

A Cross-sectional Study to Detect Inactive Hepatitis B Carriers among Dialysis Patients in a Tertiary Care Hospital in Bangalore

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Background and study aim: Inactive Hepatitis B virus (HBV) carriers constitute a major part of Chronic Hepatitis B Carriers worldwide. This study aims at the detection of inactive HBV carriers among dialysis patients who are more susceptible to undergo latent viral reactivation and develop fulminant hepatitis.

Patients and Methods: Serum and Plasma samples were collected from each of 120 dialysis patients and subjected to HBV detection by Chemiluminescent Immunosorbent Assay (CLIA) and Polymerase Chain Reaction (PCR), respectively.

Results: Out of 120 dialysis patients, 7 (5.8%) patients were found to be positive for Hepatitis B surface antigen (HBsAg) by CLIA, but no patient (0%) was found to be positive for HBV DNA by PCR (p=0.03). Out of 7 (5.8%), HBsAg

positives, 5 (4.2%) belonged to the age group of 59-68 years and 2 (1.7%) belonged to 49-58 years (p=0.05). 4 (3.3%) were males and 3 (2.6%) were females (p=0.0001). 5 (4.2%) underwent dialysis for >50 months and 2 (1.7%) for 40-49 months (p=0.04). 4 (3.3%) underwent dialysis 3 times a week, and 3 (2.6%) 2 times a week (p=0.02). After a yearly follow-up of these 7(5.8%) patients at 2 monthly intervals, low HBsAg levels continued to be found by CLIA with no detectable HBV DNA levels by PCR. A P value <0.05 is considered statistically significant.

Conclusion: This study shows that low HBsAg levels correlate with low HBV DNA levels, which is the hallmark feature of the Inactive Hepatitis B carrier state, which may reactivate and develop into fulminant HBV infection among dialysis patients.

INTRODUCTION

Hepatitis B Virus infection is a major blood-borne infection with high morbidity and mortality. It is currently affecting 2 billion people globally, with an estimated prevalence of 3.2% and an annual incidence rate of 1.5 million new cases per year, and it is responsible for 1 million deaths worldwide annually [1].

Hepatitis B Virus is known to cause chronic disease with an estimated burden of 300 million chronic hepatitis B carriers worldwide, and it is estimated to be the cause of 30% of cirrhosis and 53% of hepatocellular carcinoma (HCC) globally [2].

Around 15-40% of Chronic Hepatitis B carriers develop cirrhosis and end-stage liver failure or HCC in their lifetime, and the cause of death is mainly attributed to chronic HBV complications rather than acute HBV infection [3, 4].

Acute HBV infection becomes chronic in about 5%-10% of the general population whereas the rate is about 60%-80% in the case of dialysis patients due to an immunosuppressed state of kidney failure, long and frequent exposure of vasculature to the external environment, multiple blood transfusions, and increased rate of nosocomial HBV transmission in various dialysis settings [5].

Approximately 2%-15 % of patients on hemodialysis get infected with chronic HBV infection [6].

Dialysis patients are at increased risk of latent HBV reactivation and development of fulminant hepatitis as they are unable to mount a proper immune response against HBV vaccination compared to the general population, and only 10%-50% achieve effective seroconversion following vaccination and are at higher risk of exposure to nosocomial HBV transmission [7, 8].

Chronic HBV infection is also known to induce kidney disease in various forms, such as membranous nephropathy, membranoproliferative glomerulonephritis, and polyarteritis nodosa. Cirrhosis is known to worsen kidney failure in around 10% of HBV-infected dialysis patients [9]. Treatment of chronic HBV infection in dialysis patients is also challenging, as most anti-viral drugs require a dose adjustment and have more toxic side effects compared to the general population [10].

