CLINICAL VIGNETTE

Acute Esophageal Necrosis

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Case Presentation

Patient is a 62-year-old male presented with one day history of bloody vomiting. His past medical history includes atrial fibrillation (on rivaroxaban), HTN and lifestyle controlled- diabetes. He was in his usual state of health but awakens in the middle of the night with nausea and several episodes of vomiting which were initially yellow/green but became bloody. He denied any associated abdominal pain or melena reported burning heartburn in his chest. His last dose of rivaroxaban was the evening prior to admission. He denies any prior gastrointestinal problems and no chronic liver disease. He drinks alcohol on a daily basis and does not use non-steroidal anti-inflammatory drugs. There was no prior endoscopy.

Examination was notable for sinus tachycardia and slightly elevated blood pressure, with normal remaining vital signs. He was anxious and appeared acutely ill. The remainder of his physical examination including abdominal exam was unrevealing. Labs were significant for WBC of 21.1 (4.0 - 11.0 x10 3 /uL) with 86% neutrophils, hemoglobin 17.7 (13.5 - 17.7 g/dL) and lactate of 10.7 (0.4 - 2.0 mmol/L). Given his leukocytosis with lactic acidosis, stat CT angiogram of the abdomen and pelvis was obtained and negative for any acute findings, mesenteric ischemia or vascular compromise. It did note distal esophageal thickening.

The patient was admitted to ICU and started on aggressive fluid resuscitation and empiric IV antibiotics. Emergent upper endoscopy showed blackened esophageal mucosa consistent with acute esophageal necrosis (AEN) of the distal esophagus with sparing of the gastroesophageal junction (Figure 1). There was no other cause for upper GI bleeding found. The patient was treated with IV acid blocking medications, advancement of diet as tolerated and discharged home after a few days. After a few weeks of oral PPI therapy and alcohol abstinence, follow-up endoscopy showed entirely normal esophageal mucosa.

Discussion

AEN, also referred to as “black esophagus,” is a rare cause of upper GI bleeding with a reported prevalence of 0.001% to 0.2%. The pathogenesis is thought to be secondary to mucosal ischemia and necrosis from a combination of low vascular supply, and topical caustic injury including binge drinking and vomiting. It occurs in association with conditions that cause severe tissue hypoperfusion or a thrombo-embolic state such as severe shock, acute blood loss, lactic or keto- acidosis, sepsis, pancreatitis, arrythmias, and solid or hematological malignancies. Endoscopy is the gold standard for diagnosis and typically demonstrates a blackened mucosal lining of the distal esophagus sparing the gastroesophageal junction. This is due to the “watershed” blood supply of the distal esophagus (relative to the more proximal esophagus). Management of AEN consists of volume resuscitation with intravenous fluids, gastric acid suppression with proton pump inhibitors and treatment of the inciting cause. Follow up endoscopy after several weeks is recommended to document esophageal mucosal healing. The differential diagnosis includes malignant melanoma, acanthosis nigricans, melanosis or coal dust deposition. Imaging is necessary to rule out esophageal perforation with mediastinitis. Other complications include interval esophageal stricture or stenosis.

Figure 1