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Study of Serum Sodium Levels in Decompensated Chronic Liver Disease

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ABSTRACT

Decompensated Chronic Liver Disease is associated with disturbances in regulation of water balance leading on to abnormalities in serum sodium. Dilutional Hyponatremia due to impaired free water clearance is the most common dysnatremia while hypernatremia due to cathartic use has also been reported in few studies. The aim of this study was to study the serum sodium levels in patients with DCLD and to establish its significance.

Methods: *Data were collected from 97 patients admitted in medical wards. Patients were divided into groups based on serum sodium levels and the relevant parameters analyzed among the groups.*

Results: *Among 97 patients, 42 (43.30%)had serum sodium levels ≥136 mEq/L, while 32(32.99%)had serum sodium levels between 131 and 135 mEq/L. 23(23.71%) patients had serum sodium level ≤130. No patients had serum sodium levels greater than 145. Serum sodium levels was associated strongly with the severity of liver disease as assessed by Child Pugh and MELD score. Patients with serum sodium less than 130 mEq/L had increased frequency of complications than those with ≥136 mEq/L. Patient with serum sodium levels ≤130 had increased mortality(30.4%; p value- 0.002)*

Conclusion: *Hyponatremia is more common in DCLD and low serum sodium levels are associated with increased frequency of complications of DCLD, increased MELD, CPS score and mortality indicating the inverse relationship between serum sodium levels and severity of the disease.*

KEYWORDS : Hyponatremia, Hypernatremia, Decompensated Chronic Liver Disease.

Hyponatremia is a frequent complication in patients with decompensated liver disease. It occurs due to the impaired free water clearance by renal tubules that leads to disproportionate retention of water when compared with sodium. This leads to reduction in serum sodium and hypoosmolality. Although hyponatremia in decompensated liver disease was described 50 years ago, interest in this area increased when studies done in 1980s indicated that hyponatremia is significant prognostic indicator.

Recent studies also showed that presence of hyponatremia is associated not only with poor outcome in patients who has not undergone transplants but also in post transplant patients.

Although hyponatremia in general population is defined as the concentration of serum sodium less than 135 mEq/L, in patients with chronic liver disease and ascites, it is defined as serum sodium less than 130 mEq/L. Still, patients with serum sodium levels between 130 and 135 have pathogenic and clinical features almost similar to those with patients who have levels below 130 mEq/L. 21.6% of patients with DCLD have serum sodium less than 130 and 49.4% have levels less than 135 mEq/L.

TYPES OF HYPONATREMIA:

Patients with decompensated liver disease may develop either hypervolemic hyponatremia or hypovolemic hyponatremia.

Hypervolemic Or Dilutional Hyponatremia:

This is by far the most common type. It occurs in patients with increased extracellular fluid and edema. Hyponatremia here is due to impaired ability of kidney to excrete free water that results in disproportionate increase in water compared to sodium. Renal impairment is frequent but not always present with this type.

Hypovolemic Hyponatremia:

It is less common and occurs due to loss of fluid, mainly from kidney due to enhanced diuresis as a result of diuretic drugs or from gastrointestinal tract. It is associated with reduction in plasma volume, absence of ascites or edema, signs of dehydration and pre-renal renal failure. These patients show an improvement of sodium values after administration of normal saline or by increasing quantity of sodium in the diet.

Both types can be differentiated on the basis of volume status. In hypervolemic hyponatremia, the effective arterial blood volume is reduced in spite of the increase in absolute plasma volume owing to the marked dilatation of the arterial circulation.

PATHOGENESIS OF HYPONATREMIA IN DCLD:

Patients with decompensated liver disease and ascites have impairment in the ability of kidney to excrete solute free water[1,2]. In some patients, this impairment is only moderate and detected by measuring urine volume following a water load. These patients can eliminate water normally and maintain sodium concentration within normal limits as long as their intake of water is within normal range. When the intake of water is increased, these patients may develop hyponatremia and hypoosmolality. Figure 1 shows the mechanisms of renal water handling in these patients causing hyponatremia.

