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Review Article
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Acute Esophageal Necrosis (Gurvits Syndrome) in a 78-Year-Old Male Filipino Patient: A Case

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Abstract

Background: Acute Esophageal Necrosis, also known as Gurvits Syndrome and commonly referred to as Black Esophagus, is a rare life threatening clinical entity with an estimated prevalence of 1 to 200 in 100,000. Only 88 patients over a span of 40 years have been reported with no published report among the Filipino population. Acute Esophageal Necrosis arises from a multifactorial background of ischemic insult from hemodynamic compromise and low-flow states, corrosive injury, and inadequate mucosal barrier function, usually in the setting of critical conditions.

Case: This is a case of a 78-year-old Filipino patient who developed hematemesis in the setting of hyperosmolar hyperglycemic state. An upper gastrointestinal endoscopy revealed a distinctly diffuse circumferential necrosis of the esophagus alone with abrupt demarcation at the gastro esophageal junction. Repeat endoscopy seven days after the episode showed pink mucosa after appropriate glycemic control, bowel rest and hemodynamic stabilization.

Conclusion: This is a report of a rare clinical disease in a male Filipino patient who presented with hematemesis and a distinct isolated esophageal necrosis on upper gastrointestinal endoscopy. The potential reversibility of this life threatening condition emphasizes the importance of timely recognition and appropriate management.

Keywords: Gurvits syndrome; Black esophagus; Acute esophageal necrosis

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Introduction

Acute Esophageal Necrosis (AEN) (also referred to as Gurvits Syndrome and Black Esophagus) was first described by Goldenberg et al. [1] in 1990 and as few as 88 patients have been diagnosed with AEN [2]. It has an estimated prevalence of between 1 and 200 per 100,000 [3]. Patients have multiple comorbidities and factors that result in hypoperfusion and/or corrosive injury to the esophagus, and they almost always present with gastrointestinal bleeding and associated symptoms. It is diagnosed on endoscopy where characteristically, a diffuse, circumferential, necrotic esophageal mucosa is seen. The overall mortality of AEN is around 32%, and it can result in life-long complications [4].

Case Presentation

A 78 year old Filipino male was admitted for the management of acute gouty arthritis on a background of recurrent attacks of gouty arthritis self-managed with chronic Non-Steroidal Anti-Inflammatory Drug (NSAID) use, poorly controlled type 2 diabetes mellitus, hypertension treated with intermittent use of nifedipine, and excessive alcohol consumption. The patient was initially treated with etoricoxib, dexamethasone, paracetamol and tramadol with relief of symptoms.

On day four, the patient had an acute coffee-ground, non-bilious hematemesis with associated epigastric pain. Prior to this episode, there were no complaints of dyspnea, dysphagia, odynophagia, nausea, vomiting or abdominal pain. There was no concomitant fever, respiratory distress, hypotension, melena or hematochezia. The patient was managed as a case of upper gastrointestinal bleeding likely secondary to a bleeding peptic ulcer secondary to chronic NSAID use and alcohol abuse. The patient was started on intravenous pantoprazole and intravenous hydration. A nasogastric tube was inserted with dark coffee-ground nonbilious drainage. Four hours after the episode, the patient was noted to be drowsy and confused with no focal neurological deficits. Capillary blood glucose reading registered "too high" and a subsequent serum osmolality was 384 mOsm/kg, consistent with concomitant Hyperosmolar Hyperglycemic State (HHS). The patient was started on an insulin regimen and an upper gastrointestinal endoscopy was performed.

Figure 1: Endoscopy revealed circumferential necrosis of the esophagus, with purulent exudates.

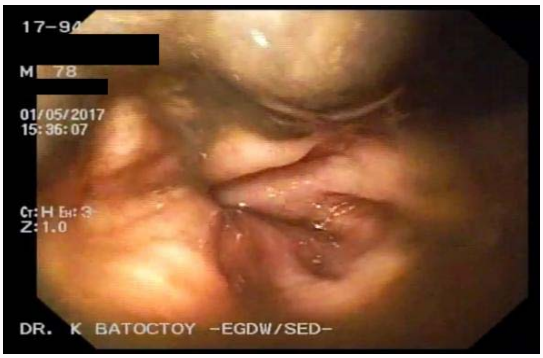

Figure 2: There was a notable abrupt normalization of the mucosa in the gastroesophageal junction in contrast with the diffusely black esophagus seen in Figure 1.

The endoscopy revealed a diffusely black non-distensible esophagus, commencing approximately 10 cm from the incisors, with thickened mucosa, multiple ulcers and thick purulent mucoid esophageal exudates (Figure 1). There was an abrupt demarcation in the gastro esophageal junction (Figure 2). The gastric mucosa was generally unremarkable with punctuate superficial erosions diffusely distributed throughout, and the duodenal mucosa was consistent with duodenitis. No active bleeding was observed. Biopsy of the esophagus was not done due to the risk of perforation. However, multiple gastric biopsy samples were taken which revealed a benign gastric mucosa with no significant histopathologic findings and were negative for Helicobacter pylori. A nasogastric tube was inserted under endoscopic guidance.

