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Research Article

A STUDY ON SERUM SODIUM LEVEL IN PATIENTS WITH HEPATIC ENCEPHALOPATHY IN A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: Hyponatremia is common in decompensated cirrhosis and may promote hepatic encephalopathy (HE) by aggravating ammonia-induced astrocyte swelling. We examined serum sodium in relation to HE severity.

Methods: Cross-sectional study at a tertiary hospital in Kolkata, India (February 2021 to October 2022) of 100 adults hospitalised with HE due to decompensated chronic liver disease. HE was graded by the West Haven criteria; sodium was classed as normal (135–145 mmol/L) or mild (126–134), moderate (110–125) or severe (<110) hyponatremia. Groups were compared by ANOVA and exact or chi-square tests.

Results: Mean age was 49.5 ± 14.9 years, 78% were men, and aetiology was alcohol-related in 53% and non-alcoholic steatohepatitis in 35%. HE grades 1 to 4 occurred in 18, 37, 34 and 11 patients. Hyponatremia was present in 83% (moderate or severe in 36%); mean sodium was 126.9 ± 8.6 mmol/L. Mean sodium was 123.3, 132.4, 124.8 and 120.4 mmol/L in grades 1 to 4 ($p<0.001$), lowest in grade 4 and highest in grade 2, without a linear trend ($p=0.25$). Moderate or severe hyponatremia occurred in 66.7%, 10.8%, 38.2% and 63.6% ($p<0.001$). Urea and creatinine, which peaked in grade 3, and platelet count also differed (all $p<0.001$). Age group, but not sex or aetiology, was associated with grade.

Conclusions: Hyponatremia affected most patients hospitalised with HE and varied significantly across West Haven grades, lowest in grade 4, but not linearly, and coexisted with renal dysfunction. Sodium and renal function should be assessed together; larger prospective multivariable studies are needed.

KEYWORDS: Hepatic Encephalopathy, Hyponatremia, Serum Sodium, Cirrhosis, West Haven Criteria, Acute Kidney Injury.

Introduction

Cirrhosis is a leading cause of morbidity and mortality worldwide[1]. Hepatic encephalopathy (HE), a neuropsychiatric syndrome that ranges from subtle cognitive change to coma, is one of the most feared complications of decompensated disease. It is commonly precipitated by infection, gastrointestinal bleeding, constipation and electrolyte disturbance[2]. Its clinical severity is graded with the West Haven criteria, which combine altered consciousness, intellectual function, behaviour and neuromuscular signs[3]. Ammonia is central to its pathogenesis: astrocytes detoxify ammonia by synthesising glutamine, and the resulting osmotic load causes low-grade astrocyte swelling[4].

Hyponatremia is among the commonest electrolyte abnormalities in advanced cirrhosis. It results from non-osmotic hypersecretion of arginine vasopressin, driven by splanchnic arterial vasodilation and reduced effective arterial volume, which impairs renal excretion of solute-free water and produces hypervolemic dilutional hyponatremia[5,6]. Low serum sodium predicts mortality in cirrhosis, including in critically ill patients[7,8].

Hyponatremia may also contribute directly to HE. Depletion of organic osmolytes such as myo-inositol in astrocytes is thought to make the brain more susceptible to ammonia-induced swelling, and the two conditions share overlapping mechanisms and clinical features[9]. In patients with cirrhosis and refractory ascites, a lower serum sodium concentration was a risk factor for HE[10], and hyponatremia has been linked to abnormal brain metabolite profiles and poorer quality of life[11]. Whether serum sodium relates to the severity of HE in hospitalised patients, particularly in South Asian cohorts where non-alcoholic steatohepatitis is emerging alongside alcohol as a major aetiology, is less well characterised. We therefore studied serum sodium in patients hospitalised with HE and its relationship with HE grade.

Materials and Methods

Study Design and Setting

This was a single-centre, cross-sectional observational study conducted in the Department of General Medicine, IPGME&R and SSKM Hospital, Kolkata, a tertiary care teaching hospital in eastern India, from February 2021 to October 2022. The report follows the STROBE recommendations for cross-sectional studies.

Participants

Adult inpatients of either sex with HE complicating decompensated chronic liver disease were eligible. We excluded patients with (i) intracranial lesions (subdural haematoma, intracranial haemorrhage, tumour, stroke or abscess); (ii) central nervous system infection; (iii) other metabolic encephalopathy (hypoglycaemia or anoxia); (iv) Wernicke encephalopathy; (v) post-seizure encephalopathy; (vi) encephalopathy attributable to drugs, including antipsychotics, sedatives, antidepressants and diuretics; and (vii) serum sodium abnormality due to renal, cardiac or endocrine causes. The target sample size of 100 was fixed in advance according to case availability.

