Eosinophilic Gastroenteritis Complicated with *Helicobacter pylori* Infection Unresponsive to Eradication Therapy

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Abstract

An adolescent girl presented with inappetence. Upper gastrointestinal endoscopy showed rough and cracked mucosa at the gastric antrum with a scarred duodenal ulcer, and a biopsy sample demonstrated abundant eosinophils. We therefore diagnosed the patient as having eosinophilic gastroenteritis. Eradication therapy for *Helicobacter pylori* (*H. pylori*) did not improve her symptoms; however, proton pump inhibitor therapy was effective in resolving her chief complaints. There are several reports of eosinophilic gastroenteritis complicated with *H. pylori* infection in which the association between eradication therapy and the patient’s symptoms is unclear. In the present case, the patient’s symptoms did not improve with eradication therapy, and there appeared to be no relationship between the two.

Key words: eosinophilic gastroenteritis (EG), *Helicobacter pylori* (*H. pylori*), eradication therapy, proton pump inhibitor (PPI)

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Introduction

Eosinophilic gastroenteritis (EG) is an uncommon disorder first reported by Kajiser et al. (1) as eosinophilic infiltration in the gastrointestinal tract. With an incidence of 1 in 100,000 individuals, EG frequently afflicts men between 30 and 50 years of age (2). Most individuals with EG have a history of allergies, with some exhibiting eosinophilia and elevated serum IgE levels (3). In addition, there are several reports of EG complicated with *Helicobacter pylori* (*H. pylori*) infection. In such cases, symptoms have been reported to improve with both successful and failed eradication therapy. Therefore, the relationship between *H. pylori* eradication and symptom improvement remains unclear.

Case Report

The present patient was a late adolescent girl with an allergy to pollen. In November 2009, she visited her primary care doctor for inappetence, epigastric aching and vomiting. She had lost 3 kg within two weeks due to poor meal intake and had been found to have peripheral blood eosinophilia; therefore, she was referred to our institution for a closer examination in late December. The patient was slender, measuring 155 cm in height and 42 kg in weight, with a BMI of 17.5. She did not have a fever or any other abnormalities on physical or abdominal examinations. However, we noted elevated levels of peripheral blood eosinophils (860/μL; 16.5%) and IgE (454 mg/dL) on the initial laboratory examination conducted at our hospital (Table). *H. pylori* antibody findings were negative. An upper gastrointestinal endoscopic examination revealed cracked, thickened and reddened mucosa.
in the gastric antrum (Fig. 1A, B), mild chronic atrophic gastritis classified as C-II according to the Kimura-Takemoto classification system and an ulcer scar on the duodenal bulb (Fig. 1C). The esophagus and descending portion of the duodenum were apparently normal. A biopsy sample of the duodenal bulb showed significant infiltration of eosinophils, lymphocytes and plasma cells (Fig. 2A), similar to the findings obtained from a gastric antrum biopsy specimen (Fig. 2B). Whereas biopsy samples taken from the esophagus and descending portion of the duodenum did not exhibit significant infiltration of eosinophils, those obtained from the gastric body showed slightly elevated infiltration of eosinophils. *H. pylori* infection was not visually apparent. In addition, we witnessed no abnormalities on total colonoscopy, and, although there was wall thickening in the vestibular and duodenal bulbs on abdominal contrast CT (Fig. 3), no ascites was detected. A double contrast radiographic examination of the small intestine showed no abnormal findings, such as stenosis or expansion. Based on these findings, the patient was diagnosed as having EG. We first administered an H2 receptor antagonist to treat the ulcer scar, which initially improved her abdominal

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**Figure 1.** Upper gastrointestinal endoscopy findings. A) Cracked, thickened and reddened mucosa in the gastric antrum. B) Chromoenodoscopy with indigo carmine staining. C) An ulcer scar in the duodenal bulb.
symptoms and inappetence, although these symptoms again returned when the medication was stopped eight months later. Repeat upper gastrointestinal endoscopy showed an apparent improvement in the patient’s cracked mucosa in the antrum; however, an ulcer had developed on the bulb, and *H. pylori* infection was recognized on a gastric biopsy. Accordingly, we prescribed a proton pump inhibitor (PPI) and commenced *H. pylori* eradication therapy. Interestingly, her improved symptoms, including nausea and inappetence, recurred when we halted the PPI treatment for a urea breath test. The administration of the PPIs was resumed after *H. pylori* eradication was confirmed, and the patient’s inappetence again quickly improved. Upper gastrointestinal endoscopy performed one year later showed no changes in the cracked gastric antrum mucosa, although the duodenal bulb ulcer had disappeared (Fig. 4). The patient’s atrophic changes remained stable at grade C-II. Biopsies conducted to assess the degree of inflammation revealed that the number of lymphocytes and neutrophils had decreased; however, the eosinophilic infiltration had not improved in either the duodenal bulb or gastric antrum (Fig. 5). Based on the patient’s biopsy findings and residual symptoms, it appeared that the eradication therapy had not significantly contributed to the treatment of the patient’s EG. Her symptoms have since been controlled with PPI therapy without the need for steroids.

