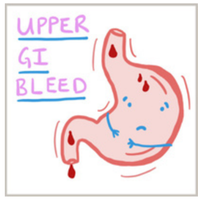
Upper gastrointestinal bleeding



Supervised by:
Dr.Mohammad Nofal

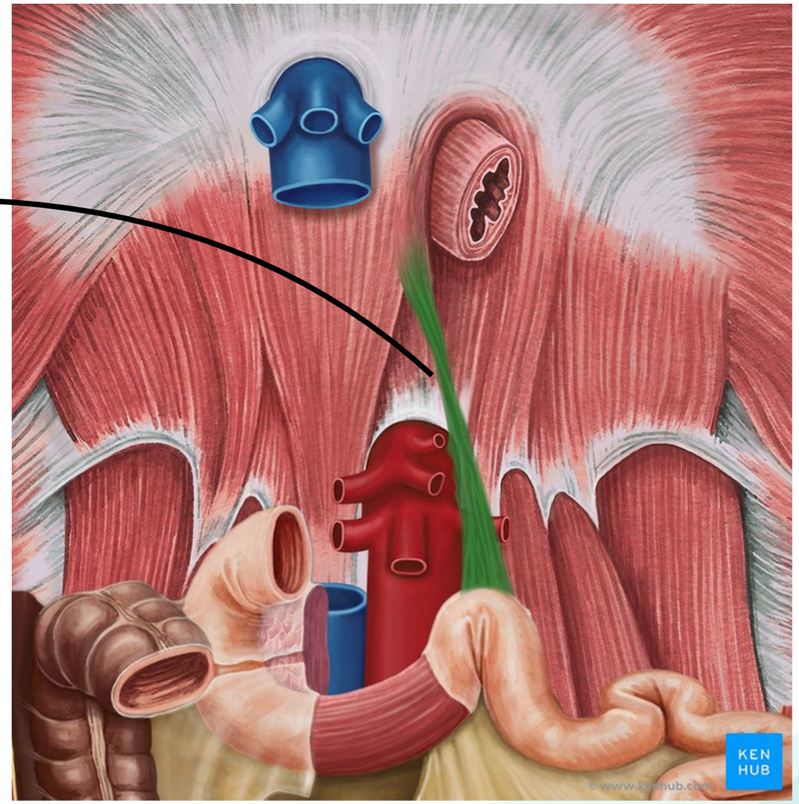
Presented by :Dima Khaled
Jana al sawadha
shaima alhroub
areen mahadeen
kawther Al-Habashneh

Presented by:Shaima alhroub

Definition:

bleeding derived from a source proximal to the ligament of Treitz which connects the duodenum of the small intestines to the diaphragm and marks the beginning of the jejunum.

Ligament of Treitz

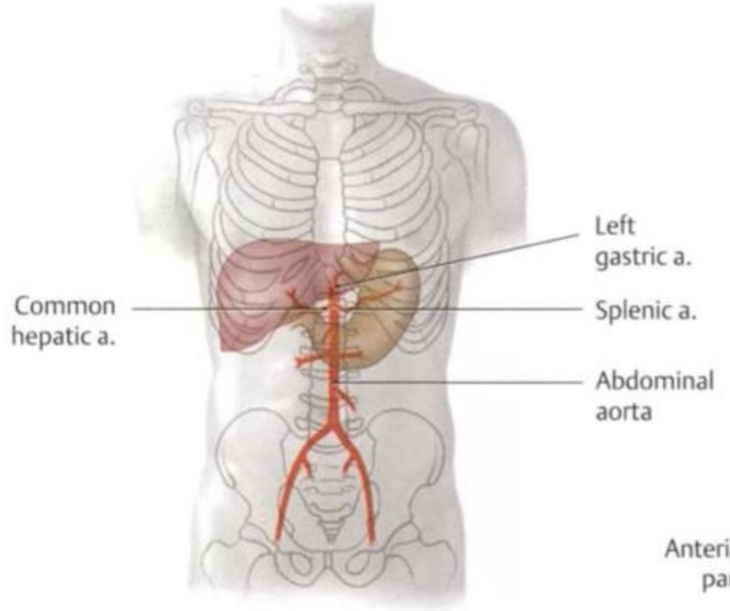


A fold of peritoneum that attaches the duodenojejunal flexure to the retroperitoneum. Can be used as a landmark to distinguish between the upper and lower gastrointestinal tracts.

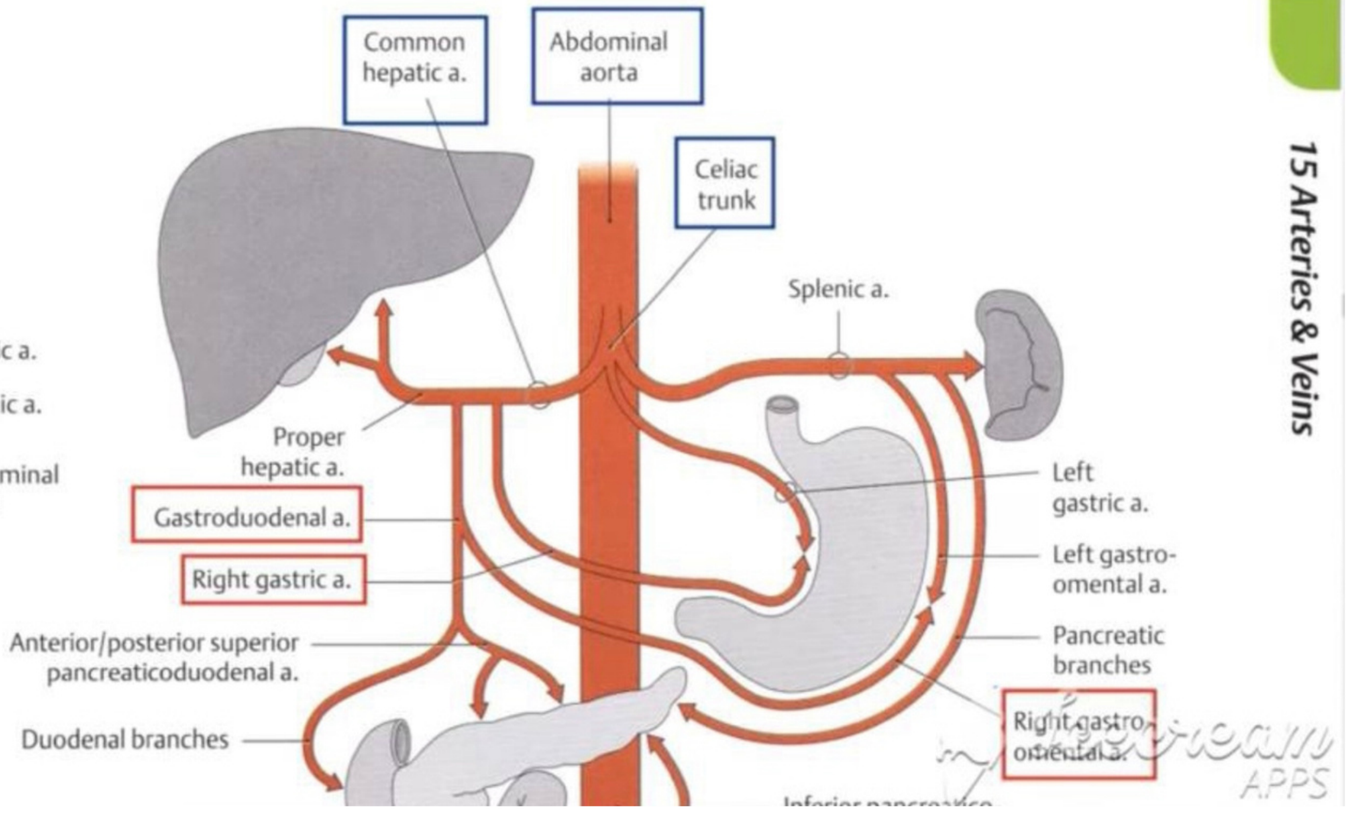
UGITB CLASSIFICATION :

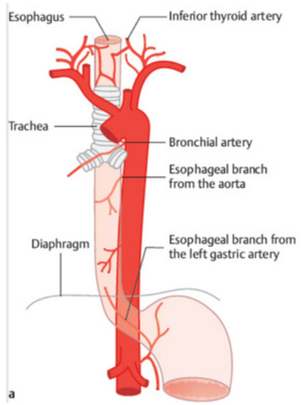
The source of bleeding either arterial (non varicela) or venous (variceal) in the UGIT i.e. within either oesophagus, stomach, Duodenum, UGITB isn't a disease it a complication of a veriety of disease that cause either venous or arterial bleeding.

***Fig. 15.2* Celiac trunk**



A Location, anterior view.

