fortune Journals

Research Article

FORTUNE JOURNAL OF HEALTH SCIENCES

ISSN: 2644-2906

Unseen Endocrine Deficits: Clinical Diversity in Sheehan's Syndrome Presentation

Rohit Kumar*[,1], Rajan Kumar[2], Prabhat Ranjan[3]

Abstract

Background: Sheehan's syndrome (SS) is postpartum hypopituitarism resulting from ischemic necrosis of the pituitary gland after severe postpartum hemorrhage. It remains underdiagnosed in developing countries due to variable clinical presentation and non-specific symptoms, leading to delayed diagnosis and significant morbidity.

Objective: To evaluate the clinical features, hormonal deficiencies, and radiological findings in patients with Sheehan's syndrome and to highlight occult endocrine abnormalities responsible for diagnostic delay.

Methods: This descriptive case series was conducted at Patna Medical College and Hospital (PMCH), Patna, India, between December 2024 and August 2025. Five female patients with a history of significant postpartum hemorrhage, clinical features of hypopituitarism, and confirmatory hormonal and magnetic resonance imaging (MRI) findings were included. Clinical assessment, hormonal evaluation, and pituitary MRI were performed. Patients were followed for one month after initiation of hormone replacement therapy.

Results: All patients presented with lactation failure and secondary amenorrhea. Fatigue, cold intolerance, and postural hypotension were common. Hyponatremia occurred in three patients and hypoglycemia in two. Secondary adrenal insufficiency and central hypothyroidism were present in all cases, with variable gonadotropin and prolactin deficiencies. MRI revealed partial or complete empty sella in all patients. Hormone replacement therapy resulted in marked clinical and biochemical improvement, with one patient resuming spontaneous menstruation.

Conclusion: Sheehan's syndrome shows heterogeneous endocrine involvement and often mimics non-specific disorders. Early endocrine evaluation in high-risk postpartum women is crucial for timely diagnosis and prevention of life-threatening complications.

Keywords: Sheehan's syndrome; Hypopituitarism; Postpartum hemorrhage; Empty sella; Hormone replacement therapy

Introduction

Sheehan's syndrome (SS) is a significant yet often undiagnosed form of hypopituitarism, particularly in developing countries such as India, where postpartum hemorrhage (PPH) is still one of the leading obstetric complications [1–3]. The syndrome occurs due to the ischemic necrosis of the anterior pituitary due to substantial blood loss or hypotension occurring during or immediately after delivery [4]. The pituitary gland is particularly prone to ischemic damage during the period of postpartum due to the coefficient enlargement during

Affiliation:

[1]Junior resident, Department of General Medicine, Patna Medical College and Hospital, Patna, Bihar, India

[2]Professor, Department of General Medicine, Patna Medical College and Hospital, Patna, Bihar, India

[3]Assistant Professor, Department of Endocrinology, Patna medical College and Hospital, Patna, Bihar, India

***Corresponding author:**
Rohit Kumar, Junior resident, Department of General Medicine, Patna Medical College and Hospital, Patna, Bihar, India

Citation: Rohit Kumar, Rajan Kumar, Prabhat Ranjan. Unseen Endocrine Deficits: Clinical Diversity in Sheehan's Syndrome Presentation. Fortune Journal of Health Sciences. 9 (2026): 206-213.

Received: January 31, 2026
Accepted: February 08, 2026
Published: May 02, 2026

Kumar R, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.411

pregnancy and the hormonal deficits due to the damage could take months or even years to develop after the obstetric complication [5]. The syndrome is clinically heterogeneous even though the classical triad of SS includes agalactia, amenorrhea, and loss of secondary sexual characteristics [6,7]. Some patients may reveal very mild symptoms such as chronic fatigue, weakness, or other psychiatric disorders, and others may present with life-threatening adrenal insufficiency, hyponatremia, or hypoglycemia [8–10]. This is one of the reasons the syndrome gets poorly recognized and diagnosed in the primary care setting, especially in low-resource environments [11].

