Microbiology
437

MED437
KING SAUD UNIVERSITY

VIRAL HEPATITIS B,C,D&G

VIRAL INFECTION OF GIT

★ What is hepatitis?

- ★ Inflammation of the liver

★ Etiology

- ★ <u>Primary infections</u> (their target is the liver)
  - ○ Hepatitis A Virus (HAV)
  - ○ Hepatitis B Virus (HBV)
  - ○ Hepatitis C Virus (HCV), was known as non-A non-B hepatitis
  - ○ Hepatitis D Virus (HDV)
  - ○ Hepatitis E Virus (HEV)
  - ○ Hepatitis F Virus (HFV)
  - ○ Hepatitis G Virus (HGV)
- ★ <u>As part of generalized infection</u>**:** (CMV, EBV, & Yellow Fever Virus)
- ★ Hepatitis F has been reported in the literature but not confirmed.
- ★ Viral hepatitis is divided into two large groups, based on the mode of transmission:
  1. Enterically transmitted hepatitis or water borne hepatitis. This group includes hepatitis A and E viruses. (they are non-capsulated and have a thick capsid that help them to live in the rough environment)
  2. Parenterally transmitted hepatitis or blood borne hepatitis. This group includes hepatitis B, C, D & G viruses.

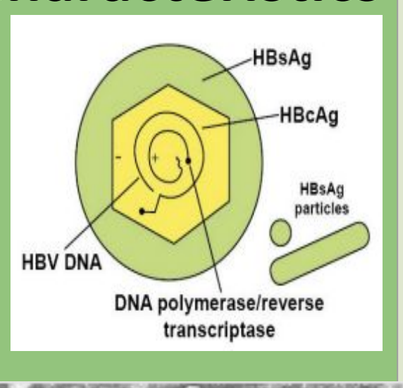
★ Groups who are at high risk to develop HBV & HCV:

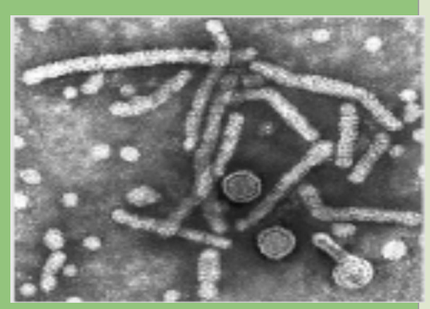
- ★ Intravenous drug users.
- ★ Hemodialysis patients.
- ★ Patients receiving clotting factors.
- ★ Individuals with multiple sexual partners and homosexuals.
- ★ Recipient of blood transfusion, before 1992
- ★ Health care workers with frequent blood contact.
- ★ Individuals who exposed to tattooing, body piercing or cupping.




Hepatitis B Virus (HBV)

Characteristics





Electron micrograph of particles in the blood of a patient infected with HBV

- Family of hepadnaviridae.
- Virion consists of
  - Outer envelope containing hepatitis B surface antigen (HBsAg).
  - Internal core (nucleocapsid) composed of hepatitis B core antigen (HBcAg).+E antigen linked to the DNA which will increase with replication
  - The viral genome which is small partially circular ds-DNA.
  - There are 8 genotypes (A-H), genotype D is dominant in Saudi Arabia.
  - The virus contains the 2 enzymes reverse transcriptase & protease enzyme
  - The serum of infected individual contains three types of hepatitis B particles:
  - Large number of small spherical free HBsAg particles.
  - Some of these HBsAg particles are linked together to form filaments.
  - The complete HBV particles (Dane particles).

Transmission	**Parentally**	**Sexually**	**From mother to baby**
	- Direct exposure to infected blood or body fluids (e.g. receiving blood from infected donor). - Using contaminated or not sterilized tools in surgical or cosmetic practice (dental, tattooing & body piercing). - Sharing contaminated needles, razors or toothbrushes.	- The virus is present in blood and body fluids. By having sexual contacts with infected person, virus is present in semen & vaginal secretion (homosexual)	- Mostly (perinatally) during delivery, nursing, breast feeding, & less likely through placenta (vertical transmission)

Acute Hepatitis B Infection

- Incubation period varies from 2 to 4 months.
- **Most** HBV infection are **asymptomatic**.
- **If symptomatic hepatitis as:**
- **An-icteric hepatitis:** (no jaundice) fever, malaise , anorexia, rash, nausea, vomiting and high upper quadrant abdominal pain with raised liver enzyme.
- **Icteric hepatitis:** (with jaundice) about 25% of (the patient become icteric Jaundice with raised bilirubin, dark bile containing urine and pale stools)

Clinical Outcomes

- About 90 % of infected adults will develop acute hepatitis B infection and recover completely
- <9 % of the infected adult, 90% of infected infants and 20% of infected children may progress to chronic hepatitis B.
- <1 % may develop fulminant hepatitis B, characterized by massive liver necrosis, liver failure and death.

