Chronic oesophagitis dissecans : a case report

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Abstract

Chronic oesophagitis dissecans is an often unrecognised cause of chronic dysphagia. It usually occurs in otherwise healthy patients and was first described as We report the case of a patient with chronic dysphagia due to chronic oesophagitis dissecans. (Acta gastroenterol. belg., **2006**, 69, **223-225**).

Introduction

Chronic oesophagitis dissecans (CED) is an often unrecognised cause of chronic dysphagia. CED usually occurs in otherwise healthy patients. It was first described by Ponsot Ph. in Gastrointestinal Endoscopy in 1997 as We report the case of a patient with chronic dysphagia due to CED.

Case report

A 62-year-old man presented with chronic dysphagia for fluids and solids since two years. He also reported a weight loss of 10 kilograms. There were no symptoms suggestive of reflux disease, no odynophagia and no history of skin lesions. His medical history was remarkable for peptic ulcer, chronic alcohol abuse, alcoholic encephalopathy, atopic dermatitis, asthma and TUR prostate. There was no history of ingestion of any corrosive substance. As daily medication he reported the use of diclofenac, iron substitution, lormetazepam and ranitidine.

Clinical examination was unremarkable. Laboratory tests including liver and kidney function, electrolytes, vitamin B12 and folate levels, were normal. There was a mild anemia (hematocrit = 35.2%) with lowered levels of serum iron and ferritin. There was no eosinophilia. The levels of serum IgE are unknown.

Because of persistent complaints of dysphagia the patient was referred for esophageal barium contrast radiography. The radiography showed an incomplete stenosis of the mid-oesophagus over a length of 7 cm (Fig. 3). At endoscopy, an incomplete stenosis was seen from 23 to 30 cm from the incisor teeth. The oesophageal mucosa had a pearly, whitish pseudomembranous appearance with vertical and circumferential fissures (Fig. 1). These membranes were peeled off the underlying mucosa during passage of the endoscope. The underlying mucosa was very friable and started to bleed immediately (Fig. 2). From 30 cm to the gastro-oesophageal junction (38 cm) there was a normal mucosa. Endoscopy of the stomach, bulbus and duodenum were normal. A dilatation with Savary bougies was performed at the end of the endoscopic examination. Histopathological evaluation of esophageal biopsy specimens revealed a hyperplastic basal layer of the epithelium with intraepithelial cleavages and few inflammatory cells, consistent with a diagnosis of oesophagitis dissecans. Control radiography after dilatation showed a normal luminal diameter and a normal passage of barium through the cardia.

Computed tomography of the chest showed a thickening of the distal esophageal wall with stenosis. There were no adenopathies or other signs of malignancy (Fig. 4).

The daily medications, such as diclofenac, iron substitution, lormetazepam and ranitidine, were discontinued and the patient was prescribed pantoprazole (40 mg daily). A program with follow-up endoscopy at six month intervals was planned.

Discussion

Oesophagitis dissecans superficialis is a rare cause of acute dysphagia that occurs in previously healthy patients who suffer trauma of their esophagus (1-6). This trauma has been described in response to chemical (alendronate (2), chemical irritants), physical (nasogastric intubation, Mallory Weiss tear) and thermal injury (hot beverages). Oesophagitis dissecans superficialis has also been reported as an idiopathic disease and in patients with cutaneous disorders (pemphigus vulgaris) or with chronic renal failure (5).

Chronic oesophagitis dissecans (CED) usually occurs in otherwise healthy patients.

Our patient did not have any skin lesions or evidence of a thymoma on computed tomography of the chest.

Patients with CED mostly present with a long-standing history of dysphagia for fluids and solids. The endoscopic lesions are characterized by areas of whitish, pearled mucosa that form incomplete rings or transverse streaks. Upon passage of the endoscope, detachment of

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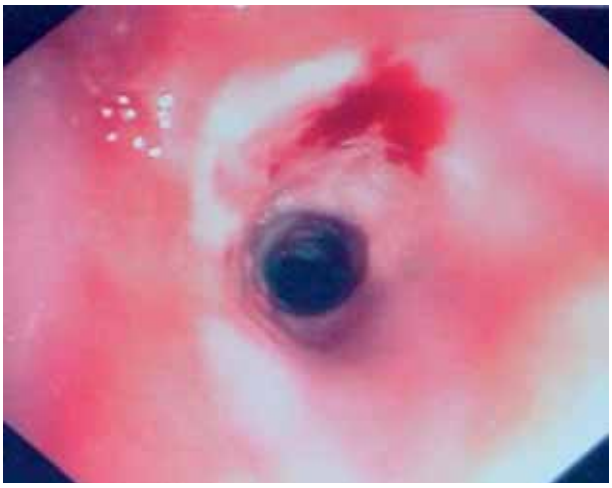

Fig. 1. — At endoscopy we noticed an incomplete stenosis of the esophagus between 23 and 30 cm from the incisor teeth. The esophageal mucosa had a pearly, whitish pseudomembranous appearance with vertical and circumferential fissures.

