Case Report

Empty sella, full story: Delayed recognition of Sheehan syndrome in a middle-aged woman

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ABSTRACT

Sheehan's syndrome is a rare but important cause of hypopituitarism that may present years after postpartum hemorrhage (PPH) with vague, non-specific symptoms, often leading to delayed diagnosis. A 53-year-old woman from Assam presented with persistent nausea, recurrent non-bilious vomiting, and severe hyponatremia. Initial evaluation revealed esophageal candidiasis, hypotension, dehydration, and elevated inflammatory markers. Scrub typhus immunoglobulin M was positive, and she improved partially with intravenous doxycycline and fluids. However, hypotension persisted, and hyponatremia recurred after stopping IV fluids, prompting evaluation for adrenal insufficiency. This case highlights that Sheehan's syndrome can present decades after the inciting obstetric event with isolated manifestations such as recurrent hyponatremia, vomiting, and hypotension. Clinicians should maintain a high index of suspicion for Sheehan's syndrome in middle-aged women with unexplained endocrine or metabolic abnormalities, particularly in regions where home deliveries and PPH remain common.

Key words: Empty sella, Hyponatremia, Hypopituitarism, Postpartum hemorrhage, Secondary adrenal insufficiency, Sheehan syndrome

Sheehan syndrome is a rare but clinically significant cause of hypopituitarism resulting from ischemic necrosis of the pituitary gland in the immediate post-partum period. The delayed presentation of Sheehan syndrome poses a considerable diagnostic challenge because symptoms are frequently attributed to other causes such as aging, menopause, chronic illness, or gastrointestinal disorders. The diagnosis is often established only after a precipitating physiological stressor unmasks previously compensated pituitary hormone deficiencies. A detailed obstetric history, particularly regarding postpartum haemorrhage (PPH) and lactation failure, remains crucial for identifying the underlying etiology.

The patient presented nearly two decades after PPH with non-specific symptoms without any overt features of panhypopituitarism. The concurrent scrub typhus infection complicated the clinical picture and delayed diagnosis. This case highlights the importance of maintaining a high index of suspicion for Sheehan syndrome in middle-aged women presenting with unexplained hyponatremia and hypotension, even many years after the inciting obstetric event.

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CASE PRESENTATION

A 53-year-old woman, a homemaker, residing in Udalguri, Assam, presented with complaints of multiple episodes of non-bile-stained, non-projectile vomiting for a month. She had complaints of nausea for 3 months before the onset of vomiting. She denied abdominal pain, oliguria, and diarrhea.

The patient was initially treated on an outpatient basis, where routine investigations and esophagealgastroduodenoscopy were advised along with symptomatic management. The patient was reviewed in the outpatient department with no relief of symptoms, with reports indicating severe hyponatremia along with esophageal candidiasis, and the patient was admitted for further management.

On admission, the patient was afebrile (36.8°C), blood pressure was 86/54 mmHg, pulse rate was 78/min, respiratory rate was 18/min, and oxygen saturation was 98% on room air.

In view of leukocytosis on admission, inflammatory markers and infectious disease profile were sent. Scrub immunoglobulin M was positive with elevated procalcitonin and C-reactive protein, and the patient was initiated on intravenous doxycycline (Table 1).

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Table 1: Investigations of the patient

