REVIEW ARTICLE

Overview of Hepatitis B and Human Immunodeficiency Virus Coinfection

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ABSTRACT

Hepatitis B Virus (HBV) and Human Immunodeficiency Virus (HIV) co-infection is a major public health issue, especially in areas where both viruses are common. Despite the serious health problems associated with having both HBV and HIV, the specific immune interactions between these viruses are not fully understood. This review looks at how these two viruses interact in the body, affecting the immune system and overall health. When someone has both infections, it can lead to faster damage to the immune system, higher levels of virus in the body, and more severe liver problems, including cirrhosis and liver cancer. This research should aim to identify specific immune responses, which could lead to better treatment options for people with both infections. Improved understanding may also help with vaccine development and optimize the use of current antiviral treatments. Treatments like antiretroviral therapy, which can help manage both HBV and HIV, have shown effectiveness. However, treating both infections at the same time is complicated due to issues like drug resistance, a condition known as immune reconstitution inflammatory syndrome (IRIS), and overlapping side effects from the medications. This review also points out important gaps in research, especially regarding the long-term effects of having both infections and how to improve treatment plans for better patient outcomes. Future research should focus on new treatment methods, the role of immune boosters, and personalized treatment strategies to tackle the unique challenges of HBV-HIV co-infection.

Keywords: Hepatits B virus, human immunodeficency virus, immunology, pathology

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INTRODUCTION

Co-infection with Hepatitis B virus (HBV) and Human Immunodeficiency Virus (HIV) represents a significant clinical challenge, particularly in regions with high endemicity such as sub-Saharan Africa and Southeast Asia. These two viruses, which share similar modes of transmission—primarily blood borne, perinatal, and sexual routes—affect millions of individuals globally.[1] According to the World Health Organization (WHO), approximately 37.7 million people were living with HIV by the end of 2020, with an estimated 10% co-infected with HBV. This co-infection is associated with accelerated liver disease progression, a higher likelihood of antiretroviral therapy (ART) complications, and increased morbidity and mortality compared to monoinfected individuals.[2]

This review aims to provide a comprehensive overview of the immune interactions between HBV and HIV, explore the implications for treatment and management of co-infected patients, and highlight the current research gaps that need to be addressed to improve clinical outcomes. By examining recent advancements

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in immunology and virology, we seek to propose new avenues for therapeutic research and clinical practices that can mitigate the impact of HBV-HIV co-infection. The interaction between HIV and HBV is complex because each virus affects the body in different ways, which can change the immune response and complicate how the diseases progress and are treated.[3] HIV mainly targets immune cells, leading to a decrease in CD4+ T-cells, while HBV primarily attacks liver cells, causing ongoing liver inflammation. In people who have both infections, the immune suppression caused by HIV can worsen HBV replication and liver damage, increasing the risk of serious conditions like cirrhosis, liver failure, and liver cancer (HCC).[4]

Despite the serious health problems associated with having both HBV and HIV, the specific immune interactions between these viruses are not fully understood. Recent studies suggest that HIV not only helps HBV persist in the body but also changes how the immune system normally responds to HBV infection.[5] Individuals co-infected with both viruses often show signs of uncontrolled immune activation, chronic inflammation, and weakened immune response, which complicates treatment.[6] Given these challenges, there is a strong need for more research to clarify the immune interactions in HBV-HIV co-infection. This research should aim to identify specific immune responses, which could lead to better treatment options for people with both infections. Improved understanding may also help with vaccine development and optimize the use of current antiviral treatments.

HIV (Human Immunodeficiency Virus) is a retrovirus (Figure 1) that attacks the immune system, specifically targeting CD4+ T cells, which are crucial for immune defense. By compromising these cells, HIV reduces the body's ability to fight infections and diseases, leading to immunodeficiency. If left untreated, HIV can progress to AIDS (Acquired Immunodeficiency Syndrome), a severe stage marked by a critically weakened immune system and opportunistic infections or cancers.

