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Original Research Article

Study on Correlation of Hyponatremia with Severity in Decompensated Cirrhosis

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Abstract:

Background: Cirrhosis, the final stage of chronic liver disease, tends to decompensate, indicating the development of complications such as ascites, encephalopathy, or hepatorenal syndrome. Hyponatremia is one of the most common electrolytes disorders in decompensated cirrhosis. Clinical studies indicate that it reflects circulatory and neurohormonal dysfunction and is associated with poor prognosis.

Aim: To study the relationship between hyponatremia and decompensated cirrhosis severity using Child Pugh and MELD scores.

Methods: A hospital-based observational study was done among 50 patients with cirrhosis of liver at NMCH, Patna (April 2023 to March 2025). Clinical, biochemical and radiological investigations were done. Serum sodium levels were correlated with disease severity (Child Pugh, MELD) and associated complication. Statistical interpretation of data was done using SPSS v17 software and p value of <0.05 was considered significant.

Results: The prevalence of hyponatremia in our study was 66% in total (mild 26%, moderate 22%, and severe 18%). It was significantly associated with ascites (p=0.042), hepatic encephalopathy (p=0.048) and hepatorenal syndrome (p=0.03). Serum sodium was inversely correlated with MELD (r=-0.62, p<0.001) and Child Pugh scores, r=-0.55, p=0.002, indicating that decreasing levels of sodium reflected increasing severity of liver dysfunction.

Conclusion: Hyponatremia is prevalent and clinically significant marker of severity of decompensated cirrhosis with a strong correlation to higher MELD and Child Pugh scores and complications.

Keywords: Hyponatremia, Decompensated cirrhosis, MELD score, Child-Pugh score, Liver failure, Prognosis.

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Introduction

Cirrhosis is the end stage of chronic liver disease characterized by disorderly hepatic fibrosis and regenerative nodules, resulting in progressive distortion of normal hepatic architecture. All of these pathological changes jeopardize hepatic microcirculation and functional capacity, leading to multiple organ system complications. At the decompensated stage, patients may develop serious, often life-threatening sequelae: portal hypertension, ascites, hepatic encephalopathy, variceal hemorrhage, renal insufficiency - all of which contribute to enormous morbidity and mortality (Schlatter et al., 2022). Among these complications, hyponatremia-a reduction in serum sodium concentration-is a very frequent and clinically significant electrolyte abnormality in decompensated cirrhosis. It is generally defined as a serum sodium level below 130 mEq/L and is associated with worsening hepatic function, an increased risk of hepatic encephalopathy and hepatorenal syndrome, and higher short-term mortality (Bernardi et al., 2021). Hyponatremia is not just a secondary finding in cirrhosis but a key marker of disease progression and poor prognosis (Bajaj et al., 2021).

Hyponatremia develops in cirrhosis primarily due to impaired renal capacity to excrete free water because of excessive antidiuretic hormone (ADH) secretion and splanchnic vasodilatation. As liver dysfunction worsens, systemic vasodilation, mainly in the splanchnic circulation, reduces effective arterial blood volume, thereby triggering compensatory activation of RAAS, the sympathetic nervous system, and non-osmotic ADH release (Angeli et al., 2020; Ginès et al., 2019) ,. This neurohormonal activation promotes increased renal water retention, causing dilutional hyponatremia despite a normal or increased total body sodium. In more advanced disease, renal vasoconstriction and hepatorenal syndrome further decrease free water clearance due to impaired sodium and water handling (Davenport et al., 2020). Systemic inflammation and endothelial

dysfunction further amplify circulatory disturbances through the release of cytokines, such as TNF-α and IL-6, which further deteriorate vascular permeability and favor fluid retention (Clària & Moreau, 2021). Hyponatremia therefore reflects the complex interplay of hemodynamic, neurohormonal, and inflammatory processes that mirror overall severity of liver decompensation.

