Natural history and prognosis of IgA nephropathy in Chinese

Jicheng Lv 吕继成

Institute of Nephrology, Peking University

Renal division of Peking University First Hospital

Outline

- **The nature history of IgA nephropathy**
- **Can/have we changed it?**
- **Perspective**

Outline

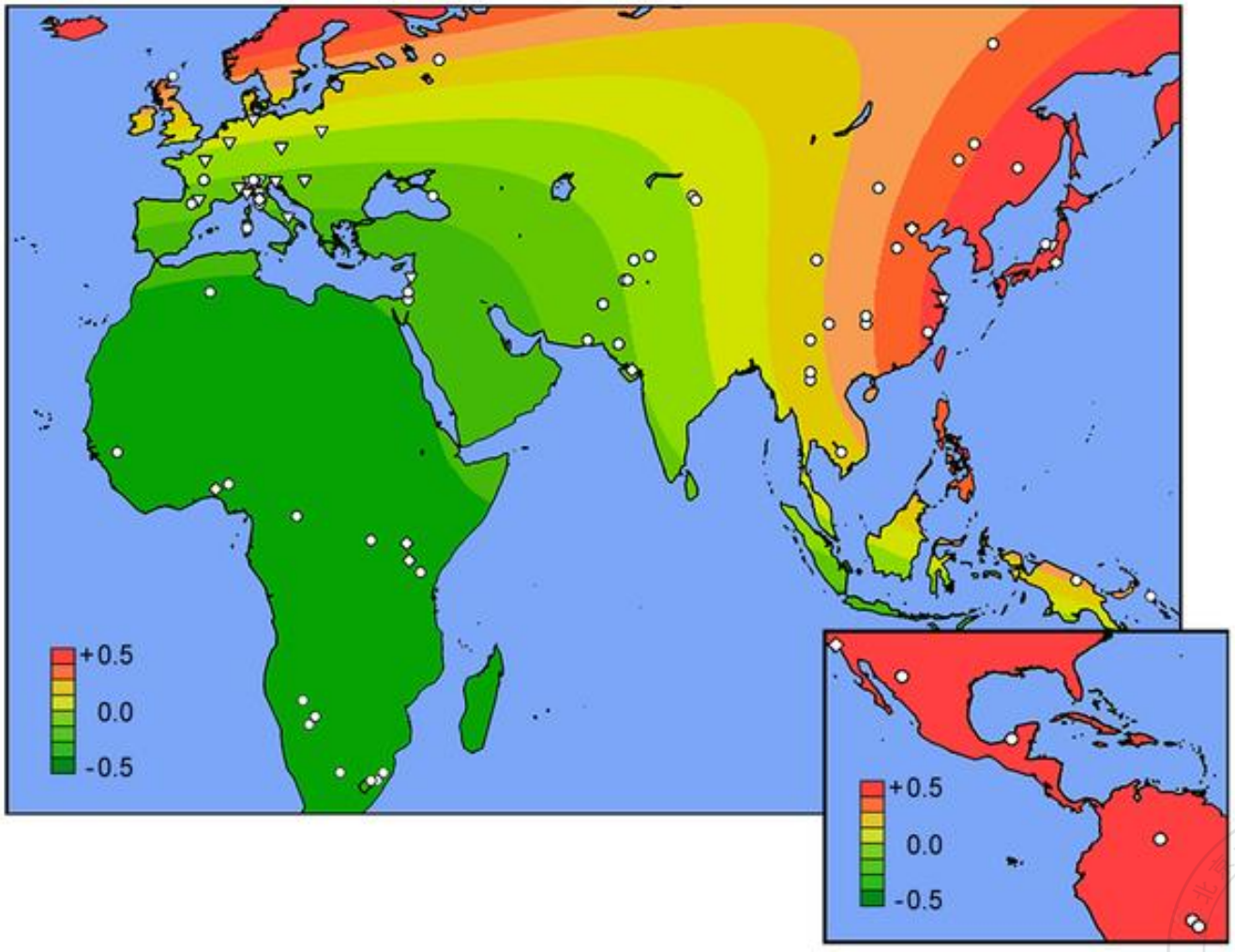
- **The nature history of IgAN**
  - **General IgA nephropathy**
  - **Different phenotype of IgA nephropathy**
- Can/have we changed it?
- Perspective

Epidemiological data regarding the frequency IgA nephropathy

Country	Author(year)	Study population (number of renal biopsy)	Proportion of primary GN (%)	Proportion of all GN (%)	Incidence (per 1 million person-years)
		Asia			
China	Zhou FD (2009) Li LS (2004)	Single Centre-north China (5714) Single Centre-south China (13,519)	58.2 45.6		
Japan	1999	National Survey (1850)	47.3		
Korea	Chang JH(2009)	Single Centre (1818)	28.3		
Singapore	Woo KT (1999)	Review	45		
		Oceania			
Australia	Briganti EM(2001)	Population-based (2030)	48.3	34.1	42.9
		Europe			
CzechRepublic	Rychlík I(2004)	National Registry of Renal Biopsies (4004)	34.5		
Italy	Schena FP (1997)	National Registry of Renal Biopsies (13835)	36.9		
	Stratta P 1996	Population based survey			14.7
Spain	Rivera F (2002)	National Registry of Renal Biopsies (7016)		17	7.9
UK	Hanko JB(2009)	Regional biopsy registry (1844)	38.8		3.4 (1976 to 1985) to 17.9 (1996-2005)
Netherland	Tiebosch AT (1987)	Population based survey			19
France	Simon P (2004)	Population based survey			28
		Americas			
USA	Nair R (2006)	Nephropathology Associates from states (4504)	24	22	
	Wyatt RJ (1998)	Population-based survey			5(1975-1979) to 12 (1990-1994)
Brazil	M. G. Polito (2010)	National biopsy data	20.1		

From TESTING study protocol

Worldwide geospatial risk analysis.



Kiryluk K, Li Y, Sanna-Cherchi S, Rohanizadegan M, Suzuki H, et al. (2012) Geographic Differences in Genetic Susceptibility to IgA Nephropathy: GWAS Replication Study and Geospatial Risk Analysis. PLoS Genet 8(6): e1002765

Nature history and kidney survival in Chinese patients with IgA nephropathy

（n=2230 for outcomes of ESKD and 2349 for eGFR decline）

Author	Sample size	Year	Age	Proteinuria (g/d)	hypertension MAP	hypertension %	Scr umol/L	eGFR ml/min/1.73m²	CKD stage 1	2	3	4	5	Follow	Therapy RASi	steroid	others	TA-pro (g/d)	TA-MAP mmHg	GFR decline	Survival (%) 5	10	15	20
Li et al. Hong Kong	168	1987-1996	33	1.67		28	152.7							7.4							88	82		
Lv et al. Beijing	204	1990-2001	31	2.7		41	132.6	81	38	35	13	10	3	6.1	56	36	22				85	77		
Le et al. Nanjing	1155	1989-2005	34	0.89		31	93.7	89	48	32	18	2	1	5.4	90	10.8	13.6	0.54	94	-1.6		85	76	67
Li et al. Beijing	703	2003-2011	34	2.5	94		101.7	84	46	32	11	6	5	3.8	96	45		1.12	90	-3.12	89			
Zeng et al. Multi-center	1026	2012	34	1.3	98	33		85	41	35	24			4.4	89	31		0.7	94	-1.5				
VALIGA cohort	1147	2014	36	1.3	98	65			60		28	7	2	4.7	86	43	16	0.8	96	-1.8		74*		
North American	187	2011	34	1.7	96			82						4.4	87	37	24	1	92	-2.6				
Toronto cohort	542	2007	38	2.4	103		129.7	77						6.5	53	13		2.2	100	-4.56				
Japan National Register	2283	1995-2005	32						36	32	25	5	2									85		
Japan	1012	1974-2011	33	1.2	90.4		78.7	79						7.9	28.9	13						84		67

Li PK et al. Nephrol Dial Transplant. 2002;17(1):64-9
Lv J et al. Nephrology (Carlton). 2008;13(3):242-6
Li XL et al. Clin J Am Soc Nephrol. 2014;9(3):484-9

Le W et al. Nephrol Dial Transplant. 2012;27(4):1479-85
Zeng C et al. Am J Kidney Dis. 2012 Nov;60(5):812-20.