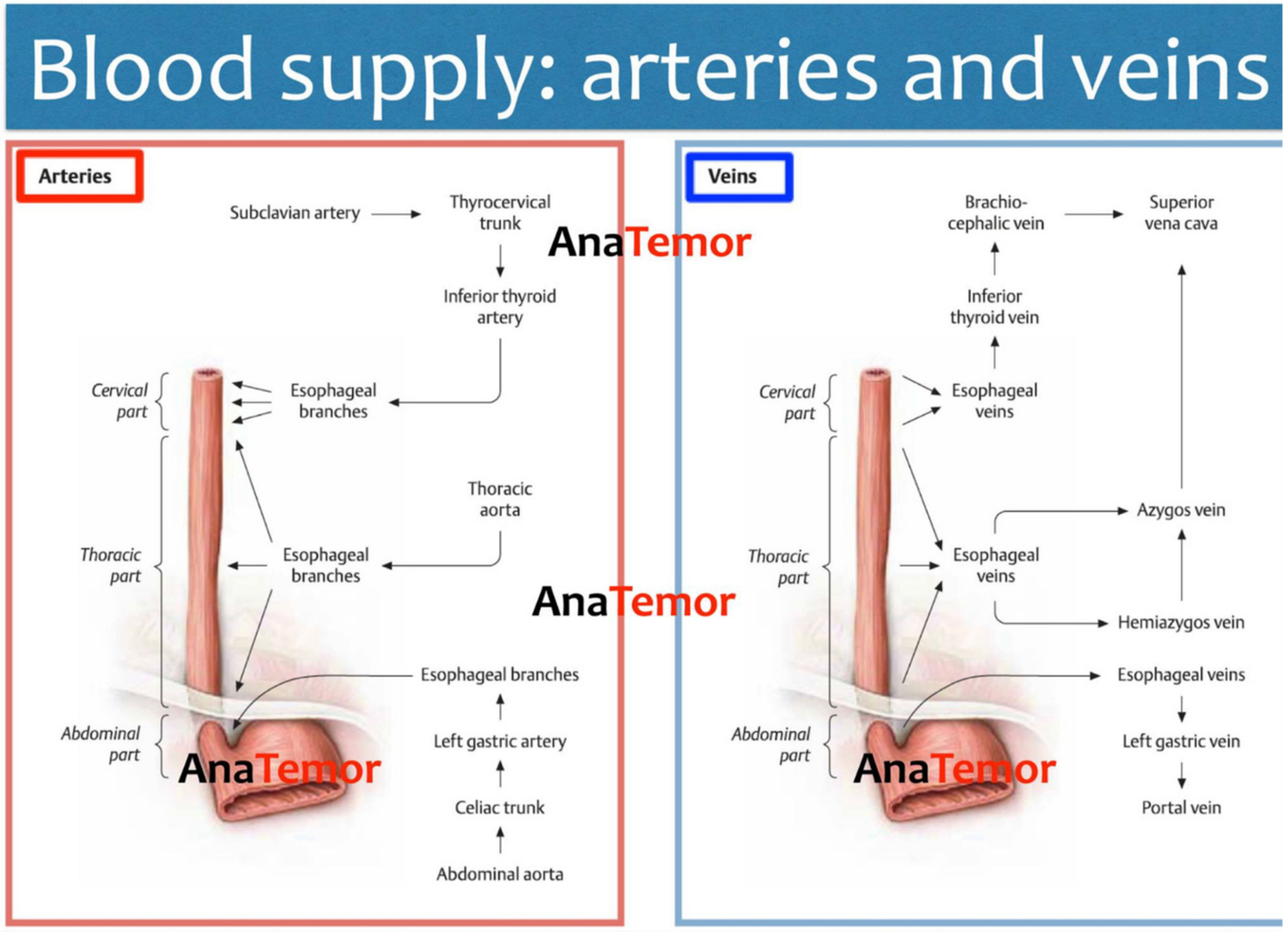


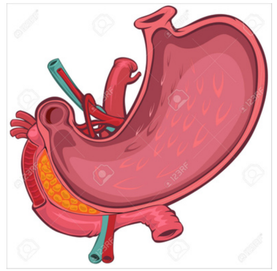


OESOPHAGUS blood supply:

	arterial	Venous
cervical	Inferior thyroid artery	into brachiocephalic vein
Thoracic	Oesophageal branches from aorta	Azygos vein
Abdominal	Oesophageal branches from short gastric arteries	Left gastric then to portal vein

OESOPHAGUS blood supply:

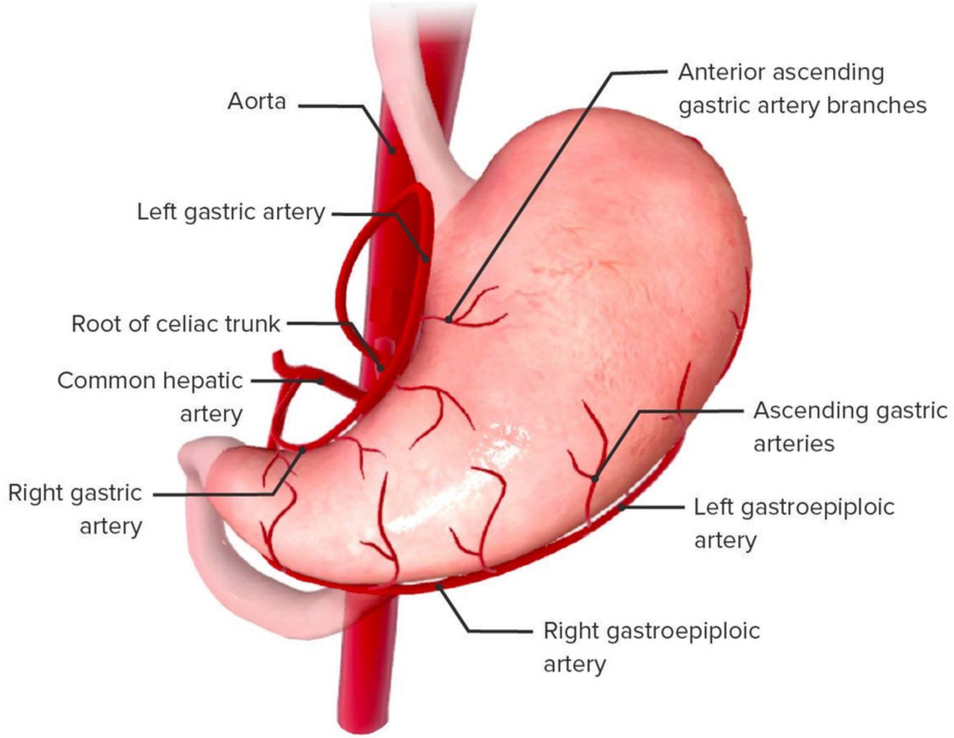


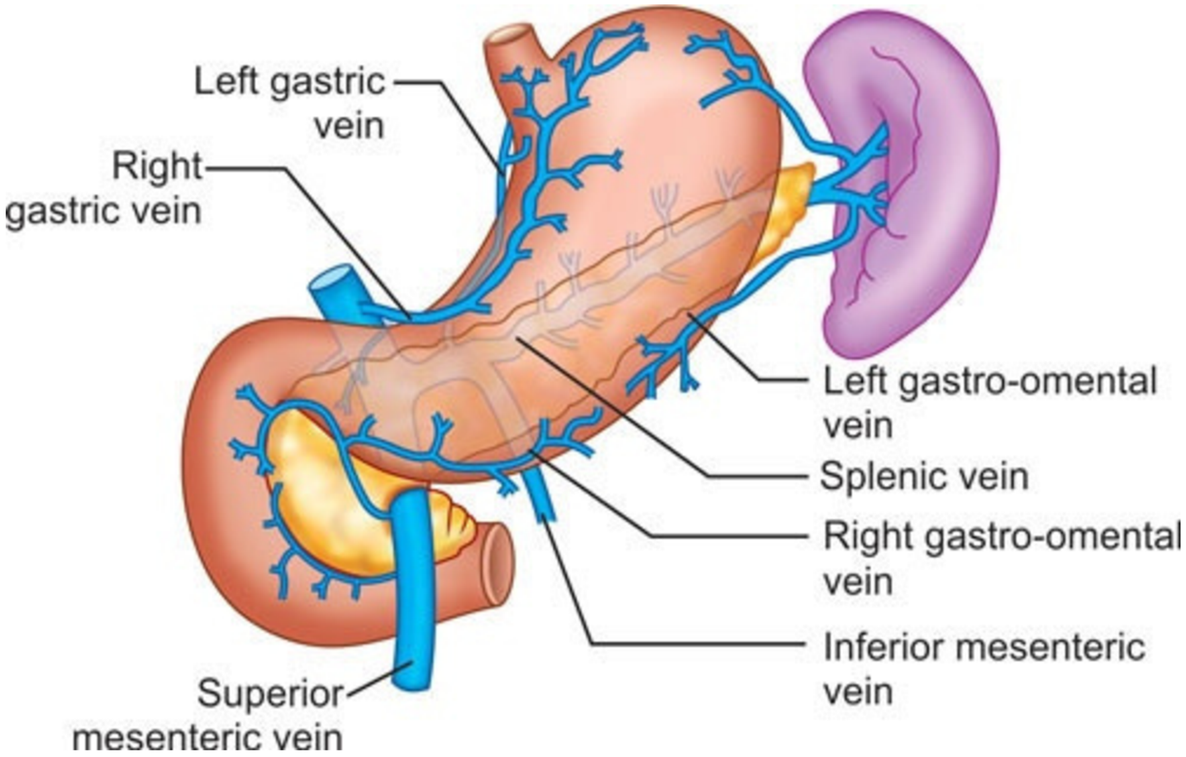


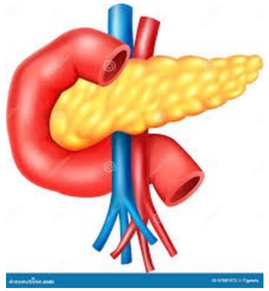
STOMACH blood supply:

arterial	Venous
along lesser curvature : RT gastric from hepatic proper from common hepatic artery from celiac trunk from abdominal aorta	rt and lt gastric veins to the portal vein
along greater curvature : RT gastroepiploic (omental) from gastro doudenal artery from hepatic artery from celiac trunk	Rt gastro omental vein to superior mesentric vein which unite with splenic to form the portal vein
Fundus : short gastric arteries from splenic through gastrosplenic ligament .	lt gastro omental vein and short gastric vein to splenic vein .

STOMACH blood supply:



