RESEARCH PAPER

Incidence of Hyponatremia in Chronic Liver Disease Patient and Correlation with Severity of the Disease

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ABSTRACT

Background: Hyponatremia is a frequent electrolyte disturbance in patients with chronic liver disease (CLD) and is considered a marker of advanced disease and poor prognosis. It results primarily from circulatory dysfunction and impaired free water excretion in cirrhosis. Despite its clinical significance, the incidence and impact of hyponatremia on disease severity and outcomes require further evaluation in the Indian population.

Aim: To determine the incidence of hyponatremia in patients with chronic liver disease and to assess its correlation with disease severity, complications, and clinical outcomes.

Materials and Methods: This hospital-based cross-sectional observational study was conducted in the Department of Medicine at a tertiary care center and included 80 patients diagnosed with chronic liver disease. Patients were evaluated clinically and with laboratory investigations including serum sodium, liver function tests, and renal parameters. Disease severity was assessed using Child-Pugh and MELD/MELD-Na scores. Hyponatremia was classified as mild, moderate, or severe based on serum sodium levels. Statistical analysis was performed using ANOVA and Chi-square tests, with $p<0.05$ considered significant.

Results: Hyponatremia was observed in 48.75% of patients, with moderate hyponatremia being the most common (26.25%). Alcohol was the predominant etiology (56.25%). A significant association was found between hyponatremia and disease severity, with mean MELD scores increasing from 15.66 in normonatremic patients to 25.60 in severe hyponatremia ($p=0.0021$), and MELD-Na scores from 16.37 to 32.00 ($p<0.0001$). Patients with moderate and severe hyponatremia had higher incidences of complications such as ascites, hepatic encephalopathy, and acute kidney injury. A trend toward higher grades of esophageal varices was also observed with worsening hyponatremia. The duration of hospital stay increased significantly with decreasing sodium levels ($p=0.0006$). Mortality was markedly higher in patients with severe hyponatremia (80%) compared to normonatremic patients (2.4%) ($p<0.0001$).

Conclusion: Hyponatremia is highly prevalent in patients with chronic liver disease and shows a strong correlation with disease severity, complications, and mortality. It serves as an important, cost-effective prognostic marker and should be routinely assessed in clinical practice for risk stratification and management of CLD patients.

Keywords: Chronic liver disease, Hyponatremia, MELD score, Hepatic encephalopathy, Mortality.

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INTRODUCTION

Liver diseases encompass a broad spectrum of pathological conditions characterized by hepatocyte injury, inflammatory cell infiltration, and hepatic stellate cell (HSC) activation, ultimately resulting in structural distortion and functional impairment of the liver[1,2]. Globally, liver disease remains a major contributor to mortality, being responsible for nearly 2 million deaths per year and accounting for approximately 4% of total global mortality.[3] According to the Global Burden of Disease 2019 study, 1.26 million deaths were attributed to cirrhosis and

chronic liver diseases in 2019, reflecting a 13% increase since 1990.[4] Notably, the sharpest rise in chronic liver disease (CLD)–related mortality has been reported in low- and lower-middle-income countries across Asia and Africa, where evolving demographic and epidemiological trends contribute to increasing disease burden.[5,6]India alone accounts for an estimated 18.3% of global liver-related deaths, positioning it as a critical epicenter of this growing health challenge.[6]

Acute liver diseases are frequently linked to hepatotropic viral infections, with drug-induced liver injury (DILI) emerging as an increasingly significant global cause. In contrast, chronic liver diseases primarily stem from prolonged alcohol consumption, hepatitis B (HBV) and hepatitis C (HCV) infections, and rising occurrences of metabolic dysfunction-associated steatotic liver disease (MASLD).[7]

The progression to advanced disease states such as cirrhosis and hepatocellular carcinoma significantly increases global morbidity and mortality.[3] Chronic liver disease is defined as progressive hepatic functional deterioration persisting for over six months, involving impaired protein synthesis, detoxification, metabolism, and bile secretion. Persistent cycles of inflammation, destruction, and regeneration of hepatic parenchyma lead to fibrosis and ultimately cirrhosis, resulting in nodular transformation, vascular remodeling, neo-angiogenesis, and extracellular matrix deposition.[8]

