

A Case of Cerebellar Infarction Caused by Acute Subclavian Thrombus Following Minor Trauma

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Subclavian steal syndrome caused by an acute thrombus is very rare. We present a case of cerebellar infarction with proximal subclavian artery thrombosis. A 56-year-old woman was admitted for sudden vertigo. One day prior to admission, she received a shoulder massage comprised of chiropractic manipulation. On examination, her left hand was pale and radial pulses were absent. Blood pressure was weak in the left arm. Downbeat nystagmus and a right falling tendency were observed. Brain MRI showed multiple acute infarctions in the left cerebellum. The findings of Doppler ultrasonography in the left vertebral artery were compatible with a partial subclavian artery steal phenomenon. Digital subtraction angiography demonstrated a large thrombus in the left subclavian artery. After heparin infusion, thrombus size markedly decreased. Cerebellar infarction caused by acute subclavian thrombosis following minor trauma is rare, but the thrombus can be successfully resolved with anticoagulation.

Key Words: Subclavian steal syndrome, brain infarction, thrombosis

INTRODUCTION

Subclavian steal syndrome is most commonly caused by atherosclerosis. However, an acute thrombus and trauma can cause this syndrome in very rare cases.^{1,2} Here, we report a case of acute cerebellar infarction presented with subclavian steal, developed by the thrombus in the subclavian artery.

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CASE REPORT

A 56-year-old woman was admitted to the emergency room for sudden onset vertigo and right sway on awakening. She had received an intense chiropractic manipulation of her shoulders the day before admission. Her medical history included

diabetes mellitus, which had been managed for six years. On examination, blood pressure was 137/71 mmHg and 113/56 mmHg in the right and left arms, respectively. Her left hand was pale and cold with an absent radial pulse on the left (Fig. 1A). An audible subclavian bruit was present on the left side. Downbeat nystagmus and a right falling tendency during tandem gait were observed. No motor and sensory deficits were shown. No musculoskeletal abnormalities that could have been implicated in compression of the subclavian artery were evident. Diffusion-weighted MRI revealed multiple acute infarctions in the left cerebellar hemisphere and vermis, compatible with the medial territory of

the left posterior inferior cerebellar artery (Fig. 1B). Doppler ultrasonography of the left vertebral artery showed bidirectional wave. Marked mid-systolic deceleration was seen with a sharp peak immediately before deceleration and return of forward direction during diastole. A provocative hyperemic cuff test (occlusive inflation of a blood pressure cuff for three minutes with subsequent release) of the left arm aggravated reverse flow. Therefore, these findings were compatible with a partial subclavian artery steal phenomenon (Fig. 2A and B). Digital subtraction angiography (DSA) showed a large thrombus in the proximal left subclavian artery as well as retrograde flow through the left vertebral ar-

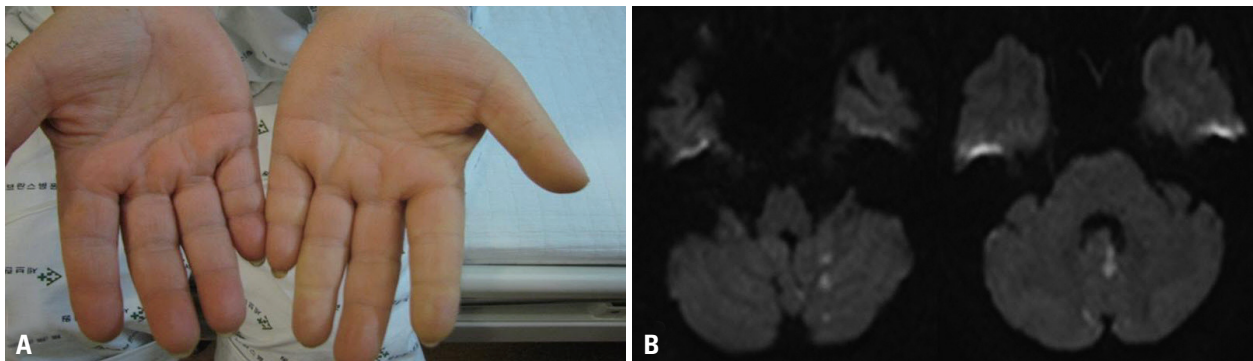
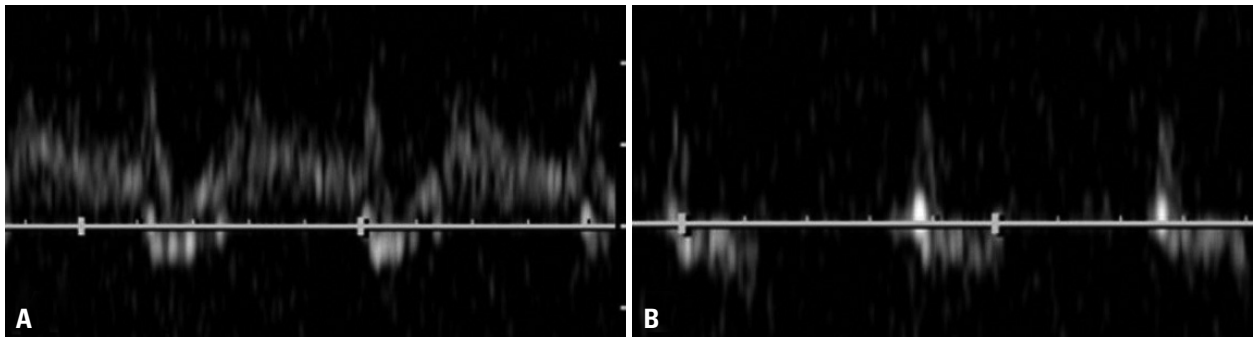


