Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/8pdz2282
Original Article

Clinical profile of hyponatremia in patients with decompensated chronic liver disease: A cross-sectional study.

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Abstract

Background

Hyponatremia is a common electrolyte abnormality in patients with decompensated chronic liver disease and is associated with severe complications and poor prognosis. This study evaluated the clinical profile and severity of hyponatremia among hospitalized patients with decompensated chronic liver disease.

Methodology

A hospital-based cross-sectional study was conducted in the Department of Medicine, Pt. J.N.M. Medical College and Dr. B.R.A.M. Hospital, Raipur, over a period of one year. A total of 198 admitted patients with decompensated chronic liver disease were included. Clinical history, physical examination findings, laboratory investigations, and serum sodium levels were recorded. Statistical analysis was performed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation, whereas categorical variables were represented as frequencies and percentages. Chi-square test and independent t-test were used for analysis. A p-value <0.05 was considered statistically significant.

Results

Among 198 patients, males constituted 74.2% and females 25.8%. The mean age was 51.8±12.6 years. Hyponatremia (<135 mEq/L) was observed in 139 patients (70.2%). Severe hyponatremia (<125 mEq/L) was present in 17.2% of cases. Ascites (86.4%), jaundice (79.3%), pedal edema (65.7%), hepatic encephalopathy (36.4%), and gastrointestinal bleeding (24.7%) were the common manifestations. Severe hyponatremia was significantly associated with a higher frequency of hepatic encephalopathy (p=0.003), spontaneous bacterial peritonitis (p=0.021), and prolonged hospitalization (p=0.001).

Conclusion

Hyponatremia is highly prevalent among patients with decompensated chronic liver disease and correlates with disease severity and complications. Early identification and correction of sodium imbalance may improve clinical outcomes.

Recommendation

Routine assessment and monitoring of serum sodium should be incorporated into the standard management protocol of decompensated chronic liver disease patients.

Keywords: *Hyponatremia, Decompensated Chronic Liver Disease, Ascites, Hepatic Encephalopathy, Serum Sodium, Cirrhosis.*
Submitted: *April 20, 2026* ***Accepted:*** *May 20, 2026* ***Published:*** *June 30, 2026*

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Background of the study

Chronic liver disease (CLD) is a major cause of morbidity and mortality worldwide and represents a significant public health burden. Cirrhosis, which is the end stage of chronic liver injury, develops secondary to viral hepatitis, alcohol-related liver disease, non-alcoholic fatty liver disease, autoimmune disorders, and metabolic diseases (1,2). Decompensated chronic liver disease is characterized by complications such as ascites, jaundice, variceal bleeding, spontaneous bacterial peritonitis, hepatorenal syndrome, and hepatic encephalopathy, which are associated with poor prognosis and reduced survival (3).

Hyponatremia is one of the most common electrolyte abnormalities encountered in patients with advanced liver disease and has emerged as an important predictor of disease severity and mortality (4). The development of hyponatremia in cirrhosis is mainly dilutional and results from impaired renal free water excretion caused by portal hypertension and marked splanchnic vasodilatation (5). This reduction in effective arterial blood volume activates the renin-angiotensin-aldosterone system and stimulates non-osmotic release of arginine vasopressin, ultimately leading to water retention and reduced serum sodium concentration (6).

Several studies have reported that hyponatremia occurs in approximately 40–60% of patients with cirrhosis, with a higher prevalence among hospitalized patients with decompensated disease (7). Serum sodium concentration has gained increasing importance as a prognostic marker and has been incorporated into the MELD-Na score used for liver transplantation prioritization (8).

Hyponatremia has been associated with refractory ascites, spontaneous bacterial peritonitis, hepatic encephalopathy, hepatorenal syndrome, prolonged hospitalization, and increased mortality (9). Furthermore, reduced serum sodium levels contribute to cerebral edema and aggravate neurological dysfunction, thereby increasing the risk of hepatic encephalopathy (10).

