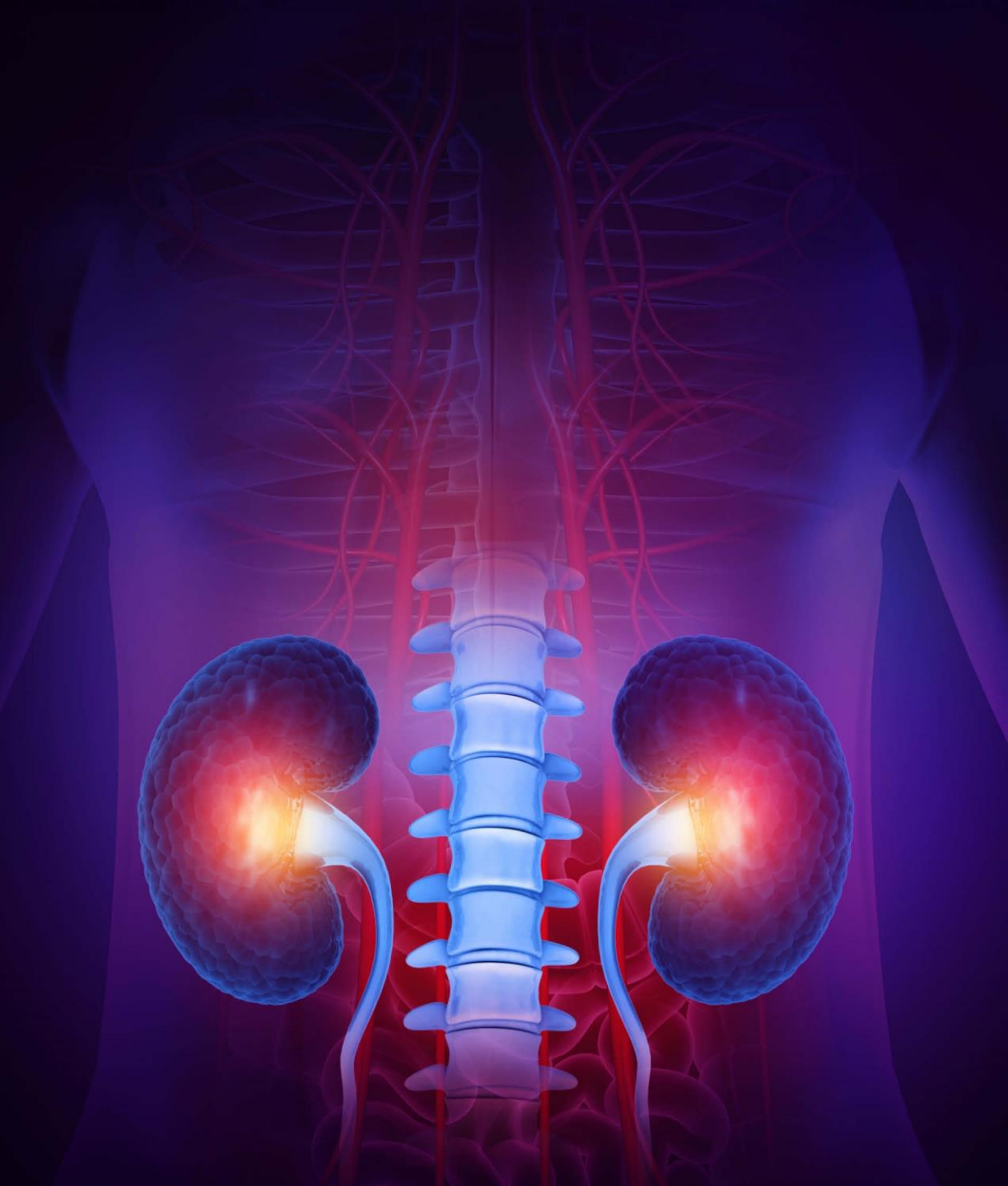
Optimizing Outcomes in Complement 3 Glomerulopathy

Managed Care Approaches to Improve Disease Awareness and Management



Managed Care | LEARNING NETWORK

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Faculty Disclosures

- **Joshua Thurman, MD**: Consultant—Q32 Bio; Stockholder—Q32 Bio, Compsit3; Royalties—Q32 Bio
- **Russell Spjut, PharmD** has disclosed no relevant financial relationship with any ineligible company (commercial interest)

Program Information

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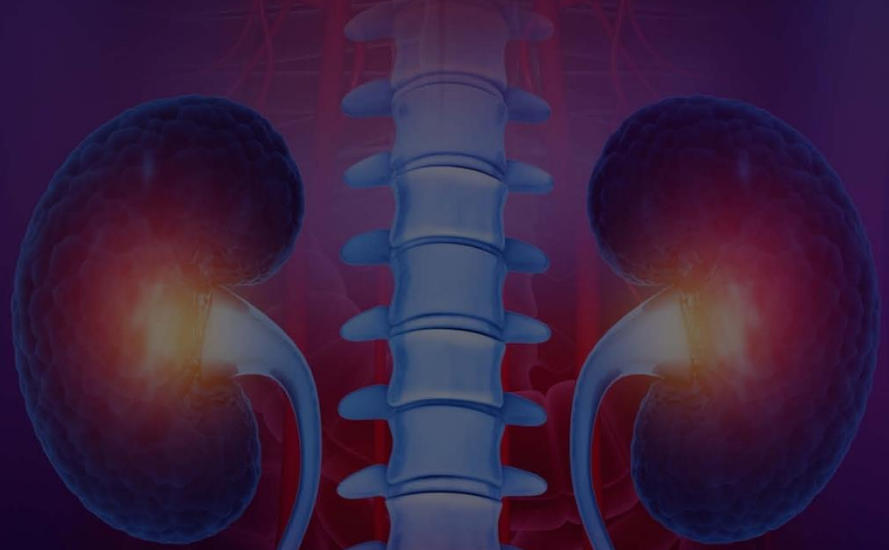
- Supported by an educational grant from Apellis Pharmaceuticals

Learning Objectives

- Assess the current clinical landscape of C3G, including its prevalence, impact on healthcare systems, and associated economic burden
- Describe the underlying pathogenesis, diagnostic approaches, and management of C3G
- Evaluate the most recent clinical findings concerning C3G treatment, including the limitations of conventional therapies and the potential of new and emerging complement inhibitors
- Implement cost-effective and evidence-based managed care policies to improve patient outcomes and minimize the burden of C3G

C3G = complement 3 glomerulopathy.

Overview and Impact of C3G



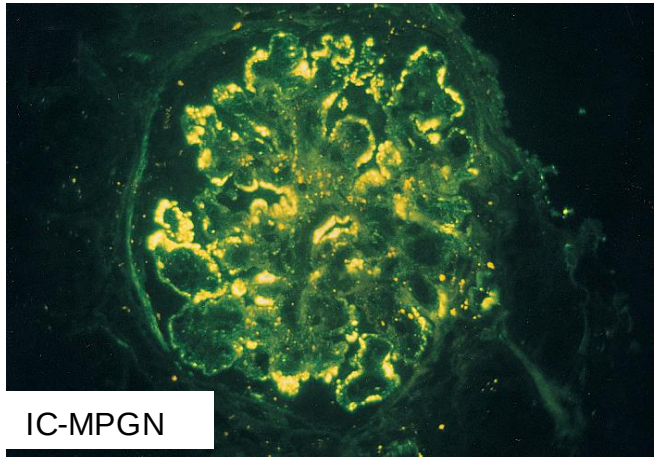
Introduction – Rare Kidney Diseases

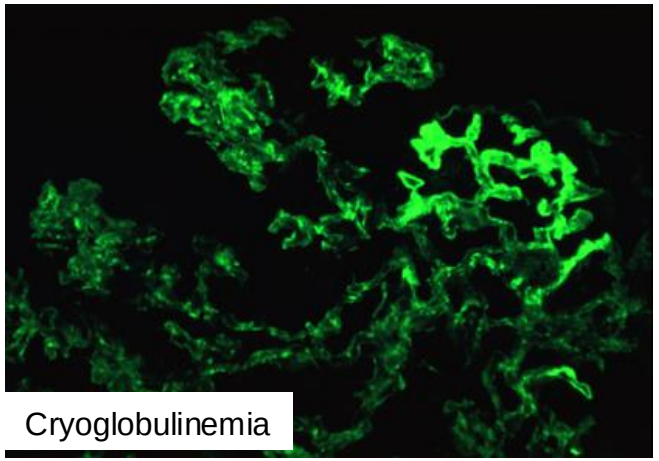
- “Orphan disease” – affects fewer than 200,000 in the US
- “Ultra-orphan disease” – fewer than 1 in 50,000
- Limited data – natural history, clinical trials
  - Within nephrology, research into C3G has been very successful
- The challenges of testing new treatments for rare diseases
  - Difficult to recruit for clinical trials
  - For most forms of glomerulonephritis, progression occurs over years
    - Reliance on surrogate endpoints
- Risks of treatment accumulate
  - Infections
  - Other medication adverse events
  - Risks of not treating
    - Irreversible damage to the kidneys
    - Chronically low complement and nephrotic range proteinuria also increases risk of infections

C3G – Complement in the Absence of ICs

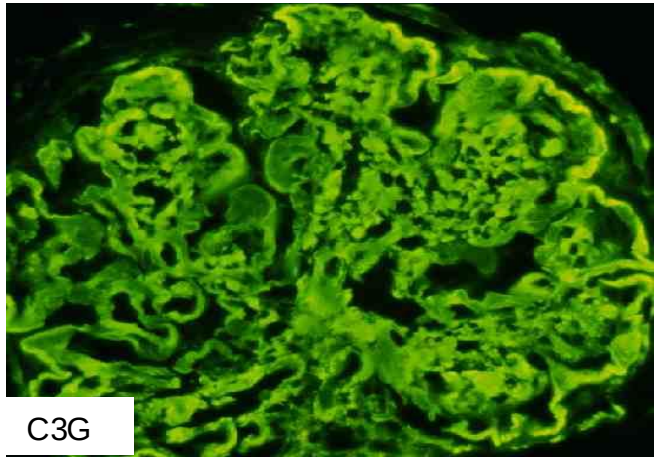
Relative

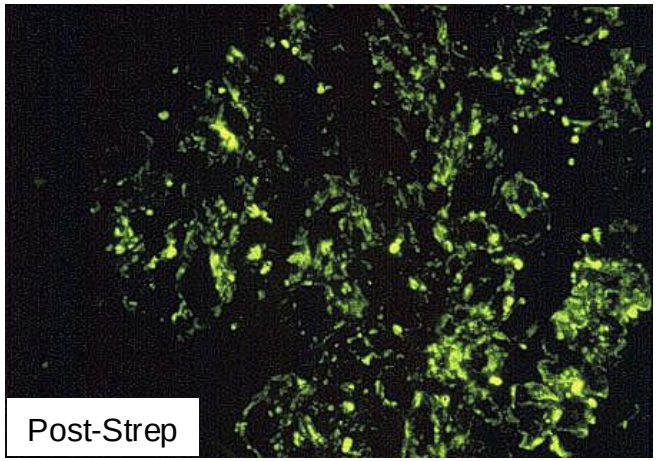
Immune-complex mediated





Non-immune-complex mediated



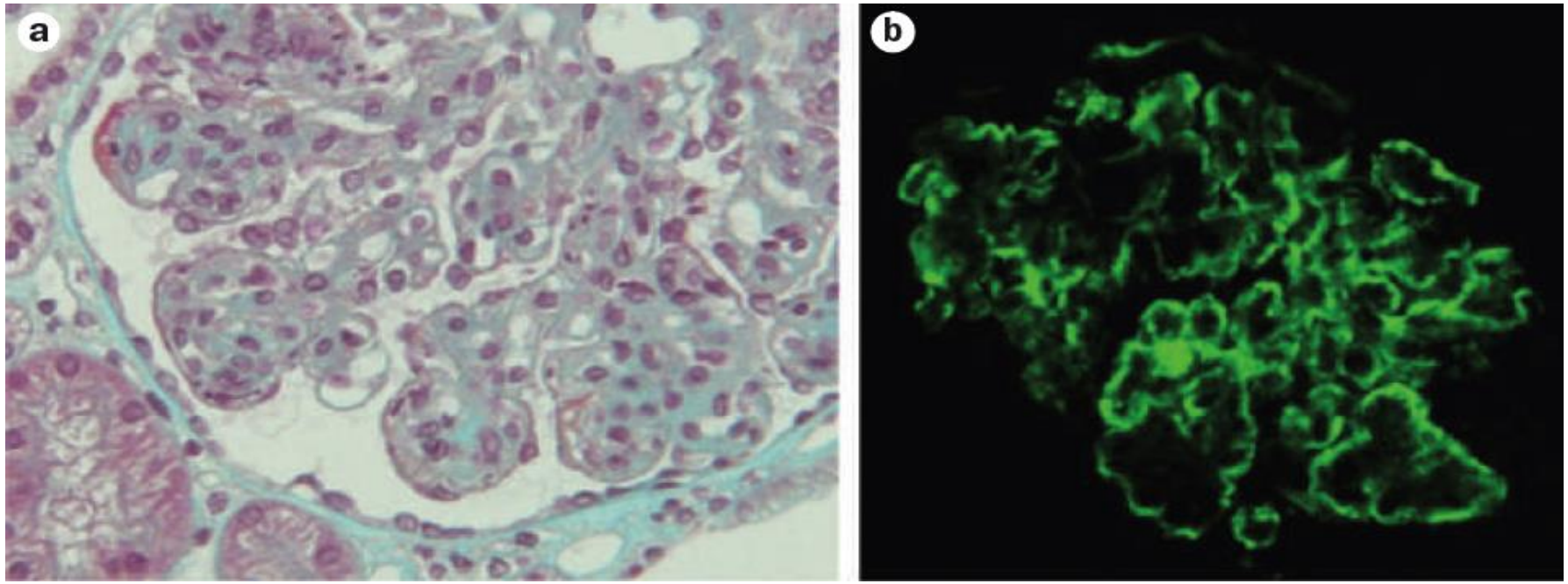


IC = immune complex; MPGN = membranoproliferative glomerulonephritis.

Kim Y, et al. *Lab Invest.* 1979;40(4):474-80.

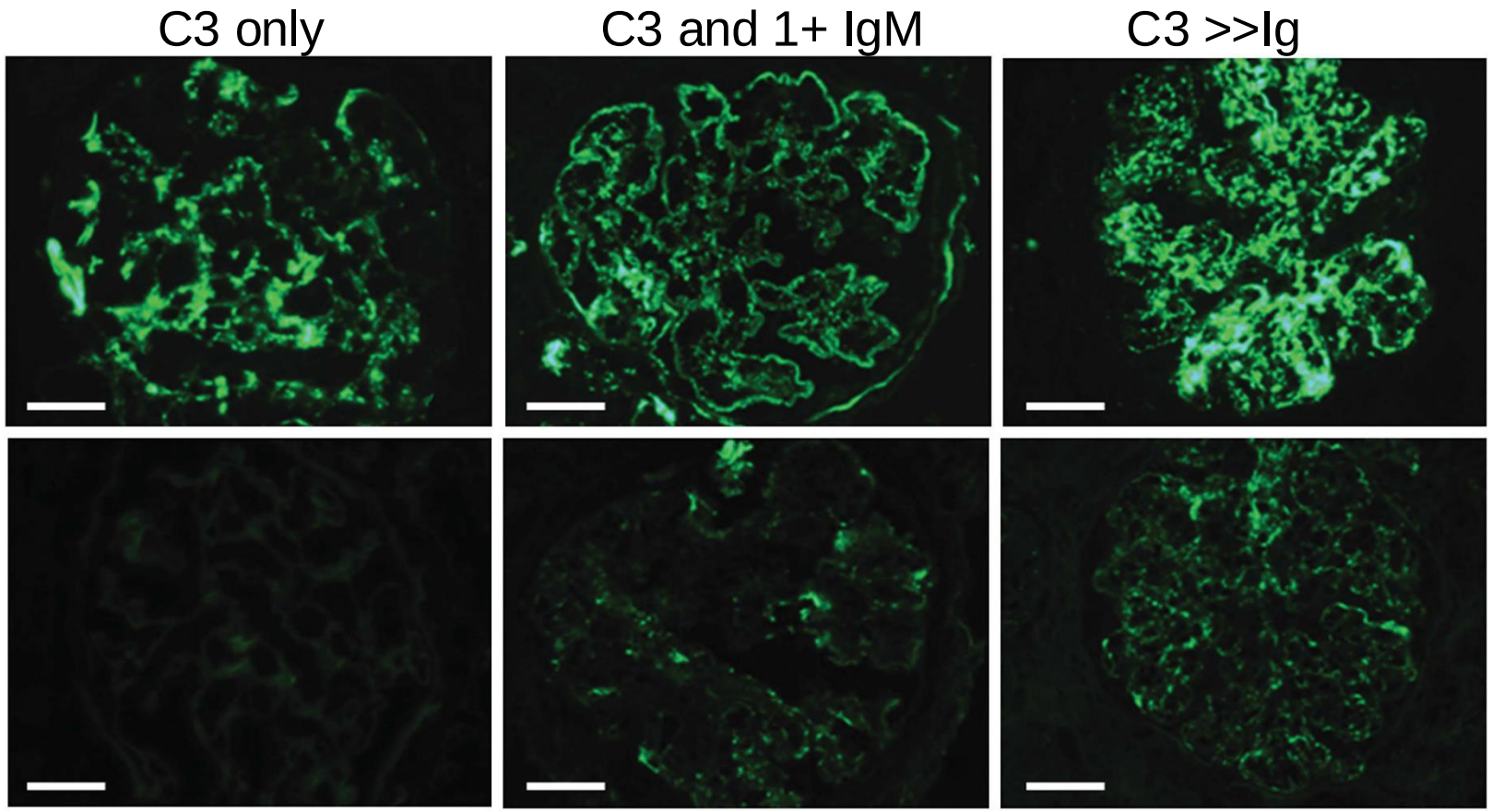
C3G – Complement without the ICs

- C3 two orders of magnitude higher than immunoglobulin



Servais A, et al. *J Med Genetics.* 2007;44(3):193-199. Fakhouri F, et al. *Nat Rev Nephrol.* 2010;6(8):494-499. Pickering MC, et al. *Kidney Int*. 2013;84(6):1079-1089.

