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Clinical Case Reports

Sheehan's Syndrome with Dilated Cardiomyopathy: An Overlooked Sequela of Postpartum Hemorrhage

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Abstract

Summary: We present the case of a 43-year-old woman who developed panhypopituitarism as a sequela of postpartum haemorrhage. She presented with recurrent vomiting, diarrhea, hypotension, and hypoglycaemia and was subsequently diagnosed with Sheehan's syndrome complicated by dilated cardiomyopathy. Prompt initiation of hormonal replacement therapy resulted in remarkable clinical improvement. This case emphasises the critical need for early recognition of Sheehan's syndrome in women with a history of obstetric haemorrhage, particularly when presenting with chronic nonspecific complaints, to prevent life-threatening complications.

Keywords: Sheehan syndrome; postpartum hemorrhage; hypopituitarism; dilated cardiomyopathy; heart failure.

Introduction

Sheehan's syndrome is a form of hypopituitarism that occurs as a result of ischaemic necrosis of the anterior pituitary gland following severe postpartum haemorrhage (PPH).[2] The pituitary gland undergoes hyperplasia during pregnancy, making it particularly vulnerable to vascular compromise in the setting of massive blood loss and hypovolemic shock.[1,2] This condition, first described by Harold Sheehan in 1937, remains a major cause of hypopituitarism in developing countries, where maternal health services and emergency obstetric care are often limited.[2,6,5]

The clinical manifestations of Sheehan's syndrome are variable and may appear immediately or many years after the inciting obstetric event. Acute presentations can include failure of lactation, severe fatigue, adrenal crisis with hypotension, and hypoglycaemia.[13,14] More chronic or delayed features include secondary amenorrhoea, loss of axillary and pubic hair, generalised weakness, cold intolerance, alopecia, coarse dry skin, and progressive features of hypothyroidism and adrenal insufficiency. Because these symptoms are nonspecific and evolve gradually, diagnosis is often delayed, sometimes until life-threatening metabolic or systemic complications develop.[2,6,15]

Although endocrine and metabolic manifestations are well documented, cardiac involvement in Sheehan~~'s~~ syndrome is exceedingly rare. Only isolated case reports have described dilated cardiomyopathy (DCM) as a complication.[4,7] The pathophysiology is thought to be multifactorial, involving prolonged untreated hypothyroidism, glucocorticoid deficiency impairing myocardial function, and possible autoimmune mechanisms of action. Importantly, DCM in this context has been shown to improve with appropriate hormone replacement, suggesting that early recognition and treatment are crucial.[8]

We present a case history of a middle-aged woman from Punjab, Pakistan, who developed Sheehan syndrome following massive PPH and later presented with panhypopituitarism complicated by dilated cardiomyopathy.[3,8] This case contributes to the scarcity of literature on cardiac manifestations of Sheehan syndrome and emphasises the role of early diagnosis in women who have experienced obstetric haemorrhage.

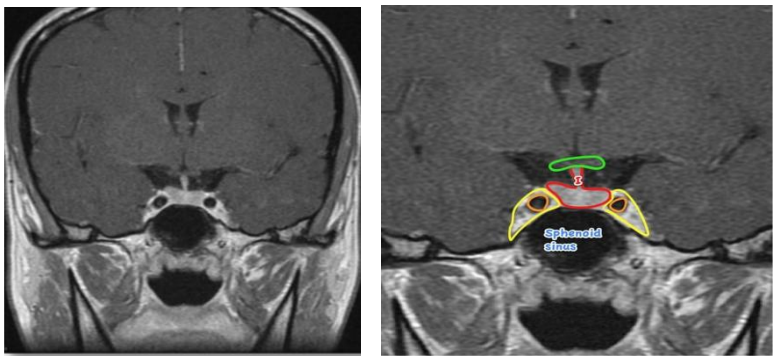

Figure 1: A.B Normal Pituitary Gland(Coronal Images)

Case Presentation

A 43-year-old homemaker, married for 18 years, with no prior medical comorbidities, presented with recurrent vomiting, diarrh~~o~~ea, and abdominal pain of three days' duration. These episodes were associated with dizziness, drowsiness, and symptomatic hypoglycaemia. The patient~~She~~ also reported cold intolerance, progressive alopecia, reduced libido, generali~~sz~~ed weakness, and increasing exertional dyspnoea over the past several years.