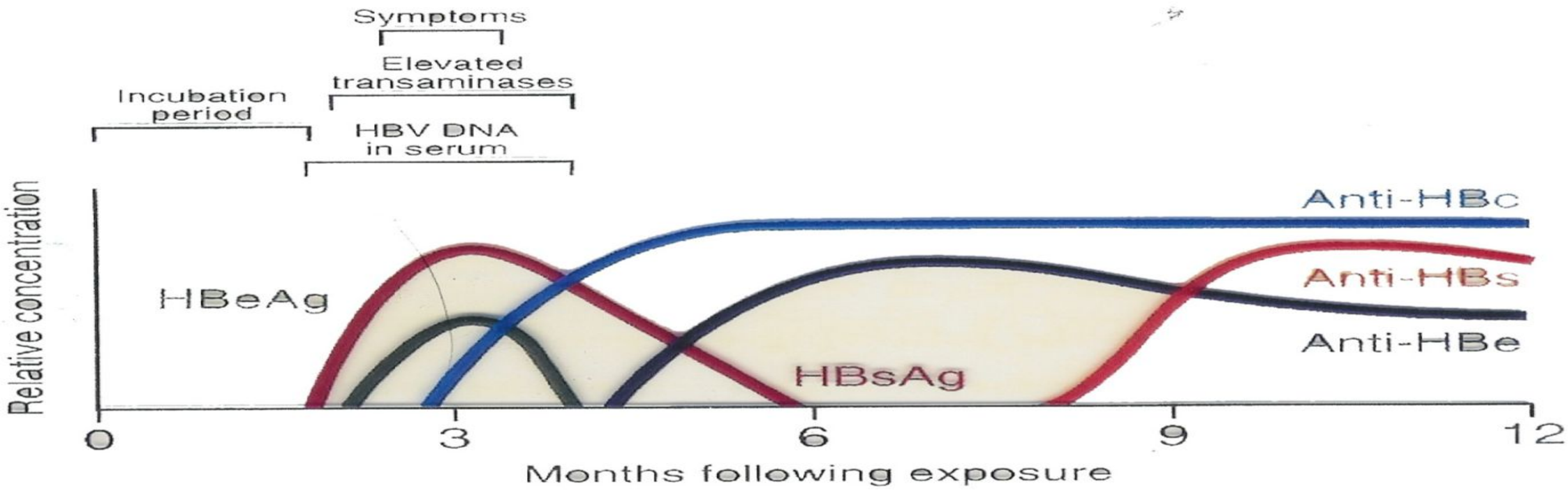
HBV Markers (Important)	
HBV DNA	Marker of infection. Contiguous
Hepatitis B surface antigen (HBsAg)	Marker of infection. Contagious
Hepatitis B e antigen (HBeAg)	Marker of active virus replication, the patient is highly infectious, the virus is present in all body fluids. Highly contiguous
Antibody to hepatitis B e antigen (Anti-HBe)	Marker of low infectivity, the patient is less infectious. Contiguous
Antibody to hepatitis B core (Anti-HBc)	Marker of exposure to hepatitis B Infection. Contiguous
Antibody to hepatitis B surface antigen (Anti-HBs)	Marker of immunity. Not contiguous

Serological Profile of Acute HBV Infection	
HBV DNA	Is the first marker that appears in circulation, 3-4 weeks after infection.
HBsAg	Is the second marker that appears in the blood and persists for less that 6 months then disappears.
HBeAg	Is the third marker that appears in circulation and disappears before HBsAg, it indicates active viral replication.
Anti-HBe	with the disappearance of HBeAg and appearance of Anti-HBe Ab which usually persists for several weeks to several months.
Anti-HBc	Is the first antibody that appears in blood and usually persists for several years. (Marker of exposure)
Anti-HBs	Is the last marker that appears in the blood, it appears few weeks after disappearance of HBsAg and persists for several years. It is the marker of immunity to hepatitis B infection.



