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"A Study of Serum Sodium Levels in Decompensated Chronic Liver Disease and Its Correlation with the Severity of the Disease"

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KEYWORDS

Decompensated Chronic Liver Disease, Hyponatremia, MELD Score, Prognosis, Serum Sodium, Cirrhosis.

ABSTRACT:

Background: Decompensated Chronic Liver Disease (DCLD) is frequently associated with disturbances in water balance, leading to abnormalities in serum sodium levels. Hyponatremia, mainly dilutional, is common due to impaired free water clearance, while hypernatremia, though less frequent, has been linked to increased mortality. Several studies have demonstrated a correlation between serum sodium levels and survival in cirrhotic patients.

Aim: The study aims to assess serum sodium levels in patients with DCLD and establish their clinical significance in predicting disease severity and adverse outcomes.

Materials and Methods: This prospective descriptive cohort study was conducted over 18 months in a tertiary care hospital on 125 patients admitted with DCLD. Patients were evaluated based on clinical history, examination, laboratory investigations, and imaging. Serum sodium levels were recorded, and their association with complications, MELD score, and outcomes was analyzed. Data were statistically assessed using SPSS v23.0.

Results: The study population had a mean age of 50.11±10.22 years and was predominantly male (93.6%). Common clinical manifestations included ascites (100%), portal hypertension (96.8%), hepatic encephalopathy (8.8%), spontaneous bacterial peritonitis (13.6%), and hepatorenal syndrome (12.8%). A significant inverse correlation was observed between serum sodium levels and both MELD ($r = -0.439$, $p < 0.05$) and Child-Pugh scores ($r = -0.433$, $p < 0.05$), indicating that mean sodium level 129 – 134mE/L were associated with greater disease severity. Hyponatremia was linked to adverse outcomes, with non-survivors exhibiting significantly lower sodium levels (125.58±8.2) compared to discharged patients (134.5±6.9) ($p < 0.05$).

Conclusion: Serum sodium levels are crucial in assessing the severity and prognosis of DCLD. Hyponatremia correlates with advanced liver dysfunction and increased mortality. Routine sodium assessment may aid in early risk stratification and management of patients with DCLD.

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Introduction:

Decompensated chronic liver disease (DCLD) disrupts water balance regulation, resulting in significant alterations in serum sodium levels. Research has consistently demonstrated a strong association between sodium concentrations and patient survival in DCLD. The most frequently observed electrolyte disturbance is dilutional hyponatremia, primarily caused by impaired free water excretion. A similar study has recently been published in which hyperaemia has also been documented in certain cases.[1–3] Cirrhosis is a major cause of mortality and is associated with several complications, including ascites, hepatic encephalopathy, portal hypertension, spontaneous bacterial peritonitis, hepatorenal syndrome, and hepatopulmonary syndrome. It often leads to disturbances in renal function and electrolyte imbalances. Hyponatremia is a prevalent issue in cirrhotic patients and can manifest as either hypovolemic or hypervolemic, further complicating disease management.[4]

Reduced renal sodium excretion, resulting from renal hypoperfusion and excessive antidiuretic hormone secretion due to decreased effective blood volume caused by peripheral arterial vasodilation, leads to dilutional hyponatremia. Early identification and management of hyponatremia are crucial in preventing complications such as hepatic encephalopathy, hepatorenal syndrome, and spontaneous bacterial peritonitis, ultimately improving patient survival rates.[5–8] Extensive studies on large cohorts of cirrhotic patients have shown a significant association between the kidney's ability to excrete free water—measured through solute-free water clearance or serum sodium levels—and overall survival. Patients with hyponatremia exhibit poorer survival outcomes compared to those with normal sodium levels.[9–12] According to recent research, hyponatremia is a critical prognostic marker in patients with CLD, and individuals with hyponatremia have a worse survival rate than those without hyponatremia. Hence serum sodium levels have a role in predicting the prognosis and institutional management in patients with cirrhosis of liver.[13,14] Recent research has shown that lower serum sodium levels are linked to higher complication rates and mortality, leading to the inclusion of sodium in the MELD score. Similarly, hypernatremia has also been associated with increased mortality. Therefore, this study was conducted at our tertiary care hospital to evaluate serum sodium levels in patients with decompensated chronic liver disease (DCLD) and determine their clinical significance. The primary objective was to assess the impact of serum sodium levels on disease outcomes in DCLD patients.

