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Plasma Sodium Abnormalities Predict Mortality and Poor Functional Outcome After Adult Traumatic Brain Injury

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Abstract:

- **Background:**
Traumatic brain injury (TBI) remains a major cause of morbidity and mortality worldwide. In addition to the primary mechanical injury, secondary physiological disturbances frequently complicate the clinical course of affected patients. Among these disturbances, abnormalities in plasma sodium concentration are particularly important because of their effects on cerebral osmotic balance and neuronal function. However, data regarding the prognostic significance of sodium abnormalities among patients with traumatic brain injury in Nigeria remain limited.

- **Objective:**
This study evaluated the prevalence of plasma sodium abnormalities and their relationship with injury severity, mortality, and functional outcome among adult patients with traumatic brain injury.

- **Methods:**
This prospective observational study included 70 adult patients with moderate or severe traumatic brain injury managed at a tertiary hospital in Ado-Ekiti, Nigeria. Plasma sodium levels were measured at admission, 12 hours, 24 hours, and daily for the first ten days of hospitalization. Sodium abnormalities were defined as hyponatremia (<135 mmol/L) and hypernatremia (>145 mmol/L). Clinical outcome was assessed at three months using the Glasgow Outcome Score (GOS). Associations between sodium abnormalities and clinical outcomes were analyzed using chi-square tests and logistic regression analysis.

- **Results:**
The study population comprised 54 males and 16 females (male-to-female ratio of 3.4:1). Plasma sodium abnormalities occurred in 24 patients (34.3%). Hypernatremia was observed in 15 patients (21.4%), while 9 patients (12.9%) developed hyponatremia. Sodium abnormalities were significantly associated with poorer clinical outcomes. Among patients who died, 13 (72.2%) had abnormal sodium levels compared with 5 (27.8%) who had normal sodium levels ($p < 0.001$). Logistic regression analysis demonstrated that plasma sodium abnormalities independently predicted mortality (OR 4.8, 95% CI 1.6–14.3, $p = 0.005$). Most sodium abnormalities were detected within the first 24 hours after admission.

- **Conclusion:**
Plasma sodium abnormalities are common among patients with moderate and severe traumatic brain injury and are strongly associated with unfavorable clinical outcomes and increased mortality. Early detection and appropriate correction of dysnatremia may represent an important strategy for improving neurological outcomes in patients with traumatic brain injury.

Keywords: *Traumatic Brain Injury, Dysnatremia, Hypernatremia, Hyponatremia, Mortality, Neurological Outcome.*

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I. INTRODUCTION

Traumatic brain injury (TBI) represents a major global public health problem and remains a leading cause of morbidity and mortality [1].

In sub-Saharan Africa, traumatic brain injury constitutes a substantial proportion of trauma-related morbidity and mortality. Limited access to prehospital care, delayed hospital presentation, and resource constraints often contribute to poorer outcomes compared with high-income countries [2].

Within Nigeria, traumatic brain injury represents a major component of neurosurgical practice. Hospital-based studies have shown that head injuries account for a large proportion of emergency neurosurgical admissions, with road traffic crashes being the most common mechanism of injury [3].

Beyond the primary mechanical damage to brain tissue, secondary physiological disturbances contribute significantly to neurological deterioration following traumatic brain injury. These secondary insults include hypoxia, hypotension, metabolic derangements, and electrolyte abnormalities [4].

Among these metabolic disturbances, disorders of sodium balance are particularly important because of their influence on neuronal function and cerebral osmotic regulation. Alterations in plasma sodium concentration can lead to shifts in intracellular and extracellular fluid balance, thereby contributing to cerebral edema or neuronal dehydration [5].

Hyponatremia following traumatic brain injury is commonly attributed to syndrome of inappropriate antidiuretic hormone secretion or cerebral salt wasting syndrome. These conditions can lead to worsening cerebral edema and deterioration in neurological status if not promptly recognized and treated [6].