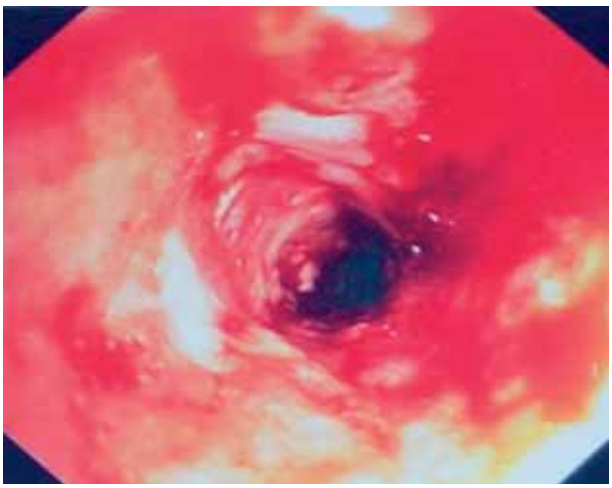

Fig. 2. — These membranes were peeled off the underlying mucosa on passage of the endoscope. The underlying mucosa was very friable and started to bleed immediately.

the mucosal fragments occurs, leaving a denuded esophageal wall which bleeds readily (1-4-5). The site and intensity of the lesions are variable.

Histopathological examination usually shows the presence of intraepithelial cleavages, located between the basal layer of the oesophageal epithelium and the lamina propria, or within the basal layers of the epithelium in the absence of an inflammatory infiltrate (1).

The etiology of CED has not been established. The microscopic lesions are reminiscent of pemphigoid type lesions, and therefore may reflect defects of cell adhesion mechanisms.

Similar to other desquamating esophageal disorders, CED usually results in stricture formation (4). As CED has been related to toxicological influences, it is appropriate to discontinue all potentially toxic

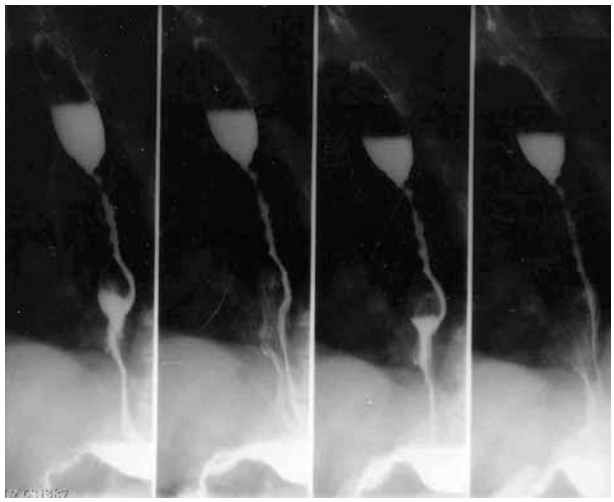

Fig. 3. — Contrast radiography with barium showed an incomplete stenosis of the mid-oesophagus with a length of 7 cm.

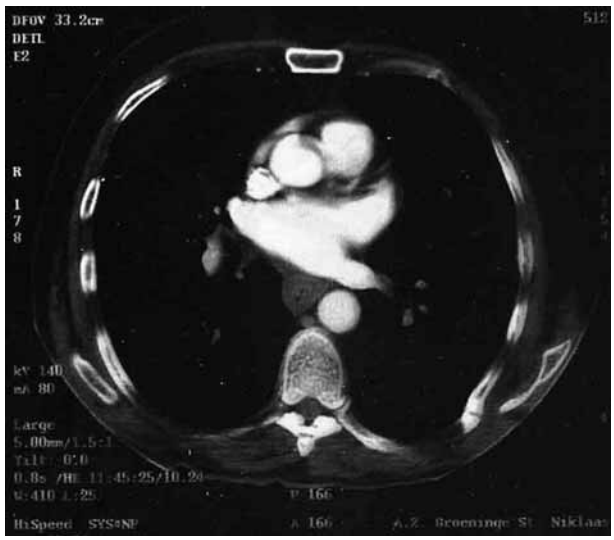

Fig. 4. — Computed tomography of the chest showed a thickening of the distal oesophageal wall with stenosis. There were no adenopathies or other signs of malignancy.

Table 1. — **Differential diagnosis of Oesophagitis dissecans superficialis**

Chemical injury	Alendronate Chemical irritants
Thermal injury	Hot beverages
Physical injury	Nasogastric intubation Mallory Weiss tear
Bullous skin lesions	Pemphigus vulgaris Bullous pemphigoid Dystrophic epidermolysis bullosa
Idiopathic	

medication. The mechanism of toxicity, which does not involve an inflammatory infiltrate, as well as the principal causative agents, remains largely unknown. None of the medications that our patient was taking has been established as a potential cause of CED. Treatment with diclofenac can cause a severe desquamative oesophagitis, but it usually recovers immediately after discontinuing the drug.

Treatment with acid secretion inhibitors is usually started, although a role for acid suppression in controlling these lesions has not been established. A survey of the literature fails to report other specific therapeutic strategies.

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