Original Article

A Study of Electrolyte Imbalances in Patients withChronic Liver Disease

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Abstract:

Background: Chronic liver disease (CLD) is a significant global health problem marked by progressive hepatic injury and impaired liver function. One of the frequently overlooked yet clinically important complications of CLD is electrolyte imbalance. Electrolyte disturbances-particularly hyponatremia, hypokalemia and hypocalcemia-are common in advanced liver disease. **Objective:** This study evaluate the patterns and prevalence of electrolyte disturbances in CLD patients, aiming to establish their association with disease severity. **Methods:** This observational cross-sectional study was conducted at Department of Hepatology, Comilla Medical College, Cumilla, Bangladesh, from January 2023 to December 2023. A total of 100 patients diagnosed with chronic liver disease (CLD) were included. Statistical analysis was performed using SPSS version 26.0. **Result:** In this study of 100 chronic liver disease patients, electrolyte imbalances were found to be highly prevalent, with hyponatremia in 58%, hypokalemia in 36%, and hypocalcemia in 41% of cases. These disturbances were more common in patients with advanced liver dysfunction (Child-Pugh Class B and C) and were significantly associated with complications such as ascites and hepatic encephalopathy. **Conclusion:** This study highlights the high prevalence of electrolyte imbalances-particularly hyponatremia, hypokalemia, and hypocalcemia-among patients with chronic liver disease. These abnormalities were significantly associated with the severity of liver dysfunction, as graded by the Child-Pugh classification, and were more frequent in patients with complications like ascites and hepatic encephalopathy.

Keywords: Hyponatremia, Hypokalemia, Hypocalcemia, Chronic Liver Disease, Hepatic Encephalopathy

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Introduction:

Chronic liver disease (CLD) represents a significant global health burden, with millions affected by progressive hepatic dysfunction that can culminate in cirrhosis, liver failure, and death. It is associated with various systemic complications, among which electrolyte imbalances are particularly common and clinically significant. These disturbances often reflect the severity of liver dysfunction and are closely linked to patient outcomes,especially in those with advanced disease stages such as decompensated cirrhosis or hepatic encephalopathy[1]. Electrolyte abnormalities in CLD patients arise from a complex interplay of pathophysiological mechanisms,including portal hypertension, hypoalbuminemia, renal dysfunction, and the use of diuretics for ascites management. Hyponatremia is the most frequent and well-documented electrolyte disturbance, often resulting from hypervolemic dilution due to increased arginine vasopressin (AVP) secretion and impaired renal free water clearance[2]. Studies have shown that even mild hyponatremia is associated with increased mortality,risk of hepatic encephalopathy, and poor post-transplant outcomes[3,4]. Hypokalemia is another common electrolyte abnormality in CLD, usually exacerbated by secondary hyperaldosteronism and potassium-wasting diuretics like furosemide. It plays a

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critical role in precipitating hepatic encephalopathy and contributes to muscle weakness and cardiac arrhythmias[5]. Conversely, hyperkalemia may occur in the context of hepatorenal syndrome or with the use of potassium-sparing diuretics such as spironolactone[6]. Hypocalcemia and hypomagnesemia are also frequently observed in CLD, especially in alcohol related liver disease. These disturbances may be secondary to vitamin D deficiency, hypoalbuminemia, malabsorption, or renal losses. Such abnormalities are linked to neuromuscular irritability, bone disorders, and cardiovascular complications[7]. Magnesium plays a protective role in liver injury and fibrosis, and its deficiency may accelerate hepatic decompensation[8]. The pathogenesis of these electrolyte imbalances is further compounded by the altered neurohormonal regulation seen in cirrhosis. The activation of the renin -angiotensin-aldosterone system (RAAS), sympathetic nervous system, and non-osmotic release of AVP leadsto significant alterations in sodium and water homeostasis[9]. Moreover, systemic inflammation and infections such as spontaneous bacterial peritonitis can further exacerbate electrolyte derangements. Recent research emphasizes the prognostic utility of electrolyte levels in CLD patients. For instance, hyponatremia has been incorporated into the Model for End-Stage Liver Disease (MELD-Na) score, improving its ability to predict short-term mortality in cirrhotic patients awaiting liver transplantation[3]. Early detection and correction of electrolyte imbalances are, therefore, essential in managing cirrhosis and improving quality of life, reducing complications, and guiding therapeutic interventions. Despite the clinical relevance, data on the prevalence and patterns of electrolyte disturbances among CLD patients in different populations, especially in low- and middle-income countries, remain scarce. Factors such as etiology of liver disease (e.g., viral hepatitis, alcohol, NAFLD), nutritional status, socioeconomic background, and availability of healthcare services may significantly influence these imbalances[11]. Understanding local patterns and associated risk factors can aid in developing region-specific treatment protocols. This study aims to investigate the prevalence and types of electrolyte imbalances in patients with chronic liver disease, identify associated clinical features, and analyze their correlation with disease severity.