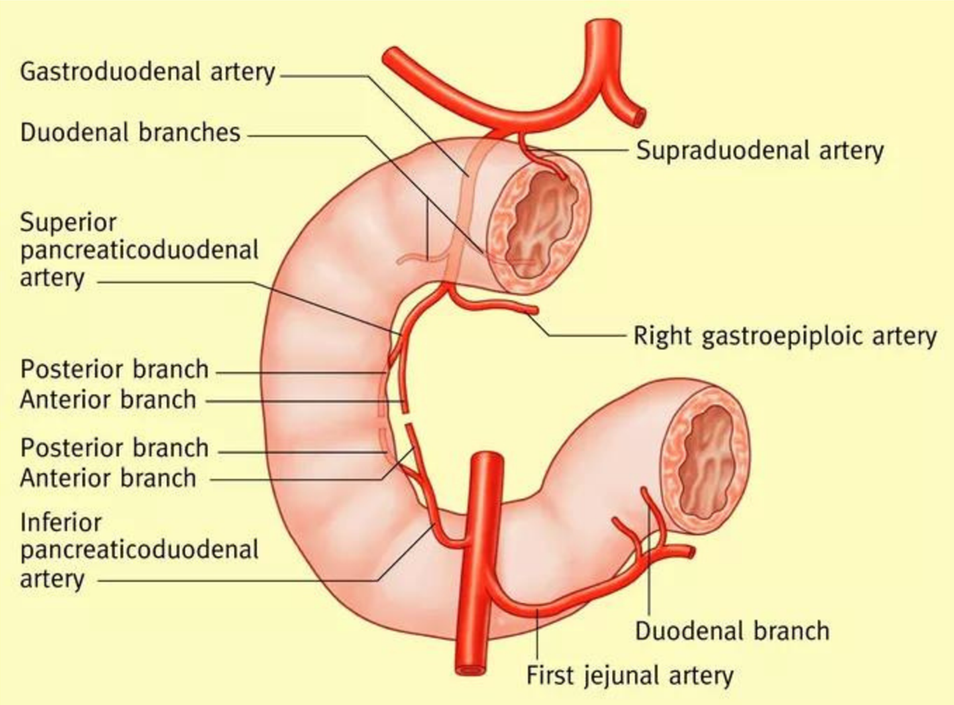


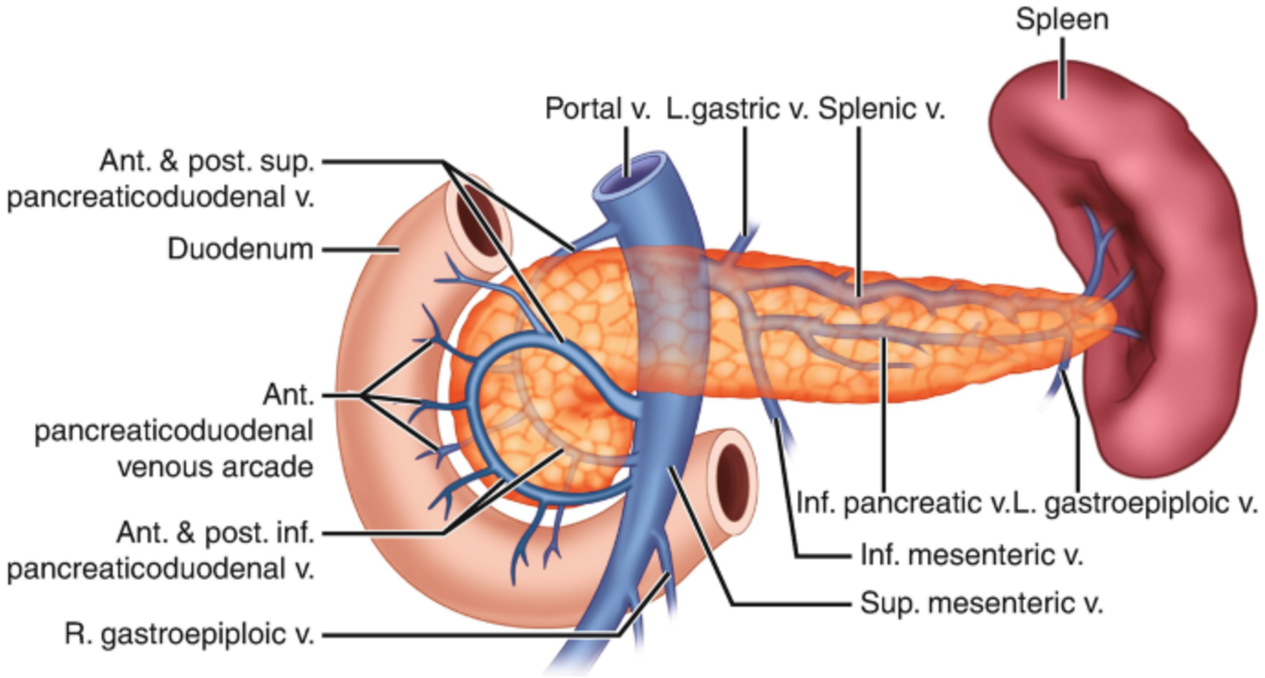


DUODENUM blood supply:

arterial	Venous
Superior pancreatico-doudenal artery from gastro-duodenal artery from common hepatic from celiac from aorta .	Pancreatodoudenal veins superior and inferior drains into superior messentric vein then to portal system.
Inferior pancreatico-doudenal artery from superior mesentric from the aorta	
supradoudenal artery from gastro-duodenal artery from hepatic artery .	

DUODENUM blood supply:



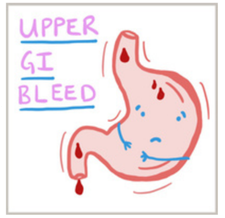


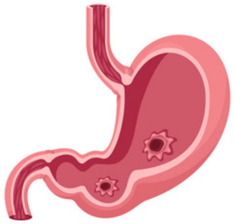
Epidemiology :

- UGIB vs LGIB = 4:1
- More in male than female
- The incidence increase with age
- The most common cause peptic ulcer (duodenal ulcer more common then the gastric ulcer)
- 80% are self limiting
- The mortality rate 5-10% for severe UGIB
- The patients who have recurrent bleeding within 48-72 hours have poor prognosis
- Anti platelet therapy has two fold increase in bleed
- Using NSAIDs increase the risk of bleeding(at least 2 fold)

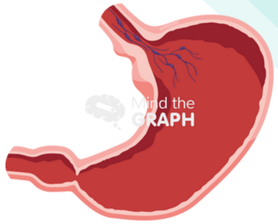
What is the difference between UGIB & LGIB ?

features	UGIB	LGIB
Site	Above Ligament of Treitz	Below Ligament of Treitz
presentation	Hemetemesis or Malena	Hematochezia
nasogastric aspiration	Blood	clear fluid
Bowel sounds	Hyperactive	Normal
BUN / creatinine ratio	increased	Normal

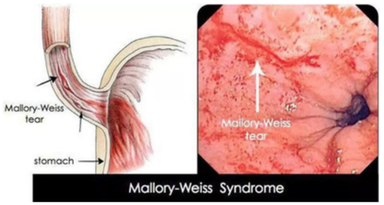


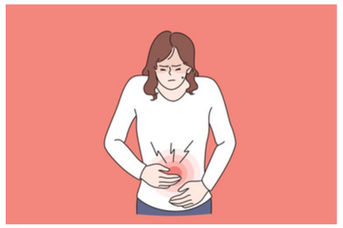


Causes :



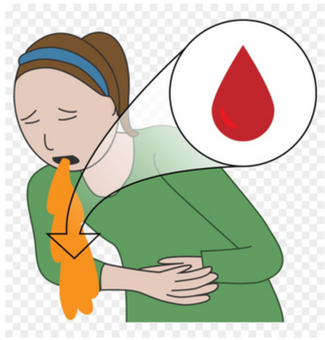
- ***Peptic ulcer disease (35-50%) most common***
- Gastroesophageal erosions **(10-20%)**
- Mallory-weiss tear **(5%)**
- Esophageal varices **(2-9%)**
- Tumors
- Vascular lesions **(5%)**
- Others





Possible Symptoms & signs of UGIB :

- Hematemesis
- Melena
- Hematochezia
- Syncope
- Dyspepsia
- Epigastric pain
- Heartburn
- Diffuse abdominal pain
- Dysphagia
- Weight loss
- Jaundice



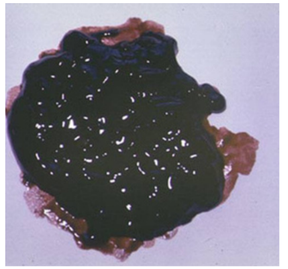
Common complication:

1. Hematemesis

- vomiting of blood ,could be: Digested blood in the stomach(coffee-ground emesis that indicate slower rate bleeding) or fresh/unaltered blood (gross blood and clots,indicates rapid bleeding).

2. Melena

- stool consisting of partially digested blood (black tar:, semi solid, shiny and has a distinctive odor, when its present it indicates that blood has been present in the GI tract for at least 14 h. The more proximal the bleeding site, the more likely melena will occur.



3. Hematochezia

- usually represents a lower GI source of bleeding, although an upper GI lesion may bleed so briskly that blood does not remain in the bowel long enough for melena to develop.

Presented by:
Dima khaled

Management :

- Patients with acute upper gastrointestinal (GI) bleeding commonly present with hematemesis (vomiting of blood or coffee-ground-like material) and/or melena (black, tarry stools), though patients with large-volume upper GI bleeding may also present with hematochezia (red or maroon blood with stool). The initial evaluation of patients with acute upper GI bleeding involves an assessment of hemodynamic stability and resuscitation if necessary. Diagnostic studies (usually endoscopy) follow, with the goals of diagnosis, and when possible, treatment of the specific disorder.

INITIAL EVALUATION :

- The initial evaluation of a patient with acute upper gastrointestinal bleeding includes a history, physical examination, and laboratory tests. The goal of the evaluation is to assess the severity of the bleeding, identify potential sources of the bleeding, and determine if there are conditions present that may affect subsequent management. The information gathered as part of the initial evaluation is used to guide decisions regarding triage, resuscitation, empiric medical therapy, and diagnostic testing.

Management :

How to mange ? (approach)

1.History taking :

patient profile: name, age, sex ,marital status,residency, occupation

History of presenting illness:

- Abdominal pain, Haematamesis, Haematochezia, Melena.

- Features of blood loss: shock, syncope, anemia

GI bleeding manifesti as:
- Overt bleeding
- Occult bleeding

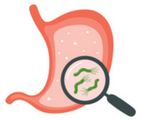
- History of epistaxis and hemoptysis to rule out the GI source of bleeding.

Management :

How to mange ? (approach)

Drug history: NSAIDS, Aspirin, corticosteroids, anticoagulants.

Past medical : previous episodes upper gastrointestinal bleeding, diabetes mellitus; coronary artery disease; chronic renal or liver disease, H pylori infection ; or chronic obstructive pulmonary disease.



