

Доклад Столяревич Е.С.

Конференция РДО в

IgA -нефропатия

современные представления

Столяревич Е.С.

Доклад Столяревич Е.С.

Конференция РДО в

Краснодаре

Краснодар 26-27 апреля 2019 г.

Management and treatment of glomerular diseases (part 1): conclusions from a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference



OPEN

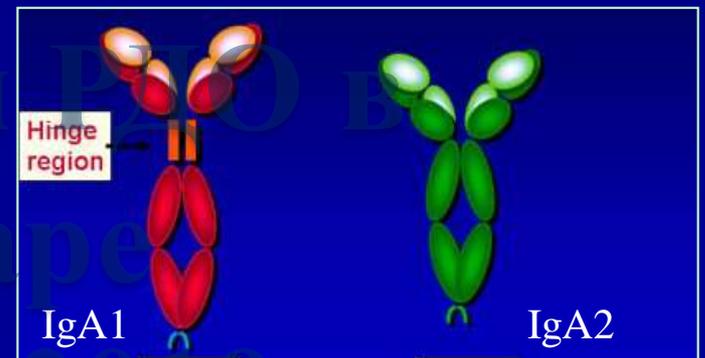
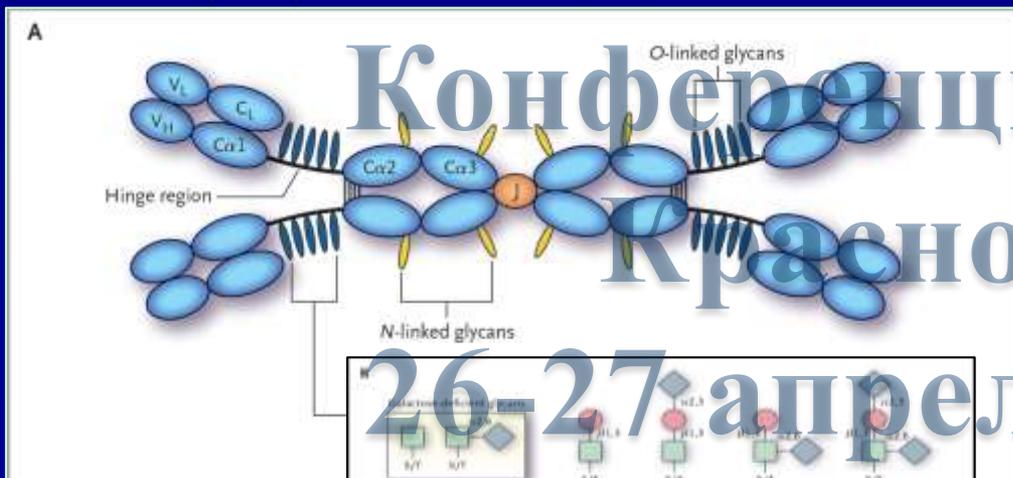
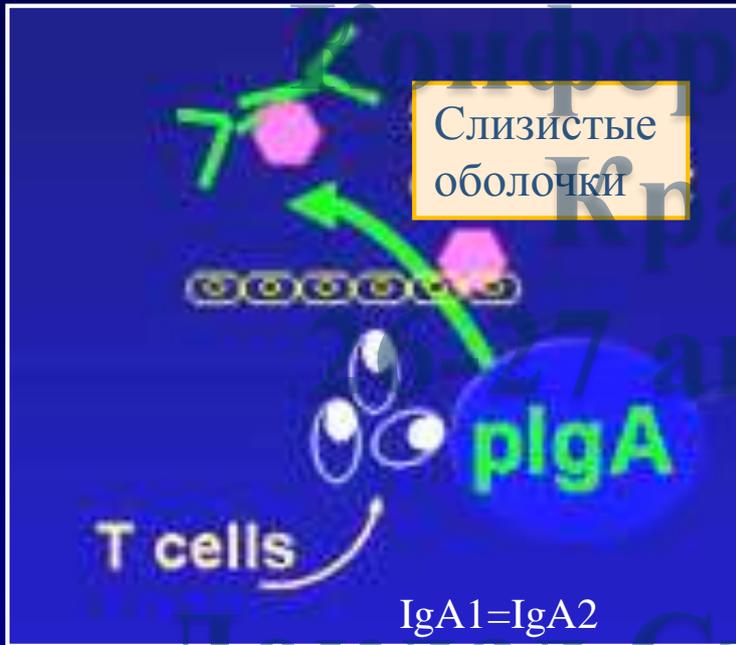
Jürgen Floege¹, Sean J. Barbour^{2,3,4}, Daniel C. Cattran⁵, Jonathan J. Hogan⁶, Patrick H. Nachman⁷, Sydney C.W. Tang⁸, Jack F.M. Wetzels⁹, Michael Cheung¹⁰, David C. Wheeler¹¹, Wolfgang C. Winkelmayr¹² and Brad H. Rovin¹³; for Conference Participants¹⁴

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- Патогенез
- Биомаркеры и предикторы прогноза
- Лечение
- Исследования новых препаратов

Kidney International (2019)
95, 268–280

Молекулярная структура IgA.



Wyatt RJ, Julian BA. N Engl J Med 2013; 368:2402-2414.

Патогенез IgA-нефропатии



Доклад Столяревич Е.С.



Патогенез IgA-нефропатии



НОВЫЕ ВОЗМОЖНОСТИ НОВЫЕ ВОЗМОЖНОСТИ ДИАГНОСТИКИ IgA- диагностики IgA-нефропатии нефропатии

Краснодаре

26-27 апреля 2019 г.

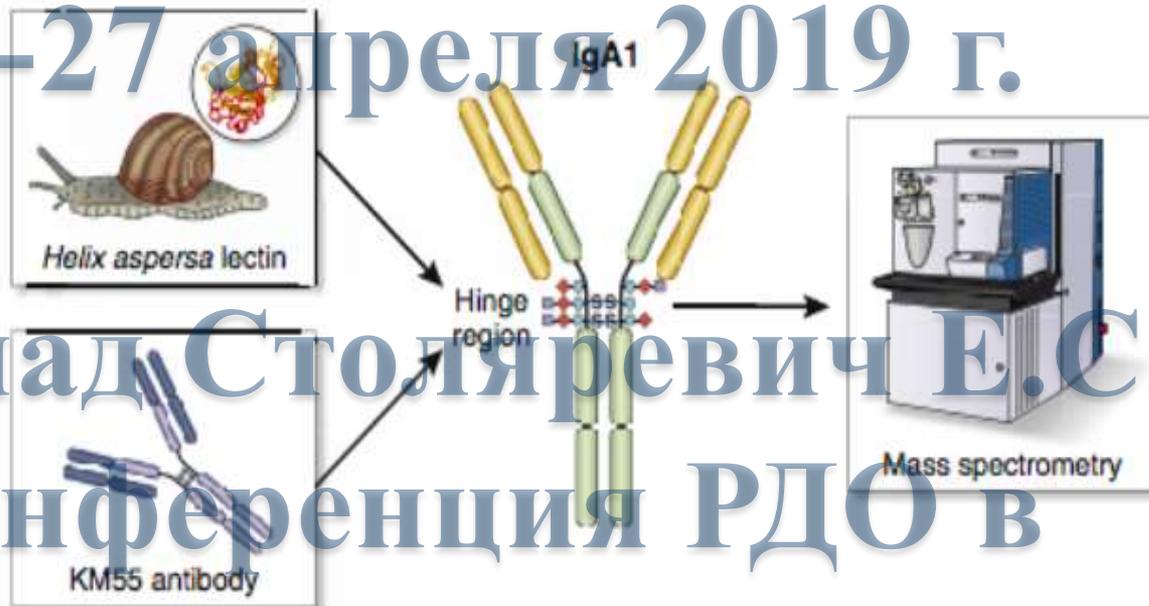


Figure 1 | Three different methods to detect undergalactosylation of the IgA1 hinge region.

26-27 апреля 2019 г.

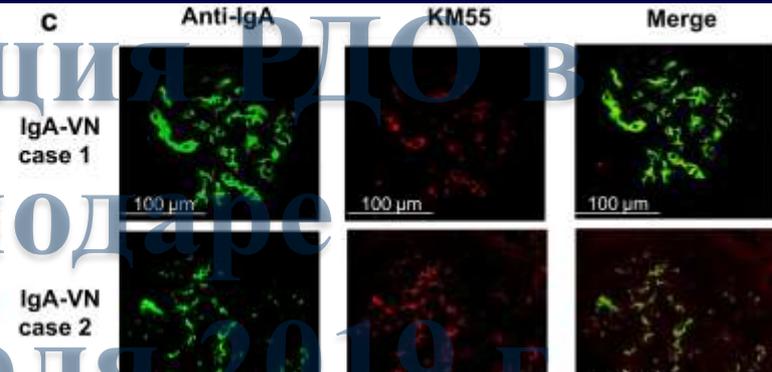
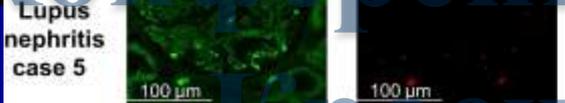
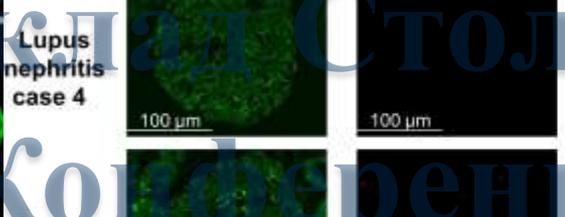
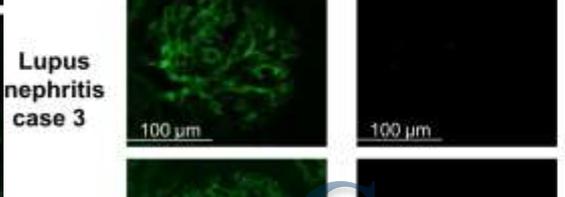
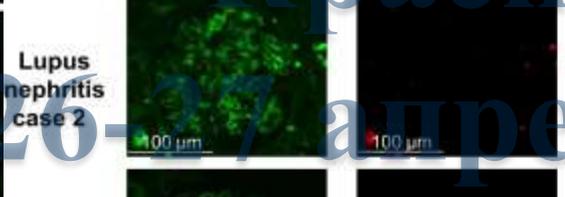
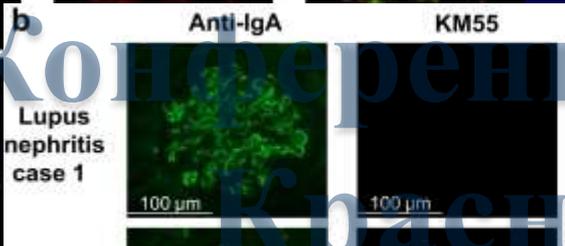
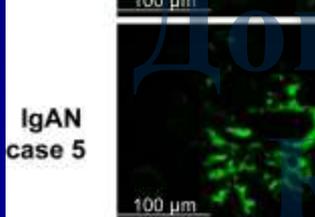
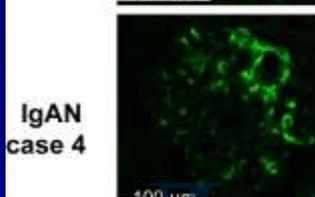
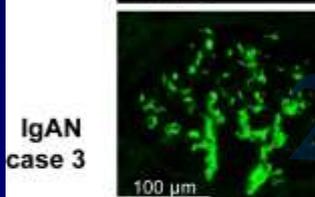
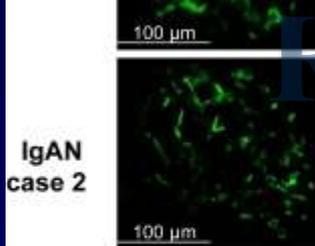


