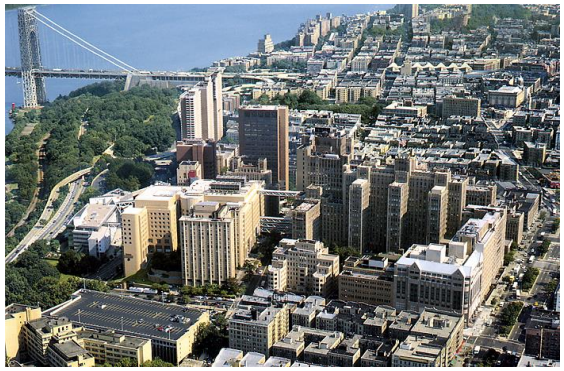


Board Review Questions on Glomerular Diseases

Gerald B. Appel, M.D.

Professor of Medicine

Founder - The Center for Glomerular Diseases

Division of Nephrology

Columbia University College of Physicians and Surgeons

G Appel Disclosures

Dr. Appel has research grants with Sanofi-Genzyme, Apellis, Mallinkrodt, Novartis, Vera, and Vertex.

He has consultantships with : Alexion-Achillion, Apellis, Aurinia, Glaxo , Calliditas, Roche-Genentech, Mallinkrodt, Pfizer, Merck, Up-to-Date, Genzyme-Sanofi , Chinook, Novartis, Vertex, Vera.

He has lectureships with Aurinia and Glaxo on LN and Calliditas on mechanisms of IgA Nephropathy.

Question 1

Which initial patient presentation is least likely to be associated with new onset minimal change disease?

1) **65 year old M with AKI and 4+ proteinuria**
2) **32 year old F with 2.1 g proteinuria daily**
3) **45 year old M with 9 g proteinuria daily**
4) **28 year old M with High BP, microhematuria and 4+ proteinuria.**

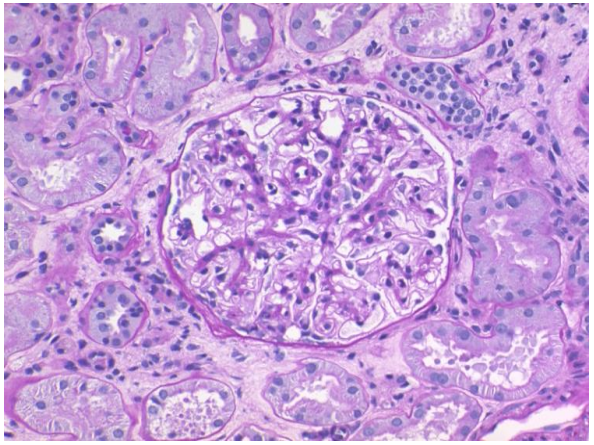
Features, Treatment, Course
Adult MCD – 5 Pearls

95 Bxed Adult MCD at CUMC

1) **Reduced GFR - GFR 72 cc/min Pcreat 1.4 mg/dl**
2) **Heavy proteinuria -24h Protein 9.9 g/day**
   hematuria 20% HBP 43%
3) **AKI at presentation 17% (eventual 24%)**
4) **No Diference in Pred response QD vs QOD (76% vs 74%) or in time to response (11 v 16 wks) or percent relapse (75% v 63%)**
5) **Relapse Common Of responders 40% relapse by 6 months , most respond again to steroids**

Waldman M…Appel GB CJASN 2 445-454 2007.

Question 2

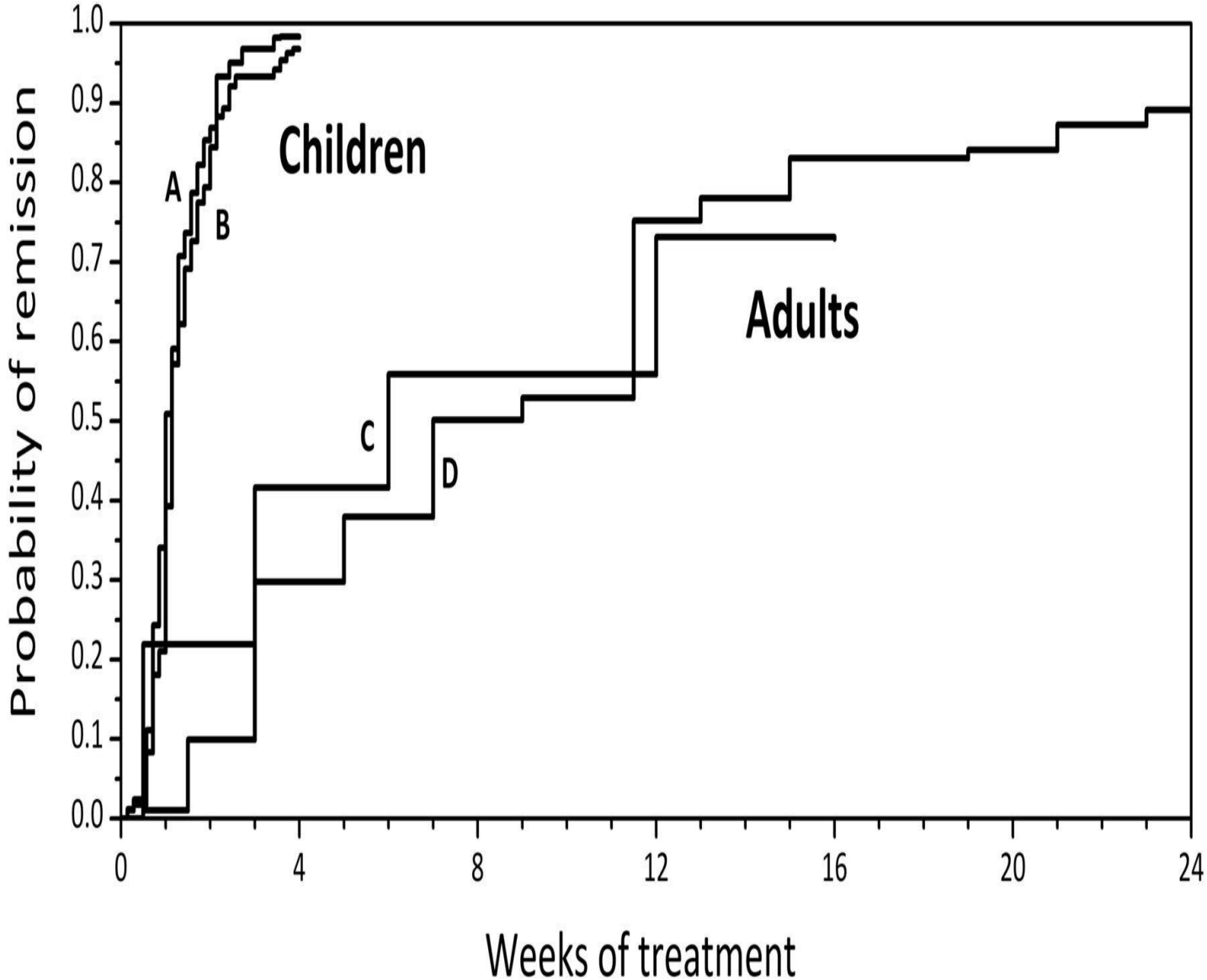


In general, adult patients with Minimal Change Disease respond to corticosteroids similar to children and can be considered steroid resistant if they do not have a reduction in proteinuria by 2 months of therapy.

True or False?

Time-to-response to prednisone is much shorter in children than in adults with MCD

M Vivarelli et al. CJASN 2017;12:332-345

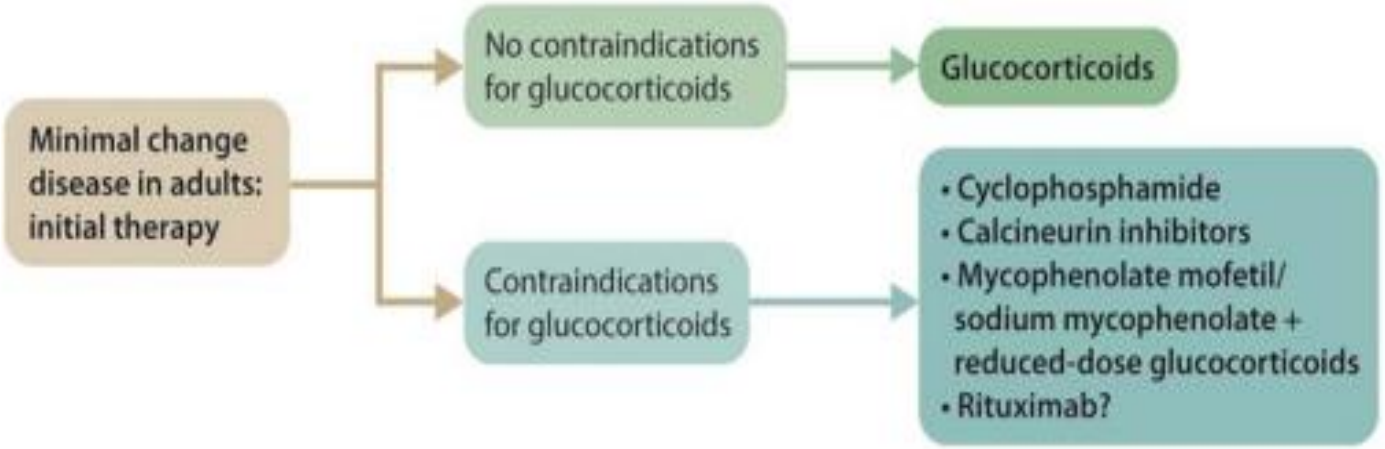


Question 3

A 38 yo F with the nephrotic syndrome due to MCD has responded to a course of prednisone with a complete remission but has relapsed x 3 whenever she discontinues the prednisone. Which therapy will give the highest remission rate for treatment at this time?

1) cyclosporine
2) cyclophosphamide
3) Tacrolimus
4) Mycophenolate mofetil
5) All above are equivalent

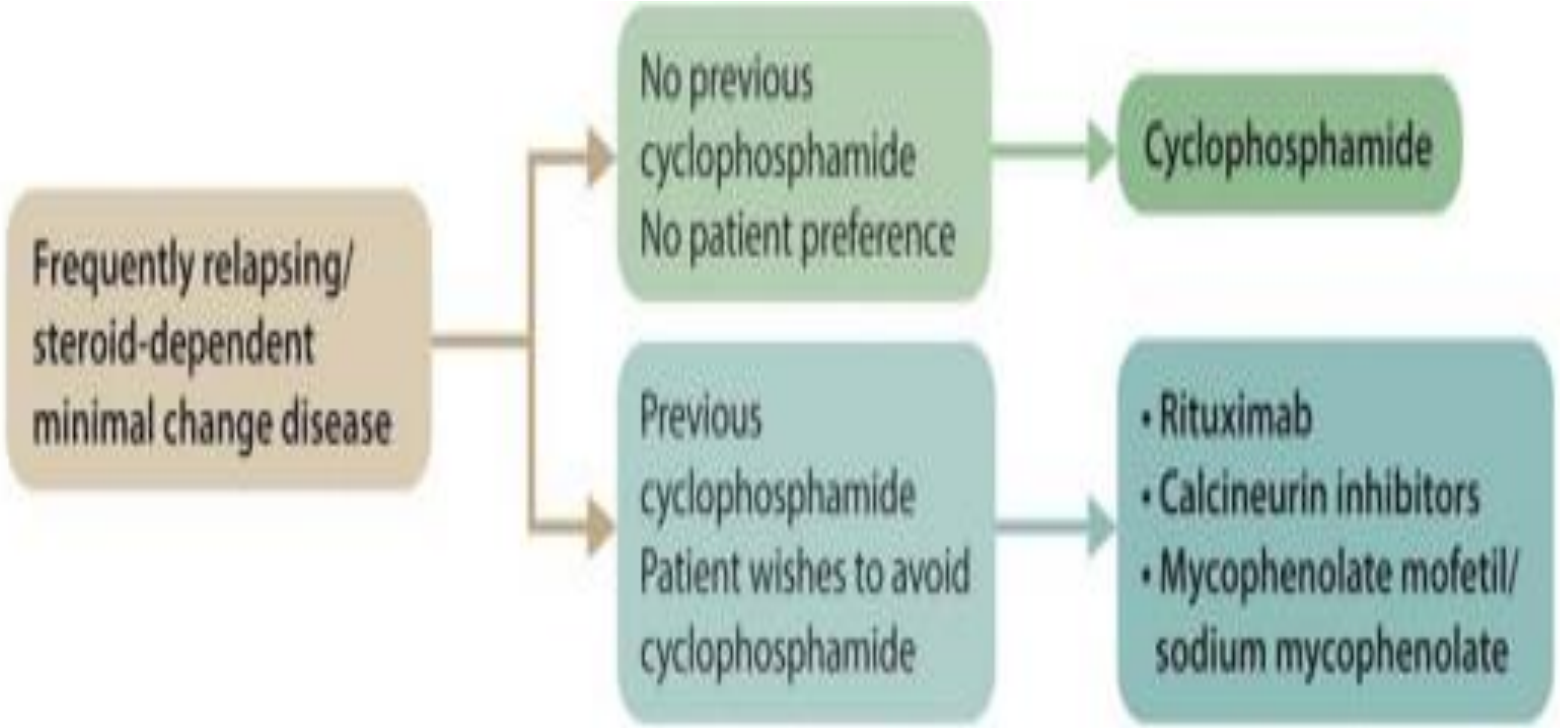
KDIGO – Initial therapy of MCD in Adults



Contraindications: All relative – Obesity, Diabetes, Cosmetic concerns, Prior problems with steroids.

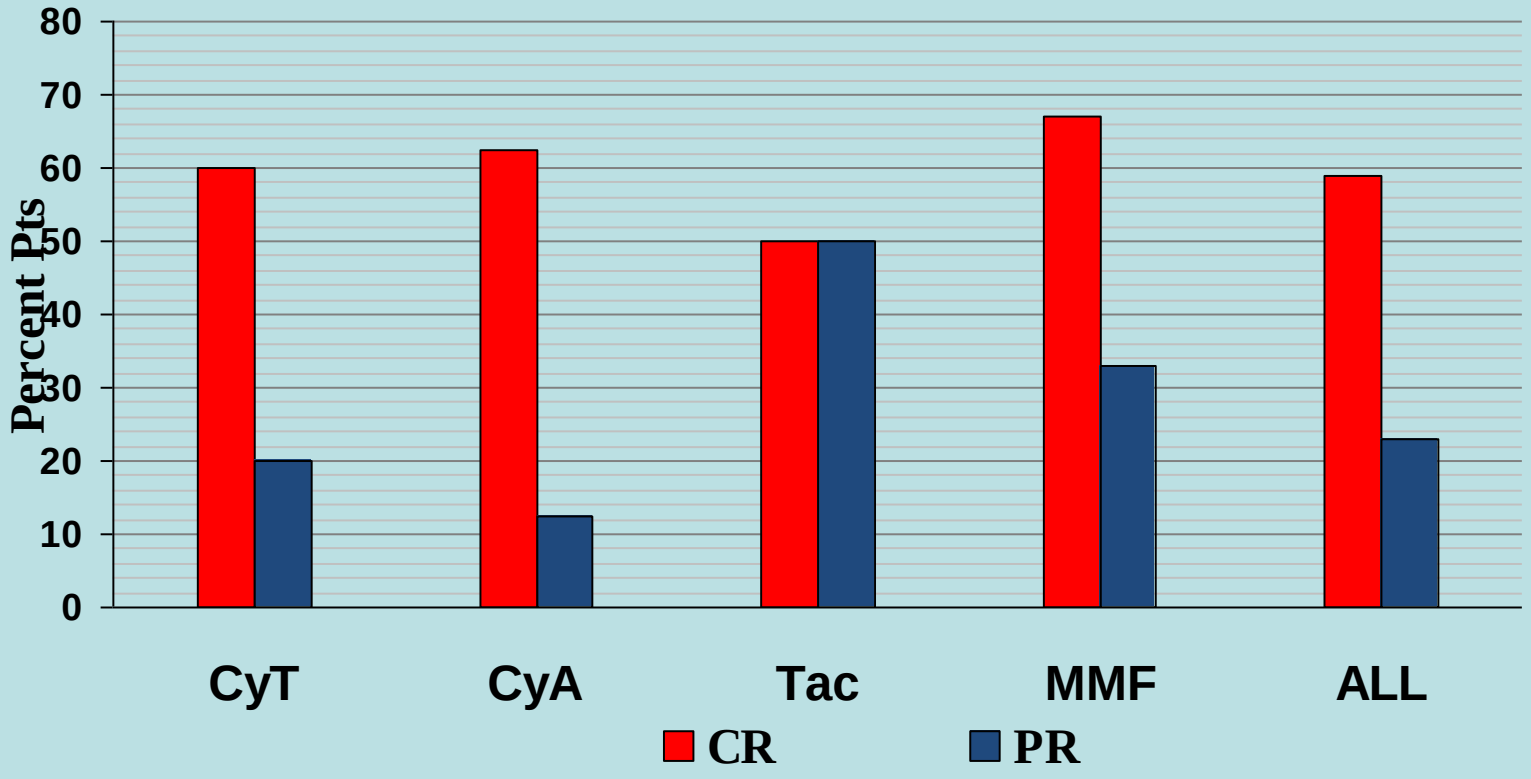
KDIGO Executive Summary 2021 Guidelines for management of Glomerular Disease. Kidney Int. 100: 753-779, 2021

