INTRODUCTION

Hepatitis B virus (HBV) and hepatitis C virus (HCV) have been identified as oncogenic viruses that might cause non-Hodgkin’s lymphomas (NHL) by the International Agency for Research on Cancer (IARC). The association between HBV or HCV infection and NHL has been demonstrated in diverse epidemiological studies and mainly observed in B-cell non-Hodgkin's lymphoma (B-NHL). Furthermore, it was reported in different interventional studies that patients with both HCV infection and indolent B-NHL such as splenic marginal zone lymphoma (SMZL), mucosa-associated lymphoid tissue lymphoma (MALT) and follicular lymphoma (FL) were successfully treated with anti-HCV agents such as (pegylated) interferon and ribavirin. Therefore, HCV activity was supposed to play a significant role in the development of indolent lymphomas, and it was shown that HCV eradication might be a promising treatment option for these patients. Contrary to HCV, the link between HBV infection and B-NHL has been explored less intensively. Koot et al reported a case of SMZL with co-diagnosed HBV infection, in which tenofovir treatment led to a...
complete remission (CR) of SMZL. This finding suggested that HBV activity might also play a role in the development of indolent B-NHL in patients with HBV infection. However, the clinical implication of HBV activity remains a matter of debate. Therefore, studies evaluating HBV activity in patients with both HBV infection and indolent or aggressive B-NHL are of high clinical significance and might provide further information for the clinical practice.

The aim of the current study was to evaluate clinical characteristics, treatment, and serological indicators of HBV activity in patients who were diagnosed with both HBV infection and indolent or aggressive B-NHL.

2 | METHODS

2.1 | Selection and collection of patients’ data

We performed a retrospective single center analysis of patients with indolent or aggressive B-NHL, which were co-diagnosed with a current or resolved HBV infection. This study has been approved by the ethics committee of the Heidelberg University Hospital. All subtypes of B-NHL were included into this study. The histology was determined according to the World Health Organization classification of NHL at the department of Pathology of the Heidelberg University Hospital. By searching in the hospitals database with support of the information technology (IT) department, we identified and retrieved data of patients who received in- or outpatient treatment from January 2000 to June 2017 at our hospital. Patients’ demographic characteristics, B-NHL-related data (time point of the first diagnosis of B-NHL, subtype, treatment, and response), serological HBV markers at the first diagnosis of B-NHL (hepatitis B core antibody [Anti-HBc], hepatitis B surface antigen [HBsAg], hepatitis B envelope antigen [HBeAg] and HBV-DNA), and anti-HBV therapy-related data were evaluated. Patients were grouped according to B-NHL subtype: indolent and aggressive B-NHL. A subgroup of patients with both HBV infection and DLBCL was evaluated in a separate analysis published by the authors and, in our previous publication, we focused mainly on the prognostic value of different serological HBV markers in the subgroup of DLBCL.

2.2 | Determination of the serological indicators of HBV activity

Serological indicators of HBV activity were tested at the first diagnosis of B-NHL. The diagnosis of HBV infection was based on the results of serological tests at the department of Virology of the Heidelberg University Hospital. Polymerase chain reaction (PCR) was used to quantify the HBV-DNA in serum at a sensitivity level of 10 IU/mL. HBsAg and HBeAg were tested via enzyme immunoassay (EIA). In some cases, only a qualitative test was performed to test these serological parameters.

2.3 | Statistical analysis

For descriptive statistics, data are given as absolute numbers and percentage, and if not otherwise stated as median and range. We used two-tailed Fisher’s exact test to investigate the difference in HBsAg and HBeAg between the subgroups. To compare the HBV-DNA in the subgroups, two-tailed Mann-Whitney U test was used. These analyses were performed with GraphPad Prism 6.0 (GraphPad Software Inc). A P-value <.05 was considered as statistically significant.

3 | RESULTS

3.1 | Patients’ characteristics and treatment

Overall, 72 patients diagnosed with both B-NHL and current or resolved HBV infection were identified. The median age at diagnosis of B-NHL was 58 (range 22-83) years, and DLBCL was the most frequent subtype of B-NHL in our cohort (n = 37, 51%). An indolent B-NHL was diagnosed in 27 patients (38%) and aggressive B-NHL in 45 patients (62%).

In the group of indolent B-NHL, there were 14 (52%) patients with FL, six (22%) patients with marginal zone lymphoma (MZL), four (15%) patients with chronic lymphocytic leukemia (CLL), and three (11%) patients with MALT. In seven (26%) patients with indolent B-NHL, watch and wait was the primary therapy strategy at diagnosis and all of them obtained either systemic chemotherapy (n = 6) or splenectomy (n = 1) as secondary therapy strategy in the course of disease due to progression of B-NHL. No patient received antiviral therapy with the intention to treat B-NHL. Patients’ characteristics and treatment-related data of the 27 patients with indolent B-NHL are summarized in Table 1A.

