

Pollen allergy and eosinophilic esophagitis

Polen allerjisi ve eozinofilik özofajit

To the Editor,

Eosinophils are normally detected in the gastrointestinal (GI) segments from the stomach to the colon, probably because of their beneficial functions in host defense (1). However, the esophagus normally does not contain resident eosinophils, and therefore, the finding of eosinophils in the esophagus always points to medical problems. Eosinophilic esophagitis (EE) is a chronic inflammatory disease of the esophagus characterized by eosinophilic infiltration in the mucosa. The most common symptoms of EE are vomiting, abdominal pain, weight loss, and dysphagia. The underlying etio-pathogenesis is presently unknown, though previous experimental and in vivo studies have shown an association between EE and allergic disease (2).

We report the case of a 12-year-old girl with allergic rhinoconjunctivitis. She complained of food sometimes becoming stuck in her throat for two years and of stomach pain, which increased when consuming milk with honey, for eight months. No gastroesophageal reflux was determined on scintigraphic examination. However, upper GI endoscopic examination showed irregular appearance of esophageal mucosa in the lower end and normal gastric mucosa. Histologic examination revealed a normal antrum and severe eosinophilic esophageal inflammation (Figure 1). Skin prick tests performed with the most frequent foods and inhalant allergens revealed pollen allergy. Honey was removed from her diet, and her complaints reduced.

Over the past few decades, the reported prevalence of allergic disease in patients with EE has increased dramatically, and a possible relationship between EE and allergy has been considered. Kelly et al. (3) first showed the association of food allergens with EE when their 10 children with un-

remitting reflux symptoms were given an elemental diet, which resulted in symptomatic and histological improvement. Aeroallergens form another potential cause of EE. Mishra et al. (4) used a murine model to demonstrate an etiological role for inhaled allergens and eosinophils in GI inflammation. Moawad et al. (2) found that approximately 50% of patients with EE in their study had coexisting atopic diseases (allergic rhinitis, food allergies, asthma, atopic dermatitis).

One possible mechanism in the development of EE from aeroallergens results from deposition of pollen in the nares and pharynx, with subsequent swallowing of nasal secretions and deposition of pollen into the esophagus (5). These results indicate that pollen plays a role in EE, but is probably not a main causative factor. With further study, we hope to better understand the etiology and pathophysiology of this complex disease.

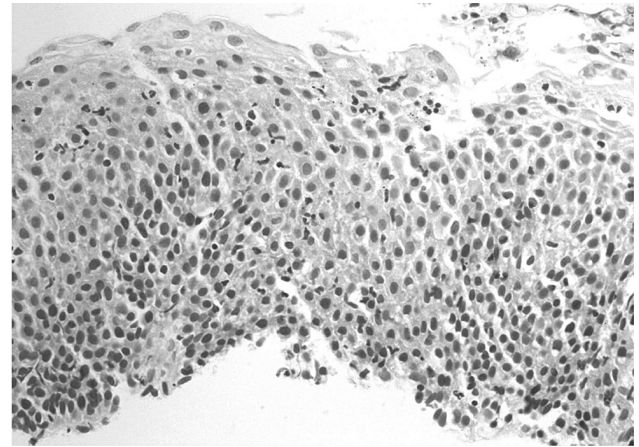


Figure 1. Esophageal endoscopic biopsy with hematoxylin-eosin staining (x400 magnification) demonstrates more than 20 eosinophils infiltrating the esophageal squamous epithelium and basement membrane thickening.

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Manuscript received: 21.02.2011 **Accepted:** 12.08.2011

Turk J Gastroenterol 2012; 23 (3): 298-312

doi: 10.4318/tjg.2012.0367

This case was presented as a poster at the 18th Congress of National Allergy and Clinical Immunology

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A case of primary gastric Burkitt-like lymphoma with chemotherapy-induced perforation

Kemoterapi ile perforasyon gelişen gastrik Burkitt benzeri lenfoma: Olgu sunumu

To the Editor,

Gastric Burkitt-like lymphoma (BLL) is a rare and very aggressive type of non-Hodgkin's lymphoma (1). According to the lymphoma classification, BLLs are considered to be borderline between classic Burkitt lymphoma (BL) and diffuse large B-cell lymphoma (2). During the course of gastrointestinal lymphomas, perforation may occur spontaneously or as a complication of treatment (3).

A 42-year-old female presented with epigastric pain and weight loss for two months. Clinical examination was unremarkable, and upper endoscopic evaluation revealed a large ulcer in the mid portion of the anterior wall of the stomach (Figure 1). Histopathologic examinations of gastric biopsy specimens showed a dense infiltrate of atypical lymphoid cells with starry sky appearance. Immunohistochemical stains were positive for CD 20 (indicating B-cell lineage), and 90% of cells were positive for Ki 67 (indicating very high proliferation rate). The diagnosis was BLL. There were no *Helicobacter pylori* organisms. Endosonographic evaluation revealed hemicircumferential thick-

ness of the gastric wall, which was infiltrated through the serosa, but no lymph node metastasis (Figure 2). There was no other involvement found in PET CT and bone marrow biopsies.

The patient underwent two courses of chemotherapy (cyclophosphamide, adriamycin, vincristine, rituximab). After the second course of therapy, she presented with acute abdominal pain and abdominal distention. Laparotomy revealed perforation of the gastric corpus, which was repaired with primary closure. Endoscopy revealed a clear improvement, and biopsies showed a complete disappearance of the lymphoma two weeks after laparotomy. The patient received four cycles of chemotherapy afterwards. She remains in excellent condition three years later with no signs of recurrence.

To the best of our knowledge, this is the first case report of gastric BLL with perforation in an adult. Gastrointestinal perforations occur rarely in association with gastric lymphomas (3). BL is known for its rapid doubling time, transmural involve-

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Manuscript received: 02.03.2011 Accepted: 15.08.2011

doi: 10.4318/tjg.2012.0373