Although numerous international studies have highlighted the prognostic significance of hyponatremia in cirrhosis, data from India are relatively limited. Variations in etiological factors, nutritional status, and healthcare accessibility necessitate region-specific studies to better understand the clinical profile and burden of hyponatremia among Indian patients with decompensated chronic liver disease (11). Early recognition and timely management of sodium imbalance may help reduce complications and improve clinical outcomes in these patients (12).

Therefore, the present study was undertaken to evaluate the clinical profile of hyponatremia in patients with decompensated chronic liver disease admitted to Pt. J.N.M. Medical College and Dr. B.R.A.M. Hospital, Raipur, Chhattisgarh.

Methodology

Study design

This hospital-based cross-sectional observational study was conducted to evaluate the clinical profile of hyponatremia among patients with decompensated chronic liver disease (DCLD) admitted to a tertiary care teaching hospital.

Study setting

The study was conducted in the Department of Medicine, Pt. J.N.M. Medical College and Dr. B.R.A.M. Hospital, Raipur, Chhattisgarh, India. The institution is a government-funded tertiary care teaching hospital that provides comprehensive inpatient and outpatient medical services, emergency and critical care, intensive care services, diagnostic laboratory facilities, radiological investigations, and specialty care. It serves as a major referral center for Raipur city and the surrounding districts of Chhattisgarh, catering to both urban and rural populations.

Study duration

The study was conducted over one year, from 1 January 2025 to 31 December 2025.

Study population

The study population comprised adult patients admitted with decompensated chronic liver disease to the Department of Medicine during the study period.

Sample size

A total of **198 patients** diagnosed with decompensated chronic liver disease who satisfied the eligibility criteria were included in the study.

Sampling technique

A **consecutive sampling** technique was employed. All eligible patients admitted during the study period who fulfilled the inclusion criteria and provided written informed consent were enrolled consecutively until the required sample size of 198 participants was achieved.

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Eligibility criteria

Inclusion criteria

Participants were included if they:
were aged 18 years or older;
provided written informed consent to participate in the study;
had a confirmed diagnosis of decompensated chronic liver disease; and
presented with one or more features of hepatic decompensation, including ascites, jaundice, hepatic encephalopathy, variceal bleeding, spontaneous bacterial peritonitis, or hepatorenal syndrome.

Exclusion criteria

Patients were excluded if they had:
chronic kidney disease;
congestive heart failure;
nephrotic syndrome;
malignancy;
pregnancy;
were receiving dialysis;
acute gastroenteritis associated with electrolyte disturbances;
received hypertonic saline before hospital admission; or
declined to participate in the study.

Data collection procedure

After obtaining written informed consent, demographic characteristics, medical history, clinical findings, and laboratory investigations were recorded using a structured and pretested data collection proforma. A detailed physical examination was performed for each participant. Serum sodium concentration was measured at admission, and additional laboratory and radiological investigations were performed according to standard hospital protocols.

Laboratory investigations

The following investigations were performed:
Hematological investigations
Complete blood count
Hemoglobin
Total leukocyte count
Platelet count
Biochemical investigations
Blood urea
Serum creatinine
Serum sodium
Serum potassium
Serum bilirubin
Serum albumin
Aspartate aminotransferase (AST)
Alanine aminotransferase (ALT)
Alkaline phosphatase
Prothrombin time
International normalized ratio (INR)
Radiological investigation
Ultrasonography of the abdomen and pelvis
Additional investigations (when clinically indicated)
Ascitic fluid examination
Upper gastrointestinal endoscopy

Operational definitions

Hyponatremia was defined as a **serum sodium concentration below 135 mEq/L**.
Based on serum sodium concentration, participants were categorized as follows:
Normal: ≥135 mEq/L
Mild hyponatremia: 130–134 mEq/L
Moderate hyponatremia: 125–129 mEq/L
Severe hyponatremia: <125 mEq/L
Decompensated chronic liver disease was defined as cirrhosis associated with one or more complications, including ascites, jaundice, hepatic encephalopathy, variceal bleeding, spontaneous bacterial peritonitis, or hepatorenal syndrome.