KDIGO – Therapy of FR/Steroid Dependent MCD in Adults



KDIGO Executive Summary 2021 Guidelines for management of Glomerular Disease. Kidney Int. 100: 753-779, 2021

Steroid-Dependent or FR Adult MCD:
Response to Second Line Agents is Similar



Waldman, Appel et al, CJASN 2007

	Li et al 2017 (n = 119)		Patil et al 2019 (n = 48)		Thomas et al 2020 (n = 50)		Chin et al 2021 (n = 144)	
Regimen	IV MP 0.8 mg/kg x 10 days + Tacrolimus	Oral Pred 1 mg/kg	Tacrolimus only Tx	Oral Pred 1 mg/kg	Tacrolimus only Tx	Oral Pred 1 mg/kg	Tacrolimus + Oral Pred 0.5 mg/kg	Oral Pred 1 mg/kg
Remission	98%	96%	80%	78%	88%	92%	79%	77%
Toxicity	n = 81	n = 128	n = 20	n = 17	n = 20	n = 17	n =127	n = 133
Relapse	45%	49%	32%	39%	72%	74%	6%	23%

Long-Term Outcomes of Rituximab in Adults w Podocytopathies

- **183 adults** (64 FSGS , 119 MCD) with difficult to treat NS (68% SD/FR , 22% SR, 85% prior treated with 2 or more immune meds)
- All Rx with **Rituximab**.
- **Complete or partial remit at 6 months in 82%**
- **55% of responders achieved long-term (3 yr) relapse free survival**
- Over 36 mo stable GFR of initial responders with complete or partial remission
- Maintenance therapy associated with better relapse free survival

Gaukler et al. JASN 36:668-678.2025

Long-Term Outcomes of Rituximab in Adults w Podocytopathies

Gaukler et Al JASN 36:668-678, 2025

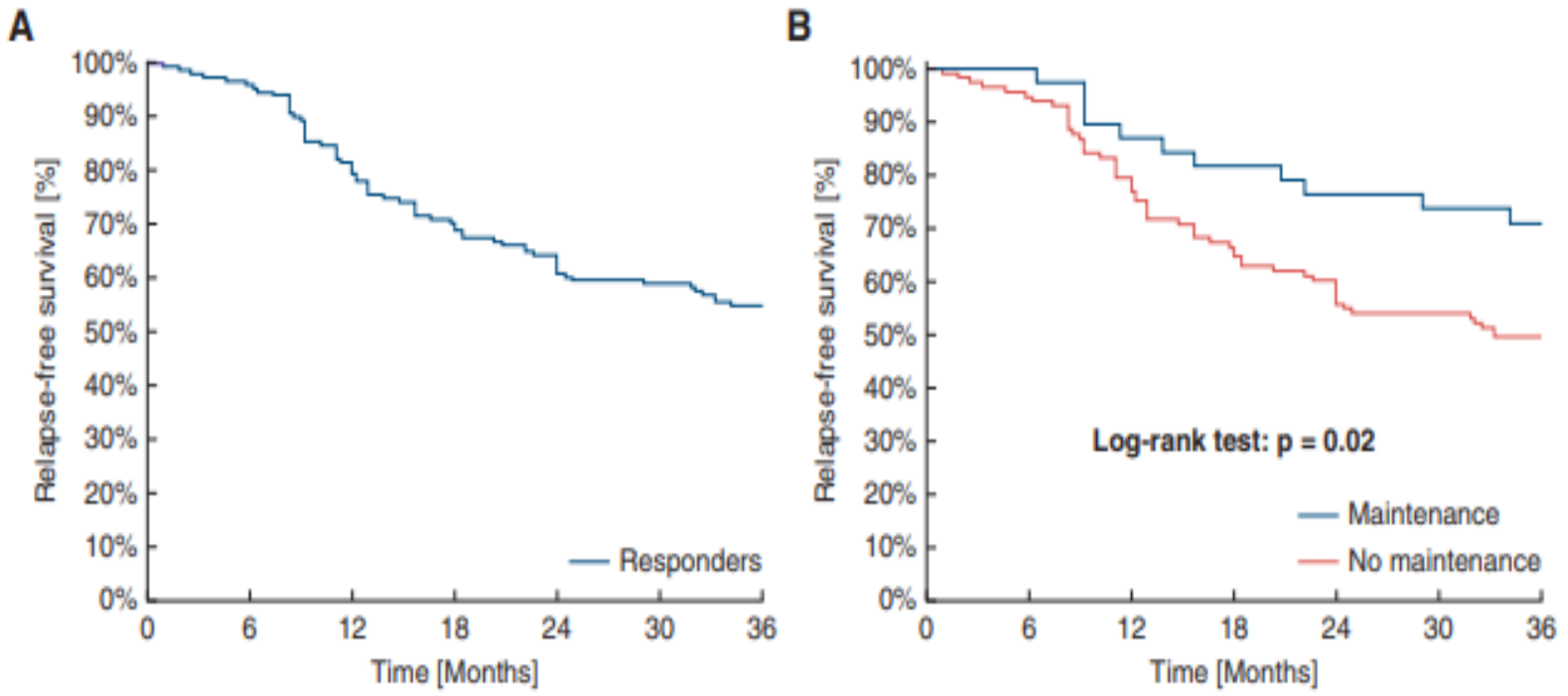
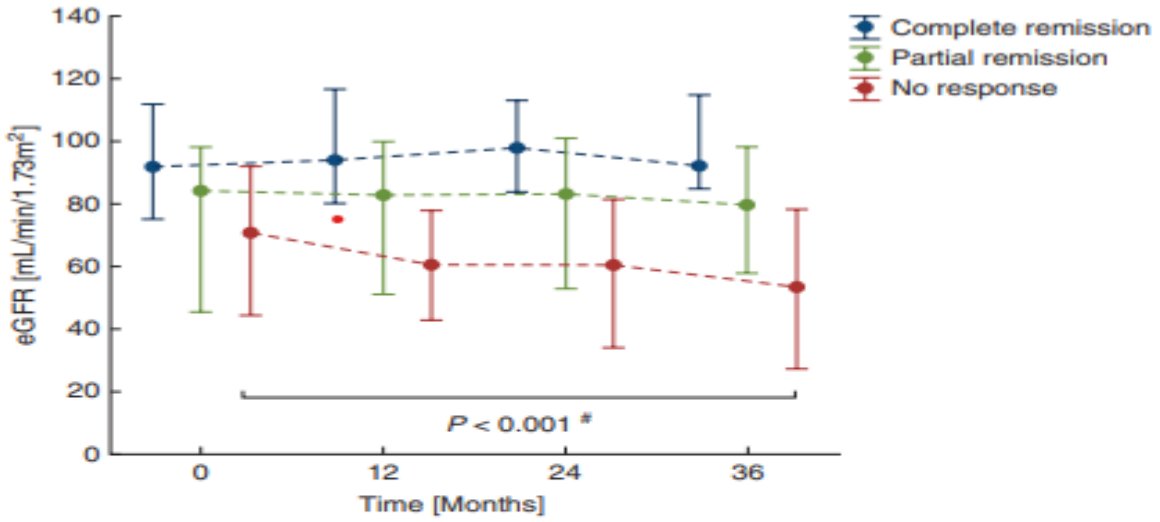


Figure 1. Three-year relapse-free survival in rituximab responders. Three-year relapse-free survival in all initial rituximab responders of the total cohort (A) and subgroup comparison with maintenance (red line) versus without maintenance (blue line) treatment with rituximab (B).

Long-Term Outcomes of Rituximab in Adults w Podocytopathies

Gaukler et Al JASN 36: 668=678.2025 2025



Group	n	eGFR [mL/min/1.73m²]					ΔeGFR
		Baseline	12 months	24 months	36 months	P – value*	0-36 months
Complete remission	107	92 (75-112)	94 (80-117)	98 (84-113)	92 (85-115)	0.99	0 (-6 to +9)
Partial remission	33	84 (46-98)	83 (51-100)	83 (53-101)	80 (58-98)	0.08	-4 (-9 to +14)
No response	28	71 (45-92)	61 (43-78)	61 (34-82)	53 (28-79)	<0.001	-11 (-33 to -6)#

igure 4. eGFR course over 3 years in complete responders, partial responders, and those with no response. The eGFR course over time

Question 4

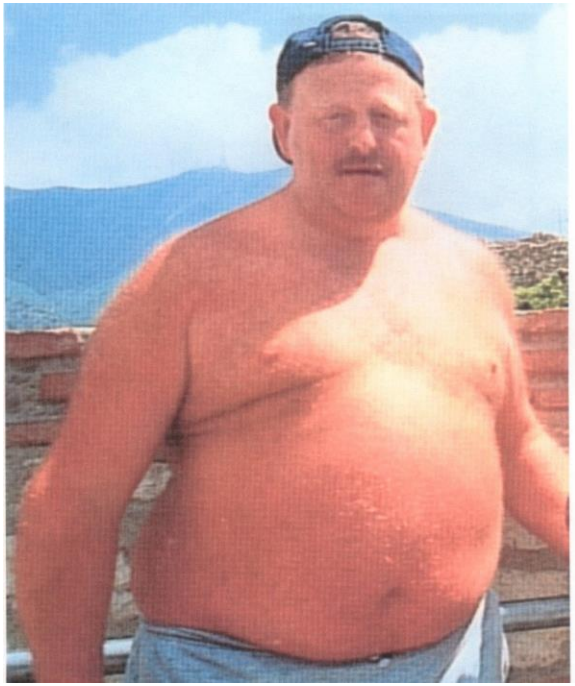
Which clinical and laboratory features

suggest an obese nephrotic patient is more

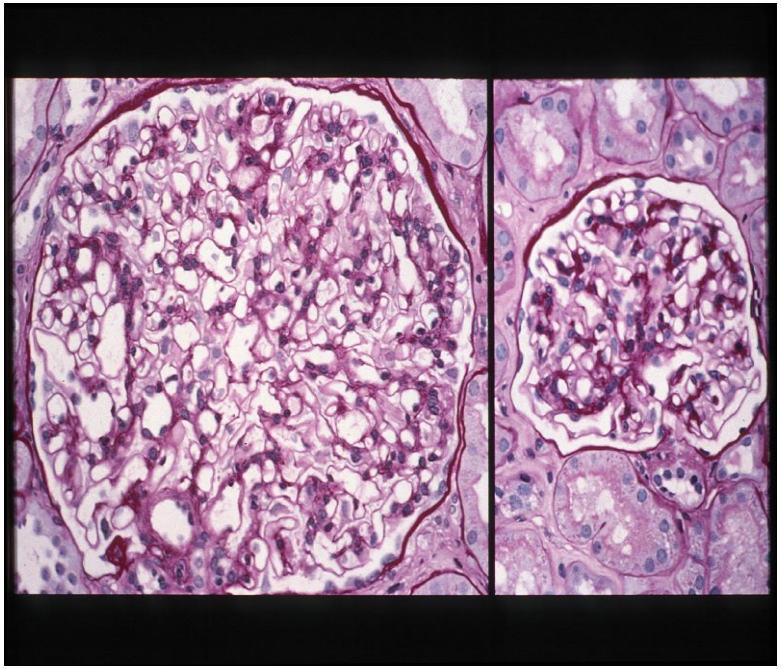
likely to have a secondary (“hyperfiltration” etiology) rather than primary FSGS?

1) Serum albumin of 2.4 g/dl and 9 g proteinuria/day
2) Serum albumin of 3.8 g/dl and 3.4 g proteinuria/day
3) Perihilar FSGS and absence of hematuria
4) BMI > 40 and plasma albumin < 2 g/dl

Obesity Related Glomerulomegaly and Secondary FSGS



- Lesser degrees of Proteinuria
- Higher P albumin
- Glomerulomegaly
- Perihilar FSGS



Question 5

Which genetic defect of FSGS is **NOT** typically associated with an autosomal dominant inheritance?

1) alpha actinin 4
2) Inverted formin 2
3) Podocin
4) TRPC6 channel

Genetic Podocyte Mutations

- Alpha actinin 4 familial autosomal dominant FSGS.
- Podocin (NPHS2) autosomal recessive steroid resistant nephrotic syndrome
- TRPC6 channel defect – autosomal dominant FSGS (Nature Genetics 2005)
- Formin INF2 gene – autosomal dominant (Nature Genetics 42:72, 2010)

Question 6

- Which disease, ultimately proven to be genetic by Whole Exome Sequencing, is most commonly mistaken for FSGS on light microscopy?
- 1) Nail patella Syndrome
- 2) Collagen 4 A 3,4,5 defects .
- 3) Fabry's disease
- 4) Tuberous Sclerosis

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

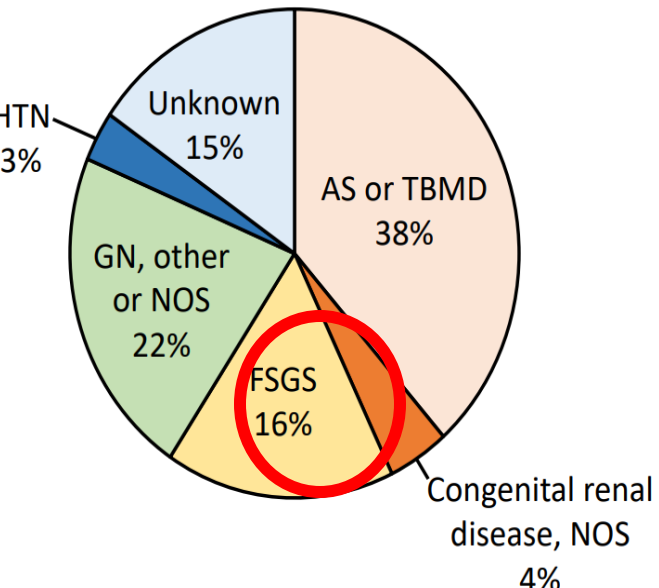
Diagnostic Utility of Exome Sequencing for Kidney Disease

E.E. Groopman, M. Marasa, S. Cameron-Christie, S. Petrovski, V.S. Aggarwal, H. Milo-Rasouly, Y. Li, J. Zhang, J. Nestor, P. Krithivasan, W.Y. Lam, A. Mitrotti, S. Piva, B.H. Kil, D. Chatterjee, R. Reingold, D. Bradbury, M. DiVecchia, H. Snyder, X. Mu, K. Mehl, O. Balderes, D.A. Fasel, C. Weng, J. Radhakrishnan, P. Canetta, G.B. Appel, A.S. Bomback, W. Ahn, N.S. Uy, S. Alam, D.J. Cohen, R.J. Crew, G.K. Dube, M.K. Rao, S. Kamalakaran, B. Copeland, Z. Ren, J. Bridgers, C.D. Malone, C.M. Mebane, N. Dagaonkar, B.C. Fellström, C. Haefliger, S. Mohan, S. Sanna-Cherchi, K. Kiryluk, J. Fleckner, R. March, A. Platt,

