

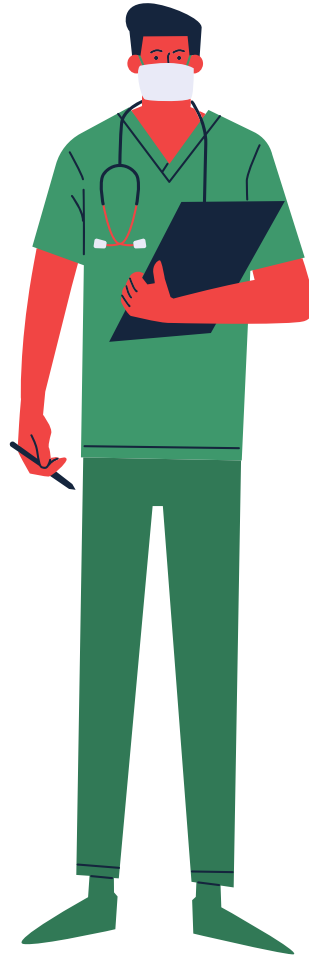
GIS



Sheet no.1

PBL

Problem-based learning



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Correction: Shahed Atiyat

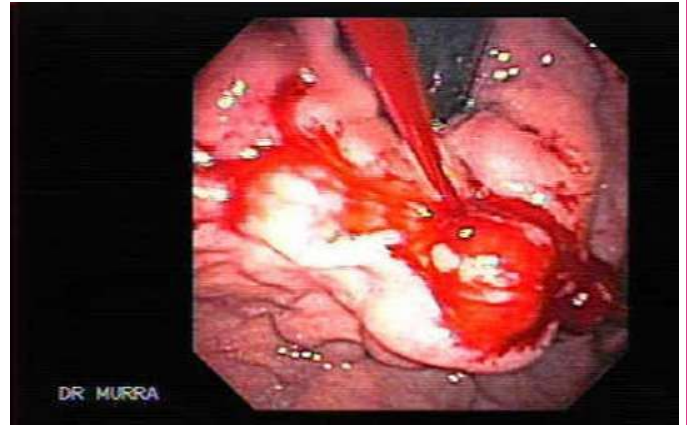
Doctor: Nadia Khamees

Upper GI bleeding:

(Underlined sentences are from the slides)

Anatomically it is defined as any bleeding above ligament of Treitz which is at the duodenojejunal junction. Any bleeding below it is considered lower GI bleeding (distal jejunal or ileal bleeding)

Note: The symptoms of both are quite similar.



There is no “negative rectal exam.” It is either empty rectum or brown or green stool but not negative

Signs and symptoms of upper GI bleeding: -

- ✓ Hematemesis: vomiting fresh blood.
- ✓ Melena: black tarry offensive shiny and loose stool. The color changes because of enzymatic oxidation of the blood and the doctor diagnoses it by a rectal exam.
 - Iron therapy makes the stool black, but it is hard not loose.
- ✓ coffee ground vomiting.
- ✓ Fresh rectal bleeding.

Massive upper GI bleeding, fresh rectal bleeding means that the patient is most likely hemodynamically unstable which means that he is in shock.

 - Features of hemodynamic instability:
 - 1- Postural tachycardia -> increased heart rate by 15 heartbeats when standing up. Some drugs mask this tachycardia such as beta blockers, so you should take proper history.
 - 2- Postural hypotension: drop in blood pressure (20 systolic or 10 diastolic).
 - 3- Frank hypotension. The doctor just mentioned it
- ✓ Dizziness: due to hypotension. Palpitations if the patient has tachycardia.
- ✓ Abdominal pain and symptoms of Peptic ulcer disease
- ✓ History of NSAID's use -which is related to peptic ulcers-
- ✓ Pallor: due to anemia. شحوب
- ✓ Hypotension, which was mentioned previously.
- ✓ Orthostasis: postural hypotension.

- ✓ Jaundice and other stigmata of chronic liver disease, related if the patient has esophageal varices.

