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Pyloric Stenosis: A Rare Manifestation of Crohn's Disease

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Abstract

We are presenting a rare case of pyloric stenosis due to Crohn's disease. A 53-year-old woman with prior history of colonic Crohn's disease was admitted to the hospital with gastric outlet obstruction. Esophagogastroduodenoscopy demonstrated pyloric stenosis and biopsy was consistent with Crohn's disease. She was treated with corticosteroids and her condition improved.

Keywords: Crohn's disease, Gastroduodenal Crohn's disease, Upper gastrointestinal Crohn's disease, Pyloric stenosis, Gastric outlet obstruction

1. Introduction

Crohn's disease (CD) is a chronic, idiopathic, inflammatory disease with segmental and transmural involvement of the gastrointestinal tract.¹ The first documented case involving the stomach was published in 1949.² Crohn's gastritis is commonly associated with Crohn's duodenitis and is referred to as "gastroduodenal CD" and is reported in 0.5 -4.0% of cases of Crohn's disease.³⁻⁷ 56% of patients with gastroduodenal CD had previous CD elsewhere in the gastrointestinal tract⁵ and one third will develop distal disease later in life.^{7,8} Isolated gastroduodenal CD is extremely rare and accounts for 0.07% of patients with CD.⁶ We are reporting a rare case of pyloric stenosis secondary to upper gastrointestinal Crohn's disease in a patient with known colonic Crohn's disease.

2. Case report

A 53-year-old woman with prior history of colonic Crohn's disease, presented to the hospital for

evaluation of worsening epigastric pain, nausea and intermittent non-bloody emesis for 5 weeks. She was diagnosed with colonic Crohn's disease at another facility and was taking prednisone 10 mg daily. On presentation, patient was tachycardic, but was otherwise hemodynamically stable. She had mild tenderness in the left upper quadrant with hypoactive bowel sounds and the rest of the examination was unremarkable.

Initial blood count, basic metabolic panel and hepatic function panel demonstrated: white blood cells 22,200/ μ L, hemoglobin 8.6 g/dL, hematocrit 33.1, platelet 363,000/ μ L, sodium 129 mEq/L, potassium 4.8 mEq/L, chloride 93 mEq/L, bicarbonate 22 mEq/L, glucose 130 mg/dL, blood urea nitrogen 10 mg/dL, creatinine 0.7 mg/dL, alanine aminotransferase 25 IU/L, aspartate aminotransferase 47 IU/L, alkaline phosphatase 65 IU/L, total bilirubin 0.5 mg/dL, total protein 5.4 mg/dL and albumin 2.9 mg/dL. Due to the presentation of epigastric abdominal pain, lipase was checked to exclude acute pancreatitis and was normal (44 U/L).

Abbreviations: CT, computed tomography; EGD, esophagogastroduodenoscopy; CD, Crohn's disease; UGI-CD, upper gastrointestinal CD; MRI, magnetic resonance imaging.

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Computed Tomography (CT) imaging of the abdomen and pelvis with oral and intravenous contrast was performed and it demonstrated a markedly distended stomach and thickened gastric walls; gallbladder, pancreas and biliary tree were unremarkable (Fig. 1).

Given the clinical presentation of vomiting and finding of gastric wall thickening in the CT images, patient underwent esophagogastroduodenoscopy (EGD) and had food in the proximal stomach limiting visualization with diffuse friable, ulcerated mucosa in the distal body and antrum with pyloric narrowing which could not be traversed with pediatric gastroscope. Biopsies from the ulcerated gastric mucosa demonstrated granulation tissue with transmural chronic inflammation (Fig. 2) consistent with Crohn's disease. Colonoscopy was performed and she had severe, edematous ulcerated mucosa in the distal rectum, with sparing of the rest of the colon and terminal ileum. Biopsies from the rectum were consistent with known Crohn's disease.

Based on the clinical presentation, endoscopy and biopsy findings, we arrived at the diagnosis of

pyloric stenosis secondary to Crohn's disease. The different treatments available were discussed with the patient and she opted for medical management. She was treated with intravenous corticosteroid. Her condition steadily improved and she was able to tolerate oral feeds at the time of discharge. She was advised to follow up at the gastroenterology team for continued follow up and transition to steroid-sparing therapy.

