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College of Medicine
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Title: Cirrhosis

Grade: Fourth

Module: GIT

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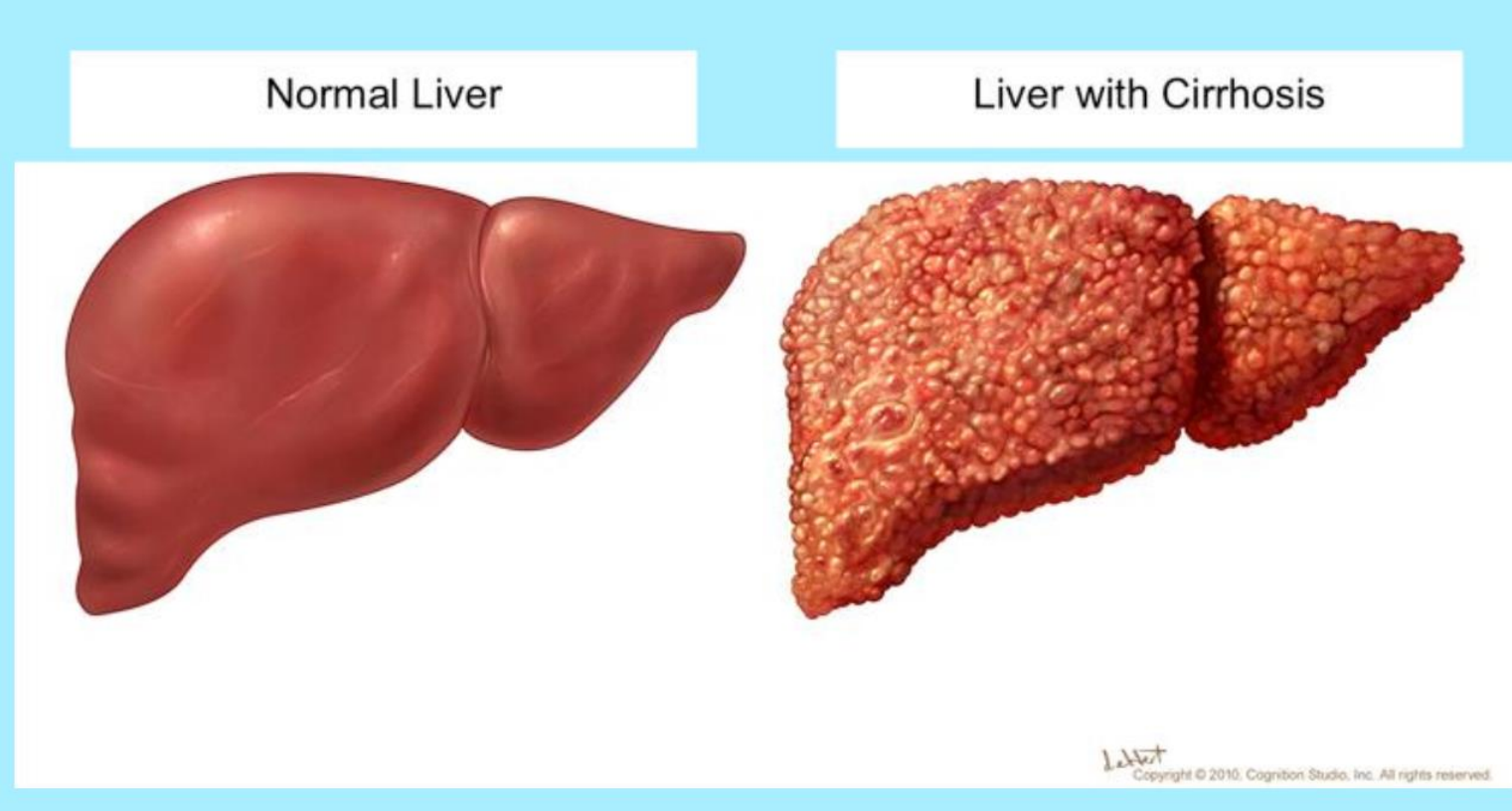
Cirrhosis


Normal Liver
Liver with Cirrhosis
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Cirrhosis

Definition:

It's the final stage of any chronic liver disease, is a diffuse process characterized by fibrosis and loss of normal architecture to structurally abnormal nodules with loss of function. These "regenerative" nodules lack normal lobular organization and are surrounded by fibrous tissue and change of blood flow

* Cirrhosis can be :Micronodular or Macronodular .

