Peptic ulcer complications

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PUD

- Peptic ulcer disease (PUD) is a chronic condition that causes ulcers, or open sores, to develop in the lining of the stomach or duodenum
- caused by an imbalance between the acid and protective factors such as mucus and bicarbonate that line the digestive tract

Epidemiology

- prevalence of 5–10% and an annual incidence of 0.1–0.3% in the general population
- The risk to develop complication from chronic PUD 2-3% per year
- Mortality from PUD related to complications

Etiology and complication of PUD

- Most common causes of PUD is :

1) H.pylori:
   * Its gram –ve spiral shaped bacteria
   * lived in the antrum of the stomach
   * have a lot of virulence factor (urease , Cag A , Vac A)
   * can cause :
     1) gastritis
     2) peptic ulcer (duodenal 90% , gastric 70%)
     3) cancer

* most important risk factor for mortality is Age

Curling ulcer → Burn ulcer

Cushing ulcer → head trauma.

2) NSAIDs :
   inhibit COX-2 which block production of prostaglandins (PG help in secretion of mucus)

3) Others :
   (advanced Age , co-morbidity , alcohol , stress ,.... Etc

Diagnosis and management of PUD

- Diagnosis :
  - non invasive :
    1) urea breath test (used for follow up in eradication)
    2) stool antigen test (used for follow up in eradication)
    3) serology (not used in follow up)
  - invasive (upper endoscopy and biopsy)
    1) Histology
    2) Tissue urease test
    3) culture

*Giant gastric ulcer more liable for bleeding.

- Management :
  - change life style
    stop smoking , manage stress , limit alcohol consumption
  - medication (eradication of H.pylori) and follow up

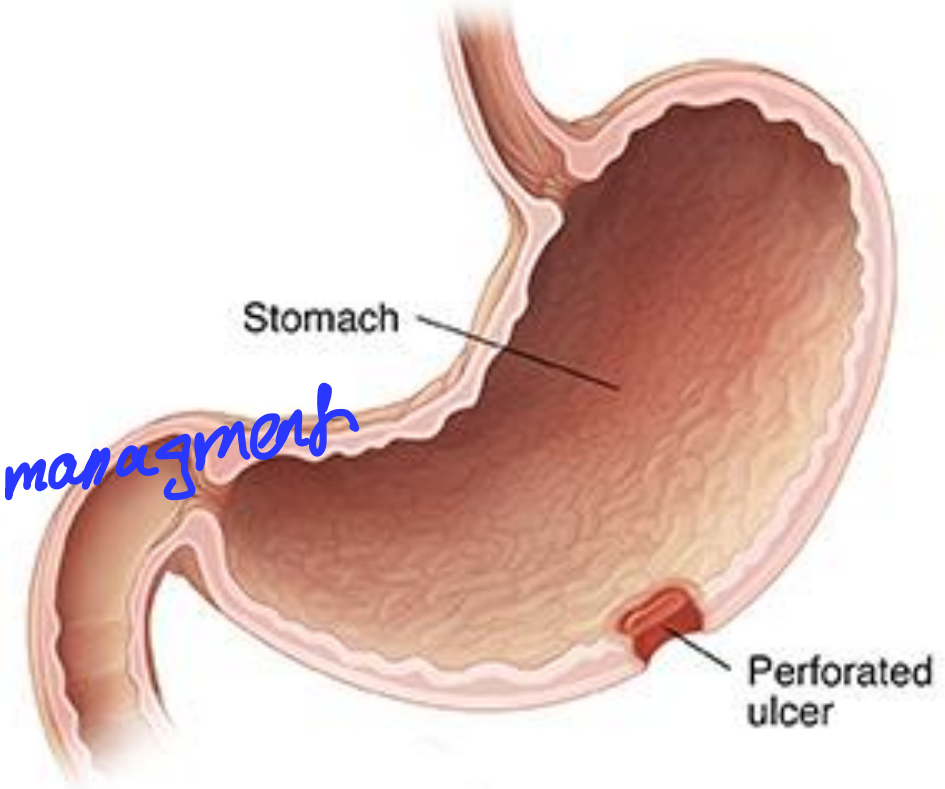
    triple therapy (PPI , Amoxicillin , clarithromycin)
    quadruple therapy (PPI , Bismuth , Tetracycline , metronidazole)

Complications of PUD

- Bleeding
- Perforation
- Gastric outlet obstruction

Perforation (PPU)

*type II, III which need secondary management



Perforation vs Penetration

- Penetration : leakage of content into adjacent organ by fistula
  ↳ mostly to Pancreaus.
- Perforation : leakage of content into peritoneal cavity

- Gastric ulcer most commonly penetrate into the left lobe of liver
- Duodenal ulcer penetrate posteriorly into the adjacent pancrease , sometimes lead to pancreatitis

Epidemiology

- Perforation is the second most common complication of peptic ulcer, but it represent the most frequent indication for emergency surgery for PUD

- Previously, most patients were middle aged, with a M:F ratio of 2:1, now occur most commonly in elderly female patients

- The lifetime prevalence of perforation in patients with PUD is about 5%
- PPU carries a mortality ranging from 1.3% to 20% .

Causes and Risk factors

❖The exact cause why some patient develop perforation is unknown

But there is multiple risk factors :

- PUD (due to H.pylori ,NSAID , stress)
- Previous Bariatric surgery
- Hyper acidic state (Zollinger-Ellison syndrome)
- Chemotherapy

Cont...

- Most common sites of perforation :

  1) Anterior wall of duodenum

  2) Posterior wall of stomach

- Present in two form :

  1) acute

  2) subacute

Acute PPU

- Three stages :

Stage 1 (chemical peritonitis) :

*leakage of gastric content into peritoneal cavity

Symptom :

Sudden severe epigastric pain which become generalized

Sign :

1) guarding and epigastric tenderness
2) Decreased liver dullness (air under diaphragm)
3) Shifting dullness (fluid in the peritoneal cavity)
4) Decreased intestinal sounds (paralytic ileus occurs late).

Cont…

Stage 2 (Illusion)

*__**Reaction of peritoneum**__ (release large amount of alkaline fluid to neutralize the acidity)

Symptoms :

decrease in pain

Sign :

like the previous stage __**but**__ with increase shifting dullness

Cont...

Stage 3 (septic peritonitis)

***Translocation of bacteria** (lead to infection and pus formation in the peritoneum)

Symptoms :
increase in pain , fever , anorexia ,vomiting ,abdominal distention

Sign :
generalized rigidity and tenderness

- As a complication it can cause :

1) Septic shock
2) paralytic ileus

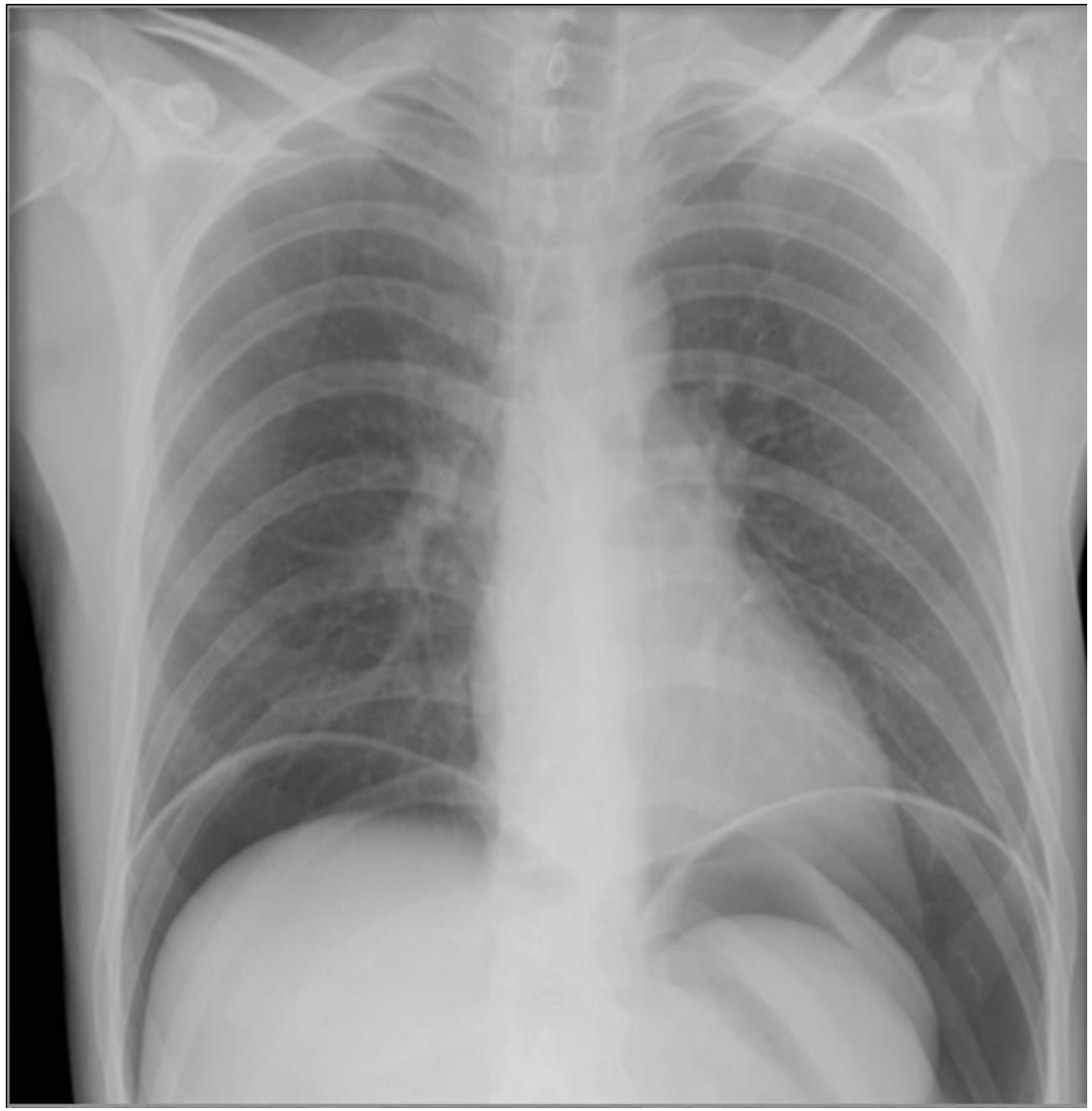
Subacute PPU

may be sealed perforation by tissues

- A small perforation allowing only a minimal amount of contents to enter the peritoneal cavity and is rapidly sealed
- It give the same early signs as perforated PU but much reduced
- If diagnosed correctly it can be treated conservatively .

Investigations

- Labs:
  serum amylase (non specific)
  CBC (elevated WBCs)
  electrolyte disturbances (hyponatremia , hypokalemia)
  KFT → suggest Prerenal injury
  Gastrin level → in Case of ZES suggestion.
    - → reccurence
    - → ass. diarrhea
- Imaging :
  X ray
  CT scan



Imaging

→ standing chest not Abdomen.

- **Erect x-ray :**
  - The erect chest x-ray is the most important initial plain film commonly performed in patients with acute upper abdominal pain suspected of perforation
  - If there is a perforated peptic ulcer, air will rise and collect beneath one or both hemidiaphragms
  - This technique is about 80% sensitive for perforation

* Left lateral decubitus position → in patient Cannot tolerate standing.