3. Discussion

Crohn's gastritis is commonly seen during the third and fourth decades of life and is the most common site of upper gastrointestinal CD.^{3,8} Gastroduodenal CD is seen equally in both genders with male:female ratio of 1.2:1.^{3,8} Gastroduodenal CD is commonly asymptomatic.^{4,6–10} Many of the symptoms mimic peptic ulcer disease and the most common symptom is epigastric abdominal pain.^{4,11,12} Continued epigastric pain with weight loss is often a sign of gastroduodenal stricture from chronic inflammation and warrants prompt evaluation.^{4,6,8}

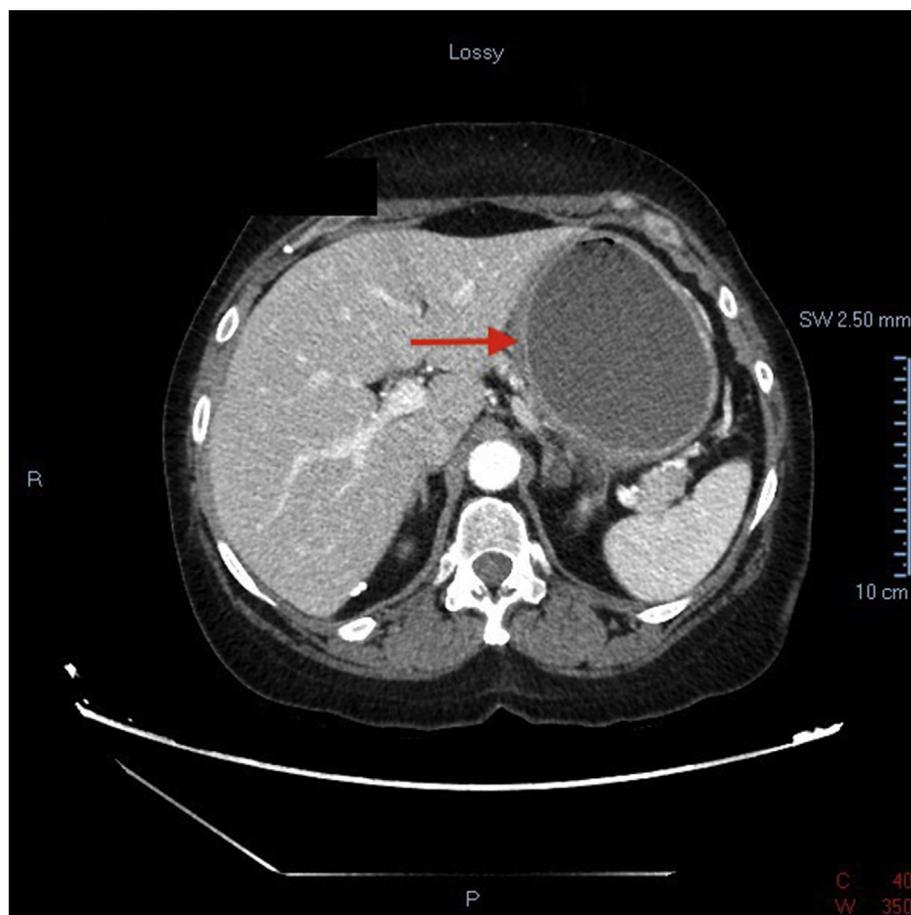


Fig. 1. CT scan: markedly distended stomach and thickened gastric wall.

