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Black esophagus: A devastating consequence - A case report and comprehensive literature analysis

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Abstract

Acute Esophageal Necrosis (AEN) is a rare condition characterized by circumferential black discoloration and necrosis of the esophageal mucosa. This report presents a case of a 79-year-old female with metastatic cancer diagnosed with black esophagus and literature reviews of 68 cases. Our literature search highlighted common presenting symptoms were hematemesis (55%), gastrointestinal symptoms (54%), and melena (19%). And comorbidities including, malignancy (~10%) being leading cause of AEN. Age, low hemoglobin, and hypotension are associated with increased mortality. Management typically involves supportive care, but in cases of advanced malignancy, treatment may focus on symptom control and palliation.

Keywords: Acute esophageal necrosis; Black Esophagus; Malignancy; Endoscopy.

Abbreviations: AEN: Acute Esophageal Necrosis; GERD: Gastroesophageal Reflux Disease; Hb: Hemoglobin; EGD: Esophagogastroduodenoscopy; ESRD: End-Stage Renal Disease; CKD: Chronic Kidney Disease; COPD: Chronic Obstructive Pulmonary Disease; HLD: Hyperlipidemia; MEN1: Multiple Endocrine Neoplasia Type 1.

Introduction

Acute esophageal necrosis, commonly known as "black esophagus," is a rare and potentially life-threatening condition characterized by the circumferential black discoloration and necrosis of the esophageal mucosa. This condition was first described by Goldenberg et al. in 1990, and since then, numerous case reports and small case series have been published in the literature [1]. The exact pathogenesis of the black esophagus remains unclear, but it is believed to result from a combination of factors leading to compromised blood supply to the esophagus, such as low-flow states, vasculopathy, and mucosal ischemia. Proposed risk factors and associated conditions include diabetes mellitus [2], alcohol abuse, malnutrition, Gastroesophageal Reflux Disease (GERD), vascular disease, hypoperfusion states (e.g., shock, cardiac disease), and certain medications or procedures that may impair esophageal perfusion [3,4]. The clinical presentation of black esophagus is often non-specific, with upper gastrointestinal bleeding (hematemesis, melena) being the most common symptom. Other associated symptoms may include epigastric pain [5], nausea, vomiting, dysphagia, and odynophagia. Diagnosis is typically made through endoscopic evaluation [6], revealing the characteristic circumferential black discoloration and necrosis of the esophageal mucosa, often involving the distal esophagus and abruptly stopping at the gastroesophageal junction [7]. Here, we present a case report of a black esophagus in a 79-year-old female with metastatic cancer.

Case report

A 79-year-old female with a past medical history of type 2 diabetes mellitus, hypertension, dementia, and hyperlipidemia, presented to the emergency department with worsening abdominal swelling, and dyspnea on exertion. Her initial vitals showed hypertension 172/92 mmHg, tachycardia 104 bpm, tachypnea 22 breaths/min, temperature 97.7°F, and oxygen saturation 98% on room air. Pertinent labs were Hemoglobin (Hb) 11.2 g/dL, platelets 373 x10^3/µL, AST 15 U/L, ALT 5 U/L, glucose 260 mg/dL, and creatinine 1.3 mg/dL. On exam, the lungs exhibited decreased breath sounds at the bases bilaterally. Imaging revealed peritoneal carcinomatosis with a large pelvic mass measuring 12.7 x 10.8 x 16.7 cm (Figure 1). Bilateral pleural effusions and ascites were also present. Paracentesis fluid analysis showed metastatic mucin-producing adenocarcinoma, tumor cells are positive for pan cytokeratin, CK20, and CDX2. The findings were suggestive of metastatic adenocarcinoma of colorectal origin. Oncology was consulted, but no treatment options were appropriate for the patient at this time as the prognosis was very poor. And recommended a palliative care consult. On hospital day 26, she developed coffee-ground emesis, her Hb dropped to 5.6 g/dL and was hypotensive to 82/64 mmHg, requiring 2 units of packed RBCs. On day 28, she underwent an Esophagogastroduodenoscopy (EGD) revealing circumferential black discoloration of the esophagus concerning for acute necrotic esophagus (Black esophagus) (Figure 2) with biopsy showing erosive esophagitis; she was treated with intravenous proton pump inhibitors, sucralfate, and hydration. Given the patient's overall poor prognosis in the setting of malignancy, no follow-up EGD was recommended. Her course required 4 paracenteses removing ~10L of fluid, and 1 thoracentesis for symptomatic management. As she was not a candidate for further cancer treatment, detailed goals of care discussions with the family were done. The patient was transitioned to hospice care; unfortunately, the patient passed away in the hospital before being discharged.

