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FREQUENCY OF HYPONATREMIA AND ITS EFFECT ON MORTALITY IN PATIENTS WITH ISCHEMIC STROKE

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Abstract

Objective: *To determine the frequency of admission hyponatremia in acute ischemic stroke and assess its independent association with 30-day all-cause in-hospital mortality.*

Study Design: *Observational cohort study.*

Place and Duration of Study: *Department of Medicine and Neurology, Tertiary Care Hospital, Pakistan, from May to Aug 2025.*

Methodology: *A total of 140 adult patients with acute ischemic stroke admitted within 24 hours of onset were enrolled. Clinical profiles and baseline serum sodium concentrations were documented upon admission. Hyponatremia was defined as serum sodium <135 mmol/L. Patients were followed for 30 days to record all-cause mortality. Group comparisons utilized Student's t-test and Fisher's exact test. Multivariable binary logistic regression was conducted to determine the independent risk of mortality.*

Results: *The mean age was 62.54 ± 9.63 years, with 77 (55.0%) males. Common comorbidities included hypertension (52.1%), diabetes mellitus (35.0%), and obesity (25.7%). Admission hyponatremia was present in 28 patients (20.0%; 95% CI: 14.2%–27.4%). The overall 30-day mortality rate was 10.0% (14/140). Patients with hyponatremia experienced a significantly higher 30-day mortality compared to normonatremic individuals (25.0% vs. 6.25%; p = 0.008; RR = 4.00, 95% CI: 1.53–10.47). On multivariable logistic regression adjusting for age, sex, and vascular risk factors, hyponatremia remained an independent predictor of 30-day fatal outcome (aOR = 5.55, 95% CI: 1.64–18.80, p = 0.006).*

Conclusion: *Admission hyponatremia is prevalent in 20.0% of acute ischemic stroke patients and confers an independent, greater than five-fold increased risk of 30-day mortality. Baseline electrolyte screening is crucial for early risk stratification.*

INTRODUCTION

Stroke is the second leading cause of mortality worldwide, accounting for approximately 5.54 million deaths annually, with nearly two-thirds of these fatalities occurring in resource-limited settings.[1] It is a major global health concern,

representing the foremost cause of adult disability and the second leading cause of death globally.[2] According to estimates by the World Health Organization (WHO) for the year 2020, stroke was projected to remain the second leading cause of death alongside ischemic heart disease in both developing and developed nations.[3]

Stroke is broadly classified into two major subtypes: ischemic and hemorrhagic. Timely differentiation between these subtypes is critical for appropriate therapeutic interventions, as the management strategies differ significantly. Ischemic stroke necessitates the administration of thrombolytic and/or antiplatelet therapy, whereas hemorrhagic stroke requires hemostatic treatment. Optimal outcomes are achieved when either thrombolytic or hemostatic therapy is initiated promptly following the onset of symptoms.[1]

Notably, stroke patients in developing countries tend to present at a younger age—approximately a decade earlier than their counterparts in developed nations—resulting in a greater burden of long-term disability among survivors.[4] Ischemic stroke constitutes approximately 60–90% of all stroke cases in Pakistan and is primarily caused by an obstruction in a cerebral blood vessel, leading to reduced perfusion to the affected brain region. Risk factors for stroke can be categorized as non-modifiable (including advanced age, family history, prior stroke, sex, and ethnicity) and modifiable (such as hypertension, diabetes mellitus, coronary artery disease, atrial fibrillation, dyslipidemia, smoking, obesity, excessive alcohol consumption, and physical inactivity).[5-6]

Hyponatremia is the most frequently encountered electrolyte disturbance in hospitalized patients and often signifies an underlying systemic pathology. Its prognostic significance in patients with acute first-ever ischemic stroke remains insufficiently elucidated. Emerging evidence suggests that hyponatremia is associated with an increased risk of both stroke and cardiovascular disease. Even mild hyponatremia has been linked to elevated 30-day mortality following myocardial infarction, and recent studies indicate that it may contribute to increased three-year mortality post-stroke.[7]

A study reported an incidence of hyponatremia in 16% of patients with ischemic stroke, with a corresponding mortality rate of 19.6% in affected individuals.[8] The presence of hyponatremia among ischemic stroke patients is associated with increased morbidity and mortality. However, there is a paucity of local data regarding the prevalence and impact of hyponatremia in ischemic stroke patients in Pakistan, where ischemic stroke predominates.

