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CASE REPORT

A Case Report on Sheehan's Syndrome Presenting with Acute Decompensated Heart Failure and Type 3C Diabetes Mellitus

Hafsa Jabeen*[1], Ayesha Fatima[2]

[1] *Clinical Research Coordinator, Department of Pharmacy Practice, ClinArion Research Pvt. Ltd, Hyderabad, India*
[2] *Assistant Professor, Department of Pharmacy Practice, RBVRR Women's College of Pharmacy, Osmania University, Hyderabad, India*

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Abstract: A 27-year-old postpartum female presented to the emergency department with grade IV orthopnea, bilateral lower limb edema, and a history of severe hemorrhage during cesarean delivery. The patient had pre-existing type 3C diabetes mellitus (Fibro calculous Pancreatic Diabetes), asthma, and hypothyroidism. Laboratory investigations revealed profound anemia (Hb 6.6 g/dL), thrombocytopenia (91 × 10^9/L), acute kidney injury (creatinine 2.43 mg/dL), and marked endocrine abnormalities. Hormonal evaluation demonstrated significantly reduced free T3 (0.89 ng/dl) with concurrent signs of hypopituitarism. ECG showed extreme tachycardia with low-voltage limb leads. The diagnosis of Sheehan's syndrome was established based on the clinical presentation, postpartum hemorrhage history, and hormonal profile. The patient developed acute decompensated heart failure with pleural effusion, necessitating immediate medical intervention. Management included diuretic therapy, hormone replacement, and cardiovascular support. The patient showed gradual improvement with stabilization of vital parameters and reduction in peripheral edema by day 5 of hospitalization. Blood pressure normalized to 130/90 mmHg, and renal function showed progressive improvement. The case highlights the importance of early recognition of Sheehan's syndrome in postpartum women, particularly when presenting with multisystem involvement. Early treatment using hormone replacement therapy along with management of associated complications remains crucial for favorable outcomes.

Keywords: Sheehan's syndrome; Postpartum hypopituitarism; Type 3C diabetes mellitus; Acute decompensated heart failure; Hormone replacement therapy

1. Introduction

Sheehan's syndrome represents a significant endocrine disorder characterized by anterior pituitary necrosis following severe postpartum hemorrhage [1]. First described by Harold Leeming Sheehan in 1937, this condition manifests as partial or complete hypopituitarism resulting from ischemic pituitary necrosis [2]. The pathophysiological cascade typically initiates during pregnancy when physiological pituitary enlargement increases the gland's susceptibility to ischemic damage [3]. The epidemiology of Sheehan's syndrome has evolved significantly over the past decades. Historical data from the 1970s reported an incidence of 10-20 cases per 100,000 women. Contemporary studies indicate a marked reduction in developed nations, attributed to advanced obstetric care and improved management of postpartum hemorrhage [4]. However, in developing countries, particularly in rural areas with limited access to specialized obstetric care, the condition remains a significant concern. Indian epidemiological studies report a prevalence of 2.7-3.9% among parous women aged over 20 years [5].

During pregnancy, the pituitary gland undergoes substantial physiological changes, including a 36% volume increase primarily due to lactotroph hyperplasia [6]. This enlargement, coupled with the increased metabolic demands of pregnancy, creates a precarious situation where sudden hypotension or severe hemorrhage can precipitate ischemic damage. The enlarged gland's compressed vascular supply, particularly in the anterior portion, increases vulnerability to necrosis during hypotensive episodes [7]. The pathophysiological mechanisms extend beyond simple ischemia. Recent research has identified potential roles for autoimmune processes and genetic predisposition in disease development [8]. Cardiovascular implications in chronic Sheehan's syndrome include increased carotid intimal-medial thickness, elevated inflammatory markers, and clustering of cardiovascular risk factors [9]. Clinical manifestations typically follow a biphasic pattern. The acute phase presents immediately postpartum with failure to lactate, amenorrhea, and varying degrees of anterior pituitary hormone deficiencies [10]. The chronic phase may develop insidiously over months to years, characterized by progressive hypopituitarism symptoms including fatigue, cold intolerance, hypoglycemia, and secondary hypothyroidism [11]. Diagnosis requires a high index of clinical suspicion, particularly in cases presenting years after the inciting event. The diagnostic approach incorporates detailed obstetric history, hormonal evaluation, and imaging studies. Modern magnetic resonance imaging typically reveals an empty sella or partially empty sella, although findings may be subtle in early stages

