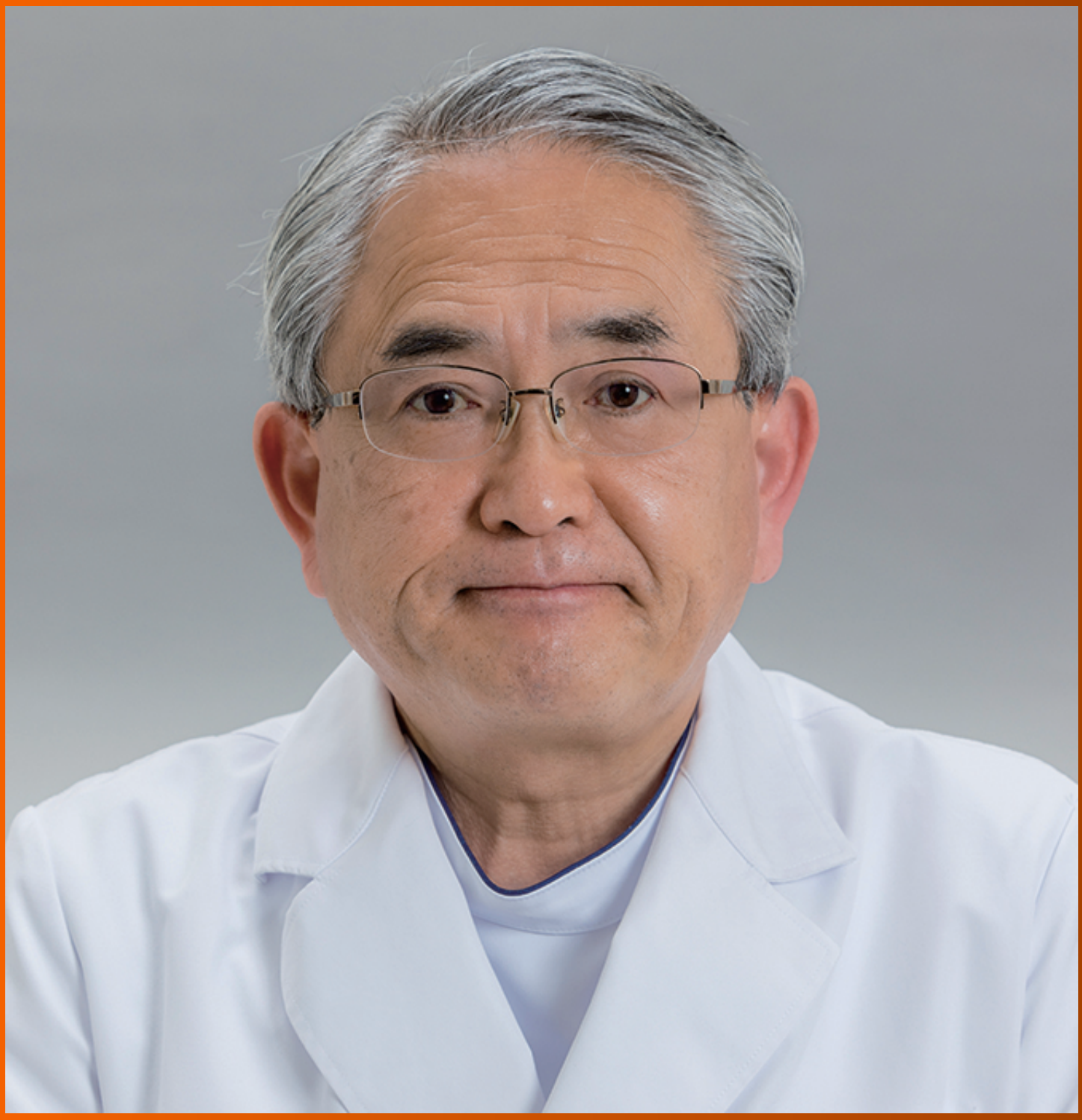
ISSN 1007-9327 (print)
ISSN 2219-2840 (online)