Among the 45 patients who were diagnosed with an aggressive B-NHL, DLBCL was the most frequent subtype of B-NHL (n = 37, 82%). The majority of patients (n = 32, 71%) was treated with (R)-CHOP regimen (rituximab, cyclophosphamide, hydroxydaunorubicin, vincristine, prednisolone). No patient obtained antiviral therapy with the intention to treat B-NHL. Characteristics of patients with aggressive B-NHL are summarized in Table 1B.

In total, six (13%) and four (15%) patients had a HCV co-infection in the subgroup of aggressive and indolent B-NHL, respectively. Human immunodeficiency virus (HIV) co-infection was registered in one patient with DLBCL, and this patient had also a HCV co-infection. Of note, 26 (36%) patients were international patients or patients with migration background. One (1%), four (6%), and 21 (29%) patients had a Southeast Asian, Eastern European, and Mediterranean origin, respectively.

3.2 | HBV reactivation and antiviral therapy

In our cohort, concurrent anti-HBV therapy was administered regularly during immunochemotherapy. Twenty-four (33%), 12 (17%), six
ZHOU et al. (8%), and one (2%) patients received lamivudine, entecavir, tenofovir, and adefovir, respectively. In 29 (40%) patients, there was no anti-HBV therapy administered in the course of the disease. Hepatitis B virus reactivation with elevation of HBV-DNA was detected in only 11 (15%) patients. Among these 11 patients, one patient with FL received lamivudine during R-CHOP. Lamivudine was stopped directly after the last cycle of immunochemotherapy, and 4 months later, the patient developed HBV reactivation. In the remaining ten patients, HBV reactivation occurred during chemotherapy, and no anti-HBV therapy was administered prior to HBV reactivation. No patient received anti-HBV therapy with the intention to treat B-NHL in both subgroups.

3.3 | Serological indicators of HBV activity

We analyzed anti-HBc status and the serological indicators of HBV activity including HBsAg, HBeAg, and HBV-DNA in patients with indolent and aggressive B-NHL at the first diagnosis of B-NHL.

Anti-HBc is a marker, which indicates an exposure to HBV. Overall, the majority of patients were anti-HBc-positive (n = 60, 83%), and no patient was anti-HBc-negative at the first diagnosis of B-NHL. The anti-HBc status was not available in 12 (17%) patients. Positive HBsAg status indicates a current HBV infection, and HBeAg is a serum marker that correlates with high HBV replication. Among patients with indolent and aggressive B-NHL, 16 out of 27 (59%) and 17 out of 45 (38%) patients were HBsAg-positive at the first diagnosis of B-NHL, respectively (Tables 1A and 1B). In the subgroup of indolent B-NHL, the proportion of HBsAg-positive patients was significantly higher when compared to the subgroup of aggressive B-NHL (59% vs 38%, P = .03). However, only one out of 27 and two out of 45 patients were HBeAg-positive at the first diagnosis of B-NHL in the subgroups indolent and aggressive B-NHL, respectively. There was no difference in
the positive rate of HBsAg between the both subgroups (4% vs 4%) (Tables 1A and 1B).

We also analyzed HBV-DNA at the first diagnosis of B-NHL in the both subgroups. HBV-DNA at diagnosis of B-NHL was available in 13 and 23 patients in the indolent and aggressive B-NHL groups, respectively. Among the patients with aggressive B-NHL, 15 patients (33%) were HBV-DNA-negative, while only three patients (11%) had negative HBV-DNA in the subgroup of indolent B-NHL at the first diagnosis of B-NHL. Overall, HBV-DNA level at the first diagnosis of B-NHL was significantly higher in patients with indolent B-NHL when compared to patients with aggressive B-NHL ($P = .01$, Figure 1).

### 3.4 Subgroup analysis of DLBCL and FL

Li et al reported in a recent updated meta-analysis of 58 studies that DLBCL and FL had the most significant association with HBV infection within the B-NHL subtypes.\textsuperscript{15} In our study, DLBCL and FL were the most common subtypes among aggressive and indolent B-NHL, respectively (Tables 1A and 1B). Therefore, we performed a subgroup analysis of DLBCL and FL.

