Available online at www.sciencedirect.com

ScienceDirect

Journal of Acute Medicine 5 (2015) 107–108

www.e-jacme.com

Case Report

Black esophagus

Chiara Casarsa, Filippo Mearelli*, Michela Zanetti, Gianni Biolo

Internal Medicine Division, Cattinara Hospital, Trieste, Italy

Received 19 September 2014; revised 26 May 2015; accepted 7 September 2015
Available online 14 November 2015

Abstract

Black esophagus is an uncommon clinical entity and its pathogenesis remains unknown. Clinical presentation is usually characterized by the combination of hematemesis and circumferential darkness of the mucosa in the distal esophagus. This case illustrates an atypical presentation of the disease. Despite its rarity, black esophagus should be considered in the differential diagnosis of acute upper gastrointestinal bleeding, especially in patients with predisposing factors.

Keywords: black esophagus; esophageal necrosis; hematemesis

1. Introduction

Acute upper gastrointestinal hemorrhage is a serious medical condition. Common causes include peptic ulcer disease, gastrointestinal tumors, gastroesophageal varices, Mallory–Weiss tears, angiodysplasia, and Dieulafoy's lesions. Besides these diseases, acute esophageal necrosis or black esophagus is an uncommon medical syndrome that usually presents with upper gastrointestinal hemorrhage.

2. Case report

A 64-year-old man was admitted to our hospital because of atypical chest pain. His past medical history included hypertension, diabetes mellitus, lower limb arteriopathy obliterans, and provoked pulmonary embolism (hemicolectomy for cancer). His medications consisted of acenocoumarol, insulin, and enalapril. At admission, he was in mild respiratory distress. Electrocardiography and chest X-ray were normal. There was no evidence of hypercoagulability (international normalized ratio = 1.7).

Due to a likely pretest probability of pulmonary embolism, computed tomography (CT) angiography of the lung was performed, but no vascular defects were detected. During the period of observation following CT, the patient abruptly developed acute hematemesis without signs of hemodynamic instability, which required phytonadione and packed red blood cells. An urgent upper endoscopy was suggestive of black esophagus (BE; Figure 1). Intravenous proton pump inhibitors, histamine H2 receptor blockers, sucralfate, and hydration were started; carbapenems were empirically added to the therapy because of the septic aspect of the patient. On further questioning, the patient denied ingesting caustic substances or alcohol or having symptoms of gastroesophageal reflux disease. Bowel rest was recommended for 5 days. The results of human immunodeficiency virus, cytomegalovirus, and Epstein–Barr virus tests were negative. Thoracic–abdominal CT and colonoscopy did not show any relapse of colonic cancer. Two weeks after admission, upper gastrointestinal endoscopy was performed to exclude common complications of BE, and complete resolution of the macroscopic picture was noted (Figure 2). Two weeks later, esophageal histology confirmed mucosal and submucosal necrosis and ruled out primary cancer. Follow-up cultures for bacteria, mycobacteria, and fungi were negative.

* Corresponding author. Internal Medicine Department, Azienda Ospedaliera Univeristaria Ospedali Riuniti, Strada di Fiume in 447, Trieste, Italy.
E-mail address: filippome@libero.it (F. Mearelli).

http://dx.doi.org/10.1016/j.jacme.2015.09.004

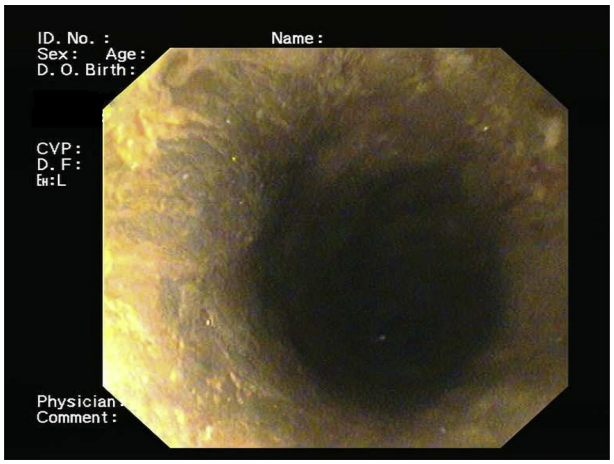

Figure 1. Black discoloration of the distal esophagus, which is highly suggestive of acute esophageal necrosis.

