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Acute esophageal necrosis in association with acute cholecystitis

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Dear Editor,

We report a unique case of acute esophageal necrosis (AEN) in association with perforated acute cholecystitis and secondary Klebsiella pneumoniae bacteremia.

Case report

An 83-year-old male with a history of diabetes mellitus, dyslipidemia, ischemic cardiomyopathy and a recent right hemicolectomy for colon adenocarcinoma presented to the Emergency Department with acute epigastric pain and hematemesis. The patient appeared cachectic and dehydrated. He was hemodynamically stable. Laboratory studies revealed anemia, leukocytosis, hyponatremia and hyperlactatemia (lactate 4.0 mmol/l). Esophagogastroduodenoscopy displayed characteristic features of acute esophageal necrosis (AEN) (Fig. 1A) and an abdominal computerized tomography revealed perforated acute cholecystitis. Percutaneous cholecystostomy was performed and fluid therapy, intravenous pantoprazole and bowel rest were started. Klebsiella pneumoniae was cultured in blood and bile and broad-spectrum antibiotic therapy was administered. Three weeks later, esophagogastroduodenoscopy demonstrated a nearly complete healing of the esophageal mucosa (Fig. 1B).

Discussion
AEN is a rare cause of upper gastrointestinal bleeding, defined endoscopically by diffuse black-appearing distal esophagus with abrupt transition at the gastroesophageal junction and variable proximal extension (1,2). This is usually followed by a healing phase dominated by residual black areas and thick white exudates covering pink friable mucosa (3). It is more common in older males with multiple comorbidities, including diabetes mellitus, hypertension, dyslipidemia, ischemic cardiomyopathy, malnutrition and malignancy. Pathophysiology involves esophageal ischemia, gastroesophageal reflux and impaired mucosal reparative mechanisms in debilitated states (1).

To our knowledge, this is the first case of AEN in association with acute cholecystitis, which is probably a relevant factor in its pathogenesis. One possible pathophysiologic explanation is that local infection and bacterial dissemination resulted in a systemic inflammatory response. This, in a background of significant risk factors for advanced vasculopathy (male gender, advanced age, diabetes mellitus, dyslipidemia and ischemic cardiomyopathy) and a debilitated physical state (malnutrition and recent history of malignancy), resulted in esophageal ischemia and necrosis. AEN has been reported in association with other inflammation-mediated diseases, including acute pancreatitis (1), peritonitis (4) or pneumonia (5), which may be mediated by similar mechanisms.

References
Fig. 1. A. Esophagastroduodenoscopy on admission demonstrating friable, diffuse black-appearing distal esophageal mucosa with an abrupt transition at the gastroesophageal junction. The proximal and middle esophagus were not involved and the gastric and duodenal mucosa were also normal. B. Esophagastroduodenoscopy three weeks after admission. Significant improvement can be seen, with a nearly complete healing of the distal esophageal mucosa. A thick, white exudate is present where it previously appeared black, which is characteristic of the healing phase of acute esophageal necrosis. There are no necrotic areas or signs of active bleeding.