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Effect of Low Dose-Steroid Therapy in Patients with IgA Nephropathy

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The effectiveness of low-dose steroid therapy has not been determined in patients with IgA nephropathy. Twenty IgA nephropathy patients whose serum creatinine levels ranged from 0.62 mg/dl to 1.58 mg/dl at the time of renal biopsy were studied. The patients, with mild inflammatory activities such as cellular and/or fibrocellular crescents, mesangial interposition with mononuclear cell infiltration, and interstitial inflammatory cell infiltration, were treated with prednisolone (20-30 mg/day). The patients were then divided into two groups according to changes in the serum creatinine levels after steroid therapy: group I, improved renal function or no significant changes in renal function; group II, deterioration of renal function. In all of the patients studied, serum creatinine levels did not significantly change during steroid therapy ($p = 0.4493$). However, proteinuria significantly reduced after 12 months of steroid therapy (1.0 ± 0.8 g/day vs. 0.5 ± 1.1 g/day, $p = 0.0202$). In addition, hematuria significantly reduced after 12 months of steroid therapy (30.0 ± 32.7 RBC/HPF vs. 6.1 ± 6.7 RBC/HPF, $p = 0.0032$). Blood pressure did not significantly change during steroid therapy. When serum creatinine levels were compared over the time course, the grade of arteriosclerosis was lower in group I than in group II (0.4 ± 0.5 vs. 0.8 ± 0.4, $p = 0.001$). In conclusion, our data suggested that low-dose steroid therapy for IgA nephropathy patients with mild inflammatory activities could reduce the amount of urinary protein excretion and prevent deterioration of renal function, provided the histological findings in the renal biopsies showed mild arteriosclerosis.

Key words: IgA nephropathy, corticosteroid, creatinine, proteinuria, arteriosclerosis

Introduction

Since IgA nephropathy was firstly described by Berger and Hinglasis in 1968[1], it is now recognized as one of the most common types of primary glomerulonephritis worldwide[2]. Although the prognosis of IgA nephropathy was considered as relatively good previously, long term observation revealed that 20-40% of IgA nephropathy patients could progress to end stage renal disease[3]. So far, no adequate therapeutic strategies have been reported.

Kobayashi et al[4] recently reported that steroid therapy has a long-term beneficial effect for stabilization of renal function in progressive IgA nephropathy with massive proteinuria of 1.0 g/day or more, when patients have a preserved renal function with a creatinine clearance (Ccr) of 70 ml/min or more, although the effectiveness of steroid therapy in IgA nephropathy is controversial.

In contrast, in patients with heavy proteinuria and preserved renal function, steroid therapy was recommended[5]. Pozzi et al[6] reported that steroid therapy reduced proteinuria and protected against deterioration in renal function in patients with IgA nephropathy, in a prospective randomized multicenter study. In addition, significant amelioration of histopathological changes was reported in IgA nephropathy patients who were received with steroid therapy[7]. However, the ideal steroid therapy dosage for IgA nephropathy has not been determined and it is essential to establish a gold standard steroid therapy protocol that has definite effects in

preventing the progression of IgA nephropathy and has minimal toxicity.

We therefore designed this study to assess the effectiveness of low-dose steroid therapy (20-30 mg/day) for IgA nephropathy patients with mild inflammatory activities such as cellular and/or fibrocellular crescents, mesangial interposition with mononuclear cell infiltration, and interstitial inflammatory cell infiltration, and to ascertain the clinical parameters that influence the effectiveness of steroid therapy.

Patients and Methods

Patients

The diagnosis of IgA nephropathy was based on the light microscopic findings showing mesangioproliferative changes, mesangial IgA and C3 deposition in an immunofluorescence study and the presence of electron dense deposits in the mesangial area with electron microscopy. Patients with systemic diseases such as diabetes mellitus, collagen disease, abnormal hyper gamma globulinemia and chronic liver disease were excluded from this study. We selected 20 patients whose serum creatinine levels ranged 0.62 mg/dl to 1.52 mg/dl at the time of renal biopsy, and investigated the clinical course after the renal biopsy. Clinical data were evaluated with age at the time of renal biopsy and blood pressure, and laboratory data including serum creatinine (mg/dl) and urinary protein excretion (g/day) at the time of renal biopsy were also evaluated.