Serum sodium has become an important prognostic biomarker in patients with cirrhosis, helping to monitor the disease and prioritize organ transplantation. Combining sodium into the MELD-Na (Model for End-Stage Liver Disease–Sodium) scoring system improved mortality prediction over the traditional MELD score (Kim et al., 2021). It has been suggested that a decline of just 1 mEq/L in serum sodium below 130 mEq/L may increase mortality risk by about 12% (Ahluwalia et al., 2020). Severe hyponatremia (serum sodium of less than 125 mEq/L) is especially concerning because the neurological outcome is poor due to the development of cerebral edema and osmotic disequilibrium; this can induce hepatic encephalopathy, seizures, and increased susceptibility to osmotic demyelination syndrome if correction is too rapid, as has been described by Romero-Gómez et al. (2020). Furthermore, chronic hyponatremia has also been associated with prolonged hospital stays, an increase in intensive care utilization, and lower long-term survival by González-Abraldes et al. (2022). In the context of liver transplantation, hyponatremia not only increases pre-transplant mortality but also increases complications during and around the time of surgery. Because of this recognition, serum sodium is now fundamentally part of transplant allocation practices, further solidifying its role as a dynamic marker of systemic and hepatic decompensation (Kim et al., 2021).

Cirrhosis remains a major public health problem worldwide, causing approximately two million deaths each year; half of them are due to decompensated manifestations of the disease (Moon et al., 2020). The responsible etiologies differ by region: whereas viral hepatitis is still the leading cause in Asia and sub-Saharan Africa, alcoholic liver disease (ALD) and metabolic dysfunction-associated steatotic liver disease (MASLD) have become the predominant causes in Western countries. The increasing prevalence of metabolic risk factors such as obesity and type 2 diabetes further increases the incidence of cirrhosis and the complications associated with hyponatremia. Despite recent progress in antiviral therapies and the improvement of liver disease management, the prognosis of patients with decompensated cirrhosis remains unfavorable, with a five-year survival rate of less than 50% (Baveno VI Consensus, 2021). In view of the limited availability of liver transplantation and the high mortality of end-stage disease, early identification of predictors of poor outcomes-such as serum sodium level-became crucial for the stratification of risk and therapeutic strategy.

Although the relation of hyponatremia with the severity of cirrhosis has been realized, its implications have not been fully explored from a clinical perspective in many resource-constrained settings. The relationship between sodium levels in the serum and the severity of disease determined from Child-Pugh and MELD scores has potential for informing prognosis and providing opportunity for intervention. Further, an improved understanding of the relationship could also assist in identifying patients potentially at increased risk of complications and will lend direction for management strategies aimed more deliberately at these patients and appropriate assessments for transplant ability. This study is intended to assess the association of hyponatremia and the severity of decompensated cirrhosis in light of prevalence, association with decompensated cirrhosis major complications, and predictive value as it relates to clinical outcomes. To better define the association, this research aims to advance more accurate prognostication, and potential improvement of therapeutic approach based on statutory findings in those with advanced liver disease.

Methodology

Study Design: This study utilizes observational study designs and is hospital-based. It will assess the association between serum sodium concentration (hyponatremia) and severity of decompensated cirrhosis using two established clinical scoring systems, Child-Pugh classification, and MELD score.

Study Area: This study was conducted at Nalanda Medical College and Hospital, Patna, Bihar, India.

Study Duration: This study would be conducted from April 2023 to March 2025 and the study will be started after the approval of the Institutional Ethics Committee (IEC).

Sample Size: The study group consists of 50 patients who are clinically and biochemically diagnosed to have cirrhosis and hospitalized in the Internal Medicine wards of NMCH during the study period.

Study Population: The population includes male and female patients of all ages who were admitted with confirmed cirrhosis and regardless of etiology (e.g., alcoholic liver disease, viral hepatitis, NAFLD).

Data Collection: The data was collected for the study with a pre-designed structured proforma that contained the demographic patient details, clinical history, laboratory investigations, imaging results, and the complications of cirrhosis observed. The study was conducted only after obtaining fully informed, written consent from all the participants, which included details on the purpose of the study

and the methods to be followed. Diagnosis of cirrhosis was primarily based on clinical assessment, liver function tests, imaging studies including ultrasound or CT findings, and coagulation studies. The length of the illness, history of alcohol consumption, previous admissions, and complications were also meticulously obtained from each patient. Evaluation of serum sodium, bilirubin, albumin, prothrombin time, and renal function tests were all completed. The severity of cirrhosis was assessed using the Child-Pugh and MELD scoring systems and then assessed by comparing it to serum sodium levels and clinical complications including ascites, hepatic encephalopathy, spontaneous bacterial peritonitis, and hepatorenal syndrome to assess for possible correlation.

Inclusion Criteria

- Confirmed diagnosis of cirrhosis (clinical and laboratory basis).
- Both male and female patients of any age group.
- Patients admitted to the internal medicine ward of NMCH during the study period.