Inactive hepatitis B carriers form a major part of chronic HBV carriers (36%), which are defined by the presence of Hepatitis B surface antigen (HBsAg), absence of hepatitis B envelope antigen (HBeAg), undetectable levels of HBV DNA and normal liver histology and other parameters [11, 12]. Though they have a low rate of seroconversion and excellent prognosis among the general population, they are known to undergo reactivation and develop into fulminant hepatitis and HCC [13] among dialysis patients due to various conditions such as spontaneous reactivation of latent HBV infection due to induced immunosuppression [14], advanced age, male gender, uremia and use of long-term steroids [15, 16].

Though the inactive HBV carriers remain latent for a longer period, the risk of HBV reactivation cannot be ruled out, as the HBV tends to remain silent or latent in hepatocytes for years owing to the unavailability of eradication therapy for HBV infection at present [17].

An inactive hepatitis B carrier state is known to undergo spontaneous reactivation in patients who are immunocompromised due to various conditions such as acquired immunodeficiency syndrome (AIDS), malnutrition, chronic kidney disease, chronic autoimmune disorders such as Rheumatoid Arthritis, Systemic lupus

erythematosus, receiving immunosuppressive therapies such as cancer chemotherapy, radiotherapy, high dose corticosteroids for a longer duration [18, 19].

Inactive HBV carriers have low HBsAg levels and undetectable HBV DNA levels because most Polymerase chain Reaction (PCR) Nucleic acid extraction and detection kits have a limit of detection between 250-1000 copies/ml, and HBV DNA levels may not be sufficient for detection by these kits. But low HBsAg positivity and HBV DNA negative reports get interpreted as false positive HBsAg reaction or past exposure, which may hold good in the general population, but results in hazardous complications in dialysis patients, as there are chances of reactivation of latent HBV infection among them [20]. This study aims to detect inactive Hepatitis B carriers among dialysis patients using serological and molecular methods and to follow them up at regular intervals for 1 year to rule out false positives, monitor HBsAg and HBV DNA levels, and prevent reactivation.

PATIENTS AND METHODS

Study design and source of data - This cross-sectional study was conducted in the department of microbiology of a tertiary care hospital in Bangalore, Karnataka, for a period of 1 year from January 2021 to December 2021.

120 patients on dialysis are selected for sample collection after applying the inclusion and exclusion criteria as follows-

Inclusion criteria are 1) Dialysis patients above 18 years of age who have given verbal consent. 2) Dialysis patients not currently infected with Hepatitis A, C, D, and E viruses. 3) Dialysis patients who are not on antiviral drugs at present.

Exclusion criteria are 1) Dialysis patients who are co-infected with Human Immunodeficiency Virus (HIV) and 2) Dialysis patients with underlying primary diseases such as Autoimmune diseases, Coronary Artery Disease, Asthma, and Diabetes Mellitus.

Sample size estimation - the prevalence of Hepatitis B among renal dialysis patients was reported to be 22% [21]. Assuming similar proportions in our study with an absolute precision of 7.5% and the desired confidence

interval of 95% (Alpha error -5%), the required sample size is 117 patients.

Sample collection- The required minimum number of participants was calculated to be 117. However, the present study encompasses 120 dialysis patients. About 4 mL of blood is collected from each of the 120 patients in yellow and purple top vacutainers and centrifuged at the rate of 4000 rotations per minute to separate serum and plasma, respectively. Serum samples were tested immediately in CLIA, and Plasma samples were transported to the PCR lab at 2-8°C for immediate DNA extraction.

HBsAg detection - the VITROS HBsAg test is performed using the VITROS HBsAg reagent pack and VITROS Immunodiagnostic Products, HBsAg Calibrator on the VITROS ECIQ Immunodiagnostic systems, and the VITROS 3600 Immunodiagnostic system. An immunometric immunoassay technique is used, which involves a simultaneous reaction of HBsAg in the sample with mouse monoclonal anti-HBs antibody coated onto the wells and a horseradish peroxidase-labeled mouse monoclonal anti-HBs antibody in the conjugate. The bound HRP conjugate is estimated by a luminescent reaction. The amount of bound HRP conjugate is indicative of the level of HBsAg present in the sample. HBsAg level values were determined according to kit literature. If the value is <0.9 (<900 IU/ml), the serum sample is negative. If the value is between 0.9-0.99 (900-999 IU/ml), the serum sample is intermediate/low reactive and tested using a fresh sample from the patient and tested reflex by RT PCR, if the value is ≥ 1 (≥ 1000 IU/ml), then serum is considered positive for HBV infection. The tested samples were stored at -20 °C for further reference.

