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CASE REPORTS

Sheehan syndrome: acute presentation with severe headache

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ABSTRACT
Postpartum headache is a common occurrence with a broad differential diagnosis. Sheehan syndrome, or postpartum pituitary necrosis, is not typically recognized as a cause of postpartum headache. We present a case of Sheehan syndrome that initially presented as severe headache after vaginal delivery complicated by retained placenta and postpartum hemorrhage. The patient was discharged home on postpartum day three but continued to have headaches and returned to hospital on postpartum day six with severe headache, failure to lactate, edema, dizziness, fatigue, nausea and vomiting. Cranial magnetic resonance imaging revealed pituitary infarction consistent with Sheehan syndrome. We discuss the differential diagnosis for postpartum headache, the pathophysiological features of Sheehan syndrome and headache as an atypical acute presentation.

Keywords: Sheehan syndrome; Postpartum headache

Introduction

Postpartum headache has an incidence of 11–80%.[1,2] The majority of headaches in the postpartum period are of primary etiology, and include migraine, tension-type, cervicogenic, cluster headaches, and other trigeminal autonomic cephalgias.[2–5] While secondary headaches are less common, they can be associated with significant morbidity and mortality, and a high index of suspicion and low threshold for neurodiagnostic imaging should be maintained. Secondary headaches may be due to complications of neuraxial anesthesia such as post-dural puncture headache (PDPH) and pneumocephalus, obstetric disease including hypertensive disorders, pre-eclampsia and eclampsia, or intracranial pathology such as cerebral venous thrombosis, ischemic or hemorrhagic stroke, subarachnoid hemorrhage, reversible cerebral vasoconstrictive syndromes, posterior reversible leucoencephalopathy syndrome and meningitis.[1–5] The obstetric anesthesiologist is frequently involved in the investigation of postpartum headache.

Sheehan syndrome, or postpartum pituitary necrosis, is not typically recognized as a cause of secondary postpartum headache, and is not included in the differential diagnosis of postpartum headache in some major obstetric anesthesia textbooks.[4,5] It is infrequently diagnosed in the early postpartum period, and may remain undiagnosed for months or years until presenting as hypopituitarism.[6–8] We present a case of Sheehan syndrome that initially presented as severe headache after vaginal delivery.

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Case report

A previously healthy 31-year-old G1P0 woman at 40+3 weeks of gestation with body mass index of 34.6 kg/m^2 presented for post-dates induction of labor in an otherwise uncomplicated pregnancy. Pre-delivery hematocrit was 29% (hemoglobin 9.2 g/dL). Labor epidural analgesia was placed uneventfully. Spontaneous vaginal delivery occurred 23 h later, but a retained placenta required manual extraction. The epidural was topped-up with 2% lidocaine 8 mL and fentanyl 100 μg in divided doses. The procedure was complicated by an estimated blood loss of 1500 mL. The patient experienced a brief episode of hypotension with systolic blood pressure in the 70s mmHg, with rapid return to >100 mmHg for the remainder of the procedure. Tachycardia (100–130 beats/min) was present throughout surgery, with a 12-min period during which heart rate was 150 beats/min. The patient remained comfortable, alert and oriented throughout the procedure apart from one short episode of mild headache and nausea. Intraoperatively, she received phenylephrine 700 μg, 1 L human plasma protein fraction 5% and 1 L lactated Ringer's solution. Postoperatively, her hematocrit was 20%

(hemoglobin 6.2 g/dL) and she complained of headache. She was given two units of packed red blood cells with initial improvement in headache and hematocrit to 23%. There was no further bleeding and the epidural catheter was removed 2 h after completion of the procedure.

Headache recurred 3 h later. It was severe, constant, localized in the center of the head from vertex to occiput and behind the eyes, intermittently worse with standing, but with no consistent postural characteristics. Intermittent dizziness, nausea and mild photophobia were present, but there was no nuchal rigidity or backache. She was afebrile with no signs of meningeal irritation, altered mental status, lateralizing signs or neurologic deficits.

