

## Eosinophilic Gastroenteritis with Ileus and Ascites

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**We report a case of eosinophilic gastroenteritis, which has features of the predominant subserosal type presented as an ileus and ascites. A 48-year-old Japanese woman was admitted to our hospital because of epigastralgia, lower abdominal pain and vomiting. She had a past history of allergic disorders. The computed tomographic scan revealed ascites, and marked wall thickening and dilatation of the intestine. This patient showed eosinophilic ascites without marked peripheral eosinophilia. Histologic examination demonstrated eosinophilic infiltrates did not predominate in the gastrointestinal tract. Conservative treatment of intravenous infusion of antibiotics and Ringer's solution was effective in this case.**

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**Key words:** predominant subserosal disease, computed tomographic scan, food allergy

### Introduction

Eosinophilic gastroenteritis is a disease reported by Kaijser (1) in 1937. Klein et al (2) had demonstrated that this disorder could be pathologically classified into three major types: predominant mucosal, predominant muscle layer, and predominant subserosal. However, its clinical features, etiology, and treatment have not yet been definitely established. We report a case of eosinophilic gastroenteritis with features of the predominant subserosal type, presented as an ileus and ascites.

### Case Report

A 48-year-old Japanese woman was admitted to our hospital in December 1994 because of epigastralgia, lower abdominal pain, and vomiting. She had no past history of abdominal surgery. However, she had twice experienced urticaria after eating a slice of raw thickener and a sardine. Four days before admission she experienced urticaria after eating of a slice of slightly putrefied beef and some tuna. Later there were symptoms of gingivitis. One day before admission she took an analgesic. There was no family history of allergic diseases.

Her height was 157.4 cm, body weight 55.4 kg and her temperature was 37.2°C. There was no swelling of the lymph nodes. Physical findings of the heart and lungs were normal. There was no marked peripheral eosinophilia (Table 1). An abdominal X-ray film showed niveau of the small intestine, the so-called "herring bone" sign (Fig. 1). An abdominal computed

tomographic (CT) scan showed ascites, and marked wall thickening and dilatation of the lower intestine (Fig. 2). We examined the ascites obtained by puncture of abdominal cavity. Eosinophils were remarkably increased in the ascites (Table 2, Fig. 3). On the fifty hospital day, diffuse red spots of the antrum were found by gastrointestinal fiberscope. The biopsy specimen showed no remarkable eosinophilic infiltration (Fig. 4). On the seventh hospital day, benign sigmoid colonic polyps (10 mm) were found by colonic fiberscope. The biopsy specimen of the terminal ileum, cecum, ascending colon, transverse colon, descending colon, sigmoid colon and rectum showed no pathological evidence of eosinophilic infiltration of the mucosa. We diagnosed her disease as eosinophilic gastroenteritis having features of the predominant subserosal type presented as an ileus and ascites. On the fourth hospital day, she recovered from her symptoms by the conservative therapy of resting, fasting, and intravenous infusion of cefmetazole sodium and Ringer's solution.

### Discussion

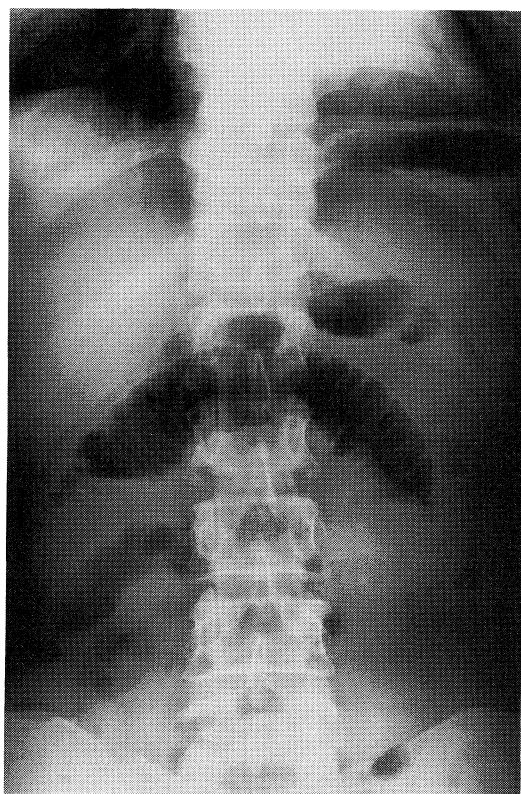
We reported a case of eosinophilic gastroenteritis that presented as an ileus and ascites. In this case, the findings of the abdominal CT scan showed marked wall thickening and dilatation of the lower intestine, as reported by Van Hoe et al (3) and Tanaka et al (4).

