CROHN'S DISEASE PRESENTING AS GASTRIC OUTLET OBSTRUCTION

A CASE REPORT

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Significance: Gastroduodenal Crohn's disease is extremely rare with an incidence of 0.5-4% of all patients with Crohn's (1). Its occurrence in Filipino patients has not been reported. Due to its rarity and nonspecific clinical presentation, its timely diagnosis and proper management are often delayed and a closer look into this clinical entity is needed.

Clinical presentation: This is a case of a 38- year old Filipino female with persistent bloating and post prandial vomiting consistent with gastric outlet obstruction. Her CT scan showed a markedly distended stomach and irregular wall thickening at the pyloric area and proximal duodenum. Upper GI endoscopy confirmed this radiologic finding, the pyloric area was noted to be edematous and nodular and aphthous ulcers were present; biopsy revealed chronic and active inflammation. Tests for *Helicobacter pylori* and *Mycobacterium tuberculosis* were negative. The young age of the patient led us to think of a possible inflammatory bowel disease and Anti-*Saccharomyces cerevisiae* antibodies (ASCA) was requested which turned out to be positive.

Management: Steroids and proton pump inhibitors, the cornerstone of treatment for gastroduodenal Crohn's, was given to the patient for three months. Significant clinical improvement was noted and after one month of treatment, she was already asymptomatic. After this, overlap treatment with Azathioprine was prescribed.

Recommendations: In patients with clinically suspected Crohn's disase, ASCA is a helpful tool to confirm the diagnosis prior to treatment. Steroids and proton pump inhibitors are recommended for active gastroduodenal Crohn's and maintenance with azathioprine is opted to reduce flares.

KEYWORDS: case report, crohn's disease, gastric outlet obstruction, ASCA

CASE REPORT

CLINICAL PRESENTATION

Patient is FP, a 38/F non-hypertensive, non-diabetic female government employee who six months prior to consult with our institution, presented with bloating and frequent belching. She sought consult and an upper abdominal ultrasound was requested which showed unremarkable results. She was treated as a case of dyspepsia and was advised diet restriction and omeprazole 40mg/tab once a day. However, her symptoms persisted and were eventually associated with multiple episodes of post prandial vomiting. This prompted consult with a gastroenterologist who performed an upper GI endoscopy revealing gastric outlet obstruction. A CT scan of the abdomen was likewise requested which revealed pyloric and duodenal wall thickening. Vomiting persisted and patient had a weight loss from 42 kilograms to 30 kilograms. There was no reported hematochezia and melena, nor fever or jaundice. Her symptoms worsened and patient decided to seek consult in our institution.

For her past medical history was only positive for postpartum depression in 2002. Her family history was negative for any malignancy. She is a non smoker and a non alcoholic beverage drinker.

The initial physical exam showed a cachectic patient with a BMI of 11.6 kg/m^2. Her abdomen was flat, the bowel sounds are hypoactive, and it was soft and non-tender. Digital rectal exam revealed a tight sphincteric tone with no mass palpate. The rest of the physical exam was unremarkable.

Initial work up was done which included a CT scan of the whole abdomen (Figure 1) which showed a markedly distended stomach from T10 to the pelvis with retained fluid and food particles with irregular wall thickening and enhancement in the pylorus and proximal duodenum.

Upper GI endoscopy was performed which confirmed the CT scan findings of a markedly distended stomach with significant amount of food particles and fluid. The irregular wall thickening

at the pyloric area seen in the CT scan was revealed to be an edematous and nodular prepyloric area with aphtous ulcers, likewise the antrum was deformed (Figure 2A and B). Biopsy was done which showed gastric antral type fragments with moderate chronic and active inflammation and intestinal metaplasia (Figure 2C).

