MICROBIOLOGY

بسم الله الرحمن الرحيم

Final – Lecture #2

# viral hepatitis

﴿ وَإِن تَتَوَلَّوْا يَسْتَبْدِلْ قَوْمًا غَيْرَكُمْ ثُمَّ لَا يَكُونُوا أَمْثَالَكُم ﴾

اللهم استعملنا ولا تستبدلنا


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# Objectives

- Discuss the structure, properties, epidemiology, clinical presentation, laboratory diagnosis, and treatment of:

5 major groups

1. Hepatitis A (HAV)
2. Hepatitis B (HBV)
3. Hepatitis C (HCV)
4. Hepatitis D (HDV)
5. Hepatitis E (HEV)

# Introduction

- Hepatitis: inflammation of liver; presence of inflammatory cells in organ tissue
- The causes of hepatitis are varied and include viruses, bacteria, and protozoa, as well as drugs and toxins (eg, isoniazid, carbon tetrachloride, and ethanol).
- Acute hepatitis: symptoms last less than 6 months
- Viral Hepatitis: is inflammation of the liver induced by viral infections
- The clinical symptoms and course of acute viral hepatitis can be similar, regardless of etiology, and determination of a specific cause depends on laboratory tests.

Inflammation

Doesn't always mean that there is an infection

Which Indicates

3 Infective causes:

Most common cause

Ameba can cause hepatic apsis

+ Autoimmune diseases

In Chronic >> last more

Between viruses

Most common

- Hepatitis may be caused by at least five different viruses (A, B, C, D, E) Other viruses, such as Epstein–Barr virus and cytomegalovirus, can also cause inflammation of the liver, but hepatitis is not the primary disease caused by them.

Primary disease → hepatitis

Rarely

primary disease → Inflammation in other organs

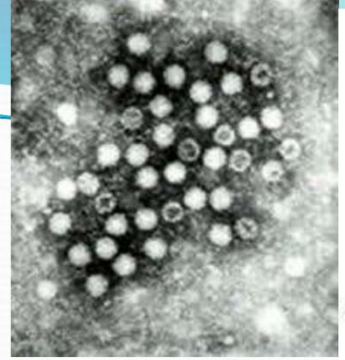
# Hepatitis A

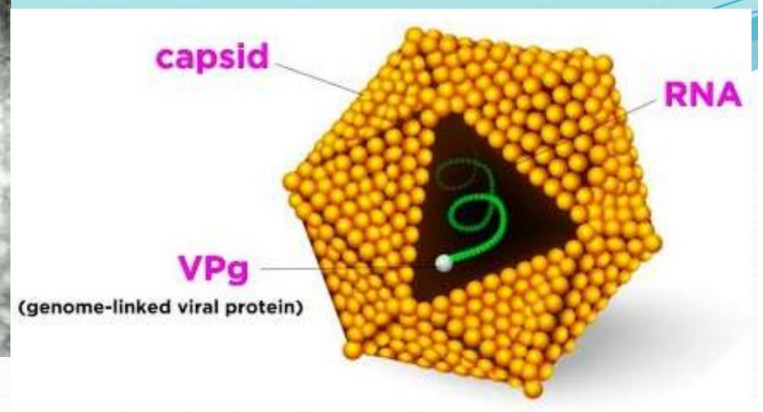
- HAV is a picornavirus,. It replicates in the liver, is excreted in bile and is then excreted in the faeces of infected persons for about 1) 2 weeks before the onset of 2) clinical illness and for up to 3) 7 days after.

Can be transmitted: Symptoms

- Hepatitis A virus is spread by the fecal–oral route, and outbreaks may be associated with contaminated food or water.
- Humans appear to be the major natural hosts of hepatitis A virus.
- The major mode of spread of hepatitis A is fecal–oral. transmission
- HAV particles can be demonstrated in the faeces by electron microscopy.