Material & Method:

The study was conducted on patients admitted with decompensated chronic liver disease (DCLD) in the medical wards of Aarupadai Veedu Medical College and Hospital. It was a prospective descriptive cohort study carried out over 18 months with a sample size of 125, determined using statistical estimation based on adverse outcome proportions. Patients were selected based on history, clinical examination, laboratory investigations, and imaging findings indicative of DCLD. Inclusion criteria encompassed individuals aged 18–80 diagnosed with DCLD, while those with cardiac failure, chronic kidney disease, or on specific medications were excluded. Ethical clearance was obtained, and informed consent was secured from all participants.

Data collection involved demographic details, clinical assessment, and investigations such as CBC, renal and liver function tests, electrolytes, and ultrasound imaging. Serum sodium levels were measured at admission, and disease severity was assessed using the MELD score, which incorporates serum bilirubin, INR, and creatinine levels. Ascites and hepatic encephalopathy were graded based on standard criteria. Patients were monitored for complications and outcomes over 30 days.

The statistical analysis was performed using SPSS v23.0. Quantitative data were analyzed using measures of central tendency and dispersion, while qualitative data were assessed using Chi-square, ANOVA, and Fisher's test. A p-value of <0.05 was considered statistically significant. The study aimed to explore the relationship between serum sodium levels and adverse outcomes in DCLD, providing insights for risk stratification and clinical management.

Result: Present study included total of 125 patients with mean age of 50.11±10.22 yrs. Among the participants included 93.6% were male and 6.4% were female.

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Table 1: Distribution of the patients presenting symptoms and signs			
		Count	N %
Gender	Female	8	6.4%
	Male	117	93.6%
Abdominal distention	Present	125	100.0%
Altered sensorium	Absent	106	84.8%
	Present	19	15.2%
Gastro Intestinal bleeding	Absent	96	76.8%
	Present	29	23.2%
Alcoholic	Absent	11	8.8%
	Present	114	92.2%
Pallor	Absent	101	80.8%
	Present	24	19.2%
Icterus	Absent	85	68.0%
	Present	40	32.0%
Clubbing	Absent	84	67.2%
	Present	41	32.8%
Pedal edema	Present	125	100.0%
Signs of liver cell failure	Present	125	100.0%
Ascites	Present	125	100.0%
Organomegaly	Absent	110	88.0%
	SPL	15	12.0%
Ascites	G II	65	52.0%
	G III	60	48.0%
Portal hypertension	Absent	4	3.2%
	Present	121	96.8%
Hepatic encephalopathy	A	103	82.4%
	G I	4	3.2%
	G II	7	5.6%
	G III	11	8.8%
Spontaneous bacterial peritonitis	Absent	108	86.4%
	Present	17	13.6%

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Hepatorenal syndrome	Absent	109	87.2%
	Present	16	12.8%
GI bleeding	Absent	101	80.8%
	Present	24	19.2%
Coagulopathy	Absent	109	83.2%
	Present	16	12.8%

At presentation, all patients presented with abdominal distension, pedal edema, signs of liver cell failure, ascites (100%). 32.8% presented with jaundice and clubbing, 23.2% GI bleeding, 19.2% with pallor, 15.2% with altered sensation.

