

Sodium Imbalance as a Marker of Prognosis of Outcome in Patients with Traumatic Brain Injury



Bikesh Khambu *MCh*¹, Rajiv Jha *MCh*², Prakash Bista *MCh*³

^{1,2,3} Department of Neurosurgery, National Academy of Medical Sciences, Kathmandu, Nepal

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Abstract

Introduction: Sodium imbalance in post-traumatic brain injury is not uncommon. It's early detection and treatment is vital as it increases morbidity and mortality if not treated on time. We study the effect of Sodium imbalance as a predictor of outcome in these patients.

Methods and Materials: Patients diagnosed with traumatic brain injury (TBI) admitted to Neurosurgery department were included in this study. Traumatic brain injury cases managed between 1st Baisakh 2074 B.S. and 29th Chaitra 2074 B.S. were analyzed prospectively. Glasgow Coma Scale, Computed Tomography scan of head, duration of hospital and Intensive Care Unit (ICU) stay, and Glasgow Outcome Scale in relation to sodium imbalance measured within 24 hours of injury were assessed. Mortality and morbidity were measured using Glasgow Outcome Scale (GOS).

Results: A total of 100 patients with traumatic brain injury admitted in the Neurosurgery department of Bir Hospital were included in this study. Sixty seven cases were categorized as mild, 18 as moderate and 15 as severe according to the severity of TBI. The maximum serum sodium level observed was 168 and lowest was 110 mmol/l. The association of sodium imbalance with severity of head injury was found to be statistically significant. The sensitivity of sodium imbalance measured within 24 hours of injury in prognosticating outcome of patient till death or before discharge from hospital on the basis of GOS and Neuro ICU stay day is significant with p value <0.05.

Conclusion: Sodium imbalance is an independent prognostic factor outcome in TBI. Continuous monitoring of sodium level in the neurosurgical ICU setting is mandatory to manage patients with head injury with or without undergoing neurosurgical intervention as it has prognostic value with outcome measures like GOS and ICU stay days.

Key words: Glasgow coma scale, Glasgow outcome score, Hospital stay, Hyponatremia, Hypernatremia, Neurosurgery, Sodium imbalance, Traumatic brain injury

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Introduction

Traumatic Brain Injury (TBI) is the spectrum of brain insult as a consequence of trauma to the brain. It can be primary or secondary. Primary injury occurs during the initial insult, and results from displacement of the physical structures of the brain.¹ Secondary injury occurs gradually and may involve an array of cellular processes can result from the primary injury or be independent of it.²

The blood brain barrier and meninges may be damaged in the primary injury giving rise to severe disability due to neuronal destruction, a large number will subsequently deteriorate owing to cerebral ischemia from brain swelling, hematoma formation, hypoxia and hypotension.¹⁻⁴ Secondary brain injury is divided into intra and extra-cranial causes. Intracranial Injury includes neurotoxic cascades; calcium channel disturbance, oxygen free radical production and hematoma formation.⁵⁻¹¹ Extracranial secondary injury include respiratory failure and blood loss. Other causes of secondary injury infection

¹ORCID id: 0000-0002-7369-589X

²ORCID id: 0000-0002-2830-7835

³ORCID id: 0000-0002-4811-6964

Address for correspondence:

Dr. Bikesh Khambu
Madhyapur Thimi-8,
Bode, Bhaktapur, Nepal
E-mail: bikeshrestha2001@gmail.com
Phone: +977 9841376383

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and seizure, epileptic seizures and sodium imbalance.¹²

Among electrolyte imbalance, sodium imbalance solely is the most important factor that indicates severity of secondary brain injury due to disturbances in hypothalamo pituitary axis, hypothalamo renal axis and severe brain edema that causes increased morbidity and mortality in TBI.¹³

The main objective of this study is to determine the effect of sodium imbalance as a predictor of outcome in the patients of TBI.

