Mesa de Nefrología clínica

Sábado, 28 de octubre de 2023. 11:30-12:45 h

RECIDIVA DE ENFERMEDAD GLOMERULAR EN TRASPLANTE RENAL

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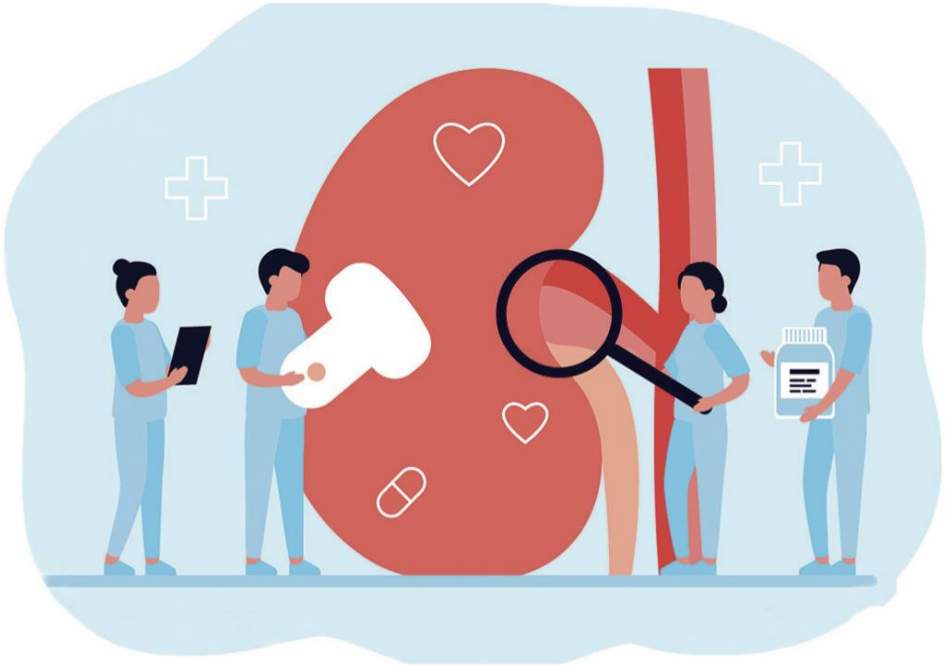
Servizo de Nefroloxía

SERVIZO GALEGO de SAÚDE | Complexo Hospitalario Universitario A Coruña
A Coruña

- Declaro que no tengo conflictos de interés

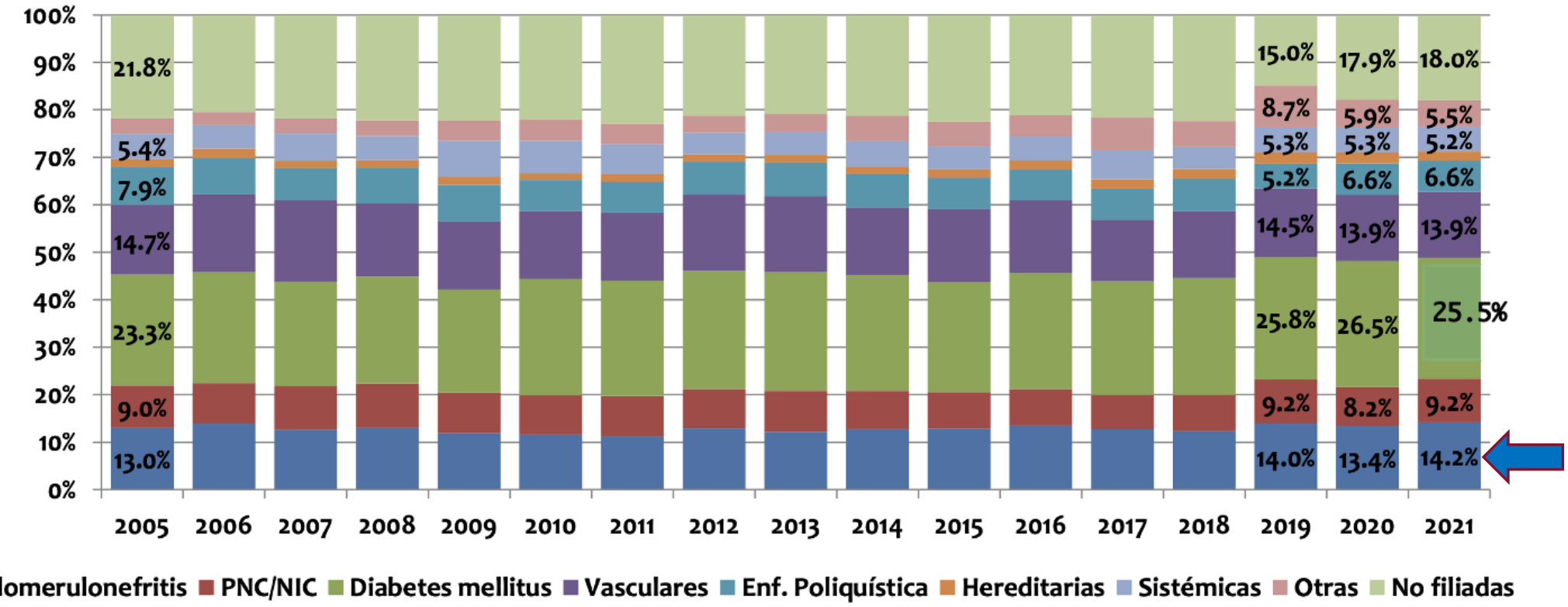
?



Incidencia

Evolución de la Incidencia por ERP (%)

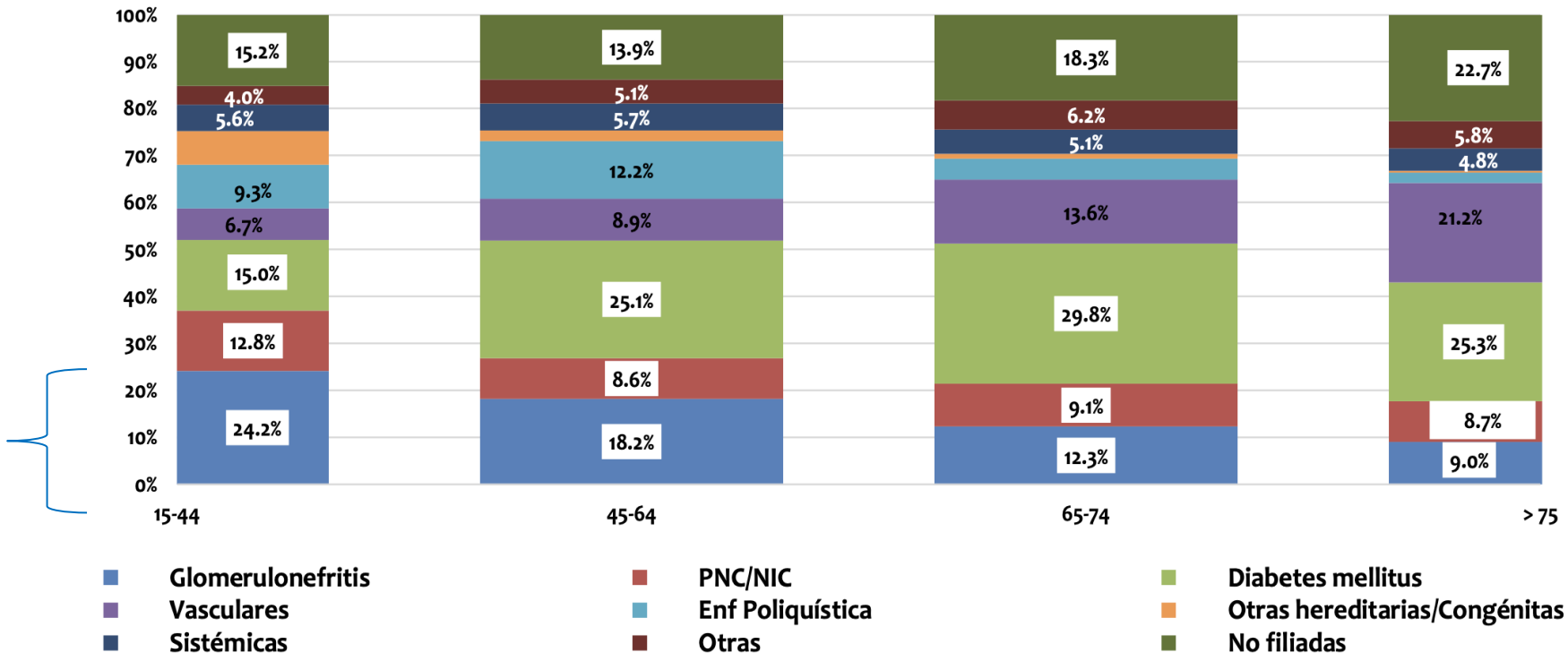
7084 (149.5 pmp)



