Manage the complication of liver cirrhosis

By

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Liver Blood Supply

- 75% blood supply by portal and hepatic veins
- 25% by hepatic arteries
Portal Vein

- Portal vein is a collection of superior and inferior mesenteric veins andsplenic vein
- Portal pressure is around 10 to 12 mmHg
Blood flow inside the liver parenchyma tissue

- Hepatocytes function as detoxification of toxic substances and synthesizer of proteins, carbohydrates, hormones and related materials
- Kupffer Cell act as phagocytic cell
Bile canalculus and Bile Duct

- Bile is secreted by hepatocytes and it helps absorb fat and fat-soluble vitamins from the gut.
- Bile salt is a corrosive agent.
- Can damage hepatocytes if not drained properly.
Healthy Liver
Definition:

Cirrhosis of the liver is the result of various disease processes and is characterized by diffuse fibrosis and conversion of the normal liver architecture into structurally abnormal nodules.
Definitions

**Fibrosis**
- Excess deposition of the components of extracellular matrix (collagens, glycoproteins, proteoglycans) within the liver
- Reversible process

**Cirrhosis**
- Diffuse hepatic process characterized by fibrosis and conversion of normal liver architecture into structurally abnormal nodules
- Irreversible process
The cirrhotic liver on the left shows the typical nodular appearance. The nodules are separated by fibrous bands. The spleen on the right is huge due to portal hypertension.
Etiology

Most common causes
- Hepatitis (26%)
- Alcoholic liver disease (21%)
- Hepatitis C+ alcoholic disease (15%)

Miscellaneous (5%)
- Autoimmune hepatitis
- Primary biliary cirrhosis
- Secondary biliary cirrhosis
- Primary sclerosing cholangitis
- Hemochromatosis
- Wilson disease
- Alpha-1 antitrypsin deficiency
- Drug induced
- Venous outflow obstruction (budd-chiari syndrome)
Pathophysiology

- Prehepatic
  1. Portal vein thrombosis
  2. Shistosomiasis

- Hepatic
  1. Hepatitis B, C, Autoimmune
  2. Alcohol

- Posthepatic
  - Venous outflow obstruction (budd-chiari syndrome)
  - Chronic right side heart failure
Sign and Symptoms and clinical finding of Liver Cirrhosis

- Anorexia, nausea, abdominal discomfort, weight loss, and malaise
- Ascites, peripheral edema, jaundice, spider nevi, palmar erythema
- Gynecomastia, testicle atrophy, amenorrhea, pubical hair lost
- Hepatomegaly, spleenomegaly, encephalopathy, and bleeding
Laboratory Findings

- Initially elevated ALT and AST level but at the end stage they can be normal or below normal
- Elevated bilirubin most of the time
- Low albumin level
- Prolong prothrombin time (PT) and APTT
- Elevated serum creatinine and blood urea nitrogen (BUN)
## Classification

### Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Points assigned to laboratory values and signs*</th>
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<tbody>
<tr>
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<tr>
<td><strong>laboratory value</strong></td>
<td>&lt;2 mg per dL (34 μmol per L)</td>
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<td><strong>serum bilirubin level</strong></td>
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<td><strong>Serum albumin level</strong></td>
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<td>Non</td>
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<td><strong>Encephalopathy</strong></td>
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*Based on total points, a patient with cirrhosis is assigned to one of three classes: Child class A = 5 to 6 points; Child class B = 7 to 9 points; Child class C = 10 to 15 points.*

Gastroenterology 2001;120:727
Complications of Cirrhosis

- **Gastrointestinal:**
  - **Ascites**
  - **Bleeding varices**
    - (esophageal, gastric, ectopic)
  - **Portal hypertensive gastropathy (PHG)**

- **Central nervous system:**
  - **Hepatic Encephalopathy (HE)**

- **Renal:**
  - **Hepatorenal syndrome (HRS)**

- **Pulmonary:**
  - **Hepatic Hydrothorax**
    - Hepatopulmonary syndrome.
    - portopulmonary syndrome
Complications

- Portal hypertension
- Variceal Hemorrhage
- Ascites
- Spontaneous Bacterial Peritonitis
- Hepatic Encephalopathy
- Hepatorenal Syndrome
Treatment Recommendations - Cirrhosis