Risk Factors for IgA nephropathy progression (published before 2000s)

Authors and Country	Clinical Features at Presentation							
	No. of Patients	Mean Age at Presentation (yr)	High Serum Creatinine (%)	High Blood Pressure (%)	Proteinuria >3 g/24 Hours	History of Macroscopic Hematuria (%)	Mean Duration of Follow-up (months)	Actuarial Renal Survival at 10 Years
Europe								
D'Amico et al (1986), Italy[5]	365	29	24	36	7%	55	79	85%*
Beukhof et al (1986), The Netherlands[6]	75	24	—	37	—	46	92	84%*
Droz et al (1984)[7]; Noël et al (1987), France[8]	280	—	—	6	10%	37	>60	85%*
Velo et al (1987), Spain[9]	153	22	—	—	1%	78	>60	81%*
Bogenschutz et al. (1990), Germany[10]	239	—	34	19	—	26	59	81%†
Rekola et al. (1989, 1990), Sweden[11,12]	209	25	16	11	1%	64	76	83%‡
Alamartine et al. (1991), France[13]	282	28	2	9	3%	27	96	94%*
Johnston et al. (1992), UK[14]	220	30	28	26	32%	—	65	83%‡
Payton et al. (1988), UK[15]	67	32	—	40	—	—	—	77%*
Australia								
Nicholls et al. (1984)[16]	244	32	36	43	6%	39	60	87%‡
Ibels et al. (1994)[17]	121	39	36	31	16%	40	107	93%*
Asia								
Woo et al. (1986), Singapore[18]	151	27	6	33	4%	24	65	91%‡
Kusumoto et al. (1987), Japan[19]	87	27	—	31	15%	—	114	80%*
Katafuchi et al. (1994), Japan[20]	225	32	36	22	16%	20	48	74%‡
Yagame et al. (1996), Japan[21]	206	30	—	—	—	—	110	87%‡
Koyama et al. (1997), Japan[22]	448	>10 in 95%	19	29	3%	24	142	85%*
Li et al. (2002), Hong Kong[23]	168	33	—	28	5%	20	88	82%‡
North America								
Wyatt et al. (1984), USA[24]	58	27					>60	78%*
Radford et al. (1997), USA[25]	148	39	59	47	30%	—	45	67%‡
Haas (1997), USA[26]	109	~40	mean = 2.2 ± 1.9 mg/dL	49	33%	35	>18	57%‡
Bartosik et al. (2001), Canada[27]	298	36	—	—	—	—	70	65%*

D'Amico G.Semin Nephrol. 2004;24(3):179-96

Risk Factors for IgA nephropathy in Chinese

Author	Year	Sample size	Follow	baseline Proteinuria		TA-proteinuria		Hypertension		TA-MAP	
				Uni	multi	Uni	multi	Uni	multi	Uni	multi
Le et al.	2012	1155	5.4	Y	Y	Y	Y	Y	Y	Y	Y
Xie et al	2012	619	3.4	Y	N	NA	NA	Y	Y	NA	NA
Lv et al.	2008	204	6.1	Y	N	NA	NA	Y	Y	NA	NA
Li et al.	2014	703	3.8	Y	N	Y	Y	Y	Y	Y	Y
Li et al.	2002	168	7.4	Y	Y	NA	NA	Y	Y	NA	NA

author	Year	GFR or Scr		Marohematuria		Hyperuricemia		Hypoalbuminemia		Anemia	
		Uni	multi	Uni	multi	Uni	multi	Uni	multi	Uni	multi
Le et al.	2012	Y	Y	Y	N	Y	Y	Y	Y	NA	NA
Xie et al	2012	Y	Y	N	N	Y	N	Y	Y	Y	Y
Lv et al.	2008	Y	Y	NA	NA	NA	NA	NA	NA	NA	NA
Li et al.	2014	Y	Y	N	N	Y	NA	NA	NA	NA	NA
Li et al.	2002	Y	Y	NA	NA	NA	NA	NA	NA	NA	NA

Li PK *et al*. Nephrol Dial Transplant. 2002;17(1):64-9
Lv J *et al*. Nephrology (Carlton). 2008;13(3):242-6
Li XL *et al*. Clin J Am Soc Nephrol. 2014;9(3):484-9

Le W *et al*. Nephrol Dial Transplant. 2012;27(4):1479-85
Zeng C *et al*. Am J Kidney Dis. 2012 Nov;60(5):812-20.
Xie J *et al*. PLoS ONE 7(6):e38904.

Outline

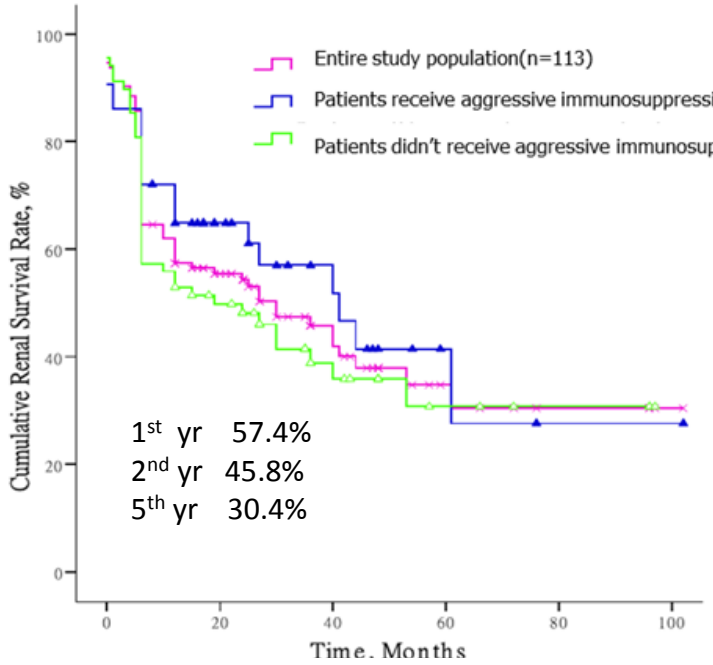
- **The nature history of IgAN**
  - General IgA nephropathy
  - **Different phenotype of IgA nephropathy**
    - **Crescentic IgA nephropathy**
    - **With minimal change like lesions**
    - **Hematuria with or without minimal proteinuria**
- Can/have we changed it?
- Perspective

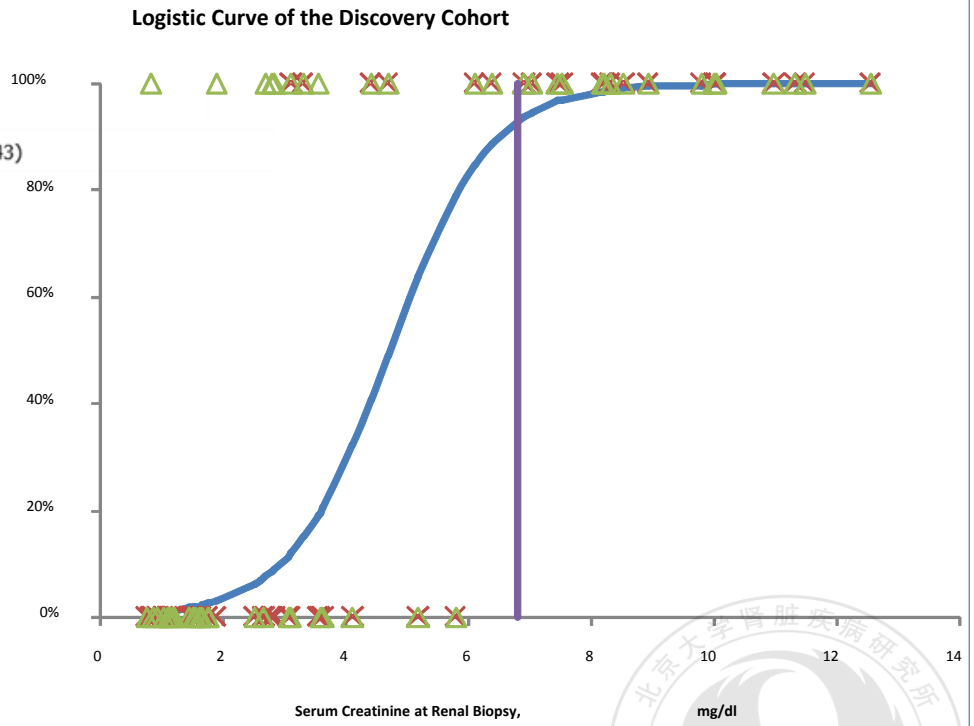
Follow-up data of 15 Chinese patients with crescentic IgAN (n=15)

Case No.	Follow-up time	Upro, g/24 h		Scr, μmol/l	
		first	end	first	end
1	26	3.35	1.59	241	188
2	8	1.48	2.50	856	HD
3	17	4.80	1.39	256	103
4	24	2.97	1.58	667	HD
5	36	3.58	0.50	124	114
6	16	3.97	3.17	577	435
7	25	1.85	1.19	109	98
8	14	3.31	1.60	304	268
9	29	8.50	0.87	325	115
10	92	5.97	0.83	760	176
11	52	2.93	1.82	168	HD
12	9	2.12	0.29	388	HD
13	19	3.20	1.60	168	126
14	42	1.68	0.25	142	250
15	38	2.99	0.58	882	PD
$\overline{X} \pm SD$	29 ± 21	3.51 ± 1.81	1.31 ± 0.87	409 ± 299	187 ± 106