% calculado sobre incidentes con ERP registrada – n= 6802/7084

Incidencia

ERP por grupo de edad (%)
7084 (149.5 pmp)

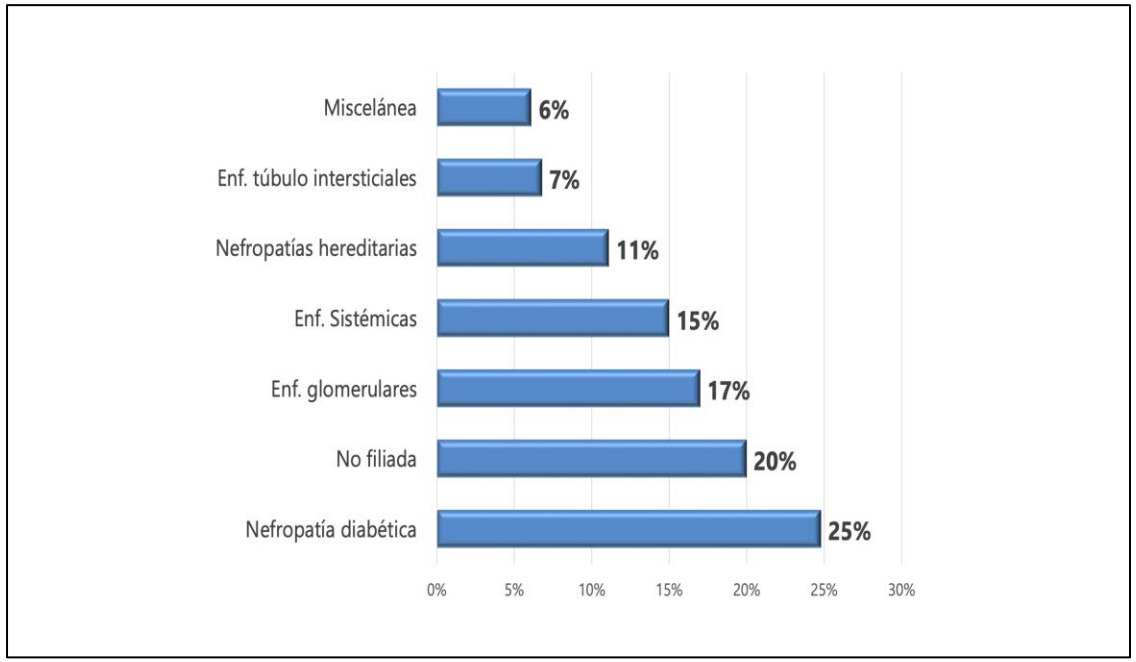


% calculado sobre incidentes con ERP registrada – n= 6802 / 7084

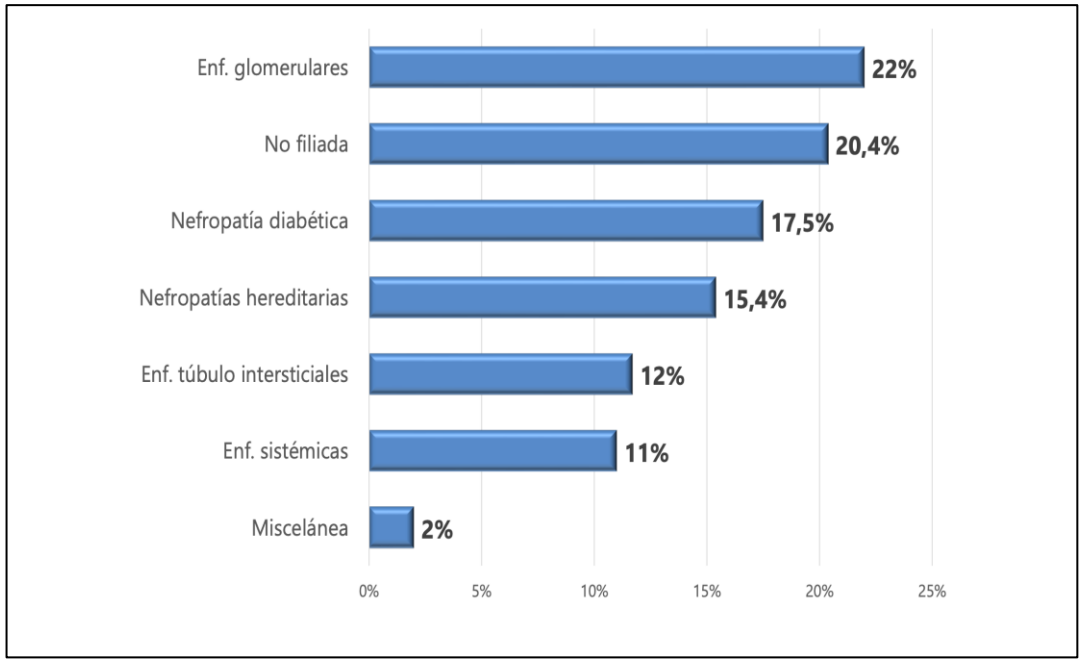
ORGANIZA:

SGAN
sociedad gallega de nefrología

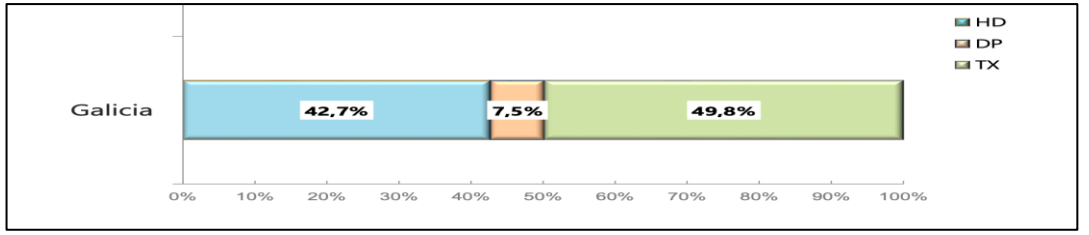
Incidentes

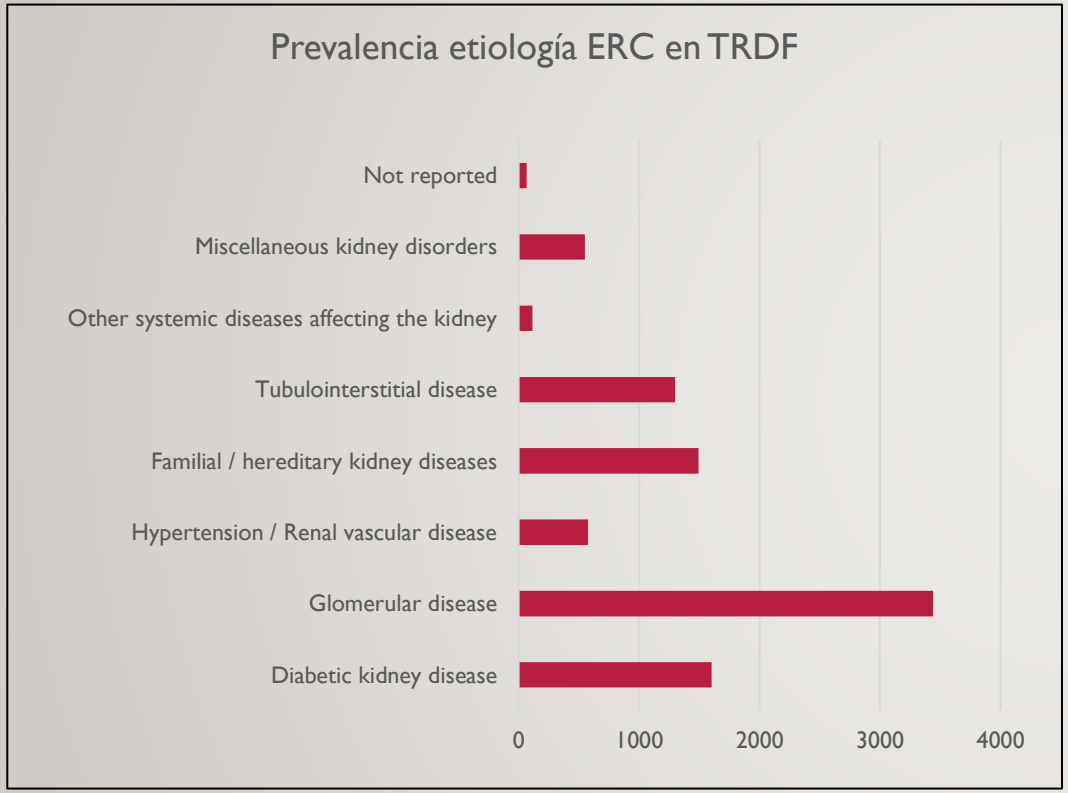


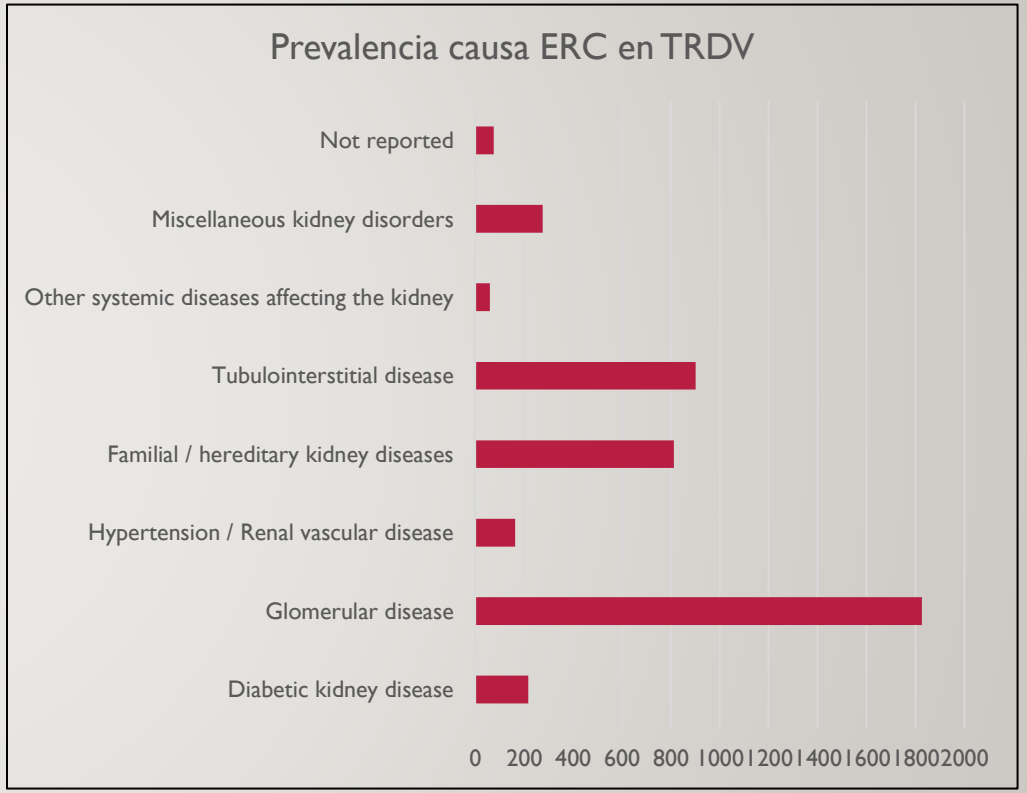
Prevalentes



REXER año 2021

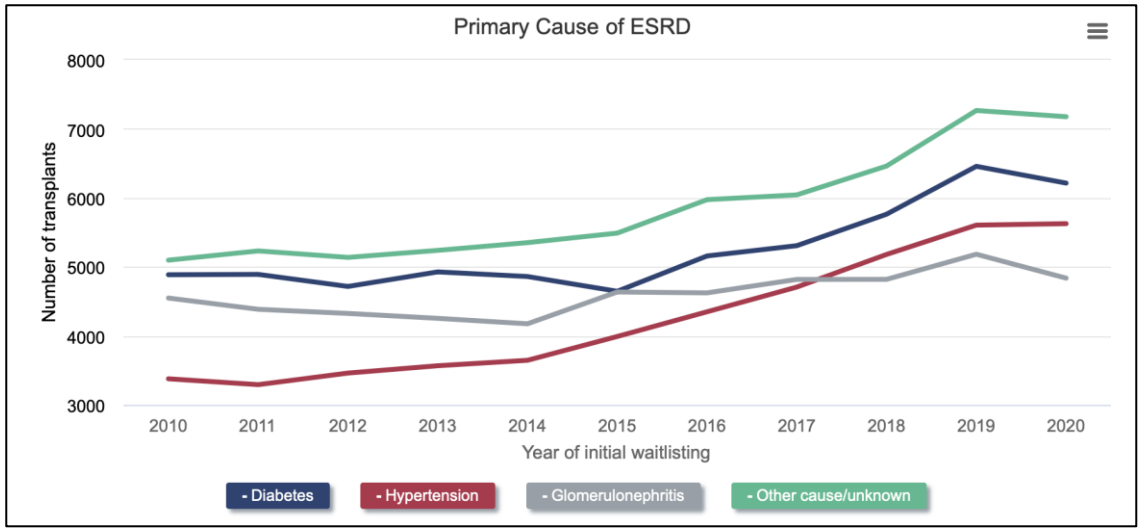


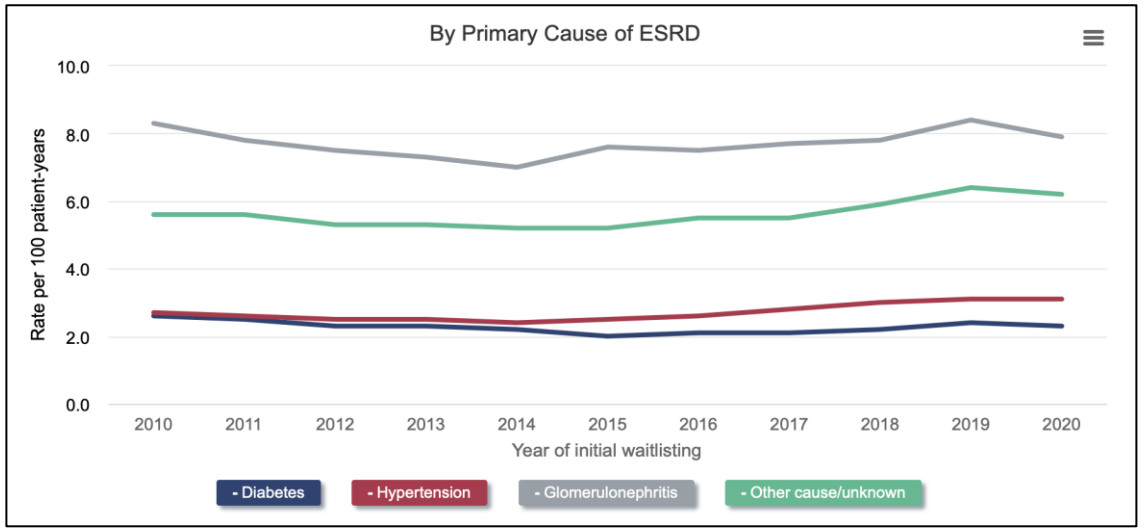




ANZDATA 46th Annual Report 2023 (Data to 2022)