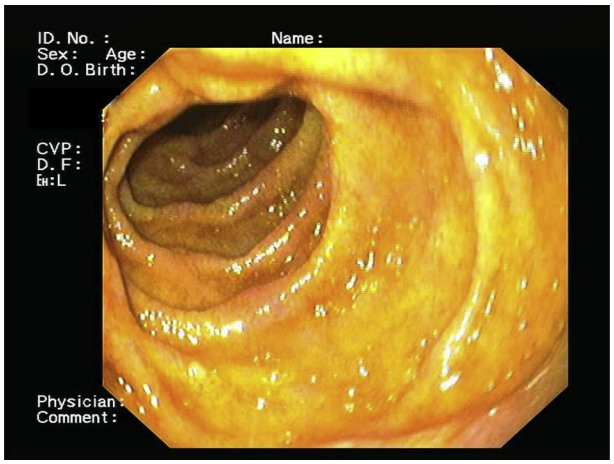

Figure 2. Normalization of the endoscopic findings.

3. Discussion

Acute esophageal necrosis (AEN) has been referred to by multiple names, including BE and necrotizing esophagitis.[1,2] It is characterized by endoscopic findings of circumferential BE with or without exudates, distal esophageal involvement that can continue proximally, but ends roughly at the gastroesophageal border, and by histological findings of uniform severe necrosis of the esophageal mucosa and submucosa without specific causative agents.[3] Ingestion of caustic substances or other injurious agents may mimic the same endoscopic features, but have to be excluded. The pathogenesis of AEN remains unknown, although ischemia seems to play a key role[4]: in fact, AEN occurs mainly in the distal third of the esophagus, which is relatively hypovascular if compared with other esophageal segments. Furthermore, AEN appears to be more common in older men with diseases that may predispose to hypoperfusion (atherosclerotic disease). Other comorbidity, like hypercoagulable state, cancer, and antiphospholipid syndrome, as well as localized infection (mainly *Candida* spp. and herpes virus) could be implicated in the pathogenesis of AEN. These initial events predispose the esophageal mucosa to topical injury caused by reflux of acid and pepsin as in gastric outlet obstruction. The damage may be exacerbated by conditions such as malnutrition, which causes an overall reduction in the mucosal defense against gastric contents. Other possible triggers implicated in the pathogenesis of AEN are gastric volvulus, paraesophageal hernia, neoplastic disease, diabetic ketoacidosis, hyperglycemia, sepsis, recurrent shock state due to hemorrhage/pump failure, aortic dissection, polyarteritis nodosa, pancreatitis, direct trauma, and prolonged vomiting.[5] It is unlikely that one single etiological factor is responsible for the disease: the presumed overall understanding is that of a “two hit” phenomenon. The diagnosis of AEN is established by upper gastrointestinal endoscopy. Biopsies should be obtained to differentiate AEN from other conditions in which the esophageal mucosa appears darkened, such as melanosis, melanoma, pseudomelanosis acanthosis nigricans, pseudomembranous esophagitis, and intraepithelial hemorrhage of the esophagus.[6]

Unlike caustic esophagitis, whose treatment is often surgical, the treatment of AEN is primarily medical and directed to optimize vascular flow, suppress acid production, treat esophageal infections, and control comorbidity. Oral intake should be avoided for at least 24 hours. The risk of perforation restricts the use of nasogastric tubes in patients with AEN, if they do not have vomiting. Sucralfate should be considered as adjunctive therapy to the combination of proton pump inhibitor and histamine H2 receptor blockers for its cytoprotective effects, its ability to bind pepsin, and to stimulate mucus secretion. A decision about antibiotic treatment is usually made on an individual basis and should be reserved for septic patients: there is insufficient evidence in favor of its prophylactic use. The high mortality related to the underlying illnesses of the patients with AEN (and the limited number of reported cases) obscures a detailed understanding of its natural history. However, deaths secondary to complications such as esophageal perforation, mediastinitis, or esophageal infection in immunocompromised individuals are seen in <6% of the patients.[6]

Conflict of interest

The authors have no conflict of interest to declare. The study was not supported by departmental research funds.

References

1. Obermeyer R, Kasirajan K, Erzurum V, Chung D. Necrotizing esophagitis presenting as a black esophagus. *Surg Endosc*. 1998;12:1430–1433.
2. Grudell AB, Mueller PS, Viggiano TR. Black esophagus: report of six cases and review of the literature, 1963–2003. *Dis Esophagus*. 2006;19:105–110.
3. Moretò M, Ojembarrena E, Zaballa M. Idiopathic acute esophageal necrosis: not necessarily a terminal event. *Endoscopy*. 1993;25:534–538.
4. Goldemberg SP, Wain SL, Marignani P. Acute necrotizing esophagitis. *Gastroenterology*. 1990;98:493–496.
5. Gurvits GE. Black esophagus: acute esophageal necrosis syndrome. *World J Gastroenterol*. 2010;16:3219–3225.
6. Grigorly EG, Shapis A, Lau N, Gualtieri N, Robilotti J. Acute esophageal necrosis: a rare syndrome. *J Gastroenterol*. 2007;42:29–38.