Swallowing: Is It a Carotid Massage Equivalent Causing Syncope in Patients With Neck Masses?

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Syncope associated with the act of swallowing (deglutition syncope) and syncope associated with head and neck cancers have been documented independently. We present a case of syncope precipitated by a combination of both these mechanisms. It is important to recognize the coexistence of different hemodynamically significant mechanisms leading to syncope. A 66-year-old male recently diagnosed with head and neck cancer presented with syncope associated with eating. Diagnosis was complicated because of multiple factors in this patient, which individually could have led to syncope. The patient was on beta-blocker therapy, had a neck mass, and the episodes were associated with swallowing. Our hypothesis is that all these etiologies together led to the events but not in their typical mechanisms and raises the possibility that swallowing can be considered a carotid massage equivalent in patients with neck masses not physically extending to the carotid sinus.

KEY WORDS: swallowing; head and neck cancer; carotid massage.
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INTRODUCTION

Syncope is a sudden, transient loss of consciousness associated with the absence of postural tone and is followed by a rapid and complete recovery. The causes of syncope can be categorized as cardiovascular causes, which include arrhythmias, mechanical problems and abnormal cardiovascular reflexes; noncardiovascular causes including metabolic, neurologic, and psychiatric causes; and finally, unexplained causes. It is important to recognize the cause of syncope as cardiac syncope may predict a risk of subsequent cardiac arrest. Carotid sinus hypersensitivity also called carotid sinus syndrome accounts for 6% to 14% of cases with syncope. Our case considers a new mechanism for carotid sinus hypersensitivity leading to syncope.

CASE PRESENTATION

A 66-year-old male presented after an episode of syncope to our hospital. He was apparently eating breakfast when he felt dizzy, became pale, and blacked out. There was no loss of bladder or bowel control, seizure-like activity, chest pain, or palpitations. He denied any relation to postural changes, urination or defecation, or previous syncopal episodes. He had a history of hypertension and his medications included atenolol and lisinopril. A recent diagnosis of squamous cell carcinoma of the tongue with extensive regional lymph node metastasis particularly to the lateral cervical lymph nodes was being worked up at an outlying facility. An echocardiogram and cardiac catheterization at the same hospital were normal.

During the evaluation in the emergency department, the patient was having a bite of his lunch when we witnessed a similar episode where he complained of lightheadedness, became pale, and unresponsive, his heart rate dropped to 37/min with a palpable systolic blood pressure of 58 mmHg. The telemetry monitor showed sinus bradycardia. He recovered quickly after being laid flat and receiving intravenous fluids. Minutes after resuscitation, he was alert and oriented, and his blood pressure and pulse were 135/71 mmHg and 50/min, respectively, without any significant findings on exam except a palpable neck mass on the left. EKG showed sinus bradycardia. Laboratory exam and chest x-ray were normal.

The patient did not have any postural (suggestive of orthostatic syncope), exertional (suggestive of obstructive valvular disease or coronary ischemia), situational (such as cough, defecation or micturition), psychiatric symptoms, or symptoms suggestive of organic cardiac or neurological disease. Physical examination did not reveal any cardiovascular or neurological signs. As there was a concern for medication-induced syncope related to atenolol, it was withheld. As the patient's EKG and telemetry monitor did not reveal any arrhythmias other than the bradycardia during the event, we did not pursue further cardiac testing with a recent normal echocardiogram and cardiac catheterization. As the complete neurological history and examination were negative, we did not see any benefit of extensive neurological testing. Computerized tomography and angiography of the head and neck revealed a multiloculated cystic structure within the left carotid space located anterior to the sternocleidomastoid muscle compressing the anterolateral margin of the left jugular vein (Fig. 1) with no extension of the mass to the esophagus. Both the episodes of syncope occurred when the patient was eating and resolved soon after he was put on a liquid and soft diet. The patient was started on induction chemotherapy, and he did not have any further episodes of syncope even when normal diet was resumed.

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Figure 1. Computerized tomography and angiography of the head and neck. A multiloculated cystic structure within the left carotid space located anterior to the sternocleidomastoid muscle compressing the anterolateral margin of the left jugular vein.

