UPPER GIT BLEEDING

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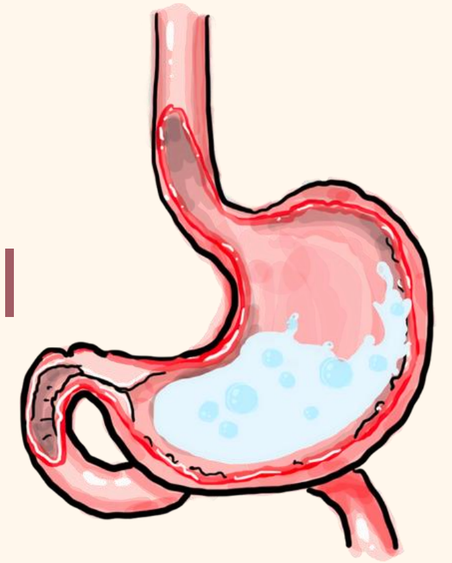
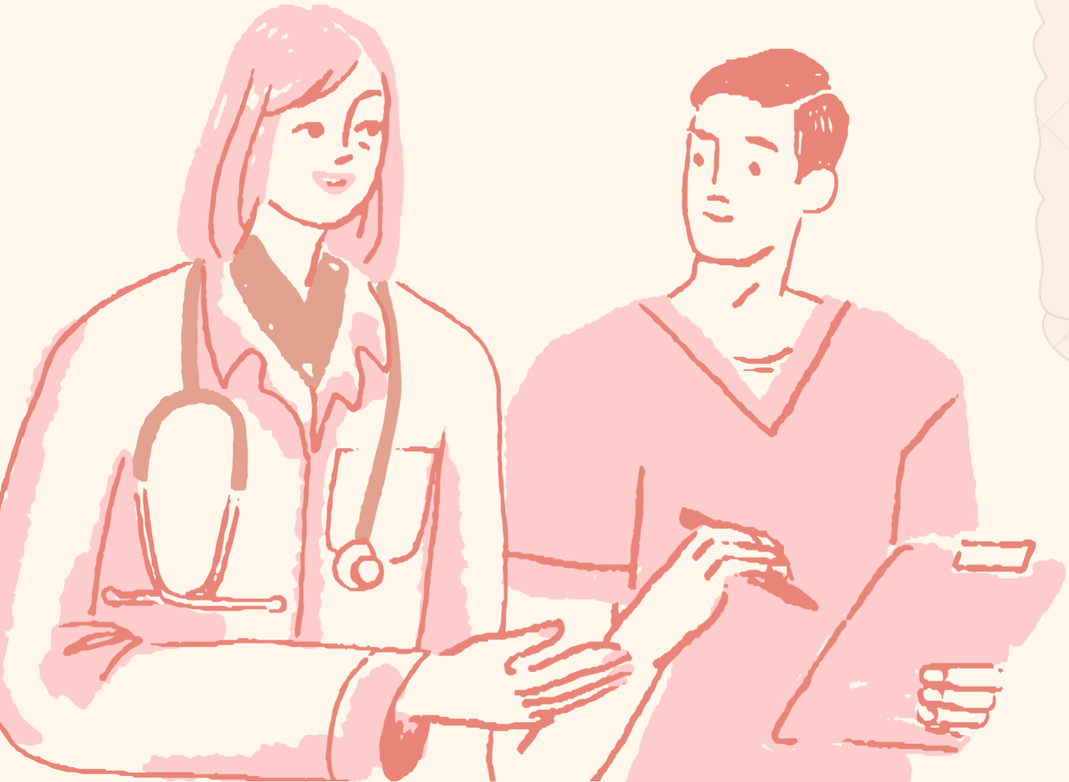
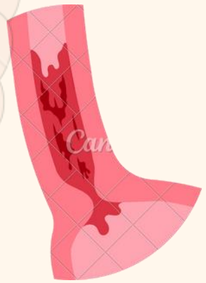
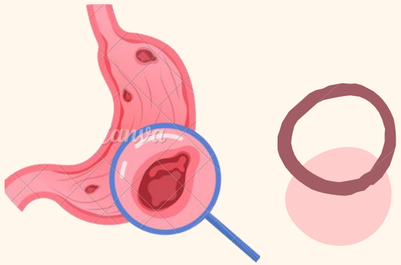




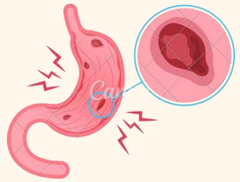




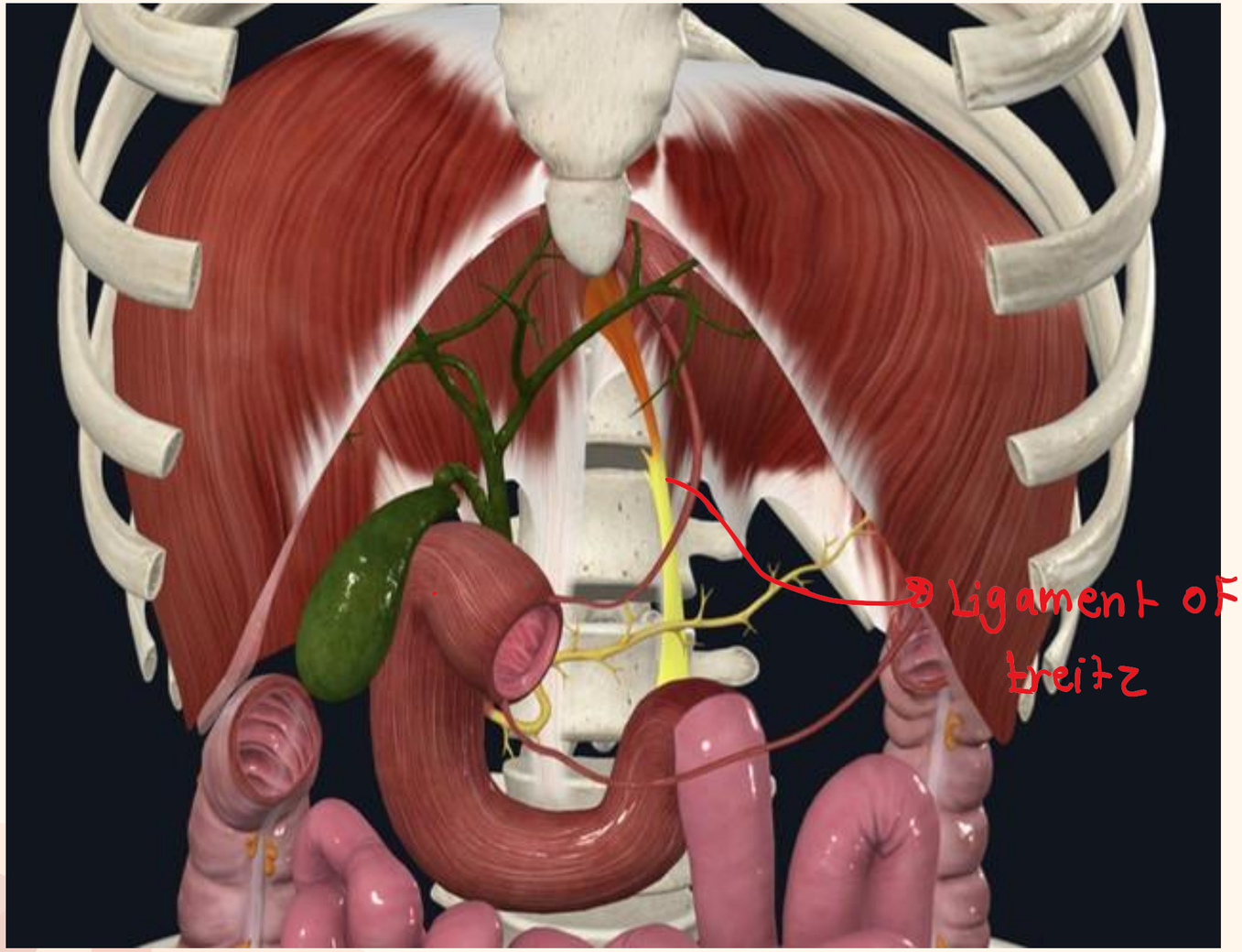
DEFINITION OF UGIT BLEED AND EPIDEMIOLOGY :

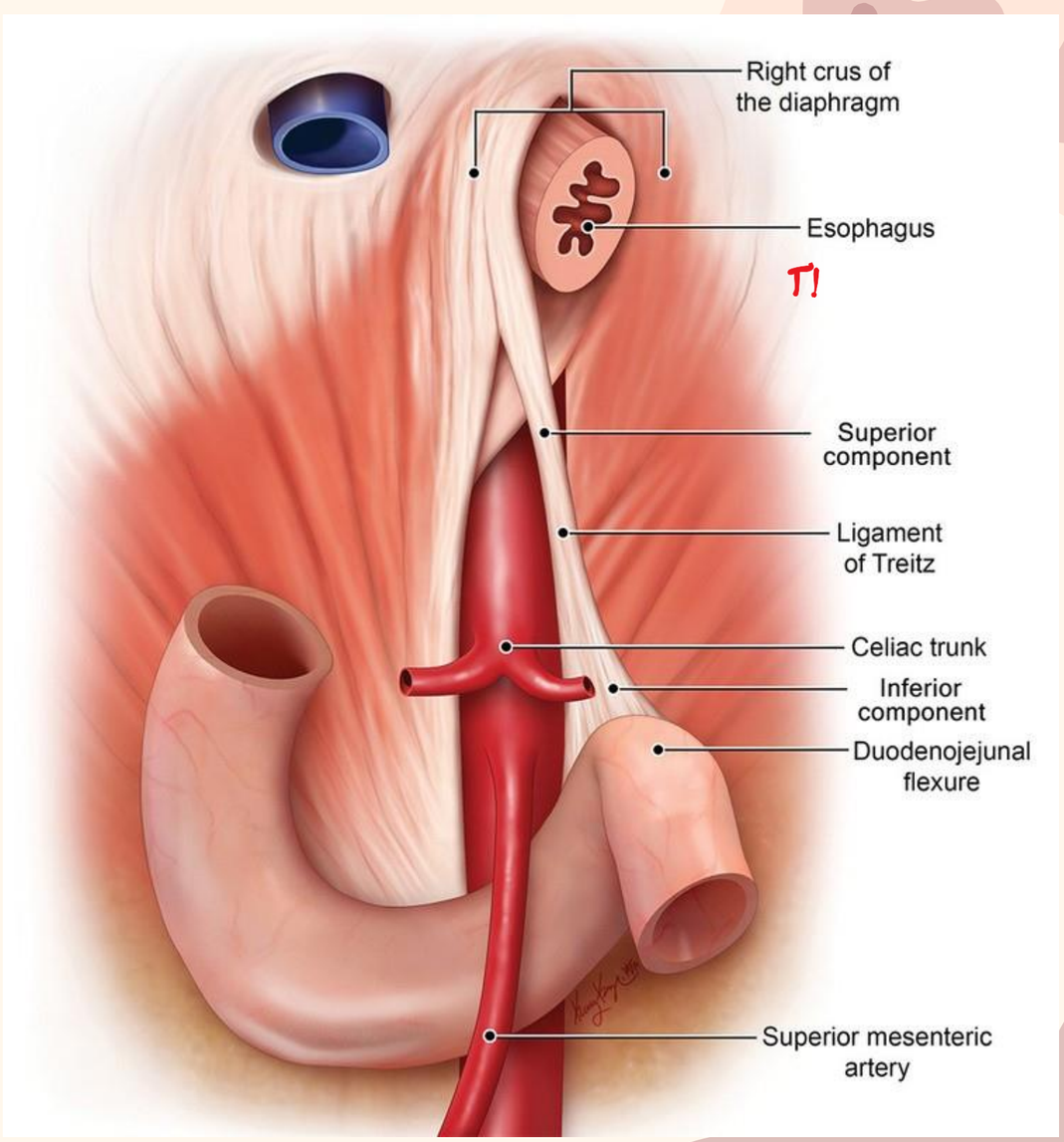
- Bleeding into the lumen of the proximal GI tract
- oesophagus , stomach and duodenum
- source of bleeding is proximal to the ligament of Treitz(suspensory muscle of the duodenum)
- account for 70-80%of GIT haemorrhage and 4 times more common than lower GIT bleeding
- It’s life threatening abdominal emergency , common cause of hospitalisation

- attended by significant mortality and hospital mortality account for 5%in the region
- increase risk for patient with ischemic heart disease , renal and c.liver disorders
- higher incidence in males and older ages (more use to NSAIDS)
- High incidence in patient Using NSAIDS (inhibit mucosal PGs which is protective factor that inhibit Hcl and increase mucous and bicarbonate)and anticoagulants



ANATOMY :


Ligament of Treitz


Right crus of the diaphragm
Esophagus
T!
Superior component
Ligament of Treitz
Celiac trunk
Inferior component
Duodenojejunal flexure
Superior mesenteric artery

UGITB CLASSIFICATION :

THE SOURCE OF BLEEDING EITHER ARTERIAL (NON VARICEAL) OR VENOUS (VARICEAL)IN THE UGIT I.E. WITHIN EITHER OESOPHAGUS , STOMACH , DUODENUM , UGITB ISN'T A DISEASE IT A COMPLICATION OF A VERIETY OF DISEASES THAT CAUSE EITHER VENOUS OR ARTERIAL BLEEDING .

BLOOD SUPPLY TO UGIT :

1)OESOPHAGUS :

ARTERIAL :

1) CERVICAL : INFERIOR THYROID ARTERY

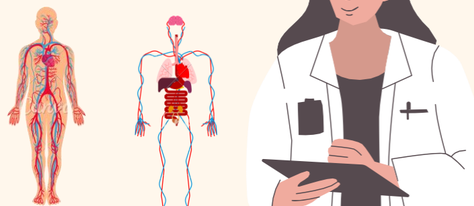
2)THORACIC : OESOPHAGEAL BRANCHES FROM TAORTA

3) ABDOMINAL : OESOPHAGEAL BRANCHES FROM SHORT GASTRIC ARTERIES

VENOUS :

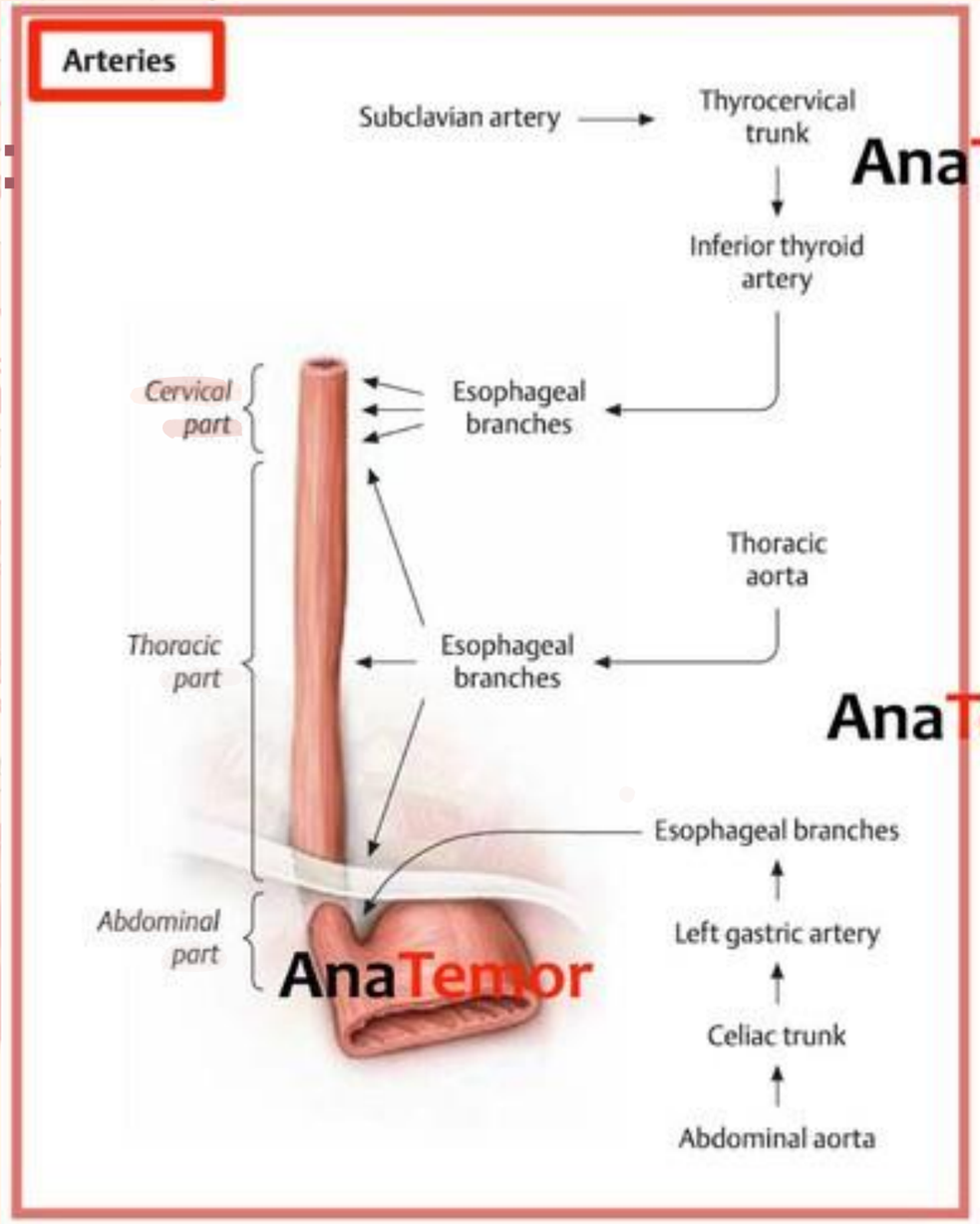
1) CERVICAL : INTO BRACHIOCEPHALIC VEIN

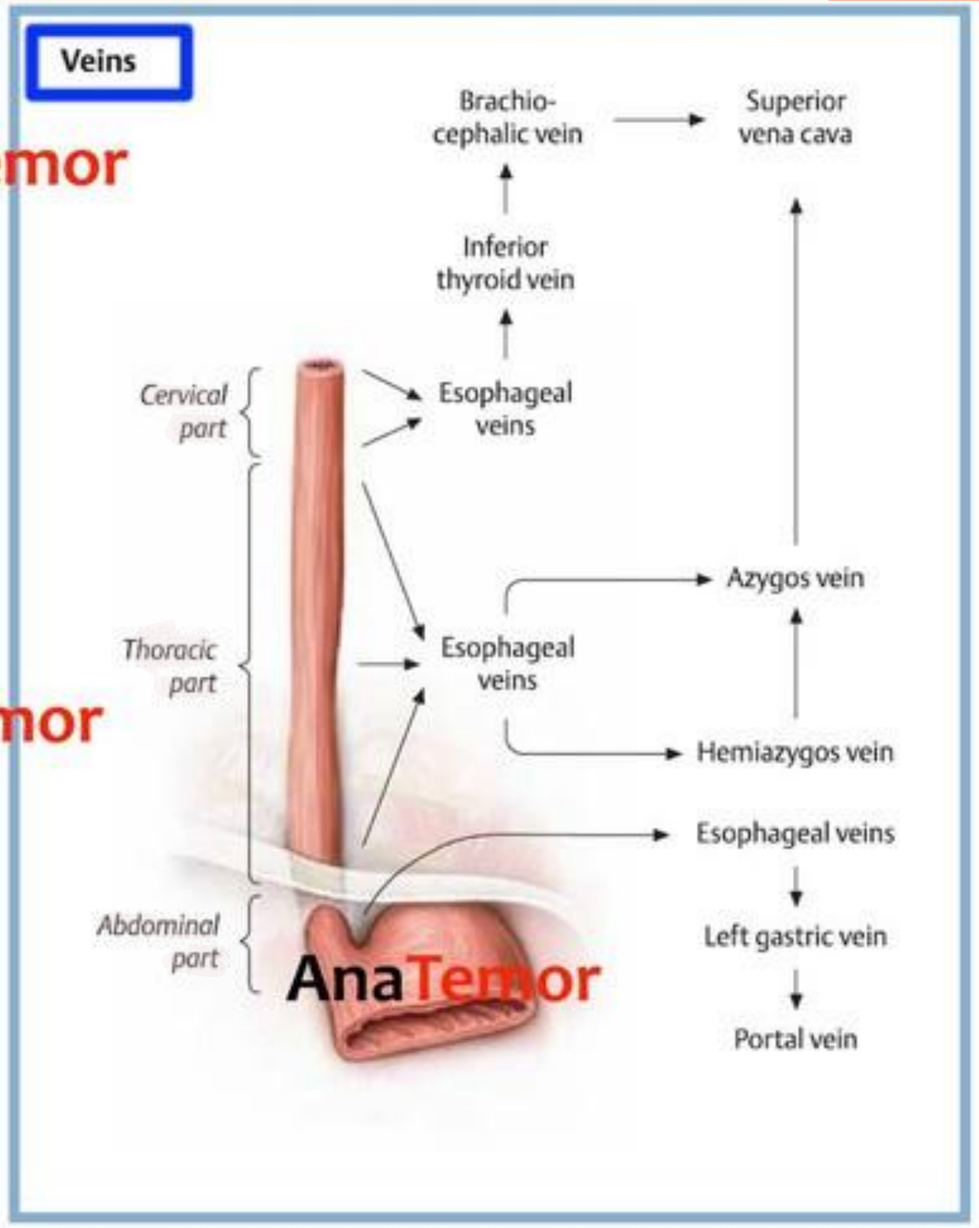
S 2)THORACIC : R.T : AZYGOZ VEIN AND L.F B)EXTRAABDOMINAL : LEFT GASTRIC VEIN THEN TO PORTAL VEIN



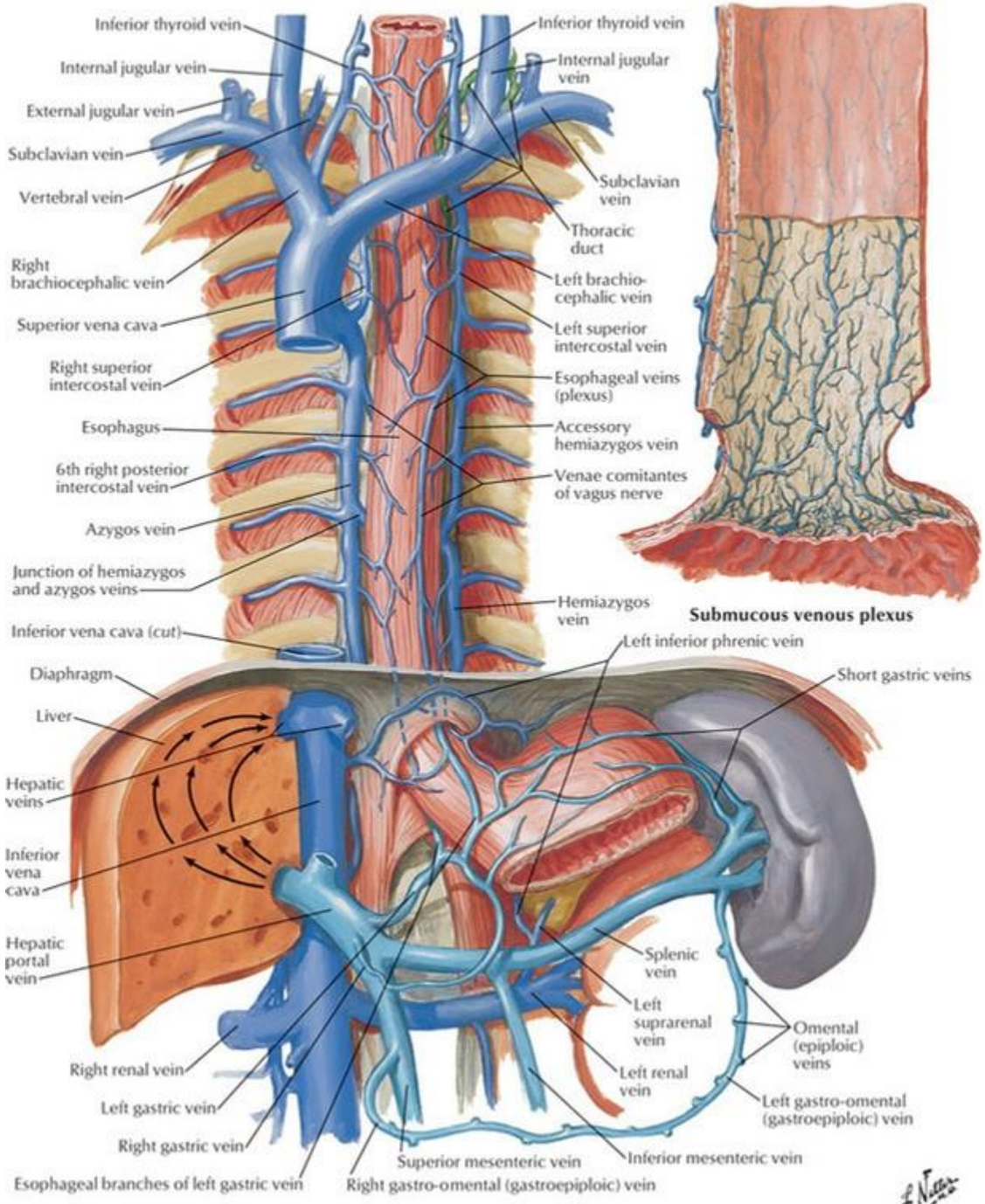
BLOOD SUPPLY TO UGIT :

1) OESOPHAGUS :

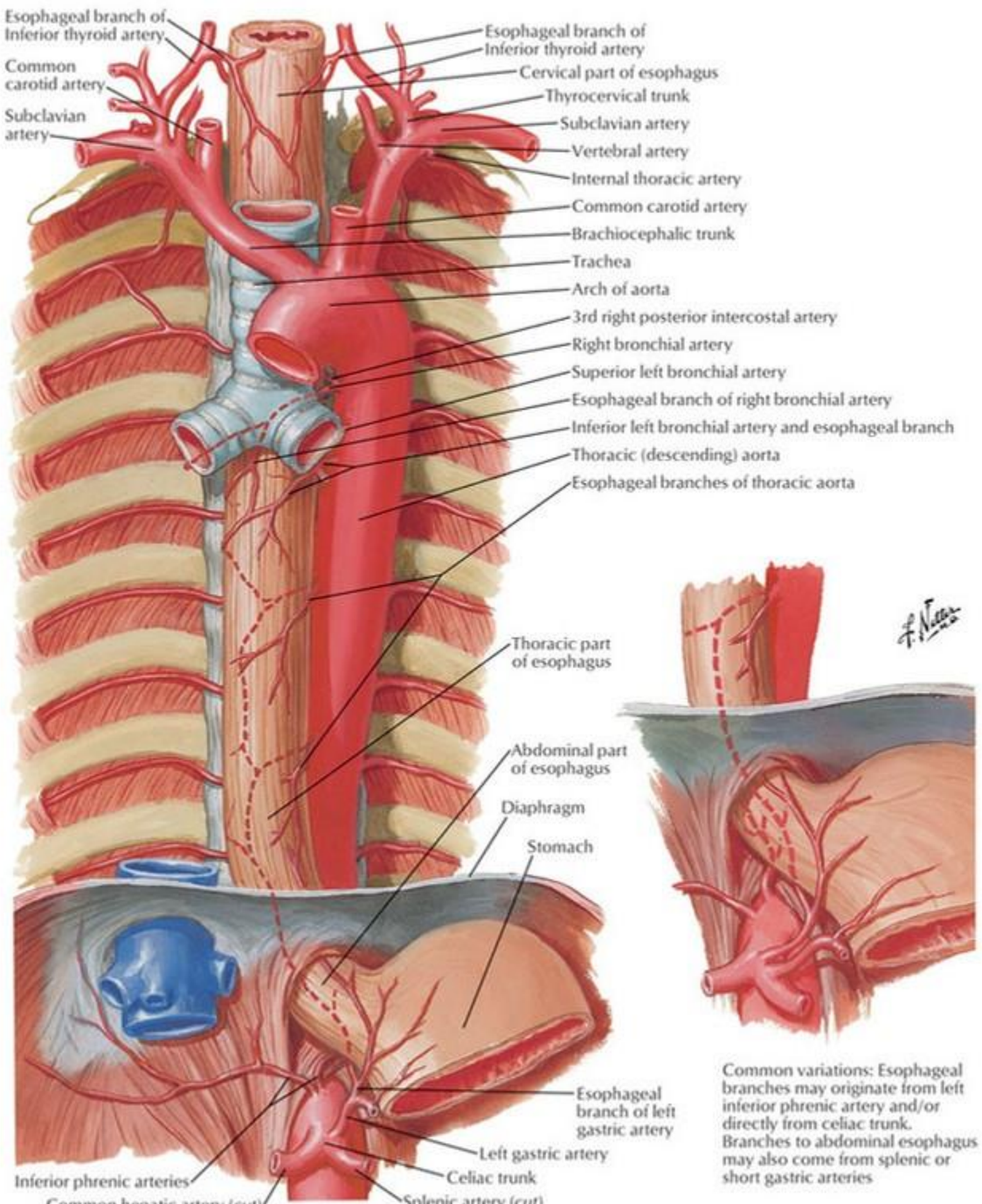




Veins of Esophagus



Arteries of Esophagus



Common variations: Esophageal branches may originate from left inferior phrenic artery and/or directly from celiac trunk. Branches to abdominal esophagus may also come from splenic or short gastric arteries

