Bilateral Esophagus: A Rare Case of Acu

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Society of American Journal of Medicine (SS

AJM), 4 (1). pp. 19-23. ISSN 2996-587X

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Black Esophagus: A Case Report

Samaan, Bishoy [1]; Tahir, Hafsa MD [2]; Mikhaiel, Elarea MS [3]; Fahim, Mark MS [4]; Maher, Miray BHSc [5]; Awad, Youana BHSc [6]; Kumar, Sunny MBBS [7]

1. Saint George's University School of Medicine, Grenada, West Indies
2. Providence Alaska Medical Center, Anchorage, Alaska
3. University of Central Lancashire, Preston, England
4. American University of the Caribbean, St. Maarten, Lesser Antilles
5. Temerty Faculty of Medicine, Toronto, ON
6. Wilfrid Laurier University, Waterloo, Ontario
7. The Wright Center for GME, Scranton, PA

Abstract:

Acute esophageal necrosis (AEN), or "black esophagus," is a rare and life-threatening condition characterized by diffuse circumferential black discoloration of the esophageal mucosa. This case report discusses a 71-year-old male with multiple comorbidities who presented with gastrointestinal bleeding and was subsequently diagnosed with AEN. The report highlights the clinical presentation, diagnostic findings, management strategies, and a review of relevant literature.

Introduction:

AEN is an uncommon clinical entity with an estimated prevalence ranging from 0.01% to 0.2% in autopsy studies [1]. It is characterized by a striking black appearance of the esophageal mucosa on endoscopy, typically involving the distal esophagus and abruptly stopping at the gastroesophageal junction [1]. The etiology is multifactorial, often involving a combination of ischemic insult, gastric acid injury, and systemic conditions leading to hemodynamic compromise [2]. The mortality rate of AEN is high, with one study approximating mortality at 30%. However, 60% of affected patients experience favorable outcomes with appropriate treatment [3]. AEN predominantly affects elderly males with multiple comorbidities [1]. Risk factors include type 2 diabetes, hypertension, alcoholism, and chronic kidney and liver disease [2].

Case Presentation:

A 71-year-old male presented to the emergency department with symptoms of gastrointestinal bleeding and a fall resulting in a head injury. Medical history remarkable for atrial fibrillation, coronary artery disease, congestive heart failure, aortic and mitral prosthetic valves, a pacemaker, chronic obstructive pulmonary disease, cirrhosis, renal disease on hemodialysis, and epilepsy. Medications included aspirin, carvedilol, Depakote, digoxin, divalproex sodium, furosemide, valsartan, and warfarin. Vitals were remarkable for a temperature of 95.1°F, heart rate of 60 bpm, respiratory rate of 20, blood pressure of 118/53 mmHg, and oxygen saturation of 96% on 4L via nasal cannula. He arrived confused and hypoglycemic, for which both improved following IV dextrose administration and red blood cell transfusion. The patient reported experiencing hematemesis, vomiting, hematochezia for three weeks, and black tarry stools for three months (which continued through most of the hospital course). Additionally, he had significant bodily weakness and a history of recurrent falls. Hemoglobin was 10.1g/dL on arrival, and this continued to drop daily. Further labs can be noted in Table 1. He was

maintained on IV fluid resuscitation and IV proton pump inhibitor (PPI). The patient underwent esophagoduodenoscopy (EGD), which was remarkable for distal esophageal ulcerations and patches of black mucosa, concerning for necrosis, which was biopsied (Figure 1). Pathology was notable for necro-inflammatory debris without intact esophageal mucosa. By day 4, there were no further reports of tarry dark stool, and Hgb had stabilized around 8g/dL. He was transfused 1U PRBC (secondary to cardiac history and transitioned to PO PPI. By day 6, he was feeling symptomatically better, his vitals remained stable, and he was cleared for discharge to rehab due to weakness. He was recommended to follow up in the clinic in 1 week for further evaluation and discussion of the need for a repeat endoscopy.

Investigations:

- *Laboratory Tests:*
  - Elevated white blood cell count
  - Renal dysfunction
  - Abnormal liver function tests
  - Elevated INR
- *Upper Endoscopy with Biopsy:*
  - Identified necro-inflammatory debris and ulcerative esophageal mucosa
- *Transthoracic Echocardiography:*
  - Mitral valve stenosis with paravalvular regurgitation
  - Reserved left ventricular systolic function
- *Liver and Gallbladder Sonogram:*
  - Benign findings with no obvious pathology
- *CT Imaging:*
  - CT Head showed cerebral atrophy with chronic microvascular ischemic changes
  - CT Chest/Abdomen revealed multiple rib fractures and pancreatic atrophy with fatty infiltrates

Table 1: Table depicting the trend of various labs from arrival to day 6 when the patient was cleared for discharge.

