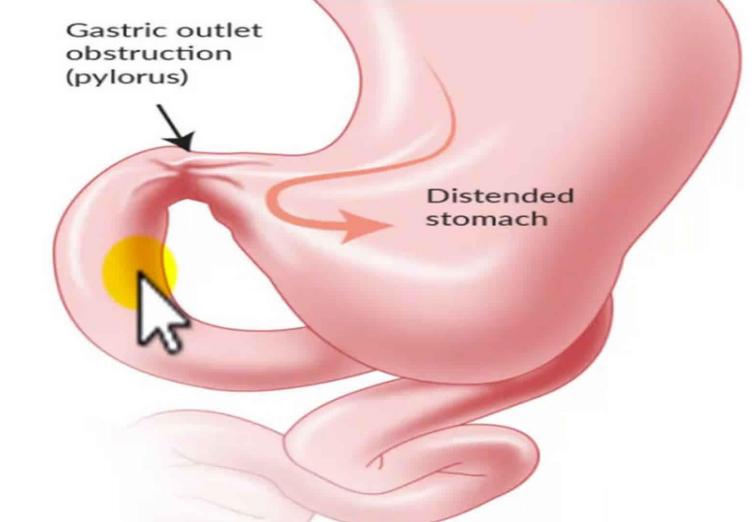
## **APPROACH TO GASTRIC OUTLET OBSTRUCTION**

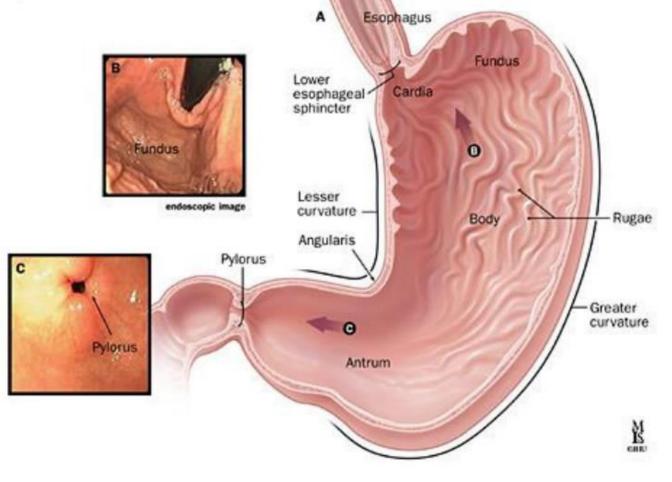


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# DEFINITION

Gastric outlet obstruction (GOO) is a clinical syndrome characterized by epigastric abdominal pain and postprandial vomiting due to mechanical obstruction.

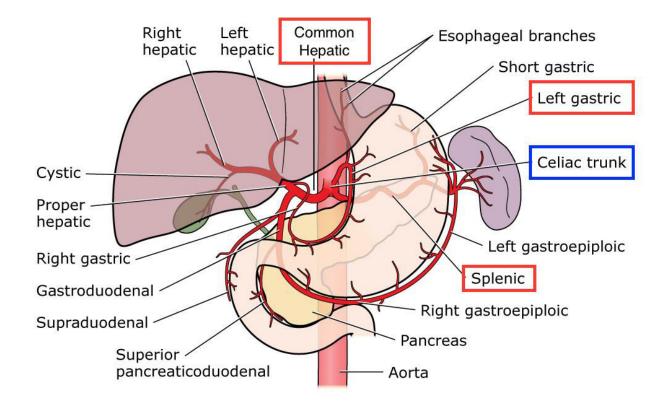
### Anatomy



Inflammation, scarring, or infiltration of the antrum and pylorus are associated with the development of GOO.

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- The stomach is supplied by a rich system of arteries derived from the celiac trunk.
- <u>The lesser curvature</u> of the stomach is supplied by the left and right gastric artery, which are branches of the celiac trunk and the common hepatic artery respectively.
- <u>The greater curvature</u> is supplied by the left and right gastro-omental (gastro-epiploic) arteries, which originate from the splenic and gastroduodenal arteries respectively.
- <u>The fundus and upper part of the</u> <u>body of the stomach</u> is supplied by the short and posterior gastric branches of the splenic artery while <u>the pylorus</u> of the stomach is supplied by the gastroduodenal artery, a branch of the common hepatic artery.