Blood pressure was usually maintained at under 140 mmHg in the systole, and 90 mmHg in the diastole with a calcium channel blocker, an angiotensin-converting enzyme inhibitor (ACE-I). The patients were divided into two groups according to changes in renal function evaluated by the serum creatinine levels over the period of steroid therapy: group I, improved renal function or no significant changes in renal function (serum creatinine after 12 months of steroid therapy did not change or decreased significantly, compared with those in the initiation of steroid therapy); group II, deterioration of renal function (serum creatinine after 12 months of steroid therapy increased significantly, compared with those in the initiation of steroid therapy).

Histological examination of renal biopsy specimens

All specimens were obtained with the percutaneous needle biopsy method. The specimens were fixed with 10% phosphate-buffered formalin (pH 7.2), embedded in paraffin, and cut into 2 μm sections. Hematoxylin and eosin, periodic acid Schiff (PAS), silver methenamine, and Masson trichrome stainings were performed for light microscopy.

Each specimen was evaluated for glomerular, interstitial, and vascular changes as previously described[8]. The changes were scored semiquantitatively by two independent observers without any knowledge of the clinical data. The percentages of glomeruli exhibiting glomerular obsolescence, crescent formation, and glomerular tuft adhesion to Bowman's capsule were estimated. Mesangial cell proliferation in each patient was semiquantitatively graded into four degrees of severity: grade 0, no abnormality; grade 1, mild; grade 2, moderate; and grade 3, severe. The extent of interstitial fibrosis was semiquantitatively graded into four categories according to the proportion of fibrotic lesions to the total cortical area: grade 0, less than 5% of total cortical area; grade 1, 5-20% ; grade 2, 20-40% ; and grade 3, more than 40%. Arterio-arteriolosclerotic changes were also evaluated using a similar grading scale: grade 0, no abnormality; grade 1, mild; grade 2, moderate; and grade 3, severe. The arterio-arteriolosclerosis indicate the hyaline deposition in their intima and media.

Protocol of the treatment

Oral prednisolone treatment was indicated in the 20 patients who met the following criteria : (1) proteinuria: 0.29-1.54 g/day, and (2) mild inflammatory activities: presence of cellular and/or fibrocellular crescents, mesangial interposition with mononuclear cell infiltration, and interstitial inflammatory cell infiltration. The patients were initially treated with a dose of prednisolone 20-30 mg/day for the first 4 weeks, and the dose was gradually reduced to 10-20 mg on alternate days during the next 11 months. An anti-platelet agent (dipyridamole or zilazep hydrochloride) was administrated in combi-

Table 1 Clinical characteristics of steroid-treated patients with IgA nephropathy at the time of renal biopsy

Case	Group	Age (year)	Male/ Female	Steroid (mg/day)	U-P (g/day)	U-RBC (/HPF)	S-Cr (mg/dl)	SBP (mmHg)	DBP (mmHg)
1	II	38	F	30	0.8	6	0.8	110	70
2	II	30	F	20	3.1	100	0.8	123	83
3	I	37	M	20	1.0	30	1.5	128	65
4	II	45	F	20	0.3	3	0.8	106	73
5	II	52	F	20	0.3	30	0.8	113	72
6	I	36	F	30	0.6	0	0.9	106	68
7	II	26	F	20	1.3	20	0.7	114	68
8	II	25	F	20	0.7	20	0.8	118	80
9	I	27	M	20	1.5	100	1.0	128	76
10	II	44	F	20	1.5	100	1.1	133	85
11	II	53	F	30	0.6	0	0.7	114	70
12	I	26	M	20	0.5	2	1.2	141	78
13	II	47	F	20	1.4	30	0.6	120	82
14	II	49	F	20	3.0	30	0.7	100	68
15	I	47	M	20	0.5	20	1.2	150	90
16	II	42	F	20	0.5	3	0.8	135	102
17	II	43	F	20	0.4	30	0.7	152	82
18	I	51	F	20	0.4	40	0.8	115	77
19	I	34	M	30	0.3	30	1.6	105	60
20	II	32	F	20	1.4	5	1.3	112	62
mean ± SD		39.2 ± 9.4			1.0 ± 0.8	30.0 ± 32.7	0.9 ± 0.3	121 ± 15	76 ± 10

U-P: proteinuria, U-RBC: urinary red blood cells, S-Cr: serum creatinine, SBP: systolic blood pressure, DBP: diastolic blood pressure, Steroid: initial dose of therapy.

nation with prednisolone in all the patients. An ACE-I was administered for the hypertensive patients (4 patients in the steroid group, 20%).

