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Atopic condition of the esophagus - eosinophilic esophagitis – a literature review

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Abstract:

Introduction and purpose

Eosinophilic esophagitis (EoE) is a chronic, immune-mediated disorder causing esophageal dysfunction in children and adults. Characterized by eosinophilic inflammation, EoE leads to dysphagia, food impaction, and esophageal remodelling. Since its classification in the 1990s, research has expanded, yet aspects of its pathogenesis, diagnosis, and management remain under investigation. This review compiles current knowledge on EoE, covering its pathophysiology, epidemiology, clinical presentation, diagnosis, treatment, and prognosis. By integrating recent findings and guidelines, it aims to enhance understanding and optimize care.

Description of the state of knowledge

EoE is defined by eosinophilic infiltration of the esophageal mucosa, leading to dysfunction. Diagnosis relies on histologic criteria, with ≥15 eosinophils per high-power field in biopsies. Endoscopic tools like the EoE Endoscopic Reference Score (EREFS) aid evaluation, though differentiation from GERD remains challenging.

Treatment includes dietary modifications, pharmacologic therapy, and endoscopic interventions. First-line options are dietary elimination, proton pump inhibitors (PPIs), and topical corticosteroids, while emerging biologics target inflammatory pathways. Endoscopic dilation is reserved for fibrostenotic cases.

Despite therapeutic advancements, EoE is a chronic condition requiring long-term management. Continued research is needed to refine treatment strategies and improve outcomes.

Conclusions

EoE requires lifelong management. While dietary, pharmacologic, and endoscopic treatments control symptoms, they do not halt disease progression. Biologic therapies offer promising advances, but further studies are needed to optimize long-term strategies and patient care.

Keywords: eosinophilic esophagitis; dysphagia; eosinophil; dupilumab

Introduction and purpose

Eosinophilic esophagitis (EoE) is a chronic, immune-mediated inflammatory disease of the esophagus that has gained increasing recognition as a significant cause of esophageal dysfunction in both pediatric and adult populations. The prevalence of EoE has risen in recent decades, with current estimates suggesting an incidence of approximately 34.4 cases per 100,000 individuals in Europe and North America [1]. EoE is primarily driven by a T-helper type 2 (Th2) immune response, leading to chronic eosinophilic inflammation, progressive esophageal remodelling, and, ultimately, luminal narrowing, which manifests as dysphagia, food impaction, and esophageal strictures [2,3].

Historically, esophageal eosinophilia was initially attributed to gastroesophageal reflux disease (GERD), and EoE was not widely recognized as a distinct clinical entity until the 1990s [3,4]. Unlike GERD, EoE does not typically respond to acid-suppressive therapy, and its diagnosis requires histopathologic confirmation of at least 15 eosinophils per high-power field in esophageal biopsy samples, alongside exclusion of alternative causes of esophageal eosinophilia [2]. The exact pathogenesis of EoE remains an area of active research, but food allergens are considered primary triggers, and genetic predisposition has been implicated in disease susceptibility [5].

Given its chronic nature, EoE imposes a significant burden on healthcare systems and patient quality of life. Advances in understanding the disease have led to the development of evolving diagnostic criteria and management strategies, including dietary modifications, pharmacological treatments such as corticosteroids and biologics, and endoscopic interventions for severe cases [1]. This review aims to provide an updated synthesis of current knowledge on the epidemiology, pathogenesis, clinical presentation and treatment of EoE, integrating recent findings and clinical guidelines to enhance understanding and optimize patient care.

Materials and methods

This literature review on eosinophilic esophagitis utilized articles published between 2015 and 2025, sourced from database PubMed. The review focused on studies addressing the pathophysiology, epidemiology, clinical presentation, treatment, and prognosis of EoE. A total of 31 articles were analyzed to provide a comprehensive synthesis of current knowledge.

Pathophysiology

The pathophysiology of EoE involves a complex interplay of genetic predisposition, immune dysregulation, and environmental factors, ultimately leading to chronic esophageal inflammation and tissue remodeling. EoE is primarily characterized by a Th2-driven immune response, similar to other atopic disorders, where interleukins IL-4, IL-5, and IL-13 play crucial roles in the recruitment and activation of eosinophils within the esophageal mucosa. Elevated levels of these cytokines, along with an overexpression of eotaxin-3, a chemokine responsible for eosinophil attraction, contribute to persistent eosinophilic infiltration and inflammation [6,7].

