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Eosinophilic Gastroenteritis associated with Food Allergy and Bronchial Asthma

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In some patients, eosinophilic gastroenteritis(EG) occurs in those with food allergy. We experienced a non-atopic asthmatic who had an EG associated with food allergy to fish and eggs, and blood eosinophilia. A skin prick test and RAST to causative food allergens showed a negative result. A fiber-optic endoscopic biopsy from the gastric mucosa showed an intense eo sinophilic infiltration. We could find symptomatic improvement and a disappearance of eosinophilic infiltration in gastric mucosa after complete avoidance from the causative food and oral cortcosteroid. It was suggested that fiber-optic endoscopic biopsy might be needed to identify coexisting EG if an allergic patient with blood eosinophilia complains of severe gastrointestinal symptoms.

Key Words : *Eosinophilic gastroenteritis, Food allergy, Bronchial asthma*

INTRODUCTION

Eosinophilic gastroenteritis(EG) is a disease characterized by eosinophilic infiltration of the gastrointestinal(GI) wall, peripheral eosinophilia, and gastrointestinal symptoms. The sites of involvement of EG include the esophagus, stomach, small intestine, colon, and rarely, extra intestinal organs. Approximately half of the cases have allergic features and some of them may be related to food allergy(Min and Metcalfe, 1991). There are few reports of EG associated with food allergy in adults. Here we report a case of EG associated with food allergy to fish and eggs in a non-atopic asthmatic patient.

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CASE SUMMARY

The patient was a 53-year-old office worker. On first admission(June, 1990), he had suffered from belching, indigestion, epigastric discomfort as well as cough and dyspnea, both of which had been more aggravated after the ingestion of fish especially cod, mackerel, hair-tail and eggs since 49 year-old. An allergy skin prick test showed all negative responses to 70 common inhalant and food allergens including cod-fish, mackerel, salmon. A methacholine challenge test showed a positive response at 1.25 mg/ml. Total blood eosinophil count was markedly elevated with 1598/mm3. An UGI study showed no abnormal findings. For his asthmatic symptoms, theophylline, β 2-agonist and oral corticosteroid were administered(The more gastrointestinal symptoms he showed, the more asthmatic symptoms were developed with higher blood eosinophils). As epigastric pain, belching and anorexia continued for several months, a fiber-

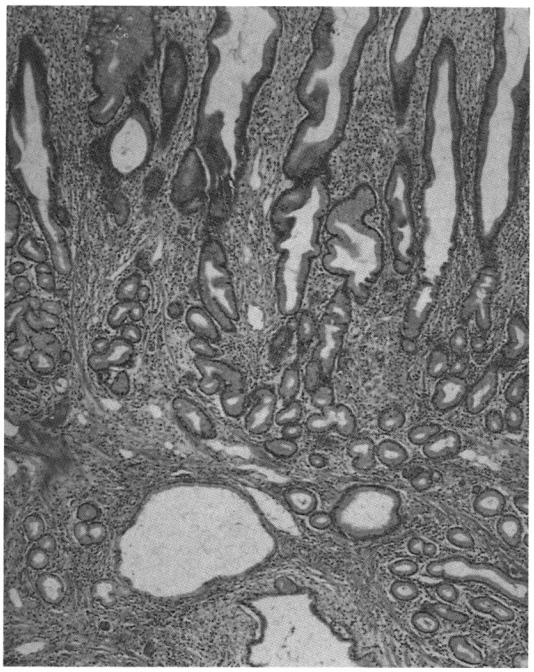

Fig. 1. A hyperplastic polyp with tortuous, elongated and cyctic dilated foveolaes which was derived from gastric mucosa

