

CASE REPORT

Dizziness and syncope after subclavian steal: A case report of a rarely symptomatic, common vascular disorder

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Abstract

Subclavian steal syndrome (SSS) is a manifestation of vertebrobasilar artery insufficiency due to stenosis of the proximal subclavian artery. It is a common vascular disorder; however, most patients are asymptomatic, and the disorder is commonly found incidentally during imaging. Nevertheless, some patients may present with arm ischemia or signs and symptoms of vertebrobasilar insufficiency, such as vertigo, dizziness, diplopia, ataxia, dysarthria, and syncopal episodes. We present a case of a male patient who presented with sudden onset dizziness, syncopal episodes, and blood pressure discrepancy between his arms. Diagnosis was a challenge, and the patient underwent several investigations, including basic bloodwork, electrocardiography (ECG), echocardiography, brain computed tomography (CT), and coronary CT angiography (CTA). Narrowing in the proximal left subclavian artery was found during coronary CTA, which confirmed the diagnosis of SSS.

Introduction

Subclavian steal is a phenomenon that occurs when there is a reversal of blood flow in the ipsilateral vertebral artery due to subclavian artery stenosis proximal to the vertebral artery.¹ This phenomenon was discovered in 1960 by Contorni when he observed retrograde blood flow in a patient with an absent radial pulse.² A year later, Reivich associated this phenomenon with neurological symptoms,³ which resulted in the introduction of the term subclavian steal syndrome (SSS) by Fisher in 1961.⁴ SSS comprises a group of symptoms that arises from the consequences of reversed blood flow in the ipsilateral vertebral artery. Right and left brachial artery blood pressure (BP) discrepancy of more than 20 mmHg should raise suspicion of SSS, in addition to symptoms of vertebrobasilar artery insufficiency.

Another clinical finding associated with SSS is delayed or reduced pulse in the affected limb.⁵ The prevalence of subclavian steal phenomenon was reported to be between 0.6% and 6.4%.⁵ Not all patients with SSS are symptomatic; in a large study on extracranial artery disease, Fields et al. found that only 5.3% of SSS patients experienced neurological symptoms.⁶ We report the case of a patient with neurological symptoms who was diagnosed with SSS after a vigorous series of investigations.

Case Presentation

A 48-year-old man with multiple comorbidities, including diabetes mellitus, hyperlipidaemia, and hypertension, presented with a sudden onset of dizziness and a spinning sensation associated with palpitations. He had previously experienced multiple episodes of syncope associated with hand elevation that subsided after rest. Otherwise, there were no other aggravating factors that led to the attack; it was unrelated to postural changes or eye closure. During the attack, the patient was unable to walk due to unsteadiness. The patient reported no complaints of chest pain or neurological deficits. Upon arrival in the emergency department, his initial examination and basic blood investigations, including packed cell volume and electrolytes, were all unremarkable. There was a discrepancy between the systolic BP of his right and left arms of more than 20 mmHg, with the BP in his right arm being higher than his left. Further investigations were carried out to determine the cause of dizziness. Electrocardiography (ECG) showed sinus rhythm with no evidence of acute ischemic changes and no evidence of previous cardiac ischemia. On echocardiography, ejection fraction was normal and there was mild mitral and tricuspid valve regurgitation. No evidence

of acute brain infarction, aside from multiple old lacunar infarctions, was observed on non-contrast brain computed tomography (CT). The patient's symptoms improved throughout the hospital stay. With multiple comorbidities, cardiac CT angiography (CTA) was performed for stratification of the coronary vessels and revealed severe short-segment stenoses at the origin of left subclavian artery (approximately 95% stenosis) and just before the left vertebral artery (approximately 80% stenosis) secondary to non-calcified atherosclerotic plaques (**Figure 1** and **2**). Interdisciplinary discussions between the vascular and interventional radiology teams

agreed on a trial of angioplasty of the stenotic part. The patient subsequently underwent percutaneous angioplasty (PTA) with a transfemoral artery approach. A 6 mm x 40 mm PTA balloon (Bard Ultraverse™ 035 PTA dilatation catheter) was advanced to the stenotic part with a 0.035-inch guidewire (Terumo GLIDEWIRE® hydrophilic coated guidewire). Angioplasty was performed at the stenotic part. Post-angioplasty imaging revealed satisfactory opening of the stenotic parts; therefore, no stent was deployed. The pre- and post-angioplasty images are presented in **Figure 3**.



Figure 1. Thoracic CT angiography in coronal view. The red arrows show severe stenoses at the origin of left subclavian artery (approximately 95% stenosis) and just before the left vertebral artery (approximately 80% stenosis) secondary to non-calcified atherosclerotic plaques.

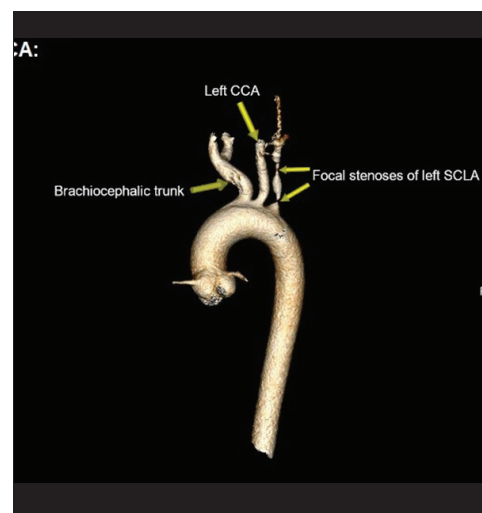


Figure 2. 3D volume rendering technique (VRT) of the main thoracic vessels. Focal stenoses at the left subclavian artery (SCLA).

