Eosinophilic esophagitis (EoE) is a chronic inflammatory condition characterized by symptoms of esophageal dysfunction and eosinophilic infiltration of the esophageal mucosa. The diagnosis requires esophageal biopsies demonstrating at least 15 eosinophils per high-powered field following a course of high-dose proton pump inhibitors. Management of EoE consists of the three Ds: drugs, dietary therapy, and esophageal dilation. In this review, we discuss the epidemiology, pathogenesis, diagnosis, and treatment of EoE to include the role of emerging therapies.

**INTRODUCTION AND EPIDEMIOLOGY**

Eosinophilic esophagitis (EoE) is a chronic inflammatory condition characterized by symptoms of esophageal dysfunction and eosinophil infiltration of the esophageal epithelium. It is three times more common in males than females, and is strongly associated with other atopic disorders. It has a prevalence of 57/100,000 persons in the United States, and is present in 12–22% of patients undergoing upper endoscopy for the evaluation of dysphagia. EoE is also present in as high as 10% of those presenting with dysphagia with endoscopically normal mucosa.

Although clinicians are becoming more familiar with EoE, the increase in prevalence observed cannot be simply attributed to heightened recognition alone. There are several theories to explain this increase in prevalence. The hygiene hypothesis asserts that, as rates of infectious diseases decrease worldwide, rates of allergic and immunologic diseases increase. This may be because of changes in immune reliance from T helper type 1 (Th1) cells to T helper type 2 (Th2) cells, or because of decreases in antigen exposure and eosinophil adhesion to fibronectin. EoE is also associated with increases in expression of thymic stromal lymphopoietin, an intercellular adhesion molecule markedly decreased in esophageal biopsies of EoE patients. This decrease leads to impaired barrier function seen in EoE. Downregulation of DSG1 also overlaps with upregulation of periostin, an extracellular matrix molecule that facilitates eosinophil adhesion to fibronectin. EoE is also associated with increases in expression of thymic stromal lymphopoietin, a cytokine that increases allergic inflammation. In biopsies of EoE patients, there is increased thymic stromal lymphopoietin and basophilic, suggesting a component of T cell-independent inflammation. However, serum levels of these biomarkers do not correlate well with EoE activity, and therefore the utility of measuring these proteins remains limited.

**PATHOGENESIS AND HISTOPATHOLOGY**

The mucosa of the esophagus consists of nonkeratinized stratified squamous epithelium in three layers: the stratum corneum, stratum spinosum, and stratum germinativum. The stratum germinativum, or basal layer, is not more than 3 cells thick in a normal esophageal epithelium. In EoE, the total epithelium is thickened, particularly in the basal zone, to more than 3 cells. Other findings include dilated intercellular spaces (spongiosis), fibrosis of the lamina propria, microabscesses, and dense eosinophilic infiltration. In addition to an increase in eosinophil count, biopsies in EoE patients demonstrate an increase in tissue mast cells, T cells, increased expression of tumor necrosis factor-α, interleukin (IL)-5, and Th2-related cytokines.

Many cytokines are implicated in the EoE inflammatory cascade. Eotaxin-3 (CCL26) is a highly upregulated chemokine in EoE that promotes eosinophil migration from the blood stream into mucosal tissue and correlates well with eosinophilia and mastocytosis. IL-13 activates local inflammation in Th2-related diseases, and is increased in the mucosa of EoE patients. IL-13 also downregulates desmoglein-1 (DSG1), an intercellular adhesion molecule markedly decreased in esophageal biopsies of EoE patients. This decrease leads to impaired barrier function seen in EoE. Downregulation of DSG1 also overlaps with upregulation of periostin, an extracellular matrix molecule that facilitates eosinophil adhesion to fibronectin. EoE is also associated with increases in expression of thymic stromal lymphopoietin, a cytokine that increases allergic inflammation. In biopsies of EoE patients, there is increased thymic stromal lymphopoietin and basophilic, suggesting a component of T cell-independent inflammation. However, serum levels of these biomarkers do not correlate well with EoE activity, and therefore the utility of measuring these proteins remains limited.

**DIAGNOSIS**

The diagnosis of EoE requires symptoms of esophageal dysfunction, mucosal biopsies that demonstrate at least 15
eosinophils per high-powered field, and persistence of esophageal eosinophilia after a trial of high-dose PPI (see Figures 1 and 2). Secondary causes of esophageal eosinophilia should be ruled out, such as eosinophilic gastrointestinal diseases, celiac disease, Crohn’s disease, hypereosinophilic syndrome, achalasia, and graft-vs.-host disease.

