Research article

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The prevalence of sodium imbalance in traumatic brain injury patients

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Abstract

Background: Serum sodium imbalances (hyponatremia and hypernatremia) are common electrolyte disturbances in patients with traumatic brain injury (TBI) resulting from the effects of direct brain injury and/or therapeutic interventions. These disturbances are directly associated with patient outcomes. In Vietnam, comprehensive data on sodium imbalances in TBI patients remain limited.

Objectives: To determine the prevalence of sodium imbalances following TBI and assess their impact on poor clinical outcomes.

Subjects and Methods: This was a retrospective descriptive study conducted on 68 patients with TBI treated in the Surgical Critical Care Unit, Department of Anesthesiology, at People's Hospital 115 from November 2023 to May 2024.

Results: The prevalence of sodium imbalances in the study population was 36.8%, including 64.0% hypernatremia, 28.0% hyponatremia, and 8.0% mixed sodium disturbances. Patients with sodium imbalances were 5.93 times more likely to experience poor outcomes at discharge compared to those without imbalances ($p < 0.05$).

Conclusion: The prevalence of sodium imbalances in TBI patients was 36.8%, and these imbalances were closely associated with poor clinical outcomes.

Keywords: sodium disturbances, hypernatremia, hyponatremia, traumatic brain injury

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1. INTRODUCTION

Sodium imbalance is an electrolyte disorder characterized by either hypernatremia or hyponatremia. It is commonly observed in patients with traumatic brain injury (TBI) due to direct brain damage and/or treatment-related mechanisms. This imbalance can worsen neurological outcomes by directly influencing cerebral edema and cerebral perfusion pressure.

In the management of TBI, elevated blood sodium levels can reduce cellular volume and cerebral blood volume, thereby helping to alleviate cerebral edema [1]. Hypernatremia in TBI may result from various causes, such as hypertonic saline therapy, decreased fluid intake, or central diabetes insipidus [2]. Although maintaining elevated sodium levels can have therapeutic benefits, hypernatremia is also associated with increased mortality and complications in

patients with severe TBI [1]. On the other hand, hyponatremia in TBI is often caused by cerebral salt wasting (CSW), syndrome of inappropriate antidiuretic hormone secretion (SIADH), pituitary insufficiency, or inadequate sodium intake. Hyponatremia is recognized as an independent risk factor for poor outcomes in TBI patients [3].

The reported prevalence of sodium imbalance among TBI patients varies widely across international studies. In Vietnam, however, comprehensive data on sodium imbalance in TBI patients remain limited. Therefore, this study aims to determine the prevalence of sodium imbalance following TBI and to evaluate its association with adverse patient outcomes.

2. METHODS

2.1. Inclusion and exclusion criteria

This study included patients with TBI who were treated at the Department of Anesthesiology and Surgical Critical Care, People's Hospital 115, between November 2023 and May 2024.

Exclusion criteria: Patients with chronic kidney disease, diabetes mellitus requiring insulin therapy, heart failure on diuretic treatment, or pregnancy.

2.2. Study design

This was a retrospective descriptive study. The required sample size was calculated using Cochran's formula:

$$\mathbf{n} = \frac{Z^2{}_{1-\alpha/2}\, p(1-p)}{d^2}$$

Where:

- n: Minimum required sample size.
- Z: Z-value for the desired confidence level (1.96 for 95%).
- α: Type I error probability (α = 0.05).
- p: Estimated prevalence of sodium imbalance.
- d: Desired margin of error.

Based on a 95% confidence level (Z = 1.96), an estimated prevalence of sodium imbalance of 68.4% [4], and a margin of error of 13% (d = 0.13), the calculated minimum sample size was 50 patients.

All TBI patients admitted during the study period were identified. Serum sodium levels were categorized as hypernatremia (>145 mEq/L), hyponatremia (<135 mEq/L), or mixed disturbances. Documented causes of sodium imbalance - such as central diabetes insipidus, hypertonic saline administration, SIADH, or CSW - were recorded when available. Relevant clinical parameters, including urine output, urinary sodium concentration, and use of desmopressin (DDAVP), were also noted.

Patient outcomes were assessed using the Glasgow Outcome Scale (GOS). A good outcome was defined as a GOS scores of 4–5 (moderate disability or good recovery), while a poor outcome was defined as a score of 1–3 (death, vegetative state, or severe disability).

2.3. Data Collection and Analysis

Data were coded using Microsoft Excel 2019 and analyzed using JASP software (version 0.18.3). Quantitative variables were presented as mean ± standard deviation or as median with interquartile range, depending on the distribution. Categorical variables were expressed as frequencies and percentages. Comparisons between groups were performed using the Chi-square test (or Fisher's exact test) for categorical variables, and the t-test (or Mann-Whitney U test) for continuous variables, as appropriate. A p-value $\leq$ 0.05 was considered statistically significant.

2.4. Ethics declarations

This retrospective study was conducted to evaluate the prevalence and impact of sodium imbalance in TBI patients. As it involved only the analysis of existing medical records, there was no direct patient contact or intervention, and no risk of harm to the participants. Patient confidentiality was strictly maintained in accordance with hospital regulations.

