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MULTIDISCIPLINARY MANAGEMENT OF A PATIENT WITH ACUTE OESOPHAGEAL NECROSIS AND SEVERE NEUROLOGICAL SYMPTOMS WITH FULL RECOVERY

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ABSTRACT

Background: Acute oesophageal necrosis is a rare and poorly understood disorder of the oesophagus. It poses significant diagnostic and therapeutic challenges as it often presents with multiple comorbidities and a poor prognosis.

Case report: We report a case of 42-year-old male with no significant medical history, who presented to our clinic due to neurological deficits and repeated vomiting for 2 weeks. He initially denied alcohol abuse. Upper endoscopy demonstrated circular black discoloration of the distal oesophagus. The patient was treated with proton pump inhibitors and received treatment for his comorbidities. Esophagogastroduodenoscopy was performed to assess improvement. The patient's symptoms gradually resolved.

Conclusion: This case is one of the few to show an association between alcohol abuse and acute oesophageal necrosis and underscores the importance of timely diagnosis and multidisciplinary treatment in patient with fully recovered condition.

KEYWORDS

Acute oesophageal necrosis, black oesophagus, oesophageal necrosis

LEARNING POINTS

- Acute oesophageal necrosis (AEN) is rare but serious condition often overlooked in patients with gastrointestinal symptoms, particularly younger patients and those with a history of alcohol abuse.
- This case illustrates how a single patient can have multiple acute conditions (AEN, pancreatitis, stomach and duodenal ulcers), necessitating a multidisciplinary approach to care.

INTRODUCTION

Acute oesophageal necrosis (AEN) is a rare condition characterized by the black appearance of the oesophagus, also known as black oesophagus. AEN affects males more frequently than females. Other risk factors include increased age, heart disease, trauma, diabetic ketoacidosis, malignancy and alcohol abuse[1]. The pathophysiology of this acute condition remains poorly understood. There

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are currently no guidelines for the management of AEN, and the disorder is associated with a poor prognosis. Esophagogastroduodenoscopy (EGD) is considered the gold standard for diagnosis. The characteristic appearance on upper endoscopy is circumferentially black appearing areas in the oesophageal mucosa, that stop at the gastroesophageal junction. Biopsy is not required to establish the diagnosis, although it is recommended to exclude local infection or malignancy. Treatment involves timely management with supportive care including proton pump inhibitors (PPI), intravenous fluid resuscitation and treatment of the underlying aetiology[2]. PPI therapy should be continued for a few months after symptoms resolve. Antimicrobial therapy is not required with exception of cases of unexplained fever, suspected oesophageal perforation or in immunocompromised patients[3].

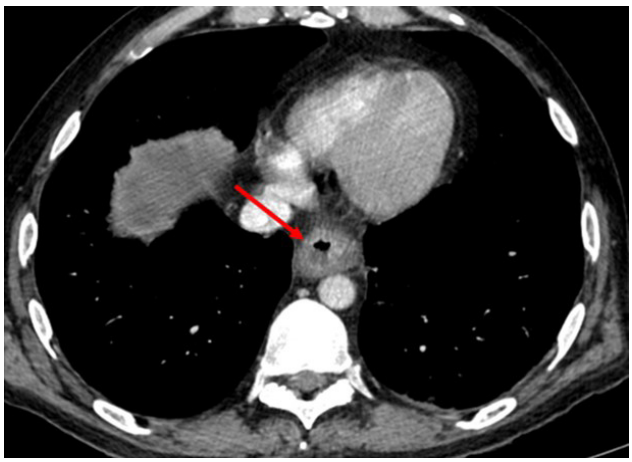


Figure 1. The distal third of the esophagus with asymmetrically thickened walls (red arrow) on axial contrast-enhanced computed tomography scan.

CASE DESCRIPTION

A 42-year-old male presented to our clinic complaining of feeling unwell, experiencing double vision and dizziness. He reported skipping meals for the last week due to high stress and repeated vomiting. The patient had a history of alcohol abuse and denied any chronic diseases. During neurological examination, the patient had ocular convergence, spontaneous rotator nystagmus to the right, left and upward view; dysarthria; dysmetria with both arms; astasis - inability to stand or walk without support due to severe combined ataxia (cerebellar and sensory). Symmetrical, distal hypoesthesia starting from the mid-shin was also present, suggesting polyneuropathy.