Overall, we identified 14 patients with FL and 37 patients with DLBCL, and data of HBsAg were available in ten and 34 patients, respectively. The rate of HBsAg positivity was significantly higher in patients with FL compared with that in DLBCL (83% vs 44%, $P = .04$). In contrast, no patient (0%) was HBeAg-positive in the FL subgroup, while the rate of HBeAg positivity was 9% (2/21) in DLBCL, and

### TABLE 1B Characteristics of patients with aggressive B-NHL (Continued)

| Parameter | Value |
|-----------|-------|
| Patients, n | 45 |
| Gender, n (%) | |
| Male | 32 (71) |
| Female | 13 (29) |
| B-NHL diagnosis | |
| Age at the first diagnosis, median years (range) | 61 (22-82) |
| Diagnosis specification, n (%) | |
| DLBCL | 37 (82) |
| PCNSL | 3 (7) |
| MCL | 2 (4) |
| BLBL | 1 (2) |
| BL | 1 (2) |
| High-grade B-NHL, NOS | 1 (2) |
| First-line treatment, n (%) | |
| DLBCL, n = 37 | |
| (R)-CHOP like | 31 (83) |
| R-Benda | 2 (5) |
| GMALL-B-ALL/NHL | 1 (3) |
| MATRix | 1 (3) |
| Local surgery | 1 (3) |
| Others | 1 (3) |
| PCNSL, n = 3 | |
| Freiburger protocol | 1 (33) |
| PRIMAIN | 1 (33) |
| Others | 1 (33) |
| MCL, n = 2 | |
| R-Benda | 1 (50) |
| Others | 1 (50) |
| BLBL, n = 1 | |
| GMALL-B-ALL/NHL | 1 (100) |
| BL, n = 1 | |
| GMALL-B-ALL/NHL | 1 (100) |
| High-grade B-NHL, NOS, n = 1 | |
| (R)-CHOP like | 1 (100) |
| Response to first-line treatment, n (%) | |
| CR | 21 (47) |
| PR | 16 (36) |
| SD | 6 (13) |
| PD | 2 (4) |
| Serological HBV markers at the first diagnosis of B-NHL | |
| HBsAg, n (%) | |
| Positive | 17 (38) |
| Negative | 23 (51) |
| NA | 5 (11) |
there was no statistically significant difference between the both subgroups. HBV-DNA level at the first diagnosis of B-NHL is significantly higher in the FL group than that in DLBCL ($P = .007$, Figure 2).

4 | DISCUSSION

In the current study, we analyze the serological indicators of HBV activity including HBsAg, HBeAg, and HBV-DNA in 72 patients who were diagnosed with both HBV infection and B-NHL upon the first diagnosis of B-NHL. Notably, 26 (36%) patients were from regions with higher prevalence of HBV infection than Germany, that is, Southeast Asia, Eastern Europa, and Mediterranean region.16

At the first diagnosis of B-NHL, patients with both HBV infection and indolent B-NHL had a significantly higher positive rate of HBsAg when compared with the group of aggressive B-NHL. Similarly, Cucuianu et al reported that positive HBsAg status was more frequent in patients with low-grade B-NHL than patients with aggressive B-NHL (37% vs 27%).17 By contrast, Xiong et al and Wang et al reported that patients with aggressive B-NHL had a significantly higher rate of positive HBsAg compared to patients with indolent B-NHL.7,18 In the current analysis, all patients had a current or resolved HBV infection with positive HBsAg or isolated positive anti-HBc and no patient was anti-HBc-negative. It was reported that subtypes of HBV with various mutations, for example, T125N, N48T, Q82P, T97N, N97T, and P93Q, might have increased or reduced HBsAg expression.19,20 Unfortunately, we could not retrospectively identify these subtypes of HBV and their impact on HBsAg status in our cohort.

We observed that patients with both HBV infection and indolent B-NHL had a significantly higher HBV-DNA level at the first diagnosis of B-NHL when compared to patients who had an aggressive B-NHL. To the best of our knowledge, this is the first study investigating HBV-DNA levels in patients with both HBV infection and indolent or aggressive B-NHL. Our results suggest that serological HBV activity is significantly higher in patients with both HBV infection and indolent B-NHL compared to the group of aggressive B-NHL.

HBeAg is a serum marker indicating high activity of viral replication and high infectivity.14 Interestingly, we observed no difference in HBeAg status upon the first diagnosis of B-NHL between both subgroups indolent and aggressive B-NHL. In contrast to our findings, it was previously reported that the prevalence of positive HBeAg status was significantly higher in patients with aggressive B-NHL in comparison to patients with indolent B-NHL.7,18 Indeed, studies suggested that diverse mutations of HBV, for example, G1862T, G1896A, Kozak 1809-1812, and 1762T/1764A might lead to reduced HBeAg expression.21-24 These mutations might also exist in our cohort and could interfere the HBeAg status.

