Black esophagus secondary to upper gastrointestinal bleeding. A case report

Emilio Vázquez Santiago M.D.
Brayant López García Montero M.D.
María Julieta Méndez Vargas M.D.
Sara Vianey González Mejía M.D.
José Miguel Palma López M.D.
Luis Daniel Rueda Luna M.D.
Valeria Monserrat Esquivel Tovar M.D.
Cinthya Gabriela Cruz Rapozo M.D.

Background: Black esophagus is a rare pathology characterized endoscopically by a black esophageal mucosa. Predisposing and triggering factors are involved in the proposed pathophysiology of this disease. The most frequent etiology is gastrointestinal tract bleeding. Clinically, patients present with data such as hematemesis, melena, etc. Diagnosis is made by direct visualization by endoscopy. Treatment depends on the underlying cause. This case report describes a male patient with clinical symptoms of upper gastrointestinal bleeding due to duodenal ulcer who developed black esophagus, which deserved in-hospital treatment and evolved favorably after medical treatment.

Keywords: Acute esophageal necrosis, black esophagus, gastrointestinal bleeding, hypovolemic shock, duodenal ulcer.

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Case Report

General Surgery

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Acute esophageal necrosis (AEN) also known as "*black esophagus*" It is a rare entity, characterized endoscopically by a diffuse, black-appearing esophageal mucosa, predominantly affecting the distal portion and often a rare complication of gastrointestinal bleeding.

With an incidence ranging from 0.01% to 0.028%, it predominantly affects men with a 4:1 ratio, typically presenting between ages 60 to 70, although it can develop at any age.[1] It is associated with different risk factors, among which the following stand out: diabetes mellitus, systemic arterial hypertension, gastroesophageal reflux disease, chronic kidney disease, malignant neoplasms (predominantly of the digestive tract), dyslipidemias, chronic malnutrition, alcoholism, liver diseases, psychiatric diseases (major depressive disorder and schizophrenia), as well as immunosuppression states.[1,2]

Although the pathophysiology remains undefined, a multifactorial or "two-strike" theory has been proposed, involving predisposing conditions and triggering events.[3] The aforementioned medical conditions increase the likelihood of developing acute esophageal necrosis (AEN) compared to the rest of the general population, which is explained by a greater predisposition to esophageal ischemia as a consequence of microvascular occlusion, chronic immunosuppression that conditions a decrease in the remodeling of the mucosal wall, epithelial damage caused by excessive reflux of gastric chemical secretions and finally a proinflammatory, thrombotic state and ischemic per se, this being the main pathogenic event.[4,5,6]

The main triggering factor is a decrease in esophageal perfusion due to hemodynamic compromise, mostly secondary to digestive tract bleeding. Other attributable causes were diabetic ketoacidosis, trauma, atrial fibrillation, gastric obstruction, and major surgery.[3,7]

Finally, it has been shown that hypovolemia and metabolic acidosis trigger hypercalcemia, which causes elevations of gastrin and acetylcholine, leading to an increase in gastric acid secretion and thus a greater risk of developing peptic ulcers, therefore becoming a vicious circle that supports the mechanism proposed above.[8,9]

Upper gastrointestinal bleeding is the most common clinical presentation in black esophagus, characterized by hematemesis, coffee-ground vomiting and melena.[10,11,12] Other symptoms include abdominal pain predominantly in the epigastrium, retrolesternal chest discomfort, nausea, vomiting, dysphagia and fever.[3] The most frequently associated clinical findings are cachexia, hypotension, tachycardia and diaphoresis. Classically, laboratories report normocytic normochromic anemia, leukocytosis (secondary to the inflammatory response given by esophageal necrosis) with predominance of neutrophils, hyperlactatemia (secondary to tissue hypoperfusion), increased acute phase reactants and hyperglycemia.[3,13]

To establish the diagnosis, it is essential to perform an endoscopy, which is characterized by a diffuse esophageal mucosa of black appearance, severe ulcerations with whitish exudates and necrotic changes that cover the entire circumference of the

From the General Surgery Department at ISSSTE Hospital Regional Tipo B de Alta Especialidad Bicentenario de la Independencia, Estado de México, México. Received on September 17, 2024. Accepted on September 24, 2024. Published on September 25, 2024

