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Original Research Article

Hyponatremia and Its Relationship with Disease Severity and Complications in Decompensated Chronic Liver Disease: A Hospital-Based Study

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ABSTRACT

Background: Decompensated chronic liver disease (DCLD) is frequently associated with electrolyte disturbances, particularly hyponatremia, which reflects advanced disease and circulatory dysfunction. It is increasingly recognized as a predictor of poor clinical outcomes and complications.

Objectives: (i) To determine the prevalence of hyponatremia and evaluate its association with disease severity in patients with decompensated chronic liver disease. (ii) To assess the relationship between serum sodium levels and the occurrence of major complications in these patients.

Materials & Methods: This hospital-based cross-sectional observational study included 94 patients aged ≥18 years with DCLD presenting with ascites, hepatic encephalopathy, or variceal bleeding. Conducted over one year at a tertiary care teaching hospital in Bareilly, Uttar Pradesh, the study utilized a pre-designed proforma to record demographic data, clinical history, examination findings, and laboratory and radiological investigations. Parameters required for Child–Pugh score calculation were documented.

Results: The mean age was 51.6 ± 12.5 years, with male predominance (81%). The main etiologies were alcoholic liver disease (48%) and viral hepatitis (45%). The mean serum sodium level was 133.68 ± 6.21 mEq/L. Hyponatremia was present in 45% of patients, while 2% had hypernatremia. Patients with hyponatremia had higher mean MELD (17.47 ± 6.63) and MELD-Na (22.84 ± 5.59) scores compared to those with normal sodium (15.12 ± 7.10 and 15.61 ± 7.04, respectively). Serum sodium levels were significantly associated with Child–Pugh class ($p = 0.013$). Complications were more frequent among patients with hyponatremia.

Conclusion: Hyponatremia is common in DCLD and is significantly associated with greater disease severity and increased risk of complications, underscoring its prognostic importance.

Keywords: Hyponatremia; Decompensated chronic liver disease; Serum sodium; MELD score; MELD-Na; Child–Pugh score; Liver cirrhosis; Ascites

INTRODUCTION

Liver cirrhosis is a major global health concern being a leading cause of morbidity and mortality. Despite advances in medical management, the burden of CLD is increasing, particularly in Asia and Africa, where healthcare access and early detection is limited.[1, 2]

The asymptomatic phase of liver cirrhosis (undetected for several years) is followed by clinical signs of decompensated chronic liver disease (DCLD), which is associated with disturbances in serum sodium levels. Sodium plays a crucial role in fluid balance maintenance; its dysregulation in CLD has serious clinical implications. DCLD is the advanced stage with significantly compromised liver's functional capacity, and its inability to maintain essential physiological processes. In CLD, cirrhosis, a common endpoint, results in hypoalbuminemia and portal hypertension, impacting sodium balance by triggering a series of events. The reduced protein synthesis, including albumin, promotes the extravasation of fluid into the peritoneal space and causing ascites, by leading to diminished oncotic pressure. Ascites further disrupts sodium excretion and retention mechanisms as it stimulates the renin-angiotensin-aldosterone system.[3]

