

A rare association of subclavian steal syndrome with bilateral carotid artery stenosis: a case report

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ABSTRACT

Bilateral carotid artery stenosis in the context of subclavian steal syndrome is an extremely rare finding. We report the case of a 75-year-old woman who presented with a transient ischemic attack. Bilateral internal carotid stenosis associated with left subclavian steal syndrome was diagnosed. Left internal carotid endarterectomy was performed under locoregional anesthesia. Inflation of a blood pressure cuff reversed the neurologic symptoms that appeared after internal carotid clamping. This rarely reported case remains a challenge to diagnose and treat because of its complex mechanisms and multiple risk factors. Our case highlights the importance of the surgical strategy adopted and the need for a good initial assessment. (*J Vasc Surg Cases Innov Tech* 2023;9:101243.)

Keywords: Bilateral carotid stenosis; Carotid endarterectomy; Subclavian steal syndrome; Vertebral artery stenosis

Subclavian steal syndrome (SSS) is defined as retrograde blood flow in the vertebral artery due to proximal subclavian artery occlusion.¹ The prevalence of this condition is believed to be between 0.6% and 6.4% and is four times more frequent on the left side.¹ Most patients are asymptomatic. Symptoms usually develop once the stenosis has become severe enough to result in vertebrobasilar insufficiency or upper extremity ischemia.² A blood pressure difference of 20 mm Hg is usually noted between the two arms of patients presenting with SSS.^{2,3} The diagnosis is made by Doppler ultrasound, followed by either magnetic resonance angiography or computed tomography angiography.³ Like internal carotid artery (ICA) stenosis, the most common cause of SSS is vascular atherosclerosis. Treatment can range from lifestyle interventions and medical treatment to endovascular or surgical interventions.

CASE REPORT

A 75-year-old woman presented with left homonymous hemianopia that lasted <10 minutes. She had a long history of hypertension and dyslipidemia. She is an active smoker and has smoked two packs a day for 45 years. In addition, she was taking

prednisone for giant cell arteritis. No other associated symptoms were reported. The patient provided written informed consent for the report of her case details and imaging studies

Physical examination revealed a bilateral carotid bruit and an arm blood pressure difference of 120 mm Hg (right vs left, 220 mm Hg vs 100 mm Hg). The peripheral upper extremity pulses were palpable and symmetric. The brain magnetic resonance imaging findings were normal. The electrocardiographic and chest radiographic findings were unremarkable. Her laboratory test results showed elevated total cholesterol and triglycerides. The remainder of the laboratory results were within the normal range, including a C-reactive protein of 3 mg/L.

Doppler ultrasound showed 95% stenosis in the left ICA with extensive atheromatous plaque. The right ICA had 70% stenosis. Doppler ultrasound also confirmed complete reversal of blood flow in the left vertebral artery (Fig 1), with a decline in flow velocity after an early systolic upstroke.

Computed tomography angiography also showed 70% stenosis of the right ICA and preocclusive stenosis (95%) of the left ICA (Fig 2). The degree of stenosis of the left ICA was estimated by combining a direct measurement using the NASCET (North American symptomatic carotid endarterectomy trial) criteria and hemodynamic parameters such as a peak systolic velocity >400 cm/s, an end-diastolic velocity >150 cm/s, and ICA/CCA peak systolic velocity ratio >5.0.⁴ In addition, the left ICA showed severe stenosis in the proximal left subclavian artery before the origin of the left vertebral artery. All the results were suggestive of permanent left SSS with bilateral carotid artery stenosis. All vessels in the intracranial domain were permeable and had a good caliber in the anterior and posterior zones, including a complete circle of Willis. From these findings and after consultation with a neurologist, a radiologist, and an internist, a transient ischemic attack in a patient presenting left SSS associated with bilateral carotid stenosis was diagnosed.

