

Eosinophilic esophagitis and food impaction: an instructive case

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Although the key features of eosinophilic esophagitis have been increasingly described over recent years, this entity is still often not considered and consequently diagnosis is often either not made or delayed. Typical endoscopic findings may be present. The diagnosis of eosinophilic esophagitis, however, relies on the histological assessment of mucosal biopsies. This case report highlights a common pattern of presentation of eosinophilic esophagitis and demonstrates the importance of considering this diagnosis.

Key words: Children, eosinophilic esophagitis, stricture, food impaction

Eozinofilik özofajit ve gıda takılması: eğitici bir vaka

Son yıllarda eozinofilik özofajitin ana bulguları giderek artan şekilde tanımlansa da, bu durum hala genellikle akla gelmemekte ve sonuçta tanı konamamakta ve gecikmektedir. Tipik endoskopik bulgular mevcut olabilir. Yine de eozinofilik özofajitin tanısı mukozal biyopsilerin histolojik değerlendirmesine bağlıdır. Bu vaka raporu eozinofilik özofajitin prezentasyonunun sık görülen bir patternini ortaya koymakta ve tanıyı düşünmenin önemini göstermektedir.

Anahtar kelimeler: Çocuklar, eozinofilik özofajit, darlık, gıda takılması

INTRODUCTION

Eosinophilic esophagitis (EE), a disorder seen in children and adults, is characterized by eosinophilic inflammation of the esophagus. EE was first reported in the 1970's (1), but until recently was rarely diagnosed or reported. Eosinophils in the esophagus had previously been considered an indication of acid reflux (2). The first case series of EE was published in 1995 and suggested that eosinophilic inflammation of the esophagus was a disease entity separate from gastroesophageal reflux disease (3).

Much of the published literature regarding EE involves children. The incidence of EE appears to be increasing in the last decade, perhaps due to in-

creasing awareness of the disorder. An Australian study reported an increase in cases from 1995 to 2004 of 0.5 to 0.89 per 10,000 children (4). A further pediatric population-based study reported an increase in cases from 0.9 to 1.28 per 10,000 children from 2000 to 2003 (5).

The diagnosis of EE is based upon the key histologic findings of eosinophils in the esophageal epithelium. A recent consensus statement defined that ≥ 15 eosinophils per high power field (HPF) was diagnostic of EE (6). Consequently, recognition of the key clinical and endoscopic features of EE is important to ensure that this diagnosis is considered and that esophageal biopsies are obtai-

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ned at the time of endoscopy. This case report highlights an adolescent diagnosed with EE after presenting with dysphagia and repeated food bolus impaction.

CASE REPORT

A 14-year-old boy was referred for subspecialty review at a tertiary referral pediatric hospital following several episodes of food impaction and a prolonged history of dysphagia. A long-standing history of swallowing difficulties since early childhood was obtained, with frequent episodes of food impaction. During these episodes, he would typically jump up and down during meals to pass food down.

The first of two episodes of significant food impaction requiring review at his local hospital occurred at the age of 6 years, when a lump of meat became lodged in his esophagus. He was unable to swallow his own saliva, leading to drooling. After review and observation for several hours, he vomited the food bolus. A contrast enhanced radiographic study undertaken shortly thereafter was reportedly normal and the patient was subsequently discharged from medical care.

At the age of 13 years, 5 months, he had a second significant impaction, again resulting in drooling. As seen previously, he eventually vomited the food bolus after several hours of observation in an Emergency Department. A second radiographic study of the upper gut was also reported as normal. Following this episode, he was referred to an adult gastroenterologist, who undertook upper gastrointestinal endoscopy. At endoscopy, an esophageal stricture was noted at 35 cm, which did not permit passage of an adult endoscope. The boy was then referred to the children's hospital for review.

Review of his other medical history was unremarkable, other than recurrent vomiting in early infancy. He was exclusively breastfed for six months and continued to vomit regularly after solids were introduced. However, there was no history of problems with the introduction of different textured foods. He had been treated for several months with omeprazole and cisapride for ongoing vomiting symptoms from the age of 19 months, with consequent improvement.

Other relevant medical history included asthma, first diagnosed at 6 years of age and requiring inhaled steroids and bronchodilators for many ye-

ars. In addition, there was a documented anaphylactic reaction to fish, with lip swelling and airway obstruction. Skin prick testing showed positive reactions to all fish tested, as well as to shellfish, cat hair and house dust mite. A family history of atopy in his mother and brother was also noted.

On repeat upper gastrointestinal endoscopy, the esophagus appeared thickened with multiple white plaques throughout and longitudinal ridging more distally (Figure 1). There was a narrowing of the esophagus at 35 cm from the lips. Histology of esophageal biopsies showed marked basal cell hyperplasia, edema of the squamous epithelium and upward prolongation of connective tissue papillae. Numerous intraepithelial eosinophils were also noted, with more than 100 eosinophils per HPF, indicative of EE (Figure 2). Due to the esophageal stricture and ongoing dysphagia, he was treated with five days of oral prednisolone prior to commencing swallowed fluticasone and omeprazole, with resolution of symptoms within two weeks.

