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# Electrolyte derangements and outcome after moderate and severe traumatic brain injury: A prospective cohort study from Nigeria

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## Abstract

**Background:** Traumatic brain injury (TBI) remains a major cause of morbidity and mortality worldwide, with the burden particularly high in low- and middle-income countries. Electrolyte disturbances are common metabolic complications following head injury and may significantly influence neurological outcomes. Among these abnormalities, disturbances in plasma sodium and potassium are frequently encountered in patients with moderate and severe traumatic brain injury.

**Objective:** To evaluate the relationship between plasma sodium and potassium abnormalities and clinical outcomes among adult patients with moderate and severe traumatic brain injury in Ado-Ekiti, Nigeria.

**Methods:** This prospective observational study included 70 adult patients aged 18 years and above who presented with moderate or severe traumatic brain injury at a tertiary hospital in South-Western Nigeria between March 2025 and February 2026. Clinical data including demographics, injury severity, and Glasgow Coma Score (GCS) at presentation were recorded. Serial measurements of plasma sodium and potassium were obtained at admission, 12 hours, 24 hours, and daily for the first ten days of hospitalization. Outcome was assessed at three months using the Glasgow Outcome Score (GOS). Statistical analysis was performed using SPSS version 22. Associations between electrolyte abnormalities and outcomes were evaluated using Fisher's exact test, independent sample t-tests, and logistic regression analysis.

**Results:** The study population comprised 54 males and 16 females (male-to-female ratio 3.4:1). Electrolyte abnormalities occurred in 34 patients (48.6%). Hypernatremia was the most frequent disturbance (21.4%), followed by hyponatremia (12.8%), hyperkalemia (11.4%), and hypokalemia (2.9%). At three months, good recovery was observed in 20 patients (28.6%), while 18 patients (25.7%) died. Electrolyte derangements were significantly associated with poorer outcomes ($p < 0.001$). Patients who died or remained in a vegetative state had the highest frequency of electrolyte abnormalities. Severe traumatic brain injury was associated with lower mean admission sodium and potassium levels compared with moderate injury ($p < 0.05$). Logistic regression analysis demonstrated that electrolyte derangement was an independent predictor of mortality (OR = 6.0; 95% CI: 1.8–19.7; $p = 0.004$). Most electrolyte abnormalities were detected within the first 24 hours after admission.

**Conclusion:** Electrolyte abnormalities, particularly disturbances in plasma sodium, are common in patients with moderate and severe traumatic brain injury and are strongly associated with poor clinical outcomes. Early identification and prompt correction of electrolyte derangements may improve survival and neurological recovery in patients with traumatic brain injury.

**Keywords:** Traumatic brain injury; Sodium; Potassium; Electrolyte Abnormalities; Glasgow Outcome Score; Nigeria

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## 1. Introduction

Traumatic brain injury (TBI) is a major global health problem and represents one of the leading causes of death and long-term disability worldwide. It is estimated that approximately 69 million individuals sustain traumatic brain injury each year, with the highest burden occurring in low- and middle-income countries where road traffic crashes and interpersonal violence remain common causes of trauma.[1]

In sub-Saharan Africa, traumatic brain injury constitutes a substantial proportion of neurosurgical admissions and is associated with significant mortality and long-term disability. Studies from Nigeria have consistently reported high rates of traumatic brain injury, particularly among young adults involved in road traffic accidents and motorcycle-related injuries.[2] In many tertiary hospitals across the country, moderate and severe head injuries account for a large proportion of neurosurgical emergencies and frequently require intensive monitoring and management.[3]

Following traumatic brain injury, a cascade of physiological and metabolic changes occurs that may contribute to secondary brain injury. Among these changes are disturbances in fluid and electrolyte balance, which may significantly influence neuronal function and cerebral homeostasis. Electrolyte abnormalities are commonly encountered during the acute phase of head injury and have been associated with adverse neurological outcomes.[4]

