Original Article

Relationship of Hypernatremia and Cortisol Levels with Severity and Survival in Pediatric Traumatic Brain Injury: A Prospective Pilot Study

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Abstract

Background: Hypernatremia and cortisol levels are associated with traumatic brain injury (TBI). The present study aimed to determine whether cortisol and sodium levels are altered in association with TBI severity and patient survival. **Material and Methods:** In the prospective pilot case-cohort study, 32 preadolescent TBI patients were enrolled. Medical history, CT, and MRI were recorded. Serum cortisol and sodium levels were measured by chemiluminescent immunoassay (Immunoassay i1000) in the follow-up sample. The association between cortisol levels and Glasgow Outcome Scale score was evaluated. Pearson's correlation coefficient was used to analyze the relationship between sodium and cortisol levels. The Kaplan–Meier test was used for survival analysis. **Results:** The mean age was 5.94±3.11 years. Nineteen patients had severe injuries (GCS 3-8), and 13(41%) had moderate injuries (GCS 9-12). The cortisol levels on day 1 were significantly higher (25.56±11.66 µg/dl) as compared to day 7 (21.43±8.88 µg/dl; p<0.0001). However, at 6 months, it was significantly lower (8.64±3.46 µg/dl) than on day 1 (p<0.0001). Severe TBI patients showed substantially higher sodium levels compared to moderate TBI patients (139.7±3.01 vs. 137.2±2.82 mmol/L, p=0.023). Cortisol and sodium levels were positively correlated (r=0.45, p=0.024). Hypernatremia was significantly associated with survival in TBI patients. Compared with normal cortisol levels (3-19.4 µg/dl), high cortisol levels (>25.0 µg/dl) were significantly associated with poor patient survival at 6 months of follow-up (p<0.0001). **Conclusion:** Severe TBI was associated with hypernatremia. High cortisol and sodium levels were associated with poor survival. A significant relationship between sodium and cortisol levels was found.

Keywords: Glasgow Coma Scale, Cortisol, Outcome, Survival, Traumatic brain injury, Hypernatremia.

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Introduction

The primary cause of death and disability among adults and preadolescents is traumatic brain injury (TBI). In India, children <15 comprise 35% of the total population and cause 20–30% of all head injuries.[1-3] These disparities are linked to age-related structural changes, the mechanism of injuries depending on the teenager's physical capacity, and difficulties in evaluating the neurological status of the pediatric population. Improvements in diagnostic imaging have enhanced the quality of care by aiding the examination and diagnosis of TBI in children. The diagnosis and prognosis of traumatic brain injury (TBI) in acute settings rely on neurological assessments and neuroimaging technologies like magnetic resonance imaging (MRI) and computed tomography (CT) scanning. Nevertheless, CT scanning exposes patients to radiation and is intolerant to diffuse brain injuries.[4-6] Although MRI may reveal the extent of diffuse injuries, its widespread usage is constrained by cost, its limited availability in many locations, and the challenge of doing MRI on patients who are physiologically unstable.[6]

Blood biomarkers may aid in predicting adverse outcomes across the spectrum of TBI severity. The pathophysiological mechanisms underlying apparent neurological deficits can be objectively and quantitatively assessed by tracking TBI biomarkers. Such information may be more useful for directing treatment than the first severity rating alone.[7]

Water-electrolyte imbalance (hypernatremia) significantly influences the TBI severity, making it more challenging to sustain individuals with TBI. Sodium concentration below 160 mmol/L is associated with 75% mortality.[8] Mild hypernatremia is common in patients with severe TBI while they are in the critical care unit. The onset of central diabetes insipidus (CDI), a credible indicator of the extent of brain injury and a distinct predictor of intensive care unit (ICU) death, is most likely to be the cause of the percentage of hypernatremia patients in this clinical scenario. Hypernatremia raises the chance of mortality on its own, regardless of the other processes involved. The most

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common electrolyte imbalance in TBI and a predictor of poor neurological outcomes is hyponatremia, which is defined as a blood sodium level of 135 mEq/L10. The two most frequent causes of hyponatremia with natriuresis—syndrome of inappropriate antidiuretic hormone secretion (SIADH) and cerebral salt wasting (CSW)—are difficult to differentiate from one another, and the prevalence of hyponatremia reported in medical literature varies widely.[9-12]

TBI patients' prognoses are affected by adrenal insufficiency. Cortisol levels rise in the morning and drop during the day, and rise again at night in healthy people. Head injuries increased mean cortisol levels, and moderate and severe head injuries increased them more; thus, serum cortisol correlated positively with GCS. Further serum cortisol levels increased immediately after trauma. Head injury severity linearly increases.[13]

This study aimed to examine the dynamic variations in cortisol levels and their association with survival and hyponatremia in pediatric patients with TBI.

