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Original Articles

Primary Eosinophilic Esophagitis in Children: Successful Treatment with Oral Corticosteroids

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Abstract

Background: The histologic appearance of esophageal eosinophils has been correlated with esophagitis and gastroesophageal reflux disease in children. Esophageal eosinophilia that persists despite traditional antireflux therapy may not represent treatment failure, but instead may portray early eosinophilic gastroenteritis or allergic esophagitis. In this study, a series of pediatric patients with severe esophageal eosinophilia who were unresponsive to aggressive antireflux therapy were examined and their clinical and histologic response to oral corticosteroid therapy assessed.

Methods: Of 1809 patients evaluated prospectively over 2.5 years for symptoms of gastroesophageal reflux, 20 had persistent symptoms and esophageal eosinophilia, despite aggressive therapy with omeprazole and cisapride. These patients were treated with 1.5 mg/kg oral methylprednisolone per day, divided into twice-daily doses for 4 weeks. All patients underwent clinical, laboratory, and histologic evaluation before and after treatment.

Results: Histologic findings in examination of specimens obtained in pretreatment esophageal biopsies in children with primary eosinophilic esophagitis indicated significantly greater eosinophilia (34.2 ± 9.6 eosinophils/high-power field [HPF]) compared with that in children with gastroesophageal reflux disease who responded to medical therapy (2.26 ± 1.16 eosinophils/HPF; $p < 0.001$). After corticosteroid therapy, all but one patient with primary eosinophilic esophagitis had dramatic clinical improvement, supported by histologic examination (1.5 ± 0.9 eosinophils/HPF, $p < 0.0001$).

Conclusions: Pediatric patients in a series with marked esophageal eosinophilia and chronic symptoms of gastroesophageal reflux disease unresponsive to aggressive medical antireflux therapy had both clinical and histologic improvement after oral corticosteroid therapy.

This article is accompanied by an editorial. Please see: Furuta GT. In 1982, Winter et al. correlated the histologic appearance of Eosinophils in the esophagus: acid is not the only cause. *J Pediatr Gastroenterol Nutr* 1998;26:468-471.

esophageal eosinophils with abnormal acid clearance⁽¹⁾. Since then, the presence of esophageal eosinophils without evidence of other gastrointestinal disorder has been the hallmark of gastroesophageal reflux and esophagitis in children. Although Winter et al. suggested that the number of esophageal eosinophils increases in proportion to the amount of acid emitted in the distal esophagus, in most cases of reflux esophagitis, only a small number of esophageal eosinophils are present (counted per high-power microscopic field [HPF]).

We report a series of 20 children with symptoms of GER and primary reflux(GER) and esophageal eosinophilia that persisted despite eosinophilic esophagitis that were unresponsive to aggressive medical therapy were often referred to a surgeon for an antireflux antireflux medical therapy but that responded both clinically and procedure^(2,3). Recently, several reports in the adult literature have histologically to oral corticosteroids. described patients with idiopathic eosinophilic esophagitis with persistent dysphagia, esophageal spasm, or esophageal stricture, with the eosinophilic esophagitis responding to steroids⁽⁴⁻⁶⁾. Kelly et al. have reported a series of 10 children with symptoms of GER and eosinophilic esophagitis who improved with the introduction of an amino acid-based formula⁽⁷⁾.

MATERIALS AND METHODS

Patients

Between January 1, 1993, and July 1, 1995, 1809 patients were evaluated for GER disease. Each patient displayed chronic gastrointestinal symptoms (>2 months' duration) including abdominal-epigastric pain or regurgitation-vomiting, and at least one of the following: nausea, globus, water brash, chest pain, dysphagia, nighttime coughing, choking, poor appetite, weight loss, or irritability. Those patients who improved during the therapy were considered to be of the reference group of children with GER. If the patients' symptoms worsened or did not show at least a 50% improvement with Upper gastrointestinal series were performed in all patients. Whenever medication, the dose of ranitidine was increased to a maximum of 3 mg/kg twice daily, and patients were given cisapride at a maximum (positioning, feeding alteration, antacids, or H₂ blockers) or when the dose of 0.3 mg/kg four times daily. After 3 months, another EGD was performed if symptoms remained despite the therapy. Patients with disease, or weight loss, an esophagogastroduodenoscopy (EGD) was continued histologic evidence of esophagitis continued receiving performed by a board-certified pediatric gastroenterologist using an Olympus video endoscope (Olympus, Columbia, MD, U.S.A.) (N30, XP20, or GIF 100—depending on the patient's age). Patients with known gastrointestinal disorders (Crohn's disease, ulcerative colitis, celiac disease), anatomic abnormalities (malrotation, hiatal hernia, duplication) or systemic disease (cancer, chronic renal disease, scleroderma) were excluded.