This results in many life-threatening severe morbid complications like upper gastrointestinal bleeding/ esophageal varices, ascites, jaundice, and hepatic encephalopathy. Later, other complications develop like acute kidney injury (AKI), hepato-pulmonary syndrome (HPS), with or without the features of HRS, porto-pulmonary hypertension (PPHT), cirrhotic cardiomyopathy (CCM), bacterial infections / peritonitis.[4-6] The serum sodium homeostasis is vital to the normal cell functioning. Abnormal sodium level result from an imbalance in the total body water regulation. DCLD leads to dysnatremia by disturbance in water homeostasis. [7,8] A disturbance in total body water regulation leads to decreased solute free water clearance. The consequent inability to match the urine output to the amount of water ingested leads to dilutional hyponatremia, which is associated with high mortality rate. Hypernatremia, is less common than hyponatremia in DCLD patients.[5] Hyponatremia is the commonest electrolyte disorder in hospitalized patients and especially in DCLD patients.[9,10] Hyponatremia has been incorporated into prognostic models such as the Model for End-Stage Liver Disease–Sodium (MELD-Na) score, emphasizing its independent role in predicting mortality and disease severity. [11] As a reasonably reliable predictor of survival in most of hepatic disease, Child–Pugh score computation predicts the major prognosis / complication of liver cirrhosis. In DCLD, the clinical significance of serum sodium levels extends beyond prognostication. Hyponatremia contributes to neurological dysfunction, including hepatic encephalopathy, through mechanisms involving astrocyte swelling and brain edema. It also worsens outcomes in hepatorenal syndrome and spontaneous bacterial peritonitis, conditions that define the transition from compensated to decompensated disease. [12] Early identification and management of hyponatremia in DCLD could significantly influence clinical outcomes, reduce hospital stay, and improve quality of life. Monitoring sodium levels can also enhance risk stratification, guide fluid management, and help predict mortality more accurately than traditional liver function parameters alone. Although the association between serum sodium levels and cirrhosis outcomes has been well documented globally, data from the Indian population remain scarce and heterogeneous, particularly regarding the relationship between the development of DCLD complications and the degree of hyponatremia. Hyponatremia in CLD patients is correctable and timely action can improve their quality of life and functional status, decreasing mortality. Among liver cirrhosis patients, serum sodium levels are independent of etiology, sex, or age, with hyponatremia prevalence ranging from 40-57% (<130 mmol/L in 22%). Even a mild serum sodium decreases in (130–134 mmol/L) has a worse prognosis. In advanced cirrhosis, resulting from profound hemodynamic changes, hyponatremia is a marker of poor prognosis; it worsens the quality of life in hepatic encephalopathy patients contributing to the disturbed consciousness. There are limited data correlating serum sodium levels with the cirrhosis complications development. This study sought to understand the hyponatremia prevalence and its association with the CLD in to help its better management. Assessment of serum sodium levels in liver disease patients can enhance patient prioritization, since hyponatremia is an independent predictive factor for survival in patients with DCLD. For stratifying cirrhotic patients, prognostic predictors are crucial in directing the therapy, estimating survival, and identifying those eligible for transplantation of liver.[13] Most existing studies have been hospital-based with limited sample sizes and varying diagnostic criteria. The basic mechanism precipitating hepatic encephalopathy because of hyponatremia is not yet fully understood. Therefore, this study determined the prevalence of hyponatremia in patients with DCLD. Monitoring sodium levels can guide therapeutic interventions with modern research continuing to unravel the DCLD complexities. Advancements in managing electrolyte imbalances hold promise for improving outcomes in this seriously challenging disease. Therefore, this study was undertaken to assess whether serum sodium levels reflect the severity of liver dysfunction and linked to complications. It is also uncertain whether low sodium levels can serve as a reliable indicator of prognosis when compared with established scoring systems.

Objectives

1. To determine the prevalence of hyponatremia and evaluate its association with disease severity in patients with decompensated chronic liver disease.
2. To assess the relationship between serum sodium levels and the occurrence of major complications in these patients.

MATERIALS & METHODS

This hospital-based cross-sectional observational study was conducted over one year in the Department of General Medicine at a tertiary care teaching hospital, Bareilly, Uttar Pradesh. Every eligible patient presenting during routine hospital visit was approached in sequence until the target sample size is achieved. Hence, Consecutive sampling was adopted for patient selection. Sample size calculation showed total 94 patients was enrolled in the study based on following formula:

$$n = \frac{Z_{\alpha/2}^{2}\, p(1-p)}{d^2}$$

Where,

- $Z_{\alpha/2} = 1.96$ for 95% confidence,
- $p = 0.433$ (anticipated proportion from James et al.[10]),
-
- $d = 0.10$ (Absolute margin of error, 10%).

$$n = \frac{1.96^2 \times 0.433 \times 0.567}{0.10^2} = 94.3 \Rightarrow \mathbf{94}$$

A pre-designed proforma was used to record patient details. The proforma captured demographic data, relevant medical history, baseline liver function tests, clinical examination findings, and laboratory/radiological investigation results. The form included all parameters required for Child–Pugh score computation.