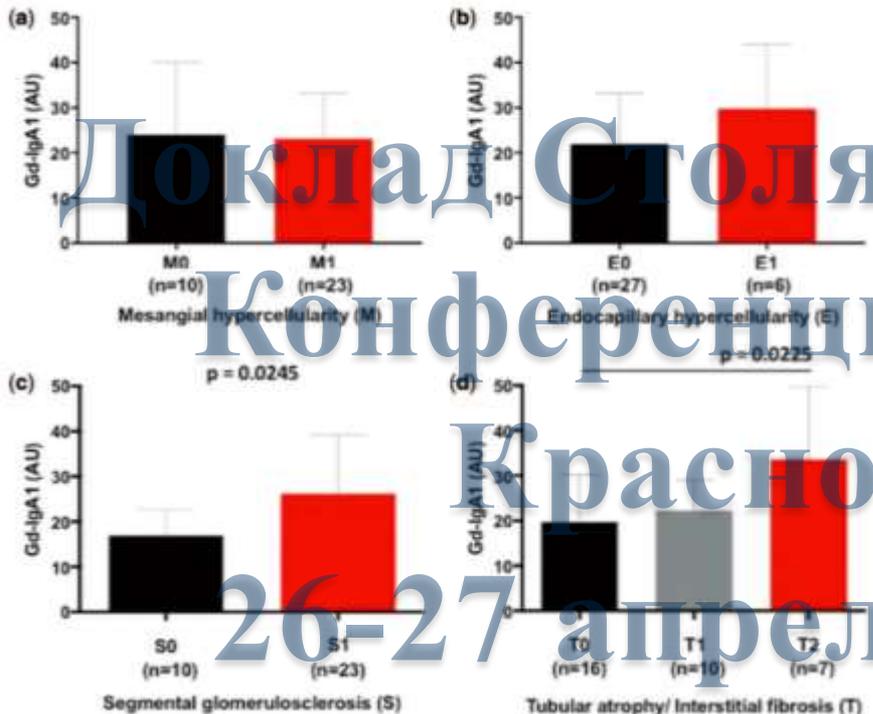
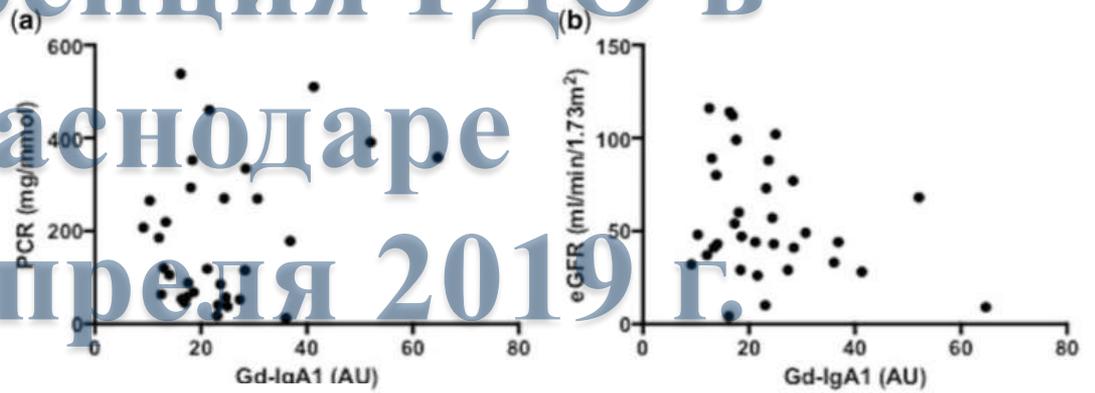
Table 1 | Biopsy samples

Diseases	Cases	Glomerular deposit	
		IgA (+)	Gd-IgA1 (+)
		Anti-IgA Ab	KM55
IgAN	48	48	48
IgA-VN	14	14	14
Lupus nephritis	8	7	0
Idiopathic MN	5	1	0
Secondary MN	4	3	0
HCV-RN	3	3	0
Hepatic glomerulosclerosis	1	1	0
ANCA-related nephropathy	2	0	0
Granulomatosis with polyangiitis	1	1	0
MPGN	1	1	0
MCNS	3	0	0
Non-IgA PGN	2	0	0
Nonspecific renal tubular atrophy	1	0	0
APSGN	1	0	0
Nephrosclerosis	1	0	0
Minor glomerular abnormalities	2	0	0
Total	97		

Ab, antibody; ANCA, antineutrophil cytoplasmic antibody; APSGN, acute post-streptococcal glomerulonephritis; Gd, galactose dependent; HCV-RN, hepatitis C



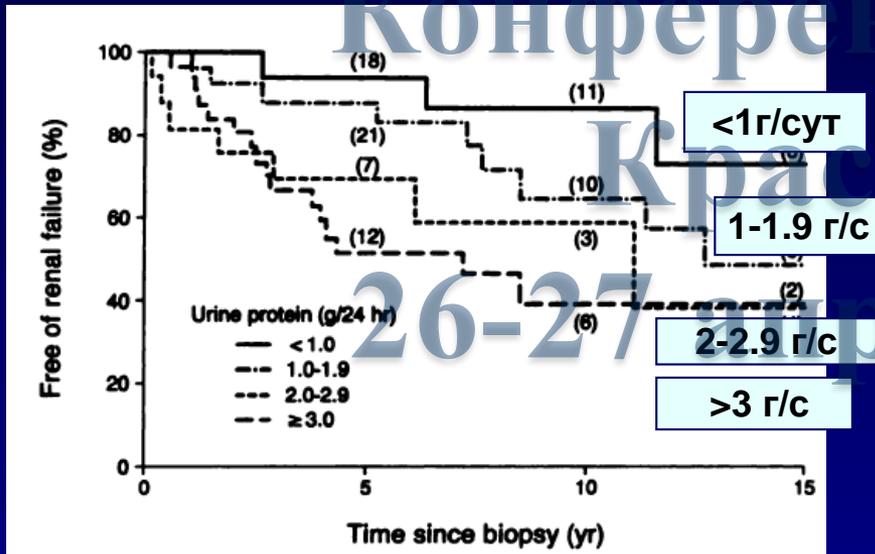
Доклад Столяревич Е.С. Конференция РДО в Краснодаре 26-27 апреля 2019 г.



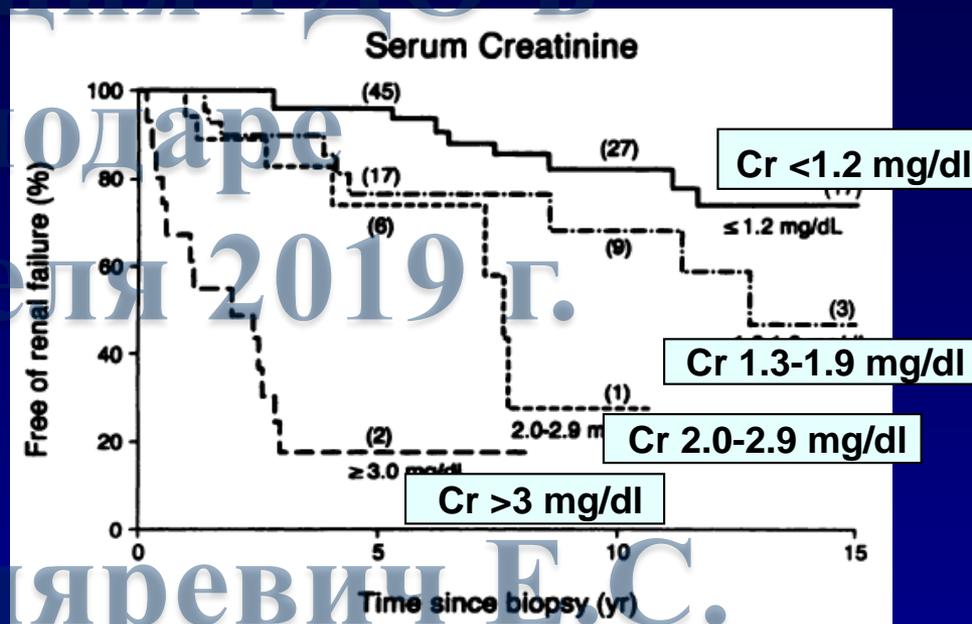
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Факторы риска прогрессирования IgA-нефропатии (клинические)