The most common clinical symptom of EoE is dysphagia that may be intermittent. There can be significant delay in diagnosis, with one study reporting the presence of symptoms for 6 years before diagnosis. In children, EoE presents with emesis, abdominal pain, feeding refusal, weight loss, and failure to thrive. Adults, however, classically present with dysphagia and food impactions. Race may influence clinical presentation. African Americans with EoE are more likely to experience heartburn, whereas Caucasians are more likely to present with dysphagia. EoE patients are likely to have other atopic diseases, most commonly allergic rhinitis, and also asthma, dermatitis, and food allergies. In one study, 50% of patients with EoE had a positive skin test to at least one food allergen, and 93% had a positive skin test to at least one inhalant. The most common food allergens identified were peanuts, egg whites, wheat, soybeans, cow’s milk, and tree nuts. Patients with EoE can have several endoscopic findings. These include concentric rings (fixed or transient) (see Figure 3), longitudinal furrows (see Figure 4), white plaques (see Figure 5), reduced mucosal vascularity, fragile or crepe-like mucosa, and stricture (dominant or diffusely narrow esophagus) (see Figure 6). Rings and furrows are the most common endoscopic findings, seen in 44% and 48% of patients respectively. The sensitivity of these endoscopic features for EoE is low (48% rings, 40% furrows, 15% strictures, and 27% white plaques), but the specificity is high (91% rings, 95% linear furrows, 95% strictures, and 94% white plaques). Up to 10% of EoE patients have no endoscopic findings, emphasizing that biopsies should be taken from all patients undergoing endoscopic evaluation for dysphagia. EREFS (Eosinophilic Esophagitis Endoscopic Reference Score), a classification system developed to standardize grading of endoscopic findings, has demonstrated good interobserver variability. This classification system assesses and scores each of the major endoscopic findings: rings (grades 0–3), exudates (grades 0–2), furrows (grades 0–1), mucosal edema (grades 0–1), and strictures (grades 0–1).
Radiographic examination is of limited value in the diagnosis of EoE, although in certain circumstances, barium esophagograms can help detect subtle strictures or diffuse luminal narrowing. 

Biopsies are necessary to establish the diagnosis of EoE. Based on current clinical guidelines, the diagnosis requires 15 eosinophils per high-powered field or more in at least one esophageal site. EoE can be patchy and a single esophageal biopsy has low sensitivity (55%) for diagnosis. Increasing to a minimum of six biopsies increases sensitivity to 99%, with biopsies in the mid and distal esophagus to help distinguish EoE from eosinophilia associated with reflux esophagitis. Targeting biopsies in furrows and exudates can also improve sensitivity. The mucosa may feel fibrotic while taking biopsies, requiring more effort to sample the mucosa. This is known as the “tug” sign or “pull” sign that has a sensitivity of 76% and specificity of 98% for EoE.

The diagnosis of EoE requires mucosal eosinophilic infiltration that persists despite therapy with a PPI. Up to 50% of patients with an EoE phenotype respond clinically and histologically to PPI therapy. This entity, called PPI-responsive esophageal eosinophilia (PPI-REE), is indistinguishable clinically, endoscopically, and histologically from EoE. There are two prevailing theories for the pathogenesis of PPI-REE. The first is that PPIs heal a defective barrier where acid increases mucosal permeability allowing the influx of allergens that activate a Th2-mediated inflammatory response. The second is that PPI therapy can reduce levels of key mediators of EoE such as eotaxin-3, IL-4, IL-5, and IL-13 independent of their antisecretory effects. Even though biomarker staining can distinguish EoE from gastroesophageal reflux disease, this technique has not been useful in differentiating EoE from PPI-REE. Recent data support that EoE and PPI-REE share a similar molecular basis. Treatment with PPIs, similar to treatment with steroids, reverses the molecular signature of EoE.

As esophageal biopsies often necessitate upper endoscopy with sedation, new diagnostic methods are being investigated. Brush cytology with the Cytosponge is 69% sensitive and 67% specific for esophageal eosinophilia in the proximal esophagus, 72% sensitive and 75% specific for the distal esophagus, and correlates well with samples obtained during endoscopy. The Enterotest (HDC, Milpitas, CA) is a noninvasive capsule filled with 90 cm of string designed to sample luminal contents; in a pediatric population, the Enterotest accurately identified eosinophil-derived proteins in patients with EoE, with good sensitivity and specificity for esophageal eosinophilia. Further studies are needed before these technologies are implemented into clinical practice. Serum studies of proteins associated with EoE (such as IL-13 and eotaxin-3) have not yielded useful biomarkers for diagnosis or measuring response to therapy to date.

