COMPLICATIONS OF LIVER CIRRHOSIS

● Portal Hypertension ○ Gastroesophageal varices ○ Portal hypertensive gastropathy ○ Splenomegaly,Hypersplenism ○ Ascites ○ SBP ● Hepatic Encephalopathy ● Hepato Renal Syndrome (HRS) ● Hepatopulmonary Syndrome ● Malnutrition	● Coagulopathy ● Bone disease ○ Osteopenia ○ Osteoporosis ○ Osteomalacia ● Hematologic abnormalities ○ Anemia ○ Hemolysis ○ Thrombocytpenia ○ Neutropenia

Varices and Portal Hypertension

- β-Blockers are standard of care for people with large varices or prior bleeding.
- Nonselective β-blockers (eg, carvedilol or propranolol) reduce portal pressure by reducing splanchnic blood flow. Because of its α-blocking effects, carvedilol also reduces intrahepatic resistance.
- Large varices are encountered on endoscopy, carvedilol (optimally dosed at 12.5 mg daily) is preferred to other β-blockers.

- Variceal bleeding should be treated with band ligation during timely endoscopy (<24 hours after presentation), vasoactive medications (compared with placebo, octreotide was associated with higher rates of hemostasis at 5 days [77% vs 58%] in a meta-analysis of randomized trials),and prophylactic antibiotics (associated with reduced short-term mortality to 18.5% vs 22.2% with placebo in a meta-analysis of randomized trials).

- In a randomized trial of 63 patients with acute variceal bleeding who achieved initial hemostasis, transjugular intrahepatic portosystemic shunt (TIPS, a stent placed in a tract created to connect branches of the hepatic and portal veins) performed within 72 hours (compared with no TIPS placement) improved 1-year survival (61% vs 86%).

HRS

- Hepatorenal syndrome is a form of functional renal failure without renal pathology that occurs in about 10% of patients with advanced cirrhosis or acute liver failure.

- Condition is marked with increased renal vascular resistance (renal vasoconstriction) and accompanied by a reduction in systemic vascular resistance. The reason for renal vascular resistance is unknown.

- It is diagnosed when a person presents with ascites with progressive rise in creatinine.

- Type 1 HRS is characterized by a progressive impairment in renal function and significant reduction in creatinine clearance within 1-2 weeks.

- Type 2 HRS is characterized by a reduction in glomerular filtration rate with an elevation of serum creatinine level, but it is fairly stable and is associated with a better outcome than type1.

- Currently patients are treated with albumin & terlipressin.

- In an RCT of 300 patients, compared with placebo, terlipressin improved kidney function (creatinine ≤1.5 mg/dL; 39% vs 18%) but was associated with an increased risk of death due to respiratory failure (11% vs 2% with placebo). In a meta-analysis, norepinephrine, 0.5 to 3 mg/h, was noninferior to terlipressin with a 50% pooled rate of kidney injury reversal.

Spontaneous Bacterial Peritonitis

- SBP is a common and severe complication of ascites characterised by spontaneous infection of the ascitic fluid without an intrabdominal source .
- Bacterial translocation is presumed mechanism for development of SBP with gut flora traversing the intestine into mesentric lymph node, leading to bacteremia and seeding of the ascitic fluid.
- Common organisms are E.Coli, gram positive bacteria including streptococcus viridans, staphylococcus aureus and enterococccus species .

- Patients present with fever, altered sensorium, elevated white blood count and abdominal pain or discomfort.
- Absolute neutrophil count >250/mcl is considered diagnostic.

- Cefotaxime is the recommended agent.
- After a first episode of spontaneous bacterial peritonitis, patients should receive secondary prophylaxis with suppressive oral antibiotics (eg, trimethoprim/sulfamethoxazole or ciprofloxacin).

Splenomegaly & Hypersplenism

- Congestive splenomegaly is common in patients with portal hypertension.
- Clinical features include enlarged spleen , thrombocytopenia and leukopenia.

Ascites

- Ascites is the accumulation of fluid within the peritoneal cavity.

- In patients with ascites,there is an increase in intrahepatic resistance, causing increase in portal pressure, but there is also vasodilation of splanchic arterial system due substances like NO, which results in increased portal venous flow.
- Both leads to increased production of splanchic lymph .

- Hemodynamic changes leads to sodium retention by causing activation of RAAS with the development of hyperaldosteronism.
- Hypoalbuminemia and reduced plasma oncotic pressure also contributes to the loss of fluid from the vascular compartment into the peritoneal cavity.

SAAG (Serum ascites to albumin gradient)

- In patients with cirrhosis, the protein concentration of ascitic fluid is low.
- SAAG is >1.1 g/dl, the cause of ascites is most likely due to portal hypertension.
- SAAG < 1.1 g/dl, infectious or malignant causes should be considered.

Treatment

- Salt restriction <2 gm/day
- Sprinolactone 100 -200 mg/day is started and may be raised upto 400 – 600mg/day , furosemide 40 -80 mg/d may be also added.

- Paracentesis is associated with temporary relief for patients with symptomatic ascites.
- Multiple paracenteses, despite attempts to optimize diuretic dosage, should prompt referral for TIPS.

- In a meta-analysis of 305 patients in randomized trials, compared with treatment without TIPS, TIPS was associated with reduced risk of recurrent ascites (42% vs 89%) and reduced 2-year mortality (51% vs 65%), but more hepatic encephalopathy episodes per year.

Hepatopulmonary Syndrome

- This is characterised by refractory hypoxemia, intrapulmonary vascular dilatation and chronic liver disease with portal hypertension.
- HPS causes digital clubbing, cyanosis, spider navei and a characteristic reduction in arterial oxygen saturation on standing.
- The hypoxia is due to intrapulmonary shunting through direct arteriovenous communications.
- It is believed to be a result of overproduction of nitric oxide.

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Reference

- Harrison
- Davidson
- https://edhub.ama-assn.org/jn-learning/module/2804599