Membranous Nephropathy Associated with Sarcoidosis

Response to Prednisolone

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Dear Sir,

Sarcoidosis may be associated with glomerulonephritis, most commonly membranous nephropathy [1]. There is limited information available on the response of this glomerulopathy to therapy [2–4]. We have observed a case of membranous nephropathy associated with sarcoidosis which showed a response to high-dose alternate-day prednisolone.

In September 1978 a 32-year-old female presented with erythema nodosum, fever, polyarthralgia, and generalized rash. Chest X-ray demonstrated hilar lymphadenopathy, and lymph nodes obtained at mediastinoscopy were replaced by granulomata showing central necrosis without caseation. Creatinine clearance was 100 ml/min and urine protein excretion 70 mg/day. Treatment with prednisolone 50 mg daily was commenced which resulted in a dramatic improvement in the constitutional symptoms and resolution of the hilar lymphadenopathy. Steroids were ceased 2 years later after progressive reduction.

In September 1983 she again presented with erythema nodosum, fever, and hilar lymphadenopathy. The serum angiotensin-converting enzyme level was 53.4 (normal 16–34) nmol/ml/min, and the erythrocyte sedimentation rate was 100 mm/h. Serum creatinine was 0.8 mg/dl (70 µmol/l) and urine protein excretion 2.0 g/day. There was a rapid clinical response of the systemic manifestations to prednisolone 25 mg daily, accompanied by a return to normal of serum angiotensin-converting enzyme level and erythrocyte sedimentation rate. However, 6 months later urine protein excretion had stabilized at 1 g/day, and renal biopsy was, therefore, carried out. This showed changes of membranous nephropathy on light microscopy, and immunofluorescence showed granular deposits of IgG and C3 along capillary loops.

Prednisolone was

increased to 120 mg on alternate days, and over the next 3 months protein excretion became normal. Prednisolone was reduced to 25 mg on alternate days and then to 12.5 mg on alternative days 4 months later.

The urine protein excretion remained normal till March 1987, when it was found to be 6.9 g/day on a regular review. Serum creatinine remained normal, the serum angiotensin-converting enzyme level was 36.1 nmol/ml/min, and the erythrocyte sedimentation rate was 23 mm/h. Renal biopsy was carried out and the diagnosis of membranous nephropathy was again made on light and immunofluorescent microscopy. Electron microscopy (fig. 1) showed subepithelial deposits and basement membrane 'spikes' indicative of membranous nephropathy. Prednisolone was increased to 120 mg on alternate days. There was a progressive fall in urine protein excretion

over the next 6 months to approximately 300 mg daily, and prednisolone was then reduced to 25 mg on alternate days. Urine protein excretion remained stable, 6 months later. Previous cases of membranous nephropathy associated with sarcoidosis which have been treated with steroids are few [2–4]. However, the response would appear to be poor, and cyclophosphamide has usually been used as adjunctive treatment. In none of the cases described did remission occur. In this patient a remission was induced on two occasions. The difference in treatment in this case would appear to be a greater dose of prednisolone given for a protracted period. Moreover, the importance of dose is suggested by the first presentation, when proteinuria persisted despite the 6-month treatment period with prednisolone 25 mg daily, but, following an increase to 120 mg on alternate days, remission occurred within 3 months. This complication of sarcoidosis seems to be more resistant per treatment than other systemic manifesta-

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Fig. 1. Electron micrograph showing subepithelial electron-dense deposits and basement membrane 'spikes'. $\times 5,363$.

tions [3, 4], and this case suggests that a course of high-dose steroids may be effective. Success has also been reported earlier in patients with idiopathic membranous nephropathy treated with high-dose alternate day steroids [5].

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