



EDITORIAL
EDİTORYAL

Hypopituitarism Due to Head Trauma: New Insights into an Old Story

Kafa Travmasına Bağlı Hipofiz Yetmezliği: Eski Hikayeye Yeni Bakış

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Ulutabanca et al. (1) have published a study investigating the pituitary functions during the acute phase of traumatic brain injury (TBI) in pediatric cases in the September 2013 issue of Erciyes Medical Journal. In their elegant study, the authors have clearly demonstrated that nearly 20% of patients had ACTH deficiency (secondary adrenal insufficiency) and 10% had TSH deficiency during the first 24 hours of head injury. In recent years, TBI induced hypopituitarism became a hot topic in the field of adult and pediatric endocrinology. Since the signs and symptoms of pituitary hormone deficiencies are subtle, hypopituitarism might be easily overlooked by the physicians. The morbidity and mortality of the patients may increase due to hypopituitarism in both the acute and chronic phases of TBI. Therefore, awareness of the clinicians regarding the pituitary consequences of head trauma is crucially important, because all the hormonal deficiencies are easily treatable if recognized.

Traumatic brain injury is an important public health problem worldwide with an overall incidence of 200-235 cases per 100.000 persons per year. Among the various causes of TBI, approximately 50% are the result of motor vehicle accidents (which is also the leading cause in Turkey), falls are the second most common cause (20-30%) and violence related incidents account for nearly 20% of TBI (2). Although TBI had been considered as an exceptional cause of hypopituitarism over the last decades, an increased prevalence of neuroendocrine dysfunction in patients with TBI has been reported in recent years (3, 4). Most of the studies related to TBI induced pituitary dysfunction in the literature were performed in adult patients. Based on the current literature, including the Turkish population data, 25-50% of patients with TBI have been reported to have some degree of pituitary dysfunction in both the acute phase and chronic phases following head trauma (4-7). In a current meta-analysis including 1015 adult TBI patients, the pooled prevalence of hypopituitarism in the chronic phase after TBI was found to be 27.5% (8). Another type of head trauma which could be considered as mild TBI is sports related chronic repetitive head trauma. While the relationship between the contact sports (boxing, kickboxing, football, soccer and ice-hockey) and TBI is well documented, pituitary consequences of chronic repetitive head trauma due to sports have not been investigated until recent years. Current studies from our clinic clearly demonstrated that sports related head trauma due to amateur boxing and kickboxing might result in pituitary hormone deficiencies (9-11). Based on the studies published in the last decade, head trauma is no longer accepted as a rare cause of hypopituitarism in adult patients (8). Nevertheless, there are an insufficient number of studies in pediatric patients to draw a firm conclusion, but the available literature shows that TBI induced hypopituitarism in children is less frequent than adult patients, especially in the chronic phase (12, 13). However, pituitary hormone deficiencies, ACTH deficiency in particular, seem to be substantially higher in the pediatric population in the acute phase after head trauma (1, 14).

Therefore, clinicians should be alert for ACTH deficiency while managing critically ill TBI patients, since physiological stress response is dependent on an intact hypothalamo-pituitary-adrenal axis in critically ill patients (15). Steroid replacement therapy in ACTH deficient patients in the acute phase of head trauma would be lifesaving if the physicians remember the risk of hypopituitarism due to TBI and make the correct diagnosis.

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Submitted/Geliş Tarihi
21.10.2013

Accepted/Kabul Tarihi
21.10.2013

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Conflict of Interest

No conflict of interest was declared by the author.

Peer-review: Commissioned, not externally peer-reviewed.

Çıkar Çatışması

Yazarlar herhangi bir çıkar çatışması bildirmemişlerdir.

Hakem değerlendirmesi: Kurul tarafından değerlendirilmiştir.

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