# World Journal of *Gastroenterology*

*World J Gastroenterol* 2024 July 7; 30(25): 3126-3184



Published by Baishideng Publishing Group Inc

World Journal of Gastroenterology

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**NAME OF JOURNAL**
*World Journal of Gastroenterology*

**ISSN**
ISSN 1007-9327 (print) ISSN 2219-2840 (online)

**LAUNCH DATE**
October 1, 1995

**FREQUENCY**
Weekly

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**PUBLICATION DATE**
July 7, 2024



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Biliary Tract Disease Institute, Fudan University

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*World J Gastroenterol* 2024 July 7; 30(25): 3147-3151

DOI: 10.3748/wjg.v30.i25.3147

ISSN 1007-9327 (print) ISSN 2219-2840 (online)

EDITORIAL

# Risk of hepatic decompensation from hepatitis B virus reactivation in hematological malignancy treatments

Michele Barone

**Specialty type:** Gastroenterology and hepatology

**Provenance and peer review:** Invited article; Externally peer reviewed.

**Peer-review model:** Single blind

**Peer-review report's classification**
**Scientific Quality:** Grade B
**Novelty:** Grade B
**Creativity or Innovation:** Grade B
**Scientific Significance:** Grade B

**P-Reviewer:** Yang Z, China

**Received:** March 25, 2024
**Revised:** May 20, 2024
**Accepted:** June 6, 2024
**Published online:** July 7, 2024
**Processing time:** 97 Days and 20.4 Hours

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## Abstract

In this editorial, we discussed the apparent discrepancy between the findings described by Colapietro *et al*, in their case report and data found in the literature. Colapietro *et al* reported a case of hepatitis B virus (HBV)-related hepatic decompensation in a patient with chronic myeloid leukemia and a previously resolved HBV infection who was receiving Bruton's tyrosine kinase (BTK) inhibitor therapy. First of all, we recapitulated the main aspects of the immune system involved in the response to HBV infection in order to underline the role of the innate and adaptive response, focusing our attention on the protective role of anti-HBs. We then carefully analyzed literature data on the risk of HBV reactivation (HBVr) in patients with previous HBV infection who were treated with either tyrosine kinase inhibitors or BTK inhibitors for their hematologic malignancies. Based on literature data, we suggested that several factors may contribute to the different risks of HBVr: The type of hematologic malignancy; the type of therapy (BTK inhibitors, especially second-generation, seem to be at a higher risk of HBVr than those with tyrosine kinase inhibitors); previous exposure to an anti-CD20 as first-line therapy; and ethnicity and HBV genotype. Therefore, the warning regarding HBVr in the specific setting of patients with hematologic malignancies requires further investigation.

**Key Words:** Hematological malignancy; Hepatitis; Hepatitis B virus-DNA; Bruton's tyrosine kinase; Previously resolved hepatitis B virus infection

**Core Tip:** All literature data on the risk of hepatitis B virus reactivation (HBVr) in patients with a previously resolved HBV infection and treated with tyrosine kinase inhibitors for their hematologic malignancies are based on retrospective studies. Different risks of HBVr in these patients may depend on the type of hematologic malignancy, the type of therapy (tyrosine kinase inhibitors or Bruton's tyrosine kinase inhibitors), previous exposure to an anti-CD20 as first-line therapy, and both ethnicity and HBV genotype. Therefore, the warning regarding HBVr in this specific clinical setting requires further investigation.

**Citation:** Barone M. Risk of hepatic decompensation from hepatitis B virus reactivation in hematological malignancy treatments. *World J Gastroenterol* 2024; 30(25): 3147-3151
**URL:** https://www.wjgnet.com/1007-9327/full/v30/i25/3147.htm
**DOI:** https://dx.doi.org/10.3748/wjg.v30.i25.3147

## INTRODUCTION

In their letter, Colapietro *et al*[1] discussed a single case of hepatitis B virus reactivation (HBVr) in a patient with chronic lymphocytic leukemia undergoing therapy with a Bruton's tyrosine kinase (BTK) inhibitor. Based on both their findings and a summary of literature data, they issued a warning on the use of tyrosine kinase inhibitors (TKIs) in this clinical setting. This article opens up an important debate on the topic, if we consider that their patient developed hepatic decompensation. In this editorial, we introduced some additional aspects that contribute to enriching the discussion on this topic, attempting to provide an explanation for the apparent discrepancy between the findings described by Colapietro *et al*[1] and the most recent literature data on the risk of HBVr in a patient with hematologic malignancies (Table 1)[2-9].

