ACUTE NECROTIZING ESOPHAGITIS: A CASE REPORT

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Acute necrotizing esophagitis is rare. The exact etiology is unknown in most cases. The esophagus appears black, necrotic and ulcerated on the upper endoscopy, thus the term "black esophagus" is used. Histologically, there is necrosis of the esophageal mucosa and submucosa. Here, we present a patient with cholangiocarcinoma who had upper gastrointestinal bleeding and was found to have acute necrotizing esophagitis on the upper endoscopy. (*J Natl Med Assoc.* 2002;94:735-737.)

Key words: endoscopy ♦ esophagitis ♦ necrotizing

Acute necrotizing esophagitis on the upper endoscopy was first described by Goldberg et al.[1] Two cases of acute necrotizing esophagitis identified at post mortem had been reported prior to the description by Goldberg et al.[2,3] The dark pigmented, black appearance of the esophagus viewed during upper endoscopy is characteristic. Histologically, there is necrosis of the mucosa and submucosa. Inflammation and partial destruction of adjacent muscle fibers may be seen occasionally. Blood vessels are sometimes thrombosed or occluded.[4] The appearance of the black esophagus endoscopically could also be seen in melanosis,[5] malignant melanoma,[5] pseudomelanosis,[6] and acanthosis nigricans,[7] but without the same histologic features. Less severe forms of esophagitis are caused by gastroesophageal reflux, medication, infectious agents, corrosives and radiation injury.[8] Here, we present a report of a patient with acute necrotizing esophagitis.

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CASE REPORT

A 61-year-old African American male presented to our hospital at midnight with severe retrosternal and epigastric pain, which woke him up from sleep a few hours earlier. He had vomited coffee grounds thrice, but did not pass any melenic stools. He had no previous history of gastroesophageal reflux, peptic ulcer disease, ingestion of corrosives or NSAID medication. His past medical history was significant for hypertension, emphysema, hypercholesterolemia and chronic renal insufficiency. He had been recently diagnosed as having a liver mass for which a biopsy was to be performed.

Examination revealed an emaciated male with icteric sclerae, T: 97; P: 100; B.P: 197/124; R: 18. The abdomen was mild to moderately distended without any tenderness on palpation. The liver was enlarged and hard, with a span of 18cm, and shifting dullness was present.

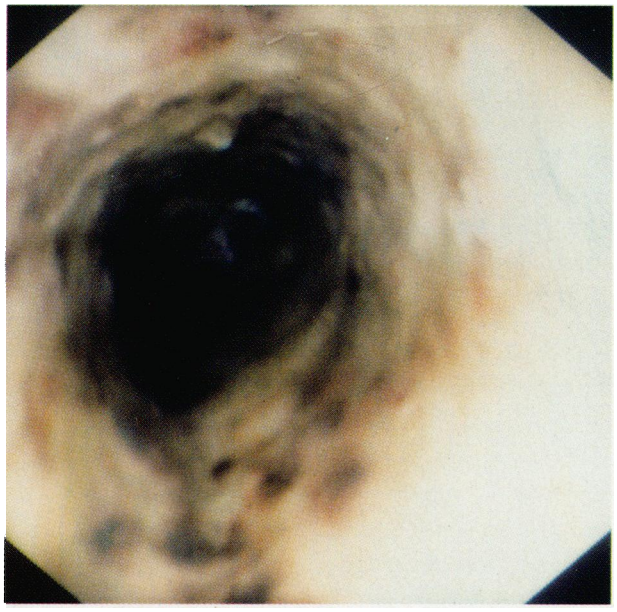

Fig. 1

Rectal examination revealed brown heme negative stool, an enlarged prostate gland and the absence of masses. Lab investigations included, WBC: 11; Hb:11; Hct: 35; platelets: 365; PT: 13.6; INR: 1.30; PTT: 33; Na: 141; K: 5.4; Cl: 103; CO_2: 24; bun: 42; Cr: 2.7; glucose: 91; Ca: 11; T.protein: 7.0; albumin: 2.0; T. bilirubin: 10.7; alk. phosphate: 583; AST: 83; ALT: 37.

