De Novo Vertebral Artery Dissecting Aneurysm After Contralateral Vertebral Artery Occlusion

—Two Case Reports—

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Abstract

Two patients developed de novo vertebral artery dissecting aneurysm after contralateral vertebral artery occlusion. A 36-year-old man presented with brainstem ischemia and was treated non-surgically. Subsequent angiography showed spontaneous vertebral artery occlusion at the site of dissection. A 45-year-old man developed subarachnoid hemorrhage due to vertebral artery dissecting aneurysm. He underwent endovascular occlusion of the vertebral artery proximal to the dissecting aneurysm. These patients developed de novo dissecting aneurysm on the contralateral vertebral artery at 13 months and 11 days after unilateral vertebral artery occlusion, respectively. These cases strongly suggest that changes in hemodynamic stress due to unilateral vertebral artery occlusion are related to de novo dissecting aneurysm on the contralateral side. The risk of de novo dissecting aneurysm may be increased by proximal occlusion or trapping of dissecting aneurysm of the contralateral vertebral artery.

Key words: embolization, cerebral infarction, subarachnoid hemorrhage, endovascular treatment

Introduction

Spontaneous dissecting aneurysms of the vertebral artery (VA) have been increasingly identified as a cause of focal neurological deficits due to vertebrobasilar artery ischemia or subarachnoid hemorrhage (SAH). Recently, non-surgical treatment of unruptured dissecting aneurysm with strict control of systemic blood pressure and serial angiography has achieved favorable outcomes.^{5,10,17)} Surgical intervention is recommended in cases with progressive dissection.¹¹⁾

Ruptured VA dissecting aneurysms have a strong tendency toward recurrent rupture associated with a high mortality.⁸⁾ Therefore, if the cerebral blood flow can be preserved, early surgical treatment such as proximal occlusion of the VA or trapping of the aneurysm is widely performed. However, hemodynamic changes following VA occlusion on the rupture side may infrequently result in the de-

velopment of contralateral dissecting aneurysm.^{4,6)}

Here we present two cases of de novo dissecting aneurysm after unilateral VA occlusion, and discuss the clinical course.

Case Reports

Case 1: A 36-year-old man was admitted to our institution with sudden onset of left occipital headache and subsequent dizziness. He had no contributory medical history such as hypertension, arteriosclerotic diseases, or head trauma. Neurological examination showed gait ataxia and hypesthesia in the right lower limb. Initial cerebral angiography demonstrated string sign in the fourth segment of the left VA on the 2nd day after onset (Fig. 1A). Right vertebral angiography showed no abnormalities (Fig. 1B). T₂-weighted magnetic resonance (MR) imaging did not reveal any ischemic lesions, but T₁-weighted MR imaging showed small hyperintense signals located in the left VA, suggesting dissecting aneurysm (Fig. 1C). He was treated non-surgically with control

Fig. 1 Case 1. Left (A) and right (B) vertebral angiograms, and T₁-weighted magnetic resonance image (C) at admission showing a dissecting aneurysm of the left vertebral artery (arrow). Left (D) and right (E) vertebral angiograms 2 weeks after onset showing spontaneous occlusion of the left vertebral artery and subsequent dilation of the right vertebral artery (arrow). Left vertebral angiogram (F) 13 months after the initial admission demonstrating a de novo dissecting aneurysm of the right vertebral artery (arrowheads).

of blood pressure and bed rest for 2 weeks. Anticoagulant or antiplatelet agents were not used. Cerebral angiography performed 2 weeks after admission revealed total occlusion of the left VA at the site of the dissecting aneurysm (Fig. 1D). Cerebellar blood flow was preserved through the dilated right VA (Fig. 1E). The patient had no further neurological deterioration and was discharged 6 weeks after the onset of the disease.

Thirteen months later, he developed sudden headache and became unconscious. Emergent vertebral angiography showed stenosis of the right VA in the fourth segment, suggesting de novo dissecting aneurysm (Fig. 1F). He developed apnea due to extensive brainstem infarction and died 6 days after the second admission. Autopsy was not performed.