Eosinophils, once activated, release cytotoxic granules containing major basic protein (MBP), eosinophilic cationic protein (ECP), and eosinophil peroxidase (EPO), which mediate tissue damage and contribute to epithelial barrier dysfunction. Additionally, eosinophils secrete transforming growth factor-beta (TGF-β), a key mediator of fibrosis, leading to subepithelial fibrosis, esophageal rigidity, and progressive luminal narrowing. This remodeling process results in the characteristic clinical manifestations of dysphagia and food impaction observed in EoE patients [8,9].

Genetic factors also play a crucial role in EoE susceptibility. Genome-wide association studies (GWAS) have identified several genetic loci linked to EoE, including variants in *CAPN14*, a gene encoding calpain-14, an esophageal-specific protease involved in epithelial barrier integrity. Dysregulation of this protease may impair barrier function, allowing antigen exposure and immune activation. Furthermore, single nucleotide polymorphisms (SNPs) in *TSLP*, *eotaxin-3*, and *TGF-β1* genes have been implicated in disease pathogenesis [7,8].

EoE is predominantly considered a non-IgE-mediated allergic disease, where food and aeroallergens act as antigens that trigger the inflammatory cascade. Although IgE-mediated sensitization may coexist, skin prick tests and serum IgE levels often fail to accurately predict causative allergens in EoE patients. Recent evidence also suggests a potential role of IgG4 in disease pathology, but its significance remains unclear [7,8,9].

Epidemiology

EoE has evolved from being a rare condition to a frequently diagnosed disorder of the upper gastrointestinal tract. The global prevalence of EoE is estimated at 0.5–1 case per 1,000 individuals, with variations between geographic regions [10]. In Europe and North America, incidence rates range from 2.1 per 100,000 per year in the Netherlands to 12.8 per 100,000 per year in Ohio, USA. A meta-analysis estimated the overall pooled incidence rate at 3.7 per 100,000 person-years, with a higher incidence observed in adults (7.0 per 100,000) compared to children (5.1 per 100,000) [11,12].

Longitudinal studies suggest a consistent increase in the incidence of EoE over the past two decades. Reports from North America and Europe show incidence increases ranging from 6-fold to over 100-fold, which cannot be attributed solely to improved diagnostic awareness. Retrospective analyses of esophageal biopsy samples from earlier decades further confirm that this surge reflects a genuine rise in EoE prevalence rather than simply improved surveillance [11].

The rising prevalence of EoE correlates with an increase in allergic diseases, supporting the hypothesis that environmental and dietary factors play a crucial role in disease development. High-income countries with improved hygiene, dietary modifications, and increased use of antibiotics during childhood appear to have higher EoE prevalence [12]. Notably, urban populations have shown higher prevalence rates compared to rural areas, further implicating environmental exposures in EoE pathogenesis [13].

Demographically, EoE predominantly affects males, with a male-to-female ratio of approximately 3:1, and is more common in Caucasian populations [10]. The average age of diagnosis ranges between 30 and 50 years in adults and between 5.4 and 9.6 years in children. While EoE has been described across all racial and ethnic groups, its highest incidence remains in Western populations [11,12].

Although some studies have suggested a link between EoE and seasonal variation, a recent meta-analysis found no significant seasonal impact on EoE diagnoses or food impaction episodes requiring medical intervention [12].

Clinical Presentation

EoE presents with a diverse range of symptoms that vary by age, disease severity, and patient adaptation. In infants and toddlers, symptoms are often non-specific and include vomiting, food refusal, and failure to thrive. As children grow, they may experience abdominal pain, nausea, and feeding difficulties, often leading to misdiagnosis with other gastrointestinal disorders. Adolescents and adults primarily present with dysphagia and food impaction, which can sometimes necessitate emergency endoscopic intervention [14,15].

Adults may also report heartburn, regurgitation, and exercise-induced chest pain, leading to confusion with gastroesophageal reflux disease (GERD). Some patients exhibit subtle symptoms and unknowingly modify their eating behaviors, such as excessive chewing, avoiding solid foods, or drinking liquids to assist swallowing. These adaptations can delay diagnosis, making clinical suspicion crucial in patients with a history of esophageal dysfunction [15].

