Gastric outlet obstruction in adults

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INTRODUCTION

Gastric outlet obstruction (GOO) is a clinical syndrome characterized by epigastric abdominal pain and postprandial vomiting due to mechanical obstruction. The term gastric outlet obstruction is a misnomer since many cases are not due to isolated gastric pathology, but rather involve duodenal or extraluminal disease. This topic will review the evaluation and management of adults with GOO. The use of enteral stents to treat malignant GOO are discussed in detail separately. (See "Enteral stents for the palliation of malignant gastroduodenal obstruction".)

EPIDEMIOLOGY

Precise estimates on the incidence of GOO are lacking [1]. Rates of surgery for GOO are likely to have declined over time because of the decline in peptic ulcer disease, which has historically been an important cause of GOO, and with the use of endoscopic means of management of GOO.

ETIOLOGY

The etiology of GOO has changed over the past several decades. Benign disease was responsible for the majority of cases of GOO in adults until the late 1970s, of which peptic ulcer

disease accounted for up to 90 percent of cases [2-6]. With the decline in peptic ulcer disease, it is estimated that 50 to 80 percent of all cases of GOO are attributable to malignancy [3,5-8]. The underlying etiology of benign GOO likely varies geographically, with peptic ulcer disease and caustic injury being more common in some countries than others [9,10].

Neoplastic

Malignant — Pancreatic adenocarcinoma with invasion into the duodenum or stomach is a common cause of malignant GOO [11]. Fifteen to 25 percent of patients with pancreatic cancer may develop GOO during their course [11]. Such patients also commonly have biliary obstruction [12-15]. (See "Clinical manifestations, diagnosis, and staging of exocrine pancreatic cancer".)

Distal gastric cancer remains a relatively common cause of malignant GOO, accounting for up to 35 percent of GOO [16]. However, the absolute number of cases has probably declined because of the decreased incidence of gastric cancer in developed nations and the increase in the proportion of gastric cancers arising from a proximal location. (See "Clinical features, diagnosis, and staging of gastric cancer" and "Epidemiology of gastric cancer".)

Other infrequent causes of malignant GOO include:

- Gastric lymphoma (see "Clinical presentation and diagnosis of primary gastrointestinal lymphomas")
- Large neoplasms of the proximal duodenum and ampulla (see "Epidemiology, clinical features, and types of small bowel neoplasms")
- Local extension of advanced gallbladder carcinoma or cholangiocarcinoma (see "Gallbladder cancer: Epidemiology, risk factors, clinical features, and diagnosis")
- Metastatic or primary malignancy in the duodenum (see "Epidemiology, clinical features, and types of small bowel neoplasms")
- Gastric carcinoid (see "Clinical characteristics of well-differentiated neuroendocrine (carcinoid) tumors arising in the gastrointestinal and genitourinary tracts")
- Gastrointestinal stromal tumors/gastric leiomyosarcomas (see "Clinical presentation, diagnosis, and prognosis of gastrointestinal stromal tumors", section on 'Clinical presentation')

Benign — Prolapse of a large antral polyp across the pylorus can rarely lead to GOO. Such polyps tend to be large (2 to 10 cm in size) and pedunculated [17-24]. (See "Gastric polyps",

section on 'Clinical and pathologic features' and "Gastric polyps", section on 'Clinical and pathologic features'.)

Inflammatory

Peptic ulcer disease — Both acute and chronic peptic ulcer disease can lead to GOO. The principal sites of involvement in cases of obstruction are the pyloric channel and the duodenal bulb. Acute peptic ulcers can cause obstruction via inflammation-induced edema and tissue deformation. By contrast, chronic peptic ulcer disease leads to scarring and tissue remodeling as part of the healing process. However, obstruction is the least common complication of peptic ulcer disease, occurring in approximately 2 percent of cases [25]. Even in patients with Zollinger-Ellison syndrome, 10 percent of whom develop duodenal or pyloric strictures, GOO occurs rarely [26]. (See "Overview of complications of peptic ulcer disease", section on 'Gastric outlet obstruction' and "Zollinger-Ellison syndrome (gastrinoma): Clinical manifestations and diagnosis", section on 'Clinical presentation'.)

Pancreatitis — Chronic pancreatitis and, to a lesser extent, severe acute pancreatitis with involvement of the duodenum can cause duodenal obstruction. The obstruction may be functional or both structural and functional in etiology. Case series report an incidence of approximately 1 to 4 percent [27-32].

The pathogenesis of GOO in chronic pancreatitis is incompletely understood. One theory is that the obstruction develops from weeping of inflammatory exudates from the anterior pancreatic surface, which leads to inflammation and fibrosis in adjacent structures, most commonly the duodenum, jejunum, and transverse colon [33]. Patients with duodenal obstruction often have associated pancreatic and biliary duct strictures [27]. Segmental chronic pancreatitis within the area between the pancreas and the duodenum has been termed "groove pancreatitis." Both duodenal and biliary obstruction can occur, and the disease is often misdiagnosed as pancreatic malignancy [30].

