Peptic Duodenal Stricture Causing Gastric Outlet Obstruction

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ABSTRACT

Gastric outlet obstruction (GOO) is an uncommon but serious problem caused by benign causes. The incidence of GOO caused by peptic ulcer disease (PUD) is decreasing due to successful Helicobacter pylori treatment. Hence, these aggressive cases are rare. We present a case of a man who experienced intermittent abdominal pain and constipation for a week but acutely worsened in the last five days. He complained of intractable nausea and vomiting, leading to intolerable oral intake. Physical examination revealed abdominal distention, tenderness in epigastrium and right hypochondrium, and hypoactive bowel sounds. Laboratory results revealed leukocytosis with neutrophilia. Gastroscopy showed erosive esophagitis, pangastritis, and large duodenal ulcer causing stricture of duodenum pars I. An open distal gastrectomy, Roux-en-Y reconstruction, and fundoplication was then performed. The patient's general condition improved after ninth day of care and was discharge on the eleventh day of care. PUD is a major cause of GOO, but incidence has decreased to 5% with the use of proton pump inhibitors. If the distal stomach or duodenum is significantly obstructed, GOO should be considered. The gold standard for diagnosis is endoscopy. Initially, conservative and supportive therapy should be done followed by endoscopic treatment in appropriate cases. If there is no improvement, surgical interventions should be done. This case demonstrated the need for definitive invasive procedures in the intervention of GOO caused by PUD. When conservative measures fail, this case also adds support to the direct anatomic treatment of duodenal strictures.

Keywords: *peptic ulcer disease, gastric outlet obstruction, duodenal stricture*

ABSTRAK

Obstruksi saluran keluar lambung karena penyebab jinak adalah kasus yang jarang namun serius. Insiden obstruksi saluran keluar lambung karena penyakit ulkus peptikum menurun karena keberhasilan pengobatan Helicobacter pylori sehingga kasus agresif seperti itu jarang terjadi. Kami melaporkan kasus seorang laki-laki dengan riwayat nyeri perut dan konstipasi secara intermiten selama seminggu, tetapi memburuk secara akut selama lima hari terakhir. Pasien tidak dapat mentolerir asupan oral dengan keluhan mual dan muntah. Pemeriksaan fisik menunjukkan distensi abdomen, nyeri tekan pada epigastrium dan hipokondrium kanan, dan bising usus hipoaktif. Hasil laboratorium menunjukkan leukositosis dengan neutrofilia. Gastroskopi menunjukkan esofagitis erosif, pangastritis, dan ulkus duodenum besar yang menyebabkan striktur duodenum di duodenum pars I. Laparotomi gastrektomi distal, rekonstruksi Roux-en-Y, dan fundoplikasi kemudian dilakukan. Kondisi umum pasien membaik setelah hari kesembilan perawatan dan dipulangkan pada hari kesebelas perawatan. Penyakit ulkus peptikum merupakan penyebab utama kasus obstruksi outlet gaster, namun kejadian obstruksi

outlet gaster akibat penyakit ulkus peptikum menurun hingga 5% dengan penggunaan inhibitor pompa proton. Obstruksi saluran keluar lambung harus dipertimbangkan pada kasus dengan obstruksi signifikan pada gaster distal atau duodenum. Endoskopi adalah standar emas untuk diagnosis. Terapi konservatif dan suportif harus dilakukan pada awal diagnosis diikuti dengan pengobatan endoskopi pada kasus yang sesuai. Jika tidak ada perbaikan meskipun pengobatan konservatif dan suportif, intervensi bedah harus dilakukan. Kasus ini menunjukkan perlunya intervensi bedah definitif dalam pengobatan obstruksi saluran lambung yang diinduksi oleh penyakit ulkus peptikum dan menambahkan dukungan untuk pengobatan anatomi striktur duodenum secara langsung ketika tindakan konservatif gagal.