Methods:

This observational cross-sectional study was conducted at Department of Hepatology, Comilla Medical College,Cumilla, Bangladesh, from January 2023 to December 2023. A total of 100 patients diagnosed with chronic liver disease (CLD), based on clinical, biochemical, and radiological findings, were enrolled after obtaining informed consent. Patients with diagnosed chronic kidney disease, recent diuretic use (at least 7 days), malignancy, or other known causes of electrolyte disturbances were excluded. Detailed demographic and clinical data were recorded, and serum electrolyte levels-including sodium, potassium, and calcium-were measured. The severity of liver disease was graded using the Child-Pugh classification. Statistical analysis was performed using SPSS version 26.0, and results expressed as frequencies, percentages, means, and standard deviations. Ethical clearance was obtained from the institutional review board before the initiation of the study.

Results:

Table-I: Demographic and Clinical Characteristics of the Study Population(n=100)

Parameter	Value
Age (mean±SD)	48.6 ± 12.4years
Sex (Male:Female)	70:30
Etiology of CLD	**n(%)**
Hepatitis B	38(38)
Alcoholic liver disease	30(30)
Hepatitis C	15(15)
NASH	12(12)
Others	5(5)
Ascites	62(62)
Hepatic encephalopathy	27(27)
Upper GI bleeding (variceal)	22(22)

The majority of the study population was males (70%). Hepatitis B (38%) was the leading cause of CLD,followed by alcoholic liver disease (30%). Ascites (62%) was the most frequent complication, while hepatic encephalopathy and variceal bleeding were observedin 27% and 22% of patients, respectively. [Table-I]

Table-II: Distribution of Electrolyte Imbalances in CLD Patients(=100)

Electrolyte Imbalance	Number of Patients	Percentage (%)
Hyponatremia(<135 mEq/L)	58	58%
Hypokalemia(<3.5 mEq/L)	36	36%
Hyperkalemia(>5.0 mEq/L)	9	9%
Hypocalcemia(<8.5mg/dL)	41	41%
Hypomagnesemia(<1.7 mg/dL)	28	28%
Combined abnormalities	32	32%

Hyponatremia was the most common electrolyte abnormality, affecting 58% of patients. Hypokalemia (36%)and hypocalcemia (41%) were also prevalent. Combined disturbances involving multiple electrolytes were present in nearly one-third of the patients. [Table-II]

Table-III: Correlation between Electrolyte Imbalances and Child-Pugh Class(n=100)

Child-Pugh Class	Number of Patients	Hyponatremia (%)	Hypokalemia (%)	Hypocalcemia (%)
A	18	4(22.2%)	3(16.7%)	3(16.7%)
B	35	21(60%)	11(31.4%)	14(40%)
C	47	33(70.2%)	22(46.8%)	24(51.1%)
p-value	-	<0.001	0.015	0.003

A statistically significant increase in electrolyte imbalances was observed with advancing Child-Pugh class.Hyponatremia and hypocalcemia were especially prevalent in Class C patients, indicating a strong association with disease severity. [Table-III]

Table-IV: Association between Electrolyte Imbalances and Major Complications

Complication	Hyponatremia (n=58)	Hypokalemia (n=36)	Hypocalcemia (n=41)
Ascites(n = 62)	45(77.6%)	28(77.8%)	32(78.0%)
Hepatic encephalopathy (n=27)	22(81.5%)	19(70.4%)	23(85.2%)
Variceal bleeding (n=22)	15(68.2%)	10(45.5%)	13(59.1%)

Electrolyte abnormalities were significantly associated with complications such as ascites and hepatic encephalopathy. Over 80% of patients with encephalopathy had hyponatremia and hypocalcemia, suggesting their potential role in neurologic deterioration. [Table-IV]

Table-V: Severity of Hyponatremia among Affected Patients (n=58)

Sodium Level (mEq/L)	Number of Patients	Percentage(%)
130-134(Mild)	26	44.8%
125-129 (Moderate)	20	34.5%
<125(Severe)	12	20.7%

Among patents with hyponatremia, 34.5% had moderate and 20.7% had severe sodium depletion. These findings highlight the need for timely correction, especially in those at risk of neurological complications. [Table-V]

Table-VI: Frequency of Multiple Electrolyte Disturbances (n=100)

