Eosinophilic Esophagitis as a Rare Cause of Dysphagia in Jordan

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ABSTRACT

Eosinophilic esophagitis (EoE) is an uncommonly diagnosed esophageal disease characterized by dysphagia and food impaction. Differentiation between the other causes of these symptoms, particularly gastroesophageal reflux disease (GERD), could be challenging, as not only can EoE and GERD overlap, but they can also impact each other. Guidelines for best practices have only recently been developed. It affects 1 in 1000 people, mostly young men. Case Report: A 27-year-old male was referred to our institution in March 2022 with a 3-year history of persistent dysphagia for solid foods with recurrent episodes of a sensation of food impaction, requiring fluid for resolution. Upper endoscopy revealed an abnormal esophagus with macroscopic features of EoE, and histologic examination showed infiltration of more than 35 eosinophils/high-power field, which confirmed the diagnosis of EoE. Treatment was initially begun with budesonide along with Esomeprazole, which was started months before endoscopy. Symptoms improved gradually during the following 3 months, and a repeat endoscopy revealed significant improvement of the endoscopic and histologic features. The patient remained in remission on follow-up at 9 and 24 months. This case discusses how to evaluate food impaction as well as the diagnostic criteria for EoE. Since the diagnostic and treatment guidelines for EoE are relatively new, it is easy for the primary care physician to overlook it, causing a delay in specialist consultation and thus delaying treatment.

Keywords: Eosinophilic esophagitis, Dysphagia, Food impaction, Hypersensitivity.

INTRODUCTION

Eosinophilic esophagitis (EoE) is a newly recognized chronic inflammatory disease of the esophagus characterized by dysphagia and esophageal food impaction[1]. EoE was essentially unknown until around two to three decades ago, its initial recognition and comprehension occurring in the 1990s[2]; hence, our knowledge of several elements of its natural history needs to be demystified[3].

EoE is thought to be caused by a CD4+ T helper type 2 (Th2) allergic inflammatory response to dietary food allergens in the esophageal mucosa[1]. EoE is characterized by a number of endoscopic and histologic characteristics that may vary according to the disease's stage and duration[4].

EoE is found in 2–6.5% of patients under going esophagogastroduodenoscopy (EGD) for any cause and in 12–22% of patients when dysphagia is the indication[3]. The specific causes of the growing incidence and prevalence are mainly unclear and cannot be explained just by greater disease identification[3]. Although EoE may affect patients of all ages, it has a bimodal peak, with the majority of cases occurring in either the juvenile age group or the third decade of life, with distinct clinical manifestations[5].

The significance of proton pump inhibitors (PPIs) in the diagnosis and treatment of eosinophilic esophagitis (EoE) has contributed to the existent complexity but also offers intriguing light on its pathophysiology[5,6], as PPIs not only have the ability to reduce stomach acid, but they also have anti-inflammatory properties that may alter the pathobiology of EoE[3].

As the EoE guidelines are still in their infancy, there is presently no agreement about the timing of patient follow-up visits, and since there are no standards for follow-up, physicians may determine widely varied timeframes for continued treatment; this is an issue that needs to be addressed. As a consequence of prolonged inflammation, esophageal remodeling, rigidity, and luminal narrowing may occur in untreated EoE cases[7,8].

CASE PRESENTATION

A 27-year-old male presented to our institution in March 2022 with a 3-year history of persistent dysphagia for solid foods, accompanied by recurrent episodes of food impaction, which were relieved by fluid intake. The patient reported no chest pain, acid regurgitation, or other significant past medical history regarding atopic disorders (rhinitis, asthma, eczema). Despite progressive symptoms over the past year, his weight remained stable. Physical examination, hematological, and biochemical investigations were unremarkable. The patient was prescribed lansoprazole 30 mg daily for three months by his primary care physician without symptomatic relief. No prior endoscopic evaluation had been performed.

A barium swallow study identified a narrowing of the proximal esophagus with significantly decreased caliber as shown in Figure 1.