Data Collection

After obtaining approval from the Institutional Ethics Committee the data collection was started. We recorded age, sex, comorbidities (hypertension, type 2 diabetes mellitus, hypothyroidism) and addiction history, and performed a general physical examination for stigmata of chronic liver disease and signs of hepatic decompensation. HE was graded 1 to 4 using the West Haven criteria[3] Investigations comprised a complete blood count, serum sodium and potassium, urea, creatinine, liver function tests (bilirubin, aminotransferases, alkaline phosphatase, total protein and albumin), prothrombin time and international normalised ratio (INR), and abdominal ultrasonography with portal Doppler.

Definitions

Serum sodium of 135–145 mmol/L was classed as normal and below 135 mmol/L as hyponatremia, graded as mild (126–134 mmol/L), moderate (110–125 mmol/L) or severe (<110 mmol/L). Aetiology was classed as alcohol-related, non-alcoholic steatohepatitis (NASH), hepatitis C virus-related or cryptogenic.

Statistical Analysis

Data were entered in Microsoft Excel and analysed with SPSS version 27.0 (SPSS Inc., Chicago, IL, USA). Continuous variables are summarised as mean ± standard deviation (SD) and categorical variables as counts and percentages. Means across the four HE grades were compared with one-way analysis of variance (ANOVA), with Tukey post hoc comparisons for sodium. Categorical variables were compared with the chi-square test; A p-value below 0.05 was considered statistically significant.

Results

Characteristics of the Study Population

One hundred patients were included. The mean age was 49.5 ± 14.9 years (range 22–80), and 78 (78%) were men (male-to-female ratio 3.5:1). The commonest aetiologies were alcohol-related liver disease (53%) and NASH (35%); hepatitis C and cryptogenic cirrhosis each accounted for 6%. HE was grade 1 in 18 patients, grade 2 in 37, grade 3 in 34 and grade 4 in 11 (Table 1).

Serum Sodium

The mean serum sodium was 126.9 ± 8.6 mmol/L (range 102–145). Only 17 patients (17%) had a normal sodium; 83 (83%) were hyponatremic, which was mild in 47 (47%), moderate in 28 (28%) and severe in 8 (8%). No patient had

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hypernatremia (Table 1).

Demographic Factors and HE Grade

Age group was significantly associated with HE grade (χ^2=37.7, 12 df; exact p<0.001): patients aged 61 years or older made up 54.5% of grade 4 but only 11.1% of grade 1, whereas those aged 31–40 years were over-represented in grade 3 (44.1%) and those aged 51–60 years in grade 2 (54.1%). There was no linear trend of age with grade (p=0.54). Sex (p=0.13) and aetiology (p=0.052) were not significantly associated with grade (Table 2).

Serum Sodium and HE Grade

Mean serum sodium differed significantly across HE grades (123.3 ± 9.4, 132.4 ± 6.1, 124.8 ± 7.8 and 120.4 ± 7.0 mmol/L in grades 1 to 4; ANOVA p<0.001; Table 3, Figure 1A). Tukey comparisons showed that grade 2 had higher sodium than each of the other grades (all p<0.001), whereas grades 1, 3 and 4 did not differ from one another (p=0.32 to 0.90). The sodium category was also associated with grade (χ^2=29.7, 9 df; exact p<0.001; Table 4, Figure 1B). Moderate or severe hyponatremia (<126 mmol/L) was found in 66.7% of patients with grade 1, 10.8% with grade 2, 38.2% with grade 3 and 63.6% with grade 4 (p<0.001), and any hyponatremia in 83.3%, 67.6%, 94.1% and 100%, respectively (p=0.009). Sodium category did not follow a linear trend with grade (linear-by-linear association p=0.25).

Other Laboratory Parameters and HE Grade

Urea and creatinine differed markedly by grade (both p<0.001), with the highest values in grade 3 (urea 77.6 ± 20.8 mg/dL; creatinine 2.70 ± 1.14 mg/dL) and the lowest in grade 2; the minimum creatinine in grade 3 was 1.7 mg/dL (Table 3). Serum potassium did not differ (p=0.13). Among other variables (Table 5), platelet count differed significantly (p<0.001), being lowest in grade 1 (87 ± 11 $\times 10^9$/L). Nominal differences were seen for total bilirubin (p=0.041), direct bilirubin (p=0.034), prothrombin time (p=0.038), INR (p=0.010), alkaline phosphatase (p=0.040) and total protein (p=0.036), but none reached the Bonferroni threshold. Prothrombin time and INR were highest in grade 1. Haemoglobin, leucocyte count, aminotransferases and albumin did not differ by grade.

