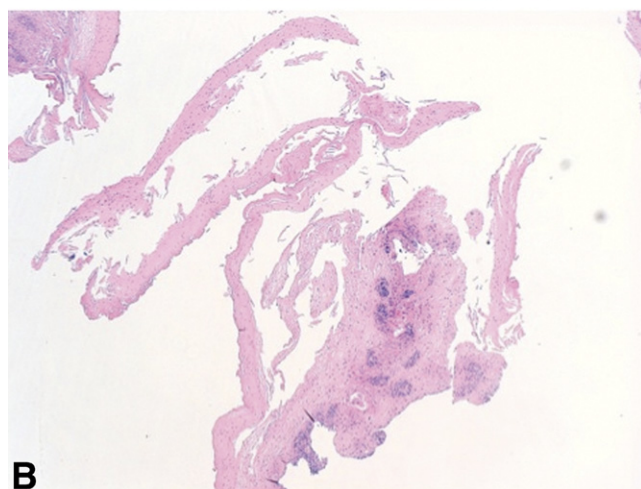


Lawrence J. Brandt, MD, *Associate Editor for Focal Points*

Esophagitis dissecans superficialis



A 59-year-old woman was evaluated for dysphagia and odynophagia. She had undergone double lung transplantation 3 months earlier for hypersensitivity pneumonitis complicated by respiratory failure. At the time of endoscopy, her immunosuppressive medications consisted of tacrolimus, azathioprine, and prednisone.

In addition to her immunosuppression regimen, she was taking an oral bisphosphonate (risedronate).

Before transplant, she experienced long-standing, mild reflux symptoms, but after transplant she developed in-

creasing dysphagia and odynophagia. Physical examination revealed no oral lesions or thrush. A barium esophagram revealed tertiary contractions in the distal esophagus with pill retention. Endoscopy showed sloughing whitish membranes that were easily removed, adjacent to intact healthy mucosa (A). The appearance was characteristic of esophagitis dissecans superficialis. Histopathologic evaluation revealed parakeratosis and desquamation of the epithelial layer (B), features that are characteristic of esophagitis dissecans superficialis (H&E, orig. mag.

×40). The patient improved with discontinuance of the bisphosphonate.

DISCLOSURE

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Commentary

Esophagitis dissecans superficialis (EDS) is the term coined by Rosenberg in 1892 that describes the endoscopic finding characterized by sloughing of large fragments of the esophageal mucosa; such sloughed squamous mucosa may be coughed up or vomited out as a cast of the esophagus. Although EDS has been reported in association with certain medications (bisphosphonates, nonsteroidal anti-inflammatory drugs, potassium chloride), hot beverages, chemical irritants, celiac disease, collagen vascular disorders, and autoimmune bullous dermatoses (pemphigus and pemphigoid), pathogenesis in most cases remains unexplained. The Italian word *dissecate* derives from the Latin *dissecare* (to dissect) and is not to be confused with a similar-looking word, *desiccate*, meaning to dry. Endoscopic features of EDS include stripped-off mucosa with or without bleeding, long linear mucosal breaks, vertical fissures, and circumferential cracks. Biopsies from patients with confirmed EDS show sloughing and flaking of superficial squamous epithelium with occasional bullous separation of the layers, parakeratosis, and varying degrees of acute or chronic inflammation; fungal elements may be associated. In spite of its sometimes-dramatic presentation, EDS usually is a benign condition that resolves without lasting esophageal pathology. Endoscopy is an important diagnostic tool and can help differentiate EDS from candidal, herpetic, and peptic esophagitis, all of which can have similar symptoms in patients who frequently are receiving glucocorticoids or immunosuppressive agents. One can remove the uplifted mucosa without concern but can cause a Nickolsky sign by biopsying or brushing against what appears to be intact mucosa. No matter, send the specimen to pathology for diagnosis. Also, please make sure to keep your EDS patients well-hydrated lest they become desiccated, because the discharge form listing these two conditions would be uncorrectable and would haunt your administrative task list for decades.

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Melena from jejunal mucosal varices caused by esophageal variceal sclerotherapy-induced splenic arteriovenous fistula

A 64-year-old man with a history of cirrhosis presented with left upper quadrant pain, severe diarrhea, and ascites, 3 months after uncomplicated endoscopic injection sclerotherapy with 5% ethanolamine oleate for esophagogastric variceal bleeding. Contrast-enhanced CT (**A**) demonstrated a splenic infarction (*yellow arrow*), a splenic arteriovenous fistula, and thrombosis of the portal vein, splenic vein, and superior mesenteric veins (*yellow arrowheads*). The portal vein became completely obstructed despite anticoagulation with danaparoid for 2 weeks and urokinase for 4 weeks after thrombolysis. Episodic melena then appeared, which required a transfusion every several weeks. Although EGD demonstrated neither esophagogastric varices nor peptic ulcers, videocapsule endoscopy (**B**) and double-balloon enteroscopy (**C**) revealed innumerable jejunal varices. We were surprised that splanchnic angiography (**D**) showed that portal vein pressure was 52 mm Hg (normal 7.4-11.1 mm Hg) by

a transducer inserted through an arteriovenous fistula (*red arrow*) from the splenic artery (*red arrowhead*) into the splenic vein (*blue arrowhead*). After we placed microcoils in the splenic arteriovenous fistula (**E**), the ascites and diarrhea gradually decreased. Videocapsule endoscopy 3 months after coiling of the fistula showed that the jejunal mucosal varices had disappeared. Contrast-enhanced CT before coiling (**F**) showed no cavernous transformation despite portal vein thrombosis, but repeat study after coiling (**G**) revealed cavernous transformation around the portal vein (*green arrowheads*). The patient was discharged and returned to his job after a 1-year interval.

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