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Frequency of Electrolyte Abnormalities in Cirrohsis Patient Presenting with Hepatic Encephalopathy

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ABSTRACT

Background: Liver cirrhosis is a chronic progressive liver disease which can develop into several complications such as hepatic encephalopathy. Hepatic encephalopathy (HE) is a serious neuropsychiatric complication of liver dysfunction that is often triggered by infection, gastrointestinal bleeding, constipation, renal insufficiency and electrolyte imbalance. Severe disease and further deterioration of cerebral function may occur in patients with cirrhosis due to electrolyte disturbances, particularly hyponatremia and hypokalemia. **Objective:** To determine the frequency of electrolyte abnormalities among cirrhosis patients presenting with hepatic encephalopathy. **Methods:** This descriptive cross-sectional study was conducted at Lahore General Hospital, Lahore, Pakistan from January 2025 to June 2025. There were 75 patients who had a diagnosis of liver cirrhosis and hepatic encephalopathy. A structured proforma was used to record demographic data, clinical history, aetiology of cirrhosis, grade of hepatic encephalopathy, precipitating factors and hospital outcomes. Blood samples were taken on admission for renal function tests, liver function tests, INR, serum sodium, serum potassium, serum chloride, serum calcium, and serum magnesium. An electrolyte abnormality was present if any electrolyte level in the serum was outside the laboratory normal reference range. The analysis of data was carried out by SPSS version 25. Qualitative variables were presented in terms of frequencies and percentages, and quantitative variables were presented as mean and standard deviation. With a p value less than 0.05 it was deemed to be statistically significant. **Results:** The mean age of patients was 52.6 ± 11.4 years. Out of 75 patients, 48 (64.0%) were males and 27 (36.0%) were females. Most patients had advanced liver disease, with 40 (53.3%) patients classified as Child-Pugh class C. Grade II hepatic encephalopathy was the most common presentation, observed in 30 (40.0%) patients. Overall, 56 (74.7%) patients had at least one electrolyte abnormality. Hyponatremia was the most frequent abnormality, present in 42 (56.0%) patients, followed by hypokalemia in 27 (36.0%), hypocalcemia in 20 (26.7%), and hypomagnesemia in 15 (20.0%) patients. Electrolyte abnormalities were significantly associated with higher grades of hepatic encephalopathy (p = 0.031) and increased in-hospital mortality (p = 0.046). **Conclusion:** Electrolyte abnormalities were highly frequent among cirrhosis patients presenting with hepatic encephalopathy. Hyponatremia was the most common abnormality, followed by hypokalemia. The presence of electrolyte imbalance was associated with more severe hepatic encephalopathy and poor hospital outcome. Routine assessment and timely correction of serum electrolytes should be part of the initial management of cirrhotic patients presenting with hepatic encephalopathy.

INTRODUCTION

Liver cirrhosis is the final stage of chronic liver injury, showing progressive fibrosis, distortion of normal liver architecture and development of regenerative nodules. Liver failure is associated with a variety of complications including ascites, variceal bleeding, spontaneous bacterial peritonitis, hepatorenal syndrome, coagulopathy and hepatic encephalopathy. Of these complications, hepatic

encephalopathy (HE) is one of the more clinically significant ones as it directly affects consciousness, cognition, hospital stay, quality of life and survival [1-4].

Hepatic encephalopathy is a (reversible or partially reversible) neuropsychiatric syndrome that occurs in patients with acute or chronic liver disease or failure. Can include mild sleep disturbance, irritability, poor concentration, disorientation, asterixis, drowsiness, stupor or coma. Hepatic encephalopathy is usually classified as grade I to grade IV based on the degree of neurological dysfunction. In clinical practice, many patients will have moderate to severe encephalopathy when they come to the doctor, and the condition will be aggravated by failure to identify and treat the precipitating factors early [5-7].