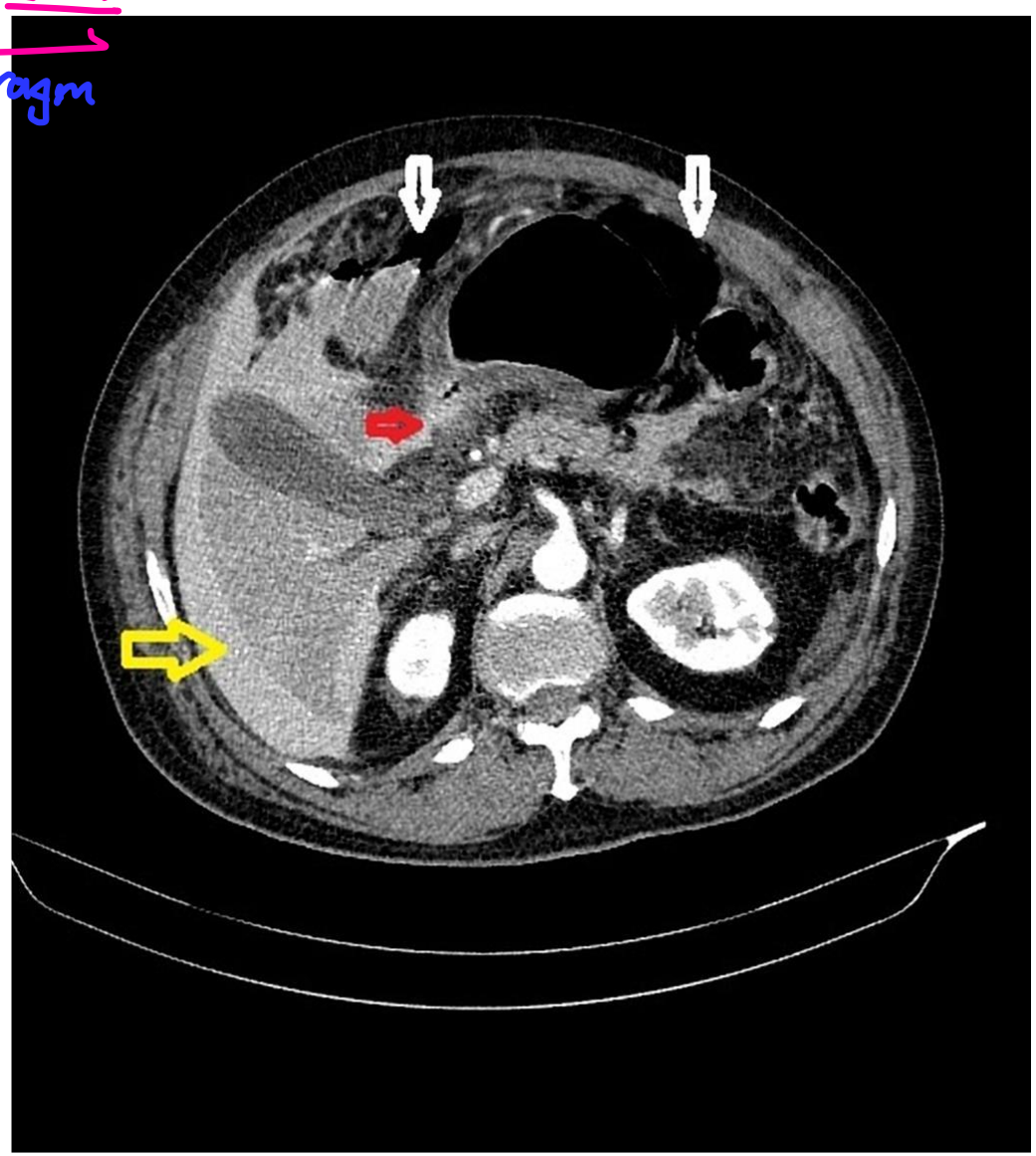
Cont...

enough for PPU dx.

Hx + PE + Labs + CXR

indications: if air under diaphragm not appear on CXR.

- **Oral contrast enhanced CT scan :**
  - It has a diagnostic accuracy as high as 98%.
  - performed in supine position and free air is usually seen anteriorly just below the anterior abdominal wall
  - Gastrografin a contrast agent used for diagnosis of PPU as Barium study is contraindicated in gastrointestinal perforation



Management

1) Non operative (conservative) :
   general management
   1) Treat underlying cause (H.pylori ....etc)
   2) Stop NSAIDs
   3) PPI
   4) Resuscitation

*used in selective patient with mild perforation like subacute PPU

2) Operative :
   (laprotomy or laproscopy)
   1) simple closure
   2) graham patch (omental patch)
   3) resection and reconstruction (billroth {1 or 2} or roux-en-Y)

*most common indication for operation is perforation.

ulcer + Biopsy.

duedenal.

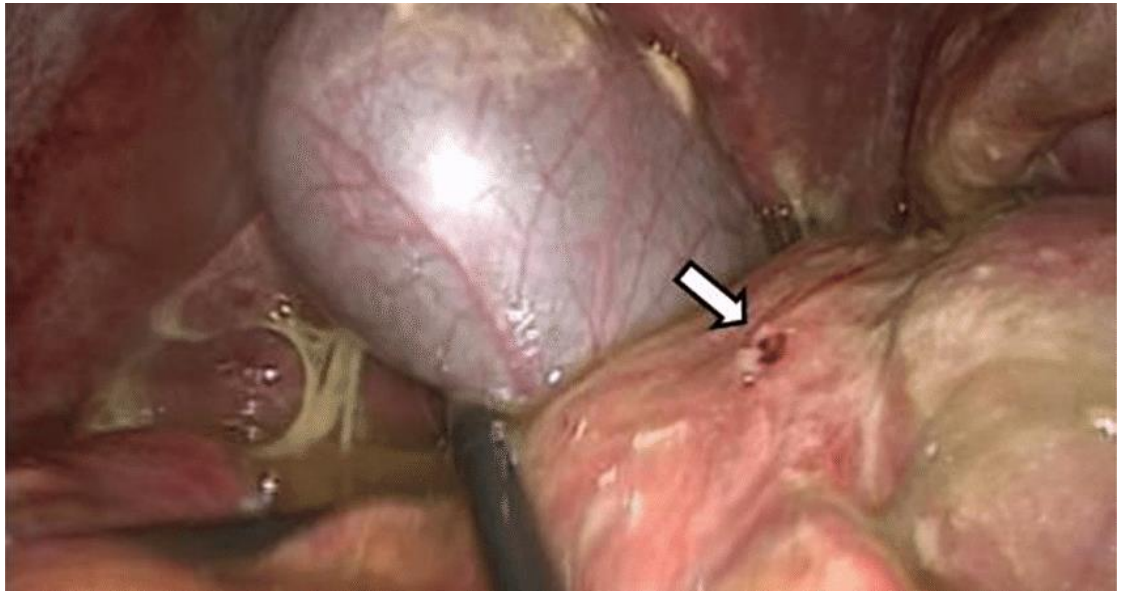
*stable or unstable.

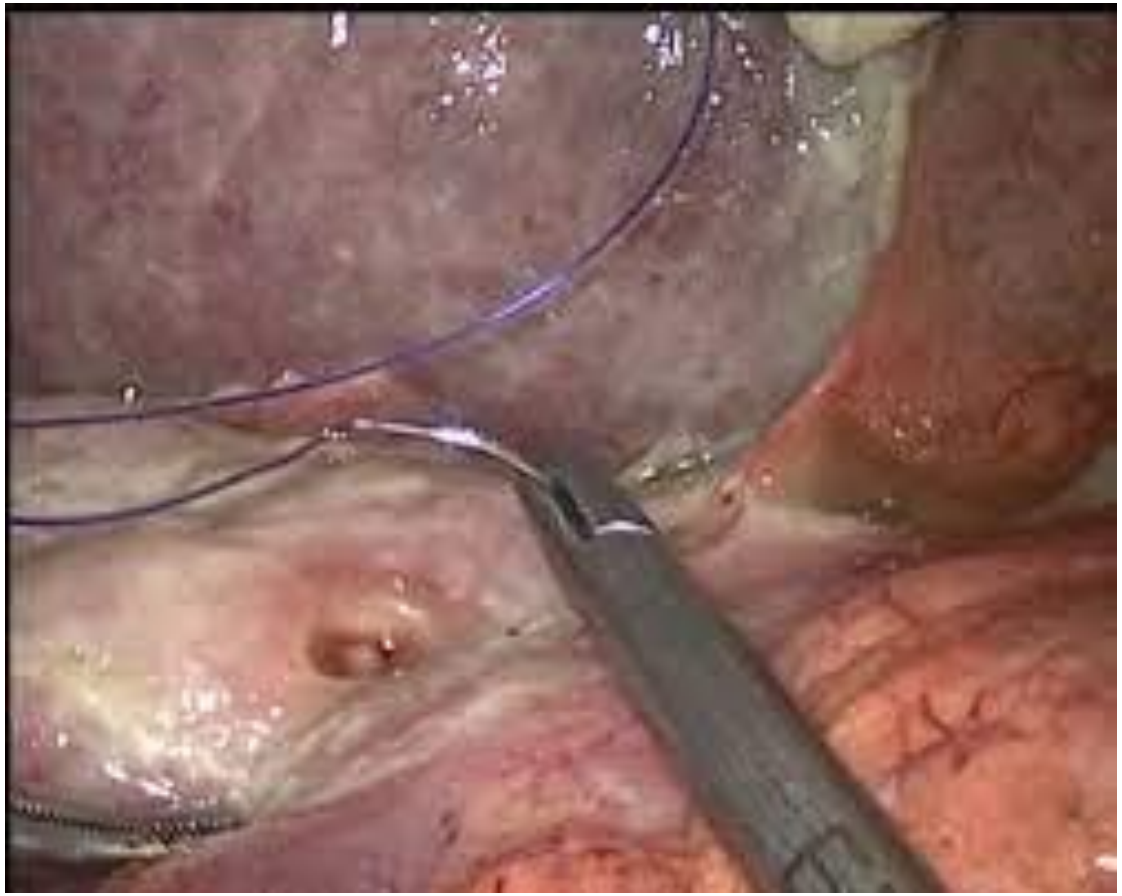
due to high Parietal cell volume.

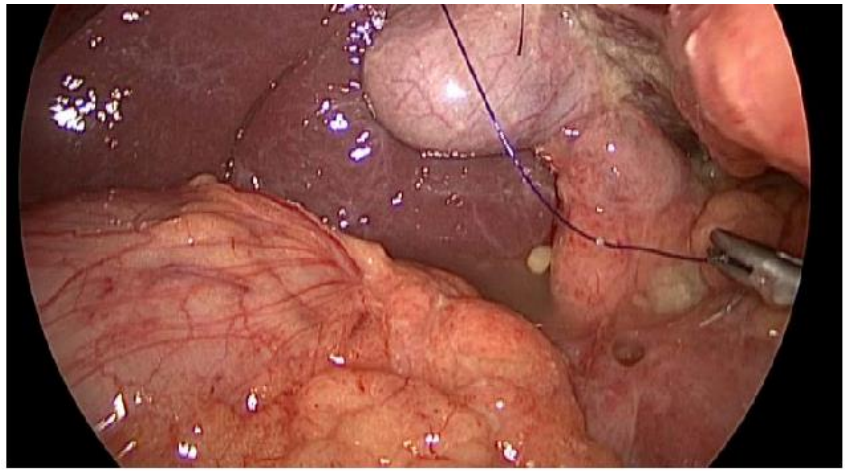
+ cut antrum + vagus + Garaham Patch.