Inflammation and oxidative stress play central roles in CLD pathogenesis across conditions including alcoholic liver disease (ALD), non-alcoholic fatty liver disease (NAFLD), and hepatocellular carcinoma (HCC).[9] Several signaling pathways, particularly Toll-like receptor (TLR), nuclear factor-κB (NF-κB), and heme oxygenase-1 (HO-1), are implicated in these processes. The dysregulated lipid metabolism further also contributes to oxidative stress and inflammatory injury.[10]

Liver cirrhosis is frequently complicated by refractory ascites, hypotension, and severe hyponatremia. This occurs due to portal hypertension-induced vascular dysregulation, impaired responsiveness to vasoactive mediators, and also reduced renal clearance of solute-free water.[11]

Hyponatremia is one of the most common and clinically significant complications in cirrhotic patients, strongly associated with impaired water excretion and increased mortality risk.[12] Hyponatremia is classified according to the serum sodium concentration, with levels of 130–134 mEq/L considered mild, 125–129 mEq/L regarded as moderate and values below 125 mEq/L defined as severe.[13] Its frequency and severity increase as cirrhosis progresses.[14] Approximately 57% of hospitalized and 40% of ambulatory CLD patients experience hyponatremia, underlining its high prevalence and strong association with disease severity.[15] This necessitates rigorous monitoring as well as management of sodium levels to prevent life-threatening outcomes.

Despite its clinical relevance, the correlation between serum sodium levels and outcomes in cirrhosis remains inadequately characterized in many regions, including India. Hyponatremia commonly develops secondary to increased extracellular fluid volume, although hypovolemic hyponatremia may occur with diuretic therapy or gastrointestinal losses.[16] Cirrhosis causes reduced effective arterial blood volume despite total body water expansion due to excessive vasodilators such as nitric oxide, endotoxins, substance P, and endogenous cannabinoids.[17] These processes activate the renin–angiotensin–aldosterone system and stimulate antidiuretic hormone (ADH)-mediated water retention in the collecting tubules while increasing sodium reabsorption proximally and excretion distally.[18] In cirrhotic patients with ascites, non-osmotic ADH release becomes dominant, resulting in impaired water clearance and dilutional hyponatremia.[19]

Hyponatremia has also been associated with hepatic encephalopathy (HE), potentially due to osmotic shifts causing astrocyte swelling. Patients with hyponatremia are reportedly eight times more prone to develop HE, with severity correlating with higher HE grades. Recent studies suggest hyponatremia as a significant prognostic indicator in chronic liver disease.[20]

The systematic evaluation of the incidence of hyponatremia and its correlation with disease severity and outcomes in chronic liver disease patients is essential, particularly in the Indian context where data remain limited. This study aims to investigate the incidence of hyponatremia in chronic liver disease patients, examine its correlation with disease severity, and assess hyponatremia-associated morbidity and mortality among hospitalized patients.

METHODOLOGY

Study design and setting

This hospital-based cross-sectional observational study was conducted in the Department of Medicine at Gandhi Medical College and its associated Hamidia Hospital, Bhopal, Madhya Pradesh. The study was carried out over a period of 18 months after obtaining

institutional approval. The tertiary care center caters to a large number of patients with chronic liver disease, providing an appropriate setting for evaluating the incidence and clinical significance of hyponatremia.

Ethical considerations

Written informed consent was obtained from all patients or their legally authorized representatives before enrolment. Confidentiality of patient information was strictly maintained throughout the study. Participation was voluntary, and no additional intervention beyond routine clinical evaluation and standard management was introduced as part of the study protocol.

Study population

The study included adult patients diagnosed with chronic liver disease who were admitted to or attended the Department of Medicine during the study period. Patients with or without hyponatremia were included. Patients with congestive heart failure, chronic kidney disease, recent use of diuretics, or non-cirrhotic portal hypertension were excluded. All eligible patients fulfilling the inclusion criteria were enrolled consecutively.

