



# Hepatitis B Reactivation and Antiviral Prophylaxis in Patients on Immunosuppressive Therapy

İmmünoşüpresif Tedavi Alan Hastalarda Hepatit B Reaktivasyonu ve Antiviral Profilaksi

Yakup Gezer, Arzu Tarakçı

University of Health Sciences Türkiye, Konya City Hospital, Clinic of Infectious Diseases and Clinical Microbiology, Konya, Türkiye

## ABSTRACT

**Objectives:** Hepatitis B virus reactivation (HBVr) may occur in patients receiving immunosuppressive therapy. The risk of HBVr varies depending on the immunosuppressive agent used and hepatitis serology. This study aimed to evaluate HBVr among immunosuppressed patients with and without antiviral prophylaxis.

**Materials and Methods:** HB surface antigen (HBsAg)-positive and/or HB core antibody-positive patients receiving immunosuppressive therapy were retrospectively evaluated at a single-center, tertiary-care hospital.

**Results:** A total of 224 patients were initially screened, and 153 were included in the study. The median age was 62 years (range, 52.5-72), and 50.3% were female. The rate of HBsAg positivity was 21.6%, while HB surface antibody positivity was detected in 52.3% of patients. Antiviral prophylaxis was administered to 81.7% of patients: entecavir (75.2%), tenofovir disoproxil fumarate (TDF) (19.2%), and tenofovir alafenamide (TAF) (5.6%). HBVr was not observed in patients receiving antiviral prophylaxis, whereas two cases occurred in patients not receiving prophylaxis ( $p=0.033$ ). One of these patients was receiving rituximab-based therapy, and the other was on corticosteroid treatment. When patients were stratified by risk group, rates of HBVr among patients who did not receive prophylaxis were 50% in the high-risk group, 25% in the moderate-risk group, and 0% in the low-risk group.

**Conclusion:** HBVr may occur in immunosuppressed patients. In these patient groups, hepatitis serologic testing should be performed, and antiviral prophylaxis should be administered according to the immunosuppressive regimen. Entecavir, TDF, and TAF appear to be both effective and safe. Patients without antiviral prophylaxis should be closely monitored.

**Keywords:** Anti-HBc-positive, antiviral prophylaxis, corticosteroids, HBsAg positive, immunosuppressive therapy, rituximab

## ÖZ

**Amaç:** Hepatit B virüs reaktivasyonu (HBVr), immünoşüpresif tedavi gören hastalarda ortaya çıkabilir. HBVr riski, kullanılan immünoşüpresif ajana ve hepatit serolojisine bağlı olarak değişir. Bu çalışmanın amacı, antiviral profilaksi alan ve almayan immünoşüpre hastalarda HBVr'yi değerlendirmektir.

**Gereç ve Yöntemler:** HB virüsünün yüzey antijeni (HBsAg) pozitif ve/veya HB çekirdek antikor pozitif hastalar, retrospektif olarak tek merkezli, üçüncü basamak bir hastanede incelendi.

**Bulgular:** Başlangıçta 224 hasta tarandı ve 153 hasta çalışmaya dahil edildi. Hastaların medyan yaşı 62 (52,5-72) yıl ve %50,3'ü kadındı. HBsAg pozitifliği %21,6 ve HB yüzey antikor pozitifliği %52,3 idi. Antiviral profilaksi hastaların %81,7'sine başlandı; kullanılan ilaçlar entekavir (%75,2), tenofovir disoproksil fumarat (TDF) (%19,2) ve tenofovir alafenamid (TAF) (%5,6) idi. Profilaksi alan grupta HBVr gözlenmezken, profilaksi almayan iki hastada HBVr saptandı ( $p=0,033$ ). Bu hastalardan biri rituksimab bazlı tedavi, diğeri ise kortikosteroid alıyordu. Risk gruplarına göre sınıflandırıldığında, profilaksi almayan hastalarda HBVr oranı yüksek risk grubunda %50, orta risk grubunda %25 iken, düşük risk grubunda gözlemlenmedi.