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- Acquisition, Analysis, Interpretation
JI SN JH - Drafting
IA - Critical Review

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Her obstetric history was unremarkable. She was Gravida 4 and para 4. Her last childbirth in 2013 was complicated by massive postpartum haemorrhage due to delayed placental expulsion. She required intensive care unit admission, surgical evacuation, and transfusion of eight units of red cell concentrate. Although she survived the acute event, she developed secondary amenorrhoea and infertility within two months of delivery, accompanied by progressive fatigue and loss of secondary sexual characteristics.

On examination, her blood pressure was 100/70 mmHg in the supine position and dropped to 80/60 mmHg in the standing position, with a pulse of 78/minute, low volume. The random capillary blood glucose level was 68 mg/dL. Physical features included dry coarse skin, thinning of scalp hair, loss of the lateral third of the eyebrows, and bilateral pedal oedema.

Table 1: Baseline Laboratory Investigations

Investigation	Result / Findings
Peripheral film	Microcytic hypochromic anemia
Serum Iron	**48 µg/dL** (Reference: 50–170 µg/dL)
Serum TIBC (Total Iron Binding Capacity)	**184 µg/dL** (Reference: 250–400.9 µg/dL)
Serum Folate Levels	**2.4 ng/mL** (Reference: >3.3 ng/mL)
Serum Vitamin D	**14.6 ng/mL** (Normal: ≥20 ng/mL)
Serum Vitamin B12	270 pg/mL (Reference: 208–964 pg/mL)
Urine routine examination	Normal
Abdominal Ultrasound	Unremarkable
Chest X-Ray	Normal
Serum Cholesterol	**211 mg/dL** (Reference: 125–200 mg/dL)
Serum Triglyceride	127 mg/dL (Reference: <150 mg/dL)
Anti-nuclear antibody (ANA)	Negative
Anti-TTG Ab (IgA)	1 (Reference: <10)
TSH	1.34 µl U/mL (0.3-4.5)
T3	1.15 ng/mL (0.69-2.15)
T4	7.97 ng/mL (52-127)
Prolactin	14.1 µl U/mL (54-490)
FSH	6.18 IU/L (3.03-8.08 IU/L)
LH	1.60 IU/L (1.80-11.78 IU/L)
Serum IGF-1 levels	14.7 nmole/L (18.4-37.12)
Estradiol	55.6 pg/mL (692-1286 pg/mL)
Progesterone	0.394 ng/mL (143 ng/mL)
C-peptide levels (Fasting)	1.9 ng/mL (1.1-5.0)
Serum Insulin (Fasting)	7.5 µl U/mL (5-25)
Serum ACTH (morning)	13.5 pg/mL (7-69)

Table 2: Follow-up Laboratory tests

Laboratory Test	16/11/2022	13/01/2023
WBC	8.5	8.0
Hemoglobin (Hgb)	**11.1**	**11.0**
MCV	76.3	85.3
MCH	29.4	29.9
Platelets	179	364
PT	13	—
APTT	36	—
Urea	**62**	26
Creatinine	**1.3**	0.7
Total Bilirubin	0.6	0.3
ALT	**66**	19
ALP	134	65
Sodium	136	137

Radiological Investigations:

Electrocardiography showed nonspecific T-wave inversions(Fig 2). Echocardiography revealed globally reduced left ventricular contractility with an ejection fraction of 35–40%, while cardiac MRI confirmed dilated cardiomyopathy with biventricular dysfunction(Fig 3). Pituitary MRI revealed an empty sella(Fig 4B). Panhypopituitarism was diagnosed by hormonal tests; the levels of cortisol, thyroid hormones, gonadotropins, oestradiol, and prolactin were reduced. Other laboratory tests, including renal and liver function tests, were normal.

