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Case Report

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# HYPOPITUITARISM FOLLOWING TRAUMATIC BRAIN INJURY (TBI)

**[1]Amer O. Al Ali, MD, [1]Mohammed A. Sweid, MD, [1]Ahmad A. Majrabi, MD, [1]Marwah AM. Al Agsam, MD, [2]*Nasir A. M. Al Jurayyan, MD**

[1]Department of Pediatrics King Fahad Hospital, Jizan, Saudi Arabia.
[2]Department of Pediatrics College of Medicine and King Khalid University Hospital King Saudi University, Riyadh, Saudi Arabia.

***Corresponding Author: Nasir A. M. Al Jurayyan, MD**

Department of Pediatrics College of Medicine and King Khalid University Hospital King Saudi University, Riyadh, Saudi Arabia.

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**ABSTRACT**

Traumatic Brain Injury (TBI) is a major cause of death and disability in children, Post-traumatic hypopituitarism was initially recognized more than a century, but it was thought to be a rare occurrence. We describe a case of Post-traumatic panhypopituitarism following head injury. He presented with recurrent hypoglycemia and short stature, and proved to have growth hormone, Thyroid-stimulating hormone (TSH) and cortisol deficiencies. Magnetic Resonance Imaging (MRI) revealed an empty Sella turcia. Accurate evaluation and long-term follow up are necessary to detect the occurrence of hypopituitarism. In order to improve the outcome and quality of life of traumatic brain injury (TBI), an adequate replacement therapy is of paramount importance.

**KEYWORDS:** Cortisol deficiency, growth hormone deficiency, hypoglycemia, hypopituitarism, short stature, traumatic brain injury, Thyroid –Stimulating Hormone (TSH).

## INTRODUCTION

Traumatic Brain Injury (TBI) is a major cause of death and disability in children. Post traumatic hypopituitarism was initially recognized more than a century, but it was thought to be a rare occurrence. TBI has a wide range of consequences, ranging from physical disabilities in long-term, cognitive, behavioral, psychological and social defects.[1-3] these long-term consequences make TBI as a major health problem. TBI has been demonstrated to be a frequent cause of hypothalamic-pituitary dysfunction. The pituitary dysfunction presents more frequently as an isolated, and more rarely as a complete deficiency. Gonadotropin and growth hormone defects appear to be the most common. Although some reports indicated on high occurrence of central hypothyroidism. Diabetes insipidus may be frequent in the early, acute phase post traumatic brain injury, but it is rarely permanent.[4-9]

Here in, we describe a 9 year-old boy who was involved in a car accident at the age of one year, and presented later with short stature and frequent hypoglycemia. He proved to have panhypopituitarism without diabetes insipidus (DI).

## CASE SUMMARY

A 9-year-old boy was seen in the endocrine clinic, King Fahad Hospital (KFH), Jizan, Saudi Arabia, for being short, and suffered from recurrent attacks of hypoglycemia for two years. He was the product of full term normal pregnancy and delivery with a birth weight of 3.2 kgs. Both parents were average in height. He had no family history of constitutional delay. He is not on any medications. Past medical history was complicated by road-traffic accident (at one year of age), where his father also died at that time. Physical examination showed a short child with height of 120 cm, the mean height of four years and six month. Laboratory investigations revealed low growth hormone concentration at 0.4 and 0.5 nmol/L normal >10,at low blood sugar of 1.7 mmol/L, serum concentration of cortisol at 8.00 am 45 nmol/L, normal; 150-830), and Adenocorticotrophin (ACTH) of 3 Pmol/L (normal; 5-18), suggestive of secondary cortisol deficiency. Serum free thyroxine ($FT_4$) of 8 Pmol/L (normal; 10-25) and thyroid stimulating hormone (TSH) of 2 mU/L (normal; 0.5 -5) indicating central hypothyroidism. Magnetic Resonance Imaging (MRI) (figure) of the brain and the pituitary region, showed changes of an old trauma and the sella turcica is empty. Bone age equivalent to four years. Hypothalamic-pituitary-gonadal axis was not tested. Normal serum electrolyte and urinary specific gravity. The diagnosis of panhypopituitarism without diabetes insipidus (DI) was entertained. Therefore, he was started initially on hydrocortisone, and later thyroxine. He would be started on growth hormone therapy, soon.

