

Temporal Evolution and Prognostic Significance of Sodium and Potassium Disturbances in Adult Traumatic Brain Injury

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Abstract

Background: Electrolyte disturbances are common complications in patients with traumatic brain injury (TBI) and may contribute to secondary brain injury and poor outcomes. However, their temporal pattern and prognostic significance remain incompletely characterized, particularly in resource-limited settings. **Objective:** To evaluate the frequency, temporal evolution, and clinical significance of sodium and potassium disturbances in adult patients with moderate and severe traumatic brain injury. **Methods:** This prospective observational study included 70 adult patients with moderate and severe TBI. Serial plasma sodium and potassium levels were measured from admission through the first 10 days of hospitalization. Electrolyte disturbances were categorized as hyponatremia, hypernatremia, hypokalemia, and hyperkalemia. Clinical outcomes were assessed at 12 weeks using the Glasgow Outcome Score. Associations were analyzed using chi-square tests and multivariate logistic regression. **Results:** Electrolyte disturbances occurred in 48.6% of patients. Hypernatremia was the most common abnormality (44.1%), followed by hyponatremia (26.5%) and hyperkalemia (23.5%). Most disturbances (73.5%) occurred within the first 5 days of injury. Electrolyte abnormalities were more frequent in severe TBI compared with moderate injury (81.8% vs 42.4%, $p = 0.018$). Mortality was significantly higher in patients with electrolyte disturbances (52.9% vs 5.6%, $p < 0.001$). On multivariate analysis, electrolyte derangement independently predicted mortality (adjusted OR 6.214; 95% CI 1.423–27.135; $p = 0.015$). **Conclusion:** Electrolyte disturbances are frequent, occur early after injury, and are independently associated with mortality in traumatic brain injury. Early detection and prompt correction may improve outcomes.

Keywords: Traumatic brain injury; Electrolyte imbalance; Hyponatremia; Hypernatremia; Potassium disturbance; Mortality; Neurocritical care.

1. Introduction

Traumatic brain injury (TBI) remains a major cause of mortality and long-term disability worldwide, particularly among young adults in low- and middle-income countries. Beyond the initial mechanical insult, secondary brain injury plays a critical role in determining patient outcomes and is often driven by systemic and metabolic disturbances that evolve during the course of care.

Electrolyte imbalance is among the most frequently encountered systemic complications in patients with traumatic brain injury. Alterations in plasma sodium and potassium levels may arise from a combination of hypothalamic-pituitary dysfunction, renal impairment, systemic inflammatory responses, and therapeutic interventions employed in neurocritical care. These disturbances can exacerbate neuronal injury, impair cerebral perfusion, and ultimately worsen clinical outcomes.

Hyponatremia is a well-recognized complication following brain injury and is commonly associated with the syndrome of inappropriate antidiuretic hormone secretion (SIADH) or cerebral salt-wasting syndrome (CSWS) [1]. In contrast, hypernatremia may

occur as a result of diabetes insipidus, osmotic diuresis, or inadequate fluid replacement [2]. Both conditions have been linked to increased morbidity and mortality in patients with neurological injury.

Potassium disturbances are also observed following traumatic brain injury and may result from catecholamine surge, cellular injury, and renal dysfunction [3]. Although less frequently emphasized than sodium abnormalities, potassium imbalance carries important clinical implications, particularly due to its association with cardiac arrhythmias and systemic instability.

Several studies have reported varying incidences of electrolyte disturbances in patients with traumatic brain injury. Usha et al. reported electrolyte imbalance in approximately 75% of patients [4], while Lohani et al. documented sodium disturbances in nearly half of patients with moderate and severe head injuries [5]. Dey et al. further demonstrated that electrolyte abnormalities may significantly influence neurological outcomes in this population [6]. Clinical outcomes in traumatic brain injury are commonly assessed using the Glasgow Outcome Scale, a validated tool for evaluating functional recovery following brain injury [7]. Despite these

observations, the temporal evolution of electrolyte disturbances during the acute phase of traumatic brain injury remains incompletely understood, particularly in resource-limited settings. Most existing studies focus primarily on the presence or type of electrolyte abnormality, with limited attention to the timing of onset and its potential relationship with injury severity and clinical outcomes.