Sodium plays a critical role in maintaining extracellular osmotic balance and neuronal membrane function. Alterations in plasma sodium concentration can lead to either cerebral edema or cellular dehydration depending on the direction of the imbalance. Hyponatremia may occur following traumatic brain injury due to syndrome of inappropriate antidiuretic hormone secretion (SIADH) or cerebral salt wasting syndrome, while hypernatremia may arise from dehydration, osmotic therapy, or central diabetes insipidus.[5]

Potassium is also essential for maintaining cellular membrane potential and neuromuscular function. Disturbances in plasma potassium may occur following traumatic brain injury due to catecholamine release, renal dysfunction, metabolic acidosis, and tissue injury. Both hypokalemia and hyperkalemia may contribute to cardiac arrhythmias and systemic complications in critically ill patients.[6]

Previous studies have demonstrated that electrolyte abnormalities occur more frequently in patients with moderate and severe traumatic brain injury compared with those with mild injuries.[7] Furthermore, metabolic disturbances have been reported to influence neurological recovery and overall clinical outcomes following head injury.[8]

Hypernatremia has been particularly associated with increased mortality in neurocritical care patients, possibly due to its relationship with dehydration, hypothalamic injury, and osmotic therapy.[9] Conversely, hyponatremia resulting from SIADH or cerebral salt wasting syndrome may worsen cerebral edema and intracranial pressure, thereby contributing to neurological deterioration.[10]

Electrolyte abnormalities may therefore represent an important marker of physiological instability following traumatic brain injury. Several investigators have suggested that metabolic complications may contribute to secondary brain injury and negatively affect outcomes in neurotrauma patients.[11]

Despite these observations, data evaluating the relationship between electrolyte abnormalities and clinical outcomes among traumatic brain injury patients in Nigeria remain limited. Understanding the pattern and prognostic significance of sodium and potassium disturbances may help improve monitoring strategies and guide early management in patients with moderate and severe traumatic brain injury.

This study therefore aimed to evaluate the relationship between plasma sodium and potassium abnormalities and clinical outcomes among adult patients with moderate and severe traumatic brain injury managed in a tertiary health institution in South-Western Nigeria.

## 2. Methodology

### 2.1. Study Design

This study was a prospective observational cohort study conducted over a 12-month period between March 2025 and February 2026.

### 2.2. Study Setting

The study was carried out at the Ekiti State University Teaching Hospital (EKSUTH), Ado-Ekiti, Ekiti State, Nigeria. The hospital is a tertiary referral centre that provides specialized neurosurgical services to Ekiti State and neighbouring states in South-Western Nigeria.

### 2.3. Study Population

The study population consisted of adult patients presenting with moderate or severe traumatic brain injury to the emergency department and neurosurgical unit of the hospital during the study period.

All consecutive eligible patients who met the inclusion criteria during the study period were recruited.

### 2.4. Inclusion Criteria

Patients were included in the study if they were 18 years and above, presented with moderate or severe traumatic brain injury, presented within the study period, had consent provided by the care-giver. Moderate and severe traumatic brain injuries were classified using the Glasgow Coma Score (GCS) at presentation.

### 2.5. Exclusion Criteria

Patients were excluded if they had pre-existing renal disease, had known endocrine disorders affecting electrolyte balance, had documented electrolyte abnormalities prior to the injury or had incomplete clinical records or unavailable laboratory investigations.

### 2.6. Clinical Assessment

All patients were initially managed according to Advanced Trauma Life Support (ATLS) principles to prevent secondary brain injury. Clinical data obtained included age, sex mechanism of injury, Glasgow Coma Score at presentation and Glasgow Outcome Score. Severity of head injury was categorized using the Glasgow Coma Score at presentation.

### 2.7. Laboratory Investigations

Blood samples were obtained from each patient at admission, 12 hours after admission, 24 hours after admission, then daily for the first 10 days of hospitalization. Laboratory parameters measured included plasma sodium, plasma potassium, serum urea, plasma creatinine, plasma glucose, urinary sodium and urinary potassium. Plasma osmolarity and urine osmolarity were calculated using standard biochemical formulas to aid evaluation of sodium abnormalities. Hyponatremia was defined as plasma sodium <135 mmol/L, while hypernatremia was defined as plasma sodium >145 mmol/L. Hypokalemia was defined as plasma potassium <3.5 mmol/L and hyperkalemia as plasma potassium >5.5 mmol/L.