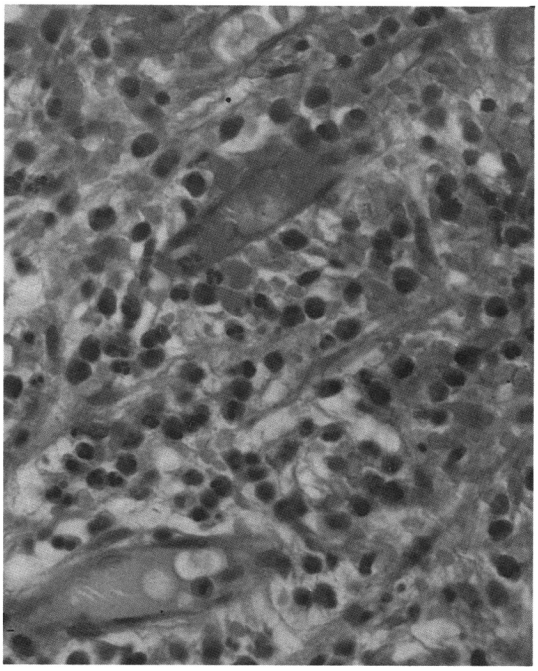

Fig. 2b. The stroma of gastirc mucosa from the angle had also a dense eosinophilic infiltrations(×400, H-E staining)

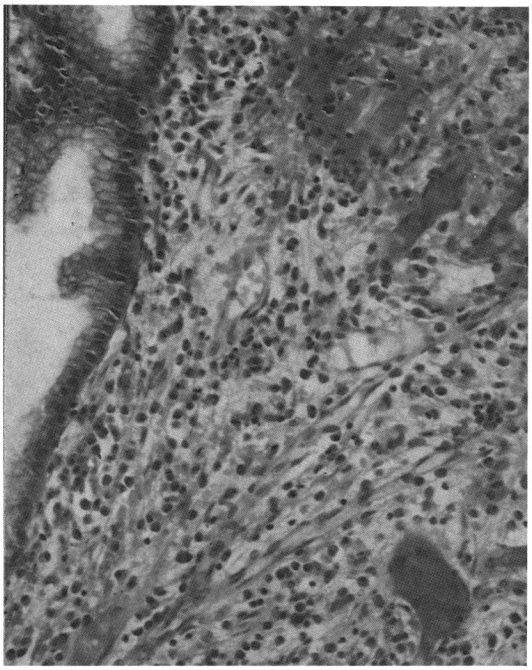

Fig. 2a. The stroma of a hyperpalstic polyp had a dense eosinophilic infiltration with increased plasma cells and some neutrophils(×100, H-E staining)

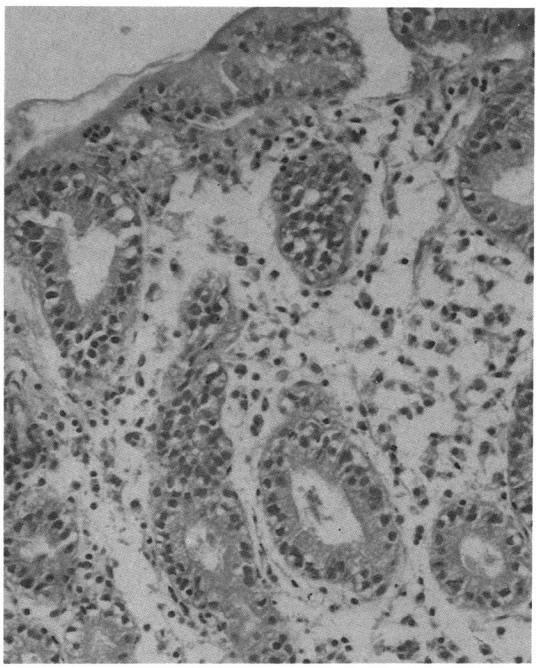

Fig. 3. A following gastric mucosal biopsy showed markedly decreased eosinophils within the mucosa.

optic endoscopy with biopsy was done. A round, 1.0 ×1.0cm sized Yamada type IV polyp was found at gastric angle and a polypectomy was done endoscopically. Pathologic findings revealed a hyperplastic polyp composed of a lot of eosinophils and the mucosal biopsy from the gastric angle also had a lot of eosinophils as shown in(Fig. 1, Fig. 2). Oral corticosteroid up to 20 mg/day was added with anti-asthmatic and G-I medications for 12 months. As his asthmatic symptoms and GI symptoms were much improved, we stopped the oral corticosteroid medication and administered only inhaled steroid for his respiratory symptoms. On a following endoscopy, diffuse scattered hyperemia was noted at the angle and antrum, which was a compatible finding with chronic superficial gastritis. A following gastric mucosa biopsy showed a normal gastric mucosa pattern with a few eosinophils(Fig. 3).

