Clinical Aspects of Glomerular Diseases

Recent advance of modern medicine in the field of renal diseases is remarkable. The favorable treatment result for experimental nephritis, and progress of cellular and humoral immunological studies as well as prevalence of renal biopsy have contributed to elucidate the pathogenic mechanism of glomerular nephritis. Renal biopsy plays an important role to combine clinical findings and morphological pictures, and makes the clinical classification possible. The advance of these diagnostic procedures has indicated the existence of difference between each treatment or therapy for glomerular diseases and the prognosis.

A group in which IgA precipitates mainly on the mesangium by an immuno-fluorescence method among several proliferative glomerulonephritis has been classified and called IgA nephropathy. The exact etiology of this disease remains obscure, but it has been reported that IgA immune complex is detected in a relatively high rate. Among the detection methods, Raji cell binding assay and anti-IgA inhibition binding assay seem to be good. It is also known that IgA immune complex mainly activates alternative pathway of complements following C3 in the renal tissue. IgA nephropathy, dysfunction of IgA specific suppressor T-cell and increase in IgA specific helper T-cell as well as increase in IgA bearing lymphocytes are observed, but there is no certain tendency of involvement of cellular immunity in IgA nephropathy.

Membranoproliferative glomerulonephritis (MPGN) manifests thickening of the basement membrane of glomeruli and hypertrophy of the mesangium cell. MPGN has three morphological types. Type I which the basement membrane of the glomeruli is thickened irregularly accompanied by mesangial interposition and type II which the basement membrane is thickened accompanied by dense deposits. Type III which is a mixed type of type I and type II. Low complement titer is observed in MPGN, the same as in acute glomerulonephritis, also type II manifests an appearance of C3 nephritic factor (C3NeF) as compared with type I. This C3NeF is considered as an autoantibody to IgG of C3 convertase (C3bBb) which is produced by the alternative pathway.

In our experience, the diagnosis of focal glomerulosclerosis (FGS) is difficult depending on the site or count of the glomeruli taken by biopsy on account of the lesional focus of FGS is located at deep in the juxtamedullary region. In such a case, the differential diagnosis from minimal change group is especially difficult. There are two hypothesis regarding pathological factors of FGS. The first hypothesis is that some of the capillary of the glomerulus will be obstructed by etiological factors (such as enhancement of local coagulation?) in minimal change group as time goes on. The second hypothesis is that FGS will be quite a different disease from the minimal change group and will be caused spontaneously. In many of FGS, predominant precipitation of IgM, C3 and fibrinogen in the sclerosis lesion was observed by the immuno-fluorescence method, but IgG and IgA are not observed. From the findings of the precipitated picture by the immuno-enzymatic electron microscopy for IgG, FGS cannot be considered as immune complex disease.

For the treatment of glomerular diseases, corticosteroids are most widely used, and pulse therapy, immuno-suppressant therapy, anti-coagulant therapy, anti-platelet drug therapy and non-steroidal anti-inflammable drug therapy are also conducted. It is thought that some patients with progressive glomerulonephritis and intractable nephrotic syndrome well respond to pulse therapy. It is also thought that τ-globulin therapy is effective for immune complex nephritis. At present, gebexate emisilate has property of multiprotease inhibitor and acts on the caogulantfibrino lytic system, platelet function and kinin-kallikrein system is also applied.

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