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Diagnostic Overlap Between Eosinophilic Esophagitis and Achalasia in a Young Adult

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Abstract

Eosinophilic esophagitis (EoE) and achalasia share overlapping clinical and histologic features, creating diagnostic ambiguity. We describe a late-adolescent male patient with progressive dysphagia and esophageal eosinophilia who was initially treated for EoE without symptomatic improvement despite histologic remission. High-resolution esophageal manometry (HREM) and functional lumen imaging probe testing (EndoFLIP) findings were consistent with type II achalasia. The patient chose to undergo a per-oral endoscopic myotomy (POEM) with symptomatic resolution, supporting achalasia as the primary diagnosis. This case highlights the importance of esophageal motility testing and patient-centered discussions on therapeutic options when dysphagia persists despite treatment of EoE.

Categories: Internal Medicine, Allergy/Immunology, Gastroenterology
Keywords: achalasia, dupilumab, endoscopy, eosinophilic esophagitis, myotomy

Introduction

Eosinophilic esophagitis (EoE) is a chronic, allergen/immune-mediated condition characterized by symptoms of esophageal dysfunction and eosinophil-predominant inflammation on esophageal biopsy [1]. It is most commonly seen in patients with concurrent atopic conditions such as asthma, allergic rhinitis, and atopic dermatitis. Dysphagia and food impaction are hallmark symptoms in adolescents and adults [1]. Achalasia is a primary esophageal motility disorder characterized by impaired lower esophageal sphincter (LES) relaxation and absent peristalsis, resulting from destruction of inhibitory neurons in the myenteric plexus [2]. Its cardinal symptoms include progressive dysphagia to solids and liquids, regurgitation, chest pain, and weight loss [2].

There is significant symptomatic overlap between EoE and achalasia, most notably dysphagia, food impaction, chest pain, and weight loss [3-7]. Distinguishing between these two disorders can be challenging given their ability to coexist with an uncertain temporal relationship; published reports and observational studies have described both EoE associated with secondary achalasia and primary achalasia associated with secondary esophageal eosinophilia, further complicating diagnostic evaluation [3-7]. In the latter scenario, esophageal stasis from impaired motility and LES dysfunction may promote food and secretion retention, leading to chronic mucosal irritation and eosinophilic infiltration that can mimic the histologic findings of primary EoE [3,6,7]. This concept is central to the diagnostic dilemma, as the eosinophilia in such cases may be a consequence rather than a cause of the underlying motility disorder.

This overlap highlights the importance of considering EoE in achalasia-like presentations, particularly because symptoms alone may be insufficient to differentiate mucosal, inflammatory, structural, and motility-related causes of dysphagia. Esophagogastroduodenoscopy (EGD) with esophageal biopsies remains critical in the evaluation of suspected EoE, as diagnosis requires symptoms of esophageal dysfunction, eosinophil-predominant inflammation on biopsy, and evaluation for alternative causes of esophageal eosinophilia [1]. High-resolution esophageal manometry (HREM) serves as the gold standard for diagnosing achalasia [2]. In equivocal cases, the functional lumen imaging probe (EndoFLIP) can provide complementary information by measuring esophagogastric junction (EGJ) distensibility and opening dynamics, helping to identify obstructive physiology when manometric or clinical findings are inconclusive [8]. Identifying the primary driver of symptoms is critical, as management strategies differ substantially. We present a case of a young adult male patient with findings of both EoE and achalasia who achieved symptomatic relief after definitive treatment for achalasia.

Case Presentation

A male patient in late adolescence with no prior medical history presented to a gastroenterology outpatient clinic with six months of progressively worsening dysphagia and the sensation of solids and liquids being “stuck” in his chest. He reported reduced dietary intake and had unintentionally lost 20 pounds over six months. He denied any history of atopic conditions, including asthma, allergic rhinitis, atopic dermatitis, or known food allergies. Family history was notable for cardiovascular risk factors, including prediabetes, heart

disease, and stroke, but was negative for EoE, atopic disease, or esophageal disorders. He was not taking any medications at the time of presentation. His diet included dairy, eggs, soy, and seafood; he subsequently attempted empirical limitation of these foods during the initial months of his evaluation without symptomatic improvement. His initial Eckardt score was 6.

