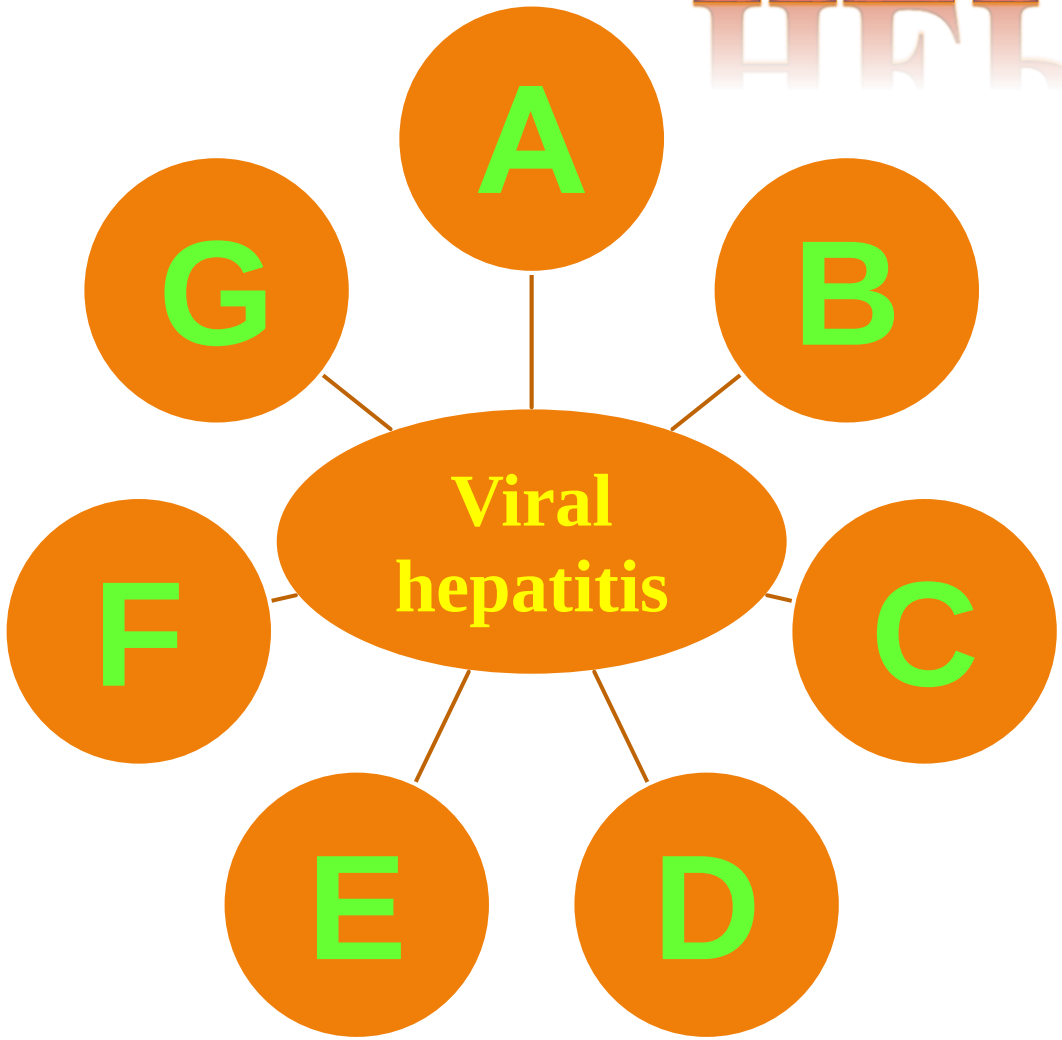
VIRAL HEPATITIS



What is Viral Hepatitis?

- **Viral hepatitis is a systemic disease with primary inflammation of the liver by any one of a heterogeneous group of hepatotropic viruses.**

The most common causes of viral hepatitis are the 7 unrelated hepatotropic viruses

Hepatitis A, Hepatitis B,

Hepatitis C, Hepatitis D, Hepatitis E, Hepatitis F, Hepatitis G.

In addition to the nominal hepatitis viruses, other viruses that can also cause liver inflammation include Herpes simplex, Cytomegalovirus, Epstein–Barr virus, or Yellow fever.

Diseases have similar clinical pattern,

but it differs in

- **etiology,**
- **epidemiology,**
- **pathogenesis,**
- **outcomes.**

Viral hepatitis is a major health problem in developing and developed countries.

In the world today:

about 4 billion people had contact with the hepatitis B virus (HBV);

400 million are asymptomatic carriers of HBV;

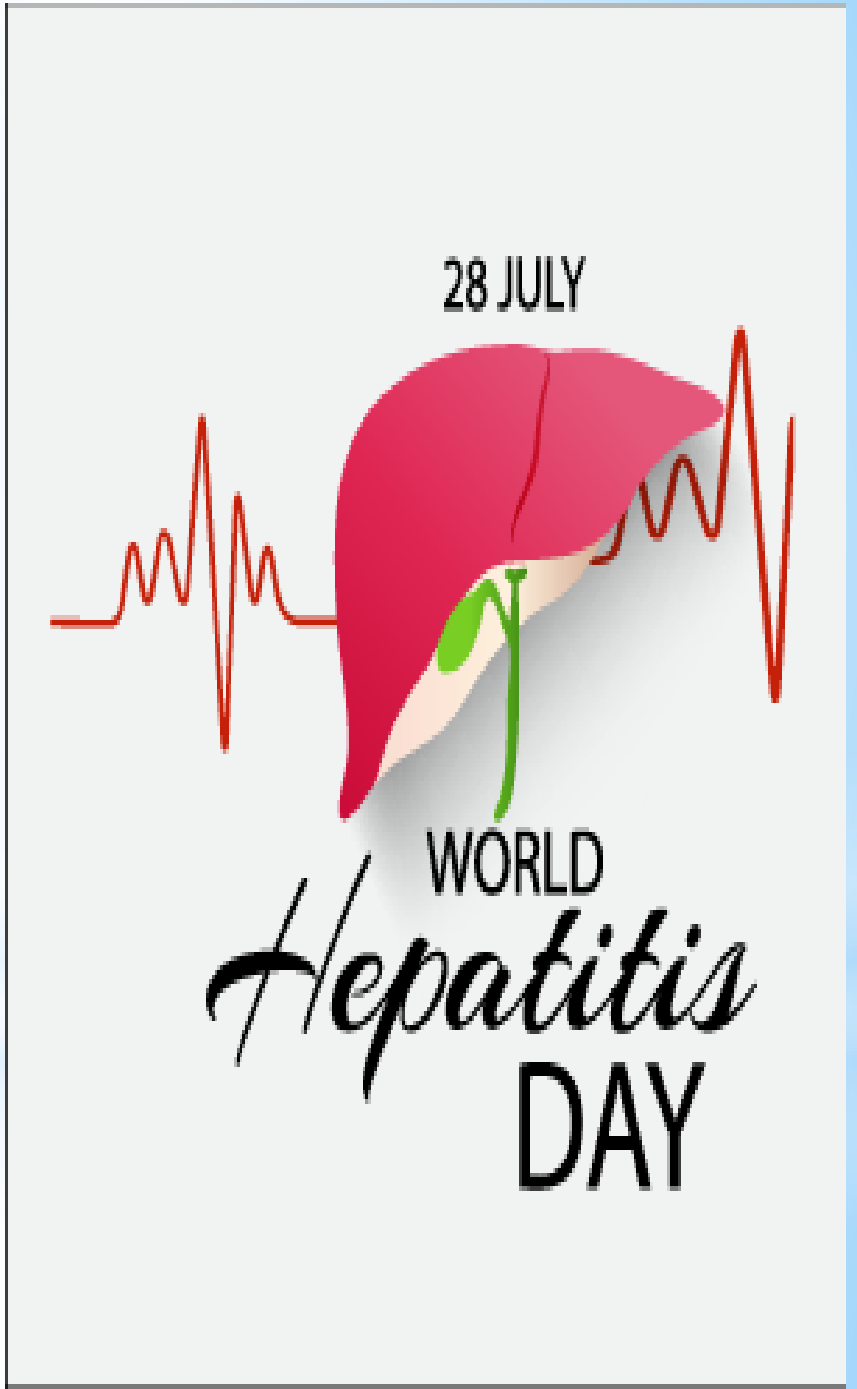
the number of newly reported cases – 4-5 million;

the number of deaths associated with HBV infection, is 1-2 million annually.

Equally widespread is hepatitis C and

From 120 to 180 million people are infected with hepatitis C virus (HCV).

Since 2010, the World Health Organization has designated a day in July as World Hepatitis Day.
That day is July 28 and the theme for the day is “Eliminate Hepatitis Day.”
It is a day to raise awareness of the 400 million people infected with hepatitis B or C around the world.
Every year, 1.4 million people die from viral hepatitis worldwide.
Last year, the World Health Organization made it a goal to eliminate hepatitis as a public health threat by 2030.



Hepatitis A, C, D, and E are RNA viruses representing four different families,

and

hepatitis B is a DNA virus

Viral hepatitis

Ways of transmission

Hepatitis A

Hepatitis E

→ **Enteral route of transmission**

Hepatitis B

Hepatitis C

Hepatitis D

→ **Parenteral route of transmission**

Dr. Shivaraj

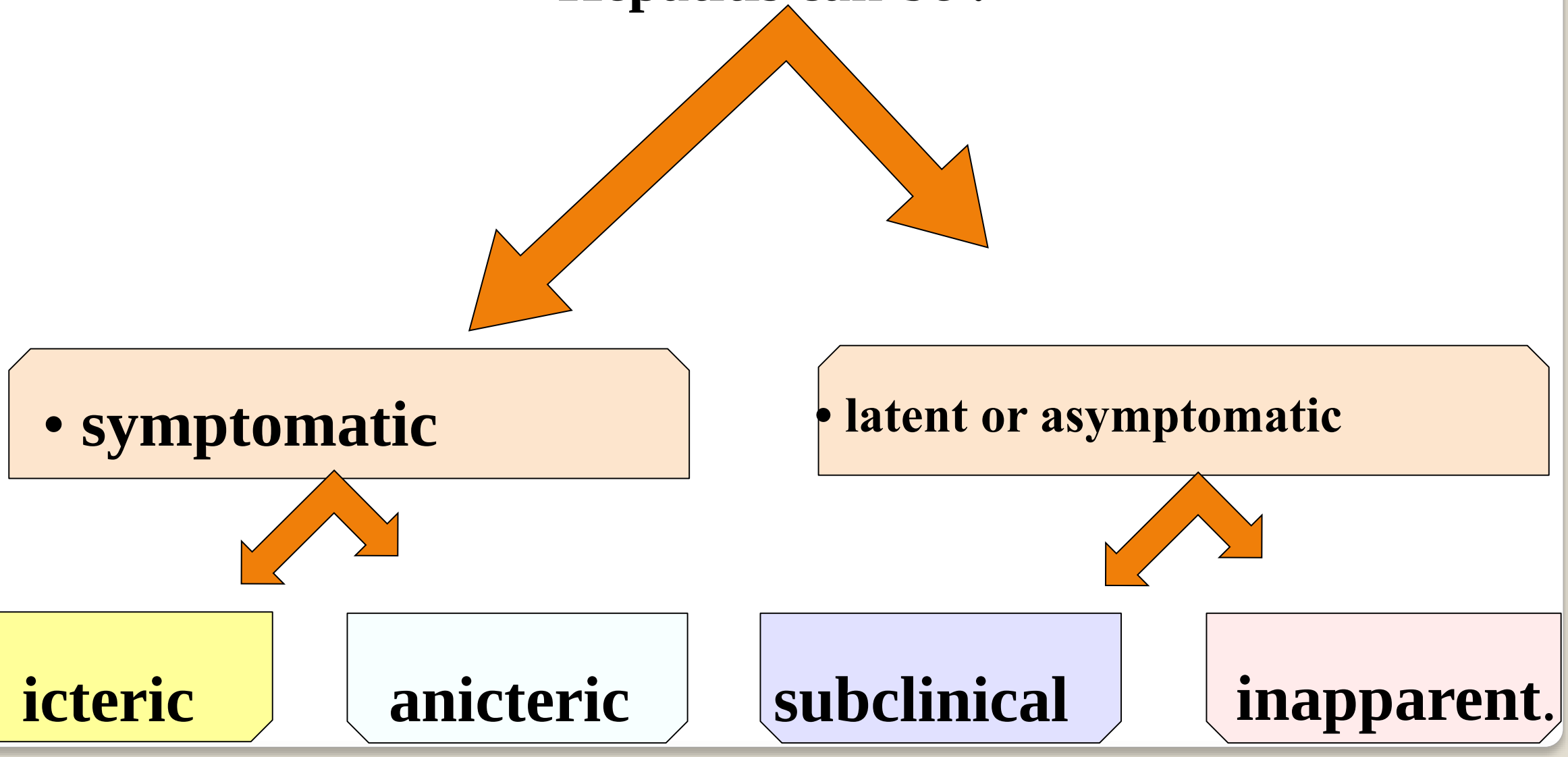
❖ Types Of Viral Hepatitis

	Viral Hepatitis A	Viral Hepatitis B	Viral Hepatitis C	Viral Hepatitis D	Viral Hepatitis E
Agent	Hepatitis A virus (HAV); ssRNA; No envelope	Hepatitis B virus (HBV); dsDNA; envelope	Hepatitis C virus (HCV); ssRNA; envelope	Hepatitis D virus (HDV); ssRNA; envelope from HBV	Hepatitis E virus (HEV); ssRNA; no envelope
Route of Transmission	Fecal-oral	Parenteral, Vertical, Sexual.	Parenteral	Parenteral	Fecal-oral
Age affected	Children	Any age	Adults	Any age	Young adults
Carrier state	Nil	Common	Present	Nil (only with HBV)	Nil
Incubation period	10-50 days (avg. 25-30)	50-180 days (avg. 60-90)	40-120 days	2-12 weeks	2-9 weeks
Chronic infection	No	Yes	Yes	Yes	No
Specific Prophylaxis	Ig and Vaccine	Ig and Vaccine	Nil	HBV vaccine	Nil