Five hundred eighty-three patients underwent EGD for GER. Of this group, reflux esophagitis was diagnosed and treated in 418. Initial treatment consisted of ranitidine with metoclopramide or cisapride. Those patients who improved during the therapy were considered to be of the reference group of children with GER. If the patients' symptoms worsened or did not show at least a 50% improvement with Upper gastrointestinal series were performed in all patients. Whenever medication, the dose of ranitidine was increased to a maximum of 3 mg/kg twice daily, and patients were given cisapride at a maximum (positioning, feeding alteration, antacids, or H₂ blockers) or when the dose of 0.3 mg/kg four times daily. After 3 months, another EGD was performed if symptoms remained despite the therapy. Patients with disease, or weight loss, an esophagogastroduodenoscopy (EGD) was continued histologic evidence of esophagitis continued receiving performed by a board-certified pediatric gastroenterologist using an Olympus video endoscope (Olympus, Columbia, MD, U.S.A.) (N30, XP20, or GIF 100—depending on the patient's age). Patients with known gastrointestinal disorders (Crohn's disease, ulcerative colitis, celiac disease), anatomic abnormalities (malrotation, hiatal hernia, duplication) or systemic disease (cancer, chronic renal disease, scleroderma) were excluded.

Patients who remained symptomatic despite more than 3 months of omeprazole and cisapride therapy again underwent EGD (11.2 ± 3.8 months after the initial EGD). Those in whom continued severe esophageal eosinophilia was demonstrated, consisting of more than 15 eosinophils/HPF without evidence of antral or duodenal eosinophilia were defined as the study population. In patients with antral or duodenal eosinophilia, in addition to esophageal eosinophilia, eosinophilic gastroenteritis was diagnosed, and the patients were excluded from the study. Figure 1 outlines selection of the study population.

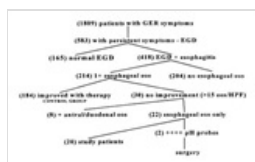


Fig. 1

Pathologic Evaluation

At least two random grasp biopsies were taken of the duodenum (third portion), stomach (antrum), and esophagus (3-5 cm above Z-line) in every patient. Additional biopsies were taken from any other site that was visually abnormal. The average number of biopsy specimens taken from each site was 2.3 ± 1.1. Each specimen was evaluated for inflammatory infiltrate (neutrophils, eosinophils) and ulceration. In addition, duodenal specimens were evaluated for villous and crypt abnormalities, antral specimens for the presence of *Helicobacter pylori*, and esophageal specimens for reflux esophagitis^(8,9). In every esophageal specimen, the number of eosinophils per HPF (magnification, ×40) was determined by averaging the number of eosinophils in sequential HPFs over the entire area (one level) of the specimen.

Study Population

All study patients underwent 24-hour pH probe testing (to determine whether acid reflux was related to esophageal eosinophilia) and quantitative IgE was performed by the Wilcoxon matched-pairs signed laboratory evaluation, including complete blood count with differential, total serum eosinophil count, quantitative immunoglobulin (Ig) E level, chemistry panel, and sedimentation rate. Patients with severe reflux revealed by pH probe (reflux index >40%) underwent surgical evaluation for fundoplication. The remaining patients began a 4-week course of oral methylprednisolone. Before therapy, each patient and family member was interviewed and completed a questionnaire regarding the type, chronicity, and frequency of symptoms (including emesis, regurgitation, heartburn, globus, dysphagia, irritability, weight loss, anorexia, water brash). Factors that precipitated symptoms—foods, position, and exercise—were also recorded. Each week, a telephone interview was conducted by the physician to record any change in clinical symptoms. All acid-suppressing medication and prokinetic agents were continued. After 4 weeks of therapy, a second upper endoscopic study, complete blood count, total serum eosinophil count, and quantitative IgE were performed.

