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SHORT REPORTS

Segmental necrotising glomerulonephritis with antineutrophil antibody: possible arbovirus aetiology?

Focal and segmental glomerulonephritis occur in mesangial IgA disease and several systemic diseases, but often the aetiology is unknown.[1] We describe eight patients seen over five years with a generalised illness associated with segmental necrotising glomerulonephritis. Clinical findings and the geographic distribution of the cases suggest that infection by a group A arbovirus may have been an aetiological factor.

Patients, methods, and results

The patients were four men and four women aged 28-71 years. All had been ill for several weeks with lethargy, weight loss, arthralgia or myalgia, and anorexia, nausea, vomiting, or diarrhoea. Renal symptoms (haematuria, loin pain, and oedema) were present in five and respiratory symptoms (dyspnoea or haemoptysis) in four. In all cases urine analysis showed microscopic haematuria, and in most there were also granular and hyaline casts and a small amount of protein. Serum creatinine concentration exceeded 0·1 mmol/l (1·1 mg/100 ml) in seven. Chest *x*-ray films showed abnormalities in five: in three these were mild and attributed mainly to oedema, but one had widespread intrapulmonary haemorrhage and another showed diffuse infiltration "consistent with a collagen disease." In all patients the erythrocyte sedimentation rate exceeded 100 mm in the first hour; five had a blood leucocyte count exceeding 10×10^9/l.

Routine tests for autoantibodies were negative, but all patients had in their serum a factor that stained the cytoplasm of neutrophil leucocytes by indirect immunofluorescence. Its maximum titre was 1/256, and it was predominantly IgG. It stained human leucocytes of different ABO blood groups equally well, but rodent leucocytes generally were unstained, although occasionally there was some weak cross-reactivity. Usually this neutrophil staining activity disappeared within a few days of the start of treatment, but in two patients it was present 12 months later, when the disease was otherwise quiescent. A retrospective serological survey for arbovirus antibody showed titres of 1/20 and 1/40 for Ross River virus in seven of the eight cases, suggesting previous infection.

Renal biopsy during the initial illness showed segmental necrotising glomerulonephritis with accumulation of fibrin and frequent formation of a crescent in the adjacent part of Bowman's space. Usually 35-75% of glomeruli were affected, but in two cases more than 90% were, while in one case fewer than 10% contained a lesion. Direct immunofluorescence showed only small irregular deposits of fibrin in damaged glomerular lobules and adjacent crescents.

All patients were treated with prednisolone (50 mg/day) and either cyclophosphamide (100 mg/day; seven patients) or azathioprine (100 mg/day; one). Three required haemodialysis, of whom two also received plasmapheresis (seven and 10 exchanges). After follow-up for one to five years all patients were alive and well; four had some residual impairment of renal function, but none required chronic haemodialysis or renal transplantation. Recurrent disease occurred in two cases, three and five years after the onset. In both this was associated with reappearance of antineutrophil factor in the serum, and renal biopsy showed active segmental glomerulonephritis on a background of long-standing glomerular scarring. Both patients again responded to treatment with prednisolone and cyclophosphamide.

Comment

The glomerular lesions in these cases were morphologically indistinguishable from those in microscopic polyarteritis nodosa.[2] Staining of neutrophil cytoplasm by these patients' sera was a characteristic diagnostic finding; this has not been previously described and has not been seen otherwise in more than 5000 sera examined during the past five years. Because of an unusual rural clustering of the cases, mainly in the Murray River valley, and the prominence of arthralgia and myalgia, this condition may be related to epidemic polyarthritis, which is common in this area[3] and caused by Ross River virus.[4] Serology showed evidence of previous infection by this virus in seven of the eight patients; the prevalence of serum antibodies to this virus in this region is 14·6%.[5]

Further serological investigation using IgM antibody as an index of recent infection is needed to establish a causal role for Ross River virus in this form of glomerulonephritis.

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