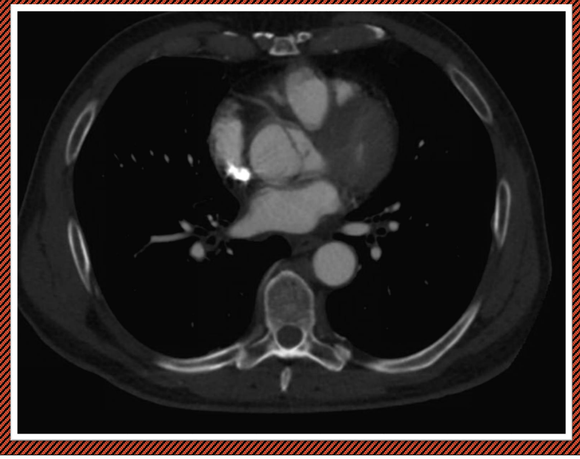
Dissecting the workup for Syncope

Padilla MA, Piro K.
Oregon Health and Science University



Syncope

Syncope is defined as a transient decrease in cerebral perfusion causing loss of consciousness that resolves quickly and without intervention.

Three general categories of etiology:

Reflex related	Hypotension related	Cardiac related
- Vasovagal	- Hypovolemia	- Bradycardia
- Situational	- Peripheral vasodilation	- Tachycardia
-Carotid sinus disease	- Drugs, toxins	- Cardiac structural
	- Primary autonomic failure	- Pulmonary embolism
	- Secondary autonomic failure	- Pulmonary hypertension
		- Aortic dissection

The combined diagnostic yield of history, physical examination, and electrocardiogram is estimated to be 50%

For unexplained syncope, it is helpful to use a risk stratifying method to determine disposition and workup.

Low risk: for instance, no evidence of heart disease, not elderly, and first episode of syncope → no work up necessary

Moderate risk: for instance, elderly, first or multiple episodes, hemodynamically stable →work up for heart disease (TTE, stress test etc), not necessarily as an inpatient

High risk: for instance, elderly, multiple episodes, evidence of heart disease, family history of sudden cardiac death, neurological signs and symptoms, trauma → admit to the hospital for thorough evaluation and monitoring

Syncope pearl

For witnessed syncope with no evidence of neurological dysfunction and no trauma, do not order carotid artery imaging or brain imaging

Introduction

Syncope is a common problem with an estimated lifetime prevalence of 40% in the general population (1). It is a problem that easily leads to hospital admission, with estimates that 1/3 of ER visits for syncope result in admission. The differential is broad and ranges from utterly benign conditions to surgical emergencies. The work up can be costly, with estimates of $5400 per admission in 2000(2). The cost may be a reflection of the litany of tests ordered for evaluation, ranging from head CT to carotid ultrasound. The Choosing Wisely ® Campaign recommends not ordering brain imaging or carotid artery imaging for syncope without evidence of neurological dysfunction because they have been shown to be of low diagnostic yield.

Case

Mr S is a 69 year old man with a history of hypertension and current smoker who experienced a witnessed episode of syncope preceded only by acute chest pain. His wife witnessed the event and confirmed total LOC for minutes and no head trauma. He denied any prior history of chest pain or syncope. He currently experienced what he called "chest tightness" in his central chest which radiated to his back. He did not complain of any weakness, numbness, vision changes, headache, incontinence, abnormal movements, incontinence, or tongue biting.

Initial vital signs included BP of 136/78 in the Left arm and 94/61 in the Right arm with concurrent decreased Right radial pulse. Initial labs were within normal limits, and initial ECG was unrevealing. Imaging included a normal head CT, and a bilateral carotid duplex study which was informally read as Right subclavian occlusion. CT dissection protocol ordered but not yet performed prior to sign out.

Before sign out was accepted, the receiving medicine team requested that the CT dissection protocol be completed. It revealed a type A thoracic aortic dissection involving the right carotid artery and brachiocephalic artery. The patient underwent an open repair that night and recovered well. After he had stabilized he was discharged home.

Discussion

Acute thoracic aortic dissection is a rare cause of syncope, with sensitivity estimates ranging from 2-14% (3) . However, it is a diagnosis which has dire consequences if missed. One study (4) suggested that this diagnosis is missed in the initial 24 hours of assessment 39% of the time. Interestingly, there is no pathophysiological basis for carotid stenosis and syncope. This is the basis of recommending against carotid imaging for syncope, provided there are no neurological deficits. The unequal pulses and blood pressure were explained by possible subclavian stenosis. Had the Choosing Wisely ® recommendations been followed, the carotid doppler would not have been ordered and possibly would not have contributed to the initial anchoring bias.

References

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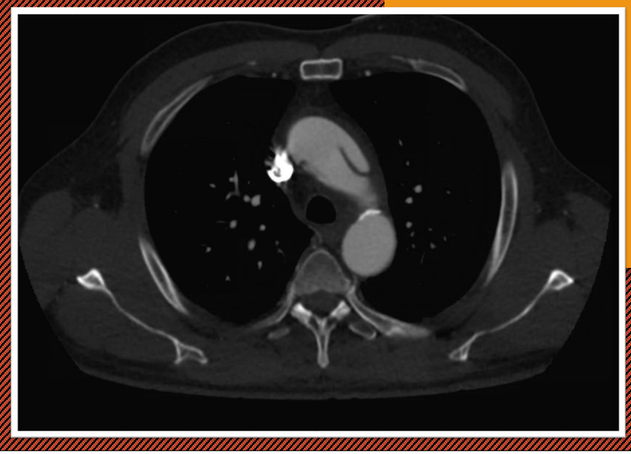

Acute thoracic aortic dissection

Intimal tear in the wall of the aorta allows blood to flow past the tunica intima into the tunica media, causing a false lumen. High pressure from cardiac contraction can cause the tear to extend, even to the point where the false lumen occludes true vessels such as the subclavian or coronaries. Proximal dissections are surgical emergencies with a high mortality rate without surgical intervention. Distal dissections are typically medically managed initially by double product control.

Risk Factors

- Hypertension
- Connective tissue disorders
- Aortic root dilation (can be seen in Turners syndrome)
- Chest trauma

Diagnosis

Imaging is the sole way to diagnose a thoracic aortic dissection, including CT angiograms, transesophageal echocardiogram, MRI. Practically, CT is most commonly used.

No single physical exam finding or history has high diagnostic yield.

Highly suspicious history or exam findings for dissection:

- Sudden onset of chest pain
- Chest pain described as tearing/ripping
- Chest pain radiating to the back
- Pulse deficit (or BP difference)
- Focal neurological findings

Acute thoracic aortic dissection pearl

Rare diagnosis. Multiple exam and history findings found together make it more likely. Pulse deficits and focal neurological deficits are the most specific findings.