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REVIEW ARTICLE **OPEN ACCESS**

Immunohistopathology of Hepatitis B Virus

Solomon Matthias Gamde[1] , Sani Fawziyyah Adamu[2] , Alheri Lawan Lailai[3] , Bukola Ajide[4] , Alexander Philip[4]
1 Department of Medical Laboratory Science, Plateau State University, Nigeria
2 Department of Medical Laboratory Science, Bingham University Karu, Nigeria
3 Public Health, Community Medicine and Primary Health Care, Bingham University Karu, Nigeria
4 Department of Microbiology, Bingham University Karu, Nigeria
Corresponding Author Email: solomonmatthias85@gmail.com
*ORCID: https://orcid.org//0000-0002-7631-8782 *Tel: +2348131943803

ABSTRACT
Hepatitis B Virus is a global health concern, causing both acute and chronic liver diseases. The immune response to the virus determines the clinical outcomes of infection, ranging from acute infection to persistent chronic conditions. In acute hepatitis B viral infection, conspicuous lymphocytes and other mononuclear cells infiltrate portal tracts and sinusoids throughout the hepatic 1obules and are often in apposition to hepatocytes. The latter showed evidence of cell injury which may present in forms, ballooning degeneration and cytolysis or coagulative necrosis and formation of acidophilic or apoptotic bodies.
Keywords*: Hepatitis B Virus, Immunology, Histopathology, Liver disease*

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INTRODUCTION

Hepatitis B virus (HBV) infection is a global health challenge, about 296 million people are affected with chronic HBV as of 2019 [1]. The infection could progress to severe liver diseases such as liver cirrhosis and hepatocellular carcinoma (HCC). Despite advancements in vaccination and antiviral therapies, immune response to HBV infection presents significant challenges complicate efforts to achieve complete viral clearance and cure. The idea of immune evasion is a clear proof that the virus avoids removal by the immune system and use some mechanisms to elude the immune response, as well as the production of large quantities of non-infectious sub viral particles that can sequester antibodies, reducing their effectiveness [2]. Additionally, HBV can mutate to escape recognition by immune cells. The precise mechanisms of immune elusion remain undefined, but likely interconnected between the innate and adaptive immunities where the innate immune system (particularly NK and dendritic cells) displays impaired function, leading to inadequate activation of the adaptive immune response and allowing the virus to persist. There are evidences that supports peripheral tolerance and the exhaustion of adaptive immunity. However, the role of the central tolerance is still developing [3, 4].

Many individuals, especially those infected perinatally or in early childhood, develop an immune-tolerant phase where the immune system does not effectively recognize or respond to the virus. This results in high viral loads with minimal liver inflammation, allowing the virus to persist. The immune system often fails to completely eliminate from the liver, leading to chronic infection HBV. This is primarily due to the virus's ability to establish a stable form of viral DNA, called covalently closed circular DNA (cccDNA), in the nuclei of hepatocytes, which acts as a reservoir for viral replication, making it difficult for the immune system and current antiviral therapies to completely eradicate the virus [5]. Over time, key immune cells like CD8+ T cells become exhausted, losing their ability to target and eliminate HBV-infected cells effectively. With the common uses of interferons alpha (IFN-α) and nucleos(t)ide analogues (NUCs) approved treatments, which suppresses HBV replication, neither of them eliminates the virus mechanistically. Hence, the risk of hepatocellular carcinoma (HCC), being a major cause of morbidity and mortality worldwide. There has been significant interest in developing therapies that could modulate immune system to restore the ability to combat HBV effectively. Therapeutic vaccines aim to boost the immune system's ability to fight HBV by enhancing T-cell responses. While prophylactic HBV vaccines are effective in preventing infection,

therapeutic vaccines for those already infected are still under development and have shown mixed results [6,7, 8].

The persistence of HBV and the problems in eradicating it highlight the need for novel treatments that address both the viral and immune components of the disease. A deeper understanding of the immune dysfunctions associated with HBV and the development of therapies that can effectively target these dysfunctions are critical for achieving long-term viral suppression or cure. The combination of new antiviral agents, immunotherapies, and personalized therapy techniques shows promise for overcoming current hurdles and improving patient outcomes.

LITERATURE REVIEW

Hepatitis B Virus is a global health concern, causing both acute and chronic liver diseases. The immune response to the virus determines the clinical outcomes of infection, ranging from acute infection to persistent chronic conditions. In acute hepatitis B, conspicuous numbers of Lymphocytes and other mononuclear cells infiltrate portal tracts and sinusoids throughout the hepatic 1obules and are often in apposition to hepatocytes. The latter show evidence of cell injury which may present in 2 forms, i.e. ballooning degeneration and cytolysis or coagulative necrosis and formation of acidophilic or apoptotic bodies [9].

