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ACUTE ESOPHAGEAL NECROSIS IN THE ALCOHOLIC PATIENT: CASE PRESENTATION

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ABSTRACT

Acute esophageal necrosis (AEN) is an uncommon but fatal cause of upper gastrointestinal bleeding with an incidence of 0.01%–0.2%. Endoscopically, it is characterized by circumferential or diffuse black pigmentation of the esophageal mucosa, conditioned by mucosal necrosis. Risk factors include gender (male), advanced age, cardiovascular disease, hemodynamic insufficiency, alcohol consumption, diabetic ketoacidosis, malnutrition, kidney disease, and trauma. Diagnosis is based on esophagogastroduodenoscopy (EGD). Treatment of AEN consists of intravenous fluids, proton pump inhibitors (PPIs), sucralfate, parenteral nutrition, and antacids. We report a rare case from our hospital, emphasizing the importance of prompt diagnosis and intervention in case management. A 64-year-old man with a history of heavy drinking was hospitalized for severe bleeding (melena) caused by a condition called acute esophageal necrosis. He underwent an emergency procedure to examine his esophagus (EGD) and found significant damage. This case illustrates a rare etiology of AEN due to active alcohol consumption that may be overlooked. The early recognition of this clinical entity is an essential factor in the therapeutic management of the disease.

Keywords: Acute esophageal necrosis (AEN), esophagogastroduodenoscopy (EGD), proton pump inhibitors (PPIs), ischemia.

Introduction:

Acute esophageal necrosis (AEN) can be fatal. It's also called black esophagus, Gurvit syndrome, or acute necrotizing esophagitis. It's a rare condition that happens when the esophagus is damaged due to a lack of blood flow and other factors. Discovered in 1990, the exact cause of AEN is still unknown (1). This considerably rare condition has an estimated prevalence of 0.01% to 0.2%, documented results based on retrospective autopsy studies performed at 30 years (2). The pathogenesis and etiology of AEN are multifactorial and may be related to various factors, such as reflux of gastric contents associated with esophageal injury, interruption of vascular supply leading to hypoperfusion and ischemia, and damage to protective barrier systems due to a weakened immune system and/or a hemodynamically unstable state (3).

Acute esophageal necrosis (AEN) is macroscopically characterized by black pigmentation of the esophageal mucosa, the pigmentation being more pronounced in the lower esophageal mucosa and never crossing the gastro-esophageal junction, i.e. the boundary between the esophageal and gastric mucosa. Histologically, it presents as necrotic tissue and acute inflammation in the mucosa, submucosa and muscularis propria accompanied by brown

pigmentation, which is limited to the mucosa (4).

AEN is more likely to occur in people with conditions like hiatal hernia, injuries, diabetes, heart problems, high blood pressure, blood clots, kidney disease, liver disease, poor nutrition, heavy drinking, radiation damage, or cancer. Studies have shown that macular sex is predominant, usually in the sixth decade of life.

Treatment for AEN usually involves the administration of blood transfusions, intravenous fluids, proton pump inhibitors (PPIs), sucralfate, parenteral nutrition, and antacids. To avoid the risk of esophageal perforation, nasogastric tubes are not recommended (5). Antibiotics should be used if there is a severe infection (sepsis) or if the esophagus has been punctured (perforated) (6).

Table 1 AEN differential diagnoses

AEN differential diagnoses
Malignant melanoma
Acanthosis nigricans
Coal dust deposition
Pseudomelosis
Melanosis of the esophagus
Ingestion of black dye
Direct caustic injuries

The differential diagnoses of AEN have been listed in Table (7); AEN is a serious condition with a high death rate of over 30%. It needs to be treated quickly to improve chances of survival (2, 8, 9). Because AEN often occurs with other health problems, it can be hard to determine if it's the cause of death. Taking this into account, the specific mortality of AEN has been observed to be as high as 5% (10).

Case presentation

In this work we present the case of a male patient, of rural origin, aged 64, an active alcohol user, who presented himself at the emergency service, within the Municipal Hospital, St. Hierarch Dr. Luca,, Onești due to multiple episodes of melena, with an onset of approximately 3 weeks, declarative, lipothymic state and hypotension. When the patient arrived at the emergency room, their heart rate was 118 beats per minute, blood pressure was 110/60 mm Hg, temperature was 36.8°C, and breathing rate was 18 breaths per minute. Clinically, upon admission, the patient had an affected general condition, enlarged abdomen due to ascites fluid, pale integuments and mucous membranes. The paraclinical picture reflects an acute and complicated chronic ethanolic hepatopathy with upper digestive hemorrhage (HDS), hemorrhagic shock, severe macrocytic anemia (chronic and acute-hemorrhagic component), significant hepatocytolysis syndrome, mild thrombocytopenia (+platelet dysfunction), coagulopathy (reduction of synthesis of plasma coagulation factors), inflammatory syndrome (chronic +/- acute inflammation caused by tissue damage and superinfections with various pathogens), lactic acidosis (through the accumulation of serum lactate due to tissue hypoxia and liver hypofunction). The initial laboratory results suggestive of prolonged alcohol consumption were noted in the Table 2.

