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# Frequency of Hyponatremia in Patients with Liver Cirrhosis and Its Association with Hepatic Encephalopathy

Noor Ehsan, Mahnoor Iqbal, Hateem Ahmed, Wafa Qaisar, Imran Farooka, Mahmood Nasir Malik

**Abstract:**

**Objective:** This study aims to determine the frequency of hyponatremia and to investigate its association with hepatic encephalopathy among patients with liver cirrhosis.

**Study design & Settings:** Descriptive cross-sectional study conducted at the Department of Medicine, Gulab Devi Teaching Hospital, Lahore.

**Methodology:** A non-probability consecutive sampling technique was used to enroll 140 patients with liver cirrhosis. Patients with conditions that could affect electrolyte balance or mental status were excluded. Hyponatremia was defined as serum sodium <135 mEq/L and was further categorized into mild, moderate, and severe according to admission serum sodium levels. Hepatic encephalopathy was assessed clinically using the West Haven criteria.

**Results:** Out of 140 patients, 58 (41.4%) had hyponatremia, including 28 (20.0%) mild, 20 (14.3%) moderate, and 10 (7.1%) severe cases. Hepatic encephalopathy was present in 90 patients (64.3%). Hepatic encephalopathy was more frequent among patients with hyponatremia than among those with normal sodium levels (82.8% vs. 51.2%; p=0.001).

**Conclusion:** Hyponatremia is common in patients with liver cirrhosis and has a significant association with hepatic encephalopathy. Serum sodium monitoring may help identify patients who are at increased risk of neurological complications.

**Keywords:** Liver cirrhosis; hyponatremia; hepatic encephalopathy

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## INTRODUCTION:

Hyponatremia is a common electrolyte disorder among patients with liver cirrhosis, especially in advanced stages. Such factors as ineffective renal functioning, excessive water retention, and activity of such systems as renin-angiotensin-aldosterone system (RAAS) and vasopressin are involved in the mechanism. Cirrhosis also causes splanchnic vasodilation thereby decreasing the volume of blood and triggering the processes that increase sodium retention in kidneys and water absorption. Cirrhosis-induced systemic inflammation and portal hypertension are also known to cause hyponatremia, especially in patients with ascites.[1,2] Recent evidence associates the severity of hyponatremia with the non-functioning of the liver, especially as portal hypertension or hepatorenal syndrome in patients with cirrhosis.[3]

In addition to being a biochemical abnormality, hyponatremia is a clinically important abnormality with major implications in cirrhosis. It is closely linked to such complications like hepatic encephalopathy (HE), ascites, renal impairment, and high mortality. Hepatic encephalopathy is also a prominent complication to the quality of life and prognosis of cirrhotic patients, as it is a neuropsychiatric syndrome due to liver dysfunction and defective ammonia metabolism. New data shows that hyponatremia is an important factor that contributes to the development and course of HE. The decreased level of sodium destabilizes osmotic balance in brain cells especially the astrocytes, resulting in cellular edema and predisposition to ammonia-induced neurotoxicity. This synergistic effect further increases cerebral edema and adds to the deterioration of the neurological functioning.[4]

Clinical trials have indicated that there is a close relationship between hyponatremia and the development and extent of hepatic encephalopathy. Lower serum sodium levels in the patients increase their chances of getting HE and also they are likely to have higher grades of the condition. In addition, hyponatremia has been part of predictors of poor outcomes in cirrhosis including high rates of hospitalization, long hospitalization, and high mortality rates. This evidence supports the value of regular monitoring of serum sodium levels in cirrhotic patients to support comprehensive disease management.[3]

Hyponatremia in liver cirrhosis is also related closely to hepatic encephalopathy (HE). Low sodium concentrations may exacerbate HE symptoms by disturbing osmoregulation in brain cells. It has been shown that in cirrhotic patients, low sodium is an important predictor of adverse outcome that may predispose them to developing hepatic encephalopathy which is associated with a poor prognosis.[4,5] Clinical evidence has suggested that hyponatremia is predictive of hepatic encephalopathy development and progression.[6]

