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Eosinophilic esophagitis: Case report

Eozinofilik özofajit: Olgu sunumu

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Eosinophilic esophagitis is an inflammatory condition of the esophagus characterized by eosinophilic infiltration. It is a condition mainly affecting children; the adult form has only recently gained recognition as a distinct entity. The major symptom among adults with eosinophilic esophagitis is dysphagia. It is often misdiagnosed as gastroesophageal reflux disease because of the similarity in symptoms. An endoscopic biopsy is required to distinguish between the conditions. The cause of eosinophilic esophagitis is poorly understood, but food allergy has been implicated. Topical steroids are the most effective and convenient method for the treatment of eosinophilic esophagitis in adults. The long-term prognosis of eosinophilic esophagitis is uncertain; however, data suggests a benign course. We herein present two eosinophilic esophagitis cases that were the first to be diagnosed in our clinic.

Key words: Eosinophil, esophagitis, dysphagia, allergic conditions

Eozinofilik özofajit; özofagusun eozinofil infiltrasyonu ile karakterize inflamatuvar bir durumudur. Başlıca çocukları etkileyen bir klinik tablodur ancak günümüzde erişkin formu farklı bir antite olarak tanımlanmıştır. Eozinofilik özofajitli erişkinlerde başlıca semptom disfajidir. Bu olgular semptomları benzer olduğu için, yanlışlıkla gastroözofagial reflü hastalığı tanısı almaktadırlar. Bu iki tabloyu ayırdetmek için endoskopik biyopsi yapılması gereklidir. Eozinofilik özofajitin sebebi tam olarak bilinmemektedir, ancak gıda allerjisi ile ilişkisi vardır. Eozinofilik özofajitli erişkinlerin tedavisinde topikal kortikosteroidler en uygun ve etkili tedavi metodudurlar. Uzun dönem prognozu bilinmemekle birlikte, mevcut bilgiler selim seyirli olduğu yönündedir. Bu yazıda kliniğimizde ilk kez tanısı konulan 2 eozinofilik özofajitli olgu sunulmuştur.

Anahtar kelimeler: Eozinofil, özofajit, disfaji, allerjik durumlar

INTRODUCTION

The esophagus is normally devoid of eosinophils, and therefore the finding of eosinophils in the esophagus indicates pathology (1, 2). There are many disorders that are accompanied by eosinophilic infiltration in the esophagus, such as eosinophilic esophagitis (EE), eosinophilic gastroenteritis, gastroesophageal reflux disease (GERD), recurrent vomiting, parasitic and fungal infections, inflammatory bowel disorders (IBD), hypereosinophilic syndrome, esophageal leiomyomatosis, myeloproliferative disorders, carcinomatosis, periarteritis, allergic vasculitis, scleroderma and drug injury (1, 3). EE is a rare disorder characterized by clinical dysphagia and food impaction (4). It is best known in the pediatric population, but its recognition in adults has increased over the past 10 years (5). It was initially included within the more general condition known as eosinophilic gastroenteritis, but is now considered an independent entity. Attwood et al. (6) called attention to EE as a distinct clinical condition in 1993. We herein present two EE cases that are the first to be diagnosed in our clinic.

CASE REPORTS

Case 1

A 42-year-old man presented to our gastroenterology division with a history of dysphagia and food impaction occurring 3-4 times per year starting from childhood. Endoscopic removal of impacted food had been required three times. There was no history of reflux, nausea, vomiting, hematemesis or any other gastrointestinal symptom. He had no constitutional features such as weight loss, fever or any other symptom suggesting a systemic disease.

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His physical examination was within normal limits. Upper endoscopy revealed the presence of concentric rings along the entire length of the esophagus (Figure 1). Biopsies from different levels of the esophagus showed a dense eosinophilic infiltrate [>24 eosinophils per high powered field (HPF)] (Figure 2). Complete blood count (CBC) and basic biochemical tests were normal and there was no eosinophilia. Allergy skin testing was negative. He was diagnosed with EE and treated with fluticasone 125 mcg inhaler, four puffs swallowed three times a day for six weeks. His dysphagia resolved and he remained in remission at the 12-month follow-up.

