Case Reports

Black esophagus: Acute esophageal necrosis due to alcohol intoxication

"Black esophagus" or acute esophageal necrosis or acute necrotising esophagitis is a rare clinical disorder identified by the characteristic finding of diffuse, circumferential, black, distal esophageal mucosa on endoscopy that stops abruptly at the gastroesophageal junction. The etiology is multifactorial. We report a case of acute esophageal necrosis secondary to acute alcohol intoxication in a young, male patient who presented with hematemesis.

Case Report

A 32 year old, male patient who was a chronic alcohol abuser came to the casualty in an inebriated state with a history of 3 bouts of hematemesis following an alcoholic binge. There was no history of corrosive ingestion and no other co-morbidities. Initial investigations revealed neutrophilic leucocytosis (TLC 22,000/ cu mm) and a hemoglobin of 8.8 gm%. Upper endoscopy revealed diffuse blackening involving the entire esophagus (**Figure 1**). However this discolouration seemed to abruptly end at the gastro-esophgeal junction and the gastric mucosa was normal (**Figure 2**).

The patient was kept nil per oral and was managed conservatively with intravenous fluids, proton pump inhibitors (PPI) and empirical antibiotics. The patient had an uneventful recovery and was discharged after he was able to tolerate oral feeds. Repeat endoscopy after one week revealed healing with some improvement in the blackish discolouration.

Discussion

First described in 1990, acute esophageal necrosis (AEN) is rare with a reported incidence of 0.008% to 0.2%.[1]

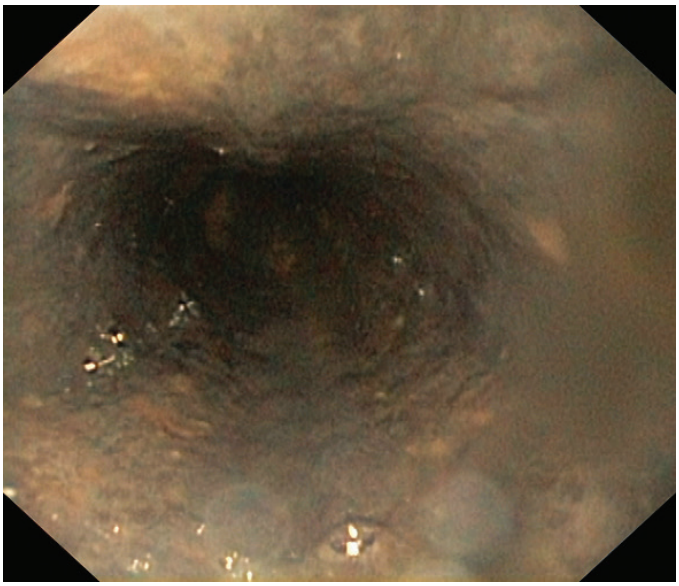

Figure 1: Diffuse blackish discoloration of esophagus.

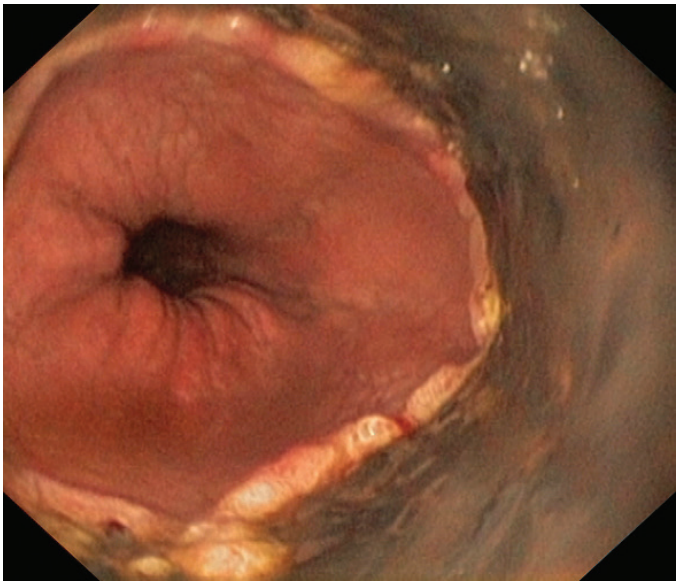

Figure 2: Clear demarcation of involved and uninvolved area at the gastro esophageal junction.

However, these numbers are likely underestimated and a study found AEN to be the fourth leading cause of upper G.I bleeding in 239 patients.[2] It is four times more common in men with a mean age of presentation at 67 years.[3] Risk factors for development of AEN include general debilitation and presence of multiple comorbidities like diabetes, hypertension, malignancy and alcohol abuse. The pathogenesis is multifactorial and combines tissue

hypoperfusion, impaired protective barriers and massive influx of gastric contents that acutely overwhelm the already vulnerable esophageal mucosa.[3] The distal esophagus may be preferentially involved because of its lesser vascularistion in comparison to the proximal and middle esophagus which makes it a "watershed area". Ulcers, erosions, inflammation and edema may occur in the duodenal bulb too because of the common blood supply to the duodenum and distal esophagus from the branches of celiac artery.[1,3,4] Relative sparing of the gastric mucosa is characteristic and can be explained by the susceptibility of the ischemic esophageal and duodenal mucosa to acid. Also, the injured gastric mucosa repairs itself within hours as compared to the esophageal mucosa which takes days.[1,3,4] Acute alcohol intoxication can lead to transient non-obstructive gastropathy which predisposes to AEN.[5]

Most patients present with upper gastrointestinal bleed although epigastric pain, vomiting, dysphagia, nausea, low-grade fever, light-headedness, and syncope can also occur. Laboratory analysis may show leukocytosis and anemia. The diagnosis of AEN is primarily based on the characteristic endoscopic finding of circumferential, black-appearing, diffusely necrotic esophageal mucosa, preferentially affecting the distal esophagus and stopping abruptly at the gastro-esophageal junction. The distal esophagus is practically always involved (97%) but proximal extension is also common. Duodenal ulcers, erosions, edema, and signs of gastric outlet obstructionmay also be seen.[3,4] The differential diagnosis of "black esophagus" includes malignant melanoma, acanthosis nigricans, coal dust deposition, pseudomelanosis, and melanocytosis of the esophagus.[3,4] It is also important to exclude corrosive ingestion.[4] Esophageal biopsy and brushings are supportive but not essential for diagnosis. Histological findings include absence of viable epithelium, abundant necrotic debris, and necrotic changes in the mucosa with variable extension including full thickness lesions. Bacterial, fungal and viral cultures may be required to exclude infectious etiologies.[1]

Management is mainly conservative with timely resuscitation, intravenous hydration, correction of anemia and intravenous PPI or H2 blockers. Ryle's tube insertion should be avoided for risk of perforation. The underlying medical conditions must be adequately treated. Surgical intervention is reserved for patients with complications like perforation or strictures not amenable to endoscopic dilatation. Prognosis depends on the co-existing medical conditions and the general condition of the patient with the overall mortality being about 32%.[3] However, AEN specific mortality is much lower (6%).[3]

VISHAL SHARMA
ARKA DE
SANDEEP LAMORIA
BRINDER MOHAN SINGH LAMBA

Department of Gastroenterology, PGIMER & Dr RML Hospital, Delhi, India.

Correspondence: Vishal Sharma
Email: docvishalsharma@gmail.com

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