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IgA Nephropathy

—Analysis of 94 cases emphasizing clinicopathologic correlation—

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Introduction

Since Berger (1969) first described nephropathy with mesangial IgA-IgG deposits, the lesion has been generally regarded as a distinct clinicopathologic entity.

This disorder is usually termed as "IgA nephropathy" or "Berger's disease" and is characterized by glomerular mesangial IgA deposit as the main immunoglobulin, without evidence of systemic diseases such as lupus erythematosus or anaphylactoid purpura.

The incidence of IgA nephropathy among the primary glomerulonephritides varies from 2.1% (Hyman et al., 1973) to 43.3% (Nakamoto et al., 1978). This disease is a frequent form of primary glomerular nephropathy in France (Berger, 1969; Berger et al., 1975; Morel-Maroger et al., 1972), Italy (D'Amico et al., 1981), Australia (Woodroffe et al., 1980), Singapor (Sinniah et al., 1976, 1979), and Japan (Nakamoto et al., 1978), while in America, England and Canada, its incidence is low (Hyman et al., 1973; McCoy et al., 1974; Finlayson et al., 1975; Sissons et al., 1975; Alexander et al., 1977; Hood et al., 1981).

The first case in Korea was discovered in 1974 (Chung et al.) and sporadic case reports were followed thereafter. However, it is still not known how often this disease would occur among general population in Korea. It was our impression that this disease is fairly common. Accordingly this study was conducted to assess the relative frequency of this disorder in this country and also to describe the clinical findings and morphologies of this disease.

Material and methods

Between July 5, 1979 and October 5, 1981, a total of 450 kidney biopsies were collected at the Department of Pathology, Seoul National Unviersity, from 11 major hospitals in Korea. Of these, the tissues of 368 cases (M:F=2.1:1) were sufficient for complete immunofluorescence studies.

Among them we found 94 patients who showed diffuse mesangial IgA deposits as a main immunoglobulin. None of these patients presented systemic lupus erythematosus, Henoch-Schoenlein purpura, or other systemic diseases. For these cases full clinical evaluations including blood pressure readings and laboratory investigations were performed. Laboratory studies included hemogram, urinalysis, serum electrolytes, blood urea nitrogen, serum creatinine, creatinine clearance, and quantitative protein excretion. Serum level of the third component of complement (C3) was obtained by radial immunodiffusion technique using monospecific

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antisera (Hyland Lab.)

Needle biopsy specimens of the kidneys were divided into three portions, each for light, electron, and immunofluorescence microscopic studies. The renal lesions were classified histologically into six categories adopted from Zollinger and Mihatsch(1978), and Churg(1981). They were minimal (nil) lesion, minor change with increased mesangial matrix and cells, diffuse mesangial proliferative glomerulonephritis, focal and segmental proliferative glomerulonephritis, membranoproliferative glomerulonephritis, and diffuse sclerosing glomerulonephritis. Minimal glomerular lesion was diagnosed when light microscopy was essentially normal. Minor change was diagnosed when light microscopy showed increased cellularity in mesangial areas (up to 3 cells/area), widening of the mesangium (up to twice normal) or occasional capillary wall abnormalities. Diffuse mesangial proliferative glomerulonephritis was characterized by uniform (>80%) increase in mesangial cells (with clusters of 4 cells or more per mesangial area) accompanied by an increase in mesangial matrix with patent capillary lumens. Focal and segmental proliferative glomerulonephritis was characterized by segmental subendothelial deposits and basement membrane doubling with mesangial interpositon and occasional loop necrosis as well as segmentally pronounced mesangial proliferation. Some of the segmental glomerular lesions were covered by crescents. Segmentally un changed glomeruli showed no or minor glomerular lesions. Membranoproliferative glomerulonephritis was characterized by circumferential mesangial interposition, variable amount of mesangial cell proliferation and increased matrix. Diffuse sclerosing glomerulonephritis was diagnosed when light microscopy showed widespread sclerosis(>80%) of glomeruli with evidence that the basic process is of glomerular origin.

In addition, the number of glomeruli showing global sclerosis, segmental sclerosis and hyalinosis, crescent formation, loop necrosis, synechia, presence of mesangial red globules, tubular atrophy, interstitial infiltrations and fibrosis, and vascular changes were also counted.

