

8 inhalants were measured. Epicutaneous SPTs were performed to 16 foods and 38 inhalant allergens. Patch testing to foods was also performed. IgE-mediated allergy was diagnosed if either serum-specific IgE or skin-prick test results were positive, whereas non-IgE-mediated allergy was diagnosed if a positive patch test result was found. A streptavidin-based immunoassay was performed to determine the presence of cross-reactive carbohydrate determinants and *Helicobacter pylori*.

**RESULTS.** Prevalence of food and inhalant allergy was 80%. The most common symptoms were dysphagia, vomiting, and abdominal pain. Food-specific IgE test results were positive to food more often than were SPT results, most commonly to milk. Serum-specific IgE detected sensitization to food in 42% of patients without a diagnosis of food allergy. Food and inhalant allergies were found with similar frequencies. Almost one-third of patients had multiple sensitivities (tree nuts, peanut, pollen, soy, and grains). Recent studies revealed allergy to plant and mammalian-derived cross-reactive carbohydrate determinants, and 3 patients were found to have a positive result (2 to bromelain and 1 patient to galactose- $\alpha$ -1,3-galactose). Patch-testing results were positive for more than one-third of the patients, most commonly to rye, without correlation to either serum-specific IgE or SPT results.

**CONCLUSIONS.** The majority of patients with eosinophilic esophagitis are atopic. The use of serum-specific IgE to foods might be useful, in particular to milk.

**REVIEWER COMMENTS.** The treatment of patients with eosinophilic esophagitis is challenging. The authors found that almost half of the patients were identified to have sensitization to a previously undiagnosed food allergen. Although the clinical significance of the serum-specific IgE might be argued, elimination diets for most patients with eosinophilic esophagitis leads to improvement. This study provides insight into another diagnostic modality, frequently used in the diagnosis of other allergic conditions, that might aid clinicians in the diagnosis and treatment of patients with eosinophilic esophagitis. However, more correlation with response to elimination of specific foods is needed.

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## Esoophageal Subepithelial Fibrosis and Hyalinization Are Features of Eosinophilic Esophagitis

Li-Kim-Moy JP, Tobias V, Day AS, Leach S, Lemberg DA. *J Pediatr Gastroenterol Nutr.* 2011;52(2):147-153

**PURPOSE OF THE STUDY.** The overlap of clinical and histologic findings between eosinophilic esophagitis (EoE) and gastroesophageal reflux disease (GERD) can lead to difficulty distinguishing these 2 conditions. These researchers sought to determine if subepithelial fibrosis could be a more specific distinguishing histologic feature of EoE.

**STUDY POPULATION.** From 358 esophageal biopsies collected from 1995-2008 in a children's hospital in Sydney, Australia, 27 children with EoE and 24 children with GERD were identified. Seventy percent of the patients were male and ranged from 7 months to 16 years of age.

**METHODS.** EoE was defined as  $\geq 15$  eosinophils per high-powered field, whereas GERD biopsies had  $< 15$  eosinophils per high-powered field. Retrospective chart reviews were performed to assess clinical symptoms, and the presence of subepithelial fibrosis was assessed with esophageal biopsy specimens.

**RESULTS.** Subepithelial fibrosis was observed in 24 (89%) children with EoE and in 9 (38%) children with GERD ( $P < .0001$ ). Fibrosis in EoE was not associated with lymphoid tissue and was less likely to occur in younger children (1.84 vs 7.02 years;  $P = .02$ ).

**CONCLUSIONS.** Subepithelial fibrosis was a common finding in children with EoE; it occurred in 89% of the children. Fibrosis was more likely to occur in older children and children with longer symptom duration.

**REVIEWER COMMENTS.** The finding of subepithelial fibrosis in children with EoE has long-term implications. If EoE pathophysiology has any similarity to asthma (which this article suggests), then early recognition and treatment to prevent fibrosis and remodeling of the esophagus are crucial. Esophageal remodeling might explain why some children with EoE have persistent symptoms despite reduction in eosinophils and why this disease is rarely short-lived.

URL: [www.pediatrics.org/cgi/doi/10.1542/peds.2011-2107DD](http://www.pediatrics.org/cgi/doi/10.1542/peds.2011-2107DD)

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## Feeding Dysfunction in Children With Eosinophilic Gastrointestinal Diseases

Mukkada VA, Haas A, Creskoff Maune N, et al. *Pediatrics.* 2010;126(3). Available at: [www.pediatrics.org/cgi/content/full/126/3/e672](http://www.pediatrics.org/cgi/content/full/126/3/e672)

**PURPOSE OF THE STUDY.** Feeding dysfunction (FD) is a symptom complex commonly associated with neurologic diseases, developmental delays, and, occasionally, gastroesophageal reflux disease. Symptoms might range from abnormal feeding behavior and immature diet preferences to sensory and motor skill deficits. The purpose of this study was to define the prevalence and feeding

characteristics of FD in children with eosinophilic gastrointestinal diseases (EGIDs).