Post endoscopy, the patient was transferred to the Intensive Care Unit (ICU) where he was maintained on parenteral nutrition. Laboratory data showed leukocytosis of 24,000/μL with neutrophilic predominance, blood cultures revealed no growth, glycosylated hemoglobin was elevated at 10.8%, urinalysis displayed glucosuria and mild proteinuria, with noted increase in creatinine levels as high as 3.07 mg/dL. Whole abdominal ultrasonography showed multiple gallbladder stones with no evidence of cholecystitis. Chest CT scan revealed a distended aperistaltic esophagus with circumferential thickening with no evidence of perforation. Medications given were: meropenem, nystatin, pantoprazole and insulin. The patient stayed in the ICU for two days with improvement in glycemic control and sensorium with no further episodes of hematemesis.

A repeat upper gastrointestinal endoscopy was performed one week later (day 11). The previously necrotic esophagus significantly

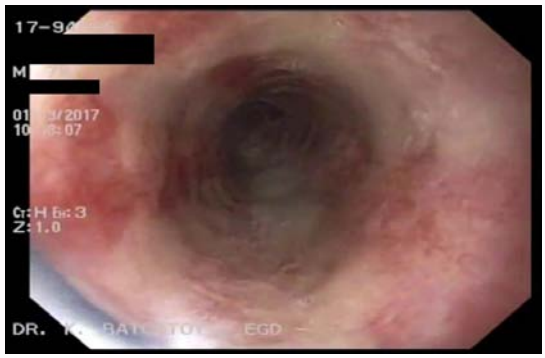

Figure 3: Repeat endoscopy one week after the initial endoscopy showed pink mucosa with occasional erosions.

improved with a pink viable esophageal mucosa and significant granulation tissue spanning throughout the esophagus, with few areas of erosion and occasional purulent discharges especially in the distal esophagus (Figure 3). The patient was subsequently discharged on day 14 in a stable and markedly improved condition.

Discussion

AEN has been associated with a number of factors and co-morbidities, including geriatric males, diabetes mellitus, hypertension, alcohol abuse, gastro esophageal reflux disease, chronic kidney disease, malnourishment, vascular disease, ingestion of caustic agents, and some medications [5, 6]. These factors predispose a patient to AEN through hemodynamic compromise, low-flow states, tissue hypoperfusion, and corrosive injury and diminished mucosal defenses [4]. The index case was an elderly hypertensive male with confounding uncontrolled diabetes mellitus in hyperglycemic state, acute kidney injury secondary to HHS, and alcohol abuse; who presented with hematemesis, which likely lead to further hemodynamic compromise and worsening AEN.

Upper gastrointestinal bleeding is the presenting complaint in around 90% of cases reported. Not surprisingly, the symptoms associated with AEN include abdominal pain, dysphagia, fever, nausea and syncope. Hypoalbuminemia is universal and commonly associated laboratory findings include anemia, renal insufficiency and hyperglycemia [5]. Diagnosis is made with endoscopy, with the hallmark characteristic of diffuse circumferential black discoloration of the esophagus, most markedly in the distal area. It classically ends abruptly at the gastro esophageal junction. The area of involvement is due to the decreased vascularity in the distal esophagus as well as the stomach's relatively inherent reparative mechanisms [5,7]. These classic endoscopic findings were evident in our case, and the decision not to biopsy was made to reduce the risk of complications. However, histologically, necrotic debris and the absence of viable epithelium with or without submucosal and muscularis propria involvement characterize this disease entity [4].

The treatment for AEN aims to address the underlying medical illness, maintain and restore hemodynamic stability with intravenous fluid resuscitation and blood transfusions, attain complete esophageal rest, gastric acid suppression with proton pump inhibitors, and antimicrobials if indicated. During the acute phase of AEN, patients are expected to present with altered vital signs, which may be the cause or the consequence of the esophageal insult [5]. Esophageal perforation is a life-threatening condition that should be suspected in the setting of acute decompensation and clinical deterioration

and requires immediate surgical intervention. Mediastinal infection or abscess formation may also develop. Subsequently, esophageal strictures, stenosis and death are possible complications anticipated in the resolution and recovery phase. This disease presents with a high clinical mortality of around 30% and is usually confounded by the underlying medical co-morbidities [2].

This is the first reported case of AEN in a Filipino patient and highlights the need for all clinicians to be mindful of it as populations develop more co-morbidities and risk factors that predispose them to AEN. Timely recognition and appropriate therapy is crucial to reduce the significant mortality risk.

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