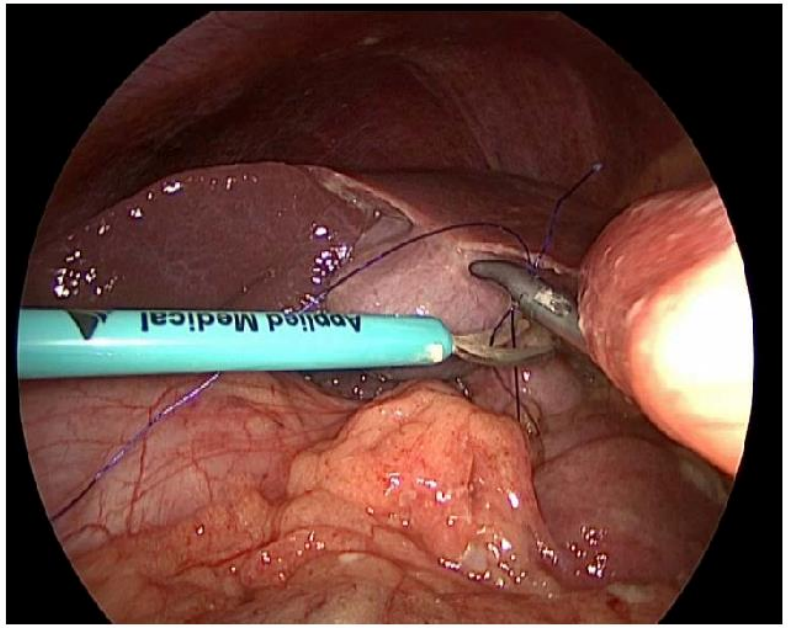
Garaham Patch. omental patch.

*High creatinine → normal saline not ringer.









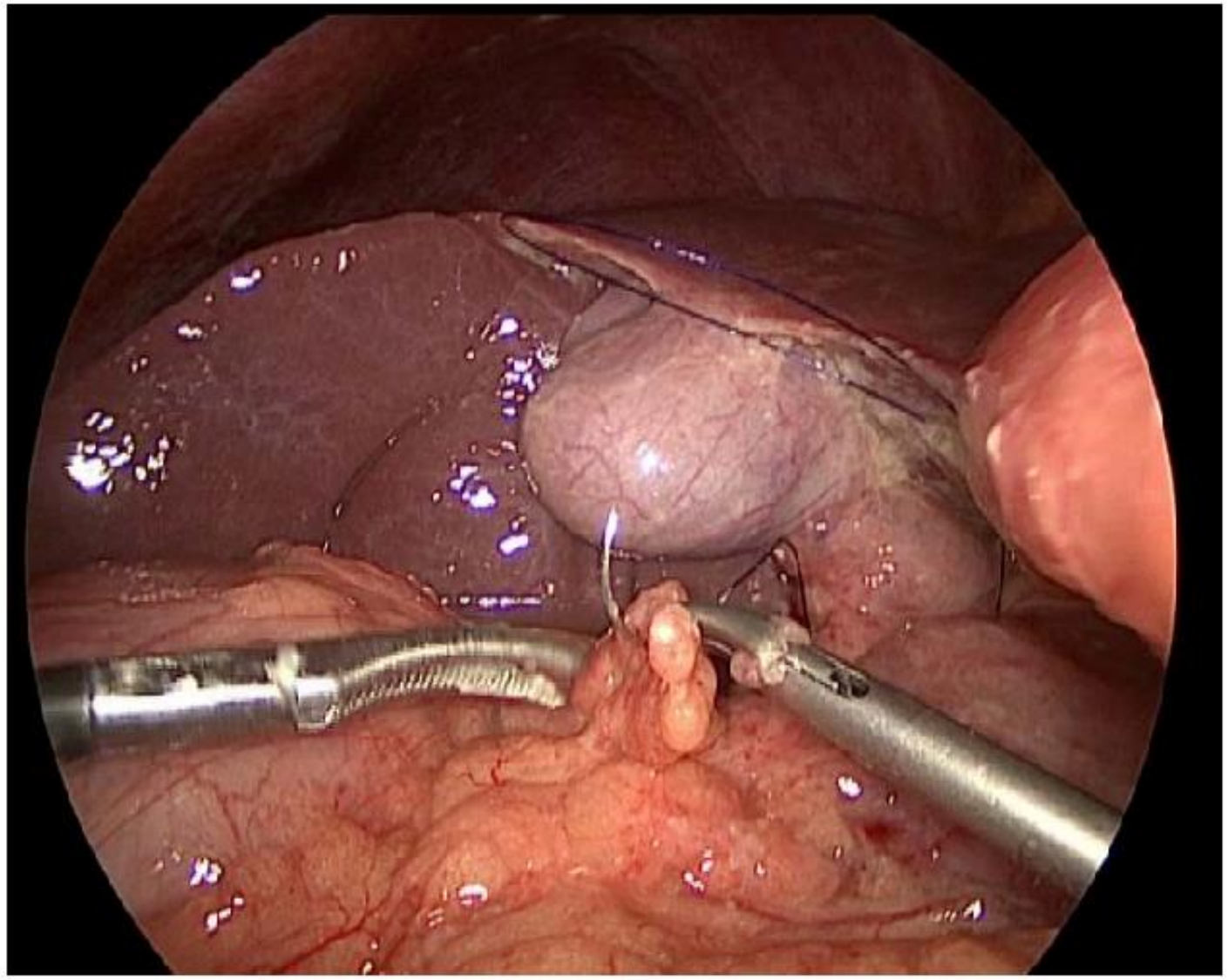
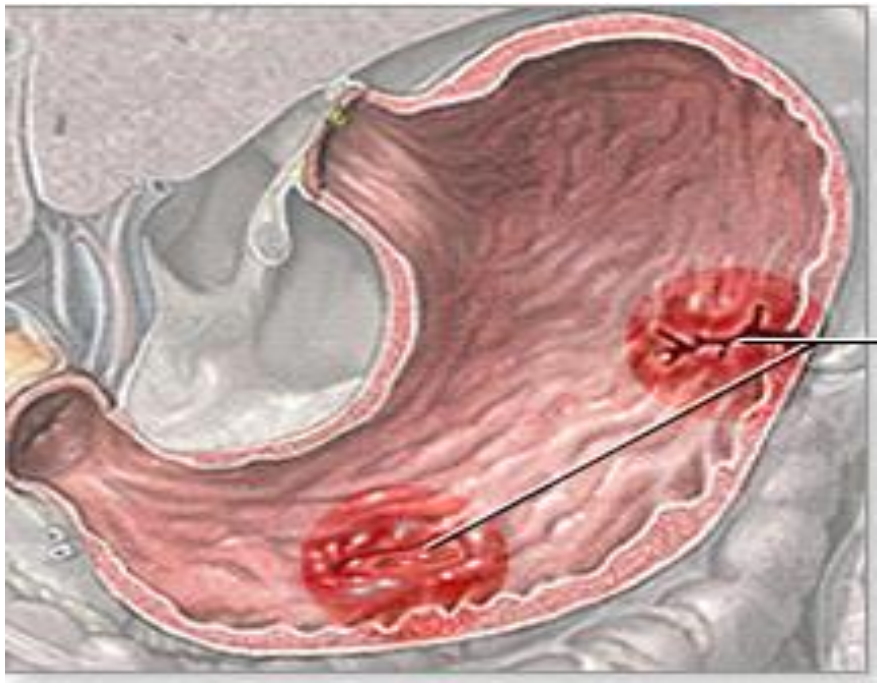
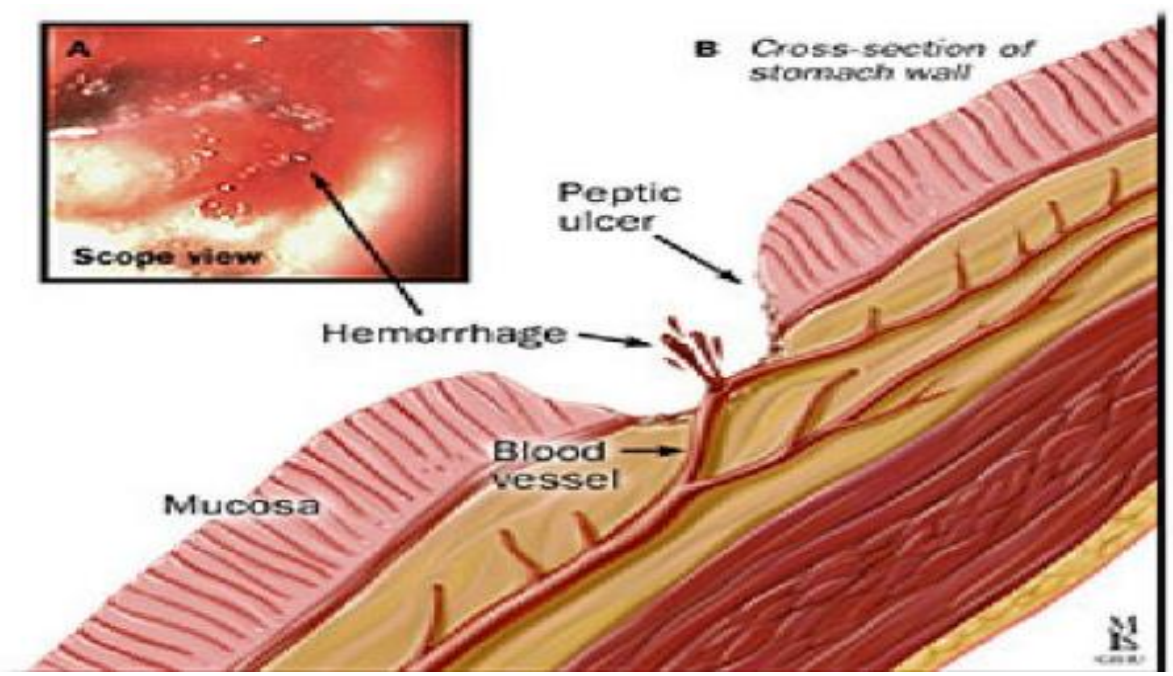


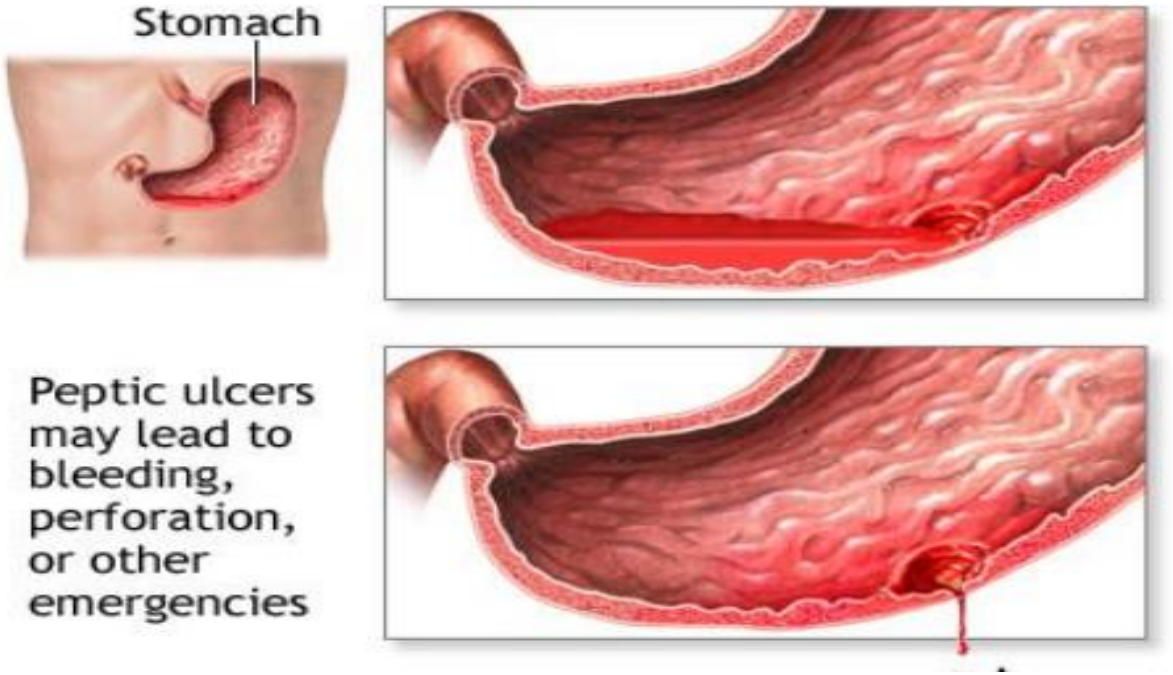
Figure 4. The final appearance of the repair.