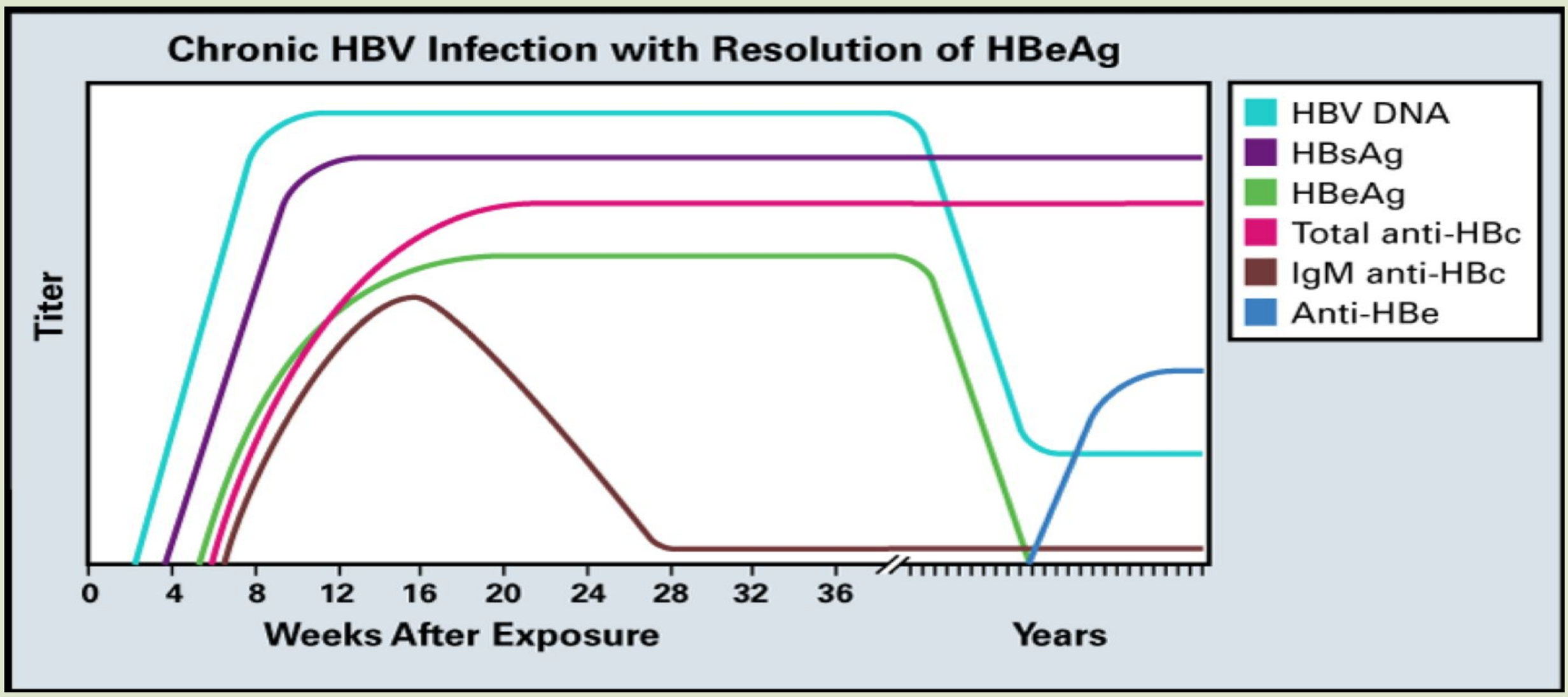

- Notice here the Anti-HBcAg IgM is found before any antibodies either Anti-HBsAg or Anti-HBeAg
- This antibodies (Anti-HBcAg) indicate viral infection in the past (marker of exposure)
- Results in 1-immune patient = having both (Anti-HBsAg + Anti-HBcAg)
- 2-Vaccinated patients = having only Anti-HBsAg. And no viral exposure so -ve Anti-HBcAg

a) **Serological profile of acute, resolving hepatitis B**



- There is a window period of the infection, where the only detectable antibody will be Anti-HBc IgM
- HBsAg & HBV DNA have to disappear before 6 months of the infection bc it it was still there after 6 months this means that the infection become chronic.

