IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

Report On Hepatitis B Virus: Comprehnsive Review Of Epidermiology, Pathogenesis And Treatment

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Abstract:

When Hepatitis-B is diagnosed, the quest for a reliable curative treatment in any medical system starts. Which physician should I see? Which doctor specializes in treating Hepatitis B or C infections, or the gastroenterologist with the largest list of alphabets after his name? How is the doctor chosen? Excellent question as well. You should see a physician with extensive clinical experience in treating infections caused by Hepatitis B or C. Instead of choosing someone with book knowledge of Hepatitis B or C, look for a physician with a long history of treating Hepatitis B and C cases alone. Practice is only focused on clinical abilities and knowledge, whereas study allows one to earn more degrees.

Hepatitis B virus (HBV) remains a major global health challenge, affecting millions of individuals and contributing significantly to chronic liver disease, cirrhosis, and hepatocellular carcinoma. This comprehensive review summarizes current knowledge on HBV epidemiology, virology, modes of transmission, clinical manifestations, and diagnostic approaches. It highlights recent advances in understanding the viral life cycle, host immune responses, and mechanisms of persistence that complicate therapeutic eradication. Current prevention strategies—including universal vaccination, maternal screening, and antiviral prophylaxis—are discussed alongside available antiviral therapies that suppress viral replication

and reduce long-term complications. Despite substantial progress, HBV cure remains elusive due to the stability of covalently closed circular DNA (ccc DNA) and viral integration into host genomes.

Keywords: Hepatitis B Virus, Hepadnaviridae, DNA Virus, Acute Hepatitis, Chronic Hepatitis

Introduction:

Hepatitis refers to the general condition of liver inflammation. This condition can arise not only from excessive activity of the immune system but also due to external factors, most commonly viral infections. An immunological response elicited by the replication and other components of a virus may lead to either an acute or chronic infection. The effects of this condition can vary significantly depending on both the host and the specific virus involved in the infection. The predominant cause of viral hepatitis is the Hepatitis B virus (HBV), which represents a significant contributor to end- stage liver disease globally. Despite the availability of an effective vaccine for HBV infection, new cases continue to emerge, attributable in part to inadequate vaccination coverage as well as challenges related to the accessibility, availability, and cost of vaccines in regions most affected by the disease. Additionally, another source of new infections arises from viral breakthrough, which may occur in up to 5% of infants who have received the anti-HBV birth-dose vaccine in a timely manner. [7, 8] Deregulated immune function is a hallmark of some liver diseases, in which the body's.

Hepatitis B virus (HBV) is a major global health concern and one of the leading causes of viral hepatitis, chronic liver disease, cirrhosis, and liver cancer. It is a small, partially double-stranded DNA virus belonging to the Hepadnaviridae family and has a unique ability to persist in liver cells through its stable ccc DNA form. HBV is transmitted mainly through exposure to infected blood and body fluids, including mother-to-child transmission, unprotected sexual contact, sharing of needles, and unsafe medical practices. The infection can present as acute hepatitis with symptoms such as fever, jaundice, fatigue, and abdominal pain, but many individuals may remain asymptomatic. When the virus remains in the body for more than six months, it can develop into chronic hepatitis B, leading to long-term complications such as liver fibrosis, cirrhosis, and hepatocellular carcinoma. Diagnosis is made through blood tests that detect viral antigens, antibodies, and DNA levels, along with liver function tests and imaging when needed. Prevention is highly effective through vaccination, which provides long-lasting immunity, along with safe injection practices, screening of pregnant women, and safe sexual behaviour. While acute HBV often resolves without treatment, chronic HBV is managed with antiviral medications like tenofovir and entecavir or interferon therapy to suppress viral replication and reduced liver damage.