Table 1: Baseline characteristics of the 100 patients with hepatic encephalopathy

Characteristic	All patients (N=100)
Age, years, mean ± SD (range)	49.5 ± 14.9 (22–80)
Age group, n (%)	
21–30	12 (12.0)
31–40	25 (25.0)
41–50	12 (12.0)
51–60	35 (35.0)
≥61	16 (16.0)
Sex, n (%)	
Male	78 (78.0)
Female	22 (22.0)
Aetiology, n (%)	
Alcohol-related	53 (53.0)
NASH	35 (35.0)
Hepatitis C	6 (6.0)
Cryptogenic	6 (6.0)
West Haven grade of HE, n (%)	
Grade 1	18 (18.0)
Grade 2	37 (37.0)
Grade 3	34 (34.0)
Grade 4	11 (11.0)
Serum sodium	
Serum sodium, mmol/L, mean ± SD (range)	126.9 ± 8.6 (102–145)
Normal (135–145)	17 (17.0)
Mild (126–134)	47 (47.0)
Moderate (110–125)	28 (28.0)
Severe (<110)	8 (8.0)

Table 2: Age, sex and aetiology according to West Haven grade of hepatic encephalopathy, n (column %)

Variable		Grade 1 (n=18)	Grade 2 (n=37)	Grade 3 (n=34)	Grade 4 (n=11)	P value
Age (years)	21–30	3 (16.7)	6 (16.2)	3 (8.8)	0 (0.0)	**<0.001**
	31–40	1 (5.6)	7 (18.9)	15 (44.1)	2 (18.2)	
	41–50	5 (27.8)	1 (2.7)	4 (11.8)	2 (18.2)	
	51–60	7 (38.9)	20 (54.1)	7 (20.6)	1 (9.1)	
	≥61	2 (11.1)	3 (8.1)	5 (14.7)	6 (54.5)	
Sex	Male	17 (94.4)	25 (67.6)	28 (82.4)	8 (72.7)	0.126
	Female	1 (5.6)	12 (32.4)	6 (17.6)	3 (27.3)	
Aetiology	Alcohol-related	14 (77.8)	13 (35.1)	21 (61.8)	5 (45.5)	0.052
	NASH	4 (22.2)	16 (43.2)	9 (26.5)	6 (54.5)	
	Hepatitis C	0 (0.0)	3 (8.1)	3 (8.8)	0 (0.0)	
	Cryptogenic	0 (0.0)	5 (13.5)	1 (2.9)	0 (0.0)	

Table 3: Serum electrolytes and renal function according to West Haven grade of hepatic encephalopathy, mean ± SD

Variable	All (N=100)	Grade 1 (n=18)	Grade 2 (n=37)	Grade 3 (n=34)	Grade 4 (n=11)	P value
Sodium, mmol/L	126.9 ± 8.6	123.3 ± 9.4	132.4 ± 6.1	124.8 ± 7.8	120.4 ± 7.0	**<0.001**
Potassium, mmol/L	3.77 ± 0.58	3.87 ± 0.43	3.71 ± 0.57	3.89 ± 0.69	3.45 ± 0.35	0.133
Urea, mg/dL	51.3 ± 30.4	43.7 ± 33.7	32.6 ± 19.5	77.6 ± 20.8	45.2 ± 25.1	**<0.001**
Creatinine, mg/dL	1.65 ± 1.14	1.50 ± 0.81	0.88 ± 0.54	2.70 ± 1.14	1.19 ± 0.47	**<0.001**

Table 4: Serum sodium category according to West Haven grade of hepatic encephalopathy, n (column %)

Sodium category (mmol/L)	Grade 1 (n=18)	Grade 2 (n=37)	Grade 3 (n=34)	Grade 4 (n=11)	All (N=100)	P value
Normal (135–145)	3 (16.7)	12 (32.4)	2 (5.9)	0 (0.0)	17 (17.0)	**<0.001**
Mild (126–134)	3 (16.7)	21 (56.8)	19 (55.9)	4 (36.4)	47 (47.0)	
Moderate (110–125)	9 (50.0)	4 (10.8)	9 (26.5)	6 (54.5)	28 (28.0)	
Severe (<110)	3 (16.7)	0 (0.0)	4 (11.8)	1 (9.1)	8 (8.0)	
Summary measures						
Any hyponatremia (<135)	15 (83.3)	25 (67.6)	32 (94.1)	11 (100.0)	83 (83.0)	0.009
Moderate or severe (<126)	12 (66.7)	4 (10.8)	13 (38.2)	7 (63.6)	36 (36.0)	**<0.001**

