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The association between hyponatremia severity and traumatic brain injury outcomes in patients undergoing conservative management: A cross-sectional study from west Sumatra, Indonesia

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Abstract
Background: Traumatic brain injury (TBI) remains one of the leading causes of morbidity and mortality worldwide. Electrolyte disturbances, particularly hyponatremia, are frequent complications that can exacerbate cerebral edema, intracranial hypertension, and poor outcomes. Although hyponatremia has been widely investigated in neurosurgical settings, limited evidence exists regarding its relationship with TBI severity in Indonesian populations.
Objective: This study aimed to evaluate the association between the degree of hyponatremia and TBI severity among patients undergoing conservative management at Dr. M. Djamil General Hospital, Padang, Indonesia.
Methods: A hospital-based cross-sectional study was conducted between January 2023 and January 2025. A total of 112 patients with TBI complicated by hyponatremia who received conservative management were included. Patients with multiple fractures, major comorbidities, inoperable injuries, or those requiring surgical intervention were excluded. Data were analyzed using chi-square tests to assess the association between hyponatremia severity (mild, moderate, severe) and TBI severity (mild, moderate, severe) based on the Glasgow Coma Scale (GCS).
Results: The mean age of participants was 46.7±17.9 years, with a predominance of male patients (74.1%). Mild hyponatremia was most common (83.9%). Among patients with severe hyponatremia, 16.7% presented with severe TBI and 83.3% with moderate TBI. In the moderate hyponatremia group, 41.7% had severe TBI, 25% moderate TBI, and 33.3% mild TBI. In the mild hyponatremia group, 20.2% had severe TBI, 63.8% moderate TBI, and 16% mild TBI. No statistically significant association was observed between the degree of hyponatremia and TBI severity ($p = 0.392$).
Conclusion: This study suggests that the degree of hyponatremia is not significantly associated with TBI severity in patients undergoing conservative management. However, the high prevalence of hyponatremia underscores the importance of early detection and appropriate correction strategies to prevent poor neurological outcomes. Future multicenter studies with larger sample sizes are warranted.

Keywords: Traumatic brain injury, hyponatremia, conservative management, Glasgow Coma Scale, electrolyte disturbance

Introduction
Traumatic brain injury (TBI) is recognized as a major global health concern, contributing substantially to morbidity, disability, and mortality across all age groups. According to the Global Burden of Disease Study 2021, there were approximately 20.8 million new TBI cases worldwide and nearly 38 million prevalent cases, representing a significant public health challenge and economic burden (GBD Neurology Collaborators, 2021). In high-income countries such as the United States, over 2.8 million individuals seek medical attention annually due to TBI, with more than 230,000 hospitalizations and approximately 60,000 deaths reported each year (CDC, 2020; Dewan *et al.*, 2018) [2]. Similarly, in Europe, traumatic brain injuries account for nearly 57,000 deaths and 1.5 million hospital admissions annually (Maas *et al.*, 2017) [8]. These figures highlight the magnitude of TBI and its impact on healthcare systems.
In developing countries, including Indonesia, the burden of TBI is expected to be higher due to rapid urbanization, increased motorization, and limited access to advanced trauma care. Data from the 2018 Indonesian Basic Health Research (Riskesdas) survey reported a national

prevalence of head injuries at 11.9%, making it one of the most frequent trauma-related diagnoses in emergency departments (Indonesian Ministry of Health, 2018). In West Sumatra, the prevalence was even higher, reaching 14.3%, ranking eighth among Indonesian provinces (Akhyar *et al*., 2023) [1]. A local study at Dr. M. Djamil General Hospital, Padang, indicated that the majority of TBI cases were attributed to traffic accidents, accounting for 88.5% of cases (Mudzakir *et al*.). These findings are consistent with global patterns where road traffic accidents, falls, and violence constitute the leading causes of TBI, particularly in younger male populations.

TBI outcomes are influenced not only by the primary injury but also by secondary insults, including hypoxia, hypotension, hyperglycemia, seizures, and electrolyte imbalances. Among electrolyte disturbances, hyponatremia is one of the most frequently observed and clinically significant (Pin *et al*., 2019). Hyponatremia, defined as a serum sodium concentration below 135 mmol/L, has been reported in 9.6-51% of patients with acute brain injury, depending on case mix and diagnostic thresholds (Rajagopal *et al*., 2017; Ha *et al*., 2021) [10, 4]. This condition is clinically important because reductions in serum sodium lower plasma osmolality, causing osmotic water shifts into glial and neuronal cells. Such shifts can precipitate cerebral edema, raised intracranial pressure (ICP), herniation, and ultimately death if not promptly managed (Sherlock *et al*., 2006; Ellison & Berl, 2007) [13, 3].

