Multidisciplinary

Biomedical Effect for Hepatitis B virus - HBV on Patient

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Abstract
All medical personnel, including those working in medical laboratories and blood banks, must be vaccinated against hepatitis B. All healthcare workers, including physicians, surgeons, dentists, nurses, medical laboratory staff, blood bank personnel, and others, must treat blood, body fluids, and materials contaminated with them as potentially containing HIV, hepatitis viruses, and other blood borne pathogens. Wearing gloves is mandatory when handling clinical specimens and any other materials that may be contaminated with blood or body fluids. Violation of this rule will not be tolerated, regardless of position. All medical laboratory staff must refrain from using their mouths to draw solutions with straws, eating, drinking, or smoking while working in these laboratories. Intravenous drug users and those who engage in promiscuous or non-monogamous sexual activity are at higher risk of contracting hepatitis B and C viruses, as well as HIV (the virus that causes AIDS). Hepatitis is a type of liver infection that affects a person's health and can lead to serious complications such as cirrhosis, liver failure, and cancer. Vaccination plays a crucial role in preventing the disease, in addition to adhering to personal hygiene practices and guidelines for preventing the spread of infection. The liver is the largest organ in the body and plays a vital role in metabolism, converting food into energy, and removing toxins from the body. It also secretes bile, a greenish fluid that plays an important role in digestion. Some patients may not show symptoms or signs of infection, which can lead to liver failure and cirrhosis, followed by the appearance of symptoms and signs that are difficult to treat and can last for a long time. There are several causes of hepatitis, not limited to viruses. Some medications can cause liver inflammation, as can autoimmune diseases. Viral infections are among the most common of these diseases. When the inflammation persists for more than six months, we refer to this type as chronic hepatitis. Acute hepatitis results from the virus settling in the liver and multiplying rapidly, leading to swelling and rupture of liver cell walls, as well as an intensive spread of various types of white blood cells throughout the liver to limit the spread of the virus. This inflammation usually lasts for a short period. It is worth noting that acute hepatitis rarely leads to chronic damage, unlike chronic hepatitis, which can cause neurological disorders and, in its severe forms, lead to hepatic coma. Therefore, infected individuals may experience symptoms of acute hepatitis for a few days or weeks, but upon recovery, the patient is completely cured, and no side effects or chronic liver damage remain. However, in rare cases, the patient's condition deteriorates during the acute phase of the inflammation to the point of death or the urgent need for a liver transplant.

Keywords: hepatitis B, HIV (the virus that causes AIDS), must treat blood, body fluids

Introduction

Viral hepatitis means the destruction of liver cells, which are among the most important organs in the human body. The liver produces approximately 1,000 different enzymes. Hepatitis has also become widespread among patients with hemophilia (a blood clotting disorder) who are treated with clotting agents that were once obtained from the blood of thousands of donors before the virus was discovered. Infection also occurs among people without these risk factors, for reasons that are not yet fully understood. Simple environmental measures can reduce the risk of infection for all healthcare workers, including those in medical laboratories, blood banks, and other healthcare settings. These measures include treating all blood, body fluids, and materials contaminated with them as potentially containing HIV, hepatitis B and C viruses, and other blood borne pathogens.

Hepatitis B virus – HBV

Hepatitis B virus (HBV), also known as hepatitis B, is widespread globally. It is estimated that over 251 million people are carriers of this virus. Approximately 25% of carriers will develop symptoms of chronic active hepatitis. Worldwide, about one million people die annually from hepatitis and liver cancer caused by this virus. HBV is transmitted through blood and blood products containing the virus, which are often obtained from healthy carriers. Many people become infected through the use of improperly sterilized needles and syringes, including for tattooing and ear piercing. The virus can also be transmitted through unprotected sex and from an infected mother to her fetus.