Causes of upper GI bleeding: -

- Peptic ulcer disease (The most common).
 - Esophageal varices which happen in patients with cirrhosis and portal hypertension
 - Mallory Weiss tear: multiple forceful attacks of vomiting (Will be explained in a bit).
 - Gastric ulcers.
 - Duodenal ulcers.
- Both are peptic ulcers
- Other **rare** etiologies:
 - Neoplasms: gastric tumor, lower esophageal tumor → patients have weight loss + family history of GI malignancies+ loss of appetite.
 - Arteriovenous malformation (AVM) like a spider web with a center, surrounded by capillaries in stomach, duodenum or colon.
 - Ectasia: *extra: dilation or distention of a tubular structure.
 - Dieulafoy lesion: submucosal artery which makes an arterial bleeding and significant drop in hemoglobin, then it buries itself. In endoscopy the bleeding appears but the source disappears, and we should search for it.
 - Stoma ulcers
 - Esophageal ulcers
 - Deodenitis
 - Hemobilia
 - Aorto- enteric fistulas

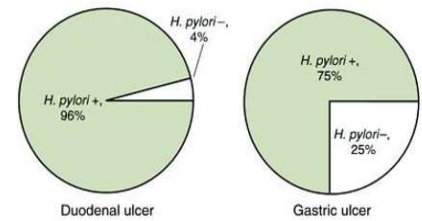
Peptic ulcer disease

- Defect in the GI mucosa extending through the muscularis mucosa.
- Decreasing incidence.
- Caused by imbalance between the aggressive and defensive factors.
- The most common causes of it: * Helicobacter Pylori *NSAID's.



*Acid hypersecretory state.

*Antral G cell hyperplasia.



- ❖ H. Pylori is very common, more than 80% of Jordanians have it, transmitted feco-orally, it doesn't cause acute gastroenteritis. It stays in the body for a long time then it gets triggered by a pill of NSAID's for example. Some patients are symptomatic others not. The infection is related to the social economic status. Ulcers occur in the antrum.
- ❖ NSAID's are the enemies of GI. So many side effects including peptic ulcers.
- ❖ Duodenal ulcer: 96% of cases are **H.Pylori +ve.** *Test for H. Pylori then treat*
: 4% of cases are **H.Pylori -ve.**



A defect in healthy mucosa with surrounding inflammatory changes around the ulcer

- ❖ Gastric ulcer: 75% of cases are caused by H.Pylori.
:25% of cases are not.

We must take a biopsy from gastric ulcers to rule out malignancy.



a defect in the normal mucosa. we call this a **clean-based ulcer.** Risk of rebleeding is low

Mallory-Weiss tear

Laceration around the GE junction.

Classical presentation as bleeding after episode of forceful vomiting (only 50%)

Self-limiting.

Multiple forceful vomiting attacks, after vomiting they have a tear in the lower esophagus then it bleeds. The blood appears after multiple vomits.

Some patients are more prone to it (who vomits a lot)

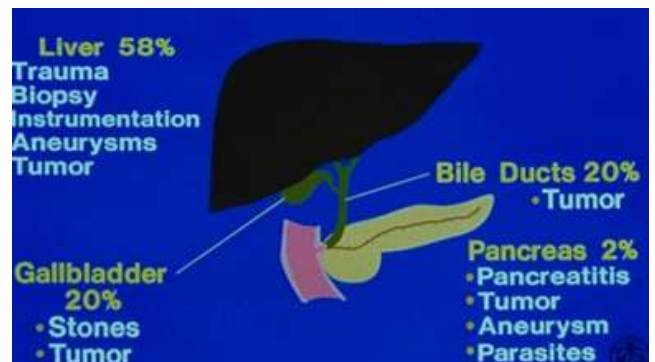
Like: Chemotherapy patients, pregnant women in first trimester who have hyperemesis gravidarum, alcoholics. It relieves spontaneously with minimal bleeding.



Hemobilia

Bleeding through ampulla of Vater and biliary tree.

Etiology: having previous procedures like ERCP (endoscopic retrograde cholangiopancreatography) used to assess the biliary tree or malignancy.



This is the ampulla of Vater, and the hemophilia is coming out of it (Black blood)



Stress ulcers

- Not our stress 😊
- Happens in critically ill patients like ICU, burns, head trauma patients.
- In extensive burn patients it's called "curling ulcer".
- In head injury patients it's called "Cushing ulcer".
- Caused by Vagal hyperstimulation and vascular hypoperfusion.
- Body and fundus more affected
- Multiple
- Prophylaxis is indicated in critically ill ICU patients, give them proton pump inhibitors especially if the patient will be given anticoagulants and NSAID's.