Light microscopy: Renal specimens were fixed in 10% neutral buffered formalin, embedded in paraffin and sectioned at 2-to 3μm. The sections were stained with hematoxylin-eosin, periodic acid shiff (PAS), Masson trichrome, and periodic acid and silver methenamine counterstained with hematoxylin eosin (PASM).

Immunofluorescence: Immunofluorescent studies were done by the direct method (Sinniah et al., 1976). The renal tissue was fast frozen in mounting medium (Ames Tissue Tek) on cryostat cutting platform by spraying it with cryokwik. Cryostat sections were cut at 4μm and fixed in ether-alcohol(1 : 1 ratio) for 20 minutes, 95% alcohol for 10 minutes, and phosphate buffered saline (pH 7.4) for five minutes. They were then stained with commercially purchased fluoresceinated monospecific antisera (Hoechst Behring Laboratories, and Hyland Laboratories) to human IgG, IgA, IgM, C3, C4, fibrinogen and albumin(all FITC-labeled antisera were tested for monospecificity by immunoelectrophoresis against normal human serum). And they were incubated in a humid chamber for 45 minutes and washed with phosphate buffered saline in medium-speed agitator for 15 minutes. The sections were covered with veronal acetate buffer (pH 8.6) and coverslips and examined under an American Optical Fluorolume microscopy.

The intensity of fluorescence was rated on a scale from 0 to 3+ as follows: 0=negative; ±=minimal; +=slight; 2+=moderate, and 3+=marked.

Electron microscopy: Specimens were fixed in 4% glutaraldehyde at 4°C, postfixed in 1% Dalton's chromium-osmium fixative and embedded

in araldite. Ultrathin sections stained with uranyl acetate and lead citrate were examined and photographed with Hitachi H-500 electron-microscope. The dense deposits were evaluated according to their location: subendothelial, intramembranous, paramesangial or mesangial. Cellularity, thickening and reduplication of capillary walls, and presence of tubulovesicular bodies were also checked.

RESULTS

1. Clinical features

Among the 368 biopsy population in which we had done complete immunofluorescence studies, IgA nephropathy was encountered in 94 cases (25.5%). Excluding 30 cases of lupus nephropathy, Henoch-Schoenlein purpura, or other systemic diseases, its frequency among primary glmerular diseases was 27.8%.

There were 64 males and 30 females (M:F= 2.1:1) aged 27.7±9.2 and 26.1±11.1 years respectively. The mean age of the 94 patients, males and females, was 27.2±9.2 with a range of 10 to 54 years. Eight patients were under the age of 15 years.

Fourty-five patients (47.9%) had intermittent gross hematuria, and the attacks were associated with upper respiratory tract infections in 21 patients (22.3%). One patient had persistent gross hematuria. And 89 patients (94.7%) exhibited intermittent or persistent microscopic hematuria. Only 5 patients had no hematuria, either gross or microscopic. All patients had proteinuria at the time of biopsy, ranged from 0.18 to 13.3 gm/day, and 19 patients (20.2%) had nephrotic range proteinuria (>3.5 gm/day). Renal function was impaired (serum creatinine>1.5 mg/dl) in 13 patients (13.8%) in which 10 patients revealed values over 2 mg/dl. Hypertension (> 140/90mm Hg) was present in 17 cases (18.1 %). Abnormal granular, waxy, or red blood cell casts in urine were seen in 17 patients (18.1%). Two patients had family history of renal disease. The serum levels of C3 were not decreased in all patients. The time interval between the initial presentation of the disease and biopsy ranged from less than 1 month to 13 years, with the mean interval of 1 year and 9 months.

Detailed clinical data of diffuse sclerosing glomerulonephritis were summarized in Table 1. The age ranged from 13 to 52 years with the

Table 1. Clinical data of diffuse sclerosing glomerulonephritis in IgA nephropathy

