EOSINOPHILIC ESOPHAGITIS (EOE)

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Conflict of interest statement

The author declares that there are no conflicts of interest.

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Introduction

Eosinophilic esophagitis (EoE) is a chronic inflammatory, immune-mediated disease of the esophagus that was first described in 1993 as a distinct clinical and pathological condition [1]. EoE is now recognized as one of the most prevalent inflammatory diseases of the esophagus and as an important cause of dysphagia and food bolus obstruction, particularly in adults [2, 3].
EoE can occur at any age, including infancy and childhood. Incidence rises during adolescence and peaks in early adulthood. Men are affected more often than women [3, 4]. If untreated, persistent symptoms and progressive inflammation can lead to esophageal remodeling, fibrosis, strictures and functional impairment. EoE can substantially impair health-related quality of life and psychosocial functioning. It may also lead to complications such as food bolus obstruction and, rarely, esophageal perforation [2–4].

This brochure focuses primarily on the recognition, diagnosis and management of EoE in adults. EoE also occurs in children, in whom its presentation, diagnostic assessment and treatment require age-specific consideration; key pediatric aspects are summarized in Supplement.
Licensed indications, age ranges and available formulations vary between countries. Please refer to the approved product information in your country.

Current treatment options for adult EoE in European and British evidence-based treatment guidelines include topical (swallowed) corticosteroids, proton pump inhibitors (PPIs), empiric food elimination diets and dupilumab. Treatment should be individualized according to the patient's characteristics and preferences and the resources available. Treatment endpoints include improvement in symptoms, endoscopic and histological findings and EoE-specific quality of life; long-term treatment is usually required to maintain remission and reduce the risk of relapse [2, 3, 5].

1 Definition of EoE, differentiating between EoE and GERD

EoE is characterized clinically by symptoms of esophageal dysfunction and histologically by eosinophil-predominant inflammation. A conclusive diagnosis requires symptoms of esophageal dysfunction and a peak count of ≥15 eosinophils per high-power field (eos/hpf) in at least one esophageal biopsy, in the absence of other causes of esophageal eosinophilia [2–4, 6, 7]. When EoE is suspected, local and systemic alternative causes of esophageal eosinophilia should be considered and excluded (Table 1.1). Findings should be interpreted in an overall clinical and endoscopic context [3, 4].

Differential diagnosis
Gastroesophageal reflux disease (GERD)
Eosinophilic gastritis/gastroenteritis/colitis with esophageal involvement
Achalasia or other primary motility disturbances of the esophagus
Hypereosinophilic syndrome
Crohn's disease with esophageal involvement
Pill esophagitis
Drug hypersensitivity reactions
Infections (fungal, viral, parasitic)
Connective tissue disorders
Vasculitis
Celiac disease

Table 1.1: Differential diagnoses of esophageal eosinophilia. Modified from [3, 4]

The most common condition to consider in the differential diagnosis of EoE is GERD. Historically, EoE and GERD were considered mutually exclusive, and an abnormal pH study was taken to indicate GERD (Table 1.2) [3, 4]. Later, patients with features of EoE who responded to PPI treatment were classified as having PPI-responsive esophageal eosinophilia (PPI-REE), irrespective of pH findings. Subsequent evidence has shown that patients classified as having PPI-REE and those with EoE were essentially indistinguishable in terms of clinical presentation, pH findings,

endoscopic and histological findings and genetic features [8]. The term PPI-REE is therefore no longer used – response to a PPI does not exclude EoE, and a PPI trial is not a diagnostic criterion [3, 4, 9].

Current evidence recognizes EoE and GERD as distinct disorders that may coexist and interact [4]. Evidence suggests complex, bidirectional interactions between them [10]. For example, GERD may disrupt mucosal integrity, increasing transepithelial allergen permeability and subsequent immune activation, and may thereby contribute to the pathogenesis of EoE [11]. Patients with EoE may also have increased acid sensitivity compared with healthy controls, potentially as a result of impaired esophageal mucosal integrity [12]. Conversely, structural and functional esophageal abnormalities associated with EoE may promote gastroesophageal reflux [13].

	EoE	GERD
Age	Children and adults	Adults > children
Sex	Male predominance	Men = women
Dominant symptom	Dysphagia, food bolus obstruction	Heartburn, regurgitation
Endoscopic findings	Normal < 10%, edema, exudates, furrows, rings, strictures, microabscesses	Normal 70–80%, erosions, ulcers, strictures, Barrett's esophagus
Histology	Eosinophils (> 15 eos/hpf), mast cells	Neutrophils, lymphocytes, low eosinophil count (< 5–10 eos/hpf)
Acid reflux (esophageal pH monitoring)	Rare	Common
Etiology	Food allergens, airborne allergens	Acid/non-acid reflux
Immune response/ cytokines	T helper 2 (T_H2) Eotaxin-3, IL-5, IL-13	T helper 1 (T_H1) IL-8, MCP-1, RANTES

Table 1.2: Comparison of EoE and GERD. Modified from [3, 4].

2 Epidemiology and risk factors

Since it was first described as a distinct clinical entity in the early 1990s [1], EoE has moved from being regarded as a rare condition reported in isolated cases to one of the most commonly recognized inflammatory disorders of the esophagus [3]. EoE may occur at any age; incidence rises with age and peaks in early adulthood [3, 4, 14]. Men have an approximately threefold higher risk of developing EoE than women [2–4, 15].
Registry-based studies from Switzerland, Denmark and the Netherlands have shown an approximately 20-fold increase in the incidence of EoE since the mid-1990s, outpacing the more modest increase in endoscopy and esophageal biopsy rates over the same period [16–18]. A 2019 meta-analysis of 29 epidemiological studies reported pooled incidence rates of 7.7 per 100,000 person-years in adults and 6.6 per 100,000 person-years in children [19]. The pooled prevalence was 34.2 per 100,000 population and was higher in adults than in children (42.2 vs 34.4 per 100,000). Across the periods studied, published prevalence estimates increased approximately fourfold, from 15.4 to 63.2 per 100,000 ($p = 0.011$) [19]. A 2023 meta-analysis of 40 studies, including 147,668 patients from 15 countries across five continents, confirmed the global rise [15]. The pooled incidence was 5.31 per 100,000 person-years and was higher in high-income countries, in men and in North America than in Europe or Asia. The pooled global prevalence was 40.04 per 100,000 inhabitants and increased significantly over the study period, 1976–2022 [15]. The rise in incidence and prevalence is believed to reflect both increased recognition of EoE and a true increase in disease frequency [3, 4].
Across population-based studies and unselected endoscopy cohorts, the prevalence of EoE has ranged from 0.4% to 6.5% [20–22]. Among adults undergoing endoscopy primarily for dysphagia, prevalence ranged from 12% to 23% [2, 14]. A meta-analysis of 14 studies of patients presenting with food bolus obstruction found EoE in 54% of those from whom esophageal biopsies were obtained [23].