HBV DNA detection is done by the PCR method using the THERMO-FISHER Quant Studio 3 Real-time PCR machine, where

The extraction of plasma samples was carried out by the BIOBEE Viral DNA extraction kit and the extracted plasma samples were tested for the HBV DNA using the NEODX HBV DNA qualitative RT PCR detection kit and the master mix-template reaction mixture prepared using NEODX kit was loaded into the THERMO-FISHER Quant Studio 3 Real-time PCR machine where it undergoes denaturation, extension, and annealing in thermal cycles to amplify the HBV

DNA if present in the sample and to analyze the final result and its validity.

Steps include- 1) Nucleic acid extraction- HBV DNA extraction from plasma samples was done immediately using a BIOBEE viral DNA extraction kit. 250 microliters of plasma are mixed with 250 microliters of lysis buffer, 5 microliters of carrier RNA, and 10 microliters of Proteinase in a sterile microcentrifuge tube and incubated at room temperature for 5 minutes. The tube is then centrifuged at 8000 rotations per minute (rpm) for 5 minutes to get the lysate. 400 microliters of this lysate are mixed with 400 microliters of binding buffer in a new 1.5 ml microcentrifuge tube and then centrifuged at 8000 rpm for 30 seconds. After discarding the flow-through, 450 microliters of wash buffer 1 are added and centrifuged at 8000 rpm for 30 seconds. The process is repeated with wash buffer 2 also. After discarding the flow through, the spin column is air-dried at 8000 rpm for 3 minutes. Then the spin column is shifted to a new 1.5 ml microcentrifuge tube, and 50 microliters of elution buffer is added and centrifuged at 8000 rpm for 1 minute. The lysate collected in the microcentrifuge tube will have viral DNA if present. The extracted samples (templates) were stored at -80 °C. HBV DNA detection from those templates is done using the NEODX HBV DNA qualitative RT PCR detection kit. 2) Master Mix preparation (120 reactions) (Tables 1 and 2) - Thaw contents of the kit at room temperature till equilibrated and the reaction mix is prepared for 100 and 20 reactions as below. 3) Template addition- 15 microliters of reaction mix are prepared for each of 120 PCR reaction tubes with 1 tube each for negative and positive controls to which 10 microliters each of sample, negative and positive control is added to respective tubes and centrifuged and transferred to PCR room where tubes are placed in the sample holder and programmed as below (Table 3) and the result is generated and data is interpreted for each run (Table 4 and 5). Follow-up – All 120 participants of the study were followed up regularly for a year at regular intervals of 2 months to monitor HBsAg and HBV DNA levels by CLIA and PCR, respectively. This follow-up was necessary to assess latent HBV reactivation among the dialysis participants. But the follow-up was solely based on HBsAg and HBV DNA parameters and other serological markers of

Hepatitis B infection such as HBeAg (Hepatitis B pre-core antigen), anti-HBs (Hepatitis B antibody) and biochemical tests such as LFT (Liver function test), serum albumin to globulin ratio among others were not performed as patients did not have any clinical features of Hepatitis B infection and also due to financial constraints of the study.

Statistical method- Descriptive statistics of the HBV positives were analyzed and summarized in terms of percentage in Microsoft Excel. Statistical significance between continuous variables was tested using the Chi-squared test, and categorical variables were tested using the t-test. Statistical analysis was done using Statistical Package for the Social Sciences for Windows (version 21.0; Armonk, NY: IBM Corp., USA). A P value of <0.05 was considered statistically significant.

