Author’s response to reviews

Title: Case report: Scleroderma with crescentic glomerulonephritis

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Author’s response to reviews: see over
Reviewer's report
Title: Case report: Scleroderma with crescentic glomerulonephritis
Version: 5 Date: 7 November 2007
Reviewer: PETER HEWINS
I am familiar with the literature and believe that this case meets one of the 7
criteria for evaluation in the journal: An unexpected association between
diseases or symptoms
Has the case been reported coherently?: Yes
Is the case report authentic?: 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?: Yes
Comments to authors:
General
This manuscript is much improved.
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Revisions necessary for publication
1) I do not agree with the statement that 'pulse methyl prednisolone .... can be
considered the standard of care' for crescentic GN. In lupus nephritis (classes iii
and iv), ANCA-associated GN and anti-GBM nephritis, for example, there is clear
evidence that steroid only therapy is inadequate and additional therapy is
needed. Cyclophosphamide is widely used and in the cases of anti-GBM
nephritis and ANCA-associated GN with severe renal failure, plasma exchange
would be the standard of care. For lupus nephritis, mycophenolate may be an
alternative to cyclophosphamide. The text should be amended accordingly.
Reply: Text has been amended accordingly.
2) Moreover, recovery from dialysis dependent renal failure is possible
(particularly for ANCA and lupus nephritides) and histopathological changes are
often patchy. Accordingly, the statement that 'She did not respond to Methyl
prednisolone also probably due to the presence of fibrous and fibro cellular
crescents' is one which could be misinterpreted and should be amended. With
the benefit of hindsight, a trial of additional immunosuppressive therapy would
probably be have been appropriate.
Text has been amended suitably.
3) I would interpret the histopathology and immunofluorescence as suggesting a
immune complex mediated disease - it would be appropriate to indicate this. I
would still contend that the features and consistent with a lupus nephritis. If
serum C3 and C4 levels were assayed, these should be recorded. If the authors
consider that the pathologic findings some other particular etiology, they should
state this.
Histopathology is interpreted as immune complex mediated disease. We do not
consider lupus nephritis as underlying disease here because C3 C4 levels are
normal and the immunoperoxide staining did not reveal a full house
immunoglobulin deposits. Patient also did not have clinical features of SLE.
4) I recommend that the authors make specific reference to HCV associated MCGN. It is important to be mindful of HCV (and other infectious) causes of crescentic GN particularly in the context of features of immune complex disease and before considering immunosuppressive therapy. In this particular case, the absence of purpura and other extra-renal manifestations, together with the absence of endoluminal thrombi make a cryoglobulinemic GN less likely but these are not invariably present. It would be worth noting if the patient were rheumatoid factor +ve or had abnormal liver enzyme levels. Patient was negative for third generation anti HCV antibodies and had RA factor level of 8 IU/ml and did not have abnormal liver enzyme levels. Text is suitably amended.

What next?: Accept after minor revisions

Quality of written English: Acceptable