Studies conducted in the 1990s in both Benghazi and Tripoli revealed that the hepatitis B virus (HBV) was present in 26% of hepatitis patients and in approximately 6% of drug addicts. The virus was also found in less than 3% of blood donors. A 2006 study found the virus in 7% of prisoners in the western region. More recently, a study found hepatitis B in approximately 3% of kidney failure patients attending dialysis units in Libya. Hepatitis B is a viral infection that affects the liver, destroying liver cells and causing cirrhosis and liver cancer. There are several other viruses that cause hepatitis and share many symptoms and clinical signs, including yellowing of the skin and eyes (jaundice), fatigue, nausea, vomiting, and

abdominal pain. However, most people infected with these viruses do not feel ill and recover completely. A small percentage of patients may develop acute hepatitis and fatal liver failure (8 to 10%). In these cases, the disease progresses to a chronic form, and over the years, the liver may become cirrhotic, increasing the risk of liver cancer. Most infections occur during childhood and early adulthood, which is where the danger of developing chronic hepatitis lies. The incidence is much higher if a person is infected during childhood, reaching up to 90% in the first year of life and approximately 30-50% in the following three years. The mortality rate from cirrhosis and liver cancer is about 25% for those infected during childhood.

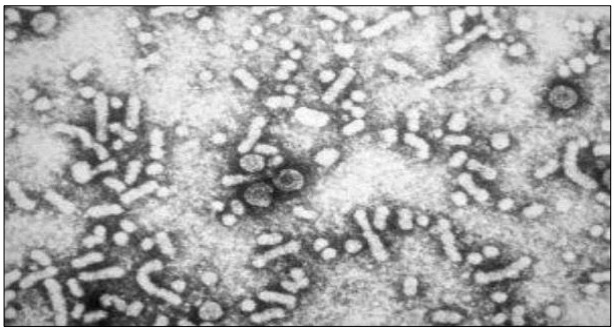

Fig 1

Signs and Symptoms of Hepatitis B

Acute hepatitis B infection is associated with acute viral hepatitis.

This disease presents with general weakness, loss of appetite, nausea, vomiting, body aches, mild fever, and darkening of the urine, which may then develop into jaundice. Skin itching has been observed as a symptom that can appear in all types of hepatitis viruses. The disease lasts for several weeks and then gradually improves in most infected individuals. However, some patients will suffer from more severe hepatitis (fulminant liver failure), which can lead to death. The infection may be asymptomatic and remain undetected.

Chronic hepatitis B infection may be asymptomatic or associated with chronic liver inflammation, leading to cirrhosis after several years. This type of infection automatically increases the incidence of hepatocellular carcinoma (liver cancer). Approximately 50% of hepatocellular carcinoma cases in Europe are caused by hepatitis B and C. Alcohol consumption should be avoided as it increases the risk of cirrhosis and liver cancer. A link has also been found between hepatitis B virus and the development of membranous hepatitis and nephritis. Extrahepatic symptoms such as acute necrotizing vasculitis (polyarteritis nodosa), membranous hepatitis and nephritis (glomerulonephritis), and infantile papular dermatitis (Crossti-Gianotti syndrome) appear in 1-10% of patients with hepatitis B. Clinical symptoms include fever, rash, and polyarthritis. These symptoms often begin shortly after the onset of jaundice but can persist throughout the course of hepatitis B infection. Acute

Approximately 30-50% of patients with acute necrotizing vasculitis (polycystic arthritis) are chronic carriers of hepatitis B virus (HBV). HBV is associated with renal impairment, which has been described in adults but is more common in children, with membranous hepatitis and nephritis being the most frequent complication. It can also be associated with other immunomodulatory hematological disorders such as aplastic anemia and cryoglobulinemia.

The nucleoplasm encapsulates the viral DNA and DNA polymerase, which is responsible for reverse transcription. The outer envelope contains embedded proteins that play a role in binding to and entering target cells.

Hepatitis B virus is one of the smallest enveloped animal viruses. Virion particles with a diameter of 42 nanometers, capable of infecting liver cells, are known as Dan particles. In addition to Dan particles, filamentous and spherical bodies lacking a core can be found in the serum of infected individuals. These particles are non-infectious and are composed of lipids and proteins that form part of the virion's surface, called HBsAg, which is produced in abundance during the virus's life cycle.