Statistical analysis

Data are given as means ± standard deviation (SD). The significance of differences between groups was examined with the Student's t-test for nonpaired samples and by the χ^2-test. The evolution of clinical parameters was analyzed in both groups by repeated-measurement analysis of variance (ANOVA). When differences could be demonstrated, values were compared with the baseline using the paired-sample t-test. A p value of less than 0.05 was considered statistically significant.

Results

Baseline characteristics of the 20 patients are summarized in Table 1, and the subjects consisted of 5 males and 15 females with a mean age of 39.2 ± 2.1 years. Prednisolone was started at an initial dose of 30 mg/day in 4 cases and 20 mg/day in 16 cases. The duration of the steroid therapy was 12 months in all patients. Daily proteinuria was more than 1.0 g/day in 8 cases, of which no patients had nephrotic-range proteinuria. The remaining 12 cases showed mild proteinuria of less than 1.0 g/day. Four cases showed mild elevation of serum creatinine ranging from 1.21 to 1.58 mg/dl.

The comparison of the clinical findings of the patients in each group at the time of renal biopsy is summarized in Table 2. The age, grade of proteinuria and hematuria, serum creatinine concentration, and blood pressure were not different between both groups. Female gender rate in group II was higher than those in group I. Six cases were complicated with hypertension and ACE-I was used in 2 cases of group I and 4 of group II. No significant differences in blood pressure control and renal function were observed in patients with or without antihypertensive agents, suggesting that antihypertensive therapy may not influence on changes in proteinuria.

Changes in renal function, daily proteinuria, grade of hematuria and blood pressure in the 20 cases were observed. In all of the patients studied,

Table 2 Comparison of clinical data at the start of steroid therapy between group I and group II

	Group I (n=7)	Group II (n=13)	p-value
Male/Female	4/3	1/12	
Age (year)	36.9±9.4	40.5±9.5	0.9702
U-P (g/day)	0.7±0.4	1.2±0.9	0.3522
U-RBC (/HPF)	31.7±33.6	29.0±33.6	0.7528
S-Cr (mg/dl)	1.1±0.4	0.8±0.2	0.1113
SBP (mmHg)	125±17	120±13	0.4159
DBP (mmHg)	73±10	77±10	0.6857

mean±SD, U-P: proteinuria, U-RBC: urinary red blood cells, S-Cr: serum creatinine, SBP: systolic blood pressure, DBP: diastolic blood pressure.

