

IgA Nephropathy in China

Long-term Outcome and Oxford Classification



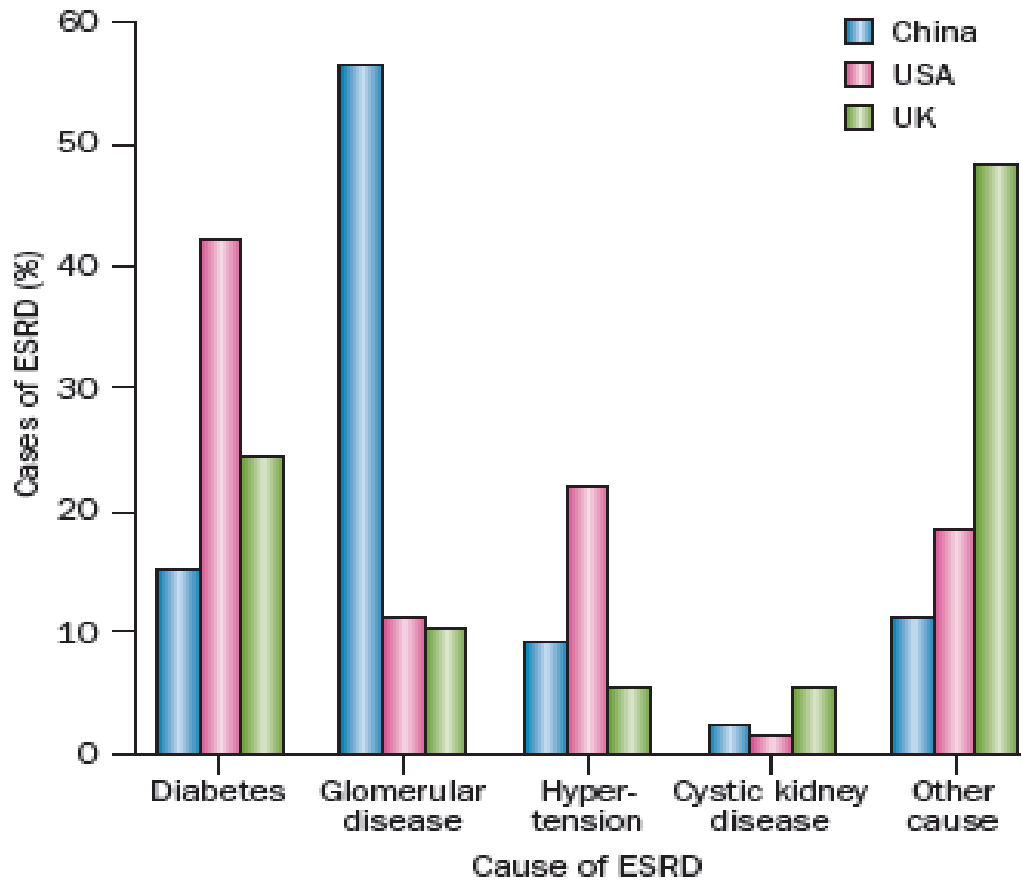
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Jinling Hospital

Nanjing University School of Medicine

Nanjing, China



Different causes of ESRD in China, the USA and the UK

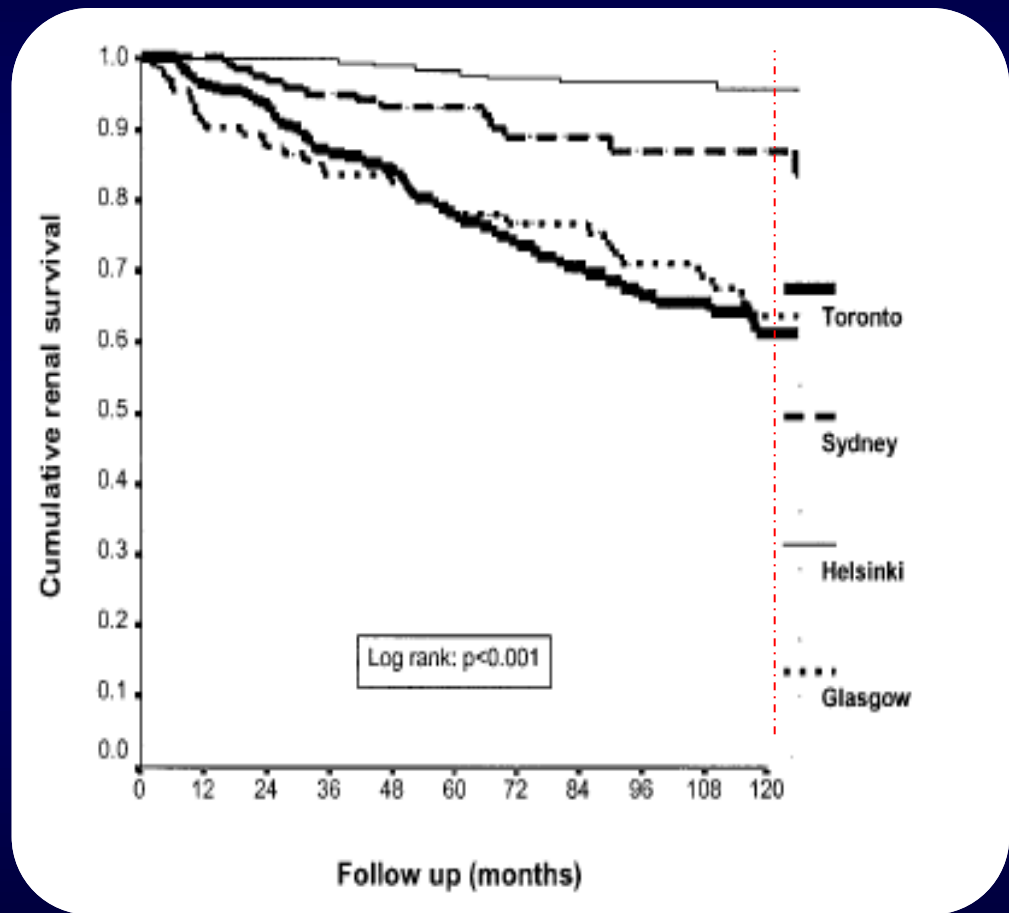
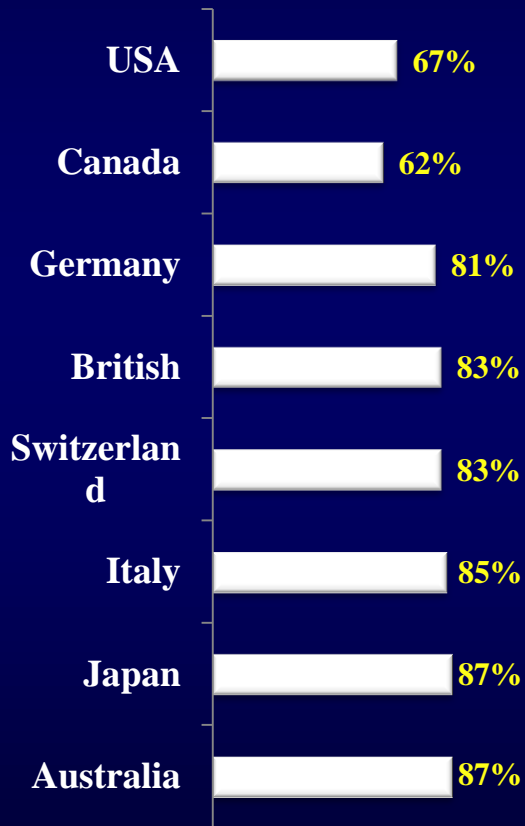
原发性肾小球疾病

(18813 cases, 2000-2010, 南京)

	n	%
IgAN	8580	45.61
FSGS	2400	12.87
MN	2422	12.76
IgMN	240	1.28
MPGN	242	1.29
EnPGN	213	1.13

Long-term renal outcome of IgAN varies in different population

10-Year renal survival



Long-term renal survival and related risk factors in patients with IgA nephropathy: results from a cohort of 1155 cases in a Chinese adult population

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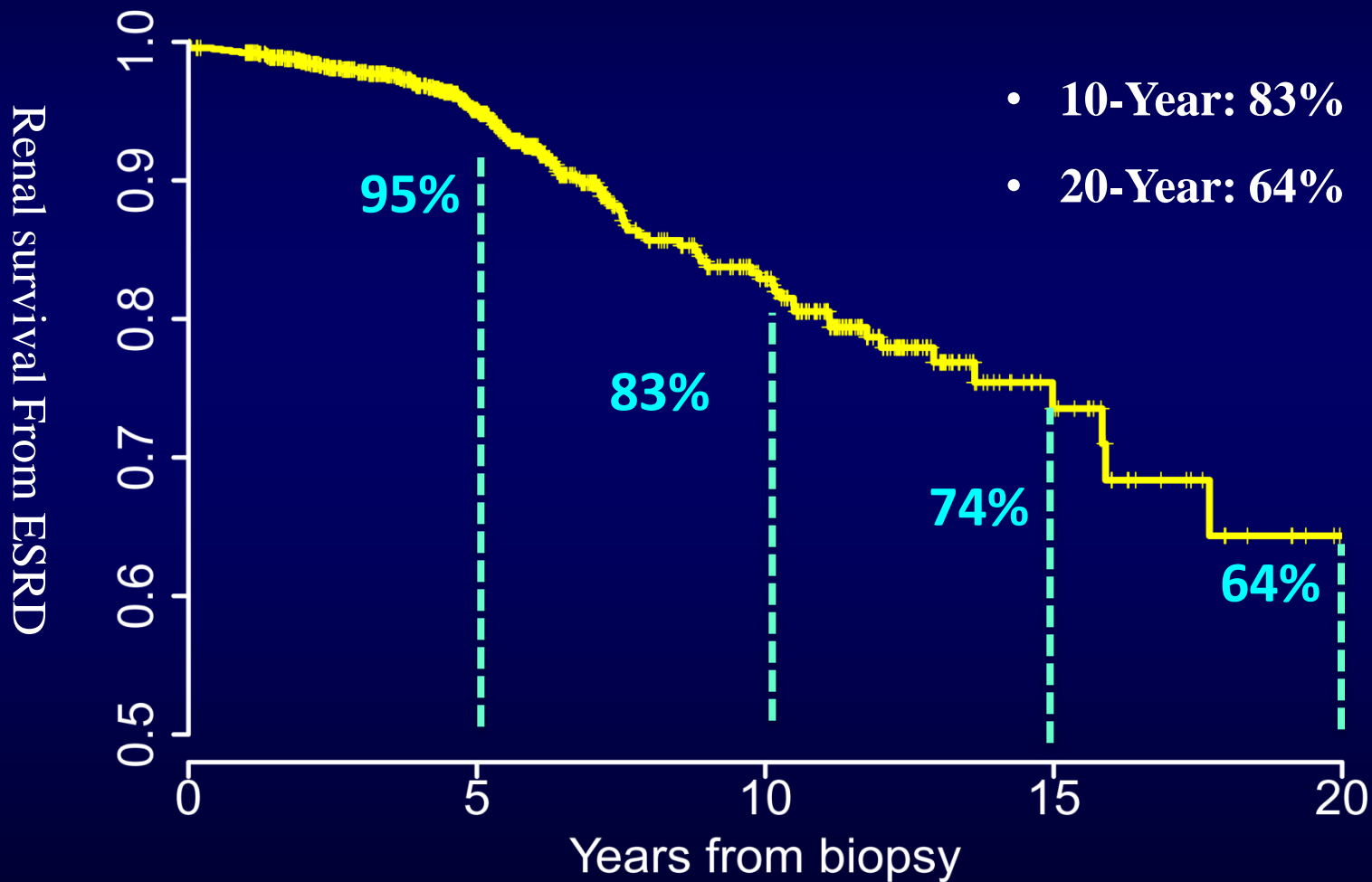
Abstract

Background. We sought to identify the long-term renal survival rate and related risk factors of progression to renal failure in Chinese adult patients with IgA nephropathy (IgAN) and to quantify the effects of proteinuria during the follow-up on outcome in patients with IgAN.