Compensated

- Hepatoma Surveillance
  - U/S, AFP q 6 months
- Varices Surveillance
- Vaccination against HAV, HBV

Decompensated

- Monitor Liver Function
  - PT, Alb, Bili q 3-6 months

- Variceal Bleed
- SBP
- Ascites
- Hepatorenal Synd.
- Encephalopathy
Risks of Complications of Cirrhosis

Cirrhosis

- Variceal Bleeding: 1.1% per year
- Ascites: 2.5%
- HCC: 1.5%
- Encephalopathy: 0.4%

Death: 11%

Liver Transplant: ≥20%+

Bennett WG et al, Ann Intern Med 1997;127:855
Portal Hypertension

- Portal pressure increases to 5 mmHg more than the pressure in the inferior vena cava
- Development of varices and alternative routes of blood flow
- Risk of varices when portal pressure exceed the vena cava pressure by \( \geq 12 \) mmHg
- Hemorrhage from varices occurs in 25-40% of cirrhotic patients
- Each episode of bleeding carries a 30% risk of death
How Varices form

- Portal hypertension causes blood flow to be forced backward, causing veins to enlarge and varices to develop across the esophagus and stomach from the pressure in the portal vein. The backup of pressure also causes the spleen to become enlarged.
Esophageal Varices
ESOPHAGEAL VARICEAL BLEEDING

- **Primary prophylaxis,**
  - Discontinue alcohol consumption

- **Pharmacotherapy:**
  - Non selective β-Blocker (Propranolol)
    - ↓Portal pressure by ↓CO → ↓ Blood flow.
    - Long acting beta blocker (Nadolol) or propanolol average dose of (60 mg/d), preferred in the evening, mean dose 160 mg/d
    - HR not less than 55 beats/min & SBP not less than 90 mm Hg.
    - Adverse effects in 27% of patients. What are they?
ESOPHAGEAL VARICEAL BLEEDING, Cont’d

- Nonselective β-blocker (Nadolol):
  - Less lipophilic and does not cross the BBB.
  - Less adv. Effects (therapy stopped in 4%)
  - 20-240 mg/d

- Long acting nitrates + Nonselective β-blockers has been shown to enhance their effect and ↓ the risk of bleeding.
  - Should be considered in patients who do not respond to β-Blockers.
ESOPHAGEAL VARICEAL BLEEDING, Cont’d

- **Endoscopic therapy:**
  - Endoscopic variceal sclerotherapy is not recommended in the primary prophylaxis of variceal bleeding.
  - Endoscopic variceal band ligation (EVL) was shown to be safe and more effective than propranolol in the primary prophylaxis of variceal bleeding.
  - Further confirmation is needed.
Prophylaxis of Variceal Hemorrhage

Diagnosis of Cirrhosis

Endoscopy

- No varices
- Small varices
- Medium/large varices

Follow-up endoscopy in 2-3 years

- Stepwise increase until maximum tolerated dose
- Continue beta-blockers lifelong

No CI

Beta-blocker therapy

CI or intolerance

Endoscopic variceal band ligation
Acute variceal Bleeding:

- Suspected patients should be managed in an ICU.
- Adequate venous access should be established.
- Replenishment of blood volume and correction of coagulopathy must be done with packed erythrocytes (to increase Hb concentration to 10 g/dL) and fresh frozen plasma, (do not over expand the plasma volume).
Acute variceal Bleeding, Cont’d

- Air way must be protected by endotracheal intubation.
- Antibiotics such as Norfloxacin should be administered at a dose of 400 mg BD to prevent SBP and G-ve systemic infection.
- Emergency endoscopy of the UGIT must be performed in all patients suspected of having a variceal hemorrhage.
- Choose the proper modality for each patient.
Acute variceal Bleeding, Cont’d

Pharmacotherapy:
– While waiting for endoscopy.

