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Acute necrotizing esophagitis

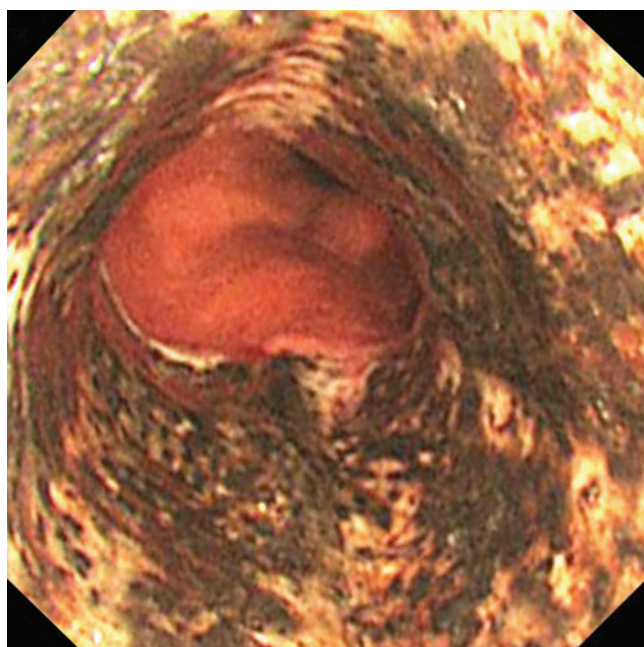


Figure 1. Upper endoscopy on the day of admission showed diffuse black discoloration of the esophageal mucosa affecting the distal esophagus and stopping abruptly at the gastroesophageal junction.

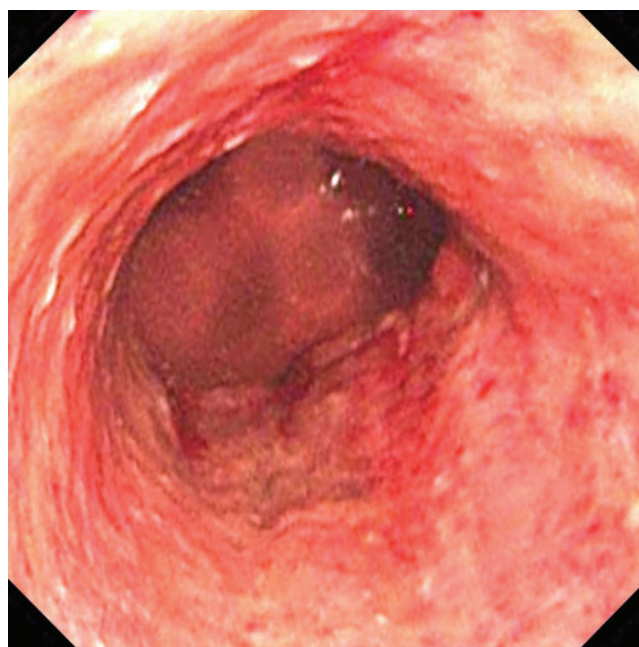


Figure 2. Upper endoscopy repeated 14 days after admission showed improvement of the mucosal lesions and esophagitis.

AN 82-YEAR-OLD MAN with poorly controlled diabetes mellitus presented to our emergency department with a 1-day history of confusion and coffee-ground emesis.

Blood test results suggested diabetic ketoacidosis. Upper endoscopy showed diffuse black discoloration of the esophageal mucosa that affected the distal esophagus and stopped abruptly at the gastroesophageal junction (**Figure 1**), with normal gastric and duodenal mucosa.

Biopsy study revealed necrosis of the esophageal mucosa. A diagnosis of acute necrotizing esophagitis was made.

The patient received nothing by mouth, and he was treated with intravenous fluids, insulin, and a proton pump inhibitor. His symptoms re-

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solved, and upper endoscopy repeated on hospital day 14 demonstrated improvement of the mucosal lesions and esophagitis (**Figure 2**).

■ ACUTE NECROTIZING ESOPHAGITIS

Acute necrotizing esophagitis is thought to arise from a combination of an ischemic insult to the esophagus, an impaired mucosal barrier system, and a backflow injury from chemical contents of gastric secretions.¹ The tissue hypoperfusion derives from vasculopathy, hypotension, or malnutrition. It is associated with diabetes mellitus, diabetic ketoacidosis, lactic acidosis, alcohol abuse, cirrhosis, renal insufficiency, malignancy, antibiotic use, esophageal infections, and aortic dissection.

The esophagus has a diverse blood supply. The upper esophagus is supplied by the inferior thyroid arteries, the mid-esophagus by the bronchial, proper esophageal, and intercostal arteries, and the distal esophagus by the left gastric and left inferior phrenic arteries.¹

■ KEY FEATURES AND DIAGNOSTIC CLUES

The necrotic changes are prominent in the distal esophagus, which is more susceptible to ischemia and mucosal injury. The characteristic endoscopic finding is a diffuse black esophagus with a sharp transition to normal mucosa at the gastroesophageal junction.

The differential diagnosis includes melanosis, pseudomelanosis, malignant melanoma, acanthosis nigricans, coal dust deposition, caustic ingestion, radiation esophagitis, and infectious esophagitis caused by cytomegalovirus, herpes simplex virus, *Candida albicans*, or *Klebsiella pneumoniae*.²⁻⁴

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■ TREATMENT AND OUTCOME

Avoidance of oral intake and gastric acid suppression with intravenous proton pump inhibitors are recommended to prevent additional injury of the esophageal mucosa.

The condition generally resolves with restored blood flow and treatment of any coexisting illness. However, it may be complicated by perforation (6.8%), mediastinitis (5.7%), or subsequent development of esophageal stricture (10.2%).⁵ Patients with esophageal stricture require endoscopic dilation after mucosal recovery.

The overall risk of death in acute necrotizing esophagitis is high (31.8%) and most often due to the underlying disease, such as sepsis, malignancy, cardiogenic shock, or hypovolemic shock.⁵ The mortality rate directly attributed to complications of acute necrotizing esophagitis is much lower (5.7%).⁵