Recurrent syncope resulting from large carotid atheroma

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Introduction

Syncope is a common cause of hospitalization in the elderly. Carotid sinus syndrome (CSS) is an often overlooked cause of syncope. CSS occurs when stimulation of the carotid artery baroreceptors in the neck results in a greater than expected fall in heart rate and blood pressure, resulting in recurrent lightheadedness, syncope, or unexplained falls in older patients. In this case, a patient with a history of severe carotid stenosis presents with syncope and bradycardia.

Case report

A 59-year-old man presented to the ER with a concussion and nondisplaced scapular fracture after an unwitnessed syncopal event. Prior to the fall, he stood up, felt dizzy, fell backwards, and hit his head on the floor. He endorsed a brief (<3 seconds) episode of loss of consciousness after the fall. He noted at least 3 unexplained syncopal events in the past year—1 resulting in a car accident. On admission, he was orthostatic with a decrease in blood pressure from 122/52 to 95/48 and increase in heart rate from 54 beats per minute (bpm) to 59 bpm from supine to standing. The patient was also in sinus bradycardia with first-degree atrioventricular (AV) block with a heart rate of 50–63 bpm. Physical examination was notable for bilateral harsh carotid bruits. Pressure from left-sided carotid sinus auscultation reproduced symptoms of dizziness and resulted in a transient decrease in heart rate by more than 10 bpm.

Past medical history included severe bilateral carotid stenosis, severe mitral stenosis (mean mitral gradient 17.3 mm Hg at heart rate 80 bpm), hypertension, hyperlipidemia, type 2 diabetes, and chronic kidney disease stage III.

KEY TEACHING POINTS

- When evaluating a patient presenting with syncope, an assessment for peripheral arterial disease, carotid disease, and carotid intervention, as well as auscultation for carotid bruits, should be documented in the history and physical exam.
- It is important to recognize the presentation of carotid sinus syndrome (CSS) as a potential reversible etiology of syncope and atrioventricular block.
- CSS is a clinical diagnosis that is defined by reproduction of symptoms in response to carotid baroreceptor stimulation, whereas carotid sinus hypersensitivity (CSH) is a physical exam finding that is defined by sinus pause of more than 3 seconds or lowering of blood pressure greater than 50 mm Hg during carotid sinus massage.
- Significant carotid arterial disease and atheromas can increase pressure on carotid baroreceptors contributing to symptoms of CSS.
- It is important to make the distinction between CSS and CSH because CSH only requires treatment if it results in CSS.

Differential diagnosis included reflex-mediated syncope, orthostatic hypotension, transient ischemic attack, cerebrovascular accident, paroxysmal atrial fibrillation, paroxysmal AV block, sick sinus syndrome, and CSS.

Investigations

The patient underwent an electrocardiogram (Supplemental Figure A), which revealed a first-degree AV block with a PR interval = 328 ms.

Inpatient monitoring during the patient's hospitalization over 10 days identified multiple spontaneous symptomatic episodes of second-degree type 1 AV block (Supplemental

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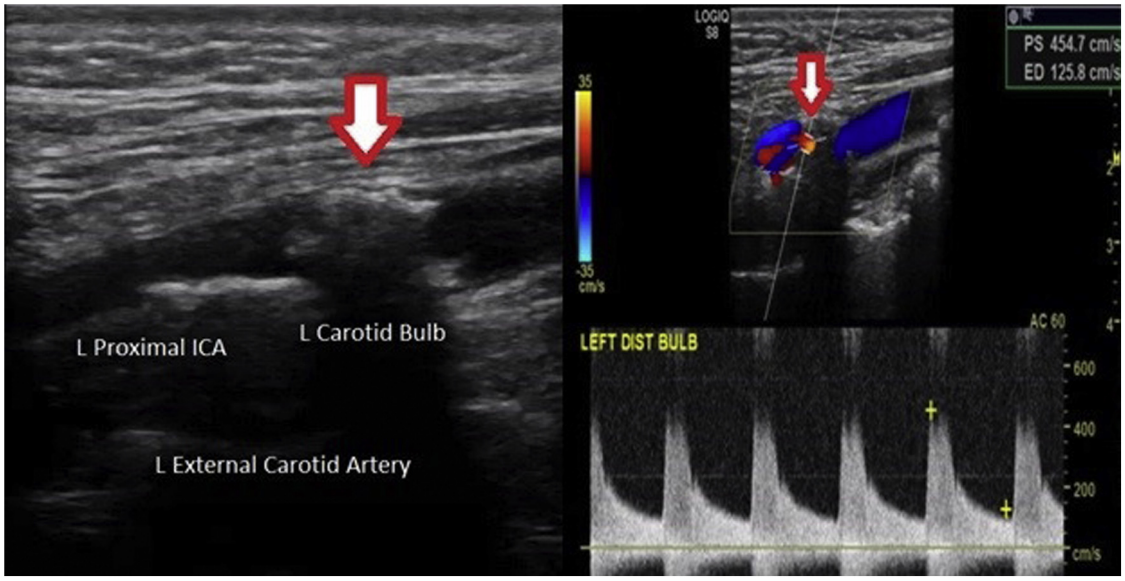