❖ Clinical Terms

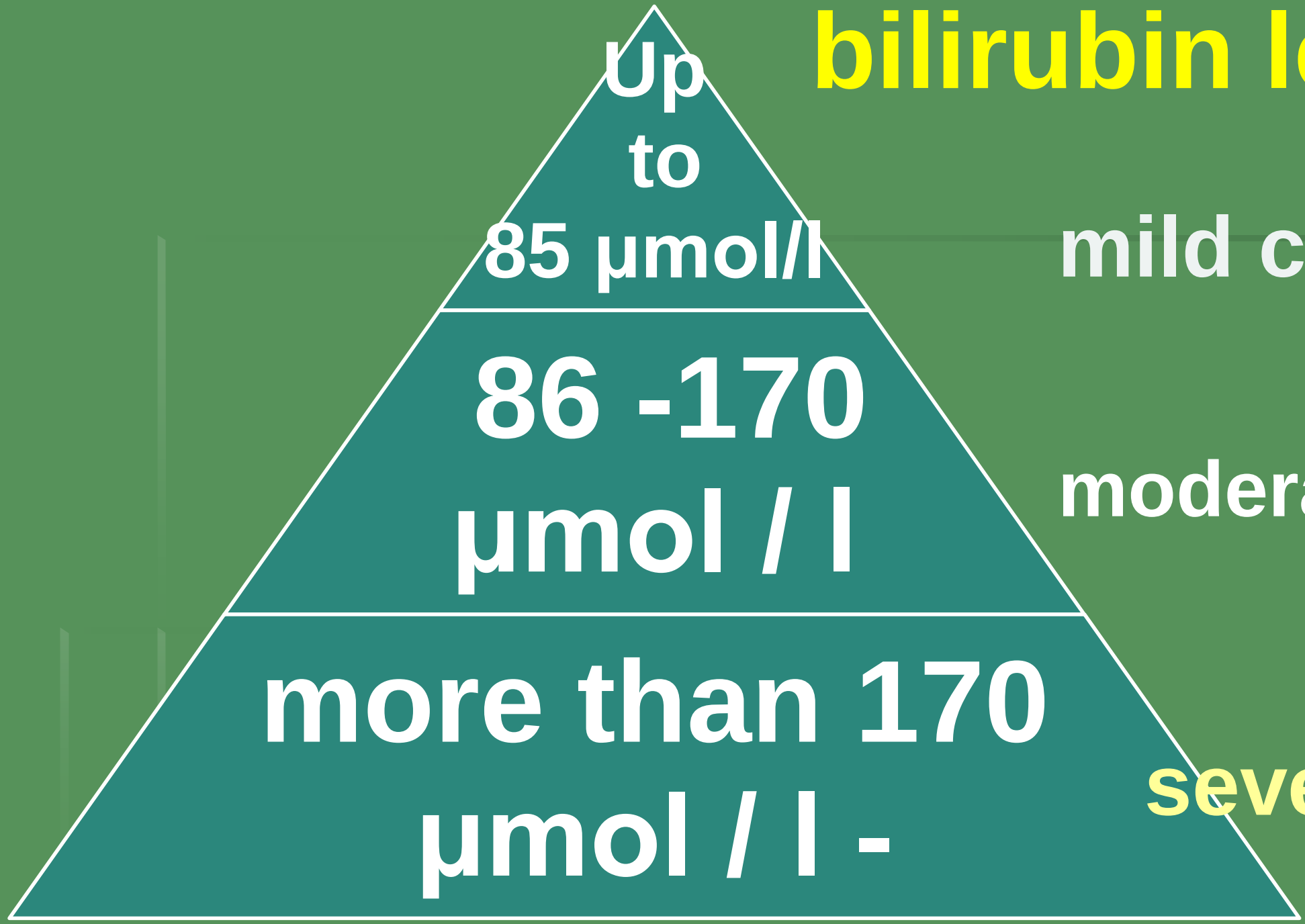
- **Hepatitis: inflammation of liver;**
- **Acute Viral Hepatitis: symptoms last less than 6 months**
- **Acute Hepatic Failure:is the appearance of severe complications rapidly after the first signs of liver disease (such as jaundice), and indicates that the liver has sustained severe damage (loss of function of 80-90% of liver cells).Massive hepatic necrosis with impaired consciousness within 8 weeks of onset of illness.**
- **Chronic Hepatitis: Inflammation of liver for at least 6 months**
- **Cirrhosis: Replacement of liver tissue→ fibrosis(scar tissue).These changes lead to loss of liver function.**
- **Fulminant Hepatitis: severe impairment of hepatic functions or severe necrosis of hepatocytes in the absence of preexisting liver disease.**

According to clinical manifestations Hepatitis can be :



By severity, they are divided into:

- mild forms of the disease
- moderate forms of the disease
- severe forms of the disease
- especially severe (fulminant) forms of the disease.



bilirubin level

mild course

moderate course

severe course

By the nature of the course of viral hepatitis, there are:

- acute cyclic (up to 3 months);
- acute prolonged or progressive course (up to 6 months);
- chronic (more than 6 months).

The classic symptoms of acute hepatitis

anorexia,
nausea,
vomiting,
severe weakness,
abdominal pain,
low-grade fever,
jaundice,
dark urine,
acholic stool

In some patients, the clinical picture resembles that when serum sickness, and characterized
arthralgia,
arthritis and
skin rashes.

These symptoms are more common in hepatitis B and have been reported in 20% of those infected.

❖Hepatitis A

- ✓ **Hepatitis A (formerly known as “infectious” hepatitis or epidemic jaundice) is an acute infectious disease caused by Hepatitis A virus (HAV).**
- ✓ **The disease is heralded by non-specific symptoms such as fever, chills, headache, fatigue, generalized weakness and aches and pains, followed by anorexia, nausea, vomiting, dark urine and jaundice.**
- ✓ **The disease is benign with complete recovery in several weeks.**

❖ Epidemiological determinants

➢Agent factors

a) **AGENT: The causative agent, the hepatitis A virus, is a small, non-enveloped hepatotropic virus classified in the genus Hepatovirus within the family Picornaviridae.**

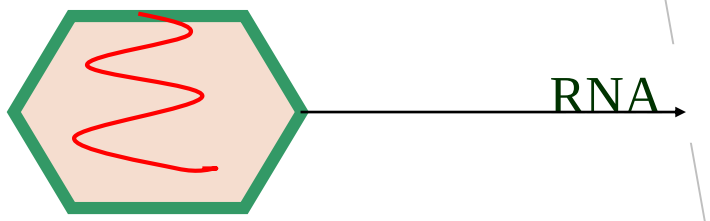
b) **It multiplies only in hepatocytes.**

c) **RESISTANCE: The virus is fairly resistant to heat and chemicals.**

-Withstands heating to 60^0 C for 1 hr. and is not affected by chlorine in doses usually employed for chlorination.

-Formalin is stated to be an effective disinfectant.

-The virus is inactivated by ultraviolet rays and by boiling for 5 minutes or autoclaving.



Naked RNA virus

c) **RESERVOIR OF INFECTION:** **The human cases are the only reservoir of infection.**

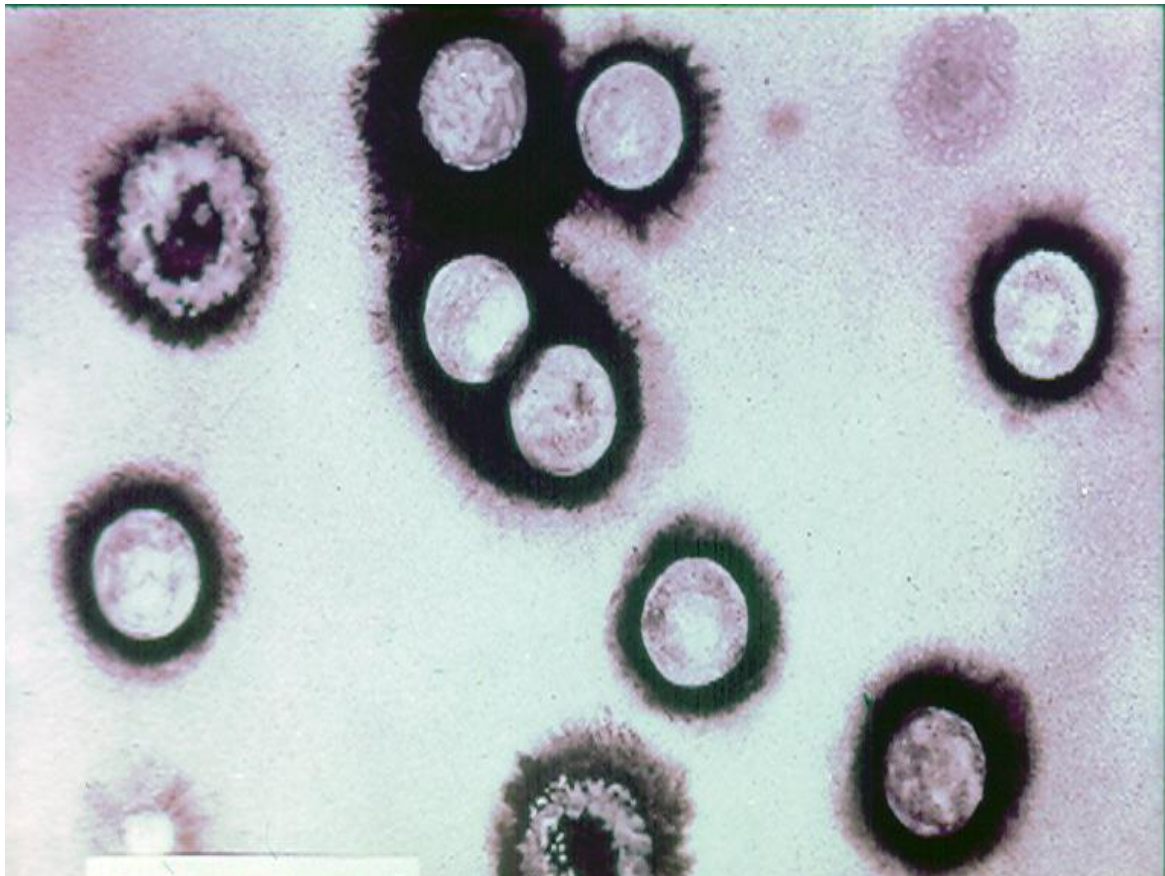
d) **PERIOD OF INFECTIVITY :** **The risk of transmitting HAV is greatest from 2 weeks before to 1 week after the onset of jaundice.**

e) **INFECTIVE MATERIAL :** **Mainly man's faeces.**

f) **VIRUS EXCRETION:** **HAV is excreted in the faeces for about 2 weeks before onset of jaundice and for up to 2 weeks thereafter.**

HAV is a 27-nm diameter, RNA-containing virus that is a member of the Picornavirus family.

It was isolated originally from stools of infected patients.
Laboratory strains of HAV have been propagated in tissue culture.
Acute infection is diagnosed by detecting immunoglobulin (Ig)M (IgM) antibodies (anti-HAV) by radioimmunoassay or, rarely, by identifying viral particles in stool.



Epidemiology.

Hepatitis A is predominantly a childhood infection, but it is not uncommon in the 15-29 age group.

Susceptibility to hepatitis A is universal. Most often, the disease is recorded in children older than 1 year (especially at the age of 3-12 years) and in young people. Children of the first year of life practically do not get sick, which is associated with the presence of transplacental immunity received by the child from the mother (most mothers have anti-HAV), the nature of nutrition and limited contact with the outside world. But since by the end of the first year of life received from the mother antibodies in a child undergo catabolism, children become susceptible to the virus.

In the overall structure of the incidence of acute viral hepatitis in children, hepatitis A accounts for about 80% cases, hepatitis B - 10% and 10% - for coinfection viruses B, C, D (mixed viral hepatitis).

The only source (reservoir) of the infectious agent is human. The virus is isolated by patients with various forms of the disease, including the most common (especially in children):
anicteric and erased, which are usually not diagnosed, respectively, those who fall ill with these forms are not hospitalized.
They lead an active lifestyle and can infect others.
In patients with icteric form, the virus is excreted with faeces most intensively at the end of the incubation period and during the preicteric period. With the advent of jaundice, in most cases, the infectivity of patients is significantly reduced. It is this feature - the most active isolation of the virus from the patient's body during the incubation period, combined with the high resistance of HAV in the environment - that causes the wide spread of hepatitis.