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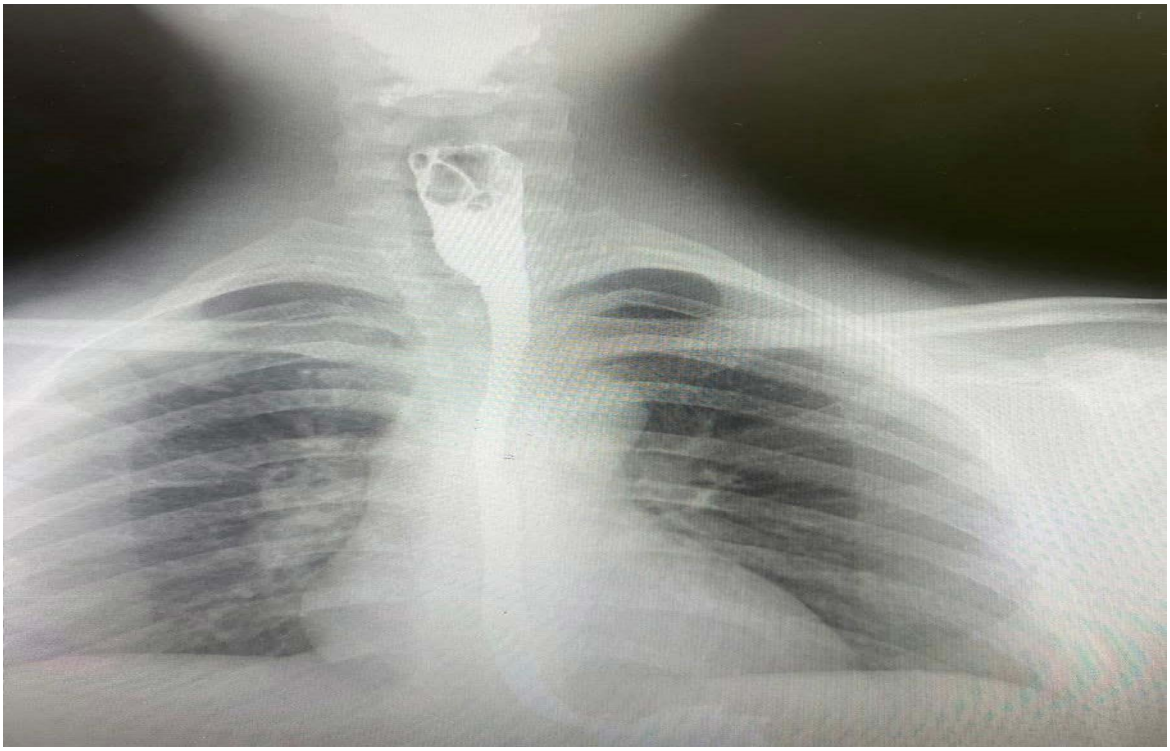

Figure 1. Barium swallow demonstrating the narrowing of proximal esophagus

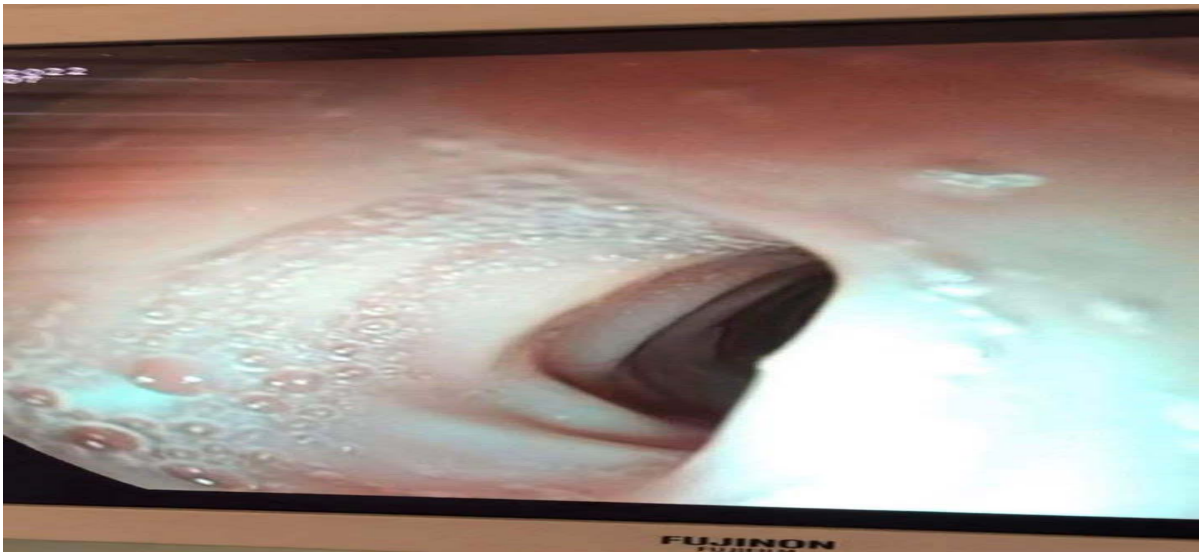

Figure 2. Upper gastrointestinal endoscopy revealed white exudates and concentric rings

