World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(CASE REPORT)

Check for updates

Eosinophilic esophagitis: Case report and a brief review of the literature

Riley Hagy [2], Allison Rojas [2], Hadiyah Page [3], Michael Mitchell [3], Ross Jenkins [2], Katherine Castrichini [2], Arash Bakian [2], Shane Ganga [4], Jessica Jahoda [1, 2] and Mohamed Aziz [1, *]

[1] *Research Writing & Publication (RWP), LLC, NY, USA.*
[2] *American University of the Caribbean School of Medicine, USA.*
[3] *Ross University School of Medicine, Barbados.*
[4] *St. George's University School of Medicine, Grenada.*

World Journal of Biology Pharmacy and Health Sciences, 2025, 23(03), 044-051

Publication history: Received on 25 July 2025; revised on 29 August 2025; accepted on 02 September 2025

Article DOI: https://doi.org/10.30574/wjbphs.2025.23.3.0799

Abstract

Eosinophilic esophagitis (EoE) is a chronic immune-mediated esophageal disease with symptoms related to esophageal dysfunction and histopathologic features of eosinophil-predominant inflammation. We present the case of a 32-year-old man who was referred for evaluation of a history of 7-month-long progressively worsening solid and subsequently liquid dysphagia. In addition, the patient reported frequent episodes of food impaction, retrosternal chest pain, and proton pump inhibitor-resistant heartburn. The patient had a background of different atopic diseases: he presented with Asthma since childhood, allergic rhinitis, eczema, and food allergies as well.

Physical examination was significant for nasal congestion, eczematous changes, and pulmonary wheezing. Laboratory findings showed a high total IgE, peripheral eosinophilia, and low vitamin B12 and folate levels. Barium esophagram displayed the presence of a ringed esophagus with concentric rings and longitudinal furrows. Endoscopy revealed several characteristics of EoE, such as mucosal rings, furrows, white exudation, and reduced vascularity. The diagnosis was confirmed by histopathologic analysis of several esophageal biopsies in which more than 15 eosinophils per high-power field were seen in more than one field, in addition to basal cell hyperplasia and eosinophilic layering. Therapy was with topical steroids (budesonide viscous slurry) swallowed and a six-food elimination diet, with usual treatment of comorbid atopic conditions.

Within five weeks, the patient experienced clinical improvement in dysphagia and fewer food impaction episodes. Histologic remission was reached after 10 weeks. Four-year follow-up showed continued remission with no significant complications in the context of a multidisciplinary care plan. The case emphasizes the need to detect EoE early in patients with chronic dysphagia, food impaction, inadequate response to PPI treatment, and atopic history, and the value of a combination of pharmacologic and dietary treatment in a lasting remission.

Keywords: Eosinophilic esophagitis; Immune-mediated; Esophagus; Endoscopy; Biopsy

1. Introduction

Eosinophilic esophagitis (EoE) is an upper GI inflammatory condition with unknown etiology. Infiltration of the esophageal mucosa by a large number of eosinophils is a hallmark of this disease. Due to improved diagnostic methods, the number of cases of this disease is significantly increasing, causing a large number of cases of upper GI morbidities. It may also be due to an actual increase in the number of cases of EoE. [1] [2] [3] Patients experiencing EoE usually present with dysphagia, food impaction, and retrosternal chest pain. Diagnosis of EoE is established with the presence of 15 eosinophils in one HPF of multiple esophageal biopsies. [4] [9]

* Corresponding author: Mohamed Aziz.

It is important to distinguish EoE from other causes of esophageal eosinophilia, especially gastroesophageal reflux disease (GERD), because the treatment options vary dramatically. [9] The disease is prominently found in persons who have had prior allergic predispositions such as Asthma, allergic rhinitis, eczema, and food allergy conditions. [4]

EoE has the potential to cause progressive esophageal remodeling, esophageal strictures, esophageal rings, and a small-caliber esophagus that can result in debilitating dysphagia and esophageal food impaction that require endoscopic treatment. Therefore, early diagnosis along with adequate management is vital to preventing any potential complications and the subsequent lowering of the quality of life of the patient. Nevertheless, there are limited therapeutic options (dietary elimination, proton pump inhibitors (PPIs), and topical corticosteroids) that are combined to offer clinical and histological remission. [2] The purpose of this case report is to shed light on the diagnostic difficulties, common clinical and radiological manifestations, pathophysiology, and successful treatment options of EoE by using a detailed patient case presentation.