# Structure






- 27- to 32-nm spherical particle
- linear single-stranded RNA
- Terminal viral protein (VP) — Genome attached with
- Icosahedral Cubic symmetry
- Nonenveloped — Its capsid isn't that much complex
- Only one serotype is known — Infectious to humans
- HAV is stable to treatment with 20% ether, acid (pH 1.0 for 2 hours), and heat (60°C for 1 hour)
- Heating food to above 85°C (185°F) for 1 minute and disinfecting surfaces with sodium hypochlorite are necessary to inactivate HAV — For prevention purposes:

# Epidemiology

- Hepatitis A is the most common type of viral hepatitis occurring world-wide, often in epidemics.
- The disease is commonly seen in children and young adults. Older people are less vulnerable to have the disease than younger, due to their strong immunity they gain by time..
- Spread of infection is mainly by the faeco-oral route and arises from the ingestion of contaminated food or water .
- Overcrowding and poor sanitation facilitate spread. + no proper control on food and drinks.
- There is no carrier state. One week after the end of the symptoms (that last for~6 months), the patient is not infectious any more!

Continuous, high transmission

- More than 90% of the adult population in many developing countries shows evidence of previous hepatitis A infection. Travelers from developed countries who enter endemic areas are particularly susceptible.
- Patients are most contagious in the 1 to 2 weeks prior to the onset of clinical disease. Hard to discover

Doctor said:

The evidence: Anti hepatitis (A) antibody (IgG) test: if +ve >> the person had been infected by the virus with or without symptoms, so he produced its antibodies.

# Clinical Manifestations

General symptoms:

- Incubation period of 10 to 50 days — Prolonged
- Followed by the onset of fever; anorexia; nausea; pain in the right upper abdominal quadrant; — Where the liver is located

Specific symptoms:

- Within several days, jaundice. Dark urine and clay-colored stools may be noticed by the patient 1 to 5 days before the onset of clinical jaundice. — Yellow discoloration of skin and mucus membrane (different from "pallor" cased by anemia)
- The liver is enlarged and tender
- Recovery occurs in days to weeks. Almost all cases (99%) of hepatitis A are self-limiting. Chronic hepatitis such as that seen with hepatitis B is very rare.
- Many persons who have serologic evidence of acute hepatitis A infection are asymptomatic or only mildly ill, without jaundice. — Presence of (IgM) antibodies

High bilirubin → inflammation → Cells under stress → Liver's ability to perform its normal function is deteriorating → Enzymes and bilirubin increase → Jaundice appears

# Diagnosis

Enzymes

- Liver biochemistry: A raised serum AST or ALT, which can sometimes be very high, precedes the jaundice. In the icteric stage (When jaundice appear) the serum bilirubin reflects the level of jaundice.
- Haematological tests There is leucopenia (↓ WBCs) with a relative lymphocytosis. (Due to viral infection → Bone marrow produces lymphocytes more than other types of cells)
- The erythrocyte sedimentation rate (ESR) is raised. (Inflammation دلالة على)
- Viral markers: antibodies to HAV IgG (Previous long term infection/immune) antibodies are common in the general population over the age of 50 years, but an anti-HAV IgM (Currently) means an acute infection.
- Immune electron microscopic identification of the virus in fecal specimens and isolation of the virus in cell cultures remain research tools.

IgM ↑ → viral PCR +ve → bilirubin and liver enzymes ↑ → recovery → immune → IgG ↑

# Regarding Hepatitis A virus

- Is there any need to do PCR, and know if the virus is in the blood or not?

  There is a case in viremia, with low percentage (20%-30%) that the virus could be +ve in blood.

- But the original location of the virus : is in the liver → So we have to do liver aspirate (biopsy)
  Or in the stool → So we do PCR of stool

- HAV is not commonly exist in blood.
- The presence of the virus in stool doesn't mean that the HAV is definitely there !

  Maybe in stool but **1) not currently in inflammation state** or **2) finished the acute state, but still shedding (to finish the weeks after symptoms)**

- CBC + liver function test are mandatory:

  If enzymes increased → Inflammation and high stress on liver → Bilirubin ↑ Jaundice ↑

- **Specific test is sufficient to know** : IgM (HA) in acute Or IgG (HA) in immune or previous infection
- PCR for stool اذا شككنا بالتشخيص بنعمل

# Treatment and Prevention

We don't give antivirals for hepatitis A since 99% of cases recover fully. Symptoms vary, lasting from a week to a month, with some experiencing mild symptoms and others more severe jaundice.