Bleeding



Bleeding can occur when an ulcer erodes into an underlying blood vessel
Ulcer → Erosion more mucosa , sub mucosa → bleeding





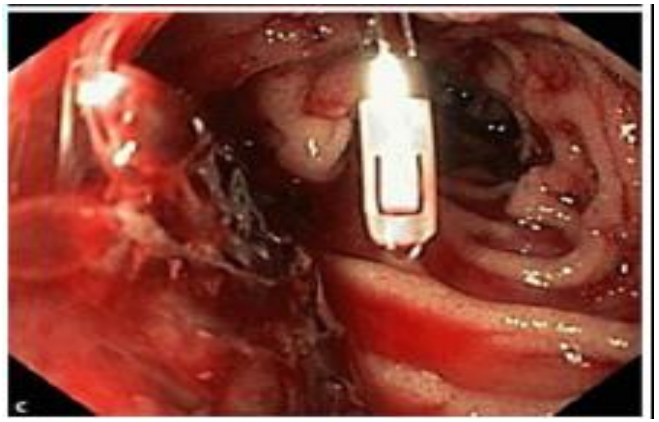
- The most common cause of upper GI bleeding(60% of all causes)
- Most common complication of PU (10-15%)
- Higher incidence to peptic ulcer bleeding noticed in patients above the 6th decade.
- Percentage of patients require surgery ~ 10%
- Percentage of patients spontaneously stop bleeding ~ 80-85%

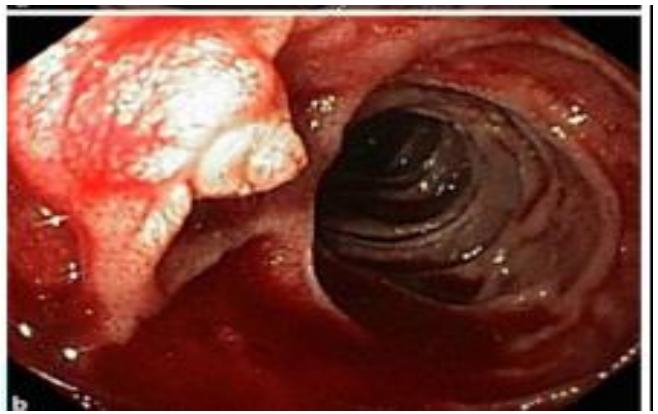
SITES OF BLEEDING :

1. Duodenal ulcer→

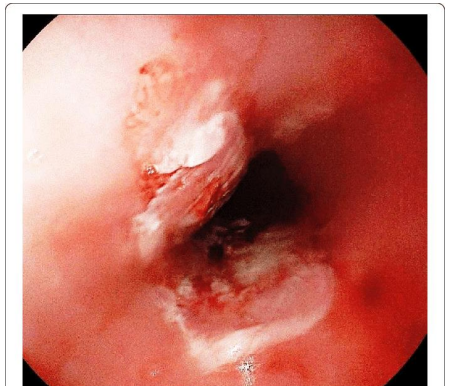
❖ is four times more common than gastric bleeding.

❖ most common site is posterior wall from gastro duodenal artery

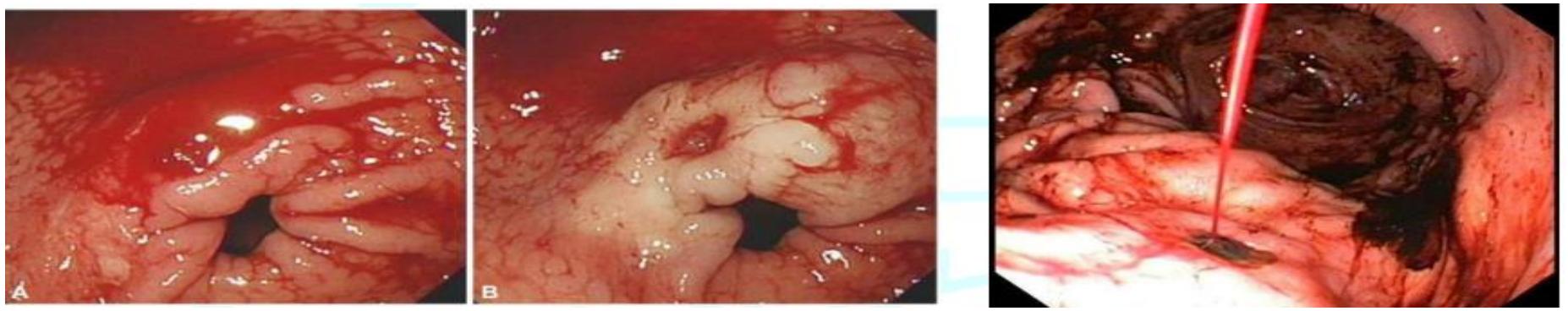




Kissing ulcer : a pair of ulcers facing each other on the opposite walls of the stomach or duodenum



2. Gastric ulcer bleeding:
Most common sites are the lesser curvature of stomach
• Large chronic ulcers may erode posteriorly into the pancreas and on other occasions, into major vessels such as Left gastric artery a branch of the celiac artery / and splenic artery.



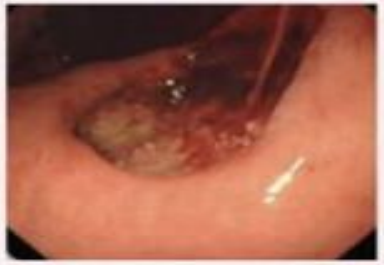
BLEEDING MAY BE:

- Mild: Due to erosion of the friable granulation tissue in the floor of the ulcer.
- Moderate: Due to erosion of a small vessel in the floor of the ulcer.
- Severe: Due to erosion of a large extra-gastric vessel (gastroduodenal or splenic).

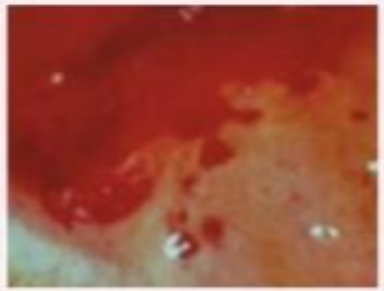
Forrest Classification

Acute Hemorrhage

2nd Re-check endoscopy.



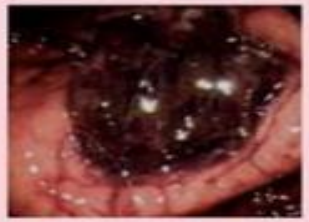
1a
Active Spurting
Rebleeding Risk:
60 to 100%



1b
Active Oozing
Rebleeding Risk:
50%

Signs of Recent Hemorrhage

IIa
Non-Bleeding Visible Vessel
Rebleeding Risk:
40 to 50%



IIb
Adherent Clot
Rebleeding Risk:
20 to 30%

IIc
Flat Spot in Ulcer Base
Rebleeding Risk:
7 to 10%

Lesions without Active Bleeding

III
Clean-Based Ulcer
Rebleeding Risk:
3 to 5%

@enrrikke

Images from Alzoubaidi, et al, 2018

- First described in 1974 by J.A. Forrest et al. in The Lancet
- Standardized classification system for endoscopists to describe peptic ulcers
- Helps prognosticate and risk stratify patients based on stigmata of recent hemorrhage and decide on discharge versus close inpatient monitoring

PRESENTATION OF A PATIENT WITH BLEEDING PUD:

Acute bleeding (heavy):

1.Melena
2. Hematemesis
3. Hematochezia " if massive"
4. Hypotension / shock

Chronic bleeding (slow):

1. Coffee ground vomiting
2. Anemia "iron deficiency

Hematochezia:

*** usually represents a lower GI source of bleeding .**

*** When hematochezia is presenting symptom of UGIB it is associated with hemodynamic instability and dropping hemoglobin.**

*** Hypovolemic shock could be a complication of bleeding peptic ulcer.**

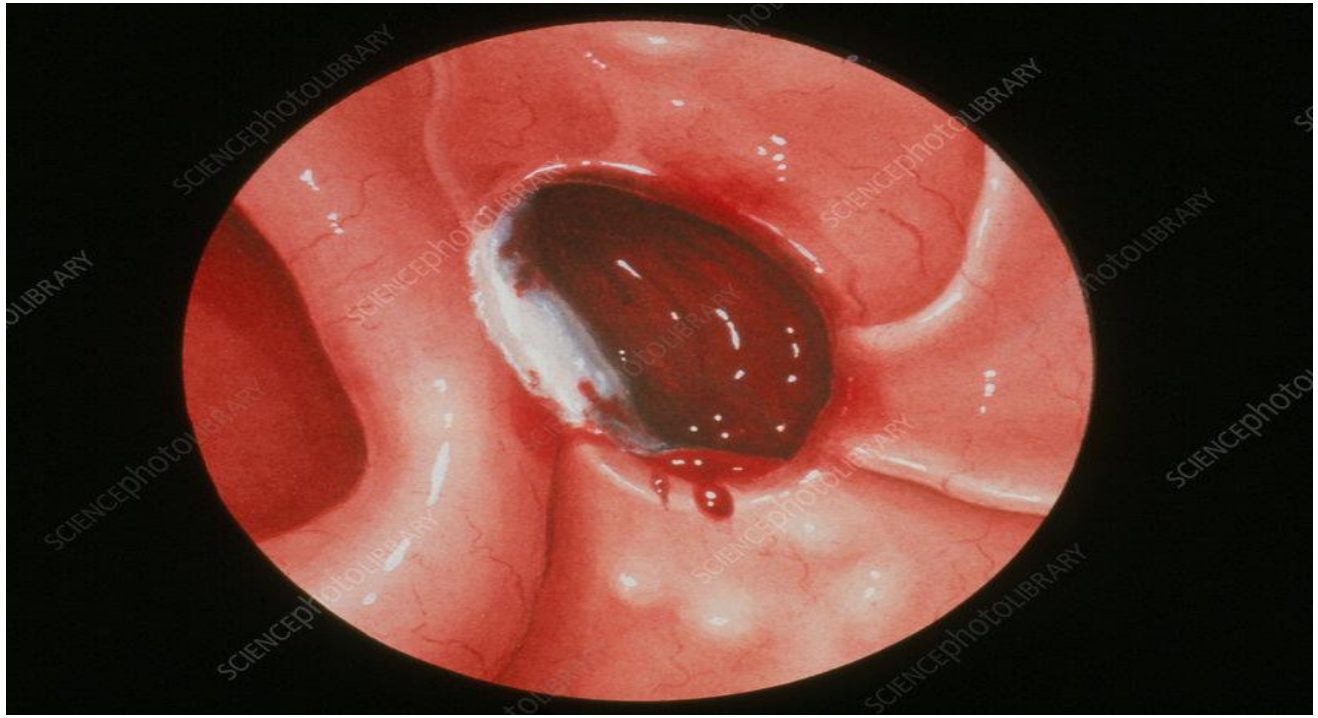
INVESTIGATION

- **Lab test**

1. **CBC : Hb / hematocreate / WBCs**
2. **KFT : pre renal injury (dehydration)**
3. **H.pylori test**