1. Vasopressin: 0.2-0.9 IU/min IV infusion for maximum of 24 to 48 hours
   – Associated with major complications such as angina, arrythmia, MI, bowel, liver and spleen infarction and local tissue necrosis and CVA
   – No longer recommended
   – The combination of vasopressin with nitroglycerin → more effective & reduced the cardiac complications.
Acute variceal Bleeding, Cont’d

2. Terlipressin:
   - Analogue of vasopressin.
   - Has a longer half-life than vasopressin and can be given as a bolus infusion q4h.
   - As effective as vasopressin with less SE.
   - Dose: 2 mg IV 6 hourly till bleeding stops and then 1 mg IV 6 hourly for
Acute variceal Bleeding, Cont’d

- Pharmacotherapy, Cont’d

3. Somatostatin
   - Has been shown to be superior to vasopressin and comparable to terlipressin, balloon tamponade, and endoscopic variceal sclerotherapy.
   - Well tolerated and has few adverse effects
   - Initial IV bolus of 250 mcg followed by continuous IV infusion of 250-500 mcg/h.
Acute variceal Bleeding, Cont’d

- Pharmacotherapy, Cont’d

4. Octreotide:
   - A synthetic analogue of somatostatin.
   - Widely used to control acute variceal hemorrhage.
   - Has been shown to be effective in controlling bleeding and in decreasing bleeding related mortality.
   - Bolus dose of 50 mcg and continuous IV infusion of 50 mcg/h for 5 days.
Acute variceal Bleeding, Cont’d

Endoscopic Therapy
- Endoscopic injection of hypertonic solution (sclerotherapy) controls active hemorrhage from esophageal varices in about 90% of patients.
- Endoscopic variceal band ligation (EVL) was shown to be comparable to endoscopic sclerotherapy with less complications.
- EVL is currently the initial procedure of choice.
- Complications of EVL include
  - Superfacial ulceration and dysphagia
  - Transient chest discomfort and rarely esophageal strictures
Esophageal varice
Sclerotherapy

Microsclerotherapy is a technique used for the removal of surface and spider veins. It involves injections with a very fine needle of a sclerosing agent which has an irritant effect on the lining of the veins causing the walls of the veins to stick together. Blood stops flowing through the veins, which are then absorbed by the body's natural defence mechanisms over a period of three months. The blood is then directed back to the deeper venous system.
Acute variceal Bleeding, Cont’d

- Other modalities
  - Balloon tamponade
  - Cold ice water lavage
  - Surgery
  - Transjugular intrahepatic portosystemic shunt (TIPS)
TIPS

- **After the TIPS procedure is performed.** A radiologist makes a tunnel through the liver with a needle, connecting the portal vein to one of the hepatic veins. A metal stent is placed in this tunnel to keep the track open.

- The shunt allows the blood to flow normally through the liver to the hepatic vein. This reduces portal hypertension, and allows the veins to shrink to normal size, helping to stop variceal bleeding.
Prevention of Rebleeding:

- Occurs in about 75% of pts. within 6 weeks of the initial episode.
- EVL is the preferred therapeutic method at weekly or twice weekly intervals until the esophageal varices are obliterated.
- Then endoscopy is performed at 3 months and then every 6 to 12 months thereafter.
- The addition of propranolol may enhance benefit.
Secondary Prophylaxis

- Lowest rebleeding rates are obtained in HVPG responders & with ligation + beta-blockers:

Ascites

- Accumulation of excessive fluid within the peritoneal cavity
- Ascites is the most common of the major complications of cirrhosis.
- 50% of patients with compensated cirrhosis will develop ascites during 10 years of observation
- Two year mortality is 50%
- Six months mortality is 50% in patient with diuretics resistant ascites
- Treatment of ascites doesn't improve mortality
Pathogenesis

CIRRHOSIS

↑ Intrahepatic resistance to portal flow

↑ SINUSODIAL PRESSURE

↑ PORTAL PRESSURE GRADIENT

↑ Portal blood inflow

↓ Portal blood inflow

↑ Portal blood inflow

↓ Arteriolar resistance

Splanchnic

Systemic

Effective arterial blood volume

Activation of neurohumoral systems

Sodium and water retention

ASCITES

COLLATERALS (VARICES)

Sodium and water retention

Gastroenterology 2001;120:727
Ascites

- Ascites results from renal retention of salt and water with localization of this excess fluid into the peritoneal cavity due to portal hypertension.