RESULTS

Out of 120 dialysis patients, 7 (5.8%) patients are found to be positive for HBsAg by CLIA, and no patient is found to be positive for HBV DNA by PCR (P value = 0.03). Out of 7 (5.8%) HBsAg-

positive patients, 5(4.2%) belonged to the age group of 59-68 years, and 2(1.7%) belonged to the age group of 49-58 years (P value = 0.05). Out of 7 (5.8%) positives, 4(3.3%) are males and 3(2.6%) are females (P value = 0.0001). Out of 7 (5.8%) positives, 5 (4.2%) underwent dialysis for >50 months, and 2 (1.7%) underwent dialysis for 40-49 months (P value = 0.04). Out of 7 (5.8%) positives, 4 (3.3%) underwent dialysis 3 times per week, and 3 (2.6%) 2 times a week (P value=0.02). [Table 6]. These patients are followed up for 1 year at regular intervals of 2 months to monitor HBsAg and HBV DNA levels and to rule out any possibility of latent viral reactivation. Over the past year, the HBsAg levels remained low reactive (<1000 IU/ml) in all participants with HBV DNA levels under the detection limit by PCR (<2000 IU/ml).

Prevalence of Inactive Hepatitis B carriers = $\frac{\text{Number of cases (patients with positive HBsAg and negative HBV DNA)}}{\text{study population}} \times 100$
 $\frac{7}{120} \times 100 = 5.8\%$.

Table 1: Master Mix preparation for 100 reactions (1st batch)

Reagents	For 100 reactions
2x master mix	1500 microliters
20x primer and probe mix	150 microliters
Nuclease-free dH2O	350 microliters
Total	2000 microliter

Table 2: Master Mix preparation for 20 reactions

Reagents	For 20 reactions
2x master mix	300 microliters
20x primer and probe mix	30 microliters
Nuclease-free dH2O	70 microliters
Total	400 microliters

Table 3: Steps of Programming the PCR Instrument

Sl no	Step	Temperature (°C)	Time	Cycle
1	Reverse Transcription	50	15 min	1
2	cDNA initial denaturation	95	2 min	1
3	Denaturation	95	15 sec	45
4	Annealing, extension, and fluorescence measurement	58	30 sec	

Min – minutes

Sec - seconds

Table 4: Result interpretation of HBV

For HBV	For the Internal control gene	Assay result
Ct <40	Ct =40	Positive
Ct >=40	Ct =40	Negative
Ct >=40	Ct >=40	Invalid

Ct – Cycle threshold

Invalid – Repeat extraction and detection

Table 5: Data interpretation for HBV

HCV (FAM)	IC (TEXAS RED/ROX)	Result
+	+	HBV detected
-	+	HBV not detected
-	-	Invalid (repeat test)

FAM, Texas Red/Rox – genes tested

IC – Internal control

Table 6: Baseline demographic characteristics of Participants

Characteristics (n=120)	Positives	Negatives	P value
Age in years			
19-28	0	6 (5.0%)	0.05
29-38	0	10 (8.3%)	
39-48	0	10 (8.3%)	
49-58	2 (1.7%)	20 (16.6%)	
59-68	5 (4.2%)	67 (55.8%)	
>70	0	0	
Gender			
Males	4 (3.3%)	57 (47.5%)	0.0001
Females	3 (2.6%)	56 (46.6%)	
Duration of dialysis (months)			
<10	0	0	0.04
10-19	0	0	
20-29	0	11 (9.2%)	
30-39	0	10 (8.3%)	
40-49	2 (1.7%)	42 (35%)	

>50	5 (4.2%)	50 (41.6%)	
Frequency of dialysis			
3	4 (3.3%)	48 (40%)	0.02
2	3 (2.6%)	65 (54.1%)	
HBV parameters			
HBsAg (CLIA)	7 (5.8%)	113 (94.1%)	0.03
HBV DNA (PCR)	0 (0%)	120 (100%)	

Age (years), Duration of dialysis (months), Frequency of dialysis (number of dialysis sessions/week), HBsAg – Hepatitis B surface antigen, CLIA – Chemiluminescence immunosorbent assay, HBV DNA – Hepatitis B virus Deoxyribonucleic acid, PCR – Polymerase Chain Reaction.