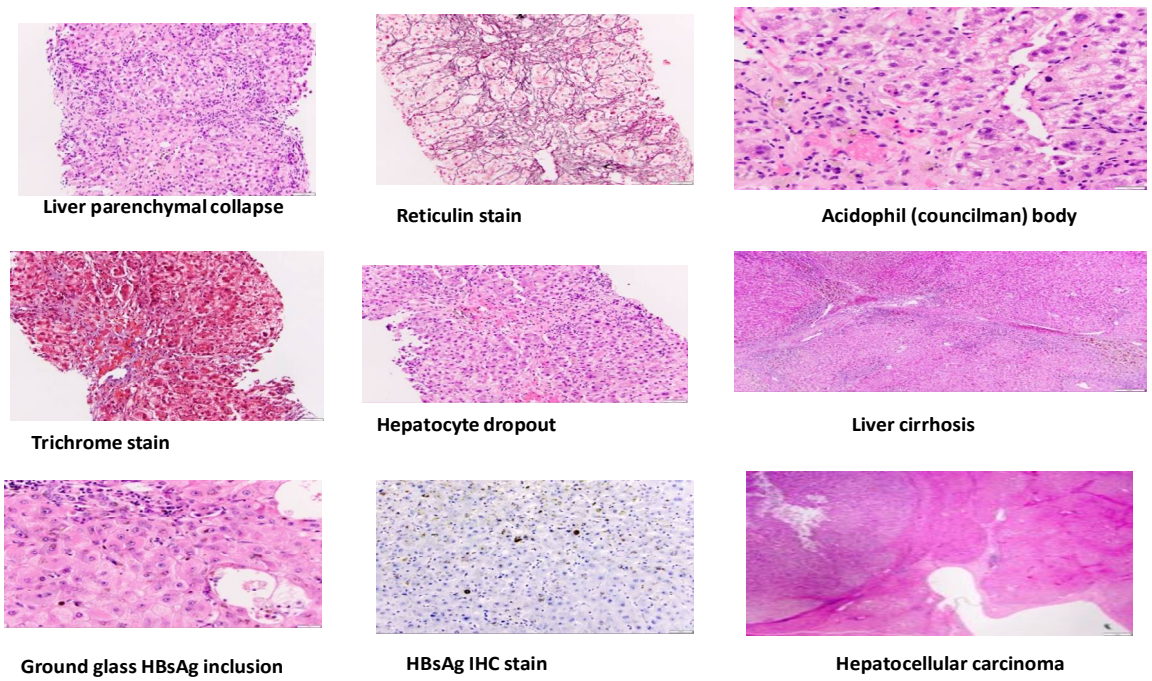

Figure 1 showing cellular injury in HBV infection

Innate Immune Response in HBV Infection

Innate immune system serves as the first line of defense against HBV infection [3]. When HBV enters hepatocytes, it does not strongly trigger pattern recognition receptors (PRRs) like Toll-like receptors (TLRs), retinoic acid-inducible gene-I (RIG-I)-like receptors, or nucleotide-binding oligomerization domain (NOD)-like receptors [9]. As a result, the virus is adept at evading the early innate immune response, which is partly why acute HBV infection can often be asymptomatic [10].

Nonetheless, natural killer cells and dendritic cells play essential roles in guiding HBV replication. NK cells exert direct antiviral activity through cytokine production (e.g., interferon-γ) and cytolytic activity, while DCs initiate adaptive immune responses [11]. However, chronic HBV infection is associated with dysfunctional NK cells and impaired DC activity, contributing to viral persistence [12].

Adaptive Immune Response in HBV Infection

The adaptive immune response is crucial in the clearance of HBV [2]. It primarily involves HBV-specific cytotoxic T lymphocytes (CTLs) and helper T cells (Th cells). During acute HBV infection, a robust and polyclonal CTL response leads to the clearance of infected hepatocytes, accompanied by the production of neutralizing antibodies by B cells [3]. These antibodies target the hepatitis B surface antigen (HBsAg),

playing a crucial role in neutralizing the virus and preventing re infection. However, in chronic HBV infection, the adaptive immune response is typically weak and ineffective [10]. CTLs often become functionally exhausted due to prolonged exposure to high levels of viral antigens. Exhausted CTLs express high levels of inhibitory receptors such as PD-1, TIM-3, and CTLA-4, reducing their ability to clear infected cells [8]. Furthermore, regulatory T cells (Tregs) are frequently upregulated in chronic HBV infection, suppressing antiviral T cell responses and promoting viral persistence.