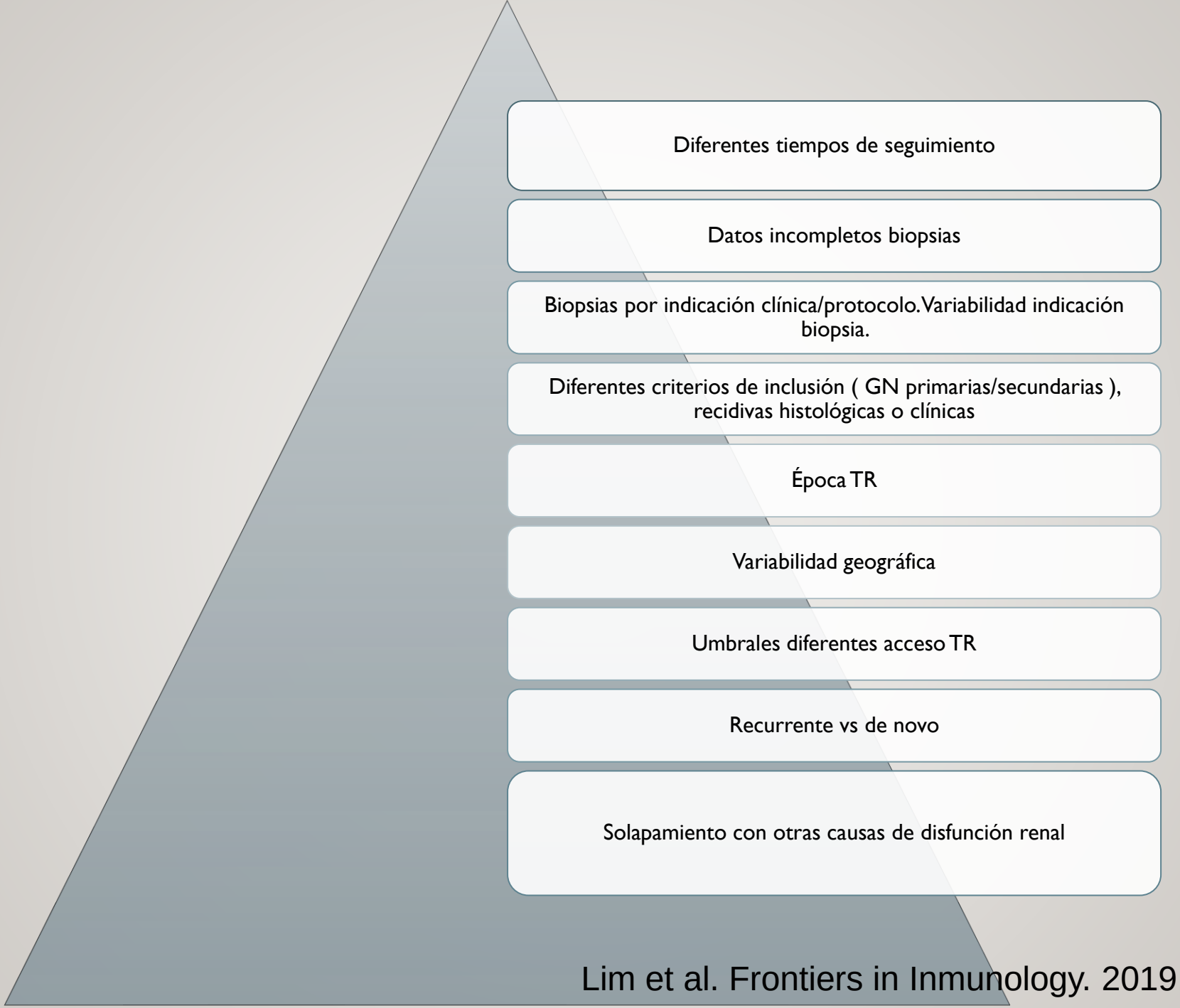
OPTN | ORGAN PROCUREMENT AND TRANSPLANTATION NETWORK





Data source: USRDS ESRD database and OPTN waitlisting history. All patients were followed until December 31, 2019. Transplants included kidney-only transplants and excluded simultaneous pancreas-kidney transplants.

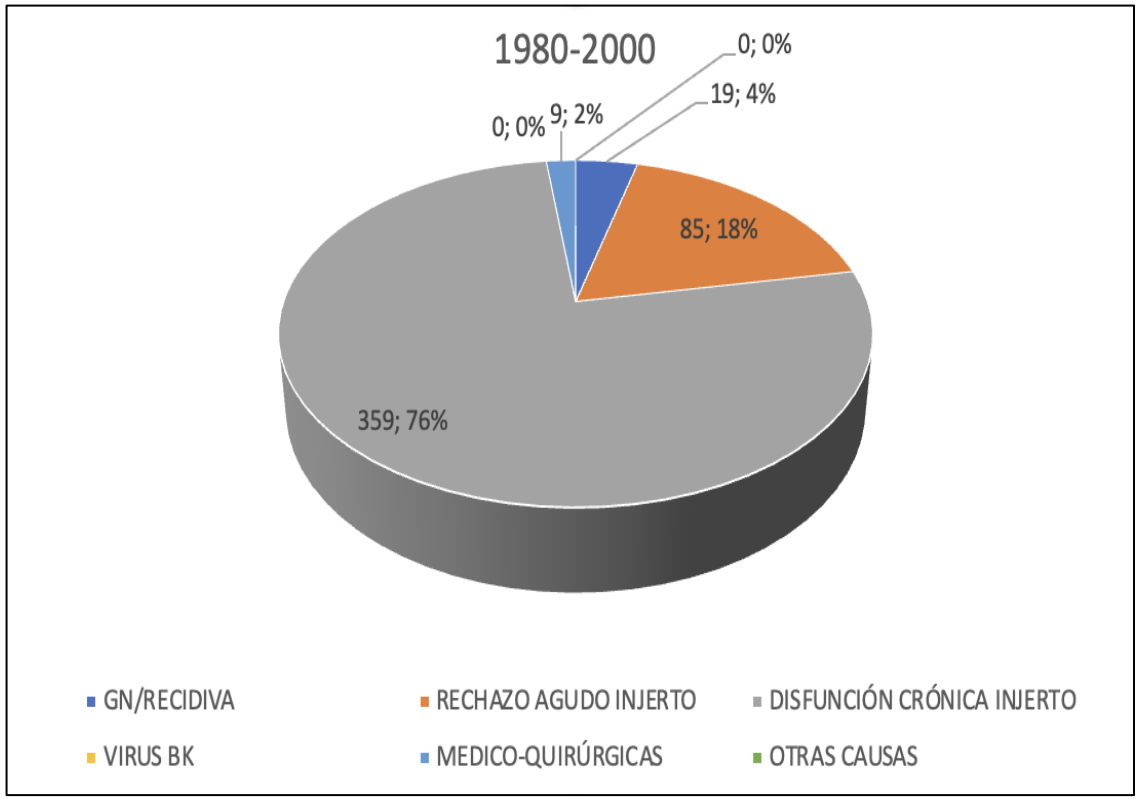
PREVALENCIA DE RECURRENCIA GN EN TR (GN como enfermedad de base)

