

## A Patient with Eosinophilic Gastroenteritis Presenting with Acute Pancreatitis and Ascites

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Eosinophilic gastroenteritis (EGE) is a rare disease characterized by focal or diffuse eosinophilic infiltration of the gastrointestinal tract, especially the stomach and duodenum. EGE has vague, nonspecific symptoms, including nausea, vomiting, abdominal pain, diarrhea, weight loss, ascites, and malabsorption. Here, we report a patient with EGE presenting with concurrent acute pancreatitis and ascites. A 68-year-old woman was admitted with abdominal pain, nausea, vomiting, and watery diarrhea. Laboratory findings revealed elevated serum titers of amylase, lipase, and peripheral blood eosinophil count. An abdominopelvic computed tomography scan showed a normal pancreas, moderate amount of ascites, and duodenal thickening. A esophagogastroduodenoscopy showed patchy erythematous mucosal lesions in the 2nd portion of the duodenum. Biopsies from the duodenum indicated eosinophilic infiltration in the lamina propria. The patient was successfully treated with prednisolone and montelukast. Despite its unusual occurrence, EGE may be considered in the differential diagnosis of unexplained acute pancreatitis, especially in a patient with duodenal edema on imaging or peripheral eosinophilia. (**Gut Liver 2014;8:224-227**)

**Key Words:** Eosinophilic gastroenteritis; Pancreatitis; Eosinophilia; Ascites

### INTRODUCTION

Eosinophilic gastroenteritis (EGE) is a rare disease characterized by eosinophilic infiltration of the gastrointestinal (GI) tract. Clinical presentation of EGE is variable and may present as an acute abdomen due to intestinal or colonic obstruction, intussusception, acute pancreatitis, and perforation.<sup>1</sup> The com-

mon causes of acute pancreatitis are gallstones or alcoholism. Eosinophilic infiltration of GI tract may be one of the etiologies in the case of recurrent pancreatitis although EGE presenting as pancreatitis is extremely rare. We report a case of EGE presenting as acute pancreatitis with concurrent ascites, and we discuss the clinical characteristics and differential diagnosis.

### CASE REPORT

A 68-year-old woman was admitted with abdominal pain, nausea, vomiting, and watery diarrhea after eating a high protein and fat diet ("Chueotang" which is loach in hot bean paste soup) 1 day ago. She had a history of recurrent epigastric pain and nausea for the past 3 years. There was no history of drug allergy, bronchial asthma, allergic rhinitis or hepatitis except for hypothyroidism. She had been taking levothyroxine, cimetidine, and rebamipide for 2 years and had not taken a new medicine recently. She was not consuming alcohol or any illicit drug. Physical examination was unremarkable except for mild epigastric tenderness. Laboratory investigations indicated a white blood cell count of 6,800/mm<sup>3</sup> with high percentage of eosinophils (18.4%). The serum biochemistry showed an aspartate aminotransferase of 50 IU/L, alanine aminotransferase of 39 IU/L, amylase of 253 U/L, lipase of 478.71 U/L, and triglycerides of 66 mg/dL. Serum immunoglobulin E (IgE) and immunoglobulin G4 (IgG4) levels were in the normal range. Antinuclear antibody was negative. Abdominopelvic computed tomography (CT) was performed under the diagnosis of acute pancreatitis. It revealed diffuse wall thickening in the duodenum at the 2nd and 3rd portions, mild edema of small bowel loops and a moderate amount of ascites. However, there was no evidence of gallstones, abnormality of the pancreas or peripancreatic fluid collection (Fig. 1). We diagnosed her as Balthazar grade A of acute

