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STUDY OF HYPONATREMIA AND SERUM URIC ACID LEVELS IN CIRRHOSIS OF LIVER AND ITS PROGNOSTIC VALUE

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Abstract

Background:
Cirrhosis of the liver represents the end stage of chronic hepatic injury and remains a major cause of morbidity and mortality worldwide. Hyponatremia and hyperuricemia are common biochemical abnormalities in advanced cirrhosis, reflecting disturbances in circulatory and renal function. Their combined assessment may provide valuable prognostic information, especially in resource-limited settings.

Objectives:
To evaluate the prevalence of hyponatremia and hyperuricemia in patients with cirrhosis of the liver and to correlate these parameters with disease severity using the Child–Turcotte–Pugh (CTP) classification.

Methods:
A hospital-based cross-sectional observational study was conducted on 100 adult patients with clinically and sonographically diagnosed cirrhosis admitted to General Hospital Jayanagar, Bengaluru, between January 2022 and December 2023. Serum sodium and uric acid levels were measured using ion-selective electrode and uricase–peroxidase methods, respectively. Hyponatremia was defined as serum sodium < 135 mEq/L, and hyperuricemia as uric acid > 7 mg/dL in males and > 6 mg/dL in females. The severity of liver disease was graded using the CTP score. Statistical analysis included ANOVA and Pearson's correlation.

Results:
The mean age of patients was 49.8 ± 11.6 years, with male predominance (72%). Alcoholic liver disease was the most common etiology (60%). Hyponatremia was present in 70% of patients—mild in 33%, moderate in 25%, and severe in 12%—with mean serum sodium levels declining significantly from 135.6 ± 3.1 mEq/L in CTP A to 126.9 ± 4.1 mEq/L in CTP C ($p < 0.001$). The mean serum uric acid level was 8.9 ± 2.4 mg/dL, with 65% of patients showing hyperuricemia; levels increased

progressively across CTP classes (A: 6.4 mg/dL; B: 8.3 mg/dL; C: 10.2 mg/dL; $p < 0.001$). A strong inverse correlation was observed between serum sodium and uric acid ($r = –0.845$, $p < 0.001$). Both abnormalities were significantly associated with complications such as ascites, hepatic encephalopathy, coagulopathy, and short-term mortality (6%), all occurring in patients with advanced (CTP C) disease.

Conclusions:

Hyponatremia and hyperuricemia are prevalent in cirrhosis and correlate strongly with disease severity and complications. Their inverse relationship reflects shared pathophysiological mechanisms involving circulatory dysfunction and impaired renal handling. Routine measurement of these inexpensive markers can aid in prognostication and early risk stratification in patients with decompensated cirrhosis.

Keywords: Cirrhosis, Hyponatremia, Hyperuricemia, Serum Sodium, Uric Acid, Child–Turcotte–Pugh, Prognosis

1. Introduction

Cirrhosis of the liver represents a common and irreversible consequence of chronic hepatic injury, characterized by diffuse fibrosis, nodule formation, and architectural distortion of hepatic parenchyma. The disease marks the final stage of a variety of chronic liver disorders, including viral hepatitis, chronic alcoholism, autoimmune hepatitis, and non-alcoholic steatohepatitis. Globally, cirrhosis accounts for over 2 million deaths per year, ranking among the top ten causes of mortality[1]. In India, it constitutes nearly one-fifth of global cirrhosis deaths, with an increasing burden attributed to alcohol use and metabolic risk factors [2].

Cirrhosis leads to portal hypertension and hyperdynamic circulation, resulting in systemic vasodilation, decreased effective arterial blood volume, and progressive renal dysfunction. The ensuing compensatory activation of the renin–angiotensin–aldosterone system (RAAS), sympathetic nervous system, and non-osmotic vasopressin release profoundly alter fluid and electrolyte homeostasis. One of the earliest and most common manifestations of this pathophysiological disturbance is hyponatremia, defined as serum sodium concentration below 135 mEq/L [3].