A clearer understanding of when electrolyte disturbances occur, and how they relate to disease severity and mortality, may help inform monitoring strategies and guide timely interventions. This study was therefore designed to evaluate the frequency, temporal pattern, and clinical significance of sodium and potassium disturbances in adult patients with moderate and severe traumatic brain injury.

2. Methodology

2.1. Study Design and Setting

This study was designed as a prospective observational study conducted at the Neurosurgery Unit of the Department of Surgery, Ekiti State University Teaching Hospital, Ado-Ekiti, Nigeria. The institution is a tertiary referral center that provides specialized neurosurgical and critical care services to patients within the region. Eligible patients were recruited consecutively over the study period.

2.2. Study Population

The study population comprised adult patients aged 18 years and above who presented with moderate or severe traumatic brain injury. A total of 70 patients who met the eligibility criteria were enrolled consecutively during the study period.

2.3. Eligibility Criteria

Patients were considered eligible for inclusion if they were adults aged 18 years or older, presented with moderate or severe traumatic brain injury, were admitted within four days of injury, and had serial serum electrolyte measurements available during hospitalization. Patients with pre-existing renal disease, known chronic electrolyte abnormalities, or prior use of medications capable of altering electrolyte balance were excluded from the study.

2.4. Clinical Assessment

All patients underwent detailed clinical evaluation at presentation. The severity of traumatic brain injury was assessed using the Glasgow Coma Scale, and patients were classified as having moderate injury if the score ranged from 9 to 12 and severe injury if the score ranged from 3 to 8. Where feasible, computed tomography imaging of the brain was performed to identify and characterize intracranial pathology.

2.5. Treatment Protocol

Patients with moderate traumatic brain injury were managed in the neurosurgical wards, while those with severe injury were admitted to the intensive care unit for closer monitoring and supportive care. Management followed standard neurotrauma care protocols and included airway stabilization, mechanical ventilation when indicated, administration of osmotic agents for intracranial hypertension, and surgical intervention when clinically required.

2.6. Electrolyte Monitoring and Definitions

Serial measurements of plasma sodium and potassium were performed at admission, 12 hours after presentation, 24 hours after presentation, and subsequently on a daily basis from day 2 to day 10 of hospitalization. Electrolyte disturbances were defined using standard laboratory reference ranges. Hyponatremia was defined as a serum sodium level below 135 mmol/L, while hypernatremia was defined as a level above 145 mmol/L. Hypokalemia was defined as a serum potassium level below 3.5 mmol/L, and hyperkalemia as a level above 5.0 mmol/L. Patients were classified as having an electrolyte disturbance if any of these abnormalities occurred at any point during the monitoring period.

2.7. Outcome Assessment

Clinical outcomes were assessed at 12 weeks following injury using the Glasgow Outcome Scale, which categorizes outcomes into death, vegetative state, severe disability, moderate disability, and good recovery [7]. For the purpose of analysis, outcomes were further grouped into favourable outcomes, comprising moderate disability and good recovery, and unfavourable outcomes, comprising death, vegetative state, and severe disability.

2.8. Statistical Analysis

Data were entered and analyzed using the Statistical Package for the Social Sciences (SPSS) version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean \pm standard deviation, while categorical variables were presented as frequencies and percentages. Associations between categorical variables were assessed using the chi-square test or Fisher's exact test where appropriate. Multivariate logistic regression analysis was performed to identify independent predictors of mortality, with variables such as electrolyte disturbance status, injury severity, and age included in the model. The strength of associations was expressed as odds ratios with corresponding 95% confidence intervals. A p-value of less than 0.05 was considered statistically significant.

2.9. Ethical Considerations

Ethical approval for the study was obtained from the institutional ethics review committee of Ekiti State University Teaching Hospital. Informed consent was obtained from patients' care-givers or their legally authorized representatives prior to participation in the study.

3. Results

3.1. Patient Characteristics

Seventy adult patients with moderate and severe traumatic brain injury were included in this study. The mean age of the cohort was 36.3 ± 10.4 years, with nearly half of the patients (47.1%) aged between 20 and 34 years (Table 1). There was a clear male predominance, with males accounting for 77.1% of the study population, resulting in a male-to-female ratio of 3.4:1.

