Case report

Rheumatoid disease presenting as a nephrotic syndrome

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SUMMARY A 62 year old man with no relevant previous history presented with a nephrotic syndrome. Renal biopsy showed a membranous glomerulopathy and coincident investigation showed high serum titres of rheumatoid factors. It was not until some months later that he developed articular and extra-articular manifestations of rheumatoid arthritis.

Key words: membranous glomerulonephritis.

The association between the development of a nephrotic syndrome secondary to membranous nephropathy in patients with rheumatoid arthritis (RA) and treatment with gold or penicillamine is well established,^{1 2} but reports of patients with RA developing membranous nephropathy in the absence of such treatment are few,³ and in all the arthropathy preceded evidence of nephropathy.

We report a patient who presented with a nephrotic syndrome due to membranous nephropathy proved by biopsy and within months developed features fully consistent with a diagnosis of rheumatoid arthritis.

Case report

In June 1985 a 62 year old retired maintenance foreman presented with swelling of his legs and was found to have a nephrotic syndrome. Blood pressure was 160/95 mmHg. He had experienced mild Raynaud's phenomenon for several years but with no arthritic symptoms or rashes and had not received gold, penicillamine, non-steroidal anti-inflammatory drugs, or other agents associated with glomerular disease and had no other significant past medical history.

Investigations showed proteinuria 7 g/24 h, plasma albumin 16 g/l, plasma creatinine 101 µmol/l, creatinine clearance 65 ml/min, Wasserman reaction negative, hepatitis B surface antigen not detected, antinuclear factor (ANF) negative, anti-DNA antibodies negative, fasting blood sugar 4.7 mmol/l, and there was no increase in urinary free light chains. A sheep cell agglutination test (SCAT) and latex test for rheumatoid factor were strongly positive at >256 IU and >64 IU respectively.

Percutaneous renal biopsy showed the characteristic changes of membranous nephropathy with thickening of the peripheral capillary basement membranes and subepithelial spikes in the absence of increased cellularity. Immunofluorescent studies demonstrated IgG, C3, and C1q only, deposited in diffuse, global, and granular fashion along the glomerular basement membranes.

He was treated initially with a high protein, high calorie, low sodium diet, and conventional diuretic agents. When the histological diagnosis was made treatment was started with prednisolone 100 mg orally on alternate days; proteinuria fell to less than 1 g/24 h within six weeks, plasma albumin rose to 29 g/l, and creatinine clearance remained stable. Three months after prednisolone was started, which by then had been reduced to 10 mg on alternate days, he developed stiff and painful joints of his hands, ankles, and shoulders. The symptoms were worse in the morning and lessened during the day.

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Physical examination showed diffuse fusiform swelling of all fingers and tenderness of the wrists. SCAT and latex tests remained strongly positive, ANF and anti-DNA antibodies were negative repeatedly, and C3 and C4 levels remained normal. Radiographs of knees, hands, and shoulders showed no evidence of erosive arthropathy, but periarticular osteoporosis of the metacarpophalangeal and proximal interphalangeal joints was noted. Prednisolone dosage was increased to 15 mg orally daily, and chloroquine 250 mg orally daily was added, with some symptomatic improvement. He subsequently developed a peripheral neuropathy and cutaneous vasculitis, which required treatment with cyclophosphamide.

Discussion

This patient, with no evidence of systemic lupus erythematosus or other systemic disease associated with membranous nephropathy, infection, malignancy, or drug exposure, presented with a nephrotic syndrome due to membranous nephropathy (proved by biopsy) in the presence of strongly positive rheumatoid factor. Over the course of the next few weeks he developed the clinical features typical of RA.

Some studies suggest that membranous nephropathy only develops in patients with RA who are exposed to gold or penicillamine.⁴ This view is not

held universally, however,⁵ and we believe that our patient provides further evidence to support the hypothesis that membranous nephropathy can develop independently of treatment in patients with RA.

The presentation of RA with such extra-articular manifestations as an isolated pleural effusion is well recognised, yet in all previous reports of membranous nephropathy in patients with RA without gold or penicillamine exposure the arthropathy had been manifest before the nephropathy.³ We suggest that in our patient a nephrotic syndrome was the extra-articular presenting feature, an observation not previously reported, with the articular features becoming overt subsequently.

References

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