ORGANIZADO POR:

Evidenze

63rd ERA CONGRESS
GLASGOW & VIRTUAL
JUNE 3-6, 2026
Challenge Your Thinking

Lo más relevante de ERA 2026 en C3G e IC-MPGN

C3 Academy

Dra. Ana Huerta Arroyo |
Hospital Universitario Puerta de Hierro, Majadahonda

sobi rare strength

AMPLIANDO EL HORIZONTE
EN EL ABORDAJE DE LAS **NEFROPATÍAS RARAS.**

Conflictos de Interés

He recibido honorarios por colaboración en docencia, formación y/o asesoría científica de Sobi, Novartis, AstraZeneca, GSK, Otsuka y Vifor.

I. Glomerulopatía C3:

Comunicaciones orales:

- **Primary and secondary GN, vasculitis and autoimmune diseases. A case-based discussion (C3G or IC-MPGN).** *Marina Vivarelli.*
- **Pegcetacoplan sustained clinical benefit in C3G and primary IC-MPGN through 1 year of therapy: Data from VALIANT/VALE.** *Fadi Fakhouri.*
- **Pegcetacoplan Treatment Response in VALIANT/VALE: Impact of Genetic and Acquired Complement Dysregulation and Disease Type in C3G and Primary IC-MPGN.** *Marina Noris.*
- **Real-world clinical profile, treatment patterns andoutcomes associated with C3G and primary IC-MPGN: Insights from the UK RaDaR registry.** *Lexy Sorrel.*

Pósteres

II. El complemento en otras enfermedades glomerulares

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A case-based discussion: C3G and Ig-MPGN

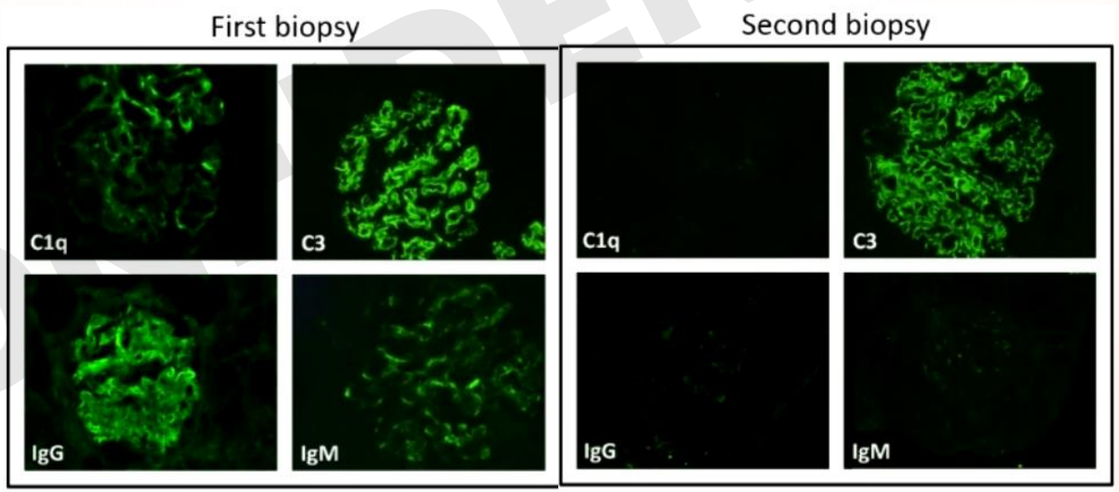
Marina Vivarelli
Bambino Gesù Children's Hospital IRCCS Rome, ITALY

sobi

El valor de las biopsias de repetición: de IC-MPGN a C3G

Niña de 13 años: ratio de proteinuria/creatinina (UPCR 3,3), hematuria microscópica función renal normal (Cr 0,7 mg/dL), C3 y C4 séricos bajos, ANA ligeramente positiva.

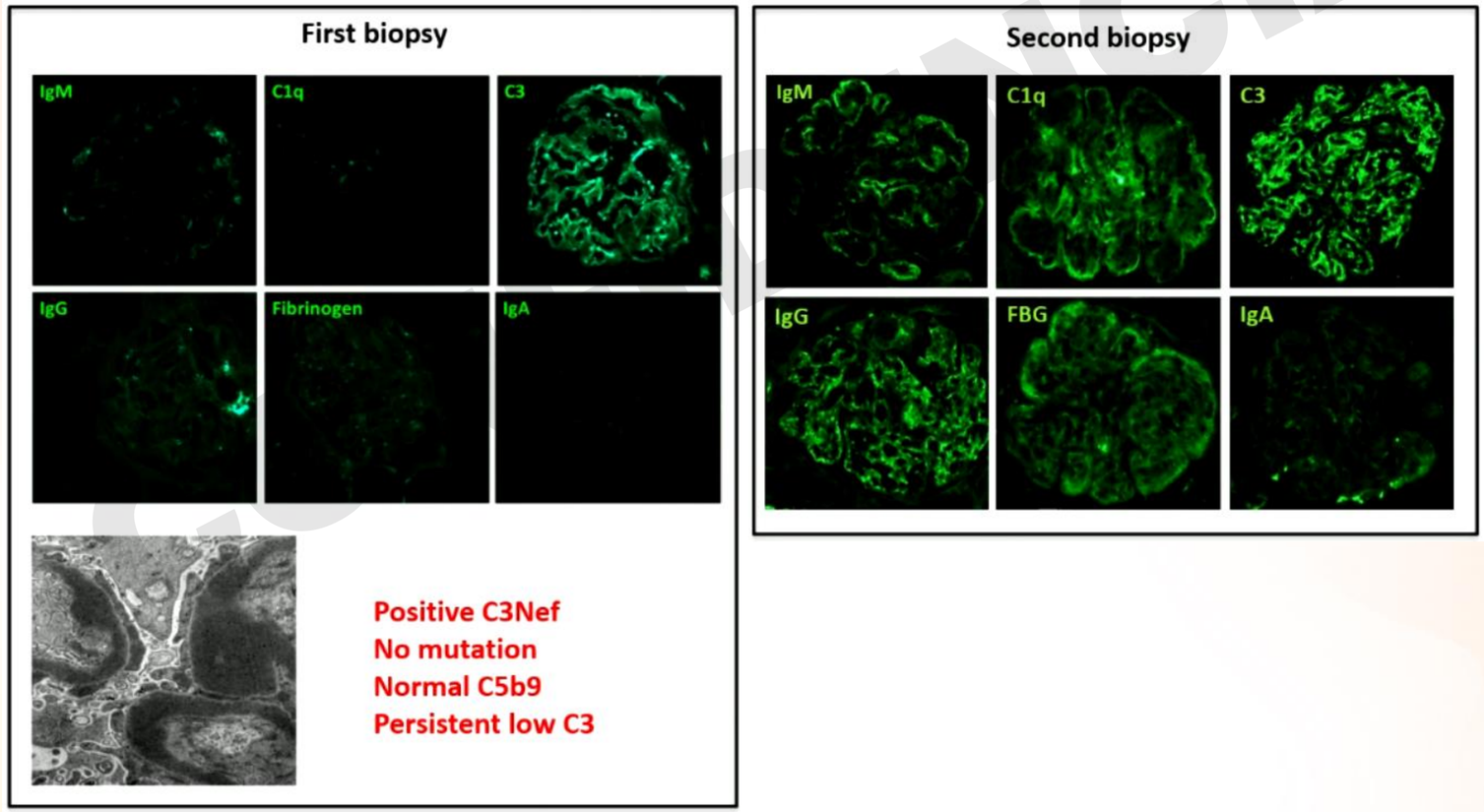
C3Nef positivo
Mutación *MCP*
C5b9 elevado
C3 bajo persistente



Cortesía del Dr. F. Diomedi-Camassei.

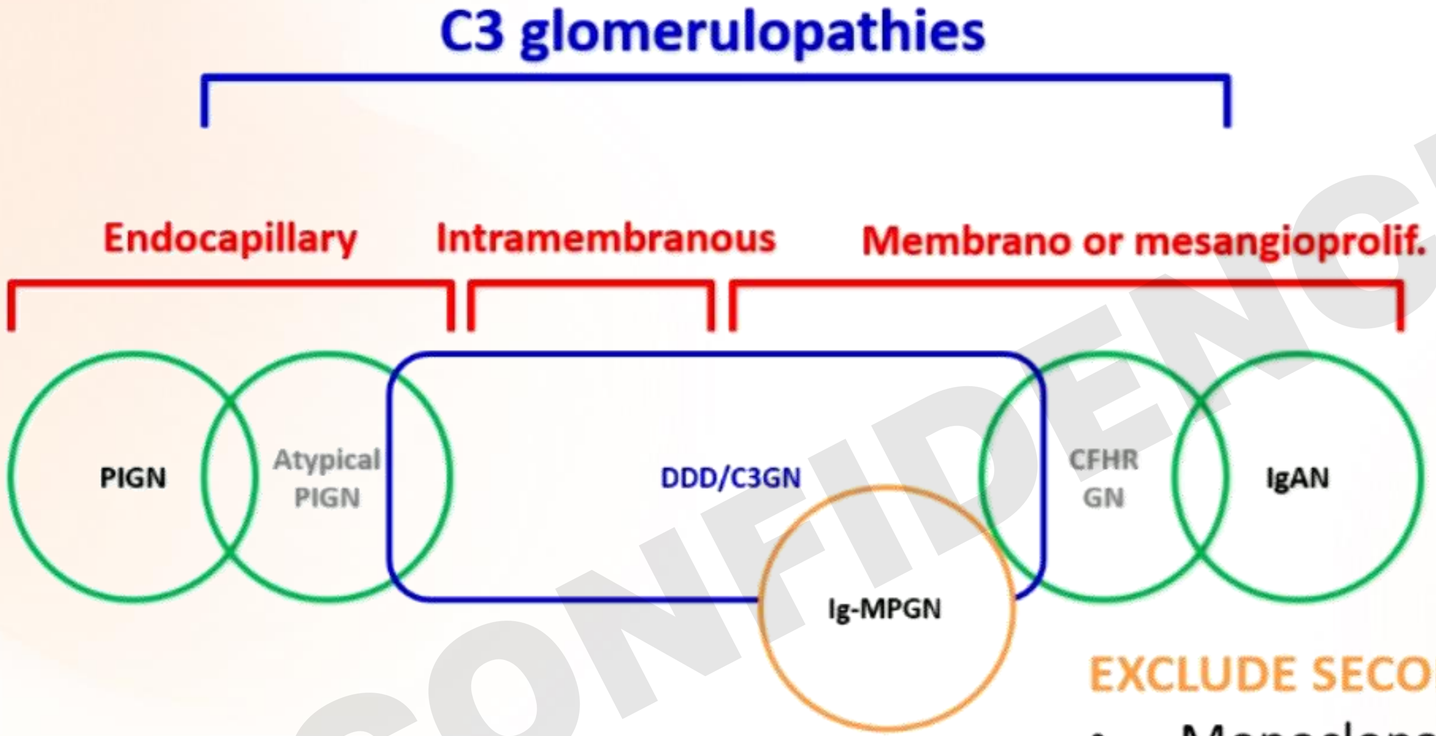
El valor de las biopsias de repetición: de IC-MPGN a C3G

Niño de 8 años: ratio proteinuria-creatinina nefrótica (UPCR 5,4), hematuria microscópica función renal normal (Cr 0,5 mg/dL), C3 sérico bajo.



Cortesía del Dr. F. Diomedi-Camassei.

La nueva clasificación de C3G/Ig-MPGN



EXCLUDE SECONDARY FORMS!

- Monoclonal gammopathy
- Infection-related GN
- Vasculitis (SLE)

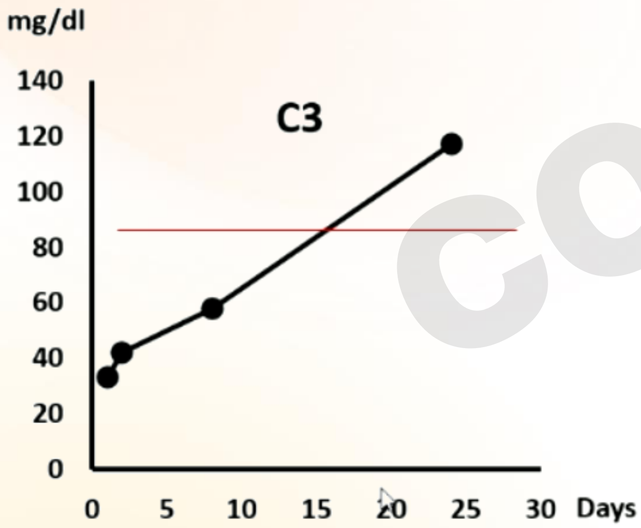
C3 deposition often predominant, not exclusive

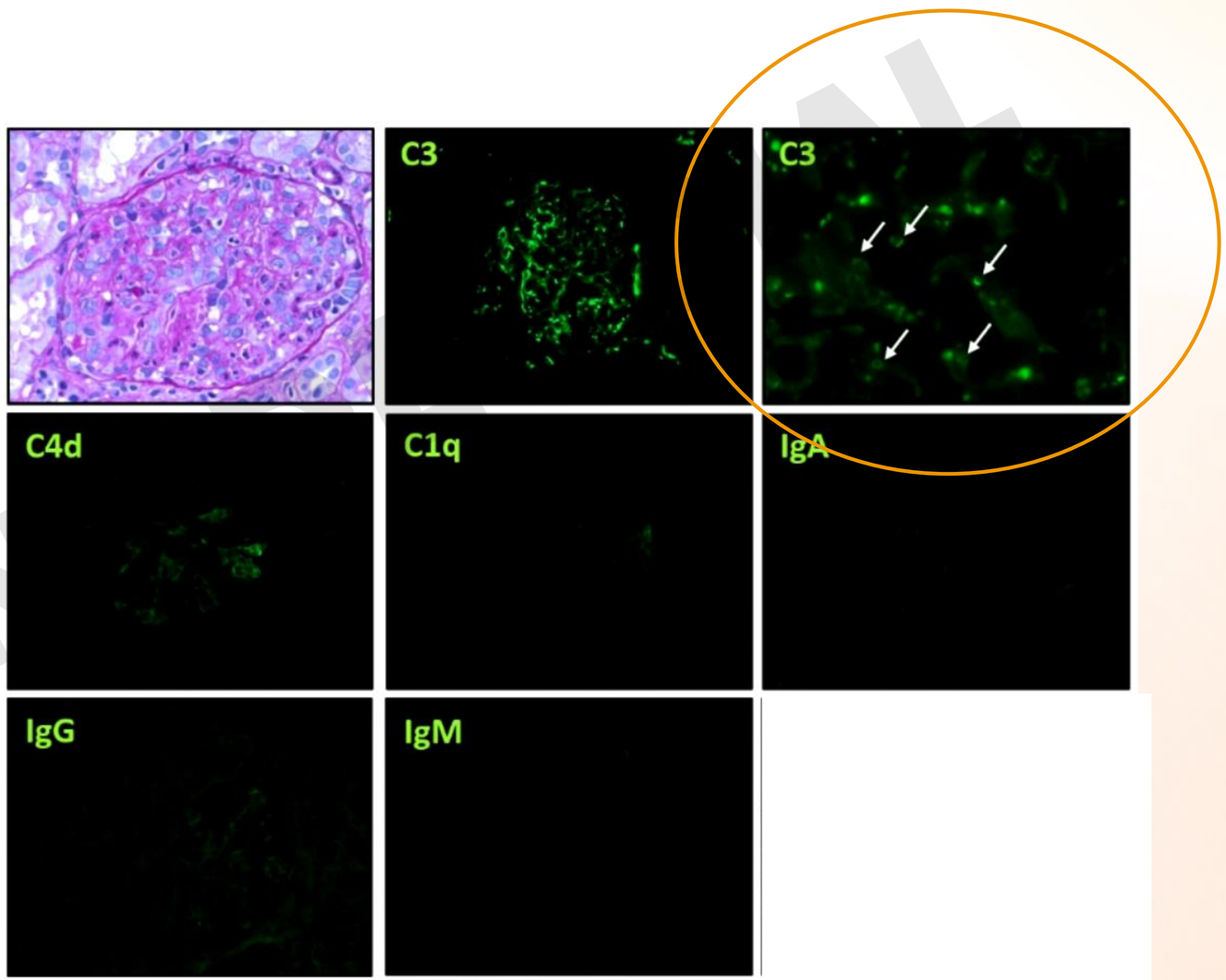
Adaptado de Zipfel et al., Molecular Immunology 2015.

Un caso "atípico-típico"

Niño de 8 años:

- Macrohematuria postinfecciosa.
- Creatinina sérica: 1,8 mg/dL.
- UPCR 4,2 mg/mg.
- C3: ↓.
- C4: normal.

