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Chronic Eosinophilic Gastritis Presenting as Gastric Outlet Obstruction in a Young Man: A Case Report

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Eosinophilic gastritis, a relatively rare condition characterized by eosinophilic infiltration of the gastrointestinal wall, has a chronic course involving relapses and remissions. Herein, we describe the case of a 25-year-old man with chronic eosinophilic gastritis complicated by gastric outlet obstruction. The patient had a history of persistent gastrointestinal symptoms since adolescence and had developed severe dyspepsia and vomiting two months before presentation. Eosinophilic gastritis was confirmed using endoscopy and biopsy. The symptoms improved after steroid treatment, although the mucosal lesions did not completely resolve. This case shows that eosinophilic gastritis can have a chronic course with persistent mucosal lesions, despite symptomatic improvement. Early diagnosis and appropriate treatment of eosinophilic gastritis are important for preventing chronic gastric mucosal changes.

Keywords Gastritis; Eosinophilic enteropathy; Adolescent; Gastric outlet obstruction.

INTRODUCTION

Eosinophilic gastroenteritis (EGE) is a relatively rare disease. The prevalence of the disease is estimated to range from 8.4 to 28 cases per 100000 people.[1] EGE is characterized by eosinophilic infiltration of the gastrointestinal (GI) tract wall. Symptoms are nonspecific and include abdominal pain, vomiting, diarrhea, weight loss, ascites, and intestinal obstruction. Clinical manifestations vary depending on the location and depth of eosinophilic infiltration. Klein et al.[2] classified EGE into three major types based on the predominant layer of eosinophilic infiltration; mucosal, muscular, and subserosal. The etiology and natural history of EGE remain unclear; however, most cases follow a chronic course with repeated relapses and remissions.[3] In Korea, Jeon et al.[4] described 17 additional cases between 1994 and 2008. However, these reports remain sporadic. Although there have been studies on the diagnosis, progression, and treatment of allergy-related eosinophilic gastritis in children,[5] there are no reports of EGE beginning during adolescence and persisting chronically into adulthood. We aimed to confirm the chronic course of eosinophilic gastritis that started in adolescence and report a case in which eosinophilic gas-

tritis manifested as gastric outlet obstruction (GOO).

CASE REPORT

A 25-year-old man visited our hospital because of abnormal findings on an upper GI endoscopy performed at another hospital. The patient had a history of severe gastritis during adolescence and experienced dyspepsia and vomiting over the past two months. Endoscopy performed at another hospital revealed nodular and erythematous mucosa in the gastric antrum and pyloric ring (Fig. 1). The pyloric ring was narrowed and deformed, preventing the endoscope from passing through. Notably, similar findings were observed on an endoscopy performed 10 years previously. Biopsy performed two months ago revealed chronic active gastritis with moderate indeterminate cell hyperplasia. The patient was treated with a proton pump inhibitor (PPI). However, his symptoms did not improve, prompting his referral to our institution. Physical examination revealed no remarkable findings.

Blood tests revealed an elevated serum IgE level of 302 IU/mL (reference range; 0–100 IU/mL), but no other significant findings. Peripheral eosinophil counts were not significantly elevated. Multiple allergy serum test showed a positive result for *Dermatophagoides farinae* (mite) at level 2 (0.89). Additional tests for carcinoembryonic antigens, hepatitis A, B, and C virus, human immunodeficiency virus, and syphilis reagent were negative. Upper GI endoscopy revealed diffuse atrophic changes in the gastric mucosa. The antrum exhibited widespread erythema with nodular and discolored mucosal lesions, and similar lesions were observed in the gastric body (Fig. 2). The lesions extended to the pyloric ring and severe narrowing prevented entry into the duodenum. Stomach computed tomography (CT) showed pyloric wall thickening, but no other significant findings. Based on endoscopic findings, we suspected atypical nodular gastritis accompanied by GOO. Differential diagnoses included lymphoma (mucosa-associated lymphoid tissue lymphoma) and Borrmann type 4 gastric cancer, which required exclusion. Endoscopic biopsies were obtained from the nodular mucosa of the antrum and lower body. Pathological examination revealed chronic gastritis with eosinophilic infiltration (eosinophil count; 90/HPF) (Fig. 3A). No evidence of *Helicobacter pylori* infection was observed.

