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Black esophagus

Mariana Leite; Cristina Silva; Marta Moreira; Francisco Fazeres

***Corresponding Author: Mariana Leite**
General Surgery Department, Hospital Santa Luzia, Viana do Castelo, Portugal
Phone: 0035-1969-526-676; Email: marianalsleite@gmail.com

Abstract

Acute esophageal necrosis is a rare clinical disorder that is defined endoscopically by a circumferential black-appearing esophageal mucosa with nearly universal involvement of the distal esophagus and abrupt transition at the gastroesophageal junction. The authors present a case of a 65-year-old male hospitalized for acute chronic pancreatitis who underwent an esophagogastroduodenoscopy that showed circumferential black mucosa in the distal esophagus – black esophagus. He received an intravenous proton pump inhibitor and was given nothing by mouth, which allowed the resolution of endoscopic findings.

Keywords

acute esophageal necrosis; black esophageal mucosa.

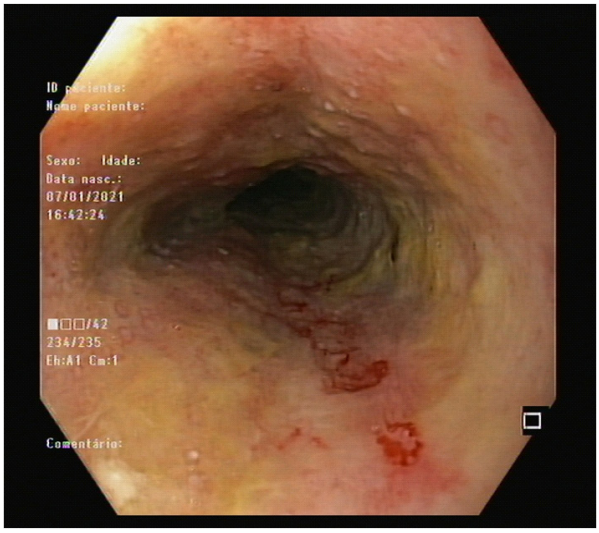
Introduction

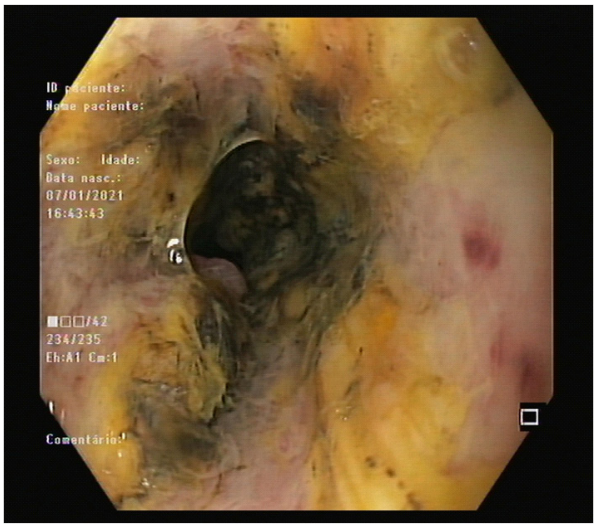
"Black esophagus" or acute esophageal necrosis is a rare clinical disorder, with an incidence of 0.01-0.28% of patients undergoing esophagogastroduodenoscopy [1,2]. It is defined endoscopically by a circumferential black-appearing esophageal mucosa with nearly universal involvement of the distal esophagus and abrupt transition at the gastroesophageal junction, with variable proximal extension [2]. This characteristic endoscopic appearance is sufficient to establish the diagnosis; biopsy is supportive but is not required to establish the diagnosis [2].

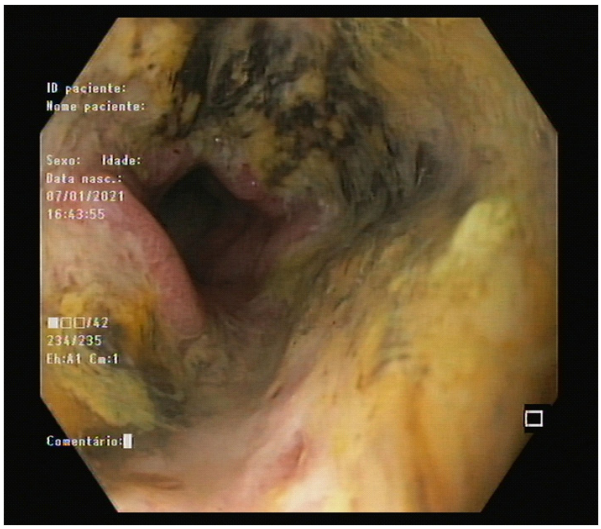
Case Presentation

A 65-year-old male with history of chronic liver disease and chronic pancreatitis of alcoholic etiology went to the hospital complaining of abdominal pain and nausea. He was hospitalized for acute chronic pancreatitis at general surgery ward for symptomatic control. After 5 days he complained worsening of nausea and vomiting, and because of that he underwent an esophagogastroduodenoscopy that showed circumferential black mucosa in the distal esophagus (shown in Figures 1, 2 and 3). This endoscopic appearance is consistent with acute esophageal necrosis, and therefore he received an intravenous proton pump inhibitor and was given nothing by mouth. Following discharge, gastric acid suppression with an oral pump

proton inhibitor was continued; however, the patient was subsequently lost to follow-up.







Figures 1,2,3: Endoscopic image of distal esophagus showing dark mucosal discoloration consistent with acute esophageal necrosis.

Discussion

Acute esophageal necrosis clearly shows sex and age predilection: men are four times more commonly affected than women, and the peak incidence occurs in the sixth decade of life [3]. The pathophysiology of acute esophageal necrosis is likely multifactorial and usually results from a combination of tissue hypoperfusion, impaired local defense barriers, and massive influx of gastric contents [2,3]. Patients usually present multiple comorbidities, such diabetes mellitus, hypertension, alcohol abuse, chronic kidney disease, coronary heart disease, dyslipidemia, peripheral artery disease, malignancy, liver cirrhosis, gastroesophageal reflux disease, congestive heart failure, and chronic obstructive pulmonary disease [2]. Potential complications of acute esophageal necrosis include esophageal perforation, superinfection, and strictures [1]. There is no specific therapy for acute esophageal necrosis: the mainstay of therapy consists in correction of the precipitating conditions and supportive care (esophageal rest and gastric acid suppression) [1,2]. Prognosis of “black esophagus” largely depends on coexisting medical conditions; the overall prognosis is poor with nearly one third of patients succumbing to the underlying critical illness [3].

References

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Authors Information: Mariana Leite[1]; Cristina Silva[1]; Marta Moreira[2]; Francisco Fazeres[1]
[1]General Surgery Department, Hospital Santa Luzia, Viana do Castelo, Portugal.
[2]Gastroenterology Department, Hospital Santa Luzia, Viana do Castelo, Portugal.

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