Acute esophageal necrosis in a patient with end-stage renal disease on hemodialysis

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ABSTRACT

Spontaneous acute esophageal necrosis (AEN) has been reported to be extremely rare. The condition is defined as a dark pigmentation of the esophagus associated with histologic mucosal necrosis. The exact pathogenesis is still unknown, but several etiologies have been suggested including ischemia, gastric outlet obstruction, hypersensitivity to antibiotics, gastric volvulus and viral infection. We herein present a case of a middle-aged man with end-stage renal disease who presented with AEN following a hemodialysis session. Its diagnosis and management are discussed with reference to the pathogenesis of the condition.

Key words: Barrett's esophagus, bleeding, endoscopy, gastroesophageal reflux, necrosis

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INTRODUCTION

Acute esophageal necrosis (AEN) in the absence of ingestion of a caustic or corrosive agent has been reported to be extremely rare. Two large retrospective series have estimated the occurrence at approximately 0.01% of all the etiologies.[1,2] AEN is defined as a dark pigmentation of the esophagus associated with histologic mucosal necrosis. The exact pathogenesis is still unknown, but several etiologies have been suggested including ischemia,[3,4] gastric outlet obstruction,[5] hypersensitivity to antibiotics,[6] gastric volvulus[7] and viral infection.[8]

We report herein a case of end-stage renal disease (ESRD) who presented with AEN after hemodialysis.

CASE REPORT

A 48-year-old male was diagnosed to have ESRD and was on renal replacement therapy for the last 2 years. There was a history of pyelolithotomy 15 years back and alcohol habituation till 1993. One night before presentation, he underwent hemodialysis during which he felt dizziness and had two episodes of forceful clear water vomiting. Four hours after dialysis, he felt nauseated and bloating, which was followed by bouts of fresh blood containing vomiting. He arrived in the emergency room and got admitted in the Department of Hepatogastroenterology.

At the time of arrival, he was well oriented with time, place and person. His supine blood pressure was 140/85 mmHg with no significant postural drop and pulse was 96 beats/min. His abdomen was soft and nontender with no visceromegaly appreciated. Gut sounds were audible. Chest and cardiac auscultation was normal.

At the time of presentation, his hemoglobin was 6.9 g/dL with mean corpuscular volume (MCV) of 87 fl and platelet count of 134,000 per cmm. He was transfused two units of packed cells.

The patient underwent upper gastrointestinal (GI) endoscopy on the same day of presentation, which showed extensive ulceration with bluish tinge mucosa in the esophagus covered with thick white exudates

and overlying slough with altered blood in the lumen. He was started empirically on omeprazole infusion, injection cefotaxime, injection fluconazole and syrup sucralfate.

The patient did not have a repeat episode of hematemesis and his blood indices remained stable. He underwent repeat endoscopy after 5 days, which showed a large ulcerated area in the mid-esophagus with overlying necrotic debris and two visible blood vessels. The Z-line was ragged with tongue of pinkish mucosa coming up for 2 cm. There was mild pangastric erythema. Biopsy taken from the ulcer margin showed features of AEN [Figure 1a and b]. Another biopsy from the pinkish tongue of the mucosa was consistent with Barrett's esophagus [Figure 1c and d]. Follow-up endoscopy performed after 1 month showed complete healing of the ulcer.

DISCUSSION

AEN is also referred to as "black esophagus" or necrotizing esophagitis. Goldenberg first described this entity.[9] It is characterized by the presence of diffuse dark pigmentation associated with esophageal mucosal necrosis in the absence of caustic or corrosive agent ingestion. Its etiology is likely multifactorial.[10,11] AEN is thought to arise from a combination of an ischemic insult to the esophagus, impaired mucosal barrier systems and a backflow injury from chemical contents of gastric secretions.[12-14]

The inciting event of AEN is unknown in most cases. Potential causes include ischemia, gastric outlet obstruction, trauma and infection.[15] The pathogenesis of AEN remains unclear, although an ischemic event is likely to play a role in precipitating the event. In support of this theory, reduction of esophageal blood perfusion experimentally can result in extensive esophageal mucosal injury that can resolve if perfusion is restored.[16,17] In addition, AEN tends to occur in the distal third of the esophagus, a relatively hypovascular "watershed region" relative to the proximal esophagus. Our patient had severe extensive necrosis that had involved the middle and distal esophagus, and the follow-up endoscopy showed persistent involvement. It is unlikely that one single factor is entirely responsible for this disease entity and the extent of damage.

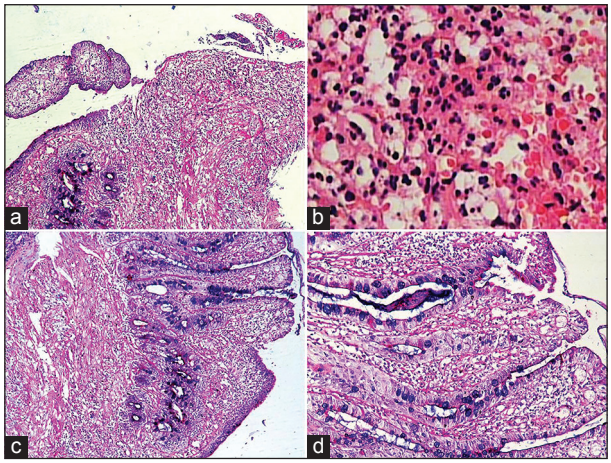

Figure 1: Histopathological features of esophageal biopsy at the time of presentation. (a) Low-power view showing partly intact glandular lining mucosa and partly ulcerated with overlying ulcer slough (Periodic acid-Schiff [PAS], ×50). (b) High-power view showing necro-inflammatory slough signifying ulcer slough. No intact esophageal tissue is seen (hematoxylin and eosin, ×400). (c) Low-power view showing extensive goblet cell metaplasia of the glandular mucosa of esophagus in Barrett's metaplasia (PAS, ×100). (d) Medium-power view showing extensive goblet cell metaplasia of the glandular mucosa of esophagus in Barrett's metaplasia. Acute inflammatory cells are seen in the lamina propria. The mucosa is intact here (PAS, ×200)

In our case, the patient became hypotensive during hemodialysis. There was evidence of gastro-esophageal reflux disease (GERD) as manifested by ragged Z line and short segment Barrett's esophagus. Reflux of gastric contents might have made the mucosa more vulnerable and had aggravated the mucosal injury in this case.

Patients with AEN can present with upper GI bleeding that may develop rapidly after an inciting event. Definitive diagnosis is made by direct mucosal visualization on upper GI endoscopy. In the initial stages, black discoloration of the esophageal mucosa with friable hemorrhagic areas can be observed. A distinct transition to normal-appearing mucosa occurs below the gastroesophageal junction. In the later stages of AEN, the mucosa is partially covered with thick whitish exudates and slough. We also observed these findings in the initial and subsequent endoscopies. To differentiate AEN from other conditions that lead to black discoloration, mucosal biopsies should be taken.

In conclusion, this 48-year-old patient with ESRD developed AEN after undergoing hemodialysis, probably secondary to hypoperfusion. The patient also showed evidence of GERD. The patient had smooth recovery. In patients with GERD and who are undergoing hemodialysis, care should be taken to prevent hypotension.

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