Case No.	Sex	Age (year)	Micro hema-turia	Gross hema-turia	Duration of known dis. before biopsy (year)	Abnormal urinary casts	Serum creatinine (mg/dl)	Urine protein (g/24hr(	Hyper-tension (>140/90)	Duration of follow up (year)	S Cr at the end of follow up(mg/dl)
1.	M	25	+	−	1/12	+	2.5	5.2	+	1	8.4 (Hemodialysis)
2.	M	25	+	−	1	−	2.2	1.9	+	1 6/12	2.5
3.	M	27	−	−	3 10/12	−	1.6	3.1	−	−	
4.	M	35	+	−	1/12	−	1.1	2.9	−	−	
5.	F	22	+	−	4	+	1.0	2.5	−	5/12	1.0
6.	M	52	+	−	10	−	3.3	3.9	+	1 10/12	11.5 (Hemodialysis)
7.	M	13	+	+	2 5/12	−	1.1	3.0	−	−	
8.	M	19	+	+	7	+	3.3	6.6	+	1	12.6 (Transplantation)
9.	F	47	−	−	1 6/12	−	3.0	3.2	+	2/12	4.0
(Mean)		29.4					2.1	3.6			

average of 29.4 years. Duration of the disease until biopsy ranged from 1 month to 10 years. Granular or waxy casts were seen in 3 cases. Average serum creatinine was 2.1 mg/dl and average 24 hr urine protein was 3.6gm. Three patients developed end stage renal failure within 1 year, and one of them received renal transplantation.

2. Histologic observation

Renal biopsies containing more than 5 glomeruli were included for light microscopic examination. The glomerular alterations are summarized in Tables 2 and 3.

Forty eight cases (51.0%) of the total IgA nephropathy showed minor changes with increased mesangial matrix and cells, whereas 18 cases (19.2%) showed diffuse mesangial proliferative glomerulonephritis, 11.7%(11) focal and segmental proliferative glomerulonephritis and 9.6% (9) diffuse sclerosing glomerulonephritis. Normal glomerular histology on light microscopic examination was observed in 7.4% (7) of the cases and membranoproliferative glomerulonephritis was observed in one patient. By careful examination of Masson trichrome stained sections small red globules in mesangium were detected in many cases, even in cases of minimal lesion. Detailed pattern of minor change, indicating the incidences of segmental and global sclerosis, segmental crescents and loop necrosis was shown in Table 3.

Among 18 cases of diffuse mesangial proliferative glomerulonephritis focal and global sclerosis was shown in one case, focal and segmental sclerosis in 4, focal segmental sclerosis/crescent in 2, focal segmental global sclerosis/crescent in 1, focal crescent in 1, and focal segmental global sclerosis in 9 cases, in two of which sclerosis (segmental and global) was over 65%. None of the diffuse mesangial proliferative glomerulonephritis was free from sclerosis or crescent. All cases of focal and segmental proliferative glomerulonephritis showed segmental sclerosis and fibrocellular crescents as well as focal marked tubular atrophy. Arteriolosclerosis was also seen in 2 cases. A case of membrano-

Table 2. Morphological patterns of glomerular lesions in IgA nephropathy

Morphological pattern	No. of cases	(% involved)
Minimal ("Nil") lesion	7	7.4
Minor change with increased mesangial matrix and cells	48	51.0
Diffuse mesangial proliferative GN	18	19.2
Focal and segmental proliferative GN	11	11.7
Membranoproliferative GN	1	1.1
Diffuse sclerosing GN	9	9.6
	94	100.0

* GN: glomerulonephritis

Table 3. Detailed pattern of minor change in IgA nephropathy

Mophorlogical pattern	No. of cases	(% involved)
Focal segmental sclerosis	10	20.8
Focal global sclerosis	9	18.7
Focal segmental and global sclerosis	9	18.7
Focal segmental sclerosis/crescent	2	4.2
Focal segmental and global sclerosis/crescent	2	4.2
Focal segmental loop necrosis (/crescent:1) (/global sclerosis:1)	3	6.3
Only increased mesangial matrix and cells	13	27.1
	48	100.0

proliferative glomerulonephritis was characterized by diffuse circumferential mesangial interposition and mesangial cell proliferation accompanied by focal segmental and global sclerosis. Tubular atrophy was severe. In all cases of diffuse sclerosing glomerulonephritis, segmental and global sclerosis comprised over 80% of whole glomeruli and uninvolved ones exhibited prominent mesangial cell proliferation and matrix increase suggesting the lesions were originated from mesangial proliferative glomerulonephritis. Tubules showed marked atrophy accompanied by areas of compensatory dilatation, and the interstitium was the seat of heavy lymphoplasma cell infiltration and fibrosis. Arterioles exhibited hyaline sclerosis in 5 cases.

As a whole, sclerosis (global and/or segmental) was seen in 75.5%(71) of the cases, crescent in 23.4%(22) and loop necrosis in 7.4%(7). Only 20 cases were free from sclerosis, crescent, or loop necrosis.

