Pan-Hypopituitarism Secondary to Sheehan's Syndrome: A Case Report and Review of the Literature

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ABSTRACT

Background: Sheehan's syndrome (SS) is an under-recognised cause of hypopituitarism resulting from ischaemic necrosis of the anterior pituitary gland following severe postpartum haemorrhage (PPH) and circulatory collapse. Despite being preventable with optimal obstetric care, SS remains clinically relevant in low- and middle-income countries where PPH-related maternal morbidity persists.
Case Presentation: We report a 30-year-old woman who presented three years after severe PPH (requiring 11 units of blood transfusion) with progressive fatigue, secondary amenorrhoea, agalactia, significant weight loss, and alopecia. Hormonal evaluation revealed pan-hypopituitarism with markedly low morning serum cortisol (<11 nmol/L), inappropriately low-normal adrenocorticotropic hormone (ACTH; 11 pg/mL), low free thyroxine (<5.15 pmol/L) with a non-elevated thyroid-stimulating hormone (TSH; 0.883 mIU/L), and suppressed gonadotrophins. Cranial magnetic resonance imaging (MRI) demonstrated an empty sella consistent with pituitary atrophy. A diagnosis of SS with secondary adrenal insufficiency, central hypothyroidism, and hypogonadotrophic hypogonadism was established.
Management and Outcome: Sequential hormone replacement—glucocorticoids (prednisolone) followed by levothyroxine. At four-week follow-up, the patient demonstrated significant clinical improvement including weight gain (4 kg), improved energy, and restored appetite, with partial biochemical recovery.
Conclusion: This case illustrates the protracted diagnostic course of SS and reinforces the importance of maintaining clinical suspicion in women with a history of severe PPH who present with multi-axis endocrine dysfunction. Prioritising glucocorticoid over thyroid hormone replacement is essential to prevent adrenal crisis. Systematic long-term endocrinological surveillance is warranted for all confirmed cases.

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INTRODUCTION

Sheehan's syndrome (SS) is an acquired form of hypopituitarism caused by ischaemic necrosis of the anterior pituitary gland during or immediately following severe postpartum haemorrhage (PPH) and haemodynamic compromise (Karaca & Kelestimur, 2020). During pregnancy, the pituitary gland undergoes physiological enlargement

predominantly due to lactotrophic hyperplasia without a commensurate increase in blood supply, rendering it highly susceptible to ischaemic injury during episodes of systemic hypotension or circulatory shock (Karaca & Kelestimur, 2020).

Although the global incidence of SS has declined in parallel with improvements in obstetric care, it remains an important and underappreciated cause of hypopituitarism in low- and middle-income countries (LMICs), where PPH continues to account for a disproportionate share of maternal morbidity and mortality (World Health Organization, 2018).

Epidemiological data suggest that SS represents up to 3-5% of hypopituitarism cases in developed settings, with substantially higher proportions reported from South Asia and Sub-Saharan Africa, where health system capacity for emergency obstetric care may be limited (Laway et al., 2019; Karaca & Kelestimur, 2020).

The clinical presentation of SS is characteristically insidious and highly variable, contributing to substantial diagnostic delay often measured in decades rather than months (Laway et al., 2019; Karaca & Kelestimur, 2020). While acute manifestations such as failure to lactate (agalactia), cardiovascular instability, and amenorrhoea may appear immediately postpartum, many patients present later with non-specific symptoms including fatigue, weakness, cold intolerance, weight changes, and neuropsychiatric features (Laway et al., 2019; Karaca & Kelestimur, 2020). These manifestations reflect deficiencies across multiple anterior pituitary axes, most critically adrenocorticotropic hormone (ACTH) and thyroid-stimulating hormone (TSH), which carry the greatest risk of life-threatening complications if untreated.

