CASE REPORT

Black Oesophagus

Olga Pereira, João Figueira-Coelho, Bárbara Picado, José Neves Costa

Centro Hospitalar Lisboa
Central, Hospital Curry Cabral,
Lisboa, Portugal

Correspondence to
Dr Olga Pereira,
olgaapp@gmail.com

SUMMARY

Acute oesophageal necrosis, also known as 'Black Oesophagus', is a rare endoscopic finding since its first description by Goldenberg in 1990. In endoscopic studies, the frequency ranged from 0.01% to 0.2%. The aetiology is undefined and is probably multifactorial. A 62-year-old woman, with chronic alcoholism, was admitted to the internal medicine department for dehydration and marked malnutrition problems. Melaena was detected, and oesophagogastroduodenoscopy showed black mucosa of the lower two-thirds of the oesophagus and candidiasis. The patient gradually recovered after conservative treatments (intravenous proton pump inhibitor and total parental nutrition) and fluconazole. Oesophagus stricture was developed after 1 month, and balloon dilatation was performed successfully.

BACKGROUND

I decided to write this article because the Black Oesophagus is a rare disease with few described cases. This article also emphasises the fact that more research is needed on the Black Oesophagus.

CASE PRESENTATION

A 62-year-old Caucasian woman with a precarious social situation came to our emergency department with dehydration and poor nutritional status. Her medical history was chronic alcohol abuse. She had no alcoholic liver cirrhosis diagnosed. At presentation, blood pressure was 84/50 mm Hg, with a heart rate in the range of 90 bpm and a regular respiratory rate. The patient was afebrile, dehydrated and very skinny. Consciousness was clear. The eyelid mucosa was anaemic. The abdomen was flat, painless on palpation and without hepatomegaly. There was no oedema.

Laboratory data revealed: 13 800 white blood cells/ μ l, haemoglobin 11 g/dl, median globular volume 102.3 fl, haematocrit 33% (38–47%), blood iron 21 μ g/dl (50–180), ferritin >1000 ng/ml (10–460), folic acid 2 ng/ml (3–20), transferrin <80 (10–300), total blood protein 4.5 g/dl (6.3–8.2) and albumin 1.93 g/dl (3.5–5). The coagulation factors were normal. Chemistries included: C reactive protein 4.5 mg/dl (normal <1), aspartate aminotransferase 212 U/l (14–36), alanine aminotransferase 45 U/l (9–52), gamma glutamyl transferase 789 U/l (12–43), lactic acid dehydrogenase 665 U/l (313–618), normal renal function, sodium 130 mEq/l.

The patient was hydrated with normal saline solution and nourished; the clinical evolution process was satisfactory.

However, 6 days after admission, melaena occurred. Blood analyses revealed a drop of haemoglobin and haematocrit (8.4/25). Emergency oesophago-gastro-duodenoscopy showed extensive candidiasis and a black-appearing oesophageal mucosa compatible with necrosis at the distal 2/3 of the oesophagus (figure 1). Biopsies of the oesophagus were not performed due to the risk of perforation and haemorrhage.

On the 21st day, a second endoscopy showed a remarkable change from pitch-black mucosa to red, friable mucosa with exudates, and there was oesophageal stenosis and stricture formation (figure 2). After this discovery, an oesophageal transit with barium was performed, and there was no oesophageal motility disorders (figure 3). The patient was submitted to oesophageal dilatation. There was no further gastro-intestinal bleeding, and her haemoglobin remained stable. She was discharged by the 30th day.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of Black Oesophagus is the ingestion of corrosive agents, viral and fungal infections, oesophageal melanosis and pseudomelanosis, acanthosis nigricans and coal-dust miner.

TREATMENT

The patient's treatment was conservative, such as intravenous proton pump inhibitor, total parental nutrition, oral intake restriction and antifungal agents (fluconazole 200 mg intravenous for 7 days).

OUTCOME AND FOLLOW-UP

A second endoscopy at the hospital on day 21 showed a remarkable change from pitch-black mucosa to red, friable mucosa with exudates, and there was oesophageal stenosis and stricture formation (figure 2). Oesophageal transit with barium was performed, and there were no oesophageal motility disorders (figure 3). The patient was submitted to oesophageal dilatation. No further gastro-intestinal bleeding was related, and her haemoglobin remains stable. She was discharged on day 35.

Follow-up oesophago-gastro-duodenoscopy at 6 weeks revealed complete resolution of the oesophageal lesion and also gastritis.

DISCUSSION

Acute oesophageal necrosis (AEN) or 'Black Oesophagus' was first described by Goldenberg *et al* in the early 1990s in the era of modern endoscopy.^{1–5} Since then, fewer than 80 cases of AEN have been described so far.^{1–2} This is a rare event,^{5–6} a severe form of acute oesophagitis in which the oesophagus shows a dark, black-

To cite: Pereira O, Figueira-Coelho J, Picado B, *et al*. *BMJ Case Reports* Published online: [please include Day Month Year] doi:10.1136/bcr-2012-008188

Figure 1 Oesophago-gastro-duodenoscopy shows extensive candidiasis and a black-appearing oesophageal mucosa.

appearing colour, with friable mucosa in the two-thirds of the distal oesophagus.^{1 3}

