Gestational Pituitary Apoplexy

Sir,

A 25-year-old woman presented with a 6-month history of headaches and galactorrhea, and primary infertility of 3-year duration. Endocrine assessment revealed a modestly elevated serum prolactin level 96 ng/mL (reference range 2.5-29.2 ng/mL) and normal thyroid function. T1-weighted contrast-enhanced magnetic resonance imaging (MRI) (Figure 1: Panels A [cornonal] and B [sagittal]) showed a 4 mm hypointense lesion on the right side of the gland (arrow), typical of a microadenoma. She was commenced on cabergoline 0.25 mg once a week, and shortly thereafter had spontaneous restoration of menses. At 3 months, she discontinued treatment following confirmation of pregnancy but unfortunately defaulted from endocrine follow-up. At 37-week gestation, she re-presented with recent onset, severe headaches. Her Glasgow Coma Scale score was 15/15, and there was no neurological deficit. Formal ophthalmic assessment showed preserved visual acuities and visual fields. A T2-weighted noncontrast MRI demonstrated a hyperintense lesion with a fluid level in an enlarged sella (Figure 1: Panels C and D, arrows), consistent with a diagnosis of pituitary apoplexy.

She was immediately treated with hydrocortisone (100 mg intravenously, 8 hourly) and recommenced on cabergoline, at an increased dose of 0.5 mg daily. Forty-eight hours later, she underwent an uneventful lower segment cesarean section and delivered a healthy baby. She expressed a strong desire to breastfeed and hence cabergoline was stopped again. She continued on physiological hydrocortisone replacement, made a good recovery thereafter, and was able to successfully

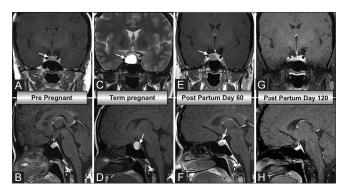


Figure 1: T1-weighted contrast-enhanced magnetic resonance imaging (Panels A [coronal] and B [sagittal]) showed a 4 mm hypointense lesion on the right side of the pituitary (arrow), T2-weighted noncontrast magnetic resonance imaging demonstrated a hyperintense lesion with a fluid level in an enlarged sella (Panels C and D), consistent with pituitary apoplexy. T1-weighted contrast-enhanced magnetic resonance imaging at 2-month postdelivery with resolution of apoplexy and persistence of the underlying adenoma (Panels E and F arrows). T1-weighted contrast-enhanced magnetic resonance imaging at 4-month postpartum revealed complete resolution of the pituitary adenoma (Panels G and H)

breastfeed. A repeat MRI (T1-weighted contrast-enhanced MRI) after 2-months postdelivery showed resolution of apoplexy but apparent persistence of the underlying microadenoma (Figure 1: Panels E and F, arrows). The patient continued to breastfeed and remained off cabergoline. Hydrocortisone was tapered following demonstration of normal adrenal function. Further imaging (T1-weighted contrast-enhanced MRI) at 4-month postpartum revealed complete resolution of the pituitary adenoma (Figure 1: Panels G and H). Serum prolactin has not been re-measured since delivery as the patient continues to breastfeed. She is otherwise eupituitary.

Pituitary apoplexy during pregnancy is a rare emergency with potentially life-threatening consequences for the mother and fetus.^[1] It should however be considered in the differential diagnosis of any pregnant woman presenting with sudden onset severe headache, especially where there is a prior history of a pituitary adenoma.^[2] To the best of our knowledge, this is only the third case of gestational pituitary apoplexy arising in the context of a previous microprolactinoma.^[3,4] Potential precipitating factors for apoplexy include treatment with dopamine agonists, anticoagulation/antiplatelet therapy (including during coronary artery bypass grafting), hypertension, and pregnancy.^[5] In our case, it is interesting to speculate whether the rise in estrogen levels during pregnancy caused growth of the microadenoma and predisposed to apoplexy. Early recognition of pituitary apoplexy ensures timely endocrine replacement therapy and combined ophthalmic and radiological assessments to guide further management. The decision for conservative versus medical (e.g., dopamine agonists) and/or surgical management should be taken on a case-by-case basis following multidisciplinary discussion between the endocrine, obstetric, neonatal, and neurosurgical teams.^[6]

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Conflicts of interest

There are no conflicts of interest.

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