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Original Research Article

Serum Sodium Levels as an Indicator of Clinical Severity in Patients with Decompensated Chronic Liver Disease

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Conflict of interest: Nil

Abstract:

Background: Decompensated chronic liver disease is frequently associated with electrolyte abnormalities, among which hyponatremia is an important marker of advanced circulatory dysfunction and poor prognosis.

Aim: To study serum sodium levels in patients with decompensated chronic liver disease (CLD) and to observe their clinical significance.

Methods: This prospective observational cross-sectional study was conducted at Dr. Pinnamaneni Siddhartha Institute of Medical Sciences and Research Foundation from July 2023 to May 2025. Sixty patients with decompensated CLD were enrolled. Clinical evaluation, liver function tests, serum electrolytes, coagulation profile, ultrasonography abdomen, portal vein Doppler, and ascitic fluid analysis were performed. Statistical analysis was done using SPSS version 23.

Results: The mean age was 52 ± 10.50 years, and 95% were males. Alcohol was the commonest etiology (95%). The mean serum sodium level was 126.45 ± 5.66 mEq/L. Hyponatremia (serum sodium ≤130 mEq/L) was observed in 66.7% of patients. Significant associations were found between low serum sodium and ascites, portal hypertension, upper gastrointestinal bleed, coagulopathy, and hepatorenal syndrome. No significant association was observed with spontaneous bacterial peritonitis.

Conclusion: Serum sodium is a valuable and easily measurable prognostic marker in decompensated CLD and may help identify patients at greater risk of complications.

Keywords: Decompensated chronic liver disease; Hyponatremia; Serum sodium; Ascites; Hepatorenal syndrome.

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Introduction

Decompensated chronic liver disease (DCLD) represents the advanced stage of cirrhosis in which complications such as ascites, hepatic encephalopathy, spontaneous bacterial peritonitis, hepatorenal dysfunction, and circulatory failure become clinically evident. In this stage, dilutional hyponatremia is a frequent biochemical abnormality caused mainly by portal hypertension, reduced effective arterial blood volume, neurohormonal activation, and impaired renal free-water excretion [1]. Recent literature shows that hyponatremia in cirrhosis is not merely a laboratory abnormality but a marker of severe systemic derangement, being associated with higher morbidity, poorer quality of life, increased risk of encephalopathy, refractory ascites, renal dysfunction, and mortality [2,3]. It has also gained prognostic importance because serum sodium is incorporated into contemporary severity models such as MELD-based systems used for risk stratification and transplant prioritization. A recent hospital-based study in patients with DCLD further emphasized that low serum sodium levels are common and clinically relevant, supporting routine monitoring in such patients [3]. Current expert guidance also recommends careful evaluation of the cause of hyponatremia in cirrhosis and serial sodium assessment during management. Hence, studying serum sodium levels in patients with decompensated chronic liver disease (CLD) and observing their clinical significance may help identify high-risk patients early, guide timely interventions, and improve prognostic assessment. The aim of the study is to find serum sodium levels in patients with decompensated CLD and to observe their clinical significance.

Methods

This prospective observational, cross-sectional study was conducted in the department of General Medicine, Dr. Pinnamaneni Siddhartha Institute of Medical Sciences and Research Foundation, over a period of two years from July 2023 to May 2025. The study included patients with DCLD who were registered or admitted during the study period. Prior approval was obtained from the Institutional Ethics Committee before commencement of the study. Participation was entirely voluntary, and informed consent was obtained from the patients or their guardians, wherever appropriate. Confidentiality of all participants was strictly maintained throughout the study, and no intervention posing risk of injury was undertaken as part of the research protocol.

All patients diagnosed with decompensated CLD based on clinical examination, laboratory investigations, and radiological imaging were considered eligible for inclusion. Patients with coexisting congestive cardiac failure, chronic kidney disease, or those receiving drugs known to alter serum sodium levels, such as selective serotonin reuptake inhibitors, tricyclic antidepressants, monoamine oxidase inhibitors, and cytotoxic drugs, were excluded from the study. After enrolment, a detailed clinical history was obtained and thorough physical examination was performed in each case.

Relevant investigations were carried out in all study participants. Blood samples were collected for liver function tests, serum electrolyte analysis, and coagulation profile including prothrombin time and international normalized ratio (INR). Ultrasonography of the abdomen was performed to assess liver echotexture and confirm the nature of CLD. Portal vein Doppler study was done to evaluate portal hemodynamics, and ascitic fluid analysis was performed wherever indicated. All data were recorded in predesigned proformas. Statistical analysis was carried out using SPSS software version 23.0. Quantitative variables were expressed as mean ± standard deviation, while qualitative variables were presented as frequencies and percentages. Differences between categorical variables were assessed using the chi-square test. Comparison of means was performed using Student's *t*-test and Wilcoxon matched-pairs test wherever appropriate. A p value of less than 0.05 was considered statistically significant, and interpretation was made at a 95% confidence interval.

