MR of nonhemorrhagic postpartum pituitary apoplexy.

G LavallÖ©e, R Morcos, J Palardy, M AubÖ© and D Gilbert

AJNR Am J Neuroradiol 1995, 16 (9) 1939-1941
http://www.ajnr.org/content/16/9/1939

MR of Nonhemorrhagic Postpartum Pituitary Apoplexy

Gilles Lavallée, Rami Morcos, Jean Palardy, Marc Aubé, and Danielle Gilbert

Summary: A 30-year-old woman had uterine bleeding and hypotension after delivery. Hyponatremic seizures and a mild headache prompted early neuroimaging, which disclosed an enlarged nonhemorrhagic pituitary gland with subsequent involution consistent with pituitary apoplexy (Sheehan syndrome). Endocrinologic investigation confirmed a partial pituitary insufficiency with subsequent improvement to almost normal status.

Index terms: Brain, infarction; Pituitary gland, hypopituitarism

Postpartum pituitary necrosis of the anterior lobe of the pituitary gland, known as *Sheehan syndrome*, is a well-established clinical entity (1). Many neuroradiologic case reports show late findings such as a partially or completely empty sella (2–4). We report a case of nonhemorrhagic postpartum pituitary infarction documented in the acute as well as in a later phase with clinical, endocrine, and sequential magnetic resonance (MR) imaging studies.

Case Report

A 30-year-old, gravida 1, para 0, aborta 0, right-handed woman was admitted to the hospital for induction of labor at 41 weeks of pregnancy. Her medical history was not significant. She was on no regular medication. The pregnancy was uneventful except for a low-percentile fetal growth curve throughout. On admission, blood pressure was 120/80 mm Hg, and Hb was 136 g/L. Labor was induced with oxytocin in a 5% dextrose-water 25% saline solution. Six hours later, she delivered a 3153-g girl with an Apgar score of 8/10/10 at 1, 5, and 10 minutes, and placental expulsion was complete. Three hours later, she had an episode of vomiting, and uterine bleeding with clot formation was noticed. She underwent a uterine revision under 5 mg intravenous midazolam hydrochloride. At that time blood pressure was 90/60 mmHg, and Hb decreased to 76 g/L.

Six hours after delivery, three generalized tonicoclonic convulsions of 1 minute's duration occurred. She received a transfusion and a loading dose of phenytoin sodium. Thereafter, she had a mild persistent headache, with nausea and vomiting 3 hours later.

The detailed neurologic exam, including visual fields, was normal with a supple neck. Goldmann perimetry obtained later was normal. Plain computed tomography (CT) of the brain (not shown) done the day after delivery was normal. Lumbar puncture revealed a clear fluid with an opening pressure of 180 mm H_2O of normal cellular and biochemical composition. Electroencephalography was normal except for drowsiness. Biochemistry was as follows: blood sodium, 125 mmol/L; potassium, 3 mmol/L; creatinine, 48 μmol/L; glucose, 7.8 mmol/L; ionized calcium, 1.2 mmol/L; magnesium, 0.46 mmol/L; plasma osmolarity, 266 mOsm/kg H_2O; urine sodium, 22 mmol/L; urine osmolarity, 251 mOsm/kg H_2O. Those results were felt to be consistent with a dilutional state with hyponatremic seizures, and correction of electrolytes was achieved with a return of plasma Na to 136 mmol/L the next day. However, the patient still had a mild nonremitting headache, and the possibility of pituitary apoplexy was raised by the clinical context.

Further investigation confirmed endocrine abnormalities. The morning after delivery, plasma cortisol was at 216 nmol/L (normal, 300 to 700 nmol/L). Three days after delivery, hormonal tests gave the following results: thyrotropin, 2.6 mU/L (normal, 0.4 to 4.5), free T4 index, 15 (normal, 13 to 49); prolactin, 25 nmol/L (normal $<$ 20; higher in postpartum); luteinizing hormone, 142 U/L (normal, 5 to 20); follicle-stimulating hormone, 19.8 U/L (normal, 5 to 20); estradiol, 1362 pmol/L (normal $>$ 221). Lactation occurred on day 4 after delivery. Pituitary function was evaluated during a stress test with a lack of response of cortisol and growth hormone during an insulin-induced hypoglycemic episode, suggesting a defect in the hypothalamopituitary-adrenal axis and the growth hormone axis.

Five days after delivery, a coronal postcontrast CT study demonstrated a peripherally enhancing mass in the sella extending in the suprasellar cistern (Fig 1) with no hemorrhagic component. MR was performed 6 days after delivery; a large intrasellar mass with superior extension was confirmed on T1-weighted (Fig 1B) and T2-weighted sequences (Fig 1C). Isointensity was remarkable on both.

Received December 2, 1994; accepted after revision March 7, 1995.

From the Departments of Radiology (G.L., M.A., D.G.), Neurological Sciences, Service of Neurology (R.M.), and Medicine, Service of Endocrinology (J.P.), Sacré-Coeur Hospital and University of Montreal, Quebec, Canada.