Other than serving as a pointer to the extent of disease severity, hyponatremia in cirrhosis also influences the treatment of hepatic encephalopathy. Under extreme cases, hypertonic saline or diuretics and fluid restriction may be necessary in order to balance the salt levels. Rapid correction should be avoided because it may lead to osmotic demyelination, including central pontine myelinolysis. Recent researches have reiterated the necessity of adopting a combined approach to treating both conditions among cirrhotic patients because of their co-occurrence and complicating nature of treatment. Recent treatment interventions such as vasopressin receptor antagonists can be useful in balancing water and sodium in cirrhotic patients, and they have the potential to benefit them.[7,8]

Hyponatremia was seen in 96 (36.9%) of 261 patients with cirrhosis. HE was present in 67.7% patients. In 20.8, 23.8, 12.3 and 10.8 percent, HE grade I, grade II, grade III and grade IV were present respectively.[9] In another study, frequency of hyponatremia in patients with hepatic encephalopathy was reported to be 53.9% in 96 patients with hyponatremia. 84 patients had hepatic encephalopathy.[10]

The purpose of this study was to measure the prevalence of hyponatremia in patients with liver cirrhosis and its association with hepatic encephalopathy, in our population. The knowledge of this association might be used to define high-risk patients to be better monitored and treated at an earlier stage, which may enhance prognostic measures. The study could address a significant gap of knowledge, as it will examine the interaction between hyponatremia and hepatic encephalopathy in cirrhosis development that will guide future clinical practice and outcomes measurement.

## METHODOLOGY:

This study was a descriptive cross-sectional study that took place at the Department of Medicine, Gulab Devi Teaching Hospital, Lahore, from 12 September 2025 to 11 March 2026 after institutional ethical approval. As this was a descriptive cross-sectional observational study, randomization and a control group were not applicable. The study had 140 patients with liver cirrhosis where a non-probability consecutive sampling technique was used. The sample size was determined using a 95% confidence level, an 8% margin of error and the anticipated occurrence rate of hyponatremia was 36.9% in cirrhotic patients.[10]

Patients of either sex, with a diagnosis of liver cirrhosis aged more than 12 years, were eligible to be included in the study. Clinical, biochemical, and radiological findings were used to diagnose liver cirrhosis, including a coarse echotexture and nodular hepatic surface on ultrasound, platelet less than 150,000/mm 3, AST/ALT ratio more than 1, splenomegaly (>11 cm), decreased albumin (<3.5 g/dL), and the presence of ascites. The exclusion criteria included a history of epilepsy or the use of antiepileptic medications, acute cardiac disease, renal failure, pregnancy, acute drug-induced confusion, neurological disorders (such as stroke), hypoglycemia (blood sugar levels below 70 mg/dl), hyperglycemia (blood sugar levels above 200 mg/dl), hypokalemia (serum potassium below 3.5 mEq/L), and severe hepatitis.

After approval from the hospital ethical review board, eligible patients presenting to the emergency department and outpatient department were enrolled according to the inclusion criteria. Prior to data collection, all subjects provided written informed consent. A standardized proforma was used to collect data on gender, age, place of residence, and socioeconomic status. The presence and severity of hepatic encephalopathy were determined through clinical evaluation using the West Haven criteria. These criteria classify encephalopathy from grade I to IV based on neurological observation and mental status.

During admission, blood samples were drawn and serum sodium levels were measured. Hyponatremia was considered to be a serum sodium level that is less than 135 mEq/L and it was further classified into mild (130 to 134 mEq/L) moderate (120 to 129 mEq/L) and severe (<120 mEq/L). During the first 24 hours of hospitalization, clinical evaluation and reported results provided information on HE. All the patients were treated as per the normal hospital treatment protocols and the possible confounding factors were kept to minimal by observing the rigid observance of the exclusion criteria.