Conversely, hypernatremia may occur due to dehydration, osmotic diuresis, or central diabetes insipidus following hypothalamic injury. Hypernatremia has been reported to occur frequently among neurocritical care patients and has been associated with increased mortality [7].

Electrolyte abnormalities in traumatic brain injury have been widely documented in several clinical studies. Some investigators have suggested that dysnatremia may worsen secondary brain injury by altering cerebral osmotic gradients and impairing neuronal metabolism [8].

Furthermore, abnormalities in plasma sodium concentration have been linked with poorer neurological outcomes and prolonged hospitalization among patients with traumatic brain injury [9].

In the African context, studies have shown that traumatic brain injury predominantly affects young adult males, largely due to road traffic accidents and occupational hazards [10].

Similar demographic patterns have been reported in several African countries, where traumatic brain injury remains a major contributor to trauma-related mortality [11].

Studies conducted in Nigerian neurosurgical centres have also demonstrated a high burden of traumatic brain injury and emphasized the need to identify factors that may influence patient outcomes [12].

Recognition of electrolyte disturbances is particularly important in neurocritical care because timely correction may prevent further neurological deterioration. Dysnatremia has been identified as a potentially modifiable factor in the management of neurologically injured patients [13].

Despite these observations, there remains limited data describing the prognostic significance of sodium abnormalities in patients with traumatic brain injury in Nigeria. This study therefore aimed to determine the prevalence of plasma sodium abnormalities and evaluate their relationship with injury severity, mortality, and functional outcomes among adults with traumatic brain injury managed at a tertiary hospital in south-western Nigeria.

II. METHODOLOGY

- *Study Design*

This study was a prospective observational clinical study designed to evaluate the relationship between plasma sodium abnormalities and clinical outcomes among adult patients with moderate and severe traumatic brain injury.

- *Study Setting*

The study was conducted at the Neurosurgery Unit of the Department of Surgery, Ekiti State University Teaching Hospital (EKSUTH), Ado-Ekiti, Ekiti State, Nigeria. EKSUTH is a tertiary referral center that provides neurosurgical services to Ekiti State and neighboring states in south-western Nigeria. The hospital receives trauma referrals from primary and secondary healthcare facilities as well as emergency presentations from road traffic accidents and other forms of head injury.

- *Study Population*

The study population consisted of adult patients who presented with moderate or severe traumatic brain injury and were admitted for neurosurgical management during the study period. A total of 70 patients who met the eligibility criteria were recruited consecutively.

- *Eligibility Criteria*

Patients were eligible for inclusion in the study if they were aged 18 years and above, had a diagnosis of moderate or severe traumatic brain injury based on clinical assessment, presented to the hospital within 24 hours of injury, and underwent serial biochemical monitoring during

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hospitalization.

Patients were excluded if they had pre-existing electrolyte imbalance prior to injury, chronic kidney disease or endocrine disorders affecting sodium regulation, or were on diuretic therapy before hospital presentation. In addition, patients with polytrauma involving severe systemic injuries that could independently influence electrolyte balance, as well as those with incomplete clinical or laboratory data, were excluded from the study.

➢ *Clinical Assessment*

All patients underwent detailed clinical evaluation at presentation. Information obtained included age, sex, mechanism of injury and Glasgow Coma Scale (GCS) score at admission.

The severity of traumatic brain injury was classified using the Glasgow Coma Scale (GCS) score. Moderate traumatic brain injury was defined as GCS score of 9–12 while severe traumatic brain injury was defined as GCS score ≤8.

Patients were managed according to standard neurosurgical protocols for traumatic brain injury.

➢ *Laboratory Evaluation*

Venous blood samples were collected for measurement of plasma sodium concentration at admission, 12 hours after admission, 24 hours after admission and daily for the first ten days of hospitalization.

Laboratory analysis of plasma sodium levels was performed using standard biochemical techniques in the hospital laboratory.

Sodium abnormalities were clearly defined. Hyponatremia was defined as plasma sodium concentration <135 mmol/L. Hypernatremia was defined as plasma sodium concentration >145 mmol/L.

