

Case Report

A rare cause of gastric outlet obstruction

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Received: 27 March 2020

Revised: 01 May 2020

Accepted: 02 May 2020

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ABSTRACT

Gastric outlet obstruction is the clinical and pathophysiological consequence of any disease process that produces mechanical impediment to gastric emptying. It may be acute from inflammatory swelling and peristaltic dysfunction or chronic from cicatrix. Chronic inflammation of the duodenum may lead to recurrent episodes of healing followed by repair and scarring ultimately leading to fibrosis and stenosis of the duodenal lumen. We would like to present a unique case of an elderly lady presenting with intractable vomiting over 3 months, gradually progressive which aggravated on consuming solids initially to consuming liquids later. After thorough investigations a provisional diagnosis of chronic duodenal ulcer with gastric outlet obstruction probably due to cicatrix was made. On laparotomy there was a chronic scarred duodenal ulcer following a previously contained perforation which was causing the gastric outlet obstruction. Cholecystectomy, duodenoplasty and loop gastrojejunostomy was performed with no complications post-operatively. This is a rare case of previous contained duodenal perforation causing gastric outlet obstruction.

Keywords: Gastric outlet obstruction, Duodenal ulcers, Peripyloric adhesions

INTRODUCTION

Gastric outlet obstruction (GOO) is the clinical and pathophysiological consequence of any disease process that produces mechanical impediment to the gastric emptying. It is commonly caused by peptic ulcer disease (PUD), locally advanced pancreatic cancer, gastric polyps, pyloric stenosis, ingestion of caustics, trichobezoars, proximal gallstone obstruction etc. Acute inflammation of the duodenum can lead to mechanical obstruction, with a functional GOO manifested by delayed gastric emptying time, anorexia, nausea and vomiting.¹ It commonly presents with non-bilious vomiting, anorexia, malnutrition, metabolic alkalosis, dehydration and marked weight loss.² Obstruction due to PUD can be acute from inflammatory swelling and

peristaltic dysfunction or chronic from cicatrix.² The gold standard procedure for obstructing duodenal or pre pyloric ulcer is distal gastrectomy with bilroth II gastrojejunostomy and truncal vagotomy.³ An alternative operation is laproscopic gastrojejunostomy and selective vagotomy.³

CASE REPORT

A 55 years old female with an average built and poor nourishment presented with intractable vomiting of 3 months duration which gradually increased in frequency, aggravating on consuming solid food initially and later progressed to liquids. Vomiting was nonbilious and was not relieved with medication. It was associated with decreased appetite and significant weight loss (15 kgs in

3 months). She had a history of open abdominal surgery 4 years back which was probably for a peptic ulcer perforation (details of which were not available). On initial evaluation all her vitals were stable. Her BMI was 15.2. Abdomen was scaphoid in shape with an upper midline scar, it was non-tender, with no guarding or rigidity and no palpable mass. Bowel sounds were present (8-10 per min) and on ausculto-percussion, the lower border of stomach was 2 cm above the umbilicus. Visible gastric peristalsis and succussion splash was present. Blood investigations were within normal limits. Arterial blood gas analysis was suggestive of hypokalaemic, hyperchloremic metabolic alkalosis.

Imaging findings

Ultrasound of abdomen and pelvis showed circumferential wall thickening of proximal duodenum measuring 10mm and also multiple gallstones. After a barium meal, contrast did not pass freely beyond 2nd part of duodenum. CECT abdomen and pelvis showed short segment of pylorus and proximal duodenum having circumferential wall thickening. No enlarged regional lymph nodes were noted. We had a suspicion of carcinoma duodenum. Upper GI endoscopy showed dilated first part of duodenum and endoscope could not be negotiated beyond the first part.

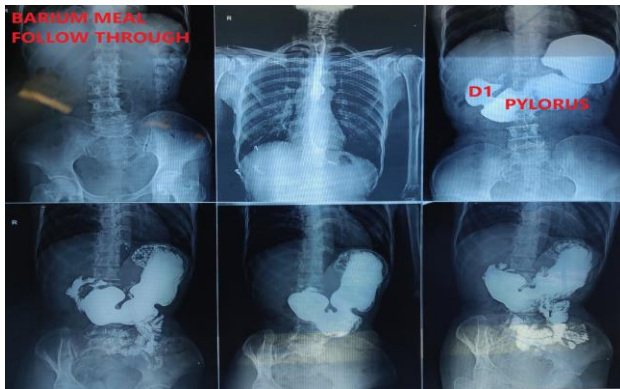


Figure 1: Barium meal follow through (arrows showing site of obstruction).

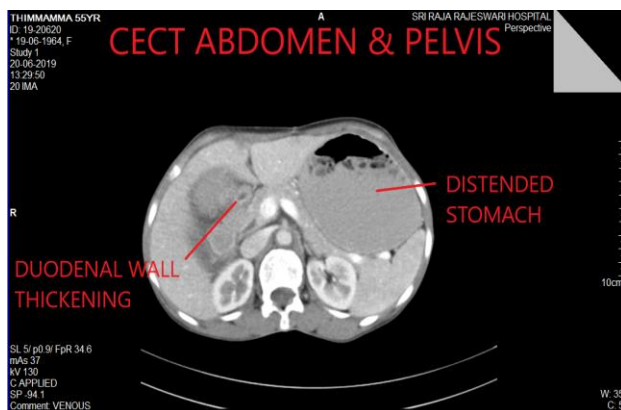


Figure 2: CECT abdomen and pelvis.