Sample size and sampling

A total of 80 patients with chronic liver disease were included in the study. Consecutive sampling technique was employed, wherein all eligible patients presenting during the study period were included until the desired sample size was achieved.

Data collection

Data were collected using a predesigned structured proforma. Information regarding demographic profile (age, sex), clinical history, probable etiology of liver disease (alcohol, viral, metabolic), duration of illness, and associated symptoms was recorded. Detailed clinical examination was performed in each patient, including assessment of pallor, icterus, ascites, hepatic encephalopathy, and other complications.

Laboratory investigations included serum sodium, liver function tests (bilirubin, transaminases, albumin), renal function tests (serum urea and creatinine), and coagulation profile (INR). Relevant investigations such as viral markers (HBsAg, Anti-HCV) were also performed where indicated.

Diagnostic grouping and severity assessment

Patients were categorized based on serum sodium levels into normal, mild, moderate, and severe hyponatremia. Disease severity was assessed using standard scoring systems including the Child-Pugh score, Model for End-Stage Liver Disease (MELD) score, and MELD-Na score. Complications such as ascites, hepatic encephalopathy, acute kidney injury, and esophageal varices were evaluated and recorded. Patients were also assessed for outcomes including duration of hospital stay and mortality.

Statistical analysis

Data were entered in Microsoft Excel and analyzed using appropriate statistical software. Continuous variables were expressed as mean and standard deviation, while categorical variables were presented as frequency and percentage. One-way Analysis of Variance (ANOVA) was used to compare means across groups, and the Chi-square test was used to assess associations between categorical variables. A p value of less than 0.05 was considered statistically significant.

RESULT

Baseline Characteristics and Etiology

A total of 80 patients with chronic liver disease were included in the present study. The age distribution showed that the largest proportion of patients belonged to the 31–40 years age group (31.25%), followed by 41–50 years (25.00%) and 51–60 years (20.00%), while only a small fraction (2.50%) were above 70 years of age. The study population demonstrated a clear male predominance, with males constituting 77.50% of cases(Table 1).

In terms of etiology, alcohol-related liver disease emerged as the most common cause, accounting for 56.25% of patients. Viral causes included hepatitis B (22.50%) and hepatitis C (8.75%), while metabolic dysfunction-associated steatotic liver disease (MASLD) was identified in 8.75% of cases. In 3.75% of patients, no definitive etiology could be established. These findings indicate that alcohol remains the leading contributor to chronic liver disease in this cohort.

Hyponatremia and Disease Severity

Hyponatremia was identified in 48.75% of patients. Among these, moderate hyponatremia was the most frequent category (26.25%), followed by mild (16.25%) and severe (6.25%) hyponatremia, whereas 51.25% of patients had normal serum sodium levels(Table 2).

Assessment of disease severity revealed that the majority of patients (62.50%) were classified as Child-Pugh Class C, indicating advanced liver disease, while 37.50% belonged to Class B. Ascites was present in 91.25% of patients, reflecting a high prevalence of decompensated cirrhosis in the study population. Hepatic encephalopathy was observed in 37.50% of cases, and acute kidney injury (AKI) was present in 23.75% of patients, underscoring the substantial burden of complications associated with chronic liver disease.

Table 1: Baseline characteristics and etiology of study population (n = 80)

Variable	Frequency	Percentage (%)
Age group (years)		
20–30	3	3.75
31–40	25	31.25
41–50	20	25.00
51–60	16	20.00
61–70	14	17.50
>70	2	2.50
Sex		
Male	62	77.50
Female	18	22.50
Etiology of CLD		
Alcohol-related	45	56.25
HBV-related	18	22.50
HCV-related	7	8.75
MASLD	7	8.75
Unknown	3	3.75

