Original Research Article

https://doi.org/10.53339/aimdr.2025.11.3.2
E-ISSN: 2395-2822 | P-ISSN: 2395-2814

Serum sodium levels and their association with complication frequency in liver cirrhosis patients in Bangladesh

Mridha Mohammad Shahinuzzaman[1], Shahana Ahmed[2], Mamun Morshed[1], Mahadi Masud[3], Mohammad Fazlul Huq[1]

[1]Department of Medicine, Faridpur Medical College, Faridpur, Bangladesh, [2]Department of Obstetrics and Gynaecology, Diabetic Association Medical College, Faridpur, Bangladesh, [3]Department of Forensic Medicine, Faridpur Medical College, Faridpur, Bangladesh

Address for correspondence: Dr. Mridha Mohammad Shahinuzzaman, Assistant Professor, Department of Medicine, Faridpur Medical College, Faridpur, Bangladesh. Email: dr.shahin38rmc@gmail.com

Abstract

Background: Liver cirrhosis is a major health issue, often complicated by hyponatremia and other clinical manifestations, such as ascites, hepatic encephalopathy (HE), and varices. Hyponatremia, a common electrolyte disturbance in cirrhosis, is closely linked to disease severity and worsened outcomes. This study aimed to explore the association between serum sodium levels and complications in liver cirrhosis patients in Bangladesh.

Methods: A hospital-based, cross-sectional study was conducted at the Department of Medicine, Sher-E-Bangla Medical College Hospital (SBMCH), Barisal, Bangladesh. A total of 80 patients diagnosed with liver cirrhosis and ascites were included. Serum sodium levels were categorized into three groups: <130 mEq/L, 131–135 mEq/L, and >135 mEq/L. Clinical complications, including ascites, HE, and esophageal varices, were recorded and analyzed based on the severity of hyponatremia.

Results: The study revealed that the majority of patients (49.4%) had serum sodium levels >135 mEq/L, with 26.3% of patients exhibiting hyponatremia (<130 mEq/L). Hyponatremia (<130 mEq/L) was significantly associated with more severe complications, particularly ascites and HE. Patients with higher serum sodium levels (>135 mEq/L) had less severe ascites and HE. Esophageal varices did not show a strong association with sodium levels, though they remained prevalent in 75% of the cohort.

Conclusion: This study underscores the critical role of serum sodium in determining the severity of complications in liver cirrhosis. Lower serum sodium levels were consistently linked to severe ascites, HE, and poor clinical outcomes. Regular monitoring of serum sodium is essential for optimizing management and improving patient prognosis in cirrhosis.

Keywords: Bangladesh, clinical complications, hepatic encephalopathy, hyponatremia, liver cirrhosis, serum sodium

Introduction

Liver cirrhosis is commonly associated with various clinical complications, including refractory ascites, severe hyponatremia, and hypotension. These conditions result from complex pathophysiological mechanisms such as portal hypertension, impaired vascular responsiveness to vasoactive agents, and reduced solute-free water clearance.[1,2] Among these, hyponatremia is particularly prevalent and

concerning, as it is closely linked to impaired water clearance, which significantly increases mortality risk in cirrhotic patients. Those with impaired clearance of solute-free water are at a substantially higher risk of death compared to those with normal water clearance.[3,4] Hyponatremia is a frequent issue in chronic liver disease, with approximately 57% of hospitalized patients and 40% of outpatients being affected by this electrolyte imbalance.[5] This highlights the widespread occurrence of hyponatremia and its strong association with the severity of liver disease, emphasizing the need for careful monitoring and management of serum sodium levels to prevent adverse outcomes.

Despite its significance, the relationship between serum sodium levels and liver cirrhosis-related complications remains insufficiently explored in certain regions, such as Bangladesh. This research gap complicates the understanding of whether serum sodium levels are directly linked to cirrhosis outcomes in the context of Bangladeshi patients. Hyponatremia is more likely to develop when extracellular fluid volume increases, though hypovolemic hyponatremia resulting from diuretic use or gastrointestinal losses can occur; it is typically not the primary cause. Therefore, it is essential to consider this condition as a critical factor when assessing cirrhotic patients with low sodium levels.[6]

Cirrhosis results in a decrease in effective arterial volume, even with an increase in total body water retention.[7] Excessive production of nitric oxide, along with potent vasodilators such as endotoxins, substance P, and endogenous cannabinoids, contributes to the reduction in splanchnic arterial blood volume.[8,9] This cascade stimulates the renal angiotensin-aldosterone system and induces excessive antidiuretic hormone (ADH)-mediated water absorption in the collecting tubule (CTU), leading to increased sodium retention in the proximal renal regions and increased sodium excretion in the CTU. In addition, baroreceptors in the left ventricle and carotid sinus play a critical role in regulating ADH secretion, overriding hypo-osmolality-induced suppression.[10] In cirrhotic patients with ascites, non-osmotic release of ADH from the anterior pituitary becomes the dominant mechanism, resulting in impaired free water excretion and dilutional hyponatremia.