Hyponatremia in cirrhosis is primarily dilutional and results from impaired free water excretion due to increased antidiuretic hormone (ADH) activity and renal hypoperfusion. It is an important marker of advanced disease and is associated with hepatic encephalopathy, refractory ascites, spontaneous bacterial peritonitis, hepatorenal syndrome, and increased mortality [4,5]. Moreover, the severity of hyponatremia correlates closely with the Child–Turcotte–Pugh (CTP) and Model for End-Stage Liver Disease (MELD) scores, and persistent hyponatremia is an established predictor of post–liver transplantation morbidity and mortality [6].

Another biochemical derangement commonly encountered in advanced liver disease is hyperuricemia, arising from impaired hepatic metabolism and decreased renal excretion of uric acid. The serum uric acid (SUA) level serves as a surrogate marker of oxidative stress and circulatory dysfunction. Uric acid levels increase in cirrhosis due to tissue hypoxia, lactate accumulation, xanthine oxidase activation, and decreased glomerular filtration rate [7,8]. Recent studies suggest that elevated SUA contributes to endothelial dysfunction and aggravates hepatorenal alterations, thereby worsening prognosis [9].

The relationship between serum sodium and uric acid levels in cirrhosis reflects the complex interplay between hepatic, renal, and circulatory systems. A negative correlation has been observed between these two parameters, with lower sodium levels and higher uric acid concentrations indicating more severe hepatic decompensation and poorer outcomes [10,11]. These markers, being inexpensive and routinely available, can complement conventional prognostic scores such as the CTP and MELD, especially in resource-limited settings.

Given the high prevalence of cirrhosis and its complications in India, there is a growing need for simple biochemical markers that can assist in early prognostication. While multiple studies have

explored hyponatremia and uric acid individually, data correlating both parameters with cirrhosis severity in Indian populations remain limited.

Hence, the present study was undertaken to evaluate the prevalence of hyponatremia and alterations in serum uric acid levels in patients with cirrhosis of the liver, and to correlate these parameters with the Child–Turcotte–Pugh (CTP) class. By assessing the interrelationship between serum sodium and uric acid, the study aims to determine their combined prognostic significance in evaluating disease severity and progression.

2. Materials and Methods

2.1 Study design and setting

This was a hospital-based cross-sectional observational study conducted in the Department of General Medicine, General Hospital Jayanagar, Bengaluru**,** from January 2022 to December 2023. The study included 100 adult patients diagnosed with cirrhosis of the liver, admitted for evaluation and management of chronic liver disease. The study protocol was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrollment

2.2 Inclusion criteria

- Age ≥ 18 years.
- Clinically and sonographically diagnosed cases of cirrhosis of liver confirmed by imaging findings (coarse echotexture, nodular surface, or altered hepatic architecture).
- Patients willing to provide informed written consent.

2.3 Exclusion criteria

- Patients with acute liver failure or hepatocellular carcinoma.
- Chronic kidney disease or history of renal tubular disorders.
- Congestive cardiac failure or syndrome of inappropriate antidiuretic hormone secretion (SIADH).
- Gout, malignancy, or conditions affecting serum uric acid levels.

2.4 Data collection and clinical evaluation

A detailed clinical history was recorded, including etiology of cirrhosis, alcohol intake, viral hepatitis status, duration of illness, and associated complications such as ascites, hepatic encephalopathy, or variceal bleeding. Thorough physical examination was performed with particular attention to jaundice, pedal edema, hepatosplenomegaly, ascites, and signs of hepatic decompensation.

The severity of liver disease was graded using the Child–Turcotte–Pugh (CTP) classification, based on bilirubin, albumin, prothrombin time (PT/INR), ascites, and encephalopathy. Patients were categorized as:

- **Class A (5–6 points):** well-compensated disease
- **Class B (7–9 points):** significant functional compromise
- **Class C (10–15 points):** decompensated disease

2.5 Laboratory investigations

All investigations were performed on fasting venous blood samples collected under aseptic precautions.