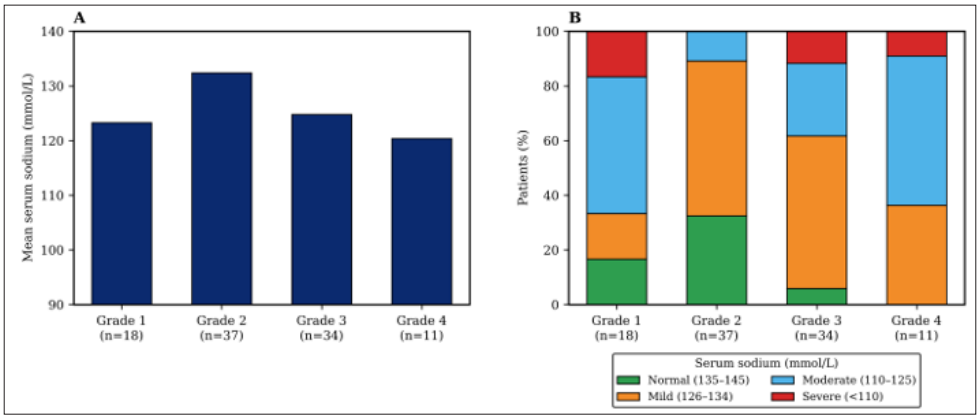


Figure 1. Serum sodium according to West Haven grade of hepatic encephalopathy (HE). (A) Mean serum sodium by HE grade. (B) Distribution of serum sodium categories within each HE grade, shown as a percentage of patients in that grade

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Table 5: Haematological, liver function and coagulation parameters according to West Haven grade of hepatic encephalopathy, mean ± SD

Variable	All (N=100)	Grade 1 (n=18)	Grade 2 (n=37)	Grade 3 (n=34)	Grade 4 (n=11)	P value
Haemoglobin, g/dL	9.86 ± 1.71	9.67 ± 1.31	9.66 ± 1.52	10.00 ± 2.10	10.46 ± 1.59	0.513
Leucocyte count, ×10³/µL	9.09 ± 4.94	7.80 ± 4.21	9.01 ± 4.51	10.44 ± 5.93	7.27 ± 3.03	0.151
Platelet count, ×10⁹/L	124.3 ± 44.5	87.2 ± 11.3	127.0 ± 40.4	138.5 ± 47.3	131.8 ± 53.3	**<0.001**
Total bilirubin, mg/dL	3.80 ± 2.17	4.67 ± 1.69	3.22 ± 1.65	3.64 ± 2.58	4.83 ± 2.51	0.041
Direct bilirubin, mg/dL	2.35 ± 1.83	3.01 ± 1.35	1.78 ± 1.09	2.34 ± 2.27	3.24 ± 2.41	0.034
Prothrombin time, s	21.3 ± 7.1	25.0 ± 5.9	21.6 ± 7.2	19.7 ± 7.8	18.6 ± 3.1	0.038
INR	1.63 ± 0.46	1.93 ± 0.54	1.62 ± 0.41	1.52 ± 0.45	1.50 ± 0.22	0.010
Alkaline phosphatase, U/L	153.4 ± 76.4	160.0 ± 71.6	134.0 ± 29.4	180.1 ± 109.2	125.5 ± 42.3	0.040
ALT, U/L	66.9 ± 40.1	75.2 ± 30.7	59.1 ± 44.2	67.6 ± 40.4	77.6 ± 37.7	0.398
AST, U/L	113.9 ± 50.9	127.4 ± 59.6	101.1 ± 42.2	112.6 ± 49.4	138.8 ± 59.3	0.097
Total protein, g/dL	6.18 ± 0.71	6.31 ± 0.70	5.91 ± 0.46	6.34 ± 0.78	6.35 ± 1.00	0.036
Albumin, g/dL	2.50 ± 0.48	2.52 ± 0.38	2.38 ± 0.49	2.50 ± 0.51	2.82 ± 0.36	0.066

ALT, alanine aminotransferase; AST, aspartate aminotransferase; INR, international normalised ratio. Platelet counts were converted from lakh/mm³ (×100 = ×10⁹/L).

Discussion

In this cross-sectional study of 100 patients hospitalised with HE, hyponatremia was present in 83% and was moderate or severe in more than a third. Serum sodium differed significantly across West Haven grades and was lowest in grade 4 (120.4 mmol/L), but the pattern was not monotonic: grade 2 patients had near-normal sodium, and patients with grade 1 had profound hyponatremia nearly as often as those with grade 4. The frequency of hyponatremia exceeds that generally reported in unselected patients with cirrhosis and ascites[6], which is plausible because a cohort defined by overt HE is enriched for advanced disease and for patients in whom water retention is prominent.