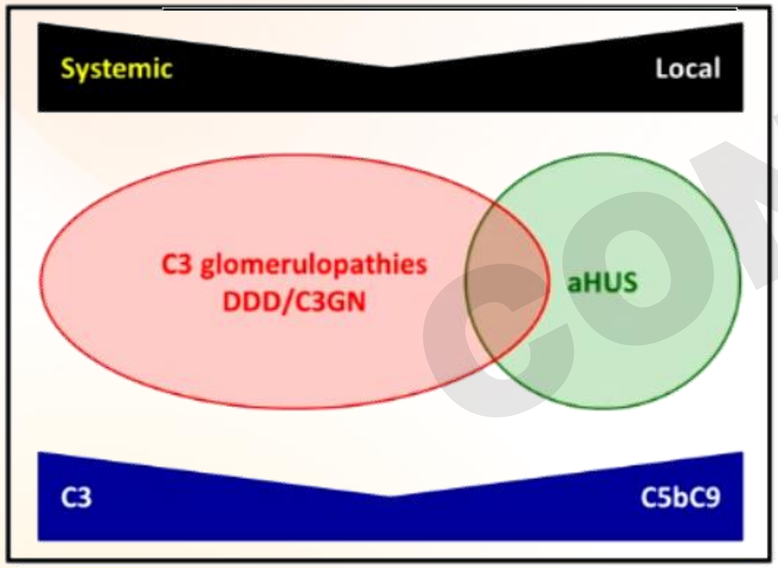


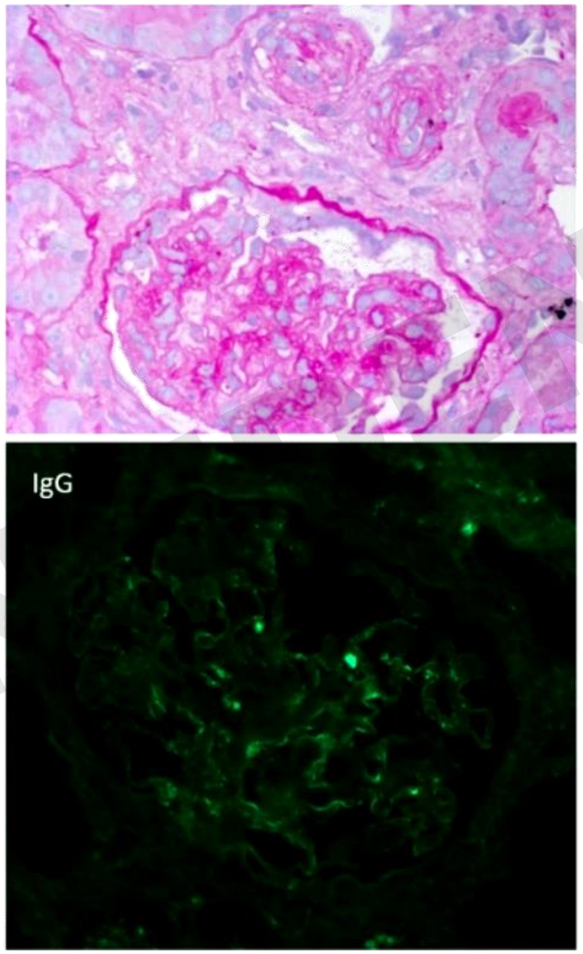


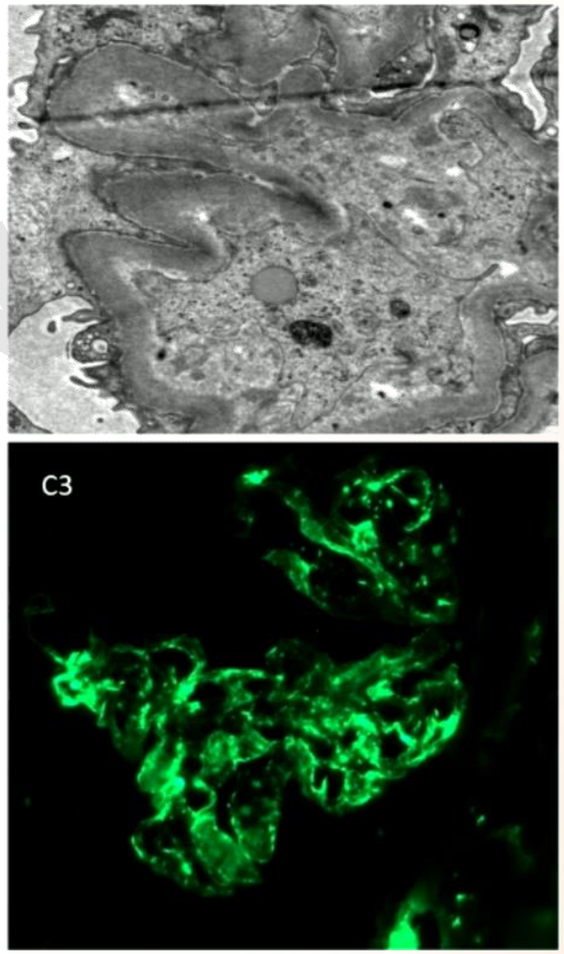
Coexistencia de C3G y TMA

5 y/o boy with aHUS:
Nephrotic syndrome following recovery from aHUS

Complete remission of proteinuria with eculizumab







Busutti et al., Nephron 2018

Caso de C3G tratado con inhibición de C5 y luego de C3

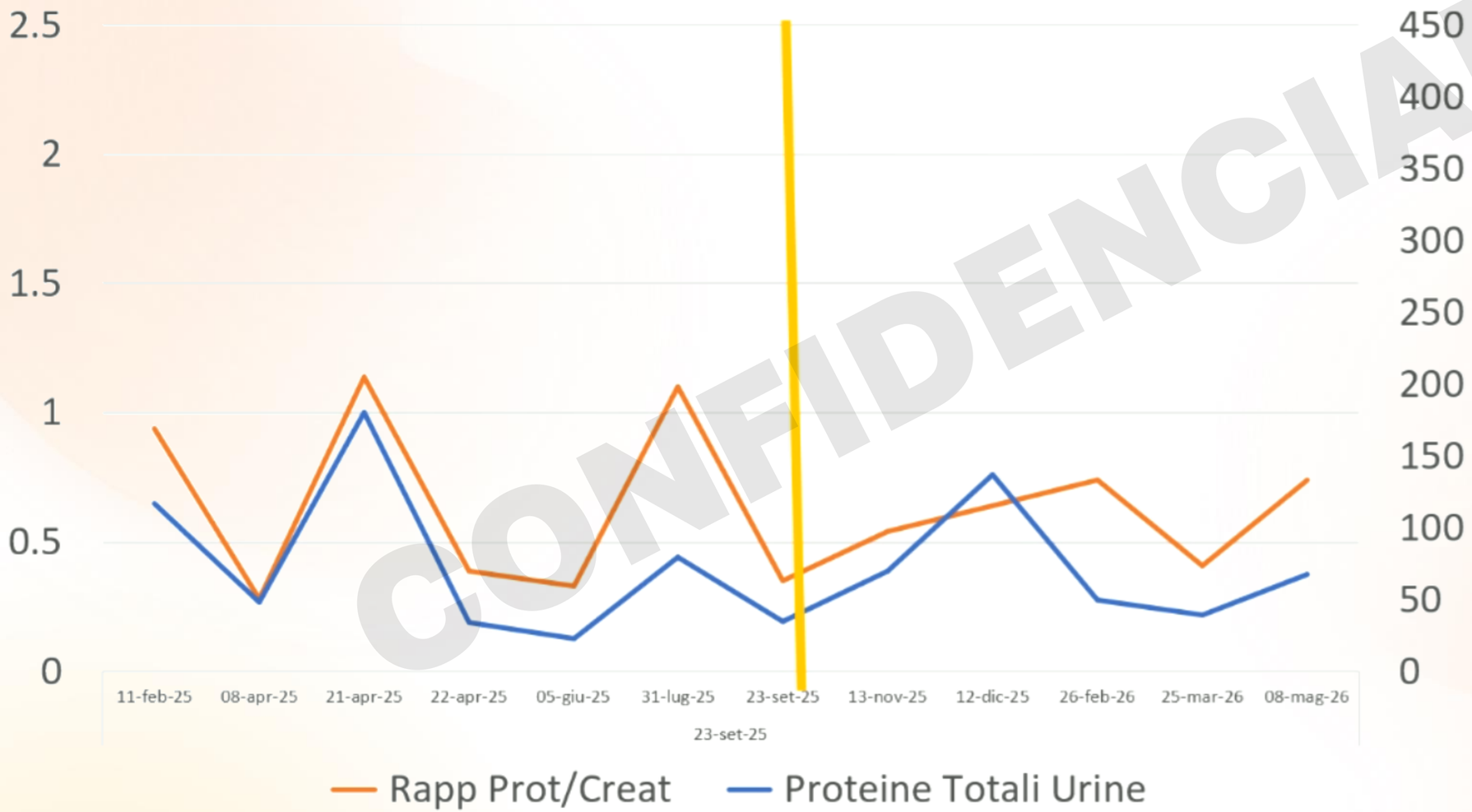
Abril 2020 (12 años): Macrohematuria (postinfecciosa) con proteinuria masiva (UPCR 20 g/g) y microhematuria persistente, creatinina sérica normal, hipoalbuminemia (1,8 g/dL), C3 sérico bajo con C4 normal; ANA, ENA y anti-dsDNA negativos.

Biopsia de riñón de mayo de 2020: C3G.
No hay respuesta a GC, MMF y ACE-i, con dependencia persistente de la albúmina intravenosa.
Cribado genético y serológico de la AP: negativo.

Marzo 2021-septiembre 2025: **eculizumab UPCR 5 reduce bruscamente a 0,3 - 0,4.** Cambio a ravulizumab, estable. C3 sérico persistente bajo. Desde el 2023 leve, aumento leve de la proteinuria a 0,7–0,8, sin síndrome nefrótico.

Marzo de 2025: Biopsia renal repetida: lesiones activas intensamente proliferativas, IF C3 +++.

Septiembre 2025: inicio con pegcetacoplán MAP



- Evolución de C3 sérico?
- Microhematuria?
- Rebiopsia renal

Adulto joven con GNMP-IC y luego C3G, primero tratado con iptacopán...

Joven adulto de 19 años (diciembre 2021):

- Proteinuria (proporción proteína/creatinina 8,32 mg/mg).
- Hematuria microscópica (60-150 células sanguíneas por campo).
- Creatinina sérica normal.
- ↓ C3 y C4 séricos, ↓ sC5b9 ANA normal y anti-dsDNA.

Primera biopsia renal: glomerulonefritis membranoproliferativa con un *'full house*' IF: Ig-MPGN.
Se inició una dieta baja en sal, MPDN vía intravenosa, luego PDN y MMF vía oral. Proteinuria no nefrótica persistente, ACEi y estatinas.

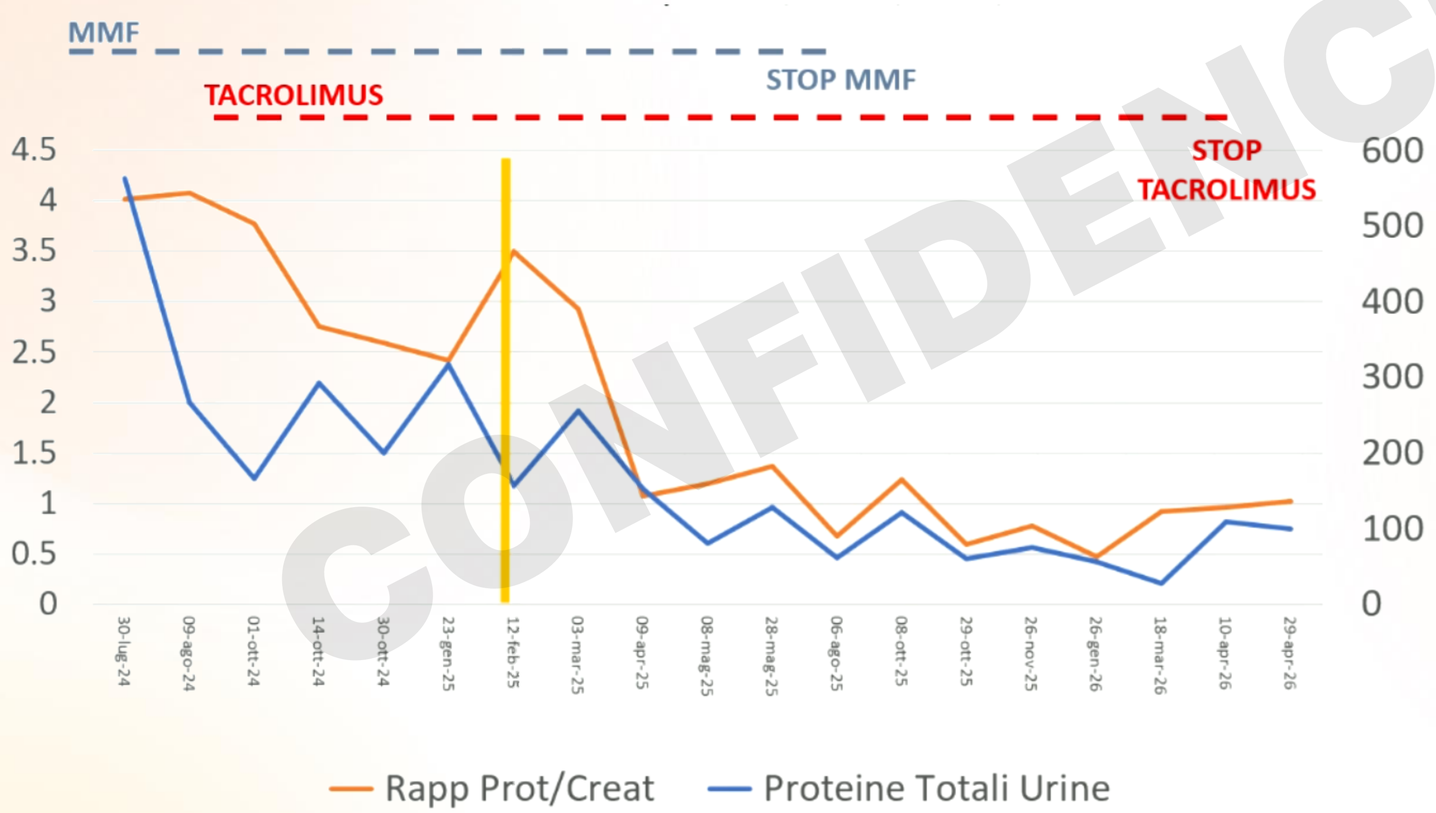
Abril de 2023 segunda biopsia: C3G.
Cribado genético y serológico: negativo.

Feb - oct 2024: iptacopán MAP: sin cambios en la proteinuria.

Se asocia Tacrolimus : no hay cambios en la proteinuria.

… y después con pegcetacoplán

March 2025: start PEGCETACOPLAN on top of FK, MMF, ACEi, statin

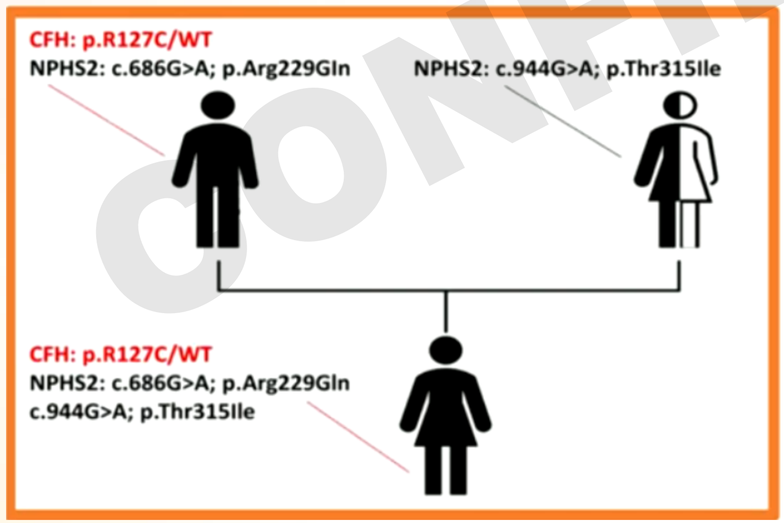


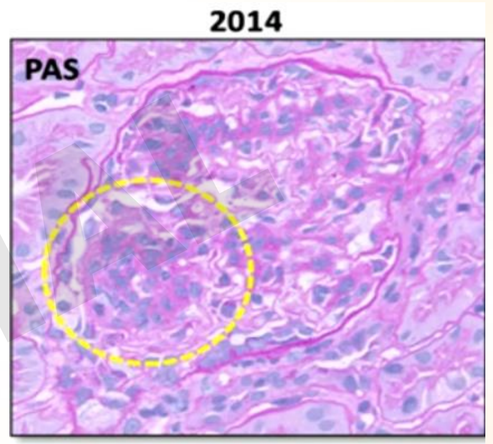
Forma genética de C3G con un giro distintivo

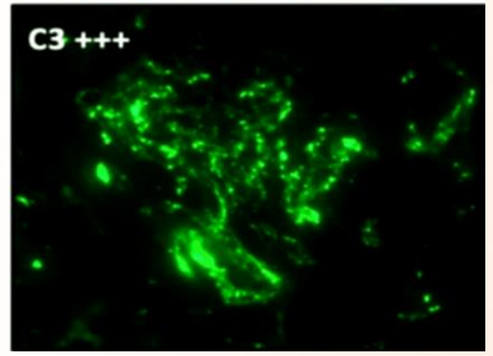
Nine months old girl (2004):

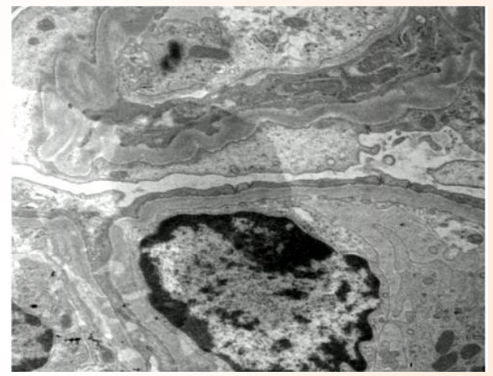
- macroscopic hematuria
- normal kidney function (Cr 0.2 mg/dL)
- hypoalbuminemia
- nephrotic proteinuria-to-creatinine ratio (UPCR 3.3)
- ↓ serum C3. Anti-FH antibodies were negative, and ↑ sC5B9 levels.