Address reprint requests to Gilles Lavallée, MD, Department of Radiology, Sacré-Coeur Hospital, 5400 Gouin Blvd W, Montreal (Quebec), Canada H4J IC5.

AJNR 16:1939–1941, Oct 1995 0195-6108/95/1609–1939

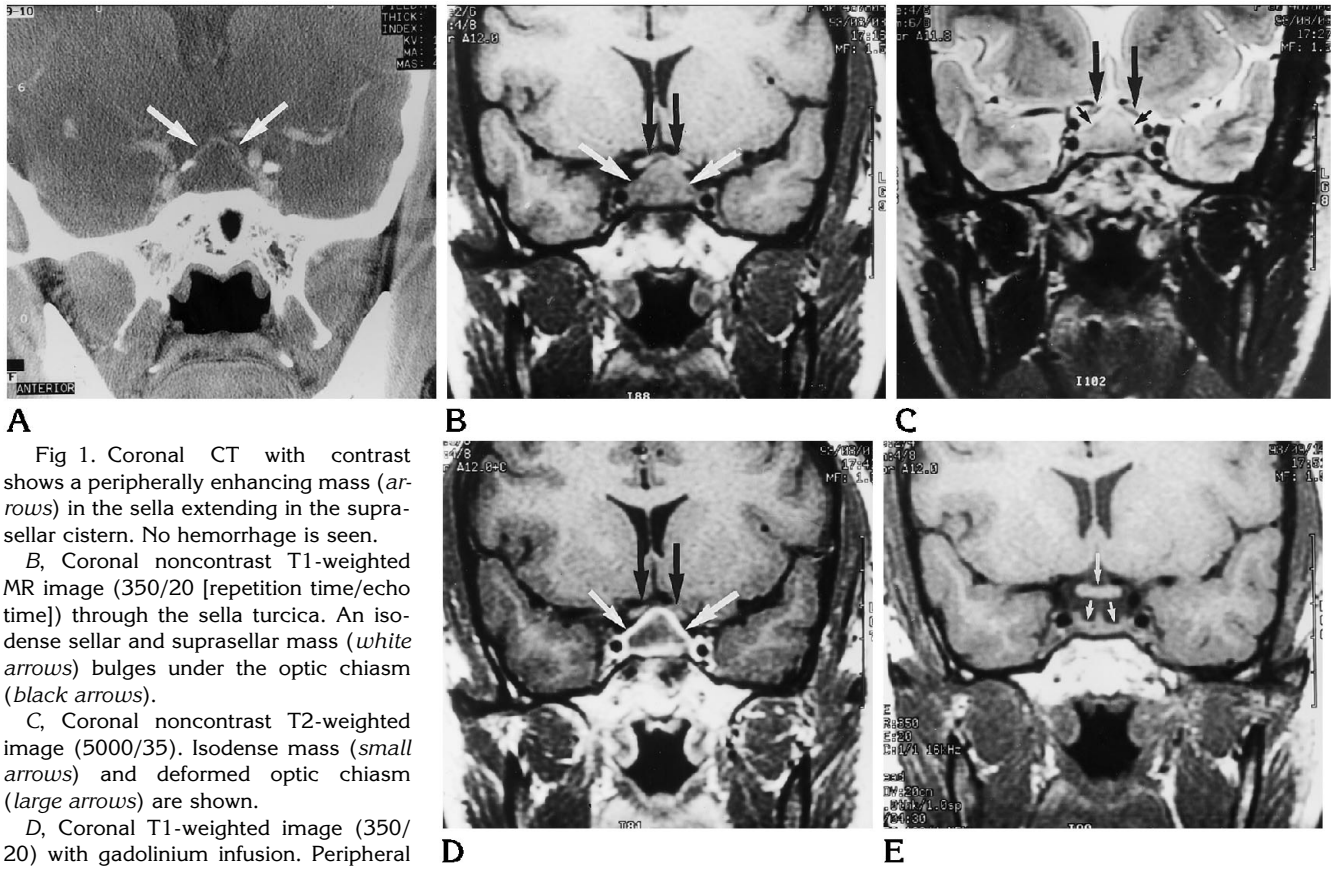

Fig 1. Coronal CT with contrast shows a peripherally enhancing mass (*arrows*) in the sella extending in the suprasellar cistern. No hemorrhage is seen.

B, Coronal noncontrast T1-weighted MR image (350/20 [repetition time/echo time]) through the sella turcica. An isodense sellar and suprasellar mass (*white arrows*) bulges under the optic chiasm (*black arrows*).

C, Coronal noncontrast T2-weighted image (5000/35). Isodense mass (*small arrows*) and deformed optic chiasm (*large arrows*) are shown.

D, Coronal T1-weighted image (350/20) with gadolinium infusion. Peripheral enhancement is noted surrounding a still isodense mass (*white arrows*). *Black arrows* indicate optic chiasm.