Routes of transmission of HAV: ◘ water, ◘ food and ◘ household.

Transfer factors -

various food products (including seafood, frozen vegetables and fruits) that have not been cooked, as well as water and dirty hands.

Large outbreaks of HAV are associated with faecal contamination of reservoir waters (sources of water supply) or with the ingress of sewage into the water supply network. In children's groups, the contact-household route of transmission through dirty hands and various household items: toys, dishes, linen, etc. is of great importance.

Very rarely, sexual transmission of HAV can occur through oral-anal contact in homosexuals.

Hepatitis A can be spread through personal contact, consumption of raw sea food or drinking contaminated water.

This occurs primarily in third world countries.

Strict personal hygiene and the avoidance of raw and unpeeled foods can help prevent an infection.

►►HAV does not pass through the placenta and is not found in breast milk, so if a woman gets HAV during pregnancy, the baby is not at risk of developing congenital hepatitis and she can breastfeed the newborn in good personal hygiene.

►► Human susceptibility to HAV is universal.

►► Immunity after suffering HA is long, almost lifelong.

HAV is characterized by a seasonal increase in the incidence in the summer-autumn period.

Along with the seasonal, there is also a cyclical increase in the incidence of HAV after 3-5, 7-20 years, which is associated with a change in the immune structure of the host population of the virus.

Clinical features.

In manifest cases of the disease, the following periods are distinguished :

- **incubation**
- **Preicteric (prodromal)**
- **icteric**
- **convalescence.**

The incubation period for HAV is on average 14-28 days (from 7 to 50 days).

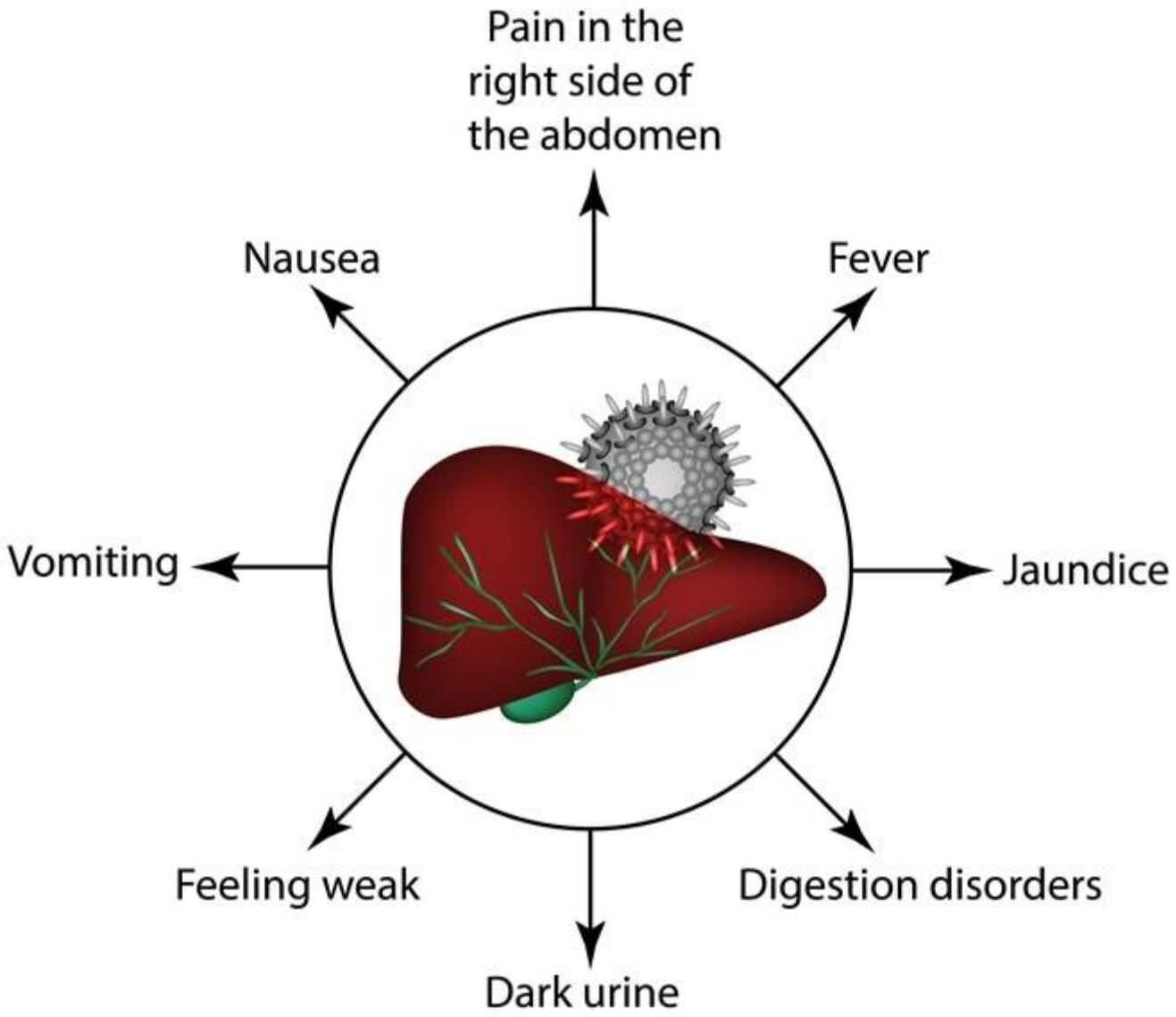
The prodromal (preicteric) period, lasting an average of 5-7 days, is characterized by the predominance of a toxic syndrome occurring in flu-like, dyspeptic, asthenovegetative and mixed variants.

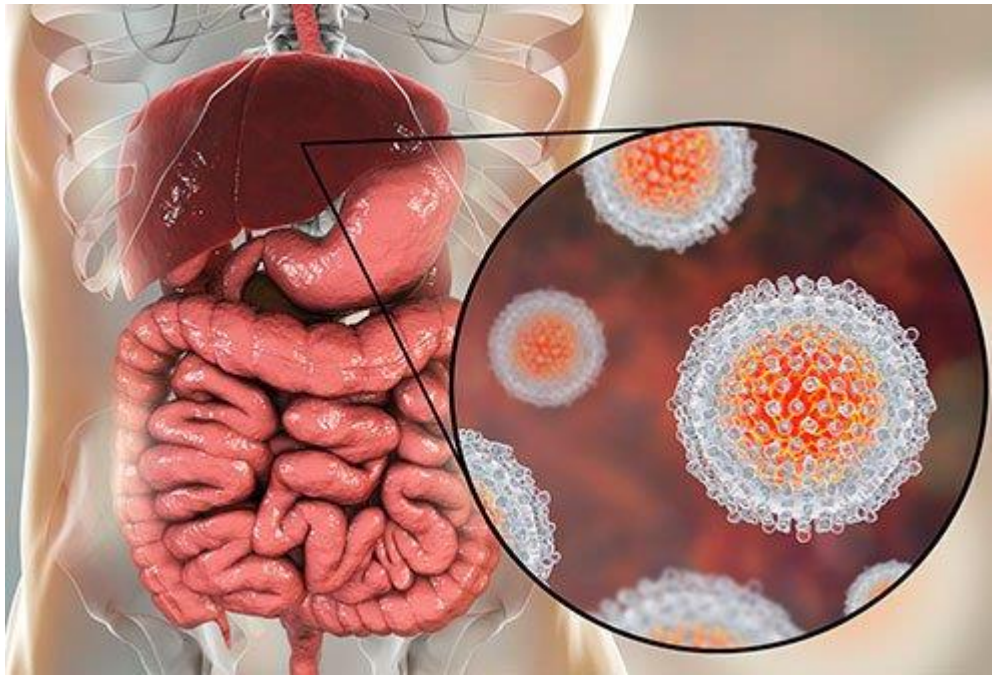
The most frequently observed "febrile-dyspeptic" variant, which is characterized by an acute onset with an increase in body temperature up to 38-40 ° C for 1-3 days, catarrhal phenomena, headache, loss of appetite, nausea and discomfort in the epigastric region.

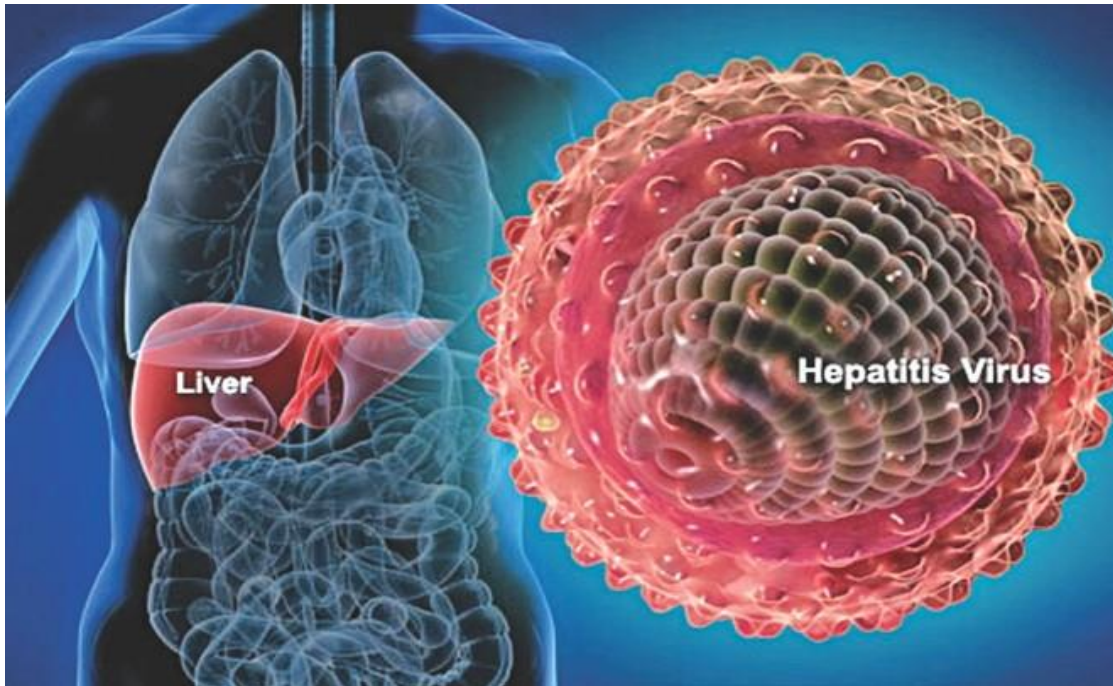
After 2-4 days, there is a change in the color of urine (choluria), which acquires the color of beer or tea. During this period, the liver increases, the palpation of which is very sensitive, and sometimes (in 10-20% of patients) the spleen. Biochemical examination reveals an increase in ALT activity.

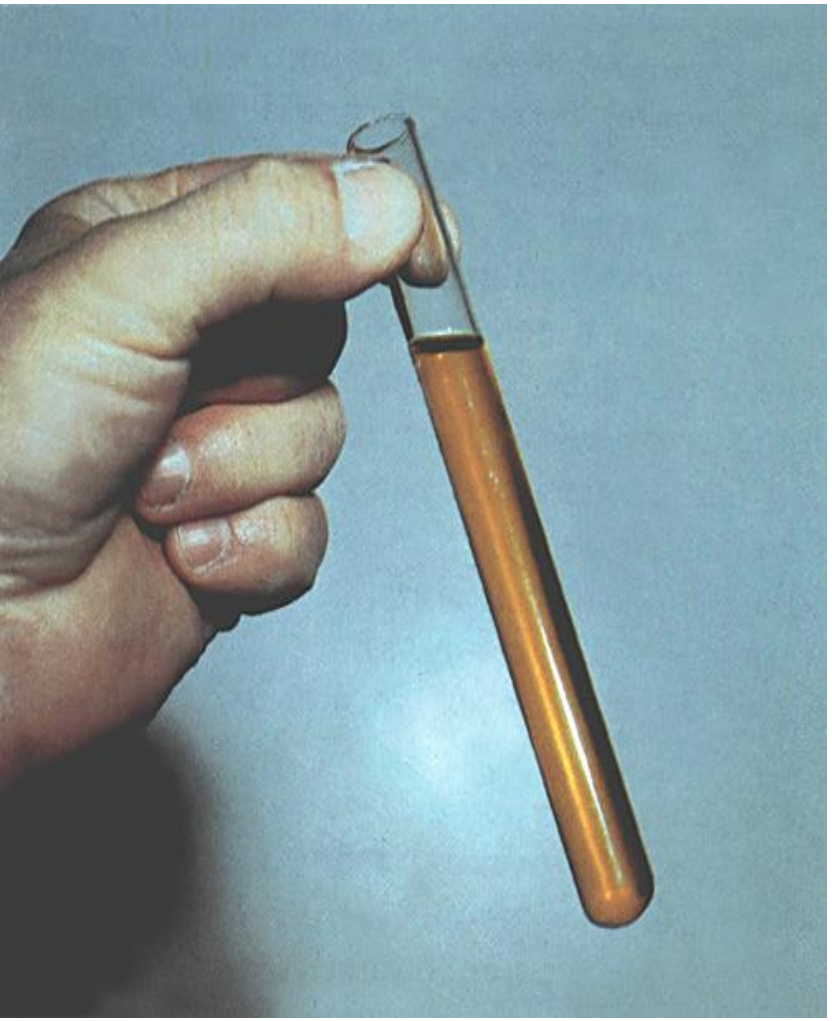
Then comes the peak period, lasting an average of 2-3 weeks. As a rule, the onset of jaundice is accompanied by fecal acholia, a decrease in body temperature to a normal or subfebrile level, a decrease in headache and other general toxic manifestations, which is an important differential diagnostic sign of hepatitis A.