## BACKGROUND

Clinical entities that can result in GOO generally are categorized into a 2 well- defined groups of causes:

- A. Benign
- **B.** Malignant

This classification facilitates discussion of management and treatment.

## **ETIOLOGY**

Major Benign causes of Gastric Outlet Obstruction (GOO) are:

- 1. PUD
- 2. Gastric polyps
- 3. Ingestion of caustics
- 4. Pyloric Stenosis
- 5. Congenital duodenal webs
- 6. Bezoars
- 7. Pancreatic pseudocysts

## **MALIGNANT CAUSES**

- 1. Pancreatic adenocarcinoma with invasion into the duodenum or stomach is a common cause of malignant GOO
- 2. Gastric cancer
- 3. Duodenal cancer
- 4. Ampullary cancer
- 5. Cholangiocarcinoma

## **BENIGN CAUSES**

- 1. Peptic ulcer disease
- 2. Caustic ingestion
- 3. inflammatory diseases such as Crohn's disease
- 4. tuberculosis
- 5. non-steroidal anti-inflammatory drug-induced strictures may also result in GOO.
- 6. Pancreatitis

- Previously, peptic ulcer diseases were a more common cause of GOO.
- Now, with the decrease incidence of peptic ulceration and the advent of potent medical treatments, gastric outlet obstruction should be considered malignant until proven otherwise.
- The need for surgery is thought to have declined because of advancements in endoscopic methods to treat GOO (such as dilation and stenting).

### **EFFECTS OF GASTRIC OUTLET OBSTRUCTION**

- Nausea and vomiting are the cardinal symptoms of gastric outlet obstruction (GOO).
- Vomiting usually is described as non-bilious, and it characteristically contains undigested food particles. In the early stages of obstruction, vomiting may be intermittent and usually occurs within 1 hour of a meal.

### **EFFECTS OF GASTRIC OUTLET OBSTRUCTION**

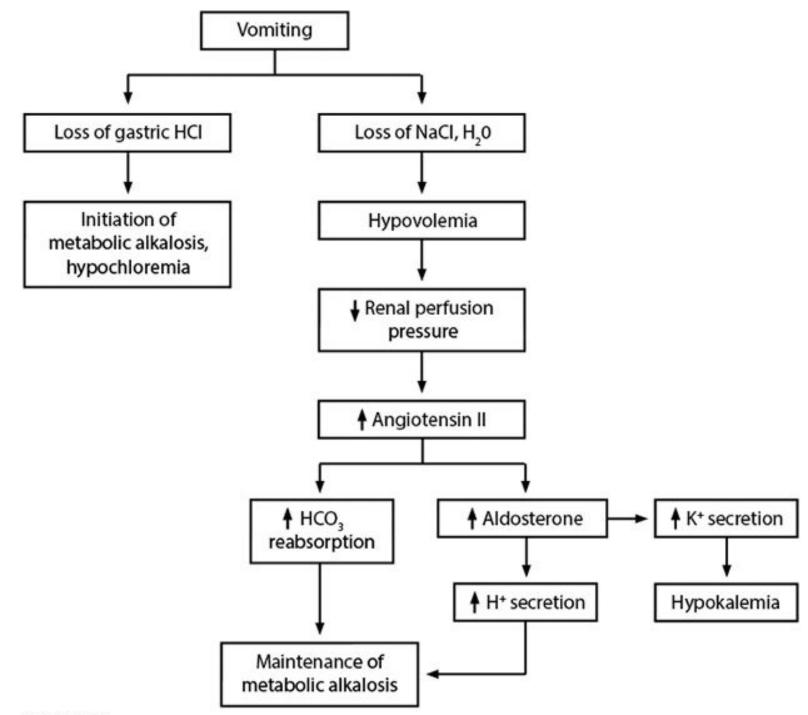
- Patients with GOO resulting from a duodenal ulcer or incomplete obstruction typically present with symptoms of gastric retention, including early satiety, bloating or epigastric fullness, indigestion, anorexia, nausea, vomiting, epigastric pain, and weight loss.
- They are frequently malnourished and dehydrated and have a metabolic insufficiency.