DISCUSSION

The cause of syncope remains undiagnosed in about 50% of patients.¹ Although syncope associated with swallowing (swallow syncope or deglutition syncope) and syncope associated with head and neck cancers have been documented independently, our case emphasizes the coexistence of 2 or more mechanisms leading to syncope in an atypical manner.

Swallow syncope is a vagally mediated syncope occurring during or immediately after swallowing with afferent impulses from the upper gastrointestinal tract and efferent impulses to the heart which can produce bradyarrhythmias with atrioventricular block.² It is usually associated with organic or functional disorders of the esophagus.³ A temporal relationship to ingestion of food, carbonated beverage, and hot, cold, or iced liquids is found and does not necessarily have to be large or rapid boluses.^{4,5} It is unlikely that our patient has swallow syncope without any underlying gastrointestinal symptoms or abnormalities. Furthermore, these episodes can occur either with eating or drinking, and our patient did not have any more syncope after diet was resumed.

Postprandial syncope is syncope that occurs 45–60 minutes after a meal, generally in the elderly or those with autonomic failure because of abnormal release of vasodilatory gastrointestinal peptides.⁶ Our clinical findings during the episode without any previous symptoms or evidence of autonomic failure occurring during and immediately after a meal makes postprandial syncope unlikely.

Syncope related to neck masses is generally because of carotid sinus hypersensitivity or glossopharyngeal neuralgia.^{7,8} Syncope related to carotid sinus hypersensitivity is because of an exaggerated response to the stimulation of the carotid sinus. The glossopharyngeal nerve carries afferent impulses

from the baroreceptor area to the medullary cardiac and vasomotor centers from where the efferents descend into the vagus leading to bradycardia or vasodilatation, resulting in hypotension, presyncope, or syncope.⁹ There are 3 types of carotid sinus hypersensitivity. (a) The cardioinhibitory type comprising 70–75% of cases where the predominant manifestation is a decreased heart rate, which results in sinus bradycardia, atrioventricular block, or asystole caused by vagal action on the sinus and atrioventricular nodes. It is defined as asystole of ≥ 3 seconds with an SBP drop of < 50 mm Hg. (b) The vasodepressor type comprising 5–10% of cases where the predominant manifestation is a vasomotor tone decrease without a change in heart rate. The significant resulting drop in blood pressure of 50 mm Hg is caused by a change in the balance of parasympathetic and sympathetic effects on peripheral blood vessels. (c) The mixed type comprises 20–25% of cases. A combined decrease in heart rate and vasomotor tone occurs.¹⁰ It is unlikely that our patient had syncope related to glossopharyngeal neuralgia as this is associated with significant pain and our patient did not complain of any pain.

In our patient, the syncope was not entirely related to the neck mass as it was not physically encroaching onto the carotid sinus. It was only during the act of deglutition that there was a mechanical compression of the carotid sinus by the tumor mass leading to the irritation of the afferent fibers of the glossopharyngeal nerve producing syncope. Our patient had carotid sinus hypersensitivity of vasodepressor kind during these episodes of carotid massage. We hypothesize that in our patient the mechanism of the hemodynamic collapse was because of intermittent carotid massage by the neck mass during deglutition on a background of inability to mount a sympathetic response because of beta-blocker usage.

Treatment is recommended in patients with more than 2 syncopal episodes. Dual chamber cardiac pacing is beneficial in patients with carotid sinus hypersensitivity who have a cardioinhibitory response.¹¹ The medical treatment of the vasodepressor type remains highly unsatisfactory although there have been case reports of limited success with beta blockade either alone or in combination with ephedrine.¹² Surgical denervation of the carotid sinus has also been tried as an option.¹³ In patients with head and neck cancer, induction chemotherapy by has been shown to be a successful treatment option for syncope.¹⁴ Because we thought that the beta-blocker had some contribution toward the syncope and only limited data was available regarding its use in the vasodepressor type of carotid sinus hypersensitivity, our patient was not continued on it. Induction chemotherapy was chosen over Surgery or radiotherapy because Surgery is often palliative only and radiotherapy poses the risk of a syncopal event in an unmonitored environment.^{15,16} Our patient did not have any further episodes of syncope.

CONCLUSION

We propose a new mechanism suggesting a carotid sinus equivalent. Our case raises the possibility that swallowing can be considered a carotid massage equivalent in patients with neck masses not physically extending to the carotid sinus.

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