2. Case presentation

A 32-year-old male presented to the clinic with a 7-month history of progressive dysphagia, which initially occurred with solid food, but gradually involved liquid food. He expressed the dysphagia as a feeling of food sticking in the chest that was usually accompanied by the need to use liquids to facilitate food going down the throat. He had a history of food impaction, especially with thick foods such as meat or bread, and sometimes had to visit the emergency department to be disimpacted. Other reported associated symptoms included chest pains that were experienced as cardiac pains, heartburn that did not show a response to proton pump inhibitors (PPI), and regurgitation of undigested food. The symptoms had been inconsistent and initially were attributed to eating too fast or stress. Standard-dose PPI therapy that had been used previously and had minimal effect in resolving the symptoms. He also reported recent weight loss based on a change of diet and avoidance of food habits.

Personal history revealed a strong atopic background, including childhood asthma, allergic rhinitis, eczema, and some food allergies. Family history included similar complaints, but to a lesser degree, with his younger brother. Significant findings in physical examination included nasal congestion, eczematous skin changes, and wheezing on pulmonary examination. The patient's weight was less than expected for his age. The clinical differential diagnosis at this presentation included gastroesophageal reflux disease, achalasia, esophageal stricture from other causes, eosinophilic esophagitis (EOE), esophageal carcinoma, pill esophagitis, infectious esophagitis, and other inflammatory esophageal conditions. The combination of dysphagia, food impaction, poor PPI response, and strong atopic background created a compelling clinical picture suggestive of EoE, warranting further diagnostic evaluation.

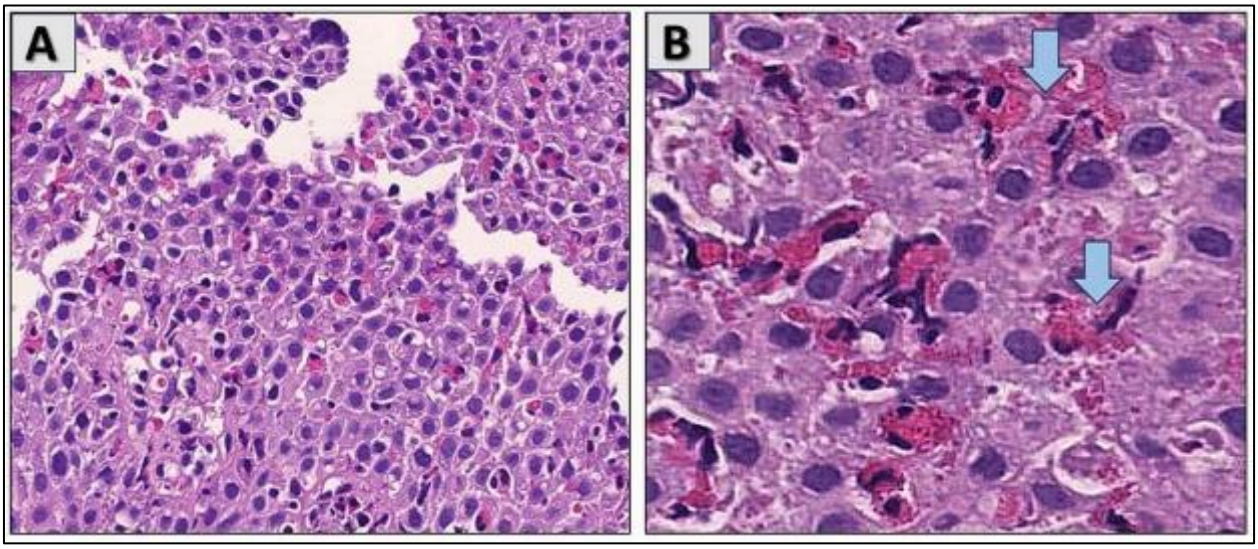
Laboratory studies were supportive of the atopic phenotype. Total IgE levels were elevated, and specific IgE testing radioallergosorbent test (RAST) to common food and environmental allergens failed to identify triggers. Complete blood count showed peripheral eosinophilia. Vitamin B12 and folate were low. A barium esophagram (BE) was performed to evaluate any obstruction and identify changes in esophageal anatomy. BE studies revealed a "ringed esophagus" appearance with multiple concentric rings and a luminal narrowing pattern due to longitudinal furrows. No mucosal tears were identified. Esophagogastroduodenoscopy "endoscopy" was performed, which revealed concentric rings, longitudinal furrows, white exudates, decreased vascular markings, mucosal friability, and potential stricture formation. Endoscopy findings were compatible with EoE.

