MEDICINE

432 Team

28

Liver Cirrhosis and Complications

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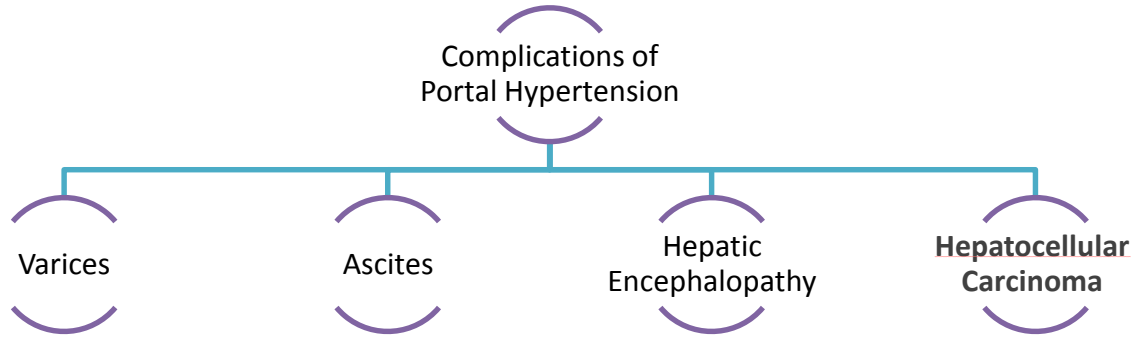
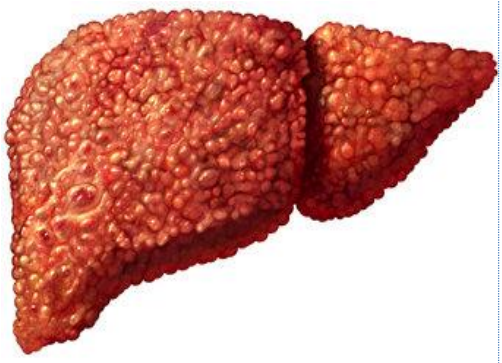
COLOR GUIDE: • Females' Notes • Males' Notes • Important • Additional

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



What is Liver Cirrhosis?



Diffuse fibrosis of the liver with nodule formation.

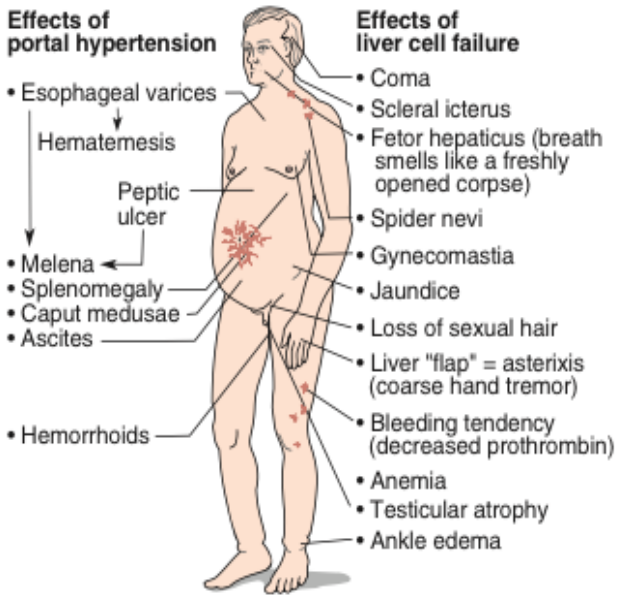
Abnormal response of the liver to any chronic injury. (Cirrhosis results from the necrosis of liver cells followed by fibrosis and nodule formation. The liver architecture is diffusely abnormal and this interferes with liver blood flow and function. This derangement produces the clinical features of portal hypertension and impaired liver cell function.)