* Corresponding author: Hafsa Jabeen

[12]. Treatment paradigms focus on comprehensive hormone replacement therapy, including thyroid hormones, corticosteroids, and gonadal steroids. Growth hormone replacement, while beneficial, requires careful consideration of cost-benefit ratios and potential side effects [13]. Long-term management necessitates regular monitoring of hormone levels and adjustment of replacement therapy to optimize clinical outcomes [14].

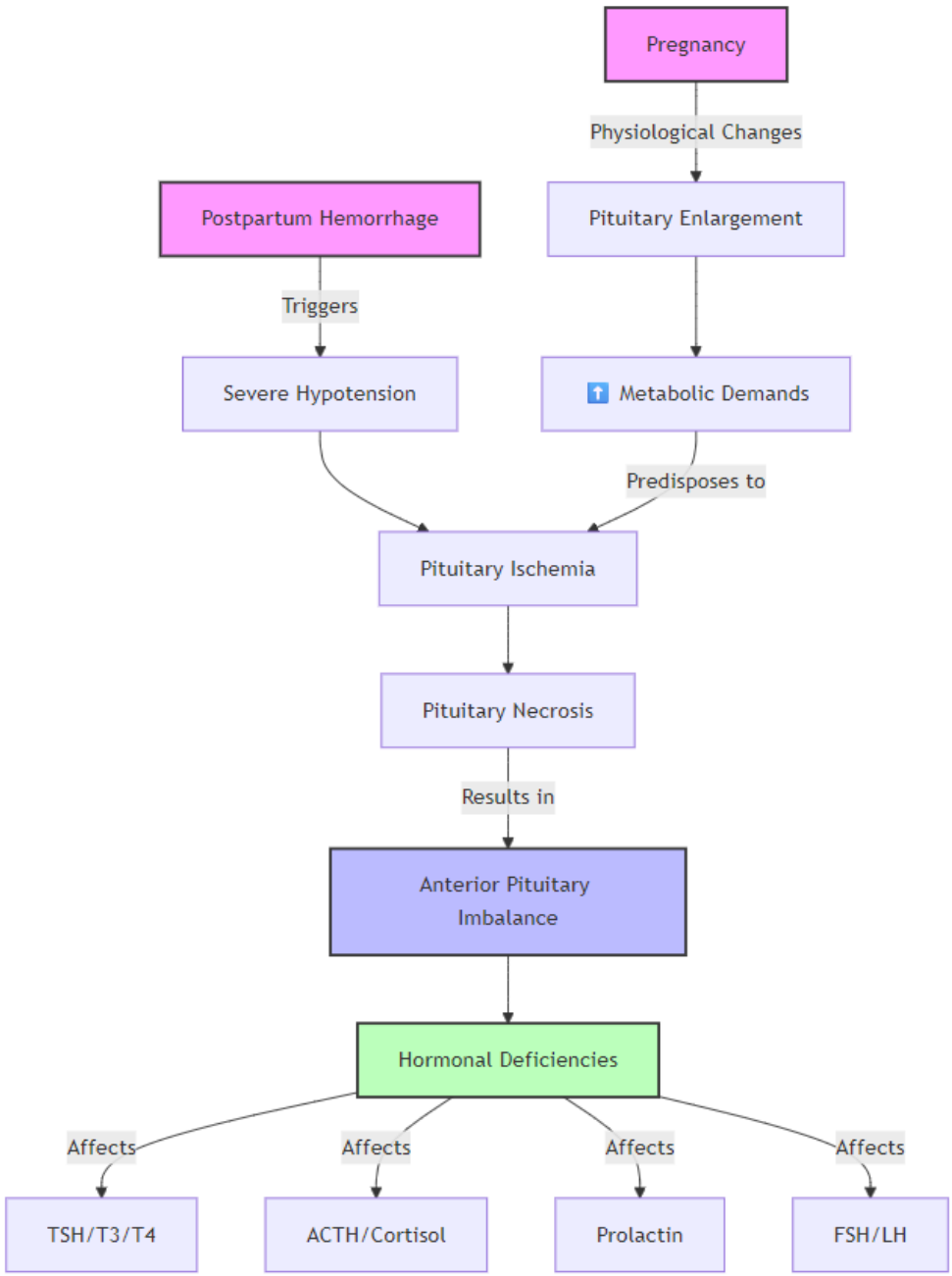

Figure 1. Pathophysiology of Sheehan's syndrome

2. Case Presentation

2.1. Initial Presentation

A 27-year-old female (P2 L1 D1) presented to the emergency department with progressive dyspnea evolving over 5-6 weeks, culminating in grade IV orthopnea. The patient reported bilateral lower limb edema persisting for one month and significant blood loss during her recent cesarean delivery. Her medical history was significant for Type 3C Diabetes mellitus (Fibro calculous Pancreatic Diabetes), asthma, and hypothyroidism, for which she was maintained on Thyronom 25 µg daily.