Immunoglobulin in C3G



- Not driven by immune-complexes
- AP-mediated

Ig = immunoglobulin; AP = alternative pathway.
Hou J, et al. *Kidney Int.* 2014;85(2):450-456.

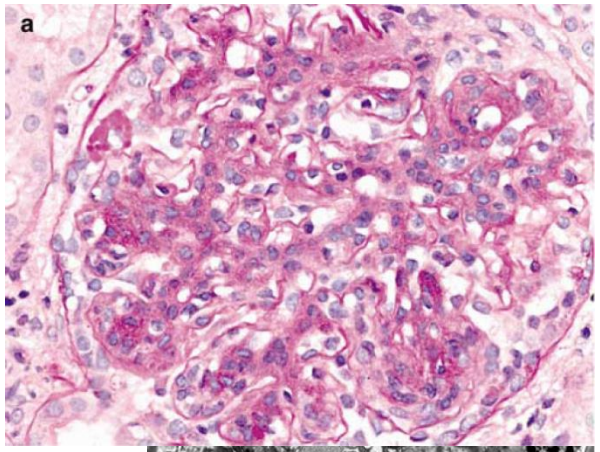
Historical Context: The Shrinking Diagnosis of MPGN

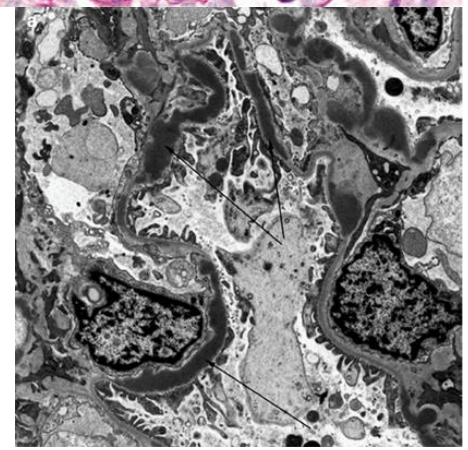
MPGN

Studied by Richard Bright in 1820s (preserved in brandy and then formalin). Reanalyzed by Weller and Nester, British Medical Journal, 1972.
Pattern described by MacCallum, 1934.

- Secondary MPGN – eg, infection, cancer, autoimmune
- MPGN type 2
- Dense deposit disease
- C3 Glomerulopathy

Idiopathic MPGN





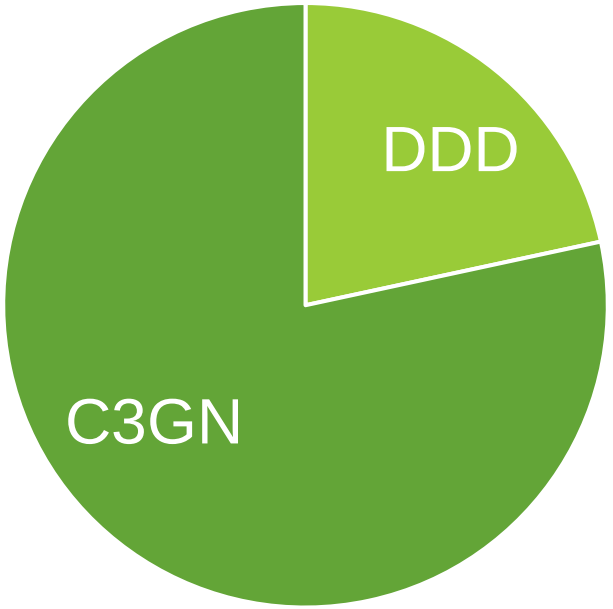
MPGN = membranoproliferative glomerulonephritis.

Weller RO and Nester B. *Br Med J*. 1972;2(5816):761-763. MacCallum WG. *Bull Johns Hopkins Hosp*. 1934;55(5):416. Löhlein M. *Med Klin*. 1910;6:375. Berger J and Galle P. *J Urol Nephrol (Paris).* 1962;68:116-122. Servais A, et al. *J Med Genet.* 2007;44(3):193-199. Walker KD, et al. *Mod Pathol*. 2007;20(6):605-616.

Prevalence and Incidence of C3G

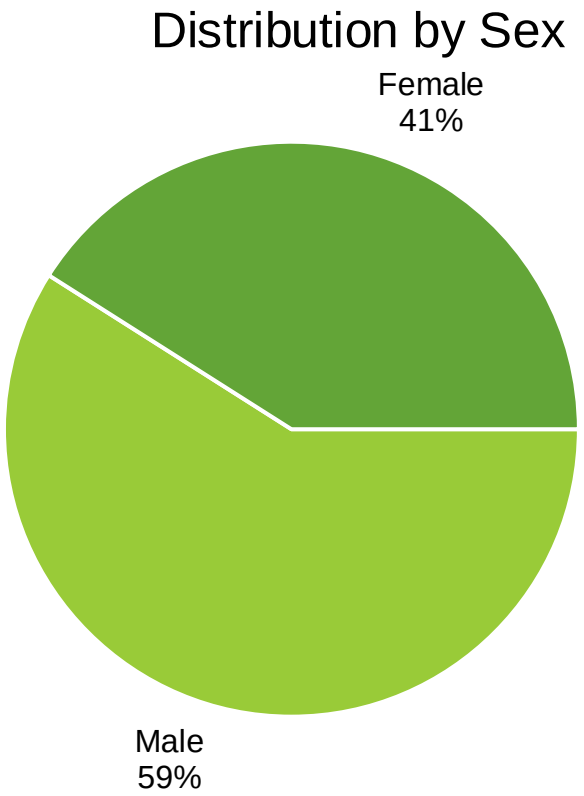
- The term C3G was defined by consensus in just 2013 making population level data an evolving metric
  - Increases in population level research
  - Increases in awareness and potential treatments
- New diagnoses have been slightly easier to track, though the data is still spread estimating around 1-3 new cases per 1,000,000 lives per year
- Prevalence data is somewhat inconclusive, though likely sits between 5-40 cases per 1,000,000 lives

Proportion of Patients With C3G Subtypes



Smith RJH, et al. *Nat Rev Nephrol*. 2019;15(3):129-143. Mehdi A, et al. *Clev Clin J Med*. 2023;90(6 suppl 1):e1-e4. Bomback AS, et al. *Kidney Int*. 2018;93(4):977-985.

Distribution of C3G



- Median age of diagnosis worldwide was recently found to be around 39 years
  - Earlier data found an average age of diagnosis of 21 years
- Awareness and understanding have increased which may account for a shift in diagnosing more at an older age
- Diagnosis can occur at any time through life with DDD primarily found in children and young adults

Lafayette RA, et al. *J Am Soc Nephrol*. 2023;34(11S):272. Heiderscheit AK, et al. *Am J Med Gen*. 2022;190(3):344-357. Complement 3 Glomerulopathy (C3G). Cleveland Clinic. Updated June 10, 2023. Accessed December 2, 2024. https://my.clevelandclinic.org/health/diseases/25074-c3g.

Landscape of Healthcare Utilization Data

- All healthcare utilization data is almost non-existent
  - It is almost a surety that outpatient utilization increases vs the general population as diagnosis is usually a long process involving multiple provider visits and tests
  - It can be difficult to adequately treat the disease with current therapies, meaning that patients often have consistent follow-up and disease progression leading to increased utilization and costs, though we do not have data on the actual numbers or extent
- In many conditions similar to C3G, hospitalization ends up being a main driver of cost
  - C3G can often progress to a point where inpatient management is needed, such as if the patient experiences an episode of acute kidney injury or complicated infection
  - Data in these types of conditions is currently not tied to the C3G diagnosis, so we do not have population data on the extent these drive costs

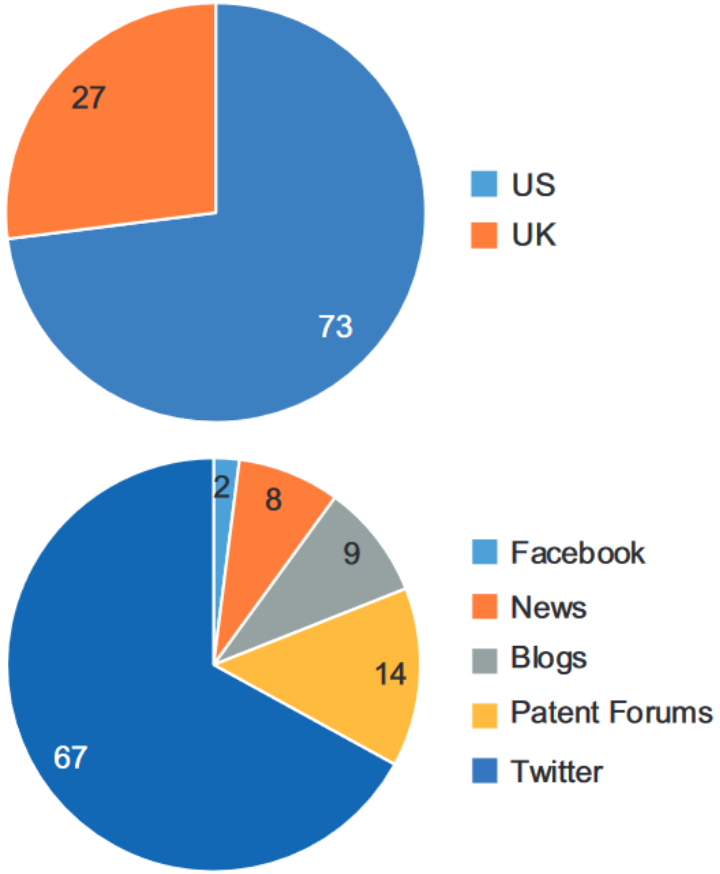
Java A, et al. *Kidney Med*. 2024 [Epub ahead of print]. Tyagi N, et al. *Value in Health*. 2019;22(Suppl 3):S919.

Landscape of Economic Data

- Comprehensive economic data is scarce – we have a knowledge gap
- Much of the available data focuses on the burden of disease progression to the point a kidney transplant is needed
  - A recent literature review report found that between the 33 publications they reviewed
    - Up to 30% of C3GN patients progressed to ESRD over a 10-year time horizon
    - Up to 100% of DDD patients progressed to ESRD over a 15-year time horizon
    - For patients in each of these ESRD groups, kidney transplants were as high as 59% and 92% for the respective subtypes

George AT, et al. *Value in Health*. 2018;21:S480.

Patient Burden – A Social Media Study: Design



- 2019 poster presentation reported findings from a social media listening study
- Medical Subject Heading (MeSH) terms were used to find and download social media posts and data related to C3G from September 2015–September 2018
- Data was anonymized and then analyzed using tools like natural language processing algorithms that could be classified under machine learning tools

Tyagi N, et al. *Value in Health*. 2019;22(Suppl 3):S919.

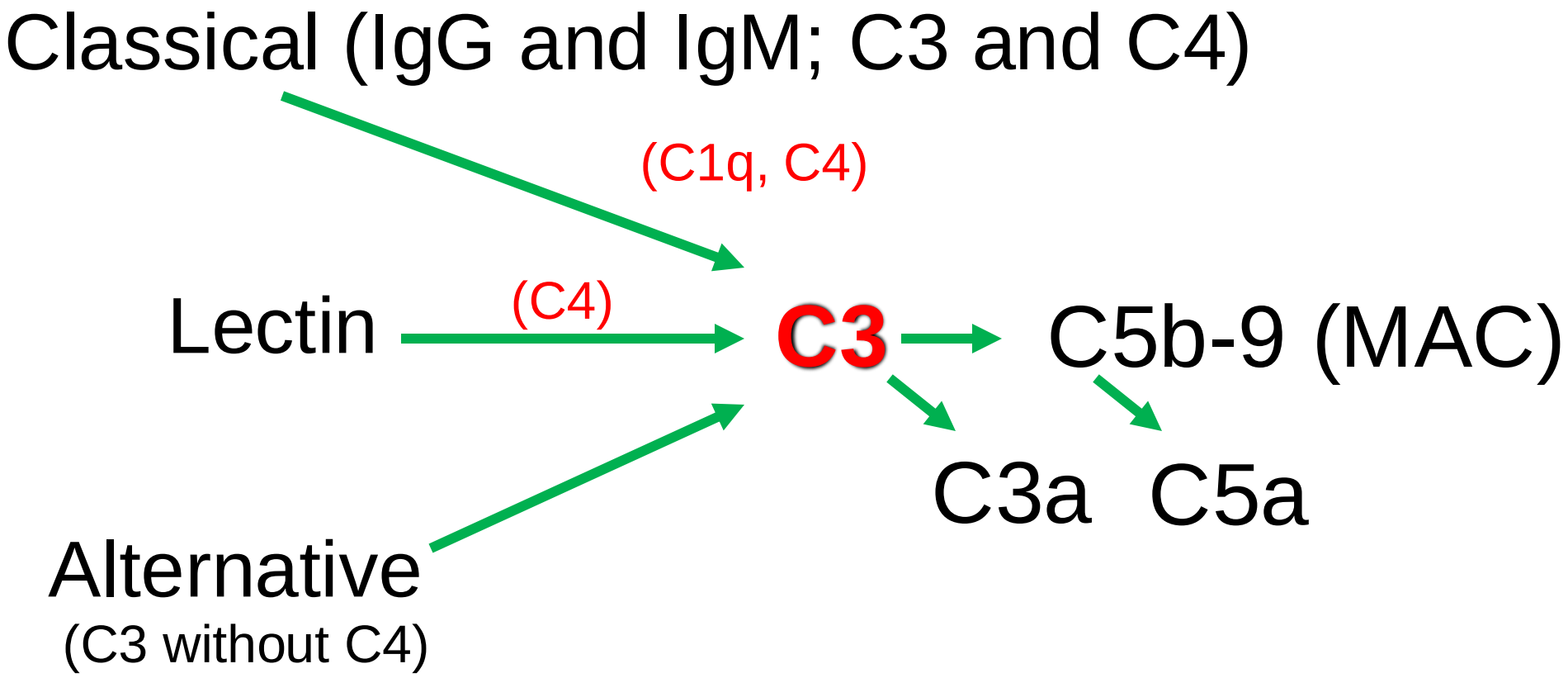
Patient Burden – A Social Media Study: Results

- Identified some seemingly large factors related to quality of life (QoL)
  - Symptoms such as fatigue, edema, proteinuria, and hematuria were commonly discussed as worrisome to many patients and caregivers
    - “I get days I’m so drained and I can’t force myself to do anything”
  - Many expressed frustration with delays in diagnosis
  - Lack of information about the disease and treatment options led many to seek help from other patients and caregivers to understand their disease
- Progression of disease leads to a lot of mental burden
  - Dialysis and transplant are tough realities to face
  - The financial burden faced as the disease progresses weighs heavy

Tyagi N, et al. *Value in Health*. 2019;22(Suppl 3):S919.

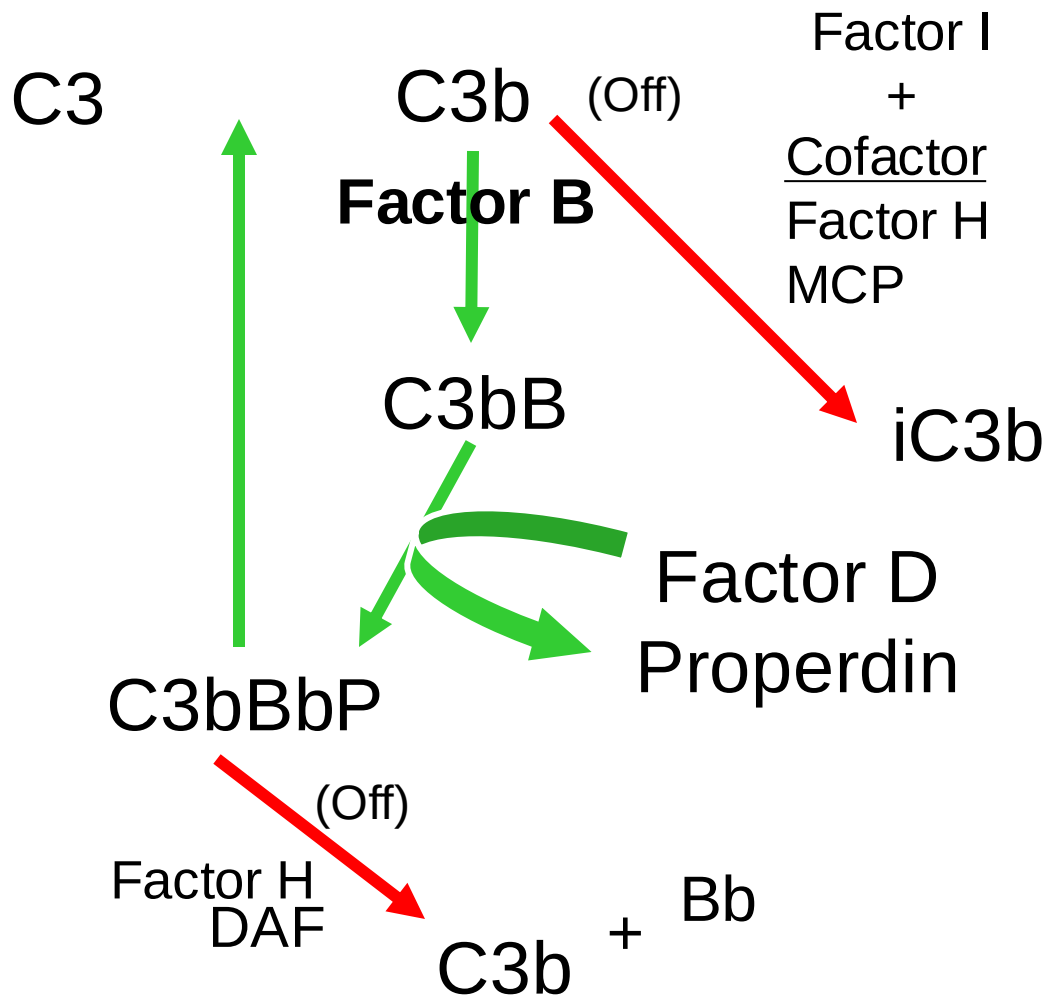
Pathogenesis

How can complement be activated in the absence of ICs?



