A Case Report of Volatile Ingestion-Induced Acute Esophageal Necrosis

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Introduction

- Acute esophageal necrosis (AEN), also known as black esophagus or acute necrotizing esophagitis, is a rare syndrome with diffuse circumferential black discoloration often of distal third esophageal mucosa due to relative hypovascularity and termination at the gastroesophageal junction.
- Despite controversial pathogenesis, there is strong consideration for a two-hit hypothesis where an initial low-flow vascular state predisposes the mucosa to a severe topical injury. There is further consideration for impaired local defense barriers.
- Chemical injury by gastric contents, toxin ingestion, severe infectious diseases, diabetic ketoacidosis and shock are associated with AEN.
- Pre-disposing risk factors for AEN include advanced age, male gender, and comorbidities like diabetes mellitus (DM), hypertension, cardiovascular disease, chronic kidney disease, malnutrition, chronic alcohol abuse and post-irradiation.

Case Report

- 74-year-old male with PMHx coronary artery disease, Type II DM, chronic obstructive pulmonary disease and prior esophageal adenocarcinoma post chemotherapy and radiation complicated by erosive esophagitis, alcoholism and homelessness presented to the hospital with shock, 3-day history of hematemesis and encephalopathy.
- Labs were significant for acute kidney injury (AKI), arterial blood gas with a pH of 7.16 and high anion gap metabolic acidosis (HAGMA) of 26 mEq/L and osmolar gap of 59 mOsm/kg, hemoglobin drop from 14 g/dl to 9 g/dl. Blood toxicology screen specifically requested for volatile acids however this lab was unfortunately not completed. Infectious work-up negative. Urine drug screen was negative.
- Patient was started on emergent hemodialysis given acute renal failure and concern for toxic volatile ingestion and intravenous proton pump inhibitor (PPI).
- A CT scan revealed a small-moderate sized hiatal hernia with fluid-filled visualized esophagus and distended, fluid-filled stomach. Thus, NGT placed for decompression which yielded 700cc coffee-ground emesis.

Imaging

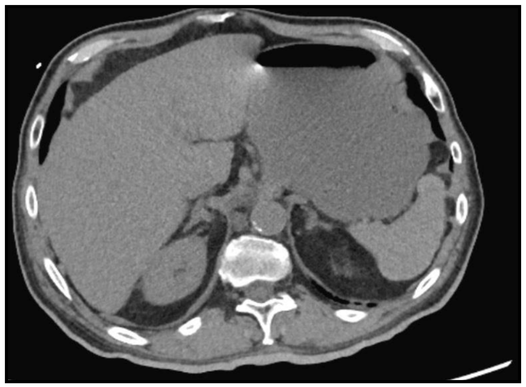


Figure 1: CT abdomen without contrast demonstrating fluid-filled, distended stomach and esophagus with hiatal hernia.

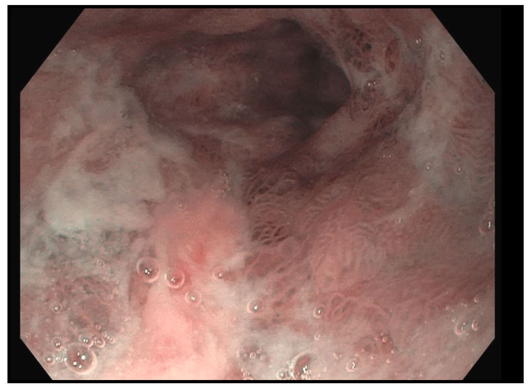
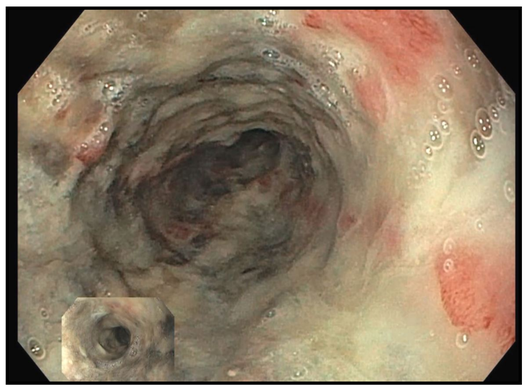
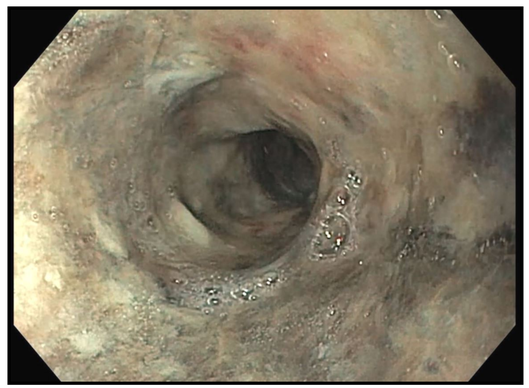


Figure 2: Second upper endoscopy demonstrating erosive, edematous, fibrotic and erythematous tissue.





Figures 3a and 3b: Initial upper endoscopy demonstrating Los Angeles Grade D esophagitis throughout esophagus with necrotic appearance.

Case Report Continued

- Upper endoscopy was performed revealing Los Angeles Grade D esophagitis throughout entire esophagus with blackened appearance as well as gastric body erosions and superficial clean-based duodenal ulcers along with a hiatal hernia (Figures 3a, 3b). No biopsies obtained given friable, severely inflamed mucosa.
- Patient was continued on oral PPI twice daily for eight weeks with plan for repeat upper endoscopy to evaluate for mucosal healing and avoidance of NGT placement.
- Patient underwent follow-up EGD noted to have a partially obstructive esophageal stricture in upper esophagus and severely erosive, edematous, fibrotic and erythematous tissue distal to this. Biopsies were taken and benign (Figure 2). Plan for follow up EGD to assess healing as patient remains on BID PPI therapy.

Discussion

- Prevalence of AEN ranges from 0.001 to 0.2%. There is a high mortality rate of 32% likely due to comorbidities and complications.
- Although biopsy is recommended, this is not required for diagnosis.
- Treatment focuses on NPO restriction, supportive red blood cell transfusion, intravenous (IV) proton pump inhibitors (PPI), restoring hemodynamic stability and correcting coexisting clinical conditions.
- This case illustrates how chemical injury via toxin ingestion in the setting of distributive shock in a male with known pre-disposing risk factors is associated with acute esophageal necrosis.
- Despite AEN's rarity, given overall 32% mortality rate and 6% mortality rate directly attributable to complications of AEN, early recognition and prompt medical management is imperative to favorable outcomes.

References

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