The features of eosinophilic gastroenteritis had been reported as the eosinophilic infiltration of gastrointestinal tract,

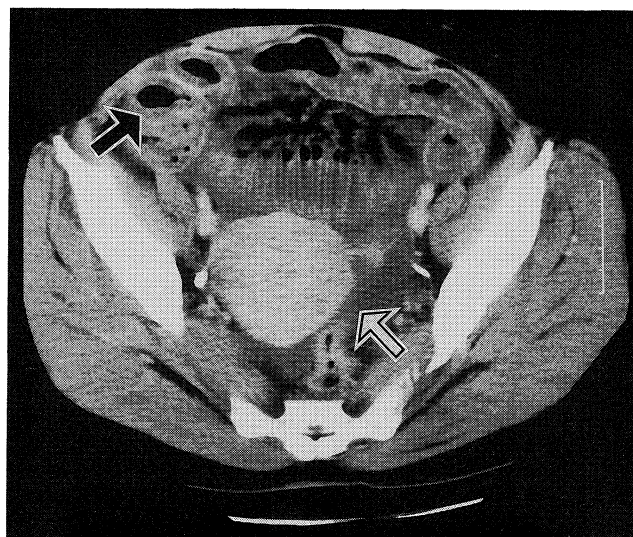
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**Table 1. Laboratory Findings on Admission**

White blood cells	6,350/mm <sup>3</sup>	Blood urea nitrogen	6 mg/dl
Neutrophil	52.40%	Creatinine	0.7 mg/dl
Lymphocyte	27.30%	Na	141 mmol/l
Monocyte	9.00%	K	5.0 mmol/l
Eosinophil	7.70%	Cl	104 mmol/l
Basophil	0.50%	Erythrocyte sedimentation rate	25 mm(1h)/60 mm(2h)
Red blood cells	363×10 <sup>4</sup> /mm <sup>3</sup>	C-reactive protein	0.81 mg/dl
Hemoglobin	12.2 g/dl	IgE	2,940 IU/ml
Hematocrit	38.40%	Urinalysis	
Platelet	20.1×10 <sup>4</sup> /mm <sup>3</sup>	Protein	(-)
Total protein	6.0 g/dl	Sugar	(-)
Albumin	3.7 g/dl	Blood	(-)
Asparate aminotransferase	18 IU/l		
Alanine aminotransferase	13 IU/l		
Lactate dehydrogenase	130 IU/l		
Alkaline phosphatase	155 IU/l		
Total bilirubin	0.3 mg/dl		
Direct bilirubin	0.1 mg/dl		
Indirect bilirubin	0.2 mg/dl		



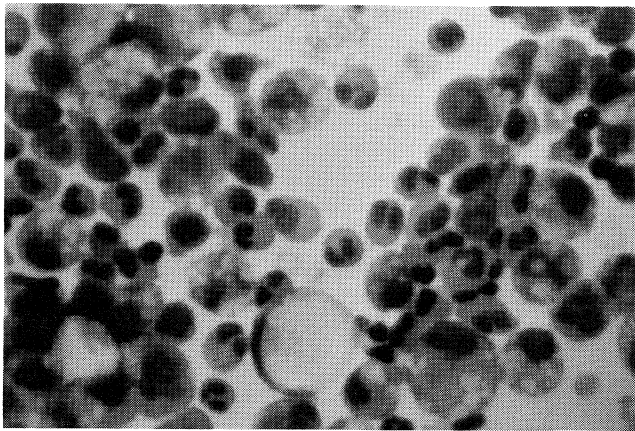
**Figure 1.** The findings of an abdominal X-ray film. The abdominal X-ray film showed niveau of the small intestine, the so-called "herring bone" sign.



