RESEARCH PAPER

Frequency of Hyponatremia in Liver Cirrhosis Patients Presenting with Hepatic Encephalopathy

Tooba Irfan[1], Abdul Rabb[2], Sidra Noor[3], Ariba Afzal[4]

[1,3,4] *MBBS, Postgraduation Trainee Internal Medicine Department, Memon Medical Institute Hospital, Karachi*
[2] *MBBS, FCPS (Medicine), Consultant & Supervisor Internal Medicine Department, Memon Medical Institute Hospital, Karachi*
Correspondence to Dr. Tooba Irfan, Email: toobairfan.94@gmail.com

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ABSTRACT

Background: Hepatic encephalopathy (HE) is one of its most serious complications and is associated with poor neurological outcomes.

Objective: To determine the frequency of hyponatremia in liver cirrhosis patients presenting with hepatic encephalopathy at a tertiary care hospital.

Study Design & Setting: This was a cross-sectional study conducted in the Department of Internal Medicine, Memon Medical Institute Hospital, Karachi from 1st December 2025 to 1st May 2026.

Methodology: A total of 192 patients aged 20–70 years with hepatic encephalopathy secondary to liver cirrhosis were enrolled through non-probability consecutive sampling. Patients meeting the inclusion criteria were included after obtaining written informed consent. Five milliliters of venous blood were collected within 30 minutes of admission for serum sodium estimation. Hyponatremia was defined as a serum sodium level of <135 mmol/L. Demographic characteristics, Child-Pugh classification, duration of chronic liver disease, duration of hospital stay, and serum sodium levels were recorded. Data were analyzed using SPSS version 26.0. Continuous variables were summarized using appropriate descriptive statistics after assessment of normality, while categorical variables were presented as frequencies and percentages. Stratification was performed for age, gender, and duration of liver disease.

Results: A total of 192 patients with liver cirrhosis presenting with hepatic encephalopathy were included in the study. The mean age was 54.18 ± 10.76 years, and 118 (61.5%) patients were male. The mean serum sodium level was 132.94 ± 5.89 mmol/L. Hyponatremia was observed in 103 (53.6%) patients. The majority of patients belonged to Child–Pugh Class C (52.1%), while Stage II hepatic encephalopathy was the most common presentation (37.5%). Stratification analysis demonstrated a statistically significant association of hyponatremia with increasing age ($p = 0.011$) and longer duration of liver cirrhosis ($p = 0.004$), whereas no significant association was observed with gender ($p = 0.276$).

Conclusion: Hyponatremia was a common electrolyte abnormality among patients with liver cirrhosis presenting with hepatic encephalopathy, affecting more than half of the study population. Its frequency was significantly associated with increasing age and longer duration of liver cirrhosis, while no significant association was observed with gender. Early recognition and prompt management of hyponatremia may help improve the clinical outcomes of these patients.

Keywords: Child-Pugh classification; hepatic encephalopathy; hyponatremia; liver cirrhosis; serum sodium.

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INTRODUCTION

Liver cirrhosis is a progressive chronic liver disease characterized by hepatic fibrosis, regenerative nodules, and irreversible deterioration of liver function. It represents the end stage of various chronic liver diseases, including chronic viral hepatitis, alcohol-related liver disease, and non-alcoholic fatty liver disease (NAFLD).[1,2] Progressive loss of liver function impairs essential physiological processes such as detoxification, protein synthesis, and bile production.[3] Hepatic encephalopathy (HE) is one of the most serious complications of liver cirrhosis. It is a potentially reversible neuropsychiatric syndrome that ranges from subtle cognitive impairment to deep coma. The condition results from impaired hepatic detoxification, leading to the accumulation of neurotoxins, particularly ammonia, which cross the blood-brain barrier and adversely affect cerebral function.[4,5] Hepatic encephalopathy develops in approximately 30–45% of patients with chronic liver disease. In Pakistan, the prevalence of hepatic encephalopathy among patients with liver cirrhosis has been reported to be approximately 18%.[5]

**Author for Correspondence: toobairfan.94@gmail.com*

Among the complications associated with liver cirrhosis and hepatic encephalopathy, hyponatremia has received considerable attention because of its association with increased morbidity and mortality. Hyponatremia is defined as a serum sodium concentration of less than 135 mmol/L.[6] It is commonly observed in patients with advanced liver cirrhosis, particularly in those presenting with hepatic encephalopathy. The relationship between hyponatremia and hepatic encephalopathy is multifactorial and involves hyperammonemia, inflammatory cytokines, oxidative stress, cerebral edema, and disturbances in brain osmolarity, all of which contribute to worsening neurological dysfunction.[7]

