Eosinophilic oesophagitis

No longer a rare cause of dysphagia

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Eosinophilic oesophagitis has transformed from a rarely seen, case reportable diagnosis to an important cause of dysphagia and food bolus impaction in adults.

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REMEMBER

- Eosinophilic oesophagitis (EoE) is a chronic immune-mediated disease characterised clinically by symptoms related to oesophageal dysfunction and histologically by eosinophil-predominant inflammation.
- EoE affects both children and adults and is more common in men than in women. In adults, the median age of onset is between 30 and 40 years.
- The incidence of EoE appears to be increasing, which may be related to increased recognition. It is found in 50% of patients presenting with food impaction and 6 to 15% of patients undergoing an endoscopy for dysphagia.[1] A recent Australian study found that the increase in prevalence of food bolus obstruction over the past 15 years was associated with an increase in the diagnosis of EoE.[2]
- The pathogenesis of EoE is not completely understood. It is thought to relate to certain environmental antigens that trigger an immune response in genetically predisposed individuals, leading to the infiltration of the oesophageal mucosa by eosinophils.[3] The majority of affected patients have a history of atopy, including asthma, eczema and allergies to food or environmental agents.
- Clinical manifestations of EoE depend on the age of the patient. Feeding disorders, vomiting, abdominal pain and failure to thrive are the common symptoms of EoE in children, whereas the hallmark presentation in adults is dysphagia or food bolus impaction. The less common symptoms include heartburn or reflux symptoms that are refractory to proton pump inhibitor (PPI) therapy.

ASSESSMENT

- In patients with suspected EoE, dysphagia or food bolus impaction, an upper endoscopy with oesophageal biopsies

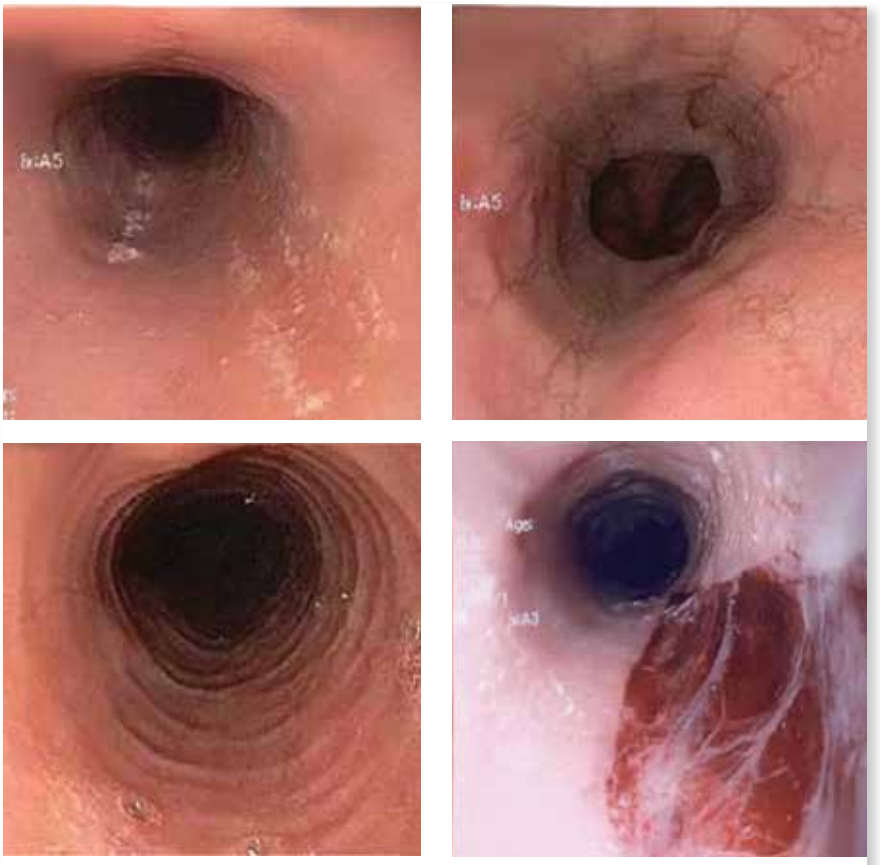

Figure. Examples of endoscopic features of eosinophilic oesophagitis, including the whitish papules (a, top left), linear furrows (b, top right), mucosa rings (c, bottom left) and mucosal laceration with nontraumatic passage of an endoscope (d, bottom right).

