Giant traumatic false aneurysm of the internal carotid artery associated with a carotid-cavernous fistula

Case report

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A case of giant traumatic false aneurysm of the intracranial internal carotid artery (ICA) with a concomitant carotid-cavernous fistula is reported. The fistula and the aneurysm persisted after ipsilateral cervical ICA ligation was performed elsewhere. Successful obliteration of the aneurysm and the fistula, with preservation of cross filling of the ipsilateral middle cerebral artery system, was accomplished by ligation of the intracranial ICA proximal to the origin of the posterior communicating artery with a 7-0 prolene suture, followed by transaneurysmal packing of the fistula.

KEY WORDS: carotid-cavernous fistula • arteriovenous fistula • cavernous sinus • traumatic cerebral aneurysm • internal carotid artery • surgical treatment

The association of carotid-cavernous fistula with a concomitant false aneurysm of the intracranial internal carotid artery (ICA) is uncommon, and only a few cases have been reported in the literature. In most of these cases, the abnormalities were secondary to head injuries, although occasionally they resulted from trauma during transsphenoidal surgery. The few reported cases were successfully treated by a trapping procedure alone, or trapping combined with controlled muscle embolization, or more recently by the insertion of a thrombogenic agent into the aneurysm. Although obliteration of the fistula by balloon-catheter techniques has been advocated, such attempts did not meet with success when there was an associated false aneurysm of the ICA. Parkinson described a direct approach for repair of carotid-cavernous fistulas, and, more recently, Mullan has developed sophisticated techniques for closure of fistulas by packing and occluding the venous drainage; however, these methods have not been employed in cases with an associated ICA aneurysm.

We are reporting a case of a giant traumatic aneurysm of the intracranial ICA with an associated fistula. This patient was successfully treated after a prior cervical ICA ligation, by ligation of the distal intracranial ICA, which had continued to feed the aneurysm, and by transaneurysmal packing of the fistula and the cavernous sinus. This repair was accomplished with preservation of the circulation of the ipsilateral middle cerebral artery (MCA) system from the contralateral ICA.

Case Report

On January 1, 1980, a 14-year-old Yugoslavian athlete, while sliding on a sled, hit his head on a stone barrier and sustained severe cranial and facial trauma, which resulted in fractures of the right orbital rim and the ethmoid sinuses. He also sustained a cerebral contusion and remained unconscious for 5 days following the injury. When he awoke, he was noted to have a blind right eye. He subsequently developed progressive proptosis of the right eye and noted a "swishing" noise in his head. Computerized tomography revealed a mass lesion in the region of the right cavernous sinus (Fig. 1). Angiography in February, 1980, identified a carotid-cavernous fistula with a large false aneurysm of the intracranial ICA protruding posteriorly into the interpeduncular space. On
FIG. 1. Computerized tomography scan revealing a giant aneurysm of the left internal carotid artery.

March 25, 1980, the cervical ICA and the common carotid artery were ligated. This procedure resulted in some restoration of the vision of the right eye and a slight decrease in the proptosis. However, postoperative angiography revealed persistence of the fistula and the false aneurysm. The patient was referred to the Mayo Clinic for further surgery.

Examination. There was mild proptosis of the right eye, and vision in that eye was limited to perception of finger movements. A high-pitched bruit was audible over the right orbit. Angiography on June 2, 1980, showed a large lobulated aneurysm arising from the right ICA in the region of the cavernous sinus, extending superiorly into the interpeduncular cistern with an associated carotid-cavernous fistula (Fig. 2). The fistula and the aneurysm filled in retrograde manner from the distal right ICA by cross flow through the A1 segment of the right anterior cerebral artery and also from the vertebrobasilar system through the posterior communicating artery. There was no antegrade flow into the right MCA, which appeared to be occluded at its origin. Presumably this occlusion occurred at the time of the injury from thrombotic reaction to the hemorrhage and tear in the ICA, and it may well have been the result of an embolus originating at the site of the tear in the ICA. This complex filled through leptomeningeal collaterals over the convexity of the hemisphere. The proximal intracranial ICA on the right was not separately visualized, and it was assumed that it had been severed at the time of the injury.