The histological findings of an oesophagus biopsy showed necrotic mucosa and submucosa with inflammatory response.^{3 5} After several days, a thick white exudate appeared over pink granulation tissue, replacing the pitch-black oesophagus. This thick exudate is composed of necrotic debris and is known to protect the tissue from perforation.¹

AEN is a rarely described entity that is controversial in many ways with regard to its incidence,⁶ which has been reported to be 0.0125–0.2%.^{3–5 7}

Moreto *et al* have identified 10 cases in a review of 80 000 upper endoscopies; its incidence was 0.0125%.⁴

The aetiology is unknown in most cases. Most investigators have suggested an ischaemic origin based on histopathology and clinical data.^{5 6}

Figure 2 Endoscopy at 21st day showed a remarkable change from pitch-black mucosa to red, friable mucosa with exudate, oesophageal stenosis and stricture formation.

Figure 3 Oesophageal transit with barium.

The association of the lesion with a low-flow status, and its resolution with haemodynamic restoration, suggested that a temporary reduction in blood flow is the cause of oesophageal necrosis. In support of this hypothesis is the fact that the lower two-thirds of the oesophagus is the most vascularised and the most affected part. Malnutrition may compromise the mucosal defence and healing capacity after injury, resulting in AEN.⁶

Risk factors include advanced age, male sex, cardiovascular disease, haemodynamically compromised status, alcoholic ingestion, malnutrition, acute gastric outlet obstruction, diabetes, acute renal failure, hypoxaemia, hypercoagulation status and trauma.^{1–4 6}

Candidiasis is a rare cause of AEN.⁵ Among 88 cases of Black Oesophagus reported until now, two were of fungal infection aetiology.⁵

It may be an incidental finding in upper endoscopy; a black wall is seen, which ends abruptly at the oesophagus–stomach junction.⁶

The diagnosis is based on endoscopic appearance, after excluding the ingestion of corrosive agents, viral and fungal infections, oesophageal melanosis and pseudomelanosis, acanthosis nigricans and coal-dust miner, based on the patient's history.^{6 7} The condition is generally seen in the elderly and those having comorbid conditions.

Acute oesophageal bleeding is the most common presentation: melaena and haematemesis.^{3 5 6}

The prognosis is variable; it depends on the patient's general status and comorbid conditions, rather than the extent of local oesophageal necrotic lesions.⁶ Most of the deaths were caused by underlying illnesses. Death secondary to oesophageal necrosis occurs in less than 6% of cases.⁵

There is no standard treatment, but there is consensual use of the proton pump inhibitor and parenteral nutrition.^{2 5 6} The current recommendation is to also treat the patient's comorbidities, to optimise vascular perfusion and to treat oesophageal infection, if present.³ In some cases, where there was an absence of transmural lesions, the treatment was conservative, like we performed on our patient, without long-term consequences, although the mortality can reach 35–60%, depending on the underlying disease.⁶

The main reported complications of AEN were oesophageal stenosis and stricture formation.⁵

We report this case by the unusual nature of oesophageal necrosis. The aetiology of this case is still uncertain; despite the presence of oesophageal candidiasis. Low oesophageal perfusion pressure is a possible cause, as the patient presented herself with hypotension on admission. Alcohol abuse led to gastric mucosal irritation and inflammation and the accumulation of large amounts of acid gastric secretion, which can overcome the oesophagus defence wall. The usual defence of the oesophagus wall can be overcome.

Besides chronic alcoholism, the patient had no other medical condition. This case shows the need for more research on Black Oesophagus, its pathogenesis and larger studies of clinical reports.

Learning points

- ▶ Acute oesophageal necrosis is a rare condition seen in upper endoscopy.
- ▶ It is necessary to exclude other conditions such as the ingestion of corrosive agents, infections, oesophageal melanosis and pseudomelanosis, acanthosis nigricans and coal-dust miner.
- ▶ There is no standard treatment, but there is a consensual use of the proton pump inhibitor and parenteral nutrition to treat possible infections and patient comorbidities.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Yamauchi J, Mitsufuji S, Taniguchi J, *et al.* Acute esophageal necrosis followed by upper endoscopy and esophageal manometry/pH test. *Dig Dis Sci* 2005;50:1718–21.
- 2 Yasuda H, Yamada M, Endo Y, *et al.* Acute necrotizing esophagitis: role of nonsteroidal anti-inflammatory drugs. *J Gastroenterol* 2006;41:193–7.
- 3 Neumann DA II, Francis DL, Baron TH. Proximal black esophagus: a case report and review of the literature. *Gastrointest Endosc* 2009;70:180–1.
- 4 Endo T, Sakamoto J, Sato K, *et al.* Acute esophageal necrosis caused by alcohol abuse. *World J Gastroenterol* 2005;11:5568–70.
- 5 Kim YH, Choi SY. Black esophagus with concomitant candidiasis developed after diabetic ketoacidosis. *World J Gastroenterol* 2007;13:5662–3.
- 6 Ramos R, Mascarenhas J, Duarte P, *et al.* Acute esophageal necrosis: a retrospective case series. *Rev Esp Enferm Dig* 2008;100:583–5.
- 7 Grudell AB, Mueller PS, Viggiano TR. Black esophagus report of six cases and review of the literature, 1963–2003. *Dis Esophagus* 2006;19:105–10.

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