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Letter to the Editor

Eosinophilic gastroenteritis caused by eating hens' eggs: A case report

Dear Editor,

Eosinophilic gastroenteritis (EGE), first reported in 1937,[1] is characterized by eosinophilia and eosinophil infiltration of the digestive organs, as well as various digestive symptoms. Although its etiology is unknown, food allergies may be involved in its pathogenesis.[2] The present report describes a young female with EGE, possibly caused by allergy to hens' eggs, as shown by a positive food challenge. Non-IgE-mediated hypersensitivity induced by increases in the number and frequency of eggs may have led to EGE in this patient.

A 14-year-old girl was admitted to our hospital with continuous epigastralgia, vomiting and loss of appetite. Despite being positive for hens' egg-specific IgE, she continued to eat eggs without any immediate-type allergic reaction. A few months prior to admission, the frequency and number of eggs she ate gradually increased; for example, she ate a Japanese rolled omelet, equivalent to one whole egg, for lunch every day. At that time, she ate a balanced diet, consisting of common Japanese-style foods, containing about 50 g of protein per day. Although she was positive for fecal occult blood, colonoscopy at another hospital showed no positive findings. At admission to our hospital, she was suspected of having irritable bowel syndrome. Physical examination showed a height of 160.9 cm (+0.6 SD), a weight of 54.8 kg (+0.4 SD), a body mass index of 21.1 kg/m^2, a pulse rate of 78/min, and a body temperature of 36.6 °C. Her bowel sounds were weak, and the area from the epigastrium to the umbilicus was tender. Blood examination revealed a leukocyte count of 7400/μL (22.0% eosinophils; 1628/μL), a total protein concentration of 5.1 g/dL and an albumin concentration of 2.5 g/dL. Her total IgE level was 2109 IU/mL, and her specific IgE levels for egg white, egg yolk, cow's milk, wheat and soybeans were 30.8 UA/mL, 9.55 UA/mL, 6.51 UA/mL, 20.3 UA/mL and 1.74 UA/mL, respectively. An allergen-specific lymphocyte proliferation test (ALPT)[3] showed stimulation indices of 864% for egg white and 367% for soybeans. Abdominal ultrasound imaging showed edema of the entire small intestine, and upper gastrointestinal endoscopy showed partial flare of the duodenal mucosa. Biopsy specimens showed eosinophil infiltration of the duodenal mucosa (Fig. 1), but not of the esophagus. The patient was diagnosed with EGE associated with an allergy to eggs, which was complicated by protein losing enteropathy (PLE). Upon admission, eggs and all egg products were eliminated from her diet, but her symptoms persisted. She was started on oral prednisolone (40 mg/day) on hospital day 16, after the biopsy results were obtained. Her symptoms improved within 1 week. The dose of oral prednisolone was tapered 10 mg/day every 2 weeks, and she was started on pranlukast (450 mg/day) on hospital day 49 (Fig. 2). Prednisolone also improved her hypoproteinemia due to PLE. She was discharged on hospital day 56 without recurrence of her symptoms. One year later, she no longer had digestive symptoms and was in good condition. She was no longer eating eggs or egg products and was no longer being treated with prednisolone or pranlukast. An egg-food challenge yielded positive results, with symptoms of nausea and vomiting 5–6 h after eating a Japanese egg roll, equivalent to one whole egg with both egg white and egg yolk. The symptoms disappeared with oral dexamethasone 10 h after challenge.

Eosinophilic gastrointestinal disorder (EGID) is characterized by eosinophilia and eosinophil infiltration into alimentary tract mucosa. EGE is an EGID, with symptoms that include nausea, vomiting, loss of appetite and stomachache, with possible subsequent anemia and hypoproteinemia. The prevalence of EGE is very low (1/100,000), whereas that of eosinophilic esophagitis is increasing.[4] Although the pathogenesis of EGE is unknown, about 50% of patients have a history of allergic disease, such as asthma or food allergy.[5] The allergic mechanism of EGE remains unclear, but one EGID, eosinophilic esophagitis, is characterized by a non-IgE-mediated food hypersensitivity, which may be triggered by foods such as eggs, milk, wheat, soybeans, and rice, particularly in children, in the absence of specific food IgE.[6] The pathogenesis of EGE may be similar to that of eosinophilic esophagitis, with delayed-type allergic reaction playing a major role. There are no standard treatments for EGE. Corticosteroids are effective in improving symptoms, but EGE often recurs after corticosteroid discontinuation. Leukotriene antagonists have been shown effective in preventing EGE recurrence after corticosteroid therapy.[7] If a food allergy is suspected, elimination of that food may improve EGE.[8]

In our patient, elimination of egg products at admission did not improve her symptoms, since the course of EGE was progressive and associated with PLE. She therefore required subsequent administration of oral prednisolone. One year after treatment, while the patient was no longer receiving prednisolone or pranlukast, an egg challenge induced digestive symptoms 5–6 h later. Although she was positive on admission for IgE specific to eggs, she had never experienced symptoms of IgE-mediated hypersensitivity. The findings in this patient, including positivity on hens' egg challenge and an increased ALPT stimulation index, suggest that eggs and egg products may have triggered EGE via non-IgE-mediated hypersensitivity. Similarly, a case report described a patient who developed eosinophilic esophagogastroenteritis after oral immunotherapy

