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Incidence, demographic, biochemical, and clinicopathological profile of primary IgAN in a tertiary care center from Northern India

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Abstract

Background: Primary IgA nephropathy (IgAN) has variable distribution and clinicopathological spectrum throughout the world. We report the incidence, demographic, and clinicopathological profile of primary IgAN from a tertiary care center in Northern India.

Methods: This is a single-center, prospective, observational study conducted at Sheri- Kashmir Institute of Medical Sciences, J&K, India, from January 2015 to December 2018. The study was approved by the hospital ethical committee.

Results: A total of 106 patients were included in this study, accounting for 19% (106/558) of all native kidney biopsies done during the period from January 2015 till December 2018. Males and females accounted for 60.4% (64/106) and 39.6% (42/106), respectively, with a ratio of 1.5:1. The mean age was 31.37±11.60 years. Edema and hypertension were the most common presenting symptoms and signs, seen in 69 (65.1%) and 72 (67.9%) patients, respectively. The baseline 24-h urine protein excretion was 2.32 ±1.34 g, Nephrotic range proteinuria (≥3.5g/day) was seen in 23/106 (21.7%). Average serum creatinine was 1.6±0.80 mg/dl and estimated glomerular filtration rate using CKD-EPI was <60 ml/min/1.73 m^2 in 48.1% of patients (51/106). In patients with < 1 g proteinuria, 36.8% had E1, 78.9% had S1, 36.8% had T1, and 42.1% had T2 lesions.

Conclusions: IgAN is common in North India and has a more severe histopathological presentation, characterized by extensive sclerosis and tubulointerstitial fibrosis. Renal dysfunction and nephrotic range proteinuria are common. Hypertension, low eGFR, and proteinuria correlate with the presence of segmental scarring, endocapillary hypercellularity, and IFTA. Screening of asymptomatic individuals might help in early diagnosis and long-term preservation of renal function.

Keywords: IgA nephropathy, Hypertension, Gross hematuria, Oxford scoring, Renal dysfunction

Introduction

First described by Berger and Hinglais in 1968, IgA nephropathy is the most prevalent pattern of glomerular diseases in most countries where renal biopsy is widely used as an investigative tool. IgAN is an immune-complex mediated disease defined by the presence of either dominant or co-dominant deposits of IgA, predominantly in the glomerular mesangium. The distribution and clinicopathological spectrum of IgA nephropathy vary in different regions throughout the world [1]. The bulk of the disease burden is borne by Asians and Caucasians [2, 3]. IgAN is reported in 10 to 15% of all kidney biopsies in India with a high proportion of nephrotic syndrome and renal dysfunction at presentation [4–9]. Indian patients seem to manifest the disease a decade earlier than Caucasian and East Asian patients [10, 11]. It is unclear whether

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these differences in epidemiology reflect differences in health care provision or are secondary to fundamental differences in the pathogenesis of IgAN. Here we report the incidence, epidemiological, clinical, biochemical, and histopathological characteristics of our IgA cohort from a tertiary care center in Northern India.

Material and methods

This is a single-center, prospective, observational study conducted by the Department of Nephrology, Sheri-Kashmir Institute of Medical Sciences, Srinagar, Jammu, and Kashmir, India, from January 2015 to December 2018. All patients with unexplained renal failure, microscopic hematuria, and any degree of proteinuria were biopsied, unless contraindicated. The renal tissue was evaluated for light microscopy and immunofluorescence. Electron microscopy was not done for financial constraints. The biopsy specimen with a diagnosis of primary IgAN was reported as per the histopathological pattern of injury and revised Oxford MEST-C scoring. All biopsy-proven primary IgAN with >8 glomeruli per biopsy were included in the study.

Statistics

Continuous variables are presented as mean ± SD. Variables with large standard deviations are expressed as a median. The chi-square test is used to compare categorical variables. Mann-Whitney *U* test and Kruskal-Wallis test are used to compare non -parametric continuous variables for 2 and more than 2 groups, respectively. Kruskal-Wallis 1-way ANOVA is used for non-parametric multiple pairwise comparisons of independent samples. Statistics is done using IBM SPSS statistics for windows version 25.