BLOOD SUPPLY TO UGIT :

2)STOMACH :

ARTERIAL :

1) ALONG THE LESSER CURVATURE:

RT GASTRIC FROM HEPATIC PROPER FROM COMMON HEPATIC ARTERY FROM CELIAC TRUNK FROM ABDO AORTA

LT GASTRIC FRON CELIAC TRUN FROM ABDOM AORTA

2)ALONG GREATER CURVATURE :

RT GASTRO EPIPLOIC(OMENTAL) FROM GASTRO DOUDENAL ARTERY FROM HEPATIC ARTRY FROM CELIA TRUNK

LEFT GASTRO EPIPLOIC ARTERY FROM SPLENIC FROM CELIAC TRUNK

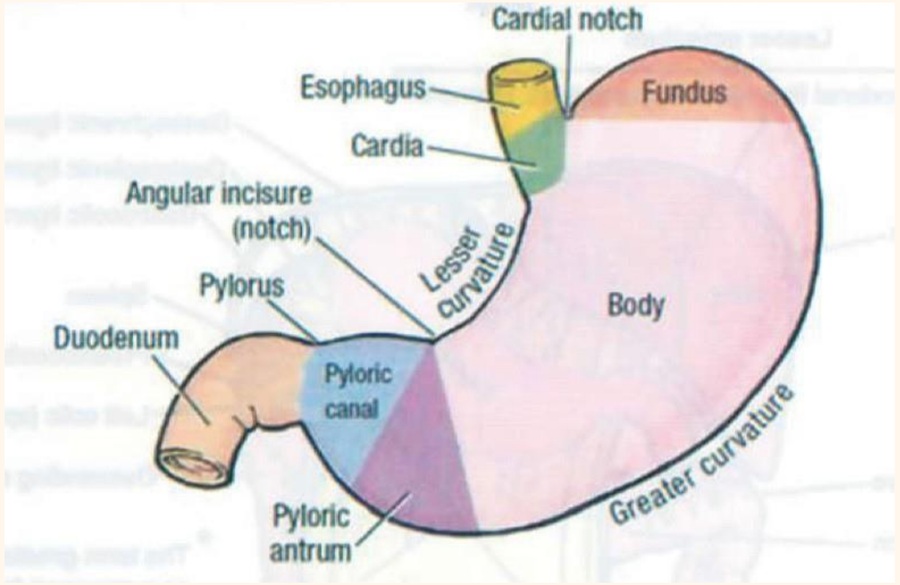
3)TO THE FUNDUS : SHORT GASTRIC ARTRIES FROM SPLENIC THROUGH GASTROSPLENIC LIGAMENT

VENOUS :

1) RT AND LT GASTRIC VEINS TO THE PORTAL VEINS

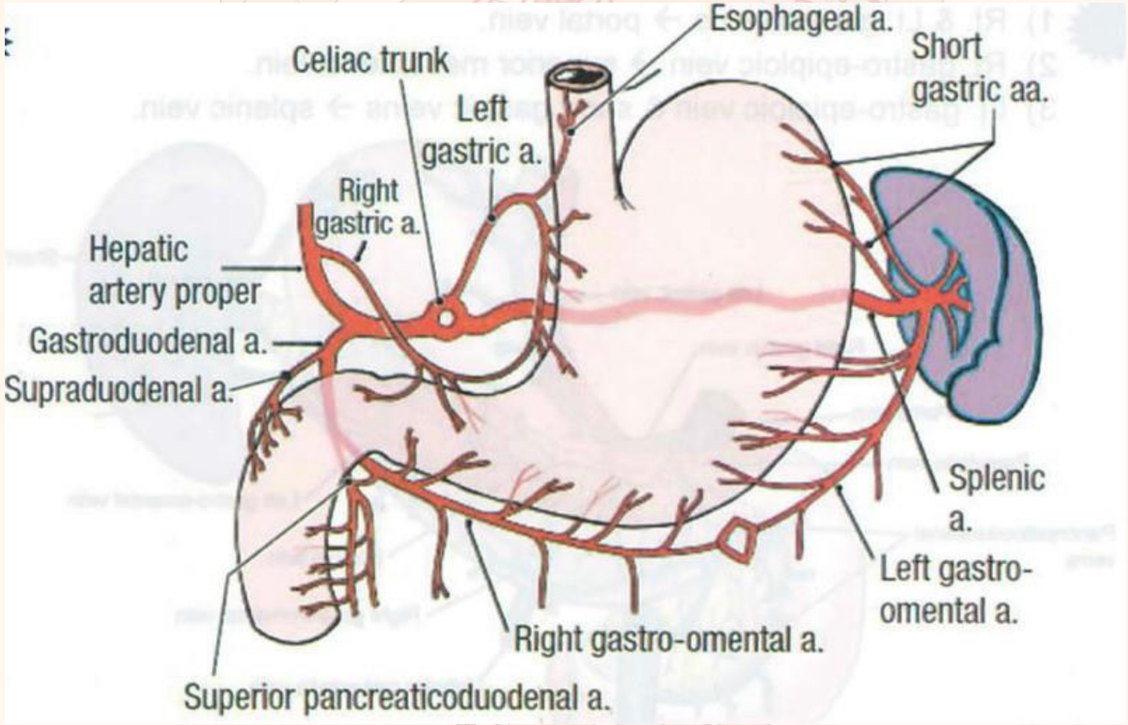
2)RT GASTRO OMENTAL VEIN TO SUPERIOR MESENTRIC VEIN WHICH UNITE WITH SPLENIC TO FORM THE PORTAL VEIN

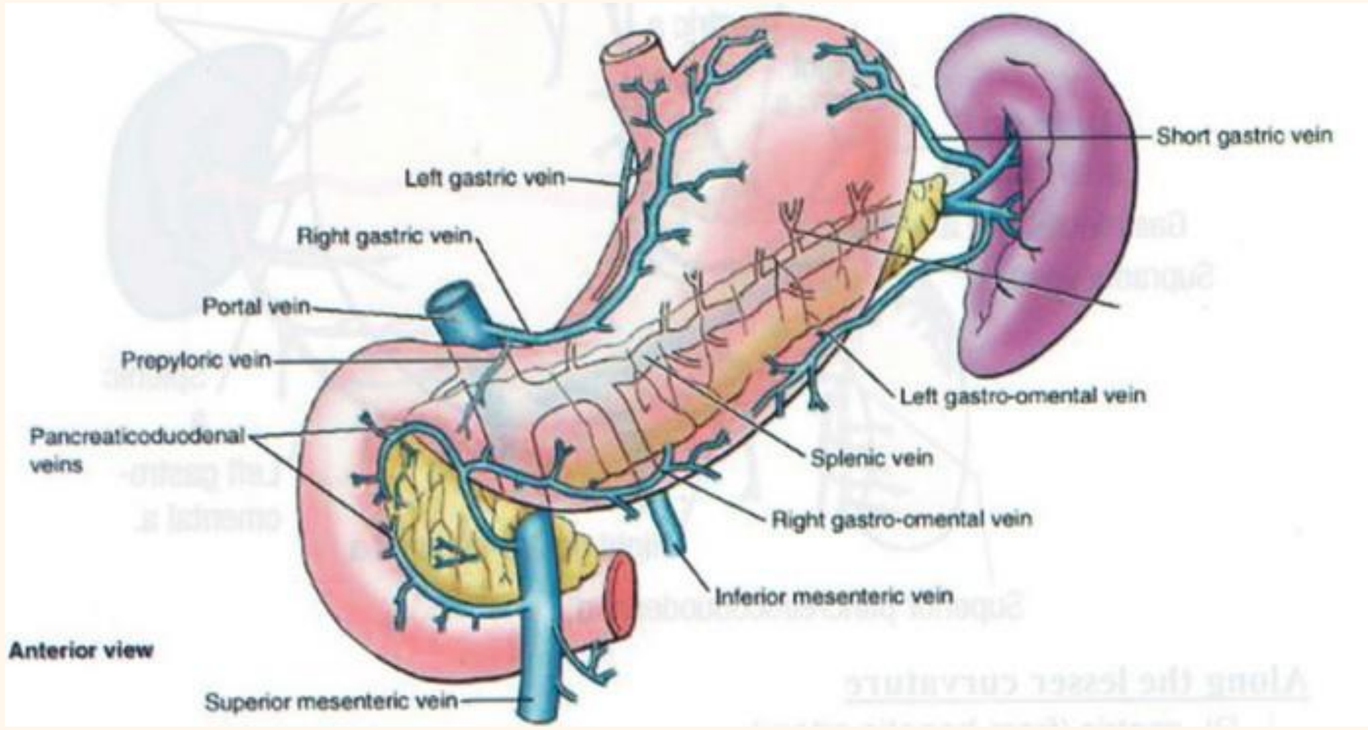
3) LT GASTRO OMENTAL VEIN AND SHORT GASTRIC VEINS TO SPLENIC VEIN



BLOOD SUPPLY TO UGIT :

2)STOMACH :





BLOOD SUPPLY TO UGIT :

3) DUODENUM:

ARTERIAL :

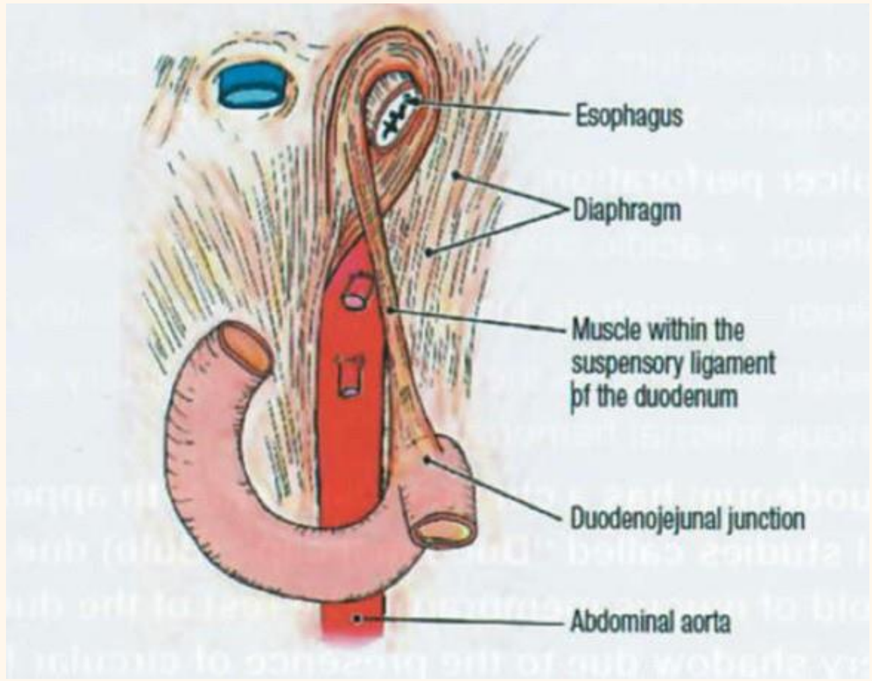
1) SUPERIOR PANCREATICO-DOUDENAL ARTERY FROM GASTRO- DOUDENAL ARTERY FROM COMMON HEPATIC FROM CELIAC FROM AORTA

2)INFERIOR PANCREATICO-DOUDENAL ARTERY FROM SUPERIOR MESENTRIC FROM THE AORTA

3)SUPRADOUDENAL ARTERY FROM GASTR- DOUDENAL ARTERY FROM HEPATIC ARTERY

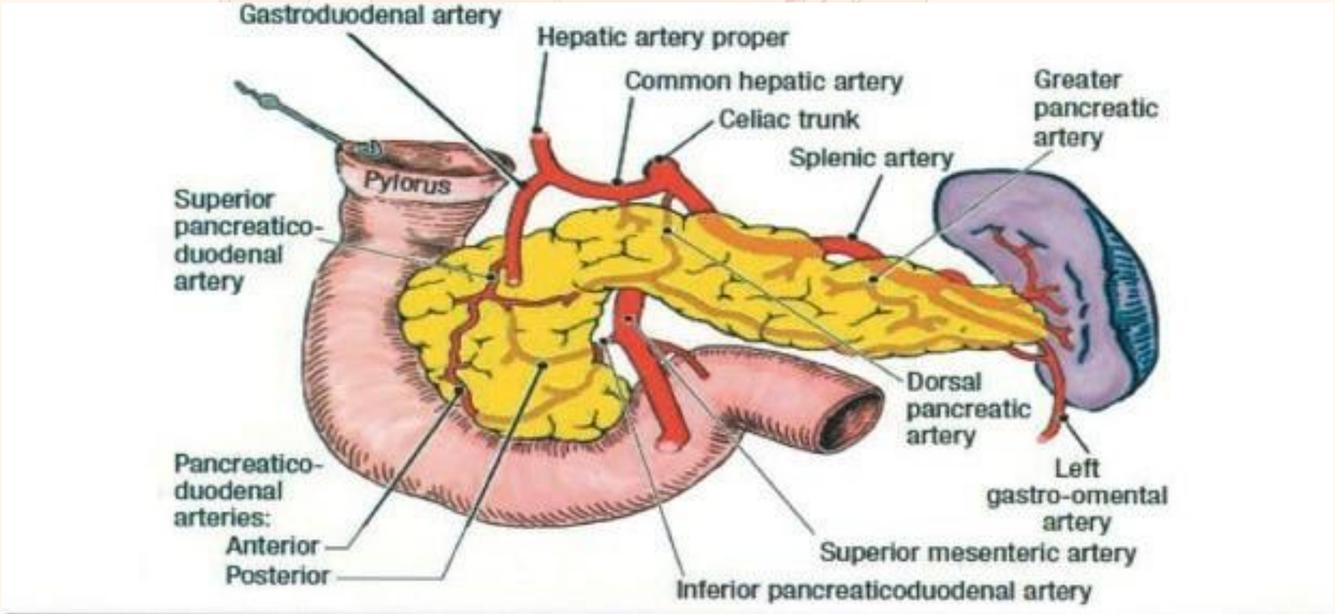
VENOUS :

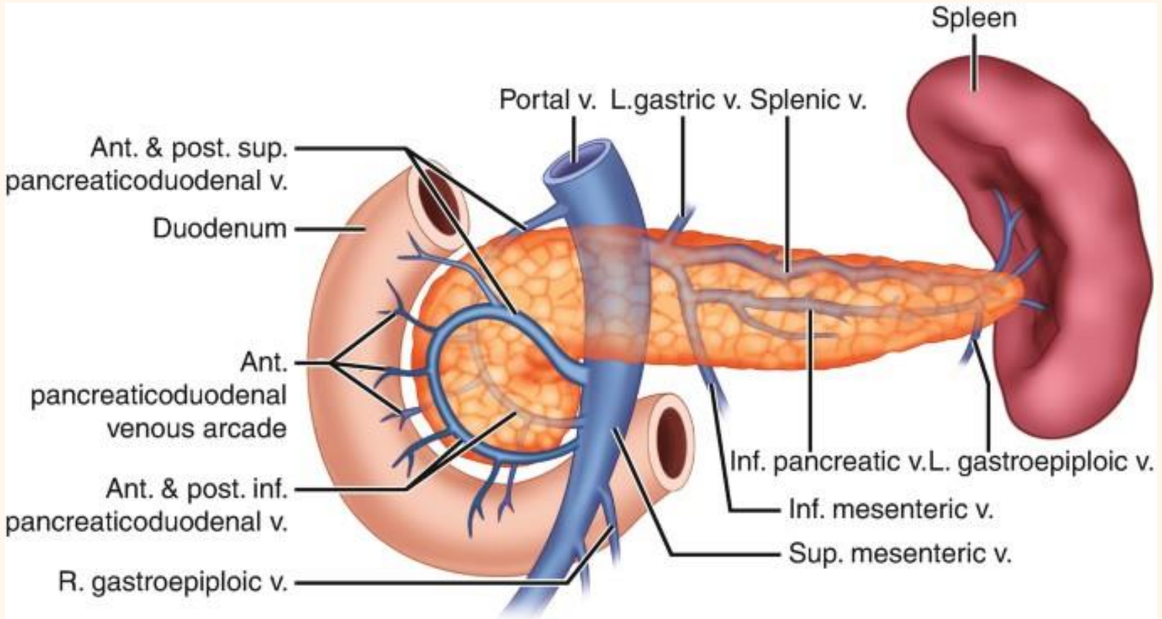
1) PANCREATODOUDENAL VEINS SUP AND INF DRAINS. INTO SUPERIOR MESSENTRIC VEIN THEN TO PORTAL SYSTEM



BLOOD SUPPLY TO UGIT :

3) DUODENUM:

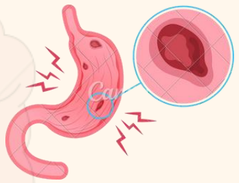




CAUSES OF UGITB :

ARTERIAL (NON VARICEAL)80%

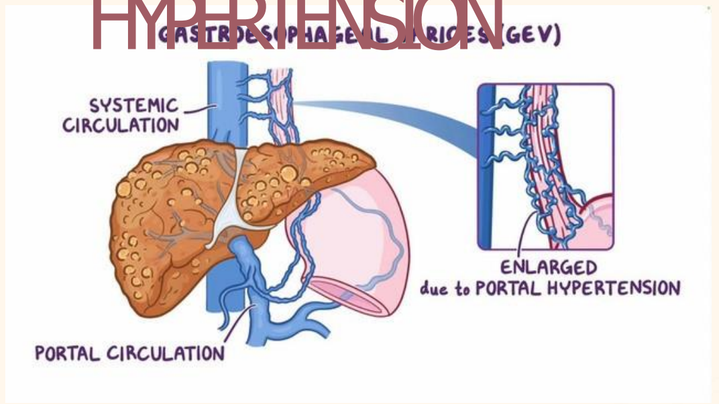
- PEPTIC ULCER DISEASE (30%) COMMON
- OESOPHAGITIS
- EROSIVE
- GASTRITIS
- DUODENITIS
- DIEULAFOY LESION
- CANCER OF OESOPHAGUS/ GASTRIC
- TRAUMA (MALLORY WEISS) HIATAL HERNIA



VENOUS (VARICEAL)20%

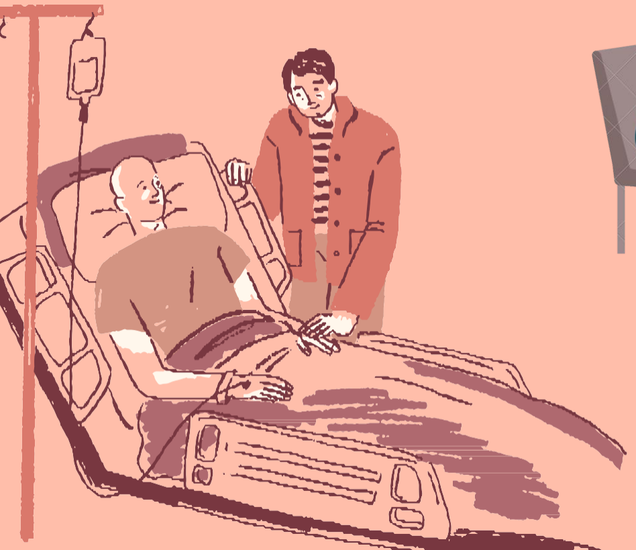
- OESOPHAGEAL COMMON
- VARICES GASTRIC VARICES

MAINLY ASSOCIATED WITH PORTAL HYPERTENSION

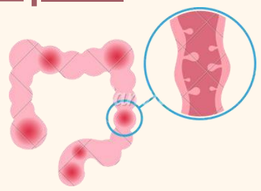


SIGNS AND SYMPTOMS OF UGITB :

- the patient can be present w/ occult. Bleed (no symptoms) or overt bleed(symptomatic)
- Anemia due to chronic blood loss
- Acute hemorrhage with significant blood loss
- Signs of circulatory insufficiency or hypovolemic shock
- Tachycardia, hypotension (dizziness, collapse, shock) syncope
- Altered mental status



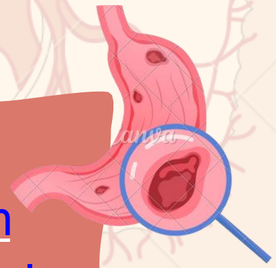
- Features of overt GI bleeding:
- Hematemesis : Vomiting blood, which may be red or coffee-ground in appearance,
- Melena Black, tarry stool with a str ong offensive odor
- Hematochezia The passage of bright red (fresh) blood through the anus (with or without stool) occur in case vigorous rate of bleed from UGITB
- epigastric pain
- dyspepsia
- dysphagia
- diffuse abdominal pain
- weight loss
- jaundice



SIGNS AND SYMPTOMS OF UGITB :

- Hematemesis :Most commonly due to bleeding in the upper GI tract (e.g., esophagus, stomach)
- Melena :Most commonly due to bleeding in the upper GI tract ,Can also occur in bleeding from the small bowel or the right colon.
- Hematochezia: Most commonly due to bleeding in the lower GI tract (e.g., in the distal colon) Rapid passage of blood from the upper GI tract may also result in hematochezia
- Unexplained iron deficiency anemia (e.g., in men or postmenopausal women) should raise suspicion for GI bleeding

Bleeding from the upper respiratory tract (e.g., nocturnal nosebleeds) can be mistaken for GI bleeding because the blood can be swallowed and vomited or appear in the stool as melena. Careful examination and history taking is the key to differentiating respiratory sources of bleeding from GI ones



CLINICAL APPROACH FOR UGITB :

1) MANAGEMENT FOR SUSPECTED UGITB AND SEEK FOR DIAGNOSIS AND TREATMENT :

The patient with suspected UGITB (HEMATEMESIS , MELENA , hematochezia)

Initially assess the severity of bleeding and according to it give fluid and blood transfusion if needed this include : Vital signs Pulse , BP, temp . Res.rate

Unstable patient (class 4 Hemorrhage):DO ABCDE

- Moderate bleeding:
orthostatic hypotension
blood loss between 30%-40%
(crystalloid and blood)

- Severe bleeding:
patient in shock, Abd
severely decreased
blood loss more than 40%
(crystalloid and blood

Stable patient :class 1/2 Hemorrhage

- Mild bleed :
normal BP blood loss less
than 30%(crystalloid)

CLINICAL APPROACH FOR UGITB :

Start basic management for all patient (stable and unstable)

- Ensure patient is NPO.
- Insert two large-bore peripheral IVs (for possible fluid resuscitation and blood transfusion)according to patient state as determined previously
- obtain blood samples for laboratory studies (e.g., CBC, type and screen)
- Conduct a focused history and examination (including DRE)
- Risk stratify to guide further management
- Pretreatment (e.g., IV PPI)
- Anticoagulant reversal (e.g., for life-threatening bleeding if INR more THAN 2.5)
- Withholding antithrombotic agents

HX AND EXAMINATION OF UPPER GITB :

History in upper GI (UGI) bleeds

1) profile of patient

- History of presenting complaint
  - Haematemesis – can be bright red OR coffee-ground
  - Melaena – distinctive smell, tar-like
    - Volume of blood loss in either case
  - Dyspesia
  - Dizziness
  - Chest pain
  - Prolonged vomiting/retching
  - Constitutional symptoms
- Past medical history
  - Previous GI bleed
  - Known PUD/varices
  - Malignancy
  - Liver disease
  - Known cardiovascular/respiratory disease (fitness to undergo sedation and/or intubation for endoscopy)
- Medications
  - NSAIDs
  - Steroids
  - Anticoagulants
- Allergies
- family history
- Social history
  - Alcohol consumption

History of epistaxis and emptysis to role out the GI source of bleeding

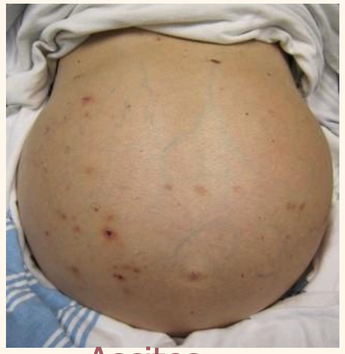
HX AND EXAMINATION OF UPPER GITB :

2) general examination :Patients with chronic upper GI bleeding usually appear fatigue, on contrast depending upon the amount of blood loss, patient appear in distress and shock in acute upper GI bleeding

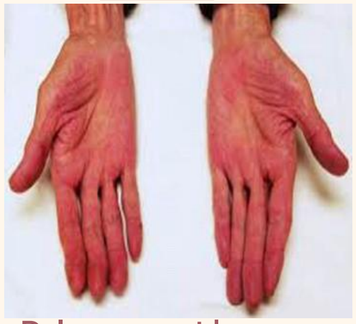
- inspection : Pale , Lymph node swelling

Feature of chronic liver disease , jaundice , cachexic

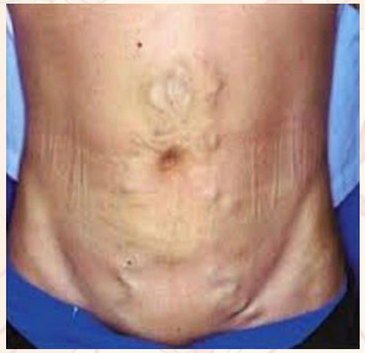
Cyanosis ,Abdominal distension ,Dilated vein ,Visible peristalsis ,Sign of dehydration (dry tongue , sunken eyes)



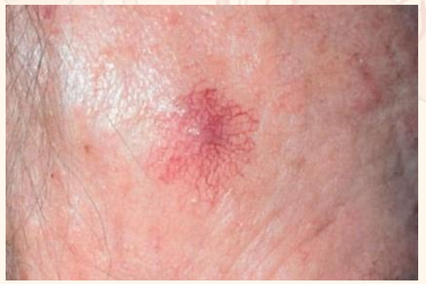
Ascites



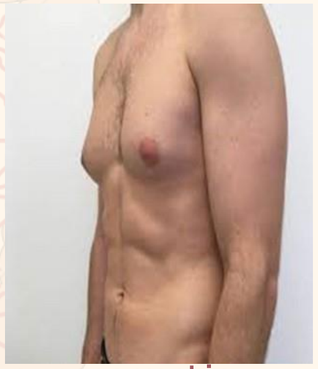
Palmar erythema



caput medusae



spider naevi

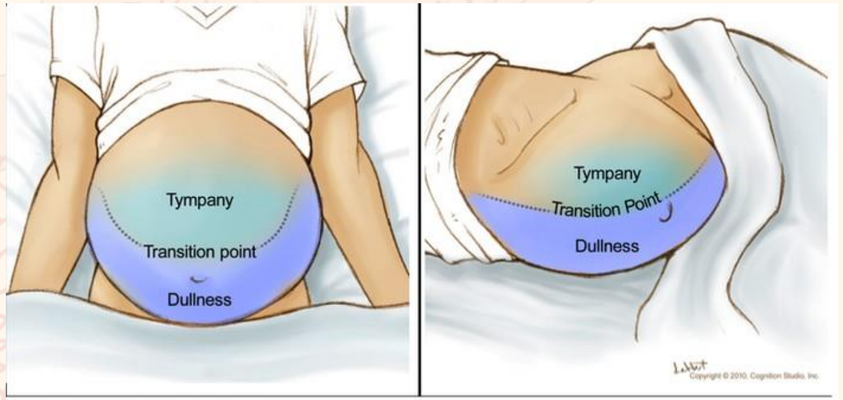


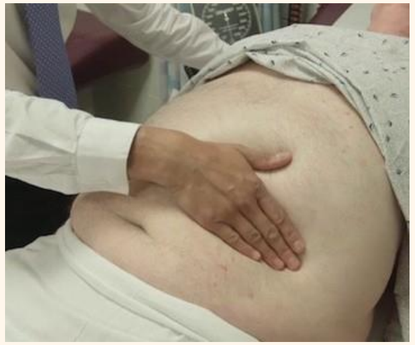
gynecomastia

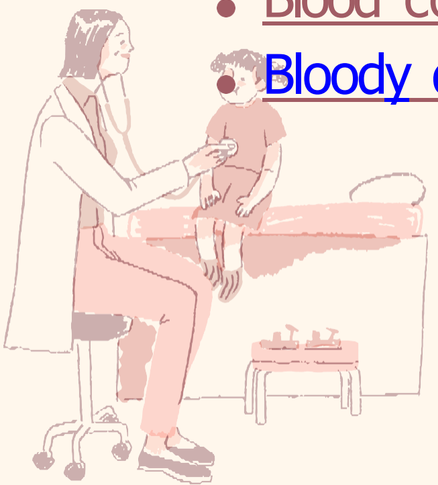
leukonychia

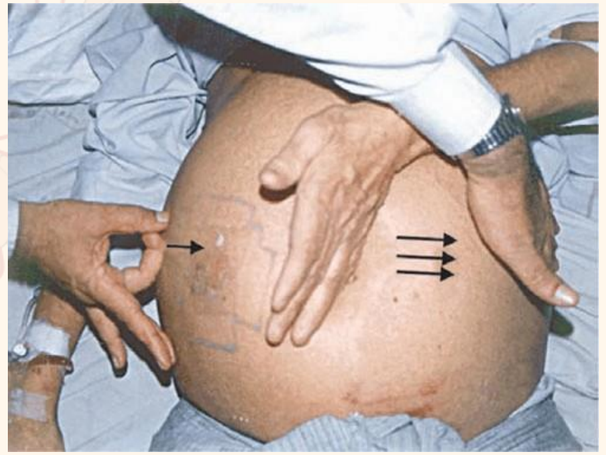
HX AND EXAMINATION OF UPPER GITB :

- palpation :tenderness / abdominal mass (in UGITB Abdominal tenderness)
- percussion :shifting dullness ,transmitted thrill(bleed due to liver disease may associated with Ascites)
- Auscultation :bowel sound and bruit (in UGIT Bleed increase the bowel sound)
- Rectal examination :
- Occult blood
- Gross blood
- Bright red blood per
- rectum Melena
- Burgundy stools
- Blood coating stools versus within stools
- Bloody diarrhea









LABORATORY STUDIES :

- _TESTS TO ASSESS THE SEVERITY OF GI BLEEDING :
  - CBC: HB, HCT LESS THA 35%, LEUKOCYTOSIS, PLATELET COUNT
  - COAGULATION PANEL

  - BMP: ↑ BUN/CR RATIO SUGGESTS A BRISK UGIB (MAINLY IF THE RATIO MORE THAN 30/1)

- BLOOD TYPE AND CROSSMATCHING
- LIVER CHEMISTRIES: IN SUSPECTED ESOPHAGEAL VARICEAL HEMORRHAGE

Based of clinical observation the A risk stratification score used to triage management in hemodynamically stable patients with upper gastrointestinal bleeding called the Glasgow-Blatchford score

THE GLASGOW-BLATCHFORD SCORE

Glasgow-Blatchford Score

Glasgow-Blatchford Bleeding Score (GBS) helps identify which patients with upper GI bleeding (UGIB) may be safely discharged from the emergency room.

