

Deglutition Syncope: A Case Report and Review of the Literature

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Abstract

Deglutition syncope (DS) is a rare, neurally-mediated syncopal syndrome arising from an aberrant vagotonic reflex during swallow-associated esophageal dilation. Its association with gastroesophageal disorders often prompts gastroenterology consultation. An 89-year-old man with recent dysphagia and otalgia was admitted after a syncopal episode occurred while eating. Esophageal imaging and endoscopy demonstrated no causative abnormalities. Maxillofacial imaging revealed chronic sinusitis and mastoiditis. Telemetry monitoring demonstrated high-grade atrioventricular block and pause associated with swallowing. His symptoms and swallow-associated arrhythmia resolved after dual chamber pacemaker implantation. DS is highly treatable once identified and multidisciplinary coordination is helpful in optimizing outcomes and avoiding superfluous testing.

Introduction

Deglutition syncope (DS) is a form of neurally-mediated syncope in which loss of consciousness occurs during or immediately after swallowing. Syncope is thought to occur via an aberrant vagal reflex in which esophageal vagal afferents are stimulated by dilation during deglutition, leading to cerebral hypoperfusion via cardiac arrhythmia and/or vasodepression.¹ This condition was first reported by Thomas Spens in 1793.²

Case Report

An 89-year-old man was brought to the emergency department after a witnessed syncopal episode that occurred while he was eating at a restaurant. He remained unconscious for several seconds, but was easily revived. His medical history included longstanding reflux controlled with a proton pump inhibitor, coronary artery disease, bioprosthetic aortic valve replacement for aortic stenosis, and a right carotid artery stenosis.

The patient had recently started an evaluation for left-sided otalgia and dysphagia to solids and liquids associated with lightheadedness and near-syncope. The symptoms were triggered by ingestion of large boluses of food and carbonated beverages. Prior cardiology evaluation included 24-hour ambulatory electrocardiography, which revealed first-degree atrioventricular block and transient, asymptomatic Mobitz type II second-degree atrioventricular block. A transthoracic echocardiogram was unrevealing. An esophagram revealed mild intermittent esophageal dysmotility, but no structural abnormalities. Maxillofacial CT showed chronic left-sided sinusitis and bilateral mastoiditis treated with a short course of steroids and clarithromycin. Upper endoscopy and esophageal manometry had been recommended, but had not yet been performed.

On current presentation, initial laboratory studies demonstrated a mild normocytic anemia and thrombocytopenia. Troponin T was negative. Electrocardiogram revealed normal sinus rhythm, first-degree atrioventricular block, and left anterior fascicular block, but was unchanged from prior. A noncontrast maxillofacial CT showed persistence of sinus disease. The patient was admitted and placed on continuous telemetry monitoring.

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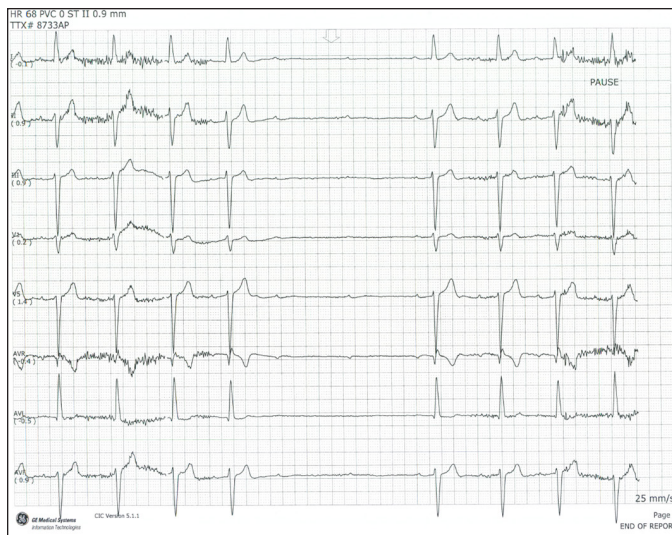


Figure 1. Electrocardiogram while patient swallowed pills. Pre-syncope symptoms (lightheadedness, dizziness) were elicited during the presented interval. Note the presence of type II, second-degree atrioventricular block and 2 non-conducted P-waves, with a total pause of approximately 3 seconds.

The following morning, he was swallowing medications and experienced recurrent symptoms accompanied by a period of atrioventricular block resulting in a 3-second pause (Figure 1). A dual chamber permanent pacemaker was placed after electrophysiology evaluation. Upper endoscopy revealed a normal esophagus without stricture, stenosis, or inflammation. There was mild chronic gastritis without *Helicobacter pylori* (Figure 2). The patient's dysphagia resolved and has not recurred at 3-month follow-up. His otalgia initially persisted, but was corrected by subsequent sinus surgery. Surveillance pacemaker interrogation has not revealed any recurrent pauses or arrhythmias requiring pacemaker correction.

