Dissecting aneurysms of the anterior circle of Willis arteries

Report of two cases

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Two cases of spontaneous dissecting aneurysm extending from the supraclinoid portion of the internal carotid artery to the middle cerebral artery are reported in two teenaged patients. Both patients collapsed with a headache on the right side, left hemiparesis, and altered consciousness due to cerebral ischemia. One patient became alert in 2 days; however, his condition rapidly deteriorated 4 days later and he died on the 8th day from massive cerebral infarction. The other patient received a right superficial temporal artery (STA)-middle cerebral artery (MCA) anastomosis 50 hours after his initial symptoms. He improved gradually and is able to walk without help. Cerebral angiograms 3 months after the operation disclosed progressive attenuation of the MCA and dilatation of the anastomosed STA. Artificial collateral flow demonstrated in the postoperative angiogram may have been useful in preventing massive cerebral infarction.

KEY WORDS: dissecting aneurysm, internal carotid artery, middle cerebral artery, revascularization

Case Reports

Case 1

This 14-year-old boy suddenly collapsed while participating in a physical education class on October 20, 1983. He complained of headache in the right temporal region and inability to move his left arm and leg. He was transferred to the hospital 4 hours after the onset of symptoms.

Examination. At admission, he had a severe left hemiparesis and an associated left central facial weakness. His eyes deviated conjugately to the right. He was somnolent but was able to speak well. There were no abnormal physical signs on general examination, and his blood pressure was 108/80 mm Hg. Lumbar puncture soon after admission yielded clear colorless cerebrospinal fluid (CSF) at normal pressure; CSF analysis revealed 14 cells/cu mm, a protein level of 45 mg/100 ml, and a glucose content of 85 mg/100 ml. Computerized tomography (CT) on admission failed to identify any cerebral lesion. Stenosis or occlusion of the MCA was considered to be the cause of his condition.
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Course. It was decided to give the patient low-molecular dextran. On the 2nd day he became alert, and neurological examination showed some improvement. A CT scan disclosed small infarctions in the region of the right basal ganglia (Fig. 1 left). A right carotid angiogram demonstrated occlusion of the distal end of the ICA, with an eccentric stenosis of the MCA. The lumen of the MCA was irregular at that point, but smooth more distally. A distinct pooling of contrast medium was seen above the dark-appearing anterior cerebral artery (Fig. 2).

The patient’s consciousness remained clear for 4 days, and his clinical deficits did not progress; however, on the 6th day, he became drowsy and deteriorated rapidly. A CT scan disclosed massive cerebral infarction of the right hemisphere (Fig. 1 right). On the 7th day, he was placed on a respirator when spontaneous respiration failed. He died 8 days after admission. Permission for a postmortem examination was not obtained.

Case 1

FIG. 1. Computerized tomography scans in Case 1. Left: Scan obtained on the 2nd day after the ictus showing small infarctions in the region of the right basal ganglia. Right: Scan obtained on the 6th day showing massive cerebral infarction in the distribution of the right middle cerebral artery.

Case 2

This 18-year-old man awoke with a severe right temporal headache at 9 a.m. on February 4, 1986. His headache improved, but 30 minutes later he suffered another severe headache and fell backward. He was found by his family at 10 a.m. and was referred to us as an emergency.

Examination. On admission the patient was drowsy with a severe left hemiparesis including the lower half of the face. Increased deep-tendon reflexes were noted on the left side. A CT scan demonstrated no definite lesion, and he was started on anticoagulant therapy. On the 2nd day he was alert. Repeat CT showed the presence of small infarctions in the region of the right basal ganglia (Fig. 3 left). Carotid angiography demonstrated spherical collapse of the right MCA at the first bifurcation.

FIG. 3. Computerized tomography scans in Case 2. Left: Scan obtained on the 2nd day after the ictus showing small infarctions in the region of the right basal ganglia. Right: Scan obtained 3 months after anastomosis showing no additional lesion.
Fig. 4. Right carotid angiograms obtained on the 2nd day after the ictus in Case 2. Left: Lateral view showing a diffuse attenuating branch of the middle cerebral artery (MCA, single arrow) and the irregular lumen of the internal carotid artery (ICA, double arrows). Right: Anteroposterior view showing spherical collapse of the MCA (single arrow) and the irregular lumen of the ICA (double arrows).

cation, attenuation of a branch just distal to it, and luminal irregularity of the supraclinoid portion of the ICA (Fig. 4).

Operation and Postoperative Course. An STA-MCA anastomosis was performed on the 3rd day. Three months after surgery a second angiogram showed severe eccentric stenosis of the supraclinoid portion of the ICA and MCA extending to a posterior branch. The contrast medium in that branch showed a pair of parallel lines. A teardrop of contrast medium extended from the narrow supraclinoid portion of the ICA, where the initial angiogram had shown an irregular lumen, and the STA-MCA bypass was dilated (Fig. 5). Three months after the operation, CT showed small infarctions, as were seen on the earlier study (Fig. 3 right), and the patient's hemiparesis has improved. He can walk without help, but has some left-arm paresis.

