Case Report



# A rare case of combined black esophagus and stomach: a case report

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**Background:** Black esophagus is a relatively rare phenomenon that seems to occur in a morbid patient population, however, this entity may be more common than we currently know. Additionally, black esophagus may even be reversible in some cases. There are far fewer cases of black stomach described. The exact etiology and cause are less well known but may have similar risk factors as black esophagus. These disease pathologies are devastating to the patients they affect and can pose clinical challenges for the providers that encounter them.

**Case Description:** This case report details our experience with a moribund patient who was found to have extensive pneumomediastinum and gastric perforation secondary to black esophagus and black stomach. This was diagnosed on endoscopy following imaging that showed extensive pneumomediastinum. Initially the intent was to temporize the disease process in an attempt to stabilize the patient however at the time of diagnosis, the degree of ischemia had led to perforation and the disease process was not reversible or recoverable. He ultimately expired after comfort directed care measures were put in place.

**Conclusions:** To the best of our knowledge, this case will be the first reported in current literature and will hopefully shed some light on this unusual and devastating pathology.

**Keywords:** Case report; esophagus; stomach

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## Introduction

Black esophagus, also known as acute esophageal necrosis (AEN) is an uncommon pathology, with a predicted incidence of 0.01–0.28% (1). It seems to present most commonly with signs of upper gastrointestinal bleeding, particularly hematemesis (2,3). It has been theorized to result from a combination of hemodynamic compromise, and baseline critical illness (1). There have been several studies highlighting an association with alcohol abuse as well (4,5). Surprisingly, the mortality seems to be relatively low, reported around 32%, with several reports describing resolution of the condition in several patients (5,6). There are extremely limited reports of black stomach, and to our knowledge, we are presenting the first case of combined black esophagus and stomach. We present the following case in accordance with the CARE reporting checklist (available at https://aoe.amegroups.com/article/view/10.21037/aoe-21-70/rc).

## Case presentation

Our case involves a 75-year-old man with relevant past medical history of reduced ejection fraction heart failure, chronic obstructive pulmonary disease, type A aortic dissection as well as metastatic pancreatic cancer was brought to our hospital following an outpatient cardiac arrest. He had initially contacted emergency medical services for worsening abdominal pain, during their evaluation of the patient he suddenly became unresponsive and was found to be pulseless. He underwent cardiopulmonary resuscitation and had eventual return of system circulation. He was subsequently admitted to the

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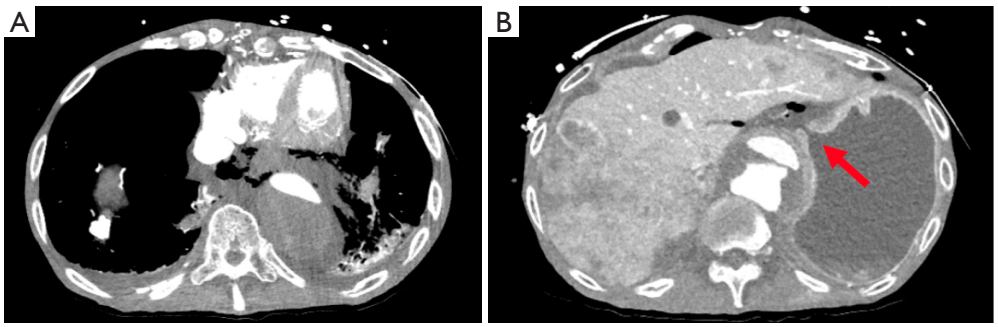


**Figure 1** Cross sectional imaging demonstrating pneumomediastinum and perforation. (A) Axial section of CTA demonstrating pneumomediastinum; (B) axial section of CTA with perforation just below GEJ (red arrow). CTA, computed tomography angiography; GEJ, gastroesophageal junction.