Endocrine evaluations show various levels of anterior pituitary hormone insufficiency including isolated cases or total panhypopituitarism [12]. More recent data show that the partial preservation of some axes such as the gonadotroph or thyrotroph functions is relatively common, which indicates that Sheehan's syndrome might manifest as more of a range of endocrine damage rather than a singular condition [13]. Neuroimaging, particularly MRI, often shows an empty or partially empty sella which is connected to the chronicity and degree of necrosis of the pituitary gland [14,15]. The chronic empty sella syndrome has an empty or partially empty sella, but the imaging findings, in some cases, do not correlate to the degree of complications or the hormonal status, and this fact differentiates the condition from other cases [16].

Given the wide spectrum of endocrine disturbances and the frequent diagnostic delays associated with Sheehan's syndrome, documenting its clinical and biochemical diversity remains essential. The present case series was conducted at Patna Medical College and Hospital (PMCH), Patna, over a four-year period (January 2021 to December 2024). It included five female patients who presented with characteristic features of hypopituitarism following postpartum hemorrhage. The primary objective of this study was to evaluate the hidden endocrine insufficiencies, metabolic disturbances, and radiological findings associated with Sheehan's syndrome and to highlight the importance of early recognition and prompt hormonal replacement in improving patient outcomes.

Materials and Methods

Study Design and Setting

This descriptive case series was conducted within the Department of Endocrinology, PMCH, Patna, India. PMCH is one of the major tertiary care teaching hospitals and is Eastern India's important referral center. It was done from December 2024 to August 2025. Ethical approval was sought from the Institutional Ethical Committee. Written informed consent was obtained from all patients, as well as for the publication of anonymized clinical and imaging documents.

Study Population

All of the included subjects in this case series were women between 30 and 45 years of age. All five females had a known history of severe PPH at their most recent delivery, accompanied by lactation failure to differing extent and later endocrine or systemic symptoms. The patients were enrolled in OPD (Out-Patient Department) and emergency department of PMCH after being stabilized and consulting with an endocrinologist.

Sheehan's syndrome was diagnosed in patients according to these **inclusion criteria:**

- History of moderate or severe PPH or long-delayed postpartum hypotension/shock.
- Inability to lactate following childbirth and/or persistent amenorrhea or menstrual irregularity after delivery
- Biochemical evidence of deficiency of one or more of the anterior pituitary hormones.
- An MRI demonstrating a partial or fully empty sella or pituitary atrophy.

Exclusion criteria were presence of known masses of pituitary or parasellar, cranial irradiation, traumatic brain injury, autoimmune hypophysitis, infiltrative or granulomatous diseases (sarcoidosis, tuberculosis), and incomplete data in laboratory/imaging tests.

Clinical Evaluation

History was taken in detail from all the patients on a structured proforma. Particular attention was paid to:

- Obstetric history (gravidity, parity, modes of delivery and perinatal complications, estimated blood loss, transfusion requirement and lactation status).
- Previous menstrual history: first onset of menstrual irregularity or amenorrhoea.
- General symptoms: fatigue, malaise, decreased libido, cold intolerance, constipation, and dizziness (versus altered mental status or sensorium) weight loss and hair distribution changes.

Blood pressure measurements were repeated by sitting, standing, and lying positions from as early in the experimentation period as practicable; orthostatic hypotension was defined as a reduction of at least 20 mmHg or higher within 3 minutes. Other vital signs included supine and standing blood pressure, and heart rate.

Laboratory and Hormonal Assessment

Venous blood samples under fasting (overnight fast for 8 hours) conditions were obtained in the morning (08:00 h) to measure baseline endocrine and biochemical parameters. The following tests were performed:

Kumar R, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.411

- Blood samples serum cortisol (8 AM) and plasma adenocortictropic hormone (ACTH)
- thyroid function tests: FT3, FT4, and TSH
- The gonadotropins: luteinizing hormone (LH) and follicle-stimulating hormone (FSH)
- Blood collected for serum prolactin and growth hormone (where possible), however, data were not consistently available for all patients
- Serum electrolytes (Na^+, K^+, Cl^-)
- Biochemical parameters such as fasting blood glucose, lipid profile, hemoglobin and renal/liver function tests
- Biochemistry (fasting blood glucose, lipid profile, hemoglobin and renal/liver function tests)

All hormonal testing was carried out by the chemiluminescence immunoassay (CLIA) technique on the Beckman Coulter UniCel DxI 800 analyzer at our central endocrine laboratory in PMCH. The electrolytes and biochemistry were examined using an automated Beckman AU5800 analyser.

