IgA nephropathy: molecular mechanisms of the disease.

Abstract
Studies of molecular and cellular interactions involved in the pathogenesis of IgA nephropathy have revealed the autoimmune nature of this most common primary glomerulonephritis. In patients with this disease, altered glycan structures in the unique hinge region of the heavy chains of IgA1 molecules lead to the exposure of antigenic determinants, which are recognized by naturally occurring antiglycan antibodies of the IgG and/or IgA1 isotype. As a result, nephritogenic immune complexes form in the circulation and deposit in the glomerular mesangium. Deposited immune complexes induce proliferation of resident mesangial cells, increased production of extracellular matrix proteins and cytokines, and ultimately loss of glomerular function. Structural elucidation of the nature of these immune complexes and their biological activity should provide a rational basis for an effective, immunologically mediated inhibition of the formation of nephritogenic immune complexes that could be used as a disease-specific therapeutic approach.
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