

RADIONUCLIDE ANGIOGRAPHY IN THE DIAGNOSIS OF CAROTID CAVERNOUS SINUS FISTULA

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Radionuclide cerebral angiography can be used to diagnose accurately carotid cavernous fistula. The use of this procedure in the evaluation of treatment of carotid cavernous fistula is stressed. The differential diagnosis of pulsating exophthalmos, cavernous sinus thrombosis, retro-orbital tumor, arterial or venous aneurysms, and arteriovenous malformations is discussed.

Radionuclide cerebral angiography is a well accepted modality for the diagnosis and treatment evaluation of many intracerebral conditions. The usefulness of this procedure in the evaluation of intracerebral vascular abnormalities has received wide attention (1). Two recent patients with post-traumatic carotid cavernous fistula have come to our attention. The radionuclide angiogram in each case was considered diagnostic. The purpose of this communication is to show how radionuclide angiography can be utilized in the differential diagnosis of this entity and to stress the simplicity and accuracy of this procedure for the evaluation of treatment.

CASE REPORTS

Case 1. A 56-year-old white man sustained a self-inflicted gunshot wound to the left face on June 8, 1973. Surgical closure was performed. Postoperatively, the patient had a left peripheral seventh nerve palsy and a right arm hemiplegia. The patient was transferred to this hospital on June 11, 1973 at which time chemosis of the left eye and a poorly reactive pupil were noted. A brain scan and cerebral blood flow study, using 15 mCi of ^{99m}Tc -pertechnetate was performed on June 14, 1973 (Fig. 1). A diagnosis of left carotid cavernous fistula was sug-

gested from this study. Thereafter, a neurosurgical consult was obtained and an orbital bruit was discovered. On June 23, 1973 a left carotid angiogram confirmed this diagnosis (Fig. 2). The predominant drainage of the fistula was through the superior ophthalmic vein. On June 27, 1973 craniotomy with clipping of the left supraclinoid carotid with embolization and ligation of the internal carotid in the neck was performed. Postoperatively the bruit disappeared. Visual acuity in the left eye was poor and ophthalmoplegia persisted. The patient died on August 31, 1973 of a pulmonary embolus. No evidence of recurrence of the fistula was documented during life or at necropsy.

Case 2. A 52-year-old woman had a history of many previous hospital visits for various injuries, usually following beatings by her husband. She was assaulted on May 28, 1973 mainly about the left side of the face. Two days after the assault she noted a "swishing" sound in her left ear. This symptom persisted through several clinic visits until she was examined on August 8, 1973. At that time a bruit was noted over the left ear and carotid. No exophthalmos was observed. A brain scan and cerebral blood flow were performed on August 17, 1973 following the intravenous injection of 15 mCi of ^{99m}Tc -pertechnetate (Fig. 3). The study was interpreted as compatible with a left carotid cavernous fistula. On August 28, 1973 a bilateral carotid angiogram was performed and confirmed this diagnosis (Fig. 4). The major venous drainage was through the inferior petrosal to the internal jugular vein with a lesser

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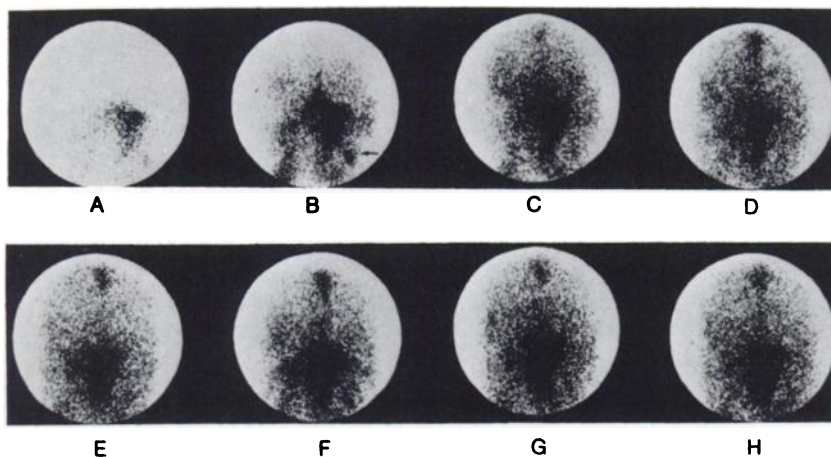