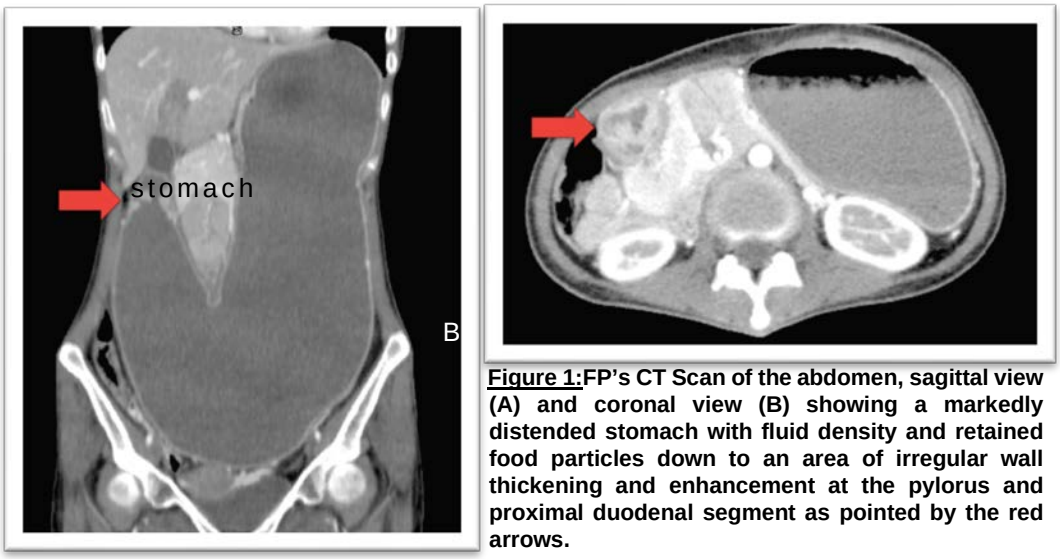

Figure 1:FP's CT Scan of the abdomen, sagittal view (A) and coronal view (B) showing a markedly distended stomach with fluid density and retained food particles down to an area of irregular wall thickening and enhancement at the pylorus and proximal duodenal segment as pointed by the red arrows.

The specimen was also tested for the presence of *Helicobacter pylori* (rapid urease test

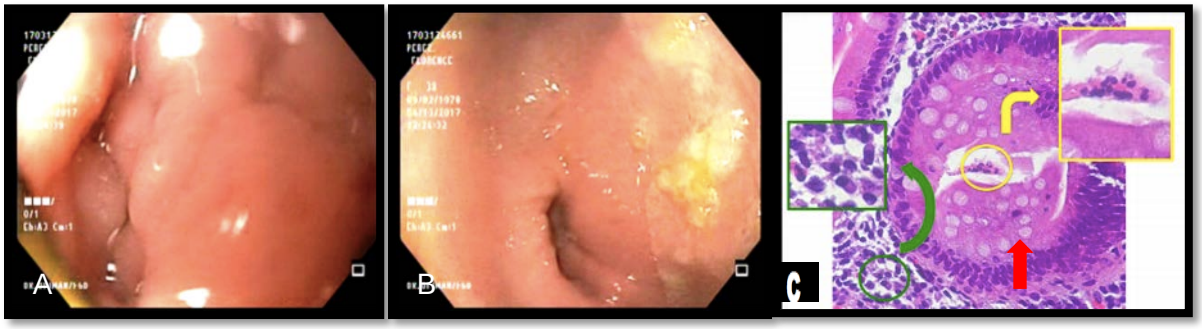

Figure 2: A: Edematous and nodular pre-pyloric area. B. Deformed antrum. C. Biopsy of the pre pyloric area showing Goblet cells (red arrow), not normally found in stomach, which connotes intestinal metaplasia, crypt with luminal neutrophils (yellow) connotes moderate active inflammation and lamina propria with plasma cells (green) which are signs of chronic inflammation.

and histopathology) which was negative. *Mycobacterium tuberculosis* was also considered and the pyloric ring mucosa and duodenal mucosa were tested for MTB RT-PCR which turned out negative.

After endoscopy and confirmation of gastric outlet obstruction, the patient underwent jejunostomy insertion for feeding.

Due to benign findings in the histopathology, she was conservatively treated with acid suppressive therapy with omeprazole 40mg/tab once a day for one month. Repeat gastroscopy showed significantly less distended abdomen but there was persistence of thickened and granular mucosa at the pylorus. A biopsy was done and again revealed active inflammation, focal surface erosion, and reactive epithelial change with no signs of malignancy.