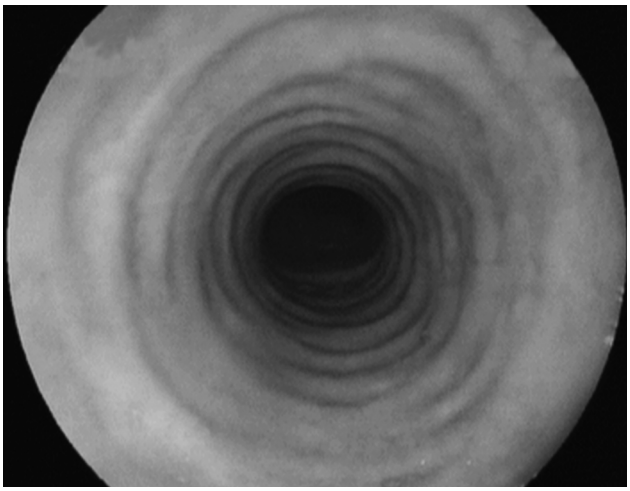

Figure 1. Concentric rings along the entire length of the esophagus

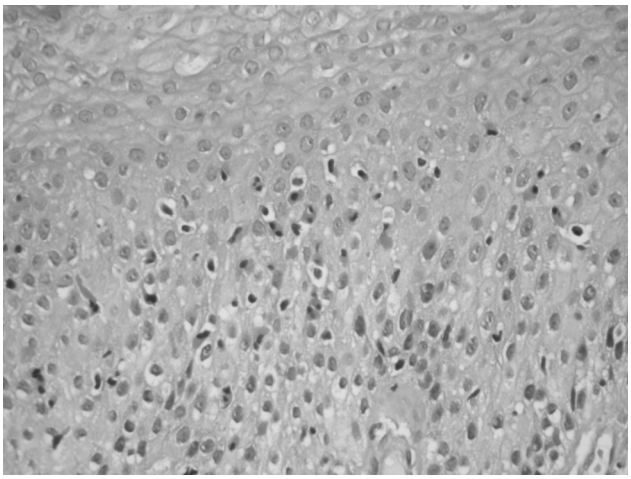

Figure 2. Dense eosinophilic infiltrates in lamina propria (intraepithelial eosinophils > 24 in any HPF)

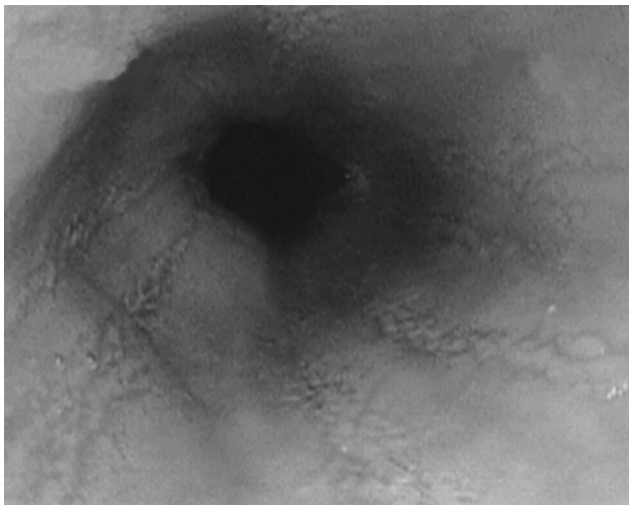

Figure 3. Diffuse edema and linear vertical furrows in esophageal mucosa

Case 2

A 35-year-old woman presented with a history of heartburn and dysphagia. Her heartburn problem had been resolved with proton pump inhibitor (PPI) treatment, but dysphagia had persisted. She was therefore referred to our clinic for endoscopy and esophageal motility study. Both her past history and physical exam were unremarkable. Esophageal motility study was performed and found normal. Upper endoscopy revealed diffuse edema and linear vertical furrows (Figure 3), and she had a 2 cm hiatal hernia. Biopsies from proximal and distal parts of esophagus showed eosinophilic infiltration (>24 eosinophils per HPF). She was diagnosed as EE. Since she was referred to us only for endoscopic and manometric inquiry, we have no information regarding her follow-up.

