**Scholars Journal of Medical Case Reports**
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

**Medical Science**

# Acute Esophageal Necrosis: A Rare but Severe Cause of Gastrointestinal Bleeding

B. Benjelloun Abidi[1*], S. El Aoula[1], M. Acharki[1], N. Kabbaj[1], M. Salihoun[1]

[1]EFD-HGE Unity, Ibn Sina Hospital, Mohamed V University, Rabat-Morocco

**DOI:** https://doi.org/10.36347/sjmcr.2026.v14i03.041 | **Received:** 11.02.2026 | **Accepted:** 19.03.2026 | **Published:** 24.03.2026

***Corresponding author:** B. Benjelloun Abidi
EFD-HGE Unity, Ibn Sina Hospital, Mohamed V University, Rabat-Morocco

**Abstract** | **Case Report**

Acute esophageal necrosis (AEN), also known as "black esophagus," is a rare but life-threatening condition characterized by diffuse circumferential black discoloration of the esophageal mucosa with a sharp demarcation at the gastroesophageal junction. It is typically associated with severe systemic illness and multiple comorbidities. We report the case of a 77-year-old male with diabetes mellitus and arterial hypertension who was admitted for critical lower limb ischemia requiring surgical amputation. Two weeks later, he developed recurrent vomiting and hematemesis with a significant drop in hemoglobin levels. Urgent upper gastrointestinal endoscopy revealed extensive circumferential black mucosal necrosis involving the entire esophagus, abruptly ending at the esophagogastric junction, along with associated gastric and duodenal lesions. The patient received intensive supportive management including fluid resuscitation, blood transfusion, high-dose proton pump inhibitors, antibiotics, and correction of metabolic disturbances. Despite initial stabilization and adequate glycemic control, the clinical course was complicated by septic shock leading to death within five days. AEN is believed to result from a multifactorial pathophysiological process involving ischemic injury, impaired mucosal defenses, and gastric acid reflux. It predominantly affects elderly patients with significant comorbidities and commonly presents with upper gastrointestinal bleeding. Diagnosis relies on endoscopic findings, while management is mainly supportive and directed at correcting underlying conditions. The prognosis remains poor, with high mortality rates largely driven by the severity of associated systemic disorders. This case highlights the importance of early recognition of AEN in critically ill patients and underscores its severe prognosis despite appropriate management.

**Keywords:** Acute esophageal necrosis, black esophagus, gastrointestinal bleeding, esophageal ischemia, endoscopy, diabetes mellitus, critical illness, septic shock.

**Copyright © 2026 The Author(s):** This is an open-access article distributed under the terms of the Creative Commons Attribution **4.0 International License (CC BY-NC 4.0)** which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

## INTRODUCTION

Acute Esophageal Necrosis (AEN), also known as necrotizing esophagitis, black esophagus, or Gurvits syndrome, is a rare disorder. It combines multiple factors such as esophageal ischemia, acid reflux, and compromised mucosal defenses.

We report a case of a diabetic patient of 77 years old, who was admitted to the hospital for the management of digestive bleeding.

## CASE REPORT

A male patient, in his seventies, with multiple cardiovascular risk factors (arterial hypertension, type 2 diabetes mellitus) presented to the Emergency Ward with critical left lower limb ischemia. He was admitted to the operation room and a forefoot amputation was performed. Two weeks later, he presented multiple episodes of vomiting complicated with hematemesis (table). The hemoglobin levels decreased from 11.4 g/dL to 6.9 g/dL. He received an intravenous normal saline infusion and blood transfusion. Intravenous omeprazole infusion was also started.

Urgent gastroscopy showed diffuse circumferential ulcerations and black mucosa affecting the esophagus from his proximal portion to his distal portion, at 20 cm to 35 cm from the incisor teeth (figure 1). The lesions gradually worsened. The lesions ended at the eso-gastric junction (figure 2). Erosions and ulcerations were detected in the pyloric antrum and in the duodenum as well (figure 3).

The treatment involved the use of antibiotics and a high-dose proton pump inhibitor, alongside the management of ketoacidosis. Even if there was good glycemic control using insulin therapy and there was no

rebleeding, the patient subsequently succumbed to a septic shock after five days.