Based on these findings, the patient was diagnosed with eosinophilic gastritis and treated with oral prednisolone at 30 mg daily for two weeks. After two weeks of steroid therapy, follow-up endoscopy showed some improvement in the GOO, allowing the endoscope to pass through the pyloric ring. However, widespread erythema with nodular and discolored mucosal lesions in the antrum and similar lesions in the gastric body were observed. Biopsy results showed chronic gastritis. Prednisolone was tapered over three months. Follow-up endoscopy performed after three months revealed that extensive nodular mucosal lesions in the antrum and body remained present, although there was some improvement in the overall mucosal erythema, nodular lesions, and obstruction of the pyloric ring (Fig. 4). Biopsy results showed chronic gastritis without any significant findings (Fig. 3B). Repeat IgE testing showed a slight decrease from 302 to 279 IU/mL, whereas the peripheral blood eosinophil count remained relatively unchanged at 339/mm^3 (6.7% of 5070/mm). The patient was monitored at an outpatient clinic and showed no notable GI symptoms. After one year, follow-up endoscopy still showed erythema and nodular mucosal lesions, but the GOO had resolved, and there were no significant changes compared with previous findings.

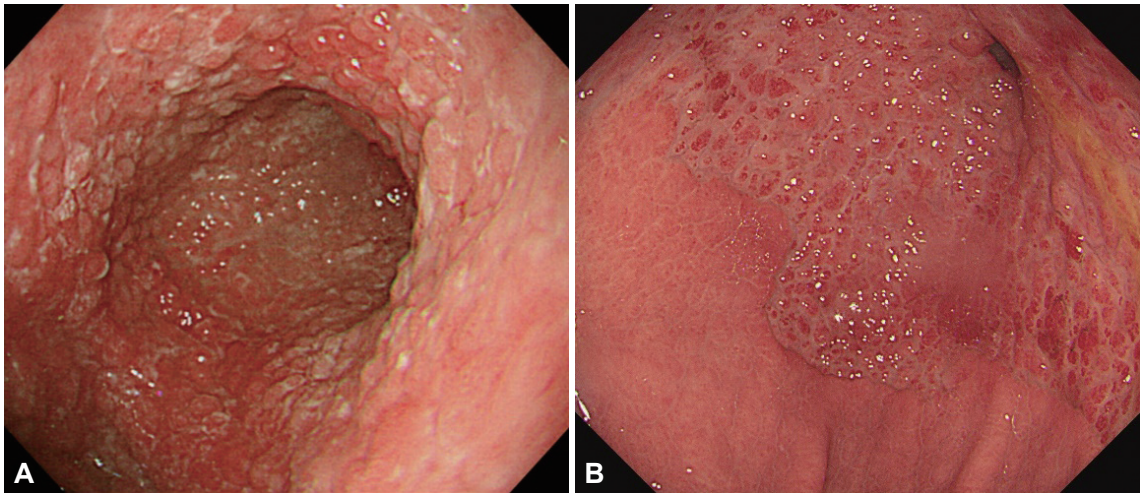

Fig. 1. Endoscopic images. A: Endoscopic image shows multiple erythematous and nodular mucosal lesions (10 years previously). B: Endoscopic image obtained two months previously.