**Sonuç:** HBVr immünoşüpre hastalarda görülebilmektedir. Bu hasta gruplarında hepatit serolojisi taranmalı ve immünoşüpresif rejime göre uygun antiviral profilaksi uygulanmalıdır. Entekavir, TDF ve TAF etkili ve güvenli seçenekler olarak görünmektedir. Antiviral profilaksi almayan hastalar takip sırasında yakından izlenmelidir.

**Anahtar Kelimeler:** Anti-HBc pozitif, antiviral profilaksi, kortikosteroidler, HBsAg pozitif, immünoşüpresif tedavi, rituksimab

**Address for Correspondence:** Yakup Gezer, MD, University of Health Sciences Türkiye, Konya City Hospital, Clinic of Infectious Diseases and Clinical Microbiology, Konya, Türkiye

**E-mail:** dryakupgezer@gmail.com **ORCID ID:** orcid.org/0000-0002-1582-7313

**Received:** 08.10.2025 **Accepted:** 12.01.2026 **Publication Date:** 26.01.2026

**Cite this article as:** Gezer Y, Tarakçı A. Hepatitis B reactivation and antiviral prophylaxis in patients on immunosuppressive therapy. Viral Hepat J. 2025;31(3):91-96



Copyright© 2025 The Author(s). Published by Galenos Publishing House on behalf of the Viral Hepatitis Society.  
This is an open access article under the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 (CC BY-NC-ND) International License.

## Introduction

Hepatitis B virus reactivation (HBVr) is a complication that can develop in patients receiving immunosuppressive therapy for autoimmune or rheumatologic diseases, or chemotherapy for cancer. People who have been previously exposed to HBV are at risk of developing this complication. Although it depends on the immunosuppressive agent used, this risk is higher in people with HB surface antigen (HBsAg)-positive/HB core antibody (anti-HBc)-positive serology than in those with HBsAg-negative/anti-HBc-positive serology. The clinical presentation of HBVr can range from asymptomatic infection to liver failure (1,2). In people with prior HBV infection, the cccDNA of HBV remains latent in hepatocytes, and when immunity is reduced by various immunosuppressive drugs, reactivation of HBV can occur (3,4). There is still no standardized approach for the prevention of HBVr. For this reason, different groupings have been made to determine the risk of HBVr, and it has been suggested that the decision regarding antiviral prophylaxis should be made according to these groupings. The risk of HBVr is classified as high if it is greater than 10%, moderate if it is between 1% and 10%, and low if it is less than 1%. Antiviral prophylaxis is recommended to be initiated two weeks before the start of immunosuppressive therapy and discontinued 6 to 12 months after the end of immunosuppressive therapy. The immunosuppressive agents responsible for HBVr are mainly cytotoxic chemotherapeutics, B-cell suppressors, anti-tumor necrosis factor (TNF) agents, immune checkpoint inhibitors, tyrosine kinase inhibitors, and corticosteroids. New targeted biologic agents are introduced daily, and the effects of many of these agents on HBVr are not fully understood (5,6,7,8). The aim of this study was to evaluate the presence of HBVr in patients receiving immunosuppressive therapy.

## Materials and Methods

We retrospectively collected and analyzed the medical records of patients for whom the infectious diseases department was consulted for evaluation of HBV prophylaxis at a tertiary care hospital between January 2021 and March 2024. Patients from different departments with hepatitis serology who were scheduled for immunosuppressive treatment for primary diseases were evaluated.

In our institution, the departments of hematology, oncology, rheumatology, gastroenterology, and neurology routinely request HBV screening prior to initiating immunosuppressive therapy. At baseline, HBV serology [HBsAg, HB surface antibody (anti-HBs), and anti-HBc] is assessed in all patients. Patients with HBsAg positivity or isolated anti-HBc positivity are referred for an infectious diseases consultation to guide antiviral prophylaxis and follow-up care planning. According to the available medical records, patients underwent liver function testing, including alanine aminotransferase (ALT) and aspartate aminotransferase (AST), approximately every three months to monitor for signs of active hepatitis. In cases of elevated liver enzyme levels, HBsAg and HBV-DNA levels were subsequently evaluated.

