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Research Article

Serum Electrolyte and Acid-Base Abnormalities in Hepatic Encephalopathy Associated with Chronic Liver Disease

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ABSTRACT
Background: Hepatic encephalopathy (HE) is a severe neuropsychiatric complication of chronic liver disease (CLD) frequently associated with electrolyte and acid–base disturbances that may worsen disease severity and prognosis.
Aim: To evaluate serum electrolyte and acid–base abnormalities in patients with hepatic encephalopathy associated with chronic liver disease and assess their correlation with disease severity and outcomes.
Materials and Methods: This prospective observational study was conducted in the Department of General Medicine, Baba Raghav Das Medical College and Nehru Hospital, Gorakhpur, Uttar Pradesh, from January to December 2024. A total of 115 adult patients with hepatic encephalopathy associated with chronic liver disease were included. Hepatic encephalopathy was graded using the West Haven criteria. Serum sodium, potassium, calcium, and arterial pH were measured at admission and discharge. Statistical analysis was performed using SPSS version26.0.
Results: Most patients belonged to the 41–50 year's age group (30.4%) and all were male. Alcohol-induced decompensated chronic liver disease was the predominant etiology (78.3%). Grade 2 HE was most common (37.4%). Significant associations were observed between etiology and diagnosis (p=0.005), admission calcium (p=0.009), admission pH (p=0.017), and all discharge biochemical parameters. Overall, 79.1% improved, while20.9% expired.
Conclusion: Electrolyte and acid–base abnormalities are important biochemical disturbances in hepatic encephalopathy. Early recognition, routine biochemical monitoring, and prompt management are essential to improve outcomes and reduce mortality.

Keywords: Hepatic Encephalopathy, Liver Cirrhosis, Electrolytes, Hyponatremia, Potassium, Calcium.

INTRODUCTION

Liver cirrhosis is characterized by irreversible changes in normal liver tissue, resulting in the degeneration of functioning liver cells and their replacement with fibrous connective tissue, resulting in chronic liver disease (CLD). [1]
Hepatic encephalopathy (HE) is a neuropsychiatric syndrome commonly seen in patients with advanced chronic liver disease (CLD) and cirrhosis. It results from the liver's inability to remove toxins, particularly ammonia, leading to cerebral dysfunction. The pathogenesis of HE is complex, involving multiple factors such as elevated ammonia levels, neuroinflammation, and alterations in neurotransmission. [2-4] However, one key aspect of HE is the profound disturbance of electrolyte balance and acid-base homeostasis, which plays a crucial role in the onset and progression of the condition.
Chronic liver disease (CLD) is characterized by electrolyte imbalances, which can worsen the severity of encephalopathy. Hyponatremia, a common electrolyte abnormality, is a result of impaired renal handling of water due to inappropriate secretion of antidiuretic hormone (ADH). It can worsen HE by affecting cerebral edema and the brain's osmotic balance. Hypokalemia is common in patients on diuretics and can exacerbate alkalosis and ammonia retention, further contributing to neurological dysfunction in HE. Hyponatremia can be caused by hypovolemia or hypervolemia, which occurs when the kidney cannot excrete solute-free water proportionate to the amount of free water ingested. [5-7]

Hypocalcemia in CLD patients can be caused by malnutrition, hypoalbuminemia, or vitamin D deficiency, leading to neuromuscular irritability and altered neuronal function. Metabolic alkalosis increases ammonia production, worsening HE, while metabolic acidosis may result from renal dysfunction and impaired lactate clearance, further compromising the patient's clinical status. [8,9]

Electrolyte disturbances, acid-base imbalances, and coagulopathy are key factors in the development and exacerbation of hepatic encephalopathy in chronic liver disease, understanding these biochemical abnormalities is crucial for managing complications. The study was conducted to assess the role of serum electrolyte and acid-base abnormalities in the progression of hepatic encephalopathy in chronic liver disease (CLD).