- There is no specific treatment for patients with acute hepatitis A. Supportive measures include adequate nutrition and rest.

Patients are typically advised to rest, maintain good nutrition, and avoid foods that strain the liver and gallbladder.

- Avoidance of exposure to contaminated food or water are important measures to reduce the risk of hepatitis A infection.
- Passive immunization with Immune serum globulin (ISG), is protective if given before or during the incubation period of the disease.

Explanation in the next slide

- Active immunization with formalin-killed vaccines induce antibody titers similar to those of wild-virus infection and are almost 100% protective.

There are exceptions

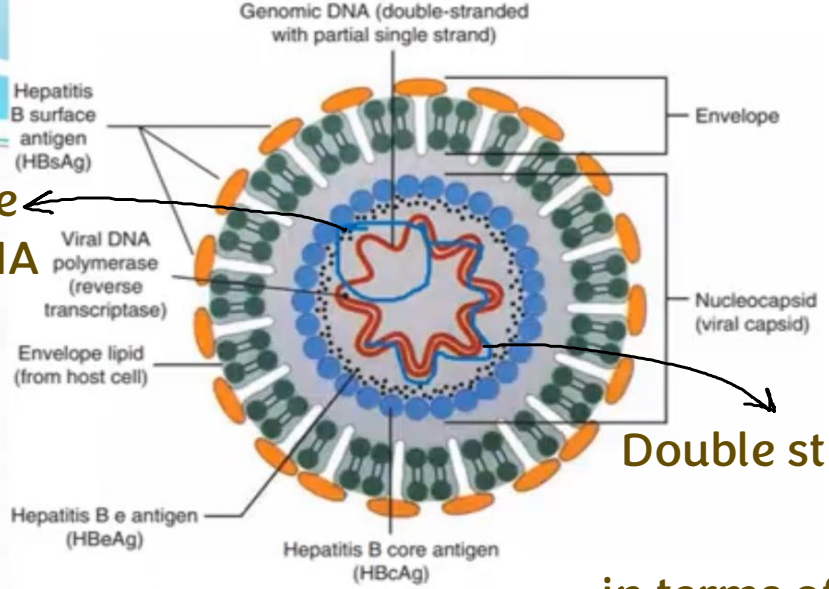
The vaccine form effective antibodies and immune memory, enabling the body to quickly fight the virus. High-risk individuals are advised to get vaccinated.

- Passive immunization with Immune serum globulin (ISG), is protective if given before or during the incubation period of the disease.

Passive immunization involves administering general antibodies (IVIg) or specific immunoglobulins. IVIg contains a broad collection of antibodies from donors, boosting the immune system to combat hepatitis A.

Specific immunoglobulins are derived from individuals who recovered from hepatitis A, purified for targeted use.
To be effective, these must be given during the incubation period before symptoms appear. For example, if a 15-year-old with hepatitis A is diagnosed after symptoms (e.g., jaundice), no immunoglobulin is needed for them. However, for a high-risk household member (e.g., an 80-year-old immune-compromised diabetic), passive immunization can protect them before symptoms develop due to close contact.

# Hepatitis B




Short , single stranded DNA

Classification based → Double stranded DNA

Double stranded DNA

- The viral genome consists of partially double-stranded DNA with a short, single stranded piece. It comprises 3200 nucleotides, making it the smallest DNA virus known.

in terms of its genome size not the virus's size

- The main components of the virus include the core - hepatitis B core antigen (HBcAg) and the pre-core- hepatitis B e antigen (HBeAg), and the envelope of the virus contains the hepatitis B surface antigen (HBsAg)

within the nucleocapsid of the virus

- Hep B is usually an asymptomatic or limited illness with fever and jaundice for days to weeks. It becomes chronic in up to 10% of patients and may lead to cirrhosis or hepatocellular carcinoma.