Atopic conditions often precede EoE and are common comorbidities [4, 24]. Allergic rhinitis, asthma, atopic dermatitis and IgE-mediated food allergy occur more frequently in patients with EoE than in the general population [2–4, 25]. EoE is a type 2 immune-mediated disease – food allergens can trigger and maintain esophageal inflammation, while aeroallergens may contribute in some patients [4, 26]. Cases of EoE developing during oral or sublingual allergen immunotherapy have been described [27, 28]. A 2014 systematic review estimated a prevalence of 2.7% after oral immunotherapy [29]; however, the relative risk remains uncertain [2].

Familial aggregation and genetic susceptibility are well recognized in EoE [3, 4, 30, 31]. Genomic studies have identified EoE susceptibility loci, some of which overlap with loci implicated in other atopic diseases, including thymic stromal lymphopoietin (TSLP), eotaxin-3 (CCL26), filaggrin (FLG), desmoglein-1 (DSG1) and calpain-14 (CAPN14) [4, 32, 33].

3 Natural course, disease progression, complications

EoE is a chronic inflammatory and potentially progressive disease of the esophagus. If untreated, persistent inflammation can lead to esophageal remodeling, fibrosis and stricture formation (Fig. 3.1) [2–4, 14, 34]. Three retrospective cohort studies have consistently demonstrated that, as the interval between symptom onset and diagnosis (the 'diagnostic delay') increases, the rates of endoscopic signs of fibrosis and esophageal strictures rise significantly (Fig. 3.2) [35–37]. A Dutch cohort study showed that the rate of acute food bolus obstruction increased significantly with diagnostic delay (Fig. 3.3) [36]. Manometric studies have also shown that longer disease duration is associated with greater impairment of esophageal motility [13].

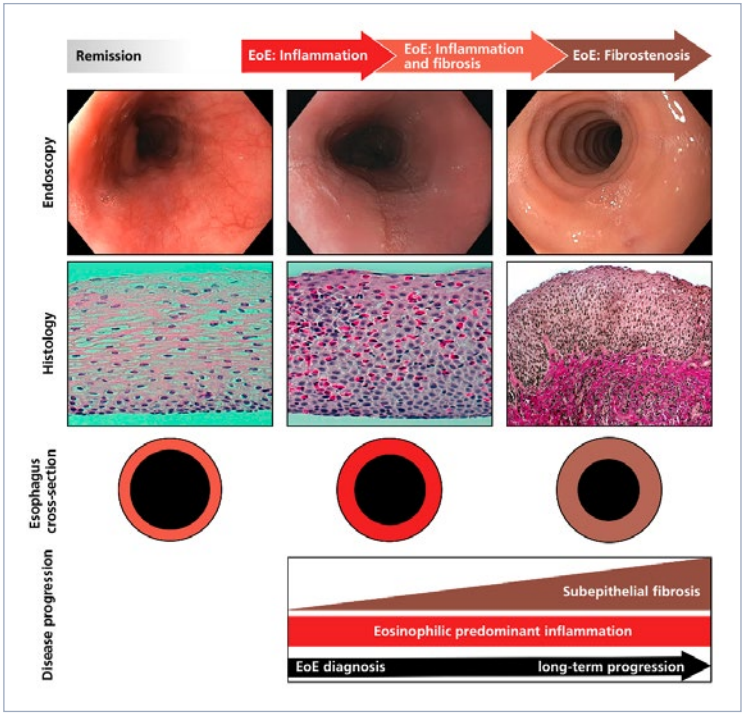


Figure 3.1: Comparison of a normal esophagus, an esophagus with active (inflammatory) EoE and an esophagus with a progressive fibrostenotic phenotype. Modified from [34].

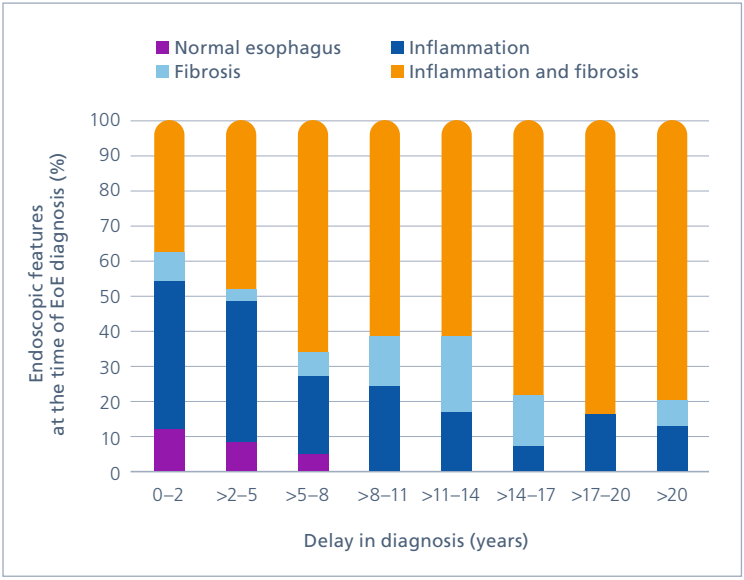


Figure 3.2: Endoscopic progression of EoE over time without treatment. Modified from [35–37].

Despite better recognition and understanding of EoE, diagnostic delay remains a relevant issue. A 2022 analysis of the Swiss EoE register found no significant improvement in diagnostic delay between 1989 and 2021, despite the publication of international consensus recommendations during this period; the delay remained approximately four years [38]. In contrast, a European registry study of more than 1,100 patients showed a gradual and significant improvement in diagnostic delay over time, from 12.7 years before 2007 to 0.7 years after 2017. The proportion of patients with stricturing disease at diagnosis also decreased, from 41% to 16% [39].

Among patients with confirmed EoE who had a gap in care of at least two years, disease progression was observed, with significant increases in esophageal fibrosis, stricture rate and the need for endoscopic dilatation. The duration of the gap in care correlated significantly with the esophageal stricture rate (Fig. 3.4) [40].

There is no evidence that EoE is a premalignant condition, although its relationship with Barrett's esophagus and esophageal cancer remains uncertain [2, 4, 14].

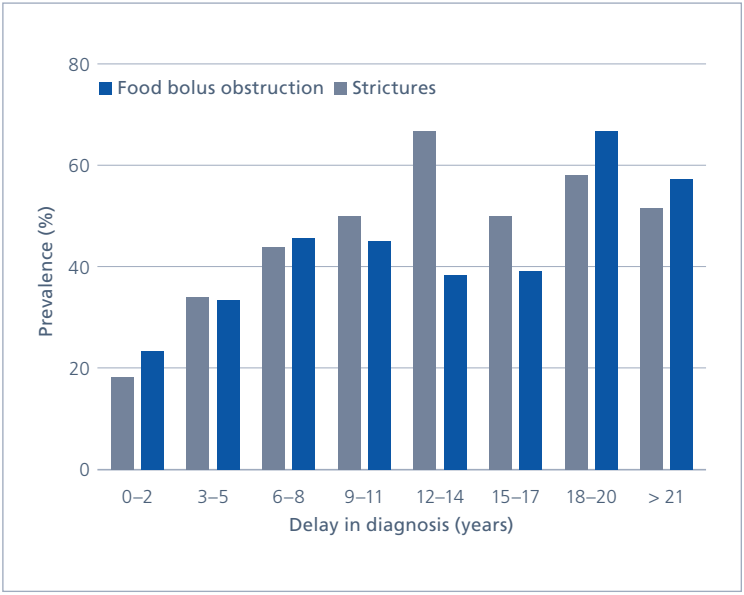


Figure 3.3: Rate of strictures and acute food bolus obstruction according to the duration of diagnostic delay. Modified from [36].

