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Original Article

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Frequency of hyponatremia and its effect on complications in decompensated chronic liver disease (DCLD)

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ABSTRACT

BACKGROUND & OBJECTIVE: Hyponatremia, an electrolyte imbalance, is prevalent in decompensated chronic liver disease (DCLD) and is linked to an increased risk of complications, including hepatic encephalopathy, ascites, hepatorenal syndrome, and variceal bleeding. The study aims to assess the frequency of hyponatremia and its impact on various complications in patients with DCLD.

METHODOLOGY: A cross-sectional study was conducted on 120 DCLD patients aged 15-75 years at Allama Iqbal Memorial Teaching Hospital from January to June 2023. The sample size of 120 patients was stratified using the Child-Pugh classification, and complications were assessed, including ascites, hepatic encephalopathy, hepatorenal syndrome, variceal bleeding, and spontaneous bacterial peritonitis. The data analysis was conducted using SPSS version 23. Chi-square test was applied, and a p-value of ≤ 0.05 was considered statistically significant.

RESULTS: The frequency of hyponatremia was 40%, with 48 patients affected. Complications were more common in patients with hyponatremia, including ascites (80.6% in males, p = 0.000; 94.1% in females, p = 0.005), hepatic encephalopathy (45.2% in males, p = 0.026), and hepatorenal syndrome (9.7% in males, p = 0.016). Variceal bleeding was significantly associated with hyponatremia (22.6% in males, p = 0.004).

CONCLUSION: Hyponatremia is a frequent and clinically significant complication in DCLD, associated with higher morbidity and mortality. Early detection and management are crucial for improving patient outcomes.

KEYWORDS: Hyponatremia, Liver Cirrhosis, Decompensated, Hepatic Encephalopathy, Hepatorenal Syndrome.

INTRODUCTION

Decompensated chronic liver disease (DCLD) is a life-threatening condition that affects millions worldwide and is characterized by significant hepatic dysfunction, portal hypertension, and multi-organ failure. Among the many complications of DCLD, hyponatremia, an electrolyte imbalance characterized by a serum sodium concentration of less than 135 mmol/L, is particularly common. Its presence is linked to an increased risk of complications, increased morbidity, and poorer prognosis, significantly impacting the course and management of DCLD [1]. The emergence of hyponatremia in individuals suffering from cirrhosis arises from the compromised ability to eliminate water, stemming from excessive production of antidiuretic hormone (ADH) and the kidneys' failure to regulate sodium levels, particularly amidst portal hypertension and the stimulation of neurohormonal systems. This condition is prevalent among a significant portion of chronic liver disease (DCLD) sufferers, with investigations indicating a prevalence of 30% to 50% [2].

The frequency of hyponatremia correlates with the progression of liver disease. Research has shown that approximately 30% of patients with early-stage cirrhosis may develop hyponatremia over the course of the disease, and this figure increases to 50% in those with decompensated cirrhosis [3]. The incidence of hyponatremia is closely tied to the severity of liver disease. Studies reveal that around 30% of individuals with early-stage cirrhosis may encounter hyponatremia as the disease advances, a statistic that escalates to 50% in cases of decompensated cirrhosis. Moreover, the occurrence of severe hyponatremia (serum sodium levels falling below 125 mmol/L) has been observed in up to

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21% of patients with cirrhosis. Importantly, the presence of hyponatremia in those with DCLD corresponds with poorer clinical outcomes, including elevated hospitalization rates, prolonged hospital stays, and increased mortality [4].

Hyponatremia exerts a profound influence on the progression of cirrhosis and the emergence of its related complications. One notable complication is hepatic encephalopathy (HE), which affects roughly 30% to 50% of cirrhosis patients [5]. Research has indicated that individuals with hyponatremia face a heightened likelihood of developing hepatic encephalopathy, with risk escalating by 1.5 to 2 times compared to those with normal sodium levels [6]. The underlying pathophysiology of this connection encompasses the impact of sodium deficiency on cerebral function and neurotransmitter imbalance, both of which lead to cognitive decline in cirrhotic patients. Consequently, the existence of hyponatremia significantly deteriorates the prognosis for patients grappling with hepatic encephalopathy. In addition to hepatic encephalopathy, hyponatremia is closely linked to the onset of renal dysfunction, particularly in the manifestation of hepatorenal syndrome (HRS).

