

Received: 2016.01.04
Accepted: 2016.02.06
Published: 2016.04.18

ISSN 1941-5923
© Am J Case Rep, 2016; 17: 259-263
DOI: 10.12659/AJCR.897403

A Case of Eosinophilic Gastroenteritis Forming a Rigid Chamber Mimicking Giant Duodenal Ulcer on Computed Tomography Imaging

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABDEF **Yoko Shimamoto**
ABDEF **Yohei Harima**

Department of Internal Medicine, Ubekosan Central Hospital Corporation, Ube-city, Yamaguchi, Japan

Corresponding Author: Yoko Shimamoto, e-mail: shima@kyosai.or.jp
Conflict of interest: None declared

Patient: Female, 67
Final Diagnosis: Eosinophilic gastroenteritis
Symptoms: Abdominal distension • abdominal pain • chronic diarrhea
Medication: —
Clinical Procedure: CT
Specialty: Gastroenterology and Hepatology





Objective: Rare disease
Background: The clinical manifestations of eosinophilic gastroenteritis are nonspecific and vary depending on which layer of the gastrointestinal tract is involved. Computed tomography (CT) is valuable for detecting and characterizing gastrointestinal wall abnormalities.

Case Report: We report a case of eosinophilic gastroenteritis that formed a chamber in the rigid duodenal wall of a 67-year-old woman. Abdominal CT showed symmetrical wall thickening of the gastric antrum and duodenal bulb, and the bowel walls consisted of 2 continuous, symmetrically stratified layers. There was a chamber mimicking a giant ulcer at the orifice of the descending duodenum. Eosinophilic inflammation was present through this rigid wall of the descending duodenum, accompanied by perienteric inflammation, which infiltrated the anterior pararenal space, gall bladder, and right colic flexure. Gastrointestinal endoscopy showed spotty erosions and reddish mucosa, with the edematous gastric antrum and duodenal bulb narrowed at their lumens. Just beyond the supraduodenal angle at the orifice of the descending duodenum, there was a chamber with only minor mucosal changes, and it was not a duodenal ulcer. Endoscopic biopsy of the duodenum showed intramucosal eosinophilic infiltration. Treatment with prednisolone resulted in normalization of radiologic and endoscopic abnormalities.

Conclusions: We present a case of eosinophilic gastroenteritis with both mucosal and muscular involvement. CT imaging and endoscopic examination confirmed the diagnosis.

MeSH Keywords: Duodenum • Endoscopy, Gastrointestinal • Eosinophils • Gastroenteritis • Tomography, X-Ray Computed

Full-text PDF: <http://www.amjcaserep.com/abstract/index/idArt/897403>

 1355   4  10



Background

Eosinophilic gastroenteritis (EGE) is a rare disease characterized by eosinophilic infiltration of the gastrointestinal tract, and it is often associated with a history of seasonal allergy, atopy, food allergies, and asthma, which may strongly suggest the role of hypersensitivity reactions in the pathogenesis of EGE [1]. Computed tomography (CT) is increasingly being used as a screening technique for patients with symptoms of intestinal disease [2–5]. Endoscopic examination primarily shows mucosal conditions, whereas CT shows the gastrointestinal bowel wall, perienteric fat, and other adjacent organs. We report a case of EGE that was treated with corticosteroids and followed using CT imaging and endoscopy.

Case Report

A 67-year-old Asian woman was admitted with a 3-month history of recurrent episodes of abdominal pain, chronic diarrhea, and abdominal distention. The patient also had a history of hypertension. There was no fever, weight loss, or rash. She had no food, pollen, or drug allergies in her medical history. She denied any tobacco smoking, alcohol use, or illicit drug use. Physical examination revealed no remarkable findings. Blood examination showed the following: leucocytes, 9750/ μ L (neutrophils: 68%, eosinophils: 0.7%, 68.3/ μ L, lymphocytes: 25%, monocytes: 5.6%, and basophils: 0.5%), hematocrit, 34.6%, platelets, 229 000/ μ L, and immunoglobulin E, 45.3 IU/mL (normal range: 0–173 IU/mL). Liver and renal functions were within normal range. Stool culture for pathogens and analysis for ova, cysts, and parasites were negative.