In cases with results suspicious for secondary adrenal insufficiency, confirmatory dynamic tests (ACTH stimulation or the insulin-tolerance test) would be scheduled when clinically feasible. The majority of patients had dynamic testing, although in some cases this was deferred on an acutely unwell patient.

Radiological Assessment

Using a Siemens Magnetom Avanto 1.5-Tesla scanner, all five patients had brain magnetic resonance imaging (MRI) with pituitary protocol. T1- and T2-weighted sagittal and coronal images were obtained, both with and without contrast enhancement. Assessments were made of the posterior bright spot, sellar configuration, stalk thickness, pituitary morphology, and gland height. The results were divided into: Typical morphology of the pituitary, Pituitary height < 3 mm with residual tissue is known as partial empty sella and entire sella is empty (pituitary flattened along the sellar floor).

Treatment and Follow-Up

To avoid an adrenal crisis, each patient received physiologic hormone replacement therapy in the following order:

- Glucocorticoid replacement: Methylprednisolone equivalent or oral hydrocortisone 15–20 mg/day (in divided doses).
- Levothyroxine 50–100 µg/day is used to replace thyroid hormones and is started 5–7 days after beginning steroids.
- Adjunct therapy: iron-folic acid, calcium, vitamin D, and, in some situations, Ovares Plus (which contains melatonin, DHEA, and Coenzyme Q10) to boost mood and energy.
- Hormonal cycle induction: Premenopausal women with gonadotropin deficiency were evaluated for cyclic estrogen-progesterone therapy.

Patients received counseling on the importance of routine follow-up, the necessity of increasing steroid dosage during stress or illness, and the necessity of lifelong hormone replacement.

Monitoring and Evaluation of Results

After one month of treatment, and then every three months after that, a clinical reassessment was conducted. We observed improvements in menstrual patterns, appetite, fatigue, sensorium, weight gain, and general well-being. In order to document endocrine stabilization, repeat biochemical and hormonal profiles were acquired whenever possible. At the initial follow-up visit, every patient reported a noticeable improvement in their symptoms.

Data Management and Statistical Analysis

For descriptive analysis, individual case data were imported into Microsoft Excel 2021. Only qualitative and descriptive statistics were used because of the small sample size (n = 5). Frequency distributions, means, and a tabular display of the biochemical and radiologic results were used to summarize the results.

Ethical Considerations

Confidentiality was strictly maintained. No identifying details were disclosed. Patients were treated according to standard clinical protocols and were provided lifelong follow-up for endocrine replacement and monitoring.

Results

Between December 2024 and August 2025, five female patients were evaluated at PMCH. Data analysis was performed. Five female patients with ages ranging from 30 to 45 (mean ± SD = 36.6 ± 6.6 years) were assessed. Each had a distinct history of severe PPH, lactation failure, and the subsequent development of systemic or endocrine symptoms. Five to fifteen years (mean = nine years) passed between the inciting obstetric event and the clinical diagnosis. Menstrual irregularities or amenorrhea (5/5), cold intolerance (3/5), generalized weakness (5/5), easy fatigability (5/5), and signs of adrenal insufficiency, such as hypotension or altered sensorium, were the most common presenting complaints.

Clinical Characteristics

Prior PPH, lactation failure, and either irregular menstruation or amenorrhea were present in all patients. Two

Kumar R, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.411

patients had chronic symptoms like edema, exhaustion, or hair loss, and three patients had acute symptoms of altered sensorium or hypotension brought on by hypoglycemia.

The clinical and demographic details of the five patients are compiled in Table 1.