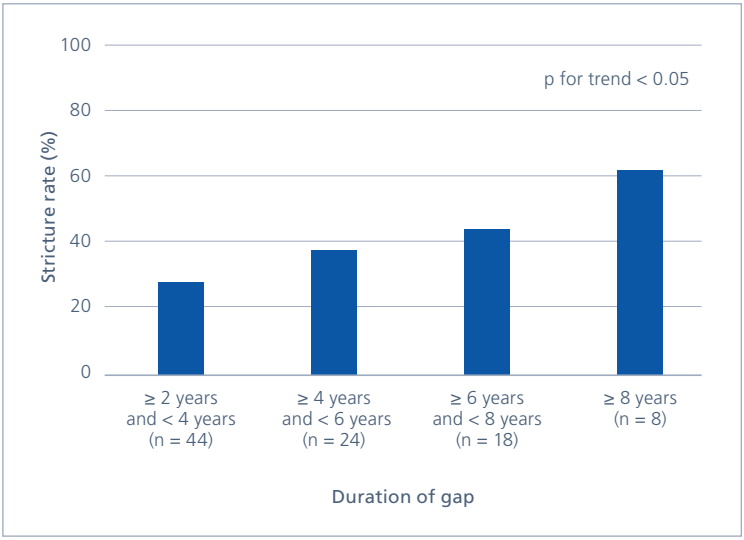


Figure 3.4: A gap in care is associated with progression of fibrosis in patients with EoE. Modified from [40].

Esophageal perforation is a rare but serious complication. A 2020 systematic literature review described 76 esophageal perforations in 70 patients (Table 3.1) [41]. In two-thirds of these cases, EoE had not been diagnosed at the time of esophageal perforation; in the remaining cases, treatment had either not started or was insufficient. In 41 cases, perforation occurred spontaneously in association with acute food bolus obstruction. In the other cases, perforation occurred during an endoscopic procedure (e.g. food bolus removal, dilatation or biopsy). Two-thirds of patients were managed conservatively, including esophageal stenting in 12 cases. One-third required surgery; half of these patients underwent thoracotomy and suturing, and 20% underwent esophageal resection [41].

The true frequency of perforation may be underestimated – a review of US hospitalizations for EoE identified esophageal perforation in 173 patients [42]. Other severe complications associated with acute food bolus obstruction have required complex endoscopic treatment or emergency esophageal resection [43–45].

The 2022 joint consensus guidelines published by the British Society of Gastroenterology (BSG) and British Society of Paediatric Gastroenterology, Hepatology and Nutrition (BSPGHAN) recommend a CT contrast study to assess the extent of extravasation, with conservative and multidisciplinary management of limited extravasation [3].

Perforations	n = 76
Spontaneous occurrence during acute food bolus obstruction	41
Endoscopic removal of food bolus obstruction	7
Dilatation	8 (balloon 6)
Biopsy Poorly tolerated endoscopy Other causes (e.g. strictures, endoscope passage, transesophageal echocardiography)	 3 3 14
Management	n = 76
Conservative (including stenting, n = 12)	51 (67%)
Surgical management	25 (33%)

Table 3.1: Esophageal perforation in patients with EoE. Modified from [41].

4 Disease presentation

In adults, the principal presenting symptoms of EoE are dysphagia and food bolus obstruction [2–4, 46–48]. Retrosternal or chest pain, heartburn and regurgitation are less common and, when present, may accompany dysphagia (Table 4.1) [2–4, 47]. Because onset may be insidious and symptoms may be masked by adaptive eating behaviors, EoE should be considered in adults presenting with dysphagia or food bolus obstruction [3].

Rarely, EoE may first present with a severe complication, such as esophageal perforation [3, 49]. Adults with EoE often develop adaptive eating behaviors that can mask symptom severity. In an observational study, patients with active EoE chewed more frequently than healthy controls while consuming a standard meal, drank more liquids and took longer to finish the meal [50]. Accordingly, the medical history should comprise any swallowing difficulty, avoidance of difficult textures, prolonged chewing, the use of liquids to aid swallowing and prolonged mealtimes [3, 49, 50].

In clinical trials, validated patient-reported outcome measures are used to assess symptoms. These include the Eosinophilic Esophagitis Activity Index patient-reported outcome (EEsAI PRO), Dysphagia Symptom Questionnaire (DSQ) and Mayo Dysphagia Questionnaire (MDQ) [51]. Their complexity or limited availability may restrict routine use. In clinical practice, simple visual analog or numerical rating scales (0–10) may support dysphagia assessment and correlate with validated measures [52]. However, symptoms do not correlate reliably with histological activity and should not be used alone to assess disease activity or treatment response [2–4].

Food-induced immediate response of the esophagus (FIRE) is a concept that describes an unpleasant esophageal sensation, such as burning, choking or pressure, which is distinct from dysphagia and occurs immediately after mucosal contact with certain foods [53]. In a survey of 368 patients with EoE, 40% reported FIRE symptoms; the most common triggers were fresh fruit, vegetables and wine [53].

Atopic comorbidities, including allergic rhinitis, asthma and atopic eczema, are common in patients with EoE [2–4, 25]. IgE-mediated food allergy may coexist [25]. However, EoE is primarily a non-IgE-mediated condition; serum-specific IgE, skin-prick testing and patch testing do not reliably identify EoE food triggers and should not be used to select dietary exclusions [2, 3, 26]. Peripheral eosinophilia and raised total serum IgE may occur but are not sufficiently accurate to diagnose or monitor EoE [2–4]. In adults with esophageal symptoms, concurrent atopy, previous food bolus obstruction, failure to respond to a proton pump inhibitor, raised serum IgE and/or peripheral blood eosinophilia increase the index of suspicion for EoE [54]. These features are not diagnostic; confirmation requires endoscopy with esophageal biopsies [2–4]. EoE is frequently associated with impaired health-related quality of life because of dysphagia, anxiety about choking or food bolus obstruction, dietary restrictions and anxiety associated with specific foods [2–4, 55, 56]. Anxiety and depression are not uncommon, while esophageal hypervigilance and symptom-specific anxiety can contribute substantially to symptom severity and disease burden [4, 56]. Psychological support should be considered when appropriate [4].

Clinical symptoms of EoE in adults
Dysphagia
Food bolus obstruction
Retrosternal/chest pain
Heartburn
Regurgitation
FIRE

Table 4.1: Disease presentation of EoE in adults. Modified from [2–4, 47, 53].

5 Pathogenesis

Several interacting factors contribute to the pathogenesis of EoE, including food and environmental antigens, epithelial barrier dysfunction and genetic susceptibility (Figs. 5.1 and 5.2) [2–4, 57]. Atopic conditions such as asthma, atopic eczema and allergic rhinitis commonly coexist (see section 2) [2–4]. Food triggers vary between individuals. Animal milk and wheat are most frequently identified; egg and soy/legumes are less common, while peanuts/tree nuts, fish and seafood appear to play a limited role [2, 3, 5].

