Swallow Syncope:
A Case Report and Review of the Literature

Subhashis Mitra, MD; Tiffany Ludka, MD; Shereif H. Rezkalla, MD; Param P. Sharma, MD; and Jiangming Luo, MD, PhD

Corresponding Author:
Jiangming Luo, MD, PhD
Department of Hospital Medicine
Marshfield Clinic
1000 North Oak Avenue
Marshfield, WI 54449
Email: luo.jiangming@marshfieldclinic.org

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Abstract

Swallow or deglutition syncope is a relatively rare syndrome. It is vagally mediated syncope induced by swallowing. Swallow syncope may occur at all age groups and is treatable when diagnosed. A 60-year-old woman presented with an episode of syncopal attack associated with swallowing a sandwich. She had a 6-month history of recurrent episodes of lightheadedness while eating solid foods. Telemetry monitoring demonstrated several episodes of severe bradycardia and complete atrioventricular (AV) block with up to 7.0 seconds pause associated with meals. Computed tomography of head and neck revealed no significant findings and barium esophagram was normal. Echocardiogram was within normal limits. Her symptoms resolved after permanent pacemaker placement. We review the diagnosis, mechanism and management of swallow syncope.

Keywords: Syncope, Swallow, Computed tomography, Bradycardia, Atrioventricular block, Pacemaker, Diagnosis
**Introduction**

Swallow syncope is a relatively rare cause of syncope which belongs to the neurally mediated reflex syncopal syndromes. It occurs due to the vagal reflex during deglutition, causing inhibition of the cardiac conduction system. Since the first case report of swallow syncope in 1773 by Spens (1), there have been only about 80 case reports of swallow syncope in the literature to date. In most of the cases there is an underlying abnormality in the esophageal or cardiac conduction system. However there have been reported cases without any esophageal or cardiac abnormality. In this article we report a case of swallow syncope and review the literature.

**Case report**

60-year-old woman was admitted with history of recurrent episodes of lightheadedness and one episode of syncope. She has been having several episodes of lightheadedness while eating solid food for the last 6 months. During these episodes she would usually stop eating and the feeling of lightheadedness would subside spontaneously. She was admitted for further evaluation after she lost consciousness while eating a peanut butter sandwich. Her past medical history was significant for hypertension and hyperlipidemia. She has been on treatment with Atenolol since 2004 and has been taking 100mg once daily since 2005. Her family history was unremarkable for any arrhythmias or syncope.

On admission, her heart rate was 54 beats per minute and blood pressure was 160/94mm Hg with no evidence of orthostatic hypotension. Rest of the physical examination including a comprehensive neurological examination, was normal. Hematological and biochemical tests were normal. 12 lead electrocardiogram (EKG) showed sinus bradycardia with rate of 54 beats per minute and 1st degree AV block with a PR interval of 224msec. Carotid massage did not produce any symptoms or conduction abnormalities. Chest X ray did not reveal any cardiopulmonary abnormality. Echocardiogram was within normal limits. Atenolol was discontinued after admission. Telemetry monitoring demonstrated multiple episodes of severe sinus bradycardia (less than 35 beats/min) and complete AV block with pauses up to 7.0 seconds in the meal times (Fig. 1a). A representative telemetry strip showing 7.0 second pause during the meal time is shown in Fig 1b. Computed tomography of head, cervical spine and neck revealed no significant findings and barium esophagram was normal. Two days after discontinuing Atenolol the patient underwent another swallow testing to see if this reproduced her symptoms. She was challenged with tap water, cold water, hot tea, cold sprite, pudding, bagel and English muffin in that order. Five to ten seconds after taking muffin, four episodes of the second degree type 2 AV block were detected on the rhythm strip. Liquids (hot or cold) and soft food did not cause any problem. She underwent a Saint Jude model #5826, dual chamber permanent pacemaker implantation before discharge. The patient remains asymptomatic after 6 months.
Discussion

Swallow syncope is a relatively rare syndrome. Swallow syncope belongs to the “reflectional syncope” syndromes. Omi et al (2) reviewed 63 reported cases of swallow syncope in children and adults from the English-language literature between 1793 and 2005. Since then another 17 cases have been reported in the English literature [Table 1]. This syndrome is more common in males and most patients were adults but some cases were also noted in young children (2). Swallow syncope has been known to occur in patients with organic or functional disorders of esophagus. These disorders include esophageal spasm, esophageal stricture, achalasia, esophageal diverticulum, esophageal cancer and hiatus hernia (2). Swallow syncope may also occur in the absence of esophageal diseases and has been observed in the setting of cardiac diseases like acute myocardial infarction, rheumatic carditis and digoxin use (2). Some cases were reported to be associated with ascending aortic aneurysm (3), thoracic surgery, advanced lung cancer and transient hypoxia (2). However in a significant number of cases, no underlying disease may be found (2). As shown in the Table 2, the distribution of underlying diseases in the previously reported 80 cases of swallow syncope are: digestive disease (38.75%), cardiac disease (15%), other (7.5%) and none or unknown (38.75%).