Intravenous contrast-enhanced CT scans during the late arterial and early portal venous phases showed mural thickening of the gastric antrum and the first and second parts of the duodenum with the presence of radiographic water halo sign (Figure 1A, 1B). A thickened bowel wall was observed, consisting of 2 continuous, symmetrically stratified layers: a higher-attenuation inner mucosal layer, which is related to hyperemia [6], and an outer ring of the lower-attenuation representing edema, which was located in the submucosa and muscularis propria (muscular layer) of the bowel wall, respectively, representing an acute inflammatory or ischemic condition, although nonspecific [2,3]. A markedly thickened wall resulted in luminal narrowing in the gastric antrum and bulb, whereas a chamber with a rigid lateral wall was shown at the orifice of the descending part of the duodenum, resembling a giant duodenal ulcer (Figure 2A). This rigid lateral wall showed only relatively reduced thickness, without submucosal edema. The inflammation penetrating through it may have spread directly toward the perienteric fat in the anterior pararenal space. An increased attenuation of perienteric fat adjacent to the rigid

wall was seen further extending to the gall bladder and right colic flexure, indicating the inflammatory process (Figure 2B). However, inflammation did not directly infiltrate the pancreatic head (Figure 2A). Neither ascites nor contrast enhancement of the subserosa was present in the whole abdomen.

Repeated upper-gastrointestinal endoscopy showed mucosal edema in the gastric antrum and large mucosal folds in the duodenal bulb, with mucosal abnormalities such as edema, erythema, whitish specks, and erosions. Markedly thickened folds in the duodenal bulb developed stenosis with decreased peristalsis, failing to dilate easily by air insufflation during the endoscopic procedure (Figure 3A, 3B). Just beyond the supraduodenal angle at the orifice of the descending duodenum, a chamber with an apparently rigid bowel wall (and no peristalsis) partly without Kerckring folds was observed with minor mucosal abnormalities such as edema and erosions (Figure 3C). The descending duodenum following a rigid chamber seemed to present only with mucosal edema and was dilated easily by air insufflation. Multiple duodenal biopsies showed intramucosal eosinophilic infiltration (>20 per high-power field) (Figure 4). There was no evidence of any parasite or malignancy.

She had been prescribed an oral proton-pump inhibitor for 1 month, but her symptoms and endoscopic and CT abnormalities had not improved. Following this treatment, she initially received prednisolone 20 mg once daily, which was gradually and carefully tapered depending on subjective symptoms and endoscopic findings to prevent relapses over a 10-month period. After 10 months, the patient's abdominal pain completely resolved. CT of the abdomen showed complete resolution of gastro-duodenal wall thickening and perienteric inflammation.

Discussion

Symptoms of EGE are nonspecific and overlap with many other gastrointestinal diseases. Although peripheral eosinophilia is very common in all subtypes of EGE, it can be absent in up to 23% of cases, as seen in our patient with 68.3/ μ L of eosinophils, which should not be considered a diagnostic criterion [7]. Gastric or duodenal biopsies are required to confirm the diagnosis; histological examination usually shows \geq 20 eosinophils in each magnification field [7,8], as demonstrated in our case. Eosinophilic infiltrates may involve various sites down the length of the gastrointestinal tract, and they may occupy various sites through the depth of the wall [9]. The clinical manifestations of EGE vary depending on which layer of the gastrointestinal tract is involved (the mucosa, muscle, or serosa) [9]. The mucosal form of EGE presents with abdominal pain, vomiting, diarrhea, gastrointestinal bleeding, or malabsorption and also manifests as fold-thickening, reddish mucosa, and erosions, as shown in the gastric antrum and the

