Major Comments		
Comment	**Response**	**Text Adjustment**
Reviewer 1		
There is a vast literature on the co-infective potential of SARS-COV-2 with hepatitis B infection (DOI: 10.3851/IMP3382) I suggest including notions on this topic. Is there any link between COVALI and hepatitis B?	We appreciate this suggestion and think it is important to comment on the relationship given the amount of existing literature. We have 1) included a statement in the recommendations for management patients with chronic liver disease, as this caveat is addressed in the AASLD guideline statements and 2) added the a statement to the pathophysiology section on chronic liver disease and drug induced liver injury	Added text - (1) Patients with chronic hepatitis B may be at particularly high risk both due to risk of severe infection and viral reactivation when receiving immunosuppressive therapy" - (2) In a combination of these, SARS-CoV2 infection treated with corticosteroids or tocilizumab has been showed facilitate reactivation and accelerate liver injury in patients with chronic hepatitis B."
Lines 250-257 A large variety of additional cytokines have been reported to be released during SARS-COV-2 infection. The authors can check PMID: 32961074	We appreciate this comment and agree with the reviewer that there are many more inflammatory markers that are known to be elevated in during SARS-CoV-2 infection. However, there is a paucity of data that discuss many of the less common cytokines noted in the suggested paper (IL-7, granulocyte-colony-stimulating factor, IP-10, monocyte chemoattractant protein-1 (MCP-1)) in clinical contexts/liver disease. To address thesis comment we amended our original statement to demonstrate why these values were selected.	Edited Text - It is well established that severe COVID-19 infection induces systemic inflammation and that concentrations of several clinically evaluated inflammatory markers are increased in patients with COVID-19, such as D-dimer, C-reactive protein, procalcitonin, ferritin, and interleukin-6 (IL-6) [92-95]."
Lines 400-407 as stated, a physiological systemic inflammation occur during advanced pregnancy. At the same time, during pregnancy, the circulating levels of alpha1-antitrypsin, which is a plasma protease inhibitor released by the liver, has been reported to increase (DOI: 10.3389/fcell.2020.550543). An impairment of this phenomenon in the liver might make pregnant females more prone to SARS-CoV-2 infection, as reported (doi: 10.4254/wjh.v13.i10.1367). In this context, a connection between SARS-CoV-2, COVALI and an alpha1-antitrypsin release impairment cannot be excluded. Authors should at least briefly include these notions and aforementioned supporting references.	We truly appreciate this reviewer's thoughtfulness in providing feedback and find this to be an interesting concept raised by the reviewer. We read the suggested articles and did additional research on the topic. Alpha 1 anti-trypsin may inhibit SARS-CoV-2 infection and has been shown to have cytoprotective role in vascular endothelial during pregnancy. However, as mentioned, A1AT levels increase during pregnancy which would argue a protective effect regarding COVID-19 infection during pregnancy. The reviewer posed that an impairment in the mechanism that increases A1AT would be a potential link in this cohort. We found no data that suggests COVID-19 infection itself reduces A1AT concentrations or its release. We did find data that A1At is reduced during pre-eclampsia which is	Added text - A potential link to the increased risk is alpha-1-antitrypsin, an enzyme that can inhibit SARS-CoV-2 infection and protects endothelial cells from oxidative stress during pregnancy, which is reduced in seen in pregnant patients with pre-eclampsia [128, 131].

