CLINICAL VIGNETTE

Subclavian Steal: An Overlooked Cause of Syncope

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Case Presentation

A 76-year-old male presented to the emergency department (ED) after an unwitnessed syncopal event. He described walking outside of his house during the day, feeling hot and flushed and then passed out falling backwards hitting the back of his head. Patient denied any other preceding symptoms such as vision changes, dizziness, chest pain, shortness of breath or palpitations. He had no prior history of syncope or seizure and denied tongue biting or bowel and bladder incontinence during the incident. He also denied any associated symptoms such as weakness in extremities, confusion, slurred speech or numbness and tingling.

This patient had a significant cardiac medical history including atrial fibrillation and atrial flutter on rivaroxaban and prior atrial septal defect (ASD) repair. His risk factors for atherosclerotic disease included diabetes and hyperlipidemia.

In the ED, he was afebrile, heart rate 60, blood pressure (BP) 112/56 and oxygen saturation 96% on room air. Left arm BP was 126/70 and right arm BP was 109/91.

An electrocardiogram and showed normal sinus rhythm without any ischemic changes, arrhythmia or heart block. Troponin was negative and labs were otherwise unremarkable. Orthostatic vitals were negative. CT head was preliminarily concerning for infarction although the final read showed no acute intracranial finding. Due to this initially concerning finding, neurology recommended MRI brain and CT angiogram head and neck to evaluate further.

On physical exam, a three cm laceration with staple placement was present on the posterior scalp. No carotid bruits were heard on exam. Cardiopulmonary exam was unremarkable, his heart was regular rate and rhythm without any murmurs. Neurological exam was unremarkable without any focal neurologic deficits.

The team was most concerned for cardiac syncope given the patient's extensive cardiac history. Transthoracic echo revealed normal ejection fraction and no residual ASD. MRI brain showed no acute intracranial infarct. CT angiogram head and neck showed no significant intracranial stenosis but incidentally did show high-grade stenosis of the proximal left subclavian artery. Vascular surgery was consulted and carotid ultrasound showed left subclavian steal with retrograde flow through the left vertebral artery. No hemodynamically significant carotid artery stenosis was noted.

Vascular surgery performed angiogram and stenting of the left vertebral artery without any complications or further syncopal events.

Discussion

Syncope is a common problem that affects 30-50% of all adults. The incidence of syncope increases with age due to decrease in cerebral blood flow.[1] As there is a recurrence rate of 35% for syncope, as well as physical injury in 29% of patients, it is important to try to identify and reduce the rate of recurrence by diagnosing and treating the underlying cause of syncope. Beyond morbidity, the economic costs are also significant as syncope accounts for 3-5% of all emergency visits with an average hospital stay of 5.5 days, leading to healthcare costs of over 2.4 billion annually in the United States.[2]

In this patient, subclavian steal syndrome was incidentally found on imaging but should generally be a consideration as a cause for syncope, especially when other cardiac tests are negative in patients with significant risk factors for atherosclerosis.

Subclavian artery stenosis is usually asymptomatic in patients with focal stenosis due to vessel collateralization. The incidence of subclavian stenosis can be 3-4% in the general population and as high as 11-18% in patients with peripheral artery disease.[3] It is associated with smoking, hypertension and diabetes and is usually caused by atherosclerosis.[4,5]

Patients usually become symptomatic once atherosclerosis affects other aortic arch vessels, such as the carotid or vertebral arteries. The left subclavian artery is four times more likely to be affected than the right or innominate arteries.[3] Subclavian steal syndrome occurs when retrograde flow occurs in the vertebral artery to compensate for increased demand of the upper extremity.[5] Common symptoms can include arm claudication or fatigue, as well as vertebrobasilar insufficiency symptoms such as visual disturbances, ataxia, vertigo, dysarthria, and, such as in this case, syncope.[3]

In terms of physical examination, an inter-arm systolic blood pressure difference of 10-20 mm Hg has high specificity of at least 85% and lower sensitivity ranging from 35-65% for subclavian stenosis.[6] Other physical assessment includes presence of bruits (carotid, cervical, supraclavicular), as well as less common findings such as finger ulcers or necrosis.[5]

Diagnosis of subclavian artery disease can be done non-invasively with duplex ultrasound with color flow imaging. Dampened waveforms, turbulent color flow imaging, and increased velocity in areas of stenosis are common findings of obstruction. Reversal of the ipsilateral vertebral artery flow, as shown in this patient's ultrasound, is characteristic of sub-clavian steal syndrome. Otherwise, invasive angiography is the definitive test.[5]

Conservative medical management is indicated in the absence of symptoms, which includes aggressive management with high-intensity statins, antiplatelet agents, and antihypertensive agents.[5] Treatment is indicated for symptomatic patients, including ipsilateral claudication or arm fatigue, ischemic symptoms, or subclavian steal. The preferred treatment is endovascular stenting, with technical success of 98%, low complication rates of 5.6% with 0.6% stroke risk, short healing times, and long-term patency of stent of 85-90% at 3 years.[3]

Subclavian steal syndrome is an often overlooked cause of syncope, especially in a patient with multiple comorbidities including a history of cardiac disease. Cardiac syncope has a one-year mortality of 20-30%; requires prompt diagnosis and treatment.[1] As in this case, cardiac etiologies may be the focus of the evaluation leading to less focus on noncardiac causes of syncope. Checking inter-arm blood pressures, listening for bruits, and getting a carotid ultrasound duplex are all tests that should be considered, especially in those with risk factors for atherosclerosis. Prompt diagnosis and intervention on syncope can decrease morbidity and mortality and help reduce overall health care costs.

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