There's a Thief in the House

IMAGE OF THE MONTH

Joanne Thanavaro, DNP

CASE PRESENTATION

A 51-year-old woman presented to her primary care provider with a 1-year history of dizziness that was not positional in nature. She had an episode of syncope for a few minutes 8 months ago, and Holter monitoring at that time was unremarkable. The patient denied any undue stress before her syncope event and had no other warning symptoms. She denied chest pain, dyspnea, and palpitations. Her symptoms were attributed to vertigo, and she was referred to an otolaryngologist for further evaluation and treatment. Her dizziness was tolerable but was never completely resolved, and 3 months ago it began to get worse with increased frequency. After she suffered another episode of syncope, a 30-day event recorder was obtained, and no arrhythmias were detected. She was subsequently referred to a cardiologist for further evaluation. She reported having no pulses in her left arm for years, and her blood pressure could not be elicited in that arm. Her dizziness and syncope were not associated with left arm activity, but she did report left arm fatigue and dropping things from her left hand in the past. Her past medical history included dyslipidemia, chronic obstructive pulmonary disease, gastroesophageal reflux, headache, and bipolar disorder. She underwent multiple surgeries for her back and cervical disc disease; she constantly required pain management for chronic pain syndrome. She was unable to exercise or work because of back and neck pain. She was on multiple medications for her complex medical problems. She had a 20 pack year smoking history and denied alcohol use.

PHYSICAL ASSESSMENT

Vital signs were as follows: blood pressure of 100/70 mm Hg using the right arm, heart rate of 64 beats/min, respirations of 16 breaths/min, and temperature of 98.4°F. Blood pressure could not be obtained from her left arm. The patient was alert and oriented with good mental function. Examination of the central nervous system, skin, head, ears, eyes, nose, throat, and thyroid was normal. She had no neck vein distention or hepatojugular reflux, and her carotid pulse was good and equal bilaterally without carotid bruits. The cardiac examination showed normal apical impulses with a normal first and second heart sound and a grade 2/6 systolic murmur. The lungs were clear to percussion and auscultation with good breath sounds. The abdomen was soft without tenderness, mass, hepatomegaly, or abdominal bruits. The lower extremities were without edema, and distal pulses in the lower extremities were good and equal bilaterally; the left brachial and radial pulses were absent.

DIAGNOSTICS

Laboratory Values

The complete blood count, comprehensive metabolic panel, lipid profile, and thyroid levels were normal.

Electrocardiogram

An electrocardiogram showed normal sinus rhythm with a normal PR interval of 148 milliseconds and a QTc of 442 milliseconds without a delta wave.

Echocardiogram

A 2-dimensional echocardiogram showed normal left ventricular systolic function with mild mitral and tricuspid regurgitation and without inflow/outflow tract obstruction or pulmonary hypertension.

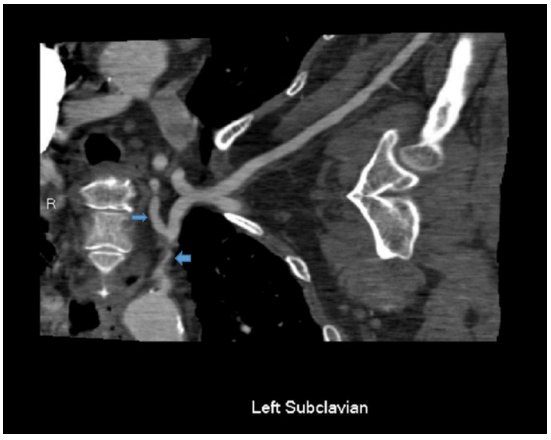
Imaging

Computed tomographic angiography of the neck with contrast revealed patent carotid and vertebral arteries bilaterally without obstruction. The right subclavian artery was unremarkable, and there was a high-grade nearly occlusive left subclavian artery that extended to the origin of the left vertebral artery (Figure 1). The rest of the left subclavian artery was unremarkable.

ETIOLOGY OF THE LEFT SUBCLAVIAN STEAL

The term subclavian steal refers to the phenomenon of flow reversal in the vertebral artery ipsilateral to a hemodynamically significant stenosis or occlusion of the subclavian artery. Frequently, subclavian steal is asymptomatic and does not require invasive evaluation or treatment; it is an appropriate physiological response to proximal arterial disease. The etiology of left subclavian stenosis includes atherosclerosis, Takayasu and giant cell arteritis, external compression, aortic dissection, and congenital anomalies of the brachiocephalic trunk.[1] The underlying risk factor leading to subclavian occlusion or stenosis is atherosclerotic arterial disease, which has a preponderance for the left side. The prevalence of subclavian stenosis varies between 3% and 18% depending on the population study.[1] Fields and Lemak[2] found that 17% of the 6,534 patients admitted to the Joint Study of Extracranial Arterial Occlusion had arteriographic evidence of subclavian or innominate stenosis greater than 30% or occlusion; only 168 patients had symptoms of subclavian steal syndrome.

Figure 1. A computed tomographic angiogram showing a high-grade nearly occlusive left subclavian artery (thick blue arrow) extending to the origin of the left vertebral artery (thin blue arrow).



DIAGNOSIS

Clinical Presentation

Most patients with subclavian artery stenosis are asymptomatic. When symptoms do occur, they are a result of ipsilateral upper extremity ischemia. Exercise-induced arm pain, fatigue, coolness, paresthesias, or numbness may occur. A significant blood pressure difference between the arms, greater than 40 mm Hg, is more commonly associated with symptoms requiring intervention.

Some patients may present with subclavian steal syndrome in which significant symptoms develop as a result of blood steal from the posterior circulation leading to arterial insufficiency of the brain. Subclavian artery occlusion or hemodynamically significant stenosis proximal to the origin of the vertebral artery results in lower pressure in the distal subclavian artery; blood flows from the contralateral vertebral artery to the basilar artery and may flow in a retrograde direction down the ipsilateral vertebral artery and away from the brainstem. Reversed vertebral artery flow is an important collateral circulation to the arm, but it may cause deleterious neurologic consequences as shown in this patient. Symptoms and signs of vertebrobasilar ischemia may include dizziness, vertigo, ataxia, disequilibrium, diplopia, bilateral visual blurring, and syncope.[3] Left subclavian coronary steal syndrome may precipitate angina pectoris in which the left internal mammary artery (LIMA) is used as a conduit for coronary artery bypass grafting. If proximal left subclavian artery disease develops or progresses in the setting of a previous LIMA graft coronary bypass, the LIMA graft may become dependent on retrograde vertebral flow; exercise of the