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REVIEW

Management of gastric outlet obstruction: Focusing on endoscopic approach

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Abstract

Gastric outlet obstruction (GOO) is a medical condition characterized by epigastric pain and postprandial vomiting due to mechanical obstruction. The obstructions typically involved in GOO can be benign or malignant. Peptic ulcer disease is the most common cause of benign GOO, and malignant causes include gastric cancer, lymphoma, and gastrointestinal stromal tumor. With the eradication of *Helicobacter pylori (H. pylori)* and the use of proton pump inhibitors, the predominant causes have changed from benign to malignant diseases. Treatment of GOO depends on the underlying cause: Proton pump inhibitors, *H. pylori* eradication, endoscopic treatments including balloon dilatation or the placement of self-expandable stents, or surgery.

Key words: Gastric outlet obstruction; Balloon dilation; Metal stent

Core tip: The causes of gastric outlet obstruction are generally divided into benign and malignant. With the eradication of *Helicobacter pylori* and the use of proton pump inhibitors, the predominant causes have changed from benign to malignant diseases. Treatment of gastric outlet obstruction (GOO) depends on the underlying cause: Proton pump inhibitor, endoscopic techniques, or surgery. In this article, we review the etiology, diagnosis, and current treatment methods of GOO, especially endoscopic techniques.

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INTRODUCTION

Gastric outlet obstruction (GOO) occurs when gastric emptying is mechanically inhibited by various diseases, most of which involve obstruction of the gastric pylorus or proximal duodenum due to intrinsic or extrinsic factors. The precise incidence of GOO is unknown. Although GOO due to peptic ulcers has been common in the past, the use of proton pump inhibitors and identification of *Helicobacter pylori (H. pylori)* have reduced the incidence of peptic ulcer disease, and malignant diseases have become the main cause of GOO in recent decades, with about 50%-80% of GOO being caused by cancer[1-5]. As the predominant cause of GOO shifts from benign to malignant diseases, treatment methods have also changed. In this article, we review the etiology, diagnosis, and current treatment methods of GOO, especially endoscopic techniques.

ETIOLOGY

Benign gastric outlet obstruction

Peptic ulcer disease is the most common cause of benign GOO, accounting for approximately 90% of cases[6]. Caustic ingestion, inflammatory diseases such as Crohn's disease or tuberculosis, and non-steroidal anti-inflammatory drug-induced strictures may also result in GOO. Other rare benign causes are large gastric polyps, gallstone obstruction (Bouveret's syndrome), annular pancreas, pancreatic pseudocyst, and bezoars (Table 1)[7]. Peptic ulcer disease was the leading cause of GOO in the past, with the use of proton pump inhibitors and identification of *H. pylori*, the incidence has declined significantly. Currently, GOO is the least common complication of peptic ulcer disease. Less than 5% of complicated duodenal ulcer disease and less than 1-2% of complicated gastric ulcer disease develop obstructive complications[8,9].

Corrosive injury caused by caustic ingestion, including acid or alkali substances, can result in GOO by antral or pyloric scarring[10,11]. The incidence rate of GOO by caustic ingestion varies from 20 to 60%[10-12]. In a study of 41 cases of acid ingestion, 44.4% developed GOO[10], and another study reported that 36.8% of the 31 alkali-ingestion patients developed GOO[11].

Inflammatory causes, such as Crohn's disease or tuberculosis, also cause GOO. Crohn's disease mostly invades the distal gastrointestinal tract, and it rarely invades the upper gastrointestinal tract such as the stomach or duodenum alone. Clinically, severe gastroduodenal Crohn's disease is rare, in which case it continuously invades the antrum, pylorus, and proximal duodenum[13]. Gastroduodenal tuberculosis is rare, occurring in only 0.3%-2.3% of patients with tuberculosis. GOO was identified in 61% of 23 patients with gastroduodenal tuberculosis which was confirmed by histopathological examination[14].

Nonsteroidal anti-inflammatory drugs (NSAIDs) can also cause GOO. NSAIDs reduce prostaglandin E2 to induce pyloric edema and scarring, and increase histamine release to increase gastric secretion, reduce mucosal absorption, and cause gastric motility disturbances, leading to GOO[15]. In a study of 10 cases of NSAID-induced GOO in 2011, the most common site of involvement was the duodenum, followed by the pylorus and duodenum, and then pylorus only[16]. Most strictures were short web-like in nature, and endoscopic balloon dilation was successful in 90% of cases.

