HEPATITIS (PATHOLOGY)

Ms.Divya K M
Associate Professor

- Acute viral hepatitis is a common disease caused by viruses such as hepatitis A ,B, C, D,E . Epstein Barr virus, cytomegalovirus, herpes virus also cause hepatitis in immunosupressed patients.

Hepatitis A

- Hepatitis A virus is RNA virus transmitted by faeco oral route
- It may occur in epidemic form or sporadic form
- The source of infection is contaminated food and water.
- Clinically characterized by mild fever, vomiting, loss of appetite and jaundice.

Hepatitis B

- Hepatitis B virus is DNA virus
- The DNA of HBV is known as dane particle.
- The antigens present are Hepatitis B Surface antigen(HBsAg) , Hepatitis B Core antigen (HBcAg) and hepatitis B Pre core antigen (HBeAg).

- HBsAg enters in circulation quite early in the disease.it can be detected in the serum in week after onset of infection.
- It disappears from blood during convalescence and is marked by appearance of anti HBsAg antibody
- Symptoms appears after transfusion of infected blood or blood products or by sexual contact.

- **Clinical features are**
- weakness
- nausea,
- vomiting,
- Hepatomegaly
- dark colored urine
- Elevated serum bilirubin level

Hepatitis D

- This is an incomplete RNA virus. This require HBV for its multiplication.
- It cannot cause infection alone
- The infection of HDV and HBV can occur simultaneously when it is called coinfection.
- If HDV infection occur in an already existing HBV infected person ,it is called superinfection.
- Superinfection causes activation of HBV carrier and progression to cirrhosis.

Hepatitis C

- This infection occur by blood transfusion and sexual contact
- The symptoms as that of HBV infection
- It is less severe and 50% cases progress into chronic hepatitis and cirrhosis and hepatocellular carcinoma

Hepatitis E

- RNA virus transmitted by faeco oral route
- Symptoms are mild
- Prognosis good

Clinicopathologic spectrum

1. Carrier state
2. Asymptomatic infection
3. Acute hepatitis
4. Chronic hepatitis
5. Fulminant hepatitis

- **Carrier state**

An asymptomatic individual without manifest disease, harboring infection with hepatotropic virus and capable of transmitting called carrier state.

- **Morphological features**
- Healthy HBV carriers may or may not show changes on liver biopsy.
- In asymptomatic chronic disease may show changes of chronic hepatitis and even cirrhosis.

2. **Asymptomatic infection**

These are cases who are detected incidentally to have infectious with one of the hepatitis virus by their serum transaminase or by detection of presence of antibodies but are asymptomatic.

3. Acute hepatitis

Acute hepatitis categorized into 4 phases

- Incubation period

  Hepatitis A- 4wks (15-45days)

  Hepatitis B 10wks (30-180days)

  Hepatitis D 6wks (30-50days)

  Hepatitis C 7wks (20-90days)

  Hepatitis E 2-8wks (15-60days)

- Pre icteric phase

Anorexia,nausea,vomitting,fatique,malaise,headache,arthralgia,low grade fever

- Icteric Phase
- Onset of clinical jaundice and constitutional symptoms diminish
- Dark coloured urine
- Clay coloured stool
- Pruritus
- Loss of weight
- Diagnosis based on liver function tests
- Serological detection of antigens and antibodies

4.Post icteric phase

- Icteric phase lasts for about 1 to 4 weeks and is usually followed by clinical and biochemical recovery in 2 to 12 weeks.
- Recovery phase is more prolonged in Hepatitis B and C

- **Morphological features**

1. Hepato cellular injury

- Mildly injured hepatocytes appear swollen with granular cytoplasm which tends to condense around nucleus.
- **Dropout necrosis** is another type of hepatocellular necrosis in this small cluster of hepatocytes undergo lysis..
- **Bridging necrosis** is more severe form of hepatocellular injury. necrosis extebd zonally from one lobule to another adjucentit is characterized by bands of necrosis linking portal tract to central hepatic vein.

2. Inflammatotory infiltrate

3.Kupffer cell hyperplasia

4.Cholestasis

5.Regeneration

- **4.chronic hepatitis**

  Continuing or relapsing hepatic disease for more than 6 months with symptoms along with biochemical,serological and histopathological evidence of inflammation and necrosis. (Hep B,C ,combination of B and D)

- Morphological features

1. Piecemeal necrosis (piece by piece)

   - necrosis that occur in fragments

   periportal destruction of hepatocytes

2. Portal tract lesions

- Inflammatory cell infiltration by lymphocytes,plasma cells and macrophages.
- Proliferated bile ductules

3.Intra lobular lesions

-bridging necrosis

-kupffer cell hyperplasia

-regeneration changes of hepatocytes

4.Bridging fibrosis

5. Fulminant hepatitis

- It is the most severe form of acute hepatitis in which there is rapidly progressive hepato cellular failure.