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Case report

Traumatic carotico-cavernous fistula presenting as delayed epilepsy

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Carotico-cavernous fistula typically presents as a pulsatile proptosis with chemosis, diplopia and a bruit. It is a recognised complication of facial trauma as well as head injury,[1] and it has been reported after closed head injury (particularly deceleration types),[2] basal skull fracture, mid facial fractures and surgery, and isolated mandibular fracture.[3-7]

CASE REPORT. A 22-year-old man was transferred from a peripheral hospital to the neurosurgery department following a car accident in which he had been a front seat passenger. Prior to transfer he had been assessed on the Glasgow coma scale at 7/14 (he did not open his eyes, made incomprehensible sounds, but localised to painful stimuli) and had undergone endotracheal intubation. There were bilateral periorbital haematomas, his pupils were small but reacting. Blood obscured the right tympanic membrane. The maxillary bone was mobile, and there was transient CSF rhinorrhoea.

X-rays showed fractures of the middle third of the face in the naso-ethmoidal region and separation of the fronto-zygomatic suture on the right. On the left the fractures extended through the floor and lateral wall of the maxillary antrum. CT scan revealed left fronto-basal contusion with associated cerebral oedema and small ventricles.

He was paralysed and artificially ventilated in the intensive care unit, and the intracranial pressure monitored. Over the following three days his sedation was reduced and at extubation his Glasgow coma scale was assessed at 13/14 (speech was confused). Further assessment of the facial injuries showed minimal displacement and limited mobility of the fractures and the occlusion of the teeth was good. Surgical intervention was therefore not warranted. Neurological examination revealed anosmia, and reduced hearing in the right ear. There was mild diplopia. Recovery continued and he was discharged from hospital three weeks after the accident. At review he remained well and reported that the diplopia had completely resolved, but that he noticed a slight buzzing in the right ear.

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Thirteen months following the injury he presented to the accident and emergency department of another hospital having had an epileptic seizure during sleep. He suffered no obvious neurological deficit at that time and was commenced on carbamazepine. CT scan showed a well-defined area of low attenuation in the left frontal lobe in keeping with the previous contusion. There was also a vascular abnormality, which with enhancement showed enlarged vessels over the right cerebral hemisphere particularly marked in the region of the right middle cerebral artery (Fig 1).

A systolic bruit was audible over the right eye, and in the right and left frontal and temporal regions. There was mild asymmetry of the orbits, the right eye being more prominent than the left but there was no palpable pulsation, no chemosis, and fundoscopy was normal. Visual acuity had not deteriorated and ocular movements were full. Audiometry revealed decreased hearing in the right ear and he again described a constant buzzing noise in this ear. He had also been aware at times of a pulsatile noise in the head.

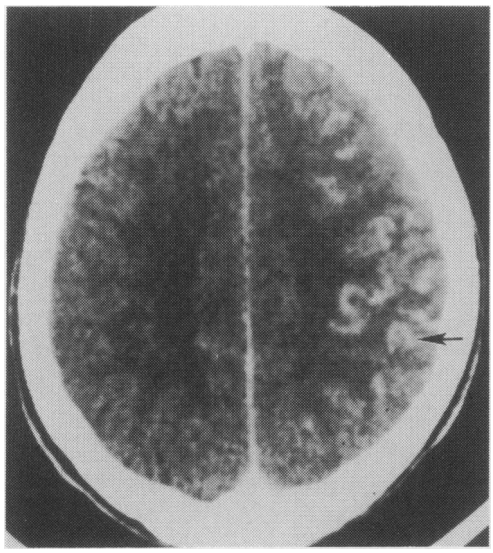

Fig 1. CT scan following intravenous contrast showing dilated vessels in right cerebral hemisphere (short arrow).

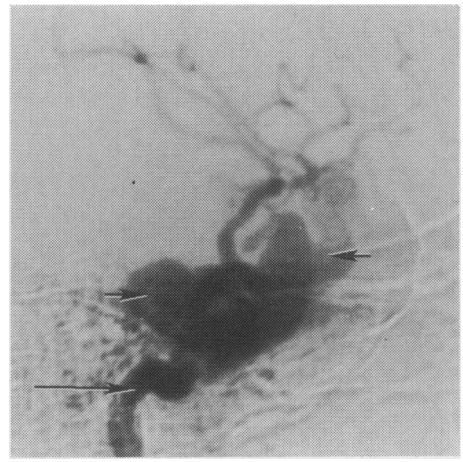

Fig 2. Right carotid arteriogram, lateral view. Contrast passing from right internal carotid artery (long arrow) to venous sinuses (short arrows).

On arteriography contrast passed directly from the right internal carotid artery into the cavernous sinus, and then into the petrosal sinus and some dilated superficial veins (Fig 2). There was incomplete filling of the ophthalmic veins which probably accounted for the absence of the classical clinical findings of a carotico-cavernous fistula (pulsating exophthalmos, chemosis and optic disc changes). There was good cross-circulation from the left internal carotid artery through the circle of Willis. The right internal carotid was therefore occluded using two detachable balloons which were placed as close to the fistula as possible.

DISCUSSION

This fistula only came to attention following an epileptic seizure 13 months after

the accident. Post-traumatic epilepsy following head injury and maxillofacial trauma is well recognised.[8] Late epilepsy, defined as a seizure occurring after the first week, may occur for the first time some years after the head injury. Over 50% of those destined to develop late epilepsy will do so within the first year. Factors predisposing to late post-traumatic epilepsy include post-traumatic amnesia of more than 24 hours, intra-cranial haemorrhage, the occurrence of early epileptic seizures, dural damage and depressed fracture.

Epilepsy is also a presenting feature of other intra-cranial lesions such as tumours or arteriovenous malformations. We have found no other cases of carotico-cavernous fistula presenting with an epileptic seizure. Kanno et al,[9] reported a case where a carotico-cavernous fistula presented with subarachnoid haemorrhage five years after a traumatic event. The epileptic seizure in this case was probably post-traumatic in origin, and the carotico-cavernous fistula discovered fortuitously on the repeat CT scan.

The cavernous sinus lies lateral to the pituitary fossa and medial to the sphenoid bone, and tapers anteriorly to the superior orbital fissure. The carotid artery is mobile within the sinus but it is tethered at its entrance and exit. The IIIrd, IVth and Vth cranial nerves as well as the ophthalmic and maxillary divisions of the Vth cranial nerve pass through the sinus. It receives the superior and inferior ophthalmic veins, the central retinal vein, and the middle cerebral veins, and is drained by the superior and inferior petrosal sinuses, which in turn drain into the internal jugular vein. A carotico-cavernous fistula allows shunting of blood from the high pressure internal carotid artery to a low pressure system, usually resulting in engorgement of the ophthalmic venous system with congestion and oedema of the orbital tissues. In this case none of the ophthalmic features were present, and there must have been an anomalous venous drainage of the area which favoured blood flow to the petrosal sinus and superficial veins rather than to the ophthalmic venous system. The apparent prominence of the right eye was more probably due to a mild enophthalmos on the left side secondary to the facial fractures.

The aims of treatment are to correct the orbital signs and symptoms, to prevent neurological damage due to pressure effects within the cavernous sinus, and to avoid subarachnoid haemorrhage or catastrophic epistaxis.[10] Observation alone may be considered for a spontaneous fistula in an older patient, but traumatic fistulas are actively managed. Treatment options have included ligation of the internal carotid artery, with or without muscle embolisation and direct surgical closure of the fistula. The use of the detachable intravascular balloon has now become the treatment of choice.[11, 12]

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