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Surgical management of an unusual carotid-cavernous fistula

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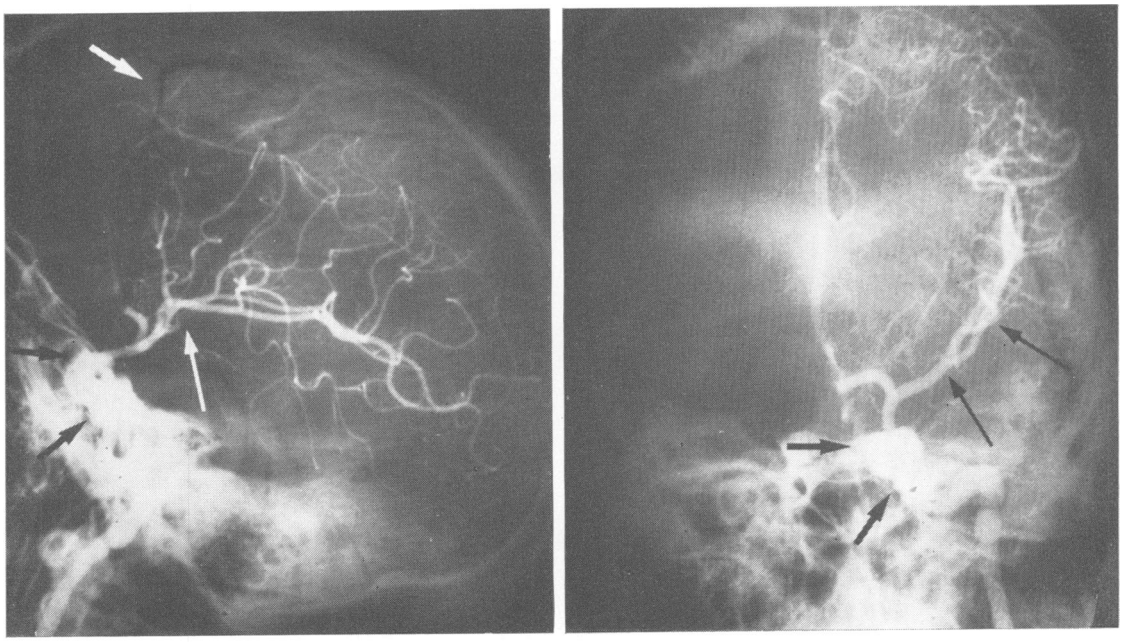
SUMMARY A case of carotid-cavernous fistula is described associated with extradural haematoma, temporal lobe contusion, ophthalmoplegia, optic nerve injury by fracture, rhinorrhoea, orbital encephalocoele, and traumatic carotid thrombosis. The fistula was occluded by clipping of the carotid artery intracranially and also of the ophthalmic artery.

Although many procedures have been proposed to treat carotid-cavernous fistula, there is not as yet a single method suitable for all cases. Direct attack on the cavernous sinus, carotid ligation in the neck, intracranial carotid occlusion, embolization, or combinations of these procedures, each carry some risk of complications, or of recurrence of the fistula. Obviously each case has to be considered on its merits, after full angiographic studies. The case described had a unique combination of features which, however, made successful treatment possible.

CASE REPORT

A 35 year old male was admitted after a car accident, in deep coma, reacting to painful stimuli and



(a) (b)

FIG. 1. *Emergency left carotid angiogram.*

with a mild paresis on the right side. There was swelling and proptosis of the left eye, with a dilated pupil not reacting to light. Straight radiographs showed a left frontal fracture radiating across the roof of the orbit to the vicinity of the sella turcica. Emergency left carotid angiography (28 March 1972) showed a carotid-cavernous fistula, and a shift of the anterior cerebral artery, with upward displacement of the middle cerebral artery (Fig. 1). At operation a left extradural haematoma was evacuated, and contusion of the temporal lobe was noted.

After operation the patient made a steady recovery, and a month later had no hemiparesis, and could walk without aid. Left exophthalmos (not pulsating) persisted, with involvement of III, IV, and VI cranial nerves. The left pupil did not react to light and there was total blindness in that eye. On the left upper eyelid a soft mass of the size of a hazel-nut was present. Neurological examination, including mental state, was otherwise normal.

Bilateral carotid angiography (27 April 1972) now showed that the left internal carotid artery was occluded in the neck; but the carotid-cavernous fistula remained unchanged and was filling via the external carotid artery and the ophthalmic artery. On the right side an excellent cross-filling circulation through the anterior communicating artery showed satisfactory vascularization of the left hemisphere (Fig. 2).

On 21 June 1972 a left frontotemporal flap was turned down. When the frontal lobe was elevated extradurally a bone defect appeared on the orbital roof. A brain hernia inside the orbit was removed and the bone defect closed with a prosthesis of acrylic material. The left optic nerve had been crushed inside the optic canal by the fracture of the orbital roof. The left internal carotid artery was occluded with a Scoville clip proximal to the posterior communicating artery. With a small rongeur and the aid of the dissecting microscope the optic canal was opened and the optic nerve divided, to expose the ophthalmic artery which was clipped.