Malignant gastric outlet obstruction

In recent decades, malignancy has been the most common cause of GOO. The most common causes are pancreatic and gastric cancer, but lymphomas, duodenal carcinoma, biliary tract carcinoma, ampullary carcinoma, and metastatic malignancies can also cause malignant GOO. In pancreatic cancer, 15%-20% of patients have been reported to develop GOO[17].

DIAGNOSIS

The diagnosis of gastric outlet obstruction is usually suggested by history and physical examination. Patients have suffered recurrent vomiting and show up electrolyte abnormalities including hypokalemia or hypochloremic metabolic alkalosis. The gastrin secretion due to gastric expansion increases serum gastrin levels (400-800 pg/mL range) and can be confused with Zollinger-Ellison syndrome[18]. Tests such as endoscopy, and barium study are helpful for diagnosis. Plain radiography

Table 1 Causes of gastric outlet obstruction

Benign	Malignant
Peptic ulcer disease	Gastric cancer
Caustic ingestion	Gastric lymphoma
NSAID induced stricture	Pancreas cancer
Bouveret syndrome	Duodenal cancer
Hypertrophic pyloric stenosis	Cholangiocarcinoma
Iatrogenic	Gallbladder cancer
Post-surgical scar or anastomosis stricture	Metastatic cancer
Endoscopic submucosal dissection	
Endoscopic mucosal resection	
Inflammatory causes	
Crohn's disease	
Pancreatitis	
Inflammatory polyps	
Infectious causes	
Tuberculosis gastroenteritis	
CMV gastroenteritis	
Infiltrative causes	
Eosinophilic gastroenteritis	
Amyloidosis	

NSAID: Nonsteroidal anti-inflammatory drugs; CMV: Cytomegalovirus.

may show a large gastric shadow. Contrast studies with barium or water-soluble contrast agents may show an enlarged stomach and provide clues as to the underlying disease. The absence of any contrast passage in the small intestine suggests a complete GOO. CT scan is helpful, especially for evaluating the mural thickness of the pylorus or gastric wall, lymph node enlargement, pancreatic or biliary tract, and retroperitoneum[9].

Endoscopy is the most useful examination to establish gastric outlet obstruction and obtain tissue specimens from obstructing areas for confirmation or exclusion of malignant GOO. Endoscopy should be performed after fasting for over 4 hours, and nasogastric tube suction is recommended before endoscopy to reduce the risk of aspiration.

TREATMENT

All patients with symptomatic GOO need to be hospitalized. Fluid resuscitation with normal saline and correction of electrolyte imbalance should be performed first. Nasogastric decompression should be initiated during hospitalization. This helps relieve discomfort and pain caused by gastric distension, clear the field during endoscopic procedure, and reduce the gastric capacity before surgery. In patients with benign gastric outlet obstruction due to acute peptic ulcer disease, patients showed improvement in symptoms due to reduced edema and spasm due to inflammation after 48-72 h with nasogastric decompression and proton pump inhibitors.

H. pylori eradication can be performed in patients with benign GOO with *H. pylori* infection. The prevalence of *H. pylori* in GOO varies from 33% to 90%[19]. Kate *et al*[20] reported a high prevalence of *H. pylori* infection in duodenal ulcers with GOO, even without active ulcers. Acute ulcers associated with *H. pylori* infection cause obstruction due to inflammation and edema, and antimicrobial treatment can help improve occlusion. Mohsina *et al*[21] summarized reports on the role of *H. pylori* in GOO.

If GOO is irreversible with medical therapy, definitive treatment is required based upon the underlying cause (Table 2). Until the development of endoscopic procedures, surgery was the only treatment for these patients. In the past, 80%-90% of ulcer related GOO patients underwent surgery[22], and the only treatment option for caustic GOO patients was surgery as well[12]. Recent reports suggest that endoscopic balloon dilation is an effective treatment option, as an alternative to surgery in the

majority of peptic ulcer disease-related and caustic GOO patients[23-31]. In benign GOO, intraluminal stent insertion is a poor treatment option. There are no commercial stents available for benign GOO, and if uncovered stents are used, stent removal is impossible, and long-term patency is not guaranteed, and stent migration occurs frequently when covered stents are used. On the other hand, if curative surgery is not possible in malignant GOO, there are palliative options such as endoscopic placement of self-expanding metal stents (SEMS), and bypass surgery such as gastrojejunostomy. Surgical gastrojejunostomy for palliative purposes has a high mortality of up to 10%[32], and previous reports have shown that palliative SEMS insertion is more cost-effective, reduces the number of days of hospitalization, and improves symptoms rapidly[33,34]. Endoscopic SEMS insertion is widely performed in malignant GOO.