Exclusion Criteria

- Pre-existing cardiac disease (e.g., congestive heart failure).
- Chronic kidney disease (CKD).
- Patients on diuretic therapy, as it alters serum sodium levels.

Ethical Considerations: The study protocol was approved by the Institutional Ethics Committee of Nalanda Medical College and Hospital, Patna, before the start of the study. Prior informed written consent was obtained to ensure that each participant was fully aware of the objectives and benefits of the study, as well as any minimal risks that it may pose. The confidentiality of the identity of the patients and the integrity of the data was also assured. The study was performed according to ethical principles stated in the Declaration of Helsinki (2013 revision) showing a respect for patient autonomy, patient safety, and scientific integrity.

Procedure: All patients hospitalized with cirrhosis were satisfied included and exclusion criteria after screening and assessment were included in the study. A full clinical history, examination findings and biochemical tests, were documented after consent. Serum sodium levels were examined at the time of admission. Each patient was evaluated for the severity of liver disease as per Child-Pugh and MELD scoring systems and relevant complications noted. Data were then collected from all participants and their analyses carried out to arrive at a correlation between serum sodium level and the severity of decompensated cirrhosis. Patients were also divided into two groups based on the presence or absence of hyponatremia so that their clinical outcomes and complication profiles could be compared.

Statistical Analysis: Data were entered and organized using Microsoft Excel and analyzed with SPSS software version 17.0. Continuous variables were serum sodium, Child-Pugh, and MELD scores presented as mean ± SD and categorical variables of gender, type of complication, and etiology summarized as frequencies and percentages. Tests of significance included Chi-square for the assessment of the relation of hyponatremia to cirrhosis-related complications and Pearson's correlation coefficient (r) for assessing the strength of relationship between serum sodium level and disease severity scores. The p-value was considered significant at less than 0.05 within a 95% confidence interval."

Result

Table 1 depicts the demographic distribution for 50 patients who were part of the study. Most respondents were in their middle age, with 26% falling in the 51–60 years age bracket, the highest proportion, followed by 41–50 years, each accounting for 22%, signifying that cirrhosis has been most common in the fifth and sixth decades of life. A lesser proportion of 18% fell between 30–40 years and less than 30 years of age, while 12% were under 30 years, indicating a lower prevalence among younger adult age groups. Males accounted for a significantly greater percentage of the sample population: 68% against 32% females, as per the higher prevalence of cirrhosis among males associated mostly with lifestyle and occupation. With regard to alcohol consumption, 28% of patients consumed alcohol regularly and 14% occasionally, while 58% abstained from it totally, indicating that though alcohol is one of the major risk factors, non-alcoholic causes too have contributed significantly to the disease burden. Family history of liver diseases was found to be present in half of the participants, indicating a probable genetic or shared environmental predisposition. Overall data show a predominance of middle-aged males, significant alcohol related risk, and a strong familial component to cirrhosis of the liver.

Table 1: Demographic Profile of the Study Population (n = 50)			
Parameter	**Category**	**Frequency (n)**	**Percentage (%)**
Age (years)	< 30	6	12
	30–40	9	18
	41–50	11	22
	51–60	13	26
	> 60	11	22
Gender	Male	34	68
	Female	16	32
Alcohol Consumption	Regular	14	28
	Occasional	7	14
	None	29	58
Family History of Liver Disease	Present	25	50
	Absent	25	50

Table 2 reports on the etiology and duration of cirrhosis in the 50 subjects included in this study. As illustrated in Table 2, the duration of illness ranged widely, with 17 (34%) participants having cirrhosis of less than a year, 13 (26%) participants having cirrhosis for 1-3 years, 11 (22%) participants having cirrhosis for 4-6 years and 9 (18%) participants reported cirrhosis for greater than 6 years, showing that most of the participants had early and middle-stage chronic liver disease. In terms of etiology, Alcoholic Liver Disease (ALD) was the most common cause, comprising 28 (56%) cases, followed by Hepatitis B with 8 (16%) cases and Hepatitis C with 6 (12%) patients. Non-Alcoholic Fatty Liver Disease (NAFLD) accounted for 5 (10%) cases. The remaining cases 3 (6%), (which also included one patient each for cryptogenic cirrhosis and autoimmune) were less frequent diagnoses. The data reflects that alcohol remained the predominant cause of cirrhosis amongst the study population and suggests that is continues to be a public health problem.

