Dear Sir,

IgA nephropathy (IgAN) is defined by the presence of diffuse deposits of dimeric IgA in the mesangium. These immunoglobulins classically coexist with the complement factor C3, while Clq and C4 are absent, or exceptional [1]. The presence of the terminal factor C9 has also been reported [2], as well as the presence of the alternate pathway component properdin [1]. These studies are however very few compared to the abundant literature on this topic [3].

We report on the glomerular distribution of 7 complement components, the presence of which was systematically looked for in 48 patients with IgAN. Clinical data related the disease to Henoch Schönlein purpura (HSP) in 9 cases. One patient also suffered from liver cirrhosis. The 38 remaining patients were assumed to suffer from primary IgAN or Berger’s disease.

The composition of glomerular deposits was analyzed in direct immunofluorescence, using a panel of fluorescein-conjugated antisera to human immunoglobulins IgG, IgA and IgE (Behring, Marburg, FRG), and complement factors Clq, C3c, C4 (Behring), factor H and properdin (Atlantic Antibodies, Scarborough, Me., USA). Indirect immunofluorescence was used to visualize complement factors B and C9 (Behring), with sheep antirabbit Ig fluoresceinated serum as second-step reagent (Behring). Positive and negative controls had been previously obtained on human tissue sections under similar technical conditions.

Mesangial deposits of IgA were obviously present in 100% of the samples, together with C3c in 94%. IgG, IgE and C4 were absent from all samples. Focal endomembranous or intraendothelial deposits of IgM were observed, often in minute amounts, in 39% of the biopsies. A similar partition was observed for Clq in the same biopsies but 4. Intraendothelial deposits of Clq alone were seen in 4 instances (8%). Small mesangial amounts of B factor were observed in one sample only. C9 was present in 89% of the cases, properdin in 73% and H factor in 69%. These three complement components coexisted in 44% of the biopsies, including the one with associated liver cirrhosis and 5 of the 9 cases of HSP.

This data support the participation of the alternate pathway of complement activation in IgAN. The presence of H factor but not B factor suggests that although it is complete (presence of C9), the complement activation is highly controlled and limited. This might explain the usually mild consequences of immune complex deposition in IgAN.
References