DISCUSSION

The seroprevalence of Hepatitis B Virus infection among dialysis patients in India is estimated to be between 3.4% and 43% [22]. In the present study, HBsAg positivity among dialysis patients is found to be 5.8% by CLIA. This study is in correlation with the prevalence data across various hemodialysis settings in India. Similarly, a study conducted by Ridha H Alkahlifah et al. showed the prevalence of HBV infection among dialysis patients to be 3.77% [23].

In this study, HBsAg positivity is seen mostly among the 50-60 years age group ($p=0.05$), in males than in females ($p=0.0001$), who underwent dialysis for a longer duration of >50 months ($p=0.04$) and who received dialysis more frequently at a rate of 3 times per week ($p=0.02$). Similar results were found in the study conducted by Dimple Raina et al. Where prevalence of HBV among dialysis patients was found to be 11.66%, with a higher prevalence among patients aged above 60 years of age, mostly in males and in those who received dialysis for more than 50 months with thrice weekly frequency [24]. Another study conducted

by Rani P et al. also showed an HBV prevalence of 11% among dialysis patients with longer duration of dialysis, multiple blood transfusions, and improper vaccination as serious risk factors [25].

However, in this study, the HBV DNA positivity among dialysis patients is found to be nil by RT PCR, as the limit of detection of the NEODX RT-PCR HBV DNA detection kit used in this study is 400 copies/ml. This contrasts with the study conducted by Elham Samadi et al., where the molecular prevalence of active HBV infection among dialysis patients was found to be 6%, of which 2.1% were vaccinated against HBV [26]. A study conducted by M R Ibrahim et al. also showed RT PCR confirmed active HBV prevalence of 3.2% among dialysis patients [27]. However, there are only a limited number of studies conducted on Inactive hepatitis B carriers and their long-term prognosis.

The findings of the present study are concordant with the study conducted by Willem P Brouwer et al. Where the Inactive HBV carrier rate was found to be 15% with repeated HBsAg positivity in the absence of HBV DNA by PCR for a long period of 8 years [28].

The HBsAg-positive HBV DNA-negative patients in the present study were followed up for 1 year at regular intervals of 2 months to check for the rise in levels of HBsAg and HBV DNA to rule out latent HBV reactivation. After following up for a year, these patients were found to maintain low reactive HBsAg levels (<1000 IU/ml) and undetectable HBV DNA levels (<2000 IU/ml). A study conducted in 2016 by Jessica Liu et al., on 1529 chronic HBV-infected patients confirmed that baseline measurement of HBsAg levels < 1000 IU/ml and HBV DNA levels <2000 IU/ml could be considered as low reactive levels and used to distinguish Inactive hepatitis B carriers from those with active Chronic HBV infection [29]. Another study conducted by Jun Cheng et al. showed that low HBsAg levels correlated with low levels of HBV DNA replication and were found most in older individuals [30]. Therefore, these patients were diagnosed as inactive HBV carriers. Inactive HBV carrier states may undergo latent HBV reactivation in dialysis patients on high-dose corticosteroids and cause fulminant hepatitis [31].