Tissue diagnosis was essential, and the biopsy protocol was crucial for diagnosis. Considering that EoE is usually patchy, multiple biopsies (7 specimens) were obtained from the proximal, mid, and distal esophagus. Biopsies were obtained regardless of endoscopic appearance, and areas of normal-appearing mucosa were also included in the sampled tissue. Microscopic findings demonstrated the hallmark feature of dense eosinophilic infiltration. The tissue sampled revealed >15 eosinophils per high-power field (hpf) in more than one microscopic field. Additional histologic features included surface epithelial layering of eosinophils and basal cell hyperplasia exceeding 30% of epithelial thickness; however, no eosinophilic abscesses were identified in the sampled tissue. (Figure 1 A, B) Pathological differential diagnosis included gastroesophageal reflux disease (but typically <5 eosinophils/hpf, and good response to PPI), pill esophagitis, infectious esophagitis (particularly herpes or candida), hypereosinophilic syndrome with esophageal involvement, and, less likely, esophageal carcinoma with secondary eosinophilia. Crohn's disease with esophageal involvement and connective tissue disorders were also included in the differential diagnosis. The clinical presentation, endoscopic, and histomorphologic findings were consistent with the diagnosis of eosinophilic esophagitis (EoE), and there was no need for immunohistochemistry (IHC) studies or molecular testing for confirmation.

Treatment started with topical steroids, budesonide viscous slurry (1-2 mg twice daily), with directions to not eat or drink for 30 minutes post-administration to maximize mucosal contact time. The patient responded well, and there was

no need for systemic corticosteroids. Dietary management included equally effective alternatives, utilizing elimination diets. The six-food elimination diet (6FED) was advised to remove milk, eggs, wheat, soy, nuts, and seafood. Other symptoms, such as asthma and eczema, received standard treatment, and the patient responded well to the treatment. In 5 weeks, there was marked improvement in dysphagia and food impaction episodes. A repeat histopathologic evaluation at 10 weeks of treatment showed histologic remission with <10 eosinophils per high-power field.

Follow-up included clinical assessment every 3-6 months, and surveillance endoscopy was performed every 1-2 years. After four years of a multidisciplinary approach involving gastroenterology, allergy/immunology, and nutrition specialists to optimize both short-term symptom control and long-term disease modification, most of the symptoms were resolved, and no significant complications.



1A Intermediate power view showing abundant intramucosal eosinophils (H&E stain X40); **1B:** High power view showing >15 eosinophils per high-power field (hpf), and eosinophilic degeneration (Blue arrow) (H&E stain X60)

Figure 1 Histomorphology of eosinophilic esophagitis

3. Discussion

3.1. History, Epidemiology, and Risk Factors

Eosinophilic esophagitis (EoE) has expanded drastically since its description in the 1990s as a distinct clinical pathology from gastroesophageal reflux disease. [5] Starting as a rare condition, it is now seen as a chronic, immune-mediated, antigen-driven esophageal disorder and identified as esophageal dysfunction and eosinophil predominant inflammation. EoE has evolved in terms of diagnostic criteria, with guidelines expressing the importance of clinical and histological thresholds in relation to therapeutic response. [3] EoE is now seen as a common cause of food impaction in young adults and a symptomatic cause of chronic dysphasia. [6]

EoE has gained increased recognition worldwide over the past 30 years, likely due to increased testing, clinical knowledge, and a rise in incidence. It is most commonly found in young males, with a male-to-female ratio of 3:1. There has been an estimated prevalence of 0.5-1 case per 1,000 in Western countries, similar to Crohn's disease. The incidence is reported at 5-10 cases per 100,000 annually in North America and Europe. [6]

