Team Medicine

Complications of Liver Cirrhosis

Writer: Mohammed Asiri
Reviser: Shatha Altahan
Leader: Alanood Asiri

431 Teams
medical students

■ Important notes ■ Doctor's notes

Complications of Liver Cirrhosis

What is Liver Cirrhosis?

- Diffuse fibrosis of the liver with nodule formation
- Abnormal response of the liver to any chronic injury

Mechanism of Portal HTN

cirrhosis → resistance of blood flow → mechanical nodule and dynamic nitric oxide

liver is cirrhotic and obstructed → blood cannot go through → portal venous system dilatation → collateral and varices open . (varices are abnormal dilated veins with low pressure. in cirrhosis they are susceptible to outflow of high pressure systemic circulation → risk of rupture and causing varecial bleeding)

Complications of Portal Hypertension

1- Varices

Common sites are

- Esophagus
- Gastric
- Colo-rectal
- Portal hypertensive gastropathy

Diagnosis

- History : Hematemesis, melena
- Physical examination
- Ultrasound abdomen
- Endoscopy

Management

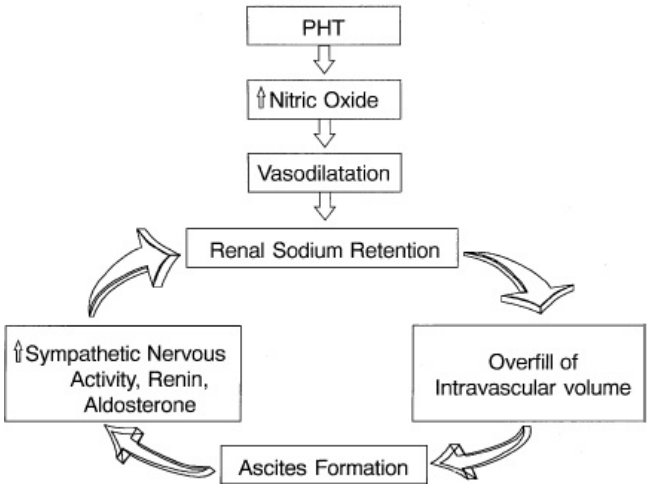
General Management	Specific Management
✓ ABC ✓ 2 IV Lines ✓ Resuscitation ❖ IVF Colloids or crystalloids, as much as the patient needs. ❖ Blood Either give the patient if it's available or cross-match.	IV vasoconstrictors (Octreotide) is B blocker working on visceral circulation Endoscopic therapy ✓ Banding Shunting ✓ Surgical→ Porto-systemic shunt, old-fashioned procedure with high risk of complications. ✓ TIPS → A stent between big portal vein and big hepatic vein

Prevention

- **Treat underlying disease**
- **Endoscopic banding protocol**
- **B-blockers used only in chronic bleeding not acute. It reduces cardiac output and causes vasodilatation which helps in a case of a hyper dynamic circulation.**
- **Liver transplantation**

2- Ascites

- **It is fluid in the peritoneal cavity.**
- **Mechanism of Ascites**



It's the mechanism that accounts for 99% of Ascites.
The body tries to compensate for the portal hypertension by producing Nitric Oxide. Nitric Oxide causes systemic vasodilatation which stimulates the Renal system which in turn, causes fluid retention.

Presentation

History:

- **Increased abdominal girth**
- **Increased weight**

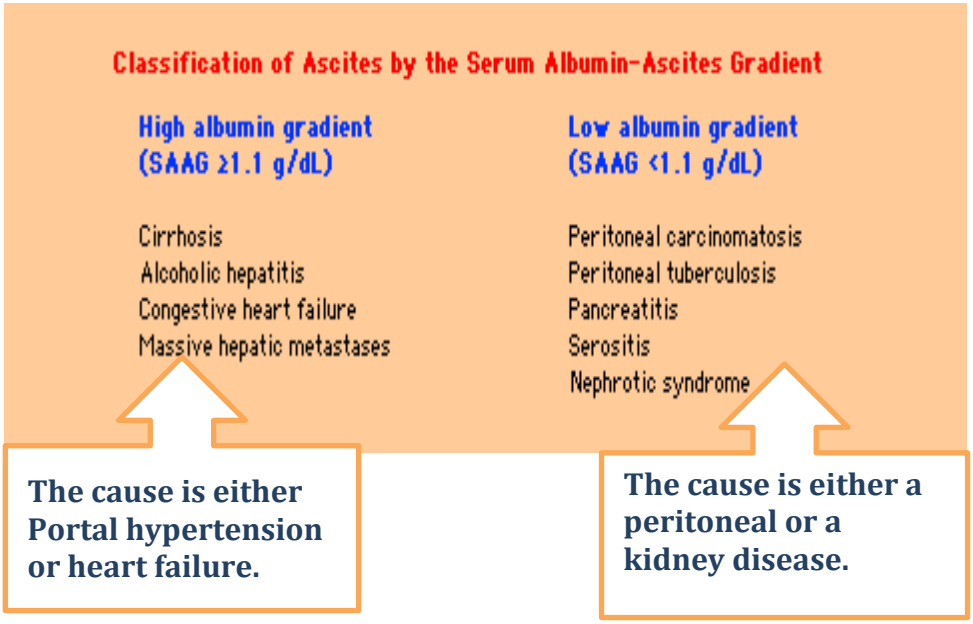
Physical exam:

