



Situational Syncope Triggered by Swallowing

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ABSTRACT

Syncope is characterized by a transient loss of consciousness. Swallow syncope, a rare cause of syncope, is caused by vagus nerve activation resulting in vasodilation and bradycardia, thus causing transient hypotension and cerebral hypoperfusion. It is diagnosed through clinical history, cardiac, and esophageal evaluation. We present a case of swallow syncope in a patient with significant cardiac history. Initial cardiac and esophageal testing was normal. Long-term telemetry revealed bradycardia with swallowing, and the patient underwent pacemaker placement. Swallow syncope is associated with a variety of esophageal and cardiac conditions. Management involves addressing the underlying cause; pacemaker placement is sometimes necessary.

KEYWORDS: syncope; swallow syncope; reflex syncope; pacemaker

INTRODUCTION

Syncope is a common presenting complaint that accounts for 1.5% of all emergency department admissions and 6% of hospital admissions.¹ Syncope is characterized as a transient loss of consciousness and postural tone, followed by a spontaneous recovery^{2,3}. Patients often describe different prodromal symptoms of syncope including dizziness, lightheadedness, nausea, or other visual disturbances.⁴ The aim of this article is to present a rare case of chronic swallow syncope in the absence of acute cardiac or esophageal etiology.

CASE REPORT

A 65-year-old White male with a medical history of pericarditis, chronic atrial fibrillation on apixaban, essential hypertension, obstructive sleep apnea (on continuous positive airway pressure at night), and nonobstructive coronary artery disease presented to his primary care provider with chronic syncope. Since his late 40s, the patient had been experiencing episodes of fainting triggered by swallowing, specifically with liquids and soft foods. It initially occurred once a month, but frequency increased to up to 3 times daily in the months before presentation. During these episodes, he described a feeling of food getting stuck and subsequent generalized weakness. He then frequently experienced a syncopal episode, however notes that he could sometimes catch himself before losing consciousness. Individuals observing these episodes witnessed the patient lose muscle control. He did not regain consciousness for a few seconds. After he regained consciousness, he was confused for a few seconds. The patient denied any prodromal symptoms such as palpitation, aura, changes in vision, or abnormal taste.

Initially, the patient's primary care physician referred him to a gastroenterologist for further testing. Because swallow syncope is a diagnosis of exclusion, a full cardiac workup including tilt table testing, echocardiogram, and long-term telemetry monitoring was needed to rule out cardiac etiology of his symptoms. The patient additionally underwent an esophagram which was notable for moderate esophageal dysmotility with intermittent spasms throughout, a small hiatal hernia with reflux (Figure 1), and a small posterior Zenker diverticulum (Figure 2). The patient also underwent an esophagogastroduodenoscopy (EGD), which showed the Zenker diverticulum (Figure 3) and gastritis in the stomach body. During the EGD, endoscopic functional lumen imaging probe and



Figure 1. Esophagram image of hiatal hernia.

high-resolution esophageal manometry (Figure 4) were performed. Hiatal hernia was not seen during the EGD. The patient experienced severe bradycardia with esophageal manipulation during the procedure which required ephedrine to resolve.

Following the EGD, an echocardiogram and tilt table testing were performed and were grossly unremarkable. The patient was referred to electrophysiology who suspected swallow syncope and placed a 30-day mobile telemetry device with hopes of capturing a syncopal event. The device recorded bradycardia while swallowing (Figure 5) and showed pauses associated with syncope which were consistent with his typical episodes.

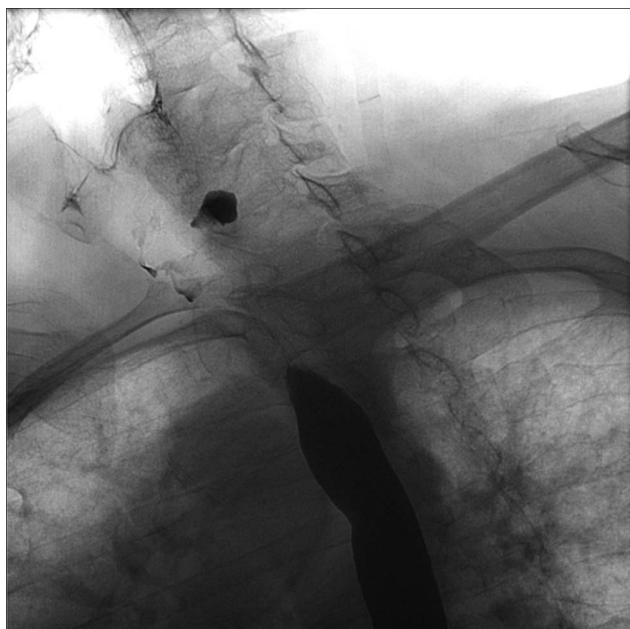


Figure 2. Esophagram image of Zenker diverticulum.