Genetic testing: **heterozygous p.R127C variant in the CFH gene**, pathogenic for C3G, which was also present in the patient's father (who also developed C3G at age 37)





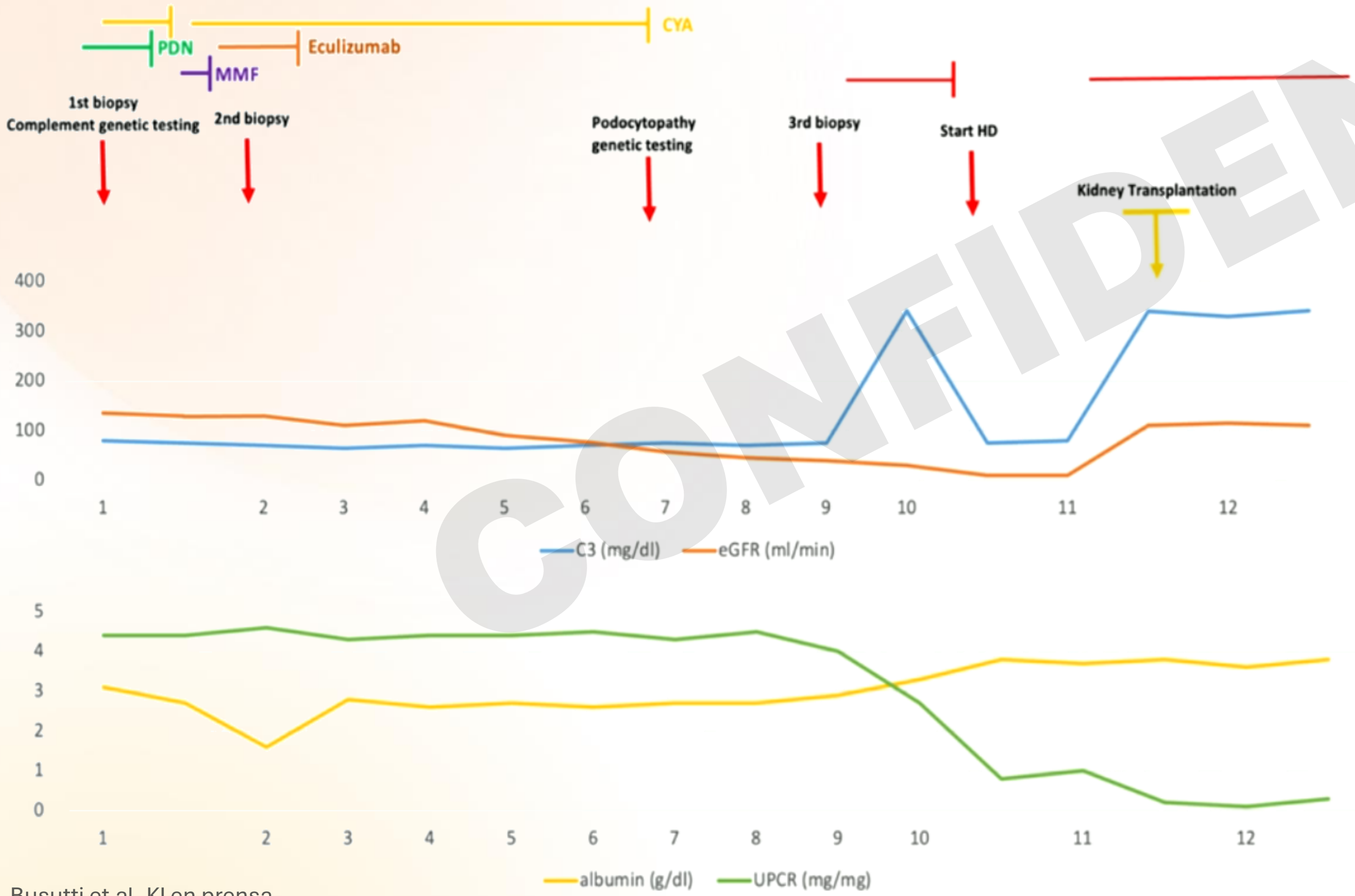


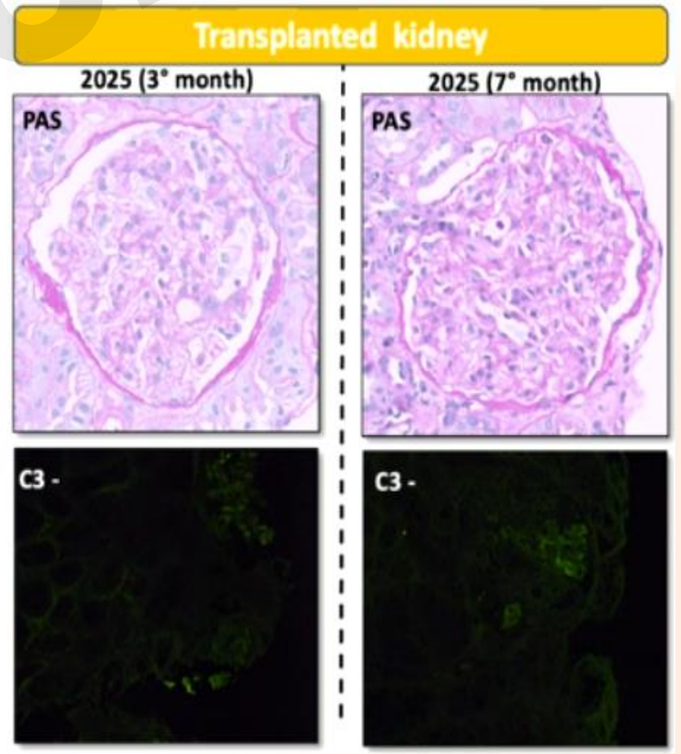


Busutti et al. KI en prensa.

Pegcetacoplán profiláctico para prevenir la recurrencia de C3G

Treatment: No response to prednisone, cyclosporine (CsA), and ACE inhibitor was initiated. From February to August 2016, the patient received Eculizumab, which was discontinued due to lack of therapeutic response. Since December 2023, renal function rapidly deteriorated and she was studied for a kidney transplant.





Busutti et al. KI en prensa.

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Pegcetacoplan sustained clinical benefit in C3G and primary IC-MPGN through 1 year of therapy: Data from VALIANT/VALE

Fadi Fakhouri[1], Gema Ariceta[2], Matthew C Pickering[3], Moglie Le Quintrec[4], Marina Vivarelli[5], Carla M Nester[6], Bradley P Dixon[7], Virginia Taliadouros[8], Johan Szamosi[8], Kathryn Gordon[9], Anwesha Mukherjee[9], Christoph Licht[10]

[1]Centre Hospitalier Universitaire Vaudois, Lausanne, Vaud, Switzerland; [2]Hospital Universitari Vall d'Hebron, Barcelona, Catalunya, Spain; [3]Department of Immunology and Inflammation, Imperial College, London, United Kingdom; [4]Department of Nephrology, Centre Hospitalier Universitaire de Montpellier, Montpellier, France; [5]Ospedale Pediatrico Bambino Gesu IRCCS, Rome, Italy; [6]University of Iowa, Stead Family Children's Hospital, Iowa City, IA, United States; [7]University of Colorado Anschutz Medical Campus School of Medicine, Aurora, CO, United States; [8]Swedish Orphan Biovitrum AB, Stockholm, Sweden; [9]Apellis Pharmaceuticals, Inc., Waltham, MA, United States; [10]The Hospital for Sick Children, Toronto, Ontario, Canada.

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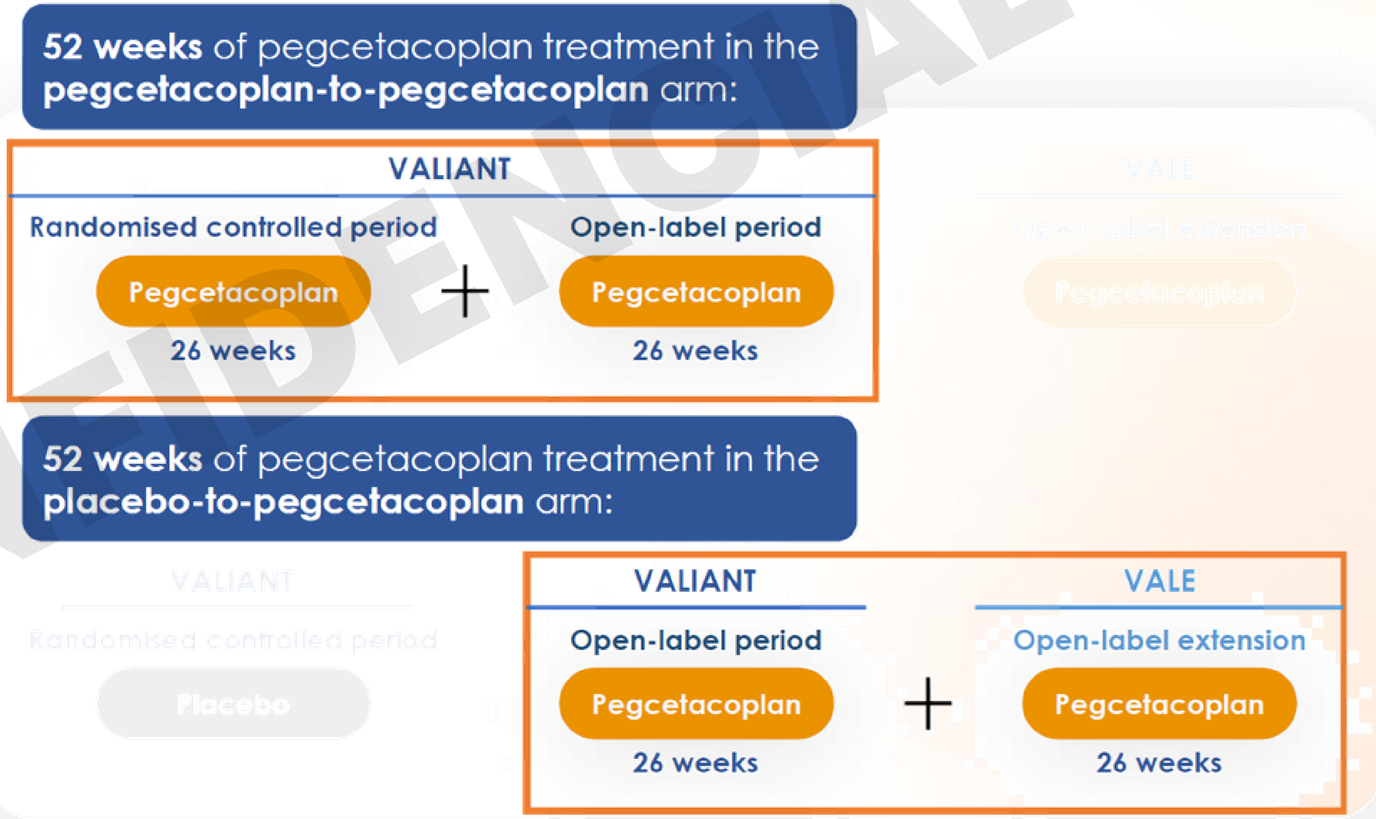
Objetivo: evaluar la **eficacia y seguridad de pegcetacoplán a 1 año** en **una población más grande** que la previamente reportada

Method:

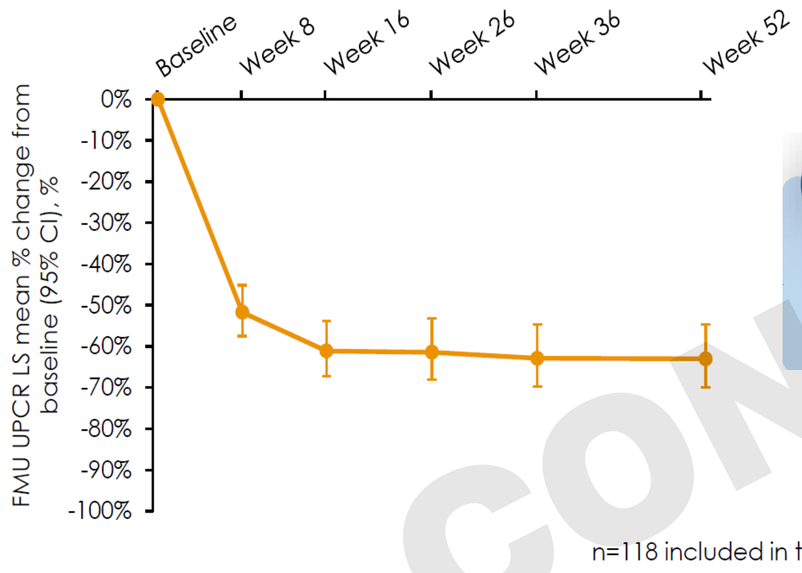
- Patients in VALIANT with <52 weeks of pegcetacoplan treatment were supplemented with up to 6 months of treatment data from the VALE extension study*
- All data were time-aligned such that baseline represented pegcetacoplan initiation for all patients



*No hay datos histológicos disponibles para este análisis, ya que la evaluación histológica no era obligatoria a las 52 semanas en VALIANT ni estaba incluida en el protocolo del estudio VALE.