Humoral Immunity in HBV Infection

Humoral immunity, mediated by B cells, is another critical component of the immune response against HBV. In acute infection, the production of anti-HBs antibodies is associated with viral clearance. These antibodies prevent the virus from infecting new cells and neutralize circulating viral particles. In contrast, in chronic HBV infection, the production of neutralizing antibodies is often inadequate or delayed, contributing to the prolonged presence of HBsAg in the bloodstream [9]. Additionally, chronic HBV carriers are characterized by elevated levels of hepatitis B core antigen (HBcAg)-specific antibodies (anti-HBc). However, the presence of anti-HBc does not necessarily lead to viral clearance but serves as an important marker of past or ongoing infection.

Cytokine Response in HBV Infection

Cytokines are signaling molecules that orchestrate immune responses. In acute HBV infection, pro-inflammatory cytokines like tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), and interferon-γ (IFN-γ) by immune cells helps control viral replication. IFN-α, in particular, has been used therapeutically to boost immune responses in chronic HBV patients, as it enhances the antiviral state of infected hepatocytes and promotes T cell activity.

In addition to the cytokines reviewed Interleukin (IL)-1 [14,15,16], Interleukin (IL)-2 [17,18,19], Interleukin -4 [20,21], Interleukin -6 [22,23], Interleukin -10 [24], Interleukin –12 [25], Interleukin -21 [26,27], Interleukin -22 [28], Interleukin -23 [25], interferon [29], Tumor Necrosis Factor-Alpha [30], and Transforming Growth Factor-Beta **(**TGF-β) [31,32].

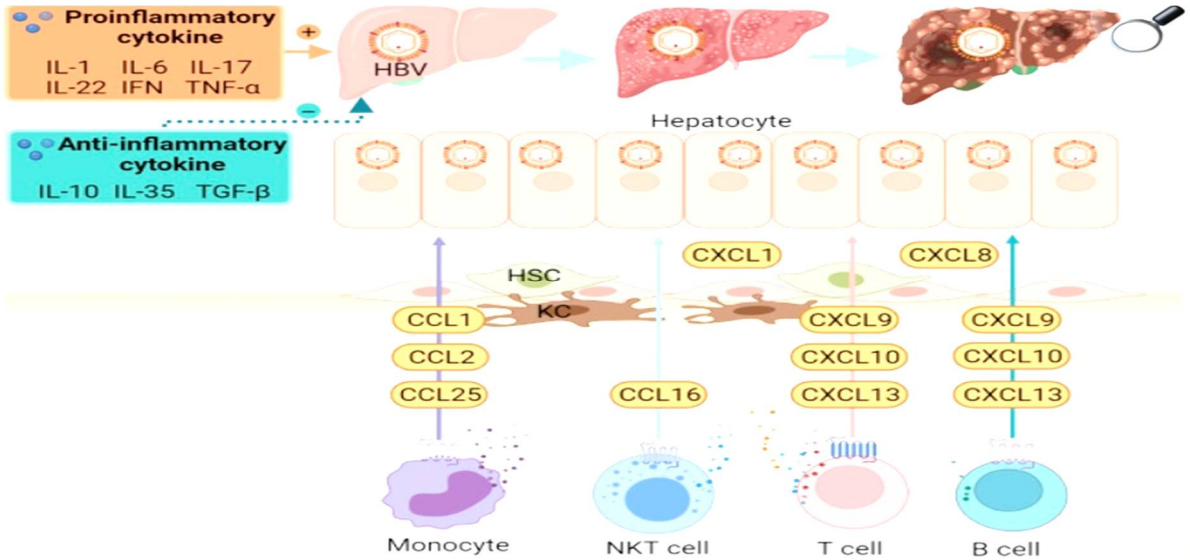

Figure 2 showing important inflammatory cells in HBV pathogenesis

Mechanisms of Immune Evasion by HBV

HBV has evolved several strategies to evade immune detection and clearance. These mechanisms include:

1. Low Innate Immune Activation: HBV has a stealth-like ability to avoid early detection by the innate immune system, resulting in minimal activation of antiviral responses.
2. T Cell Exhaustion: Chronic exposure to HBV antigens leads to T cell exhaustion, reducing their effectiveness in clearing the virus.
3. Hepatocyte Immune Privilege: The liver is considered an immunologically tolerant environment, where immune responses are naturally suppressed to prevent excessive inflammation in a vital organ. This tolerogenic environment contributes to the persistence of HBV in chronic infection.
4. HBsAg Overproduction: HBV produces large quantities of HBsAg, which acts as a decoy to neutralize anti-HBs antibodies, preventing their effective action against the virus.