Several studies have reported a high frequency of hyponatremia among patients with liver cirrhosis presenting with hepatic encephalopathy. Khan et al. (2022) reported hyponatremia in 69 (40.1%) patients.[8] Bashir et al. (2023) observed hyponatremia in 62 (53.91%) patients with hepatic encephalopathy.[9] Shah et al. (2024) reported hyponatremia in 65% of 130 enrolled patients.[10] Afridi et al. (2017) found hyponatremia in 48 (36.9%) patients.[11] Similarly, Iqbal et al. (2019) reported hyponatremia in 35 (45.45%) patients.[12] Qureshi et al. observed hyponatremia in 82.6% of patients with hepatic encephalopathy.[13] Another study by Shaikh et al. reported hyponatremia (serum sodium <130 mEq/L) in 26.7% of patients with liver cirrhosis and hepatic encephalopathy.[14]

Hyponatremia is a common complication of liver cirrhosis with hepatic encephalopathy and is associated with poor clinical outcomes. Previous studies have reported variable frequencies of hyponatremia, ranging from 26.7% to 82.6%, likely due to differences in study populations and disease severity. Limited institution-specific data are available from our setting; therefore, this study will determine the frequency of hyponatremia in liver cirrhosis patients presenting with hepatic encephalopathy at our tertiary care hospital. The findings will provide local evidence that may help in the early identification and appropriate management of these patients. The objective is to determine the frequency of hyponatremia in liver cirrhosis patients presenting with hepatic encephalopathy at a tertiary care hospital was determined.

METHODS

After approval from the Institutional Review Board (IRB) & Synopsis approval from CPSP This was a cross-sectional study conducted in the Department of Internal Medicine, Memon Medical Institute Hospital, Karachi from 1st December 2025 to 1st May 2026. A total of 192 patients with hepatic encephalopathy secondary to liver cirrhosis who fulfilled the inclusion criteria were enrolled consecutively from the Department of Internal Medicine, Memon Medical Institute Hospital, Karachi. Written informed consent was obtained from each patient or their legally authorized attendant before enrollment. Patients of either gender aged 20–70 years presenting with hepatic encephalopathy secondary to liver cirrhosis, as defined in the operational definitions, were included in the study. Only those patients or their legally authorized attendants who provided written informed consent were enrolled. Patients with diabetes mellitus (fasting blood glucose >136 mg/dL), diabetic nephropathy, or those receiving diuretic therapy were excluded. Patients with hypertension (blood pressure >140/90 mmHg), postoperative renal dysfunction (serum creatinine >2.5 mg/dL or blood urea nitrogen >100 mg/dL), intracranial vascular events (including cerebrovascular accidents, vascular malformations, aneurysms, and venous sinus thrombosis), central nervous system infections (meningitis, encephalitis, cerebral abscess), spontaneous bacterial peritonitis, or heart failure with an ejection fraction <25% were also excluded.

A detailed clinical history was recorded, and 5 mL of venous blood was collected through venepuncture within 30 minutes of admission for estimation of serum sodium levels in the hospital laboratory. The presence of hyponatremia was recorded according to the operational definition. The Child-Pugh classification, demographic characteristics, and stage of hepatic encephalopathy were documented on a structured proforma by the principal investigator. To minimize observer bias, all patients were assessed clinically by the principal investigator under the supervision of the consultant, while potential confounding variables were controlled through the exclusion criteria.

Liver cirrhosis was diagnosed in the presence of aspartate aminotransferase (AST) levels greater than two times the upper limit of normal, platelet count less than 160,000/mm³, prothrombin time (PT) activity less than 100%, and ultrasonographic evidence of liver fibrosis. Hepatic encephalopathy was diagnosed clinically according to the West Haven Criteria, and patients with Stage I to Stage IV hepatic encephalopathy were included in the study. Hyponatremia was assessed by collecting 5 mL of venous blood through venepuncture within 30 minutes of admission. The blood sample was sent to the hospital laboratory for serum sodium estimation. Hyponatremia was defined as a serum sodium level of less than 135 mmol/L.