La reducción de proteinuria se mantuvo hasta la semana 52

Change from time-aligned baseline in proteinuria over 52 weeks of pegcetacoplan treatment



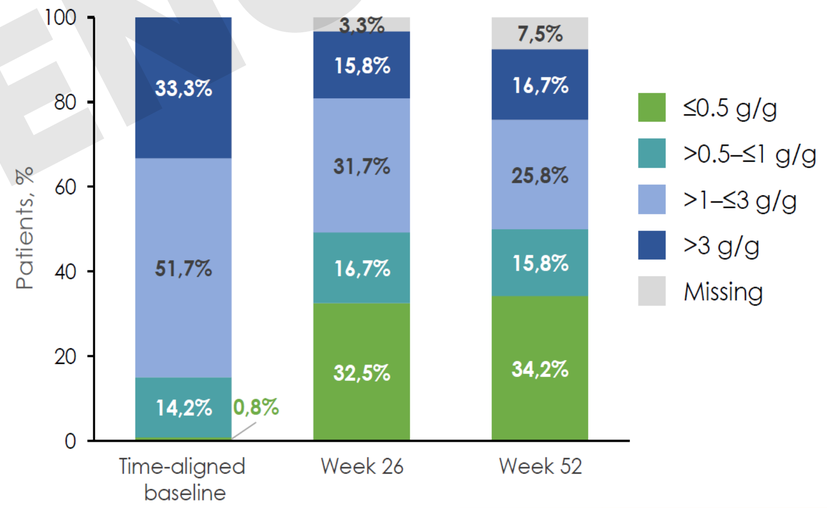
n=118 included in the MMRM model*

Geometric mean change in proteinuria between baseline and 52 weeks:

-63.0%

(95% CI -69.9, -54.7)

Proteinuria shift analysis based on FMU UPCR



Patients with UPCR ≤1 g/g at 52 weeks:

50.0%

n=60/120

Patients with UPCR ≤0.5 g/g at 52 weeks:

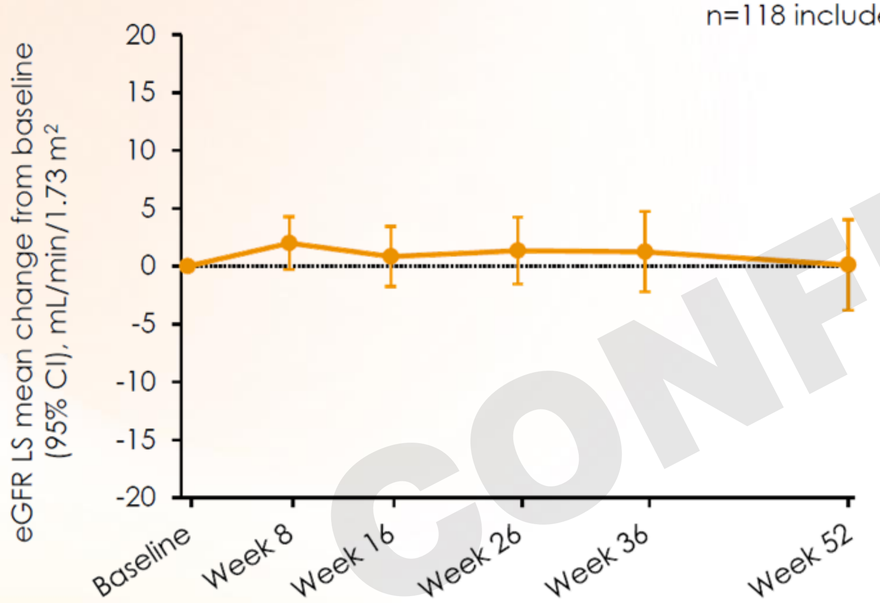
34.2%

n=41/120

Un 50% de los pacientes alcanzó una proteinuria ≤ 1 g/g

Pegcetacoplán se asoció con **la estabilización de la eGFR, mantenida durante 52 semanas**

Change from time-aligned baseline in eGFR over 52 weeks of pegcetacoplan treatment



n=118 included in the MMRM model*

LS mean change in eGFR between baseline and 52 weeks:

+0.1

mL/min/1.73 m²

(95% CI -3.8, 4.0)

*Modelo MMRM que incluye el efecto categórico fijo para el grupo de tratamiento, visita, tipo de enfermedad, uso basal de inmunosupresores y factores de estratificación, así como la interacción visita-grupo de tratamiento, además de la covariable continua fija de eGFR basal; se utiliza para analizar el cambio desde el valor basal de eGFR en cada visita en comparación con el basal de VALIANT.
IC, intervalo de confianza; eGFR, tasa de filtrado glomerular estimada; LS, mínimos cuadrados; MMRM, modelo mixto para medidas repetidas.

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Pegcetacoplan Treatment Response in VALIANT/VALE: Impact of Genetic and Acquired Complement Dysregulation and Disease Type in C3G and Primary IC-MPGN

Marina Noris[1], Nicole Van De Kar[2], Daniel P Gale[3], Sophie Chauvet[4,5], Fernando Caravaca-Fontán[6], Julia Weinmann-Menke[7], Carla M Nester[8], Lucia Quintana-Gallardo[9], Johan Szamosi[9], Kathryn Gordon[10], Anwesha Mukherjee[10], Fadi Fakhouri[11]

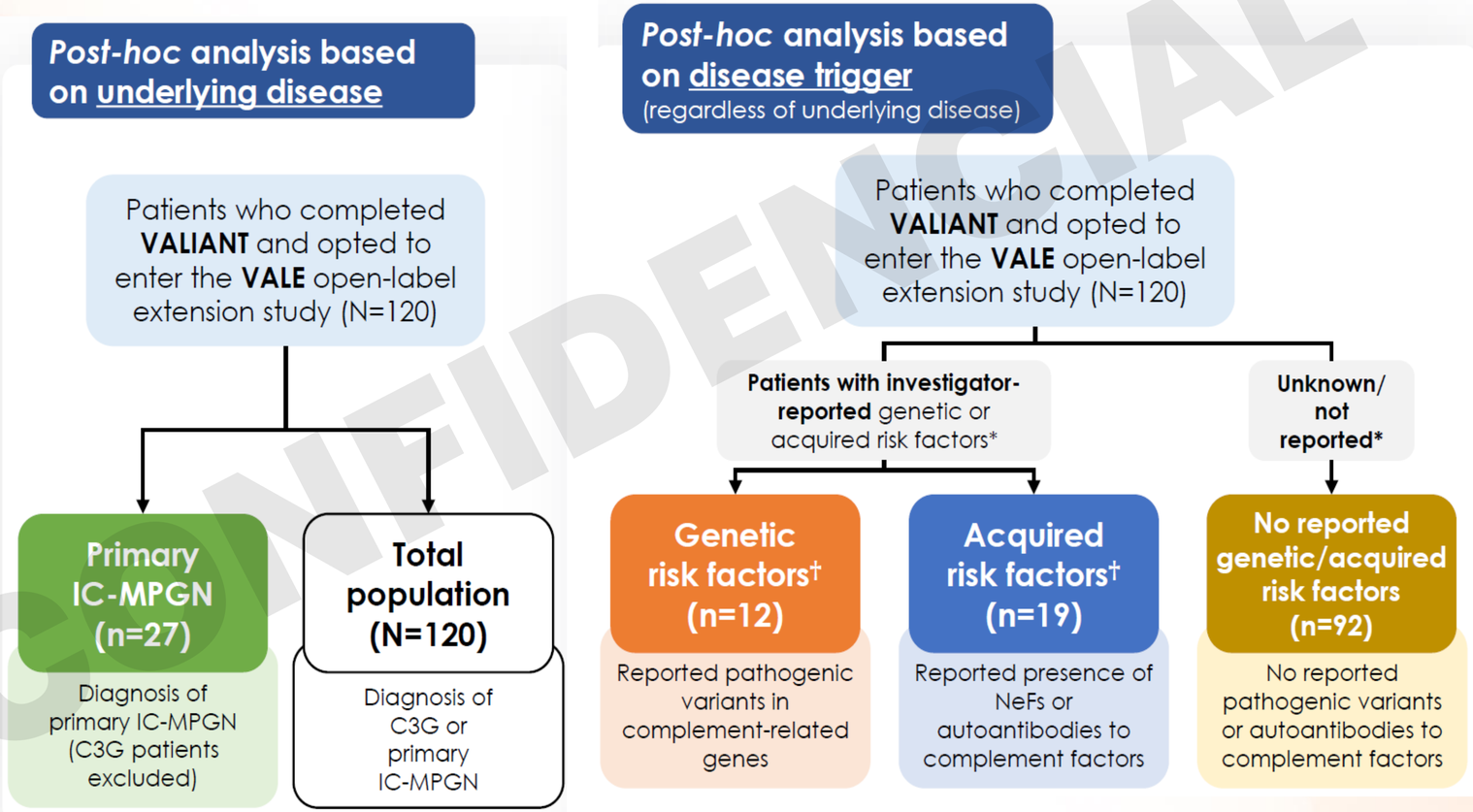
[1]Istituto di Ricerche Farmacologiche Mario Negri IRCCS, Milan, Italy; [2]Radboud University Medical Center, Amalia Children's Hospital, Nijmegen, Netherlands; [3]University College London, London, United Kingdom; [4]Centre de Recherche des Cordeliers, Sorbonne Université, INSERM, Université Paris Cité, Inflammation, Complement and Cancer Team, Paris, France; [5]Department of Nephrology, European Hospital Georges Pompidou, APHP, Paris, France; [6]Department of Nephrology, Instituto de Investigación Hospital 12 de Octubre, Madrid, Spain; [7]Department of Nephrology, University Medical Center of the Johannes Gutenberg University, Mainz, Germany; [8]University of Iowa, Stead Family Children's Hospital, Iowa City, IA, United States; [9]Swedish Orphan Biovitrum AB, Stockholm, Stockholm, Sweden; [10]Apellis Pharmaceuticals, Inc., Waltham, MA, United States; [11]Centre Hospitalier Universitaire Vaudois, Lausanne, Vaud, Switzerland

sobi

Objetivo: evaluar el efecto del tratamiento con **pegcetacoplán a 1 año en subgrupos de pacientes de particular interés**

Method:

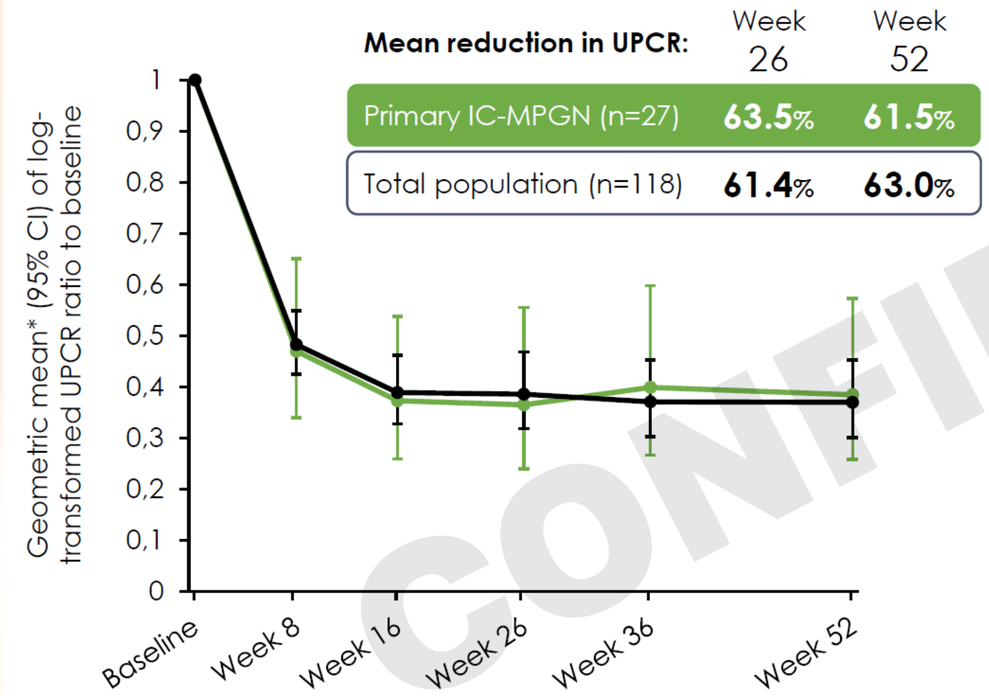
- Data from the Phase 3 VALIANT trial and its open-label extension (VALE) were pooled to enable a uniform evaluation of 1 year of pegcetacoplan exposure across all patients
- All data were time-aligned such that baseline represented pegcetacoplan initiation for all patients



*Las pruebas para factores de riesgo genéticos y adquiridos no eran obligatorias para la inclusión en VALIANT. Los factores de riesgo genéticos y adquiridos fueron reportados de forma opcional por los investigadores y no se evaluaron dentro del ensayo.
† Los pacientes con factores de riesgo genéticos reportados también podían presentar factores de riesgo adquiridos, y viceversa.
C3G, glomerulopatía por C3; IC-MPGN, glomerulonefritis membranoproliferativa por complejos inmunes; NeF, factor nefrítico.

Pegcetacoplán condujo a una reducción media sostenida de la UPCR superior al 50% entre subgrupos

Change from time-aligned baseline in UPCR over time during pegcetacoplan treatment

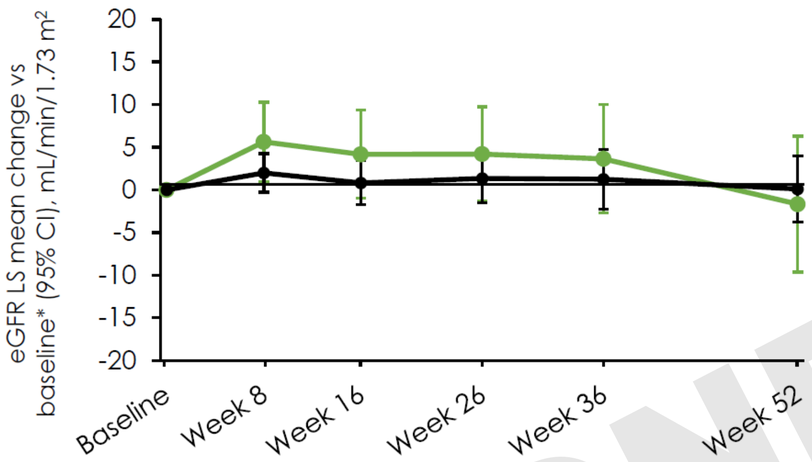


Mean reduction in UPCR:	Week 26	Week 52
Primary IC-MPGN (n=27)	**63.5%**	**61.5%**
Total population (n=118)	**61.4%**	**63.0%**

*Medias geométricas y porcentaje de cambio estimados a partir de las diferencias de medias LS exponenciadas procedentes de un modelo MMRM del UPCR transformado logarítmicamente.
n = número de pacientes incluidos en cada modelo.
CI, intervalo de confianza; FMU, primera orina de la mañana; IC-MPGN, glomerulonefritis membranoproliferativa por complejos inmunes; LS, mínimos cuadrados; MMRM, modelo mixto para medidas repetidas; UPCR, relación proteína:creatinina urinaria.

Pegcetacoplán condujo a la estabilización de la eGFR hasta 1 año entre subgrupos

Change from time-aligned baseline in eGFR over time during pegcetacoplan treatment



Mean change in eGFR between baseline and 52 weeks:

Primary IC-MPGN (n=27)	**-1.7** mL/min/1.73 m²
Total population (n=118)	**+0.1** mL/min/1.73 m²

* Modelo MMRM del cambio medio respecto a la línea inicial en la eGFR. n=número de pacientes incluidos en cada modelo.
CI, intervalo de confianza; eGFR, tasa de filtración glomerular estimada; IC-MPGN, glomerulonefritis membranoproliferativa del complejo inmunitario; LS, mínimos cuadros; MMRM, modelo mixto para medidas repetidas.

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Real-world clinical profile, treatment patterns and outcomes associated with C3G and primary IC-MPGN: Insights from the UK RaDaR registry

Lexy Sorrell, Carly Rich[2], Veruska Carboni[2], Jameel Nazir[2], David Pitcher[1,3], Edwin Wong[4], Katie Wong[1,3], Sherry Masoud[1,3], Daniel P. Gale[1,3]

[1]National Registry of Rare Kidney Diseases, Bristol, United Kingdom; [2]Swedish Orphan Biovitrum AB, Stockholm, Sweden; [3]Centre for Kidney and Bladder Health, University College London, London, United Kingdom; [4]National Renal Complement Therapeutics Centre, Newcastle Upon Tyne Hospitals NHS Foundation Trust, Newcastle Upon Tyne, United Kingdom

Métodos: objetivos y análisis

Variables	Description
Baseline characteristics	Including age, sex, ethnicity, CKD stage, reported medications
Longitudinal outcome	eGFR at baseline[a] and last follow-up[b]
Other outcomes	Proportion of patients and Kaplan–Meier estimates for dialysis, kidney transplant and death

- Results were stratified by disease type (C3G vs. primary IC-MPGN) and age (paediatrics, adults)
- No statistical comparisons between groups were conducted

Resultados: características del paciente

Characteristics at diagnosis	C3G (n=159)		Primary IC-MPGN (n=146)	
	Paediatrics (n=88)	Adults (n=71)	Paediatrics (n=62)	Adults (n=84)
Age, years; median (IQR)	11 (8–15)	39 (25–51)	9 (7–12)	51 (35–64)
Male sex; n (%)	47 (53)	43 (61)	30 (48)	38 (45)
White ethnicity, n (%)[a]	74 (84)	54 (76)	53 (85)	73 (87)
C3G subgroup, n (%)				
C3 glomerulonephritis	50 (57)	54 (76)		
DDD	37 (42)	17 (24)		
C3G-NOS	1 (1)	0		
eGFR; median (IQR)	88 (60–109) [n=61]	49 (30–73) [n=46]	88 (65–109) [n=46]	40 (30–59) [n=51]
UPCR, g/g; median (IQR)	3.6 (1.2–6.6)	3.3 (1.2–6.3)	4.5 (2.7–7.8)	4.1 (1.4–8.0)
CKD stage, n (%)				
1	42 (48)	13 (18)	30 (48)	8 (10)
2	25 (28)	15 (21)	17 (27)	11 (13)
3a	4 (5)	10 (14)	9 (15)	16 (19)
3b	9 (10)	17 (24)	6 (10)	29 (35)
4	8 (9)	16 (23)	0	20 (24)
Median follow-up, years (IQR)	10.2 (6.0–12.9)	8.9 (5.5–13.2)	11.0 (7.8–15.1)	8.7 (5.8–13.4)

[a]Ethnicity data missing for C3G adult (n=1) and IC-MPGN paediatrics (n=2).
C3, complement component 3; C3G, complement 3 glomerulopathy; CKD, chronic kidney disease; DDD, dense deposit disease; eGFR, estimated glomerular filtration rate; IC-MPGN, immune-complex membranoproliferative glomerulonephritis; IQR, interquartile range; NOS, not otherwise specified; UPCR, urine protein creatinine ratio.

