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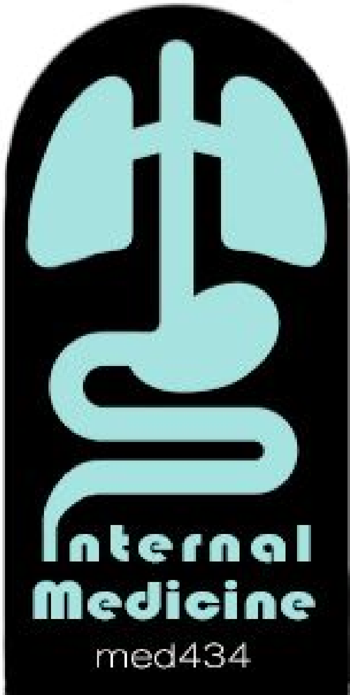
Liver Cirrhosis



★ **Objectives:**

1- Understand the basic mechanisms of portal hypertension

2- Recognized the classic presentations of portal hypertension complications

3- Get an idea on the management of these complications

★ Resources Used in This lecture:

Step up to medicine - Master of board -Doctor's slides - Doctor notes

Done by: Rawa Alohali & Latefa Alareek

Contact us at: InternalMedicineTeam434@gmail.com

Revised by : Hussain Alkaff

Liver Cirrhosis

Definition (by three characteristics)

1. **Fibrosis** in the form of delicate bands or broad scars/septa.
2. **Widespread Nodules** . (NO NODULE NO CIRRHOSIS!!)
3. **Disruption** of the architecture of the entire liver.

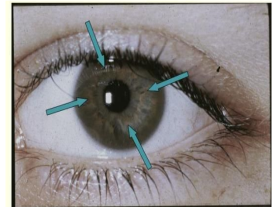
Pathophysiology

The distortion of liver anatomy causes **:**

a. **Decreased sinusoidal blood flow through the live**r (vasoconstriction) → high resistance in portal circulation (portal hypertension) → this lead widespread manifestations, including (ascites, peripheral edema, splenomegaly, and varicosity of veins)
b. **Hepatocellular failure that leads to impairment of biochemical function**s, such as decreased albumin synthesis and decreased clotting factor synthesis.

Causes

- **Alcohol abuse** (AST is higher than ALT) i **Chronic Viral hepatitis (B and C)**
- **Metabolic such as :**



- **Hemochromatosis :** inherited disorder characterized over iron deposition in the liver (high ferritin level
- **Wilson disease** : Positive Kayser-Fleischer rings
- **Alpha-1-antitrypsin** deficiency ; associated with COPD with no history of smoking
- **primary biliary cirrhosis (PBC)** : more common in women 40-50 y anad associated with fatigue ,pruritus
- **primary sclerosing cholangitis (PSC) :** more common in men 20 y and assisted with IBD
- **Autoimmune hepatitis such SLE**
- **Vascular**: Budd-Chiari Syndrome and portal vein thrombosis.

Note :

- Any condition leading to persistent or recurrent hepatocyte death may lead to cirrhosis.
- Both PBC and PSC → high levels of alkaline phosphatase and Total bilirubin
- Gold standard test for diagnosis of cirrhosis > **LIVER BIOPSY!**
- in alcoholic disease.--> Pyridoxine deficiency in alcoholics that's why AST higher than ALT

Complications

1) Portal HTN

- **definition:** portal venous pressure above 7 mmHg.
- **Pathophysiology :** constriction of **sinusoidal** veins → **Increased vascular resistance** → dilation of the collateral veins caused by the nodules"**mechanical resistance** + nitric oxide (**Dynamic resistance** .

2) Varicosity of veins :

- **Pathophysiology :** Portal HTN ➞ dilation of the collateral veins caused by the nodules"**mechanical resistance** + nitric oxide (**Dynamic resistance**)
- Including ;
- **Esophageal varices and gastric varices** : Esophageal more common than gastric , both may leads massive bleeding (hematemesis or melena) and exacerbation of hepatic encephalopathy. Management of variceal without bleeding usually give nonselective BB (propranolol or nadolol) but in active bleeding include

A. **ABC** : IV fluids (normal saline) to manian BP , PRBC if hemoglobin level is low , platelets if the the is low , plasma if PT or INT is high .
B. **IV prophylactic antibiotic** (ceftriaxone or ciprofloxacin) : to prevent infection because bleeding is good media to bactria
C. **IV vasopressin** : octreotide or somatostatin 3 to 5 days. It causes vasoconstriction of the splanchnic vessels → the varices to collapse.
D. **upper GI endoscopy :** either Endoscopic variceal ligation (EVL) or Endoscopic sclerotherapy
E. **Nonselective BB (propranolol or nadolol)** : to prevent recurrence of bleeding
F. **Transjugular intrahepatic portosystemic shunt (*TIPS) :***artificial channel between portal system and hepatic vein *used If the above drugs can't control the bleeding*