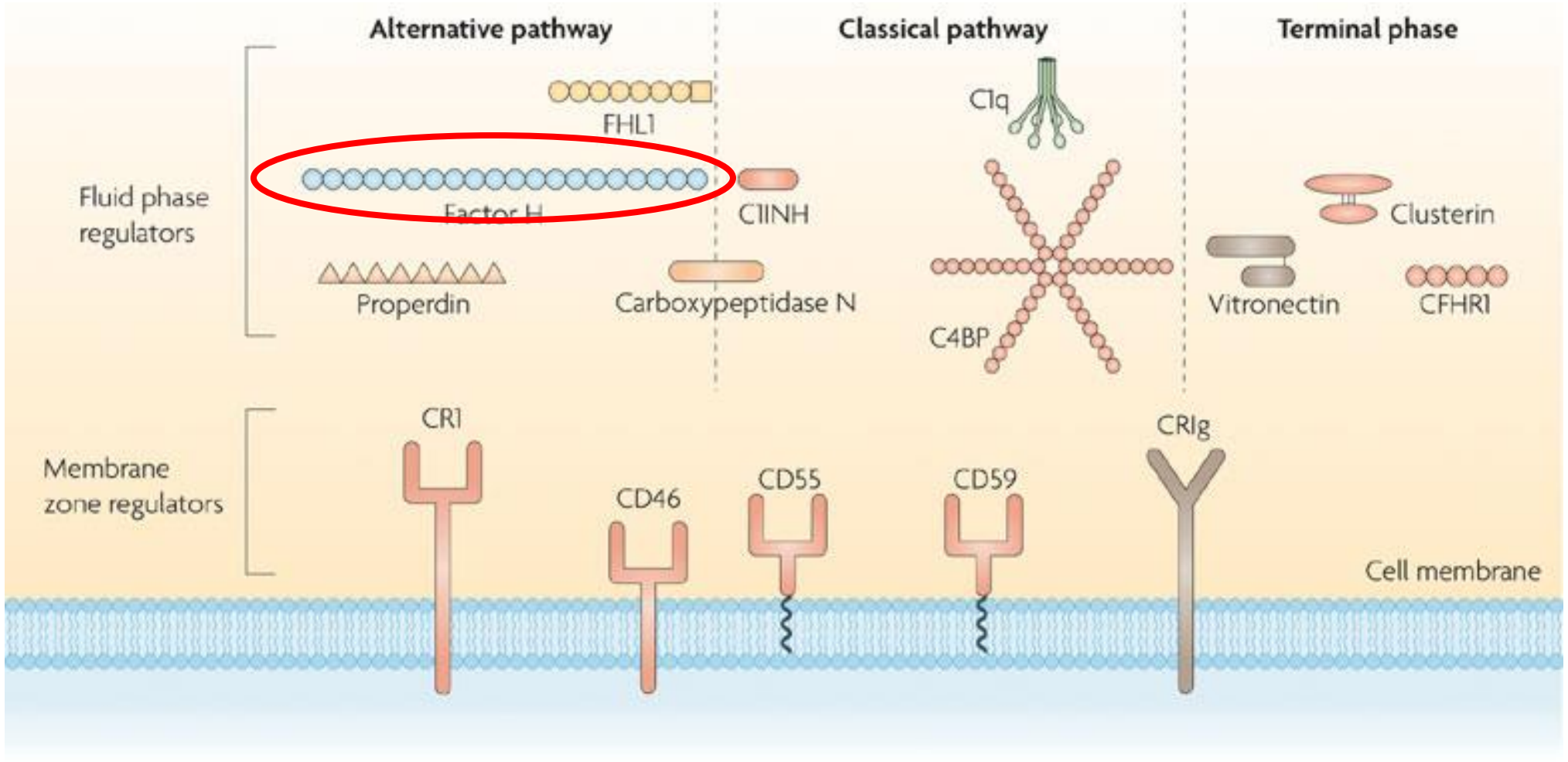
MAC = membrane attack complex.

C3G – Uncontrolled AP Activation



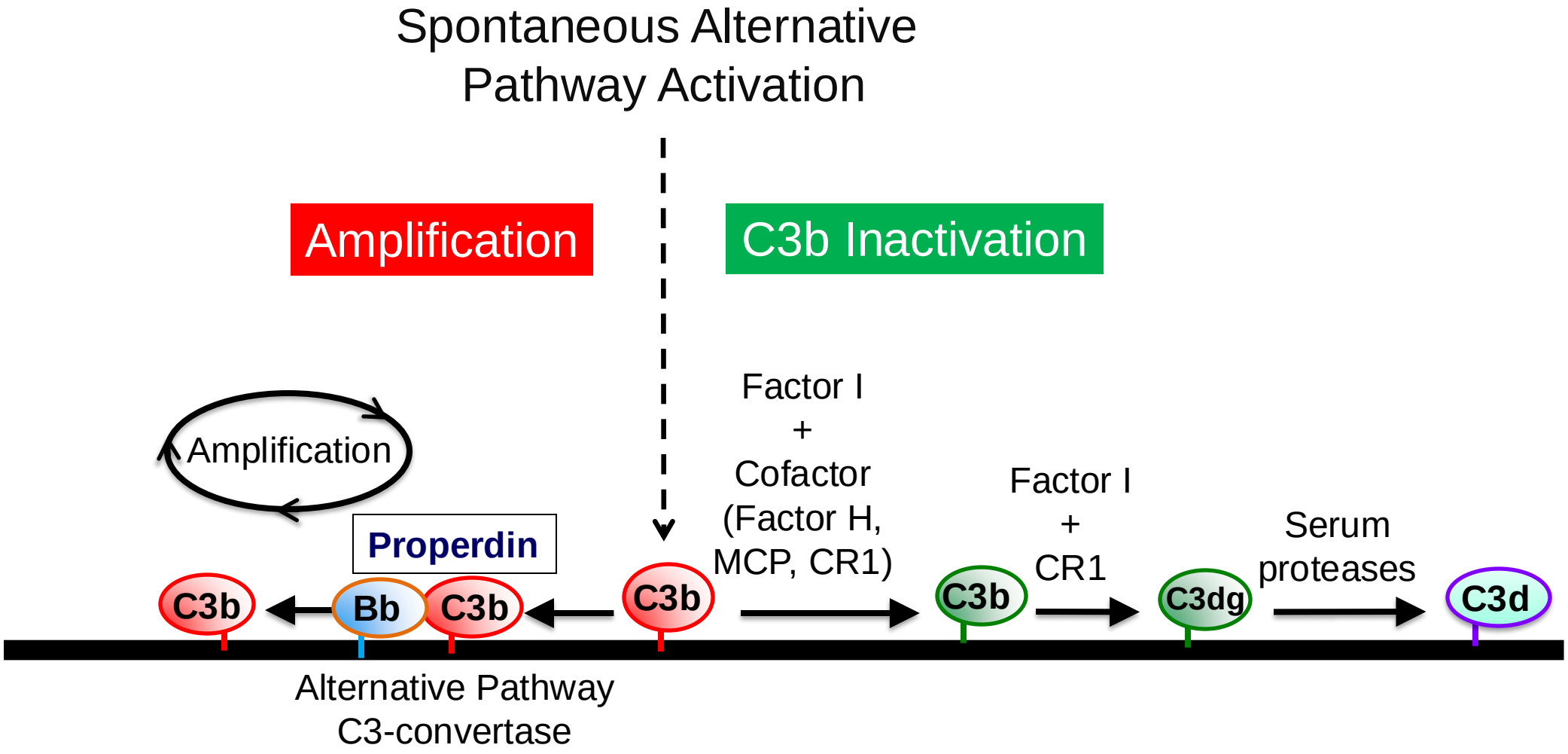
AP = alternative pathway; DAF = decay accelerating factor.

Complement Regulatory Proteins



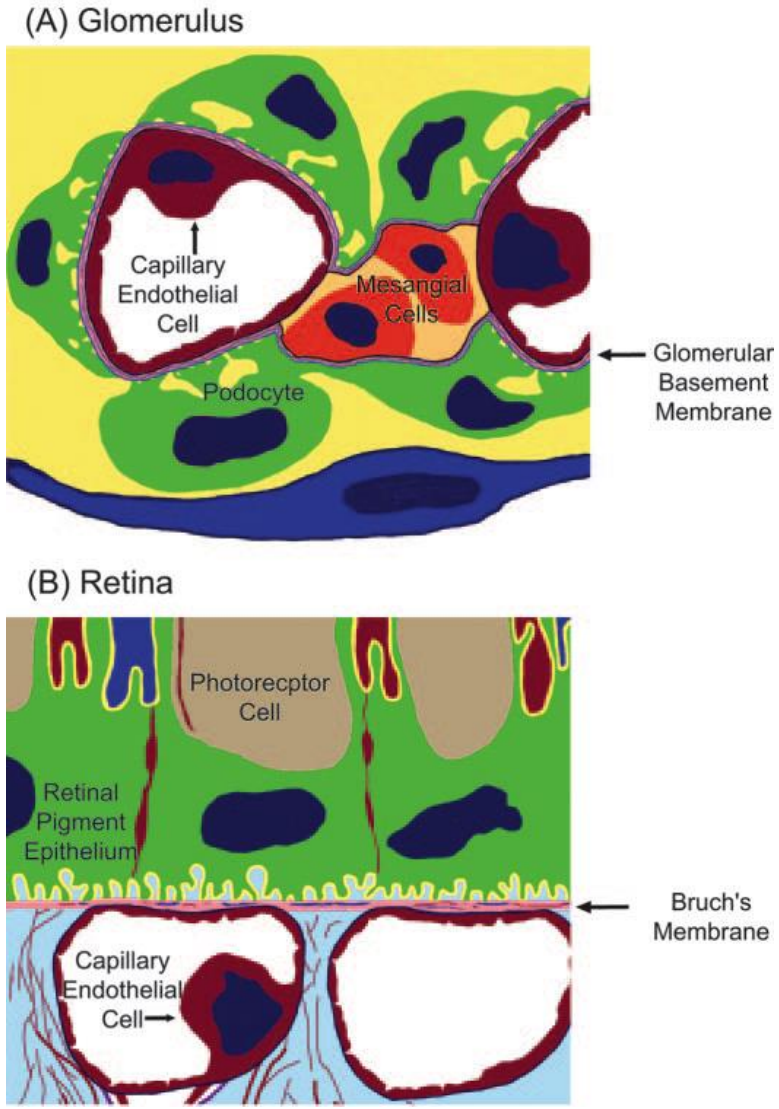
Zipfel PF, et al. *Nat Rev Immunol*. 2009;9(10):729-740.

AP "Activating" and "Inactivating" Surfaces



Thurman JM, et al. *Clin J Am Soc Nephrol*. 2016;11(10):1856-1866.

Glomerular Basement Membrane – Achilles Heel of the Glom

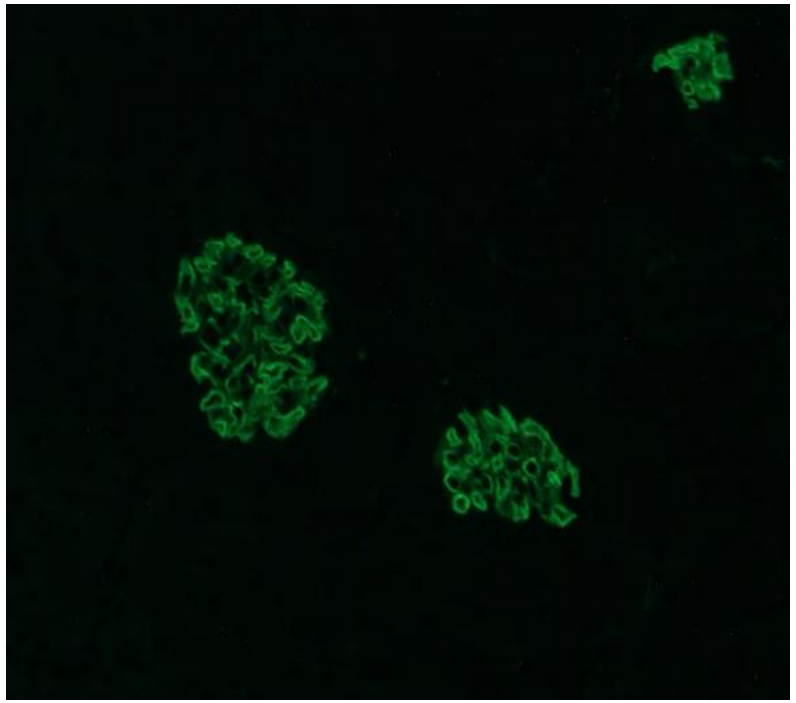


Appel GB, et al. *J Am Soc Nephrol*. 2005;16(5):1392-1403.

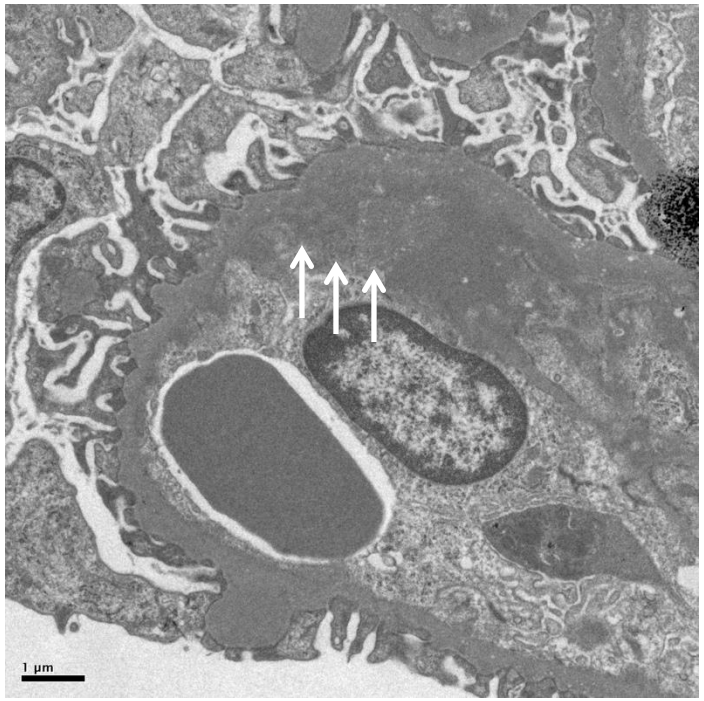
The Absence of FH is Sufficient to Cause C3G

fH$^{-/-}$ Kidney
3 month

C3b



EM



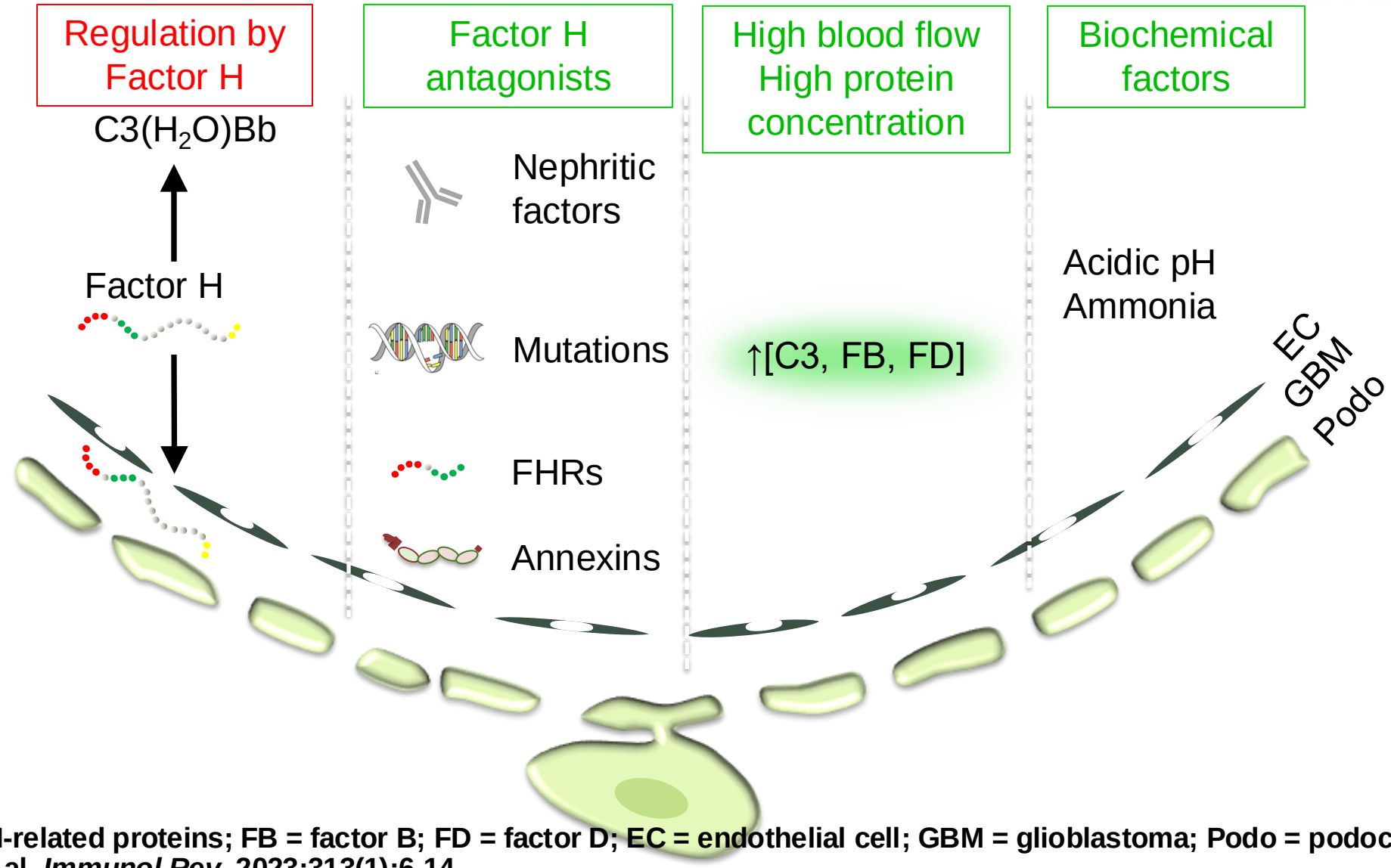
FH = factor H; EM = electron microscopy.
Laskowski J, et al. *Kidney Int*. 2016;90(1):109-122.