- Treatment of ascites is therefore aimed at creating a –ve sodium and water balance and, if this strategy is inadequate, at decreasing portal pressure by portosystemic shunting.
How Ascites Form

1. Increased hepatic resistance
2. Portal hypertension
3. Portosystemic collaterals
4. Vasodilation, hyperdynamic state (low SVR, Underfill physiology)
5. Activation of compensatory pathways (e.g., renin-angiotensin)
   (sodium retention, volume expansion, Overflow physiology)
6. Augmented portal flow
7. Excess hepatic lymph exceeds capacity to be removed
   (most fluid forms from the surface of the liver)
8. Ascites become apparent
   (when the capacity of the peritoneal lymphatics is exceeded)
Management of Ascites

1. Sodium restriction

2. Treat underlying disease (fluid restriction if hyponatremic)

3. Diuretic therapy (usually spironolactone +/- loop diuretic)

4. Large volume paracentesis (review medications and salt compliance)

5. Consider transplant evaluation depending on response and co-morbid conditions

6. Assess recurrence, malnutrition, and cardiopulmonary function

TIPS or other portal decompression in selected patients

Consider indwelling drain in those with very short life expectancy (weeks)

Peritoneovenous shunt in selected patients (usually in those not felt to be TIPS candidates)
Diagnosis

- **Physical Exam**
  - Shifting dullness
  - Flank dullness
  - Puddle sign

- **Imaging**
  - Ultrasound

- **Paracentesis**
  - Cell count
  - Culture
  - Ascites albumin

- **Serum-Ascites Albumin Gradient (SAAG)**
  - SAAG = (Serum Alb - Ascites Alb)
  - > 1.1 g/dL => Portal Hypertension
  - < 1.1 g/dL => Other
    - Peritoneal carcinomatosis (ovarian), TB, pancreatitis
Management

- Goals of therapy
  - To mobilize ascitic fluid
  - To diminish abdominal discomfort, back pain, and difficulty in ambulation
  - To prevent major complications
Management Cont,

- Nonpharmacological management
  - Na$^+$ Restriction
  - Therapeutic paracentesis
  - Peritoneovenous shunt
  - TIPS

- Pharmacological management
  - Diuretics
    - Aldactone (Aldosterone antagonist)
    - Furosemide (Loop diuretic)
Non-diuretics Therapy

- Sodium and water restriction
  - Sodium output must exceed sodium input
  - Eliminate ascites in 10-20% of patients with
    - Urinary sodium excretion > 50 mmol/d
    - Mild to moderate ascites
  - No complications associated with dietary Na restriction
  - Dietary sodium intake is restricted to 88 mmol/day
  - The goal of treatment to increase urinary sodium excretion to > 78 mmol/day
  - Only 10–15% of patients spontaneously excrete > 78 mmol/day.
ASCITES, Cont’d

- General measures:
  - Bed rest is advisable for patients with a large amount of ascites.
  - Dietary sodium is restricted to 2 gm/d if necessary.
  - Fluids are restricted to 1500 mL/d but in the presence of dilutional hyponatremia (serum sodium level <120 mEq/L) fluids are limited to less than 1000 mL/d.
Diuretics Therapy

- **Choice of agent**
  - High level of circulating aldosterone
    - Decrease execration and increase production
    - Activation of RAS
    - Hepatic impairment prolongs the half life of aldosterone
    - Low concentration of albumin
  - Spironolactone is rational choice
  - Dose 100 mg to 200 mg up to 400 mg
  - Combination with other diuretics
  - Spironolactone to furosemide ratio (100-40 mg)
ASCITES, Cont’d

Diuretic therapy:

- The choice of diuretic therapy depends on the urinary sodium levels.
- Urinary Na level >30 mEq/L, use spironolactone alone (100-400 mg/d)
- Urinary Na level is 10-30 mEq/L, use a combination of furosemide and spironolactone in a ratio of 40 mg and 100 mg
- Common complications of diuretic therapy include electrolyte imbalances (hyponatremia, hypo and hyperkalemia), hepatic encephalopathy, renal impairment, gynecomastia, and muscle cramps.
Diuretics Therapy

- Monitoring
  - Clinical response
    - Body Weight, urine output, and abdominal girth
    - Goal Weight loss of 0.5-2 kg/day (0.5-2L/day)
    - Urine output exceeds input by 300 to 1000 ml/day
    - Aggressive diuresis if peripheral edema presents (2kg/day)
  - Laboratory parameters
    - Serum Cr
    - Urinary chemistries (Na and K ratio)

- Complications
  - Hypokalemic-hypochloremic metabolic alkalosis and hyponatremia
  - Prerenal azotemia
ASCITES, Cont’d