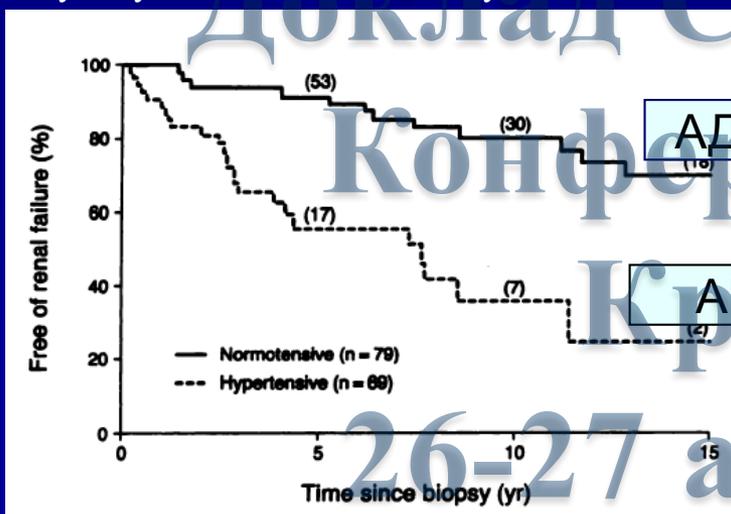
протеинурия



Уровень креатинина в дебюте



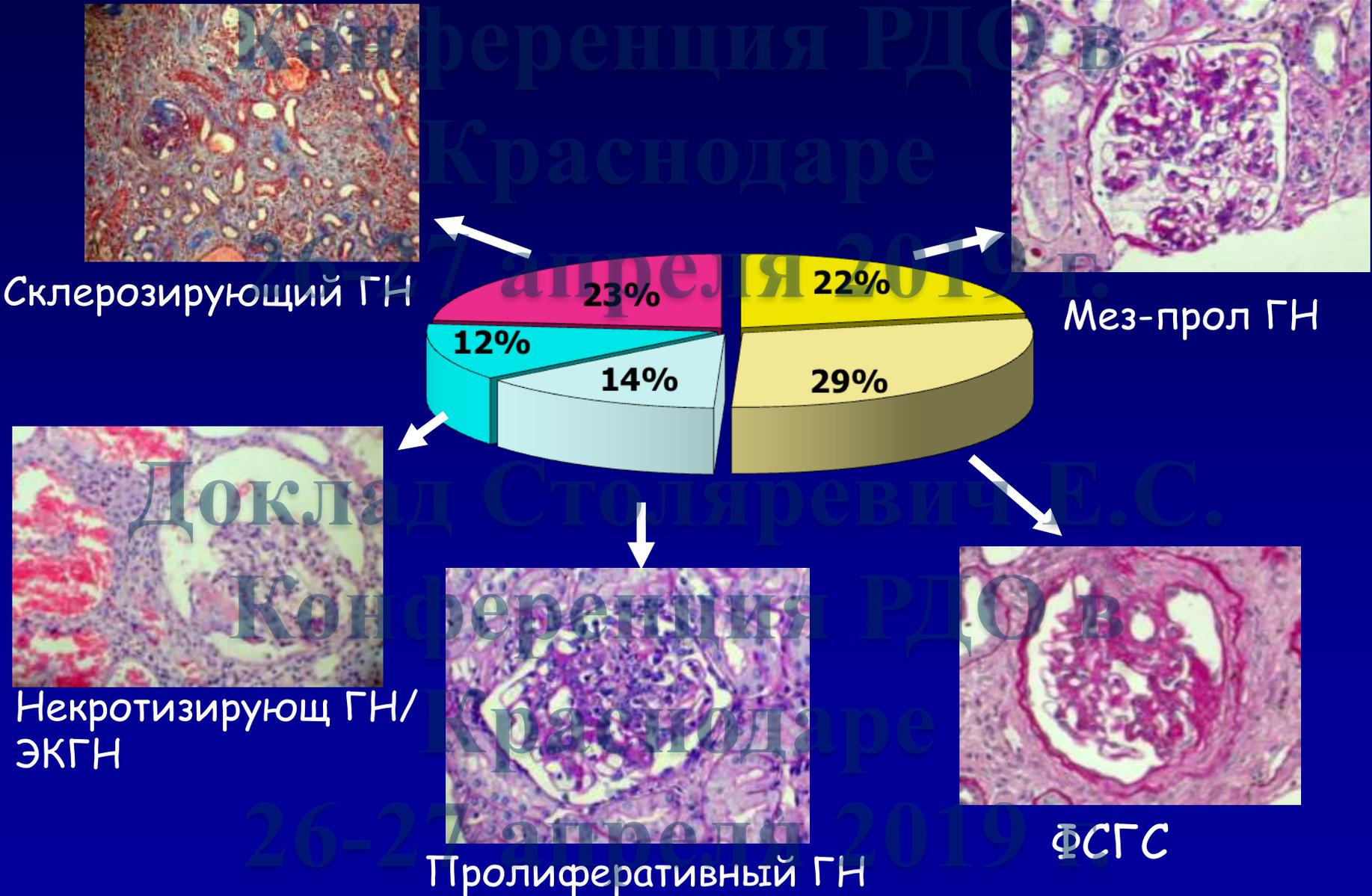
Артериальная гипертензия



Одни и те же клинические проявления могут быть следствием различных патологических процессов

Морфологическая картина при IgA-нефропатии

(Собственные данные по результатам 424 биопсий)



Морфологическая картина

Конференция РДО в

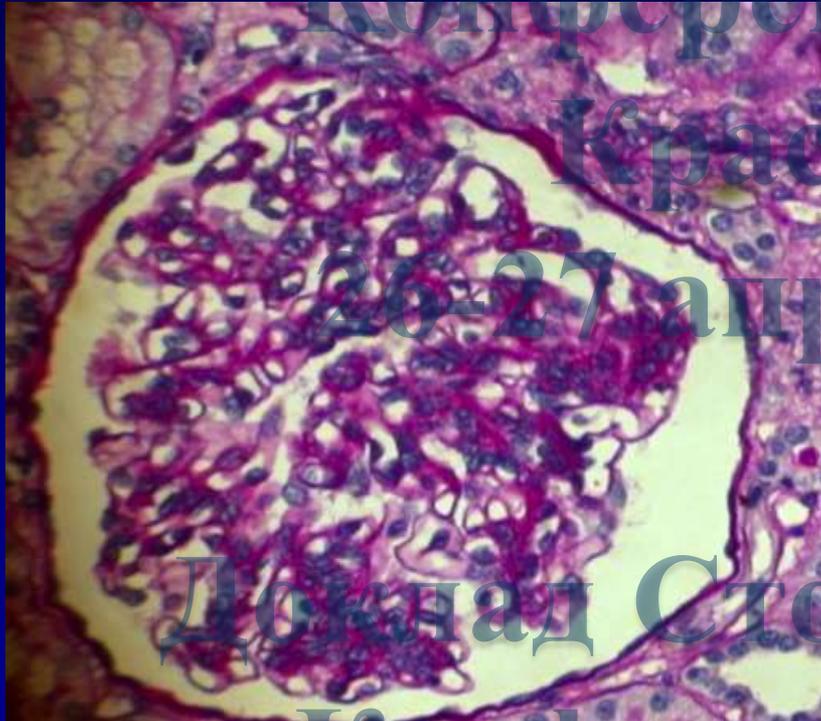
Краснодаре

26-27 апреля 2019 г.

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Конференция РДО в

Краснодаре



Обязательное условие – доминирование или кодоминирование иммунных комплексов, содержащих IgA в мезангии

Международный Консенсус по клиничко-морфологической классификации

IgA-нефропатии: Оксфордская Классификация

Сочетания MEST

2019 г.

Мезангиальная гиперклеточность



M0/M1

Эндокапиллярная гиперклеточность



E0/E1

Сегментарный гломерулосклероз



S0/S1

Тубулярная атрофия/фиброз интерстиция



T0/T1/T2

Скорость снижения почечной функции в зависимости от морфологической картины

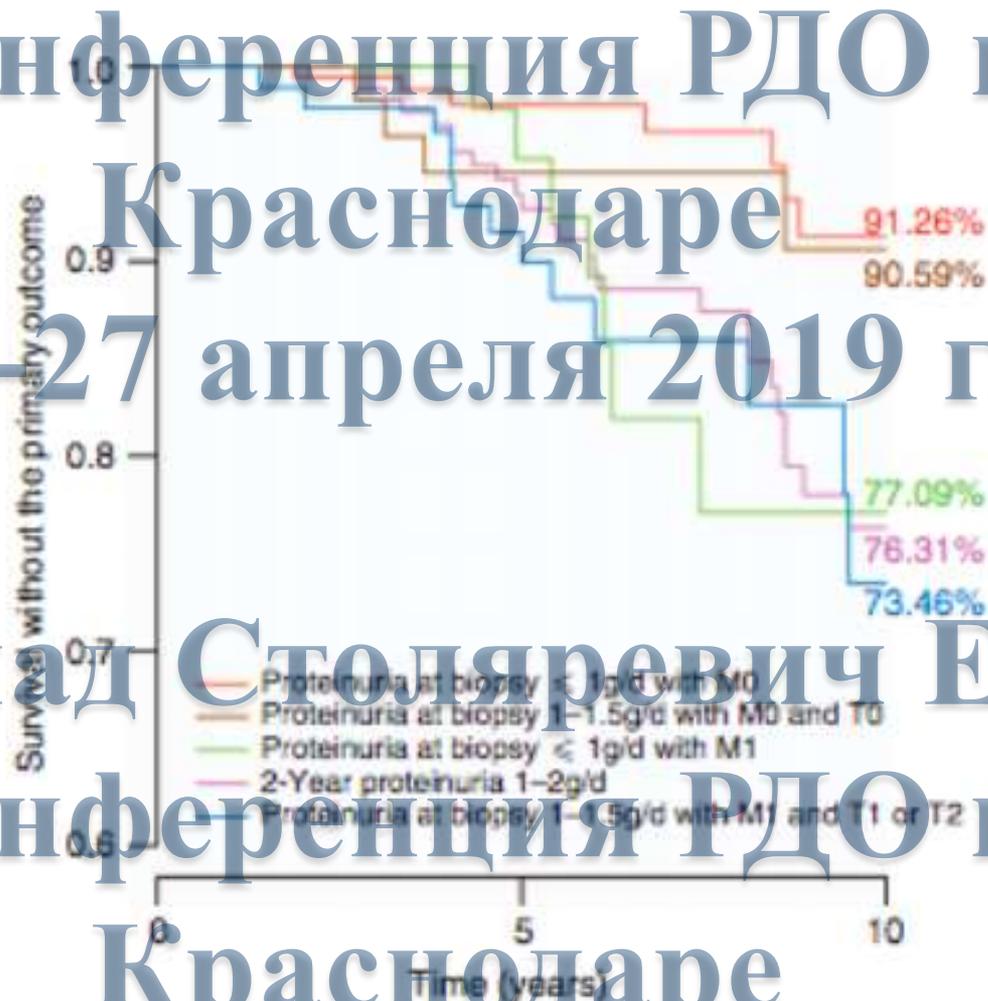
	OR	95% CI	P
Атрофия канальцев и инерст. фиброз			
0-25% (reference)	1.0		
26-50%	3.0	1.3-7.4	0.01
51-100%	21.8	2.3-206.2	0.007
Наличие сегментарного гл. склероза	2.8	1.2-6.2	0.01
Шкала мезангиальной гиперклет. >0.5	2.1	0.9-4.7	0.08

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Конференция РДО в
Краснодаре



Доклад Столяревич Е.С.

