International Journal of Life Sciences, Biotechnology and Pharma Research Vol. 13, No. 10, October 2024 Online ISSN: 2250-3137
Print ISSN: 2977-0122

DOI: 10.69605/ijlbpr_13.10.2024.146

ORIGINAL RESEARCH

To measure and correlate the serum sodium levels in patients of chronic liver disease with hepatic encephalopathy

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Received Date: 21 August, 2024 Accepted Date: 25 September, 2024

ABSTRACT

Background: Hyponatremia is the most common electrolyte abnormality seen in patients of Chronic Liver Disease(CLD).Hyponatremia is considered as a potential risk factor for the development and worsening of Hepatic Encephalopathy.This study is conducted to find association between serum sodium levels and stages of Hepatic Encephalopathy.To assess Hyponatremia as a prognostic marker in patients of Chronic Liver Disease. **Objectives:** To measure serum sodium values in patients of Decompensated Chronic Liver Diseaseover the period of 14 days and determine its association with various grades of Hepatic Encephalopathy. **Study design:** Case control study conducted in Guru Nanak Dev Hospital (GNDH), Amritsar from2023-2024. **Materials and methods:** Study was conducted on 100 patients(cases) of CLD with overt Hepatic Encephalopathy admitted in GNDH and 100 patients(controls) of CLD without Hepatic Encephalopathy presenting with other complications and patients were studied for duration of 14 days.Patients with acute intracerebral infarct or haemorrhage, with hepatorenal syndrome, patientswith sepsis and positive blood cultures and history of drug intake such as diuretic, narcotics and valproic acid were excluded. **Results:** Out of 100 cases 91 were males and 9 were females with mean age 49.79$\pm$13.52 years. Outof 100 controls 74 were males and 26 were females with mean age of 49.07$\pm$11.34 years. Alcohol was the most common cause of cirrhosis in both cases and control. Patients were divided into 4 groups based on serum sodium levels into:<120mEq/l,121-130mEq/l,131-135mEq/l and >135mEq/l. Cases showed statistical correlation between hyponatremia and Child Pugh Class (p value<0.05). Cases also showed positive correlation between Serum sodium levels and MELD score (p value<0.05). From day 1 to day 14 with improving serum sodium levels, patients shifted from higher grades tolower grades of Hepatic Encephalopathy with p value 0.001. Study showed correlation of hyponatremia with mortality in these patients with p value<0.05. **Conclusion:** Serum sodium levels may be used as a prognostic marker in patients of Hepatic Encephalopathy. Lower levels of serum sodium correlated with increased MELD score andincreased Child Pugh Class, indicating inverse relationship between hyponatremia and severity of disease. Thus, patients with hyponatremia should be considered a high-risk population because of increased frequency of complications and mortality.

Keywords: Hepatic encephalopathy, Decompensated Liver Disease, Hyponatremia

INTRODUCTION

Chronic liver disease is marked by persistent inflammation or damage to the liver,leading to reduced liver function. In its advanced stages, the liver undergoes cirrhosis, a condition characterized by irreversible changes in liver structure due to fibrosis, scarring, and the formation of abnormal nodules. Recent estimates indicate that around 5.5 million individuals globally are affected by Chronic Liver Disease (CLD), with up to 40,000 deaths attributed to the disease or its complications. In the United States, CLD ranks as the twelfth leading cause of death. Clinically, we can divide cirrhosis as compensated or decompensated. Patients of compensated cirrhosis are usually diagnosed on routine investigations or on clinical examination, as these patients are typically asymptomatic. Patients of decompensated cirrhosis presents with symptoms of Ascites, hepatic encephalopathy, bleeding varices or jaundice in combination or either alone.