Comparison of Clinical, Laboratory, and Outcome Parameters

Comparative analysis across hyponatremia severity groups demonstrated several significant associations. The mean age did not differ significantly among groups (p=0.9786), suggesting comparable baseline demographic characteristics(Table 3).Systolic blood pressure showed a statistically significant variation (p=0.0462), with relatively lower values observed in patients with severe hyponatremia. Hemoglobin levels also differed significantly (p=0.0224), with the lowest mean values recorded in the severe hyponatremia group.Renal function parameters exhibited a progressive deterioration with increasing severity of hyponatremia. Serum creatinine levels increased significantly from 1.04 ± 0.45 mg/dL in normonatremic patients to 2.10 ± 2.15 mg/dL in those with severe hyponatremia (p=0.0035), indicating a higher prevalence of renal dysfunction in this group.Markers of liver disease severity showed a strong correlation with serum sodium levels. The mean MELD score increased significantly from 15.66 in the normal group to 25.60 in the severe hyponatremia group (p=0.0021). Similarly, the MELD-Na score demonstrated a marked increase from 16.37 to 32.00 (p<0.0001), highlighting the close association between hyponatremia and advanced liver dysfunction.The duration of hospital stay increased significantly with worsening hyponatremia (p=0.0006), rising from a mean of 5.49 days in normonatremic patients to 9.80 days in those with severe hyponatremia.A significant association was also observed between hyponatremia severity and mortality (p<0.0001). Mortality rates increased markedly from 2.4% in the normal group to 28.6% in the moderate group and 80.0% in the severe hyponatremia group, indicating a strong relationship between low serum sodium levels and adverse clinical outcomes.

Table 2: Distribution of hyponatremia and disease severity parameters

Variable	Frequency	Percentage (%)
Hyponatremia		
Normal	41	51.25
Mild	13	16.25
Moderate	21	26.25
Severe	5	6.25
Child-Pugh class		
Class B	30	37.50
Class C	50	62.50
Ascites		
Present	73	91.25
Absent	7	8.75
Hepatic encephalopathy		
Absent	50	62.50
Present	30	37.50
AKI		
Present	19	23.75
Absent	61	76.25

Table 3: Comparison of outcomes and severity scores according to hyponatremia

Parameter	Normal (n=41)	Mild (n=13)	Moderate (n=21)	Severe (n=5)	p value
Age (years)	48.22 ± 13.00	48.38 ± 12.26	47.38 ± 12.46	46.00 ± 11.42	0.9786
SBP (mmHg)	109.27 ± 11.49	118.46 ± 14.63	114.95 ± 12.85	104.00 ± 16.73	0.0462*
Hemoglobin (g/dL)	7.98 ± 2.38	8.38 ± 2.70	9.78 ± 2.60	6.40 ± 3.65	0.0224*
Serum creatinine (mg/dL)	1.04 ± 0.45	1.10 ± 0.33	1.99 ± 1.66	2.10 ± 2.15	0.0035*
MELD	15.66	17.1	21.86 ±	25.6	0.0021

score	± 5.73	5 ± 6.73	9.85	0 ± 5.86	*
MELD-Na score	16.37 ± 5.72	19.46 ± 7.31	25.76 ± 10.30	32.00 ± 4.06	<0.0001*
Hospital stay (days)	5.49 ± 1.98	5.46 ± 1.90	7.14 ± 3.32	9.80 ± 1.92	0.0006*
Mortality (%)	2.4	0.0	28.6	80.0	<0.0001*

*Values expressed as mean ± SD

$p < 0.05$ considered statistically significant.

DISCUSSION

The present study evaluated the incidence of hyponatremia in patients with chronic liver disease (CLD) and its relationship with disease severity, complications, and clinical outcomes. The findings highlight the significant role of hyponatremia as a prognostic marker and are consistent with previously published literature.

In this study, hyponatremia was observed in 48.75% of patients, with moderate hyponatremia being the most common. This prevalence is comparable to earlier studies reporting hyponatremia in approximately 20–50% of cirrhotic patients [21]. Similar findings were reported by Reddy and Madhuri et al., who observed hyponatremia in 53% of patients with cirrhosis [22].Other regional studies have reported comparable prevalence rates of around 49% [23]. while more recent data have shown rates ranging from 36% to over 60% in hospitalized patients with decompensated liver disease [21].. These variations may be attributed to differences in disease stage, patient population, and sodium cut-off values. The relatively high incidence in the present study likely reflects the predominance of advanced liver disease, as most patients belonged to Child-Pugh Class C.