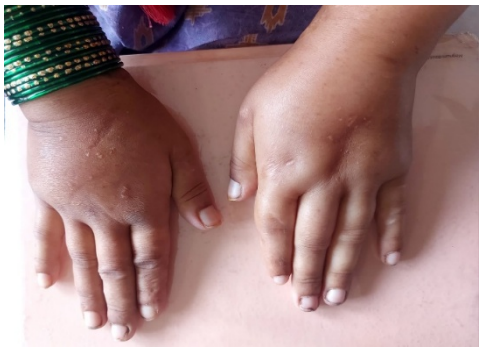
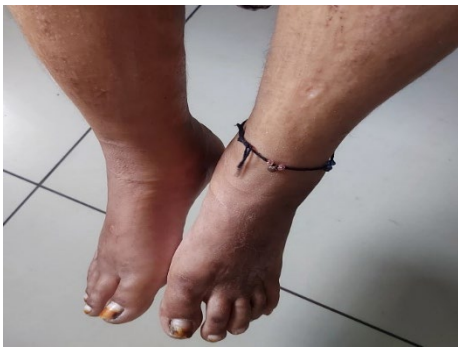

Figure 2. Patient shows physiological postpartum edema of upper and lower limb

Table 1. Laboratory Investigations during Initial Presentation

Parameter	Patient Value	Reference Range
Hemoglobin	6.6 g/dL	12.0-15.5 g/dL
RBC Count	2.45×10^{12}/L	$4.0\text{-}5.5 \times 10^{12}$/L
WBC Count	6.56×10^{9}/L	$4.0\text{-}11.0 \times 10^{9}$/L
Neutrophils	72.6%	40-70%
Platelets	91×10^{9}/L	$150\text{-}450 \times 10^{9}$/L
Serum Creatinine	2.43 mg/dL	0.6-1.2 mg/dL
Serum Urea	33.1 mg/dL	7-20 mg/dL
Serum Albumin	2.11 g/dL	3.5-5.5 g/dL

2.2. History

2.2.1. Previous Pregnancies

First gestation resulted in preterm normal vaginal delivery of a female infant who did not survive. Second gestation culminated in full-term lower segment cesarean section (LSCS) of a male infant three months prior to current presentation. Notable history of blood transfusion during the second delivery.