- Diuretic therapy, Cont’d
  - If the urinary sodium level is <10 mEq/L, large-volume paracentesis is needed in addition to diuretic therapy or for quick relieve.
  - Target weight loss should be 1 kg/d in patients with peripheral edema and 0.5 kg/d in those without peripheral edema.
Large Volume Paracentesis

- **Indications**
  - Patients with cirrhosis experiencing respiratory or cardiac symptoms

- **Procedure**
  - Removal of ascitic fluid from the abdominal cavity with a needle or a catheter
  - It is not a definitive treatment

- **Complications**
  - Hypotension, oliguria, shock, encephalopathy, hepatorenal syndrome
  - Hemorrhage, perforation, infection protein depletion
Large Volume Paracentesis

Use of Albumin

- **Rational**
  - High incidence of paracentesis induced circulatory dysfunction
  - Reduction of COP worsening renal function

- **Dose**
  - 50 ml of a 25% albumin solution per liter of ascites removed (8 grams per every liter remove)
Refractory Ascites

- Failure to lose 200g of weight in a patient despite
  - Severe Na restriction (50 meq/day)
  - Maximal doses of diuretic

- 50% 6-month mortality
HEPATORENAL SYNDROME (HRS):

- A state of functional renal failure in patients with end-stage liver disease.
- Characterized by an increased creatinine level, relatively hyperosmolar urine, and urinary sodium excretion of <10 mEq/L.
- The probability of occurrence of HRS in Pts with cirrhosis is 18%-39%
- The prognosis for HRS is extremely poor
- The only proven treatment is liver transplantation
- Correct hypovolemia and avoid use of all nephrotoxic agents.
SPONTANEOUS BACTERIAL PERITONITIS (SBP)

- In hospitalized patients the prevalence of SBP range between 10% and 30%
- A neutrophil count greater than 250/mm in the absence of an intra-abdominal source of infection in the ascitic fluid is suggestive of SBP and should prompt administration of an antibiotic.
SPONTANEOUS BACTERIAL PERITONITIS (SBP), Cont’d

- >92% of all cases of SBP are monomicrobial, with
  - Escheria coli (the most common isolate)
  - Klebsiella species
  - Other G-ve bacteria
  - G+ve organisms, Streptococcal (25%)
  - Anaerobic infection (rare <5%)
- As with polymicrobial bacteria or fungi, consider secondary bacterial peritonitis.
SPONTANEOUS BACTERIAL PERITONITIS (SBP), Cont’d

- Cefotaxime (2 g IV 8 hourly for 5-7 days) or Ceftriaxone (1-2 gm IV once daily for 5-7 days)
- Oral Ofloxacin (400 mg PO 12 hourly) has shown to be as effective as Cefotaxime in the treatment of uncomplicated SBP.
- Antibiotic therapy should be further modified based on the results of ascitic fluid C/S and antibiotic sensitivities of the isolated organisms
- Secondary prophylaxis with oral Norfloxacin (400 mg/d) may be indicated in patients after more than a few episode of SBP.
Secondary peritonitis from gut perforation should be considered in a patient with neutrocytic ascites with polymicrobial bacteria.
SPONTANEOUS BACTERIAL PERITONITIS (SBP), Cont’d

- 3 Criteria for secondary peritonitis:
  - The initial ascitic fluid is neutrocytic and fulfils 2 of the following criteria:
    i. Total protein > 1 g/dl
    ii. Glucose < 50 mg/dl
    iii. Lactate dehydrogenase > the upper limit of normal for serum.
  - In addition, a leukocyte > 10 x 10/L and the presence of multiple organisms
Hepatic encephalopathy is a neuropsychiatry syndrome caused by liver disease. It occurs most often in patients with cirrhosis but is seen in acute form in acute hepatic failure.
HEPATIC ENCEPHALOPATHY, Cont’d

- Factors precipitating HE:
  - Uremia
    - Spontaneous, diuretic induced
  - Drugs
    - Sedatives, antidepressants, hypnotics
  - Gastrointestinal bleeding
  - Excess dietary protein
  - Constipation
  - Electrolytes imbalance
HEPATIC ENCEPHALOPATHY

Factors precipitating HE, Cont’d

- Paracentesis (volumes > 3-5 liters)
- Hypokalaemia
- Infections
- Trauma (including surgery)
- Portalsystemic shunts
  - Surgical, spontaneous (large)
HEPATIC ENCEPHALOPATHY, Cont’d