The study population predominantly comprised middle-aged males, which is consistent with previous studies highlighting higher exposure to risk factors such as alcohol among males. Alcohol-related liver disease was the most common etiology (56.25%), followed by viral causes. This finding aligns with earlier Indian studies and large multicentric data, which identify alcohol as a leading cause of cirrhosis, particularly in decompensated cases [22,24].The presence of MASLD cases also reflects the evolving epidemiological trend toward metabolic risk factors.

A key finding of this study is the significant association between hyponatremia and disease severity. Patients with lower serum sodium levels demonstrated higher Child-Pugh, MELD, and MELD-Na scores. This observation is consistent with previous evidence indicating that hyponatremia correlates with advanced hepatic dysfunction and portal hypertension. The incorporation of serum sodium into the MELD-Na score has been shown to improve prognostic accuracy for mortality [25]. which is supported by the highly significant association observed in the present study ($p<0.0001$). Recent studies have also demonstrated a strong inverse correlation between serum sodium levels and disease severity scores [26,27]. further reinforcing these findings.

Ascites was highly prevalent in the present study, although its association with hyponatremia severity was not statistically significant. However, previous studies have demonstrated that hyponatremia is closely linked with circulatory dysfunction and refractory ascites. Similarly, hepatic encephalopathy was more frequent in patients with moderate and severe hyponatremia, although statistical significance was not achieved, likely due to the limited sample size. Prior studies have identified hyponatremia as an independent risk factor for hepatic encephalopathy through mechanisms involving astrocyte swelling and cerebral edema[28].

A trend toward higher grades of esophageal varices with worsening hyponatremia was also observed. This is supported by previous studies demonstrating that hyponatremia reflects advanced portal hypertension and is associated with complications such as varices [29]. Although not statistically significant, the observed trend is clinically relevant.

Hyponatremia and Renal Dysfunction

A significant association was observed between hyponatremia and acute kidney injury (AKI), with higher prevalence in moderate and severe hyponatremia groups ($p=0.0125$). This finding is consistent with earlier studies demonstrating that hyponatremia reflects severe circulatory dysfunction and reduced renal perfusion, predisposing patients to renal impairment and hepatorenal syndrome [30].

Hyponatremia and Laboratory Parameters

The present study demonstrated that worsening hyponatremia was associated with increased serum urea and creatinine levels and lower hemoglobin levels. These findings suggest that hyponatremia is associated with multi-organ dysfunction, particularly renal impairment, as reported in previous studies [31].

One of the most important findings of this study is the strong association between hyponatremia and mortality. Mortality increased progressively with worsening hyponatremia, reaching 80% in the severe group ($p<0.0001$). This finding is consistent with multiple studies that have identified hyponatremia as an

independent predictor of mortality in cirrhosis [28]. Recent studies have also confirmed that hyponatremia significantly predicts mortality in decompensated liver disease [32], with reported mortality rates increasing in parallel with the severity of sodium derangement.

The duration of hospital stay increased significantly with worsening hyponatremia (p=0.0006), indicating a higher burden of disease and complications. Similar findings have been reported in previous studies, where hyponatremia was associated with prolonged hospitalization and increased healthcare utilization [33,34].

Overall, the findings of the present study strongly support the role of hyponatremia as a reliable and clinically relevant prognostic marker in chronic liver disease. Hyponatremia reflects advanced circulatory dysfunction and is associated with increased disease severity, higher complication rates, prolonged hospitalization, and increased mortality. These observations emphasize the importance of routine monitoring and early management of serum sodium levels in patients with chronic liver disease.

CONCLUSION

Hyponatremia is a common and clinically significant finding in patients with chronic liver disease, observed in nearly half of the study population. It demonstrates a strong correlation with disease severity, as evidenced by higher Child-Pugh, MELD, and MELD-Na scores. Increasing severity of hyponatremia is associated with adverse outcomes, including renal dysfunction, prolonged hospitalization, and markedly increased mortality. Although some complications did not show statistical significance, consistent clinical trends suggest that hyponatremia reflects advanced disease and circulatory dysfunction. Serum sodium, therefore, serves as a simple, cost-effective prognostic marker and should be routinely assessed for risk stratification and management of patients with chronic liver disease.