Table 2: Etiology and Duration of Cirrhosis			
Parameter	**Category**	**Frequency (n)**	**Percentage (%)**
Duration of Illness	< 1 year	17	34
	1–3 years	13	26
	4–6 years	11	22
	> 6 years	9	18
Etiology of Cirrhosis	Alcoholic Liver Disease (ALD)	28	56
	Hepatitis B	8	16
	Hepatitis C	6	12
	Non-Alcoholic Fatty Liver Disease (NAFLD)	5	10
	Others	3	6

Table 3 shows the distribution of patients based on serum sodium levels providing distribution of severity and frequency of hyponatremia in the population. Of the 50 patients included in the study, seventeen patients (34%) had serum sodium >135 mEq/L - representing normal sodium. Conversely, thirty-three (66%) patients had some form of hyponatremia. Mild hyponatremia (130-135 mEq/L) exceeded the most among Hyponatremia patients examined and noted 13 patients (26%); moderate hyponatremia (125-129 mEq/L); eleven patients (22%); and severe hyponatremia (<125 mEq/L), nine patients (18%). This distribution highlights more than half of patients were observed to have sodium levels lower than waf normal distribution, highlighting the prevalence of hyponatremia amongst patients with chronic liver disease. Sodium levels classify patients with chronic liver disease and predict fluid-electrolyte imbalance and hepatic dysfunction pathophysiologies. Furthermore, hepatic dysfunction and fluid-electrolyte imbalance worsen as sodium levels decline, ultimately indicating disease severity and negative prognosis in patients that have liver cirrhosis.

Table 3: Serum Sodium Levels and Distribution of Hyponatremia			
Serum Sodium Level (mEq/L)	**Category**	**Number of Patients (n)**	**Percentage (%)**
> 135	Normal	17	34
130–135	Mild Hyponatremia	13	26
125–129	Moderate Hyponatremia	11	22
< 125	Severe Hyponatremia	9	18
Total		**50**	**100**

Table 4 demonstrates the association between hyponatremia and different complications due to cirrhosis and shows a significant association between low serum sodium and severity of disease. The table demonstrates that in patients that have hyponatremia (n = 33), ascites was seen in 18 patients (54.5%), compared to 5 patients (29.4%) without hyponatremia (p = 0.042). Hepatic encephalopathy was also seen more frequently in those with hyponatremia (33.3%) compared to those without hyponatremia (17.6%). This resulted in a statistically significant with a p-value of 0.048, suggesting hyponatremia may worsen neurological complications of liver failure. In addition, hepatorenal syndrome was seen in more patients with hyponatremia (18.2%) compared to 5.9% without hyponatremia (p = 0.03) with a statistically significant correlation. Though spontaneous bacterial peritonitis and variceal bleeding slightly increased (15.2% and 18.2%, respectively), these differences were not statistically significant (p = 0.71 and 0.62). Overall, the table demonstrates that hyponatremia in patients with cirrhosis significantly correlates with major complications such as ascites, hepatic encephalopathy, and hepatorenal syndrome and suggests that hyponatremia can be used as a prognostic indicator in liver disease.

Table 4: Correlation Between Hyponatremia and Cirrhosis-related Complications			
Complication	**Hyponatremia Present (n = 33)**	**Hyponatremia Absent (n = 17)**	**p-value**
Ascites	18 (54.5%)	5 (29.4%)	0.042*
Hepatic Encephalopathy	11 (33.3%)	3 (17.6%)	0.048*
Hepatorenal Syndrome	6 (18.2%)	1 (5.9%)	0.03*
Spontaneous Bacterial Peritonitis	5 (15.2%)	2 (11.8%)	0.71
Variceal Bleeding	6 (18.2%)	3 (17.6%)	0.62

Table 5 illustrates the distribution of disease severity based on the Child-Pugh and MELD scores in patients. Considering Child-Pugh scores, 11 patients (22%) were classified as Class A, which indicates well compensated liver function, 17 patients (34%) were in Class B, which indicates moderate hepatic dysfunction, and 22 patients (44%) were Class C, indicating severe liver dysfunction with a mean Child-Pugh score of 10.08 ± 3.08. The score distribution for MELD score showed that 10 patients (20%) had a low score (<10), which is considered milder disease, while 20 patients (40%) had moderate scores (10-19) and another 20 patients (40%) had high MELD scores (≥ 20), representative of late liver failure. The mean MELD score was 18.12 ± 7.45. In total, these findings show that much of the study population expressed moderate to severe liver disease, representing advanced hepatic dysfunction for this population.