Hyponatremia has also been associated with the development of hepatic encephalopathy (HE), although the underlying mechanisms remain unclear. One proposed explanation is that osmotic gradients between extracellular fluid compartments cause astrocyte swelling.[11] Furthermore, individuals with hyponatremia are found to be 8 times more likely to develop hepatitis E.[1] The severity of hyponatremia correlates strongly with the worsening grades of HE. Recent studies suggest that hyponatremia serves as a significant prognostic marker in patients with chronic liver disease.[12]

This study aims to examine the complications associated with liver cirrhosis and investigate the prevalence of hyponatremia in Bangladeshi patients. Specifically, it will focus on serum sodium levels and their potential association with cirrhotic complications, severity, and survival outcomes in hospitalized patients.

Methodology

Study design

This was a hospital-based, cross-sectional study conducted over 6 months from July 2014 to December 2014.

Place of study

The study was conducted at the Department of Medicine, Sher-E-Bangla Medical College Hospital (SBMCH), Barisal, Bangladesh.

Study population

The study included patients diagnosed with liver cirrhosis and ascites who were admitted to the medicine ward of Sher-E-Bangla Medical College Hospital (SBMCH) during the study period. A total of 80 patients were selected for the study.

Inclusion criteria

- Patients with a confirmed diagnosis of liver cirrhosis and ascites, based on clinical evaluation and diagnostic criteria.
- Patients who provided informed written consent to participate in the study.

Exclusion criteria

- Patients with hepatocellular carcinoma at the time of admission.
- Patients who had been using diuretics during the previous month or throughout the study period.
- Patients with other serious comorbid conditions that may interfere with the study outcomes.

Data collection

Demographic data such as age, gender, height, weight, and body mass index (BMI), along with medical history, were collected from each participant after obtaining informed consent.

Categorization of patients based on serum sodium levels

Patients were classified into three categories based on their serum sodium levels at the time of admission:

- Sodium <130 mmol/L
- Sodium between 131 and 135 mmol/L
- Sodium ≥136 mmol/L

Complications and classification

Common complications observed included ascites, HE, intractable ascites, bacterial peritonitis, hepatic hydrothorax, and hepatic infection. These complications were categorized based on their severity:

- Ascites: Classified into three grades:
  - Grade I: Ascites detected on imaging but not clearly present on physical examination.
  - Grade II: Ascites easily recognized on physical examination and palpation.
  - Grade III: Ascites identified with gross examination and requiring large-volume paracentesis for therapeutic purposes.

Data analysis

All data collected were analyzed using the Statistical Package for the Social Sciences version 26.0 to assess the relationship between serum sodium levels and complication frequency in liver cirrhosis patients.

Results

Table 1 presents the demographic and clinical characteristics of liver cirrhosis patients. The mean age of the patients is 53.6 years, with a higher proportion of females (63.8%) compared to males (36.2%). The mean BMI is 25.5 kg/m^2, indicating a range of weight statuses. Hepatitis B (HBV) is the most common cause of cirrhosis, affecting 59.8% of patients, followed by alcohol use (25%) and Hepatitis C (HCV) (15.6%). The majority of patients are classified as Child-Pugh Class C (50%), indicating severe liver dysfunction, whereas 37.5% are in Class B, and 12.5% are in Class A. The mean

Table 1: Demographic and clinical characteristics of liver cirrhosis patients

Variables	Frequency	Percentage
Mean age (years)	53.6±11.28	
Gender		
Female	51	63.8
Male	29	36.2
Mean body mass index (kg/m^2)	25.5±8.32	
Causes		
Hepatitis B virus	48	59.8
Hepatitis C virus	12	15.6
Alcohol	20	25
Child-Pugh class		
A	10	12.5
B	30	37.5
C	40	50
Mean±standard deviation model for end-stage liver disease	3.8±6.45	

Table 2: Prevalence of Clinical Complications in Liver Cirrhosis Patients in Bangladesh

Complications	Frequency	Percentage
Ascites	66	82.5
Hepatic encephalopathy	25	31.3
Gastric Varices	20	25
Esophageal varices	60	75
Variceal Bleeding	22	28.1
Hepatorenal Syndrome	6	7.5
Bacterial Peritonitis	17	21.9

MELD score is 3.8, reflecting variability in disease severity among the cohort (Table 1).