Results

A total of 106 patients were included, accounting for 19% (106/558) of all native kidney biopsies done during the study period. The male-to-female ratio was 1.5:1 (64/42). The mean age at presentation was 31.4±11.6 (range 15–64) years. Ninety-one (86%) patients were in the age group between 15 and 45 years. Sixty patients (56.6%) were in the age group of 15-30 years and 31 (29.2%) patients in the age group of 30–45 years. Edema was the most common presenting complaint, seen in 69 (65.1%) patients. Twelve patients (11.3%) presented with gross hematuria. Hypertension was present in 72 (67.9%) patients. Stage 2 hypertension was present in 65 (61.3%) patients. The mean 24-h urine protein at the time of biopsy was 2.32 ±1.34 (range 0.5–6.5) g. Nephrotic range proteinuria (≥3.5 g/day) was seen in 23 (21.7)% of patients. Sixty-four (60.4%) patients had proteinuria in the range of 1.0 to 3.49 g per day. Mean creatinine at the time of biopsy was 1.6±0.8 (range 0.7–4.40) mg/dl. Estimated glomerular filtration rate (CKD-EPI eGFR) less than 60 ml/min/1.73m^2 was seen in 48.1% (51/106). Mesangial hypercellularity score was > 0.5 (M1) in 57 (53.8%), endocapillary hypercellularity (E1) was present in 75 (70.8%), segmental glomerulosclerosis (S1) was present in 87 (82.1%) patients, and interstitial fibrosis with tubular atrophy (IFTA) involving 26–50% (T1), and > 50% (T2) of cortical area was seen in 46 (43.4%) and 34 (32.1%), respectively. C3 was co-deposited in 83 (78.3%) cases. Co-deposition of IgM and IgG was seen in 18 (16.9%) and 9 (8.45%) respectively. Out of 72 patients with hypertension, C3 was deposited in 59, p value 0.19. Twenty-four-hour urine protein and serum creatinine were significantly higher in patients with mean arterial pressure (MAP) ≥ 104 mmHg, p value <0.05. Patients with eGFR <30ml/min/1.73m^2 were significantly older than patients with eGFR > 60ml/min/1.73m^2, p value 0.01. There was an increased incidence of systolic, diastolic, and MAP with decreased glomerular filtration rate, p value <0.05. Mean arterial pressure in patients with 24-h urine protein >3.0 g per day was significantly higher as compared to patients with 24-h urine protein of <1.0 g per day, p value <0.05. MAP, and mean proteinuria were significantly higher in patients with E1 compared to E0, p value <0.05. MAP and mean proteinuria were higher and mean eGFR was lower in patients with S1 compared to S0, p value <0.001. There is a significant decrease in eGFR across T0 to T2, p value <0.05. The results are summarized in Tables 1, 2, 3, and 4.

Discussion

IgAN has a wide geographical variation around the globe with incidence varying from 2 to 52%. Our study incidence of 19% is like the 16.2% incidence reported by Vanikar et al. from Western India [12]. The striking geographic variation across the world might be due to the presence of particular gene alleles that alter the susceptibility for the development of IgAN or may be related to environmental factors, or the different threshold for kidney biopsy in different centers. Most of our patients were men similar to what was reported by Chacko et al. [13] and Chandrika et al. [14]. Few studies using east Asian cohorts report either equal incidence in men and women or even an increased frequency in females [15–18]. One reason for male predominance in our study can be ascribed to the fact that more males consent for kidney biopsy than females. However, the role of hormonal factors cannot be ruled out with certainty. Mean age of onset, mean serum creatinine, mean systolic, mean diastolic, and MAP were lesser in females compared to men, but they did not reach statistical significance. Similar findings were reported from China [16]. The most common age of presentation