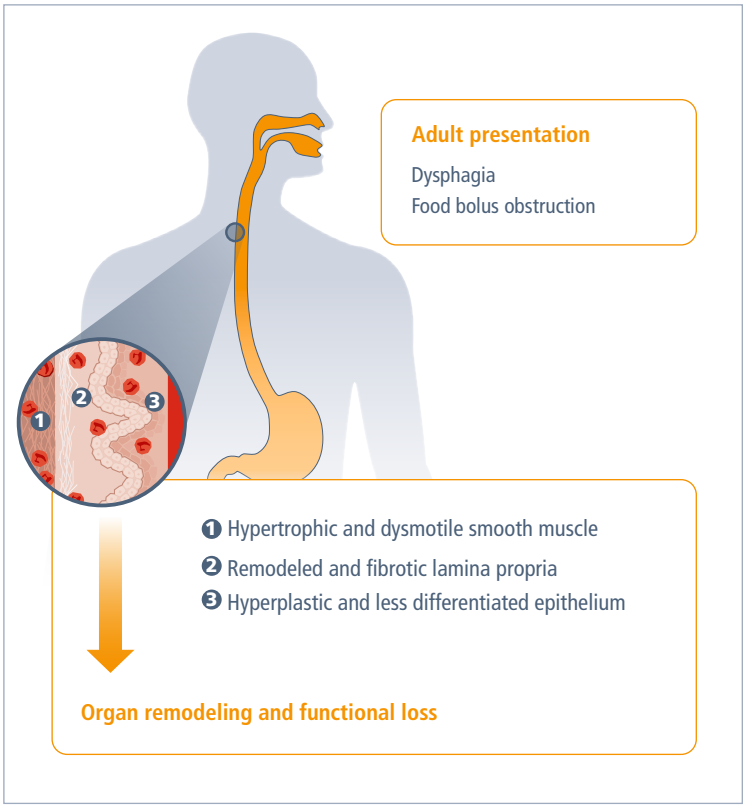


Figure 5.1: Pathogenesis and consequences of EoE in adults. Modified from [4, 57].

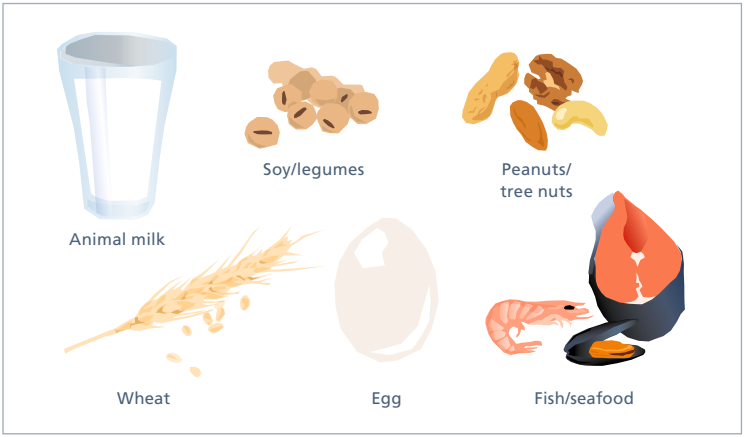


Figure 5.2: Food groups included in an empirical six-food elimination diet for EoE. Modified from [2, 3, 5].

EoE is characterized by a type 2 immune response involving eosinophils, mast cells and mediators including eotaxin-3, interleukin (IL)-5 and IL-13. Genetic variants affecting epithelial barrier function and type 2 inflammation, including *TSLP* and *CAPN14*, contribute to susceptibility [2–4, 57]. Food antigens are important triggers, and aeroallergens may contribute in some patients [2–4]. Although food-specific IgG4 deposits have been identified in the esophageal mucosa, their pathogenic role remains uncertain; EoE is not primarily an IgE-mediated disease [2–4, 58]. Persistent inflammation promotes epithelial hyperplasia, subepithelial fibrosis, smooth muscle dysfunction and esophageal remodeling [4, 57].

6 Diagnosis

EoE is diagnosed by the combination of symptoms of esophageal dysfunction and histology showing a peak eosinophil count of ≥ 15 eos/hpf in at least one esophageal biopsy, after exclusion of alternative causes of esophageal eosinophilia (Fig. 6.1) [2–4, 9].

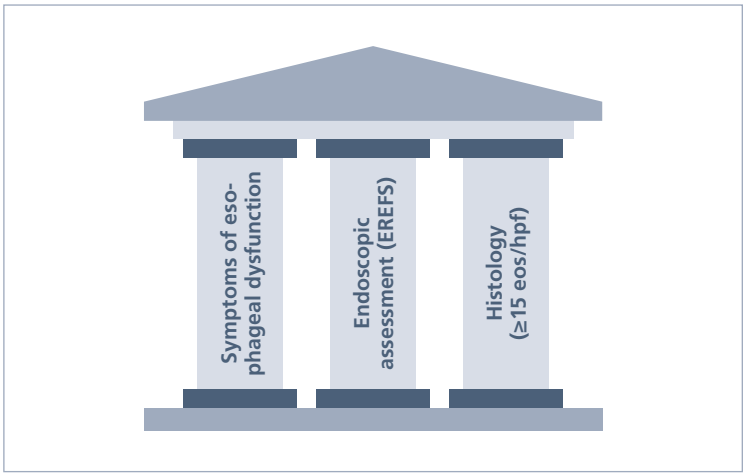


Figure 6.1: Core elements of diagnostic assessment in EoE. Modified from [3, 4, 9].

In adults with dysphagia, food bolus obstruction or other features suggestive of EoE, diagnostic assessment should include esophagogastroduodenoscopy (EGD) with esophageal biopsies, including when the esophagus appears macroscopically normal (Fig. 6.2) [3, 4, 9, 59].

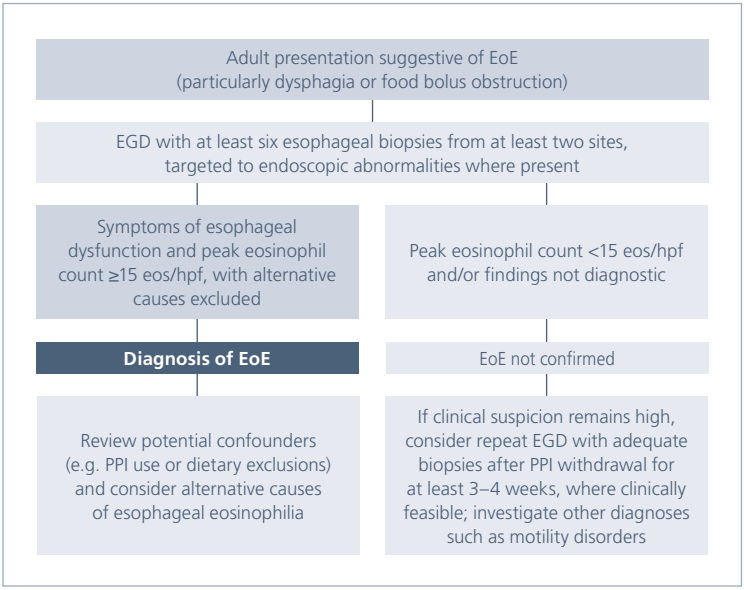
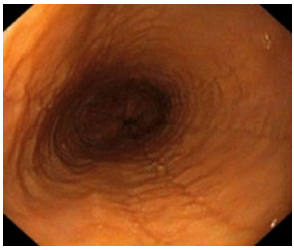


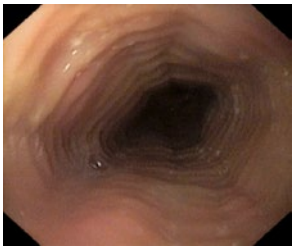
Figure 6.2: Adult diagnostic pathway for suspected EoE. Modified from [3, 4, 9, 59].