This study aims to document the frequency of hyponatremia in this patient population to establish a comprehensive local dataset that can be compared with existing international literature. The findings will aid in formulating clinical guidelines for the timely diagnosis and management of hyponatremia in ischemic stroke, thereby contributing to a reduction in disease-related morbidity and mortality. Enhanced management strategies may further improve patient outcomes, decrease hospital stays, alleviate the burden on families, and contribute to cost savings in the national healthcare system.

MATERIALS AND METHODS

The study will be conducted at Department of Medicine, Provincial Headquarter Hospital, Gilgit. Study Design is a Observational cohort study. Sampling Technique is Non-probability consecutive sampling**.** Sample size of 144 cases is estimated by using 6% margin of error with 95% confidence level and expected percentage of hyponatremia in ischemic stroke as 16%.[8]

Gender: Both male and female patients. Age: Individuals aged between 30 and 80 years. Diagnosis: Patients diagnosed with ischemic stroke, as defined by the operational criteria.

Pre-existing Cardiovascular and Hematological Disorders: Patients with a known history of coronary artery disease (CAD), coagulopathies, or bleeding disorders such as hemophilia, as documented in clinical records. Neurological Conditions: Individuals with a prior diagnosis of brain tumors, tuberculous meningitis, viral or bacterial encephalitis, or multiple sclerosis, based

on medical history and records. Radiological Findings: Patients with computed tomography (CT) brain imaging suggestive of a space-occupying lesion (SOL).

After taking approval from hospital's Ethical Committee and written informed consent from patients, 144 cases of stroke, fulfilling inclusion and exclusion criteria will be recruited from Department of Medicine of Provincial Headquarter Hospital in Gilgit. Patients will be briefed about objectives of this study, ensuring them confidentiality of the information provided and fact that there is no risk involved to the patient while taking part in this study.

Venous blood (3 ml) sample will be drawn and sent to the central laboratory of Hospital for the serum sodium levels estimation and noted in proforma. History will be taken for hypertension, smoking and diabetes. All the findings will be recorded on pre-designed questionnaire by the researcher. Those patients who will have hyponatremia will be followed for 30 days to document mortality in these patients. Any bias will be controlled through exclusion. All the data will be collected through a pre-designed structural proforma (attached).

Data will be entered and analyzed by computer program SPSS v25. Mean ± standard deviation or median (IQR) will be calculated for the age of patients and serum sodium levels after checking normality by Shapiro-Wilk test. Frequencies and percentages will be calculated for categorical variables like gender, history of hypertension, Obesity, Diabetes, mortality and hyponatremia. Effect modifiers like age, gender, hypertension, obesity and diabetes will be controlled by stratification. Post-stratification, Chi-square test or Fischer's exact test will be applied to see their effect on hyponatremia. P-value equal or less than 0.05 will be considered as significant.

RESULTS

A total of 140 patients presenting with acute ischemic stroke were enrolled and analyzed. The overall mean age of the cohort was 62.54 ± 9.63 years (range: 40 to 80 years). Males accounted for 77 (55.0%) cases and females comprised 63 (45.0%), resulting in a male-to-female ratio of 1.22:1. The majority of patients were married (n = 113, 80.7%), whereas 27 (19.3%) were unmarried or widowed. With respect to documented vascular and metabolic comorbidities, systemic hypertension was the most frequent comorbidity, identified in 73 (52.1%) patients, followed by type 2 diabetes mellitus in 49 (35.0%) and obesity in 36 (25.7%).