Hepatic encephalopathy is a complex phenomenon. It's pathogenesis includes several mechanisms, such as increased production of ammonia, impaired hepatic detoxification, portosystemic shunting, systemic inflammation, infection, gastrointestinal bleeding, constipation, dehydration, renal dysfunction, and electrolyte disturbances. However, of these factors electrolyte imbalance is important because it is common, measurable and correctable. Therefore early detection of electrolyte disturbances can potentially help to improve neurologic status and limit complications [8-11].

The most common electrolyte disturbance in cirrhosis is hyponatremia. It is generally attributed to a decrease in the effective volume of arterial blood, activation of neurohormonal mechanisms, augmented production of antidiuretic hormone and diminished renal clearance of free water. Hyponatremia in cirrhotic patients may exacerbate cerebral edema and may make them more susceptible to hepatic encephalopathy. Patients with low serum sodium levels are likely to have advanced liver disease, ascites, renal dysfunction, and a higher risk of mortality in hospital [12-14].

Also, clinically important, is the potassium disturbance in hepatic encephalopathy. Low blood potassium levels can be caused by inadequate intake, vomiting, diarrhea, use of lactulose, and diuretics. It can exacerbate encephalopathy by enhancing renal ammonia production and producing alkalosis to allow ammonia to cross the blood-brain barrier. Patients with renal dysfunction, advanced liver disease, and those taking potassium sparing diuretics are also at risk of hyperkalemia. Hence, patients with cirrhosis should be monitored for both low and high potassium [15].

Hypocalcemia, hypomagnesemia, chloride imbalance and phosphate disturbances are other electrolyte abnormalities that can occur in cirrhotic patients. These abnormalities can be due to malnutrition, renal failure, abnormal hormone levels, diuretics, infection and poor overall health. The assessment of more than one electrolyte is more useful for understanding the biochemical status of the patient with hepatic encephalopathy; although sodium and potassium are most often stressed [16].

Electrolyte abnormalities can not only be precipitating factors in liver cirrhosis, but can also be markers of more severe disease and poor prognosis. The frequency and pattern of electrolyte disturbance may be different depending on the degree of cirrhosis, diuretics, ascites, infection, renal disease and local patient characteristics. Hence, it is important to have local data to help the clinicians decide on which electrolyte disturbances are common in the cirrhotic patient with hepatic encephalopathy [17].

This study was conducted to determine the frequency of electrolyte abnormalities among patients with liver cirrhosis presenting with hepatic encephalopathy. The study also assessed the pattern of individual electrolyte disturbances and their association with the grade of hepatic encephalopathy and hospital outcome. The findings may help improve early biochemical assessment and support timely correction of reversible metabolic factors in cirrhotic patients.

METHODOLOGY

This descriptive cross-sectional study was conducted at Lahore General Hospital, Lahore, Pakistan from January 2025 to June 2025. The study was designed to determine the frequency of electrolyte abnormalities among patients with liver cirrhosis presenting with hepatic encephalopathy. All eligible patients admitted through the emergency department, medical outpatient department, or medical ward during the study period were evaluated according to the study protocol.

In this study, 75 patients with liver cirrhosis with hepatic encephalopathy were included. The number of patients available to participate in the study during the selected time frame was used to determine the sample size. Patients were admitted with clinical/radiological evidence of cirrhosis, ≥18 years of age and clinical features of hepatic encephalopathy. Chronic liver disease was diagnosed based on altered level of consciousness, disorientation, sleep disturbance, personality changes, asterixis or coma. Hepatic encephalopathy was graded as per the standard clinical grading as grade I, II, III, and IV.

Patients with acute liver failure, chronic kidney disease requiring dialysis, uncontrolled diabetes mellitus (U.D.M.) with ketoacidosis, severe dehydration (S.D.) other than as a result of liver disease and missing laboratory results were omitted. Patients who opted out were excluded from the study as well.

Informed consent was taken and a predesigned proforma was used to record demographic and clinical data. Data collected consisted of demographics such as age, sex, length of cirrhosis, etiology of cirrhosis, previous history of hepatic encephalopathy, diuretics use, ascites, jaundice, gastrointestinal bleeding, constipation, infection and other factors that might precipitate hepatic encephalopathy. Liver disease severity was determined by clinical and laboratory parameters and the Child-Pugh score.