Prior to surgical exploration, the patient's tolerance to induced hypotension with intravenous nitroprusside was determined by electroencephalographic (EEG) monitoring while he was awake and asleep. This study was thought to be of critical importance because of the marginal filling of the right MCA from collateral vessels. At a systolic pressure of 70 mm Hg, the patient became light-headed and somewhat weak. There was an associated slowing of the EEG, more

FIG. 2. Left carotid angiograms, lateral (left) and anteroposterior (right) views; subtraction films. A large lobulated aneurysm is seen arising from the intracranial right internal carotid artery (ICA) and extending superiorly into the interpeduncular cistern. The aneurysm is fed on the lateral side by the right ICA. No evidence of antegrade filling of the right middle cerebral artery is observed. There is a carotid-cavernous fistula with rapid opacification of the cavernous sinus and the superior ophthalmic vein.
False carotid aneurysm and cavernous fistula focal on the right than the left. It was determined that we would not drop the pressure below 70 mm Hg.

Operation. The false aneurysm was exposed through a pterional approach using spinal drainage (Fig. 3A-C). It was apparent that the wall of the aneurysm was formed essentially by thickened arachnoid. The Sylvian fissure was opened, the right MCA identified, and this vessel traced to its origin at the right ICA. It appeared that the MCA was patent up to the point of its origin just distal to the bifurcation of the ICA. A rete of small collateral vessels from the artery of Hübner, the anterior choroidal artery, and the posterior communicating artery had developed to anastomose with the striate arteries and anterior branches of the anterior temporal artery. It was apparent that these small collaterals had to be preserved. The cross filling from the left ICA was diverted entirely into the aneurysmal sac and the fistula, with no filling of the

FIG. 3. Diagrammatic representation of the operative procedure. A: The location of the aneurysm, the position of the patient for surgery, and the outline of craniotomy are shown. B: Closer view of the aneurysm and fistula. The aneurysm seemingly developed from a life-saving containment of the hemorrhage by the arachnoidal compartments surrounding the internal carotid artery (ICA). Note the occluded proximal middle cerebral artery (MCA). The fistulous opening into the cavernous sinus was located inferior and lateral to the ICA and medial to the third nerve. ACA = anterior cerebral artery. C: The wall of the aneurysm (thickened arachnoid) was dissected away from the ICA with the tip of a No. 11 blade knife and then retracted and elevated with a small spatula. Insufficient space to occlude the ICA with an aneurysm clip and yet preserve the posterior communicating artery made it necessary to ligate the ICA with a No. 7-0 prolene suture. D: Following ligation of the distal ICA, the aneurysm was opened and the sac emptied by a strong suction unit. Considerable bleeding occurred from this maneuver. E: The proximal ICA was clipped and the cavernous sinus packed with Surgicel.
MCA. With the patient under controlled hypotension to 70 mm Hg, the thin-walled aneurysm was carefully dissected away from adjacent structures using the tip of a No. 11 blade knife and a cotton pledget. The right ICA was traced proximally, and the right anterior choroidal and posterior communicating arteries were identified. At this point, there was not sufficient room to apply an aneurysm clip without compromising the lumen of these arteries. Hence, the segment of the right ICA feeding the aneurysm and the fistula was doubly ligated with 7-0 monofilament nylon sutures. After this was accomplished, it was noted that the right anterior choroidal artery and the right MCA appeared much larger and under a better perfusion pressure than before ligation (Fig. 3D). It was apparent that there had been a significant "steal" into the aneurysmal sac and from there into the fistula. The dome of the false aneurysmal sac was then opened, and this resulted in profuse venous bleeding arising from the cavernous sinus and the ophthalmic veins. These venous communications were identified and the bleeding controlled by packing with Surgicel (Fig. 3E) and Avitene.*

Postoperative Course. The patient had an uneventful recovery except for a mild abducens palsy, which developed 24 hours after surgery. Vision in the right eye improved markedly, the bruit subsided, and the abducens weakness was gradually improving at the time of discharge. Postoperative angiography revealed absence of filling of the giant aneurysm and the fistula (Fig. 4). The right MCA was visualized and was filling from the right A1 segment through the anastomotic vascular pathways described above. A communication from his physician in Yugoslavia in December, 1980, reported that the patient's vision had markedly improved, the abducens palsy had cleared, and he had resumed normal activities.