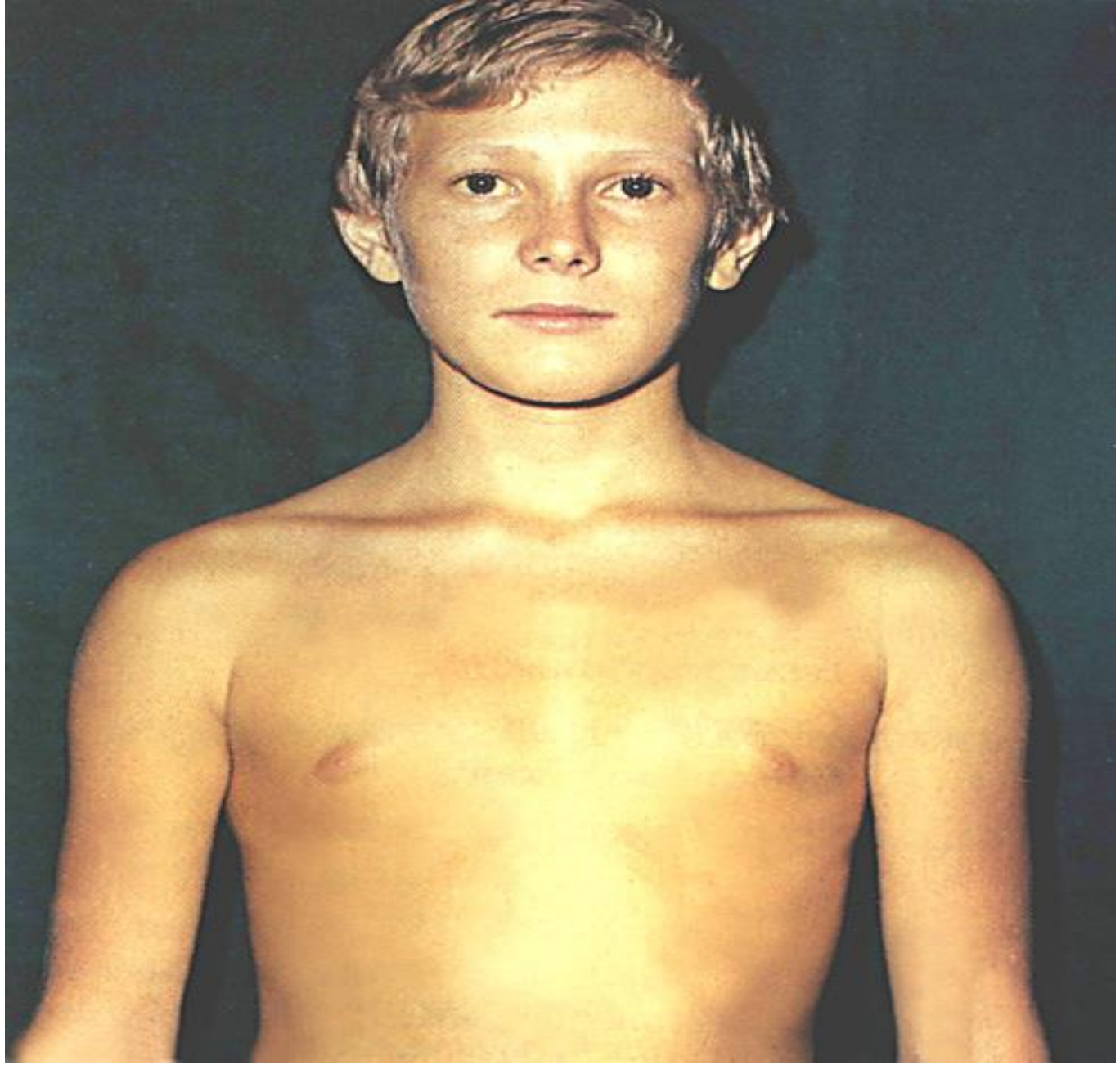
SYMPTOMS OF HEPATITIS A











The phase of extinction of jaundice usually proceeds more slowly than the phase of increase, and is characterized by the gradual disappearance of signs of the disease.

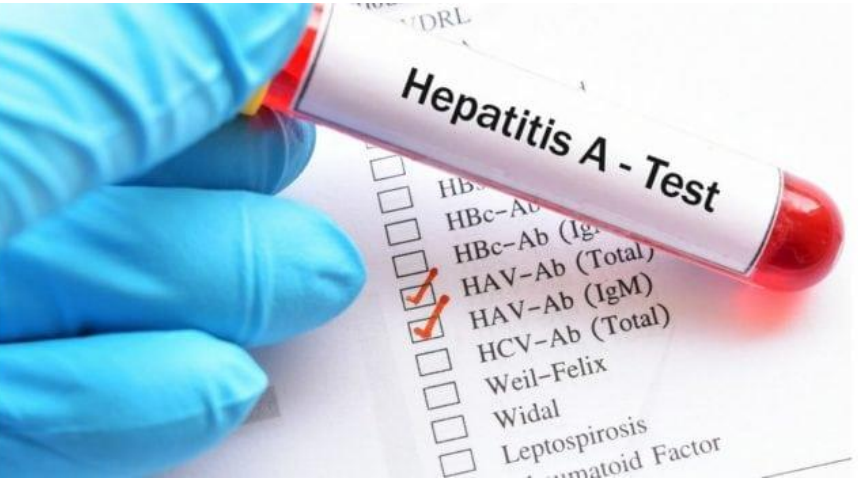
With the disappearance of jaundice, a period of convalescence begins, the duration of which is very variable (from 1-2 to 6-12 months). At this time, patients' appetite normalizes, asthenovegetative disorders are eliminated, the size of the liver and spleen are restored, and liver function tests return to normal.

Diagnosis of HA is based on clinical, epidemiological and laboratory data.

❖Diagnosis

1. Demonstration of Virus in feces, blood, bile:
   By: Immunoelectron microscopy
2. Virus Isolation:
3. Detection of Antibody :By ELISA
4. Biochemical tests:
   i) Alanine aminotransferase (ALT)
   ii) Bilirubin
   iii) Protein
5. Molecular Diagnosis : RT PCR of feces

Diagnostic methods



The laboratory diagnosis of hepatitis A can be made with specific serological tests for detection of anti-HAV antibodies. Anti-HAV IgM antibodies are generally detectable from 4 weeks to 4–6 months after infection, rarely persisting for more than 12 months. Anti-HAV IgG and IgM antibodies can be detected simultaneously 1–2 weeks after the onset of symptom. Anti-HAV IgG are then detectable lifelong.

Molecular characterisation of the virus for epidemiological purposes is performed by comparative sequencing analysis of specific regions of the viral genome.

❖Prevention:-

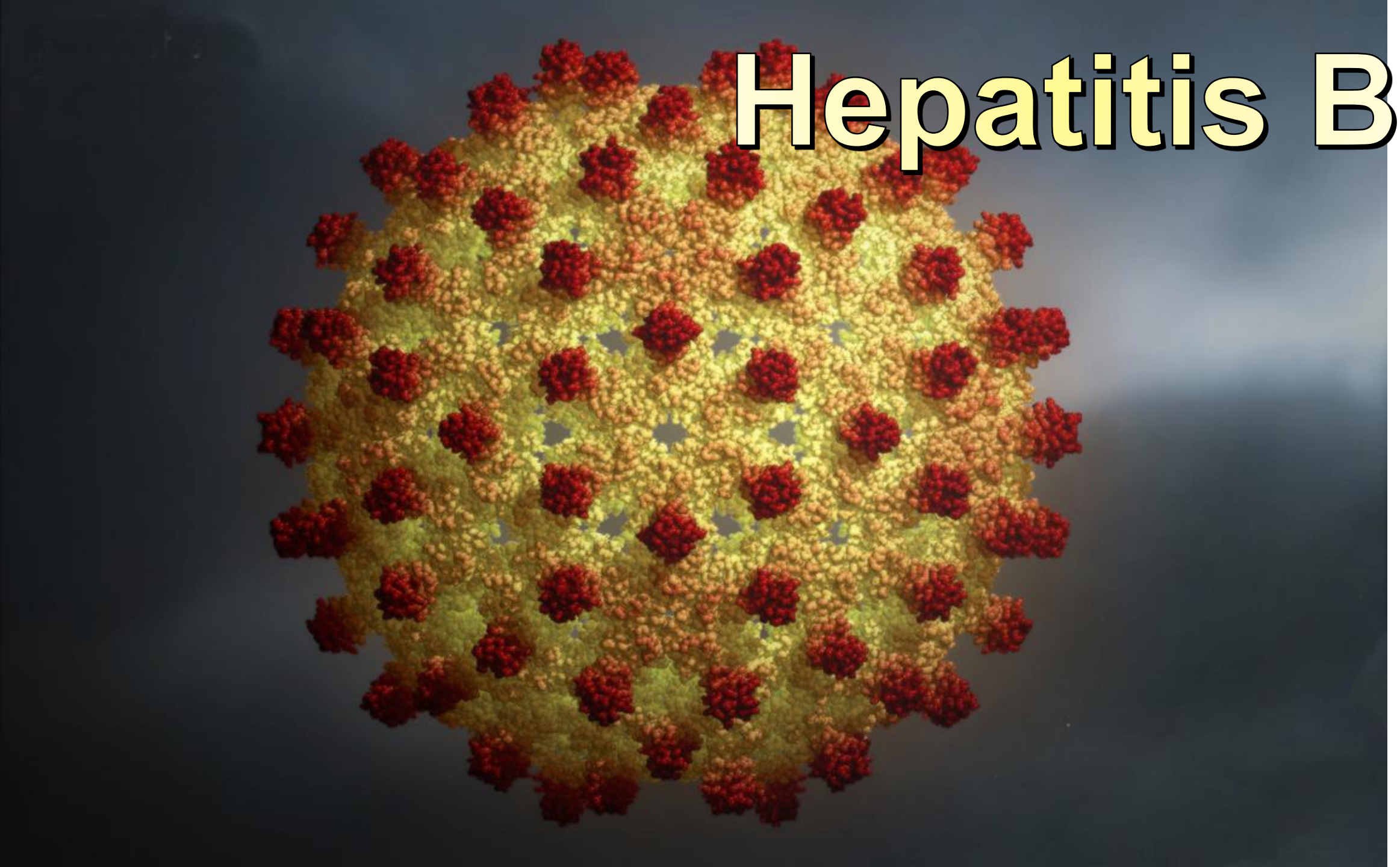
-hygienic measures and sanitation

-passive immunization(Human Immunoglobulin
Gamma globulin given before exposure to virus or early during the incubation period, will prevent or attenuate a clinical illness.

-active immunization
Several inactivated or live attenuated vaccines against hepatitis A have been developed.

❖Treatment:

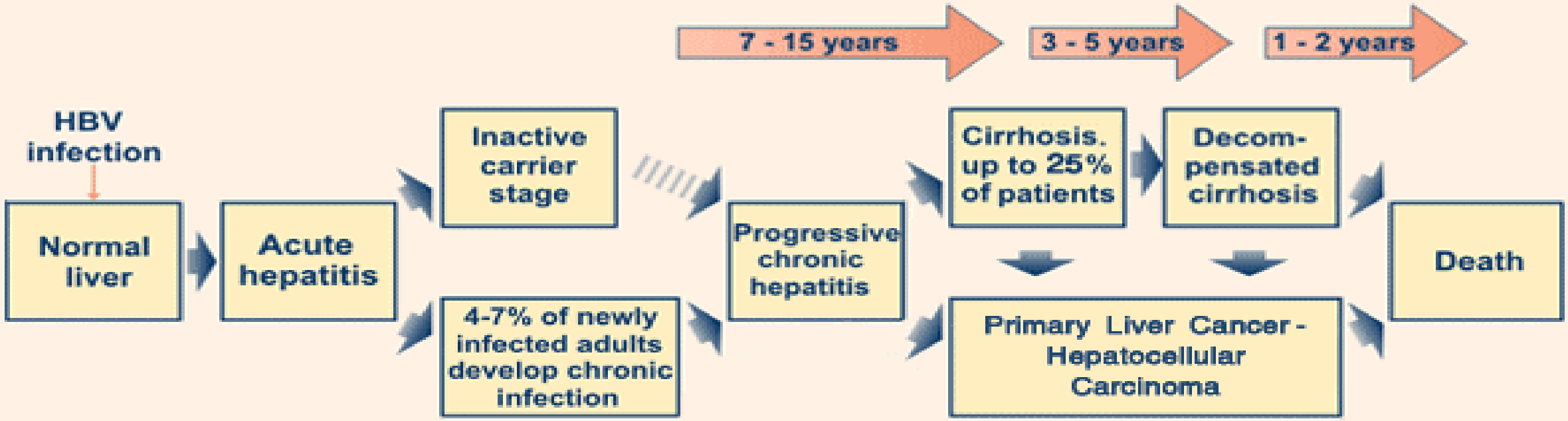
-nospecific, dietary food and long rest


Hepatitis B

❖Hepatitis B

- ✓**Hepatitis B (formerly known as “serum” hepatitis) is an acute systemic infection with major pathology in the liver, caused by hepatitis B virus.**
- ✓**Transmitted by the Parenteral route.**
- ✓**The acute illness causes liver inflammation, vomiting, jaundice, and, rarely, death. Chronic hepatitis B may eventually cause cirrhosis and liver cancer.**
- ✓**Hepatitis B is endemic throughout the world, especially in tropical & developing countries.**

Natural History of Hepatitis B Infection



Treatment with infusion of cultured hepatoblasts and/or cultured hepatocytes is indicated at the Hepatitis B stages from chronic hepatitis to decompensated cirrhosis except for liver cancer

- reversing or slowing down the diseaase with the cell therapy

© Institute of Longevity and Preventive Medicine

Hepatitis B virus has affinity to different tissues although it usually affects the liver, but the viral DNA and proteins are also found in the kidneys, spleen, pancreas, skin, bone marrow and peripheral blood mononuclear cells.

