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Original Article

Internal Medicine Section

Clinical Outcomes in Acute Stroke: A Cohort Comparison of Dysnatraemia and Normonatraemia

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ABSTRACT

Introduction: Stroke is the leading cause of mortality throughout the world, with significant economic and social implications. Electrolyte disturbances especially dysnatraemia is common in acute stroke patients and may affect clinical outcomes though its prognostic impact remains unclear.

Aim: To compare clinical outcomes between acute stroke patients with dysnatraemia and those with normonatraemia.

Materials and Methods: In the present prospective cohort study, 130 neuroimaging-confirmed acute stroke patients admitted to a tertiary care hospital in India were enrolled. Serum sodium was measured at admission and during hospitalisation and was categorised into dysnatraemia (both hyponatremia or hypernatremia) as group I and normonatraemia as group II. Primary outcomes were Intensive Care Unit (ICU), and hospital stay lengths, National Institutes of Health Stroke Scale (NIHSS) scores at admission and discharge, and in-hospital mortality. Secondary outcomes included the need for interventions such as intubation, Central Line Placement (CLP) and tracheostomy. Statistical Package for Social Sciences (SPSS) (ver_20) was used for analysis and appropriate tests of significance i.e., Chi-square test or Fisher exact test, Student t-test, Mann-Whitney test and multivariate logistic regression was used to test the significance. Values of $p<0.05$ was statistically significance.

Results: The mean age of group I was 61.2±15.1 years versus 57.6±13.2 years in group II, with approximately 71% of males in both groups. Of 130 patients, 70 (53.8%) had dysnatraemia and 60 (46.2%) had normonatraemia. Group I experienced longer ICU stays (Median 2.5 days versus 2 days, p=0.002) and higher NIHSS scores at discharge (mean 6.35±6.33 vs. 4.36±5.46, p=0.043) than group I. They also had increased rates of CLP (21.4% vs. 6.7%, p=0.018) and requires intubation (31.4% vs. 10%, p=0.003) compared to group II.

Conclusion: Dysnatraemia in acute stroke patients is linked to prolonged ICU stays, more interventions, and poorer neurological outcomes at discharge. Early identification and management of dysnatraemia may improve outcomes in acute stroke patients.

Keywords: Electrolyte imbalance, Hypernatremia, Hyponatremia, Serum sodium

INTRODUCTION

Stroke remains a leading cause of mortality and disability worldwide, with significant economic and social implications. In India, the crude annual incidence of stroke ranges from 108 to 172 cases per 100,000 individuals with one-month case fatality rates between 18% and 42% [1]. Given the high burden of stroke, identifying factors that influence patient outcomes is crucial for improving management strategies and prognosis.

Electrolyte disturbances, particularly dysnatraemia, are frequently observed in acute stroke patients. Hyponatremia, defined as serum sodium (Na^+) levels below 135 mEq/L, affects approximately 15% of patients within the first 24 hours of stroke onset. Conversely, hypernatremia (serum Na^+ levels >145 mEq/L) is less common but can also occur [2]. These electrolyte imbalances may result from various mechanisms, including Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH), Cerebral Salt Wasting Syndrome (CSWS) or iatrogenic causes [3]. The impact of dysnatraemia on stroke outcomes has been a subject of ongoing research, with several studies suggesting its potential influence on patient prognosis. A systematic review by Soiza RL et al., found that hyponatremia was associated with increased mortality in stroke patients, with a pooled odds ratio of 1.60 (95% confidence interval (CI): 1.33-1.93) [4]. Similarly, a large retrospective study by Rodrigues B et al., reported that both hyponatremia and hypernatremia were independent predictors of in-hospital mortality in acute ischaemic stroke patients [5]. However, the relationship between dysnatraemia and other important clinical outcomes remains unclear. While some studies have suggested an association between hyponatremia and prolonged hospital stays (Huang WY et al.,), others have found no significant impact on length of hospitalisation (Saleem S et al.,) [6,7].

The effect of dysnatraemia on specific outcomes such as ICU admission, intervention needs, and neurological status at discharge has been underexplored in acute stroke. Most studies focused primarily on hyponatremia, with limited data on the hypernatremia impact in these patients [4-7]. The relative paucity of research on hypernatremia in these population represents a significant gap in understanding the full spectrum of sodium imbalances in stroke. Given India's high stroke burden and unique regional factors, there is a need for localised data. Additionally, most research originates from Western populations [4-7], highlighting a gap in understanding within the Indian context. With this background, the present study aimed to compare clinical outcomes between acute stroke patients with dysnatraemia and those with normonatraemia.