Results

The study involved 68 patients diagnosed with the black esophagus, with a mean age of 60.37 (16.28) years. The majority of the patients were males (67%), while females accounted for 32%. The most common presenting symptom was hematemesis (55%), followed by other gastrointestinal symptoms (54%, nausea, vomiting, diarrhea, dysphagia, dysphonia, odynophagia), abdominal pain (22%), and melena (19%). Some patients experienced loss of consciousness (7%) or altered mental status (6%). Significant comorbidities included diabetes mellitus (42%), alcohol use disorder (25%), drug addiction (16%), and gastrointestinal conditions such as GERD, cirrhosis, and hepatitis (25%). Hypertension was present in 20% of the patients, while renal comorbidities (19%, including end-stage renal disease, chronic kidney disease, and renal transplant), cardiac conditions (12%), and lung disease (4% with COPD) were also reported. Malignancy was observed in 10% of the patients, and other comorbidities, such as hyperlipidemia and multiple endocrine neoplasia type 1, were present in 4% of the cases (as Table 1). Table 2 showing acute esophageal necrosis demographic and clinical data review. As per our literature review, Figure 3 shows the prognosis of black esophagus cases over the past 2 decades where the number of cases recorded in the literature has increased, likely from more awareness, early diagnosis, and resources like EGD availability across the health care system. Moreover, there is a clear increase in survival rate in patients diagnosed with black esophagus (Represented by the green line) with a downtrend of deceased patients (Represented by the red line).

Table 1: Demographics of all included patients with AEN.

Variables	N=68
Age	60.37 (16.28)
Gender (%)	
Male	67
Female	32
Presenting symptoms (n)	
Hematemesis	38
Melena	13
Abdominal pain	15
Other GI symptoms*	37
Loss of consciousness	5
Altered mental status	4
Comorbidities (n)	
Hypertension	14
Diabetes Mellitus	29
Drug addiction	11
Alcohol use disorder	17
GI (GERD, cirrhosis, hepatitis)	17
Cardiac	8
Renal (ESRD, CKD, Renal transplant)	13
Lung (COPD)	3
Malignancy	7
Others (HLD and MEN1)	3

*Nausea/Vomiting/Diarrhea/dysphagia/dysphonia/odynophagia
GERD: Gastroesophageal Reflux Disorder; ESRD: End-Stage Renal Disease; CKD: Chronic Kidney Disease; COPD: Chronic Obstructive Pulmonary Disease; HLD: Hyperlipidemia; MEN1: Multiple Endocrine Neoplasia type 1.

Discussion

The findings from our literature review are consistent with the existing knowledge of black esophagus. The mean age of the patients in our study (60.37 years) aligns with the reported predominance of this condition in older adults, likely due to the presence of comorbidities and compromised vascular supply.[8] The male predominance (67%) observed in our study is also consistent with previous reports, although the underlying reason for this gender difference remains unclear [9]. The presenting symptoms and comorbidities identified in our study are in line with the established literature. Hematemesis (55%) and other gastrointestinal symptoms (54%) were the most common presenting complaints, reflecting the underlying mucosal injury and bleeding associated with black esophagus. [10] Comorbidities such as diabetes mellitus (42%), alcohol use disorder (25%), and gastrointestinal conditions (25%) are well-recognized risk factors for the development of black esophagus, as they can contribute to vascular compromise, mucosal

Table 2: Summary of literature review.