Patients with severe acute pancreatitis can develop narrowing of the duodenum due to inflammation and edema in the pancreas. However, obstruction of the duodenum is rare [31,34], except in cases of walled-off pancreatic necrosis after severe acute pancreatitis and large pseudocysts that can occur as a result of both acute and chronic pancreatitis. (See "Approach to walled-off pancreatic fluid collections in adults" and "Overview of the complications of chronic pancreatitis".)

Caustic injury — GOO secondary to caustic ingestion occurs due to fibrosis after resolution of the acute injury and inflammation, most commonly 6 to 12 weeks after initial ingestion [35]. (See "Caustic esophageal injury in adults".)

Gastric injury to acid exposure can be similarly severe. In a prospective study of 41 patients who ingested corrosive acids, 84 percent developed acute gastric injury (typically severe, grade 2 or 3) [36]. Chronic sequelae (stricture, distal gastric deformity) were noted in 44 percent.

Infiltrative disease

Crohn disease — Clinically significant gastroduodenal Crohn disease is uncommon, occurring in fewer than 5 percent of patients. When present, approximately 60 percent of patients have continuous disease that involves the antrum, pylorus, and proximal duodenum [37]. The majority have concomitant disease in the distal gastrointestinal tract [2,33]. (See "Clinical manifestations, diagnosis, and prognosis of Crohn disease in adults".)

Most patients with gastroduodenal Crohn disease do not have symptoms attributable to the gastroduodenal involvement, and symptoms are usually synchronous with active disease elsewhere in the gastrointestinal tract, most commonly ileocolic involvement [38,39]. Obstruction due to Crohn-related strictures is the most common complication of gastroduodenal disease. Patients may present with upper abdominal pain, nausea, and/or postprandial vomiting. (See "Clinical manifestations, diagnosis, and prognosis of Crohn disease in adults", section on 'Other gastrointestinal features'.)

Gastric tuberculosis — Gastroduodenal disease occurs in only 0.3 to 2.3 percent of patients with tuberculosis (TB). Clinically it may resemble peptic ulcer disease or malignancy [40-46]. GOO is the most common complication of gastroduodenal TB; in one report, for example, GOO was the presenting feature in 61 percent of 23 patients with biopsy-proven gastroduodenal TB [23]. The obstruction may be secondary to infiltration of the gastric antrum or duodenum or to extrinsic compression by adenopathy or a phlegmon [41]. (See "Abdominal tuberculosis".)

Other — GOO has been described in association with infiltrative diseases, including eosinophilic gastroenteritis with disease of the muscularis, chronic granulomatous disease, and gastroduodenal amyloidosis [47-51]. (See "Eosinophilic gastrointestinal diseases", section on 'Clinical manifestations' and "Granulomatous gastritis", section on 'Clinical features' and "Gastrointestinal amyloidosis: Clinical manifestations, diagnosis, and management", section on 'Gastrointestinal tract amyloidosis'.)

Iatrogenic — GOO is a rare complication of PEG tube placement. It is typically caused by migration of the tube into the pyloric channel or duodenal bulb [52-56]. Risk factors include absence of an external bolster (such as in the case of Foley catheters) and placement of the tube close to the pylorus [53,54,57]. (See "Gastrostomy tubes: Complications and their management".)

GOO can also occur in the setting of a preserved or partially preserved stomach such as sleeve gastrectomy, placement of an intragastric balloon, pylorus-preserving Whipple procedure, and gastrojejunostomies [58]. Complications of bariatric surgery are reviewed separately.

Other rare causes

- **Bouveret syndrome** – Bouveret syndrome is characterized by the impaction of a large gallstone (a single stone in >90 percent) within the pyloric channel or, more commonly, the duodenum [59-63]. It occurs most commonly in older women. The offending stone travels from the biliary tree via a biliary enteric fistula, formed in the setting of cholecystitis and pericholecystic inflammation. Prolonged obstruction occurs in only 15 percent of cases after passage of a stone into the duodenum. (See "Gallstone ileus".)

- **Annular pancreas** – Annular pancreas is a congenital condition in which the second part of the duodenum is surrounded by an abnormal ring of pancreatic tissue, potentially leading to GOO in adults [64]. (See "Annular pancreas", section on 'Clinical manifestations'.)

- **Intramural hematomas** – Intramural intestinal hematomas may develop spontaneously in patients receiving anticoagulation or with bleeding disorders, or may be seen following abdominal trauma [65-67].

- **Gastric bezoar** – A bezoar is a concretion formed by the gradual accumulation of ingested material, both inorganic and organic, most often in the gastric lumen. While GOO from bezoars has been documented in adults, the majority of cases of obstruction have been reported in children [68-71]. (See "Gastric bezoars", section on 'Clinical manifestations'.)

- **Gastric volvulus** – Gastric volvulus, defined as an abnormal rotation of the stomach, is a rare entity that is seen most commonly in adults over the age of fifty. Both acute and chronic gastric volvulus can cause GOO. Two etiologic factors predominate: diaphragmatic defects such as a paraesophageal hernia or diaphragmatic hernia of traumatic origin, and poor fixation of the stomach due to laxity or absence of the gastrosplenic or gastrocolic ligaments. Adhesive disease from previous surgery may serve as an axis for gastric rotation. (See "Gastric volvulus in adults", section on 'Clinical presentations'.)

