Lecture 11:

Complication of Liver Cirrhosis

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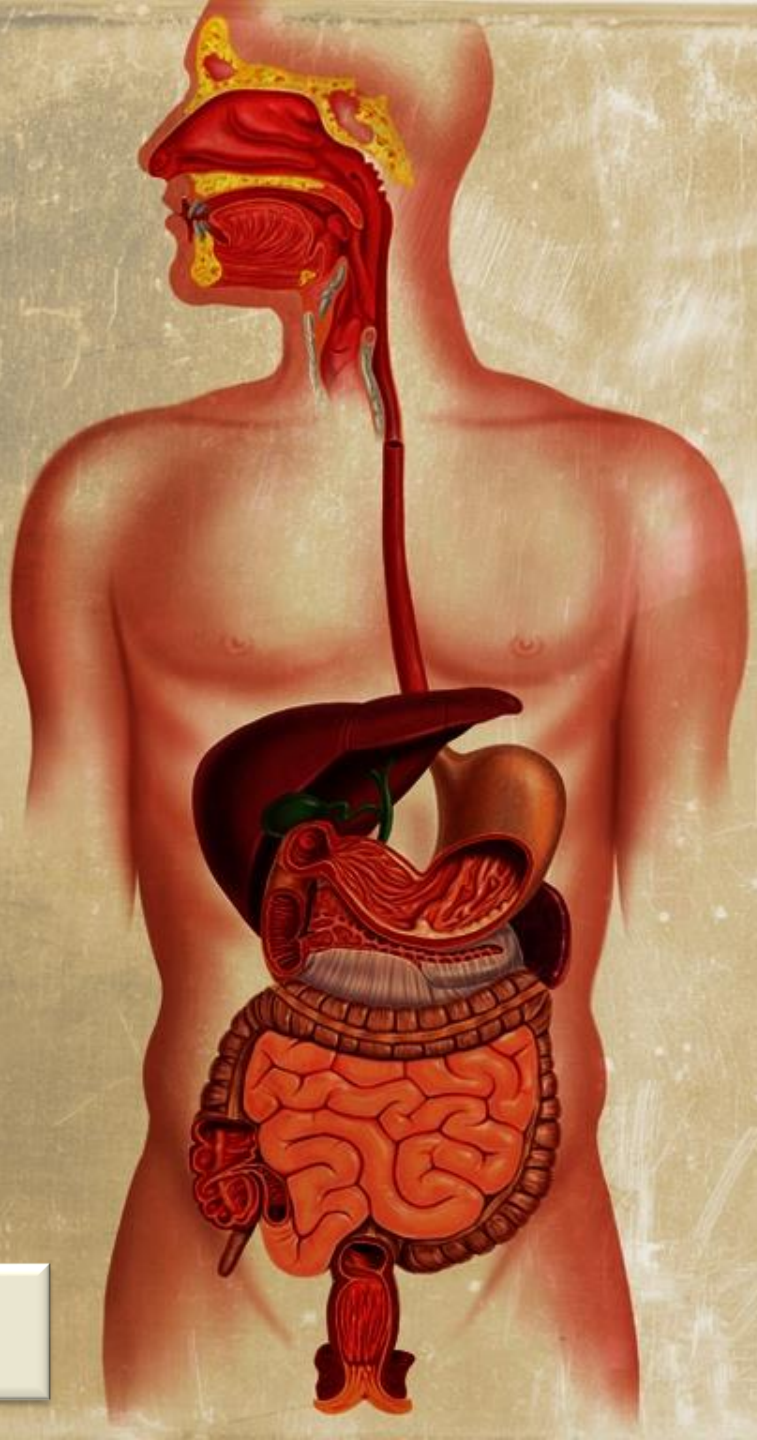

Objectives

- Recognize the major complications of cirrhosis.
- Understand the pathological mechanisms underlying the occurrence of the complications.
- Recognize the clinical features inherent to the above mentioned complications.
- Describe the pathological findings of the different complications.

