

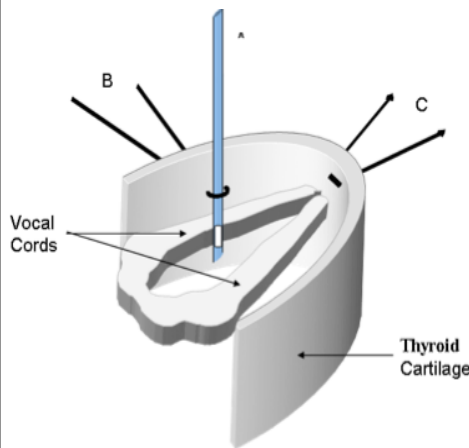


The Yale Larynx Laboratory

A Clinical Review

Zenker's Diverticulum: Toward a Unified Understanding of its Etiopathogenesis

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Laboratory Note

The purpose of this newsletter is to update our readers with the evidence-based management of certain Head & Neck disease presentations. In this issue we shall focus on a bilobed Zenker's diverticulum.

The Yale Larynx Laboratory was founded by John A. Kirchner in 1967. Since 1975 this laboratory has been in continuous operation under the direction of Clarence T. Sasaki, the Charles W. Ohse Professor and has been funded by the National Institutes of Health and by endowments of grateful patients.

Case Presentation

A 78-year-old man presented with several months of difficulty swallowing. His symptoms were worse with solid foods compared to liquids, and he also noted an increased production of "phlegm." He had no significant past medical history.

Clinical findings

On physical examination, he had a strong voice and his oral cavity was clear. Fiberoptic laryngoscopy revealed pooling of secretions in his vallecula and pyriform sinuses, but normal appearing laryngeal anatomy. A complete metabolic panel, complete blood cell count, and coagulation studies were all within normal limits. A barium swallow study was performed by a speech and language pathologist (Fig.1).

Course

The barium esophagram demonstrated a moderately enlarged, laterally displaced diverticulum of a highly unusual shape, as well as a small hiatal hernia. The patient was taken to the operating room and provided general anesthesia with orotracheal intubation. An esophagoscope was advanced to expose the upper esophageal sphincter where a Zenker's diverticulum (ZD) with a midline septum was visualized (Fig.1a, 3a). A wire guide was passed through the upper esophageal sphincter and serial dilations were performed to dilate the sphincter. The vertical septum within the Zenker's pouch was horizontally divided using a CO2 laser at 7 watts (Fig. 3b). This was followed by a vertical, transmucosal division of the CPM, opening the fundus of the diverticulum followed by insertion of a nasogastric tube (Fig. 3c). A month later the patient was noted to be tolerating solid foods without "phlegm" or regurgitation.

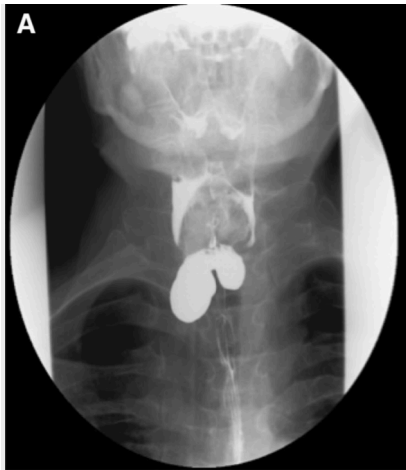


Figure 1a: Barium swallow:
Anterior-posterior view of a
bilobed diverticulum



Figure 1b: Barium swallow:
Lateral view of bilobed Zenker's

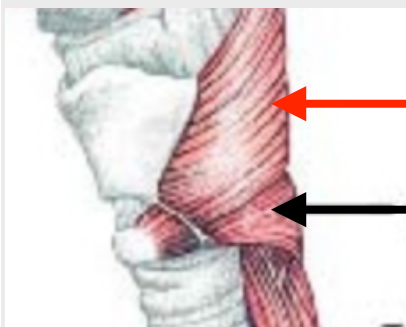


Figure 2: Cricopharyngeus
muscle (black arrow), inferior
constrictor muscle (red arrow)

Discussion

Anatomy

The pharyngeal muscle anatomy is such that the cricopharyngeus muscle (CPM) extends dorsally without a median raphe capable of anchoring it to the prevertebral fascia. In contrast, immediately superior to the CPM, the pharyngeal constrictor muscles possess a fibrous midline raphe that anchors them to the skull base superiorly and to the prevertebral fascia vertically (Fig 2). The lack of fixation allows for the CPM to be displaced caudally in relation to the inferior constrictor muscle.