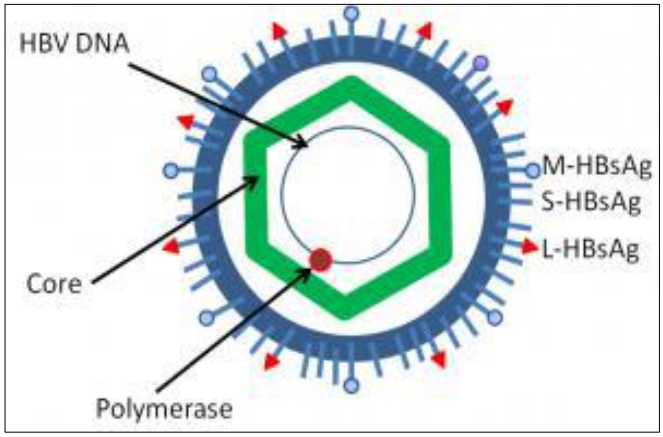

Fig 2

The Hepatitis B virus (HBV) genome

consists of circular DNA, but it has an unusual shape because the DNA is double-stranded (incomplete). One end of the completed strand is attached to viral DNA polymerase. The genome is 3020–3320 nucleotides long (the complete strand), while the shorter strand is 1700–2800 nucleotides long. The negative-sense (non-coding) strand complements the viral messenger RNA (mRNA). Viral DNA is present in the nucleus shortly after infection. The double-stranded DNA is partially restored to complete by completing the positive-sense strand and removing a protein molecule from the negative-sense strand, in addition to removing a short RNA sequence from the same strand. The non-coding bases are also removed from the ends of the negative-sense strand, and then the ends are rejoined. The genome contains four genes: s, c, x, and p. The core protein is encoded by the c gene for the antigen (HB c Ag). Its start codon is preceded by the AUG start codon, located upstream in-frame, from the start site towards the end, from which the core protein progenitor is produced. The coating antigen (HB e Ag) is produced by the lytic cleavage of the core protein progenitor.

DNA polymerase is encoded by the p gene. The s gene encodes the surface antigen (HB s Ag). The surface antigen gene is a single, long, open reading frame, but it contains three start codons. The ATG pattern within the frame divides the gene into three parts: pre-s1 & s, and pre-s2.

Because of the presence of multiple start codons, polypeptides of different sizes are produced: large, medium, and small. (Pre-s1 + Pre-s2 + s, Pre-s2 + s, or s). The protein coded by the X gene has not yet been precisely defined, but it is associated with the development of liver cancer, activating genes that promote cell growth and inhibiting the activation of growth-regulating molecules.

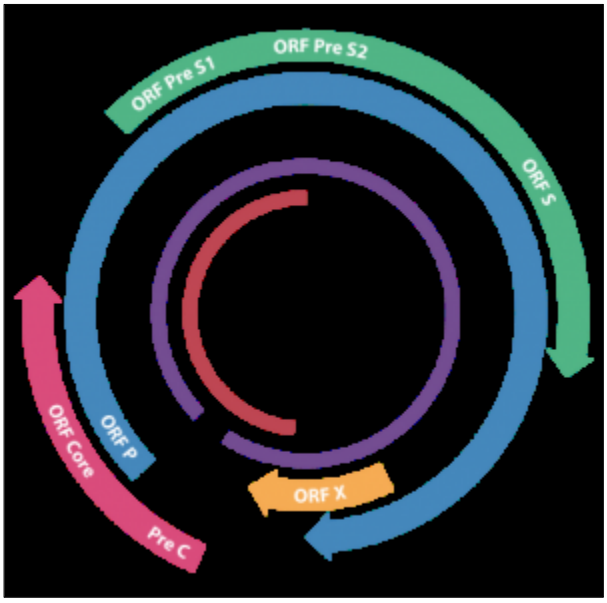

Fig 3

Serological and Genotype Classification:

The virus is classified into four main serotypes (adr), ayr, adw, and ayw). This classification is based on the epitopes present on the envelope proteins. The virus is classified genotypically into eight types (A-H) according to nucleotide sequence variations in the genome. These genotypes have diverse geographic distributions, which is used to track the evolution and transmission of the virus. Genotype variation affects disease severity, progression, and the likelihood of complications, as well as response to treatment, and may also impact vaccine development. Genotypes differ by at least 8% of their sequences. This was first documented in 1988 when the first six types (A-F) were described. The remaining two types were described later. Most genotypes are further subdivided into subclasses, each with its own distinct characteristics. The mechanism of hepatitis B virus infection: Hepatitis B virus primarily involves Liver function is affected by its replication within hepatocytes, as the functional receptor is NTCP 38. There is evidence that this receptor, which is very similar to the hepatitis B virus receptor in ducks, is carboxypeptidase.

