

Protective role of rheumatoid factor in lupus nephritis

Sirs,

Lupus nephritis is a severe manifestation of systemic lupus erythematosus (SLE) with a prevalence varying from 31 to 65% according to the studied population (1). It is associated with the presence of anti-DNA antibodies but it is questioned if other antibodies such as rheumatoid factor (RF) might also be involved in its occurrence.

Birchmore *et al.* (2) observed that RF enhanced the binding of DNA-anti DNA complexes to C3b receptors in erythrocytes *in vitro* by fixating complement to its own Fc region. Their finding suggests that the presence of RF may increase the risk of renal injury in SLE. Others (3, 4) measured RF in SLE patients and found that the frequency as well as the titer of RF in patients with nephritis did not differ from those without it. Conversely, Hill *et al.* (5) found that patients with SLE and nephritis who were RF positive had milder renal lesions when compared to those without this autoantibody.

We have studied the prevalence of IgM RF by nephelometry in 187 SLE patients (6 males and 181 females) with mean age of 30.6 years and mean disease duration of 86.5 months. Sixty-six of them (35.9%) had biopsy proven glomerulonephritis with four (2.1%) class VI, 11 (5.8%) class V; 33 (17.9%) class IV; 11 (5.8%) class III and

seven (3.8%) class II by the WHO classification. We found that 42 patients were RF positive: four (9.5%) of them with renal disease and 38 (90.5%) without it. One hundred and fifteen patients were RF negative of which 50 (43.6%) with glomerulonephritis and 65 (56.5%) without it ($p=0.001$; Fisher). The four patients with glomerulonephritis and positive RF were one class 3, two class 4 and one class 5 nephritis.

Our findings support a protective role for RF in lupus nephritis. This can be explained by the fact that: (a) RF may compete with complement for binding to immune complexes (6); (b) RF binding to antigen-antibody complexes may result in a more efficient removal by the reticuloendothelial system (6), (c) RF blocks the attachment of C3 to aggregated IgG not allowing its reaction with C3b receptors of glomeruli thus shielding them from deposition of pathogenic immune complexes (7).

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