VOLUME - 11, ISSUE - 04, APRIL - 2022 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Endocrinology

A DELAYED PRESENTATION OF POSTERIOR HYPOPITUITARISM FOLLOWING TRAUMATIC BRAIN INJURY – A CASE REPORT OF A 24 YEAR OLD ASIAN FEMALE.

Dr Akshay Balaji Junior Resident-Sri Ramakrishna Hospital.

Dr Suresh Damodharan Consultant Endocrinologist-Sri Ramakrishna Hospital.

Dr Ganesh Prasad Consultant Urologist-Sri Ramakrishna Hospital.

ABSTRACT

Background: Cases of post traumatic hypopituitarism are rarely contemplated in the Indian subcontinent. However global literatures disclose that the incidence rate of hypopituitarism following traumatic brain injury is around 15-30%. Most hospitals do not routinely screen patients for pituitary dysfunction following traumatic brain injury due to the lack of proper protocol supporting the same. Hence large number of cases with varying pituitary abnormalities is procrastinated, which could have been otherwise benefited from early interventions. This case is a classic example of delayed exhibition of posterior hypopituitarism post traumatic brain injury, which has significantly compromised the quality of life of the patient in later stage due to the failure of timely screening.

Case Presentation: This is a case of 24 year old female who sustained a traumatic brain injury 3 years ago allegedly due to domestic abuse. The patient presented to the urology department with the complaints of polyuria, polydipsia, secondary amenorrhea, sleep disturbance and weight gain for 6-7 months. The patient was not under regular follow-up for the traumatic injury. On further evaluation, all the anterior pituitary hormones were found to be normal. We further investigated the case with biochemical investigations and an MRI of brain (plain and contrast) which showed a thickened enhancing pituitary stalk with absence of posterior pituitary bright spot with a normal anterior pituitary gland – suggestive of Central Diabetes Insipidus. Upon initiation with Desmopressin, the patient showed significant symptomatic improvement.

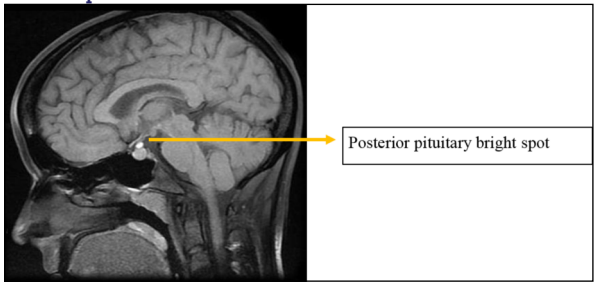
Conclusion: One of the most common complications of traumatic brain injury is hypopituitarism. Early screening for hypopituitarism in such cases will help us intervene early and improve the quality of life of the patients.

KEYWORDS : Hypopituitarism, traumatic brain injury, pituitary injury, posterior pituitary dysfunction.

INTRODUCTION:

Traumatic brain injury (TBI) often leads to permanent or temporary impairment of cognitive, physical, and psychosocial functions which occurs due to non-congenital insult to the brain from an external mechanical force [1].Global incidence rate of TBI in 2010 is around 200 cases per 100,000 people per annum [2]. TBI often manifests as neuroendocrine dysfunction, which affects the quality of life of patient and consists of both anterior and posterior pituitary insufficiency; water and electrolyte abnormalities (DI and SIADH) that are amongst the most challenging consequences [3]. The signs and symptoms of hypopituitarism may be often subtle and overlap with the neurological and psychiatric sequelae, which was then misapprehended leading to life threatening consequences occasionally.The aim of the case study is to emphasize the importance of early screening of pituitary hormones following traumatic brain injury which will be extremely useful to prevent further devastation and provide timely rehabilitation to improve the quality of life of the patient.

Case Report:



MRI Of Normal Brain With Posterior Pituitary Bright Spot

We identified a female patient of age 24 years who was admitted with a known case of traumatic brain injury 4 years ago, with complaints of polyuria, polydipsia and secondary amenorrhea for 6-7 months. The patient had a family history of diabetes mellitus and weight gain of 20 kg for the past 1 year. The fluid intake and urine output was found to be 3100ml/day and 6600ml/day respectively. The MRI of the brain revealed absent posterior pituitary bright spot-CDI as shown below in figure 1.

Figure 1: MRI Of The Patient Showing Absence Of Posterior Pituitary Bright Spot.