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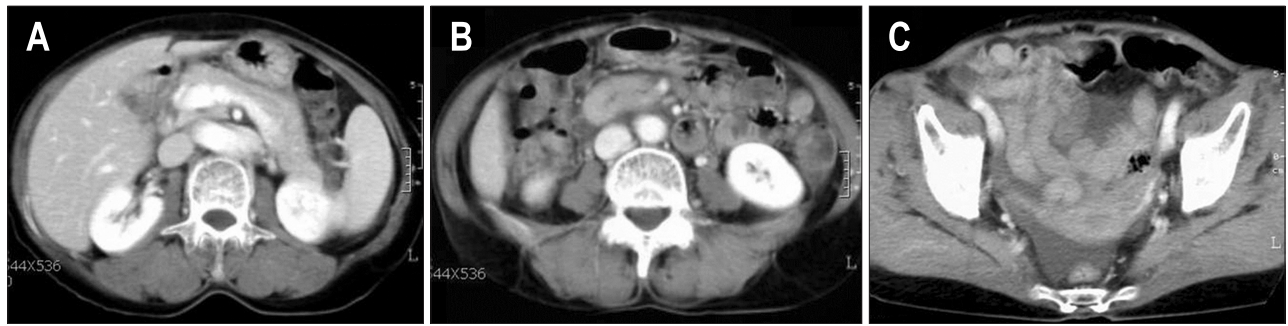
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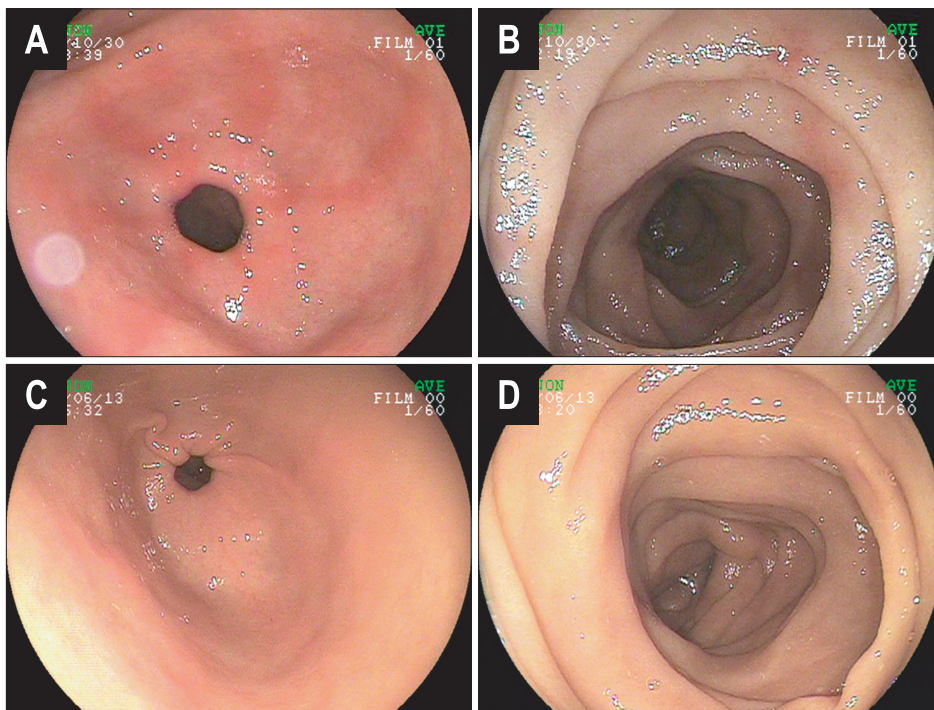
Received on May 19, 2013. Revised on July 30, 2013. Accepted on August 2, 2013.