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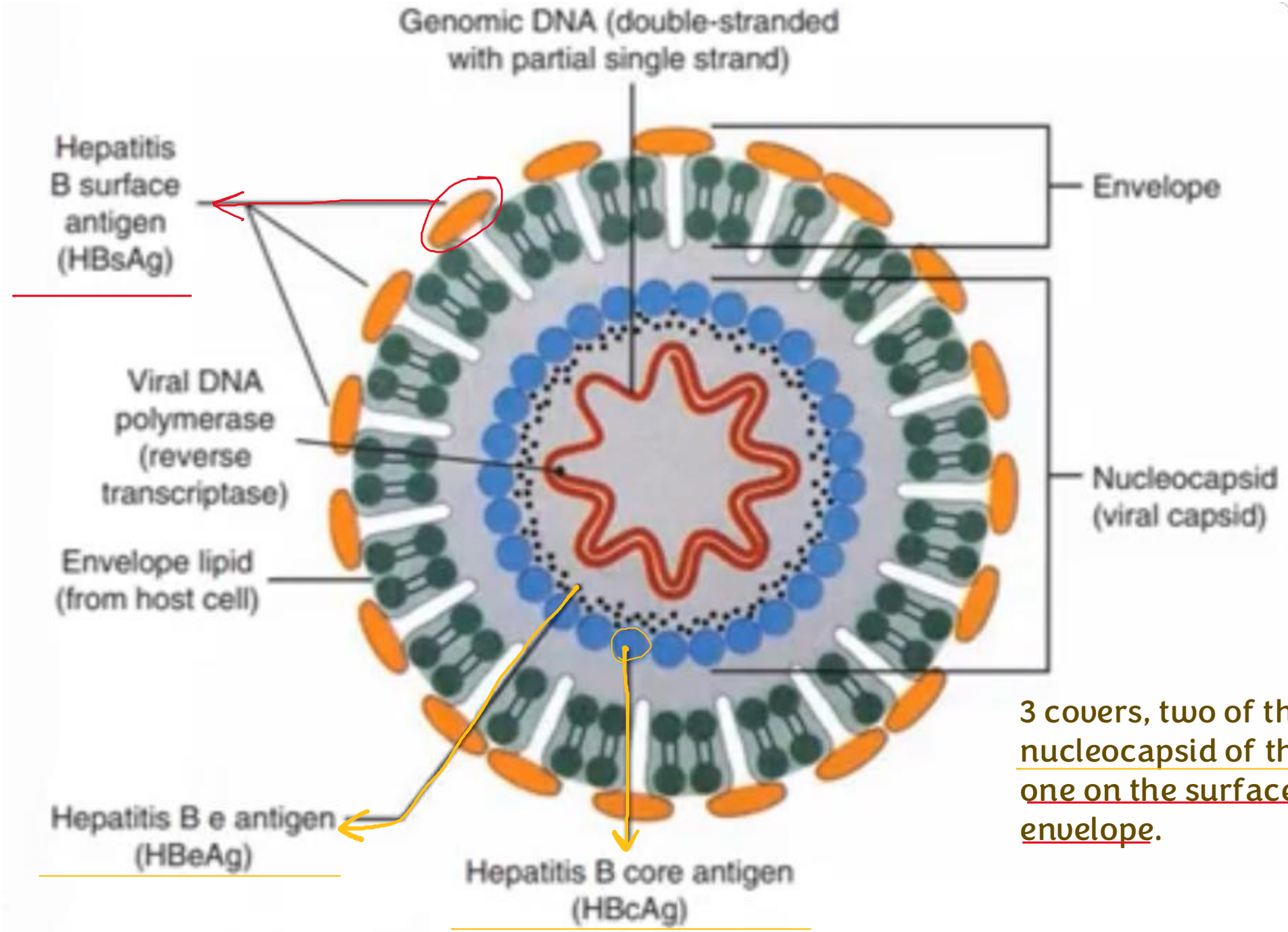


3 covers, two of them within the nucleocapsid of the virus and one on the surface of the envelope.

