

Acute Esophageal Necrosis Caused by Gallstone Pancreatitis

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INTRODUCTION

Acute esophageal necrosis is a serious, yet relatively rare clinical condition afflicting hospitalized patients. It is most often observed in medically ill elderly males and nearly always presents with upper gastrointestinal hemorrhage.^{1,2,3,4} Endoscopy typically finds black, necrotic mucosa in the distal esophagus with abrupt termination at the gastroesophageal junction.¹ The pathogenesis is unknown, although tissue hypoperfusion and gastric reflux are commonly cited contributing factors.¹ It has been associated with hyperglycemia, hypoproteinemia, hypoxemia, underlying malignancies, dehydration, various infections and antibiotic use.^{2,3,5} Treatment remains primarily supportive and overall mortality is heavily dependent on the underlying comorbid

conditions.^{1,4} While this condition remains uncommon, there are growing numbers of case reports and case series noting its presence. Here we discuss a case of acute esophageal necrosis following gallstone pancreatitis.

Case

A 50 year old man presented in the emergency department following the acute onset of severe, sharp epigastric pain radiating to his back. His symptoms were associated with nausea and non-bloody vomiting. Physical examination was notable for diffuse abdominal tenderness to palpation, worst in the epigastrium. Laboratory results revealed an elevated white blood cell count (25,000/cmm (Normal 4,000-11,000)), elevated lipase 8307 U/L (NI 73-393), a slightly elevated total bilirubin (1.2 mg/dL (NI 0.2-1)), and a normal triglyceride level. A computed tomography (CT) scan of his abdomen and pelvis with oral and intravenous contrast demonstrated diffuse enlargement of the pancreatic head and body with extensive peripancreatic fluid consistent with acute

(continued on page 34)

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A CASE REPORT

(continued from page 32)

pancreatitis. Multiple hyperdense foci were noted within the gallbladder, suggestive of gallstones. No biliary dilation was noted.

The patient was admitted with the diagnosis of acute pancreatitis, thought to be secondary to gallstones, aggressively resuscitated and his pain was managed with hydromorphone.

Over the next two days, his bilirubin rose to 2.5 mg/dL, and an endoscopic retrograde cholangiopancreatography (ERCP) was performed. On examination, a short segment of the proximal esophagus was normal (Figure A), however the mucosa in the remainder of the esophagus was black and necrotic (Figure B). The gastric mucosa was uninvolved with a clear demarcation at the gastroesophageal junction (Figure C). The major papilla could not be located due to duodenal edema.

Repeat endoscopies over the following months demonstrated complete mucosal healing, though he did require an esophageal dilatation to treat a stricture. His course was complicated by the development of pancreatic necrosis initially managed with an endoscopic ultrasound (EUS) guided cystgastrostomy. Due to chronic abdominal pain and complete obstruction of the distal pancreatic duct, he later underwent a pancreaticojejunostomy to relieve the obstruction. His clinical condition improved significantly following this surgical procedure.

Discussion

Acute esophageal necrosis, also known as black esophagus or necrotizing esophagitis, is a relatively rare and often incidentally noted clinical entity. It is characterized by diffuse circumferential black esophageal mucosa that generally involves at least the distal esophagus with abrupt termination at the gastroesophageal junction.^{1,4} It is rarely seen in clinical practice; Augusto et al. reported the condition in 29 of 10,295 patients who underwent an upper gastrointestinal endoscopy over a 5 year period (0.28%).³

Most commonly described in elderly hospitalized men, acute esophageal necrosis often presents with symptoms of upper gastrointestinal hemorrhage, though dysphagia and epigastric pain have been observed. It has been associated with diabetes mellitus, malignancy, and vascular

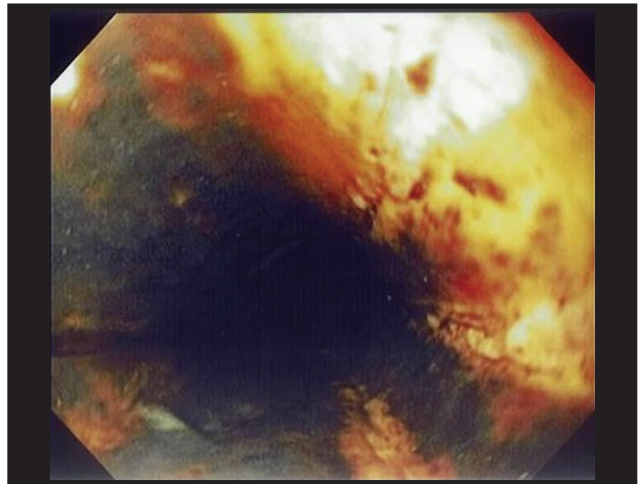


Figure A.