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Hepatic encephalopathy (HE) is characterized by alterations in mental status and cognitive function in individuals with liver failure, representing a reversible yet serious complication of CLD. In patients with cirrhosis, encephalopathy often manifests due to various triggering factors such as infections, hyponatremia, high protein intake, constipation, dehydration, diuretics, vomiting, anemia, and gastrointestinal bleeding. HE can be classified into covert hepatic encephalopathy (minimal hepatic encephalopathy and West Haven grade 1 HE) and overt hepatic encephalopathy (West Haven grade 2-4). In CLD patients the impaired liver function leads to an inability to effectively filter natural toxins like ammonia from the body. As ammonia accumulates in the bloodstream, it can cross the blood- brain barrier and affect brain function. Excess ammonia in the brain results in the accumulation of glutamine within astrocytes. Glutamine is an osmotically active substance, causing astrocyte swelling and resulting in cerebral edema. Besides elevated ammonia levels, factors such as hyponatremia, inflammatory cytokines, and endogenous benzodiazepines also contribute to astrocyte swelling.[1] Hyponatremia is a frequent electrolyte abnormality observed in patients with DCLD, and its severity correlates with the severity of liver disease itself. In individuals with CLD, serum sodium levels indicating hyponatremia become clinically significant when they drop below 130 mEq/L, particularly in cirrhotic patients. The most common type observed is hypervolemic (dilutional) hyponatremia, which arises from impaired free water clearance by the renal tubules, resulting in an excessive retention of water relative to sodium. Hypoosmolar hyponatremia, characterised by decreased serum sodium levels and resulting in astrocyte swelling due to water movement into the astrocytes, poses a significant risk for overt hepatic encephalopathy (HE). Hyponatremia is a major risk factor in cirrhotic patients with hepatic encephalopathy, underscoring the prognostic value of monitoring serum sodium levels closely in managing these patients. Hyponatremia have been linked to higher mortality rates and poorer clinical outcomes, prompting the incorporation of sodium levels into the Model for End-Stage Liver Disease (MELD) score for prioritising transplantation.[2]patients of DCLD and hyponatremia were found to have higher mortality risk regardless of the severity of their underlying liver disease.[3]

AIM

1. To measure serum sodium values in decompensated liver disease patients during variousphases of hepatic encephalopathy.
2. To determine the association of serum sodium levels with various stages of hepatic encephalopathy.

MATERIALS AND METHODS

This case control study was conducted on 100 patients of chronic liver disease with Hepatic Encephalopathy (cases) and 100 patients of chronic liver disease presenting with complications other than Hepatic Encephalopathy (controls) from 2022 to 2023.The study was conducted after approval from institutional Ethics Committee, Government Medical College, Amritsar. Written and informed consent was taken from every patient.

Sample size: Cases (100 patients) admitted in Hospital Ward with diagnosis of CLD with onset of Hepatic Encephalopathy.

Control (100 patients) previously diagnosed CLD patients attending OPD or getting admitted for complications other than Hepatic Encephalopathy.

Inclusion Criteria

- In-patients admitted in the hospital ward with diagnosis of CLD with onset of hepatic encephalopathy Grade 2-4 (as per West Haven criteria).
- Control patients will be previously diagnosed chronic liver disease patients attending the OPD or getting admitted for complications other than hepatic encephalopathy.

Exclusion Criteria

- Patients with acute intracerebral events- infarct or haemorrhage.
- Patients in sepsis with positive blood cultures that may contribute to theencephalopathy.
- H/o drug intakes such as diuretics, narcotics, valproic acid.
- Patients developing hepatorenal syndrome

Methodology and analysis

The patients diagnosed with chronic liver disease with hepatic encephalopathy were classified as per West Haven criteria and included in the study.

West Haven Grading[4]

GRADE	INTELLECTUAL FUNCTION	NEUROMUSCULAR FUNCTION
0	Normal	Normal
Minimal	Normal examination findings; subtle changes in work or driving	Minor abnormalities of visual perception or on psychometric or number tests.
1	Personality changes, attention deficits, irritability, depressed state	Tremor and in coordination
2	Changes in sleep wale cycle, lethargy, moodand behavioural changes, cognitive	Asterixis, ataxic gait, speech abnormalities (slow and slurred)

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	dysfunction.	
3	Altered level of consciousness (somnolence), confusion, disorientation, and amnesia	Muscular rigidity, nystagmus, clonus,babinski sign, hyporeflexia
4	Stupor or coma	Oculocephalic reflex, unresponsiveness to noxious stimuli

The patients who were in hepatic encephalopathy were studied from the time of admission for duration of 2 weeks.Blood samples were collected and analysed for the levels for serum sodium, Creatinine, Bilirubin, Blood Urea and Total count.

Serial monitoring of the blood parameters was done and analyzed during the period of two weeks along with their clinical outcome.5 ml of blood was collected and segregated into three vacuum tubes. One tube with 2 ml of blood was sent to the Department of Biochemistry Guru Nanak Dev Hospital, Government Medical College, Amritsar for analysis of serum sodium, blood urea and serumcreatinine.1 ml of blood was used to ascertain the serum bilirubin levels which was also analysed in the Department of Biochemistry Guru Nanak Dev Hospital, Government Medical College, Amritsar.1 ml of blood with EDTA was analysed in Pathology department, Government Medical College, Amritsar for complete blood count.In cases and controls, their serum sodium levels were analysed for the same duration of two weeks, blood samples being collected on weekly basis.