FIG. 1. Radionuclide cerebral angiogram in Case 1 demonstrates rapid accumulation of activity in area of intracavernous portion of left carotid artery (A). This is followed by accumulation over left orbit (B), which quickly regresses and disappears on venous phases (E-H). Another area of increased activity (see arrow ←, B) corresponds to external jugular vein on carotid angiogram (2B, C).

amount through the ophthalmic plexus and sphenoparietal sinus. Surgical correction was suggested but the patient declined and was discharged.

DISCUSSION

Pulsating exophthalmos was first described by Travers (1809) but a postmortem examination was not performed and the condition was attributed to a cirroid aneurysm of the orbit. Guthrie (1823) performed the first necropsy for pulsating exophthalmos and found an aneurysm of the ophthalmic artery which he regarded as the underlying cause of the

condition. It was not until 1835 that Baron, at necropsy, discovered and described a communication between the internal carotid artery and the cavernous sinus. Since then numerous reports have appeared describing the pathophysiology of this condition (2).

From these reports the concept of two general causes of carotid cavernous fistula emerge. First are those that are thought to be traumatic in origin usually related to a skull fracture or penetrating cranial injury (3) but fistula secondary to carotid endarterectomy has also been reported (4). Post-traumatic fistulas comprise approximately three-fourths of all carotid cavernous fistulas (2). The remainder are considered spontaneous and a large number of predisposing factors may exist in this group. Spontaneous fistulas are strikingly more frequent in hypertensive pregnant women. Rupture of an intercavernous aneurysm has been reported but intracavernous aneurysms are quite uncommon (3). Inherent weakness of the carotid artery due to atherosclerosis has been suggested as a cause. True weakness of the arterial wall in Ehlers-Danlos syndrome and related primary mesodermal disorders have been the cause of some spontaneous carotid cavernous fistulas (5). Finally, carotid cavernous fistula has been reported as a complication of cavernous sinus thrombophlebitis (2).

The differential diagnosis of carotid cavernous fistula in uncomplicated cases is usually not difficult when the symptom complex of pulsating exophthalmos, orbital bruit, headache, visual failure, and extraocular nerve palsy is present. The suspected diagnosis is further supported by noting relief of symptoms following carotid compression.

A number of other problems should be considered in the differential diagnosis, however. These include cavernous sinus thrombosis which has no evidence of bruit or pulsation and for which an antecedent



FIG. 2. Left lateral carotid angiogram in Case 1 shows arterial venous fistula in area of cavernous sinus (A). Predominant draining is through large tortuous superior and inferior ophthalmic veins, which drain into facial and external jugular veins.