Causes of Cirrhosis:

1. Chronic viral hepatitis
2. Metabolic: hemochromatosis, Wilson dis, alfa-1-antitrypsin, NASH
3. Prolonged cholestasis (primary biliary cirrhosis, primary sclerosing cholangitis)
4. Autoimmune diseases (autoimmune hepatitis)
5. Drugs and toxins
6. Alcohol

Pathology

The characteristic features of cirrhosis are regenerating nodules separated by fibrous septa and loss of the normal lobular architecture within the nodules



Management

Management is that of the complications seen in decompensated cirrhosis. Patients should have 6-monthly ultrasound to detect the early development of a hepatocellular carcinoma, as all therapeutic strategies work best with small single tumours. Treatment of the underlying cause may arrest or occasionally reverse the cirrhotic changes.

(From Kumar)

Complications and effects of cirrhosis:

Here we will talk about:

- Portal hypertension
- Varices
- Ascites
- Hepatic Encephalopathy
- Hepatocellular Carcinoma

Portal Hypertension:

The portal vein is formed by the union of the superior mesenteric and splenic veins. The pressure within it is normally 5–8 mmHg with only a small gradient across the liver to the hepatic vein in which blood is returned to the heart via the inferior vena cava. Portal hypertension can be classified according to the site of obstruction:

- ***Prehepatic*** – due to blockage of the portal vein before the liver
- ***Intrahepatic*** – due to distortion of the liver architecture, which can be presinusoidal (e.g. in schistosomiasis) or post-sinusoidal (e.g. in cirrhosis)
- ***Post-hepatic*** – due to venous blockage outside the liver (rare).

As portal pressure rises above 10–12 mmHg, the compliant venous system dilates and collaterals occur within the systemic venous system. The main sites of the collaterals are at the gastro-oesophageal junction, rectum, left renal vein, diaphragm, retroperitoneum and the anterior abdominal wall via the umbilical vein. (Kumar)

Mechanism of Portal HTN

Resistance portal flow:

Mechanical Nodules

- The blood go through portal vein to sinusoid then pass to hepatic vein.
- Sinusoid: The distance between portal vein and hepatic vein surrounded by nodules.
- When liver become cirrhotic the blood will not pass Easley (hypertension).

Dynamic Nitric oxide:

- Vasoconstriction of vessel inside liver (the cause is unknown)

Varices:

- Collateral:

1. Esophagus
2. Gastric
3. Colo-rectal
4. Portal hypertensive gastropathy

Pathophysiology: Because of portal hypertension the blood will move to area of low resistance, which is:

1. Esophagus causing esophageal varices the patient present with hematemesis and melena
2. Umbilicus causing caput medusa
3. Spleen causing splenomegaly
4. Spider angiomata.
5. Palmar erythema
6. Asterixis (flapping tremor)

Varices Diagnosis:

1. History: Hematemases, melena
2. Physical examination
3. Ultrasound abdomen
4. Endoscopy

Note(s):

Eighty percent of GI bleeding will stop spontaneously if the fluid resuscitation is adequate. Most patients die of inadequate fluid replacement.

Varices Management - General:

1. ABC
2. 2 IV Lines (normal saline)
3. Type and cross match (blood transfusion)
4. Resuscitation
5. IVF
6. Blood
7. Platelet transfusion (platelet <75,000)
8. Fresh frozen plasma (Correct Pt)

Varices Management-Specific

1. IV vasoconstrictors (Octreotide).
2. Endoscopic therapy. Octreotide: vasoconstrictive effects on the dilated splanchnic blood vessels reduce amount of blood going to bowel
3. Banding varics endoscope pass through esophagus When varices are found, tiny elastic bands are placed around the enlarged veins in the esophagus to tie them off so they can't bleed
4. Sclerotherapy
5. Shunting
6. Surgical (they never do surgery know because it increase mortality by 70%)
7. TIP Transjugular intrahepatic portosystemic shunting

Varices Prevention

1. Treat underlying disease (treat cirrhosis)
2. Endoscopic banding protocol (for acute and prophylactic purpose)
3. B-blockers
4. Liver transplantation if Endoscopic banding protocol and B-Blocker did not work we do liver transplant

Note(s):

TIPS is a tract created within the liver using x-ray guidance to connect two veins within the liver. The shunt is kept open by the placement of stent.
TIPS catheter pass through jugular vein ----- superior vena cava ---
RA------ inferior vena cava ------hepatic vein
The blood flow from hepatic to portal.

