

Occlusion of the Vertebral Artery Secondary to Dissection of the Subclavian Artery

—Case Report—

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Abstract

A 34-year-old man presented with occlusion of the left vertebral artery (VA) secondary to dissection of the left subclavian artery manifesting as vertigo, nausea, vomiting, and neck pain. On admission, he was alert with left limb and truncal ataxia. Magnetic resonance (MR) imaging and MR angiography showed left cerebellar infarction and occlusion of the left VA. Conventional angiography and three-dimensional computed tomography (3D-CT) angiography showed stenosis with thrombosed pseudolumen of the left subclavian artery, and occlusion of the left VA. Presumably the idiopathic dissection of the left subclavian artery had reached the orifice of the left VA, and an embolism from the dissection had caused occlusion of the VA, leading to cerebellar infarction. After one month, he was discharged without severe neurological deficits. Idiopathic dissection of the subclavian artery is very rare. 3D-CT angiography is very useful for the diagnosis of arterial dissection.

Key words: cerebellar infarction, subclavian artery, dissection, three-dimensional computed tomography angiography

Introduction

Subclavian artery dissection is usually caused by trauma,^{4,7)} for example, seat belt injury during traffic accidents,^{4,7)} sports injury such as swinging a golf club,⁵⁾ iatrogenic injury during catheterization or angioplasty,¹⁾ or following aortic dissection with or without Marfan's syndrome.⁸⁾ The typical initial clinical symptoms are subclavian steal syndrome,⁶⁾ ischemic symptoms caused by embolism from the dissection or the dissection reaching the orifice of the vertebral artery (VA),⁵⁾ and a difference in blood pressure between the right and left arms.²⁾ We describe a case of idiopathic dissection of the subclavian artery manifesting as cerebellar infarction, which was depicted clearly by three-dimensional computed tomography (3D-CT) angiography.

Case Report

A 34-year-old man, with a history of cerebral infarction and untreated hypertension 3 years previously, suddenly developed vertigo, nausea, vomiting, and

neck pain while sleeping on April 16, 2003. He observed the course of the symptoms during the night, and the symptoms improved slightly. He consulted a local doctor 3 days after the onset. Magnetic resonance (MR) imaging identified cerebellar infarction. He was then referred to our institute.

On admission, he was alert and complained of neck pain, left limb ataxia, and hemi-numbness on the left side as sequelae of the previous cerebral infarction. His blood pressure was 206/125 mmHg. Family medical history was not contributory. Laboratory testing on admission showed that protein-C, protein-S, anti-cardiolipin antibody, lupus circular coagulant, c-antineutrophil cytoplasmic antibody (ANCA), p-ANCA, anti-thrombin III, supplement body, and crioglobulin levels, and blood sedimentation rate were all within the normal limits.

Initial MR imaging in our hospital depicted an old lesion in the right basal ganglia and a fresh lesion in the left cerebellum (Fig. 1), and MR angiography identified occlusion of the left VA (Fig. 2). The initial diagnosis was dissection of the left VA. Anti-platelet therapy and blood pressure reducing therapy were begun. Angiography depicted stenosis

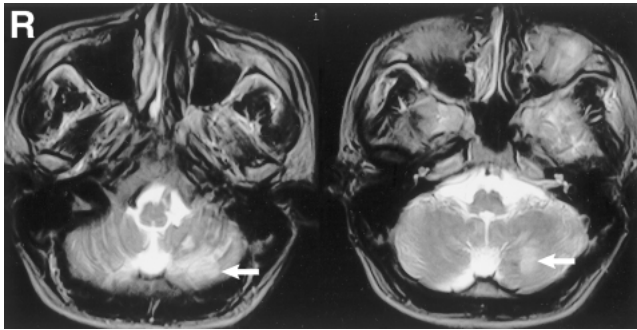


Fig. 1 T₂-weighted magnetic resonance images on admission demonstrating infarction (arrow) in the left cerebellum.

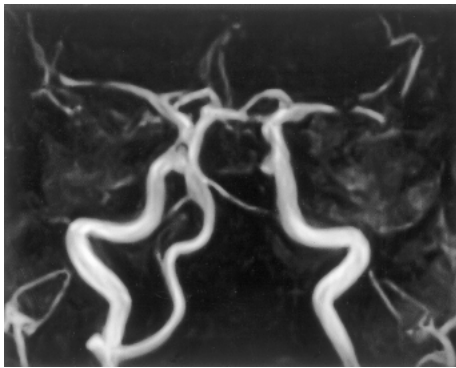


Fig. 2 Magnetic resonance angiogram on admission demonstrating occlusion of the left vertebral artery.



