# Posttraumatic hypopituitarism

Trauma is a rare cause of hypopituitarism. It may follow closed head injury (with or without basilar skull fracture) or penetrating trauma [1]. In 20 cases in the literature [2] all had deficient growth hormone and gonadotropin, 95% had corticotropin deficiency, 85% had reduced TSH, 63% had elevated PRL. Only 40% had transient or permanent DI.

Patients with traumatic brain injury (TBI) may develop pituitary dysfunction.

Early after the TBI, pituitary dysfunction/s differ than those occurring after few weeks and months. Growth hormone deficiency (GHD) and alterations in puberty are the most common. After the one to more years of TBI, pituitary dysfunction tends to improve in some patients but may deteriorate in others.

GH deficiency as well as Hypogonadism and thyroid dysfunction are the most common permanent lesions. Many of the symptoms of these endocrine defects can pass unnoticed because of the psychomotor defects associated with the TBI like depression and apathy. Unfortunately pituitary dysfunction appear to negatively affect psycho-neuro-motor recovery as well as growth and pubertal development of children and adolescents after TBI. Therefore, its important to follow the patients, especially children and adolescents for growth and other symptoms and signs suggestive of endocrine dysfunction. In addition, all should be screened serially for possible endocrine disturbances early after the TBI as well as few months to a year after the injury. Risk factors for pituitary dysfunction after TBI include relatively serious TBI (Glasgow Coma Scale score < 10 and MRI showing damage to the hypothalamic pituitary area), diffuse brain swelling and the occurrence of hypotensive and/or hypoxic episodes [3].

## Epidemiology

see Posttraumatic hypopituitarism epidemiology.

## Diagnosis

Apparent diffusion coefficient (ADC) is a sensitive and independent marker of pituitary damage following traumatic insult, which is useful to detect the microstructural damage in pituitary in normal appearing brain [4].

## Case series

A total of 105 patients have been assessed in two cohorts: (i) 58 patients in serial cohort and (ii) 47 patients in cross-sectional late cohort. Alavi et al. found that in serial cohort, 10.3% (6/58) of TBI patients had abnormalities of the pituitary-adrenal axis in the acute phase (Day 0-7 post injury). In comparison, in cross-sectional late cohort, 21.3% (10/47) of the patients developed dysfunction in at least one of their pituitary axes at 6 months or more post-TBI, with hypogonadotrophic hypogonadism being the most common. Twenty-two patients from these two cohorts had their growth hormone

assessment at 12 months or more post-TBI and 9.1% (2/22) were found to have growth hormone deficiency. The results suggest that post-traumatic hypopituitarism (PTHP), is a common condition amongst sufferers of TBI, and appropriate measures should be taken to detect and manage it [5].

# References


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