C3G – Uncontrolled AP Activation

- Autoantibodies to complement proteins
  - Nephritic factors (>70% of pts)
  - Autoantibodies to complement proteins
  - Plasma cell dyscrasias
- Genetic defects of complement (15% of pts)
  - Loss of function (regulation)
  - Gain of function (activation)
  - Disease associated variants
- Environmental triggers

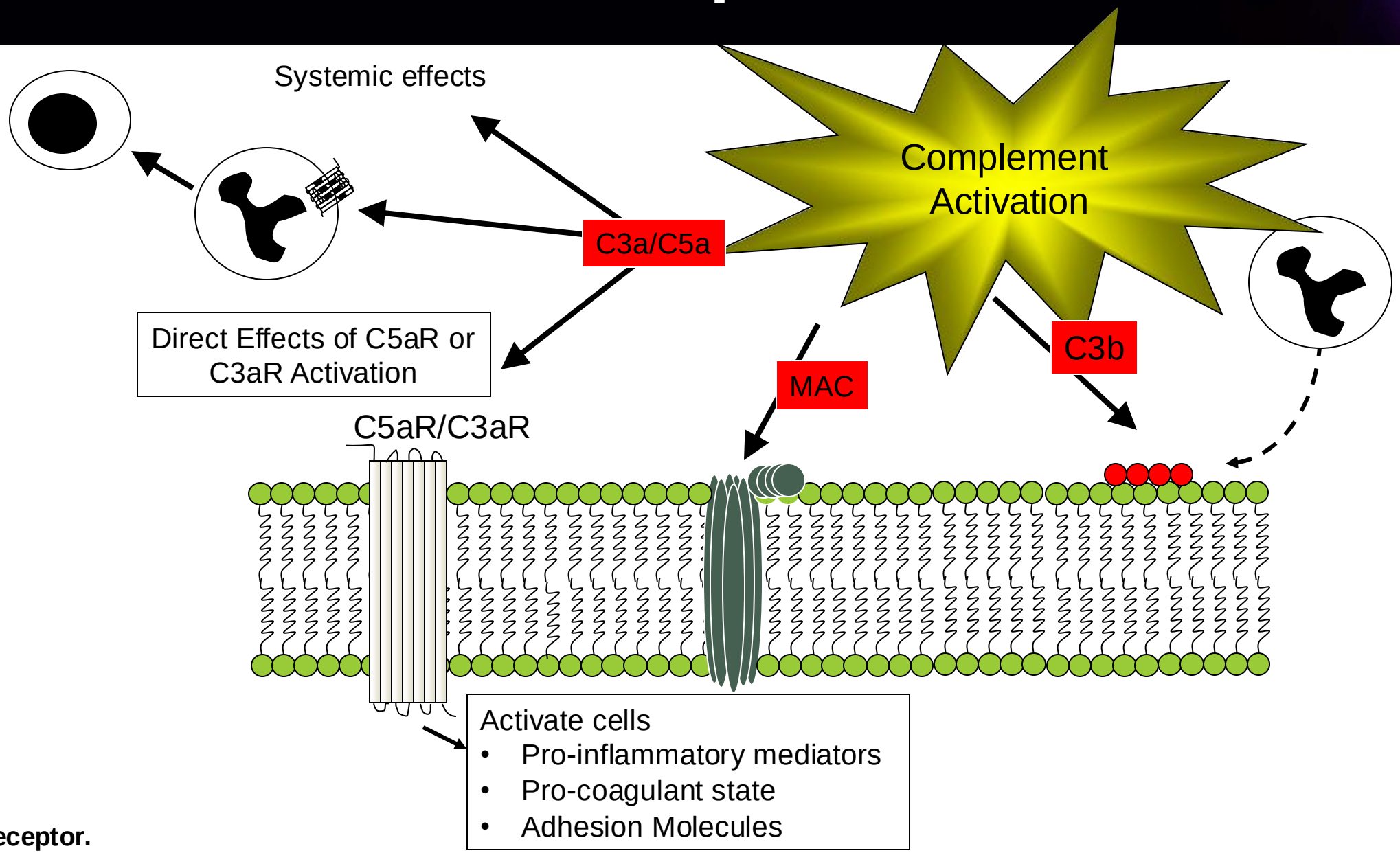
Chen Q, et al. *N Engl J Med.* 2011;365:2340-2342. Bridoux F, et al. *Clin J Am Soc Nephrol.* 2011;6(9):2165-2174. Servais A, et al. *Kidney Int.* 2012;82(4):454-464.

Why Is the Kidney the Primary Target of the AP?



FHRs = factor H-related proteins; FB = factor B; FD = factor D; EC = endothelial cell; GBM = glioblastoma; Podo = podocyte.
Harrison RA, et al. *Immunol Rev*. 2023;313(1):6-14.

Mechanisms of Complement-Mediated Injury



R = receptor.

Diagnosis and Management of C3G

Clinical Presentation

- Variable clinical course
  - 30-40% of patients present in childhood
  - ~50% of patients → ESKD within 10 years
- Infection can precede kidney disease (28.9%)
- Usually renal-limited
  - Drusen
  - Partial lipodistrophy
- Nephritic
  - Proteinuria
  - Hematuria

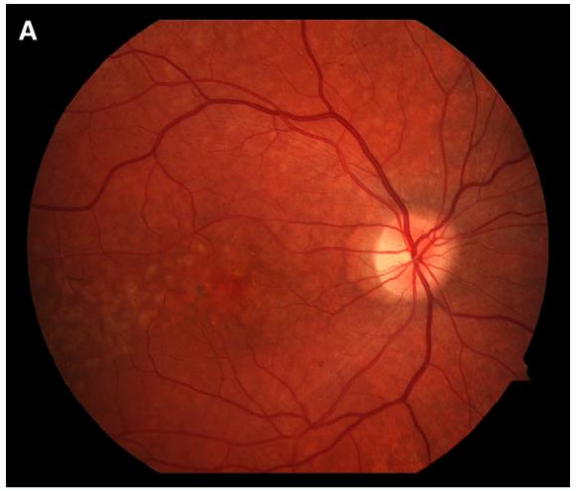
  } Biopsy
  - Hypocomplementemia 50-75%
- May have the worst prognosis of the primary GNs

ESKD = end-stage kidney disease; GN = glomerular disease.

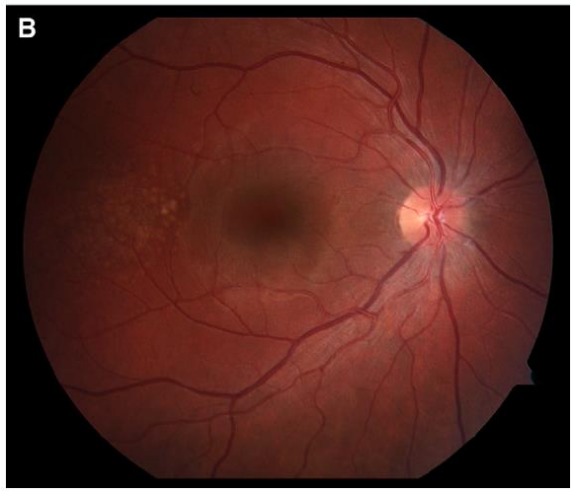
Bomback AS, et al. *Kidney Int.* 2018;93(4):977-985. Medjeral-Thomas NR, et al. *Clin J Am Soc Nephrol.* 2014;9(1):46-53. Servais A, et al. *Kidney Int.* 2012;82(4):454-464. Smith RJH, et al. *J Am Soc Nephrol*. 2007;18(9):2447-2456. Wooden B, et al. *Adv Kidney Dis Health.* 2024;31(3):223-233.

Drusen Formation in DDD and C3GN

DDD



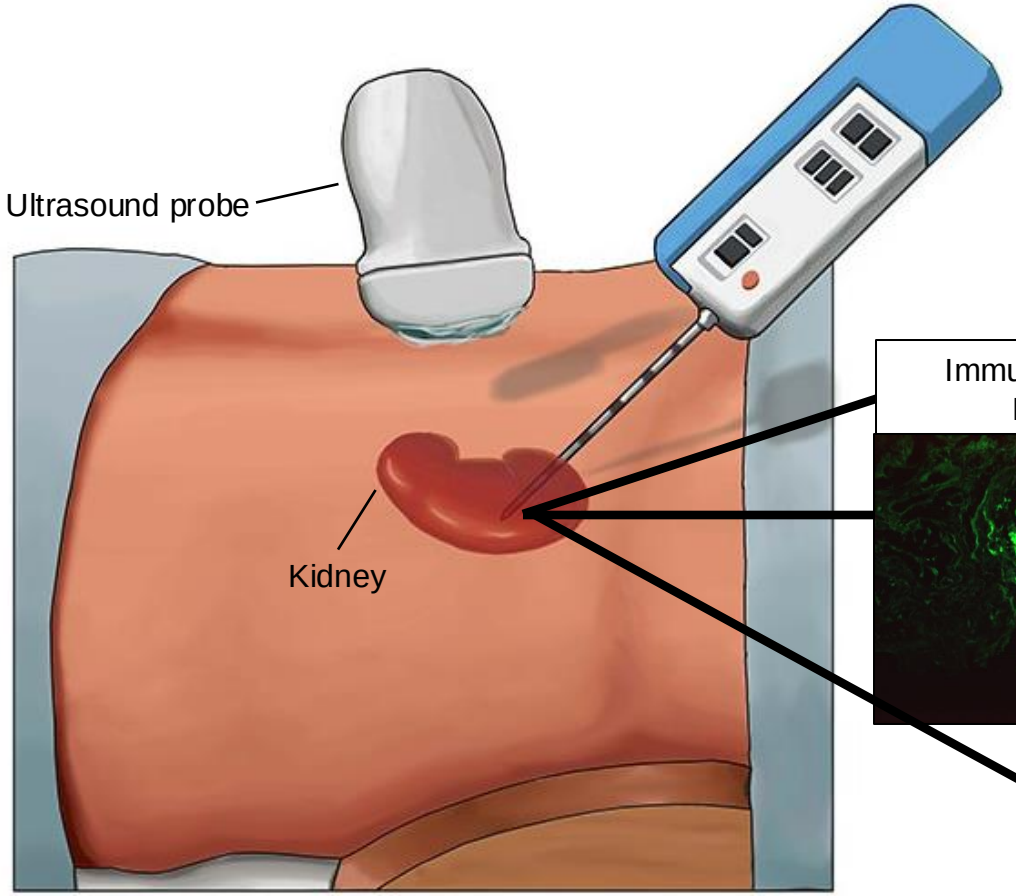
C3GN



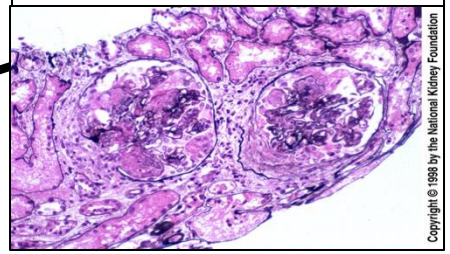
DDD = dense deposit disease.

Bomback AS, et al. *Clin J Am Soc Nephrol*. 2012;7(5):748-756.

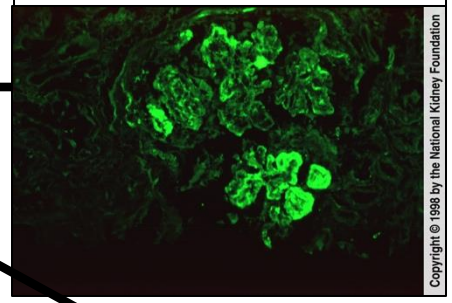
C3G – Diagnosis Based on IF and EM



Light Microscopy

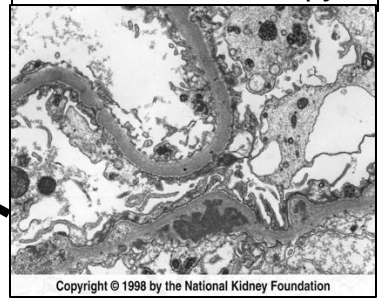


Immunofluorescence Microscopy



IF: IgG, IgM, IgA, C3, C1q

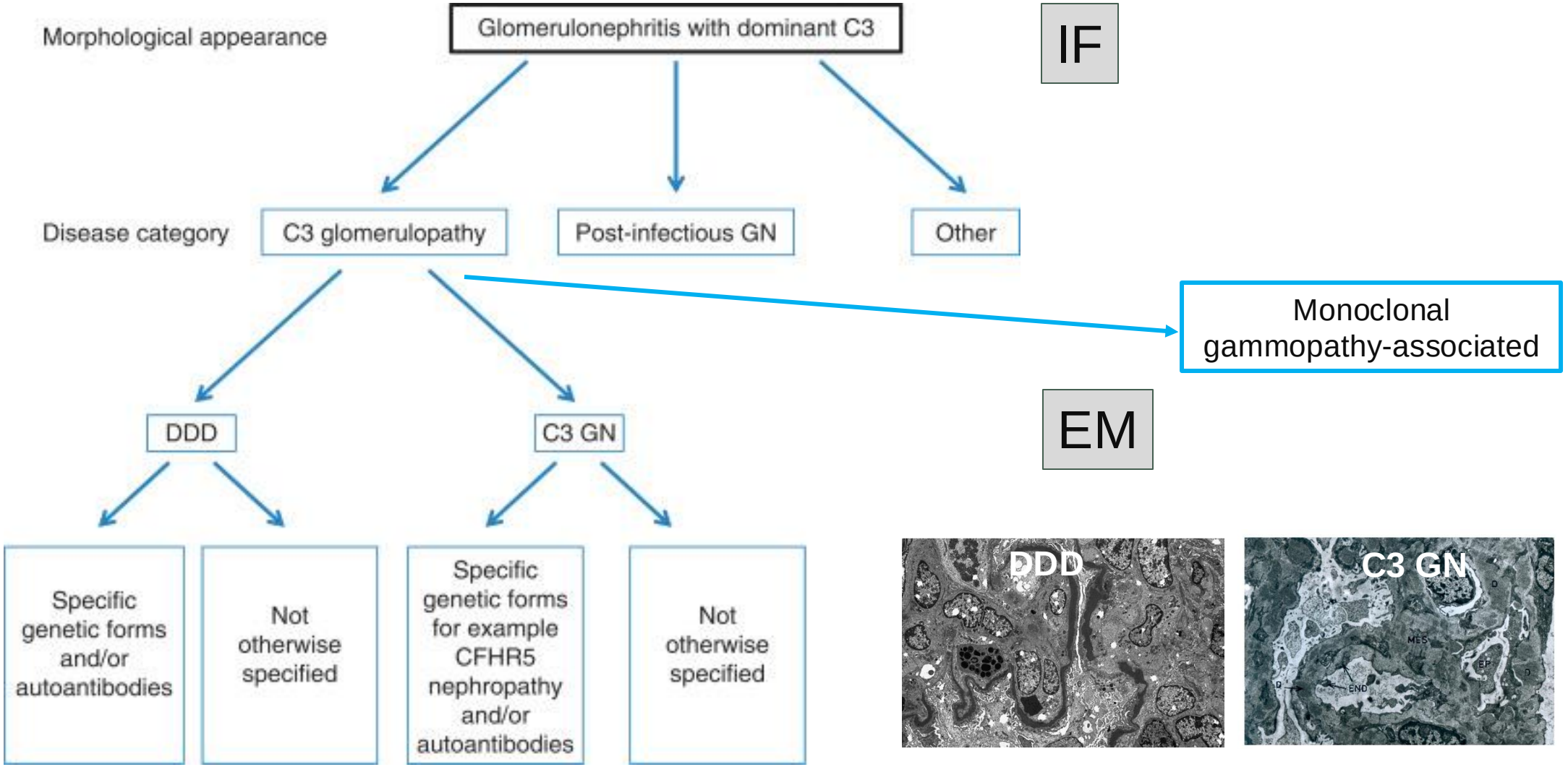
Electron Microscopy



IF = immunofluorescence.