Endoscopy and biopsy

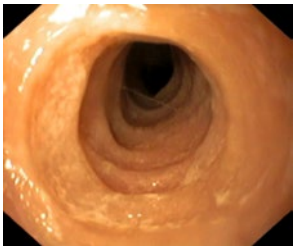
Typical endoscopic findings in EoE include white exudates or plaques, longitudinal furrows, edema with loss of vascular markings, mucosal fragility (crêpe-paper esophagus), rings, strictures and diffuse luminal narrowing (see Fig. 6.3) [2–4, 60]. Importantly, a normal endoscopic appearance does not exclude EoE [2–4]. The EoE Endoscopic Reference Score (EREFS; exudates, rings, edema, furrows and strictures) was proposed in 2013 as a validated classification and grading system for EoE [61]. This has since been validated in independent adult cohorts and demonstrated good interobserver reliability [62, 63]. EREFS provides a standardized method of recording the presence and severity of endoscopic features and is widely used in clinical studies (Table 6.1) [3, 4, 61–63].



Furrows and edema

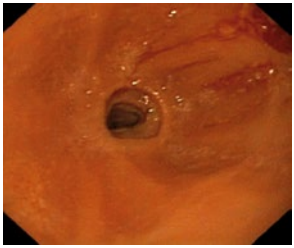


Rings and edema



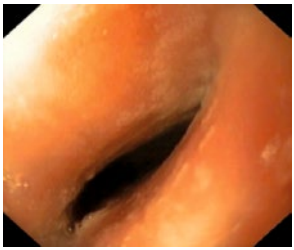
Rings (trachealization), **exudates, edema**

Exudates

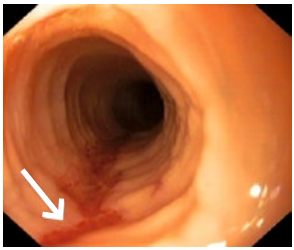


Stricture (severe)

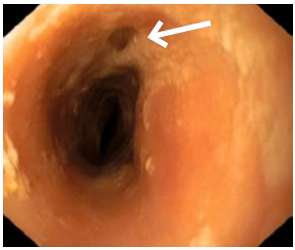
Food bolus obstruction



Severe luminal stenosis



Rings and crêpe-paper mucosa (longitudinal mucosal tear during biopsy)



Extensive luminal stenosis; blind fistula after perforation during dilatation (incidental finding)

Figure 6.3: Examples of endoscopic manifestations of EoE. Images courtesy of the author and Prof. Alex Straumann.

Grade	Major features
	Exudates (E)
0	None
1	Mild (≤ 10% of esophageal surface area)
2	Severe (> 10% of esophageal surface area)
	Rings
0	None
1	Mild (subtle)
2	Moderate (distinct rings that do not impair passage of a standard diagnostic endoscope)
3	Severe (distinct rings that do not permit passage of a standard diagnostic endoscope)
	Edema
0	Absent (distinct vascularity present)
1	Present (loss of clarity or absence of vascular markings)
	Furrows (F)
0	Absent
1	Present
	Strictures
0	Absent
1	Present
Grade	**Minor features**
	Crêpe-paper esophagus (mucosal laceration on passage of the diagnostic endoscope)
0	Absent
1	Present

Table 6.1: Modified EREFS classification. Modified from [3, 4, 61].

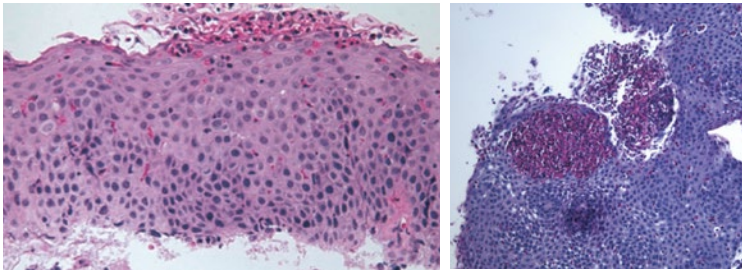
Symptoms and endoscopic findings correlate only modestly with histological activity, so biopsy is required both to diagnose EoE and to assess disease activity [3, 4, 64, 65]. Esophageal eosinophilia is often patchy [66]. At least six biopsies should therefore be obtained from at least two esophageal sites, preferably targeting exudates, furrows or other abnormalities; biopsies are required even when the mucosa appears normal [2–4]. In adults with non-esophageal gastrointestinal symptoms or corresponding endoscopic findings, gastric and/or duodenal biopsies should also be obtained at index endoscopy to exclude other eosinophilic gastrointestinal disorders [4, 67].

For diagnostic assessment, PPIs should be discontinued at least three weeks before EGD and biopsy [3, 4]. If this has not occurred and suspicion remains despite non-diagnostic histology, repeat EGD with adequate biopsies should be considered after an appropriate period without PPI therapy [3, 4]. PPI use and the duration

of withdrawal should be documented in the endoscopy report and histology request [3].

Histology

In routine clinical practice, hematoxylin and eosin (H&E) staining is sufficient for histological assessment. The accepted diagnostic threshold is a peak count of ≥15 eos/hpf (approximately 60 eos/mm^2) in at least one high-power field on esophageal biopsy [2–4]. Additional features that should be assessed include eosinophil microabscesses, basal-zone hyperplasia, dilated intercellular spaces, eosinophil surface layering, papillary elongation and lamina propria fibrosis. These features support diagnosis and assessment of disease activity but are not specific to EoE (Fig. 6.4) [2, 4].



Eosinophil-rich inflammatory infiltrate

Eosinophil-rich microabscess

Figure 6.4: Histological features of EoE.
Images courtesy of Prof. Thomas Günther, Laboratory Lademannbogen, Hamburg, Germany.

The validated Eosinophilic Esophagitis Histology Scoring System (EoEHSS) provides a standardized, semiquantitative assessment of the severity (grade) and extent (stage) of eight histological features of EoE (Table 6.2) [68]. This tool is responsive to change and may discriminate disease activity more effectively than peak eosinophil count alone [69]. EoEHSS is recommended as an outcome measure in therapeutic studies [70], although its use in routine clinical practice remains limited [4].