Adrenal insufficiency secondary to ACTH deficiency may manifest with fatigue, hyponatraemia, hypoglycaemia, and haemodynamic compromise, with a risk of adrenal crisis precipitated by physiological stress (Fleseriu et al., 2024). Central (secondary) hypothyroidism, characterised by low free thyroxine (FT4) with a non-elevated or inappropriately normal TSH, is a recognised diagnostic pitfall and requires targeted awareness (Castinetti & Brue, 2021). Neuroimaging, preferably MRI commonly reveals partial or complete empty sella in established disease, reflecting progressive pituitary involution (Karaca & Kelestimur, 2020).

Early recognition is critical, as prompt and correctly sequenced hormone replacement therapy particularly initiating glucocorticoids before thyroid hormone can prevent adrenal crisis and significantly improve patient outcomes (Fleseriu et al., 2024; Castinetti & Brue, 2021). Despite advances in diagnostic modalities, SS remains underdiagnosed, especially in resource-limited settings with limited access to dynamic pituitary function tests and specialist endocrinological services. Case reports continue to serve an important educational function by highlighting the range of clinical presentations and reinforcing the value of a thorough obstetric history in women presenting with unexplained hypopituitarism (Laway et al., 2019; Karaca & Kelestimur, 2020).

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We report a case of SS in a 30-year-old Nigerian woman presenting three years after severe PPH, with biochemical evidence of secondary adrenal insufficiency, central hypothyroidism, and hypogonadotrophic hypogonadism, and empty sella on MRI. This report underscores the importance of maintaining a high index of suspicion in at-risk individuals, particularly in resource-constrained settings where delayed diagnosis remains common.

CASE PRESENTATION

Patient History and Presenting Complaint

A 30-year-old Nigerian woman of Edo State origin presented to the endocrinology outpatient clinic with a three-year history of progressive fatigue, generalised weakness, dizziness, cold intolerance, and secondary amenorrhoea. She reported complete inability to lactate from the time of her most recent delivery, approximately three years prior to presentation.

Her most recent obstetric event was complicated by severe PPH requiring transfusion of 11 units of packed red blood cells and prolonged hospitalisation. The delivery resulted in a fresh stillbirth via spontaneous vaginal delivery. Approximately six months following that delivery, she developed persistent amenorrhoea, diffuse hair thinning with subsequent patchy hair loss, and progressive unintentional weight loss from a pre-illness weight of 70 kg to 56 kg at presentation (a loss of 14 kg over approximately two and a half years).

She denied any significant bowel habit change but reported persistent generalised sluggishness, poor appetite, and reduced motivation. There was no prior history of thyroid disease, head trauma, intracranial surgery, pituitary tumour, radiotherapy, or chronic systemic illness. She was not taking long-term medications and denied use

of hormonal contraceptives or herbal preparations. Family history was non-contributory. Two years prior to the index pregnancy, she had experienced an uncomplicated vaginal delivery of a live infant with normal lactation.

Physical Examination

On general examination, the patient appeared lethargic, pale, and chronically ill-looking. Notable findings included fluffy, sparse scalp hair with patchy alopecia, periorbital puffiness, dry and cool skin, sparse axillary and pubic hair. Her temperature was 36.1°C. Weight was 56 kg with a body mass index (BMI) of 21.8 kg/m². Vital signs revealed a resting pulse of 73 beats per minute and blood pressure of 90/60 mmHg in the sitting position, indicative of postural hypotension.

Neurological assessment revealed delayed relaxation of deep tendon reflexes bilaterally-a classical feature of hypothyroidism without focal deficits. No visual field defects were detected on confrontation testing, and cranial nerve examination was unremarkable. Thyroid gland was impalpable. Breast examination revealed no galactorrhoea.

Investigations

Initial haematological and hormonal evaluation demonstrated evidence of anterior pituitary dysfunction across multiple axes (Table 1). Notable electrolyte findings included mild hyponatraemia (sodium 133 mmol/L; reference: 135–145 mmol/L), likely attributable to secondary adrenal insufficiency. Other electrolytes, renal function indices, and random plasma glucose were within normal limits, although the plasma glucose level of 70.2 mg/dL was at the lower margin of the reference range, consistent with the risk of relative hypoglycaemia in cortisol-deficient states.

