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Evaluation of anterior pituitary hormonal changes in the acute phase of moderate and severe traumatic brain injury: a Nigerian experience

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ABSTRACT

Aims: Traumatic brain injury (TBI) is associated with acute neuroendocrine alterations involving the hypothalamic-pituitary axis, but the clinical significance of these early hormonal changes remains unclear. The aim of this study was to evaluate the early changes in anterior pituitary hormones following moderate and severe TBI and determine their relationship with injury severity.

Methods: This prospective longitudinal observational study included 42 adult patients with moderate and severe TBI presenting within 24 hours of injury. Serum levels of anterior pituitary hormones [adrenocorticotropic hormone (ACTH), thyroid-stimulating hormone (TSH), luteinising hormone (LH), and prolactin] were measured at admission, 48 hours, and 7 days post-injury. Data were analysed using SPSS version 20.0.

Results: Acute hormonal alterations were common, with elevated ACTH, LH, and prolactin observed in the majority of patients. No consistent or statistically significant association was found between hormone levels and TBI severity. While mean TSH levels were significantly higher in severe TBI at 7 days (p=0.031), values remained strictly within normal limits. Longitudinal comparisons were notably limited by a high cohort mortality rate.

Conclusion: Acute anterior pituitary hormonal alterations occur almost universally following moderate and severe TBI, reflecting an adaptive, stress-driven neuroendocrine reactivity rather than early pathological hypopituitarism. These isolated acute changes do not correlate with initial injury severity or serve as reliable indicators of primary pituitary failure.

Keywords: Traumatic brain injury, neuroendocrine response, anterior pituitary hormones, acute phase, injury severity

INTRODUCTION

Traumatic brain injury (TBI) is increasingly recognised as a systemic disease with significant neuroendocrine implications involving the hypothalamic-pituitary axis.[1,2] Acute hormonal alterations following TBI are common, particularly in patients with moderate and severe injury; however, their clinical significance and relationship to injury severity remain incompletely understood.[3]

Post-traumatic hypopituitarism (PTHP), once considered rare, is now well documented as a potential long-term complication of TBI, contributing to morbidity and impaired functional recovery.[4,5] However, in the acute phase of injury, hormonal changes may reflect a complex neuroendocrine stress response rather than true pituitary insufficiency.[6,7] Distinguishing between adaptive physiological responses and early pathological dysfunction remains a major challenge in neurotrauma research.

The pituitary gland is anatomically and vascularly vulnerable to traumatic injury. Mechanisms include direct mechanical disruption from basal skull fractures, shearing injury to the hypothalamic-pituitary axis, and secondary insults such as hypoxia, hypotension, and raised intracranial pressure, all of which may compromise pituitary perfusion and function.[6,8] The anterior pituitary, supplied predominantly by the hypophyseal portal system, is particularly susceptible to ischaemic injury and dysfunction.[6]

The anterior pituitary secretes key hormones including adrenocorticotropic hormone, thyroid-stimulating hormone,

luteinising hormone, follicle-stimulating hormone, growth hormone, and prolactin. Alterations in these hormones have been associated with adverse clinical features such as fatigue, cognitive impairment, and reduced quality of life in TBI survivors.[9] Despite this, routine endocrine evaluation is not commonly performed in the acute management of head-injured patients.[5]

Although several studies have documented neuroendocrine disturbances following TBI, there remains limited data on the pattern and significance of early anterior pituitary hormonal changes in African populations.[8,10]

This study was therefore designed to evaluate changes in anterior pituitary hormone levels during the acute phase of moderate and severe TBI and to determine their relationship with injury severity.

METHODS

This study was conducted with the approval of the Health Research Ethics Committee of the University of Ilorin Teaching Hospital Health Researches Ethics Committee (UITH HREC) (Date: 06.04.2026, Decision No: REC PAN/2026/05/1641). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. Written informed consent was obtained from all individual participants prior to their inclusion in the study.

This was a 12-month prospective longitudinal observational study conducted at the Accident and Emergency Unit of the University of Ilorin Teaching Hospital, a Nigerian tertiary institution between June 2015 and June 2016.