On physical examination, the patient appeared well nourished, with a body mass index (BMI) of 20 kg/m². There were no signs of malnutrition, dehydration, or systemic illness. The remainder of the physical examination, including oropharyngeal, cardiopulmonary, and abdominal examinations, was unremarkable.

Given his dysphagia, weight loss, and progressive symptoms, the differential diagnosis included EoE, achalasia, gastroesophageal reflux disease with peptic stricture, infectious esophagitis, pill esophagitis, pseudoachalasia or malignancy, connective tissue disease-related dysmotility, and other obstructive esophageal motor disorders. Laboratory testing obtained during the diagnostic evaluation is summarized in Table *1*.

Feature	Evidence supporting EoE	Evidence supporting achalasia
Symptoms	Dysphagia, food impaction, weight loss (non-specific; shared by both conditions)	Progressive dysphagia to solids and liquids, Eckardt score worsening from 6 to 10 despite EoE therapy
Atopic/allergy history	No personal or family history of atopy, asthma, allergic rhinitis, or food allergies (atypical for EoE)	Not applicable
Dietary modification	Empirical limitation of dairy, eggs, soy, and seafood attempted without symptomatic improvement	Not applicable
EGD findings	Scattered white plaques (though also consistent with *Candida* from stasis)	Diffusely dilated esophagus, transient resistance at GEJ, *Candida* esophagitis (secondary to stasis)
Histology	16 eos/HPF in the upper esophagus (marginally above 15 eos/HPF threshold); 3 eos/HPF in the lower esophagus	Eosinophil count only marginally above threshold; proximal predominance atypical for classic EoE
HREM	Not applicable	IRP 31.4 mmHg, 100% failed peristalsis, 60% panesophageal pressurization → type II achalasia
EndoFLIP	Not applicable	EGJ diameter 9 mm, DI 2 mm²/mmHg, absent contractility → persistent obstruction despite histologic remission
Response to PPI (8 weeks)	No symptomatic improvement	Lack of PPI response consistent with non-inflammatory etiology of obstruction
Response to budesonide (2 months)	No symptomatic improvement	Persistent symptoms despite anti-inflammatory therapy
Response to dupilumab (3 months)	Histologic remission achieved (0 eos/HPF)	No symptomatic improvement despite histologic remission; Eckardt score worsened from 6 to 10
Response to POEM	Not applicable	Complete resolution of dysphagia; Eckardt score 0 at two-week follow-up
Overall assessment	Eosinophilia may represent secondary stasis-related infiltration rather than primary EoE	Clinical course, manometric findings, EndoFLIP data, and therapeutic response favor achalasia as primary diagnosis

TABLE 1: Diagnostic evidence supporting EoE versus achalasia.

EoE: eosinophilic esophagitis; EGD: esophagogastroduodenoscopy; GEJ: gastroesophageal junction; eos/HPF: eosinophils per high-power field; HPF: high-power field; HREM: high-resolution esophageal manometry; IRP: integrated relaxation pressure; EndoFLIP: functional lumen imaging probe; EGJ: esophagogastric junction; DI: distensibility index; PPI: proton pump inhibitor; POEM: peroral endoscopic myotomy; mmHg: millimeters of mercury

He underwent EGD, which showed a diffusely dilated esophagus with scattered white plaques and mild transient resistance when passing the upper endoscope through the gastroesophageal junction (Figure *1*). No strictures were found. Esophageal biopsies were obtained from the proximal and distal esophagus per standardized biopsy protocols and showed elevated intraepithelial eosinophils, with up to 16 eosinophils/high-power field (HPF) in the upper esophagus and 3 eosinophils/HPF in the lower esophagus,

along with evidence of *Candida* esophagitis (Figure *2*) [1]. The concurrent finding of *Candida* esophagitis was considered secondary to esophageal stasis rather than the primary cause of dysphagia, as *Candida* esophagitis can occur in the setting of obstructive esophageal conditions, including achalasia, and the patient had no immunocompromising conditions or recent antibiotic use. He was treated with a standard two-week course of fluconazole without improvement in his dysphagia symptoms.