Table 2: Showing mean blood parameters

	Mean	SD
Urea	41.15	12.41
Creatinine	1.41	.72
Sodium	133.65	7.55
T. Bilirubin	2.04	1.40
D. Bilirubin	1.15	1.14
T. Protein	5.48	.81
Albumin	3.05	.53
ALT	64.93	26.51
AST	74.9	60.0
ALP	97.4	20.4
PT	17.74	3.23
INR	1.36	.24
MELD	14.2	5.4
CPS	8.3	1.7

Table 3: Showing the CPC class and presence of HBSAG, HCV infection, endoscopy and outcome among patients

		Count	N %
CPC	A	9	7.2%
	B	89	71.2%
	C	27	21.6%

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HBSAG	Negative	117	93.6%
	Positive	8	6.4%
HCV	Negative	124	99.2%
	Positive	1	0.8%
Endoscopy	Esophageal varices	87	71.2%
	Esophageal varices, PHT	26	20.8%
	Normal	10	8.0%
Outcome	Discharge	113	90.4%
	Expired	12	9.6%

Table showing the CPC severity with majority belonging to Class B (71.2%) followed by 21.6% in class C and 7.2% in class A CPC. Study found HbSAG positive in 6.4% and HCV positive in 0.8% of the cases.

Table 4: Correlation of MELD and CPS score with serum sodium level

Correlations		S. Sodium	MELD
MELD	r	-.439**	1
	p-value	.000	
CPS	r	-.433**	.681**
	p-value	.000	.000

**. Correlation is significant at the 0.01 level (2-tailed).

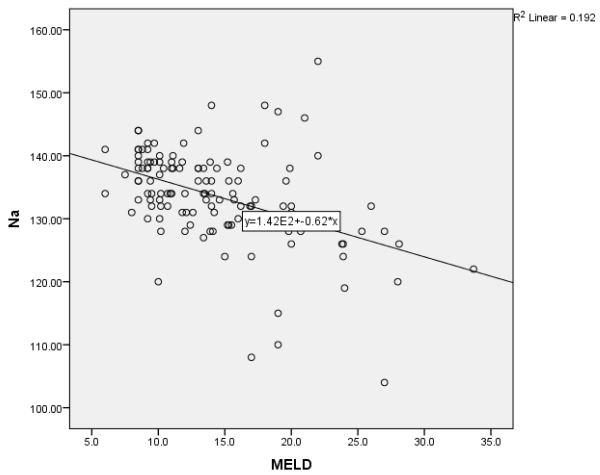

Figure 1: Correlation of MELD with serum sodium level

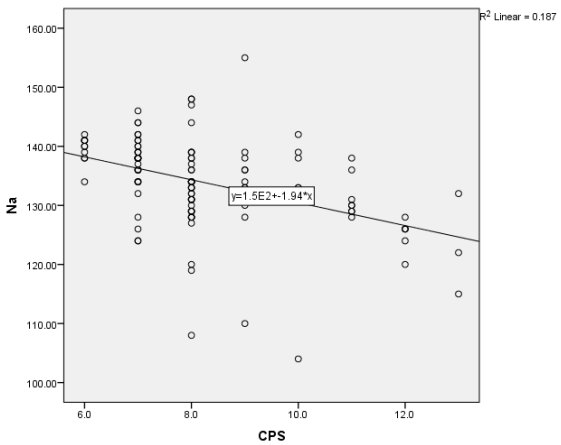

Figure 2: Correlation of CPS score with serum sodium level

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Table 5: Association of grade of severity of CPC class with serum sodium level				
		Serum Sodium		p-value
		Mean	SD	
CPC	Class A	139.33	2.45	0.01*
	Class B	134.34	7.33	
	Class C	129.48	7.55	

With severity of liver disease, with increase in grade of CPC the mean sodium level showing the decreasing trend. Showing the lower mean sodium in CPC C grade compared to CPC A grade.($p<0.05$)

Table 6: Comparison of serum sodium with outcome among patients					
	Discharge		Expired		p-value
	Mean	SD	Mean	SD	
S. Sodium	134.50	6.98	125.58	8.20	0.05*

On assessment of the sodium level with outcome of patients, there is significant hyponatremia seen in cases with worst outcome as death (125.58±8.2) compared to the patients discharged (134.5±6.9)

