

 GLOMERULAR DISEASE

# Chemotherapy for monoclonal gammopathy-associated C3G



Patients who achieved haematological response after chemotherapy had higher renal response rates



Monoclonal gammopathies are associated with a variety of renal manifestations that are induced by the deposition or precipitation of monoclonal immunoglobulin (MIg) produced by clonal B cells. New findings suggest that monoclonal gammopathies are present in a high proportion of patients with C3 glomerulopathy (C3G) and that treatment of the underlying B-cell clone is associated with improved renal outcomes.

To investigate the association between MIg and C3G, Sophie Chauvet and colleagues performed a retrospective study of 201 patients in the French C3G registry; 60 (29.7%) of these patients had monoclonal gammopathy. The prevalence of MIg was higher in patients >50 years of age (59%) and increased with increasing age. The researchers evaluated renal outcomes in 50 patients with MIg and C3G according to treatment strategy: chemotherapy adapted to the B-cell clone ( $n = 29$ ), conventional immunosuppressive therapy ( $n = 8$ ), or

renin–angiotensin system blockade ( $n = 13$ ). At diagnosis, 43% of patients had nephrotic-range proteinuria and 86% had chronic kidney disease stage 3 or higher. More patients in the chemotherapy group than in the other groups achieved a haematologic response, and patients who achieved haematological response after chemotherapy had higher renal response rates and median renal survival than those receiving conservative or immunosuppressive therapy.

The researchers are planning further studies to investigate the mechanistic link between monoclonal gammopathy and C3G. They also recommend careful haematological workup in patients with C3G and MIg and early consideration of chemotherapy targeting the underlying B-cell clonal disorder.

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**ORIGINAL ARTICLE** Chauvet, S. et al. Treatment of B-cell disorder improves renal outcome of patients with monoclonal gammopathy-associated C3 glomerulopathy. *Blood* <http://doi.org/10.1182/blood-2016-08-737163> (2017).