Section: General Medicine

Original Research Article

STUDY OF HYPONATREMIA IN CIRRHOSIS OF LIVER AND ITS PROGNOSTIC VALUE AMONG PATIENTS IN CIVIL HOSPITAL AIZAWL

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ABSTRACT

Background: Hyponatremia is the most common electrolyte abnormality in patients with advanced cirrhosis and portends a poor prognosis. It is associated with disturbances in regulation of water balance leading to abnormalities in serum sodium. Various studies have established a correlation between serum sodium levels and survival in these patients. Dilutional Hyponatremia due to impaired free water clearance is the most common dysnatremia which has been reported in studies. The aim of this study was to study the serum sodium levels in patients with Cirrhosis of liver and to establish its prognostic value.

Materials and Methods: This observational study was conducted in department of General Medicine of Civil Hospital, Aizawl, Mizoram, India from September 2019 to December 2020, on patients with cirrhosis of liver. Informed consent will be obtained from all patients to be enrolled for the study. In all the patient's relevant information will be collected in a predesigned proforma. The patients are selected based on clinical examinations, biochemical tests, and ultrasound abdomen. Data were collected from 50 patients admitted in medical wards. Patients were divided into group based on serum sodium levels and the relevant parameters analyzed among the groups.

Results: Among 50 patients, 25 (50%) had serum sodium levels ≥136 mEq/L, while 14(28%) had serum sodium levels between 131 and 135 mEq/L. 11(22%) patients had serum sodium level ≤130 mEq/L. No patients had serum sodium levels greater than 145 mEq/L. Serum sodium levels was associated strongly with the severity of liver disease as assessed by Child Pugh and MELD-Na score. Serum sodium ≤130 mEq/L indicated the existence of Portal Hypertension (p value- 0.036), Hepatic Encephalopathy (p value<0.0001), GI Bleeding (p value- 0.0314, hepatorenal syndrome (p value- 0.0401), SBP (p value <0.0001). Patients with serum sodium less than 130 mEq/L had increased frequency of complications than those with ≥136 mEq/L. Patient with serum sodium levels ≤130 mEq/L had increased mortality (27.27%; p value- 0.01581))

Conclusion: Hyponatremia is more common in DCLD, and low serum sodium levels are associated with increased frequency of complications such as hepatic encephalopathy, hepatorenal syndrome, spontaneous bacterial peritonitis, and GI bleeding. Lower serum sodium levels were associated with increased MELD CPS score and mortality indicating the inverse relationship between serum sodium levels and severity of the disease.

Keywords: Hyponatremia, Hypernatremia, Decompensated Chronic Liver Disease.

INTRODUCTION

The normal range of serum sodium is 135-145 mEq/L. Its homeostasis is vital to the functioning of the cell. Hyponatremia is defined as concentration of sodium less than 135 mEq/L. It occurs when there is excess of water in relation to sodium. It is associated with disturbance in water homeostasis leading to dysnatremias.[1] It is the most common electrolyte disorder in hospitalized patients and more so in liver cirrhosis patients.[2] A disturbance in total body water regulation leading to decreased clearance of solute free water and the consequent inability to match the urine output to the amount of water ingested leads to dilutional hyponatremia. Recent studies have reported that lower serum sodium levels were associated with increased complications and mortality leading to incorporation of sodium in the MELD- Na score.[3,4]

Therefore, we undertook this study in our tertiary hospital to study serum sodium levels in patients admitted with cirrhosis of liver and to establish its prognostic value. Hyponatremia is a common electrolyte abnormality in cirrhosis of liver, as a result of high serum level of renin/aldosterone due to portal hypertension, a decreased vascular response to vasoactive drugs and reduced solute free water clearance. In patients with cirrhosis of liver, use of diuretics is one of the causes for hyponatremia. Hyponatremia has been associated with refractory ascites, spontaneous bacterial peritonitis, and hepatic encephalopathy in patients with cirrhosis. Hyponatremia can be a key prognostic factor in patients with cirrhosis of liver when it is added to MELD Score.[5] Thus, hyponatremia could be useful in predicting prognosis & development of complications in cirrhotic patients. Various studies are currently assessing role of hyponatremia as an independent risk factor for complications of cirrhosis and its possible implication in prevention.

MATERIALS AND METHODS

The study was conducted among 50 patients admitted with Cirrhosis of liver in Department of General Medicine of Civil Hospital, Aizawl during the study period of one year from September 2019 to December 2020.