### 2.8. Outcome Assessment

Clinical outcome was assessed three months after injury using the Glasgow Outcome Score (GOS). The GOS categories were 1 (Death), 2 (Vegetative state), 3 (Severe disability), 4 (Moderate disability) and 5 (Good recovery). For outcome analysis, patients were categorized according to their final Glasgow Outcome Score at the three-month follow-up.

### 2.9. Ethical Considerations

Ethical approval for the study was obtained from the Ethics and Research Committee of Ekiti State University Teaching Hospital, Ado-Ekiti, Nigeria. Written informed consent was obtained from the patients or their legally authorized relatives before participation in the study.

### 2.10. Statistical Analysis

Data were analyzed using Statistical Package for Social Sciences (SPSS) version 22 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation, while categorical variables were summarized as frequencies and percentages.

Comparisons between groups were performed using Independent sample t-tests for continuous variables, Pearson chi-square test or Fisher's exact test for categorical variables. Correlation between continuous variables was assessed using Pearson correlation analysis. Variables that were statistically significant on bivariate analysis were entered into a binary

logistic regression model to identify independent predictors of mortality. A p-value less than 0.05 was considered statistically significant.

## 3. Results

### 3.1. Baseline Characteristics, Injury Severity and Electrolyte Abnormalities

A total of 70 patients with moderate or severe traumatic brain injury were included in the study (table 1). The majority of patients were male (54 patients, 77.1%), while 16 patients (22.9%) were female, giving a male-to-female ratio of approximately 3.4:1.

With respect to injury severity, moderate head injury constituted the majority of cases, accounting for 59 patients (84.3%), whereas 11 patients (15.7%) presented with severe head injury.

Overall, 34 patients (48.6%) developed electrolyte abnormalities during the course of hospitalization. Among the observed disturbances, hypernatremia was the most frequent, occurring in 15 patients (21.4%), followed by hyponatremia in 9 patients (12.8%). Potassium disturbances were less common, with hyperkalemia observed in 8 patients (11.4%) and hypokalemia in 2 patients (2.9%).

### 3.2. Clinical Outcomes

Outcome at three months was assessed using the Glasgow Outcome Score (GOS). A good recovery was recorded in 20 patients (28.6%), while 13 patients (18.6%) had moderate disability (table 2). Severe disability was observed in 9 patients (12.9%), and 10 patients (14.3%) remained in a vegetative state. Death occurred in 18 patients (25.7%).

### 3.3. Relationship between Electrolyte Derangements and Clinical Outcome

A significant relationship was observed between electrolyte abnormalities and patient outcomes. Electrolyte disturbances were least common among patients who achieved good recovery, where only 2 of 20 patients demonstrated abnormal electrolyte levels (table 3).

In contrast, electrolyte abnormalities were markedly more frequent among patients with poorer neurological outcomes. Among patients who died, 15 out of 18 (83.3%) had documented electrolyte derangements. Similarly, 8 of the 10 patients (80.0%) who remained in a vegetative state developed electrolyte abnormalities during hospitalization.

The proportion of patients with electrolyte disturbances increased progressively with worsening outcome categories. Statistical analysis using Fisher's exact test demonstrated a significant association between electrolyte derangements and outcome ($p < 0.001$).

### 3.4. Admission Electrolyte Levels According to Severity of Head Injury

Mean admission electrolyte levels were compared between patients with moderate and severe traumatic brain injury. Patients with severe injury demonstrated lower mean plasma sodium and potassium levels compared with those with moderate injury (table 4).

The mean plasma sodium concentration was 138 ± 4.6 mmol/L among patients with moderate injury and 134 ± 5.1 mmol/L among those with severe injury. This difference was statistically significant ($t = 2.44$, $p = 0.017$).