DISCUSSION

EG is an uncommon disease characterized by eosinophilic infiltration in the gastrointestinal tract. The pathogenesis of eosinophilic gastroenteritis is still unknown, although an IgE-mediated and mast cell-mediated mechanisms have been advocated as the immunologic basis of this disorder in some patients with an immunologic reaction to food antigens(Caldwell et al., 1978). Atopic diseases, such as childhood food allergies, eczema, allergic rhinitis, bronchial asthma, urticaria, and a positive family background for allergy tend to be common in patients with EG. Increased number of mucosal IgE-plasma cells and activated intraepithelial lymphocytes as well as eosinophils have been noted in the mucosa from allergic EG(Lucak et al., 1982). The present study demonstrated a case of EG associated with food allergy in a non-atopic asthmatic patient. The patient had suffered from severe gastrointestinal symptoms especially after the ingestion of various kinds of fish and eggs. Whenever he felt an aggravation of his gastrointestinal symptoms, his asthmatic symptoms were also aggravated concurrently. A gastric mucosa biopsy revealed an intense eosinophilic infiltration, which disappeared after oral corticosteroid therapy with symptomatic improvement.

Some EG occurs in patients with food allergy symptoms(Lee et al., 1993 : Park et al., 1989). In pediatric cases, milk, eggs and wheat flour are the most commonly incriminated causes of EG(Liu et al., 1968 : Oyaizu et al., 1985). IgE-mediated reactions to ingested foods occur infrequently in the general population. Other immunologic mechanisms had been suggested in food allergy(Sampson, 1991). Oral challenge of these reactions confirms only about one third of suspected food sensitivities in adults and children-(Bock et al, 1978). In the present study, although he had a definite history of food allergy, a skin prick test and RAST to suspected foods showed negative responses. Even though we could not confirm any immunological evidence for the etiologic agent of his food allergy, the gastrointestinal symptoms of this study might be caused by eosinophilic infiltration of gastric mucosa which was associated with food allergy. Endoscopic biopsy revealed infiltration of numerous plasma cells as well as eosinophils. These findings suggested a possible local immunological response limited to gastric mucosa without changes in serum in EG patients.

Activated degranulated eosinophils have been noted in a case of EG(Keshavarzion et al., 1985). The clinical features of EG depend upon which layer and location are involved(Cello, 1979 : Klein et al., 1970 : Talley et al., 1990). The deposition of major basic protein, which is the major toxic material from eosinophils was localized in the gastric mucosa of EG(Talley et al, 1992). It was suggested that IL-5, not GM-CSF and IL-3 might be the major cytokines in the pathogenesis of EG(Tokahashi et al., 1992). Further investigations are needed to clarify how eosinophils can infiltrate and survive in gastric mucosa according to different types of EG with heterogeneous etiologies.

The ideal treatment for EG is to identify and remove the causative agents if they exist. In adults, the causative agents are usually not as evident. Sequential withdrawal of various food substances may fail to provide sustained amelioration of symptoms. In most patients with EG who failed to respond to elimination diets, steroids have been successfully used sometimes for many months or even years. Cromolyn sodium and ketotifen have been used in some patients(Heatley et al., 1980 : Min and Metcalfe, 1991). In the present study, the patient was recommended to avoid causative foods and treated with oral corticosteroids, which induced a disappearance of eosinophilic infiltration in gastric mocosa with marked symptomatic improvement. The long-term prognosis of patients with EG is generally good. Adult patients may experience chronic recurrent disease(Carnichael, 1982).

In conclusion, these results suggested that, if a patient with allergic diseases and blood eosinophilia complains of severe GI symptoms, even though the gross findings of a fiber-optic gastroscopy seem not to be specific, a random gastric mucosal biopsy may be needed to identify co-existing EG. If EG is identified, chronic systemic careful steroid treatment should be considered with a complete removal of causative agents.

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