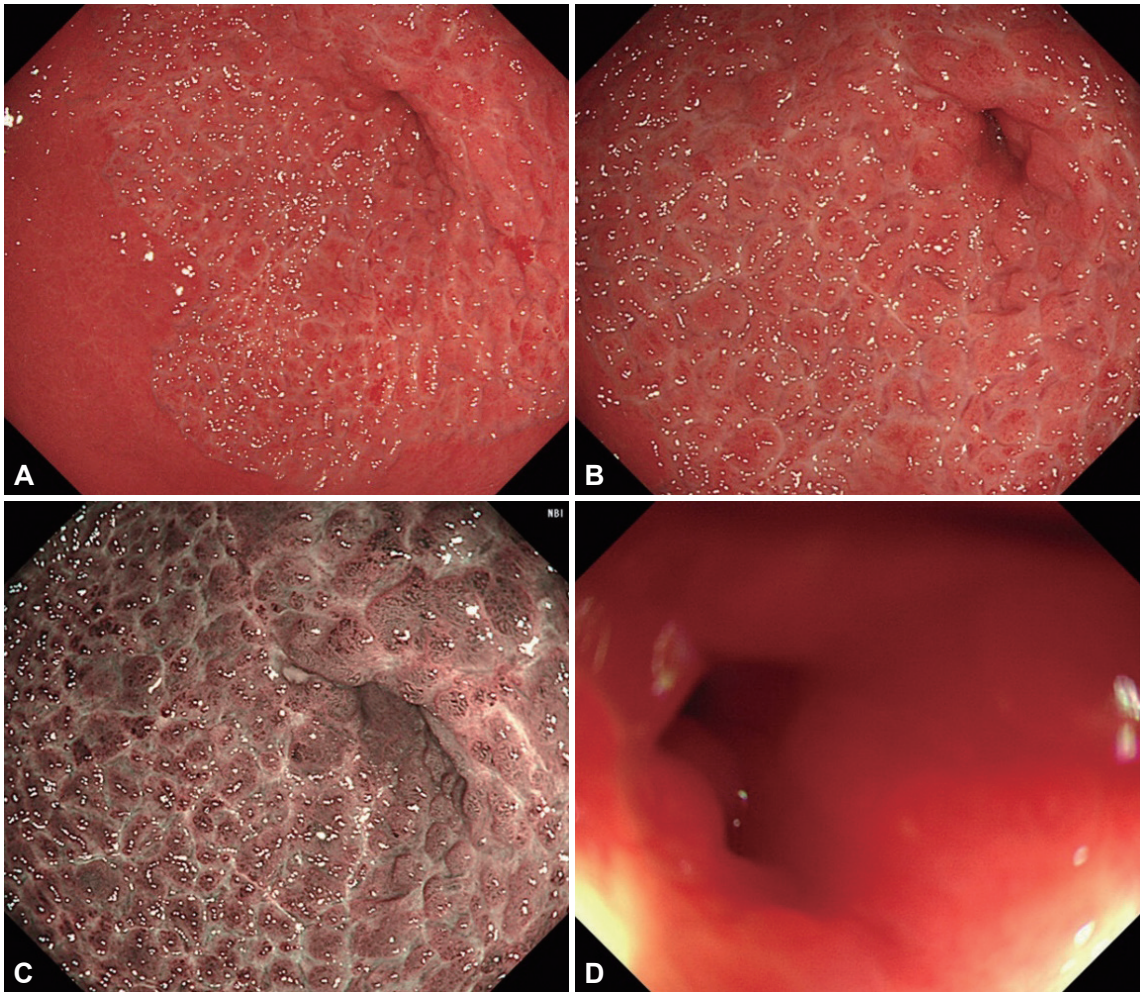

Fig. 2. Endoscopic images obtained at our hospital before steroid treatment. A-C: Nodular mucosa with discoloration and erythema is observed from the antrum to the pyloric ring, with a clear demarcation from the surrounding normal mucosa. D: The pyloric ring is narrow; therefore, the endoscope is difficult to pass through.

