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Low incidence of IgA nephropathy in Blacks

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Low incidence of IgA nephropathy in blacks. The clinical and pathologic features were evaluated in 106 IgA nephropathy patients identified in 1,753 consecutive patients undergoing renal biopsy in the southeastern United States. Special attention was paid to the proportion of Blacks among the IgA nephropathy patients compared with non-IgA nephropathy patients, and any differences in clinical or pathologic findings between Whites and Blacks with IgA nephropathy. The incidence of IgA nephropathy was approximately six times lower in Blacks (6 out of 461) than in Whites (100 out of 1,292) undergoing renal biopsy ($P < 0.001$). There were no morphologic or immunohistopathologic differences between Whites and Blacks with IgA nephropathy. The only clinical difference was a marked shift in the male to female sex ratio from 3.5:1 in Whites to 1:5 in Blacks ($P < 0.004$). The low incidence and reversed sex ratio of IgA nephropathy in Blacks were also supported by a review of previously published data.

Faible incidence de la néphropathie à dépôts d'IgA chez les noirs. Nous avons évalué les caractéristiques cliniques et pathologiques de 106 patients atteints de néphropathie à dépôts d'IgA identifiés parmi 1753 patients consécutifs ayant eu une biopsie rénale dans la région sud-est des Etats Unis. Une attention particulière a été accordée à la proportion de sujets noirs parmi les patients atteints de néphropathie à dépôts d'IgA, comparés avec des patients atteints d'autres néphropathies, ainsi qu'à toute différence dans les caractères cliniques ou pathologiques entre les sujets noirs ou blancs atteints de néphropathie à dépôts d'IgA. L'incidence de la néphropathie à dépôts d'IgA a été approximativement 6 fois moindre chez les noirs (6 patients sur 461) que chez les blancs (100 patients sur 1292) parmi les sujets ayant une biopsie rénale ($p < 0.001$). Aucune différence morphologique ou immunohistopathologique n'a été trouvé entre les noirs et les blancs atteints de néphropathie à dépôts d'IgA. La seule différence a été une modification nette du sex-ratio (H:F), de 3,5:1 chez les blancs à 1:5 chez les noirs ($p < 0.004$). La faible incidence de la néphropathie à dépôts d'IgA et l'inversion du sex-ratio chez les noirs atteints de la maladie a été confirmée par une revue des données précédemment publiées.

Since its description in 1968 by Berger and Hinglais [1], the clinical and pathologic features of IgA nephropathy have been studied extensively. Virtually every clinicopathologic discussion of IgA nephropathy notes the predilection of this disease for males, the frequency of hematuria in affected individuals, and the characteristic mesangial hypercellularity seen by light microscopy.

However, most studies have also demonstrated substantial clinical and pathologic heterogeneity. The initial clinical manifestations of IgA nephropathy may be asymptomatic gross or microscopic hematuria, acute nephritis, rapidly progressive nephritis, acute renal failure, or nephrotic syndrome. The underlying glomerular lesions can range from mild mesangial expansion to severe glomerular inflammation, even with extensive crescent formation. The nature and degree of glomerular, tubulointerstitial, and vascular injury correlate with the severity of renal dysfunction at the time of biopsy and with the prognosis.

Only a few reports point out the marked racial predilection of IgA nephropathy for Whites compared with Blacks [2–6]. The data presented here further document this predilection in the United States. The clinical and pathologic data are collected from the largest group of IgA nephropathy patients yet reported in North America. Probably due to the criteria used to select patients for renal biopsy, the overall severity of the clinical and pathologic features of our IgA nephropathy patients is greater than in any previously reported series.

Methods

The clinical and pathologic data from the last 106 consecutive patients whose renal biopsy specimens were diagnostic for IgA nephropathy were compiled. All specimens were evaluated in the Nephropathology Laboratory of the Department of Pathology, the University of North Carolina at Chapel Hill. Over 90% of the renal biopsy specimens evaluated in this laboratory are from patients residing in North Carolina, with these specimens being referred from hospitals throughout the state. The remaining specimens are sent primarily from Virginia, South Carolina, and Georgia. Over the interval when the 106 IgA nephropathy specimens were received, a total of 1,753 renal biopsy specimens were evaluated.