Validation of the Oxford classification of IgA nephropathy

Andrew M. Herzenberg^{1,6,7}, Agnes B. Fogo^{2,6}, Heather N. Reich^{1,6}, Stéphan Troyanov^{3,6}, Nuket Bavbek², Alfonso E. Massat⁴, Tracy E. Hunley², Michelle A. Hladunewich², Bruce A. Julian⁵, Fernando C. Fervenza⁴ and Daniel C. Cattran²

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У пациентов с эндокапиллярной пролиферацией:

темпы снижения СКФ:

У получавших патогенетическую терапию

- 2.3+/-5.4 мл/мин/1.73м² /год

У не получавших патогенетическую терапию

- 5.0+/-9.6 ml/min/1.73m² /год

P= 0.02

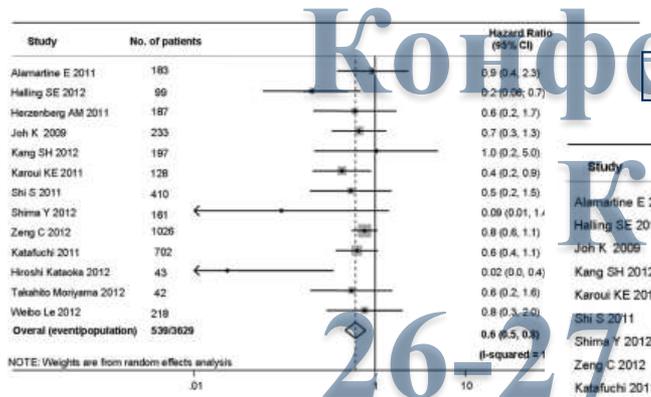
Краснодаре

187 пациентов
(взрослые и дети)
эндокапиллярной
пролиферацией)
4 центра США

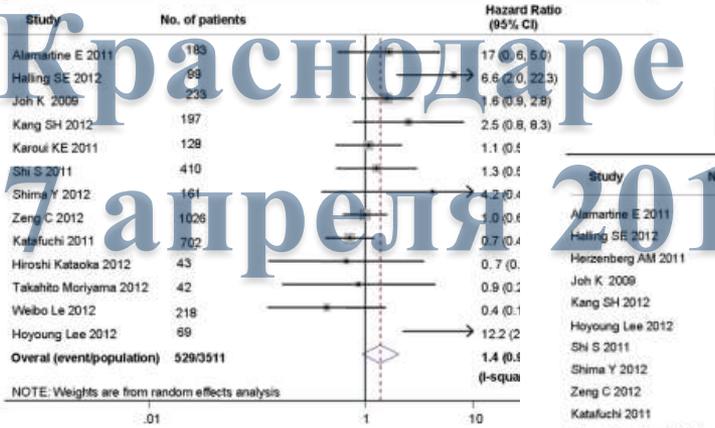
Эндокапиллярная пролиферация имеет прогностическое значение у пациентов, не получавших патогенетическую терапию

Данные метаанализа (16 ретроспективных исследований; 3,893 пациентов)

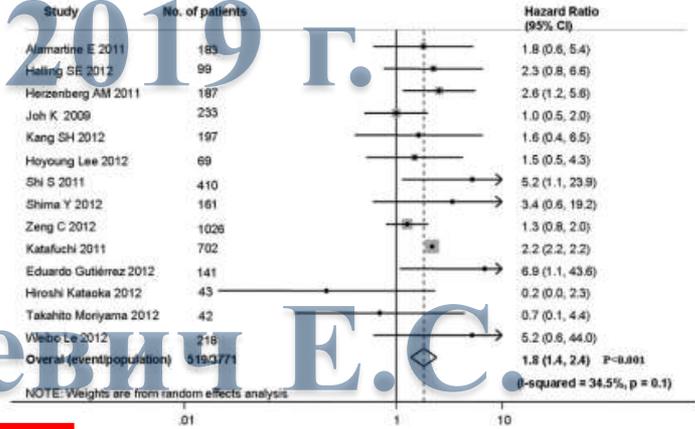
M-score HR-0,6 (0,5-0,8) p<0,01



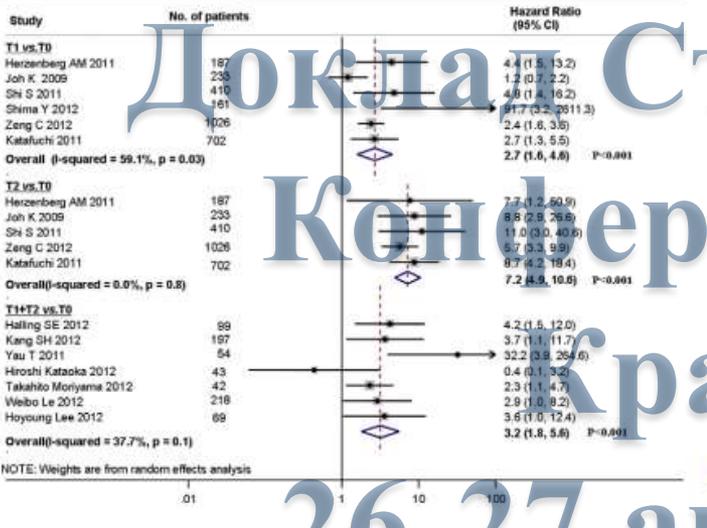
E-score HR-1,4 (0,8-2,0) p=0,1



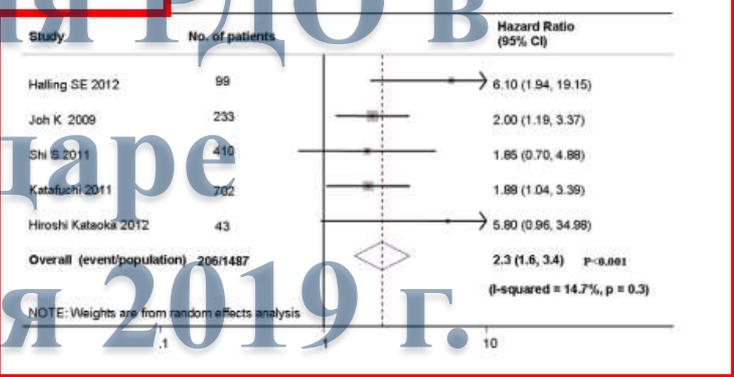
S-score HR-1,8 (1,4-2,4) p<0,01



T-score HR: T1 vs T0 -2,7 ;
T2 vs T0 -7,2 p<0,01



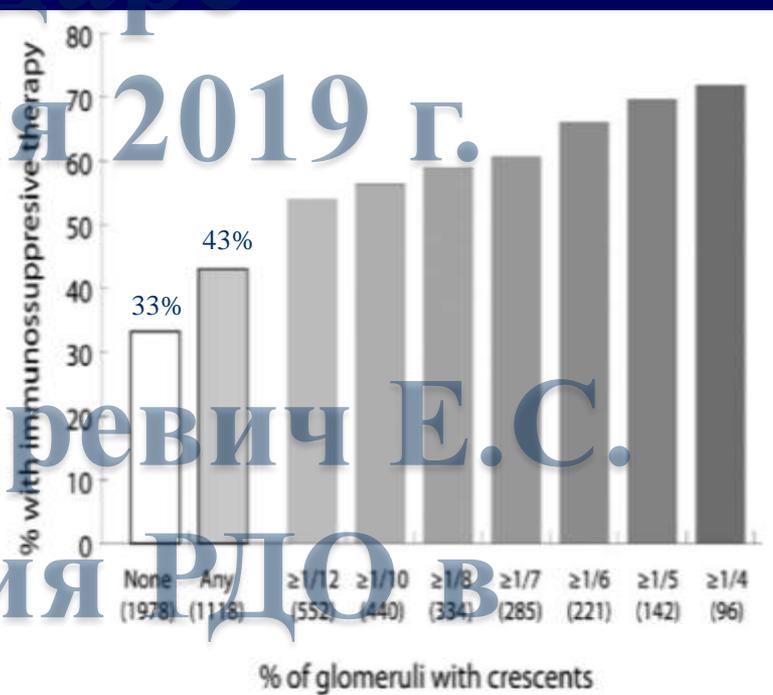
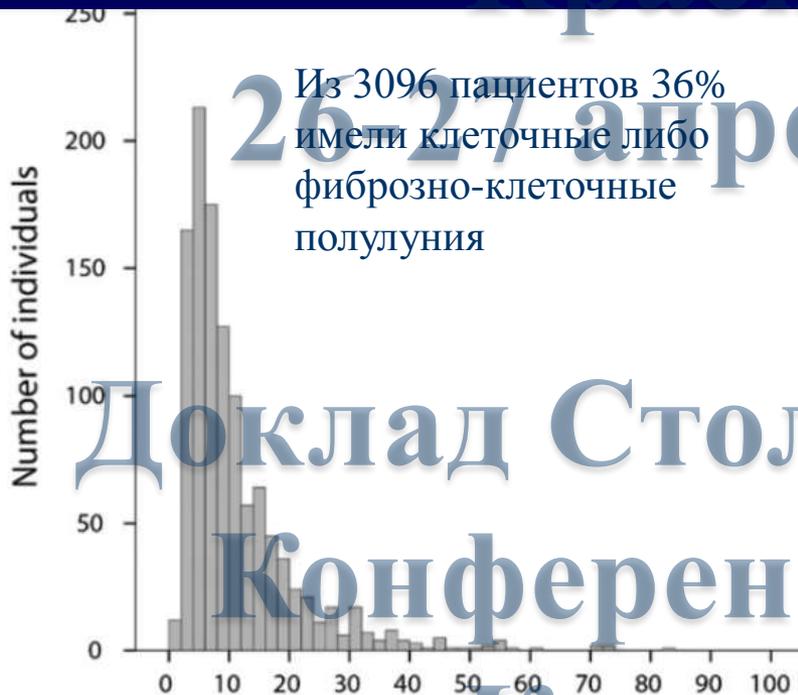
C-score HR-2,3 (1,6-3,4) p<0,01



Доклад Столяревич Е.С.