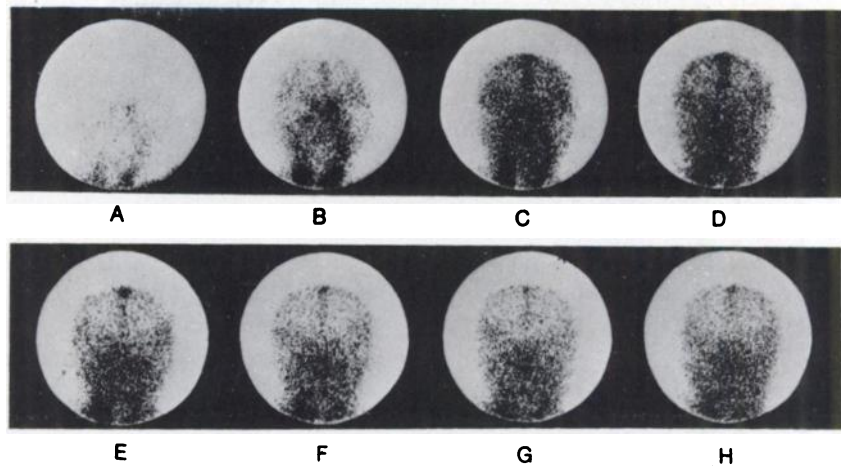


FIG. 3. Radionuclide cerebral angiogram in Case 2 shows early focal area of increased radioactivity in area of cavernous sinus (B), which persists momentarily, then disappears completely during venous phases (D-H).

history of infection is easily obtained. Retro-orbital tumors generally have an insidious onset with no evidence of bruit or pulsation. True orbital arterial or venous aneurysms are distinctly rare but do present with pulsating exophthalmos and may have a bruit. The diagnosis is generally established by contrast angiography. Arteriovenous malformations of the orbit are very rare but have clinical symptoms identical to carotid cavernous fistula. Again the diagnosis is generally made by contrast arteriography.

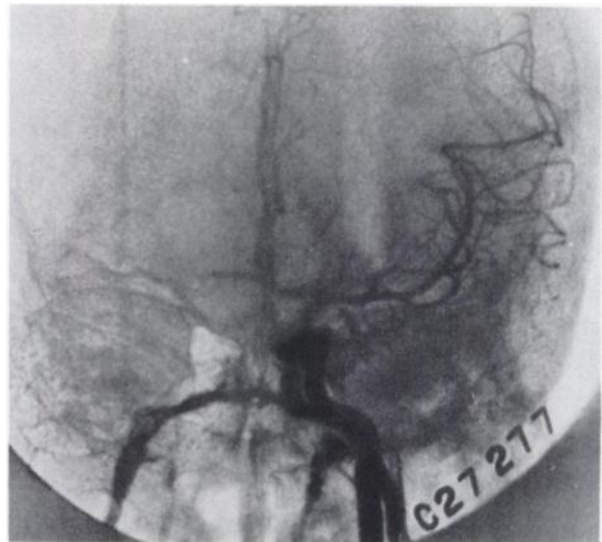
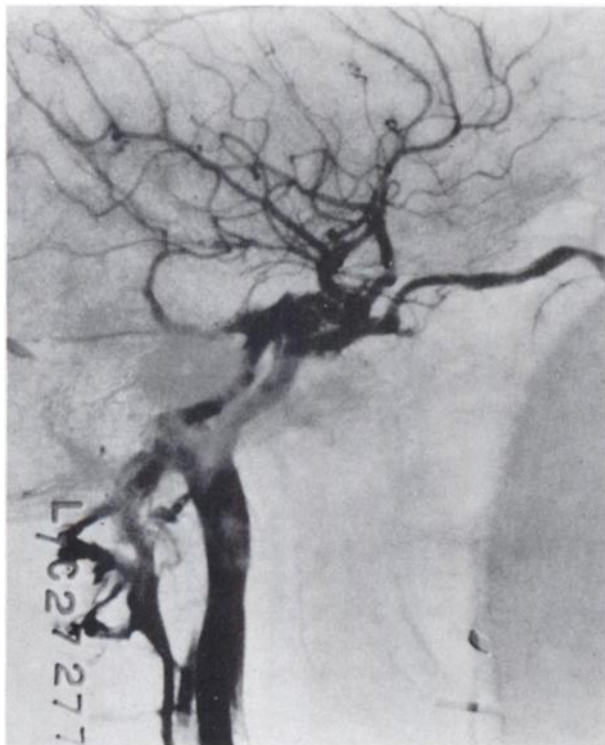
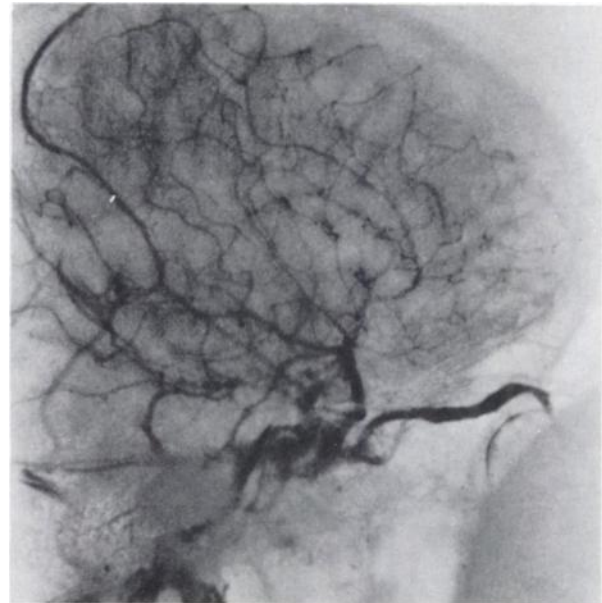


