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Radiology in Focus

Giant aneurysm of the petrous internal carotid artery: diagnosis and treatment

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Abstract
We report the case of a giant fusiform aneurysm of the petrous internal carotid artery in a 15-year-old patient who had presented with headache, hearing loss and Horner's syndrome. Definitive radiological diagnosis was made by non-invasive imaging techniques, including magnetic resonance angiography (MRA). The aneurysm was obliterated by endovascular balloon occlusion following successful tolerance of test occlusion of the internal carotid artery.

Key words: Aneurysm; Carotid artery, internal; Horner's syndrome

Introduction

Giant fusiform aneurysms of the petrous internal carotid artery are rare. These lesions are, however, of relevance to otolaryngologists, since unilateral facial pain, hearing loss and haemorrhage are common presentations. Magnetic resonance imaging (MRI) and computed tomography (CT) demonstrates a mass in the petrous bone, the complex appearance of which may be misinterpreted and lead to a potentially fatal attempt at surgical resection. In this case CT, MRI, MRA and conventional angiography were performed prior to successful endovascular balloon occlusion of the internal carotid artery.

Case history

A 15-year-old schoolboy with a two-month history of 'headache' and unilateral deafness was referred by his general practitioner for an urgent ENT opinion. The boy described headache of increasing frequency and severity, located behind his left eye and pain 'deep' within the ear, which radiated into the neck. The only past history of note was a minor head injury three years previously, when he had fallen off his bicycle. This had not required medical attention. On examination, there was almost complete hearing loss on the left and a left Horner's syndrome but the other cranial nerves were intact.

Prior to admission to our neurosciences unit an enhanced CT and MRI of the brain was performed. A provisional diagnosis of a cholesterol granuloma was made, and the patient was referred for surgery. Review of the CT demonstrated an avidly enhancing mass centred upon, and markedly expanding the (L) petrous carotid canal with evidence of surrounding sclerosis (Figure 1). The MRI more clearly delineated the lesion that had extended outside the petrous apex into the middle cranial fossa and was indenting the adjacent temporal lobe. On the T2-weighted images, the petrous carotid presented as an expanded predominantly hypointense structure with amorphous 'swirls' of high signal (Figure 2). On the T1-weighted image the lesion was largely hyperintense (Figure 3) with irregular areas of lower signal, and enhancement was observed following the administration of intravenous gadolinium. The combination of signal abnormalities on the different sequences indicated thrombus and turbulent flow within an enlarged vessel. The

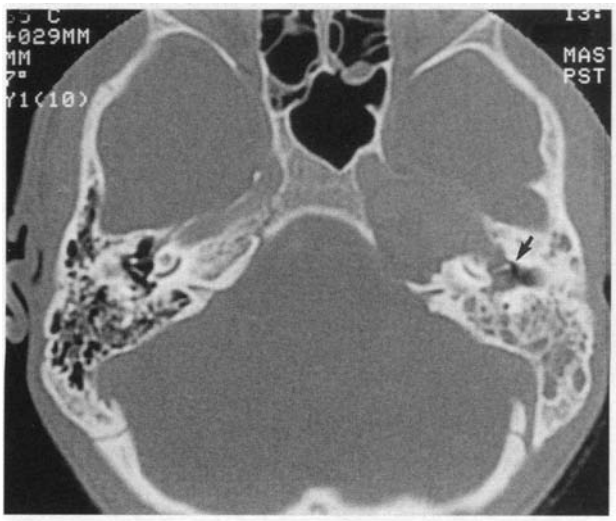

Fig. 1

Fine section axial CT of the petrous bone on bone windows demonstrates expansion of the entire length of the (L) petrous carotid canal. There is erosion into the adjacent middle cranial fossa and the lesion has expanded into the middle ear cavity (arrow). The latter is fluid-filled, as are the mastoid air cells, due to Eustachian tube dysfunction.