Outcome measures

Primary outcome

Prevalence and severity of hyponatremia among patients with decompensated chronic liver disease.

Secondary outcomes

Demographic profile of patients.
Clinical manifestations associated with hyponatremia.
Association between serum sodium categories and complications of chronic liver disease.
Relationship between serum sodium concentration and duration of hospital stay.

Quantitative variables

Continuous variables, including age, serum sodium concentration, and duration of hospital stay, were analyzed as quantitative variables and expressed as **mean ± standard deviation (SD)**. For comparative analysis, serum sodium concentration was categorized into normal, mild, moderate, and severe hyponatremia according to established clinical

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guidelines. These categories were used to evaluate the relationship between the severity of hyponatremia and clinical complications.

Measures to Minimize Bias

Selection bias was minimized by consecutively enrolling all eligible patients during the study period. Standardized definitions and uniform diagnostic criteria were used throughout the study. Data were collected using a structured proforma, and laboratory investigations were performed according to standard institutional protocols to ensure consistency and reduce measurement bias.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using **IBM Statistical Package for the Social Sciences (SPSS) version 26.0** (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as **mean ± standard deviation**, while categorical variables were presented as **frequencies and percentages**.

Comparisons between categorical variables were performed using the **Chi-square test**, while **Fisher's exact test** was applied when expected cell frequencies were less than five. Continuous variables were compared using the **independent Student's t-test**. A **one-way analysis of variance (ANOVA)** was used to compare the mean duration of hospital stay across different serum sodium categories. A two-tailed **p-value <0.05** was considered statistically significant.

Ethical considerations

Ethical approval was obtained from the **Institutional Ethics Committee of Pt. J.N.M. Medical College, Raipur, Chhattisgarh**. Written informed consent was obtained from all participants before enrollment. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Results

A total of 198 patients with decompensated chronic liver disease were included in the present study. The mean age of the study population was **51.8 ± 12.6 years** (range: 21–78 years). Hyponatremia (serum sodium <135 mEq/L) was observed in **139 patients (70.2%)**, whereas 59 patients (29.8%) had normal serum sodium levels.

Demographic characteristics

Table 1 shows the age distribution of study participants. The majority of patients belonged to the 51–60 years age group (31.3%), followed by 41–50 years (25.8%). **Table 1** presents the age distribution of the 198 study participants. The largest proportion of patients belonged to the **51–60 years** age group (31.3%), followed by the **41–50 years** age group (25.8%). Patients aged ≤30 years and >70 years each accounted for only **5.6%** of the study population, indicating that decompensated chronic liver disease predominantly affected middle-aged adults.

Table 1: Age Distribution of Study Participants (n=198)

Age Group (Years)	Frequency	Percentage (%)
≤30	11	5.6
31–40	28	14.1
41–50	51	25.8
51–60	62	31.3
61–70	35	17.7
>70	11	5.6
Total	**198**	**100**

Gender distribution

Table 2 depicts gender distribution. Males constituted 74.2% of the study population. **Table 2** presents the gender distribution of the study participants. Males constituted **147 (74.2%)** of the patients, whereas females accounted for **51 (25.8%)**, demonstrating a marked male predominance among patients with decompensated chronic liver disease.

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Table 2: Gender Distribution (n=198)

Gender	Frequency	Percentage (%)
Male	147	74.2
Female	51	25.8
Total	**198**	**100**

Distribution According to Serum Sodium Levels

Hyponatremia was present in 139 (70.2%) patients. Severe hyponatremia (<125 mEq/L) was found in 34 patients (17.2%). **Table 3** summarizes the severity of hyponatremia among the study participants. Hyponatremia (serum sodium <135 mEq/L) was present in **139 (70.2%)** patients. Mild hyponatremia was the most frequent category (33.8%), followed by moderate (19.2%) and severe hyponatremia (17.2%). Normal serum sodium levels were observed in **59 (29.8%)** patients.