Ethical consideration: This study was done after getting the approval by Institutional Ethics Committee with Ref.no. SRMSIMS/ECC/2024/41 of SRMS IMS, Bareilly. Written informed consent was obtained from each patient participating in the study. All decompensated cirrhotic patients aged ≥18 years admitted to the Department of Medicine, SRMS IMS, Bareilly, during the study period were screened for eligibility. Patients with known cardiac conditions were excluded. After obtaining informed consent, demographic and clinical details were recorded.

Statistical Analysis

The collected data was entered into Microsoft Excel 2019 and analyzed using SPSS software (version 27.0). Descriptive statistics was used to summarize the demographic and clinical characteristics of the participants. Continuous variables were presented as means ± standard deviations and categorical variables were shown in frequencies and percentages along with presented using tables, bar diagrams, and pie charts where appropriate. Association between categorical variables was tested using Chi-square/Fisher's exact test, and continuous variables was compared using ANOVA for more than two groups.[14] A p-value <0.05 was considered statistically significant.

RESULT

Table 1 shows that the mean age of the study population was 51.6 ± 12.5 years with male preponderance (80.9%). The main etiology was alcoholic liver disease (47.9%) and viral hepatitis (44.7%). The mean hemoglobin level was 9.16 ± 2.64 g/dL. The mean total leukocyte count was 8306 ± 4771/mm³, and the differential count showed a predominance of neutrophils (70.3 ± 13.3%), with lymphocytes (20.1 ± 9.8%), eosinophils (2.46 ± 2.37%), and monocytes (5.66 ± 2.65%). The mean platelet count was 120,398 ± 85,681/mm³. Mean HbA1c was 5.29 ± 1.16%.

Table 1: Baseline Characteristics and Laboratory Profile of decompensated cirrhotic patients (n = 94)

Variable	Value
Age (years), mean ± SD	51.6 ± 12.5
Sex: Male, n (%)	76 (80.9)
Etiology, n (%)	
Alcoholic	45 (47.9)
Viral hepatitis	42 (44.7)
NASH	4 (4.3)
Others	3 (3.2)
Laboratory Parameters (mean ± SD)	
Hemoglobin (g/dL)	9.16 ± 2.64
Platelet count (/mm³)	120,398 ± 85,681
Total bilirubin (mg/dL)	3.97 ± 5.16
INR	1.62 ± 0.72
Serum sodium (mEq/L)	133.68 ± 6.21
Serum creatinine (mg/dL)	1.14 ± 0.74
MELD score	16.43 ± 7.07
MELD-Na score	19.09 ± 7.37

Mean serum cholesterol was 132.8 ± 25.4 mg/dL. Mean triglycerides was124.4 ± 34.2 mg/dL. The mean HDL, LDL, and VLDL levels were 46.96 ± 10.94 mg/dL, 65.48 ± 18.55 mg/dL, and 25.71 ± 8.43 mg/dL, respectively. The mean serum sodium / potassium levels were 133.68 ± 6.21 mEq/L / 4.03 ± 0.75 mEq/L respectively. The mean SGOT and SGPT levels were 117.6 ± 219.7 U/L and 42.9 ± 40.3 U/L respectively. The mean total bilirubin was 3.97 ± 5.16 mg/dL, with direct bilirubin averaging 1.94 ± 2.80 mg/dL and indirect bilirubin 2.02 ± 2.51 mg/dL. The mean prothrombin time (PT) was 23.31 ± 9.43 seconds and INR was 1.62 ± 0.72 The mean MELD and MELD-Na scores were 16.43 ± 7.07 and 19.09 ± 7.37, respectively. The mean serum magnesium and phosphorus levels were 1.52 ± 0.26 mg/dL and 3.67 ± 0.07 mg/dL, respectively, while serum calcium averaged 9.82 ± 0.77 mg/dL. Renal parameters showed a mean serum creatinine of 1.14 ± 0.74 mg/dL and blood urea of 48.69 ± 36.72 mg/dL. Nearly half of the patients (44.7%) exhibited hyponatremia, and only 2.1% had hypernatremia (Fig 1).

