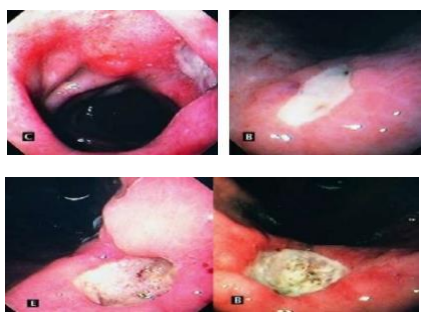
Upper GI bleeding

- Definition: Any bleeding above the ligament of Treitz.

Signs and symptoms	Causes
* **Hematemesis** (vomiting blood) and coffee ground vomiting * **Melena** (black and loose stool) * **Fresh rectal bleeding** * **Hemodynamically unstable** (posture hypotension -Orthostasis- and posture tachycardia and frank hypotension) * **Dizziness** and Palpitations * **Pallor** (due to anemia) * **Abdominal pain and symptoms of Peptic ulcer disease** * **History of NSAID's use** -which is related to peptic ulcers- * **Jaundice and other stigmatas of chronic liver disease.**	* **Peptic ulcer disease** → The most common cause. * **Esophageal varices** * **Mallory Weiss tear** * **Neoplasms** * **Arteriovenous malformation (AVM)** * **Dieulafoy lesion** * **Stoma and Esophageal ulcers** * **Duodenitis** * **Hemobilia** * **Aorto- enteric fistulas**

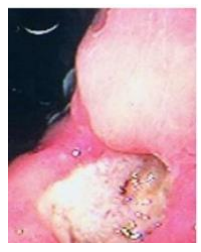
- **Peptic ulcer disease**

1. Most common cause of GI bleeding.
2. Defect in GI mucosa extend from muscularis mucosa
3. Caused by *Helicobacter Pylori (common) *NSAID's that cause imbalance between protective and damage factors.
4. There is an acid hypersecretion and G cell hyperplasia.
5. Two types:
   a. Duodenal ulcer
   b. Gastric ulcer (clean-base ulcer)



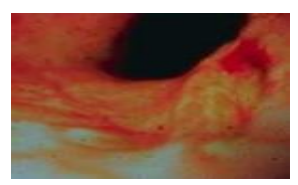
- **Stress ulcers**

1. Happens in critically ill patients like ICU, burns, head trauma patients.
2. Types:
   a. Curling ulcer → in extensive burn patients
   b. Cushing ulcer →head injury patients
3. Causes: Vagal hyperstimulation and vascular hypoperfusion.
4. Multiple/ Body and fundus more affected
5. Prophylaxis by proton pump inhibitor

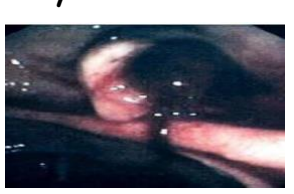


- **Mallory-Weiss tear**

1. Pathogenesis:
   Multiple forceful vomiting → Laceration around the GE junction → bleeding
2. More common in: Chemotherapy patients/ pregnant women in first trimester who have hyperemesis gravidarum/ alcoholics (because they vomit a lot)
3. Self-limiting

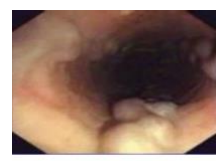


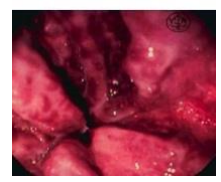
- **Hemobilia**:
  Bleeding through ampulla of vater and biliary tree (Etiology: having previous procedures)



- **Esophageal Varices**

1. Dilated tortuous veins of the lower and mid esophagus.
2. Due to thrombocytopenia and portal hypertension and liver cirrhosis.
3. 60% Rebleeding rate.
4. Two types:
   a. Non-variceal bleeding patients→Treatment: proton pump inhibitors infusion.
   b. Variceal bleeding patients → Treatment: Octreotides = Somatostatin analog
5. rectal exam and endoscopy.
6. The target hemoglobin level in varices patient is 7-8



