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ORIGINAL

Hepatitis B Incidence in Individuals with Chronic Kidney Disorder: A Meta-Analysis

Incidencia de Hepatitis B en individuos con Enfermedad Renal Crónica: un meta-análisis

Amit Kumar Kumawat[1], Sanjiv Kumar Bansal[2], Lovely[3], Wamika Goyal[4], Gujjala Srinath[5], Nitish Vashisht[6]

[1]Parul Institute of Nursing, Faculty of Nursing. Vadodara, India.
[2]Faculty of Medicine Health Sciences, SGT University, Dept. of Biochemistry, Gurugram, India.
[3]Noida Institute of Engineering & Technology, Department of Biotechnology, Greater Noida, India.
[4]Centre of Research Impact and Outcome, Chitkara University, Rajpura, India.
[5]Anurag University, Centre for Multidisciplinary Research. Hyderabad, India.
[6]Chitkara Centre for Research and Development, Chitkara University, Chandigarh, India.

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ABSTRACT

The hepatitis B virus is a major contributor to liver disease and has many non-liver symptoms. In addition to the significant impairment and mortality that HBV causes in the population, the most recent study indicates the role that HBV plays in the initiation and development of Chronic Kidney Disease (CKD). The causal relationship between HBV and CKD has yet to be fully elucidated. Antiviral nucleos (t) medicine compounds for HBV are already available, and they are very effective for individuals with chronic kidney disease. An increased probability of CKD has been identified in those with HBV, according to a recent meta-analysis of clinical data. Based on the most recent review, we identified six separate clinical trials. There was too much variation to draw any firm conclusions. Several proposed reasons account for the increased risk of CKD in HBsAg optimistic patients. Additional data links treatment with nucleos(t)ide equivalents to a decreased risk of end-stage renal disease (ESRD) and links undiagnosed HBV to an increased risk of ESRD in the CKD population. They advise that kidney activity and urine alterations be measured at the outset and periodically in HBV participants.

Keywords: Hepatitis B Virus (HBV); Chronic Kidney Disorder (CKD); End-Stage Renal Disease (ESRD); quality of life (QOL).

RESUMEN

El virus de la hepatitis B es un importante contribuyente a la enfermedad hepática y tiene muchos síntomas no hígado. Además del deterioro significativo y la mortalidad que causa el VHB en la población, el estudio más reciente indica el papel que desempeña el VHB en el inicio y el desarrollo de la enfermedad renal crónica (ERC). La relación causal entre el VHB y la ERC aún no se ha aclarado por completo. Los compuestos de medicamentos antivirales (T) para VHB ya están disponibles, y son muy efectivos para personas con enfermedad renal crónica. Se ha identificado una mayor probabilidad de ERC en aquellos con VHB, según un metaanálisis reciente de datos clínicos. Según la revisión más reciente, identificamos seis ensayos clínicos separados. Hubo demasiada variación para sacar conclusiones firmes. Varias razones propuestas explican el mayor riesgo de ERC en pacientes optimistas de HBSAG. Datos adicionales vincula el tratamiento con los equivalentes

de Nucleos (T) IDE a un riesgo disminuido de enfermedad renal en etapa terminal (ESRD) y enlaces no diagnosticados al VHB a un mayor riesgo de ESRD en la población de ERC. Aconsejan que la actividad renal y las alteraciones de la orina se miden desde el principio y periódicamente en los participantes del VHB.

Palabras clave: Virus de la Hepatitis B (VHB); Enfermedad Renal Crónica (ERC); Enfermedad Renal en Etapa Terminal (ERET); Calidad de Vida.