Materials and Methods

Selection of Subjects: This pilot observational study was conducted on 32 pediatric patients with moderate-to-severe TBI hospitalized in the Department of Neurosurgery, King George's Medical University, Lucknow. Patients with a previous corticosteroid medication, etomidate prescription, or imaging-confirmed pituitary and adrenal gland abnormalities were excluded from this study.

Clinical Assessment and Ethics: Medical history and clinical findings were recorded, including the mechanism of injury, pupillary responses, and computed tomography of the head. Serum sodium levels were measured at admission.

The experimental protocol (code no. V-PGTSC-IIA/P6) was approved by the Ethics Committee of King George's Medical University (KGMU).

Measurement of serum cortisol: For the cortisol assay, blood samples from each participant were collected within 2 hours, centrifuged, placed into polypropylene cryovials, and stored at -20°C. A chemiluminescence microparticle assay (Immunoassay I1000) was used to measure cortisol levels at admission, week (discharge), and 6 months after follow-up.

Outcomes Measure

Scoring for Glasgow Coma Scale and Glasgow Outcome Scale (E)

Radiological and clinical information on the patient was gathered. At six months, the results of the Glasgow Coma Scale (GCS) and Glasgow Outcome Scale (E) (GOS-E) were assessed. Using the patient's GCS at admission, the degree of TBI was assessed: 13–15 indicated mild TBI, 9–12 indicated moderate TBI, and 8 indicated severe TBI. The GOS-E scale, on the other hand, has the following values: death = 1, persistent vegetative state = 2, upper good recovery = 8, lower good recovery = 7, upper moderate disability = 6, lower moderate disability = 5, upper severe disability = 4, and lower severe disability = 3. GOS (E) was classified as either good (grades 6, 7, and 8) or unfavorable (grades 1–5). Furthermore, the teenagers were divided into two groups: non-survivors (grades 9–12)14 and survivors (grades 2–8). Telephone interviews were used to ascertain survivor status during a six-month follow-up.

Data analysis: The chi-squared test was used to compare categorical variables, which were represented as frequencies and percentages. All variables are expressed as the mean ± SD. Pearson's correlation coefficient was used to analyze.

The correlations: The significance level was set at $p<0.05$. All statistical analyses were performed using SPSS Statistics for Windows, version 21.0 (IBM, USA).

Results

Demographical and clinical characteristics of the pediatric TBI patients

The data are presented in [Table 1]. In the study population, there were 21 males and 11 females . Mean age was 5.94±3.11 years. The leading 22 (68.75%) causes of brain injury were motor vehicle accidents. The most common symptom presentation was loss of consciousness in 28 (86%) children, followed by vomiting in 9(28%, ear bleeding in 2(6%), and nasal bleeding in 2(6%). Categorical variables, which were represented as frequencies and percentages, were evaluated using the chi-squared test. On examination, 14 (44%) pupils responded slowly. CT scans revealed contusions in 12 (38%) patients, followed by acute subdural hematomas in 3 (10%) patients. Fractures and extradural hematomas were observed in only three (10%). There were 19 (59%) children with severe brain injuries (GCS 3-8) and 13 (41%) children with intermediate head injuries (GCS 9-12).

Dynamic changes in cortisol levels at admission, week, and 6 months

Temporal cortisol samples were collected at admission, week, and after 6 months. The cortisol levels of day 1 were significantly higher (25.56±11.66 µg/dl) in comparison to 7 days of cortisol levels (21.43±8.88 µg/dl; $p<0.0001$). However, at 6 months, cortisol levels were significantly decreased (8.64±3.46 µg/dl) compared to day-1 cortisol levels ($p<0.0001$), indicating that cases had reverted (recovered) to a normal cortisol level after 6 months [Figure 1].

Relationship between cortisol levels at admission, at week, and at 6 months, and the severity of TBI. The patients were divided into two groups according to the GCS: Severe (3-8) and Moderate (9-12). [Figure 2] shows that day1 samples cortisol levels were significantly higher in severe (3-8) as compared to moderate (severe 28.8 µg/dl vs. moderate 22.1 µg/dl; $p<0.0001$), whereas cortisol levels were also significantly changed in the severe group (23.5 µg/dl) as compared to the moderate group (18.5 µg/dl; $p<0.0001$). After 6 months, cortisol levels did not differ significantly (severe 9.7 µg/dl vs. moderate 8.5 µg/dl, $p=0.38$).

Proportionate relationship between cortisol level and GOS-E score

The results are compared in [Table 2]. According to the GOSE score, the cases were split into two groups: the unfavorable group (1–5) and the favorable group (6–8). In the unfavorable group (1-5), cortisol levels were significantly higher on admission and in the week sample ($p<0.001$) than in the 6-month sample. In the favorable group (6-8), cortisol levels also increased on admission compared to the week ($p<0.001$). There was no significant difference between the favorable and non-favorable groups in the

6-month sample (p=0.21).

