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A STUDY ON SERUM SODIUM LEVELS IN DECOMPENSATED CHRONIC LIVER DISEASE PATIENTS DURING THE PHASE OF HEPATIC ENCEPHALOPATHY AT TERTIARY CARE HOSPITAL , KALABURAGI

General Medicine

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ABSTRACT

Background: Hyponatremia is a common electrolyte disturbance in patients with decompensated chronic liver disease (DCLD) and is associated with worsening circulatory dysfunction, cerebral edema, and progression of hepatic encephalopathy (HE). Serum sodium has emerged as an important prognostic marker in cirrhotic patients and is incorporated into scoring systems such as MELD-Na. This study was undertaken to evaluate serum sodium levels in DCLD patients during the phase of hepatic encephalopathy and to assess its association with disease severity and clinical outcomes.

Objectives:

1.) To study serum sodium levels in patients with decompensated chronic liver disease during hepatic encephalopathy.

2.) To assess the association of serum sodium levels with severity of hepatic encephalopathy and clinical outcomes.

Methods: A prospective observational study was conducted in the Department of General Medicine, Basaveshwar Teaching and General Hospital, Mahadevappa Rampure Medical College, Kalaburagi, over a period of 18 months from June 2024 to November 2025. A total of 120 patients aged >18 years diagnosed with decompensated chronic liver disease and hepatic encephalopathy as per West Haven criteria were included. Patients with acute intracerebral events, sepsis, hepatorenal syndrome, cardiac failure, and those on drugs affecting sodium balance were excluded. Serum sodium levels were measured at admission and correlatedwith hepatic encephalopathy severity and clinical outcomes including ICU admission, mechanical ventilation, and in-hospital outcome.**Results:** Among 120 patients, 75% were males and 25% were females. The majority belonged to the younger and middle-aged groups. Mean serum sodium levels were significantly lower in patients requiring ICU admission (129.65 ± 4.19 mEq/L) compared to those not requiring ICU care (131.80 ± 4.42 mEq/L; p = 0.010). Patients requiring mechanical ventilation also had lower mean sodium levels (129.76 ± 4.46 mEq/L) compared to those not requiring ventilation (131.22 ± 4.43 mEq/L), though this association was not statistically significant (p = 0.222). Lower serum sodium levels were associated with greater severity of hepatic encephalopathy and poorer clinical outcomes. **Conclusion:** Hyponatremia is highly prevalent in patients with decompensated chronic liver disease presenting with hepatic encephalopathy and serves as an important marker of disease severity. Lower serum sodium levels are associated with increased risk of adverse clinical outcomes, particularly ICU admission. Serum sodium is a simple, cost-effective, and valuable prognostic indicator that can aid in risk stratification and management of patients with advanced liver disease.

KEYWORDS

Decompensated Chronic Liver Disease, Hepatic Encephalopathy, Hyponatremia, Serum Sodium, Cirrhosis, Prognostic Marker.

INTRODUCTION

Chronic liver disease (CLD) encompasses a spectrum of disorders characterized by progressive impairment of hepatic function over a period exceeding six months. This deterioration affects crucial processes such as the synthesis of coagulation factors, metabolic detoxification, and bile excretion. Pathologically, CLD involves sustained inflammation, necrosis, and regeneration of liver parenchyma, which culminates in fibrosis and ultimately cirrhosis. The range of underlying causes for CLD is broad, including prolonged alcohol abuse, exposure to toxins, persistent infections, autoimmune reactions, and genetic or metabolic abnormalities. The global burden of CLD is substantial, with mortality rates particularly high in developing countries.

Recent epidemiological data indicate a rising incidence of CLD. In developed regions, the primary etiologies are alcoholic liver disease, chronic hepatitis B and Cvirus infections, non-alcoholic fatty liver disease (NAFLD), and disorders of iron metabolism such as hemochromatosis1

Estimates suggest that there are approximately 1.5 billion individuals living with CLD worldwide, irrespective of the disease's severity2. NAFLD accounts for the majority of these cases (59%), with hepatitis B virus (HBV) and hepatitis C virus (HCV) comprising 29% and 9% respectively, and alcoholic liver disease (ALD) responsible for about 2%. Other etiologies, such as primary biliary cholangitis, primary sclerosing cholangitis, alpha-1-antitrypsin deficiency, Wilson's disease, and autoimmune hepatitis, collectively constitute about 1% of the global CLD burden3 Liver cirrhosis (LC), the advanced stage of many chronic liver diseases, is traditionally conceptualized as a continuum, progressing from mild to severe stages until the endpoint of either death or liver transplantation. "Compensated liver cirrhosis"(CLC) describes a state in which cirrhosis is present without overt complications like ascites, hepatic encephalopathy (HE), variceal hemorrhage (VH), or jaundice4.

