



Journal Homepage: - www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/20671

DOI URL: <http://dx.doi.org/10.21474/IJAR01/20671>



RESEARCH ARTICLE

ACUTE ESOPHAGEAL NECROSIS : A RARE CONDITION CASE REPORT

M. Elkhayari, A. Hamdaoui, A. Lamine, H. Abid, N. Lahmidani, A.El Mekkaoui, M.El Yousfi, M. EL Abkari, S.A. Ibrahimi and D. Benajah

Gastroenterology Department, Hassan II University Hospital, Fez Sidi Mohamed Benabdellah University, Faculty of Medicine and Pharmacy of Fez, Morocco.

Manuscript Info

Manuscript History

Received: 25 January 2025

Final Accepted: 28 February 2025

Published: March 2025

Key words:-

Acute Esophageal Necrosis, Low-Flow State, Esophagogastroduodenoscopy

Abstract

Background: Acute esophageal necrosis (AEN) is a rare life threatening condition. Diagnosis is based on esophagogastroduodenoscopy (EGD) and shows a striking black esophagus appearance with an abrupt interruption at the gastroesophageal junction. Prognosis depends on the severity of the disease and on the underlying comorbidities.

Presentation of case: We report a case of a 71 years old female with history of diabetes mellitus and cardiac arrhythmia. She has been admitted in emergency for a fracture of the upper end of the femur that has been surgically managed. The procedure was marked by a low-flow state secondary to an important bleeding during surgery. Two days later, she presented with hematemesis. Upper gastrointestinal endoscopy showed a black esophagus. Management was based on total parenteral nutrition, intravenous proton pump inhibitor and fluid resuscitation.

"© 2025 by the Author(s). Published by IJAR under CC BY 4.0. Unrestricted use allowed with credit to the author."

Introduction:-

Case Report:

We report a case of a 71 years old female with a history of diabetes mellitus under insulin and atrial fibrillation on cardiac ischemia under digoxin, acenocoumarol, propranolol and amlodipine. Anamnesis has excluded other causes of esophageal injury (caustic ingestion, radiotherapy, trauma). She presented to the emergency department of Hassan II University Hospital for a fracture of the upper end of the femur. The patient benefited of a total hip prosthesis replacement. The procedure was marked by a low-flow state secondary to important bleeding that has been successfully managed by fluid resuscitation and surgical hemostasis.

Two days later, the patient presented an episode of upper gastro-intestinal bleeding. Examination found an afebrile patient with irregular accelerated heart rate (110 pulse by min), a correct arterial tension.

Laboratory investigations revealed anemia (hemoglobin: 6.5 g/dL compared to 11.5 g/dL prior to surgery), neutrophilic leukocytosis (white blood cell count: 22.370/mm³), C reactive protein (CRP): 263 mg/L, platelet count : 260.10³/mm³, prothrombin time 86% and renal insufficiency (serum creatinine level: 2 mg/dL).