2.2.2. Present Parameters

Blood pressure monitoring revealed significant fluctuations over the initial five-day period

Table 2. Blood Pressure Monitoring During Initial Hospital Stay

Day	Morning (mmHg)	Evening (mmHg)
1	135/100	130/95
2	140/100	145/95
3	110/80	115/85
4	190/100	180/95
5	130/90	125/85

2.3. Clinical Investigations

2.3.1. Hematology

Initial laboratory evaluation revealed severe anemia (Hemoglobin 6.6 g/dL) with marked reduction in RBC count (2.45×10^{12}/L). White blood cell differential showed relative neutrophilia (72.6%) with normal total count (6.56×10^{9}/L). Significant thrombocytopenia was noted (91×10^{9}/L).

2.3.2. Biochemistry

Renal function tests indicated acute kidney injury with elevated serum creatinine (2.43 mg/dL) and urea (33.1 mg/dL). Liver function tests remained within normal limits except for hypoalbuminemia (2.11 g/dL). Electrolyte analysis revealed hyperkalemia (5.55 mEq/L) and hyperchloremia (120.9 mEq/L).

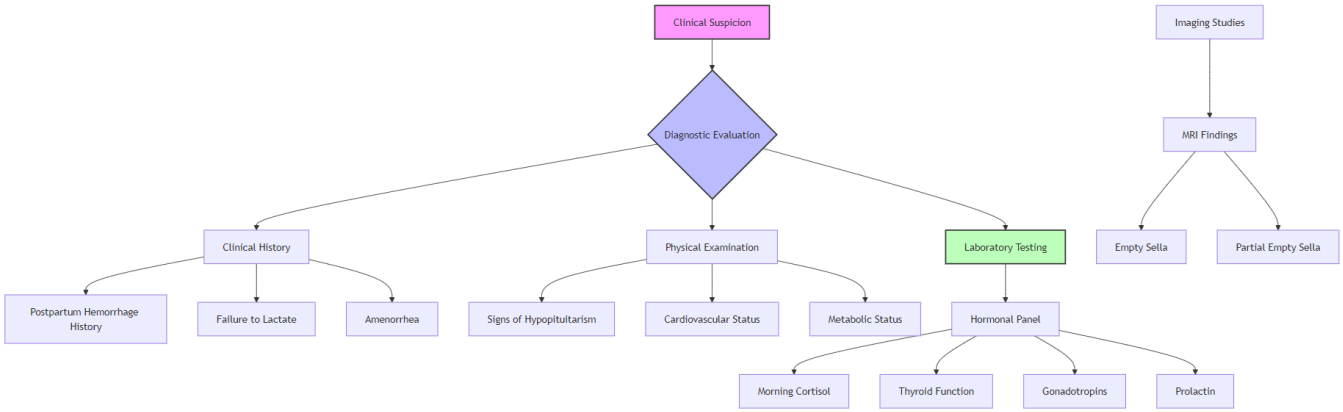

Figure 3. Diagnostic Work up for Sheehan's Syndrome

2.3.3. Lipid Profile

Notable findings included reduced total cholesterol (90.0 mg/dL), low HDL (36 mg/dL), and low LDL (40 mg/dL), suggesting metabolic derangement.

2.3.4. Endocrine Function

Thyroid function tests showed reduced Free T3 (0.89 ng/dl) with relatively normal TSH (3.935 mU/L), suggesting central endocrine dysfunction. Blood glucose monitoring revealed significant fluctuations (385 to 79 mg/dl).

Table 3. Hormonal Investigation

Hormone	Patient Value	Reference Range
Free T3	0.89 ng/dL	2.3-4.2 ng/dL
TSH	3.935 mU/L	0.4-4.0 mU/L
Cortisol (8 AM)	3.2 µg/dL	10-20 µg/dL
FSH	1.8 mIU/mL	4.7-21.5 mIU/mL
LH	0.5 mIU/mL	1.7-15.0 mIU/mL
Prolactin	2.1 ng/mL	4-23 ng/mL

2.3.5. Arterial Blood Gases

Revealed metabolic acidosis with pH 7.145, elevated pCO_2 (47.1 mmHg), and reduced pO_2 (41.3 mmHg).