Parameter	June 11, 2025	June 14, 2025	June 15, 2025	June 17, 2025	June 21, 2025	July 11, 2025	Reference values
Hb g/dL	10.4	-	-	9.7	9.8	-	12–15
Total count×10^9/L	7.21	-	-	13.11	7.42	-	4–10
Platelet×10^9/L	189	-	-	178	204	-	150–410
Erythrocyte sedimentation rate, mm	-	-	75	-	-	-	<20
Serum sodium, mmol/L	119	122	135	129	134	141	137–145
Serum potassium, mmol/L	4.3	4.5	4.1	4.3	3.8	3.1	3.5–5.1
Serum magnesium, mg/dL	-	1.6	1.6	-	1.7	2.3	1.6–2.4
Random blood sugar, mg/dL	52	-	42	-	-	-	70–140
Procalcitonin	-	16.2	4.1	0.6	-	-	<0.02
C-reactive protein	-	171	48	25	-	-	<10
Urea, mg/dL	15	16	-	-	18	-	15–36
Creatinine, mg/dL	0.7	0.9	-	-	0.7	-	0.5–1.04
Aspartate aminotransferase, U/L	34	31	-	-	28	-	14–36
Alanine aminotransferase, U/L	16	15	-	-	13	-	<35
Alkaline phosphatase, U/L	60	53	-	-	56	-	38–126
Gamma-glutamyl transferase, U/L	13	11	-	-	11	-	12–43
Albumin g/dL	3.8	3.3	-	-	3.4	-	3.5–5
Globulin g/dL	3.6	3.2	-	-	3.6	-	2.8–3.2
Total serum bilirubin, mg/dL	0.8	0.6	-	-	0.6	-	0.2–1.3
Urine sodium, mmol/L	-	-	10	-	144	-	30–90
Serum osmolarity, mmol/L	-	-	253	-	-	-	275–290
Cortisol, nmol/L	-	-	99.8	-	-	-	123–626
TSH, mIU/L	-	-	4.2	-	-	-	0.40–4.05
FSH, mIU/L	-	-	15.0	-	-	-	13.1–86.5
LH, mIU/L	-	-	3.61	-	-	-	2.58–12.1
Prolactin, ng/mL			4.2				5–25
ACTH, pg/mL	-	-	-	-	8.46	-	10–60
Venereal disease research laboratory	Non-reactive						
ANA IF	Nuclear speckled 1+ (1:100)						
Mantoux	Non-reactive						
Scrub IgM	Positive						
WIDAL, Leptospira IgM, RDT Malaria	Negative						
Dengue NS1, IgM, IgG	Negative						
HbsAg/anti-HCV/ICTC	Negative						
Sputum Gram stain	Gram-positive cocci seen						
Sputum acid-fast stain	No acid-fast Bacilli seen						
Sputum TruNAAT	Mycobacterium tuberculosis not detected						
Automated blood culture (2 bottles)	Sterile after 5 days of incubation						
Urine culture	Sterile after 48 h of incubation						
Sputum culture sensitivity	Normal upper respiratory flora grown after overnight incubation						
CECT whole abdomen	Mild gall bladder wall thickening noted with adjacent mild free fluid noted – likely cholecystitis. Both adrenals – Appear normal in size and shape.						
CECT Thorax	Subpleural nodular pleural thickening with underlying fibro atelectatic changes, bilateral upper lobe, medial segment of right middle lobe, lingular segment of left upper lobe, posterobasal segments of bilateral lower lobes – suggestive of post-infective etiology.						
MRI brain	Empty sella, Bilateral Meckel's cave prominent.						

Hb: haemoglobin, IgM: Immunoglobulin M, ANA: Antinuclear antibody, ACTH: Adrenocorticotropic hormone, FSH: Follicle-stimulating hormone, LH: Luteinizing hormone, TSH: Thyroid-stimulating hormone, MRI: Magnetic resonance imaging, HCV: Hepatitis C virus, ICTC: Integrated Counseling and Testing Centre

Evaluation revealed a hypotonic hypovolemic hyponatremia, which was initially attributed to the persistent vomiting, and the patient was started on intravenous fluids. While the patient improved symptomatically, and serum sodium started normalizing, hypotension persisted. After the vomiting episodes stopped, hyponatremia developed after IV fluids were withdrawn. Serum fasting cortisol was

measured to rule out adrenal insufficiency and was found to be low.

Further history revealed a 3-day episode of fever and non-productive cough approximately 15 days before admission. She also reported recent unintentional weight loss. She had attained menopause 2 years prior. She was a P2L2 woman with two previous home-based normal vaginal deliveries. Her last childbirth had occurred 20 years earlier. She had a history of PPH secondary to retained placenta following her last delivery, requiring hospital admission and transfusion of one unit of packed red blood cells. She also has a history of failure of lactation following the hemorrhage episode, and the child was fed only with artificial supplements.