MATERIAL AND METHODS

The prospective observational study was conducted in the Department of General Medicine, Baba Raghav Das (B.R.D.) Medical College and Nehru Hospital, Gorakhpur, Uttar Pradesh, India, over a period of one year (January 2024 to December 2024). The study was started after receiving ethical clearance from the Institutional Ethics Committee. Using a purposive sampling technique, 115 patients fulfilling the inclusion criteria were selected for the study.

Inclusion Criteria

Adult patients aged 18 years and above, diagnosed cases of chronic liver disease confirmed by clinical, biochemical, and ultrasonographic findings (coarse liver echotexture, nodularity, portal hypertension, or ascites), presenting with hepatic encephalopathy at varying grades (I–IV) based on the West Haven criteria, willing to provide written informed consent (or through legal guardian for unconscious patients).

Exclusion Criteria

Patients with acute liver failure or acute-on-chronic liver failure, with history of neuropsychiatric illness unrelated to hepatic dysfunction (e.g., epilepsy, stroke, dementia), pregnant women, patients on renal replacement therapy or dialysis, on drugs known to alter electrolyte levels (diuretics, corticosteroids, or vasopressin antagonists) before admission with uncontrolled diabetes ketoacidosis, unwilling to provide informed consent.

All eligible patients were evaluated according to a standardized study protocol immediately upon admission. After obtaining informed consent, a detailed clinical history and comprehensive physical examination were performed to confirm the diagnosis of chronic liver disease and assess the severity of hepatic encephalopathy using the West Haven criteria. Demographic details, clinical presentation, precipitating factors, and relevant medical history were documented systematically. Venous and arterial blood samples were collected within two hours of admission under aseptic precautions. Approximately 5 mL of venous blood was obtained for biochemical investigations, including serum electrolyte estimation using an automated ion-selective electrode analyzer, liver function tests, renal function tests, coagulation profile, and hematological parameters. Arterial blood gas (ABG) analysis was performed using a calibrated blood gas analyzer to determine arterial pH, bicarbonate (HCO_3^-), partial pressure of carbon dioxide ($PaCO_2$), base excess, and lactate levels. Ultrasonography of the abdomen was conducted to identify cirrhotic liver morphology, portal hypertension, and ascites. Potential precipitating factors for hepatic encephalopathy, including infections, gastrointestinal bleeding, constipation, electrolyte disturbances, and medication history, were evaluated through clinical assessment and relevant laboratory investigations such as complete blood count, urine analysis and culture, and ascitic fluid examination where indicated. Patients were monitored daily throughout hospitalization for changes in neurological status, electrolyte imbalance, acid–base disturbances, and overall clinical outcome until discharge or death. All patients received standard institutional management for hepatic encephalopathy, including lactulose therapy, antibiotics, nutritional support, and correction of precipitating factors. No experimental interventions were administered during the study period.

Data was collected using a pre-validated case record form (CRF). All collected data were analyzed using SPSS (Statistical Package for the Social Sciences) version 26.0. Descriptive statistics such as mean, standard deviation, and percentage distribution were used for demographic and clinical variables. A p-value < 0.05 was considered statistically significant.

RESULT

Table 1: Baseline Demographic Characteristics of Study Participants (n = 115)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	≤ 30	13	11.3
	31 – 40	34	29.6
	41 – 50	35	30.4
	51 – 60	16	13.9
	61 – 70	13	11.3
	> 70	4	3.5
Sex	Male	115	100.0
Total		115	100.0

The baseline demographic profile of the study participants demonstrated that the majority of patients belonged to the middle-aged population. The highest proportion of participants was observed in the 41–50 year's age group (30.4%), closely followed by the 31–40 year's group (29.6%). Participants aged 51–60 years constituted 13.9% of the cohort, while those aged ≤30 years and 61–70 years each accounted for 11.3%. Only 3.5% of participants were older than 70 years. These findings indicate that hepatic encephalopathy associated with chronic liver disease predominantly affected individuals in the economically productive middle-age group.

