EOSINOPHILIC OESOPHAGITIS

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- EOO is defined as a chronic, immune/antigen-mediated oesophageal disease, characterised clinically by symptoms related to oesophageal dysfunction and histologically by - eosinophil-predominant inflammation. However, there is over - lap with GORD-related eosinophilia and other pathologies that may also be associated with similar oesophageal eosino - philia, such as achalasia, hypereosinophilic syndrome, Crohn’s disease, coeliac disease, vasculitis, pemphigus , graft-versus-host disease and other connective tissue disorders. The incidence of EOO is increasing, ranging from 0.7/100 /uni00A0 000 to 10.7/100 /uni00A0 000 depending on the population studied. It is predominantly a disease among the white population in Western countries (white versus non-white ratio, 3:1). EOO is believed to commence with food antigen initia tion of cytokine-mediated signals that lead to eosinophilia, inflammation and subsequent remodelling by fibrosis. It is a progressive disease; thus, in infants or toddlers, it may present version and failure to thrive. In young with irritability , food a children vomiting and regurgitation predominate. In older children or early adolescents, it may present with heartburn and dyspeptic symptoms, while in adults dysphagia and food impaction become the most common symptoms. The peak age of presentation is around 20–30 years. There is a clear associa - tion with other atopic disorders such as asthma, atopic rhinitis or dermatitis and food allergies. Typical findings on endoscopy include the presence of rings, furrows, exudates, oedema, stricture , narrowing and ‘crepe paper mucosa’ (Figur e 66.36). Biopsies of the oesoph - agus show 15 or more eosinophils per high-power field (hpf). The eosinophilic infiltration is isolated to the oesophagus. Biopsies should be taken at a minimum of two separate levels of the oesophagus together with suspicious areas. Usually gas - and duodenal biopsies are also taken to exclude eosino - tric philia at these sites. Barium contrast study may demonstrate narrowing and assess the diameter of the oesophagus better than endoscopy . Treatment goals include reduction of oesophageal eosin - ophilia (to <15/hpf and preferably to <5/hpf), control of tion of endoscopic findings, although symptoms and normalisa these goals can only be completely achieved in a minority of patients. In children, the disease process is predominantly h as nausea, vomit - inflammatory , and associated symptoms suc - ing and abdominal pain are relieved with appropriate medical treatments. In adults, reduction in inflammation per se may e not relieve the fibrotic component (stricture and small-calibr oesophagus) and dilatation may be required. Topical corticosteroids are the mainstay medical treatment for EOO; swallowing topically acting corticosteroids such as

Figure 66.35 Contrast study showing a long undilatable stricture of the oesophagus secondary to caustic burn (a) . The patient underwent an oesophagogastrectomy and colonic interposition. Note that the oesophagus and the stomach were scarred (b). Figure 66.36 Endoscopic appearance of eosinophilic oesophagitis. The linear furrows and circular rings are evident.

budesonide or fluticasone is highly e ff ective in resolving symp toms. Systemic steroids should be avoided. PPIs are e ff ective in 40–50% of adults through blockage of cytokine release rather than acid suppression. Diet therapy is useful, eliminating glu ten, milk, soy , egg, nuts and seafood as the most likely antigens Endoscopy is used for diagnostic purposes, to monitor treatment progress and therapeutically to deal with strictures. Careful gradual dilatations should be performed, although perforation rates appear similar to other types of strictur

Figure 66.37 Open surgery was required to extract a broken denture stuck in the cervical oesophagus. The oesophagus was repaired in two layers.

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