Patients were monitored for the development of sodium abnormalities during the course of hospitalization.

➢ *Outcome Assessment*

Clinical outcomes were evaluated three months after injury using the Glasgow Outcome Score (GOS). Outcome categories included good recovery, moderate disability, severe disability, persistent vegetative state and death. For analytical purposes, outcomes were further grouped into favorable outcome defined as GOS of 4–5 and unfavorable outcome defined as GOS of 1–3.

➢ *Statistical Analysis*

Data obtained from the study were entered into a computerized database and analyzed using Statistical Package for the Social Sciences (SPSS) version 22. Descriptive statistics were used to summarize demographic and clinical variables and were presented as frequencies, percentages, and proportions. Associations between plasma sodium abnormalities and categorical variables such as injury severity and clinical outcomes were evaluated using the chi-square test. Correlation analysis was performed to assess the relationship between plasma sodium levels and clinical parameters. A multivariate logistic regression analysis was conducted to determine whether plasma sodium abnormalities independently predicted mortality after adjusting for injury severity. Statistical significance was set at $p < 0.05$. Variables included in the logistic regression model were selected based on clinical relevance and statistical significance in bivariate analysis, while taking into account the limited sample size to avoid model overfitting.

➢ *Ethical Considerations*

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

The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. Confidentiality of patient information was maintained throughout the study, and all data were anonymized prior to analysis.

III. RESULTS

➢ *Baseline Demographic and Clinical Characteristics of the Patients*

A total of 70 adult patients with moderate or severe traumatic brain injury were included in the study. The demographic and baseline clinical characteristics of the patients are summarized in Table 1.

The mean age of the patients was 36.3 ± 10.4 years. The majority of patients were within the 20-34 years age group (47.1%), followed by those aged 35-49 years (37.2%) and 50-60 years (15.7%).

Male patients predominated, accounting for 54 patients (77.1%), while 16 patients (22.9%) were female, giving a male-to-female ratio of 3.4:1.

Road traffic accidents were the most common mechanism of injury, occurring in 42 patients (60.0%), followed by falls in 15 patients (21.4%), assault in 9 patients (12.9%), and other causes in 4 patients (5.7%).

Based on the Glasgow Coma Scale score at presentation, 59 patients (84.3%) sustained moderate traumatic brain injury, whereas 11 patients (15.7%) had severe injury.

➢ *Plasma Sodium Abnormalities*

The distribution of plasma sodium levels among the study population is presented in Table 2.

Overall, 24 patients (34.3%) developed sodium abnormalities during hospitalization. Hypernatremia was more common, occurring in 15 patients (21.4%), while 9 patients (12.9%) developed hyponatremia. The remaining 46 patients (65.7%) maintained normal plasma sodium levels throughout the observation period.

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➢ *Sodium Abnormalities and Severity of Head Injury*

The relationship between sodium abnormalities and the severity of traumatic brain injury is summarized in Table 3.

Sodium derangements were observed more frequently among patients with severe traumatic brain injury. Hypernatremia was particularly common in this group, occurring in 45.5% of patients with severe injury, compared with 16.9% among those with moderate injury. Conversely, normal sodium levels were observed in 71.2% of patients with moderate injury, but in only 36.3% of patients with severe injury. Statistical analysis demonstrated a significant association between injury severity and sodium abnormalities (χ^2 test, $p = 0.032$).

➢ *Sodium Abnormalities and Clinical Outcome*

The relationship between plasma sodium abnormalities and clinical outcomes assessed using the Glasgow Outcome Score (GOS) is shown in Table 4.

Patients with sodium abnormalities were significantly more likely to experience poor neurological outcomes. Among patients who died, 13 (72.2%) had sodium abnormalities, whereas only 5 (27.8%) had normal sodium levels. Similarly, sodium abnormalities were frequently observed among patients with severe disability and vegetative state. In contrast, good recovery was predominantly observed in patients with normal sodium levels, with only 2 cases occurring among patients with dysnatremia. Overall, there was a statistically significant association between sodium abnormalities and functional outcome ($p < 0.001$).

