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WHEN THE POSTERIOR PITUITARY SPEAKS: SHEEHAN SYNDROME PRESENTING AS CENTRAL DIABETES INSIPIDUS FOLLOWING SEVERE POSTPARTUM HEMORRHAGE—A RARE ENDOCRINE EMERGENCY

General Surgery

Dr Anupreetta M H* MBBS, Junior Resident, Preethashree Hospital, Tirumangalam, Tamilnadu, India *Corresponding Author

Dr Hemamalini Rajamanikkam MBBS, DGO, MS (General Surgery), Consultant, Preethashree Hospital, Tirumangalam, Tamil Nadu, India

ABSTRACT

Background: Sheehan syndrome is a rare but potentially life-threatening consequence of severe postpartum hemorrhage, resulting from ischemic necrosis of the pituitary gland. While anterior pituitary hormone deficiencies are well recognized, central diabetes insipidus (CDI) is an exceptionally uncommon manifestation because the posterior pituitary is relatively protected by its direct arterial blood supply. The coexistence of panhypopituitarism and CDI often delays diagnosis and may lead to significant morbidity if unrecognized. **Case Presentation:** A 30-year-old multiparous woman developed massive postpartum hemorrhage (approximately 2.5 L) following vaginal delivery, complicated by hemorrhagic shock requiring blood transfusion and intensive resuscitation. Ten weeks later, she presented with failure of lactation, secondary amenorrhea, profound fatigue, postural dizziness, anorexia, cold intolerance, and progressive polyuria and polydipsia (9–10 L/day). Laboratory evaluation revealed hypernatremia, elevated serum osmolality, inappropriately dilute urine, and biochemical evidence of panhypopituitarism, including secondary adrenal insufficiency, central hypothyroidism, hypogonadotropic hypogonadism, growth hormone deficiency, and markedly reduced prolactin levels. A water deprivation test followed by desmopressin administration confirmed CDI. Magnetic resonance imaging demonstrated a partially empty sella, marked anterior pituitary atrophy, thinning of the pituitary stalk, and absence of the posterior pituitary bright spot. Sequential hormone replacement with hydrocortisone, levothyroxine, and desmopressin resulted in rapid biochemical correction and sustained clinical recovery. **Conclusion:** This case highlights the importance of considering Sheehan syndrome in postpartum women presenting with persistent polyuria in addition to classic symptoms such as lactation failure and amenorrhea. Early recognition of this rare association, prompt endocrine evaluation, and timely initiation of appropriate hormone replacement therapy are essential to prevent life-threatening complications and achieve favorable long-term outcomes.

KEYWORDS

Sheehan Syndrome; Central Diabetes Insipidus; Panhypopituitarism; Postpartum Hemorrhage

INTRODUCTION

Sheehan syndrome is an uncommon but potentially life-threatening cause of postpartum hypopituitarism resulting from ischemic necrosis of the pituitary gland following severe postpartum hemorrhage (PPH). Although its incidence has declined considerably in developed countries because of advances in obstetric care, it continues to be encountered in developing countries where delayed recognition contributes to significant maternal morbidity and mortality.[1] During pregnancy, the anterior pituitary gland enlarges by nearly 30–100% because of estrogen-induced lactotroph hyperplasia, increasing its metabolic demand while remaining dependent on the low-pressure hypophyseal portal circulation. Consequently, severe obstetric hemorrhage with prolonged hypotension or hemorrhagic shock can precipitate ischemic infarction of the anterior pituitary, resulting in varying degrees of hypopituitarism.[2,3]

The clinical spectrum of Sheehan syndrome is remarkably heterogeneous. Patients may present acutely in the postpartum period or remain undiagnosed for months to decades after the inciting obstetric event. Failure of lactation, secondary amenorrhea, fatigue, generalized weakness, postural hypotension, cold intolerance, anorexia, and recurrent hyponatremia are among the classical manifestations of anterior pituitary hormone deficiency. Because these symptoms are often nonspecific and evolve gradually, the diagnosis is frequently overlooked until multiple endocrine deficiencies become clinically evident.[1,4]