The collected data were entered and analyzed using SPSS version 26.0. The normality of continuous variables was assessed using the Shapiro–Wilk test. Normally distributed numerical variables, including age and serum sodium level, were presented as mean ± standard deviation, whereas non-normally distributed variables were presented as median and interquartile range. Categorical variables, including gender, Child-Pugh class, stage of hepatic encephalopathy, and hyponatremia (yes/no), were presented as frequencies and percentages. Data were stratified for age, gender, and duration of liver cirrhosis to assess the effect of these variables. Post-stratification, the Chi-square test was applied, and a p-value of ≤0.05 was considered statistically significant.

RESULTS

The study included a total of 192 patients with liver cirrhosis presenting with hepatic encephalopathy. The mean age of the patients was 54.18 ± 10.76 years. Most patients belonged to the 41–55 years age group (42.7%), followed by 56–70 years (38.5%) and 20–40 years

(18.8%). There were 118 (61.5%) males and 74 (38.5%) females. The mean duration of liver cirrhosis was 4.63 ± 2.08 years, with 126 (65.6%) patients having a disease duration of ≤5 years and 66 (34.4%) having a duration of >5 years. The mean serum sodium level was 132.94 ± 5.89 mmol/L, as shown in Table 1.

Table 1. Baseline Characteristics of Patients (n = 192)

Variables	Type	n (%) / Mean ± SD
Age (years)	Mean ± SD	54.18 ± 10.76
	20–40 years	36 (18.8)
	41–55 years	82 (42.7)
	56–70 years	74 (38.5)
Gender	Male	118 (61.5)
	Female	74 (38.5)
Duration of liver cirrhosis	Mean ± SD	4.63 ± 2.08 years
	≤5 years	126 (65.6)
	>5 years	66 (34.4)
Serum sodium (mmol/L)	Mean ± SD	132.94 ± 5.89

Most patients were classified as Child–Pugh Class C, accounting for 100 (52.1%) patients, followed by Child–Pugh Class B in 74 (38.5%) patients and Child–Pugh Class A in 18 (9.4%) patients, as shown in Table 2.

Table 2. Child–Pugh Classification of Patients (n = 192)

Child–Pugh Class	n (%)
A	18 (9.4)
B	74 (38.5)
C	100 (52.1)

Regarding the severity of hepatic encephalopathy according to the West Haven Criteria, Stage II was the most frequent presentation, observed in 72 (37.5%) patients, followed by Stage III in 61 (31.8%) patients, Stage IV in 31 (16.1%) patients, and Stage I in 28 (14.6%) patients, as shown in Table 3.

Table 3. Stage of Hepatic Encephalopathy According to West Haven Criteria (n = 192)

Stage	n (%)
Stage I	28 (14.6)
Stage II	72 (37.5)
Stage III	61 (31.8)
Stage IV	31 (16.1)

Hyponatremia was observed in 103 (53.6%) patients, whereas 89 (46.4%) patients had normal serum sodium levels, as shown in Table 4.

Table 4. Frequency of Hyponatremia Among Patients with Liver Cirrhosis Presenting with Hepatic Encephalopathy (n = 192)

Hyponatremia	n (%)
Yes	103 (53.6)
No	89 (46.4)

Stratification analysis demonstrated a statistically significant association between age and hyponatremia (p = 0.011), with the highest frequency of hyponatremia observed among patients aged 56–70 years (64.9%). Although hyponatremia was more common in males (56.8%) than females (48.6%), this difference was not statistically significant (p = 0.276). A statistically significant association was also found between the duration of liver cirrhosis and hyponatremia (p = 0.004), with patients having a disease duration of more than five years showing a higher frequency of hyponatremia (68.2%) compared with those having a duration of five years or less (46.0%), as shown in Table 5.

