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Review

Acute oesophageal necrosis: A case report and review of the literature

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ABSTRACT

Aims: We discuss a case of acute oesophageal necrosis and undertook a literature review of this rare diagnosis.
Methods: The literature review was performed using Medline and relevant references from the published literature.
Results: One hundred and twelve cases were identified on reviewing the literature with upper gastrointestinal bleeding being the commonest presenting feature. The majority of cases were male and the mean age of presentation is 68.4 years. This review of the literature shows a mortality rate of 38%.
Conclusion: Acute necrotizing oesophagitis is a serious clinical condition and is more common than previously thought. It should be suspected in those with upper GI bleed and particularly the elderly with comorbid illness. Early diagnosis with endoscopy and active management will lead towards an improvement in patient outcome.

1. Introduction

Oesophageal necrosis, which is also known as "black oesophagus" or "oesophageal stroke" is defined as a dark pigmentation of the oesophagus associated with necrosis of the oesophageal mucosa on histological examination. It is important not to assume that every case of black oesophagus is caused by oesophageal necrosis, and for this reason we prefer to use the term acute oesophageal necrosis (AON) to avoid any confusion.

We present a case of acute oesophageal necrosis and we will be discussing the literature surrounding this rare entity.

2. Case report

A 77 year-old lady was admitted to the hospital for a routine vaginal hysterectomy. Her past medical history included hypertension, ischaemic heart disease, hypothyroidism, diverticulosis and a hiatus hernia. Her medications on admission included daily thyroxine, atenolol and statins.

During the vaginal hysterectomy, a dark brownish fluid was noted coming from the pelvis. As there was a concern of possible bowel injury, a lower midline laparotomy was carried out which revealed a normal looking small and large bowel. There was an endometriotic cyst assumed to be the source of the darkish brown fluid. An uncomplicated right salpingo-ophorectomy was performed. Whilst recovering from her operation, she spiked a temperature on the 3rd postoperative day and was commenced on intravenous amoxicillin. On the 4th day, she had an episode of coffee ground vomiting for which she was treated conservatively. A nasogastric tube was placed and 1000 ml of dark fluid was drained overnight.

On the 6th postoperative day, she developed signs of peritonitis and was referred to the surgical team. A laparoscopy was performed which showed a turbid coloured fluid within the peritoneal cavity. We proceeded to perform a laparotomy through an upper midline incision which showed a partial gastric volvulus, which was reduced, and a moderate to large hiatus hernia. There were multiple pin point ischaemic perforations on the posterior aspect of the fundus which were resected using a GIA stapler (Ethicon, Route 22, West Somerville, NJ 08876) and the staple line was oversewn with absorbable sutures. The decision was made to deal with this acute episode as quickly as possible and to repair the large hiatal defect with fundoplication and gastropexy at a later date after making a full recovery.

Following an initial improvement for a week, she then became increasingly short of breath and suffered an episode of melaena on the 8th day after her second laparotomy. Her blood results showed a gradual decrease in her haemoglobin with a drop of 2 g/dl along with an increase in her inflammatory markers (CRP-176 and WCC-31.7). Her antiphospholipid antibodies were negative. An urgent oesophageo-gastro-duodenoscopy was performed which showed circumferential black pigmentation of the entire oesophagus with a large hiatus hernia (Fig. 1). There was also a gastric volvulus and a large duodenal ulcer, but there was no evidence of fresh bleeding.

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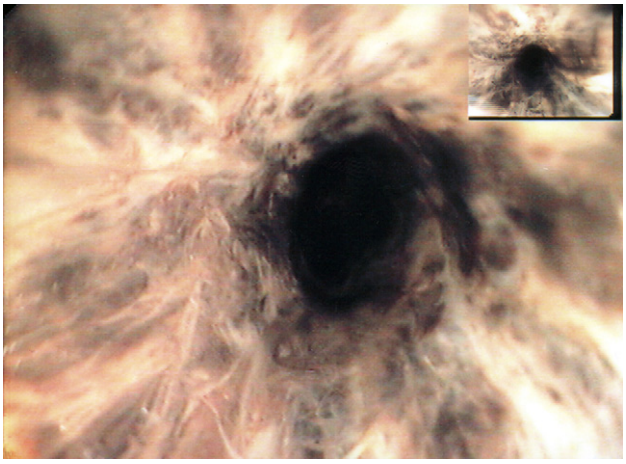

Fig. 1. Circumferential black pigmentation of the oesophagus encountered at OGD.