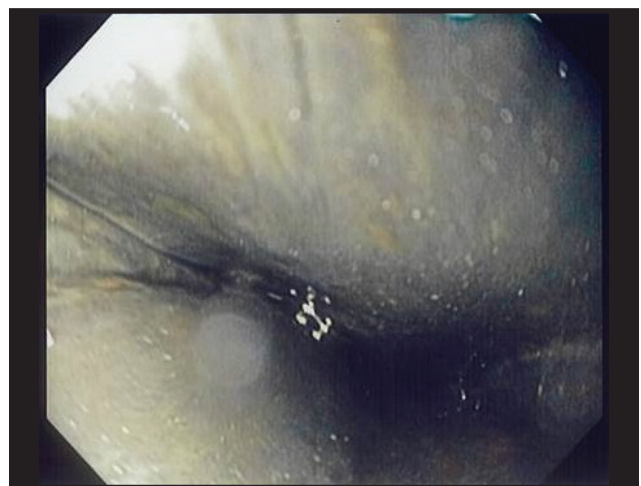


Figure B.

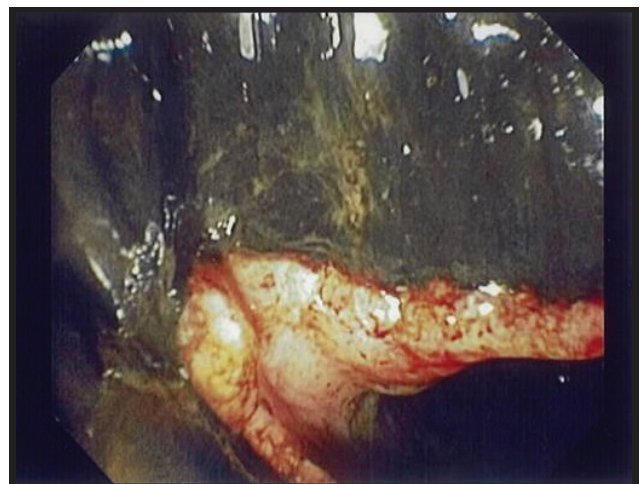


Figure C.

diseases, as well as hypoalbuminemia, hypoxemia, and dehydration.^{1,4} The pathogenesis has yet to be fully elucidated; the most commonly advanced explanation is transient tissue hypoperfusion leading to diminished esophageal mucosal defense, followed by severe gastric reflux and subsequent tissue injury.⁴

Endoscopy typically discovers diffuse, black, and necrotic mucosa in a circumferential pattern involving varying lengths of the esophagus with stark and abrupt termination at the gastroesophageal junction.^{1,2,4,5} While it most often affects the distal third of the esophagus, the condition has been noted in the mid and upper esophagus as well.⁴ Histology usually demonstrates severe mucosal and submucosal necrosis along with muscle fiber inflammation and/or destruction.²

Management is primarily supportive and typically involves oral nutritional rest, intravenous proton pump inhibitor use, short term total parental nutrition, and treatment of the underlying disorders.^{2,4} If co-morbid conditions show adequate improvement, case studies suggest that spontaneous mucosal healing can be expected.^{1,3} Mortality may be as high as 32.5% due to severe underlying medical conditions.⁵ Esophageal rupture is the most severe immediate complication, occurring in approximately 6% of cases and is typically accompanied by mediastinitis, abscess formation, and an extremely high mortality rate.⁴ Esophageal strictures are commonly encountered during the recovery period and are seen in 10-25% of cases.¹

SUMMARY

Acute esophageal necrosis is a rare but serious medical condition that often presents in medically ill older men with symptoms consistent with upper gastrointestinal bleeding. Endoscopy typically discovers black and necrotic mucosa in the distal esophagus with abrupt termination at the gastroesophageal junction. Management is generally limited to supportive esophageal care and aggressive treatment of the underlying medical issues. While immediate and devastating acute complications can occur, the high mortality associated with this condition is attributed to the severity of the underlying medical issues. Should the medical issues improve, spontaneous esophageal healing can be reasonably expected ■

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