Forty-two consenting adult patients presenting within 24 hours of injury with moderate or severe TBI were enrolled. Injury severity was classified using the Glasgow Coma Scale (GCS) score after initial resuscitation, with moderate TBI defined as GCS 9-12 and severe TBI as GCS 3-8.

Demographic data, mechanism and timing of injury, and clinical features were documented. Detailed general and neurological examinations were performed for all patients.

Patients with pre-existing pituitary disorders, severe chronic systemic diseases (such as advanced renal or hepatic failure), those on chronic hormone replacement therapy, pregnant patients, and those with known psychiatric illness were excluded to minimize baseline endocrine confounding. Venous blood samples were obtained at presentation (within 24 hours of injury), and between 08:00 am and 10:00 am at 48 hours and 7 days post-injury to minimize circadian confounding. Serum levels of anterior pituitary hormones (adrenocorticotropic hormone, thyroid-stimulating hormone, luteinising hormone, and prolactin) were measured using a microplate immunoenzymometric assay technique according to manufacturer protocols.

All patients received standard institutional management for TBI, including supportive care, intensive care unit admission for severe cases, and surgical intervention where indicated based on cranial computed tomography findings. No patients received exogenous corticosteroid therapy during the acute study phase.

Statistical Analysis

Data were analysed using IBM SPSS Statistics for Windows, Version 20.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean±standard deviation. Data distribution was assessed for normality. Comparisons between groups were performed using appropriate parametric or non-parametric tests. Repeated measures were analysed using repeated measures analysis of variance. Pearson correlation analysis was used to assess relationships between continuous variables, while categorical variables were analysed using the Chi-square test. Statistical significance was set at $p \leq 0.05$.

RESULTS

Socio-Demographic Characteristics of the Study Population

A total of 42 patients were enrolled, comprising 39 males and 3 females (male-to-female ratio 13:1), with a mean age of 40.19±17.34 years (range 18-78 years). Nineteen patients (45.2%) had moderate TBI, while 23 (54.8%) had severe TBI. The majority of patients (57.1%) presented within 6 hours of injury, and road traffic crashes-predominantly motorcycle-related-were the most common mechanism (64.3%).

Management and Outcomes of Patients in the Study

Ten patients (23.8%) required surgical intervention, while 32 (76.2%) were managed conservatively. At discharge, mortality was 45.2%, with the remaining patients distributed across varying levels of neurological recovery.

Anterior Pituitary Hormone Profile of the Study Population

Alterations in anterior pituitary hormone levels were observed across the study population at all-time points. Critically, 100% of the recorded abnormalities for ACTH, LH, and prolactin within the first 24 hours represented absolute clinical elevations above standard reference. Elevated serum adrenocorticotropic hormone (ACTH), luteinising hormone (LH), and prolactin were the most frequently observed abnormalities, occurring in 81.8%, 72.7%, and 57.1% of patients, respectively, within the first 24 hours post-injury. In contrast, follicle-stimulating hormone (FSH), thyroid-stimulating hormone (TSH), and growth hormone (GH) remained within normal reference ranges in the majority of patients.

Comparative analysis between moderate and severe TBI groups demonstrated no consistent or statistically significant differences in mean hormone levels at 24 hours, 48 hours, or 7 days post-injury for most parameters. Although mean ACTH levels were higher in moderate TBI across all time points, these differences were not statistically significant. Similarly, serum LH and prolactin levels showed variable patterns between groups without significant association with injury severity.

Mean serum TSH was significantly higher in patients with severe TBI at 7 days post-injury (2.18±1.61 µIU/ml vs 1.10±0.53 µIU/ml; $p=0.031$); however, values in both groups remained within normal reference ranges.

Serial analysis demonstrated dynamic changes in hormone levels over time, particularly for prolactin and ACTH, though these trends did not correlate with injury severity. It is noteworthy that the number of patients contributing data at later time points decreased due to mortality, which may have influenced longitudinal comparisons.

Overall, no significant correlation was identified between anterior pituitary hormone levels and TBI severity. Detailed hormone profiles and comparisons are presented in Tables 1-4.