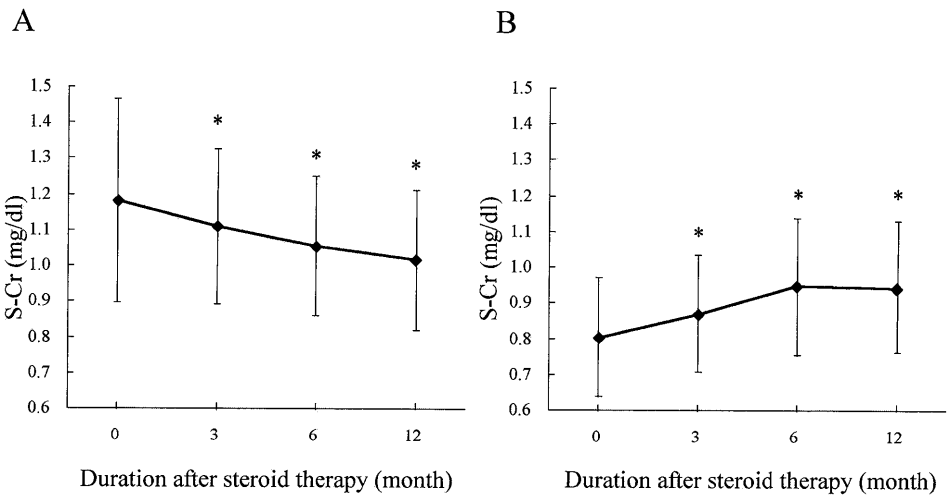

Fig. 1 Changes in serum creatinine (S-Cr) levels during steroid therapy in two group
A: group I (n=7), improved renal function or no significant changes in renal function (serum creatinine after 12 months of steroid therapy did not change or decreased significantly, compared with those in the initiation of steroid therapy).
B: group II (n=13), deterioration of renal function (serum creatinine after 12 months of steroid therapy increased significantly, compared with those in the initiation of steroid therapy).
*$p<0.05$ vs. values at the start of therapy.

serum creatinine levels did not significantly change during steroid therapy ($p=0.4493$). However, proteinuria significantly reduced after 12 months of steroid therapy (1.0±0.8 g/day vs. 0.5±1.1 g/day, $p=0.0202$). In addition, hematuria significantly reduced after 12 months of steroid therapy (30.0±32.7 RBC/HPF vs. 6.1±6.7 RBC/HPF, $p=0.0032$).

Then, clinical parameters during steroid therapy were compared between group I and group II. As shown in Fig. 1A, serum creatinine levels in group I did not significantly change during steroid therapy (1.2±0.3 mg/dl at the initiation vs. 1.0±0.2 mg/dl after 12 months, $p=0.0313$). However, serum creatinine levels in group II increased significantly during steroid therapy (0.8±0.2 mg/dl vs. 1.0±0.2 mg/dl, $p<0.0001$, Fig. 1B). Daily proteinuria in group I significantly reduced after 12 months of steroid therapy (07±0.4 g/day vs. 0.1±0.3 g/day, $p=0.0191$, Fig. 2A). However, daily proteinuria in group II did not change after 12 months of steroid therapy (1.2±0.9 g/day vs. 0.7±1.3 g/day, $p=0.1297$, Fig. 2B). As shown in Fig. 3A, the grade of hematuria did not change after 12 months of steroid therapy (31.7±33.6 RBC/HPF vs. 6.7±6.7 RBC/

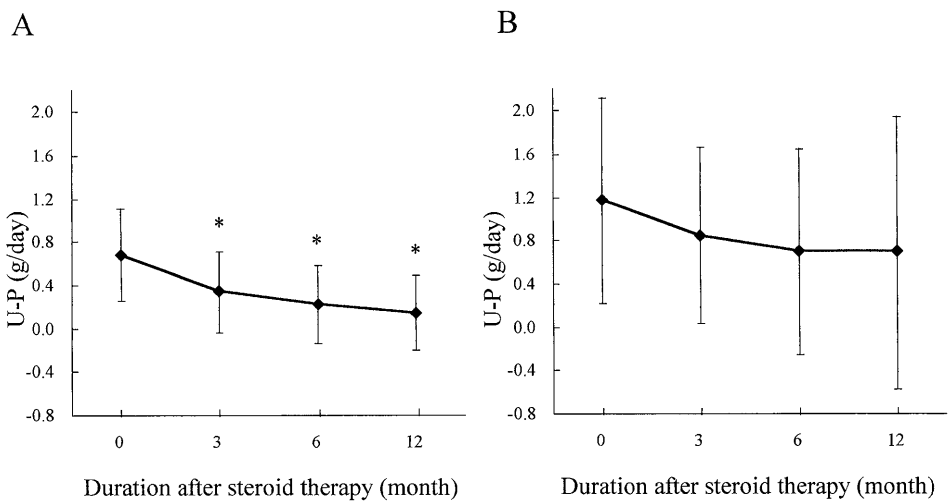

Fig. 2 Changes in daily proteinuria (U-P) during steroid therapy in two groups
A: group I (n = 7), improved renal function or no significant changes in renal function (serum creatinine after 12 months of steroid therapy did not change or decreased significantly, compared with those in the initiation of steroid therapy).
B: group II (n = 13), deterioration of renal function (serum creatinine after 12 months of steroid therapy increased significantly, compared with those in the initiation of steroid therapy).
*$p < 0.05$ vs. values at the start of therapy.