Table 5. Stratification of Hyponatremia with Respect to Age, Gender, and Duration of Liver Cirrhosis (n = 192)

Variable	Category	Hyponatremia Yes	Hyponatremia No	Total	p
Age (years)	20–40	13 (36.1)	23 (63.9)	36 (18.8)	0.011
	41–55	42 (51.2)	40 (48.8)	82 (42.7)	
	56–70	48 (64.9)	26 (35.1)	74 (38.5)	
Gender	Male	67 (56.8)	51 (43.2)	118 (61.5)	0.276
	Female	36 (48.6)	38 (51.4)	74 (38.5)	
Duration of Liver Cirrhosis	≤5 years	58 (46.0)	68 (54.0)	126 (65.6)	0.004
	>5 years	45 (68.2)	21 (31.8)	66 (34.4)	

DISCUSSION

Liver cirrhosis is a chronic progressive disease characterized by irreversible hepatic fibrosis and impaired liver function. Hepatic encephalopathy is a serious neurological complication of cirrhosis that results from the accumulation of neurotoxins, particularly ammonia. Hyponatremia is a common electrolyte disturbance in patients with advanced liver cirrhosis and has been associated with worsening cerebral edema and neurological dysfunction. The coexistence of

hyponatremia and hepatic encephalopathy contributes to increased morbidity, prolonged hospitalization, and poor clinical outcomes. Early identification of hyponatremia may facilitate timely management and improve patient care. Therefore, this study was conducted to determine the frequency of hyponatremia among patients with liver cirrhosis presenting with hepatic encephalopathy.

Liver cirrhosis is frequently complicated by electrolyte disturbances, particularly hyponatremia, which contributes to the development and progression of hepatic encephalopathy. The present study was conducted to determine the frequency of hyponatremia among patients with liver cirrhosis presenting with hepatic encephalopathy. A total of 192 patients were included, with a mean age of 54.18 ± 10.76 years, and males constituted 61.5% of the study population. Most patients belonged to the 41–55 years age group (42.7%), followed by 56–70 years (38.5%). The majority of patients were classified as Child–Pugh Class C (52.1%), reflecting advanced liver disease, while Stage II hepatic encephalopathy (37.5%) was the most common clinical presentation.

The demographic characteristics observed in our study are comparable with previous reports. Ali et al. reported a mean age of 59.84 ± 7.93 years with 45.8% males,[21] whereas Shehriyar et al. documented a mean age of 53.67 ± 13.83 years and 65.3% male patients.[23] Malik et al. also reported a predominantly male population (58.1%) with a mean age of 48.82 ± 14.94 years.[24] These similarities suggest that liver cirrhosis complicated by hepatic encephalopathy predominantly affects middle-aged and older adults and is more common among males.

The primary outcome of the present study demonstrated that 103 (53.6%) patients had hyponatremia. As shown in Table 6, the reported frequency of hyponatremia in previous studies has varied considerably, ranging from 26.7% reported by Shaikh et al.[14] to 82.5% reported by Ali et al.[21] Our findings closely resemble those of Bashir et al., who reported hyponatremia in 53.9% of patients,[9] and Hassan et al., who observed a prevalence of 58.8%.[24] However, our frequency was higher than those reported by Afridi et al. (36.9%),[11] Younas et al. (36.9%),[20] Khan et al. (40.1%),[8] Shehriyar et al. (41.5%),[23] Ehsan et al. (41.4%),[17] and Bajkani et al. (42.0%),[21] while remaining lower than the frequencies reported by Shah et al. (65.0%)[10] and Ali et al. (82.5%).[21] The observed variation across studies is likely attributable to differences in study populations, sample size, severity of liver disease, inclusion criteria, healthcare settings, and diagnostic thresholds used to define hyponatremia. Despite these differences, the collective evidence consistently indicates that hyponatremia is a common electrolyte abnormality among patients with liver cirrhosis, particularly those presenting with hepatic encephalopathy.

Table 6. Comparison of frequency of hyponatremia with previous studies

Study	Year	Sample size (n)	Frequency of hyponatremia
Shaikh et al.[14]	2020	217	26.7%
Afridi et al.[11]	2017	130	36.9%
Iqbal et al.[12]	2019	77	45.45%
Younas et al.[20]	2021	260	36.9%
Khan et al.[8]	2022	172	40.1%
Bashir et al.[9]	2023	115	53.9%
Hassan et al.[24]	2024	338	58.8%
Shah et al.[10]	2024	130	65.0%
Shehriyar et al.[23]	2025	118	41.5%
Ali et al.[21]	2025	120	82.5%
Ehsan et al.[17]	2026	140	41.4%
Bajkani et al.[21]	2026	224	42.0%
Present study	**2026**	**192**	**53.6%**

The mean serum sodium level in our study was 132.94 ± 5.89 mmol/L, which is slightly higher than the values reported by Younas et al. (129.11 ± 6.53 mEq/L)[20] and Shehriyar et al. (130.14 ± 6.46 mEq/L).[23] Rekha et al. further reported a mean serum sodium level of 127 mEq/L among patients with hepatic encephalopathy and observed elevated serum ammonia levels in these patients, suggesting that declining serum sodium is accompanied by worsening metabolic disturbances. Although serum ammonia was not measured in the present study, our findings support the concept that hyponatremia is a frequent biochemical abnormality among cirrhotic patients with hepatic encephalopathy.