Peripheral mononuclear cells may be the first targets in HBV infection.

The defeat of the blood cells may play a direct role in the development of aplastic anemia.

❖Epidemiology Determinants

➢Agent factor

a) AGENT: Hepatitis B Virus (HBV)

-It is a complex, 42 nm double-shelled DNA virus originally known as "Dane Particle".

-It replicates in liver cell.

HBV occurs in 3 morphology form in serum:

I. Small spherical particles with an average Diameter of 22nm.

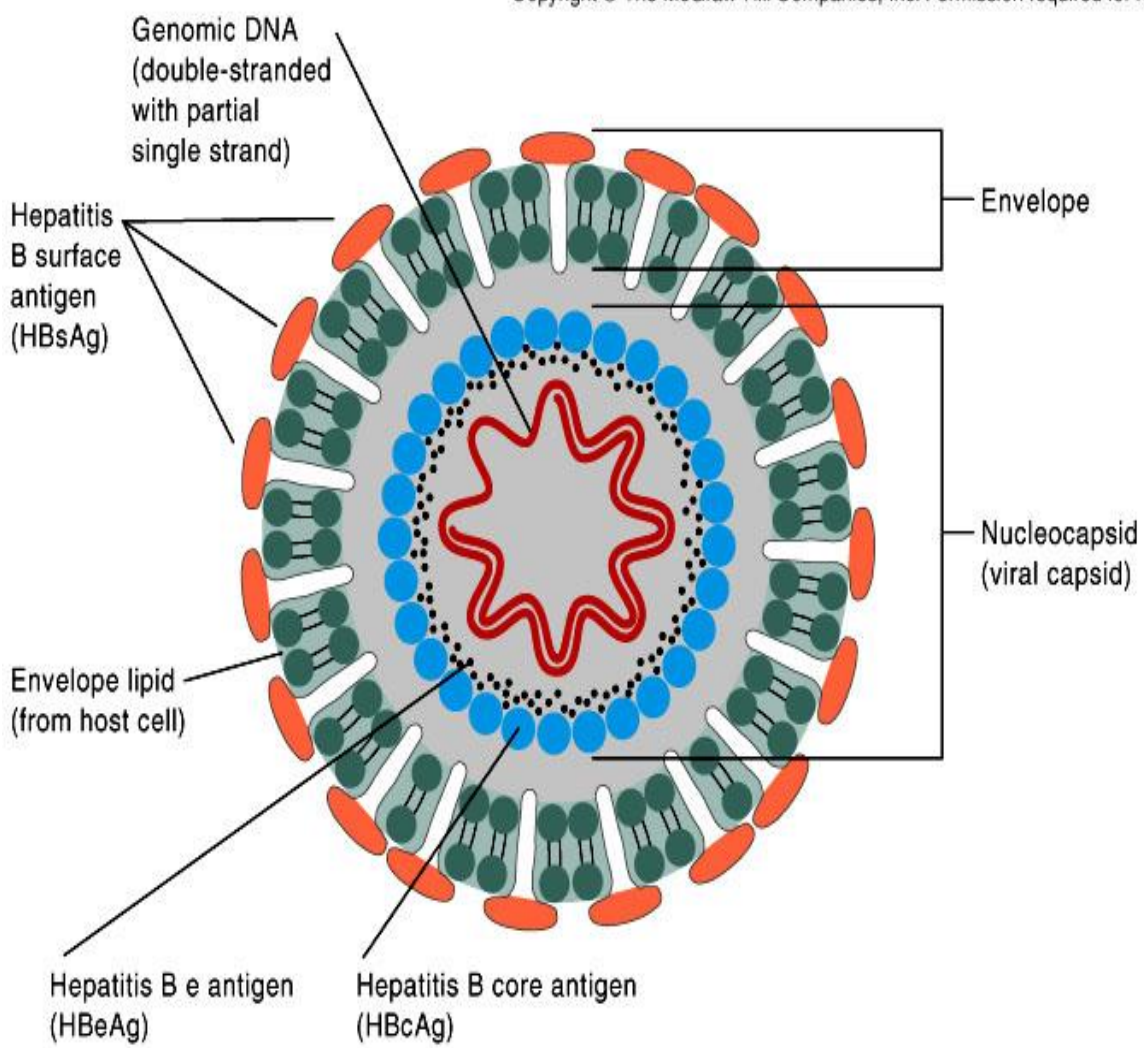
II. Filamentous or Tubules of varying length & of 22 nm diameter.

III. Dane particle.

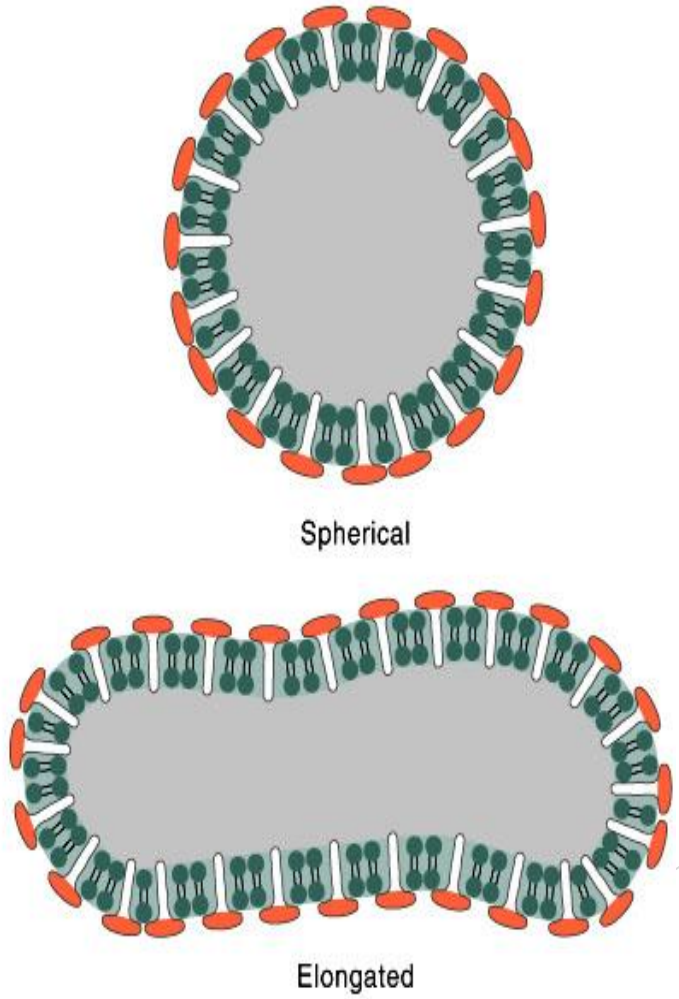
Out of 3 morphology forms, only the Dane particle is considered infectious, other circulating morphology forms are not infectious.

❖HBV : Structure

Copyright © The McGraw-Hill Companies, Inc. Permission required for reproduction or display.



(a) Complete infectious virion



(b) Viral envelope particles containing HBsAg

b)RESERVOIR OF INFECTION:

-Men is the only reservoir of infection which can be spread either from carriers or from cases.

c) Infective material:

-Contaminated blood is the main source,

-Virus has been found in body secretion such as saliva, vaginal secretion & Semen

in infected material.

d)Resistance:

-Readily destroyed by sodium hypochlorite, as is by heat sterilization in

an autoclave for 30-60 min.

c) Humoral and cellular response:

-HBV has 3 distinct antigen:

i. **HBsAg, also known as “Australian antigen,**
ii. **HBcAg antigen (core antigen)**
iii. **HBeAg envelope antigen**

They stimulate production of corresponding antibody.

❖Incubation Period

45-180 days (usually 60-90 days)

❖Mode of Transmission

- ✓ Parenteral- IV drug abusers, health workers are at increased risk.
- ✓ Sexual- sex workers and homosexuals are particular at risk.
- ✓ Perinatal (Vertical) - mother (HBeAg+) →infant. Mothers who are HBeAg positive are much more likely to transmit to their offspring than those who are not. Perinatal transmission is the main means of transmission in high prevalence populations.

By blood transfusion, usually obtained from an asymptomatic carrier or drugs (plasma, etc.).

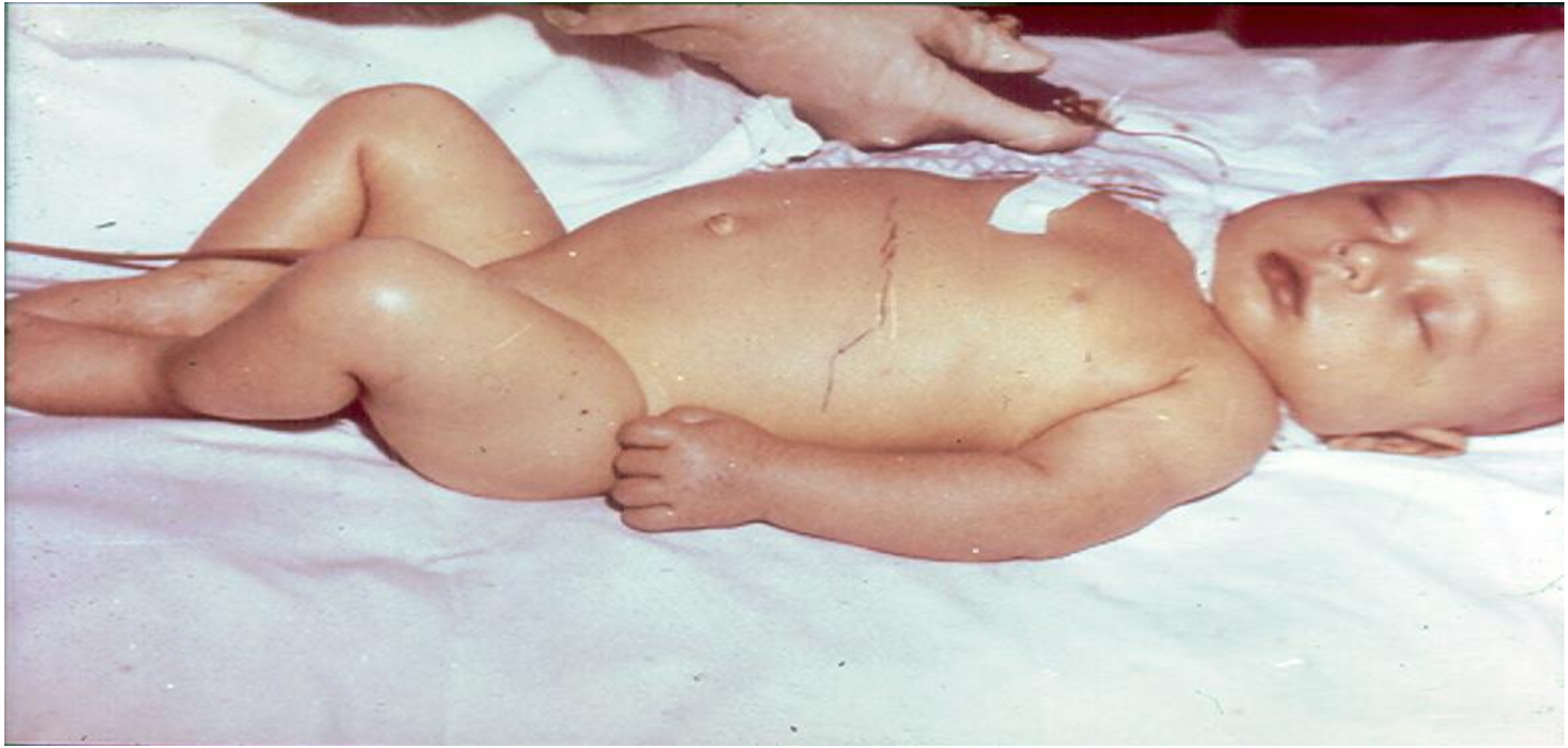
The hepatitis B virus is found in saliva swabs from the nose and throat.