EoE risk factors range from atopic predispositions, genetic susceptibility, environmental factors, demographic factors, and diet. [1] 70-80% of EoE patients have a history of asthma, allergic rhinitis, atopic dermatitis, or food/environmental allergies. [7] Dietary factors in the western world may also contribute to disease occurrence, such as vitamin D deficiency and low folate/B12 (diet or malabsorption). [2] Genetic susceptibility can be seen with familial clustering and gene loci with CAPN14 on chromosome 2p23, associated with esophageal epithelial barrier dysfunction. [8]

International recognition by many research organizations, including the American College of Gastroenterology (ACG) and the European Society of Eosinophilic Esophagitis (EUREOS), has gathered a consensus on factors that are endorsed globally. EoE is mentioned as having symptoms of esophageal dysfunction, including dysphagia, food impaction, and

PPI-resistant influx. Including endoscoping findings of rings, furrows, white plaques, edema, and strictures. Histopathological findings included ≥15 eosinophils/hpf on esophageal biopsy. [6]

3.2. Clinical presentation and Imaging findings.

EoE presentation differs greatly depending on age group, but some symptoms are highly indicative of the disease regardless of age bracket. The symptoms most frequently experienced in adults are dysphagia, especially to solid food, and food impaction, in which the food cannot pass down the esophagus and needs emergency removal by endoscopy. [9] The patients usually complain of the feeling of food sticking and chest pain and heartburn resistant to usual treatment with proton pump inhibitors (PPIs), which is the most common distinction between the disease and gastroesophageal reflux disease (GERD). [10] Other, less pronounced symptoms may consist of regurgitation, nausea, and abdominal pain. The long-term character of these symptoms tends to become a huge burden in terms of quality of life, dietary restrictions, and even weight loss, such as the one observed in our patient. [4] A personal or family history of atopic diseases, including asthma, allergic rhinitis, eczema, and food allergy, is often present in patients with EoE and should be considered a significant clue to diagnosis. [2]

In EoE, imaging studies (especially barium esophagram, BE) can demonstrate characteristic but non-specific findings. Typical BE appearances are a ringed esophagus or trachealization in which the esophagus has multiple concentric rings, and longitudinal furrows in which there are linear depressions of the esophageal mucosa. [11] Other findings can include narrowing of the lumen and strictures, as well as a small-caliber esophagus. Although BE may raise the suspicion of EoE, endoscopy is required to identify and characterize the esophageal morphology. [12] The most significant features predictive of EoE include esophageal rings, longitudinal furrows, white exudates (representing eosinophil microabscesses), and esophageal narrowing. [11] [12]

3.3. Pathology Diagnosis and Ancillary Studies

The diagnosis of eosinophilic esophagitis (EoE) begins clinically. The initial symptoms to suspect are of esophageal dysfunction, like dysphagia, which is one of the earliest symptoms. [9] Other symptoms include food impaction, heartburn, and chest pain. For children, symptoms include regurgitation, vomiting, or feeding dysfunction. Some patients will present with progressive dysphasia and a history of food allergy symptoms, which are highly suggestive of esophageal dysfunction. These observations are characteristic of EoE, particularly in adolescents and adults, and constitute the basis to proceed with further investigation. [4] [13]

An upper endoscopy (esophagogastroduodenoscopy, EGD) is performed and may reveal concentric rings (trachealization), linear furrows, and mucosal pallor. These findings are graded using the EoE Endoscopic Reference Score (EREFS). This validated classification system standardizes the endoscopic evaluation of EoE by quantifying five key features: edema, rings, exudates, furrows, and strictures. [14] Usually, 7-32% of patients may have a normal-appearing esophagus, especially during the early stages of the disease. [13] Multiple esophageal biopsies are obtained during the endoscopy—usually, three from the distal and three from the mid-to-proximal esophagus. As per current guidelines, a minimum of six biopsies from at least two different esophageal locations are recommended to address the patchy nature of eosinophilic infiltration. [5 Histological examination that demonstrates ≥15 eosinophils per high-power field (eosinophils/hpf) in multiple fields meets the diagnostic criteria for EoE. Additional findings included basal zone hyperplasia and eosinophilic microabscesses, further supporting the diagnosis. [15]

