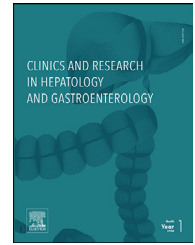




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LETTER TO THE EDITOR

Eosinophilic oesophagitis: Significantly increasing prevalence in a tertiary London paediatric gastro-intestinal and allergy department



To the editor,

We are amid a gastrointestinal allergy epidemic, with a condition once considered rare becoming a common diagnosis within our department. Eosinophilic oesophagitis (EoE) is a chronic inflammatory condition characterised by the presence of intraepithelial eosinophils in oesophageal biopsies and symptoms of oesophageal dysfunction.[1]

This is a retrospective review of all patients diagnosed with EoE between January 2010 to April 2021 within our Paediatric Gastro-Intestinal and Allergy departments. Using the HRA decision tool and 'Defining Research Leaflet' which provides guidance on what constitutes a study to be either research, audit or service evaluation. It was confirmed that this project did not appear to be generating generalisable new knowledge, as per the definition of research and was determined to be an audit. After the initial search of our electronic database, data was extracted including patient characteristics, comorbidities, allergy profile, histology results and management. The diagnosis of EoE is defined as those with more than 15 eosinophils per high power field (HPF) on oesophageal biopsy and symptoms of oesophageal dysfunction.[2]

70 children were identified with a diagnosis of EoE between 2010–2021, with mean age of diagnosis 8 years (range 0 – 17). Between 2010 and 2021 the mean age of diagnosis has increased from 6 years to 9 years. Patients who were atopic on average were 2 years older than those who were non-atopic (mean 9 versus 7 years of age). 50 (71%) were male with no sex difference in age of diagnosis. 31 (46%) of patients were White British, 14 (21%) Asian, 8 (12%) Other, 8 (12%) Black, 6 (7%) Mixed and 2 (3%) were not recorded.

Our diagnoses of EoE have increased over this period (Fig. 1), from 0.08 diagnoses per month in 2010 to 2.25 diagnoses per month in 2021. This has been reflected in recent literature, with one paper noting EoE is now the most common cause of gastrointestinal morbidity in both children and adults within the United States. [1]

In keeping with other centres, we found that the clinical presentation in our patients varied according to age.

Younger children presenting with non-specific symptoms including feeding difficulties, while adolescents present with dysphagia or food impaction. On the initial referral letter the most common presenting symptoms across all ages include food impaction, abdominal pain, and vomiting.

53 (76%) of our patients were atopic (history of asthma, eczema, food allergies and/or allergic rhinitis) and 17 (24%) non-atopic. Of the 17 non atopic individuals, 3 (18%) had a history/diagnosis of Oesophageal Atresia and/or Tracheo-Oesophageal Fistula, and 2 (12%) had a diagnosis of coeliac disease. Of the 53 atopic individuals, 25 (47%) had a history of asthma, 28 (53%) patients have eczema, and 31 (58%) patients have allergic rhinitis. 9 (13%) children had a recorded diagnosis of Pollen Food Syndrome.

IgE mediated allergy to food was found in 32 (46%) of patient (Table 1). Of these patients, 28 (88%) were allergic to 2 or more food groups.

The high prevalence of legume allergy observed in our EoE cohort is consistent with what has been reported in personal communications by colleagues in other centres but has not been noted in previous literature and contrasts with a 4% prevalence of legume allergy in a cohort of 5080 0–5-year-old food allergic patients in North Wales and North England.[3] However the demographic of the latter is very different to our population.

Treatment of EoE consists of dietary, pharmacological, and endoscopic interventions. In our patients, the initial treatment plan included a proton pump inhibitor (PPI) for 53 (76%) patients, of which 40 (75%) used a low dose (less than 40mg per day), most frequently omeprazole 20mg once daily (in 32 patients, 60%). Food exclusions, in addition to foods avoided due to IgE mediated allergies, occurred in 11 (16%) of the initial treatment plans, most commonly wheat/gluten, dairy, soya, and egg. Only 3 (4%) of patients were initially prescribed a steroid.

There is limited peer reviewed evidence surrounding long term maintenance regimens for EoE, as disease frequently recurs in patients who discontinue treatment.[4] Whilst for many, maintenance treatment is needed, for some, the disease will resolve, and maintenance can be discontinued.

Our management of EoE has evolved over time, our current patients with EoE (n=46) are less likely to be treated with a PPI, but if they are, it is more likely with a higher dose. The most common maintenance treatment remains omeprazole 20mg once daily currently prescribed for 27 (59%) patients who are managed with a PPI. Food exclusion