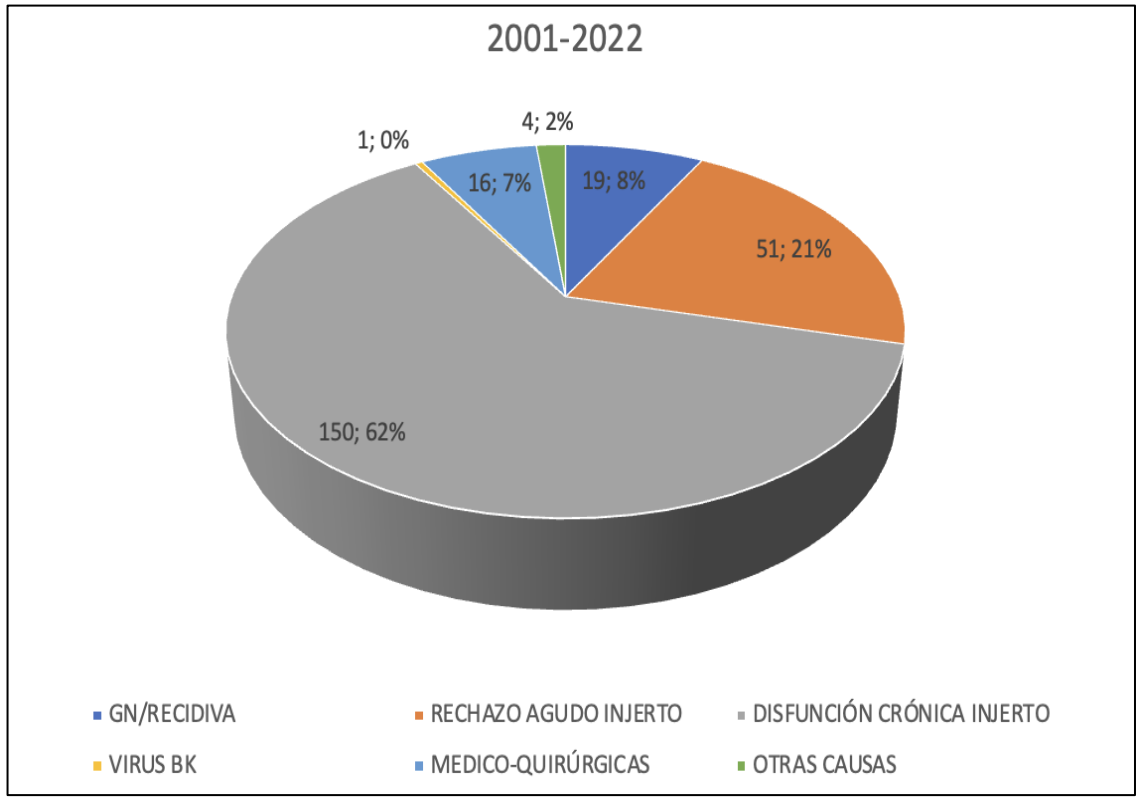


Lim et al. Frontiers in Inmunology. 2019.Vol 10,Art 1944

PÉRDIDA DE INJERTO POR RECURRENCIA GN

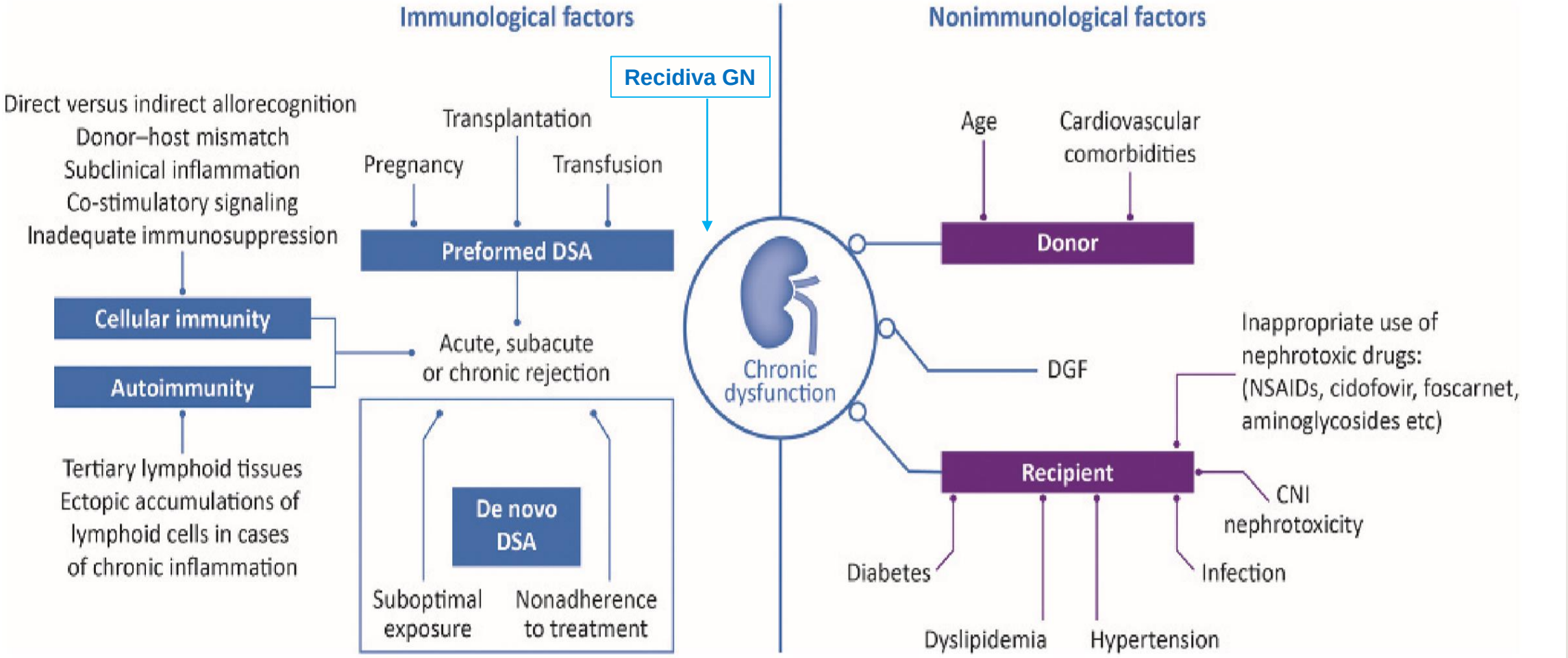
CAUSAS PÉRDIDA INJERTO*





*Censurada por muerte y trombosis vasculares precoces

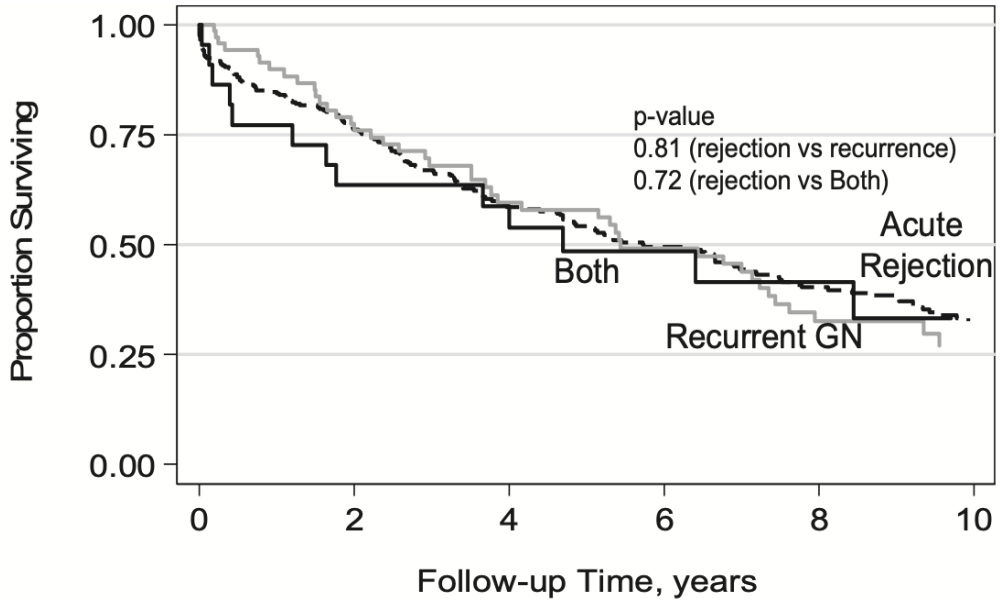
DISFUNCIÓN CRÓNICA INJERTO



CNI, calcineurin inhibitor; DGF, delayed graft function; DSA, donor-specific antibodies.

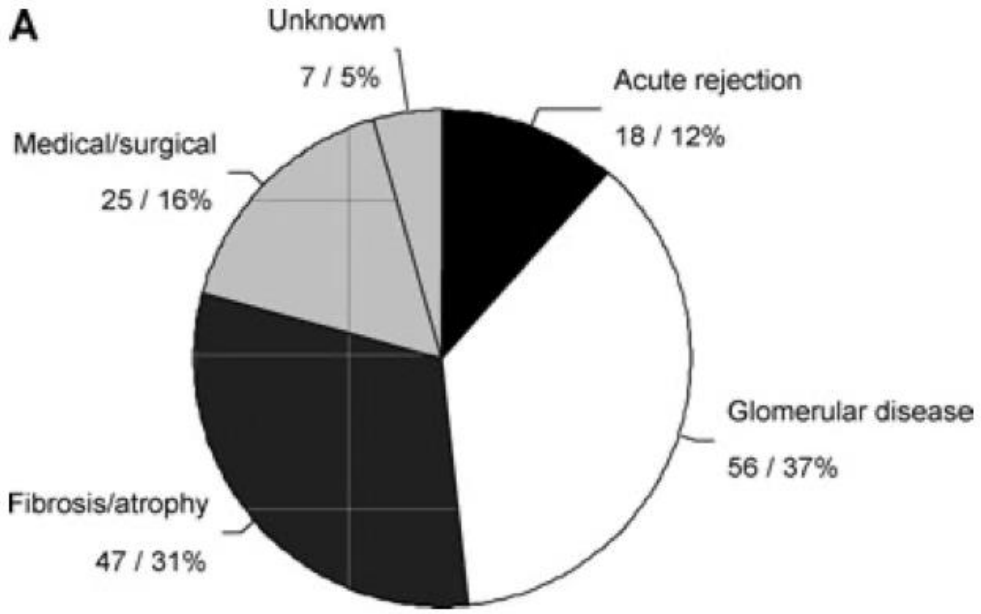
1. Jevnikar AM et al. Clin J Am Soc Nephrol 2008;3(Suppl 2):S56–67; 2. Pazhayattil GS et al. Int J Nephrol Renovasc Dis 2014;7:457–468; 3. Sellarés J et al. Am J Transplant 2012;12(2):388–399; 4. Lefaucheur C et al. J Am Soc Nephrol 2010;21(8):1398–1406. 5. Koenig A et al. Front Immunol 2016;7:646; 6. Valenzuela NM et al. Methods Mol Biol 2013;1034:41–70; 7. Siedlecki A et al. Am J Transplant 2011;11:2279–2296; 8. Puttarajappa C et al. J Transplant 2012;2012:193724.

