Complete esophageal stenosis secondary to peptic stricture in the cervical esophagus: Case report

Giovana R. Thomas, MD; Tiffany Raynor, MD

Abstract
Complete esophageal stenosis secondary to peptic stricture in the upper esophagus is rare. It is, however, a serious medical problem that may require otolaryngologic intervention because of life-threatening dysphagia and weight loss. We report the case of an elderly patient who presented with an upper esophageal stricture, without the typical symptoms of gastroesophageal reflux disease, that progressed to complete esophageal obstruction despite use of proton pump inhibitors and esophageal dilatation. Definitive management of this difficult problem required esophagectomy and gastric pull-up. We discuss the pathophysiology, clinical presentation, differential diagnosis, and multidisciplinary management of peptic esophageal strictures. This case illustrates the difficulty in managing high peptic strictures.

Introduction
Gastroesophageal reflux disease (GERD) is a common problem, with at least 10% of the U.S. population reporting symptoms of heartburn or acid reflux at least once per week. Head and neck manifestations and complications of GERD are commonly seen in a general otolaryngology practice and may present significant morbidity. Between 40 and 65% of those who suffer from GERD develop erosive esophagitis. If left untreated, reflux esophagitis may lead to peptic esophageal stricture in 4 to 23% of these patients.

Although peptic strictures are common, progression to complete esophageal stenosis in the upper esophagus occurs rarely. We report the case management of an elderly man with complete esophageal stenosis secondary to peptic stricture in the cervical esophagus, which required esophageal resection and gastric pull-up.

Case report
A 65-year-old African American man was referred to the Otolaryngology–Head and Neck Surgery service for evaluation and management of stricture in the upper esophagus. He is a widower, and had worked as a tile installer for 25 years. His symptoms had started 2 years previously with dysphagia to solids, progressing to an inability to eat, drink, or swallow his saliva. He reported losing 70 pounds during that time and required a percutaneous endoscopic gastrostomy for alimentation. He denied heartburn, caustic ingestion/inhalation, history of radiation therapy to the head and neck region, otalgia, shortness of breath, hoarseness, fevers, chills, night sweats, or aspirin use. He consumed alcohol occasionally and denied a history of tobacco use. However, he had a long history of atypical chest pain and epigastric discomfort. His medical history was significant for major depressive disorder with delusions, unresponsive to an array of medications and electroconvulsive therapy. Medications on presentation included mirtazapine (an antidepressant), haloperidol, cyproheptadine (an appetite stimulant), and lansoprazole.

Initial esophagoduodenoscopy (EGD) by gastroenterologists at an outside institution had revealed no significant esophageal pathology. Subsequent EGD evaluations revealed significant reflux esophagitis and ulcerations progressing to esophageal stricture in the upper esophagus. Biopsies in the region of the stricture did not show malignancy. The stricture was managed with dilatations, and lansoprazole 30 mg daily was started. The most recent EGD had been unsuccessful because the scope could not pass beyond the stricture, either from the oropharynx or retrograde through the percutaneous endoscopic gastrostomy.

At the patient’s initial visit to our service, examination showed a thin, debilitated elderly man with normal upper airway and cranial nerve findings. A barium esophagram showed complete obstruction of the upper part of the esophagus (figure 1). On computed tomography of the chest, the thorax showed a dilated cervical esophagus and no obvious mass compressing or involving it (figure 2). Rigid esophagoscopy revealed a blind esophageal pouch with a small granulation polyp 21 cm from the upper...
incisors. Biopsy was consistent with normal squamous mucosa, with inflammatory changes in the epithelium but no malignancy. The site of this obstruction was consistent with the site of the documented esophageal stricture.

Because this was an unusual location for peptic esophageal stricture, we investigated further to exclude other known etiologies for strictures. Autoimmune disorders such as scleroderma and systemic lupus erythematosus were excluded by laboratory studies and clinical examination. Serology results for human immunodeficiency virus were negative. A diagnosis of GERD-related esophageal obstruction was made, and a guide wire was placed with balloon dilatation of the esophagus under fluoroscopic guidance. This was initially successful in creating a small esophageal lumen, but restenosis quickly occurred. A transhiatal esophagectomy with gastric pull-up and feeding jejunostomy was performed. Pathologic examination of the esophagectomy specimen revealed ulcerated mucosa with acute and chronic inflammation and granulation tissue with no evidence of malignancy.