CLINICAL MANIFESTATIONS

Clinical presentation — The most common clinical features of GOO include:

- Epigastric pain
- Nausea and/or vomiting

- Early satiety
- Abdominal distension or bloating
- Weight loss

In one series of 49 patients, the most common clinical features were epigastric pain (94 percent), vomiting (92 percent), and weight loss (63 percent) [72]. The majority of patients (78 percent) had malignant disease. In another cohort of 30 patients with benign causes of GOO, the most common presenting features were early satiety (53 percent) and bloating (50 percent) [72].

The onset of symptoms of GOO varies depending upon the etiology of the obstruction. Symptoms generally occur abruptly with gallstone impaction, prolapse of a large gastric polyp, percutaneous endoscopic gastrostomy tube migration, and gastric volvulus. Other causes tend to follow a more indolent course. Patients with malignant disease may have a shorter duration of symptoms compared with those with benign disease [7]. Weight loss may be seen in patients with GOO due to a malignancy, tuberculosis, or a benign but chronic cause of obstruction. (See "Gallstone ileus", section on 'Introduction'.)

Physical examination — Physical examination may reveal signs of malnutrition or volume depletion. A succussion splash is suggestive of GOO, but has a low sensitivity (48 percent) [73]. A succussion splash is elicited with the stethoscope rested over the upper abdomen, and the patient is rocked back and forth at the hips. Auscultation of a "splash" is reflective of retained gastric material if noted more than three hours after a meal; a left supraclavicular lymph node (Virchow's node) or periumbilical lymph node (Sister Mary Joseph's node) may be seen in metastatic gastric cancer. A palpable abdominal mass is noted in a minority of patients.

Laboratory findings — Laboratory tests may be normal. Patients with recurrent vomiting may have electrolyte abnormalities, including hypokalemia or a hypochloremic metabolic alkalosis. Anemia may be seen in patients with peptic ulcer disease, primary or metastatic malignant disease, or large gastric polyps. (See "Peptic ulcer disease: Clinical manifestations and diagnosis", section on 'Bleeding' and "Gastric polyps", section on 'Clinical and pathologic features' and "Causes of metabolic alkalosis".)

Although not part of routine laboratory testing, elevated serum gastrin levels may be present, likely due to distention-induced gastrin release [74]. Serum gastrin levels in the 400 to 800 pg/mL range have been reported, potentially leading to confusion with Zollinger-Ellison syndrome. (See "Zollinger-Ellison syndrome (gastrinoma): Clinical manifestations and diagnosis", section on 'Serum gastrin concentration'.)

EVALUATION

GOO is suspected in patients with nausea and/or vomiting, epigastric pain, early satiety, and abdominal distension and is diagnosed on abdominal imaging (gastric distention along with retained material within the gastric lumen and an associated air-fluid level) and/or upper endoscopy that demonstrate luminal obstruction. Although history (timing of onset, duration of symptoms, associated weight loss), physical examination findings, and abdominal imaging and/or upper endoscopy may suggest an etiology, additional testing (eg, endoscopic ultrasound-guided biopsy or full-thickness surgical biopsies) may be needed in patients with a suspected malignancy or gastroduodenal tuberculosis. Radiologic testing should precede endoscopic evaluation.

Imaging

Computed tomography scan — Findings on abdominal computed tomography (CT) scan include gastric distention, along with retained material within the gastric lumen and an associated air-fluid level (image 1) [75]. CT will often also suggest the specific cause of GOO. CT is also sensitive for detecting small amounts of free air, gastric pneumatosis, or free fluid in the abdomen. In patients with suspected GOO in whom oral water-soluble contrast is required to further delineate the obstruction, the stomach should be decompressed with a nasogastric tube before in order to minimize risks of aspiration. (See 'Etiology' above.)

Other imaging — Water-soluble contrast or barium studies can be useful if a partial obstruction is expected. Failure of any contrast to pass into the small bowel suggests complete GOO. Contrast studies may give clues to the underlying etiology such as peptic ulcer disease, Crohn disease, gastric bezoars, or gastric volvulus, although in most cases the findings are nonspecific.

Abdominal radiographs are not diagnostic for GOO. Plain films of the abdomen may reveal an enlarged gastric bubble and a dilated proximal duodenum. A paucity of air in the small bowel is often noted. In a minority of patients, the plain film can suggest an underlying cause such as a gallstone or calcification suggestive of chronic pancreatitis [76]. Rarely, a calcified mass in the right upper quadrant may be seen in patients with Bouveret syndrome [77]. (See 'Imaging' above and 'Upper gastrointestinal endoscopy' below and "Gallstone ileus", section on 'Plain radiography'.)

Upper gastrointestinal endoscopy — In addition to confirming the diagnosis of GOO, upper endoscopy may identify the etiology, and allow for a therapeutic intervention such as dilation/stent placement to be performed (picture 1).