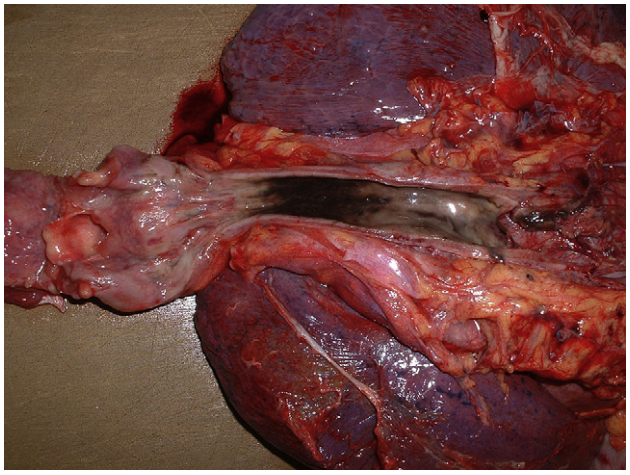

Fig. 3. A post mortem specimen showing the black oesophagus with clear demarcation at the cricopharyngeus.

The previous gastrectomy staple line was intact. The discoloration of the oesophagus was attributed to haematin staining. She was subsequently taken to the intensive care unit for resuscitation and monitoring and commenced on intravenous proton pump inhibitors.

A CT scan was performed, showing clear pulmonary arteries, large bilateral pleural effusions and consolidation. There was a fluid filled dilated gastric volvulus in the posterior mediastinum. Small pockets of air were noted adjacent to the oesophagus, but no gross free air within the abdomen. A left sub-phrenic collection was noted in addition to an ill defined mass anteriosuperior to the pancreas and posterior to the left lobe of the liver, possibly a haematoma (Fig. 2).

In view of the deterioration of her condition and the above CT findings, the patient returned to theatre for a further laparotomy. An upper midline incision was performed to drain the left subphrenic collection, which turned out to be an infected haematoma. There was an organo-axial volvulus of the stomach which was all within the chest cavity through a large hiatus hernia. A left thoracotomy was performed, which revealed a large pleural effusion and the volvulus of the stomach with necrosis of the fundus. Upon dissecting the stomach from the hiatus, the necrosis was noted to extend into the oesophagus. The upper extent of the necrosis was high and could not be determined. It was therefore not possible to anastomose the oesophagus to the distal stomach. A proximal gastrectomy and oesophagostomy was attempted. Due to the severity of the necrosis, and with deterioration of her condition and persistent sepsis she died later that night.

The post mortem showed a blackened, thinned oesophagus compatible with necrosis involving the remaining upper half of the oesophagus (Fig. 3). Histology confirmed acute necrosis confined to the mucosa but in some areas it extended more deeply to involve submucosa and muscularis propria (Fig. 4). A report of acute oesophageal necrosis of unknown aetiology was made.

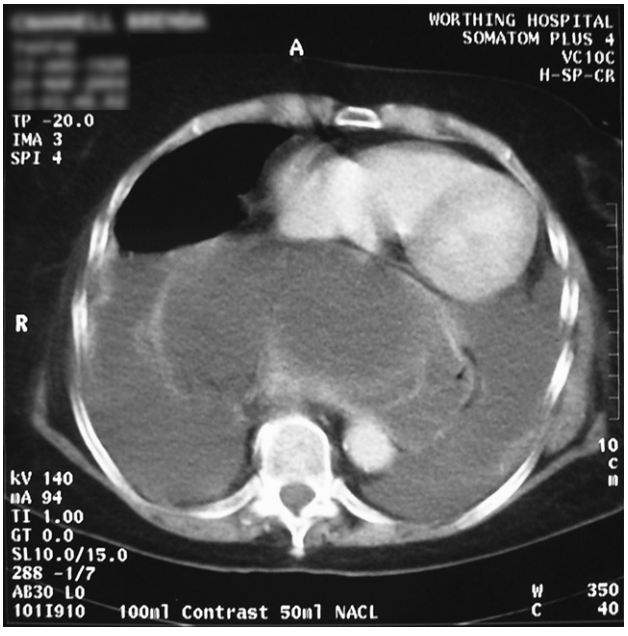

Fig. 2. A CT scan of the chest which shows a large well defined fluid filled structure representing a gastric volvulus in the posterior mediastinum.