Ex: a patient came to you during the 6th month of the infection to donate their blood. You must run some tests first right? What will make you confirm that this person is infected? Anti-HBc IgM



You can clearly see the different bw the chronic and acute infection where all the antigens are still present even after 6 months

Chronic Asymptomatic Hepatitis B Infection

Defined by:	The presence of HBsAg or HBV-DNA in the blood for > 6 months.
The majority of patients	Asymptomatic may only be detected by elevated liver enzyme (ALT & AST) on a routine blood chemistry profile.
Some patients	Have mild fatigue, RT upper quadrant abdominal pain or enlarged liver & spleen.
Serological profile	1. HBsAg may persist in the blood for life. OR 2. Some patients will become immune after years and the HBsAg disappeared and anti-HBsAb detected in the serum and persists for life.

Three Phases of Chronic Hepatitis Infection

Replicative Phase	Inflammatory Phase	Inactive Phase
The patient is +ve for HBsAg, HBeAg, & HBV-DNA High viral load >10^5 copies/ml, ALT is normal or nearly normal. Liver biopsy shows minimal damage	HBsAg is +ve for > 6 months, HBeAg +ve Decline in HBV-DNA in the blood but VL is >10^5 copies/ml, ALT is elevated. The immune system attacks before hepatocytes harboring the virus Liver biopsy shows damage to hepatocytes.	-ve for HBeAg, +ve for anti-HBe, HBV-DNA VL <10^5copies/ml & normal ALT

Chronic active hepatitis (the doctor skipped all of this)

The major long term risk of chronic HBV infection are cirrhosis with hepatic failure & hepatocellular carcinoma.

1. Cirrhosis

Characterized by	Fibrosis and nodular formation.
Results from	Liver cell necrosis and the collapse of hepatic lobules.
Symptoms	Ascites, coagulopathy (bleeding disorder), portal hypertension,hepatic encephalopathy, vomiting blood,weakness, weight loss.

2. Hepatocellular carcinoma (HCC):

One of the most common cancer in the world.Also, one of the most deadly cancer if not treated.

Cause of chronic liver diseases	Hepatitis B and C viruses
Symptoms	Abdominal pain,abdominal swelling, weight loss, anorexia,vomiting, jaundice.
Physical examination	hepatomegaly,splenomegaly and ascites.
Diagnosis	Alpha-fetoprotein measurement with multiple CT abdominal scan are the most sensitive method for diagnosis of HCC.
Treatment	Surgical resection and liver transplant.
Prognosis	Without liver transplantation, the prognosis is poor and one year survival is rare.

Lab Diagnosis of Hepatitis B Infection

- ★ Hepatitis B infection is diagnosed by detection of HBsAg in the blood
- ★ Positive results must be repeated in duplicate.
- ★ Repeatedly reactive results must be confirmed by neutralization test.
- ★ Why confirmation? It will affect his life, career e.g.(we dont bring workers with HBV)
- ★ Detection of HB-DNA by PCR
- ★ Additional lab investigations:
  1. Liver function tests (LFT).
  2. Ultrasound of the liver.
  3. Liver biopsy to determine the severity of the diseases.

Vaccine

- ★ Contains highly purified preparation of HBsAg particles, produced by genetic engineering in yeast. (It's neither live or killed vaccine it's engineered vaccine)
- ★ It is recombinant and subunit vaccine.
- ★ The vaccine is safe and protective
- ★ Dose: administered in three doses at 0,1, & 6 months.

Prevention & Control

Pre-exposure prophylaxis	Post exposure prophylaxis
Active vaccination given to all newborn, children or adult. Recombinant hepatitis B vaccine: It is prepared by cloning HBsAg in yeast cells. Given in 3 IM injection at 0-1-6 months and booster dose after 5 years. If he receives less than 3 its not affective	Persons exposed to needle prick or infant born to +ve HBsAg mother should immediately receive both: Active vaccine and hepatitis B specific immunoglobulin.

Treatment you don't have to know the dose or period just know the treatment

Treatment is limited to patients having chronic hepatitis B based on liver biopsy.