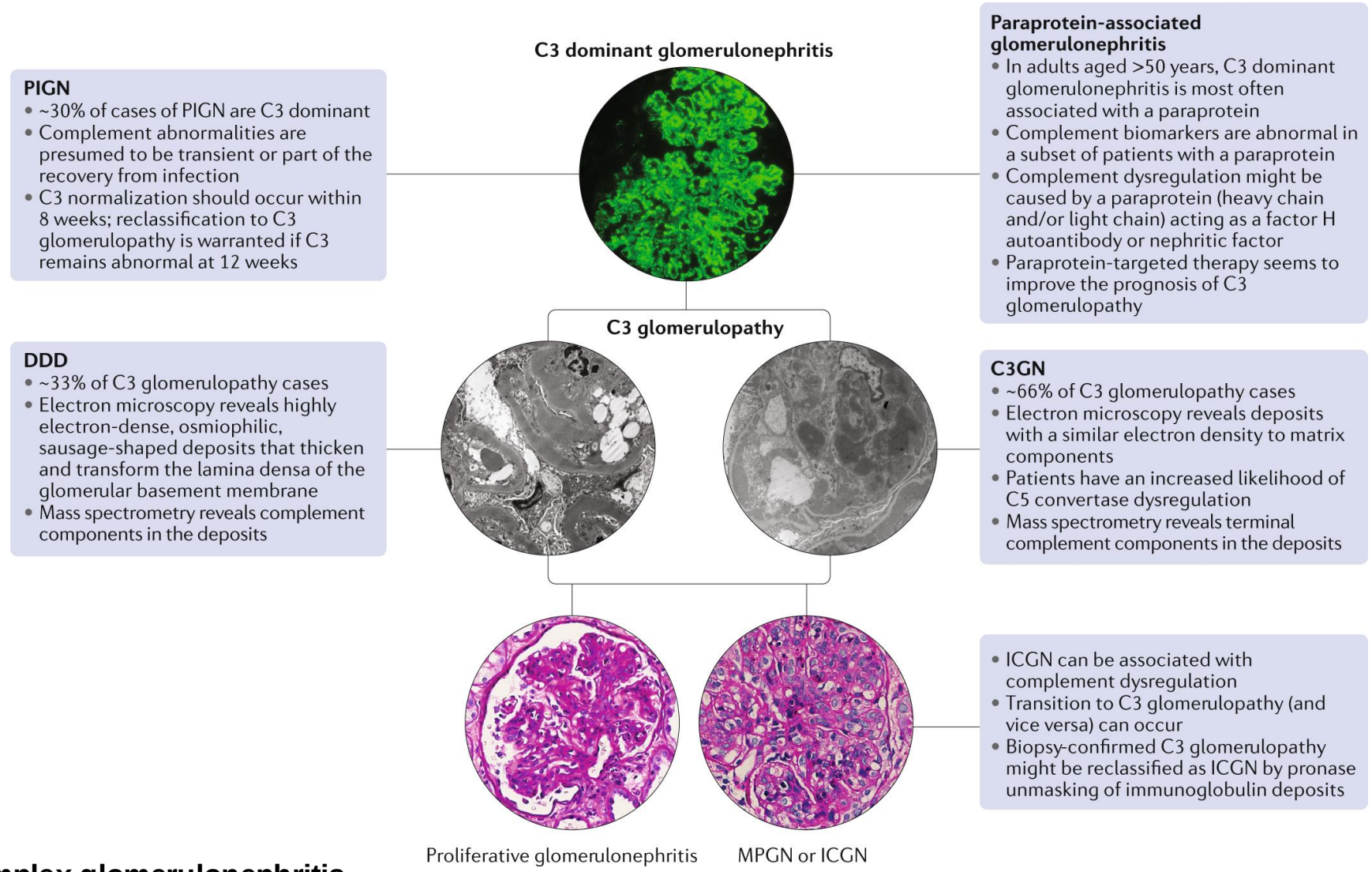
National Kidney Foundation. *Am J Kidney Dis.* 1998;31(6):E1. Health Travellers Worldwide. Accessed December 10, 2024. https://www.healthtravellersworldwide.com/treatments/kidney-biopsy/.

Diagnosis and Classification



Modified from: Pickering MC, et al. *Kidney Int*. 2013;84(6):1079-1089.

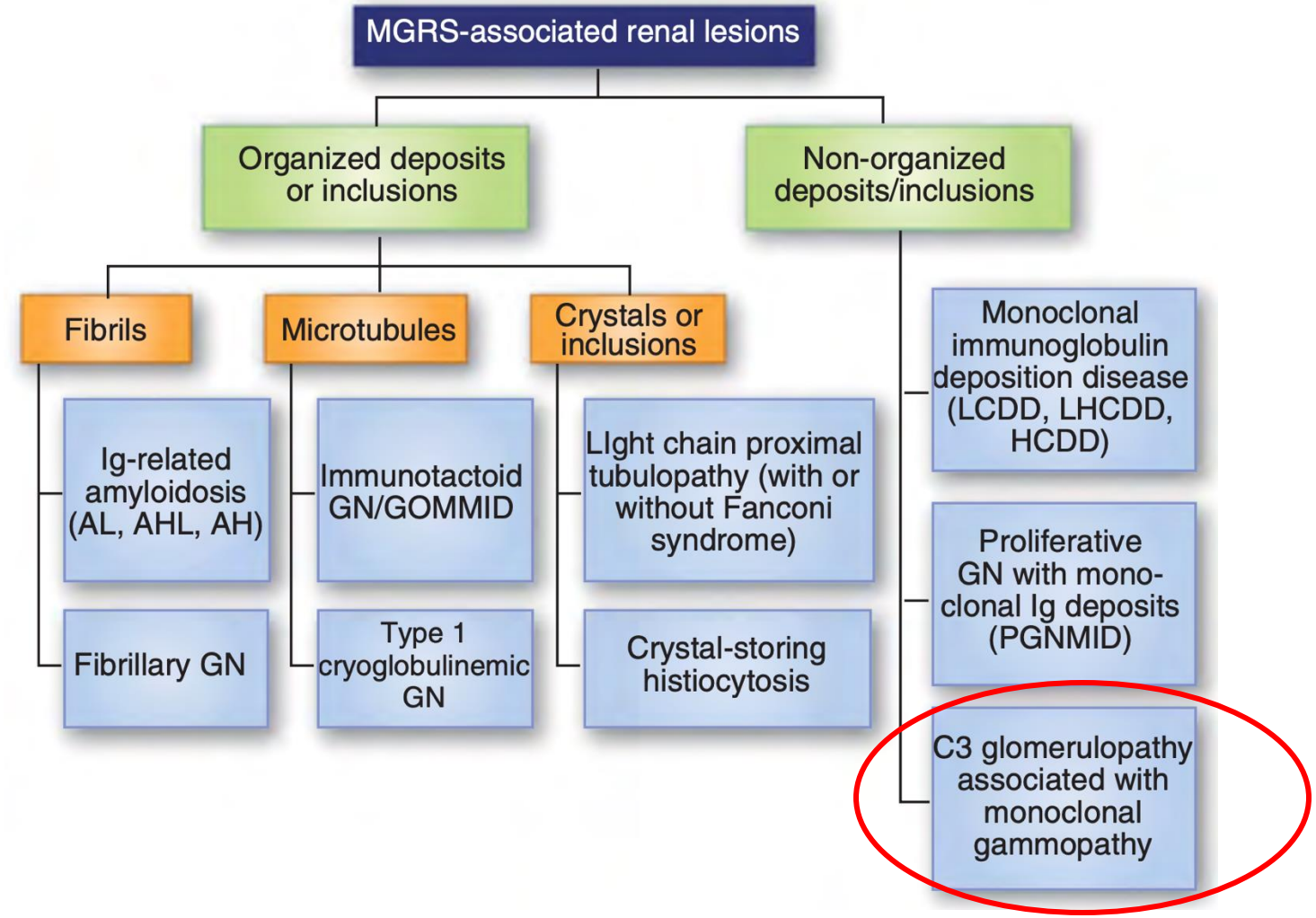
Differential Diagnosis



ICGN = immune complex glomerulonephritis.

Smith RJH, et al. *Nat Rev Nephrol*. 2019;15(3):129-143.

One of Many MGRS-Associated Kidney Diseases



MGRS = monoclonal gammopathy of renal significance; AL = immunoglobulin light chain amyloidosis; AHL = immunoglobulin heavy and light chain amyloidosis; AH = immunoglobulin heavy chain amyloidosis; GOMMID = glomerulonephritis with organized microtubular monoclonal immunoglobulin deposits; LCDD = light chain deposition disease; LHCDD = light and heavy chain DD; HCDD = heavy chain DD.

Bridoux F, et al. *Kidney Int.* 2015;87(4):698-711.

C3G Is Associated with Monoclonal Gammopathies

The prevalence is high in adult C3G patients

- 10/14 patients >49 years old
- 59-65% of patients >50 years old
- 94% of patients >70 years old
- Bone marrow biopsy
  - MGUS/MGRS (90%)
  - Multiple myeloma
  - Small lymphocytic lymphoma/CLL
  - No abnormality

MGUS = monoclonal gammopathy of undetermined significance; CLL = chronic lymphocytic leukemia.

Sethi S, et al. *Am J Kidney Dis*. 2010;56(5):977-982. Chauvet S, et al. *Blood*. 2017;129(11):1437-1447. Ravindran A, et al. *Kidney Int*. 2018;94(1):178-186.

Complement Workup in Patients with C3G

- Biopsy is definitive diagnosis
- Exclude
  - PIGN in pediatric patients
  - MGRS in patients >50 years old
- Serologies
  - Measure C3, C4, sC5b-9
  - Measure nephritic factors
  - Serum paraprotein (particularly for >50 years old)
  - Measure factor H levels
- Opthalmologic exam
- Screen for CFHR5 mutations

PIGN = post-infectious glomerulonephritis; CFHR = factor H-related protein.

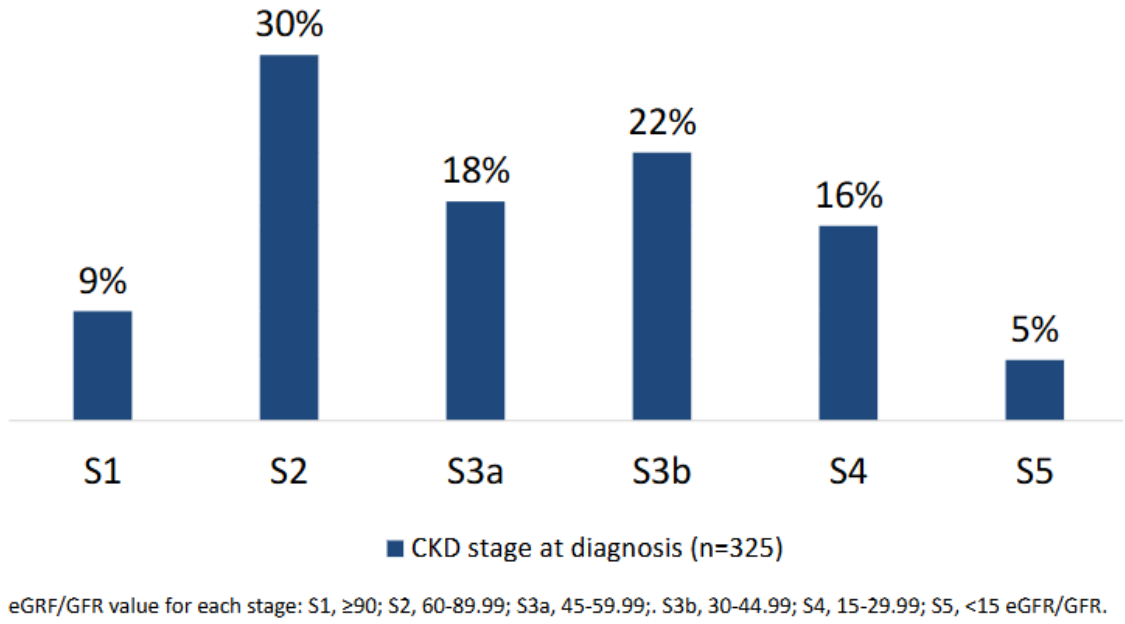
Goodship THJ, et al. *Kidney Int.* 2017;91(3):539-551.

Impact

Super rare, but really bad…

Time to Diagnosis

- Patients are waiting a long time for a correct diagnosis of C3G
- One study found that 10% of patients in their global study population waited more than 21.9 weeks
  - This may not even count the first time symptoms were seen, but not connected to C3G
  - The data collection method may also over-represent those who are likely to consult a physician, so total population results may be even higher

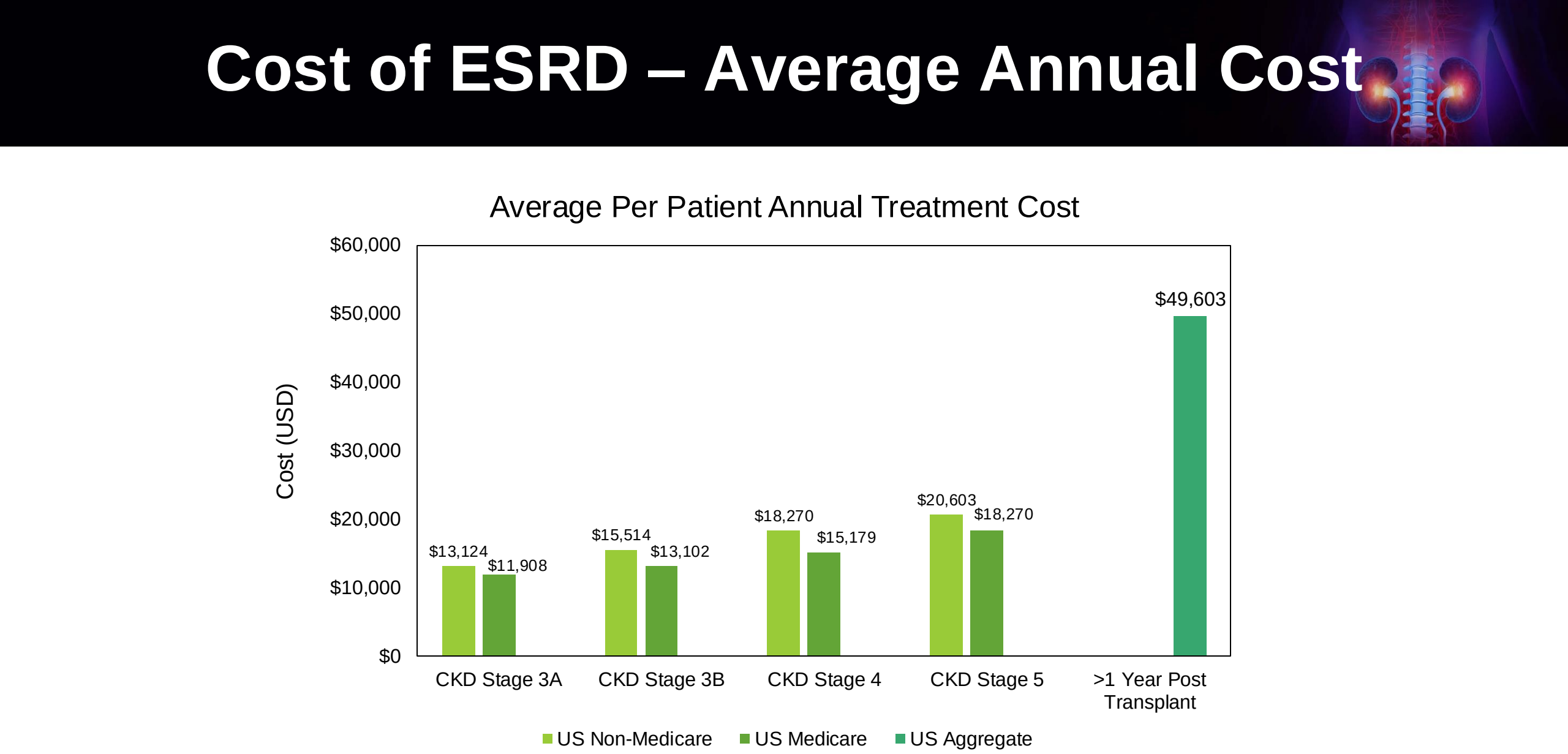


eGRF/GFR value for each stage: S1, ≥90; S2, 60-89.99; S3a, 45-59.99;. S3b, 30-44.99; S4, 15-29.99; S5, <15 eGFR/GFR.

CKD = chronic kidney disease.