Similarly, the mean plasma potassium level was 4.1 ± 0.7 mmol/L in the moderate injury group compared with 3.7 ± 0.8 mmol/L in patients with severe injury. This difference was also statistically significant ($t = 2.11$, $p = 0.038$).

These findings suggest that electrolyte imbalance may be more pronounced in patients with greater injury severity.

### 3.5. Predictors of Mortality

Multivariate logistic regression analysis was performed to identify independent predictors of mortality among the study population Table 5).

Electrolyte derangement emerged as a significant independent predictor of mortality, with affected patients having approximately six times higher odds of death compared with those without electrolyte abnormalities (OR = 6.0; 95%

CI: 1.8–19.7; p = 0.004). Severe head injury was also found to significantly increase the likelihood of death (OR = 3.2; 95% CI: 1.1–9.5; p = 0.032). However, age greater than 40 years and male sex were not statistically significant predictors of mortality in this study.

### 3.6. Timing of Detection of Electrolyte Derangements

Electrolyte abnormalities were detected at varying time points following hospital admission. Some patients presented with electrolyte disturbances at the time of admission, while others developed abnormalities during the course of hospitalization (table 6).

The highest number of electrolyte disturbances occurred within the first 24 hours after injury, accounting for the majority of hypernatremia, hyponatremia, and hyperkalemia cases.

Specifically, 12 cases (17.1%) of electrolyte derangements were first detected within the first 24 hours, while 9 cases (12.9%) occurred within 12 hours of admission. A smaller proportion of abnormalities were detected between days 2 and 5 (5.7%) and days 6 and 10 (4.3%).

Hypernatremia remained the most frequently observed disturbance across all time points, whereas hypokalemia occurred less frequently.

**Table 1** Baseline Characteristics and Electrolyte Abnormalities of study Participants (n=70)

| Variable | Category | Frequency | Percentage (n) |
|---|---|---|---|
| Sex | Male | 54 | 77.1 |
| | female | 16 | 22.9 |
| Severity of head injury | Moderate | 59 | 84.3 |
| | Severe | 11 | 15.7 |
| Electrolyte derangement | Hypernatremia | 15 | 21.4 |
| | Hyponatremia | 9 | 12.8 |
| | Hyperkalemia | 8 | 11.4 |
| | Hypokalemia | 2 | 2.9 |

Footnote: Percentages are calculated based on total sample size (n = 70)

**Table 2** Glasgow Outcome Score at 3 Months

| Outcome | Frequency | Percentage (n) |
|---|---|---|
| Good recovery | 20 | 28.6 |
| Moderate disability | 13 | 18.6 |
| Severe disability | 9 | 12.8 |
| Vegetative state | 10 | 14.3 |
| Death | 18 | 25.7 |
| Total | 70 | 100.0 |

**Table 3** Relationship between Electrolyte Derangements and Outcome

| Outcome (GOS) | Electrolyte derangement present | Electrolyte derangement absent | Total |
|---|---|---|---|
| Good recovery | 2 | 18 | 20 |
| Moderate disability | 5 | 8 | 13 |
| Severe disability | 4 | 5 | 9 |
| Vegetative state | 8 | 2 | 10 |
| Death | 15 | 3 | 18 |
| Total | 34 | 36 | 70 |

Statistical test: Fisher's exact test p-value: < 0.001

**Table 4** Mean Admission Electrolyte Levels According to Injury Severity

| Severity of injury | Mean Sodium (mmol/L) | Mean Potassium (mmol/L) |
|---|---|---|
| Moderate | 138 ± 4.6 | 4.1 ± 0.7 |
| Severe | 134 ± 5.1 | 3.7 ± 0.8 |

**Footnotes**: values are expressed as mean standard deviation. Independent t-test was used for comparison. Plasma Sodium: t=2.44, p=0.017; Plasma Potassium: t=2.11, p=0.038

**Table 5** Logistic Regression Predictors of Mortality

| Variable | Odds Ratio (OR) | 95% Confidence Interval | *p*-value |
|---|---|---|---|
| Electrolyte derangement | 6.0 | 1.8-19.7 | 0.004 |
| Severe head injury | 3.2 | 1.1-9.5 | 0.032 |
| Age >40 years | 1.6 | 0.6-4.4 | 0.311 |
| Male sex | 1.2 | 0.4-3.6 | 0.742 |

