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About A New Case Of Black Esophagus

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Acute esophageal necrosis (AEN), commonly known as black esophagus, is a rare clinical condition resulting from a combination of ischemic disorders and thromboembolic lesions of the esophagus. It is characterized by a circumferential black appearance of the esophagus. Treatment consists in correcting the causes of AEN, conditioning the patient and suppressing gastric acidity. The true incidence of AEN is uncertain, due to the lack of reliable data and the wide variability of the population studied. As a result, many cases appear to be under-diagnosed, particularly in people admitted with refractory shock [1].
In this article, we describe a new case of black esophagus revealed by upper gastrointestinal bleeding.

I. Case Report

A 77-year-old woman with a history of hypertension and dyslipidemia and treated for cirrhosis of undetermined etiology that never decompensated, poorly monitored, last FOGD in 2019 objectified stage III esophageal varices and GOV 1 with red signs, presented to the emergency department in a state of shock with hematemesis and melena associated with abdominal pain. She was apyretic, hypotensive with a BP of 78/50 mmHg, tachycardic at 125 beats/minute, tachypneic at 20 cycles/minute, and saturated well on ambient air. Her blood pressure did not respond to filling, so she was admitted to the outpatient department and put-on vasoactive drugs, sandostatin and PPIs. Her biological tests showed anemia at 5g/dl and thrombocytopenia at 50,000. An urgent initial gastroscopy showed an esophageal mucosa lined with solid non aspirable food preventing progression, with the impossibility of pushing food towards the stomach, with varicose cords without active bleeding glimpsed through. A second gastroscopy was carried out on the same day after a 6-hour fast, and initially revealed a tongue with a cardboard-like, depilated appearance (image 3). After introduction of the endoscope, an ulceration in the laryngopharynx was observed, and passage through Killian's mouth revealed an esophageal mucosa with a blackish appearance (image 1 and 2), making it impossible to progress beyond 20 cm from the dental arches. An abdominal Angio scan was performed, which revealed thrombosis of the spleno mesenteric trunk, extending to the portal trunk and its branches, with peritoneal effusion (image 4). The patient was admitted to intensive care for refractory shock. Despite rapid treatment, she suffered cardiorespiratory arrest.