Ethical Committee clearance obtained from Institution and Informed consent was obtained from the patients and enrolled in the study. The data of the patients were collected using a proforma. The first section of the proforma contains patient's demographic profile with detailed history. The second section contains detailed clinical examination that was carried out at the time of admission. The third section contains investigations that were done to aid the diagnosis and the serum sodium level. Patients were selected based on history, examination, laboratory investigations and imaging suggestive of the diagnosis of Cirrhosis of Liver. The presence of various complications and the outcome of the patients were monitored. The severity of the disease was calculated using MELD score and Child Pugh Score. Ascites was classified in to three grades:

Grade I- presence on examination not clear but observed in imaging;

Grade II- easily made out examination and palpation;

Grade III- severe abdominal distension requiring large volume paracentesis. Hepatic Encephalopathy was graded using West Haven Criteria.

Inclusion Criteria: All patients with Cirrhosis of liver diagnosed by examination, laboratory investigations and radiological imaging.

Exclusion Criteria: Patients with cardiac failure, diuretic therapy, chronic kidney disease and drugs such as SSRIs, TCA, MAO inhibitors, cytotoxic drugs etc.,

The collected data were entered in a Microsoft Excel Sheet. Graphs and tables were generated using Microsoft Word and Microsoft Excel. Statistical analyses were done using medcalc 15.8, Minitab 17, IBM SPSS 22. Quantitative data was analysed using Mean, Median, Mode and Standard Deviation (SD). Qualitative data was analysed using Chi Square Test, One way ANOVA and Fisher's test & Odds ratio & Confidence Interval. Difference between two variables is considered significant when 'p' value is less than 0.05.

RESULTS

A total of 50 patients with decompensated chronic liver disease (DCLD) admitted to the hospital during the study period were included in the study. The demographic characteristics of the study population are shown in Table 14. The mean age of the patients was 44.86 ± 10.97 years (range: 28–70 years). Of the 50 patients, 38 (76%) were males and 12 (24%) were females, giving a male-to-female ratio of 3.17:1. Alcohol-related liver disease was the commonest etiology (82%), followed by hepatitis C (10%) and hepatitis B (8%). The mean MELD score was 22.14 ± 7.04, while the mean serum sodium concentration was 134.56 ± 5.74 mEq/L. Based on serum sodium concentration, 22% of patients had serum sodium ≤130 mEq/L, 28% had serum sodium between 131–135 mEq/L, and 50% had serum sodium ≥136 mEq/L.

Table 1: Demographic characteristics of the study population

Parameter	Number of patients	Percentage	Mean	SD
Age (years)	50	–	44.86	10.97
Gender				
Male	38	76	–	–
Female	12	24	–	–

Etiology of cirrhosis				
Alcohol	41	82	–	–
HBV	4	8	–	–
HCV	5	10	–	–
Others	0	0	–	–
MELD score	–	–	22.14	7.04
Serum sodium (mEq/L)	–	–	134.56	5.74
≤130	11	22	–	–
131–135	14	28	–	–
≥136	25	50	–	–

Note: Data are presented as mean ± SD or number (%).

Patients were categorized into three groups according to serum sodium concentration (≤130, 131–135 and ≥136 mEq/L). There was no statistically significant difference in age (p=0.356), gender (p=0.8334) or etiology (p=0.6399) among the three groups. However, serum sodium concentration showed a significant association with Child–Pugh class (p<0.0001) and MELD score (p=0.0052). Patients with serum sodium ≤130 mEq/L had the highest mean MELD score (**27.90 ± 4.64**).

Table 2: Comparison of patient characteristics according to serum sodium concentration

Variable	≤130 mEq/L (n=11)	131–135 mEq/L (n=14)	≥136 mEq/L (n=25)	p-value
Mean age (years)	41 ±12.23	47 ±13.70	45 ±8.43	0.356
Child-Pugh A	0	1	2	
Child-Pugh B	3	6	11	
Child-Pugh C	8	7	12	<0.0001
Mean MELD score	27.90 ±4.64	21.42 ±7.45	20.00 ±6.49	0.0052
Male (%)	72.7	78.6	76.0	0.8334
Alcohol etiology (%)	81.8	85.7	80.0	0.6399

Note: Continuous variables were compared using one-way ANOVA. Categorical variables were analyzed using the Chi-square test.

The clinical presentation of patients at admission is shown in Table 16. Abdominal distension and bilateral lower limb edema were present in all patients (100%). Jaundice was present in 62%, gastrointestinal bleeding in 22%, and altered sensorium in 20%.