The pituitary–adrenal axis assessment revealed a critically low morning serum cortisol of <11 nmol/L (reference: 240–618 nmol/L) alongside an inappropriately normal ACTH of 11.0 pg/mL (reference: <46.0 pg/mL). This discordant pattern of profoundly low cortisol without a compensatory rise in ACTH is diagnostic of secondary (central) adrenal insufficiency and distinguishes SS from primary Addison's disease, in which ACTH would be markedly elevated (Gruber and Bancos, 2022; Lewis et al., 2023).

The pituitary–thyroid axis evaluation demonstrated markedly low FT_4 (<5.15 pmol/L; reference: 9.0–19.0 pmol/L) and FT_3 (<1.64 pmol/L; reference: 2.42–6.02 pmol/L) with a non-elevated TSH (0.883 mIU/L; reference: 0.35–4.94 mIU/L) (Table 1). This biochemical pattern of profoundly low thyroid hormones with a TSH that is inappropriately low or normal rather than elevated is pathognomonic of central hypothyroidism due to TSH deficiency (Persani et al., 2019).

Assessment of the gonadotrophic axis revealed suppressed LH (0.81 mIU/mL) and FSH (1.33 mIU/mL) consistent with hypogonadotrophic hypogonadism. Oestradiol was at the lower end of the reference range (27 pg/mL), and progesterone was below the reference threshold (<0.5 ng/mL). Serum prolactin was 6.64 ng/mL, within the reference range, suggesting variable but not absent residual lactotroph function.

Cranial MRI demonstrated an empty sella configuration on sagittal T2-weighted SSFSE sequences, with CSF-intensity signal filling the sella turcica and near-complete absence of visible pituitary parenchyma (Figure 1). No pituitary mass, haemorrhage, or suprasellar extension was identified. These imaging findings, in conjunction with the obstetric history, symptom chronology, and hormonal profile, established a diagnosis of SS with pan-hypopituitarism.

Management

Following clinical assessment and confirmatory hormonal evaluation, the patient was commenced on a structured sequential hormone replacement protocol. Given the profound secondary adrenal insufficiency, glucocorticoid replacement was prioritised to prevent adrenal crisis prior to thyroid hormone initiation-a critical sequencing principle in combined ACTH and TSH deficiency (Persani et al., 2019; Iglesias, 2024; Fleseriu et al., 2024). Oral prednisolone was started at a physiological dose of 10 mg in the morning and 5 mg in the evening, approximating the normal diurnal cortisol secretion pattern.

Following clinical stabilisation on glucocorticoid replacement, levothyroxine was introduced at 50 micrograms daily and titrated according to free T_4 levels (targeting the upper half of the reference range), with TSH acknowledged as unreliable for monitoring in central hypothyroidism (Persani et al., 2019).

Patient education was extensive and encompassed: (i) the necessity of lifelong hormone replacement adherence; (ii) stress-dose glucocorticoid protocols (doubling the daily dose during intercurrent illness or physiological stress); (iii) sick-day rules and recognition of adrenal crisis symptoms; (iv) the importance of carrying a steroid alert card/bracelet; and (v) the need for regular endocrinological follow-up.

At first review four weeks after treatment initiation, the patient reported significant subjective improvement in energy, appetite, and

general well-being. Objective findings included a 4kg weight gain and visibly improved hair regrowth. Repeat hormonal profile demonstrated partial biochemical recovery: serum cortisol increased to 58.83 nmol/L and FT_4 improved to 8.81 pmol/L with FT_3 rising to 2.02 pmol/L (Table 1). Further dose titrations were undertaken, and a structured plan for periodic reassessment of all pituitary axes including consideration of sex steroid replacement, bone density assessment, and metabolic monitoring was established as part of long-term management.