There are several approved antiviral drugs we pick one (we don't combine):

- ★ Pegylated alpha interferon → one injection per week, for 6- 12 months.
- ★ Lamivudine → nucleoside analogue, one tablet a day for at least one year.
- ★ Adefovir → nucleoside analogue, one tablet a day for at least one year.

Criteria for treatment:

- ★ Positive for HBsAg
- ★ Positive for HBV-DNA >20,000 IU/ml.
- ★ ALT > twice the upper normal limit .
- ★ Moderate liver damage.
- ★ Age > 18 years.




Hepatitis D Virus (delta virus)

- It is a defective virus, that cannot replicates by its own.
- It requires a helper virus, which is HBV → HBV provides the free HBsAg particles to be used as an envelope.
- HDV is small 30-40 nm in diameter, composed of small ss-RNA genome, surrounded by delta antigen that form the nucleocapsid.

Types of HDV Infections

Co-infection	Super infection when you have the infection and get an infection as a complication
• The patient is infected with HBV and HDV at the same time leading to severe acute hepatitis. • Prognosis: recovery is usual.	• In this case, delta virus infects those who are already have chronic hepatitis B leading to severe chronic hepatitis.

Hepatitis C Virus

Family	Flaviviridae
Genus	hepacivirus
Size	The virus is small, 60 – 80 nm in diameter.
Structure	Consists of: • An outer envelope, • Icosahedral core • Linear positive polarity ss-RNA genome • He don't have E antigens / S antigens and there antibodies like HBV
Genotypes	• There are 6 major genotypes (1 – 6) • Genotype 4 is the dominant in Saudi patients

Transmission of HCV

Parenterally
• Direct exposure to infected blood. • Using contaminated needles, surgical instruments. • Using contaminate instruments in the practice of tattooing, ear piercing & cupping. • Sharing contaminated razors 7 toothbrushes.
Sexually
From mother to child perinatally




Hepatitis C virus Cont.

Clinical Picture of HCV infection

- ★ Incubation period from 2 to 7 weeks.
- ★ Clinically ,the acute infection with HCV is milder than infection with HBV.
- ★ Fever ,anorexia, nausea, vomiting , and jaundice are common.
- ★ Dark urine ,pale feces , and elevated liver enzyme (transaminase) are seen.

Clinical Outcomes of HCV infection

About 20 % of the infected individuals will develop self-limiting acute hepatitis C and recover completely.	**About 80%** of the infected will progress to chronic hepatitis C. About **10%-30%** of them can develop cirrhosis within 30 years and liver cancer.	**Less than 1 %** will develop acute fulminant hepatitis C, liver failure and death.

Hepatitis C Markers

Hepatitis C virus RNA	Is the 1st marker that appears in circulation, it appears as early as 2-3 weeks after exposure. It is a marker of infection. (if it still present after 6 months then its chronic)
Hepatitis C Core Antigen	The 2nd marker that appears in blood, usually 3-4 weeks after exposure. It is a marker of infection
igG antibody to hepatitis C	- Antibodies to hepatitis C virus is the second marker that appears in the blood - It usually appear 50 days after exposure (long window period) - It is not marker of immunity - Can be detected in completely recovered patient and chronic and acute.

Acute hepatitis	Chronic hepatitis
Symptoms: ● jaundice, fatigue & nausea. ● Elevated serum ALT (usually greater than 10 folds). ● Presence of anti-HCV (-ve in 30-40%) in early stages of disease. (long window period) ● HCV-RNA is +ve even before the onset of symptoms.	Defined as: the presence of anti-HCV & elevated serum level of ALT for > 6 ms. ● Almost all patients with chronic hepatitis C have the genome HC RNA in serum. ● Usually asymptomatic, but if symptom present it's usually mild, non-specific & intermittent. ● Lab finding: Elevated ALT & AST ranging from 3-20 times (ALT >AST).




Lab diagnosis of Hepatitis C

By detection of both:

1. Antibody to HCV in the blood by ELISA, if +ve the result must be confirmed by RIBA/PCR.
    a. RIBA is the confirmatory test
2. HCV-RNA in the blood using PCR.