Table 1 Baseline characteristics

Parameter	Value
Mean age in years (SD)	31.37 (11.60)
Age distribution (years)	No. of patients (%)
15–30 (%)	60 (56.6)
30–45 (%)	31 (29.2)
45–60 (%)	13 (12.3)
>60 (%)	02 (1.9)
Male:female (%)	64:42 (60.4/39.6).
No. of patients with hypertension (%)	72 (67.9)
Mean systolic blood pressure in mmHg (SD)	137.60 (21.70)
Mean diastolic blood pressure in mmHg (SD)	85.22 (11.56)
Mean arterial pressure in mmHg (SD)	102.7 (14.53)
No of patients with edema (%)	69 (65.1)
No of patients with asymptomatic urine abnormality (%)	37 (34.9%)
No of patients with gross hematuria (%)	12 (11.3)
No of patients with active urinary sediment (%)	92 (86.8)
Mean proteinuria at time of biopsy in g/dl (SD)	2.32 (1.34)
Proteinuria distribution	No. of patients (%)
< 1 g daily	19 (17.9)
1.0–3.49 g daily	64 (60.4)
≥3.5 g daily	23 (21.7)
Mean serum creatinine in mg/dl (SD)	1.67 (0.84)
Mean CKD-EPI eGFR in ml/min/1.73m^2 (SD)	63.4 (31.73)
Distribution with eGFR group.	No. of patients (%)
<30 ml/min/1.73 m^2	22 (20.8)
30-60 ml/min/1.73 m^2	29 (27.4)
>60 ml/min/1.73 m^2	55 (51.9)

SD standard deviation, *CKD-EPI* chronic kidney disease epidemiology collaboration formula

in our cohort is the 2nd to 3rd decade of life which correlates with other studies [5, 9, 19]. The most common presenting symptom in our study is bilateral lower extremity edema, present in 65.1% of our cohort. An incidence of 46.5% is reported in a study from South India [19]. There was an increase in the incidence of edema with increasing proteinuria; however, it did not reach statistical significance. Hypertension is the most common sign present in 67.9% of patients. In another study from North India, hypertension was found in 78.8% of patients. This highlightens the increased prevalence of hypertension in IgAN patients [8]. At baseline, higher MAP (≥ 104mmHg) was associated with significantly higher proteinuria and lower eGFR. Similar findings were seen in the GRACE-IgANI cohort. Gross hematuria was presenting manifestation in 12% of our patients. A similar incidence (10%) was present in the GRACE-IgANI cohort from South India [19]. Clinicopathological correlations were analyzed at the time of biopsy with clinical variables like the amount of proteinuria, blood pressure, and eGFR. Endocapillary hypercellularity was found to correlate with the degree of hypertension and proteinuria. This is consistent with studies from Europe and China [20, 21]. Presence of sclerosis was found to associate with high MAP, proteinuria, and low eGFR. IFTA score of T1 and T2 was found to be associated with low eGFR. The presence or absence of mesangial hypercellularity was not found to correlate with MAP, eGFR, or proteinuria at the time of renal biopsy. This is consistent with studies from Germany [21], France [22], and North America [23]. None of the biopsies in our cohort showed a crescent or any evidence of acute /active or chronic thrombotic microangiopathy. Significant proteinuria of > 1 g per day was present in 82% of cases. Proteinuria of <1 g per day was seen in 19 (17.9%) patients. In this group, despite having low-grade proteinuria, there were already signs of significant kidney disease with 36.8% having E1, and 78.9% having S1. T1 and T2 lesions were seen in 36.8 and 42.1% of patients respectively. Even in patients with <1 g proteinuria per day and eGFR > 60 ml/min/1.73m^2. E1, S1, T1, and T2 lesions were present in 38 (69.1%), 40 (72.7%), 26 (47.3%), and 5 (9.1%), respectively. So, despite relatively preserved eGFR and low-grade proteinuria, there were signs of significant chronicity in kidney biopsy suggestive of delay in diagnosis. Nephrotic range proteinuria is uncommon in Caucasians [24, 25] but was seen in 23 (21.7%) patients in our cohort. Higher incidence (34%) was found in the GRACE-IgANI cohort likely because of the lower threshold used for the definition of nephrotic range proteinuria (3 g/day). As expected, 23 (100%) patients had E1 and S1 lesions. T1 and T2 lesions were seen respectively in 12 (52.2%) and 5 (21.7%) patients. Even in patients with nephrotic range proteinuria and eGFR > 60 ml/min/1.73 m^2 E1, and S1 lesions were seen in 13 (100%) patients. T1 and T2 lesions were present in 7 (53.8%) and 0%, respectively. It has been postulated that there may be a podocytopathic variant of IgAN marked by predominant podocyte injury [26–29]. Podocyte hypertrophy and tip variant have been described in IgAN and are associated with greater baseline proteinuria. The lack of electron microscopy in our cohort does limit podocyte evaluation. There is a disproportionate over-representation of segmental sclerosing lesions and