Sex distribution revealed an exclusively male study population, with all 115 participants being male (100%). The absence of female participants suggests a marked male predominance in the studied cohort, likely reflecting the higher prevalence of alcohol-related chronic liver disease and associated hepatic encephalopathy among men in the study setting.

Table 2: Distribution of Diagnosis among Study Participants

Diagnosis	Frequency	Percentage (%)
HE due to DCLD (HBsAg related)	20	17.4
HE due to UGI bleed with DCLD (Ethanol induced)	90	78.3
HE due to DCLD (HCV related)	5	4.3
Total	115	100

The distribution of diagnosis indicates that the majority of cases were hepatic encephalopathy due to upper gastrointestinal bleed with ethanol-induced decompensated chronic liver disease, accounting for 90 participants (78.3%). HE due to HBsAg-related DCLD was observed in 20 participants (17.4%), while HE due to HCV-related DCLD was reported in 5 participants (4.3%), out of the total 115 participants (100%).

Table 3: Etiology of Chronic Liver Disease

CLD Etiology	Frequency	Percent (%)
Alcohol (Ethanol) induced DCLD	90	78.3
HBsAg related DCLD	20	17.4
HCV related DCLD	5	4.3
Total	115	100.0

The etiology of chronic liver disease shows that alcohol (ethanol) induced DCLD was the most common cause, present in 90 participants (78.3%). HBsAg-related DCLD accounted for 20 cases (17.4%), while HCV-related DCLD was identified in 5 cases (4.3%). The total number of participants included in the analysis was 115 (100%).

Table 4: Distribution of HE Grades

HE Grade	Frequency	Percent (%)
Grade 1	18	15.7
Grade 2	43	37.4

Grade 3	30	26.1
Grade 4	24	20.9
Total	115	100.0

The distribution of hepatic encephalopathy grades indicates that Grade 2 was the most frequent, observed in 43 participants (37.4%). Grade 3 was reported in 30 participants (26.1%), followed by Grade 4 in 24 participants (20.9%). Grade 1 accounted for 18 participants (15.7%), among the total 115 participants (100%) included in the study.

Table 5: Outcome of Study Participants

Outcome	Frequency	Percent (%)
Expired	24	20.9
Improved	91	79.1
Total	115	100.0

The outcome distribution shows that 91 participants (79.1%) improved, while 24 participants (20.9%) expired during the study period. The results represent the final clinical outcome among the total study population of 115 participants (100%).

Table 6: Descriptive Statistics of Serum Electrolytes and Acid–Base Status at Admission and Discharge (n = 115)

	Minimum	Maximum	Mean	Std. Deviation
SERUM Na+ (meq/L) ADMISSION	107	183	135.68	9.758
SERUM K+ (meq/L) ADMISSION	1.7	7.7	4.357	1.1866
SERUM Ca2+ (meq/L) ADMISSION	0.62	1.32	0.9686	0.12686
pH (ACID-BASE STATUS) ADMISSION	7.080	7.640	7.39258	0.092238
SERUM Na+ (meq/L) DISCHARGE	119	175	136.96	7.296
SERUM K+ (meq/L) DISCHARGE	1.7	8.2	4.278	0.9928
SERUM Ca2+ (meq/L) DISCHARGE	0.74	1.34	1.0476	0.12754
pH (ACID-BASE STATUS) DISCHARGE	6.900	7.620	7.39965	0.095004

The descriptive statistics show that at admission, serum sodium (Na^+) ranged from 107 to 183 meq/L with a mean of 135.68 ± 9.758. Serum potassium (K^+) ranged from 1.7 to 7.7 meq/L with a mean of 4.357 ± 1.1866, while serum calcium (Ca^{2+}) ranged from 0.62 to 1.32 meq/L with a mean of 0.9686 ± 0.12686. The pH ranged from 7.080 to 7.640 with a mean of 7.39258 ± 0.092238. At discharge, serum sodium ranged from 119 to 175 meq/L with a mean of 136.96 ± 7.296, potassium from 1.7 to 8.2 meq/L with a mean of 4.278 ± 0.9928, calcium from 0.74 to 1.34 meq/L with a mean of 1.0476 ± 0.12754, and pH ranged from 6.900 to 7.620 with a mean of 7.39965 ± 0.095004.

