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CASE REPORT

Acute Esophageal Necrosis (Gurvits Syndrome): A Rare Cause of Upper Gastrointestinal Bleeding in Somalia

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Abstract: Acute esophageal necrosis, also known as Gurvits syndrome, is a rare condition due to ischemic compromise and thromboembolic injury to esophagus associated with high mortality. Endoscopically, it is characterized by the circumferential black discoloration of the esophagus. We present the case of a 55-year-old male with a history of multiple comorbidities, including uncontrolled type 2 diabetes mellitus, hypertension, smoker, peripheral arterial disease, and a right above-ankle amputation, who presented with active hematemesis and melena with hemodynamic instability. An esophagogastroduodenoscopy revealed diffuse, circumferential necrotizing esophagitis with black discoloration and ulcerations affecting the middle and distal thirds of the esophagus. The severity increased from the proximal to the distal esophagus, abruptly ending at the gastroesophageal junction. These endoscopic findings, combined with the patient's medical history, were consistent with a diagnosis of acute esophageal necrosis. The patient was admitted to the ward and managed conservatively with intravenous fluid resuscitation, IV proton pump inhibitor twice daily, sucralfate 1 gram every six hours, strict glycemic control using insulin, total parenteral nutrition, empirical IV antibiotics, and placed on nil-per-oral (NPO) for three days. A follow-up esophagogastroduodenoscopy conducted twenty-two days post-admission showed complete healing of the esophageal mucosa without stricture formation. Despite its rarity, prompt diagnosis and management of acute esophageal necrosis are crucial due to its association with high morbidity and mortality, as well as the need to minimize complications such as perforations and strictures, particularly in patients with comorbidities. This case report aims to raise awareness among clinicians in Somalia of this condition as a differential diagnosis in upper gastrointestinal bleeding and to highlight the importance of timely intervention to prevent adverse outcomes.

Keywords: hematemesis, acute esophageal necrosis, Gurvits syndrome, black esophagus, upper GI bleeding

Introduction

Black esophagus, also commonly referred to as Gurvits syndrome or acute esophageal necrosis (AEN), is a rare condition causing upper gastrointestinal bleeding. Goldenberg et al first described in the medical literature in 1990[1] and it was brought to the forefront by Gurvits et al in 2007.[2] It is an extremely rare cause of upper gastrointestinal bleeding with total reported number of cases less than 150.[3,4] The etiopathogenesis of AEN remains poorly understood, but ischemic compromise and thromboembolic injury have been considered as important risk factors.[5] The Common risk factors for the development of AEN include male sex, coronary artery disease (CAD), renal insufficiency, diabetes, hypertension, malignancy, alcohol use disorder, gastric outlet obstruction, sepsis, and malnutrition.[2,6] Classic presentation on esophagogastroduodenoscopy (EGD) includes circumferential black discoloration of the esophageal mucosa, most often distally, that abruptly ends at the gastroesophageal junction (GEJ). "Biopsy of the esophagus demonstrates necrosis of the mucosa with possible extension into the submucosa of the esophagus".[7] Clinically, it often presents with upper gastrointestinal

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bleeding in the form of hematemesis or melena.[8] Serious acute complications of AEN include perforation, mediastinitis, abscess formation and potential sepsis, and long-term complication is stenosis formation.[9] Here, we present a case of black esophagus associated with multiple comorbidities presenting with hematemesis and melena.

Case Presentation

A 55-year-old male with a history of multiple comorbidities including uncontrolled type 2 diabetes mellitus, hypertension, smoker, peripheral arterial disease, and a right above-ankle amputation secondary to peripheral vascular disease related to his diabetes. The patient presented to our emergency department (ED) with a 2-day history of two attacks of active hematemesis 300–400 mL in volume, it was associated with postprandial abdominal pain, melena, polyuria, polyphagia, and generalized fatigue. The patient denied odynophagia, hematochezia, syncope, and palpitations. He had not been taking his insulin regularly, and he has no history of chronic liver disease. He denied use of non-steroidal anti-inflammatory drugs (NSAIDs), tobacco, alcohol, or recreational drugs. On physical examination, he was relatively unstable hemodynamically on arrival with a pulse rate of 110 beats per minute, blood pressure of 141/77 mmHg, respiratory rate of 18 breaths/minute, temperature of 36.6°C and oxygen saturation of 98% on room air. Abdominal, chest and heart examinations revealed no remarkable findings. Laboratory work-up revealed a hemoglobin of 15.4 g/dl, a hematocrit of 46%, a white blood cell count of 6.43×10^9/l, a platelet count of 304×10^9/l, and an international normalized ratio (INR) of 1.6. Blood chemistry testing revealed elevated serum glucose of 316 mg/dl, HBA1C of 11.2%, blood urea of 41 mg/dl, creatinine of 0.8mg/dl, potassium of 3.9 mmol/l, sodium of 131 mmol/l, serum amylase of 19U/L. Arterial blood gas analysis showed a pH of 7.34, bicarbonate of 24 mmol/L, and anion gap of 12 mmol/L. Serology screening for HBSAg, Anti-HCV, and HIV I & II were negative. Abdominal U/S examination was unremarkable.