The overall baseline serum sodium concentration ranged from 128.3 to 145.5 mmol/L, with a mean of 138.35 ± 4.59 mmol/L. Baseline hyponatremia (defined as admission serum sodium < 135 mmol/L) was detected in 28 patients, yielding a frequency of 20.0% (28/140; 95% CI: 14.2% – 27.4%), while 112 (80.0%) patients were normonatremic. The mean serum sodium level was significantly lower in the hyponatremic cohort (131.48 ± 2.00 mmol/L; range: 128.3 – 134.6 mmol/L) as compared to the normonatremic cohort (140.07 ± 3.24 mmol/L; range: 135.1 – 145.5 mmol/L; $t = -13.435$, $p < 0.001$).

Cross-comparison between patients with and without hyponatremia demonstrated well-balanced demographic and clinical profiles across both groups. There were no statistically significant differences regarding mean age (62.96 ± 9.39 vs. 62.44 ± 9.73 years; $t = 0.258$, $p = 0.797$), male gender (57.1% vs. 54.5%; $\chi^2 = 0.065$, $p = 0.799$), marital status (82.1% vs. 80.4% married; $\chi^2 = 0.046$, $p = 0.830$), hypertension (57.1% vs. 50.9%; $\chi^2 = 0.351$, $p = 0.554$), diabetes mellitus (35.7% vs. 34.8%; $\chi^2 = 0.008$, $p = 0.929$), or obesity (14.3% vs. 28.6%; $\chi^2 = 2.393$, $p = 0.122$) (Table I).

The overall 30-day mortality rate in the entire study cohort was 10.0% (14/140; 95% CI: 6.1% – 16.1%). Notably, the 30-day mortality rate was four-fold higher in patients with admission hyponatremia compared to those with normonatremia [25.0% (7/28) vs. 6.25% (7/112); Pearson $\chi^2 = 8.750$, $p = 0.003$; continuity-corrected Yates $\chi^2 = 6.791$, $p = 0.009$; Fisher's exact test $p = 0.008$], reflecting an absolute risk increase of 18.75% (Table II). Bivariate risk assessment confirmed that hyponatremic stroke patients experienced a four-fold elevation in relative risk (RR: 4.00, 95% CI: 1.53 – 10.47) and a five-fold

elevation in unadjusted odds of death (OR: 5.00, 95% CI: 1.59 – 15.75; p = 0.006). In addition, patients who died within 30 days had significantly lower admission serum sodium concentrations than survivors (135.65 ± 3.91 vs. 138.65 ± 4.57 mmol/L; t = 2.359, p = 0.020).

In the multivariable binary logistic regression model controlling for potential confounding factors—including age, gender, hypertension, diabetes mellitus, and obesity—admission hyponatremia maintained strong and independent prognostic significance for 30-day mortality (adjusted OR [aOR]: 5.55, 95% CI: 1.64 – 18.80, p = 0.006) (Table III). None of the baseline clinical covariables reached statistical significance in the adjusted model.

Table I: Baseline Demographic and Clinical Characteristics stratified by Admission Serum Sodium Status (N = 140).

Parameter	Total Cohort (N = 140)	Hyponatremic Group (n = 28)	Normonatremic Group (n = 112)	Test Statistic	p-value
Age (years), Mean ± SD	62.54 ± 9.63	62.96 ± 9.39	62.44 ± 9.73	t = 0.258	0.797[a]
Gender, n (%)				χ^2 = 0.065	0.799[b]
• Male	77 (55.0%)	16 (57.1%)	61 (54.5%)	—	—
• Female	63 (45.0%)	12 (42.9%)	51 (45.5%)	—	—
Marital Status, n (%)				χ^2 = 0.046	0.830[b]
• Married	113 (80.7%)	23 (82.1%)	90 (80.4%)	—	—
• Unmarried	27 (19.3%)	5 (17.9%)	22 (19.6%)	—	—
Hypertension, n (%)	73 (52.1%)	16 (57.1%)	57 (50.9%)	χ^2 = 0.351	0.554[b]
Diabetes Mellitus, n (%)	49 (35.0%)	10 (35.7%)	39 (34.8%)	χ^2 = 0.008	0.929[b]
Obesity, n (%)	36 (25.7%)	4 (14.3%)	32 (28.6%)	χ^2 = 2.393	0.122[b]
Serum Sodium (mmol/L), Mean ± SD	138.35 ± 4.59	131.48 ± 2.00	140.07 ± 3.24	t = -13.435	<0.001[a]

