# Hypopituitarism Following a Traumatic Brain Injury

**Dugasa Hirpa***

*Editorial office, Journal of Steroids & Hormonal Science, Brussels, Belgium*

***Corresponding Author****

Dugasa Hirpa

Editorial office

Journal of Steroids & Hormonal Science, Brussels, Belgium

E-mail: steroids@journalsres.com

**Copyright:** ©Hirpa D. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

**Received:** 06-May-2022, Manuscript No. JSHS-22-17313; **Editor assigned:** 21-May-2022, **PreQC No.** JSHS-22-17313(PQ); **Reviewed:** 27-May-2022, **QC No.** JSHS-22-17313(Q); **Revised:** 29-May-2022, Manuscript No. JSHS-22-17313(R); **Published:** 31-May-2022, **DOI No.** 10.35248/ 2471-7536-22.13(3).237

## Abstract

The most common cause of acute brain injury is trauma. In the United States, 1.5–2.0 million Traumatic Brain Injuries (TBI) occur each year, with a cost in excess of $10 billion. TBI is the leading cause of death and disability among young adults under the age of 35. TBI can have serious consequences, such as impairments in motor function, speech, cognition, and psychosocial and emotional skills. Clinical studies have recently documented the occurrence of pituitary dysfunction following TBI and another cause of acute brain injury, Sub Arachnoid Haemorrhage (SAH). Following a moderate to severe TBI or SAH, these studies have consistently shown a 30–40% occurrence of pituitary dysfunction involving at least one anterior pituitary hormone. Growth hormone (GH) deficiency is the most common pituitary hormone disorder, affecting roughly 20% of patients when multiple GH deficiency tests are performed. Adrenal insufficiency is the most serious concern within 7–21 days of acute brain injury. Pituitary function may fluctuate during the first year following TBI, but it is well established by the end of the first year. The effects of hormone replacement therapy on motor function and cognition in TBI patients are currently being studied. Any patient suffering from a moderate to severe acute brain injury should be tested for pituitary dysfunction.

**Key Words:** Anterior pituitary hormone deficiency • Traumatic b rain injury • Screening

## Introduction

This review focuses on pituitary dysfunction as a result of traumatic brain injury. The initial focus of the research was pituitary dysfunction caused by Traumatic Brain Injury (TBI)[1-3]. Acute brain injury caused by Subarachnoid Haemorrhage (SAH) and the presence of pituitary dysfunction have also been studied in a number of recent studies. There are additional field reviews available.

This year, 1.5–2.0 million Americans will suffer a TBI; 50–70% of these injuries will be caused by motor vehicle accidents, bicycle accidents, or pedestrian-vehicle collisions [4]. TBI kills 52,000 people and hospitalises 500,000 people each year. It is the leading cause of death and disability among young adults under the age of 35, with an estimated annual cost of over $10 billion for acute care and rehabilitation. TBI is most common in the 15- to 24-year-old age group, which is prone to risky behaviour. Men are three times more likely than women to suffer a traumatic brain injury. TBI can have devastating consequences, incapacitating young, productive people and resulting in lifelong disability and reduced productivity. These findings were compiled into a consensus statement issued by the National Institutes of Health in 1998 [5].

TBI severity can be measured in a number of ways. Following an injury, the typical recovery sequence includes continuous memory (pre-injury storage), retrograde amnesia, coma, posttraumatic amnesia, and finally memory restoration. The length of coma and posttraumatic amnesia is directly proportional to the severity of the TBI. The Glasgow Coma Scale (GCS), which includes an assessment of the patient's level of consciousness and the duration of posttraumatic amnesia, is another measure of TBI severity. TBI is classified as mild, moderate or severe when the GCS score is 13–15, 9–12, or 5. Because determining coma can be influenced by a variety of factors, this scale is considered a rough estimate of TBI severity. Although initial studies of TBI and pituitary dysfunction focused on subjects who had sustained moderate to severe TBI, there have been no such studies assessing pituitary function in patients with mild traumatic brain injury and pituitary dysfunction.