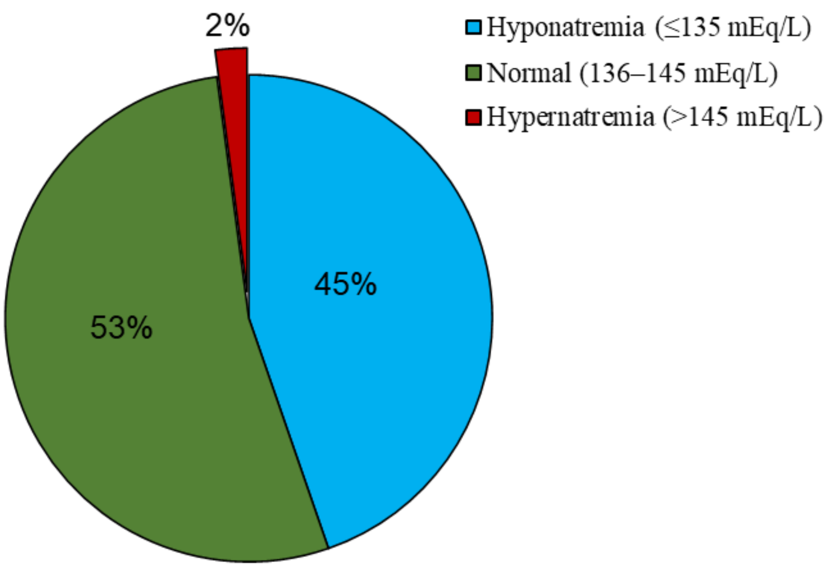

Figure 1: Distribution of Serum Sodium Levels in Patients with Decompensated Chronic Liver Disease

The mean MELD and MELD-Na scores varied significantly across serum sodium categories (Table 2). Patients with hyponatremia (≤135 mEq/L) had higher MELD (17.47 ± 6.63) and MELD-Na (22.84 ± 5.59) scores compared to those with normal sodium levels (MELD: 15.12 ± 7.10; MELD-Na: 15.61 ± 7.04). The distribution of serum sodium levels varied significantly (χ^2 = 8.72, p = 0.013) across Child–Pugh classes. Hyponatremia was most frequent in Child–Pugh Class C (70.6%), compared to 51.6% in Class B and only 6.9% in Class A. Conversely, normo-natremia predominated in Class A (89.7%) and declined progressively in Classes B and C.

Among patients with hyponatremia, the majority had alcoholic liver disease (52.4%), followed by viral etiology (40.5%), while smaller proportions were due to NASH (7.1%).

In the normonatremic group, viral (48.0%) and alcoholic (44.0%) etiologies were almost equally distributed. Cases of cryptogenic (1.1%) and other rare etiologies (2.1%) were infrequent across all sodium categories.

Table 2: Association of Serum Sodium with Disease Severity

Variable	Hyponatremia (≤135) n=42	Normal (136–145) n=50	Hypernatremia (>145) n=2	p-value
MELD score (mean ± SD)	17.47 ± 6.63	15.12 ± 7.10	27.40 ± 2.21	0.023@
MELD-Na score (mean ± SD)	22.84 ± 5.59	15.61 ± 7.04	27.40 ± 2.21	<0.001@
Child–Pugh Class, n (%)				0.013$
Class A	2 (6.9)	26 (89.7)	1 (3.4)	
Class B	16 (51.6)	14 (45.2)	1 (3.2)	
Class C	24 (70.6)	10 (29.4)	0 (0.0)	

@ANOVA Test, $Fisher exact test.

Table 3 shows complications were more frequent among patients with hyponatremia (≤135 mEq/L) compared to those with normal or elevated sodium levels. The prevalence of ascites (92.9%) and portal hypertension (83.3%) was high across all sodium categories, though not statistically significant (p = 0.082 and p = 0.144, respectively). However, hepatic encephalopathy (52.4%, p = 0.007), coagulopathy (61.9%, p = 0.031), hepatorenal syndrome (28.6%, p = 0.034), and spontaneous bacterial peritonitis (35.7%, p = 0.028) were significantly more common in patients with hyponatremia.