pISSN 1976-2283 eISSN 2005-1212 <http://dx.doi.org/10.5009/gnl.2014.8.2.224>

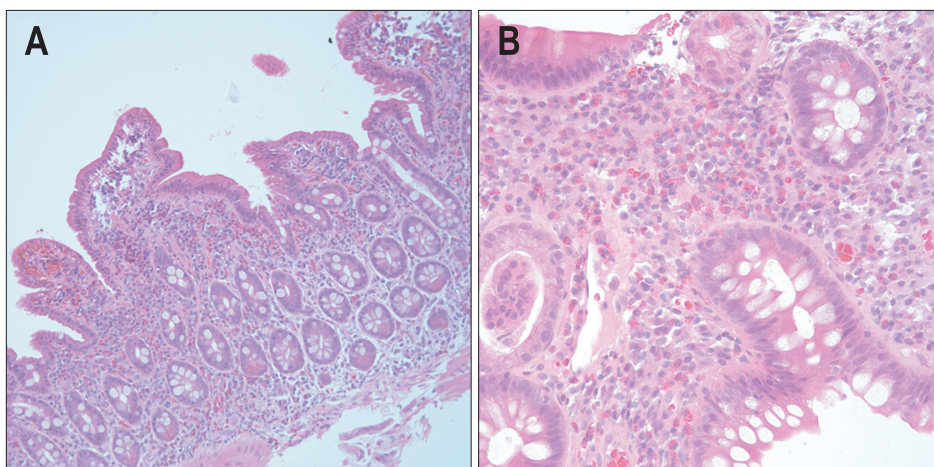
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**Fig. 1.** Abdominopelvic computed tomography (CT) scan. (A) CT image shows a normal pancreas without evidence of a pancreatic mass, edema, necrosis, or peripancreatic fluid collection. (B) Duodenal wall thickening is noted in the duodenum in the 2nd and 3rd portions. (C) Ascites is present in the posterior cul-de-sac.



**Fig. 2.** Esophagogastroduodenoscopy. (A) Second-look endoscopy shows multiple linear hyperemic mucosal lesions on the antrum. (B) Multiple hyperemic patchy mucosal lesions and mild wall thickening are noted in the 2nd portion of the duodenum. (C) On follow-up endoscopy after 8 months, the gastric antral mucosal lesions are improved. (D) The hyperemic patchy lesions in the duodenum have also resolved.



**Fig. 3.** Duodenal biopsy histology. (A) Eosinophilic infiltration with mixed inflammatory cells is noted in the lamina propria (H&E stain, x200). (B) Microscopic view shows more than 50 eosinophils/high-power field (H&E stain, x400).

pancreatitis based on the elevated amylase/lipase and normal pancreas on CT. Esophagogastroduodenoscopy (EGD) was performed to examine the duodenal wall thickening found on CT. EGD findings showed mild erythematous mucosal change with atrophy on the antrum but the duodenum was unremarkable. Bowel rest and supportive care including intravenous fluids and acid-reducing agents were initiated. The level of amylase and lipase decreased to the normal range on the following day and her abdominal pain gradually decreased within a week. However, mild epigastric pain persisted and subsequent laboratory examination on the 7th hospital day showed that blood eosinophils had increased to 39.8% and the eosinophil count was  $3.5 \times 10^3/\mu\text{L}$  (normal,  $0.04\text{--}0.5 \times 10^3/\mu\text{L}$ ). Stool examination for ova and parasites was negative. Allergen skin prick test was also negative. At that time, we thought of the possibility of EGE because her GI symptoms continued even after treatment and the eosinophilia was more aggravated without any other obvious cause. So we decided to perform EGD and colonoscopy again to take a biopsy from the GI tract. The 2nd look-EGD revealed aggravated linear mucosal erythema of the antrum and multifocal patchy erythematous mucosal lesions on the 2nd portion of the duodenum, which were not observed in the initial EGD (Fig. 2A and B). However, there was no evidence of papillitis. Colonoscopy revealed erythematous mucosal changes of the terminal ileum. Multiple biopsies were obtained from the normal mucosa to the erythematous lesions on the stomach, duodenum and ileum. Histological examination indicated chronic inflammation with increased eosinophils in the lamina propria from the duodenal erythematous lesion (Fig. 3). The patient was treated with 30 mg of prednisolone and 10 mg of montelukast for 1 month. Her symptoms improved immediately and the eosinophil count normalized within 2 weeks. Prednisolone was tapered over 8 weeks and montelukast was used 4 more months. The follow-up EGD after 8 months showed normal antral and duodenal mucosa (Fig. 2C and D). One year after presentation, her symptoms recurred, with elevated levels of amylase, lipase and blood eosinophil counts. The patient was treated with oral prednisolone and montelukast again, and then her upper abdominal pain resolved and the eosinophil count normalized.

