

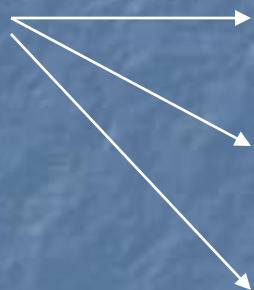
HEPATITIS B

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Hepatitis B

Etiology

Hepatitis B virus



Acute Hepatitis
Chronic Hepatitis
Hepatocellular
carcinoma

Hepadna virus

Partially double stranded DNA genome.

- Three morphological forms
 - 1.22 nm – Spherical (HBs Ag)
 - 2.tubular / filamentous 22 nm x >200 nm
 - 3.42 nm large spherical particles (Dane particles)
complete Hepatitis B virus
- Three important Ags
 - 1.HBs Ag
 - 2.HBc Ag
 - 3.Hbe Ag
- Antigenic variation of HBV gives four subtypes
- Subtypes do not differ in virulence or chronicity

HBV Stability

Resistant to physical and chemical agents.

HBs Ag & HBV stable at -20°C\ 20 years

- repeat freezing & thawing
- 37°C for 60 mins
- dried and stored at 25°C for week

HBV sensitive

- 100°C\1 min
- 60°C\10 hrs
- Ph 2.4 for 6 hrs
- 0.5 % chlorine bleach\ 3 mins

Disinfection of objects - heat sensitive

- 2% gluteraldehyde

Difficult to treat human blood.

Before screening (in blood banks)

Commonest cause of transmission
associated Hepatitis

Transmission

Virus present in blood, saliva, seminal fluid

Can spread -Blood to Blood contact

- Between IV drug abusers
- sexual partners
 - Homosexual
 - Heterosexual
- esp. : Homosexuals

- Between mother & child
 - Intrauterine infection
 - Perinatal infection
 - Postnatal infection

Transfusion → now rare

Transmissioncontd...

- Associated with
 - tattooing
 - Ear piercing
 - Acupuncture
 - use of common articles
(racers, scissors, sewing needles, tooth brushes, towels)
- Bites from infected persons

Epidemiology

- Fatality rate 1%
- Mortality as high as 20%
- Serious than Hepatitis A
- Rarely out brake
- Few → Chronic Hepatitis → Cirrhosis
- Polyarteritis nodosa
Arthritis, Nephritis →
Due to Ag-Ab complexes in persistent infections

Epidemiology contd....

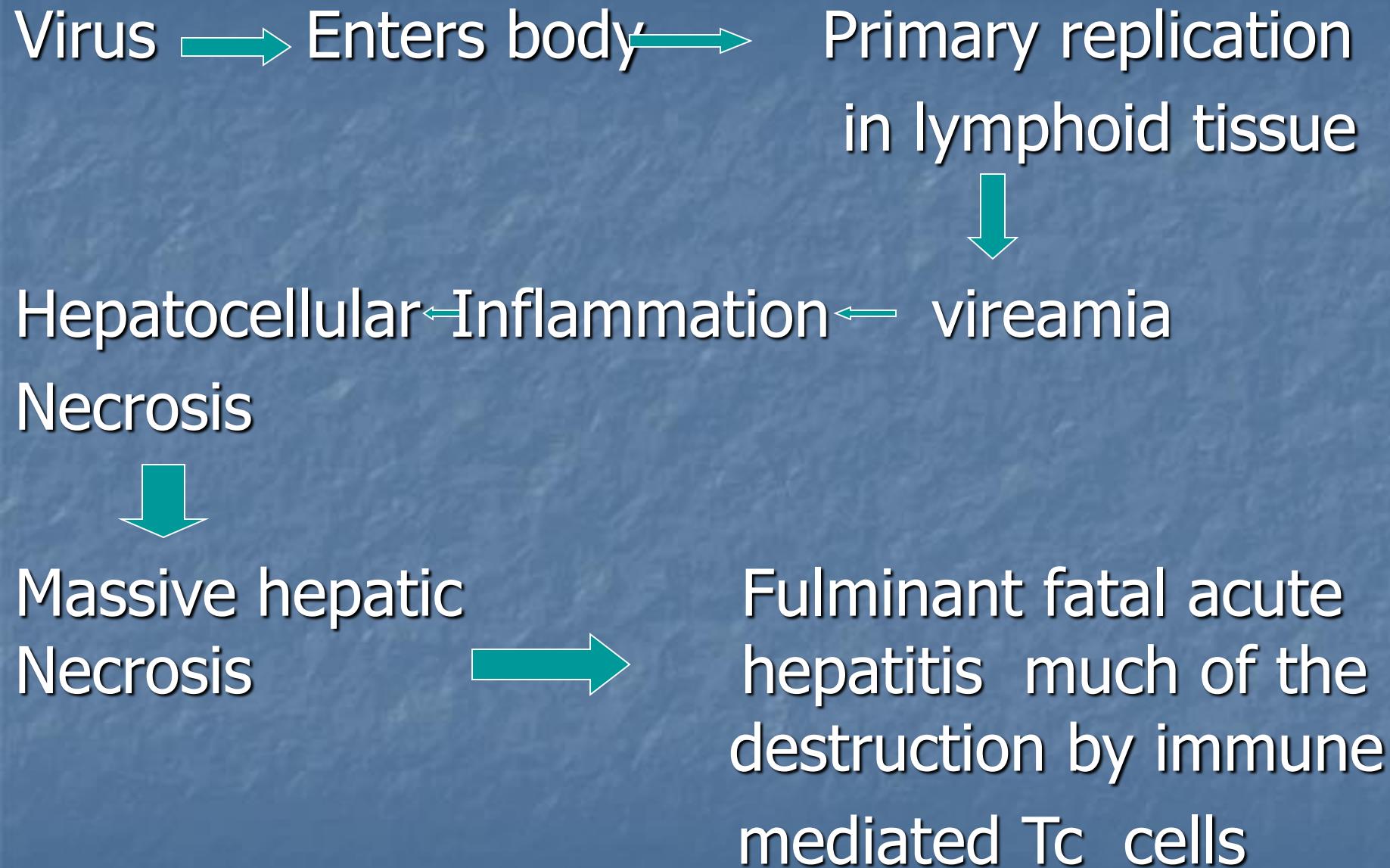
Infectious carriers (mothers)