The primary objective of the study was to assess the serum Na^+ levels in acute stroke patients and categorise them into dysnatraemia and normonatraemia. The secondary objective was to compare the clinical outcomes of dysnatraemia and normonatraemia in acute stroke patients and to estimate the in-hospital mortality rate among the stroke patients with dysnatraemia and normonatraemia.

MATERIALS AND METHODS

The present hospital-based prospective cohort study was done for the period of 18-months from February 2022 to July 2023. This study was conducted in the General Medicine department at Mahatma Gandhi Medical College and Research Institute, a tertiary

care teaching hospital, Pondicherry, India. The hospital has an annual patient volume exceeding 100,000 and is equipped with dedicated stroke units and intensive care facilities. Institutional Human Ethics Committee (IHEC) approval was obtained (MGMCRI/Res/01/2021/37/IHEC/88).

Sample size calculation: Considering the prevalence of hyponatremia among the acute stroke patient was 16%, [5] the sample size calculated was 130 using the formula:

$$n = \frac{(p)(1-p)(Z_{1-\beta}+Z_{\frac{1-\alpha}{2}})^2}{d^2}$$

(p - expected proportion of dysnatraemia (p) 0.16, absolute precision (d) - 0.09, power ($Z_{1-\beta}$) - 0.84; α error ($Z_{1-\alpha/2}$) - 1.96). Consecutive sampling technique was used to include all the patients with inclusion criteria until the desired sample size was achieved during the study period. Patients were categorised into dysnatraemia (both hyponatremia (Na^+ levels <135) or hypernatremia (Na^+ levels ≥145 mEq/L)) as group I and normonatraemia as group II (Na^+ 136 to 145 mEq/L) [2].

Inclusion criteria: Patients aged 18 years or older who were admitted to the Emergency Department with a diagnosis of acute stroke (ischaemic or haemorrhagic) within 48 hours of symptom onset confirmed by neuroimaging were included in the study.

Exclusion criteria: Patients presented with traumatic brain injury, stroke patients presented >48 hours after symptom onset, pre-existing conditions affecting serum sodium levels (e.g., chronic kidney disease, endocrine disorders) and patients on diuretic therapy prior to admission were excluded from the study.

Operational definition: Acute stroke was defined as a sudden onset of neurological deficit and confirmed by clinical examination and neuroimaging {Computed Tomography (CT) or MRI}.

Study Procedure

Using a semi-structured proforma with baseline demographic and clinical data was collected including Glascow Coma Scale (GCS) upon admission along with relevant laboratory data. Serum sodium levels were measured using ion-specific electrode technology at admission and daily throughout hospitalisation. Blood samples were collected in lithium heparin tubes and analysed within two hours of collection. The laboratory adhered to standard quality control measures, including daily calibration of the analyser and participation in external quality assessment programs.

Stroke severity was assessed using the NIHSS at admission and discharge [8]. NIHSS is a 15-item neurological examination of the stroke scale, to evaluate the acute cerebral infarction with the components of levels of consciousness, language, neglect, visual-field loss, extraocular movement, motor strength, ataxia, dysarthria, and sensory loss [8]. It is a 3- to 5-point scale with 0 as normal and the score ranges from 0 to 42, where higher score indicated greater stroke severity. It is stratified as very severe >21, moderate to severe 16-20, mild to moderate 5-15, mild 0-5 [8]. All NIHSS assessments were performed by neurologists trained and certified in the use of this scale to ensure consistency and reliability of the measurements. Patients were monitored daily for clinical progress, complications, and need for interventions.

Outcomes of the study: Primary outcomes were ICU, and hospital stay lengths, NIHSS scores at admission and discharge, and in-hospital mortality. Secondary outcomes included the need for interventions such as intubation, Central Line Placement (CLP), and tracheostomy.

STATISTICAL ANALYSIS

The collected data were entered in Microsoft Excel and analysed using IBM Statistical Package for Social Sciences for Windows (SPSS Inc. version 20.0, Chicago, IL, USA). Normality of the variables was measured using Kolmogorov-Smirnov and Shapiro-Wilk. The data was presented in the form of numbers and percentages for qualitative variables and mean±Standard Deviation (SD)/median with Interquartile Range (IQR) for quantitative variables. Appropriate tests of significance i.e., Chi-square test or Fisher-exact test, student t-test, Mann-Whitney test and multivariate logistic regression was used to test the significance. Values of p<0.05 was statistically significance. Missing data were handled using multiple imputation techniques.

