Cocaine-Induced Esophageal Necrosis: A Rare Cause of Esophageal Stricturing

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Introduction

- Acute esophageal necrosis (AEN), AKA “necrotizing esophagitis” and “black esophagus”, is a rare condition characterized by a distinct black appearance of the esophagus on esophagogastroduodenoscopy (EGD), usually starting at the distal esophagus and abruptly stopping at the gastroesophageal junction (GEJ).
- The etiology is thought to be due to an inciting event causing a state of decreased vascular flow and loss of protective characteristics of the mucosal barrier, followed by severe topical injury to the esophageal mucosa.
- Cases of AEN associated with cocaine use are even rarer, with very few reported cases in the literature.
- The mechanism of cocaine-induced AEN is not completely understood, however it has been thought that the vasoconstrictive properties of cocaine can exacerbate and contribute to esophageal ischemia.

Case Presentation

- 60-year-old male with a history of esophageal strictures requiring multiple dilations, prior small bowel obstruction (SBO) status post resection and re-anastomosis, cocaine-use disorder, and insulin-dependent diabetes mellitus presented with coffee ground emesis with associated weakness, nausea, chest pain, and abdominal pain.
- The patient was found to be in diabetic ketoacidosis as well as atrial fibrillation with rapid ventricular rate.
- Drug screen was positive for cocaine. The patient was placed on DKA protocol, and his heart rate improved with IV fluid resuscitation.
- Once stabilized, patient underwent EGD, revealing white and black mucosa from the distal to mid-esophagus, grade-D esophagitis. Surgical pathology revealed fragments of squamous mucosa in a background of necroinflammatory granulation tissue with fungal pseudohyphae. He was diagnosed with cocaine-induced esophageal necrosis.
- Treatment consisted of twice daily IV proton-pump inhibitor and carafate four times a day along with cocaine cessation counseling.
- After 4 days of therapy, symptoms resolved.

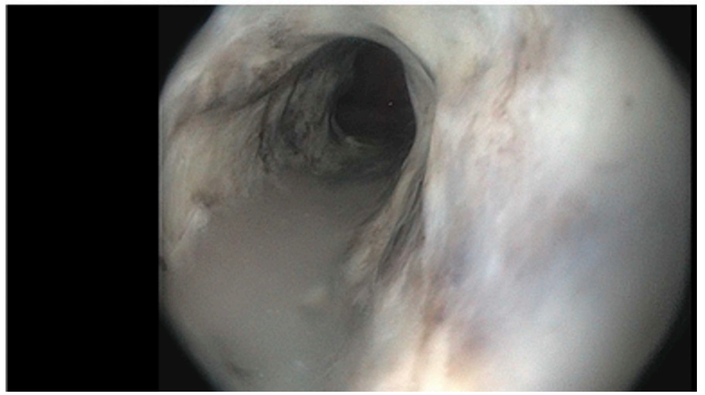

Image A

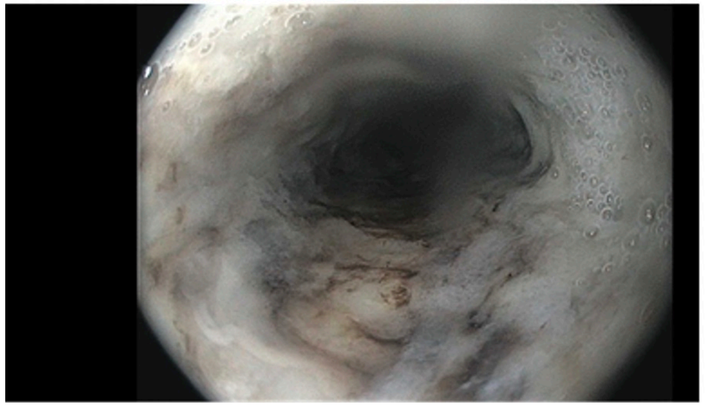

Image B

EGD revealing white and black esophageal mucosa of the lower third of the esophagus (Image A) and the middle third of the esophagus (Image B).

Discussion

- In our patient who has a history of esophageal stenosis with multiple dilations, the chronic use of cocaine likely resulted in vasoconstriction and ischemia to his already injured esophageal mucosa, leading to acute necrosis.
- Patients with AEN typically present with vague upper GI symptoms (hematemesis, coffee-ground emesis, and/or melena), along with varying degrees of severity and stability.
- EGD with biopsy is the gold standard for diagnosis of AEN. In early stages, the esophageal mucosa is typically black with areas of friable hemorrhage, and in later stages there can be thick, white exudates of sloughed mucosa.
- Further management can require surgical intervention, and is typically indicated in perforated esophagus, mediastinitis, and abscess formation.
- Stricturing of the esophagus can be a late complication and is likely in our patient with a history of esophageal strictures.
- Treatment involves supportive measures and treating the underlying diseases.

Conclusion

- The etiology of AEN is multifactorial and rare, yet AEN increases the risk for infection, stenosis, stricture, perforation, and death.
- EGD with biopsy is the gold standard for diagnosis.
- Treatment of underlying conditions, cessation of offending agents like cocaine and alcohol, and compliance with follow-up are the mainstays of treatment.