NATURAL HISTORY OF HEPATITIS B VIRUS INFECTION

The hepatitis B virus was identified in 1965 by Dr. Baruch Blumberg, who was awarded the Nobel Prize for this significant discovery. Initially, the virus was referred to as the "Australia Antigen," a name derived from a blood sample of an Australian aborigine that exhibited a reaction with an antibody found in the serum of an American patient with hemophilia. Collaborating with Dr. Blumberg, microbiologist Irving Millman contributed to the creation of a blood test for the hepatitis B infection. This test was received by blood banks in 1971 to screen blood gifts, coming about in a 25 percent lessening in the hazard of hepatitis B contaminations related with blood transfusions. Four a long time taking after the recognizable proof of the hepatitis B infection, Drs. Blumberg and Millman defined the to begin with hepatitis B immunization, which was initially a heat-inactivated adaptation of the infection.

The natural history of hepatitis B virus (HBV) infection varies depending on the age and immune status of the infected person. After exposure, individuals may develop an acute infection that can range from asymptomatic to symptomatic with jaundice and liver inflammation. Most healthy adults are able to clear the virus and develop lifelong immunity, while a small percentage progress to chronic infection. In contrast, infants and young children infected early in life have a much higher risk—up to 90%—of developing chronic hepatitis B. Chronic infection can remain inactive for years or progress through phases of immune tolerance, immune clearance, and low or high viral replication. Over time, persistent HBV infection can lead to liver fibrosis, cirrhosis, liver failure, and hepatocellular carcinoma.

First Commercial Hepatitis B Vaccine

In 1981, the FDA asserted a more present day plasma-derived hepatitis B counter acting agent for human utilize. This “inactivated” sort of counter acting agent included the collection of blood from hepatitis B virus-infected (HBsAg-positive) advocates. The pooled blood was subjected to diverse steps to torpid the viral particles that included formaldehyde and warm treatment (or “pasteurization”). Merck Pharmaceuticals made this plasma counter acting agent as "Heptavax," which was the to start with commercial hepatitis B contamination counter acting agent.

ETIOLOGY

Hepatitis B is spread through a variety of routes from infected individuals to non-immune individuals. The following are the main ways that hepatitis B is spread: Horizontal transmission: This refers to the spread of hepatitis B via intercourse or contact with mucosal surfaces. In regions with low to intermediate prevalence, unprotected sex and injectable drug use are the main ways that the disease is spread.

The etiology of hepatitis B involves infection with the Hepatitis B virus (HBV), a DNA virus from the Hepadnaviridae family that primarily targets liver cells. HBV is transmitted through exposure to infected

blood or body fluids, including perinatal transmission, unprotected sexual contact, sharing needles, and unsafe medical procedures. Once inside the body, the virus replicates in liver cells, leading to inflammation and potential liver damage.

1. Causative Agent

Hepatitis B Virus (HBV):

HBV is a little infection characterized by somewhat double-stranded DNA and is classified inside the Hepadnaviridae family. Its replication prepare is particular, including turn around translation, which adjusts it with certain retroviruses

2. Transmission:

HBV spreads through blood and body fluids. Common routes include:

1. Vertical transmission: From an infected mother to her baby during childbirth (most common in high-prevalence areas)
2. Horizontal transmission: Through contact with contaminated blood or bodily fluids, often in childhood (cuts, scrapes, or bites).
3. Sexual contact: Unprotected intercourse with an infected person.

"Transmission" applies to hepatitis B virus (HBV), not to the vaccine itself; the vaccine does not spread from person to person and cannot cause HBV infection because it contains only purified hepatitis B surface antigen, not whole live virus. Instead, vaccination is given specifically to block the main transmission routes of HBV: perinatal (mother to child at birth), early childhood household contact, sexual exposure, and blood or body-fluid contact (e.g., shared needles, unsafe injections, transfusions with unscreened blood, or needle-stick injuries in healthcare).

EPIDERMIOLOGY

Virus Structure And genome

The Hepatitis B virus (HBV) is the prototype virus of the Hepadnaviridae family of viruses. Members of this family are hepatotoxic DNA viruses known to infect birds and mammals (Orthohepadna viruses). Additionally, fish and amphibian Hepadnaviruses have been reported recently. These viruses share similarities in their genome organization as well as their replication approach, with up to 40% and 20% sequence diversity amongst orthohepadna viruses and avihepadna viruses, respectively. Three types of HBV-virion particles are usually observed in the serum of infected persons.