The headache was not considered consistent with PDPH or meningitis and was assumed to be migrainous or a tension-type headache. Treatment with intravenous metoclopramide and fluid bolus, and oral acetaminophen and caffeine yielded minimal improvement. Since the hematocrit was 23%, another unit of packed red blood cells was transfused with temporary improvement in headache. On postpartum day two, headache persisted but was not consistent with PDPH. A cranial magnetic resonance imaging (MRI) scan was suggested, but after discussion with the obstetric team she was discharged home.

Oral oxycodone, acetaminophen and ibuprofen produced partial control of headache at home. On postpartum day six, the patient returned to hospital with severe headache, failure to lactate, bilateral lower extremity and facial edema, dizziness, fatigue, pallor, mild hypertension, orthostatic hypotension, decreased appetite, nausea and vomiting. Cranial MRI revealed an infarcted enlarged pituitary with an expanded sella, which failed to enhance after contrast with the exception of its peripheral rim (Fig. 1). This was consistent with non-hemorrhagic pituitary infarction. No evidence of intracranial venous thrombosis was seen.

Table 1 Electrolyte and endocrine evaluation on postpartum day six

	Patient value	Normal range
Sodium (mmol/L)	125	135–145
Chloride (mmol/L)	93	98–108
Serum osmolality (mOs/kg)	256	277–293
Thyroid-stimulating hormone (uIU/mL)	0.37	0.34–5.66
Free thyroxine (ng/dL)	0.61	0.52–1.21
Free triiodothyronine (pg/mL)	2.51	2.20–3.80
Random cortisol (morning; μg/dL)	0.5	5–25
Adrenocorticotropic hormone (pg/mL)	<1.0	15–66
Follicle-stimulating hormone (mIU/mL)	3.6	4.4–11
Luteinizing hormone (mIU/mL)	<0.2	1.6–8.3
Prolactin (ng/mL)	3.59	3.34–26.76

Blood results are shown in Table 1, and confirmed partial hypopituitarism with corticotroph, thyrotroph, and lactotroph dysfunction. Sheehan syndrome was diagnosed. Initial treatment with intravenous hydrocortisone 50 mg 6-hourly, normal saline and thyroxine 50 μg daily was changed to oral therapy after several days. Hyponatremia, energy level, headache, nausea and dizziness improved.

The patient returned to clinic 10 weeks later with polyuria and polydipsia. Water deprivation tests demonstrated partial diabetes insipidus, and low insulin-like growth factor 3. Desmopressin (DDAVP) 0.2 mg twice daily and somatotropin were started. Six months postpartum, she continued to take prednisone, levothyroxine, DDAVP and somatropin and denied symptoms of panhypopituitarism.

Discussion

Sheehan syndrome is pituitary necrosis after postpartum hemorrhage and hypovolemia,[9] and occurs in 1–2% of women who lose 1–2 L of blood with associated hypo-

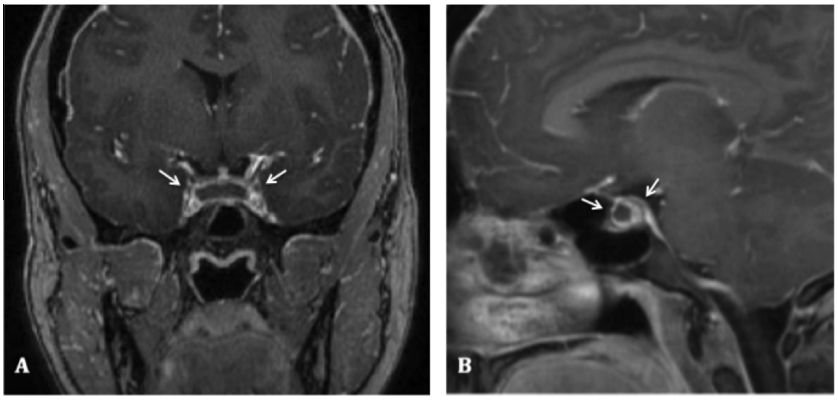

Fig. 1 Cranial magnetic resonance imaging scan performed on postpartum day six: T1-weighted coronal (A) and sagittal (B) images post gadolinium enhancement. The pituitary gland (arrows) appears enlarged with peripheral enhancement and an isodense central area.