Introduction

IgA nephropathy (IgAN) is the most common primary glomerulonephritis worldwide and is particularly prevalent in Asia. Our previous study showed that IgAN accounts for 45% of the primary glomerulonephritis in China [1]. IgAN

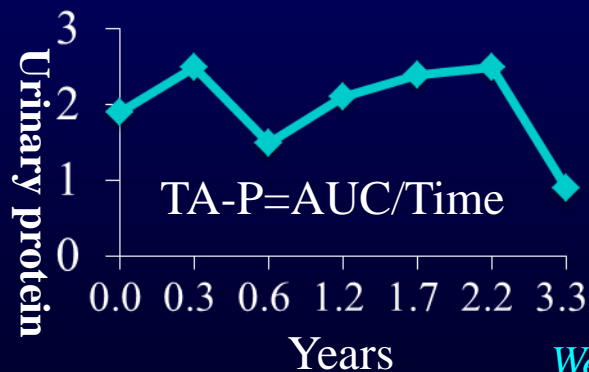
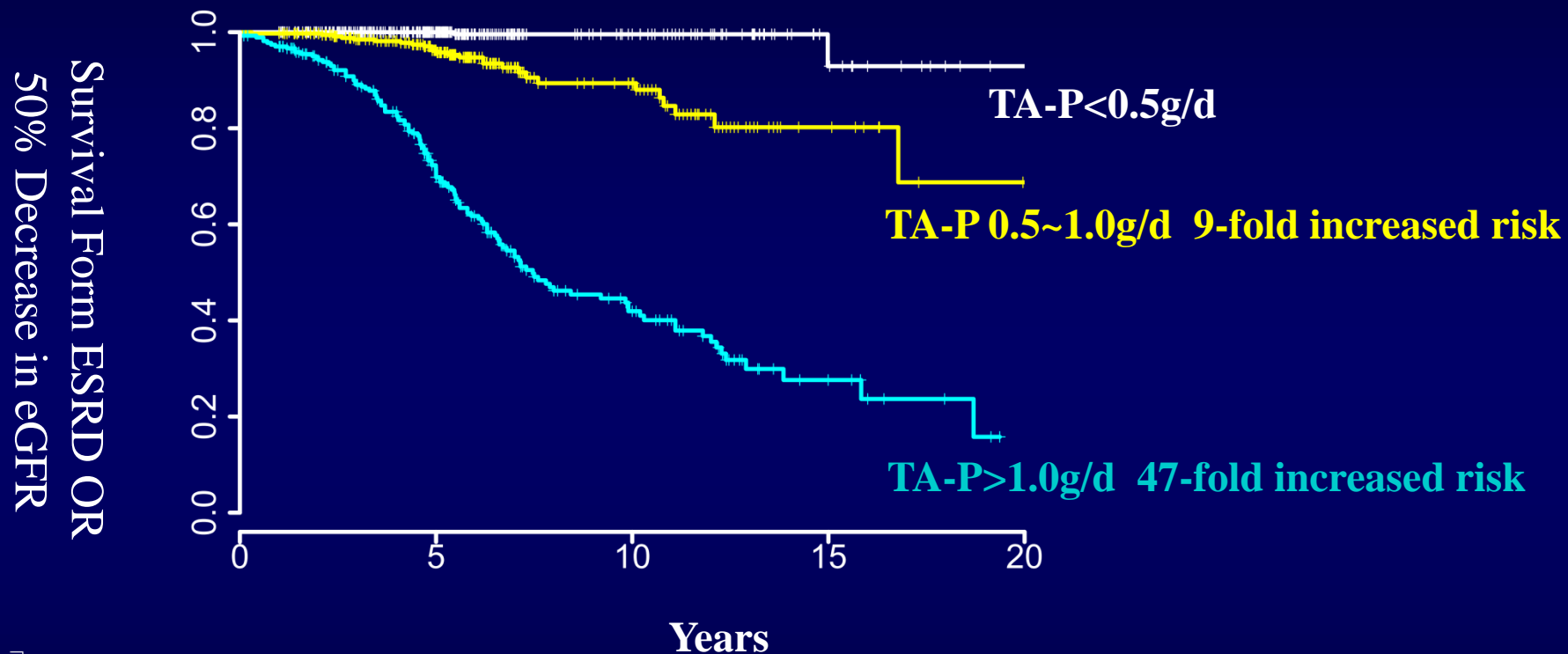
Renal survival of Chinese Patients with IgAN (1155 Cases)



Five key determinants of renal survival in IgAN

	Multivariate Cox regression	
	HR(95% CI)	P
Urinary protein>1.0g/d	3.3(2.2-4.9)	<0.001
Hypertension (>140/90mmHg)	1.9(1.3-2.7)	<0.001
eGFR<60ml/min per 1.73m ²	2.6(1.4-2.8)	<0.001
Hypoalbuminemia	2.0(1.4-2.8)	<0.001
Hyperuricemia	1.8(1.2-2.6)	0.002

Time-average proteinuria (TA-P) and renal survival



The predictors of renal survival from data during the Follow-up period

Predictors	Univariate Cox regression		Multivariate Cox regression [△]	
	HR(95% CI)	P Value	HR(95% CI)	P Value
TA-P	1.8(1.7-1.9)	<0.001	1.8(1.6-1.9)	<0.001
TA-MAP	1.04(1.03-1.06)	<0.001	1.03(1.01-1.04)	<0.001
TA-RBC	1.3 (1.1-1.7)	0.01	2.1 (1.6-2.7)	<0.001

TA-P : Time-average proteinuria

TA-MAP: Time-average mean arterial blood pressure

TA-RBC: Time-average hematuria

IgA Nephropathy Variations in

Pathological pattern

Clinical pattern

Geographical prevalence

Gender prevalence

IgA Nephropathy

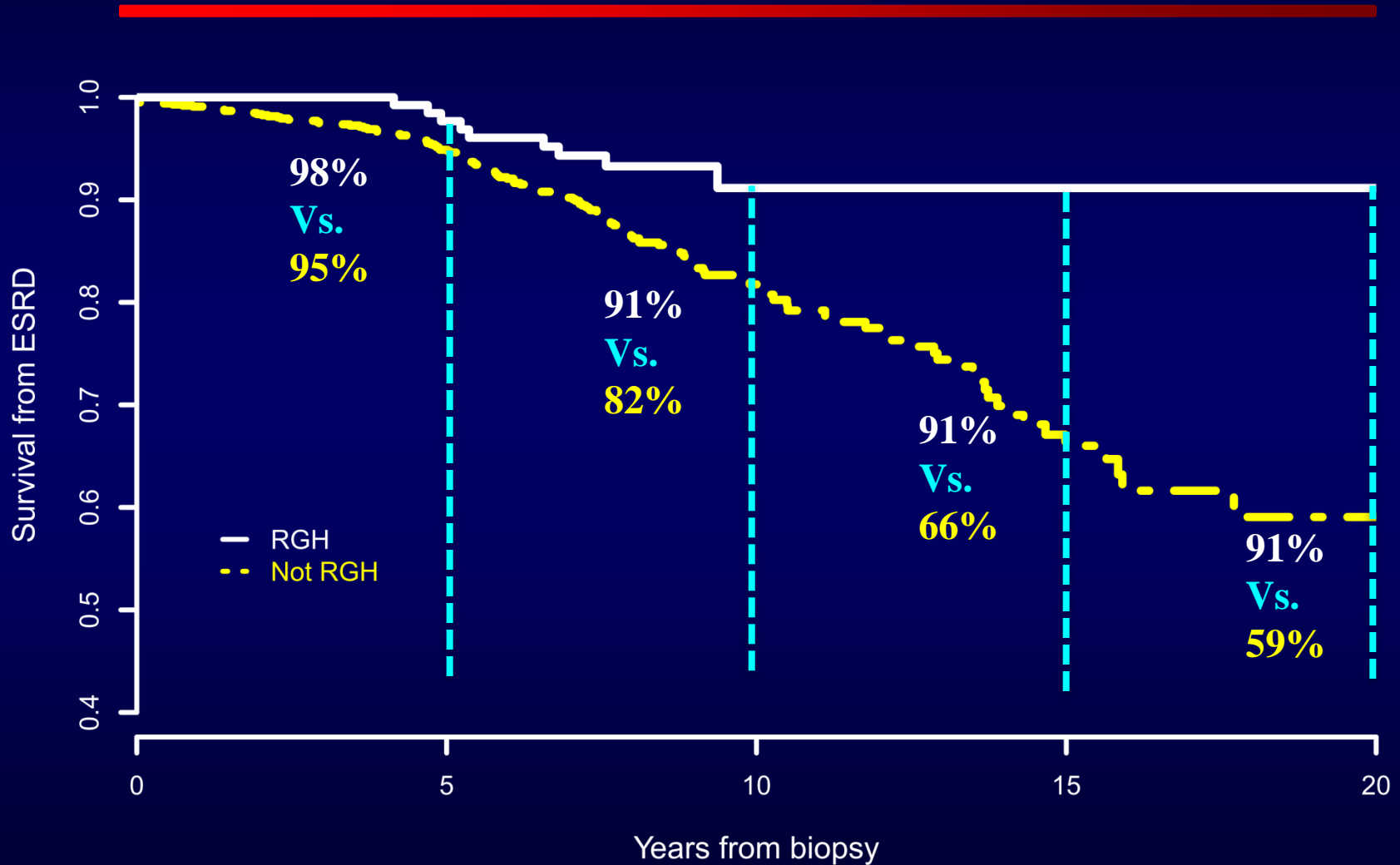
- IgAN with recurrent macroscopic hematuria
- IgAN with minimal change disease
- **IgAN in elderly patients**

The long-Term Outcome of IgAN Patients with Recurrent Gross Hematuria

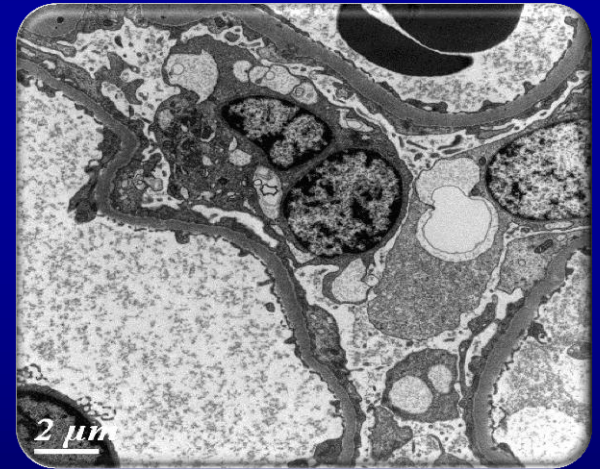
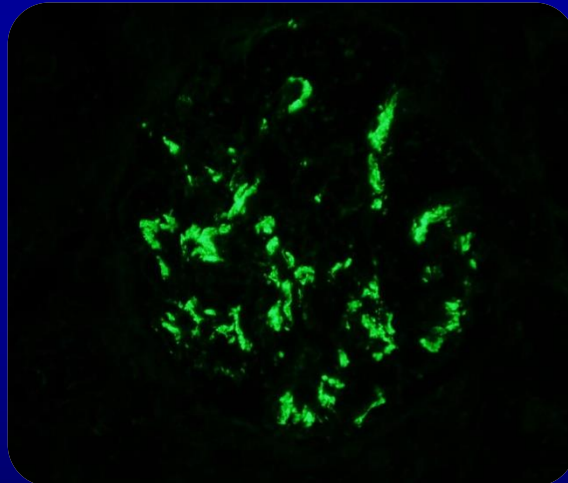
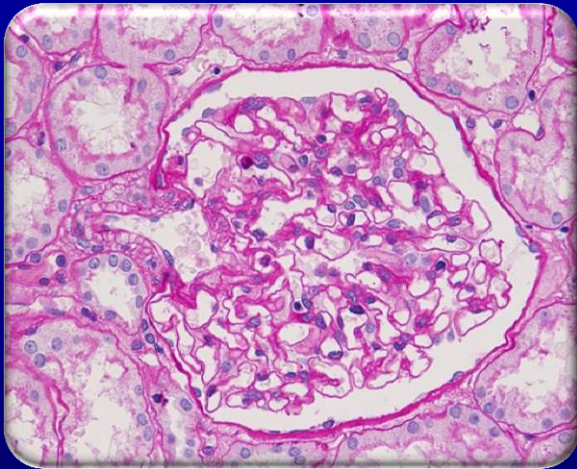
rGH: recurrent macroscopic hematuria (n= 158)

nGH: none macroscopic hematuria (n=741)