- Differential diagnosis of HE:
  - Subdural haematoma
  - Drug or alcohol intoxication
  - Delirium tremens
  - Wernicke’s encephalopathy
  - Primary psychiatric disorders
  - Hypoglycemia
  - Neurological Wilson’s disease
HEPATIC ENCEPHALOPATHY, Cont’d

Management:

- Episodes of encephalopathy are common in cirrhosis.
- The principles are
  - To treat or remove precipitating causes
  - To reduce or eliminate protein intake and
  - To suppress production of neurotoxins by bacteria in the bowel
HEPATIC ENCEPHALOPATHY, Cont’d

- Management, Cont’d
  - Dietary protein is eliminated or reduced below 20 g/d
  - Glucose (300 g/d) is given orally or parenterally in severe cases
  - As encephalopathy improves, dietary protein is increased by 10-20 g/d every 48 hours to an intake of 40-60 g/d
HEPATIC ENCEPHALOPATHY, Cont’d

- Management, Cont’d
  - Lactulose (15-30 ml 8-hourly)
    - Disaccharide which is taken orally and reach the colon intact
    - Metabolized by colonic bacteria
    - It produce an osmotic laxative effect
    - It ↓ the pH of the colonic contents
    - Limits colonic ammonia absorption and
    - Prevent the incorporation of nitrogen into bacteria
HEPATIC ENCEPHALOPATHY, Cont’d

Management, Cont’d

- **Lactitol**
  - A rather alternative to lactulose
  - With less explosive action on bowel function

- **Neomycin (1-4 g 4-6-hourly)**
  - An antibiotic which acts by reducing the bacterial flora content of the bowel
  - Alternative to lactulose in case of diarrhea
  - Poorly absorbed antibiotic
  - Can cause ototoxicity,
  - Not used very often
PULMONARY MANIFESTATIONS

- Hepatic hydrothorax
  - In about 13% of patients with cirrhosis
  - Fluid in the pleural space can be detected on chest radiograph (usually Rt sided 66%)
  - Management options include:
    - Medical control of ascites
    - Therapeutic thoracentesis for relieve of dyspnea
    - TIPS
  - Chest tube drainage is not advised unless the pleural fluid becomes infected (SBEmpyema)
  - Definitive treatment of refractory hydrothorax in the setting of CLD is liver transplantation.
Hepatopulmonary Syndrome:

- Characterized by pulmonary vascular dilatation and hypoxemia (Pao2 <70 mm Hg) in the setting of advanced liver disease
- Orthodeoxia (worsening hypoxemia when the patient stand) is characteristically seen
- Therapeutic options are limited, and only successful liver transplantation has been shown to reverse the hypoxemia in these patients.
Portopulmonary hypertension:
- An increase in pulmonary artery pressures occurs in 20% of patients with advanced liver disease.
- Intravenous prostacyclin has been shown to improve pulmonary hemodynamics in some patients.
- Liver transplantation has been successful in patients with mild pulmonary hypertension, but mortality rates are high in moderate to severe cases.
NUTRITIONAL DISORDERS

- Protein-energy malnutrition is highly prevalent in hospitalized patients with advanced cirrhosis.
- Other nutritional deficiencies of dietary components such as
  - Vitamins
  - Trace elements
  - Polyunsaturated fatty acids
- Nutritional therapy should be considered in the management of patients with cirrhosis, and some data suggest that enteral nutrition may favorably influence short-term survival.
Thyroid disorders:
- Low thyroxine levels tend to be seen only in the later stages of the illness.
- Patients with primary biliary cirrhosis and autoimmune hepatitis should be carefully monitored for the development of hypothyroidism due to Hashimoto thyroiditis.
Sexual dysfunction:
- Gonadal dysfunction commonly accompanies end-stage liver disease.
- Manifestations may include sexual dysfunction and abnormalities of the hypothalamic-pituitary-gonadal axis.
- Management of sexual dysfunction is challenging, and no treatment has been shown to be of benefit thus far.
- Studies have shown an improvement in sexual function after liver transplantation, but the exact mechanism for this remains unclear.
CONCLUSION

- Although effective therapy is available for most of the complications of cirrhosis, liver transplantation is the only treatment modality.
THANKS