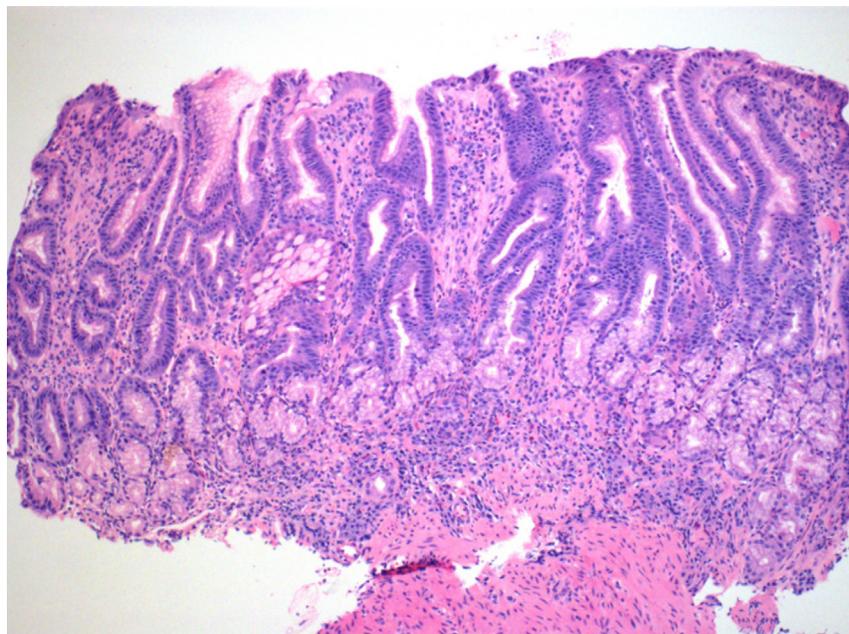


Fig. 2. Pyloric biopsy: transmural inflammation.

A classic radiographic finding is funnel shaped deformity of the antrum and duodenal bulb known as “Ram’s horn”.¹³ Findings detected during double-contrast imaging are aphthous ulcerations, thickened rugal folds, cobblestoning, pseudodiverticulum and strictures, resulting in tubularization of antrum, pylorus and duodenum.^{8,14} This resembles a gastroduodenostomy for ulcer disease and has the “pseudo-Billroth I appearance”.^{3,15} Magnetic resonance imaging (MRI) can provide some advantage in the differentiation of fibrotic strictures and acute inflammation.^{16–19} In addition to CT and MRI, enterography is a useful adjunct to diagnose and localize areas of stricturing disease.²⁰

Most common endoscopic findings are superficial, mild and diffuse inflammation.^{3,9} Bamboo joint-like appearance of the stomach is characterized by longitudinal inflamed folds with perpendicular erosions and linear furrows is pathognomonic for CD.²¹ Pathological findings include neutrophilic epithelial clusters termed as “focally enhanced gastritis”, aphthoid ulcers and noncaseating granulomas.³

Diagnostic criteria for diagnosis of UGI-CD has been established by Nugent and Roy, which consisted of one of the following two criteria: (1) non-caseating granulomatous inflammation with or without coexisting CD at other gastrointestinal tracts sites, and without an alternative systemic granulomatous disorder, or (2) clearly documented CD as another gastrointestinal site and radiologic and/or endoscopic evidence of diffuse inflammatory changes suggestive of CD.^{6,10}

Gastroduodenal CD is initially managed medically, paralleling the treatment of CD elsewhere in the GI tract. Despite scarcity of data, UGI-CD patients are treated with thiopurines, methotrexate, steroids and anti-tumor necrosis factor agents such as infliximab and adalimumab.^{1,22,23} Gastroduodenal strictures are associated with inadequate response to medical management; endoscopic balloon dilation can provide durable relief in these cases. In patients with obstruction, nasojejunal administered enteral nutrition has been proven to be effective.²⁴ The most common indications for operative interventions are gastric outlet obstruction (83%), refractory pain (11%) and bleeding (5%).⁸ Many surgical procedures such as antrectomy, vagotomy, Billroth reconstruction, gastroenterostomy, strictureplasty and laparoscopic approaches have been described for UGI-CD.^{8,25,26}

4. Conclusion

Our patient had a known history of CD with colonic manifestation and had new pyloric stenosis, as a gastroduodenal manifestation of CD which is reported in only 0.5–4.0% of patients with Crohn’s disease.^{5–7} Endoscopy and histopathological findings enabled us to arrive at the correct diagnosis and exclusion of other etiologies for the patient’s symptoms. Clinicians taking care of patients with Crohn’s disease should have a high index of suspicion and plan appropriate investigations if they develop

features of gastric outlet obstruction to exclude upper gastrointestinal manifestations of CD.

Author contributions

R.R. drafted and revised the manuscript for critically important intellectual content. E.T. drafted the manuscript. T.G. and S.P. edited the manuscript. V. G. reviewed the manuscript for important intellectual content. All authors approved the final version of the paper.

Informed consent

Informed consent was obtained from the patient for the publication of this article with anonymized information.

Author disclosures

This case was presented as a poster at the American College of Gastroenterology Conference, San Antonio, 2019.

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Conflict of interest

The authors report no conflict of interest.

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