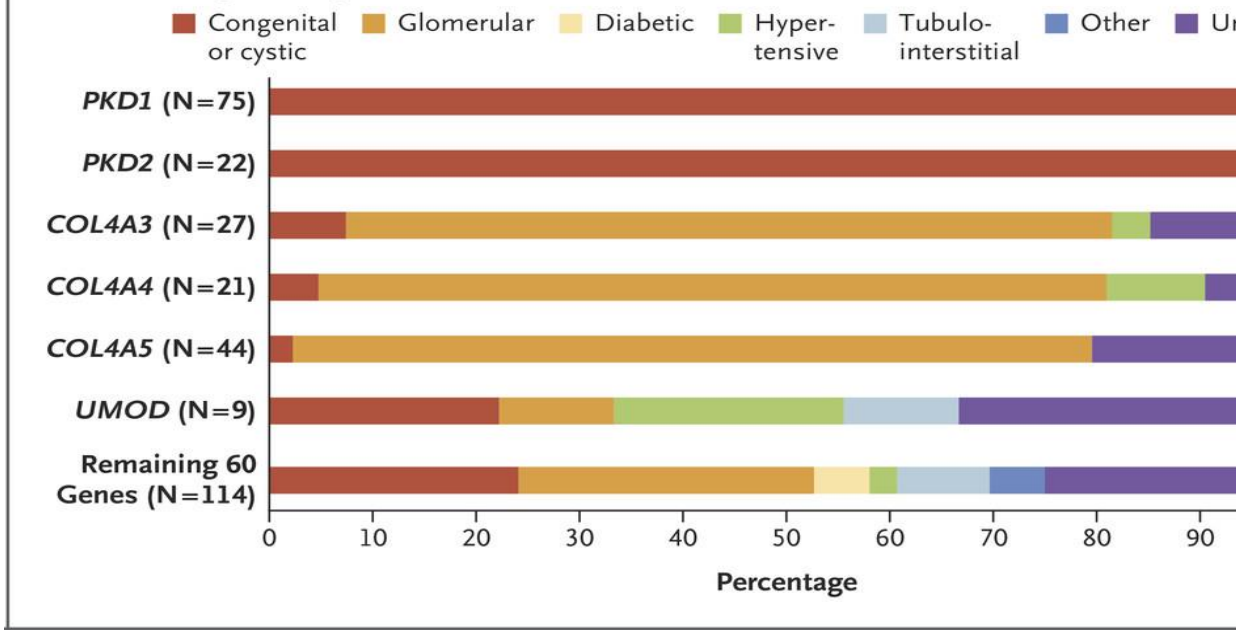
NEJM 380: 142-151
Jan 2019

307 of 3315 pts (9.3%)

A Common Genetic Findings



B Clinical Diagnostic Spectrum



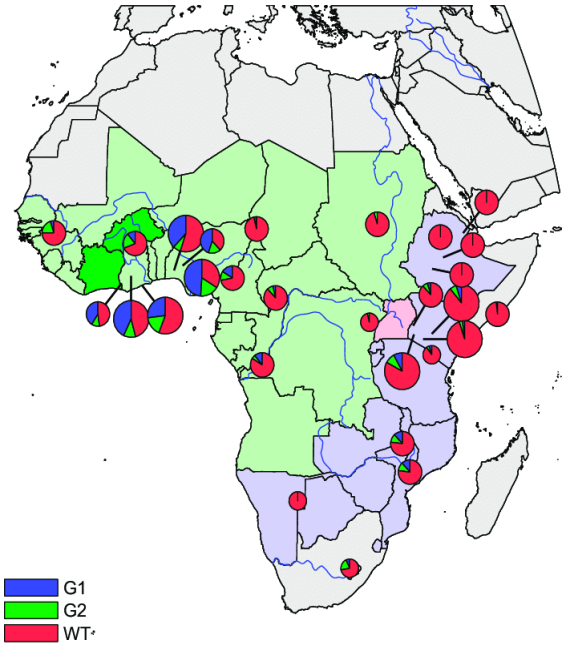
Question 7

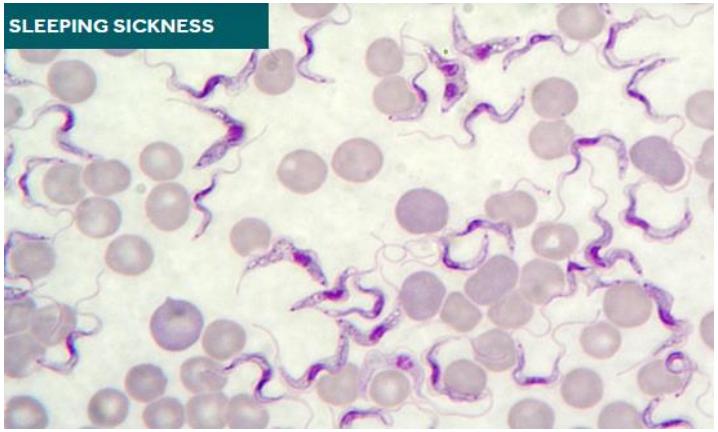
Glomerulosclerosis related to APOL1 high risk genes (G1G1,G1G2,G2G2) increase the risk of having FSGS and all of the following diseases EXCEPT which of the following?

1) HIVAN
2) COVID associated nephrotic syndrome
3) Systemic lupus nephritis
4) Diabetes mellitus

A Risk Allele for FSGS in African Americans is located in a region Containing APOL 1

- Strong association of FSGS with genes for **APOL 1**.
- **Patients with G1 and G2 alleles (G1G1,G1G2,G2G2)** have a multi-fold increase risk for developing FSGS and sclerosing glomerular diseases.
- A " genetic predisposition" not a dominant or recessive gene with all or none inheritance.
- APOL 1 alleles selected for by association with prevention of Trypanosoma brucei (sleeping sickness)!

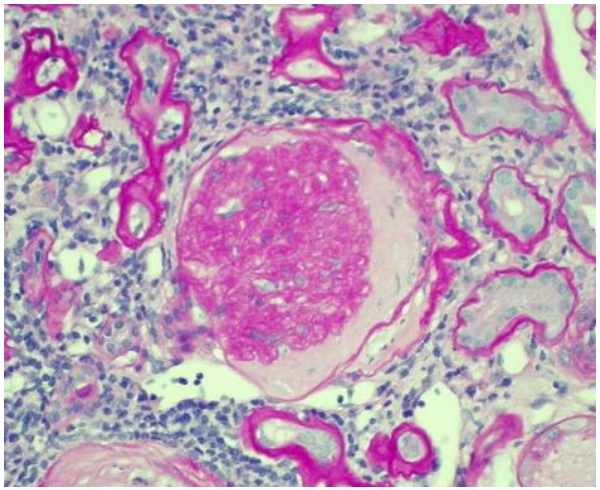




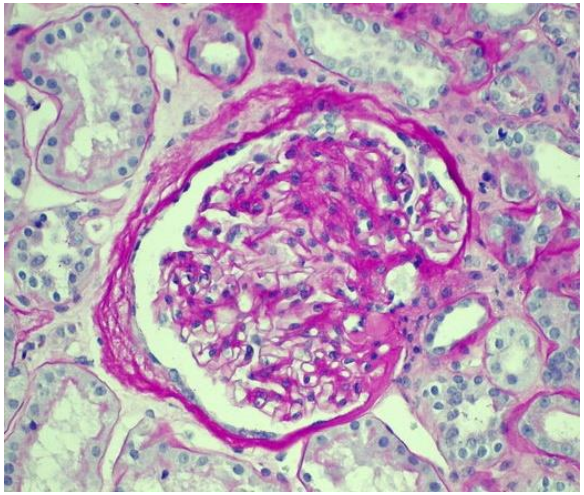
Genovese G, Tonna SJ, Knob A, Appel GB…Pollak MR. Kidney Int 78:698-704, 2010

Spectrum of *APOL1*-associated nephropathy

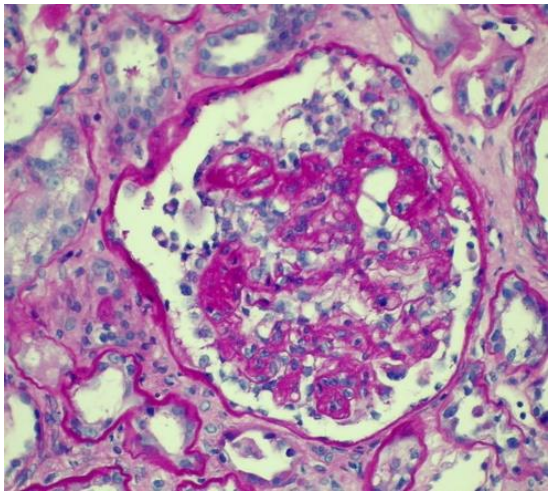
Focal Global Glomerulosclerosis
"Hypertension-attributed"



Focal Segmental Glomerulosclerosis



Collapsing FSGS (HIVAN) (COVAN)



Proteinuria & nephropathy progression rate

Severe lupus nephritis
Sickle cell nephropathy

Freedman, Bowden & Rich: Brenner and Rector's The Kidney 2012

CLINICAL RESEARCH www.jasn.org

Longitudinal Outcomes of COVID-19–Associated Collapsing Glomerulopathy and Other Podocytopathies

Satoru Kudose,[1] Dominick Santoriello,[1] Andrew S. Bomback,[2] Miroslav Sekulic,[1] Ibrahim Batal,[1] M. Barry Stokes,[1] Iman A. Ghavami,[2] Jung S. Kim,[2] Maddalena Marasa,[2] Katherine Xu,[2] Yonatan Peleg,[2] Jonathan Barasch,[2] Pietro Canetta,[2] Hila Milo Rasouly,[2] Ali G. Gharavi,[2] Glen S. Markowitz,[1] and Vivette D. D'Agati[1]

[1]Department of Pathology and Cell Biology, Columbia University Irving Medical Center, New York, New York
[2]Department of Medicine, Division of Nephrology, Columbia University Irving Medical Center, New York, New York

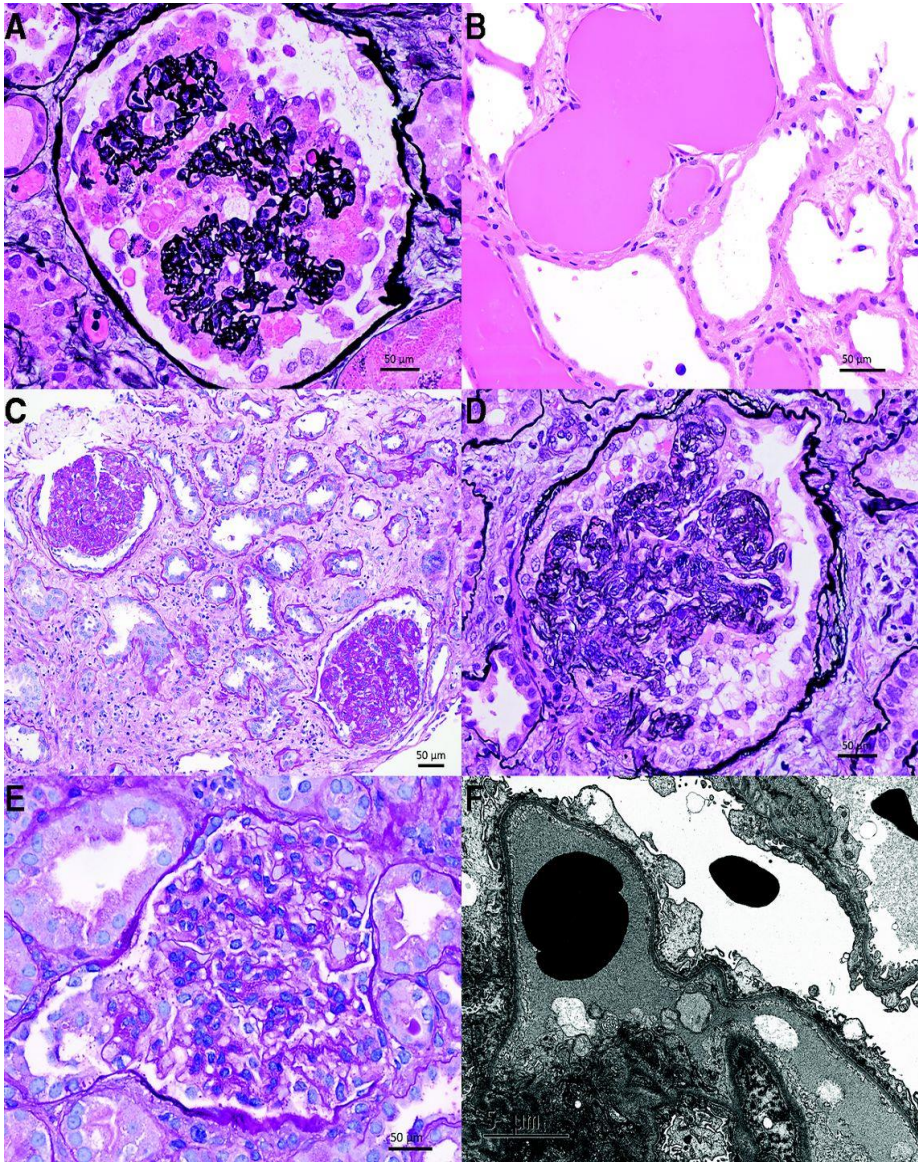
ABSTRACT

Background The long-term outcome of COVID-19–associated collapsing glomerulopathy is unknown.

Methods We retrospectively identified 76 native kidney biopsies from patients with history of COVID-19 between March 2020 and April 2021. Presenting and outcome data were obtained for all 23 patients with collapsing glomerulopathy and for seven patients with noncollapsing podocytopathies. We performed *APOL1* genotyping by Sanger sequencing, immunostaining for spike and nucleocapsid proteins, and *in situ* hybridization for SARS-CoV-2.

91% AA and 94% high risk APOL1

moderate in most (77%) patients. All but one patient presented with AKI, 17 had nephrotic-range proteinuria, and six had nephrotic syndrome. Fourteen (61%) patients required dialysis at presentation. Among 17 patients genotyped, 16 (94%) were high-risk *APOL1*. Among 22 (96%) patients with median follow-up at 155 days (range, 30–412), 11 (50%) received treatment for COVID-19, and eight (36%) received glucocorti-

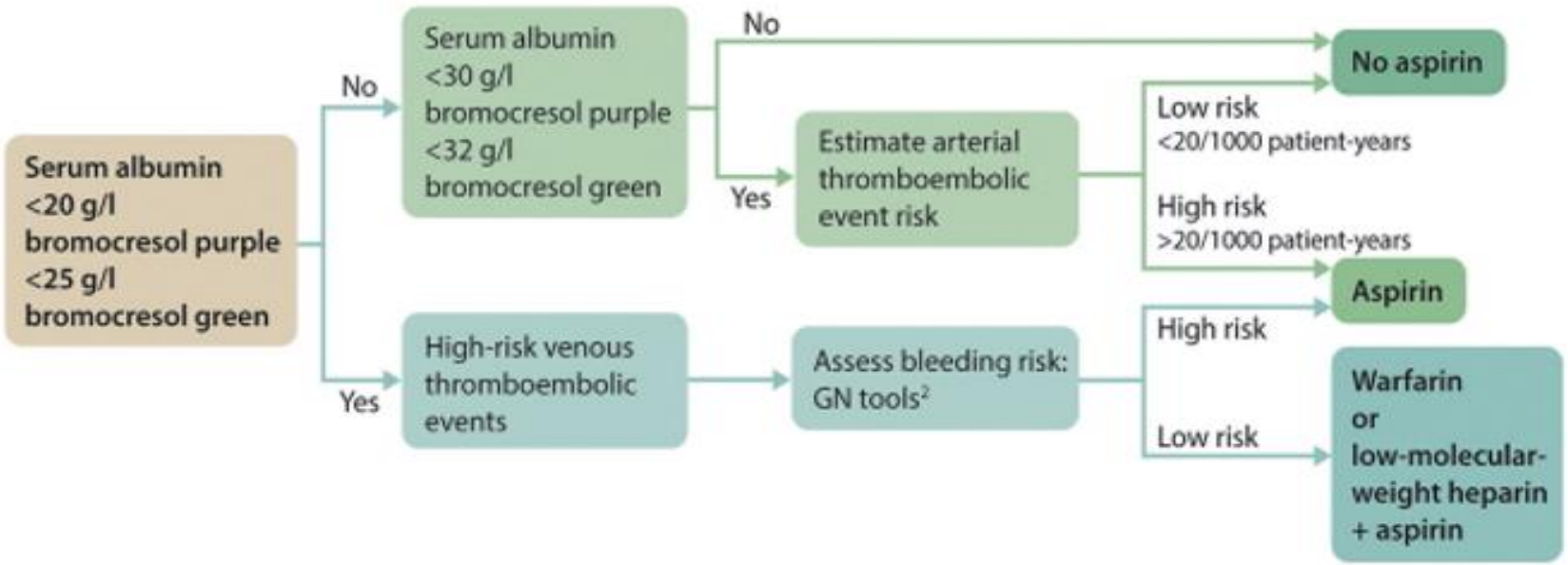


Question 8

- A 64 yo M with 12 g proteinuria daily and serum albumin of 3.2 g/dl and eGFR 28 cc/min has a bx shows MN with + anti-PLA2R staining. Which is true about thromboses in MN.