Other causes of esophageal eosinophilia should be considered and excluded. These include: gastroesophageal reflux disease (GERD); eosinophilic gastrointestinal disorders affecting other segments of the GI tract; hypereosinophilic syndrome; infections; drug reactions; and connective tissue diseases. [10] [15] Interestingly, a proton pump inhibitor (PPI) trial, which was once required to differentiate PPI-responsive esophageal eosinophilia from EoE, is no longer necessary to confirm the diagnosis, as recent evidence suggests these conditions represent the same pathogenic spectrum. [13]

Emerging diagnostic adjuncts include the esophageal string test (EST) and Cytosponge. Their use is to minimize invasiveness while sampling esophageal mucosa or eosinophil-derived proteins. However, their use is still investigational and is not yet incorporated into routine clinical practice. [16] Similarly, AI-assisted histology tools are under development to assist in quantifying eosinophilic inflammation and may become important for diagnostic reproducibility in the future. [2]

3.4. Treatment Strategies and Outcomes

The key to EoE management is symptom improvement, histologic remission (usually defined as <15 eosinophils per high-power field), prevention of disease progression (incorporating fibrotic remodeling and stricture formation), and long-term control at low doses and with reduced adverse events. [17] PPIs are an easily available first-line pharmacologic agent. They allow histologic remission in about 40-50 percent of patients, with a combined histologic response of 41.7 percent versus 13.3 percent in observational studies. Anti-inflammatory biologic agents, such as Dupilumab, can also be used in patients 12 and older who are nonresponsive to PPI therapy. [6] While systemic glucocorticoids can be used for patients with severe disease that is refractory to other treatment options, it is generally not recommended due to side effects. [10] A swallowed dose of topical steroids (mostly fluticasone or budesonide) is also a paradigm treatment modality. It has been demonstrated to induce remission by histologic criteria more than PPIs, with a rate of approximately 65% versus 13%, with placebo use. [2] There is strong evidence to support eosinophil reduction in the short-term (4-12 weeks), but less available data to support maintenance therapy. [18]

Dietary management with foods containing amino acids is also provided, and the response rate is ~90% in children and ~70% in adults. Diet elimination protocols offer a nonpharmacologic treatment option. However, they can be difficult for an individual to adhere to long-term, along with being costly, and certain foods being inaccessible or undesired. They can be in the form of six-food, four-food, or two-food elimination diets. One major limitation to an elimination diet approach involves repeat endoscopic biopsy evaluations during the food reintroduction period, which can be reduced by reducing the number of foods eliminated to 1-4 at a time. [14] These are of moderate efficacy, and step-up interventions (using less restriction initially and incrementally increasing) should be more acceptable and result in fewer endoscopies. [19] Diets based on allergy tests are less effective and less popular than the empiric elimination. [18]

Bio-therapeutics that modulate select immune pathways are an emerging modality of treatment. Dupilumab, which is an IL-4/IL-13 inhibitor, has recently been approved by FDA in patients (age >=12 years) and weight (>=40 kg). [20] Dupilumab is presently approved in patients with dysphagia or structural narrowing in whom initial medical or dietary therapy has failed. Dilation enhances the patency of the lumen, but not the counts of eosinophils. [18] Performed carefully, it is deemed to be comparatively safe, but it is necessary to counsel patients of the following risks: chest pains, bleeding, or perforation. Instead, modern recommendations (e.g., American College of Gastroenterology ACG) on the assessment of response and subsequent maintenance include a multimodal monitoring strategy by means of a combination of clinical examination, endoscopy, and histopathology. [5] Maintenance on the most efficacious first-line regimen (e.g., PPIs or topical steroids) will decrease long-term relapse risk due to the relapsing nature of EoE. [19]

With EoE being a chronic and intermittently relapsing inflammatory disorder, the prognosis can vary depending on how early the disease was diagnosed and how adherent the patient is to long-term treatments and surveillance. If diagnosed late or with inconsistent treatment, EoE can progress to long-term complications, such as esophageal remodeling, fibrosis, or strictures. [**10**]

3.5. Pathogenesis and Pathophysiology

Eosinophilic Esophagitis (EoE) is an uncommon immune-mediated disease. It is a complex interplay of genetic predisposition and environmental factors. An aberrant immune response predominantly involving type 2 helper T (Th2) cells is a central and strong factor in the etiology. [21] A long-lasting inflammation and consequent tissue remodeling of the esophageal wall occur due to heavy infiltration of the esophageal mucosa. [9] This is triggered by contact with specific food or an aeroallergen in individuals where there is a genetic predisposition leading to breakdown of the esophageal epithelial barrier. [2] A weakened epithelial defense permits more antigen invasion, which further heightens the immune response.