Discussion:

Present study included total of 125 patients with mean age of 50.11±10.22 yrs, with 93.6% were male and 6.4% were female showing male preponderance in study. At presentation, all patients presented with abdominal distension, pedal edema, signs of liver cell failure, ascites (100%). 32.8% presented with jaundice and clubbing, 23.2% GI bleeding, 19.2% with pallor, 15.2% with altered sensorium. 48% presented with ascites, 96.8% with portal hypertension, 8.8% with grade III hepatic encephalopathy, 13.6% with spontaneous bacterial peritonitis, 12.8% with hepatorenal syndrome, and 12.8% with coagulopathy.

A similar study by Azam MU et al., documented with mean age of the patients in this study to be 47.68 ± 12.89 years, with 80 (60.15%) males and 53 (39.85%) females. The average BMI was 23.20 ± 3.11 kg/m², and the average duration of decompensated chronic liver disease (DCLD) was 7.24 ± 4.12 years. [15] Similar to the present study, Hussain S et al. reported that ascites was the most prevalent complication, affecting 132 (82.5%) patients, followed by esophageal varices and hepatic encephalopathy. The study also found that the average age of affected individuals was 53.6 ± 11.28 years, with a mean BMI of 25.4 ± 8.31 kg/m². These findings provide further clinical context, emphasizing the common demographic and clinical characteristics of patients with decompensated chronic liver disease.[16]

In the present study, the majority of patients belonged to Child-Pugh Class B (71.2%), followed by Class C (21.6%) and Class A (7.2%). Additionally, Hepatitis B surface antigen (HbSAg) was positive in 6.4% of cases, while Hepatitis C virus (HCV) was detected in 0.8% of cases. Similarly, Hussain S et al. reported that Hepatitis B virus (HBV) was the leading cause of liver disease, observed in 95 (59.8%) cases, followed by alcohol-related liver disease in 40 (25%) cases. Their study found a mean MELD score of 13.9 ± 6.44, with most patients categorized under Child-Pugh Class C (80 cases, 50%). Regarding serum sodium levels, 79 (49.4%) patients had levels above 135 mmol/L, 42 (26.3%) had hyponatremia with sodium levels below 130 mmol/L, and 39 (24.4%) had sodium levels between 131 and 135 mmol/L. These findings align with the current study, emphasizing the prevalence of liver disease severity and associated biochemical parameters.[16]

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Endoscopy showing majority with esophageal varices (71.2%) followed by "esophageal varices with portal hypertension in 20.8% of the cases. There is significant negative association of serum sodium with MELD score (r-0.439, p<0.05) and with CPS score (r-0.433, p<0.05). also, the CPS score showing significant positive association with MELD score. With severity of liver disease, with increase in grade of CPC the mean sodium level showing the decreasing trend. Showing the lower mean sodium in CPC C grade compared to CPC A grade.(p<0.05)

Consistent with the findings of the present study, Godara SS et al. reported that the prevalence of hyponatremia varied according to the severity of hepatic encephalopathy (HE). Their study found that 30% of patients with grade I HE, 27.3% with grade II, 69.23% with grade III, and 81.25% with grade IV had hyponatremia, highlighting a significant association between sodium imbalance and worsening HE severity. Additionally, when analyzing the relationship between hyponatremia and the Child-Pugh classification, hyponatremia was present in 25% of patients with Child-Pugh Class A, 23.8% with Class B, and 88% with Class C. These findings further reinforce the strong correlation between declining serum sodium levels and worsening liver disease severity.[17] Present study documented with Class B (71.2%) followed by 21.6% in class C and 7.2% in class A CPC."