SD = Standard Deviation; mmol/L = millimoles per liter. [a] Independent samples Student's t-test. [b] Pearson's Chi-square test.

Table II: Cross-tabulation and risk analysis of 30-day mortality according to serum sodium status (N = 140).

Serum Sodium Status	30-Day Mortality: Deceased (n = 14)	30-Day Mortality: Survived (n = 126)	Mortality Rate (%)	Relative Risk (95% CI)	Odds Ratio (95% CI)	p-value
Hyponatremia (<135 mmol/L)	7 (50.0%)	21 (16.7%)	25.0%	4.00 (1.53 – 10.47)	5.00 (1.59 – 15.75)	0.008[a]
Normonatremia (≥135 mmol/L)	7 (50.0%)	105 (83.3%)	6.25%	1.00 (Reference)	1.00 (Reference)	—
Total Cohort	**14 (100.0%)**	**126 (100.0%)**	**10.0%**	—	—	—

CI = Confidence Interval. [a] Fisher's exact test (two-tailed). Pearson's Chi-square = 8.750, p = 0.003.

Table III: Univariate and multivariable binary logistic regression analysis for predictors of 30-day mortality.

Variable	Unadjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
Hyponatremia (Yes vs. No)	**5.00 (1.59 – 15.75)**	**0.006**	**5.55 (1.64 – 18.80)**	**0.006***
Age (per year increase)	0.98 (0.92 – 1.04)	0.434	0.98 (0.92 – 1.05)	0.518
Gender (Male vs. Female)	1.54 (0.49 – 4.83)	0.463	1.81 (0.53 – 6.19)	0.343
Hypertension (Yes vs. No)	2.50 (0.75 – 8.35)	0.137	2.52 (0.70 – 9.06)	0.157
Diabetes Mellitus (Yes vs. No)	0.72 (0.21 – 2.45)	0.597	0.85 (0.23 – 3.15)	0.811
Obesity (Yes vs. No)	1.18 (0.34 – 4.04)	0.797	1.84 (0.48 – 7.10)	0.377

* Statistically significant at $p < 0.05$. OR = Odds Ratio; CI = Confidence Interval. Model log-likelihood = -39.82; Pseudo $R^2 = 0.125$.

DISCUSSION

Acute ischemic stroke remains a preeminent global cause of mortality, persistent neurological disability, and extensive socioeconomic strain, disproportionately affecting developing and transitional economies across South Asia.[1] In countries like Pakistan, the adverse prognosis of acute neurovascular events is further compounded by delayed emergency presentation, limited regional accessibility to intravenous thrombolysis, scarce endovascular thrombectomy infrastructure, and an overwhelming burden of untreated vascular risk factors.[2,3] While traditional cardiovascular risk determinants—including chronic arterial hypertension, diabetes mellitus, dyslipidemia, and systemic atherosclerosis—have historically formed the bedrock of clinical risk stratification, increasing scientific emphasis has shifted toward evaluating secondary physiological disruptions during the hyperacute phase of stroke.[3,4] Among these secondary metabolic complications, alterations in fluid and electrolyte homeostasis, particularly disorders involving serum sodium concentration, represent the most frequent biochemical abnormalities encountered in emergency stroke units.[4,5] Baseline hyponatremia is not an innocuous bystander; rather, it actively destabilizes the ischemic penumbra, enhances cytotoxic cellular swelling, impairs intracranial volume buffering mechanisms, and triggers severe neurovascular decompensation.[5]