The histologic distribution in 8 children was: minimal (1 case), minor change (1 case), minor change with focal segmental sclerosis (1 case), diffuse mesangial proliferative glomerulonephritis with focal segmental sclerosis (2 cases), diffuse mesangial proliferative glomerulonephritis with focal segmental sclerosis/crescent (2 cases), and diffuse sclerosing glomerulonephritis (1 case). Among them two presented nephrotic syndrome and one hypertension but no case showed impaired renal function.

The incidence of nephrotic range proteinuira, impaired renal function, abnormal urinary casts, and hypertension according to histologic classification was shown in table 4. Though diffuse sclerosing glomerulonephritis showed higher proportion of poor clinical manifestation than others, it was statistically nonsignificant by Fisher's exact method possibly due to insufficient

Table 4. Clinicopathologic correlation in IgA nephropathy

Morphological pattern		Nephrotic range proteinuria (>3.5gm/day)	Impaired renal function (SCr>1.5mg/dl)	Abnormal urinary casts	Hypertension (BP>140/90mmHg)
		No. of cases(%)	No. of cases(%)	No. of cases(%)	No. of cases(%)
*Minimal lesion	(7)	—	—	—	—
*Minor change					
with FSScl	(10)	1 (10 %)	—	1 (10 %)	1(10 %)
with FGScl	(9)	4(44.4%)	1(11.1%)	1(11.1%)	1(11.1%)
with FSGScl	(9)	3(33.3%)	1(11.1%)	1(11.1%)	—
with FSN/Cr	(1)	—	—	1(100 %)	1(100 %)
with only increased mesangial matrix & cells	(13)	2(15.4%)	—	—	1(7.7%)
with other combination	(6)	—	—	—	—
*Diffuse mesangial proliferative GN					
with FSScl	(4)	—	1(25 %)	—	—
with FSScl/Cr	(2)	1(50 %)	—	1(50 %)	1(50 %)
with FSGScl	(9)	1(11.1%)	1(11.1%)	1(11.1%)	3(33.3%)
with Cr	(1)	—	—	1(100 %)	—
with other combination	(2)	—	—	—	—
*Focal and segmental proliferative GN	(11)	3(27.3%)	2(18.2%)	6(54.5%)	3(27.3%)
*Membranoproliferative GN	(1)	1(100 %)	1(100 %)	1(100%)	1(100 %)
*Diffuse sclerosing GN	(9)	3(33.3%)	6(66.7%)	3(33.3%)	5(55.6%)
Total No. of cases	(94)	19(20.2%)	13(13.8%)	17(18.1%)	17(18.1%)

F: focal G: global S: segmental Cr: crescent Scl: sclerosis N: necrosis
GN: glomerulonephritis

Table 5. Immunofluorescent findings

	No. of patients	(% involved)
Type of immunoglobulins		
IgA alone	26	27.7
IgA+IgG	40	42.5
IgA+IgM	14	14.9
IgA+IgG+IgM	14	14.9
	94	100.0
Mediators		
C_3	76	80.9
Fibrinogen	16	17.0
C_4	2	2.1

number of samples.

Immunofluorescence studies: Immunofluorescence findings are shown in Table 5. In all cases IgA was always the prominent immunoglobulin in staining intensity predominantly localized in mesangium. Thirty-three patients (36.2%) had mesangial plus capillary loop deposits of IgA. IgA was the only immunoglobulin localized in the glomeruli in 26 patients (27.7%). IgA in association with IgG in 40 cases (42.5%); IgA in association with IgM in 14 cases (14.9%), and IgA-IgG in association with IgM in 14 cases (14.9%). C3 was found in 76 cases (80.9%), fibrinogen in 16 cases (17.0%), and C4 in 2 cases (2.1%).

Electron microscopic studies: Electron microscopic studies were done on 50 cases. Mesangial and/or paramesangial electron dense deposits were seen in all including 4 cases with diffuse sclerosing glomerulonephritis. The intensity of these deposits correlated to the degree of mesangial IgA deposits in many cases. Less amount of isolated subendothelial deposit was seen in 18 out of 50 cases. Three cases showed a few subepithelial humps. Mesangial matrix was increased in most cases. One case of light microscopically minimal lesion exhibited segmental increase in mesangial matrix and cells. Segmental mesangial interposition was seen in 3 out of 50 cases. Tubulovesicular bodies were seen in one case. And endothelial arcade formation was demonstrable in 38 cases. Foot process fusion and microvilli formation were seen in all cases.