862 TR con GN

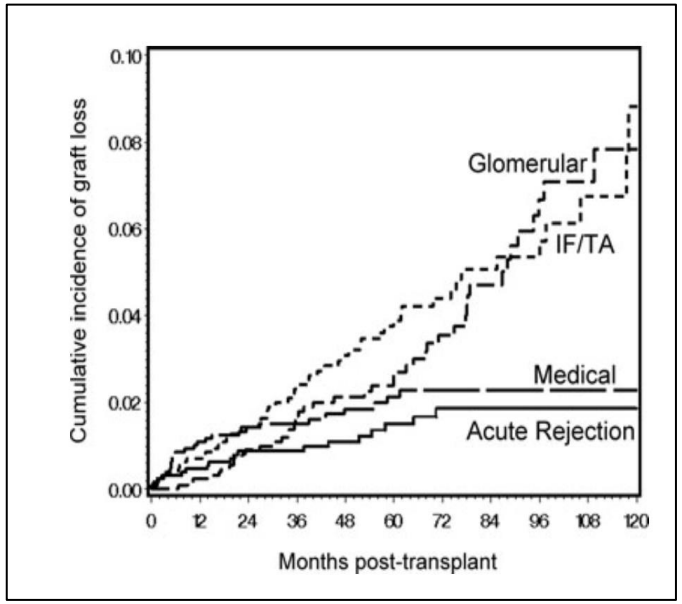


Singh et al. Clinical Transplantation. 2019;33:e13738

1996-2006
1317 TR. 330 pérdidas.
138 éxitus con injerto funcionante
39 no función primaria



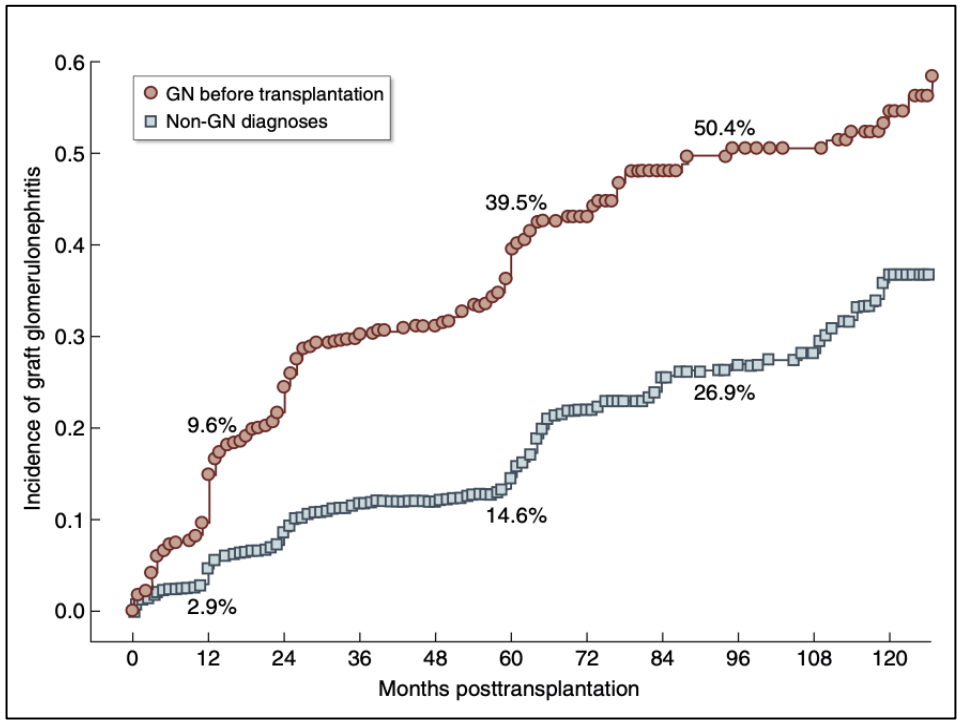
153 biopsiados (98%)



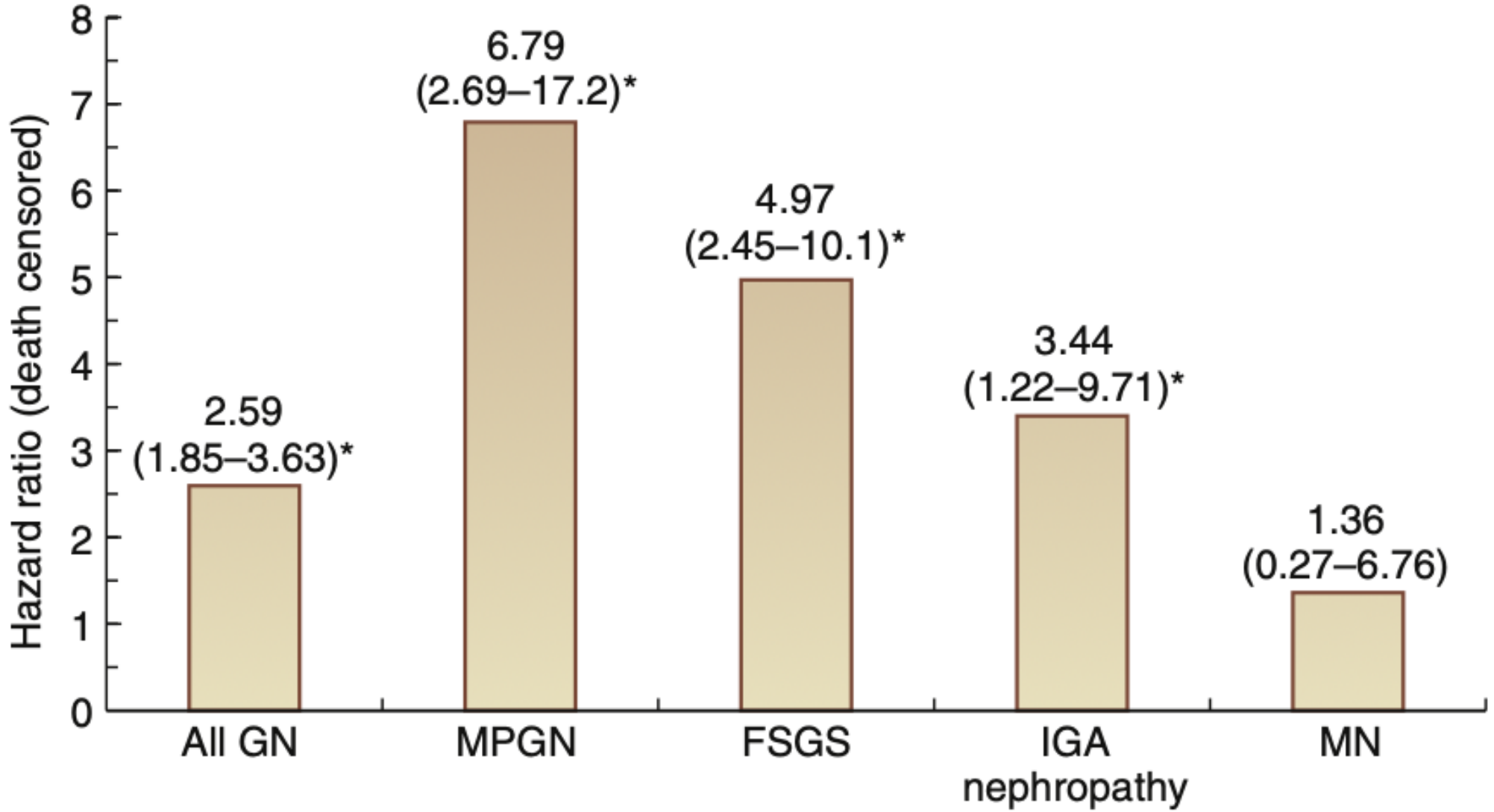
El-Zohgby et al. AJT. 2009;9;527-535

414 GN antes de TR
1282 no GN

- Estudios basados en biopsias de protocolo (0, 4, 24, 60,120 m) y no guiado por manifestaciones clínicas
- Biopsias estudiadas sistemáticamente con MO,IF y ME
- Recurrencia: histología compatible con GN
- GNM, NIgA, GNMP, GEFS



