

REGARDING MANUSCRIPT 30581

Unicentric Castleman's disease associated with end stage renal disease by amyloidosis

Dear Editor;

Thank you for your continued interest in the above manuscript! We have considered the reviewers' comments and below provide a point-by-point response as well as an updated manuscript with changes made using tracked changes.

We have tried to comply with most requests and we believe that the revised version reads better and more appropriately puts into context the results of this letter. We hope the Editor in Chief and Reviewers find that the changes performed justify the acceptance of our case report in **World Journal of Clinical Cases**.

Yours sincerely,

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Response to reviewers

Reviewer 00503254

Comments

In this manuscript, the authors report on a case of unicentric Castleman's disease associated with end stage renal disease by amyloidosis. This case report is clinically interesting. However, there are some points that need to be addressed. Minor comments: 1. In the Case Report, they show that the hemoglobin level on admission was 9.6 mg/dl, but 9.6 mg/dl should be changed to 9.6 g/dl. 2. In the Case Report, they report that urine stick test was revealed three positive protein with normal findings. What are normal findings?

Response

Thanks for the comments. **1.** According to your suggestions we changed the hemoglobin level to 9.6 g/dL. **2.** We made a mistake, so we changed the sentence to "The urine stick test revealed a result of three positive for protein but there were no erythrocyte and leukocyte casts in the microscopic evaluation of the urine sediment".

Reviewer 00503257

Comments

This is an interesting case report if the patient did not have any other etiology except for past history of Castleman's disease for underlying renal amyloidosis. Thus, the following issues should be clarified. 1. The patient's clinical status at the latest observation on 2013 should be described in more depth. How about inflammatory condition of the patient? At least, serum C-reactive protein level and erythrocyte sedimentation rate should be described. If possible sequential changes of these inflammatory parameters in this patient should be described. 2. Did the patient have any other underlying chronic inflammatory condition, such as tuberculosis, malaria infection, or rheumatoid arthritis, etc.? Please clarify this issue. 3. Any information about patient's clinical manifestations during from 2013-2016 should be added. 4. Any speculation why this patient unexpectedly developed renal amyloidosis should be needed. Proper English editing should be needed throughout the text.

Response

Thanks for the comments. **1.** According to your suggestion we described the clinical state of the patient more in depth. We added the results of the patient in terms of inflammatory status in 2013. We also added the sequential changes of the inflammatory markers including the erythrocyte sedimentation rate and C-reactive protein into the manuscript. **2.** The patient did not have any chronic inflammatory conditions such as tuberculosis, malaria, rheumatoid arthritis, or familial Mediterranean fever. The inflammatory condition is attributed to low grade inflammation due to plasma cell type Castleman's disease. **3.** The patient did not come in for routine check-ups for his disease between 2013 and 2016, thus we have no idea about his clinical condition during these three years. **4.** However, his disease was in remission radiologically, and low grade inflammation may be responsible for amyloidosis in this patient. We speculate that low grade inflammation exists in patients with Castleman's Disease and plasma cell type. These findings suggest that physicians should be careful in terms of the presence of an inflammatory state and of amyloidosis, although the disease was in radiologically remission. English editing was performed by a native English speaker. We also added these changes into the manuscript.

Reviewer 00503175

Comments

Case report "Unicentric Castleman's disease associated with end stage renal disease by amyloidosis" by Eray Eroglu et al. is according to my opinion, acceptable for publication. This article is interesting, useful and written on the properly way.

Response

Thanks for the supportive comments.

Reviewer 00503255

Comments

The authors described a patient with unicentric Castleman's disease who developed end stage renal disease caused by amyloidosis. **1.** How did amyloidosis occur in the patient with

Castleman's disease? Please provide pathogenic mechanisms for the development of amyloidosis in Castleman's disease. 2. Why did amyloidosis develop in this patient long after a complete resection of culprit lesion? Were there any cryptogenic lesions in this patient? 3. The main text does not have the introduction. Please provide it.

Response

Thanks for the comments. **1.** Although the patient's disease was in remission radiologically, a low grade inflammation may be responsible for amyloidosis in this patient. We speculate that low grade inflammation exists in patients with Castleman's Disease and plasma cell type. These findings suggest that physicians should be careful in terms of the presence of an inflammatory state and of amyloidosis, although the disease was in remission radiologically. We also added these changes into the manuscript. **2.** It is known that chronic inflammation is associated with plasma cell type UCD and this histological type may contribute to the amyloidosis development. There were millimetric lymph nodes in the paraaortic area. It is possible that millimetric lesions might be active in terms of inflammation. There were no cryptogenic lesions and there were no any inflammatory conditions such as tuberculosis, malaria, rheumatoid arthritis, or familial Mediterranean fever. **3.** The main text has an introduction.

We added these changes to our manuscript.