1) Anticoagulation will not be beneficial unless the Salb is < 2.0 g/dl.
2) Data supports prophylactic oral NOACs over warfarin to prevent thrombosis.
3) Anticoagulation has not proven beneficial in MN patients who are PLA2R antibody positive.
4) Anticoagulation should be based on an individual risk/benefit strategy

KDIGO and Up to Date – Guide to Anticoagulation in MN



UpToDate

We do not routinely prescribe direct oral anticoagulants (DOACs) as prophylactic anticoagulation in MN, given limited data. May consider DOACs in pts with side effects or inadequate therapeutic effects from warfarin and if the patient is unwilling to take low-molecular-weight hepari

UpToDate

Assess risk of thrombosis and of bleeding (http://www.med.unc.edu/gntools)

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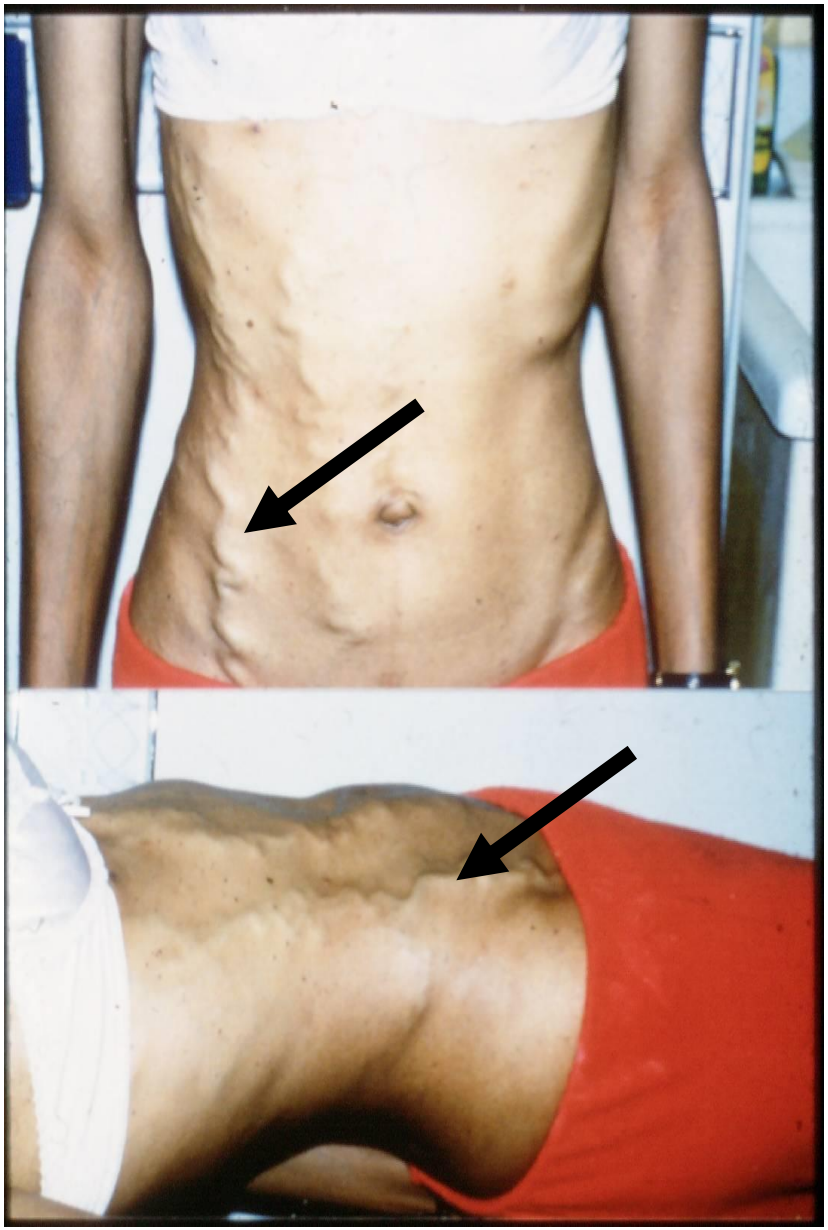
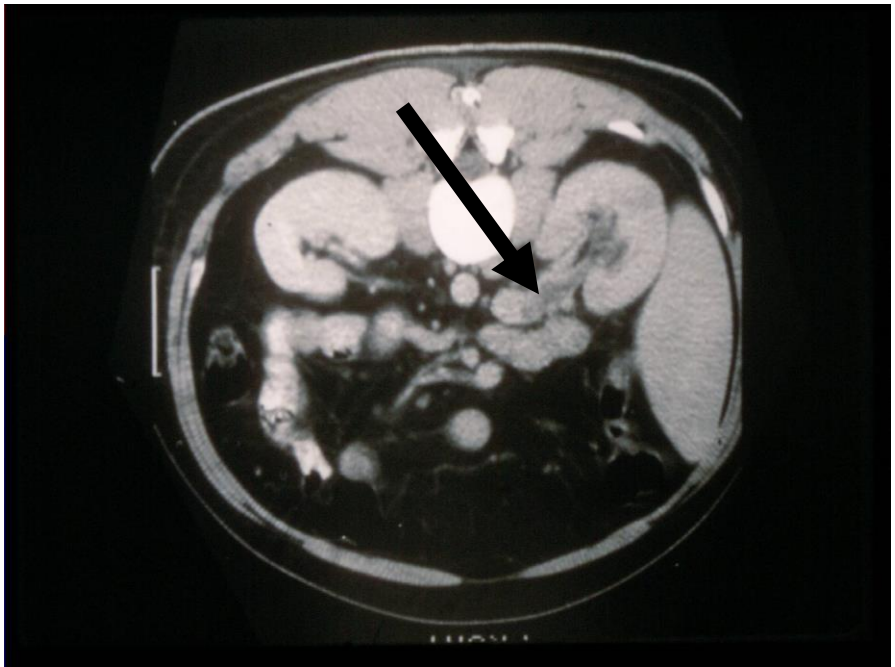
Renal Vein Thrombosis in MN





Question 9 -Auto-Antigens in Membranous Nephropathy

Which "autoantigen(s)" in biopsies of patients with MN have been associated with tumors?

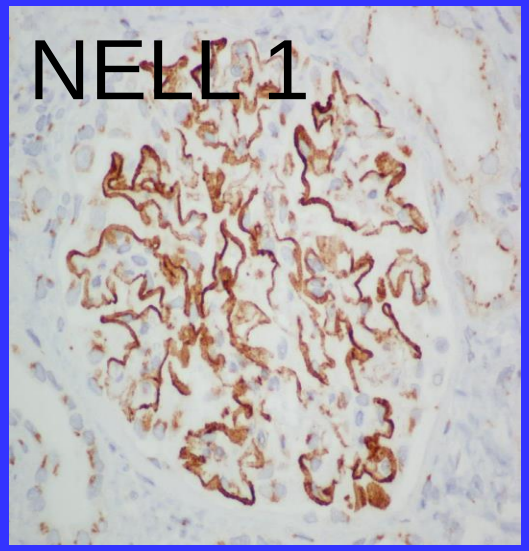
1) SEMA3B
2) Exostosin 1 and 2
3) NELL 1
4) PLA2r
5) THSD7A

Ahmad S, Appel GB. Antigens, antibodies, and membranous nephropathy: a decade of progress Kidney Int: 97: 29–31, 2020.

NELL1-associated MN

Table 3 | Increased prevalence of malignancy in NELL1-associated MN compared with PLA2R-associated MN, THSD7A-associated MN, and MN due to unknown antigens[a]

Type of neoplasm	PLA2R-positive (n = 35 of 829)	THSD7A-positive (n = 4 of 37)	NELL1-positive (n = 30 of 91)	Unknown antigen (n = 42 of 421)
Prostate adenocarcinoma	10	3	6	10
Breast carcinoma	12	1	5	8
Gastric carcinoma	0	0	1	2
Soft-tissue tumor	0	0	1	0
Glioma	0	0	1	0
Colon adenocarcinoma	1	0	1	6
Lung carcinoma	1	0	3	4
Bladder carcinoma	5	0	2	0
Renal cell carcinoma	2	1	3	5
Pancreatic carcinoma	0	0	0	1
Hepatocellular carcinoma	0	0	0	1
Thyroid carcinoma	0	0	1	2
Ovarian carcinoma	0	0	1	0
Uterine carcinoma	0	0	0	1
Cervical carcinoma	1	0	1	0
Testicular carcinoma	1	0	0	0
Skin (melanoma, BCC, SCC)	2	0	4	1
SCC (head/neck)	0	1	1	2
Laryngeal carcinoma	0	0	0	1
Nasopharyngeal carcinoma	0	0	1	0
Thymoma	0	0	1	0
Lymphoma	0	0	1	1
Total	35	6[b]	34[c]	45[d]

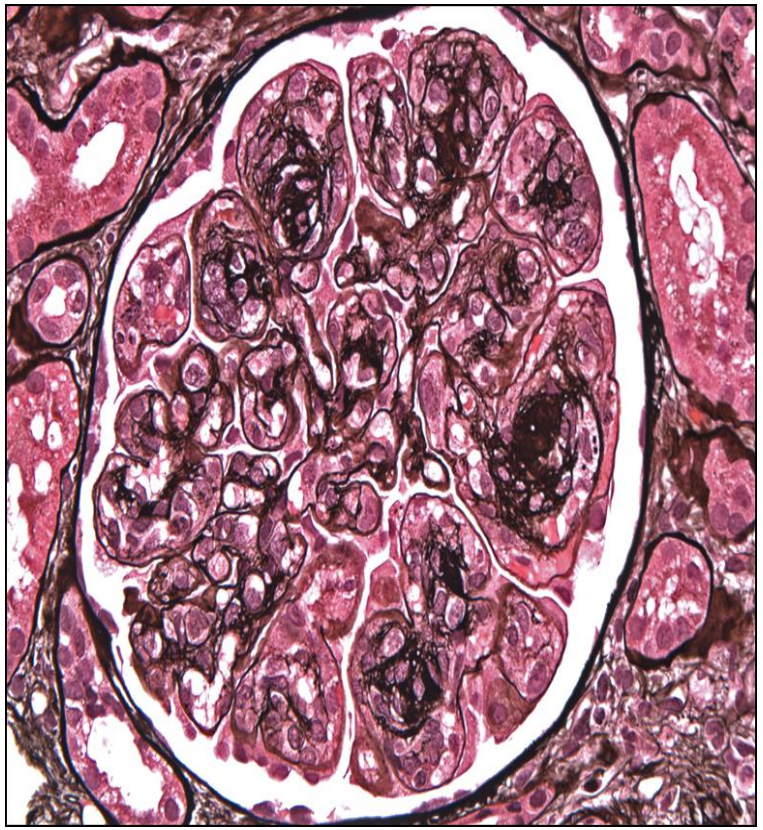


4.2% 11% 33%

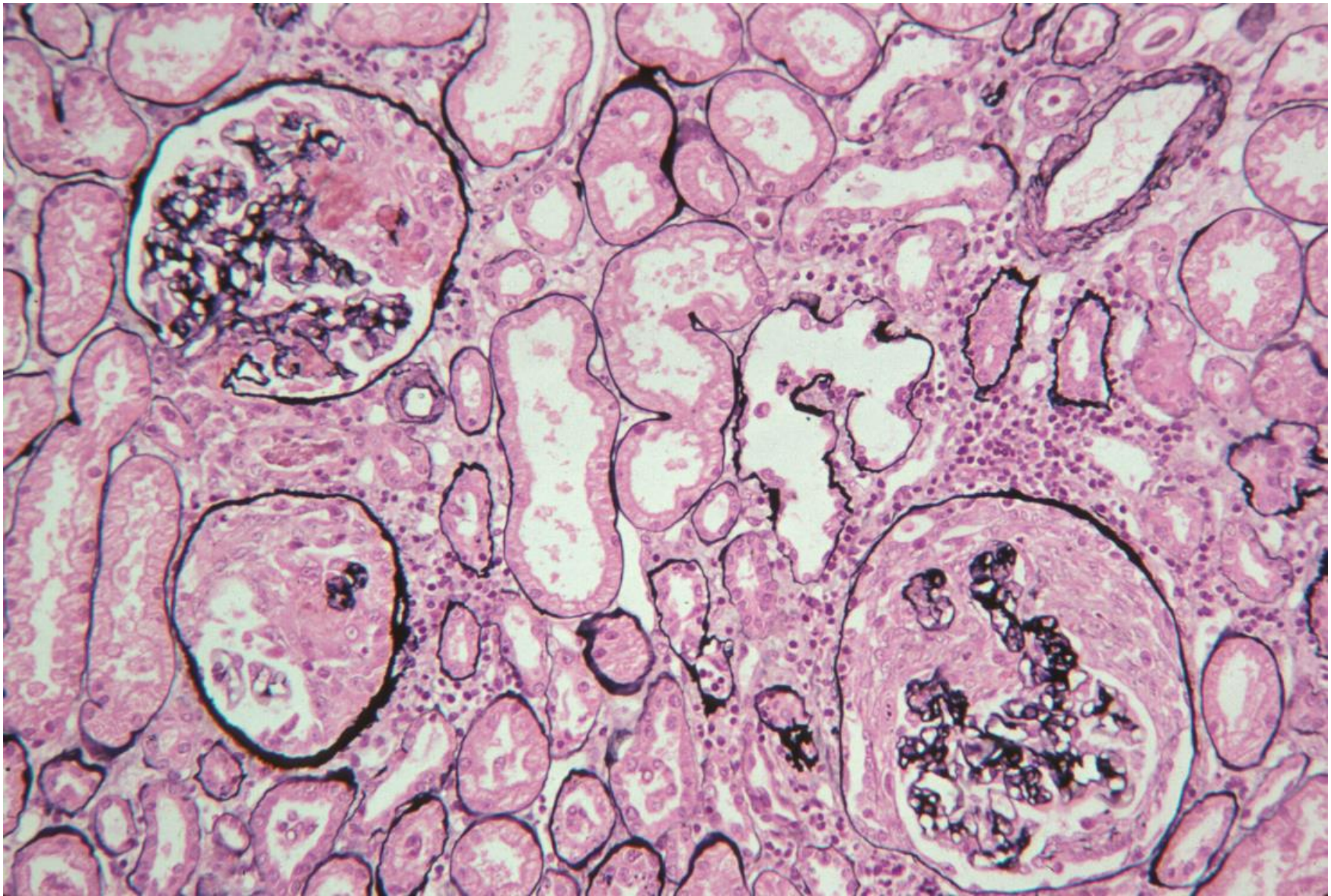
Caza et al NELL1 is a target antigen in Malignancy –assoc MN Kid Int (2021) 99:967-976

Question 10 - Which test is usually NOT part of the evaluation of a patient whose kidney biopsy shows the following Pattern?

1) HCV and HBV serology
2) S PEP , U PEP, Free-Lyte
3) ANA and complement level
4) Anti GBM antibody titer



Anti GBM Dis = A Crescentic GN

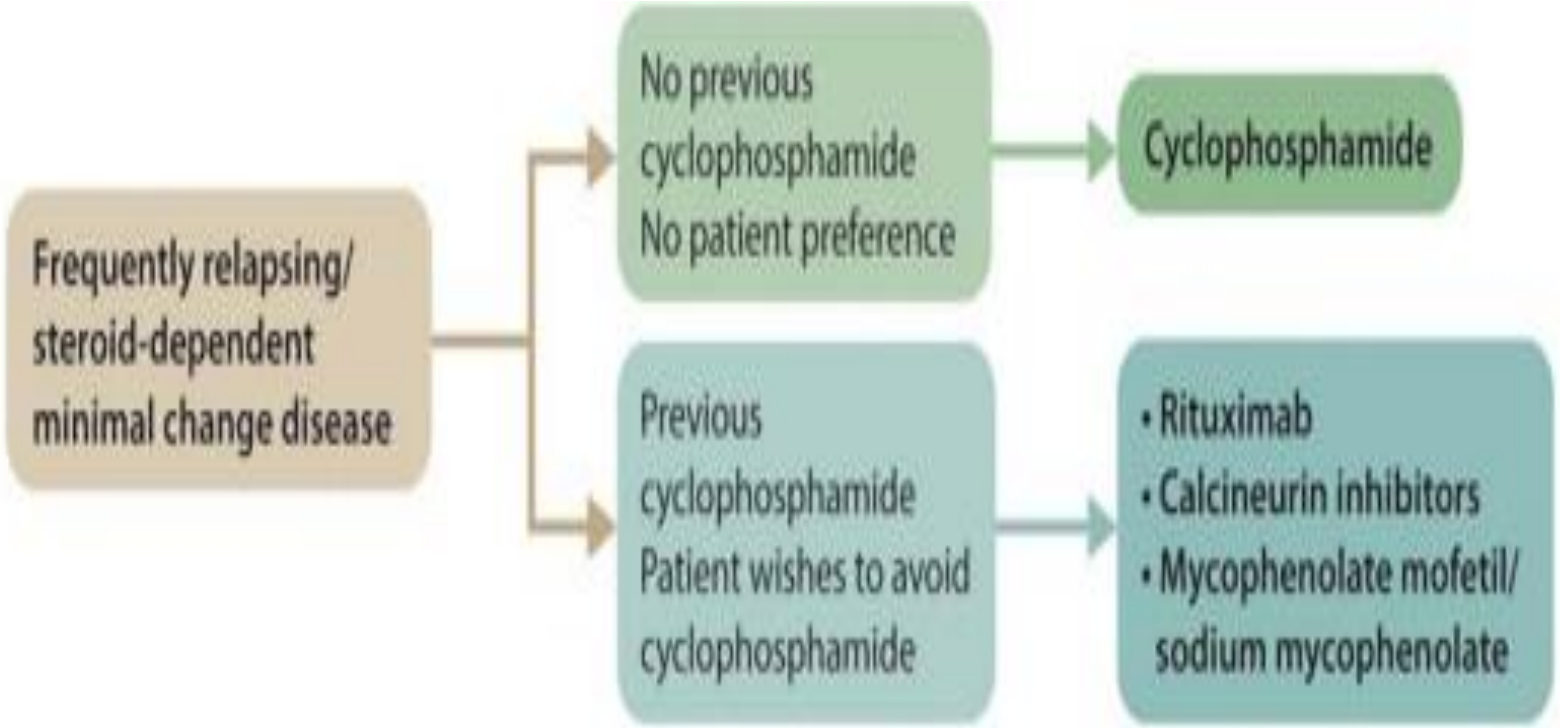


Question 11

For which glomerular disease(s) has rituximab NOT been recommended as a first line therapy?