Pathophysiology

Following liver injury, stellate cells in the space of Disse are activated by cytokines produced by Kupffer cells and hepatocytes. This transforms the stellate cell into a myofibroblast-like cell, capable of producing collagen, pro- inflammatory cytokines and other mediators that promote hepatocyte damage and tissue fibrosis .

. Fibrosis evolves over years, and the progression can be described semi-quantitatively . Fibrosis and widespread hepatocyte loss lead to distortion of the normal liver architecture that disrupts the hepatic vasculature, causing portosystemic shunts. These changes usually affect the whole liver, but in biliary cirrhosis (e.g. PBC) they can be patchy.

24.28 Causes of cirrhosis

- Alcohol
- Chronic viral hepatitis (B or C)
- Non-alcoholic fatty liver disease
- Immune:
  - Primary sclerosing cholangitis
  - Autoimmune liver disease
- Biliary:
  - Primary biliary cholangitis
  - Secondary biliary cirrhosis
  - Cystic fibrosis
- Genetic:
  - Haemochromatosis
  - Wilson's disease
  - Alpha-1-antitrypsin deficiency
- Cryptogenic (unknown – 15%)
- Chronic venous outflow obstruction
- Cardiac hepatopathy with chronic hepatic congestion
- Any chronic liver disease

Clinical features

Some patients are asymptomatic and the diagnosis is made incidentally at ultrasound or at surgery .

Others present with isolated hepatomegaly, splenomegaly, signs of portal hypertension or hepatic insufficiency. When symptoms are present, they are often non-specific and include weakness, fatigue, muscle cramps, weight loss, anorexia, nausea and upper abdominal discomfort .

Cirrhosis will occasionally present because of shortness of breath due to a large right pleural effusion, or with hepatopulmonary syndrome .

**Hepatomegaly is common when the cirrhosis is due to alcoholic liver disease or haemochromatosis.

The liver is often hard, irregular and non-tender. Jaundice is mild when it first appears and is due primarily to a failure to excrete bilirubin. Palmar erythema

Spider telangiectasias (or spider naevi) occur and comprise a central arteriole (that occasionally raises the skin surface), from which small vessels radiate. They blanch with pressure and refill from the centre outwards, are usually found on the hands, face and upper chest.

multiple spider telangiectasias are a strong indicator of liver disease. Florid spider telangiectasia and gynaecomastia are most common in alcohol-related cirrhosis .

Pigmentation is most striking in haemochromatosis and in any cirrhosis associated with prolonged cholestasis.

Clubbing of the fingers and toes is not a sign of cirrhosis but is seen in combination with hypoxia in hepatopulmonary syndrome .

Endocrine changes are noticed more readily in men, who show loss of male hair distribution and testicular atrophy. Gynaecomastia is common and can be due to drugs such as spironolactone