All 106 IgA nephropathy specimens were evaluated by direct immunofluorescence using standard procedures [7], and antibodies specific for IgG, IgM, IgA, C3, C4, C1q, and fibrin/fibrinogen. The composition, intensity (0 to 4+), and location (for example, mesangial or capillary wall) of glomerular immunostaining was determined by one of the authors (JCJ). A diagnosis of IgA nephropathy was made when IgA was found to be the most intensely staining glomerular immunoglobulin (Fig. 1). No IgA nephropathy patients had serologic or clinical data indicative of systemic lupus erythematosus, and none had evidence for liver disease. Six patients had clinical evidence for Henoch-Schönlein purpura.

Transmission electron microscopy was carried out on 102 of the specimens using standard techniques [7] and included an evaluation of the presence and location of glomerular electron dense deposits. Adequate tissue (specifically, cortical tissue with 10 or more glomeruli) was available from 99 specimens for light microscopic categorization of glomerular lesions into: *(1)*

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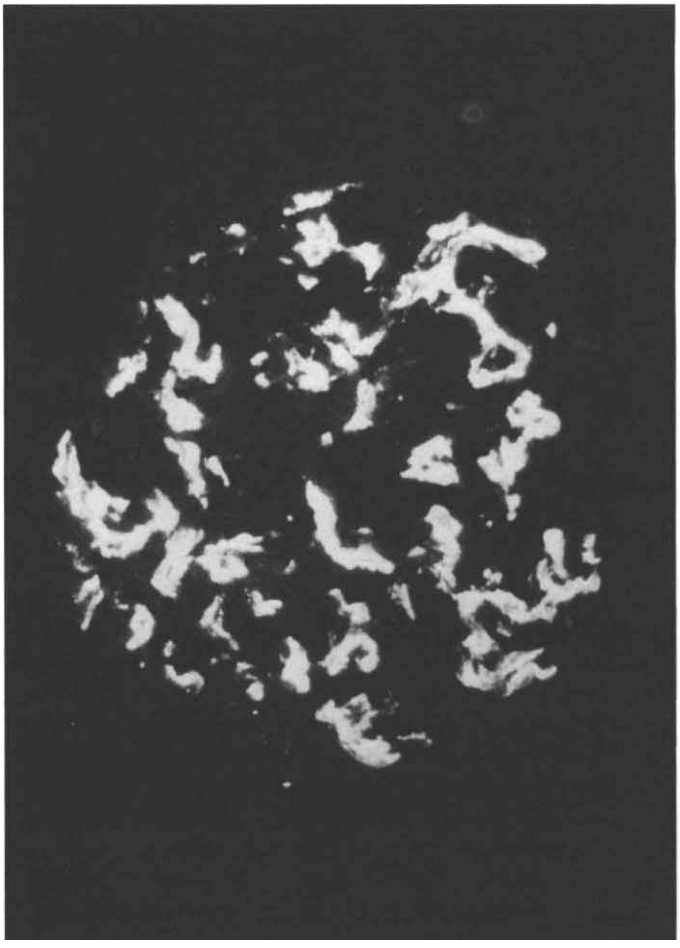

Fig. 1. *Intense IgA immunostaining in the glomerular mesangium of a patient with IgA nephropathy.* (×400)