In the current study, the prevalence of baseline admission hyponatremia (<135 mmol/L) was 20.0% (95% CI: 14.2% – 27.4%) among 140 consecutive patients presenting with acute ischemic stroke. This observed frequency demonstrates remarkable concordance with recent multi-center international registries and neurocritical cohorts, where admission hyponatremia rates consistently fluctuate between 16% and 32%.[5,6] In an updated global systematic review and meta-analysis evaluating prospective acute stroke cohorts, the pooled baseline hyponatremia prevalence was established at 19.8%, almost exactly paralleling the proportion observed in our cohort.[6] Furthermore, regional investigations conducted across tertiary stroke services in Pakistan have documented hyponatremia rates spanning 18% to 25%, affirming that approximately one out of every five presenting stroke patients suffers from significant sodium depression.[7,8] The minor variances reported across existing literature can be

attributed to differences in clinical inclusion windows, variations in pre-hospital crystalloid infusions, and differing cutoff thresholds employed to define sodium deficiency (135 mmol/L versus 136 mmol/L).[6,8]

Analysis of our cohort's baseline demographic parameters revealed an overall mean age of 62.54 ± 9.63 years alongside a modest male predominance of 55.0%, an epidemiological distribution consistent with established South Asian stroke registries.[2,4] Notably, comparative stratification between hyponatremic and normonatremic subgroups demonstrated marked equivalence across all recorded demographic and chronic vascular comorbidities, including age (62.96 ± 9.39 vs. 62.44 ± 9.73 years, p = 0.797), male sex (57.1% vs. 54.5%, p = 0.799), hypertension (57.1% vs. 50.9%, p = 0.554), diabetes mellitus (35.7% vs. 34.8%, p = 0.929), and obesity (14.3% vs. 28.6%, p = 0.122). This baseline homogeneity is of major scientific importance; it demonstrates that hyponatremia in this population cannot merely be dismissed as an artifact of advanced biological age, patient frailty, or cumulative pre-existing cardiovascular disorders, but rather represents an acute, insult-induced biological response to cerebral ischemia.[7,8]

The most pivotal finding of our investigation is the striking, independent prognostic relationship between baseline hyponatremia and 30-day all-cause in-hospital mortality. Patients presenting with admission hyponatremia exhibited a four-fold increase in 30-day mortality compared to their normonatremic peers (25.0% vs. 6.25%; p = 0.008; RR = 4.00, 95% CI: 1.53 – 10.47), representing an absolute mortality excess of 18.75%. In addition, patients who died within the 30-day observation window had significantly lower admission serum sodium concentrations than survivors (135.65 ± 3.91 vs. 138.65 ± 4.57 mmol/L, p = 0.020). After rigorously controlling for confounding variables through multivariable binary logistic regression, admission hyponatremia retained its robust, independent prognostic impact, conferring an adjusted odds ratio of 5.55 (95% CI: 1.64 – 18.80, p = 0.006) for 30-day fatal outcome. These results corroborate recent prospective clinical studies establishing admission dysnatremia as an autonomous determinant of early neurovascular death.[8,9]

The physiological mechanisms linking hyponatremia to accelerated neurological deterioration and fatal outcomes are complex, progressive, and catastrophic. Extracellular hypo-osmolality disrupts the trans-capillary osmotic gradient across the blood-brain barrier, driving excessive water movement into astrocytic foot processes and parenchymal neurons through bidirectional aquaporin water channels.[10] Within the non-compliant cranial vault, this water shift significantly aggravates both cytotoxic and vasogenic cerebral edema, leading to acute increases in intracranial pressure (ICP) and critical decreases in cerebral perfusion pressure (CPP).[10,11] In the ischemic penumbra—where microvascular autoregulation is severely compromised—decreased CPP accelerates microvascular collapse, converting salvageable neural tissue into irreversibly infarcted core.[11] Furthermore, acute extracellular sodium depletion alters cellular membrane resting potentials, lowers seizure thresholds, suppresses astrocytic glutamate reuptake, and provokes intense neurotoxic depolarization cascades that amplify ischemic neuronal death.[11,12] Systemically, hyponatremic encephalopathy depresses cortical arousal, increases coma rates, causes bulbar dysfunction with aspiration pneumonia, and elevates the risk of fatal cardiac arrhythmias in bed-bound patients.[12]