Past surgeical: previous abdominal surgery

Family history

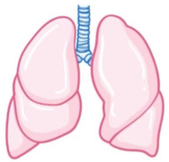
Social history : Smoking, Alcohol

2. check vitals:

to know if the paitent is → *Stable* / *unstable*

TEMPERATURE

PULSE



RESPIRATORY RATE

BLOOD PRESSURE

OXYGEN SATURATION

Management :

How to mange ? (approach)

3. Physical examination:

1.Genaral status of patient :

2.inspiction:

- Pale, Cyanosis
- Abdomenal Distension
- Lymph node swelling, visible peristalsis
- Feature of chronic liver disease
- Sign of dehydration (dry tongue , sunken eyes)

3. Palpation:

- Tenderness
- Abdominal mass

4. Percussion: Shifting dullness

5. Auscultation:

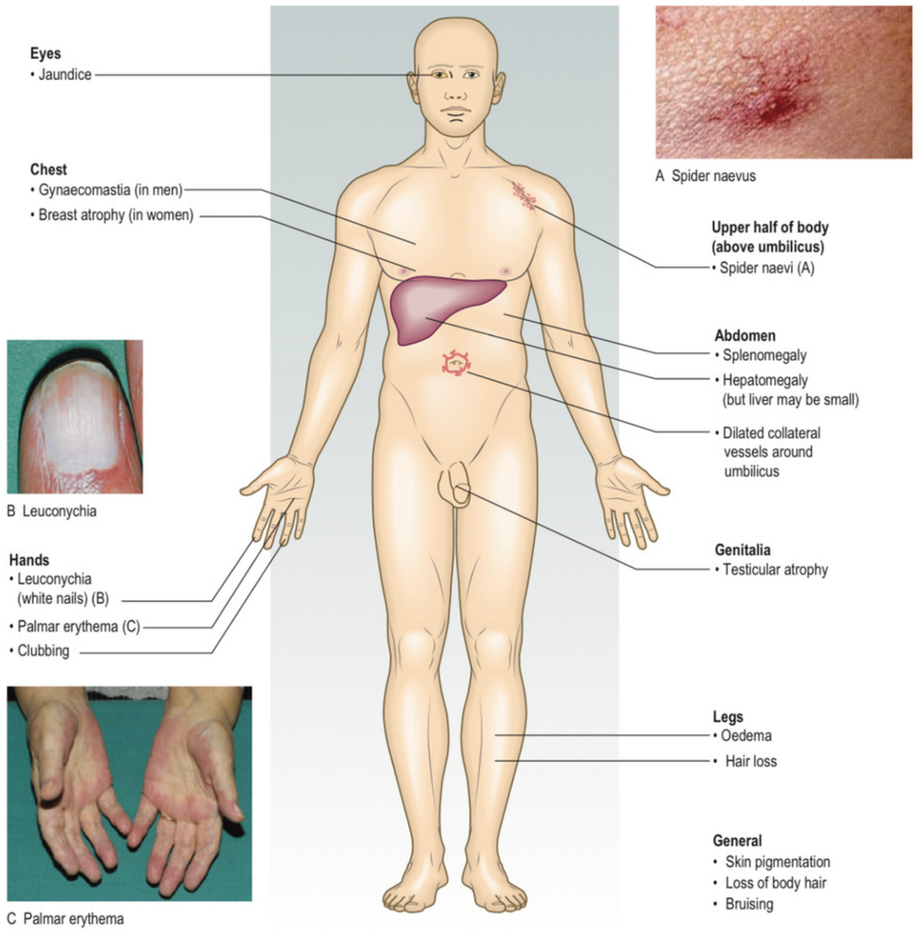
- Absent bowel sound
- Bruit

6. Digital rectal examination:

- Hemorrhoid
- Fresh blood fissures

Management :

Feature of chronic liver disease:

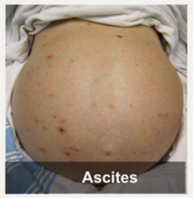


A Spider naevus

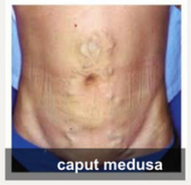
B Leuconychia

C Palmar erythema

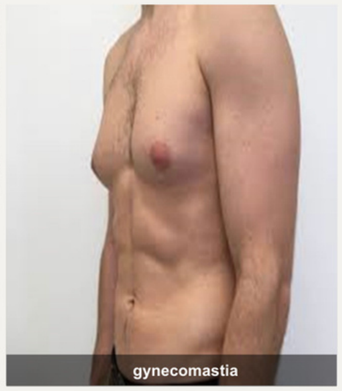
Fig. 6.9 Features of chronic liver disease.



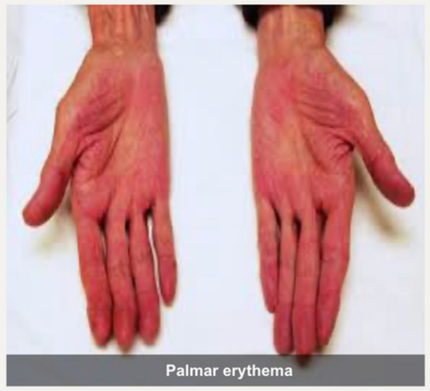
Ascites



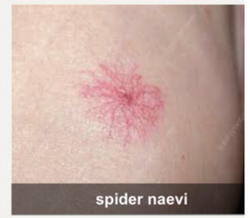
caput medusa



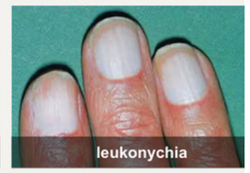
gynecomastia



Palmar erythema



spider naevi



leukonychia

Management :

How to mange ? (approach)

3. Physical examination:

6. Digital rectal examination: is very important to detect the presence of blood in the rectum.

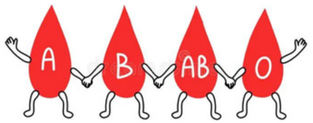
THIS DOES NOT MEAN THAT THE BLOOD IS COMING FROM THE RECTUM, but indicates that the patient IS BLEEDING. in UGIB, the blood is usually tarry and black with offensive smell, but brisk bleeding can result in detecting fresh blood.

Management :

How to mange ? (approach)

3. Investigation :

- Full blood count
- Hematocrit (PCV decrease only after 24 hours to 72 hours after bleeding) Coagulation profile
- Liver & Renal function test, Electrolytes
- Blood urea nitrogen ratio
- Blood grouping and cross matching
- Stool occult blood test(in chronic bleeding)
- ECG, Cardiac enzymes(if essential)



Management :

How to mange ? (approach)

4. Imaging:

- chest & abdomen X-ray
- CT & US
- Angiography

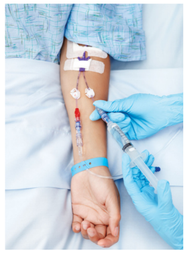
Management :

How to mange ? (approach)

INITIAL MANAGEMENT: -

The patient should be first resuscitated and then investigated urgently to determine the cause of the bleeding, this should follow the same principles of treating hemorrhage

1. ***2 wide bore venous cannulas are inserted***



For
- installing fluids
- drawing blood
  - For
    - CBC, coagulation profile, urea and electrolytes and cross- matching

- ***A central venous catheter is considered when there are signs of cardiovascular instability.***

Management :

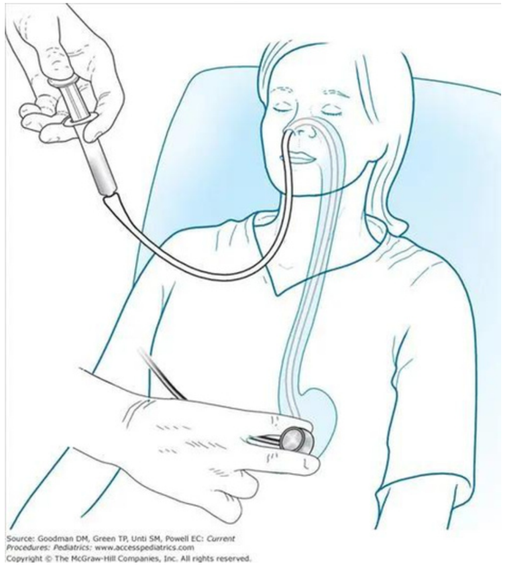
How to mange ? (approach)

3. INITIAL MANAGEMENT: -

2. Monitor adequacy of resuscitation

→ *Bladder catheterization for monitoring urine output*

3. Insertion of a nasogastric tube to confirm wither there is blood in the stomach (and thus help confirm UGIB).



Management :

How to mange ? (approach)

3. INITIAL MANAGEMENT: -

4. Risk stratify to guide for further management

ROCKALL SCORE:

↓

Assessing the risk of death and re-bleeding in patients with UGI hemorrhage

- Rockall's score>8=High risk of death
- Rockall's score<3=excellent prognosis

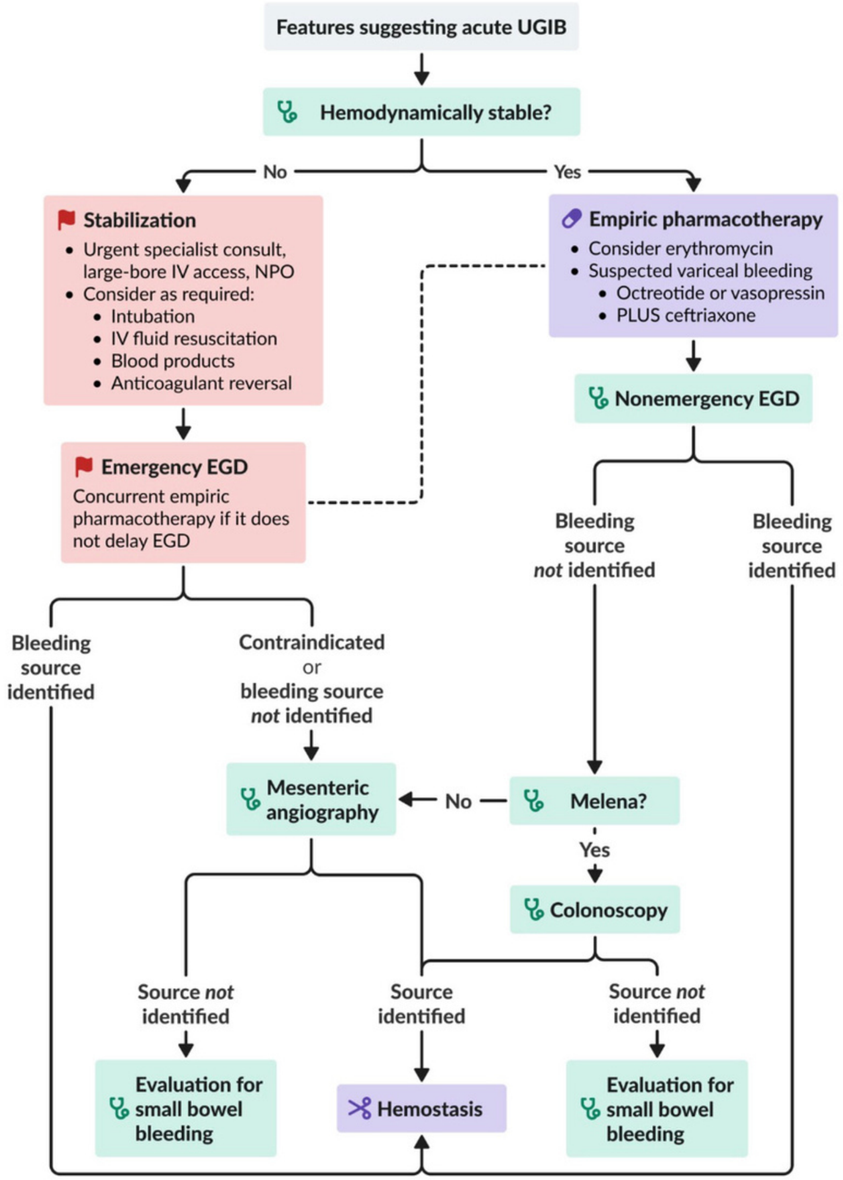
TABLE 67.5 The Rockall scoring system of bleeding severity.