Table 3: Association between Serum Sodium Levels and Complications

Complication	Hyponatremia n (%)	Normal n (%)	p-value$
Ascites	39 (92.9)	40 (80.0)	0.082
Portal hypertension	35 (83.3)	34 (68.0)	0.144
Hepatic encephalopathy	22 (52.4)	10 (20.0)	0.007
GI bleeding	8 (19.0)	5 (10.0)	0.272
Coagulopathy	26 (61.9)	18 (36.0)	0.031
Hepatorenal syndrome	12 (28.6)	4 (8.0)	0.034@
Spontaneous bacterial peritonitis	15 (35.7)	6 (12.0)	0.028

$Chi square test, @Fisher exact test.

DISCUSSION

Cirrhosis is a significant global public health burden. Chronic inflammation of liver results in cirrhosis, wherein acute liver function deterioration leads to DCLD, resulting in many complications. Dilutional hyponatremia is associated with spontaneous bacterial peritonitis, hepatorenal syndrome, and severe ascites. As a major marker of poor prognosis, hyponatremia indicates reduced survival due to its association with poor liver functioning (raised prothrombin time / total bilirubin / liver enzymes, low serum albumin levels), and worsening kidney function (high serum creatinine levels), placing a major health resources burden.[6,15]

The mean age of the present study population was 51.6 ± 12.5 years with male preponderance (80.9%). This indicates that the majority of cases occurred in individuals aged above 40 years. This indicates a marked male predominance among DCLD patients. The mean serum sodium levels in the present study were 133.68 ± 6.21 mEq/L. Nearly half of the patients (45%) exhibited hyponatremia, and only 2.1% had hypernatremia. Hyponatremia thus represented a frequent biochemical abnormality among DCLD patients in the present study. These findings indicate that lower serum sodium levels are associated with increased frequency of major complications in DCLD. Hyponatremia prevalence was reported as 49.4% by Angeli et al [5], and 57% by James et al.[10]

In Bhandari and Chaudhary study, 41% patients with mean age of 53.44±7.57 yrs (64% males and 36% females) had hyponatremia.[16]

The MELD includes hyponatremia diagnoses to better predict survival. A high mortality rate (over 50%) is seen in severe hyponatremia (<120 mEq/L) cases with, particularly alcoholics. Similarly, despite low severity scores, cirrhotic patients with hyponatremia and persistent ascites have a high mortality risk, indicating hemodynamic decompensation. In Azam et al study, 36% patients had hyponatremia (mean-118.13±5.22 mEq/L). The prevalence of hyponatremia was elevated among DCLD patients.[15]

Etiology of liver cirrhosis includes NAFLD, HCV, HBV, biliary / cardiac issues, alcohol, autoimmune / celiac / metabolic disease, hemochromatosis, Wilson's disease etc. Complications like liver insufficiency, portal hypertension, infections, nutritional deficiencies, hepatopulmonary syndrome, hepatic encephalopathy, hepatic hydrothorax, hepatorenal syndrome, cirrhotic cardiomyopathy, bone and joint disease develop in its decompensated stage. As a marker of advanced stages, due to excessive water retention, hyponatremia frequently results, reflecting the cirrhosis severity, and potential for complications.[17]

The main etiology was alcoholic liver disease (48%) and viral hepatitis (45%) in the present study. Al Kaabi et al found in Oman that liver cirrhosis was mainly due to hepatitis B (27%), alcohol (30%), and hepatitis C (28%). The mean age of their study population was 58 ± 13.8 years, with mean CLIF, MELD-Na, and CTP scores reported as 41, 18, and 9, respectively. In Azam et al study, BMI and age were the commonest risk factors for hyponatremia. Due to water retention in DCLD patients, as a complication Marker, hyponatremia was commonly linked to refractory ascites (96%), reduced survival, high mortality, advanced portal hypertension hepatorenal syndrome (41%), and hepatic encephalopathy (34%).[15] The lowest sodium levels (<130 mmol/L) had the more complications.[5] The mean MELD and MELD-Na scores were 16.43 ± 7.07 and 19.09±7.37, respectively in the present study. These scores varied significantly across serum sodium categories. Patients with hyponatremia (≤135 mEq/L) had higher MELD (17.47 ± 6.63) and MELD-Na (22.84±5.59) scores compared to those with normal sodium levels (MELD: 15.12 ± 7.10; MELD-Na: 15.61±7.04). This indicated that worsening hyponatremia was associated with increasing disease severity in patients with DCLD.

