

# Endovascular Treatment of Vertebral Artery Steal Syndrome from an Arteriovenous Fistula Between the Internal Mammary Artery and Superior Vena Cava

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Vertebrobasilar insufficiency resulting from subclavian steal syndrome and high flow through hemodialysis fistulas has been described. The present report describes a case of vertebral artery steal syndrome from an arteriovenous (AV) fistula between the right internal mammary artery and superior vena cava with the unusual pattern of antegrade flow through the vertebral artery in the setting of classic symptoms of steal. The AV fistula likely developed during earlier cardiac catheterizations. The patient presented with progressively worsening gait and limb ataxia. Angiography demonstrated the AV fistula and antegrade flow through the vertebral artery. Coil embolization of this high-flow fistula resulted in complete resolution of symptoms.

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**Abbreviations:** AV = arteriovenous, RIMA = right internal mammary artery, SVC = superior vena cava

“STEAL” is a term that has been used to describe the pathophysiologic process of blood flow diversion from one vascular bed to another lower resistance-vascular bed (1,2). Subclavian steal syndrome is perhaps the most commonly described type of steal syndrome. It classically results from stenosis of the subclavian artery proximal to the vertebral artery such that flow is retrograde in the ipsilateral vertebral artery. Subclavian steal syndrome presents clinically as upper-extremity claudication with associated vertigo, ataxia, and visual disturbances suggestive of vertebrobasilar ischemia.

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Other causes of vertebral artery steal are rare. In the literature, patients with hemodialysis fistulas have been reported to develop ipsilateral vertebral artery steal (3). We report a case of vertebral artery steal syndrome from a high-flow arteriovenous (AV) fistula between the right internal mammary artery (RIMA) and the superior vena cava (SVC) that precipitated symptoms of vertebrobasilar insufficiency. The steal syndrome was successfully treated with use of endovascular techniques.

## CASE REPORT

This case report did not require review by our center’s institutional review board. An 18-year-old right-handed man presented in April 2003 with a 7-month history of episodic loss of balance that acutely progressed to a constant feeling of disorientation and severe, unsteady gait. Of note, the patient had a history of Wolff-Parkinson-White syndrome that was treated in 1991 with two cardiac ablations. Approximately once per month, begin-

ning in September 2002, he experienced an unsteadiness and imbalance that required him to catch himself. In April 2003, his condition precipitously worsened, resulting in a fall at home. He reported nausea and several episodes of emesis. He also reported feeling confused and disoriented and increasing lightheadedness, particularly when standing. His symptoms were noticeably worsened when his position changed from lying or sitting to standing. The patient noted difficulty holding a pen and writing with his right hand. He denied any history of head injuries, including distant trauma or loss of consciousness with his recent fall.

The patient underwent magnetic resonance (MR) imaging of the brain and brainstem to evaluate a possible cerebellar lesion. The MR imaging findings were normal. A cerebral angiogram including imaging of the aortic arch, both common carotid arteries, and both vertebral arteries was then obtained to evaluate for possible vertebral artery dissection. Examination of the carotid and vertebral arteries



**Figure 1.** Flush aortogram in the right anterior oblique projection reveals tortuosity and dilation of the right subclavian artery proximal to and extending to the origin of the vertebral artery.

(extracranial and intracranial) had completely normal findings. However, angiography of the right subclavian artery revealed a high-flow AV fistula from the RIMA to the SVC (Figs 1,2). There was a subtle irregularity of the subclavian artery at the origin of the vertebral artery, which was thought to be caused by long-term increased turbulent flow. Of interest, flow was noted to be antegrade through the vertebral artery during the study. It was believed that his RIMA-to-SVC fistula was iatrogenic in nature and likely resulted from venous access attempts during earlier cardiac ablations for Wolff-Parkinson-White syndrome.