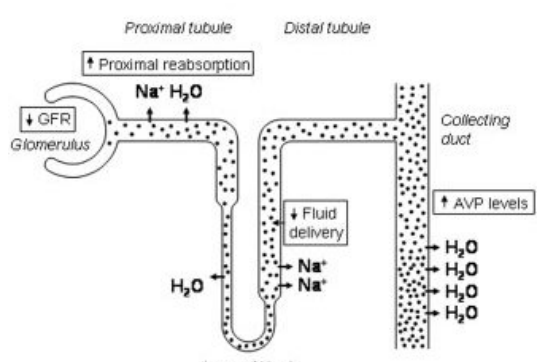


FIGURE 1: RENAL WATER HANDLING IN CIRRHOSIS

The important determinant of hyponatremia is increased secretion of AVP as a result of non-osmotic stimulation following circulatory changes in patients with decompensated liver disease.

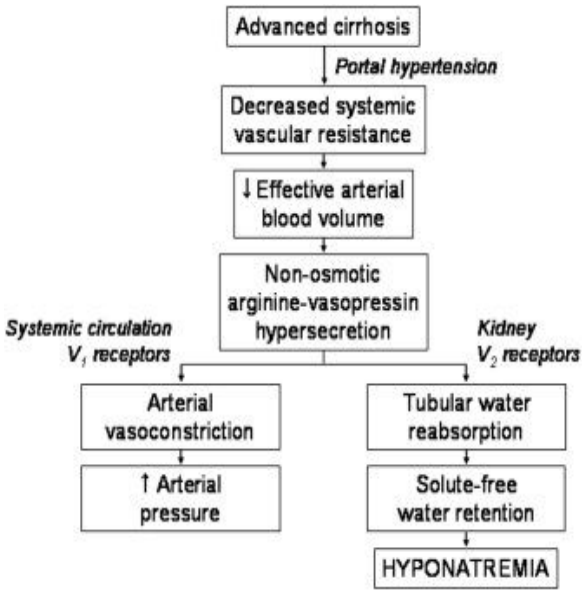


FIGURE 2: MECHANISM OF AVP SECRETION AND ITS EFFECTS

Various studies have provided evidence that AVP is a major factor responsible for water retention in cirrhosis[3].

MECHANISM OF RETENTION OF WATER AND DILUTIONAL HYPONATREMIA:

AVP is metabolized in liver and kidney. In patients with altered liver function due to chronic liver disease, metabolic clearance rate of AVP is decreased and its level increases. This alone does not explain the whole process of dilutional hyponatremia, as the increase in AVP levels should exert a negative feedback on its release if osmotic factor is alone responsible for the regulation of AVP. The decreased metabolism of AVP along with non osmotic release of AVP, reduced renal prostaglandins and decreased delivery of filtrate to distal tubule leads to hyponatremia. The pathogenesis of dilutional hyponatremia is shown in figure 3.

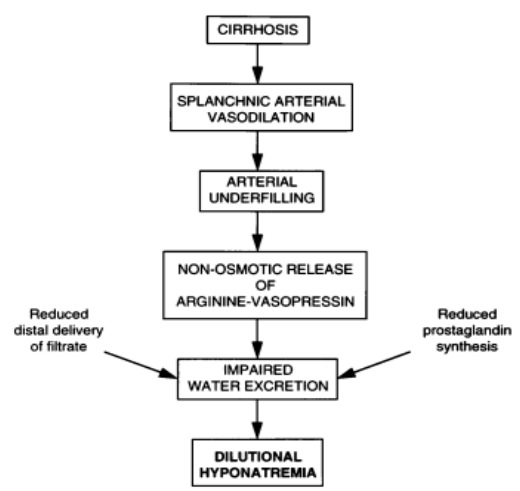


FIGURE 3: PATHOGENESIS OF DILUTIONAL HYPONATREMIA

PROGNOSTIC SCORES:

The prognostic scores in decompensated liver disease are used for various reasons. They help in identifying patients at risk for developing complications, to predict the risks involved in various procedure such as porto-systemic shunts, to prioritize the patients waiting for Orthotopic Liver Transplantation(OLT).