Since surgery, the patient has tolerated oral feedings, has gained significant weight, and has made significant improvement in his depressive disorder.

Discussion
One of the most significant long-standing sequelae of untreated reflux esophagitis from GERD is peptic esophageal stricture. Factors predisposing to stricture formation are not well understood, but studies demonstrate that patients with peptic stricture are older, have reflux symptoms of longer duration, have an associated esophageal motility disorder, and have significantly reduced lower esophageal sphincter pressures. Strictures occur when esophagitis progresses to panmural inflammation. This results in thickening and scarring of the involved segment, which is usually located in the distal esophagus at the squamocolumnar junction.

Other factors besides exposure of the esophageal mucosa to acid likely contribute to the development of erosive esophagitis. Dysphagia is the most common presenting symptom in patients with benign esophageal stricture. A history of typical heartburn symptoms is present in more than 75% of patients with peptic strictures. However, older patients are less likely to report frequent or severe heartburn, perhaps because of decreased gastric acidity and a decline in esophageal sensitivity with age.

The differential diagnosis for esophageal strictures is extensive and includes peptic strictures, which comprise 60 to 70%; esophageal tumors; caustic ingestion; nasogastric tube trauma; scleroderma; radiation therapy; infections (e.g., Candida, tuberculosis); drugs (e.g., doxycycline/tetracycline, potassium chloride, quinidine, vitamin C); Crohn’s disease; pill-induced esophagitis; and bullous dermatoses such as epidermolysis bullosa and pemphigus.

Barium esophagography is the initial diagnostic procedure for patients with GERD who present with dysphagia because it has greater sensitivity (95%) than endoscopy in detecting strictures. Biopsies should be taken at endoscopy and should include tissue both from proximal ulcerated areas and from the depths of the stricture. In the absence of a clear history of GERD, serologic testing to exclude autoimmune etiologies is recommended. Serum gastrin levels and salicylate levels are useful in detecting Zollinger-Ellison syndrome and surreptitious use of aspirin. Manometry is a useful adjunct if a motility disorder is suspected. PH monitoring may be helpful if the etiology of the stricture remains unclear. For example, pill-induced strictures may be differentiated from peptic strictures by...
esophageal pH studies. In our elderly patient, additional factors besides GERD—such as the use of medications that may decrease lower esophageal sphincter pressure, disturbance in esophageal motility from aging, and absence of typical symptoms of heartburn that delayed adequate diagnosis—likely contributed to esophageal stenosis. Oral medications that may reduce lower esophageal sphincter pressure include groups of drugs called nitrates (e.g., isosorbide dinitrate), calcium-channel blockers, diazepam, barbiturates, nonsteroidal anti-inflammatory drugs, theophylline, and nicotine. Since, prior to his visit to our service, the patient was tried on multiple medications for depression, it is uncertain which medication, if any, might have played a part in GERD and stenosis formation.

The mainstay of treatment for peptic strictures has traditionally been mechanical dilatation. Further medical management includes use of H₂-receptor blockers, proton pump inhibitors, and intralesional steroid injections. Studies have shown that aggressive acid suppression with proton pump inhibitors decreases the frequency of peptic stricture dilatation.

Esophageal resection with reconstruction for peptic strictures is reserved for strictures that cannot be adequately dilated, rapid recurrence of a stricture after dilatation, or an irreversibly damaged esophagus with transmural fibrosis and evidence of aperistalsis on manometry. Reconstruction options include segmental resection and esophagogastrotomyortalesophagectomy with jejunal or colon interposition.

In summary, physicians must keep a high index of suspicion for peptic strictures in patients with complaints of persistent dysphagia even in the absence of typical symptoms of GERD. A multidisciplinary treatment team that consists of the otolaryngologist, gastroenterologist, and swallowing therapist should manage these patients once the diagnosis has been established. Early medical treatment with proton pump inhibitors, especially in elderly patients who have other predisposing factors, should be implemented to prevent stricture progression to complete stenosis.

References