Table 3: Clinical presentation of patients at admission

Clinical presentation	Number	Percentage
Abdominal distension	50	100
Lower limb swelling	50	100
Jaundice	31	62
Altered sensorium	10	20
Gastrointestinal bleeding	11	22

Note: Values are expressed as frequency and percentage.

Ascites was present in all patients (100%), followed by coagulopathy (72%), portal hypertension (70%), gastrointestinal bleeding (58%), hepatic encephalopathy (24%), hepatorenal syndrome (14%), and spontaneous bacterial peritonitis (14%).

Table 4: Distribution of complications

Complication	Number	Percentage
Ascites	50	100
Portal hypertension	35	70
Hepatic encephalopathy	12	24
Gastrointestinal bleeding	29	58
Coagulopathy	36	72
Hepatorenal syndrome	7	14
Spontaneous bacterial peritonitis	7	14

Note: Values are presented as frequency and percentage.

Patients with lower serum sodium concentrations had significantly higher frequencies of portal hypertension, hepatic encephalopathy, gastrointestinal bleeding, hepatorenal syndrome and spontaneous bacterial peritonitis.

Table 5: Comparison of complications according to serum sodium concentration

Complication	≤130 (n=11)	131–135 (n=14)	≥136 (n=25)	p-value
Ascites	11 (100%)	14 (100%)	25 (100%)	1.000
Portal hypertension	11 (100%)	10 (71.4%)	14 (56%)	0.036
Hepatic encephalopathy	11 (100%)	1 (7.1%)	0	<0.0001
Gastrointestinal bleeding	9 (81.8%)	10 (71.4%)	10 (40%)	0.0314

Coagulopathy	10 (90.9%)	10 (71.4%)	16 (64%)	0.2532
Hepatorenal syndrome	4 (36.4%)	0	3 (12%)	0.0401
SBP	7 (63.6%)	0	0	<0.0001

Note: Chi-square test was used for comparison.

Table 6: Odds ratio of complications according to serum sodium concentration

Complication	OR (≤130 vs ≥136)	p-value	OR (131–135 vs ≥136)	p-value
Ascites	0.45	1.000	0.57	1.000
Portal hypertension	18.24	0.015	1.96	0.496
Hepatic encephalopathy	1173.00	<0.0001	5.67	0.359
Gastrointestinal bleeding	6.75	0.0312	3.75	0.0958
Coagulopathy	5.63	0.1274	1.41	0.7334
Hepatorenal syndrome	4.19	0.1665	0.22	0.5404
SBP	85.00	<0.0001	1.76	1.000

Note: Odds ratios are presented with 95% confidence intervals. The reference group was patients with serum sodium ≥136 mEq/L.

Mortality increased significantly with decreasing serum sodium concentration. Three patients (27.3%) in the ≤130 mEq/L group died compared with one patient (7.1%) in the 131–135 mEq/L group, whereas no deaths occurred in patients with serum sodium ≥136 mEq/L.

Table 7: Mortality according to serum sodium concentration

Serum sodium group	Deaths	Percentage	p-value
≤130 mEq/L (n=11)	3	27.27	
131–135 mEq/L (n=14)	1	7.14	
≥136 mEq/L (n=25)	0	0	**0.0158**

Note: Chi-square test was used to compare mortality among the three groups.

DISCUSSION

The present study evaluated the association between serum sodium concentration and the severity, complications, and mortality of decompensated chronic liver disease (DCLD). Hyponatremia is a common electrolyte abnormality in patients with advanced cirrhosis and reflects impaired renal free water excretion secondary to portal hypertension, splanchnic vasodilatation, and activation of neurohumoral mechanisms. In the present study, lower serum sodium concentration was significantly associated with greater disease severity, increased frequency of complications, and higher in-hospital mortality.

Hyponatremia was a common finding in the present study. Half of the patients (50%) had serum sodium concentrations below 136 mEq/L, including 22% with serum sodium ≤130 mEq/L and 28% with serum sodium between 131 and 135 mEq/L. No patient had serum sodium levels greater than 145 mEq/L, indicating that hypernatremia was uncommon in our cohort.

