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Acute Esophageal Necrosis: A Rare Condition Not to be Overlooked

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Abstract | **Case Report**

Acute esophageal necrosis, also known as "black esophagus," is a rare condition characterized by diffuse necrosis of the esophageal mucosa, predominantly affecting the distal esophagus. It most commonly occurs in patients with severe comorbidities. We report the case of a 57-year-old patient with no significant medical history, admitted for moderate hematemesis. Clinical examination revealed a hemodynamically stable patient. Laboratory findings showed normochromic normocytic anemia with a hemoglobin level of 7 g/dL and an elevated urea-to-creatinine ratio. Upper gastrointestinal endoscopy demonstrated a typical black esophagus involving the distal two-thirds of the esophagus. Additional investigations, including viral serologies and esophageal biopsies, did not reveal any specific infectious etiology. The patient was managed conservatively, with a favorable outcome and significant endoscopic improvement at 8 weeks.

Keywords: Acute esophageal necrosis – Black esophagus – Hematemesis – Endoscopy – Case report.

INTRODUCTION

Acute esophageal necrosis [AEN] is a rare clinical entity defined by circumferential necrosis of the esophageal mucosa, resulting in a characteristic black appearance on endoscopy. Its incidence is estimated to range between 0.01% and 0.2% of upper gastrointestinal endoscopies.

The pathophysiology of AEN is multifactorial, involving tissue hypoperfusion, impairment of mucosal defense mechanisms, and gastric acid reflux injury. It typically occurs in patients with significant comorbid conditions.

CASE PRESENTATION

A 57-year-old male with no significant past medical history, particularly no known cardiovascular disease, presented to the emergency department with moderate hematemesis.

On admission, the patient was hemodynamically stable

Laboratory investigations revealed normochromic normocytic anemia with a hemoglobin level of 7 g/dL, along with an elevated urea-to-creatinine ratio, suggesting an upper gastrointestinal source of bleeding.

Initial upper gastrointestinal endoscopy revealed diffuse black discoloration of the esophageal mucosa with extensive necrotic areas and associated congestive zones.

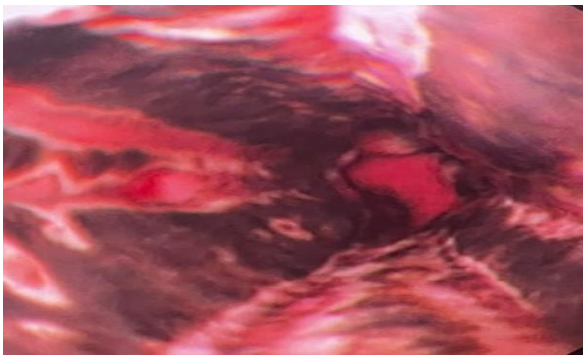

Figure 1: Black necrotic esophageal mucosa

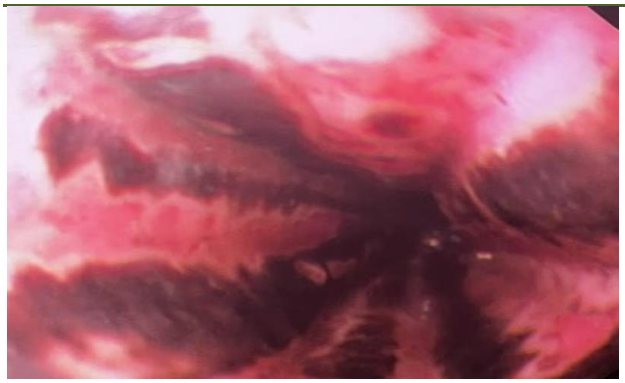

Figure 2: Extensive involvement of the distal esophagus

Viral serologies, including HIV testing, were negative. Esophageal biopsies demonstrated a nonspecific inflammatory infiltrate, with no evidence of cytomegalovirus [CMV] infection.

Management was conservative. The patient was kept nil per os [NPO], and parenteral nutrition was initiated to ensure adequate nutritional support.

