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LETTER TO THE EDITOR

COVID-19-associated liver injury (COVALI): role of hepatologists

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We read with great interest the article by Li and colleagues [1]. COVID-19 caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), which has become a global pandemic. Elevated hepatic enzymes are common in patients with COVID-19, occurring in approximately 15–65% of SARS- CoV-2-infected individuals, particularly in severe cases. Liver biopsy results in patients with SARS-CoV-2 have been characterized by non- specific findings, including steatosis, mild lobular and/or portal inflammation, and vascular pathology. It still remains unknown SARS-CoV-2 can directly infect hepatocytes and/or cholangiocytes. Mechanisms underlying COVID-19-associated liver injury (COVALI) are multifactorial and related to direct cytopathic effect of SARS-CoV-2, cytokine storm, hypoxia, abnormal coagulation, an administrated drug, and preexisting chronic liver diseases (CLD). The article extensively reviewed plausible mechanisms of COVALI. Overall, 2–11% of patients with COVID-19 were reported to have underlying CLD. Patients with cirrhosis have high rates of hepatic decompensation, acute-on-chronic liver failure and death from respiratory failure following SARS-CoV-2 infection and should be prioritized for COVID-19 vaccination. The Asia Pacific Association for the Study of the Liver (APASL) launched a pan-Asia collaborative study "APASL COVID Liver Injury Spectrum (APCOLIS). APCOLIS study showed that the outcome was poor with Child–Pugh score 9 or more among 43 cirrhosis those exposed to SARS CoV2 infection [2]. According to data on 745 patients with CLD and SARS-CoV-2 from a UK hospital network. Baseline child stage and alcohol-related liver disease are independent risk factors for death from COVID-19 [3]. Accumulating evidences have proposed that metabolism-associated fatty liver disease may be a risk factor for COVID-19 severity. Liver transplant recipients do not appear to have an increased risk of mortality following SARS-CoV-2 infection compared with the matched general population. The effects of COVID-19 on underlying CLD require detailed evaluation, with further research warranted in this area. Remdesivir, an approved drug for patients with COVID-19, may have hepatotoxic effects. Remedesivir should not be used for COVI-19 patients with baseline liver enzymes more than five times the upper limits or decompensated cirrhosis. If patients with COVID-19 receive dexamethasone or tocilizumab, HBV reactivation should be ruled out. International academic societies have recommended guidance outlining the evidence to date regarding the management of patients with COVID-19 and liver disorders, and CLD under the COVID-19 pandemic. The percentages of scheduled visits to screen HCC or varices in chronic hepatitis C after sustained virologic response declined rapidly after COVID-19 became pandemic in Japan [4]. We should avoid delay of HCC surveillance in CLD patients. If liver enzymes are commonly elevated in patients with COVID-19, we should first evaluate for the presence of other causes including acute viral infections (HAV, HBV, HEV, etc.) or pre-existing

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CLD, and strictly follow-up liver enzyme values. Hepatologists must play a certain important role in management of COVALI in collaboration with respiratory medicine and infectious disease specialists until efficacious SARS-CoV2 vaccines or effective medications are globally available [5].

Declarations

Conflict of interest The authors declare that they have no conflict of interest.

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