Table 2 Histopathological parameters at baseline. Oxford MEST-C score (*n*=106)

M1/M0 (M1%)	57/49 (53.8)
E1/E0 (E1%)	75/31 (70.8)
S1/S0 (S1%)	87/19 (82.1)
T2/T1/T0 (T2%/T1%)	34/46/26 (32.1/43.4)
C2/C1/CO	0
C3 staining (%)	83 (78.3)
IgM and IgG co-staining (%)	18 (16.9) and 09 (8.45)

Table 3 Relationship between MAP, eGFR, and other clinical variables

Characteristic	MAP (mmHg) No. of patients			eGFR (ml/min/1.73m^2) No of patients				Proteinuria in gram/day No of patients			
	<104 39	≥104 67	*p* value	>59.9 55	30–59.9 29	<30 22	*P* value	<1 19	1–2.99 57	>3 30	*p* value
Age mean±SD	27.74±7.86	33.48±12.91	ns	28.82±11.4	32±9.25	36.91±13.25	0.017[b]	31.21±9.85	33.28±11.93	27.83±11.52	0.04[c]
Gender male: female (ratio)	23:16 (1.44:1)	41:26 (1.56:1)	ns	13:9 (1.44:1)	14:15 (0.9:1)	37:18 (2.05:1)	ns	13:6 (2.1:1)	32:25 (1.28:1)	19/11 (1.72:1)	ns
Systolic BP mmHg mean±SD	113.44±9.53	151.67±12.28	<0.001	125.71±18.62	141.59±16.02	162.09±9.98	0.007[a] 0.001[b,c]	129.68±23.44	137.19±21.29	143.40±20.32	ns
Diastolic BP mmHg mean±SD	72.10±7.92	92.85±3.98	<0.001	79.35±12.03	88.38±6.58	95.73±4.33	0.01[a,c] 0.001[b]	77.79±14.23	85.88±10.43	88.67±9.96	0.012[b]
MAP in mmHg mean±SD	85.85±7.84	112.51±5.91	<0.001	94.96±14.02	105.86±9.05	94.96±14.02	0.013[a] 0.001[b,c]	95.11±16.94	103±13.62	106.93±13.09	0.031[b]
Mean (SD) 24-h urine protein	1.95 (1.21)	2.53 (1.36)	0.01	2.47±1.54	2.16±1.06	2.13±1.08	ns	0.79±0.21	1.90±0.48	4.08±0.95	0.001
Serum creatinine mg/dl mean±SD	1.11 (0.31)	2.0 (0.88)	<0.001	1.07±0.24	1.86±0.93	2.92±0.61	0.001[a,b] 0.012[c]	1.86±1.03	1.63±0.79	1.62±0.81	ns
eGFR ml/min/1.73m^2 mean±SD	86.03±24.45	50.27±27.92	<0.001	89±20.43	44.10±10.12	24.45±4.60	0.001[a,b] 0.010[c]	62.16±36.07	61.96±30.22	66.9±33.21	ns

SD standard deviation, *MAP* mean arterial pressure, *BP* blood pressure, *eGFR* estimated glomerular filtration rate (calculated using *CKD-EPI* chronic kidney disease epidemiology collaboration formula), *ns* not significant

[a] Columns 1 and 2

[b] Columns 1 and 3

[c] Columns 2 and 3

Table 4 Relationship between histopathological and clinical variables

Characteristic	MAP (mmHg)	*P* value	eGFR (ml/min/1.73m^2)	*P* value	Proteinuria (g/d)	*P* value
M0	104.08±14.75	ns	61.55±32.37	ns	2.33±1.35	ns
M	101.51±14.19		64.98±31.38		2.30±1.34	
E0	96.97±15.05	0.01	64.32±28.88	ns	1.21±0.39	0.001
E1	105.07±13.72		63.01±33.02		2.77±1.32	
S0	91.16±12.45	0.001	83.74±27	0.001	1.29±0.34	0.001
S1	105.2±13.77		55.95±31.08		2.54±1.37	
T0	94.85±12.53	ns[a]	65.28±23.03	0.002[a]	2.34±1.42	ns
T1	101.41±13.15	0.001[b]	65.28±25.05	0.001[b,c]	2.51±1.49	
T2	110.44±14.29	0.04[c]	63.40±31.73		2.03±0.98	