2.3.6. ECG

ECG demonstrated extreme tachycardia with low voltage complexes in limb leads, suggestive of cardiovascular compromise.

2.3.7. Blood Coagulation

D-dimer levels were markedly elevated (1418.00 ng/ml FEU), indicating potential thrombotic risk.

2.3.8. Microbiological Cultures

No significant growth was observed in cultural studies.

3. Treatment and Management

3.1. Initial Management

Upon confirming the diagnosis of Sheehan's syndrome with concurrent acute decompensated heart failure (ADHF), pleural effusion, and acute kidney injury (AKI), a comprehensive multidisciplinary treatment approach was initiated [15]. The complexity of the case necessitated careful consideration of multiple organ systems and potential drug interactions.

3.2. Therapeutic Interventions

Diuretic therapy formed the cornerstone of initial management, with intravenous Furosemide 20 mg administered thrice daily to address fluid overload and cardiac decompensation [16]. Calcium channel blockade using Nicardia retard (Nifedipine) 10 mg thrice daily was implemented for blood pressure control, following established protocols for management of heart failure with preserved ejection fraction [17]. Antiplatelet therapy with Aspirin 75 mg daily was incorporated based on current cardiovascular guidelines [18].

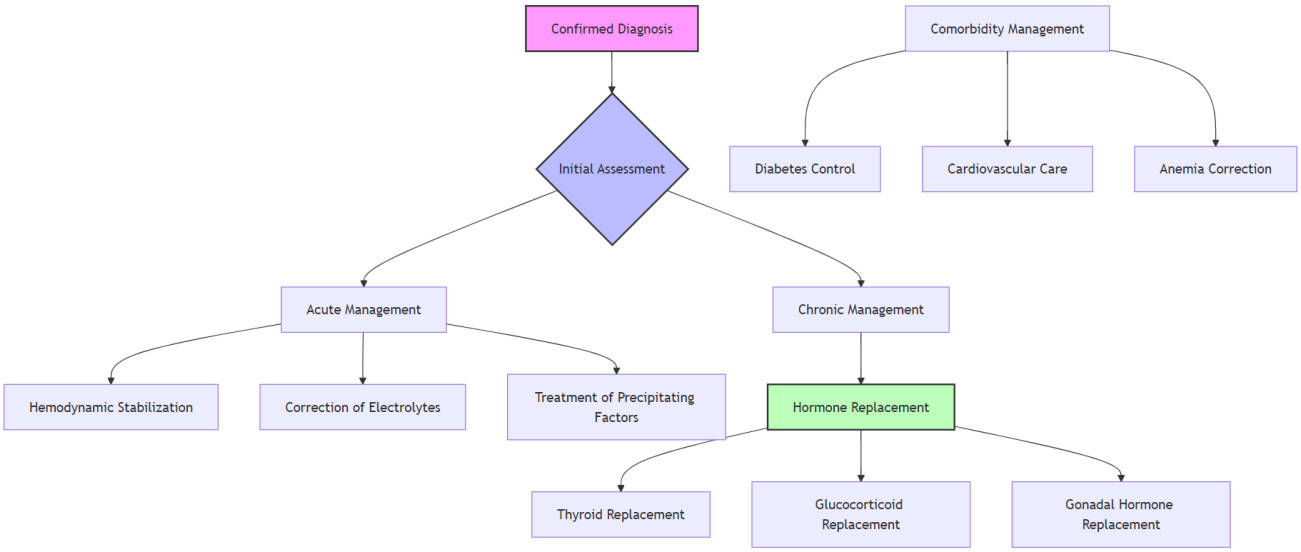

Figure 4. Management of Sheehan's Syndrome

The hormone replacement therapy centered on thyroid hormone supplementation with Thyronom 25 µg daily, carefully titrated to avoid precipitating cardiac complications [19]. Insulin therapy utilizing H.mixtard was initiated for glycemic control, with dosing adjusted based on regular blood glucose monitoring. This approach aligned with current recommendations for managing complex endocrine deficiencies in Sheehan's syndrome [20].