Patients aged 18 years and older were included in the study. Patients on antiviral therapy for chronic HBV and those with

insufficient documentation were excluded. Age, sex, primary disease, immunosuppressive therapy, HBV serology, AST, ALT, HBV-DNA, and antiviral agents initiated for prophylaxis were recorded. Patients were categorized as being at high, moderate, or low-risk of HBVr according to guideline recommendations (9). HBVr was defined as either the de novo detectability of HBV-DNA in patients with previously undetectable levels or a  $\geq 10$ -fold increase in HBV-DNA from baseline values (9).

## Statistical Analysis

Statistical analyses were performed with SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The Shapiro-Wilk test was used to assess the distribution of continuous variables. The median and interquartile range (IQR) (IQR: 25<sup>th</sup>-75<sup>th</sup> percentile) are reported for continuous variables that are not normally distributed, and categorical variables are presented as frequencies and percentages. Categorical variables were compared using the chi-square test. Fisher's exact test was employed when the expected cell counts were fewer than five. A p-value of  $<0.05$  was considered statistically significant.

## Ethics

This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from KTO-Karatay University Non-Drug and Non-Medical Device Research Ethics Committee (approval no: 2024/013, date: 07.06.2024).

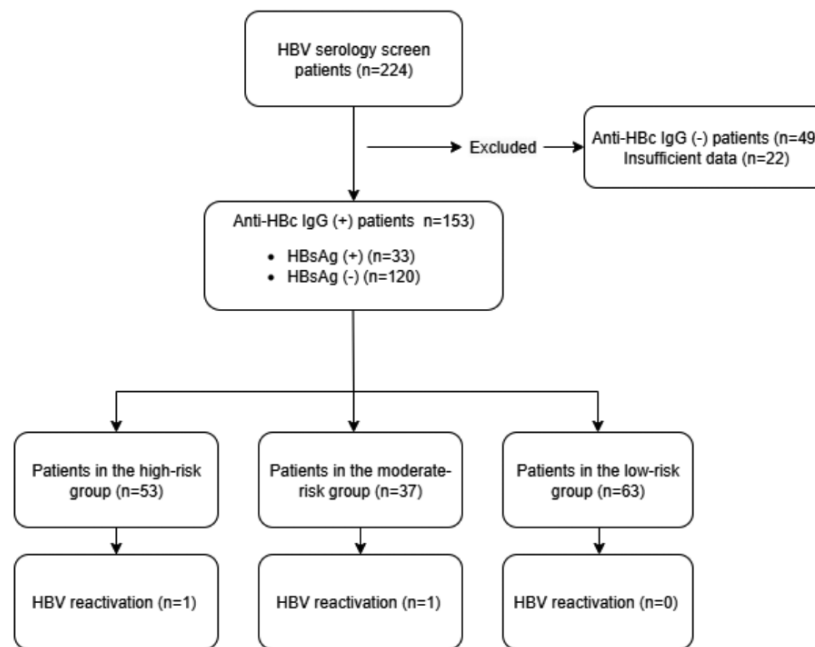
## Results

Hepatitis serology was analyzed in 224 patients who were referred from different departments of the hospital to the infectious diseases department for HBV prophylaxis. When the retrospective records were analyzed, 22 patients with missing data on immunosuppressive treatment were excluded. The 49 anti-HBc-negative patients were excluded from the study because they were not at risk of HBVr. One hundred and fifty three patients with HBsAg(+/-) and anti-HBc(+) status were included (Figure 1). Of the 153 patients, 33 (21.6%) were HBsAg-positive and 80 (52.3%) were anti-HBs-positive. The median (IQR) age of the patients was 62 (52.5-72) years, and 77 (50.3%) were female (Table 1). Among patients with detectable baseline HBV-DNA levels, the median (IQR) was 230 IU/mL (90-3009).

Patients were grouped according to the immunosuppressive treatments they received with regard to HBVr. Of the study population, 53 patients (34.6%) were classified as high-risk, 37 (24.2%) as moderate-risk, and 63 (41.2%) as low-risk.