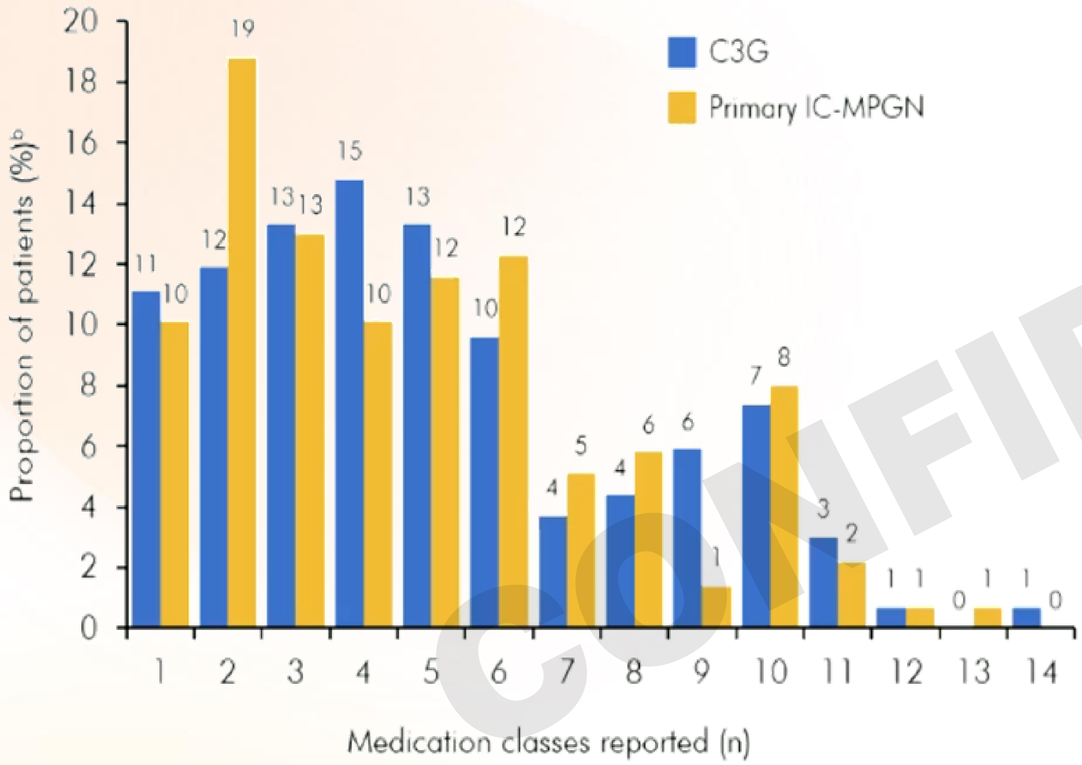
- *Peor FGe en adultos, con similar grado de proteinuria.*
- *Similares características C3G y IC-MPGN.*

Resultados: medicación

- *Gran carga de tratamiento.*
- *Similar en GC3 y GNMP-IC*

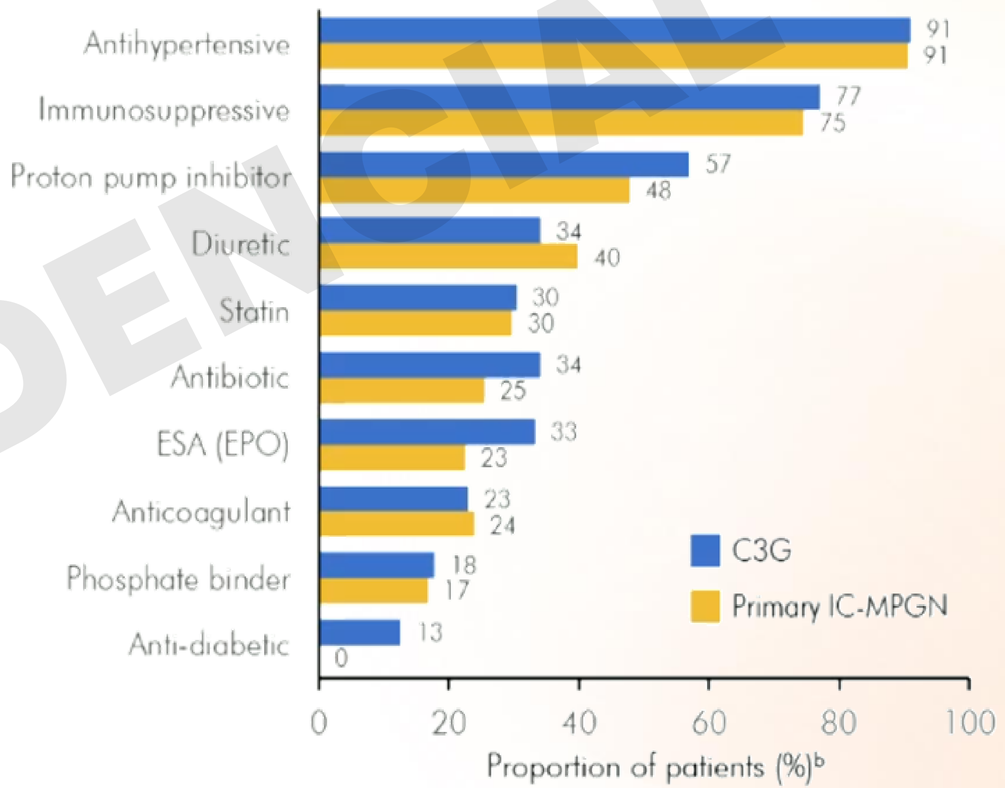
Academy

Number of medication classes reported[a]



Multiple conventional therapies were used:
median (IQR) number of medication classes per patient was **4 (2–6)**

Type of medication classes[a]



Antihypertensives and **immunosuppressants** were the **most common medications**

[a]Medication data available for 273 (90%) patients. [b]Rounded percentage values are shown.
C3G, complement 3 glomerulopathy; EPO, erythropoietin; ESA, erythropoiesis-stimulating agent; IC-MPGN, immune-complex membranoproliferative glomerulonephritis; IQR, interquartile range.

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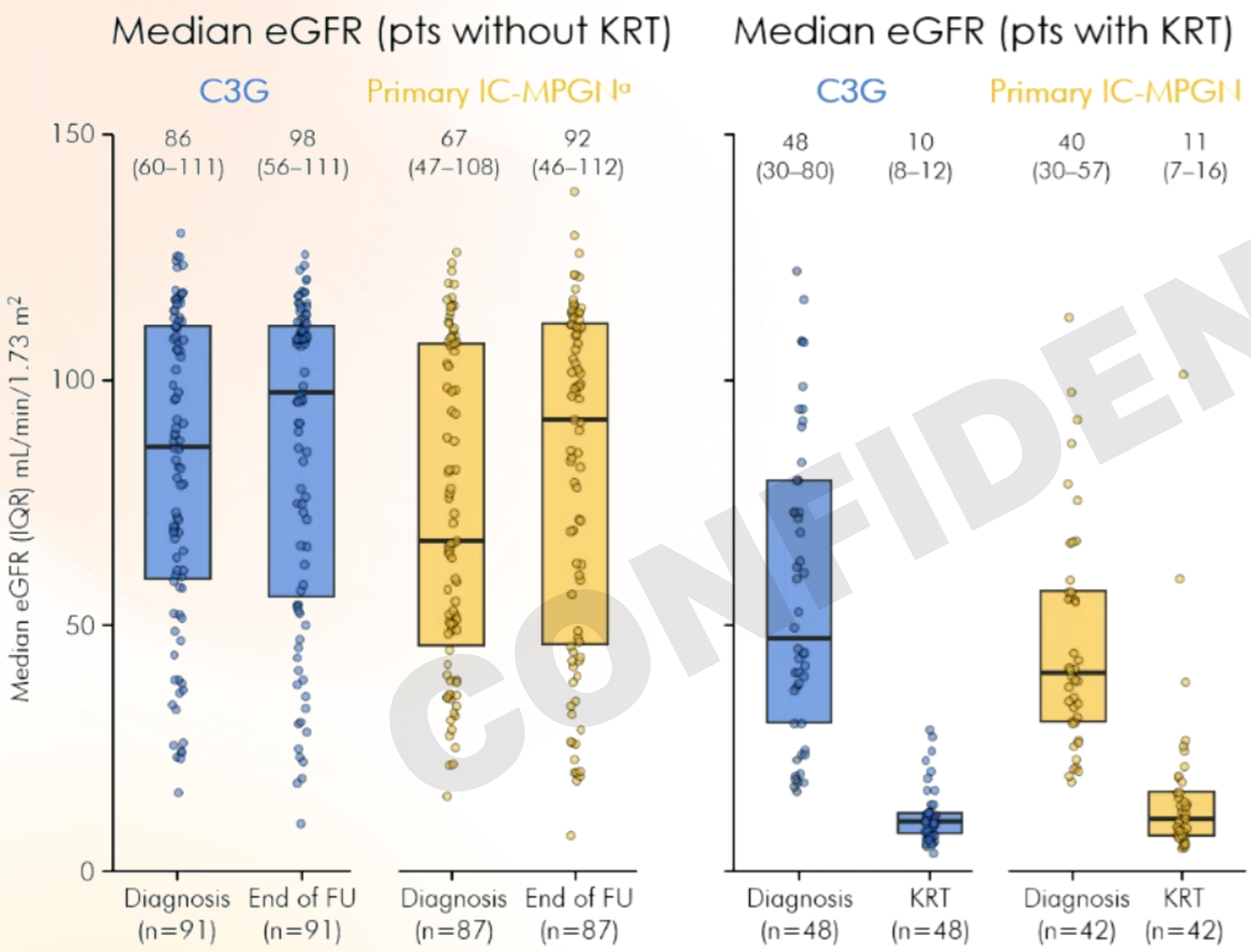
Resultados: eventos derivados de terapia sustitutiva renal

KRT events	C3G (n=159)		Primary IC-MPGN (n=146)	
	Number of events (%)	Median (IQR) years from diagnosis to event[a]	Number of events (%)	Median (IQR) years from diagnosis to event[a]
Dialysis (incl. following pre-emptive transplant)	47 (30)	2.9 (0.9–6.2)	40 (27)	2.3 (0.6–8.0)
Transplant (incl. post-dialysis)	42 (26)	5.8 (3.5–9.6)	32 (22)	4.6 (2.7–7.5)
1 transplant	38 (24)	–	25 (17)	–
2 transplants	4 (3)	–	6 (4)	–
3 transplants	0	–	1 (1)	–
First KRT (dialysis or pre-emptive transplant)	55 (35)	3.1 (1.0–8.6)	44 (30)	3.3 (0.6–6.4)
First KRT = dialysis	45 (28)	–	37 (25)	–
First KRT = pre-emptive transplant	10 (6)	–	7 (5)	–

- *Alto porcentaje de TRS.*
- *Similar en C3G y IC-MPGN.*

[a]Median time was calculated among patients who had an event.
C3G, complement 3 glomerulopathy; IC-MPGN, immune-complex membranoproliferative glomerulonephritis; IQR, interquartile range; KRT, kidney replacement therapy.

Resultados: eGFR durante el seguimiento



Patients with KRT had lower eGFR values at diagnosis and reported a substantial reduction in eGFR at follow-up

[a]Two eGFR values in the primary IC-MPGN group (>150 mL/min/1.73 m²) were omitted from the plot for clarity, one at diagnosis and one at end of FU.
C3G, complement 3 glomerulopathy; eGFR, estimated glomerular filtration rate; FU, follow-up; IC-MPGN, immune-complex membranoproliferative glomerulonephritis; KRT, kidney replacement therapy; IQR, interquartile range; pt, patient.

Deterioro renal progresivo, elevada carga clínica y malos resultados a pesar de múltiples terapias convencionales

Longitudinal data from patients with C3G or primary IC-MPGN followed for ~10 years in the UK

Patients experienced poor outcomes despite substantial exposure to conventional therapies

~30%
of patients quickly progressed to requiring dialysis or transplant, indicating substantial patient, caregiver and healthcare system burden

~10%
all-cause mortality highlights poor prognosis, particularly in such a young population

These findings highlight the need for effective therapies targeting complement dysregulation to preserve kidney function, delay or prevent kidney failure, reduce reliance on KRT, and improve survival

C3G, complement 3 glomerulopathy; IC-MPGN, immune-complex membranoproliferative glomerulonephritis; KRT, kidney replacement therapy; UK, United Kingdom.

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Epidemiology of C3G and primary IC-MPGN: A systematic literature review and meta-analysis

F. Caravaca-Fontán,[1] C. Rich,[2] V. Carboni,[2] J. Nazir,[2] C. D. Poole,[3] D. P. Gale[4,5]

[1]Instituto de Investigación Hospital 12 de Octubre (imas12), Madrid, Spain; [2]Swedish Orphan Biovitrum AB, Stockholm, Sweden; [3]Digital Health Labs, Cardiff, United Kingdom; [4]Centre for Kidney and Bladder Health, University College London, London, United Kingdom; [5]National Registry of Rare Kidney Diseases, Bristol, United Kingdom

Abstract number: 2440

C3 Academy

CONCLUSIONS

- This systematic literature review and meta-analysis indicates C3G and primary IC-MPGN have similar epidemiological profiles
- Based on data from Europe, Asia and South America, pooled estimated **prevalence** was **17.6–17.8 per million population (pmp)** and **incidence** was **0.4–0.5 cases per 10^6 person-years (PY)**
- Correcting for disease-specific mortality, which addresses the limitations of previously calculated estimates, reduced prevalence of both conditions to ~12.5 pmp
- These data support that C3G and primary IC-MPGN are **ultra-rare (or ultra-orphan) diseases**, and emphasise the unmet need for **disease-modifying targeted therapies** that can effectively prevent renal damage and reduce the risk of kidney failure in patients with C3G or primary IC-MPGN

INTRODUCTION

- C3G and primary IC-MPGN are heterogeneous, rare, complement-mediated kidney diseases[1,2]
- Up to 50% of individuals with C3G or primary IC-MPGN progress to irreversible kidney failure within a decade of diagnosis, requiring dialysis or transplantation.[1,3] Most people (~89%) experience disease recurrence after transplantation, with allograft loss occurring frequently[2,4]
- The poor long-term outcomes and disease-related complications of C3G and primary IC-MPGN create substantial clinical, economic and healthcare burdens[5]
- Robust epidemiological data are essential for informing healthcare planning, resource allocation and public health policy making[6]

AIM

- To conduct an SLR on the epidemiology of C3G and primary IC-MPGN and synthesise the data using a frequentist meta-analytic approach

METHODS

Systematic literature review

- PubMed, Embase and Cochrane databases were searched to identify real-world studies reporting prevalent or incident cases of C3G and/or primary IC-MPGN between 01 January 2010 and 30 June 2025. This analysis reflects an updated search conducted after the ERA 2026 abstract submission
- Studies were excluded if they described secondary IC-MPGN, case reports, or case series without a definable denominator
- Data extracted from eligible studies included number of cases, referral population size, study duration and reported study-level incidence/prevalence
- Quality assessment was performed using STROBE criteria[7]

Meta-analysis

- All prevalence and incidence estimates were harmonised to common units of pmp and cases per 10^6 PY, respectively
- A frequentist meta-analysis was conducted using a restricted maximum likelihood (REML)-weighted random-effects model; pooled estimates were reported with 95% CIs
- Mortality-adjusted prevalence was calculated by applying a pooled estimated correction factor to the unadjusted prevalence estimates
- Heterogeneity was assessed using the I^2 statistic, Cochran's Q and the between-study variance τ^2; publication bias was evaluated using Egger's regression test

Sensitivity analyses

- To determine the influence of: (1) excluding individual studies; and (2) excluding either adult or paediatric age cohort studies

RESULTS

Study characteristics

- The searches identified 7727 records; after removing duplicates, 5615 records underwent title/abstract screening and 155 records assessed by full-text review. Overall, 11 studies reporting data for 32–620 patients from Europe, Asia and South America were included in the meta-analysis; 8/11 studies met ≥80% of STROBE checklist items

