

Elevated Liver Chemistries in COVID-19—Is It Not a Concern?

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We read with great interest the study by Hao et al. on liver enzyme elevation in coronavirus disease 2019 (COVID-19) (1). The authors have collated data from a large number of COVID-19 patients from multiple centers across China. The authors have shown that around 28% of patients with COVID-19 have deranged liver function tests (LFTs) at admission, and furthermore, 32% of patients developed elevated liver chemistries during illness. Male patients, overweight individuals, and smokers are at higher risk of developing enzyme elevation. Another interesting observation made in this study was that deranged LFT has no impact on the outcome of COVID-19 patients. However, there are some concerns that we would like to discuss.

The incidence of elevated liver chemistries in COVID-19 patients at the time of admission is 24.1% (95% confidence interval [CI], 20–28.8) in adults and 17.8% (95% CI, 9.9–29.8) in pediatric patients (2). In the recent meta-analysis comprising 107 studies, it has been shown that non-survivors and severely infected COVID-19

patients had a higher risk of presenting with a deranged LFT. Elevated liver chemistries also aid in predicting the outcomes of COVID-19, which is in contrast to the observation made in the current study (3,4). It would be interesting to know the severity of COVID-19 in controls and individuals with deranged LFTs.

The authors further state that compared with the control group, the levels of aspartate amino transferase, total bilirubin, lactate dehydrogenase, blood urea nitrogen, C-reactive protein, and procalcitonin were higher in the deranged LFT group. C-reactive protein, aspartate amino transferase/alanine amino transferase, and lactate dehydrogenase are important markers of disease severity and predict the outcomes in COVID-19 (4,5). Despite this correlation in the current study, patients with deranged LFTs had no difference in outcomes. We believe that because more than half of the patients were still under treatment/hospitalized at the time of publication, concluding that the deranged LFT has no impact on the outcome of COVID-19 patients is precipitous.

The prevalence of chronic liver disease (CLD) among COVID-19 is ~4%. The authors have reported that COVID-19 patients with pre-existing CLD had outcomes similar to those without pre-existing CLD, which is in congruence with the observation made in the recently published meta-analysis. Still, a plausible explanation for this is not yet clear (2).

Liver injury in COVID-19 is multifactorial. Liver involvement can be because of direct cytopathic effect of virus or severe inflammatory response, uncontrolled immune reaction, sepsis, or drugs. Given that 86% of patients received some antiviral drugs, it would have been more interesting if

a detailed analysis of drug-induced liver injury was described. More prospectively designed studies collaborating data of LFTs, liver histology, the severity of COVID-19, and viral load of severe acute respiratory syndrome corona virus-2 may help to solve the debate of the precise cause of elevated liver chemistries.

CONFLICTS OF INTEREST

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