A Multicenter Study of the Predictive Value of Crescents in IgA Nephropathy

Mark Haas,* Jacobien C. Verhave,[†] Zhi-Hong Liu,[‡] Charles E. Alpers,[§] Jonathan Barratt,^{||} Jan U. Becker,[¶] Daniel Cattran,^{**} H. Terence Cook,^{††} Rosanna Coppo,^{‡‡} John Feehally,^{||}



Доклад Столяревич Е.С.

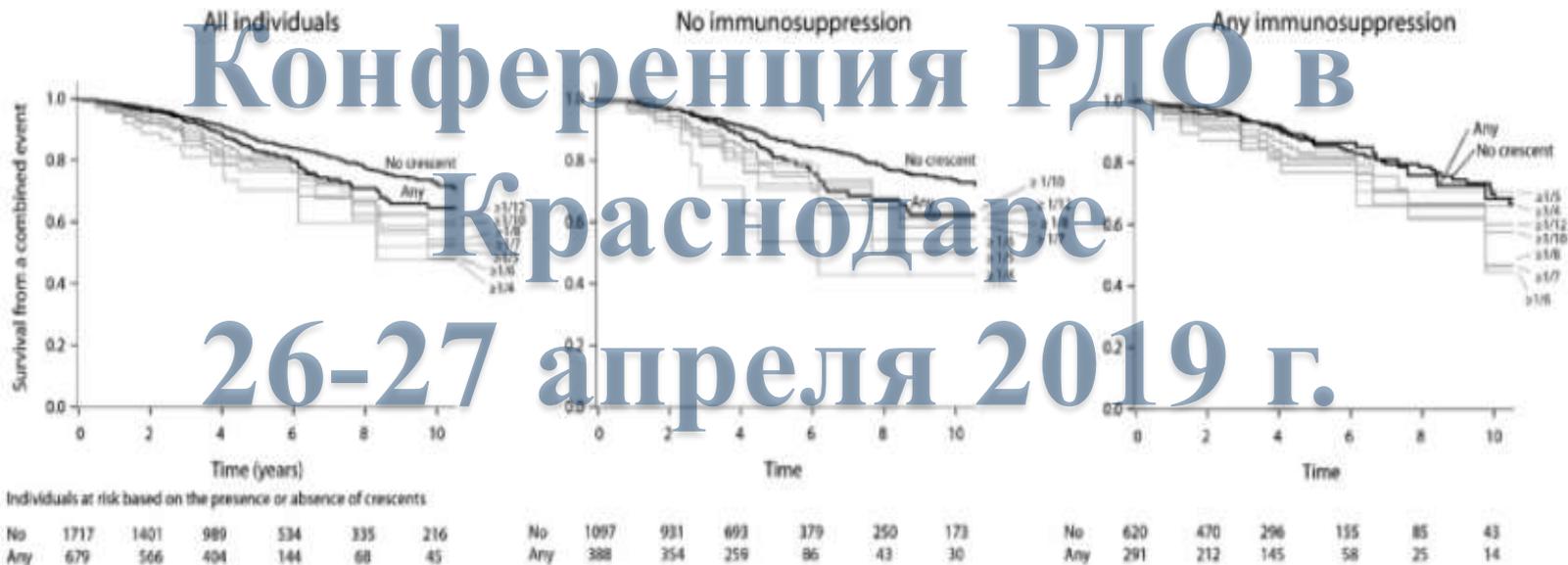
Конференция РДО в

Доклад Столяревич Е.С.

Конференция РДО в

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26-27 апреля 2019 г.



- Наличие полулуний значительно повышало риск тХТН у пациентов не получавших ИСТ
- Наличие полулуний в 25% и более клубочков значительно повышало риск тХТН у всех пациентов, независимо от терапии

Oxford Classification of IgA nephropathy 2016: an update from the IgA Nephropathy Classification Working Group



Hernán Trimarchi¹, Jonathan Barratt¹, Daniel C. Cattran³, H. Terence Cook⁴, Rosanna Coppo⁵, Mark Haas⁶, Zhi-Hong Liu⁷, Ian S.D. Roberts⁸, Yukio Yuzawa⁹, Hong Zhang¹⁰ and John Feehally² on behalf of the IgAN Classification Working Group of the International IgA Nephropathy Network and the Renal Pathology Society¹; for Conference Participants¹

¹Hospital Británico de Buenos Aires, Argentina; ²Department of Infection, Immunity, and Inflammation, University of Leicester, Leicester, United Kingdom; ³Department of Medicine, Toronto General Research Institute, University Health Network, Toronto General Hospital, Toronto, Ontario, Canada; ⁴Centre for Complement and Inflammation Research, Department of Medicine, Imperial College, London, United Kingdom; ⁵Fondazione Ricerca Biomedica, Citta' della Salute e della Ricerca Scientifica Hospital, Turin, Italy; ⁶Department of Pathology and Laboratory Medicine, University of Toronto, Toronto, Ontario, Canada; ⁷Department of Pathology, University of Turin, Turin, Italy; ⁸Department of Pathology, University of Leicester, Leicester, United Kingdom; ⁹Department of Pathology, University of Osaka Prefecture, Sakai, Japan; ¹⁰Department of Pathology, University of Leicester, Leicester, United Kingdom

Table 2 | Recommendations for updating the Oxford Classification of IgAN

- We recommend no changes to the published Oxford Classification of IgAN. A minimum of 8 glomeruli should be examined.
- We recommend that MEST criteria continue to be applied to cases of IgAN.
- We confirm the predictive value of M, S, and T.
- We confirm the predictive value of E in patients not treated with immunosuppression.
- We recommend that a C score be added to the MEST score in all cases of IgAN to indicate the frequency of cellular and/or fibrocellular crescents.
 - C0 (no crescents) or
 - C1 (crescent in a least 1 glomerulus) or
 - C2 (crescents in at least 25% of glomeruli)
- We recommend no change in the definition of S1, but adding text to indicate whether there are podocytopathic features.
- We recommend that MEST criteria are not yet applied to cases of Henoch-Schönlein purpura nephritis (IgA vasculitis).

C, crescent; E, endocapillary cellularity; IgAN, IgA nephropathy; M, mesangial hypercellularity; S, segmental sclerosis; T, interstitial fibrosis/tubular atrophy.

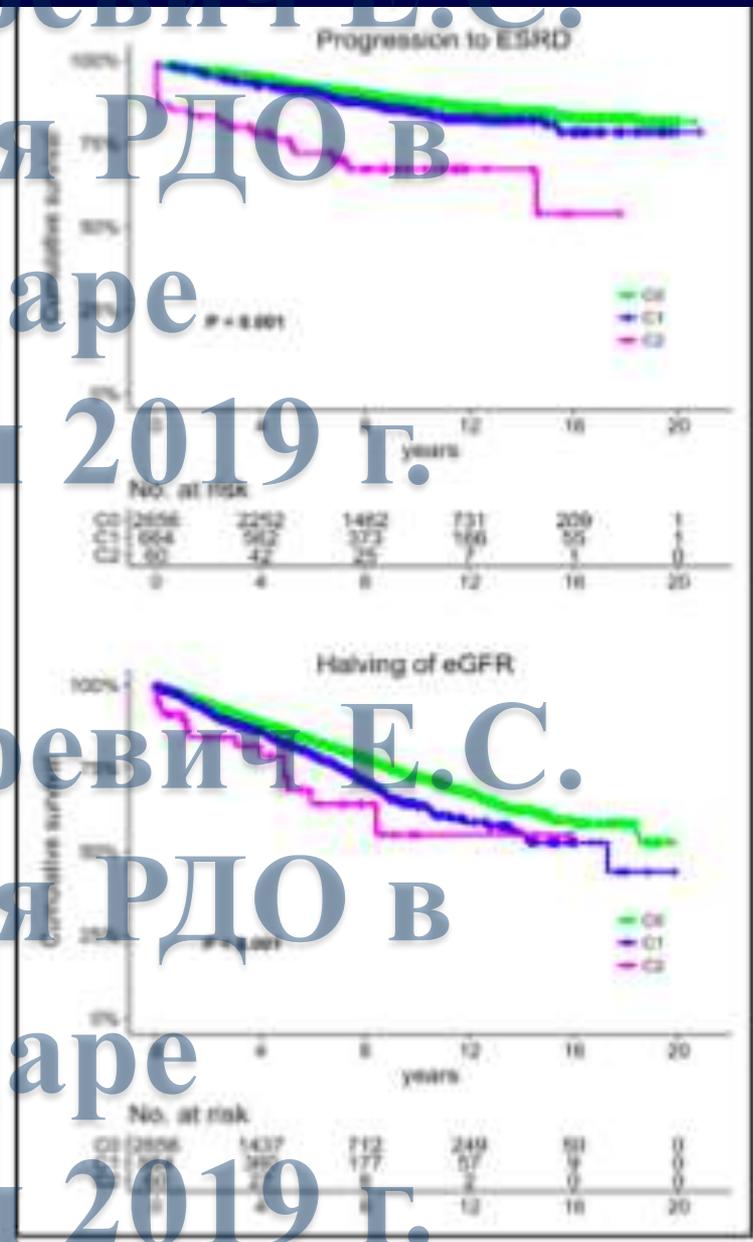
Сочетания MEST-C

Recommendations for the renal biopsy report in IgA nephropathy (adapted from refs. 1, 2, and 32)

Light microscopy	Immunohistochemistry or immunofluorescence
Electron microscopy	
Summary of 5 key pathologic features	
Mesangial score <0.5 (M0) or >0.5 (M1)	
Endocapillary hypercellularity absent (E0) or present (E1)	
Segmental glomerulosclerosis absent (S0) or present (S1); presence or absence of podocyte hypertrophy/tip lesions in biopsy specimens with S1	
Tubular atrophy/interstitial fibrosis ≤25% (T0), 26%–50% (T1), or >50% (T2)	
Cellular/fibrocellular crescents absent (C0), present in at least 1 glomerulus (C1), in >25% of glomeruli (C2)	
Quantitative data	
Total number of glomeruli	
Number of glomeruli with endocapillary hypercellularity, necrosis, extracapillary hypercellularity (cellular/fibrocellular crescents), global glomerulosclerosis, and segmental glomerulosclerosis	