The distribution of serum sodium concentration observed in the present study was remarkably similar to that reported in previous studies. Angeli et al,[6] reported that 21.6% of patients had serum sodium ≤130 mEq/L, 27.8% had serum sodium between 131 and 135 mEq/L, and 50.6% had normal serum sodium concentrations. Similarly, Jong Hoon Kim et al,[7] observed corresponding frequencies of 27.1%, 20.8%, and 52.1%, while Shaikh et al. reported 26.7%, 24.9%, and 48.4%, respectively. Borroni et al,[8] also demonstrated that 29.8% of patients had serum sodium ≤130 mEq/L. The close agreement between these studies and the present findings supports the observation that hyponatremia is a frequent manifestation of advanced cirrhosis irrespective of geographical location or patient population.

Table 8: Comparison of studies showing distribution of patients according to serum sodium concentration

Study	≤130 mEq/L	131–135 mEq/L	≥136 mEq/L
Present study	22%	28%	50%
Angeli et al[6]	21.6%	27.8%	50.6%
Jong Hoon Kim et al[7]	27.1%	20.8%	52.1%
Shaikh et al.[9]	26.7%	24.9%	48.4%
Borroni et al.[8]	29.8%	—	—

Patients with lower serum sodium concentrations had more severe ascites in the present study. Although ascites was present in all patients because only decompensated cirrhosis was included, patients with serum sodium ≤130 mEq/L had clinically more advanced disease.

This observation is consistent with previous studies. Arroyo et al,[10] demonstrated that patients with serum sodium concentrations below 130 mEq/L had reduced glomerular filtration rate and impaired free water clearance, resulting in poor response to diuretic therapy and frequent need for large-volume paracentesis. Similar observations were reported by

Angeli et al,[6] and Bernardi et al,[11] who found that hyponatremic patients had refractory ascites and poorer response to diuretics than normonatremic patients. Thus, the findings of the present study further support the close relationship between hyponatremia and severe portal hypertension with refractory ascites.

One of the most important observations of the present study was the strong association between hyponatremia and hepatic encephalopathy. Hepatic encephalopathy occurred in 100% of patients with serum sodium ≤130 mEq/L compared with 7.14% of patients with serum sodium between 131 and 135 mEq/L, while no patient with serum sodium ≥136 mEq/L developed hepatic encephalopathy. This association was highly significant (p<0.0001).

Previous investigators have also demonstrated a similar relationship, although the frequency observed in our study was higher. Angeli et al,[6] reported hepatic encephalopathy in 38%, 24%, and 15% of patients with serum sodium ≤130, 131–135, and ≥136 mEq/L, respectively. Jong Hoon Kim et al,[12] reported corresponding frequencies of 43.1%, 35.8%, and 24.4%, while Shaikh et al. reported hepatic encephalopathy in 25.8% of patients with serum sodium ≤130 mEq/L.

The higher prevalence observed in the present study may be explained by the relatively small sample size and inclusion of only hospitalized patients with decompensated cirrhosis, who generally have more advanced liver disease.

Table 9: Comparison of studies showing association between serum sodium concentration and hepatic encephalopathy

Study	≤130 mEq/L	131–135 mEq/L	≥136 mEq/L
Present study	100%	7.14%	0%
Angeli et al.[6]	38%	24%	15%
Jong Hoon Kim et al.[7]	43.1%	35.8%	24.4%
Shaikh et al.[9]	25.8%	—	—

The present study demonstrated a higher frequency of hepatorenal syndrome among patients with serum sodium ≤130 mEq/L (36.36%) compared with patients having higher serum sodium concentrations. Angeli et al,[6] reported hepatorenal syndrome in 17%, 10%, and 6% of patients with serum sodium ≤130, 131–135, and ≥136 mEq/L, respectively, whereas Jong Hoon Kim et al,[7] reported frequencies of 3.9%, 2.5%, and 3%, respectively. Although the prevalence observed in our study was higher, the overall trend remained consistent, suggesting that worsening hyponatremia is associated with progressive circulatory dysfunction and renal impairment in cirrhosis.

Table 10: Comparison of studies showing association between serum sodium concentration and hepatorenal syndrome

Study	≤130 mEq/L	131–135 mEq/L	≥136 mEq/L
Present study	36.36%	0%	12%
Angeli et al.[6]	17%	10%	6%
Jong Hoon Kim et al.[7]	3.9%	2.5%	3%

The present study found a significant association between hyponatremia and spontaneous bacterial peritonitis (SBP). Among patients with serum sodium ≤130 mEq/L, **63.64%** developed SBP, whereas no patient in the other two groups developed SBP.

Jong Hoon Kim et al. reported SBP in 33.3%, 30.7%, and 16.3% of patients with serum sodium ≤130, 131–135, and ≥136 mEq/L, respectively. Angeli et al. also observed an increased frequency of SBP in hyponatremic patients. Although the absolute frequencies differed, both studies support the finding that hyponatremia identifies patients at increased risk of bacterial infections.