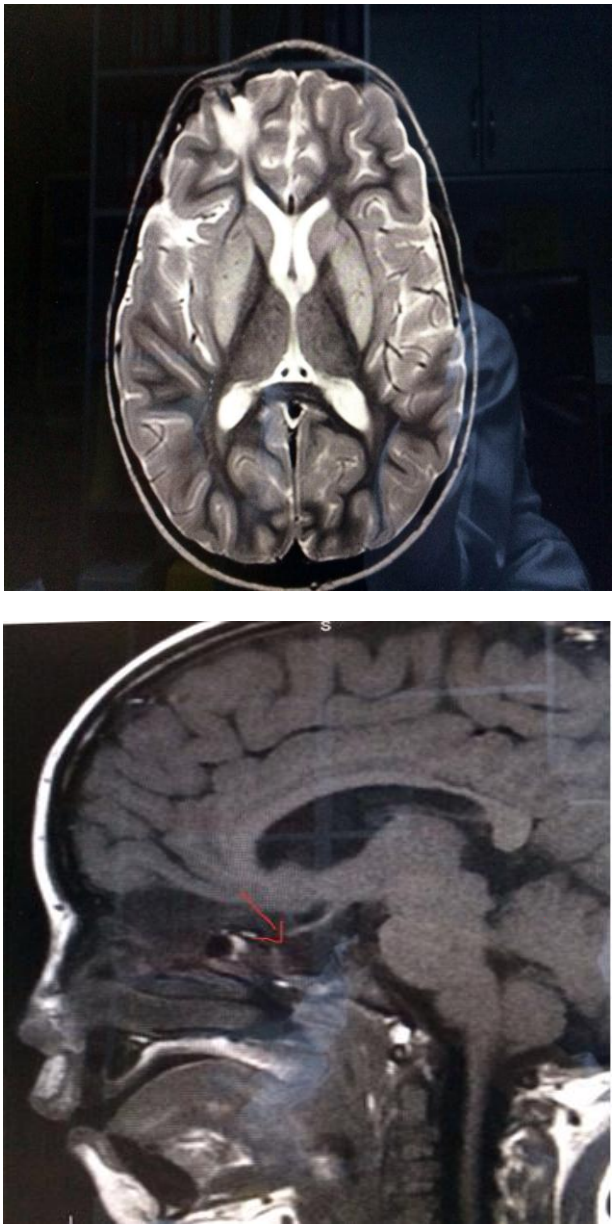

**Figure: Magnetic Resonance Images (MRI), showing changes of an old trauma and the sella turcica is empty (arrow).**

## DISCUSSION

Traumatic Brain Injury (TBI) is a major public problem that requires more effective strategies to improve the outcome. Changes in the pituitary hormone secretion may be occurred during the acute phase post trauma, representing part of the acute adoptive response. In most cases, hypopituitarism is usually diagnosed within the first year post injury, however, it may occur at any time. Pituitary dysfunction, may follow mild trauma, although, severity of TBI seems to be an important risk factor for developing post traumatic hypopituitarism.

Medical complications occur not only immediate post-injury period with implications for early rehabilitation, but may persist for many months or become chronic. Neuroendocrine dysfunction occurs in approximately 40% of patients with TBI, however, it might be transient. The association between TBI and diabetes insipidus has been recognized for many years, but generally very rare and can be transient. Also syndrome of inappropriate secretion of anti diuretic hormone (SIADH) can manifest during the immediate post-TBI period.[9-11]

The prevalence of specific pituitary hormones deficiency is variable in the literature[12] Growth hormone (GHD) and gonadotropin deficiency appear to be the most common. In accordance with the anatomical site of gonadotrope and somatotrope cells in the vascular territory of the long hypophyseal portal system, which can be easily affected by TBI.