Clinical Significance of Crescent Formation in IgA Nephropathy – a Multicenter Validation Study

Sehoon Park^{1*} Chung Hee Baik² Su-Kil Park³ Hee Gyung Kang⁴
Hye Sun Hyun⁵ Eujin Park⁶ Seung Hyeok Ham⁷ Dong-Ryeol Ryu⁸
Dong Ki Kim⁹ Kook Hwan Oh¹⁰ Yoon Wook Park¹¹ Han Su Kim¹²



Подходы к диагностике IgA-нефропатии:

Низкий риск:

- Прот < 0.5 г/с
- микрогематурия
- СКФ – N

Биопсия не обязательна

контроль

Средний риск:

- Прот 0.5-1 г/с
- СКФ – N
- АГ ±

Биопсия необходима

активность

Патогенетическая терапия

Высокий риск:

- Прот > 1-3 г/с
- СКФ – снижена/БПГН

Биопсия не обязательна

склероз

Нефропротекция

ХПН:

- СКФ < 30 мг/л
- Размеры почек уменьшены

Подготовка к ЗПТ





10.3.1: При персистировании протеинурии > 1г/сут (несмотря на лечение иАПФ или БРА в течение 3-6 мес при хорошем контроле АД) и СКФ >50 мл/мин рекомендованы КС в течение 6 мес (2С)

Краснодаре

Table 2. Corticosteroid monotherapy

Trial	Pozzi et al., Italy ^{37,36}	Katafuchi et al., Japan ³⁸	Hogg et al., United States ²⁶	Manno et al., Italy ³⁵	Lv et al., China ³⁴
Corticosteroid regimen	Intravenous methylprednisolone 1 g/d for 3 consecutive days at the beginning of months 1, 3, and 5, plus oral prednisone 0.5 mg/kg every other day for 6 months	Oral prednisolone 20 mg/d tapered to 5 mg/d at 18 months	Oral prednisone every other day 60 mg/m ² for 3 months, then 40 mg/m ² for 9 months, and then 30 mg/m ² for 12 months	Oral prednisone for 6 months (1 mg/kg/day for 2 months, then reduced by 0.2 mg/kg/day per month)	Oral prednisone for 6-8 months (0.8-1 mg/kg/day for 2 months, then reduced by 5-10 mg every 2 wk)
Control regimen	Supportive only	Dipyridamole	Placebo	Supportive only	Supportive only
RAS blockade	14% at baseline, allowed during follow-up	2% at baseline; allowed during follow-up	Enalapril if hypertensive	Ramipril in all patients	Cilazapril in all patients
Key outcome in steroid group versus control	Ten-year renal survival (=absent doubling of serum creatinine), 53% in controls versus 97% in the steroid group	Significant reduction in proteinuria but not ESRD frequency	No benefit in the steroid group versus placebo at 2 years	Mean annual loss of GFR 6.2 ml/min in controls versus 0.6 ml/min in the steroid group	Significantly fewer patients with a 50% increase in serum creatinine in the steroid group

Therapeutic regimens and outcomes in randomized controlled trials in IgAN patients. RAS, renin-angiotensin system; ESRD, end-stage renal disease.

В/в №3 по 1г/с (в I, III и V мес лечения) +
РО 0,5 мг/кг/сут – 6 мес

Эффективно (СКФ)

РО 20 мг/сут со снижением до 5 мг/с к 18 мес

Нет эффекта

РО 60мг/с ч/день 3мес, 40мг до 9мес, 30 мг- до 12 мес

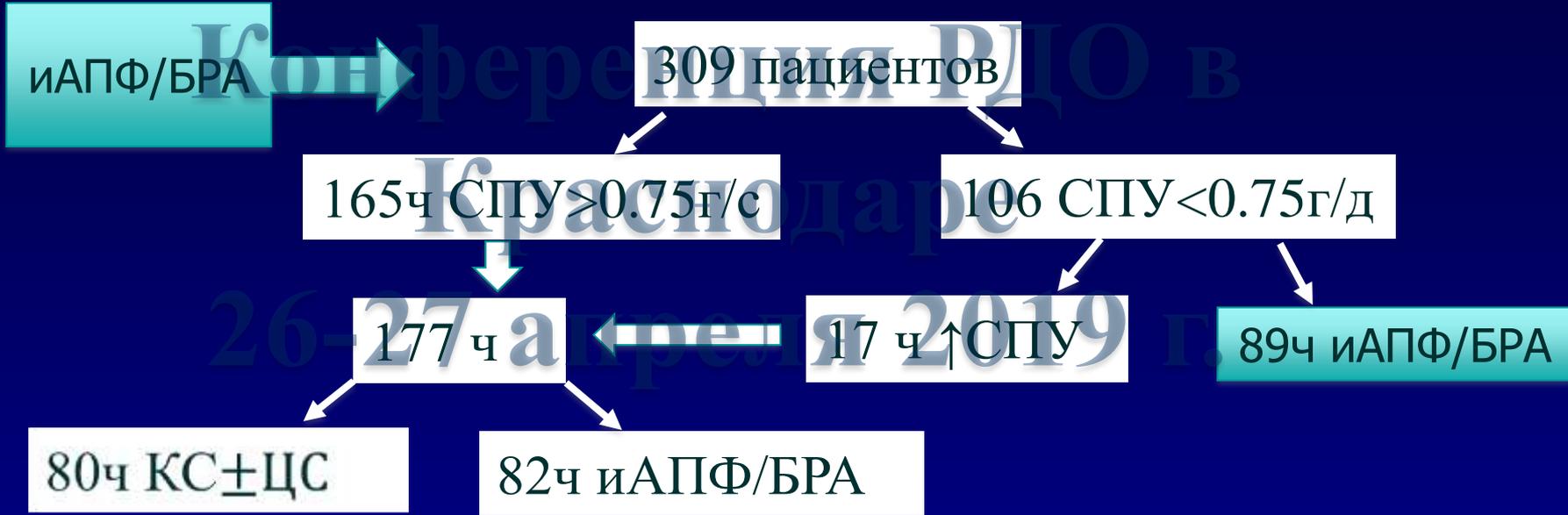
Нет эффекта

РО 6мес: 1мг/кг/с - 2мес с последующим снижением на 0.2 мг/кг за месяц

Эффективно (СКФ)

РО 6-8 мес: 0.8-1 мг/кг – 2 мес с последующим снижением на 5-10 мг каждые 2 нед

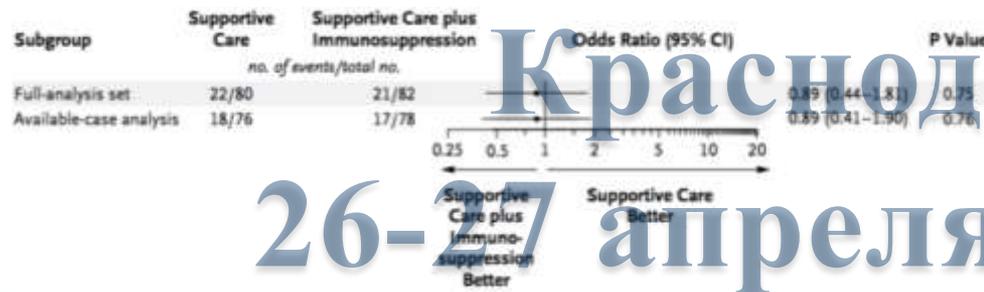
Исследование STOP-IgAN



Вероятность развития полной ремиссии



Вероятность развития ТХПН



Исследование STOP-IgAN

162 пациента, завершивших 6-месячное исследование
были включены в 3-летнее исследование