Most patients sustained moderate traumatic brain injury (84.3%), while 15.7% presented with severe injury at admission (Table 1).

Table 1: Baseline demographic and injury characteristics of study population (n=70)

Variable	Category	Frequency n (%)
Age group	20-34	33 (47.1)
	35-49	26 (37.2)
	50-60	11 (15.7)
Mean age \pm SD (years)	-	36.3 ± 10.4

Sex	Male	54 (77.1)
	Female	16 (22.9)
Male: Female ratio	-	3.4:1
Severity of TBI (GCS)	Moderate (9-12)	59 (84.3)
	Severe (3-8)	11 (15.7)

3.2. Clinical Presentation and Treatment Characteristics

Loss of consciousness was observed in all patients at presentation. Multiple clinical features were frequently observed in individual patients, and therefore the total number of reported symptoms exceeded the number of study participants. Other common clinical features included scalp lacerations (44.3%), vomiting (42.9%),

seizures (37.1%), and cerebrospinal fluid leakage (30.0%) as shown in Table 2.

More than half of the patients (55.7%) had associated extra-cranial injuries, reflecting a substantial burden of polytrauma. The majority of patients were managed conservatively (92.9%), with only 7.1% requiring surgical intervention (Table 2).

Table 2: Clinical Presentation, Associated Injuries and Treatment Characteristics (n = 70)

Variable	Category	Frequency n (%)
Clinical features*	Loss of consciousness	70 (100.0)
	Scalp laceration	31 (44.3)
	Vomiting	30 (42.9)
	Seizures	26 (37.1)
	CSF leak	21 (30.0)
Associated Injuries	Present	39 (55.7)
	Absent	31 (44.3)
Treatment modality	Conservative	65 (92.9)
	Surgical intervention	5 (7.1)

*Multiple responses allowed. Patients presented with more than one clinical feature; therefore, the total frequency exceeds the number of study participants.

3.3. Clinical Outcomes

At 12 weeks following injury, outcomes assessed using the Glasgow Outcome Scale revealed that 28.6% of patients achieved good recovery, while 18.5% had moderate disability (Table 3). Severe disability and vegetative state were observed in 12.9% and 14.3% of patients, respectively.

Overall mortality was 25.7% (18 patients). When categorized, 47.1% of patients had favourable outcomes (GOS 4-5), whereas 52.9% experienced unfavourable outcomes (GOS 1-3) (Table 3).

Table 3: Clinical Outcomes at 12 weeks (Glasgow Outcome Score)

Outcome category	Frequency n (%)
Good recovery (GOS 5)	20 (28.6)
Moderate disability (GOS 4)	13 (18.5)
Severe disability (GOS 3)	9 (12.9)
Vegetative state (GOS 2)	10 (14.3)
Death (GOS 1)	18 (25.7)
Outcome grouping	
Outcome group	Frequency n (%)
Favourable (GOS 4-5)	33 (47.1)
Unfavourable (GOS 1-3)	37 (52.9)

3.4. Occurrence and Pattern of Electrolyte Disturbances

Electrolyte disturbances were observed in 34 patients (48.6%), while 36 patients (51.4%) maintained normal electrolyte levels throughout hospitalization (Table 4).

Among those with electrolyte abnormalities, hyponatremia was the most common disturbance (44.1%), followed by hypernatremia (26.5%), hyperkalemia (23.5%), and hypokalemia (5.9%) (Table 4). These findings highlight the predominance of sodium imbalance in the acute phase of traumatic brain injury.

Table 4: Occurrence and types of Electrolyte Disturbance

A. Overall Occurrence (n = 70)	
Electrolyte status	Frequency n (%)
No derangement	36 (51.4)
Any derangement	34 (48.6)
B. Type of Disturbance	
Type	Frequency n (%)
Hyponatremia	15 (44.1)
Hypernatremia	9 (26.5)
Hyperkalemia	8 (23.5)
Hypokalemia	2 (5.9)

Footnotes: Percentages in section A were calculated based on the total study population (n=70). Percentages in section B were calculated among patients who developed electrolyte disturbance (n=34). Each patient was classified into a single category of electrolyte disturbance; therefore, percentages in section B summed up to 100%.

