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PREVALENCE AND PATTERN OF POST-TRAUMATIC PITUITARY HORMONE DYSFUNCTION IN PATIENTS WITH SEVERE TRAUMATIC BRAIN INJURY

Neurosurgery

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ABSTRACT

Background: Traumatic brain injury (TBI) is a major cause of mortality and long-term disability worldwide, particularly among young adults. Post-traumatic hypopituitarism (PTHP), resulting from damage to the hypothalamic–pituitary axis, is an under-recognized complication of severe TBI and may adversely affect neurological recovery and quality of life. Early identification and management of hormonal dysfunction may improve patient outcomes. **Objectives:** To determine the proportion and pattern of pituitary hormonal dysfunction in patients with severe traumatic brain injury and to evaluate its relationship with neurological outcome, diffuse axonal injury (DAI), and skull base fractures. **Methods:** A prospective observational study was conducted in the Department of Neurosurgery, in tertiary care center for 6 month . 50 adult patients with severe TBI (Glasgow Coma Scale score 3–8) were included. Pituitary hormonal profiles, including TSH, FSH, LH, growth hormone (GH), prolactin, and cortisol, were assessed on Day 1, Day 7, Day 30, and at 3-month follow-up. Neurological outcomes were evaluated using the Glasgow Outcome Scale (GOS). **Results:** Post-traumatic hypopituitarism was identified in 38% of patients on Day 1, with 18% exhibiting a single hormonal deficiency and 20% showing multiple hormonal deficiencies. Gonadotrophic dysfunction was the most common abnormality, followed by growth hormone deficiency. Cortisol levels demonstrated an initial increase followed by a gradual decline. At 3-month follow-up, persistent hypopituitarism was observed in 8% of patients. Hormonal dysfunction was significantly associated with poorer neurological outcomes ($p = 0.047$). Skull base fractures were identified as an important risk factor for the development of pituitary dysfunction. **Conclusion:** Post-traumatic hypopituitarism is a frequent complication of severe TBI and is associated with unfavorable neurological outcomes. Routine endocrine evaluation and timely hormone replacement therapy may facilitate improved recovery and reduce long-term morbidity in affected patients.

KEYWORDS

Traumatic Brain Injury, Post-traumatic Hypopituitarism, Pituitary Hormone Dysfunction, Growth Hormone Deficiency, Neurological Outcome.

INTRODUCTION

Traumatic Brain Injury (TBI) is one of the leading causes of death and disability worldwide, particularly among young adults and individuals in the economically productive age group. It is estimated that nearly 69 million people sustain traumatic brain injuries annually, making it a major public health concern.[1] The burden of TBI is particularly high in developing countries such as India, where road traffic accidents account for the majority of cases.[2]

Traumatic brain injury results in a wide spectrum of neurological deficits ranging from mild cognitive impairment to severe disability and death. While primary brain injury occurs at the moment of impact, secondary brain injury develops over hours to days due to cerebral edema, ischemia, inflammation, and metabolic disturbances.[3] These secondary mechanisms contribute significantly to morbidity and mortality.

In recent years, increasing attention has been directed toward endocrine dysfunction following TBI. Post-traumatic hypopituitarism (PTHP) is a recognized but frequently overlooked complication resulting from damage to the hypothalamic-pituitary axis.[4] The pituitary gland is particularly vulnerable because of its unique vascular supply and anatomical location within the sella turcica. Mechanical injury, vascular compromise, increased intracranial pressure, diffuse axonal injury, and skull base fractures can all contribute to pituitary dysfunction.[5]

The prevalence of post-traumatic hypopituitarism varies considerably among studies, ranging from 20% to 68%, depending on the severity of injury and timing of hormonal evaluation.[6–9] Growth hormone deficiency and gonadotropin deficiency are the most commonly reported endocrine abnormalities.[7,8] Hormonal dysfunction may present with fatigue, impaired cognition, mood disturbances, reduced rehabilitation potential, decreased quality of life, and poor neurological recovery.[10]

Despite its clinical importance, pituitary dysfunction often remains undiagnosed because many symptoms overlap with the neurological sequelae of traumatic brain injury. Early identification and treatment of hormonal abnormalities may significantly improve functional outcomes and quality of life.[5] Therefore, assessment of pituitary function should be considered an essential component of long-term follow-up in patients with severe traumatic brain injury.

The present study was undertaken to determine the proportion and pattern of pituitary hormonal dysfunction among patients with severe traumatic brain injury and to evaluate its relationship with neurological outcomes, diffuse axonal injury, and skull base fractures.

Objectives

Primary Objective

To determine the proportion of pituitary hormonal dysfunction in severe traumatic brain injury patients.

