



Case Report

A delayed diagnosis of Sheehan syndrome, presenting as chronic hypopituitarism

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Abstract

Background: Sheehan syndrome is a rare but important cause of maternal hypopituitarism resulting from ischemic necrosis of the pituitary gland following severe postpartum hemorrhage. The diagnosis is often delayed due to non-specific clinical manifestations that may appear years after the inciting obstetric event. We report the instance of a middle-aged women presenting with features of chronic hypopituitarism, ultimately diagnosed as Sheehan syndrome. This report highlights the importance of detailed obstetric history and early endocrine evaluation in women presenting with unexplained multi system symptoms.

Key words: Sheehan syndrome; Hypopituitarism; Hypothyroidism; Amenorrhea; Post partum hemorrhage.

Citation: Priya, Maria, P.D Aravindhan, Raghunath, Gayathiri. A delayed diagnosis of Sheehan syndrome, presenting as chronic hypopituitarism. *Kauverian Med J.* 2026;3(6):71-76.

Academic Editor: Dr. Venkita S. Suresh

ISSN: 2584-1572 (Online)



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1. Introduction

Sheehan syndrome is characterized by partial or complete anterior pituitary insufficiency following severe postpartum hemorrhage. Although its incidence has declined in developed countries, it remains prevalent in developing regions due to inadequate obstetric care. The prevalence of Sheehan syndrome was estimated to be 5 in 100000 females with prevalence as high as 3.1% reported in India. The clinical presentation ranges from acute postmortem complications to insidious, delayed manifestations occurring years later, often leading to misdiagnosis. The previous studies have discovered a range of 1- 33 years as the typical time between postpartum hemorrhage and the onset of symptoms. This case report describes a patient with delayed presentation of Sheehan syndrome and emphasizes the need for clinical suspicion in appropriate settings.

2. Case presentation

A 48-years aged woman G2 P2 L2 with no known comorbidities presented to the General Medicine outpatient department with complaints of chronic fatigue, dizziness, constipation, facial puffiness, bilateral lower limb swelling.

Clinical examination revealed delayed tendon reflex (hung-up reflex/Woltman sign) hence patient was suspected of having Hypothyroidism and advised thyroid function tests which revealed secondary hypothyroidism. TSH-2.67mIu/l, T3-0.367mcg/dl, T4-0.659ng/ml.



Further detailed history revealed that the patient had undergone home delivery 25 years ago which was complicated by severe postpartum hemorrhage, followed by which the patient became unconscious for about three hours and then regained consciousness thereafter. The patient reported failure of lactation following the delivery and did not resume her menstruation thereafter. No prior endocrine evaluation had been performed.

3. On physical examination

- The patient appeared pale with dry and coarse skin, facial puffiness and bilateral pitting pedal edema.
- Pulse rate: 62 per minute.
- Blood pressure: 90/60mmHg, with postural drop.
- Woltman sign positive- prolonged return of the muscle to its resting state after a deep tendon reflex is elicited (ankle jerk).
- Secondary sexual characters were poorly developed, with sparse axillary and pubic hair.
- Systemic examination was otherwise unremarkable.

4. Investigations

Investigation	Results	Ref Range
Hemoglobin (g/dl)	9.9	12-15
Total Count (/mm ³)	5010	4000-10000
Platelet count (/mm ³)	140000	140000-400000
Na ⁺ (mmol/L)	110	136-145
K ⁺ (mmol/L)	3.1	3.5-5.0
serum Osmolality (mOsm /kg water)	223.5	275-295
Urine Osmolality (mOsm /kg water)	112.8	300-900
Urine Spot Sodium (mEq /L)	43	80-180
T3 (mcg/dl)	0.367	0.82-1.58
T4 (mcg/dl)	0.659	5.1-14.1
TSH	1.613	0.5-5.0
Prolactin (ng/mL)	3.9	5.0-35
FSH (mIU/mL)	2.34	1.3-9.58
LH (mIU/mL)	0.412	0.8-15.5
ACTH (pg/mL)	<1.50	3.6 - 60.05
Cortisol (mcg/dl)	1.25	4.46-22.7
GH (ng/mL)	0.007	2-5

Na⁺ - sodium ion, k⁺ - potassium ion, TSH- Thyroid stimulating hormone, T3- Triiodothyronine, T4- thyroxine, FSH- Follicular stimulating hormone, LH- lutenizing hormone.

MRI



5. Report

- Empty Sella, with non-visualization of the posterior pituitary bright spot.
- No evidence of pituitary adenoma, hypophysis or other seller/suprasellar pathology.
- No imaging signs of intracranial hypertension.
- Otherwise, normal MRI brain study.

