

Viral Hepatitis

- liver Inflammation caused by viral infection.
- caused by
- Common $\rightarrow$ Hepatitis A, B, C, D, E
- herb $\rightarrow$ Cytomegalovirus, Epstein-Barr Virus.
- Rare $\rightarrow$ Herpes Simplex, Yellow Fever.

Hepatitis A (HAV)

- Only Acute state, no chronic carriers.
- Transmission $\rightarrow$ Faeco oral Route, Travellers.
- Serology $\rightarrow$ HAV IgM antibody = Active
- HAV IgG antibody = Recovery or Vaccination.

Hepatitis E (HEV)

- Only Acute state, no chronic carriers.
- Transmission $\rightarrow$ Faeco oral Route.
- Serology $\rightarrow$ HEV IgM antibody = Active
- HEV IgG antibody = Recovery or No Vaccination.

- When occurs in Pregnant women causes acute Fulminant Hepatitis.

Hepatitis C (HCV)

- Acute & Chronic.
- Transmission $\rightarrow$ Intravenous Drug abuses, Unscreened blood transfusion, Vertical transmission, Pregnancy, Tattooing, Needle stick injury.

Tests:

- $\rightarrow$ Enzyme Immunoassay $\rightarrow$ HCV IgG $\rightarrow$ Not Protective
- $\rightarrow$ Recombinant Immunoblot $\rightarrow$ $\uparrow$ Specificity, $\downarrow$ Sensitivity
- $\rightarrow$ HCV RNA Test (with PCR).
- $\cdot$ $\uparrow$ Sensitivity.
- Shows viral RNA in Blood.
- If Decreases shows recovery.

Hepatitis B (HBV)

- Acute & Chronic 80% overall population is chronic carriers, leading cause for liver cancer
- Transmission same as HCV

Serology

① PCR → Viral RNA HBV DNA in blood

② Hepatitis B surface antigen (HBsAg)

→ HBsAg is the indicator of active infection and appears very early after onset.

→ Anti-HBsAg appears late than the disappearance of the HBsAg.

→ In between gap is called window period.

→ Recovered people do not have HBsAg and have Anti-HBsAg for life.

③ Hepatitis B core Antigen (HBcAg)

HBcAg is not found in blood, but anti-HBcAg appears early.

- First appears as IgM and changes to IgG.

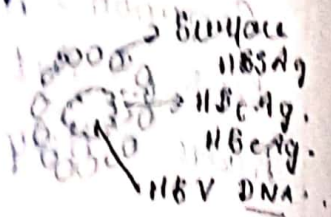
- IgM can reveal the acute infection when HBsAg has disappeared and before anti-HBsAg has appeared.

④ Hepatitis B e antigen (HBeAg)

- It indicates viral replication, and anti-HBeAg occurs immediately.

Chronic Hepatitis → ↑ HBsAg, anti-HBc (IgG), and HBeAg or anti-HBeAg indicates viral replication.

Acute Hepatitis → ↑ HBsAg, anti-HBc (IgM), and HBeAg, ↑ HBV DNA.



- It can co-exist with HIV infection
- Can cause Cirrhosis & Hepatocellular Carcinoma
- So levels of HBsAg & Alpha-fetoprotein should be checked

Hepatitis D Virus (HDV) (Delta Virus)

It can infect only if HBV is pre-existing

- Occurs by Co-infection or Superinfections.
- Serology
 - HDV IgM or IgG $\Rightarrow$ Not protective
 - $\hookrightarrow$ Active infection

Pathophysiology

Hepatitis Virus

$\downarrow$
Infection

$\downarrow$
MHC I molecule

$\downarrow$
CD8+ T cells

$\downarrow$
Cytotoxic killing

Cell Apoptosis

$\downarrow$
Councilman Body (portal tract
lobules)

$\downarrow$
Liver Damage

$\downarrow$ Bilirubin

Jaundice

$\downarrow$
ALT,

AST

ALP,

AP

$\downarrow$
Hepatomegaly

Types

1) Acute $\rightarrow$ A, E $\Rightarrow$ $\leq$ 6 months

2) Chronic $\rightarrow$ B & C $\Rightarrow$ $>$ 6 months

Symptoms

Acute

- Nausea, vomiting & Right Upper Quadrant pain
- ↑↑ Bilirubin
- Jaundice
- Pruritis, Dark urine
- Clay colored stools
- Hepatomegaly

Chronic

- Asymptomatic
- Fever, Fatigue, Loss of appetite
- Extra hepatic
- Arthralgia
- Skin rashes
- No megalia
- Symptoms of Cirrhosis if present

Lab Investigations

Serology:- CBC → ↓ Thrombocytes

Liver Function Test

- ↑ Bilirubin → Unconjugated
- ↑ Urobilinogen
- ↑ ALT, ↑ AST, ↑ ALP, Alkaline Phosphatase
- ↑ LDH
- ↓ Albumin = 3:5, A:G ratio = ↓ 1:5 to 3:1
- ↑ Immunoglobulin
- ↓ PT & APTT
- ↑ Plasma Ammonia & Aminoaciduria → Acute Fulminant Hepatitis
- ↑ Antinuclear antibody
- Ultrasound & Cirrhosis
- Biopsy, FNAC
- ERCP & MRCP

Management

- Testing during Pregnancy
- Sexual protection, Drugs