➢ *Logistic Regression Analysis*

The results of the multivariate logistic regression analysis are presented in Table 5.

After adjusting for injury severity, plasma sodium abnormalities remained an independent predictor of mortality. Patients who developed sodium derangements had approximately fivefold higher odds of death compared with those who maintained normal sodium levels (adjusted odds ratio [aOR] = 4.8, 95% CI: 1.6–14.3, $p = 0.005$).

Severe traumatic brain injury was also independently associated with mortality. Patients with severe injury (GCS ≤8) had approximately threefold increased odds of death compared with those with moderate injury (aOR = 3.1, 95% CI: 1.0–9.2, $p = 0.040$).

➢ *Timing of Sodium Abnormalities*

The timing of the first detection of sodium abnormalities during hospitalization is presented in Table 6.

Of the 24 patients who developed dysnatremia, 5 patients (20.8%) already had abnormal sodium levels at presentation, while 6 patients (25.0%) developed abnormalities within the first 12 hours of admission. The largest proportion occurred within 24 hours, affecting 8 patients (33.3%).

A smaller number of abnormalities developed later during hospitalization, with 3 patients (12.5%) developing dysnatremia between days 2 and 5, and 3 patients (12.5%) between days 6 and 10.

These findings indicate that the majority of sodium abnormalities occurred within the first 24 hours after traumatic brain injury.

➢ *Tables of Results*

Table 1 Baseline Demographic and Injury Characteristics of Study Population (n=70)

Variable	Category	Frequency (n)	Percentage (%)
Age group	20-34	33	47.1
	35-49	26	37.2
	50-60	11	15.7
Mean age ± SD (years)		36.3 ± 10.4	
Sex	Male	54	77.1
	Female	16	22.9
Male:Female ratio		3.4 : 1	
Mechanism of injury	Road traffic accident	42	60.0
	Falls	15	21.4
	Assaults	9	12.9
	Others	9	5.7
Severity of TBI (GCS)	Moderate (9-12)	59	84.3
	Severe (3-8)	11	15.7

Table 2 Distribution of Plasma Sodium Status Among Patients (n = 70)

Sodium Status	Frequency	Percentage (%)
Normal sodium	46	65.7
Hyponatremia	9	12.9
Hypernatremia	15	21.4
Total	70	100.0

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Table 3 Relationship Between Plasma Sodium Abnormalities and Severity of Traumatic Brain Injury

Severity of Injury	Hyponatremia n (%)	Hypernatremia n (%)	Normal Sodium n (%)	Total n (%)
Moderate TBI	7 (11.9)	10 (16.9)	42 (71.2)	59 (100)
Severe TBI	2 (18.2)	5 (45.5)	4 (36.3)	11 (100)
Total	9 (12.9)	15 (21.4)	46 (65.7)	70 (100)

Chi-square test: p = 0.032

Values are presented as number of patients. Percentages are calculated based on total number of severity group (moderate or severe head injury).

Table 4 Association Between Plasma Sodium Abnormalities and Functional Outcome

Outcome (GOS)	Sodium Abnormal n (%)	Sodium Normal n (%)	Total n (%)
Good recovery	2 (7.1)	18 (42.9)	20 (28.6)
Moderate disability	4 (14.3)	9 (21.4)	13 (18.6)
Severe disability	3 (10.7)	6 (14.3)	9 (12.9)
Vegetative state	6 (21.4)	4 (9.5)	10 (14.2)
Death	13 (46.5)	5 (11.9)	18 (25.7)
Total	28 (100)	42(100)	70 (100)

Chi-square p < 0.001

Percentages are calculated based on their respective column totals.

