



# First Reported Case of Deglutition Syncope With Underlying Suppurative Parotitis

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## ABSTRACT

Deglutition syncope and carotid sinus hypersensitivity are neurally mediated events, leading to potentially dangerous arrhythmias and cardiovascular events. Mostly related to underlying gastroesophageal or cardiovascular causes, sometimes, this might not be the case. We report the first-ever documented case of deglutition syncope with acute suppurative parotitis, which resolved after resolving the parotid gland's swelling.

## INTRODUCTION

Swallowing syncope, also known as deglutition syncope (DS), is one of the rarest forms of syncopal syndromes with the sparse literature on documented cases and mechanisms. DS is supposed to arise from an imbalance between the overfiring parasympathetic tract and underfiring sympathetic outflow tract, which leads to bradycardia, asystole, heart blocks, or hypotension.<sup>1-3</sup> DS has also been shown to overlap with carotid sinus hypersensitivity.<sup>4</sup> Most reported cases of DS had underlying cardiovascular and gastrointestinal causes.<sup>2</sup>

## CASE REPORT

An 82-year-old man with a known history of hypertension, hyperlipidemia, and gastroesophageal reflux disease presented to the emergency department after 2 episodes of syncope while chewing solid food. When the first episode occurred, he passed out with no preceding headache, aura, light-headedness, visual changes, or palpitations. There was no shakiness, tongue bites, and urinary or bowel incontinence preceding the event. He regained consciousness in a few seconds; however, the second episode recurred right after when the patient tried to eat again. He did not have any diaphoresis, chest pain, shortness of breath, extremities' weakness, or decreased vision during both episodes. He was sent to the emergency department, where, while having a snack, he passed out again. Of note, the patient started to notice some swelling near his left mandibular angle associated with some mild, achy, dull pain that worsened with opening mouth and mastication.

He was vitally stable with a blood pressure of 134/67, normothermic, and having a resting heart rate of 76 beats per minute on admission. Orthostatic vitals were performed, which were normal. On examination, his left jaw had marked swelling over the parotid region, which was tender to palpation and warm to touch. On manual compression and massage of the parotid gland, pus was seen exuding out of the orifice of the left-sided Stensen duct opposite to the upper second molar. The rest of the examination, including cardiopulmonary, neurological, and cerebellar, was unremarkable. The syncope was reproduced with mastication, and hence, fall precautions were enforced.

Computed tomography with contrast of the maxillofacial region was decided to pursue to study the parotid region, revealing evidence of significant inflammation of the left parotid gland, extending and involving ipsilateral carotid sheath (Figure 1). Given the patient's metabolic disorders and high risk of cardiovascular causes of syncope, the patient was admitted to medicine service with



**Figure 1.** Computed tomography maxillofacial with contrast showing the inflammation in superficial and deep lobes of the left parotid gland (red dotted lines), extending up to the carotid sheath and compressing its components. A normal carotid sheath (red circle) is also marked for comparison.

telemetry. Erythrocyte sedimentation rate and C-reactive protein were slightly elevated with no leukocytosis. Comprehensive panel was impressive for normal electrolyte, blood urea nitrogen, and creatinine denoting euvolemic status with no evidence of dehydration (Table 1). The patient was asked to chew solid food under observation, and telemetry monitoring was observed to see the effect. The patient was noted to be passed out, and bradycardia of 54–55 beats per minute lasting 16 seconds was observed on telemonitoring, returning to normal heart rate with no asystole or heart blocks (Figure 2).

To rule out structural heart disease, transthoracic echocardiography was performed, which turned out to be normal. Pus expressed from the Stensen duct was sent for culture and sensitivity, which only grew oral microbial. Because our patient was only having events on solid food intake, the diet was shifted to liquid form with sips. Considering the clinical confirmation of the diagnosis of acute suppurative parotitis, antimicrobial coverage with amoxicillin-clavulanate 875 mg twice daily for 7 days along analgesics was given. Gentle massage of parotid area, increased fluid intake, and suckling of sour candies/lozenges were highly encouraged. The patient was observed in the hospital for 2 days, during which the swelling subsided. Solid diet was reinstated, which did not cause any further syncopal events. This eradicated any possible evaluation for coronary angiography or a pacemaker. The patient had a normal follow-up 4 weeks later.