Adverse prognostic factors:

If the patient has adverse prognostic factors this means that he will have a poor course (prognosis).

🔑 Diagnosis

Esophageal varices → mortality rate reaches 30%.

Malignancy → bleeding from a gastric tumor for example.

🔑 Older age

🔑 Severe initial bleeding. The bleeding initially made a significant drop in hemoglobin → less than baseline.

🔑 Recurrence during hospitalization.

🔑 Coincidental diseases (comorbidities like DM, ischemic heart disease, hypertension).

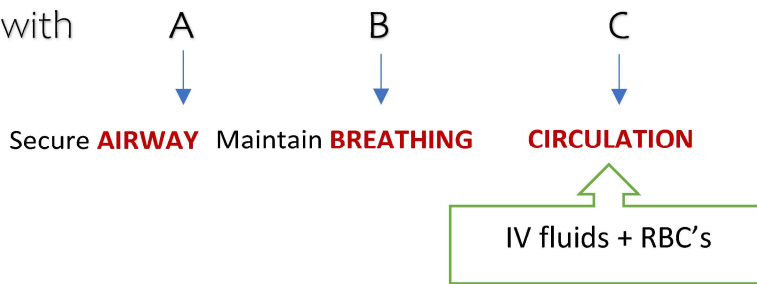
🔑 Endoscopic stigmata of recent bleeding. Clean based ulcer → no recurrence

If the vessel is spurting or a visible vessel in the ulcer carries risk for re-bleeding.

🔑 Need for emergency surgery.

Now we'll talk about **management** of upper GI bleeding

Start with



Esophageal Varices

*Dilated tortuous veins of the lower and mid esophagus.

*These veins when they dilate, they can rupture.

*Due to portal hypertension.

*30% mortality rate after the first episode.

*60% Rebleeding rate.

❖ In esophageal varices the goal is to maintain 7 to 8 mg/dL hemoglobin level, not more; to prevent increasing systemic pressure which would lead to increase in portal pressure then to another bleeding.

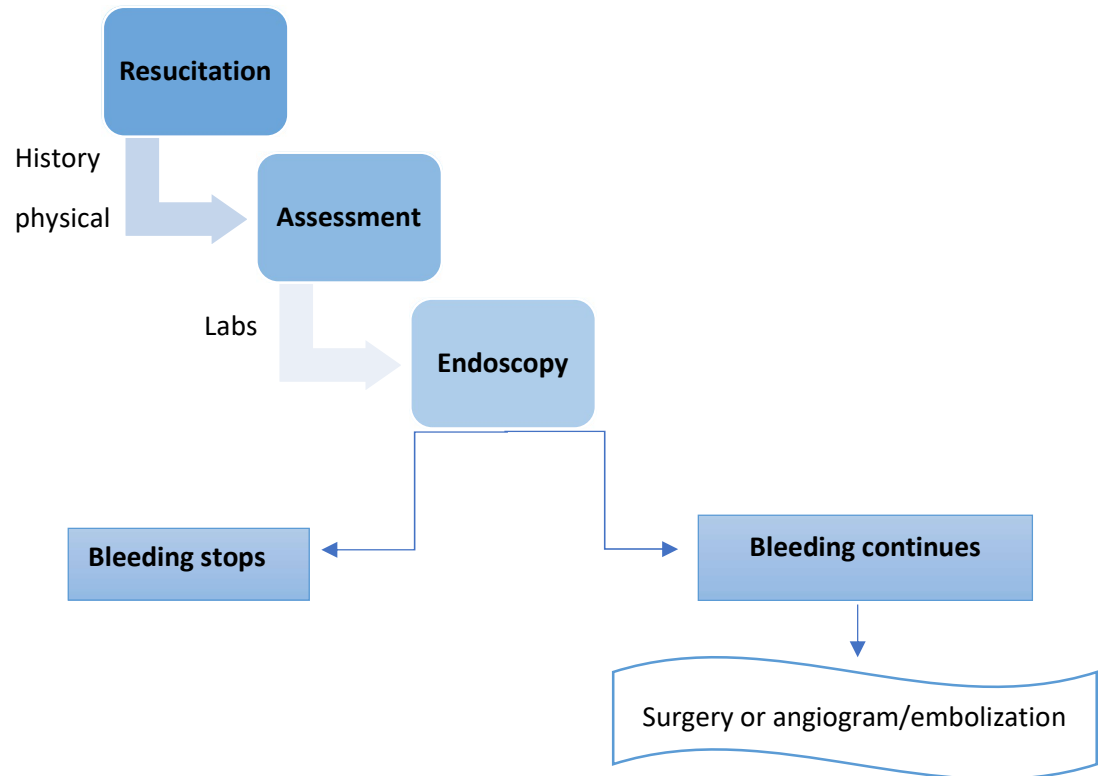
❖ If the patient has thrombocytopenia and portal hypertension and liver cirrhosis