Image 1 : Endoscopic aspect of acute esophageal necrosis with varicose cords

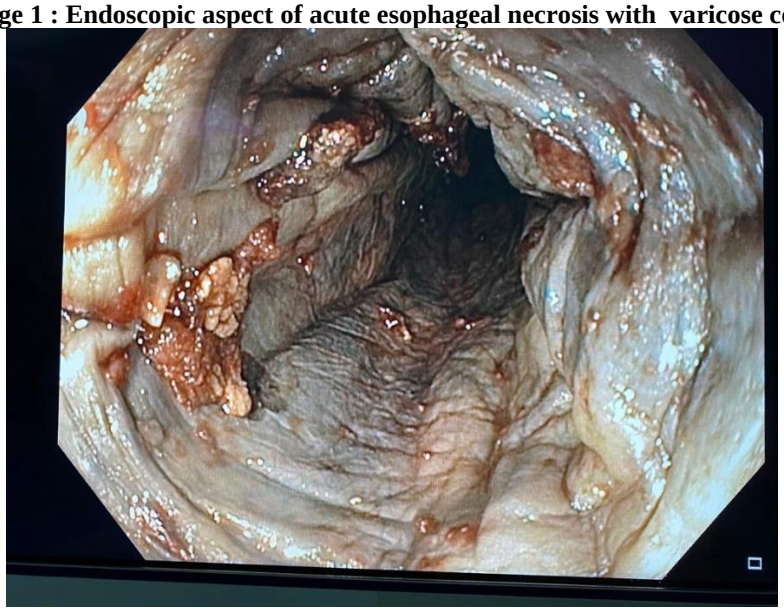


Image 2 : Endoscopic aspect of acute esophageal necrosis with varicose cords

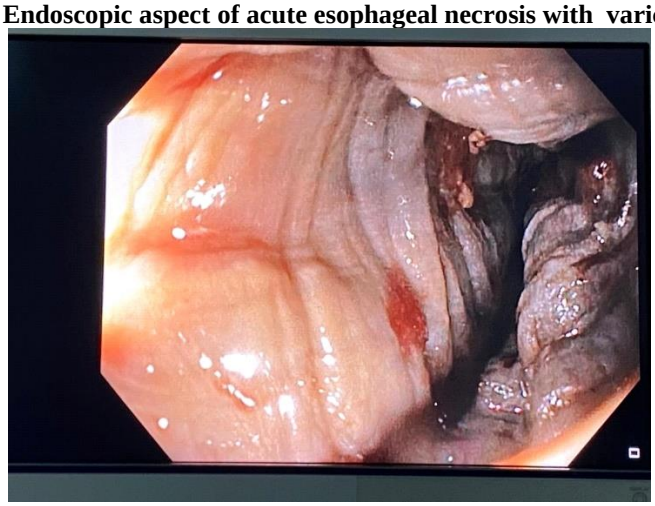


Image 3 :Aspect of tongue with a cardboard-like and depilated appearance

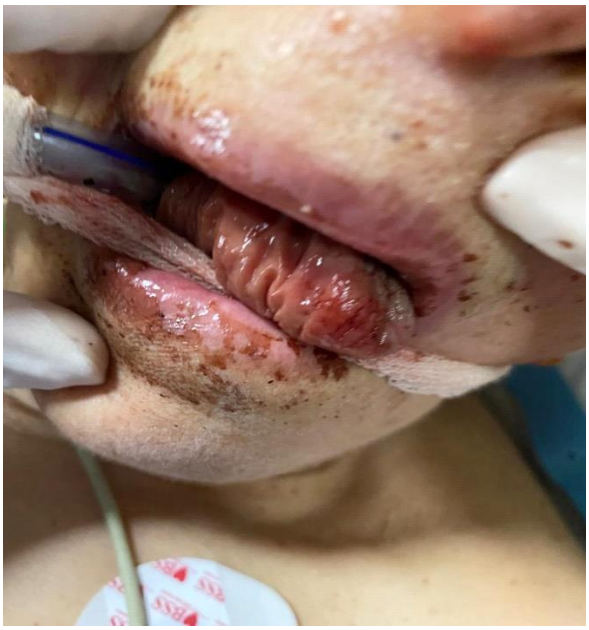
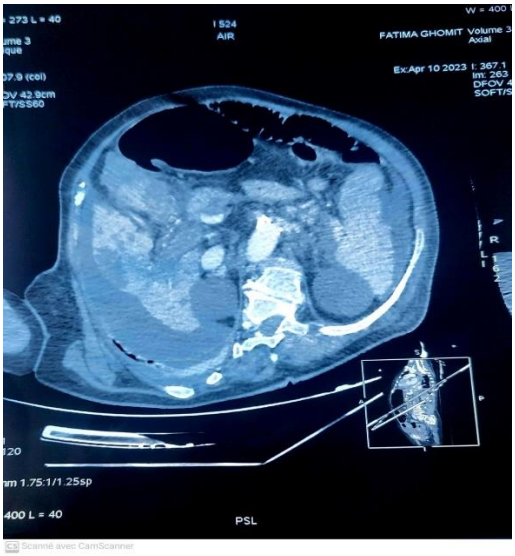


Image 4 : Angio scan image of thrombosis of the spleno-mesenteric trunk, extending to the portal trunk and its branches



II. Discussion

Acute esophageal necrosis (AEN) is a rare condition characterized by a diffuse circumferential black appearance of the esophageal mucosa [2]. AEN can affect any age group and considered to be multifactorial in origin. Tissue hypoperfusion and thromboembolic conditions play an important role in the etiopathogenesis of AEN, including shock, sepsis, congestive heart failure, acute blood loss, hypothermia, coagulopathy, solid or hematological malignancies, antiphospholipid syndrome (APS) and atherosclerosis [4-6]. Patients with a history of coronary artery disease, diabetes, high blood pressure, malignancy and alcohol use disorders are at increased risk of developing AEN. Although it can affect a variable length of the esophagus, it most often affects the distal esophagus and avoids the gastro-esophageal junction, as the lower esophagus has a lower degree of vascularization than the proximal and middle parts [6,7]. Vascular involvement of the distal esophagus also explains the duodenal pathology commonly observed in AEN, namely duodenal bulb ulcers, erosions and inflammation [8].Our patient had many risk factors that could have predisposed her to the development of AEN, including age, a history of diabetes, dyslipidemia, cirrhosis, and especially ischemic disorders due to hemorrhagic shock following variceal rupture in addition to thromboembolic disorders. She had no history of exposure to chemical agents.

The important differential diagnosis of esophageal black appearance includes malignant melanoma, melanosis, acanthosis nigricans and coal dust deposits [2]. Esophageal perforation is the most serious complication of AEN, seen in less than 7% of cases, and should be suspected in patients whose condition deteriorates rapidly [3]. Perforation can lead to mediastinitis, mediastinal abscess, empyema, and sepsis. Other complications of AEN include bleeding, superinfection and stenosis or stricture formation [7].

Treatment of AEN focuses on treating the underlying pathology, with adequate supportive care including volume resuscitation and blood and platelet transfusions (to maintain hemoglobin at >7 g/dL and platelets at >50,000/mm3).

The patient should initially be kept sedated, and the diet slowly increased.

Patients should not be placed on a nasogastric tube to avoid perforation [9]. Medical treatment includes gastric acid suppression with proton pump inhibitors (PPIs) and mucosal protection with sucralfate [6]. Intravenous PPIs are preferable initially and can be replaced by an oral form when adequate. Antibiotic treatment is initiated in the event of positive esophageal cultures and the presence of multinucleated giant cells or inclusion bodies on histopathological evaluation (if a biopsy is performed) [7]. Surgical intervention for EAN is reserved for esophageal perforations accompanied by mediastinitis and abscess formation. Endoscopic balloon dilatation may be necessary for patients with symptomatic esophageal stenosis or stricture [6].

III. Conclusion

The literature reports several cases of this type.

Ischemia appears to be the main pathogenic mechanism. Prognosis depends on the patient's general condition rather than on the appearance of esophageal lesions.

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