The patient had an upper endoscopy the following day. The findings were that of circumferential black areas of necrosis, ulceration and exudate involving the entire lower three-fourths of the esophagus (see Figure 1). A few small erosions in the body of the stomach were noted, and the duodenum appeared congested, without any ulcer or erosion. The patient was commenced on sucralfate and omeprazole. A follow up endoscopy four days later showed improving ulceration and necrosis in the esophagus. Pathology findings were that of necrotic epithelium, tissue, and fibrino purulent exudate consistent with acute necrotizing esophagitis. Special stains for cytomegalovirus, herpes simplex virus and fungi were negative.

An ultrasound-guided liver biopsy revealed cholangiocarcinoma. The patient died four weeks following admission probably from massive pulmonary embolism. No autopsy was performed.

DISCUSSION

Acute necrotizing esophagitis is rare. Moreto et al.,[4] identified 10 cases in a review of more than 80,000 upper endoscopies, which corresponds to an incidence rate of 0.0125%. Lacy et al.,[9] reported two cases in more than 20,000 upper endoscopies during a period of two decades, an incidence of less than 0.01%.

On upper endoscopy, the esophagus appears black and necrotic. The etiology is unknown in most cases, but infections — nasogastric tube trauma and ischemia — have been suggested as possible causes.[9] The likelihood of transmural ischemia, however, is low because of the complex arterial blood supply of the esophagus.[10] Lacy et al.[9] reviewed 23 cases, and reported two additional cases of acute necrotizing esophagitis.

The etiology was apparent in four of the cases. An 81-year-old man had compression of the mid-to-distal esophagus by a posterior mediastinal hematoma following spontaneous rupture of the thoracic aorta. In another case, a 50-year-old man with anticardiolipin antibody syndrome had complete occlusion of the esophageal blood supply. Two patients developed acute esophageal necrosis in the setting of erythema multiforme and Stevens-Johnson syndrome. The etiology was unknown in the remaining 21 cases.

Lacy et al.[9] proposed that gastric outlet obstruction may be the precipitating factor for acute necrotizing esophagitis because two of the reported patients had gastric outlet obstruction, and 11 of the 23 cases reviewed had duodenal ulcers, severe duodenitis, or an abnormal pylorus on endoscopy.

Our patient did have a malignancy, which is a proposed mechanism of development of acute necrotizing esophagitis, but remains unproven. Five of the 23 patients reviewed by Lacy et al.,[9] had malignancies.

Kram et al.[11] documented a case of acute necrotizing esophagitis associated with gastric volvulus. Similarly, a case of acute necrotizing esophagitis associated with antibiotic use was documented by Mangan et al.[12] None of these was a factor in our patient.

Cattan et al.[13] documented a case of black esophagus associated with herpes esophagits. A follow-up endoscopy after six weeks showed complete resolution following a two-week course of acyclovir and six weeks of omeprazole. However, definite proof of a causal relationship between herpes esophagitis and acute necrotizing esophagitis is lacking. Herpes simplex virus was not identified in our patient.

The prognosis appears to be poor in acute necrotizing esophagitis. Our patient died four weeks later, it appears from a cause unrelated to necrotizing esophagitis. Massive pulmonary embolism was a probable cause of death, but no autopsy was performed to confirm this diagnosis. He did not have any further hematemesis during the course of admission, suggesting progression of the esophagitis, and a follow up endoscopy confirmed the improvement of the necrotizing esophagitis.

In the study by Lacy et al.,[9] 20 out of 25 patients with acute necrotizing esophagitis were followed. Sixty percent either died or suffered from residual side effects. Seven patients (35%) died from underlying disease, which appeared unrelated to acute necrotizing esophagitis. Five patients (25%) developed some complication related to acute necrotizing esophagitis: four developed esophageal strictures and one developed a mediastinal abscess. Eight patients (40%) remained asymptomatic, and did not appear to have any residual effects.

There are no prospective studies of the treatment of acute necrotizing esophagitis. Treatment should include management of the underlying illness and acid suppression with H_2 receptor blockers or proton pump inhibitors. Sucralfate could be used in addition to an H_2 receptor blocker or proton pump inhibitor.[9]

In conclusion, we have presented a report of a patient with acute necrotizing esophagitis. The etiology of the disease largely remains unknown in most cases, though several factors are proposed to cause it, such as ischemia and infections. Acute necrotizing esophagitis is a rare finding on an upper endoscopy.

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