From an etiopathogenic perspective, acute neurovascular hyponatremia is predominantly driven by two distinct clinical syndromes: the Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) and Cerebral Salt Wasting Syndrome (CSWS).[12,13] Acute focal cerebral ischemia, localized tissue hypoxia, and surrounding inflammatory cascades induce hypothalamic-pituitary dysregulation, stimulating excessive non-osmotic secretion of arginine vasopressin (AVP).[13] Elevated circulating vasopressin prompts continuous insertion of aquaporin channels into renal collecting ducts,

causing free-water retention, volume expansion, and dilutional euvolemic hyponatremia characteristic of SIADH.[13] Conversely, CSWS is provoked by central overproduction of natriuretic peptides or disruption of descending sympathetic renal input, precipitating aggressive renal sodium loss accompanied by true intravascular volume depletion.[13] Accurately discriminating between SIADH and CSWS is essential in neurocritical practice: applying fluid restriction to a hypovolemic patient with CSWS worsens intravascular collapse, inducing profound cerebral hypoperfusion, arterial vasospasm, and irreversible extension of infarction.[13,14]

These observations carry immediate clinical implications for acute emergency stroke care pathways throughout Pakistan. Measuring baseline serum electrolytes is universally available, rapid, and economical, positioning admission serum sodium as an indispensable clinical biomarker for early triage risk stratification.[8,14] Identifying hyponatremia on initial hospital evaluation should immediately flag a patient subgroup at elevated risk for secondary neurological decline, progressive brain swelling, and early mortality. A frequent and dangerous clinical misstep in resource-limited emergency wards is the empiric administration of hypotonic intravenous fluids (such as 5% dextrose in water or half-normal saline), which dramatically worsens hypoosmolar cerebral edema and accelerates uncal herniation.[14] Clinicians must preferentially utilize isotonic crystalloids (0.9% normal saline) for volume resuscitation and maintenance, reserving monitored hypertonic 3% saline for severe, symptomatic cases while adhering strictly to safe correction speeds (<8–10 mmol/L per 24 hours) to prevent fatal osmotic demyelination syndrome.[14]

This investigation features several distinct strengths, including a clearly defined acute cohort, complete 30-day survival ascertainment, and robust multivariable modeling adjusting for primary metabolic risk factors. Nonetheless, certain methodological limitations should be addressed. First, this study was performed at a single referral hospital with a sample size of 140 patients, which may restrict immediate generalizability across remote primary care settings. Second, baseline stroke severity indices (such as the National Institutes of Health Stroke Scale [NIHSS]) and volumetric CT/MRI infarct measurements were not documented in the primary dataset, preventing statistical adjustment for initial anatomical infarct magnitude.[15] Finally, dynamic urinary sodium concentrations, fractional uric acid excretion, and longitudinal plasma osmolality measurements were not serially recorded, precluding definitive syndromic differentiation between SIADH and CSWS across all individual cases. Future prospective multicenter studies incorporating serialized neuroimaging volumetry and detailed fluid-electrolyte balance monitoring are warranted to validate personalized fluid correction protocols in acute stroke care.[14,15]

CONCLUSION

Hyponatremia is a frequent metabolic complication encountered in one out of five patients presenting with acute ischemic stroke. It serves as a potent, independent predictor of 30-day all-cause in-hospital mortality, conferring an over five-fold increase in the adjusted odds of death. Routine admission serum electrolyte screening provides critical, cost-effective prognostic information, allowing early identification of high-risk patients and guiding tailored fluid and electrolyte management to improve stroke survival.

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