Rupture of a giant basilar aneurysm after saphenous vein interposition graft to the posterior cerebral artery

Case report

ROBERTO C. HEROS, M.D., AND ALI M. AMERI, M.D.

Neurosurgical Service, Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts

A patient with a giant aneurysm of the top of the basilar artery presented with severe progressive symptoms of brain-stem compression. There was inadequate collateral circulation to the upper basilar system. She underwent exploration of the aneurysm and, after it was found to be impossible to clip, a tourniquet was placed on the basilar artery for future occlusion with the patient awake. A saphenous vein graft was interposed between the left external carotid and the left posterior cerebral arteries. The previously unruptured aneurysm bled fatally 36 hours after surgery, just before intended occlusion of the basilar artery.

KEY WORDS • giant aneurysm • basilar artery aneurysm • anastomosis • extracranial-intracranial bypass • saphenous vein interposition graft • subarachnoid hemorrhage

DRAKE was the first to indicate that basilar ligation could be an effective, albeit dangerous, method of treatment for inoperable giant basilar aneurysms.4 To increase the safety of arterial ligation, he later suggested using a tourniquet which could be placed at surgery and tightened afterward with the patient awake and under radiographic control. He also referred to the “Allcock test” which consists of compressing each carotid artery in turn during vertebral injection to ascertain the size of the posterior communicating arteries and thus the potential for collateral circulation to the top of the basilar artery.3 Sundt, et al.,21 have suggested using an interposition saphenous vein graft to the posterior cerebral artery to enhance the safety of basilar trunk occlusion in patients with deficient collateral circulation and inoperable basilar aneurysms.

We are reporting a patient with progressive signs of brain-stem compression secondary to a giant aneurysm of the basilar bifurcation. This patient was treated by application of a tourniquet to the distal basilar artery and by an interposition saphenous vein graft from the external carotid artery to the left posterior cerebral artery. She died from rupture of the aneurysm 36 hours after surgery, just before the planned occlusion of the basilar artery. We have not found a similar case reported.

Case Report

This 57-year-old woman was admitted in February, 1983, for evaluation of progressive gait difficulty, incontinence, headache, nausea and vomiting, and dementia of several weeks’ duration. The medical history was relevant in that she had coronary artery disease with a previous myocardial infarction and severe chronic obstructive pulmonary disease. A computerized tomography (CT) of the head showed significant obstructive hydrocephalus and a large enhancing lesion in the area of the tentorial incisura. An arteriogram showed a giant basilar artery aneurysm which was considered inoperable. A ventriculoperitoneal shunt was placed and the patient improved significantly and was discharged home.

Admission. She was readmitted in October, 1983, with difficulty in swallowing, progressive right-sided numbness, left-sided spasticity, diplopia on looking to the right side, and severe ataxia with inability to sit up in bed unassisted. She had become unable to live independently and had been in a nursing home for 2 months. For the last 3 weeks she had been practically...
bedridden and had lost about 30 lb because of her swallowing difficulty. Repeat CT scan showed a probable increase in the size of the basilar aneurysm. There was no indication of aneurysmal thrombosis. Repeat arteriography confirmed the presence of the large unthrombosed aneurysm (Fig. 1). With temporary balloon occlusion of each carotid artery during vertebral injection there was no visualization of either posterior communicating artery (Fig. 2). There was only poor and delayed visualization of the distal posterior cerebral arteries.

We considered that an attempt to treat this aneurysm was justified in view of the patient’s relentless deterioration. The “Allcock test” failed to show even a small posterior communicating artery, so we assumed that the patient would not tolerate primary basilar occlusion. The aneurysm projected to the right (Fig. 1), and therefore an exploration of the aneurysmal neck would have to be carried out from the left side.

Operation. With these considerations in mind, we performed a left subtemporal craniotomy and initially proceeded to explore the aneurysmal neck. After confirming that indeed the superior cerebellar and posterior cerebral arteries arose from the distended aneurysmal neck and therefore clipping was not possible, we proceeded to place a tourniquet around the distal basilar artery just as it began to distend into the aneurysm. The aneurysm appeared to be relatively thick-walled and there was no evidence of its ever having bled. A saphenous vein graft was then interposed without difficulty between the external carotid artery and the left posterior cerebral artery just as it started to turn around the cerebral peduncle in a short segment that was free of perforating branches.