with everyday communication

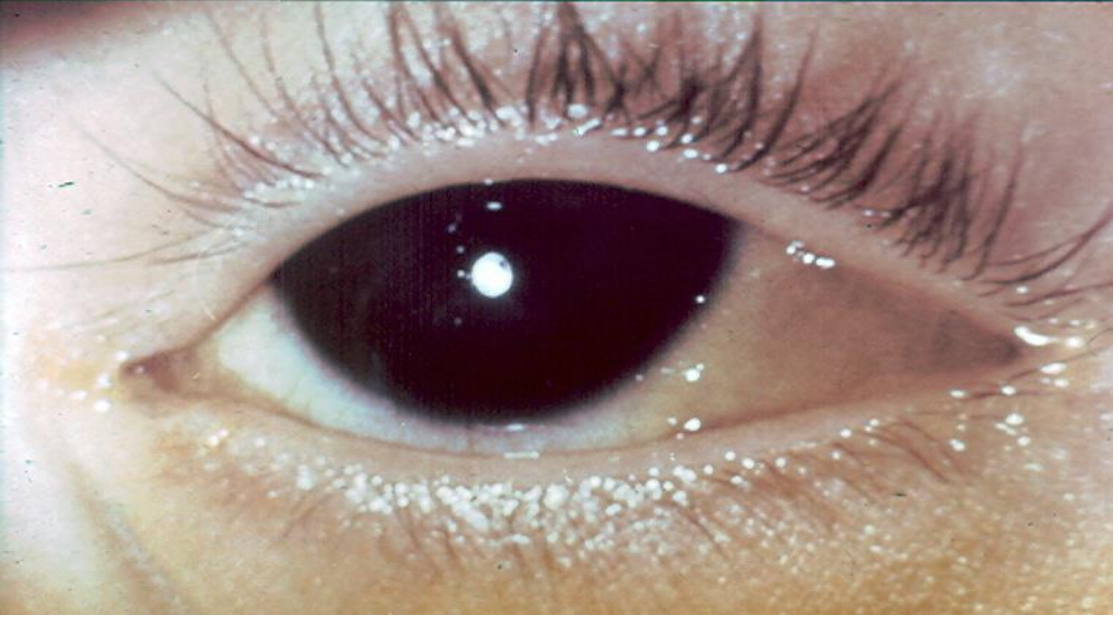
The sexual way. Damage to the skin or mucous membrane of the genitals, rectum or the mouth are the gateway to the virus present in the semen or discharge from the uterus of an infected sexual partner.
Among homosexuals antibodies to hepatitis B virus was detected in 30% of patients, and in men, with only heterosexual - 5%.
In recent years, the number of cases of heterosexual transmission of hepatitis B increased. Men - chronic carriers of hepatitis B, are a danger to their sexual partners, but are not a source of infection in everyday contacts.

Diagnosis of hepatitis is based on clinical, epidemiological and laboratory parameters

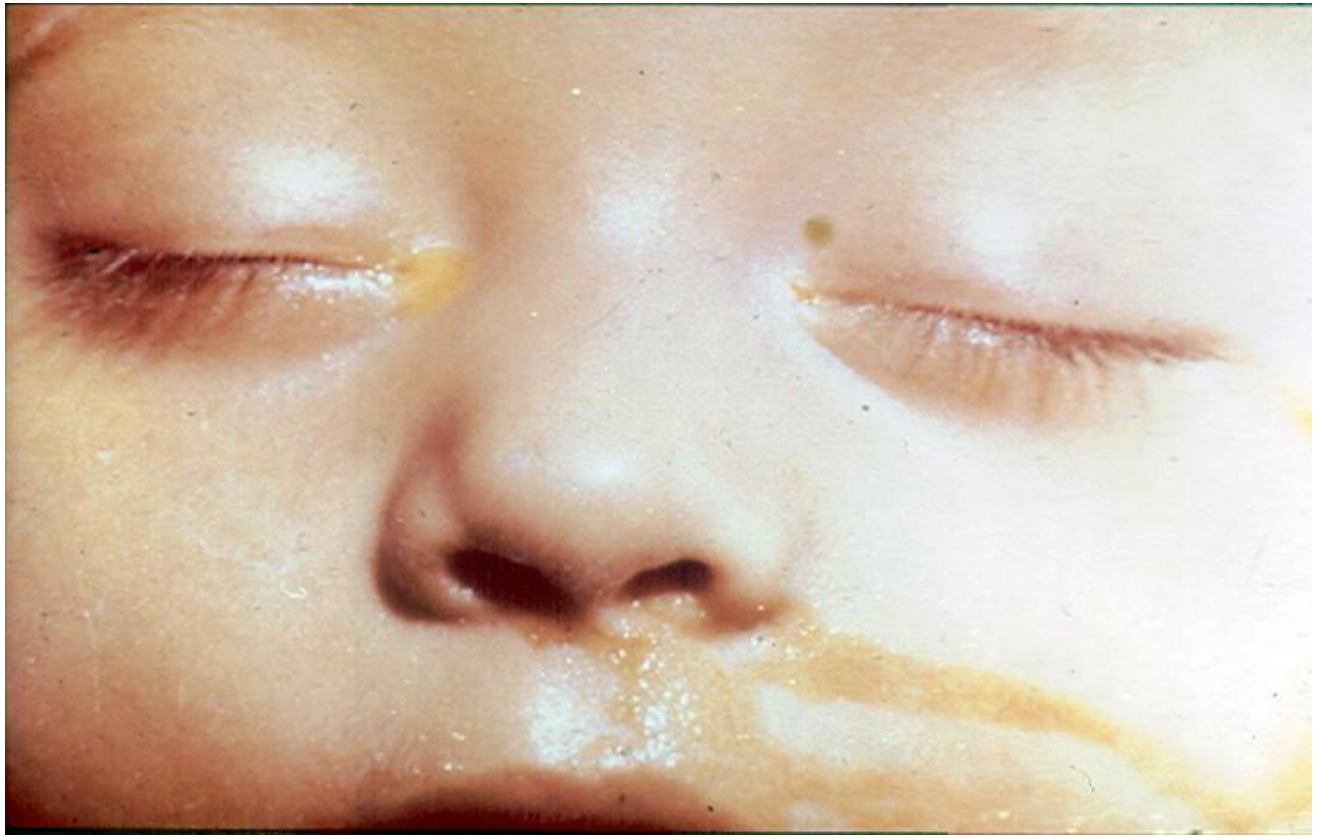
The transmission of hepatitis B virus from a pregnant woman (patient or bearer of a virus) to the fetus through the placenta, amniotic fluid, or at the time of passing through the birth canal. Transfer proshodit through poorly sterilized syringes, needles, scalpels, during tattooing. Often sick, surgeons, pathologists, dentists, nurses, laboratory workers, the staff of blood transfusion stations and others.



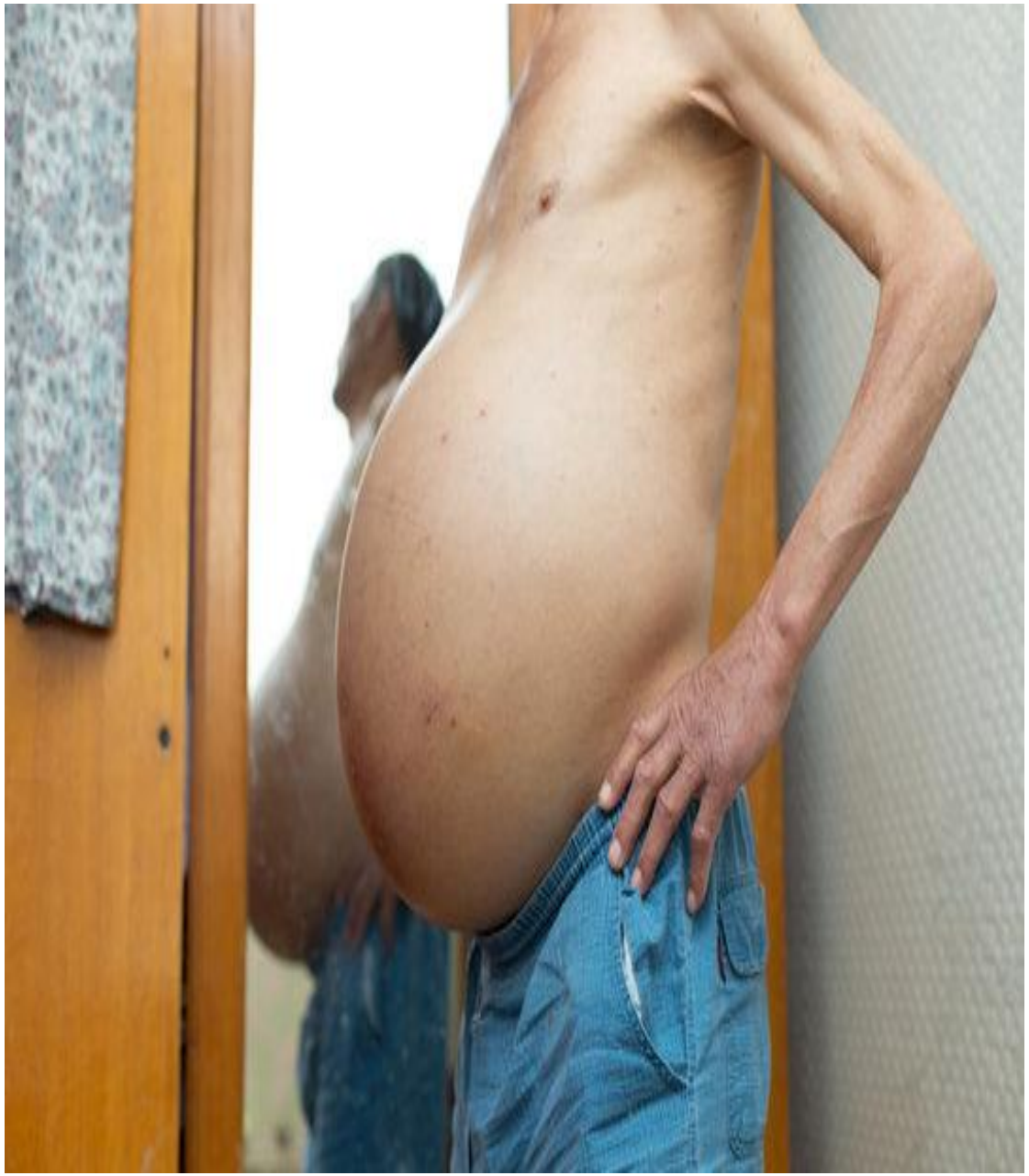
Viral hepatitis B. Fulminant form



Hepatic coma.
Floating eyeball



Hepatic coma. Vomiting "coffee grounds"



Cirrhosis

❖Diagnosis

- ✓ Serology
- ✓ Liver Chemistry tests
  - AST, ALT, ALP, and total Bilirubin
- ✓ Histology--Immunoperoxidase staining
- ✓ HBV Viral DNA--Most accurate marker of viral DNA and detected by PCR
- ✓ Liver Biopsy--to determine grade(Inflammation) and stage(Fibrosis) in chronic Hepatitis

1) HBsAg :- It is the first marker to appear in blood after infection.

2) Anti-HBs(HBsAb) :-Disappearance of HBsAg and the appearance of anti-HBs signals recovery from HBV infection, non-infectivity.

3) Anti-HBc :- IgM anti-HBc appears shortly after HBsAg is detect (HBcAg alone dose not appear in serum)
IgM-HBc may also or can persist for 3-6 months or longer.
IgG-HBc also appear during acute hepatitis B but persist indefinitely.

4) HBeAg :-

→ HBeAg appear in blood concurrently with HBsAg, or soon afterwards.
→ HBeAg is a soluble protein found only in HBeAg positive serum.
→ HBeAg indicate viral replication and infectivity.
→ Persistence of HBeAg in serum beyond 3 month indicate an increased like hood of chronic hepatitis B.

