End-to-end anastomosis of the anterior cerebral artery after excision of a giant aneurysm

Case report

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A case of a giant anterior cerebral artery fusiform aneurysm is presented. The lesion was treated by primary excision of the involved segment with an end-to-end anastomosis of the proximal-distal segments of the anterior cerebral artery. It is believed that this technique has not been reported previously for lesions involving major intracranial arteries.

KEY WORDS · fusiform aneurysm excision · anterior cerebral artery · end-to-end anastomosis · temporary occlusion

Giant intracranial aneurysms measure greater than 2.5 cm in their greatest dimension. The term “fusiform” has been applied to describe those giant aneurysms in which the outpouching is spindle-shaped and involves the total circumference of the arterial wall. The underlying pathology of the fusiform type is probably quite distinct from that of saccular aneurysms, in which only a small segment of the vessel circumference is involved. Whereas focal defects in the medial layer and internal elastic membrane are apparently progenitors of aneurysms of the saccular type, inflammatory lesions of the arterial wall and degenerative connective tissue disorders, such as Marfan’s syndrome and pseudoxanthoma elasticum, have been described in patients harboring aneurysms of the giant fusiform type. Cranial arteritis and syphilis may also play a role in the pathogenesis of the latter type.

Fusiform aneurysms have been recognized on the vertebral and basilar arteries, the internal carotid artery, the middle cerebral artery (MCA), and at the origin of the anterior cerebral artery (ACA). They have not been described previously in the distal ACA segment. These large channels provide a nidus for thrombus formation and secondary embolization. By virtue of their mass, they commonly affect adjacent cranial nerves, but rarely cause subarachnoid hemorrhage (SAH). In this report, however, we describe a giant fusiform aneurysm of the ACA which was responsible for SAH. The lesion could not be effectively managed using conventional methods, and thus it was excised and an end-to-end anastomosis carried out between the proximal and distal segments of the ACA.

Case Report

This 67-year-old man was admitted to the Jackson Veterans Administration Medical Center with a 1-week history of diffuse headache, myalgia, nausea, and vomiting. On the day of admission, he was aroused by severe headaches, radiating to the occipital area. He was confused and incontinent.

Examination. The patient was slightly lethargic, but his verbal responses were appropriate and he was oriented. He had a mildly stiff neck but no localizing neurological deficits. His blood pressure was 140/90 mm Hg and heart rate 86/min and regular. A lumbar puncture was performed and xanthochromic fluid was obtained. Computerized tomography (CT) demonstrated a calcified right frontal parasagittal lesion with minimal enhancement (Fig. 1). Angiography demonstrated a fusiform aneurysm of the right ACA, with some distal narrowing of the pericallosal artery (Fig. 2).
Operation. On March 19, 1981, under general halothane anesthesia, a bifrontal craniotomy was performed. The dura was opened bilaterally, and the sagittal sinus was ligated anteriorly, divided, and the falk was incised. The dural flap and sinus were then retracted posteriorly (Fig. 3B), exposing the interhemispheric fissure. The aneurysm, which was asymmetrically fusiform over a 6- to 7-mm segment, penetrated the right frontal lobe through the cingulate gyrus. It was firm, seemed to be filled with thrombus, and would not accept a clip without compromise of the anterior cerebral vessel. The surrounding brain was gliotic and xanthochromic.

At this point, a double-blade microvascular clip was placed on the ACA proximal and distal to the aneurysm (Fig. 3C). The left ACA was visualized and was retracted laterally. A 7-mm segment of the artery, encompassing the aneurysm, was then excised. The ACA was only slightly dilated at the point of excision. The proximal and distal segments were then freed of their arachnoid attachments, although no perforating vessels were sacrificed. Approximately 1 ml of undiluted heparin was injected into the proximal and distal segments via a No. 27 needle. The microvascular clips were then approximated until the proximal and distal segments touched. Anastomosis was carried out using 9-0 interrupted black nylon, first on one side, then the other (Fig. 3D and E). Nine sutures were required to complete the anastomosis. The anastomosis was reinforced with gelatin sponge and the microvascular clips removed. The total occlusion time was 45 minutes.

Postoperative Course. The patient maintained the strength in both his upper and lower extremities, and there was no increased deficit. Follow-up CT showed no evidence of infarction (Fig. 4 left), and a postoperative angiogram 1 week after the anastomosis showed patency of the right ACA (Fig. 4 right), with filling of the distal pericallosal branch. During the next 3 months, the patient developed no additional neurological deficits, and he has returned to his former activities.

Discussion

There is no consensus in the literature regarding management of the difficult giant aneurysm and fusiform aneurysm of the cerebral arteries. Perhaps, in

FIG. 1. Computerized tomography scan on admission showing an enhancing lesion in the right parasagittal frontal lobe.

FIG. 2. Angiogram, lateral (left) and oblique (right) views, demonstrating a fusiform enlargement of the anterior cerebral artery proximal to the callosomarginal branch. Irregularity of the distal vessel is also seen.
some cases, when the potential for intracranial ischemia or hemorrhage is not a factor, a nonoperative approach is the proper one since operative methods have not proven uniformly successful.\textsuperscript{3,11,17} In managing fusiform aneurysms of the vertebral artery, Sugita, et al.,\textsuperscript{10} have proposed the formation of a new parent artery by successive applications of a fenestrated clip developed in their center. With this technique, the aneurysm is pinched off, forming a progressively smaller tube as the primary arterial channel. The method does not take into account, however, the intra-aneurysmal thrombus which is a recognized feature of this lesion.\textsuperscript{5,10,11,15} Spetzler, et al.,\textsuperscript{18} suggested trapping procedures to exclude giant aneurysms of the internal carotid artery from the circulation. An extracranial-intracranial bypass can be used to provide distal perfusion of the MCA and to prevent distal ischemic complications. In a series of 13 patients they treated in this manner, there were no late ischemic or bleeding events. They suggested trapping of the aneurysm immediately after the bypass procedure so as to produce the best pressure gradient for the maintenance of the bypass.\textsuperscript{18} Samson, et al.,\textsuperscript{14} have warned,
however, that bypass surgery may not always be effective in preventing infarction distal to a trapping procedure involving the major trunk of the MCA. In one of their cases, there was progressive infarction in the distribution of the MCA after the procedure. Two other patients, however, tolerated permanent occlusion of the proximal MCA, whereas one other patient accepted 80 minutes of occlusion. Chater noted that the usual superficial temporal artery-MCA anastomosis only delivers 20 to 60 mm of blood per minute, while normal flow rates of 75 to 120 ml/min are recorded from the MCA. This would lead to the expectation of a high risk of infarction associated with sacrifice of the main trunk of the MCA.

There is little information available concerning sacrifice of the ACA, especially the distal portions. The tolerance time for temporary occlusion of the ACA is likewise unknown. Nornes and Wikeby found flow rates in the distal ACA (120 ml/min) comparable to those of the MCA, suggesting that tolerance for temporary occlusion of the two vessels might be similar. Harvey and Rasmussen found that after only 15 minutes of temporary occlusion of the MCA of the primate, infarction, although quite minimal, could be detected regularly in the distribution of the vessel. However, the occlusion was tolerated for periods up to 30 minutes if the animal was massively heparinized. An equivalent dosage for man would range between 300 and 400 mg administered intravenously.

It is quite likely that the tolerance for occlusion depends on how far distally the clip is applied. In the case of the MCA, when the clip is applied proximal to the distal territory of the ACA after 45 minutes of occlusion, either a collateral supply protected the distal anterior cerebral territory or perfusion was established before cellular death occurred.

References

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