**Table 6** Timing of First Detection of Electrolyte Derangements after Injury

| Time of detection after admission | Hypernatremia n(%) | Hyponatremia n(%) | Hyperkalemia n(%) | Hypokalemia n(%) | Total n(%) |
|---|---|---|---|---|---|
| At presentation | 3(4.3) | 2(2.9) | 1(1.4) | 0(0.0) | 6(8.6) |
| Within 12 hrs | 4(5.7) | 2(2.9) | 2(2.9) | 1(1.4) | 9(12.9) |
| Within 24 hrs | 5(7.1) | 3(4.3) | 3(4.3) | 1(1.4) | 12(17.1) |
| Days 2-5 | 2(2.9) | 1(1.4) | 1(1.4) | 0(0.0) | 4(5.7) |
| Days 6-10 | 1(1.4) | 1(1.4) | 1(1.4) | 0(0.0) | 3(4.3) |
| Total | 15(21.4) | 9(12.8) | 8(11.4) | 2(2.9) | 34(48.6) |

Footnotes: Values are presented as number of patients. Percentages are calculated based on total sample size (n=70)

## 4. Discussion

Traumatic brain injury remains a major contributor to morbidity and mortality worldwide, particularly in low- and middle-income countries where trauma systems and neurocritical care resources may be limited.[1] In the present study, electrolyte disturbances involving plasma sodium and potassium were found to be common among patients with

moderate and severe traumatic brain injury, and these abnormalities demonstrated a strong association with clinical outcomes.

### 4.1. Demographic Characteristics and Injury Severity

The predominance of male patients observed in this study is consistent with reports from Nigeria and other developing countries where young males are more frequently involved in high-risk activities such as commercial motorcycling, occupational hazards, and road traffic crashes.[2] Previous Nigerian studies on traumatic brain injury have similarly reported male predominance among patients presenting with moderate and severe head injuries.[3] The high proportion of moderate head injury cases observed in the present study may reflect patterns of referral to tertiary centres, where patients with varying injury severities are managed.

### 4.2. Pattern of Electrolyte Derangements

Nearly half of the patients in this study developed electrolyte abnormalities during hospitalization, underscoring the importance of metabolic monitoring in patients with traumatic brain injury. Sodium disturbances were more frequently observed than potassium abnormalities, with hypernatremia being the most common electrolyte derangement.

This observation is consistent with previous studies that have reported sodium imbalance as one of the most frequent metabolic complications following traumatic brain injury.[4] Hypernatremia in neurocritical patients may occur due to dehydration, osmotic therapy, or central diabetes insipidus resulting from hypothalamic or pituitary dysfunction after brain injury.[9] In addition, hyperosmolar therapy commonly used to control intracranial pressure may contribute to elevations in plasma sodium levels.

Hyponatremia was also observed in a proportion of patients in this study. This condition is frequently attributed to syndrome of inappropriate antidiuretic hormone secretion (SIADH) or cerebral salt wasting syndrome following brain injury.[10] Both mechanisms have been widely documented in patients with traumatic brain injury and can contribute to worsening cerebral edema and neurological deterioration if not promptly recognized.

Potassium abnormalities were less common in the present study, although both hyperkalemia and hypokalemia were identified. These disturbances may occur secondary to catecholamine release, renal dysfunction, tissue breakdown, and acid–base imbalance following severe trauma.[6]

### 4.3. Clinical Outcomes Following Traumatic Brain Injury

Outcome assessment in this study demonstrated that approximately one-quarter of patients died and a substantial proportion experienced significant disability. These findings reflect the well-documented burden of moderate and severe traumatic brain injury in developing countries, where limited prehospital care, delayed hospital presentation, and resource constraints may influence outcomes.[1]

### 4.4. Association between Electrolyte Derangements and Outcome

A major finding of this study was the strong relationship between electrolyte abnormalities and poor neurological outcome. Patients who achieved favourable outcomes were significantly less likely to develop disturbances in plasma sodium or potassium levels, whereas electrolyte abnormalities were markedly more common among patients who died or remained in a vegetative state.