HBV antiviral prophylaxis was initiated in 125 (81.7%) patients (Table 2). The antivirals used were entecavir (n=94, 75.2%), tenofovir disoproxil fumarate (TDF) (n=24, 19.2%), and tenofovir alafenamide (TAF) (n=7, 5.6%). One patient receiving entecavir was switched to TDF due to an allergic reaction. The median follow-up duration (IQR) was 10 (6-18) months.

Among the 28 patients (18.3%) who did not receive antiviral prophylaxis, two were classified as high-risk and were receiving rituximab-based regimens and anthracycline-group immunosuppressive agents. HBVr occurred in one patient



**Figure 1.** HBV reactivation associated with serologic profiles and antiviral prophylaxis

HBV: Hepatitis B virus, HBsAg: Hepatitis B surface antigen, Anti-HBc: Hepatitis B core antibody, IgG: Immunoglobulin G

treated with a rituximab-based regimen. Four patients in the moderate-risk group did not receive prophylaxis. These patients were receiving corticosteroid therapy, and one had HBVr. Twenty-two patients were identified as low-risk, including seven on corticosteroids, six on conventional synthetic disease-modifying antirheumatic drugs (DMARDs), and nine on anti-TNF agents. All nine patients receiving anti-TNF therapy (including four receiving etanercept, three receiving golimumab, and two receiving adalimumab) were HBsAg-negative. No HBVr was observed in this low-risk group.

HBVr was not observed in HBsAg-positive or anti-HBs-positive patients; two cases occurred in HBsAg-negative patients (0/33 vs. 2/120;  $p=1.000$ ) and in anti-HBs-negative patients (0/80 vs. 2/73;  $p=0.226$ ). In contrast, HBVr occurred only in patients who did not receive antiviral prophylaxis (0/125 vs. 2/28;  $p=0.033$ ). Table 3 summarizes the characteristics of patients who developed HBVr.

## Discussion

In this study, antiviral prophylaxis was administered to 96.2% of high-risk, 89.2% of moderate-risk, and 65.1% of low-risk patients. Entecavir was the predominant antiviral agent (75.2%). No cases of HBVr occurred among patients receiving prophylaxis, whereas HBVr was observed in two of 28 patients (7.1%) without prophylaxis. When stratified by risk, the HBVr rate in patients without prophylaxis was 50% (1/2) in the high-risk group, 25% (1/4) in the moderate-risk group, and not observed in the low-risk group.

In countries with HBsAg prevalence above 2%, hepatitis serology screening is recommended before initiating immunosuppressive therapy (5). In Türkiye, a seroprevalence study reported HBsAg and anti-HBc positivity rates of 4% and 30.6%, respectively (10). Because Türkiye is a country of moderate endemicity for HBV,

HBV serology screening is required prior to immunosuppressive treatment.

In a multicenter study of patients with hematologic malignancies receiving rituximab-based chemotherapy, HBVr was more common in those without antiviral prophylaxis (11). In our study, among 34 patients receiving rituximab-based therapy, only one patient—who did not receive prophylaxis—developed HBVr. No cases occurred among patients receiving prophylaxis. Current guidelines classify rituximab-containing regimens as high-risk and recommend antiviral prophylaxis (5,9). Although the number of cases in our study was small, our findings support these recommendations and highlight the importance of guideline implementation.

HBVr has been frequently reported in patients receiving anti-CD20 or anti-TNF therapy, though data on newer monoclonal antibodies remain limited (12). Recent evidence suggests that treatment with biologic or targeted synthetic DMARDs in patients with rheumatoid arthritis who are HBsAg-negative/anti-HBc-positive may increase the risk of HBVr (13). While some studies report minimal risk in anti-TNF-treated HBsAg-negative/anti-HBc-positive patients, others indicate a risk ranging from 0.4% to 6% (14,15,16,17). In our cohort, none of the nine low-risk, isolated anti-HBc-positive patients who received anti-TNF therapy without prophylaxis developed HBVr. These findings suggest that HBsAg-negative/anti-HBc-positive patients, unlike patients with HBsAg-positive serology, have a lower risk of HBVr with anti-TNF therapy. Accordingly, close clinical and laboratory monitoring with a preemptive strategy appears preferable to routine prophylaxis, minimizing unnecessary antiviral exposure.