STAGE	ENDOSCOPIC FINDINGS	HISTOLOGICAL FINDINGS	FIRST OBSERVATION	COMPLICATIONS
0	**Normal-appearing pre-necrotic viable esophagus.**	Viable mucosa and submucosa, intact epithelium.		
1	**Black esophagus.** Circumferentially black friable esophageal mucosa that abruptly ends at the GEJ.	Mucosa and submucosa with presence of necrosis, absence of viable epithelium.	Initially.	Perforation.
2	**Checkerboard esophagus.** Residual black spots accompanied by whitish exudates composed of necrotic remains covering friable pink.	Pink mucosa with elements of necrosis or necrotic debris with inflammation over the granulation tissue.	1 to 4 weeks after diagnosis.	Stenosis.
3	**Esophagus re-epithelialized.** Esophageal mucosa regains its normal pink appearance.	Normal mucosa or mild granulation tissue.	1 to 2 weeks after diagnosis.	

Table 1. Modified endoscopic classification of black esophagus.

esophagus. This pattern ends clearly and abruptly at the squamoso-junction. cylindrical.[8, 14] AEN affects in most cases the distal esophagus, since it is a so-called "watershed" area, i. e. the vessels that emerge from the splenic artery towards its mucosa tend to lack coarse collateral branches that can supply the ischemic phenomenon, thus making this portion of the esophagus more susceptible to hypoperfusion and ischemic insult.[15]

However, there are exceptional cases where esophageal involvement is involved along its entire tract.[2] Additionally, as other endoscopic findings, esophageal, gastric, duodenal ulcerations and "coffee-ground" appearance material can be found.[13,16]

An endoscopic staging (Table 1) of the disease has been proposed, classifying it into: Stage 0 (prenecrotic esophagus), Stage 1 (black esophagus), Stage 2 ("checkerboard" esophagus), and Stage 3 (reepithelialized esophagus). [11,17]

Biopsy is not necessary to establish a diagnosis, but it is useful in ruling out other causes that appear as black esophageal mucosa such as: malignant melanoma, melanosis, pseudomelanosis, acanthosis nigricans, coal dust exposure. At present there is no well-established therapy in AEN, however the priority is based on the correction of the triggering events. Therefore, measures can be adopted such as intravenous fluid therapy that promotes vascular perfusion, and progressively decreases ischemic damage, proton pump inhibitors and gastric mucosal protectors as a mechanism of protection of the esophageal epithelium, accompanied by fasting to promote esophageal re-epithelialization, complemented by parenteral nutrition.[13]

It is important to mention that management is individualized for each patient, depending on the causal event and complications that are observed in up to 5% of all cases. An example of them is the transfusion of globular packets in case of active bleeding or hypovolemic shock; antibiotic management if a superimposed infection is suspected, as well as endoscopic balloon dilation in case of stenosis. Surgical treatment such as distal esophagectomy is reserved only in case of esophageal perforation (the most lethal complication).[2,18] Other complications described in the literature are: mediastinitis, mediastinal abscesses, gastroesophageal fistula, and multiple organ dysfunction.[13]

Black esophagus has a recovery period of 7 to 14 days, lasting up to a month. It is expected to visualize residual black areas and thick white exudates covering the partially regenerated mucosa.

This condition has a variable prognosis since it does not directly depend on the black esophagus, but rather is related to the patient's age, the causal factor and the underlying medical comorbidities, reaching a mortality of 30 to 50%.3,9 Various authors report that The earlier the diagnosis, the better clinical results the patient will have, thus reducing a lethal outcome.[3,19]

Case report

A 70-year-old male patient with a history of systemic arterial hypertension under treatment, sigmoid colon adenocarcinoma in situ requiring extended left hemicolectomy, gastroesophageal reflux disease (GERD) and untreated irritable bowel syndrome and generalized anxiety disorder (GAD). He refuses transfusions., drug addictions, alcoholism and smoking. Complete vaccination schedule for SARS-Cov-2 (3 doses).