Nasogastric tube suction is recommended before endoscopy to minimize retained fluid that may increase the risk of aspiration during endoscopy. Large-bore tubes (eg, Ewald tubes) can achieve better clearance than standard smaller-bore tubes. Patients should fast for at least four hours or until returns from nasogastric suction begin to slow and clinical signs of decompression are evident. Fasting for prolonged periods is not necessary and may increase the amount of retained fluid if concurrent nasogastric tube suction is not performed since the stomach continues to produce significant amounts of secretions during fasting.

Endoscopic features suggestive of GOO include luminal narrowing of the stomach or duodenum. Based on the etiology, an intraluminal cause of obstruction (eg, stenosis or stricture, polyp) or extraluminal compression may be identified.

Passage of a standard adult endoscope across the stricture may not be possible if there is complete or near-complete obstruction. Gentle attempts at passage through the stricture can be made but are often unnecessary for a definitive diagnosis. Small-diameter scopes can also be used to navigate tight strictures. If endoscopic therapy is indicated/planned/performed, contrast injection with fluoroscopy can be helpful in delineating the stricture. Biliary retrieval balloons can be used to both define the distal extent of the stricture, but also to promote flow of contrast through the stricture to allow for fluoroscopic determination of the length.

Upper endoscopy has diagnostic limitations. In the setting of Bouveret syndrome, the offending stone may not be visualized due to mucosa overlying the embedded stone [73]. Routine biopsy techniques can also have poor sensitivity if the tumor is extraluminal or does not involve the mucosa [5,6,75].

Patients with a suspected underlying malignancy (eg, >50 years of age, no history of peptic ulcer disease, family history of gastric cancer) or gastroduodenal tuberculosis should undergo additional tunneled endoscopic biopsies or endoscopic ultrasound-guided biopsies. If these are nondiagnostic, full-thickness surgical biopsies may be needed.

MANAGEMENT

Supportive care in all patients

- Patients with GOO and symptoms of nausea/vomiting should receive nothing by mouth.
- For patients with moderate to severe or continuous vomiting, or significant abdominal distention, a nasogastric tube should be placed for gastric decompression. Large-bore nasogastric tubes can improve gastric clearance.

- Intravenous fluids should be administered to maintain normovolemia and electrolytes repleted as necessary. Replacement intravenous fluids should be administered when there is a large volume of emesis or drainage from a nasogastric tube (one or more liters per day).

- High-dose proton pump inhibitors (PPIs) should be administered regardless of the cause of GOO to decrease the volume of gastric secretions and reduce associated inflammation [78,79].

- Nutritional support with parenteral nutrition may be needed if definitive therapy is not imminent or for presurgical optimization of nutritional status. (See "Overview of complications of peptic ulcer disease", section on 'Gastric outlet obstruction'.)

  Response to conservative management can be assessed by a challenge with a liquid diet or a gastrografin contrast study with a nasogastric tube in place. Failure to tolerate a trial of liquids or failure of contrast to pass into the distal duodenum usually necessitates intervention.

Endoscopic therapy for benign GOO

- **Balloon dilation technique** – Dilation can be accomplished by using endoscopy and a through-the-scope (TTS) balloon dilator, or by using a balloon placed over a guidewire positioned under fluoroscopic guidance. While fluoroscopy may not be required for dilation using a TTS balloon in the case of mild strictures (eg, strictures that allow passage of an endoscope), most other stricture dilations are carried out using fluoroscopic guidance. The amount of dilation in a single session is determined by the initial diameter of the stricture and the presence of active ulceration. Narrow strictures may require stepwise dilation performed over multiple sessions, as is done with esophageal strictures. (See "Endoscopic interventions for nonmalignant esophageal strictures in adults", section on 'Procedure'.)

  If the pyloric channel can be identified and a balloon can be passed, dilation is an appropriate option. The balloon is inflated to the chosen diameter and inflation is sustained for 30 to 60 seconds and monitored by a pressure gauge. The dilation may then be repeated at the same diameter, though it is not certain that repeated dilations to the same diameter yield better results. Using a dilute contrast medium to fill the balloon allows progress to be followed fluoroscopically; a waist forms initially during dilation but is effaced during balloon distention. Successful dilation is usually confirmed by pulling the balloon through the strictured segment, although failure to accomplish this does not preclude a successful procedure. Symptoms are usually considerably improved with

successful dilation to 12 to 15 mm. Dilation beyond 15 mm can be performed but with notable caution, as higher rates of perforation, between 2 to 6 percent, occur with dilation beyond 15 mm. This must be weighed against a lower rate of clinical efficacy with smaller-diameter dilation. Each patient must be re-evaluated after each dilation for symptomatic improvement [25,80-82].

The frequency of repeated dilation sessions depends upon the success of initial dilation and the patient's past response to dilation. Patients undergoing dilation for the first time may require multiple sessions, especially if the gastric/duodenal lumen has a narrow diameter and exhibits significant resistance during dilation. Such patients may require repeated sessions every 5 to 14 days.