Left carotid endarterectomy was performed the next day under locoregional anesthesia, allowing for continuous clinical monitoring of the patient's neurologic status. In addition, a

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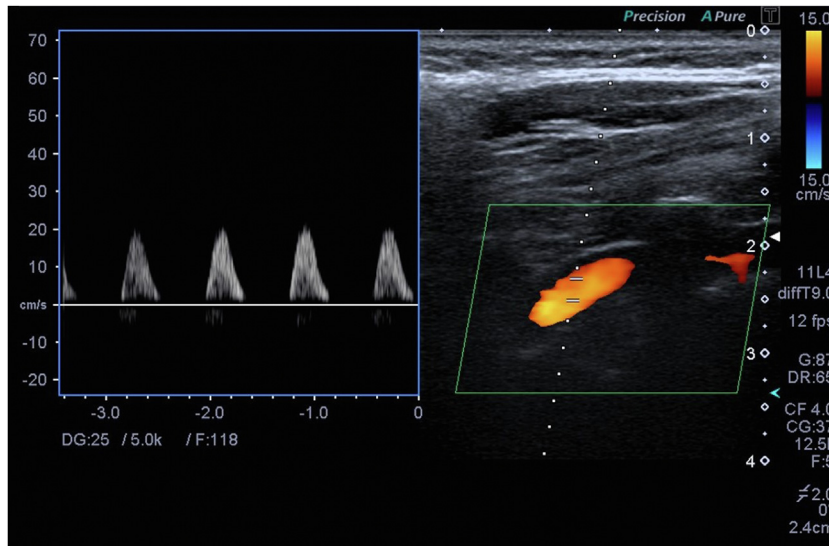


Fig 1. Color Doppler ultrasound showing reversal of blood flow in the left vertebral artery.

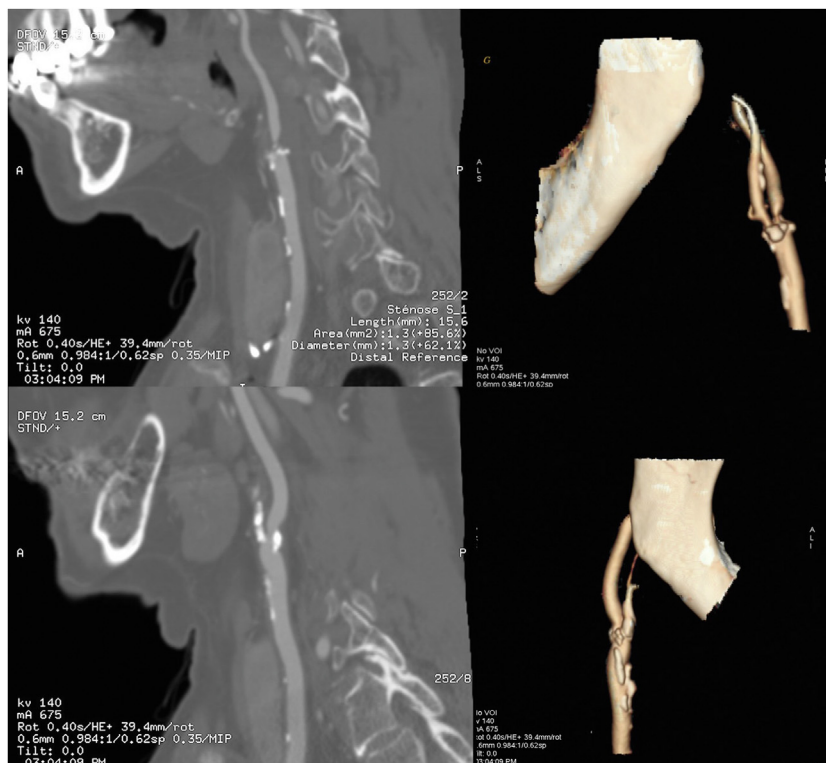


Fig 2. Computed tomography angiogram showing significant stenosis in the left internal carotid artery (ICA; Upper Row) and right ICA (Lower Row).

deflated blood pressure cuff was placed on our patient's left arm to inflate in case of decreased cerebral perfusion. The patient maintained a stable high blood pressure of 160 mm Hg during the surgical procedure until carotid artery clamping. Following carotid artery clamping, she became confused and agitated. No focal neurologic signs were noted. Carotid artery clamping was then removed, and her blood pressure increased. After complete recuperation, clamping of the ICA was again

attempted at a systolic blood pressure of 210 mm Hg. Neurologic symptoms were again obvious; however, they disappeared with inflation of the blood pressure cuff placed on her left arm. Her blood pressure was lowered to 140/52 mm Hg immediately before unclamping the carotid artery. No shunt was used during the procedure. Endarterectomy was performed using the classic eversion technique, and direct closure was performed without the use of a patch. Her postoperative course was uneventful.

