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Case Report

Sheehan's syndrome with delayed diagnosis

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ABSTRACT

Sheehan's syndrome is a rare but important cause of hypopituitarism, typically resulting from ischemic necrosis of the anterior pituitary gland following severe postpartum hemorrhage. Its presentation may be acute or delayed, often leading to diagnostic challenges, particularly in regions with limited obstetric facilities. This case reports the case of a 57-year-old woman who presented with chronic fatigue, dizziness, and hypotension. She had a history of severe postpartum hemorrhage decades earlier, followed by failure of lactation and amenorrhea. Evaluation revealed secondary hypothyroidism and adrenal insufficiency. Magnetic resonance imaging (MRI) brain demonstrated an atrophied pituitary with a thin rim of residual tissue. She was diagnosed with Sheehan's syndrome and treated with hydrocortisone replacement, followed by levothyroxine titrated according to free T4 levels. At six-month follow-up, she was asymptomatic with complete resolution of clinical features. This case highlights the importance of considering Sheehan's syndrome in women with unexplained hypothyroidism, hypotension, or amenorrhea with a history of postpartum hemorrhage. Cortisol replacement must always precede thyroid hormone therapy to avoid adrenal crisis. Early recognition and appropriate hormone replacement therapy ensure favorable long-term outcomes.

Keywords: Sheehan's syndrome, Hypopituitarism, Adrenal insufficiency, Secondary hypothyroidism, Postpartum hemorrhage

INTRODUCTION

Harold Leeming Sheehan (1900–1988), a British pathologist, first described postpartum necrosis of the anterior pituitary in 1937, based on autopsies of twelve postpartum deaths with significant association to postpartum hemorrhage (PPH).[1]

Sheehan's syndrome is rare, with an estimated prevalence of <1 in 200,000. Its incidence has declined in developed countries due to improved obstetric care but remains relevant in developing regions with inadequate facilities.[2]

During pregnancy, the pituitary gland enlarges considerably, making it vulnerable to ischemia in the setting of severe hypotension due to hemorrhage or shock. This may result in acute or delayed hypopituitarism (Figure 1). Risk factors include small sella turcica, hypercoagulable states, and genetic predisposition. Ischemic injury may also trigger autoimmunity, further damaging the gland. The posterior pituitary usually remains intact due to its rich blood supply, though rarely diabetes insipidus may occur.[3]

Symptoms depend on the extent of pituitary necrosis. About 70–80% of the gland must be destroyed before symptoms manifest. Prolactin deficiency causes failure of lactation; gonadotropin deficiency leads to amenorrhea, reduced libido, loss of axillary/pubic hair, and vaginal atrophy. Thyroid stimulating hormone (TSH) deficiency results in hypothyroidism with fatigue, constipation, weight gain, and cognitive slowing. Adrenocorticotropic hormone (ACTH) deficiency may cause chronic hypotension, fainting, and poor stress response. Growth

hormone (GH) deficiency produces subtle features such as reduced strength and altered body composition. Rare antidiuretic hormone (ADH) deficiency results in diabetes insipidus.[4]

Figure 1: Pathogenesis of Sheehan's syndrome.

Treatment consists of hormone replacement tailored to deficiencies: glucocorticoids, levothyroxine guided by FT4 levels, sex steroids, and occasionally GH or desmopressin. Prognosis is good with early recognition, but delayed diagnosis can be fatal.

CASE REPORT

A 57-year-old woman presented on 30 April 2024 with dizziness, fatigue, and lethargy. She was hypotensive on examination but alert, comfortable, and not tachycardic.

She reported longstanding fatigue, worsened after hospitalization six weeks earlier for left lower lobe pneumonia requiring ICU care, inotropes, and intravenous steroids. She had a known history of hypothyroidism, treated with 25 mcg thyroxine daily. She appeared hypothyroid with pale, finely wrinkled skin (Figures 2 and 3), suggestive of secondary hypothyroidism.

Further probing revealed severe postpartum hemorrhage (PPH) during her last childbirth decades ago, requiring intensive care unit (ICU) care and transfusions. She had never lactated and had permanent amenorrhea. A scar from venesection performed during resuscitation was noted (Figure 4).

Investigations revealed low serum cortisol, low thyroid hormones, hyponatremia, hyperkalemia, and normal renal function (Figure 5). Magnetic resonance imaging (MRI) brain showed an atrophied pituitary with a thin rim of residual tissue (Figure 6).

