PAS stain shows global and prominent thickening of the glomerular basement membranes, in the absence of evidence of cellular proliferation (PAS, x 400).
nephropathy, except in those cases associated with systemic lupus. The immunopathogenesis of this unusual transformation is unclear. It is well recognized that patients with a crescentic glomerulonephritis have severe and often rapidly deteriorating failure. Unlike membranous nephropathy, which often has an insidious course progressing to renal failure over a period of years, patients with superimposed crescentic glomerulonephritis appear to have a more aggressive clinical course. The importance of recognizing this group of patients with membranous nephropathy and crescentic glomerulonephritis is that immunosuppressive therapy may ameliorate the progression of renal damage and in some cases early treatment was associated with useful recovery of renal function. In our case, the discontinuation of prednisone and azathioprine therapy may have facilitated the rapid progression of kidney disease.

Conflict of interest
The authors declare that there is no conflict of interest associated with this manuscript.


They reported a case of membranous glomerulonephritis (MGN) with crescentic transformation in a ANCA-negative vasculitis which revealed no evidence of systemic lupus erythematosus (SLE), anti-glomerular basement membrane (GBM) glomerulonephritis, infection, malignancy and showed no improvement after immunosuppressive treatments. The case they presented was similar to the patient that Kwan JT et al. described previously. Although several authors have demonstrated the concomitance of MGN and ANCA-associated glomerulonephritis, MGN accompanied by ANCA-negative crescentic glomerulonephritis has been rarely encountered.

The light microscopic visualization of renal tissue in their case showed the formation of 11 crescents (3 cellular crescents, 1 fibrocellular crescent and 7 fibrotic crescents) and 11 out of 17 glomeruli were globally sclerotic. These histopathological changes indicate the patient has reached to an advanced stage of crescentic glomerulonephritis and the renal disease has progressed to the sclerotic phase at the time of renal biopsy. Nasr SH et al. reported that the percentage of globally sclerotic glomeruli correlated with nonresponse to immunosuppressive agents. This is why the patient showed no improvement after treated with steroid plus cyclophosphamide and started chronic haemodialysis treatment eventually. By contrast, our case showed 2 sclerosed glomeruli out of 19 glomeruli, the formation of 9 crescents including 4 cellular crescents and 5 fibrocellular crescents, as well as the fibrinoid necrosis lesions upon light microscopy. This indicates our patient might be at the relatively early stage of crescentic glomerulonephritis and the renal biopsy findings may interpret the better response to immunosuppressive treatments in our case.

To the Editor:
We were very interested by the comment submitted by Dr. Gioacchino Li Cavoli and his collaborators, regarding their similar experience of a membranous glomerulonephritis with crescentic overlap. First of all, we would like to thank them for their input.

Response:
Response to “Comment on 'Membranous glomerulonephritis associated with mieloperoxidase antineutrophil cytoplasmic antibody associated glomerulonephritis’’”
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