Table 1: Laboratory Investigations at Presentation and After 4 Weeks of Hormone Replacement Therapy

Parameter	Value at Presentation	Reference Range	Value After 4 Weeks
Haematology			
Haemoglobin (g/dL)	10.2	12.0–16.0	11.1
White blood cell count ($\times 10^9$/L)	7.2	4.0–11.0	6.9
Platelet count ($\times 10^9$/L)	250	150–400	251
Electrolytes and Renal Function			
Sodium (mmol/L)	**133**	135–145	138
Potassium (mmol/L)	3.7	3.5–5.5	3.9
Chloride (mmol/L)	96	90–110	95
Bicarbonate (mmol/L)	24	20–30	25
Urea (mg/dL)	26	10–40	27
Creatinine (mg/dL)	1.0	0.5–1.2	0.9
Glucose Metabolism			
Random plasma glucose (mg/dL)	70.2	70–140	90
Pituitary–Adrenal Axis			
8 AM serum cortisol (nmol/L)	**<11**	240–618	**58.83**
ACTH (pg/mL)	**11.0**	<46.0	**—**
Pituitary–Thyroid Axis			
TSH (mIU/L)	**0.883**	0.35–4.94	**0.26**
Free T_4 (pmol/L)	**<5.15**	9.0–19.0	**8.81**
Free T_3 (pmol/L)	**<1.64**	2.42–6.02	**2.02**
Pituitary–Gonadal Axis			
LH (mIU/mL)	**0.81**	2.95–13.65	**—**
FSH (mIU/mL)	**1.33**	4.46–12.43	**—**
Oestradiol (pg/mL)	27	20–146	—
Progesterone (ng/mL)	**<0.5**	0.50–2.0	**—**
Other Pituitary Hormones			
Prolactin (ng/mL)	6.64	4.60–25.0	—

Bold/highlighted rows indicate values outside the reference range. '—' indicates data not recorded or not repeated at follow-up.

Abbreviations: *ACTH = adrenocorticotropic hormone; TSH = thyroid-stimulating hormone; FT_4 = free thyroxine; FT_3 = free triiodothyronine; LH = luteinising hormone; FSH = follicle-stimulating hormone.*

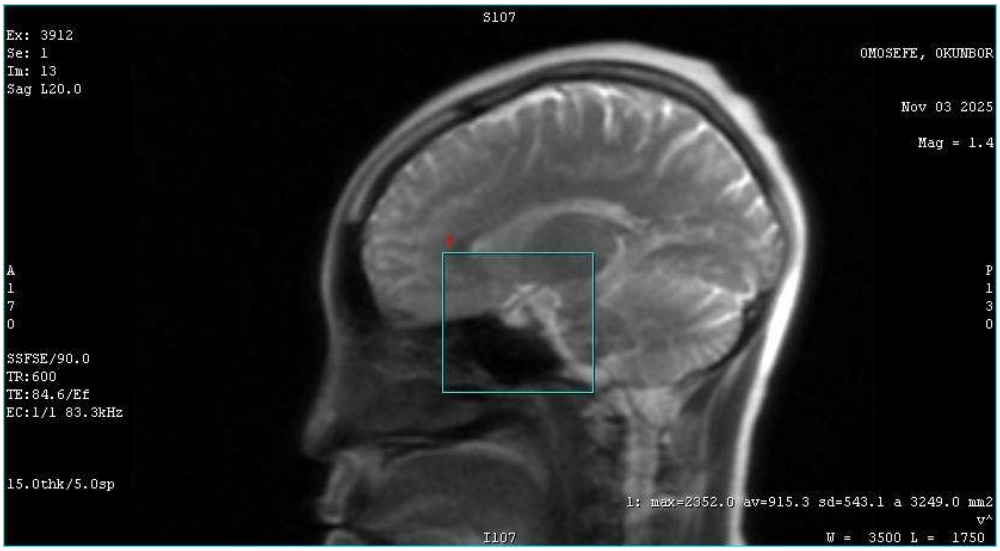

Figure 1. *Sagittal T2-weighted SSFSE MRI (slice thickness 15 mm, Series 1, Image 13) demonstrating an empty sella configuration. The sella turcica is occupied by cerebrospinal fluid-intensity signal (T2-hyperintense) with no identifiable pituitary parenchyma, consistent with pituitary involution following postpartum haemorrhage in Sheehan's syndrome. The cyan box highlights the sellar region of interest. Note: dedicated pituitary-protocol thin-section MRI (2–3 mm) was not available at the study centre; the infundibulum sign could not be resolved at this slice thickness.*

