

Anterior Cerebral Artery Dissections Manifesting as Cerebral Hemorrhage and Infarction, and Presenting as Dynamic Angiographical Changes

—Case Report—

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Abstract

A 65-year-old woman presented with multiple dissecting aneurysms of the anterior cerebral artery (ACA) manifesting as hemiparesis on the right with dominance in the lower extremity. Computed tomography revealed hematoma in the left frontal lobe, corresponding to the area perfused by the callosomarginal artery. Initial angiography showed string sign and occlusion in the distal portion of the left callosomarginal artery and abnormal feeding suggesting double lumen of the A₂ portion of the left ACA. The patient was treated conservatively under a diagnosis of multiple spontaneous dissecting aneurysms of the left ACA. Repeat angiography on Day 8 showed improvement of the string sign and occlusion in the left callosomarginal artery, and change of the double lumen of the A₂ portion into string sign. Further angiography on Day 36 showed normalization of the left callosomarginal artery and improvement of the string sign in the A₂ portion. Multiple spontaneous dissecting aneurysms of the ACA are extremely rare. Serial angiography beginning in the early stage will be important for correct diagnosis.

Key words: anterior cerebral artery, cerebral hemorrhage, cerebral infarction, multiple lesions, spontaneous dissecting aneurysm

Introduction

Dissecting aneurysms are now recognized as a cause of hemorrhagic or ischemic stroke. Dissecting aneurysms occur most frequently in the vertebrobasilar arteries (VBAs),³⁶⁾ and are relatively rare in the anterior circulation, especially in the anterior cerebral artery (ACA).^{1,4-10,12,14-18,20,21,23,25,26,29,30,32,33,37,38)} Spontaneous intracranial dissecting aneurysms usually develop as a single lesion,^{34,36)} whereas multiple spontaneous dissecting aneurysms of the ACA have not been reported. We describe a case of multiple spontaneous dissecting aneurysms of the ACA.

Case Report

A 65-year-old hypertensive woman was admitted with disturbance of consciousness and right-sided hemiparesis without obvious evidence of trauma on May 9, 1996. She did not habitually smoke or drink. On admission, her Glasgow Coma Scale score was 14. She had hemiparesis on the right with dominance in the lower extremity. Hematological and laboratory examinations found no abnormality. Computed tomography (CT) on Day 0 revealed hematoma in the left frontal lobe (Fig. 1). Angiography on Day 1 showed string sign and occlusion in the distal part of the left callosomarginal artery, and abnormal feeding suggesting double lumen of the A₂ portion of the left ACA (Fig. 2). An-

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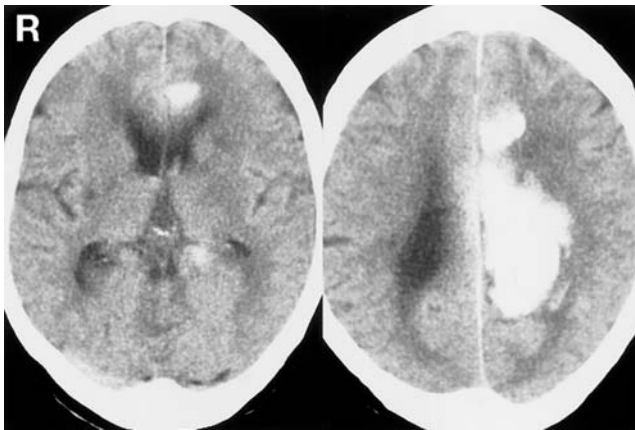


Fig. 1 Computed tomography scans on admission showing high-density lesions in the left frontal lobe and the left lateral ventricle.

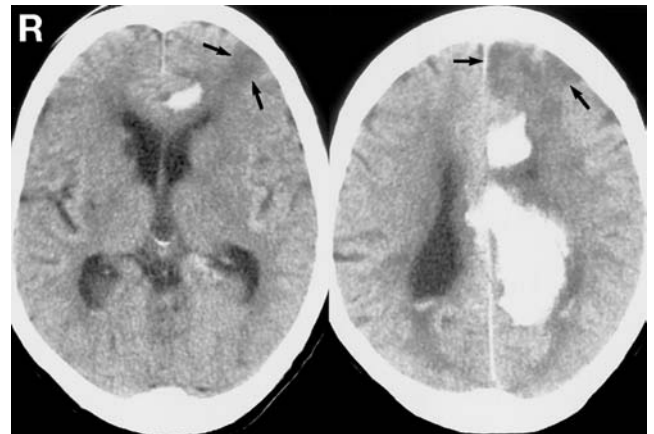


Fig. 3 Computed tomography scans on Day 3 showing a low-density lesion in the left frontal lobe (arrows).