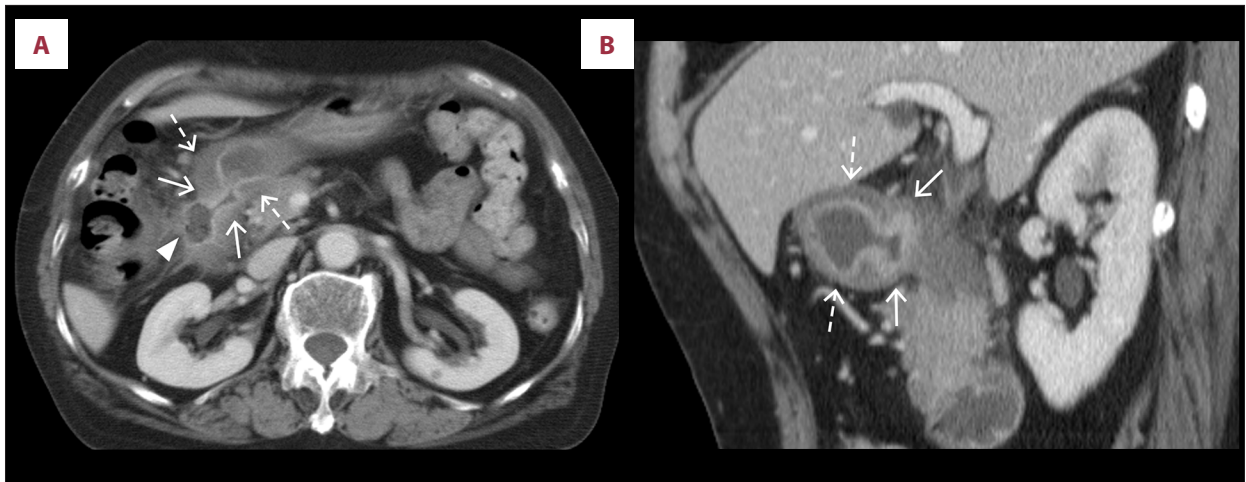


Figure 1. Axial (A) and sagittal (B) contrast-enhanced computed tomography images showing mural thickening of the gastric antrum (dashed arrow) and the duodenum (white arrow) with the radiographic water halo sign. A chamber with a rigid lateral wall (arrowhead) is shown at the orifice of the descending part of the duodenum.



Figure 2. Sagittal (A) and coronal (B) contrast-enhanced computed tomography images showing the rigid chamber (arrowhead) at the orifice of the descending part of the duodenum and soft-tissue inflammation extending to the gall bladder (arrow).

first and second parts of the duodenum in the present case. The gastric antrum and the bulb, and the rigid chamber of the duodenum, showed a sharp high-attenuation mucosal layer, representing mucosal abnormalities such as erosions on endoscopic examination, while the following descending duodenum had a thicker and fluffy mucosal layer only, with mucosal edema observed using endoscopy.

Muscular involvement results in areas of reduced distensibility, strictures, bowel wall thickening, or intestinal obstruction, in which CT imaging can help localize involved layers of the affected bowel walls. Since the stomach and duodenal bulb are wholly covered with less distensible visceral peritonea, the submucosal and muscular edema demonstrated by CT can only expand inwards, leading to narrowing of the antrum and bulb, which fail to easily dilate by air insufflation.

Furthermore, decreased peristalsis suggests muscular involvement. The rigid lateral wall in the proximal descending duodenum resulted from severe damage of the entire duodenal wall involving the muscular layer on CT imaging, and gastrointestinal endoscopy showed reduced distensibility, lacking peristalsis, with minor mucosal changes that were also indicative of muscular involvement. Anatomically, the descending duodenum, not in the intraperitoneal portion, occupies the anterior or pararenal space of the retroperitoneum. Therefore, mural thickening of the proximal descending duodenum, except of the right lateral wall, can expand outward and cause lateral displacement of its lumen, allowing a rigid chamber to form, which did not dilate by air insufflation. However, the distal descending duodenum was narrowed by mural thickening with submucosal edema, but endoscopic air insufflation easily dilated its lumen, suggesting an intact muscular layer.