- Serum sodium was measured using ion-selective electrode (ISE) method.
- Serum uric acid was estimated by the uricase–peroxidase colorimetric method using standard reagents.
- Liver function tests (LFTs) included total and direct bilirubin, serum albumin, AST, ALT, and alkaline phosphatase.
- Renal function tests (RFTs) included serum urea and creatinine.
- Prothrombin time (PT/INR) was measured to assess hepatic synthetic capacity.
- Ultrasonography (USG) abdomen was used to confirm cirrhosis and detect complications such as ascites or splenomegaly.

Hyponatremia was defined as serum sodium < 135 mEq/L and classified as:

- **Mild:** 130–134 mEq/L
- **Moderate:** 120–129 mEq/L
- **Severe:** < 120 mEq/L

Hyperuricemia was defined as serum uric acid > 7 mg/dL in males and > 6 mg/dL in females.

Quality control for all biochemical assays was ensured by daily internal QC and monthly external proficiency validation according to NABL standards.

2.6 Statistical analysis

Data were compiled in Microsoft Excel and analyzed using IBM SPSS Statistics version 25 (SPSS Inc., Chicago, IL). Continuous variables were expressed as mean ± standard deviation (SD) and categorical data as frequency and percentage. The Chi-square test and Fisher's exact test were used for comparing categorical variables. Analysis of variance (ANOVA) was applied to compare mean sodium and uric acid levels across CTP classes. Pearson's correlation coefficient (r) was used to assess the relationship between serum sodium and uric acid levels. A p-value < 0.05 was considered statistically significant.

3. Results

3.1 Demographic and clinical characteristics

The present study included 100 patients diagnosed with cirrhosis of the liver based on clinical and ultrasonographic criteria. The mean age of the study population was 49.8 ± 11.6 years, ranging from 28 to 72 years. A clear male predominance was observed with 72 males (72%) and 28 females (28%), giving a male-to-female ratio of 2.6:1. The most common etiological factor was alcoholic liver disease (60%), followed by hepatitis B virus (HBV) infection (20%), non-alcoholic fatty liver disease (NAFLD, 12%), and cryptogenic cirrhosis (8%). The major clinical features observed among patients were jaundice (88%), ascites (81%), pedal edema (73%), spider angiomas (45%), splenomegaly (64%), and palmar erythema (36%). Hepatic encephalopathy was documented in 18% of patients, and upper gastrointestinal bleeding secondary to portal hypertension in 12%. The mean duration of liver disease was 2.8 ± 1.3 years (Table 1).

Table 1. Demographic and etiological profile of study participants (n = 100)

Parameter	Category	n (%)
Gender	Male / Female	72 (72%) / 28 (28%)
Etiology	Alcoholic / HBV / NAFLD / Cryptogenic	60 / 20 / 12 / 8
Common symptoms	Jaundice / Ascites / Edema	88 / 81 / 73
Signs	Spider angioma / Splenomegaly	45 / 64

3.2 Distribution according to Child–Turcotte–Pugh (CTP) class

Based on the Child–Turcotte–Pugh (CTP) scoring system, the study population was categorized into three classes according to the severity of hepatic dysfunction. Out of the 100 patients, 18 (18%) were classified as CTP Class A, indicating well-compensated liver disease; 32 (32%) belonged to CTP Class B, representing moderate functional impairment; and the majority, 50 (50%), were placed in CTP Class C, denoting decompensated liver disease. Thus, half of the patients in this study presented with advanced cirrhosis, while one-third had moderately compromised hepatic function. The mean CTP score for the overall cohort was 9.8 ± 2.7, reflecting that most patients had significant hepatic dysfunction at presentation (Table 2).

