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A CASE REPORT OF ACUTE ESOPHAGEAL NECROSIS: A RARE BUT POTENTIALLY FATAL CONDITION

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ABSTRACT Acute Esophageal Necrosis or AEN is a rare diagnosis that is characterized by diffuse, circumferential and black discoloration of the mucosa of the distal esophagus extending proximally to involve variable length of esophagus. It is thought to arise from a combination of ischemic insult due to hemodynamic compromise, acid reflux injury and decreased function of mucosal barrier systems present in malnourished and debilitated patients. We present a case of a middle aged diabetic male who presented with hematemesis associated with alcohol abuse. Esophagogastroduodenoscopy (EGD) confirmed black esophagus. He was managed successfully by keeping the patient nil per oral along with intravenous fluid resuscitation, broad-spectrum antibiotics, acid-suppressing agents and oral sucralfate suspension.

KEYWORDS : Black Esophagus, Acute Esophageal Necrosis, AEN, Gurvits Syndrome

INTRODUCTION

Acute Esophageal Necrosis or AEN is a rare diagnosis that is characterized by diffuse, circumferential and black discoloration of the mucosa of the distal esophagus that ends abruptly at the Gastro-esophageal junction and can extend proximally up to the entire length of the esophagus.[1] It is a purely endoscopic diagnosis and based on the black discoloration of the esophageal mucosa it is also known as the Black Esophagus or acute necrotizing esophagitis or Gurvits syndrome. The most common symptom of black esophagus is upper gastrointestinal bleeding: hematemesis, coffee-ground vomiting or melena. Other manifestations include epigastric pain, retrosternal chest discomfort and dysphagia[2]. The exact cause of the condition is not known but the pathophysiology most likely involves a blend of esophageal ischemia due to any cause, backflow injury from acidic gastric contents and impaired mucosal repair mechanisms associated with general debilitated physical condition of the patient. Risk factors include old age, male gender, hypertension, diabetes mellitus, coronary artery disease, alcohol abuse, renal insufficiency and advanced malignancies[3]. Here, we report a case of chronic alcoholic with black esophagus or Acute Esophageal Necrosis (AEN) who presented to us with hematemesis and ultimately improved with supportive care.

CASE PRESENTATION

A 50 years old male presented with a history of 3 episodes of coffee-ground emesis in last 2 days, with black colored stool and general malaise. He had a significant past history of Type II Diabetes Mellitus but on irregular treatment, chronic alcohol abuse (50-60 ml/day) and a chronic smoker (Smoking Index - 250). He had no history of hypertension, asthma, chronic NSAID use or corrosive ingestion nor any history of similar episodes in the past.

On arrival in the emergency room, he was afebrile, with a regular weak pulse of 112 bpm, a blood pressure of 70/40 mm/Hg and respiratory rate of 22 breaths/minute. A test irrigation with normal saline via a nasogastric tube was conducted and a red-brown liquid with clots drained.

Laboratory results were as follows: White blood cell count 13080/µl (neutrophils 68.5%), hemoglobin 14.4 g/dl, hematocrit 41.9%, platelets 2,10,000/µl, blood urea nitrogen 7.94 mg/dl, creatinine 1.0 mg/dl, alanine aminotransferase 25 IU/L, aspartate aminotransferase 18 IU/L, total bilirubin 0.5 mg/dl, direct bilirubin 0.2 mg/dl, alkanine phosphatase 84 IU/L, sodium 133 mEq/l, potassium 3.1 mmol/l, serum glucose 318 mg/dl, Prothrombin Time (PT) 21.3 s, International Normalized Ratio 1.85 and Activated Partial Thromboplastin Time (APTT) 49.8 s. Urine chemical examination was suggestive of glucosuria (1000 mg/dl), proteinuria (30 mg/dl) and ketonuria (52 mg/dl) consistent of diabetic ketosis. On arterial blood gas analysis, Ph 7.465, PO2 94 mmHg, PCO2 32.2 mmHg, HCO3 23.2 mmol/l was present. Patient was first stabilized with IV normal saline infusion and was kept nil per oral. After initial resuscitation, an esophagogastro-duodenoscopy (EGD) was performed that showed diffuse ulcerations and erosions throughout the length of esophagus along with black-grey discoloration of mucosa till the gastroesophageal junction (Fig I) and small erosions in first and second part of duodenum. Additional findings included Hill grade 4 hiatus hernia of esophagus, moderate inflammation and multiple erosions in body and antrum of stomach. Computed tomography (CT) of chest and abdomen showed diffuse esophageal wall thickening and surrounding edema (Fig II). The patient was kept nil per oral (NPO) and treated conservatively with high dose proton pump inhibitor, IV fluids and short acting insulin for management of diabetic ketosis. Patient improved and rest of the hospital stay was uneventful. On day 5, follow up EGD was performed (Fig III) which was suggestive of edematous esophageal mucosa with multiple erosive areas and thick white membrane overlying the mucosa throughout the length of esophagus, but with resolution of the black discoloration. The patient was discharged on day 7 after transitioning to clear liquids, without any complications, on oral PPIs, antacid syrup and Sucralfate syrup and advised for alcohol abstinence and strict glycemic control. Repeat EGD was done on day 37 (Fig IV) which showed that the white membrane covering the esophageal mucosa previously had been replaced by normal pinkish mucosa along with the presence of few longitudinal erosions up to 25 cm and with Mucosal breaks > 5mm seen in lower esophagus involving > 75% of the circumference.