Tang Z et al. Am J Nephrol 2002;2:480

Prediction of Outcomes in Crescentic IgA Nephropathy in a Multicenter Cohort Study





Clinical Nephrology, Vol. 26 No. 4 – 1986 (pp. 174–180)

Corticosteroid therapy in IgA nephropathy with nephrotic syndrome: a long-term controlled trial

K. N. Lai[1], F. M. Lai[1], C. P. Ho[2] and K. W. Chan[2]

[1]*Renal Unit, Department of Medicine, Chinese University of Hong Kong, Prince of Wales Hospital, Shatin, New Territories Hong Kong*
[2]*Renal Unit, Princess Margaret Hospital, Hong Kong*

Abstract. A randomized prospective study of 34 patients with IgA nephropathy and nephrotic syndrome was conducted to determine the therapeutic value of corticosteroid therapy. The patients were divided into two groups: Group A, 17 patients receiving oral prednisolone/prednisone for four months; and Group B, 17 patients receiving no corticosteroid therapy and acting as controls. The groups are comparable in age of presentation, sex ratio, and duration of study. No difference in serum creatinine levels, creatinine clearance, serum IgA levels, severity of renal histopathological changes, incidence of hypertension or incidence of impaired renal function could be demonstrated but the Group A patients had significantly heavier proteinuria. During the mean study period of 38 months (range 12–106), no significant difference in serum creatinine levels and creatinine clearance was demonstrated between the two groups. Forty percent of the Group A patients developed complications related to steroid therapy. Despite the overall lack of therapeutic value in IgA nephropathy with nephrotic syndrome as reflected by change in renal function, corticosteroid treatment resulted in excellent remission of nephrotic syndrome in 80% of patients with mild glomerular histopathological changes. Our findings suggest that corticosteroid therapy is only beneficial to selected groups of patients with IgA nephropathy and nephrotic syndrome but its indiscriminate use should be discouraged.

Key words: IgA nephropathy – nephrotic syndrome – corticosteroid treatment – minor glomerular pathology

Introduction

Although moderate degrees of proteinuria are frequently found in patients with IgA nephropathy, nephrotic syndrome has generally been regarded as uncommon [Clarkson et al. 1977, Hood et al. 1981, Kincaid-Smith and Nicolls 1983]. In the few cases that have been reported, the prognostic significance is unclear [Abreo and Wen 1983, Mustonen et al. 1983, Katz et al. 1983]. Recently, IgA nephropathy presented with nephrotic syndrome has in some instances been reported to respond to corticosteroid therapy [Saint-Andre et al. 1980, Mustonen et al. 1983, Southwest Pediatric Nephrology Study Group 1985]. Our preliminary data suggest that the nephrotic syndrome in IgA nephropathy does not inevitably indicate the presence of poor clinical outcome and treatment with corticosteroid is encouraging in those patients with minor glomerular pathology despite heavy proteinuria [Lai et al. 1985]. In this study, we report our randomized prospective trial of corticosteroid therapy in nephrotic syndrome associated with IgA nephropathy. The result confirms our preliminary findings that corticosteroid treatment is beneficial to selected groups of patients.

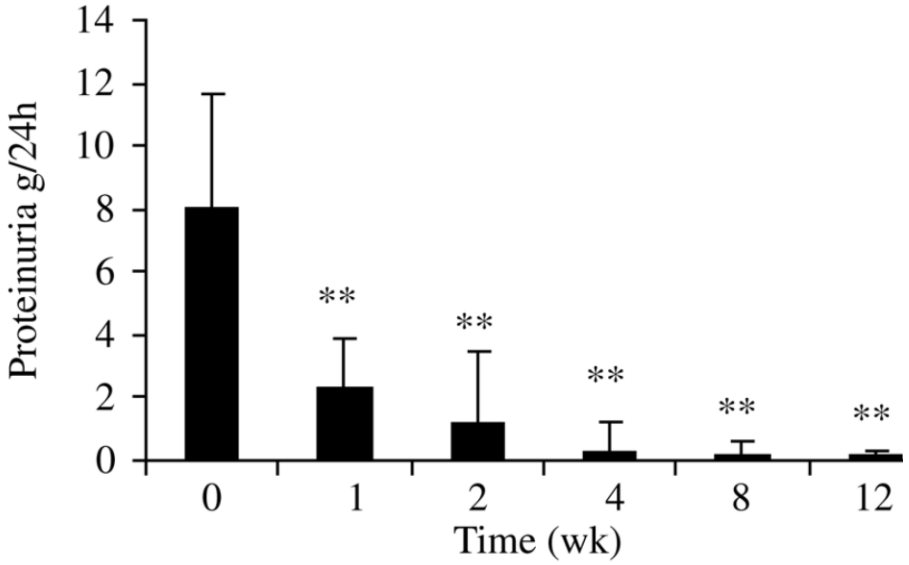
Received January 22, 1986, in revised form May 26, 1986.
Reprint requests to Dr. K. N. Lai.

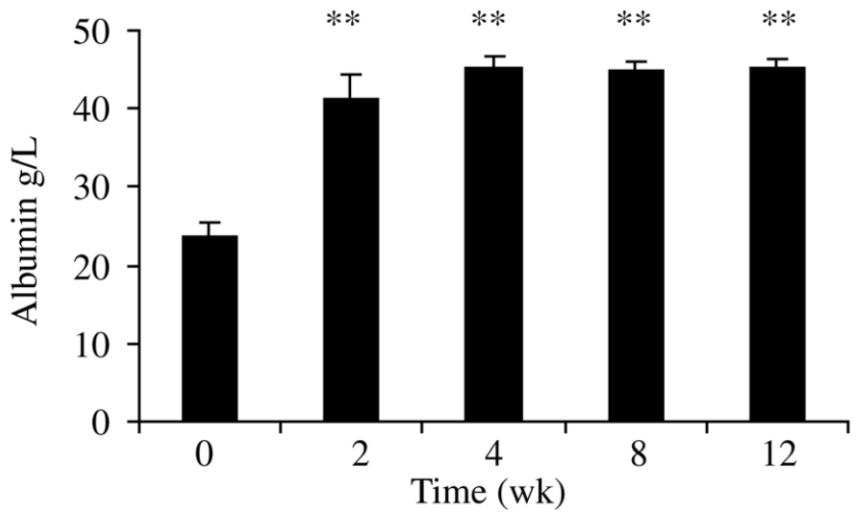
Patients and methods

Patients

From July 1977 to December 1984, IgA nephropathy was diagnosed in 145 patients. Thirty-six patients presented with nephrotic syndrome. Thirty-four nephrotic patients were selected for this study and the remaining two were excluded because of poor compliance. IgA nephropathy was diagnosed by the presence of predominant IgA deposits mainly in the mesangium and the presence of mesangial and paramesangial electron-dense deposits on ultrastructural

IgA nephropathy with minimal change-like lesions: a single-centre cohort study (n=27)



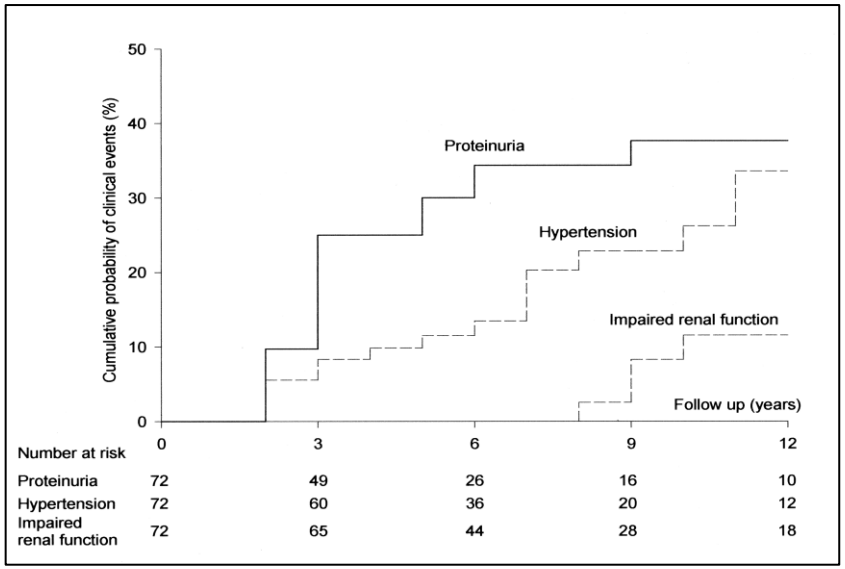


Nephrol Dial Transplant. 2013;28(9):2339

Natural history of IgA nephropathy among patients with hematuria and minimal proteinuria in Chinese