## Materials and Methods

This was a cross-sectional study based on adherence to the hospital's standard prescription form at LIEC, Korle-Bu Teaching Hospital. Prescriptions submitted and dispensed at the pharmacy are routinely held. We looked at prescriptions that were handed in to the pharmacy unit between October 1, 2015, and March 31, 2016. At the unit, all prescriptions with at least one medicament served were scanned. Patients who took medications away that were neither available nor dispensed (due to patient inability to pay) were not captured. A copy of the prescription was obtained in a particular format using a pre inserted carbon. A copy of the prescription was stored at the pharmacy unit, along with a copy of the point-of-sale receipt, which lists the names and prices of the drugs purchased. Two employees were trained to extract information from prescription paperwork. The classifications of all the drugs on the prescriptions were examined by a specialised pharmacist and a pharmacy technologist. All prescriptions were examined for the type of anti-glaucoma medication prescribed, the percentage of mono therapy vs. combination therapy prescribed, the percentage of fixed dose combinations prescribed, the percentage of medicines prescribed with generic names, and cost analysis (monthly and yearly) for all anti-glaucoma drugs, including their dose, frequency, duration, and route of administration. Each parameter was given a percentage value. After receiving approval from the institutional ethics committee, this was a six-month prospective study. The study comprised 62 patients of either gender with a well-established diagnosis of glaucoma (as per SEAGIG Guidelines) [6]. Patient demographics, past medical and drug history, socioeconomic status, and systemic comorbidities were all collected using a specifically prepared proforma. Each patient's medical history, including previous ocular surgery and current medications, was recorded. All prescriptions were analysed in patients with known and newly diagnosed glaucoma for the names, doses, frequency, duration, and class of drug prescribed, as well as the percentage of mono therapy versus fixed drug combinations and cross over between classes for those who did not achieve the target intraocular pressure.

## Pituitary Dysfunction

Acute brain injury has only recently been recognised as a cause of pituitary dysfunction. Previously, only case reports linked pituitary dysfunction to brain injury. Benvenga et al. reviewed these case reports in 2000, raising the possibility of a link between TBI and pituitary dysfunction [7].

The pathophysiology of TBI-related pituitary dysfunction is unknown, but the anatomy of the pituitary and its supporting structures suggests a disruption in blood supply to the pituitary gland. The long hypophyseal portal veins branch from the carotid artery and pass through the diaphragm to supply 70–90% of the blood supply to the pituitary gland, primarily to the outer portions of the gland. The short hypophyseal portal veins beneath the diaphragma supply less than 30% of the blood supply, primarily to the pituitary gland's central regions. Autopsy results in subjects who survived 12 hours after TBI show that 35% have infarction involving approximately 70% of the anterior pituitary in the peripheral region, where blood supply is provided by the long hypophyseal portal veins.

### TBI and Pituitary Dysfunction Research

Kelly and colleagues conducted the first study linking TBI and pituitary dysfunction, assessing pituitary function in 22 adults at least three months after TBI [2]. Eight patients (36%) with GCS scores of 10 had an abnormal response in at least one anterior pituitary axis. GH deficiency (peak GH 65 lg/L) was documented in four patients (18%) using the GH stimulation test of insulin-induced hypoglycemia, and a borderline cortisol deficiency was observed in one patient. Five patients were found to be deficient in gonadotropins (four male, one female). One patient had thyroid axis deficiencies (4.5%).

Soon after, Lieberman and colleagues [1] published a study evaluating pituitary dysfunction in 70 adults (ages 18–58 years) admitted to a TBI rehabilitation facility. Seven (15%) of the 48 patients evaluated by glucagon stimulation testing were found to be GH-deficient (peak GH 3 lg/L), which was confirmed by L-dopa stimulation or an abnormal IGF-I level. Assessments of the anterior pituitary axes produced results similar to those of Kelly et al. [2], with the exception of a higher prevalence of low serum cortisol levels (46 percent) [1]. The cortisol axis was then assessed using a cosyntropin stimulation test, which did not confirm an increased risk of adrenal insufficiency. Subsequent studies using the GHRH + arginine test for GH deficiency found that GH deficiency was present in approximately 25% of moderate to severe TBI patients [4,5]. Furthermore, no correlation has been found between the severity of the TBI and the increased risk of GH deficiency [3,4]. The third and seventh decades of life have the highest prevalence of GH deficiency following TBI [4].

