

**PART XXIX**

**WORKSHOP ON IMMUNOPATHOLOGY OF NEPHRITIS**



## IMMUNOPATHOLOGY OF NEPHRITIS

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The workshop on immunopathology contained eight papers that had been selected by the programme committee. Five papers were on clinical nephrology and three on experimental nephrology. No main theme had been chosen for this workshop.

Kazatchkine et al from Paris [1,2] demonstrated that the presence of C<sub>5</sub>b-9-neo-antigens is not necessarily associated with the presence of immune deposits in glomeruli. They showed that the neo-antigens were present on old immune deposits and cell remnants present in glomeruli but only occasionally on small newly formed immune deposits. These findings question the role of C<sub>5</sub>b-9-neo-antigens in the induction of proteinuria in a variety of glomerular disease.

Williams et al from London [3] demonstrated a role for macrophages in the increased production of IgA and IgG in patients with IgA nephropathy. Using ELISA techniques they were able to demonstrate an increased spontaneous production of IgA and IgG but not of IgM in patients with IgA nephropathy. In pokeweed mitogen stimulated cultures of peripheral blood cells plastic adherent cells stimulated the production of IgA and IgG but not of IgM. The significance of this finding has to be established.

Two papers on membranous nephropathy focused on predictive and prognostic indices in this glomerular disease. Adel Hassan et al from St Etienne [4,5] demonstrated a decrease of the T4/T8 ratio in patients with membranous nephropathy in remission, a high T4/T8 ratio and a decrease of the percentage of suppressor T cells existed in the active phase. They concluded that a helper/suppressor cell imbalance was important in the pathogenesis of membranous nephropathy.

Zucchelli et al from Milan [6] studied a group of patients with membranous nephropathy in a long-term prospective and randomized therapeutic trial based on methylprednisolone and chlorambucil for six months. They found that patients with a higher helper/suppressor cell ratio before therapy were more likely to experience a remission during therapy. It was established that in itself therapy did not influence the helper/suppressor cell ratio. In addition they found that patients with a DR3/B8 haplotype which shows a strong association

with the occurrence of membranous nephropathy have a lesser chance on a spontaneous remission than do patients with DR3<sup>+</sup>/B8<sup>-</sup> haplotype.

These two papers again stress the importance of the increased immune response and its role in the prognosis of membranous nephropathy.

Gronhagen-Riska et al from Helsinki [7] focused the importance of the fine-needle aspiration biopsy in addition to the classical renal biopsy in various glomerular diseases.

The experimental nephrology part of the workshop contained papers on nephrotoxic serum nephritis and on toxin induced membranous nephropathy in mice. Shinkai et al from London [8] as well as Bertani et al from Bergamo [9] demonstrated that an inhibition of thromboxane generation by either aspirin or the imidazole derivative OKY-046 which inhibit thromboxane generation by platelets as well as the prostaglandin synthesis in the kidney, worsens the nephrotoxic serum nephritis and increases the mortality of the animals. In addition Bertani et al showed that treatment with Sulindac which inhibits only thromboxane synthesis by platelets reduces inflammation and prevents severe renal function loss in nephrotoxic serum nephritis. This finding indirectly points to a protective effect of local prostaglandin production in the kidney in cases of nephrotoxic serum nephritis.

Fleuren et al from Leiden [10] demonstrated the induction of a membranous nephropathy in Balb/c mice on administration of low doses of mercuric chloride. The antigens involved in the induction of the disease showed similarities with antigens responsible for Heymann nephritis in rats. The development of this model opens future possibilities to study the role of genetics in the induction of an autoimmune membranous nephropathy in animals.

## Papers presented

- 1 Hinglais N, Kazatchkine M, Bhakdi S, Mandet C, Grossetete J, Bariety J. *C5b-9 in diseased human kidneys*
- 2 Hinglais N, Kazatchkine M, Bhakdi S, Appay MD, Grossetete J, Bariety J. *C5b-9 in normal kidneys*
- 3 Williams DG, Perl SI, Wilkinson AH. *Abnormal immunoglobulin production by lymphocytes and its control by adherent cells in IgA disease*
- 4 Adel Hassan A, Genin C, Le Petit JC, Laurent B, Berthoux F. *Immunogenetics of primary membranous glomerulonephritis*
- 5 Adel Hassan A, Genin C, Le Petit JC, Laurent B, Berthoux F. *T lymphocyte subpopulations in primary membranous glomerulonephritis*
- 6 Zucchelli P, Coagnoli L, Pasquali S, Beltrandi E. *B and T cell dysfunctions in patients with primary IgA nephropathy*
- 7 Gronhagen Riska C, v Willebrand E, Laasonen L, Pettersson E, Tornroth T. *Fine needle aspiration biopsy in renal disease*
- 8 Shinkai Y, Cameron JS. *Inhibition of thromboxane synthetase worsens accelerated anti-GBM nephritis in rabbits*
- 9 Bertani T, Benigni A, Cuttillo F, Rocchi G, Morelli C, Carminati C, Verroust P, Remuzzi G. *Effect of aspirin and sulindac in rabbit nephrotoxic nephritis*
- 10 Fleuren GJ, de Heer E, Burgers J, v Osnabrugge C, Hoedemaeker Ph J. *Mercuric chloride induced autoimmune glomerulopathy in Balb/c mice*