BILATERAL CAROTID-CAVERNOUS FISTULA

COLONEL H. R. OSHEROFF, M.C., U.S.A.

U.S. Army Hospital, Camp Gordon, Georgia

(Received for publication December 15, 1953)

The carotid-cavernous fistula presents an intriguing clinical and pathological picture. The specific surgical treatment has been recognized since 1809, when Travers\(^1\) cured a patient with pulsating exophthalmos by ipsilateral common carotid ligation. A cirsoid aneurysm of the orbit was thought to have been the etiological factor. In 1823 Guthrie\(^1\) recorded the autopsy of a patient with pulsating exophthalmos, who was found to have an aneurysm of the ophthalmic artery. Aneurysmal rupture within the cavernous sinus was described by Baron\(^1\) in 1835. It was not until 1856, when Nélaton\(^3\) reported 2 cases of traumatic origin, that the more common etiology was disclosed. Fracture of the base of the skull with traumatic rupture of the carotid artery into the cavernous sinus produces increasing proptosis and eventual ophthalmoplegia. The primitive trigeminal artery is the earliest communication between the carotid and basilar system.\(^12\) An arteriovenous fistula, or an aneurysm, may originate from this site of origin as the vessel becomes a rudimentary structure.

The popular form of therapy has been extracranial, carotid ligation on the affected side. Successful treatment by repeated, prolonged manual compression of the carotid artery has been reported in a few cases.\(^2\) A surprising number of bilateral carotid ligations have been performed in cases in which unilateral occlusion had failed to reduce the pulsating exophthalmos.\(^5\) Hamby and Gardner\(^6\) reported the first successful intracranial ligation of the internal carotid artery in the treatment of carotid-cavernous fistula. This patient had an ipsilateral common carotid ligation and a subsequent contralateral, common carotid occlusion before a cure resulted.

In this angiographic era, thorough planning of the surgical attack is made possible by first visualizing the communicating channels. Jefferson\(^7\) has shown bilateral filling of the cavernous sinus with a single arteriovenous fistula. Ramos and Mount\(^11\) have demonstrated that the proptosis may be on the side opposite the fistula. A case of rupture of bilateral saccular intracavernous aneurysms was reported by Poppen.\(^10\) The following case illustrates bilateral carotid-cavernous fistulas of traumatic origin. Angiographic demonstration of this condition has not been recorded previously, so far as we are aware. The sequence of treatment was repeatedly dictated by angiography.

CASE REPORT

An 18-year-old Private sustained a head injury in a motorcycle accident on April 5, 1953. He was admitted to U.S. Army Hospital, Fort Bragg, North Carolina in a comatose state with bleeding from the nose and right ear. Shock was treated with whole blood, after which he became restless and combative. A right 6th nerve paralysis was evident and Babinski’s sign was positive bilaterally. He remained agitated and disoriented, and on April 13, a prop-
tosis was noted on the right with early chemosis and ophthalmoplegia. At this time a left 6th nerve palsy became apparent. A bruit was auscultated over both eyes, but was more pronounced on the right. Skull films did not demonstrate a fracture.

On April 21, 1953, the patient was transferred to the Neurosurgical Section of U. S. Army Hospital, Camp Gordon, Georgia. Marked right proptosis and chemosis were apparent. Bilateral bruit, early protrusion of the left eye, positive left Babinski, restlessness, confusion and recent loss of weight were noted. A definite thrill was palpated over the right carotid. Compression of the right carotid greatly diminished the bruit. A diagnosis of right carotid-cavernous sinus fistula was made.

The stages of angiography and surgical treatment were as follows:

1. April 22, 1953. Bilateral percutaneous angiograms revealed a right carotid cavernous fistula and normal circulation on the left (Fig. 1).

![Fig. 1. (Left) Right carotid angiogram showing diodrast in the cavernous sinus and dilated ophthalmic veins. (Right) Normal left carotid angiogram.](image)

2. May 4, 1953. Ligation of the right internal carotid artery was done, with removal of clamp which had been applied 6 days previously and gradually closed. Bruit was markedly diminished on the right side.

3. May 9, 1953. Left percutaneous angiography was repeated because of an increase in the bruit over the left eye and the appearance of an easily palpable thrill over the left common carotid artery. Another carotid-cavernous sinus fistula was demonstrated (Fig. 2).

4. May 12, 1953. Right frontal craniotomy was done, with supraclinoid clipping of right internal carotid artery. The right bruit became almost inaudible and the left bruit was intensified.

5. June 22, 1953. The left internal carotid artery was ligated, with removal of clamp which had been inserted 16 days previously and gradually closed. The left bruit diminished to about 80 per cent of its former intensity. A bruit no longer could be heard on the right.

6. June 22, 1953. Open left external carotid angiography was performed because of persistent bruit on left (Fig. 3).

7. June 22, 1953. The left common carotid artery was ligated to block flow to external carotid, as the left internal carotid had been occluded previously.

Subsequent Course. Neurological examination at this time revealed that the patient was oriented, rational and alert. He was ambulatory and showed no evidence of hemiparesis, motor incoordination or aphasia. Anesthesia was present over the distribution of the left ophthalmic and maxillary nerves. The left corneal reflex was absent, whereas the right was brisk. The left bruit was still heard but was about 10 per cent of former maximum intensity. A soft murmur continued over the left orbit, but this was inaudible to the patient.
Thirty-two days after occlusion of the left common carotid the bruit disappeared. The proptosis receded completely on each side and the originally described bilateral palsy of the 6th nerve became apparent. Tarsorrhaphy was performed on the right to reduce the chemosis and later on the left because of a keratitis. Lateral closure of the eyelids was maintained temporarily to prevent further ophthalmologic difficulties. The bilateral 6th nerve palsy and above-described anesthesia persist but the patient is otherwise in good health.

Fig. 3. (Left) Lateral radiograph of skull following angiography, for comparison with left external carotid angiogram. (Right) Left external carotid angiogram. This shows retrograde filling of the fistula through the ophthalmic artery.
COMMENT

Why the left arteriovenous fistula did not appear radiographically until the right internal carotid artery had been occluded is a matter of speculation. It is felt that the increase in intravascular pressure on the contralateral side, subsequent to initial carotid occlusion, might have been sufficient to burst open a healing intracavernous-carotid tear. Possibly, a vestigial defect in the carotid may have been present to account for the development of the second carotid-cavernous fistula. The reduction in blood flow in the cavernous sinus by right internal carotid trapping and left internal and common carotid ligations eventually produced a thrombosis in the involved channels to close them.

The presence of bilateral carotid-cavernous fistulas may explain the persistence of proptosis in some of the previously recorded cases. Although no general conclusions can be made from a single case report, this may clarify the effectiveness of bilateral carotid ligations in some instances. The demonstration of retrograde filling of the cavernous sinus through the ophthalmic artery, when both internal carotid arteries had been occluded, modified our surgical attack. Mount has also shown evidence of this back flow from the external carotid into the intracranial portion of the internal carotid. Meningeal or pituitary arteries of the external carotid system may also be capable of enhancing this collateral circulation.

SUMMARY

The surgical treatment of a patient with bilateral carotid-cavernous fistulas was dictated by repeated angio graphic studies. The demonstration of a second arteriovenous communication on the contralateral side made the changing bruit, shifting carotid thrill, and persistent proptosis understandable. The visualization of retrograde filling of the cavernous sinus through the external carotid system verifies the assumption that potent collateral circulation develops in carotid-cavernous fistulas.

REFERENCES