MAP mean arterial pressure, *eGFR* estimated glomerular filtration rate (calculated using *CKD-EPI* chronic kidney disease epidemiology collaboration formula), *ns* not significant

[a] Row 1 and 2

[b] Row 1 and 3

[c] Row 2 and 3

tubulointerstitial fibrosis at presentation even with relatively well-preserved eGFR and low levels of proteinuria. This contrasts with studies from the east Asian population where predominant features at presentation are active glomerular lesions with little background scarring [20, 30]. It is unclear why our cohort has such advanced fibrotic lesions so early in the clinical course. This might be related to low nephron number consequent to poor maternal health and nutritional status owing to the poor socio-economic status with subsequent hyperfiltration injury resulting in an increased risk of hypertension and CKD. However, this would need confirmation from larger studies and further evaluation. The overall incidence, demographic, biochemical, and clinicopathological profile of primary IgAN from our center in Northern India is very similar to the rest of the country.

The main limitation of the present study is that being a hospital-based single-center study, it does not represent the true burden of the disease, since only the patients who presented to the hospital were biopsied. The study also lacks long-term follow-up data.

Conclusions

IgAN is common in North India and has more severe histopathological presentation, characterized by extensive sclerosis and tubulointerstitial fibrosis at diagnosis. Renal dysfunction and nephrotic range proteinuria are common. The presence of hypertension, low eGFR, and significant proteinuria correlate with the presence of segmental scarring, endocapillary hypercellularity, and IFTA on renal biopsy. Screening of asymptomatic individuals might help in early diagnosis and long-term preservation of renal functions.

Abbreviations

IgAN: IgA nephropathy; eGFR: Estimated glomerular filtration rate; IFTA: Interstitial fibrosis and tubular atrophy; MEST-C: Mesangial hypercellularity, endocapillary hypercellularity, segmental sclerosis, interstitial fibrosis, and tubular atrophy, crescents; MAP: Mean arterial pressure.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s43162-022-00109-9.

Additional file 1.

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Authors' contributions

MA, IS, RS, and IW contributed to the design and development of the study. MA, IS, RS, and IW contributed to data collection. MA, IS, RS, and IW contributed data analysis and interpretation. MA, IS, RS, and IW participated in the writing of the manuscript. MA and IW participated in the critical review. MA, IS, RS, and IW provided approval for the final manuscript.

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Availability of data and materials

Data and material will be available on request.

Declarations

Ethics approval and consent to participate

The study is approved by the institutional ethics committee of Sheri- Kashmir Institute of Medical Sciences under Reference no. SIMS/377/241/2014/332 dated 16th December 2014.

Since this is an observational study, the consent to participate has been waived off by the ethics committee of Sheri- Kashmir Institute of Medical Sciences

Consent for publication

Not applicable.

Competing interests
The authors declare that they have no competing interests.