3.5. Relationship between Injury Severity and Electrolyte Disturbances

A significantly higher proportion of patients with severe traumatic brain injury developed electrolyte disturbances compared with those with moderate injury (81.8% vs 42.4%) as seen in Table 5. Conversely, normal electrolyte profiles were more frequently observed among patients with moderate injury (57.6%) than in those with severe injury (18.2%).

This association was statistically significant ($\chi^2 = 5.62$, $p = 0.018$), indicating that increasing injury severity is strongly associated with the development of electrolyte derangements (Table 5).

3.6. Temporal Evolution of Electrolyte Disturbances

The timing of electrolyte abnormalities demonstrated a clear early clustering pattern. Among patients with electrolyte disturbances (n = 34), 26.5% developed abnormalities within the first 3 days, while 47.0% occurred between days 4 and 5 (Table 5). The remaining 26.5% occurred between days 6 and 10.

Overall, 73.5% of electrolyte disturbances occurred within the first 5 days following injury. This temporal distribution was statistically significant ($\chi^2 = 11.182$, $p = 0.042$), underscoring the early post-injury period as a critical phase for electrolyte instability (Table 5).

Table 5: Severity-stratified Electrolyte Disturbances and Pattern

A. Electrolyte Disturbance by Injury Severity				
Severity	Derangement present n (%)	Derangement absent n (%)	Total	p-value
Moderate TBI	25 (42.4)	34 (57.6)	59	0.018
Severe TBI	9 (81.8)	2 (18.2)	11	
Total	34	36	70	
B. Temporal Evolution of Electrolyte Disturbance (n = 34)				
Time interval	Frequency n (%)			
≤ 3 days	9 (26.5)			
4-5 day	16 (47.0)			
6-10 days	9 (26.5)			
C. Early vs Late Occurrence				
Category	Frequency n (%)			
Early (≤ 5 days)	25 (73.5)			
Late (> 5 days)	9 (26.5)			
Statistical test	Value			
Chi-square ()	11.182			
p-value	0.042			

3.7. Electrolyte Disturbances and Mortality

A strong association was observed between electrolyte disturbances and mortality. Among patients with electrolyte derangements, 52.9% died, compared with only 5.6% mortality among those without electrolyte abnormalities (Table 6).

This difference was highly statistically significant ($\chi^2 = 13.652$, $p < 0.001$), demonstrating a robust relationship between electrolyte imbalance and mortality (Table 6).

3.8. Predictors of Mortality

Multivariate logistic regression analysis was performed to identify independent predictors of mortality. After adjusting for injury

severity and age, electrolyte derangement remained a significant independent predictor of death (adjusted OR = 6.214; 95% CI: 1.423–27.135; $p = 0.015$) (Table 6).

Severe traumatic brain injury was also independently associated with mortality (adjusted OR = 4.102; 95% CI: 1.038–16.209; $p = 0.044$).

Age did not show a statistically significant association with mortality (adjusted OR = 1.029; 95% CI: 0.979–1.082; $p = 0.258$) (Table 6).

Table 6: Electrolyte disturbance and Mortality: Association and adjusted Logistic Regression

A. Association with mortality (n = 70)			
Electrolyte status	Alive n (%)	Dead n (%)	Total
Derangement present	16 (47.1)	18 (52.9)	34
Derangement absent	34 (94.4)	2 (5.6)	36
Statistical test	Value		
Chi-square	13.652		
p-value	<0.001		
B. Multivariate Logistic Regression (Adjusted)			
Variable	Adjusted OR	95% CI	p-value
Electrolyte derangement	6.214	1.423-27.135	0.015
Severe TBI (vs moderate)	4.102	1.038-16.209	0.044
Age (per year increase)	1.029	0.979-1.082	0.258

4. Discussion

This study provides a comprehensive evaluation of the pattern, timing, and clinical implications of sodium and potassium disturbances in adult patients with traumatic brain injury. The findings demonstrate that electrolyte abnormalities are common, occur predominantly early after injury, and are independently associated with mortality [5, 8].

Nearly half of the patients in this cohort developed electrolyte disturbances, as shown in Table 4. This finding is consistent with previous studies that have reported a high incidence of electrolyte imbalance in traumatic brain injury. Usha et al. documented electrolyte abnormalities in approximately 75% of patients [4], while Lohani et al. reported sodium disturbances in 27.2% of 33 patients [5]. The incidence observed in the present study, although higher than the findings of Lohani et al, remains clinically significant and may reflect differences in monitoring intensity or case mix.