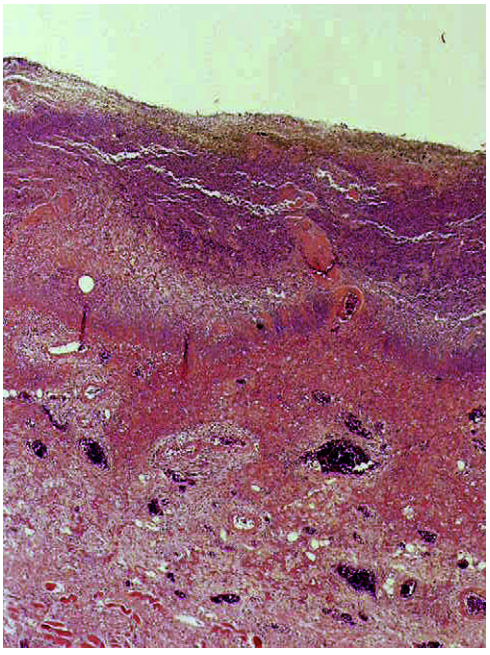

Fig. 4. Histology specimen identifying mucosal necrosis of the oesophagus.

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2.1. Introduction

Acute oesophageal necrosis or "black oesophagus" used to be considered a rare entity. It was first described in post mortem cases by Brennan[1] and Lee and colleagues[2]. A French report indicated a prevalence[3] of 0.2% and its incidence has been reported by various groups to be between 0.01[4]– 0.0125%[5]. There has been new interest in this condition over the last few years and it seems that it may perhaps occur more frequently than initially thought. In a prospective 1 year study by Ben Soussan and colleagues[6], they reported that acute oesophageal necrosis was present in 0.2% of their 3900 patients who underwent an upper GI endoscopy. Augusto et al.[7] reported the condition in 29 of 10,295 patients who had an upper gastrointestinal endoscopy (0.28%) during a 5 year period. In their large retrospective series they found that 83% of those with the disease had comorbid conditions.

It is important to differentiate acute oesophageal necrosis from other diseases causing a black oesophagus on endoscopy such as melanosis[8] (which is more common among the Japanese), malignant melanoma,[9] pseudomelanosis,[10] acanthosis nigricans,[11] exogenous dye ingestion and coal dust exposure,[46] (see Table 1). At autopsy it is often misinterpreted as haematin discolouration, which is the most important differential diagnosis.[12]

The majority of cases have no real known aetiology, but there is a large list of conditions that are associated with acute oesophageal necrosis (see Table 2). Although the majority of patients have an adverse outcome, there have been a few cases whereby the patient left the hospital with minimal or even no sequelae.

2.2. Clinical presentation and endoscopic findings

The majority of patients present with symptoms of coffee ground vomiting, frank haematemesis or melaena. There may not, initially, be a significant drop in haemoglobin. Other causes of acute oesophagitis such as infectious, toxic, traumatic, caustic and radiotherapy must be excluded. A strict definition of acute oesophageal necrosis excludes patients with caustic ingestion injury, and those with small local necrotic areas which might well be secondary to infection.[24]

The endoscopic findings vary from white plaques which can be similar to those of pseudomembranous colitis as reported by Zweiban.[19] These can be easily picked with a forceps or stripped away by the advancing scope, to reveal a more diffuse black-green patchy appearance involving the distal and sometimes the whole of the oesophagus.

Necrosis of the friable mucosa with adherent yellow exudates and ulceration is the usual endoscopic finding in the majority of patients.[63] The blackened mucosa is demarcated clearly from the cardia at the gastroesophageal junction. With the appropriate management, the process of re-epithelisation will begin leading to a normal pink mucosa granulating underneath a thick, white exudate which eventually peels away.[25,44]

Table 1
Black oesophagus.

- Acute oesophageal necrosis
- Melanosis
- Pseudomelanosis
- Malignant melanoma
- Acanthosis nigrans
- Coat dust exposure[46]
- Dye ingestion
- Haematin discolouration
- Caustic inury[22]

Table 2
Associated conditions.

- Gastric outlet obstruction[4]
- Gastric volvulus[13]
- Ischemia[2,14,43,45]
- Shock[15,39]
- Hypersensitivity to antibiotics[16,17]
- Viral infections[18,40,42]
- Associated with erythema multiforme or Stevens–Johnson syndrome[19,20]
- Others: anticardiolipin antibodies[21] and hypothermia[1]
- Irradiation
- Trauma[22,23]

2.3. Histology

AON appears as severe mucosal and submucosal necrosis with inflammation of the muscle fibres. There may be thrombosis of the blood vessels.[5] In the majority of cases, there is a circumferential necrosis which is more prominent in the lower third of the oesophagus demarcating it from the cardia which seems to be spared.[37]

In a study by Jacobsen and colleagues,[12] it was reported that oesophageal necrosis was distinguished from autolytic changes by the presence of vital reaction (i.e. fibrin exudation and presence of neutrophils). They found that oesophageal necrosis which presented as dark coloured or black, punctuate, striped or confluent areas, most frequently located in the distal oesophagus, was observed in 10.3% (32 of the 310 patients). The necrosis extended into or through the muscularis propria in nine patients.