The new endoscopic findings were compared with previous and there was no progression of the lesion. Furthermore, biopsy again revealed benign findings. This led us to think of a possible inflammatory cause, i.e. Crohn's disease which can explain the symptoms of the patient. Anti-*Saccharomyces cerevisiae* antibodies (Crohn's panel) was requested which showed positive results.

TEST	RESULT	CUT OFF	INTERPRETATION
Saccharomyces cerevisiae IgG	87.2 U/mL	10	POSITIVE
Saccharomyce cerevisiae IgM	4.5 U/mL	10	NEGATIVE

Patient was given acid suppressive therapy with omeprazole 40mg/tab once a day and prednisone 30mg/tab/ day (at 0.75/kg/day) for one month and repeat gastroscopy after two weeks showed less edematous and less granular peri-pyloric area now with a more visible opening into the duodenal bulb and the neonatal scope was able to pass through the bulb area and noted that the mucosa at the D2 junction and the rest of the duodenum is normal. Patient's diet was progressed to liquids.

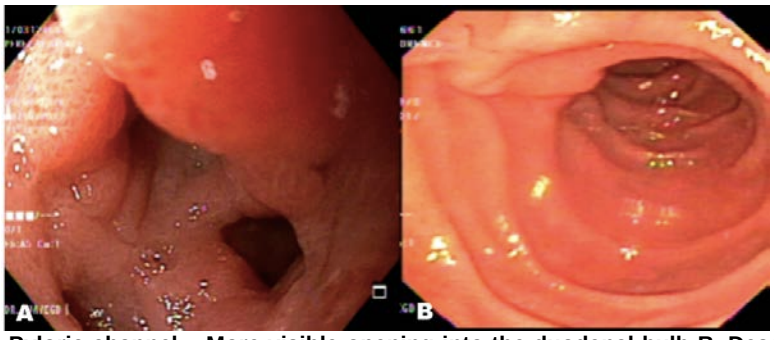

Figure 4: A. Pyloric channel – More visible opening into the duodenal bulb B. Descending part of the duodenum – It was entered endoscopically for the first time using a neonatal scope, and was noted to be normal

One month after starting the omeprazole and prednisone, an upper GI series was done which showed no delay in the transit of contrast through the upper GI tract.

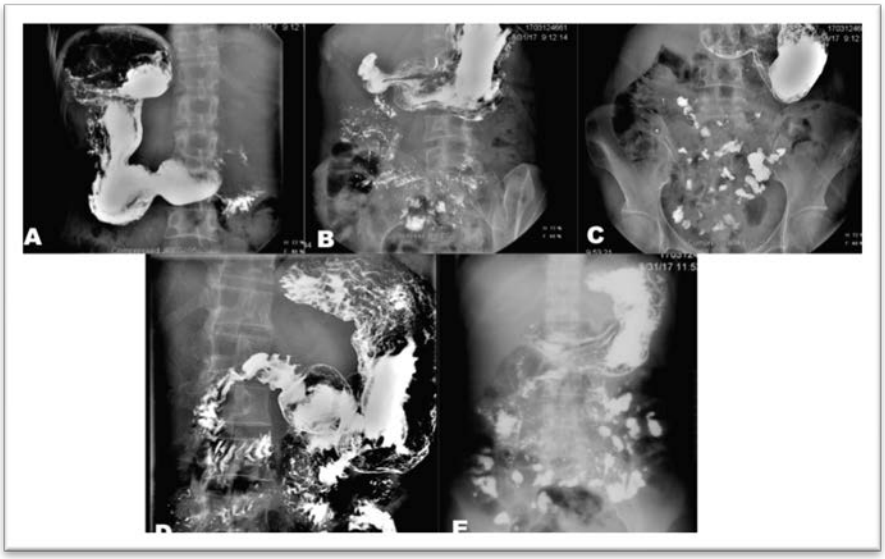

Figure 5. Upper GI Series A. At 10 minutes B. At 20 minutes C. At 40 minutes D. At 90minutes E. At 2.5 hours.