The postoperative course was uneventful. Bilateral angiography on 3 July 1972 showed no sign of the fistula. The exophthalmos subsided immediately, the ophthalmoplegia recovered and, apart from the blindness of the left eye, no neurological or mental deficiencies were detected when the patient was discharged.

COMMENTS

Because of the poor state of the patient when admitted no immediate attempt was made to treat the carotid-cavernous fistula. The primary evacuation of the extradural haematoma and subtemporal decompression were necessary to save his life. When the oedema and haematoma of the left orbit had subsided and the patient was awake and cooperative, proptosis and ophthalmoplegia were discovered with completed blindness consecutive to injury of the optic nerve in the optic canal associated with a traumatic encephalocoele and cerebrospinal fluid leaking through the left nostril, probably via the frontal

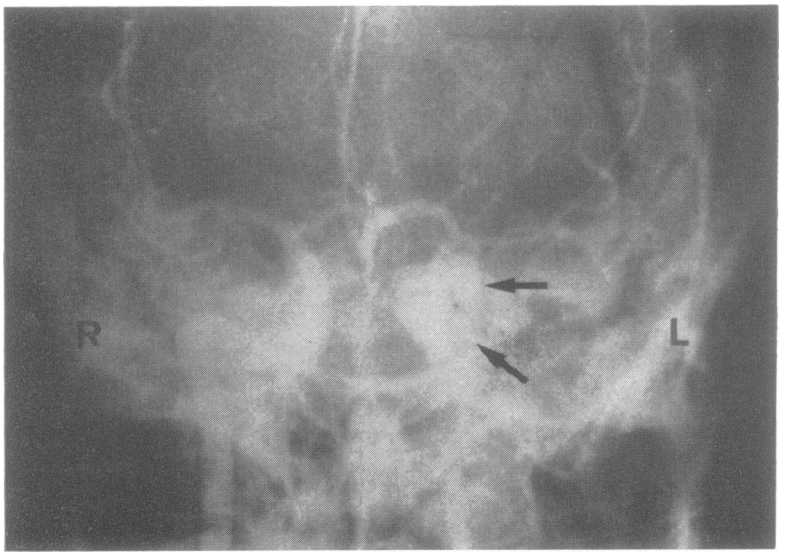


FIG. 2. *Right carotid angiogram. Excellent cross-filling circulation. The fistula is filled from the right side.*

sinus. As bruit and pulsation were absent, direct trauma could have been responsible for the exophthalmos. It was therefore considered necessary to carry out a bilateral angiogram to prove the persistence of the carotid-cavernous fistula and to evaluate the circulatory conditions from the other side. An unexpected spontaneous thrombosis in the left internal carotid artery was found which was thought to have been traumatic in origin. The patency of the carotid-cavernous fistula in spite of this event proved that a carotid ligation in the neck would have been useless.

A further operation was judged advisable to deal with the orbital swelling which was increasing, to stop the rhinorrhoea, and to close the arteriovenous shunt. As there was no neurological deficit, cross-circulation was presumably adequate to prevent ischaemia of the left hemisphere, but the 'steal' by the fistula was considered a potential threat to the blood supply of both hemispheres.

All the embolization procedures described to occlude such a fistula (Isamat *et al.*, 1970) were ruled out because of the internal carotid thrombosis. Simple clipping of the carotid artery inside the skull, as advised by Dandy (1935), was estimated to be an insufficient measure because of the good filling of the fistula through the ophthalmic artery. A determined effort was made to occlude this vessel by removing the anterior clinoid process, as advocated by Pool (cited by Hamby, 1966). This step was facilitated by the use of the microscope and by the blindness of the left eye which permitted section of the optic nerve. In this case the exit of the ophthalmic artery was very low and was lying underneath the optic nerve. In a study of necropsy material, Kempe (1968) found this position of the ophthalmic artery in 80% of the cadavers studied. If the complete blindness had not allowed section of the nerve, as advised by Tönnis (1960), occlusion of the ophthalmic artery in this particular case would have risked injury to the nerve.

After the operation ophthalmodynamometry was carried out and the tension of the central retinal artery was estimated to be within normal limits. This proved the efficiency of the anastomotic circulation from the external carotid artery in supplying the retina, and would probably have resulted in preservation of the visual acuity if the optic nerve had not been damaged. Walsh and King (1942) found that collateral circulation via the external carotid artery was sufficient to protect vision even if the thrombosis of the internal carotid artery extended to the exit of the ophthalmic artery. For this reason ophthalmodynamometry is of limited value as a diagnostic tool in carotid occlusion or stenosis. Young and associates (1964) found good correlation between arteriographic evidence of carotid stenosis and low retinal arterial pressure in only 49% of the patients studied.

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