# Subclavian steal syndrome without subclavian stenosis

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### ABSTRACT

Subclavian steal syndrome (SSS) has been well described in the setting of subclavian stenosis. We describe an unusual case of SSS caused by a high-flow arteriovenous dialysis fistula in the absence of subclavian stenosis, provide a review of the literature, and propose that arteriovenous fistula-induced SSS is an underdiagnosed cause of syncope in this population of patients. (J Vasc Surg Cases and Innovative Techniques 2017;3:129-31.)

Subclavian steal syndrome (SSS) occurs when there is symptomatic vertebrobasilar flow reversal due to "steal" from the ipsilateral subclavian artery. The most common cause is proximal stenosis of the ipsilateral subclavian artery.<sup>1</sup> Symptoms include vertigo, syncope, and arm ischemia and are often induced by exertion of the affected arm.<sup>1</sup> Here, we present a rare case of SSS due to a high-flow arteriovenous fistula (AVF) in the absence of subclavian stenosis. Written consent was obtained from the patient to publish the case report.

## **CASE REPORT**

A 73-year-old man on hemodialysis through a right arm brachiocephalic fistula presented to the hospital with episodes of syncope that were increasing in frequency. His comorbidities included coronary artery disease, hypertension, diabetes, and end-stage renal disease secondary to diabetic nephropathy. Syncopal episodes were occurring daily and would last several seconds. Attacks would occur both on and off hemodialysis. It was noted that his episodes of loss of consciousness could be reversed with compression of the fistula. The patient denied symptoms of arm ischemia or claudication, and the episodes were independent of arm use.

Evaluation of the fistula revealed aneurysmal degeneration. There were no signs of distal limb ischemia. A full cardiac and neurologic workup, including echocardiography, cardiac catheterization, computed tomography angiography of the chest, magnetic resonance imaging of the head, and electroencephalography, failed to explain his symptoms. Computed tomography angiography of the chest did not demonstrate appreciable ipsilateral subclavian stenosis (Fig 1).

Monthly fistula flow measurements during the 3 months before consultation were elevated. Flow measurements were

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**Fig 1.** Computed tomography angiography demonstrating the brachiocephalic (*B*), common carotid (*C*), vertebral (*V*, arrow), and subclavian (*S*) arteries. Note the absence of subclavian stenosis.

>2.9 L/min, with a maximum velocity of 3.7 L/min. Duplex ultrasound was performed (Fig 2), which demonstrated mild to-andfro flow in the brachial artery distal to the fistula that resolved with fistula occlusion. Duplex ultrasound evaluation of the right vertebral artery demonstrated an elevated peak systolic velocity of 516 cm/s, without an identifiable stenosis, followed by dampened waveforms and diastolic flow reversal. This constellation of findings is indicative of active vertebral artery steal. The right common carotid artery was patent with normal waveforms and velocities. Together, these investigations suggested that the patient was suffering from SSS due to a high-flow fistula in the absence of subclavian artery stenosis.

The patient underwent ligation of the fistula after placement of a dialysis catheter, with a plan to perform de novo fistula creation at a later date. His symptoms resolved immediately postoperatively, and he remained symptom free at his postoperative follow-up 2 weeks later. Additional follow-up with repeated duplex ultrasound imaging was scheduled within the first postoperative year. Unfortunately, the patient died before this could be accomplished.

#### DISCUSSION

Here, we describe a rare case of SSS caused by a highflow AVF in the absence of subclavian stenosis. The

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**Fig 2.** Ultrasound evaluation. **A**, Right vertebral artery with severely dampened waveforms and to-and-fro flow, indicative of steal in transition to reversal flow. **B** and **C**, The right subclavian and innominate arteries demonstrate high-velocity, low-resistant flow without visualization of a stenosis.

patient was successfully treated with fistula ligation. We identified only four prior case reports describing SSS without subclavian stenosis in a dialysis patient. Schenk first described this scenario in a 28-year-old man with ataxia.<sup>2</sup> Evaluation revealed a high-flow fistula, with flow speeds of 5.8 L/min. Symptoms resolved after fistula plication to reduce flow.<sup>2</sup> Boettinger et al<sup>3</sup> similarly described a patient with a high-flow fistula that resulted in a posterior circulation infarct. Maiodna et al<sup>4</sup> described a patient with ataxia and fainting spells who was treated with fistula ligation. Finally, Kargiotis et al<sup>5</sup> described a case of steal in a patient with bilateral AVFs. Symptoms failed to resolve after ligation of one fistula, but bilateral ligation was not performed<sup>5</sup>

Myocardial ischemia from subclavian-coronary steal syndrome has also been described in the setting of a left internal thoracic artery coronary bypass graft and an ipsilateral AVF in the absence of a left-sided subclavian stenosis.<sup>6-9</sup> These patients were similarly treated with fistula ligation<sup>7</sup> or implantation of the left internal thoracic artery to the ascending aorta.<sup>9</sup>

Taken together, our report and the reports of others provide mounting evidence that a high-flow AVF is a potentially underdiagnosed cause of symptomatic SSS. Given the small number of studies describing this phenomenon, it is unclear if there is a flow speed at which the probability for development of a steal syndrome increases. However, it has been suggested that AVF flow speeds in excess of 2 L/min can have clinically significant effects on cardiac function,<sup>10</sup> and these data can potentially be extrapolated to the subclavian-vertebral arterial system. Obtaining vascular studies with and without compression could be of additional diagnostic utility.

Dialysis-dependent patients often have significant cardiovascular comorbidities that can make diagnosis of AVF-induced subclavian steal challenging. In our report, the diagnosis was entertained only after cardiac and neurologic causes were excluded. We suggest determining AVF flow speeds in any hemodialysis patient who presents with symptoms of posterior circulation insufficiency and obtaining noninvasive vascular studies if flow exceeds 2 L/min or if there has been a recent increase in measured flow during hemodialysis. Obtaining vascular studies with and without fistula compression could be of additional diagnostic utility.

Once the diagnosis of SSS is made, treatment options include fistula ligation and revision.<sup>2.4.7</sup> A variety of techniques have been described to manage the AVF that is causing either high-output heart failure or steal syndromes. In general, these techniques aim to reduce flow through the fistula or to redirect flow to the ischemic organ. The described techniques include ligation, surgical plication, banding, and revascularization using distal inflow.<sup>11</sup> Characteristics of the fistula, options for

additional vascular access, and the patient's preference will dictate the surgical approach. In the case presented here, the decision was made to proceed with ligation as the patient was having significant symptoms, the fistula had undergone aneurysmal degeneration, and there were options for fistula creation in the contralateral arm.

## CONCLUSIONS

In this report, we describe a rare case of vertebrobasilar insufficiency caused by a high-flow fistula in the absence of subclavian stenosis. The patient was successfully treated with fistula ligation. We suggest that SSS should be considered in any hemodialysis patient who presents with neurologic symptoms consistent with posterior circulation insufficiency.

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