CASE REPORT

Spontaneous pituitary apoplexy during the second trimester of pregnancy, with sensory loss

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SUMMARY

A 32-year-old Hispanic woman at 23 weeks gestation presented with right-sided headache, associated with photophobia and right-sided numbness. She denied visual problems, menstrual irregularities and galactorrhoea. Examination revealed visual acuity 20/40 bilaterally with some blurriness on the left side, decreased right V1-V2 facial sensation and preserved 5/5 power, but decreased sensation over the entire right upper extremity (RUE) and right lower extremity (RLE) to touch and pinprick. Laboratories suggested normal pituitary function, but MRI of the brain revealed enlargement of the pituitary (1.7 cm), with layering haemorrhage posteriorly and mild compression of the optic nerve. The patient underwent emergent evacuation of a pituitary haematoma, and histology revealed minute fragments of adenohypophysis with haemorrhage and fibrosis.

BACKGROUND

Pituitary apoplexy is rare in pregnancy. It is an endocrine emergency and must be considered in a pregnant patient presenting with sudden onset headache. This case provides a reminder of other less commonly associated neurological symptoms and signs in such patients.

CASE PRESENTATION

A 32-year-old Hispanic woman (G6P4) at 23 weeks gestation presented with a severe, sharp and constant right-sided headache located around her face and the back of her neck. This was associated with photophobia and right-sided numbness of her body. On initial presentation, she denied any history of nausea, vomiting, fever, chills, loss of consciousness, blood clots, stroke, seizures, prior headaches, visual problems, menstrual irregularities, oligomenorrhoea or galactorrhoea. There was no history of pituitary tumours. Examination revealed visual acuity of 20/40 bilaterally with some blurriness on the left side and decreased right V1-V2 facial sensation with midline splitting at the forehead and nose, with other cranial nerves being normal. She had preserved 5/5 power in all groups except the right upper extremity (4/5), with decreased sensation over the entire RUE and RLE to touch and pinprick. Laboratories suggested an elevated prolactin at 314.2 ng/mL (in a setting of pregnancy), normal cortisol 18.2 µg/dL and adrenocorticotrophic hormone (ACTH) 14 pg/mL (6–58), follicle-stimulating hormone (FSH; 0.9 IU/L), luteinising hormone (LH) 0.19 (IU/L), insulin-like growth factor-1 (IGF-1) low at 78 ng/mL (106–

368), normal thyroid-stimulating hormone (TSH) 2.15 mIU/mL (0.34–5.6) and free thyroxine (fT4) 0.69 ng/dL (0.58–1.64). MRI of the brain revealed enlargement of the pituitary (1.7 cm), with layering haemorrhage posteriorly, and compression of the optic nerve (figure 1).

The patient described visual symptoms shortly after admission, including diplopia (side-by-side double vision) and blurriness in the peripheries. Ophthalmological examination demonstrated visual acuity of 20/20 and superotemporal hemianopsia on Goldmann visual fields. Owing to the visual symptoms, an emergent evacuation of a pituitary haematoma was subsequently performed through an endoscopic endonasal approach. Postoperative MRI revealed resolution of the optic nerve compression.

OUTCOME AND FOLLOW-UP

Postoperatively, the patient denied having any vision problems or headaches, normal visual fields by confrontation; physical examination was negative for any galactorrhoea, sensory or motor deficits. She was started on intravenous dexamethasone and this was tapered up to the day of discharge. On the second postoperative day, she developed polyuria and increased urine output (4 L in 12 h); sodium ranged from 135–145 mEq/L. The patient was started on desmopressin (DDAVP) (1 µg subcutaneous injections). After three doses of DDAVP, her urine output improved, indicating improvement of transient postoperative diabetes insipidus, and she did not require any further doses of DDAVP. Postoperative laboratory work-up showed normal TSH of 1.28 mIU/mL, fT4 of 0.82 ng/dL, and normal total T4 and total T3. IGF-1 remained slightly low at 94 ng/mL (106–368). ACTH was 24 pg/mL (6–58), with cortisol of 21.4 µg/dL (5 days off dexamethasone). Prolactin with dilutions ×10 remained elevated at 328.2 ng/mL.

Pathology revealed minute fragments of scattered nested adenohypophyseal cells within collagenised and partially haemorrhagic stroma. Reticulin stain showed the nested pattern of these cells and the rich reticulin network among them. Masson's trichrome stain highlighted presence of collagen, as well as fibrinoid-haemorrhagic areas. Pancytokeratin immunohistochemistry showed glandular cells with no epithelial lining to suggest a cystic structure. The findings were consistent with pituitary parenchyma with haemorrhage and fibrosis (figure 2).

DISCUSSION

Pituitary apoplexy may occur due to spontaneous haemorrhage into a pituitary adenoma (pituitary

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Figure 1 Precontrast T1 sagittal MRI (image 1) reveals an enlarged pituitary gland, measuring 1.7×1.3 cm, with a convex-shaped superior border abutting the optic chiasm. This image also shows an acute hyperintense layer in haemorrhage located posteriorly in the pituitary gland. Ventricles and sulci are within normal limits, there is no surrounding oedema nor midline shift, and basal cisterns are intact. Precontrast T1 axial (image 2) and coronal (image 3) views again show an enlarged pituitary gland with an acute haemorrhage that appears hyperintense in the posterior region of the pituitary gland. Diffusion imaging (not pictured) showed no foci of restricted diffusion to suggest acute infarct.

tumour apoplexy), or in a gland that is physiologically enlarged.¹ This condition was first described by Baily in 1898.² It is often an endocrine emergency. The other causes that can lead to this are head trauma or skull base fracture, or it may be in association with other risk factors. In about 80% of cases, pituitary apoplexy may be the presenting symptom of a pituitary adenoma.