An upper gastrointestinal endoscopy at our institution revealed white exudates and concentric rings, indicative of eosinophilic esophagitis (EoE) (Figure 2)**.** A proximal stricture was noted at 20 cm from the incisors that was not superable with a videogastroscope due to the narrowed esophageal lumen and esophageal mucosal retraction as shown in Figure 3**.** Histologic examination of biopsy specimens demonstrated infiltration of more than 35 eosinophils per high-power field, confirming the diagnosis of EoE (Figure 4).

The patient was treated with swallowed budesonide 1 mg twice daily along with esomeprazole 40 mg a day, started at the time of upper endoscopy. Symptoms improved gradually and no further episodes of food impaction or significant dysphagia were observed during the following 12 weeks of follow up. A follow-up endoscopy at three months showed significant improvement in the endoscopic and histological appearance. The patient remained in clinical remission at a 9-month follow-up. Swallowed corticosteroids were continued, and the patient remained in remission on follow-up at 24 months without further episodes of food impaction or dysphagia despite not undergoing any dilatation procedures.

DISCUSSION

Eosinophilic esophagitis (EoE) is a chronic condition defined clinically by esophageal malfunction symptoms and histologically by eosinophilic infiltration of the esophageal epithelium. EoE is a substantial contributor to upper gastrointestinal morbidity worldwide, an increasing health issue, and a considerable cost on healthcare systems. The rate of rise in incidence and prevalence outpaces the rate of increase in illness identification. Current incidence estimates vary

