

# Black oesophagus

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In the last decade, several case reports and some review articles (1, 2) in scientific literature have contributed to a greater awareness of a condition known as black oesophagus or acute oesophageal necrosis. This condition is characterized by a black stained circumferential segmental lesion in the oesophagus sharply demarcated against a normal gastric mucosa at the cardia extending orally, sometimes more than 15 centimetres.

Early upper endoscopy after onset of symptoms shows the characteristic black oesophageal wall as clearly demonstrated in the report by Stanislav Rejchrt et al. in this issue of the Journal (6). Biopsies show no epithelial elements but only inflammatory changes or granulation tissue and sometimes an abundance of regenerating vascular elements as an expression of initial healing. The changes look ischaemic and the necrosis is only rarely transmural with a risk of perforation to the mediastinum. If the endoscopic examination is performed days after onset, or if control endoscopic examination is carried out, the oesophageal wall is no longer black but it is covered by a white pseudomembrane (6), which may be mistaken for severe candidiasis (7). This represents an early stage in the healing process.

The mechanism behind black oesophagus is not well understood, but there seems to be an element of ischaemia involved leading to necrosis of the mucosa, submucosa and sometimes even transmural necrosis with perforation. Only rarely are similar concomitant lesions detected in the stomach, duodenum or colon. Victims of black oesophagus are often multimorbid patients suffering from septicaemia, shock, renal insufficiency, fulminant hepatitis (7), peritonitis or cardiac insufficiency. Therefore middle-aged or old patients in intensive care units are typical victims of black oesophagus.

Acute oesophageal necrosis is not always diagnosed before the patient dies, and since many years limited and sometimes extensive black oesophagus is not diagnosed until autopsy (4). Histological examination of suspicious lesions can clearly document that such lesions have features of intravital reactions,



**Autopsy specimen showing a classical case of black oesophagus in a 39-year-old patient who died from septicaemia related to a pancreatic abscess. A perforation is seen in the distal quarter of the organ (arrow).**

like in our recent publication, where lesions diagnostic for black oesophagus was found in 10 % of our prospective study (5). The earliest publication of acute oesophageal necrosis to my knowledge is from 1914 where the patient was a young man with complicated tuberculosis and septicaemia followed by severe odynophagia before he died. A completely black oesophagus was found at autopsy (3).

Our youngest victim of acute oesophageal necrosis was a 7-year-old boy with diffuse peritonitis and septicaemia. Careful gastroscopy because of haematemesis showed a black oesophagus involving the entire oesophagus with perforation in the distal third and

therefore oesophagectomy was performed. Three months later he was re-operated and had successful colon interposition. He is now doing well.

Clinically, the patients often present repeated coffee-grounds like haematemesis and accordingly upper endoscopy is requested. If the underlying disease can be controlled, most cases of black oesophagus may heal. In the early phase after onset, severe odynophagia is frequent and in about one third of the cases, healing may lead to fibrotic strictures with subsequent dysphagia and the need for dilatation. Interestingly, the healing seems to progress upwards from the cardia and downwards from the upper end of the necrotic segment with a shortening of the segment covered with white pseudomembrane. Healing may take many months.

Recent publications have found black oesophagus in 0.2 - 0.3 % of upper endoscopies (1,2), but the true incidence is difficult to calculate and it may be even higher, dependent upon the case mix of patients.

Treatment of black oesophagus is symptomatic. Most doctors prescribe proton pump inhibitors in the healing phase, and temporary tube feeding may be necessary to ensure optimal enteral nutrition. Dilatati-

on must be carried out very carefully and repeated dilatations are often needed.

#### REFERENCES:

1. Augusto F, Fernandes V, Cremers MI, Oliveira AP, Lobato C, Alves AL, Pinho C, de Freitas J. Acute necrotizing esophagitis: a large retrospective case series. *Endoscopy* 2004; 36: 411 - 415.
2. Ben Soussan E, Savoye G, Hochain P, Herve S, Antoniotti M, Lemoine F, Ducrotte D. Acute esophageal necrosis: a 1-year prospective study. *Gastrointest. Endosc.* 2002; 56: 213 - 217.
3. Brekke A. Nekrose av oesophagus. *Med. Rev. (No)* 1914; 31: 210 - 214.
4. Dalgaard JB. Oesophago-gastro-duodenal ulcerations encountered at autopsy. *Acta Pathol Microbiol Scand* 1957; 41: 1 - 18.
5. Jacobsen NO, Christiansen J, Kruse A. Incidence of oesophageal necrosis in an autopsy material. *Acta Pathol Microbiol Immunol Scand* 2003; 111: 591 - 594.
6. Rejchrt S, Douda T, Kopáčová M, Široký M, Repák R, Nožička J, Špaček J, Bureš J. Acute oesophageal necrosis (black oesophagus) - report of three cases. *Folia Gastroenterol Hepatol* 2004; 2: 87 - 91.
7. Saftoiu A, Cazacu S, Kruse A et al. Acute esophageal necrosis associated with alcoholic hepatitis: is it black or is it white? Submitted to *Endoscopy*.

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