STATISTICAL ANALYSIS

The collected data were analysed with SPSS software. The continuous variables were presented as Mean±SD and categorical data were expressed as percentages (%). The numerical values were compared using chi-square test for non-parametric data and student's "t" test for Parametric data. The (p value<0.05) was considered statistically significant.

Reference Range for Sodium:135-145 mEq/l

RESULTS

In both cases and controls, males were comparatively more than females. Gender distribution was comparable in both the group. The mean age among cases was 49.79 ±13.52 years and in controls the mean age was 49.07 ±11.34 years. No statistically significantdifference found among the study groups in terms of age distribution. In both the groups Alcohol was the most common cause of cirrhosis. Out of cases,6% patients had serum sodium levels <120mEq/l, 84% patients had levels between 120-129mEq/l, 10% patients had level between 130-135 mEq/l and there were no patients with Serum sodium levels above 135 mEq/l. In controls, there were no patients with serum sodium levels less than 130 mEq/l, 29% patients had level between 130-135mEq/l and 71% had serum sodium level more than 135 mEq/l. Out of 100 cases, hepatic encephalopathy was seen in 100% cases, while GI bleed was observed in 27% of patients, Coagulopathy was observed in 14% patientsand SBP was observed in 27% patients. Out of 100 controls, no patient had hepatic encephalopathy, 16% patient had GI bleeding, 11% patient had coagulopathy and 23%patients had SBP.Among cases there was statistical difference in the occurrence of complication of chronic liver disease such as SBP (p value-0.04), GI bleed (p value-0.03) with serum sodium levels. There was no statistical difference in presence of Ascites (0.07) and coagulopathy (0.2) in relation to serum sodium levels.There was no statistical difference in the occurrence of complication of chronic liver disease with serum sodium levels such as SBP(p value-0.2), GI bleed (p value-0.3), presence of Ascites(0.08) and coagulopathy(0.5) in control group.

TABLE 1: CORRELATION OF CHILD PUGH CLASS WITH SERUM SODIUM LEVELS IN CASES (AT ADMISSION)

This table shows that out of 6 patients with levels < 120 mEq/l, 2(33.3%) patients had CPC B, 4(66.66%) patients had CPC C and there were no patients in CPC A. Out of 84 patients having serum sodium levels between 120-129mEq/l, 10(11.9%) had CPC A, 34(40.4%) had CPC B, 40(47.61%) had CPC C. Patients with levels between 130-135 mEq/l , 1(10%) had CPC A, 8(80%)in CPC B and 1(10%) in CPC C. There were no patients with serum sodium >135mEq/l. Serum sodium had association with severity of disease. There was statistical significance of Child Pugh Class with serum sodium levels (p value-0.01).

CHILD PUGH CLASS(CPC)	<120 mEq/l (N=6)	120-129mEq/l (N=84)	130-135mEq/l (N=10)	>135 mEq/l
A	0	10(11.9%)	1(10%)	0
B	2(33.3%)	34(40.4%)	8(80%)	0
C	4(66.66%)	40(47.61%)	1(10%)	0

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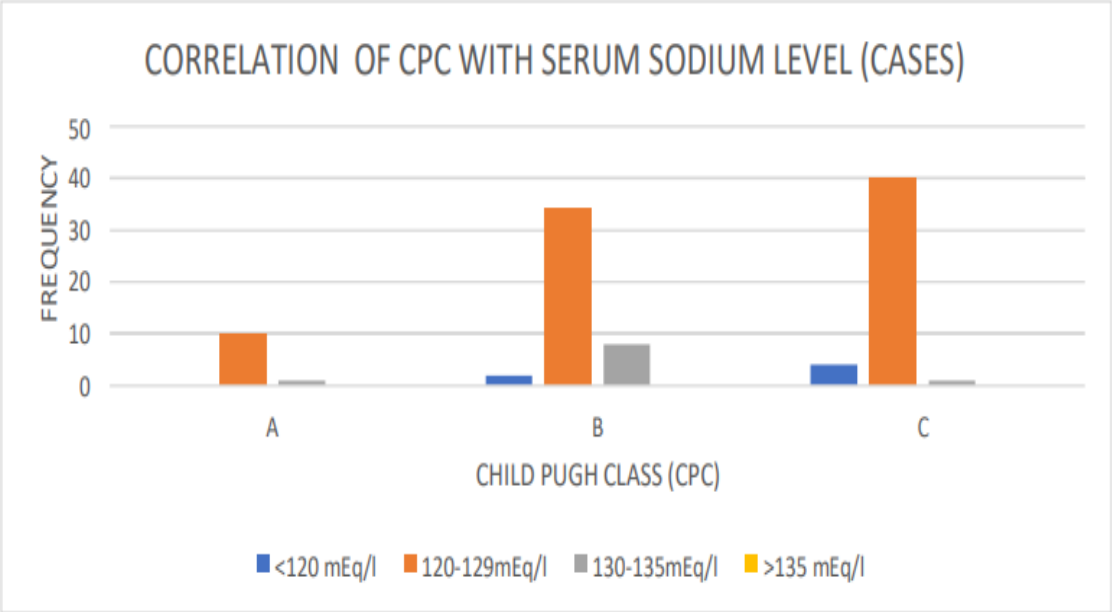