Table 2 The initial laboratory findings (data collected from the general clinical observation sheet, as well as from the database of the InfoWord program Municipal Hospital, St. Hierarch Dr. Luca, Onești)

Laboratory parameter	Value	Reference range
Leukocyte	12.66	4-10/µL
RBC	1.92	4.2-5.6 10/µL
Hemoglobin	7.6	13,1 - 17,2 g/dl
Hematocrit	22.2	39,0 - 50,0%
MCV	115.1	81-101 fL
Platelets	145	150 – 450 10/µL
Sodium	139.28	146 – 157 mmol/L
Potassium	5.2	3,7 - 5,5 mmol/L
Bicarbonate	22.0	24 - 30 mmol/L
Chloride	90.9	101 - 110 mmol/L
Lac	14.6	0.3-0.8 mmol/L
Creatinine	0.43	0.7 - 1,2 mg/dl
AP	59.4	74.4 - 120 %
INR	1.34	0.80-1.20
Quik Time	12.0	8.40-10.60
Quantitative D-dimer	1.91	0-0.5 µg/mL
Creatinine	0.97	0.7-1.2 mg/dl
Amylase	144	28-100 U/L
GT range	1475	0-71 U/L
LDH	297	135-225 U/L
Lipase	40	13-60 mg/dl
TGO	170	0-40 U/L
TGP	62	0-41 U/L
Serum urea	84	16.6-48.5 mg/dl
Creatine	99.7	20-200 U/L
Quantitative CRP	21.34	0-5 mg/L

An emergency upper gastrointestinal endoscopy was performed which revealed severe diffuse mucosal changes with black pigmentation of the esophageal mucosa in the distal third of the esophagus due to ischemic necrosis (Figure 1). No esophageal varices were observed at endoscopy. Biopsy was avoided given the increased risk of perforation.

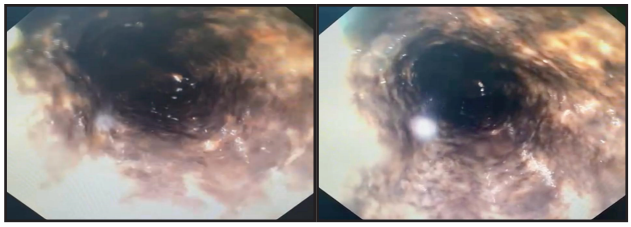

Figure 1. Endoscopic image, on the first day, in a patient diagnosed with circumferential necrosis of the esophagus

Multimodal treatment was initiated by volume repletion of hydroelectrolytic balancing, proton pump inhibitor (PPI), hemostatic agents were administered, severe anemia was corrected by blood transfusion, antibiotics, analgesics and sucralfate suspensions.

After diagnosis and administration of treatment, the patient did not show a favorable evolution. After 4 days of administered treatment, the patient's clinical condition progressively deteriorated, developing multiple organ dysfunction, he became hemodynamically and respiratory unstable, required vasopressor and positive inotropic support, ventilatory support and oxygen therapy. Cardiorespiratory arrest (CPR) was instituted, the resuscitation protocol was initiated, the result was negative, and the patient was pronounced dead. The autopsy was not performed, due to the family's refusal.

Discussion

Cases of esophageal necrosis, termed "black esophagus," have been described and attributed to alcohol abuse. Endo T, Sakamoto J, Sato K, Takimoto M, Shimaya K, Mikami T, et al. (11) hypothesized that acute esophageal necrosis is caused by lactic acidosis secondary to alcohol ingestion, which in turn may lead to a poor perfusion (12). In addition, ethanol, its metabolite acetaldehyde, and other fermentation products can cause direct mucosal damage and increase the production of gastric secretions (13, 14), all of which can contribute to gastric necrosis. Direct mucosal damage from these substances, in turn, makes the gastric mucosa more susceptible to damage from gastric acid, bile, and other digestive enzymes (12). Alcohol abuse induces irritation of the gastric mucosa and the accumulation of a large volume of gastric secretions. This can disrupt the defense mechanism of the esophagus and could be another cause of our case. In this case, the cause of AEN was heavy drinking and the health problems that came with it.