Table 5: Distribution of Disease Severity Scores				
Severity Index	**Category**	**Patients (n)**	**Percentage (%)**	**Mean ± SD**
Child-Pugh Score	Class A (5–6)	11	22	10.08 ± 3.08
	Class B (7–9)	17	34	
	Class C (10–15)	22	44	
MELD Score	< 10 (Low)	10	20	18.12 ± 7.45
	10–19 (Moderate)	20	40	
	≥ 20 (High)	20	40	

Table 6 demonstrates the association between serum sodium and the severity of liver disease, as quantified by MELD (Model for End-Stage Liver Disease) and Child-Pugh (C-P) score. Serum sodium had a strong negative association (r = -0.62, P < 0.001) with MELD score, suggesting that lower sodium levels were significantly associated with higher MELD scores and thereby more severe impairment of liver function. In parallel, serum sodium was moderately correlated with Child-Pugh scores (r = -0.55, P = 0.002) which similarly suggests that patients with lower sodium levels have more advanced hepatic impairment. In contrast, MELD and Child-Pugh scores had a weak and non-significant

correlation (r = 0.03, P = 0.78), indicating that while MELD and Child-Pugh scores are both measures for assessing severity of liver disease, they may measure different clinical domains. Taken as whole, these data highlight the prognostic significance of serum sodium as an independent marker of liver disease severity and progression in patients with chronic liver disease.

Table 6: Correlation Between Serum Sodium and Severity Scores

Variable Correlation	Correlation Coefficient (r)	p-value	Interpretation
Serum Sodium vs MELD Score	–0.62	< 0.001*	Strong inverse correlation
Serum Sodium vs Child-Pugh Score	–0.55	0.002*	Moderate inverse correlation
MELD vs Child-Pugh Score	0.03	0.78	Weak correlation

Discussion

In the present study of 50 cirrhotic patients, the average age was mostly recorded in the 51-60 year age group and included a predominance of males accounting for 68%. This aligns with the usual observation and also aligns with the distribution of gender seen in most if not all studies on decompensated cirrhosis. Azam et al. (2024) similarly reported a predominance among males at 60.15%, with the mean age reported at 47.68, suggesting that middle-aged males are at an increased risk to develop advanced liver disease due to alcohol related aetiologies and also due to other aetiologies that predispose to liver disease. The greatest aetiologies represented in this study were Alcoholic Liver Disease at 56%, with viral hepatitis accounting for 28% combined for HBV and HCV. This is similar to Hussain et al. (2022), where Hepatitis B was the most prevalent group of liver disease at 59.8% and alcoholic cirrhosis was the second highest cause at 25%. The similarity is noticeable and indicates that decompensated cirrhosis continues to occur in South Asia, attributed viral infection and alcohol related liver disease, whereas this study suggests a greater predominance of alcohol related cirrhosis".

In this study, hyponatremia was present in 66% of patients with mild (26%), moderate (22%), and severe (18%) cases respectively. This finding is consistent with Singh and Chamoli (2022) which found 63.3% of patients with chronic liver disease had hyponatremia. Furthermore, Nareddy et al. (2020) reported that 87.9% of patients with hyponatremia fell within Child-Pugh Class C, indicating lower sodium levels strongly correlate with advanced liver dysfunction. Similarly, we noted severe hyponatremia to be more commonly associated with ascites, hepatic encephalopathy and hepatorenal syndrome and all were significantly more frequent in the hyponatremic cohort. These confirm that serum sodium is an important marker of prognosis in hepatic decompensation.

In our group, ascites was seen in 54.5% of hyponatremic patients versus 29.4% of normonatremic patients (p = 0.042), and hepatic encephalopathy was seen in 33.3% and 17.6%, respectively (p = 0.048). These findings align with Kumar et al. (2023), who found that low sodium levels were significantly associated with higher rates of portal hypertension, hepatic encephalopathy, and hepatorenal syndrome ($p<0.05$). Further, Godara et al. (2023) found that there was a significantly higher prevalence of hyponatremia with increasing grade of hepatic encephalopathy (30%-grade I to 81.25%, grade IV), supporting sodium imbalance could be responsible for worsening neurological manifestations in cirrhosis. Our findings clarify the findings of Abd El-Ghany et al. (2023) that the cutoff value ≤124 mmol/L sodium had an 83.33% sensitivity for predicting hepatic encephalopathy, indicative of predicting neurological complications in end-stage liver disease.