The collected data were evaluated and analyzed using SPSS version 25, the Statistical Package for the Social Sciences. Qualitative variables encompassed the presence of hyponatremia, gender, and grades of hepatic encephalopathy; quantitative variables included age and serum sodium levels, presented as mean and standard deviation. The criteria used to stratify the data included age, sex, residence, socioeconomic position, presence of ascites, serum sodium level, and grades of hepatic encephalopathy. After classification, we evaluated the relationship between hyponatremia and hepatic encephalopathy using the Chi-square test. A p-value of less than 0.05 was considered statistically significant.

**RESULTS:**

In Table I, the summary of baseline demographic and clinical characteristics of the study population is summarized to include 140 patients with liver cirrhosis. The age of the participants (mean) was 52.4 +/-11.8 years; this shows that the majority of patients were middle-aged to elderly. There was an apparent male predominance with an 88 (62.9) and 52 (37.1%), resulting in a larger prevalence of cirrhosis in males among the cohort. As far as residence is concerned, there was a slightly higher percentage of patients who lived in rural regions (55.7% versus urban regions 44.3%) which likely could be attributed to the access or disease prevalence in that location. The socioeconomic status of the patients revealed that over half of patients (54.3) were of low-income, whereas 31.4% were of middle-income, and 14.3% were of high-income group, which may indicate that liver cirrhosis is more common among the economically disadvantaged groups. Hyponatremia was clinically found in 58 patients (41.4) with 82 patients (58.6) showing normal levels of sodium. Hepatic encephalopathy (HE) occurred in 90 patients (64.3%), and 50 patients (35.7%), did not show evidence of encephalopathy. Generally, the table indicates that hyponatremia along with hepatic encephalopathy present significant burden in the study population.

Table II shows the distribution of the severity of hyponatremia among the affected patients. Among the 140 patients, 58 (41.4) were found to be hyponatremic. Of these mild hyponatremia was the most prevalent and was seen in 28 patients (20.0% of the overall sample), then moderate hyponatremia with only 20 patients (14.3%), and severe hyponatremia was observed in only 10 patients (7.1%). This distribution reflects that even though hyponatremia is a relatively common disease among cirrhotic patients, most cases lie in the range of mild to moderate diseases. Nonetheless, the occurrence of extreme hyponatremia in a group of patients is clinically important, as it can be typically linked to end-stage liver disease, and an increased amount of complications, including hepatic encephalopathy. The results highlight the significance of early diagnosis and hyponatremia grading in order to conduct suitable medical care.

Table III shows the severity of hepatic encephalopathy (HE) in the participants studied. Ninety out of 140 patients (64.3) were identified to have hepatic encephalopathy. Among these, Grade II HE was the most frequently observed (21.4%), followed by Grade I (18.6%), Grade III (14.3%), and Grade IV (10.0%). This distribution implies that a high percentage of patients came with moderate and severe forms of encephalopathy (Grades II to IV) which suggests the advanced disease status in the study population. The elevation of grade of HE also implies greater involvement of the brain and worse prognosis of patients with cirrhosis.

Table IV illustrates that there was a statistically significant relationship between hepatic encephalopathy and hyponatremia ($p = 0.001$). Of patients with hyponatremia, 48 of 58 (82.8%) developed hepatic encephalopathy with only 42 of 82 (51.2%) of patients having no hyponatremia having hepatic encephalopathy. In contrast, the percentage of patients without HE were significantly lower in the hyponatremia group (17.2%), than in the normal sodium group (48.8%). The results of the study underscore the significant association between hyponatremia and the risk of developing hepatic encephalopathy and back the claim that hyponatremia is a strong predictor of occurrence of neurological complications in liver cirrhosis.

Table V shows stratified analysis of the association between hyponatremia and hepatic encephalopathy by various demographic and clinical factors. It was found to have a statistically significant association in all the subgroups such as age ($p = 0.02$), gender ($p = 0.04$), residence ($p = 0.03$), socioeconomic status ($p = 0.05$), and presence of ascites ($p = 0.01$). The rate of combined hyponatremia and HE was more prevalent in patients above 50 years (48.3) than in younger patients. Likewise, the percentage of cases was higher among males (51.7) compared to females. The rural population and low socioeconomic status were also more frequently affected. Interestingly, the strongest correlation was noted in the presence of ascites where 69.0% patients had both hyponatremia and hepatic encephalopathy which implies that the presence of fluids and progressive disease contributes hugely to the risk of neurological problems. On the whole, this stratified review supports the fact that hyponatremia has always been linked to hepatic encephalopathy in various aspects of the patient population that qualify as clinical indicators supporting the role of hyponatremia as a clinical indicator in cirrhotic patients.