Results

A total of 60 patients with decompensated CLD were included in the study. The mean age of the participants was 52 ± 10.50 years, and most patients belonged to the 41–50 years age group, followed by 51–60 years. There was a marked male (n = 57; 95%) predominance. Regarding etiology, alcohol was the leading cause of DCLD, accounting for 95% (57) cases, while hepatitis B-related liver disease contributed to 5% (3). Among complications at admission, ascites was the most frequent finding, present in 75% of patients, followed by portal hypertension in 58.33%, hepatorenal syndrome in 23.33%, upper gastrointestinal bleed in 20%, and both coagulopathy and spontaneous bacterial peritonitis in 16.6% each. The mean serum sodium level was 126.45 ± 5.66 mEq/L, indicating that hyponatremia was common in this cohort. Mean serum bilirubin, albumin, and INR were 6.48 ± 2.30 mg/dL, 2.62 ± 0.52 g/dL, and 1.45 ± 0.48, respectively. On subgroup analysis, 40 patients had serum sodium ≤130 mEq/L and 20 had sodium >130 mEq/L. Patients with lower sodium levels had significantly higher frequencies of ascites, upper GI bleed, coagulopathy, and hepatorenal syndrome. SBP did not show a significant association with serum sodium level.

Table 1: Baseline demographic profile

Variable	Category	n	%
Age group; Mean age: 52 ± 10.50 years	31–40	12	20
	41–50	19	31.7
	51–60	17	28.3
	61–70	11	18.3
	>70	1	1.7
Gender	Male	57	95
	Female	3	5

Table 2: Complications associated with DCLD on admission

Complication	Present n (%)	Absent n (%)
Ascites	45 (75.0)	15 (25.0)
Portal hypertension	35 (58.33)	25 (41.67)
Upper GI bleed	12 (20.0)	48 (80.0)
Coagulopathy	10 (16.6)	50 (83.4)
Hepatorenal syndrome	14 (23.33)	46 (76.67)
Spontaneous bacterial peritonitis	10 (16.6)	50 (83.4)

Table 3: Distribution of continuous clinical variables				
Variable	**Mean**	**SD**	**Minimum**	**Maximum**
Serum sodium (mEq/L)	126.45	5.6611	110	136
Serum bilirubin (mg/dL)	6.4766	2.3005	2.4	11.6
Serum albumin (g/dL)	2.6167	0.5175	1.4	3.4
INR	1.445	0.4793	0.8	3.1

Table 4: Complications in relation to serum sodium groups			
Complication	**Na in mEq/L; n (%)**		**P value**
	≤130; n = 40	**>130; n = 20**	
Ascites	34 (85.0%)	11 (55.0%)	<0.001
Portal hypertension	28 (70.0%)	7 (35.0%)	0.02
Upper GI bleed	12 (30.0%)	0 (0%)	<0.001
Coagulopathy	9 (22.5%)	1 (5.0%)	<0.001
Hepatorenal syndrome	12 (30.0%)	2 (10.0%)	0.02
SBP	6 (15.0%)	4 (20.0%)	0.74

Discussion

The present study showed that decompensated CLD was predominantly seen in middle-aged and elderly patients, with a mean age of 52 years, and there was a striking male predominance, as 95% of the study population were men. Alcohol was the principal etiology, accounting for 95% of cases, whereas hepatitis B contributed to only 5% of cases. This clinicodemographic profile is in agreement with recent Indian literature, which has consistently shown that alcohol-related cirrhosis remains a major cause of decompensated liver disease admissions in men and is commonly associated with multiple portal hypertensive and systemic complications [4, 5]. The overall complication burden in the present study was also substantial, with ascites in 75%, portal hypertension in 58.33%, upper gastrointestinal bleed in 20%, hepatorenal syndrome in 23.33%, coagulopathy in 16.6%, and spontaneous bacterial peritonitis in 16.6%, confirming that the study population largely represented advanced disease at presentation [5].

A major observation in the present study was the high prevalence of hyponatremia. The mean serum sodium was 126.45 ± 5.66 mEq/L, and 40 patients had serum sodium ≤130 mEq/L. Hyponatremia in cirrhosis is now recognized as a marker of advanced circulatory dysfunction rather than an isolated electrolyte abnormality. Splanchnic vasodilatation, reduction in effective arterial blood volume, activation of the renin-angiotensin-aldosterone and sympathetic nervous systems, and non-osmotic release of vasopressin together impair renal free-water excretion, leading to dilutional hyponatremia [4]. Indian evidence has further highlighted that low serum sodium is closely linked to disease severity, poorer quality of life, greater decompensation burden, and adverse short-term outcomes, and this is the reason sodium has become an important component of prognostic systems such as MELD-Na [6]. Thus, the low mean sodium level observed in the present study strongly suggests that a large proportion of patients were in an advanced hemodynamic stage of decompensated cirrhosis [4, 6].