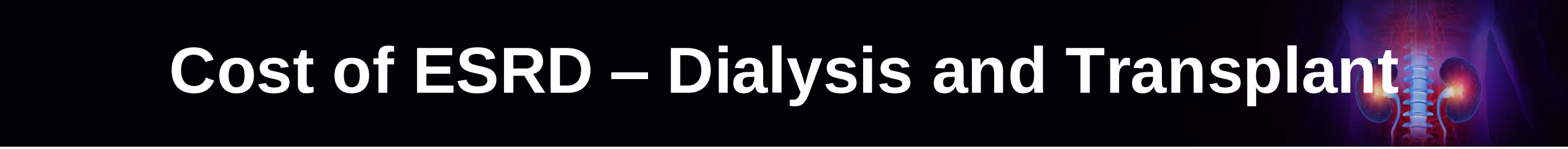
Proudfoot C, et al. Presented at: American Society of Nephrology (ASN) Kidney Week 2023; November 2-5, 2023; Philadelphia, Pennsylvania. Abstract TH-PO647.

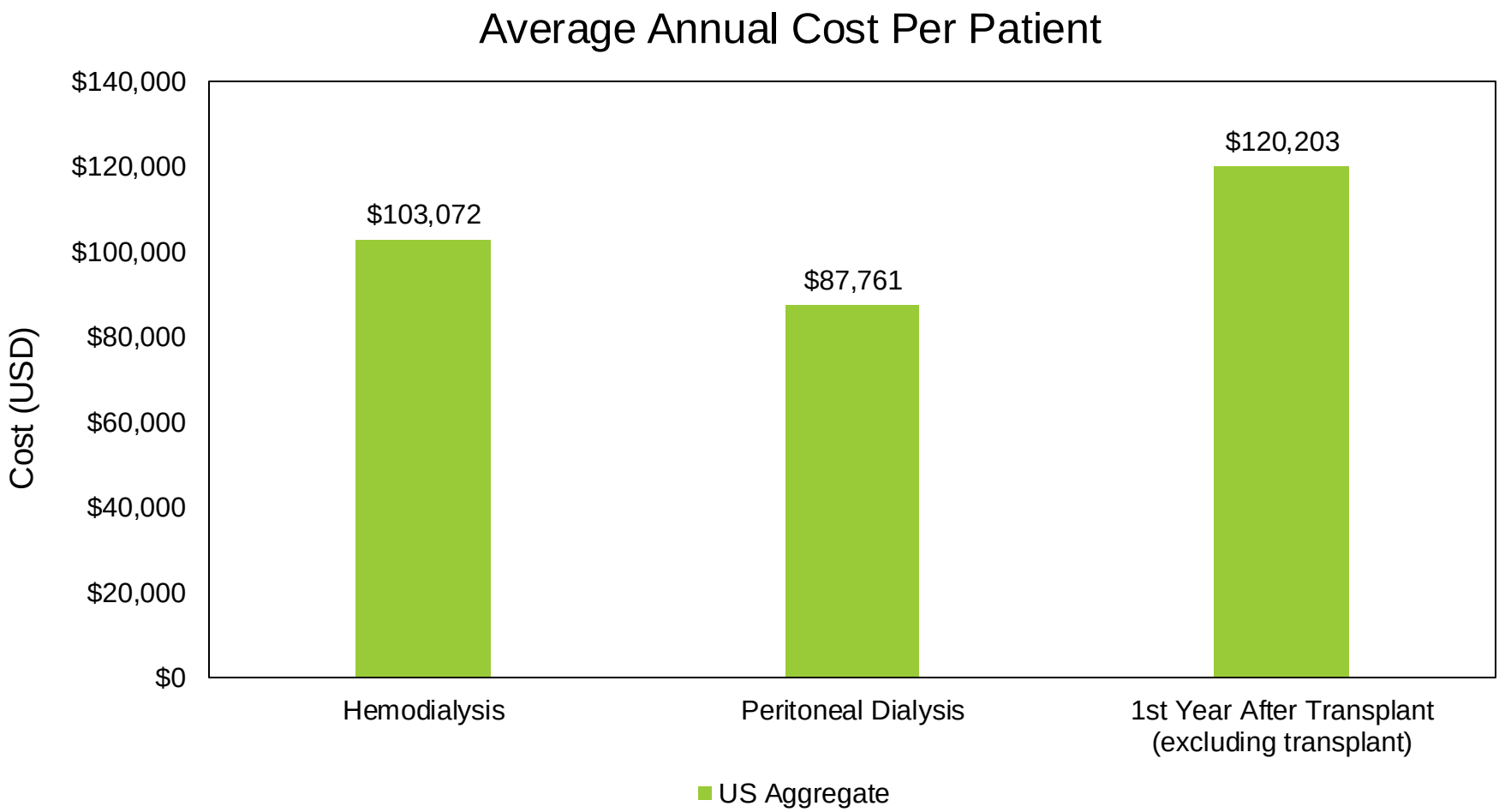
Cost of ESRD – Average Annual Cost



Jha V, et al. *Adv Ther*. 2023;40(10):4405-4420.

Cost of ESRD – Dialysis and Transplant

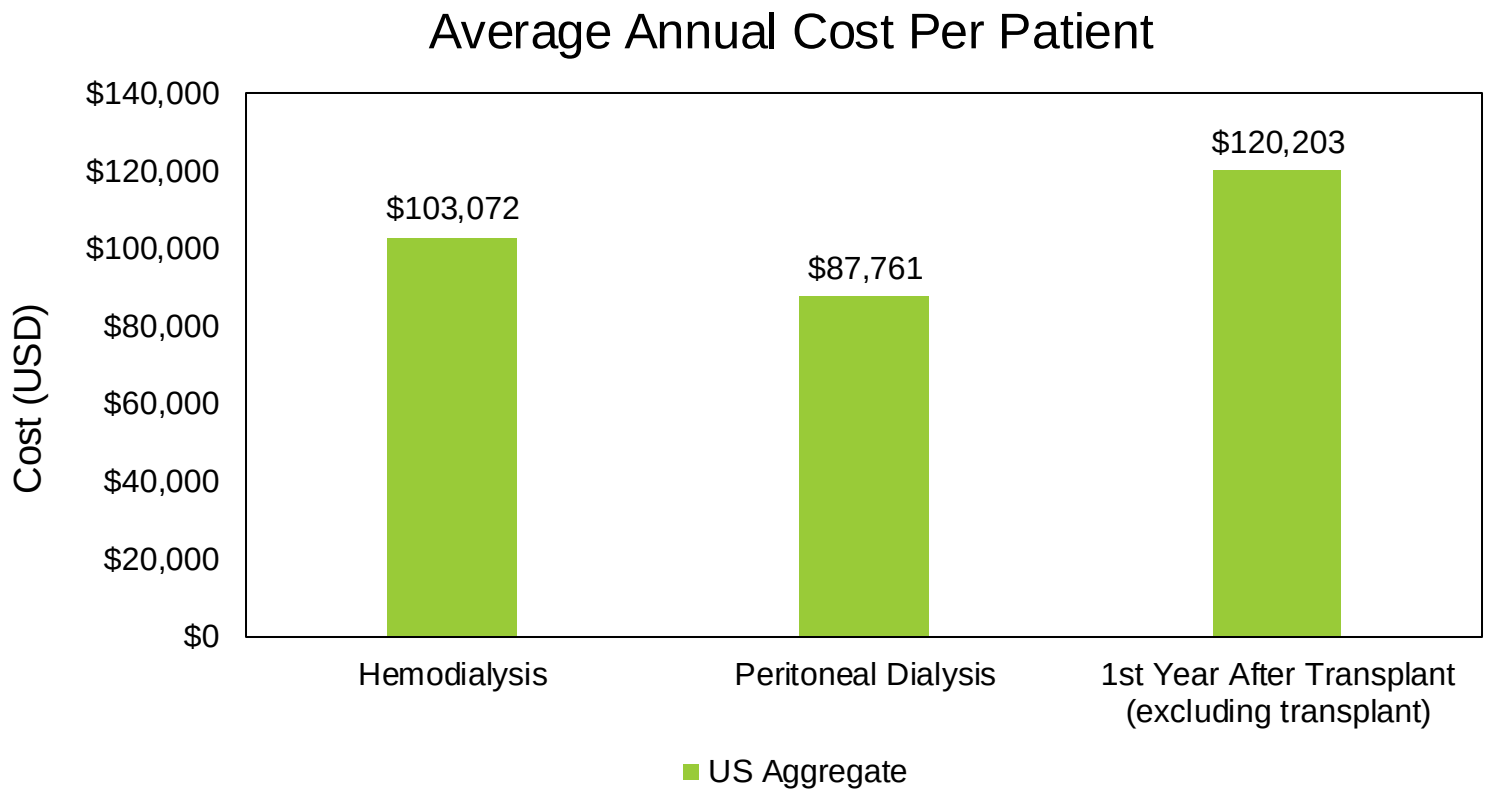




Jha V, et al. *Adv Ther*. 2023;40(10):4405-4420.

Cost of ESRD – Dialysis and Transplant

- Cost of average kidney transplant in 2020 was found to be $442,500
- Patients are waiting on average just around 2 years across all kidney transplant indications



Jha V, et al. *Adv Ther*. 2023;40(10):4405-4420.

Impact

- Morbidity
  - Hypertension and proteinuria
  - Side effects from medications
- End-stage kidney disease
  - C3G probably accounts for <0.1% of ESKD
  - Worst prognosis of the primary forms of glomerulonephritis
  - Approximately 50% of patients → ESKD within 10 years
- Transplant
  - Overall time to allograft loss is 42 months
  - Recurrence in up to 67% of transplant recipients within 2-3 years
  - Median graft failure within 18 months after recurrence

Smith RJH, et al. *Nat Rev Nephrol*. 2019;15(3):129-143. Zand L, et al. *J Am Soc Nephrol*. 2014;25(5):1110-1117. Regunathan-Shenk R, et al. *Am J Kidney Dis.* 2019;73(3):316-323.

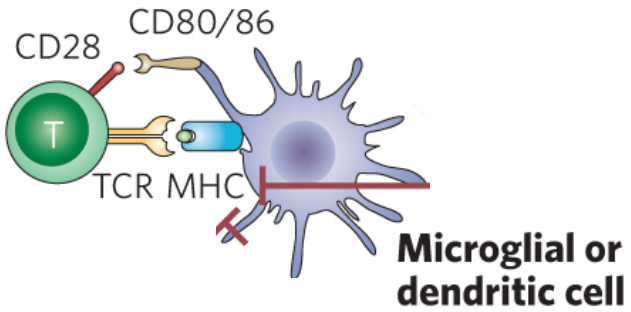
Emerging Treatments for C3G: A Focus on Complement Inhibitors

Treatment Challenges – How to Block Complement?

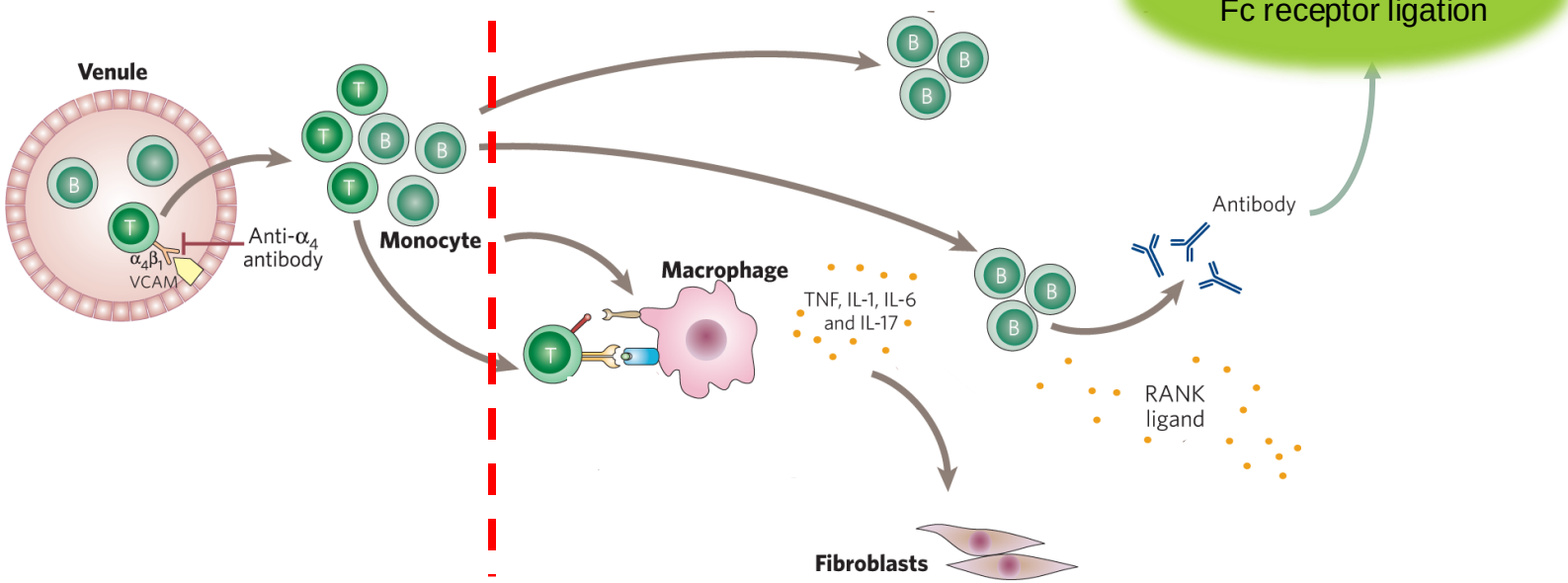
Cytotoxics

Corticosteroids

Initiation of autoimmunity



Mediators of tissue inflammation



TCR = T-cell receptor; MHC = major histocompatibility complex; TNF = tumor necrosis factor.
Modified from: Feldmann M, et al. *Nature*. 2005;435(7042):612-619.

Treatment of C3G – General Supportive Care

All patients

- BP control with RAAS inhibition
- SGLT2i?
- GLP1 agonists?

BP = blood pressure; RAAS = renin-angiotensin-aldosterone system; SGLT2i = sodium-glucose cotransporter-2 inhibitor; GLP = glucagon-like peptide.

Treatment of C3G – Standard Immunosuppression

Paradox: C3G is defined by the relative absence of immune complexes in the glomeruli…

But it is associated with certain antibodies

- Nephritic factors
- Other autoantibodies
- Paraprotein

Treatment of C3G – Standard Immunosuppression

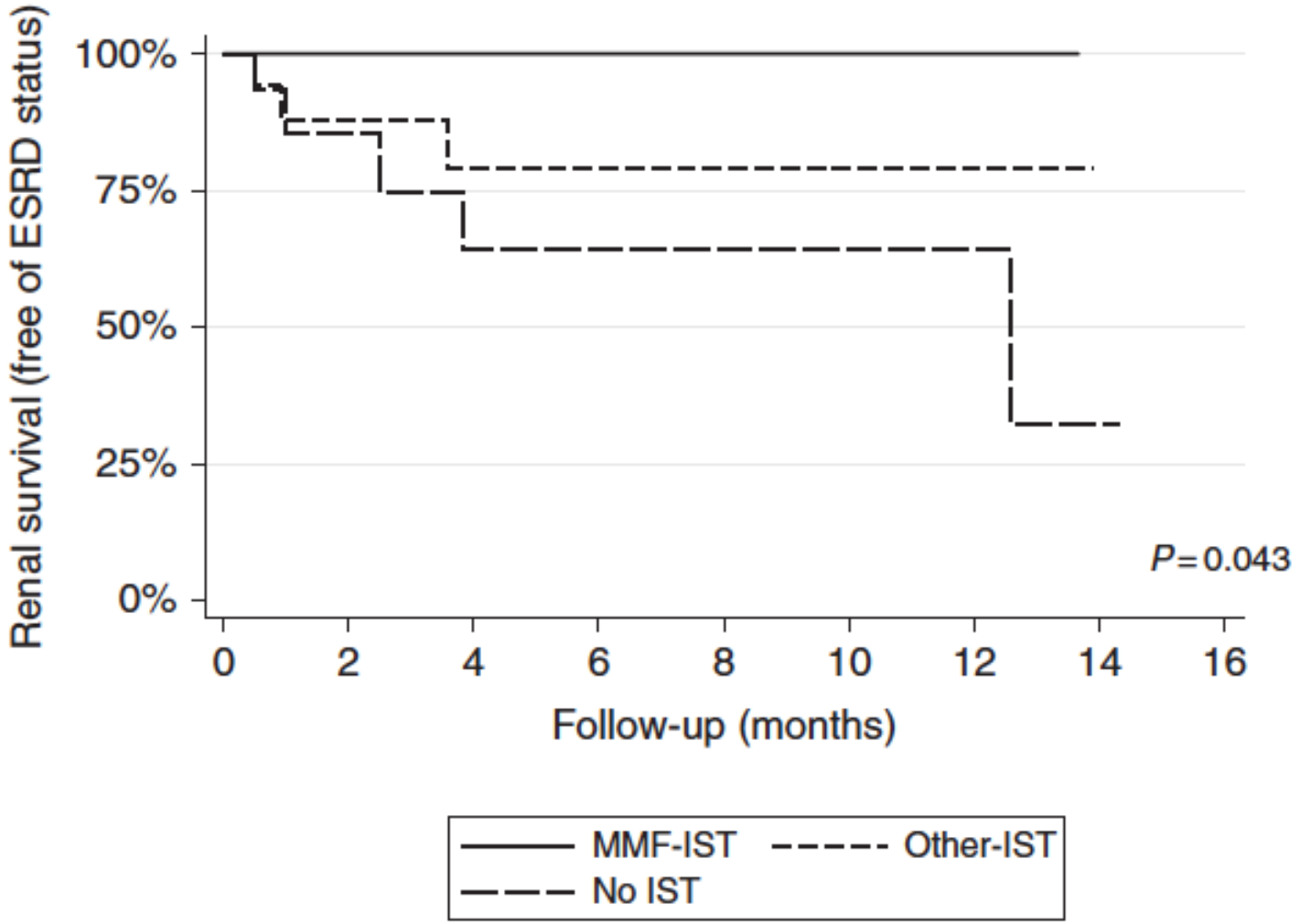
Moderate disease

- UPC >500 mg
- Inflammation on biopsy
- Rising serum Cr
- → MMF and prednisone

UPC = urine protein to creatinine ratio; Cr = creatinine; MMF = mycophenolate mofetil.