2.4. Aetiology

The disease can develop fairly rapidly,[22] but the mechanism of its development remains unclear. Two main underlying causes of oesophageal damage have been proposed, acid reflux and hypoperfusion.

As the lesion in most cases stops abruptly at the gastro-oesophageal junction, this suggests that acid reflux may play a role in the aetiology. Transient or complete gastric outlet obstruction has been projected as a cause, with a direct toxic effect of the acid or pepsin.[26,27] The oesophagus is exposed to large amounts of gastric secretions for a long period of time, causing damage to the mucosal layer with disruption of the cellular tight junctions. There is impairment of oesophageal motility as well as a reduction to the buffering capacity of mucus and saliva.

An alternative proposal is that the indirect effect of acid on the mucosal barrier leads to a drop in the oesophageal blood flow, therefore triggering the mucosal injury.[28] Alkaline reflux might also play a role especially in those presenting with yellowish discolouration in the oesophagus.

Yamauchi et al.[51] have shown, however, that this condition can still develop in the absence of gastro-oesophageal reflux. They examined a previously healthy individual who developed idiopathic oesophageal necrosis with resolution. They performed pH monitoring and pressure manometry 5 months following the event and showed no evidence of reflux with normal lower oesophageal sphincter pressure. This has also been demonstrated by Reichart et al.[53]

The oesophagus has a diverse blood supply from the inferior thyroid, aorta, bronchial, intercostals and left gastric arteries. There is mounting evidence suggesting a temporary reduction in oesophageal blood flow and decreased perfusion may lead to oesophageal necrosis, which may be reversible with restoration of a normal haemodynamic status. The frequent involvement of the distal 1/3 of the oesophagus which is less well vascularised than the rest of the oesophagus[29] clearly supports this theory. This is also suggested by the absence of gastric lesions and the association of duodenal mucosal injury.[6]

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Table 3
Acute oesophageal necrosis demographic and clinical data review.

Ref #	Presumed Dx	Complaint	Age	Gender	Histology	OGD/autopsy* findings	Outcome
Brennan, 1967[1]	Severe hypothermia	Confusion & Alteration of consciousness	84	M	No biopsy	Blackening lower $^{3}/_{4}$ *	Death
McManus et al., 1975[25]	? infectious (lactobacillus)	Retrosternal discomfort	67	F	Gram positive bacilli with cellular debris	Lower 10 cm of oesophagus was tough with a necrotic membrane	Death
Lee et al., 1977[2]	Rupture thoracic aorta	Dysphagia	81	M	Transmural infarction with necrosis	Black oesophagus in distal 2/3*	Death
Zweiban et al., 1986[19]	SJS (penicillin)	Dysphagia	19	M	Necrotic mucosa	White plaques and inflamed mucosa	Resolution
	SJS (phenytoin)	Dysphagia	36	M	Necrotic plaque formation	Thick necrotic plaque mid & distal oesophagus	Died