Sr. No	Author	EGD findings	Biopsy	Cause	Treatment	Prognosis
1	Porcu et al [18]	N/A	N/A	Thoracic endoluminal aortic repair	Cholecystostomy, Tazocilline, amikacin	Deceased (non-BE related)
2	Akkinepally et al [19]	Lower half of esophagus showed signs of necrosis (exudates and distal blackening), DU	N/A	Alcoholic cirrhosis	IVF, PPI, NPO	Deceased (non-BE related)
3	Lee et al [20]	Distal half blackening of the esophagus	Reactive glandular cells with acute inflammation and necrosis.	Alcohol-induced chemical esophagitis with Achalasia and hypotensive episodes	PPI, broadspectrum abx, IVF	Survived
4	McLaughlin et al [21]	Ischemic changes starting 25 cm from the incisors and ending abruptly at the gastroesophageal junction, shortened esophagus	N/A	N/A	IVF, NG tube, broad-spectrum abx	Survived
5	Carrillo et al [22]	Typical blackish esophageal lesions with exudates	N/A	Several hypotension episodes	N/A	N/A
6	Garas et al [23]	N/A	Features of ischaemia and associated haemorrhagic necrosis	N/A	PPI, antiemetics, NPO	Deceased
7	Altenburger et al [24]	Esophagus was black with ischemic necrosis of the mucosa, submucosa, and muscularis	Diffuse acute inflammatory infiltrate, brown pigmentation limited to the mucosa	N/A	N/A	Deceased
8	Singh D et al [25]	Blackened esophagus, necrotic appearing pale esophageal mucosa, and normal GE junction	Necrotic debris, absence of epithelium, granulation tissue, and heavy leukocytic infiltrates	DKA	NG tube, PPI, broad-spectrum abx, antifungal agent	Survived
9	Singh D et al [25]	Necrotic appearing friable areas of spontaneous bleeding	N/A	N/A	NPO, PPI,100 units of botulinim toxin injected at LES	Survived
10	Singh D et al [25]	Dusky appearance in distal 10 cm of esophagus with areas of superficial ulceration suggestive of ischemia	N/A	N/A	NPO, PPI, TPN	Deceased
11	Singh D et al [25]	Diffuse blackened esophageal mucosa, friable mucosa	N/A	N/A	PPI, broad spectrum abx	Deceased
12	Singh D et al [25]	Middle to distal esophagus appeared necrotic	N/A	DKA	NPO, PPI	Survived
13	Lahbabi et al [26]	Lower third mucosa was black and covered by an exudate of the same color associated with diffuse bleeding	Necrotic debris, mucosal submucosal necrosis with a local inflammatory response	N/A	PPI, TPN	Deceased
14	Pereira et al [27]	Extensive candidiasis and a black-appearing oesophageal mucosa compatible with necrosis at the distal 2/3 of the oesophagus	N/A	Esophageal candidiasis, hypotension episode, alcohol abuse	PPI, TPN, NPO, fluconazole	Survived
15	Singh S et al [28]	Extensive ulceration, sloughing and multiple areas of necrosis in the distal oesophagus and stomach	N/A	Dabigatran adverse effect	PPI, broad spectrum abx, NG tube	Survived
16	Kwon et al [29]	Black macerated mucosa in the mid third of the esophagus and circumferential mucosal necrosis with a huge adherent blood clot in the distal third of the esophagus	N/A	Coronary angiography complicated with hypotension	PPI	Survived
17	Shimamura et al [30]	Thick black stripes involving the distal esophagus, with a sharp demarcation at the squamocolumnar border	Leukocyte infiltration with hemosiderosis.	DKA	IVF, PPI, NPO	Survived