Venous blood samples were taken at the time of admission from each of the patients that were enrolled, prior to any electrolyte correction. Laboratory tests performed included serum sodium, serum potassium, serum chloride, serum calcium, serum magnesium, serum creatinine, blood urea, serum bilirubin, alanine aminotransferase, aspartate aminotransferase, serum albumin, prothrombin time, international normalized ratio, and complete blood count. Standard laboratory methods were used in the hospital laboratory for serum electrolyte measurements.

Electrolyte abnormality was considered to be any serum

electrolyte level that was not in the hospital laboratory normal range. Serum calcium, chloride and magnesium levels were also noted based on the reference range values of the hospital laboratory: Hypocalcemia, less than 2.5 mmol/L; Hyperkalemia, greater than 5.0 mmol/L; Hypomagnesemia, less than 1.4 mmol/L; and Chloride abnormalities, less than 95 mmol/L or greater than 110 mmol/L. Patients with more than one abnormal electrolyte level were included as mixed electrolyte abnormality.

Electrolyte abnormalities were the primary outcome studied in the cirrhotic patients who presented with HE. Other secondary variables included the type of electrolyte disturbance, grade of hepatic encephalopathy, Child-Pugh class, precipitating factors, duration of hospital stay, improvement, and in-hospital mortality.

All data collected were entered and analysed in SPSS version 25. Continuous variables like age, serum sodium, serum potassium, serum calcium, serum magnesium, serum bilirubin, serum albumin, and INR were given as mean and standard deviation. Qualitative variables like gender, etiology of cirrhosis, Child-Pugh class, grade of hepatic encephalopathy, electrolyte abnormality, and mortality were shown as frequency and percentage. The association between grade of hepatic encephalopathy, Child-Pugh class and mortality with electrolyte abnormalities was determined by Chi-square test. A p-value of <0.05 was regarded as being statistically significant.

RESULTS

The study included 75 patients who were diagnosed with liver cirrhosis and had hepatic encephalopathy. Patients' ages ranged from 28 to 76 years and the mean age was 52.6 ± 11.4 years. The average age of the patients was 52 years. Male predominance (48/75, 64.0%) was observed in 48 patients and female (27/75, 36.0%).

Table 1

Baseline Demographic Characteristics of Patients

Variable		Frequency (n)	Percentage (%)
Age group			
20–40 years		12	16.0
41–60 years		43	57.3
>60 years		20	26.7
Gender	Male	48	64.0
	Female	27	36.0
Duration of cirrhosis	<1 year	15	20.0
	1–5 years	39	52.0
	>5 years	21	28.0

The most prevalent etiology of cirrhosis was hepatitis C virus infection with 38 (50.7%) patients and hepatitis B virus infection with 16 (21.3%) patients. 13 (17.3%) patients had cryptogenic cirrhosis and 8 (10.7%) had non-alcoholic fatty liver disease.

Table 2

Etiology and Severity of Cirrhosis

Variable		Frequency (n)	Percentage (%)
Etiology of cirrhosis			
Hepatitis C		38	50.7
Hepatitis B		16	21.3
Cryptogenic		13	17.3
NAFLD		8	10.7
Child-Pugh class	Class A	6	8.0
	Class B	29	38.7
	Class C	40	53.3

Regarding the severity of liver disease, 40 (53.3%) patients were in Child-Pugh class C, 29 (38.7%) were in class B and 6 (8.0%) were in class A. This reveals the advanced liver disease at presentation in majority of patients. The most common presentation was grade II hepatic encephalopathy, seen in 30 (40.0%) patients, followed by grade III hepatic encephalopathy, seen in 23 (30.7%) patients. Twelve (16.0%) patients had grade I hepatic encephalopathy and 10 (13.3%) patients had grade IV hepatic encephalopathy.