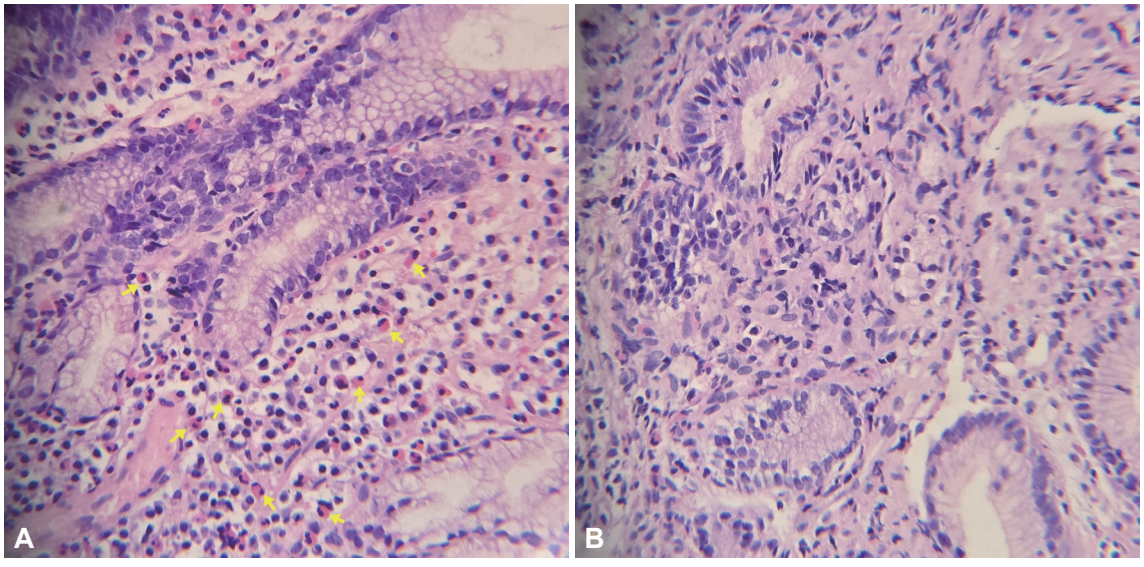

Fig. 3. Histopathological findings (hematoxylin and eosin, ×400). A: Diffuse eosinophilic infiltration is observed in gastric biopsy before steroid treatment (yellow arrows). B: No eosinophilic infiltration is observed after steroid treatment.

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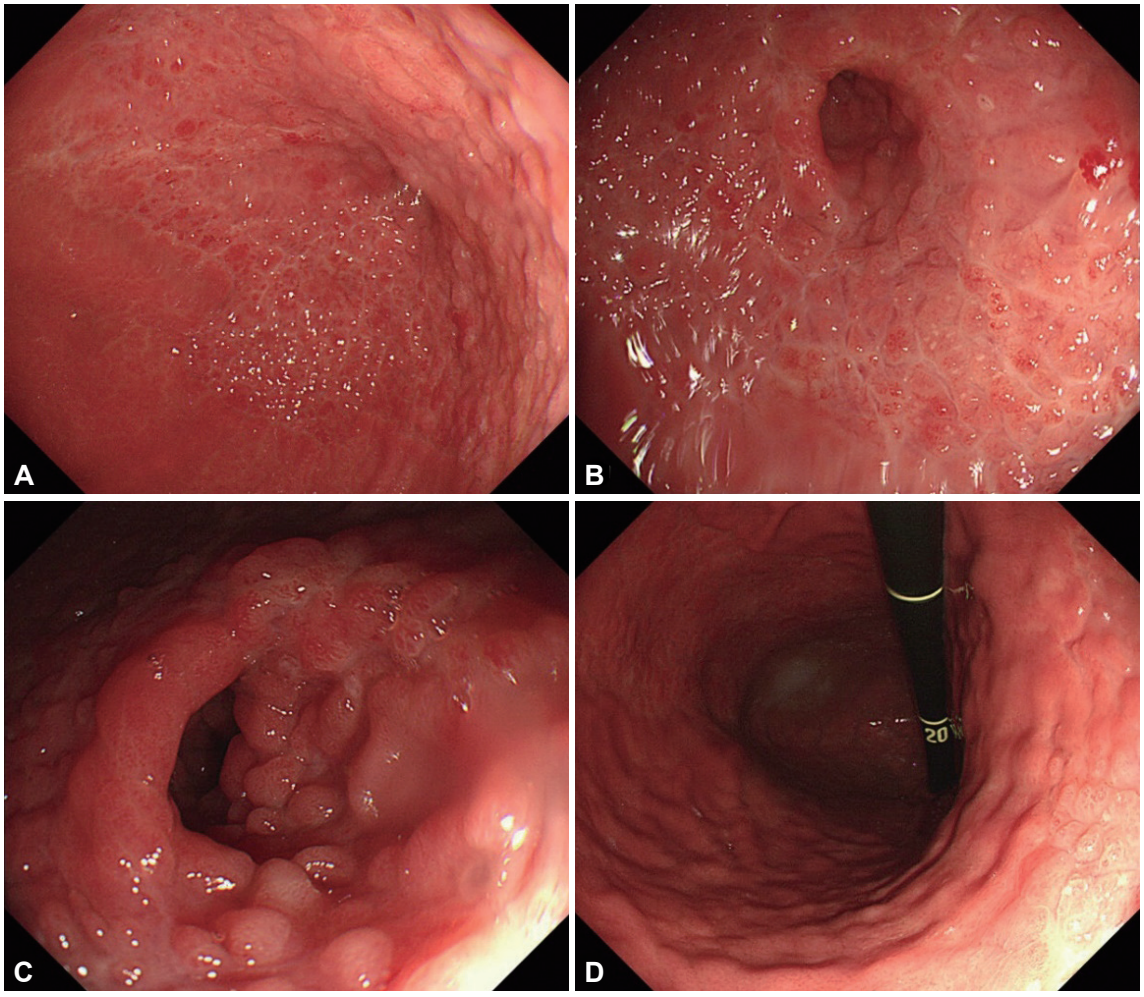