FIGURE 1: BAR DIAGRAM SHOWING CORRELATION OF CHILD PUGH CLASSWITH SERUM SODIUM LEVELS IN CASES (AT ADMISSION)

TABLE 2: CORRELATION OF MEAN MELD SCORE WITH SERUM SODIUMLEVELS IN CASES (AT ADMISSION)

This table shows that cases with serum sodium < 120 mEq/l had mean MELD score of 18.4±1.6, with serum sodium levels between 120-129mEq/l had mean MELD score of 15.2± 0.32 and those with serum sodium levels between 130-135 mEq/l had mean MELD score of 11.83+0.13. There was statistical correlation of MELD score with serum sodium levels among three groups. (P value-0.03).

MELD SCORE(MEAN±SD)	<120 mEq/l	120-129mEq/l	130-135mEq/l	>135 mEq/l
	18.4±1.6	15.2±0.32	11.83±0.13	0

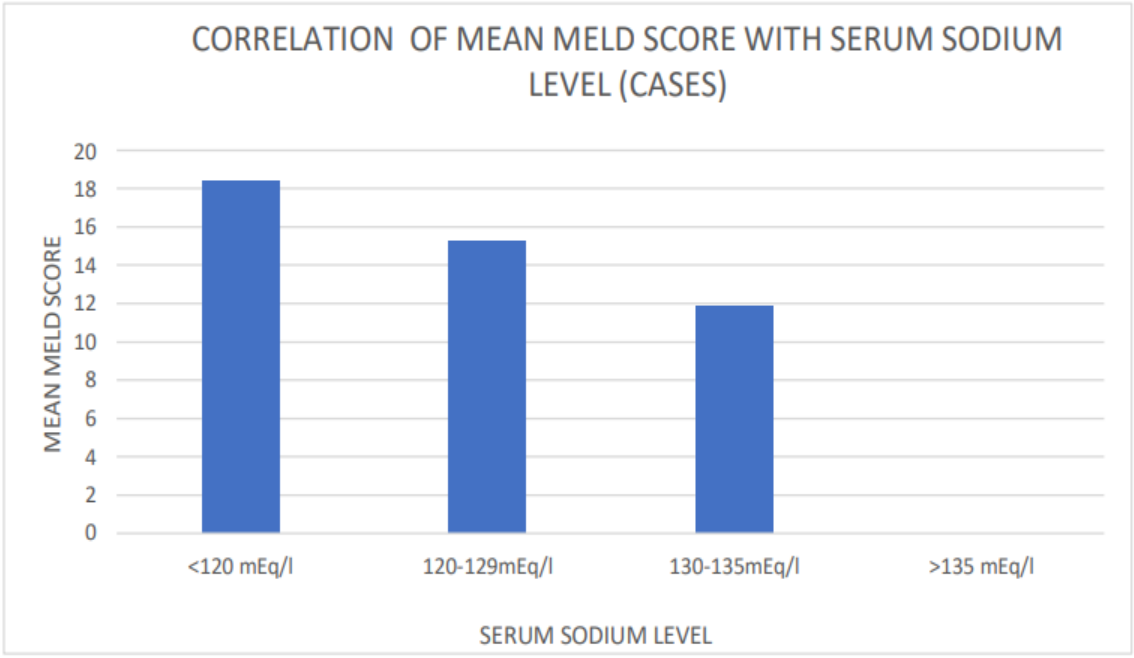

FIGURE 2: BAR DIAGRAM SHOWING CORRELATION OF MEAN MELD SCOREWITH SERUM SODIUM LEVELS IN CASES (AT ADMISSION)