Long or fibrotic duodenal strictures, such as those encountered in chronic pancreatitis, caustic injury, or Crohn disease are the most difficult to dilate. Multiple sessions using stepwise dilation for tight obstructions will likely lower the risk of perforation. Nevertheless, because of the risk of perforation, patients should be appropriately prepared for surgery before duodenal dilation and monitored closely after dilation before resuming oral intake. If there is concern about a possible perforation, or if the dilation is unusually difficult to perform, an immediate postprocedure water-soluble contrast study should be obtained either by contrast instillation by the endoscopist or more commonly, a CT with oral contrast.

- **Efficacy** – Multiple sessions of endoscopic balloon dilation are necessary in some cases [25,80,83,84]. In one study, for example, 24 of 30 patients (80 percent) achieved sustained symptom relief; 17 had a single procedure, while seven required multiple sessions [25]. Dilation failed in four patients with long duodenal strictures, while two dilated to 18 mm suffered perforation; both recovered uneventfully after surgery. However, a durable response can be obtained using large-diameter TTS balloon dilation, careful removal of inciting factors (nonsteroidal anti-inflammatory drugs [NSAIDs]/aspirin avoidance and eradication of *Helicobacter pylori)*, and acid suppression [80,84]. In another large study of 264 patients, 92 percent had resolution of symptoms and major complication rates were relatively low (7.7 percent hemorrhage, 9.1 percent pain, and 3.4 percent perforation) [9].

- **Metal stents** – Covered or partially covered self-expanding metal stents (SEMS) have been used successfully to treat benign gastroduodenal strictures in both case reports and small series [85,86].

  In a retrospective study that examined use of partially covered SEMS for 10 benign strictures, all due to peptic ulcer disease, SEMS were associated with 100 percent technical

and clinical success, but had a 20 percent risk of stent migration [87]. In second Korean study, 22 patients were treated with 18 or 20 mm diameter SEMS, 21 with strictures secondary to peptic ulcer disease. There was symptom improvement in 82 percent of patients as measured by a GOO symptom score (GOOSS) in the short term, but only 67 percent at six weeks, and stent migration was observed in 69 percent of patients [88].

Off-label use of lumen-apposing metal stents (LAMS) has been reported for treatment of short, benign gastrointestinal strictures. The results have been variable, with variable rates of stent migration and complications [89-92].

Surgery in selected patients — Surgery is indicated if the pylorus is obstructed and cannot be safely dilated, or if the obstruction persists or recurs despite endoscopic dilations to achieve a 15 mm diameter. A retrospective study found that factors predictive of surgery included need for >3 procedures and duration of treatment beyond one year [80]. Surgical management of GOO is discussed elsewhere. (See "Surgical management of peptic ulcer disease", section on 'Gastric outlet obstruction'.)

Specific management of obstruction by etiology

Peptic ulcer disease — Initial management consists of acid suppression with a PPI, avoidance of NSAIDs, and, if present, eradication of *H. pylori* infection. Although case series have demonstrated resolution of obstruction with *H. pylori* eradication alone, treatment responses vary [93-96]. Recurrence of obstruction appears to be reduced in cases of *H. pylori* infection that are successfully eradicated. Endoscopic dilation of ulcer-related strictures appears to be less successful in patients who are *H. pylori* negative at time of diagnosis, and surgical intervention appears to be more likely [1]. (See "Peptic ulcer disease: Treatment and secondary prevention", section on 'Initial management'.)

In patients with peptic ulcer disease who fail to respond to a brief (three to seven day) trial of conservative management, we suggest endoscopic dilation. SEMS have been used for the treatment of peptic ulcer-related GOO [87]. We reserve their use for patients with strictures refractory to endoscopic balloon dilation who are not surgical candidates. (See 'Endoscopic therapy for benign GOO' above.)

Chronic pancreatitis — Patients with GOO from fibrosis in the pancreas and surrounding tissues due to chronic pancreatitis are unlikely to respond to balloon dilation and usually require gastric bypass with gastrojejunostomy. GOO is also a rare complication of pseudocysts, which are often amenable to endoscopic drainage techniques. (See "Approach to walled-off pancreatic fluid collections in adults" and "Overview of the complications of chronic pancreatitis", section on 'Biliary obstruction'.)

Acute pancreatic fluid collections — Several options exist for management of GOO secondary to a large pancreatic pseudocyst: CT-guided percutaneous drain placement, transpapillary endoscopic drainage, endoscopic cyst gastrostomy or duodenostomy, and surgical internal drainage. The feasibility of endoscopic management depends upon the availability of the appropriate endoscopic expertise and characteristics of the pseudocyst itself. A mature cyst wall and apposition of the cyst and the gastric or duodenal wall are required for endoscopic therapy. (See "Approach to walled-off pancreatic fluid collections in adults" and "Endoscopic interventions for walled-off pancreatic fluid collections".)