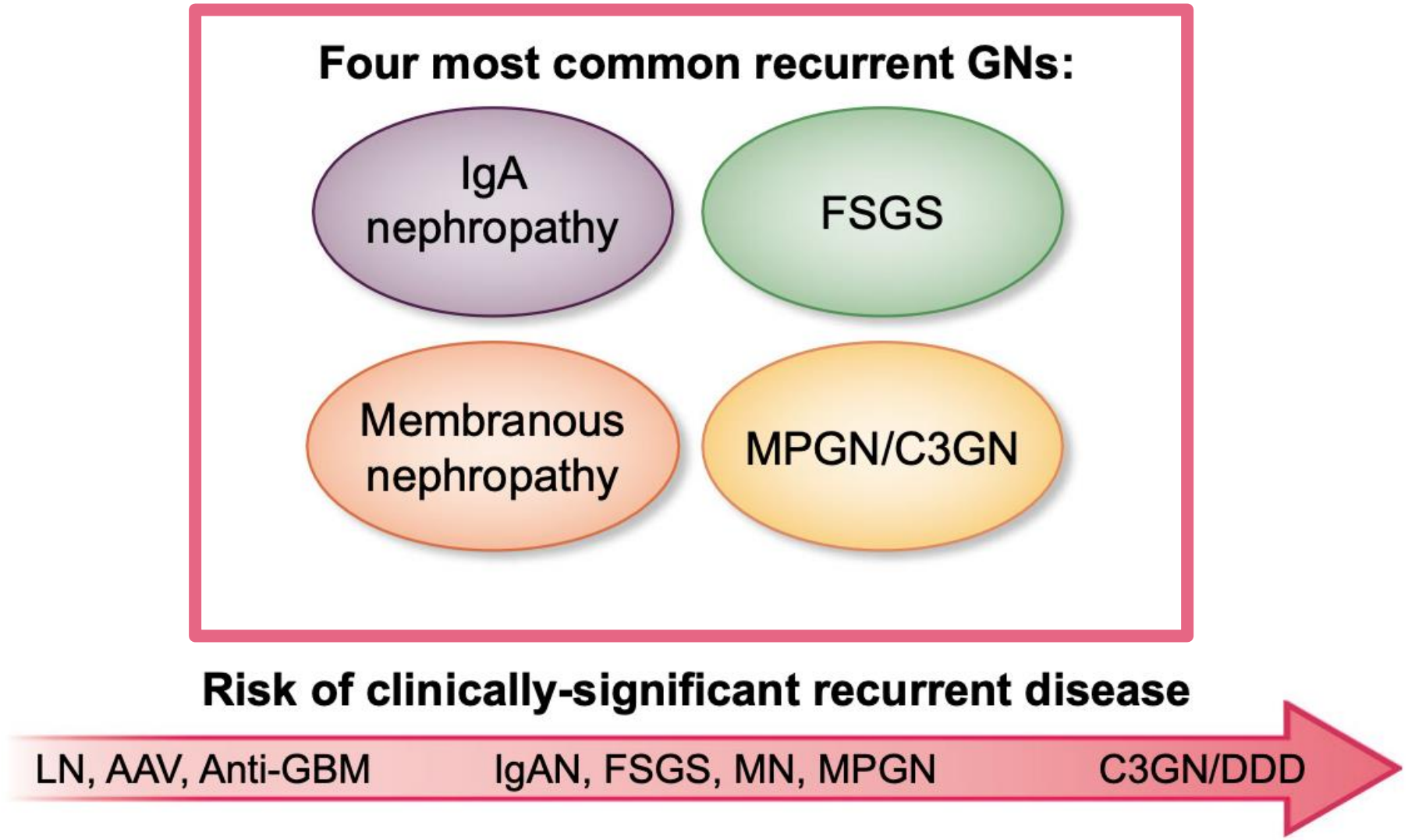
Cosio et al. Kidney International (2017) 91, 304–314



Cosio et al. Kidney International (2017) 91, 304–314

RECIDIVA GN EN TRASPLANTE RENAL

- Escasa información
- Gran variabilidad de incidencia recidiva/causa pérdida de injerto
- Dx por biopsias de protocolo/indicación clínica.
- Pacientes jóvenes con IS previa al TR
- Empeoran SV injerto ≥que RA
- Heterogeneidad


Four most common recurrent GNs:
IgA nephropathy
FSGS
Membranous nephropathy
MPGN/C3GN
Risk of clinically-significant recurrent disease
LN, AAV, Anti-GBM
IgAN, FSGS, MN, MPGN
C3GN/DDD

ORGANIZA:

SGAN
sociedad gallega de nefrología

Nefropatía IgA

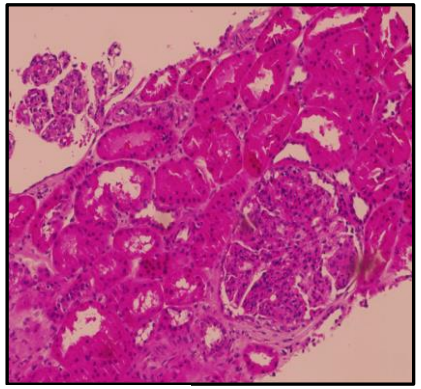
58 a

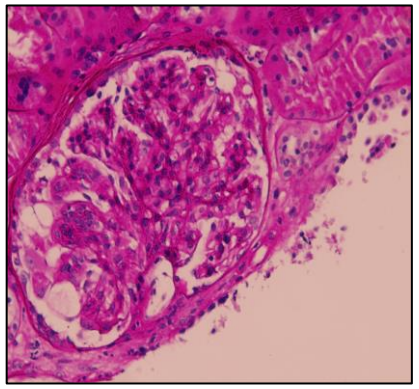
2018 Crp 1,1 mg/dl
Prot 2 g/d →BR→
NIgA (M1 E0 S0 T0 C0)

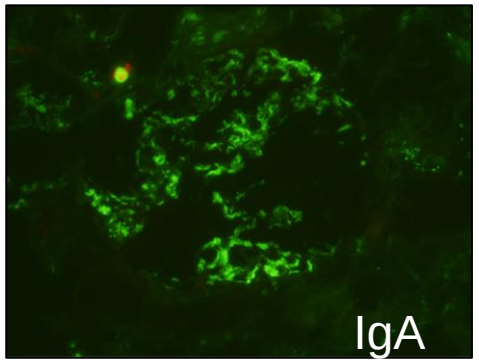
Prednisona 6 m.
→ MMF

TRDV 2022 (hermano)
Crp 1.4 mg/dl

3er m: Crp 1,7 mg/dl
Prot 1.2 g/d
DSA neg







Tto soporte
ARAII,iSLGT2

Prednisona oral
6 meses
Aumento MMF

Estudios genéticos sin alteraciones

Crp 2,6 mg/dl
Prot 1,4 g/d

Rebiopsia

RECURRENCIA NEFROPATIA IGA

- Prevalencia recurrencia 10-20 % a 10 años en biopsias por indicación clínica, hasta 50% biopsias de protocolo.
- Pérdida injerto hasta un 20-30 % a 10 a
- Tiempo medio: 3,4 años
  - *Uffing et al. TANGO Project. CJASN 16: 1247–1255, August, 2021*

- Estudios reportan más riesgo en protocolos sin esteroides vs mantenimiento (HR 4,17).
  - *Visger et al. Clin Transplant 2014: 28: 845–854*

- Estudios reportan menos riesgo en protocolos inducción timoglobulina.
  - Berthoux et al. Transplantation 2008;85: 1505–1507
- Mayor riesgo en TRDV, compatibilidad HLA
- Jóvenes, menor tiempo en diálisis.
- DSA pretrasplante y de novo > riesgo (HR 2,74; HR 6,65)