	Score			
	0	1	2	3
Age	<60	60–79	>80	
Shock	Pulse <100 bpm Systolic BP >100 mmHg	Pulse >100 bpm Systolic BP <100 mmHg	Pulse >100 bpm Systolic BP <100 mmHg	
Comorbidities	None		Circulatory failure/coronary artery disease	Renal failure Liver failure Disseminated malignancy
Endoscopic signs of bleeding	None/dark spot		Blood/adherent clot/visible or spurting vessel	
Diagnosis	Mallory–Weiss syndrome/no pathology	All other diagnoses	Malignancy of the upper gastrointestinal tract	

BP, blood pressure; bpm, beats per minute.

Timothy Alexander Rockall, contemporary, Royal Surrey County Hospital, Guilford, Surrey, UK.

Management :


Features suggesting acute UGIB
Hemodynamically stable?
No
Yes
Stabilization
• Urgent specialist consult, large-bore IV access, NPO
• Consider as required:
• Intubation
• IV fluid resuscitation
• Blood products
• Anticoagulant reversal
Empiric pharmacotherapy
• Consider erythromycin
• Suspected variceal bleeding
• Octreotide or vasopressin
• PLUS ceftriaxone
Nonemergency EGD
Emergency EGD
Concurrent empiric pharmacotherapy if it does not delay EGD
Bleeding source not identified
Bleeding source identified
Bleeding source identified
Contraindicated or bleeding source not identified
Mesenteric angiography
No
Melena?
Yes
Colonoscopy
Source not identified
Source identified
Source not identified
Evaluation for small bowel bleeding
Hemostasis
Evaluation for small bowel bleeding

Management :

How to mange ? (approach)

What is your further management and treatment?!

1. ***Endoscopic hemostasis***

2. ***Interventional radiology (angiography)***
   - *embolization*
   - *vasopressin infusion*

3. ***surgery***

Management :

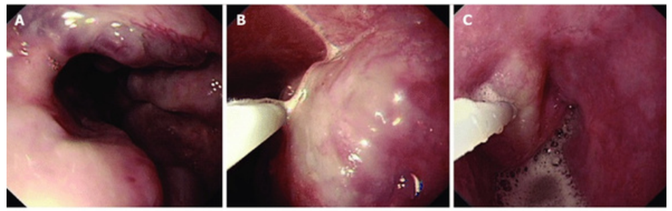
How to mange ? (approach)

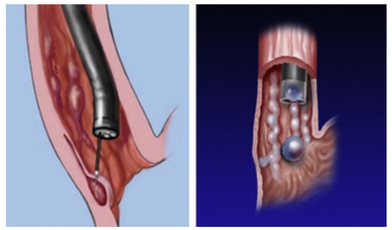
- **Endoscopy :**

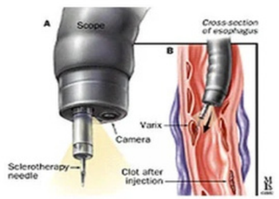
Endoscopy is the most accurate test to determine the etiology of upper Gl bleeding, and should be performed urgently in patients with hemodynamic instability. It has the marked advantage of providing therapeutic interventions after visualization of the lesion
The management is now considered for each diagnosis made by endoscopy.

- **Endoscopic management?**

1. ***sclerotherapy Injection***

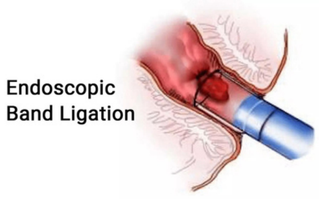


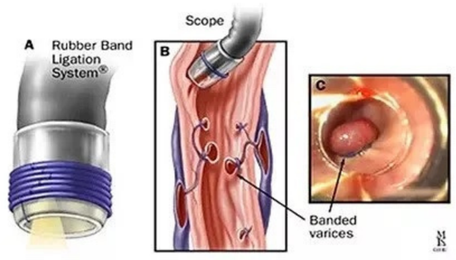




2. mechanical clipping

3. band ligation





Management :

How to mange ? (approach)

- **Indications of angiography:**
  - Unstable refractory for resuscitation
  - Negative endoscopy with ongoing bleeding

- **Indications for Surgery :**
  - Unstable refractory for resuscitation
  - Failure of endoscope after second attempt
  - Failure of endoscopic management to control bleeding

Presented by:Kawther
Al-habashneh

Peptic ulcer bleeding

The most common cause of upper Gl bleeding (35-50%)

Bleeding peptic ulcers are caused by erosion of an artery passing below the site of the ulcer

There are two types of peptic ulcer:

- *duodenal ulcers*
- *gastric ulcer*

Peptic ulcer bleeding

duodenal ulcers VS gastric ulcer

duodenal ulcers	*Gastric ulcers*
Erosion of: gastroduodenal artery	***Erosion of:*** left gastric artery
Site of ulcer: posteriorly	***Site of ulcer:*** most commen is lesser curvature
Most commen	***Less commen***
No possibilty of malignancy	***Possibility of malignancy must be considered***
No biopsy is needed	***The ulcer must be biopsied in all cases***

Peptic ulcer bleeding

Management of bleeding peptic ulcer:

Once the patient is stable (vital signs are normal)

↓

Endoscopy is commenced to identify the source of bleeding, even when the bleeding stops spontaneously

↓

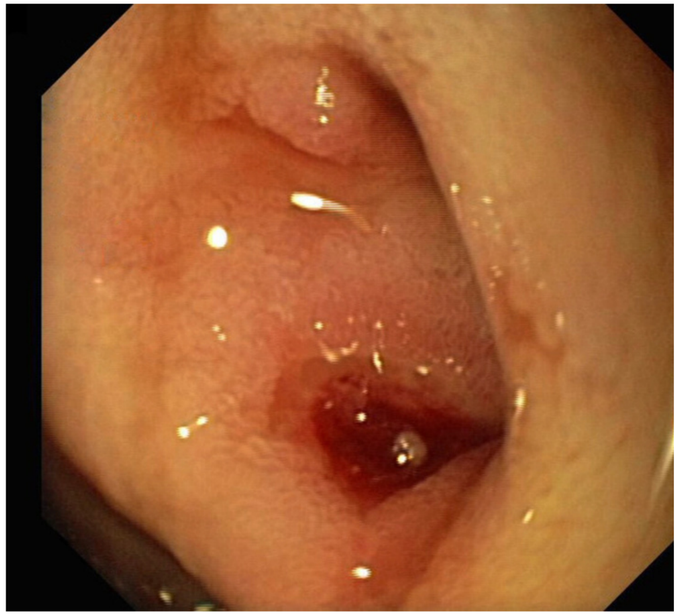
but if an active bleeding is detected: laser therapy, electro coagulation, sclerotherapy

Peptic ulcer bleeding

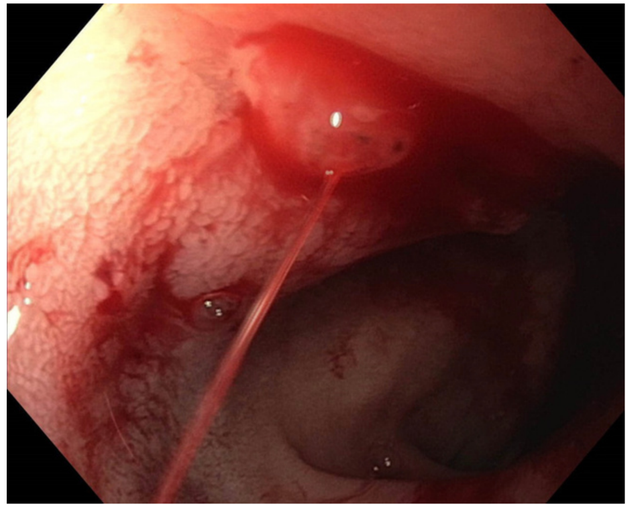
Forrest classification of bleeding peptic ulcers

stage		description	Risk of recurring hemorrhage
Active hemorrhage (Stage 1)	Ia	Active spurting	~90%
	Ib	Active oozing	~50%
Evidence of a recent hemorrhage (Stage II)	IIa	Nonbleeding ulcer with a visible vessel	~50%
	IIb	Ulcer with an adherent clot	~ 30%
	IIc	Flat pigmented spot (covered with hematin	~10%
Clean-based ulcer (Stage III)	III	Clean ulcer base (no active hemorrhage)	<5%

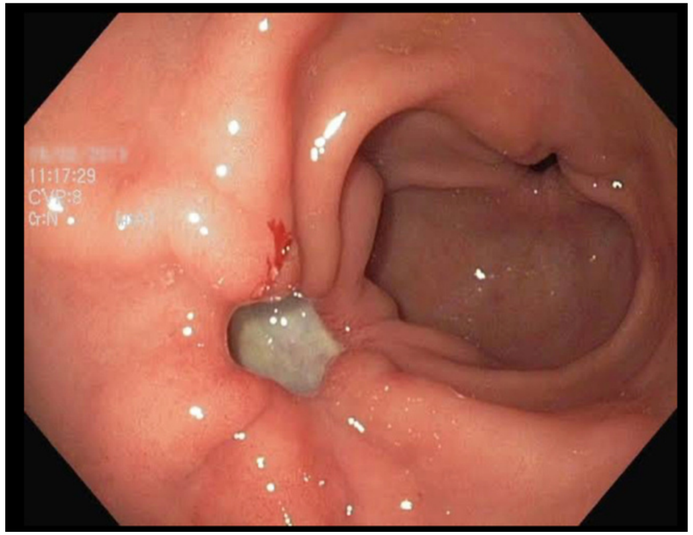
Peptic ulcer bleeding



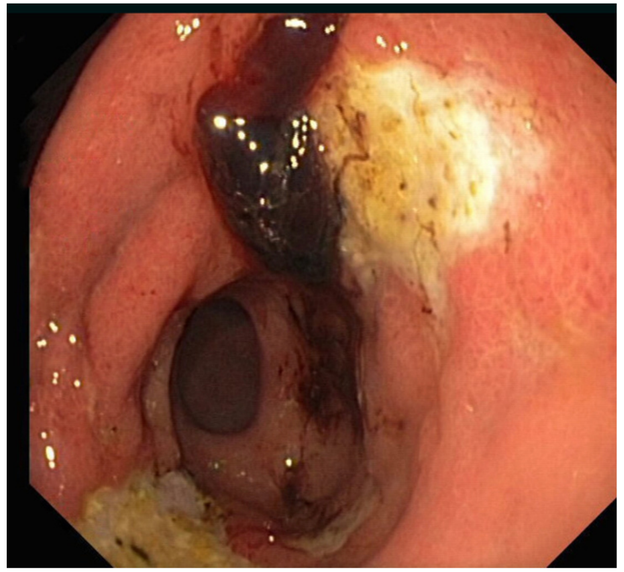
non-bleeding with visible vessel



spurting arterial Hemorrhage



flat ulcer with clean base



ulcer with an adherent clot

Peptic ulcer bleeding

Management of bleeding peptic ulcer:

