Case Report

Acute Esophageal Necrosis Following Acetaminophen Overdose: An Unreported Cause of Black Esophagus

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Abstract

Description

Acute esophageal necrosis (AEN), also known as "black esophagus" or Gurvits syndrome, is an uncommon finding with an unclear etiology and pathogenesis. This condition often presents as an upper gastrointestinal bleed in older men with multiple comorbidities. AEN is characterized by circumferential black, necrotic mucosa in the esophagus. We present a case of AEN following acetaminophen overdose. The patient was ultimately discharged from the hospital with oral omeprazole twice daily, a clear liquid diet, and a recommendation for follow-up in the outpatient setting for repeat EGD in 4 to 6 weeks. Acetaminophen overdose, although a rare cause, must be considered as a possible etiology of AEN.

Keywords

esophageal diseases; necrosis; drug overdose; attempted suicide; esophagitis; acetaminophen/poisoning; AEN; esophagus; endoscopy

Introduction

Acute esophageal necrosis (AEN) is a rare clinical entity with an estimated incidence of ~0.2%.[1] This condition is being diagnosed more frequently due to the widespread availability of endoscopy.[1,2] The incidence of AEN is more than 4 times higher in men than women, especially for those older than 65.[1,3,4] The etiology of esophageal necrosis is diverse, including shock, gastric outlet obstruction, hypoxemia, diabetic ketoacidosis, infection medications, trauma, and impaired mucosal defenses.[4,5] The majority of patients have multiple comorbidities, such as diabetes, hypertension, chronic liver disease, and/or chronic kidney disease.[6] AEN typically presents as an upper gastrointestinal bleed with hematemesis or melena, while other associated symptoms include low-grade fever, nausea, dysphagia, chest discomfort, and/or epigastric pain.[1,5-7]

Case Presentation

A 53-year-old Caucasian man with a history of alcohol dependence and compensated liver cirrhosis with a model for end-stage liver disease (MELD) score of 13 (with recently diagnosed small esophageal varices) presented to the emergency department after being found unresponsive at his home following an intentional acetaminophen overdose.

On admission, laboratory data were remarkable for an acetaminophen level of 530 ug/mL, lactic acid of 5.20 mmol/L, and mildly elevated liver function tests (AST 48 u/L, ALT 50 u/L), total bilirubin 1.7 mg/dL, alkaline phosphatase 129 u/L, hemoglobin 11.8 gm/dL, platelet count of 49 u/L, and a white blood cell count of 3.6 u/L. His urine drug screen was negative, and the patient denied using illicit narcotics. Due to worsening hemodynamic instability and severe hypoxia, the patient was intubated and transferred to the intensive care unit. Bright red blood was noted in the orogastric tube following insertion. N-acetylcysteine (NAC), octreotide, ceftriaxone, and pantoprazole infusions were initiated due to concern for possible variceal bleeding.

Within 24 hours, a decision was made to perform esophagogastroduodenoscopy (EGD)

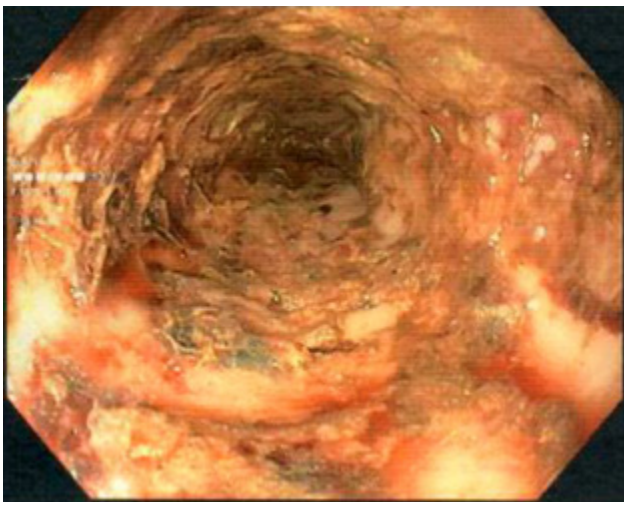

Figure 1. Image shows necrotic and sloughing esophageal mucosa, consistent with the diagnosis of AEN.