Hepatitis B Virus (HBV) is a DNA virus belonging to the family Hepadnaviridae (Figure 2). It primarily infects liver cells (hepatocytes) and is the causative agent of hepatitis B, a viral infection characterized by liver inflammation. The virus is highly infectious and transmitted through blood, bodily fluids, or vertical transmission from mother to child during childbirth. HBV can cause both acute and chronic infections, with chronic infection potentially leading to serious complications, including liver cirrhosis, liver failure, and hepatocellular carcinoma (HCC).

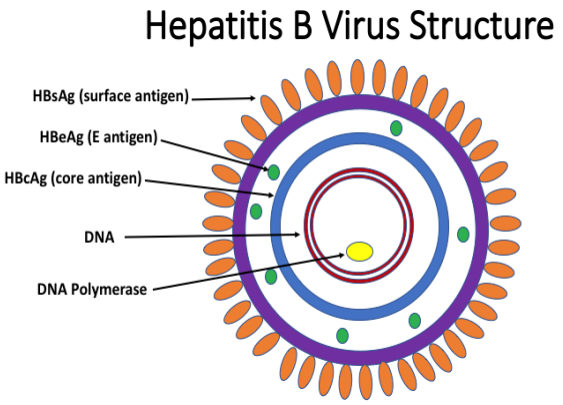

Figure 1: Ultrastructure of the human immunodeficiency virus (HIV)

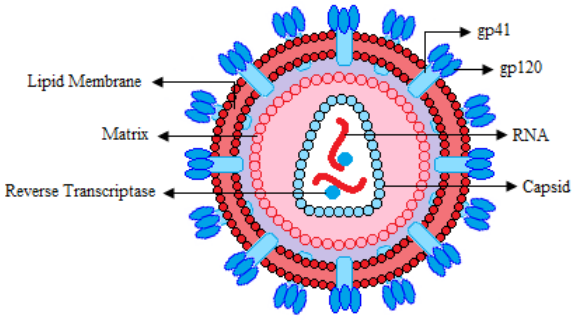

Figure 2: Ultrastructure of the hepatitis B virus (HBV)

PREVALENCE OF HBV AND HIV

HBV and HIV infections frequently coexist due to shared transmission routes, including sexual contact, perinatal transmission, and exposure to infected blood. Co-infection rates are disproportionately high in regions with endemic HBV, such as sub-Saharan Africa and East Asia.[7] Understanding prevalence is critical for guiding treatment protocols and allocating resources effectively. Hepatitis B virus (HBV) and human immunodeficiency virus (HIV) co-infection is a significant global health challenge due to shared transmission pathways and the compounded effects of co-infection on disease progression. The prevalence of HBV-HIV co-infection among people living with HIV (PLWH) is estimated to be 5–15%

globally,[8] translating to approximately 2.7–3.7 million individuals affected. The prevalence varies substantially by region, reflecting differences in HBV endemicity, HIV prevalence, vaccination coverage, and healthcare infrastructure.

In the Sub-Saharan Africa, people living with HIV ranges between 15–25%, making it the region with the highest co-infection rates.[9] Factors driving these high rates include endemic HBV and the significant burden of HIV. Transmission occurs predominantly through sexual contact and vertical transmission, compounded by limited access to HBV vaccination and early diagnosis tools. In the Asia-Pacific Region, the co-infection prevalence is approximately 10–15%. This region has a large pool of chronic HBV carriers (estimated at over 100 million), overlapping with concentrated HIV epidemics in key populations, such as men who have sex with men (MSM) and people who inject drugs (PWID).[10]

In the North America and Europe, the prevalence is lower, around 5–8%, reflecting robust HBV vaccination efforts and lower HBV endemicity with people who inject drugs (PWID). Co-infection rates are higher among specific subgroups, such as PWID and immigrants from HBV-endemic regions.[11] The Middle East and North Africa prevalence estimates are limited but range between 5–10%, influenced by intermediate HBV endemicity and the varying prevalence of HIV while in the Latin America and the Caribbean, the prevalence is relatively low, generally under 5%, except in populations with significant presentation with PWID.[12]

PATHOLOGIES OF INFECTIONS

Co-infection with hepatitis B virus (HBV) and human immunodeficiency virus (HIV) has widespread effects on various organ systems, resulting in compounded health challenges. This analysis highlights the major organs impacted by HBV-HIV co-infection with supporting evidence and quantitative data.