Based on the clinical history, two differential diagnoses were considered. The primary differential was primary adrenal insufficiency due to tuberculous adrenalitis, given the endemicity of tuberculosis in Northeast India. The other differential diagnosis was late presentation of Sheehan syndrome, although other classical features, such as hyperkalemia and manifestations of additional pituitary hormone deficiencies, were absent.

Simultaneous workup for the two differentials was undertaken. Contrast-enhanced computed tomography (CECT) Thorax revealed sequelae of previous infection, and CECT abdomen with adrenal protocol suggested inflammatory colitis with normal adrenals. The Mantoux test was negative. Sputum examination showed no acid-fast bacilli, and Mycobacterium tuberculosis was not detected on TruNAAT. Antinuclear antibody IF was weakly positive in a 1:100 dilution with a speckled pattern, and reflex testing was negative for all other antibodies. Serum adrenocorticotropic hormone (ACTH) was low (8.46 pg/mL), follicle-stimulating hormone, luteinizing hormone were normal, and thyroid-stimulating hormone was borderline raised. After primary adrenal insufficiency had been reasonably excluded, magnetic resonance imaging (MRI) of the brain was performed for further evaluation.

MRI of the brain revealed an empty sella with bilateral prominent Meckel's cave. There was no evidence of hydrocephalus or optic atrophy (Fig. 1). Ophthalmological evaluation revealed no papilledema, optic atrophy, or visual field defects. A provisional diagnosis of delayed-onset Sheehan syndrome was made. The concurrent scrub typhus infection was considered a likely precipitating stressor that unmasked the underlying endocrine insufficiency.

Oral hydrocortisone was initiated at 30 mg/day in two divided doses (20 mg in the morning and 10 mg in the afternoon). Subsequently, serum sodium normalized, and the patient was discharged with regular outpatient follow-up. The patients' hydrocortisone dose was subsequently decreased to 15 mg/day with periodic 3-monthly monitoring of serum electrolytes.

DISCUSSION

Sheehan syndrome refers to postpartum hypopituitarism resulting from ischemic necrosis of the pituitary gland,

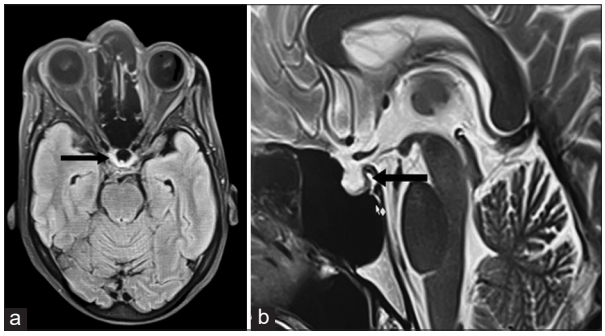


Figure 1: (a) T1-weighted magnetic resonance imaging (MRI) brain image showing empty sella; (b) T2-weighted MRI brain image showing empty sella.

usually following massive PPH or hypotension [1,2]. During pregnancy, the anterior pituitary enlarges by up to 120–136% because of lactotroph hyperplasia, while its blood supply remains predominantly dependent on the low-pressure hypophyseal portal circulation. Hence, when episodes of massive blood loss and hypotension occur, it can precipitate pituitary ischemia and infarction, leading to varying degrees of hormonal deficiency [3]. Although the incidence has declined in developed countries due to advances in obstetric care, it remains an important clinical entity in resource-limited settings where home deliveries and delayed management of PPH continue to occur.

Patients typically present in the immediate postpartum period with lactation failure, amenorrhea, and hemodynamic instability. However, delayed presentations may occur years later and can manifest as secondary adrenal insufficiency, hypothyroidism, hyponatremia, hypotension, or other pituitary hormone deficiencies. In patients who present later, symptoms are often non-specific, including features such as chronic fatigue, generalized weakness, weight loss, persistent nausea and vomiting, psychiatric disturbances like apathy or depression, and electrolyte imbalances [4,5]. Because these symptoms are subtle and frequently attributed to aging, menopause, psychiatric illness, or gastrointestinal disorders, the underlying endocrine dysfunction is commonly overlooked.

