

Multiple Cerebral Infarction Caused by Spontaneous Subclavian Artery Dissection

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ABSTRACT

Subclavian artery dissection (SAD) is mainly caused by aortic arch deformity, trauma, and vascular intervention. However, a spontaneous dissection that is idiopathic is very rare. Ischemic stroke can occur as a complication of SAD. Majority of ischemic strokes from previous reports of SAD have occurred in the posterior circulation area, such as the cerebellum, brainstem, occipital lobe, or cervical spinal cord. This report presents a rare case of multiple cerebral infarctions in the anterior circulation as well as the posterior circulation area of patients with spontaneous SAD; hence, the author aims to report this case with a literature review.

KEYWORDS: Subclavian artery; Dissection; Cerebral infarction

INTRODUCTION

Subclavian artery dissection (SAD) is mainly caused by aortic arch deformity [1], trauma [2] and vascular intervention [3], and an idiopathic cause of spontaneous dissection is very rare [4,5]. Symptoms of SAD may include chest, back, or neck pain; claudication of the upper extremities; dizziness; nausea; vomiting; and visual disturbance [1-8]. Complications of SAD can lead to ischemic strokes and subclavian steal syndrome [2,5,6,8]. In this study, the ischemic stroke due to SAD occurred in the posterior circulation area, including the vertebrobasilar and posterior cerebral artery territories [2,5-8]. The author encountered a rare instance of multiple cerebral infarctions in the anterior circulation as well as the posterior circulation areas of patients with spontaneous SAD; hence, the author aims to report this case with a literature review.

CASE PRESENTATION

A 72-year-old female patient visited the emergency room due to sudden onset of right hemiparesis while she was on a bus half an hour before her scheduled hospital visit. A few minutes before the neurological abnormality occurred, chest tightness was felt at the upper chest area. Her medical history indicated that she was

taking a calcium channel blocker (felodipine 5mg) and a diuretic (indapamide 2.5mg) once a day for hypertension. There was no history of connective tissue disorders, trauma, or vascular intervention. The patient is a non-smoker and a non-alcohol beverage drinker.

Initial vital signs were as follows: blood pressure, 160/80 mmHg; pulse rate, 78/min; respiratory rate, 20/min; and body temperature, 37.3 °C. In the neurological examination, the patient was alert and conscious and manifested dysarthria, right central type facial palsy, Medical Research Council grade 4 right hemiparesis, and positive Babinski's sign on the right side. The National Institute of Health Stroke Scale score was 5 points.

Non-enhanced computed tomography (CT) and CT angiography (CTA) of the brain showed Sylvian fissure dot sign, which is an early marker of thromboembolic occlusion of the distal middle cerebral artery (MCA) branches, and occlusion in the M2 branch of the left MCA (Figure 1). Perfusion CT images demonstrated decreased cerebral blood flow, cerebral blood volume, and delayed time to peak in the left MCA territory (Figure 1). Recombinant tissue plasminogen activator (rt-tPA) was immediately administered

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intravenously after the diagnosis of acute cerebral infarction. Diffusion-weighted magnetic resonance imaging (MRI) during rt-tPA administration showed acute cerebral infarction in the posterior inferior cerebellar artery territory of the left cerebellum;

however, no lesions were found in the left MCA territory (Figure 2). Fluid-attenuated inversion recovery (FLAIR) images revealed hyperintense vessel signs in the left MCA branches (Figure 2). All neurological deficits resolved after the rt-tPA administration.

