Dear Sir,

Glomerulonephritis (GN) is rarely a complication of rheumatoid arthritis (RA). In fact, GN was diagnosed in only 4 of 5,232 patients with RA at the UCLA Center for the Health Science [1]. We experienced 14 patients with RA complicated with hematuria and/or proteinuria, among 132 patients with RA, and describe here 2 patients with IgA nephropathy (IgA N) among them.

Patient 1, a 45-year-old woman, first noticed polyarthritis in 1971. Until 1983, nonsteroidal anti-inflammatory drugs (NSAID), gold and corticosteroids had been administered. Hematuria and proteinuria were initially indicated in July 1983 and she was admitted to hospital in October 1983. Upon admission, she was diagnosed as having classical RA (class 2; stage III). Laboratory data (table I) revealed microscopic hematuria and proteinuria, and high levels of IgA containing circulating immune complexes (CIC) (IgA-CIC, normal range: 0–3 µg/ml). On renal biopsy, we demonstrated 13 glomeruli showing mesangial expansion without hypercellularity. Immunofluorescent microscopy revealed the mesangial deposition of IgA and C3, while electron microscopy revealed mesangial dense deposits (fig. 1).

Patient 2, a 62-year-old woman, initially became aware of polyarthralgia and fever, and developed RA in 1972. NSAID had been prescribed. The patient’s condition was complicated by hypertension in June 1978, and furosemide was administered. Hematuria and proteinuria were first indicated in June 1984, where upon the patient was admitted to Hospital. On admission, she was diagnosed as having classical RA (class 2; stage III) and renal insufficiency. Laboratory results (table I) revealed mild renal dysfunction with microscopic hematuria and high levels of IgG-CIC (normal range: 0–3 µg/ml), IgA-CIC, IgA-rheumatoid factor (IgA -RF, normal range: 0–5 U) and IgM -RF (normal range: 0–6 U). On renal biopsy, 5 glomeruli were found to be completely sclerotic, and 9 were characterized by mild mesangial hypercellularity and expansion. The vessels showed arteriolar hyaliniza-tion and intimal fibrosis. Mesangial depositions of IgA and electron-dense materials were observed. These results indicated a renal histopathological diagnosis of IgA N with arteriolar sclerosis.

Membranous GN associated with gold or penicillamine therapy or amyloidosis were often seen in RA. Although GN is a rare complication in RA, a well-documented case of proliferative GN in association with RA has been reported [1]. Recently, Sellars et al [2] reported that an increase of the mesangial matrix and/or cells was a dominant abnormality in some patients with RA, although mesangial IgA deposition was not observed. Our 2 cases were a typical form of IgA N.

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**IgA Nephropathy in Rheumatoid Arthritis**

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<th>Masashi Sato</th>
<th>Hiroomi Kojima</th>
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Although the pathogenesis of IgA N is still unknown, the dysfunction of the reticuloendothelial system (RES) was confirmed in IgA N [3]. We additionally reported a decrease of phagocytic activity of polymorphonuclear leukocytes (PMN) and the presence of intracytoplasmic inclusions in PMN in IgA N [4]. The RES function may yet be proven to be one of the most important factors in IgA N. On the other hand, a defective RES function [5] and PMN abnormalities [6], as demonstrated in IgA N, have been confirmed in RA.

The origin of IgA deposited in the mesangium, which is another important problem, is still unclear. However, our data suggest that IgA-CIC is more important than IgA-RF as far as glomerular mesangial deposition is concerned.

Sato/Kojima/Koshikawa

Table I. Laboratory data on admission

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
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<tr>
<td>WBC</td>
<td>5.0</td>
</tr>
<tr>
<td>Hb</td>
<td>13.5</td>
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<tr>
<td>Platelets</td>
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Fig. 1. Diffuse mesangial IgA deposition (inset, magnification × 200) and mesangial electron-dense deposits (arrow, magnification × 3,000) are noted.

References