Postoperative Course. The patient was slow to awaken and did not obey commands, but she moved all extremities spontaneously. A CT scan showed no temporal hemorrhage or mass effect. The vein graft had an excellent pulse. We decided to wait an extra day to allow for additional neurological improvement before tightening the tourniquet under radiographic control.

![FIG. 1. Right vertebral angiogram, anteroposterior view, during temporary balloon occlusion of the left carotid artery. The aneurysm is projecting to the right, and there is lack of early filling of the posterior cerebral arteries.](image1)

![FIG. 2. Left: Lateral right vertebral injection with temporary balloon occlusion of the left carotid artery. Note poor and delayed filling of the posterior cerebral arteries and absence of demonstration of a left posterior communicating artery. Right: Lateral right vertebral injection with temporary balloon occlusion of right carotid artery. Note poor and delayed filling of the posterior cerebral arteries and absence of demonstration of a right posterior communicating artery.](image2)
Treatment of giant basilar aneurysm

At about 2:00 a.m. on the day of the planned basilar occlusion procedure, she suddenly became decerebrate, her vital signs began to fail, and blood started to pour out through the stab wound where the tourniquet was brought out. She died in a few hours and at autopsy the aneurysm was found to have bled massively. The vein graft suture line was intact and there was no indication of antemortem thrombosis of the graft.

Discussion

Retrospectively, it appears that in this case the basilar artery should have been ligated forthwith at surgery after satisfying ourselves that the vein graft was patent and flow through it was satisfactory. There is a good chance that she would have tolerated this abrupt occlusion. At the time, however, we thought that there was a significant risk of graft occlusion during the few hours immediately after surgery and, in addition, we believed that occluding the basilar artery with the patient awake would add a margin of safety. Initially, the patient failed to awaken satisfactorily from surgery which, in view of the negative CT scan, we attributed to manipulation of the already compromised brain stem during exposure of the aneurysm. This problem has already been commented upon by Drake and Peerless and also by Hopkins, et al. This is why we deferred basilar occlusion for an extra day, hoping that the patient's neurological status would continue to improve enough for adequate clinical testing at the time of occlusion. Her aneurysm bled fatally hours before the planned arterial occlusion.

We believe that the iatrogenically induced hemodynamic changes brought about by the vein graft were partially responsible for the rupture. The vein graft had a bounding pulse right up to the time of death. In addition, considerable distention of the posterior cerebral artery was evident at surgery after flow through the graft was established. It is not difficult to postulate that occluding the basilar artery with the patient awake would add a margin of safety. Initially, the patient failed to awaken satisfactorily from surgery which, in view of the negative CT scan, we attributed to manipulation of the already compromised brain stem during exposure of the aneurysm. This problem has already been commented upon by Drake and Peerless and also by Hopkins, et al. This is why we deferred basilar occlusion for an extra day, hoping that the patient's neurological status would continue to improve enough for adequate clinical testing at the time of occlusion. Her aneurysm bled fatally hours before the planned arterial occlusion.

We believe that the iatrogenically induced hemodynamic changes brought about by the vein graft were partially responsible for the rupture. The vein graft had a bounding pulse right up to the time of death. In addition, considerable distention of the posterior cerebral artery was evident at surgery after flow through the graft was established. It is not difficult to postulate that intra-aneurysmal pressure was significantly increased by retrograde flow from the left posterior cerebral artery into the aneurysm. A similar mechanism may have been operative in a case reported by Scott, et al., of a middle cerebral artery aneurysm that bled after a superficial temporal to middle cerebral artery bypass procedure. An additional factor that may have contributed to rupture of that previously unruptured aneurysm was the simple exploration and manipulation of the aneurysm. Drake and Peerless have already commented on the occurrence of aneurysm rupture shortly after simple exploration. It may be that simply removing some of the support to the aneurysmal wall by dissecting brain from it may predispose to rupture. In our case, dissection of the aneurysmal sac was very limited and the wall was thick and easily separable from brain tissue. However, the site of rupture at autopsy was precisely in the area of the aneurysm that had been dissected free.