Stage 3+ flat ulcer with dark base (of stage 2) therapeutic with PPI

Stage 1 + non bleeding ulcer with visible vessel /ulcer with an adherent clot (of stage 2) therapeutic by endoscope manovers, surgical in both of them we should use PPI

Randomized controlled trials document that high-dose constant infusion intravenous omeprazole (PPI; 80-mg bolus and 8-mg/h infusion), used to raise intra- gastric pH to between 6 and 7 and enhance clot stability, decreases further bleeding (but not mortality), even after the use of appropriate endoscopic therapy in patients with high-risk ulcers (active bleeding, non-bleeding visible vessel, and perhaps adherent clot)

Peptic ulcer bleeding

A bleeding duodenal ulcer may simply be under-runwith sutures, through a duodenotomy (opening of the anterior wall of the duodenum) to gain access to theulcer or pyloromyotomy.

When is surgery indicated in duodenal ulcer?	**The acronym "I HOP":** **Intractability** **Hemorrhage (massive or relentless) Obstruction (gastric outlet obstruction)** **Perforation**
How is a bleeding duodenal ulcer surgically corrected?	***Opening of the duodenum through the pylorus & Oversewing of the bleeding vessel***

Peptic ulcer bleeding

Important notes:

In gastric ulcer for young fit patients, it should be excised completely by taking a small wedge resection.

25% of patients with duodenal ulcers present with upper UGIB as their first manifestationand is the mostcommon cause of mortality, accounting for 40% of deaths.-80-85% of patient spontaneously stops bleeding.

Because ulcers recur (or rebleed) within the 2 years(most likely the first 3 days), thus treatment foreradication of H.pylori and avoiding NSAIDs is veryimportant to decrease the risk of recurrence .

If there is no other reason for hospitalization, such patients may be discharged on the first hospital day, following stabilization. Patients without clean-based ulcers (visible vessel, blood, clot) should usually remain in the hospital for 3 days, since most episodes of recurrent bleeding occur within 3 days .

Peptic ulcer bleeding

BLOOD SUPPLY OF GI:

1- celiac trunk

2- superior mesentric

3- inferior mesentric

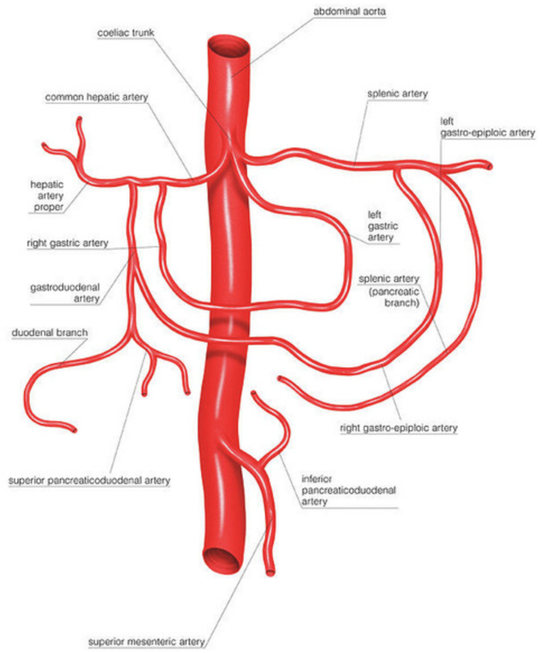
devided into:

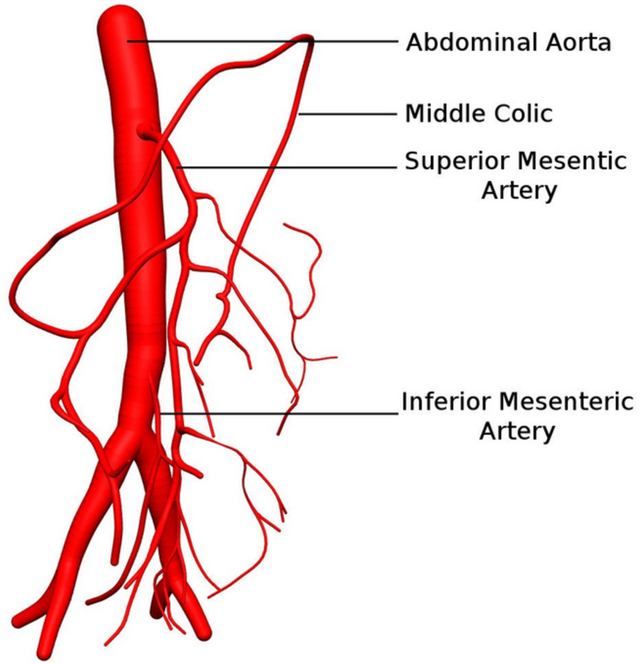
- ***Rt artery - proper hepatic:***

  a- common hepatic
  b- gastroduodenal artery

- ***Middle ⟶ splanic artery***

- ***Lt artery ⟶ left gastric artery***



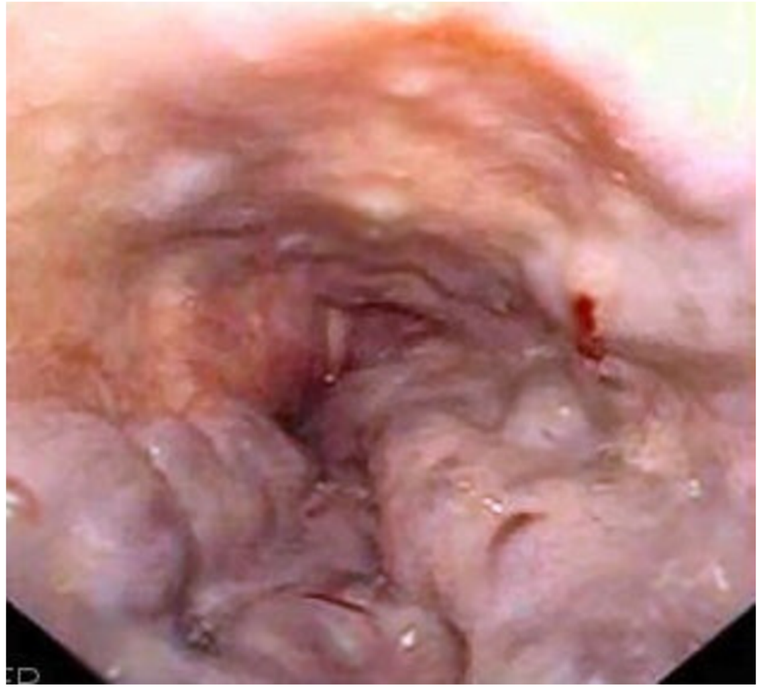


Esophageal varices

Presented by :Areen Mahadeen

What are esophageal varices?

- Varices are veins that are enlarged or swollen. The esophagus is the tube that connects the throat to the stomach. When enlarged veins occur on the lining of the esophagus, they are called esophageal varices.