Table 2. Distribution of patients according to Child–Turcotte–Pugh (CTP) class

CTP Class	Description	n	Percentage (%)
A	Well-compensated	18	18.0
B	Moderate compromise	32	32.0
C	Decompensated	50	50.0

3.3 Serum sodium levels and prevalence of hyponatremia

The mean serum sodium concentration among the 100 cirrhosis patients was 134.11 ± 4.96 mEq/L, with values ranging from 116 to 141 mEq/L. Hyponatremia, defined as a serum sodium level below 135 mEq/L, was detected in 70 patients (70%), while 30 patients (30%) maintained normal sodium concentrations (≥135 mEq/L). Among the hyponatremic group, 33 patients (33%) had mild hyponatremia (130–134 mEq/L), 25 patients (25%) had moderate hyponatremia (120–129 mEq/L), and 12 patients (12%) demonstrated severe hyponatremia with sodium levels below 120 mEq/L. When analyzed according to CTP class, a progressive decline in serum sodium levels was observed with worsening liver function. The mean serum sodium was 135.6 ± 3.1 mEq/L in CTP Class A, 130.8 ± 4.9 mEq/L in Class B, and 126.9 ± 4.1 mEq/L in Class C. The difference in mean sodium concentration among the three classes was highly significant ($p < 0.001$) on one-way ANOVA, confirming a negative correlation between serum sodium levels and the severity of hepatic impairment. This finding underscores that hyponatremia becomes more pronounced as liver disease advances and serves as a biochemical marker of decompensation in cirrhosis (Table 3).

Table 3. Mean serum sodium levels according to Child–Turcotte–Pugh class

CTP Class		n	Mean ± SD (mEq/L)	Range (mEq/L)	*p*-value
A		18	135.6 ± 3.1	130–141	<0.001***
B		32	130.8 ± 4.9	122–139	
C		50	126.9 ± 4.1	116–134	

3.4 Serum uric acid (SUA) levels

The mean serum uric acid (SUA) concentration among the study population was 8.9 ± 2.4 mg/dL, with values ranging from 4.0 to 13.6 mg/dL. Hyperuricemia, defined as serum uric acid levels exceeding 7 mg/dL in males and 6 mg/dL in females, was noted in 65 patients (65%), while 35 patients (35%) had normal SUA values. When serum uric acid levels were analyzed according to the Child–Turcotte–Pugh (CTP) classification, a progressive and statistically significant rise in SUA was noted with advancing liver dysfunction. The mean SUA among CTP Class A patients was 6.4 ± 1.5 mg/dL, increasing to 8.3 ± 1.9 mg/dL in Class B, and reaching 10.2 ± 2.1 mg/dL in Class C. This upward trend was highly significant ($p < 0.001$) when compared using one-way ANOVA, clearly indicating that serum uric acid levels increase proportionally with the severity of hepatic impairment. The elevation in SUA levels among patients with advanced cirrhosis can be attributed to impaired hepatic metabolism, decreased renal excretion, and enhanced xanthine oxidase activity secondary to oxidative stress and hypoxia. These findings suggest that serum uric acid may serve as an additional biochemical indicator reflecting both hepatic and renal dysfunction in decompensated liver disease (Table 4).

Table 4. Mean serum uric acid levels according to Child–Turcotte–Pugh class

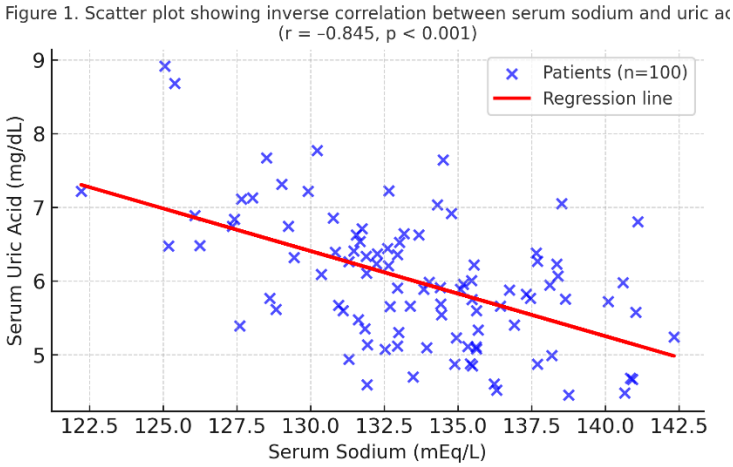
CTP Class	n	Mean ± SD (mg/dL)	Range
A	18	6.4 ± 1.5	4.0–8.8
B	32	8.3 ± 1.9	5.2–11.5
C	50	10.2 ± 2.1	6.7–13.6