ENDOSCOPIC MANAGEMENT

Endoscopic balloon dilation

Benjamin *et al*[35,36] first reported the use of endoscopic balloon dilation (EBD) of the pylorus for the treatment of GOO using a through-the-scope 5-mm balloon with good clinical outcome. Subsequent reports have shown the safety and effectiveness of EBD for GOO management[23-31]. Dilations can be performed with endoscopy and using balloon dilators inserted through the working channel of the endoscope, or using balloons placed over a guidewire under fluoroscopic guidance. If adequate dilation is achieved, the clinical response is maintained in 70%-80% of patients[25,30]. Repeated recurrence of stricture after EBD may be an indication of surgery. If more than two sessions of dilations are required, they are highly associated with the probability of surgery[28].

EBD may also be effective for GOO caused by caustic injury or endoscopic submucosal pylorus dissection[37,38]. In a single-center study published by Kochhar *et al*[39] recently, EBD had a clinical success of 97.3% and no recurrence during a 98-month follow-up period. Perforation occurred in 2 of 111 patients. However, the mean number of sessions was 2-13 times in caustic GOO, while only 1-3 times in PUD-induced GOO[27,38]. GOO caused by other causes, such as Crohn's disease and tuberculosis, may also benefit with EBD[26,40].

EBD is generally a safe procedure, with complications of bleeding and perforation in diameters less than 15 mm rare. Perforation occurred more often when the diameter was over 15 mm[7,24,41,42]. Pain and minor bleeding are common during EBD procedures, but they are self-limited, whereas arterial bleeding is rarely reported[40].

Intralesional steroids

A combination of balloon dilation and intralesional steroid injection could be performed to inhibit stricture formation. Triamcinolone blocks the cross-linking of collagen and prevents scar contracture[43]. There are few reports on the treatment of pyloric strictures with intralesional steroids. Kochhar *et al*[44] and Lee *et al*[45] reported the efficacy of intralesional steroids.

Endoscopic incision

Endoscopic incision could be further performed after endoscopic balloon dilation in pyloric stenosis refractory to EBD. Boron *et al*[46] reported an electrosurgical incision using sphincterotomy, and Hagiwara *et al*[47] used a needle-knife radial electrosurgical incision in refractory anastomotic pyloric stenosis.

Endoscopic placement of self-expanding metal stents

SEMS insertion is used as a palliative treatment for malignant GOO and is used in cases of malignant gastrointestinal obstruction that cannot be surgically treated. The goal of SEMS insertion is to relieve obstruction symptoms. To evaluate the degree of symptom relief, the GOO score, which evaluates the severity of symptoms defined as satiety, nausea, and early vomiting, scoring based on the patient's oral intake level[48].

Generally, the technical and clinical success rates are reported to be 89%-98% and 86%-89%, respectively, which is very good in terms of short-term success rates[49-52]. SEMS insertions should be considered in patients with a short life expectancy (less than 2-6 mo)[53]. In addition, there should be no other occlusion site in the distal part of the stent insertion site, and the presence of free perforation or peritonitis are contraindications to endoscopic stent placement[54].

In malignant GOOs, biliary obstruction is often coexistent. Placement of the biliary metal stent should be considered before insertion of the duodenal stent. Since the endoscopic approach to the biliary tract is very limited after the duodenal stent is inserted, the percutaneous transhepatic approach is usually required[54].

Table 2 Treatment of gastric outlet obstruction based upon the underlying cause

Underlying cause	Treatment	
Benign		
Peptic ulcer disease	PPI ± HPE (1st option)	EBD or surgery (2nd option)
Crohn disease	Corticosteroid (1st option)	EBD or surgery (2nd option)
Caustic ingestion	EBD or surgery	
Bouveret syndrome	Surgery or endoscopic removal	
Large gastric polyp	Endoscopic resection	
Malignant		
Palliative	Endoscopic stent (covered or uncovered)	
	EUS-guided gastroenterostomy	
	Surgical resection, surgical bypass (gastrojejunostomy)	
	Radiation therapy	
Curative	Surgery	
	Chemotherapy (for lymphoma)	

PPI: Proton pump inhibitor; HPE: Helicobacter pylori eradication; EBD: Endoscopic balloon dilatation; EUS: Endoscopic ultrasound.

Covered versus uncovered SEMS: Uncovered stents are widely used for the treatment of malignant GOO. It is less likely to migrate and more flexible, but the tumor can grow into the stent and result in stent obstruction. Covered stents are increasingly used in Europe because they provide the advantage of low tumor growth. However, they are more prone to migration and less flexible than uncovered stents[55,56]. According to Kim *et al*[57] stent migration rate was much higher in covered stents than in uncovered stents (28% *vs* 3%) within 8 weeks of stent insertion.