Infant at Birth become symptom less
carriers ↓

Later in life high incidence of
-Chr. Hepatitis
-Cirrhosis
-1ry Liver carcinoma

Clinical features & Pathogenesis



Clinical features & Pathogenesis

count..

- Incubation period is long 2-3 months (usually)
Rarely one month or up to six months
- As with 1st specific Abs → Brief prodromal illness with rash & arthralgia in 10-20 % of icteric patients
- Rash is urticarial in character

Out come of Hepatitis infection

- ❖ As liver damage increases → sings of hepatitis
- ❖ Immune response → slowly recover
become effective (90-96%)
- ❖ Fulminant hepatitis 1% deaths
- ❖ Persistent infections
 - 2-10% individuals become carriers
blood remain infectious for life
 - 20-25% continuous liver damage
Chr.hepatitis

Damage is slight therefore apparently healthy

Individuals with

- ❖ Vigorous immune response -clear virus rapidly
 - severe acute illness
 - (CD₈ T cell killing)
- ❖ Immunodeficient - milder disease
 - likely to become carrier
- ❖ Males more likely to become carriers than females
- ❖ Perinatally infected infants, 90-95% become carriers
- ❖ Infected 1-3 yrs - 23% become carriers

- ❖ University students 3% become carriers
- ❖ Countries with high childhood infection > 70%
Ab +ve 12-20% carriers
- ❖ Western Europe 5% Ab +ve & 0.9% are carriers

Laboratory Diagnosis

- ❖ HBs Ag → appear in serum during incubation period (1 month before symptoms)
 ↓
 disappear 2-3 months later at the end of acute stage of illness
- ❖ As HBs Ag is ↑ → Dane particles also present
- ❖ HBs Ag ↓ or disappear → recovery/ convalescence
 anti HBs Ab detectable

Laboratory Diagnosis *Contd*

- ❖ Previously infected non carriers
 - Anti HBs Ab detectable
- ❖ Preicteric illness HBs Ag
 - HBe Ag
 - HBs Ag
- ❖ Clinical hepatitis
 - HBe Ag
 - Dane particles

Self limited HBV infection

Persistent HBV infection

Treatment & prevention

- ❖ No standard viral therapy
- ❖ But large doses of α & β interferons used to clear virus from carriers
- ❖ Safe effective vaccines available
 - plasma derived
 - yeast vector
- ❖ Up to 10% fail to develop protective Ab (<10U) even when revaccinated

Vaccination

Prophylactic

vaccination → 0 1 2 & 12
 ↓
 0 1 6 Good

immunity

Ab response > 10 units

Accidental exposure

HBs Ig immediate passive protection
& Vaccination

Vaccination Contd

- ❖ To babies born to Hep. B infected mothers
 - HBIG within 48 hrs + vaccine 0,1,6 M
- ❖ Booster – 5 years after primary cause
- ❖ EPI in Sri lanka
- ❖ End of 2nd, 4th and 6rh monthys of age

Groups requiring post exposure prophylaxis

1. Babies born to infected mothers
2. Persons accidentally
 - inoculated
 - contaminated eye, mouth, fresh cuts
 - abrasions of skin with blood from known HBs Ag +ve person

Wash with soap and water → medical advise

Groups requiring post exposure prophylaxis cont....