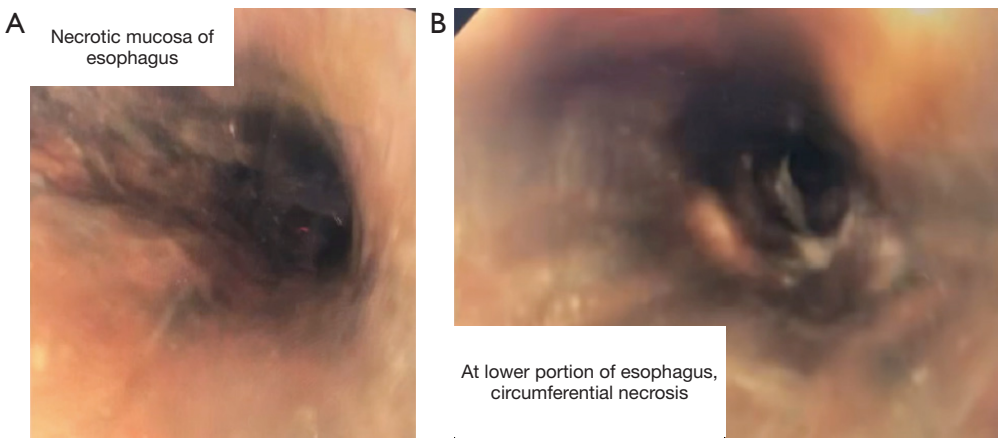


**Figure 2** Images obtained during endoscopy showing necrosis of mucosa. (A) Esophagus at 25 cm from incisors, patchy black mucosa; (B) esophagus at 35 cm from incisors, circumferential black mucosa.

intensive care unit (ICU) for further resuscitation.

Of note several months prior to presentation he was evaluated for possible gastric outlet obstruction by the gastroenterology team. At the time he had presented with gastric distention and thickening at gastrojejunal junction (patient had previously undergone a pancreaticoduodenectomy) on imaging. He was initially decompressed with a nasogastric tube but subsequently had minimal output and was able to tolerate liquid diet. He was being followed by the gastroenterology team for possible stent placement prior to his decompensation.

Once further stabilized from the code event he underwent computed tomography angiography (CTA) scan which demonstrated extensive pneumomediastinum, pneumoperitoneum and free fluid (*Figure 1A*) as well as an apparent perforation at the cardia below the gastroesophageal junction (GEJ) (*Figure 1B*). At this point our team was consulted for further evaluation. He was noted to be hemodynamically unstable with a pressor requirement. His physical exam was significant for signs of peritonitis. His laboratory results were significant for leukopenia of 3,700, and lactic acidosis of 2.9 mmol/L. Given his hemodynamic instability and medical history the decision was made to proceed with endoscopy to better elucidate the cause of these findings and for possible stent placement. The stent was considered as a temporizing measure given that the patient was not hemodynamically stable enough to tolerate more definitive surgery.

During the endoscopy, at 25 cm from the incisors, patchy black mucosa was encountered (*Figure 2A*), extending to confluent areas at 30 cm and circumferential necrosis at 40 cm from incisors (*Figure 2B*). The gastric mucosa of

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**Figure 3** Back mucosa of body of stomach.

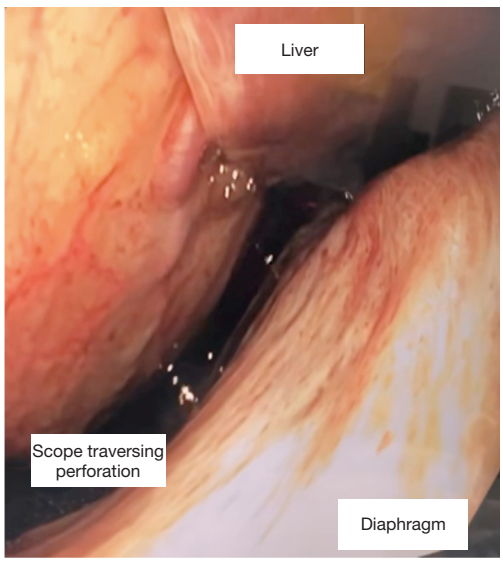


**Figure 4** Endoscope traversing perforation, edge of liver and diaphragm visualized.

the fundus, body and antrum were necrotic (*Figure 3*) with the perforation in the fundus with peritoneal viscera and liver visible through the defect (*Figure 4*). Given the extent of the necrosis, no biopsies were taken out of concern for worsening the perforation. Further interventions were deemed futile and the patient was brought to the ICU for comfort directed care and expired shortly thereafter.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

