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CASE REPORT | ESOPHAGUS

Esophagitis Dissecans Superficialis After Thermal Injury

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ABSTRACT

Esophagitis dissecans superficialis (EDS) is a rare esophageal condition characterized by sloughing of the esophageal mucosal epithelium, typically associated with a desquamating dermatologic disorder or mucosal irritants. We present a case of a 49-year-old man who presented for thermal burns sustained from an outdoor heater explosion. On body trauma imaging, he was incidentally found to have a midcervical esophageal prominence causing asymptomatic posterior tracheal compression. Endoscopy with esophageal biopsy were performed and consistent with EDS. He never exhibited symptoms associated with this diagnosis. To our knowledge, thermal injury has been a purported, albeit without case reference, etiology of EDS.

KEYWORDS: esophagitis dissecans superficialis; sloughing esophagitis; thermal injury; pseudomembranes; endoscopy

INTRODUCTION

Esophagitis dissecans superficialis (EDS), also known as sloughing esophagitis, is a condition of undetermined significance and pathogenesis in which sloughing of the esophageal mucosal epithelium occurs. This esophageal lesion has been reported in association with desquamating dermatological disorders, physical trauma, chemical irritants, anticoagulants, and immunotherapies.[1–7] Furthermore, EDS has been observed more frequently in debilitated patients.[8] Endoscopic presentation of EDS typically involves whitish strips of easily detachable esophageal mucosa underlined by normal-appearing mucosa. Histologically, detached superficial epithelia, parakeratosis, and sometimes necrotic epithelial fragments, mild inflammation, and bacterial and fungal colonies are seen.[9] We present a case of sloughing esophagitis found after thermal injury. It describes another mechanism from which EDS may be precipitated.

CASE REPORT

A 49-year-old man with phenylketonuria presented to the emergency department after an outdoor heater exploded in front of him, catching his shirt on fire. Physical examination showed second-degree burns on 13% body surface area distributed among his face, torso, and bilateral hands. His face had singed eyebrows, lashes, and nares. There was oropharyngeal erythema without soot. He remained in no acute distress with scheduled analgesics. From the time of presentation, he remained hemodynamically stable and maintained normal oxygen saturation on room air. Venous blood gas values were within normal limits, and chest x-ray did not show acute airspace disease or pulmonary edema.

Body trauma computed tomography imaging incidentally noted a soft tissue prominence of the midcervical esophagus measuring 3.3 × 1.8 cm causing posterior compression of the trachea at the level of C7. There was also a chronic appearing anterior deformity of the tracheal cartilage. Combined, these findings caused approximately 75% stenosis of the airway. A nothing by mouth diet was maintained pending further workup. Differential diagnosis included sequelae of prior trauma, diverticulum, and esophageal malignancy.

Endoscopy was performed 2 days after admission, after preprocedural intubation given concern for airway compromise, and showed esophageal mucosal changes consistent with sloughing esophagitis, erythematous stomach mucosa, and erosive gastropathy without

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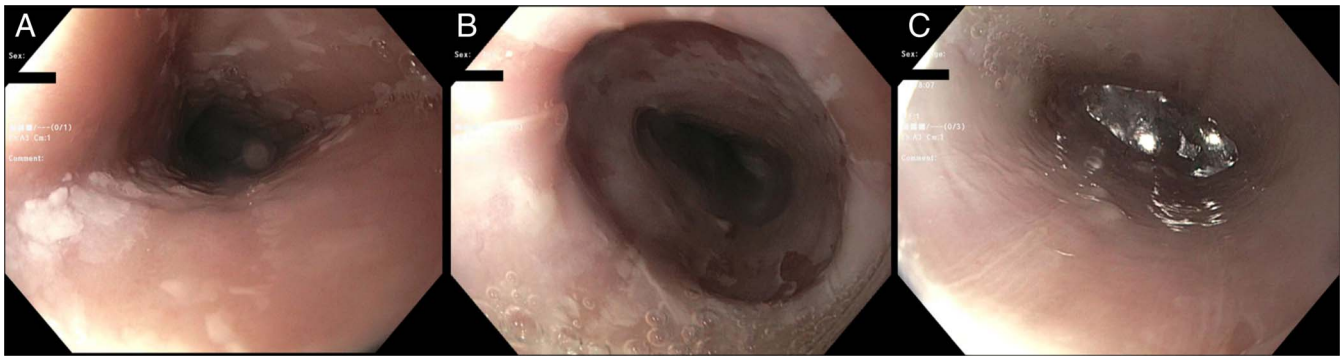

Figure 1. (A) Upper third of esophagus with patches of whitish mucosa. (B) Middle third of esophagus with peeling strips of this mucosa with underlying normal-appearing epithelium. (C) Lower third of esophagus with relatively uniform-appearing mucosa.

signs of recent bleeding (Figure 1). Esophageal biopsy confirmed sloughing esophagitis (Figure 2). No esophageal mass was visualized. The patient was started on a once-daily oral proton pump inhibitor and enteral nutrition through Dobhoff tube. Modified barium swallow study performed on day 4 of admission was significant for mild oropharyngeal dysphagia and esophageal retention of air, contrast, and a 13 mm barium tablet between C5 and C7. Diet was then advanced to regular solids and thin liquids, which the patient tolerated without issue.

