Subarachnoid hemorrhage from a dissecting aneurysm of the middle cerebral artery

Case report

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A case of subarachnoid hemorrhage (SAH) from a dissecting aneurysm of the inferior limb of the middle cerebral artery is reported. The patient's clinical status and the initial and follow-up angiographic appearance of the aneurysm are presented. Diagnosis and treatment are briefly discussed. It is suggested that, if angiography demonstrates luminal narrowing or vascular occlusion in a patient with unexplained SAH, a dissecting aneurysm of the carotid system should be considered as a cause of the hemorrhage.

Key Words: subarachnoid hemorrhage, aneurysm, dissecting, middle cerebral artery

Intracranial dissecting aneurysms are rare, although they have been reported more frequently in recent years and are recognized as a cause of stroke. Clinically, dissecting aneurysms of the vertebrobasilar system are usually associated with subarachnoid hemorrhage (SAH)\(^{4,13,14}\) whereas those of the carotid system present with cerebral infarction. An SAH is an extremely rare complication of a dissecting aneurysm in the anterior circulation.\(^{1,8,14}\) In this paper, we present a case of massive SAH from rupture of a dissecting aneurysm of the inferior limb of the middle cerebral artery (MCA).

Case Report

This 41-year-old previously healthy woman suddenly suffered a severe headache while eating breakfast on August 11, 1987, and was admitted to the Department of Neurosurgery, Kuwana Hospital 40 minutes after the episode.

Examination. On admission, the patient's blood pressure was 120/80 mm Hg, her heart rate was regular at 72 beats/min, and an electrocardiogram was normal. She was alert and cooperative. No abnormality was found on physical examination, and her neurological examination was normal except for headache. Her personal and family medical history was not contributory. Laboratory testing on admission showed normal blood and urine values. Special coagulation studies were also normal.

The initial computerized tomography (CT) scan showed a small amount of SAH in the basal cistern and right sylvian fissure (Fig. 1A), and a ruptured right MCA aneurysm was suspected. Shortly after this, the patient suddenly became unconscious and decerebrate. Rebleeding was suspected and a second CT scan showed that blood clots had increased in the basal cistern and sylvian fissure (Fig. 1B). She recovered consciousness in about 30 minutes, but thereafter exhibited a left hemiparesis. Angiography revealed no saccular aneurysm. Right internal carotid angiography showed segmental luminal narrowing of the inferior limb of the right MCA and a filling defect in the distal artery (Fig. 2A), interpreted as atherosclerotic changes. During angiography, a second rebleed occurred and contrast medium extravasated into the sylvian fissure (Fig. 2B). Left carotid angiography and left vertebral angiography were normal. After angiography the patient was comatose and a third CT scan showed an increase in SAH and a large right sylvian hematoma (Fig. 1C).

First Operation. The first operation was performed
Dissecting aneurysm of the middle cerebral artery

**Fig. 1.** Computerized tomography (CT) scans obtained on the day of admission. A: The first CT scan shows a small amount of subarachnoid hemorrhage in the sylvian fissure. B: Blood clots increase in the second scan. C: A large sylvian hematoma is observed in the third scan.

**Fig. 2.** Right internal carotid angiograms on the day of admission. A: Angiogram showing luminal narrowing of the inferior limb of the middle cerebral artery (arrow) and a filling defect in the distal artery (arrowhead). B: During angiography the second rebleed occurred and contrast medium extravasated into the sylvian fissure.

on the day of admission. This was a decompressive frontotemporoparietal craniectomy with removal of the hematoma. No aneurysm was verified at the bifurcation of the MCA. After surgery, barbiturate therapy was introduced for 3 days; on the 7th postoperative day, the patient became responsive and thereafter her consciousness improved gradually. A severe left hemiparesis persisted.

Repeat angiography was performed 1 month after the onset of the illness. The branches of the right MCA were narrowed, which was interpreted as a result of spasm due to the SAH. No aneurysm was noted. Rehabilitation was started and the left hemiparesis slowly improved. Follow-up CT scans taken 50 days after the onset of disease showed an abnormal enhancing focus in the sylvian fissure.

A third angiography was performed 2 months after the initial onset. An aneurysmal dilatation of the inferior limb of the right MCA was observed at the site where segmental luminal narrowing was shown on the first angiograms (Fig. 3). The lesion consisted of two parts; an inner part with greater contrast enhancement and an outer part with less contrast intensity. Contrast medium was retained in the aneurysm until the venous phase. The lesion was diagnosed as a dissecting aneurysm.