Cummings, 1990[24]	Severe reflux with gastric outlet obstruction	Haematemesis	67	F	No biopsy	Ulcerative oesophagitis, distal blackening, DU, gastric outlet obstruction	Oesophageal stricture
Mangan et al., 1990[16]	Erythema multiforme – 2 patients	– Haematemesis – Dysphagia	Unknown	Unknown	Necrotic muscle fibres	Diffuse black oesophagus	Unknown
Goldenberg, 1990[23]	Acute cholecystitis	Haematemesis	82	M	Necrotic debris	Proximal yellow exudates and distal blackening, multiple large DUs	Stricture/ oesophagectomy
Marie-Christine et al., 1991[22]	Pancreatic cancer, ischemia (carcinomatosis), caustic injury (endoscope detergent)	Haematemesis	67	M	Necrotic debris, neutrophils infiltration	Proximal ulceration and mostly black oesophagus	Death
Goldenberg, 1991[14]	Ischemia (sepsis)	Colon ca., peritonitis	79	M	Necrotic debris	Distal blackening, actively bleeding DUs	Death
Hoffman et al., 1992[62]	*Penicllium chrysogenum* infection in an AIDS patient	Dysphagia and epigastric pain	30	M	Fungal hyphae on biopsy	Necrotising oesophagitis mid to distal oesophagus	Death
Mahe et al., 1993[20]	Stevens–Johnson syndrome (SJS)	Haematemesis	35	M	Necrotic epithelium	No endoscopy	Resolution
Moreto et al., 1993[5]	– Cholecystitis, acute renal failure	– Coffee ground vomit	67	M	All patients had diffuse necrosis involving mucosa and submucosa	– Distal blackening, normal GOJ mucosa	Resolution
	– Rectal cancer	– Coffee ground vomit	60	M		– Distal blackening, normal GOJ mucosa	Resolution
	– Lung Cancer	– Abdominal pain & dark vomit	72	M		– Distal blackening, normal GOJ mucosa	Oesophageal stenosis
	– Ovarian cancer	– Haematemesis	57	F		Gastroduodenal erosions	Resolution
	– Hypopharyngeal cancer	– Epigastric discomfort & vomiting	65	M		– Distal blackening, normal GOJ mucosa	Resolution
	– Gastrinoma	– Haematemesis	26	M		– Distal blackening, normal GOJ mucosa	– Mediastinal abscess
	– Aortic aneurysm	– Coffee ground vomit	80	M		Pyloric stenosis	– Death
	Gastric obstruction	– Coffee ground vomit	72	M		– Distal blackening, normal GOJ mucosa, acute GU	– Resolution
	– Multiple trauma	– Melaena	68	M		– Distal blackening	– Oesophageal stenosis
	– *Cryptosporidia* colitis	– Melaena	86	M		– Distal blackening, DU – Distal blackening, DUs – Distal blackening, DUs	– Discharge no follow-up
Cappell, 1994[21]	Anticardiolipin antibody syndrome	Epigastric pain	50	M	Not performed	Necrosis & perforation of the oesophagus*	Death
Erdozain et al., 1994[30]	Gallstone pancreatitis	Haematemesis	60	M	Necrotic debris and full thickness lysis of wall	White exudate upper 1/3 oesophagus and distal 2/3 were black	Asymptomatic at 1 2 months
Haviv et al., 1996[15]	Ischemia (shock)	Haematemesis	83	M	Necrotic debris & neutrophils infiltration	Confluent pseudomembranes (black) entire oesophagus	Dysphagia, Death at 3 months
Obermeyer, 1998[31]	Ischemia (low flow)	Coffee ground vomit &dysphagia	88	F	Necrotic debris & neutrophils infiltration	Proximal & distal blackening	Resolution