Hyponatremia was the most frequently observed electrolyte abnormality, accounting for the largest proportion of disturbances (Table 4). This aligns with prior reports indicating that hyponatremia is a common complication in neurocritical care, often resulting from diabetes insipidus, osmotic diuresis, or inadequate fluid replacement [2,9]. The predominance of hyponatremia underscores its importance as both a marker of neuroendocrine dysfunction and a potential contributor to secondary brain injury.

Hyponatremia was also commonly observed and remains a well-recognized complication of traumatic brain injury, frequently associated with SIADH or cerebral salt-wasting syndrome [1]. Previous studies, including that of Al Yaqoubi et al. have demonstrated that hyponatremia is associated with increased mortality in hospitalized patients [10]. The coexistence of both hypo- and hypernatremia in this study highlights the complex and dynamic regulation of sodium balance following brain injury.

One of the most important findings of this study is the clear temporal pattern of electrolyte disturbances. As demonstrated in Table 5, nearly three-quarters of abnormalities occurred within the first five days following injury. This early clustering is consistent with previous reports by Yano et al., who observed that electrolyte imbalances tend to develop in the early post-traumatic period [11]. Similarly, Tran et al. emphasized the importance of close biochemical monitoring during this critical phase [12]. These findings reinforce the need for intensive electrolyte surveillance during the first week of hospitalization.

The present study also demonstrates a significant association between injury severity and electrolyte disturbance (Table 5). Patients with severe traumatic brain injury were substantially more likely to develop electrolyte abnormalities than those with moderate injury. This observation is biologically plausible, as severe brain injury is more likely to disrupt hypothalamic-pituitary function and trigger systemic physiological derangements.

A key strength of this study lies in the demonstration of a strong association between electrolyte disturbances and mortality. As shown in Table 6, mortality was markedly higher among patients with electrolyte derangements compared with those without. Importantly, this association remained significant after adjustment for injury severity and age, with electrolyte disturbance conferring more than a six-fold increase in the odds of death.

From a statistical perspective, the use of multivariate logistic regression enhances the robustness of these findings by accounting for potential confounding variables. The inclusion of injury severity in the model is particularly important, given its well-established relationship with mortality in traumatic brain injury. The persistence of electrolyte disturbance as an independent predictor suggests that it is not merely a surrogate for injury severity but may contribute directly to adverse outcomes.

Although the confidence intervals are relatively wide, this is expected given the modest sample size and number of outcome events. Nevertheless, the effect size is large, the direction of association is consistent, and the statistical significance is maintained, all of which support the validity of the findings.

These findings are consistent with the findings of previous studies, including that of Maggiore et al. which demonstrated that electrolyte imbalance is associated with worse outcomes in critically ill patients [13], while other investigators have highlighted electrolyte disturbances as important prognostic markers [14].

The study has several strengths, including its prospective design, systematic electrolyte monitoring, and incorporation of temporal analysis. However, certain limitations should be acknowledged. The relatively small sample size may limit the precision of effect estimates and the generalizability of findings. In addition, the study was conducted in a single center, which may reflect local practice patterns.

Despite these limitations, the findings provide clinically meaningful insights. The early occurrence of electrolyte disturbances, their strong association with injury severity, and their independent relationship with mortality collectively emphasize the importance of routine and timely electrolyte monitoring in patients with traumatic brain injury.

5. Conclusion

Electrolyte disturbances are common during the acute management of traumatic brain injury and predominantly occur within the early post-injury period. This study demonstrates that abnormalities in plasma sodium and potassium levels are not only frequent but are also significantly associated with injury severity and mortality.

The finding that most disturbances develop within the first five days following injury highlights a critical window during which patients are particularly vulnerable to secondary physiological insults. The strong and independent association between electrolyte derangement and mortality further underscores their clinical relevance as both markers of disease severity and potential contributors to adverse outcomes.

These observations emphasize the need for routine and vigilant electrolyte monitoring in patients with traumatic brain injury, particularly during the early phase of hospitalization. Prompt recognition and appropriate correction of electrolyte abnormalities may represent a simple yet important strategy for mitigating secondary brain injury and improving overall patient outcomes.

