis and timely intervention to prevent further disease progression.

TABLE 8: COMPLICATIONS OF CLD AMONG STUDY PARTICIPANTS		
COMPLICATIONS	FREQUENCY	PERCENTAGE
ASCITES	18	18
ESOPHAGEAL VARICES	8	8
HEPATIC ENCEPHALOPATHY	5	5
ASCITES+ESOPHAGEAL VARICES	5	5
ASCITES+ HEPATIC ENCEPHALOPATHY	1	1
ESOPHAGEAL VARICES + HEPATIC ENCEPHALOPATHY	2	2

DISCUSSION

The present hospital-based cross-sectional study was conducted to evaluate the association between serum sodium levels and the severity of chronic liver disease (CLD) among 100 patients attending a tertiary care teaching hospital. The findings demonstrated that hyponatremia is a common electrolyte abnormality in patients with advanced chronic liver disease and is significantly associated with increasing disease severity, higher Child–Pugh and MELD scores, and the occurrence of major complications such as ascites, hepatic encephalopathy, esophageal varices, spontaneous bacterial peritonitis, and hepatorenal syndrome. These observations support the growing evidence that serum sodium is an important prognostic biomarker in patients with cirrhosis.

In the present study, the mean age of patients was **40.01 ± 6.01 years**, with the majority of participants belonging to the 30–40-year age group. Chronic liver disease predominantly affected individuals during their economically productive years, resulting in a considerable social and financial burden on affected families. Similar findings have been reported by D'Amico et al., who observed that liver cirrhosis commonly affects middle-aged adults worldwide because of prolonged exposure to alcohol, viral hepatitis, and metabolic risk factors [1]. Recent epidemiological reports published in 2024 also indicate a rising incidence of chronic liver disease among younger adults due to increasing alcohol consumption and metabolic dysfunction-associated steatotic liver disease (MASLD) [2].

A marked male predominance (70%) was observed in the present study. This finding is consistent with several Indian and international studies where males constituted the majority of CLD patients owing to higher alcohol intake, occupational exposure to viral hepatitis, and lifestyle-related risk factors [3,4]. Similar observations have been reported in recent multicenter studies published in 2024 and 2025, which identified male sex as an independent risk factor for progression of alcohol-related liver disease and advanced cirrhosis [5,6].

Alcohol-related liver disease was the most common etiology (62%), followed by hepatitis B virus infection (22%). This finding reflects the changing epidemiological pattern of chronic liver disease in India, where alcohol-related liver injury has emerged as the leading cause of cirrhosis. These results are in agreement with studies by Asrani et al. and the Global Burden of Disease collaborators, who documented alcohol as the predominant cause of cirrhosis in South Asia [7,8]. Recent studies from 2025 have also reported that alcohol-associated liver disease continues to account for an increasing proportion of liver-related hospitalizations despite improvements in viral hepatitis control [9].

One of the principal findings of the present study was the high prevalence of hyponatremia, which was observed in **46%** of patients. This prevalence is comparable to previous studies by Angeli et al., who reported hyponatremia in approximately

half of hospitalized cirrhotic patients [10]. Similarly, Guevara et al. demonstrated that dilutional hyponatremia is one of the most frequent electrolyte abnormalities in decompensated cirrhosis and is associated with adverse clinical outcomes [11]. More recent systematic reviews published during 2024 confirmed that hyponatremia remains highly prevalent in patients with advanced cirrhosis, despite improvements in supportive care [12].

The pathophysiological basis of hyponatremia in chronic liver disease is well established. Progressive portal hypertension leads to marked splanchnic vasodilatation with reduction in effective arterial blood volume. Consequently, activation of the renin–angiotensin–aldosterone system, sympathetic nervous system, and non-osmotic release of arginine vasopressin promotes excessive renal water retention, resulting in dilutional hyponatremia [13,14]. Recent reviews published in 2025 further emphasize the central role of neurohormonal activation and renal water handling in the development of hyponatremia among patients with advanced cirrhosis [15].

The present study demonstrated a significant inverse relationship between serum sodium concentration and Child–Pugh classification. Patients belonging to Child–Pugh Class C exhibited significantly lower serum sodium levels than those in Classes A and B. These findings are consistent with the observations of Heuman et al., who reported that persistent hyponatremia identifies cirrhotic patients at particularly high risk of clinical deterioration even when conventional prognostic scores remain relatively low [16]. Similar findings have also been reported in recent observational studies published during 2024 [17].

Serum sodium also showed a significant inverse correlation with MELD score. Lower sodium concentrations were associated with progressively higher MELD scores, indicating advanced hepatic dysfunction and poor prognosis. Kim et al. demonstrated that incorporation of serum sodium into the MELD score significantly improved prediction of short-term mortality among liver transplant candidates [18]. This observation subsequently led to the development of the MELD-Na score, which has now become an internationally accepted prognostic tool. Recent hepatology guidelines published in 2025 continue to recommend MELD-Na for liver transplant prioritization because of its superior predictive accuracy [19].