The condition began 48 hours ago with generalized burning abdominal pain, predominantly in the epigastrium, VAS scale 9/10 without periods of remission or exacerbation. Later, he vomited coffee grounds on 5 occasions, which is why he was admitted to the Emergency Department.

Upon admission, the following vital signs were obtained: A/T: 80/40 mmHg, HR: 118 bpm, RR: 22 rpm, Temperature: 36.5 °C, SaO 2: 87% which

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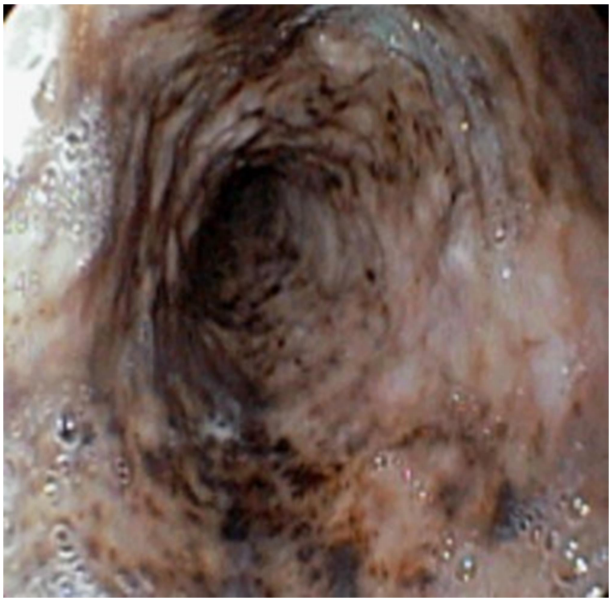

Figure 1. Distal esophageal mucosa. Typical "black esophagus" pattern.

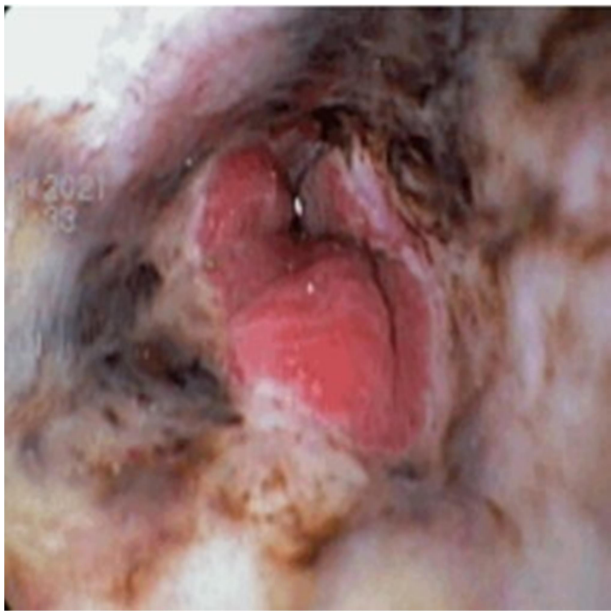

Figure 2. Esophagogastric junction with areas of black coloration alternating with whitish spots. Irregular Z line.

improved with supplemental O2 at 3 lt per minute to 91%.

On physical examination, the patient was awake, disoriented, uncooperative, Glasgow 11 at the expense of 3O, 3V, 5M, diaphoretic, generalized paleness of the integuments, sore fascia, oral cavity with blood remains, rhythmic heart sounds, increased in tone and intensity without added sounds, lung fields with good entry and exit of air, soft, depressible abdomen, painful on mid and deep palpation in the epigastrium and left hypochondrium without signs of peritoneal irritation, tympanic, normal peristalsis, negative rectal examination, intact extremities with capillary refill of 4 seconds, decreased peripheral pulses.