Similarly, Singh N et al. reported that 63.3% of patients exhibited hyponatremia. Their findings indicated that lower serum sodium levels were significantly associated with higher mean MELD and Child-Pugh scores. Furthermore, patients with serum sodium levels ≤130 mEq/L were more frequently categorized under Child-Pugh Class C compared to those with higher sodium levels, emphasizing the strong link between hyponatremia and advanced liver disease severity.[18] Similarly, the present study observed a higher incidence of hyponatremia in patients with severe MELD and CPS scores. El-Ghany H et al. reported that hyponatremia, with a cutoff value of >124 mmol/L, significantly predicted hepatic encephalopathy (HE) in patients classified under Group C. The study demonstrated an area under the curve (AUC) of 0.656 (P = 0.037), with a sensitivity of 83.33%, specificity of 46.67%, a positive predictive value (PPV) of 61.0%, and a negative predictive value (NPV) of 73.7%. Their findings highlight the role of both hyponatremia and hyperammonemia as crucial prognostic markers for HE and overall mortality in cirrhotic patients.[19] Azam MU et al., found that hyponatremia was found to be prevalent among individuals with DCLD, with age and BMI identified as significant risk factors. The study suggests that regular screening of serum sodium levels in DCLD patients is important to prevent complications such as encephalopathy, particularly in younger and underweight individuals.[15] The present study also highlights the importance of the sodium level and the patients outcome at hospital.

Also in study by Nareddy SR et al., "pateints with hyponatremia, 87.9% (29 patients) had a Child-Pugh class C classification, and the association between hyponatremia and Child-Pugh class C was statistically significant (OR 3.987; CI 1.240-12.818; p=0.029). Additionally, a positive correlation was found between low sodium levels (≤130 mEq/L) and complications such as spontaneous bacterial peritonitis (OR 4.667; CI 1.538-14.164; p=0.004) and hepatorenal syndrome (OR 5.357; CI 0.979-29.327; p=0.034). In conclusion, low sodium levels in cirrhosis are significantly correlated with disease severity, as well as the development of hepatorenal syndrome and spontaneous bacterial peritonitis."[20]

On assessment of the sodium level with outcome of patients, there is significant hyponatremia seen in cases with worst outcome as death (125.58±8.2) compared to the patients discharged (134.5±6.9). In similar Kumar H et al., found a significant difference in the complications associated with liver cirrhosis between the three serum sodium groups (≤130 mEq/L, 131-135 mEq/L, and ≥136 mEq/L). Specifically, portal hypertension, hepatic encephalopathy, and hepatorenal syndrome were significantly more prevalent in patients with lower sodium levels (p values of 0.0111, <0.0001, and 0.0140, respectively). Hyponatremia is common among cirrhotic individuals and worsens prognosis.[21]

Hyponatremia is more prevalent in decompensated chronic liver disease (DCLD) and is linked to an increased frequency of complications, including hepatic encephalopathy, hepatorenal syndrome, spontaneous bacterial peritonitis, and gastrointestinal bleeding. Lower serum sodium levels correlated with higher MELD and Child-Pugh scores, as well as increased

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mortality, highlighting an inverse relationship between serum sodium levels and disease severity.[2] Furthermore, hyponatremia is associated with increased morbidity and mortality, serving as a crucial prognostic factor both before and after liver transplantation in study by jang CM et al.[22]

Conclusion:

The study on 125 patients with decompensated chronic liver disease (DCLD) underscores the significance of serum sodium levels in assessing disease severity and prognosis. A strong negative correlation was found between sodium levels and MELD/CPS scores, with lower sodium levels indicating more severe liver dysfunction, particularly in CPC Class C patients. Hyponatremia was significantly linked to poor outcomes, as patients who succumbed had markedly lower sodium levels than those who survived ($p<0.05$). These findings highlight the potential of serum sodium as a prognostic marker in DCLD, emphasizing its role in early risk stratification and management.