Crohn disease — Options for management of GOO due to Crohn disease include medical, endoscopic, and surgical approaches. Experience with any of these approaches is limited. Corticosteroids can be used to treat the inflammatory component, but are typically ineffective in the setting of obstruction due to the presence of underlying fibrotic disease. Successful medical therapy for Crohn-related duodenal strictures with biologic medications has been described in case reports [97]. (See "Medical management of moderate to severe Crohn disease in adults", section on 'Gastroduodenal disease' and "Overview of the medical management of mild (low risk) Crohn disease in adults", section on 'Gastroduodenal disease'.)

Endoscopic balloon dilation of Crohn-related strictures can be attempted, but is often followed by symptomatic recurrence requiring repeated therapy; multiple dilations are often necessary. In an illustrative study of nine patients with GOO due to Crohn disease, initial symptom relief was achieved with multiple endoscopic dilations (up to 10) and medical treatment with acid suppression [37]. Almost all of the patients experienced recurrent symptoms during a mean follow-up of eight years. However, repeated sessions were effective in all patients. Two patients eventually underwent surgery for frequent symptomatic recurrence; both had long stenotic segments. Another study found that short strictures required fewer dilation sessions [98].

Historically up to 40 percent of patients with gastroduodenal Crohn disease will require surgery, most commonly due to GOO, but advances in medical therapy suggest that biologics may be effective for treatment of Crohn-related duodenal strictures obviating the need for surgery [2,26]. In a series of 89 patients with gastroduodenal Crohn disease, 33 patients (37 percent) required surgery, with 23 due to gastroduodenal obstruction. At one year, good to excellent results were achieved in 87 percent. Surgical options include gastrojejunostomy, gastroduodenostomy, and stricturoplasty. (See "Surgical management of Crohn disease", section on 'Surgical techniques'.)

Caustic ingestion — The traditional treatment for GOO related to ingestion of corrosive agents has been surgery [99]. Temporary interventions during the subacute phase may include a venting gastrostomy or feeding jejunostomy. Definitive treatments include subtotal

gastrectomy with a Billroth I or Billroth II anastomosis (figure 1) or, less commonly, pyloroplasty. Early definitive therapy may improve quality of life and avoid the need for a second operation [100]. Experience with endoscopic balloon dilation of corrosive-induced GOO is limited but suggests that it may be a viable alternative to surgery [101-103]. In a series of 41 patients with caustic-induced GOO, balloon dilation was successful in 39 patients over an average follow-up of 35 months (range 18 to 58 months) [102]. Patients required a mean of six dilations to achieve an endpoint of 15 mm. One patient required surgery for a perforation and another developed pain with each dilation and ultimately required surgery. However, caustic strictures required a greater number of dilations to achieve clinical success as compared with non-caustic strictures [9].

Bouveret syndrome — Bouveret syndrome (impaction of a large gallstone within the pyloric channel) usually requires surgery to remove the impacted stone, repair the fistula, and remove the gallbladder. For patients who are unable or unwilling to undergo surgery, options include endoscopic removal of the obstructing stone and electrohydraulic lithotripsy of obstructing stones. (See "Gallstone ileus", section on 'Treatment'.)

Large gastric polyps — GOO due to large gastric polyps can usually be treated by endoscopic resection. (See "Gastric polyps" and "Endoscopic removal of large colon polyps", section on 'Polyp removal techniques'.)

Gastric bezoars — The choice of therapy (chemical dissolution, endoscopic therapy, or surgery) for gastric bezoars depends on the type of bezoar and the severity of symptoms and is discussed in detail separately. (See "Gastric bezoars", section on 'Management'.)

Malignant obstruction — Treatment for GOO due to malignancy depends upon the underlying cause. Options for the palliation of locally advanced pancreatic cancer with duodenal obstruction, as well as obstructing gastric adenocarcinomas, include endoscopic or surgical bypass through a gastrojejunostomy or placement of an endoscopic enteral stent. Therapeutic options to control symptoms of local disease progression include palliative surgical resection, surgical bypass (gastrojejunostomy), radiation therapy, endoscopic stenting, and palliative decompressive gastrostomy. Endoscopic ultrasound-guided gastroenterostomy appears to be a promising means of endoscopic bypass. (See "Enteral stents for the palliation of malignant gastroduodenal obstruction" and "Supportive care of the patient with locally advanced or metastatic exocrine pancreatic cancer" and "Initial systemic therapy for locally advanced unresectable and metastatic esophageal and gastric cancer" and "Therapeutic endoscopic ultrasound".)

For patients who have a gastrointestinal tract lymphoma causing GOO, chemotherapy is usually the preferred initial treatment. (See "Treatment of extranodal marginal zone lymphoma of mucosa associated lymphoid tissue (MALT lymphoma)".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Upper endoscopy (The Basics)")
- Beyond the Basics topics (see "Patient education: Upper endoscopy (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- Gastric outlet obstruction (GOO) is a clinical syndrome characterized by epigastric abdominal pain and postprandial vomiting due to mechanical obstruction.
- The incidence of GOO is not known precisely, but is likely to have declined because of the decrease in peptic ulcer disease, which has historically been an important cause of GOO. With the decline in peptic ulcer disease, it is estimated that 50 to 80 percent of all cases of GOO are attributable to malignancy. Pancreatic adenocarcinoma with extension to the duodenum or stomach is a common cause of malignant GOO. (See 'Epidemiology' above and 'Etiology' above.)
- The most common clinical features are nausea and/or vomiting, epigastric pain, early satiety, abdominal distension, and weight loss. Symptoms generally occur abruptly with gallstone impaction, prolapse of a large gastric polyp, percutaneous endoscopic

gastrostomy tube migration, and gastric volvulus. Other causes tend to follow a more indolent course. Weight loss can be seen in patients with malignant causes, tuberculosis, or those who develop malnutrition from chronic obstruction. (See 'Clinical manifestations' above.)