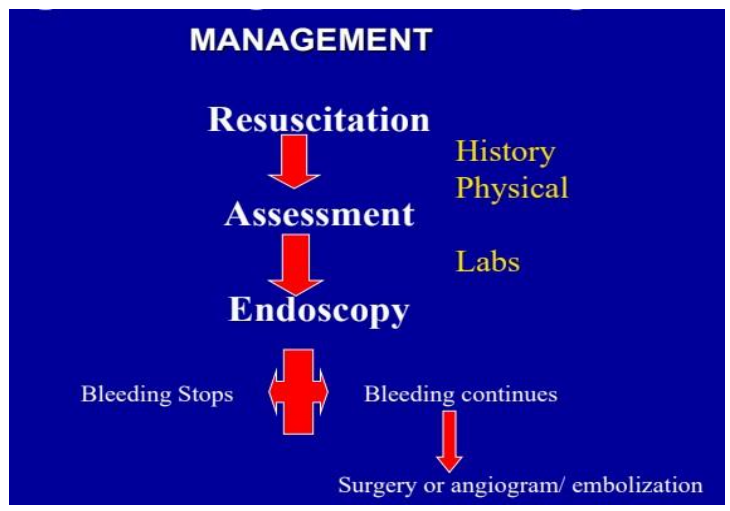


Adverse prognostic factors

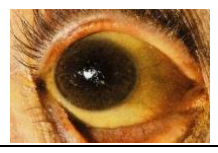
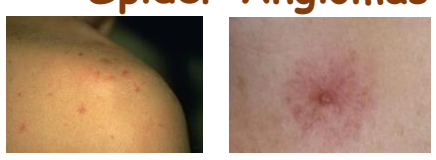
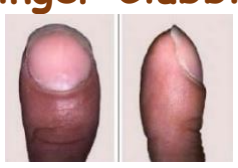
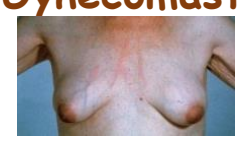
*Diagnosis (Varices + Malignancy)/ *Older age /*Severe initial bleeding (drop in hemoglobin)/ *Recurrence during hospitalization/ *Coincidental diseases (like: DM, ischemic heart disease, hypertension)/ *Endoscopic stigmata of recent bleeding/ *Need for emergency surgery/ *Need for emergency surgery.