❖ Non-variceal bleeding patients → proton pump inhibitors infusion.

❖ Variceal bleeding patients → Octreotides = Somatostatin analog

❖ Most have **rectal exam** and endoscopy.

❖ So, to summarize:



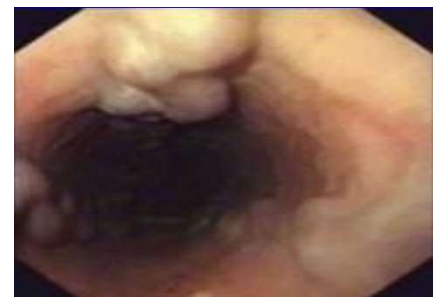
* Variceal bleeding patients has stigmata of recent bleeding.

* We give them Octreotides or vassopressin.



* Non-variceal bleeding patients does not have stigmata of recent bleeding.

* we give them protein pump inhibitors



*Spurting vessels

*Clean based

*Carries slight risk of rebleeding



UPPER GI BLEEDING

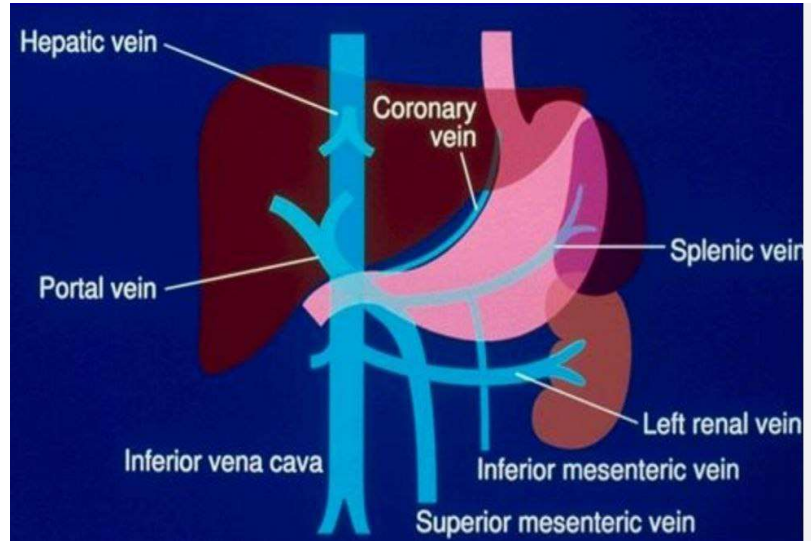
Esophageal Varices: Predictors of severity

- High Hepatic Gradient
- >12 mmHg

- Degree of Cirrhosis
- (Child Classification)