	consistent with literature that shows increased risk of COVID-19 and COVALI in pregnant patients with pre-eclampsia. We have included a sentence on this relationship to address this comment.	
Reviewer 2		
In the abstract (line 32), please specify whether the Covid-19-associated liver injury is the occurrence of a newly diagnosed hepatic biochemical injury during a Covid-19 infection or it is the exaggeration of an old hepatic injury after contracting Covid-19. The definition in its current form is a little bit confusing and should be better stated	We apologize for any confusion and appreciate this feedback. The text has been updated to clarify the definition (see right). We chose this wording rather than calling it "new" as to not imply patients with previous liver injury or diagnosed conditions that cause liver injury would be excluded from the definition.	Edited text: - Liver injury is an increasingly recognized extra-pulmonary manifestation of SARS-CoV-2 infection (COVID-19). COVID-19 associated liver injury (COVALI) is a clinical syndrome encompassing all patients with biochemical liver injury identified in the setting of SARS-CoV-2 infection"
The introduction is well-written but deviated to some extent from the main focus of this paper. It contains a lot of general information about Covid-19, which is slightly irrelevant to the aim of this manuscript. I suggest summarizing the general information about Covid-19 in a few lines and focusing the introduction on the Covid-19-associated liver injury.	We appreciate this feedback and took steps to shorten and narrow the scope of the introduction. We reduced the total word count by 30% by removing the portions highlighted on the right.	Removed text: - *At the end of 2019 and in the early months of 2020, the medical community was called to action when what was thought to be an isolated outbreak of viral respiratory infections in China began to rapidly circulate the globe [1-3] . Since this time,* - *During early stages of the current pandemic scientists relied on knowledge about previous Coronavirus outbreaks to inform recommendations during COVID-19. However, it became clear that COVID-19 was transmitted more rapidly and often asymptomatically making detection and containment difficult [10-12]. Clinical practice has evolved continually and in parallel with ongoing scientific discovery about the disease.* - *…since 2000 there have been two well-known outbreaks of Coronaviridae associated with severe respiratory syndromes (SARS and MERS)..*
In the introduction section (lines 79 and 80), I slightly disagree with this statement. Promoting open access publishing and research equity is done by publishing in open access journals (as you are doing by submitting to WJG). Focusing on papers published in open access journals only is a weakness in this review because it may lead to missing important papers published in journals with	Thank you for this feedback. Fortunately, most of the literature related to COVID-19 has been open access since the onset of the pandemic; one study of 5611 papers on Pubmed found of 97% were freely available to the public (PMC7438966). However, we agree that this statement may be viewed as a weakness outside of that context and have removed this statement from the text.	Removed text: - *Literature referenced in writing this review will be majorly from open access sources to promote equity in access to vital health information.*

subscriptions.		
In the clinical consideration paragraph (line 101), please clarify whether the liver enzymes peak "at least five times higher" or "less than five times higher" than the upper limit in patients with Covid-19-associated liver injury. The current sentence is unclear.	We thank the reviewer for this question - The liver enzyme elevation is COVID-19 is typically non-severe and enzymes do not reach 5 times the upper limit of normal (less than five times the ULN). We have updated the text to clarify this clinical feature.	Edited text - Most studies report mild liver injury with liver enzymes that peak at values less than five times the upper limit of normal [38-42].
In the paragraph Clinical Cases (lines 318-345), I believe it is not necessary to give a summary of each case. I suggest focusing on the main findings and writing a briefer paragraph. The manuscript is interesting and informative, but also long paragraphs are not really desired.	We appreciate this comment from the reviewer, however, we believe the summaries of the cases are important to the text to describe complicated cases of COVID-19 that mirrored pregnancy related liver disease. We believe that the cases demonstrate the importance of critical thinking in obstetric patients with liver injury which is an objective in this article. To avoid explaining all the cases in the table, we only gave thorough details for cases that improved without delivery, as these were most consistent with COVALI. Please note, we will remove the details at the request of the editors if they feel it is extraneous for the purposes of the journal.	N/A
The conclusions typically are a 6 to 7-line paragraph that gives recommendations for future research perspectives or clinical suggestions for improving the practice. A conclusion of 1-page length is not acceptable. Please summarize it.	Thank you for this comment and drawing this to our attention. In writing our conclusions, we aimed summarize and assess our findings, highlight key takeaways, and discuss future directions. However, after reading conclusions from similar articles published by *World Journal of Gastroenterology*, we recognize the content of the conclusion should be reduced. The conclusion was reduced by over 50% (352 words to 170 words) by removing the following sections of the text:	Removed text - *A plethora of data connects inflammation with liver injury in COVID-19 in the general population. In the obstetric population, similarities between extrapulmonary manifestations of COVID-19 with pre-eclampsia also support an inflammatory driven disease.* - *We have used available data to demonstrate the significance of COVALI in obstetric population. In addition to increased risk of severe disease and mortality, in the obstetric setting COVALI is associated with worse outcomes in the antenatal and immediate postpartum periods [114]. Management also differs between COVALI and perinatal liver diseases, where-in liver injury secondary to COVID-19 can improve with supportive management and does not require prompt delivery.* - *Our conclusions are somewhat limited by lack of high-quality data on liver injury in pregnant*

