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A 69-year-old woman with an unusual case of dysphagia

To the Editor,

Esophagitis dissecans, also known as sloughing esophagitis, is a rare, but benign, endoscopic finding. The typical endoscopic appearance is represented by sloughing of the mucosal layer. Rarely, patients develop esophageal casts that can obliterate the esophageal lumen and cause obstructive symptoms (1).

Esophagitis dissecans is most often idiopathic, but it can be associated with use of nonsteroidal anti-inflammatory drugs or bisphosphonates, consumption of hot beverages, celiac disease, or autoimmune bullous dermatoses (2). Histological features include the following: parakeratosis, intraepithelial splitting at varying degrees above the basal layer, occasional association with intraepithelial bullae and minimal or no inflammatory component, and unusually long, detached fragments of superficial epithelium (3).

Here we report the case of a 69-year-old female who presented to our observation department with a 2-year history of progressive oropharyngeal dysphagia exacerbated by solid food ingestion. Her medical history included the following: tonsillectomy, appendectomy, and episodic exacerbations of allergic dermatitis treated with corticosteroids. She smoked 10–12 cigarettes a day; alcohol consumption was not reported. She did not receive any chronic medication.

The patient initially underwent videofluoroscopic swallowing study, which did not identify a significant alteration. Esophagogastroduodenoscopy was then performed, revealing a tight stricture of the proximal esophagus that has been crossed only by an ultraslim transnasal gastroscope (Fujifilm EG-530NP) with the presence of a thin white membrane lifting from the underlying mucosa, which appears normal. The mid-distal esophagus, stomach, and duodenum were normal (Figure1).

Suspecting a microperforation of the esophagus, biopsies were not performed; instead, a computed tomography (CT) scan was performed. The CT scan showed circumferential wall thickening and was supported by thickening of the submucosa and with evidence of mucosal enhancement (Figure 2). The patient was treated with a high-dose proton pump inhibitor and showed progressive clinical improvement. After two months,

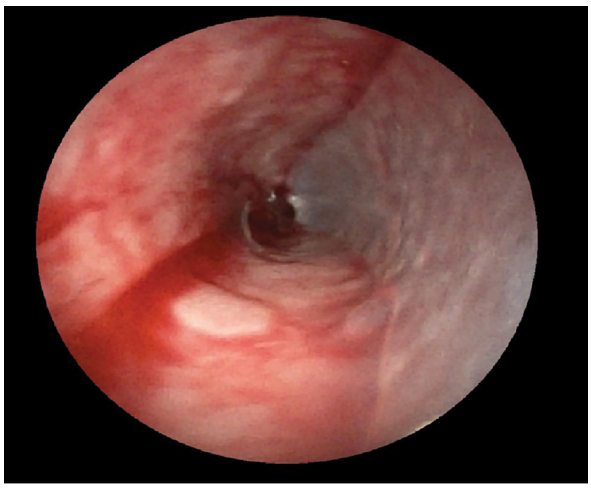

Figure 1. Intraluminal view of the patient's esophagus during gastroscopy shows the presence of a thin white membrane lifting from the underlying mucosa, which appears normal.

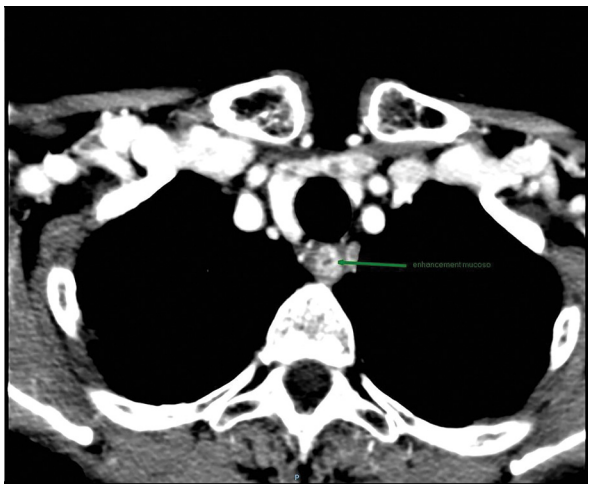

Figure 2. Axial CT scan shows circunferential wall thickening, supported by thickening of the submucosa and with evidence of mucosal enhancement.

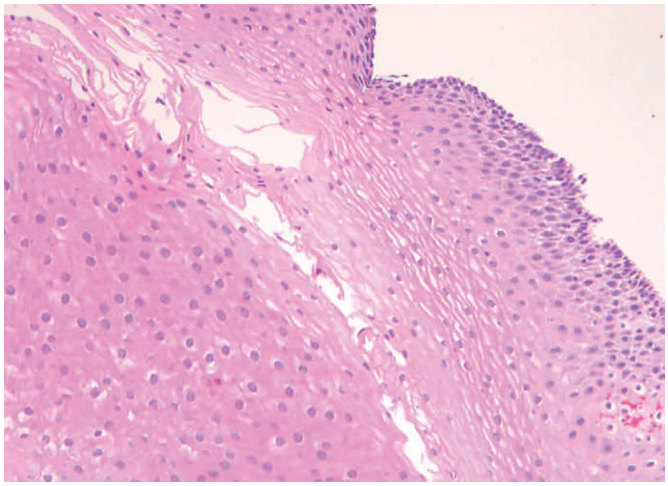

Figure 3. Histology of the esophagus shows edema and hyperplasia of the basal layer and splitting of the squamous mucosa at the suprabasal and superficial layers.

the endoscopic follow-up revealed a significant reduction in proximal esophageal strictures and the disappearance of macroscopic mucosal alterations. Multiple biopsy samples were taken during endoscopy.

The biopsies showed edema and hyperplasia of the basal layer and splitting of the squamous mucosa at the suprabasal and superficial layers, which resulted in the diagnosis of esophagitis dissecans being made (Figure 3). Our patient had no medical history of drug use and known risk factors for esophagitis dissecans, and the excellent response to the proton pump inhibitor suggests that in this case, the endoscopic and histological diagnosis is an epiphenomenon of gastroesophageal reflux. To our knowledge, this is the second case, after the one described by De Shumona et al. (4), where a patient experienced complete response with a proton pump inhibitor.

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