The infectious virion, also known as the Dane particle, is 42–45 nm in diameter, made up of HBsAg embedded in a lipid envelope, encasing the viral nucleo capsid containing a reverse transcriptase tethered to the nucleic-acid material.

The other two are sub viral particles (22–24 nm), filamentous and spherical in shape, both comprising HBs

Ag embedded in a host-derived lipid membrane but lacking viral DNA. Interestingly, the sub viral particles outnumber the infectious particles by 100 to 100,000-foldin the blood and play immune modulatory and immune inhibitory roles

. HBV infection has the potential for progression to a chronic state and thus presents as a global public health threat for its associated morbidity and mortality. While hepatitis B vaccines are available, limited access to healthcare and lack of proper health education contributes to the increasing global prevalence of hepatitis B.

Hepatitis B is a widespread global infection, with an estimated hundreds of millions of people living with chronic HBV worldwide, particularly in regions such as East Asia, Southeast Asia, and Sub- Saharan Africa where early-life transmission is common. The prevalence varies widely, ranging from low rates in North America and Western Europe to high endemicity in parts of Africa and Asia. HBV is most commonly transmitted from mother to child at birth, through early childhood exposure, unprotected sexual contact, and contaminated needles. Despite effective vaccination programs, HBV remains a major cause of chronic liver disease, cirrhosis, and liver cancer worldwide.

PATHOGENESIS

HBV can be acquired through two main routes parenatally, from infected mothers to their new-born, which accounts for a majority of cases worldwide, and horizontal transmission through contact with an infected person's body fluids, equipment for body piercing, tattoos, and injecting-drug use. Generally, the mechanisms by which HBV accesses and gains entry into hepatocytes is not fully understood.

The heparin Sulphate proteoglycans (HSG's), sodium taurocholate co-transporting peptide (NTCP), and the epidermal growth factor receptor (EGFR) are some of the receptors that mediate this internalization although there are likely to be others. Both the NTCP and HSG's are hepatocyte- specific receptors, thus explaining the virus' hepatotropic nature. In-depth studies into these receptor interactions could significantly contribute to finding a cure to HBV through inhibition of viral spread within the liver. Recently, some compound leads have been shown to selectively inhibit the virus- receptor function of NTCP.

The pathogenesis of hepatitis B is driven by the virus's ability to infect liver cells (hepatocytes) and replicate using its stable ccc DNA form, but most of the liver injury results from the body's immune response rather than the virus itself. When HBV enters hepatocytes, the immune system recognizes infected cells and attacks them, leading to liver inflammation and damage. A strong immune response can clear the infection, while a weak or immature response—especially in infants—allows the virus to persist and become chronic. Over time, chronic inflammation can cause fibrosis, cirrhosis, and increase the risk of hepatocellular carcinoma.

CAUSES AND RISK FACTORS OF HEPATITIS-B

Hepatitis B follows a similar mode of transmission as the human immunodeficiency virus (HIV), the agent responsible for AIDS. Both are transmitted through exposure to infected blood or blood products, sexual

contact and from mothers to infants primarily at birth. However, hepatitis B appears to be far more infectious than HIV. In addition to the ways in which HIV is spread, hepatitis B appears to be spread by casual contact. It can be acquired by close contact within families, or from person to person through contact with open skin lesions.

SYMPTOMS OF HEPATITIS B VIRUS

Symptoms and signs of hepatitis B can range from none to minimal in the early stages of the illness, to jaundice (yellowing of the skin), nausea, abdominal pain, fever, and malaise in the acute phase. Appetite loss, fatigue, itching, dark urine and pale stools are some common symptoms.

Acute Hepatitis B Symptoms

1. Fever
2. Fatigue or general malaise
3. Loss of appetite
4. Nausea and vomiting
5. Abdominal pain (especially in the upper right side near the liver)

Chronic Hepatitis B Symptoms

1. Persistent fatigue and weakness
2. Joint pain
3. Abdominal pain or discomfort
4. Jaundice (if liver function deteriorates) Early diagnosis and treatment can help manage symptoms, prevent complications, and reduce the risk of liver damage for those with chronic HBV.

