Gastritis profunda cystica presenting as gastric outlet obstruction and mimicking cancer: A case report

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ABSTRACT

Gastritis cystica profunda (GCP) is a rare, benign lesion of the stomach characterized by polypoid hyperplasia and/or ulcerated mucosal lesion and cystic dilatation of the gastric glands extending into the submucosa or muscularis propria of the stomach. Its etiology and pathogenesis are still incompletely understood. The most important factor is assumed to be a history of prior gastric surgery. We herein present a case of a young adult female with upper gastrointestinal (GI) symptoms. She underwent upper GI endoscopy twice, which revealed pyloric narrowing and intramural mass. Gastric endoscopic mucosal biopsies were performed, but no tumor was identified and her symptoms persisted. Imaging studies also revealed a mass lesion. Open laparotomy and partial gastrectomy with histopathology of the resected specimen revealed the true nature of the lesion. Surgery also improved her symptoms. GCP should be kept in the differential diagnosis of gastric mural mass lesions.

Key words: Endoscopy, herniation, histopathology, partial gastrectomy, pyloric mass

INTRODUCTION

Gastritis cystica profunda (GCP) is a rare, benign lesion characterized by polypoid hyperplasia and/or ulcerated mucosal lesion and cystic dilatation of the gastric glands extending into the submucosa of the stomach.[1] It has been suggested that GCP represents a manifestation of hyperplastic and metaplastic responses to mucosal injury caused by several factors, such as chronic inflammation, ischemia, gastric surgery and the presence of foreign materials.[2,3] The most important factor is assumed to be a history of prior gastric surgery.

There is no consensus on the optimal management of these lesions. Surgical interventions have ranged from simple submucosal excisions to total gastrectomies. As it is seen so rarely, we present this case to raise the awareness of differential diagnoses of stomach wall masses. Our case was managed with partial gastrectomy with good relief of symptoms.

CASE REPORT

A 38-year-old female presented with complaints of epigastric pain for 3 months along with abdominal fullness, nausea, vomiting and weight loss for the last 2 months. Initially, she was not able to tolerate solids, but, with the passage of time, she became intolerant to liquids also. She had a past history of pulmonary tuberculosis diagnosed on the basis of the symptoms and the sputum acid fast bacilli (AFB) smear positivity. There was no past surgical or blood transfusional history.

The patient underwent an upper gastrointestinal endoscopy that revealed narrowed pyloric opening with severe erythema and edema beyond which the GIF 150 scope could not be negotiated. Multiple biopsies were taken from the lesion that showed moderate active inflammation and mild *Helicobacter pylori* infection.

She was prescribed first-line therapy for *H. pylori* eradication, but the symptoms

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Access this article online

Website: www.intern-med.com

DOI: 10.4103/2224-4018.154296

Quick Response Code:

of abdominal fullness did not resolve. A computed tomography of the abdomen was performed that showed eccentric soft tissue thickening identified along the distal aspect of the stomach as well as along the pyloric canal, which was causing luminal narrowing and dilation of the proximal stomach. Adjacent fat stranding and few enhancing adjacent lymph nodes were noticed.

Because of the persistent symptoms, she underwent a second upper gastrointestinal endoscopy that showed similar findings of the mass in the pylorus with no reduction in size as compared with the previous examination. This time, the narrowed segment of the pylorus was dilated with a 12 mm CRE balloon. Subsequently, the pyloric opening was crossed successfully showing edematous bulb and normal distal duodenal folds. A repeat biopsy of the lesion showed no *H. pylori* organisms.

