A Case of Recurrent Syncope

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PATIENT HISTORY

CHIEF COMPLAINT:

- 63 y/o Hispanic male presents after 3rd syncopal episode this week
- Episode duration: 15 seconds each
- Dizziness once a week x 4 months
- Pertinent negatives: chest pain, relation to exertion, headaches, vision changes, nausea, vomiting, hematochezia, weakness

PAST MEDICAL HISTORY:

- CVA and MI two years ago
- Hypertension and hyperlipidemia
- Medications: atorvastatin and aspirin
- Smoking: 1/2 pack a day x 50 years; quit 5 months ago
- Alcohol: 5 beers/week

EXAMINATION

- **Vitals:**
  - BP 152/82 mmHg (supine)
  - BP 132/72 mmHg (standing)
- **Cardiovascular:**
  - RRR without murmur or ectopy
  - Bruit noted over left carotid artery
- **Neuro:**
  - CN II-XII grossly intact
  - Sensation to light and sharp intact
- **Musculoskeletal:**
  - Full active ROM in all extremities
  - 5/5 muscle strength bilaterally

DIAGNOSTIC WORKUP

Carotid Ultrasound Results:

- Bilateral carotids: diffuse plaques
- Left internal carotid: 70-90% stenosis
- Bilateral external carotids: 50% stenosis

Electrocardiogram:

- Normal sinus rhythm without a prolonged QT interval

Laboratory:

- Chemistry panel: Negative
- CBC: Hgb: 14 g/100 ml; normal indices
- Stool guaiac: Negative

Imaging:

- Computerized Tomography with Angiography (CTA) revealed atherosclerotic disease with narrowing of bilateral internal carotid arteries by 71%

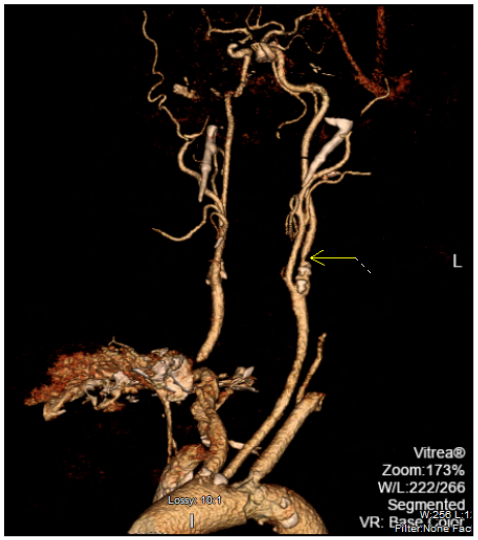

Figure 1: CTA 3-D rendering of bilateral carotid arteries

DIFFERENTIAL DIAGNOSIS

A. Cardiac arrhythmia

B. Iron deficiency anemia

C. Carotid stenosis

D. Transient ischemic attack (TIA)

OUTCOME:

The patient was referred to vascular surgery for a carotid endarterectomy (CEA).
Four weeks after surgery, the patient reported alleviation of dizzy spells and no subsequent syncopal episodes.

DIAGNOSTIC REASONING:

- Absence of arrhythmia or prolonged QT interval on EKG makes arrhythmia an unlikely cause of dizziness and syncope
- Iron deficiency was ruled out by a normal hemoglobin, MCV, and MCHC.
- Absence of neurological deficits and presence of symmetrical movements without weakness in face and extremities, makes a TIA unlikely.
- Left carotid bruit on physical exam prompted a carotid ultrasound that led to the diagnosis of carotid stenosis.

DISCUSSION

Carotid Stenosis

- Most common cause: atherosclerotic changes beginning in adolescence
- Accounts for 9% of ischemic strokes[1]
- Worsened with endothelial damage and plaque accumulation
- Indications for carotid duplex ultrasound: bruit, dizziness, syncope, or history of ischemic event
- If no TIA or stroke in past 6 months, stenosis is considered asymptomatic
- Management: antiplatelets, statins, anti-hypertensives, diabetes control, smoking cessation, and lifestyle modifications
- Immediate CEA with medication in asymptomatic patients with >60% stenosis decreases risk of ischemic stroke from10% to 5% over 10 years

REFERENCES

1. Dharmadhikari S, Chaturvedi S. Medical and revascularization therapies for asymptomatic carotid stenosis. *Curr Atheroscler Rep.* 2015;17(8):44.
2. Halliday A, Harrison M, Hayter E, et al. 10-year stroke prevention after successful carotid endarterectomy for asymptomatic stenosis (ACST-1): a multicentre randomized trial. *Lancet*. 2010;376:1074-84.
3. Brott TG, Howard G, Roubin GS, et al. Long-term results of stenting versus endarterectomy for carotid artery stenosis. *N Eng J Med.* 2016;374:1021-31.

