

# Acute esophageal necrosis: Black esophagus in setting of diabetic ketoacidosis

Brooke Colin Bear, Jacob Mathew, Calvin W. Parker III

## ABSTRACT

**Introduction:** Acute esophageal necrosis (AEN) is a rare cause of upper gastrointestinal bleeding with an unclear etiology that may be related to ischemic insults resulting in caustic injury primarily to the distal segment of the esophagus along with damage caused by underlying coronary artery disease or diabetes mellitus. If not diagnosed correctly, treatment may incorrectly focus on proton pump rather than underlying medical conditions which if not addressed, can lead to repeat episodes of the condition. **Case Report:** We report a case in which a patient presented in diabetic crises. **Esophagogastroduodenoscopy (EGD)** revealed circumferential necrotic discoloration and friable tissue of the gastroesophageal junction with a normal appearing stomach. Treatment with a proton pump inhibitor and aggressive insulin management led to cessation of symptoms. **Conclusion:** Upper gastrointestinal bleeding is a common complaint in both the inpatient and outpatient settings. While many cases may be related to bacterial infection (*Helicobacter pylori*) or iatrogenic insults (NSAID or alcohol use), rare cases may be related to poorly controlled underlying medical conditions such

as coronary artery disease or diabetes. Our case highlights the need to keep heightened suspicion for entities such as AEN in patients who have appropriate underlying medical conditions to prevent further episodes.

**Keywords:** Diabetic ketoacidosis, Endoscopy, Esophageal necrosis, Gastrointestinal bleed,

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## INTRODUCTION

Acute esophageal necrosis is a rare entity with an estimated prevalence from 0.001 to 0.2% of cases affecting men four times more than women [1, 2]. The etiology of disease is unclear but is most likely felt to be ischemic in nature that predisposes tissue to caustic injury [3]. We present a case of a 52-year-old male who presented in diabetic ketoacidosis (DKA) who was found to have melanotic stools with upper esophagogastroduodenoscopy (EGD) revealing acute esophageal necrosis. Acute esophageal necrosis is an important disorder to consider in the evaluation of patients who present with diabetic ketoacidosis that have evidence suggestive of upper gastrointestinal bleed (UGIB).

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## CASE REPORT

A 60-year-old male with poorly controlled diabetes complicated by multiple amputations presented to the emergency room with complaints of generalized weakness, dry mouth, emesis and decreased intake by mouth and elbow pain. On presentation, he was noted to have a relatively low blood pressure of 95/76 mmHg and elevated serum glucose of 632 mg/dL (baseline HgA1c of 8.2% revealing historically poor diabetes control). Chemistry panel was notable for potassium of 3.8 mEq/L, bicarbonate of 19 mEq/L (with calculated anion gap of 18) and serum creatinine of 2.3 mg/dL (which was 1.2 units higher than his historic values). Laboratory examination revealed hemoglobin and hematocrit of 15 g/dL and 47 g/dL respectively, near his baseline. An elevated serum osmolarity of 367 mOsm/kg and serum ketones. He was admitted to the ICU for the management of presumed hyperosmolar hyperglycemic state (HHS) vs DKA with acute kidney failure. He was fluid resuscitated and his glucose corrected with an insulin drip per unit protocol. These interventions led to an improvement in his acid-base disturbance, closure of his anion gap, and improvement in his renal function. Twenty-four hours into his admission, he was found to have melanotic stools. His vitals had improved with fluid resuscitation and while he had yet to be transitioned to oral intake, he denied any abdominal pain. Medical chart review showed no history of *Helicobacter pylori* infection in the past. The following day an EGD was performed revealing a normal appearing upper esophagus (Figure 1) but upon inspection of the distal segment, a circumferential black necrotic discoloration with underlying friable tissue of the gastroesophageal junction with a sharp transition point towards normal appearing mucosa was noted (Figures 2–3). No abnormalities were noted in either the stomach or the duodenum.

The patient was placed on a Protonix drip (proton pump inhibitor) for 72 hours, his mean arterial pressures goal of 65 mmHg was maintained with plasmalyte and continued cessation of his home antihypertensive medications which included Lisinopril 25 mg daily and hydrochlorothiazide 12.5 mg daily. After 72 hours of the proton pump inhibitor drip, he was placed on oral therapy with esomeprazole 40 mg twice a day dosing to complete 30 days of therapy and his diet was advanced. He was discharged on insulin therapy after appropriate in-hospital teaching took place. In a one-month follow-up with the patient to his primary care manager, he had no similar episodes of UGIB warranting hospital admission and remained stable with daily proton pump therapy and insulin administration. Medical treatment continues to focus on improvement of his diabetes.