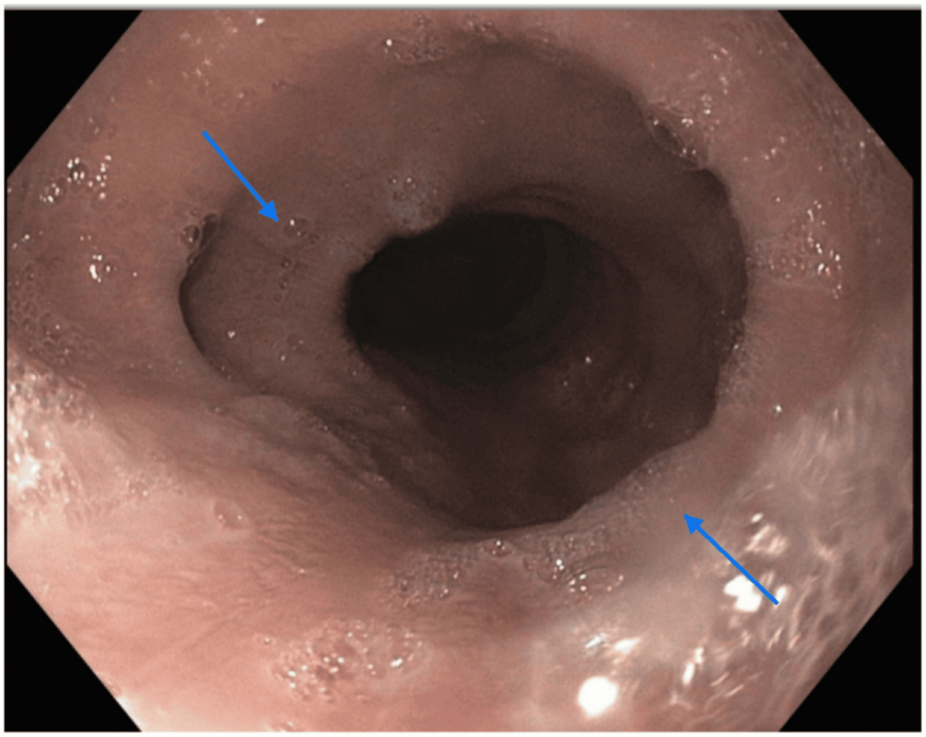


FIGURE 1: Esophagogastroduodenoscopy (EGD) demonstrating a diffusely dilated esophagus with scattered white plaques.

Arrows highlight representative areas of white plaque formation. Image obtained during diagnostic EGD.