TABLE 1: MODIFIED CPS

VARIABLE	POINTS		
	1	2	3
Serum Bilirubin(mg/dl)	<2	2-3	>3
Serum Albumin(mg/dl)	>3.5	2.8-3.5	<2.8
INR	<1.7	1.7-2.2	>2.2
Ascites	None	Mild	Moderate to Severe
Encephalopathy	None	Grade I-II	Grade III-IV
Class A- 5-6 points; Class B- 7-9 points; Class C- 10-15 points			

MELD SCORE: (The Model for End-stage Liver Disease)

- MELD = 3.78[$\log_e$ serum bilirubin (mg/dl)] + 11.2[$\log_e$ INR] + 9.57[$\log_e$ serum creatinine (mg/dl)] + 6.43
- POINTS TO BE REMEMBERED WHILE CALCULATING THE SCORE:
- The score ranges from 6-40 and any value more than 40 is taken as 40.
- It is used in patients 12 years and older.
- Maximum value for creatinine is 4. If patient is dialysed twice in the preceding week, creatinine value is taken as 4.
- If the value of a variable is less than one, it is taken as one.(Serum bilirubin value of 0.9 is taken as 1)

SERUM SODIUM AS A PROGNOSTIC MARKER:

The prognostic effect of serum sodium has been studied in patients with decompensated liver disease. A large study done on patients admitted for cirrhosis, has shown that the prevalence of hyponatremia to be 29.8%.[4] Low serum sodium levels were found to be an indicator of poor prognosis and short term in-hospital mortality. Low serum sodium levels were not found to be an independent predictor of mortality when compared with CPS.[5] Biggins et al showed that the ability of MELD score to predict three month waiting list mortality improved when serum sodium was added to it.[6] The mortality risk for patients with decompensated liver disease was found to be higher in patients with hyponatremia irrespective of the disease severity.[7]

HYPERNATREMIA IN DCLD:

Although hypernatremia is less common than hyponatremia in decompensated liver disease, Sanford E.Warren et al found that patients with hypernatremia and decompensated liver disease had a mortality of 87% and attributed the cause of hypernatremia to decreased water intake due to encephalopathy and osmotic cathartics usage.[8]

AIMS AND OBJECTIVES:

To study serum sodium levels in patients with Decompensated Chronic Liver Disease and establish its significance.

MATERIAL AND METHODS

SOURCE OF STUDY:

The study will be conducted on consecutive patients admitted with DCLD in Medical Wards (Male and Female) in Government Vellore Medical College and Hospital during the study period of one year from August 2014- July 2015.

METHOD OF COLLECTION OF DATA:

Ethical Committee clearance obtained from Institution. Informed consent was obtained from the patients enrolled in the study. The data of the patients were collected using a proforma. The first section of the proforma contains patient's demographic profile with detailed history. The second section contains detailed clinical examination that will be carried out at the time of admission. The third section contains investigations that were done to aid the diagnosis and the serum sodium level.

Patients were selected based on history, examination, laboratory investigations and imaging suggestive of the diagnosis of Decompensated Chronic Liver Disease. The presence of various complications and the outcome of the patients were monitored. The severity of the disease was calculated using MELD score and Child Pugh Score. Ascites was classified in to three grades: Grade I- presence on examination not clear, but observed in imaging; Grade II- easily made out examination and palpation; Grade III- severe abdominal distension requiring large volume paracentesis. Hepatic Encephalopathy was graded using West Haven Criteria.

INCLUSION CRITERIA:
All patients with Decompensated Chronic Liver Disease diagnosed by examination, laboratory investigations and radiological imaging.

EXCLUSION CRITERIA:
- Patients with cardiac failure
- Patients with chronic kidney disease
- Patients on drugs such as SSRIs, TCA, MAO inhibitors, cytotoxic drugs etc.,

STATISTICS:
The collected data were entered in a Microsoft Excel Sheet. Graphs and tables were generated using Microsoft Word and Microsoft Excel. Statistical analysis were done using medcalc 15.8, Minitab 17, IBM SPSS 22. Quantitative data was analysed using Mean, Median, Mode and Standard Deviation(SD). Qualitative data was analysed using Chi Square Test, One way ANOVA and Fisher's test. Difference between two variables is considered significant when 'p' value is less than 0.05.

RESULTS
Data were collected from 97 patients admitted in our hospital. The mean age of the patients was 49.69 years with a range of 28-70 years. Out of the 97 patients, 91(93.81%) were males and 6(6.19%) were females. Table 2 shows the demographic details and causes of DCLD.

