Clinical vignette: Acute esophageal necrosis

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Recommended Citation
Acute Esophageal Necrosis
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INTRODUCTION
Acute Esophageal Necrosis (AEN), also known as “Black Esophagus” is a condition which occurs after a sequence of physiologic challenges. The inciting event is diminished blood flow, followed by caustic exposure to gastric contents, ultimately leading to a compromise in the protective function of the esophageal architecture. Susceptible populations include immune compromised and elderly patients.

EPIDEMIOLOGY
Reported incidence of black esophagus is very low, ranging from 0.0125%–0.2%. [1]

DIFFERENTIAL DIAGNOSIS
- Melanoma
- Acanthosis nigricans
- Pseudomelanosis
- Melanosis
- Coal dust deposition
- Corrosive ingestion

MECHANISM / ETIOLOGY
Ischemic phenomenon is viewed by some as the major factor in rapid development of esophageal lesions. Low flow states or shock, atherosclerosis, thromboembolic disease, cardiac arrhythmias, and hypoperfusion as a result of severe lactic acidosis have been implied as possible etiologies.

CASE PRESENTATION
69-year-old male with HIV, Insulin Dependant Diabetes, Parkinsonism, who was recently discharged from the hospital to a skilled nursing facility after being treated for Clostridium Difficile presented to the emergency department with chest pain and nausea, which he attributed to heartburn and receiving an extra dose of HAART. Nitrofurantoin was given at the skilled nursing facility without any appreciable relief. On evaluation patient revealed 7/10, non-radiating, epigastric pain exacerbated by oral intake. On physical exam patient was afebrile, tachycardic, hypotensive. He appeared pale, cachectic with temporal wasting. Stool guaiac was positive. Pertinent laboratory results included an elevated creatinine, BUN, and negative cardiac biomarkers. EKG showed sinus rhythm with no S-T wave changes. Patient was started on pantoprazole drip. The morning after admission patient underwent EGD, which found circumferential pallor and blackening of the mucosa (see picture below) limited to the gastro-esophageal junction, suggestive of acute esophageal necrosis (AEN). Moreover, the patient continued to remain hypotensive in the absence of active hemorrhage or volume loss, this was later attributed to an underlying sepsis. Moving into day two of hospitalization patient reported “blood tinged diarrhea”. An infectious stool work up found that patient had recurrence of C. Diff and patient was immediately started on high intensity oral Vancomycin was started. The patient was discharged from the hospital to a skilled nursing facility after complete course of PO vancomycin was started. The patient was discharged shortly after.

DISCUSSION
Acute Esophageal Necrosis (AEN), also known as “Black Esophagus” is a condition which occurs after a period of ischemia of the esophagus. On initial evaluation one must identify risk factors, diagnosis, and pathogenesis of the acute esophageal necrosis. Risk factors include age, male sex, heart disease, hemodynamic instability, alcohol ingestion, diabetes, renal insufficiency, and hypercoagulable state. The physiologic insult is often multifactorial, ultimately leading up to ischemic compromise. Discovery of acute esophageal necrosis should be viewed as a poor prognostic factor, and may suggest eminent mortality from the underlying disease process.

TREATMENT
Treatment is primarily supportive care, with intravenous fluids, proton pump inhibitors, nothing taken orally and instituting appropriate anti-microbial/virals/fungals as needed for underlying infections. Potential complications include stricture, perforation, and death. Surgical evaluation should be obtained for the aforementioned complications. Ideally treatment is to find/reverse the inciting event and prevent further physiologic insult.

PROGNOSIS
Death secondary to esophageal necrosis occurs in less than 6% of cases[3]. Prognosis of black esophagus is very poor, and its mortality reaches up to 31.8%-50%, although most of the deaths were caused by underlying illnesses[3-4].

REFERENCE