

A Vascular Cause for Hypopituitarism

Dear Editor,

Pituitary tumours, infarction, surgery and radiotherapy cause hypopituitarism. Vascular etiologies are uncommon. We describe a patient with an internal carotid artery aneurysm who developed hypopituitarism following coil embolization.

A 42-year-old man presented with headache, diplopia and an episode of altered sensorium over 5 months. Tuberculous meningitis was diagnosed previously, based on cerebrospinal fluid (CSF) studies and he was started on antituberculous therapy. He had two recent episodes of epistaxis. On examination pressure of left lateral rectus palsy was noted.

Computerized tomography of the brain revealed a mass in the sphenoid sinus with erosion of surrounding bone and left parasellar extension. Magnetic resonance imaging showed internal carotid pathology with flow void lesions, corresponding to the left parasellar and petrous region (Figure 1). Cerebral angiography showed two aneurysms from the left internal carotid artery, (a) a fusiform aneurysm in the petrous segment, measuring 16 x 13 mm² (Figure 2) and (b) a giant saccular aneurysm of the cavernous segment, projecting into the enlarged sella, measuring 31 x 14 mm² (Figure 2) Coil embolization of the left internal carotid artery reduced the flow into the aneurysm, resulting in thrombosis. The patient was well at discharge, and antituberculous drugs were stopped.

Anorexia, vomiting and weight loss occurred over 3 months following the procedure. Examination revealed a low-blood

pressure of 90/70 mmHg, decreased body hair and testicular atrophy. Biochemistry confirmed the presence of panhypopituitarism (reference intervals in parentheses): 8:00 AM serum cortisol <1 (9–25 mcg/dl), total thyroxine: 3.45 (5–12 mcg/dl) and free thyroxine 0.63 (0.8–2.0 ng/dl), serum testosterone <20 (212–1 730 ng/dl), FSH 0.74 (0.7–11.1 mIU/ml), LH 0.55 (0.8–7.6 mIU/ml) and Prolactin 0.65 (2.5–17 ng/ml). His symptoms improved on replacement with glucocorticoids (Prednisolone 5 mg AM and 2.5 mg PM), thyroxine (0.1 mg/day) and testosterone (250 mg intramuscularly once monthly). He was well at follow-up 2 months later.

Hypopituitarism associated with aneurysms neighbouring the sella turcica is known.^[1–3] Presentations include blindness, weight loss due to hypocortisolemia,^[2] headache and cranial nerve palsies. Mechanisms for hypopituitarism in these patients are pressure effects, thrombosis,^[3] apoplexy of the pituitary adenoma,^[1] and following a subarachnoid haemorrhage.^[4] Pituitary dysfunction usually involves the pituitary-gonadal axis or pituitary-adrenal axis. Most intrasellar aneurysms arise from the internal carotid artery. Hypopituitarism following surgery of aneurysms is described.^[5] Our patient was unusual in that he developed overt hypopituitarism after embolization.

In patients with headache, cranial nerve palsy and altered sensorium, tuberculous meningitis can be rightly considered as a diagnosis. But rupture of a sellar aneurysm and pituitary apoplexy can have similar presentation. The CSF biochemistry can be misleading, as the protein is elevated in all these conditions. In the case of a sellar aneurysm (following a subclinical rupture) and apoplexy, it is due to blood in the CSF. This highlights the necessity of radiological modalities for accurate diagnosis.

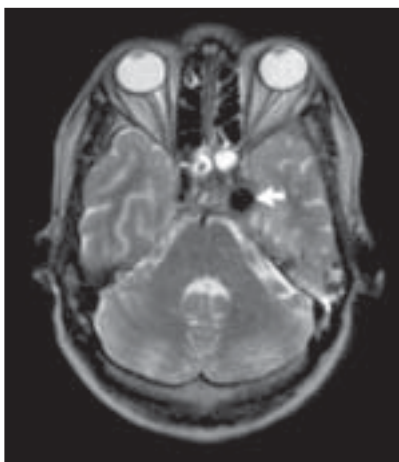


Figure 1: T2 Weighted magnetic resonance axial image, showing a flow void in the sella-left parasellar region (arrow)

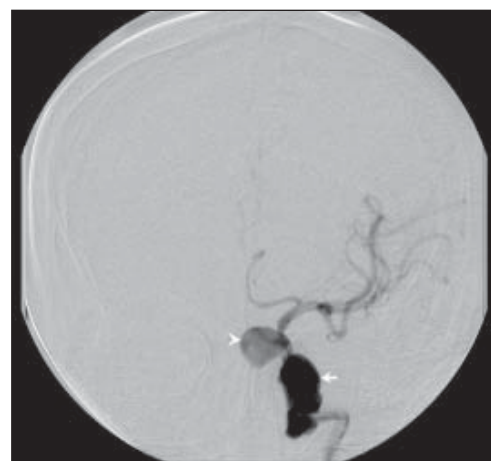


Figure 2: DSA image of the left internal carotid artery, frontal view showing two aneurysms: (A) a fusiform aneurysm in the petrous segment, measuring approximately 16 x 13 mm² (arrow) and (B) a giant saccular aneurysm of the cavernous segment measuring 31 x 14 mm² (arrowhead)

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