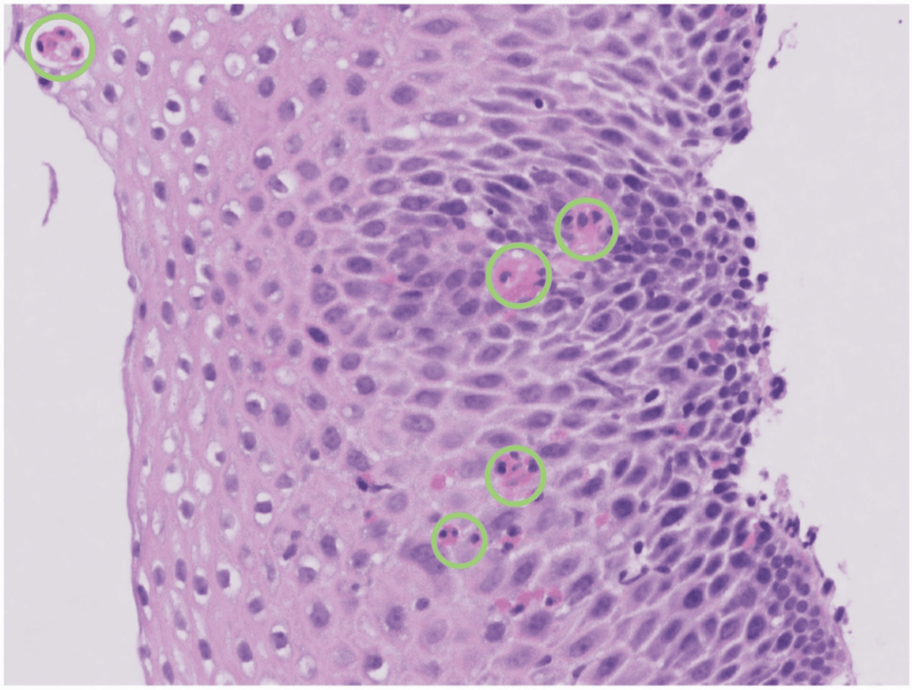


FIGURE 2: Esophageal biopsy demonstrating intraepithelial eosinophils within the squamous epithelium.

Image obtained from a hematoxylin and eosin-stained esophageal biopsy (x40 magnification). Green circles mark selected, observed eosinophils on the tissue specimen.

Given the histologic finding of esophageal eosinophilia, an eight-week course of twice-daily proton pump inhibitor (PPI) therapy was initiated concurrently. Although updated guidelines have eliminated the PPI trial as a diagnostic requirement for EoE, PPIs remain a recommended first-line therapeutic option for EoE due to their low cost, favorable safety profile, ease of administration, and anti-inflammatory properties independent of acid suppression, including reduction of eotaxin-3 expression and improvement of esophageal barrier function [1]. In this case, PPI therapy was initiated as an empirical therapeutic trial for EoE rather than as a diagnostic exclusion step. The patient did not experience symptomatic improvement with PPI therapy.

HREM, performed approximately two weeks after the initial presentation, showed impaired LES relaxation with a median integrated relaxation pressure of 31.4 mmHg, 100% failed peristalsis, and 60% panesophageal pressurization, consistent with type II achalasia (Figure 3) [2]. Given the coexistence of esophageal eosinophilia and manometric evidence of achalasia, diagnostic uncertainty was discussed with the patient. The patient was offered per-oral endoscopic myotomy (POEM) but preferred a trial of conservative medical therapy for EoE due to concern regarding the irreversibility of POEM.

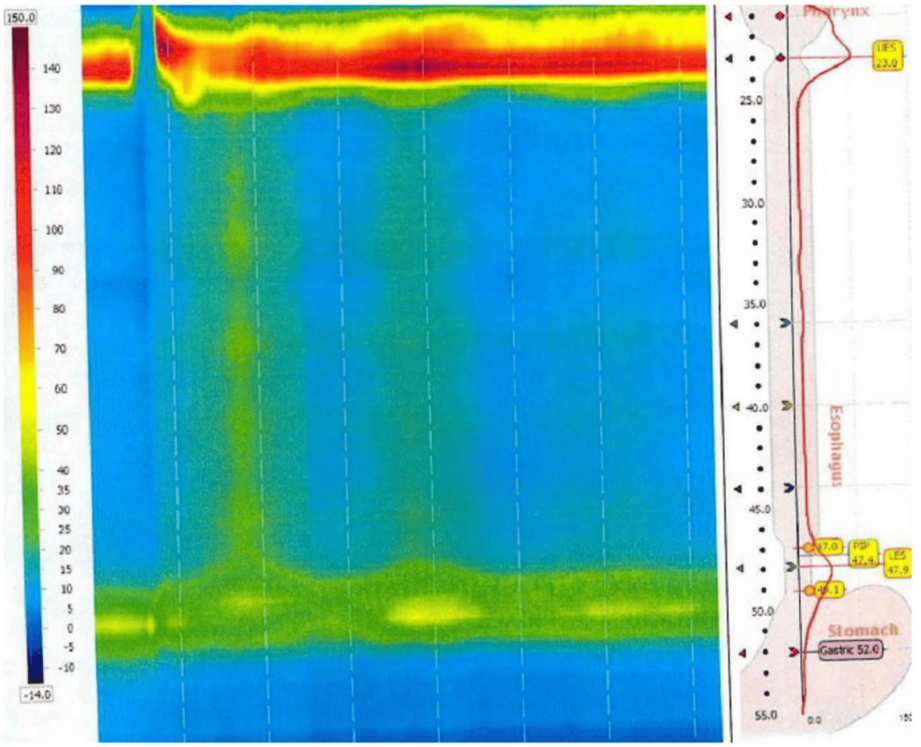


FIGURE 3: High-resolution esophageal manometry demonstrating impaired lower esophageal sphincter relaxation, failed peristalsis, and panesophageal pressurization, consistent with type II achalasia.