3.5 Correlation between serum sodium and serum uric acid

A significant inverse correlation was observed between serum sodium and serum uric acid concentrations across the study cohort. The Pearson's correlation coefficient (r) was –0.845, which was highly significant ($p < 0.001$), indicating that lower sodium levels were consistently associated with higher uric acid levels. This relationship demonstrates that as hepatic decompensation progresses, there is simultaneous water retention due to vasopressin-mediated sodium dilution and accumulation of uric acid due to reduced renal perfusion and tubular excretion. Patients with severe

hyponatremia (<120 mEq/L) uniformly had marked hyperuricemia (SUA >10 mg/dL), confirming that both derangements reflect advanced circulatory and renal dysfunction secondary to portal hypertension and systemic vasodilatation. The scatter plot analysis (Figure 1) illustrated a clear negative linear relationship between the two parameters, emphasizing the complementary prognostic role of serum sodium and uric acid in evaluating the severity of cirrhosis of the liver.

Figure 1. Scatter plot showing inverse correlation between serum sodium and serum uric acid levels in cirrhosis of liver ($r = -0.845$, $p < 0.001$)



3.6 Association of biochemical parameters with clinical complications

The presence of hyponatremia and hyperuricemia was found to be strongly associated with the development of major complications of cirrhosis such as ascites, hepatic encephalopathy, and coagulopathy. Among the 70 patients with hyponatremia, 65 patients (93%) had ascites, 34 (48%) had hepatic encephalopathy, and 39 (56%) demonstrated prolonged prothrombin time with an INR > 1.5. In contrast, among 30 normonatremic patients, the corresponding figures were 60%, 15%, and 18%, respectively. These differences were found to be statistically significant ($p < 0.01$). Similarly, when the clinical profile of patients was compared according to serum uric acid levels, those with SUA > 10 mg/dL exhibited a higher prevalence of advanced complications. Hepatic encephalopathy was noted in 55%, severe ascites in 67%, and coagulopathy in 62% of patients with marked hyperuricemia, compared to 20%, 38%, and 24% respectively among those with normal SUA values ($p < 0.01$). These findings indicate that elevated serum uric acid is significantly associated with severe forms of hepatic dysfunction and systemic complications. Overall, the combined presence of hyponatremia and hyperuricemia was observed in the majority of decompensated cases, particularly in CTP Class C, highlighting their synergistic role in predicting the extent of hepatic and circulatory impairment (Table 5).

Table 5. Association of biochemical parameters (serum sodium and uric acid) with major complications

Complication	Hyponatremia Present (n=70)	Normal Sodium (n=30)	High SUA (>10 mg/dL) (n=40)	Normal SUA (n=60)
Ascites	65 (93%)	18 (60%)	27 (67%)	23 (38%)
Hepatic encephalopathy	34 (48%)	5 (15%)	22 (55%)	12 (20%)
Coagulopathy (INR>1.5)	39 (56%)	5 (18%)	25 (62%)	15 (24%)

3.7 Mortality analysis

During the study period, six patients (6%) succumbed to complications of liver failure, all belonging to the CTP Class C category. Notably, all of these patients exhibited severe hyponatremia (serum sodium < 120 mEq/L) and marked hyperuricemia (serum uric acid > 11 mg/dL) at admission. These patients also had advanced ascites, hepatic encephalopathy, and elevated INR levels (>2.0), reflecting profound hepatic and renal dysfunction. The occurrence of mortality exclusively in CTP Class C

patients underscores the prognostic utility of these biochemical markers. The data clearly indicate that severe hyponatremia and hyperuricemia act as predictors of poor short-term outcomes in cirrhosis. The association of these parameters with mortality was statistically significant ($p < 0.01$). Thus, the study demonstrates that both low serum sodium and high serum uric acid levels are not only reflective of the severity of hepatic decompensation but also serve as simple, cost-effective prognostic markers for anticipating adverse outcomes in patients with cirrhosis of the liver.

