UPPER GI BLEEDING

↳ bleeding upper the ligament of Treitz.

↳ symptomes &-

1. Hematemesis ↳ Vomiting Fresh blood, if the blood stays for a time in the stomach ⇒ Cofee ground Vomitus.

2. Melena ↳ Black stool (offensive smell, loos stool) ↳ result of slow transit of blood. ↳ sign Following and upper GI bleeding.

3. Hematochezia ↳ Fresh rectal bleeding.

4. Dizzines ↳ Bleeding and anemia ⇒ hypotension ⇒ Dizzines (with posture)

5. Abdominal pain and symptomes of peptic ulcer disease.

6. Pallor. 7. Hypotension.

8. Orthostasis ↳ Fall in blood pressure when a person transitions from laying down to an upright position (At early stage).

↳ At later stage ↳ The patient will have hypotension no matter what his posture (Qwing) is.

a. jaundice and other stigmatas of chronic liver desease (in case of varieceal bleeding)

↳ History of NSAIDs use (Ibuprofen, voltarin, Nolpain and naproxen) which have a role in every disease (Renal Faliure, ulcers bleeding...).

↳ Causes ↳

1. Peptic ulcers (20% gastric, 25% duodinal) ↳ Most common cause.

2. Varices ↳ Patients with liver Cirrhosis.

3. Mallory Weiss tear ↳ After multiple forceful Vomiting → Less common.

4. Rare Causes ↳ Neoplasms, AVM/Ectasia, DieulaFoy's stoma ulcers, esophageal ulcers, Deodenitis, Hemobilia (bleeding from biliary tree)

↳ Aorta ↳ Colon.
Aorto-enteric Fistulas.

↳ Peptic ulcer disease ↳ Defect in the GI mucosa extending through the muscularis mucosa caused by imbalance between the aggresive and defensive factors.

95% Fo deodenal ulcer ↳ 75% of Gastric ulcers

↳ Main cases are H.Pylori and NSAIDs (Mainly in the antrum) ↳ Even one pill can cause Peptic ulcer especially in diabetic or erderly patients.

↳ 25% of gastric ulcers are associated with NSAIDs and Malignancy.

↳ Acid hypersecretory state, Antral G cell hyperplasia.

↳ Mallory-Weiss ↳

↳ around GE junction. ↳ Classical presentation ⇒ episode of Vomiting then bleeding (Foun in 50% of cases only)

↳ self-limiting (can give PPI)

↳ Hemobilia ↳ Blood coming out of ampulla of vater because of Stone tumor, ERCP...

↳ Stress ulcers ↳ Extensive burn Curling, head injury Cashing of ICU patient.

↳ same treatment (PPI)

↳ Caused by vagal hyperstimulation and vascular hypoperfusion.

↳ Body and Fundus mole affected. ↳ Multiple

↳ Prophylaxis is indicated in Critically ill ICU patients.

↳ Bleeding Esophageal Varicea ↳ Dilated tortuous of the lower and mid esophagus.

↳ 2ry to portal HTN. ↳ on of the most common causes is alcohol.

↳ the most common cause of cirrhosis in jordan Nonalcoholic steatohepatitis (NASH) ↳ result of obesity, Poorly controlled diabetes and hypertriglyceridenimia.

↳ 30% morning after the first episode. ↳ 60% Rebleedin rate.

CHRONIC LIVE DISEASES

(Chirrhosis and portal hypertension)

↳ Liver cirrhosis is irreversible, But if we catch it at early stage such as patient with NASH ↳ with weight loss and control of Diabetes it could be reversed or stopped.

↳ Clinical Features (stigmata) of chronic liver diseases &-

1. jaundice ↳ Accumulation of bilirubin in the blood stream causing yellowish discoloration of plasma and heavily perfused tisskes (sclera mucks membranes)

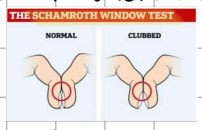
2. Spidar angioma ↳ Dilated arteriol → small, centrally raised bumps and network of dilated capillaries radiated from arteriole.

