Association of Hepatitis B and C Virus Infections with Hematological Malignancies: An Epidemiological and Pathogenetic Perspective

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BACKGROUND & OBJECTIVE

Chronic hepatitis B virus (HBV) and hepatitis C virus (HCV) infections remain major global public health concerns. Increasing epidemiological evidence suggests that these viral infections may contribute to hematological malignancies, particularly B-cell neoplasms.

OBJECTIVE
To evaluate HBV and HCV prevalence among patients with different hematological malignancies, estimate associated relative risks, compare findings with population-based epidemiological data, and discuss potential pathogenetic mechanisms.

METHODS

Study design: Retrospective observational study conducted at the National Hematology and Transfusion Center, Baku, Azerbaijan.

Population: 797 patients with hematological malignancies, including non-Hodgkin lymphoma (NHL), acute lymphoblastic leukemia (ALL), chronic lymphocytic leukemia (CLL), multiple myeloma (MM), acute myeloid leukemia (AML), and chronic myeloproliferative neoplasms (CMPNs).

Screening: At diagnosis, all patients were screened for HBsAg and anti-HCV markers.

Statistics: Infection frequencies were compared with national epidemiological data. Relative risk (RR), odds ratio (OR), 95% confidence intervals, and p-values were calculated using SPSS. $P < 0.05$ was considered statistically significant.

RESULTS

- The strongest association was observed for non-Hodgkin lymphoma:
  HCV: approximately 4.3-fold increased estimated risk
  HBV: approximately 2.4-fold increased estimated risk
- Significant associations were also observed between HCV infection and chronic lymphocytic leukemia.
- CLL demonstrated significant associations with both HBV and HCV.
- No statistically significant associations were identified for multiple myeloma, acute myeloid leukemia, or chronic myeloproliferative neoplasms.
- Findings were described as consistent with published international studies.

KEY FINDING

NHL showed the strongest epidemiological association with chronic viral hepatitis.

HCV ≈ 4.3-fold | HBV ≈ 2.4-fold

PATHOGENETIC PERSPECTIVE

Proposed mechanisms include:

- Chronic immune stimulation
- Persistent inflammation
- Genomic instability
- HBV DNA integration
- HCV-mediated activation of activation-induced cytidine deaminase (AID) expression
- B-cell clonal expansion

Together, these mechanisms provide a biologically plausible framework for virus-associated B-cell lymphomagenesis, while epidemiological associations vary across malignancy types.

CONCLUSION

Chronic HBV and HCV infections appear to contribute to the development of selected hematological malignancies, particularly B-cell non-Hodgkin lymphoma.

Routine HBV and HCV screening at the time of hematological diagnosis may improve risk stratification and clinical management. Further multicenter prospective studies and molecular investigations are required to clarify viral oncogenesis and optimize preventive and therapeutic strategies.

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