In patients with multiple sclerosis (MS) receiving ocrelizumab, HBVr occurred in 28.6% of patients not receiving antiviral prophylaxis, while no cases were observed in those receiving prophylaxis (18). However, a multicentre study reported no cases

<b>Table 1.</b> Demographic and clinical characteristics of the patients	
<b>Variables</b>	<b>n=153</b>
<b>Age</b> , years, median (IQR)	62 (52.5-72)
<b>Gender</b> , n (%)	
Male	76 (49.7)
Female	77 (50.3)
<b>Follow-up duration</b> , months, median (IQR)	10 (6-18)
<b>Diseases</b> , n (%)	
Rheumatoid arthritis	33 (21.6)
Lymphoma	29 (18.9)
Multiple myeloma	22 (14.4)
Ankylosing spondylitis	17 (11.1)
Leukemia	15 (9.8)
Immune thrombocytopenic purpura	11 (7.2)
Multiple sclerosis	6 (3.9)
Autoimmune hemolytic anemia	6 (3.9)
Psoriatic arthritis	3 (2)
Others	11 (7.2)
<b>Baseline hepatitis serology</b> , n (%)	
HBsAg (+)	33 (21.6)
HBsAg (-)	120 (78.4)
Anti-HBs (+)	80 (52.3)
Anti-HBs (-)	73 (47.7)
Anti-HBc (+)	153 (100)
<b>Anti-HBs titer**</b> (IU/L), median (IQR)	160 (60-1000)
<b>Baseline AST level</b> (U/L), median (IQR)	24 (19-29)
<b>Baseline ALT level</b> (U/L), median (IQR)	25 (20-30)
<b>Baseline HBV-DNA status</b>	
Detectable	19 (45.2)
Undetectable	23 (54.8)
<b>Immunosuppressive agents used</b> , n (%)	
<b>Hematological diseases</b>	
Rituximab-based regimens	34 (22.2)
Corticosteroids	20 (13.1)
Bortezomib-based regimen	15 (9.8)
Anthracyclines	6 (3.9)
Antimetabolites	4 (2.6)
Tyrosine kinase inhibitor	3 (2)
B-cell lymphoma 2 inhibitors	2 (1.3)
Others	7 (4.6)
<b>Rheumatological diseases</b>	
Anti-TNF	30 (19.6)
csDMARDs	9 (5.9)
csDMARDs+corticosteroids	9 (5.9)
Anti-TNF+csDMARDs	8 (5.2)
<b>Neurological diseases</b>	
Ocrelizumab	6 (3.9)

<b>Table 1.</b> Continued	
<b>Variables</b>	<b>n=153</b>
<b>Antiviral prophylaxis agent</b> , n (%)	
Entecavir	94 (75.2)
Tenofovir disoproxil fumarate	24 (19.2)
Tenofovir alafenamide	7 (5.6)
Numerical variables were shown as median (IQR 25-75%). **: Median anti-HBs titer was calculated among patients with anti-HBs positive patients ( $\geq 10$ IU/L); categorical variables were expressed as number (%). Anti-TNF: Anti-tumor necrosis factor, csDMARDs: Conventional synthetic disease-modifying antirheumatic drugs, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, HBV: Hepatitis B virus, IQR: Interquartile range, HBsAg: Hepatitis B surface antigen, Anti-HBs: Hepatitis B surface antibody, Anti-HBc: Hepatitis B core antibody	

of HBVr among anti-HBc-positive patients with MS who received rituximab or ocrelizumab, irrespective of antiviral prophylaxis (19). Although data in the literature differ regarding the risk of HBVr, the recently published American Gastroenterological Association guideline places ocrelizumab in the same high-risk category as rituximab (9). In our study, all patients treated with ocrelizumab received antiviral prophylaxis, and no cases of HBVr were observed.