Baseline data
N=72
Age 27
Proteinuria: 0.1-0.4g/d
eGFR: >100ml/min
None hypertension
Follow-up data
Follow-up: 84mon (14-180)
ESKD: 1/72
eGFR<70ml/min: 5 (7%)
Proteinuria>1g/d: 24(33%)

Szeto CC *et al*. Am J Med. 2001,15;110(6):434-7



Baseline data
N=177
Age 38
Proteinuria: 0.1-0.4g/d
eGFR: >90ml/min
None hypertension
Follow-up data
Follow-up: 111mon (109-225)
ESKD: 0/177
eGFR<60ml/min: 43 (24%)
Proteinuria>1g/d: 67(38%)

Shen P *et al*. Neth J Med. 2008;66(6):242-7

Table 3. *Clinical data at the end of follow-up*

	Early IgAN	Normal biopsy	Normal dipstick
Patients (n)	177	135	120
Haematuria:			
• Disappearance	18 (10%)	67 (50%)	0 (0%)
• Persistent	135 (76%)	41 (38%)	5 (4%)
Proteinuria:			
• Remission	10 (6%)	90 (67%)	0 (0%)
• Stable	50 (28%)	14 (10%)	0 (0%)
• Increased	79 (46%)	3 (3%)	2 (2%)
Hypertension	68 (38%)	11 (8%)	8 (7%)
Renal insufficiency	43 (24%)	0 (0%)	0 (0%)

Long-Term Outcomes of IgA Nephropathy Presenting with Minimal or No Proteinuria In Caucasian

Baseline characteristics

N=141

Age 23.7

Proteinuria: ~0.5g/d

eGFR: >60ml/min/1.73m^2

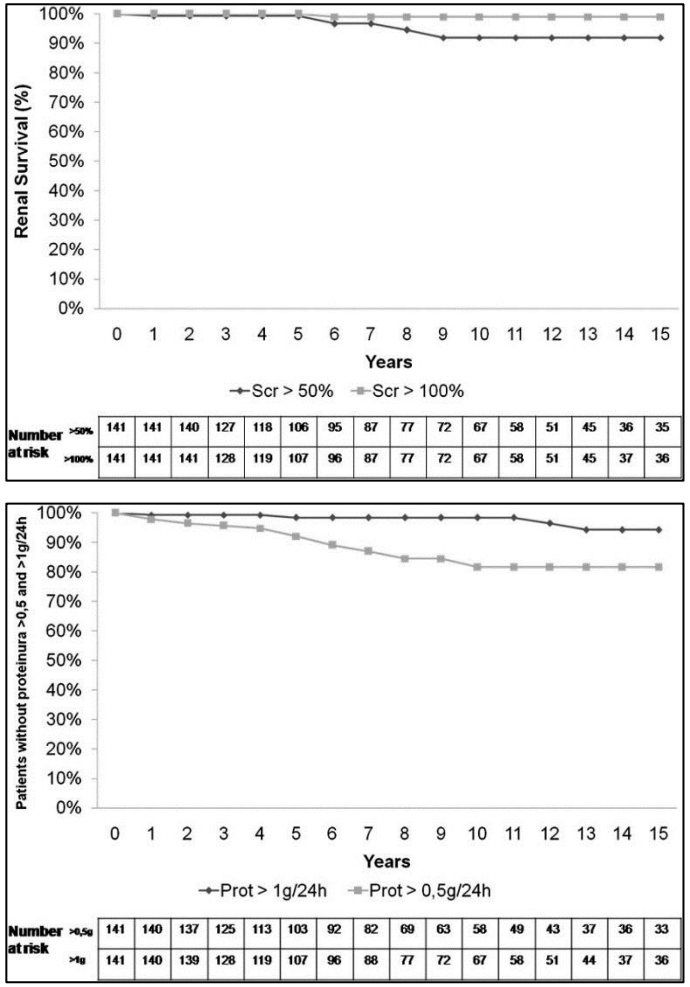
Follow-up data

Follow-up: 108mon (80-180)

ESKD: 0/141

Scr >50% : 5 (3.5%)

Proteinuria>1g/d: 6 (4.3%)



Eduardo Gutiérrez et al. JASN 2012;23:1753-1760

Outline

- **The nature history of IgAN**
  - General IgA nephropathy
  - Different phenotype of IgA nephropathy
    - Crescentic IgA nephropathy
    - IgA nephropathy with minimal change like lesions
    - Hematuria with or without minimal proteinuria
- **Can/have we changed it?**
- Perspective

KDIGO guidelines

- Blood pressure lowering
- RAS inhibitors
- Steroids

Progression of IgA nephropathy under current therapy regimen in a Chinese population

author	Sample size	Year	Proteinuria (g/d)	hypertension		Scr (umol/L)	eGFR (ml/min/1.73m²)	CKD stage					Follow	Therapy			TA-pro (g/d)	TA-MAP mmHg	GFR decline	Survival (%)			
				MAP	%			1	2	3	4	5		RASi	steroid	Steroid				5	10	15	20
Li et al. Hong Kong	168	1987-1996	1.67		28	152.7							7.4							88	82		
Lv et al. Beijing	204	1990-2001	2.7		41	132.6	81	38	35	13	10	3	6.1	56	36					85	77		
Le et al. Nanjing	1155	1989-2005	0.89		31	93.7	89	48	32	18	2	1	5.4	90	10.8		**0.54**	**94**	-1.6		85	76	67
Li et al. Beijing	703	2003-2011	2.5	94		101.7	84	46	32	11	6	5	3.8	96	45	53%	**1.12**	**90**	-3.12	89			
Zeng et al. Multi-center	1026	2012	1.3	98	33		85	41	35	24			4.4	89	31		**0.7**	**94**	-1.5				
VALIGA cohort	1147	2014	1.3	98	65				60		28	7	2	4.7	86	43		**0.8**	**96**	-1.8		74*		
North American	187	2011	1.7	96			82						4.4	87	37		**1**	**92**	-2.6				
Toronto cohort	542	2007	2.4	103		129.7	77						6.5	53	13		**2.2**	**100**	-4.56				

Li XL et al. Clin J Am Soc Nephrol. 2014;9(3):484-9

A tricontinental view of IgA nephropathy

lead-time bias

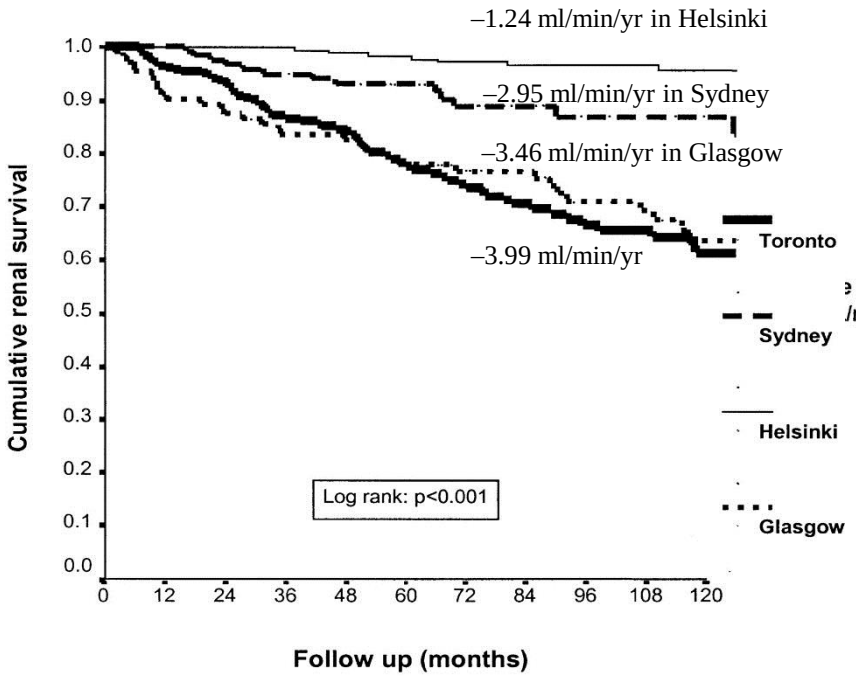
Table 1. Clinical characteristics of patients with biopsy-proven IgA nephropathy at presentation