Ascites
Medscapes
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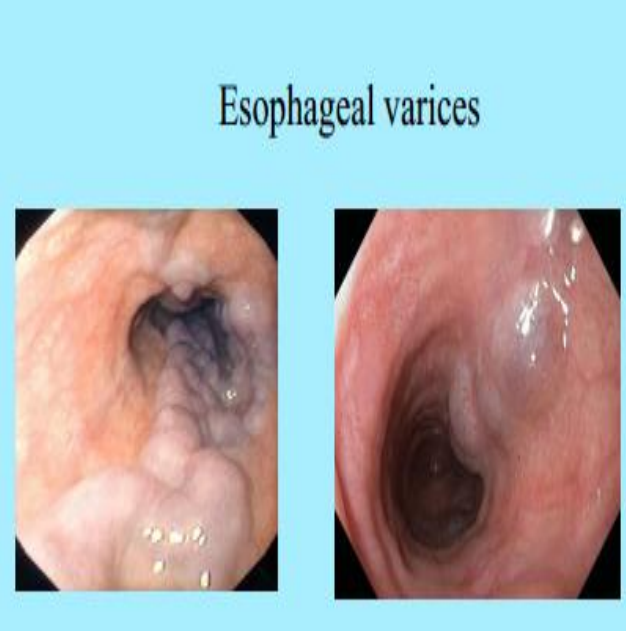
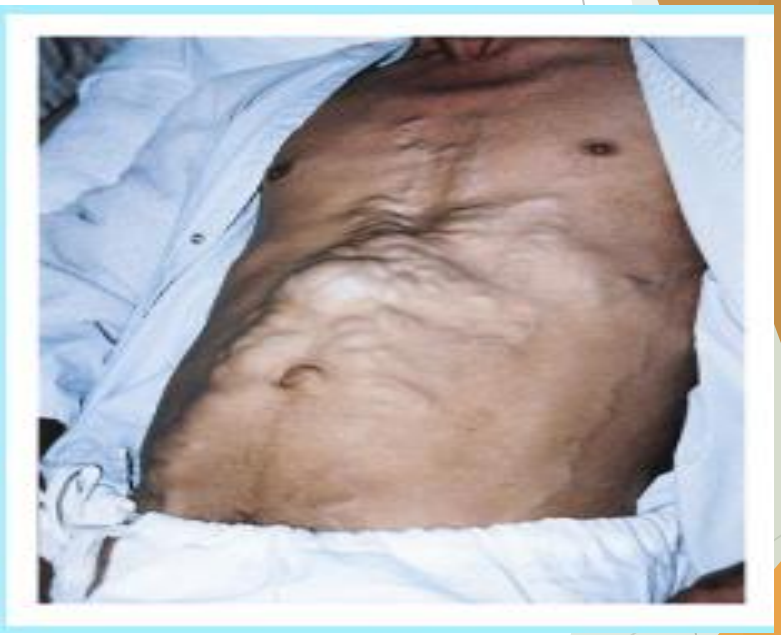
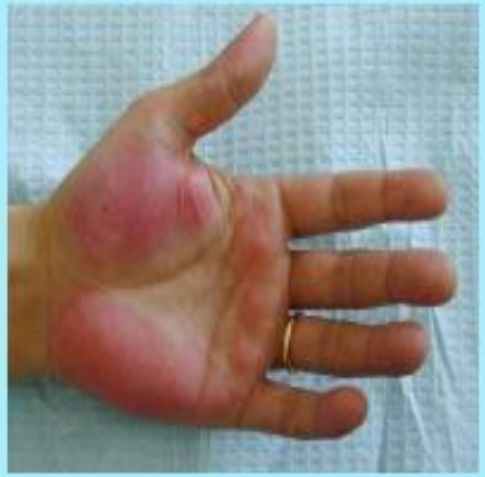
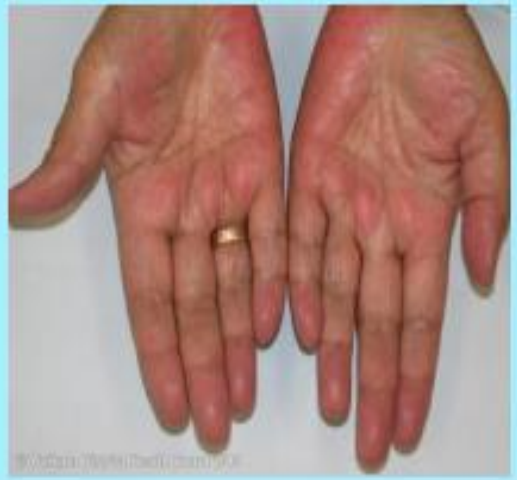

Esophageal varices



Dupuytren's contracture

Palmar erythema





**Easy bruising becomes more frequent as cirrhosis advances .

Splenomegaly and collateral vessel formation are features of portal hypertension

Jaundice, ascites, encephalopathy and variceal bleeding signify advanced disease, and are associated with a worse prognosis. The term ‘decompensated liver disease’ is often used when any of these are present .

Transition from a compensated to a decompensated stage occurs at a rate of approximately 5 to 7% per year .

peripheral oedema, renal failure, and hypoalbuminaemia and coagulation abnormalities due to defective protein synthesis.

Anaemia is common and often multifactorial, due to a combination of chronic inflammation, bone marrow suppression, haemolysis or chronic blood loss.

24.30 Features of chronic liver failure

- Worsening synthetic liver function:
  - Prolonged prothrombin time
  - Low albumin
- Jaundice
- Variceal bleeding
- Hepatic encephalopathy
- Ascites:
  - Spontaneous bacterial peritonitis
  - Hepatorenal failure

24.29 Clinical features of hepatic cirrhosis

- Hepatomegaly (although liver may also be small)
- Jaundice
- Ascites
- Circulatory changes: spider telangiectasia, palmar erythema
- Endocrine changes: loss of libido, hair loss
  - Men: gynaecomastia, testicular atrophy, impotence
  - Women: breast atrophy, irregular menses, amenorrhoea
- Haemorrhagic tendency: bruises, purpura, epistaxis
- Portal hypertension: splenomegaly, collateral vessels, variceal bleeding
- Hepatic encephalopathy
- Other features: pigmentation (haemochromatosis), Dupuytren's contracture (alcohol excess)