18	Lu et al [31]	N/A	N/A	Foreign body ingestion	Exploratory laparotomy (removed the fish bone)	Deceased
19	Zaid et al [32]	Circumferential black pigmentation, fragile esophageal necrotic mucosa, and multiple superficial ulcerations in the middle and lower thirds of esophagus.The lower one-third of esophagus was covered with dark exudates, oozing fresh red-colored blood with minimal touch	Focal area of ulceration. The ulcerated area was replaced by local inflammatory changes comprising necrotic debris, neutrophil exudates, hemorrhage, and background fibrinous material deposition.	N/A	IVF, PPI, NPO, analgesia, TPN	Survived
20	koksal et al [33]	Circumferential, black colored, necrotic distal esophageal mucosa abruptly turning to normal at the gastroesophageal junction and a giant necrotic based ulcer covering the anterior aspect of the first portion of the duodenum.	N/A	DM	IVF, PPI, NPO, sulbactam-ampicillin	N/A
21	Galtes et al [34]	N/A	Severe necrosis of the mucosa and submucosa with absence of viable squamous epithelium and an abundance of necrotic debris. Marked acute inflammatory infiltrates and partial destruction of muscle fibres	DKA	N/A	Deceased
22	Kim S et al [35]	Necrotic tissue on lower esophagus, necrotic tissue from bulb to distal area of duodenum	N/A	Likely from COVID-19 infection	NG tube, IVF, PPI, NPO, broad spectrum abx	Survived
23	Pineo et al [36]	N/A	N/A	Cocaine abuse	PPI, IV opioids, NG tube, G-CSF, broad-spectrum abx, exploratory laparotomy	Deceased
24	Shafa et al [4]	Black necrotic mucosa circumferentially throughout the entire esophagus.	N/A	Septic shock	Supportive care	Deceased
25	Shafa et al [4]	Diffuse esophageal necrosis as well as a small segment of denuded mucosa that was actively oozing blood	N/A	Alcohol abuse or heart failure	Bleeding esophageal segment was injected with epinephrine, PPI, NPO, TPN	Survived
26	Shafa et al [4]	Acute esophageal necrosis from the cricopharyngeus to the gastroesophageal junction	Inflammatory exudate and necrotic debris	N/A	IV PPI, NPO	Survived
27	Shafa et al [4]	Circumferential necrosis throughout the esophagus and a mid-esophageal stricture 5 cm in length.	N/A	Alcohol, cocaine, cannabis abuse, DKA	Surgically placed gastrostomy tube and supportive therapy	Survived
28	Shafa et al [4]	Pan-esophageal necrosis with circumferential involvement distally	N/A	N/A	Supportive care	Deceased
29	Shafa et al [4]	Necrosis in the distal third of his esophagus	N/A	Multiorgan dysfunction	N/A	Deceased
30	Alcaide et al [37]	Esophageal lumen diffusely dilated, with submucosal hemorrhages and confluent violet-blackish areas, hiatal hernia	Esophageal mucosa with micro-hemorrhages and microscopic foci of necrosis	N/a	IVF, PPI	survived
31	Nunes et al [38]	Black esophagus covered with dark fluid, hyperaemia and erosions.	N/A	Paraesophageal hernia and gastric volvulus	Emergency surgery repaired the diaphragmatic hernia, achieved volvulus reduction and a Nissen fundoplication	survived
32	Osterman et al [39]	Severe diffuse ulceration beginning in the mid esophagus, with continued severe ulceration throughout the distal esophagus, stomach and duodenum	N/A	Re-initiating clozapine and quetiapine	N/A	Deceased