### **EFFECTS OF GASTRIC OUTLET OBSTRUCTION**

- Weight loss is frequent when the condition approaches chronicity and is most significant in patients with malignant disease.
- Abdominal pain is not frequent and usually relates to the underlying cause (eg, peptic ulcer disease [PUD] or pancreatic cancer).

# METABOLIC EFFECT

- Prolonged vomiting causes loss of hydrochloric (HCl) acid and produces a hypochloremic alkalosis
- Initially Na and K levels are normal
- However as dehydration progresses, more profound metabolic abnormalities arise, partly related to renal dysfunction.
- Initially urine has low chloride and high bicarbonate
- HCO3 is excreted with Na, so the patient becomes hyponatremic and more dehydrated
- Because of this a phase of Na retention follows and K and Hydrogen are excreted .....
- This results in urine becoming Paradoxically aciduria and hypokalemia
- Alkalosis leads to a lowering in the circulating ionized calcium and tetany can occur



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### HOW TO APPROACH A PATIENT WITH GASTRIC OUTLET OBSTRUCTION ?

- History
- Physical examination.
- Investigations.
- Treatment

# 1) History

- Age ( reflect the cause )
- Symptoms : Epigastric pain (SOCRATES), Nausea and/or vomiting ( character of vomiting), Early satiety, Abdominal distention or bloating, steatorrhea, jaundice, constitutional symptoms.
- Previous attacks
- Medical and surgical history : History of PUD
- Drug history : NSAIDs
- Family history
- Social history : Smoking

# 2) physical examination

- General ( pale , Jaundice, cachectic , malnutrition or volume depletion)
- Vital signs
- Abdominal examination (any tenderness, visible masses or peristalsis, abdominal distention: upper abdomen while lower abdomen scaphoid)
- Succussion splash is suggestive of GOO, but has a low sensitivity. 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
- Lymph nodes : left supraclavicular lymph node (Virchow's node) or periumbilical lymph node (Sister Mary Joseph's node) may be seen in metastatic gastric cancer.

### **INITIAL TREATMENT**

#### Admission and start resuscitation

- 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.
- Foley catheter & I.V. line insertion
- Intravenous fluids should be administered to correct hypovolemia and electrolytes abnormalities 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.
- Nutritional support with <u>parenteral nutrition</u> may be needed if definitive therapy is not imminent or for presurgical optimization of nutritional status.

# 3) investigations

#### Labs :

- CBC with differential (Anemia may be seen in patients with peptic ulcer disease, primary or metastatic malignant disease).
- Serum Electrolytes (Patients with recurrent vomiting may have electrolyte abnormalities, including hypokalemia or a hypochloremic metabolic alkalosis).
- Kidney Function Test
- ABG

#### **Additional tests:**

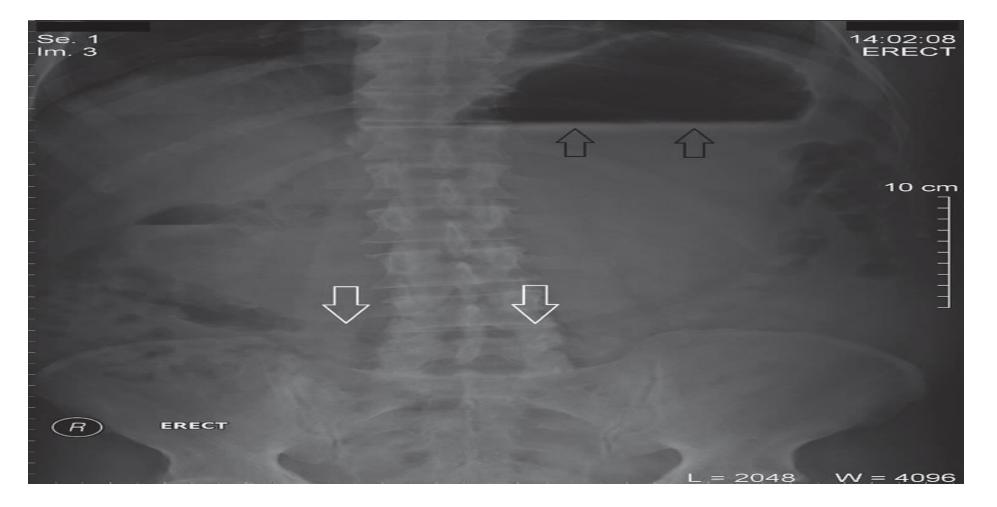
- H. Pylori test
- Gastrin level (distention induced gastrin release)
- Tumor markers CEA, CA 19-9