Table 7: Association between HE Grade and CLD Etiology

HE Grade	Alcohol induced DCLD	HBsAg related DCLD	HCV related DCLD	Total n (%)
Grade 1	13	4	1	18 (15.7)
Grade 2	34	7	2	43 (37.4)
Grade 3	24	5	1	30 (26.1)
Grade 4	19	4	1	24 (20.9)

Total	90	20	5	115 (100)

Chi-square = 0.532, df = 6, p = 0.997

The distribution shows that Grade 2 HE was most frequent with 43 cases (37.4%), followed by Grade 3 with 30 cases (26.1%), Grade 4 with 24 cases (20.9%), and Grade 1 with 18 cases (15.7%). Alcohol-induced DCLD accounted for 90 cases, HBsAg-related DCLD 20 cases, and HCV-related DCLD 5 cases. Statistical analysis showed Chi-square = 0.532, df = 6, p = 0.997.

Table 8: Association between Outcome and CLD Etiology

Outcome	Alcohol induced DCLD	HBsAg related DCLD	HCV related DCLD	Total n (%)
Expired	19	4	1	24 (20.9)
Improved	71	16	4	91 (79.1)
Total	90	20	5	115 (100)

Chi-square = 0.015, df = 2, p = 0.993

Outcome distribution shows that 24 participants (20.9%) expired, including 19 alcohol-induced DCLD, 4 HBsAg-related DCLD, and 1 HCV-related DCLD. Improvement was observed in 91 participants (79.1%), including 71 alcohol-induced, 16 HBsAg-related, and 4 HCV-related cases. Statistical analysis showed Chi-square = 0.015, df = 2, p = 0.993 among 115 participants (100%).

Table 9: Comparison of Serum Electrolytes and Acid–Base Status at Admission and Discharge According to Etiology of Chronic Liver Disease (n = 115)

		Mean	Std. Deviation	P value
SERUM Na+ (meq/L) ADMISSION	Alcohol (Ethanol) induced DCLD	135.71	10.124	**0.997**
	HBsAg related DCLD	135.53	9.401	
	HCV related DCLD	135.80	3.421	
	Total	135.68	9.758	
SERUM K+ (meq/L) ADMISSION	Alcohol (Ethanol) induced DCLD	4.343	1.1439	**0.969**
	HBsAg related DCLD	4.390	1.3742	
	HCV related DCLD	4.460	1.4223	
	Total	4.357	1.1866	
SERUM Ca2+ (meq/L) ADMISSION	Alcohol (Ethanol) induced DCLD	0.9671	0.12799	**0.009**
	HBsAg related DCLD	0.9820	0.13702	
	HCV related DCLD	0.9420	0.05586	
	Total	0.9686	0.12686	
pH (ACID-BASE STATUS) ADMISSION	Alcohol (Ethanol) induced DCLD	7.39724	0.088845	**0.017**
	HBsAg related DCLD	7.37800	0.094122	
	HCV related DCLD	7.36700	0.149650	
	Total	7.39258	0.092238	
SERUM Na+(meq/L) DISCHARGE	Alcohol (Ethanol) induced DCLD	137.17	7.261	**0.007**
	HBsAg related DCLD	135.90	8.220	
	HCV related DCLD	137.40	4.037	
	Total	136.96	7.296	
SERUM K+ (meq/L) DISCHARGE	Alcohol (Ethanol) induced DCLD	4.226	0.9117	