**STUDY POPULATION.** The study included children previously evaluated in the multidisciplinary EGID program at Children's Hospital National Jewish Health (Aurora, CO) between January and December 2008.

**METHODS.** Retrospective analysis of medical records ( $N = 200$ ) assessed patients for EGID and FD (determined by feeding therapists using a feeding assessment that addressed symptoms, observation of functional skills, learned behaviors, mealtime dynamics with caregivers, and developmental skills).

**RESULTS.** Thirty-three (16.5%) patients (age range: 14–113 months) were identified as having both EGID and FD. Food sensitivity was noted in 88% of the patients, and 52% of them had clinical evidence of other allergic disease. Twenty-five of the 33 patients (76%) had eosinophilic esophagitis, defined by  $\geq 15$  eosinophils per high-powered field. Learned maladaptive feeding behaviors were the predominant form of FD and were noted in 93.9% of the children; gagging or vomiting was seen in 84.8% of them. Twenty-one percent were diagnosed with failure to thrive, and nearly 70% required individual or group feeding therapy.

**CONCLUSIONS.** Feeding difficulties are prevalent in children with EGIDs and might persist even after eosinophilic inflammation is treated.

**REVIEWER COMMENTS.** This study highlights the importance of assessing for FD in children with EGIDs and vice versa, because appropriate management of both disease states might enhance outcomes. The authors were able to examine the records of a relatively large number of patients. Potential limitations of the study were its retrospective design, lack of a universally accepted feeding-assessment protocol, and possible referral bias.

URL: [www.pediatrics.org/cgi/doi/10.1542/peds.2011-2107EE](http://www.pediatrics.org/cgi/doi/10.1542/peds.2011-2107EE)

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### **Incidental Gastric Eosinophils in Patients With Eosinophilic Esophagitis: Do They Matter?**

Ammoury RF, Rosenman MB, Roettcher D, Gupta SK. *J Pediatr Gastroenterol Nutr.* 2010;51(6):723–726

**PURPOSE OF THE STUDY.** Some patients with eosinophilic esophagitis (EoE) demonstrate an increased number of eosinophils in gastric mucosa. These researchers sought to assess clinical and therapeutic differences in children with EoE and either no gastric eosinophils (EE-N) or an increased number of gastric eosinophils (EE-A).

**STUDY POPULATION.** Children aged 1 to 18 years who had had an esophagogastroduodenoscopy (EGD) over an 8-year period (1999–2007) were assessed. The study was conducted at a children's hospital in Indianapolis, Indiana.

**METHODS.** A retrospective chart review was performed to identify children with EE-A, defined as EoE with  $\geq 10$  eosinophils per high-powered field in a gastric biopsy. Clinical characteristics and response to swallowed fluticasone between children with EE-A and children with EE-N were compared by using 2-sample  $t$  and  $\chi^2$  tests.

**RESULTS.** A total of 356 children with EoE were identified: 41 (12%) met criteria for EE-A. When compared to a randomly selected group of 50 children with EE-N, there was no difference regarding gender, age, presenting symptoms, atopy history, or esophageal histology. Both groups had similar responses to swallowed fluticasone (significant reductions in the number of esophageal eosinophils). In 11 children with EE-A treated with swallowed fluticasone, 9 (82%) had a reduction in the number of gastric eosinophils (to  $< 5$  eosinophils per high-powered field). No differences were observed between responders and nonresponders.

**CONCLUSIONS.** Twelve percent of the children with EoE had an increased number of gastric eosinophils; however, the presence of increased numbers of gastric eosinophils does not portend a worse clinical presentation or result in a reduced response to swallowed fluticasone.

**REVIEWER COMMENTS.** Just when we thought we were starting to understand EoE, gastroenterologists are now identifying children with clinical symptoms and an increased number of eosinophils in areas distal to the gastroesophageal junction. Although the authors admitted that they did not have a study group of patients with only eosinophilic gastritis, the lack of differences between EE-N and EE-A was reassuring. This study's results offer another twist in the continuing story of eosinophilic gastrointestinal disorders.

URL: [www.pediatrics.org/cgi/doi/10.1542/peds.2011-2107FF](http://www.pediatrics.org/cgi/doi/10.1542/peds.2011-2107FF)

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### **Safe Vaccination of Patients With Egg Allergy With an Adjuvanted Pandemic H1N1 Vaccine**

Gagnon R, Primeau MN, Des Roches A, et al; PHAC-CIHR Influenza Research Network. *J Allergy Clin Immunol.* 2010;126(2):317–323

**PURPOSE OF THE STUDY.** Influenza vaccines are produced from embryonated hens' eggs and contain residual, variable amounts of egg protein. This study attempted to better characterize reaction risk in a large population of egg-allergic persons.

## Feeding Dysfunction in Children With Eosinophilic Gastrointestinal Diseases

Erin M. Cannington and William K. Dolen

*Pediatrics* 2011;128;S110

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