## DISCUSSION

Acute esophageal necrosis (also known as black esophagus) is a rare disorder that is seen more often in

men than women with an incidence rate ranging from 0.001–0.2% [1, 2]. The pathologic etiology of acute esophageal necrosis has yet to be determined but a “two hit hypothesis” involving a low flow vascular state is thought to predispose esophageal mucosa to severe ischemic injury causing the necrosis observed on endoscopy [3]. The lower two-thirds of the esophagus is anatomically at higher risk for ischemia given its relatively hypovascular state when compared with other esophageal segments [3].

A history of diabetic ketoacidosis, coronary artery disease, prolonged vomiting as well as other conditions that predispose one to diminished blood flow are thought to predispose patients for this disorder was seen with our patient. He was noted to have low blood pressures with elevated blood sugars and prolonged vomiting prior to emergency room visit with subsequent melanotic stool that improved with management of hemodynamics after initiation of fluids and treatment of his diabetic ketoacidosis, treatment of acute kidney injury, and continuous PPI drip.

The diagnosis of esophageal necrosis can be made off of characteristic findings on EGD such as blackened



Figure 1: Endoscopic view of the upper third of esophagus with healthy appearing mucosa.

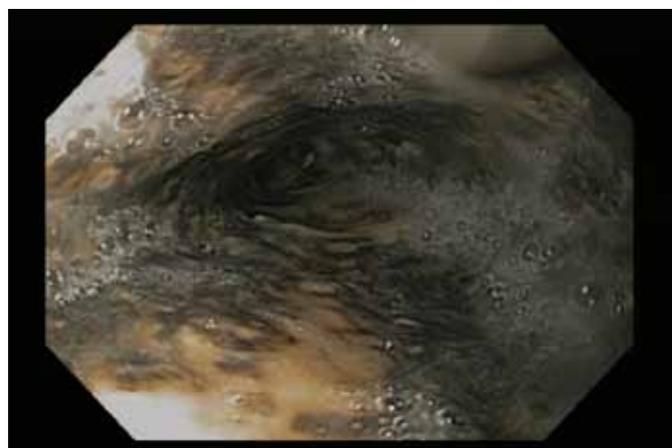


Figure 2: Endoscopic view of the middle esophagus revealing black mucosa with friable tissue.



Figure 3: Endoscopic view of the lower third of the esophagus with continued black mucosa and sparing of the gastroesophageal junction.

esophagus and clinical history to include diagnoses of diabetes mellitus, diabetic ketoacidosis, coronary artery disease, and emesis with hypotension. Histopathology can be obtained to exclude other causes, however, risk of perforation can be as high as 7% [4, 5]. Once a diagnosis is made, management is based on maintaining hemodynamic stability with optimizing hemoglobin, gastric acid suppression with PPI, and management of underlying medical conditions [6].

## CONCLUSION

Acute esophageal necrosis is a rare disorder whose exact etiology has yet to be determined but may be related to ischemic injury affecting primarily the lower two thirds of the esophagus. A strong consideration for acute esophageal necrosis should be considered in a patient with long standing diabetes, coronary artery disease who presents with hypotension, in DKA with melanotic stools. Diagnosis is made through visual inspection via EGD; histopathology is not required and in-fact may increase risk for further esophageal damage or perforation. The management is focused on PPI therapy with restoration of perfusion pressure with fluids until the underlying conditions resolve.

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## Author Contributions

Brooke Colin Bear – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Final approval of the version to be published  
Jacob Mathew – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Critical revision of the article, Final approval of the version to be published

Calvin W. Parker III – Analysis and interpretation of data, Critical revision of the article, Final approval of the version to be published

## Guarantor

The corresponding author is the guarantor of submission.

## Conflict of Interest

The views expressed in this abstract/manuscript are those of the author(s) and do not reflect the official policy or position of the Department of the Army, Department of Defense, or the US Government. I have obtained permission to reprint any figures in this manuscript. All identifying patient information has been removed to protect the patient's privacy. Neither myself nor the other contributing authors have any financial support to disclose, and have no other conflicts of interest.

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