(TIPS) is used to decrease portal pressure in those who are not controlled by Octreotide and banding.

Ascites

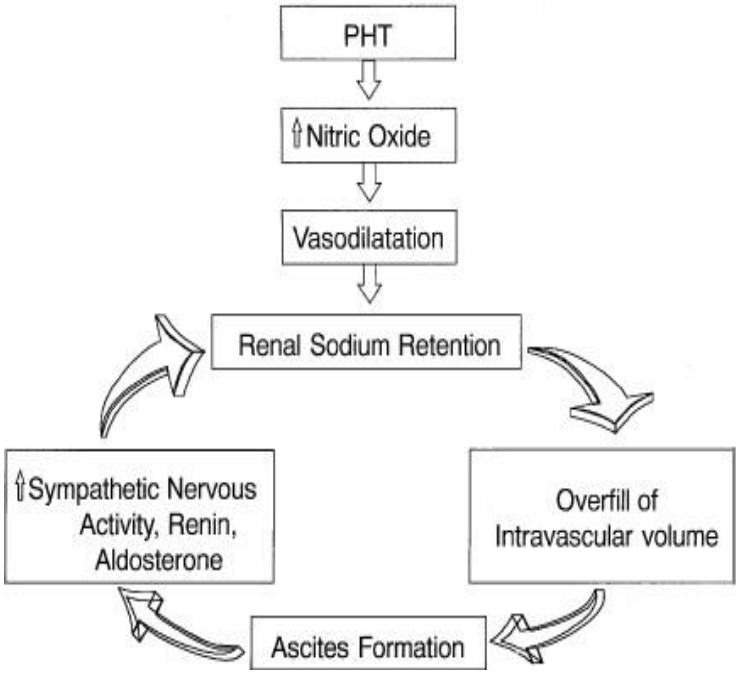
Definition: fluid in the peritoneal cavity

Mechanism of Ascites

1-PHT

Endothelial produce nitric oxide ⟶ nitric oxide goes to systemic circulation cause vasodilation ⟶ Because of vasodilation the kidney stimulate RAAS ⟶ fluid retention ⟶ ascites formation

2-Low protein



Causes of Ascites

1. Liver disease: cirrhosis
2. Right sided heart failure
3. Kidney disease (nephrotic syndrome)
4. Low albumin (malnutrition, bowel loss)
5. Peritonial infection (TB…)
6. Peritonial cancer.

The cause of ascites could be local and systemic:

Local: TB, cancer, ovarian.

Systemic: heart failure, kidney failure, liver failure,

Presentation

History:

- Increased abdominal girth
- Increased wt

Physical exam:

- Bulging flanks
- Shifting dullness
- Fluid wave

Diagnosis:

1. Physical examination
2. Ultrasound (conclusive test)
3. Ascitic tap
4. WBC (>250 PMN: SBP) SBP: Spontaneous Bacterial Peritonitis
5. RBC
6. SAAG (serum albumin to ascitic fluid albumin gradient)

Difference between serum albumin and ascetic fluid albumin

>11 mg/dl: High SAAG >> liver, heart causes • Portal hypertension • CHF • Hepatic vein thrombosis • Constrictive pericarditis

<11 mg/dl: Low SAAG >> other causes Infections (except SBP) • Cancer • Nephrotic syndrome

