Spontaneous thrombosis of a giant intracranial aneurysm and ipsilateral internal carotid artery

Case report


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Computerized tomography revealed a thrombosed giant intracavernous carotid artery aneurysm in a man who presented with ophthalmoplegia and headache. Angiography confirmed complete aneurysmal thrombosis and also revealed complete occlusion of the ipsilateral internal carotid artery. Aneurysmectomy and thrombectomy produced substantial reduction in mass effect, with symptomatic improvement. The spontaneous thrombosis of giant intracranial aneurysms is discussed.

KEY WORDS: giant aneurysm, spontaneous thrombosis, aneurysmectomy, thrombectomy

Complete spontaneous thrombosis of unruptured giant intracranial aneurysms has been recognized as an infrequent neuropathological curiosity since Lyall's initial report in 1936. Antegrade propagation of thrombosis that also occludes the aneurysm's parent artery is, however, a rare occurrence. We are reporting a case of spontaneous cure of a giant intracavernous carotid artery aneurysm by aneurysmal and ipsilateral internal carotid artery thrombosis.

Case Report

This 27-year-old Indonesian man was admitted on January 30, 1981, for investigation of headaches, diplopia, and a red left eye. Two years previously, he had noted the onset of painless diplopia and drooping of the left eyelid. Three weeks prior to admission he had experienced the sudden onset of constant left frontoparietal headaches and a red left eye; he had no other neurological symptomatology. There was no history of seizures, mental dysfunction, or hypertension.

Examination. On admission, the patient's blood pressure was 120/70 mm Hg, and his vital signs were normal. Funduscopy was unremarkable; visual acuity was 6/5 on the right and 6/9 on the left. The left eye revealed ptosis, proptosis (4 mm), absent pupillary response to direct and consensual light, suffusion of the conjunctiva, and restriction of ocular movements in all directions. Hypalgesia and hypesthesia were also present in the distribution of the left maxillary and ophthalmic nerves. The remainder of the neurological examination was normal, and there were no cranial, carotid, or orbital bruits.

Axial computerized tomography (CT) of the head revealed a large, partly calcified, globoid lesion in the left middle fossa with some contrast enhancement of its margin (Fig. 1). Coronal CT also demonstrated the globoid mass, which seemed to arise from the region of the left cavernous sinus. The lesion was 6 x 6 x 5 cm in size. Carotid angiography revealed occlusion of the left internal carotid system almost to its origin in the neck (Fig. 2 left). The left anterior and middle cerebral arteries filled from the right carotid system, and were displaced by the avascular mass (Fig. 2 right). Various serological, biochemical, hematological, and pituitary function tests were normal. A preoperative diagnosis of thrombosed giant intracavernous carotid aneurysm was made.

Operation. On February 12, 1981, exploration of the mass was undertaken through a left frontotemporal craniotomy. The dura was opened, and the gyri of the anterior temporal lobe were noted to be flattened and the Sylvian fissure elevated. The fissure...
FIG. 1. Preoperative axial computerized tomography scans. **Left:** A spherical lesion with calcified capsule occupies a substantial part of the middle fossa, and distorts the body of the sphenoid medially. **Right:** There is contrast material around but not within the lesion capsule.

split readily to expose a large nonpulsatile mass covered by a capsule that was continuous with the dura of the middle fossa. The carotid artery was noted to be reduced to a thin ribbon, and the mass was needled with progressively larger needles until some old blood was obtained. A 1.5-cm aneurysmotomy was then performed followed by thrombectomy. The laminated and more recent thrombus was removed by irrigation, suction, and curettage, causing considerable reduction in the aneurysm mass. No attempt was made at complete excision. Histological examination of the wall of the mass confirmed the diagnosis of aneurysm.

**Postoperative Course.** The postoperative course was uneventful, and the patient was discharged on the 8th day after surgery. At follow-up examination 3 months later, he was free of headaches and the proptosis appeared reduced; however, the diplopia and ophthalmoplegia were unchanged.

**Discussion**

Since Morley and Barr described aneurysms greater than 2.5 cm as "giant," various publications have described the diagnosis, management, and natural course of these lesions. Spontaneous thrombosis, either partial or complete, within some of these lesions is a well recognized phenomenon. The percentage of giant intracranial aneurysms that spontaneously totally thrombose is, however, unknown since most publications have reported only isolated cases. In Drake's surgical series of 174 patients with giant
Spontaneous thrombosis of giant intracranial aneurysm

intracranial aneurysms, none of the aneurysms was totally thrombosed preoperatively. Neuroradiological series that have defined the CT characteristics of giant intracranial aneurysms have reported the incidence of total thrombosis as between 13% and 20%.5,9,11

The site of the lesion is of no predictive value as to likelihood of spontaneous thrombosis, since this phenomenon has been described on all major branches of the anterior circulation. Factors that precipitate thrombosis are also unknown, although occurrence is almost certainly related to the ratio of aneurysm volume to orifice size, the age of the aneurysm, and the hemodynamics within the parent artery. Direct distortion of the parent artery by the aneurysm would also seem to be essential in the etiology of proximal propagation of thrombus. The only recorded case similar to ours occurred in a 19-year-old woman, who, like our patient, was unlikely to have had significant atherosclerotic narrowing.

The operative finding in our case suggested that the headache may have been a result of a significant thrombotic episode, either within the aneurysm or in the compressed cavernous sinus. Review of the previously recorded cases suggests that there is no clinical correlate to the thrombotic episodes in globoid aneurysms; indeed, some cases are almost asymptomatic.5,7,12 Epilepsy, progressive focal neurological dysfunction, and headaches due to raised intracranial pressure are generally attributable to the mass effect and location of the aneurysm.

Management of the spontaneously thrombosed globoid giant intracranial aneurysm has varied, and some asymptomatic cases have been treated conservatively.3,12 Surgery should be considered, however, in the clinically stable but symptomatic patient, since any significant spontaneous reduction in mass effect would be slow, because of the avascularity of the laminated, sometimes centrally necrotic thrombus.14 Aneurysmptomy and thrombectomy, or aneurysmectomy where technically possible, have resulted in a good symptomatic and clinical response.1,6 Particular caution is indicated, however, in thrombosed fusiform giant aneurysms, and also in aneurysms that incorporate distorted or serpiginous vascular channels with the wall, since these may be a source of profuse hemorrhage or later cerebral infarction.6,10,13

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References


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