ISSN No:-2456-2165

A Diagnostic Challenge, an Unique Case of a Psychiatric Disorder Presenting with Altered Sensorium

Dr. Dnyanesh Morkar, Dr. Akashdeep Singh*, Dr. Priyanka Patil, Dr. Hima Morkar
Department of General Medicine
KLE Academy Of Higher Education & Research, Belagavi, India

Abstract:- Severe hyponatremia, defined as serum sodium <120 mEq/L, is a life-threatening condition that usually results in neurological complications including altered sensorium, seizure episodes and ranges till death. Patients presenting with recurrent episodes of hyponatremia along with hypoglycemia should be evaluated with appropriate investigations to evaluate causes of hyponatremia and hypoglycemia apart from just correcting sodium and glucose value . This might lead to a diagnosis of occult disease.

Keywords :- *Empty Sella Syndrome, Hypotension, Hypoglycemia, Panhypopituitarism , Abortion, Sheehan's Syndrome .*

I. INTRODUCTION

When CSF fluid fills the sella Turcica, pituitary tissue is compressed until it lines the floor of the sella as well as its walls, resulting in an empty sella. There are two types of empty sella: primary and secondary. It's called When CSF enters the sella through a rent in the sellar diaphragm, it can cause a primary empty sella. This can happen with or without an increase in intracranial pressure. Secondary empty sella is brought on by pituitary injuries (pituitary apoplexy, for example) or the aftereffects of radiation or surgery. Most often observed in elderly, obese, hypertensive, and multiparous women, empty sella can go unnoticed. Clinical symptoms and endocrinopathies such as hypogonadotropism , growth hormone insufficiency or multiple pituitary hormone deficits are linked to empty sella in children. Research has demonstrated that empty sella syndrome.

II. CASE DESCRIPTION

48 year old female brought to ER with complaints of altered sensorium, irritability & excessive sleepiness since past 2 days.

Patient was alright 2 days back after which her attenders noticed a change in her sensorium in the form of excessive sleeping, drowsiness and inability to carry out her daily routine activities. She was taken to a local hospital and routine investigations were done which revealed that her serum sodium was 113 meq. An MRI was done which didn't show any evident abnormalities.

- PAST HISTORY: She is hypertensive patient on Tab Telmisartan-40 mg. She has also been prescribed Tab Nortriptyline -25mg, Tab Clonazepam- 0.25mg and Escitalopram- 10mg for depression.
- OBSTETRIC SCORE : P5L0A4D1 last delivery was 20 years back.
- ON EXAMINATION:
  PR-102 BPM
  BP-90/60 MMHG
  SP02-98%IN ROOM AIR
- SYSTEMIC EXAMINATION: Central nervous system - Patient was drowsy, she could be aroused by painful stimulus

B/L pupils were equal and reactive to light and B/L plantar reflexes were mute.

Cardiovascular system - S1 and S2 heart sound heart

Respiratory system- Bilateral air entry present

Per Abdomen - Soft and Non tender abdomen

Random blood sugar was 40 mg/dl.

She was started on Dextrose infusion to correct hypoglycaemia and fluid resuscitation was initiated to correct her low blood pressure.

Routine investigations (Table I) were evaluated to find the cause for altered sensorium.

ISSN No:-2456-2165

Table 1: Routine Investigations

PARAMETERS	VALUES	REFERNCE RANGE
Hemoglobin	12.3	12-15g/dl
HCT	36%	36-46
WBC	$8.37*10^3$/ul	4-10
Platelet	$141*10^3$/ul	150-450
T.Bilirubin	1.5 mg/dl	0-1.1
D. Bilirubin	0.8mg/dl	0-0.4
T. Proteins	5.6gm/dl	6.4-8.3
AST	117 u/l	0-31
ALT	23 u/l	0-34
Creatinine	0.8 mg/dl	0.5-1.2
Sodium	115 meq/l	136-145
Potassium	4.5 meq/l	3.5-5.5
Chloride	106meq/l	
Urine osmolality	390 mosmlkg	500-800
Serum osmolality	237 mosmlkg	280-310
Urine spot	151 meq/l	>20 meq/L

The differential diagnosis being considered were:

- Drug overdose
- Metabolic Encephalopathy
- APLA
- Sheehan's syndrome

An initial diagnosis of multiple drug overdose vs metabolic encephalopathy was made. The patient's attenders confirmed that there was no drug intake in the last 4 days which narrowed down our diagnosis pool.

Metabolic encephalopathy was considered the working diagnosis. SIADH was ruled out and sodium correction and glucose correction was initiated.