Because of the persistent symptoms, she underwent open laparotomy that showed bulky pylorus extending up to the first part of the duodenum. No extramural evidence of mass was noticed. A distal gastrectomy with controlled duodenal fistula and gastrojejunostomy was performed. The histopathology of the resected mass lesion showed irregular proliferation of a few mucinous cystically dialted glands lined by bland epithelial cells with abundant cytoplasm and basally located nuclei, intermixed with hypertrophied smooth muscle fibers reaching up to the deep muscle layer in the wall [Figure 1a-d]. Features were consistent with GPC. No dysplasia was seen. No *H. pylori* were detected. Foci of goblet cell metaplasia were noted. She made an uneventful recovery and, on follow-up, her symptoms improved markedly.

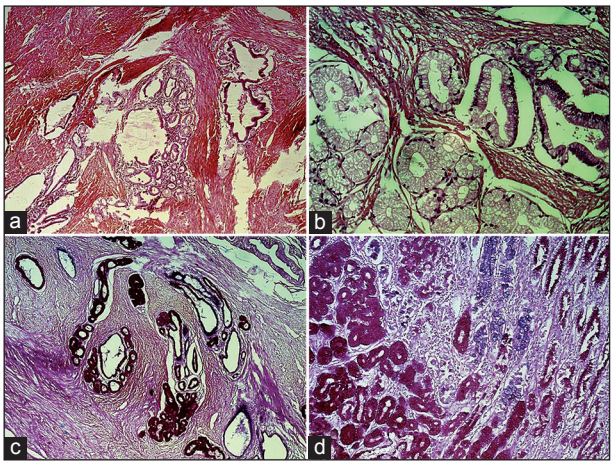

Figure 1: (a) Low-power view showing cystically dilated glands deep in the muscularis propria (hematoxylin and eosin, ×50). (b) High-power view showing benign, mucinous glands in the muscle layer (hematoxylin and eosin, ×400). (c) Low-power view showing cystically dilated mucin-positive glands deep in the muscularis propria (Periodic acid-Schiff [PAS], ×50). (d) Medium-power view of the mucosal glands showing foci of goblet cell metaplasia (Alcian Blue — PAS, ×200)

DISCUSSION

In the case reported, we have demonstrated the difficulty in accurately diagnosing a lesion with endoscopic and radiological appearances of an early gastric cancer but which is histologically benign. A number of gastroscopies were needed to be confident of an accurate diagnosis.

GCP remains a rare diagnosis and, apart from a few reported cases in intact stomachs,[4-7] seems confined to those with any form of gastro-enterostomy.[8,9] This leads to the suggestion that it is secondary to chronic mucosal irritation from reflux of small bowel contents and subsequent herniaton of the mucosal glands into the deeper layers of the stomach wall.[8-10] Because GCP has been identified alongside early gastric cancer, it has been suggested to be a precancerous lesion, but remains difficult to prove.[5,9] In a pathological study of 10,728 patients with gastric cancer, it was found in 161 patients.[10] There are some reports of GCP coexisting with Ménétrier disease[11,12] or gastric inverted hyperplastic polyps.[13]

GCP describes gastric glands covered with normal gastric mucosa that exist under the submucosal layer and form cystic expansion. The pathogenesis relates to the degradation of the integrity of the muscularis mucosa layer and the emigration of epithelial cells to the submucosa. The etiology is not clear but the most important factor appears to be the previous gastric surgery. Chronic inflammation, ischemia and a reaction to suture materials are considered to be other predisposing factors. Our patient did not have previous stomach surgery or a complaint of ulcers.

The signs and symptoms of the disease are nonspecific. It may present as abdominal pain, nausea, vomiting, bloating, acid reflux and/or bleeding.[14] Our patient had abdominal pain and vomiting and had signs of gastric outlet obstruction as evident by narrowed pyloric opening on upper GI endoscopy. To the best of our knowledge, this is the first case report of a large GCP causing gastric outlet obstruction in a previously unoperated stomach. It shows that a multicystic mass in the gastric submucosa may be GCP even in an unoperated stomach, and the diagnosis should be confirmed by histological examination because preoperative diagnosis of GCP is fraught with difficulty.