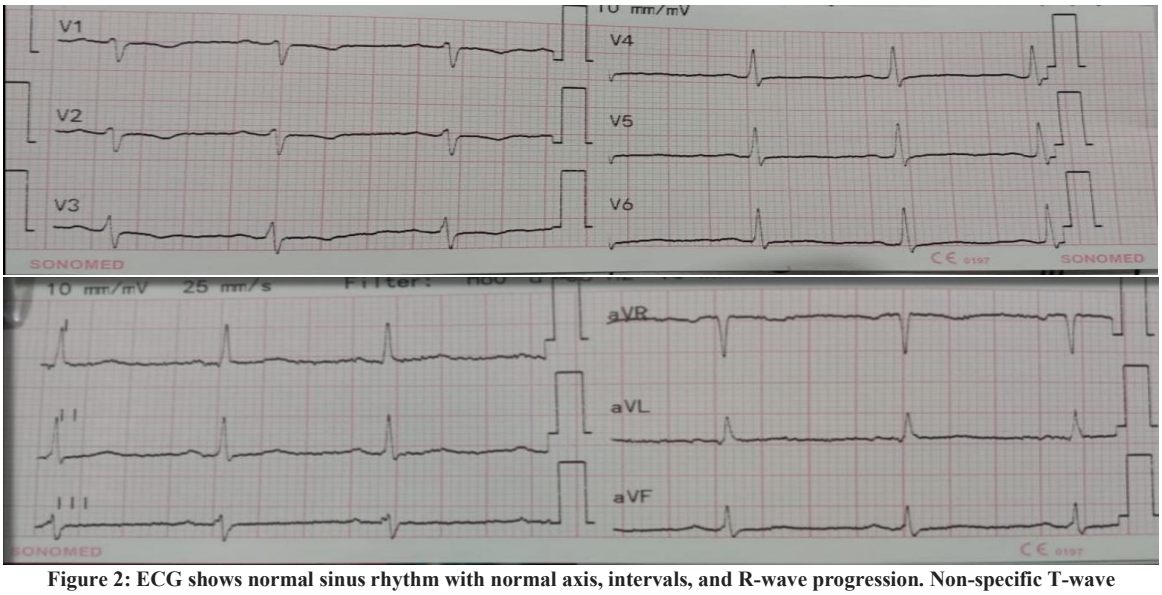

Figure 2: ECG shows normal sinus rhythm with normal axis, intervals, and R-wave progression. Non-specific T-wave inversions in V1–V3 with T-wave flattening in V4–V6 and limb leads

Outcome and Follow-up: Sheehan syndrome complicated by dilated cardiomyopathy was diagnosed. Her acute presentation was stabilized with intravenous fluids and antiemetics. Prednisolone (7.5 mg/day), levothyroxine (50 0g/day), and oestrogen-progesterone therapy were initiated. Social therapy was also administered to support the treatment of cardiomyopathy. She recovered from her symptoms of fatigue, postural dizziness, and hypoglycaemia on follow-up twice in two weeks. Her blood pressure stabilised at 100/70 mmHg, and her thyroid profile normalised. Repeat echocardiography showed an improvement in the systolic blood pressure. The patient reported a significant improvement in energy, appetite, and mood, with the resumption of daily household activities.