The incidence of HBV reactivation among dialysis patients is estimated to be 2% and is more commonly seen in older age, male gender, and longer duration with increased frequency of dialysis, multiple blood transfusions, past HBV exposure, and mainly in those on immunosuppressant therapy [31]. A study conducted by Gi-Ae Kim et al. showed that low HBsAg levels after 50 years of age act as an independent predictor of HCC and recommended maintenance of HCC surveillance in patients above 50 years of age, irrespective of whether they are cirrhotic or not [32]. Another study conducted by Huang X et al. also showed that patients with positive HBsAg and negative HBV DNA were also at greater risk of developing cirrhosis and HCC [33]. The present study did not show any latent HBV reactivation among inactive HBV carrier patients on dialysis for one year. A limitation of the study is that this study is conducted with a limited number of samples for a short period of 1 year. This study also could not afford testing for other serological and biochemical parameters of HBV infection due to financial constraints. Though this study could not detect HBV reactivation among Inactive carriers, this data is not sufficient to conclude that Inactive HBV carriers among dialysis patients

are safe from latent HBV reactivation as there are studies that have demonstrated progression of chronic HBV-infected patients with positive HBsAg and low levels or negative HBV DNA into decompensated liver cirrhosis and related complications [33]. But it is also important to note that there are not many studies conducted on Inactive HBV Carriers. Therefore, this study recommends conducting large multicentric observational studies with large sample sizes for a longer duration of time to study in detail the impact of the Inactive HBV carrier state among dialysis patients who are more vulnerable to developing HBV-related liver complications than the general population.

CONCLUSION

This study shows that the seroprevalence of the Hepatitis B virus is more commonly seen among older individuals above 50 years, especially males who are on long-term dialysis for >50 months, with thrice weekly exposure. This study fails to demonstrate HBV DNA among HBsAg-positive dialysis patients, as the limit of detection of the NEODX RT-PCR HBV DNA detection kit used is 400 copies/ml.

Though this study fails to demonstrate latent HBV reactivation among dialysis patients owing to the small sample size and short study period, it underscores the importance of regular monitoring of HBV markers such as HBsAg and HBV DNA especially in low HBsAg positive dialysis patients because there is risk of HBV reactivation among those dialysis patients as they are subjected to induced immunosuppression and increased nosocomial HBV infection which provides a conducive environment for HBV reactivation in longer run.

Conflict of Interest -The authors declare that there is no conflict of interest.

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Author's contributions – MG*: Conceptualization, methodology, Investigation, Writing – Original draft, data curation, formal analysis, visualization. AGP: Review and editing, supervision, and resource acquisition. All authors read and approved the final manuscript.

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Ethical Clearance – The study was approved by the ethics committee of M S Ramaiah Medical College in January 2021. The ethical clearance certificate number is MSRMC/EC/PG-10/01-2021. Informed consent was obtained from all the patients in the study

HIGHLIGHTS

- This article highlights a considerably higher prevalence of inactive Hepatitis B carriers among dialysis patients.
- Regular monitoring of these dialysis patients for Hepatitis B surface antigen by CLIA and HBV DNA by PCR is recommended as these patients are at a high risk of undergoing latent reactivation for HBV infection, owing to their decline in immunity.
- This article also shows a higher prevalence of inactive Hepatitis B carrier state among male gender, older age with long duration of dialysis, and increased frequency of dialysis.