# Epidemiology

- The hepatitis B virus is present world-wide with an estimated 300 million carriers. 2 billion people have markers of infection
- 400 million have chronic infection.
- Spread of this virus is either by the intravenous route (e.g. by transfusion of infected blood or blood products, or by contaminated needles used by drug addicts, tattooists or acupuncturists), or by close personal contact, such as during sexual intercourse, particularly in male homosexuals.
- The virus can be found in semen and saliva.
- Vertical transmission from mother to child during parturition or soon after birth is the usual means of transmission world-wide.
- Needle stick injuries, has resulted in a higher risk of hepatitis B in medical personnel.

Most patients have the virus without symptoms. Symptoms may appear after a year, 10 years, or when the patient becomes immune-compromised.

Hemophilia patients receiving factor 8 or 9 may acquire hepatitis B if the factor is sourced from an infected donor.

Especially when they share the needle

Tattoos contain needles and if they are not in good condition, they can transmit the virus.

Like dentists , during the extraction process, bleeding may occur, which may result in contamination of the equipments used by the dentist.

Medical staff may accidentally injure themselves with a needle after it has contacted a hepatitis B-infected patient, leading to transmission.

doctors and medical students are advised to get the hepatitis B vaccine.

# Clinical Manifestations

- The incubation period may be as brief as 7 days or as long as 160 days (mean, approximately 10 weeks).
- Acute hepatitis B is usually manifested by the gradual onset of fatigue, loss of appetite, nausea and pain, and fullness in the right upper abdominal quadrant. Early in the course of disease, pain and swelling of the joints and occasional frank arthritis may occur. Some patients develop a rash.
- With increasing involvement of the liver, there is increasing cholestasis and, hence, clay-colored stools, darkening of the urine, and jaundice. Symptoms may persist for several months before finally resolving.

Most cases are mild or asymptomatic; (20–30)% develop acute hepatitis, 10% chronic hepatitis, and (1–2)% progress to cirrhosis or hepatocellular carcinoma

Hepatitis B shares the same symptoms as hepatitis A.
*Viral hepatitis symptoms are generally similar and are distinguished by history or laboratory investigation.

· Hepatitis B virus can enter the body and be cleared by the immune system without causing symptoms.
· It may remain dormant and be transmitted to another person without showing symptoms.
· When transmitted, it can cause acute infection and then disappear.
· It may become chronic, causing ongoing liver damage.
· In some cases, it leads directly to complications like cirrhosis or liver cancer.

In severe cases of hepatitis B, the virus rapidly destroys liver cells, causing necrosis, which can lead to coma, complete liver failure, and ultimately death.

- Fulminant hepatitis, leading to extensive liver necrosis and death, develops in less than 1%.
- Development of chronic hepatitis occurs in approximately 10% of all patients with hepatitis B infection, hepatocellular carcinoma, in up to 25% of patients.

25% from 10%
In total (2-2.5)%

# Diagnosis

Is it related to asymptomatic, chronic, or hepatocellular carcinoma? This requires further investigation.

## Serology:

- **HBsAg** - used as a general marker of infection.
- **HBsAb** - used to document recovery and/or immunity to HBV infection.
- **anti-HBc IgM** - marker of acute infection.
- **anti-HBcIgG** - past or chronic infection.
- **HBeAg** - indicates active replication of virus and therefore infectiveness.
- **Anti-Hbe** - virus no longer replicating. However, the patient can still be positive for HBsAg which is made by integrated HBV.
- **HBV-DNA** - indicates active replication of virus, more accurate than HBeAg especially in cases of escape mutants. Used mainly for monitoring response to therapy.

The presence of HBsAg indicates that the patient has been exposed to the virus and it is present in the blood.

Antibodies (IgG ,IgM )

If IgM is present, it is more likely acute; if IgG is present, it suggests chronic infection or previous exposure.

This means that the patient has formed antibodies to the virus's coating.

The best antibody marker for acute hepatitis B is IgM against the core.

Pre-core

Pre-envelope

If a person has IgM against the surface antigen, it could indicate an acute infection, but it is not as specific as IgM against the core antigen.

