

## THE CLINICAL PICTURE

### SUDHA AKKINEPALLY, MD

University Medical Center at Brackenridge, and Department of Internal Medicine, University of Texas Medical Branch, Austin Programs, Austin, TX

### VIJAYARAMA POREDDY, MD

University Medical Center at Brackenridge, and Department of Internal Medicine, Division of Gastroenterology, University of Texas Medical Branch, Austin Programs, Austin, TX

### ALEJANDRO MORENO, MD, MPH

University Medical Center at Brackenridge, and Department of Internal Medicine, University of Texas Medical Branch, Austin Programs, Austin, TX

# The Clinical Picture

## Black esophagus



FIGURE 1

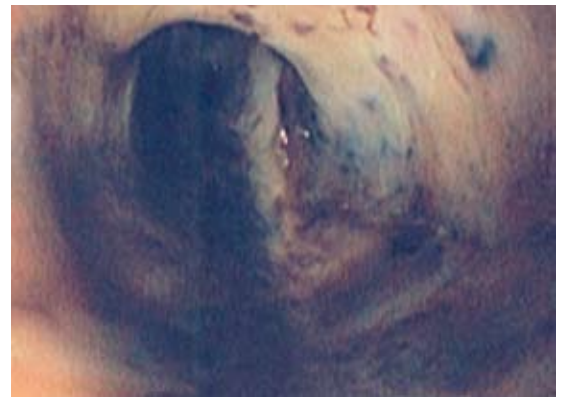


FIGURE 2

**A** 60-YEAR-OLD MAN with a history of alcoholic cirrhosis and gastrointestinal bleeding was admitted after being found unconscious. He had coffee-ground emesis and melena.

At the time of the physical examination, he was intubated and was unresponsive and hypotensive. On endoscopy, the upper half of the esophagus was normal (FIGURE 1), but the lower half showed signs of necrosis: the mucosa was black and covered by an exudate of the same color (FIGURE 2). Endoscopy also found signs of severe portal hypertensive gastropathy and multiple small ischemic ulcers in the distal duodenum. No biopsy was done because of the risk of bleeding due to coagulopathy. The patient died of hepatorenal syndrome and sepsis.

### ■ ASSOCIATED CONDITIONS

This patient had alcoholic cirrhosis and hypotension, which are significant comorbidities associated with necrotic (“black”) esophagus. Other conditions associated with black esophagus are gastric outlet obstruction, myocardial ischemia, hypersensitivity to antibi-

otics, certain viral infections, ketoacidosis, alcoholic hepatitis, acute renal failure, severe acid reflux, and acute pancreatitis.<sup>1,2</sup> The differential diagnosis includes melanosis, malignant melanoma, pseudomelanosis, acanthosis nigrans, adverse drug effects (quinidine and tetracycline), and infection (*Candida* and herpes).<sup>1,3</sup>

### ■ TREATMENT AND COURSE

The treatment is mainly supportive, with intravenous fluids, proton pump inhibitors, and nothing taken orally.<sup>1</sup> Complications include stricture, perforation, and death.<sup>1,3</sup> The death rate is about 30% in these patients.<sup>3</sup>

### ■ REFERENCES

1. Khan AM, Hundal R, Ramaswamy V, Korsten M, Dhuper S. Acute esophageal necrosis and liver pathology, a rare combination. *World J Gastroenterol* 2004; 10:2457–2458.
2. Le K, Ahmed A. Acute necrotizing esophagitis: case report and review of the literature. *J La State Med Soc* 2007; 159:330, 333–338.
3. Katsinelos P, Pilpilidis I, Dimiropoulos S, et al. Black esophagus induced by severe vomiting in a healthy young man. *Surg Endosc* 2003; 17:521.

ADDRESS: Sudha Akkinpally, MD, University Medical Center at Brackenridge, 601 East 15th Street, Annex Building, Internal Medicine, Austin, TX, 78701; e-mail suakkine@utmb.edu.