The mechanisms of hyponatremia in TBI are multifactorial, with the two principal entities being the syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting (CSW). SIADH is characterized by excessive vasopressin release leading to water retention, euvolemia, or mild hypervolemia, while CSW involves natriuresis and hypovolemia due to impaired renal sodium handling mediated by neuroendocrine dysfunction (Harrigan, 2001; Leonard *et al*., 2015) [5, 7]. Differentiating SIADH from CSW is clinically challenging but essential, since management strategies differ substantially: fluid restriction for SIADH and sodium/fluid replacement for CSW (Verbalis *et al*., 2013) [14]. Misdiagnosis can worsen patient outcomes, further complicating the course of TBI management.

Several studies have investigated the association between hyponatremia and TBI severity. In a retrospective study of 1,500 TBI patients, Rajagopal *et al*. (2017) [10] reported a 13.2% prevalence of hyponatremia, particularly among patients with severe injuries. Similarly, Parvar *et al*. (2023) [9] observed hyponatremia in 42.7% of moderate-to-severe TBI cases and found that sodium disturbances were significantly correlated with poor neurological prognosis, independent of age and sex. In South Africa, a cohort study by Oke *et al*. (2019) [16] found hyponatremia in 33.1% of patients with moderate-to-severe TBI. In Southeast Asia, Kusumaningtyas *et al*. (2018) [6] reported that 61.5% of TBI patients developed moderate hyponatremia, which was associated with a 3.6-fold increased risk of mortality. Conversely, Ha *et al*. (2021) [4], in a study of 903 patients, concluded that mild-to-moderate hyponatremia was not significantly related to adverse outcomes, although severe hyponatremia was strongly associated with in-hospital mortality (OR 5.41). These findings suggest that the degree of hyponatremia may play a critical role in determining prognosis, though evidence remains inconsistent across different populations.

In Indonesia, research remains limited. A study from RSUP Sanglah, Bali, reported a high incidence of hyponatremia (46.8%) among TBI patients (Sasmita *et al*., 2019) [17]. Another report from Yogyakarta highlighted a significant association between moderate hyponatremia and increased mortality (Kusumaningtyas *et al*., 2018) [6]. However, other Indonesian studies have failed to demonstrate a clear correlation between serum sodium levels and TBI severity or outcome (Rivaldi *et al*., 2019) [11]. Such discrepancies underscore the need for further investigation, particularly in regions like West Sumatra where epidemiological data are scarce.

The clinical significance of hyponatremia extends beyond neurological deterioration. Hyponatremia has been associated with prolonged intensive care unit (ICU) stays, higher rates of ventilator use, increased hospital costs, and reduced functional recovery (Yumoto *et al*., 2015; Selim *et al*., 2016) [15, 12]. Early detection and appropriate correction of sodium imbalances may therefore play a vital role in optimizing outcomes in TBI patients managed conservatively. Despite this, the relationship between the degree of hyponatremia and the severity of TBI remains poorly defined in Indonesian populations.

Given these considerations, this study was designed to assess the association between hyponatremia severity and TBI severity among patients undergoing conservative management at Dr. M. Djamil General Hospital, Padang, West Sumatra. Understanding this relationship is crucial for guiding clinical decision-making, developing preventive strategies, and improving patient outcomes in resource-limited settings.

Methods

Study Design and Setting

This study employed a hospital-based cross-sectional design conducted at Dr. M. Djamil General Hospital, Padang, West Sumatra, Indonesia, a tertiary referral center for neurosurgical cases in the region. Data collection was carried out over a two-year period, from January 2023 to January 2025. The hospital receives a high volume of trauma cases annually, making it a suitable setting to examine the association between hyponatremia and traumatic brain injury (TBI) severity.

Study Population and Sample

The target population included all patients diagnosed with TBI who presented to the emergency department or inpatient neurosurgical wards during the study period. A total of 112 patients who met the inclusion criteria were enrolled consecutively. Sample size was determined based on feasibility within the study period, with the aim of achieving sufficient statistical power to detect associations between sodium levels and injury severity.