Kata kunci: *penyakit ulkus peptikum, obstruksi outlet gaster, striktur duodenum*

INTRODUCTION

Peptic ulcer disease (PUD) is a condition in which the inner lining of the gastrointestinal (GI) tract becomes obstructive as a result of gastric acid or pepsin secretion. It reaches the gastric epithelium's muscularis propria layer. The stomach and proximal duodenum are the most common sites of PUD. In patients with duodenal ulcers, epigastric pain usually occurs 2–3 hours after eating. PUD is a global issue, with a lifetime risk of development of 5–10%. Generally, the incidence of PUD has decreased due to better sanitary and hygienic environments, as well as appropriate therapy and reasonable use of nonsteroidal anti-inflammatory drugs (NSAIDs).[1] Duodenal ulcers are four times more common than gastric ulcers. Males are also more likely than females to develop duodenal ulcers.

Gastric outlet obstruction (GOO) makes up a small percentage of all gastrointestinal tract obstructions (< 2%). A partial or complete obstruction in the distal stomach, pylorus, or proximal duodenum obstructs the passage of gastric contents into the duodenum, resulting in gastric outlet obstruction. GOO is an unusual complication of duodenal stricture (2–3%), with an untreated or long-standing duodenal ulcer with severe mural inflammation and/or fibrosis accounting for almost 80% of cases.[2] GOO is a chronic condition that cause abdominal pain, postprandial vomiting, early satiety, and weight loss. It is caused by either a benign or malignant mechanical obstruction or a motility disorder that prevents gastric emptying. Mechanical obstruction can occur either intrinsically or extrinsically to the stomach or anatomically in the distal stomach, pyloric tract, or duodenum. PUD was the leading cause of GOO cases. The incidence of GOO induced by PUD has decreased to 5% due to improved treatment of *Helicobacter pylori* (*H. pylori*) infection and the use of proton pump inhibitors (PPIs). GOO had been linked to an underlying cancer in 50–80% of cases recently.[3] Males are more likely than females, with a 3–4 to 1 ratio for benign and malignant causes.[4]

The causes of GOO are various. When a patient has GOO, primary care providers or emergency medicine are often the first to see the patient. A detailed examination, including a computerized tomography (CT) scan and endoscopy, must be performed to identify the cause of the obstruction. Diagnosis and treatment must be carried out as soon as possible because delay can lead to further compromise in the nutritional status, edematous tissue, and complicated invasive treatment. Hence, early management should focus on identifying the underlying cause and correcting volume and electrolyte abnormalities should be taken. This case report focuses on a gastric outlet obstruction patient caused by duodenal ulcer causing duodenal stricture.

CASE ILLUSTRATION

A 45-year-old man was admitted to the emergency room in Siloam Hospital Lippo Village with a history of abdominal pain and constipation for a week, as well as progressive abdominal distension. He could not take any liquids or solid food after two days and started vomiting, consisting of remnant of latest meal, and no history of coffee grounds-like vomit. The patient had history of significant gastroesophageal reflux disease (GERD) and gastritis. He had never had abdominal surgery before.

The patient was fully conscious, afebrile, and showed no signs of dehydration during the examination. He had general weakness, with blood pressure of 130/90 mmHg, heart rate 78 bpm, respiratory rate 18 times per minute, and temperature of 36.1°C. Abdominal examination revealed abdominal distension without any scars or obvious hernias, tenderness in the epigastrium and right hypochondrium without muscle guarding, and bowel sounds were hypoactive. A digital rectal examination was unremarkable.