Declarations

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.

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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.

Conflict of Interest Statement

The author declares that there are no conflicts of interest regarding the publication of this article.

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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.

6. References

- [1] Sherlock M, O'Sullivan E, Agha A, Behan LA, Rawluk D, Brennan P, et al. The incidence and pathophysiology of hyponatraemia after subarachnoid haemorrhage. *Clin Endocrinol (Oxf)*. 2006; 64(3): 250-4. doi: 10.1111/j.1365-2265.2006.02432.x
- [2] Capatina C, Paluzzi A, Mitchell R, Karavitaki N. Diabetes Insipidus after Traumatic Brain Injury. *J Clin Med*. 2015 Jul 13;4(7):1448-62. doi: 10.3390/jcm4071448. PMID: 26239685; PMCID: PMC4519799.
- [3] Pomeranz S, Constantini S, Rappaport ZH. Hypokalaemia in severe head trauma. *Acta Neurochir (Wien)*. 1989; 97(1-2): 62-6. doi: 10.1007/BF01577741. PMID: 2718795.
- [4] Usha S Adiga, Vickneshwaran V, Sanat Kumar Sen. Electrolyte derangements in traumatic brain injury. *Journal of Medicine and Clinical Sciences*. 2012; 1(2): 15-18

- [5] Lohani S, Devkota UP. Hyponatremia in patients with traumatic brain injury: etiology, incidence, and severity correlation. *World Neurosurg*. 2011 Sep-Oct;76(3-4):355-60. doi: 10.1016/j.wneu.2011.03.042. PMID: 21986437.
- [6] Dey S, Kumar R, Tarat A. Evaluation of Electrolyte Imbalance in Patients with Traumatic Brain Injury Admitted in the Central ICU of a Tertiary Care Centre: A Prospective Observational Study. *Cureus*. 2021; 13(8): e17517. DOI 10.7759/cureus.17517
- [7] Jennett B, Bond M. Assessment of outcome after severe brain damage. *Lancet*. 1975; 1: 480-4. doi: 10.1016/s0140-6736(75)92830-5.
- [8] Săcărescu A, Turluc MD. Electrolyte Imbalance in Acute Traumatic Brain Injury: Insights from the First 24 h. *Clin Pract*. 2024 Aug 30;14(5):1767-1778. doi: 10.3390/clinpract14050141. PMID: 39311291; PMCID: PMC11417862.
- [10] Mirza Faisal Ahmed Rafiq, Noor Ahmed, Adil Aziz Khan. Serum electrolyte derangements in patients with traumatic brain injury. *J Ayub Med Coll Abbottabad* 2013;25(1-2): 162-164
- [11] Al Yaqoubi IH, Al-Maqbali JS, Al Farsi AA, Al Jabri RK, Khan SA, Al Alawi AM. Prevalence of hyponatremia among medically hospitalized patients and associated outcomes: a retrospective cohort study. *Ann Saudi Med*. 2024 Sep-Oct;44(5):339-348. doi: 10.5144/0256-4947.2024.339. Epub 2024 Oct 3. PMID: 39368118; PMCID: PMC11454974.
- [12] Tran V, Flores J, Sheldon M, Pena C, Nugent K. Fluid and Electrolyte Disorders in Traumatic Brain Injury: Clinical Implications and Management Strategies. *Journal of Clinical Medicine*. 2025; 14(3):756. <https://doi.org/10.3390/jcm14030756>
- [13] Maggiore U, Picetti E, Antonucci E, Parenti E, Regolisti G, Mergoni M, Vezzani A, Cabassi A, Fiaccadori E. The relation between the incidence of hypernatremia and mortality in patients with severe traumatic brain injury. *Crit Care*. 2009;13(4):R110. doi: 10.1186/cc7953. Epub 2009 Jul 7. PMID: 19583864; PMCID: PMC2750153.
- [14] Bikesh Khambu, Rajiv Jha, Prakash Bista. Sodium Imbalance as a Marker of Prognosis of Outcome in Patients with Traumatic Brain Injury. *Nepal Journal of Neuroscience* 2020; 17(3): 25-30.



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