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TABLE 3: MEAN SERUM SODIUM VALUES OF CASES WITH MODE OFCLINICAL WEST HAVEN CRITERIA

This table shows on day 1 patients had mean sodium of 125.36 with standard deviation of 3.32, on day 7 patients had mean sodium of 127.68 with standard deviation of 2.58 and on day 14 patients had mean sodium of 129.16 with standard deviation of 2.43. From day 1 to day 14 patients had improving west haven grading, with maximum patients with West Haven grading of 4 on day 1 to maximum number of patients shifting to West Haven grading of 2 by day 14. It shows statistical significance between serum sodium levels and grading of hepatic encephalopathy (p value-0.001).

DAYS	MEAN SERUM SODIUM	STANDARD DEVIATION	WEST HAVENGRADE			
			1	2	3	4
DAY 1	125.36	3.32	0	22	24	54
DAY 7	127.68	2.58	0	26	52	22
DAY14	129.16	2.43	10	60	20	10

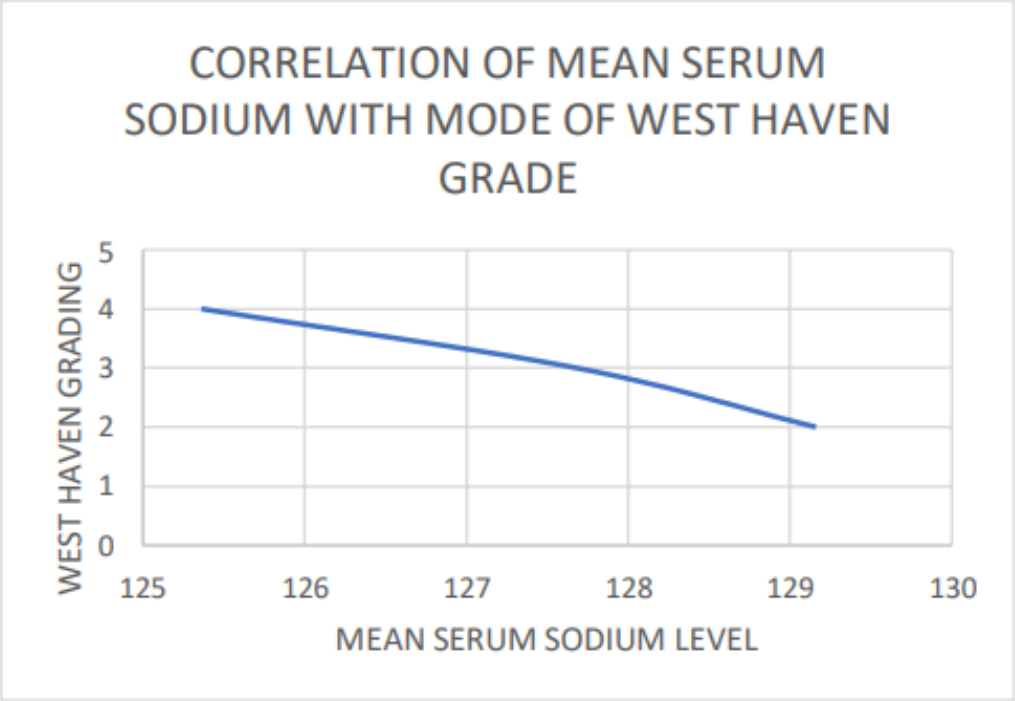

FIGURE 3: BAR DIAGRAM SHOWING CORRELATION MEAN SERUM SODIUMLEVELS WITH MODE OF WEST HAVEN GRADE

TABLE 4: SERUM SODIUM LEVELS AND RELATIVE PERCENTAGE OF CASES WITHIN EACH RANGE

This table shows that from day 1 to day 14 patients shifted from lower serum sodium levels to normal serum sodium levels showing improvement in serum sodium levels over 14 days.