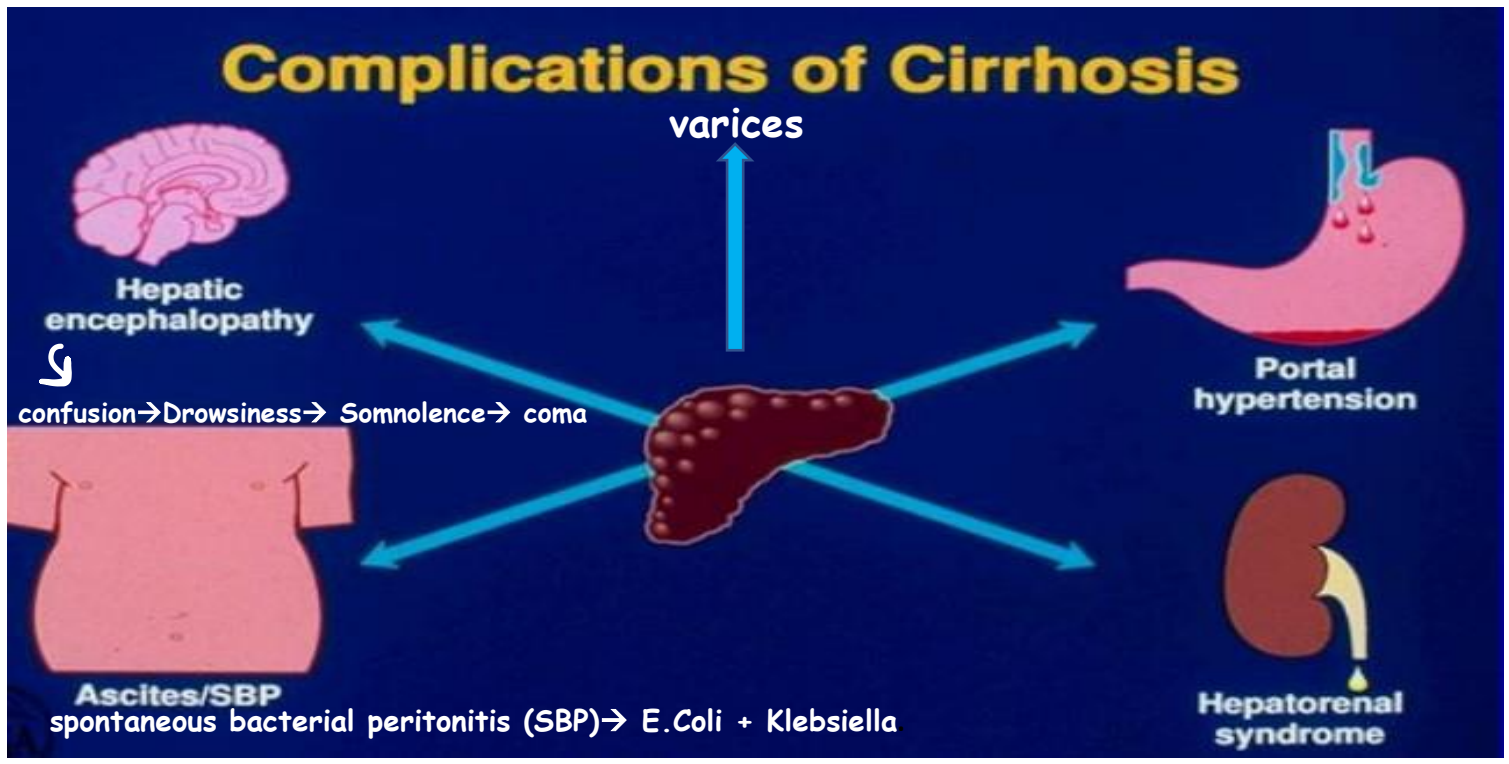
Management

Secure Airway/ Maintain Breathing/ Circulation (IV fluids+ RBC)



Cirrhosis and Portal hypertension

Stigmata (identifying marks) of cirrhosis	
Jaundice 	Accumulation of bilirubin in the blood stream causing yellowish discoloration of plasma and heavily perfused tissues
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.
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
Gynecomastia 	Breast development in men and breast atrophy in women
Dupuytren's Contractures 	Joint contractures



Caput Medusae (complication of portal hypertension)

Distended and engorged umbilical veins which are seen radiating from the umbilicus across the abdomen to join systemic veins.

Astraxia

Flapping tremors, quick arrhythmic movement in background tonic muscle contraction

Lecture 2

كتبت بس يلي ركزت عليه الدكتورة :)

Hepatitis (A-E) Viruses

Hepatitis A

1. Clinical feature: Jaundice/ abdominal pain/ vomiting (especially in adults).
2. Complications: Fulminant hepatitis/ Cholestatic hepatitis/ Relapsing hepatitis
3. No chronic sequelae/ its acute
4. Transmitted by fecal-oral rout.
5. There is a vaccine (is given for chronic liver disease)
6. Laboratory diagnosis (by ELA):
    a. Detection of IgM→ Acute infection
    b. Detection of IgA→ Past infection
7. The patient comes with hepatocyte injury, so transaminase enzymes (AST, ALT) are elevated.
8. Acquiring HAV in young → No or simple symptoms and the older the age →the more symptoms will appear, (even HBV shares this aspect with HAV).