Table 3: Severity of Hyponatremia (n=198)

Serum Sodium Level	Frequency	Percentage (%)
Normal (≥135 mEq/L)	59	29.8
Mild (130–134 mEq/L)	67	33.8
Moderate (125–129 mEq/L)	38	19.2
Severe (<125 mEq/L)	34	17.2
Total	**198**	**100**

Clinical manifestations

Table 4 demonstrates the frequency of clinical manifestations. Ascites was the most common presentation (86.4%), followed by jaundice (79.3%) and pedal edema (65.7%). **Table 4** shows the frequency of clinical manifestations among patients with decompensated chronic liver disease. Ascites was the most common clinical feature (86.4%), followed by jaundice (79.3%), abdominal distension (75.3%), and pedal edema (65.7%). Hepatic encephalopathy occurred in 36.4% of patients, while spontaneous bacterial peritonitis was documented in 16.2%.

Table 4: clinical manifestations among study participants

Clinical Feature	Frequency	Percentage (%)
Ascites	171	86.4
Jaundice	157	79.3
Pedal edema	130	65.7
Abdominal distension	149	75.3
Hepatic encephalopathy	72	36.4
Gastrointestinal bleeding	49	24.7
Fever	41	20.7
Spontaneous bacterial peritonitis	32	16.2

Etiology of Chronic Liver Disease

Alcohol-related liver disease was the commonest etiology, accounting for 47.5% of cases. **Table 5** presents the

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etiological distribution of chronic liver disease. Alcohol-related liver disease was the leading cause, accounting for **47.5%** of cases, followed by hepatitis B (18.2%), hepatitis C (14.1%), and non-alcoholic fatty liver disease (11.1%).

Table 5: Etiology of Chronic Liver Disease

Etiology	Frequency	Percentage (%)
Alcoholic liver disease	94	47.5
Hepatitis B	36	18.2
Hepatitis C	28	14.1
NAFLD	22	11.1
Autoimmune liver disease	8	4.0
Others	10	5.1
Total	**198**	**100**

Association Between Severity of Hyponatremia and Hepatic Encephalopathy

As shown in **Table 6**, severe hyponatremia was significantly associated with hepatic encephalopathy (**χ^2=13.24, p=0.003**). **Table 6** presents the association between serum sodium categories and hepatic encephalopathy. Hepatic encephalopathy was observed in **11 of 59 (18.6%)** patients with normal serum sodium levels, **18 of 67 (26.9%)** patients with mild hyponatremia, **17 of 38 (44.7%)** patients with moderate hyponatremia, and **26 of 34 (76.5%)** patients with severe hyponatremia. The frequency of hepatic encephalopathy increased progressively with worsening hyponatremia, and the association was statistically significant (χ^2 = 13.24, p = 0.003).

Table 6: Association Between Hyponatremia and Hepatic Encephalopathy

Serum Sodium Level	Hepatic Encephalopathy Present	Absent	Total
Normal	11	48	59
Mild	18	49	67
Moderate	17	21	38
Severe	26	8	34
Total	**72**	**126**	**198**

Chi-square = 13.24, p = 0.003 (Statistically Significant)

Association Between Hyponatremia and Spontaneous Bacterial Peritonitis

Patients with severe hyponatremia showed a significantly higher incidence of spontaneous bacterial peritonitis (**χ^2=9.62, p=0.021**). **Table 7** shows the association between serum sodium levels and spontaneous bacterial peritonitis. Spontaneous bacterial peritonitis occurred in **5 of 59 (8.5%)** patients with normal serum sodium, **8 of 67 (11.9%)** with mild hyponatremia, **7 of 38 (18.4%)** with moderate hyponatremia, and **12 of 34 (35.3%)** with severe hyponatremia. The incidence increased with increasing severity of hyponatremia, and this association was statistically significant (χ^2 = 9.62, p = 0.021).

