Fellows' Corner

by Cody B. Barnett and Jorge L. Herrera

A 57-year-old male presented to the emergency department with a two week history of worsening anterior chest pain, epigastric pain, nausea and vomiting. He admitted to coffee ground emesis prior to presentation as well as progressive dysphagia to solids over the last several months. He had experienced chronic epigastric pain for the prior 6 months associated with a weight loss of approximately sixty pounds over that same period of time. He denied any recent caustic substance ingestion or difficulty swallowing. He admitted to using non-steroidal anti-inflammatory medications and had a lifelong history of ethanol abuse and tobacco dependence. He had not seen a physician for many years prior to presentation.

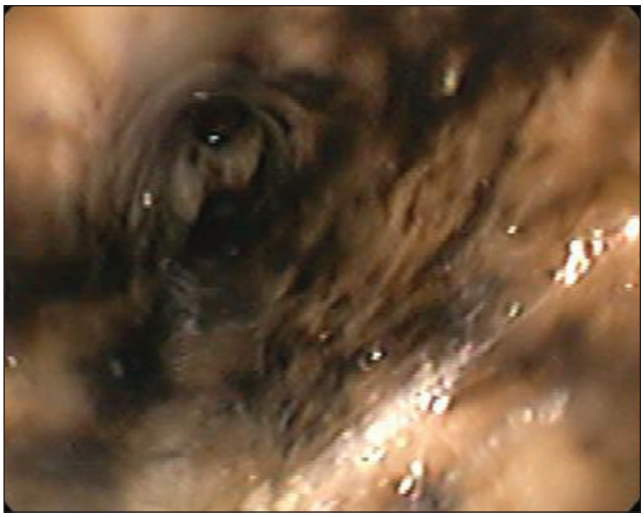

Figure 1.

Cody B. Barnett, M.D. and Jorge L. Herrera, M.D., University of South Alabama College of Medicine, Mobile, AL.

Physical exam revealed a thin, malnourished man. Vital signs included a temperature of 96.8 degrees Fahrenheit, heart rate of 109 beats per minute, respiratory rate of 18 breaths per minute, and blood pressure of 114/76 mmHg. No lymphadenopathy was palpable. Inspiratory crackles were evident in both lung fields with decreased breath sounds at the left base. Abdominal exam was tense, bowel sounds were present and there was mild tenderness in the epigastric area. Rectal exam revealed brown stool which tested negative for occult blood.

Laboratory values included a white blood cell count of 17,000/mm^3 with a differential of 74% granulocytes and 23% bands. Hematocrit was 34% and platelets were 633,000/mm^3. Blood urea nitrogen of 16 mg/dL (normal 7–18 mg/dL) and creatinine of 1.1 mg/dL (normal 0.6–1.3 mg/dL). Glucose and calcium

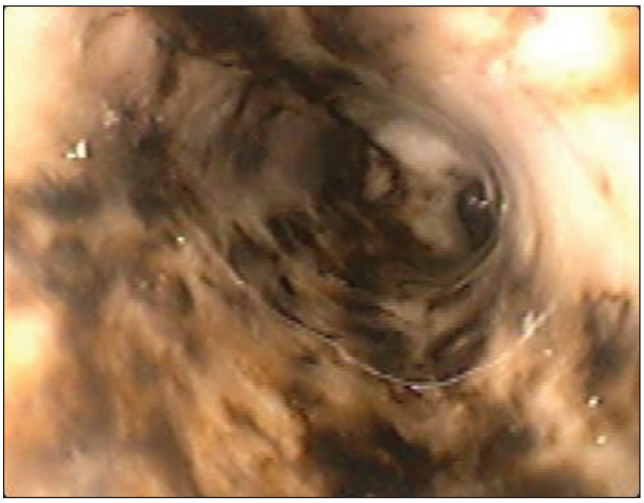

Figure 2.

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were normal. Albumin was 1.9 g/dL, with the remainder of hepatic functional panel and associated enzymes within normal limits. Lipase was elevated at 2715 U/L. Chest radiograph showed left pleural effusion and pneumonia. CT of the abdomen and pelvis with contrast showed extensive calcifications of pancreas and massive ascites with normal appearing liver. Paracentesis was performed. Ascites fluid analysis revealed a serum-ascites albumin gradient <1.1, the total protein was 2.6 g/dL, amylase 19,168 U/L, and no evidence of infection or neoplastic cells. Pleural fluid was negative for malignancy as well.

Esophagogastroduodenoscopy was done the day after presentation which showed diffuse black mucosa from gastroesophageal junction to 25 centimeters from incisors (Figures 1 and 2). Biopsies revealed necrotic debris and acute inflammation.

The patient's respiratory status worsened progressively while in the hospital precluding additional evaluation for pancreatic ascites. There was no response to octreotide and he was unable to undergo repeat esophagogastroduodenoscopy. After approximately six weeks in the hospital, the patient died from worsening respiratory status and co-morbid illnesses.

Questions

1. What is the diagnosis?
2. What is the differential diagnosis of a black appearing esophagus on endoscopy?
3. Are there predisposing factors?
4. What is the treatment and prognosis?

(Answers and Discussion on page 66)

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DISCUSSION

Acute esophageal necrosis (AEN) without ingestion of a caustic or corrosive agent is uncommon. It has been defined as the presence of diffuse dark pigmentation of the esophagus on upper endoscopy associated with the histopathologic finding of mucosal necrosis (1). It is often referred to as "black esophagus." It is important to distinguish AEN from other entities that can give the appearance of a black esophagus including malignant melanoma, melanosis, pseudomelanosis, and acanthosis nigricans (2–4). It is also important to exclude caustic ingestion as well as local necrosis secondary to various infections.

The pathogenesis of AEN is unknown. There are a number of conditions that have been associated with AEN such as nasogastric tube trauma, antibiotics, malignancy , as well as ischemia. A variety of mechanisms have been proposed to explain AEN including low systemic blood pressure perfusion, direct toxic effect, and indirect mucosal breakdown (5). Lacy, et al reviewed twenty-three cases of AEN and failed to identify underlying condition common to all. They proposed that the precipitating event in many patients may be the acute development of gastric outlet obstruction (6). Although the precise incidence and prevalence of AEN is unknown Soussan (1) prospectively evaluated 3900 patients who underwent esophagogastroduodenoscopy over a one year interval and found eight (0.2%) who were diagnosed with AEN. Nutritional status was found to be poor in six of those patients.

There are no prospective studies on the treatment of AEN. Hydration and nutritional therapy, aggressive acid suppression and treatment of the underlying disease are probably the most beneficial. There is no data to support the use of antibiotics in patients with AEN.

The predominant complication of AEN is esophageal stenosis and stricture formation. Of the eight patients in the study by Soussan, four patients died precluding adequate follow-up endoscopy (1). Lacy, et al suggested that AEN has a poor prognosis with approximately one-third of patients dying from the underlying disease or directly related to AEN (6). ■

References

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