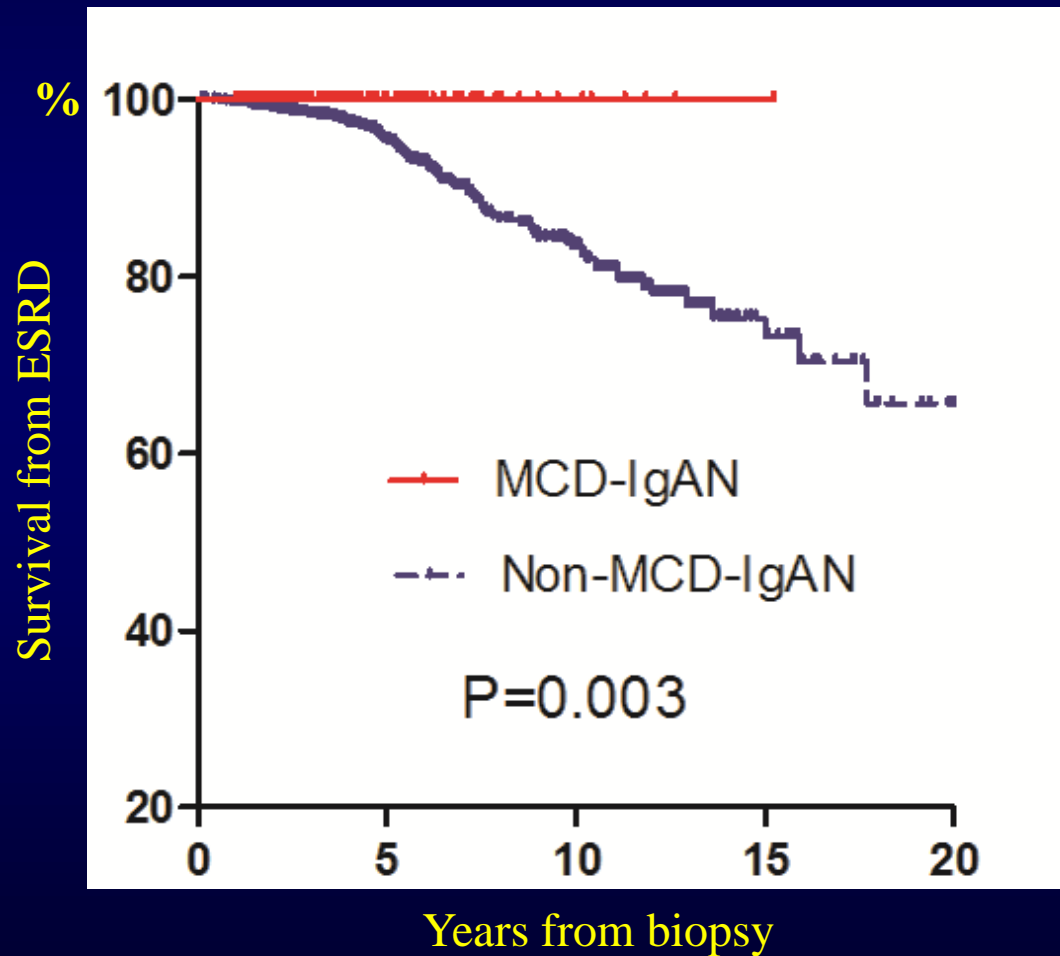
Long-term outcome of IgAN with recurrent macroscopic hematuria



IgAN with minimal change disease

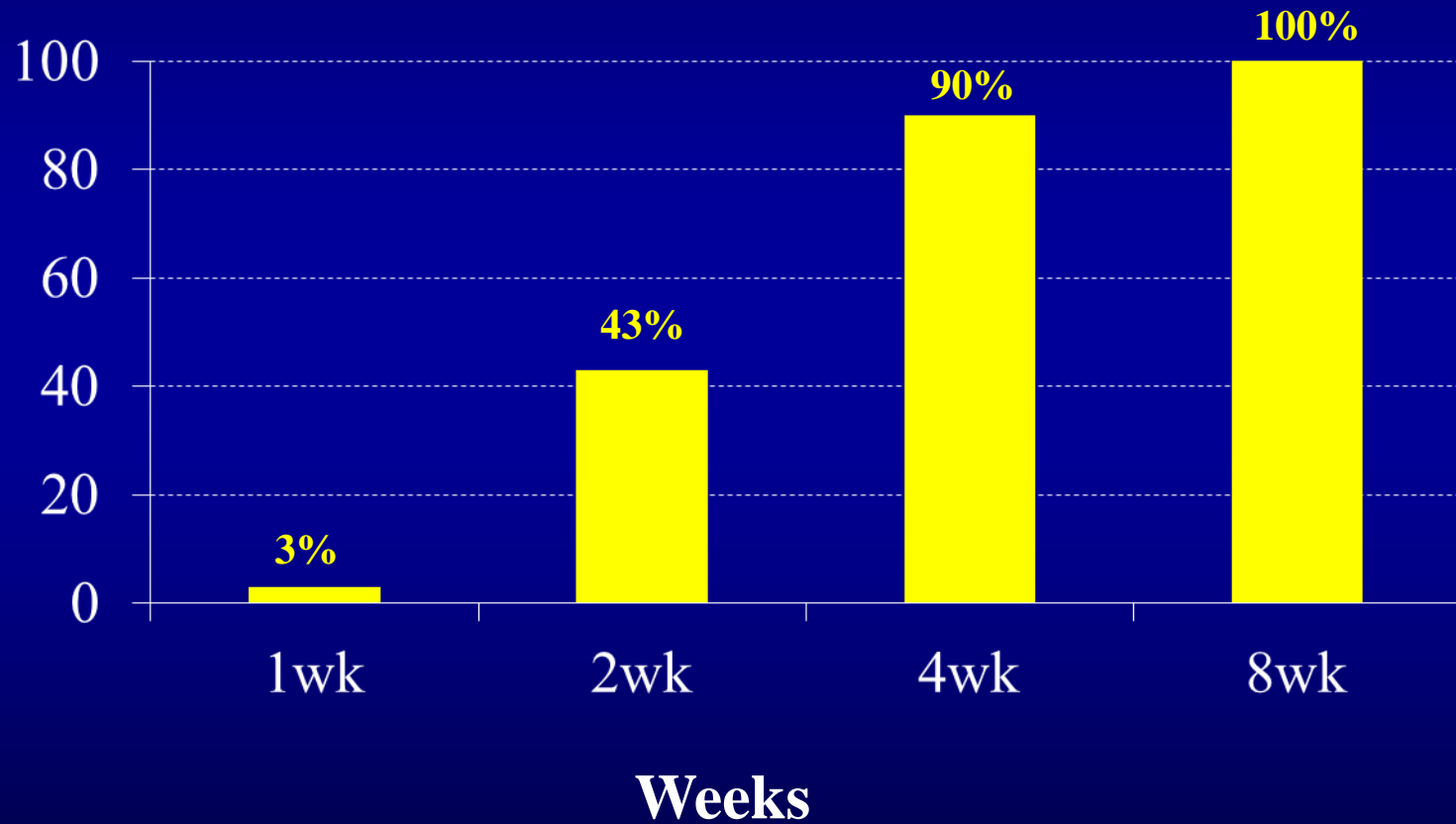


Long-term outcome of IgAN with minimal change disease

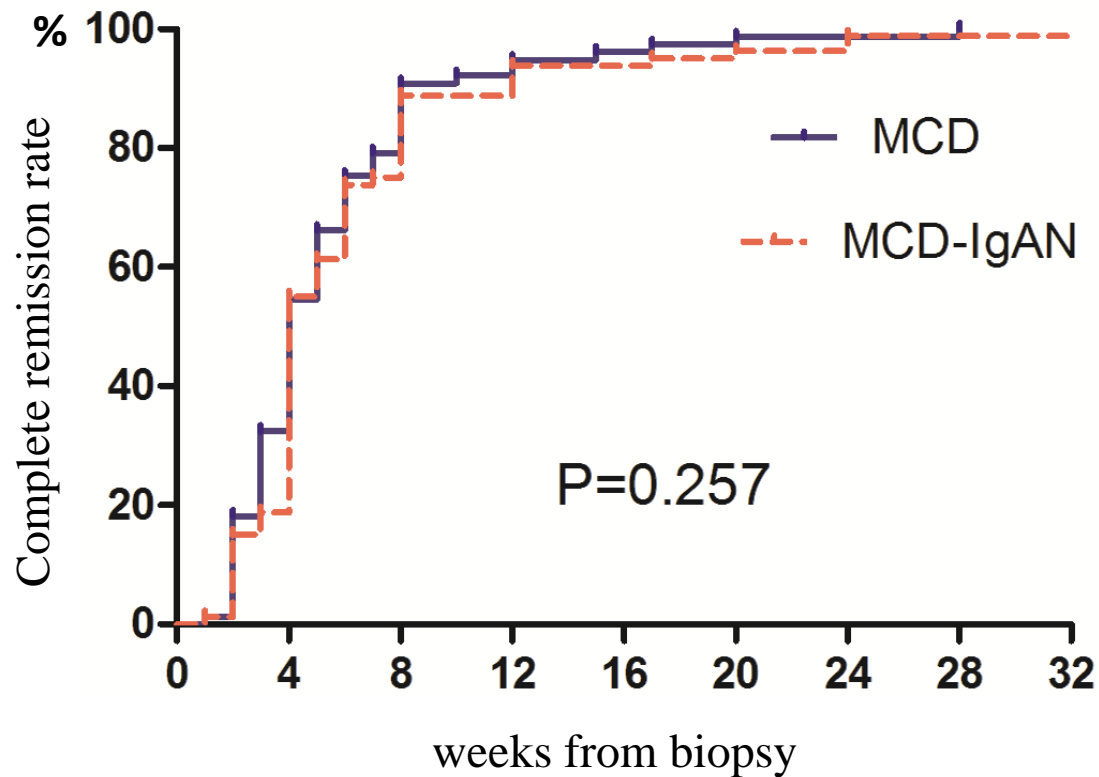


Corticosteroids treatment in IgAN with minimal change disease

Complete remission rate



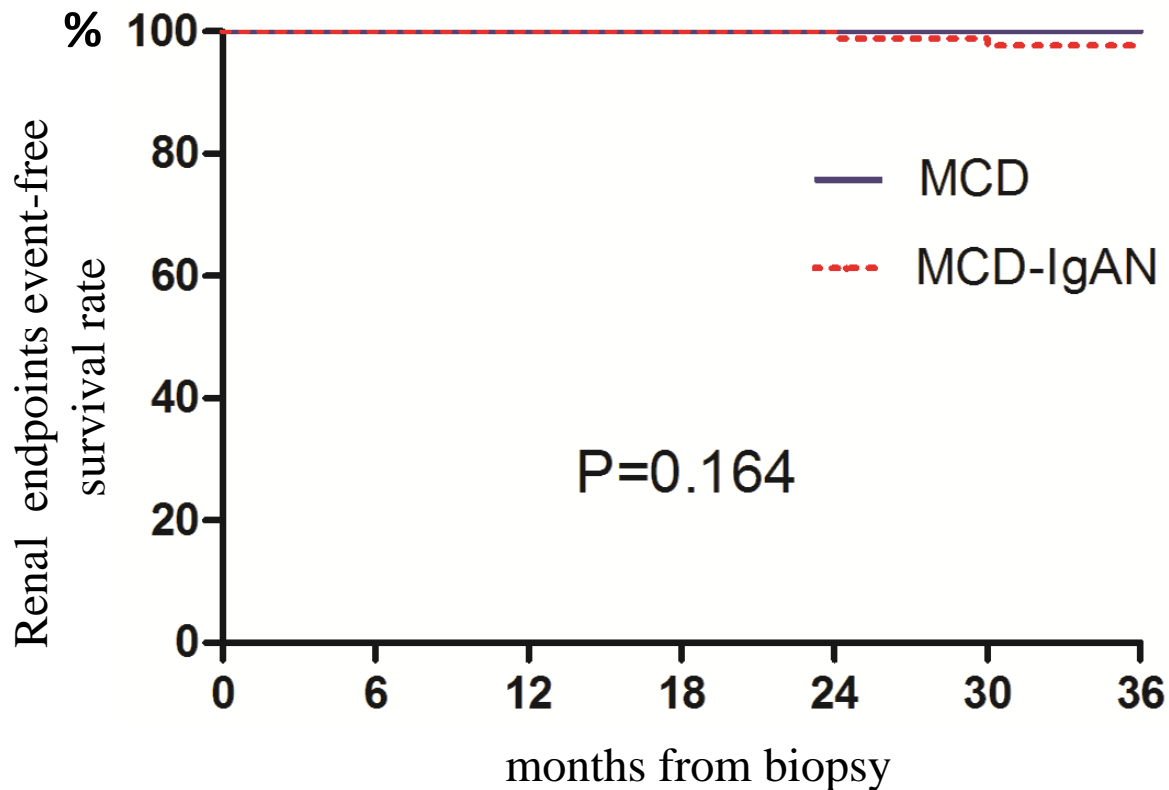
Response to steroid treatment of IgAN patients with minimal change disease



MCD: Minimal change disease (n=77)