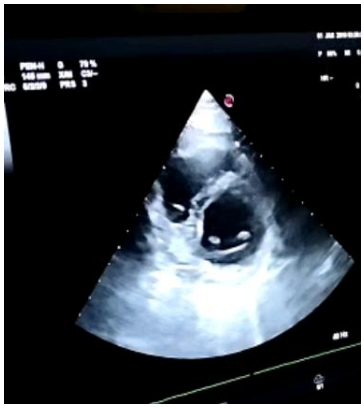

Figure 3: EF 35% -40% Moderate LV systolic dysfunction. Global hypokinesia is present

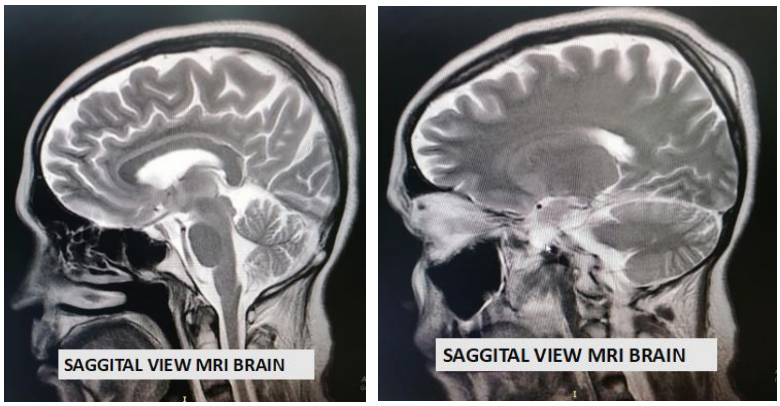

Figure 4: (A) Early — enlarged pituitary with low T1, high T2 homogeneous signal, ring enhancement; (B) Late — empty sella of normal size

Discussion

Sheehan's syndrome remains frequently underdiagnosed because of its insidious onset and non-specific manifestations. Women may present years after the inciting obstetric insult, as in our patient who developed clinical features eight years after severe postpartum haemorrhage. This delay reflects both the subtlety of the symptoms and the lack of awareness among healthcare providers.[2,3]

Endocrine deficiencies in Sheehan's syndrome often present gradually, with amenorrhea, fatigue, and generalised weakness being the earliest indicators.[3,11] Because these symptoms overlap with common post-pregnancy complaints, the diagnosis is easily overlooked. Recognition requires a high index of suspicion, especially in women with a history of complicated childbirth and subsequent menstrual irregularities.

In the present case, the most remarkable feature was dilated cardiomyopathy. While it is established that hypothyroidism and adrenal insufficiency can impair myocardial contractility, progression to overt dilated cardiomyopathy is unusual. This highlights the potential severity of long-standing, untreated hypopituitarism.[7,8]

In Sheehan syndrome, cardiomyopathy pathogenesis is likely multifactorial. Besides the direct impacts of thyroid and adrenal hormone deficiency, the autoimmune process has been suggested to be a contributory factor.[12] This intersection indicates that Sheehan's syndrome could not only be an endocrine disorder, but this syndrome could also have an immunological overlap.

Notably, timely and appropriate hormone replacement has been shown to improve the cardiac performance of such patients, and in some cases, cardiomyopathy has been reversed.[9,10] This demonstrates the importance of early diagnosis and management of hypopituitarism.

On a larger population health scale, this case highlights the persistence of postpartum haemorrhage-associated morbidity in Pakistan. To avoid the development of long-term complications, such as in the case of Sheehan, it is important to strengthen maternal healthcare services, improve obstetric care, and provide proper follow-up for high-risk women.

Conclusion

The results of this study showed that two weeks of proper hormone replacement therapy resulted in significant clinical improvement in the patient. Her fatigue, frequent instances of hypotension, and episodes of hypoglycaemia were completely cured. The blood pressure leveled at 100 / 70 mmHg without the necessity to use support measures, and her thyroid picture was restored to normal levels, which is a clear indication of sufficient endocrine treatment. There was a significant increase in the left ventricular systolic function on follow-up echocardiography, indicating the recovery of the previously reported dilated cardiomyopathy. The patient also reported a major improvement in overall well-being, such as an increase in energy level, regained appetite, and a more stable mood. All these changes indicate the reversibility of endocrine-induced cardiac dysfunction with early hormone replacement.

Author Information

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