Molecular (doctor said this is for your own information)

Qualitative Assay	Quantitative Assay
- It's the most specific test for indicating ongoing infection & almost +ve in chronic hepatitis. - It's usually –ve after recovery & effective treatment. - It's the best method for diagnosis of early stages of acute hepatitis & HCV in immunocompromised patient. - PCR & TMA(transcription mediated amplification) can detect low level of virus (50-100 copies/ml # 25-50 IU).	- It gives indirect assessment of viral load by measuring the concentration or level of virus in serum. - Methods includes quantitative PCR & branched DNA test. - Most patient have viral load between 100.000-10.000.000 copies/ml # 50.000- 500.000 IU. - It correlate with the likelihood response to therapy. - Response is higher with low level viraemia <2 million copies/ml # one million IU.

Treatment of Hepatitis C Infection & Vaccine

The currently used treatment is the combined therapy using **both**:

1. Pegylated alpha interferon
2. Ribavirin.

There is no vaccine available to hepatitis C.

<u>New Drugs:</u> There are number of approved therapies as Sovaldi may be given together with or without Ribavirin & Peginterferon. When hepatitis C treatment is working , the virus will become undetectable within 4 to 12 weeks and will remain that way throughout treatment .patients consider cured when virus remain undetectable for 12 to 24 weeks after completing therapy.

Criteria for treatment:

- Positive for HCV-RNA & anti-HCV.
- Known HCV genotype.
- ALT > twice the upper normal limit.
- Moderate liver damage based on liver biopsy.

Hepatitis G Virus: (doctor didn't read it)

Hepatitis G virus or GB-virus was discovered in 1995.

- Share about 80% sequence homology with HCV.
- Family: Flaviviridae, genus: Hepacivirus.
- Enveloped, ss-RNA with positive polarity.
- Parenterally, sexual and from mother to child transmission have been reported.
- Causes mild acute and chronic hepatitis infection.
- Usually occurs as co-infection with HCV, HBV and HIV




Doctor's Notes

(doctor mona skipped all the treatment but just read them in case just the questions came from the males' doctor)

❖ **Hepatitis B Virus features**

- The only hepatitis virus that has DNA
- It has an outer envelope composed of lipoprotein for protection (HBsAg)
- Internal core protecting the genome inside

- نتخيلها كأنه الڤايرس لابس جاكت عشان يحميه اللي هو الاوتر انڤلوب والجاكت هذا هو اول شيء بنشوفه لأنه برا صح؟ والجاكت هذا مكون من ايش؟ سرفس انتيجن واللي هو اول ماركر بيطلع لنا
- طيب تحت الجاكت هو لابس بلوزه اللي هي الانترنال كور المكونة من الكور انتيجن عشان تحمي الستركتشرز اللي موجوده جوا (and those are the viral genome and enzymes.)

❖ **Transmission**:

- HBV can survive on environmental surfaces.
  - ➔ If for example you touched a contaminated surface and then transferred the infectious material to your eyes, mouth, nose or broken skin.
- Why homosexual are more likely to get the infection? Bc the rectum area is highly vascular and it might get injured which will transfer the virus present in the body fluid of the infected person to the other uninfected one.

❖ **Markers found in serum.**

- 1st marker HBV DNA → marker of infection & can infect other ppl
- 2nd marker HBsAg → marker of infection & contagious
- 3rd marker HBeAg → part of the core, and it presence in serum means that the virus is in highly multiplication pattern thus it means it highly infectious (highly contagious)
- Anti-HBe → this is a good marker bc it tell us that the activity of the virus decreased
- Anti-HBc → tell us that this person got infected before
- Anti-HBs → the only marker of immunity
  - If a person got infected and then recovered & developed immunity from the infection they will have Anti-HBc & Anti-HBs

❖ **Hepatitis D Virus:**

➢ You can't get infected with it unless you're already infected with HBV or by direct exposure of HBV and HDV at the same time

➢ Why? Bc its a weak virus that doesn't have envelope and needs HBV surface Ag

Confirmation test for HBV? Neutralization
Confirmation test for HCV? RIBA/PCR




Doctor's Notes

★ Dr.Alhetheel **Review**:

❖ know the Hb-B markers

- Summarized in order: DNA SpECiES

➔ first the antigens: DNA.Surface,E

➔ Then antibodies against: E,C,Surface

❖ know the structure and antigens.

➔ Outer envelope containing hepatitis B surface antigen (HBsAg).