Classification of Ascites by the Serum Albumin-Ascites Gradient

High albumin gradient (SAAG ≥1.1 g/dL)	Low albumin gradient (SAAG <1.1 g/dL)
Cirrhosis	Peritoneal carcinomatosis
Alcoholic hepatitis	Peritoneal tuberculosis
Congestive heart failure	Pancreatitis
Massive hepatic metastases	Serositis
	Nephrotic syndrome
Portal hypertension or heart failure	**Peritoneal disease or kidney disease**

Do not be confused by units :

>11 mg/dl ≥1.1g/dl

<11 mg/dl ≤1.1g /dl

Treatment-General

- Treat the underlying disease
- Salt restriction (<2gm/d)
- Diuretics
- Loop diuretic (Lasix)
- Aldosterone inhibitor (Spironolactone)
  Spironolactone is the best diuretics for ascetic pt: because the cause of their ascites is activation of aldosterone but they will develop hyperkalemia so we will give loop diuretic to decrease potassium

Treatment-Resistant: If general treatment did not succeed

- Recurrent tapping
- TIPS (reduce portal hypertension)
- Liver transplantation

Spontaneous Bacterial Peritonitis

- Infection of ascetic fluid usually gram negative (E.Coli).
- Presentation variable
- Mortality is high
- Dx: ascitic tap = PMN>250 (Fluid culture is the most accurate test)
- Treatment: third generation cephalosporin IV

Note(s):

Spontaneous bacterial peritonitis (SBP)
This represents a serious complication of ascites with cirrhosis and occurs in approximately 8%. The infecting organisms gain access to the peritoneum by haematogenous spread; most are *Escherichia coli, Klebsiella* or enterococci. The condition should be suspected in any patient with ascites who clinically deteriorates. Features such as pain and pyrexia are frequently absent. Diagnostic aspiration should always be performed (see above). A raised neutrophil count in ascites is alone sufficient evidence to start treatment immediately. A third-generation cephalosporin, such as cefotaxime or ceftazidime, is used and is modified on the basis of culture results. Mortality is 10–15%. Recurrence is common (70% within a year) and an oral quinolone, e.g. norfloxacin 400 mg daily, is given for prevention, prolonging the survival. Primary prophylaxis of SBP in patients with ascites protein ‹10 g/L or severe liver disease also prevents hepatorenal syndrome and improves survival. SBP is an indication to refer to a liver transplant centre.

Hepatic Encephalopathy

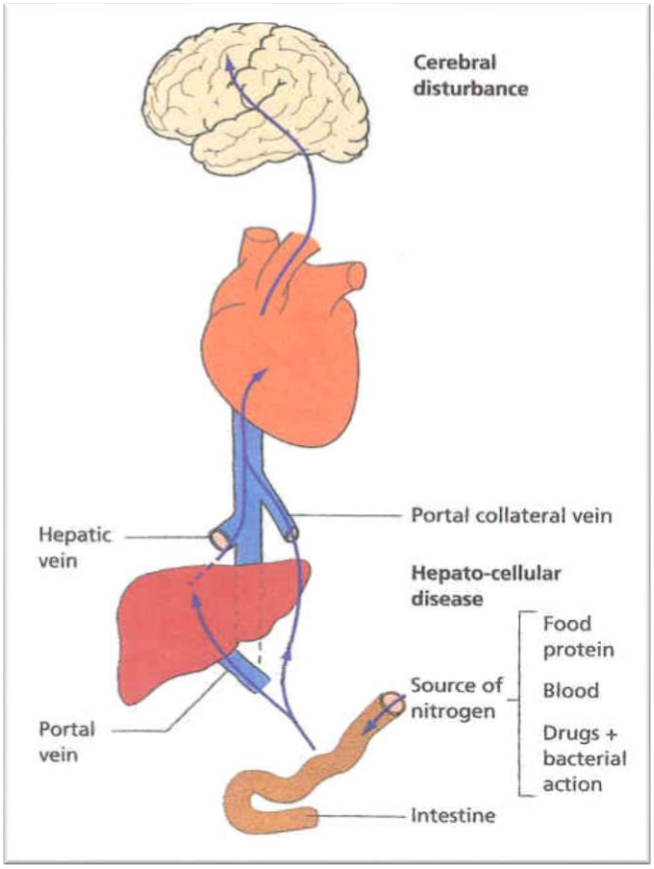
Reversible decrease in neurological function secondary to liver disease

