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CORRESPONDENCE

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Suppression of hepatitis C virus replication during COVID-19 infection

An outbreak of COVID-19 caused by Delta variant occurred in Southern Taiwan on May 2021. It is believed to have originated from two COVID-19 cases from Peru, and seventeen patients have been infected with Delta variant in the event. Here, we report a case suggesting that SARS-CoV-2 might suppress HCV during COVID-19 infection.

A 70-year-old woman presented with fever and cough for 2 days. She had tested positive for Delta 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 hospitals. She was isolated in a negative pressure ward at a government hospital in Pingtung, Taiwan. The initial biochemical analysis results were abnormal for aspartate aminotransferase (AST) (47 IU/L), and the hepatitis C antibody (Anti-HCV) was positive. This was the first time she was diagnosed with HCV infection. The concurrent HCV RNA titer was low (140 IU/mL) (Table 1). Remdesivir was administered for COVID-19 infection and she was discharged after 2 weeks (Figure 1). HCV RNA titer was rechecked 8 weeks after COVD-19 infection and HCV RNA titer elevated more than 10,000 times (1,675,038 IU/mL), but the serum AST and alanine aminotransferase (ALT) was normal. HCV Genotype 1b was detected and she received direct antiviral agents (DAA).

In late 2019, COVID-19 emerged in Wuhan, China, and resulted in a formidable outbreak that expanded globally and caused huge health and economic burden. COVID-19 causes damages to many extrapulmonary organs. The first case of COVID-19 in Taiwan was diagnosed on January 2020 and the mortality rate was 1.3% at that time.[1] Taiwan had few domestic cases until small outbreaks occurred in May 2021.[2] The dominant variants were Alpha and Beta, and Delta variant cluster emerged last June 2021. Delta variant emerged recently in India and spread gradually to other countries. It has now become the dominant cause of COVID-19 in the world.

SARS-CoV-2 might cause hepatitis B virus (HBV) reactivation[3]; however, the interaction between HCV and SARS-CoV-2 has rarely been described. One case report described no significant change in HCV RNA titer after COVID-19 infection in a nonhospitalized patient.[4] Here, we report a case that HCV RNA might be significantly elevated after COVID-19 infection in hospitalized patients, and HCV viremia flare up might occur after COVID-19 infection. In the previous literature, the mean change of viral load was less than 1 $\log_{10}$ per year and rarely exceeded 2 $\log_{10}$.[5] This case changed more than 4 $\log_{10}$ within

TABLE 1 The characteristics of the COVID-19 patient before and after the COVID-19 infection

	Pretreatment	Posttreatment[a]
AST (IU/L, mean [SD])	47	29
ALT (IU/L, mean [SD])	30	14
Bilirubin (mg/dL)	1.6	1.5
Platelet count ($\times 10^3$ U/L, mean [SD])	39	70
Albumin (g/dL)	3.1	3.4
Creatinine (mg/dL)	1.0	1.0
HBsAg	Negative	Negative
HCV RNA (IU/mL)	140	1,675,038

Note: COVID-19: coronavirus disease 2019; AST: aspartate aminotransferase.
Abbreviations: ALT, alanine aminotransferase; HBsAg, hepatitis B surface antigen; HCV, hepatitis C virus.
[a]Eight weeks after the admission.

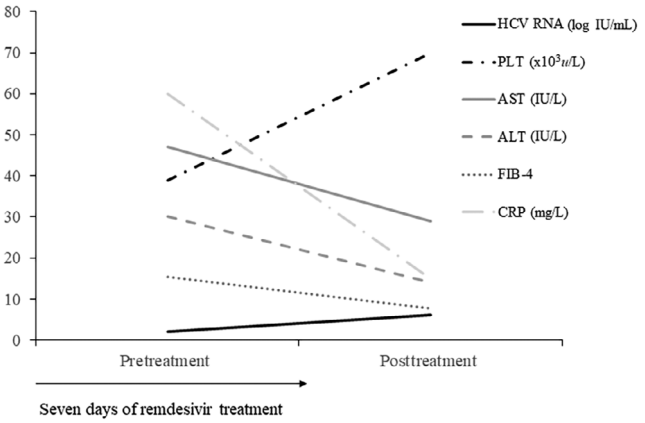

FIGURE 1 The pretreatment and posttreatment biochemical data of the patient. ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein; FIB-4, fibrosis-4 index; HCV, hepatitis C virus; PLT, platelet count

2 months, and it was unlikely to be a natural fluctuation. The patient received 7 days of remdesivir treatment, and the HCV RNA was checked after 2 days of remdesivir treatment. There was no evidence that remdesivir would suppress HCV in the current literature. We suppose that SARS-CoV-2 might suppressed HCV during COVID-19 infection, but further study is needed. The limitation to our case was that the baselines status of HCV RNA before COVID-19 infection was unknown.

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CONFLICT OF INTEREST

All authors declare no conflict of interest.

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