Table 3

Distribution of Hepatic Encephalopathy Grades

Grade of hepatic encephalopathy	Frequency (n)	Percentage (%)
Grade I	12	16.0
Grade II	30	40.0
Grade III	23	30.7
Grade IV	10	13.3

Cirrhotic patients with HE were prone to electrolyte disturbance. Of the 75 patients, 56 (74.7%) were found to have any (at least one) electrolyte abnormality and 19 (25.3%) had no electrolyte abnormality. The most common abnormality was hyponatremia (42 [56.0%]) followed by hypokalemia (27 [36.0%]). Twenty (26.7%) patients had hypocalcemia and 15 (20.0%) had hypomagnesemia. Hyperkalemia and hypernatremia were less common, reported in 6 (8.0%) and 3 (4.0%) patients, respectively.

Table 4

Frequency of Electrolyte Abnormalities

Electrolyte abnormality	Frequency (n)	Percentage (%)
Any electrolyte abnormality	56	74.7
Hyponatremia	42	56.0
Hypokalemia	27	36.0
Hypocalcemia	20	26.7
Hypomagnesemia	15	20.0
Hyperkalemia	6	8.0
Hypernatremia	3	4.0
No electrolyte abnormality	19	25.3

The mean serum sodium level was 130.8 ± 6.2 mmol/l and mean serum potassium level was 3.6 ± 0.7 mmol/l. Mean serum calcium level was 8.1 ± 0.6 mg/dL and the mean serum magnesium level was 1.7 ± 0.4 mg/dL.

Table 5
Mean Electrolyte Levels among Study Participants

Laboratory parameter	Mean ± SD
Serum sodium (mmol/L)	130.8 ± 6.2
Serum potassium (mmol/L)	3.6 ± 0.7
Serum chloride (mmol/L)	97.4 ± 5.8
Serum calcium (mg/dL)	8.1 ± 0.6
Serum magnesium (mg/dL)	1.7 ± 0.4
Serum creatinine (mg/dL)	1.3 ± 0.5
Serum bilirubin (mg/dL)	3.8 ± 1.6
Serum albumin (g/dL)	2.7 ± 0.5
INR	1.8 ± 0.4

There was a strong correlation between the severity of HE and electrolyte disturbances. In 8(66.7%) patients with grade I, 20(66.7%) patients with grade II, 18(78.3%) patients with grade III and 10(100%) patients with grade IV hepatic encephalopathy, electrolyte disturbance was present. The association between the two was statistically significant (p=0.031).

Table 6
Association of Electrolyte Abnormalities with Grade of Hepatic Encephalopathy

Grade of hepatic encephalopathy	Electrolyte abnormality present n (%)	Electrolyte abnormality absent n (%)	p-value
Grade I	8 (66.7)	4 (33.3)	
Grade II	20 (66.7)	10 (33.3)	
Grade III	18 (78.3)	5 (21.7)	
Grade IV	10 (100.0)	0 (0.0)	0.031

Patients with advanced hepatic encephalopathy also had a higher prevalence of hyponatremia. It was present in 5 (41.7%) patients with grade I, 14 (46.7%) with grade II, 15 (65.2%) with grade III, and 8 (80.0%) with grade IV hepatic encephalopathy. Statistically significant hyponatremia scores and worsening grade of hepatic encephalopathy was seen with a p-value of 0.028.

Table 7
Association of Hyponatremia with Grade of Hepatic Encephalopathy

Grade of hepatic encephalopathy	Hyponatremia present n (%)	Hyponatremia absent n (%)	p-value
Grade I	5 (41.7)	7 (58.3)	
Grade II	14 (46.7)	16 (53.3)	
Grade III	15 (65.2)	8 (34.8)	
Grade IV	8 (80.0)	2 (20.0)	0.028

The most common precipitating factor for hepatic encephalopathy was infection, seen in 22 (29.3%) patients; constipation was seen in 18 (24.0%) patients and gastrointestinal bleeding was seen in 13 (17.3%) patients. In 12 (16.0%) patients, electrolyte imbalance was an identifiable precipitating factor, and in 10 (13.3%) patients, poor drug compliance was noted.