E, Coronal noncontrast T1-weighted image (350/20) 6 weeks later. The mass effect has disappeared, and the pituitary gland (*short arrows*) is somewhat atrophic (3 mm in height). The pituitary stalk is in midline position, and the optic chiasm (*long arrow*) has resumed normal position. There is no evidence of tumor or hemorrhagic sequel.

No enhancement was found after gadopentetate dimeglumine infusion (Fig 1D) except for that in a peripheral rim of tissue.

The patient eventually was discharged from the hospital on hormone replacement therapy with hydrocortisone and levothyroxine. One month later, she resumed normal menstrual flow. Levothyroxine was ceased rapidly, and hydrocortisone was decreased slowly and discontinued 4 months after delivery. Noncontrast MR was obtained 48 days after delivery (Fig 1E). The intrasellar mass effect completely disappeared, leaving a somewhat atrophic pituitary gland with no evidence of tumoral process or hemoglobin degradation products as would have been expected with an hemorrhagic infarction. A stimulation test with synthetic corticotropin with a cortisol response during the sixth postpartum month indirectly showed the recovery of the hypothalamopituitary-adrenal axis.

Discussion

Postpartum hypopituitarism secondary to pituitary infarction during severe shock at or near the time of delivery is known as *Sheehan syndrome* (1). The anterior lobe of the gland is affected. Diabetes insipidus and involvement of the optic nerves or chiasm are rare events. However, hyponatremic and hypoglycemic convulsions have been reported (5), as in our case. Usually the disease runs a long course, with symptoms of pituitary insufficiency appearing 15 to 20 years later (6). However, many authors described partial hormonal defects during endocrine stimulation tests after severe postpartum hemorrhagic shock. Selective loss of one or two trophic hormones and spontaneous improvement occur rarely (7). Our patient had an atypical clinical course, because she presented acute symptoms and clear defects in only two of the hormones secreted by the pituitary gland (cortisol and growth hormone) with, however, a lower-than-expected prolactin level given her postpartum state, and recovered

her pituitary function rapidly and almost completely.

Pituitary apoplexy usually is associated with a preexisting macroadenoma, although it can occur with a normal gland. In the first instance, CT will show a tumor of mottled density or occasionally hyperdense hemorrhage as well as indirect signs of a tumor. Hemoglobin degradation products may appear in subsequent MR studies. Absence of concomitant adenoma and recovery of endocrine function have been reported earlier with an hemorrhagic necrosis of the pituitary gland (8). MR imaging may fail to demonstrate acute hemorrhage unless specific sequences are used; the hemorrhage may be isointense on T1-weighted images and hypointense on T2-weighted images. In the subacute phase, however, the extracellular methemoglobin should appear bright on both T1 and T2 sequences, features that are not present in our case. Finally, hyperplasia has not been considered in the differential diagnosis in this case, given the clinical evolution as well as the absence of expected enhancement on both CT and MR studies (9).

It is likely that patients with similar clinical symptoms will have early neuroimaging in the future with similar results. It is therefore important to be alert to the possibility that an enlarged nonhemorrhagic pituitary gland may be present in the postinfarction phase of Sheehan syndrome, as shown in the present case report.

Acknowledgment

We thank Mrs Marthe Dupont for typing the manuscript.

References

1. Sheehan HL, Stanfor JP. The pathogenesis of postpartum pituitary necrosis of the anterior lobe of the pituitary gland. *Acta Endocrinol* 1961;37:479–510
2. Dash RJ, Gupta V, Suri S. Sheehan's syndrome: clinical profile, pituitary hormone responses and computed sellar tomography. *Aust Ne Z J Med* 1993;23(1):26–31
3. Lee HC, Lee EJ, Lee, et al. Computed tomographic correlation with pituitary function in Sheehan's syndrome. *Korean J Intern Med* 1992;7(1):48–53
4. Bakiri F, Bendib SE, Maoui R, Bendib A, Benmiloud M. The sella turcica in Sheehan's syndrome: computerized tomographic study in 54 patients. *J Endocrinol Invest* 1991;14(3):193–196
5. Farmer TW, Flowers CA. Neurologic manifestations of postpartum pituitary insufficiency. *Neurology* 1955;5:212–214
6. De Groot LJ. *Textbook of Endocrinology*. 2nd ed. Philadelphia, Pa: Saunders; 1989:431–432
7. Rolih CA, Ober P. Pituitary apoplexy. *Endocrinol Metab Clin North Am* 1993;22(2):291–302
8. Mauerhoff T, Leveque P, Lambert AE. Spontaneous pituitary apoplexy with transient panhypopituitarism and diabetes insipidus. *Acta Clin Belg* 1991;46(1):30–36
9. Latchaw RE. *MR and CT Imaging of the Head, Neck and Spine*. 2nd ed. St Louis, Mo: Mosby Yearbook; 1990:705–706