3. Sexual partners
4. Family contacts
5. Health care workers already immunized
but
< protective Ag (**Booster**)

Close family contacts of a case or a carrier

At risk 1.sexual partners

 2.close house hold contacts

What to do

a) check hepatitis B markers

 to see if they are infected

b) contacts who are - HBs Ag +ve

- anti HBs +ve or
- anti HBC +ve

Do not require immunization

In case of sexual partners

- ❖ give first dose of vaccine while waiting for test results
- ❖ Advise on use of condoms until immunity is established
- ❖ Sexual contacts of a acute Hep B - vaccine
 - HBIG

Mother

Baby should receive

	HBV	HBIG
HBs Ag +ve, HBe Ag +ve	+	+
HBs Ag +ve, HBe Ag -ve, or not tested	+	+
Acute Hepatitis B during pregnancy	+	+
HBs Ag +ve, anti HBe +ve	+	-

Hepatitis C

Etiology

- ❖ Caused by hepatitis C virus (HCV)
- ❖ Causes 90-95% cases of transfusion associated Non A Non B hepatitis
- ❖ SS RNA genome, belongs to “Togavirus”

Transmission

- Virus present in blood 10^4 - 10^5 /inf.dose/ μ l
- Spread → same as HBV
- other methods of transfer
 - without evidence of parenteral exposure known to occur in the community.

Clinical picture & Pathogenesis

- ❖ Incubation period 2-4 months
- ❖ Disease can be mild
- ❖ Nothing known of its pathogenesis
- ❖ Virus detectable in blood after recovery
- ❖ Carries a source of infection

Complications

1. 50% patients develop chronic hepatitis
2. 20% progress to cirrhosis
3. Infection → associated with liver cancer

Treatment

- ❖ α interferon encouraging
- ❖ No vaccine

Hepatitis D

Etiology

- ❖ Small RNA virus
- ❖ HDV or delta virus
- ❖ causes hepatitis D
- ❖ SS RNA circular genome
- ❖ A defective RNA virus depend on HBV (a DNA virus)
- ❖ Multiply in a cell when cell is sametime infected with HBV
- ❖ When it buds from cell acquire envelope consisting of HBs Ag

Transmission

- ❖ Similar to HBV & HCV
- ❖ Infected blood contains up to 10^{10} inf. Doses/ μ l in chimps

Clinical effects and pathogenesis

- HBV & HDV → simultaneous infection
illness no more severe than HBV alone
- HDV infection of carriers → severe acute hepatitis
- UK mainly found in drug addicts
- Worldwide → present in 5% of HBV carriers
- Common in South American & Africa

Diagnosis

Lab tests

- ❖ HD Ag (Delta Ag) or Ab to HD Ag
- ❖ HBs Ag not necessarily detectable

Prevention

- ❖ No vaccine
- ❖ Vaccine against HBV prevents HDV

Hepatitis E

- Also known as Enteric non A non B Hepatitis
- Caused by SS RNA, a “Calcivirus”
- Virus excrete in faeces
- Spread is by faeco- oral route
- Uncommon in developed countries
- Waterborne infections - responsible for 50%
sporadic hepatitis in developing
countries
- Incubation period : 6-8 weeks

- Disease is mild
- But severe in pregnant women
- High motility (20%) with DIC during 3rd trimester
- Virus is eliminated in recovery
- No carriers
- Normal Ig potential therapeutic value
- Serological tests → under developed

Hepatitis F

❖ 5-10 % cases of known HGV - Non A-E

Hepatitis G

(GB virus C)

Family “Flavivirus” group like HCV

Hepatitis by TTV

Transfusion associated

Studies on

- ❖ HGV alone or patients have both HGV or HCV
- ❖ HGV does not cause chronic hepatitis
- ❖ HGV do not affect clinical course of A B or C
- ❖ Persistent infection common