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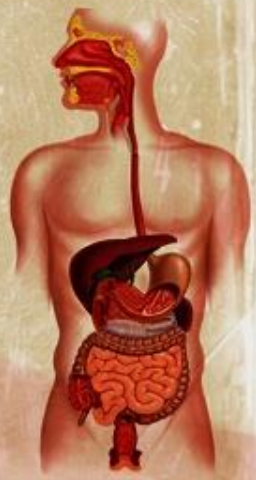


Complication of Liver Cirrhosis :

1. Ascites.
2. Portal hypertension.
3. Hepatorenal syndrome.
4. Variceal bleeding (Especially Esophageal Varices)
5. Spontaneous bacterial peritonitis. ascites increase risk of spontaneous bacterial peritonitis.
6. Hepatic encephalopathy.
7. Hepatocellular carcinoma.

We will take it in details

1. Ascites :

Ascites is the accumulation of excess fluid in the peritoneal cavity.
Most commonly caused by cirrhosis (due to portal hypertension).
Ascites generally is a serous fluid containing as much as 3 g/dL of protein (largely albumin)
There is also, decrease in serum albumin

2. Portal hypertension :

Increase resistance to portal blood flow.
May develop from prehepatic, intrahepatic, and posthepatic causes, but most common cause is intrahepatic which mostly caused by liver cirrhosis. *due to fibrosis compressing central veins *.

3. Hepatorenal syndrome :

• Appearance of renal failure in individuals with severe chronic liver disease.
• No intrinsic morphologic or functional causes for the renal failure.
• The incidence of this syndrome is about 8% per year among patients who have cirrhosis and ascites.
• *Treat the liver, and kidney will go back to normal*.
• Could be caused by :

1. Decreased renal perfusion pressure .
2. Activation of the renal sympathetic nervous system with vasoconstriction of the afferent renal arterioles
3. Increased synthesis of renal vasoactive mediators, that decrease glomerular filtration.

4. Esophageal Varices :

Diseases that interrupt the blood flow from GIT to liver *First pass for drugs and food metabolism* cause portal hypertension and can lead to the development of varices, especially in sites of portal systemic anastomosis like lower esophagus, causing esophageal bleeding.

Pathogenesis :

• Occurring for 90% of cirrhotic patients.
• Portal hypertension results in the development of collateral channels at sites where the portal and caval systems communicate. Although these collateral veins allow some drainage to occur, they lead to development of a congested subepithelial and submucosal venous plexus within the distal esophagus.

Morphology :

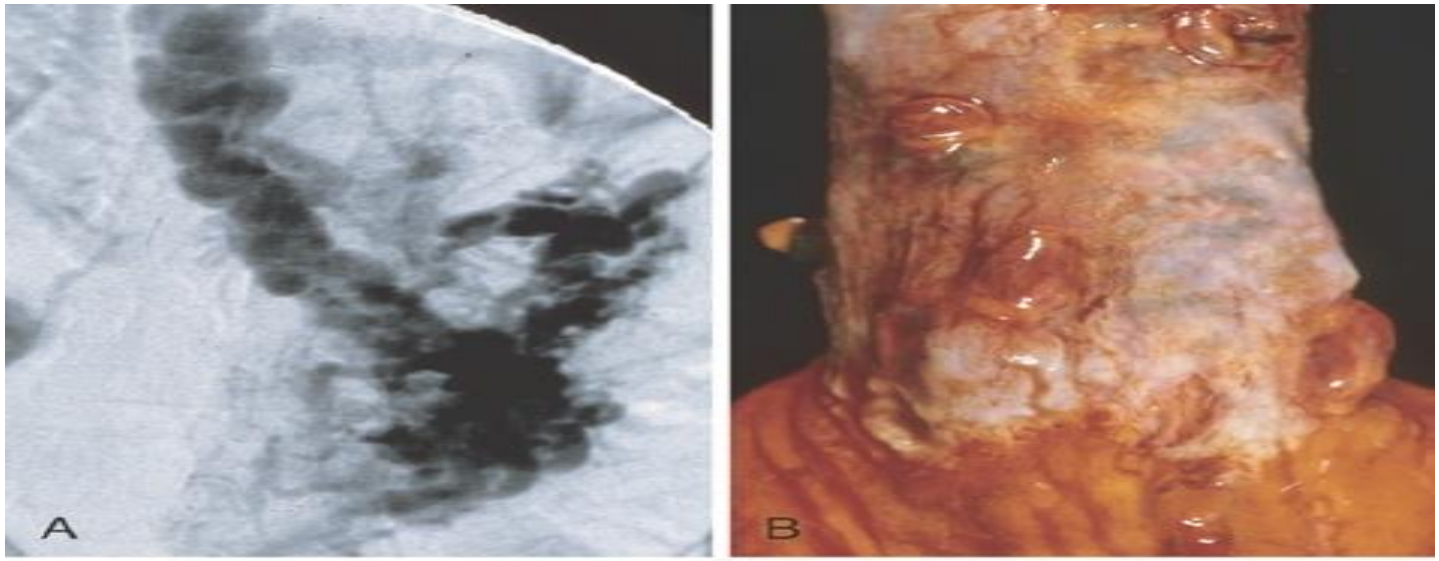
• Varices can be detected by venogram : tortuous dilated veins lying primarily within the submucosa of the distal esophagus and proximal stomach.