Even if this catastrophic hemorrhage had not occurred, two basic assumptions in our plan of management may be criticized. First, we assumed that basilar ligation would lead to thrombosis of the aneurysm in spite of the flow provided by the saphenous vein graft. Ideally, to ensure thrombosis the aneurysm should be trapped. In this case this would have required, in addition to basilar ligation, ligation of both superior cerebellar and posterior cerebral arteries as they originated from the aneurysm and, in view of the demonstrated lack of distal collateral circulation, a bypass graft to each of these arteries. Clearly, this was not a practical consideration. We simply had to hope that, after basilar ligation, the new direction of flow toward the aneurysmal base rather than directly toward the apex would produce enough stasis of the dome to allow thrombosis of the aneurysm and preserve flow into the major efferent arteries at the base of the aneurysm. We have not found a report of a patient with a giant basilar aneurysm treated by a saphenous vein interposition graft and basilar occlusion. However, in the one patient of Hopkins, et al., who survived basilar occlusion after a superficial temporal to superior cerebellar artery bypass graft, the giant basilar aneurysm was found to be thrombosed and good flow to the upper basilar circulation through the graft was demonstrated angiographically. It could be argued that a donor vessel, such as the superficial temporal artery, that would deliver less flow than a vein graft, had a better chance of allowing thrombosis of the aneurysm while at the same time maintaining sufficient flow to the distal basilar artery. Sundt, et al., however, had a very disappointing experience in seven patients with ischemic problems in whom the superficial temporal artery was anastomosed to either the superior cerebellar or the posterior cerebral artery. They had a high incidence of early graft occlusion and considered that the flow provided by the superficial temporal artery was inadequate to support the upper basilar circulation when high flow was immediately necessary. In our patient, the potential for collateral circulation was so limited that we wanted to have a large conduit immediately available to supply the distal basilar circulation.

The second basic assumption in our plan of management was that once thrombosis had occurred in the aneurysm the signs of progressive brain-stem compression would gradually improve. Even if everything else had gone well and the aneurysm had thrombosed, the patient may not have improved and in fact may have worsened. Both in our own series of giant paraclinoid aneurysms and in the series reported by Ferguson and Drake, some patients showed deterioration of vision, presumably due to increased compression of the optic apparatus, coincidentally with aneurysmal thrombosis induced by proximal arterial ligation. In both of these series, however, the number of patients who improved as a result of aneurysmal thrombosis is considerably larger than the number of patients who deteriorated. Similarly, considerable recovery after aneurysmal thrombosis, even in cases with severe disability from
long-standing aneurysmal compression of the brain stem, has been reported in a smaller number of patients with giant basilar aneurysms.1,3,4,15

Other methods of treatment, such as direct or stereotaxic injection of the aneurysmal sac with wire to induce thrombosis13,18 or attempted excision of the aneurysm and aneurysmorrhaphy under cardiac arrest with cardiopulmonary bypass,1,17,22 may have been considered. In Drake’s experience,3 wire injection produced only incomplete thrombosis and the aneurysms subsequently bled or enlarged at the unthrombosed portion of the neck. His experience with aneurysmorrhaphy under cardiopulmonary arrest has also not been satisfactory because of the high incidence of temporal lobe hemorrhage, presumably related to the clotting abnormalities that are a necessary concomitant of this procedure.3 We have no experience with the former method, and our very limited experience with cardiopulmonary bypass in cases of giant aneurysms has been catastrophic.

Finally, it could be asked whether this patient should have been treated at all in view of the predictable difficulties with any form of treatment. The natural history of giant aneurysms in general is notoriously poor.2,3,9,12,14,18 It is now clear that they bleed probably as frequently as non-giant aneurysms and about 30% to 50% of cases present with subarachnoid hemorrhage.3,7,9,10,12,14,19,20 Cerebral ischemia resulting from embolism or major vessel occlusion as the aneurysms gradually thrombose also accounts for significant morbidity in patients with these lesions.9,20 More pertinent to our case is the fact that, once progressive signs of brain or nerve compression by the large aneurysmal mass develop, most patients deteriorate relentlessly.7,12,15 It appears that, if left untreated, about 80% of patients with massive aneurysms presenting with signs of brain compression are dead within a few years.2,3,9,12,19

The patient described here presented with progressive symptoms of brain-stem compression secondary to a giant aneurysm of the top of the basilar artery. Her relentless progression to marked disability and the dismal natural history of her disease compelled us to attempt to treat her lesion. Our surgical attempt failed perhaps because of unnecessary delay in occluding the basilar artery. Given the same situation again, we would proceed in the same manner with a saphenous vein graft to the posterior cerebral artery, but we would occlude the basilar artery acutely, with a simple clip, after being satisfied that flow through the vein graft was adequate.

References