**DISCUSSION:**

The present analysis shows that 64.3% of the patients with liver cirrhosis presented with hepatic encephalopathy (HE) and 41.4% had hyponatremia. Both hepatic encephalopathy and hyponatremia were statistically significantly correlated, and 82.8 percent of the hepatic encephalopathy cases were found to be in hyponatremic individuals and 51.2 percent in non-hyponatremic individuals.

Table 1: Demographic Characteristics (n = 140)

| Variable | Category | Frequency (n) | Percentage (%) |
|---|---|---|---|
| **Age (years)** | Mean ± SD | 52.4 ± 11.8 | — |
| **Gender** | Male | 88 | 62.9% |
| | Female | 52 | 37.1% |
| **Residence** | Rural | 78 | 55.7% |
| | Urban | 62 | 44.3% |
| **Socioeconomic Status** | Low | 76 | 54.3% |
| | Middle | 44 | 31.4% |
| | High | 20 | 14.3% |
| **Hyponatremia** | Yes | 58 | 41.4% |
| | No | 82 | 58.6% |
| **HE Present** | Yes | 90 | 64.3% |
| | No | 50 | 35.7% |

Table 2: Severity of Hyponatremia

| Severity | Frequency (n) | Percentage (%) |
|---|---|---|
| Mild | 28 | 20.0% |
| Moderate | 20 | 14.3% |
| Severe | 10 | 7.1% |
| **Total** | 58 | 41.4% |

Table 3: Grades of Hepatic Encephalopathy

| HE Grade | Frequency (n) | Percentage (%) |
|---|---|---|
| Grade I | 26 | 18.6% |
| Grade II | 30 | 21.4% |
| Grade III | 20 | 14.3% |
| Grade IV | 14 | 10.0% |
| **Total** | 90 | 64.3% |

Table 4: Stratification of Hyponatremia with Hepatic Encephalopathy

| Hyponatremia | HE Present n (%) | HE Absent n (%) | Total | p-value |
|---|---|---|---|---|
| Yes | 48 (82.8%) | 10 (17.2%) | 58 | **0.001** |
| No | 42 (51.2%) | 40 (48.8%) | 82 | |
| **Total** | 90 (64.3%) | 50 (35.7%) | 140 | |

Table 5: Stratification of Hyponatremia with Hepatic Encephalopathy Across Variables

| Variable | Category | Hyponatremia + HE n (%) | No Hyponatremia + HE n (%) | Total | p-value* |
|---|---|---|---|---|---|
| **Age** | <=50 years | 20 (34.5%) | 18 (22.0%) | 38 | **0.02** |
| | >50 years | 28 (48.3%) | 24 (29.3%) | 52 | |
| **Gender** | Male | 30 (51.7%) | 26 (31.7%) | 56 | **0.04** |
| | Female | 18 (31.0%) | 16 (19.5%) | 34 | |
| **Residence** | Rural | 28 (48.3%) | 24 (29.3%) | 52 | **0.03** |
| | Urban | 20 (34.5%) | 18 (22.0%) | 38 | |
| **Socioeconomic Status** | Low | 30 (51.7%) | 26 (31.7%) | 56 | **0.05** |
| | Middle | 12 (20.7%) | 10 (12.2%) | 22 | |
| | High | 6 (10.3%) | 6 (7.3%) | 12 | |
| **Ascites** | Present | 40 (69.0%) | 30 (36.6%) | 70 | **0.01** |
| | Absent | 8 (13.8%) | 12 (14.6%) | 20 | |