Posterior pituitary dysfunction is exceptionally uncommon in Sheehan syndrome because the neurohypophysis derives its blood supply directly from the inferior hypophyseal arteries, rendering it relatively resistant to ischemic injury. Consequently, central diabetes insipidus (CDI) has been reported only rarely, with fewer than 2% of patients demonstrating clinically significant vasopressin deficiency in published series.[5,6] The coexistence of panhypopituitarism and CDI therefore represents an unusual and diagnostically challenging presentation. Persistent polyuria and polydipsia in the postpartum period are more commonly attributed to gestational diabetes insipidus, osmotic diuresis, primary polydipsia, or renal disorders, often delaying appropriate endocrine evaluation.[6,7]

Magnetic resonance imaging (MRI) of the hypothalamic–pituitary region provides important supportive evidence for the diagnosis by demonstrating partial or complete empty sella, anterior pituitary atrophy, thinning of the pituitary stalk, and, in rare cases of posterior pituitary involvement, absence of the characteristic posterior pituitary bright spot. Comprehensive hormonal assessment together with dynamic endocrine testing remains essential for establishing the diagnosis and guiding appropriate management.[2,8]

Early recognition of Sheehan syndrome is of paramount importance because untreated secondary adrenal insufficiency may be fatal. Current endocrine guidelines recommend prompt glucocorticoid replacement before initiation of thyroid hormone therapy to avoid precipitating adrenal crisis, followed by replacement of other deficient hormones according to clinical and biochemical findings. Patients with confirmed CDI require desmopressin therapy, which typically results in rapid normalization of urine output, serum sodium concentration, and overall clinical status.[3,8]

We report a rare case of Sheehan syndrome with panhypopituitarism presenting as central diabetes insipidus approximately 10 weeks after severe postpartum hemorrhage. The case highlights the importance of maintaining a high index of suspicion for posterior pituitary involvement in postpartum women presenting with persistent hypotonic polyuria in addition to classical manifestations of anterior pituitary failure. Early diagnosis, comprehensive endocrine evaluation, and timely sequential hormone replacement resulted in excellent clinical and biochemical recovery.

Case Presentation

A 30-year-old woman (gravida 2, para 2) with no previous history of diabetes mellitus, hypertension, thyroid disease, pituitary disorders, or other significant endocrine illness underwent an uncomplicated full-term vaginal delivery at a peripheral healthcare center. The immediate postpartum period was complicated by severe postpartum hemorrhage secondary to uterine atony, with an estimated blood loss of approximately 2.5 L. She developed hemorrhagic shock, with a recorded blood pressure of 70/40 mmHg, tachycardia (132 beats/min), altered sensorium, and peripheral hypoperfusion. Emergency management included aggressive intravenous crystalloid resuscitation, uterotonic agents, uterine tamponade, transfusion of five units of packed red blood cells, four units of fresh frozen plasma, one unit of platelet concentrate, and short-term vasopressor support. Following hemodynamic stabilization, she recovered without apparent neurological deficits and was discharged in stable condition after an uneventful hospital stay.

Approximately 10 weeks after delivery, she presented to our institution with progressive generalized fatigue, profound weakness, inability to lactate since childbirth, secondary amenorrhea, anorexia, constipation, postural dizziness, cold intolerance, and an unintentional weight loss of approximately 5 kg. During the preceding 2 weeks, she had developed excessive thirst and progressive polyuria, consuming nearly 8–10 L of water daily and passing an estimated 9–10 L of urine per day. She also reported frequent nocturia, dry mouth, and symptoms suggestive of progressive dehydration. There was no history of fever, dysuria, recurrent urinary tract infection, osmotic symptoms suggestive of diabetes mellitus, diuretic use, chronic kidney disease, psychiatric illness, or excessive voluntary water intake.

On physical examination, the patient was conscious, alert, and oriented. Her blood pressure was 88/56 mmHg with significant postural hypotension, pulse rate was 104 beats/min, and body mass index was 21 kg/m². She appeared dehydrated, with dry oral mucosa, reduced skin turgor, pallor, and complete absence of breast milk secretion. Cardiovascular, respiratory, abdominal, and neurological examinations were otherwise unremarkable.