- **Bulging flanks**
- **Shifting dullness**
- **Fluid wave**

Diagnosis

- **Physical examination**
- **Ultrasound**
- **Ascitic tap**
- **WBC (>250 PMN → Spontaneous Bacterial Peritonitis) Explained later on.**

- **SAAG (serum albumin to ascitic fluid albumin gradient)**
  - ✓ **SAAG = (albumin concentration of serum) - (albumin concentration of ascitic fluid) =**
  - ✓ **>11 mg/dl : portal hypertension (Either a liver or a heart problem)**
  - ✓ **<11 mg/dl : Other (everything else)**
  - ✓ **It is an old method that is rarely used for diagnosis nowadays unless the effective methods like (CT , renal function test , echo) show no significant results. But it usually comes in the exam ☺!**



Treatment

General Treatment	Resistant Treatment
▪ Treat the underlying disease ▪ Salt restriction (<2gm/d) ▪ Diuretics → Most important is loop diuretics. ▪ Loop diuretic (Lasix) ▪ Aldosterone inhibitor (Spironolactone) ▪ In practice we use both to: 1- Increase potency. 2- Reduce electrolytes imbalance.	▪ Recurrent tapping ▪ Peritoneal-venous shunt ▪ TIPS To reduce the portal hypertension. ▪ Liver transplantation

Spontaneous Bacterial Peritonitis (SBP):

- **Infection of the ascitic fluid caused by gram negative (E.Coli).**
- **Presentation variable (usually patients are asymptomatic) and Mortality is 40% ,when treated → 0%**
- **Dx: Ascitic tap CBC: if PMN(Polymorphic nuclear cells)>250 = SBP, and it is treated by third generation cephalosporin IV**

3- Hepatic Encephalopathy

- Reversible decrease in neurological function secondary to a liver disease
- Acute: seen with acute liver failure
- Acute on chronic: established cirrhosis

Hepatic Encephalopathy Mechanism

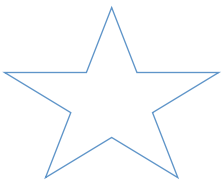
If the liver function of detoxifying is lost due to liver failure or proto-systemic shunting of blood → accumulation of neurotoxins -mainly nitrogenous substances- in the brain causing Encephalopathy.

Clinical features

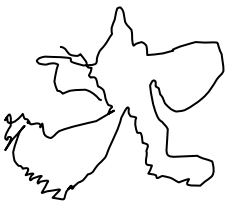
- ❖ Reversal of sleep pattern.
- ❖ Disturbed consciousness.
- ❖ Personality changes.
- ❖ Flapping Tremors.
- ❖ First, intellectual deterioration.
- ❖ Then, fluctuating consciousness.
- ❖ And finally, Coma.

Diagnosis

By recognizing the clinical features and performing the drawing test:

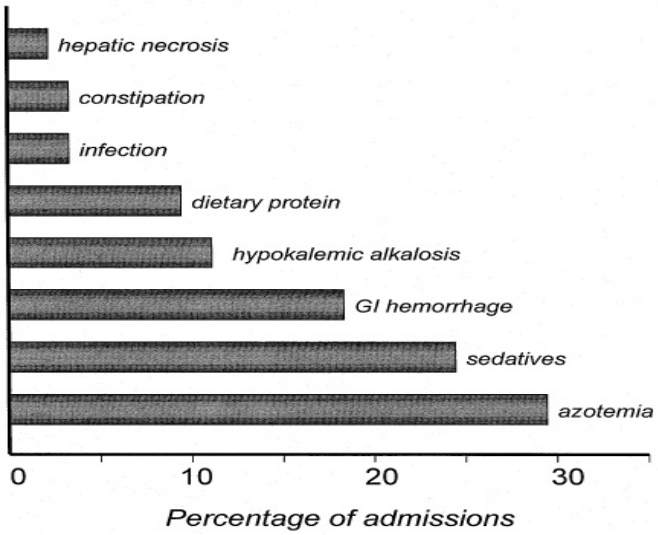


Negative Encephalopathy



Positive Encephalopathy

Exacerbating or precipitating factors



TIPS is considered a precipitating factor due to the shunt that bypasses the liver. That is why 10% of the patients develop encephalopathy after the procedure and sometimes end up in a coma. Therefore, they are always given prophylactic treatment.

Treatment

- ❖ Identify and treat precipitation factors.
- ❖ Treat underlying liver disease.
- ❖ Normal protein diet.
- ❖ Antibiotics (Neomycin, metronidazole)
- ❖ Lactolose.
- ❖ Transplantation.

4- Hepatocellular Carcinoma

- **One of the most common cancers in Saudi Men and develops in patients with cirrhosis usually. Detected by ultrasound and diagnosed by CT or MRI.**
- **Poor prognosis (we should perform ultrasound screening every 6 months for patients suffering from liver cirrhosis).**
- **Multiple treatment modalities.**

Summary

- **Mechanical compression of blood flow plus hemodynamic changes leads to portal hypertension.**
- **Common complications of portal hypertension are:**
  - ✓ **Collateral formation (Varices) – Early complication.**
  - ✓ **Ascites – Middle complication.**
  - ✓ **Hepatic encephalopathy – End stage complication.**
- **The most important step in variceal bleed management is resuscitation.**
- **The most important step in management of hepatic encephalopathy is the identification of the precipitating factors.**