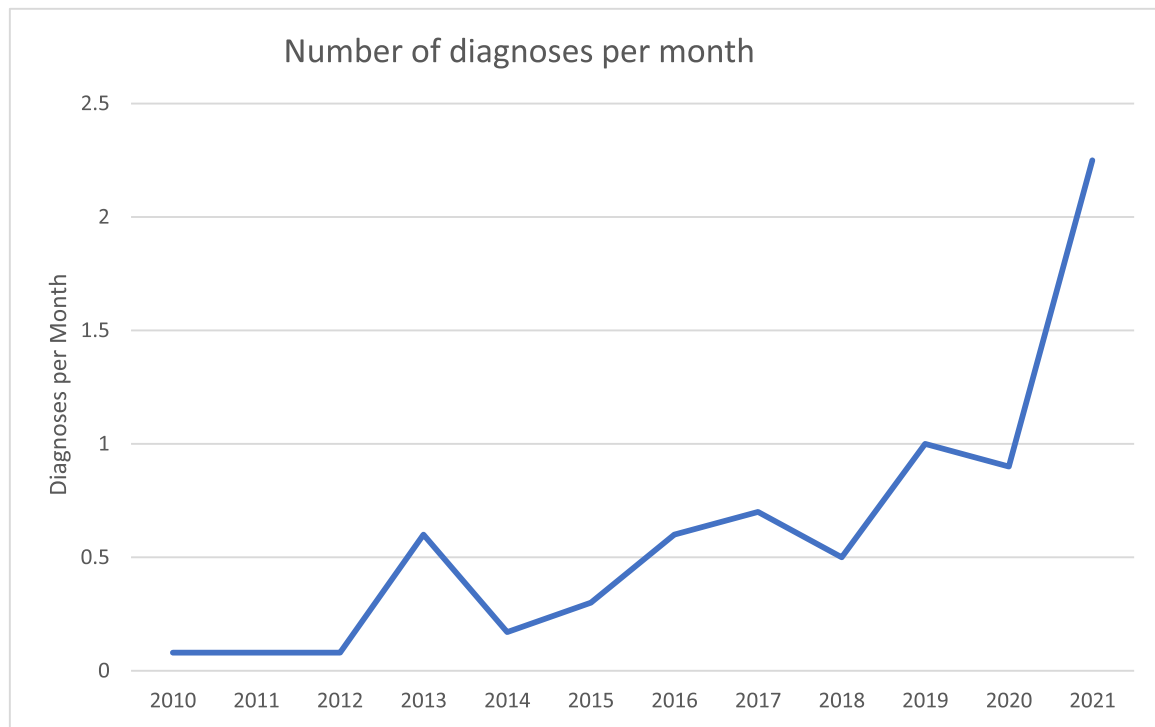


Fig. 1 Number of diagnoses of EoE per month Graph illustrating the number of EoE diagnoses averaged per month between 2010 and 2021.

is part of the management for 34 (49%) patients, an increase of 33%. Additionally, 13 (19%) of patients are currently prescribed a steroid as part of their treatment. Only 3 (4%) patients are noted to no longer require treatment for their EoE.

Within our dataset 16 (23%) of patients are in complete remission, as defined by both absence of symptoms and EoE counts <15 /HPF.² Although 23% may appear low, this reflects our pragmatic approach in tailoring individual management plans in partnership with our patients and carers; in those with no symptoms of oesophageal dysfunction, we do not necessarily aim for complete endoscopic and histological remission. This is to avoid unnecessary repeat endoscopies and multiplying risk (including that from multiple exposures to general anaesthesia which is required for

paediatric endoscopy in our unit). Over the past 3 years, we have carried out around 39 repeat endoscopies, on average once every 6 months (the ranging from 8 weeks to 12 months).

One complication of untreated EoE is stricture formation. [5] Only one patient has had a stricture, and this had already occurred at initial. None of our patients have gone on to develop strictures, suggesting that most of our cohort may have an inflammatory phenotype, and that a fibro-stenosing phenotype is rare. [6] EoE was initially described and historically managed by gastroenterologists and gastroenterological surgeons, as it is diagnosed by endoscopy. [7] As such, the treatment paradigm has been modelled on inflammatory bowel disease, where disease progression is the norm for untreated disease. However, there is increasing evidence to suggest that a more appropriate model is atopic dermatitis. [8] There are genetic similarities between eczema and EoE, both being diseases of epithelial derived mucosa, and a stricturing phenotype could be analogous to lichenified eczema. Developments in our understanding of the natural history of EoE will continue to shape treatment plans.

In this analysis we included all patients diagnosed with EoE between 2010 and 2021 in our centre. However, we recognise that our case identification has improved, potentially influencing our results. We are now collecting more comprehensive data, with a particular focus around treatment outcomes, to further assess this aspect of EoE.

Our understanding of various aspects of EoE, including the natural history and long-term management are incomplete. Long-term outcome-based research is required as well as exploring non-invasive markers for disease progression to avoid repeated endoscopy.

Table 1 Food allergies in EoE patient dataset

IgE mediated food allergy	Number of children (%) N= 32
Milk	12 (40%)
Egg	18 (56%)
Nuts	20 (67%)
Fish	6 (19%)
Wheat	11 (34%)
Sesame	6 (19%)
Legumes	14 (44%)
Shellfish	1 (0%)
Pollen Food Syndrome	9 (28%)

Contribution to authorship

Conceptualisation: RW, AO, EB; Methodology: RW, AO, EB; Data collection: EB, RW; Statistical analysis: EB; Data interpretation: EB, RW, AO; Manuscript draft: EB; Manuscript review & editing: EB, RW, AO, KM, RK, MP.

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Declaration of competing interest

No conflicts of interest to declare.

Supplementary materials

Supplementary material associated with this article can be found in the online version at [doi:10.1016/j.clinre.2022.101958](https://doi.org/10.1016/j.clinre.2022.101958).

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