1) **Frequent relapsing or steroid resistant MCD**
2) **ANCA + vasculitis.**
3) **IgA Nephropathy.**
4) **Membranous Nephropathy**

KDIGO – Therapy of FR/Steroid Dependent MCD in Adults



KDIGO Executive Summary 2021 Guidelines for management of Glomerular Disease. Kidney Int. 100: 753-779, 2021

RAVE TRIAL

N=197
1–3 (1000-mg) IV pulses methylprednisolone

‡

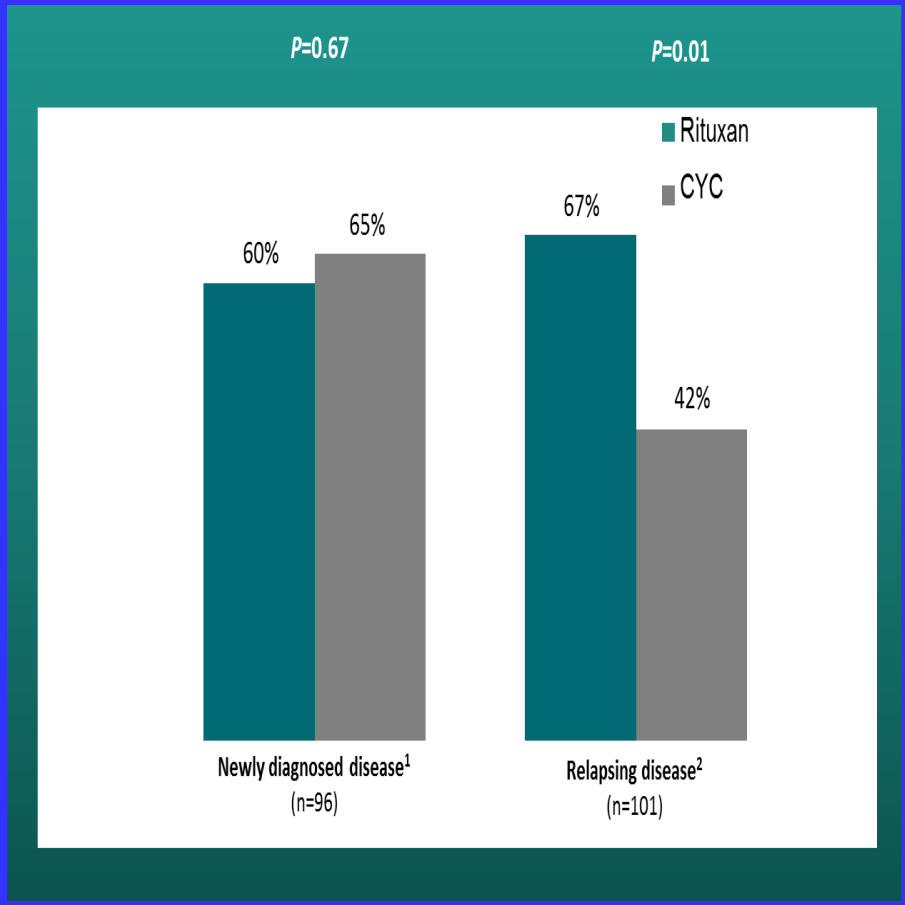
Rituximab Group (n=99)
Ritux IV (375 mg/m^2 weekly × 4)
Oral CYC-placebo daily for 3–6 months

Patients in Remission
AZA[§]-placebo (daily up to 18 months)

CYCLOPHOS Group (n=98)
Ritux-placebo IV
Oral CYC (2 mg/kg) daily[†] for 3–6 months

Patients in Remission
AZA
(2 mg/kg daily up to month18)

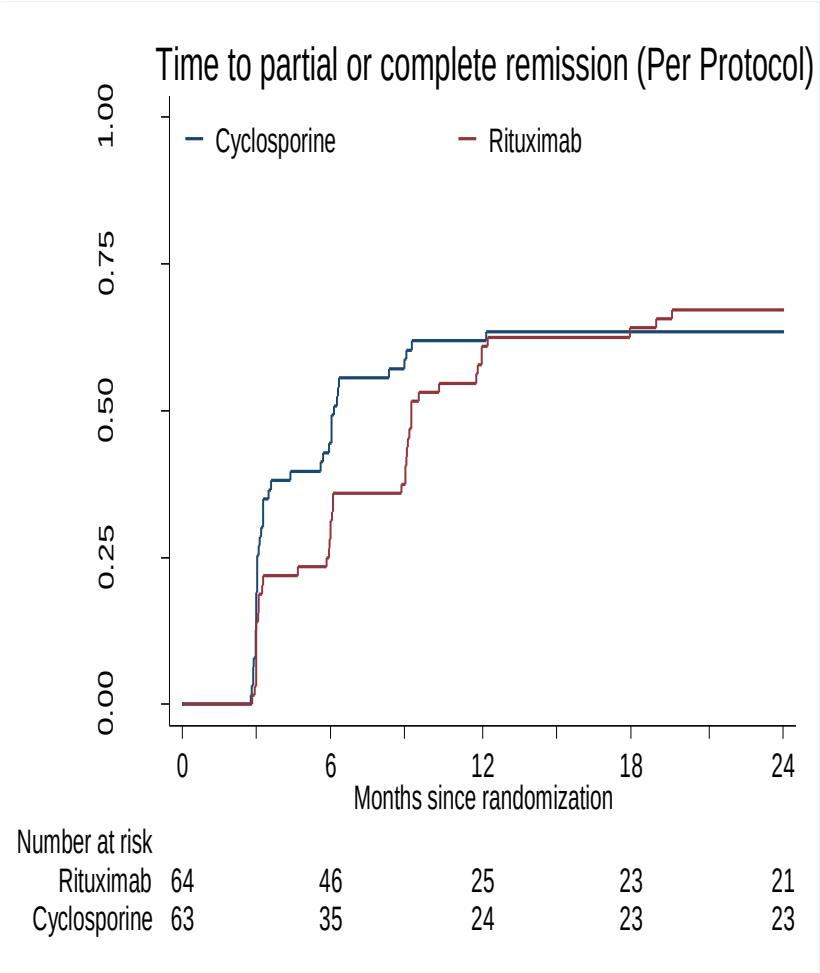
Complete remissions by 6 mo

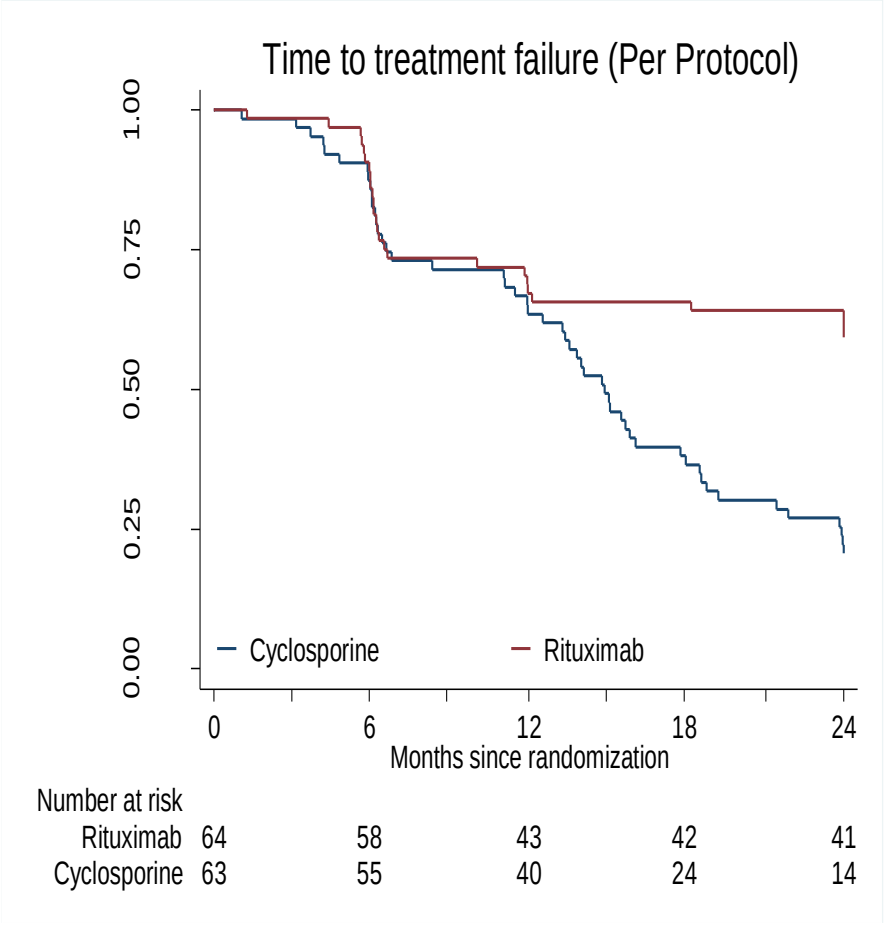


Stone J et al. N Engl J Med. 2010;363:221-232. 18 month data NEJM 2012

Rituximab or Cyclosporine Treatment of Membranous Nephropathy

Fervenza F, Appel GB, Barbour SJ et al , NEJM 381:36-46, 2109





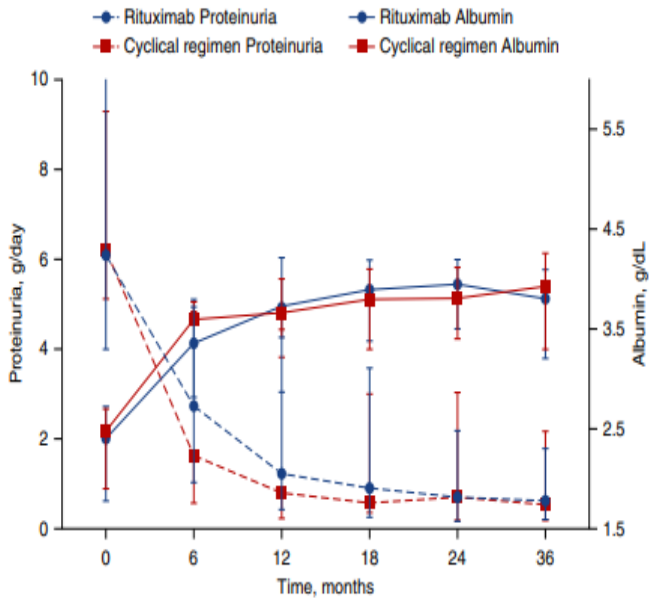
Rituximab or Cyclophosphamide in the Treatment of Membranous Nephropathy: The RI-CYCLO Randomized Trial

Francesco Scolari,[1,2] Elisa Delbarba,[2] Domenico Santoro,[3] Loreto Gesualdo,[4] Antonello Pani,[5] Nadia Dallera,[6] Laila-Yasmin Mani,[7] Marisa Santostefano,[8] Sandro Feriozzi,[9] Marco Quaglia,[10] Giuliano Boscutti,[11] Angelo Ferrantelli,[12] Carmelita Marcantoni,[13] Patrizia Passerini,[14] Riccardo Magistroni,[15] Federico Alberici,[1,2] Gian Marco Ghiggeri,[16] Claudio Ponticelli,[17] and Pietro Ravani,[18] for the RI-CYCLO Investigators*

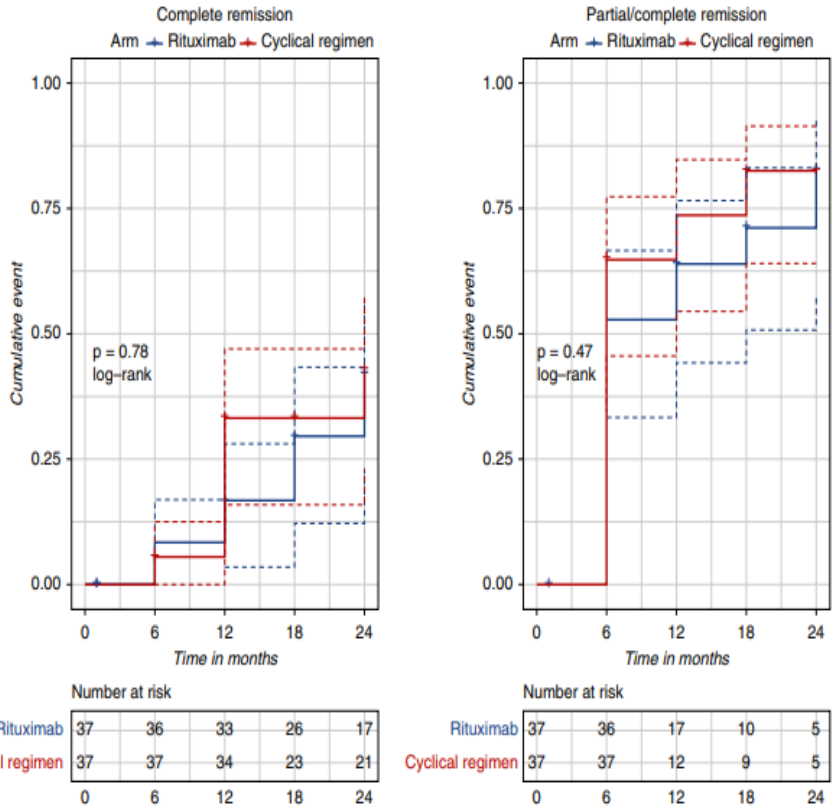
Due to the number of contributing authors, the affiliations are listed at the end of this article.