Persistent hyponatremia and hypoglycemia was seen even after correction.Serum insulin was tested and found to be normal. In view of recurrent hypoglycemia, hypotension and hyponatremia, further investigations done were serum TSH , serum 8am and 4 pm cortisol. Serum 8am and 4 pm cortisol along with serum TSH were low which further prompted testing of ACTH which also turned out to be low.(TABLE-II)

Table 2: Investigations

PARAMETERS	VALUE	REFERENCE RANGE
SR INSULIN		
SR CORTISOL (8AM)	0.988	
SR CORTISOL(4PM)	0.80	
SR PROLACTIN	1.15ng/ml	4.8-23.30
SR TSH	0.1mcl/ml	0.27-4.2
FREE T3	1.17pg/dl	2.0-4.40
FREE T4	0.28pg/dl	0.93-1.70
LH	0.21MIU/ml	
FSH	0.4MIU/ml	
SR ACTH	2.0pg/ml	7.2-63

Running Diagnosis of secondary adrenal insufficiency was made and the patient was started on Inj Hydrocortisone 100 mg TID. Patient improved symptomatically and sodium and glucose levels returned to normal levels.

Further workup was done to find the cause of secondary adrenal insufficiency. Serum prolactin, FSH , LH and growth hormone were tested and were all found to be significantly below normal ranges.

A summative diagnosis of pan-hypo-pituitarism was established.

The patient had a past obstetric history of five abortions and her last baby died at 6 months of age 20 years back. Post delivery patient also had a history of multiple blood transfusions.

Antiphospholipid antibody test was done to rule out APLA in view of the recurrent progressive abortions but it was found to be negative.

There was a history of failure to lactation in the last pregnancy.

Under the guise of a psychiatric disorder with some of the most classical symptoms of Sheehan's syndrome, a range of investigations were done and after ruling out several differentials a diagnosis was made. A diagnosis of pan-hypo-pituitarism secondary to Sheehan's syndrome was made.

An MRI Brain was repeated in our hospital which showed an empty sella turcica suggesting empty sella syndrome.

Final diagnosis of empty sella syndrome secondary to Sheehan's syndrome was made.

Patient was bed ridden for a long time leading to bed sore for which supportive treatment and physiotherepy was done.