Histological features (EoEHSS)
Eosinophil density
Basal-zone hyperplasia
Eosinophil abscesses
Eosinophil surface layering
Dilated intercellular spaces
Surface epithelial alterations
Dyskeratotic epithelial cells
Lamina propria fibrosis

Table 6.2: Histological features of EoE. Modified from reference [68].

Additional diagnostic procedures

Early studies of minimally invasive sampling methods, including the esophageal string test and Cytosponge, demonstrated promising correlations between eosinophil-derived proteins or cytology and histological eosinophilia [71, 72]. Esophageal distensibility can be assessed using a functional lumen imaging probe (FLIP; EndoFLIP) and is often reduced in active EoE [73–75]. While no minimally invasive biomarker is currently validated for the diagnosis or monitoring of EoE in routine clinical practice, investigated markers in blood, stool, saliva, urine and breath condensate include eosinophil cationic protein, eosinophil-derived neurotoxin, mast-cell tryptase, exhaled nitric oxide, eotaxin-3 and other chemokines [4, 76]. One study did find correlations between peripheral blood eosinophil counts, esophageal eosinophilia and histological remission during treatment, but with insufficient diagnostic accuracy (area under the receiver operating characteristic curve, 0.754) [77].

7 Treatment

EoE is a chronic, potentially progressive disease; active disease should therefore be treated [2, 3]. Treatment aims to control symptoms, improve histological and endoscopic disease activity and quality of life, and reduce the risk of fibrostenotic complications. Topical corticosteroids, PPIs and empiric elimination diets are established anti-inflammatory options, while dupilumab provides an additional option for selected patients. The initial approach should be individualized according to disease phenotype, comorbidities, patient preferences, treatment feasibility and access. Endoscopic dilatation may be required for clinically relevant narrowing, but does not treat the underlying inflammation (Figure 7.1) [2, 3, 5].

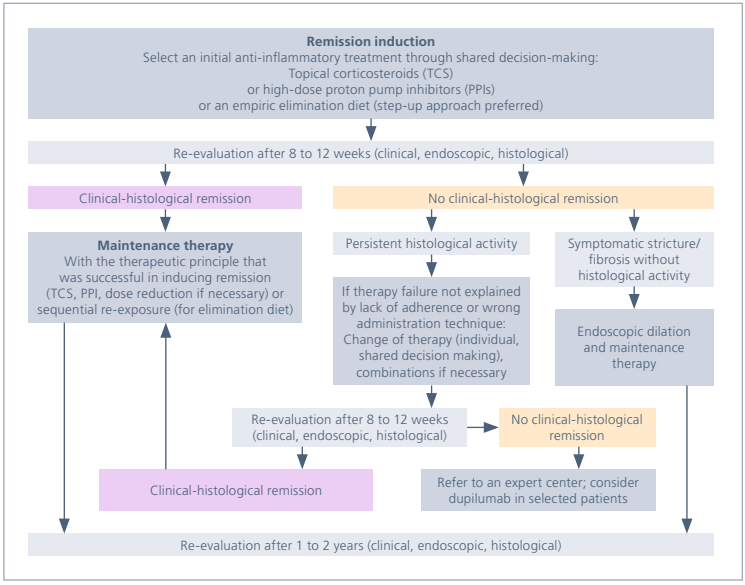


Figure 7.1: Adult treatment pathway, based on current European and British evidence-based treatment recommendations [2, 3, 5].

Assessing response and maintaining remission

Treatment response should be assessed across several domains: symptoms, endoscopic findings, histology and, where appropriate, EoE-specific quality of life. Symptoms alone correlate poorly with inflammatory activity and may be masked by behavioral adaptations, such as eating slowly, prolonged chewing or avoiding particular food textures. Endoscopy with esophageal biopsies is therefore recommended 8–12 weeks after treatment is started and after a therapeutic change [3, 5]. A peak eosinophil count below 15 eos/hpf has been widely used to define histological response in clinical research, although individual trials have used more stringent thresholds [78–81].

Relapse is common after withdrawal of pharmacological or dietary treatment [5]. An effective induction therapy should therefore generally be continued as maintenance treatment. The choice and intensity of maintenance therapy should reflect the treatment that achieved remission, disease severity, fibrostenotic risk, tolerability and patient preference, with periodic clinical, endoscopic and histological review [2, 3, 5].

Topical corticosteroids

Topical (swallowed) budesonide and fluticasone are effective for inducing clinical and histological remission [2, 3, 5, 82–85]. Earlier studies generally used topical formulations developed for asthma, whereas more recent adult trials have evaluated esophageal-targeted suspensions, orodispersible tablets and orally disintegrating tablets [80, 81, 83–86]. EoE-specific formulations are intended to prolong contact between the corticosteroid and the esophageal mucosa and are supported by the most robust evidence in this class [3, 5, 87]. In a placebo-controlled phase 3 trial in adults, budesonide orodispersible tablets at a dose of 1 mg twice daily produced clinical and histological remission in 57.6% of patients after 6 weeks; histological remission was achieved in 93%. Extending treatment to 12 weeks increased the clinical and histological response among patients who had not responded fully at week 6 [5, 84]. Budesonide orodispersible tablets at twice-daily doses of either 0.5 mg or 1 mg maintained remission for 48 weeks in approximately three-quarters of patients, compared with 4.4% receiving placebo [88].

Esophageal or oral candidiasis is the most frequently reported adverse effect with topical corticosteroids, usually mild and treatable with local anti-mycotic therapy [88–90]. Although systemic corticosteroid effects occur less frequently, monitoring for adrenal suppression and effects on bone health should be considered during prolonged or high-dose treatment, particularly in patients receiving corticosteroids for other conditions [2, 3, 5].

Proton pump inhibitors

PPIs can induce clinical and histological remission in EoE through both acid-dependent and anti-inflammatory effects, including inhibition of eotaxin-3 expression [91]. EoE and GERD may coexist, and a clinical and histological response to PPI therapy does not exclude the diagnosis of EoE [2, 3, 5]. A meta-analysis reported histological remission in approximately half of treated patients, and a large registry study reported histological remission < 15 eos/hpf in 49% [92, 93]. For induction in adults, British and European consensus recommendations support high-dose, twice-daily treatment for 8–12 weeks, such as omeprazole at a dose of 20–40 mg twice daily, with endoscopic and histological confirmation of response [3, 5].

Observational data suggest sustained remission with PPI therapy in approximately 70% of patients with evaluable follow-up, although controlled long-term evidence remains limited [3, 5, 92]. PPIs are generally considered well tolerated [5].

Dietary therapy

Empiric food elimination diets can induce clinical and histological remission in adults and may provide sustained, drug-free disease control while identified trigger foods remain excluded [2, 3, 5, 94, 95]. In a prospective adult study, a six-food elimination diet achieved histological remission in approximately 70% of patients, and systematic food reintroduction identified one or more triggers in most responders [94]. Animal milk and wheat are among the foods most frequently implicated, but triggers vary between individuals [2, 3]. Current European consensus recommendations support a step-up approach, beginning with the elimination of animal milk and escalating to a broader diet only if required. This reduces unnecessary restriction and may limit the number

of endoscopies compared with starting immediately with a six-food diet [2, 5].
Dietary treatment requires motivation and may affect nutritional adequacy, cost, social eating and quality of life. It should therefore be planned and supervised by a dietitian with expertise in EoE [3, 5]. Allergy test-directed elimination is not recommended because available tests do not reliably identify food triggers [3, 5]. An elemental diet can be highly effective but is generally reserved for selected refractory cases because of its substantial practical, nutritional and quality of life burden [2, 3, 5].

