PATHOLOGY TEAM

Complication of liver

Cirrhosis

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❖ Complications of liver cirrhosis

- Portal hypertension.
    - Ascites.
    - Portosystemic shunt and variceal bleeding.
    - Splenomegaly.
- Jaundice and cholestasis
- Hepatorenal syndrome.
- Hepatic encephalopathy
- Hepatocellular carcinoma.

❖ Portal hypertension

- Resistance to blood flow
    - prehepatic, intrahepatic, and posthepatic
    - The dominant **intrahepatic** cause is cirrhosis accounting for most cases of portal hypertension
    - Others : massive fatty liver - Hepatic schistosomiasis

Extra information from (Robbins):

Portal hypertension in cirrhosis results from increased resistance to portal flow at the level of the sinusoids and compression of central veins by perivenular fibrosis and expanded parenchymal nodules. Anastomoses between the arterial and portal systems in the fibrous bands also contribute to portal hypertension by imposing arterial pressure on the normally low-pressure portal venous system. The four major clinical consequences are (1) ascites, (2) the formation of portosystemic venous shunts, (3) congestive splenomegaly, and (4) hepatic encephalopath

Extra information :

Causes of portal hypertension are divided into prehepatic, intrahepatic and posthepatic.

A. Prehepatic causes include:Portal venous thrombosis

1. Congenital compression
2. Splenic vein thrombosis
3. Arteriovenous malformation
4. Ascariasis

B. Intrahepatic causes include:

1. Liver cirrhosis
2. Acute or chronic hepatitis
3. Schistosomiasis
4. Congenital hepatic fibrosis
5. Idiopathic portal hypertension
6. Polycystic disease
7. Primary biliary cirrhosis
8. Vitamin A toxicity
9. Sarcoidosis
10. Tuberculosis
11. Venoocclusive disease
12. Wilson disease
13. Hemochromatosis

3. Posthepatic causes include:

1. Inferior vena cava obstruction

❖ Ascites

- *The accumulation of excess fluid in the peritoneal cavity*
- 85% caused by cirrhosis
- Serous: 3 gm/dL of protein
- Mechanism : Spontaneous bacterial peritonitis

Usually you need more than 0,5 L in order to be clinically detectable

Mechanism: When there is increase in intrahepatic pressure due to fibrosis lead to shift of fluid from **hepatic sinusoids** into **space of disse (perisinusoidal space)** then this fluid will shift through lymphatic lead to over flow of fluid in lymphatic system causing back of fluid into free spaces in body like peritoneum.

❖ Splenomegaly (enlargement of the spleen)

spleen usually weighs between 150 grams and 200 grams

Long-standing congestion may cause congestive splenomegaly. (1000 gm)

– Hematologic abnormalities attributable to hypersplenism, such as thrombocytopenia or pancytopenia.

Pancytopenia is a reduction in the number of red and white blood cells, as well as platelets. If only two parameters from the full blood count are low, the term **bicytopenia** can be used.

❖ Jaundice and cholestasis

- Causes of jaundice (yellow discoloration of skin and sclera)

Bilirubin overproduction, reduced hepatic uptake/excretion, and obstruction of the flow of bile.

- ***Cholestasis*** : characterized by systemic retention of not only bilirubin but also other solutes eliminated in bile

❖ *Hepatorenal syndrome*

- *A*ppearance 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 .
- Decreased renal perfusion pressure due to systemic vasoconstriction
  - Activation of the renal sympathetic nervous system with vasoconstriction of the afferent renal arterioles
  - Increased synthesis of renal vasoactive mediators, that decrease glomerular filtration.

❖ Esophageal varices

extremely dilated sub-mucosal veins in the lower third of the esophagus

- Instead of returning directly to the heart, venous blood from the GI tract is delivered to the liver via the portal vein before reaching the inferior vena cava.
- This circulatory pattern is responsible for the *first-pass effect* in which drugs and other materials absorbed in the intestines are processed by the liver before entering the systemic circulation..
- Diseases that impede this flow cause portal hypertension and can lead to the development of esophageal varices, an important cause of **esophageal bleeding**

Pathogenesis

- Portal hypertension results in the development of collateral channels **(collateral channels might help but at same times, they are dilated, tortuous, weak and submucosal)** 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. (*varices):* 90% of cirrhotic patients

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.
- Inflammatory erosion of thinned overlying mucosa
- Increased tension in progressively dilated veins
- Increased vascular hydrostatic pressure associated with vomiting are likely to contribute

Clinical features: Asymptomatic or rupture-→ massive hematemesis.

Treatment

- Medical emergency that is treated by any of several methods: sclerotherapy
- Endoscopic balloon tamponade
- Endoscopic rubber band ligation

Sclerotherapy is a medical procedure used to treat varicose veins and "spider veins." During sclerotherapy, the physician injects a solution directly into the affected vein. The solution irritates the lining of the vessel, causing it to swell and stick together. Over time, the vessel turns into scar tissue that fades from view.

Others

- 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% within 1 year.
- Each episode has a similar rate of mortality.
- Over half of deaths among individuals with advanced cirrhosis result from variceal rupture.

❖ Hepatic encephalopathy:

- Severe loss of hepatocellular function
- Shunting of blood from portal to systemic circulation.
- Clinical manifestations.

Because of entering unfiltered blood into systemic circulation

❖ Hepatocellular Carcinoma