Goodship THJ, et al. *Kidney Int.* 2017;91(3):539-551.

Does MMF Improve Outcomes in C3G?

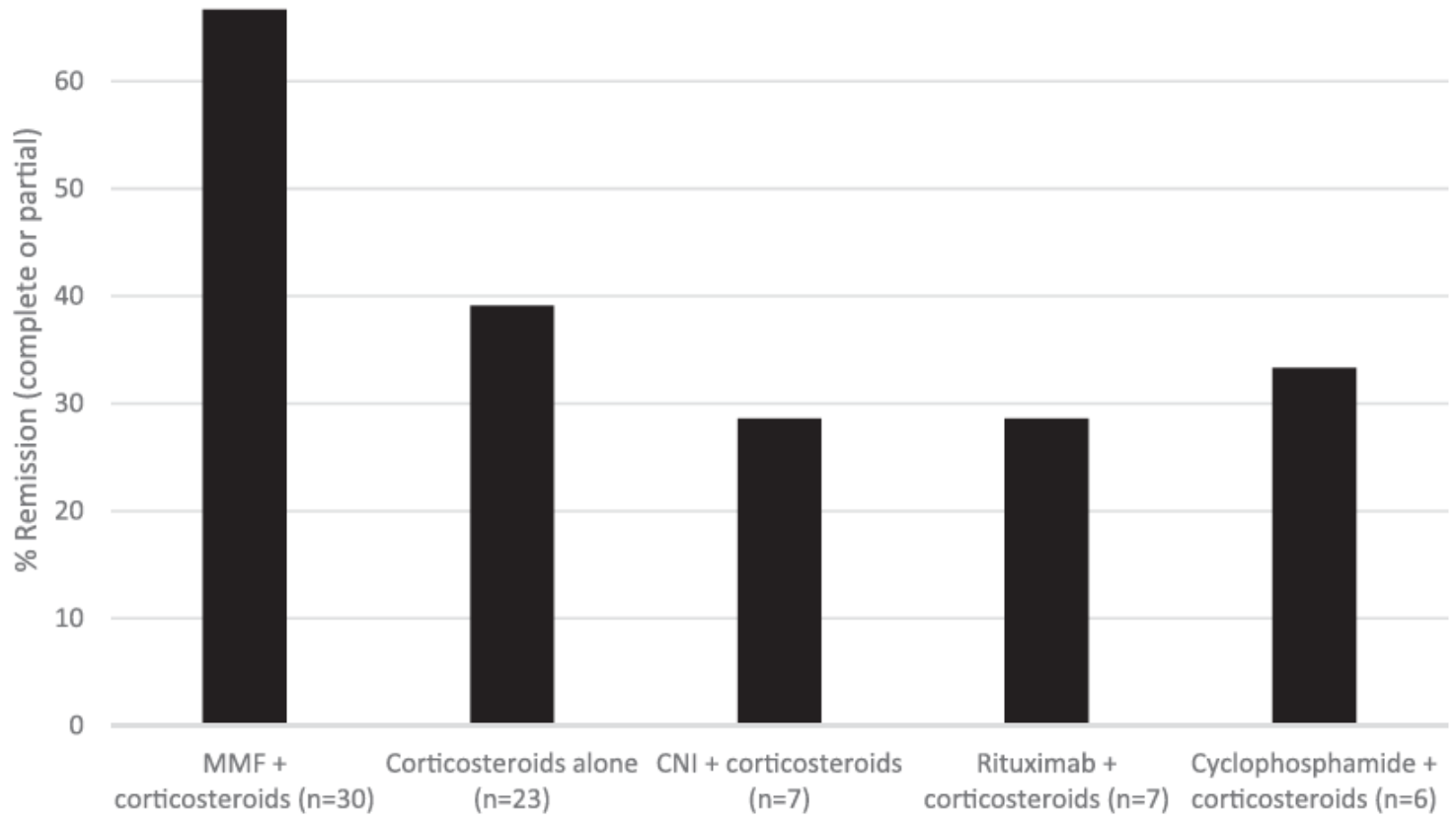


ESRD = end-stage renal disease; IST = immunosuppressive treatments.

Rabasco C, et al. *Kidney Int*. 2015;88(5):1153-1160.

Does MMF Improve Outcomes in C3G?

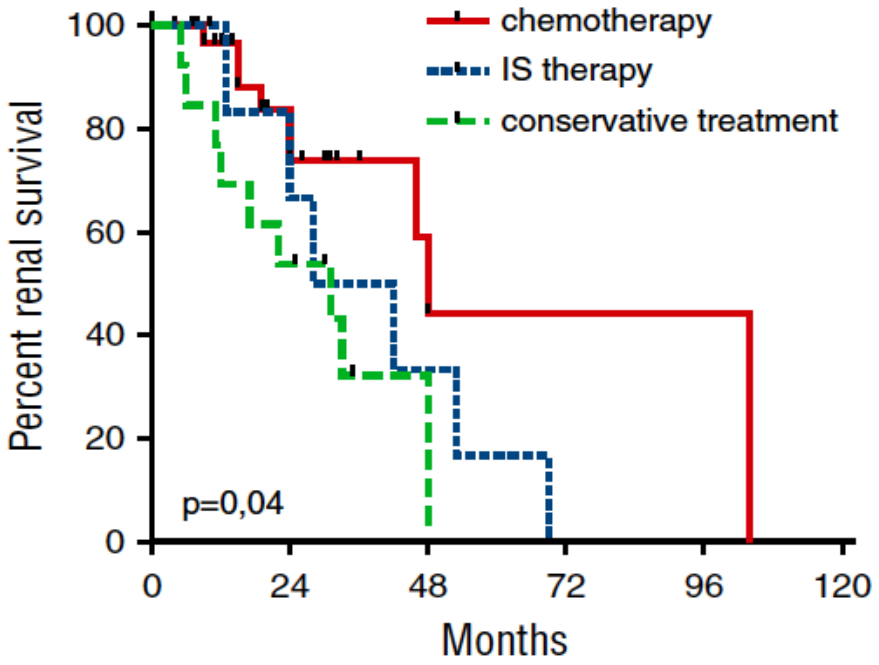
- 30 patients
- Remission in 67%

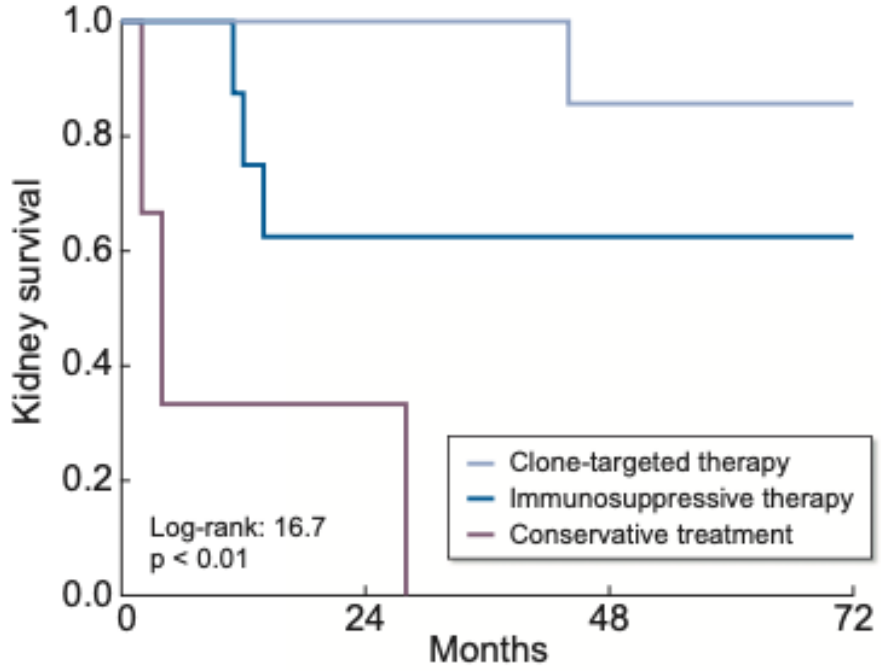


Avasare RS, et al. *Clin J Am Soc Nephrol.* 2018;13(3):406-413.

Treating MG-Associated C3G

Chemotherapy superior to standard immunosuppression





IS = immunosuppression.

Chauvet S, et al. *Blood*. 2017;129(11):1437-1447. Caravaca-Fontan F, et al. *Nephrol Dial Transplant*. 2022;37(11):2128-2137.

Treatment of C3G – Complement Inhibition

Severe disease

- UPC >2000 mg
- Severe inflammation on biopsy
- Rapidly progressing disease
  - → IV methylprednisolone, MMF, or other anti-cellular therapy
  - ?eculizumab or other complement inhibitors

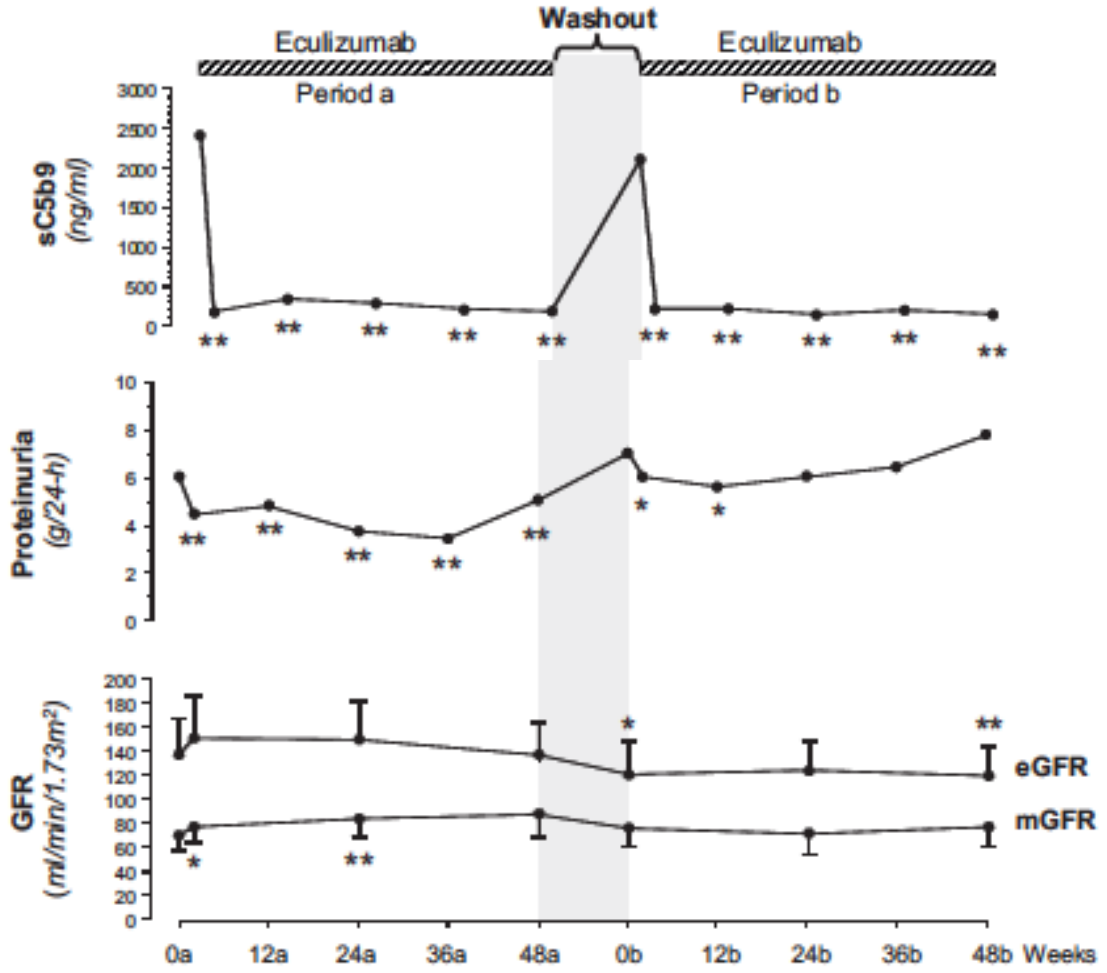
IV = intravenous.

Goodship THJ, et al. *Kidney Int.* 2017;91(3):539-551.

Is Eculizumab Beneficial in C3G?

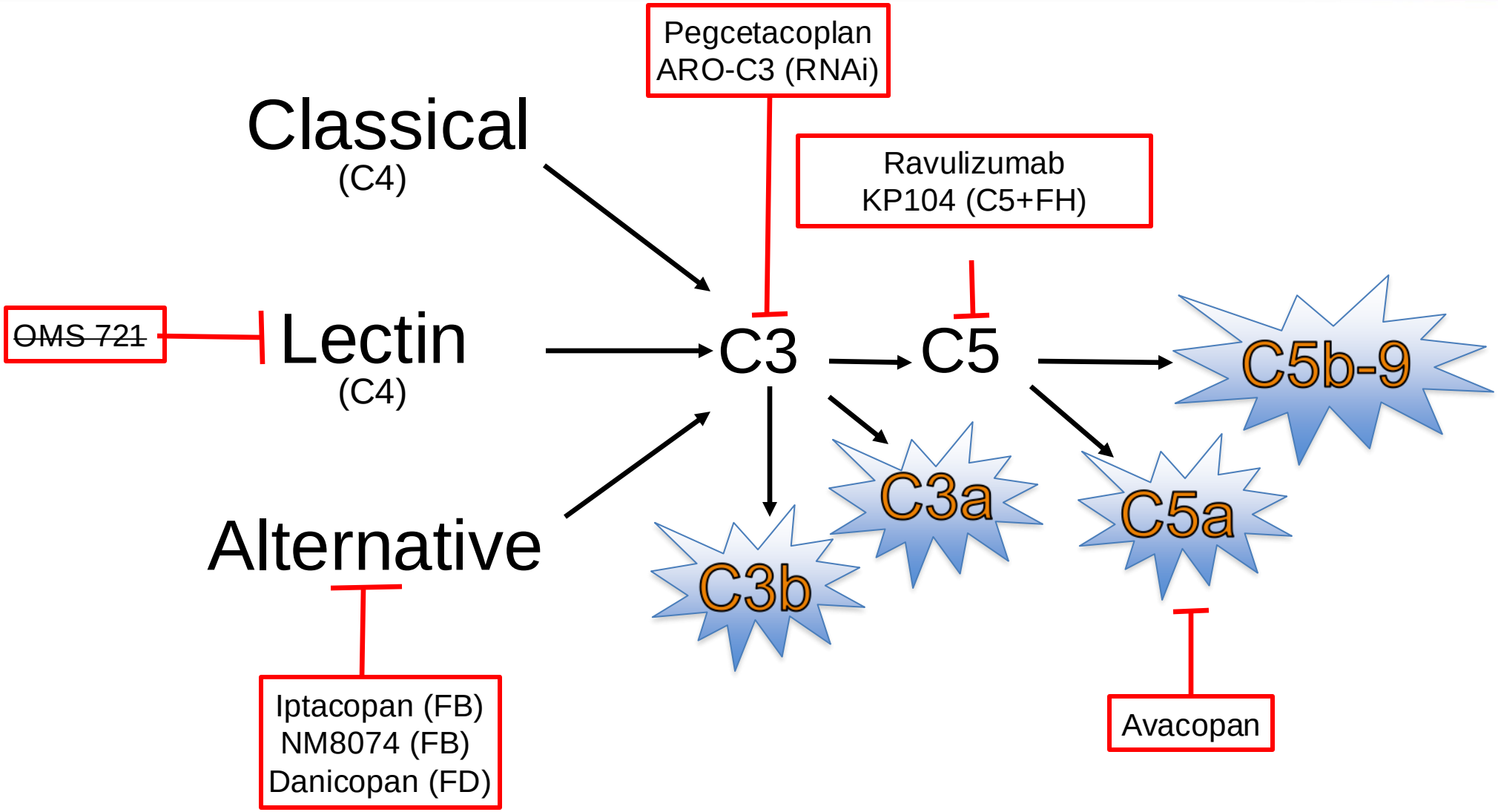
EAGLE Study

- 10 patients
- 2x 48-week treatment periods, with 12-week washout between
- 3 patients in partial remission
- GFR relatively stable in all patients



GFR = glomerular filtration rate; e = estimated; m = measured; sC5b9 = serum complement membrane attack complex.
Ruggenenti P, et al. *Am J Kidney Dis*. 2019;74(2):224-238.