Lab Type	Arrival	Day 2	Day 3	Day 4	Day 5	Day 6
WBC (10^3/µL)	17.00	15.30	10.70	7.00	7.70	10.70
Hgb (g/dL)	10.1	9.2	8.5	7.9	8.1	9.2
Platelets (10^6/µL)	228	153	133	111	107	118
Sodium (mmol/L)	138	140	138	139	139	135
Potassium (mmol/L)	4.2	4.2	4.0	3.8	4.1	4.1
BUN (mg/dL)	64	68	43	27	17	14
Creatinine (mg/dL)	2.1	2.0	1.7	1.3	1.2	1.1

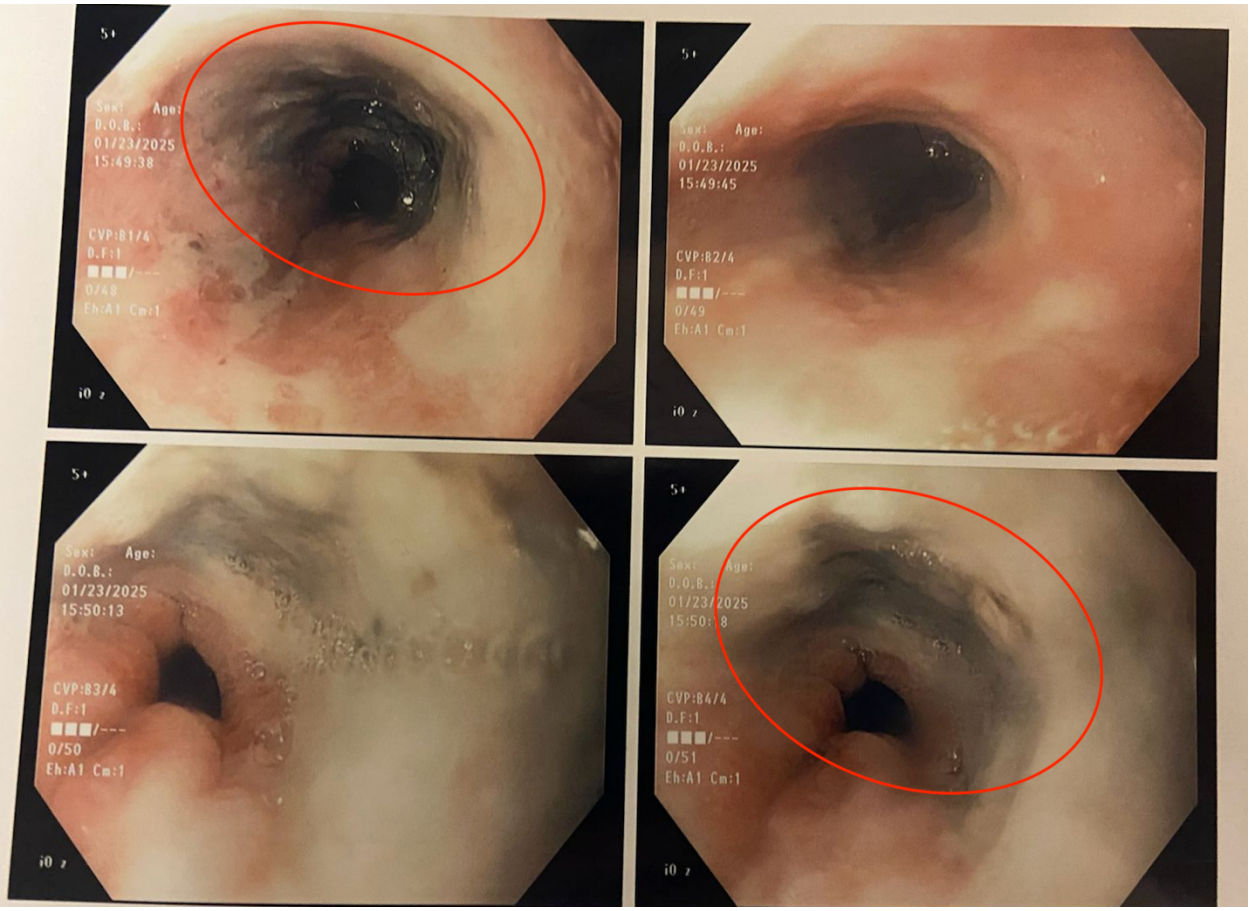

Figure 1: Endoscopic image with areas of visualized necrosis identified within the red circle.