INTRODUCTION

A key contributor to the development of hepatocyte carcinoma and chronic liver disease, hepatitis B virus (HBV) infection poses a substantial threat to people with Chronic Kidney Disease (CKD), particularly those undergoing surgery. Renal machinery, elevated blood-related substance use, frequent epidermal breakdowns, weakened immune systems, and elevated hepatitis B infection rates among kidney patients all raise the risk of HBV infection in patients receiving therapy.[1] Disruption within the kidneys' structure and function over time through various factors defines chronic renal failure. There are wide regional variations in the prevalence of chronic renal disease and its numerous causes.[2] Approximately twenty-four million individuals worldwide are living with chronic hepatitis B virus infection, which is an important contributor to hepatic cirrhosis as well as the sixth most prevalent malignancy internationally. The reason for this is currently unknown, but it is undoubtedly related to the gradual loss of structurally resolved circular DNA during development, the disruption of transcription from integrated viral genomes, and a persistent decline in hepatic antibody transcription.[3] Conversely attempts to eradicate hepatitis in people have concentrated on lowering mortality and disability from chronic liver disease by widespread testing, Hepatitis therapy in adolescence is limited to just three direct-acting antiviral regimens at present, despite the availability of in excess of eight equivalent regiments among people. Long-term antiviral treatment with entecavir or tenofovir to prevent virus proliferation and increased case detection.[4] Individuals, particularly dialysis users, commonly experience pruritus, an unpleasant condition linked to end-stage renal disease (ESRD) and chronic kidney disease (CKD). Even after ruling out all three possible causes, individuals who experience pruritus due to a systemic etiology directly linked to chronic kidney disease should be evaluated for chronic kidney disease-associated pruritus. These individuals' quality of life (QOL) is negatively impacted.[5] In addition to the significant impairment and mortality that HBV causes in the population, the most recent evidence supports the significance of HBV in the beginning and development of CKD. The goal of was to though hepatitis B virus (HBV) DNA levels in the blood of individuals with previous or persistent infections with HBV increase this phenomenon is referred to as hepatocellular B reactivated.[6] Therapeutic circumstances where recurrence is more inclined to happen involve those when the patient is immune-compromised or receiving restrictive treatment. Research Hepatitis B virus (HBV) infection that persists over time is dangerous for a person's fitness.[7] Antiviral therapies inhibiting HBV is used extensively to treat chronic hepatitis B. However, these medications perform not cure the infection entirely. Hepatitis B vaccination is the single effective method of reducing HBV transmission.[8]

The objective of was too the primary goal in this large-scale practical prospective research was to determine the possibility that prednisolone increased the probability of hepatitis flare in patients previously exposed to HBV. The research showed too persistent infection is the primary factor in developing chronic hepatitis B (CHB) by activating immunological responses inducing inflammation and facilitating fibrous renewal in CHB sufferers.[9] The objective also analyzed diversity, an issue that hadn't been well explored in the literature until that point.[10] The considered studies that compared and contrasted the risk of hepatocellular carcinoma in patients treated for a persistent hepatitis B infection with entecavir or tenofovir disoproxil fumarate. Research Long-term liver damage can be caused by several causes, including but not limited to alcoholism, exposure to contaminants, hepatitis, and immunological and metabolic abnormalities.[11] The goal of was that through the past two decades, advances in HBV treatment had raised a possibility that HBV DNA will be undetectable after medical treatment, moving compared to the initial treatments towards the previous generation equivalents.[12] The majority of recipients of kidney transplants who are infected with HBV have already been receiving this safe long-term therapy with matches.[13] Research the treatment of related conditions of blood pressure (BP) control decrease in precipitation by inhibition from the rennin angiotensin aldosterone structure and prevention least delaying additional injury to the kidneys are the mainstays of CKD care. The research also provided the findings of an investigation conducted on adults with type one diabetes about their vaccination status concerning viruses such as seasonal pneumonia, pneumococcal infections, and hepatitis B.[14] Research is hoped as outcomes will provide light on the prevalence of HBV and HCV infections among the kidney demographic throughout Vietnamese and how these infections are possibly affected by current infection prevention and control measures.[15]

METHOD

Preferred reporting items for systematic reviews and meta-analyses (PRISMA)

To create a graphical PRISMA diagram flow for the research on the prevalence of CKD in individuals infected with HBV, the simplified PRISMA flowchart illustrates the essential steps involved. These phases are commonly followed by the PRISMA diagram includes identification, screening, eligibility, and included studies. Figure 1 shows the PRISMA structure.

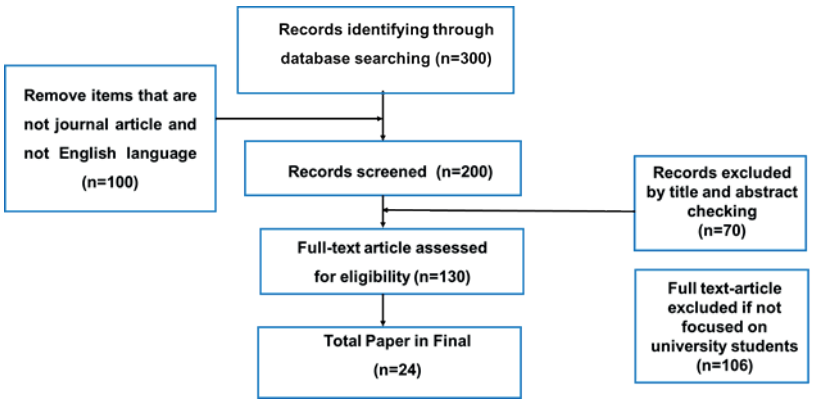

Figure 1. Framework of the PRISMA

Research design

The detection rate of HBsAg among individuals with CKD remains low. The HBV-infected individuals may develop extrahepatic manifestations, affecting both acute and chronic infections. Symptoms related to HBV include polyarteritis nodosa, renal dysfunction, and vasculitis, often presenting as combined conditions.[16]