Table 2 highlights the prevalence of various clinical complications among liver cirrhosis patients in Bangladesh. The most common complication is ascites, present in 82.5% of patients, whereas 17.5% did not experience ascites. HE was observed in 31.3% of patients, with 68.7% remaining unaffected. Esophageal varices were notably prevalent, affecting 75% of patients, whereas gastric varices were less common, seen in only 25%. Variceal bleeding occurred in 28.1% of the patients, with 78.9% not experiencing it. Hepatorenal syndrome was rare, affecting just 7.5% of patients, whereas bacterial peritonitis was found in 21.9% of the patients (Table 2).

The majority of the patients (49.4%) had a serum sodium level >135 mEq/L, with 39 patients falling into this category. This was followed by 26.3% of patients (22 individuals) having hyponatremia with serum sodium levels below 130 mEq/L. The remaining 24.4% (19 patients) had serum sodium levels between 131 and 135 mEq/L (Figure 1).

Table 3 presents the severity of complications in liver cirrhosis patients based on their serum sodium levels. For ascites, the majority of patients with serum sodium levels >135 mEq/L had non-severe ascites (41 patients), whereas the number of patients with severe ascites was highest in the <130 mEq/L group (23 patients). For esophageal varices, the distribution of severe and non-severe cases was more evenly distributed across the serum sodium

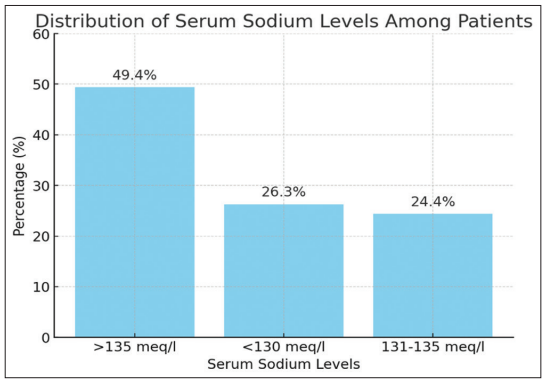

Figure 1: Frequency of sodium among cirrhotic patients

levels, with 15 severe cases in the <130 mEq/L group, compared to fewer cases in the higher serum sodium ranges. Similarly, for HE, most severe cases were observed in the <130 mEq/L group (55 patients), followed by the 131–135 mEq/L group (25 patients), and significantly fewer severe cases in those with sodium levels >135 mEq/L (23 patients) (Table 3).

Discussion

Hyponatremia is a well-documented and common complication in liver cirrhosis, often linked to disease severity and adverse outcomes. Our study explored the association between serum sodium levels and the frequency of complications in liver cirrhosis patients, focusing on ascites, HE, and esophageal varices. The findings confirm that lower serum sodium levels (<130 mEq/L) were associated with more severe complications, consistent with previous studies in the literature.

The majority of patients in our study had serum sodium levels >135 mEq/L, with the lowest prevalence of severe complications in this group, particularly for ascites and HE. This is in agreement with earlier research indicating that lower serum sodium levels are linked to the worsening of cirrhotic complications. According to Adrogué and Madias (2000), hyponatremia results from a combination of factors, including the body's inability to excrete free water and portal hypertension, contributing to sodium retention.[13]

Table 3: Severity of complications in liver cirrhosis patients based on serum sodium levels

Complications	<130 mEq/L	131–135 mEq/L	>135 mEq/L
Ascites			
Severe	23	18	28
Non-Severe	14	15	41
Esophageal varices			
Severe	15	10	10
Non-Severe	15	17	41
Hepatic encephalopathy			
Severe	55	25	23
Non-Severe	15	10	7

Our study found that ascites were more severe in patients with sodium levels <130 mEq/L, which is consistent with Gines *et al*.'s (1998) work that hyponatremia in cirrhosis often correlates with severe fluid retention and a worse prognosis.[14]

In addition, the study revealed a strong association between lower serum sodium levels and HE, particularly in patients with sodium levels <130 mEq/L. This finding is supported by the work of Younas *et al*. (2021), who emphasized that hyponatremia is a significant predictor of HE in cirrhosis patients.[15] The pathophysiology behind this is well-established; hyponatremia can lead to alterations in cerebral edema, which may exacerbate the encephalopathic state. Studies such as Guevara *et al*. (2010) have shown that the serum sodium concentration directly influences the severity and frequency of HE in patients with cirrhosis and refractory ascites.[16]

The study also observed that the severity of esophageal varices did not show a strong correlation with serum sodium levels, as severe cases were evenly distributed across the different sodium categories. However, esophageal varices remain a common complication in cirrhosis and are an important factor in assessing the overall prognosis of patients with cirrhosis. This finding aligns with earlier studies that have identified esophageal varices as a significant complication, but the correlation with hyponatremia has been inconsistent in the literature.[6]

Barakat *et al*. (2015) suggested that hyponatremia in decompensated cirrhosis not only indicates severe liver dysfunction but also correlates with a higher frequency of multiple complications, including renal dysfunction and bacterial infections.[17] Our study also noted the low prevalence of hepatorenal syndrome (7.5%) and bacterial peritonitis (21.9%) in the cohort, reflecting the variability in complications in cirrhotic patients, which may depend on the underlying etiology and individual patient factors.