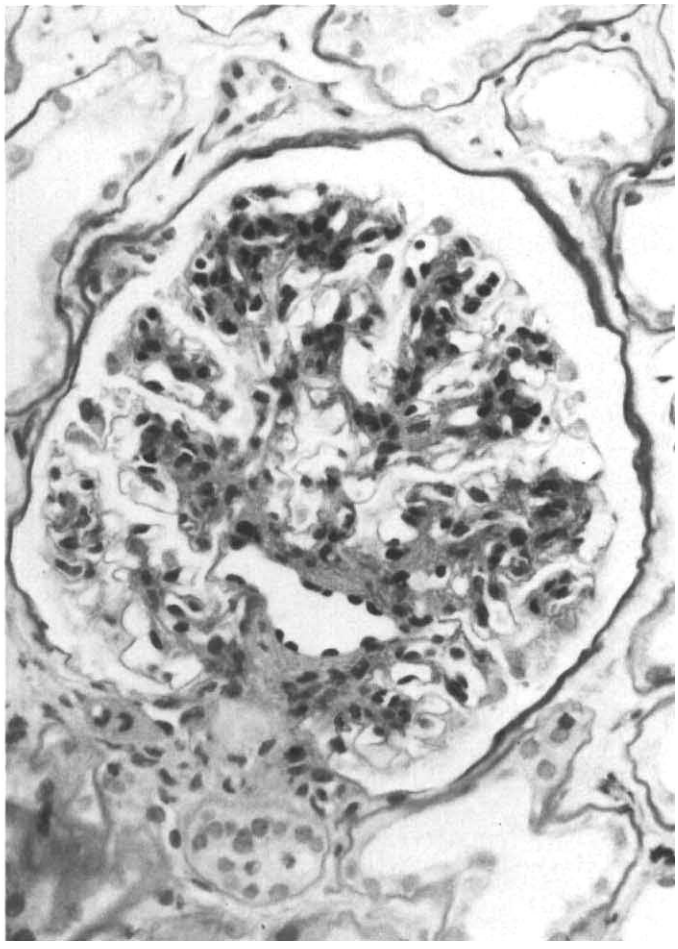

Fig. 2. *Glomerular mesangial hypercellularity and matrix expansion in a patient with IgA nephropathy.* (periodic acid Schiff stain, ×370)

no light microscopic abnormality; *(2)* focal mesangioproliferative glomerulopathy characterized by exclusively mesangial hypercellularity (Fig. 2) in less than 75% of glomeruli; *(3)* diffuse mesangioproliferative glomerulopathy characterized by exclusively mesangial hypercellularity in greater than 75% of glomeruli; *(4)* focal proliferative glomerulonephritis characterized by less than 75% of glomeruli having, in addition to mesangial hypercellularity, any more destructive lesions, such as necrosis, sclerosis, obliteration of capillary lumens, adhesions to Bowman's capsule, or crescent formation (Fig. 3); *(5)* diffuse proliferative glomerulonephritis characterized by greater than 75% of glomeruli having the lesions described for the previous category; or *(6)* crescentic glomerulonephritis characterized by 80% or more of glomeruli having crescent formation. The degree of tubulointerstitial injury was subjectively quantified from 0 to 4+; with 1+ indicating injury to less than 25% of the tubulointerstitial compartment, 2+ indicating injury to 25 to 50%, 3+ indicating 51 to 75%, and 4+ indicating greater than 75%.

For the purposes of this study, compilation of clinical data was confined to the time of biopsy. Age, sex, race, and the presence or absence of hematuria (greater than 5 red blood cell per high power field), proteinuria, and renal insufficiency (serum creatinine greater than 1.5 mg/dl) were determined for all 106 patients. The following data were available for a portion of the patients: blood pressure (79 patients), 24-hr urine protein excretion (76 patients), creatinine clearance (51 patients), serum C3 level (61 patients), serum antinuclear antibody level (63 patients), and serum ASO titer (34 patients).

For statistical analysis of the difference in incidence of IgA nephropathy between Whites and Blacks, a large-sample test procedure for the null hypothesis (proportion 1 = proportion 2) was performed. The significance of the different sex ratios between Whites and Blacks was analyzed by a Fisher's exact test. These analyses were carried out by the Department of Biostatistics, University of North Carolina School of Public Health.