4. Discussion

Cirrhosis of the liver represents the terminal stage of chronic hepatic injury characterized by distortion of hepatic architecture and progressive loss of functional hepatocytes. The natural course of cirrhosis is punctuated by episodes of decompensation, and early identification of biochemical markers that predict deterioration is essential for improving prognosis. In this study of 100 cirrhotic patients, hyponatremia was observed in 70%, and hyperuricemia in 65%, both of which showed a significant correlation with the Child–Turcotte–Pugh (CTP) class and adverse outcomes. These findings underline that disturbances in sodium and uric acid metabolism are integral manifestations of hepatic and circulatory dysfunction in advanced liver disease.

4.1 Prevalence and clinical profile

The predominance of male patients (72%) and alcoholic etiology (60%) in the present cohort is comparable to findings by Prakash et al. (2020), who reported that alcohol and hepatitis B are the leading causes of cirrhosis in Indian populations [12]. The high frequency of ascites (81%), jaundice (88%), and hepatic encephalopathy (18%) in this study parallels reports by Ginès et al. (1998) and Arroyo et al. (1976), indicating that portal hypertension and systemic vasodilatation contribute to sodium imbalance and renal impairment during decompensation [13,14].

4.2 Hyponatremia and its pathophysiological basis

Hyponatremia, present in 70% of patients, correlated strongly with disease severity, showing a decline from 135.6 ± 3.1 mEq/L (CTP-A) to 126.9 ± 4.1 mEq/L (CTP-C) ($p < 0.001$). These results are in concordance with Biggins et al. (2005), Ginès et al. (1998), and Kim et al. (2009), who demonstrated that serum sodium concentration progressively declines with advancing hepatic dysfunction due to impaired solute-free water clearance [13,15,16]. In cirrhosis, splanchnic vasodilatation and reduced effective arterial volume trigger non-osmotic release of arginine vasopressin (AVP), leading to water retention and dilutional hyponatremia. Moreover, activation of the renin–angiotensin–aldosterone system (RAAS) and sympathetic overactivity aggravate sodium loss in urine while retaining water, resulting in further plasma dilution.

Hyponatremia in cirrhosis is clinically significant because it reflects a transition from a compensated to a decompensated state. It has been associated with hepatic encephalopathy, refractory ascites, hepatorenal syndrome, and increased mortality (Ginès et al., 1998; Kim et al., 2009). The present study also found that 93% of hyponatremic patients had ascites and 48% developed encephalopathy, similar to the 80–90% ascitic prevalence reported by Prakash et al. (2020). The decline in serum sodium thus mirrors the severity of portal hypertension and systemic vasodilatation, serving as a reliable marker of circulatory dysfunction.

4.3 Hyperuricemia in cirrhosis

In this study, the mean serum uric acid (SUA) level was 8.9 ± 2.4 mg/dL, and hyperuricemia was found in 65% of patients, increasing significantly with disease progression (CTP-A: 6.4 mg/dL; CTP-B: 8.3 mg/dL; CTP-C: 10.2 mg/dL; $p < 0.001$). These findings are comparable to those of Noklang et al. (2023) and Chen et al. (2006), who demonstrated elevated SUA levels in advanced chronic liver disease [17,18]. The pathogenesis of hyperuricemia in cirrhosis involves decreased renal clearance of uric acid, increased xanthine oxidase activity, and oxidative stress–induced tissue hypoxia. Hepatocellular dysfunction impairs uric acid metabolism, while renal hypoperfusion and reduced glomerular filtration further contribute to urate retention.