REFERENCES

1. Gan C, Yuan Y, Shen H, Gao J, Kong X, Che Z, et al. Liver diseases: epidemiology, causes, trends and predictions. Signal Transduct Target Ther [Internet]. 2025;10:33. Available from: http://www.ncbi.nlm.nih.gov/pubmed/39904973

2. Han H, Desert R, Das S, Song Z, Athavale D, Ge X, et al. Danger signals in liver injury and restoration of homeostasis. J. Hepatol. 2020. p. 933–51.

3. Devarbhavi H, Asrani SK, Arab JP, Nartey YA, Pose E, Kamath PS. Global burden of liver disease: 2023 update. J. Hepatol. 2023. p. 516–37.

4. Sepanlou SG, Safiri S, Bisignano C, Ikuta KS, Merat S, Saberifiroozi M, et al. The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol Hepatol. 2020;5:245–66.

5. Sinha J. Study Of Serum Sodium Levels And Its Clinical Significance In Decompensated Chronic Liver Disease Patients Admitted In A Tertiary Care Centre Of North Eastern State, Tripura. Int J Adv Res. 2024;12:779–85.

6. Parida PK, Patnaik SK, Pradhan S, Kanungo M, Mishra D, Narayan J, et al. The Burden of Liver Disease in Eastern India : Epidemiology and Analysis of Different Biochemical Parameters to Know the Risk Score for CLD Patients. Int J Pharm Clin Res. 2023;15:717–24.

7. Wong MCS, Huang JLW, George J, Huang J, Leung C, Eslam M, et al. The changing epidemiology of liver diseases in the Asia–Pacific region [Internet]. Nat. Rev. Gastroenterol. Hepatol. 2019. p. 57–73. Available from: https://www.nature.com/articles/s41575-018-0055-0

8. Sharma A, Nagalli S. Chronic Liver Disease [Internet]. StatPearls. 2025. Available from: http://www.ncbi.nlm.nih.gov/pubmed/16970019

9. Xu J-J, Li H-D, Wu M-F, Zhu L, Du X-S, Li J-J, et al. 3-B-RUT, a derivative of RUT, protected against alcohol-induced liver injury by attenuating inflammation and oxidative stress. Int Immunopharmacol [Internet]. 2021;95:107471. Available from: http://www.ncbi.nlm.nih.gov/pubmed/33756231

10. Zhang CY, Liu S, Yang M. Antioxidant and anti-inflammatory agents in chronic liver diseases: Molecular mechanisms and therapy. World J Hepatol. 2023;15:180–200.

11. Angeli P, Wong F, Watson H, Ginès P, Capps I. Hyponatremia in cirrhosis: Results of a patient population survey. Hepatol. (Baltimore, Md.). 2014. p. 1535–42.

12. Fernández-Esparrach G, Sánchez-Fueyo A, Ginès P, Uriz J, Quintó L, Ventura PJ, et al. A prognostic model for predicting survival in cirrhosis with ascites. J Hepatol. 2001;34:46–52.

13. Miller NE, Rushlow D, Stacey SK. Diagnosis and Management of Sodium Disorders: Hyponatremia and Hypernatremia. Am Fam Physician. 2023;108:476–86.

14. Qureshi MO, Khokhar N, Saleem A, Niazi TK. Correlation of hyponatremia with hepatic encephalopathy and severity of liver disease. J Coll Physicians Surg Pakistan. 2014;24:135–7.