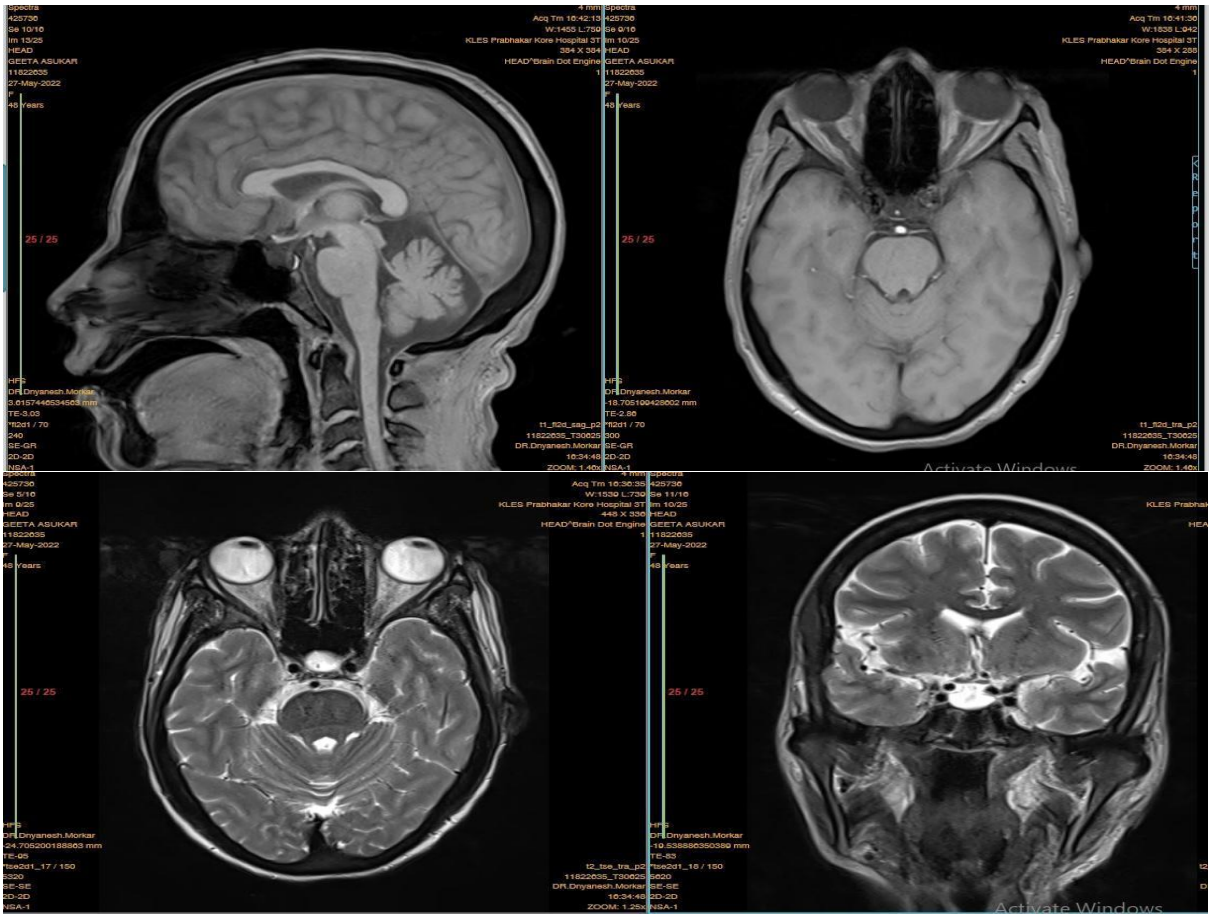

Fig. 1: MRI Scan - (Empty Sella Turcica)

III. DISCUSSION

Sheehan's syndrome (SS) is brought on by severe postpartum hemorrhage-induced ischemic pituitary necrosis. Other potential causes of this condition include thrombosis, vasospasm, and vascular constriction of the hypophyseal arteries. The pathophysiology of Sheehan's syndrome includes an enlarged pituitary gland, small sellar size, disseminated intravascular coagulation, and autoimmune. Out of one million births, five persons have Sheehan syndrome. The symptoms of empty sella syndrome include hypopituitarism, which manifests as hypopigmentation, irregular menstruation, loss of genital hair, premature aging, and inability to nurse after delivery. In rare cases, circulatory collapse, psychosis, hypoglycemia, and hyponatremia may be observed.

The most frequent cause of hypoperfusion-induced adenohypophyseal ischemic necrosis is adenohypophyseal insufficiency. Sheehan originally described it in 1939. There are other, delayed, and subtle indications of adenohypophyseal insufficiency that can go undiagnosed. For patients with Sheehan syndrome, the average diagnosis delay is 20 years. When diagnosed, 53% of patients exhibited nonspecific complaints, 31% complained of adrenal insufficiency, and 10% complained of hypogonadism. Of the patients, 45% had partial hypopituitarism and 55% had panhypopituitarism at the time of diagnosis. The causes of hypopituitarism are multifactorial. In addition to adenohypophyseal necrosis, immunological, iatrogenic, traumatic, infectious, and tumoral factors can also be responsible.

The preferred inquiry is an MRI scan. There are instances of acute postpartum hypophyseal necrosis. Early MRI in hypophyseal necrosis shows a smaller pituitary gland with hypersignal segments in T1 and T2 and hyposignal regions without contrast. After pituitary atrophy, an empty sella turcica is later seen on an MRI.

The management of Sheehan's syndrome is consistent with the therapy of hypopituitarism. The main objective is to replace missing hormones. It not only aids in the correction of endocrine problems but also lowers hypopituitarism-related mortality. Individuals who have hypocortisolism and secondary hypothyroidism should receive glucocorticoid replacement prior to thyroid hormone therapy. Hormone replacement therapy is the recommended course of treatment for hypogonadism and gonadotropin insufficiency. Desmopressin (DDAVP) is the preferred treatment for patients with diabetic insipidus (DI). Patients with GH deficiency should think about getting GH replacement therapy. The recommended initial dosage for GH is 0.1–0.3 mg/d, with a monthly titration up to 0.1 mg/d.

IV. CONCLUSION

This case proves a diagnostic challenge as hypopituitarism is rarely considered in clinical practice when a patient present with hyponatremia and hypoglycemia.This case report highlight the importance of considering Sheehan's syndrome as one of the differential diagnosis in cases of multiparous female patients presenting with adrenal insufficiency.

REFERENCES

[1]. Rondon-Berrios H. Therapeutic Relowering of Plasma Sodium after Overly Rapid Correction of Hyponatremia. What Is the Evidence? 2020;15(2):282–4.
[2]. Beck-Peccoz P, Rodari G, Giavoli C, Lania A. Central hypothyroidism - a neglected thyroid disorder. Nat Rev Endocrinol. 2017;13(10):588–98.
[3]. Auer MK, Stieg MR, Crispin A, Sievers C, Stalla GK, Kopczak A. Primary Empty Sella Syndrome and the Prevalence of Hormonal Dysregulation. Dtsch Arztebl Int. 2018;115(7):99–105.
[4]. Chiloiro S, Giampietro A, Bianchi A, Tartaglione T, Capobianco A, Anile C, et al. DIAGNOSIS OF ENDOCRINE DISEASE: Primary empty sella: a comprehensive review. Eur J Endocrinol. 2017;177(6):R275–85.