RESULTS

An EGD was performed in 583 patients: Findings were normal in 165, Findings in a second EGD examination showed 30 patients with whereas 418 had histologic evidence of esophagitis. Of these, 214 had continued esophageal eosinophilia (>15 eosinophils/HPF) and at least 1 esophageal eosinophil/HPF without evidence of gastric or persistent symptoms. In 8 patients with antral or duodenal duodenal disease (Figure 1). After treatment with ranitidine-eosinophilia (in addition to their continued esophageal eosinophilia) omeprazole and metoclopramide-cisapride, clinical symptoms eosinophilic gastroenteritis was diagnosed and the patients excluded improved in 184 patients (control group). The clinical characteristics of the study. The remaining 22 patients underwent a 24-hour pH the control group consisted of 94% with regurgitation-vomiting, 82% probe (Synectics pH probe, Irving, TX, U.S.A.), with all acid-with abdominal-epigastric pain, 69% with nausea, 53% with chest-suppressing agents and prokinetic medications withdrawn. Two pain-heartburn, 22% with weight loss, 17% with water brush-globus, patients had severe GER according to pH probe criteria (reflux index 16% with choking, 13% with dysphagia, and 8% with bronchospasm, >40%; total time and longest episodes >90 minutes) and underwent an antireflux procedure. Twenty patients demonstrated mild or no reflux in examination by pH probe but continued to have severe symptoms and eosinophilic esophagitis, despite medical therapy with

omeprazole and cisapride (Table 1). Table 2 depicts the 24-hour pH probe characteristics of the study population. The average duration of symptoms in these patients was 3.2 ± 1.8 years. Results of laboratory testing revealed 1 patient with an elevated sedimentation rate and 1 with hypoalbuminemia.



Table 1



Table 2

The average number of esophageal eosinophils/HPF was significantly greater in those children with eosinophilic gastroenteritis and primary eosinophilic esophagitis (34.7 ± 17.3 eosinophils/HPF and 34.2 ± 9.6 eosinophils/HPF, respectively), compared with the count in children with GER that responded to antireflux therapy (2.26 ± 1.16 eosinophils/HPF; $p < 0.001$); however, no statistical difference was found in the average number of esophageal eosinophils/HPF between children with primary eosinophilic esophagitis and those with eosinophilic gastroenteritis.

Results of Corticosteroid Therapy

Twenty patients entered the study and began a 4-week course of 1.5 mg/kg methylprednisolone twice daily. Thirteen of 20 patients became completely asymptomatic and 6 others had marked improvement in their clinical symptoms after 4 weeks of corticosteroid therapy. The average time for initial appearance of clinical improvement was 8 ± 4 days. One patient continued to experience abdominal pain similar to the pain described before the initiation of steroid therapy. All 20 patients demonstrated significant clinical (asymptomatic) improvement, which was supported by histologic evidence of the average number of esophageal eosinophils/HPF. In addition, a significant difference was seen in the serum eosinophil count and quantitative IgE level (Table 3). Figure 2 shows the esophageal histologic improvement after corticosteroids.



Table 3



Fig. 2

LONG-TERM FOLLOW UP

After a second EGD, each patient's corticosteroids and reflux medications were tapered and withdrawn in 6 weeks. At the 12-month follow up, 10 patients (50%) remained asymptomatic. In 9, symptoms redeveloped and dietary restrictions were imposed, as previously described (7). Of these patients, 7 (35%) responded to dietary withdrawal, whereas 2 (10%) remained symptomatic and required a second corticosteroid challenge, 6 ± 2.5 months after initial steroid therapy. One patient (5%) remained symptomatic despite corticosteroids and dietary restriction.

DISCUSSION

These results demonstrate that a significant number of children with The precise role of the esophageal eosinophil has not been defined. characteristic symptoms of gastroesophageal reflux and predominant Esophageal eosinophilia was linked to pediatric reflux esophagitis in eosinophilic esophagitis do not have GER but instead have a unique 1982 by Winter et al. (1) They correlated the observation of esophageal disorder, which does not respond to antireflux therapy but does eosinophils with abnormal esophageal acid clearance. However, none respond both clinically and histologically to oral corticosteroids. The of the 46 patients studied had more than 3 eosinophils per HPF. Since features of this disorder, observed in the patients reported here, are that time, in several reports adult patients with severe eosinophilic significant eosinophilic infiltration only of the esophagus; minimal-to-esophagitis have been identified, suggesting a cause other than acid no acid reflux detectable by 24-hour pH monitoring; and resolution of reflux. In 1985, Lee reported on a series of 11 patients with more than the esophageal eosinophilia, evident in histologic study, within 110 esophageal eosinophils per HPF who had dysphagia, heartburn, month of initiation of steroid therapy. Although not statistically vomiting, and esophageal strictures (in 3 of 11) (10). Although he significant, the clinical features of dysphagia, eczema, and suggested that reflux occurred in the majority, reflux was not indicated