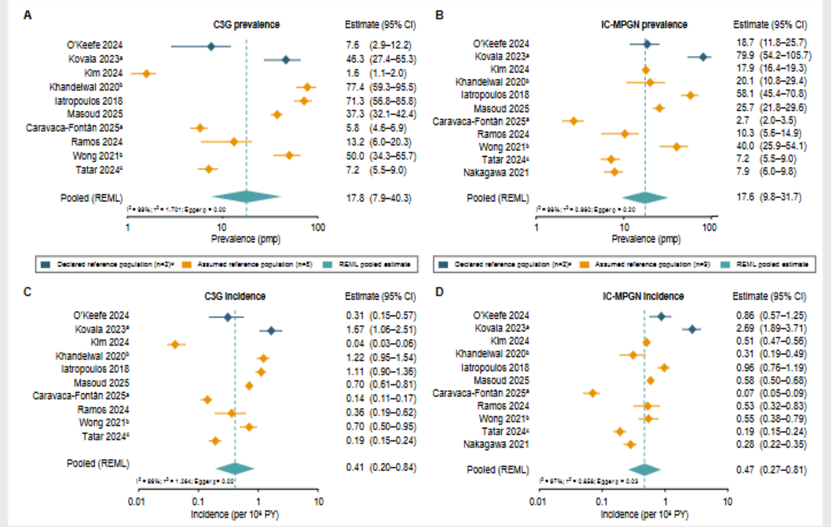
Meta-analysis

- Pooled estimated prevalence rates (95% CI) for C3G and primary IC-MPGN were 17.8 (7.9–40.3) and 17.6 (9.8–31.7) pmp, respectively (**Figure 1 A & B**). Incidence rates (95% CI) were 0.41 (0.20–0.84) and 0.47 (0.27–0.81) per 10^6 PY, respectively (**Figure 1 C & D**)
- Substantial heterogeneity in prevalence and incidence rates was observed between studies (I^2, 97–99%; Cochran's Q, 399–956 [p<0.001 for all]; τ^2, 0.86–1.70)
- Disease-specific mortality, derived from five studies, estimated a pooled correction factor of 0.71 (95% CI 0.55–0.88) with no heterogeneity. The mortality-adjusted prevalence rates (95% CI) for C3G and primary IC-MPGN were 12.7 (5.4–29.7) and 12.6 (6.7–23.7) pmp, respectively, corresponding to ~30% lower than the unadjusted rates

Sensitivity analyses

- Individual studies generally had minimal influence on pooled estimates. The change in prevalence and incidence by excluding individual studies ranged from −2.7 to 5.7 pmp and −0.06 to 0.12 per 10^6 PY, respectively
- Excluding the two paediatric studies reduced pooled estimates of C3G prevalence (Δ −4.7 pmp) and incidence (Δ −0.07 per 10^6 PY). Excluding the two adult studies minimally decreased C3G prevalence (Δ −0.40 pmp) and incidence (Δ −0.01 per 10^6 PY). Excluding studies based on age subgroups had a relatively similar impact on primary IC-MPGN prevalence and incidence

Figure 1. Forest plots of prevalence (A & B) and incidence (C & D) for C3G and primary IC-MPGN



Data reported on log scale with 95% CI error bars. Dashed line represents pooled mean estimates.
[a]Adult population. [b]Paediatric population. [c]Tatar 2024 reported 66 patients with primary MPGN as a combined cohort without subtype split; the meta-analysis applied the same case count to both C3G and primary IC-MPGN. [d]Only 2/11 studies (O'Keefe and Kovala) explicitly declared their reference populations; the remaining 9 required assumptions about catchment coverage over study periods of up to 32 years.

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Pegcetacoplan treatment response in VALIANT/VALE: Impact of disease chronicity in C3G and primary IC-MPGN

Abstract no.: 3319

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D. WALLACE[1], A. AVILA[2], A. MASTRANGELO[3], F. PROVOT[4], D. ZECHER[5], C.M. NESTER[6], A.S. BOMBACK[7], L. QUINTANA-GALLARDO[8], J. SZAMOSI[8], G. AYEHU[9], A. MUKHERJEE[9] and F. FAKHOURI[10]

[1] Royal Manchester Children's Hospital, Manchester, United Kingdom [2] Department of Nephrology, Hospital Universitario Doctor Peset, Valencia, Spain [3] Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico, Milan, Lombardia, Italy [4] Department of Nephrology, Lille University Hospital, Lille, France [5] Department of Nephrology, University Hospital Regensburg, Regensburg, Germany [6] University of Iowa, Stead Family Children's Hospital, Iowa City, IA, United States [7] New York-Presbyterian/Columbia University Irving Medical Center, New York, NY, United States [8] Swedish Orphan Biovitrum AB, Stockholm, Sweden [9] Apellis Pharmaceuticals, Inc., Waltham, MA, United States [10] Centre Hospitalier Universitaire Vaudois, Lausanne, Vaud, Switzerland

INTRODUCTION

C3G and primary (idiopathic) IC-MPGN

Rare glomerular diseases driven by uncontrolled activation of C3, causing excessive glomerular deposition of C3 breakdown products[1,2]

Indicators of disease chronicity

High levels of interstitial fibrosis/tubular atrophy (IFTA) and low estimated glomerular filtration rate (eGFR) correlate with poor outcomes[3-5]. Cumulative structural damage can develop silently without acute signs[6].Current standard of care does not address the underlying pathologic mechanism[2], permitting irreversible kidney damage

Pegcetacoplan

Targeted C3/C3b inhibitor. In the Phase 3 VALIANT study, pegcetacoplan led to clearance of C3 deposits, proteinuria reduction, and eGFR stabilisation[7] – irrespective of age, disease subtype, proteinuria levels, or concomitant immunosuppression[7-9]

AIM & METHOD

Aim

***Post-hoc* analysis** evaluating the impact of baseline disease chronicity on 1-year clinical outcomes following pegcetacoplan treatment in patients with C3G and primary IC-MPGN

Dataset

Pooled data from the Phase 3 VALIANT trial and its open-label extension study (VALE) enabling a uniform evaluation of 1 year of pegcetacoplan exposure

Realigned baseline approach:

- Continuous pegcetacoplan arm: original VALIANT baseline; outcomes at 52 weeks
- Placebo to pegcetacoplan switch: Week 26 as new baseline (26 weeks VALIANT + 26 weeks VALE)
- Patients with >50% IFTA excluded from VALIANT

Patient stratification

- IFTA at baseline biopsy (≤25% or 26–50%)
- eGFR at baseline (>90, ≤90–≥60 or <60 mL/min/1.73 m²)

Outcomes

Changes in proteinuria (based on urine protein:creatinine ratio [UPCR] from first morning urine) and eGFR over 52 weeks were analysed

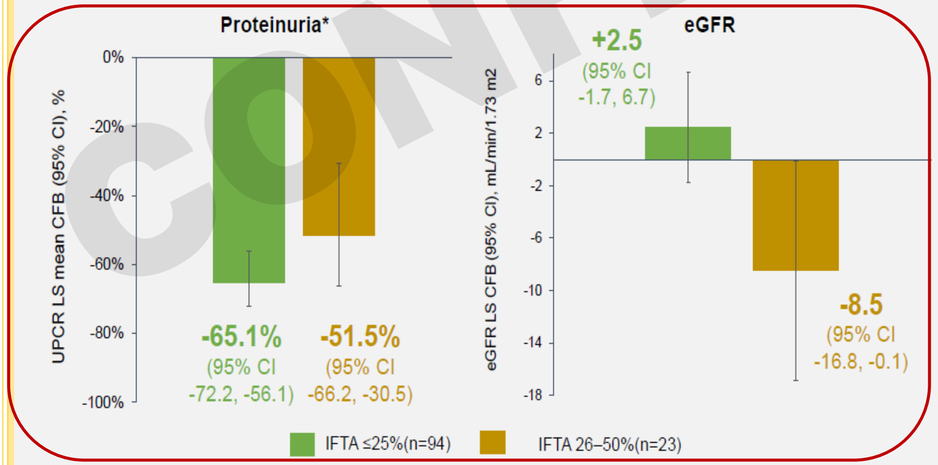
Time is Kidney

Pegcetacoplan reduces proteinuria by >50% indicating **effective suppression of ongoing complement-mediated disease regardless of baseline disease chronicity**

Data suggest that **patients with reduced eGFR may still derive clinical benefit from targeted C3 inhibition;** however, **eGFR stabilisation is maximised when treatment begins before significant kidney fibrosis**

These findings support early identification and intervention with targeted C3 inhibition before substantial fibrotic damage is established

FIGURE 1. Change from baseline in UPCR and eGFR at Week 52 by IFTA at baseline



* Geometric means and % change estimated from exponentiated LS mean differences from a MMRM of log-transformed UPCR.
CFB, change from baseline; CI, confidence interval; eGFR, estimated glomerular filtration rate; IFTA, interstitial fibrosis/tubular atrophy; LS, least squares; MMRM, mixed model for repeated measures; UPCR, urine protein:creatinine ratio.

RESULTS

~~119 (IFTA) and 129 (eGFR) patients were evaluable~~ **(TABLE 1)**

IFTA-stratified

Proteinuria reductions were consistent **(FIGURE 1)**

IFTA ≤25%: eGFR stabilisation achieved over 52 weeks

IFTA 26–50%: experienced an eGFR decline from baseline despite proteinuria reduction (lower than placebo annualised rate of -15.6 mL/min/1.73 m²/yr **(FIGURE 1)**

eGFR-stratified

Across all baseline eGFR strata, mean UPCR reduction >50% and eGFR stabilisation over the 52-weeks **(TABLE 2)**

TABLE 1. Characteristics at baseline by IFTA status and eGFR category

	Baseline IFTA in renal biopsy		Baseline eGFR (mL/min/1.73 m²)		
	≤25% (n=96)	26-50% (n=23)	>90 (n=47)	≤90–≥60 (n=34)	<60 (n=39)
Age, mean (SD), years	23.8 (14.3)	34.7 (18.9)	18.1 (7.9)	25.2 (13.4)	36.7 (19.5)
Adolescents (12–17 years), n (%)	50 (52.1)	3 (13.0)	29 (61.7)	14 (41.2)	10 (25.6)
Adults (≥18 years), n (%)	46 (47.9)	20 (86.9)	18 (38.3)	20 (58.8)	29 (74.4)
Sex, female, n (%)	56 (58.3)	11 (47.8)	28 (59.6)	23 (67.6)	17 (43.6)
Baseline triplicate FMU UPCR, mean (SD), g/g	3.0 (2.5)	2.8 (1.8)	2.2 (1.5)	2.9 (2.0)	3.9 (3.1)
Baseline eGFR, mean (SD), mL/min/1.73 m²	82.5 (34.0)	66.7 (35.0)	114.5 (17.2)	76.1 (9.7)	38.8 (10.8)
Underlying disease based on screening biopsy, n (%)					
C3G	70 (72.9)	21 (91.3)	38 (80.9)	24 (70.6)	30 (76.9)
Primary IC-MPGN	26 (27.1)	2 (8.7)	9 (19.1)	10 (29.4)	9 (23.1)
Time since diagnosis, mean (SD), years*	3.4 (2.6)	3.2 (3.4)	4.0 (2.7)	2.6 (2.5)	3.1 (2.9)

* For patients grouped by baseline IFTA: ≤25% n=90; 26-50% n=22. For patients grouped by baseline eGFR: >90 n=45; ≤90–≥60 n=33; <60 n=34.
C3G, C3 glomerulopathy; eGFR, estimated glomerular filtration rate; FMU, first-morning urine; IC-MPGN, immune complex-membranoproliferative glomerulonephritis; IFTA, interstitial fibrosis/tubular atrophy; SD, standard deviation; UPCR, urine protein:creatinine ratio.

TABLE 2. Change from baseline in UPCR and eGFR at Week 52 by baseline eGFR category

	Baseline eGFR (mL/min/1.73 m²)		
	>90 (n=45)	≤90–≥60 (n=34)	<60 (n=39)
UPCR LS mean CFB (95% CI), %*	-63.8 (-73.0, -51.5)	-63.1 (-74.3, -47.1)	-63.1 (-74.0, -47.6)
eGFR LS mean CFB (95% CI), mL/min/1.73 m²	+1.6 (-4.4, 7.7)	-3.3 (-11.8, 5.2)	+1.3 (-3.8, 6.4)

* Geometric means and % change estimated from exponentiated LS mean differences from a MMRM of log-transformed UPCR. CI, confidence interval; CFB, change from baseline to Week 52; eGFR, estimated glomerular

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Complement Factor B Inhibition in C3 Glomerulopathy: Real-World Experience

Marta Garcia Chacon[1], Veronica Coll-Brito[1], Elisabeth Massó Jiménez[1], Maya Sanchez Baya[1], Carles Cañameras Fugasot[1], Paula Rodríguez-Martínez[1], Mauricio Suárez-Franck[1], Jordi Bover Sanjuan[1], Jordi Ara[1], Iara Da Silva[1], Paula Anton-Pampols[1]

[1] University Hospital Germans Trias i Pujol, Barcelona, Spain

Germans Trias i Pujol Hospital
Institut Català de la Salut

INTRODUCTION

C3 glomerulopathy (C3G) is a rare complement-mediated kidney disease characterized by dysregulation of the alternative complement pathway, resulting in predominant glomerular C3 deposition and progressive renal injury. Current therapeutic strategies remain limited and rely mainly on supportive measures and non-specific immunosuppression. We present a case of refractory C3G successfully treated with iptacopan, a selective factor B inhibitor.

AIM

To report a real-world case of C3 glomerulopathy with prolonged follow-up demonstrating sustained clinical remission, normalization of complement activity, and preservation of renal function following treatment with iptacopan after failure of conventional immunosuppressive therapy.

CASE PRESENTATION

42-year-old man with proteinuria, microscopic haematuria and preserved kidney function. Kidney biopsy confirmed C3 glomerulopathy. Persistent disease activity despite ACEi, PDN and MMF led to initiation of iptacopan.

RESULTS

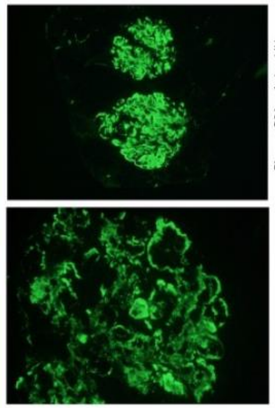

Figure 1. Immunofluorescence findings in the patient's biopsy. Upper panel, mesangial C3 deposition with nodular formation. Lower panel, C3 deposits with a granular and pseudolinear ribbon-like pattern along the capillary wall

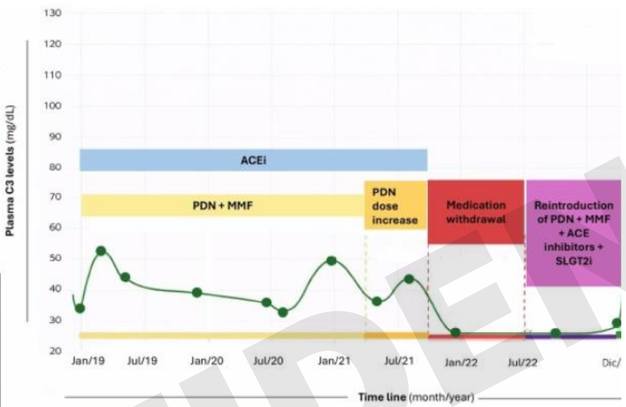

Figure 2. Longitudinal assessment of plasma complement C3 levels demonstrates poor response to no immunosuppresive therapies, with a sustained increase in serum C3 levels after iniciation of iptacopan. angiotensin-converting enzyme inhibitors; PDN, prednisone; MMF, mycophenolate mofetil; SLGT2i, sodi 2 inhibitors.

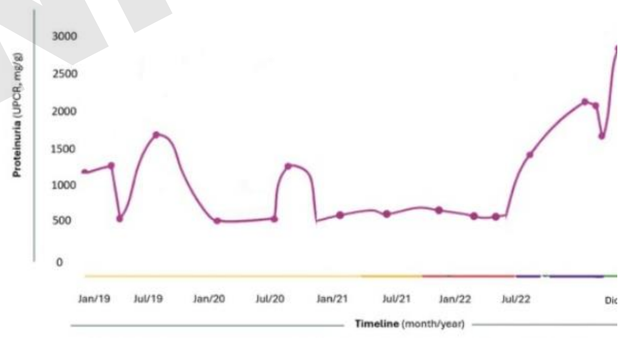

Figure 3. Longitudinal assessment of proteinuria levels demonstrates a sustained decrease in proteinuri

CONCLUSIONS

This case is noteworthy because the sustained response exceeds the follow-up reported in most published real-world experiences with Iptacopan. Furthermore, a response was also observed despite negative genetic testing, suggesting that the benefits of proximal complement inhibition may extend beyond genetically defined subgroups. The case also highlights an important unanswered question in the management of C3 glomerulopathy: the optimal duration of Iptacopan therapy and the safety of treatment discontinuation still remain unknown.

ACKNOWLEDGEMENTS

We thank the clinical and laboratory teams for their expertise in complement diagnostics and the management of patients receiving complement-targeted therapies.