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Table 7: Association Between Hyponatremia and SBP

Serum Sodium Level	SBP Present	SBP Absent	Total
Normal	5	54	59
Mild	8	59	67
Moderate	7	31	38
Severe	12	22	34
Total	**32**	**166**	**198**

Chi-square = 9.62, p = 0.021 (Statistically Significant)

Duration of Hospital Stay

The mean duration of hospitalization was **8.4 ± 3.2 days**. Patients with severe hyponatremia had significantly longer hospital stay (**11.3 ± 3.8 days**) compared with patients having normal serum sodium (**6.2 ± 2.1 days**) (**t = 7.86, p = 0.001**). **Table 8** compares the duration of hospital stay according to serum sodium levels. The mean hospital stay increased progressively with worsening hyponatremia, from **6.2 ± 2.1 days** in patients with normal serum sodium to **11.3 ± 3.8 days** in those with severe hyponatremia. The difference among the four groups was statistically significant (ANOVA F = 16.74, p = 0.001), indicating that severe hyponatremia was associated with prolonged hospitalization.

Table 8: Mean Hospital Stay According to Serum Sodium Level

Serum Sodium Level	Mean Hospital Stay (Days)	SD
Normal	6.2	2.1
Mild	7.4	2.8
Moderate	9.6	3.1
Severe	11.3	3.8

ANOVA F-value = 16.74, p = 0.001 (Highly Significant)

The present study demonstrated a high prevalence of hyponatremia among patients with decompensated chronic liver disease. Ascites and jaundice were the commonest clinical manifestations. Severe hyponatremia was significantly associated with hepatic encephalopathy, spontaneous bacterial peritonitis, and prolonged hospitalization, suggesting that serum sodium concentration serves as an important indicator of disease severity and adverse clinical outcomes.

Discussion

The present hospital-based cross-sectional study evaluated the clinical profile of hyponatremia in **198 patients** with decompensated chronic liver disease. The principal finding was that **70.2%** of patients had hyponatremia, indicating that sodium imbalance is highly prevalent in patients with advanced liver disease. In addition, severe hyponatremia was significantly associated with hepatic encephalopathy (χ^2 = 13.24, p = 0.003)**,** spontaneous bacterial peritonitis (χ^2 = 9.62, p = 0.021)**, and** prolonged hospital stay (F = 16.74, p = 0.001)**,** suggesting that worsening hyponatremia is associated with greater disease severity and poorer clinical outcomes (13).

The high prevalence of hyponatremia observed in the present study can be explained by the pathophysiological changes associated with decompensated cirrhosis. Progressive portal hypertension and splanchnic vasodilatation reduce the effective arterial blood volume, resulting in activation of the renin-angiotensin-aldosterone system and non-osmotic release of arginine vasopressin. Consequently, excessive water retention leads to dilutional hyponatremia. Similar observations have been reported by Ginès et al. and Bernardi et al., who identified hyponatremia as one of the most frequent electrolyte abnormalities in advanced cirrhosis (14).

The majority of participants were middle-aged adults (mean age **51.8 ± 12.6 years**), and males accounted for **74.2%** of the study population. This demographic profile is

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comparable with previous studies by Borroni et al. and Moreau et al. The predominance of male patients is likely attributable to the higher prevalence of alcohol-related liver disease among men in many populations (15-17).

Alcohol-related liver disease was the leading etiology, accounting for **47.5%** of cases. This finding is consistent with previous reports demonstrating alcohol as one of the most common causes of cirrhosis worldwide. The high proportion observed in the present study may reflect regional differences in alcohol consumption patterns and the referral nature of the study center.