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**Second Operation.** The second operation was performed on February 5, 1988, and consisted of: resection of the aneurysm, a superficial temporal artery (STA)-MCA anastomosis, and cranioplasty. A localized atheromatous plaque was observed in the vessel just proximal to the aneurysm. The aneurysm was resected and the STA was anastomosed to the distal artery. During manipulation of the aneurysm, a part of the outer wall was separated from the aneurysm (Fig. 4A and B).

**Pathological Examination.** The separated outer wall consisted of the adventitia, media, and internal elastic lamina (Fig. 4C). The dome of the aneurysm (Fig. 4D) lacked the internal elastic lamina, and showed fibrocellular proliferation. Intense neovascularization was seen in its outer layer. The intima was thickened circumferentially and deposition of hemosiderin was noted in the aneurysmal wall, suggesting intramural hemorrhage; however, disruption of the intima was not observed. Partially organizing thrombus was seen in the lumen of the aneurysm.

**Postoperative Course.** The patient's postoperative course was uneventful. Postoperative angiography showed disappearance of the aneurysm and patency of the bypass (Fig. 5). She was discharged, on July 21, 1988, at which time she had moderate left hemiparesis but was able to walk without aid.

**Discussion**

**Clinicopathological Correlation**

Intracranial dissecting aneurysms of the carotid system occur in young individuals, and are usually associated with complete stroke resulting from arterial stenosis or occlusion. In the vertebrobasilar system, on the other hand, dissecting aneurysms often manifest as SAH. Yonas, et al., separated these aneurysms into two types on the basis of the clinicopathological findings. Type 1 presents as stroke without SAH, and is characterized by dissection between the elastica and media. In Type 2, dissection occurs between the media and adventitia, and the patients suffer SAH. This second type occurs most frequently in the vertebrobasilar system and recently has been reported more often. Dissecting aneurysms of the carotid system associated with extravascular bleeding are extremely rare and not generally recognized as a cause of SAH.

**Etiology**

The etiology of dissecting aneurysms remains obscure in most reported cases, although dissections have been associated with syphilis, atherosclerosis, periangiitis nodosa, fibromuscular dysplasia, degenerative disease of the arterial wall, migraine, and trauma. In our case, the cause of arterial dissection was not clear, but localized atherosclerosis may have been related to the dissection in view of the operative and pathological findings.

**Angiographic Findings**

A number of angiographic characteristics of dissecting aneurysms have been reported, such as the string sign, rosette sign, and pearl reaction. However, an-
Dissecting aneurysm of the middle cerebral artery

![Image](image_url)

**FIG. 5.** Postoperative right internal (A) and external (B) carotid angiograms showing disappearance of the aneurysm and patency of the bypass.

angiography does not always permit a definitive diagnosis because these signs are also seen in atherosclerotic vascular disease and in many cases angiography reveals only a complete occlusion. The pathognomonic sign for a dissecting aneurysm has been suggested as a double lumen, but this sign is infrequently demonstrated.

After dissection, a variety of lesions can develop including a pseudoaneurysm, variable narrowing of the lumen, and occlusion of the vessel. Follow-up angiography is therefore important for the correct diagnosis when a dissecting aneurysm is suspected. In our case, the first angiograms showed luminal narrowing of the vessel (the string sign), but this finding was interpreted as an atherosclerotic change, and the diagnosis of a dissecting aneurysm was made when the third angiograms showed a pseudaneurysm at the site where luminal narrowing was observed on the first angiograms. Hemorrhage from a dissecting aneurysm may be unrecognized or misdiagnosed as unexplained SAH because of difficulty in the angiographic diagnosis. If angiography reveals luminal narrowing, vascular occlusion, or a nonaneurysm in a patient with SAH, a dissecting aneurysm of the carotid system should be considered as a likely cause of the hemorrhage and follow-up angiography should be performed for definitive diagnosis.

**Treatment**

The best treatment of intracranial dissecting aneurysms producing SAH has not been determined because of the small number of patients and lack of knowledge of the natural history of the disease. However, in dissecting aneurysms of the vertebral artery, proximal arterial ligation has been performed safely and successfully to produce oblitative thrombosis of the dissected segment. If the diagnosis of a dissecting aneurysm of the carotid system can be made preoperatively in a patient with SAH, trapping of the aneurysm or proximal ligation of the artery in combination with extracranial-intracranial bypass surgery would become the surgical treatment of choice in order to prevent rebleeding.

**References**


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