Glasgow-Blatchford Score	
Blood urea nitrogen (mg/dL)	
18.2 to <22.4	2
22.4 to <28.0	3
28.0 to <70.0	4
≥70.0	6
Hemoglobin (g/dL)	
12.0 to <13.0 (men); 10.0 to <12.0 (women)	1
10.0 to <12.0 (men)	3
<10.0	6
Systolic blood pressure (mmHg)	
100–109	1
90–99	2
<90	3
Heart rate (beats per minute)	
≥100	1
Other markers	
Melena	1
Syncope	2
Hepatic disease	2
Cardiac failure	2

Source : Harrison 20th Ed

MEDREWIND

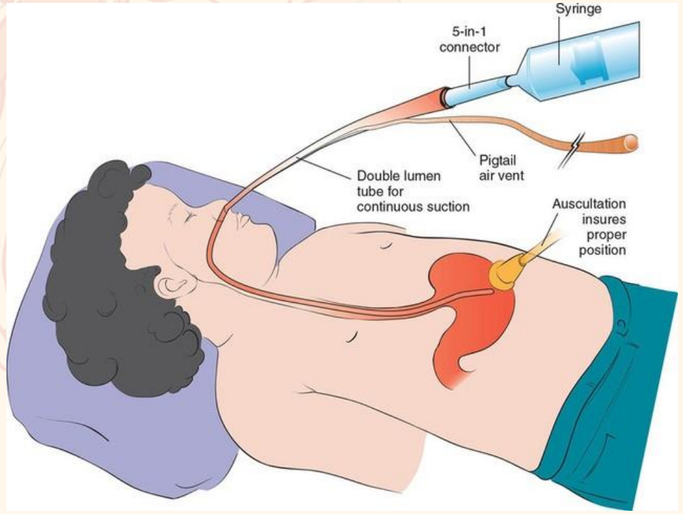
- Score 0: low-likelihood of rebleeding or need for urgent intervention
- Score ≥ 1: higher likelihood of rebleeding and/or need for urgent intervention

NASOGASTRIC ASPIRATE (NG ASPIRATE):

Nasogastric aspirate (NG aspirate)

This test is not routinely recommended other than as an adjunct in patients with hematochezia with only moderate PTP of UGIB as the source.

- Procedure: Instill 200–300 ml of warm isotonic saline via NG tube, then aspirate gastric contents for inspection.
- Findings
  - Positive: Bright red blood or coffee-grounds; active UGIB confirmed
  - Inconclusive: Nonbloody and nonbilious
  - Negative: Nonbloody and bilious; active UGIB less likely



ENDOSCOPY :

- Endoscopy:

These procedures allow for bleeding source identification, diagnostic biopsies (e.g., for gastric or colorectal cancer), and hemostatic interventions (e.g., epinephrine injection, vessel clipping). They should ideally be performed within 24 hours of admission

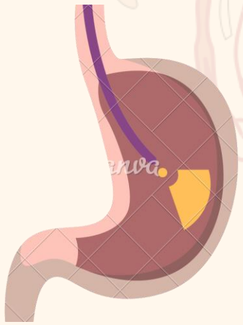
- Upper endoscopy: a procedure during which a flexible fiber-optic instrument is passed through the mouth to visualize the inner layer of the upper GI tract up to the duodenal papilla
- Colonoscopy: a procedure during which a flexible fiber-optic instrument is passed through the anus to visualize the mucosa of the colon
- Angiography :Indications:

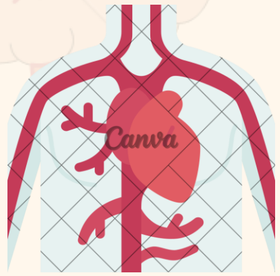
Consider as the initial test in patients with suspected LGIB and hemodynamically instability refractory to resuscitation .

Further workup of patients with ongoing bleeding and negative endoscopy

CT angiography (CTA): is a type of medical test that combines a CT scan with an injection of a special dye to produce pictures of blood vessels and tissues in a part of your body.

allows for rapid source localization to help target hemostatic interventions (e.g., angioembolizationor surgery)





ENDOSCOPY :

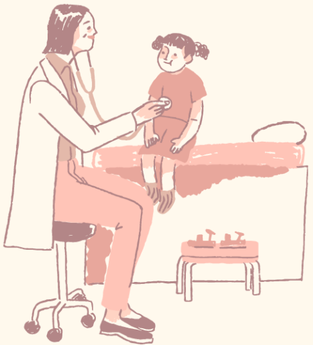
- EGD:

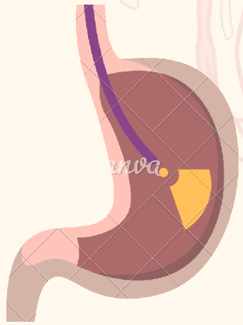
Preparation:

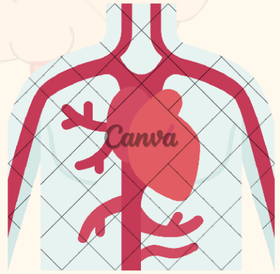
- Bowel preparation medication is not routinely required.
- Fasting (NPO) for > 2 hours (for clear liquids) and > 6 - 8 hours (for solids) is ideal.
- Consider the following to improve visualization fi large amounts of blood, clots, or food suspected in the upper GI tract
- • NG tube lavage and suction
- • Prokinetic agents: e.g., erythromycin DOSAGE

Timing :

- • Most patients: within 24 hours of presentation
- • Suspected esophageal variceal bleeding: as soon as possible in unstable patients and within 12 hours in all other patients
- • Findings and further management (see "Treatment" for details)
- • Source of GI bleeding identified (positive EGD) &A:ttempt endoscopic hemostasis. • Source of GI bleeding not identified (negative or nondiagnostic EGD)
- • Hemodynamically stable patients with hematochezia or melena: Perform colonoscopy if not performed as the first-line intervention): consider evaluation for small bowel bleeding. (301
- • Hemodynamicallyunstablepatientswithongoingbleeding:Considerangioembolization.







ENDOSCOPY :

Colonoscopy :

- Preparation:

Rapid bowel preparation, e.g., polyethylene glycol solution DOSAGE : preferred for patients with acute LGIB requiring urgent colonoscopy and are stable enough to tolerate it.

• Standard bowel preparation: for nonurgent or outpatient colo

- Timing:

•High-risk clinical features and/or ongoing GI bleeding: within 24 hours of presentation (after rapid bowel prep)

All other inpatients: at the next available opportunity (after rapid bowel prep)

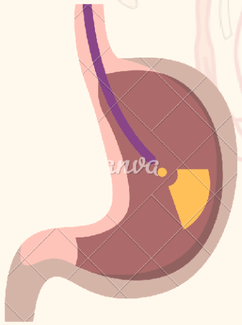
- Findings and further management :

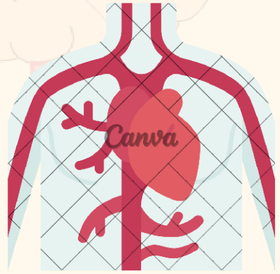
Source of GI bleeding identified :Attempt endoscopic hemostasis.

Negative (nondiagnostic) colonoscopy

• Hemodynamically stable patients: Evaluate for small bowel bleeding.

• Hemodynamically unstable patients with ongoing bleeding: Consult surgery or angioembolization.





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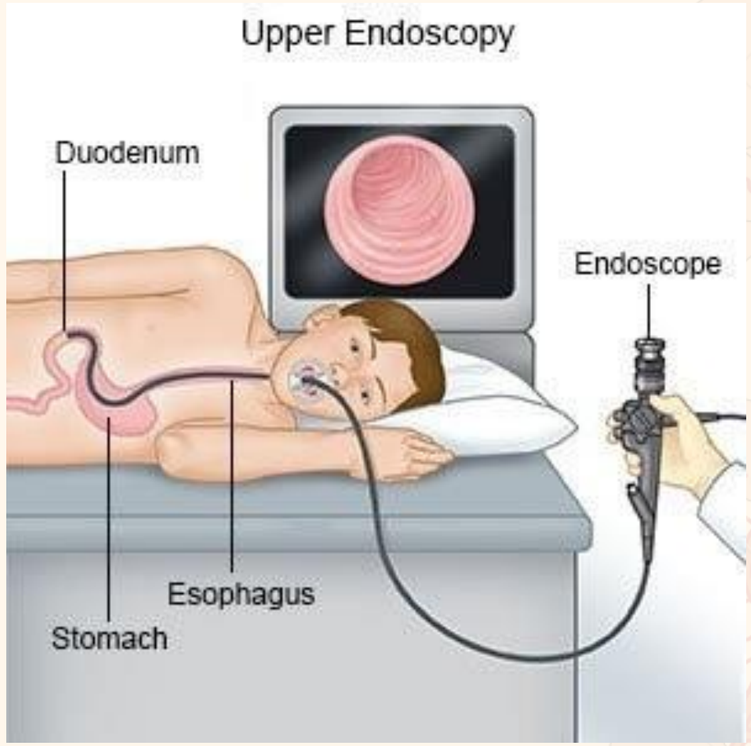
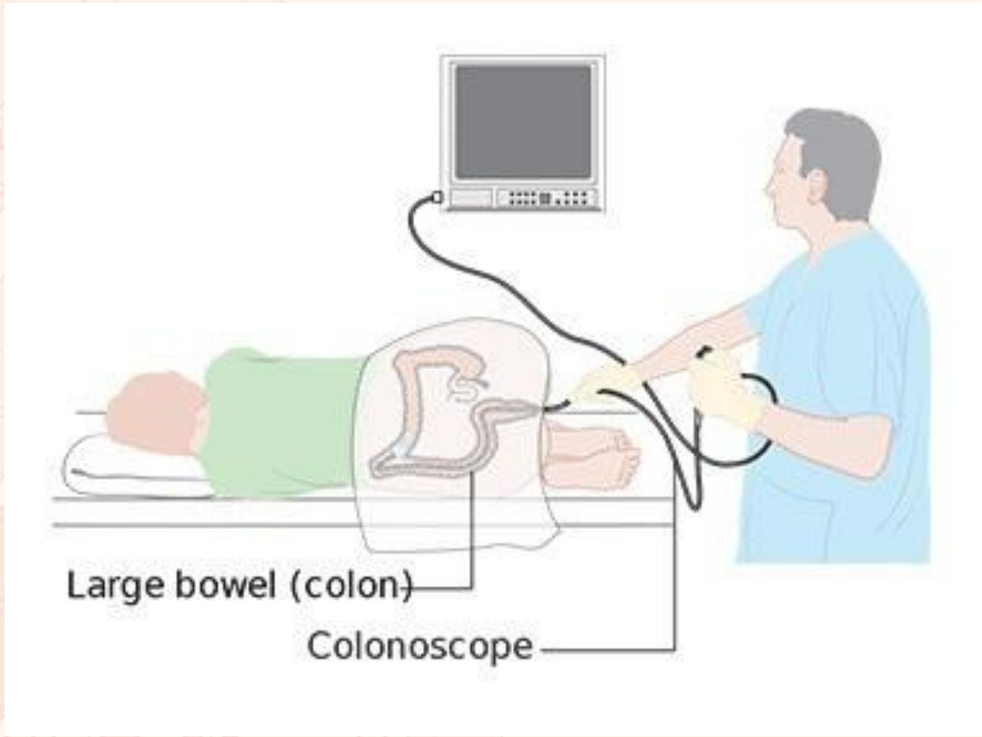
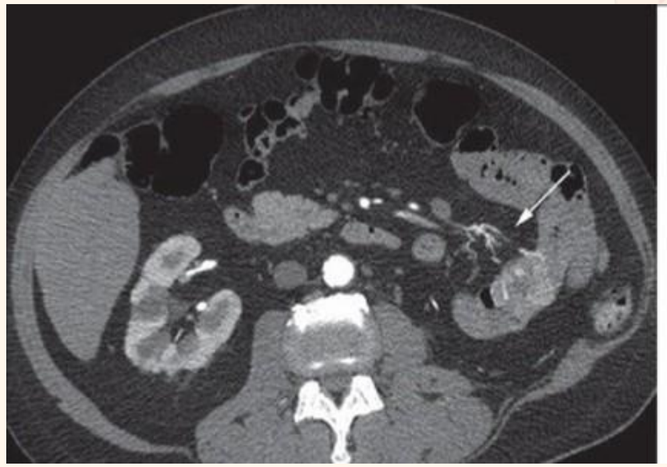
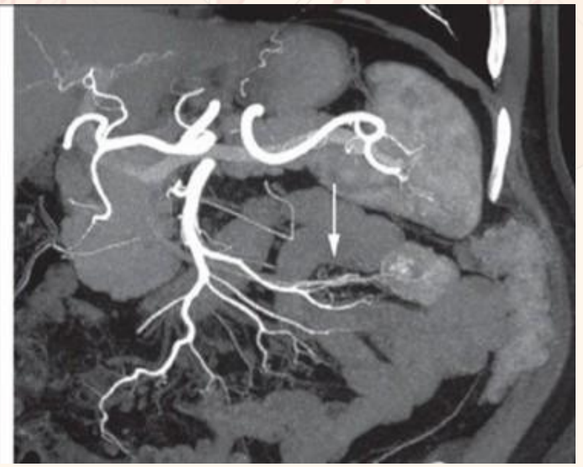

Upper Endoscopy
Duodenum
Endoscope
Esophagus
Stomach


Large bowel (colon)
Colonoscope





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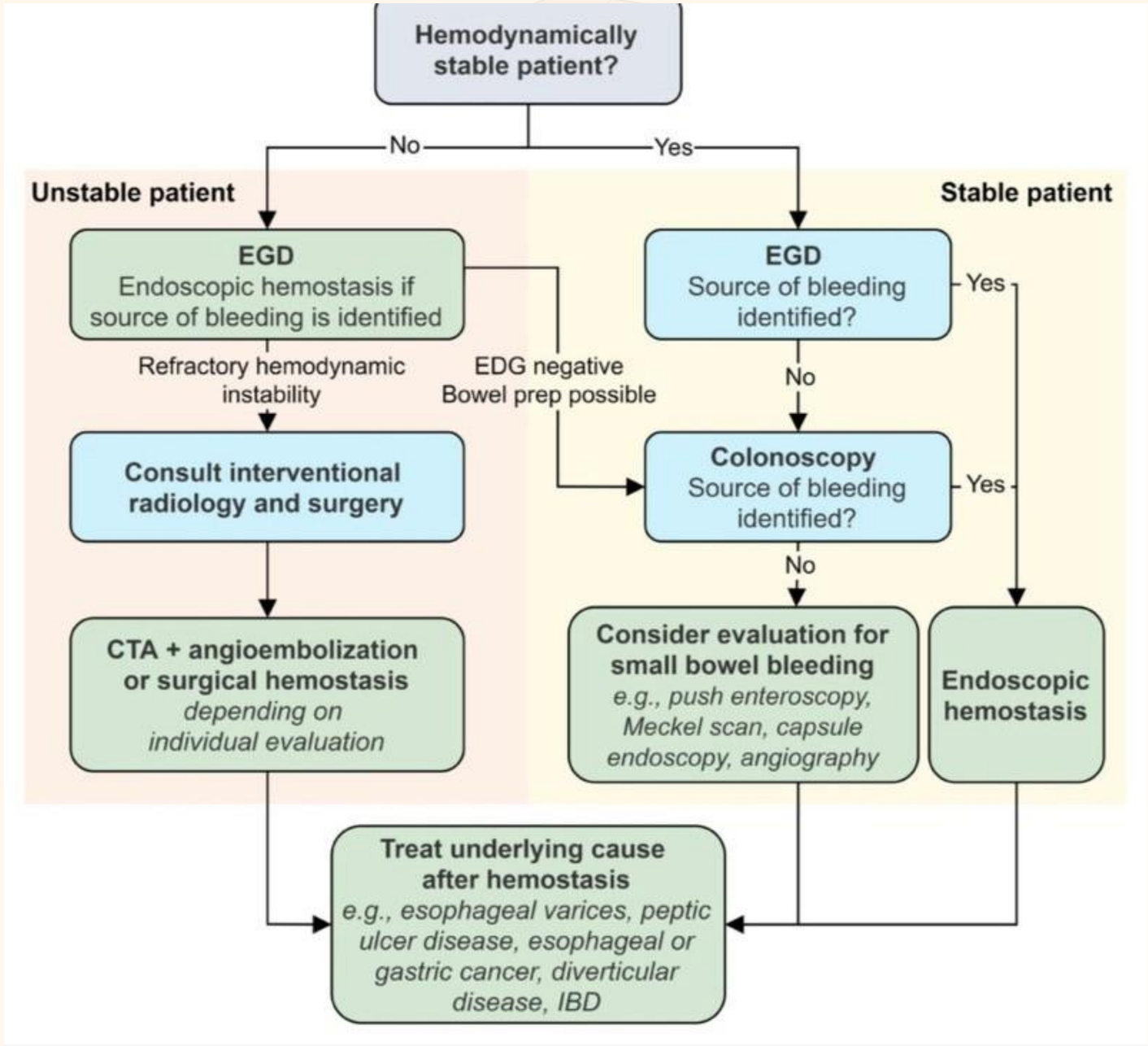

Hemodynamically stable patient?
No
Yes
Unstable patient
Stable patient
EGD
Endoscopic hemostasis if source of bleeding is identified
Refractory hemodynamic instability
EDG negative
Bowel prep possible
Consult interventional radiology and surgery
CTA + angioembolization or surgical hemostasis
depending on individual evaluation
EGD
Source of bleeding identified?
Yes
No
Colonoscopy
Source of bleeding identified?
Yes
No
Consider evaluation for small bowel bleeding
e.g., push enteroscopy, Meckel scan, capsule endoscopy, angiography
Endoscopic hemostasis
Treat underlying cause after hemostasis
e.g., esophageal varices, peptic ulcer disease, esophageal or gastric cancer, diverticular disease, IBD

ROCKALL RISK SCORING SYSTEM:

	Score			
	0	1	2	3
Age	<60	60-79	>80	
Shock	Pulse < 100 BP > 100	Pulse > 100 BP < 100	Pulse > 100 BP < 100	
Commorbidities	none		Circulatory failre/ coronary artery disease	Renal failure Liver failure Disseminated malignancy
Endoscopic sings of bleeding	None/ dark spot		Blood/ adherent clot/ visible or spurting vessel	
Diagnosis	Mallory-Weiss syndrome/ no patology	All other diagnoses	Malignancy of the upper GI tract	

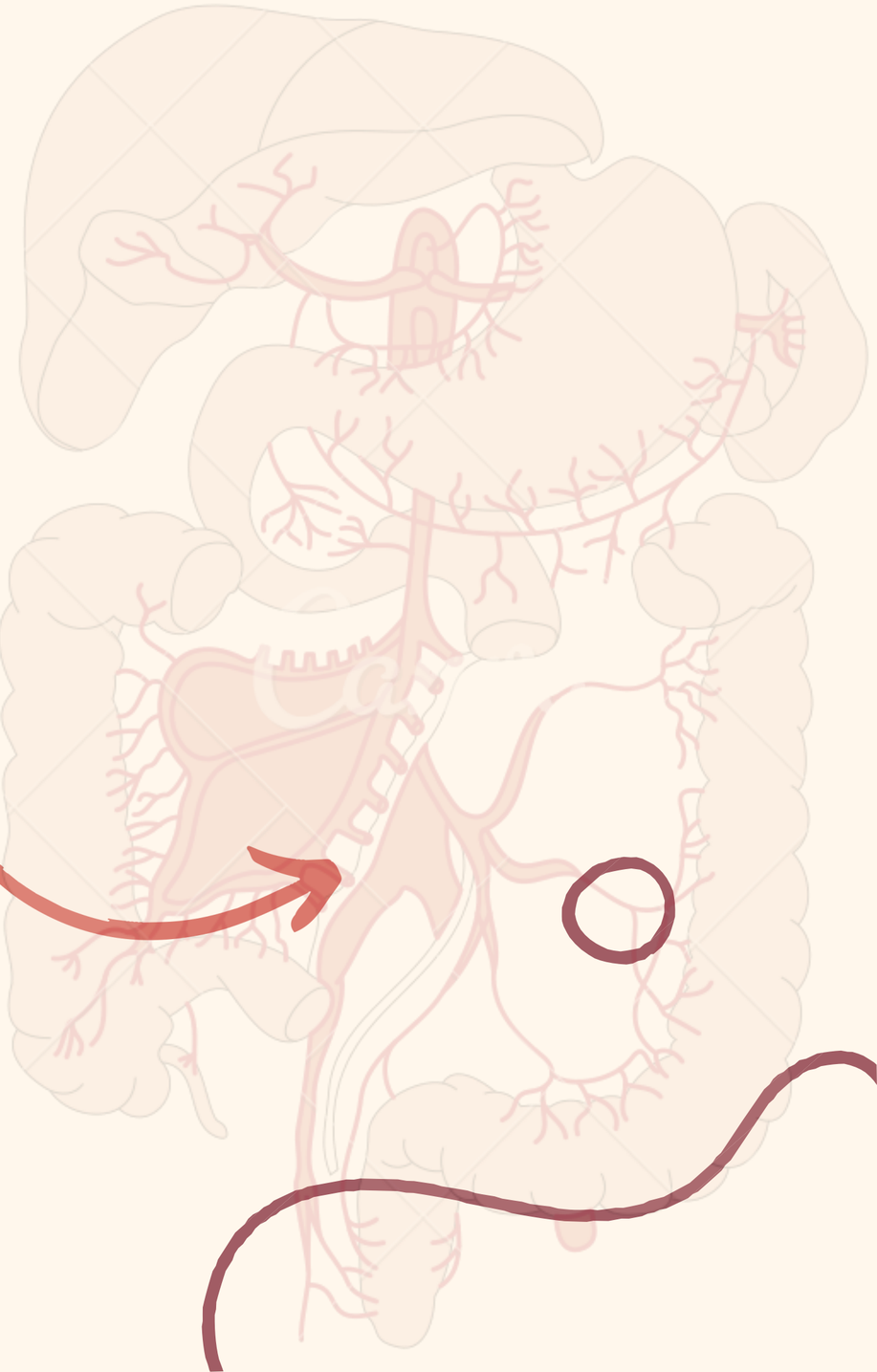
- Assessing the risk of death and re-bleeding in patients with UGI haemorrhage
- A score less than 3 carries good prognosis but total score more than 8 carries high risk of mortality

CASE :

- A30-year-old male named Joseph came to the emergency department because of sharp chest pain radiating to his back. He recently graduated from medical school and has been celebrating for the past week at local bars. He says that he was drinking to the point of vomiting and blacking out. He thinks his pain began after a particularly intense night of vomiting and retching. His vital signs show no abnormalities

UGITB VS LGITB :

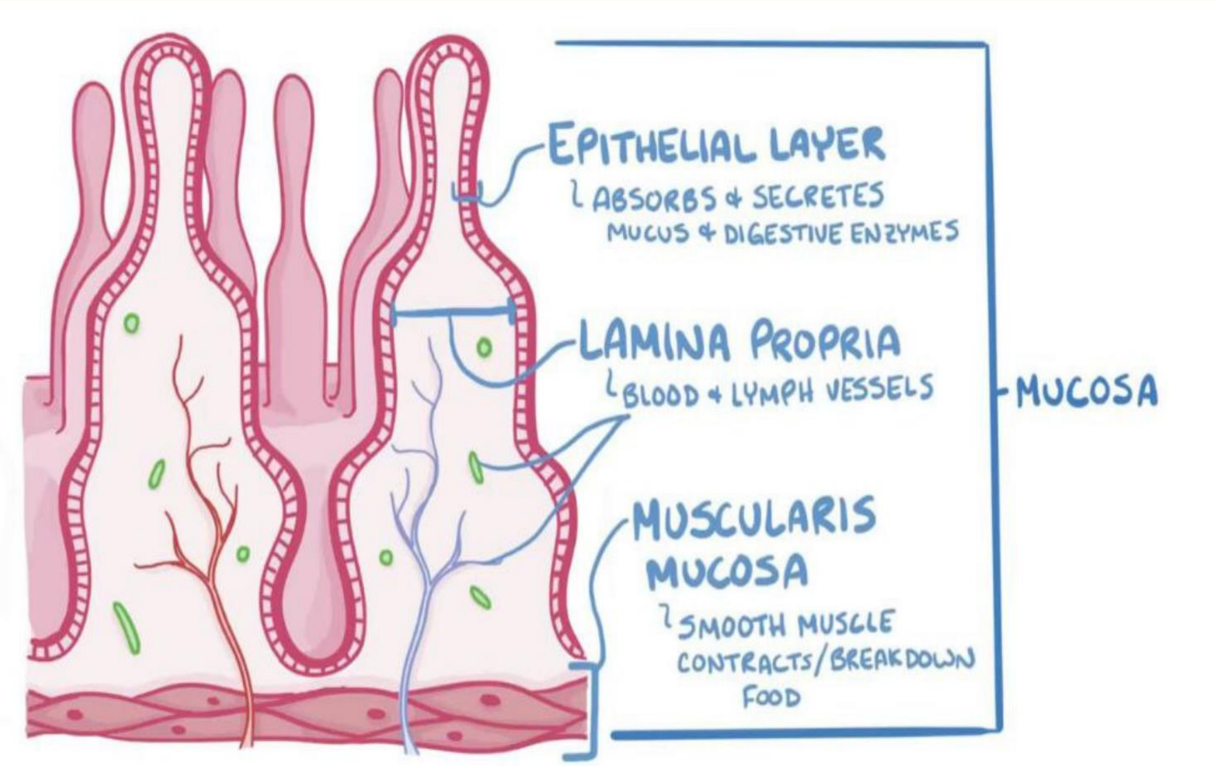
UGITB	LGITB
Proximal to ligament of Treitz	Distal to ligament of Treitz
Hemetemesis or malena blood on NA	Hematochezia clear fluid on NA
Hyperactive bowel sound increase BUN/ CREATININE R	Normal bowel sounds and normal ratio



Peptic → stomach

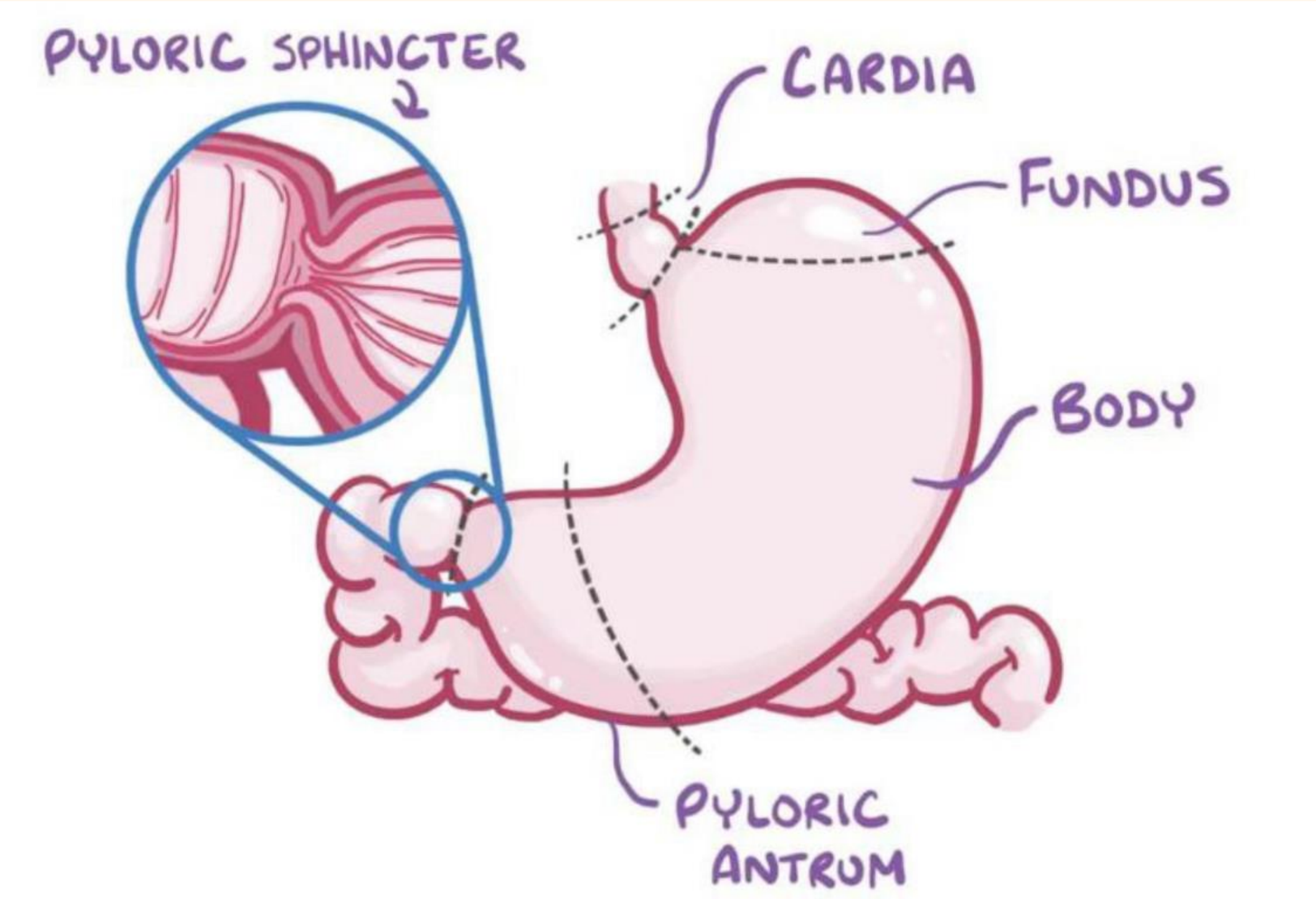
Ulcer → loss of continuity

Peptic ulcer disease PUD : loss continuation of the mucosa of the gastrointestinal tract .