Methods and Materials

It is a non-randomized prospective, observational hospital-based study done at Bir Hospital (BH) & National Trauma center (NTC) from 1st Baisakh 2074 B.S. to 29th Chaitra 2074 B.S. All the patients diagnosed with TBI admitted via emergency in Bir Hospital with or without neurosurgical intervention were included. Exclusion criteria included patients who were discharged from the emergency department, patients with previous electrolyte imbalance and associated other chest/abdomen trauma, patients on diuretics, other potential causes of sodium disorders and systemic trauma and patients who could not be followed.

The sample size was 100 consecutive patients taken by convenient sampling method. All patients underwent neurosurgical examination, cervical spine and plain chest radiography, and cerebral CT. Patients underwent CT scans on at least two occasions: on hospital admission after resuscitation and within 12 hours to 3 days after the first tomography. Hypertonic saline solution was not used in our cases, and no protocols with hypertonic saline solution for care in a rescue unit were adopted in BH. After hospital admission with resuscitation by advanced trauma life support (ATLS) protocol, all patients with TBI were admitted to Neurosurgery ward, Neurosurgery high care unit and ICU and were included in this study. They did not receive diuretics. In all patients, a standard blood sample was taken (collected directly from a peripheral vein in the arm or leg). All baseline investigations needed for the standard management were done. Baseline sodium value was done, and was repeated everyday till the patient was discharged from the hospital.

Sodium level of more than 145 mEq/L or less than 135mEq/L on two or more occasions was considered sodium imbalance. In patients with abnormal sodium, a blood sample was collected every day until correction of sodium and after that at least one blood sample per week during the first month after trauma. Decisions regarding the management of sodium disorders were left to the physicians responsible and were not influenced by the

study. For all measurements, an AVL 988-4 Electrolyte Analyzer (AVL Scientific Co, Roswell, GA) was used.

The characteristics of the patients analyzed were: serum sodium level, TBI classification (as mild, moderate and severe according to the Glasgow coma scale (GCS)),⁷ pupillary reaction to light, vital signs namely blood pressure, pulse rate, respiratory rate, temperature, oxygen saturation (SpO₂), urine output, presence of other co-morbidity, CT scan of head, hemogram, renal function test, Na/K(Serum Sodium/Serum Potassium), Prothrombin Time(PT), International Normalized Ration(INR), Random blood sugar (RBS) and Arterial blood gas (as required). Diffuse injury was diagnosed as persistent comatose patients (more than 6 hours) with no focal lesions greater than 25 cm³ as described by Marshall et al.⁸

Data was obtained from history and physical examination as well as the completed proforma mainly by the principal investigator after meeting all the criteria mentioned. Data on clinical status of the study participants was collected through the emergency and admission chart. A detailed orientation of the study and enrollment system was given to medical doctors, MCh residents and ward in-charge for admitted patients. Upon receiving a case fulfilling the inclusion criteria, the participants were explained about the study in detail. He or she was assured of full confidentiality and a written informed consent taken subsequently. The specific tests mentioned were carried out using standard laboratory protocol. Final data analysis was done upon completion of the study.

The statistical analyses were performed using *R* statistical software, applied within each subgroup analysis using chi square or fisher exact test to compare proportions. A 95% confidence interval was taken, and p-value less than 0.05 were termed as statistically significant.

Ethical committee of Bir Hospital, IRB regarding this research was received and study was done as per their guidelines.

Results

100 patients with TBI with injuries as depicted in Figure 1 (from left to right, clockwise manner) were enrolled in the study.

There was male dominance in the study with male: female ratio of 4.8:1. Mean age of the study population was 37 years (range 1 to 95). Mean duration of hospital stay was 11 days (range 1-113). Twenty-nine patients stayed at ICU. The mean duration of ICU stay was 5 days (range 1-57).

The most common mode of TBI was fall injury (62%)

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followed by RTA (31%), physical assault (5%) and struck (2%) injury (Figure 2).

Based on GCS, among the TBI cases almost two third of the cases (67%) were classified as mild, 18% as moderate and 15% as severe TBI.

The prevalence of sodium imbalance among TBI cases was 25%.

