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Case Report

Acute Esophageal Necrosis (Gurvits Syndrome) in the Setting of Diabetic Ketoacidosis: A Case Report

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Abstract:

Background:

Acute esophageal necrosis (AEN), also known as Gurvits syndrome, is a rare and severe condition characterized by circumferential black discoloration of the esophageal mucosa, typically involving the distal segment and commonly associated with systemic hypoperfusion and metabolic disturbances.

Case presentation:

We report the case of a 52-year-old man with type 2 diabetes mellitus admitted for diabetic ketoacidosis (DKA). During hospitalization, he developed persistent epigastric pain and intractable vomiting. Upper gastrointestinal endoscopy revealed diffuse circumferential black discoloration of the distal esophagus, consistent with AEN.

Management and outcome:

The patient was managed with fluid resuscitation, intravenous insulin, proton pump inhibitors, parenteral nutrition, and broad-spectrum antimicrobial therapy. Clinical and laboratory parameters improved rapidly, with complete resolution of symptoms. Follow-up endoscopy confirmed full mucosal recovery.

Conclusion:

AEN is a rare but potentially life-threatening complication of DKA. Early recognition and prompt supportive management are essential to improve outcomes.

Key words: diabetic ketoacidosis, acute esophageal necrosis

Introduction

Acute esophageal necrosis (AEN), first described by Goldenberg et al. and later termed Gurvits syndrome, is a rare clinical entity with a reported prevalence of less than 0.2% in endoscopic series [1,2]. It is characterized by a striking endoscopic appearance of circumferential black discoloration of the esophageal mucosa, most commonly affecting the distal third with a sharp demarcation at the gastroesophageal junction [3].

The condition is typically associated with states of hypoperfusion and metabolic derangement, including diabetic ketoacidosis [4].

We report a case of acute esophageal necrosis occurring in the setting of diabetic ketoacidosis, highlighting the importance of early endoscopic evaluation in patients with persistent gastrointestinal symptoms.

Case Presentation

We report the case of a 52-year-old man with a history of insulin-treated type 2 diabetes mellitus and bipolar disorder treated with antipsychotics, who was admitted for diabetic ketoacidosis.

On presentation, he reported severe epigastric pain associated with persistent vomiting, initially food-containing and later bilious with blood streaks (approximately 10 episodes/day). He was afebrile, tachycardic (110 bpm), and normotensive (120/80 mmHg). Abdominal examination revealed epigastric tenderness without peritoneal signs.

Laboratory investigations showed marked hyperglycemia (6.2 g/L), ketonuria (3+), leukocytosis (25,000/mm^3), elevated C-reactive protein (35 mg/L), acute kidney injury (creatinine 135 µmol/L), hyponatremia (131 mmol/L), and severe hypokalemia (2.4 mmol/L). Arterial blood gas

analysis confirmed metabolic acidosis (pH 7.32, bicarbonate 16 mmol/L). Liver enzymes and lipase levels were within normal ranges.

No clear precipitating factor was identified. Cardiac causes were excluded (normal electrocardiogram and troponins), and infectious workup, including urine analysis and chest X-ray, was unremarkable.

The patient was treated with intravenous insulin, fluid resuscitation, and electrolyte correction. Despite appropriate management, vomiting and epigastric pain persisted.

A thoraco-abdominopelvic CT scan revealed bilateral lower lobe bronchiolitis suggestive of aspiration, a small pericardial effusion, and diffuse circumferential thickening of the distal esophageal wall without luminal stenosis.

Upper gastrointestinal endoscopy, performed 36 hours after admission, showed longitudinal ulcerations in the proximal esophagus. From 25 cm distal to the dental arches, the esophageal mucosa appeared diffusely black and circumferential, extending to 38 cm, with a sharp demarcation line just above the gastroesophageal junction. Associated erythematous gastropathy was also noted.

Findings were consistent with acute esophageal necrosis.

Management included bowel rest, parenteral nutrition, high-dose proton pump inhibitors, broad-spectrum intravenous antibiotics (third-generation cephalosporin and metronidazole), antifungal therapy (fluconazole), continued insulin therapy, and correction of metabolic abnormalities.

Clinical and biological parameters improved within five days, with resolution of vomiting and normalization of renal function, electrolyte levels, and glycemic control. The patient was discharged on day 7.

Follow-up endoscopy at 4 weeks demonstrated complete resolution of esophageal lesions, confirming the diagnosis of reversible acute esophageal necrosis (Gurvits syndrome).