In EoE, the immune cascade is a Th2-biased inflammatory reaction. When an allergic person is exposed to an allergen, antigen-presenting cells stimulate Th2 lymphocytes that secrete important cytokines, including IL-5, IL-13, and IL-4. [22] IL-5 is a strong chemoattractant and activator of eosinophils, which stimulates their recruitment, survival, and activation in the esophagus tissue. [23] IL-13 is important in the expression of eotaxin-3 (CCL26) by the esophageal epithelial cells, which is yet another strong chemokine to attract eosinophils and increases its infiltration into the body. [23] IL-13 also plays a role in epithelial barrier dysfunction and fibrosis and tissue remodeling, which explain the typical symptoms of EoE, including strictures and rings. [24]

The inflammation and the eosinophilic infiltration are persistent, which causes structural and functional alterations of the esophagus. Eosinophil degranulation releases a variety of cytotoxic substances, such as major basic protein, eosinophil cationic protein, eosinophil-derived neurotoxin, and eosinophil peroxidase, that can destroy esophageal

epithelial cells directly. [25] This continual damage, along with the pro-fibrotic effect of Th2 cytokines, leads to esophageal remodeling, which is typified by hyperplasia of the basal cells, subepithelial fibrosis, and muscular hypertrophy. [26] These alterations are a cause of dysfunction of the esophagus, which is characterized by the occurrence of dysphagia and food impaction.

The progressive characteristic of the disease highlights the need to identify the condition early and to continue treatment over a long period of time to avoid irreversible fibrotic changes and preserve esophageal function.

3.6. What have we Learned from this Case?

This thorough case presentation of a 32-year-old male with EoE provides some useful information on how to diagnose the condition, treat it, and the long-term implications of the disorder. First, the case highlights how EoE symptoms can be insidious and progressive. The atypical presentation of the patient with dysphagia, initially to solids and then to liquids, and frequent food impactions and PPI-resistant heartburn, prompted the clinician to consider the condition in the presence of an atopic background. The fact that the initial misattribution of symptoms to eating habits or stress compounds the difficulty of diagnosing EoE early in the differential diagnosis process. This further highlights the need to consider EoE in the differential diagnosis early on.

Secondly, the case is a graphical demonstration of how a multidisciplinary approach is needed to diagnose and treat EoE effectively. It is a combination of clinical manifestations (dysphagia, food impaction, non-response to PPI, atopy), laboratory results (elevated IgE, peripheral eosinophilia), radiographic (ringed esophagus, longitudinal furrows), and endoscopic criteria (concentric rings, furrows, white exudates) that led to the diagnosis of EoE. Nevertheless, the ultimate diagnosis was based on histopathological confirmation by multiple biopsies of the esophagus, which exhibited >15 eosinophils per HPF, which further illustrates the importance of tissue diagnosis in EoE. This diagnostic workup is important to distinguish EoE from other conditions that have similar symptoms.

Lastly, the effective long-term treatment of this patient by the use of both topical steroids (budesonide viscous slurry) and a six-food elimination diet (6FED), resulting in clinical and histological remission, is an excellent example of positive treatment measures The long-term remission lasting 4 years with multidisciplinary care including gastroenterology, allergy/ immunology and nutrition specialists illustrates the significance of a comprehensive and multifaceted care to maximize patient outcomes and to avoid development of long-term complications like esophageal remodeling. This case is a lesson to clinicians to think of EoE in patients with chronic dysphagia and atopy, and to follow through a diagnostic pathway and then adopt an individualized comprehensive management to achieve permanent remission and improve quality of life.