• Venous channels directly beneath the esophageal epithelium may also become massively dilated.

• Varices may not be grossly obvious in surgical or postmortem specimens, because they collapse in the absence of blood flow.

• Variceal rupture results in hemorrhage into the lumen or esophageal wall, in which case the overlying mucosa appears ulcerated and necrotic.

• If rupture has occurred in the past, venous thrombosis, inflammation, and evidence of prior therapy may also be present.



Clinical features :

Asymptomatic or rupture leading to massive hematemesis.

Causes of hematemesis :

1. Inflammatory erosion of thinned overlying mucosa.
2. Increased tension in progressively dilated veins.
3. Increased vascular hydrostatic pressure associated with vomiting.

Treatment :

Medical emergency that is treated by any of several methods:

1. Sclerotherapy.
2. Endoscopic balloon tamponade.
3. Endoscopic rubber band ligation.

Prognosis :

• Over half of deaths among individuals with advanced cirrhosis result from variceal rupture.

• Half of patients die from the first bleeding episode, either as a direct consequence of hemorrhage or following hepatic coma triggered by hypovolemic shock.

• Additional 50% die within 1 year.

• Each episode has a similar rate of mortality.

Other Complications of liver cirrhosis :

Splenomegaly

• Long-standing congestion may cause congestive splenomegaly (1000 gm).
• Hematologic abnormalities caused by hypersplenism, such as thrombocytopenia or pancytopenia.

Jaundice and Cholestasis :

Causes of jaundice :

- Bilirubin overproduction, hepatitis, and obstruction of the flow of bile.
- *Cholestasis*, characterized by systemic retention of not only bilirubin but also other solutes eliminated in bile.

SUMMARY

Summary from Robbins

Circulatory Disorders

- Circulatory disorders of the liver can be caused by impaired blood inflow, defects in intrahepatic blood flow, and obstruction of blood outflow.
- Portal vein obstruction by intra- or extrahepatic thrombosis may cause portal hypertension, esophageal varices, and ascites.
- The most common cause of impaired intrahepatic blood flow is cirrhosis.
- Conditions of obstruction of blood outflow include hepatic vein thrombosis (Budd-Chiari syndrome) and sinusoidal obstruction syndrome, previously known as venoocclusive disease.

SUMMARY

Jaundice and Cholestasis

- Jaundice occurs when retention of bilirubin leads to serum levels above 2.0 mg/dL.
- Hepatitis and intra- or extrahepatic obstruction of bile flow are the most common causes of jaundice involving the accumulation of conjugated bilirubin.
- Hemolytic anemias are the most common cause of jaundice involving the accumulation of unconjugated bilirubin.
- Cholestasis is the impairment of bile flow resulting in the retention of bilirubin, bile acids, and cholesterol.
- Serum alkaline phosphatase usually is elevated in cholestatic conditions.