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PICTURES IN DIGESTIVE PATHOLOGY

Acute oesophageal necrosis (black oesophagus)

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BACKGROUND

Acute oesophageal necrosis (AEN) is a rare entity characterized by a black aspect in oesophageal mucosa, mainly distal and with a variable extension at the proximal level, terminating abruptly at the gastro-oesophageal junction (1).

CASE REPORT

A 54-year-old man was admitted to hospital after being found unconscious in his home. He had a history of alcoholism, multiple drug addictions, and type I diabetes mellitus. At admission, he had hyperglycaemia (550 mg/dL) with glucosuria and ketone bodies in the urine, along with septic shock refractory to bilateral alveolar infiltrates and severe respiratory failure. The patient died 24 hours post admission due to multiple organ failure, with diabetic ketoacidosis decompensated by possible respiratory infection in a patient with polytoxicomania. The autopsy confirmed the presence of acute bilateral bronchopneumonia, chronic pancreatitis, severe hepatic steatosis, and generalized congestive changes. At the oesophagus, AEN was evident (Figs. 1 and 2).

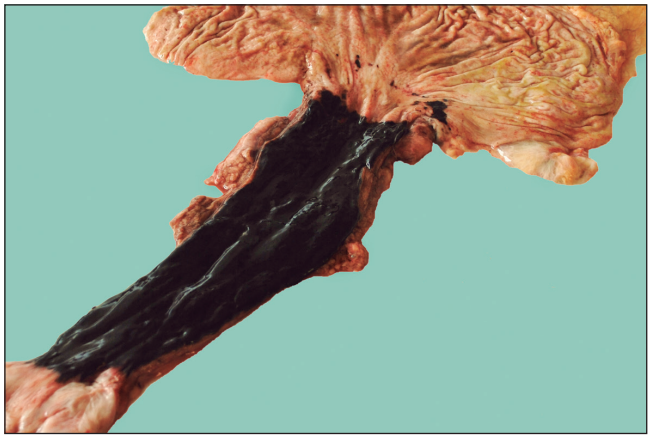

Fig. 1. Oesophagus opened longitudinally during autopsy. Non-formalinized section. Dark coloration with clear boundaries around the circumference and along the whole length of the oesophageal mucosa, with an abrupt transition at the level of the gastro-oesophageal juncture.

DISCUSSION

The incidence of AEN in endoscopic studies is less than 0.3% and contrasts with the 10.3% described in autopsies. The transitory nature of the causal stim-

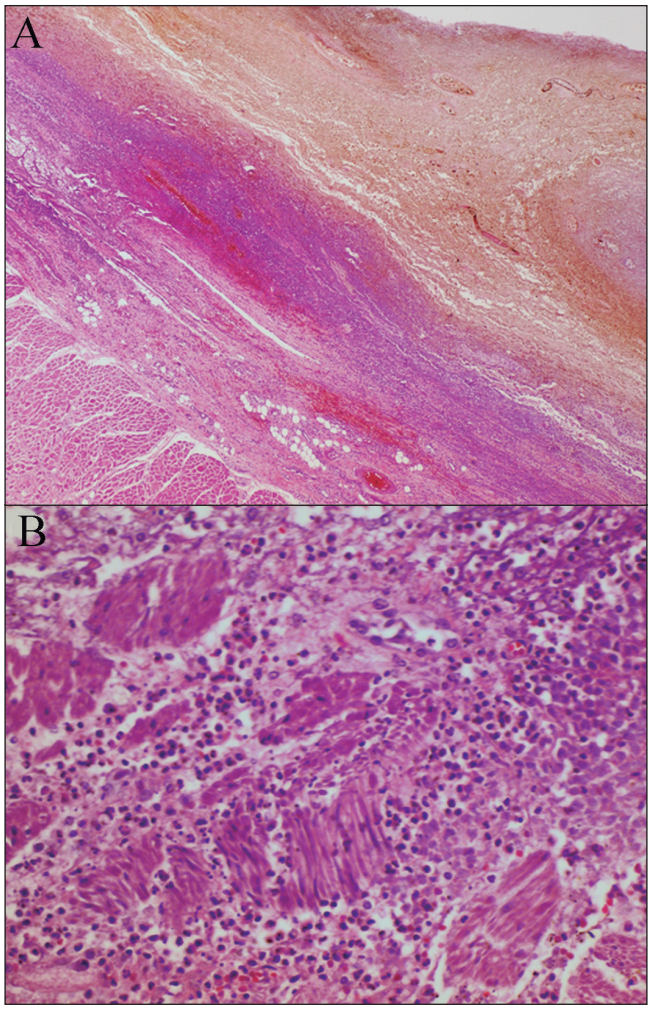

Fig. 2. A. Severe necrosis of the mucosa and submucosa with absence of viable squamous epithelium and an abundance of necrotic debris (HE 2X). B. Marked acute inflammatory infiltrates and partial destruction of muscle fibres (HE 40X).

ulus and the tendency for rapid healing could explain this discrepancy (2,3). The aetiology is multifactorial, with predisposing factors including male sex, advanced age, malnutrition, alcoholism, cardiovascular diseases, and chronic kidney disease. Hypoperfusion, decreased mucosal defensive barrier, and the presence of gastro-oesophageal reflux have been proposed as physiopathologic mechanisms (4). Diabetic decompensation could be an important factor, particularly ketoacidosis, suggesting a relationship between the degree of proximal damage and of hyperglycaemia (5). The differential diagnosis is made with melanosis, pseudomelanosis, malignant melanoma, *acantosis nigricans*, charcoal deposits, and caustic poisoning (1). Our case is unusual for the diagnosis and for its extension, despite showing no signs of bleeding. The latter is especially relevant, given the exceptional anatomopathology report on this patient.

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