### The immune responses against HBV infection

The host adaptive T and B cell responses lead to the immune response against the HBV infection[10]. The persistence in hepatocytes of the full-length, covalently closed circular DNA in a latent state acts as a reservoir for HBV reactivation in those subjects who do not completely clear the virus[11]. Vaccination protects against HBV infection by stimulating the production of hepatitis B surface antibodies (anti-HBs), and hypogammaglobulinemia is the predominant inherent immune defect in patients with hematologic malignancies[12]. All drugs, therefore, capable of reducing protective immunoglobulin levels against hepatitis B surface antigen (HBsAg) could theoretically increase the risk of HBVr in these patients.

However, a previously resolved HBV infection, with or without the persistence of covalently closed circular DNA, is identified by the presence of the hepatitis B core antibody, which may or may not be associated with anti-HBs. Therefore, the reduction of the protective anti-HBs titer cannot be used as a predictor of reactivation in anti-HBs-negative patients. In addition, experience with anti-CD20 monoclonal antibodies, which is associated with a high risk of HBVr in hematologic malignancies, has shown that HBVr is not only related to a reduced production of antibodies (in this case anti-HBs) but also depends on the depletion of CD20-positive B cells. This has an impact on the production of interleukins that are critical for memory T cell survival and T cell number[13,14].

It is widely accepted that the host immunity and HBV characteristics (genotype and HBsAg mutations) influence HBVr [15-17]. At least ten genotypes (A to J) are currently recognized, based on the intergroup nucleotide sequence divergence. In addition, more than 40 genetic subtypes have been identified[18].

Although viral genotypes influence the clinical evolution of HBV infection in terms of cirrhosis and hepatocellular carcinoma[19,20], there is no study comparing the impact of genotypes on the risk of HBVr in patients treated with immunosuppressants, immunomodulators, and TKIs. The possible role of innate immunity in controlling HBV infection remains practically unexplored[21].

### Risk of HBVr in patients with hematologic malignancies and previously resolved HBV infection

Although TKIs and BTKs are two therapies widely used in the treatment of patients with hematologic malignancies, the exact mechanism by which they would favor HBVr still remains unclear. However, as previously mentioned for the CD20 inhibitor rituximab, BTK plays an active role in B cell differentiation, proliferation, and survival and is highly expressed in a wide variety of immune cells, a condition that could explain the risk of HBVr[22]. Moreover, TKIs impair the B cell immune response in patients with chronic myeloid leukemia (CML) and inhibit antigen-specific T cell activation and proliferation *in vitro*[23,24]. Nevertheless, the hypothesis of immune restoration after therapy ends does not explain the occurrence of HBVr during the treatment[25].

The apparent discrepancy between the conclusions of Colapietro *et al*[1], based on their case report, two other case reports, and the recent study of Chiu *et al*[7], and the data from literature published from 2016-2022 can be explained

**Table 1 Hepatitis B surface antigen-negative patients with either hepatitis B core or surface antibody or hepatitis B core/surface antibody positivity treated with tyrosine kinase inhibitors for hematological malignancies**

| Ref. | Type of disease, *n* | First-line TKI therapy | Patients, *n* | HBVr, *n* | Hepatitis, *n* | Liver failure, *n* |
|---|---|---|---|---|---|---|
| Orlandi *et al*[2] | CML | NS[1] | 26 | 0 | 0 | 0 |
| Sora *et al*[3] | CML | 6 imatinib[1], 4 nilotinib[1] | 10 | 0 | 0 | 0 |
| Wang *et al*[4] | CML | NS[1] | 123 | 0 | 0 | 0 |
| Innocenti *et al*[5] | CML | Ibrutinib[2] | 108 | 2 | 0 | 0 |
| Hammond *et al*[6] | CLL (15) MCL (4) WM (2) | Ibrutinib[2] | 21 | 2 | 0 | 0 |
| Chiu *et al*[7] | CCL (22) MCL (6) | 12 ibrutinib[2], 1 zanubrutinib[2], 15 acalabrutinib[2] | 28 | 2 | 2 | 2 |
| Tsuruya *et al*[8] | CLL | Ibrutinib[2] | 1 | 1 | 1 | 0 |
| Lam *et al*[9] | | Ibrutinib[2] | 1 | 1 | 1 | 0 |