Results

Pathologic data

As shown in Table 1, and as would be dictated by definition, IgA was present in 100% of IgA nephropathy specimens, and had a mean intensity of 3.3+. IgA immunostaining was always either exclusively or predominantly within the mesangium, and, when present in capillaries, was always focal and segmental. C3 immunostaining was seen in all but one specimen, and had a

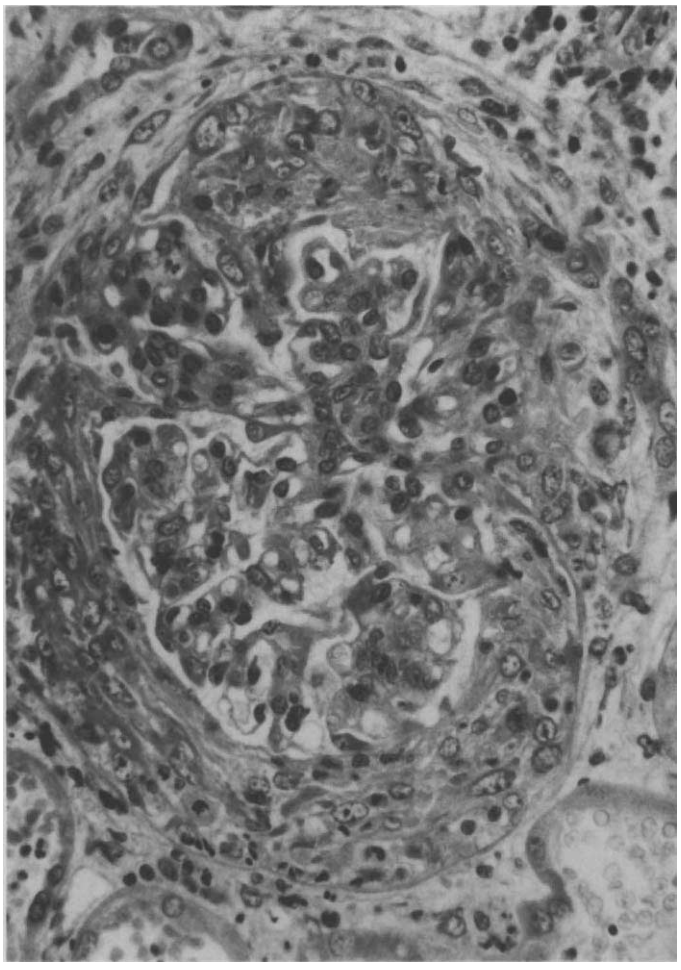

Fig. 3. *Severely injured glomerulus with hypercellularity and crescent formation in a patient with IgA nephropathy.* (H & E stain, ×380)

mean intensity of 2.6+. In spite of this relatively conspicuous C3, there was only low-frequency, low-intensity immunostaining for C4 and C1q. In most, but not all, specimens there was low-intensity IgM and IgG. In two cases, the IgG showed more extensive capillary wall localization, in a subepithelial distribution, than did the IgA; but the mesangial IgA was more intense overall in the glomeruli.

When the IgA nephropathy specimens were categorized as to light microscopic glomerular lesions, there was no difference among the categories with respect to immunoglobulin or complement composition or intensity. Specifically, there was no greater degree of IgG, IgM, or C3 immunostaining in the more destructive categories of injury. Approximately 20% of specimens had well-defined mesangial immunostaining for fibrin/fibrinogen, including seven specimens with 2+ to 3+ staining.

By light microscopy, of the 99 specimens that could be categorized based on glomerular lesions, one had no discernible lesions, ten focal mesangioproliferative glomerulopathy (FMG), ten diffuse mesangioproliferative glomerulopathy (DMG), 20 focal proliferative glomerulonephritis (FPG), 48 diffuse proliferative glomerulonephritis (DPG), and ten crescentic glomerulonephritis (CG) (that is, with 80% or more of glomeruli having crescents). Three of 20 FPG specimens had crescents, involving an average of 7.7% of glomeruli; while ten of 48 DPG specimens had crescents, involving an average of 31.0% of glomeruli.