**Discussion**

We experienced a case of EG complicated with *H. pylori* infection. The patient’s pathological findings and symptoms were not remarkably improved by *H. pylori* eradication therapy, although PPI drugs were able to control her chief complaints. Therefore, we considered that *H. pylori* infection was not involved in the pathogenesis of her primary symptoms.

A slight male preponderance has been reported for EG. The majority of patients clinically present in the third to fifth decade of life, although the disease can affect any age group (4), and lesions often appear in both the small intestine and stomach (5). Half of individuals with EG have a history of asthmatic conditions, such as bronchial asthma, atopic dermatitis or allergic dermatitis (2, 6). EG symptoms often differ depending on the location of eosinophil infiltration. When the mucosa is the primary inflammatory location, the patient may complain of a stomachache, diarrhea and/or weight loss. If the muscular layer is affected, nausea, vomiting and/or stomachache may be noted, and, if the serosa is involved, eosinophilic ascites may be detected. EG is associated with increased eosinophils in the peripheral blood in 80% of cases (7). Biopsies of the stomach, small intestine and colon often reveal inflammatory cells composed primarily of eosinophils (>20-25/HPF) infiltrating mucous membranes (2). An allergic reaction to food antigens is considered to be the main mechanism of this disease. Therefore, as in the related condition of eosinophilic esophagitis, dietary restriction of cow milk protein, soy, wheat, eggs, peanuts and seafood, all of which are common food antigens, is recommended (8). Physicians may also prescribe 20-40 mg/day of prednisolone. In addition, it has been reported that anti-
allergy drugs, such as budesonide (9), cromolyn (10), ketotifen (11) and montelukast (12), may be effective for EG.

Since there are few reports of EG complicated with *H. pylori* infection, the association of both diseases and their treatments is controversial. For example, Papadopoulos et al. reported that, when they administered primary eradication therapy in a middle-aged woman with *H. pylori*-complicating EG complaining of vomiting, diarrhea and a stomachache, her symptoms and eosinophil infiltration improved (13). On the other hand, Müller et al. reported that although primary eradication therapy failed, eosinophil infiltration improved in a woman in her sixties; the authors concluded that the diseases had no relationship with each other because the eosinophil infiltration improved despite the persistence of *H. pylori* infection (14). Against this background, a unified opinion regarding the association between *H. pylori* infection and EG remains elusive. Dellon et al. reported that the incidence of eosinophilic esophagitis is increasing along with that of other allergic diseases due to improvements in hygiene, while the frequency of *H. pylori* infection...
is decreasing in an inversely proportional relationship (15). In the present case, the degree of eosinophilic infiltration and severity of the patient’s symptoms remained unchanged after eradication therapy. Therefore, we concluded that the patient’s H. pylori infection and EG were largely unrelated.

No standard treatment for EG has been established due to the insufficient level of testing and number of randomized controlled trials. Steroid therapy constitutes the current systemic treatment for EG, and there are several reports regarding the successful application of anti-allergy medicines, although there have been few accounts of the efficacy of anti-acid therapy. The introduction of steroid therapy was discussed in the present case; however, consent was not obtained from the patient or her family since she was young, and her symptoms improved with non-steroid therapy and continued anti-acid treatment. Talley et al. reported that eosinophil infiltration in the duodenum is a characteristic biopsy finding in patients with functional dyspepsia (FD) (16). Meanwhile, Walker et al. reported that, when they compared the presence of infiltrating eosinophils between FD patients and healthy subjects, the number of eosinophils in the FD patients with postprandial distress syndrome (PDS) was greater than that observed in the controls (17). Presuming that there is some overlap between EG and FD, it is possible that anti-acid therapy may have improved our patient’s functional upper digestive tract disorders resulting from eosinophilic infiltration.

In summary, we herein presented a case of EG complicated with H. pylori infection. The patient’s EG was not improved by H. pylori eradication therapy, and we thus consider these two entities to be separate and unrelated. The administration of anti-acid medications should be considered in cases of EG that are unresponsive to eradication therapy.

The authors state that they have no Conflict of Interest (COI).

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References