DISCUSSION

The relative frequency of IgA nephropathy among the primary glomerular diseases shown in this study was 27.8%. This datum is comparable to that of Italy (D'Amico et al., 1981) but is lower than those of Japan and Singapore (Nakamoto et al., 1978; Sinniah, 1979).

Because our series were collected from 11 hospitals covering over two thirds of the large hospitals in Korea which have performed routine renal biopsy examination, we think our data can represent the general trend of glomerular diseases in this country. IgA nephropathy appears to be the most frequent form of primary glomerular disease among Koreans. Considering the difference of its frequency among different countries, ethnic or environmental factors should be counted for the pathogenesis of this disease.

When Berger (1969) first reported nephropathy with IgA-AgG deposits in 55 patients, histological diagnosis were mainly of focal glomerulonephritis and unclassified chronic glomerulonephritis. Clinically most of them presented normal renal function, light proteinuria, and persistent microscopic hematuria, along with episodic gross hematuria. Since then some authors regarded IgA nephropathy as pursuing benign long term course (Morel-Maroger et al., 1972; McCoy et al., 1974; Finlayson et al., 1975; Joshua et al., 1977; Alexander et al., 1977). But the lesion does not always have a favorable prognosis, and progression to end-stage renal fail ure has been well known (Berger et al., 1975; Sissons et al., 1975; Nakamoto et al., 1978; Clarkson et al., 1979; Woodroffe et al., 1980;

D'Amico et al., 1981; Hood et al., 1981).

Our data showing nephrotic range proteinuria in 20%, impaired renal function test in 13.8%, hypertension in 18.1%, and abnormal urinary casts in 18.1%, by the time the biopsies confirmed poor clinical outcomes.

And there was no specific morphologic parameter that we could determine for the prediction of rapid decline in renal function.

As to the histologic classification, most studies revealed that focal and diffuse mesangial proliferative glomerulonephritis comprise a large population of IgA nephropathy though cases of membranoproliferative glomerulonephritis (Alexander et al., 1977; Nakamoto et al., 1978) and partial or diffuse membranous glomerulonephritis(Finlayson et al., 1975; Nakamoto et al., 1978) were also reported.

Our cases also revealed variable mesangial involvement in broad sense rangining from minor change to diffuse sclerosing glomerulonephritis in most cases including a case of membranoproliferative glomerulonephritis. And the frequency of segmental and global sclerosis (75.5%) was higher than those of others (McCoy et al., 1974; Sissions et al., 1975; D'Amico et al., Hood et al. 1981; 1981). However, the frequency of showing crescents (23.4%) was not higher than other data (McCoy et al., 1974; D'Amico et al., 1981). It conceivably denotes that some glomeruli with crescents had already undergone sclerosing process.

The reasons why we had considerable cases with advanced clinical and morpholical manifestations at the time of biopsy are presumably two folds; firstly it could have been the long duration of known disease befeor biopsy in some cases due to different biopsy criteria applied in the 11 hospitals. Secondly the disease itself might have pursued rapidly deteriorating courses from the beginning.

It is of interst that children also have variable morphologic features comparable to adults from minimal lesion to diffuse sclerosing glomerulonephritis similar to those of Roy et al.(1973). But this is in contrast to the observations of Berger et al.(1975) that renal tissue often looked normal by light microscopy. There was no evidence that morphologic severity in IgA nephropathy was associated with aging.

French authors(Berger et al., 1969, 1975; Morel-Maroger et al., 1972) reported strict relationship of IgG and C3 with IgA localization, but concomitant mesangial IgM deposits were observed by various other studies. The proportion of accompanying IgG or IgM deposits are varied among the reporters and our study exhibited that IgG deposits (57.4%) were more frequent than IgM deposits (29.8%).