Secondary Objectives

1. To study the pattern of pituitary hormonal dysfunction.
2. To assess the relationship between pituitary dysfunction and neurological outcome.
3. To evaluate the association between pituitary dysfunction and diffuse axonal injury (DAI).

MATERIALS AND METHODS :

Study Design : Prospective observational study.
Study Setting: Department of Neurosurgery, Tertiary care center
Study Period: 6 months

Sample Size: A sample size of 50 patients was calculated using the standard formula for prevalence studies based on previously reported prevalence rates of pituitary dysfunction after TBI.

Inclusion Criteria: Age 18–80 years., Severe traumatic brain injury with Glasgow Coma Scale (GCS) score 3–8 after resuscitation.

Exclusion Criteria: Pregnancy, Known endocrine disorders,

Malignancy, Severe psychiatric illness, Previous glucocorticoid therapy, Autoimmune disorders.

Data Collection
The following information was recorded:
Demographic characteristics, Mode of injury, Clinical findings, CT brain findings,Hormonal profile.

Hormonal evaluation included:
Thyroid Stimulating Hormone (TSH), Follicle Stimulating Hormone (FSH), Luteinizing Hormone (LH),Growth Hormone (GH), Prolactin,Cortisol Hormonal assessment was performed on: Day 1, Day 7,Day 30, 3-month follow-up Neurological outcome was assessed using the Glasgow Outcome Scale (GOS).

Statistical Analysis
Data were analyzed using SPSS version 20.
4. Categorical variables: Frequency and percentage.
5. Continuous variables: Mean ± SD.
6. Chi-square test used for associations.
7. $p < 0.05$ considered statistically significant.

RESULTS:
1) Demographic Characteristics
A total of 50 patients with severe TBI were included.

Table 1 : Demographic characteristics among patient with severe TBI

Age Distribution	Percentage	Number of patient
21–30 years	26%	13
31–40 years	24%	12
41–50 years	22%	11
51–60 years	14%	7
60 years	8%	4
11–20 years	6%	3

2) Gender Distribution
Male: 79%, Female: 21%

The predominance of males reflects their increased exposure to road traffic accidents and occupational hazards.

3) Mode of Injury

Table2: mode of injury in patient of severe TBI

Mode of injury	Number of patient	Percentage
Road traffic accident	37	74%
Falls	10	20%
Assault	3	6%
Total	50	100%

Road traffic accidents were the leading cause of severe TBI.

4) Clinical Findings
Common presenting symptoms included:

Table3 : clinical finding among patient with severe TBI

Clinical finding	Percentage	Number of patient
Vomiting	52%	26
Seizures	40%	20
Weakness	24%	12
Loss of consciousness	94%	47

The majority of patients presented with altered sensorium and loss of consciousness.

5) CT Brain Findings

Table 4: CT finding among patient with TBI

CT finding	Number of percentage	Percentage
Contusion	29	58%
Subdural hematoma	28	56%
Basal skull fracture	11	22%
Subarachnoid fracture	10	20%
Extradural hematoma	6	12%
Diffuse axonal injury	8	16%

Contusions and subdural hematomas were the most frequent radiological abnormalities.

6) Prevalence of Pituitary Dysfunction
Day-1 hormonal assessment showed:
1. No dysfunction 62%
2. Single hormone dysfunction 18%
3. Multiple hormone dysfunction 20%
Overall prevalence of post-traumatic hypopituitarism = 38%

7) Pattern of Hormonal Dysfunction
The most commonly affected hormones on Day 1 were: hormones with dysfunction percentages
a. FSH 24%
b. GH 18%
c. TSH 16%
d. Prolactin12%
e. LH 12%
f. Cortisol 8%

Gonadotrophic dysfunction was the most frequent abnormality, followed by growth hormone deficiency.

At 3-month follow-up:
a. Persistent FSH abnormality: 4%
b. Persistent GH deficiency: 4%
c. Persistent TSH abnormality: 2%
d. Overall persistent hypopituitarism: 8%.

Hormonal Trends
Growth Hormone: Growth hormone levels showed a significant decline immediately after injury, followed by gradual recovery during follow-up. Persistent GH deficiency was noted in a small proportion of patients.

Cortisol :Serum cortisol levels demonstrated an initial elevation, reflecting the physiological stress response to severe injury, followed by a gradual decline toward normal values.

Gonadotropins: FSH and LH abnormalities improved progressively over time, indicating recovery of pituitary function in most patients.
Thyroid Function : Transient central hypothyroidism was observed in several patients. Most recovered during follow-up, with only a few showing persistent abnormalities.

