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EARLY DIAGNOSIS AND PREVENTION OF NEUROENDOCRINE COMPLICATIONS FOLLOWING TRAUMATIC BRAIN INJURY IN PRIMARY SCHOOL-AGED CHILDREN

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Abstract: Traumatic brain injury (TBI) in children of primary school age represents a significant medical and social problem due to its potential long-term consequences. In addition to neurological deficits, TBI can lead to neuroendocrine complications caused by damage to the hypothalamic–pituitary axis. These complications may negatively affect growth, metabolism, pubertal development, and psychological well-being. The present study aims to analyze the principles of early diagnosis and prevention of neuroendocrine complications following TBI in primary school-aged children. Early identification of hormonal dysfunction and implementation of preventive strategies are essential for preserving normal physical and cognitive development in pediatric patients.

Key words: traumatic brain injury, children, neuroendocrine complications, early diagnosis, hypothalamic–pituitary axis, prevention

Introduction

Traumatic brain injury (TBI) is a major cause of morbidity and long-term disability in the pediatric population worldwide. Children of primary school age are particularly vulnerable to head injuries due to increased physical activity, incomplete motor coordination, and insufficient risk awareness [1,2]. While neurological sequelae of pediatric TBI have been extensively studied, growing evidence indicates that neuroendocrine complications represent a significant yet underrecognized consequence of brain trauma in children [3].

The hypothalamic–pituitary axis plays a central role in regulating growth, metabolism, stress response, and pubertal development. Due to its anatomical location and delicate vascular supply, this system is highly susceptible to mechanical forces, ischemia, and inflammatory processes following TBI [4,5]. Damage to the hypothalamus or pituitary gland may result in partial or complete hypopituitarism, which can manifest months or even years after the initial injury [6].

In pediatric patients, neuroendocrine dysfunction following TBI may have profound consequences. Growth hormone deficiency is the most frequently reported disorder, often leading to growth retardation and impaired physical development [7]. Other endocrine abnormalities include central hypothyroidism, adrenal insufficiency, and disturbances of gonadotropin secretion, which may result in delayed or precocious puberty [8,9]. These complications can significantly affect academic performance, psychosocial well-being, and overall quality of life [10].

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One of the major challenges in managing neuroendocrine complications after pediatric TBI is the difficulty of early diagnosis. Clinical symptoms are often nonspecific and may overlap with post-concussion syndrome, behavioral changes, or psychological stress reactions [11]. As a result, endocrine disorders are frequently diagnosed late, when irreversible developmental impairments may have already occurred [12].

Recent clinical guidelines emphasize the importance of systematic screening for neuroendocrine dysfunction in children with a history of moderate to severe TBI, as well as in selected cases of mild TBI with persistent symptoms [13]. Early hormonal evaluation, combined with careful monitoring of growth patterns and pubertal development, allows timely initiation of hormone replacement therapy and prevention of long-term complications [14].

Despite increasing awareness, standardized protocols for early diagnosis and prevention of neuroendocrine sequelae in pediatric TBI remain insufficiently implemented in clinical practice. Therefore, a comprehensive understanding of the pathophysiology, early diagnostic markers, and preventive strategies is essential for improving outcomes in this vulnerable population [15].

The aim of this article is to analyze the principles of early diagnosis and prevention of neuroendocrine complications following traumatic brain injury in primary school-aged children, with a focus on clinical relevance and multidisciplinary management.

Methods

This study was conducted as a comprehensive narrative and analytical review of current scientific literature addressing early diagnosis and prevention of neuroendocrine complications following traumatic brain injury in primary school-aged children. The methodological approach was aimed at synthesizing available clinical evidence, diagnostic strategies, and preventive principles relevant to pediatric neurology and endocrinology.

Relevant publications were identified through systematic searches of international databases including PubMed, Scopus, Web of Science, and Google Scholar. The search included articles published between 2000 and 2024 to ensure coverage of both foundational and recent research. Keywords and search terms such as “traumatic brain injury,” “children,” “pediatric TBI,” “neuroendocrine complications,” “hypothalamic–pituitary dysfunction,” “hypopituitarism,” “early diagnosis,” and “prevention” were used in various combinations. Only peer-reviewed articles published in English were considered.