One of the main causes of kidney disease is HBV infection, especially in CKD patients who are receiving long-term dialysis, as they are more likely to experience problems from HBV. The complex and bidirectional interactions between HBV and renal function are influenced by antiviral treatments and comorbidities in HBV-infected individuals, along with the medications commonly prescribed for them.[17]

Inclusion and Exclusion criteria

Inclusion: Research on HBV therapy effects, CKD incidence, ESRD risk, and changes in renal function, including statistically supported clinical trials or meta-analyses.

Exclusion: Case reports, animal studies, studies without a CKD focus, studies with no clinical data, or studies that concentrate on other forms of hepatitis (such HCV). Excluded from consideration is research that does not evaluate renal function.

Data Sources and the Method of Search

A systematic review of the literature was conducted according to PRISMA guidelines. We searched multiple databases, including PubMed, Scopus, and Web of Science, for studies published between 2019 and 2025. The search terms included “chronic kidney disease”, “hepatitis B virus”, “HBsAg-positive”, and “kidney dysfunction”. Studies were selected based on relevance to the incidence of CKD in HBV-infected individuals.

Outcome measure

This multi-center, cross-sectional study aimed to assess the prevalence of kidney disease among individuals with chronic HBsAg-positive infection. Over a two-year period, 300 individuals who tested positive for HBsAg but had never received oral anti-HBV therapy were prospectively enrolled. CKD was diagnosed based on the KDOQI/KDIGO criteria. The distribution of kidney function stages was as follows:

- 50 individuals were classified as having normal renal function.
- No cases of CKD stage 4 or 5 were recorded.
- 35 individuals were diagnosed with stage 2 CKD.

Risk Factors and Co-morbidities

According to statistical modeling and cohort studies, arterial hypertension, diabetes, and cirrhosis were identified as risk factors for the advancement of chronic kidney disease (CKD) in people infected with HBV. While the proportion of dialysis patients testing positive for hepatic antibodies is relatively low in developed countries, it remains higher than that of the general population in these regions. These prevalence rates are derived from numerous high-quality studies. In contrast, kidney transplant recipients in developing countries face a significantly higher risk of HBV infection. However, this conclusion is largely based on limited single-

center studies, highlighting the need for broader, multi-center research.[18] Despite the available data on HBV infection in dialysis and transplant patients, there is a notable lack of information regarding its incidence in CKD patients before initiating dialysis.

A Review of Current Research on the Association in HBV and CKD

HBV is known to negatively impact the development and progression of adult chronic kidney disease. The association is supported by the meta-analysis and a systematic evaluation of observational research. Separate meta-analyses were conducted for each outcome to ensure comprehensive evaluation. All included studies assessed the prevalence and incidence of CKD and hepatitis B virus (HBV). However, there was no significant evidence indicating that occult HBV infection elevated the risk of CKD development.

Despite these findings, several limitations must be considered:

1. Study Design Limitations: most included studies relied on cross-sectional designs, preventing causal inference. The use of prevalence ratios may have overestimated relative risks. Research methodology differences could have also influenced the findings.
2. Residual Confounding: studies with adjusted estimates were included, but potential residual confounding remained due to incomplete data on key variables. For example, HBV virus characteristics and recreational drug use were not fully accounted for in the selected studies, which may have affected the accuracy of the meta-analysis.

In observational studies, addressing residual confounding remains a critical challenge, and new strategies are being developed to mitigate these effects. However, the debate continues regarding the effectiveness of statistical methods and meta-analyses in fully accounting for these biases. This study provides a comprehensive assessment of non-randomized observational investigations. While randomized controlled trials (RCTs) help reduce confounding bias, they also have significant limitations that restrict the generalizability of findings to real-world clinical practice.[19] Therefore, all HBsAg-positive patients should undergo baseline kidney function assessments and regular urine testing as part of their follow-up care. However, routine HBV serological screening in CKD patients is not currently recommended by the K/DOQI guidelines.