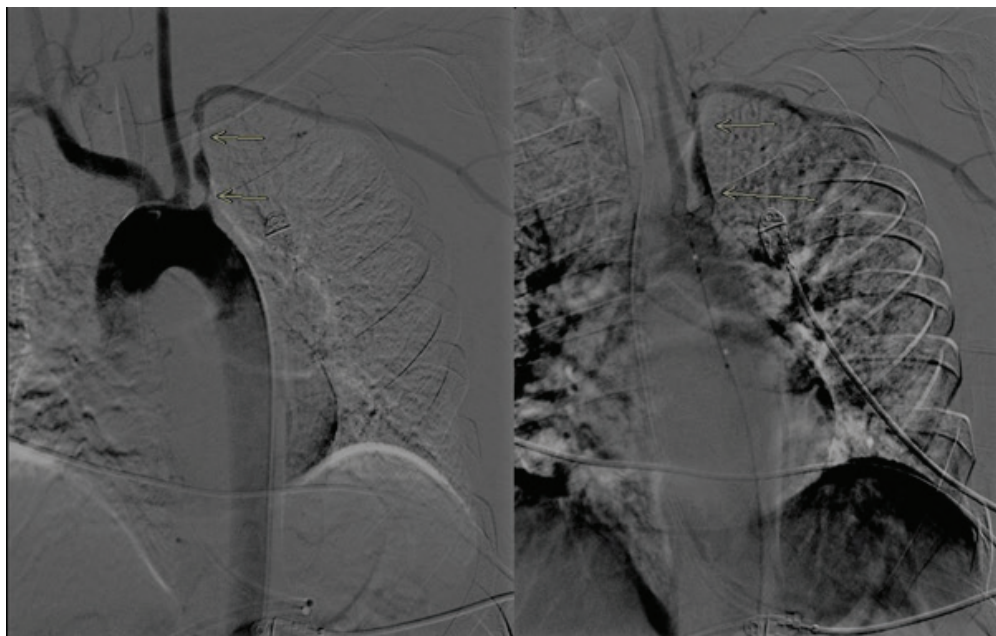


Figure 3. Pre-angioplasty digital subtraction angiography (DSA) (left) shows two significant stenoses at the proximal left subclavian artery. Post-angioplasty DSA (right) shows satisfactory re-opening of the stenotic parts.

Discussion

Whilst most of the patients who have subclavian steal phenomenon are asymptomatic, they may present with arm ischemia or neurological symptoms, such as dizziness, ataxia, vertigo, and syncope. These symptoms are due to vertebrobasilar arterial insufficiency, as blood flows retrograde from the basilar artery to the ipsilateral vertebral artery and subsequently to the distal subclavian artery. The main cause of this phenomenon is the presence of stenosis at the proximal subclavian artery, resulting in reduced blood flow distally. As a result, compensatory blood flow from the basilar artery leads to the 'steal' of blood from the brain circulation.⁷ The main aetiology of SSS is atherosclerotic vascular disease, although other causes include Takayasu arteritis, external compression of the subclavian artery, coarctation of the aorta, and congenital anomalies. Risk factors for SSS are the same as those for atherosclerotic vascular disease which include smoking, hyperlipidaemia, diabetes mellitus, hypertension, family history, and age.^{6,8}

Clinically, SSS should be suspected if there is absent or reduced ipsilateral radial pulse, a cervical or supraclavicular bruit, or an upper limb systolic blood pressure discrepancy of more than 20 mmHg.⁵ Tan et al. showed that there was a linear correlation between increasing arm blood pressure discrepancy and the occurrence of SSS symptoms.^{5,9} Further imaging with carotid duplex ultrasound, magnetic resonance (MR) angiography, or CT angiography is needed to establish the diagnosis.¹

Treatment options are either open bypass or endovascular intervention. Endovascular intervention involves angioplasty or stenting the stenotic part; it is safer than open bypass,

with low morbidity and mortality. Wang et al. reported overall survival rates of patients receiving endovascular angioplasty with stenting of 98.2 % at 12 months and 84.5% at 5 years. The main complication of stenting is intra-stent thrombosis. Inability to cross the occlusion during endovascular intervention can necessitate conversion to open axillo-axillary bypass or carotid-subclavian bypass; the latter option is preferred due to its effectiveness and improved long-term patency.¹⁰

Conclusion

Although SSS is common, it rarely presents with neurological symptoms. A thorough history of symptoms such as arm ischemia, recurrent syncopal episodes, dizziness, vertigo, diplopia, and ataxia, in addition to a BP discrepancy between arms greater than 20 mmHg, should alert general practitioners of this diagnosis. Further imaging, such as CT or MR angiography, should be performed to confirm the diagnosis.

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Conflicts of interest

The authors declare that there are no conflicts of interest.

Patients' consent for the use of images and content for publication

Written and verbal consent was obtained from the patient for publication of this case report with the accompanying images.

What is the implication to patients?

- When a patient presented with vertigo, dizziness, diplopia, ataxia, and syncopal episodes, especially when associated with strenuous activity of the hands, a physician should also be thinking of SSS as part of differential diagnosis.
- A physician must perform blood pressure measurement on both arms. A discrepancy greater than 20 mmHg has been proven to be a sensitive threshold for the detection of SSS.
- CT or MR angiography is needed to establish the diagnosis of SSS.
- SSS is now treatable with minimally invasive endovascular surgery.

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