Elevated cortisol levels associated with the outcome of TBI (GOSE) in preadolescents after 6 months of a 6-month follow-up [Figure 3] shows the association of cortisol levels with GOS-E at the 6-month follow-up in patients with TBI. Cortisol levels were not significantly associated with the GOS-E scores (p=0.72).

Relationship between cortisol level and Hypernatremia in TBI patients

Severe TBI patients showed significantly higher sodium levels (hypernatremia) than moderate TBI patients (139.7±3.01 vs. 137.2±2.82 mmol/L, p=0.023). A positive correlation was observed between cortisol and sodium levels. In TBI patients, hypernatremia was also strongly associated with survival. In patients with elevated cortisol, survival was markedly correlated with cortisol levels (> 25.0 µg/dl). Only three patients were alive, and 16 died of high cortisol levels.

Table 1: Baseline and Clinical Characteristics of pediatric traumatic brain injury

Variables	N=32
Age (Years) Mean	5.94 ± 3.11
Range	6 – 10
Gender	
Male	21 (66%)
Female	11 (34%)
Mode of Injury	
Road Accident (two-wheeler, four-wheeler, Bicycle and Pedestrian)	22(68.75%)
Assault	10 (31.25%)
Presenting Symptoms	
Loss of Consciousness	28 (86%)
Vomiting	9 (28%)
Ear bleed	2 (6%)
Nasal bleed	2 (6%)
Associated Symptoms/ sign	
CSF/brain matter Leak from wound site	6 (19%)
CSF Otorrhoea	2 (6%)
Pupillary reaction	
Normal reaction	18 (56%)
Sluggishly reaction	14 (44%)
CT Scan	
EDH	3 (10%)
ASDH	3 (10%)
Contusion	12 (38%)
Fracture	10 (31%)
DAI	8 (25%)
SAH	1 (3%)
IVH	3 (10%)
Glasgow Coma Scale (GCS)	
Severe (3-8)	19 (59%)
Moderate (9 -12)	13 (41%)

Abbreviations: CSF-Cerebrospinal fluid EDH-Epidural hematoma, ASDH-Acute Subdural hematoma, DAI Diffuse Axonal Injury SAH- subarachnoid hemorrhage, IVH-Intraventricular hemorrhage

Table 2: Association of cortisol level at day 1(admission), 7(discharge) & 6 months with Glasgow outcome scale followed by 6 months

Glasgow Outcome Scale (GOS)		Cortisol Day-1	Cortisol Day-7	Cortisol 6 months	P1	P2	P3
Unfavorable Outcome	Expired (1)	32.8±20.5	27.1±14.7	13.4±4.2	0.21	<0.001	<0.001
	Vegetative state (2)	37.1±21.6	32.6±18.9	8.6±1.7	0.43	<0.001	<0.001
	Lower Severe Disability (3)	19.5±9.8	13.8± 4.8	6.7±1.4	0.01	<0.001	<0.001
	Upper Severe Disability (4)	9.0±6.9	14.2±7.6	4.8±1.2	0.01	<0.001	0.04
	Lower Moderate disability (5)	33.1±15.9	24.9±11.3	8.2±1.8	0.04	<0.001	<0.001
Favorable Outcome	Upper Moderate disability (6)	22.2±2.9	16.8±2.4	11.5±4.7	<0.001	<0.001	<0.001
	Lower good recovery (7)	22.5±6.6	20.2±4.3	7.6±3.5	0.15	<0.001	<0.001
	Upper good recovery (8)	18.5±8.8	24.3±9.2	14.2±6.5	0.02	<0.001	0.05

Abbreviations: P1; p-value between day 1 and 7, P2; p-value between day 7 and 6 month and P3; p- value between day 7 and 6 month.

Table 3: Association of sodium level in pediatric traumatic brain injury patients during admission

Variables	Glasgow Outcome Scale (GOS)		p-value
	Severe (3-8)	Moderate (9-12)	
Sodium (mmol/L)	133.7±3.01	139.2±2.82	<0.0001
	Outcome		
	Dead	Live	
Sodium (mmol/L)	137.6±3.06	141.3±2.01	<0.0001

Table 4: Association of cortisol levels with survival of pediatric traumatic brain injury patients after 6 months follow-up

Cortisol Status	Total No.	No. of Death patients	No. of Live patients	Log Rank (Mantel-Cox) p-value
High (>25 µg/dl)	19	16	3	(15.62) <0.0001
Normal (3-19.4 µg/dl)	13	2	11	

Discussion

The present study sought to determine sequential changes in blood cortisol levels in preadolescent patients with moderate-to-severe traumatic brain injury. The present study is the first to examine changes in serum cortisol dynamics at 1-day intervals (7 days and 6 months) and the relationship between hyponatremia and cortisol levels, severity, and survival in pediatric TBI patients.