4. Conclusion

Early diagnosis and complete management of EoE must be undertaken, particularly when dealing with patients with atopy reporting chronic dysphagia and food impaction. The process taken by this patient to move through the signs and symptoms of insidious onset, ultimately leading to diagnosis, highlights the importance of having a high index of suspicion as well as a careful diagnostic workup, including endoscopy with focused biopsies. The remission attained in the long-term treatment with a multidisciplinary approach, the combination of topical corticosteroids and dietary elimination, is an example of efficient treatment methods. The current case serves as a reminder about the importance of early diagnosis and personalized, long-term treatment to prevent further remodeling of the esophagus, symptom improvement, and a positive influence on the quality of life of patients with EoE.

Compliance with ethical standards

Acknowledgments

Special thanks to Armando Piedra, Javier A. Alemán, and David Jochheim for their assistance in reviewing the final manuscript. In addition, we appreciate the assistance of Grammarly's language editor, which helped improve the manuscript by identifying and correcting errors in grammar, spelling, punctuation, and style.

Disclosure of conflict of interest

All authors make the following declarations:

- **Payment/services information:** All authors have declared that they received no financial support from any organization for the submitted work.
- **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

Consent for publication of this case was obtained from the patient.

References

[1] Mansoor E, Cooper G. Epidemiology of Eosinophilic Esophagitis in the United States: A Population-Based Study: 1621. Official journal of the American College of Gastroenterology| ACG. 2015 Oct 1;110:S693-4.

[2] Muir A, Falk GW. Eosinophilic esophagitis: a review. Jama. 2021 Oct 5;326(13):1310-8.

[3] Mona R, Hruz P. Epidemiology of Eosinophilic Esophagitis: Really a Novel and Evolving Disease? Inflammatory Intestinal Diseases. 2025 Jan 20;10(1):34-40.

[4] Kumar S, Choi SS, Gupta SK. Eosinophilic esophagitis: current status and future directions. Pediatric Research. 2020 Sep;88(3):345-7.

[5] Dellon ES, Liacouras CA, Molina-Infante J, Furuta GT, Spergel JM, Zevit N, Spechler SJ, Attwood SE, Straumann A, Aceves SS, Alexander JA. Updated international consensus diagnostic criteria for eosinophilic esophagitis: proceedings of the AGREE conference. Gastroenterology. 2018 Oct 1;155(4):1022-33.

[6] Hahn JW, Lee K, Shin JI, Cho SH, Turner S, Shin JU, Yeniova AÖ, Koyanagi A, Jacob L, Smith L, Fond G. Global incidence and prevalence of eosinophilic esophagitis, 1976–2022: a systematic review and meta-analysis. Clinical Gastroenterology and Hepatology. 2023 Dec 1;21(13):3270-84.

[7] Gawron AJ, Hirano I. Eosinophilic esophagitis—Emerging epidemic or misdiagnosed malady?. Clinical Gastroenterology and Hepatology. 2014 Apr 1;12(4):597-8.

[8] Litosh VA, Rochman M, Rymer JK, Porollo A, Kottyan LC, Rothenberg ME. Calpain-14 and its association with eosinophilic esophagitis. Journal of Allergy and Clinical Immunology. 2017 Jun 1;139(6):1762-71.

[9] Jennifer Stratton Do, MD; Sadhna Dhingra, MBBS, MD, FCAP, et.al, Medscape. Eosinophilic Esophagitis (EoE) Pathology. (2025). Available at: https://emedicine.medscape.com/article/1610470-overview

[10] Peter A L Bonis, MD, Sandeep K Gupta, MD. UpToDate. Clinical manifestations and diagnosis of eosinophilic esophagitis. (2025). Available at: https://www.uptodate.com/contents/clinical-manifestations-and-diagnosis-of-eosinophilic-esophagitis-eoe

[11] Hernandez PV, Amer S, Lam-Himlin DM, DiSantis DJ, Menias CO, Horsley-Silva JL. Eosinophilic esophagitis: imaging features with endoscopic and pathologic correlation. Abdominal Radiology. 2020 Mar;45(3):591-600.