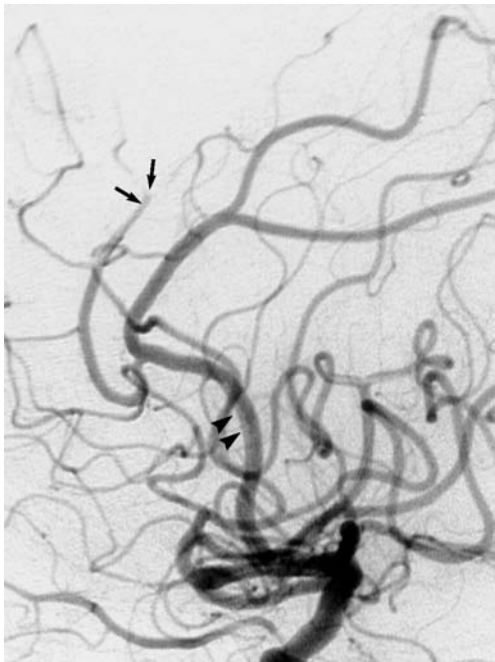


Fig. 2 Left carotid angiogram on Day 1, arterial phase, lateral view, showing stenosis of approximately 20% with a length of 7 mm and occlusion in the distal part of the left callosomarginal artery (arrows), and abnormal feeding suggesting double lumen of the A₂ portion (arrowheads).

angiography found no abnormality in the other vessels, including the proximal portion of the left callosomarginal artery. The patient was treated conservatively under a diagnosis of multiple dissecting aneurysms of the left ACA. She became semi-

comatose on Day 3. CT showed a low-density area in the left frontal region (Fig. 3). Repeat angiography on Day 8 showed improvement of the stenosis and occlusion of the left callosomarginal artery, and change of the double lumen of the A₂ portion of the left ACA into string sign with stenosis of approximately 50% and a length of 1.5 cm (Fig. 4). Third angiography on Day 36 showed normalization of the left callosomarginal artery and improvement of the stenosis in the A₂ portion (Fig. 5). At 6 months after onset, she was transferred to another hospital with moderate disability.

Discussion

Multiple intracranial dissecting aneurysms are very rare. A nationwide study of 357 patients with intracranial arterial dissection found that 35 (10%) had multiple lesions: 15 (43%) had onset of hemorrhagic stroke and 20 (57%) had onset of ischemic stroke.³⁶⁾ In another study of 260 patients with intracranial arterial dissection, 27 (10%) had multiple lesions: 21 (78%) were located in the bilateral VBAs, three (11%) in the unilateral anterior circulation and VBA, and three (11%) in the bilateral anterior circulations.³⁴⁾ However, neither study discussed multiple lesions. We found 13 cases of multiple intracranial dissecting aneurysms, 12 located in the bilateral VBAs^{2,3,13,22,24,27,35)} and one in the anterior choroidal artery and basilar artery.²⁾ On the other hand, only 36 reported cases of spontaneous dissecting aneurysms of the ACA have been reported: 11 with hemorrhage,^{4-6,16,18,29,33,37)} 21 with infarction,^{1,8,9,12,15,18,20,21,23,25,26,29,30,32)} and four with both hemorrhage and infarction.^{7,10,38)} However, multiple dissecting