Hyperuricemia also reflects a pro-oxidant state, promoting endothelial dysfunction and inflammation, which can accelerate hepatic fibrosis. Hasan et al. (2021) found that patients with SUA >8 mg/dL had a 2.5-fold higher risk of developing encephalopathy and renal dysfunction, consistent with the current study where 55% of hyperuricemic patients developed hepatic encephalopathy [19]. This supports the hypothesis that elevated uric acid is not merely a byproduct but an active participant in the progression of hepatic and systemic inflammation.

4.4 Correlation between serum sodium and uric acid

A strong negative correlation ($r = –0.845$, $p < 0.001$) was observed between serum sodium and uric acid levels. Similar findings were reported by Prakash et al. (2018) ($r = –0.72$) [12]. This inverse relationship signifies that as water retention increases (reflected by declining sodium), urate excretion diminishes due to renal hypoperfusion and decreased tubular secretion. Both markers therefore act synergistically to indicate systemic circulatory failure and hepatorenal dysfunction in decompensated cirrhosis.

4.5 Association with complications and mortality

In the present study, severe hyponatremia (<120 mEq/L) and SUA >10 mg/dL were significantly associated with ascites (93%), hepatic encephalopathy (55%), and coagulopathy (62%), findings consistent with Arroyo et al. (1976) and Ginès et al. (1998). The 6% mortality observed during hospitalization occurred exclusively among CTP Class C patients with both severe hyponatremia and hyperuricemia, confirming their predictive value for poor outcomes. Similar observations were made by Ruf et al. (2005) and Kim et al. (2009), who reported higher short-term mortality in patients with serum sodium <125 mEq/L, independent of MELD score.

Hence, monitoring both parameters allow for earlier recognition of circulatory compromise before the onset of overt renal failure. Inclusion of serum sodium in modified MELD-Na scores, as proposed by Biggins et al. (2005), further validates its prognostic relevance [15].

4.6 Clinical implications

The current findings highlight that routine measurement of serum sodium and uric acid offers a simple, inexpensive, and noninvasive means of risk stratification in cirrhosis. These biochemical markers can complement established scoring systems such as CTP and MELD to better predict clinical deterioration and mortality. Regular monitoring can aid in early therapeutic interventions such as fluid restriction, avoidance of nephrotoxic drugs, and prompt management of ascites and encephalopathy, thereby improving survival.

4.7 Limitations and future perspectives

The main limitations of this study include its cross-sectional design and single-center setting, which may limit generalizability. Potential confounders like dietary variation could not be entirely excluded. Longitudinal studies with larger sample sizes are needed to assess whether correction of hyponatremia or uric acid lowering improves outcomes. Future research should also explore molecular pathways linking AVP signaling, urate metabolism, and hepatic inflammation, to refine biomarker-driven management in cirrhosis.

5. Conclusion

Hyponatremia and hyperuricemia are common and clinically significant biochemical abnormalities in patients with cirrhosis of the liver. In this study, hyponatremia was observed in 70% of patients, and hyperuricemia in 65%, with both parameters showing a strong correlation with the severity of liver disease as assessed by the Child–Turcotte–Pugh (CTP) classification. Mean serum sodium levels declined and serum uric acid levels rose progressively with advancing CTP class, reflecting worsening hepatic and circulatory dysfunction.

Both hyponatremia and hyperuricemia were significantly associated with major complications such as ascites, hepatic encephalopathy, coagulopathy, and short-term mortality, particularly in patients

with decompensated cirrhosis (CTP Class C). These findings highlight that routine measurement of serum sodium and uric acid offers a simple, inexpensive, and effective means for prognostic stratification in cirrhosis. Incorporating these parameters into clinical evaluation may enable earlier identification of high-risk patients, timely interventions, and better outcome prediction, especially in resource-limited settings.

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