		individuals with COVID-19. Limited data may be due to ethical policies that qualify pregnant individuals as members of vulnerable populations which can create a barrier to inclusion in many clinical studies. It may also be due to the fact COVALI can mimic serious gestational conditions and therefore may be under-diagnosed. However, it is unlikely to be due to low prevalence as evidenced by early studies reporting elevated liver enzymes is 5-10 times more common in perinatal population than the general population.
Reviewer 3		
The abstract is lack of the major point of the review conclusion. The readers may feel not know the key conclusion after reading the abstract.	The authors thank the reviewer for this feedback. To better represent the conclusions of our review, the second half of the abstract has been edited accordingly.	Edited + Added text: - This manuscript reviews: clinical features of COVALI, leading theories of COVALI, and existing literature on COVALI during pregnancy, a topic not widely explored in the literature. Ultimately, we 1) synthesized data from the general and perinatal populations that demonstrates COVALI to be a hepatocellular transaminitis that is likely induced by systemic inflammation and that is strongly associated with disease severity and poorer clinical outcome and 2) offered perspective on approaching transaminitis in the potentially COVID-19 positive patient in the obstetric setting.

Minor Comments and Grammar

- Reviewer 1:
  - Line 30 COVID-19 should be mentioned as Coronavirus disease 2019 (COVID-19) when mentioned for the first time → Completed ("The Severe Acute Respiratory Syndrome -2 Coronavirus (SARS-CoV-2) disease pandemic (COVID-19)")
  - Line 35 better pregnant females → The authors did not agree with this change as to use language that respects all individuals regardless of how they identify in regard to their gender.
  - Line 44 SARS-2-CoV should be replaced with SARS-CoV-2, while its complete name is severe acute respiratory syndrome coronavirus 2 → Completed
  - Line 48 coronaviridae should be in italic style → Completed
  - Lines 54, 60 as well as many others: unnecessary spaces between sentences should be removed → Completed
  - Lines 65, 66 given the topic of the work, reproductive system as a possible infective target of SARS-COV-2 infection should be mentioned DOI: 10.1186/s43043-020-00046-z → Completed with different references
  - Line 99 it can be replaced with COVALI → The authors did not agree with this suggestion because sentences should not start with an acronym.
  - Line 123 as well as other acronyms should be carefully checked and mentioned with their complete name the first time being mentioned → Completed (alanine aminotransaminase – ALT)

- Line 201 I would mitigate this point, this is not completely true DOI: 10.1038/s41575-021-00426-4 → Adjusted statement ("However, the understanding of SARS-2-CoV hepatotropism of is still evolving."
- Lin 244 In vivo should be in italic style. Please revise the entire manuscript for additional style errors → Completed
- Lines 233 and 250 please uniform the style → Completed (Adjusted a space for IL-6)
- In line 250 is should be IL-6 → Completed (Adjusted a space for IL-6)
- Lines 376-377 please revise the typo after the word "thus" → Completed

- Reviewer 2
  - In the whole text, please correct the name of the Covid-19 virus from "SARS-2-CoV" to "SARS-CoV-2". The first abbreviation is incorrect. → Completed
- Reviewer 3
  - "Covid associated liver injury (COVALI)" was used in the title and most places of the manuscript, but in some places and in figure 1, the author used COVID-19-induced liver injury. It could be better to use one, or please explain the difference. → Completed
  - Line 117, improve grammar(period?) → Completed