For this reason, reperfusion therapy with parenteral fluids, prokinetics, proton pump inhibitor and ethamsylate was indicated. A nasogastric tube was placed, draining 700 cc. Panendoscopy was performed where it is reported; esophagus with preserved shape and distensibility, from 10 centimeters from the upper dental arch pale, edematous mucosa is observed, with black color areas (figure 1), this pattern continues until the esophagogastric junction which coincides with diaphragmatic impingement, irregular "Z" line (figure 2). Duodenum: shape and compliance preserved, edematous and hyperemic mucosa, in the bulb an excavated lesion of approximately 10 mm in diameter is observed with irregular, raised and poorly defined edges with hematin and fibrin in the background. Second portion of duodenum with edematous mucosa, "salt and pepper" pattern (figure 3). Biopsies were not taken due to the high risk of bleeding and perforation from acute esophageal necrosis. The endoscopic diagnosis was: black esophagus, acute gastropathy, Forrest III duodenal ulcer and duodenitis.

Laboratory reports are: Leukocytes 16.6 x10^3/uL, Erythrocytes 4.52 x10/uL^6, Hemoglobin 8.5 g/dL, Hematocrit 26%, MCV 96.2 fL, MCH 29.7 pg, Platelets 168,000, Neutrophils 91%, Lymphocytes 7%, TP 15.2 sec, TTP 32 sec, INR 1.12 sec, Serum Creatinine 1.2 mg/dL, Glucose 141 mg/dL, Urea 56 mg/dL, BUN 26.01 mg/dL Ab anti SARS-COv 2 IgM and IgG negative

The patient was hospitalized for 8 days with the diagnoses of; black esophagus secondary to ATLS grade III hypovolemic shock due to upper gastrointestinal bleeding from a Forrest III duodenal ulcer. Medical treatment previously indicated was continued based on fasting that progressed to a liquid diet, parenteral solutions, proton pump inhibitor, prokinetic, antihypertensive and gastric mucosa protector. During his hospital stay, he had no evidence of rebleeding and remained hemodynamically stable, with a favorable clinical and biochemical evolution. For this reason, it was decided to discharge him from the Surgical service.

Subsequently, 14 days later a control endoscopy was performed, showed: Reactive gastropathy, nonspecific esophagitis, Forrest III duodenal ulcer, and Duodenitis. Thus, the remission of the black esophagus is evident.

Discussion

Black esophagus is a rare pathology, characterized endoscopically by a diffusely distributed black esophageal mucosa, which mainly affects its distal third, typically occurring in elderly male patients

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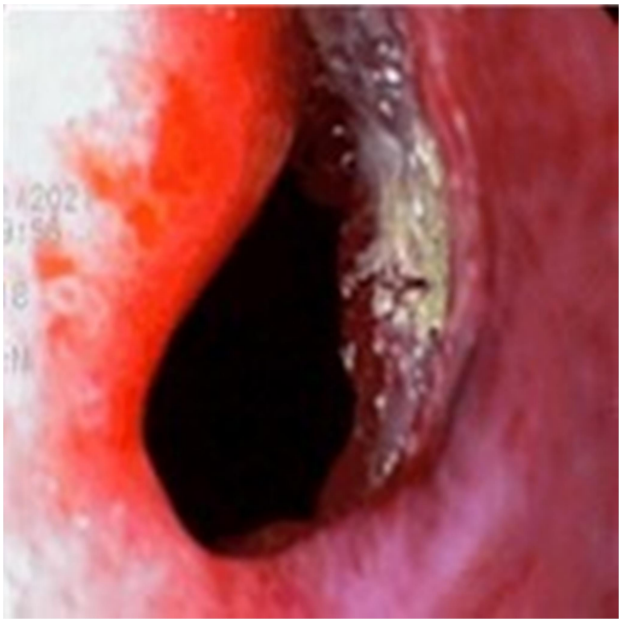

Figure 3. Duodenal bulb with presence of Forrest III ulcer.Distal esophageal mucosa in healing phase.

with multiple comorbidities, which predispose them to a higher risk of developing AEN compared to the rest of the population. However, a triggering event such as gastrointestinal tract bleeding that causes significant hemodynamic compromise in the patient is req for digestive perfusion, specifically the esophagus, to be compromised.

This case report describes a 70-year-old male patient with clear signs of gastrointestinal bleeding characterized by coffee-ground vomiting, along with epigastric pain, and signs suggesting hypovolemic shock, which was secondary to a duodenal ulcer, with the finding of black esophagus.