ABSTRACT

Background A cyclic corticosteroid-cyclophosphamide regimen is the first-line therapy for membrano nephropathy. Compared with this regimen, rituximab therapy might have a more favorable safety profil but a head-to-head comparison is lacking



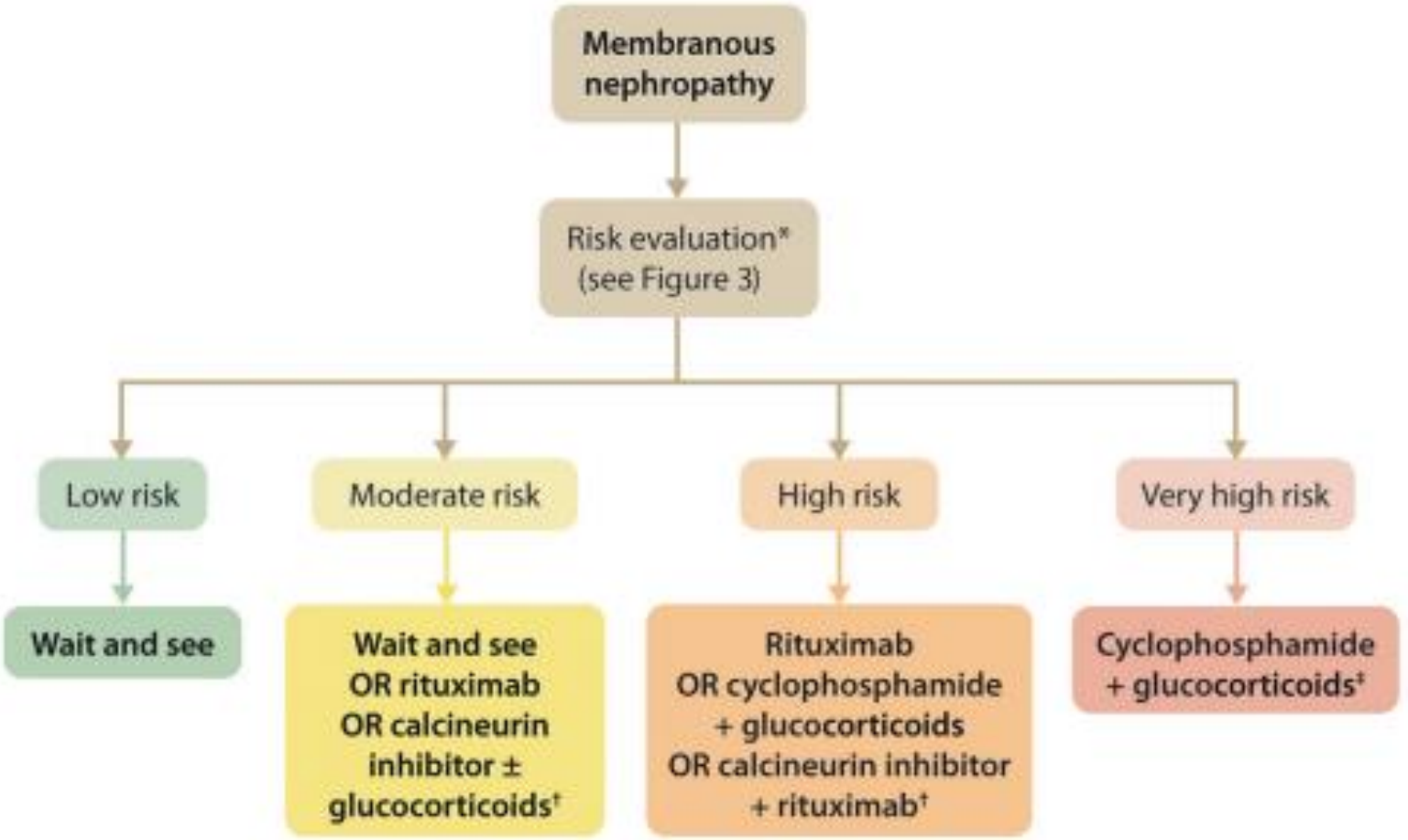
gure 3. Proteinuria and serum albumin over time. Data are presented as median (IQR) over time, by assigned treatment.



This pilot trial found no signal of more benefit or harm w ritux vs cyclic corticosteroid-cyclophosphamide in MN.

Scolari F, et al RI-Cyclo Investigators JASN 32: 972-982 2021

KDIGO Guidelines for the Treatment of MN



KDIGO Exec Summary 2021 Guidelines for management of Glomerular Disease. Kidney Int. 100: 753-779, 2021

Question 12

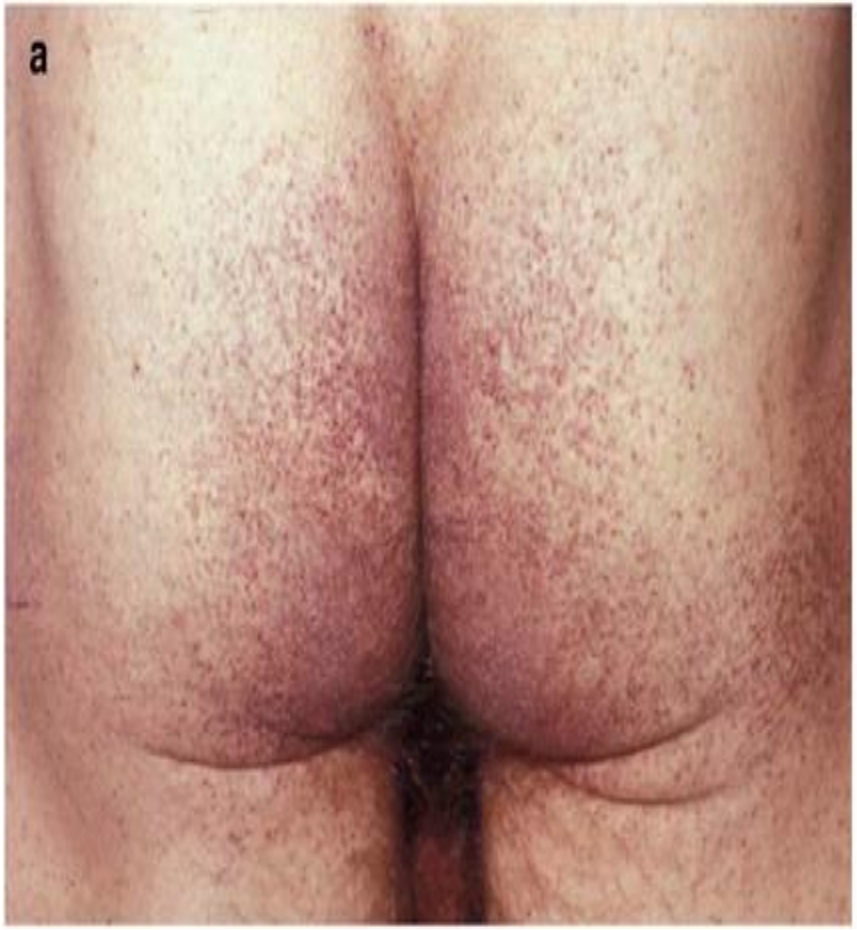
You are discussing the current treatments options fo IgA Nephropathy with a patient.

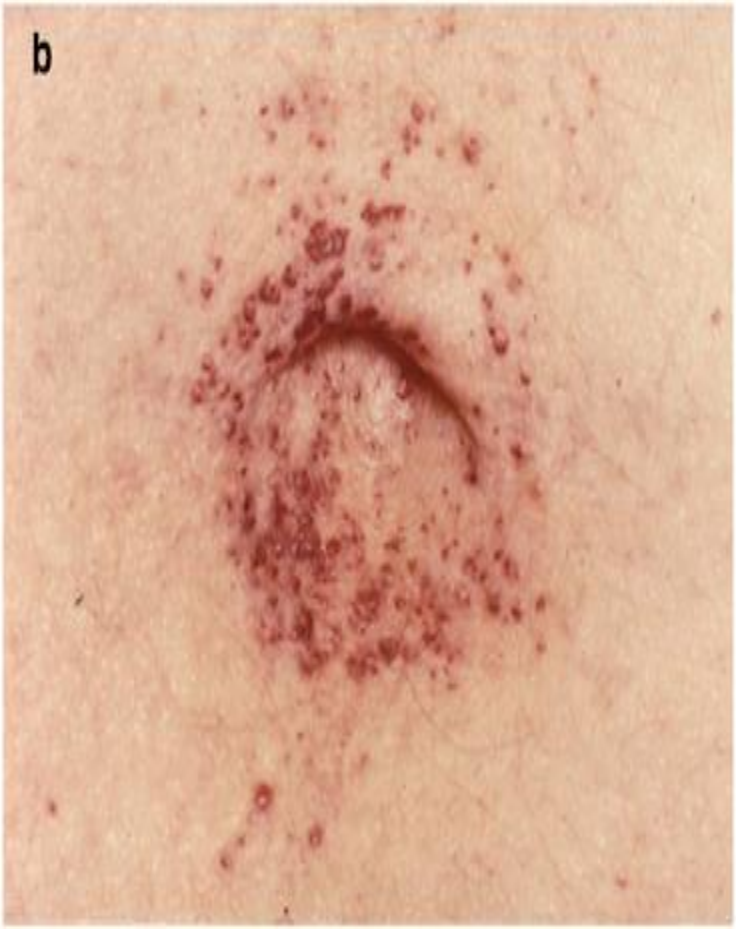
Which agents are FDA approved specifically to treat IgAN?

1) Sparsentan, Atrasentan, and Mycophenolate
2) Obinituzumab , Iptacopan, and Mycophenolate
3) Targeted Release budesonide, Iptacopan, and Atrasentan
4) Targeted release budesonide, Sparsentan and Mycophenolate

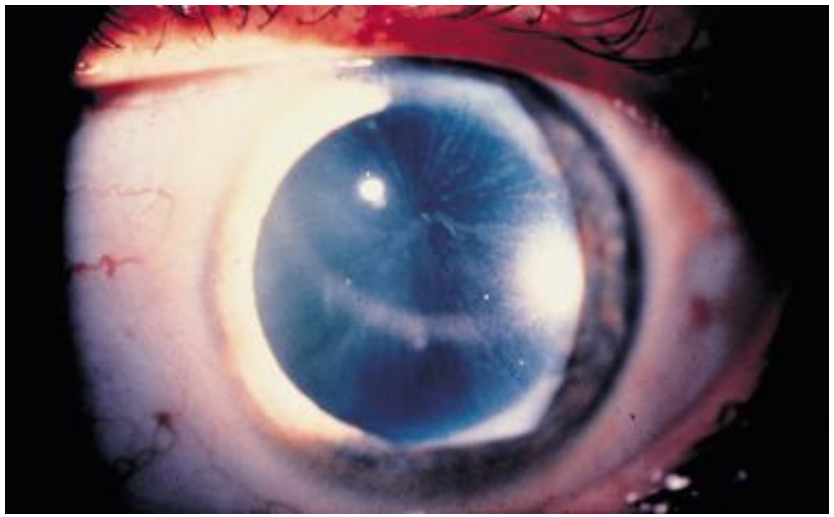
Question 13

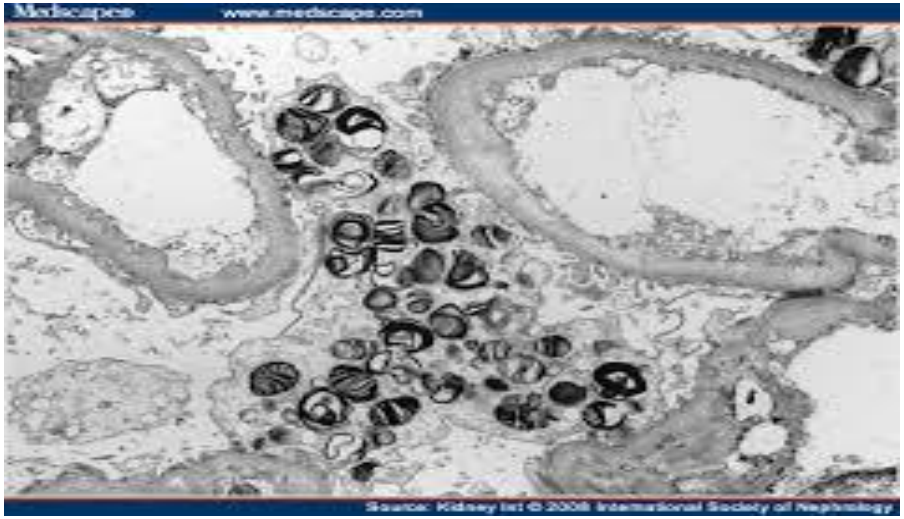
22 yo M is seen with a creatinine of 1.8 mg/dl and 2.4 gram proteinuria/day . On physical exmination he has the following skin changes.

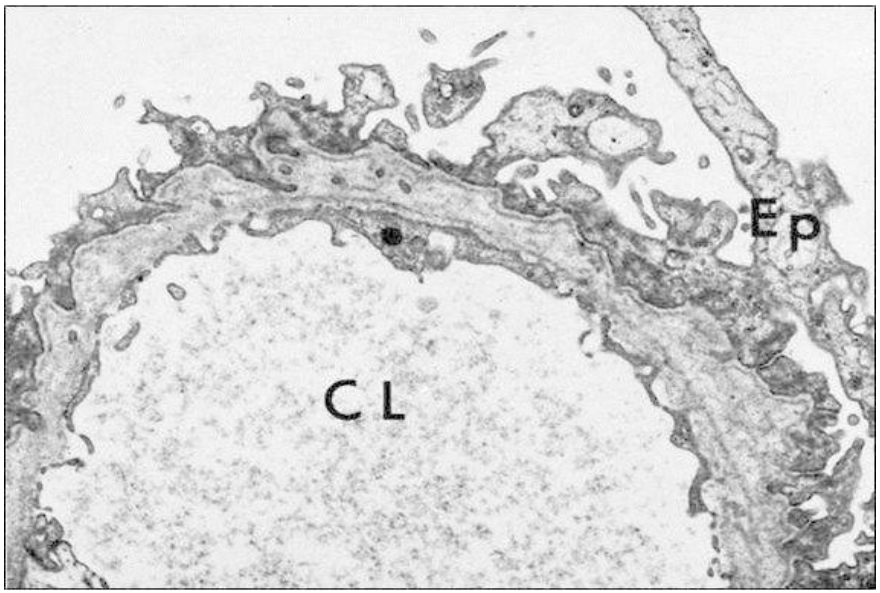




A 22 yo M creatinine of 1.8 mg/dl and 2.4 g/d prot.
Which would **NOT** be typical findings in this patient?

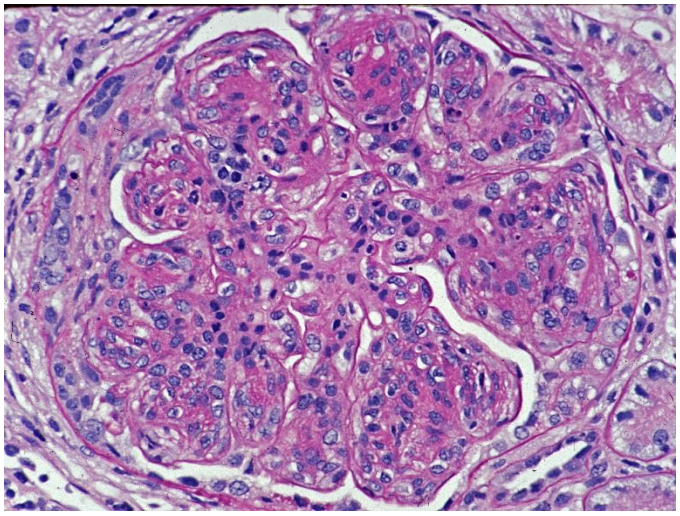






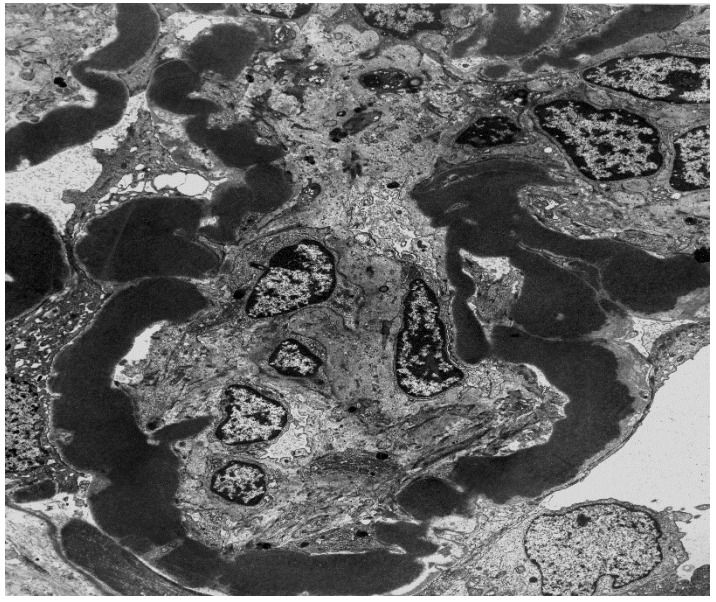
Question 14

A 59 yo M with a 2 yr history of proteinuria and hematuria has this biopsy.