The diagnosis of GHD is performed by clinical and biochemical criteria. The current consensus is that patients with appropriate clinical history should have the diagnosis of GHD confirmed by a provocative test of growth hormone (GH) secretion[13,14] Corticotrope function is generally tested by measuring serum cortisol concentration from an early morning samples on two or more occasion, coupled with ACTH concentration. When the morning cortisol value is persistently in the Lower limit of the normal range, a test of ACTH reserve should be performed. Also, recurrent hypoglycemia is an important clinical measure of childhood central hypoadrenalin.[15] The lack of unrecognized pituitary factors such as B-endorphin has been suggested. A low thyroxine level in the absence of cleveted thyroid stimulating hormone (TSH) indicates secondary hypothyroidism and rarely the response to thyroid-realizing hormone (TRH) stimulation test is required. This is not rare.

In conclusion, accurate evaluation and long-term follow up of patients with traumatic brain injury (TBI are necessary in order to detect the occurrence of hypopituitarism, regardless of clinical evidence for pituitary dysfunction. It is therefore, necessary that medical professional involved in the management of TBI patients, be aware of this issue in order to timely diagnose pituitary dysfunction and adequately replace the deficiency if indicated in order to improve outcome and quality of life of patients with traumatic brain injury.

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## REFERENCES

1. Salazar AM, Warden DI, Schwab K et al. Cognitive rehabilitation for traumatic brain injury: a randomized trial. Defense and Veterans Head Injury Program (DVHIP) study group. J AM M Association, 2000; 283: 3075-3081.

2. Khan F, Baguley IJ, Cameron ID. Rehabilitation after traumatic brain injury. Med J Australia, 2003; 178:290-295.
3. Consensus conference. Rehabilitation of persons with traumatic brain injury. NIH Consensus Development Panel on Rehabilitation of persons with traumatic brain injury. J Am Med Association, 1999; 282: 974-983.
4. Poomthavorn P, maixner W zacharin M. Pituitary function in pediatric survivary severe traumatic brain injury. Arch Dis Child, 2008; 93: 133-137.
5. Bondanelli M, Ambrosio MR, Zatelli MC,De Marinis L, Degli liberti Éc. Hypopituitarism after traumatic brain injury. Eur J Endocrinology, 2005; 152: 679-691.
6. Bondanelli M, de Marinis I, Ambrosio MR et al. Occurrence of pituitary dysfunction following traumatic brain injury. J Neurotrauma, 2004; 21: 685-698.
7. Aimaretti G, Ambrosio MR, Di Somma C et al. Traumatic brain injury and sub-arachnoid haemorrhage are conditions at high risk for hypopituitarism: screening study at 3 months after brain injury. Clin Endocrinol, 2004; 61: 320-326.
8. Lieberman SA, Oberoi AL, Gilkinson CR, Masel BE, Urban RJ. Prevalence of neuroendocrine dysfunction in patients recovering from traumatic brain injury. J Clin Endocrinol Metab, 2001; 86: 2752-2756.
9. Agha A, Thornton E, OI Kelly P, Tormey W, Phillips J, Thompson CJ. Posterior pituitary dysfunction after traumatic brain injury. J Clin Endocrinol Metab, 2004; 89: 5987-5992.
10. Doczi T, Tarjanyi J, Huszkae, Kiss J. Syndrome of inappropriate secretions of antidiuretic hormone (SIADH) after head injury. Neurosurgery, 1982; 10: 685-688.
11. Iglesias P, Gomez-Pan A, Diez JJ. Spontaneous recovery from post traumatic hypopituitarism. J Endocrinol Invest, 1996; 19: 320-323.
12. Benvenga S, Campenni A, Ruggeri RM, Trimarchi F. Hypopituitarism secondary to head trauma. J Clin Endocrinol Metab, 2000; 85; 1353-1361.
13. Saggese G, Ranke MB, Saenger P et al. Diagnosis and treatment of growth hormone deficiency in children and adolescents towards a consensus. Horm Res., 1998; 50: 320-340.
14. Cohen P, Rogol AD, Deal CL et al. Consensus statement on the diagnosis and treatment of children with idiopathic short stature: a summary of growth hormone research society. The Lawson with Wilkins Pediatric Endocrine Society and the European Society for Pediatric Endocrinology Workshop. J Clin Endocrinol Metab, 2008; 93; 4210-4217.
15. Al Jurayyan NAM. Isolated adrenocortitropin deficiency as a rare cause of hypoglycemia in children. Horm Res., 1995; 44: 238-240.