Hepatitis B and Kidney Involvement: A Focus on Glomerular Diseases

This condition, though rare, is a recognized manifestation of chronic HBV infection. There is growing evidence that end-stage glomerular disease is highly associated with chronic hepatitis B virus (HBV) infection, particularly in patients who have had liver or kidney transplants. The main cause of HBV-associated glomerular illness is thought to be the buildup of immunological complexes in the glomerulus. The immune response involves viral antigens and the antibodies that neutralize them. These immune complexes may form within the glomerulus or originate from the circulatory system, becoming trapped in the glomerular structure. Depending on the size of the immune complexes and antigens, the subepithelial region is frequently the site of immune complex deposition, although mesangial and subendothelial sections can be impacted. Several studies suggest that nephropathy regression correlates with HBeAg clearance, highlighting its role in HBV-related kidney disease.[20] Proximal and distal kidney tubule epithelial cells are key sites of cell apoptosis, although retinal kidney apoptosis appears less common. Histological findings in kidney tissue samples commonly reveal membranous changes, with membranoproliferative glomerulonephritis (MPGN) and mesangial proliferation being the predominant pathological patterns associated with HBV-induced glomerular dysfunction.

Membranous Glomerulopathy (MG)

Autoimmunity and inflammation are often seen in kidney illnesses caused by HBV. These immune complexes usually have a sub epithelial location and include HBeAg and Immunoglobulin. These ICs cause glomerular injury by causing inflammation and fibrin build-up.[21] Most of those with HBV end up with MG. This relatively glomerular condition carries a decent prognosis among kids. However, it may lead to kidney failure later on in adults and, in up to 30 % of cases, lead to ESRD if ignored. Proteinuria is the predominant clinical symptom in adulthood.

Membranoproliferative glomerulonephritis (MPGN)

Regarding current developments about the complement's operations, a novel categorization of MPGN has been suggested. Patients who have MPGN caused by HBV typically have ICs containing HBsAg and HBeAg that are deposited in the mesangium and sub endothelial region.[22] The underlying mechanisms of HBV-associated MPGN are currently under investigation. Key clinical manifestations of HBV-related MPGN include glomerulonephritis and hypertension.

Mesangial proliferative glomerulonephritis (MsGN)

Hepatic antibody and IgA-containing circulation complexes of immunity are typically accumulated in the sub

endothelial and mesangial spaces. Urine abnormalities are common in those whose MsGN is due to hepatitis

Hepatitis B virus-induced cryoglobulinemic vasculitis

Cryoglobulinemic hepatitis has been related to a positive HBsAg serologic condition. The extremely small to large veins of different tissues take the brunt of the damage caused by HBV mixed cryoglobulinemia. Histologically it seems to be a leukocytoclastic vasculitis characterized by the accumulation of immunoglobulin and the vessel's exterior neutrophils. There's also the possibility of necrotic hepatitis. In individuals with HBV-induced cryoglobulinemic vasculitis, the most frequent glomerular illness is membranous proliferation the condition. Cryoglobulinemic vasculitis caused by HBV is unusual, and its therapy is unclear. A form of mixed freezing constituted the most prevalent presentation. After treatment with NUCs, no patients experienced an additional illness development, and the HBV DNA in their samples remained invisible. Five patients succumbed to cirrhosis and renal failure during antiviral therapy, despite a reduction in purpura and cryocrit levels. The prevalence of HBV and its complications has declined with widespread vaccination efforts in developed countries. Figure 2 illustrates the distribution of CKD stages among participants at cohort entry, determined using the MDRD equation.

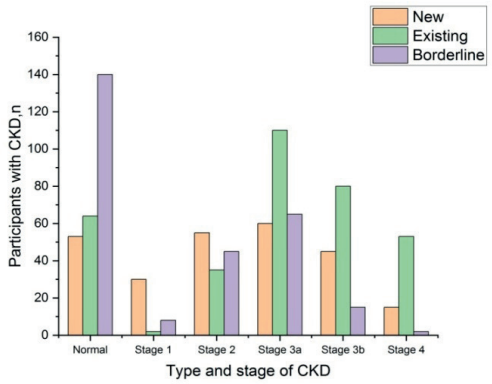

Figure 2. CKD stage at cohort entry was determined using the MDRD equation

Cryoglobulinemic vasculitis caused by HBV is declining due to the introduction of nucleus idea analogs, which are altering HBV's natural background/history. Effective care of hepatitis B and its related consequences requires routine screening and monitoring, especially in immigrant groups where chronic HBV infection is highly prevalent.