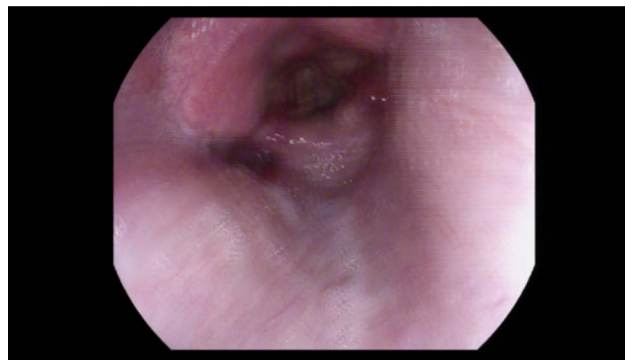


Figure 3. Endoscopic view of Zenker diverticulum.

These results contributed to the diagnosis of swallow syncope, and the patient underwent pacemaker placement. Roughly 4 months after his initial pacemaker procedure, the patient contacted electrophysiology with symptoms consistent with pacemaker infection requiring pacemaker extraction and reimplantation. Two months following reimplantation, he returned for a follow-up where he reported feeling well and denied any cardiac complaints including chest pain, palpitations, shortness of breath, dizziness, orthopnea, syncope, or near syncope. The most recent note with gastroenterology, 4 months after the second pacemaker placement, shows no further syncopal episodes.

DISCUSSION

Swallow syncope, a subset of reflex syncope, is characterized by a brief period of hypoperfusion to the brain induced by swallowing.⁵ The neurological mechanism of swallow syncope has been postulated but is poorly understood. The most reported mechanism involves vagal nerve overactivation, leading to increased parasympathetic tone. When swallowing, afferent vagal fibers travel to the solitary nucleus in the medulla, activating vagal efferents and leading to increased parasympathetic tone and bradycardia.⁶ Multiple studies have shown that atropine, an anticholinergic agent, can prevent bradycardia in cases of swallow syncope, supporting the presence of a reflex arc between afferent sensory fibers of the esophagus and efferent parasympathetic fibers affecting the heart.^{6,7} Our patient exhibited severe bradycardia during esophageal manipulation, implicating esophageal mechanoreceptors in the pathogenesis of swallow syncope.

Swallow syncope is suspected when syncopal episodes consistently follow swallowing, and cardiac evaluation is normal. In this case, the patient's echocardiogram, tilt table test, and EGD were unremarkable. The 30-day mobile telemetry study demonstrating bradycardia associated with swallowing was diagnostic.

The most documented and successful form of treatment for swallow syncope is a pacemaker.⁶⁻⁸ Our patient received a permanent dual-chamber pacemaker which demonstrated success