➔ Internal core (nucleocapsid) composed of hepatitis B core antigen (HBcAg).

➔ circular dsDNA. Linked to E antigen

➔ The virus contains the enzyme reverse transcriptase.

❖ what are the clinical outcomes for hepatitis B (percentages)?

➔ Adult: 90% of infections will have an acute HB (self-limiting)

➔ 9% adult will have chronic infections

➔ Children : 20% will have Chronic

➔ Infants: 90% will have chronic

❖ Know Hepatitis treatment

➔ HBV:alpha-Interferon,Lamivudine, adefovir(not combined)

➔ HBC: combined alpha-Interferon , ribavirin

- For screening do ELISA for all the hepatitis viruses
- confirmation :HBV⟶ neutralization, for HCV⟶ PCR or Riba




Summary

Blood born hepatitis

1- Hepatitis B virus

Family	hepadnaviridae	**viral genome**	ds-DNA.	**Markers**	HBV DNA	Marker of infection contagious
virus enzymes	1- reverse transcriptase 2-protease	**Genotype**	(A-H) D is dominant in KSA.		HB surface antigen (HBsAg)	
					HB e antigen (HBeAg)	Marker of active virus replication, the patient is highly infectious
					Antibody to HB e antigen (Anti-HBe)	low infectivity, the patient is less infectious, contiguous.
Characters	1- Outer envelope containing hepatitis B surface antigen (HBsAg) 2-Internal core composed of hepatitis B core antigen(HBcAg)	**Transmission** Similar to HCV	1-Parentally 2- Sexually (unprotected sex) 3- From mother to the fetus: (perinatally)		Antibody to HB core (Anti-HBc)	Marker of exposure to HB infection, contiguous.
					Antibody to HB surface antigen (Anti-HBs)	Marker of immunity not contiguous.
Either	Acute hepatitis B infection			or	Chronic hepatitis B infection	
Clinically	asymptomatic	If symptomatic		**Clinically**	Many are asymptomatic	
	Many are asymptomatic	An-icteric hepatitis	Icteric hepatitis	**symptom**	right upper quadrant pain, enlarged liver & spleen. ±Jaundice, fatigue. elevated liver enzyme	
The clinical outcome	90 % of infected adults will recover completely. < 9 % of the infected adult and 90% of infected infants and 20% of infected children may progress to chronic HB. < 1 % may develop fulminant hepatitis B			**Complication**	Chronic active hepatitis: Lead to cirrhosis with hepatic failure and hepatocellular carcinoma	
Serological profile of acute HB	1-HB DNA: 3-4 weeks after infection 2-HBsAg: persists for < 6m 3- HBeAg: disappears before HBsAg 4- Anti-HBc Ab: persists for several years.	5- anti-HBe Ab: persists for several weeks or months. 6- Anti-HBsAg: persists for several years		**Serological profile of chronic HBV**	the presence of HBsAg or HBV-DNA in the blood for more than 6 months	
Diagnosis	1-Detection of HBsAg in the blood. 2- if Positive results must be repeated in duplicate 3-confirmed by neutralization test. PCR for HB-DNA Additional lab investigations: Liver function tests-US or biopsy of liver			**Prevention**	**Pre-exposure prophylaxis:** prepared by cloning HBsAg in yeast cells IM injection at 0-1-6 months and booster dose after 5 years.	
IMPORTANT	• Anti –HBcAg **IgM** is found before any antibodies • Anti-HBc Ag indicate viral infection in the past. • Immune patient : Anti –HBsAg + Anti-HBc Ag • Vaccinated patients: only Anti-HBsAg.				**Post exposure prophylaxis:** immediately receive both Active vaccine and hepatitis B specific immunoglobulin.	