MCD-IgAN: IgAN with minimal change disease (n=80)

Comparison of long-term outcome between patients with MCD-IgAN and MCD

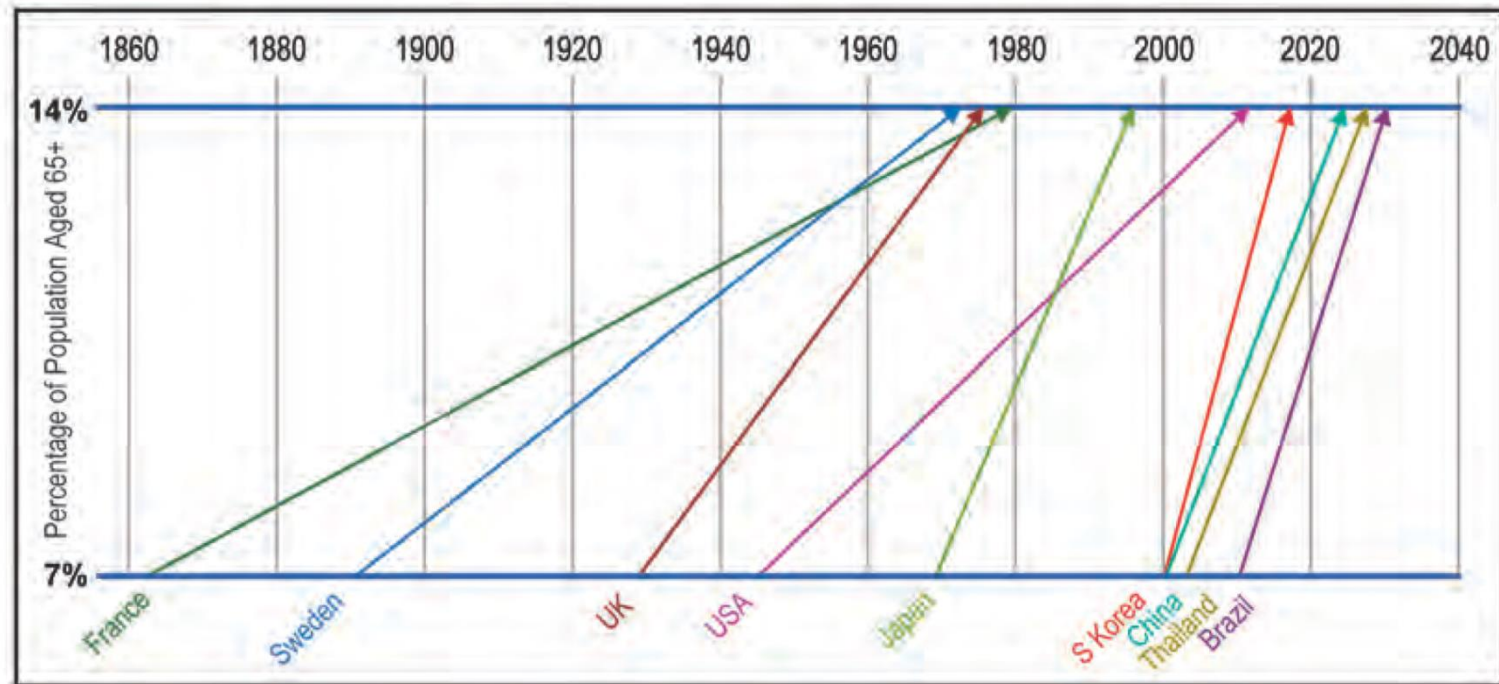


MCD: Minimal change disease (n=77)

MCD-IgAN: IgAN with minimal change disease (n=80)

The Speed of Population Aging in China

Time required or expected for percentage of population aged 65 and over to rise from 7 percent to 14 percent



China is one of countries with the highest speed of population aging in worldwide

Source: Kinsella K, He W, *An Aging World*, 2008.

Washington DC. National Institute on Aging and US Census Bureau, 2009

The spectrum of biopsy-proven kidney diseases in elderly Chinese patients

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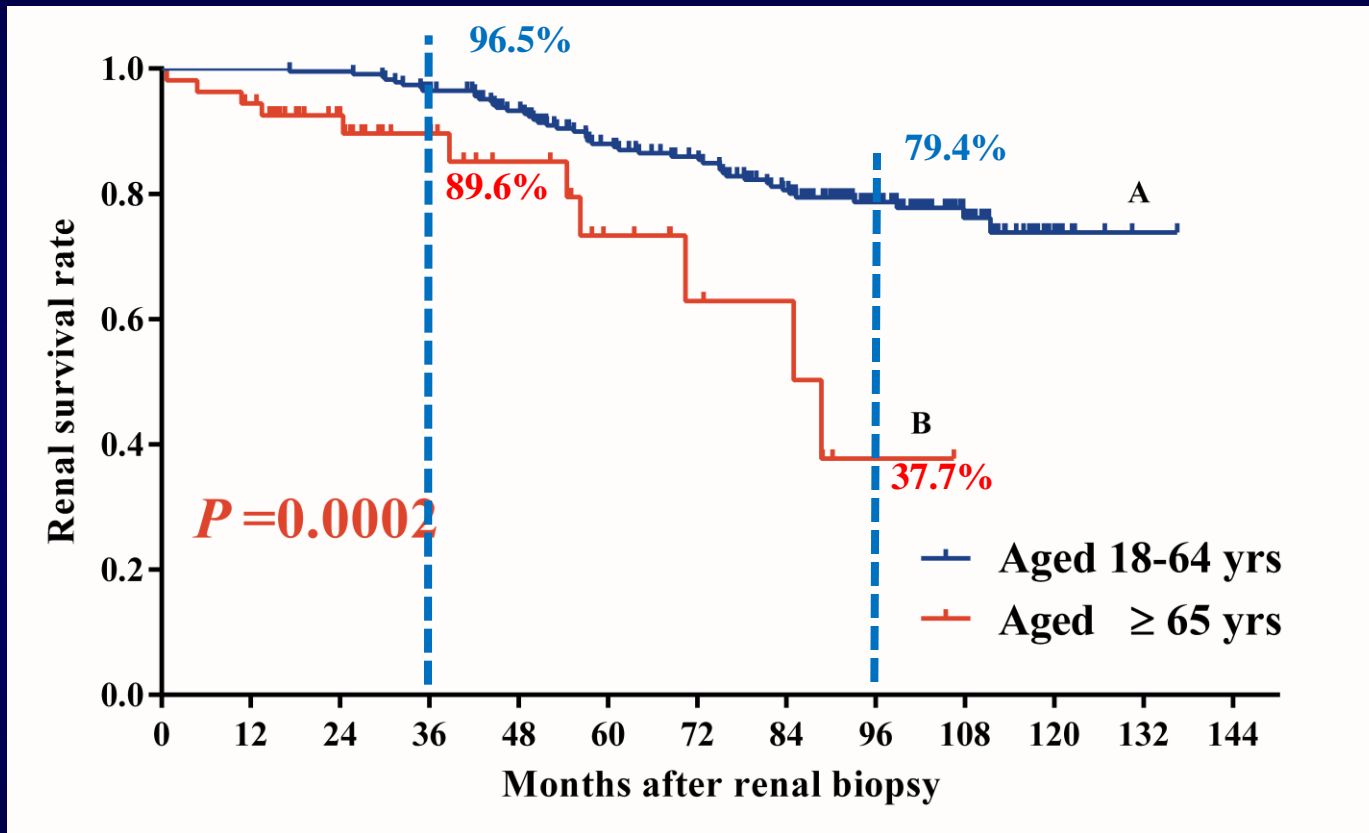
>65 y

N=851

Table 1. Classification of renal diseases based on renal biopsies in elderly patients

	Number of cases	%
Primary glomerular disease	459	53.94
MN	245	28.79
IgAN	82	9.64
MCD	41	4.82
FSGS	40	4.70
MsPGN	24	2.82
IgMN	9	1.06
MPGN	7	0.82

老年与非老年IgAN患者肾脏累积生存率的比较



老年与非老年IgAN患者临床特征的比较

	≥65岁 (n=82)	18-64岁 (n=328)	P值
女性, n (%)	36 (43.9)	167 (50.9)	0.256
年龄 (岁)	68.9 ± 2.9	35.1 ± 9.0	-
收缩压 (mmHg)	135 (125-148)	125 (116-138)	<0.0001
舒张压 (mmHg)	81 (77-90)	80 (73-90)	0.051
MAP (mmHg)	100 (93-109)	94 (87-103)	0.001
高血压, n (%)	51 (62.2)	118 (36.0)	<0.0001
尿蛋白定量 (g/24h)	1.65 (0.72-3.68)	1.18 (0.78-1.93)	0.011
≥3.5g/24h, n (%)	22 (26.8)	35 (10.7)	<0.0001
≥1-3.5g/24h, n (%)	52.332 (39.0)	151 (46.0)	
<1g/24h, n (%)	28 (34.2)	142 (43.3)	

老年与非老年IgAN患者临床特征的比较

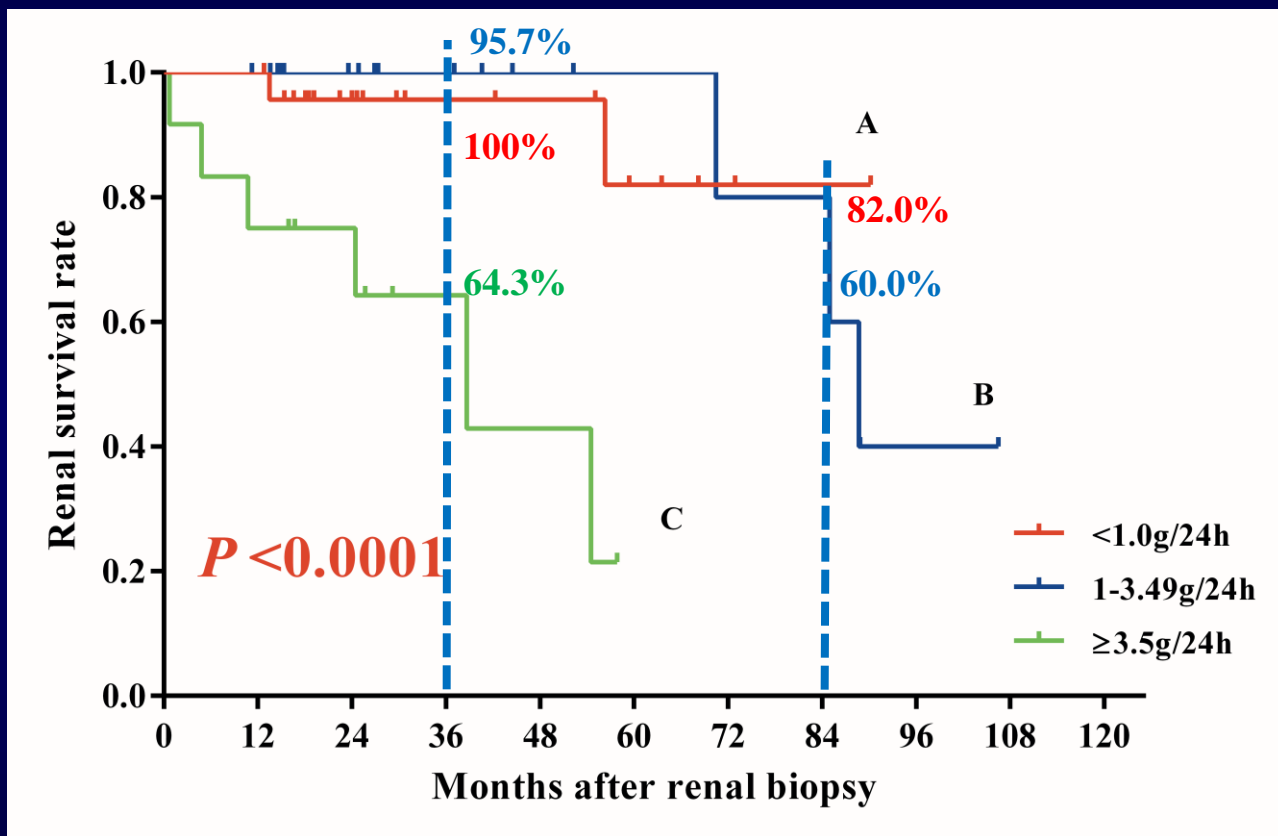
	≥65岁 (n=82)	18-64岁 (n=328)	P值
血白蛋白 (g/L)	35.5 (31.5-39.8)	37.3 (34.1-39.7)	0.105
Scr (mmol/L)	104.8 (84.0-162.0)	83.1 (63.6-113.2)	<0.0001
Scr >109.6mmol/L, n (%)	39 (47.6)	88 (26.8)	0.001
eGFR (ml/min/1.73m ²)	55.3 ± 24.8	89.2 ± 28.2	<0.0001
CKD 1 stage	8 (9.8)	174 (53.0)	
CKD 2 stage	23 (28.0)	85 (25.9)	
CKD ≥3 stage	51 (62.2)	69 (21.0)	<0.0001
血尿酸 (mmol/L)	425 (340-500)	383 (316-453)	0.012
总胆固醇 (mmol/L)	5.56 (4.65-6.84)	4.55 (3.94-5.33)	<0.0001
三酰甘油 (mmol/L)	1.61 (1.21-2.15)	1.51 (1.08-2.13)	0.541