Ascites (**86.4%**) and jaundice (**79.3%**) were the most frequent clinical manifestations. These findings are consistent with studies by Runyon and D'Amico et al., which reported that portal hypertension, sodium retention, and impaired hepatic synthetic function contribute to fluid accumulation and jaundice in patients with decompensated cirrhosis (18-21).

A significant association was observed between severe hyponatremia and hepatic encephalopathy. The frequency of hepatic encephalopathy increased progressively from **18.6%** among patients with normal serum sodium to **76.5%** among those with severe hyponatremia. This finding supports previous reports by Córdoba et al. and Guevara et al., who demonstrated that hyponatremia contributes to cerebral edema and increases susceptibility to hepatic encephalopathy. Reduced serum sodium lowers plasma osmolality, facilitating astrocyte swelling and worsening neurological dysfunction.

Similarly, spontaneous bacterial peritonitis occurred more frequently in patients with severe hyponatremia (**35.3%**) than in those with normal serum sodium (**8.5%**). Comparable findings have been reported by Ginès and Cárdenas, suggesting that severe hyponatremia reflects advanced circulatory dysfunction and immune impairment, thereby predisposing patients to infectious complications.

Another important finding was the progressive increase in hospital stay with worsening hyponatremia. Patients with severe hyponatremia required a mean hospitalization of **11.3 ± 3.8 days**, compared with **6.2 ± 2.1 days** among patients with normal serum sodium levels. This observation indicates that hyponatremia is associated with increased disease complexity, greater need for supportive care, and delayed recovery (22-25).

Overall, the findings of the present study demonstrate that hyponatremia is not merely a laboratory abnormality but an important clinical marker of disease severity in decompensated chronic liver disease. Routine assessment of serum sodium may facilitate early identification of high-risk patients, improve clinical decision-making, and enable timely interventions to reduce complications.

Generalizability

The findings of this study are primarily applicable to adult patients with decompensated chronic liver disease receiving treatment at tertiary care hospitals similar to the study setting. Because the hospital serves as a major referral center for both urban and rural populations, the results are likely to be relevant to comparable healthcare institutions across India. However, caution should be exercised when generalizing these findings to primary care settings, community-based populations, or healthcare systems with different patient characteristics and management practices.

Conclusion

The present cross-sectional study demonstrated that hyponatremia is a common electrolyte abnormality among patients with decompensated chronic liver disease, with an overall prevalence of 70.2%. Ascites and jaundice were the most frequent clinical manifestations, while alcohol-related liver disease was the predominant etiology. Severe hyponatremia was significantly associated with hepatic encephalopathy, spontaneous bacterial peritonitis, and prolonged hospital stay. Thus, serum sodium concentration serves as an important indicator of disease severity and prognosis. Early identification and timely management of hyponatremia may contribute to improved clinical outcomes and reduced morbidity in patients with decompensated chronic liver disease.

Limitations

The present study has several limitations. First, the cross-sectional design precludes establishing a causal relationship between hyponatremia and adverse clinical outcomes. Second, the study was conducted at a single tertiary care center, which may limit the generalizability of the findings. Third, long-term clinical outcomes, including mortality and readmissions, were not evaluated because participants were not followed beyond hospitalization. Finally, serial measurements of serum sodium were not performed, limiting the assessment of changes in sodium concentration during the course of treatment.

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Acknowledgement

The authors express their sincere gratitude to the Department of Medicine, Pt. J.N.M. Medical College and Dr. B.R.A.M. Hospital, Raipur, Chhattisgarh, India, for providing the necessary facilities and support for conducting the study. The authors are also thankful to all the patients who participated in this study for their valuable cooperation.