### 1) Plain films:

- Enlarged gastric bubble and a dilated proximal duodenum.
- Paucity of air in the small bowel.
- Calcified mass in the right upper quadrant is seen in up to 25% of patients with Bouveret's syndrome.
- Pancreatic calcifications are suggestive of chronic pancreatitis.

### **ENLARGED GASTRIC BUBBLE**



### **CALCIFICATION OF PANCREAS**







### 2) Contrast studies:

- Water soluble contrast 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's disease, or gastric volvulus, although findings are often nonspecific.

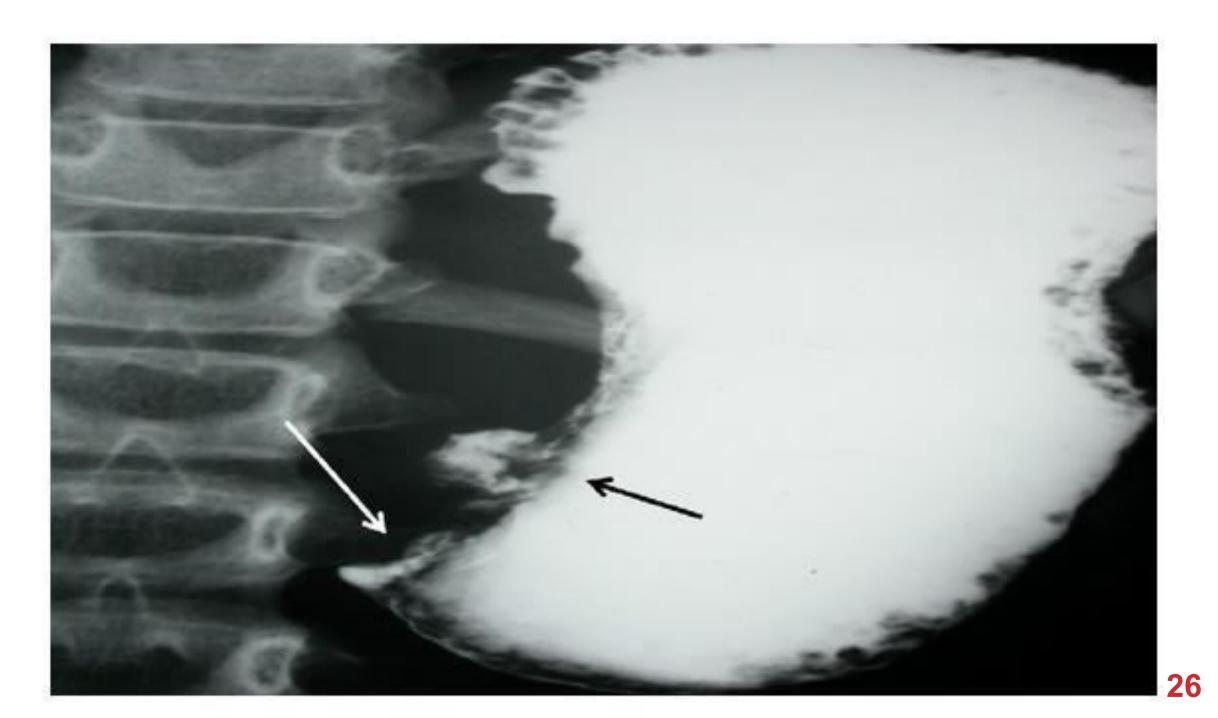
# **CONTRAST FINDINGS**

oMarkedly dilated stomach

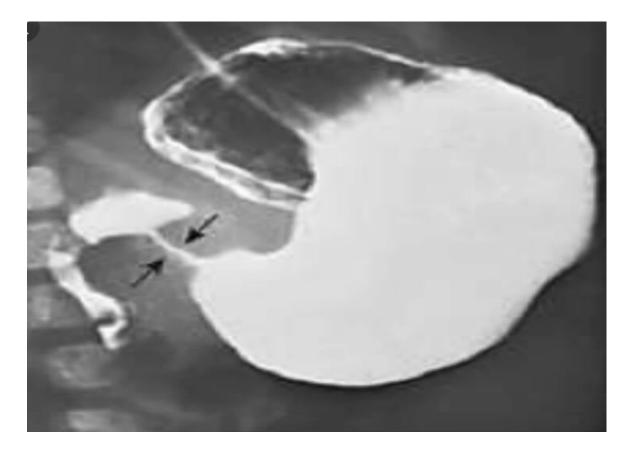
oLarge amount of residue.

oUlcer craters in case of PUD

oFilling defects at or near the pylorus.

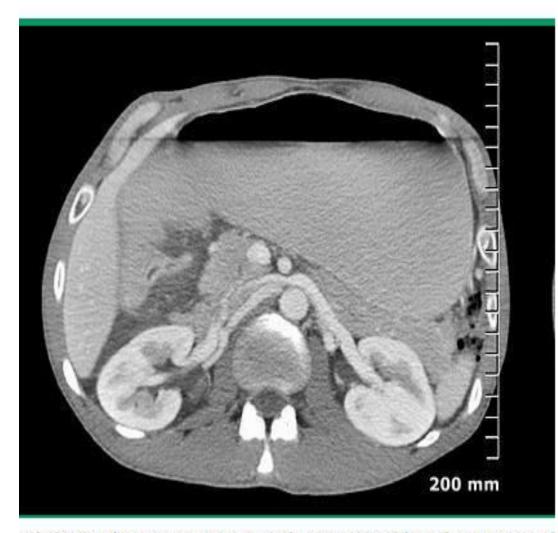








- An abdominal CT scan may reveal gastric distention along with retained material within the gastric lumen and an associated air-fluid level.
- CT scan may also give additional details such as the thickness of the pylorus or gastric wall, and it can also reveal if lymph nodes or pancreatic lesions are present