Virions bind to the host cell via the pre-S domain of the viral surface antigen and enter the cell sequentially through endocytosis. Pre-S domain receptors are primarily found on hepatocytes, and viral DNA and proteins have been detected at extrahepatic sites, suggesting the possibility of extrahepatic cell receptors for hepatitis B virus.

During hepatitis B infection, the host's immune response causes hepatocyte damage in addition to viral clearance. Although the innate immune response does not play a significant role in this process, the adaptive immune response, particularly cytotoxic T lymphocytes (CTLs) specific to hepatitis B virus (HBV), is responsible for most of the liver damage associated with HBV infection. CTLs clear HBV infection by killing virus-infected cells and producing antiviral cytokines, which are subsequently used to clear viable hepatocytes of HBV.⁴⁸ While liver damage is initiated and mediated by CTLs, non-specific inflammatory cells can further impair CTL function, leading to pathological immunopathology. Additionally, platelet activation at the site of infection can facilitate the accumulation of CTLs in the liver.

Diagnosis of Hepatitis B Virus (HBV) Infection

Investigations to determine hepatitis B virus (HBV) infection include serum and blood tests to identify either viral antigens (proteins produced by the virus) or antibodies produced by the host. Detection of the hepatitis B virus surface antigen (HBsAg) is the most commonly used test for HBV infection. It is the first detectable viral antigen during infection, but it may not be detectable in the early stages. It may also be undetectable in later stages due to clearance by the host. The infectious virion contains an endospore that encapsulates the viral genetic material. This icosahedral endospore consists of 180 or 240 copies of the endospore protein and is known as the hepatitis B virus core antigen (HBcAg). During the window in which the host remains infected but has successfully cleared the virus, acute-phase antibodies (IgM) are produced. The hepatitis B virus (HBV) core antigen may be the only serological evidence of infection; therefore, most diagnostic kits for HBV include surface antigen and total antibodies directed against the virus's core antigen (acute phase antibodies + chronic phase antibodies).

Shortly after the appearance of the surface antigen, another antigen called the envelope antigen (HbeAg) appears. Typically, the appearance of these antigens in the host serum is associated with very high rates of viral replication and severe infectious activity. However, in some cases, the hepatitis B virus does not produce the envelope antigen, so this rule is not always true. During natural hepatitis B infection, the envelope antigen can be cleared, and antibodies directed against the envelope antigen appear directly. This serological shift is always associated with a significant decrease in viral replication. If the host is able to clear the infection, the surface antigen will eventually become undetectable, and antibodies against the hepatitis B virus surface antigen and the hepatitis B core antigen (anti-HBs and anti-HBc IgG) will rise.³¹ The period between the disappearance of the surface antigen and the appearance of antibodies against the surface antigen (anti-HBs IgG) is called the window period. A person who is negative for the surface antigen but positive for antibodies against the surface antigen indicates a previous

infection or that the person has previously received the hepatitis B virus vaccine. Individuals who remain positive for the surface antigen for at least six months are considered carriers of the hepatitis B virus. Individuals carrying the hepatitis B virus (HBV) can develop chronic HBV infection. This is reflected in elevated serum liver enzymes (ALT) and inflammation of the liver, which occurs when they are in the process of clearing chronic infection.

Carriers who have undergone seroconversion from positive to negative serostatus, particularly those who were infected as adults, have very low viral replication and therefore may have a lower risk of long-term complications and a lower rate of transmission to others compared to HBV-positive patients. PCR tests are used to determine and measure the number of HBV DNA copies, also known as viral load. These tests are used to assess the patient's infection status and monitor treatment. Patients with a high viral load are characterized by hepatocytes that appear glassy on biopsy.