In specimens categorized as DPG, the more destructive lesions were usually focal, and superimposed on diffuse mesangial hypercellularity and matrix expansion. The severity of tubulointerstitial injury corresponded to the severity of glomerular injury (Fig. 4), with FMG and DMG having 0.5+ mean tubulointerstitial injury, FPG 1.3+, DPG 2.1+, and CG 3.3+. Hyperplasia of mural smooth muscle and sclerotic arterial and arteriolar lesions showed a similar correlation with the degree of glomerular injury.

By electron microscopy, mesangial electron dense deposits were found in 97% of specimens, subendothelial dense deposits in 24%, and subepithelial dense deposits in 20%. Subendothelial and subepithelial dense deposits were observed most frequently in IgA nephropathy specimens having crescentic glomerulonephritis.

Clinical data

Clinical data are detailed in Table 2. The IgA nephropathy patients had a mean age of 32 years. There was a predilection for males versus females (2.9:1) and for Whites versus Blacks (16.7:1). These predilections remained true for all the categories of glomerular injury.

Except for a minority of the patients with FMG, all patients had hematuria. As shown in Table 2 and Figure 4, the degree of proteinuria, renal insufficiency, and hypertension correlated with the severity of glomerular injury, with progressively greater dysfunction in the following order: FMG, DMG, FPG, DPG, CG.

The serum C3 was low in only one of 61 patients. One of 63 patients had a positive antinuclear antibody determination. Four of 34 patients had an ASO titer of greater than 200 Todd units.

Of the 106 IgA nephropathy patients, 100 were White and only six Black, for a ratio of 16.7:1. According to the 1980 United States Census, the ratio of Whites to Blacks in North Carolina is 3.5:1. The overall ratio of Whites to Blacks in patients whose renal biopsy specimens were evaluated in our laboratory during the study was 2.8:1. Of 1,292 Whites, 100 had IgA nephropathy; while only six of 461 Blacks had IgA nephropathy. This was a statistically significant difference ($P < 0.001$). Therefore, there was an approximately sixfold decrease in the proportion of Blacks with IgA nephropathy compared with other renal diseases leading to renal biopsy. Evaluation of 100 of the idiopathic (nonlupus) membranous glomerulopathy patients diagnosed over the same time interval showed a White to Black ratio of 2.2:1.

Of the six Blacks with IgA nephropathy, two had FMG, three FPG, and one CG. They had the same distribution and composition of immunostaining as Whites. As shown in Table 3, the clinical findings in Blacks were similar to that in Whites, with slightly less severe average dysfunction. The most striking difference was a shift in the male:female ratio from 3.5:1 for Whites to 1:5 for Blacks. Even with the small number of Blacks available for comparison, this difference was statistically significant when analyzed by Fisher's exact test ($P < 0.004$).

Table 3 also details the comparative clinical data between males and females, and between younger (less than 30 years old) and older (30 or more years old) patients with IgA nephrop-

Table 1. Pathologic data for IgA nephropathy specimens

	All patients	Light microscopic lesion				
		FMG	DMG	FPG	DPG	CG
Number of patients	106	10	10	20	48	10
IgA deposits	100/3.3[a]	100/3.1	100/3.3	100/3.5	100/3.4	100/3.3
IgG deposits	65/1.3	40/1.3	70/1.4	85/1.3	69/1.3	40/1.0
IgM deposits	80/1.1	90/1.1	90/1.0	70/1.1	81/1.1	80/1.2
C3 deposits	99/2.6	100/2.4	100/2.7	100/2.6	98/2.6	100/2.9
C4 deposits	22/1.0	20/1.0	10/1.0	15/1.0	28/1.1	22/1.0
C1q deposits	12/1.0	12/1.0	0/-	6/1.0	12/1.0	14/1.0
% with mesangial dense deposits	97	100	100	100	96	100
% with subendothelial dense deposits	24	20	20	10	27	50
% with subepithelial dense deposits	20	0	30	5	22	40
% with crescents	23	0	0	15	21	100
Tubulointerstial injury (1–4+)	1.7	0.5	0.5	1.3	2.1	3.3

Abbreviations are: FMG, focal mesangial glomerulopathy; DMG, diffuse mesangial glomerulopathy; FPG, focal proliferative glomerulonephritis; DPG, diffuse proliferative glomerulonephritis; CG, crescentic glomerulonephritis.

