Case report

Acute Esophageal Necrosis: A Case of Black Esophagus with DKA

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Abstract

Acute esophageal necrosis is a rare cause of upper gastrointestinal bleeding, usually caused by hypoperfusion. In this report, we present a 34-year-old male patient suffering from acute esophageal necrosis presenting as hematemesis in a patient with Diabetic ketoacidosis. Diabetic ketoacidosis is rarely reported as a cause of acute esophageal necrosis and it is vital to diagnose it in a patient with Diabetic Ketoacidosis as a potential cause of mortality.

Keywords: Acute esophageal necrosis, black esophagus, diabetic ketoacidosis.


Introduction

Acute esophageal necrosis (AEN) has been referred to by multiple names, including black esophagus and necrotizing esophagitis, and is a rare endoscopic finding since its first description by Goldenberg in 1990.1,2 The incidence of esophageal necrosis varies from 0.008% to 0.2% of endoscopic cases.1 Its etiology is multifactorial, namely male sex, diabetes, renal insufficiency and et cetera.1 Ischemia is the commonest cause of AEN, but preferential esophageal involvement despite its rich blood supply is amazing.3

Case Report

A 34-year-old male patient presented with hematemesis for one day. He also had abdominal pain, nausea, vomiting associated with fatigue for two days and concomitant disorientation. He was on irregular insulin treatment for two years and had received none for one day. Examination revealed drowsiness, moderate dehydration with blood pressure of 80/50 mmHg, PR of 120/min, RR of 18/min and oral temperature of 36.2°C with no other positive findings except for oral candidiasis. The patient had normal hemoglobin with leukocytosis and neutrophilia (WBC: 16700, neutrophil: 88%). Biochemical tests showed hyperglycemia (glucose 577 mg/dL), normal liver enzymes with normal PT and PTT. He was diagnosed as a case of diabetic ketoacidosis (DKA) and appropriate treatment was started. For his hematemesis, esophagogastroduodenoscopy revealed no abnormality except for black esophagus with the proximal third spared and biopsy was taken (Figure 1). Rapid urease test was negative. Pathologic findings of esophagus included necrotic material with infiltration of lymphoplasmacytic cells associated with PMN leukocytes and eosinophils (Figure 2). Finally, based on endoscopic and microscopic findings, black esophagus, acute esophageal necrosis, was diagnosed.

Discussion

Black esophagus or acute esophageal necrosis (AEN) is a rare disease with an incidence of 0.01%–0.2% in autopsies and clinical trials.4 Men are affected four times more than women with its peak incidence in the sixth decade of life and the average age of 67 years.5 Its pathogenesis is unknown but ischemia plays a role which is supported by the evidence of histopathologic and clinical data.6,7 Diabetic ketoacidosis is rarely reported as a cause of acute esophageal necrosis and it is vital to diagnose it in a patient with Diabetic ketoacidosis as a potential cause of mortality. Therefore, in a DKA patient with upper GI bleeding, this diagnosis should be considered. The etiology of AEN is unknown but the condition is thought to be multifactorial,2,3,6 namely vascular/hypoperfusion (atherosclerotic vascular disease, shock), chemical (acid reflux, alcohol, antibiotics, corrosives), metabolic (hyperglycemia, uremia, sepsis, lactic acidosis), infectious, mechanical and co-morbidities of other conditions (diabetes mellitus, renal insufficiency).8 The commonest manifestation reported in the majority (>80%) of cases and as seen in our case is upper GI bleeding. Other symptoms may vary from abdominal pain to asymptomatic black esophagus that was found during a percutaneous gastrostomy tube placement.1,3

Diagnosis is made with upper endoscopy, showing the characteristic black discoloration with a sharp transition to normal mucosa of the gastroesophageal junction.1 Biopsies should be taken to differentiate it from other causes of black esophagus.1 Microscopic study reveals severe necrosis of the mucosa and submucosa.4 Most patients recover with adequate hydration and treatment including correction of underlying disease, intravenous proton pump inhibitors and sucralfate suspension.1 With appropriate treatment, mortality specific to AEN is about 6% while the overall mortality may reach 32% due to the underlying condition.1,4 Esophageal perforation occurs in less than 7% while esophageal strictures are seen in more than 10% of patients.1 AEN is a rare cause of upper GI bleeding with high mortality in comparison to
other causes, both caused directly by it or indirectly due to its underlying disease, but early diagnosis and treatment may save the patient’s life as in our case. Therefore, it is necessary for clinicians and pathologists to have sensitization and awareness about these diagnostic challenges.

References


**Figure 1.** Black mucosa of lower two-third of esophagus.

**Figure 2.** Photomicrograph shows extensive necrosis and absence of esophageal epithelium, except for a small focus in upper part (arrow). (Hematoxylin & Eosin stain, ×100).