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Longitudinal Proteinuria and eGFR Patterns as Predictors of Graft Failure in Recurrent C3 Glomerulopathy and Immune-Complex MPGN

JE. Ruiz-Cabello[1], M. Praga[2], A. Andrés[1,3], F. Caravaca-Fontán[1,3] on behalf of the International Recurrent C3G/IC-MPGN Collaborative Group*

1 Department of Nephrology, Hospital Universitario 12 de Octubre, Madrid, Spain.
2 Department of Medicine, Complutense University, Madrid, Spain.
3 Department of Nephrology, Research Institute Hospital 12 de Octubre (imas12), Madrid, Spain

GRUPO DE TRABAJO Sociedad Española de Nefrología
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Club Nefropatología Grupo Enfermedades Glomerulares
SENTRA

INTRODUCTION

C3 Glomerulopathy (C3G) and Immune-Complex Membranoproliferative Glomerulonephritis (IC-MPGN) frequently recur after kidney transplantation. The natural history of recurrent disease and the prognostic value of longitudinal kidney biomarkers remain poorly defined, limiting our ability to risk-stratify patients for emerging proximal complement-targeted therapies.

AIM

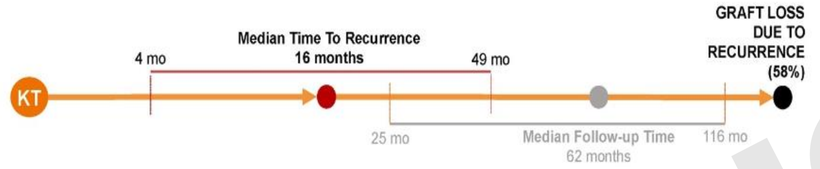
To characterize the **natural history** of biopsy-proven recurrent C3G and primary IC-MPGN, specifically evaluating whether **longitudinal changes in proteinuria** and **estimated glomerular filtration rate (eGFR)** can serve as dynamic **surrogate markers of graft failure.**

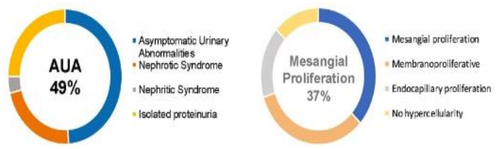
METHODS

- **Study Design**: Multinational, retrospective cohort study (2001 – 2023) involving 48 centers across 11 countries.
- **Population**: 134 KT recipients with biopsy-proven recurrent C3G or primary IC-MPGN
- **Inclusion criteria**: ≥4 consecutive measurements of eGFR and proteinuria after recurrence, with ≥6 months follow-up.
- **Statistical Analysis**: Linear mixed-effects and Bayesian joint longitudinal-survival models evaluated biomarker trajectories and graft failure risk. Complement genetic and autoantibody testing

RESULTS

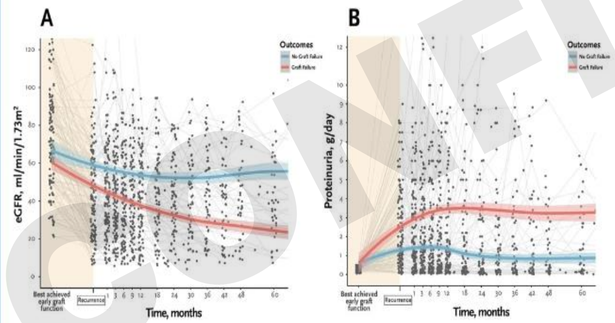
N = 134
80 C3GN (60%)
16 DDD (12%)
38 IC-MPGN (28%)



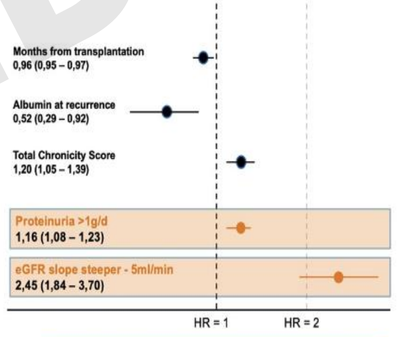


Protocol vs **Indication** Biopsy
11 % vs 89%
3 [IQR 2–4] vs 23 [IQR 7–58]
p<0.001

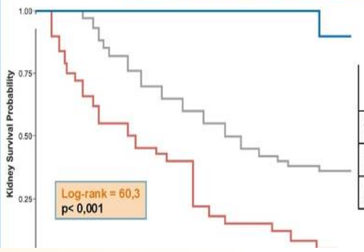
Longitudinal trajectories of proteinuria and eGFR



Graft failure predictors - Joint model analysis



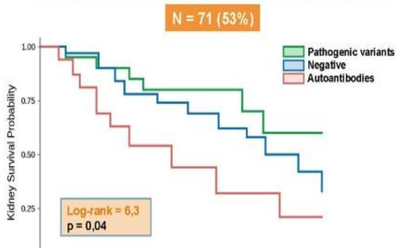
Prognostic cut-off for eGFR-slope



eGFR Decline (ml/min)	Stable (< -1,5)	Moderate (-1,5 to -5)	Rapid (> -5)	p
Age at recurrence	42 (34–51)	45 (35–61)	31 (22–49)	0,03
Time to recurrence	31 (3–115)	18 (6–42)	4 (2–15)	0,004
Proteinuria at recurrence	1.3 (0.6–3)	2 (0.9–4)	3.2 (1.5–4.5)	0,003

Impact of pathogenic drivers on graft survival

N = 71 (53%)



CONCLUSIONS

1.- Recurrent C3G and primary IC-MPGN after transplantation are associated with poor long-term outcomes, substantial histological injury, and marked biological heterogeneity.

2.- Longitudinal eGFR and proteinuria provide powerful prognostic information, and clinically meaningful thresholds (>1 g/day time-averaged proteinuria; eGFR slope steeper than –5 mL/min/1.73 m²/year) to refine risk stratification.

3.- These dynamic biomarkers may serve as surrogate endpoints, underscoring the need for prospective validation with emerging proximal complement therapies.

4.- While the overall rate of graft failure is similar across the different biological groups, the underlying pathogenic mechanism may dictate how quickly that failure occurs.

ACKNOWLEDGEMENTS

* This was an international collaborative study that involved 53 co-authors across 48 centers in 11 countries. We acknowledge their valuable contribution. Work in this study was supported by the Instituto Salud Carlos III (ISCIII), project PI24/00140.

CONTACT INFORMATION

fcaravacaf@gmail.com
@JoseRCS

63rd ERA CONGRESS
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Challenge Your Thinking

¿Se puede ampliar el uso de la inhibición del complemento en la enfermedad glomerular?

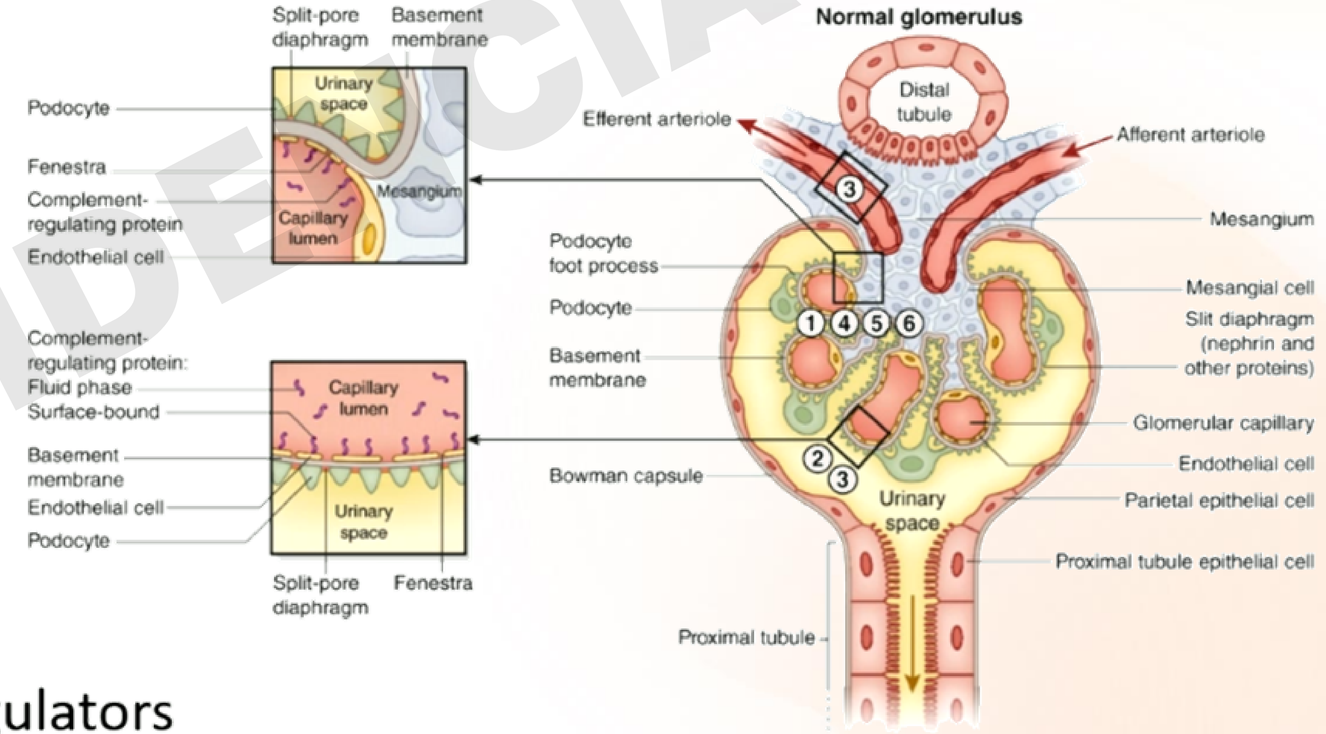
Marina Vivarelli
Bambino Gesù Children's Hospital IRCCS Rome, ITALY

¿Por qué el riñón se ve afectado con tanta frecuencia?

1) High blood hydrostatic pressure and filtration of plasma

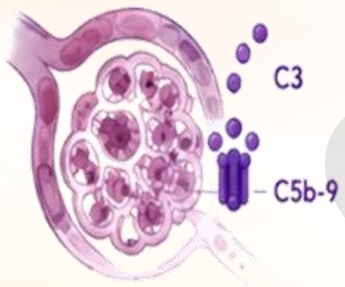
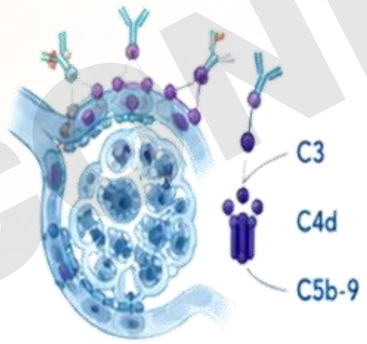
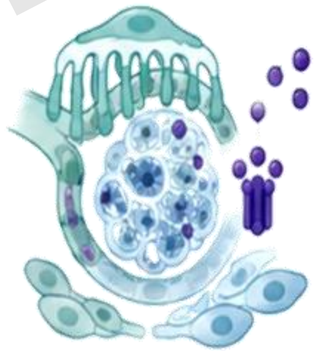
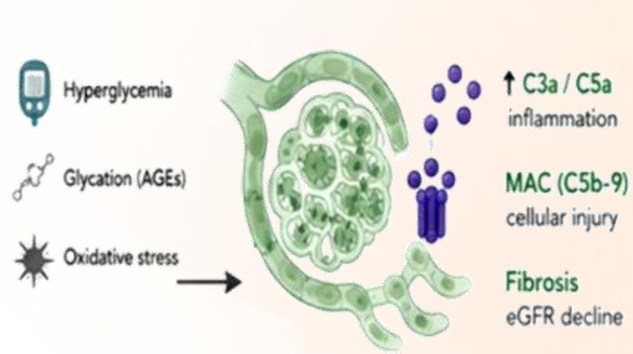
2) Fenestrae in glomerular endothelial cells

3) Absence of intrinsic complement regulators on the GBM



El papel del complemento en el espectro de las enfermedades renales

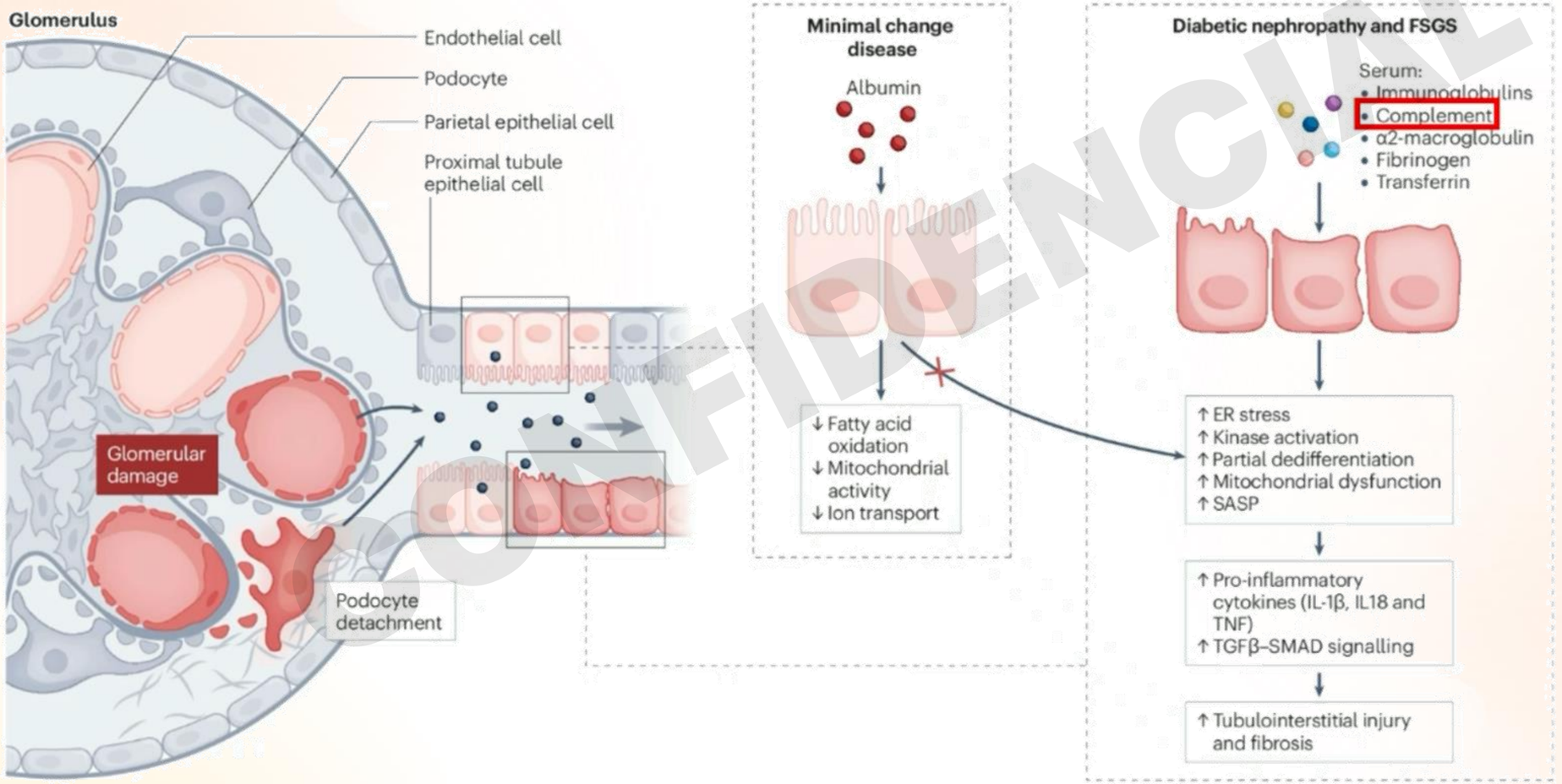
Complement activation exists across a broad spectrum of kidney diseases, with a role that shifts from initiator to amplifier.

