Letters to the Editor

were seen in two and three cases respectively. Ultrastructural examination showed electron-dense deposits localised in the mesangium in nine cases. Patients were treated during the first month with prednisone 1 mg/kg per day. During the next two months the dose was reduced to 1 mg/kg on alternate days and then gradually decreased. Steroid-resistant patients were treated with 0.2 mg/kg per day of chlorambucil for three months. In no case was the total dose of chlorambucil greater than 25 mg/kg. One nephrotic patient experienced spontaneous remission and eight others were steroid-responsive. Only one of the five steroid-resistant patients treated with chlorambucil showed clinical remission. Five steroid-responsive patients relapsed. At present, four patients are "healthy," having not relapsed for the past two years.

We think it is important to determine if the presence of IgM in renal biopsies indicates a poor prognosis in idiopathic nephrotic syndrome. Cohen et al.1 have suggested a relatively poor prognosis in five such patients after treatment, showing clinical remission in only one case. On the other hand, Bhasin et al.4 described six of eight patients who initially achieved clinical remission, although four of these later required cytotoxic drugs or were steroid-dependent. It therefore appears that whereas the short-term prognosis in IgM associated mesangial proliferative glomerulonephritis is good, the long-term outlook must be much more guarded.

We do not know whether IgM mesangial nephropathy is really a single disease entity or whether the IgM deposition is simply a concomitant immunological finding not necessarily related to the pathogenesis of the nephrotic syndrome. Only a small number of cases have been reported of this ill-defined glomerular disease. A definitive statement regarding the course and prognosis is not possible. More studies are necessary to confirm that IgM mesangial nephropathy is indeed a separate entity.

Dr Lawler and his colleagues comment as follows:

Thank you for inviting us to comment on the letter by Gonzalez et al. The cases which they describe appear to be very similar to ours1 in structural and immunopathological terms, although criteria for selection were different; thus all their 14 patients had the nephrotic syndrome, whereas 9 of our 23 patients had asymptomatic proteinuria.

In our experience, based on these 23 cases2 and another unpublished group of 20 similar cases of IgM-associated primary diffuse mesangial proliferative glomerulonephritis, both clinical remission during steroid therapy and spontaneous improvement are uncommon, the majority of patients pursuing a chronic indolent course which, in a minority, progresses to end-stage renal failure. It may well be that what we and others3,4,5 including Gonzalez et al., have described is a heterogeneous group, and that the patients who improve, either spontaneously or with steroid therapy, may represent a different pathogenetic mechanism. Nevertheless, the fact that the majority do not improve suggests that they should be considered as a distinct clinicopathological group.

We agree that further long-term studies are required to confirm IgM mesangial nephropathy as a separate entity and, if so, to determine its course and ultimate prognosis.

References


Misappellation of Russell's name

The paper by Bartolini et al. (October 1980;33:936) contains clerical slips in the spelling: Russell's bodies, but a more serious error in suggesting that the intracellular bodies described by Russell (1890) occur within plasma cells. Russell stated that these fuschinophil bodies occurred within and around cancer cells, but not in sarcomata or in simple tumours.

Modern staining methods make it fairly certain that the inclusions seen in carcinoma cells are of fibrin, and this gains support by the greater number of inclusions within carcinoma cells adjacent to fibrinous coagula. On the other hand, the inclusions within plasma cells are now linked to immunological changes, do not stain exactly as fibrin, and surely should never be called Russell bodies.

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Reference


Ethanol-induced vacuolation in red cells

The article "Cytoplasmic vacuolation of peripheral blood cells in acute alcoholism" (J Clin Pathol 1980;33:1193-6) found our interest. Working in the field of haemorheology, we carry out studies of red cell deformability (RCD) using a filtration method based on a technique originally developed in our laboratory and described in this Journal.1,2 Studying healthy volunteers, we observed that RCD was reduced after alcohol intake during the night before the measurement (on average 13%). Subsequently we performed a series of in vitro experiments, determining the RCD of physiologically deformable red
Misapplication of Russell's name

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