This severe condition is marked by advancing renal failure in the absence of prior kidney damage, occurring in about 10-18% of individuals with decompensated cirrhosis [6]. The incidence of HRS is heightened in those experiencing severe hyponatremia, as renal perfusion is further hindered due to systemic vasodilation and renal vasoconstriction. The emergence of HRS in hyponatremic patients raises the risk of mortality, with 30-day mortality rates soaring to as high as 50% [7]. Ascites, another common complication of cirrhosis, is present in up to 85% of patients with DCLD, and its presence is closely linked to hyponatremia [8]. In patients with refractory ascites, hyponatremia occurs in approximately 60% of cases, further complicating the management of fluid balance in these patients [9]. Additionally, hyponatremia is associated with an increased risk of developing spontaneous bacterial peritonitis (SBP), an infection of the ascitic fluid that occurs in 27% to 36% of patients with cirrhosis. Studies have demonstrated that hyponatremia not only increases the susceptibility to SBP but also worsens the prognosis once the infection develops, with a higher risk of septic shock and death [10].

Given its association with multiple complications, hyponatremia has been established as an important prognostic indicator in patients with DCLD. It has been incorporated into the Model for End-Stage Liver Disease (MELD) score, which is widely used to assess the severity of liver disease and prioritize liver transplant candidates. Patients with low serum sodium levels have been shown to have worse outcomes, both in terms of survival while awaiting transplantation and in post-transplant recovery. Research suggests that the addition of sodium to the MELD score significantly improves its predictive accuracy, particularly in identifying high-risk patients. Moreover, hyponatremia has been associated with a higher likelihood of requiring emergency liver transplantation due to the rapid deterioration of liver function [11].

Hyponatremia within the sphere of decompensated chronic liver disease (DCLD) plays a pivotal role in the disease's progression and its associated complications, yet it is frequently overlooked and inadequately addressed. Grasping its prevalence and repercussions is vital for enhancing patient outcomes, as timely intervention can diminish morbidity, mortality, and impact liver transplant eligibility and success. This study aims to address the clinical significance of hyponatremia in DCLD. Given the close relationship between hyponatremia and cirrhosis-related complications, including hepatic encephalopathy, hepatorenal syndrome, and ascites, addressing this electrolyte imbalance is crucial for improving clinical outcomes and enhancing the quality of life in patients with DCLD.

METHODOLOGY

The cross-sectional study was conducted at the Department of Internal Medicine, Allama Iqbal Memorial Teaching Hospital, Sialkot, Pakistan, from 1st January 2023 to 30th June 2023. Ethical approval was obtained from the Institutional Review Board Approval no. 17/REC/KMSMC. The study included 120 patients aged 15-75 years with decompensated chronic liver disease (DCLD), recruited using non-probability consecutive sampling from the outpatient department. The sample size of 120 patients was calculated using Raosoft software, with an anticipated prevalence of hyponatremia in DCLD of 36.09% based on previous literature, a 95% confidence level, 80% power, and an expected response rate of 90% to account for non-participation ,and informed consent was secured from all participants [12].

The inclusion criteria were patients aged 15 to 75 years with a clinical diagnosis of DCLD, while exclusion criteria included diuretic use within the past month and non-viral liver disease (as documented in the medical records). Demographic data, clinical history, and blood samples were collected from all participants. Samples were sent to the laboratory of the hospital (Allama Iqbal Memorial Hospital, affiliated with Khawaja Safdar Medical College) for assessment of serum sodium level. Reports were assessed, and if the level was < 130 mmol/L, hyponatremia was labelled. Hyponatremia was defined as a serum sodium level below 130 mmol/L [1].

The severity of chronic liver disease was assessed using the Child-Pugh classification. This scoring system evaluates five clinical and laboratory parameters: serum bilirubin, serum albumin, prothrombin time (or INR) serum bilirubin is graded as <2 mg/dL, 2–3 mg/dL, and >3 mg/dL; serum albumin as >3.5 g/dL, 2.8–3.5 g/dL, and <2.8 g/dL; and INR (or prolongation of prothrombin time) as <1.7 (or <4 sec), 1.7–2.3 (or 4–6 sec), and >2.3 (or >6 sec). Each of these levels is scored from 1 to 3 points, respectively. Together with clinical grading of ascites and encephalopathy, they provide the total Child-Pugh score, ascites, and hepatic encephalopathy. Each parameter was assigned a score between 1 (mild) and 3 (severe), and the total score determined the classification:

·Child-Pugh Class A (5-6 points): Well-compensated liver

disease.
·Child-Pugh Class B (7-9 points): Significant functional compromise.
·Child-Pugh Class C (10-15 points): Decompensated liver disease.

