

Gurvits Syndrome or “*Black esophagus*”: A Rare Cause of Upper Gastrointestinal Bleeding. A Case Report and Review of the Literature

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Abstract

Introduction: Acute esophageal necrosis (AEN), or “black esophagus,” is a rare but serious condition that mainly occurs in elderly patients with vascular frailty and significant comorbidities. It is thought to result from acid injury combined with esophageal ischemia. It frequently presents with upper gastrointestinal bleeding. Diagnosis relies on esophagogastroduodenoscopy (EGD), which shows diffuse, circumferential black discoloration of the esophageal mucosa, predominantly distal and ending sharply at the Z-line. AEN may progress to severe complications, including esophageal stricture and perforation, and remains associated with high mortality. **Case Presentation:** We report the case of a 50-year-old man with alcohol-related liver cirrhosis, still actively drinking and not under medical follow-up, admitted for mild acute alcoholic pancreatitis. His course was complicated by delirium tremens in the setting of abrupt alcohol withdrawal. Clinically, he presented with blackish vomiting and melena. Laboratory tests revealed severe anemia (hemoglobin 6.3 g/dL), acute kidney injury, and severe hypokalemia requiring admission to the intensive care unit. EGD revealed grade D ulceronecrotic esophagitis according to the Los Angeles classification, without active bleeding, consistent with black esophagus syndrome. Biopsies were deferred given the acute bleeding context. Caustic ingestion was ruled out by history. The patient was managed with fluid resuscitation, parenteral nutrition, correction of fluid and electrolyte disturbances, high-dose intravenous proton pump inhibitor (PPI) therapy followed by double-dose oral PPI combined with sucralfate, and initial fasting. Follow-up EGD on day 8 showed marked improvement without esophageal stricture. The outcome was favorable, with resumption of oral feeding, continuation of PPI therapy for eight weeks, and referral to an addiction medicine clinic. **Conclusion:** Our case shows that early and optimal management can lead to healing of esophageal lesions. Control of comorbidities is an essential component of treatment, together with supportive resuscitative measures and combined PPI and sucralfate therapy. The outcome is often favorable, but mortality remains high owing to the frail background frequently associated with this condition.

Keywords: Melena; EGD; esophageal necrosis; black esophagus; PPI.

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INTRODUCTION

Acute esophageal necrosis (AEN), also known as *black esophagus* or Gurvits syndrome, is an uncommon entity with a potentially severe prognosis, first described by Goldenberg *et al.*, in 1990 [1]. It is characterized by diffuse necrosis of the esophageal mucosa, producing a characteristic circumferential black appearance on esophagogastroduodenoscopy (EGD) [1,2], and most often presents with upper gastrointestinal bleeding. Its incidence is estimated at 0.01% to 0.28% in published series [2,3]. It mainly affects elderly patients with multiple comorbidities, including cardiovascular disease, diabetes mellitus, chronic kidney disease, shock, or any other state of systemic hypoperfusion [2–4].

CASE PRESENTATION

A 50-year-old man had a medical history of alcohol-related cirrhosis with portal hypertension, with ongoing alcohol use and no medical follow-up, peripheral polyneuropathy, and previous alcohol withdrawal seizures. He was admitted to the emergency department for acute alcoholic pancreatitis and delirium tremens, the latter occurring after abrupt cessation of alcohol intake. The clinical picture developed in a context of general deterioration and was accompanied by blackish vomiting. On admission, the patient was pale, and digital rectal examination revealed melena. He had a generalized tonic-clonic seizure that resolved after intravenous diazepam. Laboratory tests showed an inflammatory syndrome, anemia (hemoglobin 6.3 g/dL),