Table 8
Precipitating Factors of Hepatic Encephalopathy

Precipitating factor	Frequency (n)	Percentage (%)
Infection	22	29.3
Constipation	18	24.0
Gastrointestinal bleeding	13	17.3
Electrolyte imbalance	12	16.0
Poor drug compliance	10	13.3

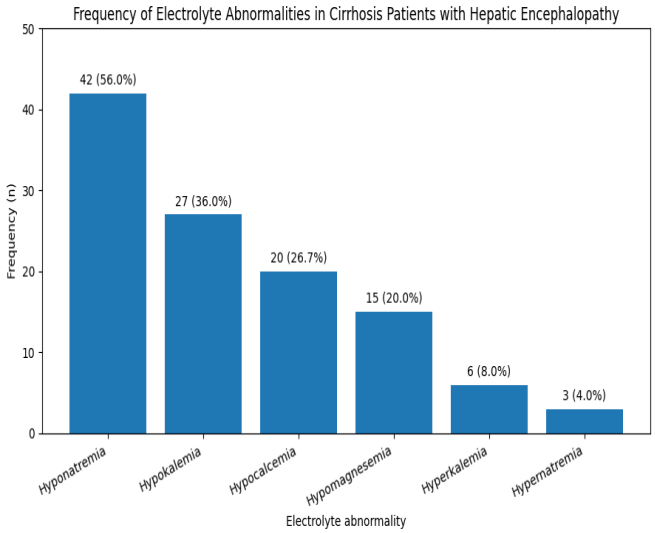
The outcome of the hospital was better for 61 (81.3%) patients, who were discharged, and worse for 14 (18.7%) who died in hospital. There was a higher mortality rate among patients with electrolyte abnormalities: 13 of 56 (23.2%) had electrolyte abnormalities and died, while 1 of 19 (5.3%) patients with no electrolyte abnormality died. This difference was statistically significant with a p-value of 0.046.

Table 9
Association of Electrolyte Abnormalities with Mortality

Electrolyte status	Survived n (%)	Died n (%)	p-value
Electrolyte abnormality present	43 (76.8)	13 (23.2)	
Electrolyte abnormality absent	18 (94.7)	1 (5.3)	0.046

In general, electrolyte abnormalities occurred in all cirrhotic patients with hepatic encephalopathy, and were very common. The most prevalent abnormality was hyponatremia, which was followed by hypokalemia and hypocalcemia. Electrolyte imbalance was more common the higher the grade of hepatic encephalopathy and was also a risk factor for in-hospital mortality.

Figure 1
Frequency of electrolyte abnormalities among cirrhosis patients presenting with hepatic encephalopathy. Hyponatremia was the most common abnormality, followed by hypokalemia, hypocalcemia, and hypomagnesemia.



DISCUSSION

The present study was conducted to determine the frequency of electrolyte abnormalities among patients with liver cirrhosis presenting with hepatic encephalopathy. In this study, electrolyte disturbance was observed in a large proportion of patients, as 56 out of 75 patients (74.7%) had at least one abnormal electrolyte level at the time of admission. This finding shows that electrolyte imbalance is a common clinical problem in cirrhotic patients with hepatic encephalopathy and should be actively looked for during initial assessment. Patients with chronic liver disease are already physiologically unstable due to impaired hepatic function, portal hypertension, altered renal handling of water and sodium, and frequent use of diuretics; therefore, even mild electrolyte changes may worsen neurological status.

The most frequent electrolyte abnormality in the current study was hyponatremia (42 [56.0%] patients). Clinically this result is important as sodium imbalance is closely associated with altered mental status in liver cirrhosis patients. With advanced cirrhosis, decreased effective arterial blood volume causes increased release of neurohormones such as renin-angiotensin-aldosterone (RAA) and antidiuretic hormone (ADH). These mechanisms cause water retention and dilutional hyponatremia. Low sodium can exacerbate cerebral edema in patients with hepatic encephalopathy and lead to confusion, lethargy and coma. Thus, serum sodium is an important biochemical parameter in cirrhotics who develop encephalopathy [18].