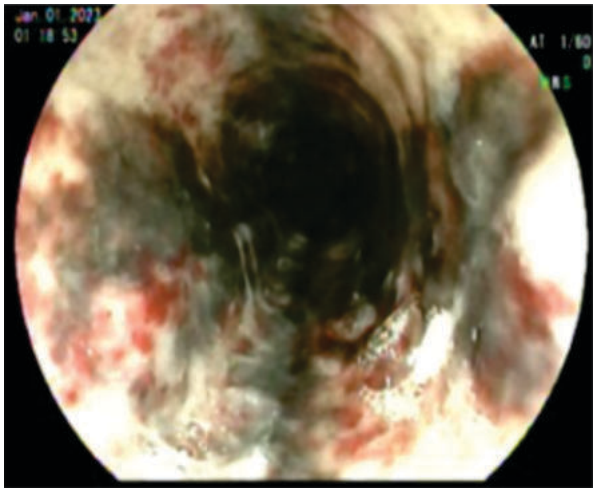

Fig I - EGD On Day 1 Showing Diffuse Ulcerations And Erosions Throughout The Length Of Esophagus Along With Black-grey Discoloration Of Mucosa

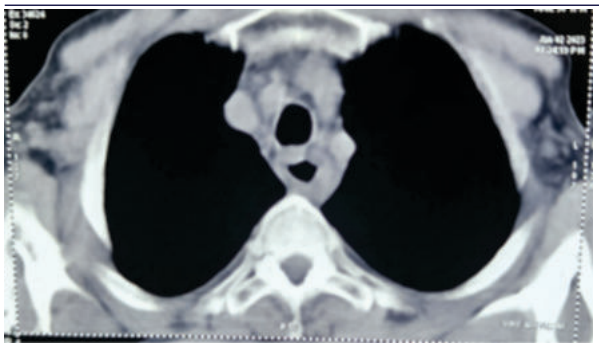

Fig II Computed Tomography (CT) Of Chest Showing Diffuse Esophageal Wall Thickening And Surrounding Edema

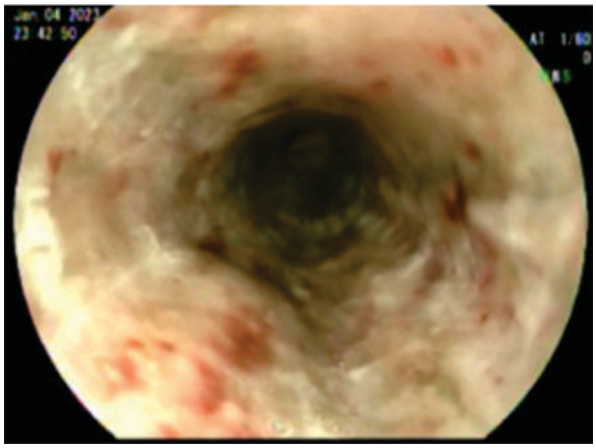

Fig III - EGD On Day 5 Showing Edematous Esophageal Mucosa With Multiple Erosive Areas And Thick White Membrane Overlying The Mucosa