- CT will often also suggest the specific cause of GOO.



Abdominal CT in a patient with gastric outlet obstruction due to peptic ulcer disease showing a distended and fluid filled stomach. *Courtesy of Ashley Davidoff, MD.* 



Very large cyst in the gastro-hepatic ligament extrinsically compressing the pylorus causing gastric outlet obstruction, i.e. distended fluid-filled stomach.



### **4) Endoscopy:**

- Can be used to establish a diagnosis, determine the causes by taking biopsies and treat the cause.
- It is essential to biopsy the area around the pylorus to exclude malignancy.
- A nasogastric tube should be inserted and suction should be done before endoscopy to reduce the risk of aspiration.

### **5)Gastric function tests:**

•Following gastric decompression, to further evaluate mechanical outlet obstruction, a saline load test can be helpful.

• The saline load (750 ml) is emptied into a patient's stomach through a nasogastric tube. If more than 400 mL gastric contents are aspirated after 30 minutes, it is considered a positive test to diagnose GOO.

# MANAGEMENT

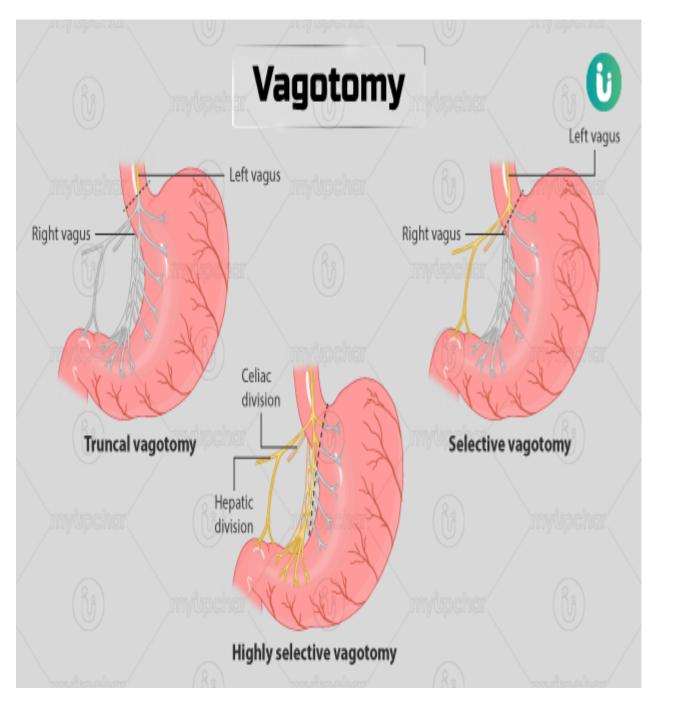
#### After initial treatment, treat underlying cause