is required to confirm the diagnosis of EoE. The following are diagnostic criteria for EoE.[3]

- Endoscopic findings in patients with EoE include fixed oesophageal rings, linear furrows, whitish papules, strictures, oedema, small calibre oesophagus and mucosal lacerations induced by passage of the endoscope (Figures a to d).
- The presence of at least 15 eosinophils per high power field in oesophageal biopsy specimens is diagnostic of EoE. The other histological features of EoE include eosinophil microabscesses, subepithelial and lamina propria fibrosis and inflammation, and basal cell hyperplasia.
- The diagnosis of EoE should include the exclusion of other causes of oesophageal eosinophilia, including eosinophilic gastrointestinal disease, PPI-responsive oesophageal eosinophilia, coeliac disease, Crohn's disease, parasite infection, hypereosinophilic syndrome, drug hypersensitivity, vasculitis, pemphigus, connective tissue diseases and graft versus host disease.

- A response to treatment supports, but is not required for, diagnosis of EoE.
- More than half of patients with EoE have elevated serum IgE levels.
- The use of skin-prick tests and patch tests to identify food allergies in patients with EoE is not useful. These tests predict only 13% of the associated food detected by an elimination diet.[3]

MANAGEMENT

- In children with EoE, a six-food elimination diet is often considered as the first-line therapy because it has been shown to be effective and is 'drug-free'.[4,5] The foods eliminated are milk, soy, wheat, nuts, eggs and fish/shellfish. To improve compliance, however, dietary therapy should be tailored based on the available resources and preferences of the patient as well as the family.
- If dietary therapy fails, swallowed topical corticosteroids (off-label use; e.g. fluticasone propionate 250 μg/dose, two puffs twice daily in children and three puffs twice daily in adults; or viscous budesonide liquid prepared by a specialist pharmacist comprising 1 mg/2 mL of aqueous budesonide with 5 g of sucralose, twice daily) can be used alone or in conjunction with a PPI (e.g. pantoprazole 40 mg/day; off-label use).
- Although topical corticosteroid alone or in combination with PPI therapy has been used as first-line pharmacological therapy for the treatment of EoE in adults, the six-food elimination diet has been increasingly adopted as it has been recently shown to be effective in about 80% of patients.[5]
- Systemic corticosteroids (e.g. budesonide 2 mg/day or prednisolone 2 mg/kg/day to a maximum dose of 60 mg/day) can be useful when topical corticosteroids are not effective or in patients who require rapid improvement in symptoms.[3,4,6]
- In patients whose symptoms persist despite medical or dietary therapy, careful oesophageal dilation is effective, especially in those with obvious fibrotic

oesophageal strictures.[3,4]

- Data on the use of mast cell stabilisers, leukotriene inhibitors or immunomodulators for the treatment of patients with EoE are limited and currently these biologic therapies remain experimental.[6]
- Maintenance therapy with either dietary manipulation or drugs (swallowed corticosteroid and/or PPI) should be considered in all patients with EoE because the recurrence of symptoms is high when treatments are discontinued.[3,4]
- If EoE is left untreated then chronic inflammation can lead to fibrosis and the development of oesophageal strictures. There is no evidence to suggest an increased risk of malignancy in patients with EoE.
- GPs play an important role in co-ordinating the long-term management of patients with EoE, especially in monitoring compliance with either dietary or pharmacological treatment.

CONCLUSIONS

- The incidence of EoE appears to be increasing in both children and adults, which may be at least partly a result of increased recognition.
- Unexplained dysphagia or recurrent food bolus obstructions should raise suspicion for the diagnosis of EoE, and oesophageal biopsy should be performed.
- In all patients, dietary therapy should be the first-line treatment and if that fails, pharmacotherapy and/or oesophageal dilation can be used. MT

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COMPETING INTERESTS: None.