	HBsAg related DCLD	4.580	1.3109	**0.008**
	HCV related DCLD	4.020	0.9121	
	Total	4.278	0.9928	
SERUM Ca2+ (meq/L) DISCHARGE	Alcohol (Ethanol) induced DCLD	1.0452	0.12406	**0.041**
	HBsAg related DCLD	1.0710	0.15266	
	HCV related DCLD	0.9960	0.06504	
	Total	1.0476	0.12754	
pH (ACID-BASE STATUS) DISCHARGE	Alcohol (Ethanol) induced DCLD	7.41111	0.074596	**0.047**
	HBsAg related DCLD	7.35650	0.156450	
	HCV related DCLD	7.36600	0.058992	
	Total	7.39965	0.095004	

At admission, mean serum sodium (Na^+) levels were 135.71 ± 10.124 meq/L in alcohol-induced DCLD, 135.53 ± 9.401 meq/L in HBsAg-related DCLD, and 135.80 ± 3.421 meq/L in HCV-related DCLD (p = 0.997). Mean serum potassium (K^+) values were 4.343 ± 1.1439, 4.390 ± 1.3742, and 4.460 ± 1.4223 meq/L (p = 0.969). Mean serum calcium (Ca^{2+}) values were 0.9671 ± 0.12799, 0.9820 ± 0.13702, and 0.9420 ± 0.05586 meq/L (p = 0.009), while pH values were 7.39724 ± 0.088845, 7.37800 ± 0.094122, and 7.36700 ± 0.149650 respectively (p = 0.017).

At discharge, mean serum sodium values were 137.17 ± 7.261, 135.90 ± 8.220, and 137.40 ± 4.037 meq/L (p = 0.007). Mean serum potassium values were 4.226 ± 0.9117, 4.580 ± 1.3109, and 4.020 ± 0.9121 meq/L (p = 0.008). Mean serum calcium values were 1.0452 ± 0.12406, 1.0710 ± 0.15266, and 0.9960 ± 0.06504 meq/L (p = 0.041). Mean pH values were 7.41111 ± 0.074596, 7.35650 ± 0.156450, and 7.36600 ± 0.058992 respectively (p = 0.047).

DISCUSSION

The present study aimed to evaluate serum electrolyte and acid–base abnormalities in patients with hepatic encephalopathy (HE) associated with chronic liver disease (CLD). It focused on analyzing serum sodium, potassium, calcium, and pH levels at admission and discharge, and correlating these parameters with demographic profile, etiology (alcoholic, HBsAg-related, and HCV-related CLD), HE grade, and clinical outcomes.

The present study showed that hepatic encephalopathy (HE) associated with chronic liver disease (CLD) predominantly affected middle-aged males, particularly those aged 31–50 years. This finding reflects the chronic progression of alcohol-related liver disease, which commonly manifests in the fourth and fifth decades of life. Qazi, Khan, and Umar [10] also reported alcoholic liver disease as the most common etiology of HE. The exclusively male cohort likely reflects the predominance of alcohol-induced CLD in the study population.

Most patients had HE precipitated by upper gastrointestinal (UGI) bleeding in the setting of ethanol-induced decompensated CLD. Gastrointestinal bleeding is a recognized precipitating factor that increases ammonia production and worsens encephalopathy. Similar findings were reported by Qazi, Khan, and Umar [10].

Alcohol-induced decompensated CLD was the predominant etiology, followed by hepatitis B- and hepatitis C-related CLD. These findings are consistent with Qazi, Khan, and Umar [10], who identified alcoholic liver disease as the leading cause of HE. Sahani and Das [11] highlighted the role of inflammation in worsening cirrhosis severity.

Grade 2 HE was the most common presentation, and most patients had moderate-to-severe encephalopathy (Grades 2–4). Kumar et al. [12] reported lower serum sodium levels in advanced HE, while Shah Zeb and Khan [13] and Kowshik et al. [14] associated electrolyte imbalance with severe HE and poor outcomes.