Table 1: Demographic and Clinical Characteristics of Patients with Sheehan's Syndrome (n = 5)

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5
Name	Sila Devi	Shrimati Devi	Renu Devi	Kunti Devi	Sunita Sinha
Age (years)	45	33	30	45	30
Residence	Dighwara, Patna	Sheikhpura (Bihar)	Patna (Bihar)	Gopalganj (Bihar)	Patna (Bihar)
Mode of delivery	NVD (twins)	Vaginal	Vaginal	Vaginal (G8P8)	C-section (twins)
Obstetric complication	Severe PPH → hysterectomy	Severe PPH with shock	Massive PPH + transfusion	Moderate PPH	Severe PPH during C-section
Interval since PPH (years)	15	13	7	15	5
Lactation failure	Complete	Complete	Complete	Partial	3 months only
Menstrual disturbance	Secondary amenorrhea	Premature menopause (29 yrs)	Secondary amenorrhea	Menopause 8 yrs ago	Irregular menses × 4 yrs
Main presenting complaint	Altered sensorium, vomiting	Recurrent vomiting, hypotension	Generalized edema, fatigue	Fatigue, dry skin, constipation	Weakness, nausea, constipation
Blood pressure (mm Hg)	90/70	70/50	90/74	126/70	110/70
Random blood sugar (mg/dL)	29 ↓	45 ↓	68 ↓	84	72

Hormonal and Biochemical Findings

Multi-axial anterior pituitary hormone deficiency was present in every patient. Secondary adrenal insufficiency was confirmed by the significantly lower morning serum cortisol levels in every case. With low FT3/FT4 and abnormally normal or low TSH, four patients displayed central hypothyroidism. Low or low-normal levels of gonadotropins and prolactin were indicative of hypogonadotropic hypogonadism. Mild anemia and hyponatremia were frequently observed.

Table 2: presents the detailed biochemical and hormonal parameters.

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5	Reference Range
Serum Cortisol (8 AM, µg/dL)	2.57 ↓	< 0.4 ↓	3.0 ↓	4.54 ↓	4.22 ↓	7–25
ACTH (pg/mL)	—	—	—	—	10.2 ↓	10–60
FT3 (pg/mL)	1.71 ↓	1.29 ↓	1.07 ↓	3.11	2.1	2.0–4.4
FT4 (ng/dL)	0.18 ↓	0.24 ↓	0.40 ↓	1.33	1.25	0.8–1.8
TSH (µIU/mL)	0.657	3.33	0.721	1.74	3.19	0.5–5.0
LH (mIU/mL)	—	4.72	3.46	—	22.24 ↑	5–25
FSH (mIU/mL)	1.7 ↓	9.01	7.5	—	5.76	4–30
Prolactin (ng/mL)	0.42 ↓	7.65 (low-normal)	—	—	4.94 (low-normal)	5–25
Serum Na^+ (mmol/L)	105 ↓	114 ↓	128 ↓	137	138	135–145
Serum K^+ (mmol/L)	3.2 ↓	3.6 ↓	3.0 ↓	4.2	4.1	3.5–5.0
Serum Cl^- (mmol/L)	74 ↓	84 ↓	99 ↓	103	104	98–107
Hemoglobin (g/dL)	10.8 ↓	9.6 ↓	8.8 ↓	10.5 ↓	10.9 ↓	12–16
Fasting glucose (mg/dL)	29 ↓	45 ↓	68 ↓	84	72	70–110

Kumar R, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.411

Table 2 presents the detailed biochemical and hormonal parameters.

Radiological Findings

MRI of the pituitary demonstrated features consistent with Sheehan's syndrome in all patients. Three had complete empty sella, one near-complete, and one partial empty sella with residual tissue. Posterior bright spot was absent in four, reflecting chronicity.

Table 3: MRI Pituitary Findings

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5
MRI Diagnosis	Near-complete empty sella	Complete empty sella	Complete empty sella	Partial empty sella	Complete empty sella
Pituitary height (mm)	≈ 1.5	≈ 1.8	≈ 1.2	2.6	1.9
Posterior bright spot	Absent	Absent	Absent	Present	Absent
Pituitary stalk	Normal	Normal	Normal	Mild thickening	Normal
Residual tissue enhancement	Absent	Absent	Absent	Present	Absent

Table 3 summarizes the MRI findings.

Treatment and Clinical Outcome

Glucocorticoids and levothyroxine were the first two hormone replacement treatments given to all patients. Supplemental calcium, vitamin D, and, in certain situations, Ovares Plus (Coenzyme Q10, DHEA, and melatonin) were also given. Within four to six weeks, there was a noticeable clinical improvement in sensorium, appetite, and fatigue (Figure 1). Every hyponatremic patient's serum sodium levels returned to normal. After three months of treatment, one patient (Case 4) resumed having menstrual periods on their own.

