Clinical/PBL Lecture #1

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①Upper GI Bleeding

Anatomically, upper GI bleeding is defined as bleeding where the source is above the ligament of Treitz. Clinically, however, bleeding below the ligament of Treitz can sometimes manifest as upper GI bleeding; so this definition doesn't always apply in real life.

Signs and symptoms of upper GI bleeding

- Hematemesis (vomiting fresh blood)
- Melena
- Dizziness
- Abd. Pain and symptoms of Peptic ulcer disease
- Hx of NSAID's use
- Pallor
- Hypotension
- Orthostasis
- Jaundice and other stigmatas of chronic liver diseases
- Coffee-ground vomiting

•**Melena**: stool is **black, tarry, shiny, loose, sticky and offensive in smell.**

Not all black stool is melena, therefore it's very important to know its characteristics in order to distinguish it from other conditions:

Taking iron supplements can also cause black stool, but unlike melena it's hard and matte (not shiny), and the patient usually suffers from constipation

It is mandatory for doctors to perform a digital rectal exam (DRE) whenever a patient comes in with symptoms of upper GI bleeding; you need to see stool with the features of melena in order to confirm

*Many patients with upper bleeding present with symptoms similar to anaemia:

-Dizziness

-Pallor

-Easily fatigued

-Palpitations due to tachycardia

-Chest pain

-Exertional dyspnea (shortness of breath during exercise or heavy movement)

Sometimes there's massive upper bleeding, which can lead to fresh red blood in the stool; it's usually accompanied with orthostatic hypotension (blood pressure decreases when the patient stands up) and tachycardia. In these cases, patients tend to have a low haemodynamic status (haemodynamically unstable patients don't have enough pressure in the circulatory system to keep blood flowing reliably to all the various parts of the body where it needs to be at the same time)

Causes of upper GI bleeding

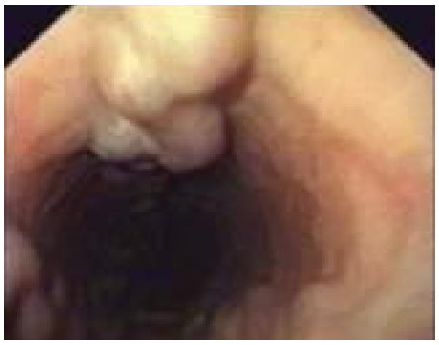
The most common cause is peptic ulcer disease (PUD) i.e. gastric ulcers, duodenal ulcers and gastric erosions.

Other causes include:

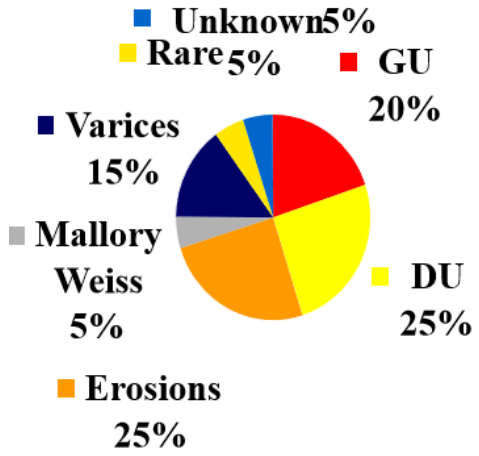
Mallory-Weiss tears in the oesophagus: happens in patients with recurrent forceful vomiting. They differ from PUD in that blood doesn't come out from the first vomitus (patient vomits many times before blood starts to expel). These cases need no intervention as they will recover spontaneously, and are not associated with massive bleeding most of the time.

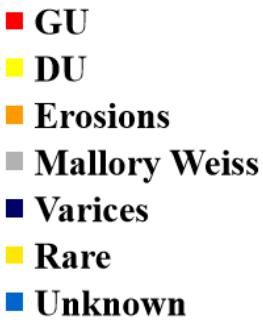
Malignancy: in any gastric ulcer, we should first rule out malignancy

Oesophageal varices: in patients with portal hypertension or cirrhosis; causes massive bleeding that needs intervention by band ligation.



