





Comparison of hepatitis B reactivation in patients with resolved hepatitis B infection receiving rituximab or non-rituximab based immunosuppressive therapy. Does the presence of hepatitis b surface antibody reduce risk of reactivation? Abstract 110/ Poster 41

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BACKGROUND AND AIMS

Hepatitis B virus reactivation (HBVr) and flare may occur in

Table 1. Logistic regression analysis of rituximab exposure and
 anti-HBs levels in predicting HBVr, all patients

patients with resolved HBV infection (HBsA negative, anti-HBc antibody positive) who receive immunosuppressive therapy. Compared to most other immunosuppressive therapy, rituximab is associated with a higher risk of HBVr in such patients. The presence of anti-HBs may reduce the risk of HBV reactivation.

We compared the outcome of patients with resolved HBV received rituximab or infection who non-Rituximab immunosuppressive therapy and explored the utility of anti-HBs levels in these patients.

METHODS

We retrospectively collected data of patients who were on follow up at the HBV immunosuppression clinic in Singapore General Hospital from 2016 to 2022. The lower limit of quantification of HBV DNA was 10 IU/ml.

HBV reactivation (HBVr) was defined as a new detectable HBV

| Factors | Odd ratio (OR) | Confidence Interval (C.I) | P value |
|---------------------------------|-------------------|------------------------------|---------|
| Rituximab | 3.73 | 1.36-10.21 | 0.011 |
| Hepatitis B Surface Ab > 100 | 0.417 | 0.13-1.31 | 0.134 |

Table 2. Logistic regression analysis of anti-HBs levels in predicting HBVr, Rituximab exposed patients

| Factors | Odd ratio (OR) | Confidence Interval (C.I) | P value |
|---------------------------------|-------------------|------------------------------|---------|
| Hepatitis B Surface Ab > 100 | 0.012 | 0.001-0.785 | 0.038 |

Table 3. Logistic regression analysis of anti-HBs levels in predicting HBVr, Non – rituximab exposed patients

DNA from previously undetectable DNA.

HBV flare was defined as raised alanine aminotransferase of >3 times of upper limit normal in the presence of detectable DNA.

RESULTS

- A total 153 patients who did not receive prophylactic nucleoside analogue (NA) were included in the study.
- Of the 153 patients, 70 were male and the median age was 64 years (IQR 15).
- The median duration of follow-up following initiation of immunosuppressive therapy was 21.4 months (IQR 25.5).
- 112 patients received non-rituximab therapy, and 41 patients received rituximab therapy.
- 11 patients in each group developed HBVr. No patient developed HBV flare.
- HBVr resolved without needing NA in 8 patients in the rituximab group, and 7 in the non-rituximab group.

| Factors | Odd ratio (OR) | Confidence Interval (C.I) | P value |
|---------------------------------|-------------------|------------------------------|---------|
| Hepatitis B Surface Ab > 100 | 0.97 | 0.25-3.762 | 0.965 |

- Patients who received rituximab had higher risk of HBVr when compared with non-rituximab group, 26.8% vs 9.8%, p=0.01.
- Of the 41 patients receiving rituximab therapy, a baseline anti-HBs Ab of > 100 IU/ml was associated with lower risk of HBVr (6.7% vs 41.7%, *p*=0.02), as well as a lower odd ratio of HBVr (Table 2).
- Amongst patients on non-rituximab therapy, an anti-Hbs Ab of > 100 IU/ml was not associated with any statistically significant differences in HBVr.

CONCLUSION

Although the risk of HBVr was higher in patients receiving rituximab therapy, none of the patients developed HBV flare and HBVr resolved spontaneously in the majority. A hepatitis B surface antibody level of more than 100 IU/L may confer additional protection against HBVr in patients receiving rituximab therapy. Regular monitoring instead of prophylactic NA should be considered in this specific group of patients, as well as in patients receiving non-rituximab therapy.