- **Acute:** seen with acute liver failure
- **Acute on chronic:** established cirrhosis

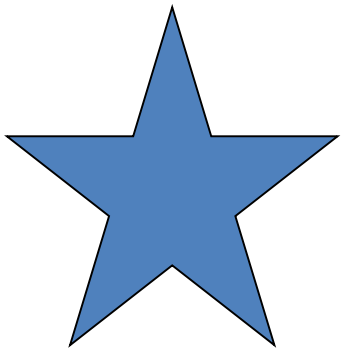
Hepatic Encephalopathy Mechanism: Decrease detoxification by liver causing increase ammonia and urea affect CNS

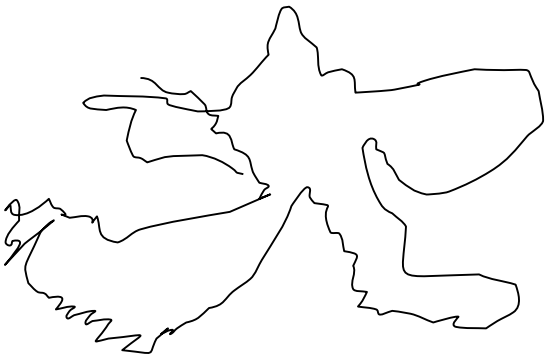
Clinical features:

1. Reversal of sleep pattern
2. Disturbed consciousness
3. Personality changes
4. Intellectual deterioration
5. Fetor hepaticus
6. Astrexis (flapping tremor) which rapid relaxation of flexed wrist because toxin prevent nerve from conducting normaly.
7. Fluctuating



