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Case Report

Between the devil and the deep blue sea: puerperal sepsis and pituitary apoplexy following obstetric hysterectomy for postpartum haemorrhage

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ABSTRACT

Sheehan's syndrome, or postpartum pituitary necrosis, is a rare but life-threatening complication of severe postpartum hemorrhage (PPH). Its diagnosis is often delayed or obscured when confounded by concurrent critical illnesses. We report a case of acute Sheehan's syndrome masked by severe puerperal sepsis to highlight the diagnostic challenges involved. A 38-year-old multiparous woman developed severe atonic PPH following a vacuum-assisted delivery, necessitating an emergency subtotal hysterectomy. Her postoperative course was complicated by severe sepsis, multi-organ dysfunction, and recurrent hypoglycaemia. Despite aggressive sepsis management, her condition remained critical. A pivotal diagnostic clue was her complete failure to lactate. Hormonal assays revealed panhypopituitarism (low follicle-stimulating hormone (FSH), luteinizing hormone (LH), thyroid-stimulating hormone (TSH), and cortisol), and a computed tomography (CT) scan of the brain confirmed an empty sella. The patient stabilized only after the initiation of hormone replacement therapy (hydrocortisone and thyroxine). Sheehan's syndrome should be suspected in any patient with a history of severe PPH who presents with non-specific signs of critical illness, particularly refractory hypoglycaemia and failure to lactate. Concomitant sepsis can mask these features, delaying diagnosis. Early recognition and multidisciplinary management are essential for survival.

Keywords: Sepsis, Sheehans, PPH, Endocrine dysfunction, Pituitary failure, Obstetrics

INTRODUCTION

Postpartum hemorrhage (PPH) remains a leading cause of maternal mortality, particularly in low-resource settings. A rare but devastating sequela of massive blood loss during childbirth is Sheehan's syndrome, defined as ischemic necrosis of the pituitary gland.[1] During pregnancy, the pituitary gland undergoes physiological hyperplasia, increasing its metabolic demand and making it highly vulnerable to ischemic injury from hypovolemic shock.[2] The resulting anterior pituitary insufficiency typically manifests as failure to lactate (agalactia) and amenorrhea, with more severe cases presenting as adrenal crisis or myxoedema coma.[3] However, the diagnosis is often challenging in the acute setting because symptoms such as fatigue, hypotension, and tachycardia may be attributed to anaemia, postpartum recovery, or infection.[1-4] This case report presents an acute manifestation of Sheehan's syndrome where the diagnosis was significantly obscured by severe puerperal sepsis, emphasizing the need for a high index of suspicion in critically ill postpartum women.

CASE REPORT

A 38-year-old multiparous woman (Para 4, Living 4) was referred to our tertiary care centre with severe postpartum complications. She was a migrant labourer with no history of antenatal care during this pregnancy. Her obstetric history included three previous uncomplicated vaginal deliveries. She presented to a peripheral hospital in

spontaneous labor at approximately 37 weeks gestation with severe anaemia (haemoglobin 3.7 g/dL). Following the vacuum-assisted vaginal delivery of a healthy 2.5 kg female infant, she developed atonic PPH. Medical management with oxytocics (including tranexamic acid and methylergometrine) and mechanical maneuvers failed to control the bleeding. The patient developed hypovolemic shock (shock index 1.85), necessitating the initiation of noradrenaline and an emergency obstetric subtotal hysterectomy with bilateral salpingectomy. Over the initial 48 hours, she received massive transfusion support comprising 8 units of packed red blood cells (PRBCs), 10 units of platelets, and 8 units of cryoprecipitate.

Initial findings and sepsis

Upon transfer to our institution on postoperative day (POD) 2, the patient was stable on inotropic support. Initial laboratory investigations revealed haemoglobin of 6.9 g/dL, thrombocytopenia (platelets 73,000/µL), and hypoalbuminemia (2.1 g/dL). Renal and liver function tests were otherwise normal. By POD 6, the patient developed high-grade fever. Investigations indicated a rising total leukocyte count (16,000/µL) and elevated inflammatory markers (ESR 59 mm/hr). Imaging revealed a right pleural effusion and a complex collection in the pouch of Douglas (6x5 cm). Despite broad-spectrum antibiotics (piperacillin-tazobactam, later escalated to meropenem and vancomycin), the patient remained febrile and clinically unstable. On POD 13, she developed a focal seizure progressing to a generalized tonic-clonic seizure. A contrast-enhanced computed tomography (CECT) of the abdomen confirmed a peripherally enhancing collection, necessitating a laparotomy for drainage of 50 mL of pus.

Endocrine evaluation and diagnosis

Throughout the septic episode, the patient exhibited recurrent, unexplained hypoglycaemia (blood glucose nadir 49 mg/dL) requiring frequent dextrose correction.