What is the difference between having IgM against the surface and IgM against the core?
explanation in the next slide

It helps with prognosis: if hepatitis B DNA increases, it indicates poor prognosis or ineffective treatment. If it stabilizes, replication is prevented; if it decreases, the treatment is effective.

# What is the difference between having IgM against the surface and IgM against the core?

IgM against the surface antigen may indicate an acute infection, but it's less specific than IgM against the core. The core is formed first during viral replication, followed by the pre-core and surface antigen. The body can produce antibodies against the core, pre-core, or surface.
Core antibodies (IgM) suggest acute infection and viral replication. Surface antigen or IgM against the surface suggests the body is responding to the surface antigen, but it doesn't necessarily mean active viral replication.

- The laboratory diagnosis of acute hepatitis B is best made by demonstrating the IgM antibody to hepatitis B core antigen in serum.
- Almost all patients who develop jaundice are anti-HBc IgM positive at the time of clinical presentation. HBsAg may also be detected in serum.
- Past infection with hepatitis B is best determined by detecting IgG anti-HBc, anti-HBs, or both.
- Liver Chemistry tests
  - AST, ALT, ALP, and total Bilirubin are elevated
- 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

All the previous markers indicate the presence of the virus. The only way to determine the stage of infection is through a liver biopsy.

# Treatment

- There is no specific treatment for acute hepatitis B. A high-calorie diet is desirable.
- Corticosteroid therapy has no value in uncomplicated acute viral hepatitis,
- For chronic hepatitis, interferon alpha provides long-term benefit in (~33%) of patients.
- Lamivudine (3TC), a potent inhibitor of HIV is also active versus hepatitis B virus both in vitro and in initial clinical trials, but resistance to this agent develops in about 25% of patients after 12 months of therapy.
- Adefovir, a nucleotide analog of adenosine monophosphate, is newly approved for the treatment of chronic hepatitis B.

# Prevention

- Safe practices and avoidance of needle stick injuries or injection drug use are approaches to diminishing the risk of hepatitis B infection.
- Vaccination - highly effective recombinant vaccines are now available. Vaccine can be given to those who are at increased risk of HBV infection such as health care workers. It is also given routinely to neonates in many countries.
- Hepatitis B Immunoglobulin - HBIG may be used to protect persons who are exposed to hepatitis B. It is particular efficacious within 48 hours of the incident. It may also be given to neonates who are at increased risk of contracting hepatitis
- Other measures - screening of blood donors, blood and body fluid precautions.

After 20 years, antibodies may disappear, requiring a booster dose.

# Hepatitis D – Delta Hepatitis

- Delta hepatitis is caused by the hepatitis D virus. This small single-stranded RNA virus **requires** the presence of **hepatitis B** surface antigens for its transmission and is thus found only in persons with acute or chronic hepatitis B infection.

If he's HDV+ then he must be HBV+! *Coinfection*
And when tested tested for HBsAg it should be (+) if not then there's something wrong in the diagnosis

- Delta hepatitis is most prevalent in groups at high risk of hepatitis B. Injection drug users, and as many as 50% of such individuals may have IgG antibody to the delta virus antigen. Other risks include dialysis. Non parenteral and vertical transmission can also occur

# Clinical Manifestations

Two major types of delta infection have been noted:

- Simultaneous delta and hepatitis B infection: Simultaneous infection with both delta and hepatitis B results in clinical hepatitis that is indistinguishable from acute hepatitis A or B; however, fulminant hepatitis is much more common than with hepatitis B virus alone due to the presence of two viruses..

- Delta superinfection in those with chronic hepatitis B. Persons with chronic hepatitis B who acquire infection with hepatitis D suffer relapses of jaundice and have a high likelihood of developing chronic cirrhosis and it could be sooner as well.

# Diagnosis

- Diagnosis is made most commonly by demonstrating IgM or IgG antibodies, or both, to the delta antigen in serum.
- IgM antibodies appear within 3 weeks of infection and persist for several weeks.-> acute
- IgG antibodies persist for years. -> either past infection or immune.

