Life-threatening postpartum hemorrhage is uncommon as a result of the advances in obstetric care. As this case illustrates, however, the consequences of postpartum hemorrhage can be severe.

Case Report
A healthy 39-year-old woman, gravida 2 para 1, came to the Emergency Department with a 2-day history of nausea, vomiting, diarrhea, dizziness, and fatigue. Twelve days earlier, she had undergone a primary low transverse cesarean section at term for failure to descend after premature rupture of membranes, oxytocin augmentation, and 2½ hours of active pushing. A viable male infant weighing 3,832 g was delivered with Apgar scores of 9 and 9 at 1 and 5 minutes, respectively. The patient’s uterus was found to be boggy and patulous after delivery. A lower uterine segment laceration was repaired, and the uterus and skin incisions were closed. She had considerable postpartum bleeding and received misoprostol and dinoprostone.

The patient continued to hemorrhage despite these interventions and was taken back to the operating room for an emergent supracervical hysterectomy. Because her right ovary was hemorrhagic, she also had a right oophorectomy. She continued to hemorrhage after the hysterectomy and underwent an embolization of her right vaginal artery. The embolization finally stopped her bleeding. During these procedures she was hypotensive and coagulopathic and received numerous units of blood, platelets, and fresh frozen plasma. After the embolization, the patient remained in the intensive care unit for 2 days and spent a total of 10 days in the hospital. A sodium level measured on the 5th day of her hospitalization was normal. She had been home for 2 days when she came to the Emergency Department. She started feeling nauseous the last 2 days of her initial hospitalization. The nausea worsened, and she developed vomiting, diarrhea, dizziness, and fatigue.

In the Emergency Department, the patient was normovolemic. Her blood pressure, pulse, respiratory rate, and temperature were normal, and she did not have orthostatic hypotension. Her sodium was 108 mEq/L. Her hemoglobin was 10.7 mL/dL, hematocrit 29%, white cell count 6,000/μL, potassium 4.6 mEq/L, chloride 81 mEq/L, carbon dioxide content 16 mEq/L, blood urea nitrogen 5.0 mg/L, and creatinine 0.7 mmol/L. She was alert and oriented and had normal findings on neurologic examination. She was admitted to the hospital for severe hyponatremia. Initially her hyponatremia rapidly corrected to 119 mEq/L by active hydration with normal saline solution and then furosemide diuresis. The patient subsequently had fluids restricted; however, her sodium level was slow to correct. At admission, her serum osmolality was 220 mOsm/L, and her urine osmolality was 120 mOsm/L. Her cortisol level and thyroid-stimulating hormone (TSH) level were also measured at admission and were low at 1.9 μg/dL and 0.44 ng/dL, respectively. Later her free thyroxine and triiodothyronine (T3) levels were found to be <0.4 ng/dL and 44 ng/dL, respectively. These laboratory values suggested pituitary failure. To ascertain whether this patient did in fact have pituitary failure, an insulin tolerance test was conducted on day 4 of her hospitalization; the results are displayed in Table 1.

Based on these results, pituitary failure caused by Sheehan syndrome was diagnosed. The patient was started on cortisone acetate while still an inpatient and on levothyroxine and estrogen 1 week after her discharge. She was advised to wear a Medic Alert bracelet indicating adrenal insufficiency. The patient will need to remain on both estrogen and levothyroxine therapy for the rest of her life. She will need adrenocortical steroid replacement whenever she has surgery or is severely ill.
Discussion

Sheehan syndrome, or necrosis of the pituitary gland, is a rare complication of postpartum hemorrhage initially described in 1937.1 This case illustrates an example of Sheehan syndrome at 2 weeks postpartum with severe hyponatremia. The pituitary gland is physiologically enlarged in pregnancy and is therefore very sensitive to the decreased blood flow caused by massive hemorrhage and hypovolemic shock. Women with Sheehan syndrome have varying degrees of hypopituitarism, ranging from panhypopituitarism to only selective pituitary deficiencies.2–4 The anterior pituitary is more susceptible to damage than the posterior pituitary.

Failure to lactate or difficulties with lactation are common initial symptoms of Sheehan syndrome.5 Many women also report amenorrhea or oligomenorrhea after delivery. In some cases, the diagnosis is not made until years later, when features of hypopituitarism, ranging from panhypopituitarism to only selective pituitary deficiencies,2–4 The anterior pituitary is more susceptible to damage than the posterior pituitary.

In this case, the diagnosis of Sheehan syndrome was suspected because of her history, hyponatremia, and low baseline cortisol and thyroid hormone levels. Neither cortisol, corticotropin, nor growth hormone levels responded to a hypoglycemic state. The insulin tolerance test induces transient hypoglycemia, which in a person with a normal pituitary gland stimulates corticotropin production and cortisol release. Normal stimulated levels of cortisol range in the hundreds. This patient’s cortisol level never went higher than 3 μg/dL. The insulin tolerance test can also test for growth hormone deficiency but is an unpleasant test for the patient and is contraindicated in patients who have coronary artery disease or seizures.

Radiologic imaging with either computed tomography or magnetic resonance imaging is usually not helpful in the acute phase and has not been used frequently in acute diagnosis.11 Several studies have shown an empty sella, however, in women.

Table 1. Insulin Tolerance Test Results, by Minutes After Insulin Administration.

<table>
<thead>
<tr>
<th>Components Measured</th>
<th>30 min</th>
<th>45 min</th>
<th>60 min</th>
<th>90 min</th>
<th>120 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corticotropin (ACTH) (pg/mL)</td>
<td>16</td>
<td>18</td>
<td>17</td>
<td>15</td>
<td>12</td>
</tr>
<tr>
<td>Cortisol (μg/dL)</td>
<td>1.6</td>
<td>1.8</td>
<td>3.2</td>
<td>3.3</td>
<td>3.0</td>
</tr>
<tr>
<td>Glucose (mg/dL)</td>
<td>47</td>
<td>38</td>
<td>50</td>
<td>74</td>
<td>104</td>
</tr>
<tr>
<td>Growth hormone (ng/mL)</td>
<td>0.5</td>
<td>0.8</td>
<td>2.4</td>
<td>1.7</td>
<td>1.3</td>
</tr>
</tbody>
</table>
with Sheehan syndrome further into the disease process.\textsuperscript{12–14} Imaging can be helpful in situations where the diagnosis is not apparent.

Treatment of young women with hypopituitarism usually includes replacement of hydrocortisone first and then replacement of thyroid hormone and estrogen with or without progesterone depending on whether she has a uterus. Hydrocortisone is replaced first because thyroxine therapy can exacerbate glucocorticoid deficiency and theoretically induce an adrenal crisis.\textsuperscript{9,15} The standard dose of hydrocortisone is 20 mg/d for an adult (15 mg every morning and 5 mg every evening). Both thyroxine replacement and gonadotropin replacement are common, and doses are titrated to each individual. Replacement of growth hormone is necessary in children with hypopituitarism but is controversial in adults. Some people with severe growth hormone deficiency derive great benefit from replacement, but standard recommendations are not available.\textsuperscript{16}

Although Sheehan syndrome is uncommon as a result of improved obstetric care, it should be a consideration in any woman who has a history of a postpartum hemorrhage and who reports signs or symptoms of pituitary deficiency.

References