1. Liver: The liver is the most affected organ in HBV-HIV co-infection due to the direct pathogenicity of HBV and the immunosuppressive effects of HIV.[13] The co-infection accelerates liver fibrosis and cirrhosis, with increased risks of hepatocellular carcinoma (HCC) and liver-related mortality. Studies show a 3–6-fold higher risk of advanced liver fibrosis in co-infected individuals compared to HBV mono-infected persons.[14] The incidence rate of HCC in HBV-HIV co-infected individuals is reported to be 114 per 100,000 person-years, compared to 52 per 100,000 in HBV mono-infected populations.[15]

2. Immune System (Lymphoid Organs): HIV primarily depletes CD4+ T cells, weakening immune defences, while chronic HBV further dampens immune responses through persistent immune exhaustion.[4] Co-infection elevates systemic immune activation, contributing to faster HIV progression and reduced immune reconstitution during antiretroviral therapy (ART). A 50–70% reduction in CD4+ T cell counts is observed in co-infected individuals compared to those with HIV alone. Immune activation markers, such as IL-6 and soluble CD14, are significantly elevated in co-infected patients, correlating with higher morbidity risks.[16]

3. Kidneys: Both viruses contribute to kidney disease through direct infection, immune complex deposition, and drug toxicity. HBV-associated glomerulonephritis and HIV-associated nephropathy (HIVAN) are common in this population.[17] Up to 40% of co-infected individuals experience renal impairment, significantly higher than in mono-infected cohorts prevalence exceeds 30%, indicating substantial renal involvement in many co-infected individuals.[18]

4. Cardiovascular System: Co-infection elevates cardiovascular risk due to chronic inflammation, metabolic derangement, and ART-related lipid abnormalities.[19] Persistent immune activation exacerbates endothelial dysfunction, atherosclerosis, and thrombosis. Cardiovascular event rates, including myocardial infarction, are significantly increased in co-infected populations.

5. Central Nervous System (CNS): HIV directly affects the CNS, leading to neurocognitive disorders, while HBV contributes indirectly through systemic inflammation and liver-related encephalopathy.[20] Co-infection amplifies CNS immune activation, resulting in more severe neurocognitive impairments. Cognitive impairment rates in co-infected patients range from 35–50%, higher than in HIV mono-infected individuals. Biomarkers of neuroinflammation, such as neopterin and β2-microglobulin, are significantly elevated in co-infected cohorts, reflecting heightened CNS involvement.[21]

IMMUNE INTERACTIONS

The immune interactions in HBV and HIV co-infection are multifaceted and involve both the innate and adaptive immune systems. HIV's attack on CD4+ T-cells and the resulting immune suppression critically affects the body's ability to control HBV, leading to persistent viral replication and chronic liver disease. In contrast, HBV's chronic infection state leads to immune exhaustion, which exacerbates HIV progression by creating a more permissive environment for viral replication.[5]

1. Innate Immune Response: The innate immune system is the body's first line of defense against infections like HBV and HIV. In people who are co-infected with both viruses, these immune responses are significantly weakened, mainly because important immune cells, such as dendritic cells (DCs) and natural killer (NK) cells, are reduced. These cells are vital for recognizing viruses through specific receptors known as pattern recognition receptors (PRRs), including Toll-like receptors (TLRs), which help activate the interferon (IFN) signaling pathway.[22]