RESULTS

The study population comprised 130 patients, with 70 (53.8%) in the group I and 60 (46.2%) in the group II. All patients in the dysnatraemia group presented with hyponatremia and no cases of hypernatremia were observed during the study period. The demographic and co-morbidity details were presented in [Table/Fig-1].

Variables	Group I Dysnatraemia (n=70)	Group II Normonatraemia (n=60)	p-value
Age (years)	61.2±15.1	57.6±13.2	0.14[a]
Male	50 (71.4)	43 (71.7)	0.97[b]
Stroke type			
Ischaemic	59 (84.3)	50 (83.3)	0.88[b]
Haemorrhagic	11 (15.7)	10 (16.7)	0.88[b]
Co-morbidities			
Hypertension	38 (54.3)	32 (53.3)	0.99[b]
Diabetes mellitus	25 (35.7)	22 (36.7)	
No comorbidities	7 (10.0)	6 (10.0)	

[Table/Fig-1]: Baseline characteristics of the study population.
[a]Independent t-test; [b]Pearson's chi-square test. p-value<0.05 were statistically significant and indicated in boldface. Continuous variables were presented as mean±Standard Deviation (SD), categorical variables were presented as frequencies. Values in brackets represents percentages

Among the study participants, the most common vascular territory involved in the acute stroke was the right middle cerebral artery in 53 patients (40.8%), followed by the left middle cerebral artery in 45 patients (34.6%) [Table/Fig-2]. Patients' assessment at the time of admission and serum sodium levels are presented in [Table/Fig-3]. According to NIHSS scores at admission, 51 patients (39%) had mild strokes, 64 patients (49%) had moderate strokes, and 15 patients (12%) had severe strokes. The differences were not statistically significant. However, patients with dysnatraemia tended to have slightly higher NIHSS scores and lower GCS scores at admission, although these differences did not reach statistical significance.

[Table/Fig-4] explains the primary and secondary outcomes of the study population. Where duration of ICU stays, interventions such as endotracheal tube intubation and CLP were higher in dysnatraemia (group I) when compared to normonatraemia (group II) and were statistically significantly. Comparison of NIHSS at the time of admission and discharge in both groups showed improvement. For ischaemic stroke, the median NIHSS score improvement was three points in both groups (p<0.001). For haemorrhagic stroke, the median improvement was one point in both groups (p=0.004).

Similarly in the correlation analysis, a weak negative correlation between admission sodium levels and discharge NIHSS scores (rho -0.22, p=0.012), suggesting that lower sodium levels were associated with worse neurological outcomes at discharge. Kaplan-Meier analysis showed that group I patients had a longer time to ICU discharge compared to group II patients (log-rank test, p=0.15) [Table/Fig-5].

Territory	n (%)
Anterior communicating artery	1 (0.8)
Bilateral middle cerebral artery	1 (0.8)
Basilar artery	5 (3.8)
Bilateral anterior cerebral artery	1 (0.8)
Left anterior cerebral artery	3 (2.3)
Left middle cerebral artery	45 (34.6)
Left posterior cerebral artery	4 (3.1)
Left vertebral artery	3 (2.3)
Pontine branch of basilar artery	1 (0.8)
Right anterior cerebral artery	6 (4.6)
Right middle cerebral artery	53 (40.8)
Right posterior cerebral artery	5 (3.8)
Right pontine artery	1 (0.8)
Thalamic branch of left internal carotid artery	1 (0.8)

[Table/Fig-2]: Table showing the distribution of vascular territory among the study participants.

Variables	Group I Dysnatraemia (n=70)	Group II Normonatraemia (n=60)	p-value
GCS at admission	14 (12-15)	15 (13-15)	0.06[a]
NIHSS at admission	7 (3-12)	6 (2-9)	0.08[a]
Serum sodium (mEq/L)	131.2±2.8	138.5±2.1	**<0.001**[b]