Inclusion and Exclusion Criteria

Inclusion criteria

1. Patients aged ≥18 years diagnosed with TBI (based on history, clinical examination, and radiological findings).
2. Presence of hyponatremia, defined as serum sodium <135 mmol/L, detected during hospitalization.
3. Managed conservatively without neurosurgical operative intervention.

Exclusion criteria

1. Patients with pre-existing chronic medical conditions that may independently influence sodium levels (e.g., chronic kidney disease, adrenal insufficiency, liver cirrhosis).
2. Patients with multiple fractures or polytrauma requiring operative management.
3. Patients undergoing surgical intervention for TBI (e.g., craniotomy, decompressive craniectomy).

4. Inoperable head injuries deemed unsuitable for conservative management.

Variables and Operational Definitions

- **Independent variable:** Degree of hyponatremia, categorized as:
- **Mild:** 130-134 mmol/L
- **Moderate:** 125-129 mmol/L
- **Severe:** <125 mmol/L (Verbalis *et al*., 2013) [14].

- **Dependent variable:** Severity of TBI, assessed using the Glasgow Coma Scale (GCS) on admission:
- **Mild:** GCS 13-15
- **Moderate:** GCS 9-12
- **Severe:** GCS ≤8 (Teasdale & Jennett, 1974) [18].
- **Covariates:** Age, sex, mechanism of injury, and comorbidities.

Data Collection Procedures

Sociodemographic and clinical data were collected from patient medical records, including age, sex, mechanism of injury, clinical presentation, laboratory results, and GCS score at admission. Serum sodium levels were measured using an ion-selective electrode method at the hospital's central laboratory. All measurements were conducted within 24 hours of admission and repeated as necessary during hospitalization. Patients were followed throughout their inpatient stay to confirm eligibility and ensure accurate classification into hyponatremia categories. Data were extracted using a standardized proforma by trained research assistants to minimize transcription errors.

Statistical Analysis

Data were entered and analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables (e.g., age) were expressed as mean ± standard deviation, while categorical variables (e.g., sex, degree of hyponatremia, TBI severity) were presented as frequencies and percentages. The association between hyponatremia severity and TBI severity was assessed using the Chi-square test of independence. A p-value <0.05 was considered statistically significant. Where expected cell counts were small, Fisher's exact test was applied. Results were presented in tabular and graphical formats to enhance clarity.

Ethical Considerations

This study was approved by the Ethics Committee of the Faculty of Medicine, Andalas University / Dr. M. Djamil General Hospital, Padang (Approval No: [placeholder]). Written informed consent was obtained from patients or their legal guardians prior to participation. Patient confidentiality was maintained by anonymizing data, and all procedures were conducted in accordance with the Declaration of Helsinki (2013 revision) on ethical principles for medical research involving human subjects.

Results

Patient Characteristics

A total of 112 patients with traumatic brain injury (TBI) and hyponatremia who underwent conservative management were included in this study. The mean age of patients was 46.7 ± 17.9 years (range: 18-82 years). The majority were male (74.1%), reflecting the higher exposure of men to high-risk activities such as driving and manual labor.

With respect to hyponatremia severity, mild hyponatremia was the most common category (83.9%), followed by moderate hyponatremia (11.6%) and severe hyponatremia (4.5%). Regarding TBI severity, most patients presented with moderate TBI (60.7%), while 22.3% had severe TBI and 17.0% had mild TBI.

Table 1: Demographic and Clinical Characteristics of Patients (n=112)

Variable	n (%) / Mean ± SD
Age (years)	46.7±17.9
Sex	
Male	83 (74.1)
Female	29 (25.9)
Hyponatremia severity	
Mild (130-134 mmol/L)	94 (83.9)
Moderate (125-129 mmol/L)	13 (11.6)
Severe (<125 mmol/L)	5 (4.5)
TBI severity (GCS)	
Mild (13-15)	19 (17.0)
Moderate (9-12)	68 (60.7)
Severe (≤8)	25 (22.3)

Association between Hyponatremia and TBI Severity

When stratified by hyponatremia degree, the distribution of TBI severity is presented in Table 2.

In the severe hyponatremia group (n=5), 16.7% of patients had severe TBI and 83.3% had moderate TBI.

In the moderate hyponatremia group (n=13), 41.7% of patients had severe TBI, 25.0% had moderate TBI, and 33.3% had mild TBI.