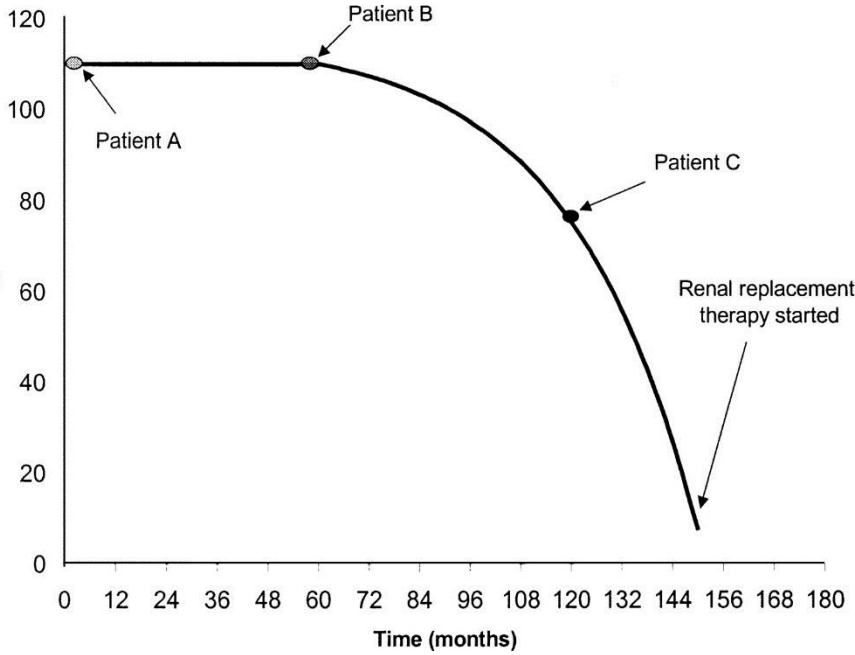
	Glasgow ($n=112$)	Helsinki ($n=204$)	Sydney ($n=121$)	Toronto ($n=274$)	P
All subjects					
Median year of presentation (range)	1989 (77–95)	1987 (80–95)	1985 (59–93)	1984 (63–97)	–
Median follow up (months)	86	123	73	53	$<0.001^{a}$
Mean age (years)	37.3	34.9	33.9	37.0	0.05^{b}
Male:female ratio	4.6	1.7	1.5	1.8	$<0.001^{c}$
Median serum creatinine (μmol/l)	118	90	110	115	$<0.001^{a}$
Mean CrCl (ml/min)	79.1	98.7	82.6	74.2	$<0.001^{b}$
Median urine protein (UP) excretion (g/day)	1.72	0.64	1.28	1.75	$<0.001^{a}$
Proportion with UP <0.5 g/day	22.9%	46.4%	26.2%	5.8%	$<0.001^{c}$
Mean of mean arterial blood pressure (mmHg)	105	94.0	103	105	$<0.001^{b}$
Subjects presenting CrCl <75 ml/min					
Number (%)	52 (46.4)	34 (16.7)	51 (42.1)	132 (48.2)	$<0.001^{c}$
Mean age (years)	45.3	47.6	37.7	39.1	$<0.001^{b}$
Mean CrCl (ml/min)	44.1	55.8	52.1	52.2	0.003^{b}
Median urine protein (UP) excretion (g/day)	2.24	1.28	1.28	2.2	0.006^{a}
Mean of mean arterial blood pressure (mmHg)	107	102	109	108	0.223^{b}

Nephrol Dial Transplant. 2003;18(8):1541-8

A tricontinental view of IgA nephropathy

lead-time bias





Number available for study	1yr	3yr	5yr	10yrs
Toronto	258	192	123	41
Sydney	121	111	73	28
Helsinki	204	196	166	109
Glasgow	102	90	78	34

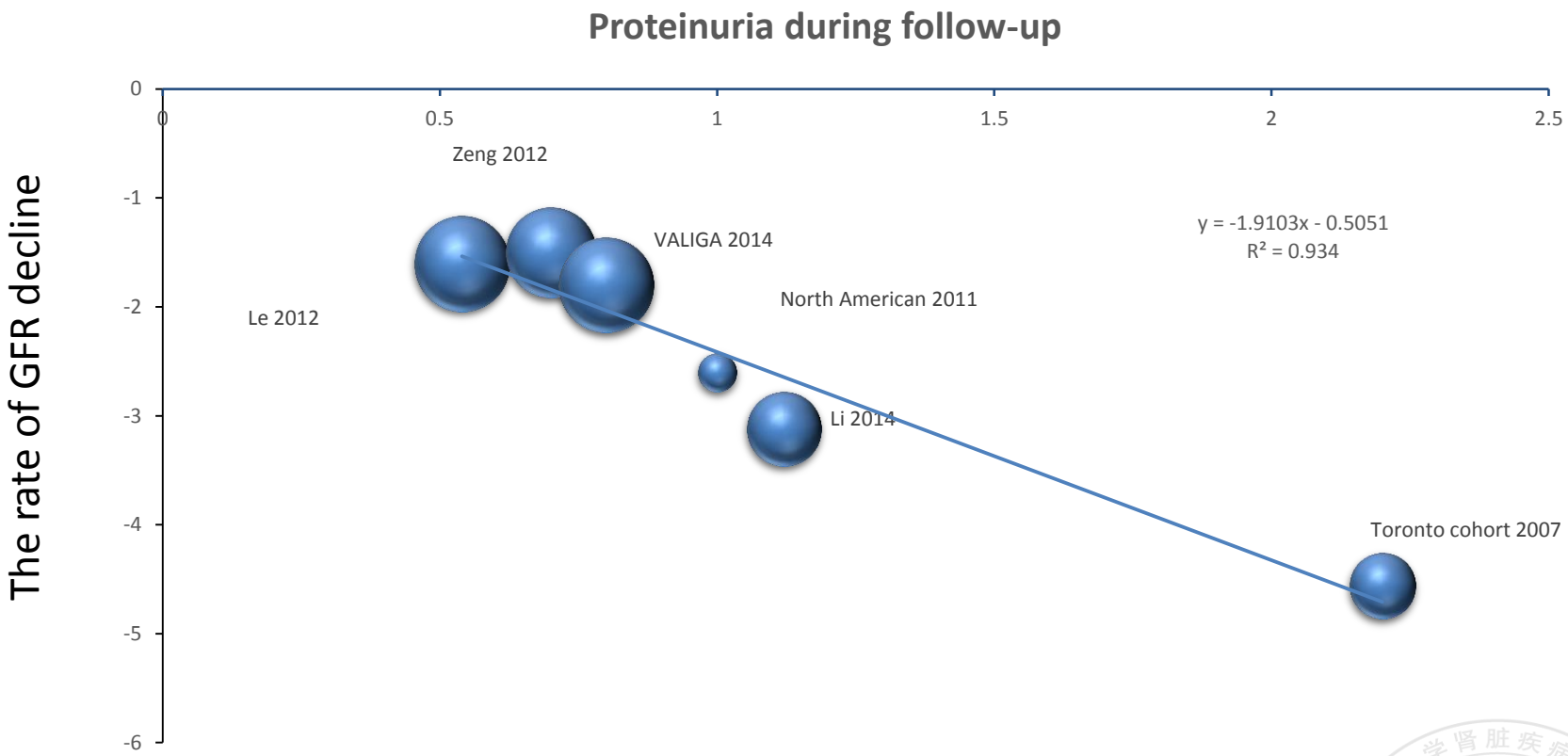
Nephrol Dial Transplant. 2003;18(8):1541-8

Progression of IgA nephropathy under current therapy regimen in a Chinese population

author	Sample size	Year	Proteinuria (g/d)	hypertension		Scr (umol/L)	eGFR (ml/min/1.73m²)	CKD stage					Follow	Therapy			TA-pro (g/d)	TA-MAP mmHg	GFR decline	Survival (%)			
				MAP	%			1	2	3	4	5		RASi	steroid	Steroid				5	10	15	20
Li et al. Hong Kong	168	1987-1996	1.67		28	152.7							7.4							88	82		
Lv et al. Beijing	204	1990-2001	2.7		41	132.6	81	38	35	13	10	3	6.1	56	36					85	77		
Le et al. Nanjing	1155	1989-2005	0.89		31	93.7	89	48	32	18	2	1	5.4	90	10.8		0.54	94	-1.6		85	76	67
Li et al. Beijing	703	2003-2011	2.5	94		101.7	84	46	32	11	6	5	3.8	96	45	53%	1.12	90	-3.12	89			
Zeng et al. Multi-center	1026	2012	1.3	98	33		85	41	35	24			4.4	89	31		0.7	94	-1.5				
VALIGA cohort	1147	2014	1.3	98	65				60	28	7	2	4.7	86	43		0.8	96	-1.8		74*		
North American	187	2011	1.7	96			82						4.4	87	37		1	92	-2.6				
Toronto cohort	542	2007	2.4	103		129.7	77						6.5	53	13		2.2	100	-4.56				