3. RESULTS

From April to June 2024, a total of 68 patients with TBI met the inclusion criteria. Their baseline characteristics are summarized in Table 1, 2, and 3.

Table 1. Baseline characteristics of the study population

Age*		**48 ± 19**
Gender	Male	51 (75%)
	Female	17 (25%)
Comorbidities	None	49 (72.1%)
	Hypertension	10 (14.7%)
	Diabetes	7 (10.3%)
	Neurological disorders (Parkinson's disease, epilepsy)	2 (2.9%)
	Others	9 (13.2%)
Treatment modality	Surgical	49 (72.1%)
	Non-surgical	19 (27.9%)
Polytrauma		14 (20.6%)
Time to hospital admission	< 12 hours	53 (77.9%)
	> 12 hours	13 (19.1%)
Treatment duration**	Hospital stay (days)	10 (4 – 15)
	ICU stay (days)	4 (1 – 10)

*Mean ± SD

**Median (IQR)

Table 2. Severity of TBI on GCS

Severity	n (%)
Mild	25 (36.8)
Moderate	24 (35.3)
Severe	19 (27.9)

Table 3. CT-scan findings

Injury type	n (%)
Isolated epidural hematoma	8 (11.8)
Isolated subdural hematoma	7 (10.3)
Isolated subarachnoid hemorrhage	2 (2.9)
Isolated cerebral contusion	1 (1.5)
Combined injuries	50 (73.5)

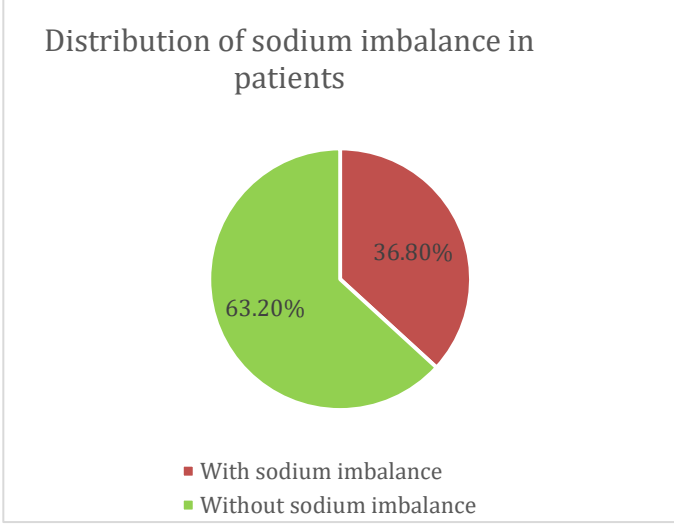

Figure 1. Prevalence of sodium imbalance following TBI

Table 4. Types of dysnatremia

Type	n (%)
Isolated hypernatremia	16 (64.0)
Isolated hyponatremia	7 (28.0)
Mixed disturbances	2 (8.0)

While dysnatremia etiologies in TBI are well-documented, this study could not fully investigate them due to insufficient diagnostic data. However, selected clinical parameters such as 24-hour urine volume and urinary sodium concentration were utilized to partially assess sodium homeostasis.

Table 5. Parameters evaluated for diagnosing causes of sodium imbalance

Parameters	**n (%)**
24-hour urine volume measured	68 (100)
Urinary sodium concentration measured	2 (2.9)
Desmopressin (DDAVP) used	0 (0)

Table 6. Discharge Outcomes based on GOS

Outcome	**n (%)**
Good recovery (GOS 5)	24 (35.3)
Moderate disability (GOS 4)	13 (19.1)
Severe disability (GOS 3)	9 (13.2)
Vegetative state (GOS 2)	7 (10.3)
Death (GOS 1)	15 (22.1)

Table 7. Outcome comparison by sodium imbalance status.

	Poor outcome (n=31) n (%)	**Good outcome (n=37) n (%)**	**p-value**	**Odds ratio**
With imbalance	18 (58.1)	7 (18.9)	0.001	5.93
Without imbalance	13 (41.9)	30 (81.1)		

Patients with sodium imbalance were 5.93 times more likely to experience poor outcomes compared to those with normal sodium levels (p=0.001).

Table 8. Outcome comparison by type of sodium imbalance

	Poor outcome (n=31) n (%)	**Good outcome (n=37) n (%)**	**p-value**	**Odds ratio**
Hypernatremia	12 (38.7)	4 (10.8)	**0.007**	**5.21**
Hyponatremia	4 (12.9)	3 (8.1)	0.517	
Mixed	2 (6.5)	0 (0.0)	0.117	

Isolated hypernatremia was significantly associated with a higher risk of poor outcomes, with an odds ratio of 5.21 (p=0.007), compared to other types of sodium disorders. Furthermore, the presence of comorbid conditions markedly increased the risk of poor outcomes, with an odds ratio of 5.27 ($p < 0.01$) relative to patients without comorbidities.

