

Passive transfer of hepatitis B surface antibodies from intravenous immunoglobulin

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To the editor,

Prior to initiating immunosuppressive therapy in autoimmune systemic disease, it is a requirement to screen for viral serology such as hepatitis B (HBV), hepatitis C (HCV) and human immunodeficiency virus (HIV). Intravenous human immunoglobulin (IVIg) is a blood product prepared and purified from human plasma used to treat a variety of autoimmune and inflammatory conditions. IVIg can transmit clinically important molecules, including antibodies. The passive transfer of viral antibodies from IVIg has been previously reported, but still with limited awareness. As so, passive antibody transfer may not be routinely considered in the interpretation of viral serology results after IVIg administration. We report a case of a 70-year-old female patient with granulomatosis with polyangiitis (GPA) with passive anti-HBV surface antibody transfer following IVIg infusions.

A 70-year old female patient presented at the emergency department with fever, anemia, acute renal failure, pulmonary infiltrates, sensorineural hearing loss and lower limbs mononeuritis multiplex. She was diagnosed with GPA and started treatment with systemic corticosteroids and Rituximab (RTX). The viral serology was screened before initiating treatment and were negative, including hepatitis B core antibody (HBcAc), hepatitis B surface antigen (HBsAg) and surface antibody (HBsAc). After 6 months she was started on IVIg monthly infusions due to persistent and very symptomatic mononeuritis multiplex. After the fifth IVIg infusion and due to persistent disease activity, a second RTX cycle was scheduled and viral screening was repeated. The screening revealed detectable HBcAc and HBsAc in the absence of HBsAg. She had no epidemiologic context for a possible HBV infection, normal liver function tests and was negative for HBV viral load. It was unlikely that the patient had asymptotically acquired and cleared hepatitis B infection in the period between the two laboratory tests. Therefore, the possibility that HBcAc appeared due to passive acquisition

from IVIg therapy was considered. The acknowledgement that HBV antibodies are transmitted to patients through IVIg has been described in some case reports, mainly in hematology patients¹. One cross sectional study with 80 patients receiving IVIg, 70 patients tested positive for HBsAc and 36 for HBcAc after IVIg. There was a progressive increase with each infusion in the percentage of patients testing positive for HBcAc among patients initiating IVIg².

This case shows that passive transfer of HBcAc can be a rare consequence of the administration of IVIg and does not confer an infectious risk. This is of particular relevance for rheumatology patients who are scheduled for future immunomodulatory treatment (such as rituximab), where previous hepatitis B infection can often be a barrier to treatment. Our patient performed the second RTX cycle with no delay and no complications were registered. Six months after IVIg treatment suspension, viral serology for HBV was repeated and was negative.

We did not find any rheumatology case reports or studies in the literature about passive transfer of viral antibodies from IVIg, so we consider that this case is of particular importance to divulge. Increased awareness and active consideration of the passive transfer of clinically significant antibodies from IVIg treatments will help prevent needless further investigations, unnecessary administration of anti-viral treatment, unwarranted referrals to gastroenterology services and withholding of essential immunosuppressive agents.

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