SERUM SODIUM	DAY 1 (N=100)	DAY 7 (N=100)	DAY 14(N=100)
>120 mEq/l	6%	0%	1%
120-129 mEq/l	84%	74%	45%
130-135mEq/l	10%	26%	54%
>135mEq/l	0%	0%	0%

DOI: 10.69605/ijlbpr_13.10.2024.146

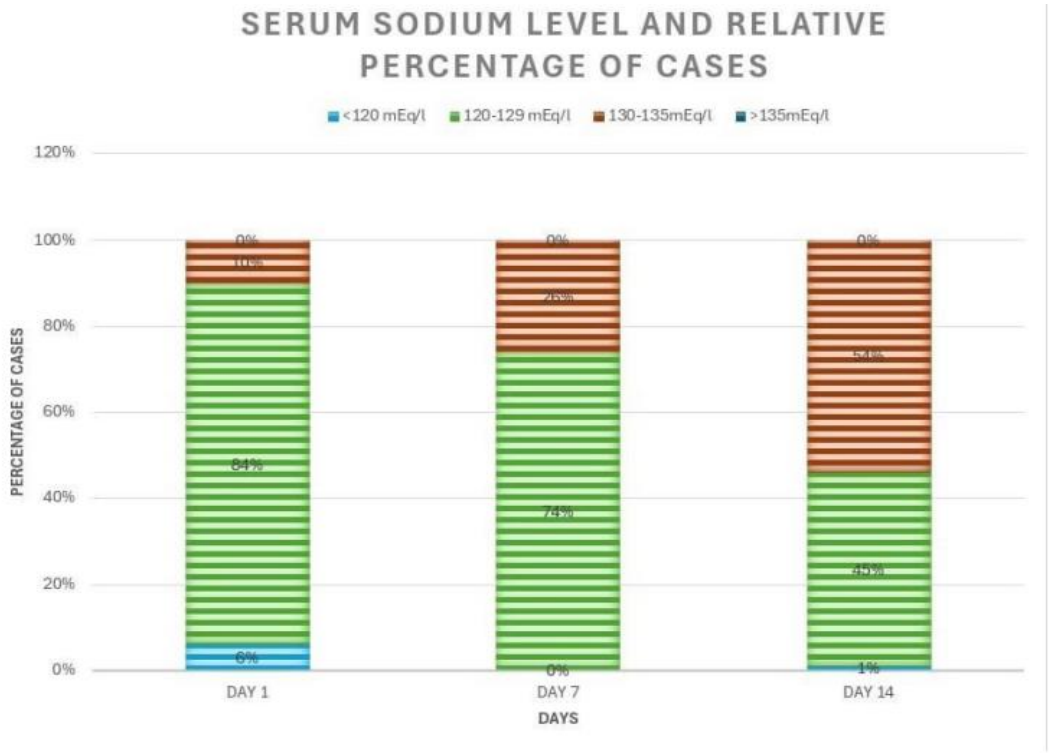

FIGURE 4: BAR DIAGRAM SHOWING SERUM SODIUM LEVELS ANDRELATIVE PERCENTAGE OF CASES WITHIN EACH RANGE

TABLE 5: MEAN SERUM SODIUM VALUES ON DAYS 1,7,14 IN CASES ANDCONTROL

This table shows that there is statistical significance on comparing the serum sodium levels inboth the groups on days 1,7 and 14. Cases mean serum sodium levels improved over the period of 14 days while no such fluctuations were found in controls. This signifies that patient with improving sodium levels had improving grades of hepatic encephalopathy. There was statistically significant difference in mean serum sodium levels between case and controls over 14 days duration (p value-0.001).

DAY	CASES MEAN SERUM SODIUM LEVELS	CONTROL MEANSERUM SODIUM LEVELS	P value
1	125.36+3.3	137.86+3.3	0.004
7	127.68+2.6	139.09+2.2	0.005
14	129.16+2.4	139.24+2.3	0.006