Table 9. TBI pathology and sodium imbalance association

	With imbalance (n = 25) n (%)	**Without imbalance (n = 43) n (%)**	**p-value**	**Odds ratio**
Combined injuries	22 (88.0)	28 (65.1)	**0.039**	**3.93**
Isolated injuries	3 (12.0)	15 (34.9)		

In addition, patients with combined intracranial injuries were significantly more likely to develop sodium imbalance than those with isolated injuries (OR = 3.93, p = 0.039), suggesting that more extensive brain damage may be associated with greater disruption of sodium regulatory mechanisms.

4. DISCUSSION

Among 68 TBI patients treated in the Surgical Intensive Care Unit (SICU) of the Department of Anesthesiology and Surgical Critical Care at People's Hospital 115, we observed a hypertension (HTN) prevalence of 14.7% and a type 2 diabetes mellitus (T2DM) rate of 9.6%, with no prior insulin treatment recorded. These findings differ from Vedantam et al.'s study on hypernatremia in severe TBI patients, where psychiatric disorders were the most common comorbidity (1.2%) and HTN accounted for only 0.7% [5]. Additionally, our cohort included patients with epilepsy and Parkinson's disease - conditions influencing injury mechanisms, as affected individuals are more prone to sudden falls during daily activities, leading to TBI [6, 7].

TBI Severity and Injury Patterns

Per the Glasgow Coma Scale (GCS), mild TBI was most prevalent (36.8%) in our study, contrasting with Pin-on et al. 's findings, where severe TBI accounted for 70% of cases [8]. This discrepancy may be attributed to differences in data collection methodology, as GCS scores in our study were recorded only at emergency department admission, with subsequent neurological deterioration during hospitalization not systematically documented.

Among isolated intracranial injuries, epidural hematoma (EDH) was most frequent (11.8%), while isolated hemorrhagic cerebral contusion was rarest (1.5%). These findings diverge from prior studies, likely due to differing classification criteria. For instance, Dey et al. reported subdural hematoma (SDH) as the most common CT finding (58%) and diffuse axonal injury as the least (14%) [9].

Sodium Imbalances and Outcomes

Abnormal serum sodium levels occurred in 25 patients (36.8%), including isolated hypernatremia (64%) and isolated hyponatremia (28%). This distribution aligns with Deveduthras et al.'s reported prevalence of hypernatremia (35.3%) versus hyponatremia (33.1%) but contrasts with Dey et al., who found hyponatremia to be predominant (85.4%) [9].

Diagnostic and Therapeutic Challenges

Although the etiologies of dysnatremia are well-established, they could not be thoroughly investigated in this study due to limited diagnostic testing. Only two patients (2.9%) underwent urinary sodium measurement, which is essential for differentiating SIADH (urinary Na^+ > 20 mEq/L) from CSWS. Notably, neither patient had hyponatremia, limiting the utility of these data. We speculate that many cases of dysnatremia lacked definitive etiologic diagnosis. Furthermore, emergency surgical interventions often precluded preoperative correction of electrolyte disturbance. Intraoperative fluid and electrolyte management may have masked early trends, complicating causality assessment.

Long-Term Outcomes and Comorbidities

Of the total cohort, 54.4% experienced good recovery (GOS 4-5), while 45.6% had poor outcomes (GOS 1-3), including disability or death. These findings are consistent with the outcome rates reported by Deveduthras et al. (48.4%) [4]. Poor outcomes were significantly more common in patients with sodium imbalance (58.1%) compared to those with normal sodium levels (41.9%), indicating a strong association ($p = 0.001$). Specifically, hypernatremia was a key contributor to this trend, reinforcing previous literature on its prognostic value in severe TBI [4].

Combined intracranial injuries posed the highest risk for dysnatremia ($p < 0.05$), likely due to the greater potential for disruption of the hypothalamic-pituitary axis-an essential center for fluid and electrolyte regulation. In contrast, isolated injuries (e.g., EDH) rarely involve these midline structures and thus are less frequently associated with sodium imbalance.

Comorbid conditions also exacerbated the impact of TBI, consistent with Hanafy et al.'s finding that pre-existing illnesses compound cognitive and physical impairments post-injury [11]. However, standardized protocols for managing TBI patients with comorbidities remain lacking and warrant further investigation.

Study Limitations

This study has several limitations. The sample size was relatively small and may not reflect broader population trends. Moreover, limited access to laboratory testing, particularly for urinary sodium and osmolality, hindered accurate etiologic classification of sodium disorders. Emergency surgical needs often took precedence over comprehensive metabolic workups, leading to missing data. Future studies should involve larger patient cohorts, prospective designs, and standardized sodium monitoring protocols to better elucidate the relationship between dysnatremia and TBI outcomes.

5. CONCLUSION

Sodium imbalance occurred in 36.8% of patients with TBI, with hypernatremia being the most common form (64.0% of cases). It was significantly associated with poor neurological outcomes. Early recognition and targeted management of sodium disorders are essential for improving outcomes in patients with TBI.

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