Initial laboratory investigations demonstrated mild anemia (hemoglobin 9.2 g/dL) with normal leukocyte and platelet counts. Serum sodium was elevated at 154 mmol/L, accompanied by increased serum osmolality (314 mOsm/kg), while urine osmolality was markedly reduced (92 mOsm/kg) with a urine specific gravity of 1.002, indicating hypotonic polyuria. Random blood glucose, renal function tests, and serum potassium were within normal limits, making osmotic diuresis and intrinsic renal disease less likely. Comprehensive endocrine evaluation revealed panhypopituitarism, including secondary adrenal insufficiency, central hypothyroidism, hypogonadotropic hypogonadism, growth hormone deficiency, and markedly reduced serum prolactin levels. A formal water deprivation test demonstrated persistent excretion of dilute urine despite rising plasma osmolality, whereas administration of desmopressin resulted in a marked increase in urine osmolality, confirming the diagnosis of central diabetes insipidus. Magnetic resonance imaging of the hypothalamic–pituitary region demonstrated a partially empty sella, marked anterior pituitary atrophy, thinning of the pituitary stalk, and absence of the normal posterior pituitary bright spot, without evidence of a pituitary adenoma or other sellar mass lesion.

Based on the antecedent history of severe postpartum hemorrhage, characteristic postpartum endocrine manifestations, biochemical evidence of panhypopituitarism, dynamic confirmation of central diabetes insipidus, and supportive MRI findings, a diagnosis of Sheehan syndrome with panhypopituitarism presenting as central diabetes insipidus was established. Differential diagnoses considered included gestational diabetes insipidus, lymphocytic hypophysitis, pituitary apoplexy, infiltrative hypothalamic–pituitary disorders, and primary polydipsia; however, these were considered less likely in view of the patient's obstetric history, hormonal profile, imaging findings, and response to desmopressin therapy.[1–5]

Management and Clinical Outcome

The patient was admitted for urgent stabilization with careful correction of dehydration and electrolyte imbalance while simultaneously evaluating the underlying endocrine abnormalities. Initial management consisted of intravenous isotonic fluid replacement with close monitoring of urine output, serum sodium concentration, serum osmolality, and hemodynamic status. Because biochemical evaluation demonstrated secondary adrenal insufficiency, glucocorticoid replacement was initiated immediately before thyroid hormone therapy in accordance with current endocrine recommendations to prevent precipitation of adrenal crisis.[3,9]

Intravenous hydrocortisone (100 mg) was administered as an initial bolus, followed by 50 mg every 6 hours for the first 24 hours. After clinical stabilization, the patient was transitioned to physiological oral hydrocortisone replacement (10 mg in the morning, 5 mg in the afternoon, and 5 mg in the evening). Following adequate glucocorticoid replacement and improvement in blood pressure, oral levothyroxine was initiated at a dose of 50 µg/day and gradually titrated to 100 µg/day over the subsequent four weeks based on clinical response and serial free thyroxine measurements.

Persistent hypotonic polyuria despite correction of intravascular volume, together with a positive water deprivation test, confirmed central diabetes insipidus. Oral desmopressin was therefore commenced at a dose of 60 µg twice daily, resulting in a rapid reduction in urine output and marked improvement in thirst. The patient also received oral iron supplementation for anemia, calcium and vitamin D supplementation, nutritional counseling, and detailed education regarding lifelong hormone replacement therapy. She was instructed on stress-dose glucocorticoid administration during intercurrent illness, the importance of adherence to replacement therapy, and the need for regular endocrinology follow-up.

A significant clinical response was observed within the first 72 hours of treatment. Daily urine output decreased from approximately 9.5 L to nearly 2 L, excessive thirst resolved, serum sodium returned to the normal range, and postural hypotension improved substantially. The patient reported increased energy levels, improved appetite, and resolution of dizziness, allowing discharge in a clinically stable condition with scheduled endocrine follow-up.