The patient was monitored for 24 hours in the intensive care unit and was discharged the next day with daily low-dose aspirin and a higher dose of a statin. At 18 months postoperatively, the patient remained totally asymptomatic.

DISCUSSION

SSS results from a narrowed subclavian artery, causing reversed blood flow in the vertebral artery and stealing blood from the unaffected subclavian artery to supply the upper limb.¹ A difference of >20 mm Hg is usually noted between the two upper extremities.¹ Most patients are asymptomatic because the residual blood flow remains greater than that required for daily activities. Symptoms are more frequent in patients with a higher difference in blood pressure, with 38.5% of patients with a >50-mm Hg difference presenting with symptoms.⁵ Symptoms can vary from vertebrobasilar insufficiency to, more rarely, upper limb ischemia.²

The main determinant of the appearance of neurologic symptoms in such patients is the capacity to increase the collateral blood flow.⁶ Therefore, an association with bilateral carotid artery stenosis might precipitate neurologic symptoms via a reduction in the compensatory arterial flow.⁷ In the present case, the pressure-directed vertebral artery flow preferentially supplied the requirements of the upper arm. The concomitant reduction in compensatory arterial flow might have precipitated the symptoms.

A transient ischemic attack (TIA) was suspected in our patient. The American Heart Association and American Stroke Association recommend a number of tests to evaluate a patient with a suspected TIA.⁸ These include neuroimaging within the first 24 hours of symptom onset, a cervical vasculature assessment for atherosclerotic lesions, a cardiac assessment, and routine blood tests. The normal magnetic resonance imaging findings, the carotid artery duplex ultrasound and computed tomography angiography findings, and the normal cardiac assessment support the diagnosis of a TIA. With her history of giant cell arteritis and current cortisone maintenance therapy, an initial inflammatory origin for the subclavian artery stenosis could not be eliminated. However, with the low level of C-reactive protein at her presentation and the computed tomography findings, it was highly unlikely that vasculitis played a role in the current episode.

Our treatment goal was to primarily recanalize our patient's left ICA. According to the 2021 European Society for Vascular Surgery guidelines for atherosclerotic carotid disease,⁹ left endarterectomy was indicated. Our patient underwent left carotid artery endarterectomy under locoregional anesthesia with continuous neurologic evaluation. In the case of reduced cerebral perfusion, elevating the blood pressure frequently results in symptom reversal. This was not the case with our patient. However, inflating the blood pressure cuff placed on

her left arm did reverse the symptoms. Inflation of the cuff placed on her left arm to a higher level than her systolic pressure resulted in a reduction of the flow in the left brachial artery, diminishing the left subclavian steal phenomenon and improving our patient's cerebral circulation.

SSS is a rare finding, and its association with bilateral carotid artery stenosis is extremely uncommon. Only two similar cases have been reported in the literature, both of which involved chronic neurologic symptoms and were treated differently. Aketa et al¹⁰ reported the first case in 2017. Their patient was treated with percutaneous angioplasty of the innominate and right ICAs. Song et al⁷ reported the second case in 2021. Their patient was treated with left carotid artery endarterectomy and left subclavian angioplasty. In contrast, our patient was completely asymptomatic before experiencing a TIA, despite having 70% and 95% stenosis in the right and left ICAs, respectively, and significant occlusion of the left subclavian artery.

CONCLUSIONS

SSS is mostly seen in patients with cardiovascular risk factors. Although it is most frequently asymptomatic, it should be suspected in patients presenting with chronic neurologic signs. High-risk patients should be assessed for carotid artery stenosis, which could aggravate their prognosis. Treatment options include medical, surgical, and endovascular procedures. Treatment should be tailored to each patient depending on the patient's overall clinical situation. When operating on such cases, we insist on placing a blood pressure cuff and maintaining good cerebral perfusion.

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