Management

This includes treatment :

A. the underlying cause.

B. maintenance of nutrition

C. treatment of complications, including ascites, hepatic encephalopathy, portal hypertension and varices.

Nutrition in cirrhosis

Malnutrition is a frequent complication, affecting up to 50% of patients with decompensated cirrhosis. Loss of muscle mass (sarcopenia) is associated with a higher rate of complications including infections, encephalopathy and ascites

Daily calorie intake should be no lower than 35 kcal/kg body weight with a daily protein intake of 1.2–1.5 g/kg. A late evening snack will help to minimise overnight fasting which could otherwise trigger a catabolic state in cirrhosis.

patients are unable to achieve adequate oral intake, enteral feeding should be considered .

Varices and Variceal Bleeding

Reducing portal pressure. Nonselective β-adrenergic blockers (propranolol, nadolol) reduce portal pressure by producing splanchnic vasoconstriction and decreasing portal venous inflow. **Propranolol** should be titrated to produce a resting heart rate of about 50 to 55 beats per minute.

Endoscopic variceal ligation. Endoscopy should be repeated every 2 to 3 years in patients with no varices, every 1 to 2 years in patients with small varices.
The most effective specific therapy for the control of active variceal hemorrhage is the combination of a vasoconstrictor with endoscopic therapy. Safe vasoconstrictors include terlipressin, and the somatostatin analogues ,octreotide, which is used as a 50-μg intravenous bolus followed by an infusion at 50 μg/hour

Transjugular intrahepatic portosystemic shunt (TIPS), should be used in patients whose variceal bleeding has persisted .

Ascites

Salt restriction and diuretics constitute the mainstay of management of ascites.
Dietary sodium intake should be restricted to 2 g/day.
Spironolactone, should be started at a dose of 100 mg/day to a maximal effective dose of 400 mg/day.
Furosemide, at an escalated dose from 40 to 160 mg/day.
The goal is weight loss of 1 kg in the first week and 2 kg/week subsequently.
In the 10 to 20% of patients with ascites who are refractory to diuretics,

large-volume paracentesis, aimed at removing all or most of the fluid, plus albumin at a dose of 6 to 8 g intravenously per liter of ascites removed.

in patients requiring frequent large-volume paracentesis (more than twice per month), TIPS stents should be considered .

Hepatorenal Syndrome

The mainstay of therapy is liver transplantation.
Terlipressin, plus albumin. use of terlipressin, which at a dose 0.5 to 2.0 mg intravenously every 4 to 6 hours.
The most used combination is octreotide (100 to 200 µg subcutaneously three times a day) plus midodrine.

Spontaneous Bacterial Peritonitis

Empirical antibiotic therapy with an intravenous third-generation cephalosporin. the minimal duration of therapy should be 5 days.Repeat diagnostic paracentesis should be performed 2 days after starting antibiotics.

The renal dysfunction associated with spontaneous bacterial peritonitis can be prevented by the intravenous administration of albumin, Albumin has been used at a dose of 1.5 g/kg of body weight at diagnosis.

Hepatic Encephalopathy

Treating the precipitating factor and reducing the ammonia level.

Precipitating factors include infections, overdiuresis, GI bleeding, a high oral protein load, and constipation. Narcotics and sedatives contribute to hepatic encephalopathy by directly depressing brain function.

lactulose (15 to 30 mL) orally twice daily or orally administered nonabsorbable antibiotics such as neomycin, metronidazole (250 mg two to four times per day), or rifaximin (550 mg two times per day).

• protein from an animal source to a vegetable source may be beneficial.

PROGNOSIS

The 10-year survival rate of patients who remain in a compensated stage is approximately 90%, whereas their likelihood of decompensation is 50% at 10 years.

Four clinical stages of cirrhosis:

Stage 1 patients without varices or ascites, the mortality rate is about 1% per year.

Stage 2 patients, or those with varices but without ascites or bleeding, have a mortality rate of about 4% per year.

Stage 3 patients have ascites with or without esophageal varices that have never bled; their mortality rate while remaining in this stage is 20% per year.

Stage 4 patients, or those with portal hypertensive GI bleeding with or without ascites, have a 1-year mortality rate of 57%, with nearly half of these deaths occurring within 6 weeks after the initial episode of bleeding.

THANK YOU