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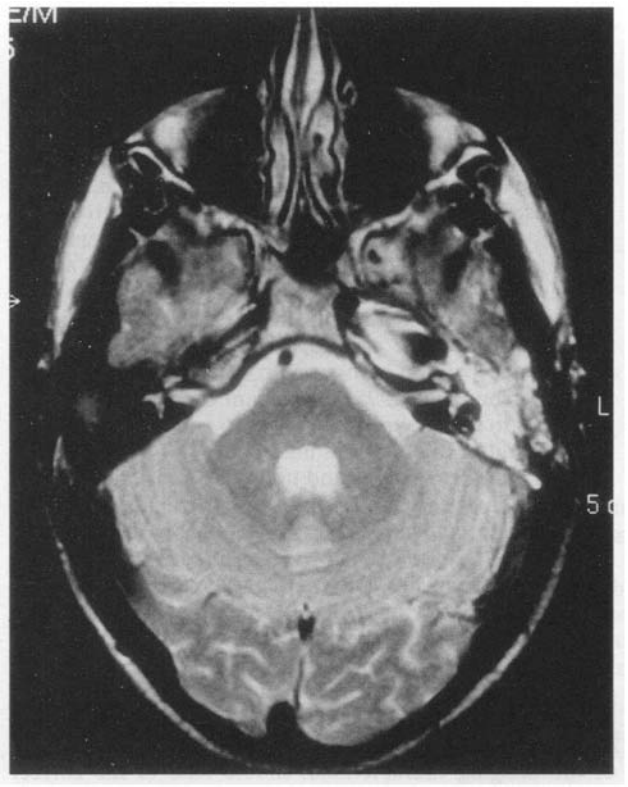

FIG. 2

Axial T-2 weighted MR (TR 2413 ms, TE 120 ms) at the level of the internal auditory canals depicts the petrous carotid aneurysm as an expansile structure of mixed signal which is predominantly hypointense with areas of amorphous peripheral high signal. The high signal is due to the presence of thrombus and complex intralesional flow. High signal is noted in the mastoid air cells.

mastoid air cells were hyperintense on the T2 sequence due to obstruction of the Eustachian tube.

Following admission to our institution, MRA and conventional percutaneous arteriography (Figure 4) were performed and confirmed the presence of a giant thrombus-containing fusiform aneurysm of the left petrous carotid. The expansion of the vessel extended from the distal cervical internal carotid artery (ICA), at a point just beneath the skull base, to the origin of the cavernous segment of the ICA.

Endovascular occlusion was performed the following day. A preliminary transfemoral test occlusion of the left ICA was conducted using a 7.5 mm non-detectable balloon (Boston Scientific, Fremont, CA). A test balloon was advanced up the left ICA to the point immediately proximal to the origin of the aneurysm through a 7-French guiding catheter (Medtronic Micro Interventional Systems, Sunnyvale, CA). Under systemic heparinization (5000 iu/70 kg) the balloon was inflated for 30 minutes. During this period the patient was examined by a neurologist and was not observed to develop any additional neurological deficit. During the test occlusion, right internal carotid and left vertebral arteriograms, performed via the contralateral femoral artery, confirmed complete filling of the vascular supply to the left hemisphere, which occurred without delay in venous drainage. A minimal fall in the mean systolic velocity of the left middle cerebral artery (MCA) from 65 to 60 m/s was recorded by transcranial Doppler.

The test occlusion catheter was withdrawn and replaced by a 7.5 mm ∉ 13.5 mm detachable silicone balloon (Boston Scientific). The balloon catheter was advanced

