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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Conflicts of interest
The authors disclose no conflicts.

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Reply. We thank Dr Jindal for comments regarding our article on liver injury and COVID-19. Herein we provide a response to the 3 points raised.

First, we entirely agree with Dr Jindal's assessment of our study. We show that severe liver injury reflects severity of COVID-19 vis-à-vis therapeutic strategies aimed at treating the disease (COVID drugs) and/or at keeping the patient alive (eg, vasopressors). Notably, severe liver injury only occurred in a minority of patients (3%). In the absence of liver biopsy (which would not have been justified) that would confirm ischemic or drug-induced injury, we had to rely on associations and temporal relationships as is often done clinically. Autopsy studies of patients who died from COVID-19 have shown histopathologic changes in the liver consistent with disturbed intrahepatic circulation[1] and upregulation of proteins involved in fibrosis, necrosis, fatty acid oxidation, and other markers of immune activation, with very little viral replication.[2] Unfortunately, these studies lack a clinical-pathologic correlation.

Second, we could not find an association between mortality by COVID-19 and the presence of preexisting liver disease. In our study, which included consecutive patients admitted with COVID-19, only 38 (4.5%) had chronic liver disease without cirrhosis and 13 (1.5%) had cirrhosis. This is very similar to rates reported in another large cohort from an academic center with only 31 (1.4%) patients with cirrhosis.[3] As pointed out in that study, the lack of association between chronic liver disease/cirrhosis and mortality could be caused by these low numbers. Additionally, and as mentioned by Dr Jindal, international registries that include large numbers of patients with cirrhosis have reported worse outcomes mostly in patients with decompensated cirrhosis.[4] Only 3 patients in our cohort had decompensated cirrhosis. Interestingly, in a careful analysis of patients with cirrhosis admitted during the initial stages of the COVID-19 pandemic, mortality in patients admitted for complications of cirrhosis (without COVID-19) was not significantly different from that of age- and gender-matched patients with cirrhosis admitted with COVID-19 but was significantly higher than that of patients admitted with COVID-19 (without cirrhosis).[5]

Third, in our definition of ischemic liver injury, we required that a patient be on vasopressors for at least 2 days. We defined the ischemic state as 2 consecutive days on vasopressors to avoid capturing patients who may have required transient vasopressor use peri-intubation. We agree that patients who received vasopressors are more severely ill and have a higher mortality. The multivariate analysis in Table 3 of our study has the objective of identifying different predictors of death with the main objective of examining whether significant liver injury was an independent predictor of death. It was not.

The comments by Dr Jindal highlight our 2 main conclusions, that significant liver injury in patients hospitalized with COVID-19 mainly results from concomitant processes/drugs during hospitalization; and that this significant liver injury does not result in liver insufficiency and is not an independent predictor of death.

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Conflicts of interest
The authors disclose no conflicts.

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Network Meta-analysis of Ulcerative Colitis Pharmacotherapies: Carryover Effects From Induction and Bias of the Results

Dear Editor:

In their network meta-analyses (NMAs) of treatments for ulcerative colitis (UC), Singh et al[1] did not take into