High-resolution esophageal manometry showed impaired lower esophageal sphincter relaxation, with a median integrated relaxation pressure of 31.4 mmHg, 100% failed peristalsis, and 60% panesophageal pressurization. Normal manometry demonstrates coordinated esophageal peristalsis with appropriate lower esophageal sphincter relaxation. In this patient, the elevated integrated relaxation pressure and absent peristalsis indicate esophagogastric junction outflow obstruction, while panesophageal pressurization supports a diagnosis of type II achalasia.

Image generated from high-resolution manometry software.

Beginning approximately one month after initial presentation, the patient trialed two months of budesonide oral suspension without symptomatic improvement, followed by three months of dupilumab beginning at approximately month three, during which his Eckardt score remained 6. Repeat EGD, performed three months after initiation of dupilumab and approximately six months after initial presentation, showed persistent moderate dilation of the esophagus, although biopsies of the upper and lower esophagus obtained per standardized protocols demonstrated complete resolution of eosinophilia, with 0 eosinophils/HPF [1]. EndoFLIP performed during the same session showed a narrow EGJ diameter of 9 mm, a distensibility index of 2 mm^2/mmHg, and a pressure of 30 mmHg at 60 cc balloon volume (Figure *4*). Absent contractility was noted [8].

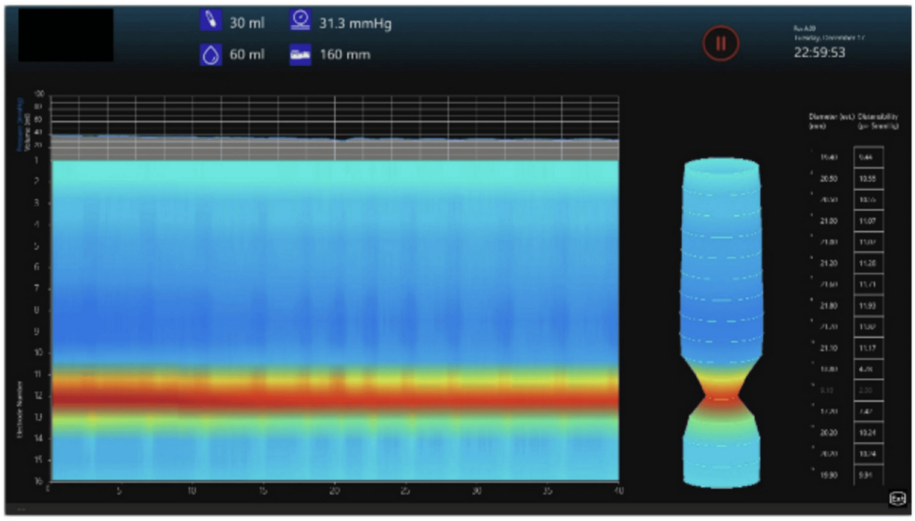


FIGURE 4: Endolumenal functional lumen imaging probe demonstrating reduced esophagogastric junction diameter and reduced distensibility.

EndoFLIP demonstrated a narrow esophagogastric junction diameter of 9 mm, a distensibility index of 2 mm²/mmHg, and a pressure of 30 mmHg at 60 cc balloon volume, with absent contractility. Normally, the esophagogastric junction should open and distend adequately during balloon distension. In this patient, the reduced esophagogastric junction opening and low distensibility index supported persistent obstructive physiology despite histologic resolution of eosinophilia.

Image generated from EndoFLIP software and de-identified for publication.