At the emergency department, computed tomography (CT) scan of the head and lumbar puncture showed no pathological changes. Chest CT with contrast revealed the distal third of the esophagus with an asymmetrically thickened wall *(Fig. 1)*, proximal esophageal stasis, and bilateral pneumonia. Later magnetic resonance imaging (MRI) of the brain was performed and findings were consistent with Wernicke encephalopathy. Blood tests indicated electrolyte imbalance including hyponatremia (121 mmol/l), hypokalaemia (3.4 mmol/l) and low folic acid <2.2 ng/ml), high lipase (157 U/l), hyperglycaemia (7.3 mmol/l), elevated C-reactive protein (CRP) (28.93 mg/l). The complete blood count revealed leucocytosis (22.3 $\times 10^3$/µl), and neutrophilia (19.6 $\times 10^3$/µl). Coagulation tests showed coagulopathy; prothrombin time 17.2 s, prothrombin index 43.3% and international normalized ratio (INR) 1.58. All values returned to normal as a result of treatment.

During hospitalization the patient had melena and diarrhoea. Therefore, EGD was performed, revealing AEN characterized by circular ulcers covered in black hematin *(Fig. 2)* and multiple ulcers in the gastric pylorus (Forrest III), bulbus and descending part of duodenum (Forrest IIc). Additionally, hiatal hernia, hyperchromic macrocytic anaemia and acute pancreatitis were diagnosed.

Thiamine supplementation, PPI and antimicrobial therapy, betamethasone, and intravenous (IV) fluids were administered throughout hospitalization. EGD 8 days later showed a healing process: fibrin granulation tissue in the distal part of the oesophagus *(Fig. 3)*. Subsequently, the patient was discharged with recommended lifestyle and dietary modifications and oral PPI, antacid and folic acid therapy.

At discharge the patient had mild neurological symptoms: binocular horizontal fixation nystagmus to right and left, mild dysarthria, action tremor (postural and kinetic). He was able to stand and walk freely without assistance or walking aids. Three months later the patient did not have any neurological or gastrointestinal symptoms.

DISCUSSION

AEN, also known as acute necrotizing esophagitis and 'black oesophagus', is an uncommon condition observed on EGD, usually characterized by dark mucosa in the distal third of the oesophagus. A review of the literature confirms that AEN is a rare disease, highlighting that the true prevalence of AEN is unknown and most likely underestimated due to the occasionally subclinical presentation[4].

This case report described a 42-year-old male with a history of alcohol abuse who exhibited neurological symptoms alongside signs of gastrointestinal bleeding, ultimately diagnosed with Wernicke encephalopathy and AEN. The combination of neurological symptoms and gastrointestinal bleeding in this patient underscores the complex interplay between alcohol abuse, nutritional deficiencies, and gastrointestinal health.

Most previous case reports have documented cases of AEN primarily in older patients with significant comorbidities[1]. This case illustrates that even in the absence of overt chronic disease, acute stressors can precipitate severe gastrointestinal complications in individuals with a history of alcohol abuse. Although AEN is a serious clinical condition, mortality is generally related to underlying disease and can reach 32%[4].

This case highlights the importance of considering AEN in patients presenting with gastrointestinal symptoms,



Figure 2. Esophagogastroduodenoscopy images showing black pigmentation in the oesophagus of the patient.

particularly those with history of alcohol abuse. It emphasizes the need for prompt endoscopic evaluation in similar clinical scenarios.