Figure 1 Carotid ultrasound and Doppler of the distal left common carotid artery. Carotid ultrasound identified a large calcification (*red arrow*) in the left bulb of the distal left common carotid artery. Carotid Doppler identified a high-velocity color jet with a peak systolic velocity of 454.7 cm/s, consistent with severe stenosis (>70%). ICA = internal carotid artery; L = left.

Figure B). These episodes and symptoms were reproducible with left carotid sinus massage (CSM) but did not occur with right carotid massage. Owing to the finding of bilateral carotid bruits, we obtained a carotid duplex ultrasound. There was an atheroma in the left carotid bulb (Figure 1) producing a high-velocity color flow jet with a peak systolic velocity of 454.7 cm/s (Figure 1), consistent with severe carotid stenosis (>70%). There was also an atheroma in the right carotid with a peak systolic velocity of 340 cm/s approaching 70% carotid stenosis. Computed tomography of the neck with intravenous contrast identified a complex left carotid stenosis (Figure 2).

Management

The patient underwent left carotid endarterectomy with removal of left carotid atheroma (Figure 3) and was subsequently started on aspirin 81 mg daily and clopidogrel 75 mg daily. He noted symptomatic improvement of his presyncope postoperatively; CSM post carotid endarterectomy failed to elicit syncope or a significant decrease in heart rate (>10 bpm) or blood pressure (>20/10 mm Hg).

Follow-up

The patient remained asymptomatic without syncope or dizziness postoperatively and was discharged on hospital day 10. An implantable loop recorder was placed prior to undergoing endarterectomy. PR interval after the carotid endarterectomy decreased from 328 ms on admission electrocardiogram to 292 ms on postoperative electrocardiogram (Supplemental Figure C). Loop recorder interrogation and remote monitoring over the course of 4 months failed to identify any episodes of AV block or sinus pauses with heart rate <40 bpm.

Discussion

When diagnosing CSS, it is important to make a distinction between CSS and carotid sinus hypersensitivity (CSH). CSH is a physical examination finding that is defined by ACC/AHA/HRS guidelines as a sinus pause of more than 3 seconds or lowering of blood pressure greater than 50 mm Hg during CSM.[1] In contrast, CSS is a clinical diagnosis that is defined by reproduction of symptoms, such as syncope or dizziness in response to carotid baroreceptor stimulation.[1] Therefore, CSH only requires treatment if it is causing