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References

1. D'Amico G (1987) The commonest glomerulonephritis in the world: IgA nephropathy. Q J Med 64:709–727
2. Seedat YK, Nathoo BC, Parag KB, Naiker IP, Ramsaroop R (1988) IgA nephropathy in blacks and Indians of natal. Nephron 50:137–141
3. Jennette JC, Wall SD, Wilkman AS (1985) Low incidence of IgA nephropathy in blacks. Kidney Int 28:944–950
4. Narasimhan B, Chacko B, John GT et al (2006) Characterization of kidney lesions in Indian adults: towards a renal biopsy registry. J Nephrol 19:205–210
5. Das U, Dakshinamurty KV, Prayaga A (2011) Pattern of biopsy-proven renal disease in a single center of south India: 19 years experience. Indian J Nephrol 21:250–257
6. Bhuyan UN, Dash SC, Srivastava RN et al (1992) IgA-associated glomerulonephritis. J Assoc Physicians India 40:310–313
7. Siddappa S, Kowsalya R, Mythri KM (2011) IgA nephropathy in a tertiary care center from south India. Indian J Nephrol 21:230–234
8. Mittal N, Joshi K, Rane S et al (2012) Primary IgA nephropathy in north India: Is it different? Postgrad Med J 88(1035):15–20. https://doi.org/10.1136/postgradmedj-2011-130077 Epub 2011 Nov 3. PMID: 22052882
9. Muthukumar T, Fernando ME, Jayakumar M (2002) Prognostic factors in immunoglobulin-A nephropathy. J Assoc Physicians India 50:1354–1359
10. Prakash S, Kanjanabuch T, Austin PC et al (2008) Continental variations in IgA nephropathy among Asians. Clin Nephrol 70:377–384
11. Berthoux FC, Mohey H, Afiani A (2008) Natural history of primary IgA nephropathy. Semin Nephrol 28:4–9
12. Vanikar AV, Kanodia KV, Patel RD et al (2005) Primary immunoglobin A nephropathy in Western India. Indian J Nephrol 15:227–231
13. Chacko B, John GT, Neelakantan N et al (2004) Primary IgA nephropathy: a ten-year analysis on renal outcomes and a model for estimating of risk of progression. Indian Journal Nephrol 14:163–171
14. Chandrika BK (2009) IgA nephropathy in Kerala, India: a retrospective study. Indian J Pathol Microbiol 52:14–16
15. Li PKT, Ho KKL, Szeto CC et al (2002) Prognostic indicators of IgA nephropathy in the Chinese—clinical and pathological perspectives. Nephrol Dial Transplant 17:64–69
16. Deng W, Tan X, Zhou Q et al (2018) Gender-related differences in clinicopathological characteristics and renal outcomes of Chinese patients with IgA nephropathy. BMC Nephrol 19:31
17. Moriyama T, Tanaka K, Iwasaki C et al (2014) Prognosis in IgA nephropathy: 30-year analysis of 1,012 patients at a single center in Japan. PLoS One 9:e91756
18. Knoop T, Vikse BE, Svarstad E et al (2013) Mortality in patients with IgA nephropathy. Am J Kidney Dis 62:883–890
19. Alexander S, Varughese S, Franklin R, Roy S, Rebekah G, David VG, Mohapatra A, Valson AT, Jacob S, Koshy PM, Rajan G, Daha MR, Feehally J, Barratt J, John GT (2020) Epidemiology, baseline characteristics, and risk of progression in the first South-Asian prospective longitudinal observational IgA nephropathy cohort. Kidney Int Rep 6(2):414–428. https://doi.org/10.1016/j.ekir.2020.11.026 PMID: 33615067; PMCID: PMC7879115
20. Zeng CH, Le W, Ni Z et al (2012) A multicenter application and evaluation of the oxford classification of IgA nephropathy in adult chinese patients. Am J Kidney Dis Off J Natl Kidney Found 60:812–820
21. Schimpf JI, Klein T, Fitzner C et al (2018) Renal outcomes of STOP IgAN trial patients in relation to baseline histology (MEST-C scores). BMC Nephrol 19:328
22. Alamartine E, Sauron C, Laurent B et al (2011) The use of the OxfordClassification of IgA nephropathy to predict renal survival. Clin J Am Soc Nephrol 6:2384–2388
23. Herzenberg AM, Fogo AB, Reich HN et al (2011) Validation of the Oxford classification of IgA nephropathy. Kidney Int 80:310–317
24. Donadio JV, Grande JP (2002) IgA nephropathy. N Engl J Med 347:738–748
25. Delclaux C, Jacquot C, Callard P et al (1993) Acute reversible renal failure with macroscopic haematuria in IgA nephropathy. Nephrol Dial Transplant Off Publ Eur Dial Transpl Assoc—Eur Ren. Assoc. 8:195–199
26. Herlitz LC, Bomback AS, Stokes MB et al (2014) IgA nephropathy with minimal change disease. Clin J Am Soc Nephrol 9:1033–1039
27. Hill GS, Karoui KE, Karras A et al (2011) Focal segmental glomerulosclerosis plays a major role in the progression of IgA nephropathy.I. Immunohistochemical studies. Kidney Int 79:635–642
28. El Karoui K, Hill GS, Karras A et al (2011) Focal segmental glomerulosclerosis plays a major role in the progression of IgA nephropathy. II. Light microscopic and clinical studies. Kidney Int 79:643–654
29. Cook HT (2011) Focal segmental glomerulosclerosis in IgA nephropathy: a result of primary podocyte injury? Kidney Int 79:581–583
30. Kang SH, Choi SR, Park HS et al (2012) The Oxford classification as a predictor of prognosis in patients with IgA nephropathy. Nephrol Dial Transplant 27:252–258

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