Immune Therapies for HBV

Immune therapies for hepatitis B virus (HBV) are designed to enhance the immune system's ability to control or eliminate the virus, aiming for a functional cure characterized by sustained loss of hepatitis B

surface antigen (HBsAg) and viral control without long-term medication. Unlike nucleos(t)ide analogs, which suppress viral replication, immune therapies target immune pathways and responses [4].

1. Therapeutic Vaccines

Therapeutic vaccines aim to stimulate HBV-specific immune responses. These include vaccines containing HBV antigens such as HBsAg or core antigens combined with adjuvants to enhance T cell activation [9].

2. Immune Checkpoint Inhibitors

Immune checkpoint inhibitors, such as anti-PD-1 or anti-PD-L1 antibodies, block inhibitory pathways that suppress immune responses. By doing so, they rejuvenate exhausted HBV-specific T cells, allowing them to better target HBV-infected cells [8,33].

3. Cytokine-Based Therapies

Cytokines such as interferon-alpha (IFN-α) and interleukins (e.g., IL-2, IL-12) promote immune activation. Pegylated interferons, with improved pharmacokinetics, remain a key component of HBV immune therapies, though side effects limit their use [34].

4. Toll-Like Receptor (TLR) Agonists

TLR agonists activate innate immune pathways to stimulate antiviral responses. TLR7 and TLR8 agonists, like vesatolimod, promote cytokine production and adaptive immune activation [35].

5. Engineered T Cells

T-cell therapies involve engineering T cells to specifically target HBV-infected cells. Examples include chimeric antigen receptor (CAR) T cells and T-cell receptor (TCR)-redirected therapies, both of which show promise in preclinical studies [8].

6. RNA-Based Therapies

RNA-based therapies, such as small interfering RNAs (siRNA) and antisense oligonucleotides, reduce viral antigen expression, alleviating immune exhaustion and potentially restoring immune function [36].

7. Innate Immune Modulators

Stimulating the innate immune system through agents like STING (stimulator of interferon genes) agonists or NKG2D receptor activators can enhance antiviral responses [4].

8. Combination Therapies

Combining immune therapies or pairing them with direct-acting antivirals has shown synergistic effects, improving the likelihood of functional cure [9].

Implications for Treatment and Prevention of HBV Infection

The treatment and prevention of Hepatitis B Virus (HBV) infection are critical public health concerns due to its potential to cause chronic liver disease, cirrhosis, and hepatocellular carcinoma (HCC) [37]. The following sections discuss the implications for treatment and prevention based on recent advances in HBV research.

Treatment of HBV Infection

1. Antiviral Therapy

The primary goal of antiviral therapy is to suppress viral replication, reduce liver inflammation, and prevent disease progression to cirrhosis or HCC. The two main classes of antiviral treatments for HBV are nucleos(t)ide analogs (NAs) and interferon (IFN) therapy.

- Nucleos(t)ide Analogs (NAs): These are oral antiviral drugs, including tenofovir and entecavir, that inhibit HBV DNA replication. NAs are highly effective in reducing viral load and improving liver histology. However, they require long-term use, as they do not target the covalently closed circular DNA (cccDNA) form of HBV, which remains in hepatocytes and is responsible for viral persistence. Long-term suppression can prevent liver damage but may not completely eradicate the virus, necessitating indefinite treatment for some patients [38].

- Interferon Therapy: Pegylated interferon-α is used to boost the immune system's ability to control HBV. Although treatment with interferon is limited by its side effects and variability in patient response, it can offer finite treatment duration, with some patients achieving a functional cure (defined as sustained HBsAg loss).

Implications: Antiviral therapy with NAs significantly reduces the risk of cirrhosis and HCC but is not curative. The development of therapies that can eliminate cccDNA or improve immune responses remains crucial for achieving a complete cure. Combining NAs with new therapies that target immune pathways, such as checkpoint inhibitors or therapeutic vaccines, may offer more effective long-term outcomes.

2. Immune-Based Therapy

Given the role of immune exhaustion in chronic HBV infection, restoring immune function is a promising approach.