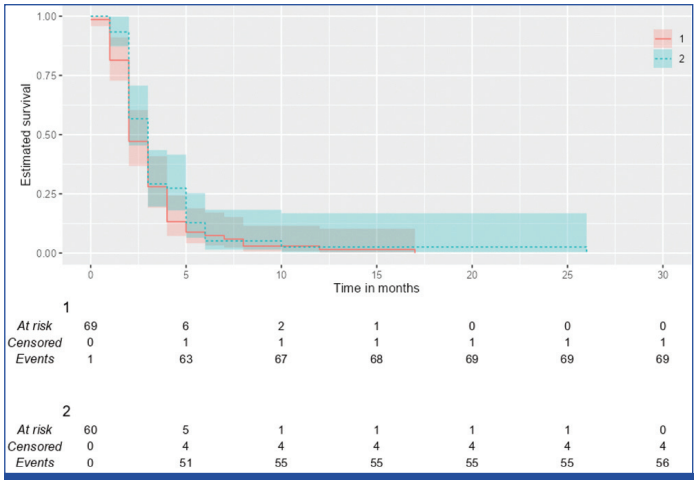
[Table/Fig-3]: Initial assessment and sodium levels of the study population. [a]Mann-Whitney U-test; [b]Independent t-test; p-value<0.05 were statistically significant and indicated in boldface. Continuous variables were presented as mean±Standard Deviation (SD), ordinal variables were presented as median {Interquartile Range (IQR)}. GCS: Glasgow coma scale; NIHSS: National institute of health stroke scale

Outcomes	Group I Dysnatraemia (n=70)	Group II Normonatraemia (n=60)	p-value
Primary outcomes			
ICU stay (days)	2.5 (2-4)	2 (1-3)	**0.002**[a]
Hospital stays (days)	4 (4-6)	4 (4-5)	0.076[a]
NIHSS at discharge	6.35±6.33	4.36±5.46	0.043[b]
In-hospital mortality	4 (5.7)	1 (1.7)	0.373[c]
Secondary outcomes			
Central Line Placement (CLP)	15 (21.4)	4 (6.7)	**0.018**[c]
Endotracheal intubation	22 (31.4)	6 (10.0)	**0.003**[c]
Tracheostomy	3 (4.3)	1 (1.7)	0.450[c]

[Table/Fig-4]: Primary and secondary outcomes for the study population. [a]Mann-Whitney U-test; [b]Independent t-test; [c]Pearson's chi-square test; p-value <0.05 were statistically significant and indicated in boldface. Ordinal variables were presented as median (interquartile range (IQR)); categorical variables were presented as frequencies. Values in brackets were percentages. ICU: Intensive care units; NIHSS: National institute of health stroke scale

DISCUSSION

The present prospective cohort study to compare clinical outcomes between acute stroke patients with dysnatraemia and those with normonatraemia among 130 acute stroke patients resulted that 53.8% had dysnatraemia, suggesting that Na^+ imbalances are a common yet underrecognised complication of stroke. Patients with dysnatraemia had significantly longer ICU stays, higher NIHSS scores at discharge and required more intubations including CLP and intubation than normonatraemia group. Logistic regression confirmed dysnatraemia as an independent predictor of prolonged ICU stay and higher intubation rates. Also, weak negative correlation presents between admission Na^+ levels and NIHSS scores at discharge. Kaplan-Meier analysis showed longed ICU stays in dysnatraemia patients. These findings underscore the impact of Na^+ disturbances on stroke prognosis and highlight the need for early identification and correction.



[Table/Fig-5]: Kaplan-Meier curve shows the length of the ICU stay in both groups. Group I: Dysnatraemia; Group II: Normonatraemia. At-risk: patients who are improved during the course of the study (not dead); censored: exact time of event not observed as before the events happens (dead); Event - occurrence of event (death)

The incidence of dysnatraemia in our study was 53.8% which was higher than previously reported rates 30.6% and 35% by Ng PY et al., and Khan A et al., in patients with acute stroke [9,10]. In a meta-analysis by Shima S et al., showed the range of prevalence of hyponatremia was 7 to 59.2% in acute stroke patients [11]. Similarly, the incidence of dysnatraemia reported in various studies ranges from 0.5-35.3% in patients with acute stroke [7,12-16]. The discrepancies in the incidence of the dysnatraemia was due to the geographic variation, due to the differences Na^+ thresholds and pre-existing comorbidities considered in the study. The mechanism causing dysnatraemia in acute stroke is still in debate. Hyponatremia in stroke is often linked to mechanisms such as secondary adrenal insufficiency, Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH), Cerebral Salt Wasting Syndrome (CSWS), or iatrogenic causes [3,7,17,18]. While, other causes for the Na^+ insufficiency include poor oral intake and also due to area postrema syndrome results in vomiting by activation of CTZ receptors [19].