8)Neurological Outcome:
Glasgow Outcome Scale at 3 months:
- Good Recovery 16%
- Moderate Disability 32%
- Severe Disability 36%
- Persistent Vegetative State 16%

The majority of patients had residual disability despite survival.

9)Association Between Pituitary Dysfunction and Outcome
Pituitary dysfunction on Day 1 was significantly associated with poorer neurological outcomes.

Patients without hormonal dysfunction were more likely to achieve favorable recovery compared with patients having pituitary dysfunction.

Chi-square = 7.93
$p = 0.047$

This indicates that endocrine dysfunction may serve as an important predictor of neurological recovery after severe TBI.

DISCUSSION:
The present prospective observational study evaluated pituitary hormonal dysfunction in 50patients with severe traumatic brain injury. The study demonstrated that post-traumatic hypopituitarism is a common complication following severe head injury, with an overall prevalence of 38% during the acute phase. This finding is consistent with previous studies that reported prevalence rates ranging from 28% to 54%.[7,9]

The majority of patients in the present study were young adult males, with road traffic accidents being the predominant mechanism of injury. These findings are comparable to national and international epidemiological data, which identify young males as the population

most vulnerable to severe traumatic brain injury because of greater exposure to vehicular accidents and occupational hazards.[1,2]

Hormonal assessment revealed that gonadotrophic dysfunction was the most common endocrine abnormality, followed by growth hormone deficiency. Similar observations have been reported by Aimaretti et al.[8] and Agha et al.[9] Gonadotroph and somatotroph cells are particularly susceptible to ischemic injury because of their dependence on the portal circulation supplied by the long hypophyseal vessels. Damage to these vessels during traumatic brain injury may explain the high frequency of these hormonal deficiencies.[5]

Growth hormone deficiency was observed in a substantial proportion of patients during the acute phase. Previous investigators have also reported growth hormone deficiency as one of the most common endocrine consequences of traumatic brain injury.[6] Growth hormone plays an important role in cognitive function, muscle strength, metabolism, and rehabilitation; therefore, its deficiency may adversely affect neurological recovery.

The study also demonstrated transient abnormalities in thyroid and cortisol secretion. Cortisol levels showed an initial increase followed by gradual normalization during follow-up. This finding likely reflects activation of the hypothalamic-pituitary-adrenal axis in response to physiological stress caused by severe trauma. Similar hormonal trends have been described by Zheng et al.[13]

At three-month follow-up, only 8% of patients exhibited persistent hormonal abnormalities. This observation suggests that many endocrine disturbances occurring in the acute phase are reversible and related to temporary dysfunction rather than permanent structural damage. Nevertheless, the persistence of hormonal deficits in a subset of patients highlights the importance of long-term endocrine surveillance.

An important finding of the present study was the significant association between pituitary dysfunction and poorer neurological outcomes. Patients with hormonal abnormalities demonstrated lower Glasgow Outcome Scale scores compared with those without endocrine dysfunction ($p = 0.047$). This supports previous reports suggesting that pituitary dysfunction contributes to delayed recovery, impaired rehabilitation, and reduced quality of life following traumatic brain injury.[4,5]

The study further identified skull base fractures as an important risk factor for pituitary dysfunction. Direct mechanical injury to the pituitary gland, stalk, or hypothalamus may account for this association. Similarly, diffuse axonal injury was associated with a higher frequency of hormonal abnormalities, emphasizing the role of shearing injury in hypothalamic-pituitary damage.

The findings of the present study have important clinical implications. Routine endocrine screening should be considered in patients with severe traumatic brain injury, particularly those with diffuse axonal injury, skull base fractures, or poor neurological recovery. Early diagnosis and timely hormone replacement therapy may improve functional outcomes and reduce long-term morbidity.

In conclusion, post-traumatic hypopituitarism is a frequent and clinically significant consequence of severe traumatic brain injury. Regular endocrine evaluation should form an integral component of comprehensive neurotrauma care to facilitate early intervention and optimize recovery.

CONCLUSION

Post-traumatic hypopituitarism is a frequent complication of severe traumatic brain injury. In the present study, 38% of patients demonstrated pituitary hormonal dysfunction during the acute phase, with gonadotroph and growth hormone deficiencies being the most common abnormalities. Although most hormonal disturbances improved over time, persistent dysfunction remained in 8% of patients at three months.

Pituitary dysfunction was significantly associated with poorer neurological outcomes and was more common in patients with skull base fractures. These findings highlight the importance of early hormonal assessment and appropriate endocrine management in patients with severe traumatic brain injury.

Routine screening for pituitary dysfunction should be incorporated into the follow-up protocol of severe TBI patients to improve functional recovery and long-term quality of life.

Conflict of interest: no

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