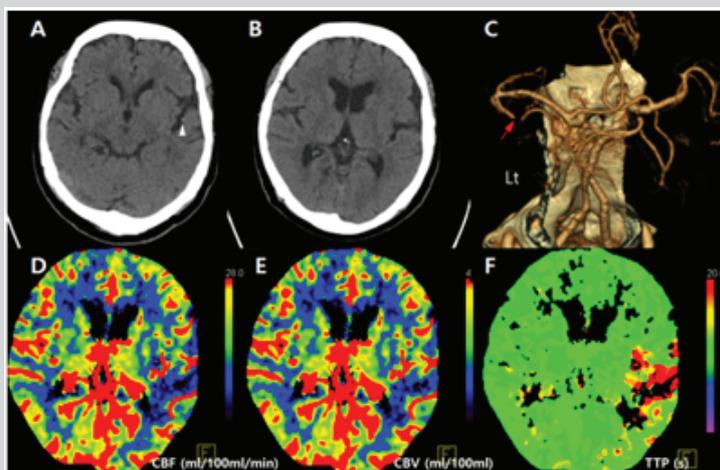


Figure 1: Initial brain computed tomography (CT), CT angiography (CTA), and CT perfusion images of the patient. Non-enhanced brain CT images (A and B) show Sylvian fissure dot sign, which is an early marker of thromboembolic occlusion of the distal middle cerebral artery (MCA) branches (arrowhead). Brain CTA image (C) reveals occlusion in the M2 branch of the left MCA (arrow). Perfusion CT images (D, E, and F) demonstrate decreased cerebral blood flow (D) and cerebral blood volume (E), and delayed time to peak (F) in the left MCA territory.

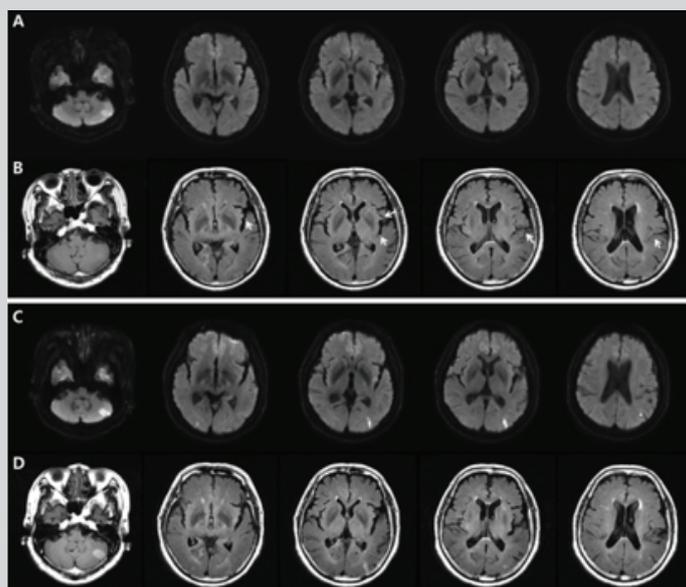


Figure 2: Diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) images of the patient. Initial brain DWI (A) and FLAIR images (B) show acute left cerebellar infarction and hyperintense vessel signs in the left middle cerebral artery (MCA) branches (arrow), but there is no diffusion restriction in the left MCA territory. Follow up brain DWI (C) and FLAIR images (D) reveal multiple focal infarctions in the left cerebellum, left parieto-occipital, left insular cortex, and right occipital region. Hyperintense vessel signs of the left MCA have disappeared.

Laboratory examinations such as complete blood count, blood chemistry, lipid profile, coagulation profile, blood sugar levels, erythrocyte sedimentation ratio, high-sensitivity C-reactive protein levels, fibrinogen, and homocysteine levels were normal. Serum anti-nuclear antibody and rheumatoid factor tests were negative. Serum D-dimer level was 0.79 $\mu\text{g}/\text{mL}$ (normal: 0-0.5). Electrocardiogram and echocardiogram were normal. Atrial fibrillation was not noted

during the three-day cardiac monitoring at the stroke unit or in the 24-hour Holter monitoring.

In the neck CTA conducted on the second day of admission, severe segmental stenosis was found in the left subclavian artery proximal to origin of the vertebral artery with a double lumen, intimal flap, and an intraluminal hematoma compatible with arterial dissection (Figure 3). In the follow-up brain MRI conducted on the

third day of admission, in addition to the left cerebellar lesions in the initial images, newly developed focal lesions were found in the bilateral occipital lobe, the left insula, and the left parietal lobe (Figure 2). The left MCA was recanalized on brain MR angiography.

From the second day of admission, 75 mg of clopidogrel and 100 mg of aspirin were administered for secondary prevention of cerebral infarction and discharged a week later without any neurological deficits.



Figure 3: Neck computed tomography angiography of the patient. Maximum intensity projection images (A and B) show segmental severe stenosis in the left proximal subclavian artery (white arrow). Axial images (C and D) reveal double lumen, hypodense intimal flap (black arrowhead), and intraluminal hematoma (black arrow) compatible with arterial dissection.