that subsequently revealed severe esophagitis with diffuse circumferential necrosis and black sloughing mucosa in the mid- and distal esophagus **(Figure 1)**. No esophageal or gastric varices were seen. Given the extensive nature of necrosis observed during EGD, a chest X-ray was requested to rule out esophageal perforation. Imaging was negative for pneumatosis or free air, and the patient was treated conservatively without surgical intervention. Given these findings, the patient was diagnosed with AEN. Hence, octreotide was discontinued and the orogastric tube was removed to avoid any further esophageal trauma. The patient was switched to oral omeprazole twice daily following extubation. The patient was ultimately discharged from the hospital with oral omeprazole twice daily, a clear liquid diet, and was recommended to follow up in the outpatient setting for a repeat EGD in 4 to 6 weeks. Unfortunately, the patient was lost to follow-up and did not undergo a second EGD to assess for mucosal healing.

Discussion

The reported incidence of black esophagus is very low, ranging from 0.0125% to 0.2%. AEN is a relatively rare clinical disease process with an estimated mortality rate of 38%.[8,9] The pathogenesis of AEN has been proposed to involve low systemic perfusion, direct toxic effect, diminished mucosal defenses, and mucosal breakdown.[2,6] Due to the multifactorial etiology of AEN, patients with comorbid conditions including diabetes, hypertension, chronic liver disease, and/or chronic kidney disease are at higher risk.[2] Acetaminophen-induced acute esophageal necrosis has not been described in the current review of the literature.

Currently, the "two-hit" hypothesis has been the widely accepted theory to describe the pathophysiology of AEN.[3] The initial event consists of a low-flow vascular state and an inciting event resulting in esophageal injury.[1] It is important to note that the ingestion of corrosive agents excludes the diagnosis of AEN[9]; caustic ingestion was ruled out in our patient. We believe our patient developed a direct mucosal injury from the ingestion of a large amount of acetaminophen in combination with the development of a state of hemodynamic instability with shock and reduced perfusion to the region of the esophagus, leading to overt mucosal necrosis.

AEN is diagnosed 4 times more often in men than in women.[7,10] Peak incidence of AEN occurs in the sixth decade of life.[8] AEN commonly presents as an upper gastrointestinal bleed with hematemesis and/or melena. Other associated symptoms include low-grade fever, nausea, epigastric pain, dysphagia, chest discomfort, tachycardia, hypovolemia, and/or hypotension.[2,3,5,7]

While a thickened distal esophagus may be seen on computed tomography imaging, EGD is considered the gold standard for diagnosis.[4,5] The typical endoscopic appearance of AEN is a circumferential black mucous membrane in the

distal esophagus halting at the gastroesophageal junction.[1,4] Histology is not necessary for diagnosis, but typically reveals necrotic epithelium, polymorphonuclear infiltrates, inflammatory changes, and necrosed mucous membrane.[2,4]

There is no standardized treatment for AEN, and management largely consists of supportive care and treatment of the underlying condition.[2,4] Surgical intervention is typically reserved for cases of esophageal perforation.[11] The mainstays of treatment have been nil-per-os (NPO), intravenous hydration, intravenous proton pump inhibitor, and short-term parenteral nutrition.[4,6] Nasogastric tube placement should be avoided due to the high risk of perforation.[11] In most cases, repeat endoscopy reveals a normal esophageal mucus membrane after 1 to 2 weeks.[4] The major complications of AEN include perforation, stricture formation, and stenosis. However, the majority of reported patients do not develop long-term sequela.[2,6] AEN has variable prognoses and recovery results depending on the severity and the extent of esophageal involvement. Unfortunately, our patient was lost to follow-up, making it difficult to establish the extent of recovery and possible development of complications.

Conclusion

This case demonstrates that conservative therapy, even in extremely severe cases of AEN, should be considered, and the general gastroenterologist should be aware that acetaminophen overdose, although a rare cause, must be considered as a possible etiology of AEN.

Conflicts of Interest

The authors declare they have no conflicts of interest.

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