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Clinical Observations and Interactions Between HBV Infection and B-NHL: Comments on High Prevalence of Hepatitis B Virus Infection in Patients with Aggressive B Cell non-Hodgkin's Lymphoma in China

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As a hematological physician in China mainly focus on malignant blood disorders, prevalent HBV infection in lymphoid neoplasm attracts my interests increasingly. Not only because of the well known fact that China is a highly endemic area with the prevalence of HBV infection of 7.2% [1] and approximately 170 million chronic carriers (350 million globally) [2], but also HBV is a lymphotropic virus whose infection and replication in lymphocytes might contribute to the development of lymphoproliferative disorders [3], disclosed by recent researches.

Non-Hodgkin's lymphoma (NHL) is a heterogeneous disease resulting from the malignant transformation of lymphocytes and includes multiple subtypes each with specific molecular and clinical characteristics [4]. Several studies have found a high prevalence of HBV infection in NHL, and some study indicated that the rate of positive hepatitis B surface antigen (HBsAg) was much greater among patients with B cell NHL (B-NHL) compared with T cell NHL (T-NHL) [5-6]. However, it is hardly to find research probes the differences rate of HBV between aggressive B cell non-Hodgkin's lymphoma (aggressive B-NHL) and indolent B cell non-Hodgkin's lymphoma (indolent B-NHL). Actually, the discussion in the latter is more closely connected with clinical practice since it emphasized on their functional relationship, which is promising to uncover therapeutic targets and prognostic factors. In my clinical observations, NHL patients with HBV apparent infection or carrier are not only predispositions to progress to a worse malignant degree and quickly deteriorate, but also easily suffer from complications especially liver dysfunction that limits anti-tumor drug selection. Therefore, uncovering the correlation between HBV infection and NHL aggressiveness appeals more attention for the specialist in this field.

A latest clinical analysis reported by Wang [7] which depicted a preliminary profile that answered my concerns recently. In this study, an enzyme-linked immunosorbent assay was used to test serum samples from the study for HBsAg, hepatitis B surface antibody (anti-HBs), hepatitis B e antigen (HBeAg), hepatitis B e antibody (anti-HBe), and hepatitis B core antibody (anti-HBc). Also, an impressive case number (373 aggressive B-NHL, 255 indolent B-NHL) was introduced in this study. According to WHO classification and cell differentiated origin, solitary plasmacytoma (SP, n=75) and multiple myeloma (MM, n= 327) was enrolled as well. Based on these materials, they observed a significant difference between aggressive B-NHL and other groups, evidence as aggressive B-NHL exhibiting significantly higher HBV prevalence than others. Notably, HBV positive cases were enriched in a subtype of aggressive B-NHL, which was named diffuse large B cell lymphoma-not otherwise specified (DLBCL-NOS). In turn, they observed HBV infection may have significant correlation with aggressive B-NHL for the evidence of the positive rates for all other HBV markers (HBeAg, anti-HBe, anti-HBc) were higher in the aggressive B-NHL subgroup than those in the other groups.

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In my opinion, though the shortage for this study was lacking of etiological discussion in term of these findings, which inspired attempts to be conquered through experimental studies to clearly determine the role of HBV infection in the pathogenesis of aggressive B-NHL, it was still constructive for effectively decreasing HBV-related events through early detection of HBV infection and anti-HBV drugs interventions.

Competing Interests

The author declare no competing interests.

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