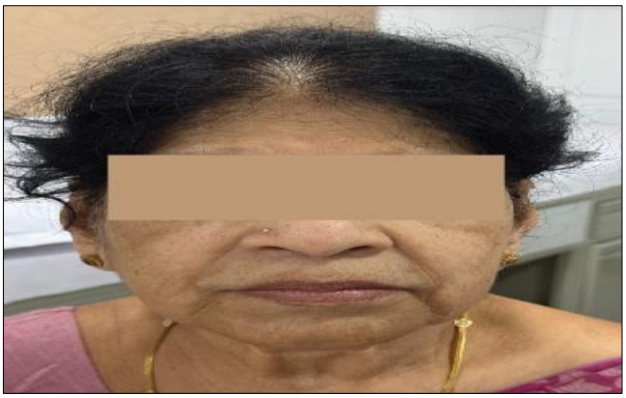

Figure 2: Fine wrinkles of face.

Figure 3: Shiny, non-myxedematous hands.

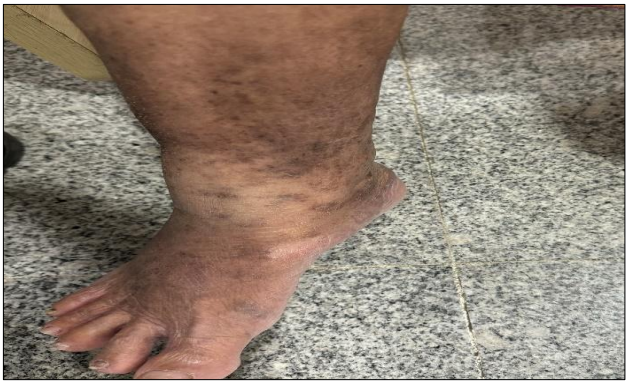

Figure 4: Scar of venesection.

Investigations

FBS : 95 mg/dl
Serum creatinine: 1.0 mg/dl
Serum Sodium: 125 meq/L
Serum Potassium: 5.2 meq/L
Serum Chloride: 92 meq/L
T3. 0.2. ng/ mL (0.6 - 2.3)
T4. 2.3 ug/ dl (4.8. - 12.0)
TSH 5.2. mcIU (0.35. - 5.5)
Serum Cortisol 1.2 (4.30 to 22.40)
Absolute Esinophilic count 200 cells / cmm (200-800)

Figure 5: Investigations.

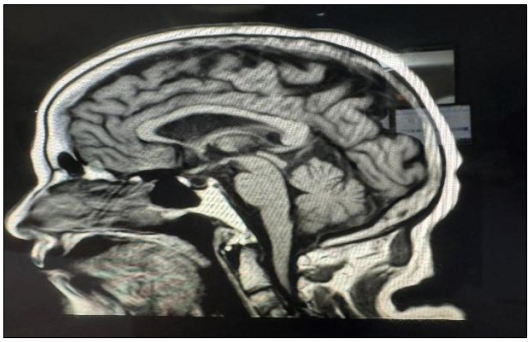

Figure 6: MRI brain showing atrophied pituitary.

She was diagnosed with Sheehan's syndrome. Hydrocortisone 20 mg AM and 10 mg PM was started, followed by gradual titration of thyroxine up to 100 mcg/day, guided by FT4 levels. At six months follow-up, she had complete resolution of symptoms and improved physical appearance (Figure 7).

Figure 7: Facial appearance six months after replacement therapy.

DISCUSSION

This case illustrates delayed diagnosis of Sheehan's syndrome more than three decades after the inciting obstetric event. Delayed recognition is common due to nonspecific symptoms and lack of awareness. Patients often fail to link current symptoms with obstetric history unless directly questioned.[5]

The patient had been incorrectly managed as primary hypothyroidism with low-dose thyroxine. In central hypothyroidism, TSH is unreliable and FT4 should guide therapy.[6] Importantly, thyroxine should never be initiated before glucocorticoid replacement, as it may precipitate adrenal crisis.[7]

A useful clinical clue is skin texture: pale and finely wrinkled in Sheehan's syndrome versus coarse, myxedematous skin in primary hypothyroidism.[8] Other common findings include hyponatremia, hypoglycemia, and elevated eosinophil counts—all of which improve with steroid therapy.[9]The patient improved significantly after appropriate hormone replacement.

CONCLUSION

Sheehan's syndrome, although rare, can remain undiagnosed for decades. A history of PPH, failure of lactation, amenorrhea, and chronic fatigue should raise suspicion. Clinicians must recognize that TSH is unreliable in central hypothyroidism, FT4 should guide levothyroxine therapy. Importantly, cortisol replacement must always precede thyroxine initiation to avoid adrenal crisis.

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