Fig. 4. Follow-up endoscopic images obtained three months after steroid treatment. A and B: Nodular mucosal lesions in the antrum are still present, but overall mucosal erythema and nodularity have improved compared to the initial findings. C: Pyloric stenosis has partially improved, allowing endoscopic passage through the pylorus, which was previously difficult. D: Mucosal lesions in the gastric body have also shown overall improvement.

DISCUSSION

EGE is a primary eosinophil-associated disorder of unknown etiology characterized by eosinophilic infiltration of the GI tract. Currently, diagnostic criteria have been established only for eosinophilic esophagitis, while criteria for other regions have not yet been clearly defined. As a result, the diagnostic criteria proposed by Talley et al.[6] in 1990 are commonly used. Diagnosis is confirmed when all four of the following conditions are met: 1) the presence of GI symptoms, 2) evidence of eosinophilic infiltration (eosinophils >20 HPF) or eosinophilic ascites in one or more locations on GI biopsy or surgical tissue, 3) absence of eosinophilic infiltration in areas outside the digestive tract, and 4) no evidence of parasitic infection.[6] EGE diagnosis requires the exclusion of other diseases that may cause eosinophilia, such as malignant lymphoma, hypereosinophilic syndrome, malignancies, parasitic infections, and drug exposure.[3] In cases with GOO accompanied by pyloric stenosis, eliminating Borrmann type 4 gastric cancer is crucial. This case was diagnosed and treated as EGE based on the absence of other conditions causing eosinophilia, presence of GI symptoms, and significant eosinophilic infiltration observed on biopsy. Malignant GOO was excluded using CT, follow-up endoscopic biopsies, and a review of endoscopic images obtained 10 years previously.

Endoscopic findings in EGE range from normal to nonspecific gastritis and often include mucosal erythema, mucosal friability, and nodular mucosa. Other findings, such as ulcers, thickened folds, and polypoid mucosa, have been reported.[3,7] In rare cases,[8,9] GOO may occur if the gastric antrum and pyloric ring are affected, as was observed in this case. Despite symptom improvement following medical treatment, the mu-

cosal lesions remained partially unresolved. Endoscopic images obtained 10 years previously showed similar findings, including nodular mucosal changes with erythema and mucosal friability, which were similar to those observed in a recent study. However, no pyloric stenosis was observed. The patient was presumably treated with medications for atypical gastritis. Although the mucosal inflammation improved to some extent, the lesions were never completely resolved. Recent endoscopy findings were considerably unchanged from 10 years previously, excluding marked narrowing of the pyloric ring, which impedes endoscope passage. After steroid therapy, the extent of the lesions, nodularity, and erythema improved, and pyloric stenosis eased to allow endoscopic access, although it remained significantly narrower than that 10 years previously.