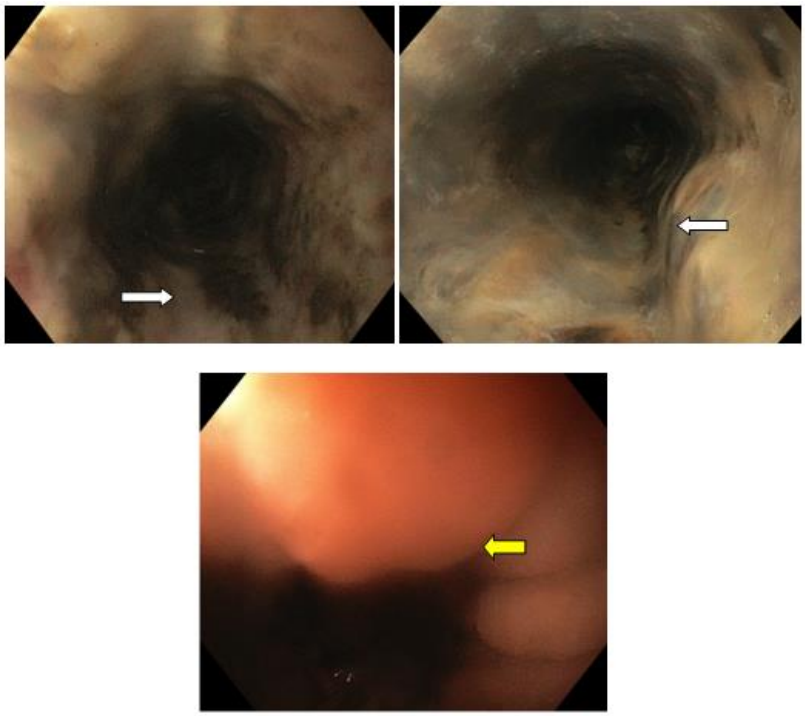

Figure 1: Endoscopic view of circumferential necrosis of the distal third of the oesophagus (White arrows indicate areas of esophageal necrosis) with a demarcation line at the cardia (The yellow arrow indicates the gastroesophageal junction)

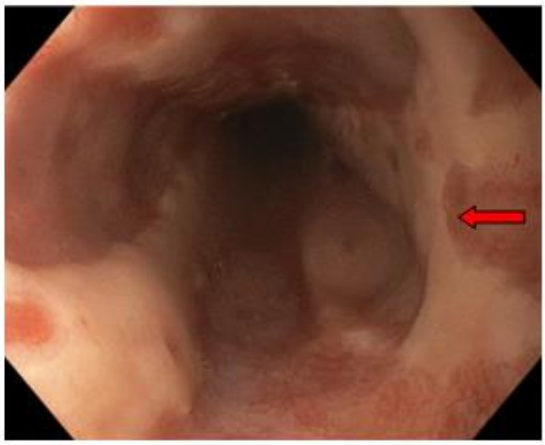

Figure 2: Endoscopic appearance of ulcerative oesophagitis in the proximal third of the oesophagus (red arrow)

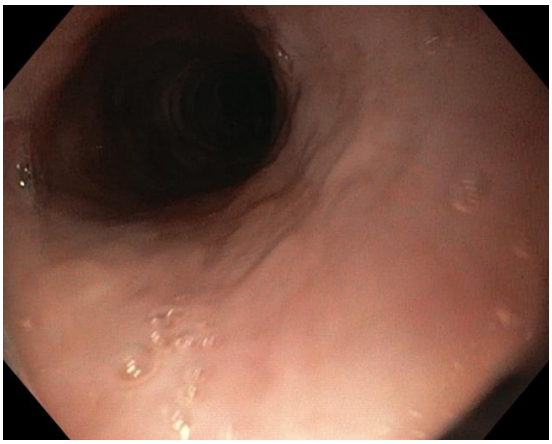

Figure 3: Normal endoscopic appearance of the oesophageal mucosa after 4 weeks

Discussion

Acute esophageal necrosis (AEN), or "black esophagus," is a rare condition with a reported prevalence of 0.01–0.3% [1,2,5]. First described by Goldenberg et al. in 1990, it predominantly affects older men with multiple comorbidities [6]. Diabetes mellitus, cardiovascular disease, renal insufficiency, and states of hypoperfusion are frequently associated [7,8].

The pathophysiology is multifactorial and classically explained by a "two-hit" mechanism combining ischemic injury and gastric acid exposure [2]. The distal esophagus is particularly vulnerable due to its relatively poor vascularization. Diabetic ketoacidosis (DKA) represents a typical precipitating condition, as hypovolemia, metabolic acidosis, and adrenergic vasoconstriction contribute to splanchnic hypoperfusion, while repeated vomiting increases mucosal exposure to gastric acid [9]. Nevertheless, cases without clear ischemic triggers suggest additional mechanisms [10,11].

Clinically, AEN most often presents with upper gastrointestinal bleeding, epigastric pain, or dysphagia [12]. Diagnosis is based on endoscopy, which reveals a circumferential black discoloration of the esophageal mucosa, typically involving the distal segment with a sharp demarcation at the gastroesophageal junction [13]. Differential diagnoses include melanoma, melanosis, acanthosis nigricans, and caustic injury [11–14]. Biopsies can help rule out other possible diagnoses but are not mandatory due to the risk of oesophageal perforation, given the fragility of the mucosa [14].

Management is mainly supportive, including fluid resuscitation, bowel rest, proton pump inhibitors, and treatment of the underlying cause [12]. Antibiotics are reserved for selected cases [15,16]. Prognosis depends largely on comorbidities, with a reported mortality of approximately 30% [6,17]. Complications include perforation, infection, and, in the longer term, esophageal strictures [2,6].

This case highlights diabetic ketoacidosis as a key reversible trigger of AEN, emphasizing the importance of early recognition and supportive management to achieve complete mucosal recovery.

Conclusion

AEN should be considered in patients with DKA and persistent vomiting. Early endoscopy is essential to establish the diagnosis and initiate appropriate management.

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