The association between serum sodium and ascites was particularly noteworthy in the present study. Ascites was present in 85% of patients with serum sodium ≤130 mEq/L compared with 55% among those with sodium >130 mEq/L, and this association was highly significant. These findings are biologically plausible because both hyponatremia and ascites arise from the same pathophysiological framework of portal hypertension, arterial underfilling, and renal sodium and water retention [4, 5]. Likewise, portal hypertension was more frequent in the lower sodium group, and the tabulated p value of 0.02 indicates a significant association. Upper gastrointestinal bleed was also seen exclusively in the hyponatremic group in a clinically important proportion, again suggesting that low sodium may reflect more severe portal hypertensive disease and poorer hepatic reserve [7, 8]. Indian studies on advanced cirrhosis have similarly demonstrated that patients with more severe portal hypertension are more likely to develop new decompensation events, variceal bleeding, and worse survival, supporting the interpretation that hyponatremia can serve as a practical surrogate marker of severe portal circulatory disturbance [5, 8].

Another clinically important finding in the present study was the significant association of low serum sodium with coagulopathy and hepatorenal syndrome. Coagulopathy was present in 22.5% of patients with sodium ≤130 mEq/L versus 5% in patients with sodium >130 mEq/L, while hepatorenal syndrome was seen in 30% and 10%, respectively. This pattern indicates that hyponatremia paralleled worsening synthetic liver

dysfunction and progressive renal circulatory impairment. In cirrhosis, renal vasoconstriction, systemic inflammation, and worsening portal hypertensive hemodynamics contribute to hepatorenal syndrome, and falling serum sodium often precedes or accompanies renal dysfunction [4, 9]. Recent Indian studies have reinforced the major prognostic role of kidney injury in cirrhosis, demonstrating that AKI biomarkers, baseline creatinine, and response models for terlipressin have strong associations with mortality and treatment response in patients with hepatorenal syndrome-AKI [10, 11]. Therefore, the present data support the clinical view that serum sodium should be routinely monitored not merely as a biochemical parameter, but also as an early warning signal for renal decompensation in DCLD [9 – 11].

In contrast, spontaneous bacterial peritonitis did not show a statistically significant association with serum sodium in the present study. This lack of significance may be attributable to the modest sample size and relatively smaller number of SBP events rather than true absence of clinical interaction. In advanced cirrhosis, infection, circulatory dysfunction, and hyponatremia frequently coexist and worsen one another. Indian studies evaluating albumin therapy have shown that albumin administration can improve not only kidney dysfunction but also hyponatremia, hepatic encephalopathy, and infection-related decompensation, underlining the interconnected nature of these complications [12, 13]. Similarly, Indian data on long-term albumin use in decompensated cirrhosis have suggested reductions in recurrent decompensation, bacterial infection, kidney dysfunction, and mortality, indirectly supporting the concept that low sodium identifies a clinically fragile subgroup requiring closer monitoring and more aggressive supportive care [12, 13]. Hence, even though SBP itself was not statistically linked with sodium in this cohort, the overall evidence still places hyponatremia within a broader syndrome of advanced systemic decompensation in cirrhosis.

Overall, the present study establishes that serum sodium has important clinical significance in decompensated CLD. Hyponatremia was common and was significantly associated with ascites, portal hypertension, upper gastrointestinal bleed, coagulopathy, and hepatorenal syndrome, indicating that lower sodium levels accompany more severe decompensation. These findings are in line with contemporary Indian literature showing that hyponatremia, renal dysfunction, inflammatory burden, and advanced portal hypertension are major determinants of adverse outcomes in cirrhosis [9 – 13]. The major strengths of the study are its prospective design and use of simple bedside and laboratory variables that are readily applicable in routine practice. However, the single-center nature of the study, relatively small sample size, and absence of longitudinal survival or transplant-related outcome analysis limit broader generalization. Even so, the study clearly supports routine serum sodium assessment in all patients with decompensated CLD, both for early risk stratification and for timely intervention before irreversible multiorgan dysfunction develops [7].

Conclusion: The present study showed that hyponatremia was a common biochemical abnormality in patients with decompensated CLD. Lower serum sodium levels were significantly associated with major complications such as ascites, portal hypertension, upper gastrointestinal bleed, coagulopathy, and hepatorenal syndrome, indicating more advanced disease and poorer clinical status. These findings suggest that serum sodium is a simple, inexpensive, and clinically useful marker for assessing severity in DCLD. Routine measurement of serum sodium at admission and during follow-up may help in early identification of high-risk patients, guide closer monitoring, and support timely therapeutic intervention to reduce morbidity and improve overall outcome.

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