The degree of immunosuppression induced by corticosteroids depends on dose and duration. In patients receiving moderate- to high-dose corticosteroids for more than four weeks, HBsAg-positive individuals are classified as high-risk, while HBsAg-negative/anti-HBc-positive individuals are considered moderate-risk. When treated with low-dose corticosteroids for four weeks, HBsAg-positive and HBsAg-negative/anti-HBc-positive patients are classified as moderate- and low-risk, respectively (9). In our study, nine HBsAg-negative/anti-HBc-positive patients received corticosteroids without antiviral prophylaxis (two moderate-risk and seven low-risk). HBVr occurred in one of the two moderate-risk patients (50%, 1/2). Risk stratification for HBVr should consider corticosteroid dose and duration; and clinicians should remain vigilant even for moderate-risk patients.

Previous studies have suggested that anti-HBs positivity, particularly an anti-HBs titer above 100 IU/L, may have a protective effect against HBVr (13,20). The findings of our study are consistent with this observation. Among the 14 patients who were anti-HBs positive and did not receive antiviral prophylaxis (11 of whom had anti-HBs titers  $>100$  IU/L), none experienced HBVr. These patients were classified as belonging to the low-risk group. Although a generalization cannot be made because of the small sample size and inclusion of only low-risk patients, anti-HBs positivity may contribute to the prevention of HBVr; this finding should be investigated further in larger patient populations.

Increasing awareness of HBVr has led to more frequent hepatitis serological screening and identification of patients who are HBsAg(-)/anti-HBc(+). However, this has also resulted in unnecessary antiviral prophylaxis among low-risk individuals (21). Consistent with this observation, 65.1% of our low-risk group received prophylaxis. In accordance with current guidelines, close clinical and laboratory monitoring may be preferred for these patients.

**Table 2.** Use of HBV antiviral prophylaxis according to immunosuppression risk stratification

	High (n=53)	Moderate (n=37)	Low (n=63)	Total (n=153)
Use of antiviral prophylaxis (yes)	51 (96.2%)	33 (89.2%)	41 (65.1%)	125 (81.7%)
Use of antiviral prophylaxis (no)	2 (3.8%)	4 (10.8%)	22 (34.9%)	28 (18.3%)

HBV: Hepatitis B virus

**Table 3.** Clinical and demographic characteristics of patients with HBV reactivation

Patient characteristics	Patient 1	Patient 2
Age	49	78
Gender	Male	Female
Disease	Non-Hodgkin's lymphoma	Autoimmune hemolytic anemia
Treatment for the primary disease	Rituximab-based chemotherapy (cyclophosphamide, adriamycin, vincristine, methylprednisolone)	Dexamethasone+mycophenolate mofetil
Antiviral prophylaxis	No	No
HbsAg	Negative	Negative
Anti-HBc	Positive	Positive
Anti-HBs (IU/L)	Negative	Negative
HBV-DNA (IU/mL)	-	967
HBVr risk status	High	Moderate
After reactivation		
HBV-DNA (IU/mL)	20000000	8560518
ALT (U/L)	45	223
Initiated antiviral therapy	Entecavir	Tenofovir disoproxil fumarate
6 <sup>th</sup> month follow-up		
HBV-DNA (IU/mL)	18193	2670
ALT (U/L)	42	53
12 <sup>th</sup> month follow-up		
HBV-DNA (IU/mL)	0	0
ALT (U/L)	31	34

HBVr: Hepatitis B virus reactivation, ALT: Alanine aminotransferase, HbsAg: Hepatitis B surface antigen, Anti-HBs: Hepatitis B surface antibody, Anti-HBc: Hepatitis B core antibody

### Study Limitations

This study has some limitations because it was a single-center, retrospective study. Because the number of patients who did not receive antiviral prophylaxis was small, the results cannot be generalized. We believe that this study contributes to the literature by reporting outcomes for patients who did or did not receive antiviral prophylaxis with respect to HBVr.