Discussion:

AEN is a rare condition often associated with multiple comorbidities and systemic complications. The pathophysiology involves a combination of ischemia, gastric acid injury, and impaired mucosal defense mechanisms. Peak incidence occurs in the sixth decade of life and is more common in males with multiple comorbidities and poor nutritional status. Chronic alcohol use, illegal drug use, malignancy, solid organ transplantation, CAD, diabetes, hypertension, various infectious states causing immunocompromise, including HIV, herpes simplex virus, cytomegalovirus, and fungal infections, have been associated with diffuse AEN [4][5].

It primarily affects the distal esophagus due to its limited vascularization. Chronic microvascular ischemia can diminish perfusion, leading to intermittent low-flow states [5]. As the distal esophagus is considered a “watershed zone” compared to the remainder, as it receives blood flow from more distal branches, this makes it more susceptible to ischemic injury during these times of decreased perfusion [1] [6].

The classic presentation includes upper GI bleeding, hematemesis, coffee-ground emesis, melena, and dysphagia. Diagnosis is confirmed via esophagogastroduodenoscopy (EGD), which reveals characteristic black discoloration of the esophageal mucosa [8].

Histology is notable for necrotic changes, leukocytic infiltrate, and absence of squamous epithelium. Regeneration of damaged mucosa appears as whitish exudates in between the areas of black discoloration, giving it a “chessboard” like appearance [7]. The histopathology also helps distinguish AEN from other conditions, such as malignant melanoma, melanosis, coal deposition, etc.

The management of AEN includes symptomatic management and close monitoring for signs of hemodynamic compromise. Volume resuscitation and appropriate transfusion (to maintain hemoglobin > 7 g/dL and platelets at > 50,000/mm3). Patients should initially be kept NPO for the first 24 hours with a slow progression of diet once more hemodynamically stable with less concern for bleeding/complications. Medical management includes gastric acid suppression with PPI and can include mucosal protection with sucralfate. Intravenous PPI is preferred initially (intermittent boluses vs infusion), which can later be changed to oral form [7]. Antimicrobial therapy is started for biopsy-positive esophageal cultures or in the presence of multinucleated giant cells or inclusion bodies. Nasogastric tubes should be avoided due to the risk of perforation. Bleeding should be rapidly controlled using submucosal epinephrine and some other form of hemostasis (i.e, self-expanding stents, clips, etc). Balloon tamponade should be avoided due to the risk of perforation. Perforations and strictures should be managed either surgically or with an expanding stent if surgery is not feasible (Chinta). Management can be summarized in Figure 2.

Management:

- **Supportive Care:**
  - Hgb transfusions for levels < 7g/dL (8 for those with cardiac comorbidities)
  - maintaining platelet numbers > 50,000/mm^3
  - V Fluid resuscitation,
- **Pharmacological Care:**
  - IV proton pump inhibitor infusion with transition to PO when stable
  - Sucralfate
  - Antibiotics (i.e. ceftriaxone)
  - Octreotide infusion if suspected variceal bleed
- **Nutritional Support:**
  - Maintain NPO initially, with slow advancement of diet
- **Anticoagulation Management:**
  - Can resume in the absence of active bleeding.
- **Lifestyle and Medications:**
  - Avoidance of NSAIDs, close INR monitoring.
- **Monitoring and Follow-up:**
  - Repeat laboratory tests and imaging to assess treatment effectiveness

Figure 2: Figure depicting overall management for acute eosinophilic necrosis.

The prognosis varies, with a mortality rate of 30-50%, largely depending on the patient’s underlying health conditions [9]. Complications such as esophageal perforation, strictures, and superinfection contribute to poor outcomes [8]. Effective management includes aggressive resuscitation, hemodynamic stabilization, gastric acid suppression, and infection control. At this time, there is no official consensus on the timing of initial endoscopic intervention, but ideally, it should be performed within the first week [9]. Repeat endoscopic follow-up is recommended to monitor healing and prevent complications.

Conclusion:

AEN is a rare but life-threatening condition characterized by partial or complete circumferential blackening of the distal esophagus, primarily due to ischemic insult and systemic instability. Early recognition, aggressive resuscitation, and supportive care are critical in reducing mortality. Given the high association with comorbidities, a multidisciplinary approach is necessary for optimal patient outcomes.

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