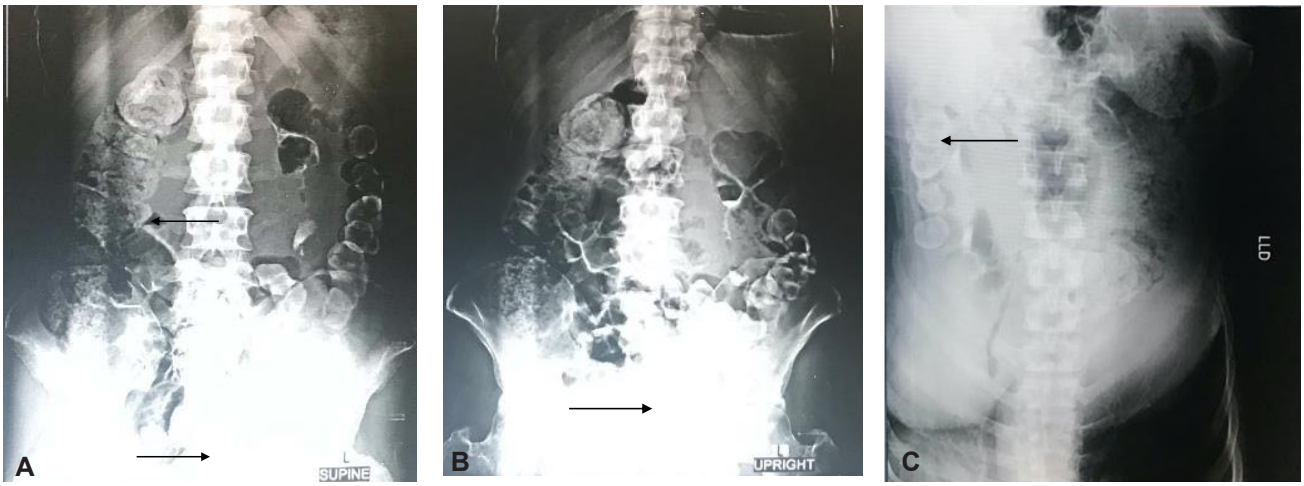

Figure 1. Plain abdominal X-ray film showed radio-opaque lesions occupying the lumen of intestines intra-abdominally and lesser pelvic cavity (arrow). (A) Supine; (B) Upright; (C) Left lateral decubitus.

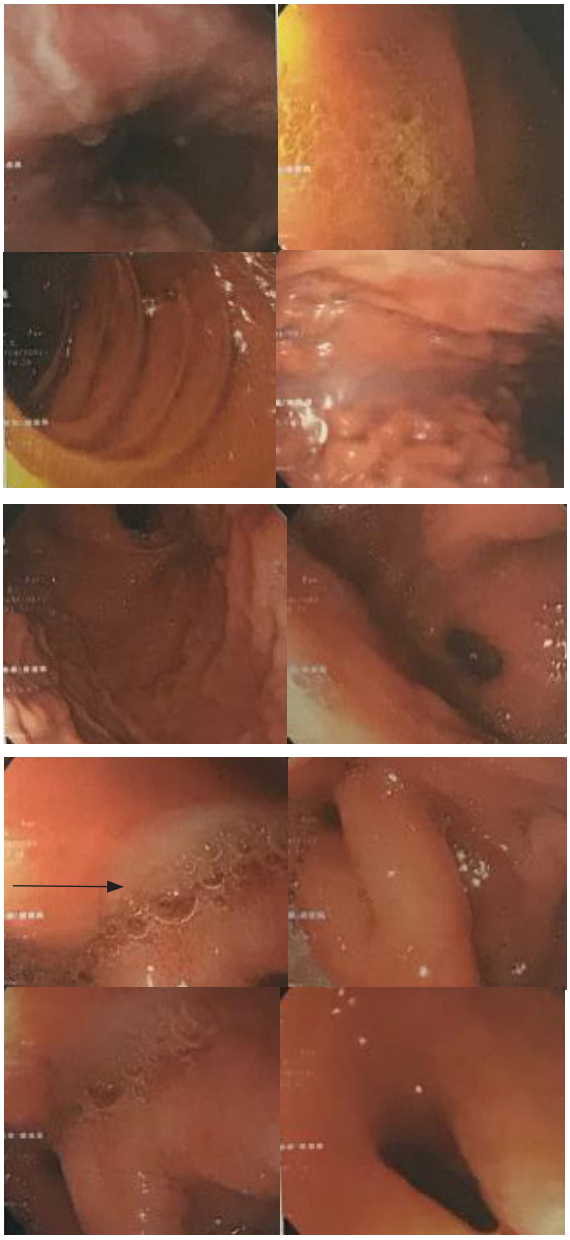

Figure 2. Gastroscopy showed erosive esophagitis (arrowhead), pangastritis, and large duodenal ulcer causing duodenal stricture in the first part of the duodenum (arrow)