DISCUSSION

The clinical manifestations of EE vary with age. Dysphagia and food impaction, the presenting symptoms in our patient, are the most common presentations in adults and children over the age of 12 years (7). Feeding problems and failure to thrive are the most common presentations in children under 2 years of age, whilst school age chil-



Figure 1. White plaques and furrowing present in the upper esophagus. When viewed endoscopically, multiple white plaques were evident throughout the esophagus, along with the appearance of vertical furrowing.

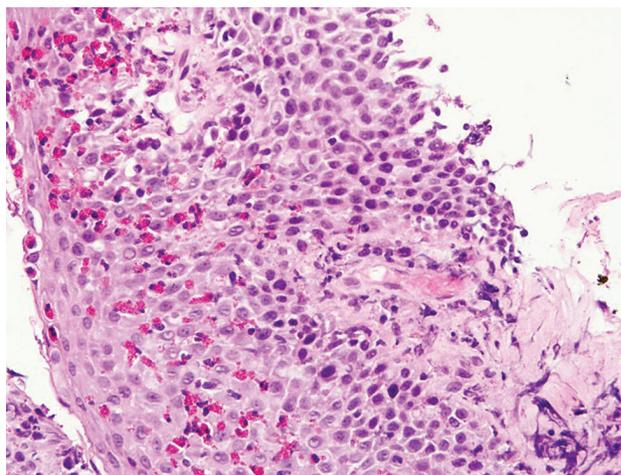


Figure 2. Photomicrograph of esophageal biopsy. Histological examination of mucosal esophageal biopsy demonstrated numerous intraepithelial eosinophils, consistent with eosinophilic esophagitis.

dren more commonly present with vomiting, abdominal pain and reflux symptoms (8). EE is linked with a higher incidence of atopy and peripheral eosinophilia (9,10), as seen in the current patient.

Diagnosis of EE is based on typical endoscopic and histological findings. The classic endoscopic finding is of a ringed appearance described as “trachealization”. Longitudinal ridging, strictures, white plaques, erythema, erosions, and a loss of vascularity may also be seen (11,12). Endoscopic features are not always present, however, indicating the importance of mucosal biopsies when EE is likely (12). The pathophysiology of EE remains unclear. Literature supports food allergy as a causative trigger for some patients (10). More than one food allergen may be identified: common allergens include milk, eggs, nuts, beef, and shellfish (13). Alternative evidence suggests that aeroallergens such as pollen or house dust mite may cause esophageal inflammation leading to EE (11). It is likely that different allergens in individuals will lead to the inflammatory response seen in EE. Skin prick and patch testing can be undertaken to identify possible immunoglobulin (Ig)E and non-IgE allergens prior to consideration of therapeutic options.

Treatment options for EE include medications and dietary therapy. Swallowed corticosteroids are re-

ported to be effective and improve symptoms in children and adults (7,8). Our patient was treated with swallowed fluticasone administered from a metered dose inhaler. More recently, leukotriene receptor antagonists have also been shown to be effective in the resolution of symptoms (8).

Dietary manipulation is another beneficial treatment modality for EE. A recommendation of the North American Consensus Group is that elemental formulae or specific antigen removal should be considered an effective treatment option, but lifestyle and family limitations have to be considered (6).

Eosinophilic esophagitis (EE) is a chronic disease, and long-term complications likely relate to the ongoing inflammation. Reported long-term sequelae include esophageal narrowing and strictures, as seen in our patient, and consequent dysphagia (8,12). Narrowing and esophageal rings can be reversible (13), but it is thought that persistent inflammation may lead to more permanent fibrosis. Studies have demonstrated that pediatric patients with EE undergo esophageal remodelling similar to that seen in chronic asthma, contributing to complications such as strictures (14). The reversibility of esophageal remodelling and subepithelial fibrosis with standard treatments for EE is still unknown (8). It is possible that an earlier diagnosis in the current patient might have resulted in earlier treatment, preventing stricture formation. Treatment of esophageal stricture in EE may include esophageal dilatation; however, this carries a risk of perforation (6,8). Esophageal metaplasia has not been reported as a long-term complication to date (11).

In conclusion, EE is increasingly prevalent. The combination of dysphagia, recurrent food impaction and atopy strongly suggest EE. Typical endoscopic findings in this setting should prompt mucosal biopsies, which are required to diagnose EE. This case illustrates the importance of the appropriate assessment of individuals with food bolus impaction and emphasizes that consideration be given to diagnostic endoscopy. It is likely that early identification and treatment of EE will decrease the long-term sequelae of this chronic disease.

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