Diet was progressively resumed and patient already able to tolerate regular diet. Prednisone was tapered gradually to 20mg/tab for 2 weeks then 10mg/tab for 2 more weeks and maintained on prednisone 5mg/tab once a day for one month overlap with Azathioprine 50mg/tab, 1 ½ tab once a day. Patient's symptoms completely resolved.

DISCUSSION:

Crohn's disease (CD) can affect any region of the gastrointestinal tract from mouth to the anus. Gastroduodenal Crohn's disease is rare which only occurs in around 0.5% to 4% of all patients with Crohn's disease and isolated disease is extremely rare with an incidence of 0.07% (1,5). Around one third of patients with proximal Crohn's disease do not have evidence of distal Crohn's disease at the time of diagnosis, but virtually all develop distal disease in time. The male to female predilection is 1.2:1. Patient's with this type of Crohn's disease tend to be younger at the time of diagnosis and most commonly presents with abdominal pain, most commonly dyspepsia or epigastric pain as the main symptoms and is associated with malaise. In some patients, gastric outflow is compromised and they present with obstructive symptoms such as early satiety, nausea, vomiting, and weight loss as seen in our patient (4).

The diagnosis of gastroduodenal Crohn's is difficult due to its rarity, and increased clinical suspicion is the key to proper diagnostics. Symptoms, radiographic, endoscopic and histologic characteristis are non specific further making the diagnosis difficult. Recently serologic tests are found to be helpful in confirming the diagnosis.

According to the study of Kefalas et. al. endoscopic findings in patients with gastroduodenal Crohn's include patchy erythema, mucosal friability, thickened folds, and ulcerations, both aphthous and linear instead of the usual circular ulcers as seen in peptic ulcer disease. Another common feature is a nodular or cobblestoning mucosa (1). In our patient, findings in the pyloric and pre pyloric area include edematous and nodular mucosa with aphthous ulceration with no signs of bleeding, these resulted to luminal narrowing in the gastric outlet.

As mentioned, the biopsy findings in gastroduodenal Crohn's are often nonspecific, with the most common finding include granulomas without caseation (noted in 5% to 83% of cases) and chronic inflammation. The differential diagnosis of granulomatous gastritis are *H. pylori* infection, gastric sarcoidosis, tuberculosis and syphilis. In our patient, these causes of granulomatous gastritis were already excluded based on tests. In all the biopsies done in the patient, chronic inflammatory changes and no signs of granuloma formation or malignancy were

noted.

Due to the non specificity of radiologic, endocopic and histologic findings in Crohn's disease especially in the gastroduodenal type, serological testing currently emerges to have a vital role in its diagnosis. Recent studies have suggested that anti-*Saccharomyces cervisiae* antibody is a useful tool in which the diagnosis of Crohn's disease cannot be ascertained based on the said diagnositic modalities. (5) In a study by Walker et. al. ASCAs were present in 57% of CD and ASCA-positive status was a predictor for CD with sensitivity of 57%, specificity of 87%, positive predictive value of 78% and negative predictive value of 68%. Furthermore, ASCA was associated with proximal (gastroduodenal and small bowel involvement) rather than purely colonic disease ($P < 0.001$). In our patient, ASCA IgG was strongly positive, clinching the diagnosis. Patient was then treated for Crohn's.

According to the case series by Miehsler et. al (2) comparing the efficacy of the different pharmacologic therapies for upper GI Crohn's, initial therapy with a proton pump inhibitor and steroids showed significant clinical improvement after 2-12 months of treatment. Hence this was used in our patient. After 2 weeks, there was improvement based on endoscopic findings of less edematous and nodular pyloric area with less stenosis at the pyloric channel for which the neonatal scope was already able to pass. The patient was then started on liquids. After 1 month of treatment, UGIS showed normal transit of barium in the upper GI tract. Patient was instructed to eat full diet which the patient tolerated with no episodes of vomiting. Prednisone was eventually tapered off with a total time of treatment of 2 months. Patient was then given Azathioprine 2mg/kg/day and according to the of Miehsler et. al., use of this drug for at least 6 months induced remission.

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