Subclavian Steal Syndrome

Brian J. Potter, MD, MSc; Duane S. Pinto, MD, MPH

Case Presentation
A 90-year-old woman who underwent coronary artery bypass graft surgery 15 years ago was brought to the Emergency Department from her assisted living facility after developing chest pain, dyspnea, and diaphoresis. On arrival, she continued to have chest pain and exhibited signs of congestive heart failure. Her ECG revealed 2-mm ST-segment depressions across the precordium associated with 2-mm ST-elevations in aVR (Figure 1). Given this clinical picture, she was urgently brought to the cardiac catheterization laboratory, where angiography revealed totally occluded native coronaries and an occluded saphenous vein graft to an obtuse marginal artery. The saphenous vein graft to the right coronary circulation was patent as was the left internal mammary artery (IMA) bypass to the left anterior descending artery. However, there was a severe (99%) left subclavian artery stenosis (Movie I in the online-only Data Supplement). Stenting of the left subclavian artery resulted in the resolution of the patient’s chest pain and electrocardiographic abnormalities. She was discharged to home 9 days later.

Overview
“Subclavian steal” refers to a phenomenon of flow reversal in a branch of the subclavian artery that is the result of an ipsilateral hemodynamically significant lesion of the proximal subclavian artery.1,2 Subclavian stenoses, however, are most often asymptomatic and therefore do not require specific therapy other than that directed at the underlying etiology. “Subclavian steal syndrome” can become manifest in some patients with symptoms of arterial insufficiency afflicting the brain,1–3 the upper extremity,2 or even the heart if part of the coronary circulation is supplied via an IMA graft,4 as was the case in this patient.

Pathophysiology of Subclavian Steal
A subclavian steal syndrome may occur when a significant stenosis in the subclavian artery compromises distal perfusion to the IMA, vertebral artery, or axillary artery. As the degree of subclavian stenosis progresses, the pressure distal to the stenosis will eventually fall below the pressure transmitted by the contralateral (noncompromised) vertebral artery via the basilar artery or by the carotid artery through the Circle of Willis and basilar artery (Figure 2). The result is a pressure gradient favoring reversed blood flow (retrograde flow) in the vertebral artery distal and ipsilateral to the subclavian stenosis.1

Atherosclerosis is the most common cause of subclavian stenosis and, thus, steal syndromes, irrespective of the clinical manifestation.1,2,5,6 However, large artery vasculitis, thoracic outlet syndrome, and stenosis after surgical repair of aortic coarctation or tetralogy of Fallot (with a Blalock-Taussig shunt) are other possible causes. Congenital abnormalities, such as a right-sided aortic arch with an isolated left subclavian artery, can also lead to subclavian narrowing and steal syndromes and should be considered, particularly if a steal syndrome develops in a younger patient.

When the proximal subclavian stenosis is at least moderate (>50%), >90% of patients will have either intermittent or continuous flow reversal in the vertebral artery,7 though not all will be symptomatic. Duplex ultrasonography and transcranial Doppler appears more sensitive than conventional angiography for detecting flow reversal. In the majority of patients, a subclavian
stenosis with or without flow reversal in the ipsilateral vertebral artery is asymptomatic. However, patients with flow reversal can become symptomatic if collateral blood supply from the vertebrobasilar circulation cannot accommodate increased demand, such as during exercise or in the setting of an arteriovenous fistula.

In the minority of patients who manifest symptoms related to subclavian stenosis, arm claudication is the most common complaint, consisting of exercise-induced arm pain or fatigue. Occasionally, coolness or paresthesias in the extremity may be noted at rest or with exertion. Unilateral reversal of vertebral flow may cause vertebrobasilar transient ischemic attacks in rare circumstances. Upper extremity exercise, by reducing arterial resistance, increases blood flow to the arm and can precipitate lateralizing symptoms of vertebrobasilar insufficiency among persons without sufficient collateral flow.1,3 Similarly, a steal phenomenon may also occur in dialysis patients with an ipsilateral arteriovenous fistula. It should also be noted that bilateral vertebral flow reversal has been associated with nonlateralizing cerebral ischemia.

Vertebrobasilar insufficiency presents classically as “drop attacks,” but may also manifest as dizziness, diplopia, nystagmus, tinnitus, or even hearing loss.1,3 However, even when flow reversal in the vertebral system is observed, ischemic neurological events may be attributable to other processes, such as thromboembolism or embolization of atherosclerotic material from proximal subclavian disease, and is not necessarily related to a steal phenomenon.

Finally, a coronary-subclavian steal phenomenon4 may occur in patients who have undergone coronary artery bypass graft if a stenosis occurs in the subclavian artery proximal to the take-off of an IMA graft utilized to perfuse the heart. If arterial flow demand is increased in another vascular bed, as with ipsilateral upper extremity exercise, a share of the coronary circulation may be “stolen”, leading to angina or even infarction.