The patient was resuscitated with intravenous (IV) fluid, and control of blood glucose with insulin was initiated. After his clinical condition improved, a gastroenterologist was consulted for the endoscopic evaluation of his hematemesis, and melena.

Esophagogastroduodenoscopy (EGD) (Figure 1) was performed and showed diffuse, circumferential patchy necrotizing esophagitis with black discoloration and ulcerations affecting the middle and distal thirds of the esophagus, beginning 25 cm from the incisors, consistent with a diagnosis of acute esophageal necrosis. The severity increased from the proximal to the distal esophagus, abruptly ending at the gastroesophageal junction.

Biopsies were not taken due to the risk of perforation. He was initiated on intravenous fluid resuscitation, IV proton pump inhibitor twice daily, sucralfate 1 gram every six hours, strict glycemic control using insulin, total parenteral nutrition, empirical IV ceftriaxone, and placed on nil-per-oral for two days. Thereafter, Soft diet was started as patient tolerated.

As his general condition improved over the following days, he was discharged home on oral PPI and sucralfate, with follow-up with his gastroenterologist for repeat EGD after three weeks to reassess for mucosal healing. A follow-up esophagogastroduodenoscopy (EGD) (Figure 2) conducted twenty-two days post-admission showed complete healing of the esophageal mucosa without stricture formation.

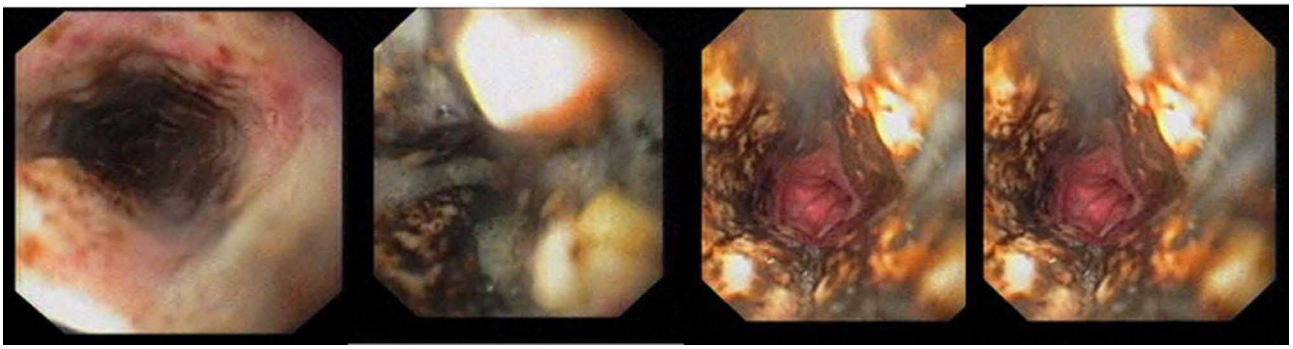

Figure 1 Esophagogastroduodenoscopy (EGD). EGD images showing diffuse, circumferential patchy necrotizing esophagitis with black discoloration and ulcerations affecting the middle and distal thirds of the esophagus, beginning 25 cm from the incisors, consistent with a diagnosis of acute esophageal necrosis. The severity increased from the proximal to the distal esophagus, abruptly ending at the gastroesophageal junction.