Electrocardiography showed a sinus rhythm. Laboratory investigations showed hemoglobin (Hb) 14.00 g/dL, hematocrit (HCT) 41.20%, white blood cell (WBC) count 15,160/mm^3, neutrophil count 87%, and platelet count (PLT) 372,000/mm^3. It also showed blood sugar level 89 mg/dL, blood urea nitrogen (BUN) 11.7 mg/dL, serum creatinine (SC) 0.92 mg/dL, sodium level 136 mEq/L, potassium level 3.9 mEq/L, and chloride level 95 mEq/L. A plain abdominal x-ray film showed radio-opaque lesions occupying the lumen of intestines intra-abdominally and lesser pelvic cavity (Figure 1). A contrast-enhanced CT of the abdomen showed gastric distension with predominance of intraluminal fluid with luminal narrowing in the pyloric region, suggesting stenosis due to inflammation. A gastroscopy showed erosive esophagitis, pangastritis, and large duodenal ulcer causing duodenal stricture (Figure 2).

The patient was assessed with gastric outlet obstruction due to stricture of duodenum pars I, GERD, and gastritis. He was put on supportive care, which included intravenous fluids, antibiotics, and nasogastric tube and urinary catheter insertion. A broad-spectrum antibiotic was administered intravenously, consisting of ceftriaxone 1 gram. The patient also received omeprazole intravenously 40 mg twice daily, ondansetron intravenously 8 mg thrice daily, and glycerin suppository twice daily. Conservative approach was taken as the patient was given pantoprazole intravenously 40 mg twice daily and sucralfate orally 1 g thrice daily, and rebamipide 100 mg orally thrice daily.

We consulted with a digestive surgeon and was suggested open distal gastrectomy, Roux-en-Y reconstruction, and fundoplication. Under general anesthesia, a midline incision was made. There was

pyloric antrum attached to the anterior abdominal wall, hence it was sharply released using monopolar electrocautery. Distal gastrectomy was performed at the site of obstruction. Roux-en-Y reconstruction (end to side gastrojejunostomy; end to side jejunojejunostomy) and fundoplication at +/-2 cm esophageal junction proximally 180–200° was also performed. A tube drain was inserted in the subhepatic space, and his abdomen was closed in layer upon layer.

Postoperative outcome was acceptable. The patient was discharged on the third postoperative day after successfully adjusting to an oral diet. There was no recurrence of symptoms and was recovering well from his surgery on subsequent follow-up.

DISCUSSION

Peptic ulcer disease can lead to serious complications, such as bleeding, perforation, penetration, or GOO in approximately 25% of patients.[5] Benign GOO is the least common complication of PUD, occurring in just approximately 3% of cases.[6] This complication affects less than 5% of patients with duodenal ulcer disease. In addition, *H. pylori* is responsible for up to 95% of duodenal ulcers.[7] In the past, PUD was the major cause of GOO, but this has decreased in recent years, and malignancy is currently the major cause of gastric outlet obstruction. Several factors can contribute to gastric outlet obstruction, which are rapidly reversible factors (spasm, edema, inflammation, pyloric dysmotility associated with ulcers or inflammatory changes), slowly reversible factors (fibrosis, scarring, and deformity), prolonged factors (gastric atony), causing gastric retention. The severity of GOO symptoms is determined by the underlying disease. Abdominal or epigastric pain, as well as vomiting, are the most frequent symptoms. Dehydration and splashing are the most common signs. Indigestion, bloating, early satiety, nausea, vomiting, anorexia, epigastric pain, and weight loss are all symptoms of gastric retention. Abdominal distension may be present, indicating GOO. Reduced visceral sensitivity occurs in some patients with PUD, leading to high-grade GOO without symptoms of gastric distress. Chronic pain, weight loss, and malnutrition, may be the major symptoms in chronic cases or cases of malignancy.[8] The patient in our case had a history of gastritis which resulted in ulcers and obstruction of the duodenum pars I. Ulcers most prevalently occur in the gastric antrum and duodenal blub, where the ulcer and its stricture are located.[6] Gastritis refers to inflammation of the gastric lining, which includes acute gastritis, chronic gastritis, and atrophic gastritis. Chronic gastritis is a long-term condition that causes the gastric lining to slowly erode, leading to the formation of ulcer.[9]