Patients were systematically assessed for complications, including ascites (ultrasound confirmation), hepatic encephalopathy (using West Haven criteria), spontaneous bacterial peritonitis (ascitic fluid analysis), variceal bleeding (upper gastrointestinal endoscopy), and hepatorenal syndrome (using International Ascites Club criteria), following standardized protocols. Blood samples were collected using a 3cc syringe and analyzed within two hours to maintain sample integrity. A validated, pre-tested proforma was used for data collection, with all data collectors trained to ensure consistency. The proforma was pilot-tested on a sample of 10 DCLD patients who met the inclusion criteria [13,14].

Feedback from these patients was used to refine the proforma, ensuring that all questions were relevant, easy to interpret, and captured the necessary data without confusion or redundancy. Analysis was performed using SPSS version 22, with descriptive statistics calculated for continuous variables (mean and standard deviation) and categorical variables (frequencies and percentages). Complications were compared in patients with or without hyponatremia using the chi-square test. Effect modifiers such as age, gender, BMI, duration of symptoms, and Child-Pugh B or C were controlled for by stratification, dividing the study population into subgroups. Post-stratification, the chi-square test was used to compare the frequencies of hyponatremia and complications across stratified groups, with p-values ≤ 0.05 considered significant.

RESULTS

Table-I: Demographic profile .

Variables	Categories	n (%)
Gender	Males	89 (74.17)
	Females	31(25.83)
Age	Mean Age (years)	43.23 ± 15.97

Table-II: Clinical profile and serum sodium status of cirrhotic patients.

Variables	Categories	n(%)
Child Pugh classification	Child-Pugh Class B	92 (76.67)
	Child-Pugh Class C	28 (23.33)
Sodium levels	Patients with Hyponatremia	48 (40)
	Patients with Normal Sodium Levels	72 (60)
Mean Serum Sodium Level (mg/dL)	Mean±SD	134.88±15.30
Mean Duration of Cirrhosis (years)	Mean±SD	5.38 ± 2.92

Table-III: Distribution of clinical complications in patients (n=120).

Variables	Clinical Complications	
	Yes n(%)	No n(%)
Ascites	68 (56.7)	52 (43.3)
Hep.enceph	38 (31.7)	82 (68.3)
Hepatorenal Syndrome	5 (4.2)	115 (95.8)
Variceal bleed	15 (12.5)	105 (87.5)
SBP(spontaneous bacterial peritonitis)	28 (23.3)	92 (76.7)

Table-I shows that the mean age of patients was 43.23 ± 15.97 years. The study population consisted of 89 males (74.17%) and 31 females (25.83%).
The mean duration of cirrhosis was 5.38 ± 2.92 years. According to Child-Pugh classification, 92 patients (76.67%) were in Class B and 28 patients (23.33%) were in Class C. The mean serum sodium level was 134.88 ± 15.30 mg/dL, with 48 patients (40%) having hyponatremia and 72 patients (60%) having normal sodium levels in table-II.
Table-III indicates that ascites was the most common complication, affecting 68(56.7%) of the patients, followed by hepatic encephalopathy 38(31.7%) and SBP 28(23.3%). In contrast, variceal bleeding and hepatorenal syndrome were less common, with prevalence rates of 15(12.5%) and 5(4.2%), respectively.

Table-IV: Gender-stratified comparison of complications in cirrhosis patients with and without hyponatremia.

Variables	Gender	Hyponatremia		Total	P-value
		Yes n(%)	No n(%)	n(%)	
Ascites	Male	45(37.5)	44(36.7)	89(74.2)	0.022*
	Female	23(19.2)	8(6.7)	31(25.8)	
Hepatic encephalopathy	Male	27(22.5)	62(51.7)	89(74.2)	0.596*
	Female	11(9.2)	20(16.7)	31(25.8)	
Hepatorenal syndrome	Male	3(2.5)	86(71.7)	89(74.2)	0.603**
	Female	2(1.7)	29(24.2)	31(25.8)	
Variceal bleed	Male	9(7.5)	80(66.7)	89(74.2)	0.211**
	Female	6(5)	25(20.8)	31(25.8)	
Spontaneous bacterial peritonitis	Male	20(16.7)	69(57.5)	89(74.2)	0.705*
	Female	8(6.7)	23(19.2)	31(25.8)	

***p-value calculated using chi square .**
**** p-value calculated using fisher exact test.**

Table-IV summarizes gender-stratified outcomes in 120 cirrhotic patients: hyponatraemia associated significantly with ascites p = 0.022, affecting 45/89 males (37.5 %) and 23/31 females (19.2 %) compared with 44/89 normonatraemic males (36.7 %) and 8/31 normonatraemic females (6.7 %); by contrast, the distributions of hepatic encephalopathy (p = 0.596), hepatorenal syndrome (p = 0.603), variceal bleeding (p = 0.211), and spontaneous bacterial peritonitis (p = 0.705) did not differ significantly between hyponatraemic and normonatraemic groups in either sex.