Nagaraja et al also found that 56% patients had hyponatremia (mean -134.0 ± 5.5 mEq/L). The patients with hyponatremia had higher MELD / MELD-Na and Child-Pugh scores.[18]

The liver disease severity was associated strongly with serum sodium levels of as per MELD and Child Pugh score. Patient with serum sodium levels ≤130 had increased mortality (30.4%; p = 0.002).[10]In 40-57% patients Pradhan et al reported hyponatremia, which was associated with higher MELD and Child–Pugh scores, and increased risk of complications.[11]

In Younas et al study, hyponatremia was present in 37% patients. The hyponatremia severity was correlated with HE grades.[19] Mean age was 48.38±11.8 yrs in Nareddy et al study, with male preponderance (97%). Hyponatremia was noted in 34.7% ;87.9% belonged to Child Pugh C. The association of hyponatremia and Child Pugh C was highly significant. A positive correlation was found between low sodium levels and complications like spontaneous bacterial peritonitis and hepatorenal syndrome. Low sodium levels in cirrhosis had a positive correlation with the disease severity, spontaneous bacterial peritonitis and hepatorenal syndrome.[20] Mei et al prospective study on 3880 patients with CLD, 712 developed within 90 days some adverse outcomes. Overall, 2% hypernatremic,0.72% severe hyponatremic, and 21% mild hyponatremic patients were there at admission. The risk of 90-day adverse outcomes decreased by 5% after adjusting for all confounding factors, (OR 0.95; $p < 0.001$), 24% (OR 0.76; 95% CI 0.70–0.84; $p < 0.001$), and by 42% (OR 0.58; $p < 0.001$) as Na level increased by 1, 5, and 10 mmol/L, respectively. Non-correction of hyponatremia on days 4 and 7 was associated with 2.05-fold (hazard ratio [HR], 2.05; $p < 0.001$) and 1.46-fold (HR 1.46; $p = 0.028$) higher risk of adverse outcomes. In patients with CLD, hyponatremia was an independent risk factor for a poor 90-day prognosis. Failure of hyponatremia correction a week after admission was associated with increased mortality.[21]

In Chaudhary et al study, coagulopathy, hepatic encephalopathy, and hepatorenal syndrome) were significantly more common among Group A patients vs B or C. Mean MELD, CPS score and mortality were also significantly higher among group A patients. Significantly higher MELD/ CPS score, more severe CLD/ complications, and mortality were seen in patients with lower serum salt levels.[22] Sardar et al observed hyponatremia in more than half of the patients, was the most prevalent anomaly with patients most severely affected. HE was seen in 53.5%.[23]

Kim et al reported dilutional hyponatremia prevalence as 47% strongly associated with Child-Pugh / MELD scores and severe complications.[24]

Hyponatremia prevalence was 57% in Kumar and Ashok study, associated with complications and higher MELD /Child Pugh scores. The most common etiology of CLD was alcohol abuse and hepatitis B. Serum sodium level was not associated with etiology of CLD, age/ gender. A higher frequency of complications /mortality was seen in patients with serum sodium <130 mEq/L.[25] Pradeep and Sindhura found hyponatremia in 75% cirrhotic patients, mostly chronic alcoholics. Mean serum sodium was 130.9 ± 6.1 mEq/L, lowest in Child–Pugh Class B and C, with significant association between hyponatremia and hepatic encephalopathy.[26]