A strong association exists between EoE and atopic conditions, including asthma, eczema, allergic rhinitis, and IgE-mediated food allergies. While children with EoE often exhibit hypersensitivity to common food allergens like milk, eggs, soy, and wheat, adults are more frequently sensitized to aeroallergens, suggesting a role for environmental triggers in disease pathogenesis [15, 16].

Diagnostic Approach

The diagnosis of EoE requires a combination of clinical assessment, endoscopic evaluation, and histopathological confirmation. Endoscopic features suggestive of EoE include esophageal rings, linear furrows, white exudates, and mucosal fragility. However, up to 10% of patients may have a normal-appearing esophagus, underscoring the necessity of esophageal biopsies for definitive diagnosis [17].

Histological analysis remains the gold standard for EoE diagnosis, with the presence of at least 15 eosinophils per high-power field in esophageal tissue being the primary diagnostic criterion. To maximize diagnostic accuracy, current guidelines recommend obtaining at least six biopsies from different esophageal regions due to the patchy distribution of eosinophilic infiltration [18].

Historically, a trial of proton pump inhibitors (PPIs) was required to distinguish EoE from PPI-responsive esophageal eosinophilia (PPI-REE). However, recent guidelines recognize that EoE and PPI-REE share overlapping genetic, histological, and clinical features. As a result, PPI responsiveness is no longer considered a diagnostic criterion, and PPIs are now regarded as a first-line treatment option rather than a diagnostic tool [19].

A validated endoscopic grading system, the EoE Endoscopic Reference Score (EREFS), aids in the objective assessment of disease severity. This scoring system evaluates five key features: edema, rings, exudates, furrows, and strictures, with higher scores correlating with more severe disease. The EREFS score is particularly useful for monitoring treatment response, as it tends to decrease with effective therapy [18, 20].

Treatment

Eosinophilic esophagitis (EoE) therapy focuses on reducing chronic inflammation, preventing disease progression, and relieving the esophageal dysfunction that leads to dysphagia and food impaction. Although treatment should always be tailored to the individual patient's clinical presentation and disease severity, management options generally include dietary modifications, pharmacological therapy, and endoscopic interventions. In many cases, these approaches are complementary rather than exclusive [21].

Patients often respond to a combination of therapies, and the best course of action typically depends on factors such as age, disease severity, comorbidities, and patient preference. Below, we introduce three primary treatment strategies: dietary interventions, pharmacological therapy, and endoscopic procedures [22].

Dietary Therapy

Dietary therapy aims to reduce or eliminate exposure to allergenic food proteins that drive the eosinophilic inflammatory process in EoE. Multiple studies have shown that limiting certain foods can lead to substantial improvements in both clinical symptoms and histopathological findings, although patient adherence and quality of life remain key concerns [22].

Three main dietary approaches are recognized in EoE management: elemental diets, empiric elimination diets, and diets guided by allergy tests[23].

Elemental diets rely on amino acid–based formulas that are free of intact protein antigens, thereby minimizing any allergenic exposure [23]. While highly effective in both children and adults, these diets can be challenging to maintain due to their taste, cost, and social implications. Empiric elimination diets, which often restrict the most common food triggers (milk, wheat, eggs, soy, nuts, and seafood), typically show good efficacy, although they also require careful monitoring of nutritional intake [22]. In many cases, a stepwise approach is used, starting with the elimination of one or two major allergens and expanding if needed. Diets guided by allergy tests rely on skin prick tests or serum IgE analyses to identify foods for removal, but the accuracy of these methods varies widely and may not reliably predict individual triggers [24]. Ultimately, the choice of dietary intervention should be individualized, taking into account each patient's dietary habits, comorbidities, and access to professional support. Regular follow-up, which may include repeat endoscopy and biopsy, enables clinicians to confirm treatment effectiveness and make timely adjustments if symptoms or tissue eosinophilia persist [22].