- Dilated tortuous veins of the lower and mid esophagus.
- Secondary to portal HTN
- 30% mortality after the first episode.
- 60% Rebleeding rate





RARE CAUSES

- Neoplasms
- AVM/Ectasia
- Dieulafoy's
- Stoma ulcers
- Esophageal ulcers
- Deodenitis
- Hemobilia
- Aorto-enteric fistulas

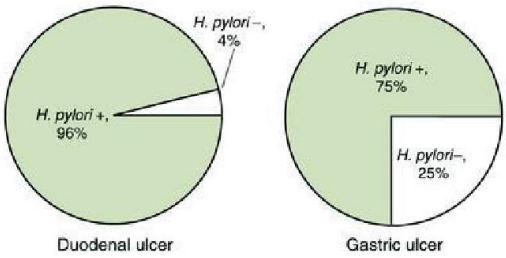
Peptic Ulcer Disease (PUD)

The most common causes are NSAIDs and H.pylori infection.

Around 80% of the Jordanian population are infected with H.pylori, especially those with low socio-economic status, since the mode of infection is faeco-oral. Patients who test positive for H.pylori should be treated since the complications include peptic ulcers and MALToma.

*95% of patients with duodenal ulcers have H.pylori

*75% of patients with gastric ulcers have H.pylori



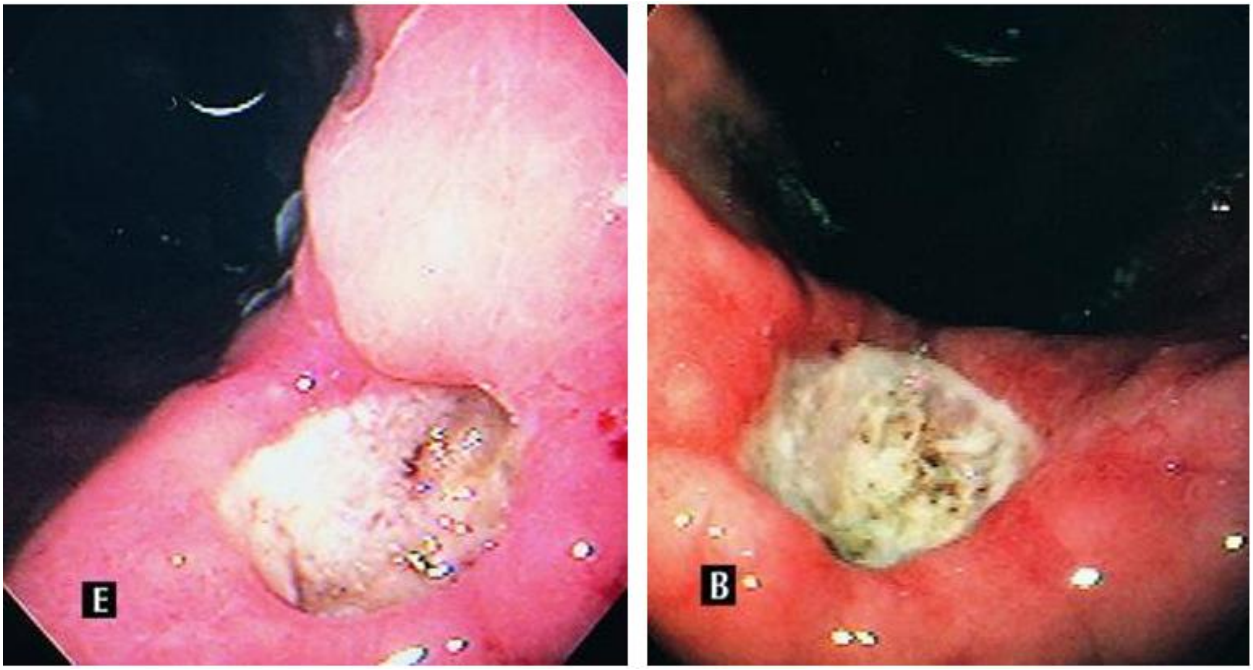


Figure: Gastric cream-based ulcer; inflammation is present, cream base with no visible vessels

Stress Ulcers

Gastric ulcers in patients under stress: ICU patients, burn patients and those with head injuries. They differ from the usual gastric ulcers: multiple, mostly located in the body & fundus of the stomach… (recall what we took in pathology)

Prophylaxis is usually indicated in critically ill ICU patients since they're at risk.