Hyponatremia was found in 30% patients in Borroni et al study; Case fatality rate was more in hyponatremia patients.[27]Barakat et al reported hyponatremia in 60% cirrhotics, associated with higher MELD/MELD-Na scores / complications.[28] In Mobin et al study,the mean age was 42.2±SD yrs (63% male). Most patients (90%) had hyponatremia, negatively correlated with complications.[29] In Udagani et al study, 59.9% patients with severe liver failure, had low serum sodium levels, irrespective of their age / sex of patients.[30]

In Khyalappa and Bardeskar study, the serum sodium levels were -< 130meq/l (73%),130-135 meq/l (17%) > 135meq/l (10%). The most common complication in hyponatraemia patient was ascities (96%), followed by hepatic encephalopathy (34%), hepato-renal syndrome (41%) and variceal bleeding (37%). Higher mortality rate (35%) and complications were seen in patients with serum sodium <130meq/L.[31] Mumtaz et al study found A grade Child-Pugh was seen in 34%, B grade in 30.7% and C grade in 35.3% cases. The mean sodium level was 142.7±15.5 mmol/L. In 1/3rd cases hyponatremia was present (Mild - 48%, moderate 50% and severe in2%).[32] Hyponatremia was seen in 25.5% class A, 39.1% in class B and 35.8% in class C Child-Pugh (P>0.05). The hyponatremia frequency of in CLD patients was high. In the present study, the distribution of serum sodium levels varied significantly across Child–Pugh classes. Hyponatremia was most frequent in Child–Pugh Class C (70.6%), compared to 51.6% in Class B and only 6.9% in Class A. Conversely, normo-natremia predominated in Class A (89.7%) and declined progressively in Classes B and C. Among patients with hyponatremia, the majority had alcoholic liver disease (52.4%), followed by viral etiology (40.5%), while smaller proportions were due to NASH (7.1%).

In the normo-natremic group, viral (48.0%) and alcoholic (44.0%) etiologies were almost equally distributed. Cases of cryptogenic (1.1%) and other rare etiologies (2.1%) were infrequent across all sodium categories. The prevalence of ascites (92.9%) and portal hypertension (83.3%) was high across all sodium categories, though not statistically significant. However, hepatic encephalopathy (52.4%), coagulopathy (61.9%), hepatorenal syndrome (28.6%), and spontaneous bacterial peritonitis (35.7%) were significantly more common in patients with hyponatremia. This study demonstrated that many cirrhosis patients had low serum sodium levels (43%), having no significant correlation with cirrhosis etiology, age, and gender. MELD / MELD-Na scores had negative correlation with serum sodium levels. In Sunil et al study, 46% patients were aged 41-50 years with male predominance (96%). Hyponatremia was correlated with increasing severity of liver disease. Child-Pugh Class C patients had a significantly higher hyponatremia incidence; so, lower sodium levels were associated more severe liver dysfunction.[12]In a prospective observational study, Kohir et al reported hyponatremia in 47% patients (18% mild, 16% moderate, and 14% severe). Highest one-month mortality (54%) and more complications were seen in severe hyponatremia patients (≤125 mmol/L). The mean age was 47.4 years, majority being over 40 years old. The male-female ratio was 5.3:1. Alcohol-related liver disease was the commonest cause (69.5%), besides HBV infection (15.8%), and NAFLD (7.4%) etc. Severe hyponatremia patients had more advanced decompensation. Ascites was present in all cases, HRS in 38.5%, jaundice in 92%, SBP in 15%, and HE in 46%. MELD-Na superiority as predictor of short-/ long-term mortality was strongly supported by their results.[17]

CONCLUSION

Hyponatremia is a common finding in patients with decompensated chronic liver disease and is strongly associated with advanced disease severity. Lower serum sodium levels correlate with higher MELD and MELD-Na scores and with worsening Child–Pugh class, being most prevalent in Class C patients. These findings reinforce the role of hyponatremia as a clinically relevant marker of disease progression and severity. Its high prevalence, particularly among patients with alcoholic and viral etiologies, highlights the importance of routine monitoring and early identification to aid risk stratification and optimize clinical management.

Recommendations: At regular intervals, serum sodium levels should be monitored in DCLD patients to avoid complications. The hyponatremia patients may need intensive care and careful monitoring, to increase their survival rate.

Declaration by Authors

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