Pharmacological Therapy

Pharmacological therapy for EoE aims to suppress the underlying inflammatory process, prevent progression to fibrostenotic changes, and alleviate symptoms such as dysphagia or food impaction [21]. Several medication classes have demonstrated efficacy in achieving these goals. Among the most widely used are proton pump inhibitors (PPIs), which were historically employed to distinguish PPI-responsive esophageal eosinophilia (PPI-REE) from EoE [22,25]. However, PPIs are now recognized as a viable first-line treatment, both for reducing gastric acid exposure and mitigating proinflammatory mediators. Standard or high-dose PPI therapy is typically prescribed for at least 8–12 weeks, followed by endoscopic and histologic re-evaluation to determine response [26,27].

Swallowed topical corticosteroids constitute another cornerstone of pharmacological therapy, particularly fluticasone and budesonide, administered as slurries, aerosolized sprays, or orodispersible tablets. These agents effectively decrease eosinophil infiltration and improve esophageal mucosal integrity [22]. Although local candidiasis may occur, the overall safety profile is favorable, and clinicians often adjust dosages according to clinical and histological remission [22,26].

More recently, biologic therapies have emerged, targeting key cytokines implicated in EoE's Th2-driven inflammation (e.g., IL-4, IL-5, IL-13). Dupilumab, an IL-4 receptor alpha antagonist, is approved for certain age groups and offers a new option for patients refractory to standard therapies [22]. Other biologics, such as mepolizumab and reslizumab, focus on IL-5 blockade, although their clinical benefits have been somewhat variable [22,26].

In many cases, a combination of pharmacological and dietary interventions is employed, with endoscopic procedures reserved for addressing fibrostenotic complications. Treatment choices depend on disease severity, patient preferences, and coexisting conditions. Clinicians typically monitor both symptomatic improvement and histopathologic remission, adjusting therapies as needed to maintain durable disease control [21].

Endoscopic Therapy

Endoscopic interventions form a crucial aspect of EoE management, especially for patients with significant fibrostenotic changes. The most common procedure is esophageal dilation, performed with bougies or balloon dilators, which helps to mechanically disrupt or widen constricted segments of the esophagus. Patients often experience rapid relief of dysphagia after dilation, although mild post-procedural chest discomfort can occur [22,28,29].

It is important to note that while dilation can alleviate luminal narrowing, it does not address the underlying inflammatory process driving disease progression. For this reason, endoscopic therapy is often combined with dietary measures or pharmacological treatments to achieve both symptomatic improvement and long-term histological remission. Clinicians typically gauge the need for repeat dilations by monitoring patient symptoms, endoscopic appearance, and the esophageal luminal diameter over time [22,27].

Prognosis

The long-term prognosis for patients with eosinophilic esophagitis (EoE) remains uncertain due to the relatively recent recognition of the disease. Some individuals remain asymptomatic, while others experience persistent symptoms and progressive esophageal narrowing, which can lead to esophageal remodelling, including the development of strictures, Schatzki rings, or even achalasia, significantly impairing swallowing function [30].

EoE follows a chronic, relapsing course, with symptomatic flare-ups and periods of remission. While spontaneous resolution has been reported in some cases, most patients require maintenance therapy to prevent progression [31]. The duration of untreated disease correlates with complication risk, emphasizing the importance of early diagnosis and intervention [30,31]. While dietary and pharmacologic treatments effectively manage symptoms and reduce eosinophilic inflammation, they do not alter the natural course of EoE. Many patients require long-term therapy, with periodic esophageal dilation for fibrostenotic complications. Despite its chronic nature, EoE remains benign, with no established link to esophageal malignancy. Further research is needed to clarify disease progression and refine long-term management strategies [30,31].

Conclusions

Eosinophilic esophagitis (EoE) is a chronic, immune-mediated disease that significantly impacts patients' quality of life. Over the past decades, advancements in understanding its pathophysiology, epidemiology, and treatment strategies have led to improved management approaches. While no definitive cure exists, a combination of dietary modifications, pharmacological therapy, and endoscopic interventions provides effective symptom relief and disease control. Early diagnosis and long-term maintenance therapy are critical to preventing complications such as esophageal remodelling and strictures. Further research is necessary to refine treatment protocols, explore novel therapeutic options, and better understand the disease’s natural progression. Continued collaboration among specialists in gastroenterology, allergology, and immunology will be essential in optimizing patient outcomes and advancing the field of EoE management.

Disclosure

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