

CLINICAL VIGNETTE

Symptomatic Bradycardia from Carotid Sinus Syndrome

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Case Presentation

Our patient is a 50-year-old woman admitted to the medicine service with a presumptive diagnosis of syncope. The day prior to admission she developed intermittent pre-syncope episodes without clear inciting factors. Symptoms were recurrent, non-positional, and not associated with any chest discomfort. Review of her history was notable for recent diagnosis of squamous cell carcinoma of the base of her tongue for which she underwent radical neck dissection one month prior to admission with an uncomplicated post-operative recovery.

Following admission, she was witnessed to have spontaneous sinus bradycardia to a heart rate of 30. Her bradycardia was treated successfully with intravenous atropine. These episodes were recurrent and associated with relative hypotension to a systolic blood pressure of 80 mmHg. CT of her head and neck revealed a phlegmon between the branches of her carotid artery concerning for an abscess. Given her history, consideration was given that this could represent a malignant mass or inflammatory process. She was admitted to the intensive care unit for temporary transvenous pacer placement as well as antibiotic treatment.

Discussion

Carotid sinus syndrome presents with episodes of syncope and is caused by stimulation of the carotid sinus, a baroreceptor. The carotid sinus is a collection of nerves that sits at the base of the internal carotid artery superior to the bifurcation of the internal and external carotid arteries. In a normal baroreflex, pressure changes to the internal carotid wall stimulates the carotid sinus, signaling through the glossopharyngeal nerve to the nucleus tractus solus in the brainstem. The efferent limb of the reflex is carried via sympathetic and parasympathetic nerves to the heart and blood vessels decreasing contractility, heart rate, and peripheral resistance.

Therefore, when the blood pressure increases, the baroreflex is activated and subsequently the blood pressure is reduced.¹

Abnormalities of the baroreflex resulting in fainting or falling may be due to several conditions in which the carotid sinus is affected or impaired, one of which is carotid sinus hypersensitivity. The Guidelines of the European Society of Cardiology defines carotid sinus hypersensitivity as presentation of cardioinhibitory (>3s asystole), vasodepressor (systolic blood pressure falls >50 mmHg), or mixed (both cardioinhibitory and vasodepressor) symptoms upon carotid

sinus massage for 10 seconds. Essentially, carotid sinus hypersensitivity is an exaggerated response of the stimulation of the baroreceptor, such that minimal mechanical pressure on the neck may lead to unexpected bradycardia and syncope.² This condition presents primarily in older patients with a mean age of 75 years with males twice as more likely to have this condition as females. The pathophysiological mechanism underlying a hypersensitive carotid sinus is not fully understood. Proposed mechanisms involve abnormal processing of baroreceptor input in the central nervous system and changes in the structure of the arterial wall.³

Abnormalities of the baroreflex may also result from an adjacent mass or fluid collection compressing on the carotid sinus. Pertinent to our patient, squamous cell carcinoma can metastasize to the retropharyngeal or parapharyngeal spaces and the resulting tumor may press on the carotid sinus causing syncope. Risk of metastasis of squamous cell carcinoma to the carotid sinus is increased when a patient has undergone a radical neck dissection. The carotid sheath serves as a barrier against invasion by squamous cell carcinoma and disruption of this barrier allows tumors to form around the carotid sinus. The mechanism of syncope caused by squamous cell carcinoma metastasis is broken down into two categories. One mechanism involves the lesion triggering syncopal episodes through pre-existing carotid sinus hypersensitivity. The other mechanism does not involve carotid sinus hypersensitivity, but results from the lesion irritating nerve fibers of the baroreflex and causing syncope.⁴

Post-surgical phlegmon or abscess in the area of the carotid sinus would present similar to a metastatic tumor. Phlegmons may result from bacterial infection in the area of a surgical procedure. A collection of cervical fluid has a similar appearance to an abscess or phlegmon on CT scans and should be considered as well as a non-infectious source of a mass compressing the carotid sinus.⁵

Rarely, carotid body tumors, or chemodectomas, develop and compress peripheral nerves around the carotid sinus, such as the vagus nerve, and cause syncope. The carotid body is a chemoreceptor located in the bifurcation of the carotid artery that detects changes in oxygen, carbon dioxide, pH, and temperature. These tumors present as palpable and painless cervical masses with elastic consistency under the angle of the mandible. A key feature of syncopal events caused by these tumors is that there is a premonitory phase prior to the syncope

in which the patient feels nauseous, perspires, and feels distress in the abdomen.⁶

In a differential of symptomatic bradycardia that includes carotid sinus syndrome, sinus node dysfunction should be considered. The sinoatrial node serves the pacemaking function of the heart. A dysfunction in this node results in arrhythmias that may cause bradycardia and associated syncope. Intrinsic causes of this dysfunction include infiltrative disease processes, such as amyloidosis and sarcoidosis, ion channel dysfunction, and remodeling of the node. The most common cause of intrinsic nodal dysfunction is degenerative fibrosis of the node associated with aging. Carotid sinus syndrome and sinus node dysfunction can present similarly clinically and require further investigation to differentiate.⁷

Conclusion

When evaluating patients presenting with syncope, carotid sinus syndrome should be considered. In addition to carotid sinus hypersensitivity, mass-effect compression of the carotid sheath may also lead to carotid sinus syndrome. In our patient, there was a high level of concern for recurrent squamous cell carcinoma, and she was referred for radiation treatment in consultation with her surgical oncologist. In addition, an abscess could not be completely ruled out as the location of her phlegmon precluded a biopsy, thus she was empirically treated with broad-spectrum antibiotics. After initiation of antibiotic treatment, she had no further episodes of bradycardia. However, given concern for recurrence, she was referred for pacemaker placement prior to hospital discharge.

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