Editorial



# A unique case of black esophagus and black stomach

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Acute esophageal necrosis (AEN) "black esophagus" is a rare clinical entity characterized by the circumferential, diffuse black appearance of the esophagus on endoscopic assessment. It commonly occurs in the distal esophagus and rarely extends beyond the gastroesophageal junction. The esophagus can be divided into three segments; cervical esophagus, intrathoracic esophagus, and abdominal esophagus. The cervical esophagus is supplied by branches of the inferior thyroidal artery, the intrathoracic esophagus is supplied by branches of the bronchial arteries, 3$^{rd}$ and 4$^{th}$ intercostal arteries, as well as smaller branches from the descending aorta, and the abdominal esophagus is supplied by the left gastric artery and left inferior phrenic artery with variable collateral circulation from splenic, celiac, short gastric or left hepatic artery. The abdominal esophagus passes through the right crus of the diaphragm at T10 and terminates at the level of the gastric cardia at T11 (1). The abdominal and terminal part of the thoracic segment of the esophagus are relatively more prone to ischemia compared to the more proximal parts of the esophagus and is hence considered a "watershed" area. As a result, ischemic insults are likely to manifest in the distal esophagus initially. Ischemic injury results in predisposition of the esophageal mucosa to topical injury which can be due to; acid reflux, gastric outlet obstruction, gastroparesis, or hiatal hernia. In addition, the esophagus lacks a serosa which leads to an increased risk of perforation and bacterial translocation with resultant infection (2). Moreover, several factors have been reported in case reports of AEN such as; diabetic ketoacidosis, use of broad-spectrum antibiotics, sepsis, severe vomiting, alcoholic hepatitis, and aortic dissection (3-7).

AEN has a prevalence of 0.01% to 0.28% which was observed in endoscopic studies (8,9). In a study conducted by Postlethwait *et al.*, examination of 1,000 autopsy specimens revealed that 17 specimens revealed black esophagus findings with the degree of staining correlating with anatomical location. The degree of staining was significantly higher in the distal esophagus (10). In a retrospective analysis of 10,295 endoscopies over a 5-year period, 0.28% of endoscopies revealed findings of AEN with a mortality of approximately 35% (9). A prospective study by Ben Soussan *et al.* assessed the frequency of AEN over a 1-year period. The prevalence of AEN was 0.2% with a mortality rate of 50% (11). It is important to note that the mortality rates in the aforementioned studies is not directly linked to the esophageal disease and could be a result of the comorbidities and overall clinical condition which predisposed these patients to AEN.

The most common presentation (approximately 70%) of AEN is symptoms of upper gastrointestinal bleed such as; hematemesis, coffee ground emesis, and melena. Chest pain, dysphagia, and epigastric pain have been reported (12).

AEN can be staged from stage 0 to 4 based on endoscopic and histological findings. Stage 0 has normal endoscopic appearance of the esophagus (pre-necrotic) with no abnormalities on histology. Stage 1 is characterized by friable, black circumferential discoloration, with potential yellow exudates which terminates abruptly in at the gastroesophageal junction. Histologically, epithelial disruption, necrosis of the mucosal and submucosal layer, as well as abundance of inflammatory cells. Stage 1 is observed

the earliest in the disease process. Stage 2 is characterized by the predominance of thick white exudates that can be scraped revealing underlying friable pink mucosa. Residual black spots can be seen. Histologically, necrotic debris can be seen along with granulation tissue with surrounding inflammation. This stage is seen 1 to 4 weeks following diagnosis of AEN and can be complicated by stricture formation. Stage 3 refers to normalization of esophageal appearance on endoscopic examination, with pathology revealing granulation tissue and normal mucosa (13). The management of AEN is primarily supportive with keeping the patient nils per oral (NPO) (potential use of total parenteral nutrition), intravenous hydration, proton pump inhibitors, and sucralfate after at least 24 hours of being NPO. Addressing the underlying pathology is crucial for improvement of AEN. The use of broad-spectrum antibiotics should be reserved for patients with evidence of perforation, mediastinitis, or signs of systemic infection. The insertion of nasogastric tube should be avoided due to possible iatrogenic perforation. Esophagectomy can be considered in patients who are surgical candidates and have evidence of perforation. The use of covered metallic esophageal stents has been performed in non-surgical candidates with success (14).

Magarinos *et al.* describe a patient with acute necrosis extending to the gastric cardia and leading to perforation (15). The involvement of the stomach in patients with AEN has not been reported thus far in the literature. The patient in the authors' case had a history of type A aortic dissection (with surgical repair), heart failure with reduced ejection fraction and suffered a cardiac arrest requiring cardiopulmonary resuscitation. The duration of the cardiac arrest is unknown. The aforementioned factors are all risks for esophageal and gastric ischemia. Of note, the patient in this case also had a history of gastric outlet obstruction which increases the risk of topical esophageal and gastric injury leading to necrosis and perforation. Gastric ischemia is extremely rare due to the gastric vascular supply. The stomach receives its arterial blood supply from the right and left gastroepiploic arteries, right and left gastric arteries, and the short gastric arteries (16). In fact, in animal models, ligation of 95% of the gastric arterial supply did not result in significant gastric mucosal injury (17). The authors stated that the patient underwent computed tomography with angiography of the abdomen which showed stable type A aortic dissection without involvement of the celiac or the superior mesenteric artery. They identified potential etiologies of gastric ischemia in this case such as chronic ischemia secondary to aortic dissection with potential acute ischemic injury during cardiac arrest. The patient had underlying sepsis and lactic acidosis, both of which have been reported to be associated with AEN.

AEN could be underreported as patients are in a critical state and significant diagnostic and therapeutic measures are often foregone with the primary goal being the comfort of the patient. Despite AEN being a result of an underlying primary pathology, early recognition and management of this condition can lead to complete or partial resolution of AEN. The mortality rates are derived primarily from small studies and case series and hence the results are heterogenous. The higher mortality rates observed in some studies can be secondary to the underlying pathologies that patients with AEN possess rather than due to AEN specifically. Diagnosis is made by endoscopic assessment with or without obtaining biopsies.

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