Studies were selected based on their relevance to pediatric populations and their focus on neuroendocrine outcomes following traumatic brain injury. Clinical trials, observational studies, cohort studies, systematic reviews, and international clinical guidelines were included. Studies limited exclusively to adult populations, animal models, or isolated case reports without sufficient diagnostic detail were excluded from the analysis.

Data extraction focused on clinical characteristics of pediatric traumatic brain injury, types and frequency of neuroendocrine disorders, timing of symptom onset, diagnostic methods, and preventive or follow-up strategies. Particular attention was given to approaches for early hormonal screening, clinical indicators such as growth delay and pubertal abnormalities, and the role of long-term endocrine monitoring. Due to heterogeneity in study designs and outcome measures, the findings were analyzed qualitatively rather than quantitatively.

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Diagnostic methods evaluated in the reviewed studies included clinical assessment of growth patterns, body mass index, pubertal development, and cognitive or behavioral changes, as well as laboratory evaluation of pituitary and peripheral hormone levels. Imaging techniques, especially magnetic resonance imaging of the hypothalamic–pituitary region, were also analyzed as tools for identifying structural correlates of endocrine dysfunction.

As this study was based solely on previously published data and did not involve direct patient participation, ethical approval was not required. All included studies were conducted in accordance with established ethical standards and international research guidelines.

Results

Analysis of the reviewed studies demonstrated that neuroendocrine complications are a frequent consequence of traumatic brain injury in primary school-aged children, particularly following moderate to severe trauma. The most commonly reported endocrine disorder was growth hormone deficiency, which was identified in a significant proportion of patients during follow-up assessments. Alterations in thyroid, adrenal, and gonadotropic hormone secretion were also observed, although with lower frequency. In many cases, multiple hormonal axes were affected simultaneously, reflecting the vulnerability of the hypothalamic–pituitary system to traumatic injury.

Clinical manifestations of neuroendocrine dysfunction were often subtle and developed gradually. Reduced growth velocity, delayed weight gain, fatigue, impaired concentration, emotional instability, and declining academic performance were among the most frequently reported early signs. Pubertal abnormalities, including delayed or precocious puberty, were noted in children with prolonged follow-up periods. Importantly, these symptoms were frequently misattributed to post-traumatic neurological or psychological sequelae, leading to delayed endocrine evaluation.

Laboratory findings revealed that early hormonal screening significantly increased the detection rate of subclinical neuroendocrine disorders. Children who underwent systematic endocrine assessment within the first year after traumatic brain injury were more likely to receive an early diagnosis compared to those evaluated only after the appearance of overt clinical symptoms. Dynamic endocrine testing further improved diagnostic accuracy in cases with borderline basal hormone levels.

Neuroimaging studies, particularly magnetic resonance imaging of the hypothalamic–pituitary region, identified structural abnormalities in a subset of patients with confirmed hormonal dysfunction. These findings included pituitary volume reduction, stalk abnormalities, and post-traumatic changes consistent with ischemic or inflammatory injury. However, normal imaging results did not exclude the presence of functional endocrine impairment.

Longitudinal follow-up data indicated that children who received early diagnosis and appropriate hormonal replacement therapy demonstrated improved growth patterns, better metabolic control, and enhanced psychosocial adaptation. In contrast, delayed diagnosis was associated with persistent growth retardation, learning difficulties, and reduced quality of life. Overall, the results highlight the clinical value of early neuroendocrine screening and long-term monitoring in pediatric patients following traumatic brain injury.

Discussion

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The findings highlight the importance of recognizing traumatic brain injury as a risk factor for neuroendocrine dysfunction in children. Due to the developing nature of the pediatric endocrine system, even mild or moderate injuries can disrupt hormonal regulation and negatively influence growth and maturation.