Table 5 Logistic Regression Model for Mortality

Variable	Adjusted Odds Ratio (aOR)	95% Confidence Interval	p-Value
Sodium abnormality (Yes vs No)	4.8	1.6-14.3	0.005
Severe traumatic brain injury (GCS 3-8)	3.1	1.0-9.2	0.04

- *Model Statistics:*

✓ Outcome variable is mortality (dead vs alive at 3 months)
✓ Reference categories are normal sodium and moderate TBI (GCS 9-12)

Table 6 Timing of First Detection of Plasma Sodium Abnormality after Admission

Time after admission	Hyponatremia n (%)	Hypernatremia n (%)	Total n (%)
At presentation	2 (22.2)	3 (20.0)	5 (20.8)
Within 12 hrs	2 (22.2)	4 (26.7)	6 (25.0)
Within 24 hrs	3 (33.4)	5 (33.3)	8 (33.4)
Days 2-5	1 (11.1)	2 (13.3)	3 (12.5)
Days 6-10	1 (11.1)	1 (6.7)	2 (8.3)
Total	9 (100)	15 (100)	24 (100)

Values are presented as number of patients. Percentages are calculated by column

IV. DISCUSSION

Traumatic brain injury (TBI) remains a major global public health challenge and continues to account for substantial morbidity and mortality, particularly in low- and middle-income countries where trauma systems are still evolving [1, 2]. In such settings, secondary physiological insults often contribute significantly to neurological deterioration following the primary injury. Among these secondary factors, electrolyte disturbances-especially abnormalities of plasma sodium-have been increasingly recognized as important determinants of outcome in neurotrauma patients [4, 5]. The present study evaluated the occurrence of plasma sodium abnormalities and their relationship with injury severity and clinical outcomes among adult patients with moderate and severe traumatic brain injury.

➢ *Demographic Characteristics of Patients*

The demographic distribution observed in this study showed a clear predominance of male patients, with males accounting for over three-quarters of the study population. This pattern has been consistently reported in studies of traumatic brain injury both within Nigeria and internationally [10]. The higher incidence of TBI among men is generally attributed to greater exposure to high-risk activities such as road traffic crashes, occupational hazards, and interpersonal violence. Similar findings have been reported in several African trauma studies where young

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adult males constitute the majority of patients presenting with head injuries [11]. The predominance of moderate traumatic brain injury in the present study may reflect the pattern of hospital presentation commonly seen in tertiary centres. Patients with extremely severe injuries may succumb before reaching hospital care, whereas individuals with mild injuries are often managed in peripheral facilities or discharged without admission. Consequently, tertiary neurosurgical units frequently receive a larger proportion of moderate injury cases.

➢ *Prevalence of Plasma Sodium Abnormalities*

One of the principal findings of this study was that plasma sodium abnormalities occurred in approximately one-third of patients with traumatic brain injury. This observation confirms that disturbances in sodium balance are relatively common during the acute phase of neurotrauma.

Among the identified abnormalities, hypernatremia was more frequent than hyponatremia. Previous neurocritical care studies have also reported hypernatremia as a common metabolic complication among patients with severe brain injury [7].

Hypernatremia may develop through several mechanisms including dehydration, osmotic diuresis, hypothalamic dysfunction, or the development of central diabetes insipidus following injury to the hypothalamic-pituitary axis [7].

Hyponatremia was observed in a smaller proportion of patients in the present study. Hyponatremia in traumatic brain injury has frequently been linked to conditions such as syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting syndrome [6]. Both mechanisms result in disturbances of water and sodium balance that can exacerbate cerebral edema and worsen neurological injury if left untreated.

The relatively high frequency of sodium abnormalities observed in this study highlights the importance of routine electrolyte monitoring in patients with traumatic brain injury.

➢ *Relationship between Sodium Abnormalities and Injury Severity*

The present study demonstrated a significant association between plasma sodium abnormalities and the severity of traumatic brain injury. Dysnatremia occurred more frequently among patients with severe injuries compared with those who sustained moderate injury.