## DISCUSSION

EGE is a rare disease of unknown etiology and is defined as a GI disorder of undetermined cause characterized by infiltration of eosinophils in the GI tract.<sup>2</sup> EGE presenting with acute pancreatitis was first reported in 1973.<sup>3</sup> Thereafter, several papers on EGE associated with pancreatitis have been reported.<sup>4-12</sup> The diagnosis of EGE may be difficult and requires a high index of suspicion since EGE has a wide spectrum of clinical presentations. The patient in the current case presented with abdominal pain, nausea, diarrhea and elevated levels of amylase and lipase. She also had peripheral blood eosinophilia at presentation.

However, it was overlooked carelessly and she was initially diagnosed as having acute pancreatitis. EGE should be considered in any patient with GI symptoms associated with peripheral eosinophilia because it is associated with peripheral blood eosinophilia in nearly 30% to 80% of the cases.<sup>13</sup>

The definite diagnosis of EGE is established by demonstrating eosinophilic infiltration on endoscopic, laparoscopic or laparotomic biopsies. Endoscopic findings in EGE may vary from normal mucosa to mild erythema, thickened mucosal folds, nodularity, and frank ulceration.<sup>1</sup> This patient was finally diagnosed with EGE according to the pathologic findings from the duodenal erythematous mucosal lesion and there was no evidence of other causes for eosinophilia, such as parasitic infestation or drugs.

Patients of EGE are divided by the Klein classification into those with predominantly mucosal, muscle layer or subserosal disease. The less common form of EGE is the subserosal disease, defined by the presence of eosinophilic infiltration of the gut and eosinophilic ascites.<sup>13</sup> We thought our case might be the subserosal type, although we could not identify eosinophilic ascites by paracentesis or take a biopsy specimen from deeper tissue. The reason is that she had eosinophilic infiltration of the duodenum and ascites concurrently. In addition, initial endoscopic findings showed nonspecific duodenal mucosa despite the duodenal wall thickening and ascites on CT. The duodenal mucosal changes were found later when the eosinophilia became more aggravated.

One of the important clues for the diagnosis of EGE in this patient was the duodenal wall thickening on CT. It is believed that eosinophilic infiltration can cause edema, fibrosis and distortion in the ampulla and periampullary duodenum, leading to pancreatitis,<sup>8</sup> but there was no evidence of papillitis in this patient. Not only our case but also most reported cases of EGE presenting with acute pancreatitis exhibited duodenal edema or thickening of mucosal folds on imaging studies.<sup>6-8</sup> Therefore EGE should be considered in patients with acute or recurrent idiopathic pancreatitis who have duodenal wall thickening on imaging study. During endoscopy in a patient with unexplained pancreatitis, the duodenum should be carefully examined and multiple biopsies should be taken from normal mucosa as well as from the abnormal mucosal lesion.

The differential diagnosis of EGE includes a variety of disorders that may have GI symptoms and peripheral eosinophilia, such as Crohn's disease, intestinal lymphoma, hypereosinophilic syndrome, and parasitic infestation.<sup>2</sup> Because autoimmune pancreatitis (AIP) is also associated with peripheral eosinophilia, with a prevalence of 28%,<sup>14</sup> it should be differentiated from the current case. AIP can be diagnosed by typical CT findings such as diffuse enlargement with homogeneous attenuation and the peripheral rim of a hypoattenuation, and elevated serum IgG4.<sup>15</sup> However, our patient exhibited a normal pancreas on CT and normal serum IgG4.



Steroids remain the mainstay of therapy in patients of EGE, although no controlled trials are available. Recently, some alternatives to steroids, such as sodium cromoglycate, montelukast, suplatast tosilate, and ketotifen, have been used.<sup>2</sup> We successfully treated this patient with steroids and montelukast initially. And the efficacy of these mediations was not reduced in the recurrent attack.

In conclusion, EGE may present as acute pancreatitis or a pancreatic mass. Duodenal edema or thickening caused by eosinophilic infiltration are common findings in these patients. EGE may be considered in the differential diagnosis of unexplained acute pancreatitis, especially in a patient with duodenal edema on an imaging study or peripheral eosinophilia.

### CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

### ACKNOWLEDGEMENTS

This paper was supported by Wonkwang University 2011.

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