Human albumin 20% (100 ml) was administered daily for four consecutive days to address severe hypoalbuminemia, with careful consideration of fluid balance given the concurrent cardiac dysfunction [21]. The volume management strategy required precise calibration to avoid exacerbating either the cardiac or renal complications.

Table 4. Treatment Regimen

Medication	Dosage	Frequency	Purpose
Furosemide	20 mg	TID	Diuresis
Thyronom	25 µg	Daily	Thyroid replacement
Nicardia retard	10 mg	TID	BP control
Human Albumin 20%	100 mL	Daily for 4 days	Protein replacement
H.mixtard	30/70 mix 16-0-8 units	BID	Glycemic control
Aspirin	75 mg	Daily	Antiplatelet
Pantoprazole	40 mg	Daily	Gastric protection

Supportive therapy included gastric protection with Pantoprazole 40 mg daily, lipid management using Atorvastatin 40 mg daily, and anemia correction through iron supplementation (IFA 200 mg daily). Antacid therapy with intravenous Nodosis 500 mg thrice daily was incorporated to maintain gastric pH balance [22].

3.3. Therapeutic Monitoring

Blood pressure monitoring revealed initial significant fluctuations ranging from 135/100 to 190/100 mmHg, with gradual stabilization achieved by day 5 (130/90 mmHg) [23]. Regular electrocardiographic monitoring demonstrated progressive improvement in cardiac rhythm parameters.

Serial laboratory evaluations included daily complete blood count, renal function tests, electrolyte levels, and arterial blood gas analysis. Blood glucose monitoring was performed at regular intervals to optimize diabetes management [24].

The patient demonstrated progressive improvement in orthopnea and peripheral edema, accompanied by stabilization of vital parameters and enhanced urine output, indicating positive response to the implemented therapeutic regimen [25].

3.4. Long-term Management Plan

3.4.1. Hormone Replacement Therapy

Long-term management focused on regular assessment of thyroid function with corresponding adjustments in hormone replacement doses [26]. The protocol included vigilant monitoring for potential complications associated with chronic hormone replacement therapy.

3.4.2. Cardiovascular Management

The cardiovascular management plan incorporated regular cardiac assessment and optimization of heart failure therapy, with particular attention to blood pressure control and volume status [27].

3.4.3. Metabolic Management

Glycemic control optimization remained a central focus, requiring regular monitoring of diabetes status and appropriate nutritional support [28]. The management strategy accounted for the complex interplay between endocrine deficiencies and metabolic derangements.

3.5. Follow-up

The patient showed significant clinical improvement over the two-week hospitalization period. Initial severe anemia (Hb 6.6 g/dL) improved with iron supplementation and supportive care. Renal function demonstrated gradual recovery, with serum creatinine decreasing from 2.43 mg/dL to 1.4 mg/dL by discharge. The metabolic acidosis resolved with appropriate fluid and electrolyte management [29].

Cardiovascular status stabilized with diuretic therapy and blood pressure control. The orthopnea improved from grade IV to grade I, and peripheral edema showed marked reduction. Blood pressure stabilized at 130/90 mmHg with regular antihypertensive medication [30].

Endocrine parameters showed gradual improvement with hormone replacement therapy. Thyroid function tests began normalizing, and blood glucose levels achieved better control with regulated insulin therapy. The patient was discharged on a maintenance dose of thyroid hormone replacement and insulin, with instructions for regular endocrine follow-up [31].