老年与非老年IgAN患者肾组织病理特征的比较

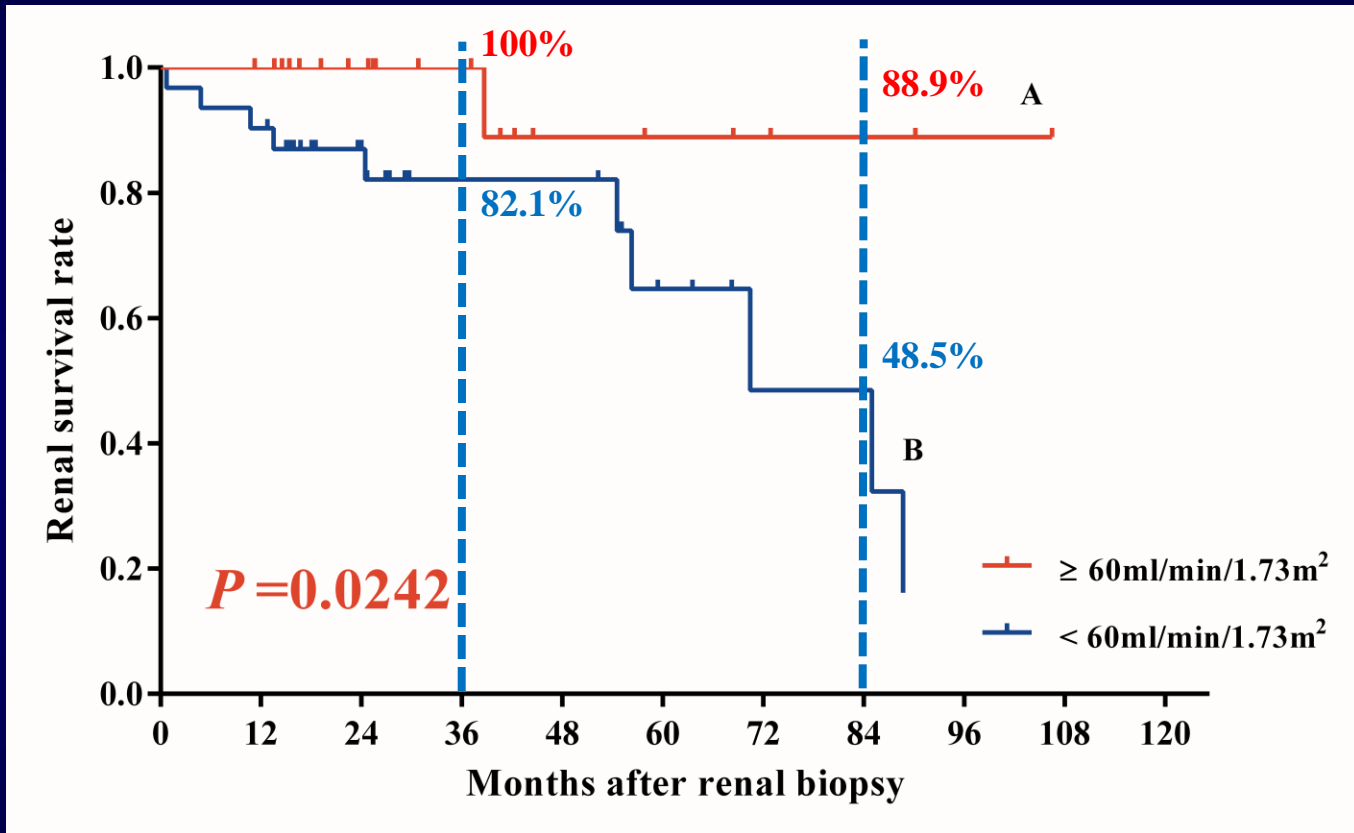
	≥65岁 (n=82)	18-64岁 (n=328)	P值
肾小球球性硬化比例 (%)	19.8 ± 18.3	12.5 ± 11.0	0.001
系膜增生 (M1), n (%)	35 (42.7)	154 (47.0)	0.488
毛细血管内增生 (E1), n (%)	17 (20.7)	47 (14.3)	0.153
节段肾小球硬化 (S1), n (%)	59 (72.0)	278 (28.0)	<0.0001
TAIF (T1 or T2), n (%)	32 (39.0)	81 (24.7)	0.009
袢坏死 (N1), n (%)	14 (17.1)	58 (17.7)	0.897
新月体 (C1), n (%)	31 (37.8)	125 (38.1)	0.959
血管硬化, n (%)	67 (81.7)	96 (29.3)	<0.0001

TAIF 肾小管萎缩/间质纤维化

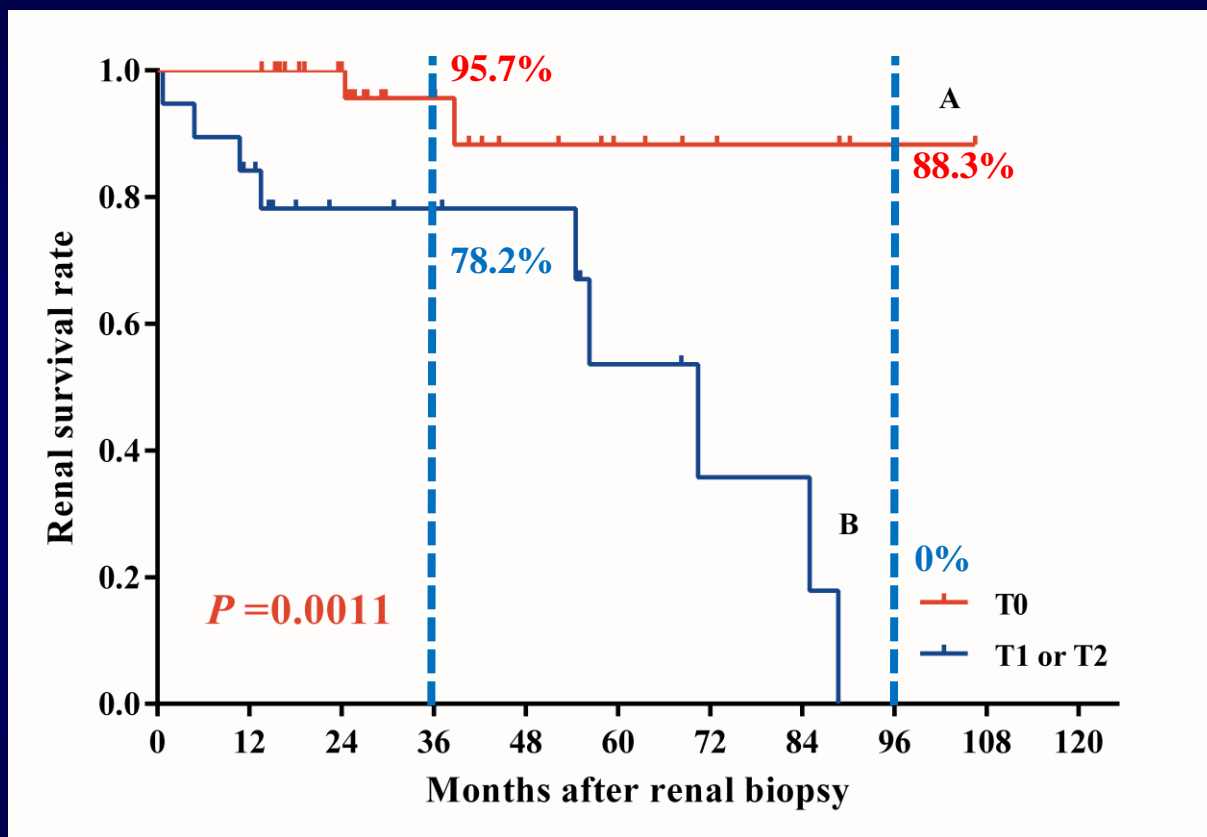
尿蛋白定量对肾脏累积生存率的影响



eGFR对肾脏累积生存率的影响



肾小管间质病变对肾脏累积生存率的影响



The Oxford Classification and Validation studies

The Oxford Classification of IgA Nephropathy

original article

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see commentary on page 477

The Oxford classification of IgA nephropathy: rationale, clinicopathological correlations, and classification

A Working Group of the International IgA Nephropathy Network and the Renal Pathology Society: Daniel C. Cattran^{1,†}, Rosanna Coppo^{2,†}, H. Terence Cook^{3,†}, John Feehally^{4,†}, Ian S.D. Roberts^{5,†}, Stéphan Troyanov^{6,†}, Charles E. Alpers⁷, Alessandro Amore², Jonathan Barratt⁴, Francois Berthoux⁸, Stephen Bonsib⁹, Jan A. Bruijn¹⁰, Vivette D'Agati¹¹, Giuseppe D'Amico¹², Francesco Emma¹⁴, Franco Ferrario¹⁵, Fernando C. Fervenza¹⁶, Colin C. Geddes¹⁹, Hermann-Josef Groene²⁰, Mark Haas²¹, Ar Ronald J. Hogg²⁴, Stephen I. Hsu²⁵, J. Charles Jennette²⁶, Ker Tetsuya Kawamura²⁹, Fernand M. Lai³⁰, Chi Bon Leung³¹, Lei Bruce Mackinnon¹⁹, Sergio Mezzano³³, F. Paolo Schena³⁴, Ya Haiyan Wang³⁷, Jan J. Weening³⁸, Nori Yoshikawa³⁹ and Hong Zhang^{37,*}

original article

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see commentary on page 477

The Oxford classification of IgA nephropathy: pathology definitions, correlations, and reproducibility

A Working Group of the International IgA Nephropathy Network and the Renal Pathology Society: Ian S.D. Roberts¹, H. Terence Cook², Stéphan Troyanov³, Charles E. Alpers⁴, Alessandro Amore⁵, Jonathan Barratt⁶, Francois Berthoux⁷, Stephen Bonsib⁸, Jan A. Bruijn⁹, Daniel C. Cattran¹⁰, Rosanna Coppo⁵, Vivette D'Agati¹¹, Giuseppe D'Amico¹², Steven Emancipator¹³, Francesco Emma¹⁴, John Feehally⁶, Franco Ferrario¹⁵, Fernando C. Fervenza¹⁶, Sandrine Florquin¹⁷, Agnes Fogo¹⁸, Colin C. Geddes¹⁹, Hermann-Josef Groene²⁰, Mark Haas²¹, Andrew M. Herzenberg²², Prue A. Hill²³, Ronald J. Hogg²⁴, Stephen I. Hsu²⁵, J. Charles Jennette²⁶, Kensuke Joh²⁷, Bruce A. Julian²⁸, Tetsuya Kawamura²⁹, Fernand M. Lai³⁰, Lei-Shi Li³¹, Philip K.T. Li³², Zhi-Hong Liu³¹, Bruce Mackinnon¹⁹, Sergio Mezzano³³, F. Paolo Schena³⁴, Yasuhiko Tomino³⁵, Patrick D. Walker³⁶, Haiyan Wang³⁷, Jan J. Weening³⁸, Nori Yoshikawa³⁹ and Hong Zhang^{37,*}

The Oxford Classification of IgA Nephropathy

Feature	Classification
<i>Mesangial hypercellularity</i>	
Present in $\leq 50\%$ of the glomeruli	M0
Present in $> 50\%$ of the glomeruli	M1
<i>Segmental glomerulosclerosis</i>	
Absent	S0
Present	S1
<i>Endocapillary hypercellularity</i>	
Absent	E0
Present	E1
<i>Tubular atrophy/interstitial fibrosis</i>	
0–25% of cortical area	T0
26–50% of cortical area	T1
$> 50\%$ of cortical area	T2

Permission obtained from Nature Publishing Group Ltd © Cattran, D. C. *et al.* The Oxford classification of IgA nephropathy: rationale, clinicopathological correlations, and classification. *Kidney Int.* 76, 534–545 (2009).