Endoscopic dilatation

Endoscopic dilatation, using either a balloon or bougie technique, is effective for dysphagia caused by fibrostenotic disease, strictures or a narrow-caliber esophagus [3, 96]. Meta-analyses report symptomatic improvement in approximately 75–85% of patients [97, 98], with no clear difference in efficacy between balloon and bougie techniques [3]. The procedure may need to be staged or repeated, depending on the severity and length of the narrowing and the response achieved.
Dilatation does not reduce esophageal inflammation and should therefore be combined with effective anti-inflammatory treatment [2, 3]. Mucosal tears and transient post-procedural chest pain are relatively common and should be discussed with the patient. Perforation, significant bleeding and hospitalization are uncommon; pooled perforation rates in a 2017 meta-analysis were well below 1% [96].

Dupilumab

Dupilumab is a monoclonal antibody directed against the IL-4 receptor alpha subunit, thereby inhibiting both IL-4 and IL-13 signaling [99]. The pivotal 52-week TREET trial, which included 222 adults with active EoE, showed that weekly subcutaneous dupilumab significantly improved histologic outcomes and alleviated EoE symptoms compared with placebo. The most common adverse event that occurred during the treatment period was injection-site reaction [100].

Current European consensus recommendations include dupilumab among the available treatment options for EoE [3, 5]. Trial populations have generally included patients with disease inadequately controlled by PPIs and, frequently, previous dietary or topical corticosteroid treatment; dupilumab may therefore be particularly relevant after an inadequate response, intolerance or contraindication to conventional therapy. Dupilumab may also be considered earlier when a patient has a coexisting type 2 inflammatory condition for which biologic treatment is indicated [5].

Treatments not routinely recommended

Systemic corticosteroids are not recommended as standard treatment for EoE because effective topical alternatives are available and systemic exposure carries a greater risk of adverse effects. Immunomodulators such as azathioprine and mercaptopurine, and anti-allergic medicines such as leukotriene antagonists, are not recommended because supporting evidence is insufficient; additionally, monoclonal antibodies without regulatory approval for EoE should be used only within the setting of randomized controlled trials [3, 5].

8 Summary and future developments

EoE is a chronic, immune-mediated and potentially progressive esophageal disease. In adults, dysphagia and food bolus obstruction are characteristic symptoms, while endoscopic findings support the diagnosis and esophageal biopsies are required to confirm it [3].

Topical (swallowed) corticosteroids, PPIs and empiric elimination diets are established anti-inflammatory treatments, and dupilumab is an additional option for selected adults. There is no single preferred treatment for every patient – the choice should reflect disease phenotype, comorbidities, patient preferences, feasibility and access [2, 3, 5].

Endoscopic dilatation is an effective adjunct for fibrostenotic disease but should be combined with anti-inflammatory treatment. Because relapse is common when treatment is withdrawn, effective therapy should generally be continued to maintain remission, with response and follow-up assessed using symptoms, endoscopy and histology [2, 3, 5].

Care is best coordinated by a multidisciplinary team that may include gastroenterologists and endoscopists, specialist nurses, pathologists, dietitians, allergists, pharmacists and general practitioners, according to the patient's needs.

Further research should clarify treatment sequencing and combination strategies, optimize long-term monitoring and develop reliable, less invasive measures of disease activity.

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Supplement: Pediatric EoE – presentation, diagnosis and management

This supplement summarizes aspects of EoE that are especially relevant to children and adolescents, based primarily on the 2024 update of the European Society for Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) treatment recommendations with support from British and European guidance as appropriate [2–5, 101].

Pediatric EoE requires age-appropriate assessment and management. Symptoms evolve with age, treatment may affect growth, nutrition, feeding behavior and family life, and care must ultimately be transferred to adult services. These considerations support a dedicated pediatric approach within a multidisciplinary service [3, 101].

S1 Overview

Recognized risk factors of pediatric EoE include male sex, atopy, a family history of EoE and previous repair of esophageal atresia. Asthma, allergic rhinitis, IgE-mediated food allergy and eczema are more common in children with EoE; clinicians should therefore maintain a high index of suspicion when compatible gastrointestinal or feeding symptoms occur in a child with atopic disease [3, 101]. As in adults, EoE may progress from inflammation to esophageal remodeling and fibrostenotic disease. Although strictures are less common in children than in adults, they can occur. Early recognition, effective anti-inflammatory treatment and long-term follow-up are important to reduce the risk of remodeling and stricture formation [3, 101].

S2 Clinical presentation

The presentation of pediatric EoE varies substantially with age. Infants and younger children often have non-specific symptoms resembling gastroesophageal reflux, including vomiting, abdominal or epigastric pain, food refusal, coughing during feeding and feeding difficulties. Slow eating, drinking repeatedly during meals and poor weight gain or faltering growth may provide additional clues [3, 101]. Older children and adolescents more commonly

report solid-food dysphagia and may present with food bolus obstruction. Chest pain can also occur. Children may adapt by avoiding foods with difficult textures, taking small bites, chewing excessively, eating slowly or using fluids to help food pass; these behaviors should be sought actively because they can mask the severity of esophageal dysfunction (Table S1) [3, 101].

Clinical symptoms of EoE in children
Abdominal pain
Chest pain
Heartburn
Coughing
Reduced appetite
Dysphagia
Food refusal
Growth failure
Choking
Nausea
Regurgitation
Sleep disorder

Table S1: Disease presentation of pediatric EoE. Modified from [3, 47, 101].

In the same way as for adult EoE, symptoms alone cannot establish the diagnosis or reliably indicate inflammatory activity. ESPGHAN recommends the validated Pediatric Eosinophilic Esophagitis Symptom Score (PEESS v2.0) to assess symptoms at diagnosis, during monitoring and when evaluating treatment response. The instrument supplements, but does not replace, endoscopic and histological assessment [101].

S3 Diagnosis

Endoscopy and esophageal biopsies

Upper gastrointestinal endoscopy with esophageal biopsies remains the diagnostic gold standard. At least six biopsies should be obtained from the upper and lower esophagus, with visible

lesions targeted. Biopsies are required whenever EoE is suspected, even if the esophagus appears macroscopically normal, because a normal endoscopic appearance does not exclude EoE [3, 101]. Inflammatory features, such as edema, exudates and furrows, and fibrostenotic features, including rings and strictures should be recorded using EREFS. Inflammatory findings predominate in younger children, but endoscopy cannot replace histological assessment [3, 4, 101].

Histology

A peak eosinophil count of ≥15 eos/hpf supports the histological diagnosis in the appropriate clinical context. Because eosinophilic infiltration may be patchy, samples from more than one esophageal level are essential. Reports should use a standardized eosinophil density or state the high-power-field area to support comparison between microscopes and centers [3, 101]. Other histological features, including basal-zone hyperplasia, eosinophilic microabscesses, surface layering, dilated intercellular spaces and lamina propria fibrosis, can provide additional information. Alternative causes of esophageal eosinophilia should be considered before the diagnosis is confirmed [2–4, 101].