- Size of Varices
- Larger > smaller

- Endoscopic Appearance
- Cherry red spots
- Red wale



UPPER GI BLEEDING

Esophageal Varices: Management

Assessment/ Resuscitation

Pharmacological Therapy

- Somatostatin or Octreotide
 - Vassopressin

Endoscopy for diagnosis and therapy

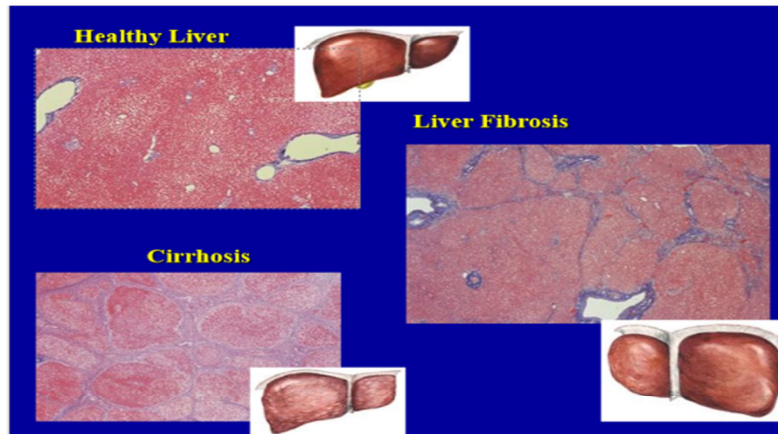
- Variceal Banding

Balloon Temponade

The first half is done! 😊

Let's go to the second ^_^

Cirrhosis and Portal hypertension



Stigmata(identifying marks) of cirrhosis:

☞ **Jaundice:** accumulation of bilirubin (hyperbilirubinemia) in the blood stream causing yellowish discoloration of plasma and heavily perfused tissues.

High bilirubin level test the true function of the liver. Transaminases -AST+ALT- elevation don't reflect the true function of the lever. **True synthetic function** of the liver tests: bilirubin, low albumin, prolonged prothrombin time, cholesterol, and glucose. Not only for cirrhosis, but patient with biliary stones could also have hyperbilirubinemia.



☞ **Spider angiomas:** Small, centrally raised bumps (papules) caused by a dilated arteriole (small artery). A network of dilated capillaries (tiny blood vessels) radiates from the arteriole. Pressing on the lesion causes the redness to disappear briefly, and there is a rapid return of redness once the pressure is lifted. Seen on anterior chest due to hyperestrogenemia. In females >5 we must suspect cirrhosis and portal hypertension.

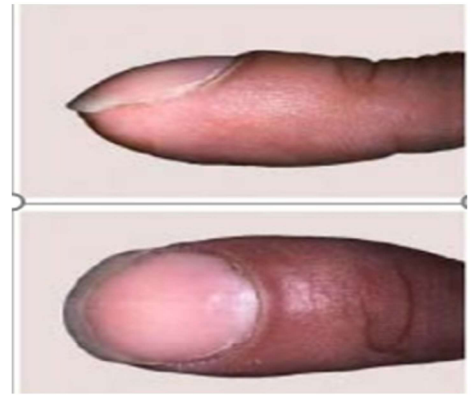


🔗 **Finger clubbing:** a condition where there is enlargement of the terminal end of the digit over the distal phalanx. It is usually symmetrical and affects the fingers

Loss of nail bed -nail angle, initially lost then the nail curvature increases

Drumstick appearance.

Can be seen in other diseases like congenital heart disease, inflammatory bowel disease, TB, sarcoidosis, celiac disease, and lung cancers.



🔗 **Gynecomastia:** breast development in men, and breast atrophy in women-the opposite-



🔗 **Dupuytren's Contractures:** contractures in hand joints

Seen in cirrhosis and Diabetes Mellitus.



Complications of cirrhosis:

*Hepatic encephalopathy → غيوبية الكبد

toxins accumulation: ammonia goes to brain

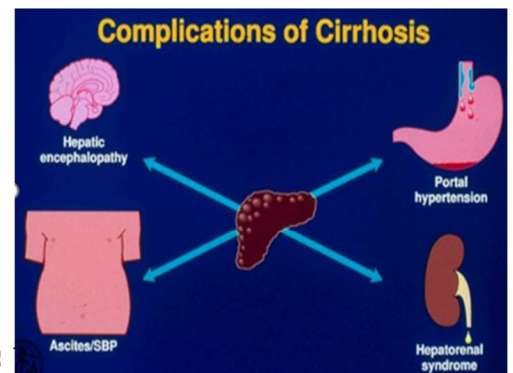
*Ascites +/- spontaneous bacterial peritonitis (SBP)

*Portal hypertension pts have translocation in GI bacteria peritoneum. Most common GI flora bacteria is E.coli which causes SBP with klebsiella.