Fig. 3 Left subclavian angiograms showing stenosis of the proximal portion of the left subclavian artery (arrow) and occlusion of the left vertebral artery (right) not visible at the time of the previous cerebral infarction (left).

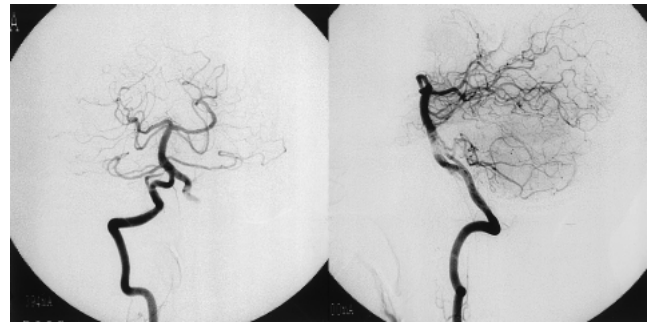


Fig. 4 Right vertebral arteriograms showing the stump of the distal portion of the left vertebral artery.

of the proximal portion of the left subclavian artery and occlusion of the left VA, which were not visible on the angiogram performed at the time of the previous cerebral infarction (Fig. 3), and right vertebral arteriography depicted the stump of the distal portion of the left VA (Fig. 4). 3D-CT angiography showed the stenotic portion of the subclavian artery consisted of thrombosed pseudolumen caused by dissection of the subclavian artery (Fig. 5). Based on these findings, the diagnosis was idiopathic dissection of the left subclavian artery extending to the orifice of the left VA, and embolism from the dissection causing occlusion of the left VA, resulting in cerebellar infarction. One month after the stroke, he was discharged with numbness on the left.

Discussion

Our patient had no systemic disease or history of trauma, so this case is thought to be idiopathic

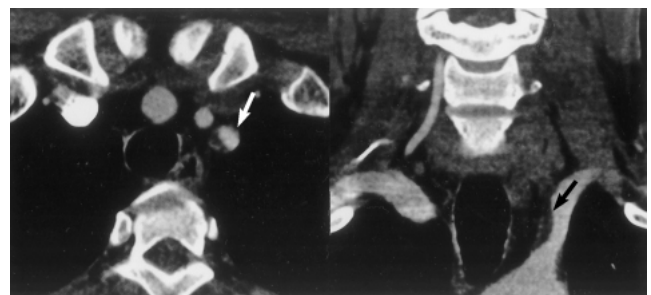


Fig. 5 Three-dimensional computed tomography angiograms showing that the stenotic portion of the subclavian artery consisted of thrombosed pseudolumen caused by dissection of the subclavian artery (arrow).

subclavian artery dissection. The mechanism causing aortic dissection is thought to involve the angle between the ascending and descending aorta,

because the abrupt change in direction of blood flow may cause damage to the aortic wall.⁸⁾ A higher incidence of arterial dissection is associated with left VA of aortic origin, compared to left VA of left subclavian artery origin or right VA of right subclavian artery origin, suggesting that the incidence of dissection is related to direct arterial pulsatile flow.³⁾ The left subclavian artery is thought to receive stronger pulsatile flow than the right subclavian artery. In our case, we think that the left subclavian artery received more severe stress because of his hypertension.

The typical initial clinical symptoms are due to reduced blood flow in the subclavian artery and embolism from the dissection or the dissection reaching the orifice of the VA. In our case, right vertebral arteriography showed only the stump of the left VA. Therefore, occlusion of the left VA was probably caused by emboli from the dissection of the subclavian artery or VA. Our patient presented with vertigo due to embolic infarction of the cerebellum, but the symptom was mild and atypical. The stenosis of the subclavian artery at the dissection was not severe. Therefore, the dissection was probably not the direct cause of the symptom. The neck pain may have been caused by dissection of the orifice of the VA.

The only angiographic finding indicating dissection in our case was the stenosis, which was not visible on the angiogram performed at the time of the previous cerebral infarction. No double lumen, pooling, slow filling, or intimal flap were detected. We think that idiopathic dissection of the subclavian artery occurs more often than is believed, and should be included in the differential diagnosis. In our case, 3D-CT angiography clearly showed the double lumen and wall irregularity, strongly suggesting dissection. We consider that 3D-CT angiography is useful for the identification of dissection because the thrombosed pseudo-lumen of the dissection can be visualized.

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