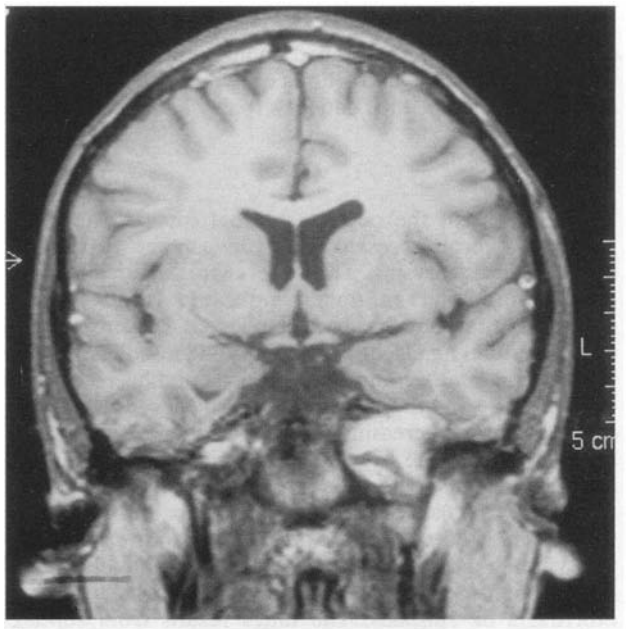

FIG. 3

Coronal non-enhanced MR (TR 550 ms, TE 20 ms) demonstrates the lesion as almost completely high signal due to flow-rated vascular enhancement. This appearance was initially mistaken for a cholesterol granuloma.

to the exact position at which the test occlusion was conducted, using an angiographic 'road-map' facility. The balloon was then inflated under direct fluoroscopic visualization. A second balloon was deployed immediately proximal to the first as a safety measure. Injection via the guiding catheter confirmed complete occlusion of the distal left ICA. Left vertebral arteriography demonstrated continued filling of the left MCA and patency of the ipsilateral cavernous carotid and ophthalmic artery. Only a sliver of contrast entered the left petrous carotid on the delayed images.

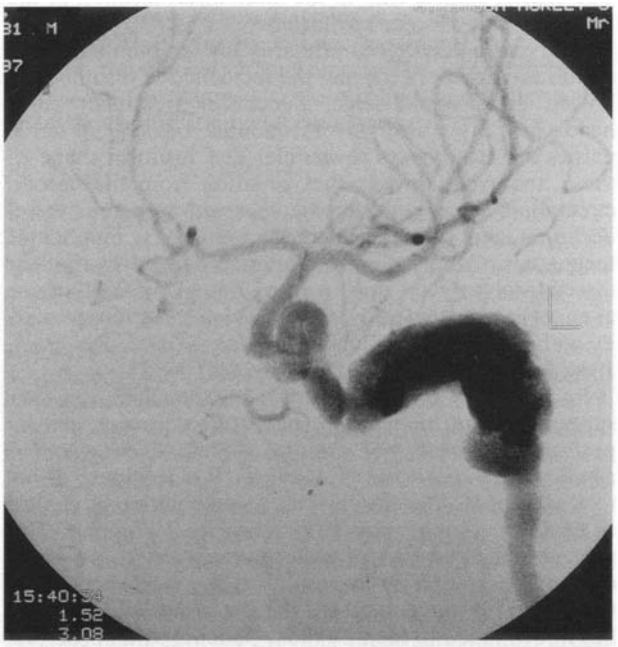

FIG. 4

Oblique AP conventional arteriogram of the (L) internal carotid artery confirms the presence of the giant fusiform aneurysm. The lesion extends from the skull base to the cavernous carotid artery.

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For 24 hours following the procedure, intravenous (iv) heparinization was continued maintaining the ratio at 2–2.5 times baseline and 4 litres of iv fluid was prescribed. Within 48 hours the patient's pain had dramatically improved.

Discussion

Aneurysms of the petrous internal carotid artery are rare and typically present at a younger age than other intracranial aneurysms. They are characteristically giant and fusiform and an underlying cause is not usually identified but trauma has been implicated. Patients present with unilateral headaches (often misdiagnosed for several years as migraine), cranial nerve palsies and haemorrhage. Palsies of the eighth (43 per cent), sixth, seventh and fifth nerves occur in decreasing order of frequency (Halbach *et al.*, 1990). Only one previous case of Horner's syndrome has been reported with a giant petrous aneurysm (Wemple and Smith, 1966). This is surprising in view of the intimate relationship of the petrous carotid artery to the surrounding sympathetic fibres to the orbit. Approximately one in four patients will present with haemorrhage, either otorrhagia or epistaxis. The latter results from rupture into the middle ear and drainage down the Eustachian tube. Patients do not seem to be at high risk of thromboembolism despite the presence of intraluminal thrombus (Halbach *et al.*, 1990).