In many cases, the diagnosis is only suspected when the patient experiences an adrenal crisis during a physiological stressor such as infection, surgery, or fluid depletion. Important diagnostic clues include a historical account of PPH, failure to lactate, or prolonged amenorrhea following childbirth, even if the event occurred decades earlier [6]. Biochemical testing typically reveals secondary adrenal insufficiency, often with low ACTH and cortisol, and variable abnormalities in other pituitary hormones. Neuroimaging, particularly MRI, may demonstrate an empty sella, indicating pituitary atrophy. Late diagnosis delays appropriate hormone replacement and can result in serious complications. Timely recognition and initiation of glucocorticoid therapy (and eventually thyroid and other hormonal replacements, if needed) can lead to rapid clinical improvement.

The patient initially presented with persistent vomiting and severe hyponatremia, which was thought to be secondary to hypovolemia. Recurrence of hyponatremia despite clinical stabilization and persistent hypotension necessitated evaluation for adrenal insufficiency, ultimately revealing hypocortisolism. Hyponatremia in Sheehan syndrome is multifactorial and may result from cortisol deficiency, secondary hypothyroidism, impaired free water clearance, and inappropriate vasopressin secretion [7]. Cortisol normally suppresses antidiuretic hormone (ADH) release; therefore, cortisol deficiency leads to increased ADH secretion and water retention, resulting in dilutional hyponatremia. Unlike primary adrenal insufficiency, mineralocorticoid secretion is usually preserved because aldosterone production is primarily regulated by the renin–angiotensin–aldosterone system. Consequently, hyperkalemia is often absent, as observed in our patient [7]. Several reports have identified severe hyponatremia as the presenting feature of previously unrecognized Sheehan syndrome, occasionally leading to seizures, altered sensorium, or adrenal crisis [7,8].

MRI findings of an empty sella provided radiological confirmation of pituitary atrophy, consistent with chronic Sheehan syndrome. Previous MRI studies have reported empty sella in approximately 70–100% of patients with established Sheehan syndrome, making it one of the most consistent imaging findings in chronic disease [9,10]. Although an empty sella is not pathognomonic and may occur in other conditions, its presence in conjunction with a history of PPH and biochemical evidence of hypopituitarism strongly supports the diagnosis.

The patient's remote history of PPH, and failure of lactation following her last delivery provided the crucial diagnostic clue. In this patient, an acute scrub typhus infection likely precipitated adrenal decompensation and unmasked previously compensated cortisol deficiency [11]. The differential of primary adrenal insufficiency, especially tuberculous adrenalitis in endemic regions, must always be considered. However, normal adrenal imaging, absence of hyperkalemia, negative tuberculosis workup, and low ACTH levels ruled this out. This highlights the importance of systematic evaluation, including biochemical markers and imaging, in reaching a definitive diagnosis.

Management revolves around the prompt initiation of hormone replacement therapy. Glucocorticoid replacement must precede thyroid hormone supplementation to avoid precipitating adrenal crisis. Patient education regarding stress dosing of steroids is critical to prevent future decompensations. A long-term follow-up is required to monitor for evolving deficiencies in other pituitary hormones and to optimize replacement therapy.

This case emphasizes the importance of considering Sheehan syndrome as a differential diagnosis in middle-aged women presenting with unexplained hyponatremia, hypotension, or nonspecific systemic complaints, especially in regions where home deliveries and PPH remain prevalent. Early recognition and timely intervention can dramatically improve outcomes and prevent life-threatening crises.

CONCLUSION

This case highlights that Sheehan syndrome, though classically acute, may present decades later with subtle, isolated, and nonspecific features such as persistent vomiting and recurrent hyponatremia. A detailed obstetric history, particularly of PPH and lactation failure, remains pivotal for diagnosis even years after the inciting event. Biochemical evaluation demonstrating secondary adrenal insufficiency, along with characteristic MRI findings such as empty sella, confirms the condition. Early initiation of glucocorticoid replacement leads to rapid symptom resolution and prevents life-threatening adrenal crisis. Thus, clinicians must maintain a high index of suspicion for Sheehan syndrome in middle-aged women with unexplained endocrine or metabolic abnormalities.

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