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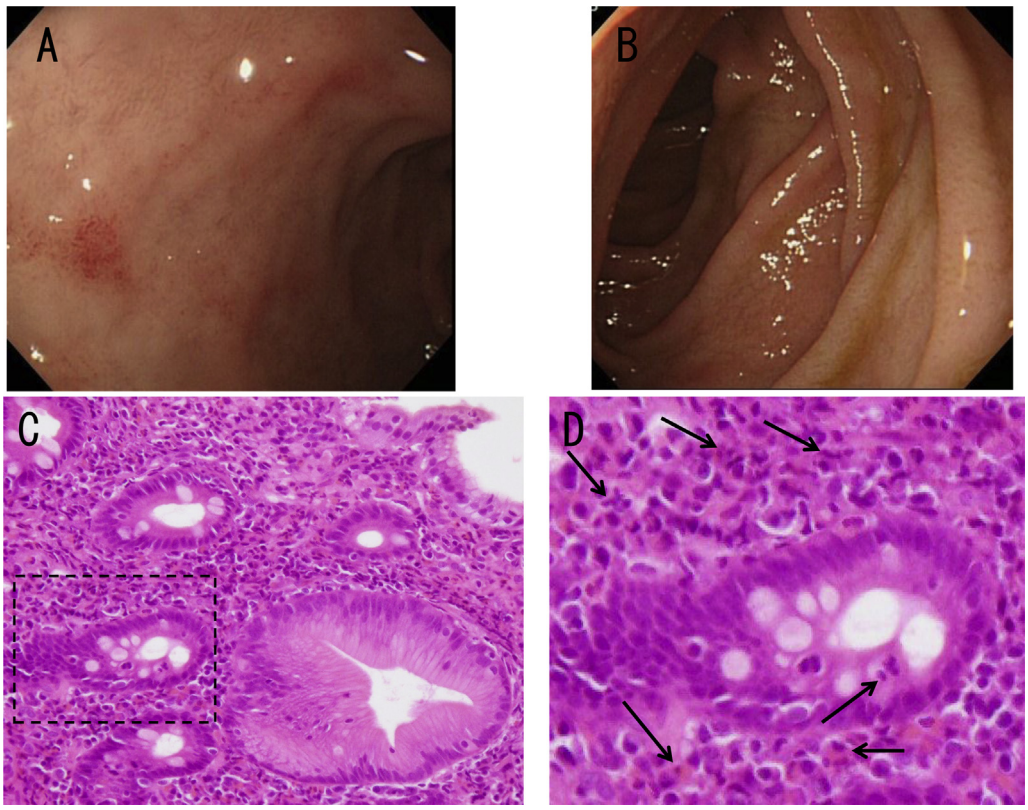

Fig. 1. Upper gastrointestinal endoscopy findings and histological findings of a biopsy specimen of the duodenal bulb of our patient. **A)** Duodenal bulb and **B)** duodenal descending part. Thickened and reddened mucosa and telangiectasia were observed from the duodenal bulb to the descending part. **C)** Stromal infiltration by eosinophils (arrows) and plasma cells. **D)** View along the dotted line in **C)** (hematoxylin and eosin staining, original magnification ×400). The eosinophil count in this patient was 136 per high powered field (HPF) (normal, <20/HPF).

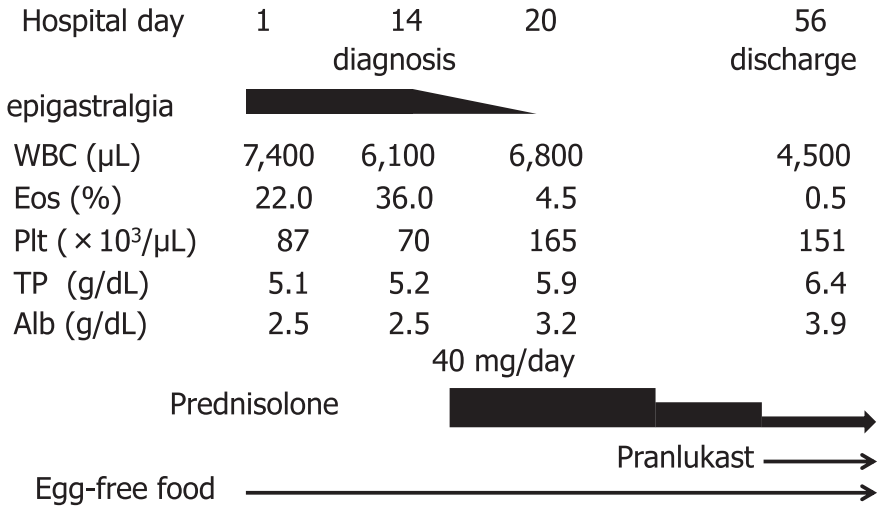

Fig. 2. The clinical course of this patient. Abbreviations: WBC, white blood cells; Eos, eosinophils; Plt, platelets; TP, total protein; Alb, albumin.

for an egg allergy and increased ingestion of eggs, suggesting that increased consumption may have triggered esophagogastroenteritis.[9] ALPT in our patient declined with improvements in her digestive symptoms and reductions in the numbers of peripheral eosinophils, as well as with the elimination of egg products. Although the cause of EGE in this patient remains unknown, our findings suggest that EGE may have been triggered by non-IgE-mediated hypersensitivity reactions induced by increases in the quantity and frequency of consumption of eggs and egg products.

In conclusion, this report described a young woman diagnosed with EGE, caused by an allergy to hens' eggs. Findings in this patient

support the pathogenic role of non-IgE-mediated hypersensitivity in EGE.

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Conflict of interest

The authors have no conflict of interest to declare.

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