Once any of these decompensating events occur, the diagnosis shifts to "decompensated liver cirrhosis" (DLC). In this state, persistent pathogenic mechanisms continue to drive further clinical deterioration. Precipitating factors for complications, particularly HE, include constipation, esophageal variceal bleeding, and bacterial infections such as spontaneous bacterial peritonitis5 Fluid and electrolyte imbalances are common in liver cirrhosis, with hyponatremia—defined as a serum sodium concentration ≤136 mEq/L6—being a notable manifestation. In cirrhotic patients, clinically significant hyponatremia is generally considered at serum sodium levels below 130 mEq/L7. Hyponatremia is categorized based on the patient's volume status into hypovolemic, euvolemic, and hypervolemic forms, and further classified by serum osmolality and antidiuretic hormone (ADH) activity8.

Pseudohyponatremia, an artifactually low sodium level seen in laboratory analysis, may also arise in acute or chronic hepatic insufficiency due to the displacement of plasma water by excessive lipids or proteins9

Hypovolemic hyponatremia in cirrhosis most frequently results from overtreatment with diuretics, though severe gastrointestinal fluid loss (such as persistent diarrhea) can also play a role. Patients often show clinical evidence of dehydration, and in advanced cases, this can progress to prerenal azotemia or kidney failure10

OBJECTIVES OF THE STUDY

1. To study serum sodium levels in Decompensated chronic Liver disease patients during the phase of Hepatic Encephalopathy
2. To study the association of serum sodium levels with severity of patients in Hepatic encephalopathyMATERIALS AND METHODS

Source of Data

The study included patients with decompensated chronic liver disease and hepatic encephalopathy who were admitted to Basaveshwar Teaching & General Hospital, affiliated with Mahadevappa Rampure Medical College, Kalaburagi. Study Design and Setting This was a prospective observational study conducted in the Department of General Medicine at Basaveshwar Teaching & General Hospital, Mahadevappa Rampure Medical College, Kalaburagi. Study Duration The study was conducted over a period of 18 months, from June 2024 to November 2025.

Inclusion Criteria

1.) Patients aged above 18 years of any gender.
2.) Patients diagnosed with decompensated chronic liver disease and hepatic encephalopathy, as per West Haven's criteria.

Exclusion Criteria

1.) Patients with acute intracerebral events (infarct or hemorrhage).
2.) Patients with sepsis and positive blood cultures.
3.) Patients with a history of drug intake (narcotics, valproic acid).
4.) Patients who developed hepatorenal syndrome.Patients on SSRIs, TCAs, MAO inhibitors, or cytotoxic drugs.
5.) Patients with cardiac failure.

RESULTS

Age distribution

Age Group (years)	Frequency	Percent
≤29	28	23.3%
30–39	18	15.0%
40–49	21	17.5%
50–59	17	14.2%
60–69	17	14.2%
70–79	17	14.2%
≥80	2	1.7%
Total	**120**	**100%**

Table :Age Distribution

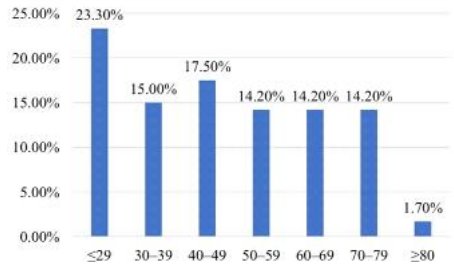
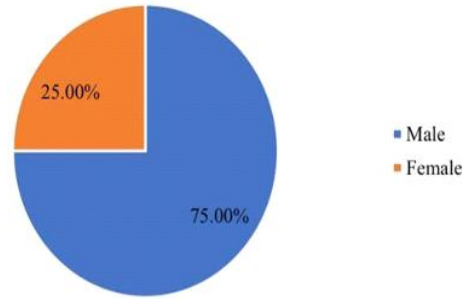


Figure: Age Distribution

The age distribution of the study population shows that patients were widely distributed across age groups ranging from ≤29 to ≥80 years. The highest proportionof patients belonged to the ≤29 years category, accounting for 23.3% of the total cohort, indicating a relatively younger predominance in this study population. The 40–49 year age group constituted 17.5% of cases, followed by 30–39 years at 15.0%. The proportions of patients in the 50–59, 60–69, and 70–79 year groups were equal at 14.2% each, suggesting a consistent representation across middle-aged and elderly groups. Patients aged ≥70 years collectively contributed to a significant portion of the cohort, while only 1.7% were aged ≥80 years. Overall, the distribution reflects a broad age spectrum with a higher concentration in younger and middle-aged individuals.

Figure:Sex Distribution (n = 120)



The sex distribution indicates a clear male predominance in the study population. Out of 120 patients, 90 were males, constituting 75.0% of the sample, whereas females accounted for only 25.0% (n = 30)

Table:Sex Distribution (n = 120)

Sex	Frequency	Percent
Male	90	75.0%
Female	30	25.0%
Total	**120**	**100%**

DISCUSSION

Decompensated chronic liver disease represents the advanced stage of hepatic dysfunction characterized by the development of complications such as ascites, hepatic encephalopathy, variceal bleeding, and renal impairment. Among these, hepatic encephalopathy is a serious neuropsychiatric manifestation resulting from the accumulation of neurotoxins, particularly ammonia, in the setting of impaired hepatic detoxification. It significantly contributes to morbidity, mortality, and healthcare burden in cirrhotic patients. In recent years, increasing attention has been directed toward electrolyte disturbances, particularly hyponatremia, as an important pathophysiological and prognostic factor in these patients.