LIST OF ABBREVIATIONS

CLD	Chronic Liver Disease
DCLD	Decompensated Chronic Liver Disease
SBP	Spontaneous Bacterial Peritonitis
MELD	Model for End-stage Liver Disease
INR	International Normalized Ratio
ALT	Alanine Aminotransferase
AST	Aspartate Aminotransferase
SD	Standard Deviation
SPSS	Statistical Package for Social Sciences
NAFLD	Non-Alcoholic Fatty Liver Disease

Funding

This research received **no specific grant** from any funding agency in the public, commercial, or not-for-profit sectors. The funding source had **no role** in the study design, data collection, data analysis, interpretation of results, manuscript preparation, or the decision to publish.

Data availability

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

Author contributions

Venugopal Margekar: Conceptualization, study design, supervision, interpretation of results, critical revision of the manuscript, and final approval.

Anusha Chouhan: Data collection, patient recruitment, statistical analysis, literature review, manuscript drafting, and final approval.

Vanini: Data interpretation, manuscript editing, literature review, critical revision, and final approval.

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References

1. Asrani SK, Devarbhavi H, Eaton J, Kamath PS. Burden of liver diseases in the world. J Hepatol. 2019;70(1):151-71. https://doi.org/10.1016/j.jhep.2018.09.014
2. Schuppan D, Afdhal NH. Liver cirrhosis. Lancet. 2008;371(9615):838-51. https://doi.org/10.1016/S0140-6736(08)60383-9
3. D'Amico G, Garcia-Tsao G, Pagliaro L. Natural history and prognostic indicators of survival in cirrhosis: A systematic review of 118 studies. J Hepatol. 2006;44(1):217-31. https://doi.org/10.1016/j.jhep.2005.10.013
4. Arroyo V, Ginès P, Gerbes AL, Dudley FJ, Gentilini P, Laffi G, et al. Definition and diagnostic criteria of refractory ascites and hepatorenal syndrome in cirrhosis. Hepatology. 1996;23(1):164-76. https://doi.org/10.1002/hep.510230122 https://doi.org/10.1053/jhep.1996.v23.ajhep023016 4
5. Ginès P, Berl T, Bernardi M, Bichet DG, Hamon G, Jiménez W, et al. Hyponatremia in cirrhosis: From pathogenesis to treatment. Hepatology. 1998;28(3):851-64. https://doi.org/10.1002/hep.510280337
6. Schrier RW, Arroyo V, Bernardi M, Epstein M, Henriksen JH, Rodés J. Peripheral arterial vasodilation hypothesis: A proposal for the initiation of renal sodium and water retention in cirrhosis. Hepatology. 1988;8(5):1151-57. https://doi.org/10.1002/hep.1840080532
7. Bernardi M, Angeli P, Claria J, Moreau R, Ginès P, Jalan R, et al. Albumin in decompensated