Macedo et al., 1998[59]	Acute fatty liver of pregnancy	Haematemesis	26	F	No biopsy	Necrotic debris over lower oesophagus with black oesophagus of distal 6 cm	Resolution
Watanabe et al., 1999[48]	Mediastinal haematoma from rupture thoracic aortic aneurysm	Haematemesis	76	M	Transmural necrosis	Annular ulcer 8 cm in length from the mid to distal oesophagus	Died from mediastinitis
Lacy et al., 1999[4]	Gastric outlet obstruction, DU, diabetes, atrial fibrillation, coronary heart disease, renal impairment, hypertension	Coffee ground vomit, abdominal pain	74	M	Not performed	Black mucosa 5 cm to GOJ, dilated stomach, deformed pyloric channel	Resolution
	Multiple trauma following RTA, hypertension, diabetes mellitus	Haematemesis	44	M	Not performed	Blackened oesophagus from cricopharyngeus to GOJ, large DU	Resolution
Cattan et al., 1999[18]	Herpes oesophagitis (post chemotherapy)	Epigastric pain, dysphagia & haematemesis	74	M	Necrotic basis with clusters of multinucleated giant cells	Blackened oesophagus with ulcerations from upper 1/3 to cardia	Died 3 months later from disseminated malignancy
Kram et al., 2000[13]	Hiatus hernia with volvulus	Coffee ground vomit	75	F	Not performed	Blacken oesophagus distal 1/3, large hiatus hernia, gastric volvulus	Resolution after surgery
Baffy et al., 2000[43]	Post left hemicolectomy	Upper gastrointestinal bleeding	73	M	Mucosal ulceration with purulent exudates	Adherent yellow exudate speckled with black spots in the distal 2/3	Developed stricture
Reichart et al., 2000[53]	Post anterior resection	Severe interscapular pain	73	M	No biopsy	Black oesophagus from 5 cm below the cricopharyngeus to the cardia	Resolution
Katsinelos et al., 2001[32]	Coronary insufficiency & hypertension	Epigastric pain & melaena	76	M	Necrotic debris & inflammation	Black oesophagus, with ulceration, pre-pyloric ulcers	Resolution
De la Serna-Higuera et al., 2001[44]	Pancreatitis	Coffee ground vomit	83	M	Necrotic debris	Black oesophagus from the proximal third to the cardia	Died from sepsis
Barjas et al., 2001[42]	CMV infection	Upper gastrointestinal bleeding	50	M	Unspecified oesophagitis with necrosis	Black diffuse mucosa in the lower oesophagus	Died from multiple organ failure
Casella et al., 2001[58]	– Diabetic ketoacidosis	Acidotic	67	M	Necrotic tissue	Black oesophagus through entire length	Died
	– Hip fracture	Melaena	69	M	No biopsy	Black oesophageal mucosa in the mid to distal 1/3	Died
Katsinelos et al., 2002[33]	Alcohol overindulgence	Vomiting epigastric pain & coffee ground vomit	23	M	Necrosis & ulcerated mucosa	Black oesophagus from the middle down to the cardia, with submucosal haemorrhage in the stomach	Resolution
Odelowo et al., 2002[49]	Cholangiocarcinoma	Coffee ground vomitus	61	M	Necrotic epithelium, fibrinopurulent exudates	Necrosis of the lower $^{3}/_{4}$ of the oesophagus	Died from PE after 4 weeks
Mall et al., 2002[47]	Post PDT for oesophageal carcinoma	Sepsis and perforation	62	M	Necrotic oesophagus	No info	Oesophagectomy and gastric pull through
Ben Soussan et al., 2002[6]	– Diabetic mellitus hyperglycemia ketoacidosis, acute renal failure	Epigastric pain	21	M	– Mucosal & submucosal necrosis & vascular thrombosis	Ischemia involving the distal 1/3 of the oesophagus	Resolution
	– Myocardial infarction & linitis plastica, acute renal failure	– Haematemesis & melaena	87	M	– Mucosal & submucosal necrosis	– Ischemia of the middle and distal 1/3+ linitis plastica	Death (day 9)
	– Atrial fibrillation & acute renal failure	– Melaena	89	M	– Not performed	– Ischemia distal 1/3	Death (day 6)
	– Alcoholic cirrhosis, diabetes mellitus, atrial fibrillation, anaemia	– Haematemesis	53	M	– Mucosal & submucosal necrosis	– Ischemia distal 1/3 & Hiatus hernia	Resolution
	– Alcoholic cirrhosis, stroke, acute renal failure	– Haematemesis	79	M	– Mucosal, submucosal & muscular necrosis, vascular thrombosis	– Ischemia middle & distal 1/3	Death (day 10)
	– Terminal prostatic cancer, acute renal failure, anaemia	– Haematemesis	68	M	– Mucosal & submucosal necrosis, vascular thrombosis, mycotic infection	– Ischemia distal 1/3	Death (day 13)
	– Diabetes mellitus prostatic ca., myocardial infarction, anaemia	– Haematemesis	79	M	Not performed	– Ischemia distal 1/3	Resolution
	– Alcoholic cirrhosis, anaemia	– Anaemia	66	F	Not performed	– Total oesophagus	Resolution

