

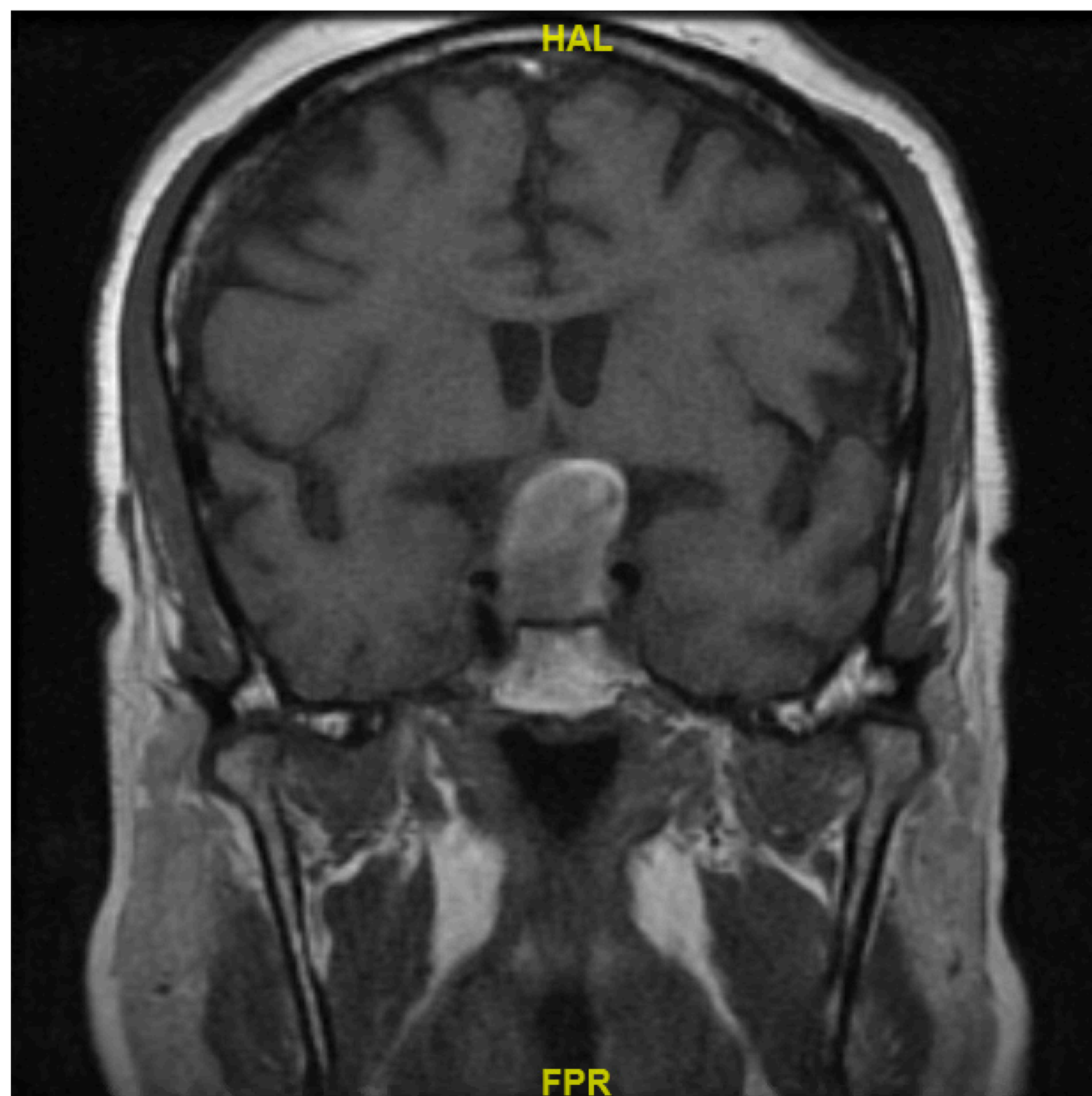
Missed Pituitary Apoplexy in a HIV Patient

