Reviewer’s report

Title: Cerebral involvement in a case of Goodpasture syndrome, a serious complication due to shortened induction therapy: a case report

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Which of the following best describes what type of case report this is?: Unexpected or unusual presentations of a disease

Has the case been reported coherently?: Yes

Is the case report authentic?: Yes

Is the case report ethical?: Yes

Is there any missing information that you think must be added before publication?: Yes

Is this case worth reporting?: Yes

Is the case report persuasive?: Yes

Does the case report have explanatory value?: Yes

Does the case report have diagnostic value?: Yes

Will the case report make a difference to clinical practice?: No

Is the anonymity of the patient protected?: Yes

Comments to authors:

Interesting to read an account of a GD patient with CNS complications and MRI changes because, whilst a well recognised association, the volume of such reports is very low and the interest high on account of the occurrence of a3 and (in other reports) antibody-deposition on the choroid plexus. I’d prefer if the case was described as having Goodpasture’s disease or anti-GBM disease rather than the less specific Goodpasture syndrome that is widely applied to mean RPGN + pulmonary haemorrhage (with many causes).

The report would be improved with clearer accounting of the immunosuppressive therapy given to the patient and the degree of BP control achieved. In particular, since much is made of the use of pulsed rather than continuous oral cyclophosphamide, it is important to record the number and timing of the pulses.
administered before as well as after the neurological admission. This would be best achieved by expanding Table 1 (shouldn’t this be a figure- its not a table!) to record events from the date of original admission with acute renal failure including boluses of CyP and PEX treatments. Also, I am unclear what if any steroid was given. It would be unusual to not give highish dose steroids for at least a while. Please clarify.

Note that reference 6 should not be cited to support recommendations on duration of therapy in GD (as in introduction)- its correctly cited later as informative on puse vs continuous CyP in ANCA-vasculitis. There are several sets of ‘recommendations’ in the literature directly pertinent to GD that could be cited in stead, and not all advocate more than 3 months immunosuppression: a duration should probably be influenced by the rate of disappearance of antoantibodies in individuals.

Lastly, its simplistic to talk about the pathology in GD as a type II hypersensitivity reaction when so much data is now available (mouse and rat models and studies of T cells in man) to support roles for CD4 T cells as well and anti-GBM antibody in the pathogenesis and in particular in the genesis of glomerular crescents.

Quality of written English: Needs some language corrections before being published

Declaration of competing interests:

'I declare that I have no competing interests'