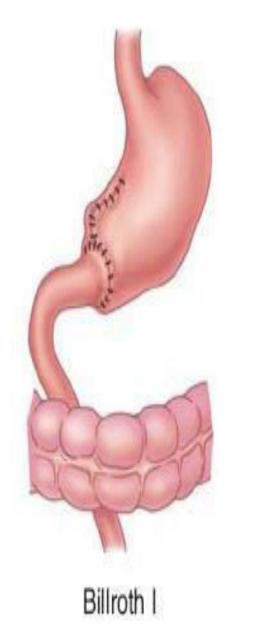
#### **PEPTIC ULCER DISEASE**

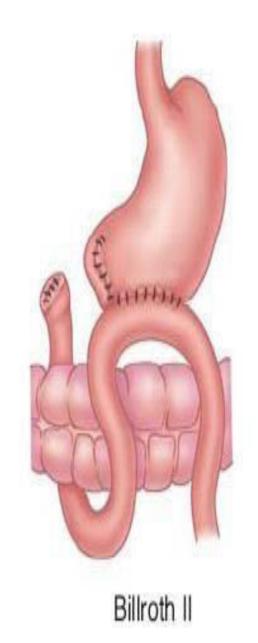
- Conservative therapy: eradication of H.pylori, PPI.
- Endoscopic therapy: patients who fail to respond to medical therapy may require endoscopic dilation or surgery.
- **Surgical therapy:** surgery is indicated if the pylorus is obstructed and can't be safely dilated, or if the obstruction persists or recurs despite medical and endoscopic management.

...vagotomy(decrease of stimulation)

...antrectomy to decrease gastrin secretion (Antrectomy to include the ulcer and truncal vagotomy is the ideal operation for most patients). ...gastrojejunostomy







# Endoscopic balloon dilatation (EBD)

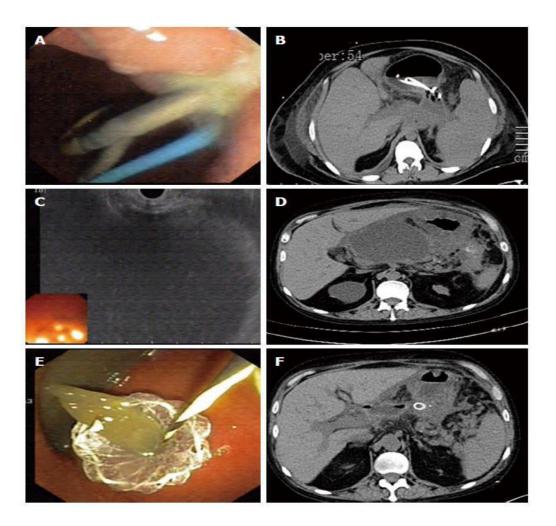
- Safe and effective alternative in the management in surgically unfit patients.
- A through-the-scope (TTS) 5 mm balloon with a 150 cm long catheter is used.
- Balloons are available from 6 mm to 20 mm
- The procedure is repeated 1-2 weekly until adequate dilatation of 15-18 mm is achieved



Figure 4: Endoscopic appearance of caustic GOO before and after CRE balloon dilatation; (a) narrowed pyloric opening; (b), (c) pyloric opening after serial dilatation; (d) post CRE dilatation opening of pyloric channel expanded to 15 mm

# **Pancreatic pseudocyst :**

 Computed tomography (CT)guided percutaneous drain placement



# **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 <u>surgical</u> bypass through a gastrojejunostomy or placement of an endoscopic enteral stent.

# Thank you