DISCUSSION

Eosinophilic esophagitis was first reported in 1978 (8), but Attwood et al. (6) called attention to it as a distinct clinical condition in 1993. EE has gained widespread recognition during the last 10 years. Normally there are no eosinophils in the esophagus, thus their presence indicates pathology. Since different conditions can cause eosinophilic infiltration into the esophagus, such conditions should be excluded to diagnose EE.

These two cases with EE are the first to be diagnosed in our clinic. Diagnoses were done based on endoscopic suspicion and histopathologic confirmation of eosinophilic infiltration. In both cases, other conditions that cause eosinophil-associated

esophagitis were excluded. Endoscopic suspicion is necessary for the diagnosis of EE. Endoscopic features of EE are vertical linear furrows, concentric rings resembling feline esophagus, uniform small-caliber esophagus, proximal and/or distal strictures, 1 to 2 mm whitish vesicles scattered over the mucosal surface, edema, fragility (crepe paper mucosa) and exudates. Gastroenterologists observing these findings during endoscopy should biopsy for histopathologic confirmation of the diagnosis of EE.

Presence of GERD symptoms in our second case made differentiation difficult. The presence of more than 24 eosinophils (per HPF) at the proximal part of esophagus indicates the diagnosis of EE. However, in this case, although there were no typical erosive lesions of reflux disease, non-erosive reflux disease could not be excluded because of the presence of heartburn and also hiatal hernia. Her heartburn was resolved with PPI. GERD is a prevalent disorder; therefore, in this case, the coincidence of GERD and EE can be accepted. pH meter would have been helpful in the diagnosis of GERD, but we could not perform it because she was referred to us for limited investigations.

The cause of EE is poorly understood, but food allergy has been implicated. Recent clinical and basic studies suggest an allergic etiology, but the precise allergen remains unknown and is likely unique in each patient. In fact, the majority of patients have evidence of food and aeroallergen hypersensitivity, as defined by skin prick tests, radioallergosorbent tests (RASTs), or both; however, only a minority have a history of food anaphylaxis (1, 2, 4).

There is no consensus regarding the treatment of EE. Standard recommendations include nutritional restrictions, medical management with systemic or topical steroids, and esophageal dilation when indicated. Treatment of children with EE is challenging. Optimal treatment involves identification of a responsible allergen, typically food-associated. In consultation with the allergist, single food is eliminated to see if symptoms and histopathology improve (4). In adults, food allergy is less responsible. Only a minority of adults with EE develop such severe disturbances that therapy is desired (4, 8). When obstructive symptoms and esophageal stricture are present, medical management should be strongly considered, and if it is not effective, dilatation is indicated. Adults have also received treatment with topical corticosteroids leading to remission of symptoms and normalization of histopathology. As we did in the first case, this treatment involves spraying an actuation of fluticasone from a metered-dose inhaler into the mouth and having the patient swallow. The patient should not eat and drink for 30 minutes following the actuation (4).

Eosinophilic esophagitis is now an established cause of dysphagia. Gastroenterologists should have a high suspicion for this condition if the characteristic clinical and endoscopic findings are present. Biopsies of the proximal and distal esophagus should be taken for histopathologic confirmation. Presence of eosinophilic infiltration is necessary for the diagnosis. In addition, given the differences in management, EE must be differentiated from GERD as the cause of dysphagia. In children, dietary therapy is probably the treatment of choice, but data on the efficacy of such therapy in adults is lacking. Topical steroids are the most effective and convenient method for the treatment of EE in adults, and have relatively few side effects (7). The long-term prognosis of EE is uncertain; however, data suggests a benign course (9).

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