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Conflict of interest: Nil

Reference:

1. John S, Thuluvath PJ. Hyponatremia in cirrhosis: pathophysiology and management. World J Gastroenterol. 2015;21(11):3197–205.
2. Shetty VN, Nayak UB. A Study of Serum Sodium Levels in Decompensated Chronic Liver Disease and its Clinical Significance: A Study in AJ Institute of Medical Sciences, Mangalore. Age (Omaha). 2023;97(49.69):10–26.
3. Channakeshava SP. A Study of Serum Sodium Levels in Decompensated Chronic Liver Disease and Its Clinical Significance. Rajiv Gandhi University of Health Sciences (India); 2019.
4. Premkumar M, Anand AC. Overview of Complications in Cirrhosis. J Clin Exp Hepatol. 2022;12(4):1150–74.
5. Castello L, Pirisi M, Sainaghi PP, Bartoli E. Hyponatremia in liver cirrhosis: pathophysiological principles of management. Dig Liver Dis. 2005;37(2):73–81.
6. Attar B. Approach to hyponatremia in cirrhosis. Clin Liver Dis. 2019;13(4):98–102.
7. Kaplan M, Ateş I, Gökcan H, Kayhan MA, Kaçar S, Akpınar MY, et al. Prognostic utility of hypokalemia in cirrhotic patients. Acta Gastroenterol Belg. 2018;81.
8. Arzhan S, Lew SQ, Ing TS, Tzamaloukas AH, Unruh ML. Dysnatremias in Chronic Kidney Disease: Pathophysiology, Manifestations, and Treatment. Front Med. 2021;8:769287.
9. Cosby RL, Yee B, Schrier RW. New classification with prognostic value in cirrhotic patients. Miner Electrolyte Metab. 1989;15(5):261–6.
10. 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(1):46–52.
11. Llach J, Ginès P, Arroyo V, Rimola A, Titó L, Badalamenti S, et al. Prognostic value of arterial pressure, endogenous vasoactive systems, and renal function in cirrhotic patients admitted to the hospital for the treatment of ascites. Gastroenterology. 1988;94(2):482–7.
12. Angeli P, Wong F, Watson H, Ginès P, Investigators C. Hyponatremia in cirrhosis: results of a patient population survey. Hepatology. 2006;44(6):1535–42.
13. Cárdenas A, Ginès P. Predicting Mortality in Cirrhosis — Serum Sodium Helps. N Engl J Med. 2008;359(10):1060–2.
14. Younas A, Riaz J, Chughtai T, Maqsood H, Saim M, Qazi S, et al. Hyponatremia and its correlation with hepatic encephalopathy and severity of liver disease. Cureus. 2021;13(2):e13175.
15. 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(9):e68907.

16. Hussain S, Yaseen M, Mushtaq K. Frequency of Complications by Serum Sodium Level Among Patients with Liver Cirrhosis. Pakistan J Med Heal Sci. 2022;16(03):1215.

17. Godara SS, Gupta N, Singh S, Kapila S. To study serum sodium levels in patients admitted with hepatic encephalopathy and its value as a prognostic marker. Int J Life Sci Biotechnol Pharma Res. 2023;12(2):1926–30.

18. Singh N, Chamoli A. An observational cross-sectional study of the serum sodium levels and their association with severity in chronic liver disease patients in a tertiary care center in Haryana. Asian J Med Sci. 2022;13(7):97–102.

19. Abd El-Ghany H, Gawish H, Al Mukhtar AA, Shaker S. Hyponatremia as A Prognostic Indicator for Hepatic Encephalopathy in Cirrhotic Patients. Zagazig Univ Med J. 2023 Oct 2;30(4):1226–44.

20. Nareddy SR, Aroor ARAO, Bhat A. Clinical Significance of Serum Sodium Levels in Liver Cirrhosis: A Crosssectional Observational Study. J Clin Diagnostic Res. 2020;14(11):1–6.

21. Kumar H, Singh SK, Kishore K. Study of Hyponatremia in Cirrhosis of Liver and Its Prognostic Value. Eur J Cardiovasc Med. 2023;13(2).

22. Jang C, Jung YK. Hyponatremia in liver cirrhosis. Korean J Gastroenterol. 2018;72(2):74–8.