**Table 1**

| Laboratory | Value | Reference range |
|---|---|---|
| Hemoglobin (g/dL) | 6.9 | 13–16.5 |
| Hematocrit (%) | 19.8 | 40-49 |
| White blood cell (103/µL) | 22.5 | 4-10 |
| Platelets (103/µL) | 380 | 150-400 |
| Blood urea nitrogen (g/L) | 2.30 | 0.15-0.5 |
| Creatinine (mg/L) | 19.8 | 7.2-12.5 |
| C-reactive protein (mg/L) | 140 | < 5 |
| Total proteins (g/L) | 63 | 64-83 |
| Aspartate aminotransferase (U/L) | 25 | 5-34 |
| Alanine transaminase (U/L) | 13 | 0-55 |
| Alkaline phosphatase (U/L) | 103 | 40-150 |
| Gamma-glutamyl Transferase (U/L) | 20 | 11-59 |

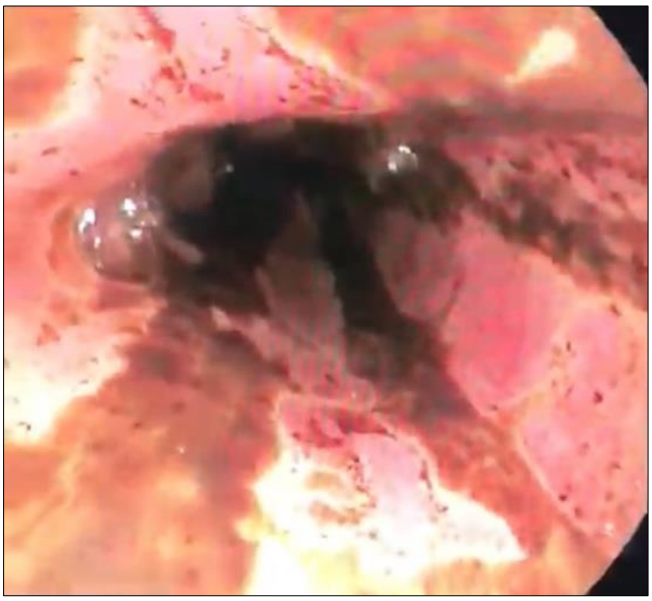

**Figure 1: Circumferential blackish mucosa at the proximal esophagus**

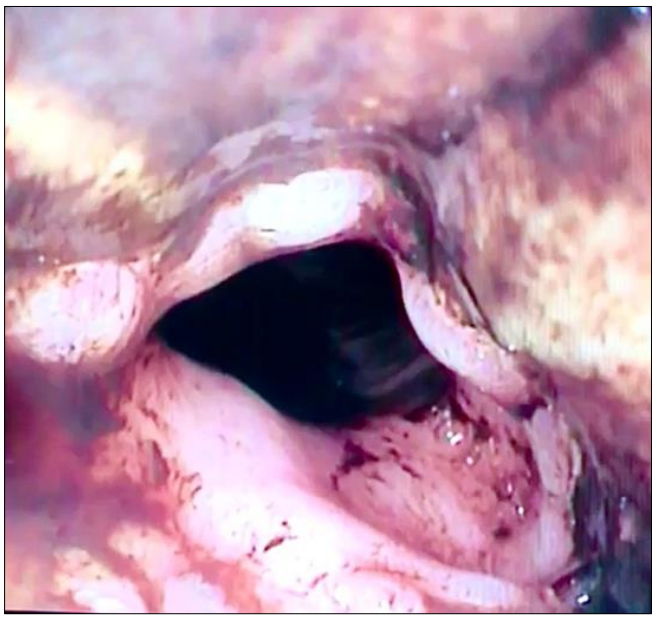

**Figure 2: Distal esophagus widely affected up to the esophagogastric junction (EGJ), where it is abruptly interrupted**

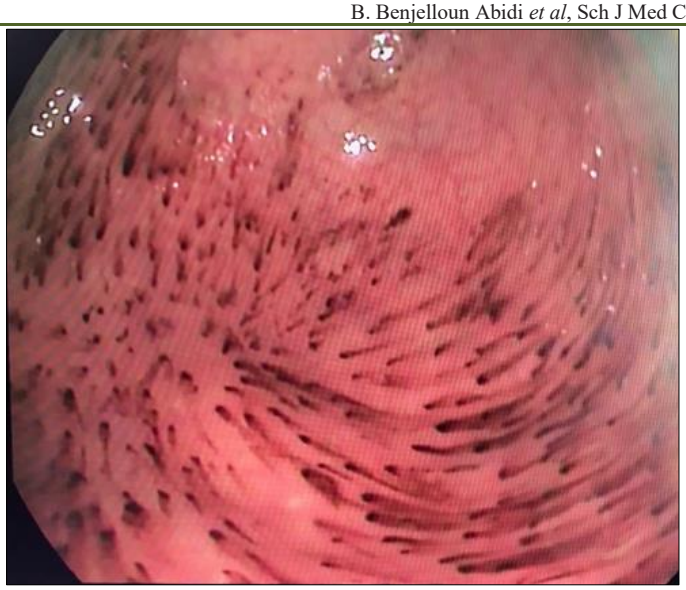

**Figure 3: Erosions in the pyloric antrum**

## DISCUSSION

Acute esophageal necrosis (AEN), also referred to as "black esophagus," is a rare but severe clinical syndrome characterized by circumferential black discoloration of the esophageal mucosa with a sharp demarcation at the gastroesophageal junction. The condition was first described by Goldenberg *et al*., in 1990 and has since been recognized as a distinct entity associated with severe systemic illness and high mortality rates [1].

