World Journal of Physical and Rehabilitation Medicine

Letter to the Editor
Published: 26 Feb, 2018

Hypothalamic-Pituitary Dysfunction Post Acquired Brain Injury

*Moheb Gaid**

Department of Rehabilitation Medicine, Colman Centre for Specialist Rehabilitation Services, UK

Introduction

Acquired brain injury (ABI) as a result of trauma (TBI) and subarachnoid haemorrhages (SAH) are major health problems; with respective incidences of 235/100,000 and 6-10/100,000 [1]. ABI often results in significant physical and psychological consequences. Hypopituitarism following these has recently be shown to be a common problem (1), and the signs of symptoms of such are often difficult to distinguish from those of the brain injury itself [1].

Basic Physiology

The anterior pituitary gland produces several peptide hormones;

-Adrenocorticotropic hormone

-Thyroid stimulating hormone

-Luteinizing hormone, and follicle-stimulating hormone

-Prolactin and growth hormone.

The release of these hormones is controlled by both the hypothalamus, and via feedback from peripheral organs. The posterior pituitary gland is a storage organ for hypothalamic hormones (oxytocin and antidiuretic hormone) [1].

Current Literature

There are no current guidelines to the screening or management of hypothalamic-pituitary dysfunction post ABI. In the last decade, there has been more awareness of the problem as a complex syndrome that could mimic the ABI impairments but potentially reversible. A meta-analysis of 19 clinical studies identified 1137 patients with traumatic brain injury or subarachnoid haemorrhage [1]. The prevalence of endocrine dysfunction in traumatic brain injuries was 27.5% (15% to 68%), and in subarachnoid haemorrhage 47% (37.5% to 55%). Results in the early phase post injury were excluded to avoid the confounding factor of acute critical illness on neuroendocrine function, which is believed to be reversible.

In traumatic brain injury, luteinizing hormone and follicle-stimulating hormone deficiencies were the most common, followed by adrenocorticotrophic hormone then thyroid stimulating hormone. In subarachnoid haemorrhage, there were increased rates of overall hypopituitarism, growth hormone deficiency and adrenocorticotrophic hormone deficiency. Posterior hypopituitarism is reported in 2.8% at 12 months but this has less evidence. Hypopituitarism is more common in severe traumatic brain injury. It has also been found that the severity of subarachnoid haemorrhages does not alter the risk. The pathology behind the hypopituitarism is considered a combination of micro-haemorrhage, ischaemia and necrosis to both the pituitary gland and/ or hypothalamus [1,2].

Untreated hypopituitarism is associated with serious morbidity and premature mortality. Adrenocorticotrophic hormone deficiency can result in a life-threatening adrenal crisis. A study indicated that following traumatic brain injury, hypopituitarism is associated with poor quality of life (factors included energy, sleep and mobility), abnormal body composition and adverse metabolic profile. Its conclusion was that hypopituitarism after both traumatic brain injury and subarachnoid haemorrhage is associated with poor recovery and a worse outcome.

Patients who are identified with hypopituitarism require hormone replacement, which in general reverses the clinical signs and symptoms. Borderline results should be followed up and repeated [1].

Recent audit at Level-I highly specialist rehabilitation unit in the UK and survey of other

OPEN ACCESS

Correspondence:
Moheb Gaid, Department of Rehabilitation Medicine, Colman Centre for Specialist Rehabilitation Services, Norwich, England NR2 2PH, UK, E-mail: moheb3mn@doctors.org.uk

Received Date: *19 Jan 2018*
Accepted Date: *20 Feb 2018*
Published Date: *26 Feb 2018*

Citation:
Gaid M. Hypothalamic-Pituitary Dysfunction Post Acquired Brain Injury. World J Phys Rehabil Med. 2018; 2(1): 1005.

specialist rehabilitation unit demonstrated that this condition is under investigated with potentially correctable symptoms.

In a local study done in 2014 then repeated in 2016; half of the patients admitted to our unit (level-I Neuro-Rehabilitation unit in England) with severe ABI were found to have abnormal pituitary functions at 3-6 months interval from the onset of their ABI (54.6% in 2014 and 55.6% in 2016) nearly one third of those remained to have abnormal pituitary functions at 12 months (36.4% in 2014 and 35% in 2016) the incident was higher in traumatic brain injuries (63.6%in 2014 and 65% in 2016).

Recommendations

Due to the high prevalence and impact of hypothalamic-pituitary dysfunction on this vulnerable cohort of patients; screening is an important aspect of detecting and potentially managing this condition. There has been a high incidence of abnormal pituitary functions in the first 3 months post ABI [2] these are likely reversible. It is recommended that the optimal screening time is between 3-6 months post injury and subsequent screening if clinically indicated. We also recommend referral to experienced endocrinology team to assess if these patients would benefit from hormone replacement therapy.

References

1. Schneider HJ, Kreitschmann-Andermahr I, Ghigo E, Stalla GK, Agha A. Hypothalamopituitary dysfunction following traumatic brain injury and aneurysm subarachnoid haemorrhage: A systematic review. JAMA. 2007;298 (12):1429-38.

2. Schneider HJ, Schneider M, Kreitschmann-Andermahr I, Tuschy U, Wallaschofski H, Fleck S, et al, Structured Assessment of Hypopituitarism after Traumatic Brain Injury and Aneurysmal Subarachnoid Hemorrhage in 1242 Patients: The German Interdisciplinary Database. J Neurotrauma. 2011;28(9):1693-8.