Most patients in our study belonged to Child–Pugh Class C (52.1%), indicating advanced liver disease. Bajkani et al.[21] similarly demonstrated that hyponatremia was significantly associated with markers of advanced liver disease, including lower hemoglobin and serum albumin levels, the presence of ascites, elevated international normalized ratio, total bilirubin, Child–Turcotte–Pugh score, and Model for End-stage Liver Disease score ($p<0.05$ for all). Likewise, Malik et al. found a highly significant association between the severity of hyponatremia and the severity of ascites ($p<0.001$), with 93.4% of patients with Grade III ascites exhibiting severe hyponatremia compared with only 1.1% among those with Grade I ascites.[24] These findings support the concept that hyponatremia becomes increasingly prevalent as liver disease progresses and decompensation worsens.

With regard to hepatic encephalopathy, Stage II (37.5%) was the most common presentation in our study, followed by Stage III (31.8%), Stage IV (16.1%), and Stage I (14.6%). Younas et al. similarly reported that Grade II hepatic encephalopathy (23.8%) was the most frequent presentation, followed by Grade I (20.8%), Grade III (12.3%), and Grade IV (10.8%).[20] Rekha et al. observed that 61% of patients had hyponatremia and that patients with hepatic encephalopathy had a mean serum sodium level of 127 mEq/L, while elevated serum ammonia levels were also present. Ehsan et al. reported hepatic encephalopathy in 64.3% of patients and demonstrated that hepatic encephalopathy occurred significantly more frequently among patients with hyponatremia than among those with normal serum sodium levels (82.8% vs. 51.2%; $p=0.001$).[17] Collectively, these studies support the

important contribution of hyponatremia to the development and progression of hepatic encephalopathy.

Our stratification analysis demonstrated a statistically significant association between increasing age and hyponatremia (p=0.011), with the highest frequency observed among patients aged 56–70 years (64.9%). A significant association was also found between longer duration of liver cirrhosis and hyponatremia (p=0.004), as patients with disease duration greater than five years had a markedly higher frequency of hyponatremia (68.2%) than those with disease duration of five years or less (46.0%). In contrast, although hyponatremia was numerically more common among males (56.8%) than females (48.6%), this difference was not statistically significant (p=0.276). Similarly, Shehriyar et al. found no significant association with gender but reported significant associations between hyponatremia and body mass index greater than 25 kg/m² (p=0.013) as well as rural residence (p=0.015). Ali et al. demonstrated a significant inverse correlation between serum sodium levels and the severity of hepatic encephalopathy (Pearson's r = −0.285; p=0.002) and also reported a highly significant association between hyponatremia and hepatic encephalopathy severity (p<0.001).[21] These findings, together with the results of the present study, suggest that hyponatremia is associated with disease progression and increasing clinical severity in patients with liver cirrhosis.

Overall, the findings of the present study are consistent with the available literature demonstrating that hyponatremia is a frequent complication of liver cirrhosis and hepatic encephalopathy. The observed association of hyponatremia with older age and longer duration of liver cirrhosis highlights the importance of regular monitoring of serum sodium levels in cirrhotic patients, particularly those with advanced disease, to facilitate early identification and timely management of this potentially preventable complication.

STUDY LIMITATIONS

This study was conducted at a single tertiary care hospital, which may limit the generalizability of the findings to the wider population. The cross-sectional study design did not permit assessment of temporal or causal relationships between hyponatremia and hepatic encephalopathy. In addition, serum sodium levels were measured only at the time of admission, and changes during hospitalization were not evaluated.

CONCLUSION

Hyponatremia was a frequent finding among patients with liver cirrhosis presenting with hepatic encephalopathy. The frequency of hyponatremia was significantly associated with increasing age and longer duration of liver cirrhosis. Early detection and appropriate management of hyponatremia may help improve the clinical outcomes of patients with liver cirrhosis and hepatic encephalopathy.

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