Formation of Zenker's Diverticulum

Although, it has been hypothesized that acidic reflux may contribute to CPM spasm, there is much controversy about the exact effect of acid on the upper esophageal sphincter (UES).¹⁻³ The most prevalent theory for the formation of ZD is an increased CPM resting tone resulting in a pulsion pseudodiverticulum through Killian's triangle above it. However, many individuals with increased sphincter tone never incur a ZD. In addition to well documented hypotheses as to the etio-pathogenic cause of ZD including anatomic weakness and muscular dysfunction of the upper esophageal sphincter (UES), esophageal reflux exposure to local tissue, may play an added role in ZD formation, and could explain its known co-association with hiatal hernia.^{4,5} The exact mechanism by which gastroesophageal reflux (GERD) contributes to the formation of Zenker's Diverticulum is still debatable. According to prevailing thought, episodic gastroesophageal reflux gradually induces changes in the length of esophagus as esophageal exposure to acidic fluids produces permanent scarring and shortening of the esophagus, predisposing a patient suffering from reflux to develop a hiatal hernia secondary to intrathoracic traction-elevation of the stomach.⁵ A similar mechanism may explain a downward force acting on the un-anchored CPM resulting in its inferior displacement in relation to the inferior constrictor muscle creating a posterior gap deformity above the CPM. Reflux would then account for the development of ZD and serve as an explanatory cofactor in hiatal hernias seen in up to 40% of patients with a pulsion pseudodiverticulum.⁶

Bilobed Zenker's Diverticulum: A Rare Presentation of ZD

Pharyngeal diverticuli may vary in size, shape, number, and position. An interesting phenomenon is the formation of bilobed pseudodiverticulum of the hypopharynx and since 1942, 10 such cases have been described.⁷⁻¹⁴ Those prior to 1990 were managed with external diverticulectomy while more recent cases were treated with endoscopic division and stapling of either the larger lobe or both lobes.¹³ A bilobed pseudodiverticulum would likely originate from a developmental attachment of a midline raphe to the upper fibers of the CPM. Shortening of the esophagus secondary to esophageal reflux coupled with this fibrous connection may account for a bilobed deformity.

A Unified Understanding of the Etiopathogenesis of ZD

An elucidation of the association between Zenker's diverticulum and gastroesophageal reflux disease (GERD) may help understand the pathogenesis of ZD. Acid reflux can induce sub-mucosal injury that may lead to longitudinal esophageal shortening. Given that the CPM is not vertically anchored by a median raphe, it is pulled inferiorly creating a gap between the vertically anchored pharyngeal constrictor muscles and CPM. If a midline fibrous raphe from the inferior constrictor remains developmentally attached to the CPM, a bilobed ZD can ensue. A bilobed diverticulum is not only rare and unusual, but it also may provide rare insight into the etiopathogenesis of ZD.

Key points

- Unlike the inferior constrictor muscle, the CPM has no midline raphe connecting it to the pre-vertebral fascia
- CPM hypertrophy, esophageal shortening secondary to esophageal reflux, and increased esophageal resting tone likely all play a role in ZD formation
- Dividing a bilobed Zenker's septum, using CO₂ laser or other methods, is a safe maneuver to allow for one continuous sac to be opened to the true esophagus.
- This report of an uncommon clinical presentation suggests a multifactorial etiopathogenesis, and a rare opportunity guiding us toward a unified causal understanding of ZD

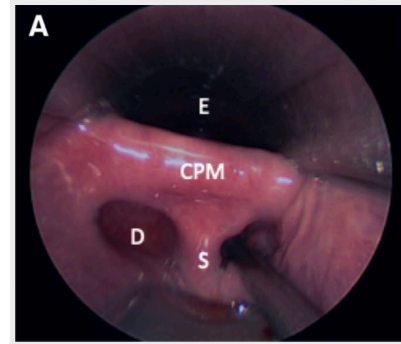


Figure 3a - Endoscopic view of bilobed Zenker's diverticulum.

(E esophagus, CPM cricopharyngeus muscle, D diverticulum, S septum)

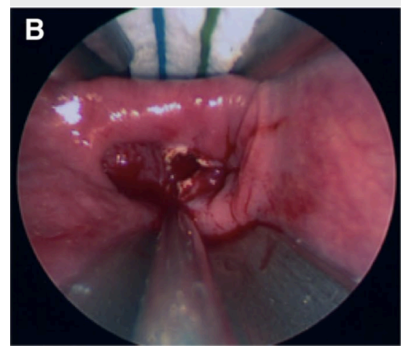


Figure 3b - Bilobed Zenker's diverticulum: Post-midline ablation of CO₂

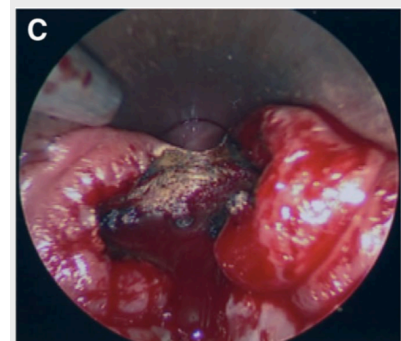


Figure 3c - Transmucosal division of the CPM with opening of the diverticular fundus to esophageal lumen

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