Sl.No	Parameter	No Of Patients	% Of Patients	Mean	SD
1.	Age	97		49.69	10.26
2.	Gender				
	Male	91	93.81%		
	Female	6	6.19%		
3.	Cause of cirrhosis				
	Alcohol	89	91.75%		
	HBV	7	7.22%		
	HCV	1	1.03%		
	Other	0	0.00%		
4.	MELD score			13.54	5.50
5.	Serum sodium			134.18	5.02
	1) ≤130 meq/L	23	23.71%		
	2) 131-135 meq/L	32	32.99%		
	3) ≥136 meq/L	42	43.30%		
	4)>145	0	0%		

TABLE 2: DEMOGRAPHY DETAILS
Alcoholic liver disease was the commonest cause of DCLD in this study accounting for 91.75% while chronic hepatitis B and hepatitis C was found to be the causative factor in 7.22% and 1.03% respectively. The mean concentration of sodium of all patients was 134.18 with a range of 120-144. Based on the serum sodium levels, 23.71% of patients had serum sodium levels less than or equal to 130. 32.99% of patients had serum sodium levels between 131 and 135, while 43.3% of patients had serum sodium levels ≥136. No patients presented with serum sodium greater than 145. The mean MELD score was found to be 13.54 with a range of 7.5- 33.7 .

Ratio of male and female in this study was 15.2:1.
Patients were classified into three groups based on the serum sodium level to assess the association between serum sodium levels and patient characteristics, complications and severity of disease as calculated by MELD score and CPS. Those with serum sodium levels less than or equal to 130 formed one group while those with serum sodium levels between 131-135 and those with ≥136 were the other two groups. Mean age of patients with sodium levels ≤130 was 50.50±11.08, while in those with serum sodium levels 131-135 and ≥136 were 49.11±11.49 and 50.82±10.67 respectively. No statistical difference was found among the three groups. (p value - 0.877).

TABLE 3: CHARACTERISTICS OF PATIENTS ACCORDING TO SERUM SODIUM CONCENTRATION

Sl No	PARAMETERS	≤130 meq/L	131-135 meq/L	≥136 meq/L	P value
		N=23	N=32	N=42	
1	Age(years) (Mean +SD)	50.50±11.08	49.11 ±11.49	50.82±10.67	0.877*
2	SEX: Male Female	22 1	31 1	38 4	0.479$
3	CAUSE OF DCLD: Alcohol HBV HCV Others	20 3 0 0	31 1 0 0	38 3 1 0	0.376$
4	MELD score (Mean ±SD)	18.89±6.70	13.17±4.40	10.90±2.95	<0.0001*
5	Child-Pugh Score	10.00 ±1.86	8.53±1.27	7.48 ±1.33	<0.0001*
6	Child Pugh Class Class A Class B Class C	 0 11 12	 1 24 7	 9 29 4	<0.0001$

*- calculated using one way annova; $- calculated using chi square test.

Serum sodium levels had a strong association with severity of disease as calculated by Child Pugh Class. Among those with serum sodium levels ≤130, 11 belonged to class B and 12 belonged to class C. Among patients with serum sodium levels between 131-135, 1 belonged to class A, 24 belonged to class B and 7 belonged to class C. Among patients with serum sodium levels ≥136, 9 belonged to class A, 29 belonged to class B and 4 belonged to class C.(p value <0.0001)

Patients with serum sodium levels ≤130 had a mean MELD score of 18.89±6.70, while those with levels between 131-135 and ≥136 had mean scores of 13.17±4.40 and 10.90±2.95 respectively. The difference in MELD scores among the three groups was statistically significant. (p value <0.0001)