Our case had *H. pylori* infection. It is well known that *H. pylori* is the recognized precursor of gastric carcinoma, but whether *H. pylori* is the risk factor of GCP remains to be explored. Further studies are needed in this regard. The natural history of GCP is unclear and needs further exploration.

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How to cite this article: Butt MO, Luck NH, Hassan SM, Abbas Z, Mubarak M. Gastritis profunda cystica presenting as gastric outlet obstruction and mimicking cancer: A case report. J Transl Intern Med 2015;3:35-7.
Source of Support: NIL, **Conflict of Interest:** None declared.

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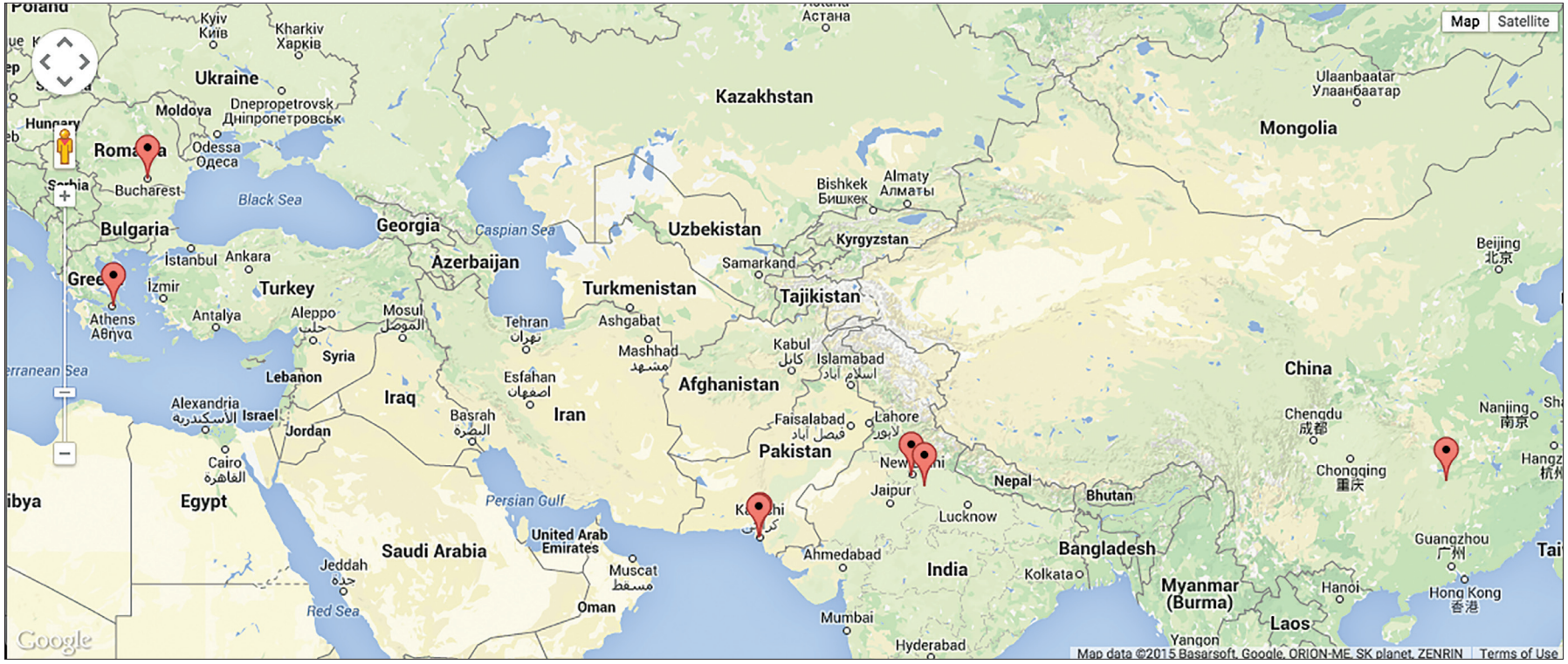

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