6. Diagnosis

Based on the history of severe postpartum hemorrhage, failure of lactation, secondary amenorrhea, biochemical evidence of pan hypopituitarism and MRI findings diagnosis of Sheehan syndrome was made.

7. Management

The patient was initiated on hormone replacement therapy in a step wise manner:

- Acute phase: correction of hypoglycemia and hyponatremia.
- Oral hydrocortisone 40 MG per day in divided doses.
- Levothyroxine 75 mcg/day (started after Cortisol replacement).

She was counseled regarding lifelong hormone replacement, stress dose steroid requirements and the importance of regular follow up.

8. Outcome and follow up

The patient was discharged with the above medications and advised for follow up in about two weeks.

9. Discussion

Sheehan syndrome pathogenesis involves the unique physiological vulnerability of the pituitary gland during pregnancy. During pregnancy, there is estrogen mediated Lactotroph hyperplasia which causes marked pituitary enlargement, without proportional increase in blood supply. The anterior pituitary, supplied by a low-pressure portal venous system, becomes highly susceptible to ischemia. Posterior pituitary function is usually preserved due to its direct arterial blood supply. Severe postpartum hemorrhage causes hypovolemia, hypotension and reduced pituitary perfusion leading to ischemic infarction of anterior pituitary. Ischemia causes coagulative necrosis of pituitary tissue followed by gradual resorption of necrotic tissue which is then replaced by fibrous tissue. Over time this leads to marked pituitary atrophy. Loss of pituitary volume allows herniation of the subarachnoid space into the sella turcica, resulting in a secondary empty sella, with resultant hypopituitarism. Small sella turcica size, vasospasm, thrombosis and coagulation disorders may predispose to ischemia. The necrosis of pituitary gland and sequestered antigens can trigger autoimmune response worsening hypopituitarism over the years. This leads to hormonal consequences of which typical hormones and their sequence of involvement are as follows - Growth hormone—> Gonadotropins (LH, FSH)—> ACTH—> TSH.

Clinical features of hypopituitarism and its association with destructive lesion in pituitary were originally described by Simmonds in 1914 [1]. Hutchinson observed the occurrence of syndrome in early pregnancy with postmortem finding of pituitary necrosis in a woman who died at 10th week of pregnancy following septic endometritis and pulmonary embolism [2]. In Sheehan syndrome, necrotic damage to anterior pituitary causes partial or complete loss of thyroid, adrenocorticoid and gonadal functions. The diagnosis can be made reliably in the presence of lactation failure, prolonged amenorrhea and hypoglycemic crisis. However other signs of pituitary insufficiency are often delayed and subtle because of which the diagnosis is being missed. Inability to lactate after delivery due to prolactin deficiency and the development of amenorrhea from gonadotropin deficiency occurs classically in Sheehan syndrome but the clinical presentation ranges variably. Delayed presentation with non-specific symptoms like fatigue, depression, weight changes and failure of lactation are common.

Failure of postpartum menstruation due to FSH, LH deficiency is quite common, but spontaneous pregnancies have been reported [3].

Furthermore, this syndrome can also be associated with anxiety and depression. Acute illnesses or stress can unmask Sheehan's syndrome by precipitating adrenal crisis, revealing their underlying pituitary damage [4]. The diagnosis of Sheehan syndrome is based on features of hormone deficiency, obstetric history with decreased level of basal hormones (T3, T4, TSH, FSH, LH, GH, Prolactin, Cortisol and Insulin are like growth factors). In India 90% patients presented with lactation failure. Anemia, hypotension, hypogonadism, hypothyroidism and altered lipid profile were the most common findings.

The mean systolic blood pressure (BP) was 80.95mmHg and diastolic BP was 51.6 mmHg at the time of presentation. Hyponatremia was the most common electrolyte abnormality noted, and low HDL was the commonest lipid abnormality [11]. Differential diagnosis includes pituitary adenoma and lymphocytic hypophysitis, craniopharyngioma. [5,6].

In our case, in addition to positive obstetric history for postpartum hemorrhage, the patient presented clinical signs of thyroid and Cortisol deficits and reported frequent hospital visits in view of generalized weakness and depressive illness, hypoglycemia. Common initial clues include failure of lactation and amenorrhea following delivery, which may be overlooked. MRI findings of empty Sella support the diagnosis in chronic cases [7,8]. Early recognition and appropriate hormone replacement can significantly avert potentially fatal crises [9].

10. Conclusion

Sheehan syndrome should be considered in women presenting with features of hypopituitarism and history of postpartum hemorrhage even many years after childbirth. A thorough obstetric history and targeted hormonal evaluation are key to diagnosis. Timely hormone replacement therapy is life saving and improves long term outcomes.

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