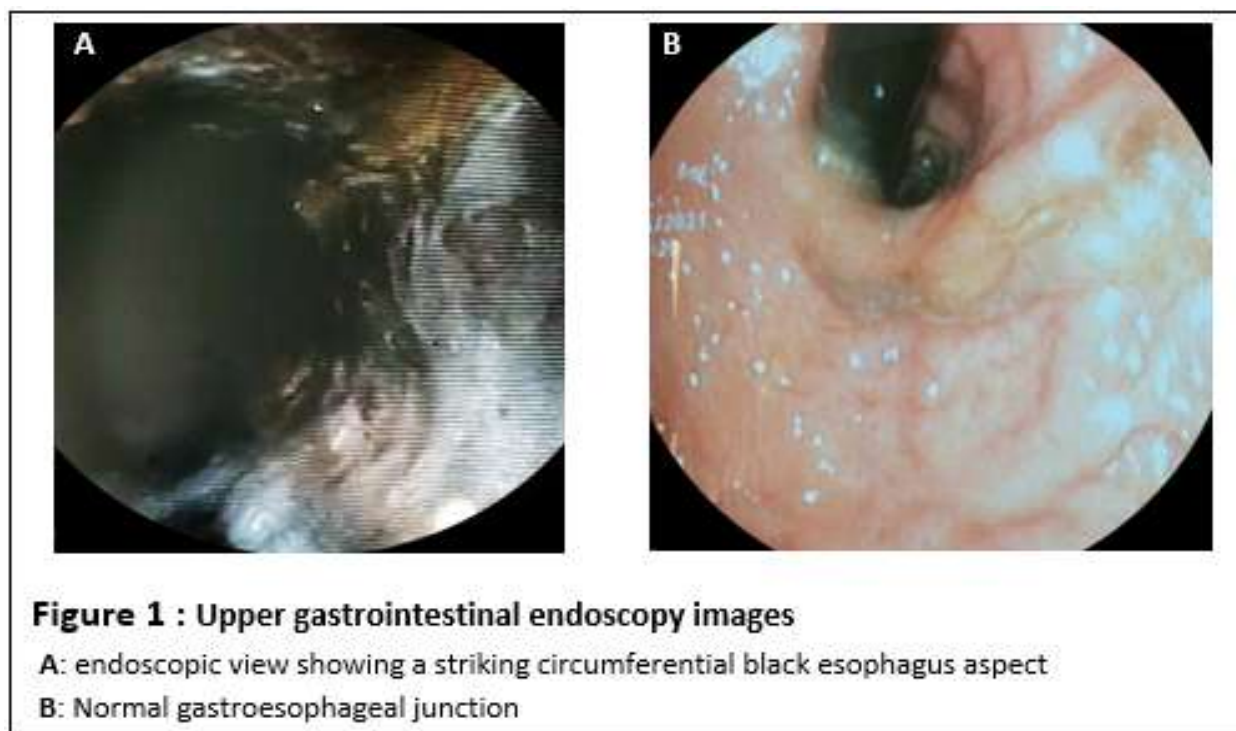
Corresponding Author:- M. Elkhayari

Address:- Gastroenterology Department, Hassan II University Hospital, Fez Sidi Mohamed Benabdellah University, Faculty of Medicine and Pharmacy of Fez, Morocco.

EGD showed ulcerations of the proximal esophagus and a striking black mucosa discoloration of the distal two thirds with an abrupt interruption at the GEJ, the stomach and duodenal were normal (**Figure 1**). Biopsies weren't taken because of the poor tolerance of the unsedated EGD. With the high endoscopic suspicion of esophagus necrosis, a computed tomography (CT) angiography was performed and showed a thickening of the lower third of the esophagus with no apparent signs of perforation.

Management consisted on total parenteral nutrition, acid suppression with intravenous proton pump inhibitor (PPI), antibiotics and fluid resuscitation including red cell concentrates transfusion. We also prohibited the use of a nasogastric tube.

Evolution was marked by a clinical worsening of the patient state. She installed a septic shock within the next 24 hours and has been admitted to the intensive care unit. Unfortunately, despite resuscitation measures, she died the next day.



Discussion:-

Acute esophageal necrosis (AEN) is a rare condition characterized by a striking black-appearing esophageal mucosa more marked in the lower third and abruptly stopping at GEJ.[1]

Men-women sex-ratio is 4:1. It has an estimated incidence of 0.01-0.28% of patients undergoing EGD and ranges from 0 to 0.2% in autopsy series[2]. Incidence increases with age and peaks in the seventh decade of life.[3]

AEN physiopathology has complex intricate mechanisms. It is multifactorial and typically occurs in a background of debilitation that put the patient at a greater risk of developing an AEN in association with an acute triggering event.

The risk factors correspond to chronic medical comorbidities responsible of an impaired esophageal mucosal repair and increase patient susceptibility to esophageal ischemia. They include, among others, congestive heart failure, diabetes mellitus, hypertension, liver cirrhosis, chronic kidney disease and gastroesophageal reflux. The above-mentioned risk factors, when coupled to a triggering event such as acute blood loss[4], diabetic ketoacidosis, septicemia[3], surgery[5], chemotherapy and others, can ultimately lead to AEN.

However, it can develop in young healthy patients triggered by some events such as binge drinking.[6]

In the case of our patient, multiple risk factors were present : diabetes mellitus, hypertension, atrial fibrillation on ischemic cardiopathy and the major triggering event was probably the low-flow state secondary to blood loss during surgery.

It's classic presentation is that of an upper gastrointestinal bleeding (hematemesis, associated or isolated melena) in a debilitated elderly male. Other symptoms can be associated such as abdominal pain, nausea, vomiting, dysphagia, fever, and syncope. Physical examination may show an abdominal tenderness, fever, signs of hemodynamic instability such as tachycardia and hypotension but none of these are specific of this condition.[7]

Laboratory findings may include anemia due to GI bleeding, leukocytosis (reflecting the inflammatory process within the esophagus mucosa). Other findings are also possible due to the patient comorbidities and complications.[1]

The cornerstone of diagnosis is EGD with the typical striking circumferential black aspect of the distal third esophageal with abrupt interruption at the GEJ[8]. In a recent systemic review of the literature by Abdullah and al, 50 % of the patients had a distal disease only with the involvement of the distal one-third of the esophagus. 34% had a pan esophageal involvement while only 2% had a proximal disease. Besides 5 % of the patients had concomitant ulcerations of the healthy esophagus[7]. The same lesions were described in our patient.