[a] Percentage of specimens with deposits/mean intensity (1–4+) when present.

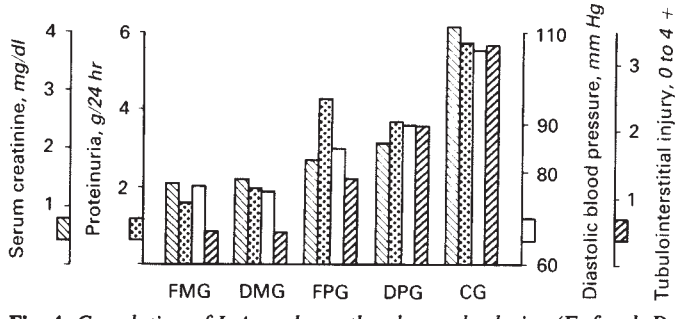

Fig. 4. *Correlation of IgA nephropathy glomerular lesion (F, focal; D, diffuse: MG, mesangioproliferative glomerulopathy; PG, proliferative glomerulonephritis, CG, crescentic glomerulonephritis) with serum creatinine, proteinuria, blood pressure, and degree of tubulointerstitial injury.*

athy. Females had a lower White:Black ratio than males, but age had no effect on the Black:White ratio.

Discussion

IgA nephropathy is defined by its distinctive immunohistopathologic features, and is now known to have a broad spectrum of clinical presentations, related to the severity of the underlying renal parenchymal injury. The ubiquitous finding of hematuria and the male predominance are widely recognized clinical characteristics of IgA nephropathy. Although there has been much discussion about the varying incidence of IgA nephropathy among different populations and the importance of genetics in susceptibility for this disease, there has been little attention paid the markedly lower incidence of this disease in Blacks.

Table 4 is a compilation of published data that detail the clinicopathologic features of IgA nephropathy in the United States. In addition to our data, there are six reports that give the racial distribution of IgA nephropathy [2–6, 15]. Of the 321 patients with IgA nephropathy, 11 were Black. Only 3.4% of IgA nephropathy patients in the United States are Black, while 11.8% of the total population is Black [16]. In our biopsied patients, one out of 13 Whites (100/1,292) had IgA nephropathy, compared to one out of 77 Blacks (6/461) ($P < 0.001$). Thus, the incidence of IgA nephropathy in biopsied patients is approximately six times greater in Whites than Blacks. Evidence that the difference between Whites and Blacks is not due to failure to biopsy Blacks is our observation that the proportion of Blacks to Whites in our renal biopsy population (1:2.8) is actually greater than in the referral population (1:3.5). Also, in another specific glomerular disease category, namely, patients with idiopathic membranous glomerulopathy, the proportion of Blacks (1:2.2) is comparable to that in the total biopsy population. In another clinicopathologic study of patients in our renal biopsy population [17], we observed a Black to White ratio of 1:2.3 in 223 patients with nonlupus proliferative glomerulonephritis, 3.8:1 in 30 proliferative lupus glomerulonephritis patients, and 1.5:1 in a newly described form of glomerulonephritis called C1q nephropathy. The first group shows that the majority of our proliferative glomerulonephritis patients have a racial distribution similar to the total biopsy population, and the latter two groups show that some specific types of proliferative glomerulonephritis have markedly increased proportions of Blacks.