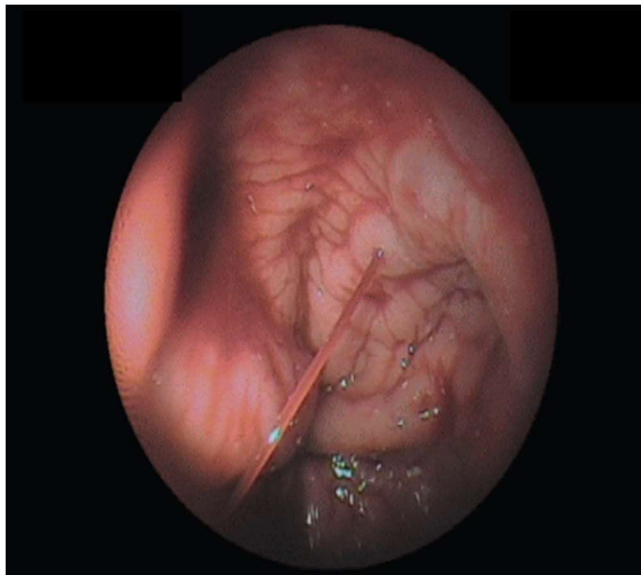
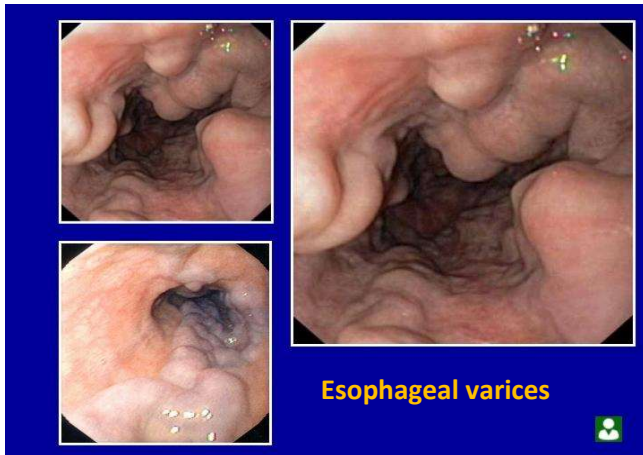
Spontaneous means it's not iatrogenic or by secondary causes

*Hepatorenal syndrome

*Varices



- ❖ **Caput Medusae:** Distended and engorged umbilical veins which are seen radiating from the umbilicus across the abdomen to join systemic veins.



Spurting vessel from a varix

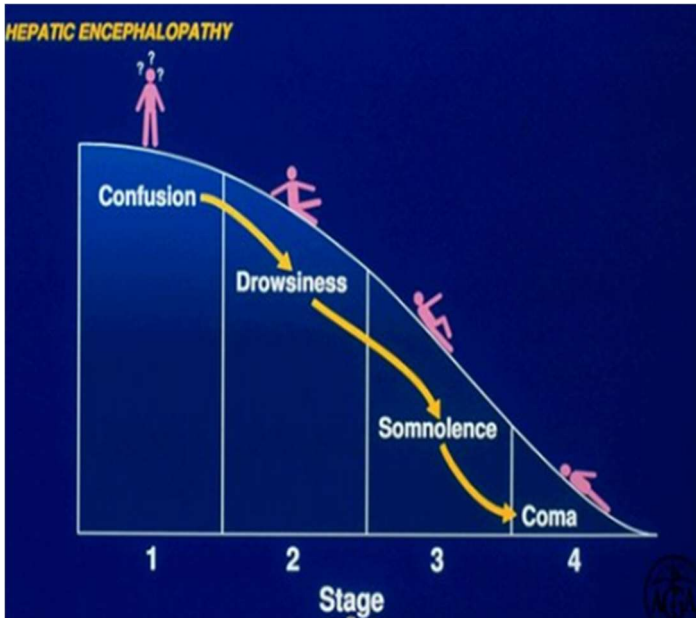


This is a ligated vessel
Treatment of varices: band ligation



Massive ascites with umbilical hernia, if the bowel enters the hernia, it becomes strangulated that's when surgeons operate to prevent gangrene and ischemia

❖ **Astraxia:** Flapping tremors, quick arrhythmic movement in background tonic muscle contraction
Happens in failures: respiratory, liver, heart.
Tell the patient to extend his elbow. In astraxia it flaps.



The stages of hepatic encephalopathy according to West Haven criteria on hepatic encephalopathy

1. it starts from some confusion and reversal of the sleep/wake cycle.
2. Drowsiness; the patient still arousable in this stage.
3. Somnolence; the patient is sleepy all the time.
4. Finally, complete coma

Good luck “)