- **Rectal hemorrhoids** :due varicosity of Rectal vein, which leads to hematochezia
- **Caput medusae** : dilation of abdominal wall veins

3) ASCITES

- **Pathophysiology : twa ways**
  - Cirrhosis→ Portal HTN → low perfusion of kidney → activation RAAS → salt & water retention → increase in hydrostatic pressure → fluids pass to third space
  - Cirrhosis → hypoalbuminemia → reduced oncotic pressure → fluids pass to third space
- **Diagnosis :**
  - **History and physical examination** : abdominal distention , Bulging flanks , Positive Shifting dullness and Fluid wave
  - **Abdominal Ultrasound** : can detect as little as 30 ml
  - **Measure SAAG** (serum albumin to ascitic fluid albumin gradient) :

	SAAG > 1.1	SAAG < 1.1
Albumin	High	Low
Causes	Cirrhosis (portal HTN) Congestive Heart failure (RSHF) Constrictive pericarditis Massive hepatic metastases Hepatic vein thrombosis	Infection (except SBP) Cancer Pancreatitis Serositis Nephrotic syndrome

- **Paracentesis with ascitic fluids WBC , gram stain, culture ,and albumin** : done only in New onset ascites , fever , abdominal pain and tenderness c (SBP suspected).

❑ **Mangement :**

- Treat the underlying cause
- Salt dietary restriction
- Diuretics both lasix and spironolactone
- Recurrent tapping : done when asites not respond to above
- TIPS : done when asites not respond Recurrent tapping
- Liver transplantation : last option when asites cannot control

6) Spontaneous bacterial peritonitis (SBP)

❑ **Definition** : Infected ascitic fluid

❑ **Eitilogy :** E.coli (most common) , Klebsiella or Streptococcus pneumoniae.

❑ **Clinical features** : asitis with fever , abdominal pain and rebound tenderness

❑ **Diagnosis** : Paracentesis is done and then :

- WBC of asiatic fluid → WBC > 500 and PMN > 250
- Culture asiatic fluid → positive

❑ **Treatment (both)**

- IV third generation cephalosporin (ceftriaxone or cefotaxime)
- Albumin

Note:

- SBP recurrence rate is very high (70 % in first year)
- Best initial test is WBC test of asiatic flud
- Paracentesis is repeated after 2-3 days to make sure decrease of PMN

5) **Hepatic encephalopathy**

- **Pathophysiology :** Liver cirrhosis → liver unable to detoxify the ammonia → high levels of Level of Ammonia that pass through collaterals vein to brain
- **Exacerbation factors :**
- Constipation is the most common.(build up of bacteria.)
- GI bleed. (bacterial growth) Blood is a good media for growth.
- Hepatic necrosis
- Infections
- renal failure
- Electrolytes imbalance (mainly hypokaemima and alkalosis)
- Drugs (like narcotics and , diuretics sedative drugs.)
- **Clinical features :**
- Reversal of sleep pattern
- Disturbed consciousness , Personality changes , Intellectual deterioration
- Reversal of sleep pattern
- Hyperreflexia and rigidity
- Fetor hepaticus. (breath of the dead)
- Asterixis (Flapping tremor) (You ask the patient to extend his hands, Toxins impair the conduction then hands stop, brain goes no, extend again!)
- Fluctuating
- **Treatment (both)** :
- Lactulose : changes the colonic PH, making it acidic by forming NH4 →.. prevents absorption of ammonia and promote excretion .
- Rifaximin : kills the flora → decrease the ammonia production

6) **Hepatorenal syndrome :**

- **Definition** : Progressive renal failure in advanced liver disease,
- **Pathophysiology :** vasoconstriction of renal artery → hypoperfusion of kidney
- **Clinical Features : a**zotemia, oliguria, hyponatremia, hypotension, low urine sodium
- **Treatmen**t : Liver transplantation i

Note :

- Hepatorenal syndrome : it's functional renal failure which means the kidney normal in morphology
- Hepatorenal syndrome not respond to volume expansion

7) **Hyperestrinism**

- **Pathophysiology :** decrease the Hepatic catabolism of estrogens
- **Clinical Features**
- Spider angiomas
- Palmar erythema .
- Gynecomastia.
- Testicular atrophy.

8) **Coagulopathy :**

- ❑ **Pathophysiology** : decrese production of clotting factors
- ❑ **Characterized by** :prolonged of PT and PTT and elevation INT
- ❑ **Treatment by** : Fresh frozen plasma

Note : Vitamin K can;t be used because the liver is affected

9) Hepatocellular carcinoma (HCC) :

- ❑ **Epidemiological :**One of the most common cancers in Saudi Men.
- ❑ **Causes :**
- Cirrhosis (most common)
- Non cirrhosis such as HBV
- ❑ **Diagnosis :**
- Ultrasound (initially)
- CT or MI to (confirm the diagnosis)

Remember

- Complication of POrtal HTN : **varices** , ascites , Splenomegaly . peripheral edema
- Patient with Liver cirrhosis should undergo screening every 6-12 Months to check for HCC