❖ Interpretation of common serological patterns in HBV infection

Virus/Antibody markers					Interpretation
HBsAg	HBeAg	Anti-HBc	Anti-HBs	Anti-HBe	
+	+	IgM	-	-	Acute HBV infection; highly infectious
+	+	IgG	-	-	Late/Chronic HBV infection or carrier state; highly infectivity
+	-	IgG	-	+/-	Late/Chronic HBV infection or carrier state; low infectivity
-	+/-	IgM	-	+/-	Seen rarely in early acute HBV infection; infectious
-	-	IgG	+/-	+/-	Remote HBV infection; infectivity nil or very low
-	-	-	+	-	Immunity following HBV vaccine

❖Prevention

❑ Vaccination

- highly effective recombinant vaccines

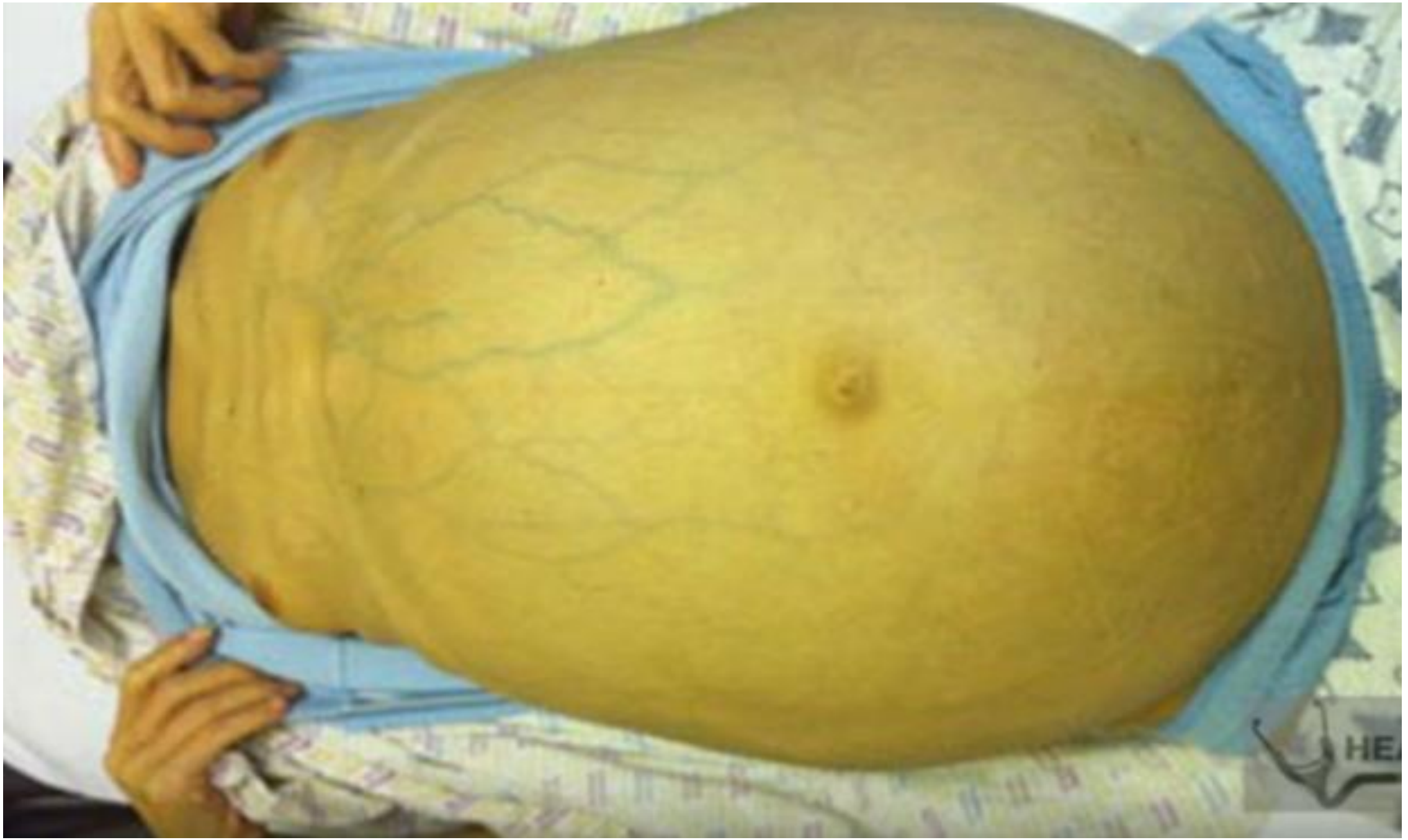
❑ Hepatitis B Immunoglobulin (HBIG)

-exposed within 48 hours of the incident/

neonates whose mothers are HBsAg and HBeAg positive.

❑ Other measures

-screening of blood donors, blood and body fluid precautions.



❖National Immunization Schedule

Vaccine	When to Give	Dose	Route	Site
Hepatitis B	**At birth or as soon as possible with in 12 hours.**	**0.5 ml**	**IM**	**Antero lateral side of mid thigh**
Hepatitis B 1,2,3	**At 2,4,6 months**	**0.5 ml**	**IM**	**Antero lateral side of mid thigh**

❖Treatment

- ✓ **Interferon Alfa (Intron A)**
  Response rate is 30 to 40%.
- ✓ **Lamivudine (Epivir HBV)**
  (relapse ,drug resistance)
- ✓ **Adefovir dipivoxil (Hepsera)**

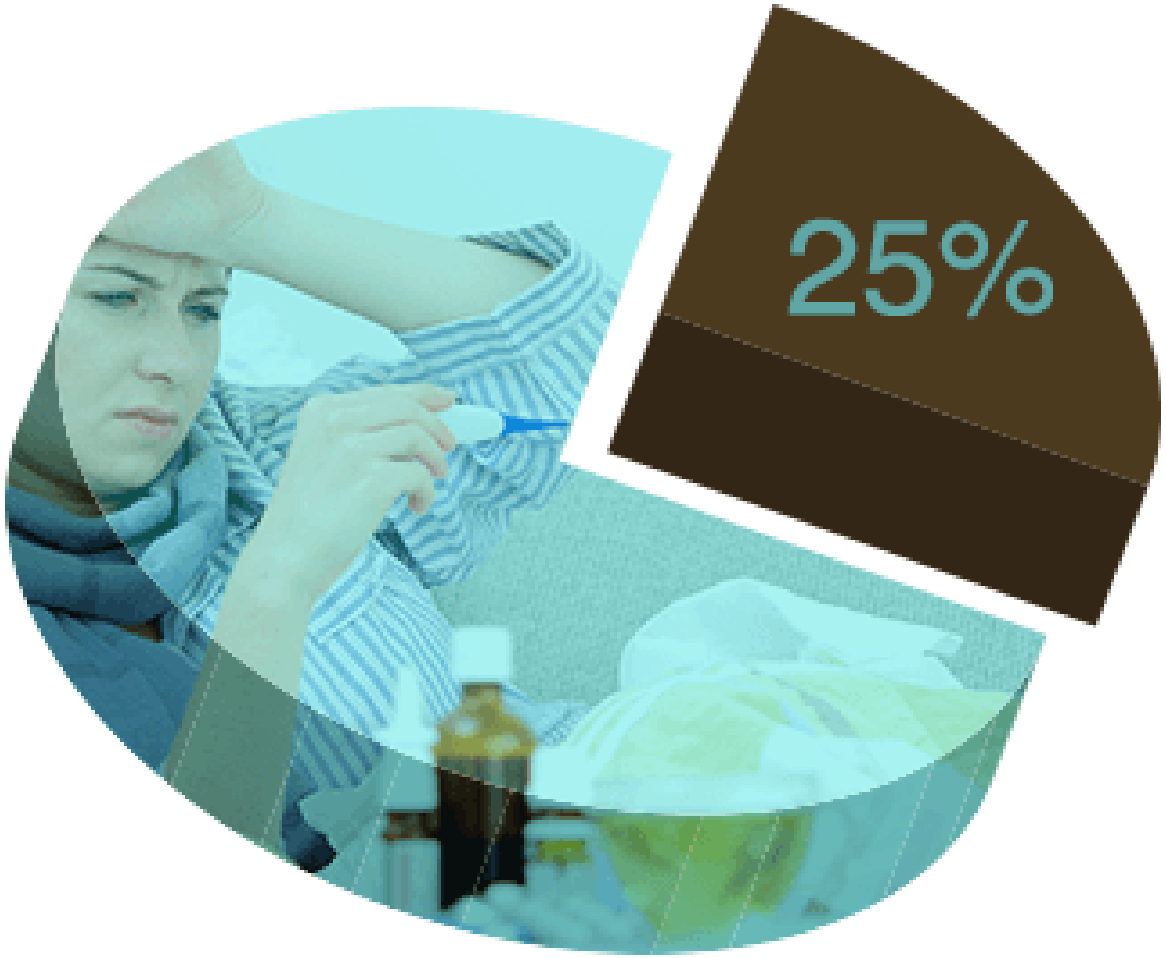
There are six treatment options approved by the U.S. Food and Drug Administration (FDA) available for persons with a chronic hepatitis B infection: alpha-interferon, pegylated interferon, adefovir, entecavir, telbivudine andlamivudine. About 65% of persons on treatment achieve a sustained response.

Hepatitis C

❖Hepatitis C

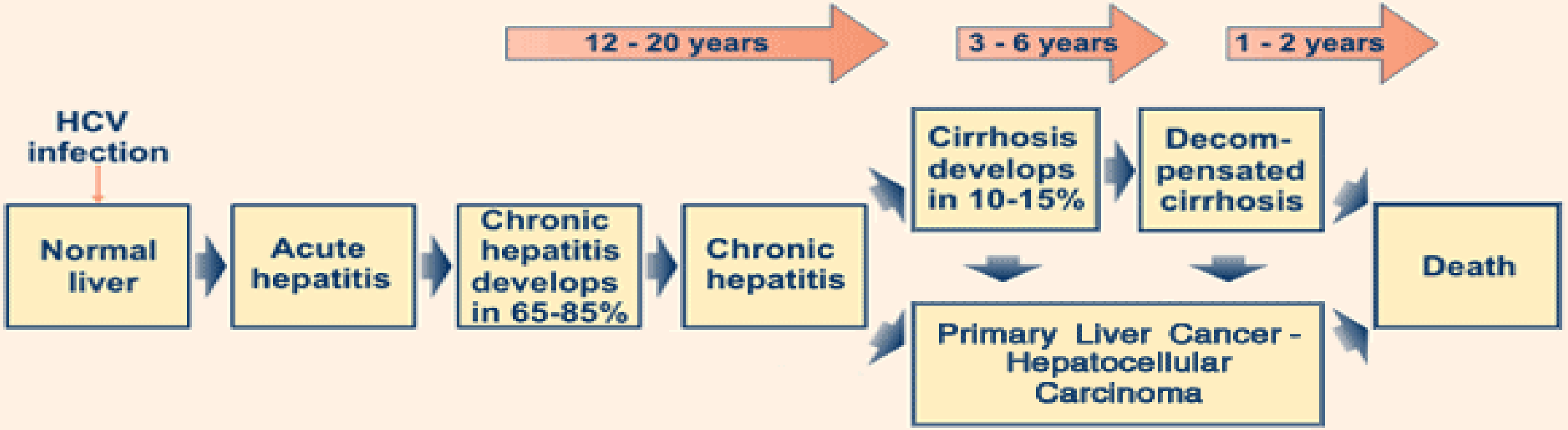
- ✓ Hepatitis C is an infectious disease affecting primarily the liver, caused by the hepatitis C virus (HCV).
- ✓ The infection is often asymptomatic, but chronic infection can lead to scarring of the liver and ultimately to cirrhosis, which is generally apparent after many years.
- ✓ It is estimated that 150-200 million people, or ~3% of the world's population, are living with chronic hepatitis C.
- ✓ Overt jaundice is seen in about 5 % of patients only.
- ✓ The important part in type C hepatitis is the chronic illness.
- ✓ About 50 to 80 % of patients progress to chronic hepatitis.

25% of all individuals infected with the acute version of hepatitis C will experience symptoms of:



- tiredness
- jaundice
- fever
- muscular aches

Natural History of Hepatitis C Infection



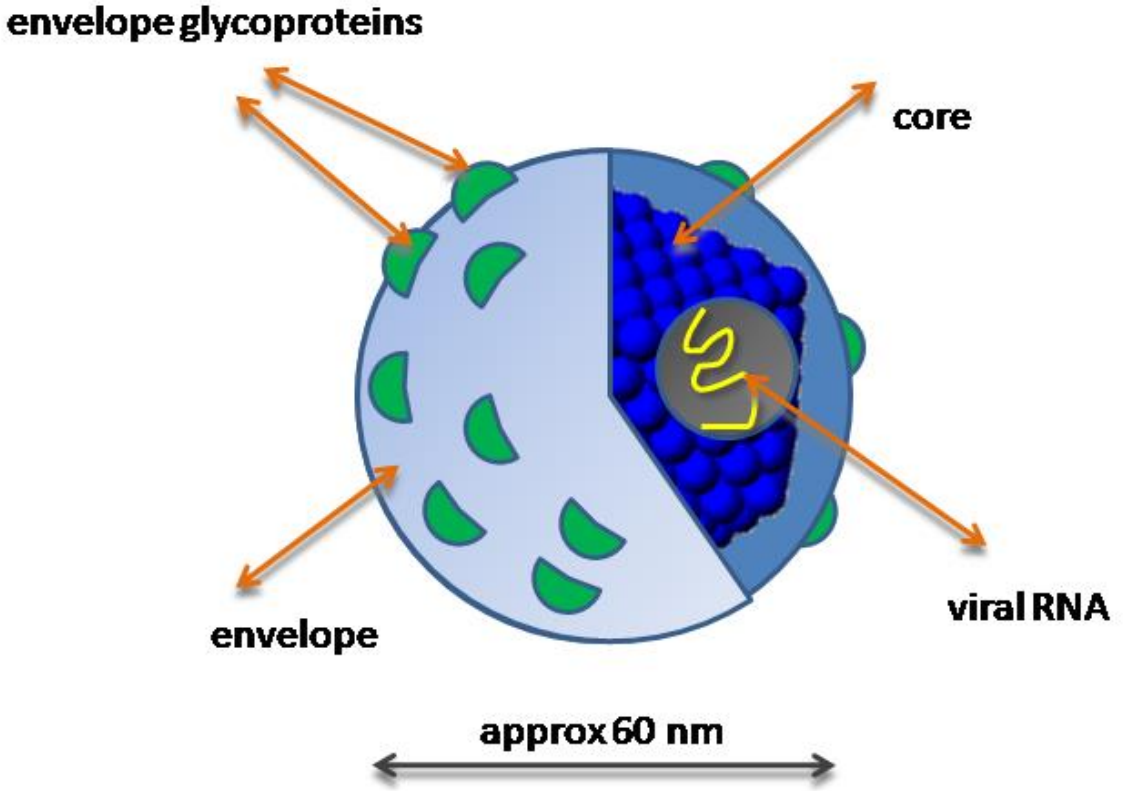
Treatment with infusion of cultured hepatoblasts and/or cultured hepatocytes is indicated at the Hepatitis C stages from chronic hepatitis to decompensated cirrhosis except for liver cancer

- reversing or slowing down the disease with the cell therapy

© Institute of Longevity and Preventive Medicine

❖Hepatitis C Virus

- ✓ HCV is a 50-60 nm virus with a linear, single stranded RNA genome, enclosed with in a core and surrounded by an envelope, carrying glycoprotein spikes.
- ✓ It is a member of the *Hepacivirus* genus in the family *Flaviviridae*.
- ✓ The half life of the virus particles in the serum is around 3 hours and may be as short as 45 minutes.
- ✓ In addition to replicating in the liver the virus can multiply in lymphocytes.