The excess of low sodium in grade 4 is biologically coherent. Hyponatremia lowers osmolyte reserves in astrocytes, increasing their vulnerability to ammonia-induced swelling, and the two encephalopathies frequently coexist[9]. Lower serum sodium has been identified as a risk factor for HE in cirrhosis with refractory ascites[10], and hyponatremia and HE each independently impair health-related quality of life and brain metabolite profiles[11]. Our findings are therefore consistent with hyponatremia contributing to the deepest encephalopathy, although a cross-sectional design cannot establish the direction of effect.

The high frequency of moderate or severe hyponatremia in grade 1 is less expected. Hyponatremia can itself cause inattention, sleep disturbance and tremor[9], so mild neurological features in a profoundly hyponatremic patient may be recorded as low-grade HE even when ammonia-driven encephalopathy is minimal. The West Haven criteria are also inherently subjective at the lower grades. The relatively preserved sodium of grade 2 patients may reflect a different mix of precipitants, which we did not record. The absence of a linear trend means that serum sodium alone did not scale with HE grade in this sample; the relationship appears to be one of association at the extremes rather than a graded dose–response.

Renal function is an important potential confounder. Urea and creatinine were markedly higher in grade 3, in which every patient had a creatinine of at least 1.7 mg/dL. Renal dysfunction limits free-water excretion and promotes hyponatremia, and it is itself associated with cognitive impairment in cirrhosis[12]. In patients hospitalised with HE, organ dysfunction and acute-on-chronic liver failure are common and carry a high mortality[13]. That renal dysfunction was present despite our exclusion of sodium abnormality attributable to renal causes suggests that these patients had functional renal impairment of cirrhosis, such as hepatorenal physiology, which cannot be clearly separated from the cirrhotic haemodynamic mechanism that causes hyponatremia. We did not adjust for it, and the sodium–HE association should be read with this in mind.

Most other biochemical measures showed no robust gradient across HE grades. The nominal differences in bilirubin, prothrombin time, INR, alkaline phosphatase and total protein did not survive correction for 16 comparisons, and

the higher INR in grade 1 runs opposite to what would be expected if coagulopathy tracked encephalopathy severity, so these are best regarded as chance or small-sample effects. The lack of association with albumin, bilirubin and transaminases suggests that HE grade in this cohort was not simply tracking routine markers of hepatic synthetic function. In Indian patients with cirrhosis, HE grade has been shown to correlate with arterial ammonia and inflammatory mediators[14], which we did not measure. Platelet count was lowest in grade 1, a finding for which we have no clear explanation.

These data have practical implications. Serum sodium is cheap and universally available, and its measurement together with renal function should be routine in patients with HE, prompting review of volume status, diuretics and renal function. Sodium is already incorporated into liver-transplant allocation because of its association with wait-list mortality[15], and it is also related to quality of life in cirrhosis with ascites[16]. Vasopressin V2-receptor antagonists correct hyponatremia in cirrhosis[17], but whether this prevents HE remains unproven[18]. Treatment of HE continues to rest on identification and correction of precipitants together with lactulose and rifaximin[3,19,20].

Strengths and Limitations

Strengths are the standardised West Haven grading, the broad panel of laboratory data and the inclusion of NASH, an emerging aetiology. Limitations are important. The study was single-centre with only 100 patients, and group sizes were small, particularly for grade 4 (n=11), leaving many contingency cells sparse. Sodium was measured once, and the design is cross-sectional, so causality and temporal order cannot be inferred. We did not measure ammonia, did not record precipitating events (infection, bleeding, diuretic exposure before admission), and did not report Child–Pugh or MELD scores. Analyses were unadjusted, so confounding by renal function and age is likely. Patients with known renal causes of hyponatremia were excluded, yet renal dysfunction remained prevalent. HE grading is subjective, and 16 continuous comparisons raise the risk of false-positive findings, which we addressed with a Bonferroni threshold. No clinical outcomes were collected.

Conclusion

Hyponatremia was present in more than four of five patients hospitalised with HE. Serum sodium differed significantly by West Haven grade and was lowest in grade 4, but the association was not linear and was accompanied by marked renal dysfunction in grade 3. Serum sodium and renal function should be evaluated together in every patient with HE. Larger, multicentre prospective studies with multivariable adjustment for renal function, ammonia and precipitants, and with outcome follow-up, are needed to determine whether sodium independently predicts HE severity and prognosis.

Funding

None

Conflicts of Interest

None

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