At the six-week review, she reported complete resolution of polyuria, polydipsia, nocturia, fatigue, and postural symptoms. Repeat laboratory investigations demonstrated normalization of serum sodium (140 mmol/L), improvement in serum osmolality (287 mOsm/kg), increased urine osmolality (620 mOsm/kg), stabilization of urine output at approximately 1.8 L/day, and improvement in hemoglobin concentration to 11.8 g/dL. Free thyroxine levels normalized with appropriate replacement, while thyroid-stimulating hormone remained suppressed, consistent with central hypothyroidism. Blood pressure remained stable without orthostatic hypotension, and electrolyte abnormalities had completely resolved.

At the six-month follow-up, the patient remained clinically stable on maintenance therapy with oral hydrocortisone, levothyroxine, and desmopressin. She had experienced no episodes of adrenal crisis, recurrent dehydration, or relapse of polyuria and had resumed normal daily activities with a satisfactory quality of life. Continued endocrinology follow-up was planned for long-term monitoring of pituitary hormone replacement, dose adjustment, and surveillance for potential complications associated with chronic hypopituitarism.

DISCUSSION

Sheehan syndrome is an uncommon cause of hypopituitarism resulting from ischemic necrosis of the pituitary gland following severe postpartum hemorrhage. Although advances in obstetric care have reduced its incidence, delayed diagnosis remains common, particularly in resource-limited settings where postpartum endocrine symptoms may be overlooked.[1,2]

Our patient developed the classical manifestations of anterior pituitary failure, including agalactia, secondary amenorrhea, fatigue, postural hypotension, and cold intolerance, approximately 10 weeks after severe postpartum hemorrhage. However, the most striking feature was the presence of central diabetes insipidus (CDI), manifested by profound polyuria, polydipsia, hypernatremia, low urine osmolality, and an excellent response to desmopressin. Posterior pituitary involvement in Sheehan syndrome is exceptionally rare because the neurohypophysis receives a direct arterial blood supply from the inferior hypophyseal arteries, making it less susceptible to ischemic injury than the anterior pituitary.[3,4]

The diagnosis of CDI in this patient was supported by biochemical evidence of hypotonic polyuria, a positive water deprivation test, and characteristic MRI findings demonstrating partial empty sella, anterior pituitary atrophy, and absence of the posterior pituitary bright spot. The coexistence of panhypopituitarism and CDI suggests extensive ischemic damage involving both the adenohypophysis and neurohypophysis. Similar presentations have been described only in isolated case reports, emphasizing the rarity of this clinical entity.[5,7]

This case also highlights important diagnostic challenges. Polyuria during the postpartum period is frequently attributed to uncontrolled diabetes mellitus, gestational diabetes insipidus, excessive fluid intake, or renal disease, potentially delaying recognition of underlying pituitary dysfunction. A careful obstetric history, particularly previous severe postpartum hemorrhage, together with the presence of lactation failure and amenorrhea, provides important clues that should prompt comprehensive endocrine evaluation.[6,8]

Management of Sheehan syndrome requires sequential hormone replacement. Glucocorticoid therapy should always precede thyroid hormone replacement to avoid precipitating adrenal crisis.[3] Once

adrenal insufficiency and central hypothyroidism are adequately treated, desmopressin effectively controls vasopressin deficiency in patients with confirmed CDI. Our patient demonstrated rapid clinical improvement following initiation of hydrocortisone, levothyroxine, and desmopressin, with normalization of serum sodium, urine output, and functional status during follow-up.

In conclusion, this case underscores that persistent polyuria and polydipsia after severe postpartum hemorrhage should not be attributed solely to common postpartum conditions. Although rare, CDI may occur in Sheehan syndrome and should be considered when hypotonic polyuria coexists with features of anterior pituitary failure. Early recognition, appropriate endocrine evaluation, and prompt sequential hormone replacement are essential to reduce morbidity and improve long-term outcomes.