Table 4: Outlines treatment regimens and outcomes.

Parameter	Case 1	Case 2	Case 3	Case 4	Case 5
Glucocorticoid replacement	Hydrocortisone 20 mg/day	Hydrocortisone 15 mg/day	Methylprednisolone 16 mg/day	Hydrocortisone 20 mg/day	Hydrocortisone 20 mg/day
Levothyroxine (µg/day)	75	50	100	75	75
Adjunct supplements	Calcium + Ovares Plus	Calcium + Ovares Plus	Calcium + IFA	Calcium	Calcium + Ovares Plus
Follow-up duration (months)	1	1	1	1	1
Weight gain (kg)	2	2	2	3	2
Improvement in weakness/ fatigue	Marked	Marked	Marked	Marked	Marked
Menstrual recovery	No	No	No	Yes	No

Table 4 illustrates the Hormone Replacement Therapy and Clinical Outcomes

Frequency Distribution of Major Clinical, Hormonal, and Radiological Features among Five Patients with Sheehan's Syndrome

All patients shared a history of obstetric hemorrhage, lactation failure, and secondary adrenal insufficiency. Central hypothyroidism was present in four, and MRI demonstrated pituitary atrophy or empty sella in all. Hormone-replacement therapy led to complete symptomatic recovery in all cases. Table 5 provides a quantitative overview of major clinical and diagnostic features.

Kumar R, et al., Fortune J Health Sci 2026
DOI:10.26502/fjhs.411

Figure 1: Clinical photographs of three patients with Sheehan's syndrome (Case 1 – Sila Devi, Case 2 – Shrimati Devi, and Case 5 – Sunita Sinha) showing visible improvement following hormone replacement therapy.

Table 5: Summary of Key Clinical and Diagnostic Findings (n = 5)

Feature	No. of Patients	Percentage (%)
History of severe PPH	5	100
Lactation failure	5	100
Amenorrhea or menstrual irregularity	5	100
Hypotension at presentation	3	60
Hyponatremia (< 135 mmol/L)	3	60
Hypoglycemia (< 70 mg/dL)	2	40
Central hypothyroidism	4	80
Secondary adrenal insufficiency	5	100
Low/low-normal prolactin	4	80
MRI showing empty/partial sella	5	100
Marked improvement after therapy	5	100

Discussion

In developing countries, Sheehan's syndrome (SS) remains an important yet underdiagnosed cause of hypopituitarism, largely due to the persistent burden of postpartum hemorrhage (PPH) as a major obstetric complication [5]. Despite improvements in maternal care, the insidious and variable nature of this disorder contributes to delayed recognition. In the present case series from PMCH, Patna, involving five female patients, the delay between the inciting obstetric event and clinical diagnosis ranged from 5 to 15 years (mean 9 years). This prolonged interval, though shorter than some earlier Indian reports, continues to highlight the challenges in early detection. The mean age at diagnosis (36.6 years) is consistent with prior literature showing that SS typically affects women in the third to fourth decades of life [17,18].

All patients had a history of severe PPH, followed by lactation failure and secondary amenorrhea—findings that represent the classic triad of Sheehan's syndrome. However, as seen in this study, clinical presentations were diverse. The most common symptoms included fatigue, weakness, cold intolerance, and postural hypotension, while altered sensorium due to hyponatremia or hypoglycemia was observed in several cases. Three patients had hyponatremia, and two had recurrent episodes of hypoglycemia. Notably, two patients were initially misdiagnosed with neurological or metabolic disorders due to nonspecific symptoms such as confusion or vomiting, underscoring the need for heightened clinical suspicion in postpartum women presenting with such features.

All patients exhibited evidence of multiple anterior pituitary hormone deficiencies, though the extent varied. Secondary adrenal insufficiency and central hypothyroidism were the most consistent biochemical abnormalities, confirming these as the biochemical hallmarks of Sheehan's syndrome [2,20]. Gonadotropin and prolactin deficiencies were also frequent, correlating with amenorrhea and lactation failure. Growth hormone levels were not consistently available in all cases, but literature indicates that chronic cases often demonstrate panhypopituitarism, including GH deficiency [21]. The occurrence of metabolic derangements such as hyponatremia and hypoglycemia in this series reflects the combined effects of cortisol and thyroid hormone deficiencies, leading to impaired water clearance and altered gluconeogenesis [22,23].