There is variability in the proportion of Blacks in the IgA nephropathy populations reported from different states. This is most likely due to the proportion of Blacks in the population from which the patients came; in other words, IgA nephropathy patients from urban areas or states with high proportions of Blacks would be expected to be more often Black. For instance, there were six Blacks out of 126 IgA nephropathy patients from North Carolina, which has 22.4% Blacks; while there were no Blacks out of 82 patients from Kentucky (7.1% Blacks in the population) or out of 62 patients from New Mexico (1.8% Blacks) [16].

The incidence of IgA nephropathy varies among different populations throughout the world, accounting for less than 10% of all biopsied patients in North America, approximately 20% of all patients with primary glomerulonephritis in Europe and Australia, and more than 30% of primary glomerulonephritis patients in Japan [18]. There is little difference between North America and Europe when the incidence of IgA nephropathy is calculated by a standard approach, for example, based either on the percentage of all biopsied patients, or on the percentage of patients with primary glomerulonephritis. Since the United States has a higher proportion of Blacks, who have a low

Table 2. Clinical data at the time of biopsy in IgA nephropathy patients

	All patients	Light microscopic lesion				
		FMG	DMG	FPG	DPG	CG
Mean age	31.7	28.2	21.5	33.0	34.7	27.0
Sex, *male:female*	79:27	6:4	7:3	12:8	40:8	8:2
Race, *White:Black*	100:6	8:2	10:0	17:3	48:0	9:1
% with hematuria	96	80	100	100	100	100
% with proteinuria	91	50	70	100	100	100
Grams protein/24-hr	3.5	1.6	2.0	4.3	3.7	5.7
% with creatinine > 1.5 mg/dl	49	30	30	37	54	90
Mean creatinine	2.2	1.4	1.5	1.8	2.1	4.1
Mean creatinine clearance	78	90	72	85	83	27
Mean blood pressure (BP)	141/89	129/77	119/76	137/85	141/90	168/106
% with diastolic BP > 90	27	0	0	25	25	57

Abbreviations same as Table 1.

Table 3. Comparison of IgA nephropathy between Whites and Blacks, males and females, and young and old

	White	Black	Male	Female	Younger *<30 years*	Older *≥ 30 years*
Number of patients	100	6	79	27	53	53
Mean age	31.6	32.5	32.6	29.1	18.9	44.5
Sex, *male:female*	78:22	1:5	—	—	29:14	40:13
Race, *White:Black*	—	—	78:1	22:5	50:3	50:3
% with hematuria	97	83	96	96	98	94
% with proteinuria	91	83	95	78	89	92
Grams protein/24-hr	3.5	4.2	3.4	3.9	3.2	3.9
% with creatinine > 1.5 mg/dl	51	17	53	37	40	58
Mean creatinine	2.2	1.3	2.3	1.8	2.2	2.2
Mean creatinine clearance	78	82	78	81	82	74
Mean blood pressure (BP)	141/89	134/86	143/90	135/85	136/88	143/90
% with diastolic BP > 90	27	20	25	30	24	29

incidence of IgA nephropathy, the incidence of IgA nephropathy in the United States might be expected to be slightly lower than that in Europe. In addition to genetic factors, environmental factors and criteria for performing renal biopsies could influence the incidence of IgA nephropathy observed in a given population.

Some racial groups appear to have a higher incidence of IgA nephropathy than do Whites, such as the Japanese [18] and American Indians [15]. The similarly high incidence in these geographically distant populations could be due to the common Mongoloid lineage of both Japanese and American Indians.

Important data supporting the low incidence of IgA nephropathy in Blacks would be renal biopsy findings obtained from a predominantly Black population. Unfortunately, we were unable to locate such data. Two recent reviews of renal disease in Africa note the high frequency of nephritis and nephrotic syndrome, often associated with infectious disease, but do not mention IgA nephropathy [19, 20]. Two cases of IgA nephropathy reported from Johannesburg, South Africa, were both in White patients [21].