Drawing Tests

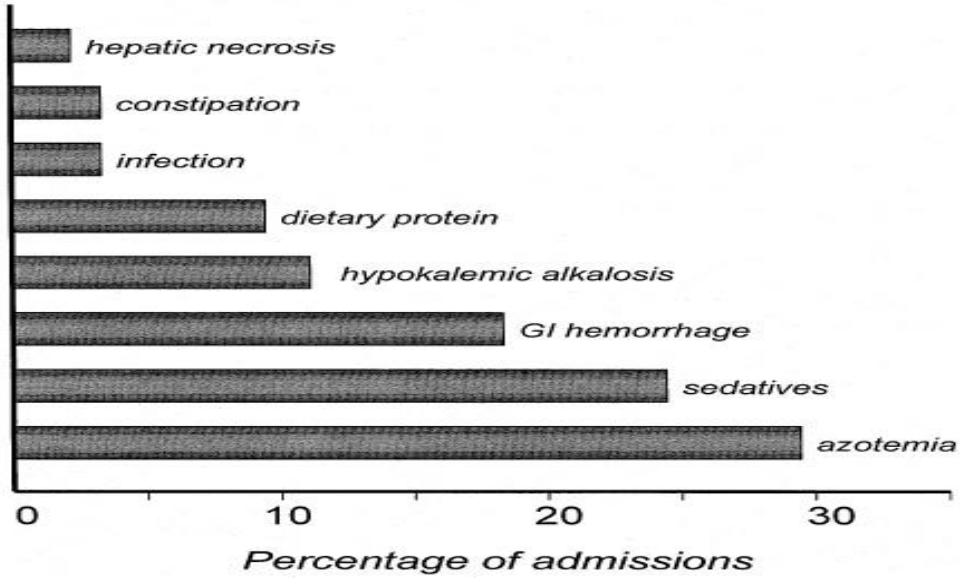




Clinical stage

Table 79–1 | Clinical Stages of Hepatic Encephalopathy

CLINICAL STAGE	INTELLECTUAL FUNCTION	NEUROMUSCULAR FUNCTION
Subclinical	Normal examination, but work or driving may be impaired	Subtle changes on psychometric or number connection tests
Stage 1	Impaired attention, irritability, depression, or personality change	Tremor, incoordination, apraxia
Stage 2	Drowsiness, behavioral changes, poor memory and computation, sleep disorders	Asterixis, slowed or slurred speech, ataxia
Stage 3	Confusion and disorientation, somnolence, amnesia	Hypoactive reflexes, nystagmus, clonus, and muscular rigidity
Stage 4	Stupor and coma	Dilated pupils and decerebrate posturing; oculocephalic reflex; absence of response to stimuli in advanced stages



- Constipation
- Stool has high amount of protein colon bacteria digest that protein produce ammonia
- GI bleeding
- Blood has protein digested by colon bacteria produce ammonia

Hepatic Encephalopathy Treatment

1. Identify and treat precipitation factor
2. Treat underlying liver disease
3. Normal protein diet.
4. Antibiotics (Neomycin, metronidazole). kill colon bacteria
5. Lactolose cause diarrhea so no stool in bowel reduce chance of producing ammonia
6. Transplantation

Hepatocellular Carcinoma

- One of the most common cancers in Saudi Men
- It develops in patients with cirrhosis usually
- Rarely with non-cirrhotic liver, some hepatitis B pateint develop HCC with non- cirrhotic liver
- It preventable because
- Detected by ultrasound and diagnosed by CT or MRI
- Poor prognosis usually discover late
- Every Cirrhotic pt should do HCC screening:
- US, a- phetpprotein every six month
- Multiple treatment modalities

SUMMARY

1. Mechanical compression of blood flow plus hemodynamic changes leads to portal hypertension
2. Common complications of portal hypertension are:
   a) Varices
   b) Ascites
   c) Hepatic encephalopathy
3. The most important step in variceal bleed management is resuscitation.
4. The most important step in management of hepatic encephalopathy is the identification of the precipitating factor.

IMPORTANT NOTES FROM EXTERNAL RESOURCES

	Notes
Step – up to Medicine	**Cirrhosis** **a. General characteristics** . Cirrhosis is a chronic liver disease characterized by fibrosis, disruption of the liver architecture, and widespread nodules in the liver. The fibrous tissue replaces damaged or dead hepatocytes. . Cirrhosis is generally irreversible when advanced. . The distortion of liver anatomy causes two major events: **Decreased blood flow** through the liver with subsequent hypertension in portal circulation **(portal hypertension)**—This has widespread manifestations, includ- ing ascites, peripheral edema, splenomegaly, and varicosity of veins "back stream" in the circulation (e.g., gastric/esophageal varices, hemorrhoids). **Hepatocellular failure** that leads to impairment of **biochemical functions**, such as **decreased albumin synthesis** and **decreased clotting factor synthesis** **B. Causes** . **Alcoholic liver disease—most common cause** a. . **Chronic hepatitis B and C infections—next most common causes**

Questions

1) What is the most common cause of liver cirrhosis?
   a. Hepatitis B
   b. Hepatitis C
   c. Alcohol
   d. Idiopathic

2) Ascites fluid finds to have more than 300 neutrophils, what is the most likely diagnosis?
   a. Inflammation
   b. Spontaneous Bacterial Peritonitis (SBP)
   c. Normal
   d. Non of the above

3) What is the most important step in the initial managing of varices?
   a. 2 normal saline lines
   b. IV vasoconstrictors (Octreotide).
   c. Endoscopy
   d. Banding varics

This lecture's extra notes are taking from: **Master the Board USMLE Step 2 CK by Conrad Fischer**

432 Medicine Team Leaders

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Answers:

1st Questions: C

2nd Questions: B

3rd Questions: A