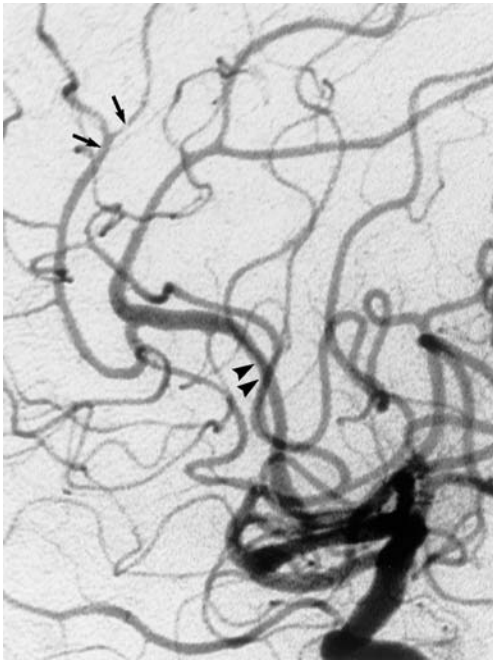


Fig. 4 Repeat left carotid angiogram on Day 8, arterial phase, lateral view, showing improvement of the string sign and occlusion of the left callosomarginal artery (arrows). The double lumen of the A₂ portion of the anterior cerebral artery, observed in the first angiogram, has changed into string sign with stenosis of approximately 50% and a length of 1.5 cm (arrowheads).

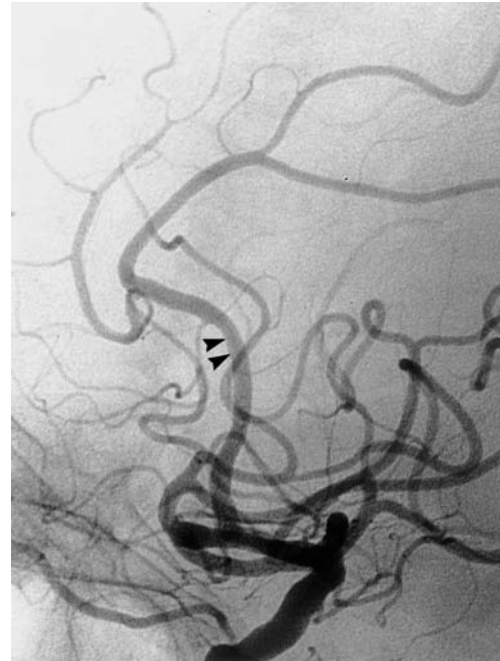


Fig. 5 Left carotid angiogram on Day 36, arterial phase, lateral view, showing normalization of the left callosomarginal artery and improvement (arrowheads) of the string sign in the A₂ portion.

aneurysms of the ACA have not been reported. In addition, the natural history of multiple intracranial dissecting aneurysms has not been elucidated because of the small number of patients with such lesions and the difficulty in detection.

The association between the mechanism of dissection of the arterial wall and clinical onset may involve: Dissection between the internal elastic lamina and the media (subintimal dissection) resulting in ischemic onset through stenosis or occlusion of the dissecting lesion by hemodynamic mechanism; dissection between the media and the adventitia (subadventitial dissection) resulting in hemorrhagic onset; or both of these.³⁹⁾ Pearl and string, double lumen, irregular dilatation, and string and occlusion signs are well known angiographical indicators of dissecting aneurysms. There are no obvious differences in these findings between patients with hemorrhagic onset and those with ischemic onset.^{11,19,25,28,31,40)} These signs often change and sometimes improve due to absorption of hemorrhage into the arterial wall and/or thrombolysis of the thrombus.^{11,19,25,28,31,40)} During the course of

thrombolysis, it is possible that a part of the thrombus can cause remote embolism,^{11,28,32)} and in such a case, angiography may show double lesions.

In this case, we speculated that the double lesions were due to multiple dissecting aneurysms, and not due to remote embolism, because of the following reasons. First, cerebral hemorrhage and infarction on CT corresponded to the area perfused by the callosomarginal artery. Second, the changes of angiographic findings of the A₂ trunk and the distal portion of the callosomarginal artery were demonstrated by serial angiography, whereas the proximal portion of the callosomarginal artery was always normal. Third, embolus from the dissecting part of the A₂ trunk is more likely to occlude the pericallosal artery than the callosomarginal artery. The difficulty in detecting dissecting aneurysms of the ACA, including multiple lesions, may be due to the time lag between CT and angiography, especially at the onset of ischemic stroke. Cerebral infarction due to dissecting aneurysms of the ACA was observed on CT from 1 to 8 days after onset (mean 2.2 days), whereas the initial angiography was performed from 0 to 38 days (mean 6.8 days).^{1,6,8-10,15,23,26,30,32,38)} Therefore, specific angiographical findings of dissecting aneurysms may already have changed during the in-

tervening period. Dissecting aneurysms of the ACA are very rare, so serial angiography beginning in the early stage will be important to detect multiple dissecting aneurysms.

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