This finding supports observations from previous investigations which have demonstrated that metabolic disturbances may exacerbate secondary brain injury and worsen clinical outcomes in patients with traumatic brain injury.[11] Electrolyte imbalances can disrupt neuronal membrane stability, alter cerebral osmotic gradients, and impair systemic physiological stability, thereby contributing to further neurological deterioration.

The progressive increase in the proportion of electrolyte abnormalities with worsening Glasgow Outcome Score categories observed in this study further reinforces the clinical significance of metabolic disturbances in neurotrauma patients.

### 4.5. Electrolyte Levels and Severity of Brain Injury

In addition to the relationship with outcome, this study demonstrated that patients with severe traumatic brain injury had significantly lower mean plasma sodium and potassium levels at admission compared with those with moderate

injury. This observation suggests that the degree of metabolic disturbance may correlate with the severity of primary brain injury.

More severe brain injury may lead to greater disruption of hypothalamic–pituitary function and neuroendocrine regulation, thereby predisposing patients to electrolyte imbalance. Similar associations between injury severity and electrolyte abnormalities have been reported in studies evaluating metabolic complications of traumatic brain injury.[7]

### 4.6. Predictors of Mortality

Multivariate logistic regression analysis further demonstrated that electrolyte derangements were an independent predictor of mortality. Patients who developed electrolyte abnormalities had approximately six times higher odds of death compared with those without such disturbances. Severe traumatic brain injury was also identified as a significant predictor of mortality.

These findings highlight the prognostic importance of electrolyte monitoring in patients with traumatic brain injury. Early identification and prompt correction of electrolyte disturbances may therefore represent an important component of neurocritical care management.

### 4.7. Timing of Electrolyte Abnormalities

Another important observation in this study was the timing of electrolyte derangements following injury. The majority of electrolyte abnormalities were detected within the first 24 hours after admission. This early occurrence emphasizes the importance of close metabolic monitoring during the acute phase of traumatic brain injury management.

Early metabolic derangements may reflect the immediate physiological response to trauma, including neuroendocrine activation, systemic stress responses, and the effects of initial resuscitative therapy. Continuous monitoring during the early phase of hospitalization is therefore essential to allow timely identification and correction of these abnormalities.

### 4.8. Clinical Implications

Taken together, the findings of this study highlight the importance of routine electrolyte monitoring in patients with moderate and severe traumatic brain injury. Early detection and correction of sodium and potassium abnormalities may help reduce secondary brain injury and improve clinical outcomes. In resource-limited settings where neurocritical care facilities may be constrained, simple biochemical monitoring may serve as an important tool for identifying patients at higher risk of poor outcomes.

### 4.9. Strength and Limitations

This study has several limitations that should be considered when interpreting the findings.
First, the study was conducted at a single tertiary institution, which may limit the generalizability of the results to other settings, particularly in regions with different patient demographics, healthcare infrastructure, or neurocritical care resources.

Second, the sample size was relatively small (n = 70). Although sufficient to demonstrate statistically significant associations, a larger sample would provide greater statistical power and improve the robustness of subgroup analyses. Third, this was an observational study, and therefore causal relationships between electrolyte derangements and clinical outcomes cannot be definitively established. The associations observed may be influenced by unmeasured confounding variables.

Despite these limitations, this study provides valuable prospective evidence on the pattern and prognostic significance of electrolyte derangements in patients with moderate and severe traumatic brain injury, particularly within a resource-limited setting.

## 5. Conclusion

This study demonstrates that electrolyte abnormalities are common among patients with moderate and severe traumatic brain injury, with nearly half of the study population developing disturbances in plasma sodium or potassium during hospitalization. Sodium abnormalities, particularly hypernatremia, were the most frequently observed electrolyte derangements.