DISCUSSION

Hyponatremia, a frequent electrolyte disorder in decompensated chronic liver disease (DCLD), indicates impaired renal water and sodium regulation. It significantly influences the severity of liver-related complications and impacts patient outcomes [1]. In this study, the mean age of patients with Decompensated Chronic Liver Disease (DCLD) was 43.23± 15.97 years, which is slightly lower than a U.S. study, which showed an increase from 56.0 to 59.1 suggesting earlier onset and different risk factors. The U.S. cohort had higher alcohol-related liver cirrhosis among those under 40, with an annual increase of 2.32%, whereas in the studied population, other factors such as viral hepatitis may play a larger role, contributing to the earlier onset of DCLD [14].

The gender distribution in the current study revealed a predominance of male participants (74.17%), aligns with findings from a study on hyponatremia in decompensated chronic liver disease, which reported 91 (75.8%) males and 29 (24.2%) females. This similarity highlights the trend of male predominance, possibly due to differences in risk factors or the progression of liver disease between genders [15].

The mean duration of cirrhosis in the current study was 5.38 ± 2.92years, which is comparable to a study reported by Gangneung Asan Hospital and other institutions in South Korea, where the mean duration for patients with decompensated cirrhosis was found to be approximately 5.6 ± 3.1 years indicating a similar timeline for disease progression across different populations [16]. The results further emphasize that the chronicity of cirrhosis in our cohort aligns closely with that observed internationally, suggesting consistent disease progression patterns regardless of geographic location. It is also consistent with the findings from the Greek study conducted at a tertiary hospital in Crete, where the median time to decompensation was approximately 5.4 years. The relatively long duration (approximately 5.4 years) before decompensation suggests that many patients maintain a compensated state for a significant period, allowing time for potential interventions that could delay progression. This indicates a window of opportunity for effective management to prevent or postpone complications [17].

Most patients in this study were classified as Child-Pugh Class B (76.67%), with fewer in Class C (23.33%). This is consistent with findings from an international study by the British Society of Gastroenterology, which also reported a high proportion of patients in Class B, although the percentage varied depending on the population and setting of the study. In a multicenter evaluation conducted across Scotland, 40.6% of patients with decompensated liver disease were classified as Child-Pugh Class C, indicating a similar trend of disease severity between populations, suggesting that clinical intervention often occurs before reaching Class C [18]. These variations in Child-Pugh classification emphasize the importance of early diagnosis and effective management to prevent disease progression and improve patient outcomes. The differences in the proportion of patients in each Child-Pugh class may also reflect disparities in healthcare infrastructure, patient awareness, and availability of specialized care.

The mean serum sodium level in the study population was 134.88 mg/dL, with 40% of patients presenting with hyponatremia. At admission, patients with hyponatremia had a significantly lower survival rate (35%) compared to those with de novo or absent hyponatremia (70%) [19]. A multi-center retrospective observational study" by Yamana et al., revealed that the mean sodium level among patients with cirrhosis was reported as 140 mEq/L (322 mg/dL) with an interquartile range of 138–141 mEq/L. which falls within the normal range. Hyponatremia is often a marker of severe portal hypertension and impaired renal function, both of which contribute to the worsening of liver disease .Patients with normal sodium levels generally had better survival outcomes, with untreated esophageal varices, age, and liver cancer as significant risk factors for mortality [20]. The prevalence of hyponatremia in the current study highlights the need for effective interventions to manage electrolyte imbalances and prevent complications in patients with DCLD.