Combination of Electrolytes Affected	Number of Patients	Percentage (%)
Hyponatremia+Hypokalemia	21	21%
Hyponatremia+Hypocalcemia	18	18%
Hypokalemia+Hypomagnesemia	12	12%
All four(Na,K,Ca,Mg)	9	9%

Simultaneous disturbances in multiple electrolytes were common. Nearly one-fifth of patients had bothhyponatremia and hypokalemia, and 9% had disturbances in sodium, potassium, calcium, and magnesium together,complicating management strategies. [Table-VI]

Discussion:

This study investigated the prevalence and clinical significance of electrolyte imbalances in patients with chronic liver disease (CLD) and revealed several important findings. Electrolyte disturbances were highly prevalent, with hyponatremia 58% being the most common, followed by hypocalcaemia41%, hypokalemia 36%, and hypomagnesaemia 28%. These imbalances were significantly associated with disease severity (Child-Pugh class) and clinical complications such as ascites, hepatic encephalopathy, and variceal bleeding. The prevalence of hyponatremia 58% in our study is comparable to findings from Sarin et al., who reported hyponatremia in 61% of cirrhotic patients, with higher rates in those with complications like ascites and encephalopathy[12]. Similarly, a study by Kim et al. identified hyponatremia as a strong independent predictor of mortality and poor liver transplant outcomes, highlighting its prognostic importance[4]. In our study, the severity of hyponatremia increased with advancing Child-Pugh class, a finding supported by Bernardi et al.,who emphasized the role of systemic vasodilation and non-osmotic vasopressin release in cirrhosis-related hyponatremia[13]. Hypokalemia was observed in 36% of patients in our study, consistent with findings from Runyon, who noted that potassium loss is common in cirrhotic patients due to secondary hyperaldosteronism and diuretic therapy[5]. Our study further confirmed that hypokalemia was more common in patients with ascites (77.8%) and hepatic encephalopathy (70.4%), a trend also described by Ginès et al., who emphasized its role in triggering encephalopathy through ammonia retention and altered membrane excitability[6]. Hypocalcemia was found in 41% of our CLD patients, a rate slightly higher than the 32-38% reported by Choudhary and Sarin[12]. This may be attributed to reduced albumin levels, malabsorption, and vitamin D deficiency commonly observed in advanced liver disease. Notably, 85.2% of patients with hepatic encephalopathy in our study had hypocalcemia, suggesting a possible contributory role. Similar findings were reported by Swaminathan et al., who emphasized the neurological implications of calcium imbalance in CLD patients[14]. Hypomagnesemia (28%) was another notable finding. Geiger and Wanner

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described magnesium as a "forgotten electrolyte" in CLD, often overlooked despite its protective role in hepatocyte function and membrane stability[8]. Our data also show that the frequency and severity of electrolyte abnormalities increased significantly with advancing Child-Pugh classification. Patients in Class C had the highest incidence of hyponatremia (70.2%), hypokalemia (46.8%), and hypocalcemia (51.1%). These findings corroborate those of Fernández et al., who linked electrolyte derangements with decompensation and systemic inflammation in cirrhotic patients[10]. Multiple electrolyte abnormalities were present in 32% of the study population. The most common combination was hyponatremia with hypokalemia (21%), which mirrors patterns noted by Rahman et al. in their Bangladeshi cohort of liver disease patients[12]. Such combinations significantly complicate fluid-electrolyte management and increase the risk of hospital readmission, as highlighted by Schrie[9]. The strong association between electrolyte disturbances and complications such as ascites, hepatic encephalopathy,and variceal bleeding reflects the interconnected nature of hepatic, renal, and neuroendocrine systems. The incorporation of serum sodium into prognostic models like MELD-Na further emphasizes the clinical relevance of these abnormalities, as supported by Kim et al.[4].

Conclusion:

This study highlights the high prevalence of electrolyte imbalances-particularly hyponatremia, hypokalemia, and hypocalcemia-among patients with chronic liver disease. These abnormaities were significantly associated with the severity of liver dysfunction, as graded by the Child-Pugh classification, and were more frequent in patients with complications like ascites and hepatic encephalopathy.

Limitations of the study:

The study was conducted in a single centre with a small sample size. Biopsy and histological confirmation CLD is not confirmed. So, the results may not represent the whole community.

Recommendation:

Routine monitoring and early correction of electrolyte imbalances should be an integral part of the management protocol for patients with chronic liver disease. Clinicians should be particularly vigilant in patients with advanced disease (Child-Pugh Class B and C) and those presenting with complications such as ascites and hepatic encephalopathy. Early intervention may help prevent further deterioration and improve both quality of life and prognosis.

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