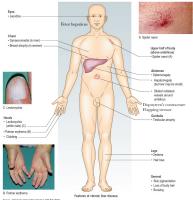
# Liver cirrhosis

- ✓ **Blood supply of liver** : 75% portal vein , 25% hepatic artery
- ✓ Obstruction of hepatic veins > Budd Chiari syndrome
- ✓ Irreversible late fibrosis , distortion of hepatic structure , and nodule

✓ Clinical presentation: most patient are asymptomatic and are diagnosed incidentally, stigmata of chronic liver disease, decompensated liver disease (variceal bleeding, ascites)

stigmata of chronic liver disease



#### ✓ Labs:

- ALT, AST: Mildly elevated, AST > ALT, AST/ALT>1
- ALK phosphatase: elevated by 2-3 fold, mainly if the cause of the cirrhosis is cholestatic (primary sclerosing cholangitis
- **GGT**: with ALK phosphatase, elevated mainly in alcoholic patients
- Albumin: hypoalbuminemia, but not specific for liver disease
- Bilirubin: normal if well compensated and rise progressively
- PT: prolonged, and reflect the hepatic dysfunction
- **Globulin**: elevated ( IgG > primary biliary cirrhosis , IgM > autoimmune hepatitis )
- Sodium: hypervolemic hyponatremia
- CBC: Anemia, thrombocytopenia, leukopenia, neutropenia

## ✓ Diagnosis :

- U/S: small liver with nodule, increase diameter of veins, collateral veins
- **CT**,**MRI**: if HCC is suspected
- **Fibroscan**: harder tissue > faster wave
- Liver biopsy: gold standard but rarely used
- Upper endoscopy: esophageal varices

 $\checkmark$  Child classification : INR , bilirubin , hepatic encephalopathy , ascites , albumin

A <7 compensated , B 7-10 and C >10 non compensated

✓ MELD score : bilirubin , INR , Cr



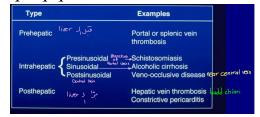
- ✓ Treatment: transplantation and management of complication
- **✓** Complications of cirrhosis
  - > Portal HTN



High risk of variceal bleeding with high mortality rate

Not usually done but it is Measured by: transhepatic, transjagular, laparotomy, catheterization splenic pulp puncture

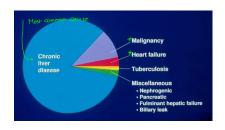




Ascites (abd discomfort or pain, SOB, wt gain, early satiety)

**Diagnosis**: by diagnostic paracentesis and fluid analysis (proteins, cells count, albumin, culture > SAAG) – no contraindications

Indications of paracentesis:
New onset ascites
Refractory ascites and clinical detorration
Worsening kidney fuction
Fever
Admision to hospital





#### **Therapy**:

- Low sodium diet (less than 2 g /day) -without fluid restriction-
- **Diuretics** (furosemide 40mg and spironolactone 100 mg)-Initial doses-For refractory ascites
- large volume paracentesis with albumin supplement (8 g for each litter removed after removing 4 litter) albumin to prevent hepatorenal syndrome
- **TIPS**: stent is put between hepatic circulation and portal circulation > blood goes to systemic circulation without detoxification > increase risk of hepatic encephalopathy and HF
- Peritoneovenous shunt, liver transplantation

**Complication of ascites:** 

## • Hydrothorax : (transudative pleural effusion)

2/3 right sided, but can be left sided and bilateral

Fluid is similar to ascites (high SAAG, low protein) and treated as ascites by diuretics, large volume paracentesis and TIPS -Not by chest tube-

#### • Umbilical hernia

Firstly, it is Treated by ascites control, and if it doesn't resolve, it is repaired surgically unless incarceration happens it it is repaired by urgent surgery.