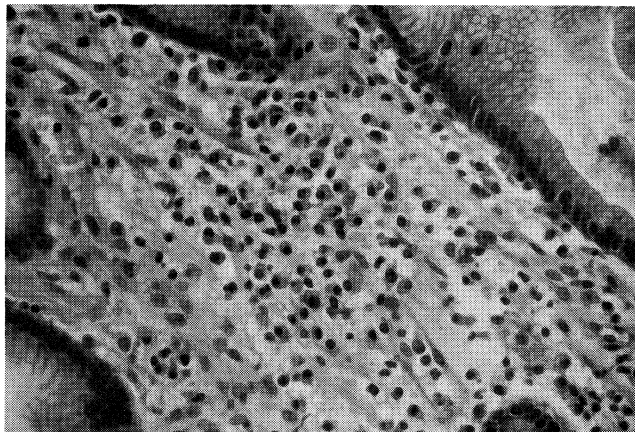
**Figure 2.** The findings of an abdominal CT scan. The abdominal CT scan showed ascites (white arrow), and marked wall thickening and dilatation of the lower intestine. The black arrow indicates the constriction of three layers in the small intestine.

**Table 2. Analysis of Ascites**

Specific gravity	1.034
Rivalta reaction	negative
Protein	5.0 g/dl
Sugar	109 mg/dl
Cell	
Eosinophil	50%
Monocyte	43%
Lymphocyte	7%



**Figure 3.** The photograph of microscopic findings of the ascites. Eosinophils were remarkably increased in the ascites (Giemsa stain,  $\times 1,000$ ).



**Figure 4.** The histologic section of the stomach. Eosinophils did not remarkably infiltrate into the mucosa (HE stain,  $\times 400$ ).

peripheral eosinophilia, and gastrointestinal symptomatology (5). However, according to Caldwell et al (6), these conditions are not necessarily all met. Fortman et al (7) reported a case of eosinophilic ascites without peripheral eosinophilia. Talley et al (8) demonstrated that peripheral eosinophilia was absent in 9 of 40 cases (22.5%). In the present case, eosinophilic ascites was present without marked peripheral eosinophilia, and there was no histological eosinophilic infiltration of the gastrointestinal tract. The findings of the CT scan played an important role in the diagnosis of eosinophilic gastroenteritis. The diagnosis of this disease seems to be aided by the findings of CT scan (3, 4).

In 1970, Klein et al (2) reported that this disorder could be pathologically classified into three major types: predominant mucosal, predominant muscle layer, and predominant subserosal. According to the Klein classification, the incidence of the predominant subserosal type was the lowest as in the following reports: 9% (5 of 54 cases) (4), 4.5% (2 of 44 cases)

(9) in Japan, 37.5% (3 of 8 cases) (10) in Taiwan, and 12.5% (5 of 40 cases) (8) in USA. The present case had features of the predominant subserosal type.

The etiology of eosinophilic gastroenteritis is currently unknown. Allergies to certain food and immunological abnormalities have been speculated as its etiology (11–13). On the other hand, some investigators (5, 6, 14) have demonstrated that the etiology of this disorder is not associated with allergic reactions. In our case, there was a past history of food allergy. However, we could not determine which food was the source of the allergy. The findings of eosinophilic ascites and high serum IgE levels suggested allergic disorders, parasitic disorders, and abdominal lymphoma. The likelihood of parasitic disorders and abdominal lymphoma was remote because of the history of the present illness, physical findings, and the findings of CT scan.

Many investigators (6, 7, 9, 10, 14–16) have reported the clinical usefulness of corticosteroid, ketotifen, sodium cromoglycate and surgery as the treatment of eosinophilic gastroenteritis. In the present case, we considered operative therapy as the symptoms and findings did not improve with conservative therapy within 24 hours of admission. Fortunately, the symptoms and findings tended to improve the next day. Accordingly we continued the conservative treatment of intravenous infusion of antibiotics and Ringer's solution. In light of the clinical course, in which the ileus was induced by specific foods and improved over the course of time, we selected the conservative therapy. In this case, there was little need for treatment with steroids, ketotifen, or sodium cromoglycate because of the good clinical course.

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