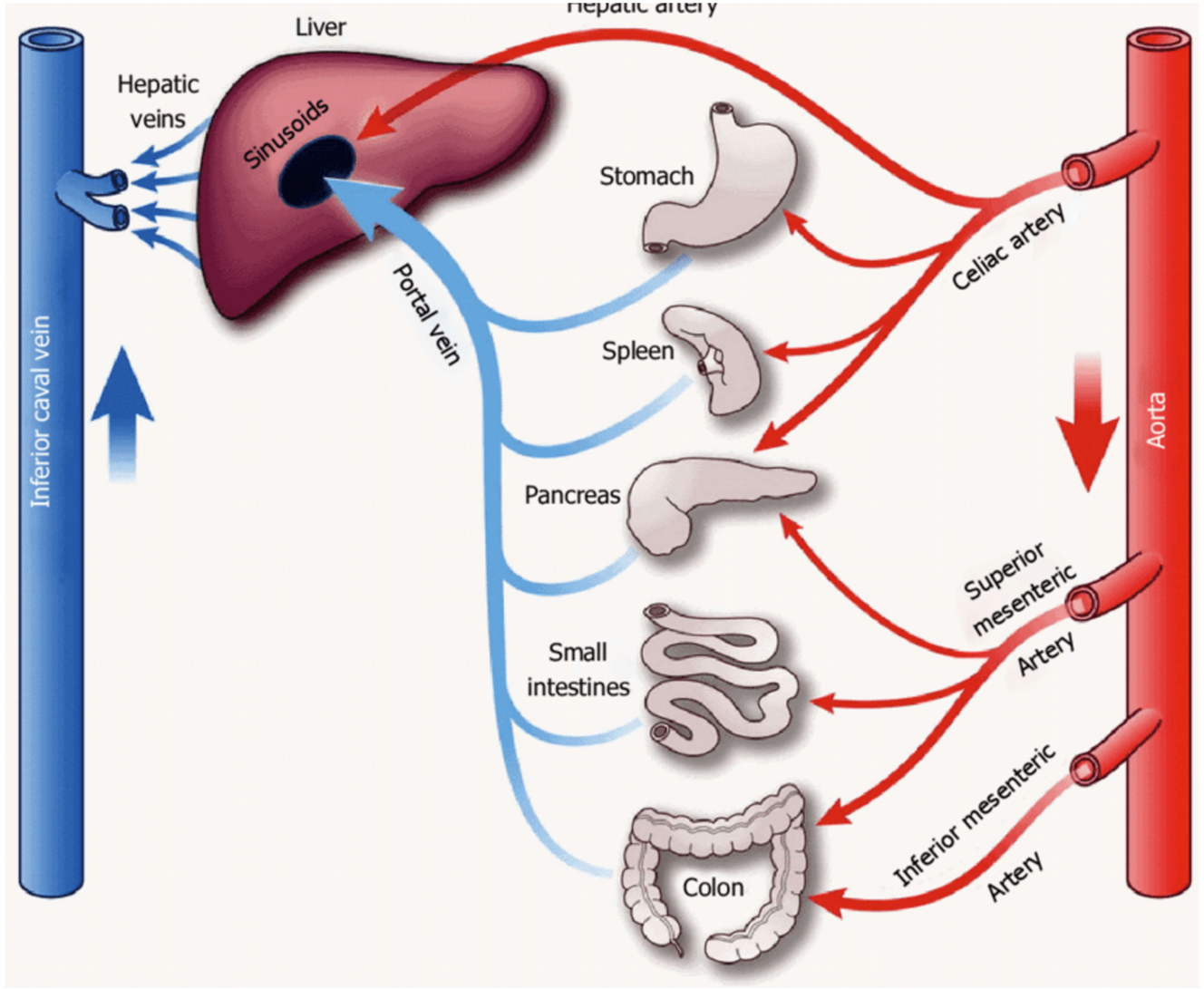


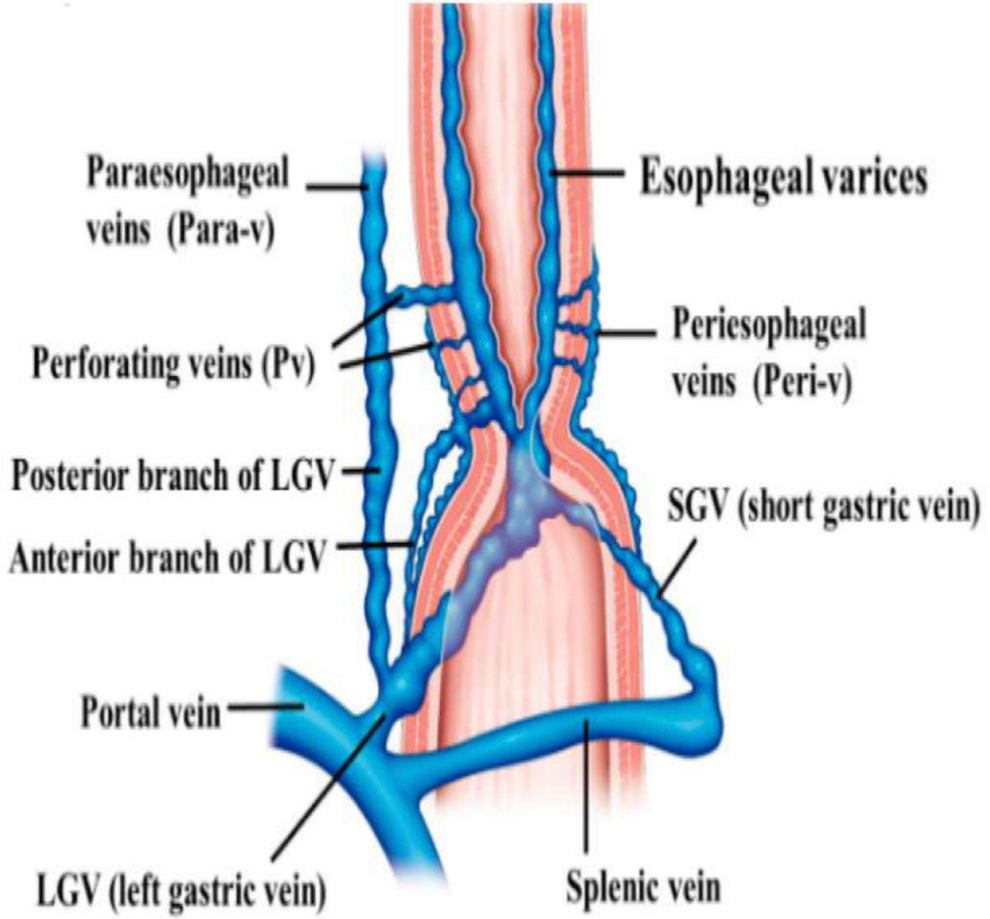
Esophageal varices

What causes esophageal varices?

- Esophageal varices usually occur in people who have liver disease. Blood flow through the liver slows in people who have liver disease. When this happens, the pressure in the portal vein goes up.
- High blood pressure in the portal vein (portal hypertension) pushes blood into surrounding blood vessels, including vessels in the esophagus. These blood vessels have thin walls and are close to the surface. The extra blood causes them to expand and swell.
- If the pressure caused by the extra blood gets too high, varices can break open and bleed. Bleeding is an emergency that requires urgent treatment. Uncontrolled bleeding can quickly lead to shock and death.
- Thrombosis (blood clot) in the portal vein or the splenic vein, which connects to the portal vein, can cause esophageal varices.

Esophageal varices





Esophageal varices

What causes esophageal varices?

Who is at risk for esophageal varices that break open and bleed?

Not everyone who develops esophageal varices will have bleeding. Factors that increase the risk for bleeding include:

High portal blood pressure:
The higher the portal pressure, the greater the risk of bleeding.

Large varices:
Risk of bleeding increases with size of varices.

Severe liver disease:
Advanced cirrhosis or liver failure increases the risk.

Ongoing alcohol consumption:
In patients with varices due to alcohol, continuing to drink increases the risk of bleeding.

Esophageal varices

What are the symptoms of esophageal varices?

- Most people do not know they have esophageal varices until the varices start to bleed. When bleeding is sudden and severe, the person vomits large amounts of blood. When bleeding is less severe, the person may swallow the blood, which can cause black, tarry stools.

- If bleeding is not controlled, the person may develop signs of shock, including pale, clammy skin, irregular breathing and loss of consciousness.

Esophageal varices

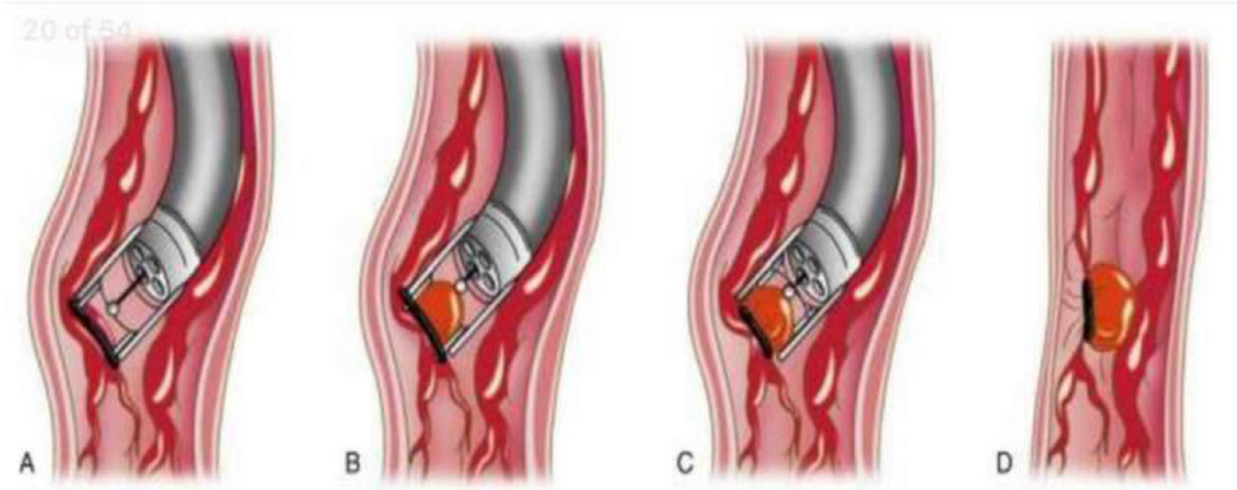
Mangment

1)Non bleeding varices:

prevention of first episode of variceal bleeding by :

1- pharmacothyerapy (NSBB)

2- EVL



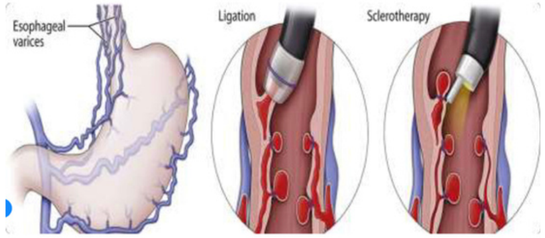
Esophageal varices

Managment

2)variceal bleeding: (active hemorrhage)

1. Approch to patient with UGIB
2. Management of varices (pharmacologic+endoscopic)

Endoscopic(immediately) :
1- variceal ligation (preferd)
2- sclerotherapy injection (when first choice is difficult)



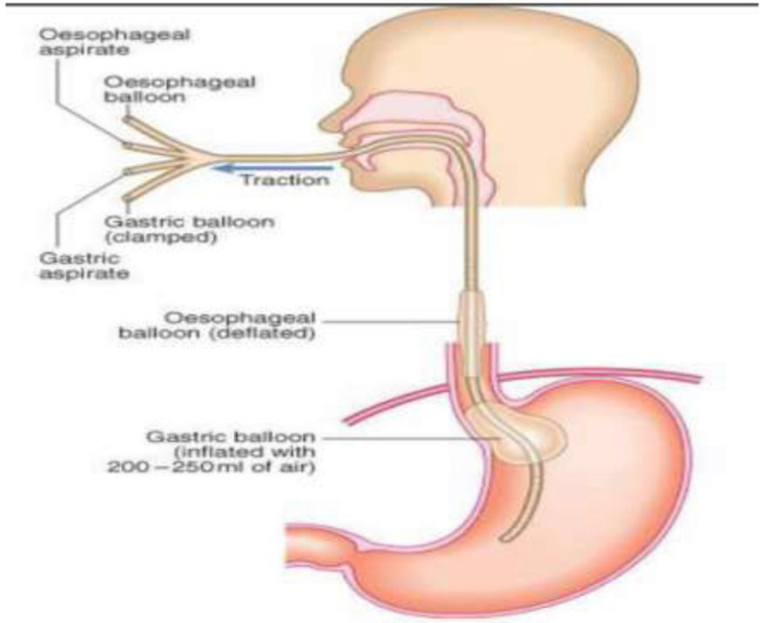
Pharmacological:
1- vasoactive agent to reduce splanchic blood flow and mortility . **Ex: -**Octreotide (a somatostatin analogue; 50-μg bolus and 50-μg/h intravenous infu-sion for 2 to 5 days)may help in the control of acute bleeding. • Agents such as somatostatin (250-500 μg/h) and terlipressin, available outside the United States, are also effective
2-antibiotic therapy(ceftriaxone)
3- prevetion of hepatic encephalopathy (lactulose)

Esophageal varices

Managment

Other interventional treatment

- ***Sengstaken-Blakemore tube(ballon tamponade)*** is used when there is suspicion of variceal hemorrhage with life threatening situation.
- It temporizing agent due to risk of pressure include necrosis.