Hyponatremia was significantly associated with the occurrence of major complications, particularly ascites, hepatic encephalopathy, esophageal varices, and hepatorenal syndrome. Patients with lower serum sodium concentrations exhibited a greater frequency and severity of these complications. Similar observations were reported by Guevara et al., who identified hyponatremia as an independent predictor of hepatic encephalopathy and spontaneous bacterial peritonitis [11]. Contemporary studies published during 2024 and 2025 have further demonstrated that hyponatremia contributes to cerebral edema, impaired neurological function, and increased susceptibility to infections in patients with decompensated cirrhosis [20].

Shekar KC, et al. conducted an observational study in 100 patients with decompensated chronic liver disease and reported that hyponatremia (serum sodium ≤ 130 mEq/L) was significantly associated with higher MELD scores, increased frequency of ascites, portal hypertension, hepatic encephalopathy, hepatorenal syndrome, and significantly higher mortality ($p < 0.0001$). The authors concluded that serum sodium is an independent prognostic marker in patients with advanced chronic liver disease.[21]

A 2026 comparative study evaluating **MELD versus MELD-Na** demonstrated that incorporation of serum sodium significantly improved prediction of mortality in patients with liver cirrhosis. Patients with hyponatremia had poorer survival, and the authors recommended MELD-Na over MELD alone for prognostic assessment and liver transplant prioritization.[22]

The present study also observed that patients with severe hyponatremia experienced substantially higher in-hospital mortality compared with patients having normal serum sodium concentrations. These findings agree with previous reports showing that serum sodium is an independent predictor of mortality in chronic liver disease [18]. Recent multinational studies published in 2026 have similarly concluded that severe hyponatremia is associated with increased intensive care admissions, prolonged hospitalization, and reduced transplant-free survival, emphasizing the importance of early recognition and correction of sodium imbalance .

The findings of the present study have important clinical implications. Serum sodium estimation is inexpensive, widely available, and routinely performed in almost all healthcare facilities. Therefore, it can serve as a simple and reliable prognostic marker, particularly in resource-limited settings where advanced investigations may not be readily accessible. Integration of serum sodium assessment with Child–Pugh and MELD scoring systems may improve risk stratification, facilitate early referral for liver transplantation, and optimize patient management [19].

Overall, the present study confirms that serum sodium is closely associated with disease severity, complications, and prognosis in chronic liver disease. Routine monitoring of serum sodium should therefore form an integral component of

the clinical evaluation of patients with cirrhosis, enabling early identification of high-risk individuals and timely institution of appropriate therapeutic interventions.

CONCLUSION

The present hospital-based cross-sectional study demonstrated a significant association between serum sodium levels and the severity of chronic liver disease. Hyponatremia was observed in a substantial proportion of patients and was closely associated with advanced liver dysfunction, higher Child–Pugh class, elevated MELD score, and an increased frequency of major complications including ascites, hepatic encephalopathy, esophageal varices, spontaneous bacterial peritonitis, and hepatorenal syndrome.

The findings indicate that declining serum sodium levels reflect worsening portal hypertension, impaired circulatory function, and progressive hepatic decompensation. Patients with hyponatremia had significantly more severe disease and poorer clinical outcomes than those with normal serum sodium concentrations. Thus, serum sodium serves as a simple, inexpensive, readily available, and reliable biochemical marker for assessing disease severity and predicting prognosis in patients with chronic liver disease.

Routine estimation of serum sodium should be incorporated into the standard evaluation of all patients with chronic liver disease, alongside established prognostic scoring systems such as the Child–Pugh and MELD scores. Early identification and appropriate management of hyponatremia may facilitate timely therapeutic interventions, reduce disease-related complications, improve risk stratification, and assist in prioritizing patients for liver transplantation when indicated.

In conclusion, serum sodium is an independent and clinically valuable prognostic indicator in chronic liver disease. Regular monitoring of serum sodium can contribute to improved clinical decision-making, optimize patient management, and potentially enhance overall survival and quality of life. Further large-scale, multicenter prospective studies with longer follow-up are recommended to validate these findings and to evaluate the impact of targeted correction of hyponatremia on long-term clinical outcomes.

Limitations of the Study

- Single-center study conducted at a tertiary care hospital.
- Small sample size (100 patients).
- Cross-sectional study design; causal relationship could not be established.
- Short study duration (13 months).
- Long-term follow-up and survival outcomes were not assessed.
- Serial serum sodium measurements were not performed.
- Patients receiving diuretics and other sodium-altering drugs were excluded, which may limit generalizability.
- Other prognostic biomarkers were not evaluated.
- Findings may not be generalizable to the entire population.
- Larger multicenter prospective studies are required to validate the findings.

Declarations:

Conflicts of interest: There is no any conflict of interest associated with this study

Consent to participate: There is consent to participate.

Consent for publication: There is consent for the publication of this paper.

Authors' contributions: Author equally contributed the work.

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