The pathophysiology of the swallow syncope is not completely understood, and several different mechanisms may exist. Vagal reflex plays a major role as pretreatment with atropine is effective in preventing bradycardia and syncpe in a significant number of patients. The common innervation of the esophagus and heart by the vagus nerve has been postulated to play an important role in swallow syncope. Mechanoreceptors in the esophagus, which are activated by stretching, may play an important role (4). They sense distension and send signals along the esophageal plexus via the vagus nerve to the brainstem. The efferent impulses from the brainstem reach the sinoatrial node via the right vagus nerve and the AV node via the left vagus nerve. The efferent signals leads to various types of paroxysmal bradyarrhythmias and temporary reduction of cardiac output which leads to cerebral hypoperfusion and possibly, syncope. Others have observed that swallow syncope can occur even in the absence of bradycardia, as increased afferent vagal stimulation may lead to syncope via sympathetic withdrawal, resulting in peripheral vasodilation and hypotension (5). AV block has been reported most frequently, though other arrhythmias like sinus bradycadia, sinoatrial block and complete atrial and ventricular asystole has also been observed. Paroxysmal atrial fibrillation could also be associated with swallow syncope (6).

The diagnosis of swallow syncope requires careful elicitation of the temporal relationship between swallowing solid or liquid food and lightheadedness and syncope. Provocative testing with various types of solid and liquid foods should be attempted. As esophageal disorders may be associated with a majority of cases of swallow syncope, further work-up to exclude possible structural or functional
esophageal pathology has been suggested. Echocardiogram and resting and ambulatory EKG should be performed to rule out any underlying cardiac pathology.

Management of swallow syncope involves withdrawal of all medications that can cause delay in cardiac conduction and inappropriate vasodepression. Avoidance of carbonated fluids or other agents associated with symptoms and behavioral modification to change eating habits may be successful in patients with infrequent episodes of syncope (7). Various anti-cholinergic medications like atropine, scopolamine and propantheline have been used to prevent bradyarrhythmias by blocking vagal conduction. Sympathomimetic agents like adrenaline and isoprenaline have been used to increase the ventricular rate directly. However the results have been inconsistent and limited by side effects [2]. Beta-blockers may be effective in carefully selected patients (8). In the appropriate clinical setting correction of any underlying esophageal pathology should be attempted. Permanent pacemaker placement is usually effective in patients in whom bradyarrhythmias are the major cause of syncope.

Because swallow syncope is a rare disease, there is no randomized controlled trial to compare the efficacy of different treatment remedies such as medications, surgery or pacemakers. However, it is logical to treat the underlying diseases of swallow syncope if they can be found and are treatable. For example, gastroesophageal reflux disease can be treated with proton pump inhibitors; hiatal hernia can be treated with surgery. In the large majority of cases of swallow syncope there is an associated reflex bradycardia leading to cerebral hypoperfusion and syncope. Of the previously reported 80 cases of swallow syncope, 68 cases (85%) including many underlying causes as digestive diseases or unknown cause were found to have sinus arrest, SA block, sinus bradycardia or AV block. Therefore, placement of a pacemaker represents a reasonable treatment option which allows for over-riding of the bradycardia, although it does not treat the cause of the problem. All 11 cases listed in the table 1 who had pacemaker implantation had resolution of symptoms over 2 to 26 months of follow up, indicating that pacemakers are an effective treatment. However, pacemaker implantation is not without risks that need to be balanced with the potential benefits. Approximately 4-5% of patients will have a complication during or following pacemaker placement, such as infection, lead dislodgment, pneumothorax and cardiac perforations (21). There were cases where pacemakers failed to resolve the symptoms. Armstrong (22) reported a case of swallow syncope who had an esophageal web, moderate hiatal hernia and spastic narrowing of the esophagus but no bradycardia was documented. Pacemaker placement was unsuccessful in this case and the patient died of congestive heart failure 2 years later. It is important that a definitive diagnosis of cardiac rhythm disturbance is established prior to insertion of a permanent cardiac pacemaker (22). In patients who do not respond to pacing therapy, volume repletion, increased salt intake, leg stockings and avoiding ingestion of specific beverages or behavior associated with symptoms (22) may be of value, but no systemic study on this patient population was reported. A careful search for
underlying causes not involving bradycardia may be warranted. For instance, the left atrial compression should be ruled out by echocardiography (23). A large hiatal hernia can cause extrinsic compression of the left atrium during meals and impede cardiac output leading to hypotension and syncope. These patients can get relief of their symptoms by surgical repair of the hernia (23, 24). A pacemaker would not have provided any benefit as bradycardia is not involved.

Swallow syncope, though generally considered a benign condition, can cause significant impairment of quality of life and can result in significant injury if it occurs at certain times like driving or operating heavy machinery. However, it is a treatable disease. A thorough history including asking about any relationships between eating and syncope or lightheadedness is important for the diagnosis of swallow syncope.

References

Author Affiliations
Subhashis Mitra, MD†; Tiffany Ludka, MD‡; Shereif H. Rezkalla, MD‡, Param P. Sharma, MD‡; Jiangming Luo, MD, PhD*
*Department of Hospital Medicine, Marshfield Clinic, Marshfield, Wisconsin, USA; Email: luo.jiangming@marshfieldclinic.org
†Department of Internal Medicine, Marshfield Clinic, Marshfield, Wisconsin, USA
‡Department of Cardiology, Marshfield Clinic, Marshfield, Wisconsin, USA
†Current affiliation: Fellow, Division of Infectious Diseases, Department of Medicine Wayne State University School of Medicine, Detroit, Michigan, USA