In the mild hyponatremia group (n=94), 20.2% had severe TBI, 63.8% had moderate TBI, and 16.0% had mild TBI.

Table 2: Distribution of TBI Severity According to Hyponatremia Degree

Hyponatremia Severity	Severe TBI n (%)	Moderate TBI n (%)	Mild TBI n (%)	Total n (%)
Severe (<125 mmol/L)	1 (16.7)	5 (83.3)	0 (0.0)	6 (100)
Moderate (125-129)	5 (41.7)	3 (25.0)	4 (33.3)	12 (100)
Mild (130-134 mmol/L)	19 (20.2)	60 (63.8)	15 (16.0)	94 (100)
Total	25 (22.3)	68 (60.7)	19 (17.0)	112 (100)

The Chi-square test demonstrated no statistically significant association between hyponatremia severity and TBI severity (p=0.392).

Graphical Presentation

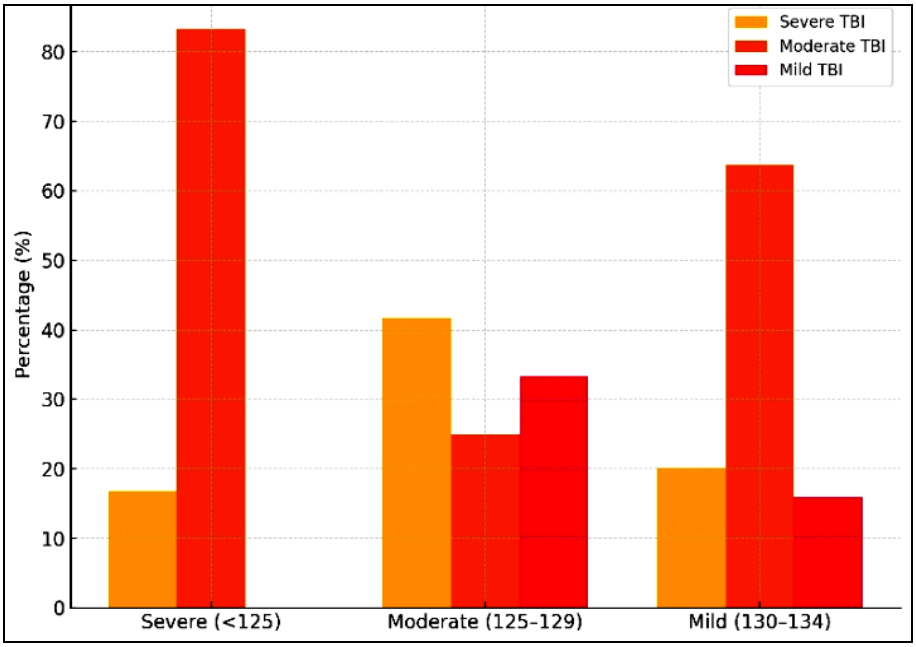

Fig 1: Distribution of TBI Severity by Hyponatremia Degree

A bar chart showing three groups of hyponatremia on the x-axis — mild, moderate, severe — and percentages of mild, moderate, and severe TBI stacked or grouped on the y-axis.

Discussion

Summary of Key Findings

This cross-sectional study involving 112 patients with traumatic brain injury (TBI) and concurrent hyponatremia found no statistically significant association between the degree of hyponatremia and the severity of TBI as measured by the Glasgow Coma Scale (GCS) ($p = 0.392$). While mild hyponatremia was the most common presentation (83.9%), moderate and severe hyponatremia were less frequent (11.6% and 4.5%, respectively). The majority of patients presented with moderate TBI (60.7%), followed by severe TBI (22.3%) and mild TBI (17.0%). These findings suggest that although hyponatremia is common among TBI patients, its severity may not directly correlate with the initial neurological severity as defined by GCS.

Comparison with Previous Studies

Our findings differ from several international reports that demonstrated a relationship between hyponatremia and worse neurological outcomes. For instance, Rajagopal *et al.* (2017) [10] conducted a retrospective study of 1,500 TBI patients and reported a 13.2% prevalence of hyponatremia, with significantly higher rates among those with severe injuries. Similarly, Parvar *et al.* (2023) [9] found that 42.7% of moderate-to-severe TBI patients developed hyponatremia, which was significantly associated with poorer prognoses independent of confounding factors such as age and sex.