Summary

<table>
<tr><th colspan="6">2- Hepatitis D virus</th></tr>
<tr><td>Characteristics</td><td colspan="2">It is a defective virus, that cannot replicates by its own.
It requires a helper virus which is HBV.
HBV provides the free HBsAg particles to be used as an envelope</td><td>Types of infections</td><td colspan="2">1- Co-infection: patient is infected with HBV and HDV at the same time.
2- Super infection: HBV infects those who are already have chronic hepatitis B.</td></tr>
<tr><th colspan="6">3-Hepatitis C virus</th></tr>
<tr><td>Family</td><td>Flaviviridae</td><td>viral genome</td><td>linear + polarity ss-RNA gemone</td><td>Genotypes</td><td>(1 – 6), genotype 4 is the dominant in KSA</td></tr>
<tr><td>The clinical outcome</td><td colspan="5">20 % of the infected individuals will recover completely.
80 % of the infected will progress to chronic hepatitis C. about 10%-30% of them can develop cirrhosis within 30 years and liver cancer
Less than 1 % will develop acute fulminant hepatitis C</td></tr>
<tr><td rowspan="2">Markers</td><td>HC virus RNA.</td><td colspan="4">1st marker that appears, it appears 2-3 weeks after exposure. It is a marker of infection</td></tr>
<tr><td>HC Ab antibody to hepatitis C.</td><td colspan="4">second marker usually appear 50 days after exposure (long window period), it is not marker of immunity, can be detected in completely recovered patient and chronic and acute.</td></tr>
<tr><td></td><td colspan="3">Acute hepatitis</td><td colspan="2">Chronic hepatitis</td></tr>
<tr><td></td><td colspan="3">Presence of anti-HCV negative in early stages of disease. HCV-RNA is +ve even before the onset of symptoms.</td><td colspan="2">Defined as the presence of anti-HCV & elevated serum level of ALT for >6 ms.
have the genome HC RNA in serum.</td></tr>
<tr><td>Diagnosis</td><td colspan="5">1- Antibody to HCV in the blood by ELISA, if positive the result must be confirmed by RIBAor PCR.
2- HCV-RNA in the blood using PCR.</td></tr>
<tr><td>Treatment</td><td colspan="5">Pegylated alpha interferon and ribavirin - sovaldi</td></tr>
</table>




MCQs:

1- What is the marker for immunity in HBV ?
A- HBsAg.
B- Anti-HBe.
C- HBeAg.
D- Anti-HBsAg.

2- What is the confirmation test for HCV?
A- Neutralization.
B- RIBA.
C- ELISA.
D- IF.

3-Patient suffering from Chronic active hepatitis. What do you expect to in serum?
A- HBsAg only.
B- HB-DNA only.
C- HBsAg and HBeAg.
D-Anti-HBs Ab.

4-Which type of hepatitis could lead to development of hepatocellular carcinoma (HCC)?
A- Hepatitis D virus.
B- Hepatitis C virus.
C- Hepatitis G virus.
D- Hepatitis B virus.

5-Which of the following drug is used to treat a patient with chronic hepatitis C virus?
A- Lamivudine.
B- Pegylated alpha interferon & ribavirin.
C- ribavirin and Lamivudine .
D- SOVALDI and Lamivudine.

6- What is the first antibody to appear in a patient after he got infected with HBV ?
A- Anti-HC Ab.
B- Anti-HBs Ab.
C- Anti-HBe Ab.
D- Anti-HBc Ab.

SAQ:

1-D 4-D
2-B 5-B
3-C 6-D

- While performing a surgery, the surgeon cut himself by accident. He knew that the patient have chronic hepatitis.

1- What is the best way to treat the surgeon?

If he did not receive any vaccine for hepatitis B we should immediately give him both Active vaccine and hepatitis B specific immunoglobulin.

2- How did the surgeon know that the patient have chronic hepatitis ?

The patient must have HBsAg and HB-DNA in the serum for more than 6 months.

2- What type of test has been done to the patient to diagnosis him/her?

- First Detection of HBsAg in the blood - Then if Positive results must be repeated in duplicate - finally confirmed by neutralization test.
- PCR for HB-DNA
- Additional lab investigations:

Liver function tests- Ultrasound of liver - biopsy of liver to determine the severity of the diseases

3- When you performed a blood test for the surgeon and found out that he have been infected by HBV. What indicate that ?

Anti –HBsAg + Anti-HBc Ag in the serum




THANK YOU!

TEAM LEADERS:

ALANOUD AL-MANSOUR & KHALED AL-OQEELY

TEAM MEMBERS:

GHADA AL-QARNI

MUNIRA AL-HADLG

NOUF AL-OTAIBI

RAHAF AL-THNAYAN

REEM AL-QAHTANI

SARA AL-SULTAN

Designed by:
Aseel Badukhon (: