Hepatitis Viruses

- Hepatitis: inflammation of liver; presence of inflammatory cells in liver organ tissue
- The causes of hepatitis are varied and include infectious agents, such as viruses, bacteria, and protozoa, as well as non-infectious agents, such as drugs and toxins. (we will focus on the virus causes)
- Among infectious liver diseases, viruses are the leading (major) cause.
- Acute hepatitis: symptoms last less than 6 months.
- Viral Hepatitis: is inflammation of the liver induced by viral infections.
- Hepatitis viruses are well characterized viruses, exclusively causing sporadic cases of hepatitis. While other viruses (e.g. Cytomegalovirus (CMV), Ebstein Barr virus (EBV), Yellow fever virus, Herpes simplex virus (HSV), Rubella virus and other enteroviruses) can still cause hepatitis, but not as exclusive, because they can cause infections in other sites in the body.

acute viral hepatitis caused by hepatitis viruses can be similar, they can't be distinguished clinically, and determination of a specific cause depends on laboratory tests, Liver enzymes test (serum enzymes levels) increase:

• ASpartate aminoTransferase (AST). • ALanine aminoTransferase (ALT).

General rule: serum ALT/AST ratio more than 1 ((ALT/AST) >1) suggests viral hepatitis, reflecting cytoplasmic liver cell damage.

- Although the target organ (liver) and basic symptoms of hepatitis viruses are similar, they differ in their structure, mode of replication, mode of transmission, time course, and sequelae of the disease they cause

Most cases of acute viral hepatitis in both children and adults are caused by one of the following agents:

- A: Picornavirus: +ssRNA, Non enveloped.

  HAV, causes hepatitis A, previously named infectious hepatitis.

- B: Hepadnavirus Ds DNA, enveloped.

  HBV, causes hepatitis B, known as serum hepatitis.

- C: Flavivirus, +ssRNA genome, enveloped.

  HCV, common cause of post-transfusion hepatitis.

- D: Deltaviruses, a Defective virus –ssRNA virus.

  HDV, always dependent on the co-infection with hepatitis B virus, as it's defective.

- E: Hepevirus, +ssRNA non enveloped.

  HEV is an agent of enterically transmitted hepatitis, similar to the hepatitis A virus.

Regardless of the virus type, identical histopathological lesions are observed in the liver during the acute disease. These viruses are not cytopathic; rather, it is mainly an immunological response that leads to those lesions.

Hepatitis A

• A typical Enterovirus, and a distinct member of the Picornaviridae, also known as entervirus 72. , non enveloped ,RNA virus , One serotype, while there're seven genotypes of HAV. (Viridae = family)

1. After ingestion and reaching the liver by hematogenous spread, HAV replicates in liver.
2. Then it's excreted in the bile.
3. After that it gets excreted in feces of those infected persons for about two weeks before the onset of clinical illness and up to one week post symptoms in those affected patients.

• Asymptomatic persons still transmit the virus to others by typical feco-oral transmission.

• Viral particles can be detected in feces using electron microscopy.

- •Hepatitis A is the most common cause of viral hepatitis, more than 40–50%. (most often associated with outbreaks or epidemics.)

Humans appear to be the major natural hosts of HAV.

- Enterically transmitted (fecal/oral route):
- There are always outbreaks associated with contaminated food and water.
- HAV can never cause chronic hepatitis; initially IgM antibody response then followed by IgG antibody response, which usually give a lifelong immunity post-infection with HAV. Also, there're no carriers. (This also applies to HEV).

  • Commonly seen in children mainly and in young adults, 90% of infected children and up to (25-50%) of infected adults have inapparent but productive infection with HAV.
- Spread of HAV infection: mainly by fecal-oral route and arise from the ingestion of contaminated food and water, so overcrowding and poor sanitation facilitate the spread.
- HAV causes infectious hepatitis, which is an acute disease that is clinically milder or asymptomatic in young children.
- More than 90% of adults in many developing countries show evidence of past hepatitis A infection. Travelers from developed countries are particularly susceptible when visiting these endemic areas.
- Patients are most contagious during the two weeks prior to the onset of clinical symptoms, which are primarily characterized by jaundice.

- Infectious dose: less than 100 viral particles, which are sufficient to establish infection.
- Jaundice is observed in almost 70–80% of adults with hepatitis A, but in only about 10% of children (particularly those under 6 years of age).
- Incubation period: 2-6 WEEKS.
- The most common presentation of HAV infection is asymptomatic, especially in children. In contrast, adults are more likely to develop symptomatic disease, which typically progresses through two distinct phases:

1.Pre-icteric (prodromal) phase:

- Characterized by the abrupt onset of a flu-like illness accompanied by nausea, vomiting, and anorexia.

2. Icteric phase:

- Characterized by jaundice, abdominal pain, hepatosplenomegaly, pale stools, and dark-colored urine, which is often noticed 1–5 days before the appearance of clinical jaundice
- A sudden onset of fever, anorexia, and pain, particularly in the right upper quadrant of the abdomen, may occur within a short timeframe and represents a classic presentation of symptomatic hepatitis A infection.
- On physical examination, the liver is typically enlarged and tender.

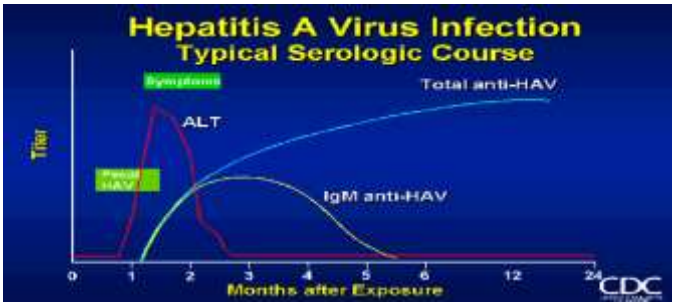
recovery occur after days or weeks.

- Many individuals with hepatitis A are asymptomatic or experience only mild illness without jaundice.
- "Resolve spontaneously in 2-4 weeks"
- Approximately 99% of HAV cases are self-limiting, with no progression to chronic hepatitis or carrier state. However, in fewer than 1% of cases, patients may develop fulminant hepatitis, these cases carry a mortality rate of 0.1–0.2%.

Comparing HBV and HAV:

HAV： No chronic cases or carriers Not associated with hepatic cancer (hepatocellular carcinoma).

HBV： Can be chronic Associated with hepatic cancer (hepatocellular carcinoma).

- Clinically: diagnosis begins with a thorough history and physical examination.
- Liver biochemistry typically reveals elevated liver enzymes, especially AST and ALT. These levels usually rise before jaundice appears in the icteric stage. Serum bilirubin may also be elevated, indicating hepatic inflammation or injury.
- Hematological tests in affected patients: often reveal leukopenia with relative lymphocytosis.
  Additionally, the erythrocyte sedimentation rate (ESR) may be elevated
- Serology or viral markers: IgM, IgG (lifelong immunity)
  ➢IgM: indicates Acute infection and remains high for 3-6 months, which is followed by the production of IgG.
  ➢IgG: indicates Past infection or vaccine.
  ▪ Anti-HAV IgG becomes predominant later in life and is commonly found in the general population over the age of 50.
- Again, using immune electron microscopy, HAV particles can be identified in fecal specimens.
- However, isolation of the virus in cell culture is used for research purposes (research tool) and is not routine in clinical practice.
- 

- This graph shows the typical serological course in patients infected HAV.

- As previously mentioned, the virus can be initially detected in stool samples, and liver function tests, particularly ALT and AST are elevated early in the course.
- Anti-HAV IgM antibodies begin to rise about one month post-exposure, peak around the third month, and are eventually replaced by anti-HAV IgG antibodies, which indicate recovery and longterm immunity.
- Both previous infection and vaccination confer lifelong protection against HAV

. Summarizing, first elevated are the liver enzymes along fecally excreted HAV (even before symptoms), followed by IgM Anti-HAV which is later replaced by IgG, resulting in long lived immunity.

Treatment of Hepatitis A:

➢Acute:

- There is no specific antiviral treatment for acute hepatitis .
- Mainly Supportive: includes adequate nutrition, hydration, and bed rest. Do not give Paracetamol and Alcohol.
- In regard to immunization, two important terms are discussed:

1. Passive immunization involves the administration of immune serum globulin, which is most effective when given before or within 2 weeks of exposure to HAV. It provides short-term protection and is approximately 80–90% effective in preventing clinical illness.
2. Active immunization is achieved with the hepatitis A vaccine , which contains a formalin inactivated/killed virus. It induces the production of anti-HAV antibodies like those produced after natural infection and offers nearly 100% protection with longterm immunity.

Prevention :

- Preventive measures, such as avoiding contaminated food and water, are essential to reduce the risk of HAV infection.
- Therefore, as a preventive measure, the spread of hepatitis A is reduced by interrupting the fecal oral transmission route.
- This is typically achieved through:

1. The avoidance of potentially contaminated food and water, especially undercooked shellfish, and through proper hand hygiene, particularly in high-risk settings such as childcare centers and mental health institutions.
2. Chlorination of drinking water is also highly effective, as it is generally sufficient to inactivate the HAV.

Hepatitis E

- Hepatitis E virus is RNA virus, similar but distinct from calicivirus.
- The viral particles in stool are spherical, 30nm in size, and unenveloped and exhibit spikes on their surface.
- Similar to HAV in transmission: Feco-oral transmission.

❖ Clinical course:

- Like HAV, HEV infection is often subclinical in children.
  - However, when symptomatic, HEV typically causes only acute disease(no developed to chronic) , which may rarely progress to fulminant hepatitis.
- Clinically, acute hepatitis E resembles hepatitis A, but a key difference is that HEV infection often presents with higher bilirubin levels and more intense, prolonged jaundice.
- In endemic and developing regions, hepatitis E has the highest attack rate among young adults.
- Most cases occur in areas with poor sanitation, where recurrent epidemics have been reported.

❖ Transmission:

- The infection is typically associated with the consumption of contaminated food and water.

- Does not appear to spread from person to person.

❖ Incubation period: it's approximately 40 days, but usually 2-8 weeks.

❖ Diagnosis: confirmed by* detecting specific anti-HEV IgM antibodies in the patient's serum. Also, by* excluding other types, and *molecular real-time PCR.

❖ High risk group: include pregnant women and malnourished individuals, these patients are most likely to develop severe hepatitis E, e.g. fulminating hepatitis.

❖Treatment: no specific treatment, but supportive treatment as in HAV is used

❖Case fatality rate: normally it's 1-2% but significantly increased to 10-20% in high-risk group.

Hepatitis B virus

- The smallest DNA virus known, with 3200 nucleotides.
- Hepadnavirus, Partially Double stranded circular DNA with a short and a single stranded piece genome ,Enveloped ,Icosahedral nucleocapsid.
- Antigens:
- The main components of the virus include the (1) core - hepatitis B core antigen (HBcAg) and the (2) pre core hepatitis B e antigen (HBeAg), and the (3) envelope of the virus contains the hepatitis B surface antigen (HBsAg).
- The pre-core hepatitis B e antigen (HBeAg) indicates active replication and infectivity.
- For replication, HBV has its own DNA-dependent DNA polymerase. And it has only single serotype.

❖ Hepatitis B infection:

**asymptomatic or presents as a mild, self limited illness in approximately 90% of cases, typically with jaundice and fever lasting a few days to weeks.

** However, up to 10% of patients may develop chronic infection, which can lead to liver cirrhosis or hepatocellular carcinoma. (unlike HAV infection)

- ❖ HBV particle forms: virions, spheres, and filaments
- ❖ HBV serotypes: serotypes are classified based on a common group-specific antigen (A) and two pairs of mutually exclusive epitopes (either D or Y, and W or R) which are determined by the serological properties of the hepatitis B surface antigen (HBsAg) This classification results in four main serotypes. • The four serotypes are: adr, adw, ayr, ayw
- ❖ Treatment: there's an HBV vaccine.

- HBV has a global distribution, with an estimated 2 billion people showing serological markers of past or present HBV infection.
- Among them, around 400 million have chronic HBV infections, thus carriers.
- The global incidence of death due to HBV-related complications is approximately 1 million people per year.
- Infection of hepatitis B is called serum hepatitis So…

❖Transmission:

1. through intravenous routes such as transfusions of infected blood or the use of contaminated needles, including among people who inject drugs, tattooing and acupuncture. (Needle stick injury, pose a high risk of transmission especially among medical personnel)
2. Additionally, transmission can occur through close personal contact, including sexual intercourse, particularly among men who have sex with men (MSM).
3. Vertically: mother to baby, during or soon after birth.

The virus can be found in semen and saliva.

- Pathogenesis:

Piecemeal necrosis (interface hepatitis):

- Common form of necrosis, with a characteristic histological finding in hepatitis Involves an inflammation that extends from the portal tract into the surrounding periportal zone (around portal vein), leading to necrosis of the periportal hepatocytes and destruction of limiting plate. ("hepatocytes injury and necrosis ")

- Clinically:
  - Incubation period:7 to 160 days with an AVG of 70 days (10 weeks)
  - Based on the immune response, patients can be: ✓Asymptomatic OR ✓Symptomatic ✓Also, acute and chronic.

Asymptomatic: 90% of children and 50% of adults (increased liver enzymes)

✓Symptomatic:

- Pre-icteric phase: flu like symptoms nausea, anorexia, malaise
- Icteric phase: Jaundice, pale stool, dark- coloured urine, increased liver enzymes and bilirubin.

- Acute hepatitis B infection often begins gradually , with symptoms such as fatigue, reduced appetite, nausea, and discomfort or a sense of fullness in the right upper abdominal area.

- Early in the illness, some patients may also report joint pain and swelling, and occasional frank arthritis (Antigen - Antibody mediated).

. A skin rash may also appear in some individuals as part of the body's immune reaction to the virus.

✓ Rule: increased involvement of the liver, increased risk of cholestasis.

- Hepatitis can cause cholestasis, leading to light-colored stools, dark urine, and jaundice due to impaired bile flow.
- Having antibodies to the Hepatitis B surface antigen (HBsAg) gives lifelong immunity

❖ Fulminant hepatitis:

- A rare complication of hepatitis B infection, occurring in less than 1% of cases (icteric patients), and can be life threatening; leads to death.
- Characterized by extensive liver necrosis.

❖ Chronic hepatitis B:

- Develops in approximately 10% of infected individuals, with a significantly higher risk among younger patients, especially neonates. Around 20% of those with chronic hepatitis B may eventually develop hepatocellular carcinoma .
- Approximately 90% of neonates infected with hepatitis B become chronic carriers (The likelihood of developing chronic disease is inversely related to the age at which the infection occurs)
- Hepatocellular carcinoma: caused by the integration of the viral DNA with the DNA of the hepatocyte.

Hepatocellular injury in hepatitis B infection is primarily mediated by the host immune response, especially $CD8^+$ cytotoxic T lymphocytes.
While extrahepatic manifestations such as arthritis and vasculitis result from circulating antigen– antibody complexes.

Jaundice

Increased involvement of the liver ⟶ Jaundice caused by cholestasis Patients are in icteric phase ⟶ Symptoms may persist for several months before resolving.

• HBV in children is generally less severe than in adults and is often asymptomatic.
• Clinically apparent illness occurs in about 25% of infected patients.
• The majority (~90%) recover, typically marked by declining fever and return of appetite.
• Chronic hepatitis develops in 5–10% of cases, often following mild or asymptomatic initial disease.

❖ In those who develop chronic hepatitis:

• About one-third progress to chronic active, with continue destruction of the liver leading to :

1. Liver scarring (cirrhosis)
2. Liver failure
3. Primary hepatocellular carcinoma hepatitis

• The other two-thirds develop chronic passive which is less likely to cause serious issues. (inactive carrier) hepatitis,
• Chronic hepatitis is often discovered incidentally through elevated liver enzyme levels in routine blood tests.
• Chronically infected individual：
  ·Are the main source of HBV transmission.
  ·Are at increased risk of fulminant hepatitis, especially when co-infected with HDV.

Diagnosis of HBV:

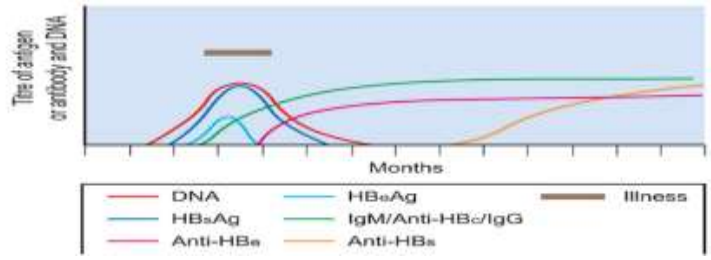
1. Clinical picture
2. Liver chimestry tests (ALT,AST, alkaline phosphatase, total bilirubin) all of them will be find elevated ,
3. Serology:

. Patients with jaundice tend to have IgM antibodies against the hepatitis B core antigen (HBcAg) and may also test positive for hepatitis B surface antigen (HBsAg).

. Previous infections show (either or both) anti-core and anti-surface antibodies in the form of IgG; as it’s a past infection not acute.

• Viral DNA detection by PCR is the most accurate marker.

• Also, to determine the grade of the inflammation and stage of fibrosis, thus prognosis, liver biopsy is examined (especially in chronic hepatitis patient)



This image show typical serology of HBV infection

• IgM antibody to hepatitis B core antigen (Anti-HBc IgM) indicates an acute infection. While, IgG antibody to hepatitis B core antigen (Anti-HBc IgG) suggests either a past resolved infection or chronic hepatitis B.

• Hepatitis B surface antigen (HBsAg) is a general marker of HBV infection, If it stay high for more than 6 months indicates chronic infection.

• The presence of antibodies against HBsAg (anti-HBs) is used to document recovery and immunity from HBV infection .

• The presence of hepatitis B e antigen (HBeAg) indicates active viral replication and infectivity.

• The appearance of antibodies to HBeAg (anti-HBe) suggests a cessation of viral replication. however, patients may still test positive for hepatitis B surface antigen (HBsAg) made by the integrated HBV, indicating ongoing infection .

• HBV DNA detected by PCR is a more accurate indicator of active viral replication than HBeAg, especially in cases of escape mutants.

• DNA is used for monitoring the response to therapy.

TABLE 41–4 Serologic Test Results in Four Stages of HBV Infection

Test	Acute Disease	Window Phase	Complete Recovery	Chronic Carrier State
HBsAg	Positive	Negative	Negative	Positive
HBsAb	Negative	Negative	Positive	Negative[1]
HBcAb	Positive[1]	Positive	Positive	Positive

• This table summarizes serology of HBV infection.

• In the acute disease HBsAG & HBcAb will be positive

• Window phase: the virus is still active, you can't detect both the surface antigen (HBsAg), and antibodies against the surface antigen (anti-HBs) , while at the same time HBcAb is positive .

• How to detect the virus in the window phase? By Anti-HBc IgM.

• In the chronic state HBsAG & HBcAb (IGg) they are positive

• One can notice that the antibody for HBs antigen doesn't appear in blood spontaneously with the antigen itself.

For example, in the acute phase HBsAg is detected while HBsAb is not. Also, after complete recovery HBsAb is detected, while HBsAg is not.

	HBsAg HBeAg* HBV-DNA	HBcAb IgM	HBcAb IgG	HBeAb	HBsAb
Acute infection	+	+	–	–	–
Window period	–	+/–	+	+	–
Prior infection	–	–	+	+	+
Immunization	–	–	–	–	+
Chronic infection	+	–	+	+/–	–

➢ In case of antibodies produced:

▪ Prior infection results in the production of Ab to all Ag.

▪ While immunization results in the production of Ab to only the HBsAg.

Treatment of Hepatitis B virus:

1. No specific treatment for HBV is present.
2. Interferon alpha, used in cases of chronic hepatitis, provides a long-term benefit for 1/3 of patients.
3. High calorie diet is desirable.

Corticosteroid therapy has no proven benefit in the management of uncomplicated acute viral hepatitis

❖ Chemical agents:

1. Lamivudine:

• A potent inhibitor of HIV and also shows activity against hepatitis B virus (HBV), both in vitro and initial clinical trials. However, resistance to the drug develops in approximately 25% of patients after 12 months of therapy.

2. Tenofovir, entecavir

• Because of resistance to Lamivudine, these agents are used.

- Nucleotide analogs of adenosine monophosphate.
- Newly approved for the treatment of chronic hepatitis B.

Prevention of Hepatitis B virus:

1. Avoidance of needle stick injuries by safe practices, for medical personnel and injection drug users.
2. Vaccine (Recombinant HBsAg), 3 I.M doses at 0, 1, 2 OR 6 months

- Vaccination is highly effective.
- Vaccine can be given for individuals at increased risk of infection, such as healthcare workers. It is also routinely administered to neonates in many countries.
- Check response to vaccine by measuring anti HBsAg antibodies 2 months after last dose (>10mIU/ml is protective) → if more than 10 million international units per ml, it's protective.

3.Hepatitis B immunoglobulin (HBIG) may be used to:

1. Protect individuals who exposed to hepatitis B virus (HBV):
   - It is effective as post-exposure prophylaxis if administered within 48 hours of exposure.
2. Provide protection to neonates at high risk of HBV infection

4. Other measures of prevention:
   - Screening of blood donors, blood, and body fluids.

❖ Prevention of HBV in Jordan:

- The Hepatitis B virus component is included in the Hexaxim vaccine (المطعوم السداسي), which is typically administered in 3 doses at the 2nd, 3rd, and 4th months of an infant's life.

Hepatitis D virus

- Small ssRNA virus.
- Causes Delta hepatitis (Hepatitis D).

• HBV provides the envelop, so HDV requires the HBsAg for transmission, that's why HDV is only found in presence of chronic or acute hepatitis B infection

• Delta hepatitis is most prevalent among groups at high risk for hepatitis B, such as injection drug users, up to 50% of these individuals may have IgG antibodies to the delta virus antigen.

• Other at-risk populations include patients undergoing dialysis.

❖ Two Major Types of Hepatitis D (Delta) Infection:

1. Co-infection with HBV and HDV:
   • Occurs when an individual is simultaneously infected with both Hepatitis B virus (HBV) and Hepatitis D virus (HDV).
   • Results in acute clinical hepatitis that is clinically indistinguishable acute hepatitis A or B. from
   • Fulminant hepatitis is significantly more common than with HBV infection alone.
2. Superinfection with HDV:
   • Occurs when a person with chronic HBV infection later acquires HDV.
   • Clinical consequences include:
   • Relapse of jaundice , High risk of developing chronic cirrhosis

Diagnosis:

• Most diagnosed by detecting IgM and/or IgG antibodies against delta antigen in serum.

• IgM appears within 3 weeks of infection and persists for several weeks.

• IgG persists for years.

❖ Treatment:

• The response to treatment (interferon alpha) is poorer in HDV-HBV co-infection compared to HBV infection alone.

• Treatment usually requires:

• Higher doses.

• Sustained improvement is seen in only ~25% of patients.

➢This is due in part to HDV's dependence on HBsAg for its replication.

Prevention:

(limiting transmission of HBV, limits transmission of HDV)

• Individuals with hepatitis B or D should not donate blood, organ tissues or semen.

• Reducing the use of contaminated needles and syringes among people who inject drugs will decrease HBV transmission.

• Use of needle safety devices by healthcare workers.

Hepatitis C

• Flavivirus, Enveloped, RNA virus; a very simple genome, consisting of just 3 structural and 5 non-structural genes. Also, there're regions called quasi species, which are hyper variable regions in the envelop glycol protein

• 6 genotypes, and multiple subtypes

• Spread is still less well understood when compared to transmission of HAV, HBV, and HDV. But can be via:

1.Infected blood, well documented, until screening blood for transfusions was introduced, it caused the great majority of cases of post-transfusion hepatitis.

➢Hepatitis C was the major cause of post-transfusion hepatitis until a serologic test for screening blood donors was developed.

2. Sexual contact, but to a lesser extent than HBV.

3. Needle sharing, accounts for 40% of the cases

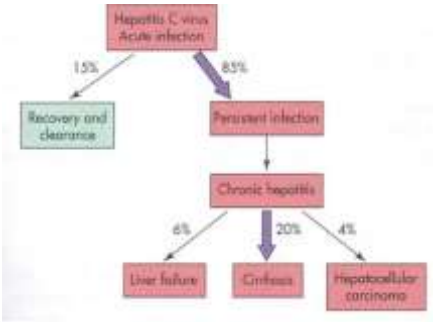
is an insidious disease that typically does not cause a clinically apparent acute illness.

• In about one-quarter of infected individuals, the first manifestation may be smoldering chronic hepatitis, which can eventually progress to liver failure.

•Screening donor blood for hepatitis C antibodies has significantly reduced the incidence of post transfusion hepatitis C by 80% to 90%

• Individuals at risk of HCV infection include chronic hemodialysis patients and spouses of those infected with hepatitis C.

• In the United States, approximately 3.5 million people have antibodies to the hepatitis C virus (HCV)



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6 - 12 weeks incubation period

• After the incubation period, HCV infection can either be: ▪ .Asymptomatic.

.Mild and anicteric, that can result in a chronic infection (carrier state) in up to 85% of infected persons.

• Chronic hepatitis can result in a liver failure and some late sequelae like cirrhosis liver and hepatocellular carcinoma .

• The average time from hepatitis C virus infection to the development of chronic liver disease typically ranges from 10 to 18 years.

Diagnosis:

Anti HCV IgM

• Antigens of hepatitis C are not detectable in the blood; therefore, diagnostic tests rely on detecting antibodies to HCV

. • Unfortunately, the antibody response during acute infection remains negative for 1 to 3 weeks after the onset of clinical symptoms and may never become positive in up to 20% of patients with self-limited acute hepatitis C.

• Now, antibody testing and detection is done by:

• Enzyme immunoassay (EIA)

• Immunoblot assay

• Even with newer assays, IgG antibodies to hepatitis C may take up to 4 months to develop, making the serological diagnosis of acute hepatitis C challenging.

• Quantitative assays of hepatitis C RNA can be used for diagnosis, prognosis estimation, prediction of response to interferon therapy, and monitoring treatment. However, there is a poor correlation between viral load and liver histology.

Treatment:

• The current treatment of choice for hepatitis C is combination therapy with interferon-alpha and ribavirin.

! Corticosteroids are not beneficial in the management of hepatitis C infection.

Prevention:

Important preventive measures include:

1. Avoidance of injection drug use.
2. Screening the blood products.

•No vaccine; not clear whether prophylactic immune serum globulins protect against Hepatitis C.

جدول رقم (9) الاجراءات الفورية بعد اصابة عمل

المريض مصدر الاصابة	الوضع التطعيمي للموظف	الاجراء
التهاب الكبد (B) موجب HBsAg (positive)	- لم يتم تطعيمه - غير مكتمل الجرعات - ثلاث جرعات من التطعيم	-اعطاء التطعيم فورا + جرعة جلوبيولين مناعي* - إكمال كل الجرعات و اعطاء جلوبيولين مناعي* - فحص الاجسام المناعية (اذا كان أكثر أو يساوي 10 وحدة دولية لا شيء) **
التهاب الكبد (B) سالب HBsAg (negative)	- لم يتم تطعيمه - تم تطعيمه	- يتم تطعيمه - لا شيء
غير معروف اصابته بالتهاب الكبد ب	- لم يتم تطعيمه - غير مكتمل الجرعات - ثلاث جرعات من التطعيم	- يعامل كما لو كان مصدر الاصابة ايجابيا - يعامل كما لو كان مصدر الاصابة ايجابيا - يعامل كما لو كان مصدر الاصابة ايجابيا
حامل لمضاد فيروس التهاب الكبد (C)	لا يوجد لقاح للالتهاب الكبد (C)	فحص الموظف بعد الاصابة مباشرة ثم بعد اسبوعين و بعد شهر ثم بعد 3 اشهر بطريقة HCV-Ab و PCR و اذا ظهرت بوادر اصابته يحول الى أخصائي جهاز هضمي
غير معروف اصابته بالتهاب الكبد (C)	لا يوجد لقاح للالتهاب الكبد (C)	فحص الموظف بعد الاصابة مباشرة ثم بعد اسبوعين و بعد شهر ثم بعد 3 اشهر بطريقة HCV-Ab و PCR و اذا ظهرت بوادر اصابته يحول الى أخصائي جهاز هضمي
حامل لفيروس العوز المناعي البشري HIV	لا يوجد لقاح لفيروس العوز المناعي البشري HIV	- مدة اربعة اسابيع يتم فيه تناول ثلاثة ادوية مضادة للفيروسات (مثل زيدوفودين ولاميفودين) ويجب الرجوع الى البرنامج الوطني لمكافحة الايدز *** - يبدأ العلاج فوراً(خلال ساعات)

* يتم ذلك خلال 24 ساعة من التعرض للعدوى
* *تقاس الاستجابة المناعية لمطعوم الكبد (B) بفحص الاجسام المضادة (Hbs Ab) وتعتبر ايجابية اذا كانت اكبر او يساوي 10 وحدة دولية

This high-value table explains post-exposure prophylaxis for different scenarios.