Discussion

DS is a situational form of the reflex, or neurally-mediated, syncope syndromes. Patients may present with overt syncope or more subtle pre-syncope symptoms, such as dizziness, lightheadedness, or weakness.¹ Dysphagia and odynophagia have been previously described as presenting symptoms.^{1,3,4} Given its rarity, the overall prevalence of DS cannot be accurately estimated. It occurs most commonly in adult males.⁵ Association with gastroesophageal disorders is frequently reported, but not required. Of the 80 cases reported between 1793 and 2011, 31 (38.8%) occurred in the setting of digestive disease.⁶ Associated conditions include esophageal stricture, web, and spasm, as well as gastroesophageal reflux, esophagitis, achalasia, and esophageal carcinoma.^{5,6} Although less common, underlying cardiac disease is well-described. The precise mechanism of syncope in DS remains speculative, and several pathways may be involved.

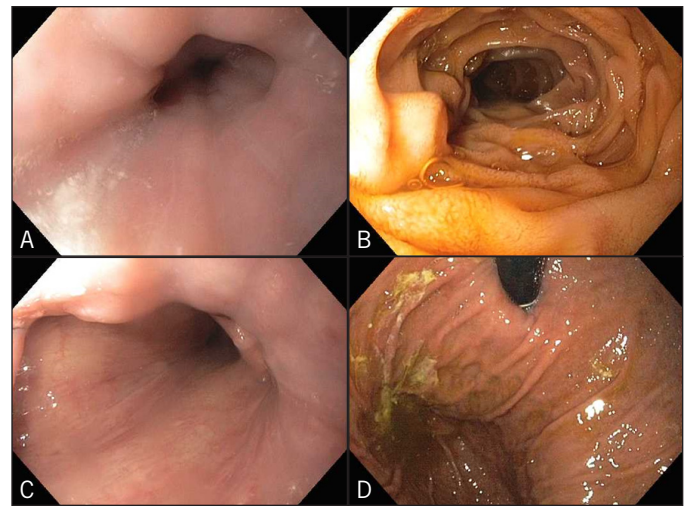


Figure 2. Examination of the (A) esophagus and (B) duodenum demonstrated normal landmarks and endoscopic appearance. (C) Antral erythema in a patchy distribution was noted. (D) Retroflexion and examination of the fundus and cardia was also normal.

Current theories suggest the irritation or aberrant activation of a vagal reflex, resulting in cerebral hypoperfusion. This vagal mechanism is supported by numerous reports documenting the prevention of syncope with pre-administration of atropine.⁷

Diagnosis is established by a clinical history suggesting the temporal association of swallowing with syncopal or presyncopal symptoms. A careful history of episodes and associated triggers should be taken. As in our case, documentation of an associated arrhythmia via continuous cardiac monitoring is critical for determining the appropriate treatment. If unsuccessful, electrophysiology studies should be strongly considered. Once the diagnosis is established, attempts at identification of an underlying cause should be undertaken. Given the association with gastroesophageal disease, esophageal radiography and upper endoscopy are important initial studies. If negative, esophageal manometry, pH, and impedance studies should be considered.¹

In our patient, the existence of numerous plausible etiologic conditions precluded the precise identification of an underlying cause. Despite a history of coronary artery disease and cardiothoracic surgery, no cardiac conduction disease was detected. Despite dysphagia, no gastroesophageal pathology was identified. The chronic sinusitis and mastoiditis bears mention, as a case of DS associated with periodontitis and mastoiditis has been reported.⁸

Fortunately, DS is highly treatable. Although no randomized trials have been conducted, there are several accepted tenets to its management. Identification and avoidance of trigger substances is vital. Carbonated beverages have been

frequently implicated as the inciting agent for DS, as the dissolved carbon dioxide distends the gastric lumen and elicits the vagal reflex.^{6,9} Cold beverages, sandwiches, and sticky foods have also been linked.¹⁰⁻¹² Common underlying conditions, such as gastroesophageal reflux and esophageal spasm, can be treated in the conventional manner. Cases of DS due to achalasia and hiatal hernia have also been reported and resolved with pneumatic balloon dilation and Nissen fundoplication, respectively.^{3,13,14}

Withdrawal of medications that slow cardiac conduction (e.g., beta blockers, calcium channel blockers) or facilitate vasodepression (e.g., angiotensin blockade) is also a mainstay of therapy.^{5,6} Anticholinergic agents (e.g., atropine, scopolamine) and sympathomimetics (e.g., epinephrine, isoprenaline) have been utilized to abolish the vagal reflex and increase the ventricular rate^{6,12}; however, their side effects limit their practical applicability.⁵ Ultimately, permanent pacemaker implantation is the most efficacious method of resolving symptoms and preventing life-threatening bradyarrhythmias.⁶

Disclosures

Author contributions: A. Kahn drafted the manuscript. LM Koepke assisted with drafting and critically revising the manuscript. S. Umar conceptualized and designed the manuscript, assisted with critical revision, and is the article guarantor.

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