PICTORIAL Primary eosinophilic oesophagitis

A 56-year-old woman was admitted to our hospital as an emergency with a food bolus stuck in her oesophagus in September 2010. She had a history of hypertension and epilepsy; her medications included atenolol and ramipril. She had no history of allergy or asthma. She had had similar episodes in the past 2 years that resolved spontaneously. Since the

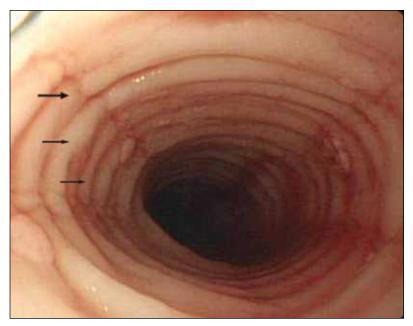


FIG 1. Oesophagogastroduodenoscopy demonstrating longitudinal furrows and concentric rings in oesophagus called 'trachealisation of oesophagus

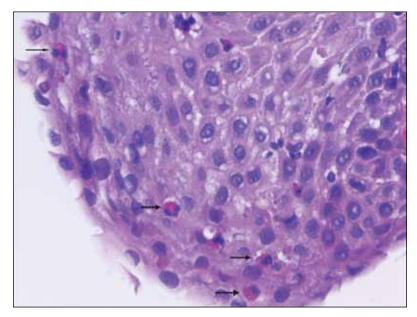


FIG 2. Oesophageal biopsy demonstrating eosinophilic infiltration of oesophageal mucosa

current episode was prolonged she presented to the emergency department. Routine blood results were normal apart from a slightly raised eosinophil count. After intravenous buscopan, her symptom abated. in-patient oesophagogastroduodenoscopy An (OGD) showed longitudinal furrows and concentric oesophagus rings also called 'trachealisation of oesophagus' (Fig 1). Biopsies taken from the oesophagus showed eosinophilic infiltration of the mucosa (Fig 2). Based on the typical history, peripheral eosinophilia, characteristic OGD and histology findings, a diagnosis of primary eosinophilic oesophagitis (PEO) was made. The patient was commenced on topical budesonide therapy and followed up in an out-patient gastroenterology clinic. She has remained stable with no further episodes of dysphagia for at least 3 months.

Primary eosinophilic oesophagitis is one of the three primary eosinophilic gastrointestinal disorders, the others being primary eosinophilic gastroenteritis and hypereosinophilic syndrome with gastrointestinal involvement. In 2007, a consensus meeting of the American Gastroenterological Association Institute and the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition recommended diagnostic criteria in PEO. Accordingly it was defined as a clinicopathological entity, combining clinical data on relevant symptoms, oesophageal biopsies with adequate histological findings (>15 eosinophils/high-power field) and exclusion of other diseases with overlapping features, especially gastro-oesophageal reflux disease.¹

The reported male-to-female ratio of PEO is about 4:1. It is found in all age-groups, peaks in early childhood and again between the ages of 20 to 40 years. Adults present with attacks of dysphagia for solid foods, which can last a few minutes to several hours, sometimes leading to prolonged episodes of food impaction. Between the episodes, patients are completely free of symptoms. What causes PEO is unclear; transient oesophageal contractions may be involved. Apart from OGD and oesophageal biopsy, biopsies should also be taken from the duodenum and stomach to rule out eosinophilic gastroenteritis.

Endoscopic signs of PEO are often unremarkable, may be misleading or even absent (10-25% in adults).² Positive oesophageal findings include vertical furrowing, strictures, white plaques, exudates, longitudinal shearing following passage of the endoscope in a fragile oesophagus, a narrowcalibre oesophagus and fixed or transient rings or oesophageal 'trachealisation'. Such findings are not pathognomonic, but a narrow oesophageal lumen, associated with concentric rings, with or without proximal stenosis of the oesophagus, is highly suggestive.³ Medical treatment relies on systemic and topical corticosteroids. The latter can be used for acute episodes and topical steroids for long-term disease control.⁴ Topical application relies on swallowed steroid administered through a metred-dose inhaler, used without a spacer to allow significant delivery to the oesophagus.

Regarding long-term implications, there is no

evidence of epithelial malignancy or premalignancy. The major complication of PEO is oesophageal remodelling and narrowing, but the risk of fibrosis remains to be defined.

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