AEN often causes stomach pain, vomiting blood, nausea, and bleeding in the stool. An EGD procedure is usually needed to diagnose AEN because it can directly see the damaged, black areas in the esophagus (15). Sometimes, a biopsy is done to rule out other infections that can look like AEN. While a biopsy is not always necessary for diagnosis, it can help determine the exact cause of the damage and guide treatment. A biopsy usually shows inflammation and tissue death in the esophageal wall (15-17). In our case, a biopsy was not done because of the risk of causing a hole in the esophagus.

The main goal of AEN treatment is to replace fluids, blood, and use medications to reduce stomach acid and prevent bleeding (15, 16).

Conclusions

If someone who drinks heavily has bleeding in the upper part of their digestive system, AEN should be considered. An EGD procedure is the best way to diagnose AEN, and a biopsy is recommended to confirm the diagnosis, unless there's a high risk of damaging the esophagus. To prevent AEN, it is important to address other health problems like heart disease, liver disease, and kidney disease. There is no known way to medically prevent AEN, and more research is needed to understand how heavy drinking can cause this condition.

References

1. Goldenberg SP, Wain SL, Marignani P. Acute necrotizing esophagitis. Gastroenterology.

1990 Feb;98(2):493-6.
2. Day A, Sayegh M. Acute oesophageal necrosis: a case report and review of the literature. Int J Surg. 2010;8(1):6-14.
3. Rehman O, Jaferi U, Padda I, Khehra N, Atwal H, Parmar M. Epidemiology, Pathogenesis, and Clinical Manifestations of Acute Esophageal Necrosis in Adults. Cureus. 2021 Jul;13(7):e16618.
4. Sano H, Kibayashi K, Shimada R, Nakao KI. Analysis of the pathogenesis of acute necrotizing esophagitis (black esophagus): A report of three autopsy cases. J Forensic Leg Med. 2023 May;96:102515.
5. Shafa S, Sharma N, Keshishian J, Dellon ES. The Black Esophagus: A Rare But Deadly Disease. ACG Case Rep J. 2016 Jan;3(2):88-91.
6. Dias E, Santos-Antunes J, Macedo G. Diagnosis and management of acute esophageal necrosis. Ann Gastroenterol. 2019 Nov-Dec;32(6):529-40.
7. Richards J, Wei R, F A. Esophageal Necrosis. StatPearls Treasure Island. StatPearls Publishing2024.
8. Sheikh AB, Mirza S, Abbas R, Javed N, Nguyen A, Hanif H, et al. Acute Esophageal Necrosis: An In-depth Review of Pathogenesis, Diagnosis and Management. J Community Hosp Intern Med Perspect. 2022;12(1):96-103.
9. Gurvits GE, Cherian K, Shami MN, Korabathina R, El-Nader EM, Rayapudi K, et al. Black esophagus: new insights and multicenter international experience in 2014. Dig Dis Sci. 2015 Feb;60(2):444-53.
10. Gurvits GE, Shapsis A, Lau N, Gualtieri N, Robilotti JG. Acute esophageal necrosis: a rare syndrome. J Gastroenterol. 2007 Jan;42(1):29-38.
11. Endo T, Sakamoto J, Sato K, Takimoto M, Shimaya K, Mikami T, et al. Acute esophageal necrosis caused by alcohol abuse. World J Gastroenterol. 2005 Sep 21;11(35):5568-70.
12. Silvers J, Dixit S, Tan K, Masia R, Pratt A, Bulauitan C, et al. A novel presentation of alcohol-induced gastric necrosis: a case report. J Surg Case Rep. 2022 Dec;2022(12):rjac601.
13. Capurso G, Lahner E. The interaction between smoking, alcohol and the gut microbiome. Best Pract Res Clin Gastroenterol. 2017 Oct;31(5):579-88.
14. Rocco A, Compare D, Angrisani D, Sanduzzi Zamparelli M, Nardone G. Alcoholic disease: liver and beyond. World J Gastroenterol. 2014 Oct 28;20(40):14652-9.
15. Gurvits GE. Black esophagus: acute esophageal necrosis syndrome. World J Gastroenterol. 2010 Jul 14;16(26):3219-25.
16. Talebi-Bakhshayesh M, Samiee-Rad F, Zohrenia H, Zargar A. Acute Esophageal Necrosis: A Case of Black Esophagus with DKA. Arch Iran Med. 2015 Jun;18(6):384-5.
17. Uhlenhopp DJ, Pagnotta G, Sunkara T. Acute esophageal necrosis: A rare case of upper gastrointestinal bleeding from diabetic ketoacidosis. Clin Pract. 2020 May 19;10(2):1254.