In the present study, ICU stays among the dysnatraemia group was higher when compared to normonatraemia patients and significant statistically (p=0.002) and also, it was independently associated with longer ICU stay (OR 2.1; p=0.002). In Kaplan-Meier analysis dysnatraemia patients had a longer time to ICU discharge compared to normonatraemia (log-rank test, p=0.003). Study done by Ng PY et al, resulted that patient with hypernatremia had 27% of ICU admission [9]. Shima S et al., also reported that patients with hyponatremia required longer hospital stay (10.68 days (mean difference) in their pooled result [11]. Moreover, patients with dysnatraemia had increased ICU stay 25.35 days when compared to normonatraemia (20.06 days) by Zhang YZ et al., [20]. The pathophysiology behind the longer admission in ICU were due to the volume overload in hyponatremia patients which requires longer duration to correct and to monitor regularly to reduce the mortality among these patients [9,16,21,22]. Thus, early correction of Na^+ imbalance could potentially reduce ICU admissions and improve the patient outcomes.

Hyponatremia may contribute to persistent neurological deficits due to their effect on the brain function and homeostasis and thus contribute to worse neurological recovery [7,18,21]. In the present study, NIHSS at the time discharge was 6.35±6.33 in dysnatraemia group and 4.36±5.46 in normonatraemia group and it was significant statistically, also there were improvement in the score at the time of discharge from admission in both groups, where in correlation a weak negative correlation between admission sodium levels and discharge NIHSS scores (ρ-0.22, p=0.012), suggesting that lower sodium levels were associated with worse neurological outcomes at discharge. Study by Malhotra A et al., also reported

that NIHSS was >16 among the patients with acute stroke with dysnatraemia which was higher when compared to our study results [19]. Study by Kasem AZM et al., showed that patients with NIHSS >15 had highest rates of dysnatraemia along with other electrolyte disturbances [23]. Rodrigues B et al., reported that poor NIHSS scores was the independent prognostic factor for the hyponatremia in acute stroke patients especially among ischaemic stroke at the time of admission and also at discharge which was similar to our study findings [5]. Khan A et al., Lath R et al., Aiyagari V et al., Siddiqui MR et al., also reported the similarities between the poor NIHSS score and hyponatremia [5,10,22-25]. This is due to the presence of cerebral oedema and the osmotic demyelination in patients with hyponatremia if the volume overload were corrected aggressively results in increased NIHSS scores [26,27]. These findings emphasise the need for continuous monitoring of serum Na^+ levels as part of the stroke management.

Thus, given the high burden of stroke and the potential impact of electrolyte imbalances, further research is needed to explore the targeted interventions for Na^+ management in acute stroke. Most existing studies focus on hyponatremia, leaving gaps in our understanding of the broader implications of dysnatraemia in stroke care. Future studies should assess the long-term effects of dysnatraemia on functional outcomes and mortality, as well as investigate the region-specific factors that may influence electrolyte disturbances. Implementing standardised sodium correction protocols may improve stroke outcomes and reduce the ICU resource utilisation. These findings emphasise the need for the routine serum Na+ monitoring as part of comprehensive stroke care, ensuring timely intervention to mitigate the adverse outcomes.

Limitation(s)

The study has several limitations that should be considered when interpreting the findings. First, as a single-centre study conducted in a tertiary care hospital, the generalisability of the results to other healthcare settings may be limited. Next, the study sample size was relatively small as it is single-centric study which warrants the multi-centric study. Followed that, the exclusion of patients with pre-existing condition that affects the Na^+ levels may have led to selection bias, limiting the applicability of results to broader stroke populations. The study also focused on short-term in-hospital outcomes, without long-term follow-up on functional recovery, disability, or recurrence which are critical for assessing the full impact of dysnatraemia. Furthermore, while serum Na^+ levels were monitored, the study did not account for fluid balance, correction strategies, or variations in treatment protocols, which could also influence the patient outcomes. Despite these limitations, the study provides valuable insights into the association between the dysnatraemia and stroke outcomes, emphasising the need for further large-scale, multicentre studies with long-term follow-up.

CONCLUSION(S)

Dysnatraemia, particularly hyponatremia, is a common yet underrecognised complication in acute stroke patients, significantly impacting clinical outcomes. Patients with dysnatraemia experienced longer ICU stays, higher NIHSS scores at discharge, and increased rates of interventions such as intubation and CLP compared to those with normonatraemia. Logistic regression analysis confirmed that dysnatraemia was independently associated with prolonged ICU admission and the need for intubation. A week negative correlation between admission sodium levels and discharge NIHSS scores suggests that lower sodium levels may contribute to worse neurological recovery. These findings highlight the importance of routine sodium monitoring and early correction of dysnatraemia as part of acute stroke management to improve patient outcomes. Further research is warranted to explore optimal sodium correction strategies and their long-term effects on stroke recovery.

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