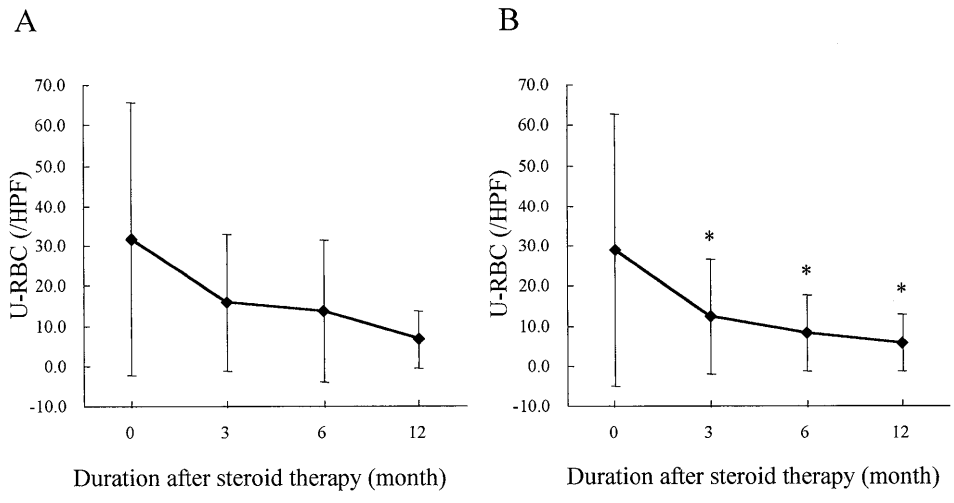

Fig. 3 Changes in the grade of hematuria (U-RBC) during steroid therapy in two groups
A: group I (n = 7), improved renal function or no significant changes in renal function (serum creatinine after 12 months of steroid therapy did not change or decreased significantly, compared with those in the initiation of steroid therapy).
B: group II (n = 13), deterioration of renal function (serum creatinine after 12 months of steroid therapy increased significantly, compared with those in the initiation of steroid therapy).
*$p < 0.05$ vs. values at the start of therapy.

HPF, p = 0.0851), whereas the grade of hematuria reduced significantly after 12 months of steroid therapy (29.0 ± 33.6 RBC/HPF vs. 5.8 ± 7.0 RBC/HPF, p = 0.0246, Fig. 3B). As shown in Fig. 4A and 4B, systolic and diastolic blood pressures in both groups did not significantly change during steroid

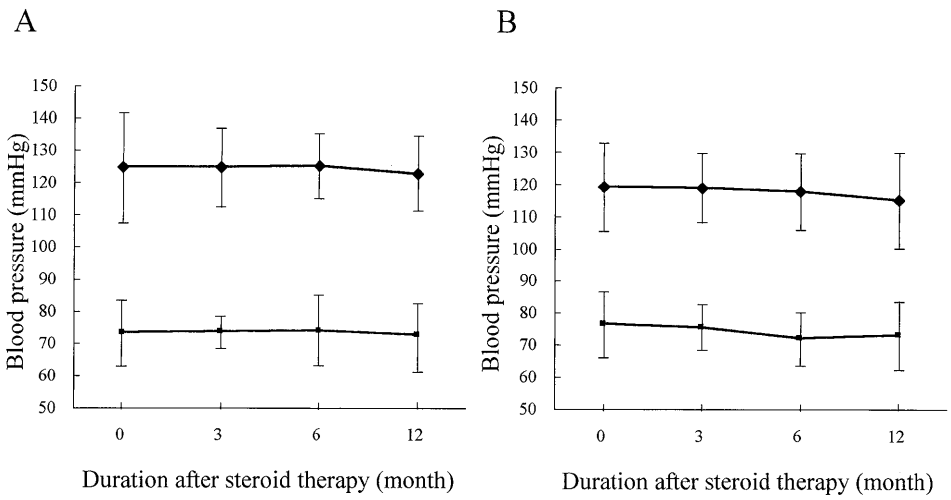

Fig. 4 Changes in systolic (◆) and diastolic (■) blood pressure during steroid therapy in tow groups
A: group I (n = 7), improved renal function or no significant changes in renal function (serum creatinine after 12 months of steroid therapy did not change or decreased significantly, compared with those in the initiation of steroid therapy).
B: group II (n = 13), deterioration of renal function (serum creatinine after 12 months of steroid therapy increased significantly, compared with those in the initiation of steroid therapy).