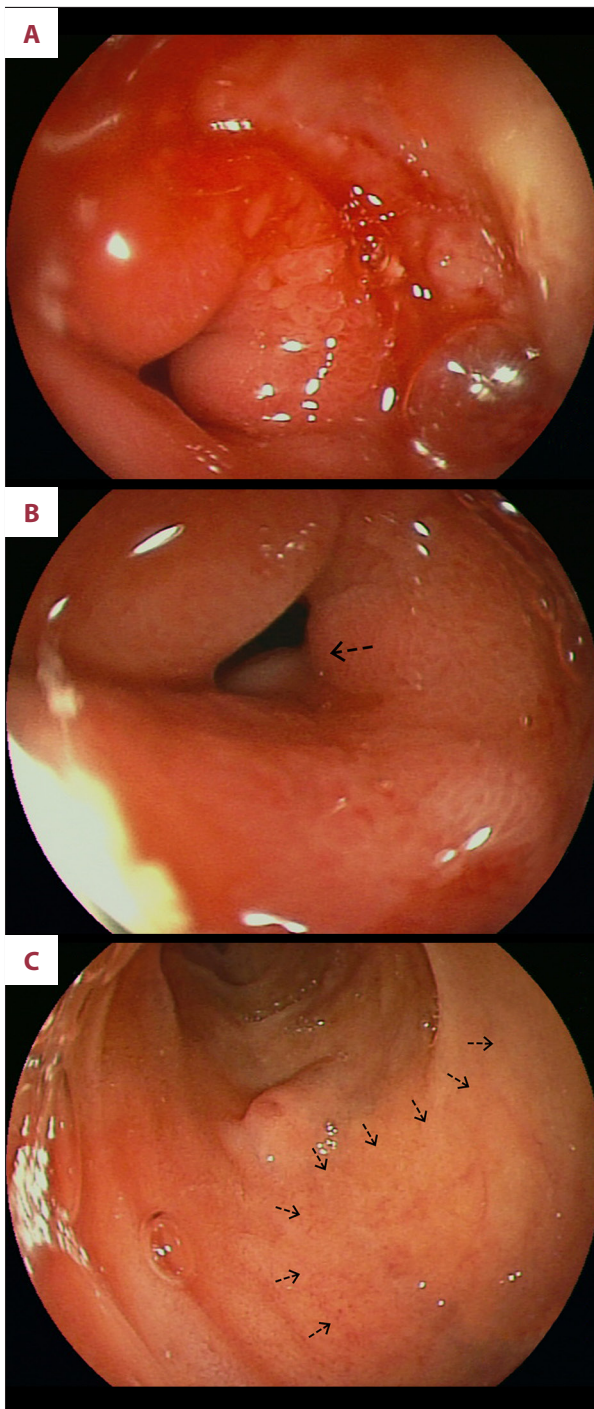


Figure 3. Upper gastrointestinal endoscopy showing mucosal edema and large mucosal folds in the duodenal bulb (A), resulting in stenosis at the supraduodenal angle (dashed arrow) (B). Just beyond the supraduodenal angle at the orifice of the descending duodenum, a chamber with a rigid bowel wall (dashed arrow) is observed (C).