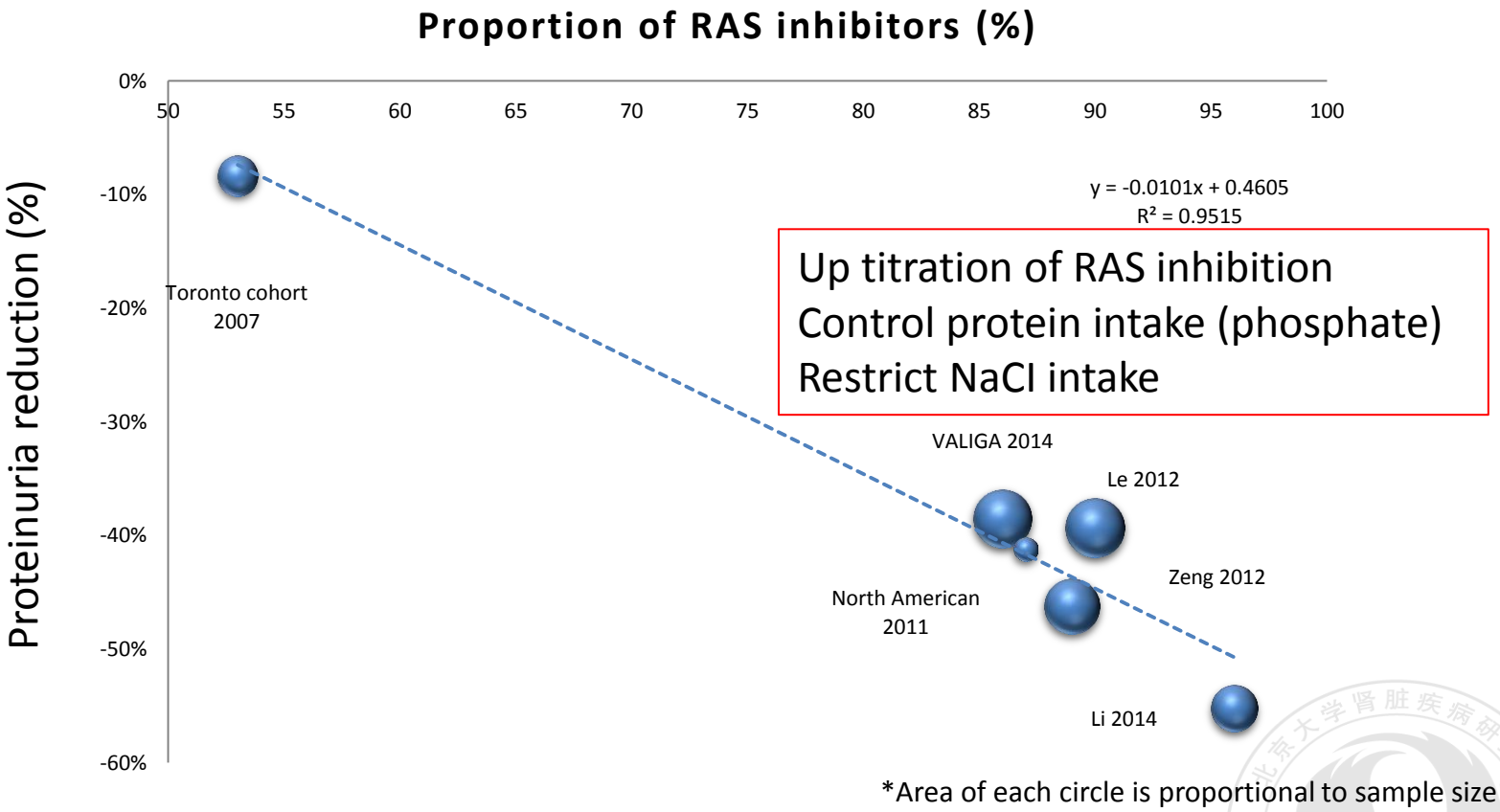
Li XL et al. Clin J Am Soc Nephrol. 2014;9(3):484-9

Association of time average proteinuria and rate of GFR decline



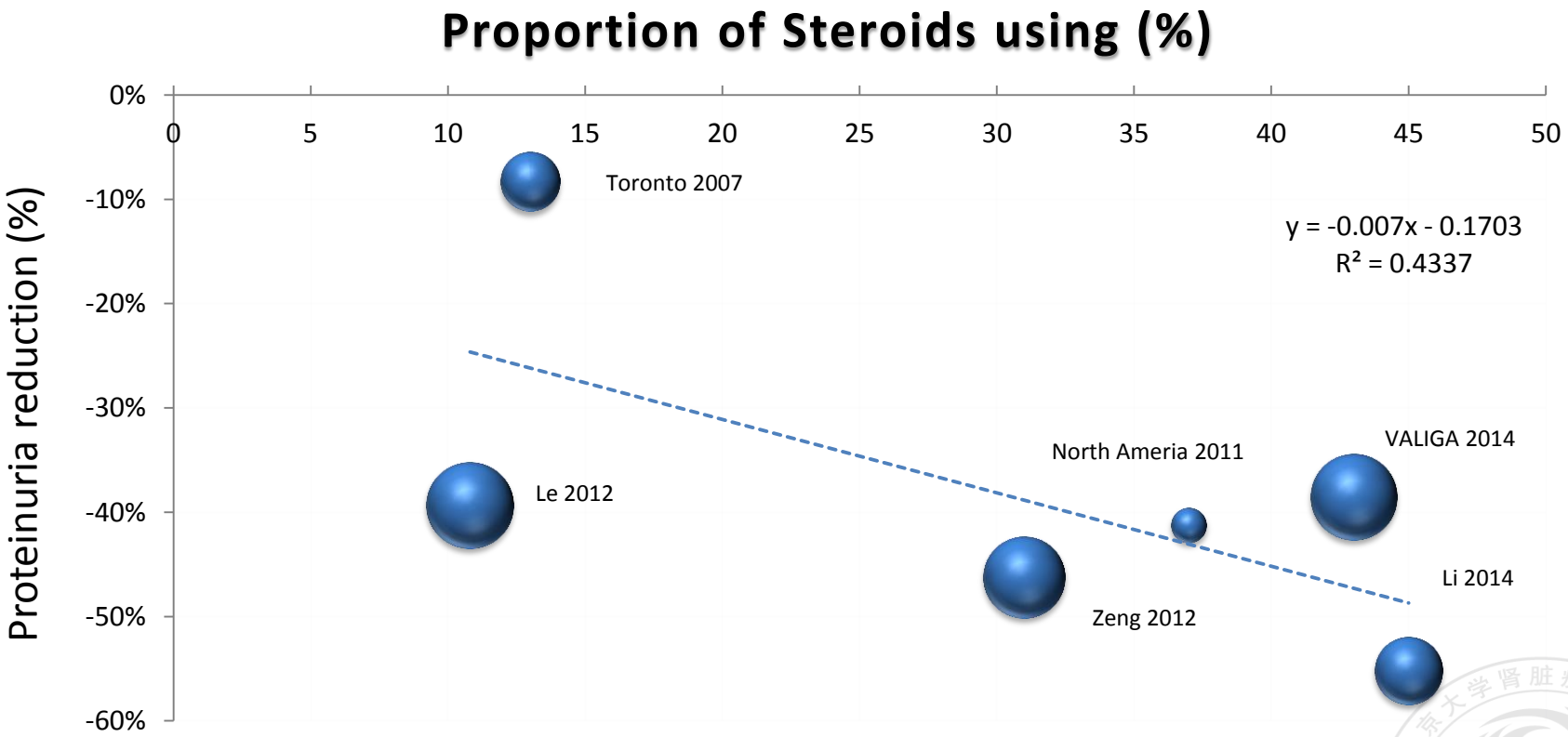
*Area of each circle is proportional to sample size

Interventions: RAS inhibitors



Association of proportion of RAS inhibitors and proteinuria reduction during follow-up

Interventions: Steroids using



*Area of each circle is proportional to sample size

Steroids reduced the risk of Kidney failure or persistent proteinuria reduction??