Magnetic resonance imaging of the neck performed approximately 1 week later showed persistent circumferential wall thickening of the midcervical esophagus measuring up to 6 mm with well-maintained mucosal lining and submucosa with unchanged tracheal luminal narrowing. The patient remained without respiratory or gastrointestinal signs or symptoms from this time until outpatient burn clinic follow-up approximately 2 months after admission. We decided not to perform repeat endoscopy because he had no esophageal symptoms.

DISCUSSION

Sloughing esophagitis is a rare condition that endoscopists should be familiar with. We present a patient who underwent endoscopy to investigate a midcervical esophageal mass seen on imaging and was found to have EDS. Given the preceding burn injuries involving his face and oropharyngeal erythema on examination, which likely extended to thermal esophageal injury, it is possible that this was a contributor or precipitant of EDS. This mechanism is proposed, given prior associations of this condition with other known esophageal irritants, such as nonsteroidal anti-inflammatory drugs, bisphosphonates, and oral iron.[5]

We established the diagnosis of EDS rather than acute esophageal thermal injury (ETI) based on the symptoms, endoscopy findings, and biopsy histology observed in this case. Our patient was asymptomatic, while ETI is typically associated with dysphagia, odynophagia, and chest pain.[10] Pseudomembranes are often present in both conditions. However, neither ulcers nor mucosal friability were found in this case, while they are commonly seen with ETI.[10,11] Histologically, we observed parakeratosis, which is nearly universal with EDS, rather than anucleate epithelial mucosa seen in ETI cases.[10,12] Perhaps, the severity of ETI is the differentiating factor in the development of EDS vs ETI.

Our patient had several other risk factors that have been associated with EDS. His classification as debilitated has a significant association with EDS.[8] He had a baseline inability to work and lived in an assisted living facility. These debilitation criteria

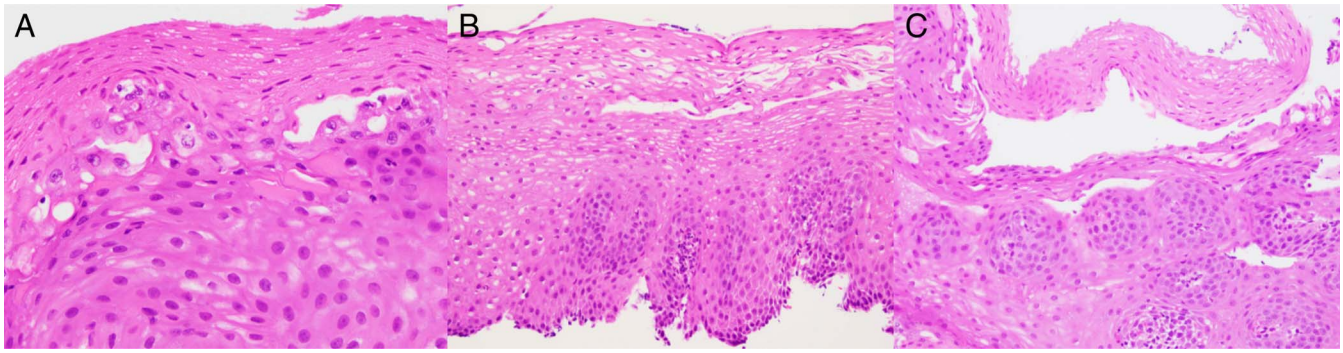

Figure 2. (A) Superficial layers of parakeratosis with early separation from underlying squamous epithelium (hematoxylin and eosin, 400×). (B). Extension of superficial separation, note the lack of inflammatory infiltrate (hematoxylin and eosin, 200×). (C) Complete separation of parakeratotic layer from subjacent normal epithelium (hematoxylin and eosin, 200×).

were expanded by his hospitalization and bedridden status secondary to acute burn injuries. In addition, the use of psychoactive agents, specifically selective serotonin reuptake inhibitors or serotonin and norepinephrine reuptake inhibitors, preceding diagnosis has commonly been reported in patients diagnosed with EDS, although there has not yet been a reported mechanism for this.[8] Our patient's medication list included fluoxetine and trazodone. Finally, it is possible that EDS resulted from the systemic effects of his recent skin trauma. Burn wounds are known to cause a systemic inflammatory response, which has been proposed to contribute to the loss of mucosal integrity and intestinal permeability through inflammatory mediators, such as cytokines and chemokines.[13]

Diagnosis of EDS is of clinical significance. Hart et al found that this condition was resolved in approximately 86% of affected patients at endoscopic follow-up.[12] These authors proposed clinicopathologic diagnostic criteria based on retrospective case series review, which include esophageal mucosal strips greater than 2 cm in length, normal underlying mucosa, and lack of ulcers and friability of immediately adjacent mucosa, all of which were present in our case. Further research into this condition is warranted as EDS can be misdiagnosed, leading to unnecessary treatment. Different diseases can have similar endoscopic appearances to EDS, such as lichen planus, candida esophagitis, squamous cell carcinoma in situ, and verrucous squamous cell carcinoma of the esophagus. This highlights the importance of clinical presentation and histology for supportive or negative evidence.

DISCLOSURES

Author contributions: A. Ahmed and A. Sharma: conception and design, acquisition of data, analysis and interpretation of data, drafting and reviewing the work. W. King and CE Middleton: acquisition of data, analysis and interpretation of data, drafting and reviewing the work. A. Ahmed is the article guarantor.

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