A Multicenter Application and Evaluation of the Oxford Classification of IgA Nephropathy in Adult Chinese Patients

Cai-Hong Zeng, PhD,^{1} Weibo Le, MD,^{1*} Zhaohui Ni, PhD,² Minfang Zhang, PhD,² Lining Miao, PhD,³ Ping Luo, PhD,³ Rong Wang, MD,⁴ Zhimei Lv, PhD,⁴ Jianghua Chen, MD,⁵ Jiong Tian, PhD,⁵ Nan Chen, PhD,⁶ Xiaoxia Pan, MD,⁶ Ping Fu, PhD,⁷ Zhangxue Hu, MD,⁷ Lining Wang, MD,⁸ Qiuling Fan, PhD,⁸ Hongguang Zheng, PhD,⁹ Dewei Zhang, MD,⁹ Yaping Wang, MD,¹⁰ Yanhong Huo, MD,¹⁰ Hongli Lin, MD,¹¹ Shuni Chen, MS,¹¹ Shiren Sun, PhD,¹² Yanxia Wang, MD,¹² Zhangsuo Liu, PhD,¹³ Dong Liu, MD,¹³ Lu Ma, MD,¹⁴ Tao Pan, MD,¹⁴ Aiping Zhang, MD,¹⁵ Xiaoyu Jiang, MD,¹⁵ Changying Xing, PhD,¹⁶ Bing Sun, PhD,¹⁶ Qiaoling Zhou, MD,¹⁷ Wenbing Tang, MD,¹⁷ Fuyou Liu, MS,¹⁸ Yinghong Liu, PhD,¹⁸ Shaoshan Liang, MD,¹ Feng Xu, MD,¹ Qian Huang, MD,¹ Hongbing Shen, PhD,¹⁹ Jianming Wang, PhD,¹⁹ Yu Shyr, PhD,²⁰ Sharon Phillips, MS,²⁰ Stéphan Trojanov, MD,²¹ Agnes Fogo, MD,²² and Zhi-Hong Liu, MD¹*

Background: The Oxford classification of immunoglobulin A (IgA) nephropathy (IgAN) provides a histopathologic grading system that is associated with kidney disease outcomes independent of clinical features. We evaluated the Oxford IgAN classification in a large cohort of patients from China.

Study Design: Retrospective study.

Setting & Participants: 1,026 adults with IgAN from 18 referral centers in China. Inclusion criteria and

Validation study

Patients:

1,026 adults with IgAN
from 18 renal centers in China.

Inclusion Criteria:

eGFR >30 mL/min/1.73 m²

Proteinuria >0.5 g/24 h

Biopsy specimen with 10 or more total glomeruli

Followed up for 12 or more months

Clinical Characteristics

	Chinese Cohort (N = 1,026)	Oxford Cohort (N = 265)
<u>At Time of Biopsy</u>		
Age (y)	34 (18-73)	30 (4-73)
Female sex	50	28
BMI (kg/m ²)	24 ± 4	25 ± 6
MAP (mm Hg)	98 ± 16	98 ± 17
Hypertensive before biopsy	33	31
eGFR (mL/min/1.73 m ²)	85 ± 32	83 ± 36
CKD stage ^a		
1	41	36
2	35	38
3	24	26
Proteinuria (g/d)	1.3 (0.5-18.4)	1.7 (0.5-18.5)
Previous macroscopic hematuria	27	34
Previous immunosuppression	1.5	14
Previous use of fish oil	0	6
Treated with RAS blockade	10	20

	<u>During Follow-up</u>	
Duration of follow-up (mo)	53 (7.6-169)	69 (12-268)
50% Decline in eGFR	15.5	22
End-stage renal disease	8.8	3
MAP (mm Hg)	94 ± 9	95 ± 10
ΔeGFR (mL/min/1.73 m ² per y)	-1.5 ± 10.4	-3.5 ± 8.4
Proteinuria (g/d)	0.7 (0.1-11.1)	1.1 (0.1-9.3)
Immunosuppression	31	29
Prednisone >6 mo	27	29
Others	22	9
Treated with RAS blockade >6 mo	89	74
Known tonsillectomy	3.7	NA

Reproducibility of pathological grading (intraclass correlation coefficients, ICCs)

	Oxford Study	Nanjing study
M (Mesangial hypercellularity)	0.64	0.80
E (Endocapillary hypercellularity)	0.57	0.66
S (Segmental sclerosis)	0.46	0.75
T (Interstitial fibrosis)	0.79	0.94

Associations between pathological lesions and renal survival (n=1026)

Pathological grading	Oxford Study	Nanjing study
M (Mesangial-cell hypercellularity)	☆☆	☆☆
E (Endocapillary hypercellularity)	—	-
S (Segmental sclerosis or adhesion)	☆☆	-
T (Interstitial fibrosis)	☆☆☆☆	☆☆☆☆
C (Crescents)	-	-
N (Necrosis)	Not available	-

A Multi-center Validation of the Oxford Classification of IgAN in Chinese patients

- We confirmed the associations of mesangial hypercellularity (M) and tubular atrophy/interstitial fibrosis (T) with kidney disease outcomes.
- We did not find the associations between the lesion of S,E, C, and N and disease outcomes.

Validation of the Oxford classification of IgA nephropathy for pediatric patients from China

Weibo Le¹, Cai-Hong Zeng¹, Zhangsuo Liu², Dong Liu², Qing Yang³, Rui-Xia Lin³, Zheng-Kun Xia⁴, Zhong-Min Fan⁴, Guanghua Zhu⁵, Ying Wu⁵, Hong Xu⁶, Yihui Zhai⁶, Ying Ding⁷, Xiaoqing Yang⁷, Shaoshan Liang¹, Hao Chen¹, Feng Xu¹, Qian Huang¹, Hongbing Shen⁸, Jianming Wang⁸, Agnes B Fogo^{9*} and Zhi-Hong Liu^{1*}

Abstract

Background: The Oxford classification of IgA nephropathy (IgAN) provides a useful tool for prediction of renal prognosis. However, the application of this classification in children with IgAN needs validation in different patient populations.

Methods: A total of 218 children with IgAN from 7 renal centers in China were enrolled. The inclusion criteria was similar to the original Oxford study.

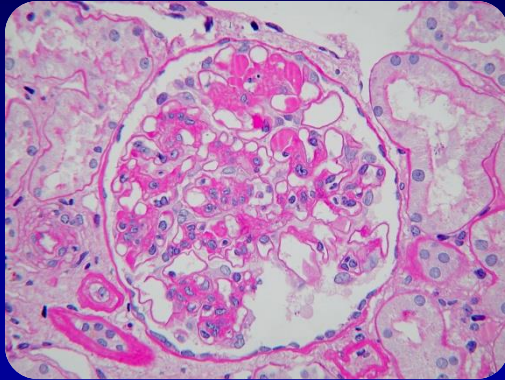
Results: There were 98 patients (45%) with mesangial proliferation (M1), 51 patients (23%) with endocapillary proliferation (E1), 136 patients (62%) with segmental sclerosis/adhesion lesion (S1), 13 patients (6%) with moderate tubulointerstitial fibrosis (T1 26-50% of cortex scarred), and only 2 patients (1%) with severe tubulointerstitial fibrosis (T2, >50% of cortex scarred). During a median follow-up duration of 56 months, 24 children (12.4%) developed ESRD or 50% decline in renal function. In univariate COX analysis, we found that tubular atrophy/interstitial fibrosis (HR 4.3, 95%CI 1.8-10.5, $P < 0.001$) and segmental glomerulosclerosis (HR 9.2 1.2-68.6, $P = 0.03$) were significant predictors of renal outcome. However, mesangial hypercellularity, endocapillary proliferation, crescents, and necrosis were not associated with renal prognosis. In the multivariate COX regression model, none of these pathologic lesions were shown to be independent risk factors of unfavorable renal outcome except for tubular atrophy/interstitial fibrosis (HR 2.9, 95%CI 1.0-7.9 $P = 0.04$).

Conclusions: We confirmed tubular atrophy/interstitial fibrosis was the only feature independently associated with renal outcomes in Chinese children with IgAN.

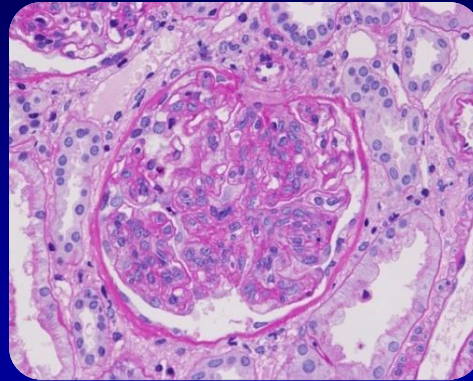
Keywords: Glomerulonephritis, IgA nephropathy, Oxford classification, Children, Pediatrics

- A recent review of 13 Oxford classification replication studies confirmed the independent prognostic value of tubular atrophy and interstitial fibrosis in 10 studies, mesangial hypercellularity in 4 studies, and segmental sclerosis in 4 studies.

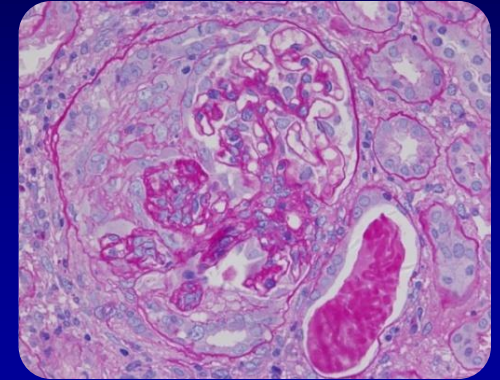
Pathological Features



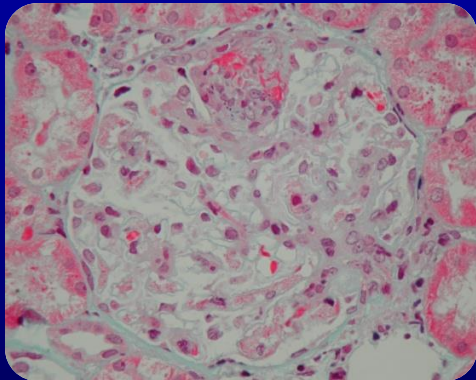
Mesangial cellularity score (M)



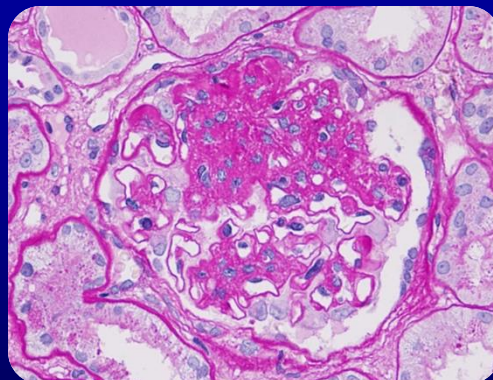
Endocapillary proliferation (E)



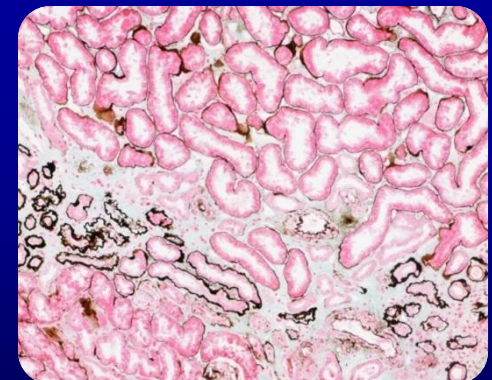
Crescents (C)



Necrosis (N)



Segmental glomerulosclerosis (S)



Tubular atrophy/
interstitial fibrosis (T)

J Nephrol

DOI 10.1007/s40620-014-0165-x

ORIGINAL ARTICLE

Reversal of active glomerular lesions after immunosuppressive therapy in patients with IgA nephropathy: a repeat-biopsy based observation

**Xia-Hong Shen · Shao-Shan Liang · Hui-Mei Chen ·
Wei-Bo Le · Song Jiang · Cai-Hong Zeng · Min-Lin Zhou ·
Hai-Tao Zhang · Zhi-Hong Liu**

Repeat Renal Biopsy

- **Patients: 60 cases**
- **Biopsy interval: >6 months**
- **Follow up: >12 months; Median time: 88m**
- **Treatment: Predinison (16); CTX (5); MMF (39)**

