Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Correspondence

Hepatitis B virus reactivation during severe acute respiratory syndrome coronavirus-2 infection

Dear Editor:

An outbreak of coronavirus disease 2019 (COVID-19) caused by the Omicron variant started in Taiwan in April 2022.[1] COVID-19 patients emerged abruptly after strategy modification from COVID-zero to co-existing. Here, we report a case suggesting that SARS-CoV-2 might cause hepatitis B virus (HBV) reactivation during COVID-19 infection.

A 70-year-old man had a history of idiopathic thrombocytopenic purpura (ITP) and chronic hepatitis B (CHB) infection. He had received prednisolone 5 mg per day for the past three years. CHB was followed up regularly at our hospital and was with normal alanine aminotransferase (ALT) and negative value of hepatitis B e-antigen (HBeAg). He did not receive nucleos(t)ide analogue (NA) in the past three years. This time he presented with fever and vomiting for one day. He had tested positive for the Omicron variant of SARS-CoV-2 through nasopharyngeal swab real-time reverse transcriptase-polymerase chain reaction (RT-PCR) and virus culture; these tests are carried out at the Taiwan CDC laboratory or in a designated laboratory at a hospital. He was isolated in a negative pressure ward at a government hospital in Pingtung, Taiwan. The initial biochemical analysis results were abnormal for ALT (268.0 IU/L) and bilirubin 2.0 mg/dL, and the HBeAg was positive. The concurrent HBV DNA titer was high (542,113 IU/mL) (Table 1). The baseline HBeAg and ALT level were examined 6 months and 1 month before COVID-19 infection, respectively. Molnupiravir was administered for COVID-19 and he was discharged after five days. The patient also received entecavir for the HBV reactivation. He did not receive any immunomodulatory agents or dexamethasone during the COVID-19. The serum aspartate transaminase (AST) and ALT gradually recovered to their normal values. The antibodies against hepatitis C and D were both negative.

One of the definitions of HBV reactivation is HBV DNA $\geq$10,000 IU/mL with unknown baseline.[2] Reactivation can be triggered in response to immunosuppressants such as corticosteroids. The patient received only low dose of prednisolone for years and without acute hepatitis before. However, HBV reactivation occurred during COVID-19 infection, and there have been other reports about this.[3] COVID-19 infection might cause liver injury in about 20 percent of patients.[4] CHB infection is prevalent at 10—15% in Taiwan[5] and COVID-19 affected at least one-sixth of the Taiwanese population between April to July 2022. The interaction between two viruses seems crucial, but the mechanism of HBV reactivation during COVID-19 infection remains elusive. However, it's our limitation to exclude that SARS-CoV-2 infection and prednisolone might also contribute to ALT elevation. We report this case so that physicians can pay more attention of HBV reactivation during COVID-19 infection and administer NA promptly.

Table 1 The characteristics of the COVID-19 patient before and during the COVID-19 infection.

	Before COVID-19	During COVID-19
AST (IU/L)	29.0	230.0
ALT (IU/L)	26.0	268.0
Creatinine (mg/dL)	0.6	0.7
Bilirubin (mg/dL)	0.8	2.0
Platelet count (x10^3u/L)	81.0	51.0
HBsAg (IU/mL)	—	4381.0
HBeAg	Negative	Positive
HBV DNA (IU/mL)	—	542,113

Note: COVID-19: coronavirus disease 2019; AST: aspartate aminotransferase; ALT: alanine aminotransferase; HBsAg: Hepatitis B surface antigen; HBeAg: Hepatitis B e-antigen; HBV: hepatitis B virus.

Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

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