↳ Pressing cause redness to disappear, and then return rapidly when the pressur is lifted.

↳ Dilation is because hyperstrogenemia (↑ estrogen) in Patients with liver cirrhosis.

↳ They are normally present on the chest of pregnant woman.

3. Finger Clumping ↳ Enlargement of the terminal end of the digit over the distal phalanx.



↳ usually symmetrical.

4. Gyne comastia ↳ Breast development in men and breast atrophy in females.

5. Dupuytren's contractures ↳ Join Contractures (could be associated with diabetes)

↳ Complication of Cirrhosis &-

1. Portal hyper tension ∴ cause ↳ 2. variceal bleeding.

3. Hepatic encephalopathy ↳ ↑ Ammonia or NO ↳ accumulation ↳ cross BBB ↳ ↓ cosciousness.

4. Ascites ↳ Fluid accumulation in the abdomin.

↳ complication of Ascites ⇒ SBP (Spontaneous bacterial Peritonitis)

5. Hepatorenal syndrom.

↳ Caput Medusae ↳ Dilation of superficial abdominal veins.

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

↳ manifestation in patients with liver cirrhosis.

↳ Some patients have ascites with paraumbilical hernia.

↳ Asterixis (Patients with liver cirrhosis) ↳ Flapping tremors, quick arrhythmic movement in background tonic muscle contraction.

↳ Stages of Encephalopathy ↳ confusion and reversal of the sleep (stage 1) / wake cycle → Drowsiness (2) → somnolence (3) → coma (4).

Hepatitis (A-E) Viruses

Viruses	source	Route of transmission	Chronic infection	prevention
↳ D, B, C (دِبِس)	blood / blood derived body Fluids	percutaneous permucosal (blood, sexual or vertical)	yes	D↳ Pre/Post exposure immunization, risk behavior modification B↳ Pre/Post-exposure immunization. C↳ Blood donor screening risk behavior modification
↳ A, E	Feces	Fecal-oral	No Acute infection.	A↳ Pre/Post-exposure immunization E↳ safe drinking water

↳ Hepatitis D cant cause infection by itself (Need presence of B)

↳ we have Vaccines against A+B: ↳ Every child in jordan gets it.

Patients with chronic liver disease.

↳ No vaccine against Hepatitis C (RNA), but it has a very effective treatment (99%)

↳ Hepatitis A Virus ↳

↳ Possibility of developing symptoms is directly proportional with age:

- Jaundice ↳ <6 yrs ↳ <10% / 6-14 yrs ↳ 40-60% / >14 yrs ↳ 70-80%

↳ Incubation Period = Range 15-50 days, Average 30 days.

↳ Complications: 1. Fulminant (Server acute) hepatitis. 2. Cholestatic hepatitis.

3. Relapsing hepatitis.

↳ Transmission ↳ close personal contact (household, Child day care Centers) contaminated Food or water (raw shellfis), Blood exposure (Rare)

↳ Diagnosis: ~ Acute infection ↳ HAV-IgM by EIA.

~ Past infection ↳ HAV-IgG by EIA.

↳ Fecal HAV is highe Before the appearance of symptomes.

↳ Hepatitis B Virus ↳

↳ Incubation period ↳ Range 45-180 days, Average ↳ 60-90 days.

↳ Clinical illness (jaundice) ↳ <5yr ↳ 10% / 5years ↳ 30-50%

↳ Acute case-Fatality rate ↳ 0.5-1%

↳ chronic infection ↳ <5years ↳ 30-90% / 5 years ↳ 2-10%

↳ Mortality From chronic liver disease ↳ 15-25%

↳ is a pregnant woman has hepatitis, it will be transmited to the baby during delivery ↳ this baby will have no symptoms, but 95% he will be a chronic carrier. (Because of immune tolerant phase.)

↳ when his immune system recognize the virus (immune active phase) he will show symptoms.