两次肾活检时的临床表现

	第一次肾活检	第二次肾活检	P值
MAP (mmHg)	95 ± 11	92 ± 12	0.276
尿蛋白定量 (g/24h)	1.98 (1.36 - 3.06)	0.80 (0.34 - 1.63)	< 0.001
尿沉渣红细胞 (万/mL)	195 (83 - 689)	23 (4 - 105)	< 0.001
SCr (mg/dL)	90.17 (72.49 - 127.30)	82.21 (67.18 - 114.26)	0.067
eGFR (mL/min/1.73m ²)	75.7 ± 29.3	88.9 ± 44.3	0.122
总胆固醇 (mmol/L)	5.75 ± 2.43	5.67 ± 2.53	0.794
甘油三酯 (mmol/L)	2.08 ± 1.28	1.87 ± 0.97	0.539

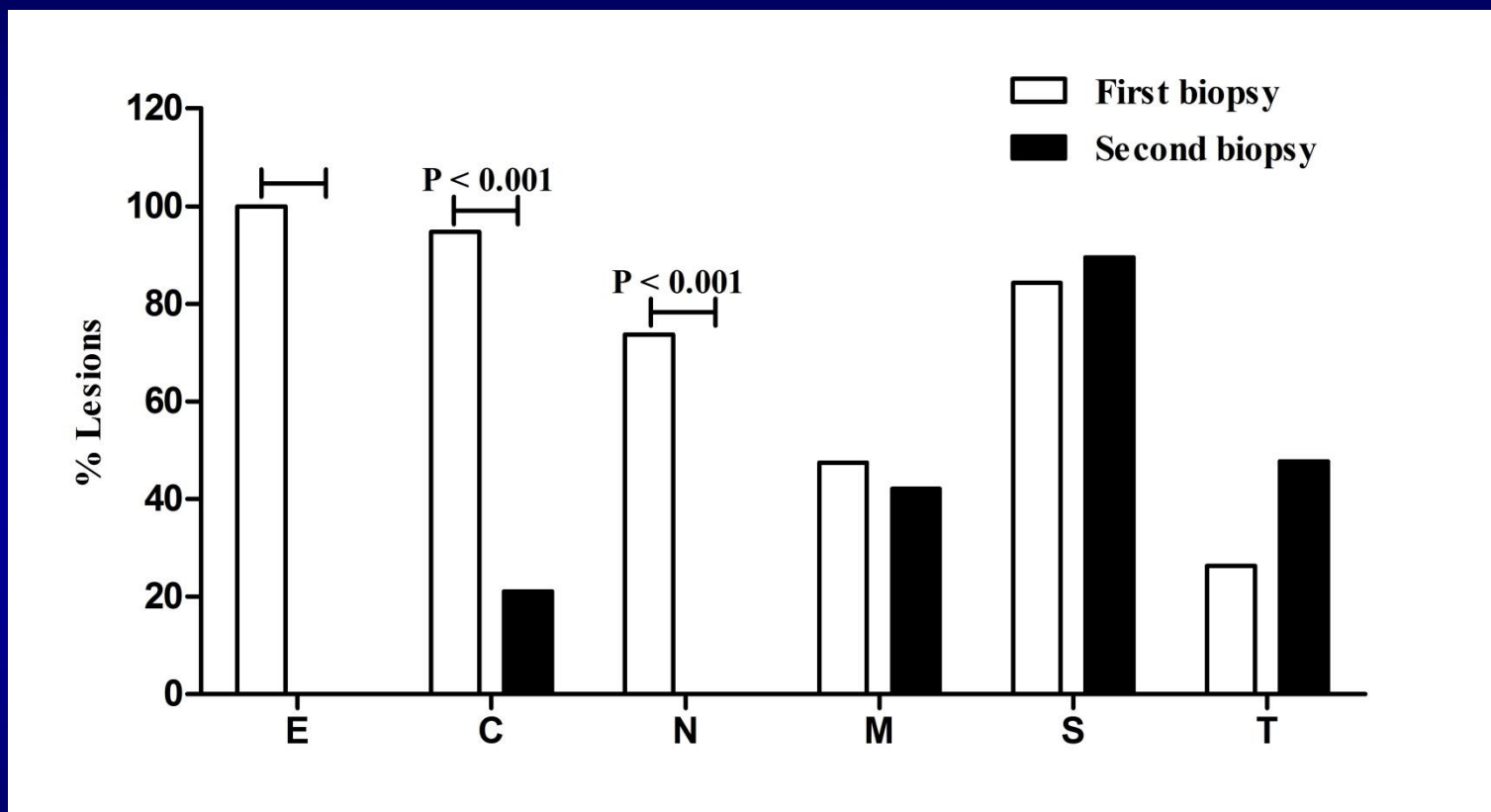
免疫抑制剂治疗后伴E病变患者的临床表现变化

	E病变好转 (n = 19)		E病变持续 (n = 3)	
	第一次肾活检	第二次肾活检	第一次肾活检	第二次肾活检
尿蛋白定量 (g/24h)	2.54 (0.85 - 7.75)	0.80 (0.18 - 4.92)^a	1.32 (0.65 - 2.43)	1.64 (1.38 - 2.65)
尿沉渣红细胞 (万/mL)	395 (9 - 15000)	40 (1 - 3000)^a	80 (58 - 520)	450 (44 - 450)
SCr (mg/dL)	1.18 (0.53 - 4.58)	0.81 (0.43 - 1.87)^b	1.23 (0.79 - 1.39)	1.30 (0.61 - 2.50)
eGFR (mL/min/1.73m ²)	72.6 ± 36.2	84.0 ± 57.2	69.5 ± 21.6	67.7 ± 47.2

a: E病变好转患者的第一次和第二次肾活检比较P < 0.01;

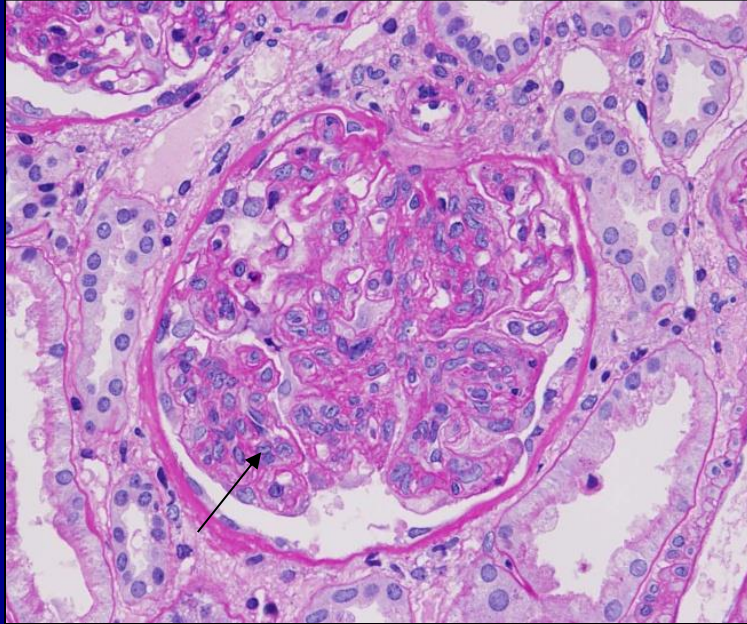
b: E病变好转患者的第一次和第二次肾活检比较P < 0.05。

免疫抑制剂治疗后伴E病变患者肾组织病变的变化

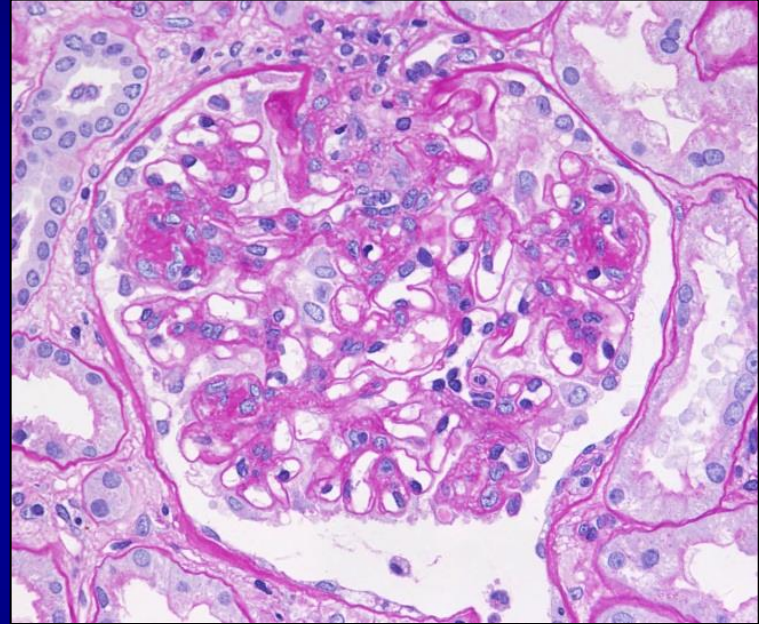


第二次肾活检E病变完全消失，同时伴C和N病变患者比例明显下降

免疫抑制治疗可以改善肾小球E病变



第一次肾活检



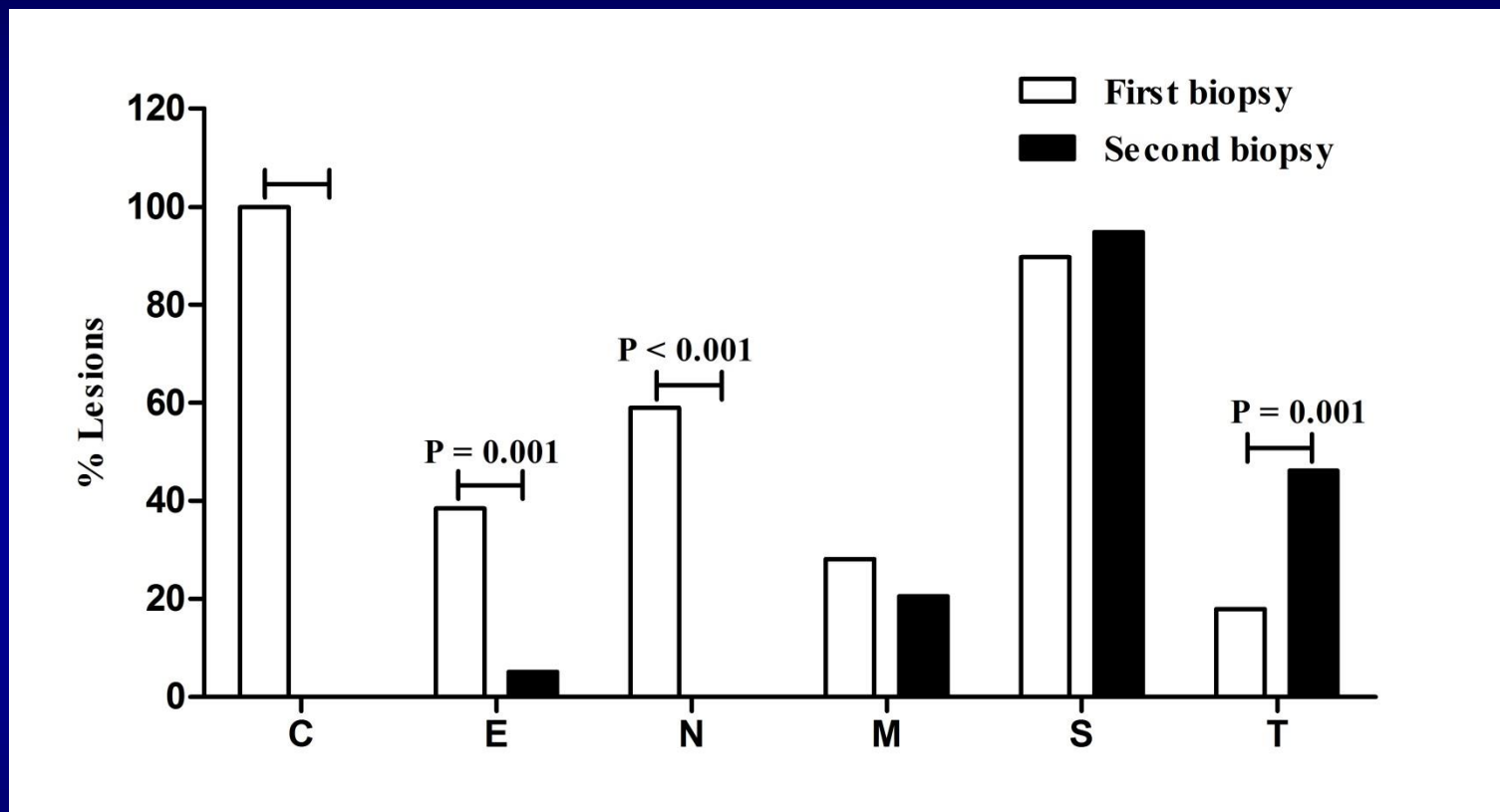
第二次肾活检

免疫抑制剂治疗后伴C病变患者临床表现变化

	C病变好转 (n = 39)		C病变持续 (n = 12)	
	第一次肾活检	第二次肾活检	第一次肾活检	第二次肾活检
尿蛋白定量 (g/24h)	1.92 (1.44 - 2.77)	0.61(0.31 - 1.07)^a	2.73 (1.09 - 3.42)	1.62 (0.46 - 3.90)
尿沉渣红细胞 (万/mL)	375 (100 - 750)	26 (4 - 80)^a	175 (99 - 955)	250 (55 - 1050)
SCr (mg/dL)	0.99 (0.82 - 1.35)	0.93 (0.76 - 1.13)	1.15 (0.82 - 2.14)	0.96 (0.63 - 2.04)
eGFR (mL/min/1.73m ²)	80.5 ± 25.7	89.6 ± 29.5	65.1 ± 37.0	83.2 ± 79.5