DISCUSSION

This case is a rare example of multiple cerebral infarctions involving the anterior and posterior circulation areas in a patient with spontaneous SAD. Spontaneous SAD is very rare and can cause cerebral infarction due to its complications. Previous reports of cerebral infarction caused by SAD involved the posterior circulation area, such as the cerebellum, medulla, occipital lobe and cervical spinal cord [2,5,6,8]. Pathomechanisms of cerebral infarction caused by SAD include thrombosis of the dissection itself, artery-to-artery embolism to the distal region of the dissection, and hypoperfusion [8]. The thrombus of the dissection area involving the origin of the vertebral artery can directly block the vertebral artery, or an artery-to-artery embolism can occur in the distal portion of the dissection involving branches of the vertebrobasilar artery and posterior cerebral artery. If a dissection occurs in the proximal portion of the vertebral artery origin, subclavian artery stenosis can cause subclavian steal syndrome, which can cause a decrease in cerebral perfusion.

The patient in this case was admitted to the emergency room due to right hemiparesis. Although no lesions were seen in the diffusion-weighted imaging, occlusion in the M2 branch of the left MCA was identified on CT angiography, and decreased perfusion was confirmed in the CT perfusion study. Therefore, the right hemiparesis of the patient was caused by the left M2 occlusion. On the third day of admission, brain MRI and MR angiography showed newly developed focal lesions in the bilateral occipital lobe, left insula, and left parietal lobe other than the left cerebellar lesion in the initial image, and the left MCA was recanalized. The path mechanism of the left cerebellar and both occipital lesions may account for the artery-to-artery embolization from the left vertebral artery, and the left insula and parietal lesions may account for the dissolution of the thrombus following administration of the rt-tPA. Whether the mechanisms of the left MCA occlusion and left cerebellar infarction occurred around the same time are unclear. Several hypotheses were considered. First, it is possible that as the patient was older, had a history of high blood pressure, the left MCA infarction caused by arteriosclerosis may have occurred simultaneously, apart from the SAD. Second, although no atrial

fibrillation was identified on electrocardiogram, 24-hour Holter monitoring, and during the three-day cardiac monitoring in the stroke unit, an undetected paroxysmal atrial fibrillation may have caused the cardiac embolism. Third, severe stenosis from the SAD may have caused turbulence and backflow of blood to the aortic arch, causing MCA occlusion along the left carotid artery. Although no evidence was seen in the human subclavian artery, 55-75% of MCA stenosis in animal models using transcranial Doppler ultrasonography can cause blood turbulence and backflow and may lead to distal embolism [9]. In the study of transesophageal echocardiographic evaluation of mobile plaque motion, retrograde and rotational blood flow in the thoracic aorta probably exists in all patients with systemic emboli and mobile, protruding aortic atheroma's [10]. Therefore, the authors of this study asserted that retrograde cerebral embolism from distal aortic plaques is theoretically possible. Severe stenosis in the left proximal subclavian artery in this case may have caused the turbulent flow and backflow of blood.

The use of rt-tPA when arterial dissection is present may aggravate the dissection, and the guidelines for acute stroke care do not recommend the use of rt-tPA in a patient suspected with aortic dissection [11]. There are no clear guidelines for SAD; however, the use of rt-tPA could aggravate it. Because thrombolytic therapy should be performed as soon as possible if it is adaptive, no further tests are recommended to detect arterial dissection. In this case, the patient manifested chest pain prior to the onset of neurologic symptoms; however, arterial dissection was not suspected because of its rarity. Therefore, rt-tPA was used, and arterial dissection was found on follow-up imaging. It is not known whether arterial dissection became aggravated due to the absence of a vascular examination prior to thrombolytic therapy. As there were no neurologic symptoms suggestive of worsening arterial dissection, the clinical significance is thought to be negligible. If arterial dissection had been known, thrombolysis should have not been performed.

CONCLUSION

Spontaneous SAD without trauma, vascular malformation, or vascular intervention is uncommon. Most cerebral infarctions

caused by SAD occur in the posterior circulation area; however, they may occur in the anterior circulation area.

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