A number of patients have died following imprudent surgical biopsy or attempted 'tumour' resection. A definitive pre-operative radiological diagnosis is, however, always possible, since a giant petrous aneurysm is readily demonstrated angiographically, either by conventional catheter arteriography or non-invasive MR angiography. Pointers to the correct diagnosis on cross-sectional imaging are i) the location of the lesion, which is centred upon, and orientated within the carotid canal, ii) fusiform, well-corticated expansion of the canal, iii) enhancement of the lesion with contrast (except for regions of thrombus) and iv) the MRI demonstrates complex mixed signal, which is not concordant in each plane, due to flow within the lesion. The crucial clinical clue to the anatomical location of the lesion was the Horner's syndrome.

Intervention is offered primarily for symptomatic relief and in the hope of reducing the incidence of thromboembolism and haemorrhage. Successful treatment cures headaches, often after several months, but cranial nerve palsies are not always reversible. The fusiform shape of these aneurysms means that isolation from the carotid circulation can only usually be achieved by parent vessel occlusion and this has been achieved by a number of techniques. Common carotid/internal carotid ligation or direct local surgery is performed but increasingly these aneurysms are treated by endovascular occlusion, primarily with the use of detachable balloons (Berenstein *et al.*, 1984; Halbach *et al.*, 1990; Larson *et al.*, 1995).

Endovascular carotid occlusion has the advantage over surgical ligation that a controlled test occlusion can be performed immediately prior to definitive occlusion to assess tolerance to vessel sacrifice. We routinely assess tolerance of ICA occlusion with a combination of clinical assessment, angiography and transcranial Doppler. The principal complication of balloon occlusion is ipsilateral cerebral infarction as a result of hypoperfusion, even where patients have tolerated the test occlusion. Attempts have been made to identify those patients with borderline collateral reserve by inducing hypotension during test occlusion, measuring ICA stump pressures or using various qualitative and quantitative measures of regional cerebral perfusion, such as ^{99m}Tc-HMPAO SPECT and stable Xenon CT. The efficacy, however, of these more complex, subsidiary techniques is not established (Niimi *et al.*, 1996). Reports estimate the incidence of subsequent infarction is in the order of four per cent for cavernous carotid aneurysms treated by ICA-occlusion (Higashida *et al.*, 1990). This figure compares favourably with surgical data, which suggests that there is a 17 per cent long-term incidence of stroke with ICA ligation (Roski *et al.*, 1981). Endovascular balloon occlusion would now seem to be the method of choice for unclippable giant carotid aneurysms.

Long-term follow-up of occluded giant petrous aneurysms is not well documented. In the short term, however, there is a very low incidence (three per cent) of continued filling of unclippable carotid aneurysms following placement of the balloon across or proximal to the aneurysm neck (Larson *et al.*, 1995). Placement of an initial balloon just distal to the aneurysm, in order to anatomically isolate the lesion at both ends does not appear to be necessary in the vast majority of cases and it also risks dislodgement of thrombus from the aneurysm during manipulation of the catheter. Rarely the aneurysm may remain partially patent, filling from the distal ICA or via caroticotympanic, vidian or periosteal branches. Such patients are candidates for the surgical placement of a distal 'trapping' clip above the aneurysm or even embolization with detachable coils from the contralateral carotid artery.

In summary, petrous aneurysms are rare lesions that present as an expansile fusiform mass and cause local pain, cranial nerve dysfunction or haemorrhage. Careful analysis of the imaging characteristics should lead to the correct diagnosis being made prior to surgery so that an endovascular procedure may be considered. Endovascular balloon occlusion appears to be the treatment of choice at the present time, since in experienced hands it is relatively safe, minimally invasive and results in symptomatic relief.

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