# Treatment and Prevention

- Response to treatment with interferon alpha in patients with delta hepatitis (and by default exists with hepatitis B ) is less than in those with hepatitis B alone. because two viruses are acting together so obviously the response will be weaker than just one
- Recommended doses are higher and may produce sustained improvement in only 15–25% of patients.
- Because the capsid of delta hepatitis is HBsAg, measures aimed at limiting the transmission of hepatitis B to prevent the transmission of delta hepatitis. since HDV depends on HBV; preventing B will also limit D
- Individuals infected with hepatitis B or D **should not** donate blood, organ, tissues, or semen.
- Methods of reducing transmission include decreased use of contaminated needles and syringes by injection drug users and use of needle safety devices by health care workers.

# Hepatitis C

- Hepatitis C virus is an RNA virus in the flavivirus. It has a very simple genome, consisting of just three structural and five nonstructural genes.
- Hepatitis C is an insidious disease in that it does not usually cause a clinically evident acute illness. Instead, its first manifestation (in 25% of those infected) may be the presence of smoldering chronic hepatitis that may ultimately lead to liver failure it's more likely to enter directly into chronic-stage hepatitis. Its transmission is less well understood than for hepatitis A, B, and D.
- Hepatitis C was the major cause of post-transfusion hepatitis until a serologic test for screening blood donors was developed. the method of transmission is unknown but it's thought to be through blood transfusion.

- The transmission of hepatitis C by blood is well documented: indeed, until screening blood for transfusions was introduced, it caused the great majority of cases of post-transfusion hepatitis although there are other ways for it to be transmitted, in Egypt for instance, individuals are HCV+ even though they haven't received any blood transfusion or come in contact with contaminated blood( FYI chatGPT says that the high prevalence of HCV in Egypt is mainly attributed to unsafe medical practices during historical public health campaigns). Dialysis is thought to be another route of transmission.
- Hepatitis C may be sexually transmitted but to a much lesser degree than hepatitis B.
- Needle sharing accounts for up to 40% of cases. In the United States, 3.5 million people (1.8%) have antibody to hepatitis C.
- Screening of donor blood for antibody has reduced post- transfusion hepatitis by 80–90%.
- Other individuals considered at risk for hepatitis C are chronic hemodialysis patients and spouses doing dialysis for HCV- patient on the same machine of HCV+ without proper control and hygiene measures leads to infection, even if the machine was properly cleaned( 15-20mins between patients), there's still a high risk of infection.

# Clinical Manifestations

- The incubation period of hepatitis C averages 6–12 weeks.
- The infection is usually asymptomatic or mild and anicteric but results in a chronic carrier state in up to 85% of adults of patients.
- The average time from infection to the development of chronic hepatitis is 10–18 yrs.
- Cirrhosis and hepatocellular carcinoma are late sequelae of chronic hepatitis.

# Diagnosis

- Antigens of hepatitis C are not detectable in blood, so diagnostic tests attempt to demonstrate antibody.
- Unfortunately, the antibody responses in acute disease remain negative for 1 to 3 weeks after clinical onset and may never become positive in up to 20% of patients with acute, resolving disease.
- Current tests measure antibodies to multiple hepatitis C antigens by either enzyme immunoassay or immunoblot
- Even with these newer assays, IgG antibody to hepatitis C may not develop for up to 4 months, making the serodiagnosis of acute hepatitis C difficult.
- Quantitative assays of hepatitis C RNA may be used for diagnosis, estimating prognosis, predicting interferon responsiveness, and monitoring therapy, but there is not a very good correlation between viral load and histology.

# Treatment and Prevention

- Combination therapy with interferon alpha and ribavirin is the current treatment of choice for patients with evidence of hepatitis due to hepatitis C.
- Corticosteroids are not beneficial.
- Avoidance of injection drug use and screening of blood products are important preventive measures.
- It is not clear whether prophylactic ISG protects against hepatitis C. In addition, it is questionable whether a vaccine will be effective; patients may be reinfected by wild-type virus.

# Hepatitis E

- Hepatitis E is the cause of another form of hepatitis that is spread by the fecal–oral route and therefore resembles hepatitis A.
- Hepatitis E virus is an RNA virus that is similar to but distinct from caliciviruses. The viral particles in stool are spherical, 27 to 34 nm in size, and unenveloped and exhibit spikes on their surface.
- Like hepatitis A, infection with this virus is frequently subclinical. When symptomatic, it causes only acute disease that may fulminate, especially in pregnant women.