Fig. 1. (A) The left hand of the patient was pale and cold. (B) Acute infarctions in the left cerebellar hemisphere and vermis are shown in diffusion-weighted MRI.

Before anticoagulation



After anticoagulation

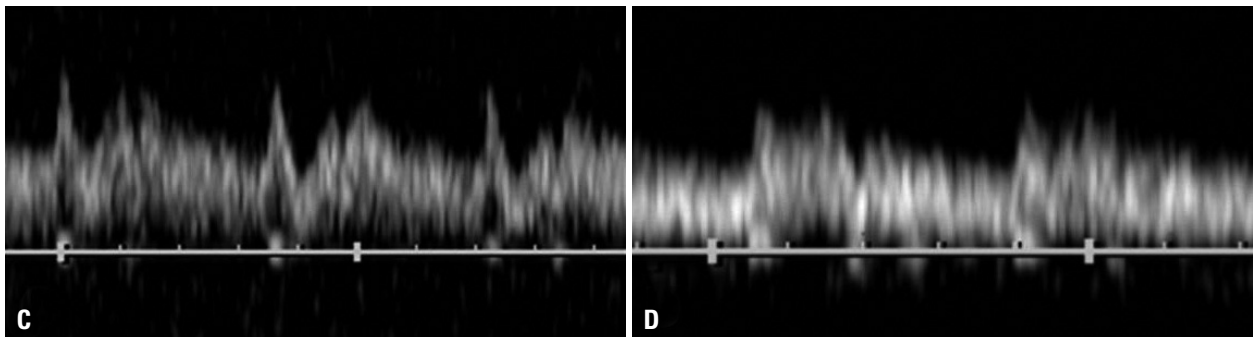


Fig. 2. (A) Initial Doppler ultrasonography of the left vertebral artery shows bidirectional wave. (B) A provocative hyperemic cuff test on the left arm aggravates flow reversal, which is compatible with a partial subclavian steal syndrome. (C) After anticoagulation, follow-up Doppler ultrasonography shows that a systolic deceleration is still seen, but there is no retrograde flow. (D) No augmented flow reversal is noted in the hyperemic cuff test, which is compatible with a pre-steal phenomenon.

tery. No vertebral artery dissection was observed (Fig. 3A). Neck CT showed a narrowed space between the anterior and lateral scalene muscles in the left side, more so than on the right. Blood tests for coagulation abnormalities, including protein C, S, lupus anticoagulant, and anti-cardiolipin antibody, were normal. After two days of treatment with heparin, warmth returned to her left arm. Follow-up Doppler ultrasonography showed improvement of the steal phenomenon. A systolic deceleration was still seen, but there was no retrograde flow and no augmented reversal flow was noted in the hyperemic cuff test, which was consistent with pre-steal phenomenon (Fig. 2C and D). Follow-up DSA at 10 days after admission revealed markedly decreased thrombus size (Fig. 3B).

DISCUSSION

In this case, a cerebellar infarction was caused by acute subclavian thrombosis following minor trauma. After anticoagulation, the thrombus successfully resolved without complication.