- **Imaging :**

1. **Endoscopy**
2. **X-ray**
3. **CT scan**
4. **U/S**
5. **CT angio**

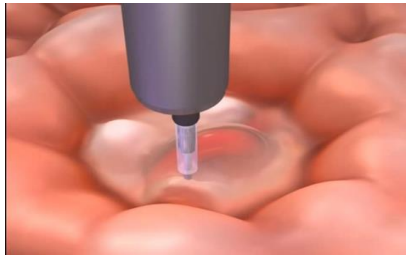


RESUSCITION

- 2 IV cannulas
- Cross match : for blood transfusion
- Folys catheter : monitor urine output
- NG tube : suctionong
  - confirming
  - decompression (endoscopy)
  - to monitor the bleeding

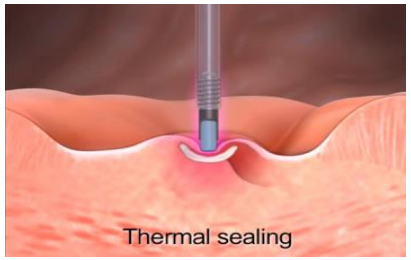
TREATMENT

1. Endoscopic : → Gold standard for Bleeding.



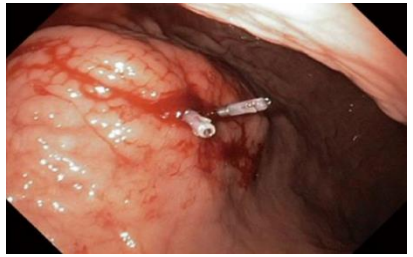
- Nor-adrenaline near the submucosa → 2 attempts before going to surgery.

- Thermal (cautrization)



- Mechanical : - clips (7-10 days)

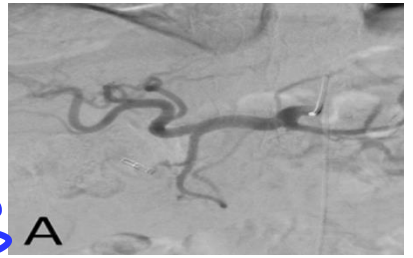
  Multiple clips maybe achieve hemeostasis .



- interventional radiology
  (Theraputic embolization)

*Tagged RBCs for LGIB

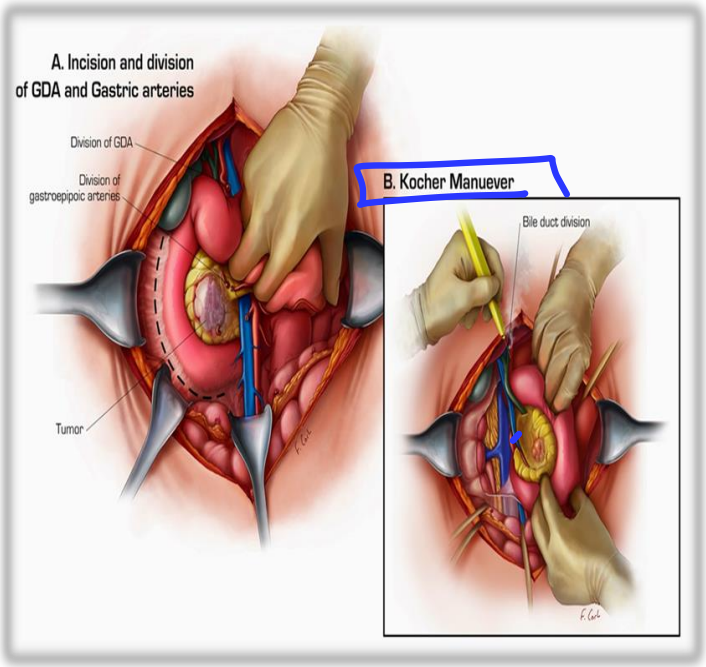
→ inject embolizing material in UGIB



2. Surgical

Indication :

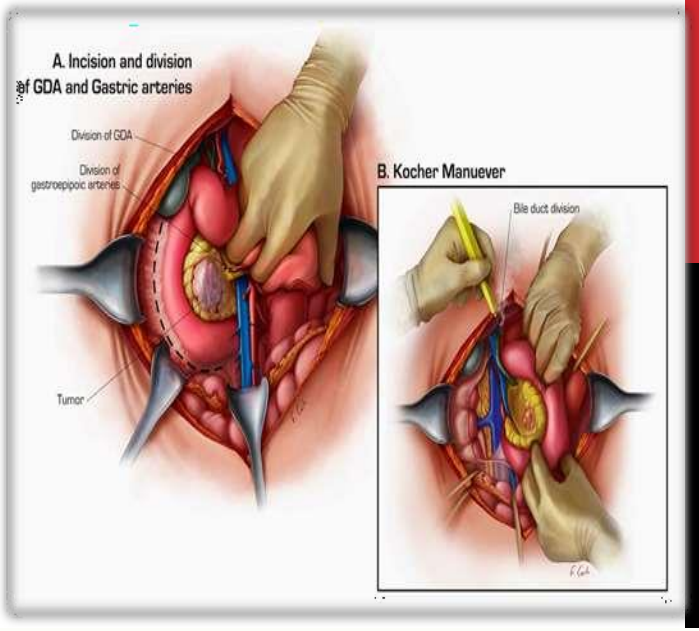
- **Recurrence after 2 attempts (endo)**
- **Active bleeding despite endoscopy trials**
- **Hemodynamically unstable despite strong resuscitation**



Upper endoscopy is the best initial diagnostic and therapeutic procedure in the management of bleeding peptic ulcers.

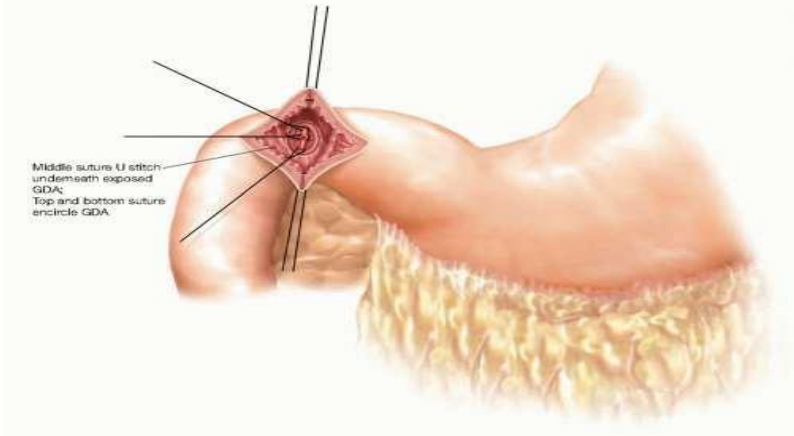
SURGICAL :

In tackling this, it is essential that the duodenum is fully mobilized.
This should be done before the duodenum is opened as it makes the ulcer much more accessible and also allows
the surgeon's hand to be placed behind the gastroduodenal artery, which is commonly the source of major bleeding.



So first we do **Kocher maneuver**

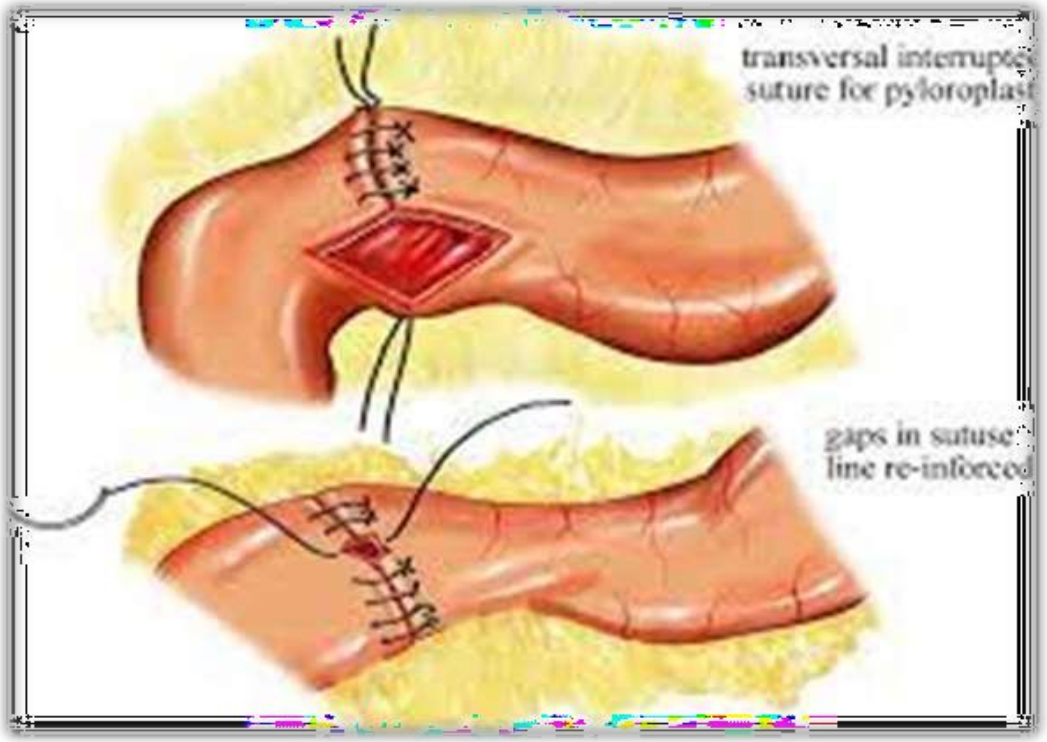
(the dissection of the lateral peritoneal attachments of the duodenum to allow inspection of the duodenum, pancreas, and other retroperitoneal structures over to the great vessels)



THEN , Following mobilization, the duodenum, and usually the pylorus, is opened **longitudinally**.
This allows good access to the ulcer, which is usually found posteriorly or superiorly

THEN oversewing of the bleeding vessel , It is the vessel within the ulcer that is bleeding and this should be controlled using well-placed sutures

Finally , we close the opening with interrupted sutures in a transverse direction