The ultrasound of abdomen was normal. The pre-void volume was found to be 760ml and post void volume was found to be 60ml. The biochemical investigations were examined as shown in the table 1:

Table 1: Biochemical Investigations Results Of The Patient

S. No	Biochemical parameters	Observed values	Reference values
1.	Haemoglobin	11.9g/dl	12.5-16 g/dl
2.	RBC	4.52 million cells/mcl	3.8-4.8 millioncells/mcl
3.	WBC	11.4 cells/mcl	4-10 cells/mcl
4.	HCT	40.6%	36-46%
5.	MCV	89.8 fL	78-100fL
6.	MCH	26.3 pg	27-32 pg
7.	MCHC	29.3 g/dl	31.5-34.5g/dl
8.	Platelets	307 cells/mcl	150-410 cells/mcl
9.	Polymorphs	62.3%	40-80%
10.	Lymphocytes	27.9%	20-40%
11.	Monocytes	6.2%	2-10%
12.	Eosinophils	3.1%	1-6%
13.	Basophils	3.1%	0-1%

14.	ESR	70 mm/Hr	3-20mm/Hr
15.	Urine osmolality	65 mOsm/ kg H_2O	500-850 mOsm/kg H_2O
16.	Serum osmolality	291 mOsm/kg	285-295 mOsm/kg
17.	Blood urea	15 mg/dl	17-49 mg/dl
18.	Creatinine	0.6 mg/dl	0.5-1.4 mg/dl
19.	Prolactin	16.4 ng/ml	3-18.6ng/ml
20.	Serum Cortisol	6 mcg/dl	4.46-22.7mcg/dl
21.	TSH	2.19 mIU/l	0.4-4 mIU/l
22.	LH	10.3 mIU/ml	2-9 mIU/ml
23.	FSH	6.38 mIU/ml	1.3-9.58mIU/ml
24.	Free T4	1.09 ng/dl	0.9-2.3 ng/dl
25.	Albumin	4.1g/dl	3.5-5 g/dl

The patient showed normal vital signs and stable cardiac and respiratory status. Based on the initial symptoms, the patient was diagnosed to have Central Diabetes Insipidus (CDI). The patient received following medications throughout the stay in the hospital as shown in table 2.

Table 2:

S. No	Medications	Dose	Frequency
1.	T. Mirabegron	25 mg, PO	1-0-1
2.	T. Desmopressin	0.1 mg, PO	1-0-1
3.	T. TolterodineTatarate	2 mg, PO	0-0-1
4.	T.Alprazolam	0.25 mg, PO	0-0-1
5.	T.Zinc	1 tab	0-0-1
6.	T.Vitamin C	1 tab	0-0-1

The patient responded very well upon treatment with oral Desmopressin. Following treatment, the patient showed a urine output around 2 litres.The patient was advised to follow-up after 7 days. During discharge, the patient was discharged with following medications as shown table 3:

Table 3:

S. No	Medications	Dose	Frequency
1.	Tab. Medroxyprogesterone	10 mg	BD 3 days
2.	Clindamycin-Clotrimazole vaginal pessary	-	5 days
3.	Tab. Desmopressin	0.1 mg	BD 7 days
4.	Multivitamin supplements	-	7 days

DISCUSSION AND CONCLUSION:

This is a classic case of a delayed presentation of hypopituitarism following traumatic brain injury. The prevalence of hypopituitarism following TBI is 50 in 1, 00,000 patients. Since most cases of hypopituitarism following traumatic brain injury leads to a lot of cases like Acromicria, Dwarfism, Simmond's disease, Diabetes Insipidus, dystrophiaadiposogenitalis which go unnoticed for prolonged period leads to severe impairment of quality of life of the patients. Our case highlights a rare and delayed presentation of posterior hypopituitarism with normal anterior pituitary functioning.

There is no data on delayed presentation of posterior hypopituitarism following traumatic brain injury. However there are a few studies of delayed presentation which highlights anterior hypopituitarism [4].Our patient had late onset symptoms suggestive of posterior hypopituitarism like polyuria, polydipsia, weight gain, secondary amenorrhea and sleep disturbance. Delayed presentation of hypopituitarism usually results in anterior pituitary dysfunction. However our case is a presentation of delayed posterior hypopituitarism which is very rarely reported. So this case throws a major highlight on the importance of early screening for anterior/posterior pituitary hormones which will help pick-up such cases and early intervention will lead to significant improvement in the quality of life of patients.

Abbreviations:

TBI - Traumatic brain injury
PTHP - Post traumatic hypopituitarism
CDI - Central Diabetes Insipidus
SIADH - Syndrome of inappropriate antidiuretic hormone secretion
QoL - Quality of life

REFERENCES:

1. Syed AT, Lone NA, Wani MA, Bhat AS. Clinical management of patients with minor head injuries. Int J Health Sci (Qassim). 2007 Jan;1(1):131-40. PMID: 21475463; PMCID: PMC3068669.
2. Bryan-Hancock C, Harrison J The global burden of traumatic brain injury: preliminary results from the Global Burden of Disease Project*Injury Prevention* 2010;**16:**A17
3. Capatina C, Paluzzi A, Mitchell R, Karavitaki N. Diabetes Insipidus after Traumatic Brain Injury. J Clin Med. 2015 Jul 13;4(7):1448-62. doi: 10.3390/jcm4071448. PMID: 26239685; PMCID: PMC4519799.
4. Kolli S, Mallipedhi A, Hughes T, Evans P. Delayed diagnosis of hypopituitarism following severe traumatic brain injury. BMJ Case Rep. 2010 Sep 29;2010:bcr1220092526. doi: 10.1136/bcr.12.2009.2526. PMID: 22778379; PMCID: PMC3028089.