**Eosinophilic gastroenteritis forming rigid chamber mimics giant duodenal ulcer on CT imaging**

**Abstract**

We report a case of eosinophilic gastroenteritis forming a chamber of the rigid duodenal wall in a 67-year-old woman. Abdominal CT revealed symmetrical wall thickening of the gastric antrum and duodenal bulb, whose bowel walls consisted of two continuous, symmetrically stratified layers. There seemed a chamber mimicking a giant ulcer at the orifice of the descending duodenum. Eosinophilic inflammation was penetrated through this rigid wall in the descending duodenum, accompanied by perienteric inflammation infiltrating the anterior pararenal space, gall bladder and right colic flexure. Gastrointestinal endoscopy showed spotty erosions and reddish mucosa with the gastric antrum and duodenal bulb edematously narrowed in their lumen. Just beyond the supraduodenal angle at the orifice of the descending duodenum, there was not a duodenal ulcer but a chamber. Endoscopic biopsy of the duodenum showed intramucosal eosinophilic infiltration. Treatment with prednisolone resulted in normalization of radiologic and endoscopic abnormalities.

**Key words:** Eosinophilic gastroenteritis; CT; Duodenum; Endoscopy

**Core tip:** Computed tomography (CT) is valuable for detection and characterization of gastrointestinal wall abnormalities. We present a case of eosinophilic gastroenteritis with both mucosal and muscular involvements. CT imagings as well as endoscopic examination supported the diagnosis.

**INTRODUCTION**

Eosinophilic gastroenteritis (EGE) is a rare disease characterized by eosinophilic infiltration of the gastrointestinal tract and is often associated with a history of seasonal allergy, atopy, food allergies and asthma that 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, 3]. Endoscopic examination primarily reveals mucosal conditions, whereas CT demonstrates assessments of gastrointestinal bowel wall, perienteral fat and adjacent other organs. We report a case of EGE that was treated with corticosteroids and followed up using CT imagings and endoscopy.

**CASE REPORT**

A 67-year-old woman was admitted with 3-month recurrent episodes of abdominal pain, chronic diarrhea and abdominal distention. The patient had a history of hypertension. There was no fever, weight loss, or rash. She had no food, pollen, or drug allergy in her medical history. There was no remarkable feature in her physical examination. Leucocyte 9750 / μl (neutrophil: 68 %, eosinophil: 0.7 % ; 68.3 / μl, lymphocyte: 25 %, monocyte: 5.6 %, basophil: 0.5 %), hematocrit: 34.6 %, platelet: 229000 /μl, immunoglobulin E: 45.3 IU / ml, (normal range: 0 – 173 IU / ml) was counted in her blood examination. Liver and renal functions were in normal range. Stool culture for pathogens and analysis for ova, cysts and parasites were negative.

Intravenous contrast-enhanced CT scan during the late arterial and early portal venous phases revealed mural thickening of the gastric antrum and the first and second parts of the duodenum with the radiographic water halo sign there (Figure 1A, 1B). A thickened bowel wall consisted of two continuous, symmetrically stratified layers; a higher-attenuation inner mucosal layer which is related to hyperemia [4] and an outer ring of the lower-attenuation representing edema located in the submucosa and the 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 the bulb, whereas a chamber with the rigid lateral wall was demonstrated at the orifice of the descending part of the duodenum, resembling a giant duodenal ulcer (Figure 2A). This rigid lateral wall only showed a relatively reduced thickness without submucosal edema. Penetrating through it, inflammation might spread directly toward 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 an inflammatory process (Figure 2B). On the other hand, 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 (with no peristalsis) partly without Kerckring folds was observed with minor mucosal abnormalities like edema and erosions (Figure 3C). The descending duodenum following a rigid chamber seemed to only present with mucosal edema and 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 one month, but her symptoms or endoscopic and CT abnormalities had not been improved. Following this treatment she received prednisolone 20 mg once a day with gradual tapering over a 10-month period. After the elapse of ten 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 as high as 23 % of cases as seen in our case with 68.3 / μl eosinophil, which should not be considered a diagnostic criterion [5]. Gastric or duodenal biopsies are required for confirming diagnosis, where more than 20 eosinophils were determined in each magnification field histologically [5,6] as demonstrated in our case. The eosinophilic infiltrates not only may involve various sites down the length of the gastrointestinal tract, but also may occupy various sites through the depth of the wall [7]. The clinical manifestations of EGE vary depending on which layer of the gastrointestinal tract is involved (i.e., mucosa, muscle, or serosa) [7]. 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 first and second parts of the duodenum in this case. The gastric antrum and the bulb and the rigid chamber of the duodenum showed a sharp high-attenuation mucosal layer, representing mucosal abnormalities like erosions on the endoscopic examination, while the following descending duodenum had a thicker and fluffy mucosal layer only with mucosal edema seen by endoscopy.

Muscular involvement results in areas of reduced distensibility, strictures, bowel wall thickening, or intestinal obstruction, where the CT imagings can help in localizing involved layers of affected bowel walls. Since the stomach and the duodenal bulb are wholly covered with less-distensible visceral peritonea, submucosal and muscular edema demonstrated by CT could only expand inwardly lead to the narrowing antrum and bulb, failing to easily dilate by air insufflation. Furthermore, decreased peristalsis suggested the 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 the CT imagings and gastrointestinal endoscopy demonstrated reduced distensibility lacking of peristalsis with minor mucosal changes, also indicative of muscular involvement. Anatomically, the descending duodenum, not in the intraperitoneal portion, occupies the anterior pararenal space of the retroperitoneum. Therefore, mural thickening of the proximal descending duodenum except the right lateral wall could expand outwardly and caused lateral displacement of its lumen, permitted to form a rigid chamber reluctantly dilated by air insufflation. On the other hand, the distal descending duodenum was made narrow by mural thickening with submucosal edema but endoscopic air insufflation easily dilated its lumen, suggesting intact muscular layer.

The serosal form presented with ascites and a higher eosinophil count seems quite distinct from the mucosal and muscular forms. The absence of ascites and lacking contrast enhancement of the subserosa denied the 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 perienteric fat (existing in the anterior pararenal space) adjacent to the descending duodenum, then reaching the gall bladder and the right colic flexure. There was one case report that large ulcerations in the duodenal bulb induced by eosinophilic gastroenteritis caused enterobiliary fistula through the peritoneum [8]. Therefore, eosinophilic infiltration may spread transperitoneally to the gall bladder followed by the right colic flexure or may extend through the duodenohepatic and duodenocolic ligaments to the gall bladder and the right colic flexure, respectively.

**CONCLUSION**

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

**FIGURE LEGENDS**

**Figure 1** Axial (A) and sagittal (B) contrast-enhanced CT images show mural thickening of the gastric antrum (arrow) and the duodenum (arrowhead) with the radiographic water halo sign.

**Figure 2** Sagital (A) and coronal (B) contrast-enhanced CT images show the rigid chamber at the orifice of the descending part of the duodenum and soft-tissue inflammation extending to gall bladder.

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

**Figure 4** Photomicrograph (original magnification, 200 x; Hematoxyllin-Eosin stain) of a biopsy specimen of the duodenal mucosa obtained during endoscopy shows eosinophilic infiltration of the lamina propria (arrowheads), a finding indicative of eosinophilic gastroenteritis.