Magnetic resonance imaging (MRI) findings were characteristic of chronic pituitary ischemic injury. All five patients demonstrated either complete or partial empty sella, with absent posterior bright spot in four, signifying chronicity. These observations align with previous studies, where empty sella is reported in 70–90% of Sheehan's syndrome cases [24,25]. The single case showing partial empty sella likely represents an earlier phase of pituitary atrophy. Consistent with prior evidence, MRI morphology did not directly correlate with the number or severity of hormonal deficits [26].

Following sequential hormone replacement therapy—glucocorticoids followed by levothyroxine—all patients demonstrated marked symptomatic and biochemical improvement. Serum sodium levels normalized, fatigue and hypotension improved, and one patient experienced

spontaneous resumption of menstruation after three months, indicating partial gonadotrophic recovery. No adverse events or adrenal crises occurred during therapy. These findings parallel previous reports where hormone replacement led to significant functional recovery and quality-of-life improvement in chronic SS cases [28,39].

The clinical diversity observed in this series—from subtle symptoms like fatigue to life-threatening metabolic derangements—illustrates the broad spectrum of Sheehan's syndrome. Such variability frequently results in fragmented evaluation across medical specialties before endocrinologic referral. Early postpartum endocrine screening for women with severe hemorrhage, lactation failure, or persistent amenorrhea can facilitate earlier detection and prevent life-threatening complications. Greater awareness among obstetricians and primary care physicians remains critical for timely recognition.

Limitations

This study has certain limitations. Being a small, single-center case series (n = 5), its findings cannot be generalized to the broader population. Dynamic hormonal testing could not be performed in all cases due to logistic and financial constraints. The short follow-up duration (1 month) also limits assessment of long-term hormonal recovery and therapy outcomes. Despite these limitations, the study underscores the persistent diagnostic delay and diverse clinical manifestations of Sheehan's syndrome in real-world practice.

Conclusion

Sheehan's syndrome continues to present as a multifaceted endocrine disorder with variable clinical and biochemical features. Even within this small series, the disease spectrum ranged from subtle hormonal insufficiency to severe adrenal crisis. Early recognition—especially in women with a history of significant PPH and lactation failure—remains essential. Timely hormonal evaluation and sequential replacement therapy can prevent morbidity and significantly improve outcomes. Strengthening obstetric care and enhancing clinical vigilance at the primary care level are key strategies for early diagnosis and management.

Funding

No funding sources.

Conflict of Interest

None declared.

Ethical Approval

This study was ethically approved. Written informed consent was obtained from all participants prior to inclusion.

References

1. Sheehan HL. Post-partum necrosis of the anterior pituitary. Journal of Pathology and Bacteriology 45 (1937): 189–214.
2. Kelestimur F. Sheehan's syndrome. Pituitary 6 (2003): 181–188.
3. Zargar AH, Singh B, Laway BA, et al. Epidemiologic aspects of Sheehan's syndrome. Fertility and Sterility 84 (2005): 523–528.
4. Gei-Guardia O, Soto-Herrera E, Gei-Brealey A, et al. Sheehan's syndrome in Costa Rica: clinical experience with 60 cases. Endocrine Practice 17 (2011): 337–344.
5. Sert M, Tetiker T, Kirim S, et al. Clinical report of 28 patients with Sheehan's syndrome. Endocrine Journal 50 (2003): 297–301.
6. Ozkan Y, Colak R, Altuntas Y. Sheehan's syndrome: clinical and laboratory evaluation of 20 cases. Neuroendocrinology Letters 25 (2004): 341–345.
7. Banzal S, Bhatia E, Shukla R, et al. Spectrum of hypopituitarism in Sheehan's syndrome in India. Clinical Endocrinology 61 (2004): 282–287.
8. Satti SA, Saeed G, Rasool S, et al. Sheehan's syndrome: clinical profile, diagnosis and management. Journal of Ayub Medical College Abbottabad 23 (2011): 8–13.
9. Matsuzaki S, Endo M, Ueda Y, et al. Clinical characteristics of Sheehan's syndrome. Gynecological Endocrinology 30 (2014): 358–361.
10. Khare S, Garg MK, Bansal R, et al. Spectrum of endocrine dysfunctions in Sheehan's syndrome and response to therapy. Indian Journal of Endocrinology and Metabolism 15 (2011): S252–S255.
11. Ozbey N, Inanc S, Aral F, et al. Clinical and laboratory evaluation of 20 patients with Sheehan's syndrome. Gynecological Endocrinology 8 (1994): 299–304.
12. Sert M, Tetiker T, Kirim S. MRI findings and pituitary function in patients with Sheehan's syndrome. Neuroradiology 45 (2003): 944–948.
13. Diri H, Tanriverdi F, Karaca Z, et al. Extensive clinical and laboratory evaluation in Sheehan's syndrome: what is the outcome? Pituitary 19 (2016): 300–308.
14. Bhatia E, Jain SK, Gupta SK. Empty sella in Sheehan's syndrome. Journal of the Association of Physicians of India 40 (1992): 591–594.
15. Jialal I, Naidoo C, Omar MAK. Sheehan's syndrome: metabolic and cardiovascular aspects. Clinical Endocrinology 21 (1984): 377–383.