EndoFLIP: functional lumen imaging probe; mmHg, millimeters of mercury

Due to worsening symptoms with an Eckardt score of 10 and persistent evidence of ongoing obstruction despite EoE therapy with histologic remission, achalasia was thought to be the more likely primary diagnosis. The decision to proceed with POEM was based on several converging factors: failure of sequential EoE-directed therapies, including PPI, budesonide, and dupilumab, despite achieving histologic remission; worsening obstructive symptoms with a rising Eckardt score; and EndoFLIP findings demonstrating persistently reduced EGJ distensibility consistent with achalasia [8]. POEM was selected over pneumatic dilation and Heller myotomy based on patient preference and institutional expertise, consistent with current guideline recommendations for type II achalasia [2,9,10]. Following patient-centered discussion of the risks and benefits, the patient opted for POEM, which was performed approximately seven months after initial presentation. At the two-week follow-up after POEM, the patient reported complete resolution of his dysphagia symptoms with an Eckardt score of 0. Overall, this patient's clinical course from initial presentation to clinical remission lasted approximately seven and a half months (Figure *5*).

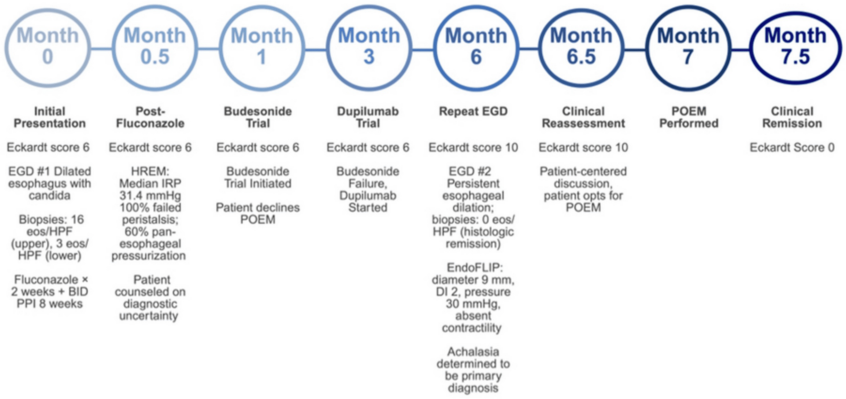


FIGURE 5: Timeline of diagnostic evaluation and therapeutic interventions over the patient's clinical course.

The figure summarizes the patient's clinical course.

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EGD: esophagogastroduodenoscopy; eos/HPF: eosinophils per high-power field; PPI: proton pump inhibitor; HREM: high-resolution esophageal manometry; IRP: integrated relaxation pressure; POEM: per-oral endoscopic myotomy; EndoFLIP: functional lumen imaging probe; DI: distensibility index

Discussion

This case highlights the diagnostic difficulty that may arise when EoE and achalasia-like physiology appear concurrently. Current diagnostic criteria for EoE require symptoms of esophageal dysfunction, eosinophil-predominant inflammation on esophageal biopsy, and evaluation for non-EoE causes of esophageal eosinophilia [1]. In this setting, the role of EGD with biopsy is particularly important, as symptoms alone may be insufficient to differentiate mucosal, inflammatory, structural, and motility-related causes of dysphagia. In the present case, the patient initially met histologic criteria for esophageal eosinophilia, but his lack of symptomatic response to EoE-directed therapy and persistent obstructive physiology despite histologic remission favored achalasia as the primary driver of symptoms.

A key question raised by this case is whether the observed eosinophilia represented primary EoE or secondary eosinophilic infiltration related to esophageal stasis from achalasia. In primary achalasia, impaired LES relaxation and absent peristalsis lead to food and secretion retention, which may cause chronic mucosal irritation and secondary eosinophilic infiltration that mimics the histologic appearance of EoE [3,6,7]. Several lines of evidence in this case favor secondary eosinophilia: the eosinophil count of 16 eosinophils/HPF was only marginally above the diagnostic threshold of 15 eosinophils/HPF; the patient lacked a personal or family history of atopic disease; empirical dietary limitation of common EoE-associated triggers, including dairy, eggs, soy, and seafood, did not improve symptoms; and histologic remission with EoE-directed therapy did not produce any symptomatic improvement. Conversely, it remains possible that EoE was a true coexisting condition or even a contributor to the development of achalasia, as proposed by Spechler et al., who hypothesized that a muscle-predominant form of EoE could cause irreversible motility disorders through eosinophil-mediated destruction of myenteric neurons [6]. A summary of the diagnostic evidence supporting each diagnosis is presented in Table *2*.

