Sheehan Syndrome: A Rare Complication of Postpartum Hemorrhage

Sarina Schrager, MD, and Laura Sabo, MD

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 (T_3) 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.

Submitted, revised, 16 January 2001.

From the Department of Family Medicine (SS, LS), University of Wisconsin, Madison. Address reprint requests to Sarina Schrager, MD, Department of Family Medicine, University of Wisconsin, 777 S. Mills St, Madison, WI 53715.

J Am Board Fam Pract: first published as on 1 September 2001. Downloaded from http://www.jabfm.org/ on 15 November 2026 by guest. Protected by copyright.

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

Components Measured	30 min	45 min	60 min	90 min	120 min
Corticotropin (ACTH) (pg/mL)	16	18	17	15	12
Cortisol (μg/dL)	1.6	1.8	3.2	3.3	3.0
Glucose (mg/dL)	47	38	50	74	104
Growth hormone (ng/mL)	0.5	0.8	2.4	1.7	1.3

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, such as secondary hypothyroidism or secondary adrenal insufficiency, become evident in a woman who had a postpartum hemorrhage. A woman with undiagnosed hypopituitarism from Sheehan syndrome might be relatively asymptomatic until her body is stressed by a severe infection or surgery years after her delivery, and she goes into an adrenal crisis.

Hyponatremia is an uncommon acute presentation of Sheehan syndrome.[6–8] There are several possible mechanisms by which hypopituitarism can result in hyponatremia. Hypothyroidism can cause decreased free-water clearance and subsequent hyponatremia. Glucocorticoid deficiency can also cause decreased free-water clearance independent of vasopressin. Hypopituitarism itself can stimulate vasopressin secretion and can cause severe inappropriate secretion of antidiuretic hormone, which can also cause hyponatremia. The potassium level in these situations is normal, because adrenal production of aldosterone is not dependent on the pituitary. In this case, the patient responded slightly to fluid restriction, but her sodium levels did not return to normal until she received hydrocortisone replacement therapy.

Diagnosis of Sheehan syndrome can be difficult. The diagnosis is based on clinical evidence of hypopituitarism in a woman with a history of a postpartum hemorrhage. Deficiencies of specific anterior pituitary hormones will cause varied symptoms. Corticotropin deficiency can cause weakness, fatigue, hypoglycemia, or dizziness. Gonadotropin deficiency will often cause amenorrhea, oligomenorrhea, hot flashes, or decreased libido. Growth hormone deficiency causes many vague symptoms including fatigue, decreased quality of life, and decreased muscle mass.

Secondary hypothyroidism is clinically indistinguishable from primary hypothyroidism, but patients with hypothyroidism caused by hypopituitarism have low T_3 and T_4 levels with normal or even inappropriately low TSH levels. Diagnosis of panhypopituitarism is straightforward, but partial deficiencies are often difficult to elicit.[9] A woman with panhypopituitarism will have low levels of pituitary hormones (luteinizing hormone, corticotropin, and thyrotropin) as well as the target hormones (cortisol and thyroxine). Stimulation tests (insulin-induced hypoglycemia or metyrapone stimulation test) are often necessary for diagnosis in the acute phase or in situations where a partial deficiency is suspected.[10]

In this case, the diagnosis of Sheehan syndrome was suspected because of her history, hyponatremia, and low baseline cortisol and thyroid hormones 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

J Am Board Fam Pract: first published as on 1 September 2001. Downloaded from http://www.jabfm.org/ on 15 November 2026 by guest. Protected by copyright.

with Sheehan syndrome further into the disease process.[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.[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.[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

1. Sheehan HL. Postpartum necrosis of the anterior pituitary. J Path Bacteriol 1937;45:189–214.
2. DiZerega G, Kletzky OA, Mishell DR. Diagnosis of Sheehan's syndrome using sequential pituitary stimulation tests. Am J Obstet Gynecol 1978;132:348–53.
3. Ozbey N, Inanc S, Aral F, et al. Clinical and laboratory evaluation of 40 patients with Sheehan's syndrome. Isr J Med Sci 1994;30:826–9.
4. Lakhdar AA, McLaren EH, Davda NS, McKay EJ, Rubin PC. Pituitary failure from Sheehan's syndrome in the puerperium. Two case reports. Br J Obstet Gynaecol 1987;94:998–9.
5. Roberts DM. Sheehan's syndrome. Am Fam Phys 1988;37:223–7.
6. Boulanger E, Pagniez D, Roueff S, et al. Sheehan syndrome presenting as early post-partum hyponatremia. Nephrol Dial Transplant 1999;14:2714–5.
7. Shoji M, Kimura T, Ota K, et al. Cortical laminar necrosis and central pontine myelinolysis in a patient with Sheehan syndrome and severe hyponatremia. Intern Med 1996;35:427–31.
8. Putterman C, Almog Y, Caraco Y, Gross DJ, Ben-Chetrit E. Inappropriate secretion of antidiuretic hormone in Sheehan's syndrome: a rare cause of postpartum hyponatremia. Am J Obstet Gynecol 1991;165(5 Pt 1):1330–3.
9. Lamberts SW, de Herder WW, van der Lely AJ. Pituitary insufficiency. Lancet 1998;352:127–34.
10. Vance ML. Hypopituitarism. N Engl J Med 1994; 330:1651–62.
11. Dejager S, Gerber S, Foubert L, Turpin G. Sheehan's syndrome: differential diagnosis in the acute phase. J Intern Med 1998;244:261–6.
12. Banzal S, Ayoola EA, Banzal S. Sheehan's syndrome in Saudi Arabia. Int J Gynaecol Obstet 1999;66: 181–2.
13. Otsuka F, Kageyama J, Ogura T, Hattori T, Makino H. Sheehan's syndrome of more than 30 years' duration: an endocrine and MRI study of 6 cases. Endocr J 1998;45:451–8.
14. Dash RJ, Gupta V, Suri S. Sheehan's syndrome: clinical profile, pituitary hormone responses and computed sellar tomography. Aust N Z J Med 1993; 23:26–31.
15. Orrego JJ, Barkan AL. Pituitary disorders. Drug treatment options. Drugs 2000;59:93–106.
16. Davies JS, Obuobie K, Smith J, et al. A therapeutic trial of growth hormone in hypopituitary adults and its influence upon continued prescription by general practitioners. Clin Endocrinol 2000;52:295–303.

J Am Board Fam Pract: first published as on 1 September 2001. Downloaded from http://www.jabfm.org/ on 15 November 2026 by guest. Protected by copyright.