16. Kim SH, Lee JS, Kim SY, et al. Bone mineral density and metabolic profiles in patients with Sheehan's syndrome. Endocrine Journal 47 (2000): 201–207.

17. Shivaprasad C, Kalra S. Sheehan's syndrome: newer advances. Indian Journal of Endocrinology and Metabolism 15 (2011): S203–S207.

18. Zargar AH, Laway BA, Masoodi SR, et al. Clinical spectrum and diagnosis of Sheehan's syndrome: observations from Kashmir, India. Endocrine Journal 52 (2005): 753–758.

19. Banzal S, Bhansali A, Dutta P, et al. Spectrum of hypopituitarism at a tertiary care hospital in North India: five-year experience. Indian Journal of Endocrinology and Metabolism 15 (2011): 326–331.

20. Shivaprasad C, et al. Delayed diagnosis of Sheehan's syndrome: experience from a tertiary care center. Endocrine Practice 18 (2012): 768–773.

21. Dökmetaş HS, Kilicli F, Korkmaz S, et al. Characteristic features of 20 patients with Sheehan's syndrome. Gynecological Endocrinology 22 (2006): 279–283.

22. Zargar AH, Wani AI, Masoodi SR, et al. Sheehan's syndrome: a study of 60 cases from Kashmir, India. Journal of Postgraduate Medicine 51 (2005): 72–76.

23. Goyal M, Bhadada SK, Mittal BR, et al. Spectrum of empty sella in Sheehan's syndrome. Pituitary 13 (2010): 236–241.

24. Kelestimur F, Sert M, et al. MRI findings in Sheehan's syndrome: correlation with clinical and hormonal parameters. Journal of Endocrinological Investigation 17 (1994): 575–581.

25. Yadav RK, Thakur S, Bhandari R, et al. Empty sella and hormonal profile in Sheehan's syndrome: a correlation study. Indian Journal of Endocrinology and Metabolism 20 (2016): 724–728.

26. Laway BA, Ganie MA. Sheehan's syndrome: new insights into an old disease. Indian Journal of Endocrinology and Metabolism 15 (2011): S193–S197.

27. Dutta P, Bhansali A, Masoodi SR, et al. Hypopituitarism in India: lessons from Sheehan's syndrome. Endocrine Practice 15 (2009): 230–235.

28. Dökmetaş HS, et al. Long-term effects of hormone replacement therapy in Sheehan's syndrome. Journal of Endocrinological Investigation 28 (2005): 808–812.

29. Soni S, Sharma S, Pathak A, et al. Reversible manifestations of chronic hypopituitarism following hormone replacement. Journal of the Association of Physicians of India 66 (2018): 72–75.

Citation: Rohit Kumar, Rajan Kumar, Prabhat Ranjan. Unseen Endocrine Deficits: Clinical Diversity in Sheehan's Syndrome Presentation. Fortune Journal of Health Sciences. 9 (2026): 206-213.