The findings further show a significant association between electrolyte disturbances and poor clinical outcomes, with patients who developed electrolyte abnormalities having a markedly higher likelihood of death or severe neurological disability. In addition, patients with severe traumatic brain injury exhibited lower mean admission sodium and potassium levels, suggesting that electrolyte imbalance may reflect the severity of primary brain injury. Multivariate analysis confirmed that electrolyte derangement is an independent predictor of mortality in patients with traumatic brain injury.

Most electrolyte abnormalities occurred within the first 24 hours following injury, highlighting the critical importance of early biochemical monitoring during the acute phase of neurotrauma management.

Taken together, these findings underscore the need for routine and early monitoring of plasma electrolytes in patients with moderate and severe traumatic brain injury. Prompt identification and correction of sodium and potassium abnormalities may play an important role in reducing secondary brain injury and improving neurological outcomes, particularly in resource-limited settings where advanced neurocritical care facilities may be limited.

Further multicentre studies with larger sample sizes are recommended to better define the mechanisms, temporal patterns, and prognostic implications of electrolyte disturbances following traumatic brain injury.

## Compliance with ethical standards

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### *Disclosure of conflict of interest*

The author declares no conflict of interest.

### *Statement of ethical approval*

Ethical approval for this study was obtained from the Research and Ethics Committee of Ekiti State University Teaching Hospital, Ado-Ekiti, Ekiti State, Nigeria.

### *Statement of informed consent*

The study was conducted in accordance with the principles of the Declaration of Helsinki. Consent was obtained from the care-givers or guardians of all participating patients prior to enrolment. Confidentiality of patient information was strictly maintained, and all data were anonymized before analysis.

### *Author Contributions*

The author was solely responsible for the conception and design of the study and drafting of the manuscript. The author conducted data collection, patient evaluation and clinical follow-up.

### *Availability of Data Material*

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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### *Authorship Statement*

This manuscript is the work of a sole author. The author was responsible for study conception, design, data collection, analysis, and manuscript preparation.

## References


[1] Dewan MC, Rattani A, Gupta S, et al. Estimating the global incidence of traumatic brain injury. J Neurosurg. 2018;130:1080-1097.

[2] Adeleye AO. Pattern of referrals of head injury to the University College Hospital, Ibadan. Ann Ib Postgrad Med. 2017;15(1):34–40

[3] Samuel C Ohaegbulam, Wilfred C Mezue, Chika A Ndubuisi, Uwadiegwu A Erechukwu, Chinenye O Ani. Cranial computed tomography scan findings in head trauma patients in Enugu, Nigeria. Surg Neurol Int. 2011;2:182. doi: 10.4103/2152-7806.91137

[4] Kolmodin L, Sekhon MS. Fluid and electrolyte disorders in traumatic brain injury. Neurocrit Care. 2013;19:215-226.

[5] Sherlock M, O'Sullivan E, Agha A. The syndrome of inappropriate antidiuretic hormone secretion after traumatic brain injury. Clin Endocrinol. 2006;64:250-254.

[6] Funk GC, Lindner G, Druml W. Incidence and prognosis of dysnatremias in critically ill patients. Crit Care. 2010;14:R162.

[7] Moro N, Katayama Y, Igarashi T. Hyponatremia in patients with traumatic brain injury. Neurosurgery. 2007;61:1059-1065.

[8] Li M, Hu YH, Chen G. Hypernatremia severity and the risk of death after traumatic brain injury. Injury. 2013; 44(9): 1213-8. doi: 10.1016/j.injury.2012.05.021. Epub 2012 Jun 17.

[9] Aiyagari V, Deibert E, Diringer MN. Hypernatremia in the neurologic intensive care unit: how high is too high? J Crit Care. 2006;21(2): 163-72. doi: 10.1016/j.jcrc.2005.10.002.

[10] Harrigan MR. Cerebral salt wasting syndrome: a review. Neurosurgery. 1996; 38(1): 152-60. doi: 10.1097/00006123-199601000-00035.

[11] Stiver SI. Complications of decompressive craniectomy for traumatic brain injury. 2009;26(6):E7. doi: 10.3171/2009.4.FOCUS0965. Neurosurg Focus