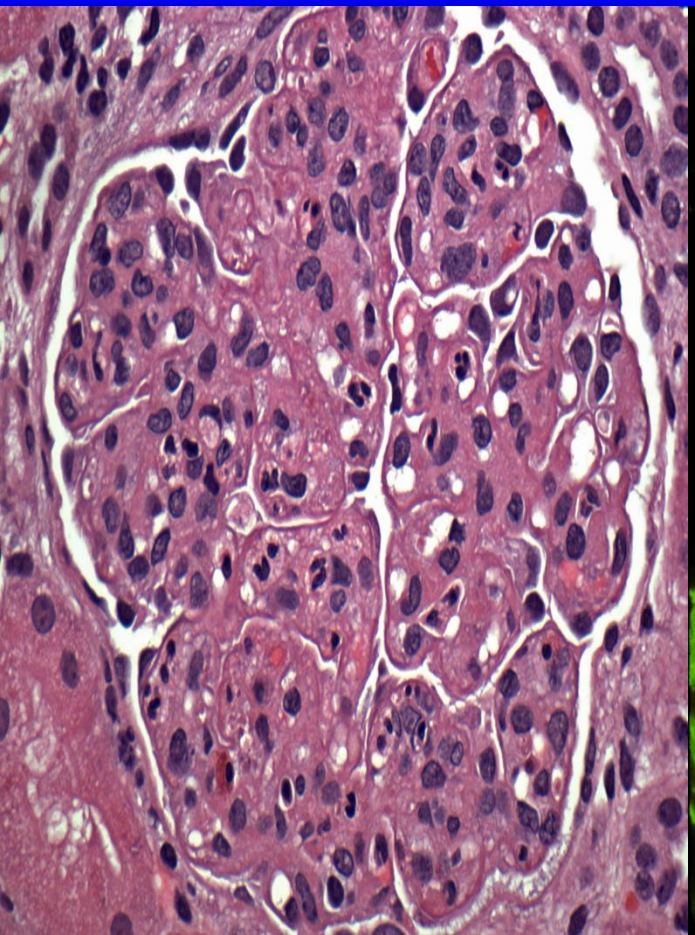
Which is NOT likely to be found with this lesion?

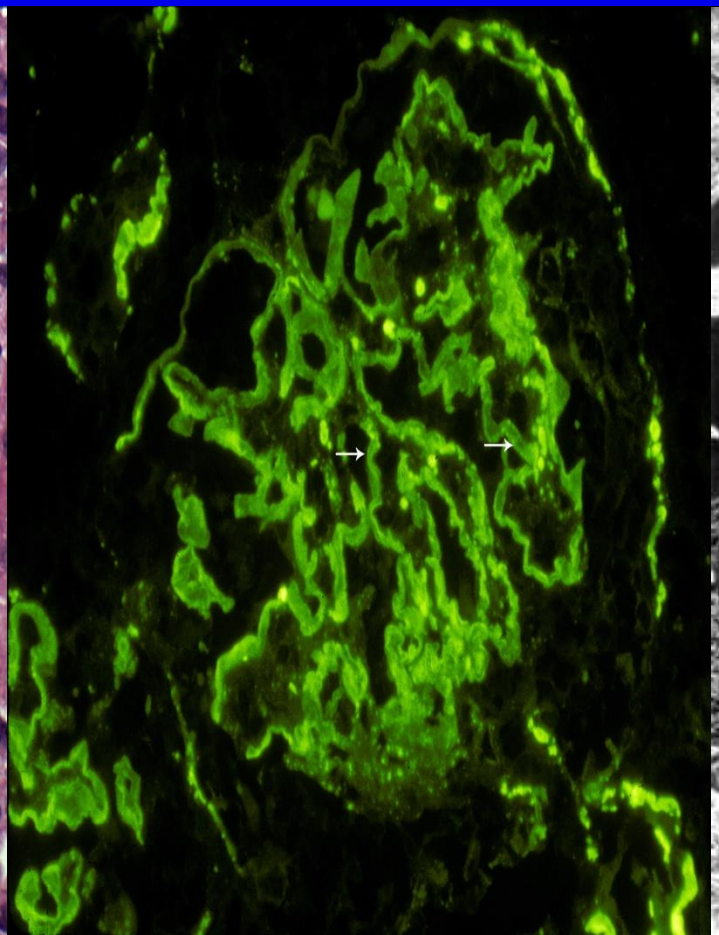
1) **+SPEP and + UPEP**
2) **+C3 Nephritic Factor**
3) **Low C3 level**
4) **+anti-PLA2R**

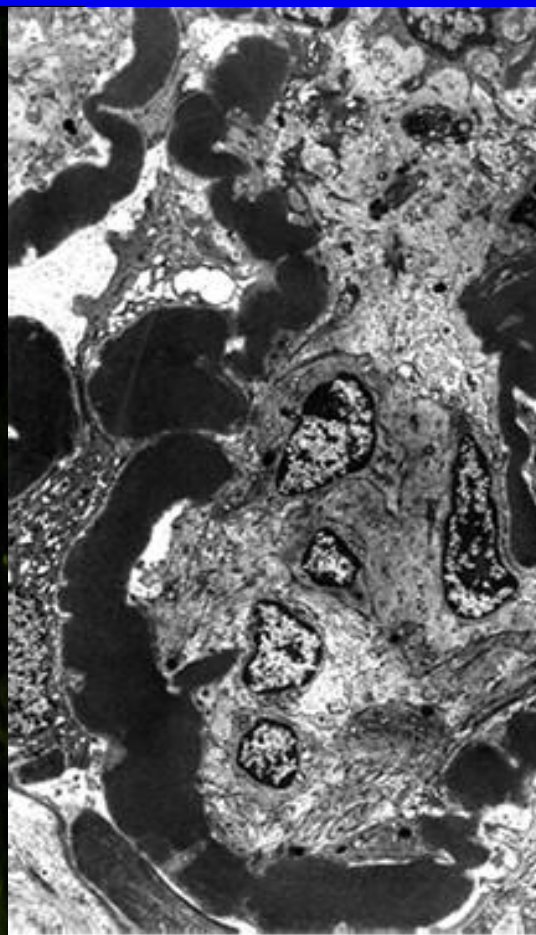


Dense Deposit Disease

LM - MPGN IF C3 EM GBM

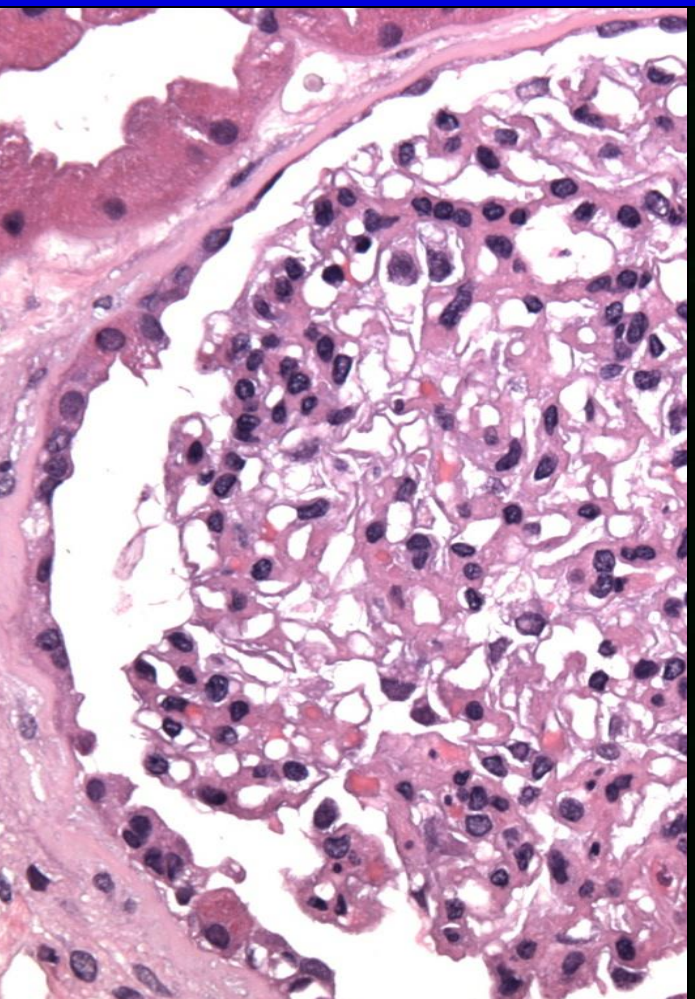


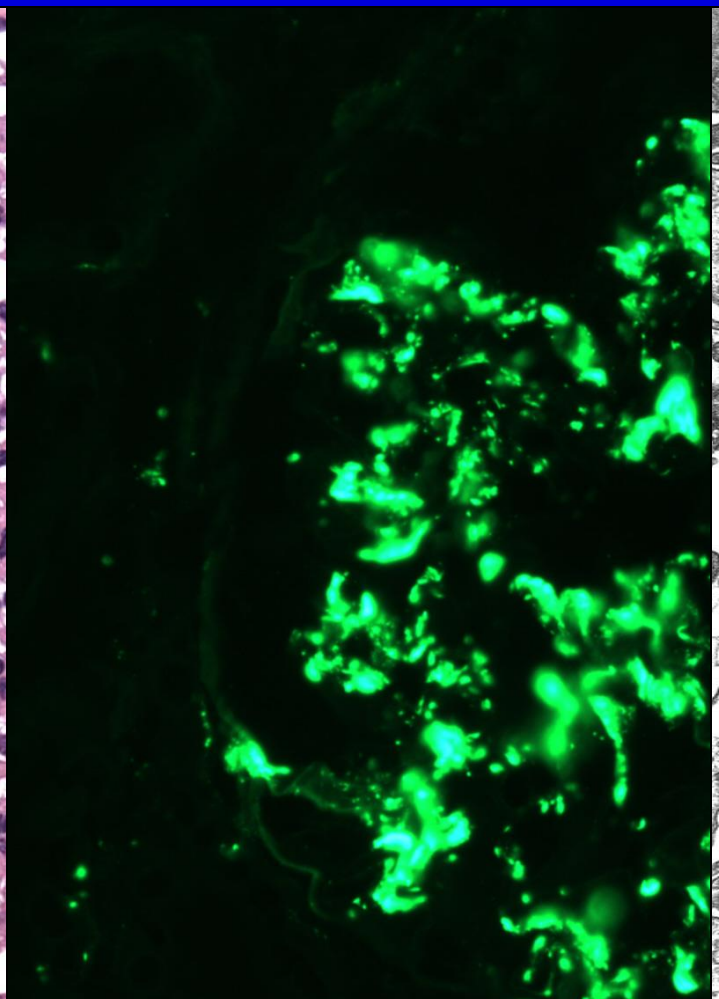


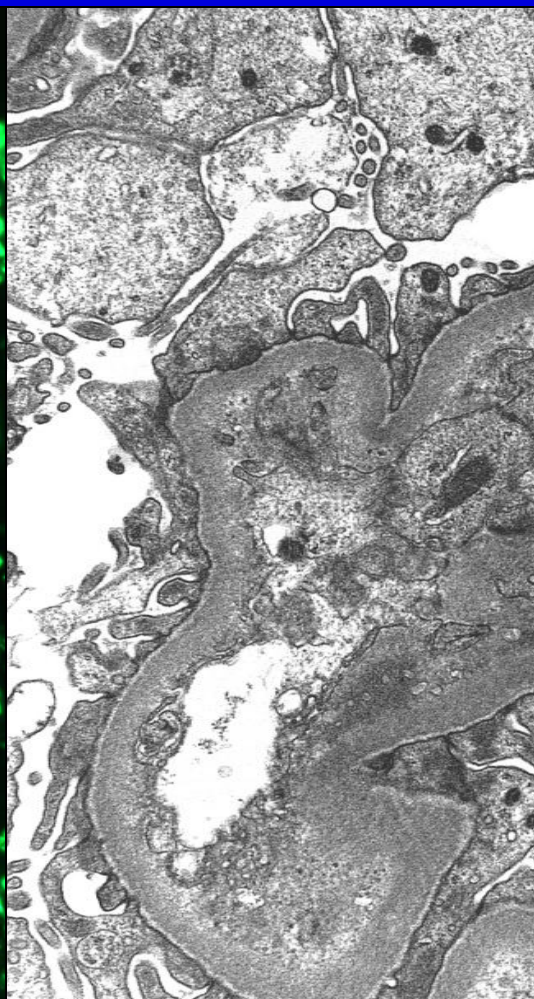


C 3 Glomerulonephritis

LM IF C3 EM GBM







Complement Pathway

Classical

Alternative

C1

C3

ALWAYS ON AT LOW RATE

C3 convertase

C3

C3b

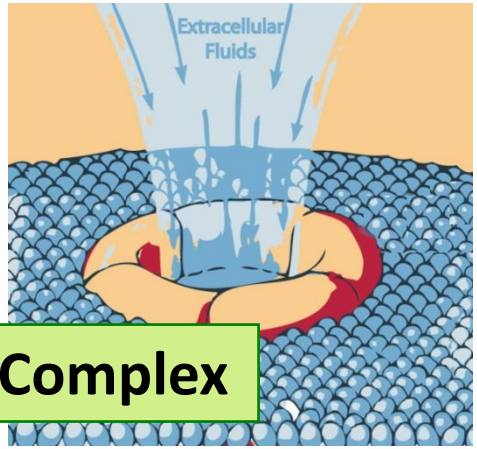
C3a

C5

C5 convertase

C5b

C5a



Membrane Attack Complex

C3 Glomerulopathy with monoclonal Ig is a distinct subtype

RavindranA , Fervenza F, Smith R, Sethi S. Kidney Int 94: 178-186, 2018

36 C3G with monoclonal spikes vs 59 w/o monoclonal

Table 1 | Clinical and laboratory findings

Data variable	C3G with monoclonal Ig, *N* = 36	C3G without monoclonal Ig, *N* = 59
Age at diagnosis (yr)		n: 28
Sex (M/F)		4–84 (47.5%) (52.5%)
Race		(78.0%) (1.7%) (3.4%) laskan Native: 0
Ethnicity		(5.1%) 7 (11.9%) Latino: 0 atino: 51 (86.4%) 3 (13.6%)
Blood urea nitrogen (mg/c		n: 18
Serum creatinine at diagn		5.5–72 n: 1.3
Serum albumin at diagnos		.3–7.9 n: 3.5
Hematuria		.2–4.5 (86.4%)
Proteinuria (g/24 h)		3 (13.6%) n: 1.7 .3–24.2
Immunology		
Anti-GBM antibody		tive: 21; NA: 37
ANCA/PR-3/MPO		tive: 46; NA: 13
Antistreptolysin		tive: 15; NA: 42
Anti-DNase B		tive: 6; NA: 53
ANA		tive: 50; NA: 2
Anti-ds DNA		tive: 43; NA: 12
Cryoglobulins		tive: 36; NA:22
Cryofibrinogen		tive: 35; NA: 24
Complement		
C3		al: 30; NA: 1

Average age 60 yo, 70% Male.
Mean creatinine 1.9 mg/dl,
89% hematuria on U/A
24 Hr protein 3.0 g/day,

1/3 low C3 level

Most MGRS (77%) some MM (14%), smoldering MM (6%), CLL (6%) , cryos(3%).

APPEAR-C3G trial: How effective is iptacopan in treating patients with C3G?

KidneyNews

Phase 3 trial

- Randomized, multicenter, double-blind study
- Age >18 years
  N = 74
- Patients with biopsy-proven C3 glomerulopathy (C3G)

Intervention

Results

58.1 % Completed 12 months' treatment at data cutoff.

In the iptacopan arm

- **Statistically significant reduction in 24-hour UPCR at 6 months, sustained up to 12 months**
  35.1 % (one-sided; p = 0.0014; 95% CI:13.8%–51.1%)
- **Sustained improvement in composite renal endpoint**
  (≥50% reduction UPCR + ≤15% reduction in eGFR at 12 months)
  43.5% (iptacopan versus placebo); **25.0%** (switched to iptacopan)
- **Improvement in eGFR trajectory compared with historical eGFR decline**
- **Favorable safety profile**

CI, confidence interval.