The findings of this study contribute to the growing body of evidence highlighting the critical role of serum sodium in determining the severity and frequency of complications in cirrhotic patients. As noted by Angeli *et al*., serum sodium levels are an important prognostic factor in cirrhosis, and their careful monitoring is crucial for guiding treatment and improving patient outcomes.[1] In clinical practice, managing hyponatremia in cirrhosis, especially in patients with advanced disease, is essential for reducing complications and improving survival.

Conclusion

Our study underscores the importance of serum sodium as a marker for the severity of liver cirrhosis complications. Lower sodium levels were consistently associated with more severe ascites, HE, and a greater likelihood of poor outcomes. These findings support the need for

regular monitoring of serum sodium in cirrhotic patients to optimize management and improve clinical outcomes. Further longitudinal studies are warranted to explore the causal relationships between sodium levels and long-term prognosis in liver cirrhosis.

References

1. Angeli P, Wong F, Watson H, Gines P, CAPPS Investigators. Hyponatremia in cirrhosis: Results of a patient population survey. Hepatology 2006;44:1535-42.
2. Fernandez-Esparrach G, Sanchez-Fueyo A, Gines P, Uriz J, Quintó L, Ventura PJ, *et al*. A prognostic model for predicting survival in cirrhosis with ascites. J Hepatol 2001;34:46-52.
3. Cosby RL, Yee B, Schrier RW. New classification with prognostic value in cirrhotic patients. Miner Electrolyte Metab 1989;15:261-6.
4. Fernandez-Esparrach G, Sanchez-Fueyo A, Gines P, Uriz J, Quintó L, Ventura PJ, *et al*. A prognostic model for predicting survival in cirrhosis with ascites. J Hepatol 2001;34:46-52.
5. Gines P, Berl T, Bernardi M, Bichet DG, Hamon G, Jiménez W, *et al*. Hyponatremia in cirrhosis: From pathogenesis to treatment. Hepatology 1998;28:851-64.
6. Kim JH, Lee JS, Lee SH, Bae WK, Kim NH, Kim KA, *et al*. The association between the serum sodium level and the severity of complications in liver cirrhosis. Korean J Intern Med 2009;24:106-12.
7. Schrier RW. Water and sodium retention in edematous disorders: Role of vasopressin and aldosterone. Am J Med 2006;119 7 Suppl 1:S47-53.
8. Ginès P, Cárdenas A. The management of ascites and hyponatremia in cirrhosis. Semin Liver Dis 2008;28:43-58.
9. Iwakiri Y. The molecules: Mechanisms of arterial vasodilatation observed in the splanchnic and systemic circulation in portal hypertension. J Clin Gastroenterol 2007;41 Suppl 3:S288-94.
10. Tivers MS, Handel I, Gow AG, Lipscomb VJ, Jalan R, Mellanby RJ. Hyperammonemia and systemic inflammatory response syndrome predicts presence of hepatic encephalopathy in dogs with congenital portosystemic shunts. PLoS One 2014;9:e82303.
11. Yun BC, Kim WR. Hyponatremia in hepatic encephalopathy: An accomplice or innocent bystander? Am J Gastroenterol 2009;104:1390-1.
12. Shaikh S, Mal G, Khalid S, Baloch GH, Akbar Y. Frequency of hyponatraemia and its influence on liver cirrhosis-related complications. J Pak Med Assoc 2010;60:116-20.
13. Adrogué HJ, Madias NE. Hyponatremia. N Engl J Med 2000;342:1581-9.
14. Gines P, Berl T, Bernardi M, Bichet DG, Hamon G, Jiménez W, *et al*. Hyponatremia in cirrhosis: From pathogenesis to treatment. Hepatology 1998;28:851-64.
15. Younas A, Riaz J, Chughtai T, Maqsood H, Saim M, Qazi S, *et al*. Hyponatremia and its correlation with hepatic encephalopathy and severity of liver disease. Cureus 2021;13:e13175.
16. Guevara M, Baccaro ME, Ríos J, Martín-Llahí M, Uriz J, Ruiz Del Arbol L, *et al*. Risk factors for hepatic encephalopathy in patients with cirrhosis and refractory ascites: Relevance of serum sodium concentration. Liver Int 2010;30:1137-42.
17. Barakat AA, Metwaly AA, Nasr FM, El-Ghannam M, El-Talkawy MD, Taleb HA. Impact of hyponatremia on frequency of complications in patients with decompensated liver cirrhosis. Electron Physician 2015;7:1349-58.