Hepatitis C

1. Causing liver cirrhosis.
2. Serology: Firstly, we test HCV antibodies → If the patient is positive against Hep C antibodies, we have 2 possible scenarios either he has chronic hepatitis C infection or that he had the infection and fully recovered. so, to be sure we make another test: the HCV-RNA test → if it came positive, it means he has a chronic hepatitis C virus.
3. No vaccine

Hepatitis D

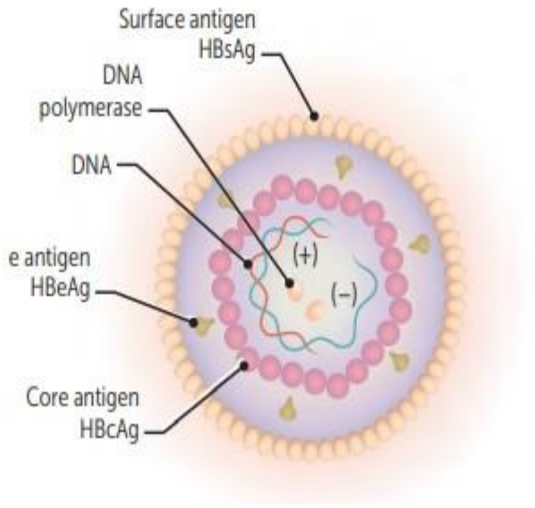
1. Can't be infectious on its own, it can only infect people who are already infected with hepatitis B.
2. **Co-infection:**
    a. Someone gets infected with HBV & HDV at the same time from the same source.
    b. Sever acute disease
    c. Low risk of chronic disease
3. **Super-infection**:
    a. Someone who suffers from HBV from a long time and acquire HDV later on
    b. Chronic infection
    c. High risk of chronic liver disease

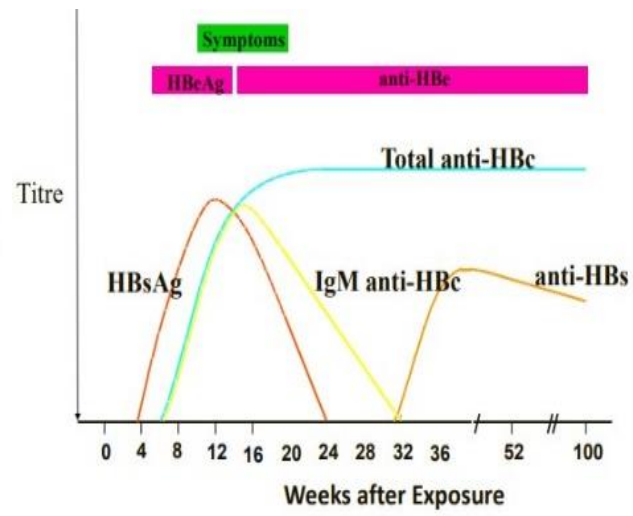
Hepatitis E

HEV usually silent except in pregnant women

Hepatitis B

1. Vertical transmission/ Has a vaccine.
2. Spectrum of Chronic Hepatitis B Diseases:
    a. Chronic hepatitis B
    b. Cirrhosis of Liver
    c. Hepatocellular Carcinoma (without causing cirrhosis)
3. Firstly, increase in HBsAg (indicate active replicating) then it will decline. Then the window period where only **IgM antiHBc** is +ve





	HBsAg	Anti-HBs	Anti-HBc
Susceptible	Negative	Negative	Negative
Vaccinated	Negative	Positive	Negative
Past Infection	Negative	Positive	Positive
Acute Infection	Positive	Negative	IgM Positive
Chronic Infection	Positive	Negative	IgG Positive

4. In infected pregnant mother we measure the titer in the third trimester if it is high→ give her a treatment to suppress the viral load. Then at the stage of delivery, the child should be given IVIG as soon as possible and in the other hand we give him the vaccine

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

Done by:

Shahed Atiyat