The patient had several risk factors predisposing him to acute esophageal necrosis AEN, however the triggering factor was the duodenal ulcer. This type of lesion tends to develop black esophagus more frequently unlike other etiologies, justified by the shared blood supply between the duodenum and the lower esophagus, which is provided by the branches of the celiac axis. Therefore greater attention should be paid to this association since it can be a strong predictor of AEN. Emphasizing the above, we suggest that when there are clinical data compatible with upper gastrointestinal bleeding and the endoscopic presence of black esophagus, an intentional search for duodenal ulcerous lesions should be performed.

Due to the few reported cases and the clinical research described to date, we do not have guidelines that establish an approach that can be developed in all patients, therefore we continue to support therapeutic praxis where the cornerstone of treatment is to correct the triggering events and control the predisposing factors, this in order to reduce the damage caused by microvascular occlusion (diabetes mellitus, systemic hypertension); gastric erosion (due to gastroesophageal reflux disease) and decreased mucosal remodeling (due to chronic states of immunosuppression such as malnutrition).

Although the duodenal ulcer causes considerable bleeding and affects the esophageal mucosa, the patient improved favorably with conservative treatment. Several authors such as *Eder and Ali et al.* have described this same satisfactory evolution in patients who had this same etiology in common and developed black esophagus.[20,21]

In this clinical case, the subject was a carrier of multiple risk factors that are present in a large part of the elderly population and that are closely related to AEN, however, not all patients with these comorbidities develop black esophagus regardless of any etiology. This is why it is important to continue investigating why only a limited group of patients have a greater predilection to develop this entity.

We believe that the incidence of this pathology is underestimated due to its variable natural history, explained by its broad esophageal repair mechanism. That is due to the different recovery period of the esophagus, this conditioned by the clinical context of each patient, since many times the black esophagus is overlooked, since in the event of bleeding of the digestive tract in patients with multiple comorbidities, an endoscopy is not performed early and for this reason this finding is not visualized.

Therefore, further research is needed to understand the pathophysiology, thereby establishing an early diagnosis and implementing an early therapeutic approach, with the aim of reducing complications and therefore mortality in a large percentage of these patients.

Conflicts of interests

The authors declare no conflicts of interest.

References

1. Schizas, D., Theochari, N. A., Mylonas, K. S., Kanavidis, P., Spartalis, E., Triantafyllou, S., et al. (2020). Acute esophageal necrosis: A systematic review and pooled analysis. World Journal of Gastrointestinal Surgery, 12(3), 104–115. https://doi.org/10.4240/wjgs.v12.i3.104
2. Abdullah, H. M., Ullah, W., Abdallah, M., Khan, U., Hurairah, A., & Atiq, M. (2019). Clinical presentations, management, and outcomes of acute esophageal necrosis: a systemic review. Expert Review of Gastroenterology and Hepatology, 13(5), 507–514. https://doi.org/10.1080/17474124.2019.1601555
3. Khan, H., Ahmed, M., Daoud, M., Philipose, J., Ahmed, S., & Deeb, L. (2019). Acute esophageal necrosis: A view in the dark. Case Reports in Gastroenterology, 13(1), 25–31. https://doi.org/10.1159/000496385