Seven patients (7%) presented with hypernatremia with serum sodium level of more than 145 mEq/L and 18 (18%) with hyponatremia with sodium level less than 136mEq/L. The present study demonstrated sodium imbalance to be significantly associated with increase in severity of TBI (Table 1).

The Glasgow outcome scale (GOS) being one of the most commonly used neurosurgical overall outcome, significant association was also observed between GOS and sodium imbalance with p value<0.0001 (Table 2).

Of the total cases, 25 patients received surgical treatment. The surgical treatments were intracranial pressure monitoring with EVD for posterior fossa bleed with fourth ventricle involvement for one patient and 24 patients undergoing hematoma removal and/or evacuation of the acute extradural, subdural, and intraparenchymal bleed with or without extension to ventricle.

Though serum sodium imbalance did not increase hospital stay, it did prolong ICU stay (p=0.007) (Table 3).

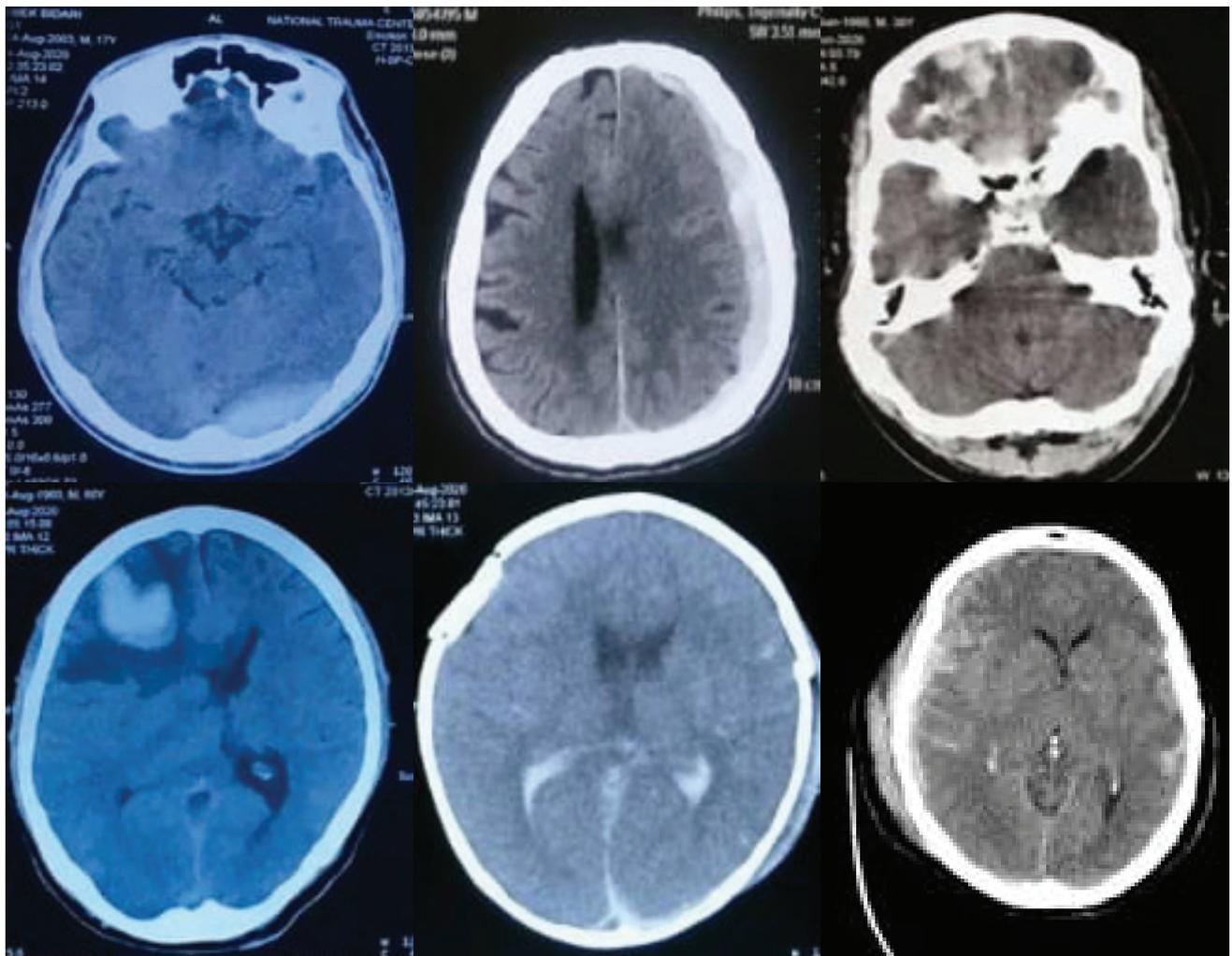


Figure 1: Axial CT scans of TBI patients (Source: Department of Neurosurgery, NTC and Bir Hospital)

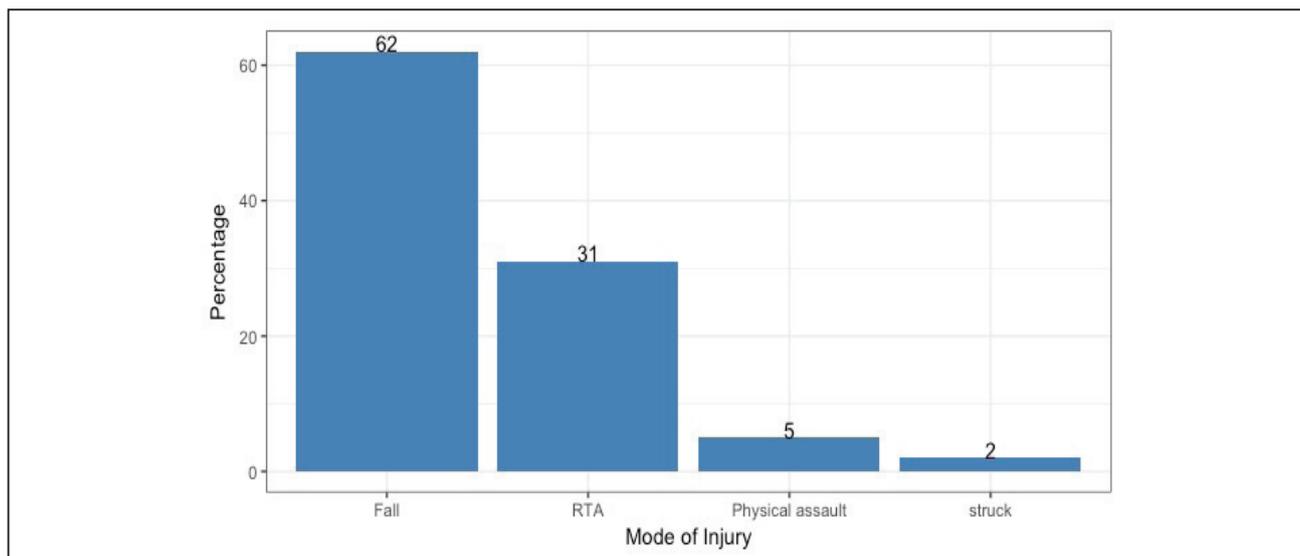


Figure 2: Modes of injury of the studied TBI patients (n=100)

Sodium level	Severity of Traumatic Brain Injury			P-value
	Mild	Moderate	Severe	
Normal Sodium level	56	14	5	0.00024
Sodium imbalance	11	4	10	

Table 1: Relationship of Sodium imbalance TBI with its severity

Sodium level	Glasgow outcome scale					P-value
	1	2	3	4	5	
Normal level of Sodium	5	1	8	9	52	<0.0001
Sodium imbalance	12	1	5	2	5	

Table 2: Relationship of Sodium imbalance in TBI with GOS

Days	Total patients(n=100)		P-value
	Normal level of sodium (n=75)	Sodium Imbalance (n=25)	
Hospital Stay			0.46
<8	49	15	
8-14	15	4	
>14	11	6	
ICU stay	n=75	n=25	0.007
0	53	8	
1-4	10	7	
5-9	6	6	
>9	6	4	