A 5-F H1 catheter (Cook, Bloomington, IN) was used to selectively catheterize the right subclavian artery. With use of the same 5-F H1 catheter, the RIMA was selectively catheterized. Angiography demonstrated a 1-cm

AV fistula between the RIMA and SVC located approximately 3–4 cm distal to its origin from the subclavian artery. Multiple 3-mm Gianturco coils (Cook) were placed in the RIMA beyond the AV fistula. Multiple 5-mm Gianturco coils and one 8-mm Gianturco coil were then deployed along the ostium of the AV fistula and proximal to the fistula. Postembolization digital subtraction arteriograms demonstrated successful embolization of the AV fistula (Fig 3).

Immediately after embolization, the patient had complete resolution of his symptoms, including ataxia and disorientation. The patient was asymptomatic with normal findings on a neurologic examination at 1-month follow-up, and he continues to remain without symptoms 2 years after embolization.

## DISCUSSION

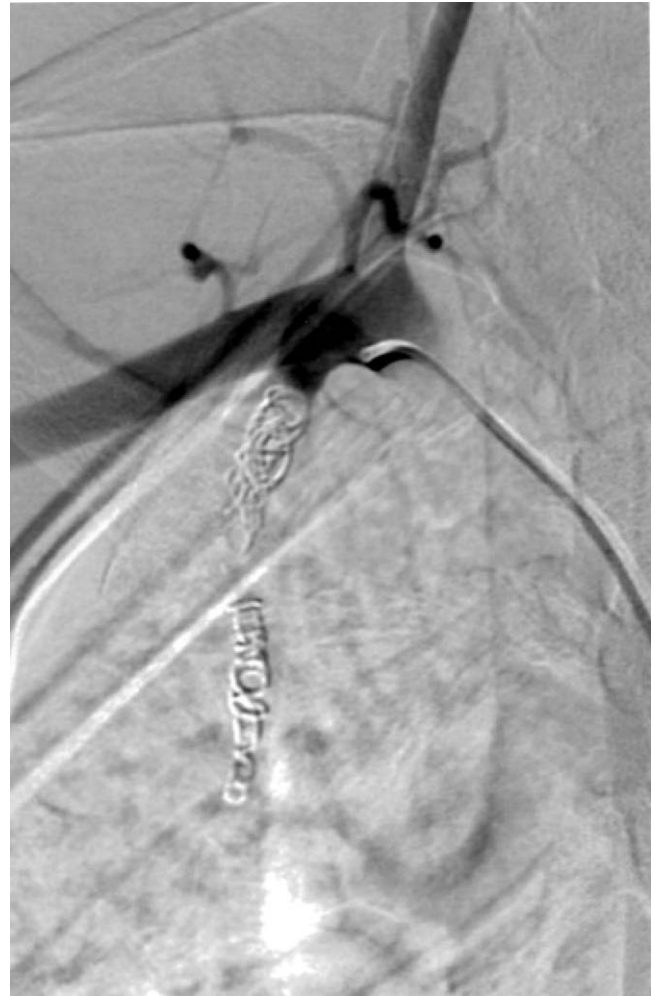
Embolism, atherosclerosis, and arterial dissection are the most common causes of vertebrobasilar artery disease (4). Steal syndromes are an infrequent cause of vertebrobasilar insufficiency. We describe a patient with a RIMA-to-SVC fistula causing vertebral artery steal syndrome with the unusual finding of antegrade flow through his vertebral artery and clinically significant vertebrobasilar insufficiency. The patient presented with classic vertebrobasilar ischemic symptoms of gait and limb ataxia, as well as episodes of emesis. Vertebral artery insufficiency commonly manifests as a sudden loss of balance, with ataxic events occurring when there is a reduction in blood flow or blood pressure, which happens with standing (4). Our patient also described increased unsteadiness with standing.