Contrast studies can reveal information about the underlying cause. Complete obstruction should be considered if there is no contrast transition to the small bowel. CT scans can reveal details such as the thickness of pylorus and gastric wall, as well as showing whether there are lymph node or pancreatic lesions. In cases of suspected gastric outlet obstruction, endoscopy is used to make a definite diagnosis of the underlying cause. Malignancy must be excluded in all cases of outlet obstruction because malignancy is responsible for approximately 40% of causes of GOO.[8]

The management of benign GOO should be based on the underlying cause, which can be divided into two categories: non-surgical treatments and surgical treatment. When the cause of benign GOO is reversible, conservative measures and supportive care, such as intravenous PPIs, sucralfate, discontinuation use of NSAIDs, treatment of *H. pylori,* and gastric decompression, are used. Endoscopic balloon dilation (EBD), intralesional steroids, endoscopic incisions, endoscopic ultrasound-guided gastroenterostomy with lumen-appropriate metal stent (LAMS), and self-expanding metal stent (SEMS) are some of the other non-surgical options. Resective surgery and non-resective (divertive) surgery are two surgical options.[10]

Patients with GOO symptoms should be admitted to the hospital. Fluid resuscitation and electrolyte replacement should be favored. Decompression should be done with a nasogastric tube, which relieves pain and discomfort caused by a full stomach, cleans the area before endoscopic procedures, and reduces abdominal capacity before surgery.[10]

PPI can reduce acid secretion, initiates ulcer healing, improves inflammatory edema, and helps resolve obstruction. This treatment works in about half of the cases, especially when the obstruction is due to associated inflammation, edema, spasm, or pyloric dysmotility instead of dense scar tissue. The aims of the treatment are to alleviate symptoms, deflate stomach to restore tone, and take account of loss of fluid. Parenteral nutrition must be considered if the patient is severely malnourished. However, some patients who do not respond to conservative and supportive treatment, may eventually require surgery or endoscopic dilatation.

Since GOO is not a medical emergency, endoscopic or surgical treatment could be postponed once the

patient is stable and hydration status and electrolyte balance has been restored. Delay is also recommended if the patient's nutritional status is poor (albumin < 2.8 is generally a strong indicator of a poor surgical outcome) or if the stomach is very dilated, as postoperative gastric atony is more likely to occur and can be avoided by preoperative decompression.

Patients with benign gastric obstruction, such as PUD, should consider endoscopic dilatation as a treatment option. Endoscopic balloon dilation (EBD) can be used to resolve gastric outlet obstruction induced by PUD. In most patients with non-malignant obstruction, EBD has become the first-line therapy. It is unclear the maximum diameter of the stenosis at which symptoms appear. However, many institutions recommend a dilation of up to 15 mm, which has been linked to a reduction of symptoms. This treatment most often help ease symptoms immediately; however, long-term response varies between 16% and 100%, and patients may need more than one dilation procedure.[8] If two or more dilation procedures are required, surgery may be required.[11]

Surgery is reserved for patients with benign GOO who have failed to respond to medical treatment and endoscopic dilation. Surgery should, ideally, remove the obstruction while also addressing the underlying cause, such as PUD. Various surgeries to block ulcers have been described. Patients are preferred to undergo laparoscopic surgery rather than laparotomy since patients can resume oral intake sooner, stay in the hospital for less time, and lose less blood during the procedure. Due to its positive effects on patients, laparoscopic surgery may become the gold standard in the future. Gastrojejunostomy is the simplest surgical procedure for removing obstruction.[8] However, patients with GOO and PUD require vagotomy along with drainage procedures, either gastrojejunostomy or pyloroplasty, antrectomy or distal gastrectomy. This eliminates the obstructive factor as wells as the stimulus for gastric secretion.[12] A highly selective vagotomy combined with stricture widening is another option. For post-pyloric obstruction, other types of duodenoplasty have been described. Surgical resection of the pathological segment or pyloroplasty are used to treat GOO caused by peptic stricture. Depending on the etiology, a vagotomy procedure may be added to the surgery. In patient with preserved anatomy, a gastrojejunostomy may be taken into account. If the anatomy has been altered, however, Roux-en-Y jejunostomy should be preferred.[10] Vagal denervation surgery is always associated with a risk of prolonged gastric atony, which may necessitate additional gastrectomy. The percentage of patients who have gastric dysfunction after surgery varies from 10–50%.[13]