- SBP (spontaneous bacterial peritonitis)
  - -Caused by: E.coli, klebsiella, strep pneumoniae
  - -In advanced cirrhosis and 40 -50 % is asymptomatic and detected in hospital admission but can present with Fever , abdominal pain , tenderness , change in mental status , jaundice , PMN count >250 in ascitic fluid
  - **-Treatment**: culture and empirical antibiotics ( cefotaxime , ceftriaxone ) for 5 days

Then Life long ciprofloxacin when SBP happened once and reduce ascites

Patient with (upper GI bleeding and cirrhosis), (cirrhosis, renal and liver failure) should take prophylactic antibiotics for 7 days.

#### • Tense ascites

## **▶** Hepatorenal syndrome :

- -Renal failure with cirrhosis and ascites
- -High mortality rate, (death within weeks)
- -Azotemia , hyponatremia , urine sodium < 10 , no response to plasma expansion , high CVP > 10
- **-Treatment**: supportive with fluid, restrict sodium and water, hemodialysis, liver transplantation, avoid nephrotoxic drugs (NSAID, gentamicin)

#### > Hepatic encephalopathy

-Reversible neuropsychiatric changes with normal EEG, asterixis

-There is a correlation between ammonia and stages of encephalopathy but not used in diagnosis, used when HE diagnosis in question

## Differential diagnosis:

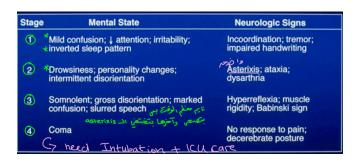
- 1. Intracranial lesions (stroke, hematoma, abscess)
- 2. Upper GI bleeding
- 3. Alcohol intoxication or withdrawal
- 4. Drugs :sedative, hypnotics, new diuretics usage
- 5. Hypoglycemia or hyperglycemia
- 6. Uremia
- 7. Hypokalemia
- 8. Metabolic alkalosis
- 9. Hypoxia, hypercapnia
- 10.Infections (meningitis, sepsis, UTI, SBP)
- 11.psychatric, post seizures

## Precipitant:

- 1. HCC
- 2. High protein diet
- 3. Constipation
- 4. Upper GI bleeding
- 5. TIPS
- 6. Infection
- 7. Alcohol and drugs
- 8. recent surgery

#### Treatment:

- -First line : Lactulose : 30 to 45 mL [20 to 30 g] / two to four times per day should be titrated to achieve two to three soft stools per day
- -rifaximin if no improvement after 48 h
- -Treatment of the underlying cause and to prevent recurrence of the HE.
- Diet
- Daily energy intakes should be 35-40 kcal/kg ideal body weight.
- Daily protein intake should be 1.2-1.5 g/kg/day.
- Small meals or liquid nutritional supplements evenly distributed throughout the day and a late-night snack should be offered .
- Oral BCAA supplementation may allow recommended nitrogen intake to be achieved and maintained in patients intolerant of dietary protein.



# Budd-Chiari Syndrome

Extra: but there were 3 past papers questions about it

- 1. Liver disease caused by occlusion of hepatic venous outflow, which leads to hepatic congestion and subsequent microvascular ischemia.
- 2. The course is variable, but most cases are indolent, with gradual development of portal HTN and progressive deterioration of liver function.
- 3. Rarely the disease is severe and leads to acute liver failure, which may be fatal without immediate therapy

**Causes**; hypercoagulable states, myeloproliferative disorders (e.g., polycythemia vera), pregnancy, chronic inflammatory diseases, infection, various cancers, trauma.

-Condition is idiopathic in up to 40% of cases.

Clinical Features (resemble those of cirrhosis)-hepatomegaly, ascites, abdominal pain (RUQ), jaundice, variceal bleeding.

**Diagnosis**: hepatic venography; serum ascites albumin gradient >1.1 g/dL.

#### Treatment:

- 1. Medical therapy (e.g., anticoagulation, thrombolytics, diuretics) is usually unsatisfactory
- 2. Surgery is eventually necessary in most cases (balloon angioplasty with stent placement in inferior vena cava, portacaval shunts).
- 3. Liver transplantation if cirrhosis is present.