↳ Adults will show more symptoms (their immune system will recognize the virus)

↳ Chronic Hepatitis B Disease ↳

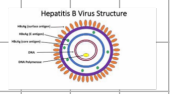
1. Chronic persistent Hepatitis (Asymptomatic).

2. Chronic Active Hepatitis (Symptomatic)

3. Cirrhosis. 4. Carcinoma.

↳ Hepatitis B surface antigen (HBsAg) ↳ Then ↳ Anti-HBs (Antibody)

↳ Window Period ⇒ when the HBsAg has disappeared and the anti-HBs hasn't appear yet ↳ We have IgM anti-HB c ↳ core.



↳ Transmission:

1. sexual. 2. Parenteral (وريدي) ↳ IVDA, Health workers.

3. Perinatal ↳ Mothers with HBeAg positive are more likely to transmit the virus. ↳ The main means of transmission in high prevalence population.

↳ concentration of virus in Body Fluids ↳

High ↳ Blood, Serum, Wound exudates.

Moderate ↳ semen, Vaginal Fluid, Saliva.

Low ↳ Urine, Feces, Sweat, tears, breastmilk.

↳ Diagnosis ↳ Serological tests for Both acute and chronic.

~ HBsAg ↳ general marker (we use it in acute infection)

~ HBsAb ↳ Recovery or immunity to HBV infection.

~ Anti-HBc IgM ↳ Marker of acute infection at the window period.

~ Anti-HBc IgG ↳ past or chronic infection.

~ HBeAg ↳ Active replication of virus and therefore infectiveness.

~ Anti-HBe ↳ virus no longer replicating, but can be still positive for HBsAg.

~ HBV-DNA ↳ Active replication of virus, it is more accurate than HBeag especially in cases of escape mutants, Mainly for monitoring response to therapy.

↳ Prevention ↳

1. Highly effective recombinant Vaccines (universal vaccination for neonate).

↳ Antibody titer should >10 to be protective.

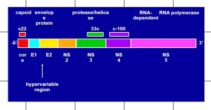
2. HB Immunoglobulin ↳ For persons who are exposed, efficacious within 48 hours of the incident.

↳ may be given to neonates at increased risk of the virus they should given the vaccin in arm and Ig in the other.

↳ Hepatitis C Virus ↳

↳ Incubation Period ↳ Range 2-26 weeks / Average 6-7 weeks

↳ Clinical illness ↳ 30-40% (20-30%)



↳ Chronic hepatitis ↳ 70%

↳ Persistent infection ↳ 85-100%

↳ No protective antibody

↳ Same as chronic hepatitis B infection.

↳ Risk Factors ↳ 1. Transfusion of transplant from infected donor.

2. Injected drug use. 3. Hemodialysis (غسيل كلى)

4. Accidental injuries with needles/sharps. 5. sexual/household exposure to positive contact.

⇒ Birth to HCV-infected mother.

↳ Diagnosis :-

~ HCV antibody ↝ Not useful in acute phase (it take ↳ weeks (atleast) after infection -> produce antibody).

~ HCV-RNA ↝ by PCR and branched DNA.

↳ may be used to diagnose the infection in acute phase.

↳ Mainly used in monitoring the response of therapy.

~ HCV-antigen ↝ EIA for HCV antigen is available.

↳ same capacity as HCV-RNA but it is much easier.

~ HCV antibody ~ positive ↝ RNA [positive → chronic; negative → previous infection.

↝ Hepatitis D ↝

1. Coinfection

↳ sever acute disease.

↳ low risk of chronic infection.

2. Superinfection

↳ Usually chronic infection.

↳ May present as a acute hepatitis.

↳ High risk of sever chronic liver disease.

↳ Transmission ↝ percutaneous or permucosal exposures, sex contact and injecting drug use.

↝ Hepatitis E ↝

↳ Incubation period ↝ Range 15-60 days / Average 40 days.

↳ Case-Fatality rate ↝ overall 1-3% / (Higher risk) pregnant women 15-25%

↳ severity ↝ ↑ with age.