The subclavian steal syndrome is a well known phenomenon that is comprised of a stenosis or occlusion of the subclavian artery.³ Because the subclavian artery is close to the origin of the vertebral artery, subclavian artery stenosis can cause retrograde blood flow through the ipsilateral vertebral artery. Along with ischemic symptoms of the ipsilateral upper arm, neurological symptoms, including dizziness and blurred vision, could occur due to impaired circulation to the posterior brain.² Subclavian artery stenosis is usually as-

ymptomatic. Reported prevalence of subclavian steal syndrome is between 0.6% and 6.4%.^{4,5} Because of the abundant collateral blood supply to the head, neck and shoulder, neurological symptoms are infrequent. Among the patients with subclavian artery steal syndrome, only 5.3% suffer neurological symptoms.⁶

In this patient, the mechanism of stroke might be an embolic occlusion of the medial territory of the posterior inferior cerebellar artery because these lesions do not correspond to border zone cerebellar infarction.⁷ Moreover, initial Doppler ultrasonography showed only a partial steal, which would be consistent with an embolism from a thrombus in the left subclavian artery that caused posterior circulation infarction. Nevertheless, a hemodynamic mechanism due to the subclavian steal could not completely be ruled out.

The question of why there is thrombus in the subclavian artery is uncertain. Our patient did not have a history of coagulopathy and there was no laboratory evidence of a coagulation disorder either. One day prior to the stroke event, the patient had chiropractic manipulation involving pulling her shoulders back forcefully. According to the Virchow triad, formation of a thrombus depends on constituents of the blood, vessel, and hemorrheology. The finding of a narrower inter-scalene triangle in the left on neck CT suggests an unfavorable anatomical structure for which repetitive strong posturing may induce thrombus formation. Our patient did not have a vertebral artery dissection, this can occur after chiropractic maneuvering, which can also induce posterior circulation ischemia.⁸

It is noteworthy that serial Doppler ultrasonographies were useful for monitoring thrombus resolution in the sub-

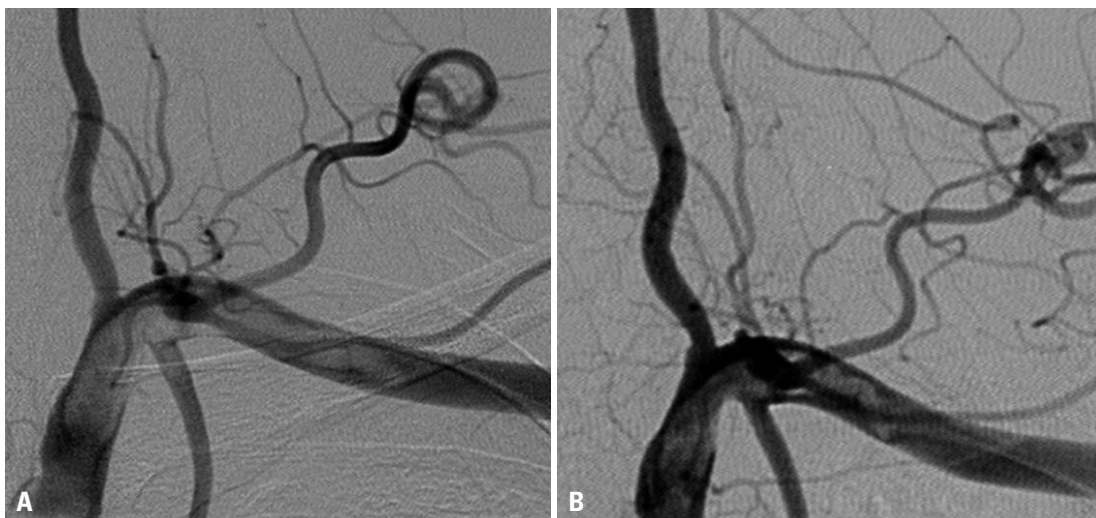


Fig. 3. (A) Digital subtraction angiography shows a large thrombus in the proximal left subclavian artery. (B) Ten days after admission, a follow-up study shows markedly decreased thrombus size.

clavian artery. Before anticoagulation, a partial subclavian steal waveform was seen on Doppler ultrasonography. The systolic flow reversal in the partial subclavian steal is related to the pressure gradient between the vertebral artery and ipsilateral arm distal to the stenosis. A partial steal can be converted to a near or complete steal through a provocative hyperemic cuff test. After anticoagulation, the Doppler changed from a partial steal to a pre-steal wave pattern. In the pre-steal, the vertebral artery flow is always antegrade, but with a transient sharp deceleration of blood flow after the first systolic peak.⁹

Patients with subclavian steal syndrome caused by atherosclerotic stenosis have been successfully treated with surgery.⁵ Recent treatment with percutaneous transluminal angioplasty is also promising.¹⁰ However, when the cause of the subclavian steal is an acute thrombus, as in this case, surgical or interventional treatment poses a high risk for recurrent embolism. Anticoagulation seems to be the most reasonable therapy to prevent recurrent embolization from a subclavian thrombus, and it may be helpful in thrombus resolution.

In summary, we report a patient with cerebellar infarctions caused by acute subclavian thrombus following minor trauma. In a patient with cerebellar infarction and discordant blood pressure readings between arms, subclavian thrombus should be suspected.

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