Discussion

Dissecting aneurysms of the cerebral arteries are diagnosed by the angiographic or pathological findings. Characteristic angiographic findings have been likened to "string," "false and true lumen," "pearl and string," "pseudoaneurysm," and "tapered occlusion." In Case 1, a string-like narrowing of the MCA was found. Double density of the anterior cerebral artery was revealed on the anteroposterior view, and a pseudoaneurysm arising from the narrowed ICA was visualized on the lateral view. These findings may indicate an intramural pooling of contrast medium in the anterior cerebral artery. On the initial angiogram in Case 2, the MCA at the first bifurcation was pearl-shaped, and a branch showed string-like narrowing. At angiography 3 months later, the MCA was narrower and a pseudoaneurysm was found in the supraclinoid portion of the ICA where the initial angiogram had shown a mild irregular line of contrast medium. This irregular line may indicate an elevated intima. A new dissection of the internal elastic lamina might begin from this point, extend to the MCA, and narrow the lumen of the MCA even more. The follow-up angiogram in Case 2 showed contrast medium in a branch of the MCA forming two parallel lines on the lateral view. The parallel lines may be due to a narrowing of the central part of lumen caused by an elevated intima.

Twenty previous cases of dissecting aneurysms of the circle of Willis arteries have been reported with sufficient clinical, pathological, and angiographic data to form a basis for comparative study. Two of these were apparently induced by head trauma, while the others had no clear etiology. These 20 cases may be divided into three types according to the clinical course: in Type 1 the initial ictus was severe with rapid deterioration and death in several days; in Type 2 the initial event was of moderate severity with obtundation and hemiparesis (with or without aphasia), and the patients showed some improvement for several days before rapid deterioration and death; and in Type 3 the patients improved gradually after the onset but some hemispheric deficits remained.

The primary neuropathological finding in Types 1 and 2 is massive cerebral infarction caused by ischemia resulting from the occlusion or stenosis of the MCA and ICA lumina. In one exception, a patient died due to intracerebral hemorrhage caused by anticoagulant therapy. Angiographic studies in Type 1 showed occlusion or severe stenosis of the MCA and ICA. Peripheral branches of the MCA were barely opacified; in fact, peripheral blood flow in the hemisphere should be minimal. Angiography was repeated in two Type 2 cases: the initial angiogram (when their deficit was improving) showed moderate narrowing of the MCA and ICA, and apparently sufficient perfusion of the
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FIG. 5. Right carotid angiograms 3 months after the ictus in Case 2. Left: Lateral view showing two parallel lines in a branch of the middle cerebral artery (MCA, single arrow) and the dilatation of the superior temporal artery connected to a branch of the MCA (double arrows). Right: Anteroposterior view showing a teardrop of contrast medium extending from the narrowed internal carotid artery (single arrow) and eccentric stenosis of the MCA (double arrows).

hemisphere; however, the second angiogram (during the period of deterioration) disclosed severe stenosis or occlusion of the MCA and ICA, indicating further progression of the dissection in the arterial wall. In other Type 2 cases, angiography was performed only when the patients' condition deteriorated and also showed occlusion of the MCA and ICA. This leads us to the speculation that perfusion in the hemisphere failed when the patients deteriorated. Angiograms performed in Type 3 cases showed sufficient perfusion from spontaneous collateral circulation or via the moderately stenosed MCA and ICA. A CT scan was obtained in only one patient; that patient had a Type 2 disease. In this case, the admission CT scan showed no definite lesion, but a repeat scan during deterioration showed massive cerebral infarction in the distribution of the MCA.

Both of our patients should be included in Type 2 based on the clinical course and/or the neuroradiological findings. The neuroradiological and neuropathological findings in these 22 cases may indicate that if an anastomosis procedure is performed before the second ictus, perfusion through the artificial bypass may prevent massive cerebral infarction. In our Case 2, an STA-MCA anastomosis was performed to gain enough peripheral perfusion before the second collapse. Fortunately, peripheral perfusion beyond this bypass and the stenosed MCA was enough to prevent an increase in cerebral ischemia. In patients with severe injury at the first bifurcation of the MCA, a single anastomosis may not be enough to prevent the cerebral ischemia from worsening; in such cases, a double anastomosis may be necessary. If the caliber of the STA is small, an anastomosis using a venous graft may be effective. We were not able to judge whether proximal ligation of the ICA with the STA-MCA anastomosis was safe or useful. Such a surgical approach should be effective if sufficient perfusion is obtained through the bypass immediately after surgery.

The role of early operative revascularization for patients suffering from cerebral occlusive lesion remains controversial. Some authors have indicated the potential hazards of this approach when brain damage has progressed beyond a certain level. Other authors have reported that early operative revascularization is effective for patients with peripheral perfusion through collateral flow. They indicated that the adequacy of collateral flow, as judged angiographically, is the best predictor of outcome. We suggest that early STA-MCA anastomosis be considered for patients with peripheral perfusion beyond the dissecting aneurysm of the MCA and ICA, even though this policy may lead to unnecessary surgery for Type 3 patients due to the difficulty in separating them from Type 2 patients at an early stage. Perhaps emergency bypass surgery within several hours after the onset of symptoms may be able to save some Type 1 patients.

References


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