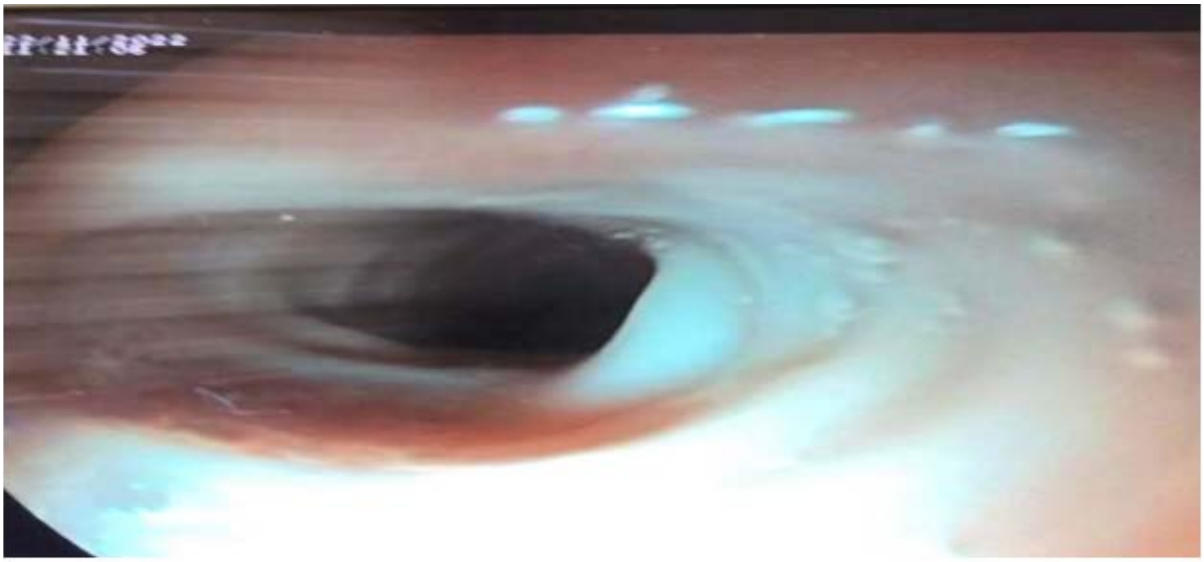

Figure 3. A proximal esophageal stricture

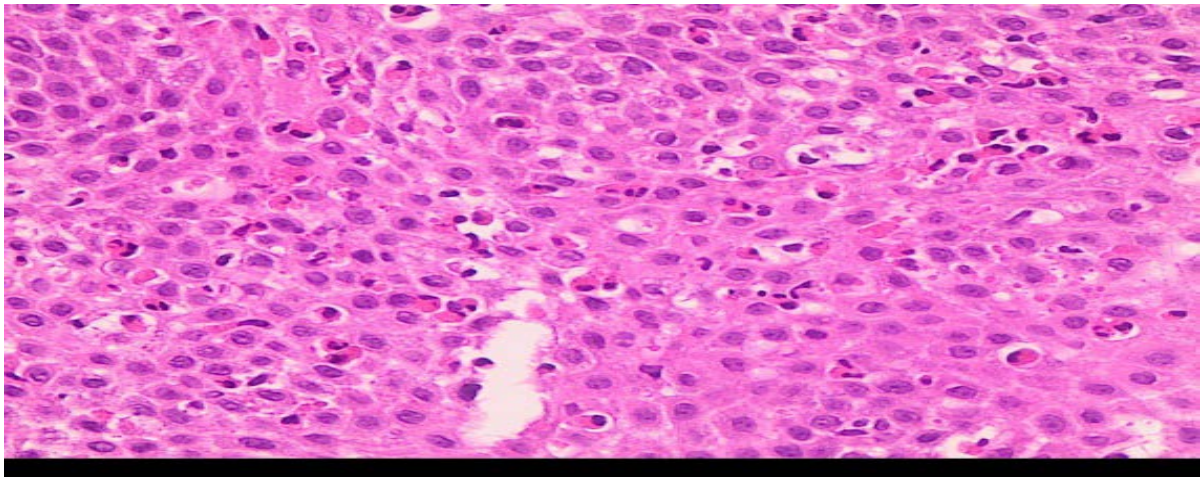

Figure 4. Histopathology showing increased eosinophils in esophageal specimens

from 5 to 10 cases per 100,000, with a prevalence estimate of 0.5 to 1 case per 1000. Patients of any age might be affected. EoE is more prevalent among Caucasians, and there is a definite male predominance (3:1). Furthermore, EoE is responsible for 5-16% of patients with dysphagia and almost half of patients with food impaction[9-11].

In recent years, several research projects have begun to look into potential risk factors for EoE. One of these proposed was geography, which includes population density and weather. When rural and urban regions were evaluated, a negative connection was found between population density and the probability of EoE. However, temperature zones, which are the primary determinants of aeroallergen dispersion, have been widely studied for the relationship between EoE and geography, with cold climate zones being related to increased probabilities of EoE compared to tropical and dry zones[12-14]. In the studied case, the patient spent his life in Jordan, which can be considered a Mediterranean climate area characterized by **hot, dry summers** and **mild, wet winters**.

Early-life exposures, genetic factors, and an atopic status are all thought to enhance the risk of eosinophilic esophagitis. When exposed to antigens, the esophageal epithelium produces alarmins, IL-33, and thymic stromal lymphopoietin (TSLP). These cytokines then induce the release of IL-13, IL-4, and IL-5 by T-helper type 2 (Th2) cells. IL-13 and IL-4 cause esophageal epithelial alterations such as basal cell hyperplasia and dilated intracellular spaces. Granulocyte infiltration is caused by chemotaxins, eotaxin-3, and IL-5. The mixed cytokine environment also promotes fibroblast activation in the lamina propria, collagen deposition, and tissue stiffness[15].

The symptoms of EoE are dissimilar when comparing pediatric patients and adults. Infants and toddlers frequently experience a variety of general features, including feeding difficulties, vomiting, and gastroesophageal reflux. As a result, diagnosis of EoE in the pediatric age group can be harder to establish. On the other hand, dysphagia to solids, episodes of food impaction, and chest pain establish stereotyped patterns in adolescents and adults. GERD-like symptoms, including heartburn and precordial discomfort, are, on the other hand, frequent regardless of age[11,16]. Our patient suffered from chronic dysphagia for solid foods, with recurring bouts of food impaction that resolved with fluid intake. He had no chest pain or acid regurgitation.

Individual's adaptability (extended meal duration, predilection for finely chopped or ground foods, regular consumption of water or beverages during meals) may cause symptoms to be misunderstood. It is not clear if such symptom variations are related to the capacity to articulate symptoms, the length of sickness, or a difference in pathogenesis[17,18].