In contrast, our results are consistent with findings by Ha *et al.* (2021), who examined 903 patients and concluded that mild-to-moderate hyponatremia was not significantly related to adverse outcomes, though severe hyponatremia was strongly associated with in-hospital mortality (OR 5.41). This indicates that while extreme derangements in sodium levels may predict worse prognosis, milder abnormalities might not correlate with initial injury severity.

Regionally, Kusumaningtyas *et al.* (2018) [6] studied 52 Indonesian patients and reported that moderate hyponatremia was significantly associated with a 3.6-fold increased risk of mortality. Conversely, Rivaldi *et al.* (2019) [11] found no significant association between sodium abnormalities (hyponatremia, normonatremia, hypernatremia) and outcomes in 41 patients with severe TBI. These contrasting results highlight the heterogeneity of findings in different populations and emphasize the need for larger, multicenter studies.

Possible Explanations for Non-significant Findings

Several factors may explain the lack of statistical association in our cohort. First, the distribution of hyponatremia severity was skewed toward mild cases (83.9%), with relatively few patients in the moderate (11.6%) and severe (4.5%) categories. This imbalance may have reduced the statistical power to detect significant differences across groups.

Second, the pathophysiology of hyponatremia in TBI is complex and multifactorial. While SIADH and CSW are the most common mechanisms, other factors such as fluid resuscitation strategies, osmotic therapy, and variations in clinical management could influence sodium levels independently of injury severity (Sherlock *et al.*, 2006; Verbalis *et al.*, 2013) [13, 14]. Therefore, sodium concentration alone may not fully reflect the underlying neurological insult.

Third, our study measured hyponatremia on admission and during hospitalization, but did not assess temporal dynamics. Previous studies (Yumoto *et al.*, 2015) [15] have shown that hyponatremia often develops within 3-7 days post-injury, and its impact may vary depending on timing. Acute vs. delayed hyponatremia may have different clinical consequences, which were not captured in our design.

Clinical Implications

Despite the absence of a direct association, the high prevalence of hyponatremia in our cohort (100% by design, since all patients included had hyponatremia) emphasizes the need for vigilant monitoring and management. Even in the absence of a correlation with initial GCS-based severity, hyponatremia remains clinically relevant due to its potential to worsen cerebral edema, prolong ICU stays, and increase the risk of

complications such as seizures (Ellison & Berl, 2007; Selim *et al*., 2016) [13, 2].

In resource-limited settings such as Indonesia, early recognition of electrolyte imbalances can improve patient outcomes by guiding individualized fluid therapy. Distinguishing between SIADH and CSW remains challenging but crucial, as inappropriate management (e.g., fluid restriction in CSW) could exacerbate hypovolemia and secondary brain injury (Harrigan, 2001; Leonard *et al*., 2015) [5, 7].

Strengths and Limitations

The strengths of this study include its prospective design, standardized data collection, and relatively large sample size compared to prior Indonesian studies. It adds valuable evidence to the limited literature from Southeast Asia, particularly in West Sumatra.

However, several limitations should be acknowledged. First, the cross-sectional design precludes causal inference, limiting our ability to establish temporal relationships between sodium abnormalities and TBI outcomes. Second, the sample distribution was unbalanced, with few moderate and severe hyponatremia cases, reducing statistical power. Third, we relied on GCS as the sole measure of severity; incorporating neuroimaging findings (e.g., Marshall classification) could provide a more nuanced assessment. Fourth, long-term outcomes such as mortality, functional recovery, and quality of life were not evaluated, which may underestimate the clinical significance of hyponatremia.

Future Directions

Future research should adopt longitudinal, multicenter designs with larger, more balanced cohorts to clarify the role of hyponatremia severity in TBI outcomes. Serial monitoring of serum sodium and integration of neuroimaging and biochemical markers could improve diagnostic accuracy and prognostication. Moreover, interventional studies comparing management strategies for SIADH vs. CSW in TBI patients are warranted to optimize clinical outcomes.

Conclusion

This study found no significant association between hyponatremia severity and TBI severity in patients undergoing conservative management at a tertiary hospital in West Sumatra. Nonetheless, the high prevalence of hyponatremia highlights its clinical importance, warranting careful monitoring and tailored management. Further large-scale studies are required to validate these findings and inform evidence-based guidelines for managing electrolyte disturbances in TBI.

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