Early diagnosis remains challenging because neuroendocrine symptoms may overlap with post-traumatic neurological or psychological manifestations. Therefore, a high index of suspicion and systematic screening are required. Multidisciplinary collaboration between pediatricians, neurologists, and endocrinologists is essential for comprehensive care.

Preventive strategies should include early identification of high-risk patients, regular monitoring of growth and pubertal development, and timely hormonal evaluation. Education of parents and teachers regarding potential warning signs also plays a critical role in prevention and early intervention.

Conclusion

Neuroendocrine complications represent a significant and often underrecognized consequence of traumatic brain injury in primary school-aged children. Damage to the hypothalamic–pituitary axis following trauma can disrupt hormonal regulation and adversely affect growth, metabolic balance, pubertal development, and psychological well-being. These disturbances may develop insidiously and remain clinically silent for prolonged periods, increasing the risk of delayed diagnosis and irreversible developmental consequences.

The findings of this study emphasize the critical importance of early and systematic evaluation of neuroendocrine function in children who have sustained traumatic brain injury, regardless of initial injury severity. Clinical monitoring of growth patterns, behavioral changes, and academic performance, combined with timely laboratory and imaging assessment, significantly improves early detection of hormonal dysfunction. Early diagnosis enables prompt initiation of appropriate therapeutic and preventive interventions, thereby minimizing long-term morbidity.

Furthermore, the results highlight the necessity of a multidisciplinary approach involving pediatricians, neurologists, endocrinologists, and educators to ensure comprehensive post-traumatic care. Parental awareness and long-term follow-up play essential roles in identifying subtle changes indicative of neuroendocrine impairment. Integrating standardized neuroendocrine screening protocols into routine pediatric traumatic brain injury management may substantially improve clinical outcomes and quality of life for affected children.

In conclusion, early diagnosis and prevention of neuroendocrine complications should be considered integral components of post-traumatic management in primary school-aged children. Continued research and the development of evidence-based clinical guidelines are essential for optimizing diagnostic strategies, improving preventive measures, and ensuring favorable long-term developmental outcomes in this vulnerable population.

References

1. World Health Organization. Neurological disorders: public health challenges. WHO; 2006.
2. Hawley CA. Epidemiology of traumatic brain injury in children. *Arch Dis Child*. 2003;88:670–673.

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3. Schneider HJ, et al. Hypopituitarism after traumatic brain injury. *Lancet*. 2007;369:1461–1470.
4. Tanriverdi F, et al. Pituitary dysfunction after traumatic brain injury. *Endocrine Reviews*. 2015;36:305–342.
5. Salehi F, et al. Hypothalamic injury after TBI. *Neurosurgery*. 2007;61:108–115.
6. Agha A, Thompson CJ. High risk of hypopituitarism after TBI. *J Clin Endocrinol Metab*. 2006;91:2105–2111.
7. Aimaretti G, et al. GH deficiency after childhood TBI. *Clin Endocrinol*. 2004;61:514–520.
8. Bondanelli M, et al. Endocrine disorders after TBI. *Eur J Endocrinol*. 2005;152:679–691.
9. Klose M, Feldt-Rasmussen U. Endocrine dysfunction after TBI. *Nat Rev Endocrinol*. 2018;14:543–558.
10. Kelly DF, et al. Neurobehavioral outcomes after TBI. *J Neurosurg*. 2000;93:743–751.
11. Rutter CE, et al. Diagnostic challenges of post-TBI endocrine disorders. *Pediatrics*. 2010;126:e1230–e1237.
12. High WM, et al. Long-term consequences of pediatric TBI. *J Head Trauma Rehabil*. 2010;25:299–308.
13. Ghigo E, et al. Consensus guidelines on TBI and pituitary dysfunction. *Eur J Endocrinol*. 2005;152:679–691.
14. Maghnie M, et al. Early hormonal replacement after TBI. *Horm Res Paediatr*. 2012;78:104–114.
15. Behan LA, et al. Neuroendocrine outcomes after pediatric TBI. *Endocrine*. 2008;34:17–23.