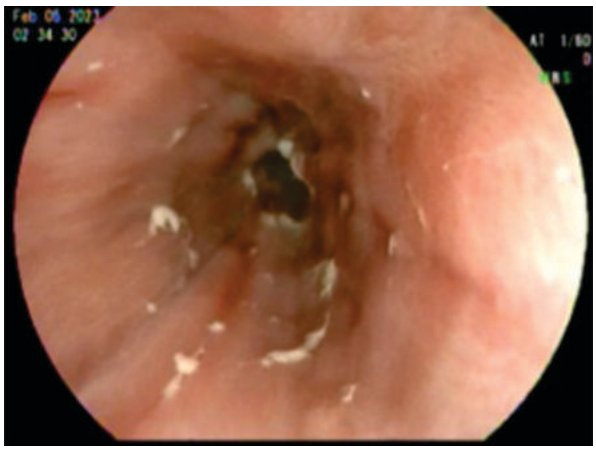

Fig IV - EGD On Day 37 Showing Pink Esophageal Mucosa Along With Few Longitudinal Erosions

DISCUSSION

Black esophagus was first reported as a clinical entity in 1990 by Goldenberg et al [1]. The reported incidence of black esophagus is very low, ranging from 0.0125% to 0.2%.[4,5] It is mostly seen in the elderly usually in the sixth decade with a male predominance of 2.3:1 [6].

Endoscopic appearance shows a diffuse blackish discoloration of the distal esophagus that affects it circumferentially [2].

Similar endoscopic appearance of black esophagus may also be seen in Malignant melanoma, acanthosis nigricans, pseudomelanosis, melanosis, coal dust deposition and corrosive ingestion. However, the term 'black esophagus' most often represents acute esophageal necrosis.

Although the etiology of this condition remains unclear, it is likely multifactorial in origin. A combination of two factors, one that can possibly lead to mucosal injury, that is, reflux of gastric contents, and another that decreases the blood supply of the esophagus such as shock, is thought to be involved in the pathogenesis of this disease [3,7,8]. This "two-hit" model of ischemia and acid reflux injury has been proposed in the literature as a possible explanation of esophageal necrosis [9].

Multiple comorbidities can precipitate this condition. Diabetes mellitus is one of the common accentuating factors, and hyperglycemia is found in 90% of the patients presenting with esophageal necrosis [10]. Malnutrition has also been suggested to be one of the factors involved in accelerating the disease process [11]. Other risk factors mentioned in previous reports include gastric outlet obstruction, gastric volvulus, shock, aortic dissection, diabetic ketoacidosis, alcohol abuse, etc.

In our case, risk factors including male gender, diabetes mellitus, alcohol abuse with acute hypotensive state may have predisposed the patient to esophageal ischemia. The average daily consumption of alcohol was 50-60 mL in our patient. Alcohol ingestion has been reported to reduce lower esophageal sphincter pressure and esophageal peristalsis and increase acid exposure in the esophagus. [12] Excessive alcohol consumption also causes irritation of the gastric mucosa and the accumulation of large volume of gastric secretions which disrupts the defense mechanism of the esophagus.

The treatment is usually supportive care by keeping the patient nil per oral, intravenous fluid resuscitation, broad-spectrum antibiotics, acid-suppressing agents, oral sucralfate suspension, and avoidance of factors that can precipitate the illness such as hyperglycemia. Short-term total parenteral nutrition can be given to malnourished individuals. Additionally, in our case, abstinence from alcohol is recommended which may have been an aggravating factor.

Although perforation is rare, it is the most fatal acute complication of esophageal necrosis. If it occurs, esophagectomy is the surgical management [13]. Stricture formation is also seen in the chronic phase of the disease in some patients. The mortality of esophageal necrosis ranges from 30% to 50% depending upon the etiology [3]. However, in our case, the esophageal lesions improved significantly in the gross appearance of the esophageal mucosa, without any complication so far. In conclusion, we report a rare case of black esophagus in a middle aged male patient with several comorbid conditions predisposing to esophageal ischemia and a history of alcohol abuse which was successfully treated conservatively, with IV fluid resuscitation, hydration, acid suppression and control of hyperglycemia along with management of diabetic ketosis.

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