Site of ulcers

1. stomach (gastric ulcer)
2. proximal duodenum &
   distal duodenum (duodenal ulcer)
3. Jejunum
4. lower esophagus

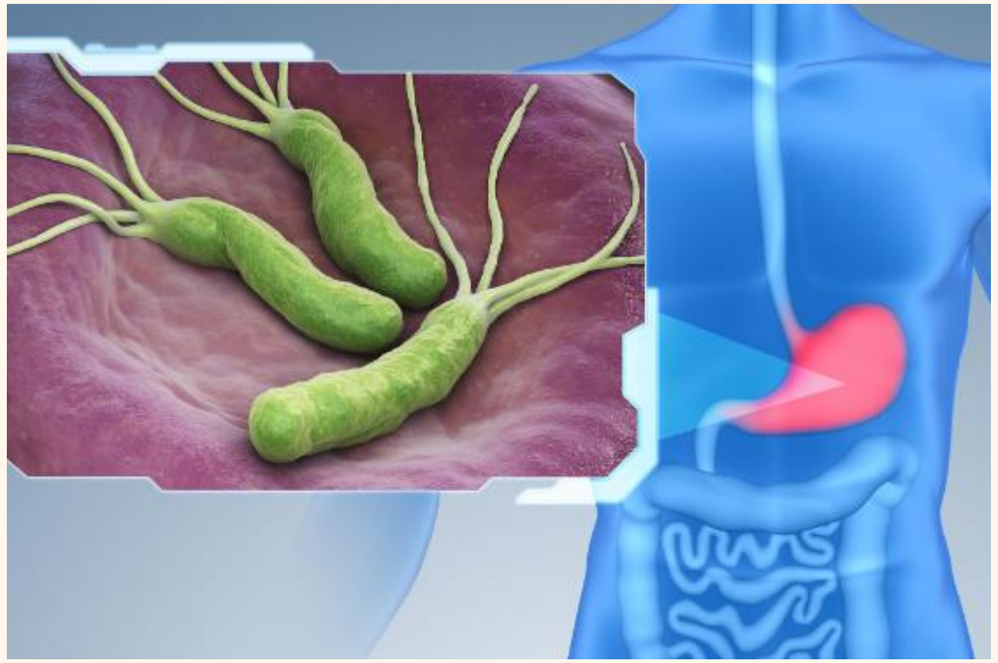


1. Gastric ulcer (mostly in the antrum, most susceptible part to bleed is the lesser curvature)

2. Duodenal ulcer (Duodenal bulb, the posterior wall is the most common site for bleeding)

NOTE: Bleeding from duodenal ulcers is four times more common than from gastric ulcers

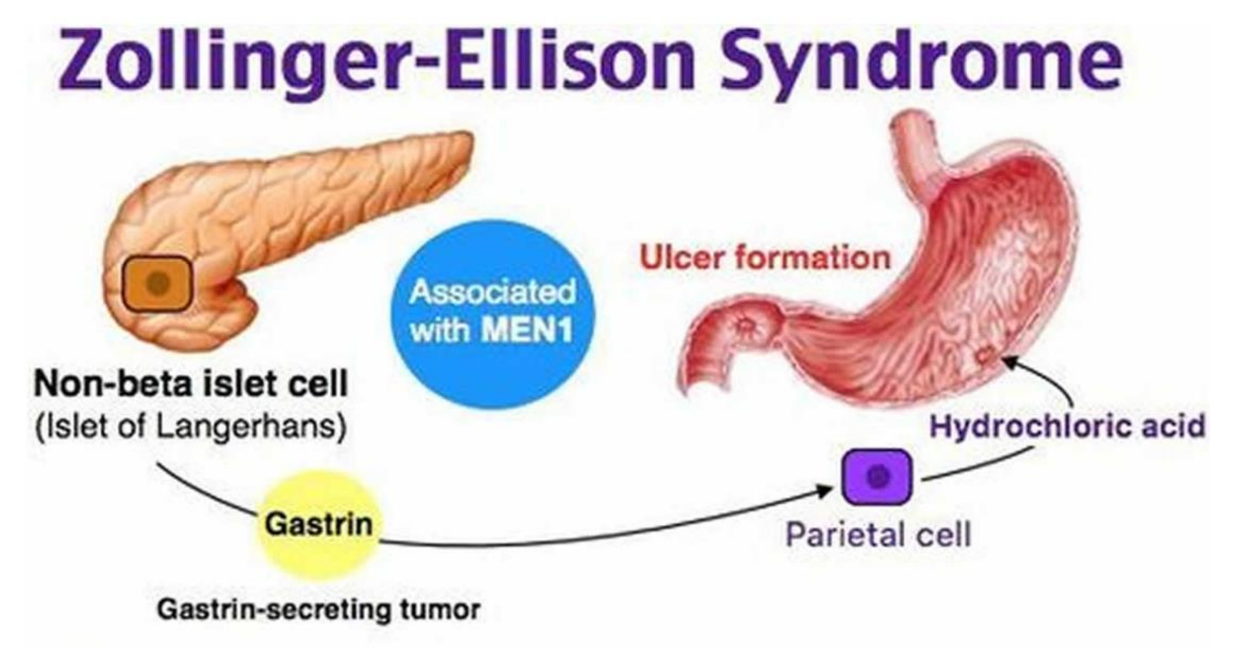
Causes of ulcers



H. Pylori : gram negative bacteria colonize the mucosa of stomach (antrum) and duodenum ,has a protease enzyme

NSAIDS ; due to inhibition of cyclooxygenase → decrease prostaglandin formation

gastrinoma (Zollinger Ellison syndrome)



What is the differential diagnosis?

1.pancreatitis

2.cholecystitis

3.ZES

4.Gastritis

5.MI

6.reflux

Complication of PUD ;

Bleeding ****

Perforation (2ry peritonitis)

Ulcer penetration and fistula formation

Air under diaphragm :(lead to phrenic nerve irritation

→ pain up to the shoulder)

Definition: Penetration of a peptic ulcer through the gastric/duodenal wall into adjacent organs (e.g., pancreas, biliary tree, colon) without leaking of gastric contents into the peritoneal cavity

Etiology: Duodenal ulcers are the most common cause of penetration.

Clinical features: a change in clinical symptoms that are related to the affected neighboring organs

Colon: Gastrocolic or duodenocolic fistulas may manifest with copremesis and postprandial diarrhea.

Liver, spleen, or diaphragm: Penetration may result in visceral abscesses (fever, abdominal tenderness, and sepsis).

Gastroduodenal artery or aorta: Vascular fistulas may result in severe hemorrhage.

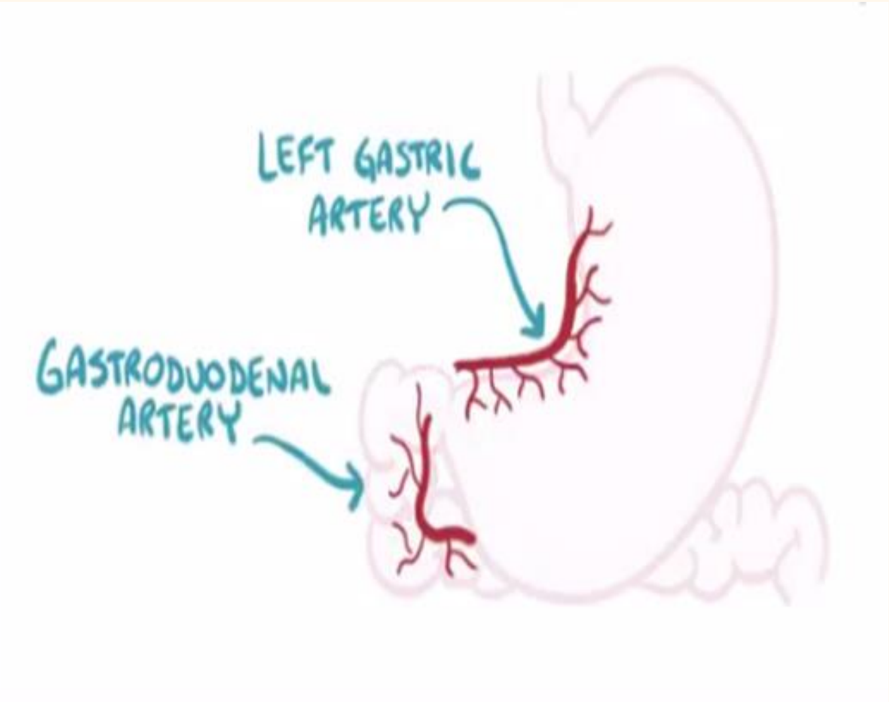
Biliary tree: Choledochoduodenal fistulas may manifest with biliary tract obstruction (fever, jaundice, RUQ pain).

Pancreas: increased epigastric pain and peritonitis

Source of bleeding in Peptic ulcers ?

These ulcers appear like holes of mucosa whit clean base , in this base you can find scar tissue .

If the erosion goes deeply to the vessels (spatially left gastric artery and gastroduodenal artery) will lead to rapid blood loss and then hypovolemic shock .



These ulcers appear like holes of mucosa whit clean base , in this base you can find scar tissue .

If the erosion goes deeply to the vessels (spatially left gastric artery and gastroduodenal artery) will lead to rapid blood loss and then hypovolemic shock

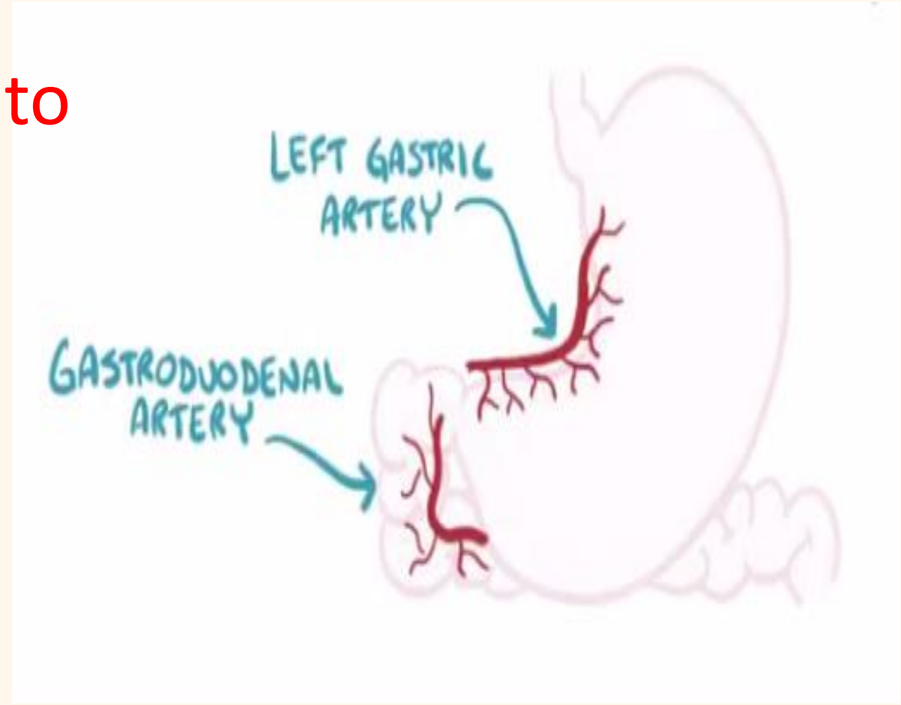
rate of bleeding :

- slowly and chronically (symptoms will be similar to that of anemia):

1.Pallor of skin and mucous membranes
2.Shortness of breath and lack of energy
3.Fatigue
4.Palpitations

- heavily and acutly cause :

1.Melena
2.Hematemesis
3.Hematochezia
3.Hypovolemic shock



Hypovolemic shock lead to ;

Tachycardia
Hypotension
Little or no urine output
Loss of consciousness or confusion

Other signs

Burning stomach pain & Heartburn.
local signs (epigastric tenderness).
Intolerance to fatty foods.
Nausea.

Note :

Gastric ulcer pain increase whit eating and relive by fasting (weight loss) .

Duodenal ulcer pain decrease whit eating and exacerbated by fasting (weight gain) .

General Management (intervention) :

Rapid resuscitation

mean arterial pressure 65 mm Hg or higher
urine output of 0.5 mL/kg/h or greater
lactate normalization

Laboratory tests for H. pylori

Endoscopy

preferred diagnostic and therapeutic tool in suspected bleeding peptic ulcers due to low complications and the high accuracy in decreasing the risk of rebleeding
performed as soon as possible (up to 24 hours after presentation is considered early endoscopy).

Tretmnt of PUD bleeding:

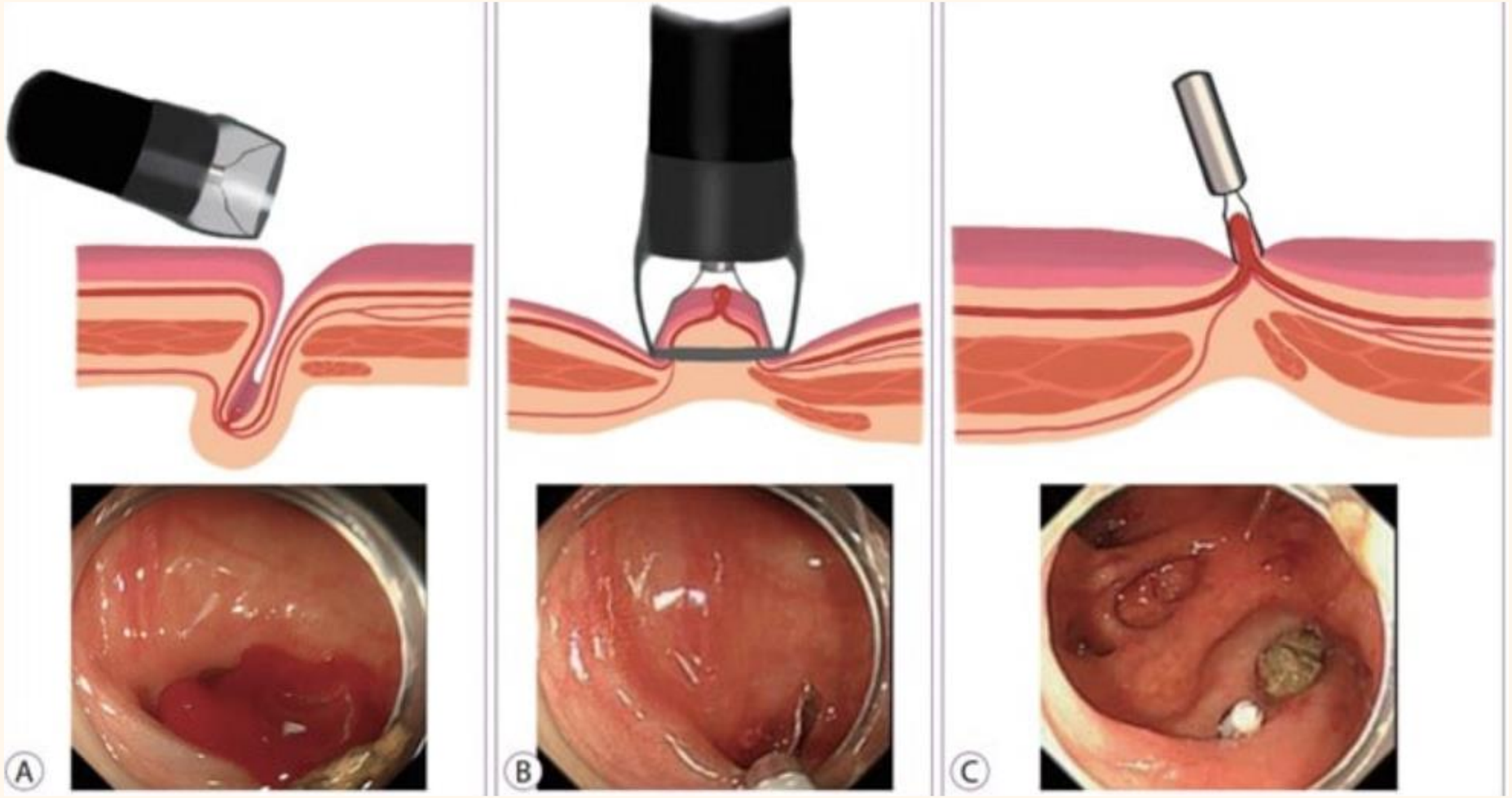
1. Endoscopic hemostasis

Endoscopic hemostasis is the first-line treatment for most patients with PUD bleeding. This minimally invasive procedure is performed using an endoscope, which is a thin, flexible tube with a camera and light at the end. The endoscope is inserted through the mouth or rectum and guided to the bleeding site. Once the bleeding site is identified, a variety of techniques can be used to stop the bleeding, such as injection therapy, thermal therapy, or mechanical therapy.

Injection therapy: This involves injecting a substance, such as epinephrine into the bleeding vessel to seal it off.

Thermal therapy: This involves using heat to seal off the bleeding vessel. This can be done using a laser

Mechanical therapy: This involves using clips, bands, or sutures to close off the bleeding vessel.


A
B
C

2. Proton pump inhibitors (PPIs);

PPIs are medications that reduce the production of stomach acid. (Omeprazole & lansoprazole)

3.H. pylori eradication ;

H. pylori is a bacteria that can cause PUDs. If H. pylori is present, it is important to eradicate it with antibiotics

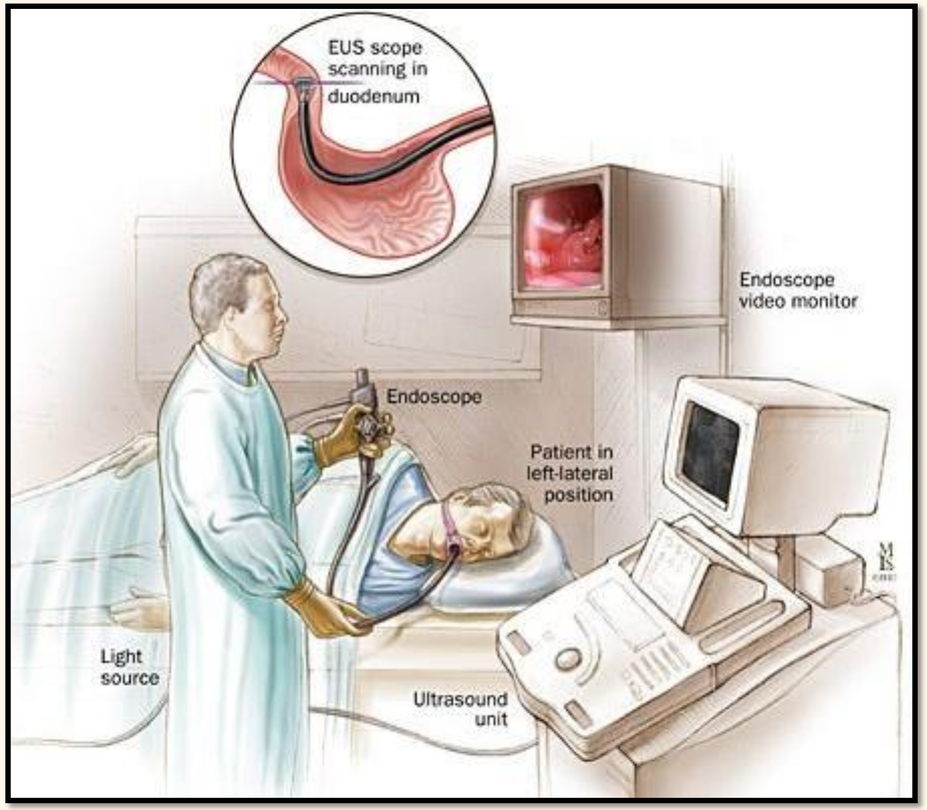
4.Surgery;

may be necessary if the bleeding cannot be controlled endoscopically or if there is a perforation of the ulcer.

5.Angiographic embolization;

is a procedure that involves injecting a material into the blood vessel that is supplying blood to the ulcer. This can help to stop the bleeding.

If there is clinical evidence of ulcer rebleeding, the guideline is to repeat endoscopy with an attempt at endoscopic hemostasis. This is because endoscopic hemostasis is the most effective way to stop bleeding from ulcers.



Surgical procedures

Vagotomy:

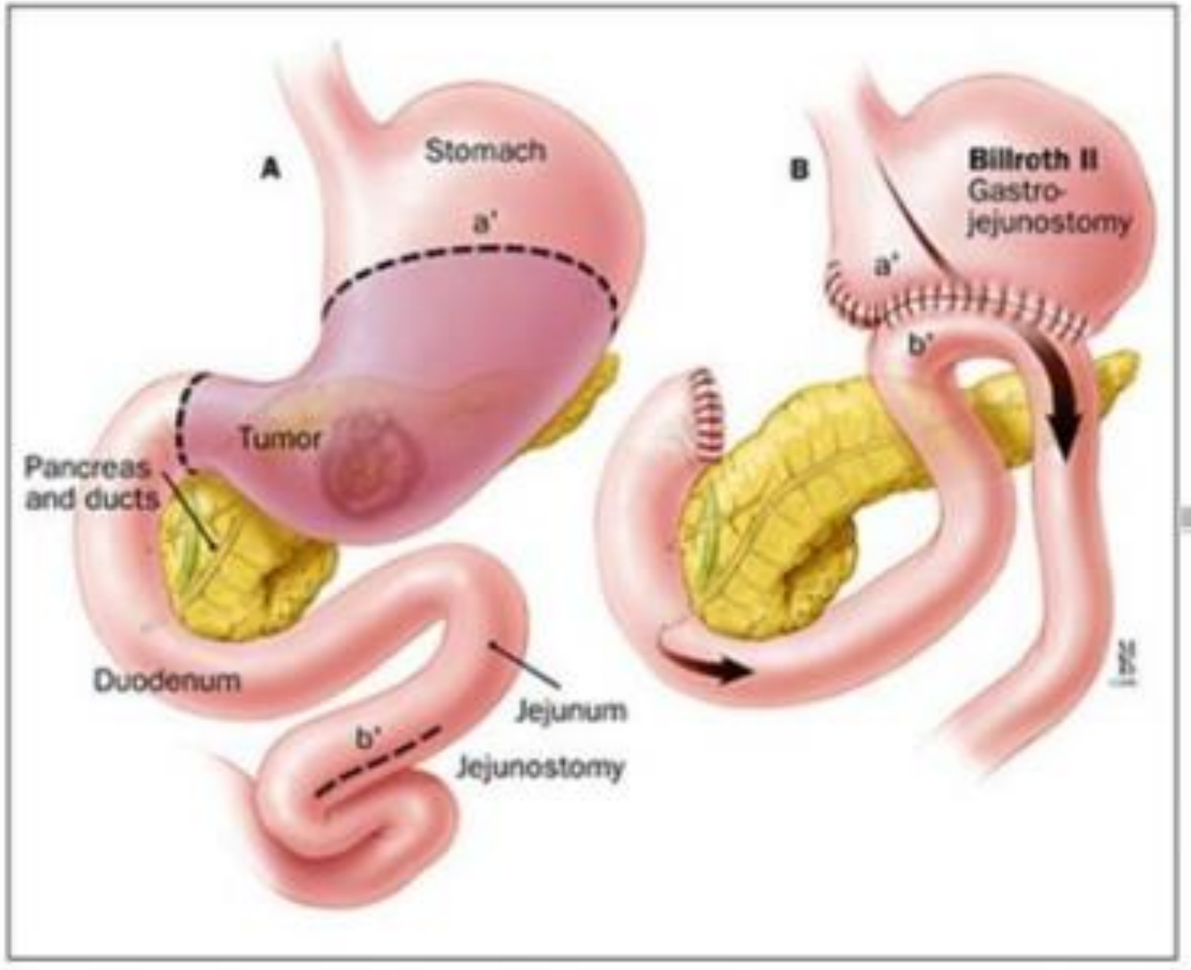
This procedure involves cutting the vagus nerve, which is the nerve that controls the release of stomach acid. A vagotomy can help to reduce the amount of stomach acid that is produced, which can help to prevent ulcers from forming and bleeding.

Pyloroplasty:

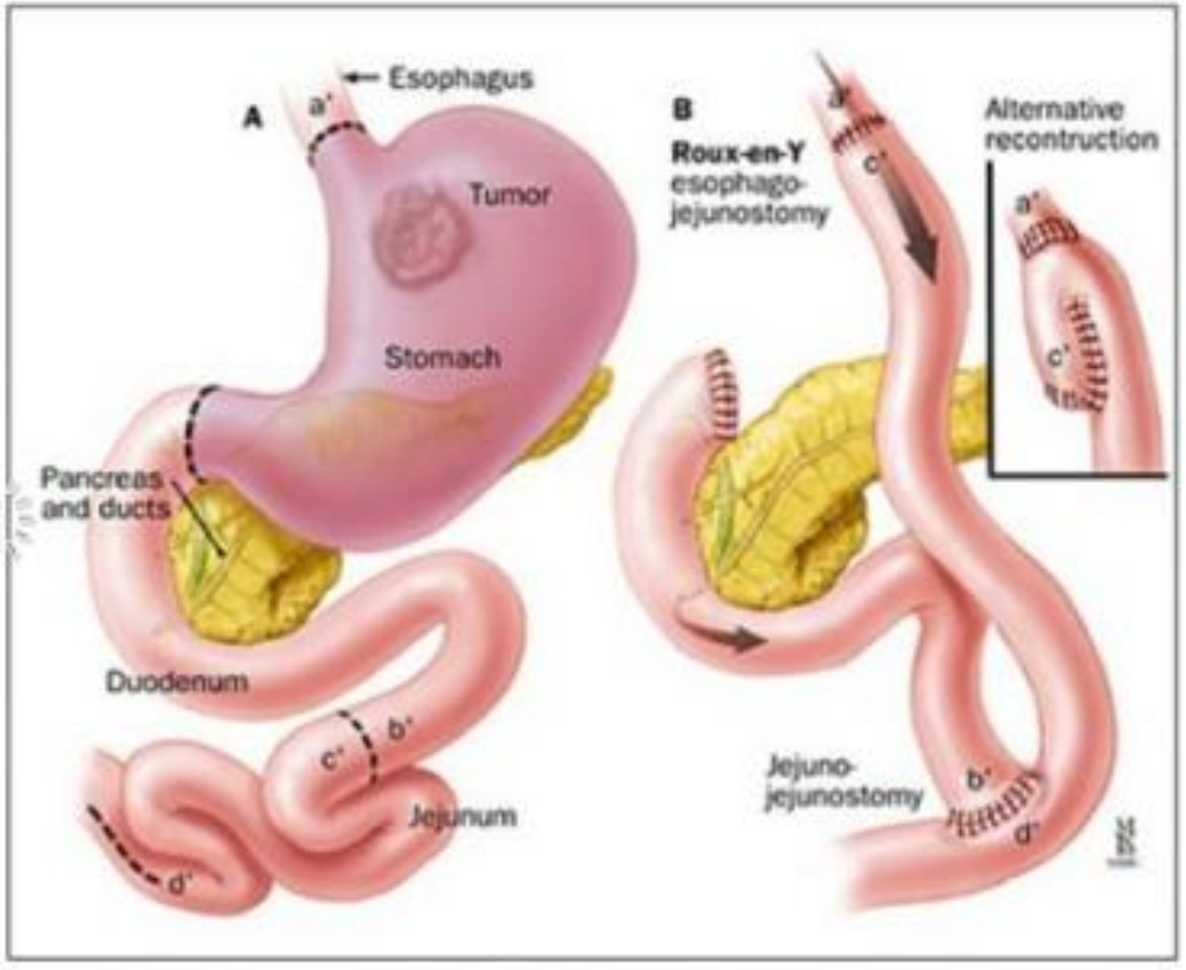
This procedure involves widening the pylorus, which is the opening between the stomach and the small intestine. A pyloroplasty can help to improve drainage from the stomach, which can help to reduce the risk of ulcers forming and bleeding.