In the case of benign GOO, there is insufficient evidence to support surgical management compared with endoscopic management. However, endoscopic management is usually attempted first, with the goal of successfully resolving benign GOO, whereas surgery is reserved for "rescue" therapy if endoscopic intervention fails or if severe side effects from endoscopic therapy occur that require surgical intervention (e.g. perforation). Depending on the etiology of stricture, the surgical intervention approach to treatment of benign GOO may vary. Due to acid suppressive therapy and identification of *H. pylori* as the cause of PUD, surgery for benign GOO is uncommon. Laparoscopic gastrojejunostomy and pyloroplasty have been shown to be safe and effective treatment with low morbidity and mortality.[14]

However, due to patient's consideration on the cost of surgery as the patient receives benefits from the National Health Insurance Organized Body and our hospital as a secondary care center only have the facility to complete an open exploration. As our digestive surgeon suggested, our patient underwent an open distal gastrectomy, which enabled for thorough and substantial duodenal mobilization and tension-free repair. Metabolic and nutritional changes, gastroparesis, dysmotility, alkaline reflux gastritis, afferent or efferent blind loop syndrome, dysplasia, long-term risk of metaplasia, gastric malignancy, and future marginal ulceration are all possible complications.[6] The operation went well, and one month later, the patient had a wide patent duodenum without signs of obstruction. At follow-up, the patient had no complaint of abdominal pain, food restrictions or intolerance, nausea, or vomiting.

There is a significant heterogeneity in surgical and endoscopic management of benign GOO, where indirect comparative analysis is not feasible. Hence, factors linked to EBD response rates must be assessed and addressed. Future research is needed to compare endoscopic and surgical approaches to the management of patients with benign GOO.

REFERENCES

1. Lanas A, Chan FKL. Peptic ulcer disease. The Lancet 2017;390:613–24.
2. Massimo T, Adriana V. Peptic duodenal stricture causing gastric outlet obstruction: CT diagnosis with endoscopic correlation. Eurorad Abdom 2016;13857:x.

3. Tringali A, Giannetti A, Adler DG. Endoscopic management of gastric outlet obstruction disease. Ann Gastroenterol 2019;32:330–7.
4. Sukumar V, Ravindran C, Prasad RV. Demographic and etiological patterns of gastric outlet obstruction in Kerala, South India. N Am J Med Sci 2015;7:403–6.
5. Narayanan M, Reddy KM, Marsicano E. Peptic ulcer disease and *Helicobacter pylori* infection. Mo Med 2018;115:219–24.
6. Mirande MD, Mirande RA. Management of a postbulbar duodenal ulcer and stricture causing gastric outlet obstruction: a case report. Ann Med Surg 2018;29:10–3.
7. Fashner J, Gitu AC. Diagnosis and treatment of peptic ulcer disease and *H. pylori* infection. Am Fam Physician 2015;91:236–42.
8. Koop AH, Palmer WC, Stancampiano FF. Gastric outlet obstruction: a red flag, potentially manageable. Cleve Clin J Med 2019;86:345–53.
9. Sipponen P, Maaroos HI. Chronic gastritis. Scand J Gastroenterol 2015;50:657–67.
10. Iliklerden ÜH, Kalayci T, Kotan MÇ. Benign gastric outlet obstruction surgery: a tertiary center experience. East J Med 2021;26:450–6.
11. Shah SK, Walker PA. Gastric outlet obstruction. In Moore LJ, Todd SR, eds. Common Problems in Acute Care Surgery. 2nd ed. Switzerland: Springer Int Publ. 2017:II.p.221–32.
12. Lagoo J, Pappas TN, Perez A. A relic or still relevant: the narrowing role for vagotomy in the treatment of peptic ulcer disease. Am J Surg 2014;207:120–6.
13. Khorgami Z, Zhang C, Messiah SE, Cruz-Munõz N. Predictors of postoperative aftercare attrition among gastric bypass patients. Bariatr Surg Pract Patient Care 2015;10:79–83.
14. Jue TL, Storm AC, Naveed M, Fishman DS, Qumseya BJ, McRee AJ, et al. ASGE guideline on the role of endoscopy in the management of benign and malignant gastroduodenal obstruction. Gastrointest Endosc 2021;93:309–22.