TABLE 4: FREQUENCY OF COMPLICATIONS BY SERUM SODIUM CONCENTRATION

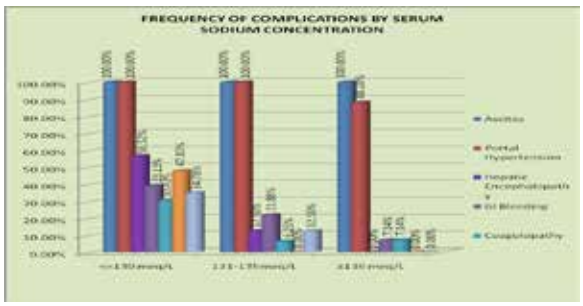
Sl No.	COMPLICATIONS	≤130 meq/L Number (%)	131-135meq/L Number(%)	≥136 meq/L Number (%)	p value*
1	Ascites	23(100%)	32(100%)	42(100%)	0.51
2	Portal Hypertension	23(100%)	32(100%)	37(88.10%)	0.031
3	Hepatic Encephalopathy	13(56.52%)	4(12.50%)	0(0%)	<0.0001
4	GI Bleeding	9(39.13%)	7(21.88%)	3(7.14%)	0.0074
5	Coagulopathy	7(30.43%)	2(6.25%)	3(7.14%)	0.0106
6	Hepatorenal Syndrome	11(47.83%)	0(0%)	0(0%)	<0.0001
7	SBP	8(34.78%)	4(12.50%)	0(0%)	0.0002

***- calculated by chi square test.**

There was significant difference in the occurence of complications of DCLD such as Portal Hypertension (p value- 0.031), Hepatic Encephalopathy(p value <0.0001), GI Bleeding(p valuve- 0.0074), coagulopathy(p value- 0.0106), hepatorenal syndrome(p value<0.0001), SBP(p value- 0.0002)among the three groups. There was no significant difference in the presence of ascites among the three groups(p value- 0.51)

Figure 4 shows the increased occurrence of complications in patients with lower serum sodium levels.

FIGURE 4: FREQUENCY OF COMPLICATIONS ACCORDING TO SERUM SODIUM CONCENTRATION



When compared to patients with serum sodium levels ≥136, patients with serum sodium levels ≤130 had a significantly increased risk for complications: 109.29 for Hepatic Encephalopathy(p value <0.0001), 8.36 for GI bleeding(p value = 0.0026), 5.69 for Coagulopathy(p value = 0.0265), 78.2 for Hepatorenal syndrome(p value <0.0001) and 46.61 for SBP(p value <0.0001). Ascites and portal hypertension did not have statistical difference and increased risk.(p value – 1, 0.152 respectively)

When compared to patients with serum sodium levels ≥136, patients with serum sodium levels between 131 and 135 had a significantly increased risk for complications: 13.42 for Hepatic Encephalopathy(p value- 0.0312); 1.3077 for SBP (p value- 0.0312). Other complications did not have statistical difference among the two groups.

TABLE 5: COMPARISION OF COMPLICATIONS ACCORDING TO SERUM SODIUM CONCENTRATION

Sl No	COMPLICATION	≤130 meq/L N=23 ODDS RATIO (95%CI)	P Value*	131-135meq/L N=32 ODDS RATIO (95% CI)	P value*
1	Ascites	0.5529 (0.0106 to 28.7818)	1	0.7647 (0.0148 to 39.5772)	1
2	Portal Hypertension	6.8933 (0.3642 to 130.4817)	0.152	9.5333 (0.5075 to 179.068)	0.0653
3	Hepatic Encephalopathy	109.2857 (5.9987 to 1990.979)	<0.0001	13.4211 (0.6954 to 259.0198)	0.0312
4	GI Bleeding	8.3571 (1.9755 to 35.3548)	0.0026	3.6400 (0.8601 to 15.4055)	0.0900
5	Coagulopathy	5.6875 (1.3046 to 24.7955)	0.0265	0.8667 (0.1361 to 5.5199)	1
6	Hepatorenal Syndrome	78.2000 (4.2991 to 1422.437)	<0.0001	1.3077 (0.0253 to 67.6793)	1
7	SBP	46.6129 (2.5370 to 856.4415)	<0.0001	1.3077 (0.0253 to 67.6793)	0.0312

***- calculated by fisher's test.**

When compared to patients with serum sodium levels ≥136, patients with serum sodium levels ≤130 had a significantly increased risk for complications: 109.29 for Hepatic Encephalopathy(p value <0.0001), 8.36 for GI bleeding(p value = 0.0026), 5.69 for Coagulopathy(p value = 0.0265), 78.2 for Hepatorenal syndrome(p value <0.0001) and 46.61 for SBP(p value <0.0001). Ascites and portal hypertension did not have statistical difference and increased risk.(p value – 1, 0.152 respectively)