- The diagnosis may be suspected based upon presenting clinical features and physical examination and is confirmed by radiologic evaluation and/or endoscopy (picture 1). (See 'Evaluation' above.)

- Patients who are suspected of having GOO should take nothing by mouth, receive adequate fluid and electrolyte replacement, and have a nasogastric tube placed for gastric decompression. We suggest all patients be treated with a proton pump inhibitor (**Grade 2C**). Definitive treatment of the obstruction should be based upon the underlying etiology. (See 'Management' above.)

- The treatment of GOO is specific to the underlying etiology:

  - Patients with malignancy typically require placement of a self-expanding metal stent or a surgical bypass. This is discussed in detail separately. (See "Enteral stents for the palliation of malignant gastroduodenal obstruction" and "Supportive care of the patient with locally advanced or metastatic exocrine pancreatic cancer".)

  - GOO in the setting of peptic ulcer disease or Crohn disease may resolve with treatment of the underlying disorder.

    For patients with persistent GOO despite medical management we suggest endoscopic balloon dilation (**Grade 2C**).

  - Patients with GOO due to caustic ingestion are treated with either surgery or endoscopic balloon dilation depending on the extent of injury and available expertise.

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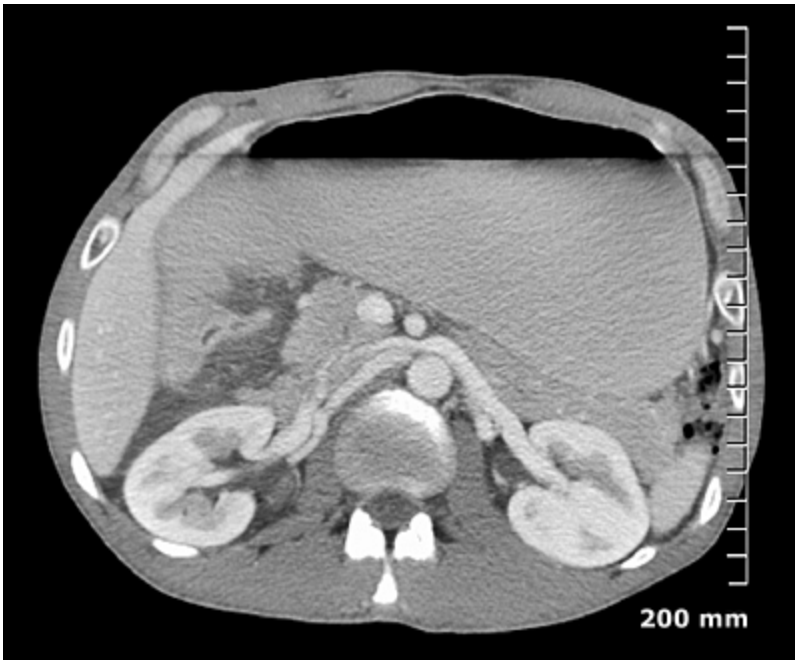
Topic 2621 Version 29.0

GRAPHICS

CT of the abdomen in a patient with gastric outlet obstruction



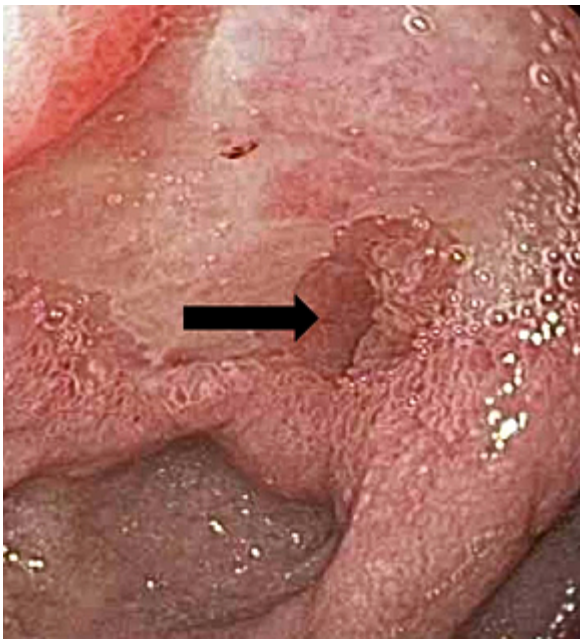
Abdominal CT in a patient with gastric outlet obstruction due to peptic ulcer disease showing a distended and fluid filled stomach.

