Fatal Bleeding From Arterial Dissection After Clipping of a Ruptured Aneurysm —Case Report—

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Abstract

A 67-year-old man died of subarachnoid hemorrhage (SAH) resulting from dissection of the distal part of the anterior cerebral artery (ACA). A saccular aneurysm in the anterior communicating artery had ruptured and was successfully clipped on Day 0. The patient recovered consciousness after surgery but his condition deteriorated due to another SAH on Day 1. A second surgical procedure disclosed bleeding from a laceration in the opposite wall of the ACA distal to the clipped aneurysm. Histological examination of the autopsy specimens revealed damage to the internal elastic lamina and inflammatory infiltration of leukocytes. The fatal dissection may have resulted from atherosclerosis, hemodynamic stress caused by hypertension, or trauma due to surgical manipulation.

Key words: dissection, subarachnoid hemorrhage, surgical complication

Introduction

Dissecting aneurysm in the brain occurs more often in the posterior circulation, particularly in the vertebral artery, than in the anterior circulation. The clinical manifestations are cerebral ischemia if the dissection originates in the subintimal layer, or subarachnoid hemorrhage (SAH) if the dissection originates in the subadventitial layer. SAH resulting from intracranial dissection occurs with moderate frequency, and is most often involves the vertebrobasilar artery.12) In contrast, dissection in the anterior cerebral artery (ACA) is much less common.13) Here we report a case of progressive dissection of the ACA after successful clipping of an anterior communicating artery (AcomA) aneurysm, which was confirmed by autopsy.

Case Report

A 67-year-old man suffered onset of severe headache at home in the morning and was transported to a local hospital by ambulance. His initial Glasgow Coma Scale score was 12 (E3, V4, M5) and no focal neurological deficit was observed. He had received inadequate treatment for hypertension and hyperlipidemia for some time. Computed tomography (CT) revealed thick and diffuse SAH in the basal cistern as well as the interhemispheric and sylvian cisterns. He was immediately transferred and admitted to our clinic.

Digital subtraction angiography disclosed a small saccular aneurysm on the AcomA (Fig. 1 left) and occlusion of the left internal carotid artery (ICA). Collateral blood flow was supplied by the ipsilateral posterior communicating artery as well as by the AcomA. A fusiform dilatation at the tip of the basilar artery and wavy atherosclerotic vessels were also observed (Fig. 1 right).

His preoperative World Federation of Neurosurgical Societies grade was IV and his SAH was classified as Fisher group 3. He underwent emergency surgery on Day 0. The aneurysm was exposed via the right pterional approach, which showed that the tip of the aneurysm dome was ruptured. A temporary clip was applied to the right proximal ACA and the aneurysm was uneventfully clipped (Fig. 2).

The patient regained consciousness after surgery. Postoperative CT revealed no problems on Day 1.
Dissection After Clipping

Soon after he returned to his bed, he became unresponsive to noxious stimuli, fell into a coma, and then into respiratory arrest. The bilateral pupils were unreactive to light and fully dilated. Immediate CT disclosed a new SAH, which was not visible on the previous CT scan. We believed that the SAH originated from the aneurysm remnant or slipping of the applied clip.

A second emergency surgical procedure was performed. The brain was severely swollen and severe cortical surface irritation was observed. Dense SAH and clotting were observed around the aneurysm. Meticulous cleaning of the clot revealed that the aneurysm remained completely clipped. Removal of the clot and mobilization of the right ACA distally from the aneurysm resulted in sudden arterial bleeding on the opposite surface of the vessel. The bleeding was brought under control by applying numerous clips to the artery proximally and distally. No avulsion of tiny branches was observed. The arterial wall became thin and discolored. Longitudinal laceration was also observed and was thought to be the cause of bleeding. The bleeding point was tentatively closed by a clip placed parallel to the wall of the parent artery (Fig. 3). Various clipping procedures were then tried, and finally fenestrated and non-fenestrated angled clips were applied to preserve the lumen of the ACA. He remained comatose after surgery and died on Day 4.

An autopsy was performed. Macroscopic examination found that the ruptured aneurysm was
Fig. 4 Photomicrographs obtained at autopsy. A: Cross-section of the wall dissection showing the lumen of the vessel occupied by fibrin-rich thrombus, as well as leukocytes, atheromatous change, and bleeding into the wall. HE stain, ×40. B: Cross-section of the wall dissection showing the disrupted internal elastic lamina (IEL) and subintimal bleeding in the wall. Arrow indicates the disrupted IEL. Elastica van Gieson stain, ×40. C: Higher magnification of Fig. 4A showing foamy and fibrinoid degeneration in the endothelial layer confirming atherosclerosis. HE stain, ×100. D: The aneurysm wall was devoid of IEL, but this was unrelated to the dissection. Elastica van Gieson stain, ×40. Asterisk indicates the luminal surface.

Discussion

Angiography can indicate dissection of an artery by various findings including string and pearl sign, double lumen, pseudoaneurysm, and tapered occlusion. Unfortunately, there was no chance to perform repeated angiography before the second surgery in our patient.

The autopsy findings indicated that the secondary SAH was due to laceration of the opposite wall of the ACA, located distal to the ruptured aneurysm. Microscopic examination showed that the IEL was torn and subintimal hematoma was believed to have developed. Abundant leukocyte infiltration was probably derived from the dissection and the insult caused by the surgical manipulation of the second operation.

The repeated application of temporary as well as permanent clips at the second operation may have modified the histological findings, in particular the dissection and inflammatory reaction. We found that the active bleeding was not from the aneurysm but from the opposite wall of the ACA, which was torn and had become thin and dark red, so we thought that the histologically verified dissection had mainly occurred following the first operation (Fig. 3).

The potential causes of dissection are numerous and include congenital medial defect,8 fibromuscular dysplasia, polyarteritis nodosa,10 syphilis,14 migraine,2 and moyamoya disease.11 However, the present patient had suffered from none of these diseases, and initial angiography found no signs of arterial dissection. However, head injury,5,7 atherosclerosis,1,2 hypotension are also known to cause dissection. The present patient was known to have hypertension.

We are uncertain as to why the dissection progressed so rapidly following surgery. However, we speculate that the dissection may have resulted from the following mechanisms. First, some atherosclerosis was present in the vessels around the aneurysm. Disruption of the intima is common in
atherosclerotic vessels and intramural hemorrhage or rupture of atheroma sometimes occurs, leading to cerebral thrombosis. Dissection may also originate from bleeding atheromatous plaque, producing a transmural extension. Second, hemodynamic stress from long-lasting hypertension due to ICA occlusion and collateral blood flow can erode the mechanical resistance of the vessel wall. This kind of hemodynamic stress is a known causative factor in de novo dissecting aneurysms in the vertebral artery. Third, surgical manipulation can cause blunt trauma to the vessel. Although the surgical procedures were subtle and not extensive, a small amount of shearing or rotating force on the vessel was likely, particularly in the vicinity of the aneurysm. This additional injury may have helped the dissection to progress.

Only two previous cases of histologically confirmed intracranial dissection related to surgical complications are known. The first case developed after direct clipping and arteriotomy of a ruptured aneurysm in the middle cerebral artery. The second case occurred after tonsillectomy, so inflammatory change may have been involved in the progress of dissection. Neither of the previous patients were salvaged and the diagnosis was determined at autopsy.

This case illustrates any of the numerous predisposing factors for dissection indicate meticulous care during surgery. However, maturation of wall dissection and rupture cannot be completely prevented, although the danger can be substantially reduced by taking appropriate precautions.

References


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