Case Report
A 42-year-old woman presented herself to the emergency services in cardiogenic shock. She had a history of progressively worsening breathlessness for the past year; however, no medical evaluation had been performed earlier. At presentation, her pulse rate was 110 beats per minute, regular, and low volume, and her blood pressure was 90/70 mm Hg. On auscultation, third heart sound was present, but no murmurs were heard. Soon after presentation, she had an asystolic cardiac arrest. However, she was resuscitated successfully and mechanically ventilated, and she required inotropic support with dopamine and noradrenaline for maintaining blood pressure.

Her ECG showed normal sinus rhythm and nonspecific ST-T changes, and chest radiograph showed cardiomegaly with features of pulmonary venous hypertension (Figure 1). Her echocardiogram showed severe left-ventricular systolic dysfunction with a left-ventricular ejection fraction of 20% (Figure 2). Her routine blood biochemistry, including liver and renal function tests, was within normal limits, except for mild anemia with hemoglobin of 9.5 g/dl. A diagnosis of dilated cardiomyopathy with cardiogenic shock was made, and the patient was treated accordingly. Subsequently, she was weaned off the ventilator, but she continued to require inotropic support.

She remained markedly symptomatic with fatigue and complained of blackouts on getting up. There was a postural drop of 25 mm Hg in systolic blood pressure on sitting up. Furthermore, it was noticed that her heart rates did not go beyond 110 beats per minute, despite being on high doses of inotropes and remaining hypotensive. On close scrutiny, patchy pigmentation of face was noticed along with loss of axillary and pubic hair.

Further inquiry into her past history revealed that she had nearly bled to death in her last pregnancy 14 years ago. Since then, she had no mensturation and lactation. In light of this, she was investigated for endocrine disorder and was found to have panhypopituitarism (Table)—thus a case of Sheehan syndrome. The diagnosis remarkably changed her management because along with cardiogenic shock, she also had additional hypoadrenal shock. She was started on glucocorticoids, thyroxine, and fludrocortisone, and within 2 days she was weaned off inotropes. Her blood pressure...
and postural symptoms improved remarkably. Symptoms of heart failure regressed, and repeat echocardiogram revealed left-ventricular ejection fraction of 40% at discharge. MRI of the brain was suggestive of pituitary atrophy (Figure 3). At 6 months follow-up, she was asymptomatic with normal chest radiograph (Figure 4) and normal left-ventricular ejection fraction (Figure 5).

**Discussion**

Sheehan syndrome is a postpartum panhypopituitarism, first described in 1937 by Sheehan, after postpartum hemorrhage and vascular collapse. The mechanism of pituitary apoplexy is its growth to twice its usual size during pregnancy, which makes it prone to ischemia in case of postpartum hemorrhage or shock. Additionally, autoimmunity has also been cited as a mechanism for Sheehan syndrome, especially to explain cases of delayed presentation. The extent of pituitary damage determines the time of onset and magnitude of pituitary hypofunction. The gland has a large secretory reserve, and >75% must be destroyed before clinical manifestations are evident. The usual symptoms are failure of lactation, menstrual irregularity, loss of secondary sexual characteristics, and features of other hormonal deficiencies. Sheehan syndrome presenting as heart failure is rarely reported. Hypothyroidism, adrenal insufficiency, and growth hormone deficiency, individually, have been associated with heart failure, with reversal of heart failure on hormone replacement. Thus, reversible heart failure attributable to collective deficiency of all these hormones, as in panhypopituitarism, can be explained, as reported in case of an adult male with pituitary apoplexy. To date, to the best of our knowledge, only 2 such cases have been reported—a 33-year-old woman symptomatic within 2 weeks of delivery and a 25-year-old woman from India, who became symptomatic 2 years after her delivery. However, no case of Sheehan syndrome with such late presentation primarily with heart failure is reported in literature to the best of our knowledge.

**Implications for Clinical Practice**

This case report highlights the importance of diligent history taking in diagnosis of a reversible cause of cardiomyopathy. It highlights that it may be prudent to look for Sheehan syndrome in appropriate clinical settings, that is, young and middle-aged women in their reproductive age group presenting with congestive heart failure, especially, because some of these patients may be misdiagnosed as peripartum cardiomyopathy if they present early after childbirth. Finally, it adds to the list of reversible cardiomyopathies.

### Table. Pituitary Hormone Levels

<table>
<thead>
<tr>
<th>Hormone, Unit</th>
<th>Patient’s Value</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tri-iodothyronine-T3, ng/mL</td>
<td>0.37</td>
<td>0.58–1.59</td>
</tr>
<tr>
<td>Thyroxine (T4), μg/dL</td>
<td>3.56</td>
<td>4.87–11.72</td>
</tr>
<tr>
<td>Thyroid stimulating hormone, mIU/mL</td>
<td>1.92</td>
<td>0.35–4.94</td>
</tr>
<tr>
<td>Prolactin, ng/mL</td>
<td>&lt;0.6</td>
<td>5.18–26.53</td>
</tr>
<tr>
<td>Follicle stimulating hormone, mIU/mL</td>
<td>2.26</td>
<td>26.7–133.41</td>
</tr>
<tr>
<td>Luteinizing hormone, mIU/mL</td>
<td>0.76</td>
<td>10.39–64.75</td>
</tr>
<tr>
<td>Growth hormone, ng/mL</td>
<td>0.5</td>
<td>0.5–17</td>
</tr>
<tr>
<td>Fasting cortisol, μg/dL</td>
<td>6.0</td>
<td>5–25</td>
</tr>
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Figure 3. T2-sagittal image—CSF filled sella turcica suggestive of empty sella.

Figure 4. Chest X-ray at follow-up—normal cardiothoracic ratio.

Figure 5. M-mode echocardiogram at follow-up showing normal left-ventricular dimensions and function. EDV indicates end diastolic volume; EF, ejection fraction; ESV, end systolic volume; FS, fractional shortening; IVS, interventricular septum; LVID, left ventricular internal diameter; and LVPW, left ventricle posterior wall.
Disclosures

None.

References


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Dilated Cardiomyopathy: A Ghost From the Past
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