15. Ginés P, Berl T, Bernardi M, Bichet DG, Hamon G, Jiménez W, et al. Hyponatremia in cirrhosis: From

pathogenesis to treatment. Hepatology. 1998;28:851–64.
16. Jong HK, June SL, Seuk HL, Won KB, Kim NH, Kim KA, et al. The association between the serum sodium level and the severity of complications in liver cirrhosis. Korean J Intern Med. 2009;24:106–12.
17. Ginès P, Cárdenas A. The management of ascites and hyponatremia in cirrhosis. Semin. Liver Dis. 2008. p. 43–58.
18. Tivers MS, Handel I, Gow AG, Lipscomb VJ, Jalan R, Mellanby RJ. Hyperammonemia and systemic inflammatory response syndrome predicts presence of hepatic encephalopathy in dogs with congenital portosystemic shunts. PLoS One [Internet]. 2014;9:e82303. Available from: http://www.ncbi.nlm.nih.gov/pubmed/24392080
19. Bernardi M, Zaccherini G. Approach and management of dysnatremias in cirrhosis. Hepatol Int. 2018;12:487–99.
20. Yun BC, Kim WR. Editorial: Hyponatremia in hepatic encephalopathy: An accomplice or innocent bystander. Am J Gastroenterol. 2009;104:1390–1.
21. Azam MU, Javed S, Memon MYY, Aftab MA, Shafqat MN, Sadiq HZ, et al. Hyponatremia Prevalence in Decompensated Chronic Liver Disease: Insights from a Tertiary Care Hospital. Cureus. 2024;16:e68907.
22. Reddy DDK. Study of Hyponatremia in Cirrhosis of Liver and It's Prognostic Value. J Med Sci Clin Res. 2019;7.
23. Singh Y, Nagar D, Singh M, Maroof M. Study of electrolyte disturbance in chronic liver disease patients attending a hospital in Kumaon region. J Fam Med Prim Care. 2022;11:4479–82.
24. Patel UB, Kanani TJ, Patel DA. Original Article A Study of Hyponatremia in Patients of Liver Cirrhosis in a Tertiary Care Hospital of South Gujarat.
25. 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;359:1018–26.
26. Namal AV, S.Vithiavathi, Sakar G, Kumar VAV. A Study of Serum Sodium Levels in Decompensated Chronic Liver Disease and Its Correlation with the Severity of the Disease. J Chem Heal Risks. 2025;15:1286–94.
27. Choudhury BN, Baruah BJ, Gandhi N, Bhattacharyya M, Deka UJ, Nanda J, et al. A Clinical Study of Dyselectrolytemia in Patients with Cirrhosis of Liver and its Association with Severity of Disease and Development of Complications. J Indian Med Assoc. 2023;121:22–6.
28. Gaglio P, Marfo K, Joseph Chiodo, Chiodo J. Hyponatremia in cirrhosis and end-stage liver disease: treatment with the vasopressin V_2-receptor antagonist tolvaptan. Dig Dis Sci [Internet]. 2012;57:2774–85. Available from: http://www.ncbi.nlm.nih.gov/pubmed/22732834
29. Garcia-Tsao G, Abraldes JG, Berzigotti A, Bosch J. Portal hypertensive bleeding in cirrhosis. Hepatology [Internet]. 2017;65:310–35. Available from: https://journals.lww.com/01515467-201701000-00032
30. Londoñdo MC, Cárdenas A, Guevara M, Quintó L, De Las Heras D, Navasa M, et al. MELD score and serum sodium in the prediction of survival of patients with cirrhosis awaiting liver transplantation. Gut. 2007;56:1283–90.
31. Bai Z, Yin Y, Xu W, Cheng G, Qi X. Predictive model of in-hospital mortality in liver cirrhosis patients with hyponatremia: an artificial neural network approach. Sci Rep. 2024;14.
32. Shekar KC, Mohan TS, Sirisha BN, Sivaiah T, Sruthi BN, Kolli JR. An Observational Descriptive Study on the Prognostic Significance of Serum Sodium Levels Among Decompensated Chronic Liver Patients. Eur J Cardiovasc Med. 2025;15:375–80.
33. Rondon-Berrios H, Velez JCQ. Hyponatremia in Cirrhosis. Clin Liver Dis [Internet]. 2022;26:149–64. Available from: http://www.ncbi.nlm.nih.gov/pubmed/35487602
34. Thuluvath PJ, Alukal JJ, Zhang T. Impact of Hyponatremia on Morbidity, Mortality, and Resource Utilization in Portal Hypertensive Ascites: A Nationwide Analysis. J Clin Exp Hepatol. 2022;12:871–5.