Comparative analysis of serial endoscopic images showed that during recurrence or worsening of the disease, nodular lesions increased in height, erythema intensified, and mucosal abnormalities expanded concentrically from pre-existing lesions, creating a clearer demarcation from the normal mucosa. With improvement, the nodules became flatter, erythema diminished, lesion extent was reduced, and boundary with the normal mucosa blurred. Although symptom resolution correlated with reduced severity of mucosal abnormalities, the lesions and stenosis never completely disappeared. Chronic inflammation may cause scarring and pyloric stenosis. These findings suggest that eosinophilic gastritis is a chronic condition characterized by repeated exacerbations and remissions, with persistent mucosal changes resulting in long-term complications, such as GI stenosis, obstruction, and irreversible mucosal remodeling.

Several factors may explain the persistence of the mucosal lesions. First, repeated inflammatory responses can cause irreversible mucosal damage. Chronic tissue changes, such as fibrosis, may persist despite symptomatic improvement. Second, the resolution of symptoms may not equate to complete pathological remission. Residual eosinophils in the mucosa, even in small amounts, may continue to cause damage. Third, damaged mucosa may take time to regenerate and repeated chronic inflammation may delay mucosal healing. Finally, persistent exposure to allergens, such as specific foods or environmental factors, may contribute to chronic inflammation and ongoing mucosal changes. Other potential factors include immune dysregulation, eosinophilic hyperactivation, and underlying conditions, such as eosinophilia or autoimmune disease.

In this case, the most likely cause of recurrent inflammation and symptoms is persistent exposure to environmental allergens and inadequate treatment. As mentioned above, the patient has been treated with PPIs for symptom control rather than with steroids or allergen avoidance, which are the fundamental treatments, over the past 10 years. While PPIs may provide some benefit in the initial treatment of EGE, they have limited effectiveness in treating recurrent EGE. Furthermore, the patient was identified as being allergic to *D. farinae* through multiple allergy serum test. *D. farinae* is a dust mite commonly found on sofas, beds, and cotton fabrics, and it is likely that the patient has been persistently exposed to it.

Unlike eosinophilic esophagitis, there are no established treatment guidelines for eosinophilic gastritis, although steroids are considered the standard therapy.[3,10] Steroids reduce inflammation by inhibiting cytokines, such as interleukin-3 (IL-3), IL-5, and GM-CSF, which are involved in eosinophil survival and activation, and suppressing chemokines that direct eosinophil migration to inflamed areas.[10,11] Initial steroid therapy reportedly achieves a response in >90% of cases, typically involving 30–40 mg of oral prednisolone daily for 6–8 weeks, followed by gradual tapering. Here, persistent mucosal lesions observed on follow-up endoscopy despite clinical improvement suggested that irreversible mucosal damage had occurred, increasing the likelihood of chronic disease progression and recurrence. Long-term low-dose steroid therapy, steroid-sparing agents or biological agents may be considered in such cases.[12,13]

Steroid-sparing agents include selective leukotriene receptor antagonists and mast cell stabilizers. Currently, biological agents targeting inflammatory mediators such as type 2 inflammatory cytokines are being studied.[14] Dupilumab is a fully human monoclonal antibody that blocks the common receptor components of IL-4 and IL-13, central mediators of type 2 inflammation. Dupilumab is approved for the treatment of several type 2 inflammatory diseases, including atopic dermatitis, asthma, chronic rhinosinusitis with nasal polyps, and eosinophilic esophagitis.[14] Benralizumab is a monoclonal antibody targeting IL-5Rα and induces rapid and almost complete eosinophil depletion through antibody-dependent cell-mediated cytotoxicity.[15]

Overall, this case demonstrates that eosinophilic gastritis can follow a chronic course with permanent mucosal damage if appropriate treatment is not initiated early. Prevention of chronic mucosal changes through timely and appropriate therapy is crucial. Long-term steroid therapy or treatment with steroid-sparing agents should be considered in patients with chronic symptom recurrence.

Authors' Contribution

Conceptualization: Jin Lee. Data curation: Na Young Kim. Investigation: Na Young Kim. Methodology: Ji Eun Na, Yong Eun Park. Supervision: Jin Lee, Jongha Park, Tae Oh Kim. Writing—original draft: Na Young Kim. Writing—review & editing: Jin Lee. Approval of final manuscript: all authors.

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Ethics Statement

All procedures were conducted in compliance with the tenets of the Helsinki Declaration. Informed consent was obtained from patient for publication of this study.

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