Esophageal diverticulosis associated with Barrett’s esophagus and erosive esophagitis

A 62-year-old man with a history of symptomatic gastroesophageal reflux disease presented to our clinic with long-standing solid food dysphagia. Physical examination and laboratory values were unremarkable. Esophagogastroduodenoscopy (EGD) was performed and showed multiple mid-esophageal diverticula associated with erosive esophagitis and mild stenosis of the mid-esophagus, 32–34 cm from the incisors (Fig. 1, Video 1). Mucosal biopsies demonstrated Barrett’s esophagus without dysplasia, and cytology brushings were negative for malignancy. A barium esophagram revealed narrowing at the mid-esophagus, proximal dilation, and multiple sinus tracts without mucosal irregularities (Fig. 2). Endoscopic ultrasound (EUS) revealed an intact, thickened muscularis propria of the mid-esophagus and benign-appearing lymph nodes (Fig. 3). The patient was initiated on proton pump inhibitor therapy with complete symptom resolution. Mucosal healing with persistence of mid-esophageal diverticulosis was demonstrated on several follow-up EGDs (Fig. 4), and no dysplasia was detected in repeated mucosal biopsies. Esophageal diverticula are outpouchings of one or more layers of the intestinal wall that may occur at any level of the esophagus [1, 2]. Epidemiological data are sparse, though Zenker’s diverticulum is the most common with a prevalence of 0.01%–0.11%. The Rokitansky classification defines traction diverticula as those that result from a chronic inflammatory process in the mediastinum and involve the entire esophageal wall, whereas pulsion diverticula result from high intraluminal pressures from obstructions against weaknesses in the gastrointestinal tract wall. A distinct condition that rarely presents with stricture formation of the esophagus is esophageal intramural pseudodiverticulosis [3, 4]. This was examined by EUS and narrow-band imaging in previous case reports [4, 5]. In the current patient, mid-esophageal pulsion diverticula were diagnosed secondary to Barrett’s esophagus and peptic stricture. We hypothesize that relative obstruction caused by the Barrett’s esophagus led to increased luminal pressure, causing esophageal diverticulosis.
Competing interests

None

The authors

Supriya Rao1,2, Ashish Sharma1, H. Christian Weber1,2
1 Section of Gastroenterology, VA Boston Healthcare System, Boston, Massachusetts, United States
2 Section of Gastroenterology, Boston University School of Medicine, Boston, Massachusetts, United States

Corresponding author

H. Christian Weber, MD
Medicine Service, Section of Gastroenterology, Room 6A-46, VA Boston Healthcare System, 150 South Huntington Ave, Jamaica Plain, MA 02130, United States
Fax: +1-857-364-4179
christian.weber@va.gov

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