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TABLE 6: MORTALITY ACCORDING TO SERUM SODIUM CONCENTRATION

	≤130 meq/L(N=23)	131-135 meq/L(N=32)	≥136 meq/L(N=42)	P value
Mortality	7(30.4%)	2(6.25%)	0(0%)	0.0002

Among 23 patients with serum sodium levels ≤130, 7 patients(30.4%) died. Among 32 patients with serum sodium levels between 131 and 135, 2 patients(6.25%) died. There were no deaths among patients with sodium levels ≥136. The difference in mortality among these three groups was statistically significant. (p value- 0.0002)

DISCUSSION

A significant proportion of patients with DCLD have abnormal serum sodium concentration. Hyponatremia is the most common occurrence in our study. No patients presented with serum sodium levels greater than 145.

56.7% of patients had serum sodium levels less than 135, while 23.71% patients had serum sodium levels than 130. Serum sodium levels less than 120 were uncommon.

TABLE 7: COMPARISON OF VARIOUS STUDIES SHOWING DISTRIBUTION OF PATIENTS ACCORDING TO SERUM SODIUM LEVELS

STUDIES	DISTRIBUTION OF PATIENTS		
	≤130 mEq/L	131-135 mEq/L	≥136 mEq/L
PRESENT STUDY	23.71%	32.99%	43.3%
ANGELI P ET AL[9]	21.6%	27.8%	50.6%
JONG HOON KIM ET AL[10]	27.1%	20.8%	52.1%
SHAIKH ET AL[11]	26.7%	24.9%	48.4%
BORRONI ET AL[4]	29.8%		

The results of the present study extend the observations made by the above mentioned studies that decompensated liver disease is associated with abnormal serum sodium concentration. It also shows that hyponatremia is the common abnormality with more than half of the patients having serum sodium levels less than 135 mEq/L.

Various studies have established that lower sodium levels were associated with ascites that are difficult to manage with diuretics and requiring frequent large volume paracentesis. Arroyo et al noted that patients having serum sodium less than 130 mEq/L had a relatively low GFR and subsequently decreased free water clearance. These patients responded poorly to diuretics when compared with those who had sodium levels more than 130 mEq/L. Angeli P et al and Bernardi et al[12] also found that poorer response to diuretics was associated with lower serum sodium concentration compared to patients who showed response to diuretics. The present study also found that patients with lower sodium levels had higher grade of ascites.

In the present study, patients with serum sodium levels ≤130 mEq/L had increased frequency of hepatic encephalopathy compared to the other two groups.

TABLE 8: COMPARISION OF STUDIES SHOWING ASSOCIATION BETWEEN SERUM SODIUM CONCENTRATION AND HEPATIC ENCEPHALOPATHY.

STUDIES	FREQUENCY OF HEPATIC ENCEPHALOPATHY		
	≤130 mEq/L	131-135 mEq/L	≥136 mEq/L
PRESENT STUDY	56.52%	12.50%	0.00%
ANGELI P ET AL	38%	24%	15%
JONG HOON KIM ET AL	43.1%	35.8%	24.4%
SHAIKH ET AL	25.8%		

In present study, patients with serum sodium levels less than or equal to 130 mEq/L had increased frequency of hepatorenal syndrome compared to other two groups.

TABLE 9: COMPARISION OF STUDIES SHOWING ASSOCIATION BETWEEN SERUM SODIUM CONCENTRATION AND HEPATORENAL SYNDROME.

STUDIES	FREQUENCY OF HEPATORENAL SYNDROME		
	≤130 mEq/L	131-135 mEq/L	≥136 mEq/L
PRESENT STUDY	47.83%	0%	0%
ANGELI P ET AL	17%	10%	6%
JONG HOON KIM ET AL	3.9%	2.5%	3%

Our study shows that 34.78% of patients with serum sodium levels ≤130 mEq/L had SBP compared to 12.5% of patients with serum sodium levels between 131-135 mEq/L.