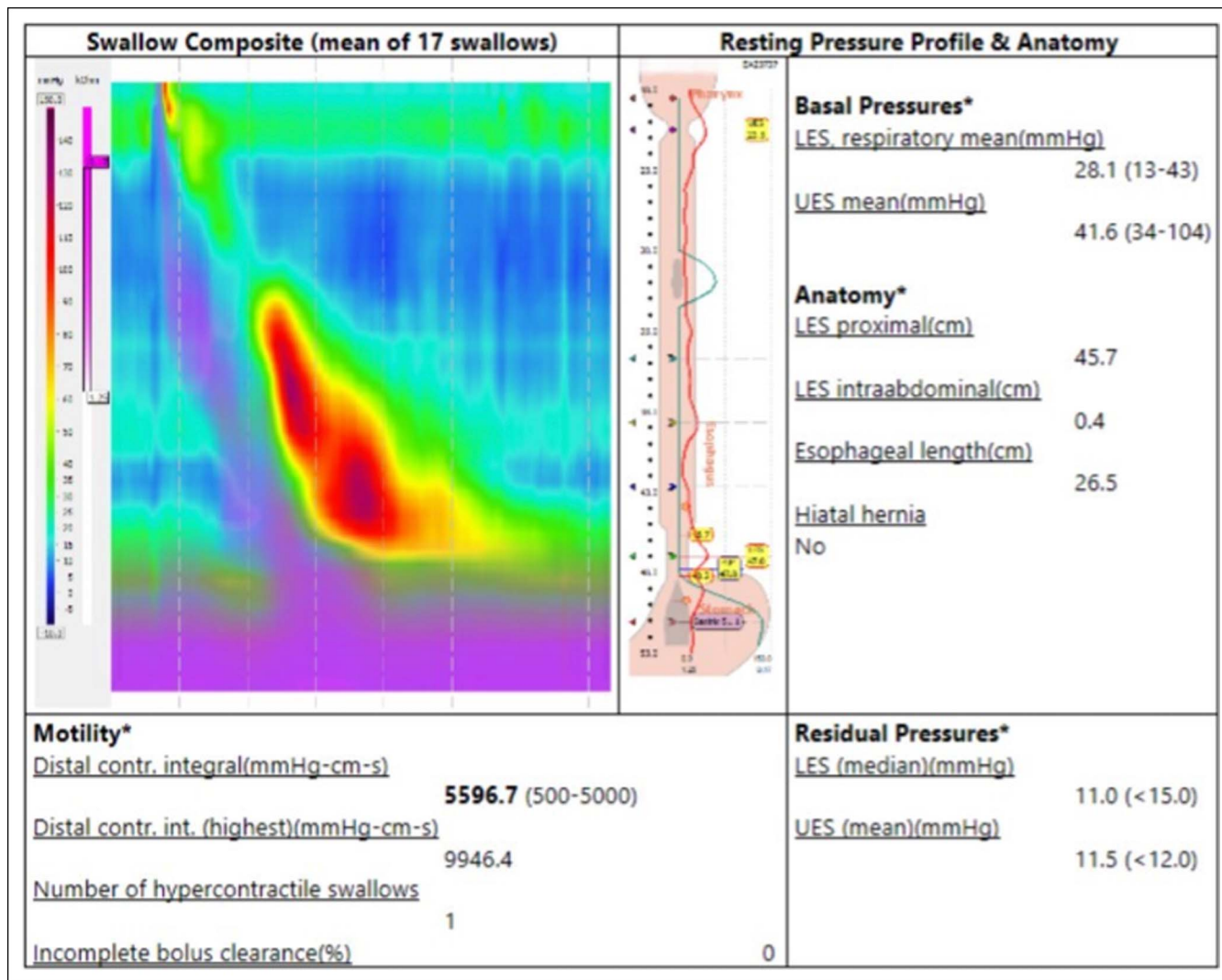


Figure 4. Manometry showed normal esophageal motility according to the Chicago classification version 4. LES, lower esophageal sphincter; UES, upper esophageal sphincter.

in reducing bradyarrhythmia and syncopal episodes⁹. As seen in our patient, pacemaker implantation carries a risk of infection that may require removal and reimplantation.

Treating the underlying causative factor has also been shown to be successful in swallow syncope. Four patients with underlying hiatal hernias have been treated by surgical correction of the hernia, all of whom had resolution of symptoms.⁸ Similarly, one patient with esophageal stricture and one patient with achalasia were cured of their syncope with esophageal dilatation.⁶ Our patient’s hiatal hernia was quite small and was not redemonstrated on EGD, thus surgical correction would likely not have yielded much benefit. In addition, correction of Zenker diverticulum is typically only advised if the diverticula is larger than 2 cm, and our patient’s was only 1.2 × 0.7 cm. That being said, surgical correction is also considered in symptomatic cases. One could argue the possibility of a Zenker diverticulum compressing or affecting the function of the vagus nerve, which would make this patient’s case symptomatic and qualify him for

correction. A surgical correction of the Zenker diverticulum can be done by an endoscopic or open approach. In our patient, previous endoscopic procedures were complicated by significant bradycardia, making this procedure higher risk. The permanent pacemaker was a less invasive and safer procedure for our patient in this setting, although, had it not worked, open correction of the Zenker diverticulum may have been the next step for symptom control.

Avoidance of the triggering factor may be beneficial in reducing the number of syncopal episodes experienced by an individual.⁴ In swallow syncope, the trigger cannot be avoided but may be modified with changes in diet.⁶ Pharmacological interventions, such as anticholinergic drugs, have been researched with inconsistent results and efficacy.⁸ However, before the invention of the pacemaker, atropine demonstrated efficacy in limiting syncopal events. Some cases of swallow syncope have been successfully treated with long-term proton-pump inhibitor use or surgical denervation of portions of the esophagus.⁶