In the present study, there was an important inverse correlation of serum sodium with both MELD (r = –0.62, p < 0.001) and Child-Pugh scores (r = –0.55, p = 0.002), indicating that lower sodium concentrations were associated with worse liver dysfunction. This was similar to the findings from Hussain et al. (2022), who also demonstrated a negative association between serum sodium and the markers of severity of the disease. Singh and Chamoli (2022) similarly found patients with sodium ≤130 mEq/L had higher MELD and Child-Pugh scores, which supports our observations that hyponatremia was not an incidental finding, but rather a marker of disease progression. Additionally, Nareddy et al. (2020) demonstrated a strong association of hyponatremia with complications such as spontaneous bacterial peritonitis (OR 4.667, p = 0.004) and hepatorenal syndrome (OR 5.357, p = 0.034), further underscoring our observation that hepatorenal syndrome was significantly more common in hyponatremic patients compared to non-hyponatremic patients (18.2% compared to 5.9%, p = 0.03).

In our study, in the assessments of severity by Child-Pugh and MELD classifications, 44% were Group C and 40% had high MELD scores (≥20), which reflects advanced state of disease severity. This distribution supports a study report by Azam et al. in 2024 with decompensated cirrhotic had significant distorted lengths of present of disease and were mostly severe classification of hepatic dysfunction. Moreover, in our research, we also showed that the prevalence of hyponatremia increased with disease

severity in proportion, in agreement with report by Godara et al., in 2023 where 88% of patients were hyponatremic in Child-Pugh Class C versus only 25% in Class A. Therefore, when the consistency of responses across studies, including our research can continue to lend support to the evidence that serum sodium levels are inversely related to hepatic impairment severity.

The prognostic sludge of hyponatremia is reported previously. Jang and Jung (2018) emphasized that a low serum sodium level is not only a marker of advanced disease but also a predictive indicator of mortality as well as post-transplant outcomes in patients with cirrhosis. Similarly, the strong association between hyponatremia and higher MELD score in our study suggests that serum sodium may be an adjunctive prognostic variable for outcome prediction, supplementing conventional scoring systems. Of importance, the poor correlation between MELD and Child-Pugh scores in our present series ($r = 0.03$, $p = 0.78$) would attest to the fact that both tools reflect different aspects of hepatic dysfunction-biochemical versus clinical parameters-further validating the independent predictive role of serum sodium.

However, some studies found the prevalence of hyponatremia to be somewhat lower. For example, Hussain et al., (2022), saw that 50.6% of their patients had normal sodium levels, while only 34% of our patients did. The variability could be due to a difference in demographics and etiology since there was a higher proportion of alcohol-related cirrhosis in our cohort, which is generally associated with severe portal hypertension and disturbances in sodium holding. Furthermore, nutritional and health care factors may vary geographically and influence the biochemical profile at presentation.

These results, therefore, are in close agreement with literature and establish hyponatremia as a significant and independent correlation of cirrhosis severity. The high occurrence rate of hyponatremia in our sample size, the strong inverse correlation with MELD and Child-Pugh scores for liver status in Cirrhosis, and associations with the other further poor outcomes at the population-level (ascites, hepatic encephalopathy, hepatorenal syndrome) highlight the pathophysiology and prognostic significance of sodium imbalance in patients with liver cirrhosis overall. Indeed, as many comparative studies would demonstrate, hyponatremia is among the most reliable biochemical predictors of decompensation and poor outcomes in cirrhosis, and compelling evidence supports its use for normal use in clinical evaluation and prognostication.

Conclusion

The research showed that decreased serum sodium closely related to the progression of liver disease. Hyponatremia was a common manifestation in patients with advanced cirrhosis and had a close relationship with MELD and Child-Pugh scores, representing an increase in hepatic dysfunction and an increase in mortality. Patients with low sodium levels had an increased frequency of ascites, hepatic encephalopathy, and hepatorenal syndrome; therefore, hyponatremia is a relatively good clinical marker of severity of disease and systemic decompensation. While alcohol abuse and viral hepatitis were generally the accountable underlying etiology, it appeared that the degree of hyponatremia was more related to the degree of functional impairment of the liver, rather than the underlying cause. The inverse relationship of serum sodium and both severity indices underscores the prognostic value of serum sodium in estimates of severity and outcomes in cirrhosis. All in all, it appears that hyponatremia should not simply be viewed as a biochemical abnormality in cirrhosis, but rather as an important indicator of advanced disease, potential for more complications, and the need for more proactive clinical intervention in those with decompensated cirrhosis.

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