33	Matsuo et al [40]	Black esophagus with ulcerated longitudinal necrosis in the lower esophagus	N/A	N/A	PPI, NPO	Survived
34	Crescenzi et al [41]	Diffuse, circumferential, blackappearing mucosa in the distal third of the esophagus	N/A	Septic shock	IVF, PPI, TPN and broad-spectrum abx	survived
35	Ullah et al [42]	Circumferential necrotic, friable oesophagus that extended from 21 cm to 40 cm from the incisors. There was associated friable red mucosa	N/A	Cocaine abuse	IVF, PPI, sucralfate suspension	Deceased
36	Sato et al [43]	Black discoloration from the esophagus to the gastric junction, esophageal perforation and black discoloration in the duodenem	N/A	N/A	Thoracic cavity drain, esophageal stent (FSEMS), SBT gastric balloon, IVF	Survived
37	Haghbayan et al [44]	Circumferential black mucosa in the distal esophagus, immediately proximal to the gastroesophageal junction	N/A	DKA	PPI, NPO, insulin, IVF	Survived
38	Kondo et al [45]	Diffuse black discoloration of the esophageal mucosa that affected the distal esophagus and stopped abruptly at the gastroesophageal junction	Necrosis of the esophageal mucosa	DKA	PPI, NPO, insulin, IVF	Survived
39	Ullah et al [46]	Circumferential necrotic, friable oesophagus extending from 21 to 38 cm from the incisors	N/A	Dialysis-induced hypotension	NG tube, IVF, PPI, IV sucralfate	Deceased
40	Tomori et al [47]	Necrotic esophagitis in the middle and lower parts of the esophagus and duodenal erosions	No findings suggestive of esophageal necrosis	Strongyloides stercoralis hyperinfection and dissemination	NG tube, ivermectin, broad spectrum abxkim s	Survived
41	Dias et al [48]	Friable, diffuse black-appearing distal esophageal mucosa with an abrupt transition at the gastroesophageal junction	N/A	Septic shock	Percutaneous cholecystostomy, IVF, PPI, NPO, broad-spectrum abx	Survived
42	Uyar et al [49]	Diffuse circumferential black appearance throughout the entire esophagus mucosa	Histological appearance of Candida hyphaes and necrosis in esophagus tissue	N/A	PPI, IVF, anti-fungal for candida in the bx	N/A
43	Tanaka S et al [50]	Diffuse erosive esophagitis with black discoloration predominantly affecting the lower esophagus and abruptly interrupted at the gastroesophageal junction	N/A	N/A	PPI, IVF, abx	N/A
44	Siddiqi A [51]	Inflammation of the epiglottis, arytenoid cartilages, and dark mucosal pigmentation of the distal two-thirds of the esophagus with associated hiatus hernia	Fragments of fibrinopurulent exudate, and necrotic tissue with predominant neutrophil infiltration indicating severe inflammation	N/A	PPI, IVF, abx	N/A
45	Deliwala Ss et al [52]	Black necrotic-appearing mucosa encompassing the entire esophagus and ending abruptly at the GEJ with associated gastric erosions and proximal duodenal ulcerations	Acute esophageal sterile gangrenous necrosis	N/A	PPI, IVF, abx	N/A
46	Bhattacharya et al [53]	Circumferential black oesophageal mucosa extending proximally from the gastro-oesophageal junction.	The duodenum contained microscopic foci of neuroendocrine tumour consistent with gastrinoma.	MEN related	PPI, IVF, abx	Survived
47	Kim NY et al [54]	Circumferential black pigmentation, and edematous mucosa covered by exudates was noted at the mid-lower esophagus	An ulcer, and immunohistochemistry showed negative cytomegalovirus (CMV), herpes simplex virus (HSV-1), and HSV-2 polymerase chain reaction findings.	Post renal transplantation	PPI, abx	Survived
48	Kroner et al [55]	Diffuse severe mucosal changes of the middle and lower third of the esophagus characterized by black-discoloration, erythema, and friable tissue with ulceration, sloughing and contact bleeding	Biopsies were consistent with acute esophageal necrosis, also known as “black esophagus”.	Post renal transplantation	PPI, and TPN	Survived