Tiffany Tsang, MD; Maya Raghuwanshi, MD

Department of Endocrinology, Rutgers New Jersey Medical School

Background

Pituitary hemorrhage has a prevalence of up to 25% in macroadenomas. In apoplectic hemorrhage, loss of pituitary function is associated with significant mortality. Sudden hemorrhagic enlargement of a preexisting adenoma compresses surrounding structures; ophthalmoplegia, mydriasis and ptosis occur when cranial nerves in the cavernous sinus are affected. The classic clinical syndrome of headache, visual deficits, altered mental status and hypopituitarism, combined with imaging, confirms the diagnosis of pituitary apoplexy.



Clinical Case

A 73 year-old smoker with a history of transsphenoidal surgery 20 years ago for a pituitary adenoma, HIV (CD4 928), hypertension, diabetes, coronary artery disease presented with two days of altered mental status, lethargy and headaches. Patient was febrile to 104.1°F on arrival. Head CT was done prior to a lumbar puncture, which showed a 1.7 x 2.1 x 2.2 cm pituitary mass. CSF analysis was positive for xanthochromia, and revealed 220 RBCs, 275 WBCs, glucose 143 mg/dL, protein 154 mg/dL and an opening pressure of 9 mm H2O. Meropenem and vancomycin were started for presumed meningitis. A hypopituitary state was found on labs: prolactin 1.9 ng/mL (4.6-21.4 ng/mL), ACTH 3.2 pg/mL (7.2-63.3 pg/mL), cortisol 3.6 ug/dL, TSH 0.164 uIU/mL (0.27-4.0 uIU/mL), free T4 0.6 ng/dL (0.7-1.5 ng/dL), T3 0.3 ng/mL (0.6-1.6 ng/mL), IGF-1 33 ng/mL (41-179 ng/mL), total testosterone 4 ng/dL (193-740 ng/dL), LH 0.3 mIU/mL and FSH 1.2 mIU/mL. Subsequent MRI showed a 2.2 x 2.4 x 2.9 cm pituitary macroadenoma extending into the suprasellar region with mass effect on the optic chiasm and lateral displacement of the cavernous sinus segment of internal carotid arteries bilaterally.

Coronal T1-weighted MRI reveals a 2.2 x 2.4 x 2.9 cm heterogeneously enhancing pituitary mass in the sella with suprasellar extension. The pituitary infundibulum is not visualized. There is mass effect on the optic chiasm and lateral displacement of the cavernous segment of internal carotid arteries. There is soft tissue packing along the floor of the sella from previous left sphenoid approach transsphenoidal resection of a pituitary macroadenoma. No pituitary apoplexy was noted in the radiologic report.

An ophthalmologic exam could not be performed due to altered mentation. Endocrinology recommended cosyntropin testing to assess for adrenal insufficiency. The following day, the patient developed a fixed and dilated right pupil, and was taken to the OR for emergent decompression. IV dexamethasone and levothyroxine were started post-operatively.

Conclusion

Differential diagnoses of pituitary apoplexy include bacterial meningitis and subarachnoid hemorrhage with aneurysmal rupture. A nonpupil-sparing third nerve palsy suggests a mass lesion. Given his symptoms and xanthochromia, this patient likely presented with pituitary apoplexy that was overlooked on initial scans. Hemorrhage that is iso-dense to normal brain tissue would not have the typical “pituitary ring sign”, a radiographic feature of peripheral enhancement around a non-enhancing infarcted center. Predisposing factors include hypertension, diabetes mellitus, pituitary dynamic testing, bromocriptine, estrogens or radiotherapy. This patient had multiple comorbidities. A lumbar puncture possibly worsened preexisting pituitary apoplexy, acutely expanding the area of hemorrhage and causing compressive symptoms.

References

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