Gastrectomy:

This procedure involves removing part or all of the stomach. A gastrectomy is usually only performed as a last resort if other treatments have been unsuccessful.



Partial gastrectomy



Total gastrectomy

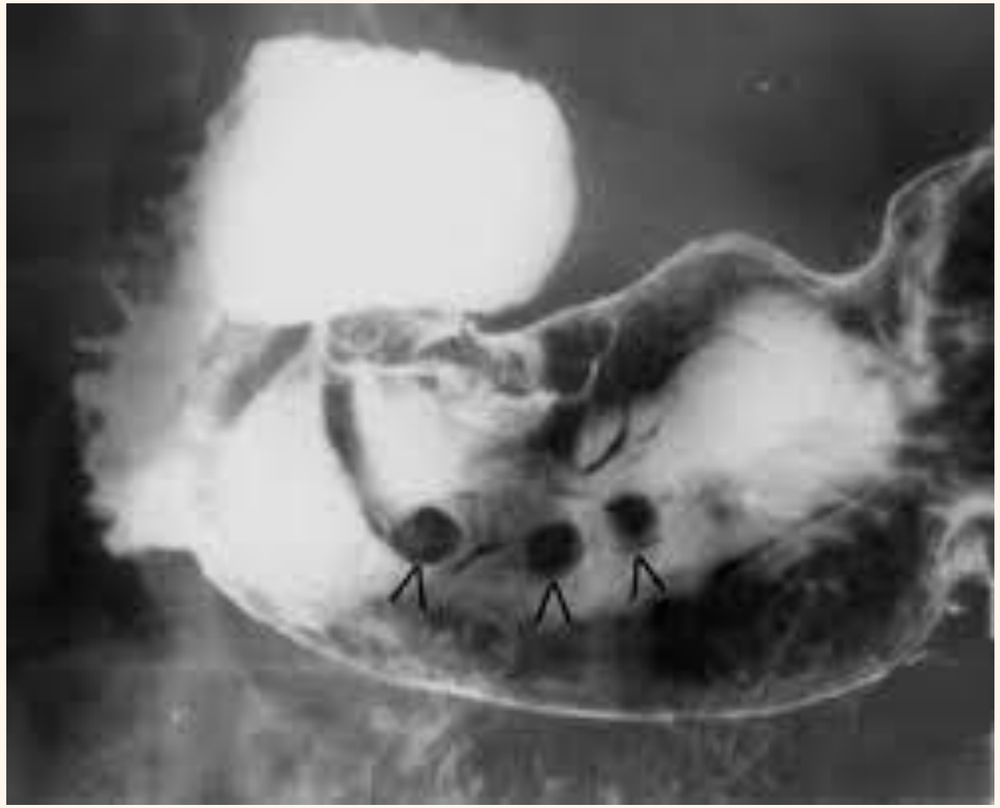
Erosive gastritis

Acute mucosal inflammatory process caused by agents that disturb the gastric mucosal barrier ;NSAIDs and alcohol are common causes.

Clinical presentation

Patients with mild erosive gastritis are often asymptomatic, although some complain of dyspepsia, nausea, or vomiting. Often, the first sign is hematemesis, melena, or blood in the nasogastric aspirate, usually. Bleeding is usually mild to moderate, although it can be massive if deep ulceration is present, particularly in acute stress gastritis.

Diagnoses



An upper GI series, also known as a barium swallow, is a type of X-ray that can be used to diagnose erosive gastritis. During the procedure, you will drink a liquid containing barium, that coats the lining of your upper GI tract. The barium makes the lining of your stomach and other organs more visible on the X-rays.

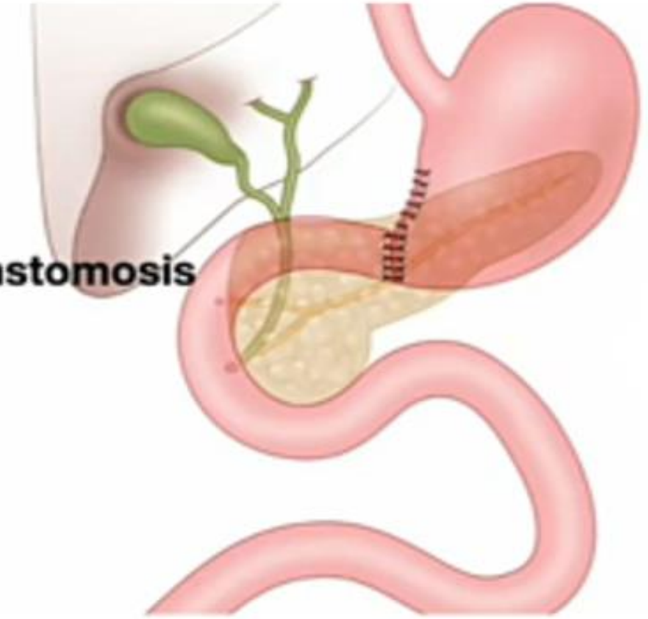
management

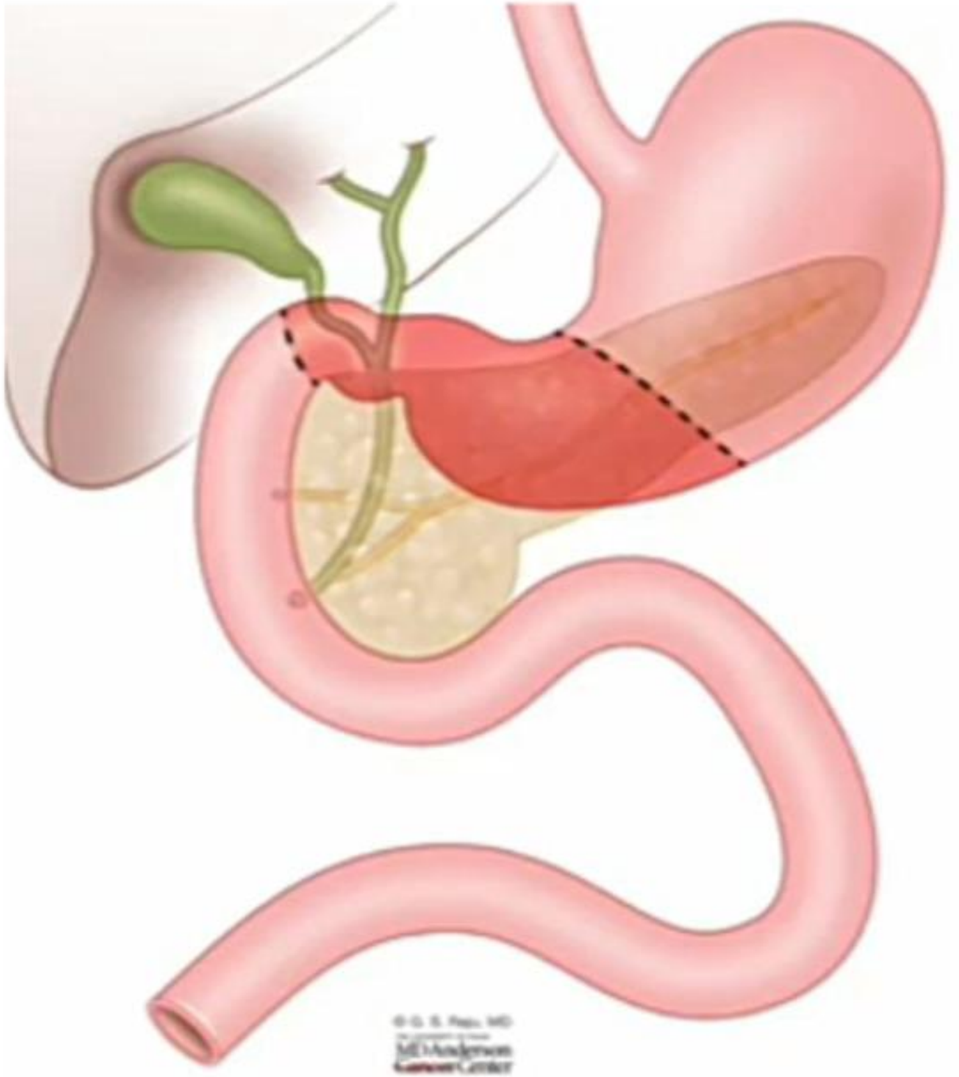
diagnosed by history of medications or any erosive agent and by endoscope
Interventional treatment: Endoscopic hemostasis For bleeding
Medical treatment : A proton pump inhibitor or H2 blocker For acid suppression ,PGE2 analog may be given to promote healing sufficient for milder gastritis,
In severe gastritis, bleeding is managed with IV fluids and blood transfusion as needed. Endoscopic hemostasis should be attempted, with surgery a fallback procedure if bleeding cannot be controlled endoscopically. Acid-suppressing drugs should be started if the patient is not already receiving it.

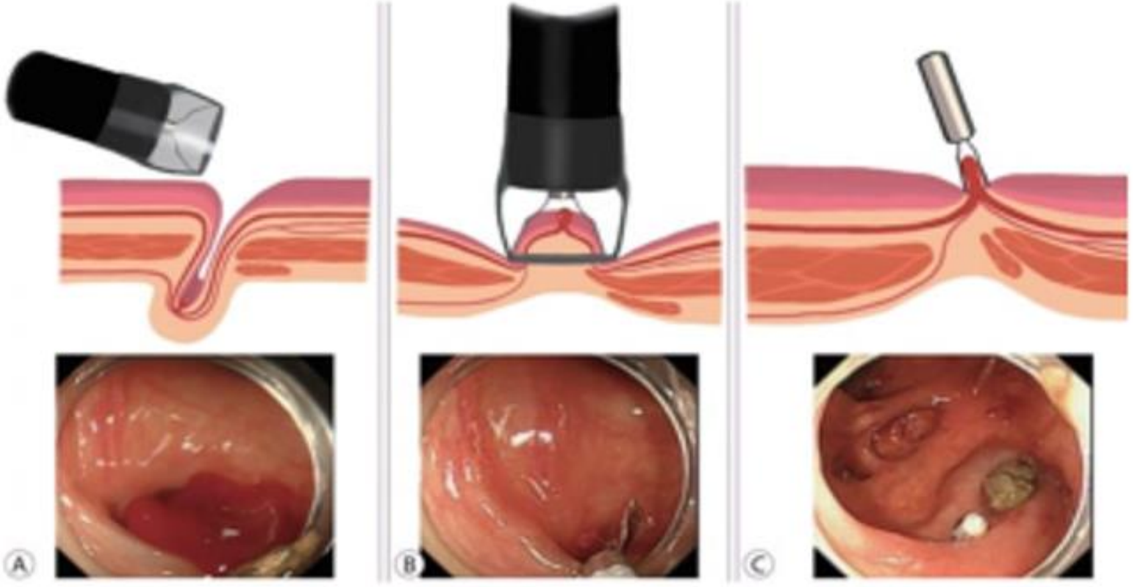
Surgery

Only indicated if bleeding cant be controlled by endoscope
Billroth 1 anastamosis is the procedure

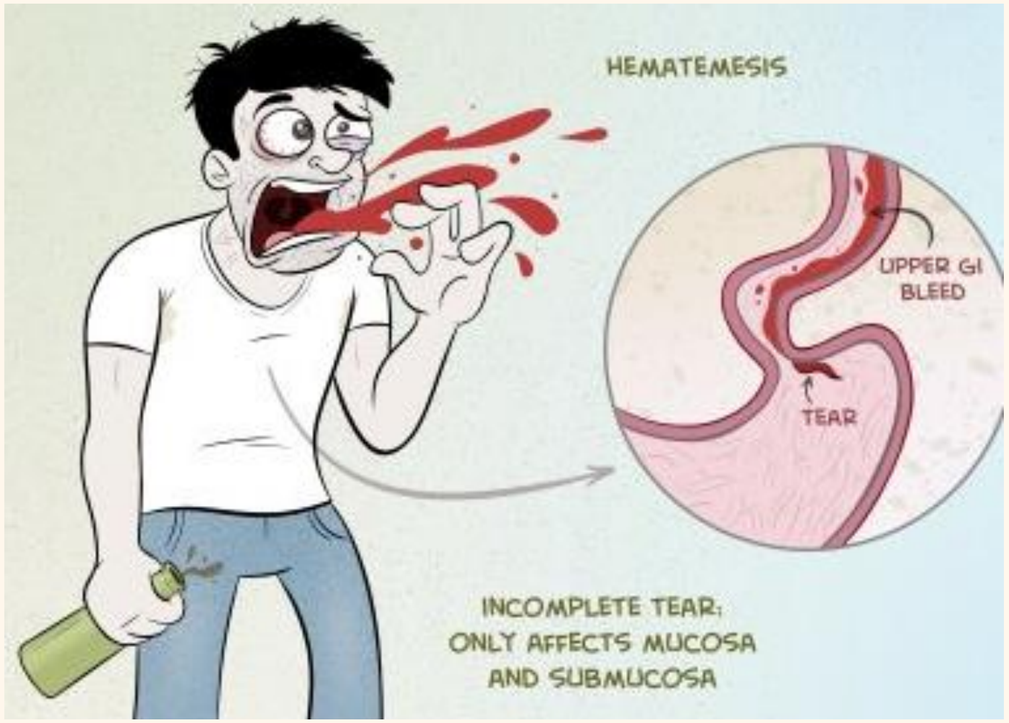
Gastroduodenal anastomosis
Completed

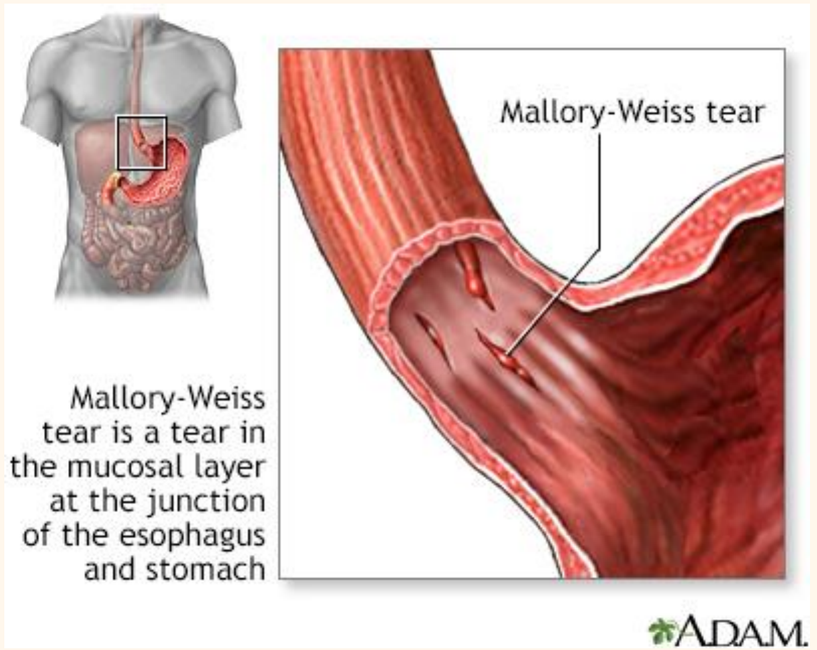




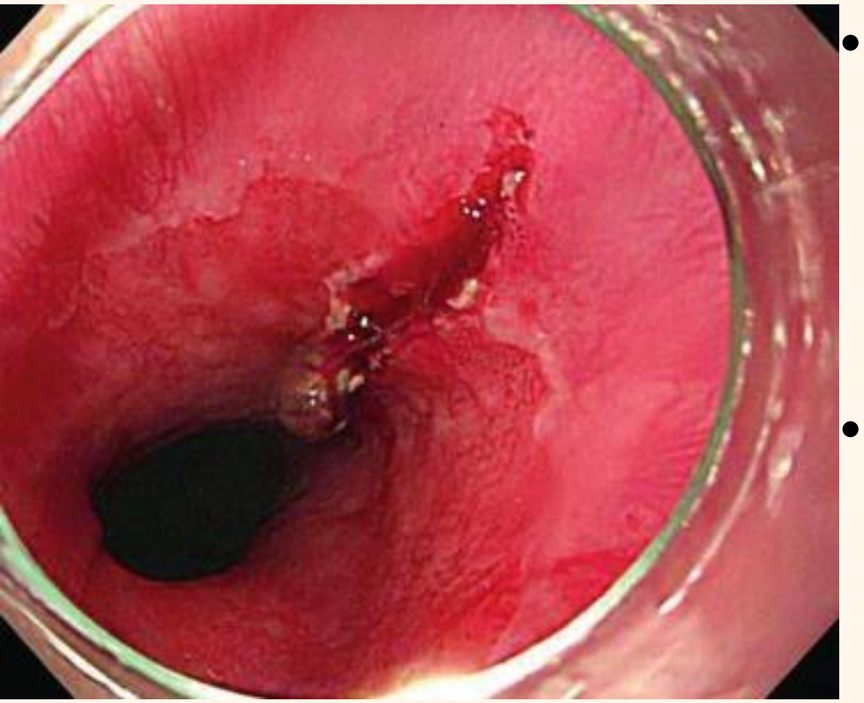


Mallory-Weiss tears





Definition



- Mucosal tear or lacerations (vertical splits) in the inner layer of esophagus with only 10% of cases, while 90% of cases at the gastroesophageal junction with <1 inch or 2.5cm length and occur in males more than females.
- It leads to hematemesis (vomiting blood); which is usually not sever but require endoscopy, heal within 7 to 10 days without treatment. Patients with sever bleeding require immediate hemodynamic support.

Etiology

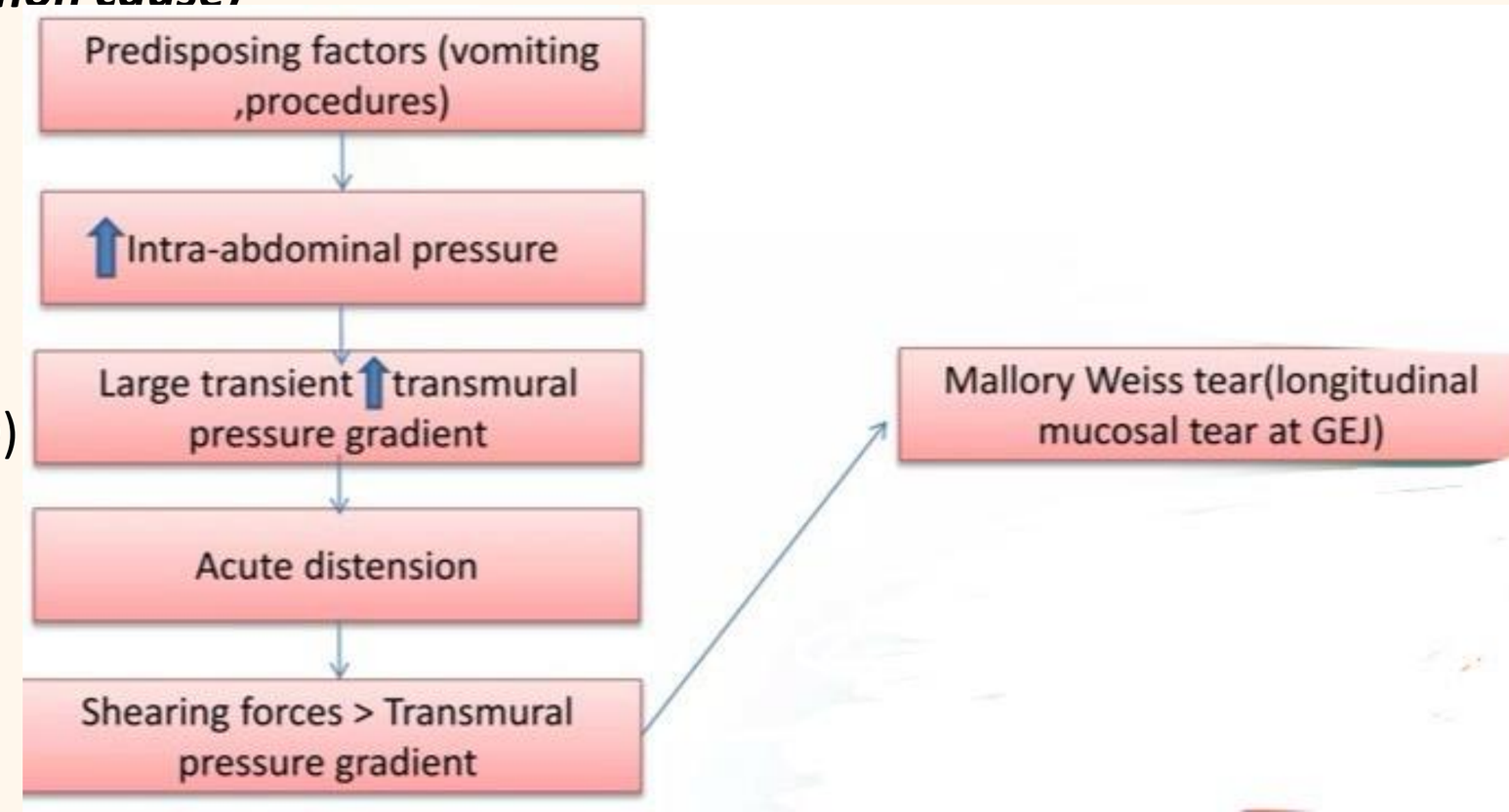
Mechanism: a sudden and sever rise in the esophageal pressure.

Precipitating factors:

1. ***Sever vomiting or retching (most common cause)***
2. Blunt abdominal trauma
3. Strained defecation
4. Prolonged coughing

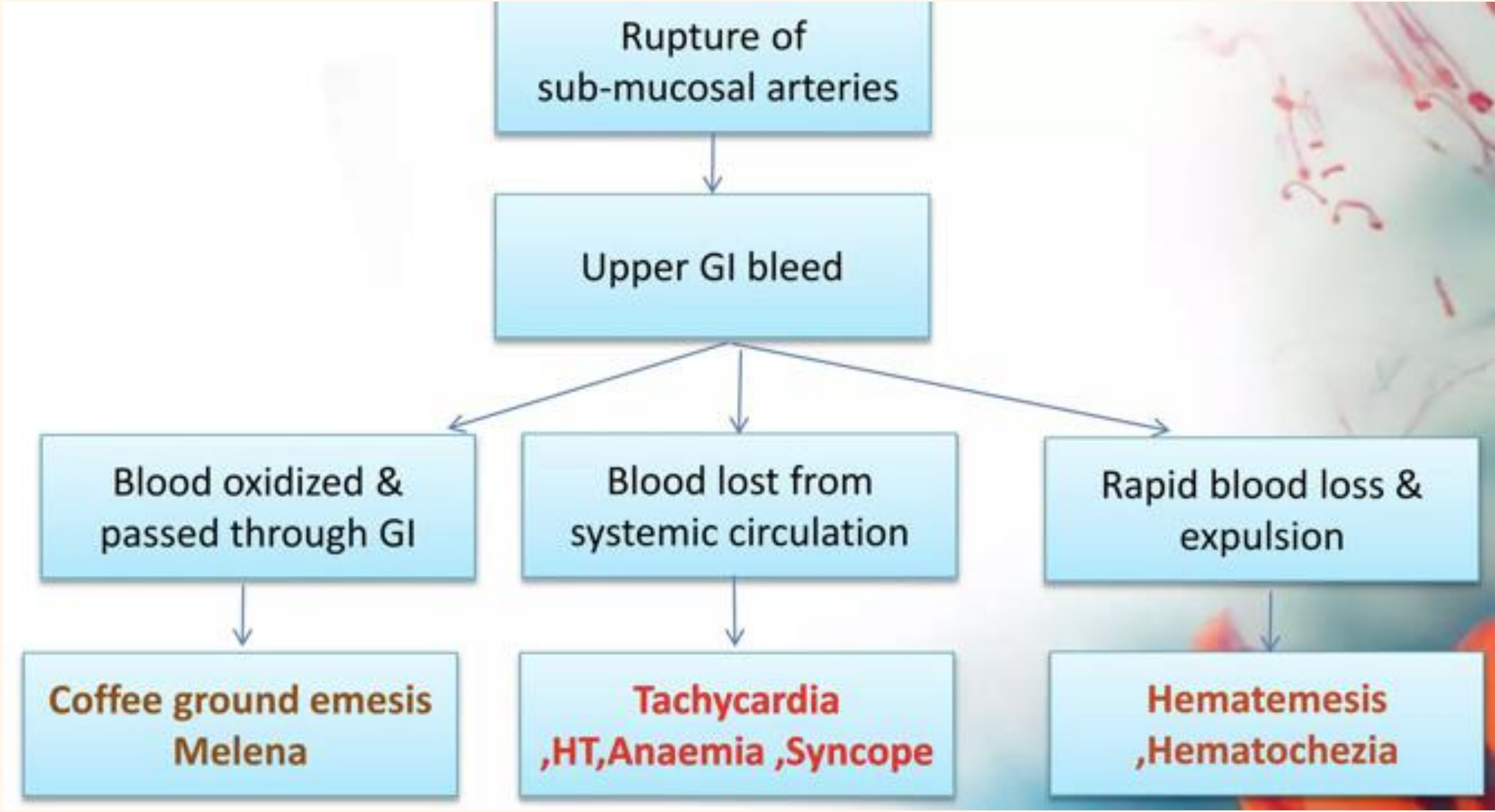
Predisposing factors:

1. ***Alcohol use***
2. Gastroesophageal reflux disease (GERD)



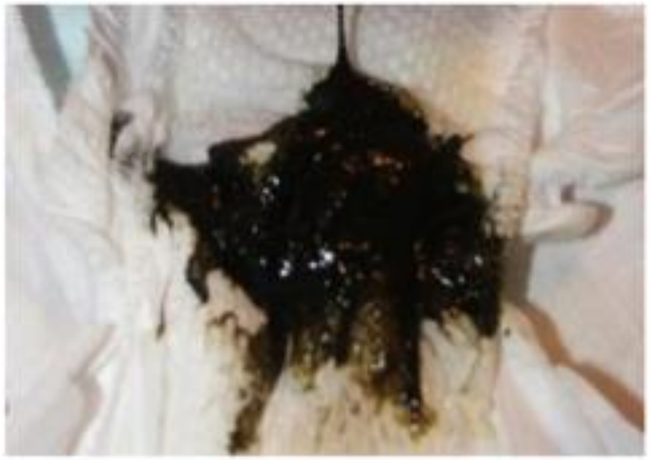
Clinical features

- May be asymptomatic
- Symptomatic patients present with:
  - Hematomesis (which occur in 85% of cases), the blood is bright red, clotted and has the appearance of “coffee grounds”.
  - Epigastric pain.
  - Sticky dark as tar stool (melenic).
  - Weakness, dizziness, fainting.


Rupture of sub-mucosal arteries
Upper GI bleed
Blood oxidized & passed through GI
Blood lost from systemic circulation
Rapid blood loss & expulsion
Coffee ground emesis Melena
Tachycardia ,HT,Anaemia ,Syncope
Hematemesis ,Hematochezia

COFFEE GROUND EMESIS

"Coffee Ground Emesis"



Melena

complications

- If untreated, blood loss can cause:

Anemia
Fatigue
Shortness of breath
oesophageal shortening
Barret’s oesophagus
Stricture

- Possible shock with massive hemorrhage and internal bleeding.

diagnostics

- ❑Patient with upper GI bleeding and a history of precipitating or predisposing factors.
- ❑Initial studies:

CBC: may reveal anemia and/or thrombocytopenia
Coagulation studies: may reveal coagulopathy
Cardiac enzymesand bedside ECG: to rule out acute coronary syndrome
Pretransfusion testing
EGD
Mallory Azan stain

treatment

❖In most patient the bleeding stops and start healing on its own within 72 hours.