49	Riascos et al [56]	Extensive esophageal necrosis from the cricopharyngeal muscle to the esophageal-gastric junction transmurally at 30 cm, with a perforation of approximately 15 mm at 35 cm.	N/A	N/A	PPI, abx, fluconazole	Survived
50	Laverick et al [57]	A black discolouration of the distal oesophagus with deep black ulcers and diffuse oozing of blood.	N/A	N/A	PPI, abx	Survived
51	Mustafa et al [58]	Two large 1.5–2 cm wide-based ulcers in the distal oesophagus without active bleeding	An ulcerated squamous mucosa with extensive necrosis extending to the muscularis propria. Coccoid bacterial colonies and rare fungal forms suggestive of Candida species were seen in the necrotic areas.	N/A	PPI, fluconazole	Survived
52	Iwamoto et al [59]	Entire circumference of the whole esophagus was shown to have turned black	N/A	HHS	treated the HHS	Survived
53	Okamoto et al [60]	N/A	N/A	N/A		Survived
54	Okamoto et al [60]	N/A	N/A	N/A		Survived
55	Okamoto et al [60]	N/A	N/A	N/A		Survived
56	Okamoto et al [60]	N/A	N/A	N/A		Survived
57	Okamoto et al [60]	N/A	N/A	N/A		Survived
58	Okamoto et al [60]	N/A	N/A	N/A		Survived
59	Okamoto et al [60]	N/A	N/A	N/A		Survived
60	Okamoto et al [60]	N/A	N/A	N/A		Survived
61	Okamoto et al [60]	N/A	N/A	N/A		Survived
62	Kitawaki [61]	Circumferential necrosis of the middle and distal esophagus, immediately proximal to the gastroesophageal junction	N/A	N/A	PPI	
63	alsakarneh et al [62]	Severe inflammation with black discoloration consistent with acute esophageal necrosis in the middle and lower esophagus, and erythematous duodenitis	N/A	N/A	PPI, sucralfate, NPO	Survived
64	Jeican et al [63]	N/A	N/A	N/A	N/A	Deceased
65	Ifuku [64]	Circumferential mucosal injury and partial necrosis from the middle esophagus to the gastroe- sophageal junction with a sudden transition to normal mu- cosa at the distal portion	N/A	Cynotic spells from co-morb	PPI and NPO for 2 days	Survived
66	Greco S et al [65]	Circumferential black appearance of the esophageal mucosa, as in concentric necrosis of the distal esophagus with possible fungal superinfection.	Fungal hyphae by Candida spp.	N/A	IV fluconazole	Survived
67	Patil et al [66]	Diffuse ischemic and necrotic mucosa beginning approximately 20 cm from the teeth and extending distally, and a large distal esophageal perforation extending into the stomach.	Gross surgical specimen analysis and immunohistochemistry were consistent with diffuse necrosis.	N/A	Surgical esophagectomy and cervical esophagostomy	Survived

68	Gonzalez diaz et al [67]	Diffuse circumferential black mucosa covered by fibrin, affecting the middle third and distal esophagus. These changes progressively worsened from the proximal to distal esophagus, until reaching the eso- phagogastric junction	N/A	N/A	N/A	N/A

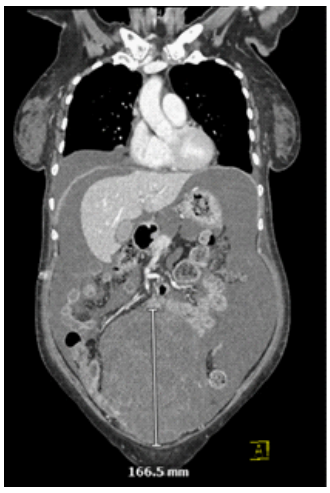

Figure 1: Computed tomography (CT) scan imaging showing peritoneal carcinomatosis with a large pelvic mass measuring 166.5 mm in size.

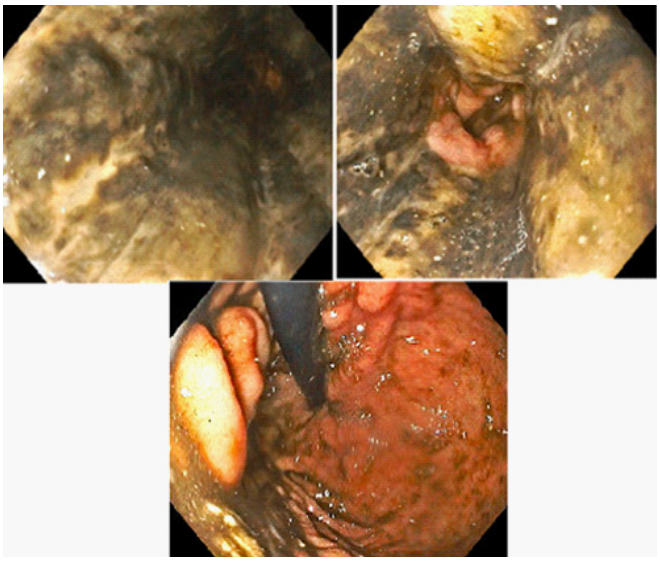

Figure 2: Esophagogastroduodenoscopy (EGD).
Endoscopic features of black esophagus showing diffuse esophageal necrosing in all the three images.