CONCLUSION

Sheehan syndrome remains an important but frequently under-recognized cause of postpartum hypopituitarism. Although anterior pituitary dysfunction is the hallmark of the disease, central diabetes insipidus is an exceptionally rare manifestation that may delay diagnosis if not actively considered. This case emphasizes the importance of obtaining a detailed obstetric history in women presenting with persistent polyuria, polydipsia, lactation failure, amenorrhea, and other symptoms of hypopituitarism following severe postpartum hemorrhage. Early endocrine evaluation, appropriate neuroimaging, and prompt initiation of sequential hormone replacement therapy are essential to prevent life-threatening complications and achieve favorable clinical outcomes. Increased awareness of this rare presentation may facilitate earlier diagnosis and improve long-term prognosis in affected patients.

Learning Points

Severe postpartum hemorrhage should prompt long-term surveillance for delayed hypopituitarism.

Persistent postpartum polyuria and polydipsia should raise suspicion for central diabetes insipidus, particularly when accompanied by agalactia and amenorrhea.

Central diabetes insipidus is a rare manifestation of Sheehan syndrome and indicates extensive pituitary injury.

Glucocorticoid replacement must precede thyroid hormone replacement to prevent adrenal crisis.
Early recognition and timely hormone replacement therapy result in excellent clinical recovery and improved quality of life.

Ethics Statement

Ethical approval was not required for this case report in accordance with the institutional policy, as it describes a single patient and all identifying information has been removed to preserve patient confidentiality.

Conflict of Interest

The authors declare that they have no competing interests.

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Author Contributions

Dr Anupreetta M H: Conceptualization, patient evaluation, data collection, literature review, manuscript drafting, and final manuscript approval.
Dr Hemamalini Rajamanikkam: Clinical supervision, critical revision of the manuscript, interpretation of clinical findings, and final manuscript approval.
Both authors have read and approved the final manuscript.

Data Availability Statement

All data generated or analyzed during this study are included in this published article. Additional information is available from the corresponding author upon reasonable request while maintaining patient confidentiality.

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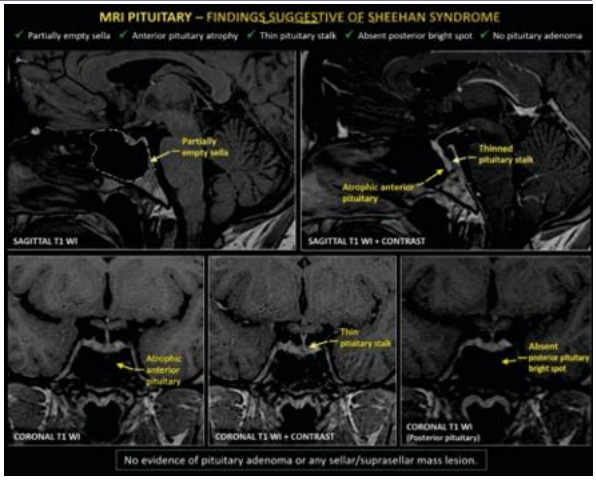

Figure 1. Magnetic Resonance Imaging of the Sellar Region Demonstrating a Partially Empty Sella with Marked Anterior Pituitary Atrophy, Thinning of The Pituitary Stalk, And Absence of the Normal Posterior Pituitary Bright Spot, Consistent with Sheehan Syndrome Involving Both the Anterior and Posterior Pituitary.

Parameter	Before	6 Weeks After
Hemoglobin (g/dL)	9.2	11.8
Serum Sodium (mmol/L)	154	140
Serum Osmolality (mOsm/kg)	314	287
Urine Osmolality (mOsm/kg)	92	620
Urine Output (L/day)	9.5	1.8
Free Thyroxine (ng/dL)	0.42	1.21
Blood Pressure (mmHg)	88/56	118/76

Figure 2. Results of Endocrine Investigations Demonstrating Biochemical Evidence of Panhypopituitarism and Hypernatremic Hypotonic Polyuria, Including Low Anterior Pituitary Hormone Levels, Elevated Serum Osmolality, Reduced Urine Osmolality, and Findings Consistent with Central Diabetes Insipidus.

Abbreviations

CDI – Central diabetes insipidus
MRI – Magnetic resonance imaging
PPH – Postpartum hemorrhage
FFP – Fresh frozen plasma
ACTH – Adrenocorticotropic hormone
TSH – Thyroid-stimulating hormone
FT4 – Free thyroxine
PRBC – Packed red blood cells

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