Recently, next-generation sequencing (NGS) data revealed HBV-DNA presence in plasma, B-cell, and tumor tissue of patients with DLBCL, and therefore, the lymphotropic nature and oncogenic potency of HBV could be supported by these findings.25 To date, serological HBV activity and its clinical implication in patients with HBV infection and B-NHL has been much less frequently investigated than HCV activity in B-NHL patients with a co-diagnosed HCV infection, although both HBV and HCV have been identified to be lymphotropic and oncogenic.26,27 In B-NHL patients with HCV infection, HCV activity was identified to be a possible mechanism in the development of indolent B-NHL. This hypothesis was supported by studies in which indolent B-NHL was successfully treated with anti-HCV therapy.27 Accordingly, HBV activity was also suggested to play a relevant role in the pathogenesis of indolent B-NHL.28 However, there were only few interventional studies supporting this hypothesis. In a case report published by Koot et al, a patient with both HBV infection and SMZL who had bone marrow involvement was treated with tenofovir monotherapy and achieved complete remission (CR) of SMZL.11 In our study, no patient received anti-HBV therapy with

![FIGURE 1](image1.png)  
**FIGURE 1** HBV-DNA at the first diagnosis of B-NHL in patients with HBV infection and aggressive or indolent B-NHL. At the first diagnosis of B-NHL, patients with indolent B-NHL ($n = 13$) had significantly higher HBV-DNA level compared to those with aggressive B-NHL ($n = 23$, $P = .01$)

![FIGURE 2](image2.png)  
**FIGURE 2** HBV-DNA at the first diagnosis of B-NHL in patients with HBV infection and DLBCL or FL. At the first diagnosis of B-NHL, patients with FL ($n = 7$) had significantly higher HBV-DNA level compared to those with DLBCL ($n = 22$, $P = .007$)
the intention to treat B-NHL, and all patients with indolent B-NHL were treated with immunochemotherapy or splenectomy in the course of the disease due to progression of B-NHL. Nevertheless, our results emphasized the relevance of serological HBV activity in indolent B-NHL and provide rationale for further interventional studies. Furthermore, our findings suggested that serological HBV activity probably did not play a significant role in the pathogenesis of aggressive B-NHL. Similar to HCV-related B-NHL, permanent genetic cell damage caused by a transiently intracellular virus might be more relevant in the pathogenesis of aggressive B-NHL rather than virus activity in serum.27

Li et al reported in a meta-analysis that DLBCL and FL were most significantly associated with HBV infection among the B-NHL subtypes.15 In our study, FL and DLBCL were the most common subtypes within indolent and aggressive B-NHL. Of note, we also observed significantly higher HBV activity with higher rate of HBsAg positivity and higher HBV-DNA level in FL compared with the DLBCL subgroup.

HBV reactivation is a well-known complication in patients treated with immunochemotherapy, for example, rituximab.29 In our cohort, HBV reactivation was observed in 11 patients. Ten of them had not received anti-HBV therapy prior to HBV reactivation, and one patient developed HBV reactivation 4 months after the last cycle of R-CHOP. To date, it is recommended that anti-HBV prophylaxis should be continued for 12 months after the last chemotherapy.30 However, the optimal duration of monitoring and prophylactic anti-HBV therapy is still a matter of debate, since HBV reactivation may also occur even 1 year or more after cessation of the treatment, and therefore, follow-up for HBV reactivation should be continued for as long as practicable.31,32

There are several limitations of our current study. First, the retrospective design and, therefore, the missing values in the analysis represented the main limitation of our study. Second, HBV-DNA testing was not part of standard screening for HBV at the first diagnosis of B-NHL. Therefore, patients with occult HBV infection and only isolated positive HBV-DNA might not have been included into the current analysis. Our findings should be interpreted with caution and investigated in further studies.

In conclusion, our findings suggested that serological viral activity of HBV was significantly higher in indolent B-NHL than aggressive B-NHL. Due to the lymphotropic and oncogenic nature of HBV, similar to HCV, HBV suppression with anti-HBV therapy might also represent a promising therapy strategy for patients with both HBV infection and indolent B-NHL. In contrast, anti-HBV therapy might not be suitable as a B-NHL therapy for patients with HBV infection and aggressive B-NHL. Our findings provide rationale for further studies investigating the relevance of HBV activity in the pathogenesis and treatment of B-NHL.

CONFLICT OF INTEREST
All authors declare that they have no conflicts of interest relevant to the submitted manuscript.

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