

Denudation of the esophageal mucosa following ingestion of a foreign body and vinegar

Esophagitis dissecans superficialis (EDS) is a rare condition that involves complete sloughing of the squamous esophageal mucosa to produce a benign esophageal cast; it is most commonly associated with systemic bullous pemphigoid [1,2]. EDS is distinct from diseases associated with pseudomembranous esophagitis and has also been described in previously healthy people who have experienced trauma to the esophagus [3,4]. Esophageal injury from various caustic agents is well described in the medical literature, however, there have been only two case reports of household vinegar as a cause [4,5]. One pattern of injury resulting from ingestion of a corrosive agent that has been described is esophageal mucosal cast formation with complete separation of the squamous mucosa [6]. Here we report a characteristic presentation of EDS following ingestion of a foreign body and vinegar, which to our knowledge has never been described.

A 28-year-old Chinese woman, who was 7 months pregnant, presented to the emergency department with a foreign body sensation in her throat. She first developed the sensation 12 hours earlier during a meal that consisted of fish with numerous small bones. In line with a Chinese folk remedy, the patient had ingested several tablespoons of vinegar with the intention of “softening” the bone. She was otherwise in good health and there were no pertinent findings on examination. A soft tissue neck radiograph was taken but it did not show any evidence of a foreign body. The patient underwent a laryngoscopy, which was also unremarkable. Upper endoscopy (esophagogastroduodenoscopy [EGD]) was carried out and revealed concentric and complete denudation of the mucosa involving the entire length of the esophagus (● Fig. 1). A 3.5-cm collection of clotted blood was found in the proximal stomach, “wrapped” in a piece of membranous, gray-white tissue (● Fig. 2). The clot was removed and on separating the tissue from the clot, a 15-cm long tube was recovered (● Fig. 3) and submitted for pathological evaluation. The histological appearance of the specimen was consistent with non-keratinized squamous epithelium with no

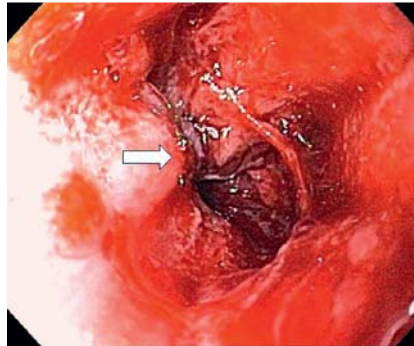


Fig. 1 Endoscopic image of distal esophagus and gastroesophageal junction (GEJ) showing absence of mucosa in the esophagus proximal to the GEJ. The arrow indicates the point of “tethering” of the esophageal mucosa to the GEJ.

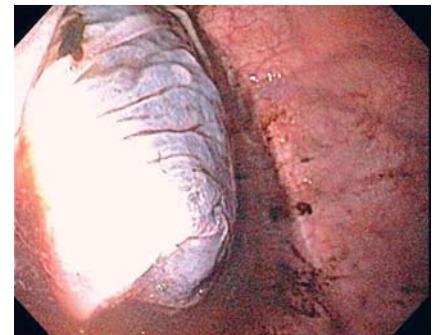


Fig. 2 A 3.5-cm aggregate of clotted blood in the proximal stomach, “wrapped” in a single piece of membranous appearing, gray-white tissue.

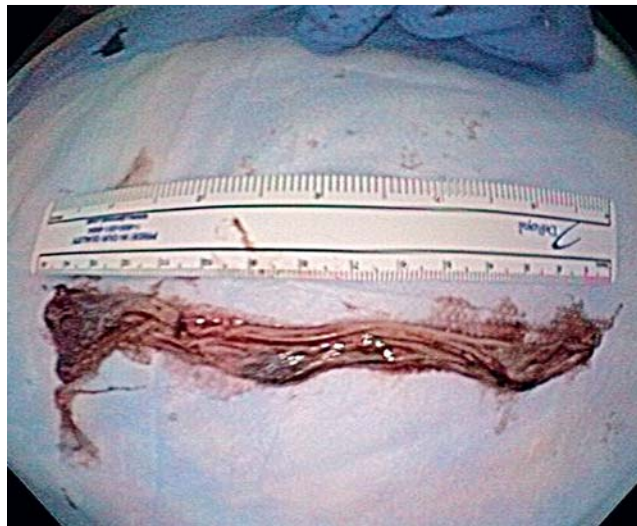


Fig. 3 The membranous appearing gray-white tissue (esophageal mucosa) measured 15 cm in length.

mucosal ulceration or lesions. A barium swallow was performed, which did not reveal any evidence of esophageal perforation. The patient was discharged on day 2; she was in a stable condition with no immediate post-episode complication. At 16-month follow-up the patient reported being asymptomatic and there was no evidence of any long-term adverse events.

We hypothesize that the initial mechanism of injury in the present case was cricopharyngeal mucosal injury caused by swallowing a fish bone. The mucosal tear was then propagated by several factors: acetic acid ingestion, significant clot formation in the submucosal layer, circum-

ferential tearing of the esophageal mucosa and the persistent peristaltic action of the esophagus, which gradually denuded the mucosa of the esophagus as the cast was propelled into the gastric fundus. Acid ingestion typically produces a superficial coagulation necrosis that causes thrombosis of the underlying mucosal blood vessels and consolidates the connective tissue, thereby forming a protective eschar [7]. To our knowledge there are only two reports in the literature describing esophageal mucosal injury caused by vinegar [4,5]. It is not known if direct exposure of the submucosal layer to diluted acetic acid (because of a preceding esophageal tear) would precipitate a more

serious reaction, but theoretically this remains a possibility.

Although pregnancy can affect esophageal motility and some connective tissues, there is no known effect on mucosal strength, integrity and adhesion to the underlying layers of the esophageal wall. Kaplan et al report a case of an esophageal cast that resulted from tissue sloughing secondary to pemphigus vulgaris as proved by immunofluorescence [3]. Several other authors have reported EDS secondary to a variety of causes, including: bisphosphonate treatment; celiac disease; chronic esophagitis; alendronate and renal failure; ingestion of hot, spicy food; nasogastric tube insertion; and infection [1,2,8–12].

A chronic form of EDS termed chronic esophagitis dissecans (CED) is characterized by chronic dysphagia, shedding of mucosal fragments, localized esophageal strictures, and a lack of concurrent chronic skin and oral lesions [13]. It has been proposed that the underlying disorder involves cell–cell adhesion and cytoskeleton components [13]. However, it is not known whether patients with EDS may also have disorders of cytoskeleton components or structural and/or functional alterations of cell–cell adhesion molecules that would predispose to esophageal cast formation secondary to an acute episode of mucosal trauma or caustic agent ingestion.

In conclusion, we propose that the multifactorial injuries to the esophageal mucosa in the present case were caused by di-

rect trauma and ingestion of a mild caustic agent, resulting in a clinical diagnosis of EDS. We postulate that a seemingly harmless household product could have a role in further propagating esophageal mucosal injury. Our report highlights another potential etiological agent of EDS and demonstrates subsequent spontaneous and complete recovery without long-term complications.

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