Tests not used routinely

Specific IgE testing, skin-prick testing and atopy-patch testing do not reliably identify the foods that trigger EoE and should not be used to select foods for elimination. IgE-based testing may nevertheless be relevant when assessing coexisting immediate food allergy or the risk of an acute reaction during food reintroduction [3, 101].

No currently available blood, urine, stool or other biomarker is sufficiently accurate to replace endoscopy and biopsy. Esophageal pH-impedance monitoring is not required to diagnose EoE, although it may be useful in selected children when concomitant gastroesophageal reflux is suspected or symptoms and histological findings are discordant [101].

S4 Treatment

Principles of treatment

Treatment aims to control symptoms, achieve histological and endoscopic improvement, preserve normal growth and nutrition, improve quality of life and prevent fibrostenotic complications. PPIs, swallowed topical corticosteroids and empiric elimination diets are all recommended first-line options for inducing remission in childhood EoE, which is consistent with the broader European emphasis on individualized treatment choice (Fig. S1) [2, 3, 5, 101]. The initial approach should be selected through shared decision-making with the child and family. Relevant considerations include age, disease phenotype and severity, feeding and nutritional status, atopic comorbidities and total corticosteroid exposure, treatment burden, likely adherence, family preference, access to specialist dietary support and the need for repeated endoscopy [3, 101].

Proton pump inhibitors

High-dose PPI therapy can induce clinical and histological remission in a proportion of children and is a practical first-line option, particularly when gastroesophageal reflux may coexist. Response must be assessed by endoscopy and biopsy. In children who achieve remission with a PPI, treatment can be continued as maintenance therapy, with dose adjustment based on response and clinical review [3, 101].

Topical corticosteroids

Topical (swallowed) corticosteroids, including budesonide and fluticasone, can induce and maintain remission. No available topical corticosteroid has been shown conclusively to be superior to another in children. Treatment is generally well tolerated, with oral or esophageal candidiasis being the most frequent adverse effect which is usually readily treated. The total corticosteroid burden, including inhaled, intranasal, topical and systemic treatment for coexisting atopic disease, should be reviewed. Monitoring for adrenal suppression should be considered in children receiving prolonged or higher-dose treatment or corticosteroids by several routes [3, 101].

Dietary therapy

Empiric food elimination is the recommended dietary approach. The ESPGHAN recommendations suggest a step-up strategy that begins with limited elimination and becomes more restrictive only when required. Cow's milk is generally the first food considered, followed where necessary by wheat-containing cereals and egg. Remission and food triggers must be established by systematic food reintroduction with endoscopic and histological assessment [101]. Dietary treatment should be supervised by an experienced pediatric dietitian to protect nutritional adequacy, growth, feeding development and quality of life. The burden on the child and family increases as the diet becomes more restrictive. An elemental diet with amino acid-based formula is highly effective but is usually reserved for selected children with severe disease, multiple food allergies, faltering growth, or inability to follow or respond to other approaches [3, 101].

Refractory disease, biologic therapy and strictures

When response is inadequate, adherence, administration technique and the original diagnosis should be reviewed before treatment is changed. An alternative first-line modality or, in selected cases, combination treatment may be considered. ESPGHAN recommends that dupilumab may be used in selected children over 1 year of age and weighing more than 15 kg when EoE is refractory to conventional treatment or when there is a substantial coexisting atopic burden with an approved indication for biologic therapy. Use must follow locally approved product information [101]. Other biologics should not be used routinely outside specialist centers or clinical trials [5, 101].

Mild inflammatory strictures may improve with medical or dietary treatment. Endoscopic dilatation is reserved for highly selected children with severe persistent narrowing or a need for rapid relief of significant dysphagia. It relieves obstruction but does not treat inflammation and should be combined with anti-inflammatory therapy. ESPGHAN also allows consideration of a short course of systemic corticosteroids as an alternative to dilatation for moderate-to-severe inflammatory strictures with severe symptoms. If the anatomy or caliber is not adequately defined endoscopically, a barium swallow may be helpful before dilatation [101].

S5 Monitoring and long-term management

For PPIs, topical corticosteroids and elimination diets, the ESPGHAN recommendations suggest an induction period of at least 8–12 weeks before endoscopic reassessment; an elemental diet should be assessed after no less than four weeks. Treatment efficacy should be evaluated using symptoms, endoscopy and esophageal biopsies because symptomatic improvement may occur without histological remission [101]. Maintenance therapy is recommended for all children after histological remission has been achieved, initially for at least one year. Decisions to continue, reduce or withdraw treatment should be individualized according to disease severity, phenotype, treatment burden and the needs of the child and family [3, 101].

Endoscopic and histological reassessment is required if symptoms recur during maintenance therapy. In stable clinical remission, the timing should be individualized; ESPGHAN suggests reassessment after 1–3 years [101].

S6 Quality of life and transition to adult care

EoE and its treatment can affect eating, school attendance, social participation, emotional wellbeing and family life. Validated health-related quality of life questionnaires, including the pediatric EoE module of the PedsQL, can be used alongside symptoms, endoscopy and histology when evaluating treatment response [101].

Care should be multidisciplinary and coordinated. Depending on the patient's needs, the team may include pediatric and adult physicians, a specialist nurse, dietitian, and psychologist, with contributions from other professionals as clinically appropriate [101]. The transition to adult healthcare services should be a planned, individualized process that begins well before adulthood. Pediatric and adult teams should review the diagnosis, disease course, previous responses, current treatment, dietary restrictions, endoscopic and histological findings and the patient's ability to manage the condition independently. A coordinated handover involving the clinical team working closely with the young person and their family can help maintain continuity of care [3, 101].

S7 Summary and conclusions

Pediatric EoE is a chronic immune-mediated disease whose presentation evolves with age. Reflux-like symptoms, vomiting, feeding difficulties and faltering growth predominate in younger children, whereas dysphagia and food bolus obstruction are more typical in older children and adolescents. Diagnosis requires compatible symptoms and esophageal histology showing a peak count of ≥ 15 eos/hpf, after alternative causes have been considered. At least six biopsies should be obtained from the upper and lower esophagus even when endoscopic appearances are normal. PPIs, topical (swallowed) corticosteroids and empiric elimination diets are all first-line options. Treatment should be selected with the child and family, taking account of disease severity, growth and nutrition, atopic comorbidity, treatment burden, adherence and access to specialist support [3, 101].

Response and long-term control cannot be judged from symptoms alone. Endoscopy and biopsy are required after induction and at clinically appropriate intervals during maintenance. Maintenance treatment, safety surveillance and attention to growth, feeding and quality of life within the framework of a coordinated multidisciplinary team and planned transition to adult healthcare services are central to long-term care [3, 101].

Quiz

Your knowledge about EOE:

Have you been able to refresh your medical expertise on EOE with the help of this brochure? Test your knowledge!

This QR code (Microsoft Forms) will take you to 10 questions. Only 1 answer out of 5 possible is correct.

Good luck!

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