FIG. 4. A-P and lateral left carotid angiogram in Case 2 shows fistula between internal carotid artery and cavernous sinus (A). Venous drainage is mainly through inferior petrosal to internal

jugular and occipital veins. There is some drainage to ophthalmic vein and sphenoparietal sinus (B). A-P view demonstrates flow to opposite side from communication between cavernous sinuses (C).

The venous outflow patterns of carotid cavernous fistulas have been the subject of much discussion (6). The following pathways have been described.

1. Superior ophthalmic vein to the facial veins and the inferior ophthalmic vein to the pterygoid plexus and facial veins
2. Superior petrosal vein to the sigmoid sinus
3. Basilar vein to the straight sinus
4. Sphenoparietal sinus and cerebral veins to the sagittal sinus
5. Inferior petrosal sinus to the internal jugular vein

In most cases of spontaneous carotid cavernous fistula, the drainage is exclusively through the ophthalmic veins (6). In traumatic fistulas a great variability in venous outflow is seen, probably related to the location of the fistula and subsequent thrombosis formation (2).

The treatment of carotid cavernous fistula depends on the clinical status of the patient. Conservative external compression of the carotid may be the only method available for patients who are unlikely to survive surgery. The success rate is disappointingly low, however. Spontaneous closure occurs in less than 4% of patients. At present the surgical treatment of choice would appear to be a "trapping" operation in which internal and external occlusion of the internal carotid artery is performed in conjunction with an embolization procedure to occlude the ophthalmic artery. This method results in a high success rate and prevents continued feeding of the arteriovenous fistula by the ophthalmic artery (3).

The role of radionuclide angiography has not received its due recognition in the diagnosis and management of carotid cavernous fistula. A single case report was offered by Curl, et al (7), who also emphasized the value of the study in evaluation of treatment, particularly in the early postoperative period when carotid angiography may be technically difficult or even hazardous.

A case of nontraumatic external carotid cavernous sinus fistula which was diagnosed only by selective catheterization of the external carotid artery has been reported (8). The author suggested from this

case that the lesion could be missed with contrast internal carotid angiography alone. Radionuclide angiography, however, should detect this rare lesion.

In the differential diagnosis of carotid cavernous fistula noted previously, two rare lesions, true ophthalmic artery aneurysm and arteriovenous malformation of the orbit, must be considered. The more lateral position of these lesions on the anterior cerebral blood flow study may help in localizing these lesions. A lateral radionuclide cerebral blood flow study, however, would be a more definitive procedure, suggesting the true orbital location of these entities.

Arterial venous malformations and some vascular tumors are dramatically demonstrated on radionuclide angiograms. Even though these lesions may be regressive during the study, the great majority would be expected to persist well into the venous and even static phases of the examination. An arterial venous fistula, on the other hand, appears early and regresses rapidly with no evidence of the lesion on late venous phases.

In some cases of carotid cavernous fistula an accurate assessment of the venous outflow is possible with radionuclide angiography but the practical significance of this observation is unknown at present.

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