Table 3 Comparison of histological findings between group I and group II

	Group I (n = 7)	Group II (n = 13)	p-value
Glomerular obsolescence	19.9 ± 27.1	24.9 ± 21.1	0.4600
Adhesion	19.6 ± 24.4	26.8 ± 14.1	0.8996
Crescent formation	1.4 ± 3.8	10.9 ± 8.5	0.1340
Mesangial proliferation	1.3 ± 0.5	1.5 ± 0.5	0.6891
Interstitial fibrosis	1.0 ± 0.8	1.8 ± 0.6	0.1723
Vascular changes	0.4 ± 0.5	0.8 ± 0.4	0.0010

mean ± SD.

therapy.

The comparison of the histological findings in the renal biopsies in both groups is summarized in Table 3. No significant differences were observed in the percentage of glomerular obsolescence, percent of glomerular crescents, grade of mesangial cell proliferation and the extent of interstitial fibrosis. However, the grade of vascular changes was lower in group I than in group II (0.4 ± 0.5 vs. 0.8 ± 0.4, p = 0.001).

Discussion

The main purpose of this study was to evaluate the effectiveness of low-dose steroid therapy for IgA nephropathy patients with mild inflammatory activities such as cellular and/or fibrocellular crescents, mesangial interposition with mononuclear cell infiltration, and interstitial inflammatory cell infiltration. The present study demonstrated that low-dose steroid therapy significantly reduced proteinuria and hematuria in association with no changes in serum creatinine levels in the IgA nephropathy patients studied. Moreover, the patients in whom renal function improved or showed no significant changes were the cases who had minor vascular lesions such as arteriosclerosis. In the present study, the primary outcome was a change in urinary protein excretion from the base-line because it has been proven that the degree of proteinuria is

one of the most important prognostic indicators in IgA nephropathy[9)~11)].

Pozzi C et al[6)] reported that the steroid therapy for IgA nephropathy was effective when the patient met the following clinical criteria; serum creatinine level was under 1.5 mg/dl, and amount of urinary protein excretion was 1.0-3.5 g/day. The patients treated with steroid therapy (40-50 mg/day as an initial dose) showed a significant reduction in the amount of urinary protein excretion (2.0 ± 0.6 g/day to 0.67 ± 0.5 g/day) and a higher percentage of renal survival after steroid therapy. They suggested that the effect of steroid treatment was associated with a decrease in urinary protein excretion. Our regimen using low-dose prednisolone (20-30 mg/day as an initial dose) had an antiproteinuric effect, but its effect was not sufficient to improve renal function. An insufficient dose of prednisolone in our protocol may be the reason for the discrepancy between the effects on proteinuria and the serum creatinine levels.

Ballardie and Roberts[12)] reported a controlled trial of prednisolone. They included only patients with impaired renal function (serum creatinine > 1.5 mg/dl) in the study. The study showed a significant effect on improving renal outcome by treating patients with prednisolone (40 mg/day as an initial dosage) and cyclophosphamide for an initial 3 months, and then azathioprine, continued for a minimum of 2 years. One patient in the treatment group developed pulmonary tuberculosis and was successfully treated. There was no evidence of other adverse effects. It was concluded that their therapy was beneficial in improving the clinical course of progressive IgA nephropathy with an acceptably low risk for side effects.

Nolin and Courteau[5)] showed in their review that steroid therapy is only beneficial in selected IgA nephropathy patients with nephrotic syndrome and minor grade histological characteristics. However, patients with minor histological characteristics and IgA deposition and who clinically demonstrate nephrotic syndrome and an excellent response to steroids are supposed to have an overlapping syndrome of IgA nephropathy and lipoid nephrosis[13)].

When considering the effects of steroid therapy in IgA nephropathy, we should distinguish between such patients and those with massive proteinuria caused by IgA nephropathy.