Table 3: Relationship of sodium imbalance in TBI patients with Hospital and ICU stay days

Discussion

Our study showed 25% patients of TBI had sodium imbalances (Hypo/Hyponatremia). The incidence of sodium disorders was similar as compared with other studies, and the results were similar to those of other series.¹⁴⁻¹⁵

The substantial rate of detection of disorders within 24 hours of injury indicates severe brain lesions. The mortality rates in TBI patients with sodium imbalance are higher than patients with normal sodium concentration. As the serum sodium concentration is altered, it can result in remarkable brain swelling.¹⁶

Arieff et al found that the acute onset of severe hyponatremia following TBI was associated with a poor neurological outcome or death after sudden onset of seizures, followed by coma, apnea, and brainstem compression.¹⁷ Our study also had worst outcome with hyponatremia. Among 7 patients 5 patients died and 1 had GOS 4 and other had GOS 3 on discharge.

In a retrospective cohort study, Maggiore et al verified that hyponatremia was common; occurring in 51.5% of patients for 31% of the duration of their ICU stay.¹⁸ Hyponatremia was associated with a threefold increase in hazard of ICU death, even after adjustment for baseline risk. In the present study we have similar results. 7 patients had hyponatremia and all stayed in ICU.

Horn and Glenn screened patients with severe head injury and found that 20% suffered from one or more hormone disturbances.¹⁹ The cause of sodium disorders is diverse and the associated risk of morbidity varies widely. Therefore, early diagnosis and effective treatment of hyponatremia is critical for hyponatremic patients with intracranial disease. Neurological dysfunction is the principal manifestation of hyponatremia, which may be exacerbated by other disease processes or underlying conditions, especially in those patients in whom a pathological condition is located intracranially.¹⁵

The high rate of serious alterations during the evolution of traumatic brain injury suggests the possible occurrence of associated neuroendocrinological conditions such as Diabetes Insipidus (DI), Syndrome of Inappropriate ADH secretion (SIADH), and cerebral salt wasting. The mechanism by which an intracranial disease leads to the occurrence of the syndrome has yet to be fully elucidated. There were fewer patients with DI in the present study and it is probably due to less number of severe TBI patients. As there was insufficient data on pituitary function and sodium excretion in patients, although syndrome of inappropriate antidiuretic hormone hypersecretion is the most common cause of hyponatremia, the cause of their hyponatremia remains unclear.²⁰

Hyponatremia may also be caused by the activity of the brain natriuretic factor (BNF), probably secreted by the thalamus, exerting an action identical to that of the hormone secreted by the atrial cardiocytes.²¹⁻²² BNF is a potent diuretic, natriuretic, vasodilation agent, and an inhibitor of the secretion of aldosterone, renin, and vasopressin.²³⁻²⁵ It also decreases plasma volume. Increased BNF is most commonly found in patients with subarachnoid hemorrhage or hemorrhage at the base of the brain or in the third ventricle.²⁶⁻²⁷ The incidence of sodium disorders was high in study patients with moderate to severe head injury patients. No association was found between specific types of TBI and the proportion of

sodium disorders, but the incidence of sodium disorders was greater in moderate and severe TBI than in mild TBI.

Limitations of the study

We believe that the selection criteria allowed the exclusion of confounding variables, however the clinical reality is different. Besides the limited number of cases in this study along with non-consideration of other factors like radiological findings and patient factors in the analysis are limitations of the study.

We believe that further studies with larger numbers of patients and blind comparison can respond to questions not answered in this study.

Conclusion

Sodium imbalance in TBI patients is more prone to have more morbidity and mortality during hospital admission. Sodium imbalance prolongs the ICU stay day in moderate and severe head injury.

So, early sodium correction in TBI patients managed with or without surgery is one of the most important independent prognostic factors especially who were treated at hospital in ICU setting in order to minimize morbidity and mortality.

Conflict of Interest: None

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