It has been said that mesangial IgA deposition is usually accompanied by C3 without C1q or C4, suggesting alternate pathway activation (McCoy et al., 1974; Morel-Maroger et al., 1972; Berger et al., 1975; Sissons et al., 1975; Sinniah et al., 1976; Alexander et al., 1977). But other sutides showed that C3 deposits were not necessarily present (Nakamoto et al., 1978; Woodroffe te al., 1980; D'Amico et al. 1981) and that early complement deposits were also present in considerable cases (Nakamoto et al., 1978; Woodroffe et al., 1980; D'Amico et al., 1981; Hood et al., 1981). In present study 18 cases (19.1%) showed no complement deposits at all, and concomitant mesangial C3~C4 deposits were present in 2 cases (2.1%) suggesting activation of the classic complement pathway in very small number. Compared to much higher proportion of mesangial fibrinogen deposit in other series (Morel-Maroger et al., 1972; McCoy et al., 1974; Nakamoto et al., 1978; Sinniah et al., 1976) our result exhibited only 17.0% of cases rather comparable to that of Zollinger and Mihatsch (1978).

The significance of predominant mesangial IgA

deposits is still unknown. Predominant mesangial IgA deposits are frequently found in association with other diseases, such as Henoch-Schoenlein purpura, chronic liver disease and systemic lupus erythematosus, which might be the key to a basic common etiology and pathogenesis (Spichten et al., 1980). Rarely were reported cases with predominant mesangial IgA localization combined with psoriasis (Sissons et al., 1975), familial thrombocytopenia (Spichtin et al., 1980), and ankylosing spondylitis (Sissons et al., 1975; Bailey et al., 1980). Of our biopsy population not included in this study predominant mesangial IgA deposits were also seen in one case of ankylosing spondylitis, and in one case localized form Wegener's granulomatosis. Our case associated with ankylosing spondylitis was very similar to that of Bailey et al. (1981). However we could not refer any literature that described combination of predominant mesangial IgA localization with Wegener's granulomatosis. All of the above combinations could be just fortuitous, but warrant further studies to elucidate the pathogenesis of IgA nephropathy. Although some authors stressed high incidence of family history of renal disease, or familial clustering case (Sissons et al., 1975; Sinniah, 1979) our study showed no significant findings in this aspect.

SUMMARY

Through the immunoflurescence study on 368 renal biopsy specimen that were collected between the year of 1979 and 1981, we found 94 cases of IgA nephropathy without evidence of systemic diseases, thus showing 27.8% relative frequency among primary glomerulonephritides among Koreans. The deposits were IgA alone in 27.7%, IgA-IgG combination in 42.5%, IgA-IgM in 14.9% and IgA-IgG-IgM in 14.9%. Early complement C4 deposits were seen in 2 cases.

The glomerular morphological patterns were: minor change with increased mesangial matrix and cells in 51.0%, diffuse mesangial proliferative glomerulonephritis in 19.2%, focal and segmental proliferative glomerulonephritis in 11.7%, and diffuse sclerosing glomerulonephritis in 9.6% of the cases. Areas of sclerosis were seen in 75.5% and crescents in 23.4%. Morphologic severity was not related to age. Electron dense deposits were seen in mesangium in all cases, with less amounts of subendothelial deposits in 36%, and subepithelial humps in 6%.

Clinically the common presentations were proteinuria, microscopic and gross hematuria. Nephrotic range proteinuria was seen in 20.2%, impaired renal function test in 13.8%, hypertension in 18.1%, and abnormal urinary casts in 18.1% of cases.

It is assumed that IgA nephropathy is fairly common disease among the Korean population, and might be the commonest primary glomerular disease in this country, and it might represent significant proportion of advanced clinical and morphological manifestations.

=國文抄錄=

Ig A 신사구체염

—94례의 병리학적 및 임상상 분석—

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1979년부터 1981년까지 서울대학교 의과대학 병리학교실에 450례의 신장 생검 조직 검사가 의뢰된 바 충분한 면역형광현미경적 검색이 이뤄졌던 368예 중 IgA 신사 구체염은 94례에서 발견되었다. 이상의 368예 중 건신성 질환 30예를 제외한 338예에서 IgA 신사구체염의 빈도는 27.8%이었다.

이들의 면역형광현미경적 검색결과는 IgA 단독침착이 27.7%, IgA-IgG 복합이 42.5%, IgA-IgM 복합이

14.9%, IgA-IgG-IgM 복합침착은 14.9%에서 발견되었다.

신사구체의 형태학적 양상은 미소 증식성 변화 51%, 미만성 세포증식성 신사구체염 19.2%, 국소분엽증식성 신사구체염이 11.7%, 미만성 경화성 신사구체염 9.6%등이었다.

전자현미경적으로 mesanguim 침착물은 전 예에서 관찰된 바 소량의 침착물이 subendothelium (36%)과 subepithelium (6%)에서도 관찰되었다.