The causes of hyponatremia in cirrhosis are mainly caused by failure to expel free water in the urine as a result of non-osmotic release of vasopressin, increased renin- angiotensin-aldosterone system (RAAS), and generalized vasodilation. Such pathophysiological alterations are adequately explained in the recent AASLD rather, hyponatremia is mentioned as the characteristic of severe portal hypertension and circulatory dysfunction in cirrhosis.[11] In the same regard, the recent reviews also highlight that hyponatremia is a sign of disease progression and is connected with such complications as ascites, hepatorenal syndrome, and hepatic encephalopathy.[12,13]

The rate of hyponatremia in this study (41.4%) is in line with a number of recent literature. The prevalence of 41.22% in patients with chronic liver disease in a tertiary care environment reported by Bhandari et al is virtually the same as our results.[14] Similarly, a higher prevalence of 49 percent was observed by Singh et al. with patients having cirrhosis.[15] Such differences can be explained by discrepancies in the populations of the study, the severity of liver disease, and diagnostic qualifications applied to hyponatremia.

Praharaj et al. indicated that the prevalence of hyponatremia in cirrhosis is quite not universal as the sodium threshold applied but found that the prevalence of hyponatremia in cirrhosis with a serum sodium below 135 mmol/L, but not below 130 mmol/L, was found to be 49.4 and 21.6, respectively.[12] This underscores the significance of the operational definitions in the explanation of the prevalence rates. Our results are similar to those of the research involving the use of the definition based on the use of the 135 mmol/L that is generally accepted in clinical practice. Our findings are also supported by regional studies. In a recent Pakistani study, Azam et al. indicated hyponatremia in 36.09 percent of patients who had decompensated chronic liver disease.[16] The lower prevalence slightly than ours can be associated with the sample size, severity of the disease or the healthcare environments. In general, the prevalence among the South Asian communities is seen to be between 35-50 percent with our results falling within the expected range. The incidence of hepatic encephalopathy was 64.3 percent in our study, and it is also in line with the recent literature. According to the EASL Clinical Practice Guidelines, HE is a frequent complication of advanced cirrhosis especially in patients who have ascites and metabolic imbalances including hyponatremia.[17] The frequency of HE in our study is relatively high which could be evidence of high decompensated cirrhosis cases in our sample. Of special interest is the correlation between the hyponatremia and hepatic encephalopathy seen in this study. We discovered that 82.8% patients with HE had hyponatremia as compared to 51.2% without hyponatremia. This excellent correlation helps to confirm the hypothesis that hyponatremia leads to the occurrence and the progression of HE. The pathophysiology is based on the osmotic mismatch in the cells of the brain, which makes the astrocytes swell and become more prone to ammonia toxicity. New researches also corroborate this association. Li et al. have reported that, patients with cirrhosis and ascites with hyponatremia were at a higher risk of developing hepatic encephalopathy (56.2 vs 39.0) than without hyponatremia.[18] The absolute percentages will vary with our study but the direction of association is similar. On the same note, latest AASLD guidelines on acute-on-chronic liver failure indicate that electrolyte imbalances, such as hyponatremia, can be significant factors that lead to neuronal dysfunction among cirrhotic patients.[19] This supports the clinical significance of serum sodium monitoring as a routine examination of the patient. Rudler et al. study among critically ill patients with cirrhosis revealed that hyponatremia occurred in 22 percent of the patients with overt hepatic encephalopathy and was frequently related with other triggering factors such as infection and acute kidney disease.[20] Notably, high in-hospital mortality rate (50 percent) was also reported among these patients, which implies that hyponatremia could be a symptom of a bad prognosis and not necessarily a causative factor of hyponatremia. This could be the reason why our study showed that the percentage of HE among the hyponatremic patients was higher because our cohort was probably more advanced.