High-dose intravenous proton pump inhibitors were administered. Blood transfusion with packed red blood cells was performed due to severe anemia.

The clinical course was favorable. A follow-up endoscopy performed at 8 weeks showed marked improvement of the lesions.

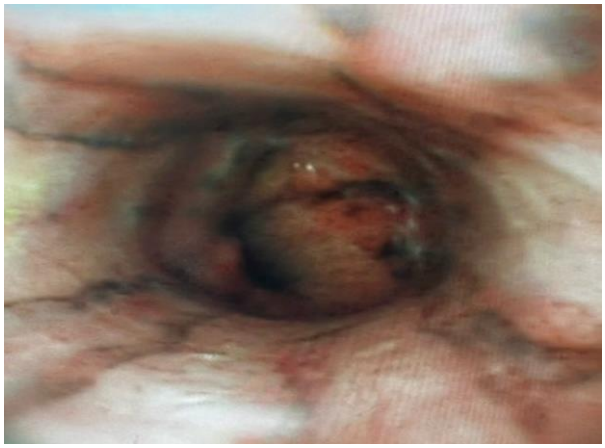

Figure 3: Regression of necrotic lesions on follow-up endoscopy

DISCUSSION

Acute esophageal necrosis is a rare condition with a reported prevalence ranging from 0.01% to 0.2% [4,6,7]. It predominantly affects males, with a male-to-female ratio of approximately 4:1, and typically occurs in elderly patients, with a mean age of around 67 years [2,8].

Several risk factors have been identified, including diabetes mellitus, malignancy, hypertension, chronic pulmonary disease, chronic alcohol abuse, coronary artery disease, liver cirrhosis, and renal insufficiency [2,6,7,9,12]. Other associated conditions include postoperative states, immunosuppression, and malnutrition.

AEN is generally considered a marker of severe underlying illness and is often associated with poor prognosis due to comorbid conditions. Mortality is more commonly related to underlying diseases than to the esophageal lesion itself.

The pathophysiology is multifactorial, involving impaired mucosal defense, ischemia, and gastric acid injury [2,13]. Ischemia appears to play a central role, supported by the distal predominance of lesions and vascular vulnerability of this segment [2,4,6,14].

In our case, the typical distal distribution of lesions with an abrupt transition at the gastroesophageal junction supports this mechanism, although the absence of hemodynamic instability suggests a possible transient or microvascular hypoperfusion.

Gastroesophageal reflux is also an important contributing factor, reported in approximately 40% of cases [15]. Infectious etiologies such as CMV, herpes simplex virus, bacterial infections [e.g., Klebsiella pneumoniae], and fungal infections [Candida] have also been described [5,10,11,16].

Clinically, upper gastrointestinal bleeding is the most common presentation, occurring in approximately 70% of cases [2,15,18].

Diagnosis is based on endoscopic findings showing circumferential black mucosa, typically involving the distal esophagus with a sharp demarcation at the gastroesophageal junction [2].

Biopsies are not routinely required but may be useful to exclude infections or malignancy [5,14].

Management is mainly supportive, including fluid resuscitation, nil per os status, proton pump inhibitors, and treatment of underlying conditions [2,23]. Parenteral nutrition is often preferred.

Complications include esophageal perforation [approximately 7%] and esophageal stricture formation [approximately 10%] [2].

Overall mortality is approximately 30%, mainly related to underlying conditions, while mortality directly attributable to AEN is lower [around 6%] [2].

In our case, the outcome was favorable, with complete clinical and endoscopic improvement.

CONCLUSION

Acute esophageal necrosis is a rare but serious cause of upper gastrointestinal bleeding. Diagnosis relies primarily on characteristic endoscopic findings.

This case highlights that AEN can occur in patients without significant comorbidities, emphasizing the importance of considering this diagnosis even in low-risk individuals.

Conservative management, including fasting, proton pump inhibitors, nutritional support, and correction of metabolic disturbances, can lead to a favorable outcome, as demonstrated in our case.

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