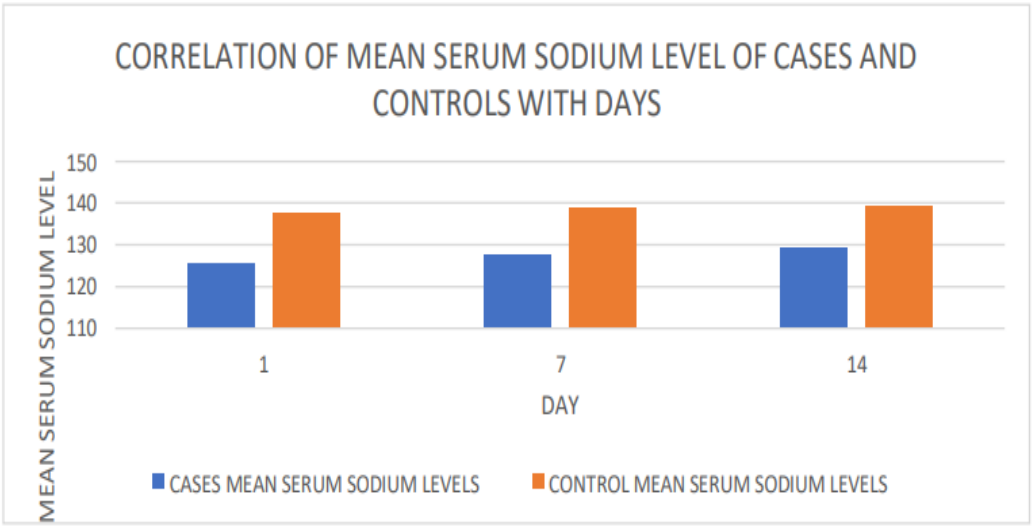

FIGURE 5: BAR DIAGRAM SHOWING MEAN SERUM SODIUM LEVELS INCASES AND CONTROLS ON DAYS 1, 7 AND 14

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TABLE 6: MORTALITY IN CASES ACCORDING TO SERUM SODIUM LEVELSAT DAY 14

This table shows that there was 1 patient with levels<120mEq/l and that patient had mortality,out of 45 patients with levels between120-129 mEq/l,14 (31%) patients had mortality and outof 54 patients with levels between 130-135 mEq/l, 5 patients (9.25%) had mortality.

The difference in mortality among these groups is statistically significant (P value-0.0001).

MORTALITY	<120 mEq/l(N=1)	120-129mEq/l(N=45)	130-135mEq/l(N=54)	>135 mEq/l
No of patientsExpired	1	14	5	0
% of patients	100%	31%	9.25%	0%

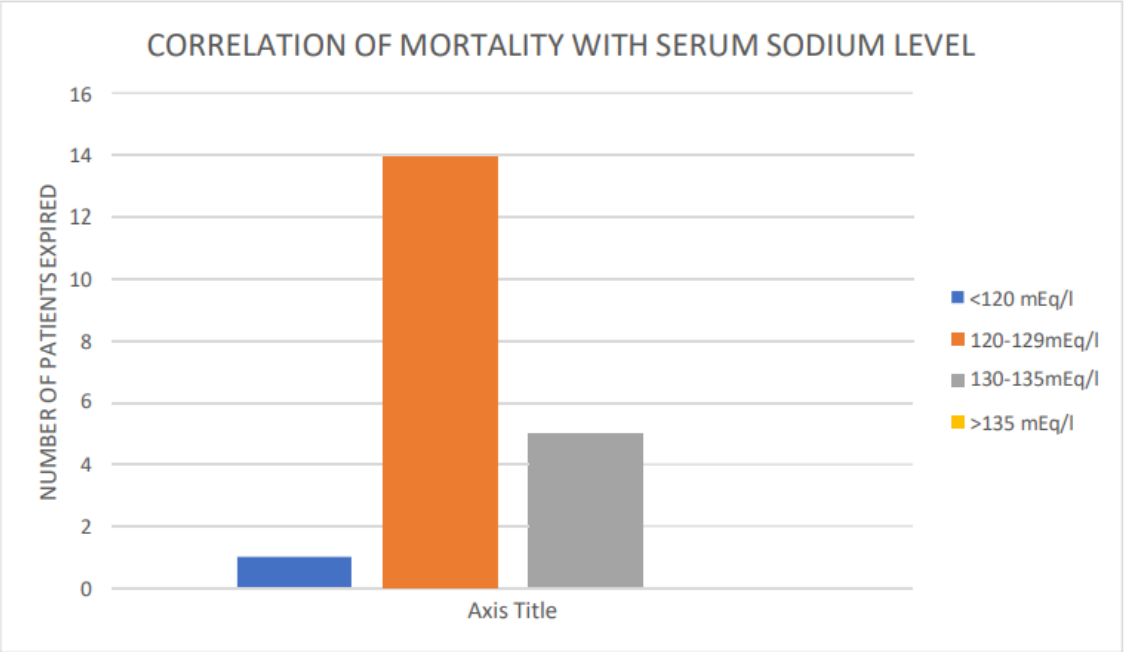

FIGURE 6: BAR DIAGRAM SHOWING CORRELATION OF MORTALITY WITHSERUM SODIUM LEVELS

DISCUSSION

The majority patients in both cases and controls were in the age group of 30-40 years with mean age of cases 49.79±13.52 years while that of controls was 49.07±11.34 years. Similarto our study **Nareddy et al** also had study population with mean age of 48.38 years.[5]