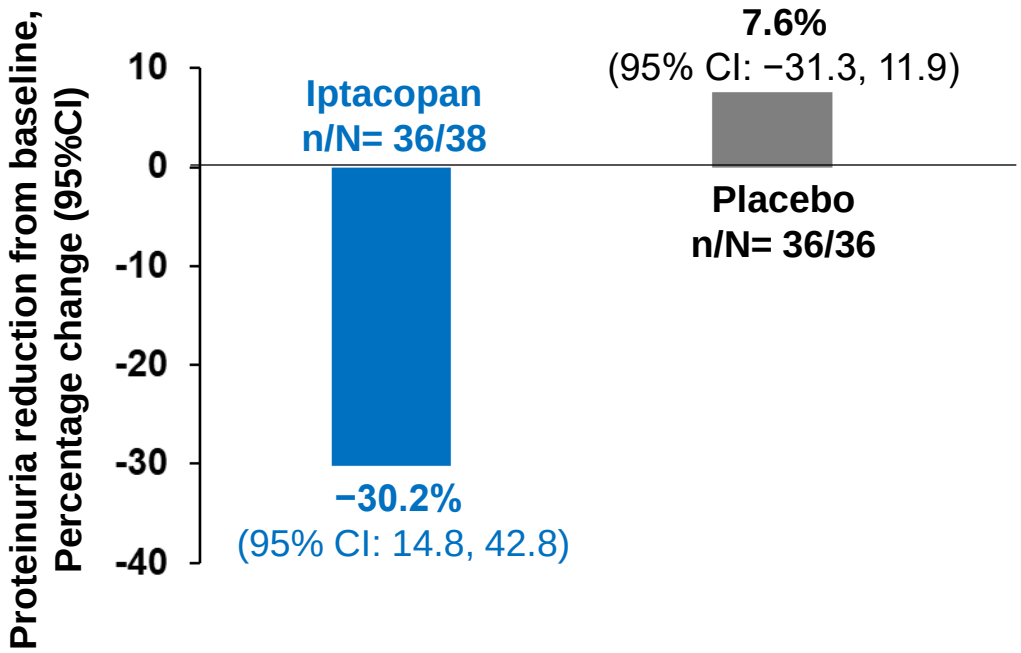
Conclusions: Iptacopan demonstrated a significant and clinically meaningful proteinuria reduction on top of supportive care at 6 months and sustained up to 12 months in patients with C3G and was well tolerated with a favorable safety profile.

Nester CM, et al. **Efficacy and Safety of Iptacopan in Patients With C3 Glomerulopathy: 12-Month Results From the Phase 3 APPEAR-C3G Study [Abstract].** *J Am Soc Nephrol* 2024; 35(10S):SA-OR66. doi: 10.1681/ASN.2024f5gka890

Visual abstract by Krithika Mohan, MD, DNB @krithicism

PRIMARY ENDPOINT

Iptacopan achieved a statistically significant and clinically meaningful reduction in 24h-UPCR at Month 6

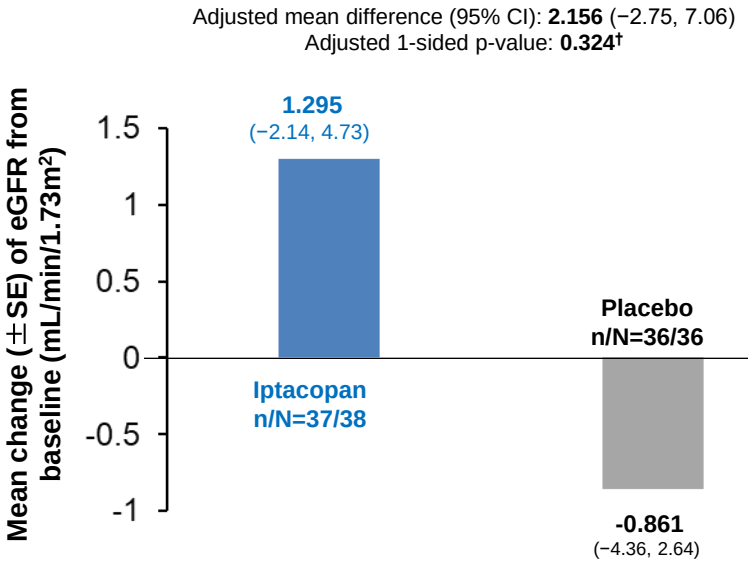


Relative percent reduction between iptacopan and placebo at Month 6 (95% CI): **35.1%;** 1-sided p-value: **0.0014**

Full analysis set. Note that there was one intercurrent event of increasing corticosteroid dose on Day 106 in the iptacopan group Robustness of primary endpoint results demonstrated by consistency of results across sensitivity analyses. CI, confidence interval; N, number of all participants included in the analysis (with non-missing baseline and covariates); n, number of participants with values non-missing and not imputed as per intercurrent event handling strategy at designate visit; UPCR, urine protein–creatinine ratio.

SECONDARY ENDPOINT

A numerical improvement in eGFR was observed in the iptacopan compared to placebo arm at 6 Months

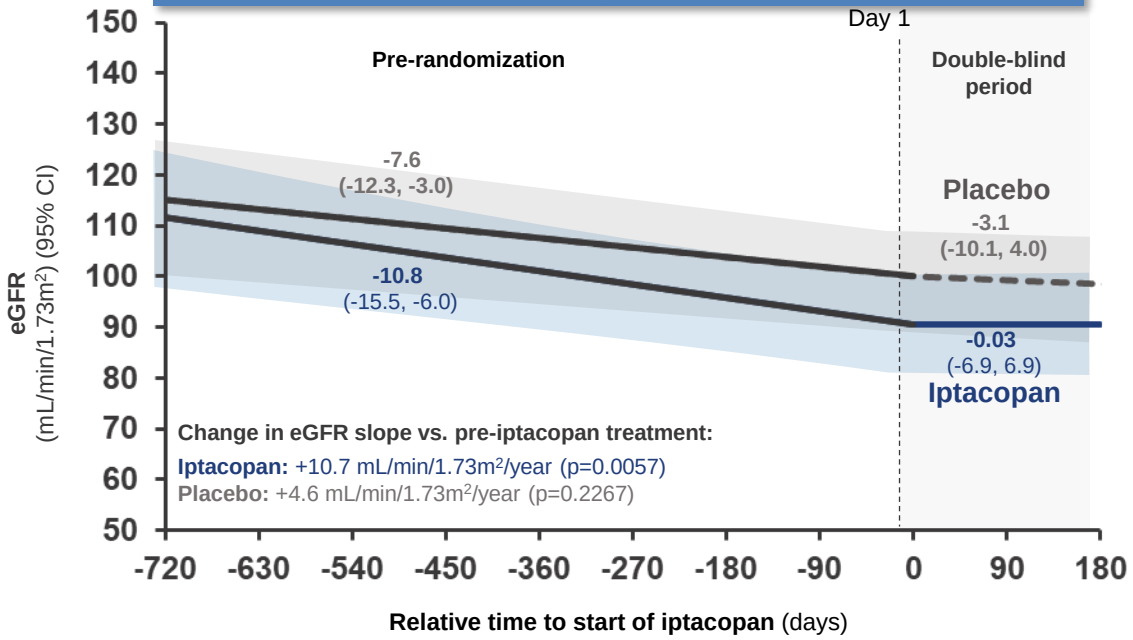


- The improvement in eGFR with iptacopan treatment was observed as early as Day 30 and continued up to 6 months compared to placebo arm

†Adjusted for multiplicity. Numerical improvement; not statistically significant
b.i.d; twice daily; CI, confidence interval; eGFR, estimated glomerular filtration rate' ; N, number of all participants included in the analysis (with non-missing baseline and covariates); n, number of participants with observed values (non-missing and not imputed as per intercurrent event handling strategy at designate visit); SE, standard error;

Exploratory endpoint

eGFR slope by treatment group pre- and post- Iptacopan treatment initiation up to 6 months



References

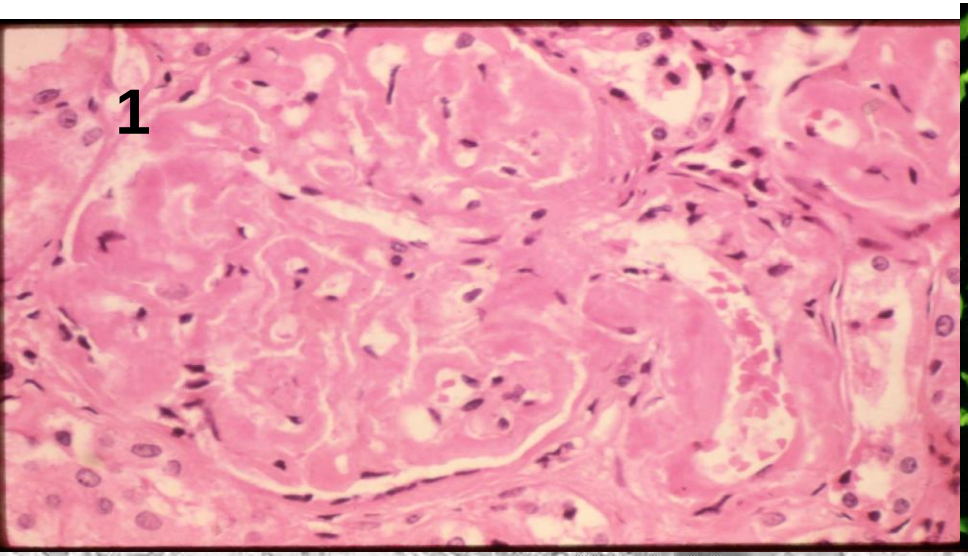
Question 15

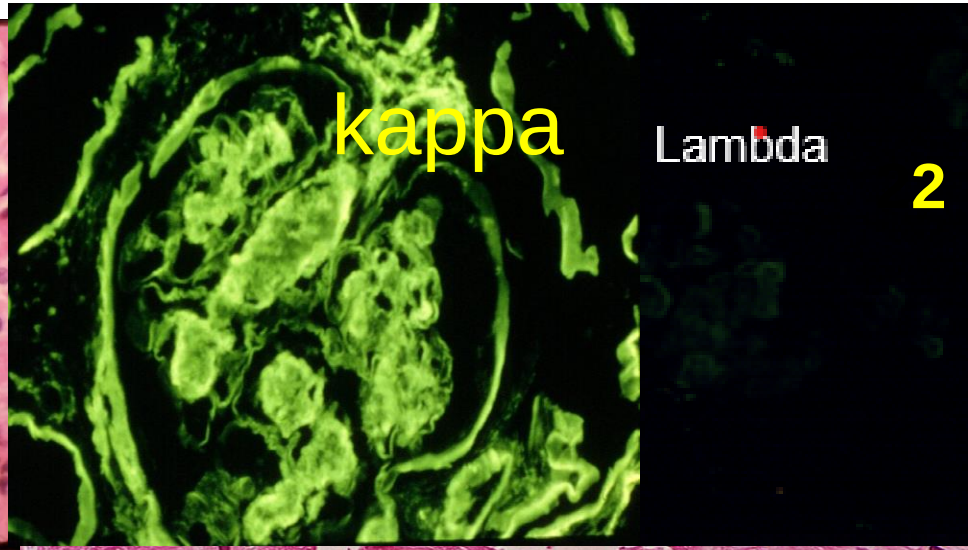
A 62 yo is evaluated for proteinuria and edema.

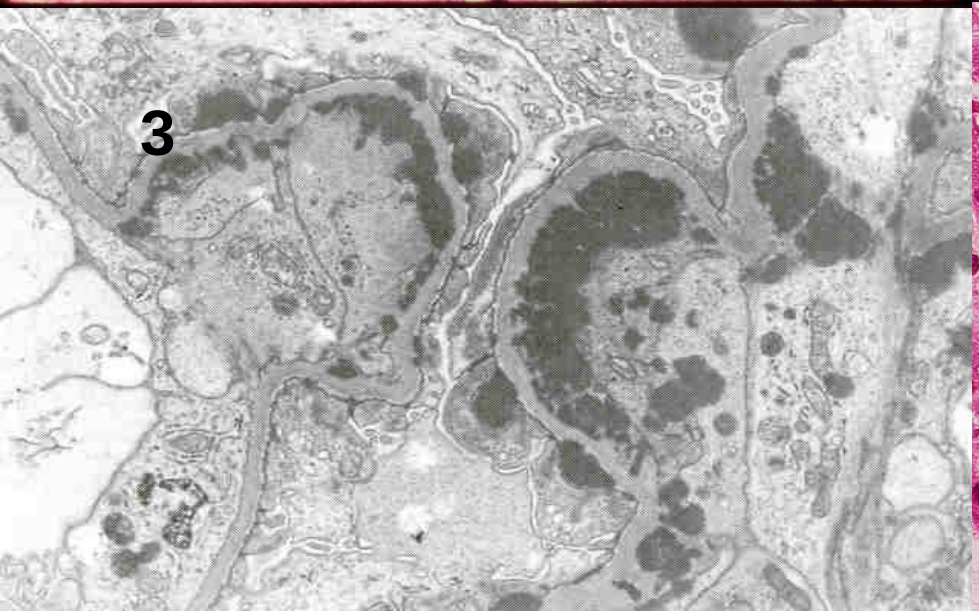
- Px BP 142/84, Cor S1S2 S4 G, Chest clear, Abd no LKKS, ext 2-3+ pedal edema
- Lab. BUN 12, Pcreat 0.7 mg/dl, Urine dipstick in office 4+ prot, 2+ heme, 24 hr urine protein 4.2 g/d,
- ANA , complement , Hep B-C and HIV neg
- Spep 1.8 g IgG kappa, Palb 2.2 g/dl

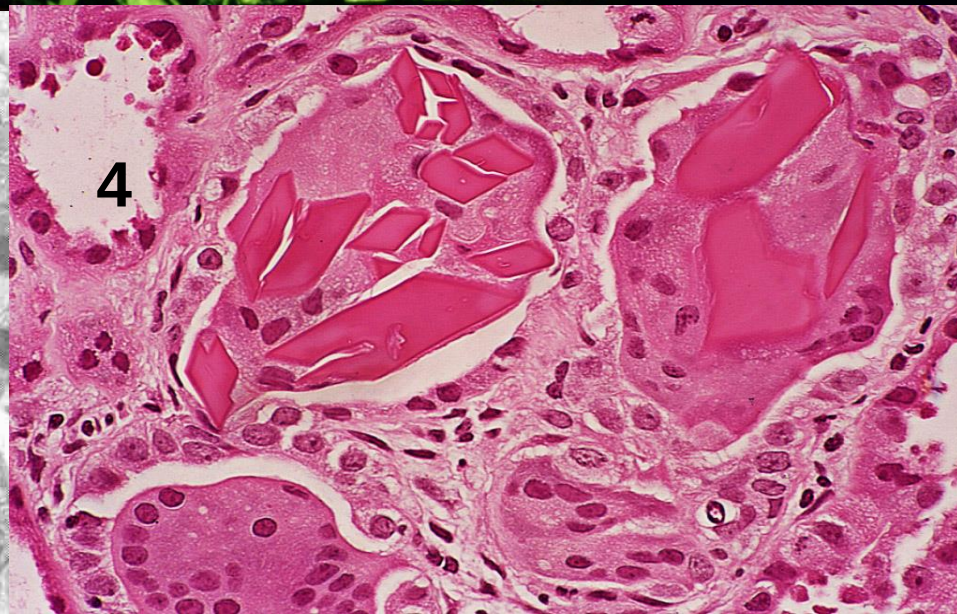
Question 15

A Kidney Biopsy is least likely to show which of the Following ?





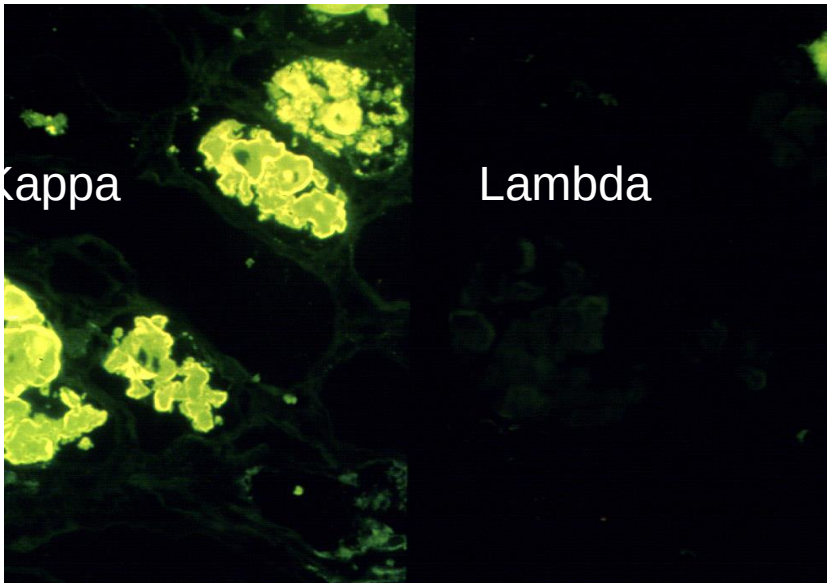




Clinical Pearls about MGRS

1. Lambda or Kappa IF staining without the other light chain.

Think monoclonal disease.



2. Light chains minimally affect the urinary dipstick protein. Tr-1+ dipstick and heavy proteinuria on the 24 hr or Up/Ucr.

Think urinary light chains.

Light Chain and light chain Fragments in Glomeruli

Leung N, Bridoux F, Nasr SH NEJM 384: 1931-1941,2021

AL Amyloid, LCDD,
And PGNMID all
Are associated with
glomerular proteinuria
and albuminuria. All
Give + dipstick for protein.

LC cast nephropathy
often does not.