Laboratory test	Patient value	Reference range	Interpretation/relevance to differential diagnosis
WBC	5.2 K/mcL	3.8-9.8 K/mcL	Normal; no leukocytosis to suggest systemic infection or inflammation
RBC	5.39 M/mcL	4.03-5.29 M/mcL	Mildly elevated
HGB	14.6 g/dL	11.0-14.5 g/dL	Mildly elevated; no anemia despite weight loss
HCT	45.3%	33.9%-43.5%	Mildly elevated
Platelet count	319 K/mcL	175-332 K/mcL	Normal; no thrombocytosis to suggest significant systemic inflammation
Absolute neutrophils	2.02 K/mcL	1.54-7.04 K/mcL	Normal
Eosinophils	4.0%	0.0%-4.0%	Upper limit of normal
Absolute eosinophils	0.21 K/mcL	0.00-0.38 K/mcL	Normal; no peripheral eosinophilia despite esophageal tissue eosinophilia
Sodium	140 mmol/L	136-146 mmol/L	Normal; no electrolyte abnormality contributing to symptoms
Potassium	3.9 mmol/L	3.5-5.1 mmol/L	Normal
CO_2, total	26 mmol/L	22-32 mmol/L	Normal
Glucose	92 mg/dL	70-125 mg/dL	Normal
Urea nitrogen	11 mg/dL	7-21 mg/dL	Normal
Creatinine	0.7 mg/dL	0.4-0.8 mg/dL	Normal renal function
Albumin	4.3 g/dL	3.9-4.9 g/dL	Normal; preserved nutritional marker despite reduced intake and weight loss
Total protein	7.7 g/dL	6.7-8.4 g/dL	Normal
Calcium	9.4 mg/dL	9.1-10.3 mg/dL	Normal
Alkaline phosphatase	210 units/L	115-471 units/L	Normal
AST	20 units/L	15-36 units/L	Normal
ALT	9 units/L	16-31 units/L	Low; not clinically suggestive of hepatic injury
Total bilirubin	0.6 mg/dL	0.1-1.2 mg/dL	Normal
Anion gap	9 mmol/L	6-14 mmol/L	Normal
Trypanosoma cruzi Ab, IgG	0.5 IV	≤1.0 IV	Negative; argues against Chagas disease as a secondary cause of achalasia-like dysmotility

TABLE 2: Pertinent laboratory evaluation during diagnostic workup.

Laboratory evaluation was largely unrevealing, with normal white blood cell count (WBC), normal absolute eosinophil count, preserved albumin, and negative *Trypanosoma cruzi* IgG antibody testing. These findings were considered in the broader differential diagnosis of dysphagia and achalasia-like physiology, including eosinophilic, infectious, metabolic, nutritional, and secondary causes of esophageal dysmotility.

ALT: alanine aminotransferase; AST: aspartate aminotransferase; CO_2: carbon dioxide; HCT: hematocrit; HGB: hemoglobin; IgG: immunoglobulin G; RBC: red blood cell count

The concurrent finding of *Candida* esophagitis in this patient warrants discussion. *Candida* esophagitis can occur in the setting of esophageal stasis from obstructive conditions, including achalasia. In this case, the patient had no immunocompromising conditions, and his dysphagia did not improve after antifungal treatment, supporting the interpretation that *Candida* esophagitis was an incidental finding secondary to stasis rather than the primary cause of his symptoms.

EndoFLIP can provide complementary information in equivocal cases of EGJ outflow obstruction and can

help characterize EGJ distensibility [8]. In this case, EndoFLIP supported persistent obstruction at the EGJ despite resolution of eosinophilia, further strengthening the diagnostic suspicion for clinically significant achalasia. This finding was particularly useful because the patient had overlapping features of inflammatory and motility-related disease, making treatment sequencing more complex.