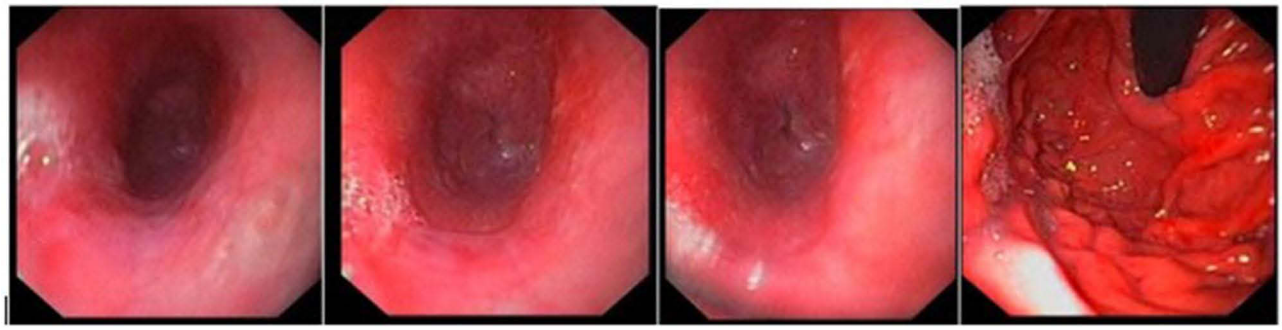

Figure 2 Esophagogastroduodenoscopy (EGD). Follow-up esophagogastroduodenoscopy (EGD) imaging conducted twenty-two days post-admission showing complete healing of the esophageal mucosa without stricture formation.

Discussion

Acute esophageal necrosis, also known as Gurvits syndrome or black esophagus, is a rare life-threatening cause of gastrointestinal bleeding that should be suspected in patients with cardiovascular risk factors with an EGD images of a black esophagus that is abruptly interrupted at the EGJ.[7,10] It is four times more common in males compared to females, and more common in older patients.[2] The most common comorbidities are diabetes mellitus, hypertension, and chronic kidney disease.[6] Other comorbidities of AEN include sepsis, congestive heart failure, pancreatitis, hypothermia, and shock.[11] Our patient suffered from both diabetes mellitus and hypertension. The exact cause of acute esophageal necrosis is unknown, but suggested mechanism is multi-factorial that results from ischemic compromise and thromboembolic injury to the mucosa of the esophagus.[9] Such ischemic compromise to the esophageal mucosa is commonly found in the distal third of the esophagus due to known its watersheds lower blood supply causing decreased vascularity of the area.[7] Common presentations of AEN include epigastric abdominal pain, hematemesis, nausea, and melena.[12] The diagnosis of AEN is based on EGD showing diffuse, circumferential patchy black mucosa of middle and distal thirds of the esophagus, with the abrupt transition of necrotic mucosa to normal mucosa at the gastroesophageal junction (GEJ).[9] Moreover, EGD is crucial for identifying complications of AEN, including esophageal perforation and stricture formation.[13] Esophageal perforation is the most serious complication of AEN and should be suspected in rapidly deteriorating patients.[2] Esophageal perforation may result in mediastinitis, mediastinal abscess, empyema, and sepsis. Additional complications of AEN include bleeding, superinfection, and the development of strictures or stenosis.[14] The differential diagnosis for AEN includes conditions such as malignant melanoma, acanthosis nigricans, melanosis, and coal dust deposition.[15] When feasible, a biopsy can be obtained to exclude bacterial, fungal, or viral infections, which are often associated with AEN.[16] Histopathological analysis of the affected area reveals inflammation and necrosis extending through the full thickness of the esophageal wall.[17] A biopsy was not taken in our case due to the risk of perforation.

No standard treatment exists for AEN; however, conservative management with bowel rest and proton pump inhibitors (PPIs) is recommended if complications are absent.[18,19] Management of AEN is focused on correcting the underlying disease, hemodynamic resuscitation with intravenous fluids, administering blood transfusion as needed, IV proton pump inhibitors, placing on nil-per-oral (NPO), total parenteral nutrition when necessary, and empirical antibiotics if there is a concomitant infection or concerns about esophageal perforation.[13] Surgical intervention for AEN is reserved for cases with complications such as esophageal perforation, mediastinitis, and abscess formation.[5] Patients with symptomatic esophageal stenosis or stricture may require endoscopic balloon dilation.[7] Acute esophageal necrosis (AEN) is associated with nearly 36% mortality rate, with an increased risk due to complications such as strictures and esophageal perforations.[13] Endoscopic intervention should be considered for patients with multiple risk factors, shock, and anemia. A follow-up EGD should be performed to evaluate mucosal healing.

Conclusion

Acute esophageal necrosis (AEN) is endoscopically characterized by a circumferential black discoloration of the esophagus, resulting from a combination of ischemic injury and thromboembolic damage. Esophageal perforation is

the most serious complication of AEN and should be suspected in rapidly deteriorating patients. Management of AEN is focused on treating the underlying disease, bowel rest, and PPIs. This case report aims to raise awareness among clinicians in Somalia of this condition as a differential diagnosis in upper gastrointestinal bleeding and to highlight the importance of prompt initiation of treatment to prevent adverse outcomes.

Ethics Approval

Institutional approval was not required for the publication of this case report.

Consent

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images. The patient was informed about the purpose of this publication, and consent included permission to share details of the case anonymously for educational and research purposes. Patient identity was protected to maintain confidentiality.

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Disclosure

The authors report no conflicts of interest in this work.

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