Mechanistic Insights into HBV-Associated Chronic Kidney Disease

Investigations are ongoing to determine the underlying causes of renal damage in those infected with HBV. HBV-related glomerular disease is partially characterized by the accumulation in the urinary tract of antibodies containing HBV antibodies, as mentioned above. Higher amounts of the growth factor converting growth factor-beta are frequently observed in the plasma of individuals with persistent HBV infection; this protein impacts apoptotic and renal fibrosis. Figure 3 illustrates the patient distribution across different stages of CKD.[23]

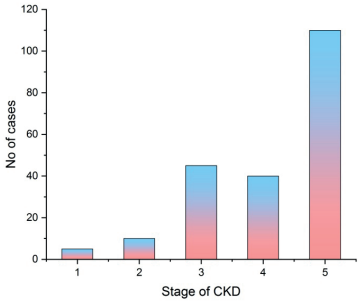

Figure 3. Graphical representation illustrating the distribution of patients across different stages of CKD

Treatment Approaches for HBV-Associated Glomerular Disease

HBV-associated kidney disease is managed using antiviral agents and immunosuppressive therapies. The two main treatments for HBV at the moment are pegylated interferon (peg-IFN) and nucleos(t)ide analogs

(NAs). However, in patients with renal impairment, NAs are preferred over peg-IFN. Due to the rarity of HBV-induced kidney disease, most treatment research has been conducted in limited, single-center studies. To gain a broader understanding, findings from meta-analyses have been reviewed. Patients receiving NA treatment were the sole subjects of one meta-analysis, whereas patients receiving both IFN and NA treatment were included in another. Three meta-analyses looked at combination treatments that included immunosuppressive medications, peg-IFN, and NAs.[24]

DISCUSSION

Compared to the non-NA group, patients receiving nucleos(t)ide analogs (NAs) had greater rates of complete remission (CR). At the moment, there are no recognized clinical recommendations for the management of HBV-associated cryoglobulinemic vasculitis. Nonetheless, those with mild to moderate cryoglobulinemic vasculitis caused by HBV are thought to be the best candidates to start antiviral treatment with NAs. They recommend plasma exchange, rituximab, and traditional immunosuppressive medications for people with severe illness. Because of the potential for HBV reactivation, concomitant treatment with NAs is advised. Due to the high risk of immunosuppressant-related reactivation of HBV, immunosuppressive drugs should be used with great caution. It is important to adjust the dosage of NAs based on how well the kidneys are working. Currently, entecavir and tenofovir are considered to be scientists demonstrated that compared to the drug disoproxil fumarate because the kidney safety rating of the infectious agent alafenamide is much improved. Nucleos(t) ide analog (NA) therapy's impact on kidney survival has not been well studied. Furthermore, propensity scores were used to CKD patients who had an HBV infection and were receiving NA treatment. Findings from Chen and colleagues indicate that uncontrolled HBV infection significantly raises the likelihood of end-stage renal disease (ESRD), while NA treatment reduces this risk.

CONCLUSIONS

Many revisions have pointed to a negative effect of HBV in each development and spread of CKD in adults. Several hypothesized processes might explain the phenomenon to be researched on the prevalence of CKD in populations outside of Asia. According to certain evidence, antiviral therapy using nucleos (t) dies analogy may lower the incidence of ESRD in CKD patients with HBV infection. Kidney function and urine alterations should be evaluated at baseline and during follow-up in HBV-infected people. Patients with CKD should undergo HBV testing and regular monitoring for renal disease development. After treatment with NUCs, no patients experienced an additional illness development, and the HBV DNA in their samples remained invisible. Five patients succumbed to cirrhosis and renal failure during antiviral therapy, despite a reduction in purpura and cryoglobulin levels. They have previously hypothesized that individuals who chronically carry HBsAg exhibit chronic systemic inflammation, and now we know that these patients also suffer from severe endothelial dysfunction. As a result, there is a higher chance of cardiac issues and persistent kidney disease.

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CONFLICT OF INTEREST

None.

AUTHORSHIP CONTRIBUTION

Conceptualization: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Data curation: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Formal analysis: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Acquisition of funds: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Research: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Methodology: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Project management: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Resources: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Software: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Supervision: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Validation: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Display: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Drafting - original draft: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.

Writing - proofreading and editing: Amit Kumar Kumawat, Sanjiv Kumar Bansal, Lovely, Wamika Goyal, Gujjala Srinath, Nitish Vashisht.