Clinical practice updates support the use of POEM as an effective therapeutic option for achalasia, particularly when selected based on patient preference, disease subtype, and institutional expertise [9,10]. The American College of Gastroenterology published guidelines in 2020 for achalasia; these guidelines recommend an initial EGD to rule out pseudoachalasia followed by HREM, which serves as the gold standard for diagnosis [2]. For type II achalasia, pneumatic dilation, Heller myotomy, and POEM are all notable treatment options, with the final choice of treatment based on patient preference and institutional expertise [2,9,10]. It should be noted that the follow-up period in this case was limited to two weeks after POEM; longer-term follow-up will be important to confirm sustained symptomatic relief and to monitor for potential recurrence of eosinophilia or dysphagia.

There is growing evidence suggesting a strong association between EoE and achalasia. A 2021 Italian multicenter study by Ghisa et al. found that achalasia and obstructive motor disorders occurred in 14.7% of patients with EoE, with 7.3% of patients having achalasia on HREM [4]. Notably, patients with clinical findings of both EoE and achalasia were less likely to respond to topical steroids, and 50% required invasive achalasia treatment for symptomatic relief, similar to the patient presented in this case [4]. A 2025 population-based study by Eindor-Abarbanel et al. further characterized the overlap between EoE and achalasia, demonstrating that patients with both conditions represent a distinct clinical phenotype [5]. Another 2024 population-based study by Reddy et al. demonstrated that patients with achalasia had a 32.9-fold increased risk of developing EoE compared to matched controls [7].

Dysphagia has a broad differential diagnosis, including mucosal disease, structural obstruction, infection, malignancy or pseudoachalasia, connective tissue disease-related dysmotility, neurologic disease, and primary esophageal motility disorders [1,2,11]. This case illustrates that histologic evidence alone may be insufficient to establish the primary cause of symptoms [3,6,7]. It also raises the question of whether EoE can lead to achalasia or whether some cases may instead represent achalasia-related stasis changes mimicking EoE [3,6,7]. No single test can accurately differentiate between EoE and achalasia in all cases; however, EGD with biopsies, HREM, and EndoFLIP can provide valuable complementary data to help ascertain the primary diagnosis [1,2,8]. The lack of clinical response to standard EoE therapy should prompt clinicians to evaluate for an underlying motility disorder [2,4]. Due to this diagnostic uncertainty, careful counseling and discussion of the risks and benefits of treatment are critical. This case contributes to the growing body of literature suggesting that the relationship between EoE and achalasia is more complex than previously recognized. Longer-term follow-up after POEM in patients with concurrent EoE and achalasia will be important to determine whether symptomatic relief is sustained and whether eosinophilia recurs after definitive motility-directed therapy.

Conclusions

Histologic evidence of EoE may be insufficient to determine the primary cause of dysphagia, particularly when esophageal eosinophilia may be secondary to stasis from an underlying motility disorder. Due to the permanent nature of LES-disruptive therapy for achalasia, careful investigation and patient counseling must be performed when the diagnosis of achalasia is uncertain. If diagnostic uncertainty exists between primary EoE and primary achalasia, HREM and EndoFLIP may serve as adjunctive tools for differentiation. The lack of clinical response to standard EoE therapy should prompt clinicians to evaluate for a possible underlying motility disorder. Patient-centered discussions regarding treatment options and their effects are critical in treatment planning. It should be acknowledged that the findings presented here are based on a single case and therefore cannot establish causality or determine the true pathophysiological relationship between EoE and achalasia. Future prospective studies with larger cohorts are needed to better characterize this relationship and to identify optimal diagnostic algorithms for patients presenting with overlapping features of both conditions.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Praveen Wickremasinghe, Han Zhang

Acquisition, analysis, or interpretation of data: Praveen Wickremasinghe, Han Zhang

Drafting of the manuscript: Praveen Wickremasinghe

Critical review of the manuscript for important intellectual content: Praveen Wickremasinghe, Han

Zhang

Supervision: Han Zhang

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received 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 have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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