The adult pituitary weighs approximately 600 mg (range 400–900) and measures approximately 13 mm in the longest transverse diameter, 6–9 mm in vertical height and about 9 mm anteroposteriorly. The gland grows by 40% in the second trimester and 70% in the third trimester; the size may increase in any dimension, by twofold or more, and pituitary weight increases up to 1 g. This growth during pregnancy occurs as a result of a marked increase in the number of prolactin-producing cells and a relative decrease in other hormone-secreting cells. This is physiological and is stimulated by high oestradiol levels and usually resolves by 6 months postpartum.³

Apoplexy can occur at any age but has a lower incidence among children than in adults.⁴ Even though the pituitary

enlarges during pregnancy, apoplexy is not a frequent phenomenon, and if it occurs, is usually seen in people with a known pituitary tumour.⁵ Spontaneous pituitary apoplexy is even rarer in occurrence.

The spectrum of clinical features of pituitary apoplexy includes headache (84–100%), nausea (80%), decreased visual acuity (56%), visual field deficits (34–70%) and, less commonly, ocular paresis and impaired mental status.¹ Classically, symptoms of pituitary apoplexy evolve over hours to 1–2 days, but sometimes, subacute symptoms may precede the full-blown picture of acute pituitary tumour apoplexy.⁶

Pathogenesis of pituitary tumour apoplexy has been attributed to different hypotheses, which include an imbalance between blood flow and perfusion, causing the tumour to outgrow its vascular supply, and compression of portal vessels by a growing pituitary mass, resulting in ischaemic necrosis, with or without haemorrhage.⁴ Some risk factors have been postulated as predisposing conditions and include conditions that may cause a sudden increase in hypophyseal blood flow, decreased blood flow to the pituitary tumour, hormonal stimulation of the

Figure 2 Top left: biopsy of the pituitary gland shows anterior pituitary parenchyma (black arrow), fibrosis (*) and haemorrhage (white arrow) (H&E; original magnification: ×100). Top right: reticulin stain highlights the preservation of the usual nested pattern of anterior pituitary parenchyma (arrow) in the background of dense reticulin fibres (white arrow; reticulin; original magnification: ×100). Bottom left: dense collagenous background (white arrow) and fibrin due to haemorrhage (black arrow) are seen (Masson trichrome; original magnification: ×100). Bottom right: epithelial cells of the anterior pituitary parenchyma are highlighted (arrows), but no epithelial lining on the surface of the tissue (bottom) to indicate a cyst lining or cystic areas in the parenchyma is seen (pancytokeratin immunohistochemistry; original magnification: ×200).

pituitary gland, exogenous administration of hormones or drugs and an anticoagulated state such as from liver disease or immunodeficiency.⁴

Pituitary imaging without contrast (CT or MRI) usually reveals signs of intrapituitary or intra-adenoma haemorrhage, stalk deviation and compression of normal pituitary tissue, as well as signs of parasellar haemorrhage in severe cases.

Work-up should include a complete laboratory panel including prolactin, cortisol, ACTH, TSH, T4, fT4, IGF-1, FSH, LH, oestrogen in females and testosterone in males, and full ophthalmological examination. Visual field testing in pregnancy should be performed in each trimester.⁷

Indication for emergent trans-sphenoidal surgery is visual field compromise. Some individuals may recover spontaneously but may develop long-term pituitary insufficiency. Pituitary function does not commonly recover after resolution of the acute haemorrhage, and patients require adrenal, thyroid and/or gonadal steroid hormone replacement in the long term.

A suggested follow-up regimen in these patients includes evaluation 1 week after surgery, and reassessment of pituitary function should be carried out 6 weeks after surgery or delivery, with annual monitoring thereafter.⁷ A repeat MRI is obtained 12 weeks after surgery and then annually.

The novelty in our case includes a rare presentation of spontaneous pituitary apoplexy in a patient—during the second trimester of pregnancy—who had no history of pituitary abnormality and, most importantly, the presentation with unilateral numbness and sensory loss, which has not been described previously in the literature. Interestingly, motor abnormalities due to internal carotid artery occlusion secondary to pituitary apoplexy—but not purely sensory symptoms—have been previously described.⁸

Contributors RRA and PAG saw the patient, provided clinical care, performed the literature search, wrote the manuscript and approve submission. REP provided the MRIs and MG provided the pathology images.

Competing interests None declared.

Patient consent Obtained.

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Learning points

- Pituitary apoplexy is very rare in pregnancy. It is an endocrine emergency and must be considered in a pregnant patient presenting with sudden onset headache and sensory abnormalities.
- Our case educates clinicians about a novel sensory presentation of spontaneous pituitary apoplexy, and makes them cognisant of other less commonly associated neurological symptoms and signs.
- Recurrent problems from the tumour during subsequent pregnancies can occur and close monitoring is advisable.

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