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thrombocytopenia, acute kidney injury, hyperlactatemia, serum lipase at 24 times the upper limit of normal, abnormal liver function tests, and fluid and electrolyte disturbances, including severe hypokalemia requiring admission to the intensive care unit. Initial management included fluid resuscitation, parenteral nutrition, antibiotic prophylaxis with a third-generation cephalosporin (3GC) for 5 days to prevent bacterial translocation, a proton pump inhibitor (PPI) as a continuous infusion at 8 mg/h, octreotide (50 µg bolus followed by continuous infusion at 25 µg/h), transfusion

of two units of packed red blood cells (PRBCs), correction of hypokalemia, and fasting. EGD revealed grade D ulceronecrotic esophagitis according to the Los Angeles classification, without active bleeding (**Figure 1**). Biopsies were deferred given the acute bleeding context. Caustic ingestion was ruled out by history. Octreotide was discontinued, whereas the continuous PPI infusion was maintained for 5 days and then switched to double-dose oral PPI, with the addition of sucralfate, three sachets daily.

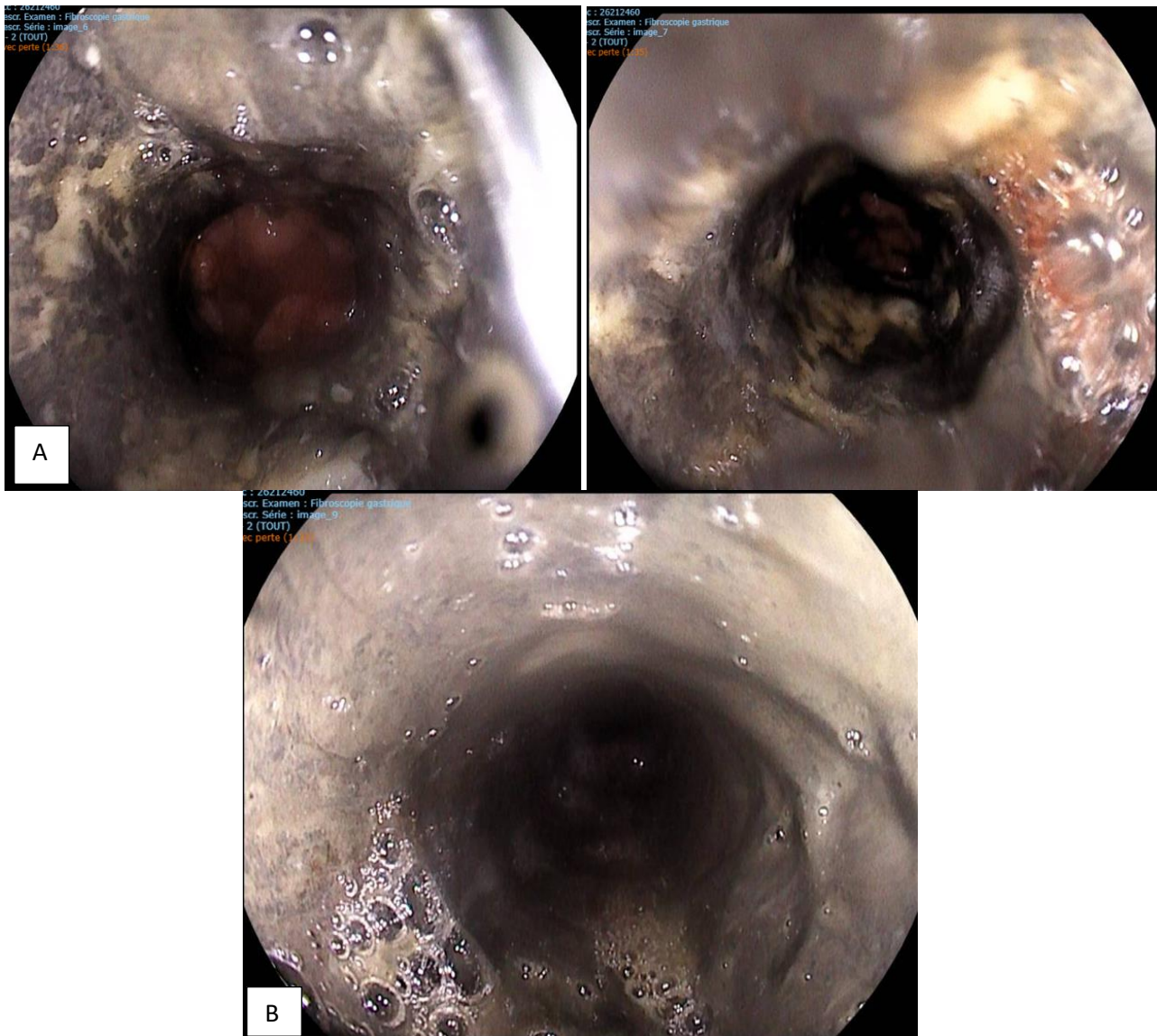


Figure 1: Endoscopic appearance of acute esophageal necrosis: A. Necrosis ending abruptly at the esophagogastric junction; B. Circumferential necrosis and ulceration of the middle and lower thirds of the esophagus

Follow-up EGD performed on day 8 of PPI therapy showed marked improvement of the necrotizing esophagitis, without esophageal stricture (**Figure 2**).

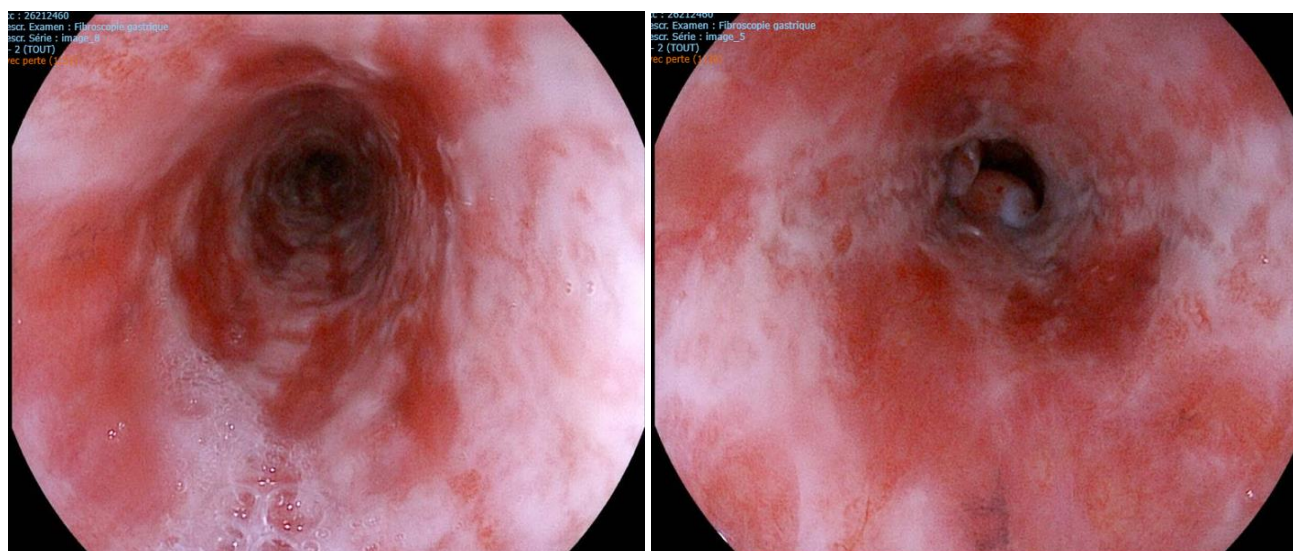


Figure 2: Healing appearance of the esophageal mucosa after PPI therapy

The outcome was favorable, with no recurrence of melena or blackish vomiting. The patient resumed a normal diet. PPI therapy was continued for eight weeks, and an addiction medicine consultation was scheduled to support long-term abstinence.

DISCUSSION

Acute esophageal necrosis (AEN) is a rare and serious condition. It is characterized by necrosis of the esophageal mucosa, resulting in blackish ulcerated and necrotic mucosal lesions on EGD [5]. It most often involves the lower third of the esophagus and stops at the esophagogastric junction [6]; it mainly affects elderly men, with a median age of 75 years and a male-to-female ratio of 3:1 [6–8].