Table 11: Comparison of studies showing association between serum sodium concentration and spontaneous bacterial peritonitis

Study	≤130 mEq/L	131–135 mEq/L	≥136 mEq/L
Present study	63.64%	0%	0%
Jong Hoon Kim et al.[7]	33.3%	30.7%	16.3%

Unlike previous reports by Angeli et al,[6] Jong Hoon Kim et al,[7] and Shaikh et al,[9] which found no significant association between serum sodium concentration and gastrointestinal bleeding, the present study demonstrated a significantly higher frequency of gastrointestinal bleeding among patients with serum sodium ≤130 mEq/L (81.81%). This difference may reflect the more advanced disease severity of patients included in the present study and the relatively smaller sample size.

The present study demonstrated that patients with lower serum sodium concentrations had significantly higher MELD scores and Child–Pugh scores, indicating more advanced liver disease. Patients with serum sodium ≤130 mEq/L had a mean MELD score of **27.90 ± 4.64**, compared with **21.42 ± 7.45** and **20.00 ± 6.49** among patients with serum sodium concentrations of 131–135 and ≥136 mEq/L, respectively. Similarly, the mean Child–Pugh score

was highest among patients with serum sodium ≤130 mEq/L (**11.54 ± 2.84**).

These findings agree with those of Jong Hoon Kim et al., who also reported increasing MELD and Child–Pugh scores with decreasing serum sodium concentration, supporting the concept that hyponatremia reflects advanced portal hypertension and circulatory dysfunction.

Table 12: Comparison of studies showing association between serum sodium concentration and MELD score

MELD score	**≤130 mEq/L**	**131–135 mEq/L**	**≥136 mEq/L**
Present study	27.90 ± 4.64	21.42 ± 7.45	20.00 ± 6.49
Jong Hoon Kim et al.[7]	17.20 ± 5.10	16.30 ± 5.20	13.90 ± 4.60
Child–Pugh score			
Present study	11.54 ± 2.84	9.21 ± 1.67	9.12 ± 1.81
Jong Hoon Kim et al.[7]	10.50 ± 1.60	9.80 ± 1.70	8.10 ± 1.60

Mortality increased progressively with decreasing serum sodium concentration. In the present study, mortality was **27.27%** among patients with serum sodium ≤130 mEq/L, **7.14%** among those with serum sodium between 131 and 135 mEq/L, and **0%** among patients with serum sodium ≥136 mEq/L.

Table 13: Mortality according to serum sodium concentration

Serum sodium concentration	**Mortality**
≤130 mEq/L	27.27%
131–135 mEq/L	7.14%
≥136 mEq/L	0%

These findings indicate that hyponatremia is a strong predictor of poor prognosis in patients with decompensated cirrhosis. The progressive increase in mortality with decreasing serum sodium concentration supports the incorporation of serum sodium into prognostic models, such as the MELD-Na score, for improved risk stratification and prioritization of patients for liver transplantation. Overall, the findings of the present study are consistent with previously published literature and further emphasize that serum sodium concentration is a simple, inexpensive, and clinically useful marker of disease severity, complications, and prognosis in patients with decompensated chronic liver disease.

CONCLUSION

Cirrhosis of Liver is associated with abnormal serum sodium concentration. Hyponatremia is the most common abnormality in this study. Age, gender and cause of DCLD did not have any association with serum sodium levels. Serum sodium levels less than 135 mEq/L is associated with increased frequency of complications such as Hepatic Encephalopathy, Hepatorenal Syndrome, Spontaneous Bacterial Peritonitis and GI Bleeding when compared to patients with serum sodium levels ≥136 mEq/L. Patients with serum sodium concentration less than 130 mEq/L are the most affected. Lower serum sodium levels are associated with increased MELD score, increased CPS score and increased mortality indicating the inverse relationship between serum sodium levels and the severity of disease. Thus, patients with decreased serum sodium levels should be considered a high-risk population because of the increased frequency of complications and mortality.

Limitations

This study did not assess the serum sodium concentration on the risk for developing complications but showed the concurrent presence of complications and serum sodium levels.

Recommendations

Further studies are needed to determine the clinical significance of Hyponatremia and identify its correlation with the incidence of possible complications. Moreover, studies should be conducted on incorporating serum sodium in new Liver Cirrhosis prognostic models and on demonstrating the efficacy of other sodium incorporated models.

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