Exploratory laparotomy was performed. A thickened gallbladder adherent to the duodenum with frozen Calot's region was noted. Proximal D2 was completely obstructed due to previous duodenal ulcer scar and adhered to Hartmann's pouch of gallbladder. There was no cholecystoduodenal fistula. There was localised collection (around 5ml yellowish fluid) near the duodenum-Hartman's junction. The duodenum opened up during dissection. Multiple gallstones (around a 100) - yellow cholesterol stones were also noted. Adhesiolysis, cholecystectomy, duodenoplasty and loop gastrojejunostomy was performed. Patient's postoperative period was uneventful and she improved symptomatically.

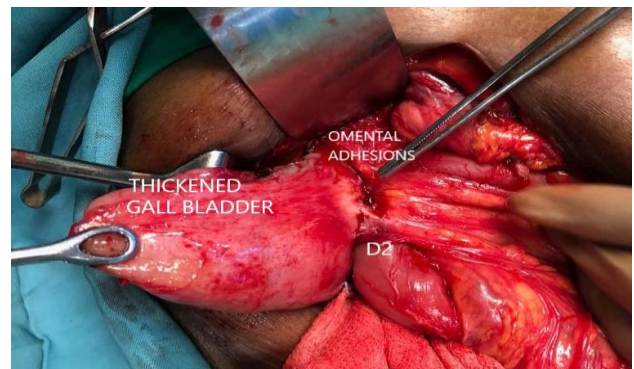


Figure 3: Intraoperative findings.



Figure 4: Cavity of the contained previous perforation.



Figure 5: Loop gastrojejunostomy performed.

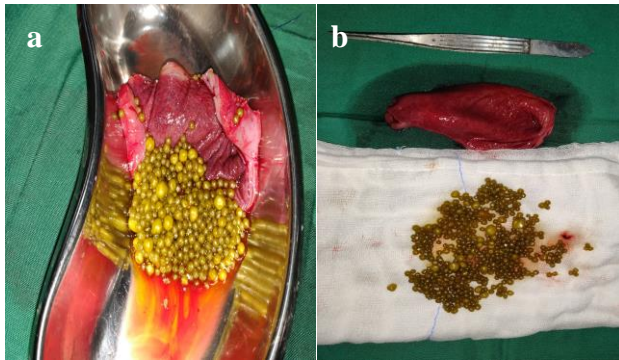


Figure 6 (a and b): Gall bladder with multiple gallstones.

DISCUSSION

GOO occurs in no more than 5% of patients with PUD.² Approximately (1-2) % of patients with peptic ulcer disease develop GOO, and about 80% of GOO due to peptic ulcer disease is caused by duodenal ulcers.⁴ Obstruction need not be due to cicatrization alone, peripyloric adhesion, perhaps from previous perforation is often the cause of obstruction.⁴ Prolonged gastric drainage before and after surgery is not necessary.⁴

In a retrospective study done on 58 patients who had surgery for obstructing duodenal ulcer, 48% was due to cicatrization, 28% due to adhesions and cicatrization, 16% due to adhesions alone and 8% due to inflammatory edema.⁴ patients with adhesion had evidence of sealed or treated perforation.⁴

GOO is now the commonest indication for duodenal ulcer surgery.⁴ Endoscopic balloon therapy may be attempted as an alternative to surgery, with balloon dilation reporting success rates of 76% after repeat dilations.⁵ Number of cases with cicatrized duodenal ulcer as the chief etiological factor for GOO is diminishing, and the number of cases of antral carcinoma of stomach as the cause of GOO is increasing.⁶ Acquired GOO is more commonly owing to malignancy than ulcer disease.⁶ The incidence of malignancy in patients presenting with GOO is greater than 50%.⁶ Endoscopy is the preferred method for diagnosis of patients presenting with GOO.⁷

Outcomes may be improved with effective ulcer therapy with acid reduction and eradication of *Helicobacter pylori*.⁷ Surgery is associated with significant morbidity and mortality and should be reserved for endoscopic treatment failures.⁷ The endoscopic treatment of GOO should be approached with caution because malignancy cannot be reliably excluded by endoscopic or radiological

studies.⁸ The aetiology of gastric outlet obstruction cannot be predicted by age, history of peptic ulcer disease, or non-steroidal anti-inflammatory drug use.⁸

CONCLUSION

Duodenal ulcer can cause gastric outlet obstruction acutely due to inflammatory swelling and peristaltic dysfunction or chronically due to cicatrix. Peripyloric adhesion, perhaps from previous perforation can also be the cause of obstruction. Rarely a previous contained duodenal perforation can also cause gastric outlet obstruction.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

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Cite this article as: Murali A, Krishnappa R, Murugesh RB, Rajagopalan S. A rare cause of gastric outlet obstruction. Int Surg J 2020;7:2062-4.