임상적으로는 단백뇨, 현미경적 및 육안적 혈뇨가 주증상을 이룬 바 신증후군 범위의 단백뇨는 20.2%에서, 신부전증은 13.8%에서, 고혈압은 18.1%에서, 원주뇨는 18.1%에서발견되었다.

한국인에서 IgA 신사구체염은 매우 흔한 질환이고 만성신부전증을 가져오는 가장 중요한 신사구체염의 하나라고 생각되었다.

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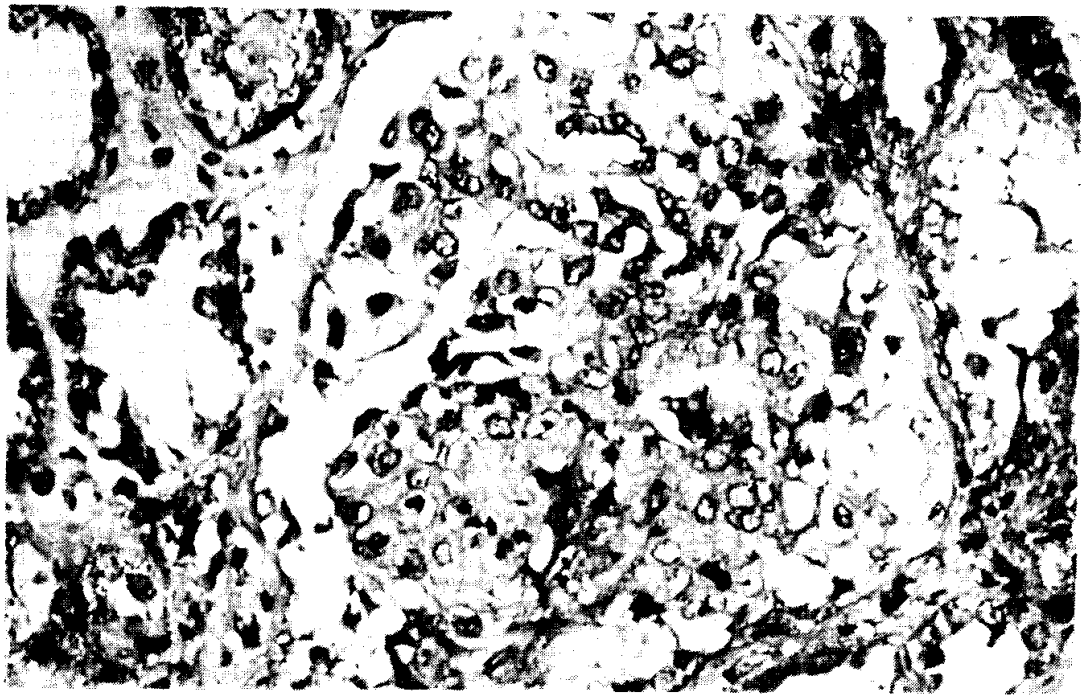

Fig. 1. Focal and segmental proliferative GN with segmental loop necrosis, fibrin exudation and sclerosing process. (PAS×400)