Structure of Hepatitis C Virus

❖Incubation Period

40-120 days

❖Mode of Transmission

- ✓ **Intravenous Drug Use**
- ✓ **Healthcare Exposure: Blood Transfusion, transfusion**
- ✓ **of Blood products, Organ Transplant without HCV screening carry significant risk of infection.**
- ✓ **Hemodialysis**
- ✓ **Accidental injuries with needles/sharps**
- ✓ **Sexual/household exposure to anti-HCV-positive contact**
- ✓ **Multiple sex partners**
- ✓ **Vertical Transmission: Vertical transmission of hepatitis C**
- ✓ **from an infected mother to her child**

❖Diagnosis

- HCV antibody – ELISA used to diagnose hepatitis C infection. Not useful in the acute phase as it takes at least 4 weeks after infection before antibody appears.

- HCV-RNA - various techniques are available e.g. PCR and branched DNA. May be used to diagnose HCV infection in the acute phase. However, its main use is in monitoring the response to antiviral therapy.

- HCV-antigen - an EIA for HCV antigen is available. It is used in the same capacity as HCV-RNA tests but is much easier to carry out.

❖Prevention

- ✓ Only General Prophylaxis, such as blood, tissue, organ screening, is possible.
- ✓ No specific active or passive immunizing agent is available.

❖Treatment

Interferon - may be considered for patients with chronic active hepatitis. The response rate is around 50% but 50% of responders will relapse upon withdrawal of treatment.

Ribavirin - there is less experience with ribavirin than interferon. However, recent studies suggest that a combination of interferon and ribavirin is more effective than interferon alone.

Treatment

- First-line treatment for acute HCV includes pegylated interferon plus ribavirin.
  - once-weekly **PEG-IFN** and a daily oral dose of **ribavirin in two divided doses**

Genotype	Pegylated-IFN Dose	weight	Ribavirin Dose	Duration
1	Peginterferon α2a 180 mcg/wk	Less than 75 Kg	1000 mg	48 weeks
	Peginterferon α2b 1.5 mcg/wk	More than 75 kg	1200 mg	
2,3	Peginterferon á2a 180 mcg/wk		800 mg	24 weeks
	Peginterferon α2b 1.5 mcg/wk			

At week 1, 2, 4 and then interval of 4-8 weeks monitor:

- **Symptom of Disease**
- **Side Effects of therapy**
- **Blood count**
- **Aminotransferases**

Interferon

- INF α 2 b:
  - Used for HBV and HBC infections
  - Half life : 2-3 hours
  - Dose: 3 MIU subcutaneous 3 times /week
    - Geno-1: 4/48 weeks
    - Geno2: 3/24 weeks
- Peg IFN α2 a: with branched peg chain
  - Used for HBV and HVC infections
  - Half life: 160 hours
  - Dose: 180 mcg s/c once weekly
    - Geno-1 4/48
    - Geno-2 3/24
- PegIFN -α2b : linear peg chain
  - Half life: 40 hours
  - Dose 1.5 mcg/kg s/c once weekly
- IFN alfaxon-1:
  - Used for HCV treatment
  - Dose:
    - naïve: 9 mcg
    - Non responder: 15 mcg s/c 3 times a week
    - Naïve for 24 weeks
    - INF non responder: 48 weeks

Hepatitis D

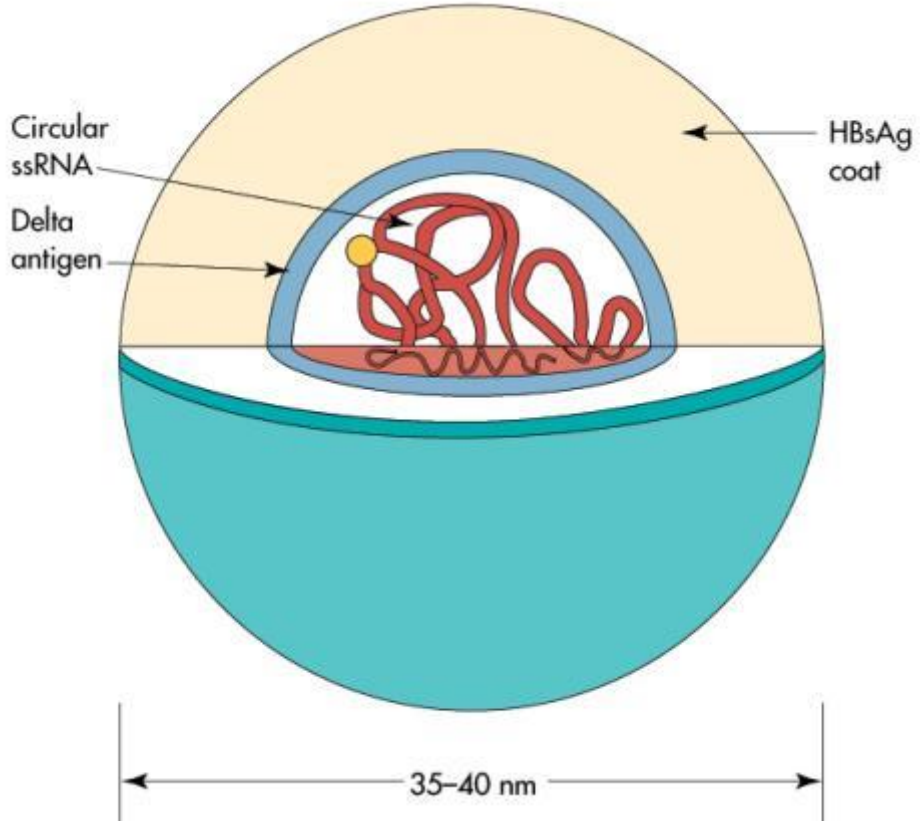
❖Hepatitis D

- ✓ **Hepatitis D**, also referred to as **hepatitis D virus** (**HDV**) and classified as *Hepatitis delta virus*, is a disease caused by a small circular enveloped RNA virus.
- ✓ HDV is considered to be a subviral satellite because it can propagate only in the presence of the hepatitis B virus (HBV).

❖Hepatitis D virus

VIRION: spherical, 36-38 nm particle with an outer coat composed of the HBsAg surrounding ssRNA genome.

Satellite virus : replicates only in the presence of HBV



❖Incubation Period

2-12 weeks

❖Mode of Transmission

The primary route of Transmission are believed to be similar to those of HBV, though HDV does not appear to be sexually transmitted disease.

❖Clinical Features

- ✓ Infection is dependent on HBV replication, as HBV provides an HBsAg envelop for HDV.
- ✓ Two types of infection are recognisesd, coinfection and superinfection.

In Coinfection, delta and HBV are transmitted together at the same time.

In Superinfection, delta infection occurs in a person already harbouring HBV.

❖Diagnosis

- ✓ **Delta antigen is primarily expressed in liver cell nuclei, where it can be demonstrated by immunofluorescence.**
- ✓ **Anti-delta antibodies appear in serum and can be identified by ELISA.**
  IgM antibody appears 2-3 weeks after infection and is soon replaced by the IgG antibody in acute delta infection.

❖Prevention

- HBV-HDV Coinfection

  Pre or post exposure prophylaxis to prevent HBV infection. Screening of blood donor for HBsAg.

- HBV-HDV Superinfection

  Education to reduce risk behaviors among persons with chronic HBV infection.

Hepatitis E

❖Hepatitis E

- ✓ **Hepatitis E is a viral hepatitis (liver inflammation) caused by infection with a virus called hepatitis E virus (HEV).**
- ✓ **Although Hepatitis E often causes an acute and self-limiting infection (in that it usually goes away by itself and the patient recovers) with low mortality rates.**
- ✓ **It bears a high risk of developing chronic hepatitis in immunocompromised patients with substantial mortality rates.**
- ✓ **Hepatitis E occasionally develops into an acute, severe liver disease, and is fatal in about 2% of all cases.**
- ✓ **In pregnant women the disease is more often severe and is associated with a clinical syndrome called fulminant hepatic failure.**

❖Signs and Symptoms

Acute Infections:

→ The incubation period of hepatitis E varies from 3 to 8 weeks.

→ After a short prodromal phase symptoms lasting from days to weeks follow. They may include jaundice, fatigue and nausea.

→Viral RNA becomes detectable in stool and blood serum during incubation period.

→Serum IgM and IgG antibodies against HEV appear just before onset of clinical symptoms.

→Recovery leads to virus clearance from the blood, while the virus may persist in stool for much longer.

→Recovery is also marked by disappearance of IgM antibodies and increase of levels of IgG antibodies.

Chronic Infections:

→While usually an acute disease, in immunocompromised subjects—particularly in solid organ transplanted patients—hepatitis E may cause a chronic infection.

→Occasionally this may cause liver fibrosis and cirrhosis.

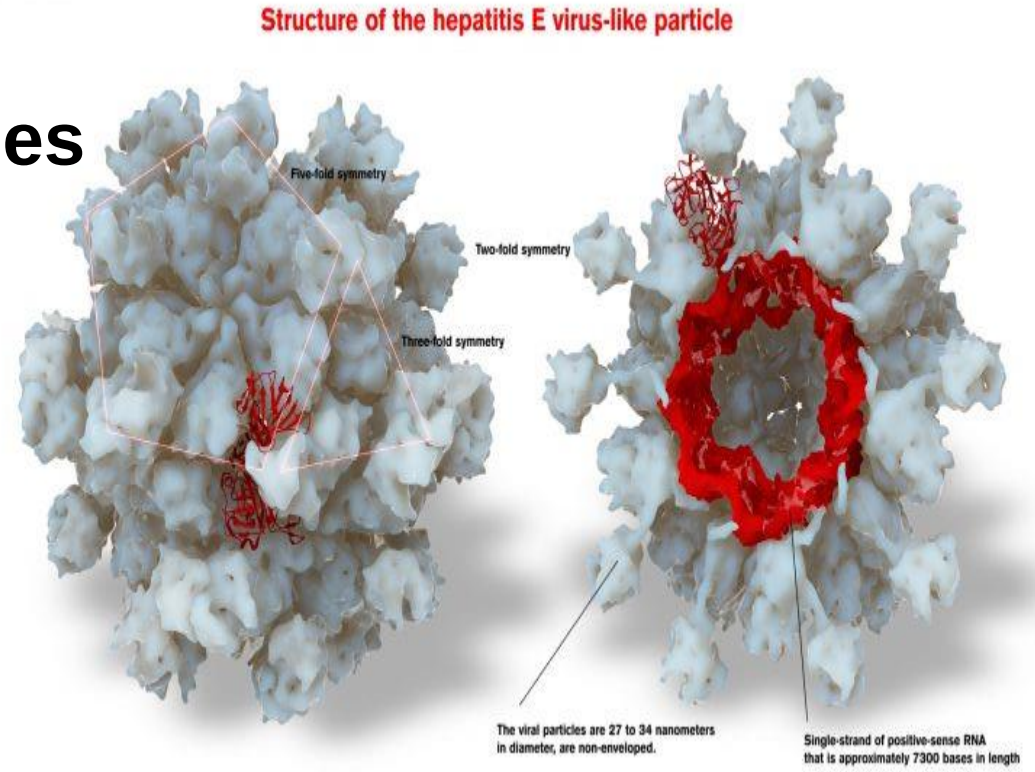
❖Hepatitis E virus

- ✓ **HEV is spherical nonenveloped virus, 29-nm to 32 nm in diameter, with a ssRNA genome.**
- ✓ **The surface of the virion shows indentation and spikes.**
- ✓ **The Virus is very labile.**
- ✓ **Il has been classified in the genus Herpes virus under the family Caliciviridae.**

❖Animal Reservoir: Pigs

❖Incubation Period

2-9 weeks



❖Mode of Transmission

It is spread mainly by the fecal-oral route due to fecal contamination of water supplies or food; person-to-person transmission is uncommon.

❖Diagnosis

ELISA kits are available for IgG and IgM antibodies, using recombinant and synthetic peptide antigens.

❖Prevention

Sanitation:

Avoid drinking water of unknown purity, uncooked shellfish, and uncooked fruit/vegetables not peeled or prepared by traveler.

❖Hepatitis G

- ✓ **GB virus C** (**GBV-C**), formerly known as **hepatitis G virus** (**HGV**) and also known as HPgV is a virus in the *Flaviviridae* family and a member of the *Pegivirus* genus, is known to infect humans, but is not known to cause human disease.
- ✓ HGV RNA has been found in patients with acute, chronic and fulminant hepatitis, hemophiliacs, patients with multiple transfusions and hemodialysis, intravenous drug addicts and blood donors.