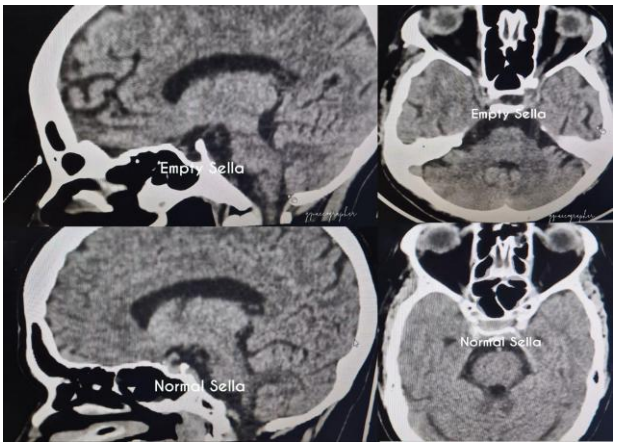

Figure 1: CT brain (sagittal and coronal views) showing an empty sella turcica (white text), consistent with pituitary necrosis.

A critical retrospective history revealed that the patient had failed to lactate since delivery, a marked deviation from her previous successful breastfeeding experiences. Suspecting Sheehan's syndrome, a hormonal profile was obtained (Table 1). The hormonal assay confirmed central hypothyroidism and secondary adrenal insufficiency. A non-contrast CT scan of the brain revealed an empty sella turcica, confirming the diagnosis of pituitary necrosis (Figure 1).

Management and outcome

The patient was immediately started on intravenous hydrocortisone (100 mg every 8 hours), followed by oral thyroxine (100 mcg daily). Following the initiation of hormone replacement, her hemodynamic status improved rapidly, hypoglycaemic episodes ceased, and her sensorium cleared. She was discharged on maintenance oral steroids and thyroxine with advice for lifelong follow-up.

Table 1: Hormonal profile at POD 13 revealing panhypopituitarism.

Hormone	Patient value	Reference range
TSH	1.0 µiu/ml	0.4 - 4.0 µiu/ml (inappropriately normal)
FSH	0.8 miu/ml	1.5-12.4 miu/ml
LH	<0.2 miu/ml	2.4-12.6 miu/ml
Cortisol (8 am)	6.0 µg/dl	6.0-23.0 µg/dl (inappropriately low for stress)
Estradiol	18 pg/ml	30-400 pg/ml
Prolactin	Not detectable	Non-pregnant: 4-23 ng/ml

DISCUSSION

This case highlights a life-threatening presentation of Sheehan's syndrome where the diagnosis was obscured by severe postpartum sepsis. The primary diagnostic challenge was the symptomatic overlap between sepsis and the adrenal crisis secondary to hypopituitarism. Both conditions can present with hypotension, tachycardia, and altered mental status. However, in this case, the persistence of shock and hypoglycaemia despite adequate sepsis control was the key indicator of underlying endocrine failure.

The systemic inflammatory response from sepsis likely exacerbated the patient's condition. Recent literature suggests that infections, such as dengue, can unmask underlying Sheehan's syndrome by precipitating an adrenal crisis in a patient with limited pituitary reserve.[5] In our patient, the metabolic stress of sepsis likely overwhelmed the compromised adrenal axis, leading to over crisis.

The two most critical diagnostic clues in this case were failure of lactation and recurrent hypoglycaemia. Failure to lactate is the most common and earliest sign of Sheehan's syndrome, occurring in up to 67% of cases due to prolactin deficiency.[6] Hypoglycaemia, caused by cortisol and growth hormone deficiency, is a dangerous manifestation that can be easily overlooked in a septic patient where glucose fluctuations are common.[4,5] Diagnostic imaging is confirmatory. In the acute phase, the pituitary may appear enlarged or haemorrhagic, but the classic "empty sella" sign, as seen in our patient (Figure 1), is typically a later finding indicating arachnoid herniation into the sella turcica due to gland shrinkage.[7,8] Management requires a multidisciplinary approach. While treating the inciting hemorrhage and sepsis is paramount, stabilization is often impossible without addressing the adrenal insufficiency. It is crucial to administer glucocorticoids before thyroid hormone replacement to avoid precipitating an adrenal crisis.[2]

CONCLUSION

Sheehan's syndrome is a critical diagnosis to consider in any woman with a history of severe PPH who fails to recover as expected. This case demonstrates that the clinical presentation can be acute and masked by overwhelming sepsis. Failure to lactate and refractory hypoglycaemia are cardinal signs that should prompt immediate endocrine evaluation. Early recognition and initiation of hormone replacement therapy are life-saving.

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