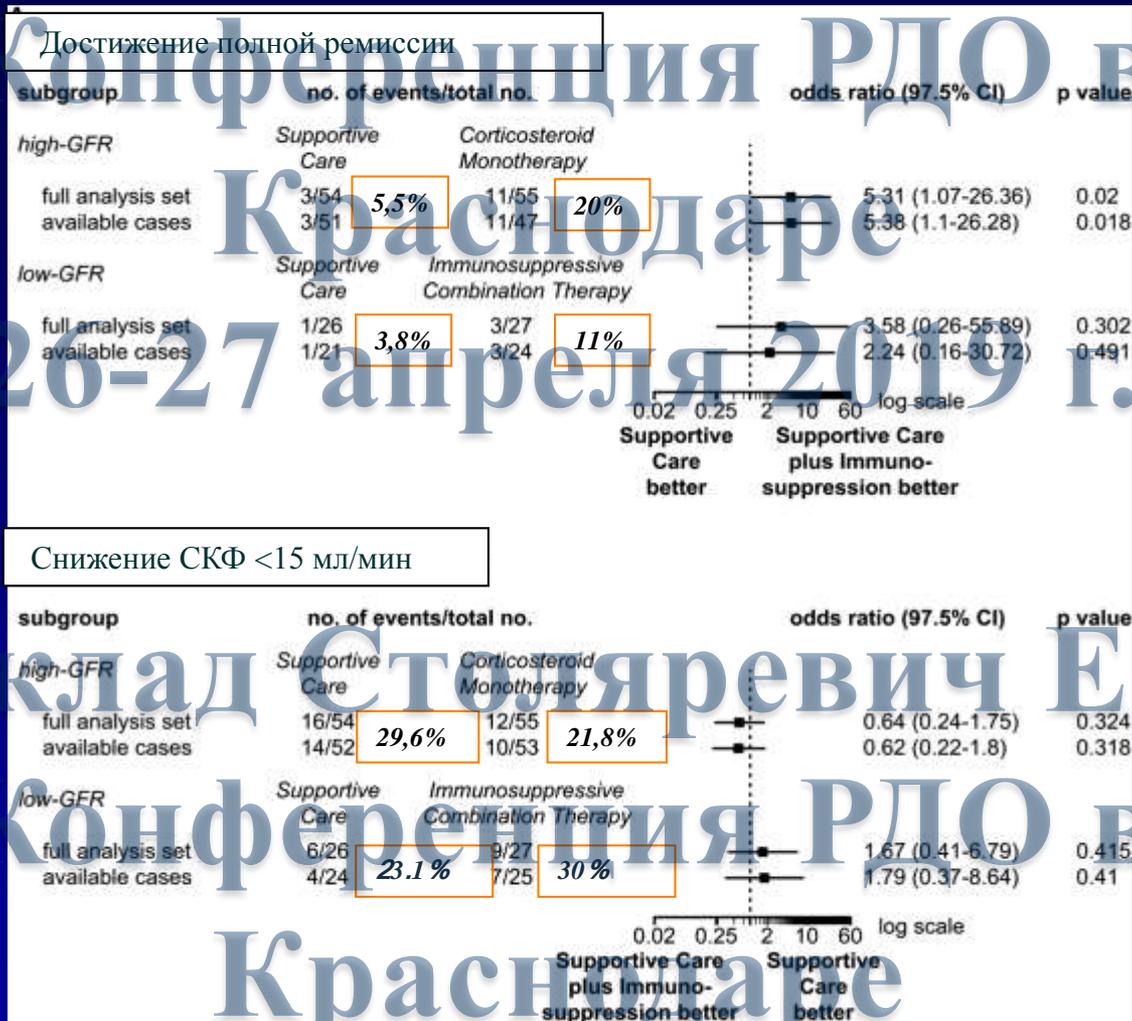


Краснодаре
26-27 апреля 2019

Thomas Rauen et al. JASN 2018;29:317-325

JASN

Исследование STOP-IgAN



Thomas Rauen et al. JASN 2018;29:317-325

JASN

Мета-анализ 7 IgAN исследований : отсутствие дополнительного эффекта при использовании КС в сочетании с ЦС

Краснодаре

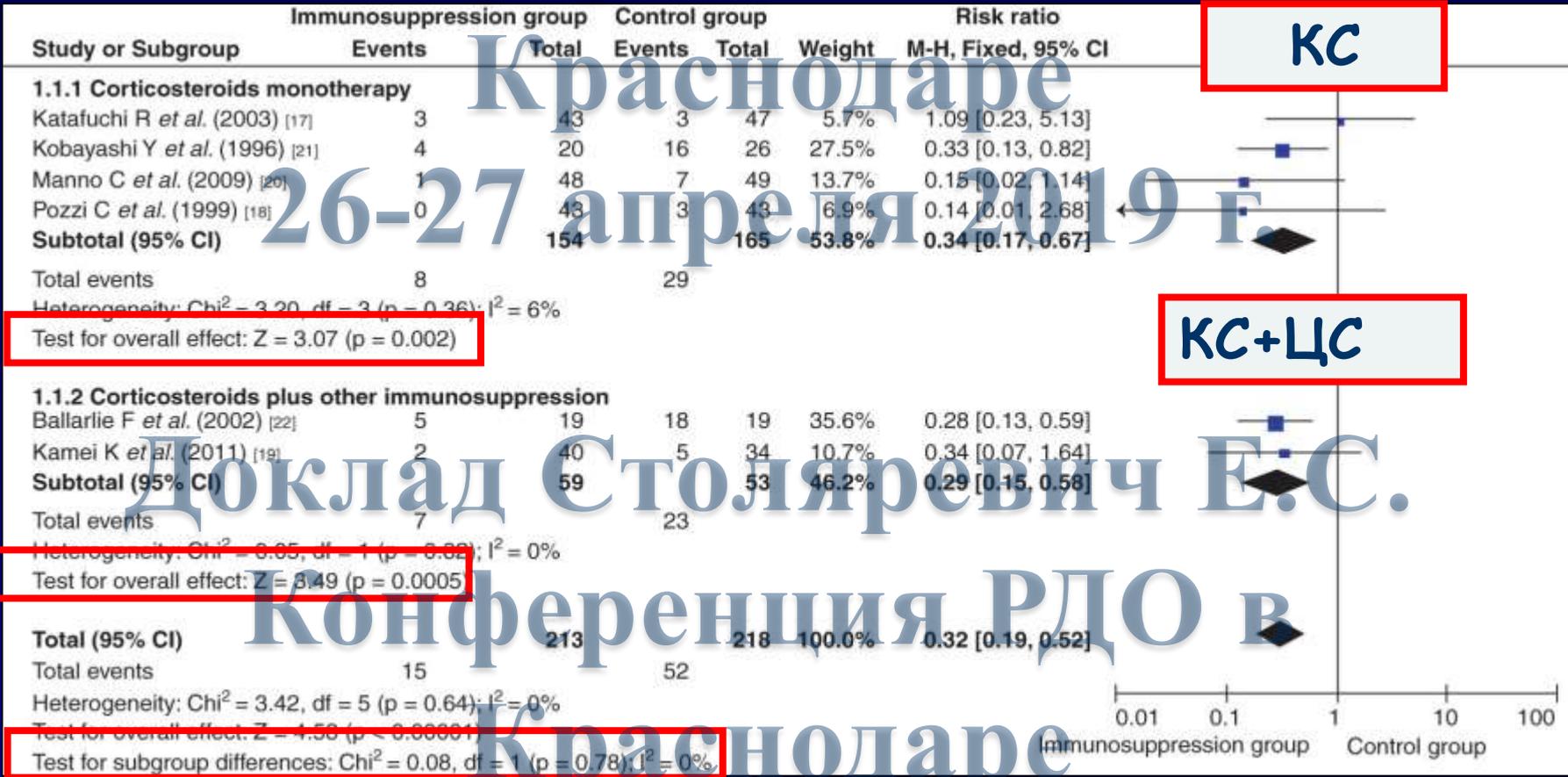
26-27 апреля 2019 г.

Доклад Столяревич Е.С.

Конференция РДО в

Краснодаре

26-27 апреля 2019 г.



Effect of Oral Methylprednisolone on Clinical Outcomes in Patients With IgA Nephropathy

The TESTING Randomized Clinical Trial

Jicheng Lv, MD; Hong Zhang, PhD; Muh Geot Wong, PhD; Meg J. Jardine, PhD; Michelle Hladunewich, MD

Vivek
Dar
Lau
Joh
Hai

Key Points

Question Do corticosteroids safely prevent loss of kidney function in patients with IgA nephropathy receiving optimal supportive therapy?

Findings This randomized clinical trial that included 262 participants was stopped early (after 28 of the 335 planned events) due to a significantly increased risk of serious adverse events with oral methylprednisolone vs placebo (14.7% vs 3.2%, primarily excess infections); at that point, the primary efficacy outcome favored methylprednisolone (5.9% vs 15.9%).

Meaning Oral corticosteroid therapy was associated with an increased risk of serious adverse events; the effect on kidney outcomes remains uncertain due to the limited number of events.

Вероятность



No. at risk	0	6	12	18	24	30	36
Methylprednisolone	136	116	115	106	94	71	51
Placebo	126	122	122	118	107	83	64

ИВАЕМОСТЬ



No. at risk	0	6	12	18	24	30	36
Methylprednisolone	136	129	111	89	60	34	16
Placebo	126	122	107	78	57	36	18

Targeted-release budesonide versus placebo in patients with IgA nephropathy (NEFIGAN): a double-blind, randomised, placebo-controlled phase 2b trial



Bengt C Fellström, Jonathan Barratt, Heather Cook, Rosanna Coppa, John Feehally, Johan W de Fijter, Jürgen Floege, Gerd Hetzel, Alan G Jardine, Francesco Locatelli, Bart D Maes, Alex Mercer, Fernanda Ortiz, Manuel Praga, Søren S Sørensen, Vladimir Tesar, Lucia Del Vecchio, for the NEFIGAN Trial Investigators

Summary

Background IgA nephropathy is thought to be associated with mucosal immune system dysfunction, which manifests as renal IgA deposition that leads to impairment and end-stage renal disease in 20–40% of patients within 10–20 years. In this trial (NEFIGAN) we aimed to assess safety and efficacy of a novel targeted-release formulation of budesonide (TRF-budesonide), designed to deliver the drug to the distal ileum in patients with IgA nephropathy.

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 See Online/Comment
<http://dx.doi.org/10.1016/>

протеинурия

СКФ

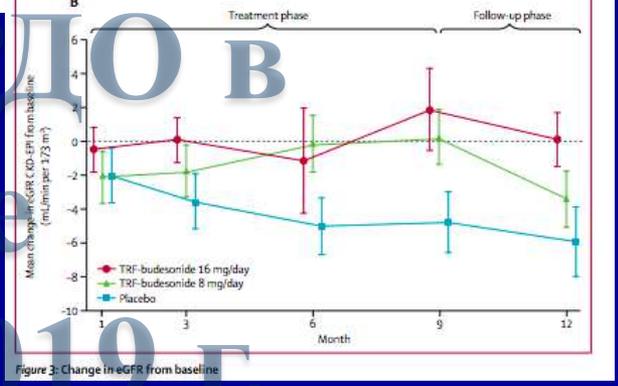
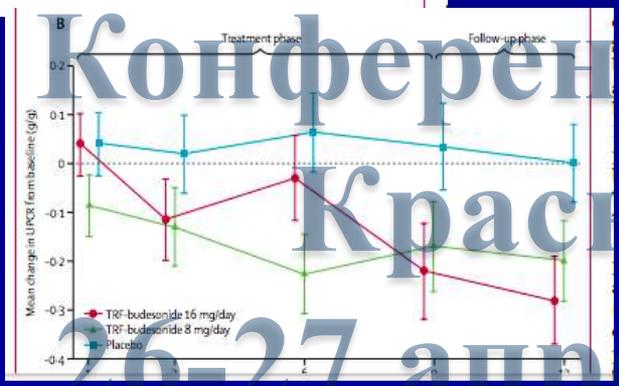
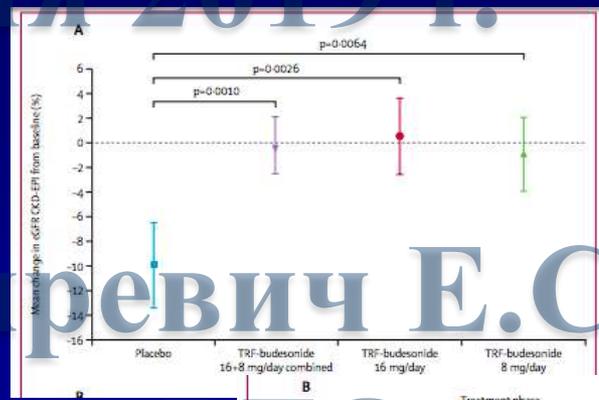
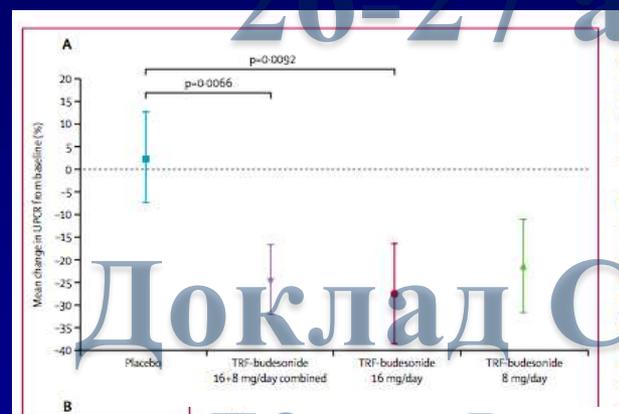