PRIMARY DRIVER (INITIATOR)	CONTRIBUTOR + AMPLIFIER (MIXED ROLE)		SECONDARY DRIVER (AMPLIFIER)
Complement-mediated diseases C3 glomerulopathy aHUS	IgA nephropathy (IgAN) Complement contributes to inflammation and injury	FSGS Complement amplifies podocyte injury and tubulointerstitial fibrosis	Diabetic nephropathy (DKD) Complement amplifies metabolic injury and drives progression
			
Complement activation is the initiating event	Complement acts as a pathogenic contributor and amplifier	Complement acts primarily as a secondary amplifier of chronic injury and fibrosis	Complement acts primarily as a secondary amplifier of chronic kidney injury and fibrosis

Academy

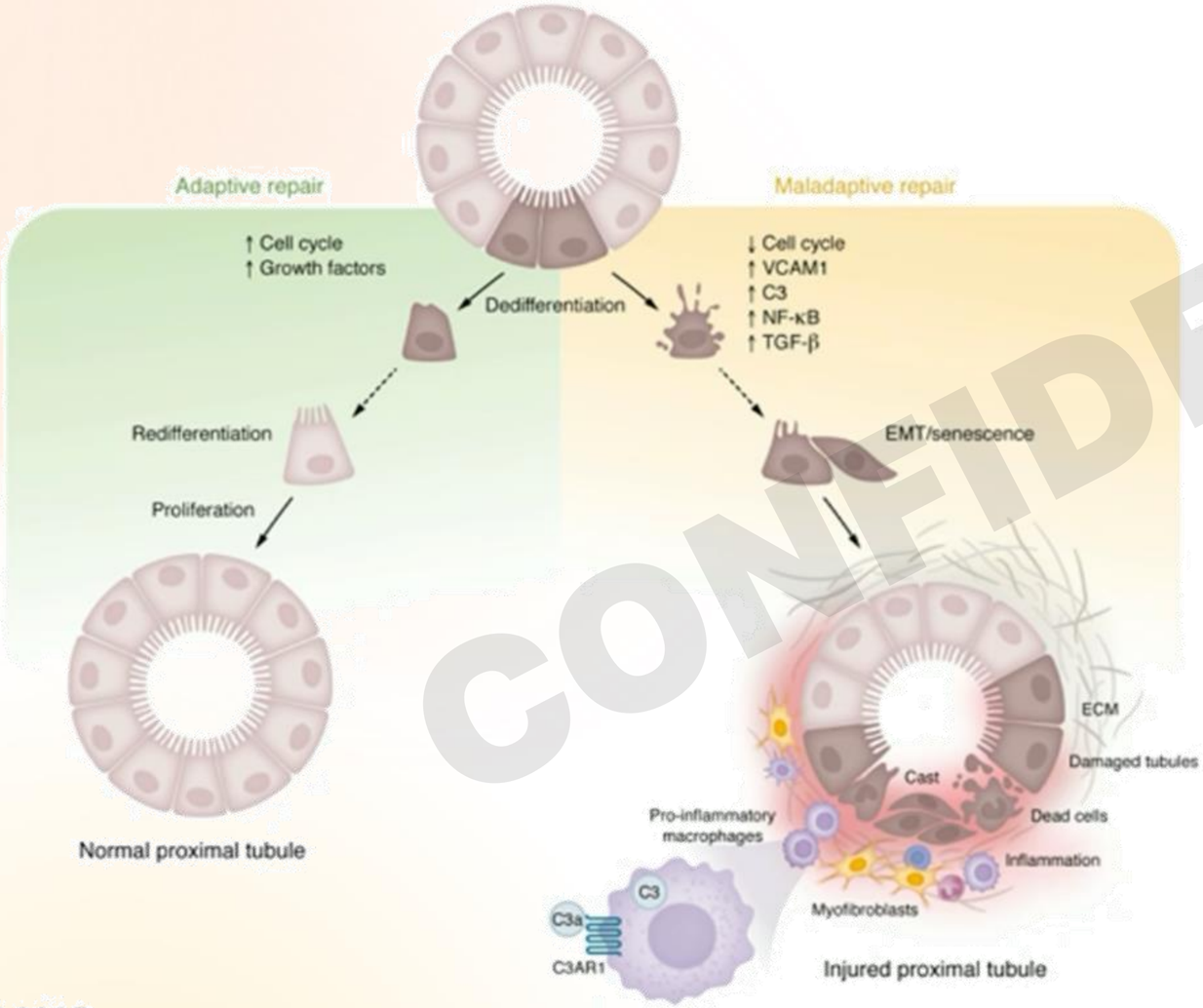
Daño extraglomerular mediado por el complemento

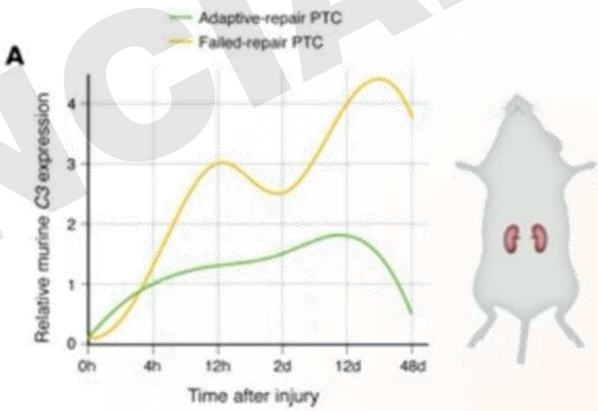
El daño en la barrera de filtracion glomerular puede exponer las células tubulares al complemento y puede promover el TIF y el deterioro progresivo de la función renal

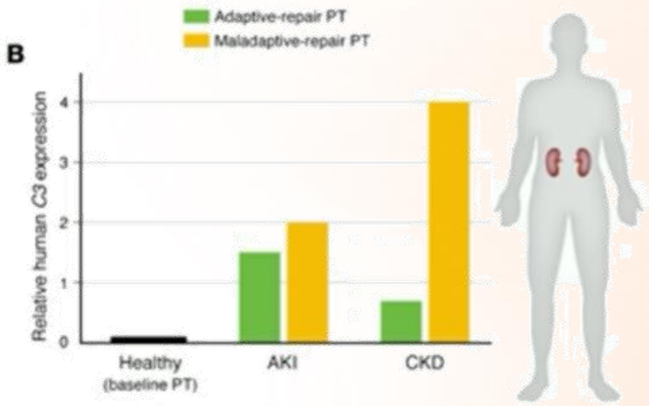


Fogo & Harris, Nat Rev Nephrology 2025.

La expresión tubular de C3 es una característica distintiva de la respuesta maladaptativa a una lesión

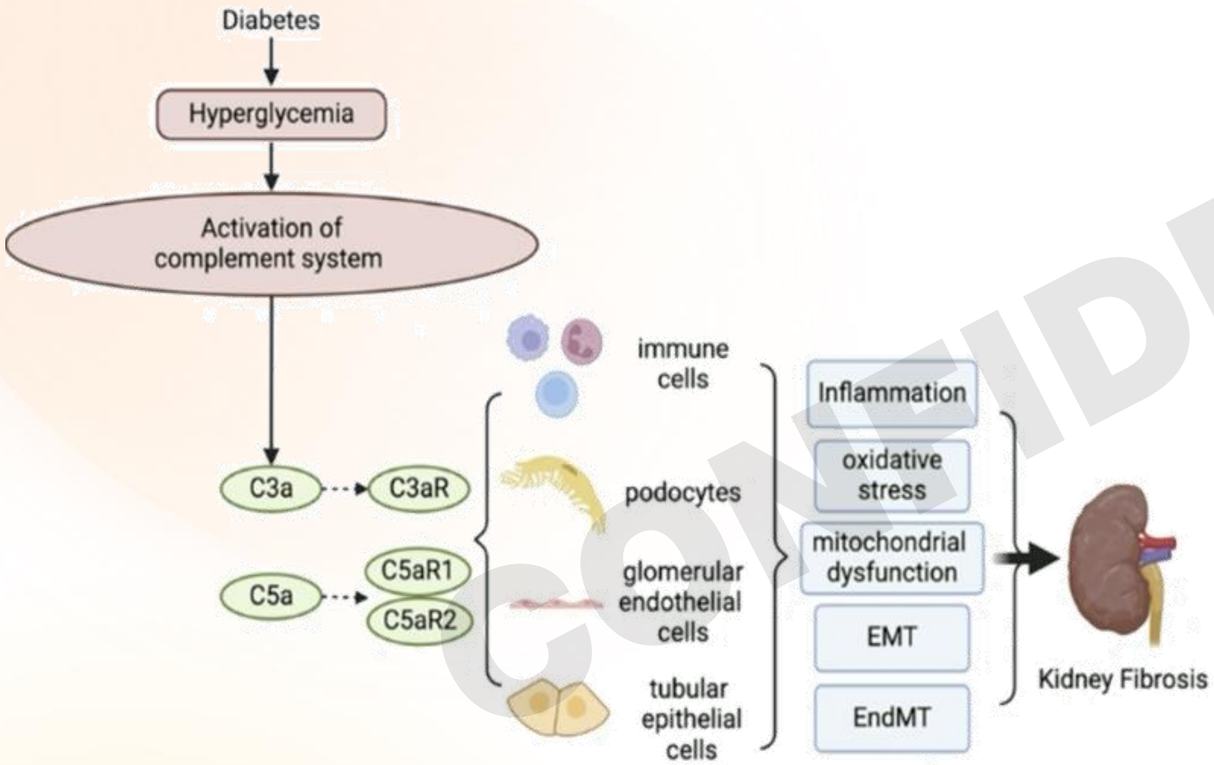




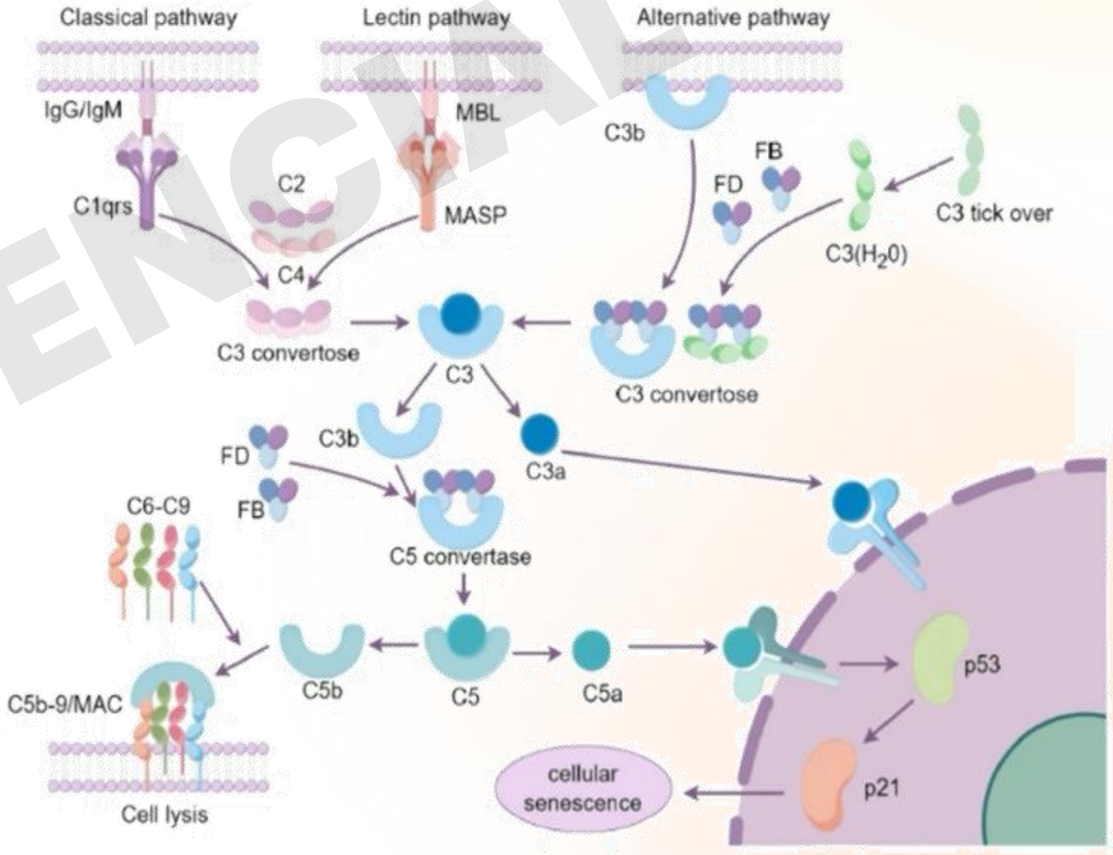


Portilla, JCI 2025

En la DKD, la hiperglucemia induce la senescencia de las células epiteliales tubulares a través de C5a y C3a

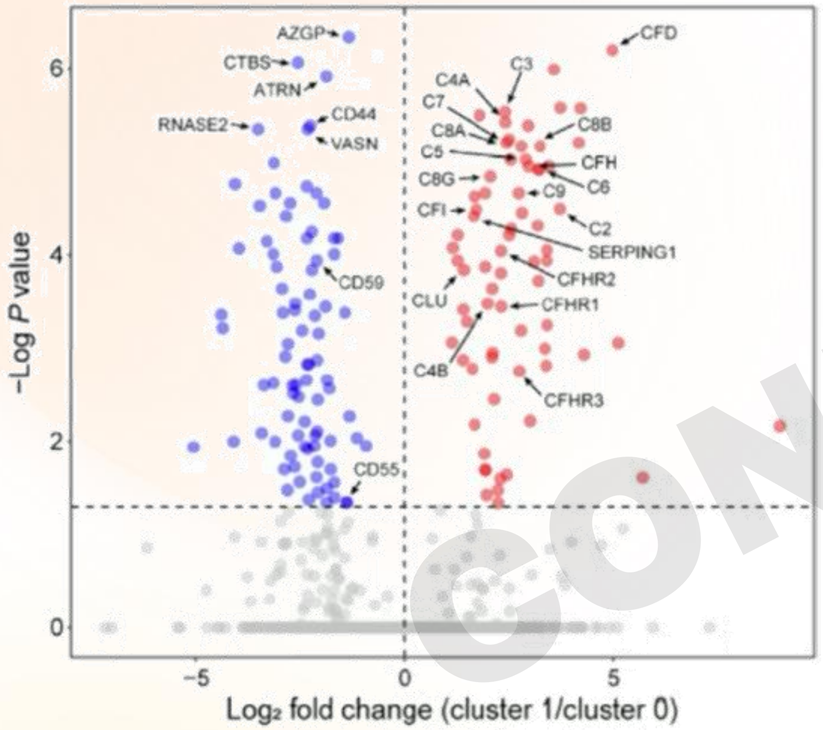


Ma, Diabetic Medicine 2024



Guo, Annals Medicine 2025

En la DKD, un score de complemento (espectrometría de masas en muestras de orina) predice una progresión rápida de la nefropatía.



Variables	Total ($N = 64$)	Cluster 0 ($n = 40$)	Cluster 1 ($n = 24$)	P
Age (yrs)	53.3 ± 11.1	52.1 ± 10.9	55.2 ± 11.5	0.282
Male (%)	71.9	75.0	66.7	0.667
Body mass index (kg/m^2)	25.0 ± 3.3	25.2 ± 3.1	24.7 ± 3.5	0.594
Hypertension (%)	92.2	95.0	87.5	0.355
Diabetes duration (yrs)	10 (4–14)	8 (2–12)	11 (4–20)	0.278
Diabetic retinopathy (%)	54.7	52.5	58.3	0.846
Diabetic neuropathy (%)	29.7	30.0	29.2	1.000
Ischemic heart disease (%)	7.8	12.5	0	0.148
Cerebrovascular disease (%)	4.7	2.5	8.3	0.551
Hemoglobin A1c (%)	7.2 (6.6–8.4)	7.2 (6.6–7.6)	7.4 (6.8–8.9)	0.178
PCR (g/g)	3.1 (1.7–7.0)	2.2 (1.2–3.6)	7.1 (4.9–9.5)	<0.001
eGFR (ml/min per 1.73 m^2)	56 (44–75)	61.2 (48–85)	43 (33–60)	<0.001
Glomerular classification (%)				
I	14.1	17.5	8.3	0.003
IIA	12.5	17.5	4.2	
IIB	32.8	42.5	16.7	
III	12.5	10.0	16.7	
IV	28.1	12.5	54.2	
Interstitial fibrosis and tubular atrophy (%)				
0	4.7	7.5	0.0	<0.001
1	35.9	47.5	16.7	
2	46.9	45.0	50.0	
3	12.5	0.0	33.3	

Yun, KI Reports 2025

Conclusiones

- La activación del **complemento** en diferentes partes del riñón desempeña un **papel** importante en el desarrollo del daño en un **gran número** de **enfermedades renales**.
- Esto sucede:
    1. Dentro de los **capilares** glomerulares y arteriolas, provocando microangiopatía trombótica en el aHUS mediado por complemento pero también en la vasculitis APS/rehat.
    2. Dentro de los **glomérulos**, lo que impulsa la proliferación mesangial y la alteración de la integridad de la GBM en varias enfermedades glomerulares.
    3. La producción **tubular** de C3 es uno de los factores que impulsan la transición epitelio→ mesénquima que precede a la **fibrosis tubulointersticial**.
- La inhibición del complemento es **transformadora** en el **aHUS** y ha demostrado eficacia en **C3G, IC-MPGN primaria, AAV** e **IgAN**. Su papel beneficioso **podría extenderse** a muchas otras enfermedades renales, ya sea agudas o crónicas. Sin embargo, esto sigue siendo especulativo.

Conclusiones

- En la práctica clínica, debemos hacer un **buen diagnóstico** del paciente con los datos clínicos, histológicos, funcionales (y genéticos en los casos necesarios), y en la medida de lo posible, **perfilarlo/estratificarlo**, para poder decidir el **tratamiento más adecuado** en cada caso.

- Aún **quedan muchas cuestiones por resolver**….
  - Ahondar en los perfiles de pacientes.
  - Tiempos de tratamientos.
  - Combinación de tratamientos.

Academy

¡Muchas gracias!