A Case of Rheumatoid Arthritis with Renal Tubular Amyloidosis

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Dear Sir,

Renal amyloidosis is well known as one of the most frequent renal involvements in rheumatoid arthritis (RA) [1]. Conversely, rheumatic disease – notably RA and juvenile RA – is also regarded to be the most common cause of reactive amyloidosis [2]. Most of the patients are considered to have developed amyloidosis due to the gradual increase of urinary protein, up to the nephrotic range and progressive renal dysfunction. To the best of our knowledge [3-7], we present the first case of RA with unusual renal distribution of amyloid A deposits (predominant and massive deposition along the tubular basement membrane).

A 40-year-old Japanese female with RA since 1974 was admitted in October 1990 because of progressive renal dysfunction. Her urinalysis and renal function had been within normal limits (creatinine, 0.9 mg/100 ml) until May 1988. In February 1989, renal dysfunction (serum creatinine, 1.5 mg/100 ml) was found. Since then, renal function gradually deteriorated to reach a level of 4.4 mg/100 ml in September 1990. During the whole duration of the progressive renal dysfunction, minimal abnormality was observed in the urinalysis and urinary volume. The anti-inflammatory drugs prescribed for RA had not been changed. Treatment with gold, D-penicillamine or steroid had not been carried out over these 10 years.

At the time of admission, she was free from any symptoms except for mild to moderate arthralgia (bilateral ankle, wrist and shoulder joints). Physical examination revealed a blood pressure of 126/80 mm Hg, pulse rate of 72/min, and moderate anemia. Swan neck finger deformity was observed on both hands. The laboratory findings on admission were as follows; urinalysis: protein (+, 0.2 g/day), blood (-), sugar (-), Bence-Johns protein (-), β_2 -microglobulin 2,720 μ g/l, N-acetyl- β -D-glucosaminidase (NAG) 6.6 U/l; CBC: WBC $7.0 \times 10^7/\text{mm}^3$, RBC $2.98 \times 10^6/\text{mm}^3$; hemoglobin 9.2 g/100 ml; ESR 75 mm/h; blood chemistry: total protein 7.0 g/100 ml, albumin 3.9 g/100 ml, BUN 36.0 mg/100 ml, creatinine 3.3 mg/100 ml, uric acid 5.7 mg/100

ml; serum chemistry: CRP 883 µg/ml. RA test (+), LE test (-), ANA (+), C3 69 mg/100 ml, C4 44 mg/100 ml, IgG 2,380 mg/100 ml, IgA 555 mg/100 ml, IgM 280 mg/100 ml; renal function: C_{cr} 18.2 ml/min, CpAH 68.3 ml/min; ultrasound: kidney size right 96 mm, left 89 mm, heart normal: electrocardiography showed no particular change, and immunoelectrophoresis no monoclonal protein.

On the 7th hospital day, percutaneous renal biopsy was performed. The most prominent finding was diffuse thickening of the tubular basement membrane (TBM) by homogeneous eosinophilic materials (fig.1).

Fig. 1. Light-microscopic finding of renal biopsy specimen. HE. × 167.

This material was stained pale orange by Congo red. There was widening of the interstitium due to fibrosis and small round cell infiltration. Small amounts of Congo-red-positive material were also observed within the interstitium and along the wall of small arteries. The specimen contained 18 glomeruli. Nine of them were totally hyalinized. Congo-red-positive deposit was segmentally observed in only one glomerulus. The remaining glomeruli showed minor abnormalities. Peroxidase anti-peroxidase (PAP) method revealed that the deposits were strongly positive for anti-amyloid A protein antibody, but neg-

ative for anti-κ and -λ. Immunofluorescence showed mild IgA and C3 deposits in glomeruli with a mesangial pattern. IgG, IgM, C4, C1q and fibrinogen were negative. Typical amyloid fibrils were observed within the tubular basement membrane by electron microscopy. We have described the first case of RA complicated with massive AA-type amyloid deposits along the tubular basement membrane and minimal deposits in glomeruli. The patient was not expected to have renal amyloidosis because of the small amount of urinary protein. The case unearthed important clinical criteria for the diagnosis of renal amyloidosis and provided some new information concerning the intrarenal distribution of amyloid deposits.

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