- Checkpoint Inhibitors: Chronic HBV infection leads to T-cell exhaustion due to continuous exposure to viral antigens. Immune checkpoint inhibitors (PD-1 blockers) are being studied to restore T-cell function and allow for more effective viral clearance. Although these inhibitors have shown promise in cancer

therapy, their application in HBV is still under research, with early studies focusing on their ability to reverse immune exhaustion.
- Therapeutic Vaccines: Therapeutic vaccines aim to boost immune responses in chronic HBV patients by stimulating HBV-specific T-cells. Unlike preventive vaccines, these are designed to enhance the immune system's ability to control or eliminate the virus in infected individuals.
Implications: Immune-based therapies could offer a way to re-engage the immune system in controlling chronic HBV infection. By combining these therapies with antivirals, it may be possible to achieve long-term control of the virus without lifelong medication.
3. Gene-Editing Technologies
CRISPR/Cas9 and other gene-editing tools are being explored to target and disrupt HBV cccDNA in infected hepatocytes. By editing the viral genome or eliminating cccDNA, these technologies aim to achieve a permanent cure.
Implications: Gene-editing technologies have the potential to cure HBV by eliminating the source of viral persistence. However, challenges remain in safely delivering these tools to infected cells and avoiding off-target effects. Continued research is needed to ensure the efficacy and safety of these approaches [39].

Prevention of HBV Infection

1. Vaccination
The HBV vaccine is the most effective preventive measure, dramatically reducing the incidence of new infections. The recombinant HBV vaccine induces protective antibody responses by targeting HBsAg, preventing the establishment of HBV infection. Vaccination programs have been highly successful in many countries, particularly when integrated into routine childhood immunization schedules.
- Universal Vaccination: The World Health Organization (WHO) recommends that all infants receive the HBV vaccine, with the first dose administered within 24 hours of birth. This early vaccination significantly reduces mother-to-child transmission, one of the primary routes of HBV infection in high-prevalence regions.
- Targeted Vaccination for High-Risk Groups: Other high-risk populations, such as healthcare workers, individuals with multiple sexual partners, and intravenous drug users, should also receive the HBV vaccine to prevent infection.
Implications: Vaccination remains the cornerstone of HBV prevention. Efforts to increase vaccination coverage, particularly in low- and middle-income countries, are essential to achieving global HBV control. Continued investment in vaccination programs, along with initiatives to improve vaccine accessibility, can reduce the incidence of chronic HBV and associated complications.
2. Prevention of Mother-to-Child Transmission (PMTCT)
Perinatal transmission of HBV is a significant cause of chronic HBV infections, particularly in highly endemic areas. To prevent transmission from mother to child, WHO recommends that all newborns of HBV-infected mothers receive HBV immunoglobulin (HBIG) and the first dose of the HBV vaccine within 12 hours of birth? Pregnant women with high viral loads can also be treated with NAs to reduce the risk of transmission.
Implications: Preventing mother-to-child transmission is critical to reducing the global burden of chronic HBV. Improved access to maternal screening, antiviral therapy, and vaccination at birth are essential components of this strategy. As more countries adopt the universal birth-dose vaccination, chronic HBV infection in the future generations will decline.
3. Behavioral and Public Health Measures
Prevention strategies also include public health campaigns aimed at reducing behaviors that increase the risk of HBV transmission, such as needle sharing and unprotected sexual activity. Harm reduction programs, including needle exchange and safe injection practices, can help reduce HBV transmission among people who inject drugs.
Implications: Education and harm reduction initiatives are vital components of HBV prevention, particularly in high-risk populations. These efforts, combined with vaccination, can significantly reduce the transmission of HBV [40].

CONCLUSION

While a robust immune response can lead to viral clearance in acute infection, chronic HBV is marked by immune dysfunction, including T cell exhaustion and immune evasion mechanisms. Understanding these immune dynamics is critical for developing novel therapeutic strategies aimed at enhancing antiviral immunity and ultimately achieving a functional cure for HBV infection. The treatment and prevention of HBV infection have seen significant advancements, particularly with the development of effective antiviral therapies and vaccines. However, challenges remain, especially in achieving a functional or complete cure for chronic HBV infection. Future research into immune-based therapies, gene-editing technologies, and

improved global vaccination programs will be essential for reducing the incidence of HBV and its associated health complications. Preventive measures, particularly vaccination and PMTCT programs, will continue to play a pivotal role in controlling HBV infection worldwide.

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