LETTERS TO THE EDITOR

Unilateral proptosis secondary to partially thrombosed giant carotid ophthalmic artery aneurysm

A wide variety of conditions have been described as a cause of unilateral proptosis. The case we describe had unilateral proptosis due to partially thrombosed giant carotid ophthalmic artery aneurysm, the diagnosis of which was confirmed with angiography and magnetic resonance imaging (MRI). So far as we are aware, unilateral proptosis secondary to giant partially thrombosed aneurysm of the carotid ophthalmic artery demonstrated on MR has not been previously reported.

A 70 year old woman was admitted complaining of a gradual bulging of the right eye and loss of vision over a period of six months. The bulging of the right eye was painless and nonpulsatile. Except for cholelithiasis, she had no other past significant illness. On examination, the right eye showed nonpulsatile proptosis with complete loss of perception to light. Left eye had 6/18 vision with glasses. Fundus examination of the right eye showed primary optic atrophy. No other neurological deficit was found. Plain radiograph of the skull and orbits did not reveal any abnormality.

Contrast enhanced computerised tomography (CT) of the head and orbit showed a huge retrobulbar mass of the right side with mixed density with the area of enhancement associated with proptosis of the right eye.

Based on the CT findings, an aneurysm with partial thrombosis was suggested and angiogram was advised. A digital subtraction right carotid angiogram revealed an aneurysm at the origin of the ophthalmic artery from the internal carotid artery above the cavernous sinus with non filling of the ophthalmic artery (fig 1). MR was performed with a 1.5-T superconducting system (Magnetom, Siemens) using spin echo (SE) technique. It showed a flow void phenomenon on all SE sequences in the right carotid artery and patent portion of the lumen of the aneurysm. It was associated with laminated thrombus of mixed stages in the clotted

Figure 2 First-echo image of axial MR through the orbits (SE 2800/28) shows patent portion of the aneurysm arising from right internal carotid artery. Note the laminated thrombus of mixed stages (arrows) around the patent aneurysm causing proptosis.

portion of the lumen. It measured $3.8 \times 4 \times 3$ cm and filled the whole right retrobulbar space with proptosis (fig 2). Based on MRI and angiography, the diagnosis of right partially thrombosed giant carotid ophthalmic artery aneurysm was established.

Carotid ophthalmic artery aneurysms arise in the first 2 mm of the internal carotid artery above the cavernous sinus and below the origin of the posterior communicating artery. The aneurysm frequently originates beneath the optic nerve and may extend into and dilate the optic canal.¹ Due to their dual component nature, partially thrombosed giant aneurysms have a characteristic appearance on spin echo MRI and identification of the signal intensity pattern is specific for this lesion.² Our case showed all the characteristic features of a partially thrombosed giant aneurysm. A more complete delineation of the giant aneurysm components and associated extra aneurysmal parenchymal abnormalities is possible with MRI compared with either CT or angiography.

RK GUPTA

Institute of Nuclear Medicine and Allied Sciences, Lucknow Road, India

VS MEHTA*

NK MISRA†

Department of Neurosurgery* and Neuroradiology,† All India Institute of Medical Sciences, New Delhi, India

1 Peris JB, Ross Russell RW. Giant aneurysms of the carotid system presenting as visual field defect. *J Neurol Neurosurg Psychiatry* 1980;43:1053-64.

2 Atlas SW, Grossman RI, Goldberg HI, et al. Partially thrombosed giant intracranial aneurysms: correlation of MR and pathologic findings. *Radiology* 1987;162:111-4.

Figure 1 Right common carotid angiogram, lateral projection shows aneurysm at the origin of the ophthalmic artery (arrow).

Pituitary abscess with recurrent aseptic meningitis

Since the first description of pituitary abscess by Simmonds in 1914,¹ less than 40 cases have been adequately reported. The diagnosis has rarely been made before operation, the radiological findings being those of a pituitary tumour. We report a patient in whom the diagnosis of pituitary abscess was made pre-operatively, to draw attention to the association with aseptic lymphoplasmocytic menin-

gitis, which provided a clue to the diagnosis. This association has been observed only twice before.^{2,4}

In 1983 a 67 year old woman developed severe frontal headaches progressively worsening over several days. Her temperature was 38.5°C and she showed signs of meningism. The cerebrospinal fluid (CSF) was xanthochromic with a lymphoplasmocytic pleiocytosis and increased protein content, but bacteriological and virological investigations were negative, including tests for tuberculosis. Computed tomography (CT) showed moderate enlargement of the subarachnoid spaces. Viral meningitis was presumed and symptoms disappeared gradually with symptomatic treatment. In a second episode a month later, she lapsed into a coma for two days. The laboratory findings were similar, treatment was not modified and she recovered after several weeks.

One year later, headaches, slowed thinking and meningism recurred. Her sedimentation rate was raised. She had anaemia, polymorphonuclear leucopaenia and a relative lymphocytosis. She had circulating immune complexes and antinuclear antibodies, but tests for anti DNA antibodies were negative. Tests of thyroid function showed that TSH was low, with low free T3 but normal free T4. Her CSF had an increased protein content (1.2 mg/ml) and lymphoplasmocytosis (60 lymphocytes and 15 plasmocytes/ml), but glucose and chloride concentrations were normal. The CT was unchanged. She again recovered without specific treatment.

Six months later, she still had a lymphocytosis in her blood. Antinuclear antibodies and immune complexes were still present. Her free T3 had fallen and she was treated with 0.1 mg L-thyroxine daily. During the following months, she experienced several episodes of mental slowing, vertigo, orthostatic hypotension with falls and headaches but without hyperthermia or signs of meningism. She was referred to the Department of Neurology, University of Liège, in February 1987. She was moderately obese and hypertensive with brittle hair and thin eyebrows. There was no pubic hair and her prepubertal skin was infiltrated. She had a subjective bilateral global diminution of vision, with no apparent hemianopia, but the rest of the neurological examination was normal.

Routine blood and urine tests were normal. Antinuclear antibodies were still positive ($1/20$), but anti DNA antibodies and circulating immune complexes were negative. Endocrine tests showed normal basal thyroid function (with 0.1 mg L-thyroxine daily) with undetectable TSH and evidence of hypopituitarism. Injection test of TRH did not increase TSH values. There was only a slight increase in prolactin values (from 110 to 190 $\mu\text{IU/ml}$). Follicle-stimulating hormone (2.9 mIU/ml) and luteinising hormone (<2.5 mIU/ml) were subnormal for a post menopausal woman and Gn RH did not increase FSH and LH values. Cortisol and ACTH were below the minimal detectable values throughout the circadian cycle. Free urinary cortisol was low at 3 $\mu\text{g/day}$. CRF test was followed by no increase of ACTH or cortisol. Urinary gravimetry was normal.

Ophthalmological examination showed senile bilateral cataract with severe reduction of visual acuity, but no defect in her visual fields. Plain radiographs of the skull showed an enlarged sella with thinning of the right part of the floor; abnormal calcification was