There are different names for different stress ulcers (the dr. said not to focus on the naming):

Cushing ulcers in cases of head injury

Curling ulcers in cases of extensive burns

②Liver Cirrhosis and Portal Hypertension

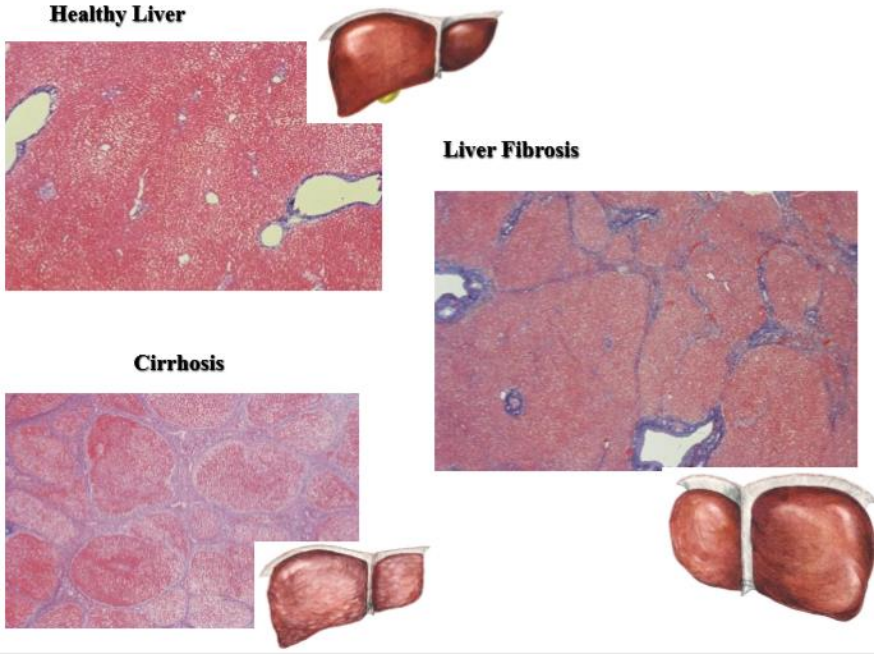
Causes of cirrhosis

-Viral hepatitis B and C

-Alcoholic hepatitis

-Schistosomiasis (uncommon in Jordan but prevalent in Egypt)

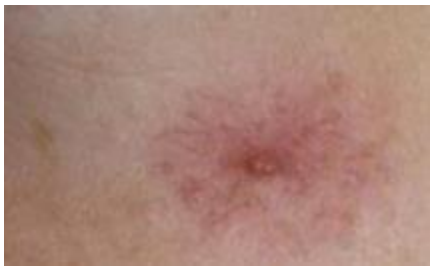
-NASH (NonAlcoholic SteatoHepatitis), previously know as cryptogenic liver cirrhosis. It's the most common cause of cirrhosis here in Jordan. It features steatosis, which leads to inflammation and fibrosis. Common in obese patients who have hypertriglyceridemia and uncontrolled diabetes.



Manifestations of liver cirrhosis:

-Jaundice: yellowish discolouration of the sclera, skin and mucous membranes due to accumulation of bilirubin in the bloodstream.

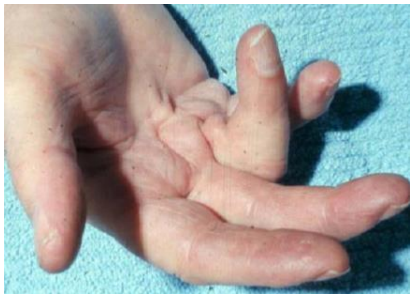
-Spider angiomas: capillaries radiating from centrally raised bumps caused by a dilated arteriole (small artery). They're caused by hyperestrogenemia. It's normal to find a few on the chest of healthy people, but in patients with cirrhosis, there's more than 5 of them, and they're more widely distributed.



-Finger clubbing: the angle between fingernails disappears, followed by curving of nails (usually symmetrical). Can be normal in familial cases.