Diagnosis

Subclavian stenosis should be suspected in any patient with vertebrobasilar territory neurological symptoms, arm claudication, or coronary ischemia where the IMA has been used for coronary artery bypass graft surgery. Physical examination findings suggestive of subclavian stenosis include a discrepancy of >15 mmHg in blood pressure readings taken in both upper extremities, delayed or decreased amplitude pulses in the affected side, and a bruit in the supraclavicular fossa.2,8–10 A bruit in the suboccipital area may also be heard. The skin and nails of the affected side should be examined to rule out atrophic changes attributable to arterial insufficiency. A variety of noninvasive imaging modalities can be selectively used to diagnose subclavian stenosis when a steal phenomenon is suspected. Continuous wave Doppler and duplex ultrasonography are readily accessible, inexpensive, and accurate when performed by an experienced operator.12 Transcranial Doppler may be more useful in the setting of neurological symptoms. Magnetic resonance angiography and computed tomography angiography are also alternatives, but are perhaps best used to quantify the degree of subclavian artery stenosis when
Doppler techniques are inconclusive, if the etiology of subclavian stenosis is uncertain, or for planning interventions. Magnetic resonance angiography offers comparable resolution to computed tomography angiography, but, for reasons of cost and availability, is used most commonly when computed tomography angiography is contraindicated or indeterminate.

**Management**

Even asymptomatic subclavian artery stenosis is associated with increased risk of morbidity and mortality related to underlying atherosclerotic disease burden in other vascular beds. The presence of subclavian stenosis is associated with increased total mortality (hazard ratio, 1.40) and cardiovascular disease mortality (hazard ratio, 1.57) and with an increased risk of cerebrovascular ischemic events related to progressive carotid stenosis and compromised collateral pathways. Thus, subclavian stenosis is a marker of cardiovascular risk and identifies a population that will benefit from aggressive secondary prevention. Medical therapy that includes aspirin, β-blockade, angiotensin converting enzyme inhibition, and a statin reduces long-term mortality in lower extremity peripheral artery disease. Such a regimen is recommended for patients with subclavian stenosis.

Incidental subclavian stenosis, in the absence of symptoms, rarely requires revascularization therapy, even if flow reversal is demonstrated. The lone exception is among patients in whom coronary artery bypass graft with an ipsilateral IMA graft is planned. Here, pre-emptive treatment of the subclavian stenosis is recommended.

For patients with mild symptoms, medical therapy and observation are appropriate, because symptomatic improvement without intervention has been described.

Patients with burdensome symptoms and proximal subclavian artery occlusive disease can be successfully treated either surgically or percutaneously. Balloon angioplasty and stenting can be performed when stenting is unlikely to compromise the vertebral circulation. Technical success of the percutaneous approach can be achieved in >90%, with 5-year patency rates of 85%. Continuous flow reversal appears to be a marker for a higher risk of restenosis compared with those with intermittent flow reversal at baseline. Ultimately, up to 10% of patients will have symptomatic restenosis, 95% of whom can be managed by repeat endovascular therapy.

Longer or more distal occlusions may be better addressed surgically. Surgical revascularization consists of bypass in the form of carotid-subclavian bypass, carotid transposition, or axillo-axillary bypass surgeries that, overall, have patency rates in excess of 70% at 5 years. However, the success rate is >80% if the common carotid is used in the bypass. Axillo-axillary bypass, by contrast, has significantly lower patency rates (46%) and is therefore typically reserved for patients with elevated perioperative risk. Patients in whom central nervous symptoms predominate should have carotid stenoses addressed before any attempt at surgical revascularization of the subclavian system. Neurological symptoms abate after re-establishment of normal carotid anterograde flow, even in the setting of significant subclavian lesions. No prospective randomized comparisons of percutaneous and surgical revascularization for subclavian stenoses exist.

Patients with both prohibitive surgical risk and unfavorable anatomy for percutaneous treatment can be managed with antiplatelet therapy and general cardiovascular prevention strategies.

**Summary**

“Subclavian steal” refers to a syndrome of symptoms relating to arterial

![Figure 2. Schematic drawing of the circulation involved in subclavian steal phenomena and the flow adaptation in response to a proximal subclavian stenosis.](http://circ.ahajournals.org/doi/abs/10.1161/CIRCULATON.114.008865)
insufficiency in a branch of the subclavian artery stemming from flow reversal, attributable to occlusive disease in the subclavian artery proximal to that branch that is usually atherosclerotic in cause. Most patients are asymptomatic, but patients with IMA bypass grafts may manifest angina elicited by exercise of the upper extremity ipsilateral to the graft and stenosis (so-called “coronary-subclavian steal”). Subclavian steal may also manifest as verteobasilar insufficiency or, most commonly, arm claudication. Subclavian steal should be considered among patients exhibiting suggestive symptoms. A meticulous examination of segmental pulses and pressures, as well as judicious use of duplex ultrasonography, magnetic resonance angiography, computed tomography angiography, or conventional angiography, or conventional angiography can confirm the presence of subclavian stenosis. Symptomatic patients benefit from either percutaneous or surgical revascularization, depending on both anatomic and patient factors. Subclavian stenosis, regardless of symptoms, is a marker of atherosclerotic disease and increased risk for cardiovascular and cerebrovascular events.

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Disclosures
None.

References

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