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Peer Review File
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Round 1:

Reviewer A

We want to thank the reviewer for taking the time to review our article. It is sincerely appreciated. Here are our responses:

Comment 1: 1. As the author has shown, collagen disease-like findings and the appearance of autoantibodies such have been reported after the diagnosis of COVID-19. So please show whether there was positive of the autoantibodies associated with collagen diseases other than SLE and increase of immunoglobulins in the present case. Also, please show how you differentiated the overlap of other collagen diseases.
Reply 1: The other autoimmune disease tests that were ordered have been added and shown. Furthermore, the dsDNA and the low complement levels added to the diagnosis of SLE
Changes in the text: Page #2 Lines 28-30

Comment 2: Please show changes in complement, dsDNA antibodies to reveal changes in disease activity of SLE over time.
Reply 2: C3 and C4 levels on admission and prior to discharge have been noted
Changes in the text: Page#2 Lines 30-31 and 41-42

Comment 3: The results of a renal biopsy should be shown by figure
Reply 3: Unfortunately, the actual biopsy images were not obtainable as it was done at at a lab that the slides were sent to, despite the authors' best efforts to get this image
Changes in the text: None

Comment 4: Is SLE the only possible cause of chronic kidney disease in the present case? Please show whether the present case has causes of chronic kidney disease such as potential diabetes or hypertension, a history of streptococcal infection, or positive MPO-ANCA or PR3-ANCA.
Reply 4: Our patient did have essential hypertension, which was likely the cause of our patient's chronic CKD. His A1c was normal. We hypothesize that the CKD was likely from hypertension (but there were no prior tests to confirm this) and the post-COVID exposure resulted in his SLE. Another possibility is that he had SLE and his SLE flareup only after COVID exposure. However, in the last paragraph in our discussion, we mention this as a possibility and state how people with underlying autoimmune diseases typically have a severe response to COVID infection, which our patient did not

.An ANCA test was done and was <1: 20, and as such a MPO-ANCA or a PR3-ANCA was not deemed necessary.
Changes in the text: Page 2, Line 27

Comment 5. Please show the diagnostic criteria when this case is diagnosed as SLE.
Reply 5: We used the 2019 EULAR/ACR criteria. The details have been added in a a new paragraph
Changes in the text: An entire new paragraph has been added in page 3

Reviewer B

We want to thank the reviewer for taking the time to review our article. It is sincerely appreciated. Here are our responses:

Comment 1: This patient's acute presentation is not consistent with SLE. He has a positive ANA 1:1280 NS, which is fairly nonspecific, and is certainly adequate for an SLE diagnosis.
Reply1: We have added other lab parameters, particularly the Anti dsDNA, low C3 and C4 levels, and criteria we have used to diagnose SLE in our patient.
Changes in text: Page#2 Lines 30-31 and 41-42 and Page 3, Lines 53-62

Comment2: He has inflammatory arthritis, which has a broad differential, and is nonspecific as well. Kidney biopsy showed FSGS, with no immune deposits. This is not consistent with lupus nephritis.
Reply2: There have been several case reports that have shown collapsing FSGS variant in SLE patients. Putting the biopsy results, the lab values and the clinical presentation lead to the diagnosis of SLE
Changes in text: None

Comment 3: The case report is missing several important elements of diagnostic evaluation, including urine studies, cardiovascular studies etc. Overall, I do not see enough evidence of SLE or another autoimmune etiology of his kidney failure.
Reply3: We have provided more lab values and the criteria for diagnosis of SLE as stated in our reply to comment 1. Urine studies are mentioned in line 27 with the protein to creatinine ratio.
Changes in text: Page #2 Lines 28-30, Page#2 Lines 30-31 and 41-42 and an entire paragraph in page 3

Round 2:

Reviewer A

Comment 1: Was the dyspnea attributed to the renal component or were there signs of serosal effusions? Did X-ray reveal anything? Ultrasound?
Reply 1: The dyspnea was attributed to the fluid overload from the kidney failure. CXR showed bilateral effusions and pulmonary congestion. Moreover, his pleural effusions improved after dialysis
Changes in the text: The CXR findings have been added in Page 2 and Line 29.

Comment 2: It may be beyond the scope of the manuscript, but do the authors know how frequent SLE without serosal or mucocutaneous manifestations is?
Reply 2: While not addressed in the case report, mucocutaneous manifestations are noted in about 80% of SLE cases, but cutaneous manifestations are not required for the diagnosis. The source for the statistic is: https://www.ncbi.nlm.nih.gov/books/NBK535405/
Changes in the text: No changes in text were made

Comment 3: However, when the patient presented with stage 3 chronic kidney failure, was there any knowledge on the etiology and basis of this? Had a biopsy ever been performed? Had the CKD presented gradually or was it a case of AKD leading to CKD?
Reply 3: Patient never had an biopsy done. However patient was not compliant with hypertension and as such it was attributed to his uncontrolled hypertension.
Changes in the text: The information stated above have been added to Page 2, Line 25-26

Comment 4: Minor issue: p3, lines 52-54, a sentence is repeated twice.
Reply 4: We apologize for the error. This was overlooked in our proof reading. Thank you for pointing this out
Changes in the text: The extra line has been deleted

Reviewer B

Comment: Importance and relevance of this case
Reply 1: Our case highlights a rare case of SLE after COVID-19. While we do discuss the possibility of an underlying SLE that was exaebrated by COVID-19, we also elaborate why this could be less likely in our case in Page 5, Lines 93-106
Changes in the text: None

Reviewer C

Comment 1: Lines 52-54 need clarification, because I think this is the crux of this case. The feature of FSGS is frequently observed in lupus nephritis, however we will need

more information about the biopsy, whether it had any subepithelial or subendothelial proliferation, formal class (IV? V?) and NIH scoring etc. The complete absence of immune complex deposits raise question about the lupus nephritis diagnosis.
Reply 1: The biopsy results were discussed with the pathologist in detail and intracapillary deposits were found. The biopsy results were classified as Class IV. Thank you for pointing these out and we believe that this strengthens our case further.
Changes in the text: The biopsy results have been added to Page 2 Lines 36-41.

Comment 2: Also of note, line 52-54 are duplicates?
Reply 2: We apologize for the error. This was overlooked in our proof reading. Thank you for pointing this out
Changes in the text: The extra line has been deleted

Comment 3: Lines 74-83: Depending on what audience you are targeting, not sure re-iterating and counting classification criteria are really necessary (especially if it's for rheum audience). I will also add the combination of ANA+ and lupus nephritis will satisfy the diagnosis of SLE.
Reply 3: Thank you for pointing this out. This is a decision we as authors struggles with in deciding whether to mention or not. But, ultimately as internists, often there is confusion on the diagnosis of SLE and with the recent update in 2019 guidelines we decided to include this. This is also beneficial as internal medicine residents about outlining how to use the criteria.
Changes in the text: Lines 69-71 on page Page 4 have been added

Comment 4: The subsequent section describing the possible mechanisms are too vague. Though there are no RCT level data, there should be enough mechanistic papers and reviews out there to more clearly delineate or hypothesize how COVID19 may or may not induce autoantibodies. The role of interferon (IFN) was also not really discussed in these pathways.
Reply 4: Data suggesting the connection between B cell activation in COVID patients and unexposed patients have been cited. Thank you for pointing out the importance of interferons in COVID-19 and autoimmunity. After doing further research, we the authors have added a paragraph and further citations supporting the role of interferons, particulary Type I in autoimmunity and COVID-19
Changes in the text: Lines 76-87 on page Page 4 have been added