A trial of complement inhibition in a patient with cryoglobulin-induced glomerulonephritis

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Cryoglobulinemia induces an immune complex-mediated glomerulonephritis that is characterized by the presence of large immune deposits, including complement C3 and C5b-9, marked macrophage influx and mesangial cell proliferation. The precise role of complement in cryoglobulin-induced glomerulonephritis in humans remains unclear, whereas in mice there has been evidence that complement activation might be a central factor favoring glomerular inflammation, particularly by the recruitment of neutrophils. We report on an exceptional case of cryoglobulin-induced glomerulonephritis in a patient with mixed essential cryoglobulinemia type II. The clinical features included relapsing proteinuria and renal function impairment that were controlled by plasmapheresis. Complement was low in plasma and two renal biopsies at 1-year interval showed prominent immunoglobulin and complement deposits, with unusual high numbers of neutrophils. In a 1-patient clinical trial, we tested whether the monoclonal anti-C5 antibody eculizumab would be sufficient to control renal function at the time of a relapse. Although during the initial weeks renal function was stabilized, slow increase in creatinine could not be controlled by this treatment, so that plasmapheresis was reinstituted. This result suggests that despite evidence for a role of complement in enhancing renal damage in this patient, other inflammatory processes dominated.

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