### Conclusion

The incidence of HBVr may vary depending on the patient's immunosuppressive status. Therefore, these patients should undergo hepatitis serology screening. The decision to administer antiviral prophylaxis should be based on the patient's level of risk. In this study, no cases of HBVr were observed among patients who received prophylaxis. Entecavir, TDF, and TAF represent effective and safe options for HBVr prevention. Patients not receiving antiviral prophylaxis should be closely monitored for HBVr.

### Ethics

**Ethics Committee Approval:** Ethical approval was obtained from KTO-Karatay University Non-Drug and Non-Medical Device Research Ethics Committee (approval no: 2024/013, date: 07.06.2024).

**Informed Consent:** It was waived due to the retrospective nature of the study.

### Footnotes

#### Authorship Contributions

Concept: Y.G., A.T., Design: Y.G., A.T., Data Collection or Processing: Y.G., A.T., Analysis or Interpretation: Y.G., A.T., Literature Search: Y.G., A.T. Writing: Y.G., A.T.

**Conflict of Interest:** No conflict of interest was declared by the authors.

**Financial Disclosure:** The authors declare no financial support.



## References

- Visram A, Feld JJ. Defining and grading HBV reactivation. *Clin Liver Dis*. 2015;5:35-38.
- Hoofnagle JH. Reactivation of hepatitis B. *Hepatology*. 2009;49(5 Suppl):S156-S165.
- Lok ASF, Liang RHS, Chiu EKW, Wong KL, Chan TK, Todd D. Reactivation of hepatitis B virus replication in patients receiving cytotoxic therapy: report of a prospective study. *Gastroenterology*. 1991;100:182-188.
- Mason AL, Xu L, Guo L, Kuhns M, Perrillo RP. Molecular basis for persistent hepatitis B virus infection in the liver after clearance of serum hepatitis B surface antigen. *Hepatology*. 1998;27:1736-1742.
- Lau G, Yu ML, Wong G, Thompson A, Ghazian H, Hou JL, Piratvisuth T, Jia JD, Mizokami M, Cheng G, Chen GF, Liu ZW, Baatarkhuu O, Cheng AL, Ng WL, Lau P, Mok T, Chang JM, Hamid S, Dokmeci AK, Gani RA, Payawal DA, Chow P, Park JW, Strasser SI, Mohamed R, Win KM, Tawesak T, Sarin SK, Omata M. APASL clinical practice guideline on hepatitis B reactivation related to the use of immunosuppressive therapy. *Hepatol Int*. 2021;15:1031-1048.
- European Association for the Study of the Liver. EASL 2017 Clinical Practice Guidelines on the management of hepatitis B virus infection. *J Hepatol*. 2017;67:370-398.
- Papatheodoridis GV, Lekakis V, Voulgaris T, Lampertico P, Berg T, Chan HLY, Kao JH, Terrault N, Lok AS, Reddy KR. Hepatitis B virus reactivation associated with new classes of immunosuppressants and immunomodulators: a systematic review, meta-analysis, and expert opinion. *J Hepatol*. 2022;77:1670-1689.
- Cohen EB, Regev A, Garg A, Di Bisceglie AM, Lewis JH, Vierling JM, Hey-Hadavi J, Steplewski K, Fettiplace A, Chen CL, Pehlivanov N, Kendrick S, I Avigan M. Consensus guidelines: best practices for the prevention, detection and management of hepatitis B virus reactivation in clinical trials with immunosuppressive/immunomodulatory therapy. *Drug Saf*. 2024;47:321-332.
- Ali FS, Nguyen MH, Hernaez R, Huang DQ, Wilder J, Piscocoy A, Simon TG, Falck-Ytter Y. AGA clinical practice guideline on the prevention and treatment of hepatitis B virus reactivation in at-risk individuals. *Gastroenterology*. 2025;168:267-284.