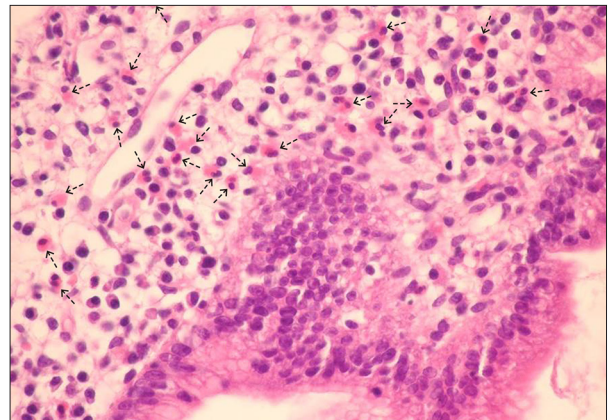


Figure 4. Photomicrograph (original magnification, 200x; hematoxylin-eosin stain) of a biopsy specimen of the duodenal mucosa obtained during endoscopy, showing eosinophilic infiltration of the lamina propria (dashed arrow), which is a finding indicative of eosinophilic gastroenteritis.

The serosal form presents with ascites and a higher eosinophil count, which seems quite distinct from the mucosal and muscular forms. The absence of ascites and lack of contrast enhancement of the subserosa negated serosal involvement in our case with a normal eosinophil count.

Because the duodenal loop occupies the anterior pararenal space of the retroperitoneum along with the pancreas and vertical colon segments, inflammatory processes affecting one of these organs often spread to affect the others. As shown in this case, CT demonstrated eosinophilic inflammation extending to the perienteric fat (existing in the anterior pararenal space) adjacent to the descending duodenum, and then it reached the gall bladder and right colic flexure. There was 1 case report of large ulcerations in the duodenal bulb induced by eosinophilic gastroenteritis caused by an enterobiliary fistula through the peritoneum [10]. Therefore, eosinophilic infiltration may spread transperitoneally to the gall bladder followed by right colic flexure, or it may extend through the duodenohepatic and duodenocolic ligaments to the gall bladder and the right colic flexure, respectively.

Conclusions

We present a case of EGE with both mucosal and muscular involvement. Since the clinical presentation may vary depending on the sites and depth of involvement of the gastrointestinal tract, both CT and endoscopic imaging can confirm the diagnosis.

Acknowledgments

We would like to thank Editage (www.editage.jp) for English language editing.

Institutional review board statement

The study was reviewed and approved by Ubekosan Central Hospital Corp. Institutional Review Board.

References:

1. Klein NC, Hargrove RL, Sleisenger MH, Jeffries GH: Eosinophilic gastroenteritis. *Medicine*, 1970; 49: 299–319
2. Macari M, Balthazar EJ: CT of bowel wall thickening: Significance and pitfalls of interpretation. *Am J Roentgenol*, 2001; 176: 1105–16
3. Wittenberg J, Harrisinghani MG, Jhaveri K et al: Algorithmic approach to CT diagnosis of the abnormal bowel wall. *Radiographics*, 2002; 22: 1093–109
4. Isik A, Firat D, Peker K et al: A case report of esophageal perforation: Complication of nasogastric tube placement. *Am J Case Rep*, 2014; 15: 168–71
5. Isik A, Deniz Firat Y, Peker K et al: How could such a wide piece of tree root pass through the narrow pyloric orifice? An extremely rare case. *Am J Case Rep*, 2014; 15: 284–87
6. Balthazar EJ: CT of the gastrointestinal tract; principles and interpretation. *Am J Roentgenol*, 1991; 156: 23–32
7. Talley NJ, Shorter RG, Phillips SF, Zinsmeister AR. Eosinophilic gastroenteritis: a clinicopathological study of patients with disease of the mucosae, muscle layer, and subserosal tissues. *Gut*, 1990; 31: 54–58
8. Baig MA, Qadir A, Rasheed J: A review of eosinophilic gastroenteritis. *J Natl Med Assoc*, 2006; 98: 1616–19
9. Khan S: Eosinophilic gastroenteritis. *Best Pract Res Clin Gastroenterol*, 2005; 19: 177–98
10. Kim HM, Woo JY: Enterobiliary fistula as a complication of eosinophilic gastroenteritis: A case report. *Korean J Radiol*, 2008; 9: 275–78

Informed consent statement

The involved person gave her written informed consent prior to study inclusion.

Conflict of interest statement

There is no conflict of interest with regard to this manuscript.