-Gynaecomastia: development of breast tissue in males (NOT fat tissue)

-Dupuytren's contractures



Complications of liver cirrhosis

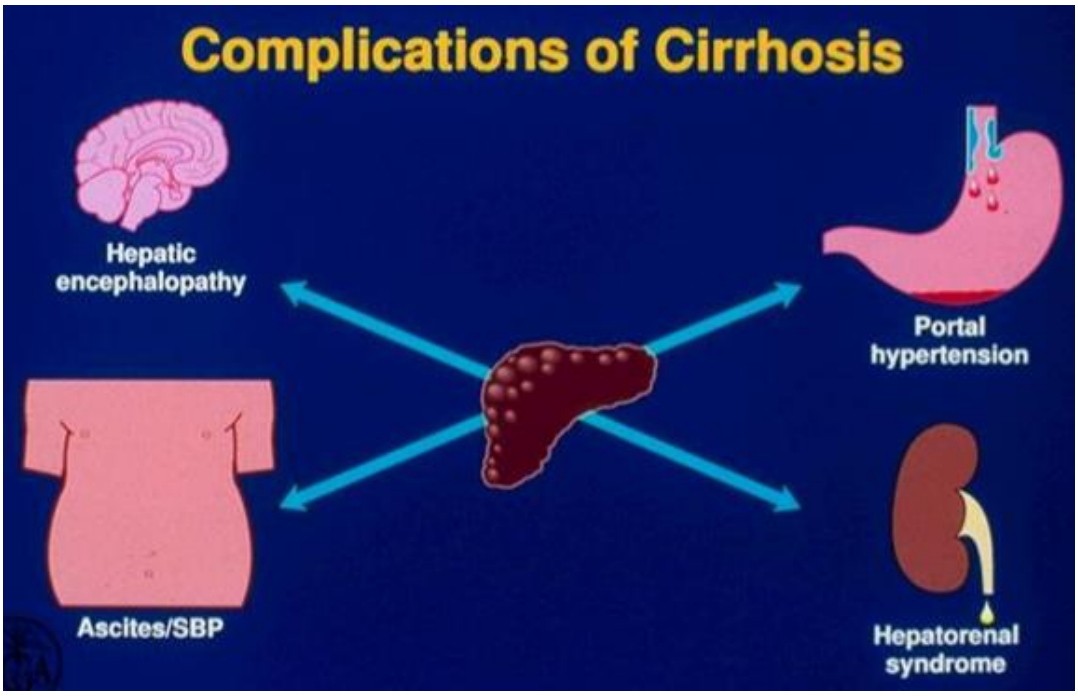
-Hepatic encephalopathy: one of the liver's functions is filtering out toxins. Failure to do so causes toxins to go to the brain, impairing liver consciousness. May progress to coma.

-Ascites: is due to portal hypertension, so peritoneal fluid accumulates. This can cause SBP

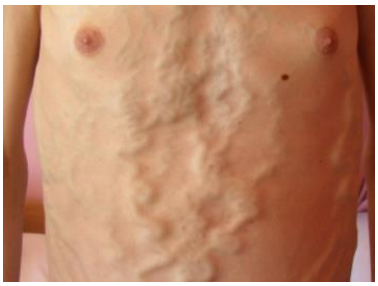
- SBP (Spontaneous Bacterial Peritonitis): bacteria from the GI flora (mainly E.coli) is translocated to the peritoneal fluid to portal hypertension. Peritoneal fluid analysis reveals elevated WBC count.

-Hepatorenal syndrome

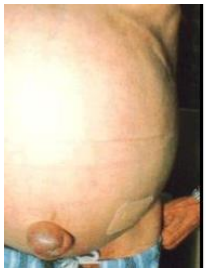
-Oesophageal varices due to portal hypertension

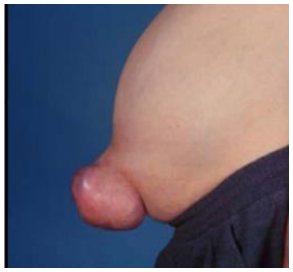


-Caput Medusae: dilation of the external veins over the abdomen (distended umbilical veins are seen radiating from the umbilicus across the abdomen to join systemic veins)