- Tozun N, Ozdogan O, Cakaloglu Y, Idilman R, Karasu Z, Akarca U, Kaymakoglu S, Ergonul O. Seroprevalence of hepatitis B and C virus infections and risk factors in Turkey: a fieldwork TURHEP study. *Clin Microbiol Infect*. 2015;21:1020-1026.
- Clerico M, Dogliotti I, Ghione P, Zilioli VR, Merli F, Botto B, Al Essa W, Battaglini M, Grimaldi D, Cervi L, Ragaini S, Ferrero S, Peri V, De Luca G, Marzano A, Cavallo F. HBV reactivation in patients with past infection affected by non-Hodgkin lymphoma and treated with anti-CD20 antibody based immuno-chemotherapy: a multicenter experience. *J Pers Med*. 2022;12:285.
- De Pauli S, Grando M, Miotti G, Zeppieri M. Hepatitis B virus reactivation in patients treated with monoclonal antibodies. *World J Virol*. 2024;13:1-6.
- Tien YC, Yen HH, Li CF, Liu MP, Hsue YT, Hung MH, Chiu YM. Changes in hepatitis B virus surface antibody titer and risk of hepatitis B reactivation in HBsAg-negative/HBcAb-positive patients undergoing biologic therapy for rheumatic diseases: a prospective cohort study. *Arthritis Res Ther*. 2018;20:1-8.
- Sayar S, Kürbüz K, Kahraman R, Öztürk O, Çalışkan Z, Doğanay HL, Özdil K. Risk of hepatitis B reactivation during anti-TNF therapy; evaluation of patients with past hepatitis B infection. *Turkish J Gastroenterol*. 2020;31:522-528.
- Taş Aygar G, Haykır Solar A, Yılmaz OK, Karataş H, Çevirgen Cemil B, Kartal SP. Risk of HBV reactivation during immunosuppressive therapy in psoriasis: a retrospective analysis. *Viral Hepat J*. 2025;31:59-65.
- Fidan S, Capkın E, Arica DA, Durak S, Okatan IE. Risk of hepatitis B reactivation in patients receiving anti-tumor necrosis factor- therapy. *Int J Rheum Dis*. 2021;24:254-259.
- Pappa M, Koutsogianni A, Karamanakos A, Kyriazi N, Cheila M, Moschou D, Mole E, Gazi S, Papadimitriou E, Atzeni F, Sebastiani M, Argyropoulou OD, Vasilakis KD, Papagoras C, Fragoulis GE, Androutsakos T. Similar hepatitis B virus reactivation risk for patients with inflammatory arthritis or connective tissue diseases: a multicenter retrospective study. *Rheumatol Int*. 2025;45:15.
- Çelik M, Baba C, Irmak Ç, Özakbaş S, Avkan-Oğuz V. Risk of hepatitis B virus reactivation in people with multiple sclerosis treated with ocrelizumab: an observational study from Turkey. *J Neurol*. 2024;271:4131-4137.
- Buonomo AR, Viceconte G, Calabrese M, De Luca G, Tomassini V, Cavalla P, Maniscalco GT, Ferraro D, Nociti V, Radaelli M, Buscarinu MC, Paolicelli D, Gajofatto A, Annovazzi P, Pinardi F, Di Filippo M, Cordioli C, Zappulo E, Scotto R, Gentile I, Spiezia AL, Petruzzio M, De Angelis M, Brescia Morra V, Solaro C, Gasperini C, Cocco E, Moccia M, Lanzillo R; Raising Italian Researchers in Multiple Sclerosis (RIREMS) study group. Management of hepatitis B virus prophylaxis in patients treated with disease-modifying therapies for multiple sclerosis: a multicentric Italian retrospective study. *J Neurol*. 2022;269:3301-3307.
- Poola S, Kratzer M, Sewell K, Tillmann HL. Size matters! Anti-HBs titer and HBV reactivation during anti-TNF therapy. *Dig Dis Sci*. 2023;68:4511-4520.
- Yenilmez E, Cetinkaya RA. Overuse of prophylaxis in HBsAg and/or anti-HBc positive patients after increasing awareness to prevent reactivation in patients receiving immunosuppressive therapies: how rational are our prophylaxis decisions according to the literature? *Infez Med*. 2019;27:299-307.