EoE diagnosis can be achieved during upper gastrointestinal endoscopy. The disease causes a variety of non-specific anatomical alterations in the esophagus, which might vary depending on the patient's age. Adults are more likely to have fibrostenotic signs such as esophageal rings or strictures, whereas children are more likely to have inflammatory abnormalities such as white plaques/exudates, linear furrows, and edema and decreased vascularity[19].

The EoE endoscopic reference score (EREFS), which stands for the five major findings of edema, rings, exudates, furrows, and strictures, is widely used to define endoscopic findings of EoE. This method improves consistency in the reporting of results, distinguishes between non-EoE and EoE patients, and corresponds with therapy[20].

In addition to that, the biopsy findings revealing increased intraepithelial esophageal eosinophils without concurrent eosinophilic infiltration in the stomach or duodenum remain the gold standard for EoE diagnosis. Because eosinophilic infiltration of the esophagus may be unevenly distributed, samples from the proximal and distal esophagus should be collected to maximize diagnostic yield. To increase sensitivity, at least five samples should be collected at different esophageal levels[21].

In individuals with esophageal eosinophilia, a positive result to proton pump inhibitors was utilized to rule out GERD. However, recent investigations have shown that 23-61% of individuals with a diagnosis of EoE based on symptoms and histology react well to PPI treatment[22].

Difficulties in managing EoE incorporate the disease's persistent aspects and the ambiguity of when, if feasible, to discontinue acute medication due to insufficient data on the disease long-term outcomes. Treatment frequently necessitates several modalities prompted by patient complaints or evaluation of sustained treatments. The primary therapy dilemma is whether patients should be treated only with the objective of full histologic response or symptom relief, regardless of histopathologic remission. The danger of stenosis owing to continuing esophageal fibrosis and remodeling caused by continuous inflammation weighs in favor of the former aim[22,23].

Previous diagnostic criteria recommended a 6- to 8-week PPI trial to rule out PPI-responsive esophageal eosinophilia (PPI-REE). Strong evidence implies that EoE and PPI-REE are most likely two different diseases. As a result of the amended criteria, a PPI study has been omitted from the diagnostic algorithm. Despite this, PPI's remain a significant therapeutic strategy, with up to 50% of EoE patients improving with PPI treatment[24,25].

Following PPI's, therapeutic options include swallowed corticosteroids, elimination diets, and esophageal dilatation. Patients now take corticosteroids via inhalers or viscous liquid corticosteroids meant for nebulization. Several novel viscous corticosteroid suspensions are being researched. Corticosteroids cause histologic remission in 50-90% of patients; nevertheless, they must be maintained in virtually all patients permanently to preserve disease remission[24,26].

Furthermore, diet elimination strategies might be allergy test-directed, empiric, or entirely remove dietary proteins (i.e., elemental formula). Recent research indicates that skin and atopy patch tests offer no advantages over empiric elimination procedures[27].

In contrast, biologic treatments targeting IgE and eosinophils (anti-IL-51, IL-13 drugs) have mainly failed in clinical trials. Early phase II study findings for dupilumab (anti-IL-4/IL-13 drug) indicate promise, with improvements in both symptoms and histology endpoints[28].

In Jordan, there is still a notable scarcity of data, as no adult studies have examined the true burden of EoE properly. One study by Altamimi et al., examined 700 upper gastrointestinal endoscopies of pediatric patients, revealed that 3% of them showed changes favoring EoE, with the vast majority being male[29]. In the adult population, the little evidence available points toward low physician awareness of this disorder, especially in primary care settings, leading to late specialist referral and causing EoE to be diagnosed very late, elevating the risk of fibrostenotic complications. Improving access to gastroenterology clinics and endoscopy services and further training programs for young physicians should be implemented along with national protocols for dealing with such presentations.

CONCLUSION

EoE is frequently seen in emergency rooms, general care settings, and gastroenterology and allergy practices. Its incidence and prevalence are rising quickly. Treatment recommendations include esophageal dilatation, topical steroids, empiric diet elimination, and proton pump inhibitors. Clinical, endoscopic, and histologic evaluations are advised to monitor treatment response and track patients over time with maintenance therapy. Assessing and managing the inflammatory and fibrostenotic components of the disease should be taken into account while evaluating and monitoring patients with EoE.

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