There are two distinct phenotypes described in EoE. Patients with endoscopic findings limited to transient rings, furrows, and white plaques have the inflammatory phenotype, and those with fixed rings or strictures have the fibrostenotic phenotype. Although fibrostenosis is uncommon in children, adult EoE patients present with both phenotypes. A study of the Swiss EoE database demonstrated that the duration of symptoms before diagnosis was the only factor that predicted stricture formation, suggesting that untreated inflammation is a major determinant of symptom development. These results were validated in a US institution where a significant difference in delayed diagnosis was observed in patients with an esophageal stricture vs. those with a patent esophageal diameter. Another retrospective study analyzing the differences between EoE phenotypes demonstrated that the likelihood of fibrostenotic disease doubled for every 10-year increase in age. These studies collectively suggest that the natural course of EoE includes the development of strictures over time that may be delayed or halted with treatment. Although there are no guidelines that delineate intervals for endoscopic surveillance of EoE patients, we conduct endoscopy annually in patients who have not shown resolution of esophageal eosinophilia.

The EndoFLIP system (Crospon Medical Devices, Galway, Ireland) is a device and program that uses impedance planimetry of a cylindrical bag to assess intraluminal pressure, and can give a measure of esophageal distensibility. In a study comparing EndoFLIP assessments in 35 EoE patients to 15 control patients, EoE patients had significantly less distensibility that did not correlate to eosinophilic density. This implicates esophageal dysmotility in symptom generation, and...
may explain why symptom resolution may not parallel resolution of eosinophilia.

MANAGEMENT

The management of EoE consists of the three Ds: drugs, diet, and dilation (see Figure 7). A major challenge in the management of EoE is the lack of well-defined therapeutic end points. From a patient's perspective, the goal is to improve symptoms and reduce the recurrence of food impaction. However, clinical end points are difficult to assess as patients develop adaptive techniques by chewing more carefully, eating slowly, and eliminating problem foods. The development of EEsAI (Eosinophilic Esophagitis Activity Index), a validated EoE symptom questionnaire that uses patient-reported outcomes, may help standardize clinical outcomes in EoE patients. Histologic outcomes are also difficult to assess. It is unclear which threshold for eosinophilia defines response to therapy, and symptoms do not accurately reflect degree of histologic inflammation. Finally, endoscopic findings may not correspond to either symptoms or histology. It may be that future studies may use distensibility and submucosal fibrosis (as measured by systems like EndoFLIP) as treatment outcomes.

Topical corticosteroids. Topical steroids remain the mainstay of EoE treatment. Steroids inhibit proinflammatory cytokines in the mucosa, thereby reducing eosinophil mucosal migration. Both swallowed aerosolized fluticasone and oral viscous budesonide induce a histologic and clinical response in randomized controlled trials. In a pediatric study,
swallowed fluticasone at 440 μg twice daily led to histologic improvement (> 90% reduction in eosinophilia) after 3 months of treatment in half of subjects.66 In adults, histologic response was seen in 62% of subjects after treatment with 880 μg twice daily for 8 weeks.67 Recently, 65% of adult and pediatric patients treated with high-dose fluticasone (1,760 μg) for 3 months experienced histologic response.56 Extending therapy beyond 3 months in steroid-resistant patients did not help induce remission. It is noteworthy that in all three controlled trials, there was no significant difference in clinical symptoms between fluticasone and placebo.56,66,67 Although symptoms commonly recur in responsive patients after discontinuation of fluticasone,71 one recent study concluded that treatment with swallowed corticosteroids had a significant reduction in the frequent of food impactions over a 5-year follow-up period.72