❖If it doesn’t stop bleeding on its own then start treatment by:

✓**Initial management of GI bleeding, potentially including:**

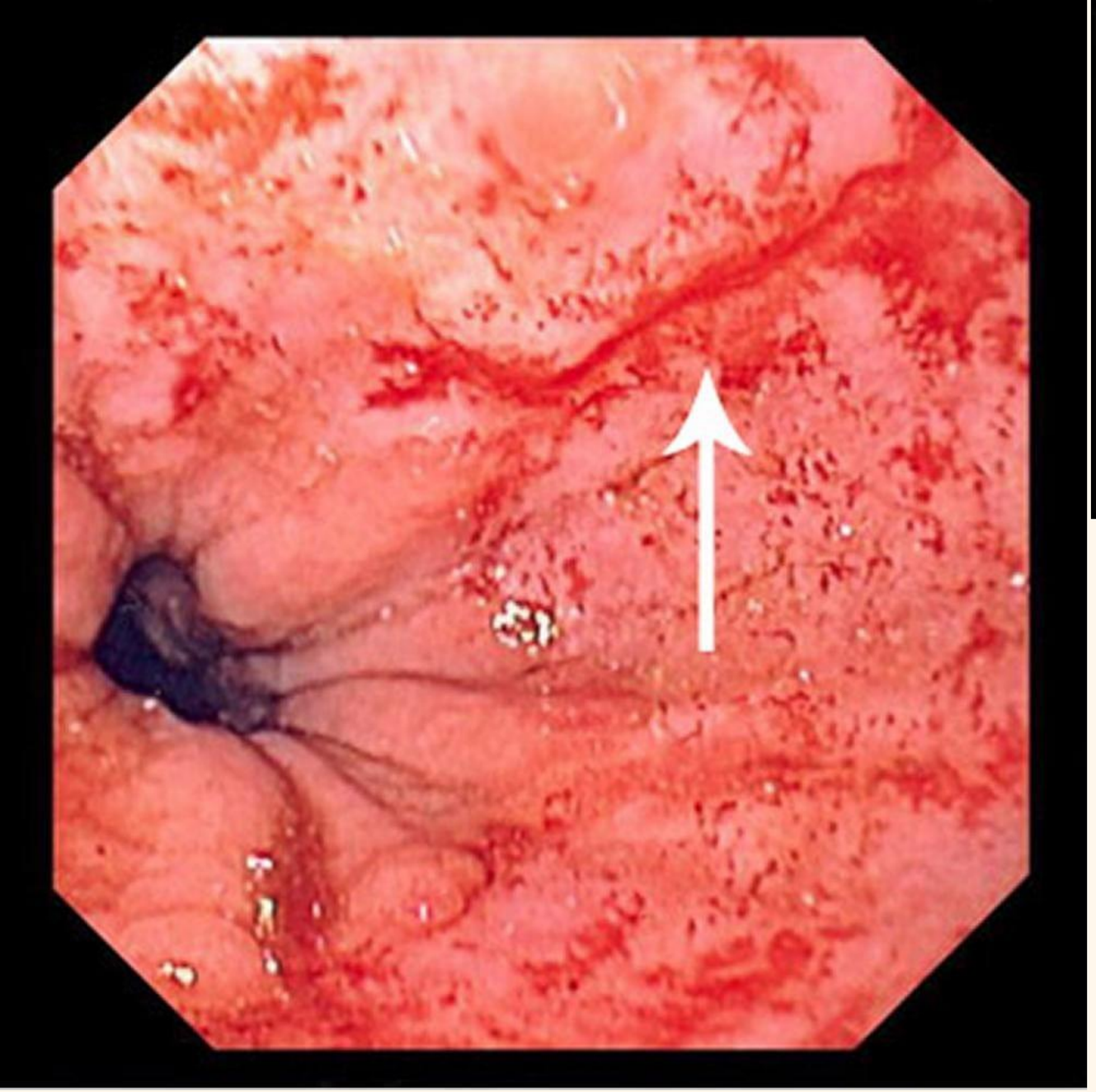
- Immediate hemodynamic support
- Emergency blood transfusion
- Epinephrine injection

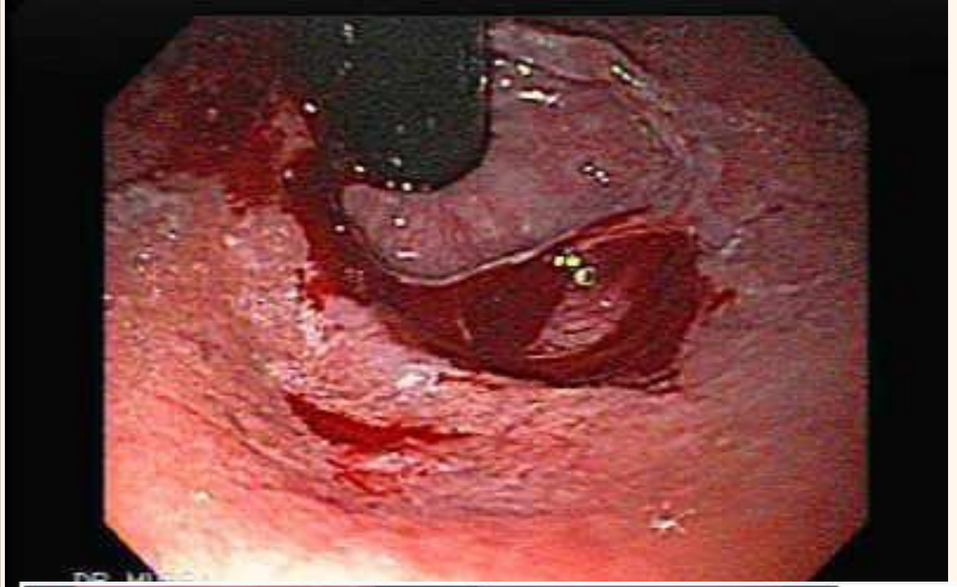
✓**Surgery:**

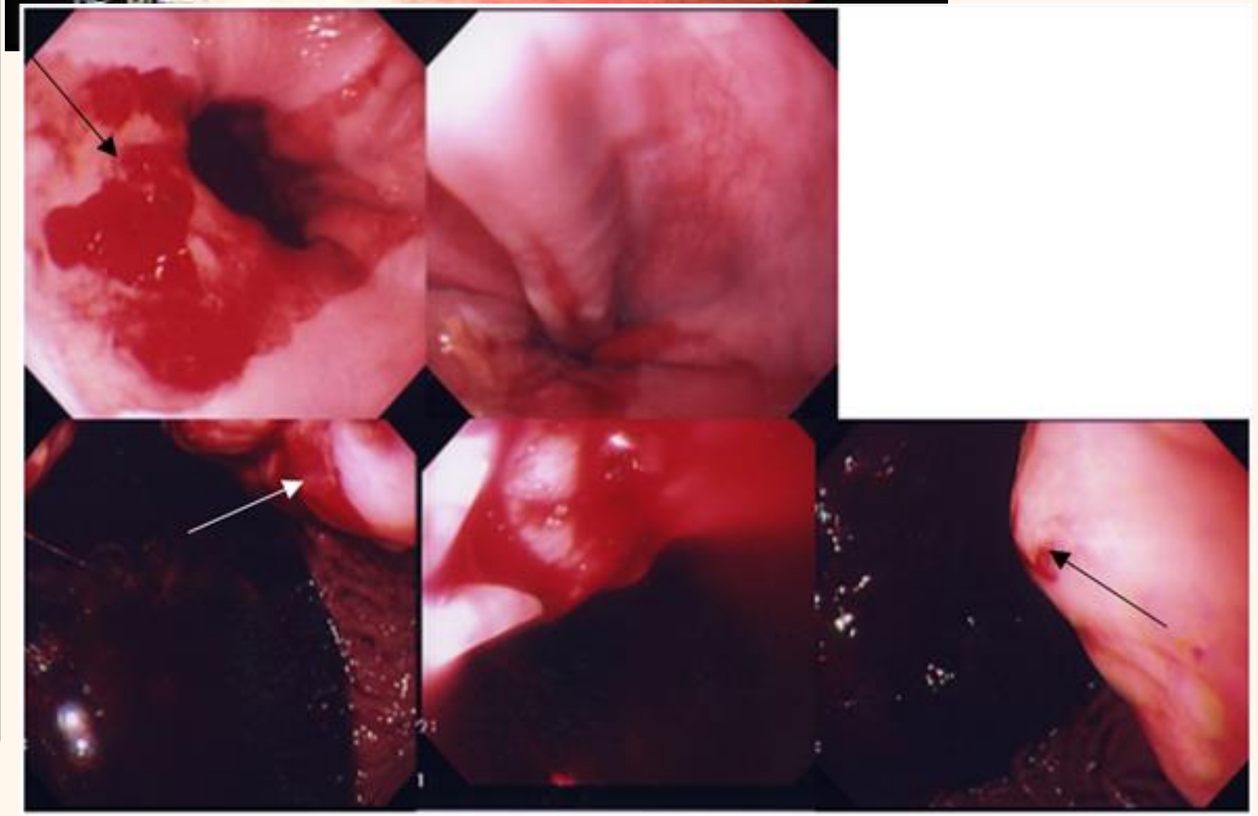
- ***Endoscopic*** (First line treatment)
  - Hemoclips placement
  - Electro coagulation or argon plasma coagulation
  - Endoscopic band ligation
- Uncomplicated gord
- Laparoscopic fundoplication
- Surgical ligation of blood vessels

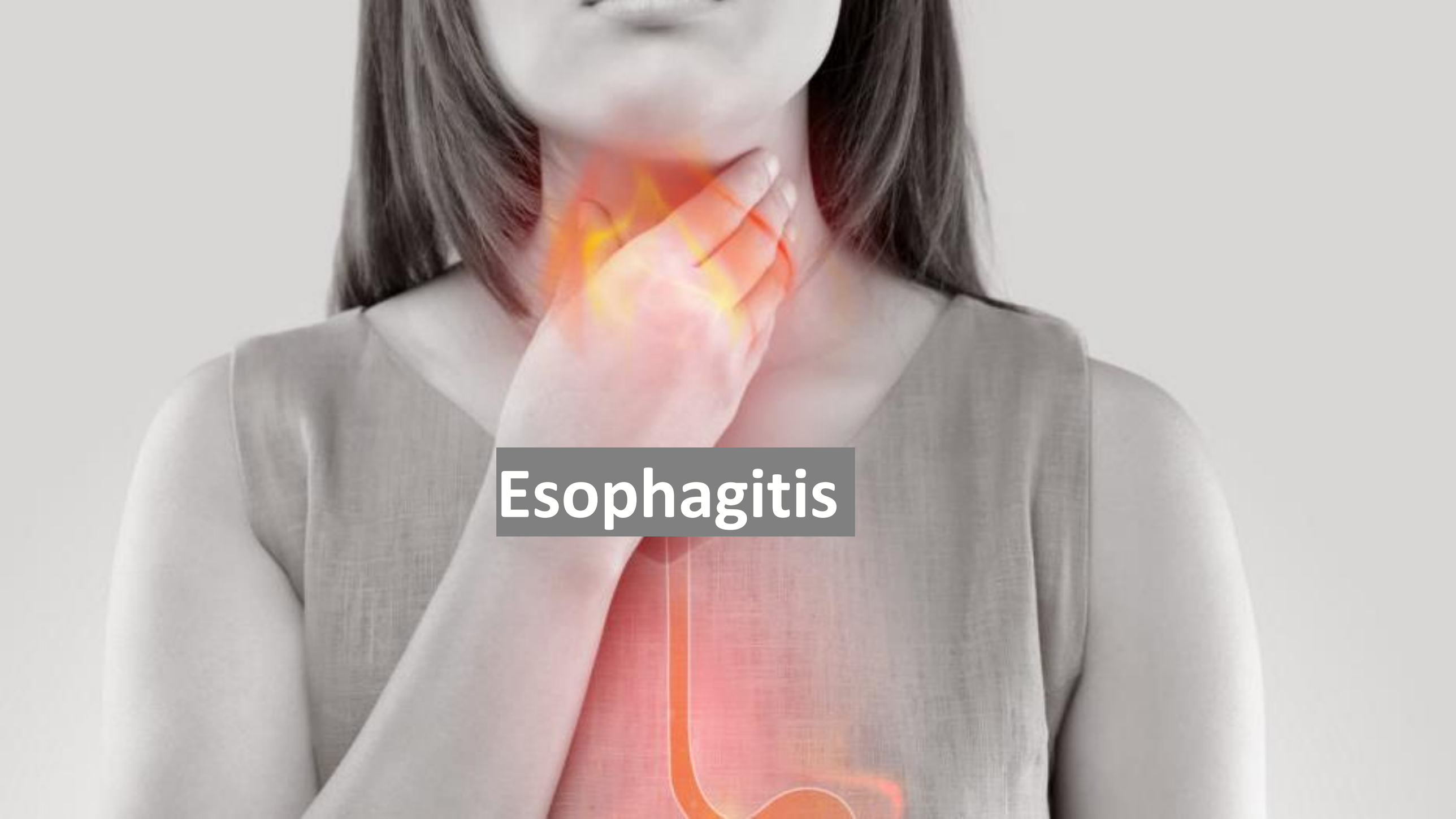
✓**Pharmacological treatment :**

- Orally or IV PPI (Acid suppression)
- Antiemetic therapy



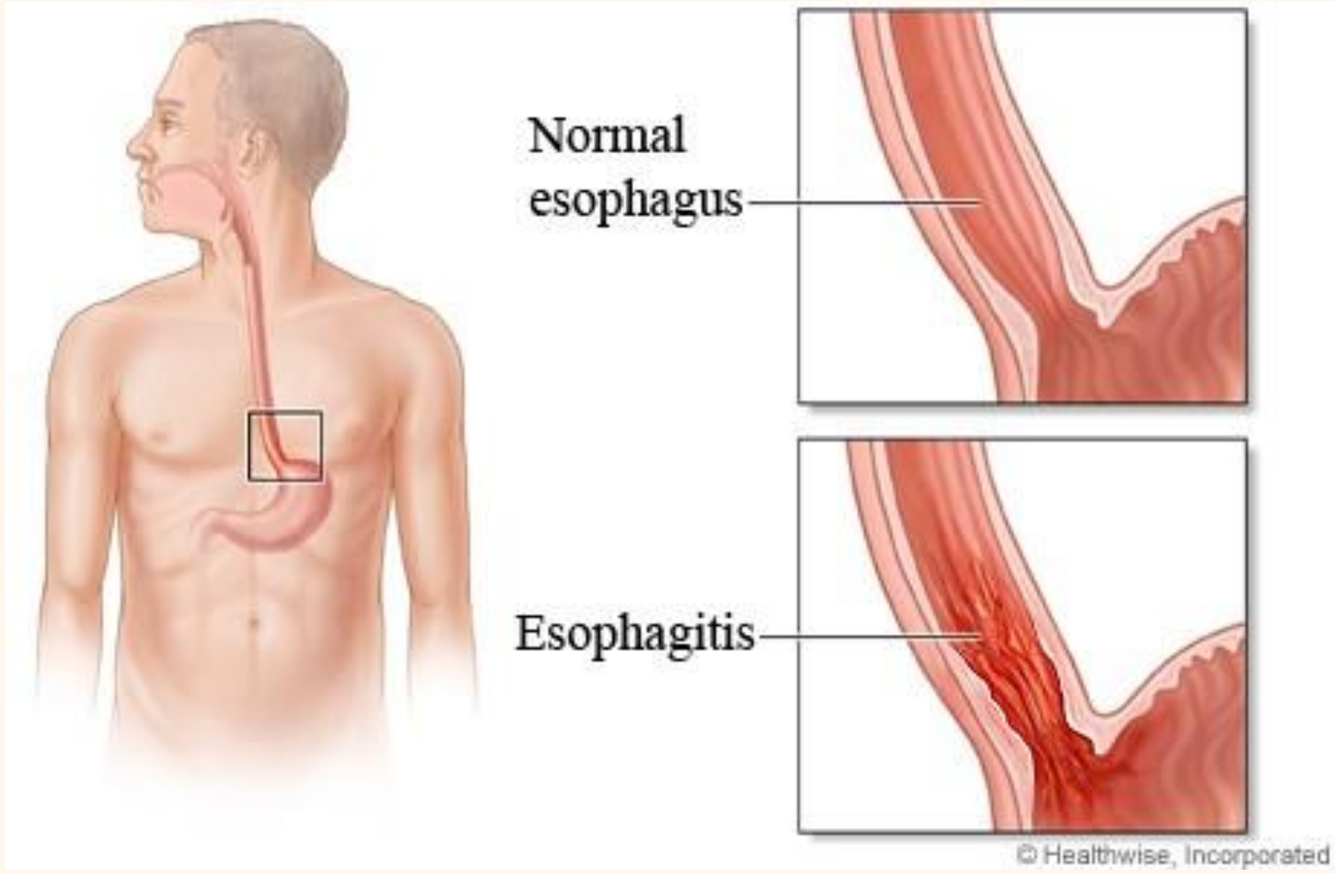





Esophagitis

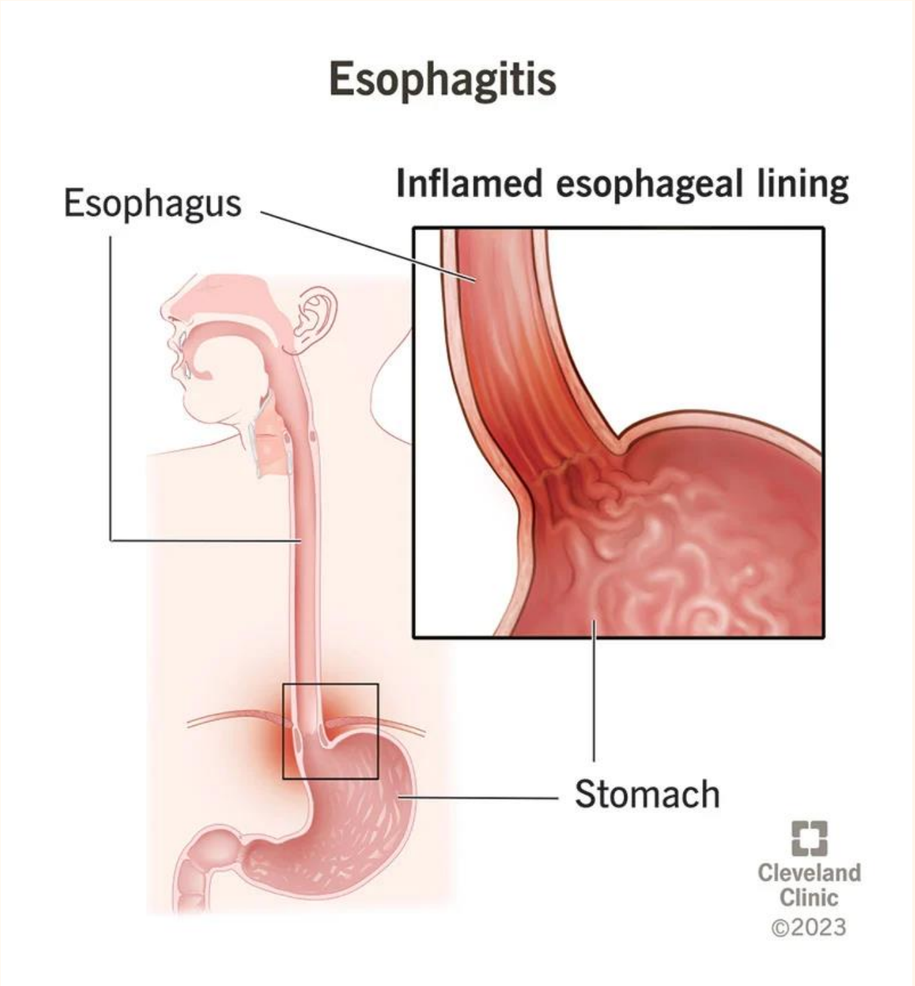
Definition:

Inflammation of the esophageal mucosa secondary to direct mucosal injury (e.g., GERD) or to an inflammatory process (e.g.., eosinophilic esophagitis).
5 – 10% of non variceal upper gastrointestinal tract bleeding cases are caused by esophagitis



Types

1. Eosinophilic esophagitis.
2. Infectious esophagitis.
3. Substance-induced esophagitis.
4. Medication-induced esophagitis.



Etiology:

Mechanism	Possible causes

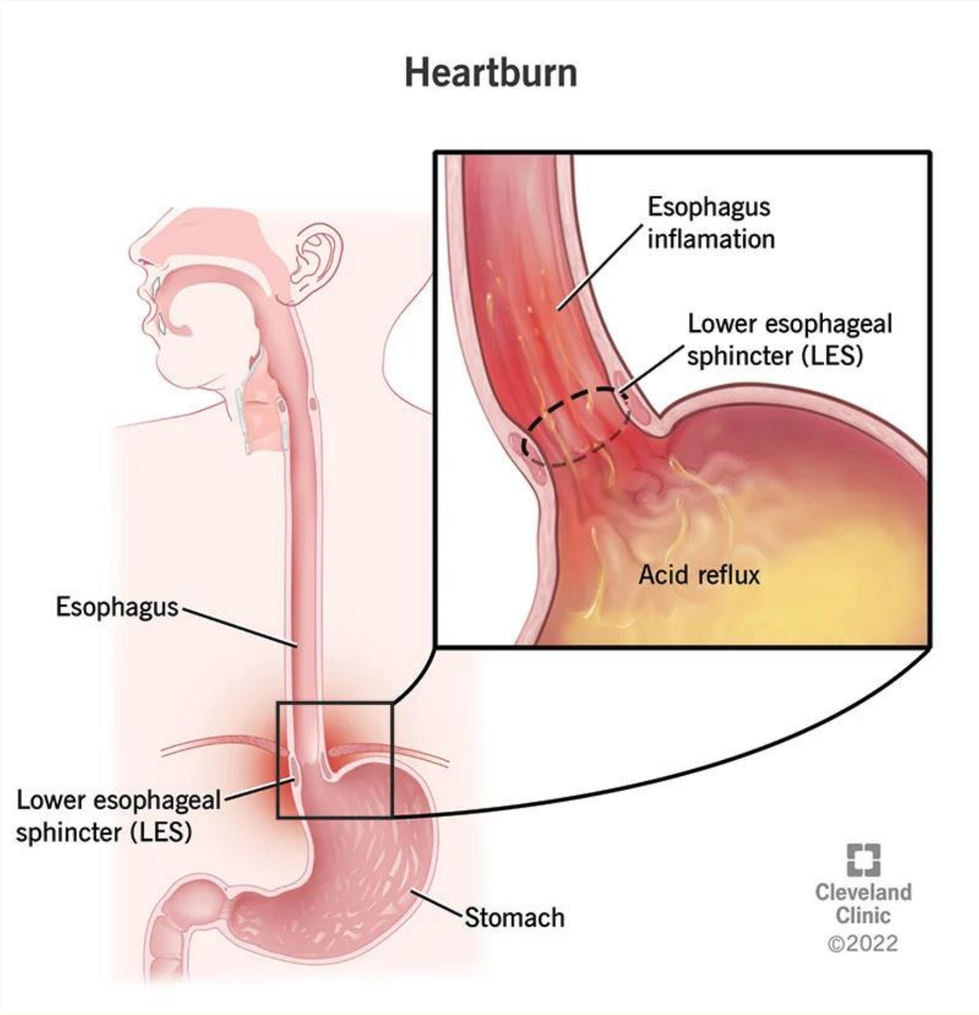
Mucosal injury	• Gastroesophageal reflux disease (GERD)
	• Infections
	  ◦ *Candida* spp.
	  ◦ Herpes simplex virus (HSV)
	  ◦ Cytomegalovirus (CMV)
	• Substance-induced esophagitis
	• Radiotherapy

Specific infiltrates	• Eosinophilic esophagitis • Lymphocytic esophagitis

Others	• Immune-mediated disorders, including:
	  ◦ Crohn disease
	  ◦ Autoimmune diseases (e.g., scleroderma, Behcet disease, systemic lupus erythematosus, Sjogren syndrome)
	  ◦ Graft-versus-host disease
	• Anatomical causes (e.g., hiatal hernia, esophageal rings, webs, diverticula)
	• Motility disorders (e.g., achalasia, muscular dystrophy)

Clinical features

- Retrosternal burning chest pain (heartburn)
- Dyspepsia.
- Regurgitation.
- Belching.
- Globus sensation.
- Nausea
- Vomiting
- Sore throat



Diagnosis

- **Endoscopy:**
- During this procedure, a long, thin tube equipped with a tiny camera is guided down the patient’s throat and into the esophagus. This instrument is called an endoscope.
- **Barium X-ray**
- **Laboratory tests and tissue sample**
- **Allergy tests**
- Skin-prick test or elimination diet

Treatment / Management

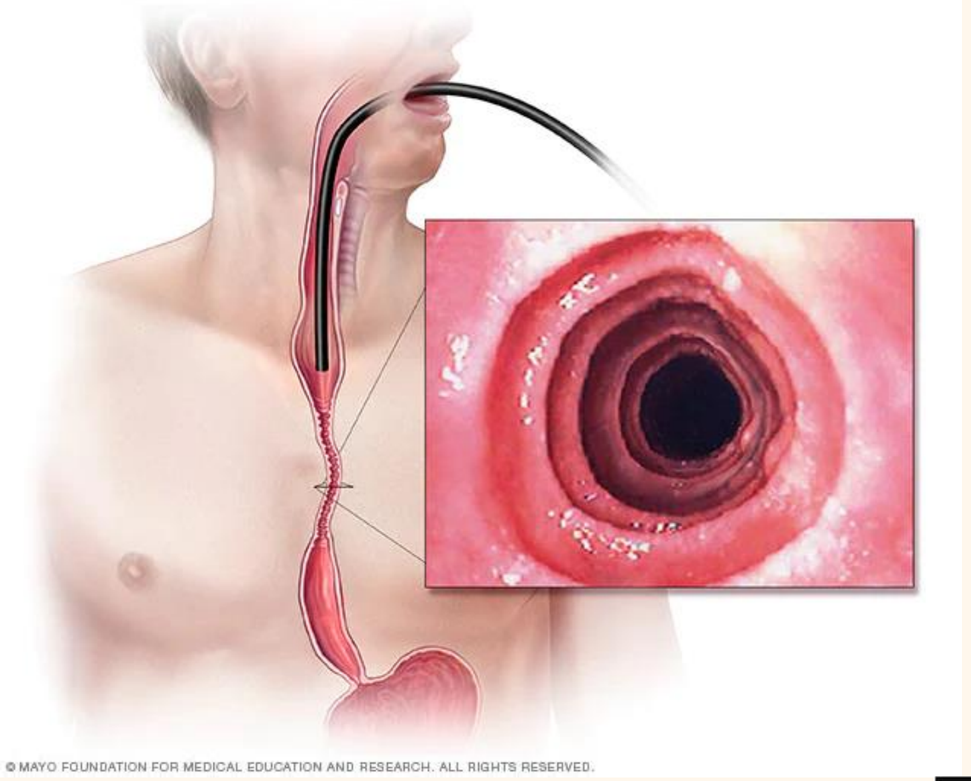
Treatment depends on the etiology but core principles of treatment include:

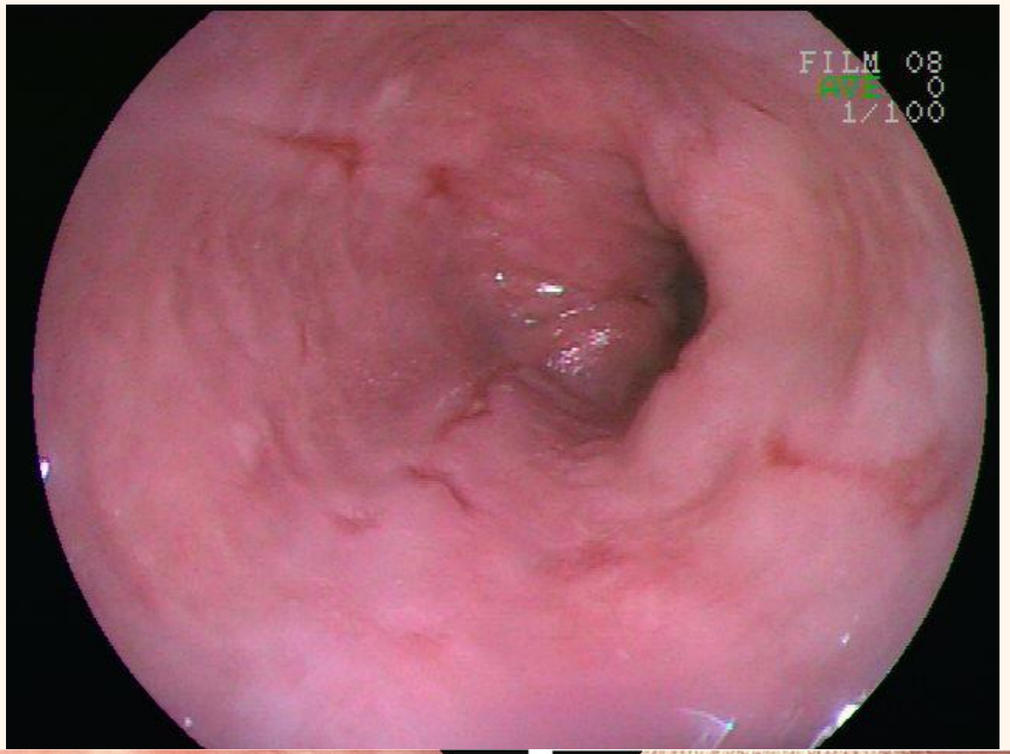
- acid suppression with PPI or H2 blockers
- lifestyle modification
- liquid to soft or pure diet to allow adequate time for healing and dietary modification.

