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. 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 subtrac- tion 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 portion of the lumen. It measured 3.6 x 4 x 3 cm and filled the whole right retroorbital space with protein (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 aneurysmal components and associated extra aneurysmal parenchymal abnormalities is possible with MRI compared with either CT or angiography.

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Pituitary abscess with recurrent aseptic meningitis

Since the first description of pituitary abscess by Simmonds in 1914,4 less than 40 cases have been adequately reported. The diagnosis has rarely been made before operation, and the radiological findings being those of a pituitary tumour. We report a patient in whom the diagnosis of pituitary abscess was made preoperatively, to draw attention to the association with aseptic lymphoplasmocytic meningitis, which provided a clue to the diagnosis. This association has been observed only twice before.5

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, treatrotic hypotension with falls and headaches recovered after several weeks.

One year later, headaches, slowed thinking and meningism recurred. Her sedimentation rate was raised. She had anaemia, polymorphonuclear leucopenia and a relative lymphocytosis. She had circulating immune complexes and anitibodies, but tests for anti DNA antibodies were negative. Tests for 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/ml and 15 plasmocytes/ml), but glucose and chloride contents 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, frontal headaches and vision disturbance. Viral meningitis was still present. 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. Inmunonephelometric test of TRH did not increase TSH values. There was only a slight increase in prolactin values (from 110 to 190 µU/ml). Follicle-stimulating hormone (29 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 cortisol was low at 3 µg/day. CRF test was followed by no increase of ACTH or cortisol. Urinary gravimetry was normal. Visual acuity was decreased bilateral maculae, which 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