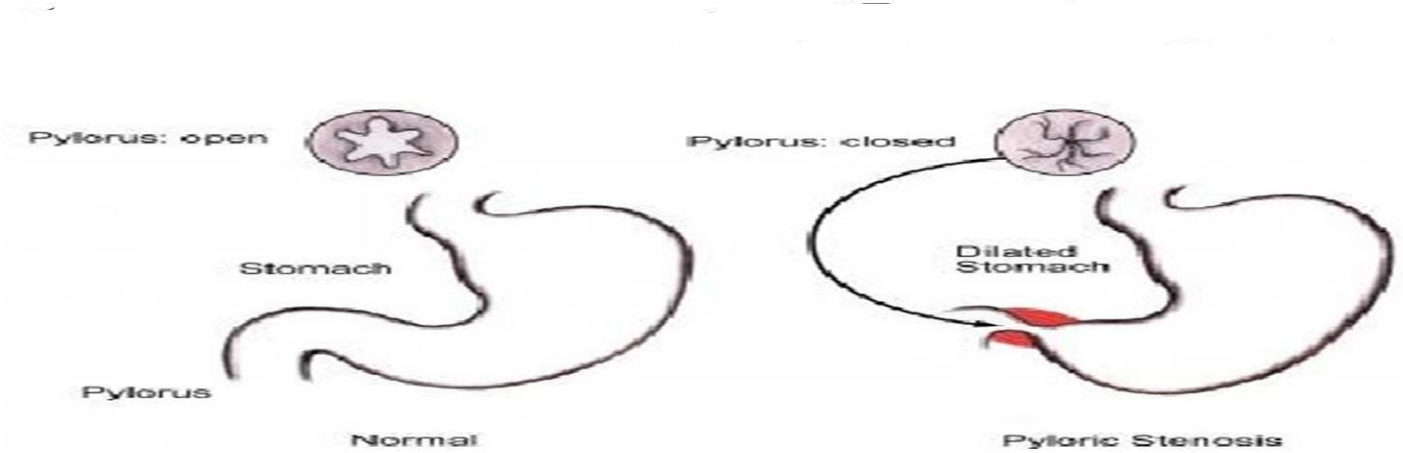


RISK FACTORS

Any factor leading to acute exacerbations and inflammation of the ulcer

- **NSAID**
- **Helicobacter pylori infection**
- **Smoking**
- **alcohol**
- **nervousness and stresses**

3- Gastric outlet obstruction



Done by : Saleh Maaitah

GOO :

- **Gastric outlet obstruction** (GOO) is a clinical syndrome characterized by **epigastric abdominal pain and postprandial vomiting** due to mechanical obstruction resulting in impaired gastric emptying.
- **Etiology** : Two major causes of GOO are **Benign gastric disorders as Peptic ulcer** which leads to pyloric stenosis and **Gastric malignancy** .
- Due to **decline** in PUD (PPI use and H. pylori eradication), the **majority of cases (50-80%) of GOO are due to Malignancy** , so **Endoscopic biopsy is needed to determine the cause of obstruction.**
- **Anatomically**, the mechanical obstruction can be at the distal stomach, pyloric channel, or duodenum; and can be intrinsic or extrinsic to the stomach.

*now Malignancy is the MC cause of GOO.

GOO :

- **Peptic ulcer induced GOO :**
- **Acute PUD : can cause obstruction by inflammation-induced edema and tissue deformation. However , Chronic PUD cause obstruction of Gastroduodenal junction by scarring and remodeling as a part of healing process.**
- **Note that GOO is the least common complication of PUD occurring in 2-3% of cases.**
- **Other benign causes include : Crohn disease and Pancreatitis , Hypertrophic pyloric stenosis in infants.**
- **Malignant causes include : adenocarcinoma , lymphoma, and gastrointestinal stromal tumours.**

GOO-clinical pictures

important

- Nausea and vomiting as chief complain.
- Epigastric pain , anorexia and weight loss.
- Electrolyte disturbance .
- Note that the vomiting is the main feature and it is :

1. Projectile .
2. Non-Bilious .
3. Contain undigested food .
4. Post prandial .

* nausia & pain → relieved by vomiting.

* early satiety.

GOO-Examination

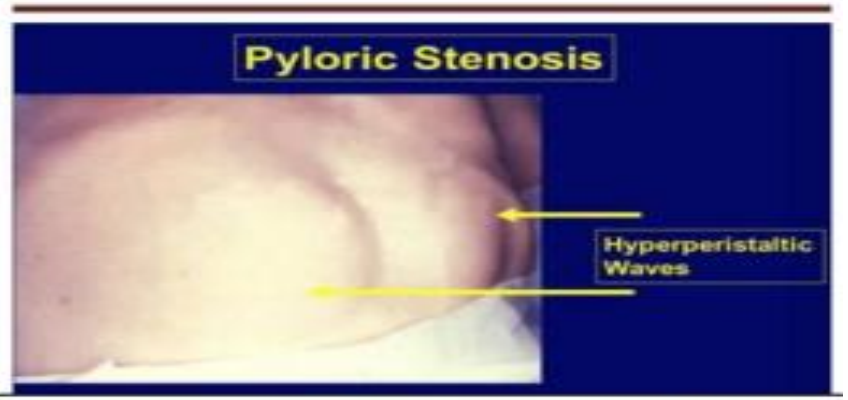
- **Abdominal distention is seen.**
- **Succussion splash, If a succussion splash is noted more than four hours after a meal, it is suggestive of GOO with a 50% sensitivity .**
- **Visible gastric peristaltic waves is also seen .**

*not marked abdominal distension,

Epigastric fullness

succession splashes

rocking.



hyperperistaltic waves LR

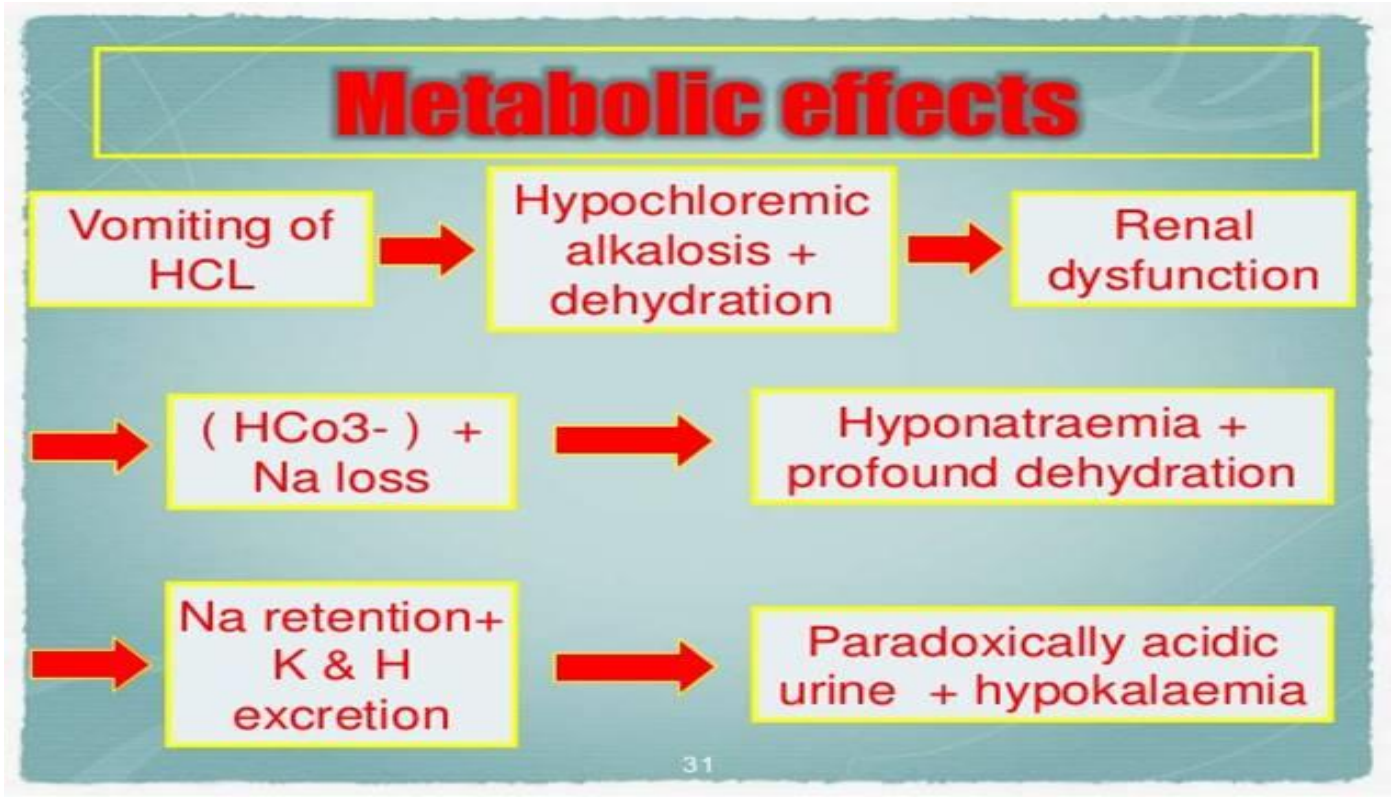
GOO-Metabolic effect

important written.

- Classically, GOO causes a - hypokalemic, hypochloraemic metabolic alkalosis with paradoxical aciduria and hypocalcaemia.
- Prolonged vomiting causes loss of hydrochloric acid and create a hypochloremic metabolic alkalosis.
- Dehydration activates the RAAS and this leads to more Na retention with increases K and H excretion in the distal tubule -> hypokalemic alkalosis.
- Paradoxical aciduria:- Initially urine has low Cl and high bicarbonate content (Urine is initially alkaline).
- The bicarbonate is excreted with Sodium as a compensatory mechanism and with time the patient becomes progressively hyponatremic and more dehydrated.
- Dehydration leads to sodium retention and K and H are excreted instead, this leads to acidic urine.

GOO-Metabolic effect cont.

- **Electrolyte Changes In GOO :**
- **metabolic alkalosis**
- **Hypochloremia**
- **Hyponatremia**
- **Hypokalemia**
- **low circulating ionized calcium. (alkalosis)**
- **Hypomagnesemia**
- **paradoxical aciduria**



GOO-INVESTIGATION

- **Endoscopy**

- **Imaging :**

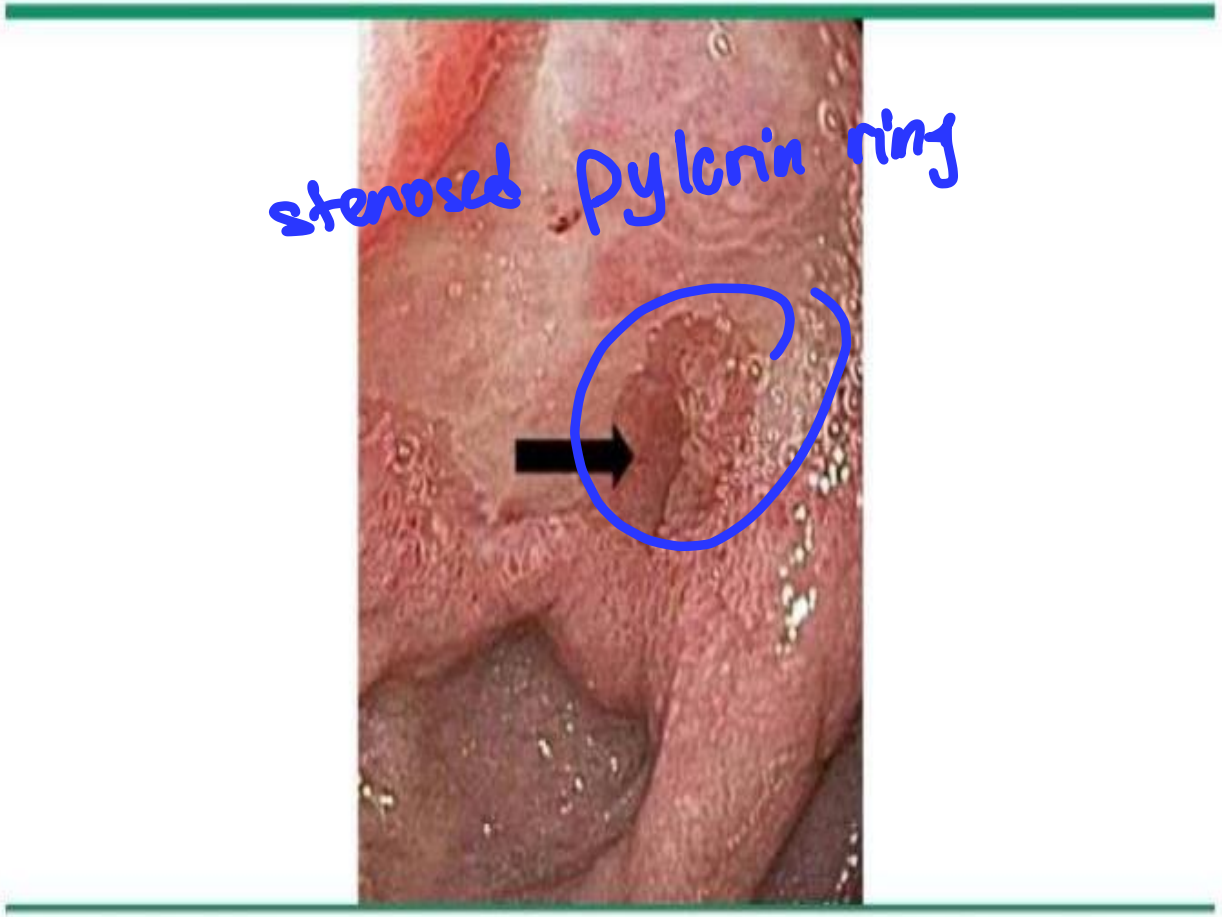
1. **Barium meal.**
2. **Abdominal x-ray.**
3. **CXR for mets .**
4. **CT.**