Most patients improved with treatment; however, mortality remained high. Kowshik et al. [14] linked electrolyte abnormalities with poor prognosis and mortality in HE. Kumar et al. [12] and Shah Zeb and Khan [13] also emphasized the prognostic role of serum sodium. These findings highlight the importance of early correction of metabolic abnormalities.

The study demonstrated considerable electrolyte and acid–base disturbances in HE patients, with partial improvement at discharge. Coukos et al. [15] reported frequent

hyponatremia and hypokalemia during HE management. Izhar et al. [16] and Beck et al. [17] also highlighted the significance of hypokalemia and acid–base imbalance in advanced CLD.

No significant association was found between HE grade and CLD etiology, suggesting that HE severity depends more on metabolic disturbances than underlying etiology. Similar findings were reported by Kumar et al. [12], Shah Zeb and Khan [13], and Kowshik et al. [14].

No significant association was observed between outcome and CLD etiology. Kowshik et al. [14] reported that outcomes in HE is more closely related to electrolyte abnormalities than etiology. Kumar et al. [12] and Shah Zeb and Khan [13] also highlighted the prognostic importance of serum sodium.

Significant differences in electrolyte and acid–base parameters were observed across etiologies, particularly at discharge, suggesting variable biochemical recovery patterns. Kumar et al. [12], Shah Zeb and Khan [13], and Kowshik et al. [14] and Coukos et al. [15] similarly emphasized the importance of electrolyte imbalance in HE severity and prognosis.

Strength & Limitations

The present study has several strengths, including a comprehensive evaluation of serum electrolytes and acid–base status in patients with hepatic encephalopathy (HE), enabling integrated assessment of biochemical disturbances in chronic liver disease (CLD). Inclusion of 115 participants allowed analysis of demographic profile, etiology, HE grading, clinical outcomes, and biochemical changes at admission and discharge. Comparison among alcohol-induced, HBsAg-related, and HCV-related decompensated CLD enhanced the clinical relevance of the findings, while assessment at two time points demonstrated dynamic biochemical changes during treatment. The study also explored associations between clinical and laboratory variables, adding analytical value, and utilized routine laboratory investigations, improving applicability in resource-limited settings.

However, the study has certain limitations. Being a single-center hospital-based study, generalizability is limited. The relatively small sample size, particularly for HCV-related cases, restricted subgroup analysis. Inclusion of only male participants prevented sex-based comparisons, and the predominance of alcohol-related CLD may limit applicability to other populations. Additionally, only selected biochemical parameters were evaluated, serial day-to-day trends were not assessed, and outcomes were limited to short-term in-hospital status without long-term follow-up. As an observational study, causal relationships could not be established, and potential confounding factors such as medications, nutritional status, renal dysfunction, and infections were not comprehensively analyzed.

CONCLUSION

In conclusion, hepatic encephalopathy in chronic liver disease predominantly affected middle-aged males and was chiefly associated with alcohol-induced decompensated chronic liver disease. Upper gastrointestinal bleeding emerged as the most important precipitating factor, particularly in alcohol-related cases. Most patients presented with moderate to severe hepatic encephalopathy, reflecting advanced metabolic and neurological impairment at admission. Significant electrolyte and acid–base disturbances were observed, with partial biochemical improvement during hospitalization, underscoring the importance of continuous monitoring and correction of metabolic abnormalities. Although etiology was not significantly associated with age, HE grade, or short-term outcome, it demonstrated a significant relationship with precipitating diagnosis and biochemical recovery patterns. Despite a considerable mortality rate, the majority of patients improved with timely management, indicating that hepatic encephalopathy is a potentially reversible condition when promptly recognized and treated. The study highlights the critical role of early identification of precipitating factors, routine biochemical assessment, and aggressive supportive management in improving clinical outcomes and reducing mortality in hepatic encephalopathy.

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