- In endemic, developing areas, it has the highest attack rate in young adults, and infection is usually associated with contaminated drinking water.
- It does not appear to spread from person to person. Most cases have been identified in developing countries with poor sanitation, and recurrent epidemics have been described in these areas.
- The incubation period is approximately 40 days.

The diagnosis may be confirmed by demonstrating the presence of specific IgM antibody. No treatment is available.

Prognosis for HEV is v. good but still rare unlike A, B, and C.

# Hepatitis G

- In 1995, hepatitis G, a newly discovered agent, was identified in sera from two different patients. Hepatitis G is an RNA virus similar to hepatitis C and members of the flavivirus family.
- An antibody assay can detect past, but not present, infection, and detection of acute infection with hepatitis G requires a PCR assay for viral RNA in serum. Up to 2% of volunteer blood donors are seropositive for hepatitis G RNA, which is a blood- borne virus.

- In addition to being closely related to hepatitis C, data suggest that the majority of patients infected by hepatitis C are also infected by hepatitis G. Given this association, it has been difficult to ascertain the contribution of hepatitis G to clinical disease.
- Patients infected with both viruses do not appear to have worse disease than those infected by hepatitis C virus only.
- Currently, there is no useful serologic test and no therapy is established.

## Comparison of A, B, D (Delta), C, and E Hepatitis

Know the table!! The boxes are just to note certain differences.

| Feature | A | B | D | C[a] | E |
|---|---|---|---|---|---|
| Virus type | Single-stranded RNA | Double-stranded DNA | Single-stranded RNA | RNA | RNA |
| Percent of viral hepatitis | 50 | 41 | <1 | 5 | <1 |
| Incubation period (days) | 15–45 (mean, 25) | 7–160 (mean, 60–90) | 28–45 | 15–160 (mean, 50) | ? |
| Onset | Usually sudden | Usually slow | Variable | Insidious | ? |
| Age preference | Children, young adults | All ages | All ages | All ages | Young adult |
| Transmission | | | | | |
| Fecal–oral | +++ | ± | ± | – | +++ |
| Sexual | + | ++ | ++ | + | +? |
| Transfusion | – | ++ | +++ | +++ | – |
| Severity | Usually mild | Moderate | Often severe | Mild | Variable |
| Chronicity (%) | None | 10 | 50–70 | >50% | None |
| Carrier state | None | Yes | Yes | Yes | ? |
| Immune serum globulin protective | Yes | Yes[b] | Yes[c] | Uncertain | ? |

Shortest incubation

Because it's not clear if it's caused by it.

Could be transmitted by saliva

More than the others

Because we said it's insidious and low clinical presentation

*Abbreviation*: Plus and minus signs indicate relative frequencies.

[a] Many individuals with hepatitis C virus are also infected with the hepatitis G virus, which is similar to hepatitis C.

[b] Hyperimmune globulin more protective.

[c] Prevention of hepatitis B prevents hepatitis D.

# For any feedback, scan the code or click on it.

Corrections from previous versions:

| Versions | Slide # and Place of Error | Before Correction | After Correction |
|---|---|---|---|
| V0 → V1 | | | |
| V1 → V2 | | | |

# Additional Resources:

# رسالة من الفريق العلمي:

وَإِنْ تَصْبِرُوا وَتَتَّقُوا لا يَضُرُّكُمْ كَيْدُهُمْ شَيْئًا إِنَّ اللَّهَ بِمَا يَعْمَلُونَ مُحِيطٌ

اللهم أعني على الدراسة ولا تجعل قلبي يمل منها وكن معي في كل لحظة ووفقني لما تحب وترضى، اللهم لا تجعل الدرجات أكبر همي ومبلغ علمي ورضني بما قضيت لي... اللهم سهل علي ما صعب حفظه ويسر لي ما استغلق فهمه واجعل هذا العلم حجة لي لا علي