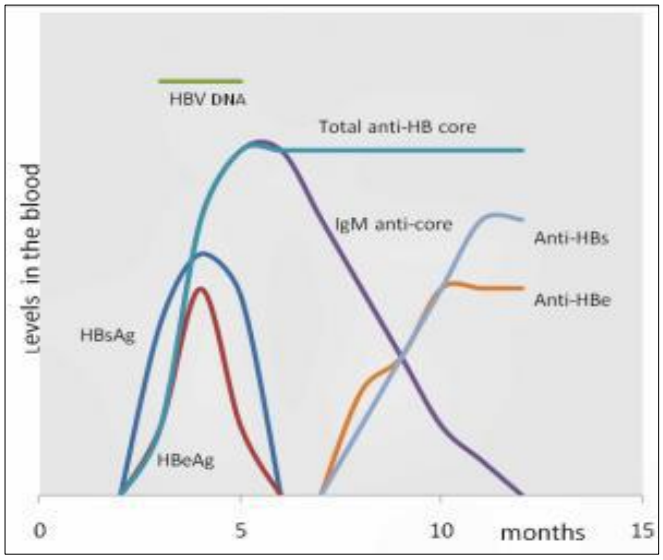

Fig 4: Antibodies and antigens that appear in the blood of a person infected with acute hepatitis C

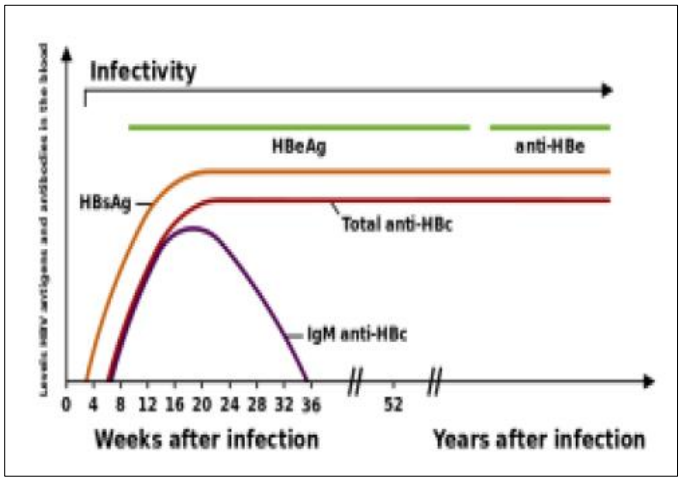

Fig 5

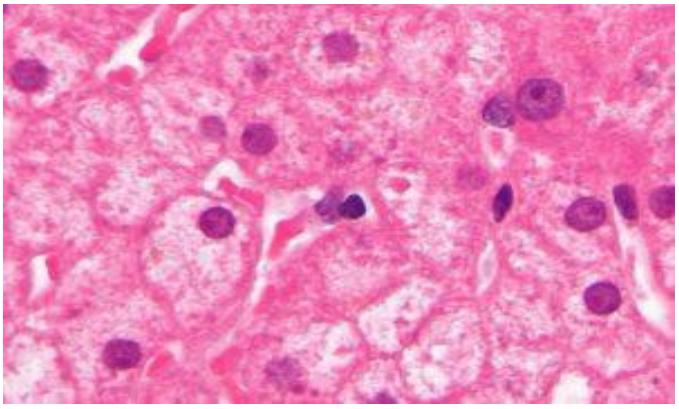

Fig 6: Liver cells as seen in a sample taken from the liver of a person with chronic hepatitis B. Hematoxylin and eosin staining.

Prevention of Hepatitis B

Vaccination has been routinely recommended for the prevention of hepatitis B since 1991 in the United States. Most vaccines are given in three doses over several months. The protective response to the vaccine is defined as the concentration of antibodies against the hepatitis B virus surface antigen, which is at least 10 mIU/ml in the recipient's serum.

The vaccine is most effective in children, with 95% of them having sufficient antibody levels to protect against hepatitis B infection. This percentage decreases to 90% by age 40 and to 75% over age 60. The protection provided by the vaccine lasts for a long time, even after the antibody level falls below 10 mIU/ml. It is also recommended that all newborns born to mothers infected with hepatitis B be vaccinated. Concomitant administration of hepatitis B immunoglobulin with the vaccine immediately after birth prevents perinatal transmission of hepatitis B in 86% to 99% of cases. All individuals at risk of exposure to bodily fluids such as blood should receive the hepatitis B vaccine if they have not already been vaccinated.⁵⁶ Testing is also recommended.

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