The incidence of AEN is estimated to range between 0.01% and 0.28% of patients undergoing upper gastrointestinal endoscopy, highlighting the rarity of this condition [2-4]. It predominantly affects elderly male patients with multiple comorbidities, including diabetes mellitus, arterial hypertension, chronic kidney disease, and cardiovascular disease [2-6]. These conditions contribute to systemic hypoperfusion and impaired mucosal defenses, which play a key role in the development of esophageal necrosis [4, 5].

The pathophysiology of AEN is multifactorial and is commonly explained by the "two-hit hypothesis." The first event is reduced blood flow causing ischemia of the esophageal mucosa, while the second involves additional injury due to gastric acid reflux and weakened mucosal defenses [2-5]. The distal third of the esophagus is particularly vulnerable because of its relatively limited vascular supply compared with the proximal segments - 2, 3].

Clinically, AEN most commonly presents with upper gastrointestinal bleeding, which occurs in up to 90% of reported cases and usually manifests as hematemesis or melena [2-6]. Other symptoms may include epigastric pain, dysphagia, vomiting, or signs of hemodynamic instability [7, 8]. Because the disease may develop rapidly in critically ill patients, early recognition and prompt endoscopic evaluation are essential for diagnosis.

Upper gastrointestinal endoscopy remains the gold standard for diagnosis, revealing the typical circumferential black appearance of the esophageal mucosa with an abrupt termination at the gastroesophageal junction [2-4]. Although biopsy can be performed to exclude other etiologies such as infectious or caustic esophagitis, it is not always necessary and may carry a risk of perforation due to the fragility of necrotic tissue [4].

Management of AEN is mainly supportive and focuses on correcting the underlying precipitating factors [4-6]. Treatment usually includes aggressive fluid resuscitation, bowel rest, high-dose proton pump inhibitor therapy, and correction of metabolic disturbances [4, 5]. Blood transfusion may be required to treat severe anemia, and parenteral nutrition may be considered until oral feeding can be safely resumed [6]. Antibiotic therapy is not routinely recommended but may be used when infection or sepsis is suspected [6].

The natural evolution of AEN generally occurs in two phases. The initial phase is characterized by the necrotic black appearance of the esophageal mucosa, followed by a healing phase during which the mucosa becomes friable and pink with residual pseudomembranes [2]. In many cases, endoscopic healing occurs within 7 to 14 days, although recovery may vary depending on the patient's clinical condition [2-4].

Although rare, complications such as esophageal perforation, mediastinitis, and esophageal strictures may occur [4-9]. Esophageal perforation represents the most severe complication and may require surgical intervention [9].

The overall mortality rate associated with AEN has been reported to be approximately 30–32%, largely reflecting the severity of the underlying systemic diseases rather than the esophageal lesion itself [6-10]. In the present case, despite appropriate supportive therapy and correction of metabolic disturbances, the patient died from septic shock, illustrating the poor prognosis frequently associated with severe comorbid conditions in patients with acute esophageal necrosis.

## CONCLUSION

AEN is a rare and severe cause of upper gastrointestinal bleeding and can be life-threatening. It concerns mostly elderly patients, polyvascular with multiple comorbidities such as diabetes, hypertension, and chronic kidney insufficiency. The treatment is based on the management of the underlying condition associated with high-dose proton pump inhibitors and supportive care.

## REFERENCES

1. Goldenberg SP, Wain SL, Marignani P. Acute necrotizing esophagitis. Gastroenterology. 1990.
2. Gurvits GE. Black esophagus: acute esophageal necrosis syndrome. World J Gastroenterol. 2010.
3. Gurvits GE *et al.,* Acute esophageal necrosis: a rare syndrome. J Gastroenterol. 2007.
4. Dias E *et al.,* Diagnosis and management of acute esophageal necrosis. Ann Gastroenterol. 2019.
5. Sheikh AB *et al.,* Acute esophageal necrosis: review of pathogenesis and management. Cureus. 2022.
6. Abdullah HM *et al.,* Acute esophageal necrosis: systematic review. World J Gastrointest Endosc. 2019.
7. Lahbabi M *et al.,* Acute esophageal necrosis: case report and literature review. Case Rep Gastroenterol. 2013.
8. Khan H *et al.,* Acute esophageal necrosis: a view in the dark. Case Rep Gastroenterol. 2019.
9. Choksi V *et al.,* Black esophagus: a rare but deadly disease. Case Rep Gastrointest Med. 2017.
10. Day A *et al.,* Acute oesophageal necrosis: case report and literature review. Int J Surg Case Rep. 2010.