REFERENCES

1. Devin RS, Ivane G, Calvin P, Thomas B, Richard G, Young-Suk Lim, et al.: Polaris Observatory Collaborators. The global prevalence, a cascade of care, and prophylaxis coverage of hepatitis B in 2022: a modeling study. *Lancet Gastroenterol Hepatol*. 2023, 8:879-907 .
2. Niu J, Jiang J, Ye S.: Liver Cancer Group, Chinese Society of Hepatology. Expert consensus on antiviral treatment of HBV/HCV-related hepatocellular carcinoma (2021 update) [J]. *Chinese Journal of Hepatology*. 2021, 29:948-966 .
3. Lin J, Wu JF, Zhang Q, Hong Wei Zhang, Guang Wen Cao.: Virus-related liver cirrhosis: Molecular basis and therapeutic options. *World J Gastroenterol*. 2014, 20:6457-6469 .
4. Lok AS. Chronic hepatitis B. *N Engl J Med*. 2002, 30:1682-3 .
5. Sit D, Esen B, Atay AE, Hasan Is hemodialysis a reason for unresponsiveness to hepatitis B vaccine? Hepatitis B virus and dialysis therapy. *World J Hepatol*. 2015, 7:761-768 .
6. Miller E.R., Alter M.J., Tokars J.I: Protective effect of hepatitis B vaccine in chronic hemodialysis patients. *Am. J. Kidney Dis*. 1999, 33:356-360 .
7. Grzegorzewska A.E: Hepatitis B vaccination in chronic kidney disease: Review of evidence in non-dialyzed patients. *Hepat. Mon*. 2012, 12:7359 .
8. Fabrizi F., Messa P., and Martin P: Hepatitis B virus infection and the dialysis patient. *Seminars in Dialysis*. 5:2-2 .
9. Bhimma R, Coovadia HM: Hepatitis B virus-associated nephropathy. *Am J Nephrol*. 2004, 24:198-211 .
10. Sharma P, Sawtell R, Wang Q, Meghan E Sise.: Management of Hepatitis C Virus and Hepatitis B Virus Infection in the Setting of Kidney Disease. *Adv Kidney Dis Health*. 2023, 30:343-355 .
11. Lok AS, Heathcote EJ, Hoofnagle JH: Management of hepatitis B: 2000 - summary of a workshop. *Gastroenterology*. 2001, 120:1828-1853 .
12. Hui Z: Correction note on the estimated number of patients in the immune-tolerant phase of hepatitis b virus infection in China and globally. *J Clin Hepatol* 785-6 .
13. Hsu YS, Chien RN, Yeh CT, Hung Yi Chiou, Chia-Ming Chu, Yun Fan Liaw.: Long-term outcome after spontaneous HBeAg seroconversion in patients with chronic hepatitis B. *Hepatology*. 2002, 35:1522-1527 .
14. Chen JD, Yang HI, Iloeje UH, San Lin You, Sheng Nan Lu, Li Yu Wang.: Risk Evaluation of Viral Load Elevation and Associated Liver Disease/Cancer in HBV (REVEAL-HBV) Study Group. Carriers of inactive hepatitis B virus are still at risk for hepatocellular carcinoma and liver-related death. *Gastroenterology*. 2010, 138:1747-54 .
15. Pita I, Horta-Vale AM, Cardoso H, Guilherme M.: Inactive hepatitis B carriers: an overlooked population. *GE Port J Gastroenterol*. 2014, 21:241-249 .
16. Oh GJ, Waldo A, Paez-Cruz F, Patrick P G, Anne Pasenson, David TS et al Steroid-Associated Side Effects in Patients With Primary Proteinuric Kidney Disease. *Kidney Int Rep*. 2019, 9:1608-1616 .
17. Fanning GC, Zoulim F, Hou J, Antonio Bertoletti: Therapeutic strategies for hepatitis B virus infection: towards a cure. *Nat Rev Drug Discov*. 18:827-844 .
18. Dropulic LK, Lederman HM: Overview of Infections in the Immunocompromised Host. *Microbiol Spectr*. 2016, 4 .