Figure 3: Change in eGFR from baseline

Другие иммуносупрессанты*



10.4.1 Мы предлагаем не лечить КС в сочетании ЦФ или Аза пациентов с IgA-нефропатией за исключением случаев БТГН с полулуниями (2D)

10.4.2 Мы предлагаем не использовать ИСТ у пациентов с СКФ < 30 мл/мин за исключением случаев IgA-нефропатии с полулуниями и картиной БТГН (2C)

10.4.3 Мы предлагаем не использовать ММФ при IgA-нефропатии (2C)

* Противоречивые результаты исследований, недостаточные для формулирования четких рекомендаций

Доклад Столяревич Е.С.

AJKD
Original Investigation
Mycophenolate Mofetil Combined With Prednisone Versus Full-Dose Prednisone in IgA Nephropathy With Active Proliferative Lesions: A Randomized Controlled Trial

Jin-Hua Hou, MD,¹ Wei-Bo Le, PhD,^{1*} Nan Chen, MD,² Wei-Ming Wang, PhD,² Zhang-Suo Liu, MD,³ Dong Liu, PhD,³ Jiang-Hua Chen, MD,⁴ Jiong Tian, PhD,⁵ Ping Fu, MD, PhD,⁵ Zhang-Xue Hu, MD,⁵ Cai-Hong Zeng, PhD,¹ Shao-Shan Liang, MD,¹ Min-Lin Zhou, MD,¹ Hai-Tao Zhang, MD,¹ and Zhi-Hong Liu, MD¹

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Доклад Столяревич Е.С.

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Ритуксимаб в лечении IgA-нефропатии

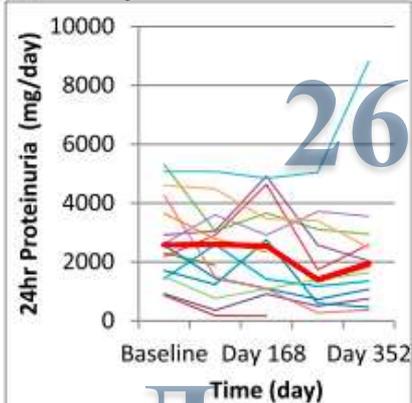
Доклад Столяревич Е.С.

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А Ритуксимаб



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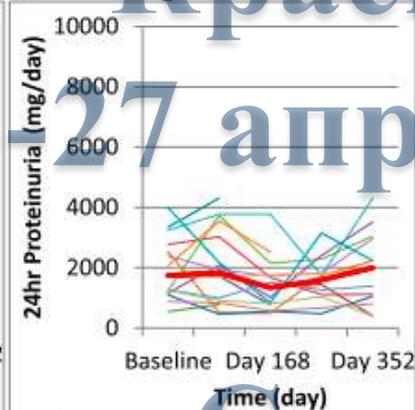


Figure 3. Proteinuria trends in (A) rituximab versus (B) control groups. The red line represents median data.

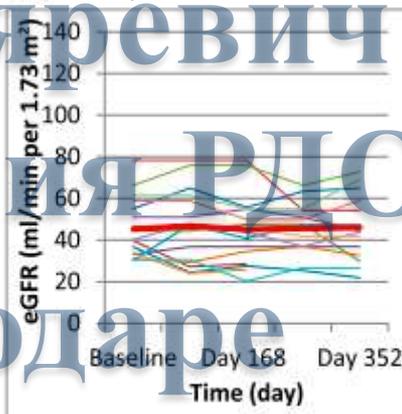
CLINICAL RESEARCH www.jasn.org

A Randomized, Controlled Trial of Rituximab in IgA Nephropathy with Proteinuria and Renal Dysfunction

Richard A. Lafayette,¹ Pietro A. Canetta,¹ Brad H. Rovin,² Gerald B. Appel,¹ Jan Novak,³ Karl A. Nath,⁴ Sanjeev Sethi,⁵ James A. Tumlin,^{**} Kshama Mehta,^{*} Marie Hogan,¹ Stephen Erickson,¹ Bruce A. Julian,^{††} Nelson Leung,¹ Felicity T. Enders,^{‡‡} Rhubell Brown,[§] Barbara Knoppova,^{§§} Stacy Hall,[¶] and Fernando C. Fervenza¹

¹Division of Nephrology and Hypertension, Stanford University, Stanford, California; ²Division of Nephrology and Hypertension, Columbia University Medical Center, New York, New York; ³Division of Nephrology, Ohio State University, Columbus, Ohio; Departments of ⁴Microbiology and ⁵Medicine, University of Alabama at Birmingham, Birmingham, Alabama; ^{††}Department of Laboratory Medicine and Pathology, and

А Ритуксимаб



В КОНТРОЛЬ

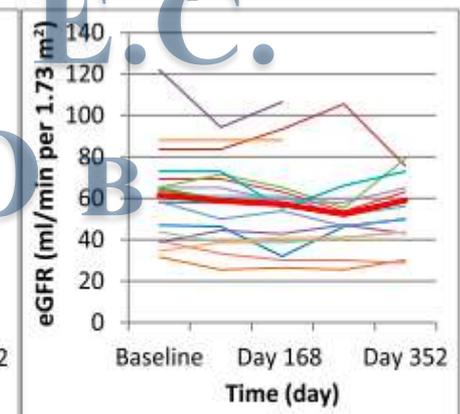


Figure 2. eGFR trends in (A) rituximab versus (B) control groups. The red line represents average data.

Ингибиторы кальцинейрина в лечении IgA-нефропатии

протеинурия

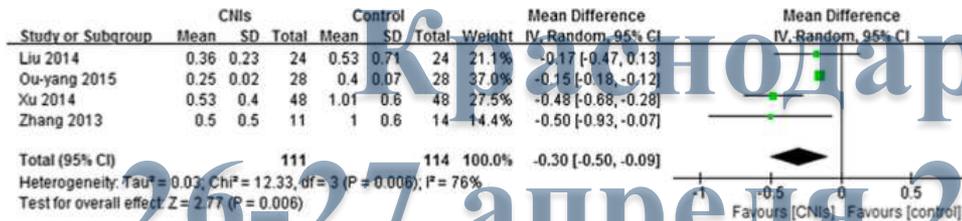


Fig. 3 Forest plot of the Effect of CNIs for proteinuria (g/d) at the end of treatment or during follow-up

СКФ

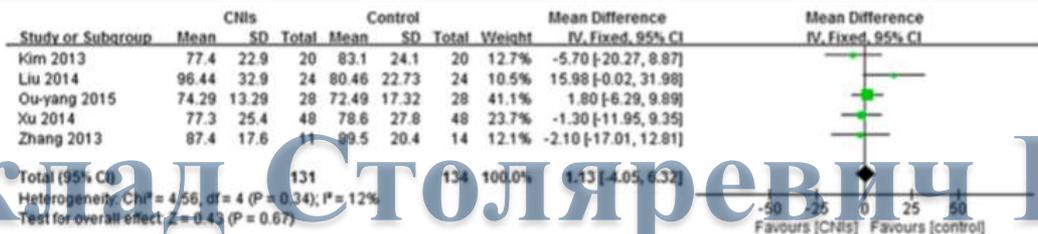


Fig. 4 Forest plot of the Effect of CNIs on eGFR at the end of treatment or during follow-up

Scr

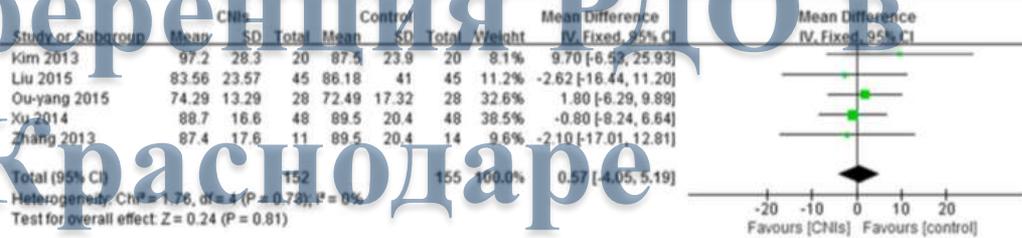


Fig. 5 Forest plot of the Effect of CNIs on Scr at the end of treatment or during follow-up

Tакролимус в лечении IgA-нефропатии

Tacrolimus Decreases Albuminuria in Patients with IgA Nephropathy and Normal Blood Pressure: A Double-Blind Randomized Controlled Trial of Efficacy of Tacrolimus on IgA Nephropathy

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Abstract

Background: Treatment remains uncertain for IgA nephropathy if antiproteinuric medication or the RAS blocker is not applicable.

Trial design: A double-blinded randomized trial.

Methods: The anti-proteinuric effect of tacrolimus was explored in 100 patients with IgA nephropathy. We randomly assigned patients either to receive tacrolimus or

Yong-Chul Kim et al.
PLOS ONE | www.plosone.org
2013 | Volume 8 | Issue 8 | e71545

Mi-yeon Yu et al.
PLOS ONE |
<https://doi.org/10.1371/journal.pone.0188375>
November 20, 2017

RESEARCH ARTICLE

Short-term anti-proteinuric effect of tacrolimus is not related to preservation of the glomerular filtration rate in IgA nephropathy: A 5-year follow-up study

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Подходы к лечению IgA-нефропатии:

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