- **Others :**
- **CBC**
- **Test for H.pylori if PUD is suspected**
- **LFT : if elevated may indicate mets**
- **ABG : metabolic alkalosis**
- **KFT : for pre-renal failure.**
- **Sodium chloride load test**

GOO-INVESTIGATION

- **Endoscopy : Most important investigation .**
- **The stomach should be emptied using a wide-bore gastric tube.**
- **It shows stenosed inactive pyloric ring.**
- **Identifies the level of obstruction.**
- **Detect the cause & take biopsy to exclude malignancy.**

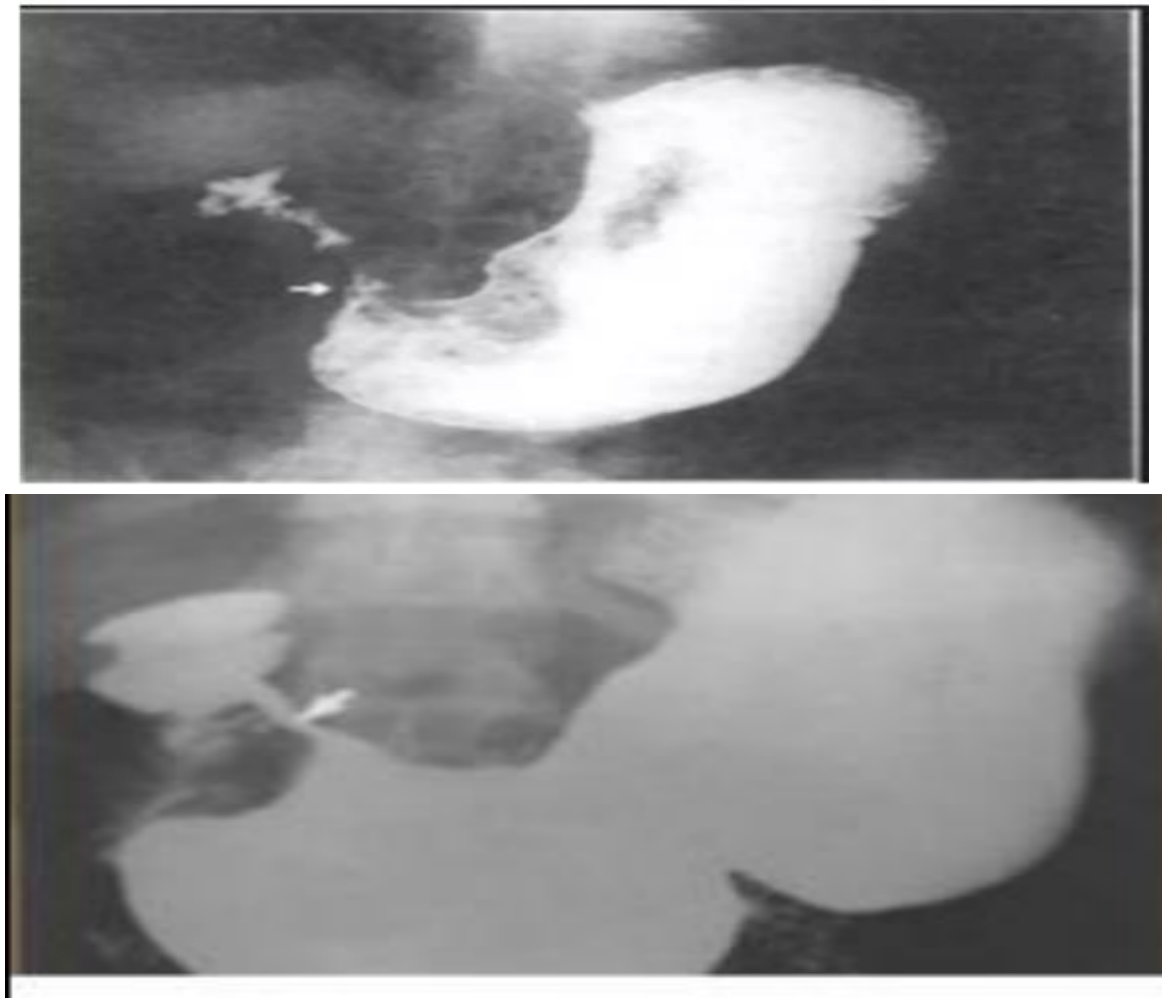
Gastric outlet obstruction due to peptic ulcer disease



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.

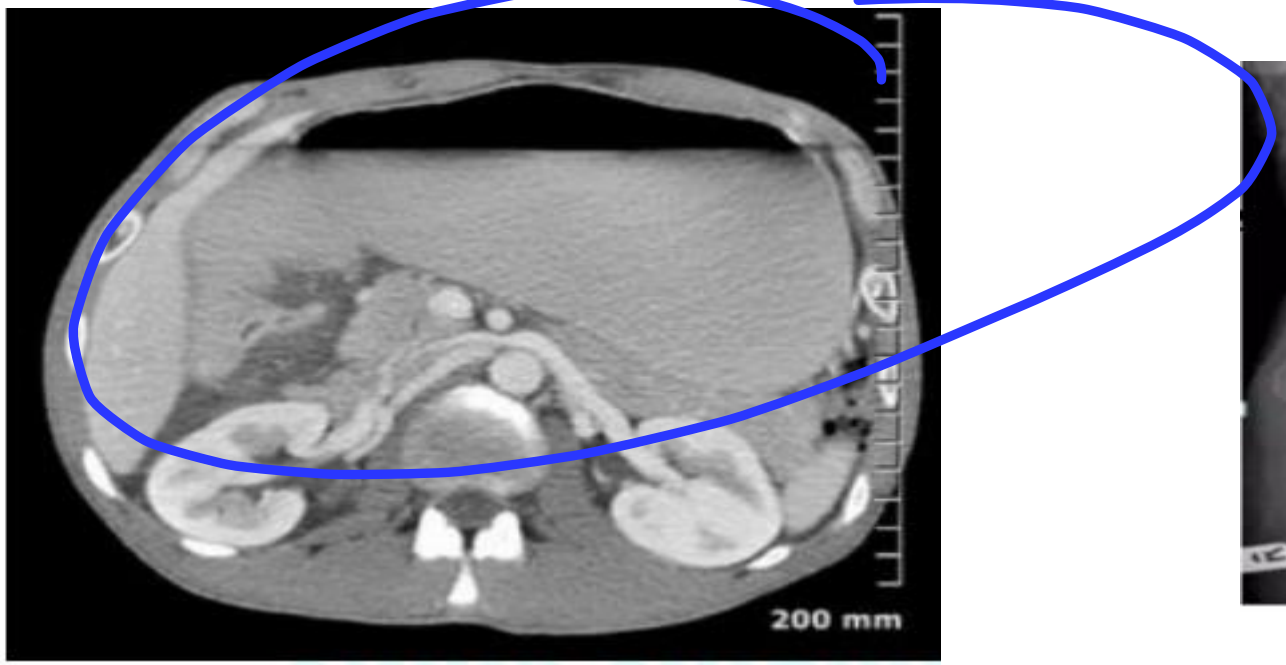
GOO-INVESTIGATION

- **Barium Meal** : After 6 hours of fasting, barium is administered orally then x ray is taken in supine position at intervals of 20-30 min.
- Hugely dilated stomach (may reach the pelvis) with wide smooth antrum ,and the point of obstruction is visualized by string sign. **White arrow.**



GOO-INVESTIGATION

CT , showing dilated stomach.



Plain abdominal x-ray showing dilated stomach.

GOO- Management

- By correcting the metabolic abnormality and dealing with the mechanical problem.
- **Conservative management** :

1. Nasogastric suction by NG tube .
2. Acid suppression by PPI .
3. Fluid replacement..
4. Correct the electrolyte abnormalities.
5. Eradication therapy of H.pylori .

normal saline + 2g KCl

GOO + renal failure →

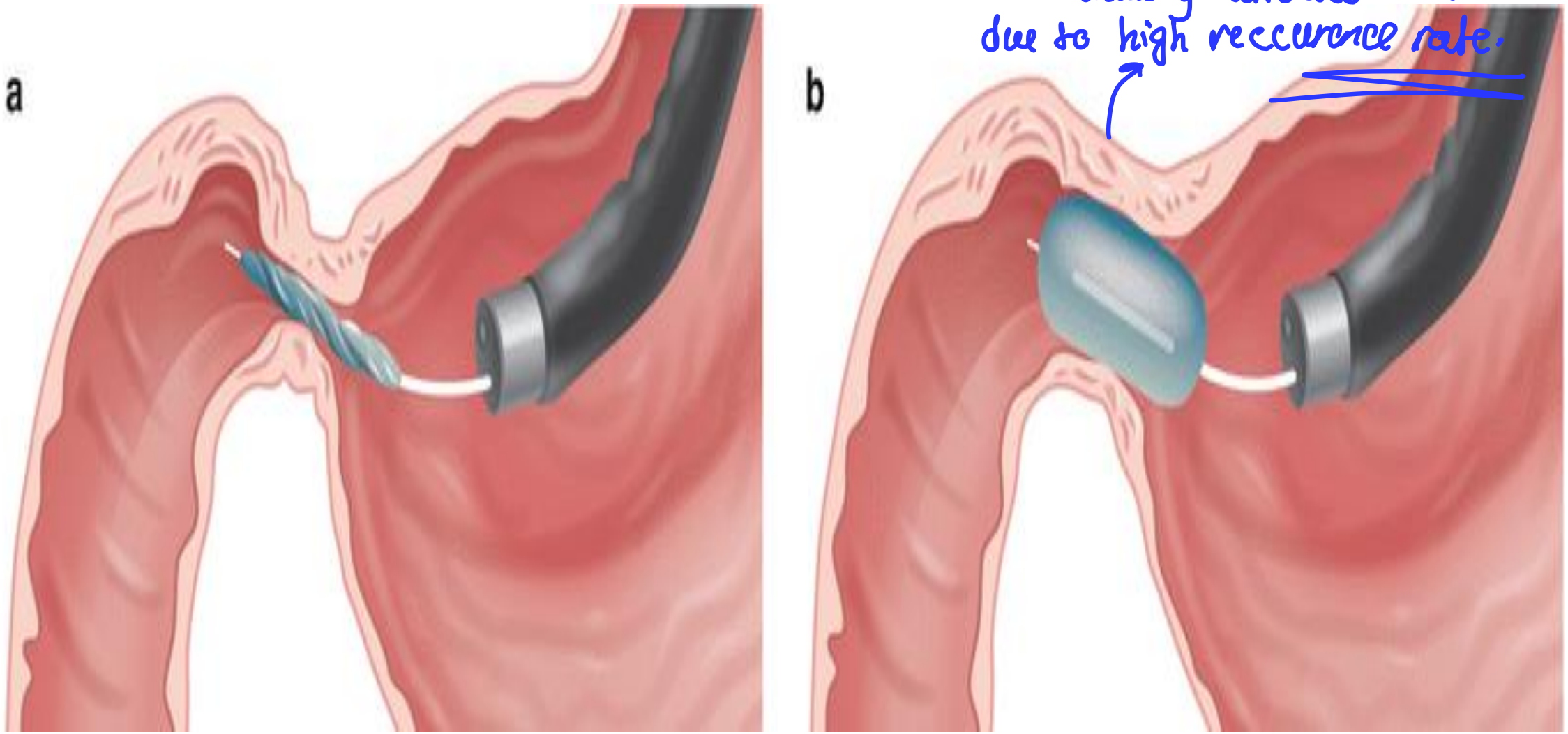
- **Interventional Treatment** :

1. Endoscopic balloon dilatation→Multiple dilatations are required, durable response seen in 70-80% of cases Immediate improvement of symptoms → Perforation risk 3-7%.