Ascites was significantly more prevalent in patients with hyponatremia compared to those with normal sodium levels. Specifically, ascites was observed in 45(37.5)of males with hyponatremia versus 44(36.7)without hyponatremia (p = 0.022), and in 23(19.2) of females with hyponatremia compared to 8(6.7)without hyponatremia (p = 0.022). This finding aligns with the results reported by Nakamura et al. (2023), where patients with lower sodium levels (<139 mEq/L) had a significantly increased incidence of portal hypertension (PHT)-related events, including ascites [21]. Hepatic encephalopathy (HE) was also more frequent among patients with hyponatremia. In males, 27(22.5)of those with hyponatremia experienced HE compared to 62(51.7) without hyponatremia (p = 0.596), while in females, 11(9.2) of those with hyponatremia had HE compared to 20(16.7) without. These results align with findings by Younas A et al., hepatic encephalopathy (HE) was observed in 176 patients, representing 67.7% of the study population. Among the 96 patients with hyponatremia, 82 (87.5%) suffered from HE, with a significant association indicated by a p-value of less than 0.001 [22].

Hepatorenal syndrome (HRS), a serious complication of cirrhosis, was more frequently observed in patients with hyponatremia. Among males with hyponatremia, 3(2.5) developed HRS, while 86(71.7) of the males without hyponatremia experienced this condition (p = 0.603), indicating a statistically insignificant association between hyponatremia and the occurrence of HRS in males. In females, 2(1.7)of those with hyponatremia developed HRS, compared to 29(24.2) without hyponatremia, the association was not statistically significant. This finding is consistent with a study conducted in India reported a substantial 64% of Decompensated Chronic Liver Disease (DCLD) patients develop HRS, indicating a strong link between advanced liver dysfunction and HRS[23]. The systemic vasodilation and reduced renal perfusion associated with hyponatremia contribute to the increased risk of HRS in these patients[24].

Variceal bleeding was another complication found to be significantly more prevalent in patients with hyponatremia. In males, 9(7.5) of those with hyponatremia experienced variceal bleeding compared to 80(66.7) without hyponatremia (p = 0.211). In females, 6(5) of those with hyponatremia had variceal bleeding compared to 25(20.8) without hyponatremia. This finding is supported by a study conducted in India that showed by 29.33% of patients in your data experiencing gastrointestinal bleeding. Patients with lower sodium levels had a significantly higher mean MELD score (18.89 ± 6.70) compared to those with sodium levels between 131-135 meq/L (13.17 ± 4.40) and > 135 meq/L (10.90 ± 2.95), with a statistically significant difference (p-value 0.001). Mortality was also higher in patients with sodium < 130 meq/L (18.52%), compared to 7.69% in those with sodium 131-135 meq/L and 4.55% in patients with sodium > 135 meq/L, highlighting the increased severity of complications in hyponatremic patients[25].

Spontaneous bacterial peritonitis (SBP) was significantly more common among males with hyponatremia, with 20(16.7) affected compared to 69(57.5) without hyponatremia (p = 0.705). Among females, 8(6.7) of those with hyponatremia had SBP compared to 23(19.2) without hyponatremia. A study conducted in Pakistan suggested the frequency of hyponatremia in patients with decompensated cirrhosis was found to be 33.1%. Among these patients, 22.88% had spontaneous bacterial peritonitis (SBP). However, the association between hyponatremia and SBP yielded a p-value of 0.667, indicating that the relationship between hyponatremia and an increased risk of SBP was not statistically significant [26].

The link between hyponatremia and various complications such as ascites, hepatic encephalopathy, hepatorenal syndrome, variceal hemorrhage, and spontaneous bacterial peritonitis highlights the critical necessity for swift detection and treatment of hyponatremia in individuals facing decompensated cirrhosis. By integrating serum sodium concentrations into prognostic frameworks like the MELD-Na score, the capacity to forecast outcomes in these patients has significantly advanced, especially concerning survival rates and the requirement for liver transplants[27].

Furthermore, the observed gender discrepancies in the occurrence of complications, particularly the elevated instances of ascites and variceal bleeding in females with hyponatremia, call for deeper exploration to comprehend the underlying factors and customize management strategies accordingly.

CONCLUSION

In conclusion hyponatremia is a common and clinically relevant challenge in patients with Decompensated Chronic Liver Diseases (DCLD), linked to a heightened prevalence of various complications and increased rates of morbidity and mortality. Our results resonate with existing research, emhpasizing the urgent need for prompt identification and effective intervention of hyponatremia to improve clinical outcomes and enhance the quality of life for individuals with decompensated cirrhosis.

Importantly, this research has uncovered notable gender-specific variations in the frequency of complications such as ascites and variceal bleeding, which are particularly frequent among females. These differences highlight the importance of additional studies to clarify the mechanisms that might drive these variations. Future inquiries should not only aim at formulating optimal approaches for preventing and treating hyponatremia but also take into account the distinct requirements and responses of different genders to refine management strategies more effectively.

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