Answers to Reviewers' Comments

We thank the reviewers for their thorough critique and insightful comments. We have addressed each of the points raised during the review process and introduced changes in the revised version of the manuscript accordingly. We believe that the manuscript has been substantially improved after these modifications. We hope that the revised version be now acceptable for publication in World Journal of Gastroenterology.

Our answers to the reviewers` concerns and comments are provided below.

Comments of Reviewer #1:

Comment 1: The study is interesting and well performed; the clinical significance needs to be confirmed by additional studies. I have some issues to mention: Question1: We didn't know the patient's alcohol consumption, and didn't see the patient's detailed data. The hepatic steatosis may be caused by alcohol, non-alcohol or hepatitis. Pleasea give a brief explanation.

Answer: Since it is a systematic review from autopsies published in the literature, we did not have access to relevant clinical data such as alcohol consumption. In fact, Table 1 summarizes all the clinical information provided by the included studies. Therefore, we mentioned it in the limitations of the study as follows:

"Finally, since it is a systematic review from autopsies, relevant clinical information to interpret steatosis (such as alcohol consumption or body mass index) was not available."

Comment 2: Some patients have obesity, diabetes and hyperlipidemia. This phenomenon can lead to nonalcoholic fatty liver disease (NAFLD). NAFLD is

described as a presence of non-alcoholic hepatic steatosis and liver tissue inflammation. The hepatic steastosis may be caseud by NAFLD or COVID-2019. Pleasea give a brief explanation in your disscusion.

Answer: We agree with this comment and it was previously mentioned in the discussion as follows:

"The most frequent histopathological finding was steatosis. This prevalence is higher than the general population[26]. This can be partially explained by the baseline characteristics of the population. Admitted COVID-19 patients suffer from more severe disease and more frequently exhibit chronic diseases (i.e. diabetes mellitus, age, hypertension, obesity, and cardiovascular disease), which are also associated to hepatic steatosis[27]. However, existing data suggest that SARS-COV-2 may affect lipid metabolism[28]. In general, all viruses alter lipid synthesis and signaling in host cells as they highjacks and utilizes the cellular machinery to produce lipids for their envelope. Also, it has been shown that COVID-19 patients have elevated serum levels of fatty acids and infection with other SARS virus determine long lasting alterations in lipid metabolism[29]. It has been shown that metabolic-dysfunction associated fatty liver (MAFLD)[30] condition is independently associated with a higher risk of severe COVID-19 (odds ratio 2.67)[31]. Also, advanced MAFLD (i.e. fibrotic diseases) have been als shown to be associated to a more severe disease irrespective of metabolic comorbidities. Since the liver host a significant mass of innate immune cells, hepatic release of proinflammatory cytokines may contribute COVID-19 severity[32]. Also, some authors suggest NAFLD progression could be accelerated or exacerbated by COVID-19[32]."