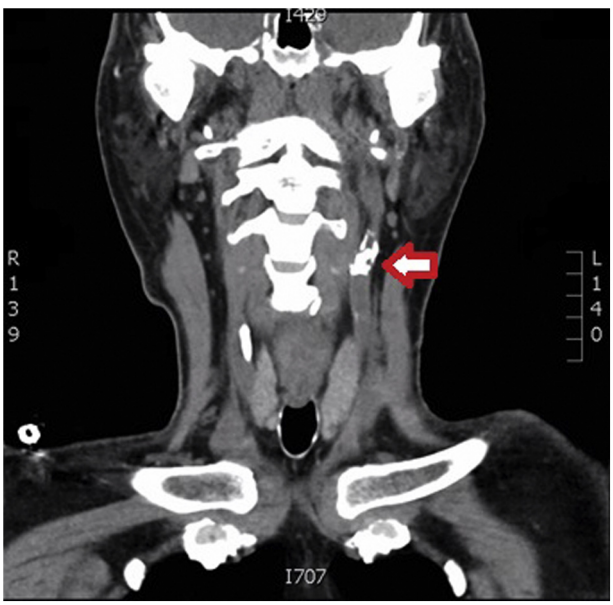

Figure 2 Computed tomography of the neck with intravenous contrast identified a complex calcified atherosclerotic plaque (*red arrow*) in the left common carotid artery.

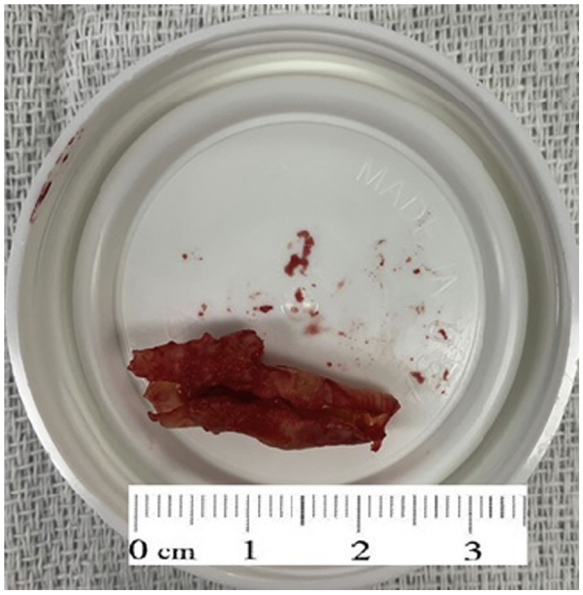

Figure 3 Left carotid plaque surgical specimen. The left carotid plaque (2.2 cm × 0.6 cm), which caused significant stenosis of the left internal carotid artery, was removed via left carotid endarterectomy.

symptoms resulting in CSS. The patient outlined in this case was diagnosed with CSS owing to his history of recurrent falls and reproduction of symptoms with CSM.

The mechanism of CSS is stimulation of carotid baroreceptors causing reflex bradycardia and AV block through vagal nerve mediation.[1] It is often triggered by progressive calcific carotid atherosclerosis, neck movements, tight collars, and extrinsic head and neck tumors compressing the carotid bulb; prior neck surgeries; or radiation. When performing CSM, it is important to auscultate for a carotid bruit prior to palpation. If a carotid bruit is heard, CSM can still be performed; however, the risk of embolization and clinical precautions should be considered prior to CSM. Patients should be on continuous telemetry monitoring with intravenous access, and with Advanced Cardiac Life Support supplies readily available. Potential complications of CSM include prolonged transient ischemic attack / cerebrovascular accident secondary to carotid plaque embolization, prolonged sinus pauses, and hypotension.[2] In most cases, a heart rate of 35–40 bpm may be tolerable without syncope; however, in this case the patient's mitral stenosis likely increased his sensitivity to sudden pauses of 1.6 seconds occurring with second-degree type 1 AV block.

There is currently limited evidence regarding permanent pacing for patients with CSS-related syncope.[3] According to the most recent ACC/AHA/HRS guidelines, in patients who had a symptomatic AV block attributable to a known reversible and nonrecurrent cause, and who have had complete resolution of the AV block with treatment of the underlying cause, permanent pacing should not be performed[4] [Evidence 3C-LD]. In this case, the patient experienced resolution of his syncope without recurrence of symptomatic bradycardia after removal of his carotid atheroma. Therefore pacemaker implantation was deferred and the patient was discharged with an implantable loop recorder to monitor for potential future recurrence of significant AV block.

Conclusion

When evaluating a patient with symptomatic bradycardia, a thorough history and physical examination, including auscultation for carotid bruits, is a cornerstone for identifying the etiology of the bradycardia. CSS due to carotid atheroma should be considered as a potentially reversible cause of symptomatic bradycardia and AV block in the appropriate clinical context.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2020.11.014.

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