In the present study, patients were aged between 6 and 10 years. Male sex was predominant (66%). In 68.74% of the patients, a traffic collision was the most common cause of brain damage. In 59 percent of patients, the GCS score ranged from 3 to 8. CT findings revealed contusions in 62.6% of patients, followed by acute subdural hematoma (39.5%), skull vault fracture, and extradural hematoma (15.1%). Muthusami et al,[15] Adatia et al,[16] and Dennis et al,[17] also reported contusions as the most common finding in TBI. However, some brain contusions "blossom" require surgical intervention with craniotomy and clot evacuation.[18] According to Sahin et al. (2005), early surgical intervention for extradural hematomas (EDH), subdural hematomas (SDH), and depressed fractures might enhance these patients' results.[19]

The mean cortisol in our sample of patients was 25.6 µg/dl on admission, while it was 9.1 µg/dl at 6 months, consistent with previous studies.[20,21] Based on their cortisol levels, patients were separated into two categories: normal and high. Serum cortisol levels were significantly higher than normal in both groups, with mean levels at intake (>19.4 µg/dl) decreasing at 7 days and 6 months, demonstrating that cases recovered to normal levels (3.7-19.4 µg/dl).

Cortisol levels were stratified based on TBI severity. The cortisol levels at the time of injury (admission) were significantly higher at 28.6 µg/dl in patients with a GCS 3-8 compared to 22.1µg/dl in those with a GCS 9-12. Trauma patients' elevated cortisol levels are caused by inputs from the injured area that reach the limbic and reticular formations. These impulses then travel to the median eminence, releasing cortisol, and critical patients have elevated cortisol levels in response to the severity of their Condition and the human body in response to stress.[22] After a 6-month follow-up, 19 (59.4%) patients had normal cortisol levels (3-19.4µg/dl). Furthermore, the patients with initial (day 1) mean cortisol levels of 32.8±20.5 had expired, and the increased cortisol level trend on day 1 was significantly associated with the patient's unfavourable (severity and survival).

In contrast, the results demonstrated a decreasing trend in cortisol levels after 6 months, which was associated with the patient's clinical status (favorable recovery or vegetative state). Trauma patients' elevated cortisol levels are caused by inputs from the injured area that reach the limbic and reticular formations. Cortisol is then released when these impulses reach the median eminence. The anterior pituitary is stimulated to release ACTH as a result. The adrenal cortex is then stimulated to produce cortisol. As a result, cortisol levels rise during the acute stage of traumatic brain injury. Feibel et al,[23] and Chesnokova et al,[24] have found similar findings. Additionally, in individuals with moderate to severe brain lesions, our study discovered a substantial correlation between cortisol levels and GCS. Cortisol levels were significantly different between mild and severe TBI patients.

Kakati et al. showed that cortisol levels higher than 550 mg/dL prospectively enrolled 164 patients with TBI and concluded that dynamic changes in pituitary hormones in patients with TBI might reflect the severity of the injury and influence its outcome.[25] According to Iqbal et al., 74% of TBI patients either die or remain vegetative.[23] However, these changes do not occur in patients with non-severe injuries or in a significant proportion of head-injured patients with a GCS score of 8 or higher. Alexandra et al., after 1 year, and Svingos et al. at 6, 12, and 24 months reported that individuals with mild TBI had better outcomes (GOS) than those with severe TBI.[26,27]

The link between hypernatremia and TBI is complicated. Thirty to forty percent of TBI 28 patients had hypernatremia (≥ 150 mEq/L). It may be difficult to pinpoint the exact cause of hypernatremia in TBI patients. Hyperosmolar treatment, hypovolemia, insensible free water loss, or a high sodium load in IV fluids and meals can all contribute to this condition26. Nonetheless, our findings showed that a considerable risk of early death is linked to hypernatremia upon hospitalization in patients with severe TBI. Furthermore, there is a strong correlation between hypernatremia and GCS and GOS-E.

Conclusion

Cortisol levels were higher on day 1 and gradually decreased on days 7 and 6 months, as patients' status improved. The unfavorable group (1-5) had greater cortisol levels on admission, followed by a week, and at 6 months. Hypernatremia is considerably more common in patients with severe TBI. The cortisol and sodium levels were positively correlated. Hypernatremia predicts TBI severity and patient survival. Cortisol levels ≥ 25.0 µg/dL were associated with poor survival.

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Conflicts of interest

There are no conflicts of interest.

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