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- Books and Other Monographs

Personal author(s): Ringsven MK, Bond D. Gerontology and leadership skills for nurses. 2nd ed. Albany (NY): Delmar Publishers; 1996.

Editor(s), compiler(s) as author: Norman IJ, Redfern SJ, editors. Mental health care for elderly people. New York: Churchill Livingstone; 1996.

Chapter in a book: Phillips SJ, Whisnant JP. Hypertension and stroke. In: Laragh JH, Brenner BM, editors. Hypertension: pathophysiology, diagnosis, and management. 2nd ed. New York: Raven Press; 1995. p. 465-78.

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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.

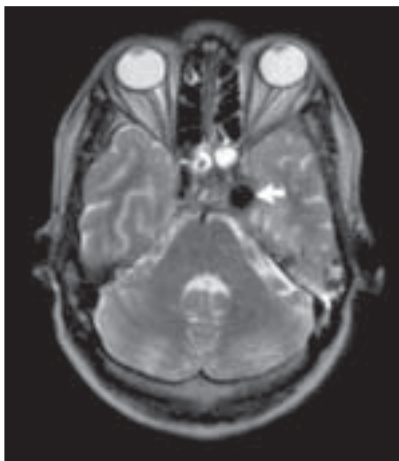


Figure 1: T2 Weighted magnetic resonance axial image, showing a flow void in the sella-left parasellar region (arrow)

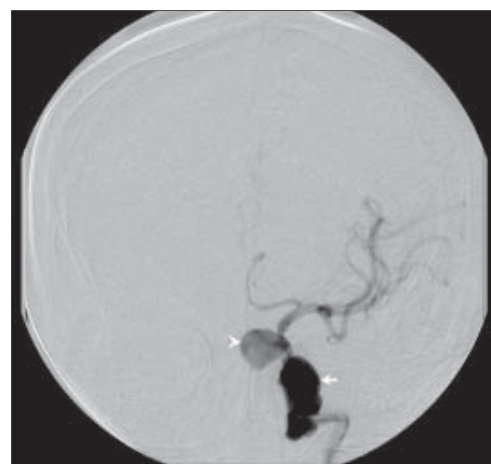


Figure 2: DSA image of the left internal carotid artery, frontal view showing two aneurysms: (A) a fusiform aneurysm in the petrous segment, measuring approximately 16 x 13 mm² (arrow) and (B) a giant saccular aneurysm of the cavernous segment measuring 31 x 14 mm² (arrowhead)

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PubMed ID :

Bilateral massive pleural effusion – a rare presentation of sarcoidosis

Sir,

A middle-aged lady presented to her physician with breathlessness on exertion, dry cough and weight loss. Histopathology of an enlarged right supraclavicular lymph node revealed necrotising granulomatous lymphadenitis. Hence with a diagnosis of tuberculosis adequate first line antituberculous therapy was indicated. As her symptoms worsened, Ciprofloxacin, Ethionamide and Kanamycin were added. These drugs were taken regularly for 13 months. Thereafter she discontinued all medications due to further worsening. Her chest radiograph showed bilateral interstitial shadows and small pleural effusions. Repeat right supraclavicular lymph node biopsy showed necrotising granulomas, and culture grew one colony of *Mycobacterium tuberculosis*. She was given Isoniazid, Rifampicin, Pyrazinamide, Sparfloxacin and Cycloserine. She still had no relief of symptoms despite good adherence to therapy for 11 months. She presented to us with worsening of breathlessness and dyspnoea at rest.

On examination, she was afebrile with tachypnea, tachycardia, central cyanosis, asterixis and bilateral pitting pedal oedema without elevation of JVP. She had three enlarged, firm, discrete, right supraclavicular lymph nodes. Chest examination revealed stony dullness to percussion over both lung bases with absent breath sounds over these areas. Rest of the physical examination was unremarkable.

Hemogram and blood biochemistry were normal. Mantoux test was negative. ABG showed pH- 7.41; pCO₂ 52.2mmHg; pO₂ 44.6mmHg; HCO₃ 32.6mEq/l and saturation of 80.3%. Chest

radiograph showed bilateral pleural effusions. HRCT scans [Figure 1] revealed nodularity and marked thickening, predominantly along the central peribronchovascular interstitium. There was moderate bilateral pleural effusion. Subcarinal, paratracheal and anterior mediastinal nodes were enlarged, up to 12mm with some exhibiting areas of peripheral calcification [Figure 2]. Pleural fluid analysis revealed an exudate with lymphocytic pleocytosis, bacterial, fungal and mycobacterial cultures were negative. Cytology was negative for malignant cells. Pleural biopsy revealed non-caseating granulomas and Mycobacterial culture of the pleural tissue was negative. Histology of right supraclavicular lymph node showed non-caseating granulomatous inflammation, and cultures were negative. Based on these clinico-radiologico-pathological features and non-response to anti-tuberculous therapy, she was diagnosed to have sarcoidosis.

She was initiated on corticosteroids and all antituberculous medications were discontinued. She had significant improvement of dyspnea and cyanosis disappeared. Repeat chest radiograph showed marked decrease in the effusion. Her follow up HRCT scans showed evidence of residual fibrosis with significant reduction in nodularity.



Figure 1: Chest radiograph showing bilateral large pleural effusions



Figure 2: HRCT thorax, mediastinal window: Image shows bilateral pleural effusion with mediastinal adenopathy (arrowheads)