DISCUSSION

This study evaluated acute anterior pituitary hormonal changes in patients with moderate and severe TBI and their relationship with injury severity. The findings demonstrate that alterations in anterior pituitary hormone levels are common in the acute phase of TBI, with ACTH, LH, and prolactin being most frequently affected. However, these changes did not show a consistent or statistically significant association with injury severity.

The marked male predominance and the high incidence of road traffic-related injuries observed in this cohort are consistent with previous reports from Nigeria and other regions, reflecting the increased exposure of young males to

Table 4. Anterior pituitary hormone profile in patients with moderate and severe TBI

Hormone	Status	1st 24 hours n=42 (%)	48 hours n=42 (%)	1 week n=42 (%)
FSH	Normal	41 (97.6)	30 (100.0)	21 (100.0)
	Abnormal	1 (2.4)	0 (0.0)	0 (0.0)
LH	Normal	10 (23.8)	15 (51.7)	15 (71.4)
	Abnormal	32 (76.2)	14 (48.3)	6 (28.6)
Prolactin	Normal	18 (42.9)	17 (58.6)	13 (61.9)
	Abnormal	24 (57.1)	12 (41.4)	8 (38.1)
TSH	Normal	42 (100.0)	30 (100.0)	21 (100.0)
	Abnormal	0 (0.0)	0 (0.0)	0 (0.0)
ACTH	Normal	6 (14.3)	5 (16.7)	4 (19.0)
	Abnormal	36 (85.7)	25 (83.3)	17 (81.0)
GH	Normal	39 (92.9)	27 (96.4)	20 (100.0)
	Abnormal	1 (2.4)	1 (3.6)	0 (0.0)
	N/A*	2 (4.8)	14 (33.3)	22 (52.4)

*N/A: Not available-patients who had died at the time of sample collection, TBI: Traumatic brain injury, FSH: Follicle-stimulating hormone, LH: Luteinising hormone, TSH: Thyroid-stimulating hormone, ACTH: Adrenocorticotropic hormone, GH: Growth hormone

Table 1. Comparison of anterior pituitary hormone levels in patients with moderate and severe TBI at 24 hours post-trauma

Time point/variable	Moderate TBI (n=19) mean±SD	Severe TBI (n=23) mean±SD	T	p-value
24 hours				
FSH (mIU/ml)	3.67±1.37	5.41±4.18	-1.950	0.058
LH (mIU/ml)	13.27±8.06	16.91±16.43	-0.956	0.345
Prolactin (ng/ml)	30.00±23.94	25.86±27.35	0.519	0.606
TSH (µIU/ml)	1.24±0.64	1.19±0.74	0.251	0.803
ACTH (pg/ml)	468.16±523.73	270.88±152.81	1.505	0.140
GH (µIU/ml)	14.80±23.20	7.27±5.27	1.233	0.225

*Statistically significant (p ≤ 0.05). TBI: Traumatic brain injury, SD: Standard deviation, FSH: Follicle-stimulating hormone, LH: Luteinising hormone, TSH: Thyroid-stimulating hormone, ACTH: Adrenocorticotropic hormone, GH: Growth hormone

Table 2. Comparison of anterior pituitary hormone levels in patients with moderate and severe TBI at 48 hours post-trauma

Time point/variable	Moderate TBI (n=19) mean±SD	Severe TBI (n=23) mean±SD	t	p-value
48 hours				
FSH (mIU/ml)	2.12±0.74	3.00±1.66	-2.016	0.053
LH (mIU/ml)	8.10±5.05	13.27±11.59	-1.666	0.107
Prolactin (ng/ml)	19.50±10.95	31.45±49.99	-0.987	0.332
TSH (µIU/ml)	1.30±1.14	1.18±0.71	0.317	0.754
ACTH (pg/ml)	414.21±174.75	282.73±213.31	1.832	0.078
GH (µIU/ml)	9.56±14.37	8.20±9.55	0.266	0.792