In individuals with HBV-HIV co-infection, NK cells show reduced effectiveness. This means they have lower ability to kill infected cells and produce important signaling proteins like IFN-γ and TNF-α.[23] Furthermore, the constant activation of DCs in response to ongoing viral presence leads to a state known as immune exhaustion, where these cells become less effective. These problems in the innate immune response allow the viruses to persist in the body and contribute to the ongoing inflammation seen in co-infected individuals.[24]

2. Adaptive Immune Response: The adaptive immune response to HBV and HIV is mediated primarily by CD4+ and CD8+ T-cells. In co-infected individuals, HIV significantly depletes CD4+ T-cells, which impairs the ability to mount an effective immune response to HBV. The depletion of CD4+ T-cells not only reduces the capacity to generate effective HBV-specific cytotoxic T lymphocyte (CTL) responses but also impairs the production of neutralizing antibodies, allowing HBV to persist in the liver.[25] Moreover, CD8+ T-cell exhaustion is a hallmark of HBV-HIV co-infection. These T-cells, which are responsible for killing HBV-infected hepatocytes, express high levels of exhaustion markers such as PD-1, TIM-3, and CTLA-4, leading to their functional impairment.[26] As a result, HBV-specific immune responses are blunted, allowing for unchecked viral replication and progressive liver damage.

3. Chronic Inflammation and Immune Activation: Chronic immune activation is a key feature of both HIV and HBV infections. In co-infected individuals, this activation is more pronounced, driven by ongoing viral replication and persistent antigen stimulation.[27] Elevated levels of pro-inflammatory cytokines such as IL-6, TNF-α, and IFN-γ are commonly observed, contributing to liver fibrosis and cirrhosis.[28] Additionally, HIV's ability to impair the immune system's regulatory mechanisms exacerbates the inflammatory response to HBV, creating a vicious cycle of immune activation and liver damage.

IMPLICATIONS FOR TREATMENTS

The management of HBV-HIV co-infection presents significant challenges due to the interplay between the two viruses and their impact on the immune system. Current treatment strategies for co-infected individuals must consider both HIV suppression and HBV control while minimizing the risk of drug resistance and hepatotoxicity.

Antiretroviral Therapy (ART) and HBV Coinfection: The cornerstone of HIV treatment is antiretroviral therapy (ART), which has dramatically improved survival rates and quality of life for people living with HIV. In the context of HBV co-infection, certain ART regimens, particularly those containing tenofovir and lamivudine, have dual activity against both HIV and HBV.[4] Wang et al. reported that patients receiving TDF experienced significant reductions in HBV DNA levels, leading to improved liver function.[29] However, the prolonged use of these drugs can lead to HBV resistance, particularly in patients who discontinue treatment or have suboptimal adherence.[30] Furthermore, ART-related hepatotoxicity is a concern, as the liver is already compromised in co-infected individuals.

HBV Treatment in Coinfected Individuals: HBV treatment in the context of HIV co-infection is primarily focused on reducing viral replication and preventing liver disease progression. Nucleoside/nucleotide analogues, such as tenofovir, are the mainstay of HBV therapy, as they effectively suppress HBV DNA levels and reduce the risk of liver fibrosis and hepatocellular

carcinoma.[31] However, long-term suppression of HBV is difficult to achieve, and viral reactivation remains a risk, particularly when ART regimens are altered or discontinued.

Management of HBV Reactivation: Reactivation of HBV during ART for HIV is a critical concern. A systematic review by emphasized the importance of screening for HBV before initiating ART. Prophylactic antiviral therapy with NRTIs should be considered for all HIV-positive individuals with a history of HBV infection. Drug Interactions is very common with co infected individuals, Clinicians must be aware of potential drug interactions between ART and HBV treatments. For instance, the use of certain protease inhibitors may exacerbate liver toxicity.[32] Regular monitoring of liver enzymes and HBV DNA levels is crucial for managing co-infected patients. Regular monitoring every 3–6 months can help in timely intervention.