(*continued on next page*)

Table 3 (*continued*)

Ref #	Presumed Dx	Complaint	Age	Gender	Histology	OGD/autopsy* findings	Outcome
Khan et al., 2004[34]	Chronic alcohol abuse, cirrhosis, mental confusion	Confusion & Alteration of consciousness	59	M	Fibrinous debris with acute inflammatory cells	20 cm segment of Necrosis with exudates, ulceration and friable middle and distal oesophageal mucosa	Resolution
Rejchrt et al., 2004[35]	GI bleed	Haematemesis	55	F	Necrosis of mucosa and submucosa	Black oesophagus in mid oesophagus	No mention
Augusto et al., 2004[7]	Wide range of pathology	Haematemesis and or melaena	Range 40–91	23 M 6 F	Necrosis of the mucosa, with fibrin thrombi in capillaries	Oesophageal necrosis mainly the distal 2/3 plus other coexisting endoscopic findings	10 died due to serious comorbid factors
Park et al., 2004[55]	Traumatic aortic transaction and compressing haematoma	Dysphagia	47	M	Ischaemia on histology	No endoscopy	Died
Pelletier et al., 2004[65]	Lymphoma postop shock state	Anaemia	65	M	Necrotic mucosa and submucosa	Diffuse necrosis throughout all the mucosa	Resolution
Tsokos et al., 2005[36]	Chronic alcohol abuse	Death in the community	Range 43–86	4 M 1 F	Necrotic mucosa with neutrophilic infiltrate*	Autopsy specimens	All died
Husova et al., 2005[37]	Carcinomatosis/sepsis	Haematemesis	56	M	Necrosis of the mucosa down to muscularis mucosae*	Oesophageal necrosis in more than 2/3 of the oesophagus	death
Yamauchi et al., 2005[51]	Alcohol excess	Haematemesis	60	M	No initial biopsy	Circumferential necrosis from middle to lower oesophagus	Resolution
Softoiu et al., 2005[26]	Alcoholic abuse Alcoholic hepatitis	Haematemesis & melaena	66 42 57	F F M	Severe inflammation and necrosis	Ranges from black mucosa to Thick white exudate (pseudomembrane)	Resolution
Sako et al., 2005[38]	Nephrotic syndrome & malnutrition	Coffee ground vomit	64	F	Ulcerated mucosa & massive necrosis	Patchy black appearance of the oesophagus with adherent yellow exudates.	Resolution
Endo et al., 2005[52]	Alcohol excess Lactic acidosis	Coffee ground vomit	41	M	No biopsy	Black circumferential mucosa form 4 cm below the cricopharyngeus to the cardia	Resolution with stricture requiring balloon dilatation
Grudell et al., 2006[54]	– Ischaemia	Haematemesis	79	M	Necrotic debris with small amount of brown pigment	All had black oesophagus but there is no specific mention of each individual case	Death
	– Ischaemia	Epigastric pain with sour taste	80	M	Necrotic debris with small amount of brown pigment		Distal oesophageal stricture
	– Candida oesophagitis	Vomiting and dysphagia	29	M	Necrotic debris with small amount of brown pigment		Death from sarcoma
	– Ischaemia	Haematemesis	73	M	No biopsy		Recovery
	– Ischaemia	Melaena	49	M	Necrotic debris with large amount of brown pigment		Recovery
	– Corrosive injury from ethanol	Haematemesis and odynophagia	41	M	No biopsy		Recovery
Trappe et al., 2007[60]	CMV infection in transplant patient	Epigastric pain	54	M	CMV positive	Distal 1/3 of oesophagus was black	Resolution
Kim et al., 2007[39]	Diabetic ketoacidosis and hypotension	Haematemesis	34	M	No biopsy	Black and friable mucosa for entire oesophagus	Developed stricture that required surgery
Nagri et al., 2007[40]	Herpes simplex	Coffee ground vomit and melaena	54	M	Multinucleated giant cells with Cowdry type A intranuclear inclusion bodies in epithelial cells	Grade D oesophagitis with black lower third of the oesophagus	Resolution
Tanaka et al., 2007[41]	Diabetes mellitus	Vomiting and chest pain	67	M	Necrotic debris	Black oesophagus from proximal 2/3 to the cardia	Resolution on PPI
Hwang et al., 2007[50]	Paraoesophageal hernia with gastric volvulus	Coffee ground vomit	84	F	No biopsy	Dusky friable oesophagus along most of the length of the oesophagus	Resolution following reduction of the hernia and closure of the defect
Burtally et al., 2007[56]	Ischaemia from a low flow state	NGT revealed dark brown fluid	77	M	Necrotic debris	Black oesophagus in the middle and distal 1/3	Resolution

Watermayer et al., 2007[57]	Drug induced hepatitis	Melaena	63	F	No biopsy	Black oesophagus along entire length	Died from liver failure
Carter et al., 2007[61]	Diabetic ketoacidosis	Pneumomediastinum	25	F	No biopsy	Circumferential black oesophagus from 20 cm to 36 cm	Resolution
Le et al., 2007[63]	Leukaemia	Odynophagia and dysphagia	62	F	Necrotic tissue	Black oesophagus from mid oesophagus to stricture. Yellow exudates above	Death from respiratory failure
Gurvits et al., 2007[64]	Acute pancreatitis	Coffee ground vomit	73	M	Necrotic debris	Black oesophageal mucosa from mid to distal oesophagus	Death from septic shock
Sayegh et al., 2008 (our case)	Gastric volvulus, ischemia	Coffee ground vomit	77	F	No biopsy	Blackened oesophagus down to the stomach, large DU, volvulus of the stomach	Death

Table 4
Summary of demographics, clinical features and outcomes of 112 cases of AON.

Feature	Value
Age:	
Mean	68.44
Range	19–91
Sex:	
Male	88
Female	22
Unknown	2
Presenting feature:	
Upper GI bleed	73 (65%)
Epigastric pain	9 (8%)
Dysphagia	8 (7%)
Melaena	6 (5%)
Unknown	5 (4%)
Other	11 (10%)
Comorbid condition:	
Shock/hypotension	22 (20%)
Liver dysfunction	17 (15%)
Malignancy	12 (11%)
Infectious	8 (7%)
Unknown	29 (26%)
Other	24 (21%)
Outcome:	
Death	43
Resolution	55
Stricture	8
Unknown	4
Other	2

It is likely, therefore, that the cause is multifactorial with hypoperfusion as perhaps the most important among all the above factors being implicated in the pathogenesis.