There were 91% males and 9% females in cases while 74 % males and 26% females in controls. There was male preponderance in both the groups. Similar to our study there wasmale preponderance in **Nareddy et al** with 95.8% patients being male.[5]In concordance to with our study **Sardar et al** also found male preponderance in the study.[6]

In cases out of 6 patients with levels < 120 mEq/l, 2(33.3%) patients had CPC B, 4(66.66%)patients had CPC C and there were no patients in CPC A. Out of 84 patients having serum sodium levels between 120-129mEq/l, 10(11.9%) had CPC A, 34(40.4%) had CPC B, 40(47.61%) had CPC C. Patients with levels between 130-135 mEq/l , 1(10%) had CPC A, 8(80%)in CPC B and 1(10%) in CPC C. There were no patients with >135mEq/l. Levels of serum sodium showed association with severity of disease. There was statistical significance of higher Child Pugh Class with low serum sodium levels (p value-0.01). Similar to our study **Kim et al** found that lower levels of serum sodium were seen in patients with higher Child Pugh Class.[7] In similar study conducted by **Godara et al** significant correlation was found between hyponatremia and higher Child Pugh Class.[8]

In cases with levels < 120 mEq/l, patients had mean MELD score of 18.4± 1.6, with serum sodium levels between 120-129mEq/l patients had mean MELD score of 15.2± 0.32 and those with levels between 130-135 mEq/l had mean MELD score of 11.83+0.13. There was statistical correlation of MELD score with levels of serum sodium among three groups. (P value-0.03). Similar to our findings, **Kim et al** found that lower levels of serum sodium were associated with highrer MELD score.[7] Similarly, **Chaudhary RD et al** found higher mean MELD scores in patients with levels of serum sodium less than 130 mEq/l.[9]

In our study, cases had mean serum sodium of 125.36±3.32 mEq/l on day 1, mean serum sodium of 127.68±2.58 mEq/l on day 7, mean serum sodium of 129.16±2.4 mEq/l on day 14.Maximum patients were in West Haven Grade 4 on day 1 and over the duration of 14 days maximum patients shifted to West Haven Grade 2.This shows statistical significance that with

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improving serum sodium levels patients had improvement in West Haven Grading of Hepatic Encephalopathy. (p value-0.001). In concordance to our study, **Gurunamasivayam et al** found that as mean levels of serum sodium improved, the relative number of patients with higher grades of hepatic encephalopathy shifted to lower grades (p value-0.0005).[10] Similar to our study **Godara et al** showed correlation of hyponatremia with grades of Hepatic Encephalopathy.[8]

The mean levels of serum sodium of cases improved from 125.36±3.32 mEq/l on day 1 to 129.16±2.4 mEq/l on day 14.Such fluctuations in serum sodium levels were not observed in controls over duration of 14 days.There was statistical difference between mean serum sodium levels between cases and controls with p value-0.001, implying that majority of patients improved from day 1 to day 14 as shown by improving grades of hepatic encephalopathy which can be attributed to improving serum sodium levels in cases.Similar to our study, **Gurunamasivayam et al** also observed improvement in serum sodium levels in cases but not many fluctuations in controls, attributing the improvement of grading of hepaticencephalopathy with improving serum sodium levels.[10]

On day 14 there was 1 patient with serum sodium levels<120mEq/l and that patient had mortality, out of 45 patients with serum sodium between120-129 mEq/l,14 (31%) patients had mortality and out of 54 patients with levels of serum sodium levels between 130-135 mEq/l, 5 patients (9.25%) had mortality.The difference in mortality among these groups is statistically significant (P value-0.0001). Similar to our study, **Visampally et al**, found similar significance, as mortality rates were higher in patients with level of serum sodium less than 130 mEq/l.[11]

CONCLUSION

Hepatic encephalopathy is correlated with abnormal serum sodium concentration. Patients with level of serum sodium less than 130 mEq/l are correlated with higher west haven grades, indicating that level of serum sodium can be used as prognostic marker in patients of CLD with hepatic encephalopathy. There is increased frequency of other complications such as GI bleed, SBP, coagulopathy and increased mortality rates in patients with lower serum sodium levels. Lower levels of serum sodium correlated with increased MELD score and increased

Child Pugh Class, indicating inverse relationship between hyponatremia and severity of disease. Thus, patients with hyponatremia should be considered a high-risk population because of increased frequency of complications and mortality. Therefore, monitoring of serum sodium levels is a crucial aspect of managing chronic liver disease, enabling timely identification and effective management of disease progression.

Conflict of interest: The authors do not have any conflicts of interest, and all authorscontributed equally in developing the manuscript.

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