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Hepatitis B and Hepatitis C Co-infections in CLL: Implications and Management Strategies

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Abstract

Hepatitis B virus (HBV) and hepatitis C virus (HCV) are classified as oncogenic human viruses. Chronic HBV and HCV infections are associated with higher risk of haematological malignancy development. Direct and indirect oncogenic mechanisms have been demonstrated for both HBV and HCV in several studies. HCV and overt/occult HBV infections in patients with oncohaematological disease constitute an impediment and a threat during immunosuppressive chemotherapy treatment. We review the HBV and HCV oncogenic mechanisms and the impact and the safety of antiviral treatment in patients with haematological malignancies.

Keywords: Leukemia; Occult hepatitis;Viral hepatitis

Introduction

Both the hepatitis B virus (HBV) and hepatitis C virus have been implicated in the pathogenesis of chronic lymphocytic leukemia. In support of this hypothesis are: epidemiological data describing associations between each virus and other hematological malignancies; the presence of viral replicative intermediates in peripheral blood mononuclear cells; and findings that each virus encodes for proteins with oncogenic potential. However, studies specifically designed to document the prevalence of HBV and/or HCV in patients with chronic lymphocytic leukemia are limited and have reported variable results [1]. This variability could, in part, reflect the small number of chronic lymphocytic leukemia subjects studied; different background prevalence of HBV and HCV in the general population; the use of serological markers alone for documenting evidence of viral exposure; and limiting viral nucleic acid testing to patient sera or plasma without including peripheral blood mononuclear cells or lymphocytes. Accordingly, in the present study, we tested a large number of North American adults who had been followed in a chronic lymphocytic leukemia clinic for evidence of previous or ongoing HBV and HCV infection using both serological and nucleic acid assays of patient sera and peripheral blood mononuclear cells. The results of the viral studies were correlated with patient clinical parameters, prognostic variables and survival times [2].

Chronic lymphocytic leukemia is a type of cancer characterized by the accumulation of mature lymphocytes in the blood, bone marrow, and lymphatic tissues. While CLL itself poses significant health challenges, patients with this condition may also face additional complications due to coexisting viral infections [3]. Two such infections are Hepatitis B and Hepatitis C, both of which can have a detrimental impact on the overall health and prognosis of CLL patients. This article explores the relationship between CLL and HBV/HCV infections, the potential risks involved, and the importance of effective management strategies.

HBV and HCV testing

HBV: All patient and control sera were tested for immunoglobulin G antibody to HBV core antigen by the CORE 1Mx system. Samples that tested positive were then tested for HBV surface antigen and antibody to HBsAg using third-generation monoclonal enzyme immunoassays according to manufacturer's instructions. A commercially available kit was used to extract DNA from sera and peripheral blood lymphocytes [4]. These extractions were then tested for HBV-DNA by real-time polymerase chain reaction (PCR) using the Light Cycler Real-Time PCR HBV, Absolute Quantification kit according to manufacturer's instructions.

HCV: Patient and control sera were tested for antibody to HCV using the HCV EIA version 2.0 kit; reactive samples were retested using the Innogenetic assay. Samples were considered to be anti-HCV positive when both tests were positive. RNA was extracted from sera and peripheral blood lymphocytes using the High Pure Viral Nucleic Acid Isolation kit. HCV-RNA testing was performed using the Cobas Taqman assay according to the manufacturer's instructions.

To prevent carryover contamination during PCR, each step of the procedure was performed in a separate room with dedicated equipment and directional flow from the beginning of the procedure to the end [5]. Negative control samples containing serum or water were also included in each extraction run, and an extra negative control containing water was included during each PCR run.

Hepatitis B and CLL

HBV is a DNA virus that primarily affects the liver. In CLL patients, the risk of acquiring HBV infection is increased due to several factors, including impaired immune function, decreased antibody production, and potential exposure to blood or blood products during treatments such as chemotherapy or blood transfusions [6]. Additionally, CLL patients who require immunosuppressive therapies are at higher risk of HBV reactivation if they have a previous infection. The consequences of HBV infection in CLL patients can be severe, leading to liver dysfunction, liver failure, and even death.

Hepatitis C and CLL

HCV is an RNA virus transmitted primarily through exposure

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to contaminated blood. Similar to HBV, CLL patients may be more susceptible to HCV due to their compromised immune system and potential exposure to infected blood products. Although the risk of HCV in CLL patients is relatively lower compared to HBV, the consequences can still be significant. Chronic HCV infection can lead to liver cirrhosis, hepatocellular carcinoma, and extrahepatic manifestations, all of which can complicate the management of CLL [7].