Agha and colleagues looked at pituitary function changes in 50 adults who had moderate to severe TBI (GCS 3–13) in the first 7–21 days after injury. While the prevalence of GH deficiency as determined by glucagon stimulation testing was comparable to previous studies, adrenal insufficiency was found to be the primary concern in the acute setting, affecting eight (16%) patients. GHD development was not related to age, GCS score, CT scan findings, or other anterior pituitary defects, according to multivariate analysis. Three studies examining pituitary dysfunction in adults following SAH found that the occurrences of GH deficiency and other anterior pituitary deficiencies are similar to those seen in the TBI studies. Aimaretti and colleagues found hypopituitarism in 38% of patients screened three months after injury in the largest of these trials (N = 100). The most common defect was severe GHD (25%) followed by secondary gonadal (12.5%), thyroid (7.5%), and adrenal (2.5%) deficiencies.

### Pituitary Dysfunction Screening

The recent study of Aimaretti and colleagues [7] clarified the question of when to screen for pituitary dysfunction following an acute brain injury. In a prospective 12-month study of adults with TBI (n = 70) or SAH (n = 32), they discovered that a diagnosis of panhypopituitarism made 3 months after injury did not change at a 12-month reassessment [7]. Adults with isolated anterior pituitary deficiency at 3 months, on the other hand, were not consistently deficient at 12 months. Adults who have suffered an acute brain injury should be evaluated for adrenal insufficiency within the first 21 days, at 3 months to determine if multiple hormone deficiencies are present (and to begin replacement therapy if necessary), and at 12 months to determine which deficiencies are permanent.

A thorough history and physical exam, as well as baseline serum levels of free T4, thyroid-stimulating hormone (TSH), Follicle-Stimulating Hormone (FSH), cortisol, prolactin, and Insulin-like Growth Factor-I (IGF-I), can all be used to screen for anterior pituitary dysfunction. No additional hormonal evaluation of gonadotropins is required in a menstruating female. A total testosterone level in men is also advised. Both men and women should have their prolactin levels checked. Free T4 and TSH levels will assess the thyroid axis, and a morning cortisol measurement will reveal significant adrenal insufficiency. Endocrinologists can perform cosyntropin and GH stimulation testing to determine cortisol or GH deficiency and initiate replacement therapy as needed. As previously stated, GH stimulation testing with the three standard stimulation tests yields roughly the same occurrence of GH deficiency in patients with acute brain injury. Because pituitary deficits may not be identified for several years after TBI, clinicians should monitor patients with a history of moderate to severe TBI for signs and symptoms of pituitary–hypothalamic impairment.

## Conclusion

Pituitary dysfunction should be evaluated in any patient suffering from moderate to severe acute brain injury. Mild brain injury may also result in pituitary dysfunction, but no studies have been conducted to determine the frequency of this occurrence.

## References

1. Lieberman SA, *et al.* Prevalence of neuroendocrine dysfunction in patients recovering from traumatic brain injury. J Clin Endocrinol Metab.2001;86(6):2752-2756.
2. Agha A, *et al.* Anterior pituitary dysfunction in survivors of traumatic brain injury. J. Clin. Endocrinol. Metab. 2004 Oct 1;89(10):4929-36.
3. Bondanelli M, *et al.* Occurrence of pituitary dysfunction following traumatic brain injury. J. neurotrauma. 2004 Jun 1;21(6):685-96.
4. Aimaretti G, *et al.* Traumatic brain injury and subarachnoid haemorrhage are conditions at high risk for hypopituitarism: screening study at 3 months after the brain injury. Clin. endocrinol. 2004 Sep;61(3):320-6.
5. Brain TR. Rehabilitation of persons with traumatic brain injury. Jama. 1999 Sep 8;282(10):974-83.
6. Benvenga S, *et al.* Hypopituitarism secondary to head trauma. The Journal of Clinical Endocrinology & Metabolism. 2000 Apr 1;85(4):1353-61.
7. Aimaretti G, *et al.* Residual pituitary function after brain injury-induced hypopituitarism: a prospective 12-month study. J. Clin. Endocrinol. Metab. 2005 Nov 1;90(11):6085-92.

**Cite this article:** Hirpa D. Hypopituitarism Following a Traumatic Brain Injury. J Steroids Horm Sci. 2022, 13 (3), 001-002.