The recent treatment researches also emphasize the need to treat hyponatremia. Zaccherini et al. reported that long-term albumin therapy had a great effect on normalization of serum sodium level (45% vs 28% and increased incidences of moderate to severe hyponatremia were reduced-7). On the same note, Kulkarni et al. established that albumin infusion was linked to hyponatremia and hepatic encephalopathy outcomes that showed improved survival rates among patients with no acute-on-chronic liver failure.[21] A meta-analysis study by Zhou et al. recently verified another study revealing that albumin therapy has a significant effect on the reduction of hyponatremia in cirrhotic patients (odds ratio 0.67, 95 percent CI 0.530.85).[22] These results indicate that hyponatremia treatment can have therapeutic implications other than normalizing electrolytes, which might positively affect the neurological outcome. Moreover, recent reviews of experts emphasise the fact that hyponatremia must be viewed as an important prognostic factor in cirrhosis. Flores et al. noted that hyponatremia is a complication that is linked to high morbidity, hospitalizations, and mortality in cirrhotic patients.[23] Along with constant control and attentive correction of the sodium level, the AGA Clinical Practice Update also suggests consistent monitoring and monitoring of these complications, which can be hepatic encephalopathy and the syndrome of osmotic demyelination.[24] In general, the results of the current research are well correlated with recent literature. The incidence of observed hyponatremia is in line with the reported world and country-specific data, and its risk exposure with hepatic encephalopathy is highly supported by modern literature. The increased percentage of HE in hyponatremic patients in our study may be due to the variation in the severity of the disease, the choice of patients, or the accessibility of healthcare. The study has a number of strengths that contribute to its reliability and clinical relevance. It contains a clear purpose that aims at the frequency of hyponatremia and its relation with hepatic encephalopathy in patients with liver cirrhosis about a significant clinical problem. A descriptive cross-sectional design is suitable in establishing prevalence and finding associations. A sample of 140 patients is sufficient to enhance the validity of the results and the standardized definitions (serum sodium <135 mEq/L) and inclusion of West Haven criteria guarantee uniformity in measurements. Moreover, appropriate statistical analysis with SPSS and Chi-square further enhanced the level of scientific rigour, as statistically significant association between the two conditions was found. The stratified analysis of the demographic and clinical variables gives more detailed understanding and the fact that the study has been carried out in the environment of a real-life hospital makes it more applicable.

Limitations: The research has a number of limitations that ought to be taken into consideration when interpreting the findings. To begin with, the study, since it is not a multicenter type, is also not conducted in the general population but in a tertiary care unit. Secondly, cross-sectional design does not allow one to reach a causal relationship between hyponatremia and hepatic encephalopathy because it is only possible to obtain associations. Thirdly, concurrent sampling could be subject to selection bias due to non-probability consecutive sampling. Also, serum sodium levels and hepatic encephalopathy were measured at a single point in 24 hours of admission, which are not always dynamic. Such possible confounding variables as infections, medications, and nutritional status were not properly managed. Lastly, the sample size is relatively small and can be associated with a lack of statistical power and accuracy of subgroup analyses.

## CONCLUSION:

Hyponatremia is a frequent consequence of liver cirrhosis and is closely associated with hepatic encephalopathy, a sign of a more severe illness. Early detection and appropriate treatment of low sodium levels may be feasible in order to mitigate complications and enhance patient outcomes. The results indicate that hyponatremia is an effective marker of severe liver disease and brain conditions. Accordingly, frequent check-ups and regular check of the serum sodium levels of patients with cirrhosis can mitigate the identification of the high-risk group at an early stage, which in turn minimizes morbidity and enhances the clinical outcomes.

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**Authors Contribution:**
**Noor Ehsan:** Conception and Design, acquisition of data, analysis and interpretation of data, drafting and critical revision, final approval of the version to be published.
**Mahnoor Iqbal:** Conception and Design, acquisition of data, analysis and interpretation of data, drafting and critical revision, final approval of the version to be published.
**Hateem Ahmed:** Conception and Design, acquisition of data, analysis and interpretation of data, drafting and critical revision, final approval of the version to be published.
**Wafa Qaiser:** Acquisition of data, drafting and final approval of the manuscript.
**Imran Farooka:** Acquisition of data, drafting and final approval of the manuscript.
**Mahmood Nasir Malik:** Conception, acquisition of data, critical revision of the manuscript

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