Response to M. Amin: COVID-19 and the liver: overview

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Dear Editor

I read with interest the review by M. Amin [1]. It has undoubtedly clarified how coronavirus disease 2019 (COVID-19) interacts with the liver, especially in patients with chronic liver disease and a history of liver transplantation. However, there are several concerns regarding the author's comments on COVID-19 and chronic liver disease.

The author proposed that COVID-19 in such patients could exacerbate hepatic injury, thus leading to hepatic decompensation. However, this statement has the potential to oversimplify matters. The review failed to mention that although there may be background liver injury, other factors can lead to poorer survival outcomes. A more holistic approach is required. Marjot and colleagues suggested even though COVID-19 patients with cirrhosis are at higher risk of mortality, invasive ventilation, ICU admission, and hospitalisation, in a severity-dependent fashion, most patients died of COVID-19 lung disease (71%) instead of liver-related causes (19%) [2]. Although liver injury might have occurred, it is insufficient to be the main cause of mortality. The role of systemic inflammation and multi-organ involvement should also be discussed since patients with chronic liver disease are more likely to have severe COVID-19 [3].

This is even more significant if we take into account the positive correlation between new requirements for renal replacement therapy, and the severity of cirrhosis in COVID-19 patients ($P < 0.001$) [2]. This can indicate COVID-19-induced kidney injury (either through viral-mediated reactions, or drug use), and hepatorenal syndrome. This can be a significant factor leading to acute decompensation and death.

The author is also advised to consider the correlations between worsening liver function and its impact on the lungs, since exacerbation of lung disease can directly induce death already, without acute decompensation of cirrhosis. Examples include hepatopulmonary syndrome and portopulmonary hypertension [4]. Both mechanisms involve, to varying degrees, the retention of vasodilators due to inadequate removal by the liver, and vascular remodelling [4].

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Conflicts of interest

There are no conflicts of interest.

References

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