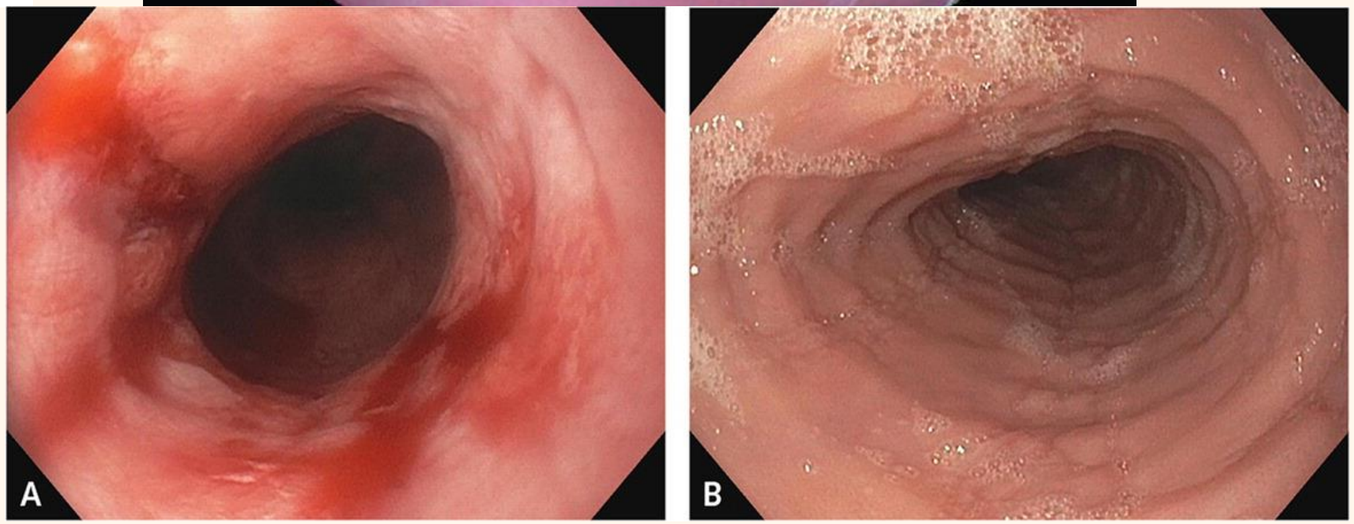
Complications

Complication of chronic and untreated esophagitis includes :

- Bleeding
- Stricture
- Barrett esophagus
- Perforation
- Laryngitis
- Aspiration pneumonitis



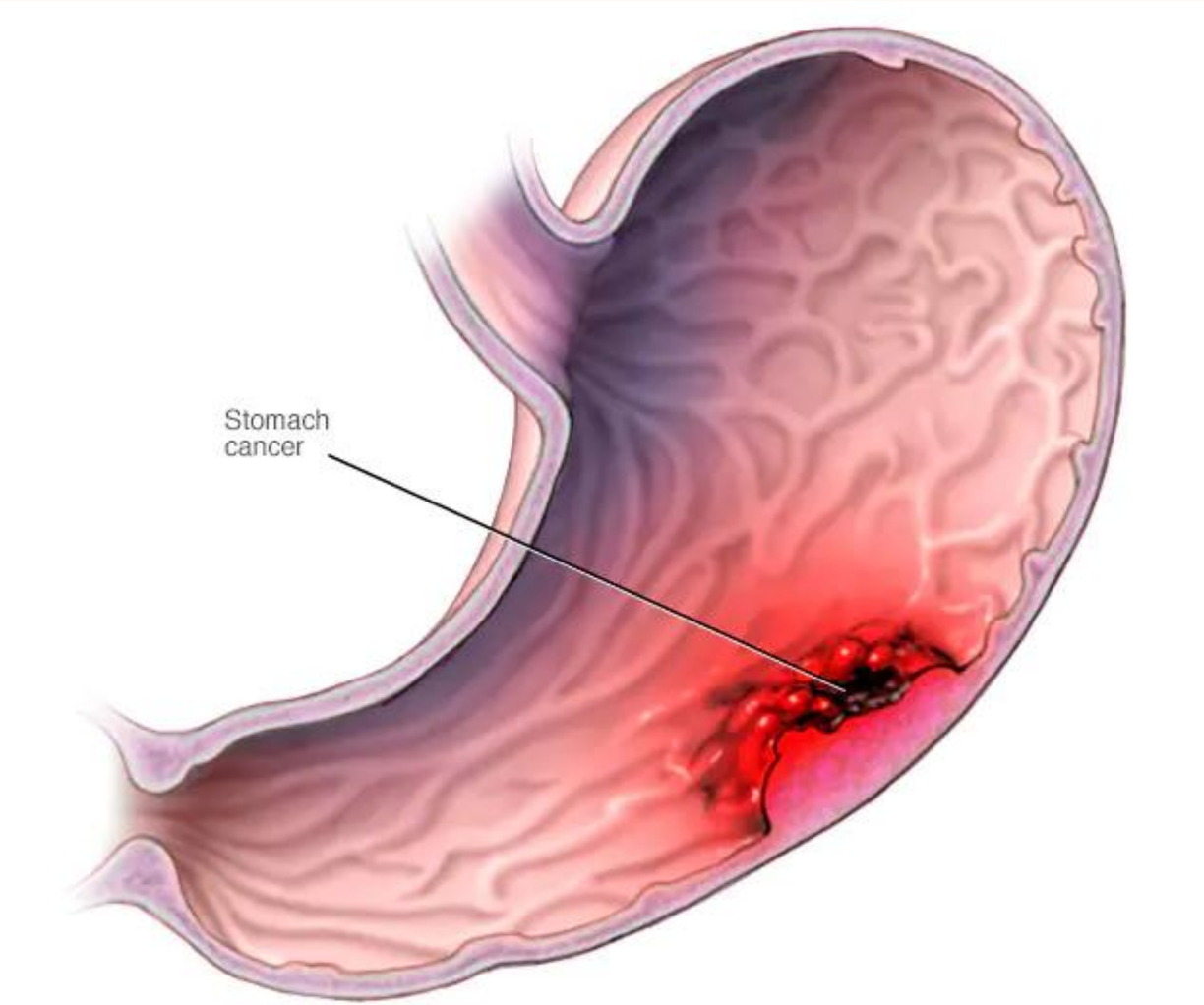




Tumors causing upper gastrointestinal tract bleeding:

Previous studies have reported that between 1% and 5% of non variceal upper GIB cases are secondary to malignancies.

Malignancies causing upper GIB are most commonly primary gastric cancers followed by primary esophageal and duodenal cancers.



Esophageal varices

Variceal bleeding <20%
gastroesophageal >90%

Presented by mahmoud hamdan

Definition

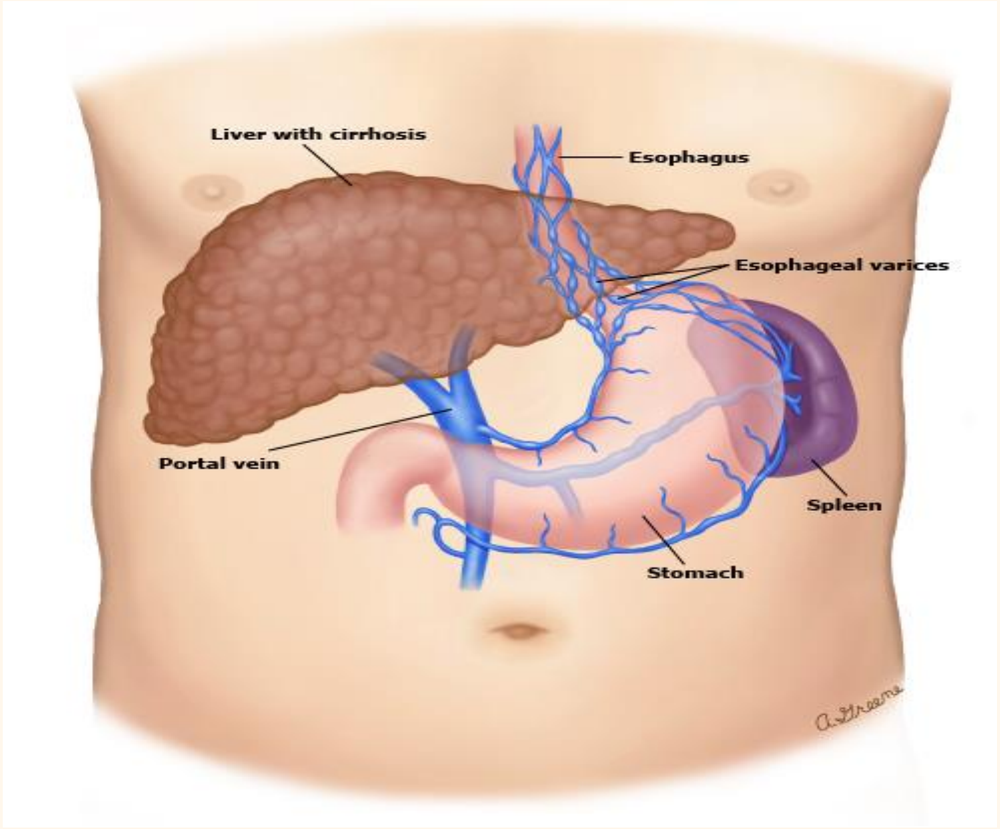
Esophageal varices are dilated collateral veins resulting from increased blood flow due to portal hypertension, often caused by cirrhosis. Nonbleeding varices are typically asymptomatic.
Affects ~ 50% of patients with cirrhosis [2][3]
Variceal hemorrhage is the most common lethal complication in patients with cirrhosis
How we classified the esophagel varices ?
Several classification methods exist for esophageal varices.
Bleeding (i.e., esophageal variceal hemorrhage) vs. non-bleeding
Degree of extension into the stomach
Size on endoscopy: [5]

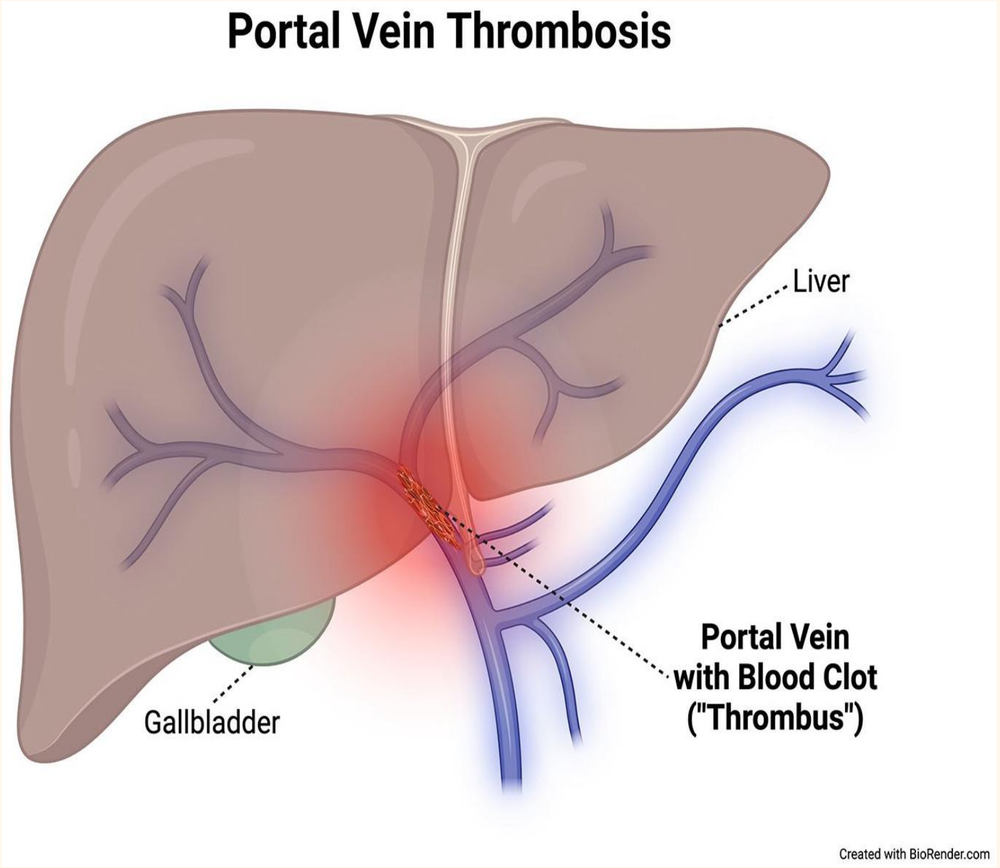
- Small esophageal varices: < 5 mm
- Medium/large esophageal varices: ≥ 5 mm

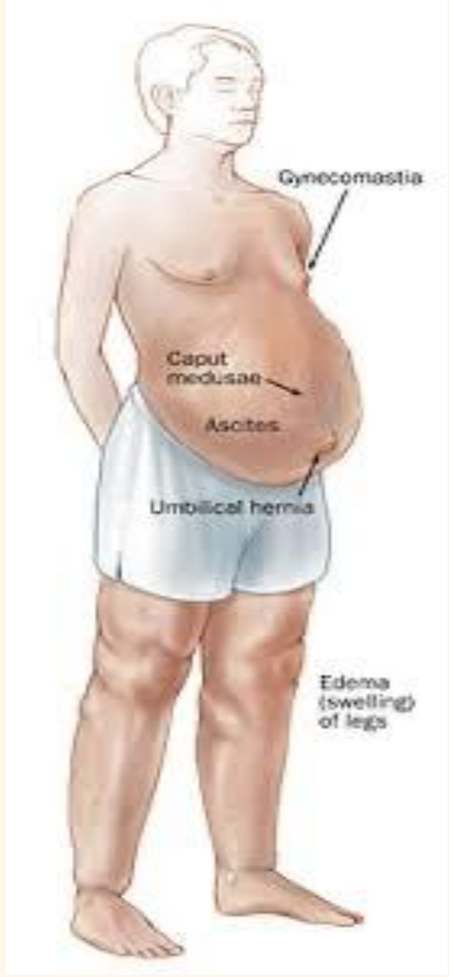
Acute variceal hemorrhage is a potentially life-threatening condition. Patients present with clinical features of gastrointestinal bleeding, e.g., sudden hematemesis and melena, and, in some cases, hypovolemic shock. In addition to stabilizing the patient, management involves administration of vasoactive medication and antibiotic prophylaxis in combination with endoscopic treatment. If the hemorrhage persists, balloon tamponade of the bleeding and/or an emergent transjugular intrahepatic portosystemic shunt (TIPS) may be necessary. Secondary prophylaxis of variceal bleeding involves nonselective beta blockers, EVL, and/or TIPS placement.

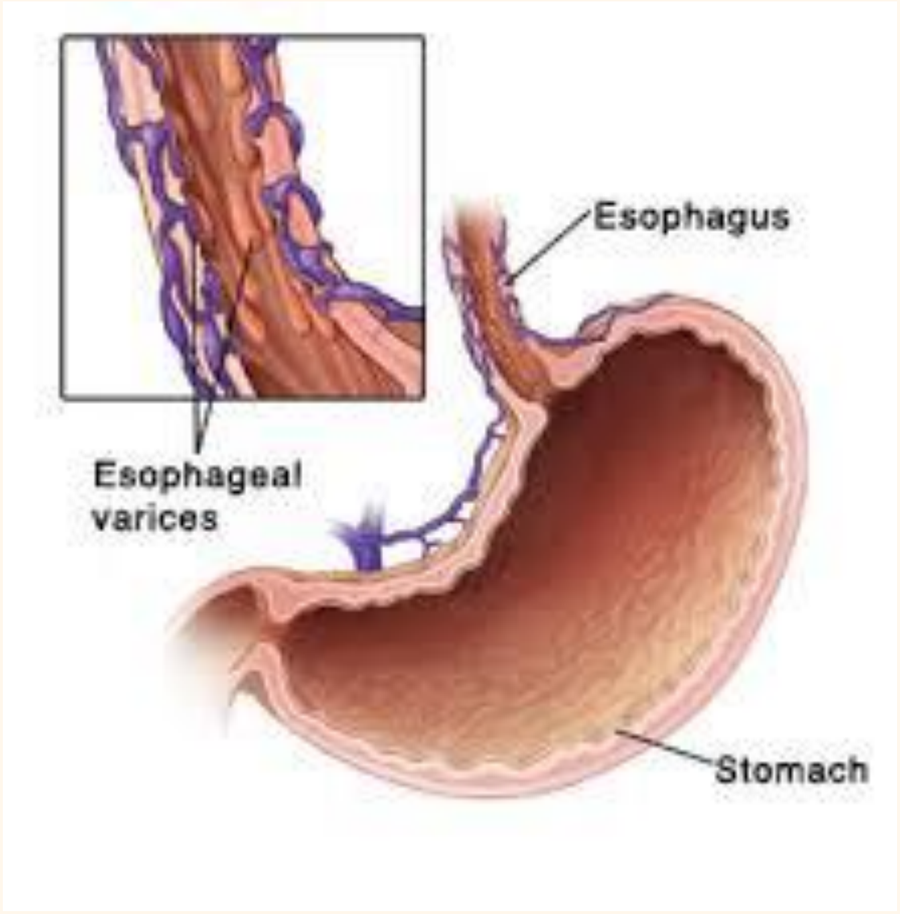
Etiology

Cirrhosis → portal hypertension → dilated sub-mucosal veins (varices) of the distal esophagus
See “Etiology” in “Portal hypertension.
Cirrhosis:The replacement of liver tissue with scar tissue secondary to chronic liver damage. Etiologies include alcohol use, hepatitis, diabetes, hypertension, and hemochromatosis. Clinical findings include jaundice, hepatosplenomegaly, ascites, skin changes (spider angiomata, palmar erythema, caput medusae), and hormonal changes (gynecomastia, hypogonadism, sexual dysfunction).

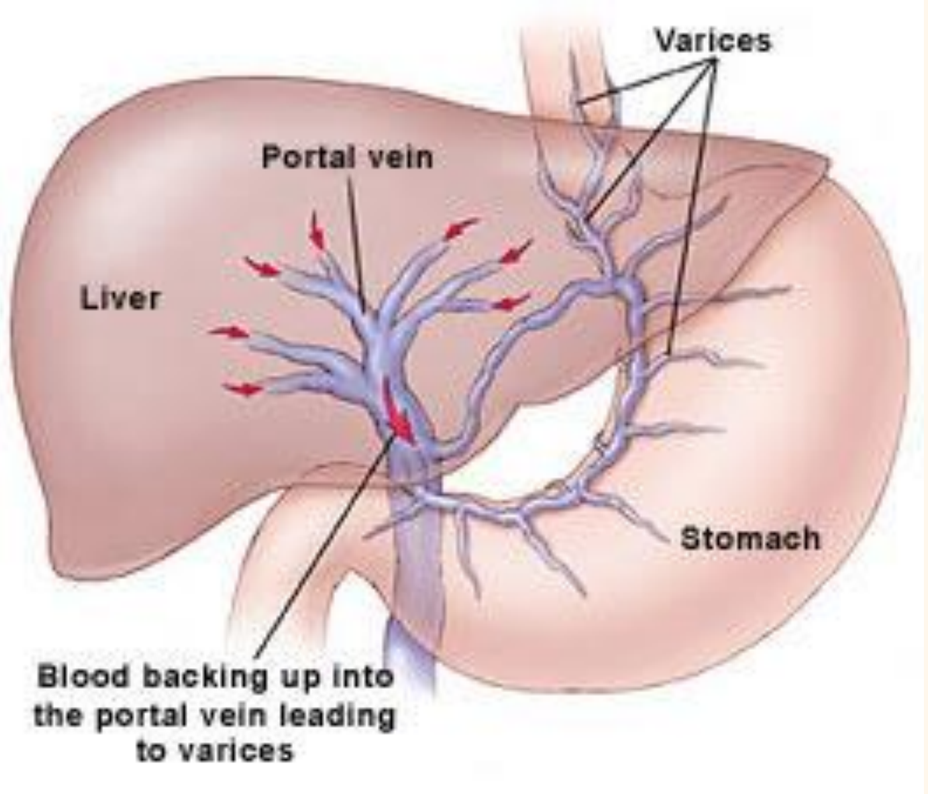

Liver with cirrhosis
Esophagus
Esophageal varices
Portal vein
Spleen
Stomach

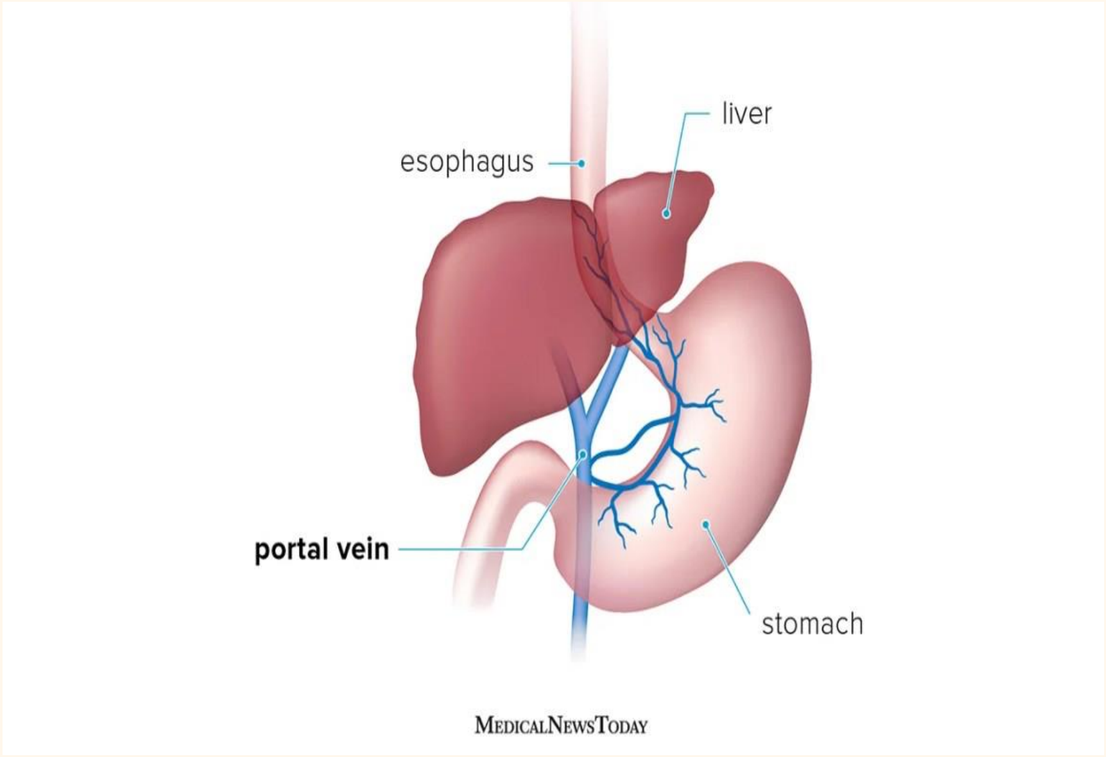

Portal Vein Thrombosis
Liver
Gallbladder
Portal Vein
with Blood Clot
("Thrombus")
Created with BioRender.com

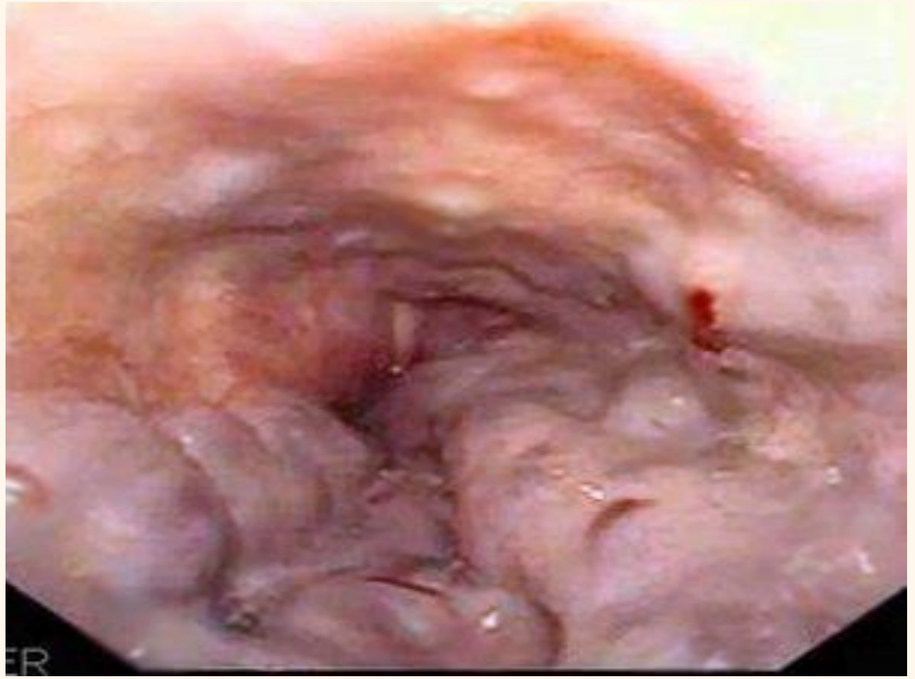

Gynecomastia
Caput medusae
Ascites
Umbilical hernia
Edema (swelling) of legs

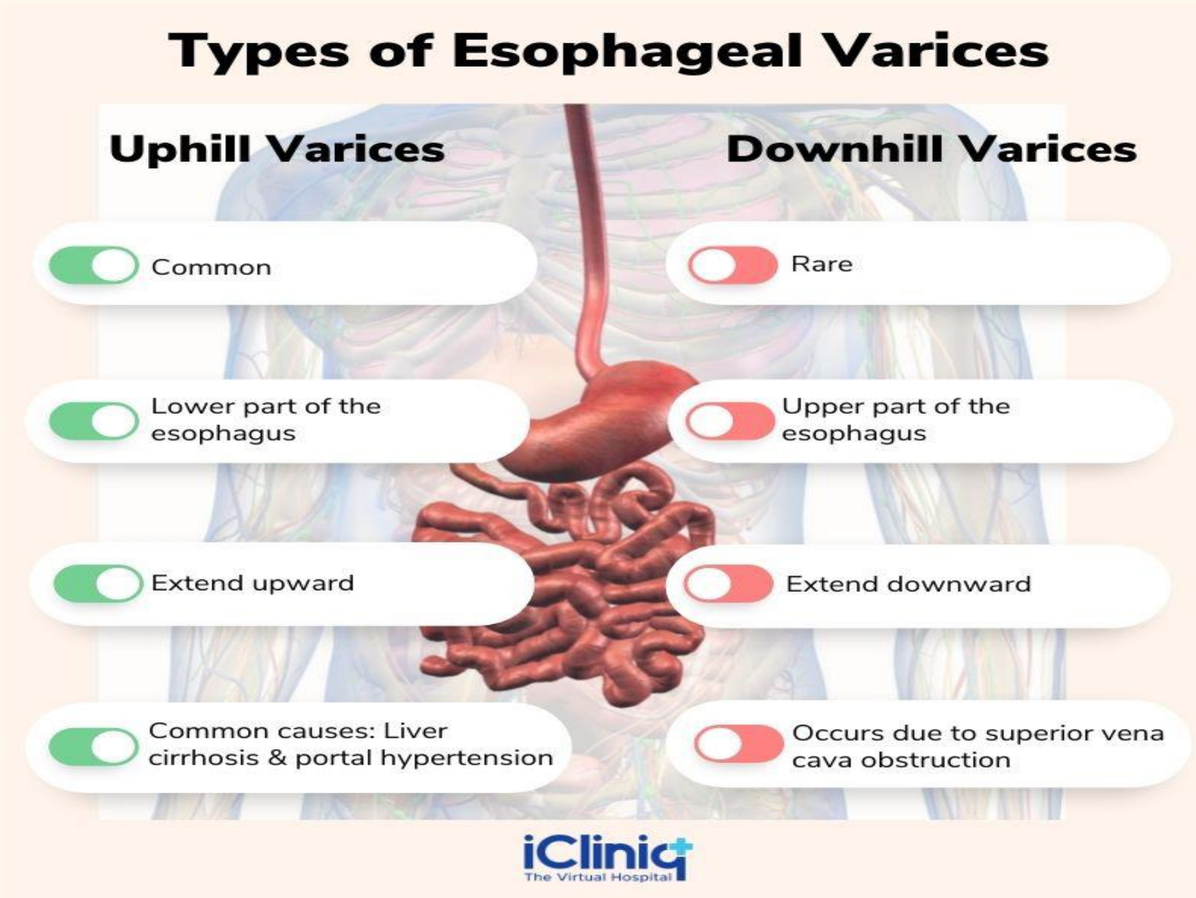

Esophagus
Esophageal varices
Stomach

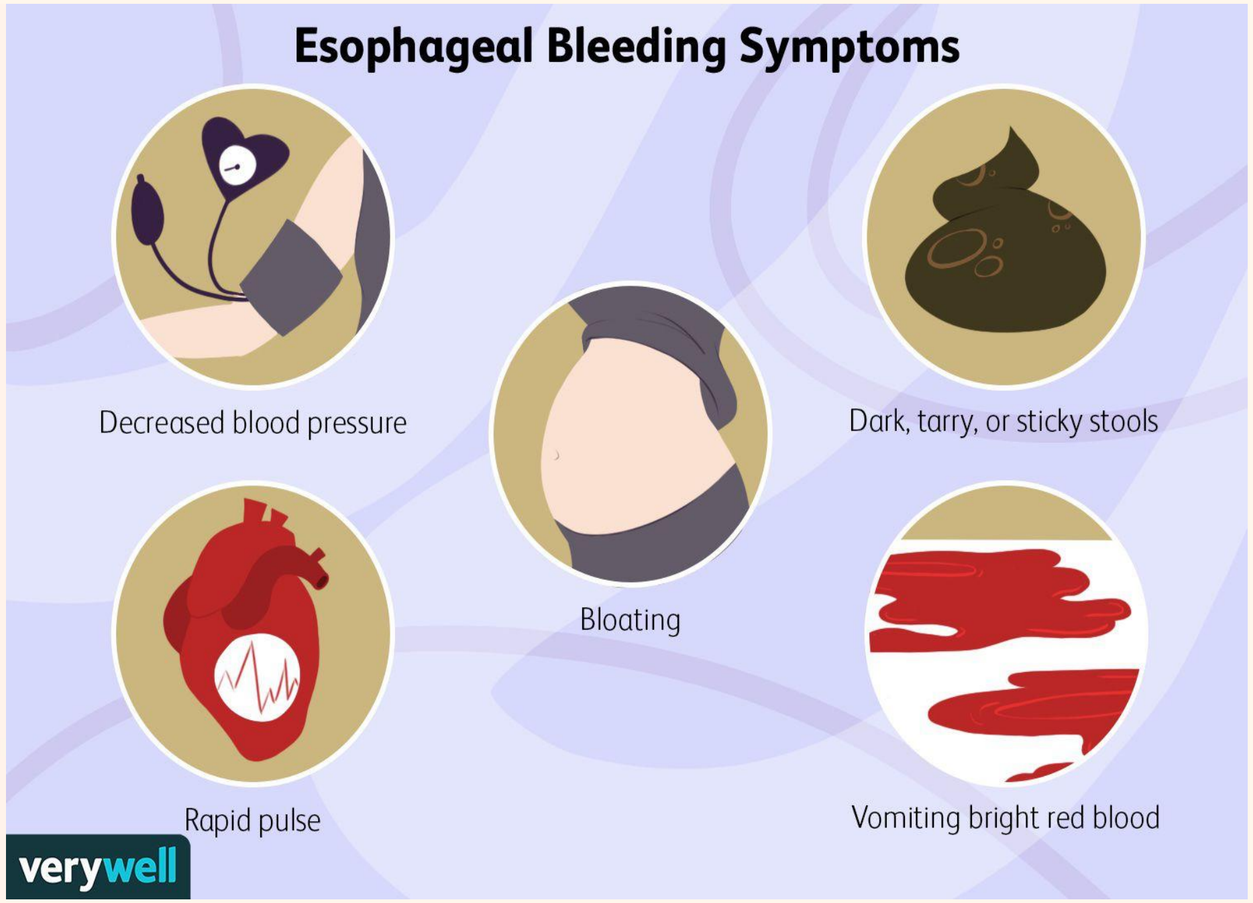

Cirrhosis of the liver
Late stage liver disease
Healthy liver
Compensated cirrhosis
(stage three liver disease)
Liver location
Decompensated cirrhosis
(stage four liver disease)
Cleveland Clinic ©2023