Table 5. Comparison of Parameters at Admission and Discharge

Parameter	At Admission	At Discharge
Hemoglobin	6.6 g/dL	9.2 g/dL
Serum Creatinine	2.43 mg/dL	1.4 mg/dL
Blood Pressure	135/100 mmHg	130/90 mmHg
Blood Glucose	385 mg/dL	156 mg/dL
Serum Albumin	2.11 g/dL	3.2 g/dL
Orthopnea Grade	IV	I

4. Discussion

Sheehan's syndrome, while decreasing in incidence in developed nations, remains a significant concern in regions with limited obstetric care resources [32]. The present case exemplifies several unique aspects of this condition. First, the combination of Sheehan's syndrome with type 3C diabetes mellitus (Fibro calculous Pancreatic Diabetes) represents a rare association not frequently reported in literature [33]. The pathophysiological mechanism in this case likely involved severe postpartum hemorrhage leading to pituitary ischemia, complicated by pre-existing endocrine dysfunction. The enlarged pituitary gland during pregnancy, coupled with significant blood loss during cesarean delivery, created optimal conditions for pituitary necrosis [34].

The case highlights three important aspects in the management of Sheehan's syndrome:

1. The importance of early recognition, particularly in cases presenting with multisystem involvement [35].

2. The need for careful balance in hormone replacement therapy, especially in patients with concurrent cardiovascular complications [36].

3. The significance of a multidisciplinary approach in managing complex endocrine disorders with multiple comorbidities [37].

Recent literature suggests that the timing of hormone replacement initiation significantly influences long-term outcomes [38]. In this case, the prompt initiation of thyroid hormone replacement, along with careful cardiovascular and metabolic management, contributed to favorable clinical outcomes.

5. Conclusion

This case highlights the importance of maintaining a high index of suspicion for Sheehan's syndrome in patients presenting with multiple endocrine abnormalities following significant postpartum hemorrhage. The successful management of this case shows that even in the presence of multiple comorbidities, including Type 3C Diabetes mellitus and cardiovascular complications, appropriate hormone replacement therapy combined with supportive care can lead to favorable outcomes. Sheehan's syndrome should be considered in any postpartum patient presenting with multiple endocrine abnormalities, particularly following significant obstetric hemorrhage. The management of Sheehan's syndrome with concurrent diabetes and cardiovascular complications requires careful titration of hormone replacement therapy and close monitoring of multiple organ systems. Early recognition and initiation of appropriate hormone replacement therapy significantly influence the prognosis and long-term outcomes. During follow-up, the patient reported significant improvement in her quality-of-life following treatment initiation. She emphasized the importance of regular medication compliance and medical follow-up in managing her condition. The patient's understanding of her condition and active participation in the treatment process also contributed to the positive outcome.

Compliance with Ethical Standards

Acknowledgments

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Conflicts of Interest

The authors declare no conflicts of interest related to the publication of this case report.

Ethical Approval

This case report did not involve any experimental studies on human subjects or animals.

Informed Consent

Written informed consent was obtained from the patient for the publication of this case report, including clinical information, images, and investigation results. All identifying information has been removed to maintain patient confidentiality.

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Author's short biography

Dr. Hafsa Jabeen

Dr. Hafsa Jabeen completed her Pharm D from Osmania University. She is interested in clinical research and is presently working as a Clinical Research Coordinator in ClinArion Research Pvt.Ltd at Niloufer Hospital for Women's and Children. She is involved in case reporting, clinical monitoring, and publishing scholarly articles. Her interests include promoting clinical practice education and contributing to impactful clinical research.

Dr. Ayesha Fatima

Dr. Ayesha Fatima holds a Pharm D degree and is currently working as an Assistant Professor in the Department of Pharmacy Practice at RBVRR Women's College of Pharmacy, with over three years of academic experience. She has made numerous contributions in the field of teaching and is a passionate researcher with several publications to her credit.