Hyponatremia in cirrhosis is primarily dilutional in nature, resulting from impaired free water excretion due to non osmotic release of vasopressin, systemic vasodilatation, and activation of neurohumoral mechanisms. This disturbance not only reflects the severity of circulatory dysfunction but also plays a direct role in cerebral edema and worsening of hepatic encephalopathy. Serum sodium, therefore, has emerged as a simple, cost-effective, and clinically significant biomarker that reflects both the severity of liver disease and the risk of complications. It has been incorporated into prognostic models such as the MELD-Na score, underscoring its relevance in predicting survival and guiding management decisions, including liver transplantation.

Several studies have demonstrated that lower serum sodium levels are associated with higher grades of hepatic encephalopathy, increased hospitalizations, need for intensive care, and mortality. Furthermore, dynamic changes in serum sodium during hospitalization have been shown to correlate with clinical improvement or deterioration.In this context, the present study was undertaken to evaluate serum sodium levels in patients with decompensated chronic liver disease during hepatic encephalopathy and to analise its association with disease severity and clinical outcomes. The findings of this study are discussed in comparison with previously published literature to better understand the role of hyponatremia as a prognostic marker and its clinical implications in the management of hepatic encephalopathy.

DEMOGRAPHIC PROFILE

In our study, the age distribution showed a predominance of younger individuals, with ≤29 years accounting for 23.3%, followed by 40–49 years (17.5%) and 30–39 years (15.0%), while patients aged ≥70 years also constituted a notable proportion, indicating a wide age spectrum with clustering in younger and middle-aged groups. Males constituted 75.0% and females 25.0%, demonstrating a marked male predominance. Similar findings were observed in Younas et al.,12 where males constituted 75% and females 25%, although the mean age was higher at 53.7 ± 11.3 years, indicating a relatively older population. Sardar et al. 115also reported a predominance of older age groups, with most patients in 51–60 years (38.8%) and 61–65 years (37.6%), and a strong male predominance (82.4%). Comparable gender distribution was noted by Chhabra et al.,14 who also reported male predominance with a mean age of 49.79 ± 13.52 years.

SERUM SODIUM AND CLINICAL OUTCOMES

In our study, patients requiring ICU admission had lower mean serum sodium levels (129.65 ± 4.19 mEq/L) compared to those not requiring ICU care (131.80 ± 4.42 mEq/L; p = 0.010), indicating that hyponatremia is associated with increased disease severity and need for intensive care. A similar association between sodium imbalance and adverse outcomes was observed in Shahrad et al116., where patients with hypernatremia had significantly longer ICU stay (20.8 ± 20.6 vs 9.4 ± 11.2 days; p < 0.001) and hospital stay (33.2 ± 28.2 vs 16.4 ± 16.3 days; p < 0.001), suggesting that electrolyte disturbances, whether hypo- or hypernatremia, are linked with worse clinical outcomes. In our study, serum sodium levels were lower in patients requiring mechanical ventilation (129.76 ± 4.46 mEq/L) compared to those not requiring ventilation (131.22 ± 4.43 mEq/L); however, this association was not statistically significant (p = 0.222). In contrast, Shahrad et al116. reported that patients with hypernatremia had a significantly higher requirement for intubation (81% vs 47%; p < 0.01), indicating more severe clinical deterioration.

LIMITATIONS

This study has certain limitations that should be considered while interpreting the findings. Firstly, the study was conducted in a single tertiary care center with a relatively limited sample size of 120 patients, which may restrict the generalizability of the results to the broader population. Secondly, the cross-sectional design limits the ability to establish a causal relationship between serum sodium levels and severity of hepatic encephalopathy, as only associations can be inferred. Thirdly, dynamic changes in serum sodium levels during hospitalization were not assessed,which could have provided better insights into prognostic trends. Additionally, other potential confounding factors such as variations in treatment protocols, fluid management, and use of diuretics were not fully controlled. The study also relied on clinical grading systems like the West Haven criteria, which may have some degree of subjective variability. Finally, long-term follow-up data were not included, limiting the understanding of the impact of hyponatremia on long-term outcomes and survival.

CONCLUSION

This study demonstrates that hyponatremia is highly prevalent among patients with decompensated chronic liver disease presenting with hepatic encephalopathy and serves as a significant marker of disease severity. Lower serum sodium levels were strongly associated with higher grades of hepatic encephalopathy, worse prognostic scores (Child–Pugh, MELD, and MELD-Na), increased need for ICU admission, and higher in-hospital mortality. A clear inverse relationship between serum sodium levels and disease severity was observed, highlighting its clinical importance. These findings underscore the role of serum sodium as a simple, cost-effective, and valuable prognostic indicator in the management of patients with advanced liver disease. Early recognition and appropriate management of hyponatremia may contribute to improved clinical outcomes and reduced mortality in this high-risk population.

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