3. Management

There is no specific treatment for AON. The management revolves around ample hydration, gastric acid suppression with proton pump inhibitors, short term parenteral nutrition and treatment of the underlying cause. Sucralfate has been suggested based on its ability to bind pepsin and stimulate mucus secretion in addition to its cytoprotective effects.[4] Urgent endoscopy is warranted to make the diagnosis and possibly treat any bleeding ulcers.[4] Re-endoscopy is indicated to assess healing.

Surgery has a definite role in the acute phase dealing with the gastric volvulus, perforation and other possible causes. It is also needed later on for those who survive and develop oesophageal strictures requiring an oesophagectomy.[14] There is some suggestion that patients should remain on proton pump inhibitors to prevent a relapse of the condition.[41]

4. Discussion

We have reviewed all the individual reported cases ($n = 112$) of acute oesophageal necrosis to date (Table 3). We are able to draw several conclusions about this condition from the above data. The condition predominantly occurs in men (78%) with a male to female ratio of 4:1, two patients' sex was unknown. The mean age of presentation is 68.44 years with a range of 19–91.

There appears to be no obvious unifying comorbid condition other than to say that the majority have significant comorbidities and generally present when they are very unwell. We found that the most common comorbid condition was shock/hypotension, accounting for 20% (22/112) of the reported cases. Another common contributing factor was liver dysfunction 15% (17/112), caused by a variety of underlying conditions ranging from alcoholic cirrhosis to acute fatty liver of pregnancy. A total of 7% (8/112) of the reported cases concerned an infective cause, with 3% (3/112) suffering from sepsis. The

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other common aetiological factor was advanced malignancy, which was reported in 11% (12/112) of the reported cases. Rarer causes included gastric outlet obstruction, gastric volvulus, erythema multiforme, aortic rupture and Stevens–Johnson syndrome.

The majority of patients presented to hospital with upper gastrointestinal bleeding (65%) with or without haemodynamic compromise. Other presentations included epigastric pain 8% (9/112), melaena 5% (6/112) and dysphagia 7% (8/112), with the more uncommon presenting complaints including confusion 1.8% (2/112), peritonitis 0.9% (1/112) and acidosis 1.8% (2/112).

During endoscopy the majority of cases had a black oesophagus in the distal 1/3 to 2/3 with sparing of the cardia. A reason for variation in the endoscopy findings is likely to be secondary to the timing of the procedure in relation to the onset of the symptoms. The appearance of the oesophagus will evolve over time as the condition progresses through its various stages. This will also account for the variability in the biopsy results.

When looking at the outcome of these patients it is very difficult to conclude whether the death was directly related to AON or because of the associated conditions. Sharma et al.[8] conclude that death is most likely to occur from the underlying condition. From this analysis of currently available published literature the mortality rate of this condition is 38%, which is a similar figure to that obtained by Augosto et al.[7] Resolution was seen in 49% (55/112) of patients. A total of 7% (8/112) developed a stricture and one required oesophagectomy. The outcome was unknown in 3.6% (4/112) patients and one developed a mediastinal abscess (Table 4).

In the case reported above endoscopy showed necrosis involving the full length of the oesophagus, and also demonstrated some necrosis of the fundus of the stomach. Looking at the literature there are two other reported cases of AON that involve a gastric volvulus as a potential precipitant. Kram et al.[13] reported a complete hiatus hernia with mesentero-axial gastric volvulus and Hwang et al.[50] reported an organo-axial volvulus again with a large hiatus hernia. Both of these cases did not report necrosis of the fundus of the stomach, as in the above case. It may be that endoscopy in this case was performed at a later stage, therefore demonstrating a possible progression to fundal necrosis. There is also the possibility that it may have been a manifestation of the underlying septic state of the patient.

We feel that the probable cause of AON and subsequent death in our patient was multifactorial, with sepsis and mucosal hypoperfusion being the precipitants. It is feasible to conclude that the poor general condition of our patient contributed to the extensive acute oesophageal necrosis and finally death.

5. Conclusion

Acute necrotizing oesophagitis is a serious clinical condition and is more common than previously thought. It should be suspected in those with upper GI bleed and particularly the elderly with comorbid illness. Early diagnosis with endoscopy and active management will lead towards an improvement in patient outcome, but in general, outcome is poor with a mortality of 38%.

Conflict of interest
None declared.

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