 DOI 10.5281/zenodo.13841296

4. Jessurun, J., Cui, I., & Aristi-Urista, G. (2019). Acute (gangrenous) esophageal necrosis (black esophagus). A rare form of injury with specific histologic features and diverse clinical associations with a common pathogenesis. Human Pathology, 87(2019), 44–50. https://doi.org/10.1016/j.humpath.2019.02.003
5. Siddiqi, A., Chaudhary, F. S., Naqvi, H. A., Saleh, N., Farooqi, R., & Yousaf, M. N. (2020). Black esophagus: A syndrome of acute esophageal necrosis associated with active alcohol drinking. BMJ Open Gastroenterology, 7(1). https://doi.org/10.1136/bmjgast-2020-000466
6. Gurvits, G. E. (2010). Black esophagus: Acute esophageal necrosis syndrome. World Journal of Gastroenterology, 16(26), 3219–3225. https://doi.org/10.3748/wjg.v16.i26.3219
7. Manno, V., Lentini, N., Chirico, A., Perticone, M., & Anastasio, L. (2017). Acute esophageal necrosis (black esophagus): a case report and literature review. Acta Diabetologica, 54(11), 1061–1063. https://doi.org/10.1007/s00592-017-1028-4
8. Okamoto, T., Suzuki, H., & Fukuda, K. (2021). Clinical and endoscopic characteristics of acute esophageal necrosis and severe reflux esophagitis. Medicine (United States), 100(44). https://doi.org/10.1097/MD.0000000000027672
9. Na, J. Y. (2021). Acute necrotizing esophagitis (black esophagus): An autopsy case with alcoholic ketoacidosis. Journal of Forensic and Legal Medicine, 78(May 2020), 102110. https://doi.org/10.1016/j.jflm.2020.102110
10. Gurvits, G. E., Cherian, K., Shami, M. N., Korabathina, R., El-Nader, E. M. A., Rayapudi, K., Gandolfo, F. J., Alshumrany, M., et al. (2015). Black Esophagus: New Insights and Multicenter International Experience in 2014. *Digestive Diseases and Sciences*, *60*(2), 444–453. https://doi.org/10.1007/s10620-014-3382-1
11. Gurvits, G. E., Shapsis, A., Lau, N., Gualtieri, N., & Robilotti, J. G. (2007). Acute esophageal necrosis: A rare syndrome. *Journal of Gastroenterology*, *42*(1), 29–38. https://doi.org/10.1007/s00535-006-1974-z
12. Inayat, F., Hurairah, A., & Virk, H. U. H. (2016). Acute esophageal necrosis: An update. *North American Journal of Medical Sciences*, *8*(7), 320–322. https://doi.org/10.4103/1947-2714.187159
13. Dias, E., Santos-Antunes, J., & Macedo, G. (2019). Diagnosis and management of acute esophageal necrosis. Annals of Gastroenterology, 32(6), 529–540. https://doi.org/10.20524/aog.2019.0418
14. Kim, S. M., Song, K. H., Kang, S. H., Moon, H. S., Sung, J. K., et al. (2019). Evaluation of prognostic factor and nature of acute esophageal necrosis: Restropective multicenter study. Medicine (United States), 98(41). https://doi.org/10.1097/MD.0000000000017511
15. Rehman, O., Jaferi, U., Padda, I., Khehra, N., Atwal, H., & Parmar, M. (2021). Epidemiology, Pathogenesis, and Clinical Manifestations of Acute Esophageal Necrosis in Adults. *Cureus*. https://doi.org/10.7759/cureus.16618
16. Grisham, E., Abu Khalaf, S., & Kuwajima, V. (2020). Acute Esophageal Necrosis in a Patient With Prostate Cancer Postchemotherapy. *ACG Case Reports Journal*, *7*(4), e00366. https://doi.org/10.14309/crj.0000000000000366
17. Bonaldi, M., Sala, C., Mariani, P., Fratus, G., & Novellino, L. (2017). Black esophagus: acute esophageal necrosis, clinical case and review of literature. *Journal of Surgical Case Reports*, *2017*(3). https://doi.org/10.1093/jscr/rjx037
18. Averbukh, L. D., Mavilia, M. G., & Gurvits, G. E. (2018). Acute Esophageal Necrosis: A Case Series. Cureus, 10(3), 3–7. https://doi.org/10.7759/cureus.2391
19. Martins, D., Marques, R., Costa, P., & Sousa, J. P. de. (2021). The dark side of the esophagus. Autopsy Case Reports, 11, e2021284. https://doi.org/10.4322/acr.2021.284
20. Eder, P., & Dobrowolska, A. (2021). Black esophagus: An unusual etiology of upper gastrointestinal bleeding. Polish Archives of Internal Medicine, 131(4), 377–378. https://doi.org/10.20452/pamw.15876
21. Ali, M., Khan, N., Yaseen, A., & Raees, A. (2022). Gurvits Syndrome: Black Esophagus in the Postoperative Setting. *Cureus*. https://doi.org/10.7759/cureus.21240

Emilio Vázquez Santiago
General Surgery Department
ISSSTE Hospital Regional Tipo B de Alta Especialidad
Bicentenario de la Independencia
Estado de México, México.

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