Our case is notable for the occurrence of AEN in a relatively young patient, which may be explained by a frail background combining several risk factors, including chronic alcohol use complicated by cirrhosis and acute pancreatitis, fluid and electrolyte disturbances, acute kidney injury, and a context of hypoperfusion.

The pathophysiology of AEN is multifactorial and involves a combination of esophageal wall ischemia, extensive exposure to gastric acid-peptic reflux, and impaired esophageal mucosal defense mechanisms [2,3]. This combination explains the predominance of lesions in the distal third of the esophagus, a region particularly vulnerable to hypoperfusion [2].

AEN is promoted by various conditions, particularly vascular disorders that may compromise esophageal perfusion and gastrointestinal diseases that may impair mucosal integrity. The main associated comorbidities are diabetes mellitus, malignancy, hypertension, alcohol use disorder, coronary artery disease, and gastroesophageal reflux disease [3,9].

Several triggering factors have also been identified, including hemodynamic disturbances such as hemorrhage or dehydration, and infections, particularly pneumonia and sepsis [10]. Reported infectious etiologies include viral infections with *cytomegalovirus* [11] and *herpes simplex virus* [12], bacterial infections with *Klebsiella pneumoniae* [13], and fungal infections with *Candida* [14]. Iatrogenic causes may also be involved, either drug-related, notably nonsteroidal anti-inflammatory drugs [15] or certain antibiotics such as cefoxitin, gentamicin, and ampicillin [16], or procedure-related, particularly after thoracic surgery [17].

Upper gastrointestinal bleeding is the main presenting feature of AEN, found in 71% of cases. Blackish “coffee-ground” vomiting is also observed in 18.2% of cases and reflects the presence of blood altered by gastric acid. Other symptoms may be associated, such as abdominal pain, nausea, or dysphagia. Rare asymptomatic cases have been reported [5,15,18].

EGD is the reference examination to confirm the diagnosis of AEN, showing circumferential black discoloration of the mucosa, preferentially located in the lower third (97% of cases) and ending abruptly at the Z-line [18].

Biopsies are not mandatory for diagnosis but can confirm it [19]. They are particularly indicated when infection or bacterial, viral, or fungal superinfection is suspected, and in immunocompromised patients [14]. Histopathological examination reveals severe necrosis of the mucosa and submucosa, whereas transmural involvement remains exceptional, unlike caustic injury [2]. An inflammatory infiltrate with partial destruction of the adjacent muscularis propria may also be observed. Biopsies also help exclude differential diagnoses of AEN, including primary esophageal melanoma, esophageal melanosis, and *acanthosis nigricans*.

[2,20,21]. Finally, caustic ingestion must be systematically sought and ruled out by careful history-taking.

Management of AEN is mainly conservative, based on hemodynamic stabilization, treatment of the underlying cause, esophageal rest with exclusive parenteral nutrition, and intravenous PPI administration [2,9]. The addition of sucralfate to PPIs appears beneficial owing to its protective effect on the esophageal mucosa [18,19]. Treatment duration depends on the clinical course. Upon improvement, oral feeding and oral PPIs can be gradually resumed. Oral feeding and nasogastric tube placement should be avoided initially [10].

The role of antibiotics remains uncertain. Empirical broad-spectrum parenteral antibiotic therapy may be considered in cases of documented esophageal infection, suspected perforation or mediastinitis, and in immunocompromised patients (AIDS, asplenia, transplantation, cirrhosis, etc.) [12,16]. To date, no guidelines specify the choice or duration of antibiotic therapy.

The most serious complication of AEN is esophageal perforation, with an estimated incidence of 7% [18]. Another frequent complication is esophageal stricture, reported in 10.2% of cases, which may develop between the 1st and 4th weeks of the disease course. It is treated with antisecretory therapy and endoscopic dilation. AEN is associated with a mortality rate of approximately 32%, mainly related to the patients' general condition and comorbidities [18].

CONCLUSION

Acute esophageal necrosis is a rare entity whose prognosis depends more on the underlying condition than on the extent of the esophageal lesions. Despite progress in understanding this condition over recent decades, its rarity explains the lack of specific treatment guidelines and the low level of available scientific evidence [2,9].

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