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ABSTRACT

CASE
A 63-year-old Hispanic male presented with episodic dizziness and his third episode of syncope. The dizziness would appear suddenly, occurring about once a week for the past four months, and each episode lasted about 15 seconds. It sometimes occurred after standing quickly. He denied any exertion or chest pain associated with these episodes. After the syncopal episodes, the patient was alert without confusion, amnesia or deficits. He denied headache, vision changes, nausea, vomiting, bloody stools or weakness.
The patient had a history of stroke and myocardial infarction in 2014 and a traumatic subdural hematoma in 2012. He also had a history of hyperlipidemia, controlled hypertension, and chronic obstructive pulmonary disease, and no significant family history. He was currently taking aspirin, lisinopril, metoprolol, atorvastatin, nifedipine, and albuterol. The patient smoked half a pack a day for 50 years and quit 5 months ago. He admitted to drinking five beers a week and denied illicit drug use.

PHYSICAL EXAMINATION
The patient's blood pressure (supine) was 152/82 mm Hg. All other vital signs were unremarkable. Upon standing, his blood pressure dropped to 132/72 mm Hg with subjective lightheadedness. He had a regular cardiac rhythm without murmur or ectopy. Bruits were heard over the left carotid artery. Cranial nerves II-XII were grossly intact. Sensation to light and sharp touch were intact throughout. He had full active range of motion in bilateral upper and lower extremities with 5/5 strength. An electrocardiogram (EKG), complete blood count, stool guaiac, and carotid duplex ultrasound were ordered.

DIFFERENTIAL DIAGNOSIS
• Cardiac arrhythmia
• Iron deficiency anemia
• Carotid stenosis
• Transient ischemic attack (TIA)

DIAGNOSTIC RESULTS AND OUTCOME
The EKG revealed normal sinus rhythm without arrhythmia or prolonged QT interval, making arrhythmia less likely a cause of his syncope. The stool guaiac was negative and his hemoglobin was 14.0, ruling out anemia. The patient showed no indication of neurological deficits and had symmetrical movements without weakness in the face and extremities, deeming a TIA unlikely.
The carotid ultrasound reported diffuse plaques within bilateral carotids with 70-90% stenosis of the left internal carotid artery and 50% stenosis of bilateral external carotid arteries. The patient was diagnosed with carotid stenosis. A computerized tomography angiogram (CTA) with contrast was ordered (Figure 1) and the patient was referred to vascular surgery. The vascular surgeon recommended a carotid endarterectomy (CEA). Four weeks after surgery, the patient reported alleviation of his dizzy spells and no subsequent episodes of syncope.

DISCUSSION
Carotid stenosis is a narrowing of the lumen of the carotid artery, most commonly due to atherosclerotic changes beginning in adolescence and worsening as endothelial damage and plaques accumulate. According to several studies worldwide, the prevalence of carotid stenosis is about 4.4 to 7%, and it is considered a cause of 9% of all ischemic strokes.1 If a carotid bruit is auscultated, a carotid duplex ultrasound screening is often clinically done, especially if symptoms are suggestive of an ischemic event, or if the patient has a history of ischemic events and presents with dizziness or syncope. Despite these symptoms, patients with carotid stenosis are considered "asymptomatic" if they have not had a TIA or stroke in the past 6 months.
Medical management of carotid stenosis has improved significantly to address risk factors for vascular disease and includes antiplatelet therapy, statin therapy, anti-hypertensives, diabetes management, smoking cessation, and lifestyle modifications. Revascularization through CEA or stenting is another form of therapy, although it is more controversial in asymptomatic patients. Asymptomatic patients with 60% or more stenosis receiving immediate CEA in combination with medication, however, halved their ischemic stroke rate to 5% risk over the next 10 years.2 These benefits were greatly decreased if patients are over 75, or have less than 10 years life expectancy.
When considering revascularization, there was no significant difference between CEA and stenting in risk of stroke, myocardial infarction (MI) or death over 10 years. However, stents have slightly higher risks of perioperative stroke or death (4.4% vs. 2.3%), while CEA has a slightly higher risk of MI (2.3% vs. 1.1%).3 As a result, patients should be individually assessed for severity of stenosis and risk factors when considering a revascularization approach.

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1. Dharmadhikari S, Chaturvedi S. Medical and revascularization therapies for asymptomatic carotid stenosis. *Curr Atheroscler Rep*. 2015;17(8):44.
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