A Case of Steroid Dependent Eosinophilic Gastroenteritis Presenting as a Huge Gastric Ulcer

INTRODUCTION

Eosinophilic gastroenteritis is quite rare disease and characterized by eosinophilic infiltration of the gastrointestinal tract in the absence of other causes for eosinophilia. It can affect both children and adults, but typical presentations are in the third through fifth decade with a male predominance. It is associated with various clinical gastrointestinal manifestations and it depends on which layer and site are involved. In eosinophilic gastroenteritis, endoscopic findings are non-specific: thickened folds, erythema, friability, nodularity, gastric outlet obstruction, small gastric or duodenal ulcer, and even normal mucosa. However, huge ulceration is extremely rare finding. Herein, we present a case of eosinophilic gastroenteritis, which is characterized by huge gastric ulceration like a Borrmann type III advanced gastric cancer. It is a unique presentation of eosinophilic gastroenteritis.

CASE REPORT

A 38-year-old female patient suffered from nausea, vomiting and epigastric pain. She lost 5 kg of her weight for 2 weeks. She had no personal and familial history of specific disease. She felt tender in the epigastrium by palpation. In laboratory investigation, white blood cell count was $5.7 \times 10^9/L$ with 7%
eosinophils. There were no other specific findings in laboratory examination, chest and abdominal radiography. ELISA of parasite was negative. Esophagogastroduodenoscopy revealed huge and deep ulceration measuring 7×8 cm, located on the lesser curvature of the stomach, just like a Borrmann type III advanced gastric cancer (Fig. 1). Although there was no malignant cell in biopsy specimen, it was difficult to rule out malignancy. Subtotal gastrectomy with Billroth II anastomosis was done. The surgical specimen showed marked stromal reaction and dense eosinophilic infiltration in full thickness of gastric wall. There was no evidence of malignancy on histological examination (Fig. 2, 3). Symptoms disappeared and blood eosinophil count was normalized after surgery without any medication.

After 5 years, she complained diarrhea, abdominal pain and generalized edema. Blood eosinophil count was elevated up to 10.5% (absolute eosinophil count up to $0.87 \times 10^9/L$) and serum IgE level were also elevated to 2,179 IU/mL. Serum albumin level fell to 20 g/L. Computed tomography of abdomen showed diffuse wall thickening of the whole small bowel and colon (Fig. 4). Endoscopy showed no active mucosal lesion, we performed biopsy procedure on remnant stomach, terminal ileum and colon for surveillance. It showed eosinophilic infiltration and lymphoid hyperplasia (Fig. 5). Steroid therapy was started and her symptoms progressively improved. However, she had frequent relapses and was dependent on steroid therapy to maintain remission. The side effects of steroid therapy were fre-

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**Fig. 1.** Initial endoscopic findings. A huge and deep ulceration was observed in the antrum and it looked like advanced gastric cancer.

**Fig. 2.** Microscopic findings of the surgical specimen. (A) Marked stromal reaction was seen, and there was no evidence of malignancy on histological examination (H&E, ×100). (B) Dense eosinophilic infiltration was observed in mucosa and submucosa (H&E, ×400).
Fig. 3. Microscopic findings of the surgical specimen. There was eosinophilic infiltration in muscle layer (H&E, A; ×100; B; ×400).

Fig. 4. Abdominal computed tomography. It revealed diffuse wall thickening of the small bowel and colon.

Quently responsible for discontinuation of therapy. Azathioprine (1 mg/kg) and other immune suppressors were used, but it could not prevent symptoms from appearing. After 3 years, she developed dyspnea and suddenly died. We suspected that the cause of death would be infective endocarditis due to the prolonged use of immune suppressants.

**DISCUSSION**

The diagnosis of eosinophilic gastroenteritis is provided by the following criteria; presence of gastrointestinal symptoms, eosinophilic infiltration in one or more biopsied areas, absence of eosinophilic involvement in other organs and absence of a parasite infection. It is categorized into mucosal, muscular, and serosal types. Mucosal disease is the most common type and produces symptoms such as vomiting, diarrhea, gastrointestinal bleeding, malabsorption, or protein-losing enteropathy. Involvement of muscular layer produces the symptoms of bowel obstruction, whereas serosal involvement results in ascites or peritonitis. Eosinophilic gastroenteritis can affect any part of gastrointestinal tract from the esophagus to the rectum, and stomach is the most common site of involvement, especially antrum. Some cases of small and shallow gastric ulceration have been reported, but deep and giant gastric ulcers are extremely rare presentation of eosinophilic gastroenteritis. Until now, two cases of giant ulcers as initial manifestation of eosinophilic gastroenteritis were reported in the world. In muscular type of this disease, infiltration of eosinophils predominantly in muscular layer is a characteristic finding. Because mucosal involvement is not always accompanied with muscular type, it is probably insufficient to diagnose with mucosal biopsy only. Full-thickness surgical biopsies may be required for accurate diagnosis, if the disease process is confined to the muscle layer. Most of the reported cases are diagnosed by full-thickness operative biopsy or surgical resection for obstruction or suspicion of malignancy. Previous studies suggested that EUS would be of value in ascertaining muscular involvement in eosinophilic gastroenteritis. The role of EUS for diagnosis of eosinophilic gastroenteritis will be evaluated particularly in muscular...
Another possible etiology of our patient’s symptoms was HES. The criteria required to diagnose HES are peripheral eosinophilia exceeding 1,500 cells/mL for more than 6 consecutive months, absence of an underlying cause of hyper eosinophilia despite extensive evaluation and presence of organ damage or dysfunction related to hypereosinophilia. HES has variable target organ dysfunction such as skin, heart, lung, and central or peripheral nerve system but eosinophilic gastroenteritis doesn’t have any other target organ damage except gastrointestinal tract. In our patient, apart from the involvement of gastrointestinal tract, there was no other organ involvement which makes the diagnosis of HES unlikely.

Although there is no treatment consensus in eosinophilic gastroenteritis, steroid therapy is a cornerstone of treatment. About 90% patients respond to steroid, especially in serosal type. In muscular type, especially presenting as gastric ulcer, the exact response rate for steroid therapy is not known. In previous cases presenting with huge gastric ulcer, steroid therapy was successful. However, in our case, the patient had frequent relapses and was dependent on steroid therapy to maintain remission. Several studies suggested that azathioprine or mycophenolate mofetil could have a benefit as a steroid-sparing agent in patients requiring high doses for maintenance, but there were no clinical trials on azathioprine treatment. Ketotifen, mast cell stabilizers, or leukotriene antagonist might be effective, and further evaluation to prove benefit is required.

In conclusion, endoscopic features of eosinophilic gastroenteritis can be various, but huge gastric ulcer is very rare manifestation of the disease. Huge gastric ulcer should be considered as one of the manifestations of eosinophilic gastroenteritis. It showed bizarre clinical course that was quite different from what we know. New therapeutic modality should be needed for the cases that were refractory to the traditional therapy. The relationship between this disease and huge gastric ulcer will be elucidated.

REFERENCES

5. Scolapio JS, DeVault K, Wolfe JT. Eosinophilic gastroenteritis