Illustration of Endoscopic balloon dilatation procedure.

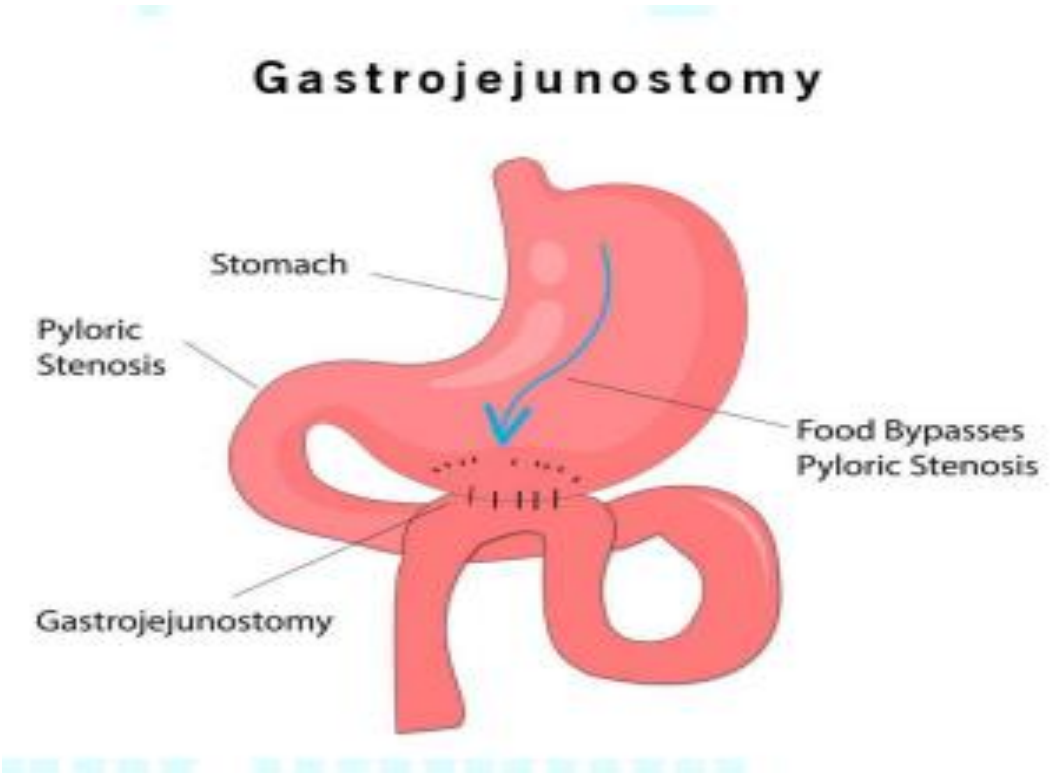
*GI

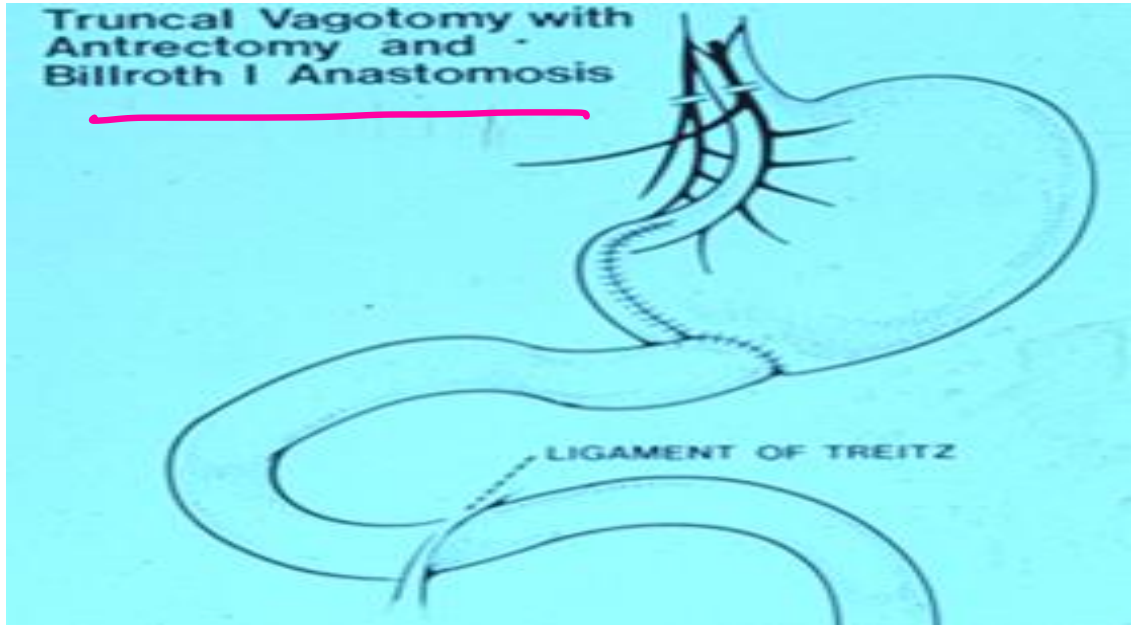
usually unsuccessful due to high reccurence rate.

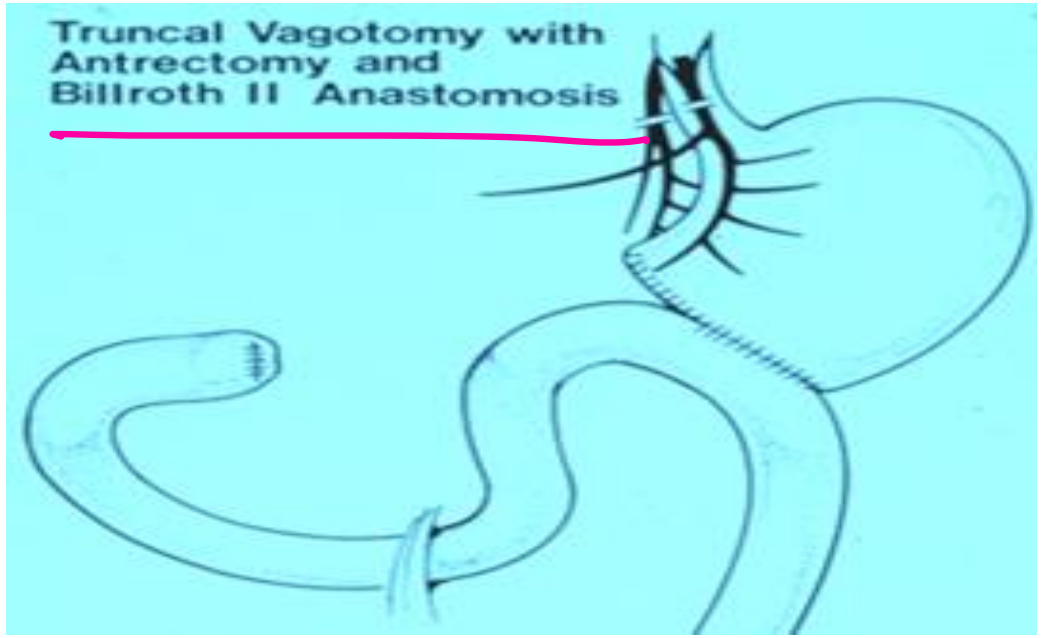


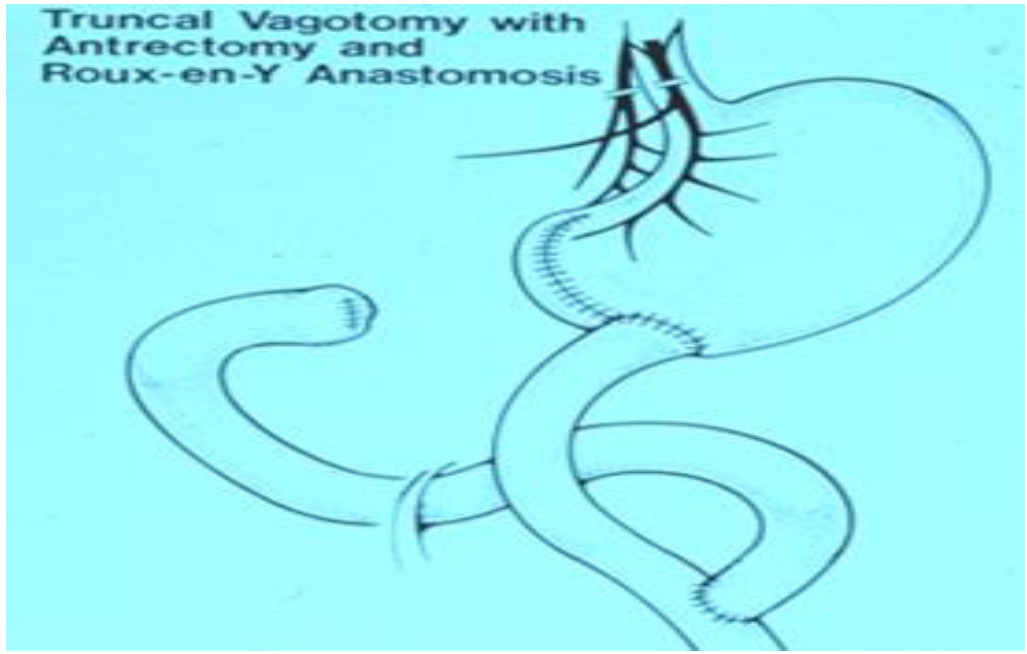
Surgical treatment options for GOO caused by peptic ulcer :

- **Pyloromyotomy** : This procedure involves cutting the muscle at the pylorus to widen the opening and relieve the obstruction.
- **Gastrojejunostomy** : This procedure involves creating a new opening between the stomach and the jejunum, bypassing the duodenum.
- **Vagotomy** : This procedure involves cutting the vagus nerve which reduces the amount of acid produced by the stomach.




Truncal Vagotomy with
Antrectomy and
Billroth I Anastomosis
LIGAMENT OF TREITZ


Truncal Vagotomy with
Antrectomy and
Billroth II Anastomosis


Truncal Vagotomy with
Antrectomy and
Roux-en-Y Anastomosis

Thank you