Other cases of AEN caused by alcohol consumption have also been described[5], but most of the cases were associated with other etiological factors, such as diabetes mellitus, gastroesophageal reflux disease[5], paraesophageal hernia[6], infections (untreated hepatitis C, human immunodeficiency virus)[7] or complex and serious general condition[8]. All of the previously mentioned cases involved patients in their sixth and seventh decade of life, who were predominantly male, some showing symptoms of upper gastrointestinal bleeding. There are no widely accepted guidelines on managing AEN, but in our case timely diagnostics, PPI therapy and treatment of other comorbidities resulted in improvement and the patient was discharged. The case also shows the importance of repeated endoscopies to monitor treatment and screen for likelihood of developing strictures. In this case, the second endoscopy 8 days later clearly showed fibrin granulation tissue indicating healing of mucosal lining. Other case reports also use repeat EGD to assess resolution of endoscopic findings[5,7]. There is no consensus to the timing of repeated EGD. Endoscopy has a pivotal role in the diagnosis, risk stratification, and treatment of significant upper gastrointestinal haemorrhage (UGIH). In case of refractory bleeding post-endoscopic haemostasis (first line treatment in UGIH), transcatheter arterial embolization (TAE) is the treatment of choice. TAE provides non-invasive, accurate, sensitive, and rapid diagnosis of haemorrhage in a wide spectrum of clinical scenarios. TAE also aids in the planning of embolization, reducing subsequent angiographic radiation dose, contrast volume, and procedure time.

A high percentage (95-97%), as in this case of nonvariceal UGIH, is controlled by combination of medical therapy with endoscopic interventions for lesions that are actively bleeding or carry a high risk of recurrent bleeding. Embolization and surgery are reserved for those patients for whom endoscopy has failed to achieve definitive haemostasis. Since its initial role in the management of poor surgical candidates, it has expanded to become the preferred treatment when endoscopic intervention fails[9,10]. The endoscopist can assist the interventionist by placing endoscopic clips at the site of an endoscopically identified but uncontrollable hemorrhage to facilitate embolization, even if the patient has temporarily stopped bleeding[11]. There are no absolute contraindications for embolization. Embolization is rarely indicated in low-volume, non-life-threatening hemorrhage as in this case. Complications of AEN include oesophageal strictures and stenosis, perforation and life-threatening bleeding. A case of AEN, oesophageal perforation and mediastinitis was reported in a 67-year-old female with a history of diabetes mellitus. Perforation was treated surgically with oesophagectomy with oesophagostomy and enterostomy. Subsequently, the patient developed stenosis in the upper oesophagus and cervical esophagectomy, tracheotomy and reconstruction with free jejunum interposition were performed, allowing gastric tube insertion[12].

After patient discharge from hospital, in case of complaints, barium oesophagography could be suggested. In case of oesophageal strictures that can occur as the result of collagen deposition and fibrous tissue formation there are no absolute contraindications for intervention with ballons or stents. As this is a single case report, caution should

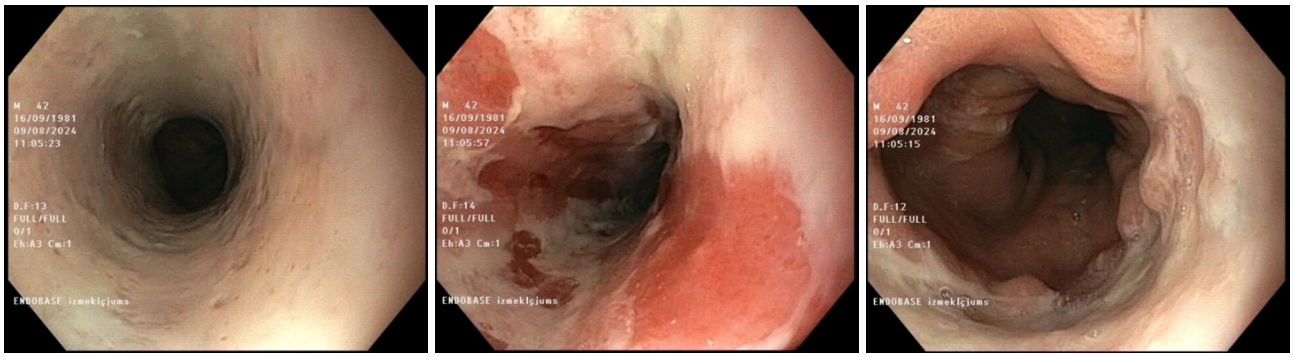


Figure 3. Esophagogastroduodenoscopy showing resolving lesions 8 days later.

be exercised when generalizing these findings to broader patient groups. Further studies are needed to explore the prevalence of AEN in similar patient demographics and results to treatment.

CONCLUSIONS

AEN is a rare condition, which often manifests non-specifically and poses a major challenge in diagnosis and treatment. AEN can be found in patients with multiple comorbidities, making a multidisciplinary approach the cornerstone of therapy.

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