DISCUSSION

This case illustrates the classic diagnostic triad of chronic Sheehan's syndrome: biochemical hypopituitarism involving multiple hormonal axes, central adrenal insufficiency, and sellar imaging demonstrating empty sella. The three-year diagnostic delay observed in our patient is consistent with published literature median delays of 5–20 years have been reported and underlines the persistent challenge of recognising SS in clinical practice (Laway et al., 2019; Karaca & Kelestimur, 2020; Karaca & Kelestimur, 2025).

Pathophysiology

The pathophysiological basis of SS relates to the unique haemodynamic vulnerability of the enlarged pregnant pituitary gland. During pregnancy, the pituitary nearly doubles in volume due to oestrogen-driven lactotroph hyperplasia; however, its vascular supply does not expand commensurately (Karaca & Kelestimur, 2020). During severe PPH or circulatory shock, a precipitous fall in portal blood flow results in ischaemic necrosis of the anterior pituitary parenchyma, with the posterior pituitary relatively spared due to its direct arterial supply. The degree of necrosis correlates with the severity and duration of hypotension, explaining the spectrum from partial to complete pituitary failure (Karaca & Kelestimur, 2025).

Secondary Adrenal Insufficiency

The most immediately life-threatening finding in our patient was secondary adrenal insufficiency, evidenced by a profoundly subnormal morning cortisol (<11 nmol/L) alongside an inappropriately low-normal ACTH (11 pg/mL). In primary adrenal insufficiency (Addison's disease), ACTH would be expected to be markedly elevated; the pattern observed here of low cortisol with non-elevated ACTH is the biochemical hallmark of secondary ACTH deficiency (Gruber & Bancos, 2022; Lewis et al., 2023; Charoensri & Auchus, 2024). Contemporary diagnostic guidance suggests that a random cortisol below 100–110 nmol/L in a clinically appropriate context is highly predictive of adrenal insufficiency, rendering dynamic stimulation testing a confirmatory rather than essential step where clinical and biochemical evidence is compelling (Lewis et al., 2023; Charoensri & Auchus, 2024).

The clinical sequelae of unrecognised secondary adrenal insufficiency include fatigue, postural hypotension (blood pressure 90/60 mmHg in our patient), hyponatraemia, relative hypoglycaemia, and the risk of life-threatening adrenal crisis during physiological stress. Prompt glucocorticoid replacement is therefore both diagnostic and therapeutic, and adherence to stress-dose protocols is a cornerstone of ongoing management (Lewis et al., 2023; Charoensri & Auchus, 2024).

Central Hypothyroidism

Central (secondary) hypothyroidism in this patient was characterised by markedly depressed FT_4 (<5.15 pmol/L) and FT_3 (<1.64 pmol/L) in conjunction with a TSH that was low-normal (0.883 mIU/L) rather than elevated. This biochemical signature represents a well-recognised diagnostic pitfall: clinicians relying on TSH alone would fail to identify central hypothyroidism, as TSH is low or inappropriately normal due to the underlying TSH deficiency (Persani et al., 2019). Current endocrinological guidance recommends diagnosis based on low FT_4 with an inappropriately normal or suppressed TSH in the clinical context of pituitary disease, with levothyroxine dose titration targeting FT_4 in the upper half of the reference range (Persani et al., 2019).