TABLE 10: COMPARISION OF STUDIES SHOWING ASSOCIATION BETWEEN SERUM SODIUM CONCENTRATION AND SBP

STUDIES	FREQUENCY OF SBP		
	≤130 mEq/L	131-135 mEq/L	≥136 mEq/L
PRESENT STUDY	34.78%	12.5%	0%
JONG HOON KIM ET AL	33.3%	30.7%	16.3%

Angeli P et al, Jong Hoon Kim et al and Shaikh et al found no association between GI bleeding and sodium levels. The present study showed increased frequency of GI bleeding in patients with low sodium levels.

Sanford E.Warren et al reported that 15 out of 25 patients with decompensated liver disease had hypernatremia in their study. The present study had no patients with serum sodium levels more than 145 mEq/L.

Jong Hoon Kim et al found that lower sodium levels were associated with increased MELD score and Child Pugh score. This indicates that lower serum sodium levels were associated with severe disease.

The present study also showed that patients with sodium levels ≤130 mEq/L had higher MELD score and Child Pugh Score compared to other two groups.

TABLE 11: COMPARISION OF STUDIES SHOWING ASSOCIATION BETWEEN SERUM SODIUM CONCENTRATION AND MELD SCORE

STUDIES	MELD SCORE		
	≤130 mEq/L	131-135 mEq/L	≥136 mEq/L
PRESENT STUDY	18.89±6.70	13.17±4.40	10.90±2.95
JONG HOON KIM ET AL	17.20±5.10	16.30±5.20	13.90± 4.60

TABLE 12: COMPARISION OF STUDIES SHOWING ASSOCIATION BETWEEN SERUM SODIUM CONCENTRATION AND CPS

STUDIES	CHILD PUGH SCORE(CPS)		
	≤130 mEq/L	131-135 mEq/L	≥136 mEq/L
PRESENT STUDY	10.00 ±1.86	8.53±1.27	7.48 ±1.33
JONG HOON KIM ET AL	10.50±1.60	9.80±1.70	8.10± 1.60

The present study also shows increased mortality among patients with lower sodium levels.

TABLE 13: MORTALITY AND SERUM SODIUM

SERUM SODIUM	MORTALITY
≤130 mEq/L	30.4%
131-135 mEq/L	6.25%
≥136 mEq/L	0%

SUMMARY OF RESULTS:

- The study was conducted on 97 patients admitted with DCLD in medical wards in Government Vellore Medical College.
- Alcohol is the most common etiology of DCLD in this study followed by Hepatitis B.
- Hyponatremia is the most common sodium abnormality(56.7%). No patients presented with hypernatremia.
- Patients were divided into three groups based on serum sodium; those with serum sodium levels ≥136 mEq/L comprised one group while those with 131-135 mEq/L and ≤130 mEq/L formed the other two groups.
- Serum sodium level was not associated with gender or etiology of DCLD.
- There was significant difference in occurrence of complications such as Portal Hypertension(p value – 0.031), Hepatic Encephalopathy(p value <0.0001), Hepatorenal Syndrome(p value <0.0001), Spontaneous Bacterial Peritonitis(p value-0.0002), Coagulopathy(p value- 0.0106). Increased frequency of complications was noted among patients with lower serum sodium levels.
- There was significant difference in severity scores such as MELD and CPS among the three groups.
- There was significant difference in mortality among the three groups(p value-0.0002). Patients with lower sodium levels had increased mortality.

CONCLUSION:

Decompensated Chronic Liver Disease is associated with abnormal serum sodium concentration. Hyponatremia is the most common abnormality in this study. Age, gender and cause of DCLD did not have any association with serum sodium levels. Serum sodium levels less than 135 mEq/L is associated with increased frequency of complications such as Hepatic Encephalopathy, Hepatorenal Syndrome, Spontaneous Bacterial Peritonitis and GI Bleeding when compared to patients with serum sodium levels ≥136 mEq/L. Patients with serum sodium concentration less than 130 mEq/L are the most affected. Lower serum sodium levels are associated with increased MELD score, increased CPS score and increased mortality indicating the inverse relationship between serum sodium levels and the severity of disease. Thus patients with decreased serum sodium levels should be considered a high risk population because of the increased frequency of complications and mortality.

CONFLICT OF INTEREST: NIL

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