if any relationship exists between the presence of e and the eventual progression to the $HB_sAg$ carrier state, it is probably related to the persistence of e but is not necessarily related to the detection of e antigen early in the acute phase of hepatitis B.

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## IgA localisation in glomerular diseases

We read with interest the article by Lawler *et al.* from Manchester in the October issue of the *Journal of Clinical Pathology*, in which they described the structural changes on light and electron microscopical study of 25 renal biopsy specimens that showed significant IgA deposition on immunofluorescence and correlated their findings with relevant clinical data.

Nephrologists differ considerably in the use and indications for renal biopsy in the management of their patients. For this reason analysis of renal biopsy material may vary widely between different centres. In Bristol, the use of biopsies in renal medicine has increased rapidly with the introduction of immunofluorescent techniques, and we are now using repeat biopsies both to study the natural history of certain types of renal disease and to assess response to treatment. With this background we thought your readers would be interested in our results on a larger series of biopsies.

A total of 500 percutaneous renal biopsy specimens have been studied by direct immunofluorescence. Of these 31 were strongly positive and 46 were moderately positive for IgA. These 77 biopsies came from 60 patients (48 with one biopsy, 7 with two biopsies, and 5 with three biopsies) and formed 15·4% of the total.

IgA, usually with the $C_3$ component of complement, was the sole localising immunoglobulin in only five cases; in 25 it was present with IgM; in 36 with both IgM and IgG; and in 11 cases with IgG. In 52 cases IgA was the predominant localising immunoglobulin, in 14 there was no predominant immunoglobulin, and in 11 cases IgG predominated.

Our morphological findings were in general agreement with those described in the Manchester series, and on light microscopy a similar proportion of patients could be designated as mesangial proliferative glomerulonephritis (*viz*, 35 (58%) of our 60 patients in comparison with 15 (60%) of the 25 Manchester cases).

It is not necessary in this letter to analyse further the morphology of our cases. However, there are significant differences when considering the clinical data and clinicopathological correlations. Irrespective of the morphological findings, we excluded from the diffuse mesangial proliferation group those cases which had been diagnosed on clinical or immunological grounds as suffering from Henoch-Schönlein nephritis or lupus nephritis. Eight patients came into each of these two groups, and in Henoch-Schönlein nephritis IgA was always present as the predominant immunoglobulin situated in the mesangium. In contrast, out of 23 patients (36 biopsies) with immunologically proven lupus nephritis, only eight had moderate or severe IgA and in only one of these was it the predominant immunoglobulin.

The clinical presentation of the 35 cases with diffuse mesangial proliferation is also of interest:

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|---|---|
| Recurrent haematuria | 17 cases |
| Proteinuria | 9 cases |
| Haematuria and proteinuria | 5 cases |
| Loin pain | 1 case |
| Haematuria and loin pain | 1 case |
| Nephrotic syndrome | 2 cases |

The sex ratio in these patients was 27 male to 8 female. We agree that the prognosis in some of these patients was unfavourable and, although this was usually associated with increasing glomerular sclerosis, crescents were unusual in our series, and the presence of IgA, which was persistent in serial biopsies, did not seem to be related to prognosis. More important in assessing prognosis seems to be the development of hypertension and/or proteinuria occurring between attacks of haematuria. In some cases renal failure developed in an alarmingly short period of time.

It is not possible to analyse our results further in this short communication but, without detracting from the Manchester authors' findings, study of a larger series widens the spectrum of glomerular disease in which IgA may be found. Analysis and publication of renal biopsy material is essential, but the material must be interpreted in the light of local conditions. Only by close co-operation between pathologists and nephrologists at different centres can sufficient material be accumulated to present a true overall picture of renal disease. The correspondence column of this journal might act as a forum to accumulate more data from centres where time and personnel are limited by heavy workloads.

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