CT: computed tomography.

Courtesy of Ashley Davidoff, MD.

Graphic 58277 Version 3.0

Gastric outlet obstruction due to peptic ulcer disease

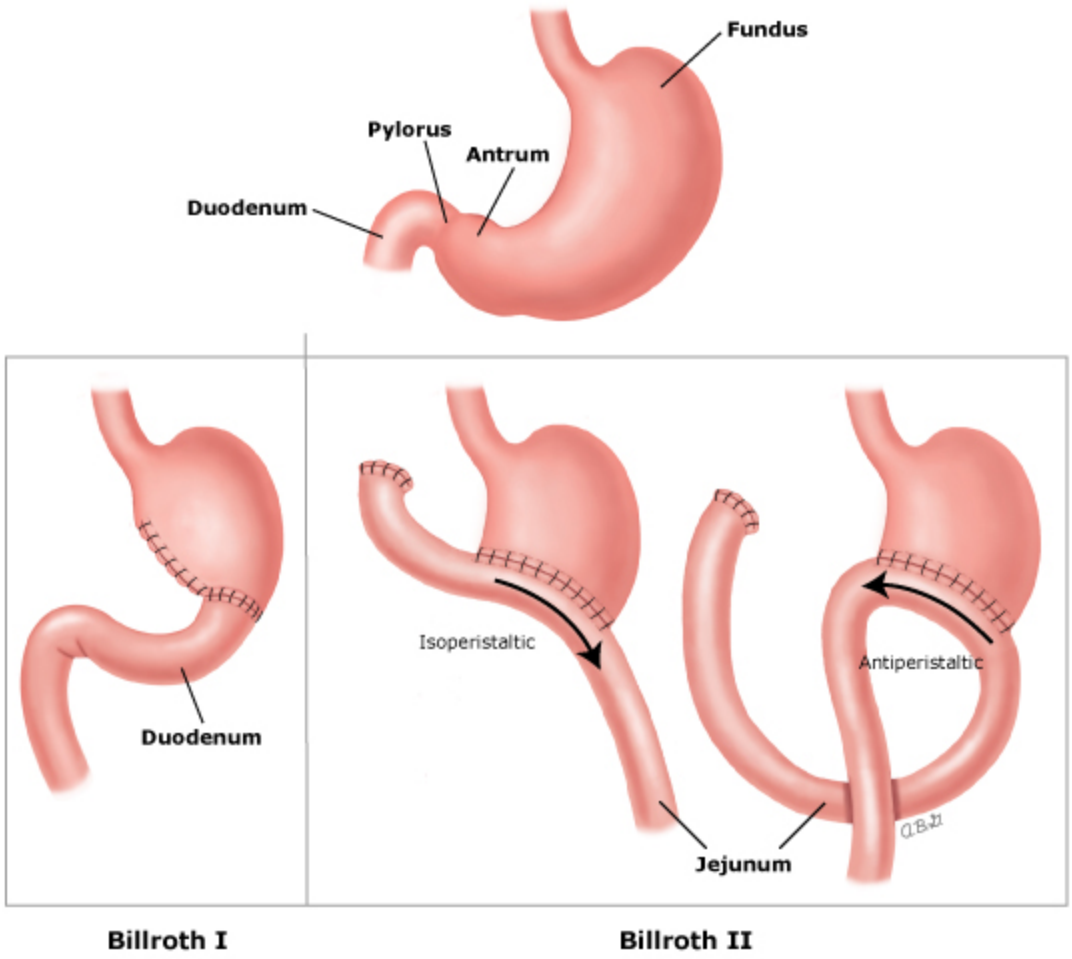


Endoscopic view of the pre-pylorus in a patient with acute on chronic peptic ulcer disease and associated gastric outlet obstruction. The black arrow indicates the narrowed pylorus.

Courtesy of Derek Frederickson, MD.

Graphic 55405 Version 2.0

Billroth reconstruction following gastrectomy



This illustration depicts the Billroth I and Billroth II methods of reconstruction following vagotomy and antrectomy. The Billroth I consists of an end-to-end gastrodoudenal anastomosis; in contrast, the Billroth II consists of an end-to-side gastrojejunal anastomosis.

Modified from: Sedgwick CE. Gastrectomy. In: Atlas of Abdominal Surgery, Braasch JW, Sedgwick CE, Veidenheimer MC, Ellis FH (Eds), WB Saunders Company, Philadelphia 1991. p.33.

Graphic 60974 Version 9.0

Contributor Disclosures

Seng-Ian Gan, MD, FRCP(C) Equity Ownership/Stock Options: Romark Labs [Antiviral]. Consultant/Advisory Boards: Boston Scientific [Therapeutic endoscopy, pancreaticobiliary disease]. All of the relevant financial relationships listed have been mitigated. **John R Saltzman, MD, FACP, FACG, FASGE, AGAF** No relevant financial relationship(s) with ineligible companies to disclose. **Shilpa Grover, MD, MPH, AGAF** No relevant financial relationship(s) with ineligible companies to disclose.

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