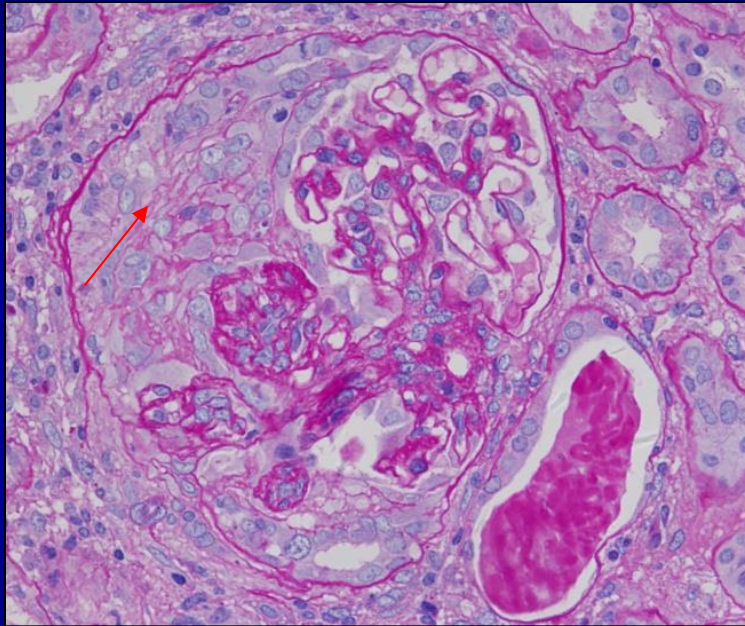
a: C病变好转患者的第一次和第二次肾活检比较P < 0.001。

免疫抑制剂治疗后伴C病变患者肾组织病变的变化

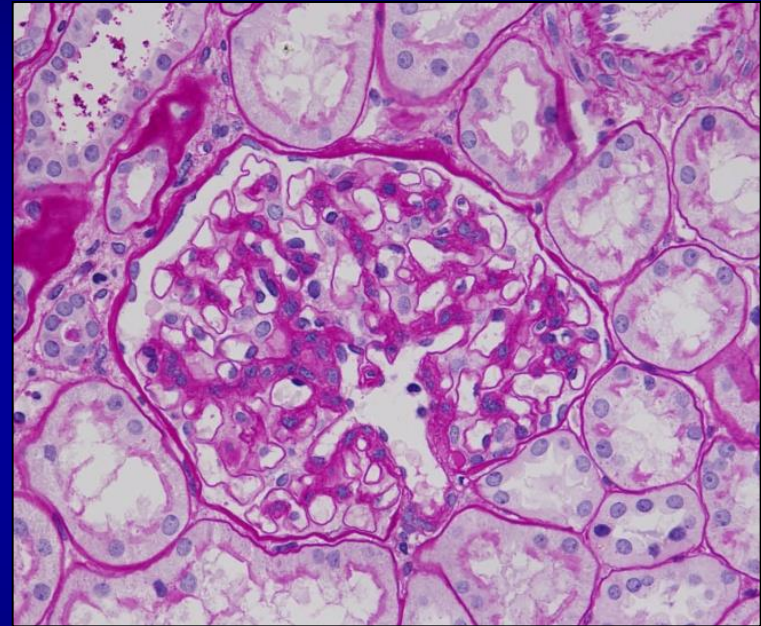


第二次肾活检时C病变消失，伴E和N病变比例明显下降

免疫抑制治疗可以改善肾小球C病变



第一次肾活检

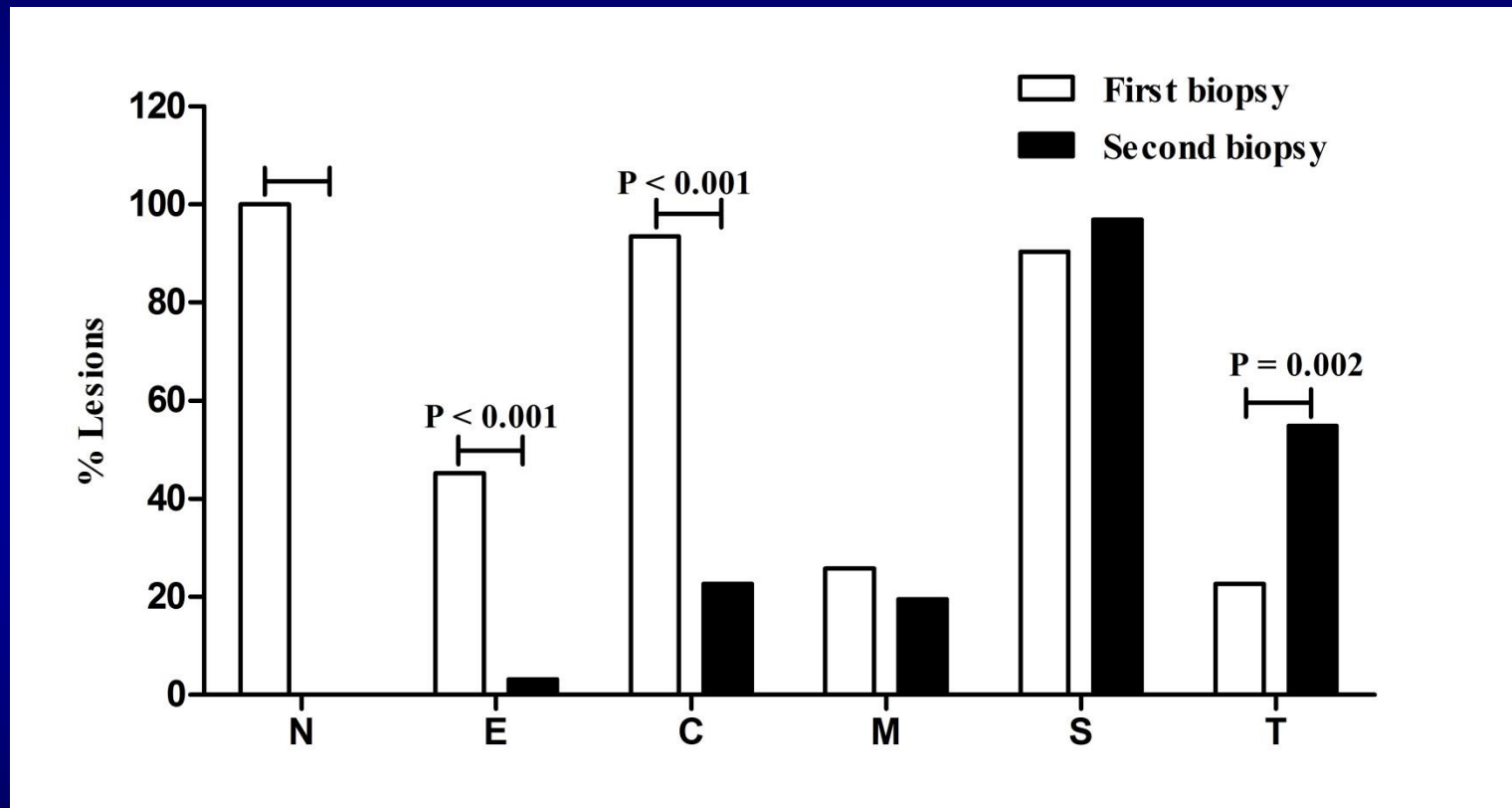


第二次肾活检

免疫抑制剂治疗后伴N病变患者临床表现变化

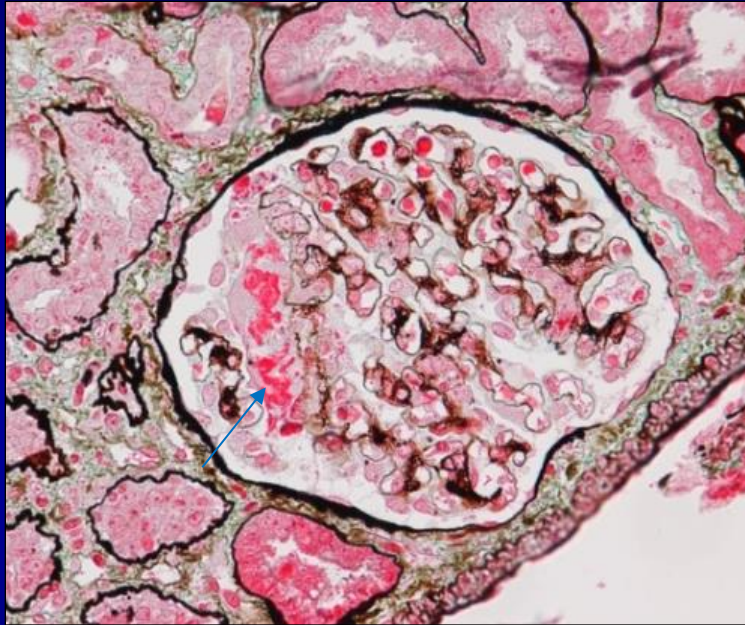
	N病变好转 (n = 31)		P值
	第一次肾活检	第二次肾活检	
尿蛋白定量 (g/24h)	1.76 (1.36 - 2.76)	0.55 (0.32 - 1.04)	< 0.001
尿沉渣红细胞 (万/mL)	375 (120 - 1000)	37 (5 - 80)	< 0.001
SCr (mg/dL)	0.94 (0.79 - 1.44)	0.82 (0.76 - 1.03)	0.102
eGFR (mL/min/1.73m ²)	78.8 ± 31.3	86.4 ± 49.2	0.098

免疫抑制剂治疗后伴N病变患者肾组织病变的变化

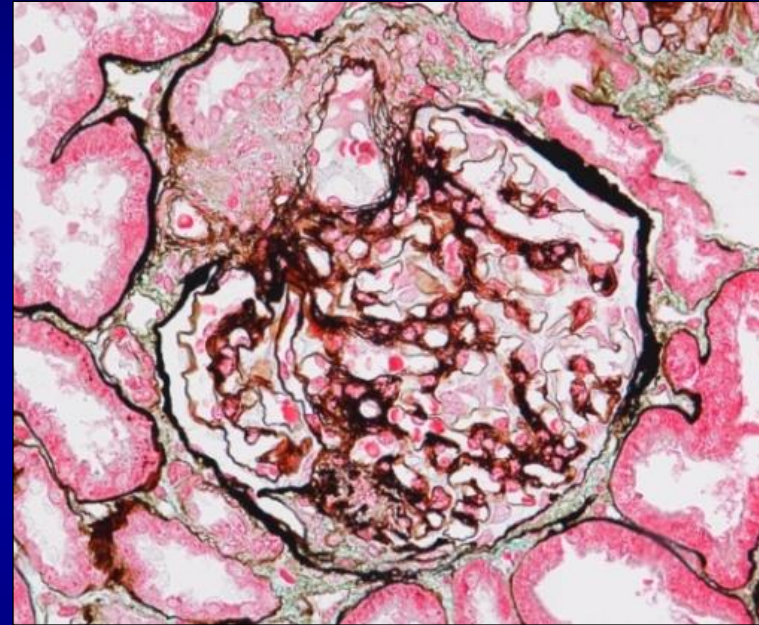


第二次肾活检时N病变好转，伴E和C病变患者比例明显下降

免疫抑制治疗可以改善肾小球C病变



第一次肾活检



第二次肾活检

- 免疫抑制剂治疗，在降低IgAN患者蛋白尿和血尿的同时可以明显改善肾小球的急性增殖性病变，包括：毛细血管内增殖性病变（E），新月体（C）和袢坏死（N）病变。基于肾组织病变特点，选择治疗方案应该是IgAN治疗中的一个方向。

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Thank you !