Our patient had an AV fistula between the RIMA and SVC, with flow diverted from the right vertebral artery to the low-resistance SVC. Although retrograde flow in the vertebral artery is classically seen in steal syndromes, our patient's angiogram demonstrated antegrade flow through the right vertebral artery. Antegrade flow in the vertebral artery has been reported in six cases with angiographically proven subclavian steal syndrome (5), which underscores the concept that it is the volume of blood the brain receives and not the direction of flow that controls symptoms. In the six cases of subclavian steal syndrome with antegrade flow, factors influencing symptoms were largely related to extensive collateralization and concomitant carotid atherosclerotic disease (5). Although no collateral vessels were observed and intra- and extracranial vessels were normal except for the high-flow AV fistula between the RIMA and SVC, our patient's clinical presentation was pathognomonic of vertebrobasilar insufficiency and subclavian steal syndrome.

One explanation for the angiographic findings of antegrade flow in the vertebral artery in the setting of a classic steal syndrome is that the right vertebral artery may have been able to maintain its intraluminal pressure while our patient was in a supine position. Therefore, the pressure in the right vertebral artery did not decrease



**Figure 2.** Flush aortogram in the left anterior oblique projection demonstrates the AV fistula (arrow) between the RIMA and SVC.



**Figure 3.** Completion embolization angiogram with multiple coils distal and proximal to the AV fistula and along the ostium of the AV fistula demonstrates a lack of residual flow of contrast medium.

below the pressure in the left vertebral artery and flow was antegrade during angiography. However, with a positional change, the vessel walls may not have been able to sufficiently compensate such that flow may have become retrograde, accounting for the worsening of our patient's symptoms with standing. The angiogram also showed irregularity and widening of the right subclavian artery at the origin of the right vertebral artery, suggesting an area of turbulent flow. This may have contributed to the decreased perfusion of the posterior fossa.

The etiology of steal syndrome in this patient was likely multifactorial. Decreased inflow coupled with inadequate collateralization and decreased

perfusion pressure caused by the AV fistula likely contributed to his symptomatic vertebral artery steal syndrome. Resolution of the patient's symptoms after embolization of his AV fistula confirms that the AV fistula was the source of symptoms. Also, the patient's classic presentation of symptomatic vertebrobasilar insufficiency with a high-flow AV fistula without any other radiographic or angiographic abnormalities further confirms the diagnosis of a vertebral artery steal syndrome in this patient.

We postulate that this patient's RIMA-to-SVC fistula was associated with the cardiac catheterizations he had undergone earlier. In ablation therapy for Wolff-Parkinson-White

syndrome, the right heart is accessed through the femoral vein, subclavian vein, or internal jugular vein (6). During this patient's cardiac catheterization, it is likely that the fistula was created unknowingly from the SVC to the RIMA, as cardiac catheterizations are often performed with use of a right-sided subclavian approach. The patient had no other history of blunt or penetrating trauma to his chest. The patient was asymptomatic for more than 10 years as his fistula increased in size sufficiently that symptoms developed.

To our knowledge, antegrade flow in the vertebral artery with vertebral artery steal is uncommon, and this cause of vertebral artery steal has not been de-

scribed in the literature. In young patients with posterior fossa symptoms and a history of procedures in which the central veins are used for access, an AV fistula causing vertebrobasilar insufficiency should be considered.

#### References

1. Taylor CL, Selman WR, Ratcheson RA. Steal affecting the central nervous system. *Neurosurgery* 2002; 50:679–689.
2. White JG, Kim A, Josephs LG, et al. The hemodynamics of steal syndrome and its treatment. *Ann Vasc Surg* 1999; 13:308–312.
3. DeCaprio JD, Valentine RJ, Kakish HB, et al. Steal syndrome complicating hemodialysis access. *Cardiovasc Surg* 1997; 5:648–653.
4. Savitz SI, Caplan LR. Vertebrobasilar disease. *N Engl J Med* 2005; 352:2618–2626.
5. Mirfakhraee M, Benzel EC, Guinto FC, et al. Unusual subclavian steal: bidirectional and antegrade blood flow in the vertebral artery. *Neuroradiology* 1995; 37:503–511.
6. Morady F. Radio-frequency ablation as treatment for cardiac arrhythmias. *N Engl J Med* 2005; 340:534–544.