Case 2: A 45-year-old man suffered sudden onset of severe headache. He had no particular medical

history. On admission to our institution, he was alert and displayed no neurological deficits. Cranial computed tomography failed to show SAH clearly, but lumbar puncture revealed bloody cerebrospinal fluid. Emergent cerebral angiography showed irregular stenosis and dilation of the left VA proximal to the posterior inferior cerebellar artery (Fig. 2A). The right VA appeared normal (Fig. 2B). Two days after onset, T₁-weighted MR imaging showed hyperintense signals corresponding to the lesion, suggesting arterial dissection (Fig. 2C). The diagnosis was SAH due to ruptured VA dissecting aneurysm. Test occlusion of the left VA showed no changes in his neurological condition, so endovascular occlusion of the left VA was performed proximal to the dissecting aneurysm to prevent subsequent rupture. After a detachable coil (IDC18, 5 mm × 15 cm; Boston Scientific Japan, Tokyo) was placed just proximal to the aneurysm, the left VA

Fig. 2 Case 2. Left (A) and right (B) vertebral angiograms at admission showing irregular stenosis (arrow) and dilation of the left vertebral artery and normal right vertebral artery. T₁-weighted magnetic resonance image (C) 2 days after onset showing a high intensity area corresponding to the stenosis of the left vertebral artery (arrow). Right vertebral angiogram (D) and T₁-weighted magnetic resonance image (E) 2 weeks after occlusion of the left vertebral artery showing de novo dissections of the right vertebral and basilar arteries (arrowheads).

was occluded at the proximal portion of the coil using two detachable balloons (GVB15; Nippon BXI Inc., Tokyo). Postoperative angiography showed good filling of the left posterior inferior cerebellar artery through the right VA.

Two weeks after VA occlusion, the patient complained of numbness of his hands and legs, and tetraparesis developed within several hours. Vertebral angiography demonstrated irregular dilation and stenosis of the right VA, extending to the basilar artery (Fig. 2D). The left VA remained occluded. T₁-weighted MR imaging showed hyperintense signals through the basilar artery, suggesting de novo arterial dissection (Fig. 2E). Barbiturate, hypervolemic, and anticoagulant therapy were started under controlled respiration. The patient was weaned from the respirator, but severe tetraparesis remained due to brainstem infarction.

Discussion

Cerebral angiography and MR imaging initially showed no abnormal findings of the unilateral VA in our patients, so we concluded that the de novo dissecting aneurysms developed after occlusion of the contralateral VA. In our institution, 27 patients with dissecting VA aneurysm underwent endovascular treatment due to SAH or progressive dissection and 10 patients were treated non-surgically in the last 10 years. Two of these 37 patients developed de novo dissecting aneurysm, so we speculate that the incidence is not low. One case of de novo dissection was reported to occur after unilateral VA occlusion among 31 patients with intracranial vertebrobasilar artery dissection.⁴⁾

The mechanism responsible for de novo dissection after unilateral VA occlusion is not obvious.

Changes in hemodynamic stress and the unique clinicopathological features of dissecting aneurysms may contribute to the pathogenesis of de novo VA dissection. The diameter of the contralateral VA increases after occlusion of the unilateral VA, as seen in our cases. In cases of bilateral VA dissecting aneurysms, the contralateral VA dissection increased in size after trapping of the unilateral ruptured aneurysm, and dilation of the contralateral VA after unilateral VA occlusion indicates substantial elevation of arterial hemodynamic stress.^{12,15)} Increasing hemodynamic stress may also be a significant factor.⁶⁾

The clinicopathological features of dissecting aneurysms have been discussed in detail.^{1-3,9,10,13,14,16)} The main mechanism of arterial dissection is diversion of the arterial stream into a weakened arterial wall. The characteristic pathological features are defect or fragmentation of the internal elastic lamina, intimal thickening, and medial degeneration.^{1-3,9,10,13,14)} Multiple small intramural hemorrhages, which are isolated and non-contiguous in the walls of the VA, are an important factor.³⁾ Generally, these intramural hemorrhages are thought to be associated with the disruption of vasa vasorum or new vessels.¹⁶⁾ Underlying connective tissue defect, such as reduction of elastic fibers in the media or external lamina, may be related to the bilaterality of VA dissections.^{1,3)}

Based on these findings, our cases may have carried the risk of the dissection in the contralateral VA due to occult vulnerability. In this situation, increased hemodynamic stress following unilateral VA occlusion may have been significant in the development of the de novo VA dissection.

Recent advances in endovascular approaches to treatment may resolve these problems associated with changes in hemodynamic stress. To preserve the normal blood flow and treat the dissecting aneurysms, Guglielmi detachable coils were positioned in the dissecting aneurysms after placing stents in the true lumen of the VAs.⁷⁾ This method seems to be more effective than conventional occlusion therapy for parent arteries, but is less accessible at present due to the lack of an appropriate stent.

In conclusion, ruptured or progressive VA dissecting aneurysms need to be treated with surgical or endovascular occlusion of the parent arteries. However, our experience suggests that some patients may develop additional dissecting lesions after VA occlusion. Under such circumstances, increased hemodynamic stress and occult vulnerability of the VA walls may be critical in the development of contralateral de novo dissecting aneurysms. The risk of de novo dissecting aneurysm

may be increased by proximal occlusion or trapping of dissecting aneurysm of the contralateral VA.

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