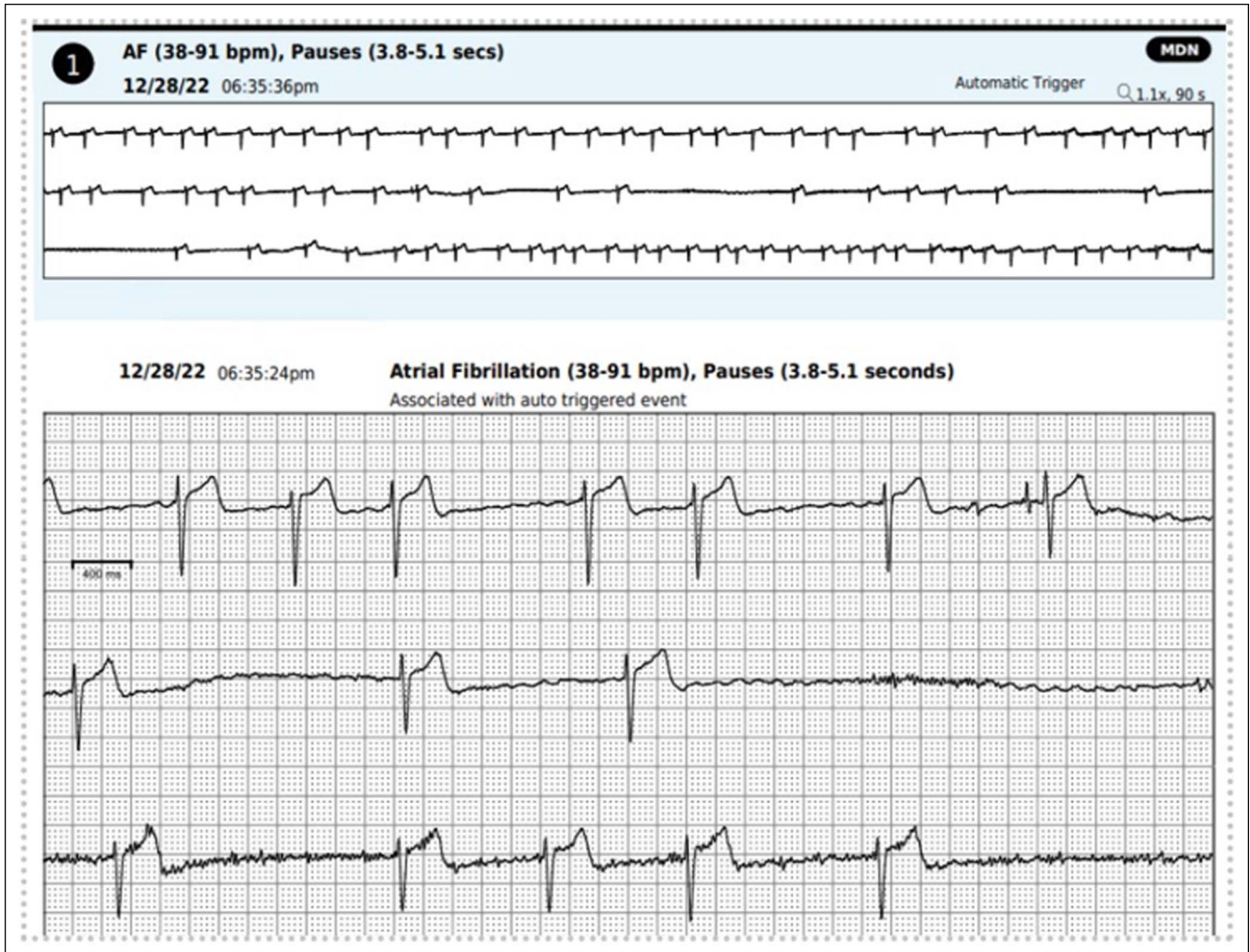


Figure 5. Mobile cardiac telemetry indicated a 3.8–5.1 second pause likely triggered by swallowing. AF, atrial fibrillation.

Swallow syncope, while uncommon, should be considered in patients with a temporal relationship between swallowing and syncopal episodes. A detailed history of the illness, extensive esophageal and cardiac testing, in addition to individualized therapeutic strategies are crucial for proper diagnosis and treatment. Here, we present a case of chronic swallow syncope in the absence of acute cardiac or esophageal etiology. This case demonstrates the importance of considering swallow syncope in patients with unexplained syncope and stresses the role of bradycardia management in treatment.

DISCLOSURES

Author contributions: AL Ellington: article Guarantor, data Interpretation, and manuscript drafting; ST Terry: data interpretation and manuscript drafting; MNH Ellis: data acquisition and analysis, manuscript editing; SB Clayton: project conceptualization, data interpretation, manuscript editing.

Acknowledgments: Acknowledgment to Karl Richardson of Department of Internal Medicine, Section of Cardiology, Wake

Forest School of Medicine, Winston-Salem, NC for interpretation of cardiac studies.

Financial disclosure: None to report.

Previous presentation: This case report was previously presented at American College of Gastroenterology Annual Meeting on October 22, 2023, in Vancouver, BC, Canada.

Informed consent was obtained for this case report.

Received August 25, 2024; Accepted December 9, 2024

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