Several controlled trials have used oral budesonide as a treatment option in EoE. In one pediatric study, oral viscous budesonide improved symptoms, endoscopic findings, and histologic eosinophilia in 87% of patients compared with a 0% placebo response.68 Another study in EoE children demonstrated that administration of medium-dose (cumulative dose > 1.4 mg/day) and high-dose (cumulative dose > 2.8 mg/day) budesonide led to significant decreases in histologic eosinophilia and EoE symptoms scores.69 In adults, 1 mg budesonide administered in nebulized form for 15 days reduced symptoms and improved histology.70 After 50 weeks of therapy, patients maintained on low-dose budesonide (0.5 mg daily) had a better response compared with placebo, yet experienced an increase in eosinophilia without a significant difference in symptom response.73 In an open-label trial, viscous budesonide had more mucosal contact time and was more effective at reducing esophageal eosinophilia than swallowed nebulized budesonide.74 As budesonide respules are not intended for esophageal delivery, a slurry mixture (e.g., with a sugar substitute or honey) can be prepared by patients or their families. Otherwise, budesonide can be administered by continuously swallowing a nebulizer. A recent study showed equal efficacy of an effervescent budesonide formulation compared with an oral viscous formulation, with 80% of patients preferring the effervescent formulation and only 17% preferring the viscous formulation.75 There are no trials comparing fluticasone with budesonide. Adverse events with topical steroids are minimal, with esophageal candidiasis being the most common (5–26%).67,68,70,76,77 that appears to be dose dependent.

**Dietary therapy.** Specialized diets are an effective treatment strategy in most EoE patients. There are three types of dietary treatment: elemental, targeted elimination, and empiric elimination of the most common allergen culprits. Studies in children have demonstrated that an elemental diet, which consists of an amino-acid free formula that eliminates all food triggers, is highly effective for both inducing histologic response and improving clinical symptoms.78–80 Elemental diet was 96% effective in inducing response in 10 children with EoE.80 Upon food challenge, patients redeveloped clinical symptoms and esophageal inflammation. In the only controlled study in adults, elemental diet was 72% (13/18) effective in inducing histologic response, defined as achieving ≤10 eosinophils/high-powered field. However, 38% failed to adhere to the diet and all patients experienced esophageal inflammation when food was introduced.79 Although elemental diet is an effective treatment option, it has several drawbacks. It is expensive, may require a feeding tube for administration in children, can limit quality of life, has poor tolerability particularly in adults, and is not sustainable in the long term.

Given the challenges in maintaining this treatment, a diet tailored to remove specific allergens implicated in inflammation is more desirable. In a retrospective study of 22 adult EoE patients undergoing targeted food elimination based on results of allergy testing, 68% experienced clinical improvement, and 53% experienced endoscopic improvement with a significant improvement in posttreatment eosinophil count.81 In a retrospective cohort study in EoE children, 75% experienced clinical and histologic improvement after eliminating foods that tested positive on skin prick testing and atopic patch testing.82 Despite these studies demonstrating favorable outcomes when using allergy testing to eliminate foods, other controlled studies have not had similar outcomes. Gonsalves et al.83 reported that the predictive value of skin prick testing was only 13% in patients undergoing empiric elimination diets. Another effective strategy in treatment is the six-food elimination diet (SFED). This diet eliminates the six most common alimentary allergens: wheat, milk, eggs, nuts, soy, and seafood. Patients are monitored clinically and histologically as each food is slowly introduced to allow for identification of allergens. Some patients have more than one food trigger. In children, SFED led to 74% histologic remission. Eggs, soy, milk, and wheat were the most common allergens identified.84 In adults, a similar approach achieved 64% histologic remission with wheat and milk being the most common allergens identified.81 A prospective controlled study from Spain showed similar results with a 73% histologic response to empiric diet elimination.85 Of note, legumes were also eliminated in this study and found to be a cause in 23% of patients. Elimination diets, with culprits identified upon reintroduction, appear more effective than identification and elimination of allergens via skin prick testing.83 The treatment was durable with histologic remission seen after 3 years of therapy in patients who continue to avoid trigger foods.85 A recent study showed that a four-food group elimination diet (FFGED) eliminating dairy, eggs, legumes, and wheat yielded a 54% remission rate. In nonresponders, SFED led to an additional 18% remission.86 The number of endoscopies required to assess for histologic response is not practical in nonresearch settings and is a potential limitation of empiric diets. In a motivated patient who can adhere to specialized diets, FFGED or SFED are reasonable first-line approaches in lieu of topical steroids.