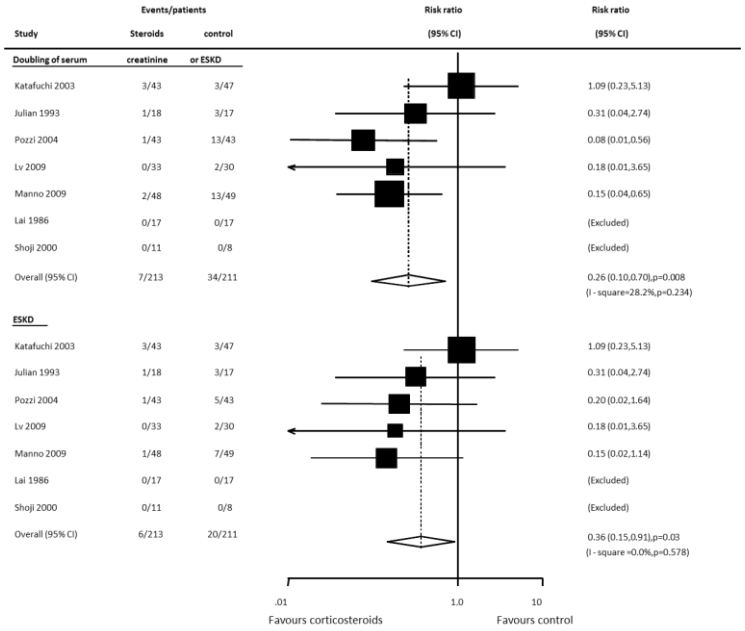


Figure : corticosteroids therapy on the outcomes of doubling of serum creatinine or ESKD

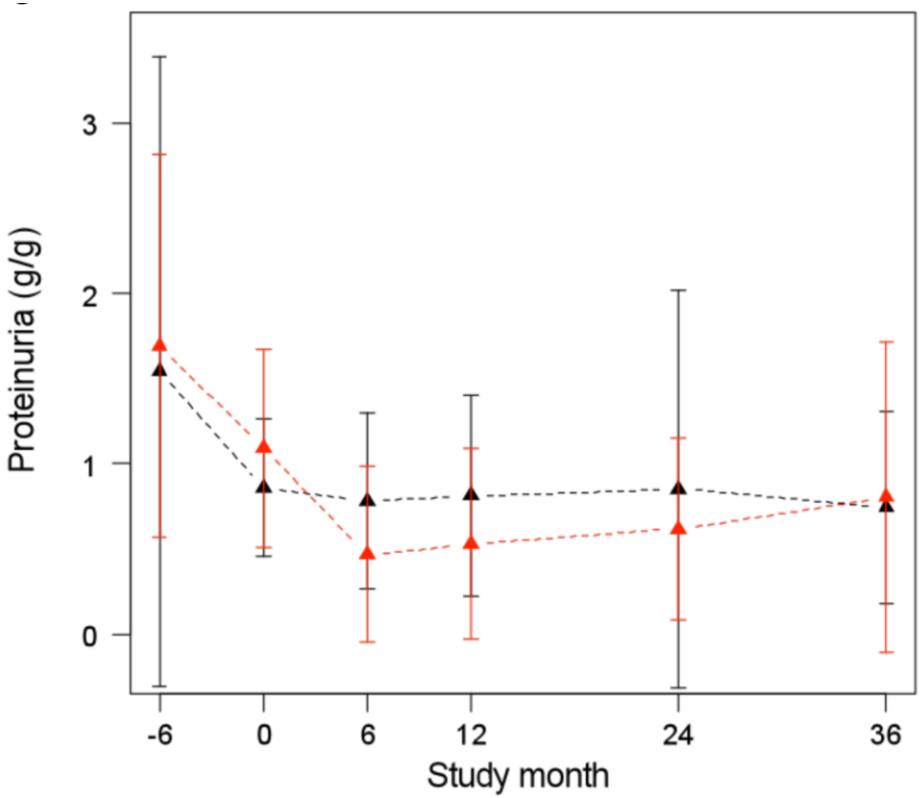
Lv J et al. JASN 2012

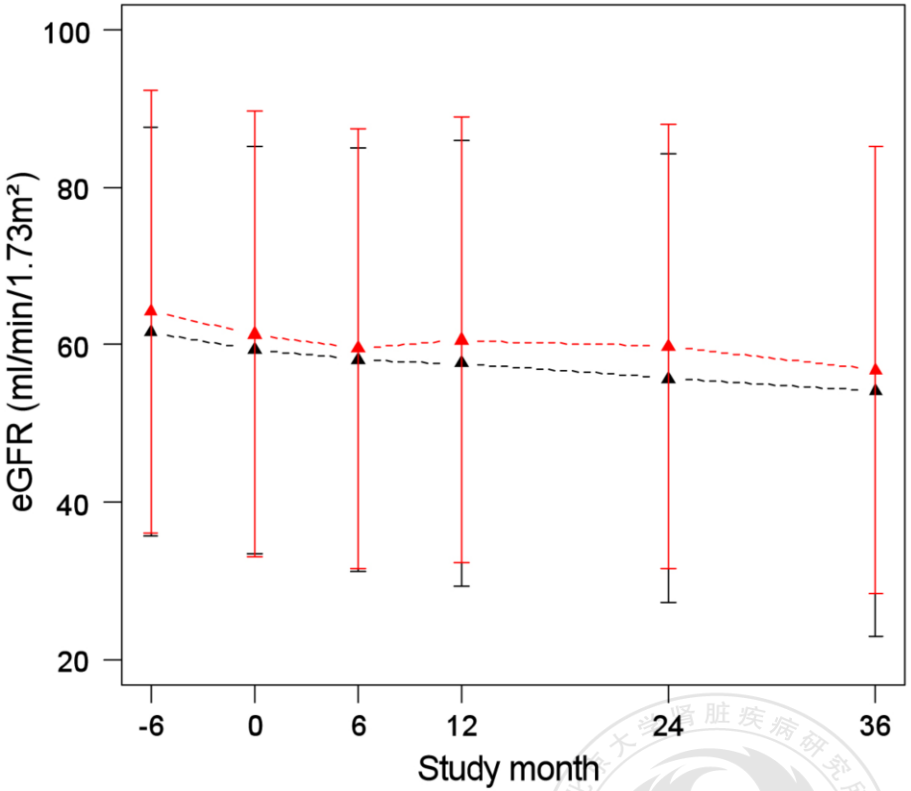
Table 2. Secondary End Points on the Basis of the Analysis of Available Cases at the End of the Trial Phase.*

Secondary End Point	Supportive Care (N=80)		Supportive Care plus Immunosuppression (N=82)		Odds Ratio (95% CI)	P Value
	Patients with Available Data	End-Point Value	Patients with Available Data	End-Point Value		
	no.	*mean ±SD or no. (%)*	*no.*	*mean ±SD or no. (%)*		
Absolute eGFR change at 36 mo — ml/min/1.73 m^2	71	−4.7±12.3	72	−4.2±14.1	Not determined	0.32
Mean annual change in the slope of the reciprocal of serum creatinine concentration — mg/dl	77	−0.02±0.06	74	−0.01±0.06	Not determined	0.60
At 12 mo	67	0.80±0.67	59	0.57±0.53	Not determined	0.01
At 36 mo	64	0.85±0.66	59	0.76±0.90	Not determined	0.66
eGFR decrease ≥30 ml/min/1.73 m^2	76	7 (9)	78	10 (13)	1.45 (0.51–4.10)	0.49
Onset of end-stage renal disease	76	6 (8)	78	6 (8)	0.97 (0.29–3.22)	0.96
Disappearance of microhematuria	55†	9 (16)	57†	24 (42)	3.73 (1.52–9.14)	0.004

STOP study. NEJM 2015

Steroids reduced the risk of Kidney failure or persistent proteinuria reduction??





STOP study. NEJM 2015

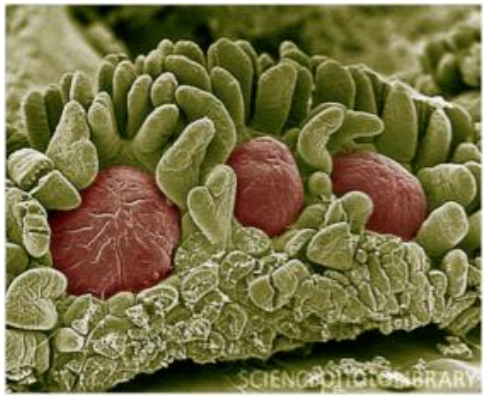
The NEFIGAN Trial

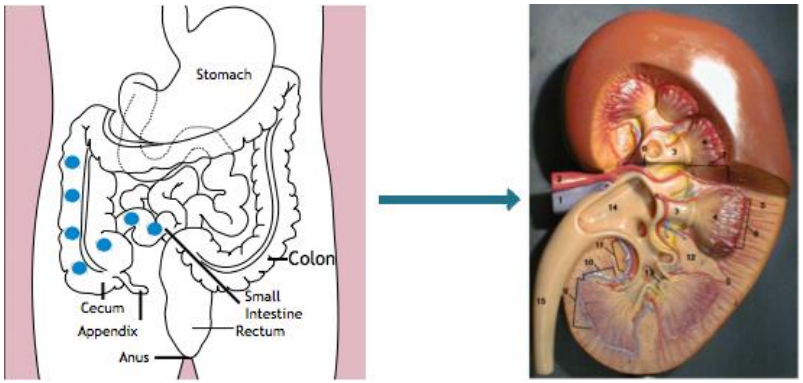
The NEFIGAN Trial is a clinical study evaluating the safety and effectiveness of the drug, NEFECON, in patients with IgA nephropathy. The study is sponsored by Pharmalink AB.