## Discussion

The stomach has a robust blood supply of multiple sources, consisting of the left and right gastric and gastroepiploic arteries, making it incredibly resistant to ischemia. The patient we described had a prior history of type A aortic dissection and repair, and chronic dissection flap extended into the abdominal aorta and iliac arteries. The dissection was stable and appeared unchanged from prior CT, with the celiac and superior mesenteric artery (SMA) arteries patent on imaging. It is possible the visceral circulation was compromised with lower perfusion pressure as a consequence of chronic dissection. This could expose the visceral circulation to hemodynamic insults, especially during the cardiac arrest. It is unclear if the cardiac arrest was secondary to worsening sepsis from primary gastroesophageal ischemia or if primary cardiac arrest resulted in visceral hypoperfusion. However, in either scenario, severe visceral ischemia led to extensive gastroesophageal necrosis.

The reported outcomes of black esophagus are varied in the literature. Some cases described resolution of the necrosis following further resuscitation, with patient not requiring any further surgical intervention (7-9). The management of these so-called “stable” patients is typically supportive, and involves correction of the underlying pathologies, usually involving resuscitative efforts, antibiotics, acid-suppression, and bowel-rest with parenteral nutrition (10). However, the patients in these reports did not have pan-esophageal necrosis, and in most cases only the distal esophagus was involved. A report by Riascos and colleagues demonstrated a case of near pan-esophageal necrosis with perforation (11). The patient underwent a left cervicotomy and right thoracotomy with wide mediastinal drainage for source control initially, followed by return to operating room (OR) for esophagectomy and feeding gastrostomy several days after improved hemodynamics. The patient improved clinically initially, however eventually decompensated and succumbed, likely due to pulmonary embolism. The mortality rate of AEN has been quoted to be as high as 32%, however, this number is likely related to other concurrent illnesses. The mortality specific to AEN

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is approximately 6% (10). The incidence of perforation secondary to black esophagus is surprisingly only 5% (12), however this is likely an underreported phenomena as patients may be too unstable and expire prior to presentation or procedures that allow for diagnosis.

The most common long-term sequelae of those who survive the acute phase of black esophagus is esophageal stricture, which accounts for 70% of all AEN long-term complications in survivors (13,14).

Black stomach is an even more rare phenomenon, with only a handful of cases described. Two cases of black stomach were reported, secondary to ingestion of caustic substances (15,16), and one case reported in the setting of polycythemia vera (17). The management was supportive until the patients succumbed to their disease. This likely represents a very late stage in their illness and gravity of the disease in this ischemia resistant organ.

There is only one other recently published report of combined necrosis of multiple foregut organs (18). The case presented a combined black esophagus and duodenum with the stomach being spared. This case, to the best of our knowledge, is the first reported case of combined black esophagus and stomach with unfortunately fatal but expected outcome. It is also important to note that given the patients long standing underlying malignancy his death was likely the result of a multitude of factors. Unfortunately, there were no viable operative interventions to correct the problem and all efforts would have been futile. More attention is needed to address early diagnosis of the condition and a meaningful impact on the outcomes. It is important to recognize that both these pathologies are secondary to a primary pathology driving the ischemia. In order to adequately treat patients with this pathology providers should also aim to identify and treat the primary underlying pathology. Finally recognizing the extent of the disease and that mortality associated with this complex may be underreported due to self-selecting nature of patients may help providers offer appropriate treatment options such as comfort care measures.

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## Footnote

*Reporting Checklist:* The authors have completed the CARE reporting checklist. Available at https://aoe.amegroups.com/article/view/10.21037/aoe-21-70/rc

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*Conflicts of Interest:* All authors have completed the ICMJE uniform disclosure form (available at https://aoe.amegroups.com/article/view/10.21037/aoe-21-70/coif). The authors have no conflicts of interest to declare.

*Ethical Statement:* The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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