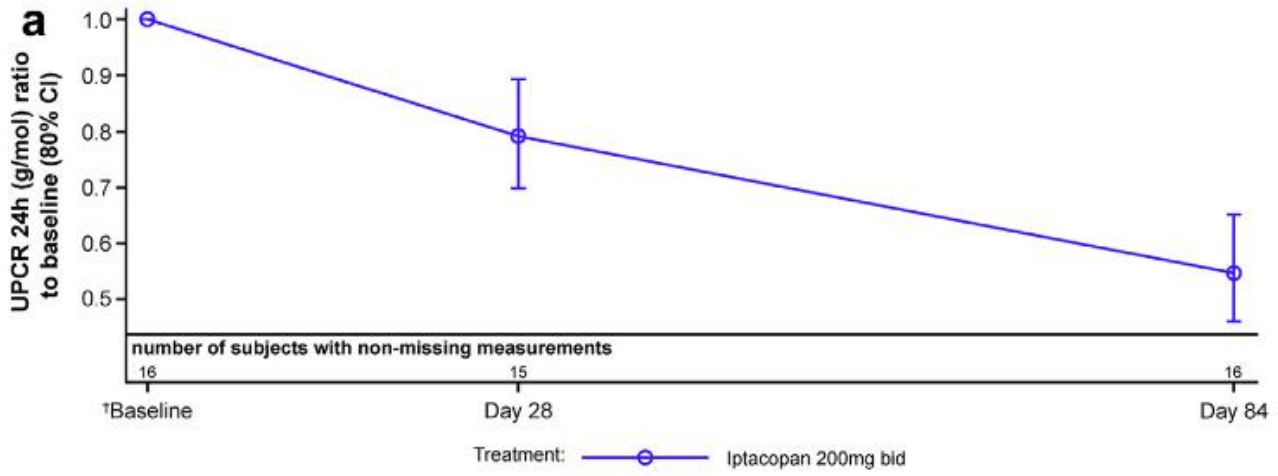
Treatment of C3G – Other Complement Inhibitors

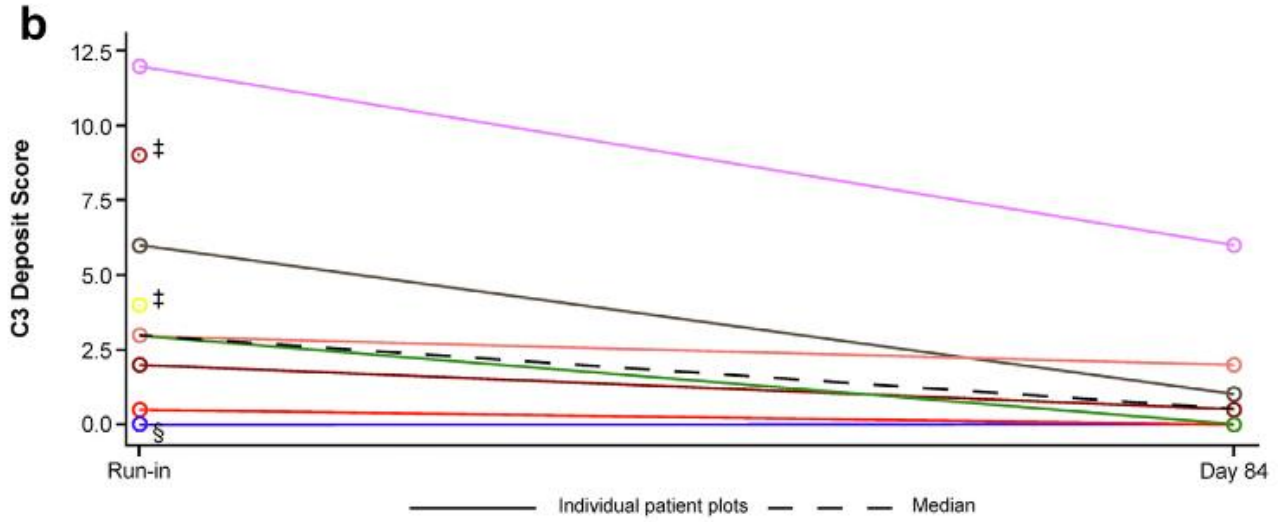


RNAi = RNA interference.

Modified from: Thurman JM, et al. *Kidney Int*. 2016;90(4):746-752.

Iptacopan – Alternative Pathway Inhibitor





Phase 2 open label study

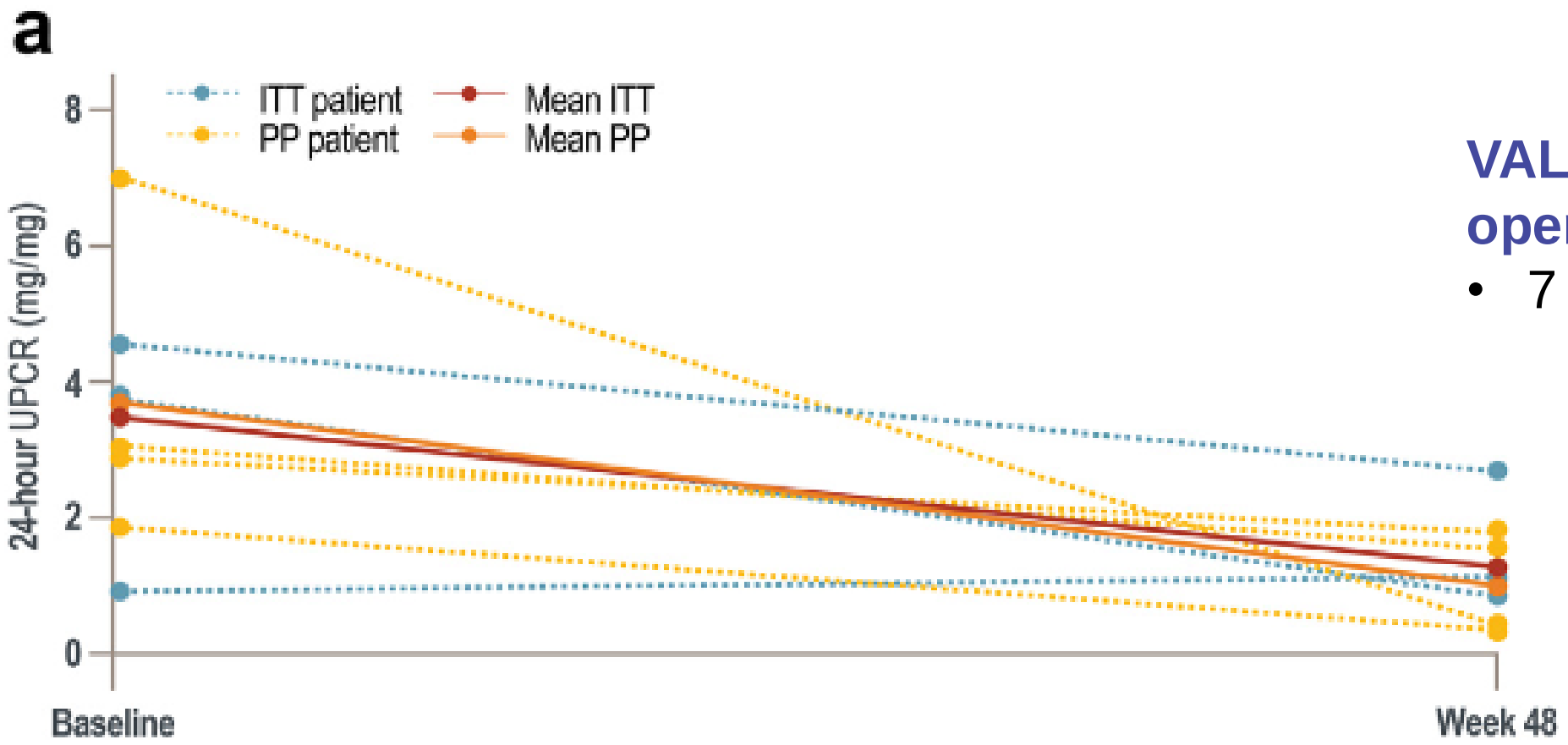
- 27 patients

C3 deposit score: 0-3

- Mes
- Capillary
- x2 if global

Wong E, et al. *Kidney Int Rep*. 2023;8(12):2754-2764.

Pegcetacoplan – C3 Inhibitor



VALIANT – Phase 2 open label study

- 7 patients

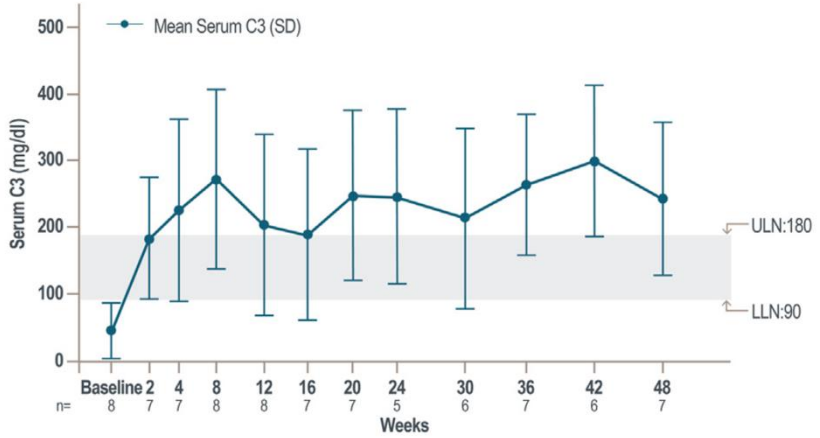
PP = per protocol; ITT = intention to treat.

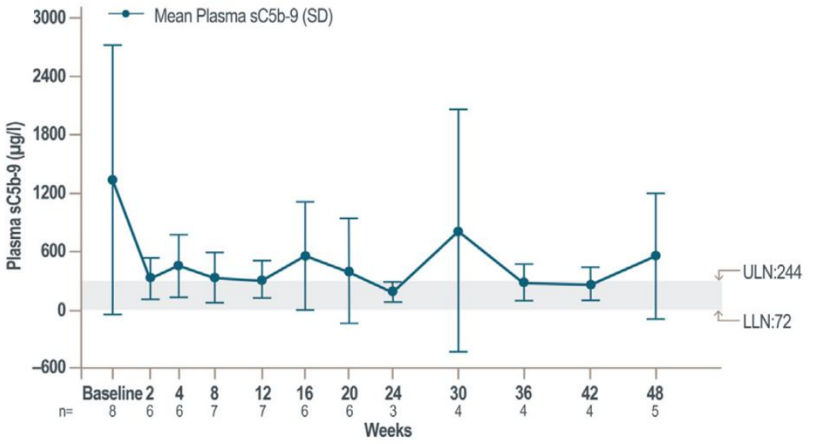
Dixon BP, et al. *Kidney Int Rep*. 2023;8(11):2284-2293. NIH. Accessed December 10, 2024. https://clinicaltrials.gov/study/NCT05067127.

Pegcetacoplan – C3 Inhibitor

Table 2. Primary end points at baseline and week 48 for patients with C3G (ITT and PP populations)

Parameter, mean (SD)	ITT	PP
Baseline[a]		
Number of patients	8	4
24-hour UPCR, mg/mg	3.3 (1.7)	3.5 (2.1)
Week 48		
Number of patients	7	4
24-hour UPCR, mg/mg	1.2 (0.8)	1.0 (0.7)
Individual CFB (SD) in 24-hour UPCR, mg/mg[b]	−2.0 (2.0)	−2.5 (2.5)
Individual %CFB (SD) in 24-hour UPCR[b,c]	−50.9 (39.1)	−65.4 (26.4)

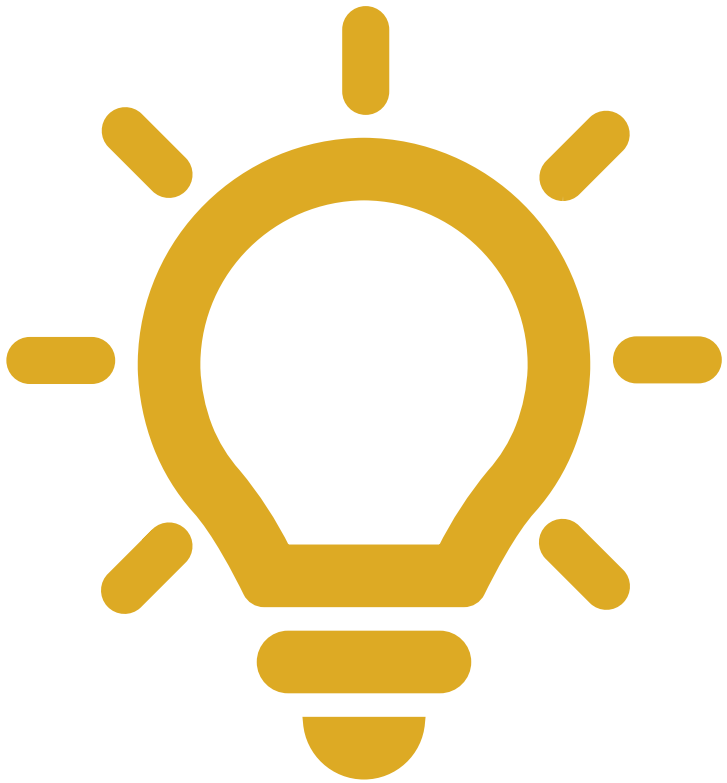




Patients with native or recurrent C3G or IC-MPGN receiving pegcetacoplan showed a statistically significant and clinically meaningful reduction in proteinuria.

[a]Baseline was the most recent result prior to the first dose; [b]The means were calculated at each visit with only non-missing values; [c]The %CFB was determined for each individual patient as induvial CFB divided by baseline UPCR, and then th emean of these individual %CFB was calculated.
SD = standard deviation; CFB = change from baseline.
Dixon BP, et al. *Kidney Int Rep*. 2023;8(11):2284-2293.

Key Learning Points



- C3G is caused by complement-mediated injury of the glomerulus
- Dysregulation via alternate complement pathway
  - Can be driven by autoantibodies
  - Associated with monoclonal gammopathies
- Definitive diagnosis is made by kidney biopsy (IF and EM)
  - Serologic and genetic workup is recommended (but controversial)
- There may be a role for conventional immunosuppressives (eg, MMF)
- Complement inhibitory drugs are currently being tested in clinical trials

Managed Care Considerations for C3G

The Future Pharmaceutical Costs

- Current annual global market size (revenue) is estimated to be $45 million
  - Reported that North America alone represents 36% of this size
  - With few pharmaceutical options, we can infer that this is likely driven by medical and procedure costs
- By 2036, it is estimated to grow to $68 million annually ($23 million increase)
  - Reported that growth is expected to be driven by increased incidence of kidney disorders, “growing research” AKA new pharmaceutical products expected to be employed, and an increased awareness as new treatments become available
  - The US market proportion is expected to remain stable at 36% meaning $8.3 million per year is expected to be tied to these growth drivers

Research Nester. Accessed December 5, 2024. https://www.researchnester.com/reports/complement-3-glomerulopathy-market/5781.

Optimizing Cost-Effective Best Clinical Practices

- Encouraging and facilitating early diagnosis and early intervention
  - Catching the patients before progression to later stages of CKD optimizes both financial, clinical, and quality of life outcomes for the patient
- Minimize patient progression to ESRD after diagnosis
  - ACE/ARB therapy may not be the golden bullet, but appears to slow progression for at least some patients
  - Although the data is not terribly robust for C3G specifically, other non-pharmacologic best practice options for kidney disfunction should be strongly considered: diet, hydration, low sodium intake, controlling blood pressure, physical activity, etc.
- Value-based care focusing on patients where the evidence indicates likely good clinical outcomes

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker.

Ndife BC, et al. Presented at: ASN Kidney Week 2023; November 2-5, 2023; Philadelphia, Pennsylvania. Abstract TH-PO643. Rovin BH, et al. *Kidney Int*. 2021;100(4S):S1-S276. KDIGO. Accessed December 5, 2024. https://kdigo.org/guidelines/ckd-evaluation-and-management/.

Cost-Effective Considerations For Novel Therapy

- Consider known predictors of progression to ESRD
  - Proteinuria, kidney function at diagnosis, change in proteinuria over time, and features found in biopsy
- As therapies emerge, data on identifying the correct patient for the correct mechanism will be imperative
  - This could include considerations like genetic testing or complement assays
- Drug pricing vs annual cost of care vs cost avoidance vs QoL will create a delicate balance to determine what cost-effectiveness will look like

Estebanez BT, et al. *Kidney Int Rep*. 2023;9(3):569-579.

Patient-Centered Care

- Shared decision making has been a pivotal part of increasing patient satisfaction and perceived quality of life among patient with CKD nearing kidney transplant
  - The patient experience represents a significant portion of the burden of C3G, making parallels to seeing decreases in the overall burden by focusing on patient-centered care a minor jump
- As novel therapies become available, having frank discussions about the treatment, clinical effects, and potential adverse effects can help identify those who may or may not be great candidates for treatment
- Optimal outcomes are often dependent on the patient understanding the differences between potential decisions

Ladin K, et al. *Am J Kidney Dis*. 2022;79(5):623-625. Davis AD. Published March 31, 2021. Accessed December 3, 2024. https://www.acpdecisions.org/bringing-a-patient-centered-approach-to-kidney-disease-care-to-the-forefront-three-reasons-why-it-matters/.

Key Learning Points

- Novel therapies are likely to represent a real budget impact, and multiple factors will need to be considered when evaluating the most cost-effective use
- New pharmacologic options show promise of offering optimal patient outcomes for patients needing treatment beyond today’s options
- Patient-centered care with shared decision making is a crucial part of ensuring patient outcomes and increased quality of life