NEFECON, an oral formulation that releases budesonide, a glucocorticosteriod, in the lower ileum and ascending colon of the gastrointestinal (GI) tract.

NEFECON significantly reduced proteinuria as compared to placebo at 9 month by 25-30%

The long-term outcomes on proteinuria or GFR decline?





ASN 2015 late breaking trials
From http://www.nefigan.net/

TESTING

Therapeutic Evaluation of Steroids in IgA Nephropathy Global Study

Institute of Nephrology Peking University

北京大学肾脏疾病研究所

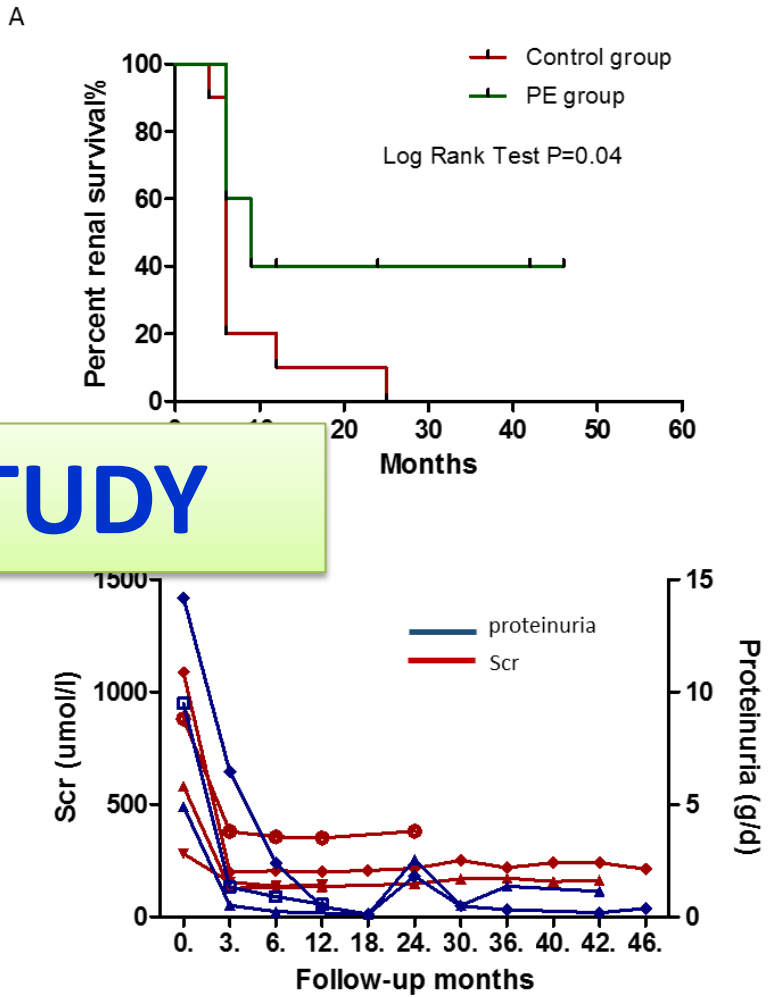
THE GEORGE INSTITUTE for Global Health

Plasma Exchange as an Adjunctive Therapy for Crescentic IgA Nephropathy

Treatment and clinical outcomes

	PE group (n=10)	Control group (n=10)
Age	44(16)	40(15)
Gender(male)	7	10
Scr (μmol/L)	657(248)	658(270)
Dialysis at presentation	7	8
Total crescent (%)	71.0(17.0)	71.3(17.6)
Follow-up months		
Follow-up months		
Courses of PE/patient	7(5-10)	0
Pulse methylprednisolone	8	9
Steroids +immunosuppresants	8	8
Steroids alone	2	2
RAS blocker	5	5
ESRD	5（50%）+1	10（100%）
Death	1	3

RESCUES STUDY



Outline

- The nature history of IgAN
  - General IgA nephropathy
  - Different phenotype of IgA nephropathy
    - Crescentic IgA nephropathy
    - IgA nephropathy with minimal change like lesions
    - Hematuria with or without minimal proteinuria
- Can/have we changed it?
- **Perspective**

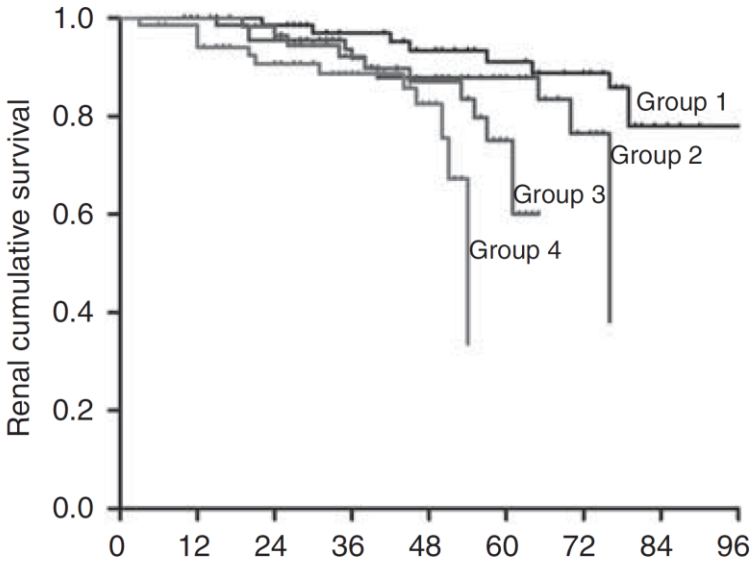
How to define IgA nephropathy in terms of pathogenesis

Who determine the heterogeneity of IgAN Behind clinical parameters?

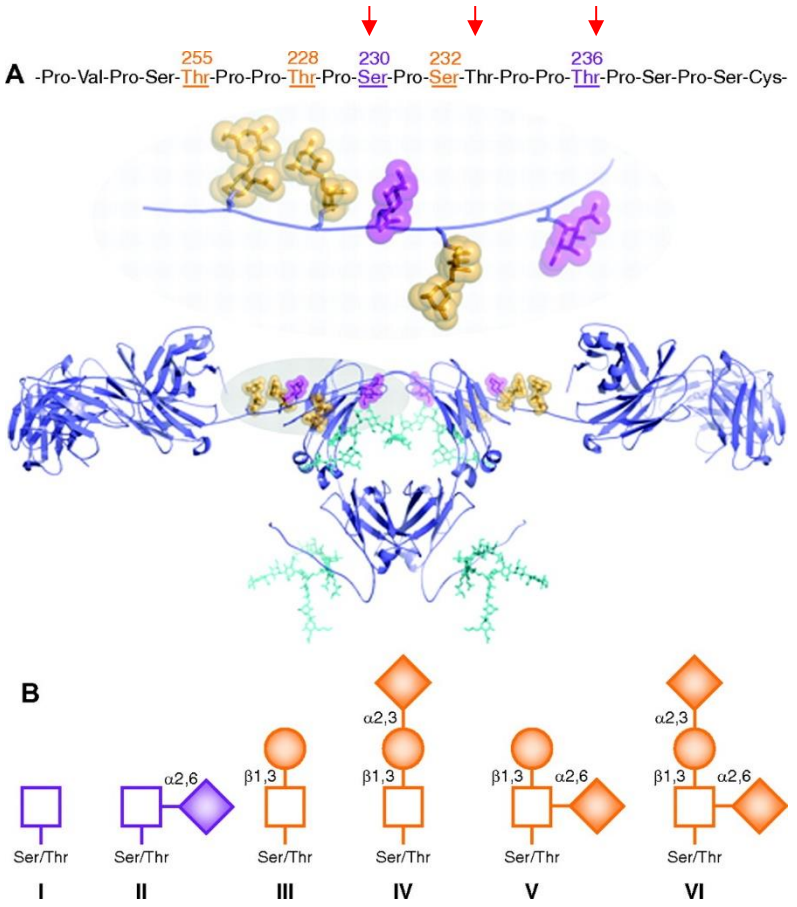
What's the profiles of galactose-deficient IgA1 ?

original article http://www.kidney-international.org

The level of galactose-deficient IgA1 in the sera of patients with IgA nephropathy is associated with disease progression



Zhao N et al. Kidney Int 2012



J Am Soc Nephrol 2011,22: 1795

THANKS FOR YOUR ATTENTION