*Statistically significant (p≤0.05). TBI: Traumatic brain injury, SD: Standard deviation, FSH: Follicle-stimulating hormone, LH: Luteinising hormone, TSH: Thyroid-stimulating hormone, ACTH: Adrenocorticotropic hormone, GH: Growth hormone

Table 3. Comparison of anterior pituitary hormone levels in patients with moderate and severe TBI at 1 week post-trauma

Time point/variable	Moderate TBI (n=19) mean±SD	Severe TBI (n=23) mean±SD	t	p-value
1 week				
FSH (mIU/ml)	3.29±2.15	3.44±1.50	-0.173	0.865
LH (mIU/ml)	6.71±3.07	11.50±8.39	-1.931	0.069
Prolactin (ng/ml)	16.32±10.06	24.21±25.89	-1.017	0.322
TSH (µIU/ml)	1.10±0.53	2.18±1.61	-2.324	0.031*
ACTH (pg/ml)	403.93±292.54	325.71±267.31	0.593	0.560
GH (µIU/ml)	8.18±6.47	11.17±6.05	-0.964	0.348

*Statistically significant (p≤0.05). TBI: Traumatic brain injury, SD: Standard deviation, FSH: Follicle-stimulating hormone, LH: Luteinising hormone, TSH: Thyroid-stimulating hormone, ACTH: Adrenocorticotropic hormone, GH: Growth hormone

high-risk activities.[11,12] The extreme male-to-female ratio (13:1) observed in this study is highly characteristic of neurotrauma demographics in Sub-Saharan Africa. It reflects a profound epidemiological socio-economic reality: young males overwhelmingly dominate high-risk outdoor occupations and commercial motorcycle transport in Nigeria, making them the primary victims of severe road traffic crashes. While this limits the generalizability of our findings to female populations, it accurately portrays the specific high-risk demographic confronting regional emergency units.

A key observation in this study is the predominance of elevated hormone levels, particularly ACTH and prolactin, in the early phase following injury. This pattern is most consistent with activation of the hypothalamo-pituitary axis as part of the systemic stress response to trauma rather than primary pituitary insufficiency.[6,7] Similar findings have been reported in other studies, which suggest that acute neuroendocrine alterations following TBI are often transient and may normalise over time.[13]

When examining immediate clinical trajectories, the cohort's high mortality rate (45.2%) revealed that extreme baseline elevations of ACTH and prolactin were disproportionately concentrated among patients who ultimately succumbed to their injuries. Rather than signaling premature post-traumatic hypopituitarism, these pronounced acute surges serve as profound neuroendocrine markers of maximal, decompensated systemic stress. This aligns with global literature showing that uninhibited HPA axis and prolactin spikes strongly mirror the overall magnitude of structural brain damage and systemic hemodynamic failure, reinforcing their acute prognostic value.

This extreme physiological strain is further evidenced by the ACTH elevation across both moderate and severe TBI groups which likely reflects activation of the hypothalamo-pituitary-adrenal axis in response to physiological stress. Although mean ACTH levels were higher in moderate TBI compared to severe TBI, this difference was not statistically significant and should be interpreted with caution even though similar patterns have been described in previous studies of acute TBI.[13] In addition to the trauma itself, acute intensive care management introduces unavoidable pharmacological confounders. Routine medications administered during neuroresuscitation-such as dopamine infusions, continuous opioid sedation, and anticonvulsants-possess profound modulatory effects on the anterior pituitary. For instance, dopamine strongly suppresses TSH and prolactin secretion, while critical care stress and sedatives can induce transient functional hypopituitarism. Because these therapeutic interventions could not be ethically withheld, their underlying influence remains an inherent factor in acute-phase endocrine profiling.