IgA nephropathy is morphologically and immunohistopathologically indistinguishable in Whites and Blacks. The only striking clinical difference between Whites and Blacks with IgA nephropathy is a marked shift in the male:female ratio from 3.5:1 in Whites to 1:5 in Blacks ($P < 0.004$) (Table 3). The male predominance in the White pateints is similar to that reported in other studies of IgA nephropathy from North America, Europe, and Asia [18]. In addition to our six patients, we found four other Black IgA nephropathy patients whose sex was reported in the literature [4, 5] and all were females, bringing the total to nine females and one male out of ten Black IgA nephropathy patients. This change, in Blacks, of the sex ratio is also indicated by the lower proportion of Blacks to Whites among males with IgA nephropathy (1:78) compared to females (5:22). Young and old patients had the same White to Black ratio.

The pathologic findings in our 106 IgA nephropathy patients resemble those reported previously [1–15], except for a higher proportion of more severe lesions, including crescentic glomerulonephritis. Similarly, the clinical findings are similar to previously reported data [1–15], but with a greater proportion of patients having renal insufficiency and proteinuria (Table 4). These somewhat more severe clinical and pathologic features in our IgA nephropathy patients are most likely due to the criteria used to select patients for biopsy. Most nephrologists referring specimens to our renal biopsy service do not biopsy patients with hematuria but normal renal function and no or low level proteinuria. Therefore, most of our biopsied IgA nephropathy patients have, in addition to hematuria, substantial proteinuria and/or renal insufficiency.

In conclusion, IgA nephropathy has a much lower incidence in Blacks than Whites. When it occurs in Blacks, females are much more often affected, which is a reversal of the sex ratio in Whites. There is no difference in the pathologic lesions or renal dysfunction induced by IgA nephropathy in Blacks compared with Whites. In both Blacks and Whites, IgA nephropathy has

Table 4. IgA nephropathy in the United States

	N	State	% of all biopsies	Race NB/B	Sex M/F	Mean age	% with Hem	% with Prot	% with Insuff	% with HT
Lowance [8]	15	TX	NA[b]	NA	11/4	27	100	93	13	27
McCoy [2]	20	NC	4.3 (20/470)	20/0	18/2[e]	NA	100	70	NA	10
Finlayson [3]	10	FL	2.2 (10/445)	9/1	NA	24.2	100	90	40	10
Burkholder [9][a]	54	WI	5.1 (54/1050)	NA	43/11	25.5	96	74	56	28
Hood [10]	37	MN	4.6 (37/806)	NA	27/10	25.7	100	86	30	22
SPNSG [11]	62	SW[d]	NA	NA	46/16	PED	100	48	25	6
Lee [4]	20	IL	1.5 (20/1293)	18/2	14/6	26.2	80	75	35	30
Feiner [12]	43	NY	NA	NA	29/14	27.6	88	88	12	28
Croker [13]	81	NC	NA	NA	61/20	27	100	88	32	NA
Kher [5]	21	OH	NA[c]	19/2	15/6	PED	100	69	0	0
Dysart [14]	11	MN	NA	NA	9/2	21.6	100	55	9	27
Wyatt [6]	82	KY	NA	82/0	56/26	NA	NA	88	15	10
Smith [15]	62	NM	9.3 (62/664)	62/0	48/13	31.2	NA	NA	21	32
Jennette	106	NC	6.0 (106/1753)	100/6	79/27	31.7	96	91	49	27
TOTALS	624	USA	4.8 (309/6481)	310/11	427/143	28.3				

Abbreviations are: NB, nonblack; B, black; Hem, hematuria; Prot, proteinuria; Insuff, renal insufficiency; HT, hypertension; SPNSG, Southwest Pediatric Nephrology Study Group; PED, pediatric patients only.

[a] A portion of these data were published earlier by Zimmerman and Burkholder (1975).

[b] 16% of all glomerulonephritis specimens.

[c] 9.5% of all chronic glomerulonephritis specimens.

[d] A multicenter study from the southwestern USA.

[e] The patient population studied had a high proportion of males.

a broad spectrum of clinical presentations that correlate with the severity of the underlying renal parenchymal injury.

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