Clinical challenges and management

The presence of HBV or HCV infection in CLL patients presents several challenges for clinicians. First and foremost, these viral infections may remain asymptomatic for an extended period, leading to delayed diagnosis and treatment initiation. Moreover, the coexistence of CLL and viral hepatitis may result in overlapping symptoms such as fatigue, hepatomegaly, and abnormal liver function tests, making accurate diagnosis and differentiation crucial.

Management strategies for CLL patients with HBV or HCV infections involve a multidisciplinary approach. Regular monitoring of viral markers, liver function tests, and imaging studies can aid in early detection of viral reactivation or liver complications. In CLL patients with active HBV infection [8], antiviral therapy is recommended to suppress viral replication and prevent liver damage. Similarly, patients with chronic HCV infection may benefit from direct-acting antiviral (DAA) therapy, which has shown excellent efficacy in achieving viral eradication.

Preventive measures

Prevention plays a crucial role in mitigating the risks associated with HBV and HCV infections in CLL patients. Vaccination against HBV is strongly recommended for all CLL patients who are not immune, ideally before the initiation of CLL-specific therapies. Prophylactic antiviral therapy may be considered for CLL patients with a high risk of HBV reactivation, such as those with positive HBV serology or previous exposure [9].

Regarding HCV, universal precautions should be followed to minimize the risk of exposure to contaminated blood products. Screening for HCV should be performed in CLL patients with a history of blood transfusions, intravenous drug use, or other risk factors. Timely diagnosis and appropriate treatment of HCV infection can help reduce the burden on the liver and improve overall outcomes in CLL patients.

Discussion

The results of the present study do not support the hypothesis that HBV or HCV infection play an important role in the pathogenesis of chronic lymphocytic leukemia. Although the number of infected patients was small, the results also argue against either virus contributing to a more aggressive course of chronic lymphocytic leukemia. Finally, the results suggest that the prevalence of occult HBV and HCV infection are low in this particular patient population.

Although there have been numerous reports describing chronic HBV and HCV infections in patients with various hematological malignancies, there have been few studies specifically documenting the prevalence of these viruses in patients with chronic lymphocytic leukemia. Moreover, in the limited number of studies that have been published to date, conflicting results have been reported. In the present study, we did not observe a significant increase in the prevalence of either HBV or HCV markers relative to our healthy control population. The possible exception was a higher prevalence of anti-HBc in chronic lymphocytic leukemia patients, in whom the difference achieved a P value of 0.054. It should be noted, however, that the rate of false-positive anti-HBc testing is relatively high, particularly in the setting of negative HBsAg, anti-HBs and HBV-DNA results [10]. It should also be noted that our chronic lymphocytic leukemia population was older and included more males than the control population and, therefore, these demographic features would have favoured a higher overall prevalence of HBV infection in the chronic lymphocytic leukemia group. Whether the higher prevalence of anti-HBc could also be explained by differences in the ethnicity of patients and controls cannot be determined because ethnicity was not recorded in either group.

Regardless of whether HBV or HCV contributes to the development of chronic lymphocytic leukemia, it remains conceivable that either virus could alter the natural history or aggressiveness of the malignancy. Further supporting this contention were the survival times and mortality rates, which were similar in infected and uninfected patients. However, the numbers of subjects with evidence of HBV or HCV infection were low and, therefore, no definitive conclusion can be drawn regarding whether either virus alters the course of chronic lymphocytic leukemia.

Conclusion

Hepatitis B and Hepatitis C viral infections in patients with chronic lymphocytic leukemia pose significant challenges to both patients and healthcare providers. The compromised immune function in CLL patients increases their vulnerability to these infections, which can result in severe liver complications. Effective management strategies, including regular monitoring, antiviral therapies, and vaccination, are essential to prevent viral reactivation and minimize the associated risks. By addressing these challenges head-on, healthcare professionals can optimize the care provided to CLL patients and improve their overall prognosis and quality of life. The results of the present study do not support the hypothesis that HBV and HCV infections play an important role in the pathogenesis or course of chronic lymphocytic leukemia. However, larger studies including liver biopsies and age- and sex-matched controls are required to confirm these results.

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