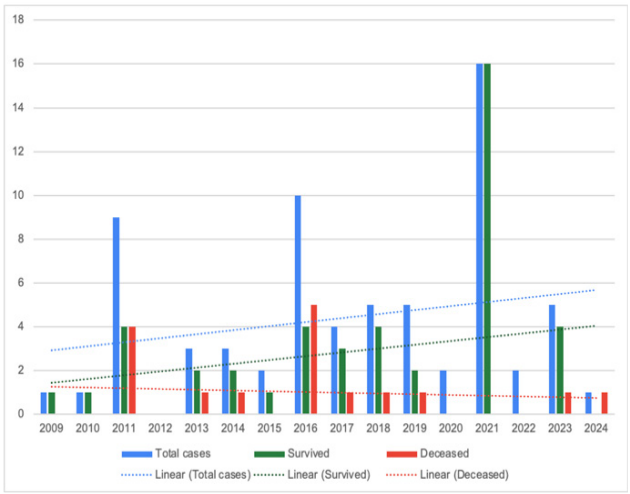

Figure 3: Prognosis of black esophagus cases from 2009 to 2024, per our literature review.
Demonstrates the prognosis of black esophagus cases over the past 2 decades where the number of cases recorded in the literature. Additionally, showing linear trends, blue line representing increase in total number of cases, red line indicating decrease in mortality and green line indicating improved survival.

ischemia, and esophageal injury [11,12]. In the current case report, the patient was an elderly female with multiple comorbidities, including diabetes mellitus, hypertension, and hyperlipidemia. While she did not have any known history of alcohol or drug abuse, her advanced metastatic cancer and overall poor prognosis likely contributed to a compromised vascular supply and increased susceptibility to esophageal necrosis. Similarly, a study by [13], providing a broader perspective on prognostic factors for mortality, showed that age, low hemoglobin, and hypotension were factors associated with increased mortality. Notably, my patient had all three of these risk factors, which increased her mortality risk. Interestingly, our analysis found a relatively high prevalence of malignancy (10%) among patients with black esophagus. While malignancy itself may not directly cause black esophagus, the associated treatments or complications (e.g., chemotherapy-induced mucositis, malnutrition, immunosuppression) could potentially predispose patients to this condition. This finding is particularly relevant in the context of our case report [14], where the patient's underlying malignancy and poor prognosis likely played a significant role in the development of black esophagus. It is noteworthy that Day et al. published a case report with a review of the literature in 2009, which included most of the cases reported until 2008. In our review, we excluded those studies and focused on more recent cases [15], incorporating literature from 2009 to 2023, making our case report one of the most up-to-date compilations of black esophagus cases. The management of black esophagus typically involves supportive care, including intravenous fluid resuscitation, proton pump inhibitors, antibiotics, and nutritional support. In our case report, the patient received appropriate management with intravenous proton pump inhibitors [16], sucralfate, and hydration. However, given the patient's overall poor prognosis due to advanced metastatic disease, no further interventions or follow-up endoscopy were pursued, aligning with the palliative care approach. It is important to recognize that black esophagus can be a manifestation of underlying systemic illness or compromised vascular supply, and prompt diagnosis and management are crucial to prevent potential complications, such as esophageal perforation, mediastinitis, or sepsis. In cases of advanced malignancy or poor prognosis [17], as in our case report, the management may be tailored toward symptom control and palliation, with careful consideration of the risks and benefits of invasive interventions.

Conclusion

Acute esophageal necrosis is an uncommon but severe con-

dition typically affecting older individuals with underlying medical conditions that compromise blood flow to the esophagus. Rapid diagnosis through endoscopy and prompt supportive treatment, such as intravenous fluids, proton pump inhibitors, and antibiotics, are essential. However, in cases where patients have advanced malignancy or a poor overall prognosis, the management approach may focus more on palliative care and symptom relief, carefully weighing the potential risks and benefits of invasive procedures.

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