**Dilation.** EoE patients with features of fibrostenosis (fixed rings or strictures) benefit clinically from dilation, even though dilation does not influence esophageal inflammation or degree of eosinophilia. For this reason, clinicians should use dilation in conjunction with medical or dietary therapy. A study followed adult EoE patients for several years and demonstrated that dilation was effective in improving symptoms of 10/11 patients for a mean of 8 months.87 In a study of steroid-naïve EoE patients treated only with PPI, dilation every other year was
effective in maintaining clinical remission.\textsuperscript{68} Options for dilation include bougie, wire-guided dilator, or through-the-scope balloon dilator. No studies to date have compared the efficacy of these methods of dilation. In several retrospective studies, dilation improved dysphagia in 67–83% of patients, but improvement appears to be transient (~1–2 years).\textsuperscript{89–91} Although there has historically been a concern about increased perforation rates when dilating patients with EoE,\textsuperscript{92,93} recent studies have shown a perforation rate of 0.3%, similar to that of dilation of other esophageal conditions.\textsuperscript{90,94,95} Multiple dilations, especially in patients presenting with a critical esophageal stricture, appears to be a safe treatment.\textsuperscript{96} One study assessing esophageal distensibility has suggested that a target diameter of 17 mm may correlate to a lower risk of food impactions.\textsuperscript{90} Patients should be counseled about chest pain that commonly occurs after dilation in EoE.\textsuperscript{89,95} Once a mucosal tear develops after passage of the dilator or with the balloon, no further dilation is recommended during that session. Our practice is to target an esophageal diameter of at least 15 mm for patients to experience a sustained improvement in swallowing. Although there are no trials comparing dilation methods, we generally rely on bougie dilators that may result in dilation of strictures that were not otherwise detected during endoscopy. Empiric dilation (i.e., without an identified fixed ring or stricture) in EoE patients is controversial and data are lacking for its effectiveness. In such patients, dysphagia may be secondary to other causes, such as reduced esophageal compliance.\textsuperscript{97}

**Emerging therapies.** Given that at least one-third of EoE patients are unresponsive to topical steroids or specialized diets, researchers continue to explore options for antieosinophilic medications. Leukotriene inhibitors, such as montelukast, have not shown efficacy in maintaining steroid-free remission in EoE patients.\textsuperscript{98,99} Therapies include drugs targeting key cytokines involved in the pathogenesis of EoE, such as IL-4, IL-5, and IL-13. There have been three well-designed studies targeting IL-5 cytokine.\textsuperscript{100–102} The largest was conducted in 226 children with EoE. All subjects received 2–4 infusions of anti-IL-5 treatment and achieved a significant eosinophil reduction, but symptom improvement was not significantly different from placebo.\textsuperscript{102}

In the only anti-IL-13 study performed to date, 23 adult EoE patients were randomized in a 2:1 ratio to receive 3 doses of anti-IL-13 every 4 weeks.\textsuperscript{103} The primary end point (75% decrease in eosinophilia) was achieved in 40% of drug patients receiving anti-IL-13 treatment vs. 13% of placebo patients. Again, a significant difference was noted in histologic response, but there was no significant difference in clinical response.\textsuperscript{103} A larger international multicenter study evaluating the role of anti-IL-13 treatment for EoE is underway.

A recent randomized controlled trial compared Omalizumab, an anti-IgE medication, with placebo in EoE patients.\textsuperscript{104} Neither the primary end point (histologic improvement) nor the secondary end point (clinical response) was met in this study. This study concluded that EoE is not primarily an IgE-mediated allergy.\textsuperscript{104}

Chemotractant receptor-homologous molecule (CRTH2) is expressed in Th2 cells and mediates recruitment and activation of eosinophils.\textsuperscript{105} One randomized double-blinded placebo-controlled trial examined the role of CRTH2 antagonist (OC000459) in patients with severe EoE refractory to topical steroids.\textsuperscript{106} Although a significant improvement in eosinophil count was demonstrated in the 14 EoE patients treated with 100 mg of OC000459 twice daily for 8 weeks, patients did not achieve complete histologic response.\textsuperscript{106}

In conclusion, EoE is a chronic inflammatory condition that is one of the most common causes of dysphagia. Its diagnosis requires endoscopic biopsies with demonstration of dense eosinophilia that persist after a course of PPI therapy to rule out PPI-REE. Treatment options available for patients include topical steroids, specialized diets, and dilation for critical strictures. Given the complexity in management of EoE, we recommend a multimodal approach with close follow-up and monitoring of symptoms to guide management. A multidisciplinary approach is useful, particularly in those patients with significant coexisting atopic conditions. Emerging therapies, although effective in reducing esophageal eosinophilia, have not yet demonstrated complete histologic response or improvements in clinical response.

**CONFLICT OF INTEREST**

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