Due to the risk of perforation, biopsies are not mandatory. However, histology can be helpful in supporting the diagnosis and excluding associated infectious conditions (candidiasis, herpes simplex virus, cytomegalovirus).[9]

The role of angiography has not been clearly defined yet. Still, computed tomography can be used for complications detection.[10]

Possible complications of AEN are perforation, strictures and peristalsis abnormalities.[1]

Singh and al. published a five case series where a single patient had an abnormal dilated esophagus with pooled secretion. [11]

Moretó described a series of 10 cases where two esophageal stenosis developed and, in one case, a full-thickness necrosis made surgery with colon interposition necessary.[8]

Management of this condition relies on three cornerstones: 1) treating the coexisting comorbidities 2) resuscitation measures using, among others, fluid resuscitation, packed red blood cell transfusion, vasoactive drugs in shocked patients, antibiotics and 2) therapies aiming to treat the AEN : keeping the patient nil-per-os, prohibit nasogastric tube placement, parenteral nutrition, acid suppression using PPI and surgery in case of perforation.[10]

Outcome depends on the underlying comorbidities but also on the presence of factors that point to a worse prognosis : patients who present with shock, those who require packed red cell transfusion, antibiotics and surgery. Also the pan esophageal extension of the necrosis has been found to be predictive of higher mortality rates[7]. AEN has been associated with a high mortality rate of approximately 32%.[1]

To sum up, an AEN is a rare condition that generally occurs on a debilitated state, diagnosis is based on EGD after a GI bleeding episode. Management is based on supportive care, acid suppression therapy and nutritive support when needed. Prognosis is variable and gripped with a mortality varying from 35% to 50% depending on the underlying comorbidities but also on the severity of the esophageal lesions and the presence of complications specially perforation. Increased awareness of this pathology might lead to early recognition and timely institution of proper management, thereby increasing chances of survival.

References:-

[1] E. Dias, 'Diagnosis and management of acute esophageal necrosis', aog, 2019, doi: 10.20524/aog.2019.0418.

- [2] A. Kerschen, G. Schmit, E. De Boosere, C. Palmiere, and J. Vanhaebost, 'Black esophagus as an autopsy discovery: a challenging interpretation', *Egypt J Forensic Sci*, vol. 10, no. 1, p. 4, Dec. 2020, doi: 10.1186/s41935-020-0177-8.
- [3] G. E. Gurvits, A. Shapsis, N. Lau, N. Gualtieri, and J. G. Robiloti, 'Acute esophageal necrosis: a rare syndrome', *J Gastroenterol*, vol. 42, no. 1, pp. 29–38, Jan. 2007, doi: 10.1007/s00535-006-1974-z.
- [4] M. H. Loghmari, W. B. Mansour, A. Guediche, W. Bouhlel, M. Gahbiche, and L. Safer, 'l'œsophage noir: à propos d'un cas Black esophagus: a case report'. *La tunisie médicale* - 2018 ; Vol 96 (02)
- [5] M. Ali, N. Khan, A. Yaseen, and A. Raees, 'Gurvits Syndrome: Black Esophagus in the Postoperative Setting', *Cureus*, Jan. 2022, doi: 10.7759/cureus.21240.
- [6] A. Siddiqi, F. S. Chaudhary, H. A. Naqvi, N. Saleh, R. Farooqi, and M. N. Yousaf, 'Black esophagus: a syndrome of acute esophageal necrosis associated with active alcohol drinking', *BMJ Open Gastroenterol*, vol. 7, no. 1, p. e000466, Aug. 2020, doi: 10.1136/bmjgast-2020-000466.
- [7] H. M. Abdullah, W. Ullah, M. Abdallah, U. Khan, A. Hurairah, and M. Atiq, 'Clinical presentations, management, and outcomes of acute esophageal necrosis: a systemic review', *Expert Review of Gastroenterology & Hepatology*, vol. 13, no. 5, pp. 507–514, May 2019, doi: 10.1080/17474124.2019.1601555.
- [8] M. Moretó, E. Ojembarrena, M. Zaballa, J. G. Tánago, and S. Ibáñez, 'Idiopathic acute esophageal necrosis: not necessarily a terminal event', *Endoscopy*, vol. 25, no. 8, pp. 534–538, Oct. 1993, doi: 10.1055/s-2007-1009121.
- [9] G. E. Gurvits et al., 'Black Esophagus: New Insights and Multicenter International Experience in 2014', *Dig Dis Sci*, vol. 60, no. 2, pp. 444–453, Feb. 2015, doi: 10.1007/s10620-014-3382-1.
- [10] G. E. Gurvits, 'Black esophagus: Acute esophageal necrosis syndrome', *WJG*, vol. 16, no. 26, p. 3219, 2010, doi: 10.3748/wjg.v16.i26.3219.
- [11] D. Singh, R. Singh, and A. S. Laya, 'Acute esophageal necrosis: a case series of five patients presenting with "Black esophagus"', *Indian J Gastroenterol*, vol. 30, no. 1, pp. 41–45, Feb. 2011, doi: 10.1007/s12664-011-0082-z.