Recently, clinical studies in Japan reported that steroid therapy (40-50 mg/day as an initial dose) was beneficial for IgA nephropathy even if the disease was in the advanced stage. Tamura et al[14)] observed 8 patients with impaired renal function, and showed that the urinary protein excretion significantly decreased (2.57 ± 1.12 g/day to 1.12 ± 0.84 g/day, $p < 0.005$), and the serum creatinine levels did not change (2.76 ± 1.32 mg/dl to 2.56 ± 1.10 mg/dl) after treatment. They suggested that steroid therapy could delay the progression to end-stage renal failure. Tomiyoshi et al[15)] suggested that steroid therapy (40-50 mg/day as an initial dose) was effective for IgA nephropathy patients who showed a high percentage of cellular and fibrocellular crescents and/or segmental glomerular sclerosis, although their Ccr was under 70 ml/min. However, the effectiveness of low-dose steroid therapy for IgA nephropathy remains unclear.

Previously, we have reported the histological indices that predict the effectiveness and ineffectiveness of steroid therapy (40-50 mg/day as an initial dose) in IgA nephropathy[16)]. According to that study, the severity of acute glomerular extracapillary lesions (cellular and/or fibrocellular crescents) predicted the possibility of proteinuria reduction, while the severity of interstitial inflammation suggested a high risk of renal dysfunction even after steroid therapy. However, the efficacy of the indication of steroid therapy for IgA nephropathy patients with mild inflammatory activitives such as crescent formation and interstitial change has not been elucidated. To reduce the side effects of steroid therapy, we used low-dose prednisolone for IgA nephropathy patients with mild inflammatory lesions. In the present study, no significant differences were observed in the percentage of glomerular obsolescence, percent of glomerular crescents, grade of mesangial cell proliferation and the extent of interstitial fibrosis in two groups divided by the serum creatinine levels. However, the grade of arte-

riosclerosis was lower in group I than in group II, suggesting that the extent of vascular lesions such as hyalinosis and arteriosclerosis in the renal vasculature may be a marker for the effectiveness of low-dose steroid therapy in patients with mild inflammatory activities.

In conclusion, we suggest that low-dose steroid therapy for IgA nephropathy patients with mild inflammatory activities such as cellular and/or fibrocellular crescents, mesangial interposition with mononuclear cell infiltration, and interstitial inflammatory cell infiltration could reduce the amount of urinary protein excretion and may restore the residual renal function, if the histological findings in renal biopsies showed mild vascular lesions. Careful histological interpretation of renal biopsy specimens is necessary to determine the indication for low-dose steroid therapy in combination with the clinical evaluation of the disease activity in IgA nephropathy patients. We consider that low-dose steroid therapy is one of the beneficial strategies for IgA nephropathy patients with mild inflammatory activities and mild vascular lesions during every clinical stage of IgA nephropathy.

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IgA 腎症患者に対する低用量ステロイド剤の有効性に関する検討

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IgA 腎症患者に対するステロイド剤の有効投与量の決定に関しては不明な点が多い．今回われわれは，腎生検時の血清クレアチニン値が 0.62 mg/dl から 1.58 mg/dl の範囲の IgA 腎症患者 20 例を対象に，低用量ステロイド剤の有効性について検討した．腎生検で軽度の炎症性変化を示した症例に，1 日 20～30 mg のステロイド剤の投与を行った．ステロイド治療前後の血清クレアチニン値の変化によって 2 つの群に分類した．グループ 1 は血清クレアチニン値が低下または変化がなかった群，グループ 2 は血清クレアチニン値が上昇した群である．検討した 20 例すべてにおいて，ステロイド治療による血清クレアチニン値の有意な変化は認められなかった．しかし，ステロイド治療 12 ヵ月後に蛋白尿は有意に減少した（1.0±0.8g/day vs. 0.5±1.1g/day，p＝0.0202）．また，ステロイド治療 12 ヵ月後に血尿は有意に減少した．(30.0±32.7RBC/HPF vs. 6.1±6.7RBC/HPF，p＝0.0032)．ステロイド治療中の収縮期および拡張期血圧の有意な変化はなかった．腎生検所見を比較すると，グループ 2 に比しグループ 1 で動脈硬化の程度は軽度であった(0.4±0.5 vs. 0.8±0.4，p＝0.001)．今回の結果から，腎生検で軽度の炎症性変化を示し，軽度の血管病変を有する IgA 腎症患者では，低用量ステロイド剤治療により，蛋白尿が有意に減少し腎機能障害の進展が阻止される可能性が示唆された．