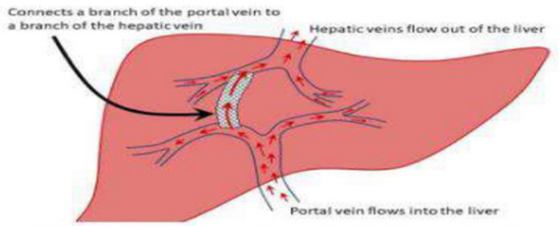
Esophageal varices

TIPS

- In this technique, a guidewire is passed from the jugular vein into the liver and an expandable metal shunt is forced over it into the liver substance to form a channel between the systemic and portal venous systems.

- It reduces the hepatic sinusoidal and portal vein pressure by creating a total shunt, but without the risks of general anaesthesia and majorsurgery. TIPS is most appropriate for patients with more severe liver disease and those inwhom transplant is anticipated. Patients with milder, well-compressive cirrhosis should probably undergo decompressive surgery (e.g.distal splenorenal shunt).

- ***Portal hypertension*** is also responsible for bleeding from gastric varices, varices in the small and large intestine, and portal hypertensive gastropathy and enterocolopathy



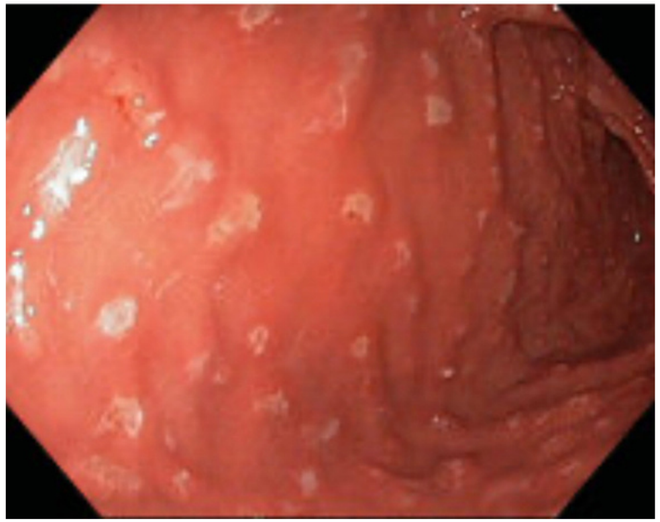
Transjugular Intrahepatic Portosystemic Shunt: TIPS

Erosive gastritis

Presented by: Jana Alsawadha

Definition :

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



Pathophysiology:

direct **mucosal** injury → edema → hyperemia → erosion → **ulceration**

Erosive gastritis

Causes:

Common causes :

- NSAIDs
- Alcohol
- Stress

Less common causes :

- Radiation
- Viral infection (eg, cytomegalovirus)
- Vascular injury
- Direct trauma (eg, nasogastric tubes)
- Crohn's disease

Erosive gastritis

Clinical presentation

- Patients with mild erosive gastritis ⟶ are often asymptomatic although some **complains of :**

1)dyspepsia 2)nausea 3)vomiting

- Often, the first sign is* hematemesis,* melena, or* blood in the nasogastric aspirate, usually within 2 to 5 days of the inciting event.

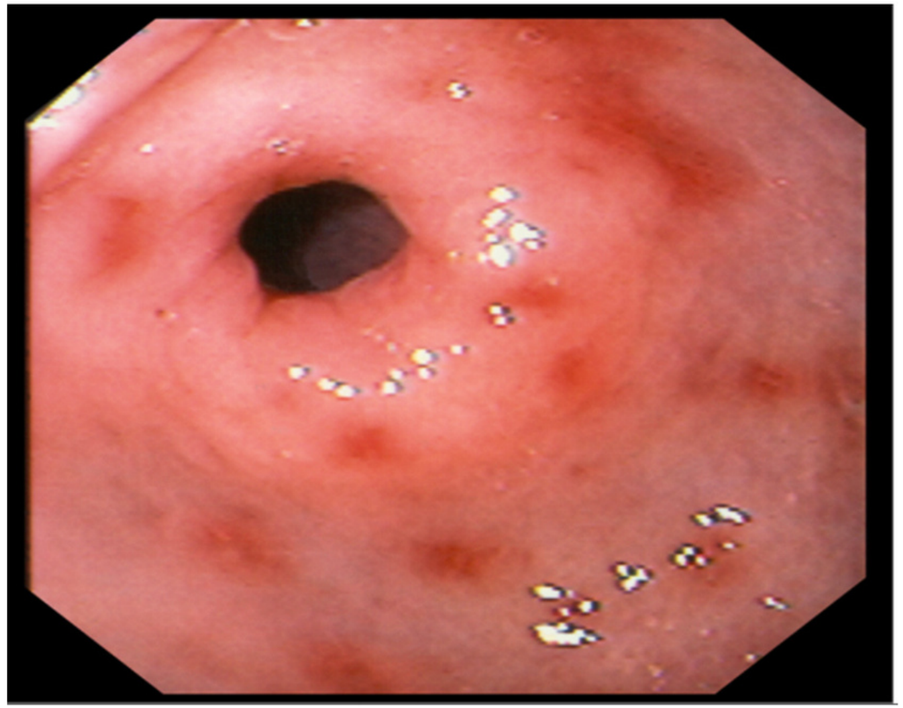
- Bleeding is usually **mild to moderate**, although it can be massive if deep ulceration is present, particularly in acute stress gastritis.

Erosive gastritis

Diagnoses

- diagnosed by:

*history of medications or any erosive agent and ***by endoscopy.**



This photo shows eroded and erythematous areas in the stomach lining resulting from prolonged nonsteroidal anti-inflammatory drug use.

Erosive gastritis

treatment

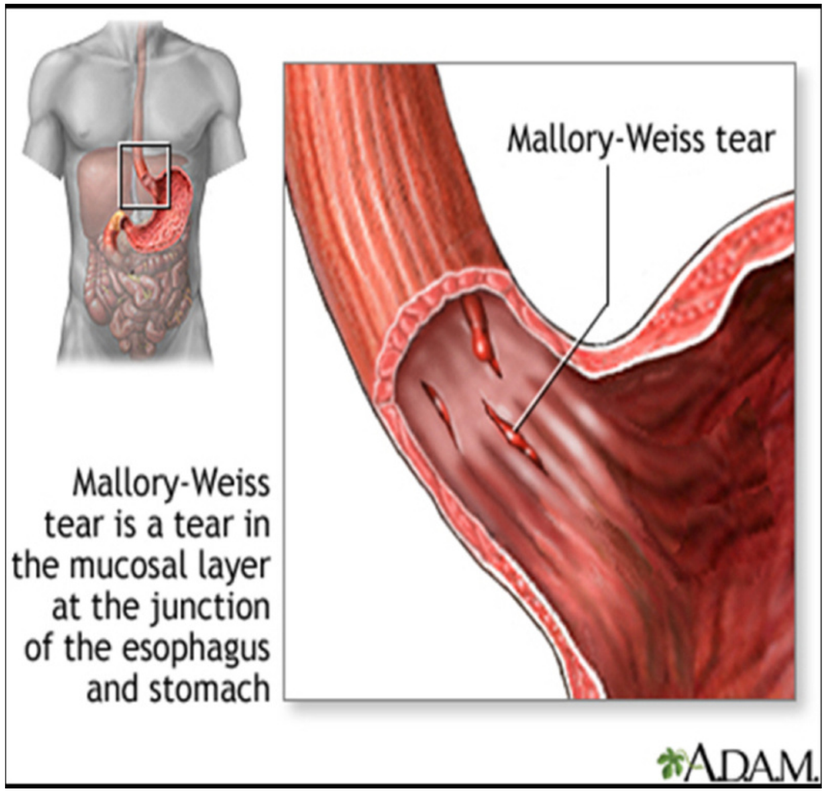
- **For bleeding:**
  - *Endoscopic hemostasis*
- **For acid suppression:**
  - *should be started if the patient isn't already receiving it*
  - *PPIs*
  - *H2 blockers (drug of choice)*
- **In severe gastritis:**
  - *bleeding is managed with IV fluids and blood transfusion as needed*

Mallory Weiss Tear

Definition :

acute upper gastrointestinal bleeding caused by mucous membrane lacerations (longitudinal tear) at the gastroesophageal junction, which is induced by repetitive and strenuous vomiting.

It makes up around 4% of UGIB



Mallory Weiss Tear

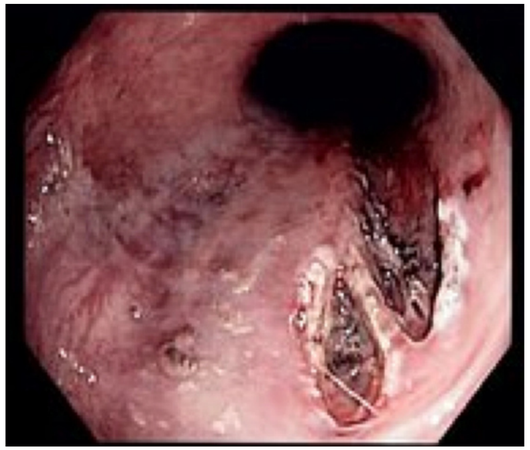
Causes:

- Mallory Weiss tears are caused by increased pressure in the abdomen. Scientists think that the abdominal pressure forces the contents of the stomach into the esophagus, leading to tears.

Causes of increased abdominal pressure include:

- Severe vomiting or retching.
- Heavy lifting or straining.
- Prolonged coughing.
- Trauma to the abdomen or chest.

Mallory Weiss Tear



diagnosis:

Ask about medical history, especially conditions that cause vomiting, retching, straining or coughing.

- **EGD(Esophagogastroduodenoscopy)**

Is the gold standard test and can rule out other differential diagnoses of upper GI bleeding.

- **Indications :**

Should be performed in all patients to confirm the diagnosis

- **Angiography**

- **Indications :**

Signs of active bleeding without successful location of the tear on EGD
**EGD unavailable*

Mallory Weiss Tear

Treatment

- *Bleeding from these tears stops spontaneously in 80 to 90% of patients(within 72 hours) and recurs in only 0 to 5%.*
- *endoscopic injection therapy is needed when the tear is actively bleeding and is usually sufficient*
- *angiographic therapy with (intra-arterial vasopressin)* ***or*** *surgical therapy (under running with sutures) are needed when the tear continues to bleed despite endoscopic therapy, but are rarely required.*

THANK YOU