Letter to the editor: Reactivation of HBV triggered by SARS-CoV-2 in a patient with cirrhosis

To the editor,

HBV reactivation can occur spontaneously, but it is more frequently triggered by immunosuppressors, immunologic diseases, or transplantation. It can also be caused by coinfection with HCV or HDV, or even by elimination of HCV in patients treated with direct-acting antivirals. In patients receiving antivirals for the treatment of chronic hepatitis, reactivation can also be caused by low levels of the drug (due to lack of therapeutic adherence or interaction with other treatments) or new mutations of the virus.[1]

Reactivation is characterized by a sudden rise of HBV DNA, which can be followed by an elevation of alanine transaminase (ALT) levels (with or without bilirubin) several weeks after.[2] It should be treated with a nucleoside/nucleotide analogue (tenofovir or entecavir) as early as possible, regardless of ALT levels. Despite antiviral treatment, up to 25%–50% of patients may still develop severe hepatitis, hepatic failure, or even death.[3]

CASE REPORT

A 37-year-old male was admitted with asthenia, hyperoxia, and jaundice for the last 4 days. The patient had a cirrhosis caused by HBV chronic infection; he had never developed previous decompensations and was taking tenofovir-disoproxil-fumarate (TDF). Initial laboratory tests were made (Figure 1), and acute-on-chronic liver failure was diagnosed. He was taking TDF properly and denied consuming alcohol, drug abuse, or unprotected sexual intercourses. A complete study for acute liver injury was made (including viral and autoimmunity tests), and all results were normal.

On admission, the patient was tested for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), resulting positive. He assumed a risk contact 10 days before, but no breathing symptoms since then. DNA HBV was tested, and 4 million copies were detected (previously undetectable); treatment with entecavir was started. The virus was also tested for mutations but none of them were identified. Unfortunately, liver function worsened progressively as shown in Figure 1, leading finally to death 3 weeks later.

DISCUSSION

In our case, other more common causes were initially excluded: Medication adherence was appropriate, serology tests were negative, no new immunosuppressive
therapies were initiated, and viral mutations were excluded. For this reason, and judging by the temporary coincidence with infection by SARS-CoV2, we propose the coinfection with this virus as the cause of HBV reactivation. The underlying pathogenesis of transient reactivation of HBV in COVID-19 remains unclear, although it could be similar to HCV or HDV, causing reactivation of HBV replication.

In summary, we would like to highlight the singularity of our case report, as it is the first patient with cirrhosis with chronic infection of HBV who presumably developed a reactivation of HBV triggered by SARS-CoV-2.

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