Discussion

Traumatic aneurysms of the intracranial ICA with an associated carotid-cavernous fistula are rare. Traumatic aneurysms can be either true or false aneurysms. True aneurysms develop secondary to incomplete rupture of the arterial wall, and false aneurysms are the result of complete interruption of the arterial wall with formation of a perivascular hematoma which subsequently develops a fibrous wall by organization. Most of the reported cases have been a result of head injuries, and these were usually associated with basilar skull fractures. However, Lister and Sypert reported a case of a traumatic aneurysm with carotid-cavernous fistula following a sphenoidotomy for chronic sinusitis, and Paullus, et al., reported another case found after transsphenoidal surgery for a pituitary adenoma. Traumatic aneurysms, when they occur in association with a carotid-cavernous fistula, are usually located distal to the fistula and only occasionally proximal to it. These aneurysms are usually single, but rarely they can be multiple.

On angiography, the early opacification of the cavernous sinus may mask the presence of an associated aneurysm, and its presence may be overlooked unless good-quality subtraction films are routinely obtained. Waga, et al., advocated intraoperative an-
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giography at the time of embolization of the fistula, not only to confirm the patency of the carotid circulation, but also to diagnose the presence of these unsuspected aneurysms.

The common clinical manifestations of this combined presentation are: progressive visual loss, chemosis, pulsating proptosis, paralysis of extraocular muscles, and an orbital bruit. Occasionally, rupture of a false aneurysm into the sphenoid sinus may result in catastrophic epistaxis.9,10,19 False aneurysms tend to progressively increase in size,1,11 and the onset of the symptoms may sometimes be delayed for several weeks or even months following the trauma.1,21

An array of ingenious techniques for the management of an isolated carotid-cavernous fistula with preservation of the carotid artery have been described. These include: direct repair of the fistula through the cavernous sinus,12 electrothrombosis,7 packing of the cavernous sinus,11 occlusion with detachable balloon catheters,2,17 and more recently intracavernous injection of cyanoacrylate.8 Most of the reported cases of carotid-cavernous fistula with an associated aneurysm were, however, treated by either trapping alone9 or by trapping with controlled muscle embolization.1,11,22

Some authors have advocated an extracranial-intracranial anastomosis to prevent possible ischemic complications of the ICA ligation in the management of isolated ICA aneurysms3,4 or carotid-cavernous fistulas,13 but this approach has not been used when both these lesions existed concomitantly.2,17 Mullan11 reported a case where successful thrombosis of the aneurysm and the fistula with preservation of the carotid artery was accomplished by insertion of 40 feet of wire into the false aneurysm. Although balloon catheter techniques2,17 have the added advantage of avoiding a craniotomy, these procedures have not been effective in the presence of an associated aneurysm.9 Furthermore, Debrun, et al.,2 indicated that use of balloon catheters might be contraindicated in this situation because of the significant risk of rupture of the aneurysm.

We believe that a carotid-cavernous fistula with a concomitant false aneurysm in most instances requires direct surgical exploration. The neck of the aneurysm or the feeding vessels should be clipped or ligated and the cavernous sinus packed. The packing can be done through the aneurysm sac if the fistula communicates with the sac, as in the present case, or by one of the various techniques described by Mullan.11 It is imperative that as many collateral channels of flow as possible be preserved.

A final comment seems appropriate concerning the amazing capacity of the arachnoid to contain a hemorrhage of this severity. Yasarigit, et al.,23 pointed out that the arachnoid in the basal cisterns is actually a series of compartments, and knowledge of their location and shape facilitates the dissection of aneurysms. Presumably, daughter sacs of recently ruptured aneurysms are confined or reinforced by these arachnoidal membranes and in some instances are actually comprised of these structures. The findings of this case underscore the importance of and protective effect of the arachnoid on some intracranial aneurysms.

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