This relationship is biologically plausible because severe traumatic brain injury is more likely to involve structural damage to the hypothalamus or pituitary gland, regions that play a central role in the regulation of water and sodium balance. Disruption of neuroendocrine pathways can lead to disturbances in antidiuretic hormone secretion, which in turn may produce either hyponatremia or hypernatremia.

In addition, patients with severe traumatic brain injury often require intensive therapeutic interventions such as osmotic therapy, fluid resuscitation, and mechanical ventilation. These treatments may further influence electrolyte balance and contribute to the development of dysnatremia. Previous clinical studies have similarly reported that electrolyte disturbances occur more frequently in patients with severe neurological injury [8].

➢ *Sodium Abnormalities and Clinical Outcome*

Another important finding of this study was the significant relationship between plasma sodium abnormalities and functional outcome following traumatic brain injury. Patients who developed dysnatremia were more likely to experience unfavorable outcomes including severe disability, vegetative state, or death. The mechanisms underlying this association are likely multifactorial. Alterations in plasma sodium concentration affect cerebral osmotic gradients and can lead to abnormal fluid shifts within brain tissue. Hyponatremia may aggravate cerebral edema, while hypernatremia can result in neuronal dehydration and impaired cerebral perfusion. Both processes may exacerbate secondary brain injury and worsen neurological recovery. Previous studies have reported similar findings, indicating that abnormalities of sodium balance are associated with poorer outcomes among patients with traumatic brain injury and other neurological conditions [8, 9]. These observations suggest that dysnatremia may contribute to the cascade of secondary injury processes that occur after the initial traumatic insult.

➢ *Independent Prediction of Mortality*

The present study demonstrated that plasma sodium abnormalities independently predict mortality among patients with moderate and severe traumatic brain injury, even after adjustment for injury severity. Patients who developed dysnatremia had nearly fivefold higher odds of death compared with those who maintained normal sodium levels. This finding underscores the clinical significance of sodium imbalance as more than a mere biochemical disturbance, suggesting that it may play an active role in the cascade of secondary brain injury.

The persistence of this association after controlling for injury severity is particularly important. Traumatic brain injury severity, as reflected by the Glasgow Coma Scale, is a well-established determinant of outcome. The fact that sodium abnormalities remained independently associated with mortality indicates that dysnatremia contributes additional prognostic information beyond that explained by the primary injury alone. This supports the concept that secondary physiological insults-including electrolyte disturbances-can significantly influence patient trajectory [4, 5].

Several pathophysiological mechanisms may explain this relationship. Hyponatremia can exacerbate cerebral edema through osmotic shifts, thereby increasing intracranial pressure and impairing cerebral perfusion [5, 6]. Conversely, hypernatremia may lead to neuronal dehydration, disruption of cellular metabolism, and reduced

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cerebral blood flow [7]. Both conditions can aggravate ongoing neuronal injury and contribute to poorer outcomes. In addition, sodium derangements may reflect underlying neuroendocrine dysfunction, including hypothalamic or pituitary injury, which is more common in severe forms of traumatic brain injury [6, 9].

The independent association observed in this study is consistent with findings from previous neurocritical care research, where dysnatremia-particularly hypernatremia-has been linked to increased mortality among patients with acute brain injury [7, 8]. These studies similarly suggest that disturbances in sodium balance are not merely epiphenomena but may represent clinically relevant contributors to adverse outcomes.

From a clinical perspective, these findings have important implications. Unlike the primary injury, which is often irreversible at presentation, sodium abnormalities represent a potentially modifiable factor. Early detection through routine electrolyte monitoring and timely correction of dysnatremia may therefore offer an opportunity to mitigate secondary brain injury and improve survival. This is particularly relevant in resource-limited settings, where optimizing basic physiological parameters may have a substantial impact on outcomes [13].

However, the interpretation of these findings should be made with caution. The regression model included a limited number of variables due to the relatively small sample size and number of outcome events, which constrained the extent of adjustment for potential confounders. As a result, the possibility of residual confounding cannot be entirely excluded. Future studies with larger sample sizes and more comprehensive modeling are needed to further elucidate the independent contribution of sodium abnormalities to mortality in traumatic brain injury.