Hypokalemia was the second most common abnormality seen in this study with 27 (36.0%) patients having this disorder. This could be due to inadequate oral intake, vomiting, diarrhea, use of lactulose and/or diuretics. Hypokalemia is of special concern in hepatic encephalopathy, as it may stimulate the production of ammonia in the kidney and may play a role in the exacerbation of neurologic symptoms. Therefore, management of potassium imbalance is an integral component of these patients. Hypocalcemia and hypomagnesemia were also seen in significant number of patients, which could be attributed to poor nutrition, impaired absorption, renal dysfunction and advanced liver disease [19].

The present study also revealed that the frequency of electrolyte disturbances was found to be more in patients with more severe grade of hepatic encephalopathy. All 100% patients of grade IV hepatic encephalopathy had electrolyte disturbance and the association between the grade of encephalopathy with electrolyte disturbance was statistically significant with a p value of 0.031. Likewise, worsening encephalopathy grades were associated with increased hyponatremia and there was a significant association between disease severity and hyponatremia. The present data indicate that an electrolyte imbalance is not only a common laboratory finding, but may be related to the degree of neurological dysfunction in cirrhotic patients [19].

In this study, the majority of patients were of Child-Pugh class C (advanced liver disease). Advanced cirrhosis frequently carries with it the following complications that can affect electrolyte balance: ascites, reduced renal perfusion, infection, gastrointestinal bleeding and repeated hospital admissions. This is, therefore, a possible explanation of the frequent electrolyte abnormalities seen in the present study, as the patients included were all at an advanced stage of liver disease. This also emphasizes the importance of early screening of electrolytes and periodic monitoring in cirrhosis, particularly in those with decompensated liver disease [20].

In the current study, infection was the most frequent precipitating cause of hepatic encephalopathy followed by constipation and gastrointestinal bleeding. Electrolyte imbalance was one of the important precipitants noted in a significant proportion of cases. These findings are consistent with the hypothesis that most cases of hepatic encephalopathy are multifactorial and that a detailed attempt should be made to identify reversible triggers. The recognition and correction of the above conditions (infection, constipation, gastrointestinal bleeding, dehydration, and electrolyte disturbance) at an early stage may enhance the clinical recovery and possibly decrease mortality [10].

Patients who had electrolyte abnormalities had higher mortality rates. In the current study, 23.2% of the patients with electrolyte disturbance died, whereas only 5.3% of the patients without electrolyte abnormality died, and this difference was statistically significant ($p = 0.046$). This is a novel finding that suggests that electrolyte imbalance could be linked to poor outcome in the hospital setting in patients with cirrhosis who present with HE. Though the present study does not prove a causal link, the correlation does indicate that monitoring of electrolytes and prompt correction will be important to the patient's outcome [8, 21].

This study has some limitations. The study was carried out in a small and limited number of patients (75) and in one study centre, which may limit the applicability of the results for other patients and settings. The study was cross sectional in design and so long term outcome of the patients and recurrence of hepatic encephalopathy after discharge could not be determined. Not all patients had levels of serum ammonia measured, and treatment effects on correcting electrolyte abnormalities were not ascertained in detail. The study, although limited, offers some local information on the prevalence and significance of electrolyte disturbances in patients with HE and cirrhosis.

CONCLUSION

Electrolyte abnormalities were highly frequent among patients with liver cirrhosis presenting with hepatic encephalopathy. Hyponatremia was the most common abnormality, followed by hypokalemia, hypocalcemia, and hypomagnesemia. The frequency of electrolyte disturbance increased with the severity of hepatic encephalopathy and was significantly associated with higher in-hospital mortality. These findings suggest that serum electrolytes should be routinely assessed in all cirrhotic patients presenting with hepatic encephalopathy. Early detection and timely correction of electrolyte imbalance may help improve neurological status, reduce complications, and improve hospital outcome.

Regular monitoring of serum sodium, potassium, calcium, and magnesium is recommended in cirrhotic patients, especially those with advanced liver disease, ascites, infection, gastrointestinal bleeding, or diuretic use. Further multicenter studies with larger sample sizes are recommended to evaluate the role of electrolyte correction in reducing the severity and recurrence of hepatic encephalopathy.

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