19. Reddy KR, Kimberly LB, Sarah P, Joseph K Lim, Yngve T Falkk- Ytter.: American Gastroenterological Association Institute Guideline on the Prevention and Treatment of Hepatitis B Virus Reactivation. During Immunosuppressive Drug Therapy, *Gastroenterology*, Volume. 148:215-219 .
20. Chan HL, Wong VW, Wong GL, Chi hang Tse, Hoi Yun Chan, Joseph Jao-Yao Sung: A longitudinal study on the natural history of serum hepatitis B surface antigen changes in chronic hepatitis B. *Hepatology*. 2010, 52:1232-1241 .
21. Perumal A, Ratnam, Philip VJ, Nair Shashikala, Anitha Patricia, Illangovan Veerappan, et al.: Seroprevalence of hepatitis B and C in patients on hemodialysis and their antibody response to hepatitis B vaccination. *Journal of Current Research in Scientific Medicine* 2(1): p 20-23, Jan-Jun. 2016 .
22. Duong MC, Nguyen VTT, Otsu S, Mary LM.: Prevalence of hepatitis B and C virus infections in hemodialysis patients in Vietnam: A systematic review and meta-analysis. *JGH Open*. 2019, 17:29-38 .
23. Alkhalifah RH, Alhaddad MJ, Alhashem AT, Hussain A, Abdullah AA, Abdullah AK .:Prevalence of Hepatitis B Virus, Hepatitis C Virus, and HIV Infections in Hemodialysis Patients at Kano Kidney Center. *Cureus*. 2023, 12:41769 .
24. Raina D, Rawat N, Pandita AK: Prevalence of Hepatitis B and Hepatitis C in Patients undergoing hemodialysis at a teaching hospital in Uttarakhand. *J Family Med Prim Care*. 2022, 11:1348-1353 .
25. Rani P, Bilolihar AK, Reddy S: Seroprevalence of hepatitis B virus and hepatitis C virus; its incidence and risk factors in incident chronic kidney disease (CKD) stage - V patients on hemodialysis in a tertiary care center. *J Med Sci Res*. 2018, 6:37-43 .
26. Samadi E, Mirshahabi H, Motamed N, Hamid S.: Prevalence of Occult Hepatitis B Virus Infection in Hemodialysis Patients Using Nested PCR. *rbmb.net*. 2020, 9:82-88 .
27. M.R. Ibrahim N, Sidiq Mohammed Saleem Z, R Hussein N: The Prevalence of HIV, HCV, and HBV Among Hemodialysis Patients Attending Duhok Hemodialysis Center. *Int J Infect*. 2018, 5 (1.)
28. Brouwer WP, Chan HL, Brunetto MR, Michelle MP, Pauline A, Marcus C, et al.: Good Practice in using HBsAg in Chronic Hepatitis B Study Group (GPs-CHB Study Group). Repeated Measurements of Hepatitis B Surface Antigen Identify Carriers of Inactive HBV During Long-term Follow-up. *Clin Gastroenterol Hepatol*. 2016, 14:1481-1489 .
29. Liu J, Yang HI, Lee MH, Jen CL, Batrla-Utermann R, Lu SN, et al., Serum Levels of Hepatitis B Surface Antigen and DNA Can Predict Inactive Carriers With Low Risk of Disease Progression. *Hepatology*. 2016 Aug;64(2):381-9.
30. Cheng J, Dai Y, Yan L, Zhou H, Xu X, Sun C, Wang Z. Clinical Characteristics and Correlation Analysis of Subjects with Chronic Hepatitis B Virus (HBV) Infection and Sustained Low Levels of Hepatitis B Surface Antigen (HBsAg). *Med Sci Monit*. 2018 Mar 29;24:1826-1835 .
31. Catarina Menga b, Carolina Belinoc, Luciano Pereira b, Ana P, Susana S, Isabel T, et al.: Reactivation of Hepatitis B virus in kidney transplant recipients with previous clinically resolved infection: A single-center experience. *nefrologia* 2018;38(5):545-550 .
32. Kim GA, Lee HC, Kim MJ, Ha Y, Park EJ, An J, Lee D, Shim JH, Kim KM, Lim YS. Incidence of hepatocellular carcinoma after HBsAg seroclearance in chronic hepatitis B patients: a need for surveillance. *J Hepatol*. 2015 May;62(5):1092-9 .
33. Huang X, Yan M, Deng Z, Lei Yao, Dan Han, Lihua Sun: Natural history of decompensated cirrhosis with serum hepatitis B DNA < 2000 IU/mL: a retrospective study. *BMC Gastroenterol* 22. 452.

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