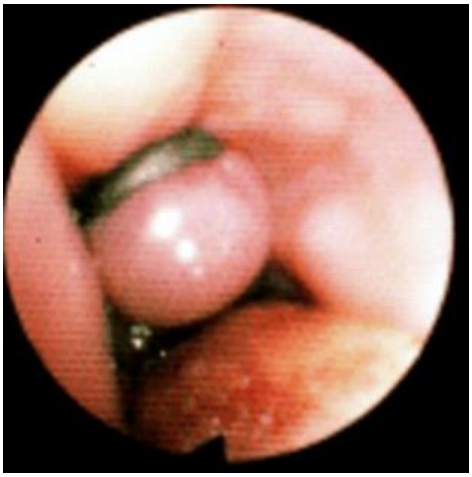


-Herniation: umbilical hernias, or incisional hernias (through a previous scar/wound)

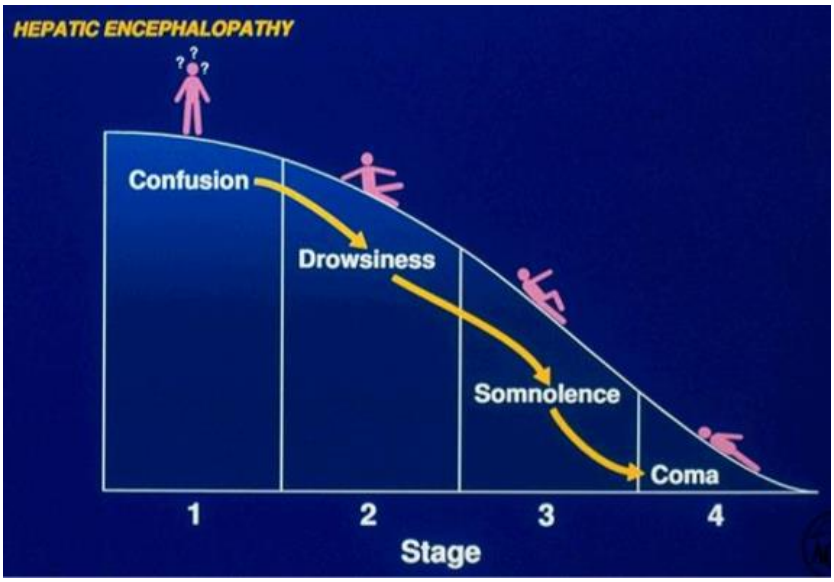




-Asterixis (flapping tremor): in normal people, the hand stays still, but in those with cirrhosis, the hands start shaking/trembling.



In this figure, we can see a column of varices tied with a band (in a process called band ligation). The bands are fired through the endoscope.



The stages of hepatic encephalopathy.

③Viral Hepatitis

Hepatitis B,C,D are transmitted parenterally while A,E are transmitted faeco-orally.

There's a vaccine for Hepatitis A and B (the vaccine for HepB is included in the vaccination programs for new-borns here in Jordan, but the HepA vaccine isn't).

Vaccine for HepA liver is mandatory for those with liver cirrhosis and chronic liver diseases, since there's a high mortality rate.

•Hepatitis A

-Occurs more in areas with low hygiene; transmitted via contaminated food/water and close personal contact (and rarely through blood exposure)

-Commonly contracted by children, and can go unnoticed since the symptoms are very mild (slight jaundice and mild fever)

-Adults, however, present with **acute** clinical illness and more severe manifestations. Less than 1% of those with acute HepA will develop <u>fulminant hepatitis</u> which leads to liver failure. Very few patients will need a liver transplant since there are no antivirals (treatment is supportive)

•Hepatitis B&C

-Parenterally transmitted

-Diagnosis: serology; may need PCR testing sometimes

•Hepatitis D

-Either as a superinfection (patient already has chronic HepB) or coinfection of Hepatitis B&D

•Hepatitis E

-<u>Pregnant women & infants</u> are at risk of developing severe **acute** HepE

Type of Hepatitis

	A	B	C	D	E
Source of virus	feces	blood/ blood-derived body fluids	blood/ blood-derived body fluids	blood/ blood-derived body fluids	feces
Route of transmission	fecal-oral	percutaneous permucosal	percutaneous permucosal	percutaneous permucosal	fecal-oral
Chronic infection	no	yes	yes	yes	no
Prevention	pre/post-exposure immunization	pre/post-exposure immunization	blood donor screening; risk behavior modification	pre/post-exposure immunization; risk behavior modification	ensure safe drinking water