[1]First-generation or second-generation tyrosine kinase inhibitors (imatinib and dasatinib/nilotinib, respectively) used alone or in combination.
[2]Bruton's tyrosine kinase inhibitors.
[3]Patients with remote use of anti-hepatitis B virus therapy or anti-CD20 monoclonal antibodies were included in this study.
All studies reported in the table were retrospective. CML: Chronic myeloid leukemia; CLL: Chronic lymphocytic leukemia; HBVr: Hepatitis B virus reactivation; MCL: Mantle cell lymphoma; NS: Not significant; TKI: Tyrosine kinase inhibitors; WM: Waldenstrom macroglobulinemia.

making use of different considerations. First, different hematologic malignancies and different types of therapies (first-generation or second-generation TKI and BTK inhibitors) were reported in the studies; second, the patients participating in the studies were from different geographical areas, which is an aspect linked to both ethnicity and HBV genotype.

As shown in Table 1, the first three studies[2-4] enrolled only patients with CML treated with TKIs, and none of the 159 patients experienced HBVr. The fourth study[5] enrolled 108 Caucasian patients with a similar hematologic malignancy (CML) but who were instead receiving a first-generation BTK, namely ibrutinib. Interestingly, the 2 patients (1.8%) who experienced HBVr had previously received chemoimmunotherapy and did not develop signs of hepatitis. Unlike the previous one, the study by Hammond *et al*[6] involved 21 patients with different hematologic malignancies treated with the same BTK inhibitor. In this study, a higher percentage of HBVr (9.5%) was observed with no signs of hepatitis, suggesting a different risk of HBVr due to the underlying disease.

The most recent study by Chiu *et al*[7] reported HBVr in 2 of the 28 patients who both experienced liver failure. In this study, they had received a second-generation BTK inhibitor (acalabrutinib or zanubrutinib) as second-line therapy. One of the two patients was 92-years-old, and both patients had previously received anti-CD20 monoclonal antibodies as first-line therapy. Interestingly, acalabrutinib was the same BTK inhibitor used by Colapietro *et al*[1].

Finally, the two case reports reported in Table 1 both possess some peculiarities that may justify the elevated risk of HBVr. The patient described by Tsuruya *et al*[8] was anti-HBs positive and HBV-DNA negative at screening. However, it is a known fact that low HBV-DNA levels can fluctuate. Lam *et al*[9] described a case of HBVr in an asymptomatic 82-year-old female with seronegative occult HBV infection (*i.e.* negative for HBsAg and anti-HBs), who was not tested for HBV-DNA before the commencement of therapy.

## CONCLUSION

Our data support a simple follow-up in patients with hematologic malignancies and a history of previously resolved HBV infections who were treated with TKI therapy. On the other hand, those patients who are treated with BTK inhibitors deserve more attention. Notably, it would seem that Asian patients are more at risk of HBVr with clinical complications compared to Caucasian patients. To definitively clarify the risk of HBVr in this clinical setting, it is necessary to perform prospective studies taking into consideration several aspects: The type of underlying hematologic disease; the patients' clinical history especially if they had already received a first-line therapy or have important comorbidities; and advanced age.

## FOOTNOTES

**Author contributions:** Barone M conceived and wrote this manuscript.

**Conflict-of-interest statement:** The author reports no relevant conflicts of interest for this article.

**Country of origin:** Italy

**ORCID number:** Michele Barone 0000-0001-8284-5127.

**S-Editor:** Li L
**L-Editor:** Filipodia
**P-Editor:** Zhang L

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