bronchospasm occurred more often in the study group, compared with results of 24-hour pH probe. One patient was given steroids and occurrences of those symptoms in the control group with reflux showed clinical improvement. esophagitis. After administration of corticosteroids, rapid improvement in clinical symptoms was observed in 19 of the 20. In 1993, Attwood et al. described 12 patients with dysphagia who had patients (noted within as few as 4 days); histologic evidence of more than 20 esophageal eosinophils/HPF (mean, 56/HPF) (11). Normal findings in pH monitoring were noted in 92%, whereas 56% had evidence of an allergic disorder. He compared results in this group with those in patients with medically responsive GER (documented by 24-hour pH probe) of whom only 45% had eosinophils in the esophagus and to a much lesser degree (mean, 3.3 eosinophils/HPF). Vitellas et al. reported 13 male patients with primary esophageal eosinophilia, dysphagia ($n = 12$), allergic manifestations ($n = 10$), peripheral eosinophilia ($n = 12$) and proximal esophageal strictures ($n = 10$) (12). None had reflux observed in barium contrast study and 85% improved with steroid therapy. In one patient, results of a second barium contrast esophagram showing resolution of the stricture.

Although all of our study patients responded rapidly to corticosteroids, our results demonstrate that eosinophilic esophagitis may take and 50% remained asymptomatic 12 months after therapy, allergic months to years to evolve. One fourth of our patients did not enteritis should always be considered in these patients. A food demonstrate severe esophageal eosinophilia on initial endoscopic elimination and challenge test should be conducted when parents or examination. In each of these patients, pronounced and persistent physicians choose not to use corticosteroids or when symptoms recur. esophageal eosinophilia was found in a second EGD after failure to Kelly et al. reported on a series of children with esophageal eosinophils obtain a clinical response to antireflux medication. Similarly, whereas who did not respond to antireflux therapy but did respond to an amino 70% of patients demonstrated peripheral eosinophilia, 35% did not acid-based formula (7). Only 1 patient underwent a 24-hour pH probe, until 5 months after initial evaluation. In 26%, patients with isolated in which the results showed no evidence of reflux. The patients were esophageal eosinophilia had evidence of antral or duodenal given solely an amino-acid based formula for a median of 17 weeks. eosinophilia in a second endoscopic study (eosinophilic Symptomatic improvement was seen within an average of 3 weeks; gastroenteritis). subsequently, regular foods were slowly reintroduced. Kelly et al. In several reports, it has been suggested that eosinophilic suggested an immunologic basis secondary to delayed hypersensitivity or to a cell-mediated hypersensitivity response.

gastroenteritis can involve the esophagus (13-15). Eosinophilic gastroenteritis is a poorly characterized, chronic disorder in which eosinophils infiltrate various layers of the alimentary tract, causing myriad gastrointestinal symptoms, including abdominal pain, vomiting, diarrhea, gastrointestinal bleeding, protein losing enteropathy, weight loss, and failure to thrive (16). In the past, the diagnosis of eosinophilic enteritis was not made unless eosinophils were observed in another portion of the gastrointestinal tract. Eosinophilic tissue infiltration in eosinophilic gastroenteritis can occur in all layers and in every part of the gastrointestinal tract (17). In children, findings of antral or duodenal eosinophilia, coupled with peripheral eosinophilia, suggest the diagnosis. Other gastrointestinal sites, including the esophagus and colon, have been reported to be affected in eosinophilic gastroenteritis but always in association with gastric or small bowel disease (14). The majority of patients manifest peripheral eosinophilia; an elevated IgE level; and other allergic symptoms, including asthma, rhinitis, and eczema.

Although the findings in this study do not answer the question of We believe that children in whom gastroesophageal reflux and cause, they indicate that children with presumed severe reflux esophagitis are diagnosed, who do not respond to aggressive antireflux esophagitis that is unresponsive to aggressive antireflux medical therapy, should undergo another round of testing for primary therapy should not be immediately referred for antireflux surgery but eosinophilic esophagitis and for the development of eosinophilic instead should be evaluated for other immunologic and allergic causes gastroenteritis. If significant esophageal eosinophilia is noted on of the esophageal eosinophilia. This study would have benefitted from histologic study of a biopsy specimen, or if peripheral eosinophilia inclusion of a placebo-controlled trial; however, the ethical develops, primary eosinophilic esophagitis should be considered. considerations of using a placebo in children with severe clinical and Steroid therapy may offer advantages as the initial therapy; our results histologic symptoms far outweighed the desirability of such a trial. demonstrated rapid clinical and histologic improvement while the Future research should be performed to determine the correlation of child consumed a diet of conventional foods. the number of esophageal eosinophils to specific disease (neonatal gastroesophageal reflux, recurrent reflux in infants and older children, allergic esophagitis and immunologic-based disease). In addition, research should be performed to determine the role of the enteric eosinophil and the cause of primary eosinophilic esophagitis.

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