- *Timing of Sodium Abnormalities*

Another notable observation from this study was that most sodium abnormalities were detected within the first 24 hours after hospital admission. This finding suggests that metabolic disturbances frequently develop during the early phase following traumatic brain injury.

Several physiological mechanisms may account for this early occurrence of dysnatremia. Acute neuroendocrine responses to traumatic brain injury can disrupt the normal regulation of water and electrolyte balance. In addition, early resuscitative interventions and osmotic therapies may influence sodium levels during the first days after injury.

The early timing of sodium abnormalities observed in this study emphasizes the importance of frequent electrolyte monitoring during the initial stages of patient management. Early detection of dysnatremia may allow timely therapeutic intervention and potentially reduce the risk of secondary brain injury.

- *Clinical Implications*

The findings of this study have several important implications for the clinical management of patients with traumatic brain injury. First, the relatively high prevalence of sodium abnormalities underscores the need for routine electrolyte monitoring in patients with moderate and severe traumatic brain injury.

Second, the association between dysnatremia and poor clinical outcomes suggests that disturbances in sodium balance may serve as useful prognostic indicators during the acute phase of neurotrauma.

Finally, the observation that most sodium abnormalities occur within the first 24 hours highlights the need for close biochemical surveillance during the early stages of hospitalization.

- *Study Limitations*

This study has several limitations that should be considered when interpreting the findings. First, the study was conducted at a single tertiary healthcare institution, which may limit the generalizability of the results to other settings. The sample size was relatively modest, potentially reducing the statistical power to detect smaller effect sizes.

Second, as an observational study, the findings demonstrate associations rather than causal relationships between plasma sodium abnormalities and clinical outcomes. Although multivariate analysis was performed, residual confounding from unmeasured variables-such as variations in fluid therapy, medication use, and critical care interventions-may still be present.

Third, the timing and frequency of biochemical measurements, although systematic, may not have fully captured transient fluctuations in sodium levels, and laboratory variability may have influenced measured values. In addition, early mortality in some patients may have limited the observation of later-onset sodium abnormalities.

Finally, functional outcome assessment using the Glasgow Outcome Score at three months, while widely accepted, may be subject to some degree of subjectivity and may not fully capture long-term neurological recovery.

Despite these limitations, the study provides valuable prospective data on the prevalence and prognostic significance of plasma sodium abnormalities among patients with traumatic brain injury in a resource-limited setting.

- *Future Research*

Further research involving larger multicenter studies would be valuable in confirming the prognostic significance of sodium abnormalities in traumatic brain injury. Future investigations should also examine the impact of early correction of dysnatremia on neurological outcomes and survival among patients with traumatic brain injury.

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V. CONCLUSION

Plasma sodium abnormalities are common among patients with moderate and severe traumatic brain injury and are significantly associated with mortality and unfavorable functional outcomes. Patients with dysnatremia were more likely to experience severe disability, vegetative state, or death compared with those with normal sodium levels. These findings highlight the importance of routine monitoring of plasma sodium levels during the acute management of traumatic brain injury. Early recognition and appropriate correction of sodium abnormalities may represent a modifiable factor capable of improving outcomes in patients with traumatic brain injury, particularly in resource-limited settings.

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- *Conflict of Interest Statement*

The author declares no conflict of interest.

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- *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.

- *Ethical Approval Statement*

Ethical approval was obtained from the Hospital Research and Ethics Committee prior to commencement of the study. The study was carried out in accordance with the ethical standards of the institutional research committee and the principles of the Declaration of Helsinki.

- *Data Availability Statement*

The datasets generated and analyzed during the study are available from the corresponding author upon reasonable request.

- *United Nations Declaration of Human Rights*

The author confirms that he accepts and agrees with the UN's Declaration of Human Rights.

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