## DISCUSSION

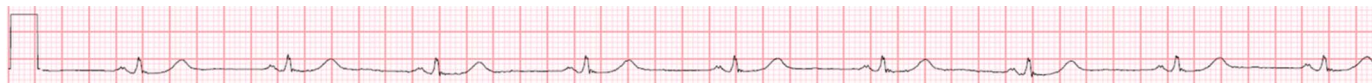
Swallow syncope, also known as DS, is a rare, situational, neural-mediated syncopal event. DS can manifest as either a subtle event such as light-headedness, weakness, or dizziness or overt such as overt syncope. It is associated with various cardiovascular disorders such as coronary artery disease, rheumatic heart disease, sick sinus syndrome, atrial fibrillation, and aortic aneurysm; gastrointestinal disorders, mainly hiatal hernia, esophagitis, and esophageal webs or strictures; and metabolic disorders such as hypertension, diabetes mellitus, hyperlipidemia, and glossopharyngeal neuralgia. The incidence of DS is more clustered at an older age and in males but can occur in anyone, irrespective of sex and age.<sup>1,2</sup>

Given the sparsity of literature, the mechanism of DS is poorly understood. It is likely due to imbalance and aberrancy of the vagal circuit with sympathetic tone outflow. Afferent impulses from the glossopharyngeal nerve in the pharyngeal plexus and the cranial vagus nerve are transmitted to the medulla oblongata’s nucleus solitarius from which signals are transmitted through reticular formation to the posterior nucleus which propagates signals to the efferent esophageal nerve, branch of the vagus nerve carrying parasympathetic signals and initiates peristalsis. Presence of reflex arcs between afferent sensory fibers and parasympathetic fibers of the cardiac branch results in inappropriate vagal activation, leading to bradycardia, atrioventricular delays, asystole, and hypotension. Sympathetic outflow can also be blunted, leading to overt parasympathetic outflow.<sup>2–4</sup> Moreover, mechanoreceptors in the lower esophageal region might also contribute to DS’ pathogenesis, which can be reproduced by a fizzy drink challenge, causing abrupt pressure changes, leading to distension in the esophagus and balloon inflation test during esophagogastroduodenoscopy.<sup>2,5</sup> Because various reasons can compound to DS, its treatment can range from conservative management, avoidance of triggers such as medication including beta-blockers and calcium channel blockers, diet modification, and treatment of underlying cause to evaluation for and placement of permanent pacemakers. Before proceeding to definitive treatment, a thorough history and physical examination compounding various syncope causes should be elicited.<sup>2</sup> However, it is worth mentioning that about half of syncope cases will not show any specific causation.<sup>6</sup> As most DS causes are related to either gastroesophageal or cardiac causes, after doing 24 hours of telemonitoring and echocardiography for evaluating structural

**Table 1. Abnormal laboratory values at presentation**

Laboratory	Patient	Reference range
Glucose (mg/dL)	119	70–99
ESR (mm/hr)	46	1–10
hs-CRP (mg/dL)	12	<0.3

CRP, C-reactive protein; ESR, erythrocyte sedimentation rate.



**Figure 2.** Sinus Bradycardia with the heart rate of 54–55 beats per minute.

heart disorders, complete gastroesophageal evaluation with, but not limited to, barium swallow and esophagogastroduodenoscopy should be performed.<sup>2,3</sup>

Carotid sinus hypersensitivity (CSH) has also been described in certain cases of syncopal events, and it is unclear whether they have an association with DS.<sup>7</sup> The carotid sinus is a dilated neurovascular structure at the junction of the internal and external carotid artery, which is enriched with baroreceptors; increased pressure in these sinuses can be sensed by afferent glossopharyngeal nerve fibers which transmit signals to nucleus solitarius and outflow of parasympathetic fibers, thereby cause bradycardia and/or hypotension.<sup>8</sup> Like DS, CSH is also common in the elderly population.<sup>2,8</sup> Part of the reason it is more common in the elderly could be attributed to the degeneration of autonomic nuclei in the medulla oblongata and overt responses.<sup>9</sup> To date, literature mentions 3 varieties of CSH: cardioinhibitory type: leading to a decrease in heart rate, asystole, and heart blocks. Asystole should be above 3 seconds, but systolic blood pressure should not drop more than 50 mm Hg, vasodepressor type: isolated systolic blood pressure drops of more than 50 mm Hg, and mixed: having mixed manifestation of both cardioinhibitory and vasodepressor type.<sup>8</sup>

Cases of CSH have been documented in mastoiditis, nasopharyngeal carcinoma, and head and neck tumors and have been shown to resolve with the resolution of underlying tumor mass.<sup>4,10–12</sup> Given the parotid gland position and its proximity to parapharyngeal space and internal carotid artery, it might be possible that our patient could also have had a component of CSH apart from DS, which has been described in a few cases of head and neck cancers in literature.<sup>10–13</sup> It proposes an unusual link between DS and carotid sinus hypersensitivity, which can compound together and cause an alarming syncope presentation in elderly patients. Before this case, there has not been any case that reported DS with parotid swelling, which opens doors for further work in the field of syncope and warrants a better understanding of etiopathogenesis.

## DISCLOSURES

**Author contributions:** SHB Waqar and AA Khan wrote the manuscript and revised it for intellectual content. O. Mohiuddin

edited the manuscript and reviewed the literature. O. Mohiuddin and T. Sheikh revised the manuscript for intellectual content. SHB Waqar and T. Sheikh approved the final manuscript.

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