[12] Vendrami CL, Kelahan L, Escobar DJ, Goodhartz L, Hammond N, Nikolaidis P, Yang GY, Hirano I, Miller FH. Imaging findings of eosinophilic gastrointestinal diseases in adults. Current Problems in Diagnostic Radiology. 2023 Mar 1;52(2):139-47.

[13] Dellon ES, Gonsalves N, Hirano I, Furuta GT, Liacouras CA, Katzka DA. ACG clinical guideline: evidence-based approach to the diagnosis and Management of esophageal eosinophilia and eosinophilic esophagitis (EoE). Official journal of the American College of Gastroenterology| ACG. 2013 May 1;108(5):679-92.

[14] Hirano I, Schroeder S, Chehade M, Menard-Katcher C, Hundal NV, Liu R, Abdulai R, Maloney J, Louisias M, Radin A. Eosinophilic oesophagitis: Mechanisms. In an international forum for clinical practice and research in digestive health 2024 (Vol. 12, No. S8, p. 614).

[15] Lucendo AJ, Molina-Infante J, Arias A, Tack J, van Rhijn BD, Bredenoord AJ, et al. Guidelines on eosinophilic esophagitis: evidence-based statements and recommendations for diagnosis and Management in children and adults. *United European Gastroenterol J*. 2017;5(3):335–58.

[16] Furuta GT, Dellon ES, Hamilton RG, Spergel JM, Aceves SS, Atkins D, et al. Eosinophilic gastrointestinal diseases (EGIDs). *J Allergy Clin Immunol Pract*. 2020;8(6):1754–63.

[17] Franciosi JP, Gordon M, Vassiliki Sinopoulou, Dellon ES, Gupta SK, Reed CC, et al. Medical treatment of eosinophilic esophagitis. The Cochrane Library. 2023 Jul 20;2023(7).

[18] Rank MA, Sharaf RN, Furuta GT, Aceves SS, Greenhawt M, Spergel JM, Falck-Ytter YT, Dellon ES, Chachu KA, Day L, Lebwohl B. Technical review on the Management of eosinophilic esophagitis: a report from the AGA institute and the joint task force on allergy-immunology practice parameters. Annals of Allergy, Asthma & Immunology. 2020 May 1;124(5):424-40.

[19] Feo-Ortega S, Lucendo AJ. Evidence-based treatments for eosinophilic esophagitis: insights for the clinician. Therapeutic Advances in Gastroenterology. 2022 Jan;15:175628482110686.

[20] Le Floc'h A, Allinne J, Nagashima K, Scott G, Birchard D, Asrat S, Bai Y, Lim WK, Martin J, Huang T, Potocky TB. Dual blockade of IL-4 and IL-13 with dupilumab, an IL-4Rα antibody, is required to inhibit type 2 inflammation broadly. Allergy. 2020 May;75(5):1188-204.

[21] Underwood B, Troutman TD, Schwartz JT. Breaking down the complex pathophysiology of eosinophilic esophagitis. Annals of Allergy, Asthma & Immunology. 2023 Jan 1;130(1):28-39.

[22] O'Shea KM, Aceves SS, Dellon ES, Gupta SK, Spergel JM, Furuta GT, Rothenberg ME. Pathophysiology of eosinophilic esophagitis. Gastroenterology. 2018 Jan 1;154(2):333-45.

[23] Marc E Rothenberg, MD, PhD. UpToDate. Eosinophilic esophagitis (EoE): Genetics and immunopathogenesis. (2025). Available at: https://www.uptodate.com/contents/eosinophilic-esophagitis-eoe-genetics-and-immunopathogenesis

[24] Lewis NE, Aceves SS. Eosinophilic esophagitis: Shifting immune complexity beyond the eosinophil. Journal of Allergy and Clinical Immunology. 2024 Mar 1;153(3):669-71.

[25] D'Alessandro A, Esposito D, Pesce M, Cuomo R, De Palma GD, Sarnelli G. Eosinophilic esophagitis: from pathophysiology to treatment. World journal of gastrointestinal pathophysiology. 2015 Nov 15;6(4):150.

[26] Cheng E, Souza RF, Spechler SJ. Tissue remodeling in eosinophilic esophagitis. American Journal of Physiology-Gastrointestinal and Liver Physiology. 2012 Dec 1;303(11):G1175-87.