TREATMENT OF HEPATITIS B

Getting advice or professional opinion is NOT a free service, as you are communicating with the world's one & only clinical specialist on Hepatitis B. Once you are diagnosed to have Hepatitis B or C, you have only 2 options, either meet Dr. John K C OR die of Liver Cancer/Cirrhosis. This statement looks awkward, but it is a frank reality, you should admit it. Public behaves like a fool or idiot when this health issue occurs to them or to any family members. They select a doctor on what basis? They assume that if they meet a specialist in a very big hospital, their all problems would be solved permanently. They do not know anything that is happening in a medical field. All specialists work in any hospital for a monthly pay of 2 lakhs rupees after signing an agreement with management to provide that hospital, a fixed income on monthly basis -‘target’ it is called.. In short, their monthly salary is based on this ‘target’. So specialists do wanted and mostly unwanted tests on the patients like blood tests, , CT scan, Endoscopy, MRI scan, Biopsy etc...

PREVENTION

Hepatitis B is preventable with a vaccine. All babies should receive the hepatitis B vaccine as soon as possible after birth (within 24 hours). This is followed by two or three doses of hepatitis B vaccine at least four weeks apart. Booster vaccines are not usually required for people who have completed the three-dose vaccination series. To reduce the risk of getting or spreading hepatitis B: practice safe sex by using condoms and reducing the number of sexual partners avoid sharing needles or any equipment used for injecting drugs, piercing, or tattooing wash your hands thoroughly with soap and water after coming into contact with blood, body fluids, or contaminated surfaces get a hepatitis B vaccine if working in a healthcare setting.

Prevention of hepatitis B relies mainly on vaccination, which is highly effective and provides long- term protection when given as a three-dose series starting at birth. Preventive measures also include screening pregnant women to prevent mother-to-child transmission, practicing safe sex, avoiding sharing needles or sharp objects, and ensuring the use of sterile equipment in medical and dental settings. Safe blood transfusion practices and proper infection control in healthcare environments further reduce the risk of transmission. Together, these strategies play a crucial role in controlling and preventing the spread of HBV.

CURRENT RESEARCH

Current research on Hepatitis B virus (HBV) is primarily focused on finding a cure, understanding immune system interactions, and improving prevention. Key areas include developing novel antiviral drugs, gene-editing techniques, and therapeutic vaccines to eliminate HBV or enhance the immune response against it. Additionally, there's a focus on discovering biomarkers for better disease monitoring and advancing personalized medicine to tailor treatments. Research also addresses HBV-related liver cancer risks, co-infection with HIV, and expanding vaccination efforts globally to prevent new cases. These efforts aim to improve outcomes for the millions affected by HBVworldwide

LITERATURE REVIEWS

The point of view on Hepatitis B virus (HBV) centres around its significant public health impact and the urgent need for improved management and a cure. HBV is recognized as a major global cause of chronic liver disease, cirrhosis, and liver cancer. Current perspectives emphasize the importance of advancing antiviral therapies, developing effective therapeutic vaccines, and understanding immune interactions to achieve functional cures. Additionally, there's a focus on expanding prevention strategies, especially in high-prevalence areas, through widespread vaccination and better diagnostics. Ultimately, the goal is to reduce the HBV burden and prevent disease progression, ideally leading to global eradication.

CONCLUSION:

The observations we made indicate a need for prevention and control of, generally, serum hepatitis in hyperendemic and low-resourced countries, especially in the West African sub-region. There is the need for operative strategies which requires comprehensive investments to interrupt the transmission of serum hepatitis and reduce the consequential morbidity and mortality. The importance of expanding research in the field of HBV cannot be overstated. There is a pressing need to elevate efforts in HBV research to precisely assess prevalence rates, identify at-risk populations, establish treatment priorities, and deepen our comprehension of host-pathogen interactions that could ultimately lead to a cure.

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