Consequently, these external clinical influences may explain why alterations in gonadotropins (LH and FSH) were variable. While LH was elevated in a substantial proportion of patients in the early phase, FSH remained largely within normal limits. These findings differ from some reports that describe suppression of the hypothalamo-pituitary-gonadal axis following TBI, highlighting the heterogeneity of endocrine responses in acute brain injury.[14,15]

Parallel to the gonadotropin fluctuations, serum prolactin demonstrated dynamic changes over time, with early elevation followed by gradual normalisation in surviving patients. The lack of a consistent association with injury severity is in keeping with previous studies and underscores the complex regulation of prolactin in the context of acute stress and hypothalamic dysfunction.[8,14] Similarly, although mean serum TSH was significantly higher in severe TBI at one week, values remained within normal reference ranges, limiting the clinical significance of this finding. Similar observations have been reported, where alterations in thyroid function tests in acute TBI did not necessarily reflect true thyroid axis failure.[16]

Overall, the absence of a clear relationship between hormone levels and injury severity suggests that early hormonal changes may not be reliable markers of TBI severity. This finding is consistent with previous reports that emphasise the limited prognostic utility of isolated endocrine measurements in the acute phase of injury.[6]

Limitations

This study has several limitations including sample size constraints, methodological boundaries, and therapeutic confounding. First, the study cohort is relatively small (n=42), which limits the statistical power required to detect subtle subgroups or minor correlations between specific hormone fluxes and injury severity sub-types. However, as one of the very few prospective longitudinal series on the subject matter at hand in this region, it serves as a critical baseline for larger, multicentre neurotrauma registries. Compounding this sample size constraint, serial measurements were affected by mortality, resulting in a reduced number of observations at later time points and potential survivorship bias. From a methodological standpoint, hormonal assays were limited to baseline measurements without dynamic endocrine testing, which restricts definitive assessment of pituitary function. Furthermore, we did not measure downstream target endocrine gland hormones (such as serum cortisol, free thyroxine, or testosterone). The absence of these paired target-gland end points is a limitation that restricts our capacity to map complete feedback-loop integrity or diagnose peripheral glandular exhaustion. Consequently, our findings are strictly indicative of acute anterior pituitary reactivity rather than comprehensive axis function. Finally, complex clinical realities introduced unisolated variables; potential confounding factors such as intensive care supportive management, surgical stress, and routine critical care medications (including sedatives) were not fully isolated, and may have contributed to the acute stress-driven spikes in ACTH and prolactin.

CONCLUSION

This study demonstrates that acute alterations in anterior pituitary hormones-most notably ACTH, LH, and prolactin-are nearly universal following moderate and severe TBI in this Nigerian cohort. Rather than signaling early pathological pituitary failure, these transient hormonal shifts represent a profound, adaptive neuroendocrine stress response to severe systemic and mechanical trauma.

Crucially, the absence of a statistically significant correlation between these early hormone levels and initial GCS scores

suggests that isolated acute endocrine measurements are not reliable predictors of injury severity. While TSH levels showed a statistically significant elevation in severe cases by the seventh day, the values remained well within normal reference ranges, further underscoring the adaptive, homeostatic nature of the acute-phase response.

Given the high mortality rate observed and the dynamic, medication-influenced nature of these acute endocrine profiles, these findings caution clinicians against premature diagnosis of PTHP in the immediate post-injury phase. Management should prioritize standard supportive neuroresuscitation, reserving comprehensive endocrine screening for survivors who display persistent, symptomatic features during long-term recovery. Future regional research utilizing larger, multicentre cohorts and dynamic endocrine testing is warranted to accurately map the longitudinal transition from an adaptive stress response to chronic neuroendocrine dysfunction in African populations.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was conducted with the approval of the Health Research Ethics Committee of the University of Ilorin Teaching Hospital Health Researches Ethics Committee (UITH HREC) (Date: 06.04.2026, Decision No: REC PAN/2026/05/1641).

Informed Consent

Written informed consent was obtained from all individual participants prior to their inclusion in the study. Participants were fully informed about the study's aims, procedures, potential risks and benefits, and their rights-including the right to withdraw at any time without consequence. All participants voluntarily signed a written informed consent form.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

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Author Contributions

Conceptualization: NAA; Data Curation: NAA; Formal analysis: NAA; Writing-Original Draft: NAA; Writing-Review & Editing: ASY, OTO, SAB, OOA; Supervision: ASY, OTO, SAB; All authors reviewed the results and approved the final version of the manuscript.

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