Varices
Portal vein
Liver
Stomach
Blood backing up into
the portal vein leading
to varices


liver
esophagus
portal vein
stomach
MedicalNewsToday




Types of Esophageal Varices
Uphill Varices
Downhill Varices
Common
Rare
Lower part of the esophagus
Upper part of the esophagus
Extend upward
Extend downward
Common causes: Liver cirrhosis & portal hypertension
Occurs due to superior vena cava obstruction
iClinic
The Virtual Hospital


Esophageal Bleeding Symptoms
Decreased blood pressure
Dark, tarry, or sticky stools
Bloating
Rapid pulse
Vomiting bright red blood
verywell

Clinical features

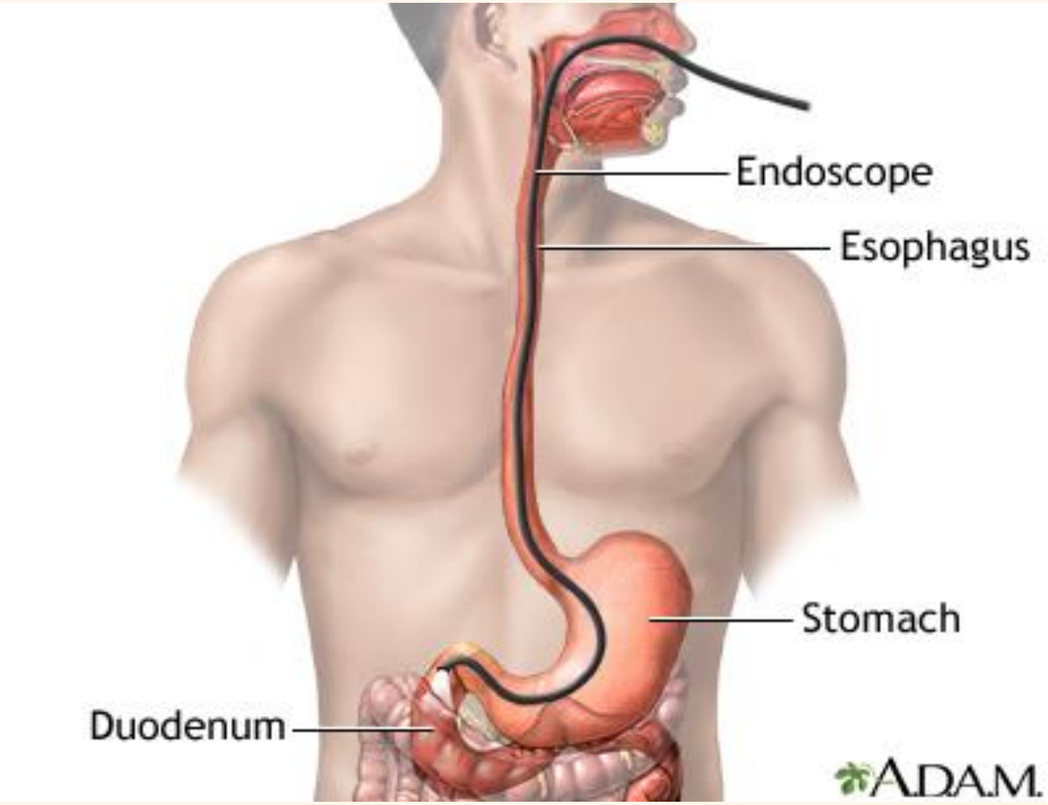
- **Nonbleeding varices**: typically asymptomatic
  - Patients may have other clinical features of portal hypertension.
- **Bleeding varices**: sudden onset of severe symptoms of gastrointestinal bleeding
  - Signs of hemorrhagic shock
  - **Hematochezia**
  - **Melena**
  - **Hematemesis**

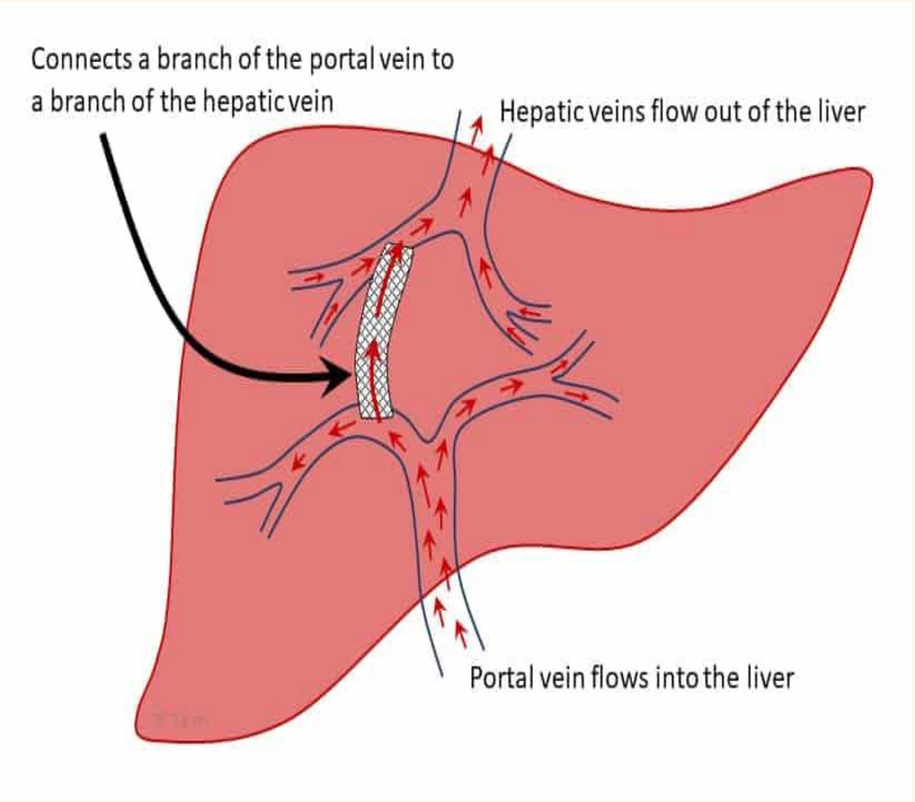
Risk factors

- Alcohol consumption
- Advanced liver disease
- Hypertension
- Size of esophageal varices
- Age

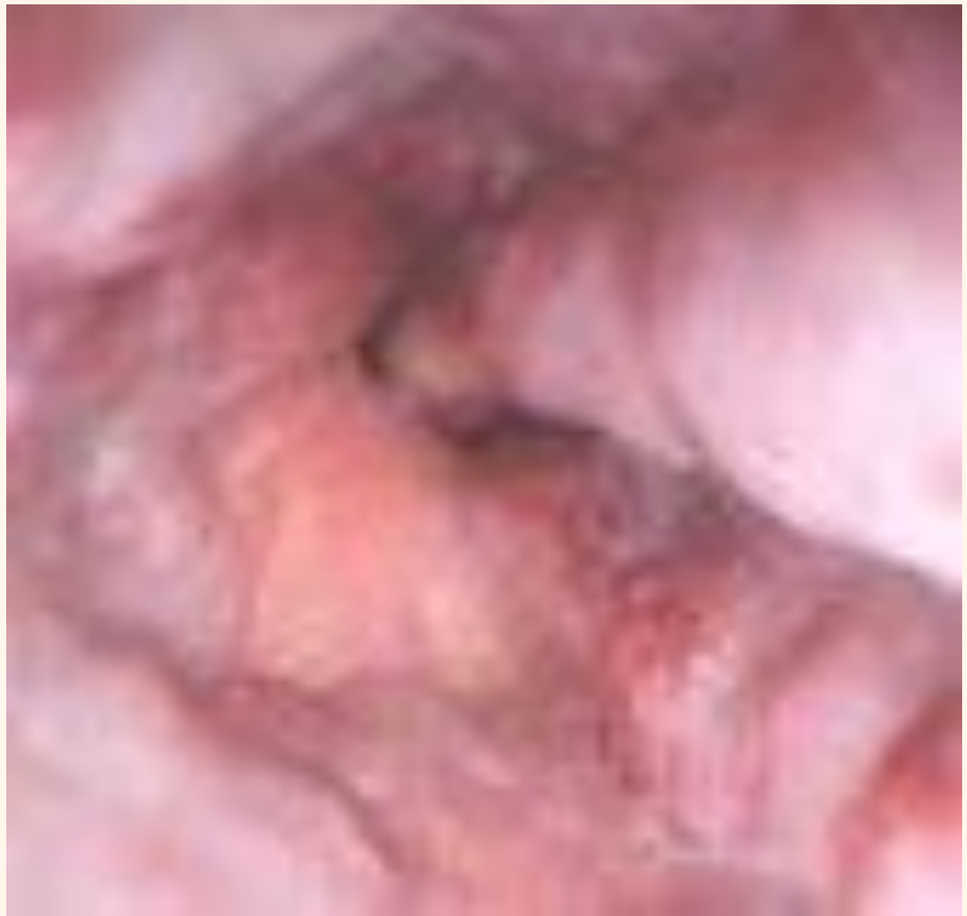
Diagnosis

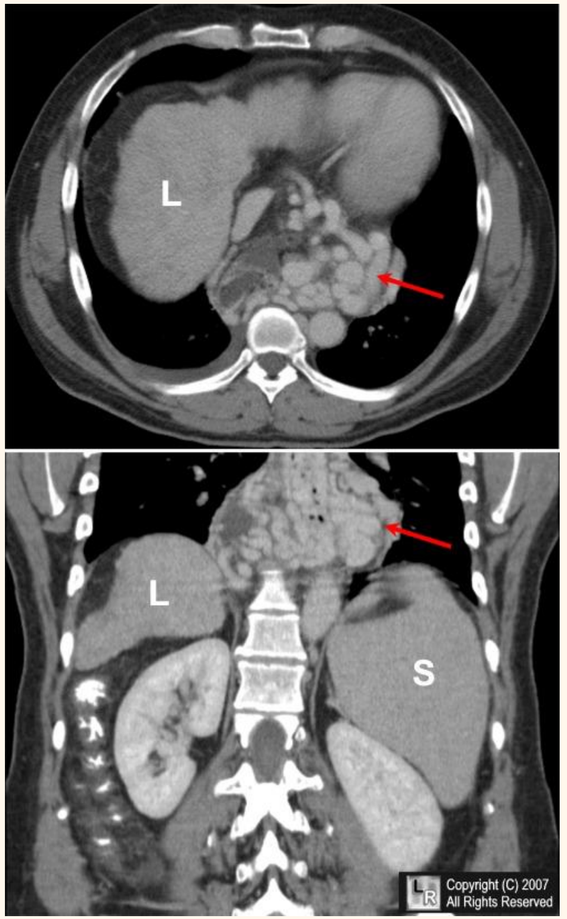
- Diagnosis and surveillance of esophageal varices requires esophagogastroduodenoscopy (EGD), with the goal of establishing: [2]
- Presence of varices
- Size of varices
- Stigmata of recent or impending bleeding (i.e., high-risk endoscopic findings): [4][7]
  - Red wale marks: longitudinal red streaks on the surface of a varices
  - Cherry-red spots
  - Hematocystic spots: raised spots that appear as blisters


Endoscope
Esophagus
Stomach
Duodenum
A.D.A.M.


Connects a branch of the portal vein to
a branch of the hepatic vein
Hepatic veins flow out of the liver
Portal vein flows into the liver

Imaging is not routinely indicated but large esophageal varices may be incidentally identified. Transient elastography and CBC may be used to rule out high-risk esophageal varices but are not routinely used for confirming the diagnosis.





Esophageal varices. An enhanced axial (top) and coronal reconstruction CT scan of the upper abdomen shows markedly tortuous and dilated varices surrounding the lower esophagus. The liver (L) is small and nodular from cirrhosis and the spleen (S) is enlarged from portal hypertension.

Management

- Management of nonbleeding esophageal varices focuses on the prevention of bleeding and involves regular surveillance and, in some cases, primary prophylaxis of bleeding using nonselective beta blockersor eradication of varices using endoscopic variceal ligation (EVL).
- **Pharmacological prophylaxis** [3]
- Nonselective beta blockers (recommended) [2]
  - Propranolol OR nadolol
  - Carvedilol (alternative)
- Dangerous!!!!!!!!!!!
- Reduce the dose or discontinue beta blockers if ascites or hepatorenal syndrome develop or systolic blood pressure is < 90 mm Hg


NDC 0517-1030-01 Rx Only
Vasopressin
Injection, USP
200 Units per 10 mL
(20 Units per mL)
For Intravenous Infusion.
Must be diluted prior to use.
10 mL Multiple-Dose Vial


Octreotide
Injection
Octride 100
100 µg/ml

Management

- **Endoscopic variceal ligation (EVL)**
- An endoscopic procedure in which esophageal and/or gastric varices are ligated using rubber bands.
- Repeat every 1–8 weeks until varices are eradicated.
- Obtain surveillance EGD within 1–6 months of eradication and every 6–12 months thereafter
- Combination therapy with EVL and pharmacotherapy is not recommended for primary prophylaxis of esophageal variceal hemorrhage.
- Medium or large esophageal varices: Provide either pharmacological prophylaxis or EVL. [2]
- Small esophageal varices with high-risk features for esophageal variceal hemorrhage: Provide pharmacological prophylaxis as indicated.

Management of esophageal variceal hemorrhage

- Esophageal variceal hemorrhage is a medical emergency.
- What is the main approach of the condition?
- Stabilize the patient: See "Initial management of overt gastrointestinal bleeding."
  - Initiate IV fluid resuscitation.
  - Transfuse packed red blood cells to maintain hemoglobin > 7 g/dL.
  - Intubate patients who have altered mental status and/or severe, ongoing hematemesis.
- Consult gastroenterology for EGD and further management immediately.
- Start vasoactive medication: octreotide OR vasopressin infusion
- Administer antibiotic prophylaxis.
- Begin prophylaxis to prevent recurrence.

Pharmacological treatment

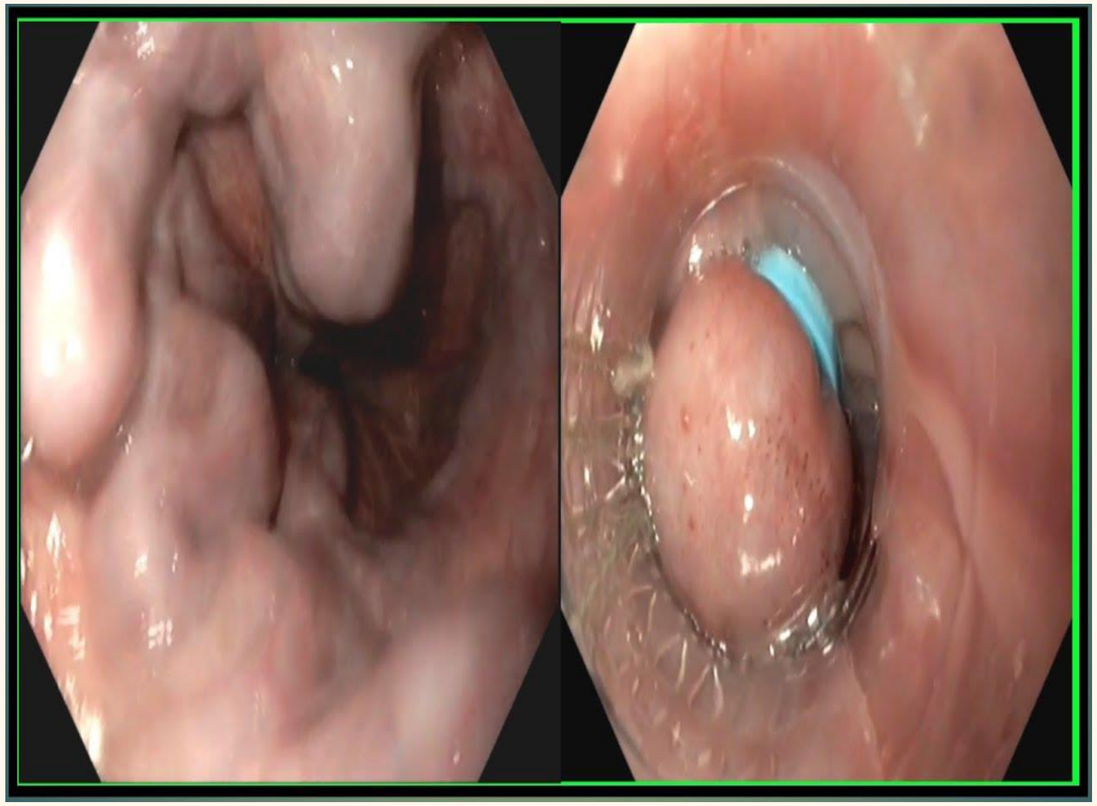
- Vasoactive medication and antibiotic prophylaxis are indicated for all patients. [2][5]
- **Vasoactive medication**
  - Reduces mortality and the need for blood transfusions by reducing splanchnic blood flow
  - Preferred agents
    - Octreotide for 2–5 days
    - OR vasopressin for 24 hours [2][5]
- **Antibiotic prophylaxis** [2]
  - Associated with decreased rates of infection, recurrent hemorrhage, and death
  - Preferred agent: ceftriaxone for a maximum of 7 days
- **Hepatic encephalopathy prevention**: Consider lactulose
- Esophageal variceal bleeding is a consequence of portal hypertension, and therefore treatment focuses on reducing portal hypertension rather than the correction of coagulation abnormalities.

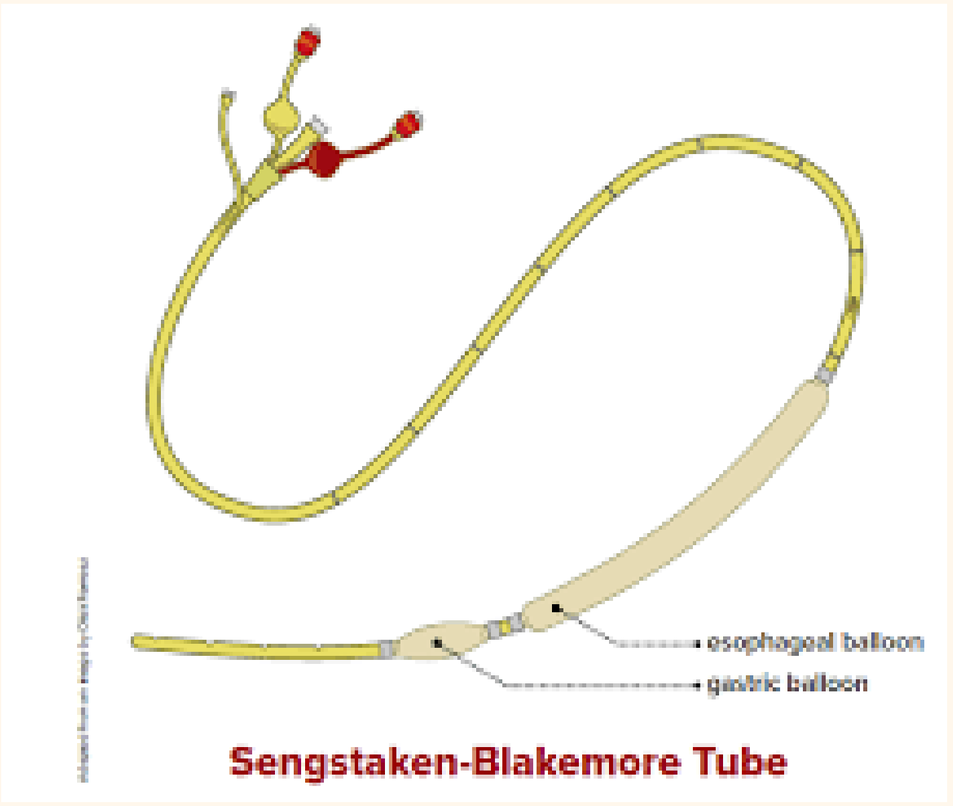
Endoscopic treatment

- **Variceal ligation**
  - Preferred intervention
  - Consider prokinetic treatment with **erythromycin** prior to EGD. [12]
- **Variceal sclerotherapy**
- intravariceal or paravariceal injection of a sclerosant agent such as ethanolamine. These agents provoke a severe inflammatory reaction within or around the varix that leads to variceal thrombosis and obliteration.
  - Injection of a sclerosant into, or adjacent to, the varix
  - Used when variceal ligation is technically difficult
  - **Balloon tamponade**
- **Definition**: orogastric tubes with esophageal and gastric balloons that tamponade bleeding when inflated
  - Sengstaken-Blakemore tube: orogastric tube with an esophageal balloon, gastric balloon, and gastric aspiration port
  - Minnesota tube: modification of Sengstaken-Blakemore tube with an additional aspiration port above the esophageal balloon and larger gastric balloon
- A Sengstaken–Blakemore tube is **a medical device inserted through the nose or mouth** and used occasionally in the management of upper gastrointestinal hemorrhage.

Prevention of recurrent variceal bleeding

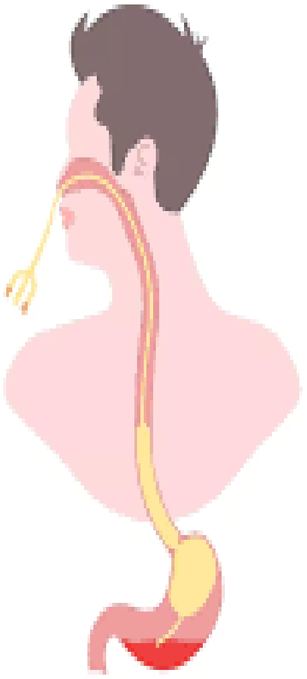
- **Patients without TIPS**: combination therapy with nonselective beta blockers and EVL
- **Patients with TIPS**: No additional treatment is indicated.
- The combination of EVL and nonselective beta blockers for the prevention of recurrent esophageal variceal hemorrhage is more effective than either therapy alone.

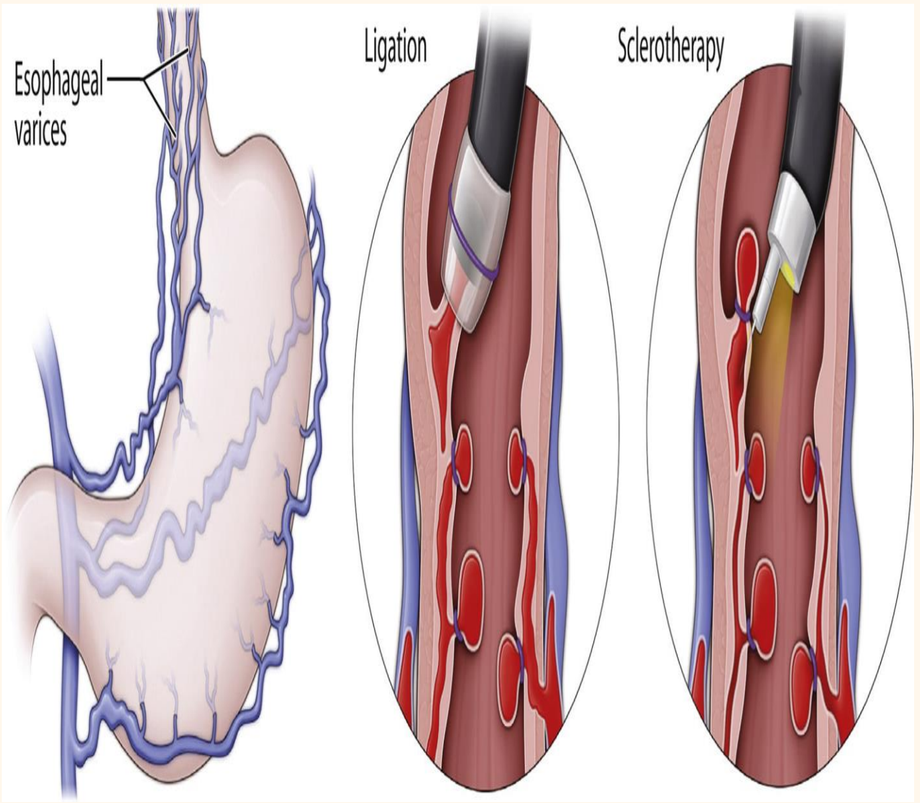



esophageal balloon
gastric balloon
Sengstaken-Blakemore Tube

What is Balloon Tamponade?

Balloon tamponade is a medical procedure in which a balloon is inserted into the body and inflated to stop or control bleeding.





Important complications

- Esophageal perforation
- Infection: e.g., aspiration pneumonia, spontaneous bacterial peritonitis, bacteremia
- Acute kidney injury: e.g., hepatorenal syndrome, acute tubular necrosis
- Hepatic encephalopathy
- Hematologic: anemia, coagulopathy, thrombocytopenia

Prognosis

- Six-week mortality rate after a variceal bleeding event is ~ 20%. [3][6]
- Risk of rebleeding within 1 year if left untreated is ~ 60%

Gastric varices [very rare]

- Isolated gastric varices (IGV) are gastric varices in the absence of esophageal varices.
- IGV is one of the rare causes of gastrointestinal bleeding and an uncommon complication of pancreatic neuroendocrine tumors (PNET).
- The gold standard diagnostic tool of varices is esophagogastroduodenoscopy (EGD).

REFERENCES :

AMBOSIS

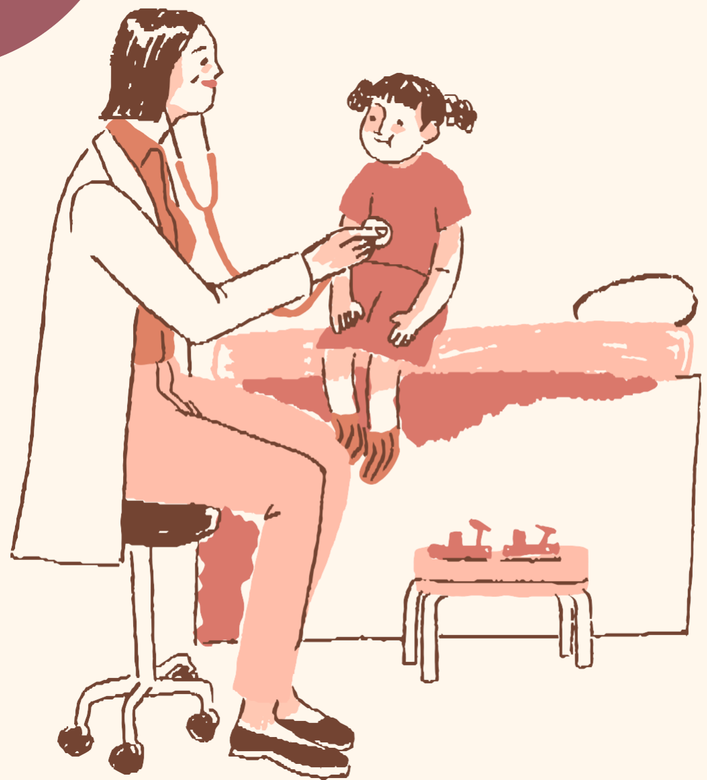
BEILY AND LOVES SURGERY

MACLEOD CLINICAL EXAMINATION
SURGICAL RECALL

OSMOSIS

KEN HUP

ALAMTARY



Finally !!!!!!

Thank you
God bless you all