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cirrhosis: New concepts and perspectives. Gut. 2020;69(6):1127-38. https://doi.org/10.1136/gutjnl-2019-318843
8. Biggins SW, Kim WR, Terrault NA, Saab S, Balan V, Schiano T, et al. Evidence-based incorporation of serum sodium concentration into MELD. Gastroenterology. 2006;130(6):1652-60. https://doi.org/10.1053/j.gastro.2006.02.010
9. Cárdenas A, Ginès P. Management of patients with cirrhosis awaiting liver transplantation. Gut. 2011;60(3):412-21. https://doi.org/10.1136/gut.2009.179937
10. Angeli P, Wong F, Watson H, Ginès P. Hyponatremia in cirrhosis: Results of a patient population survey. Hepatology. 2006;44(6):1535-42. https://doi.org/10.1002/hep.21412
11. Kim WR, Biggins SW, Kremers WK, Wiesner RH, Kamath PS, Benson JT, et al. Hyponatremia and mortality among patients on the liver-transplant waiting list. N Engl J Med. 2008;359(10):1018-26. https://doi.org/10.1056/NEJMoa0801209
12. European Association for the Study of the Liver. EASL clinical practice guidelines for the management of patients with decompensated cirrhosis. J Hepatol. 2018;69(2):406-60. https://doi.org/10.1016/j.jhep.2018.08.009
13. Borroni G, Maggi A, Sangiovanni A, Cazzaniga M, Salerno F. Clinical relevance of hyponatremia for the hospital outcome of cirrhotic patients. Dig Liver Dis. 2000;32(7):605-10. https://doi.org/10.1016/S1590-8658(00)80844-0
14. Moreau R, Jalan R, Gines P, Pavesi M, Angeli P, Cordoba J, et al. Acute-on-chronic liver failure is a distinct syndrome developing in patients with acute decompensation of cirrhosis. Gastroenterology. 2013;144(7):1426-37. https://doi.org/10.1053/j.gastro.2013.02.042
15. Arroyo V, Moreau R, Jalan R, Ginès P. Acute-on-chronic liver failure: A new syndrome that will reclassify cirrhosis. J Hepatol. 2015;62(1 Suppl): S131-43. https://doi.org/10.1016/j.jhep.2014.11.045
16. Ginès P, Quintero E, Arroyo V, Terés J, Bruguera M, Rimola A, et al. Compensated cirrhosis: Natural history and prognostic factors. Hepatology. 1987;7(1):122-28. https://doi.org/10.1002/hep.1840070124
17. Bernardi M, Ricci CS, Zaccherini G. Role of human albumin in the management of complications of liver cirrhosis. J Clin Exp Hepatol. 2014;4(4):302-11. https://doi.org/10.1016/j.jceh.2014.08.007
18. Schrier RW, Martin PY, Lee SS. Pathogenesis and management of sodium and water retention in cirrhosis. Semin Liver Dis. 1994;14(3):202-13.
19. Runyon BA. Introduction to the revised American Association for the Study of Liver Diseases practice guideline management of adult patients with ascites due to cirrhosis. Hepatology. 2013;57(4):1651-53. https://doi.org/10.1002/hep.26359
20. D'Amico G, Morabito A, D'Amico M, Pasta L, Malizia G, Rebora P, et al. Clinical states of cirrhosis and competing risks. J Hepatol. 2018;68(3):563-76. https://doi.org/10.1016/j.jhep.2017.10.020
21. Nusrat S, Khan MS, Fazili J, Madhoun MF. Cirrhosis and its complications: Evidence-based treatment. World J Gastroenterol. 2014;20(18):5442-60. https://doi.org/10.3748/wjg.v20.i18.5442
22. Córdoba J, Guevara M. Hyponatremia and hepatic encephalopathy in cirrhosis. Gastroenterol Hepatol. 2003;26(1):29-37.
23. Guevara M, Baccaro ME, Torre A, Gómez-Ansón B, Ríos J, Torres F, et al. Hyponatremia is a risk factor of hepatic encephalopathy in patients with cirrhosis. Hepatology. 2009;50(4):1133-40.
24. Heuman DM, Abou-Assi SG, Habib A, Williams LM, Stravitz RT, Sanyal AJ, et al. Persistent ascites and low serum sodium identify patients with cirrhosis and low MELD scores who are at high risk for early death. Hepatology. 2004;40(4):802-10. https://doi.org/10.1002/hep.1840400409
25. Ginès P, Cárdenas A. The management of ascites and hyponatremia in cirrhosis. Semin Liver Dis. 2008;28(1):43-58. https://doi.org/10.1055/s-2008-1040320

Student's Journal of Health Research Africa
e-ISSN: 2709-9997, p-ISSN: 3006-1059
Vol.7 No. 2 (2026): June 2026 Issue
https://doi.org/10.51168/8pdz2282
Original Article

PUBLISHER DETAILS:

Student's Journal of Health Research (SJHR)
(ISSN 2709-9997) Online
(ISSN 3006-1059) Print
Category: Non-Governmental & Non-profit Organization
Email: studentsjournal2020@gmail.com
WhatsApp: +256 775 434 261
Location: Scholar's Summit Nakigalala, P. O. Box 701432, Entebbe Uganda, East Africa