Patient Management and Care: Multidisciplinary Approach is an effective management of HBV and HIV co-infection it requires a collaborative approach involving hepatologists, infectious disease specialists, and primary care providers. Regular follow-up and patient education on liver health and ART adherence are vital.

Psychosocial Considerations is addressing the psychosocial aspects of co-infection and is crucial. Patients may experience stigma and mental health challenges, which can impact adherence to treatment. Integrating mental health services into routine care can enhance treatment outcomes.

Challenges and Future Directions for Treatment: One of the major challenges in the treatment of HBV-HIV co-infection is the development of drug resistance. Mutations in the HBV polymerase gene can lead to resistance to nucleoside analogues, complicating the management of co-infected patients.[33] Additionally, the hepatotoxic effects of ART necessitate careful monitoring of liver function, particularly in patients with advanced liver disease.

Emerging therapies, such as PD-1 inhibitors and TLR agonists, offer promising new avenues for the treatment of HBV-HIV co-infection.[23] These therapies aim to restore immune function and enhance the clearance of infected cells, potentially leading to better control of both viruses. However, more clinical trials are needed to determine the safety and efficacy of these therapies in co-infected individuals.

Future Directions for Research and Clinical Practice: Despite the critical nature of HBV and HIV co-infection, several research gaps persist. There is a need for longitudinal studies to assess the long-term impact of immune interactions on treatment outcomes. Furthermore, investigations into the mechanisms of immune evasion and the role of co-infection in the development of liver-related complications are under explored. Studies focusing on the role of microbiota and its influence on immune modulation in co-infected patients also warrant further attention.[34]

Investigating Immune Evasion Mechanism: A deeper understanding of the immune evasion strategies employed by both HIV and HBV is critical for the development of new therapies. HIV's ability to deplete CD4+ T-cells and induce immune exhaustion in CD8+ T-cells remains a significant barrier to effective treatment.[35] Immunotherapies that target these immune dysfunctions, such as checkpoint inhibitors, hold promise for enhancing immune control of HBV in co-infected individuals.

Long-term Outcomes and Public Health Implications: There is a need for long-term studies to evaluate the outcomes of co-infected patients on ART and HBV therapy. These studies should focus on the incidence of liver-related complications, including fibrosis, cirrhosis, and hepatocellular carcinoma, as well as the impact of drug resistance on treatment efficacy.[36] From a public health perspective, expanding HBV vaccination programs, particularly in HIV-positive populations, is essential for reducing the global burden of co-infection. Additionally, the potential of gene-editing technologies, such as CRISPR, to cure both HBV and HIV should be explored in future research.[37]

Research Innovation: There is a need for more research on HBV and HIV co infection in the future in other to properly manage it, new therapeutic agents under investigation include nucleotide analogs and immune modulators. For example, EASL 2023 presented data on a new class of HBV-targeting therapies that may reduce HBV replication without exacerbating HIV.[38] Ongoing trials are evaluating the efficacy of therapeutic vaccines for HBV, which could play a role in preventing HBV reactivation in co-infected patients.

Personalized Medicine: Genetic profiling may guide the choice of antiretroviral therapy (ART) and Hepatitis B (HBV) therapy. Studies indicate that HLA typing can predict response to specific antiviral regimens, allowing for personalized treatment strategies. Machine learning algorithms are being developed to analyze large datasets to predict treatment responses, side effects, and long-term outcomes in co-infected individuals.

CONCLUSION

HBV-HIV co-infection presents significant clinical and immunological challenges that require a multidisciplinary approach to treatment and management. The complex interplay between the two viruses, coupled with the immune dysfunction caused by HIV, complicates disease progression and treatment outcomes. Current therapies, while effective at suppressing viral replication, are associated with drug resistance and hepatotoxicity. Future research should focus on understanding the immune mechanisms that underlie co-infection, developing therapeutic approaches, and addressing the public health implications of co-infection.

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