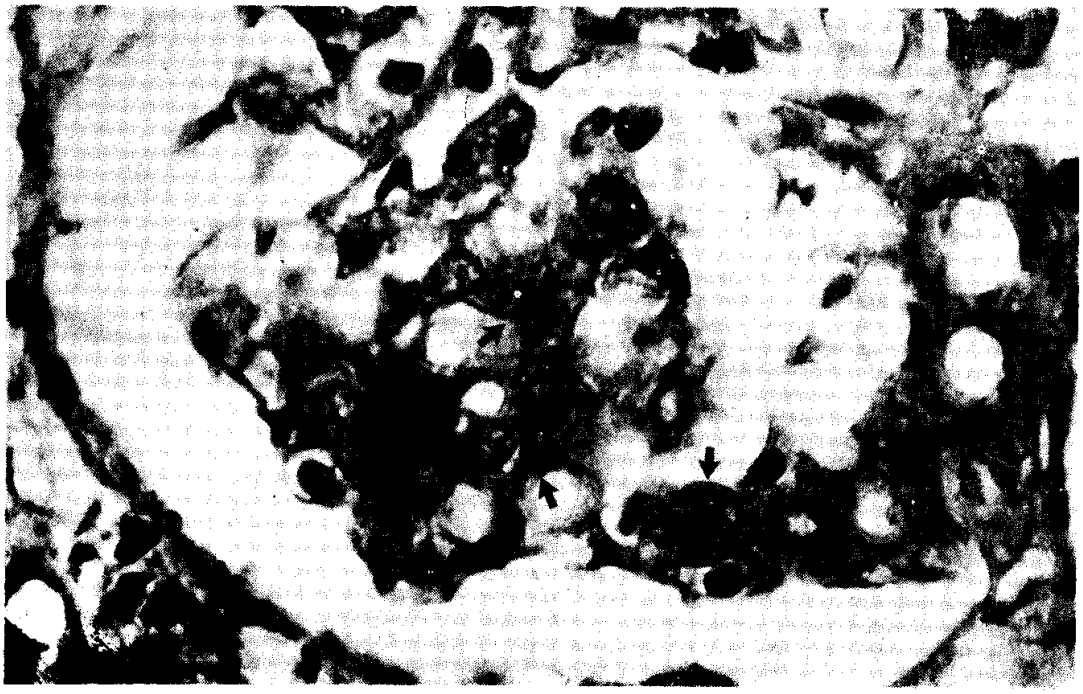

Fig. 2. Mesangial globular deposits (arrows). (Trichrome×1,200)

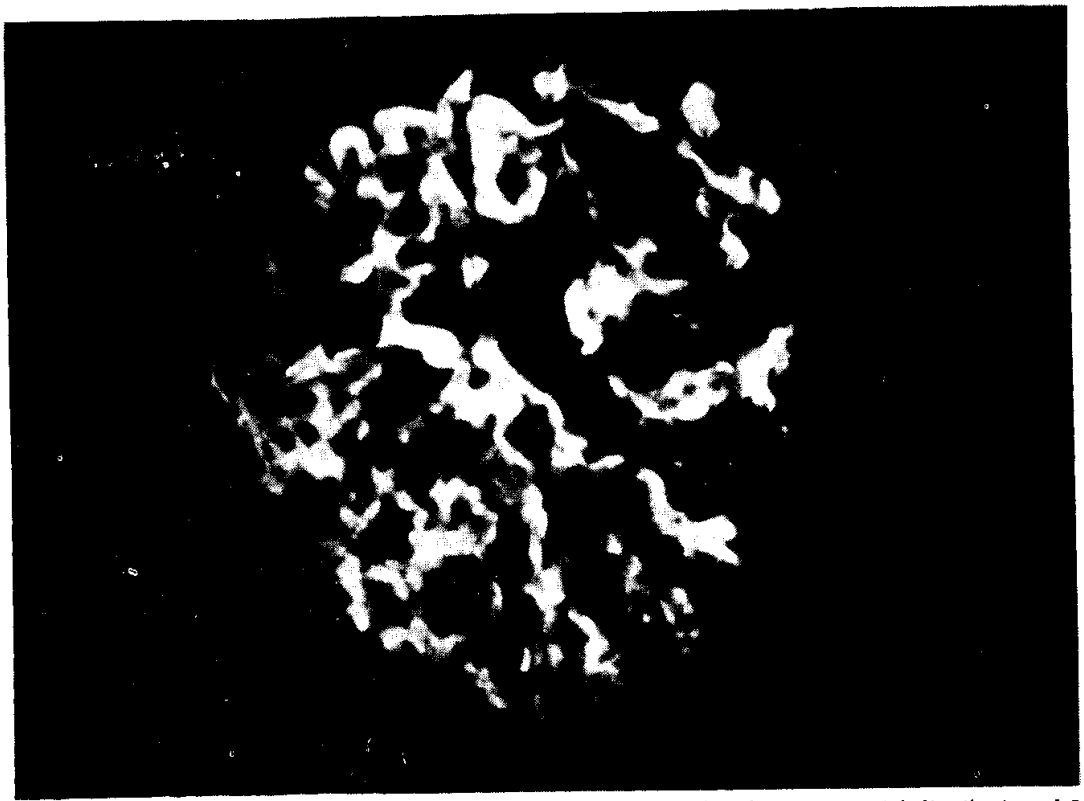

Fig. 3. Immunofluorescence of glomerulus with widespread mesangial and paramesangial distribution of IgA. (400 ASA 25 secs ×400)

Fig. 4. Electron microscopy showing heavy deposition of electron dense material in the mesangium principally along the paramesangial reflection of basement membrane. (×12,000)

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