According to a systematic review by Yang *et al*[58], there were no significant differences in technical or clinical success rate, long-term patency, or complications in three meta-analyses, in which comparison of efficacy and safety between covered or uncovered SEMS for malignant GOO were assessed.

Currently uncovered SEMS, rather than fully or partially covered stents, have been shown to be a standard treatment for managing malignant GOO, with low migration rates and better bile outflow[55,56,59]. Tumor ingrowth/overgrowth has been reported in 17.2% of patients receiving bare metal stents and in 6.9% of patients with covered stents[60,61]. This stent obstruction can be managed with a stent-in-stent technique, and stent occlusion rate was reported to be 10%-34% after the secondary SEMS insertion[62,63]. The development of stents to compensate for the shortcomings of the existing stents continues, and recent new covered stents with anti-migration designs have been suggested to be superior in terms of stent patency and complications[64].

SEMS *vs* surgery: The comparison of the effects and safety of surgical methods and endoscopic stents as palliative treatment for malignant GOO have been presented in various studies. Compared to surgery, the advantages of endoscopic stents are; shorter procedure time, less time to ingestion, and shorter hospitalization periods, but repeated procedures are often required due to frequent stent failures[65-67]. According to one systematic study, patients treated with enteral stents showed shorter hospitalization periods (average 12 d) and faster oral intake (average 7 d) than those treated with gastrojejunostomy, and there was no significant difference in mortality, overall complications, and survival rates[68]. Most studies have shown that there is no difference between both treatments in technical or clinical success rate of the procedure, but one meta-analysis reported that the success rate was higher in stent placement patients[69,70]. There was no difference in the frequency of mild and severe complications in the early stage of complications after SEMS insertion or surgery, but it is known that the time of severe complications in the late stage is relatively earlier and more common in stent patients[71]. Nevertheless, there was no difference in stent insertion or surgery-related mortality[72]. In larger randomized trials with longer follow-up, late complications including recurrent obstruction and need for reoperation were more common in SEMS than gastrojejunostomy, which confirms the previous retrospective study, which reported that gastrojejunostomy surgery has more benefits and is associated with a longer life expectancy[70,73,74].

Endoscopic ultrasound-guided gastroenterostomy

Endoscopic ultrasound-guided gastroenterostomy (EUS-GE) using lumen-apposing metal stents has emerged as a safe and effective alternative method. EUS-GE can allow sustained palliation of surgical bypass while maintaining a minimally invasive endoscopic approach[75,76]. EUS-GE was first described by Binmoeller *et al*[77] in 2012, and has shown significant efficacy in palliating malignant GOO in patients who are suitable for surgical bypass[78]. In EUS-GE, a bypass is created by inserting a lumen-apposing metal stent from the stomach to the small bowel distal to the obstruction under EUS and fluoroscopic guidance.

EUS-GE can be used for palliative management of malignant GOO and can be a treatment option for benign GOO. Two recent case studies showed high technical (90%-92%) and clinical (85%-92%) success rates, with a variable percentage of adverse events (0-11.5%)[79,80]. Tyberg *et al*[79] showed there were fewer side effects (12% *vs* 41%) and similar technical success (88% *vs* 100%) with EUS-GE compared to surgical laparoscopic gastrojejunostomy. A retrospective study in 2020 by James *et al*[81] reported EUS-GE as a bridge therapy for definitive treatment of benign gastric outlet obstruction. EUS-GE was performed in 22 patients with benign GOO, and 83.3% of patients were prevented from surgery. Lumen-apposing metal stents was maintained for a mean of 8.5 mo until GOO was resolved, and the low recurrence rate of GOO (5.6%) has been reported after lumen-apposing metal stents removal. Future prospective, large-scale, randomized studies comparing surgical gastroenterostomy and EUS-GE are needed.

SURGERY

Surgery is the preferred method of treatment in patients with refractory GOO, or for whom endoscopic treatment has not been indicated. In the past, open gastrojejunostomy was widely performed, but recently laparoscopic gastrojejunostomy has become the main treatment. The laparoscopic surgical approach is more effective than open surgery for rapid postoperative recovery and is associated with a shorter hospital stay[82].

CONCLUSION

With the eradication of *H. pylori* and the use of proton pump inhibitors, the predominant causes of GOO have changed from benign to malignant diseases. Treatment of GOO depends on the underlying cause, and multiple treatment methods exist, including both endoscopic and surgical approaches. Therefore, determining the appropriate treatment for individual patients is important for treatment success and prognosis.

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