Hypogonadotrophic Hypogonadism and Other Axes

Gonadotrophic axis suppression (LH 0.81 mIU/mL; FSH 1.33 mIU/mL) was also evident, consistent with hypogonadotrophic hypogonadism, a recognised feature of SS that contributes to amenorrhoea, infertility, impaired bone health, and reduced quality of life (Karaca & Kelestimur, 2025; Mandal et al., 2020). While sex steroid replacement was deferred pending dose stabilisation, this axis warrants systematic reassessment in the long-term management plan. Growth hormone/IGF-1 assessment was not undertaken at this presentation but should form part of comprehensive subsequent evaluation, as GH deficiency may contribute to adverse metabolic and cardiovascular outcomes (Fleseriu et al., 2024).

Neuroimaging

The MRI findings in our patient are consistent with the radiological hallmark of chronic SS. The sagittal T2-weighted SSFSE sequence demonstrated CSF-intensity signal filling the sella turcica with near-complete absence of pituitary parenchyma, corresponding to the empty sella configuration (Figure 1). Contemporary cohort data demonstrate high rates of partial or complete empty sella in confirmed SS cases on dedicated pituitary MRI (Mandal et al., 2020). It is acknowledged that the 15 mm slice thickness used in this study falls short of the 2–3 mm slices recommended for dedicated pituitary-protocol imaging, and as a consequence the infundibulum sign and any residual thin pituitary rim could not be resolved. However, the T2 CSF-signal filling of the sella, in the context of the clinical and hormonal findings, is sufficient to support the diagnosis. Clinicians in resource-limited settings should be aware that standard brain MRI sequences may demonstrate empty sella even when dedicated pituitary protocols are unavailable.

Management Principles and Sequencing

A key lesson from this case is the principle of hormone replacement sequencing. In the setting of concurrent ACTH deficiency and central hypothyroidism, thyroid hormone replacement must not be initiated without prior glucocorticoid cover, as levothyroxine accelerates cortisol clearance and increases overall metabolic demand, potentially precipitating acute adrenal crisis (Persani et al., 2019; Iglesias, 2024; Fleseriu et al., 2024). Patient education on steroid sick-day rules and emergency glucocorticoid use is integral to long-term safety and remains the most modifiable risk factor for preventable adrenal crisis-related mortality (Lewis et al., 2023; Charoensri & Auchus, 2024).

Context and Implications

This case carries particular relevance for healthcare providers in Nigeria and other LMICs, where the combination of high PPH incidence, limited access to specialist endocrinology, and restricted availability of dynamic pituitary function tests creates conditions in which SS is systematically underdiagnosed. A detailed obstetric history, specifically enquiring about PPH, blood transfusion, failure to lactate, and postpartum amenorrhoea should be an integral component of the evaluation of any woman presenting with unexplained fatigue, amenorrhoea, or evidence of multi-axis endocrine dysfunction, regardless of the interval since delivery. Structured referral protocols between obstetric and endocrine services, and improved access to basic hormonal assays, represent viable system-level responses to this ongoing diagnostic gap (World Health Organization, 2018; Makharia et al., 2021).

CONCLUSION

This case of Sheehan's syndrome presenting with pan-hypopituitarism three years after severe postpartum haemorrhage exemplifies the protracted and heterogeneous natural history of this condition. The diagnostic triad of multi-axis anterior pituitary failure, secondary adrenal insufficiency with central hypothyroidism, and empty sella on MRI, in the context of a detailed obstetric history, should prompt clinical recognition even in the absence of dynamic stimulation testing. Glucocorticoid replacement must precede thyroid hormone initiation to prevent adrenal crisis. A high index of clinical

suspicion particularly in resource-limited settings where severe PPH remains prevalent is the most critical tool for timely diagnosis. Lifelong, systematically monitored, multi-axis hormone replacement is essential for optimising patient outcomes and quality of life.

Patient Consent for Publication

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images, including the MRI scan presented in Figure 1.

Ethics Statement

This case report was prepared in accordance with the CARE (Case Reports) guidelines (Riley et al., 2017). No identifiable personal data other than that necessary for clinical illustration are disclosed. Institutional review board approval was not required for the publication of a single anonymised case report as per local institutional policy; however, written patient consent was obtained as stated above.

Competing Interests

The authors declare that they have no competing interests.

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