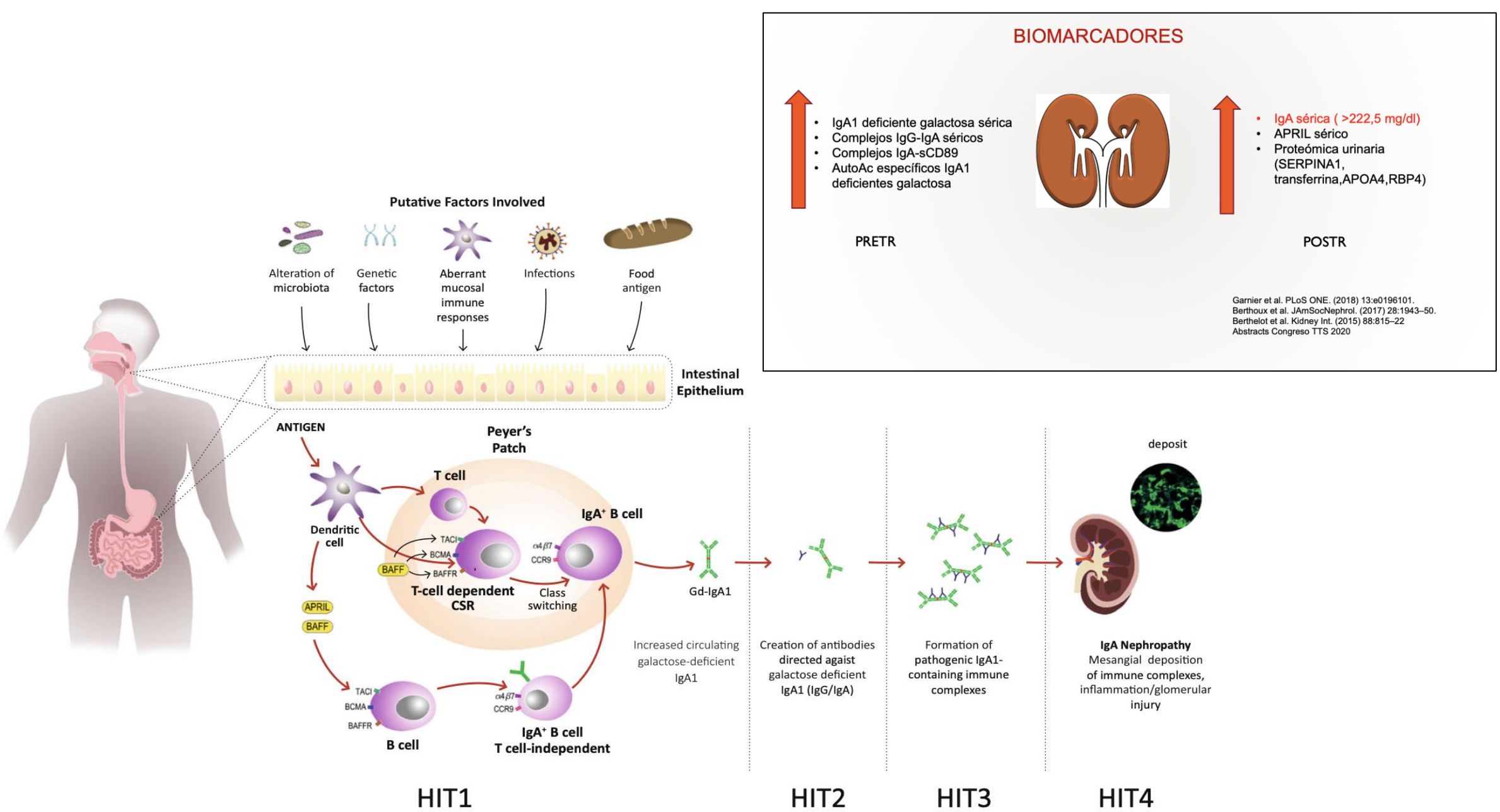
Uffing et al. TANGO Project. CJASN 16: 1247–1255, August, 2021

- Curso agresivo/semilunas en RN pueden predecir > riesgo
- Variantes genéticas en CFHR protein 5 (CFHR5) se han asociado a susceptibildad en RN (recidiva TR?)

Mousson et al. Transplantation Proceedings, 39, 2595–2596 (2007)

Tan et al. Sci Rep. 2021 Mar 9;11(1):5467.

Zhai et al. J Am Soc Nephrol. 2016;27(9):2894



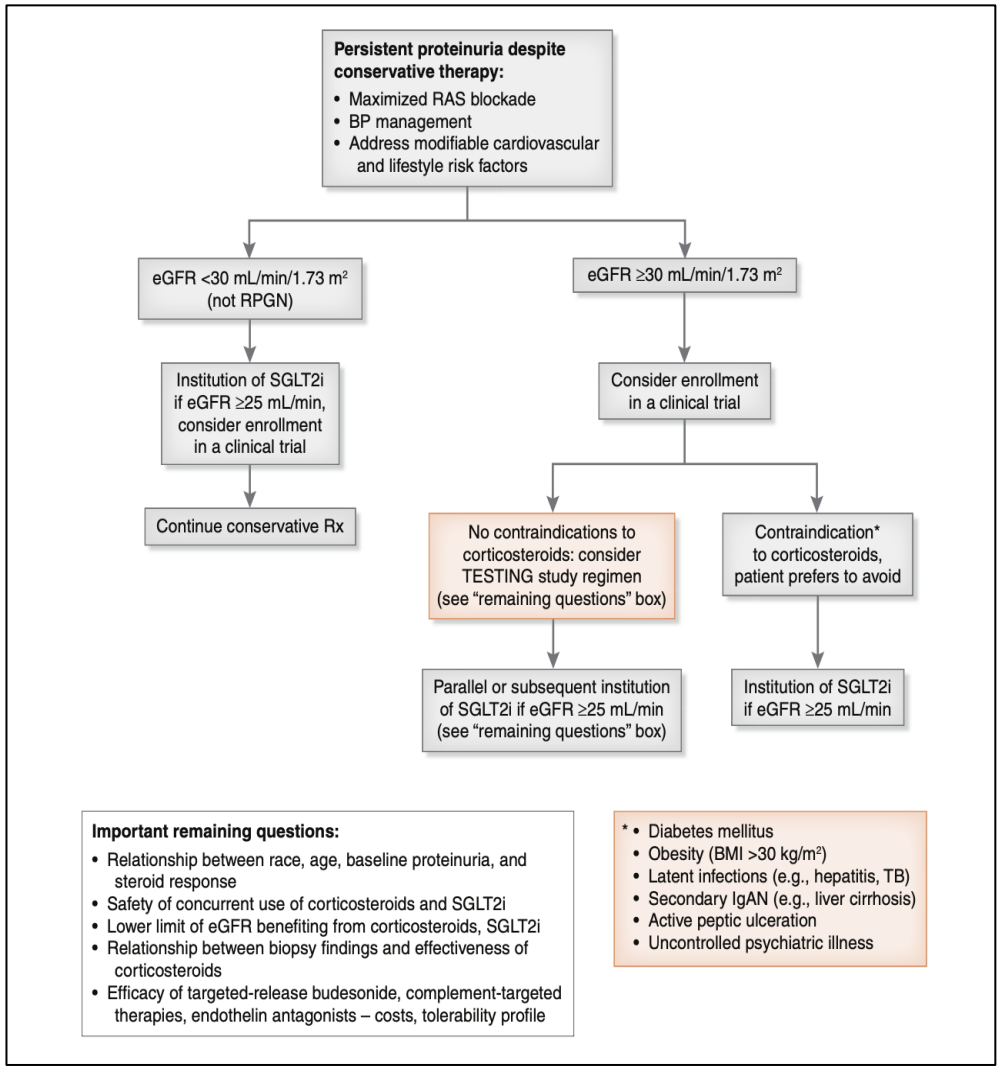
Gesualdo et al. Seminars in Immunopathology (2021) 43:657–668

Cómo trataría la recurrencia

Extrapolado de estudios de RN
Triple terapia TAC+MMF+PRD
Si iMTOR→convertir

	Treatment strategies	Level of evidence
IgA nephropathy	Antiproteinuric	♦
	Corticosteroids	◊
	Calcineurin-inhibitor[a]	◊
	Corticosteroid + calcineurin-inhibitor	◊
	Antimetabolite	◊
	Alkylating agent (for crescentic form)	◊ ◊ ♦
	Tonsillectomy	◊ ♦

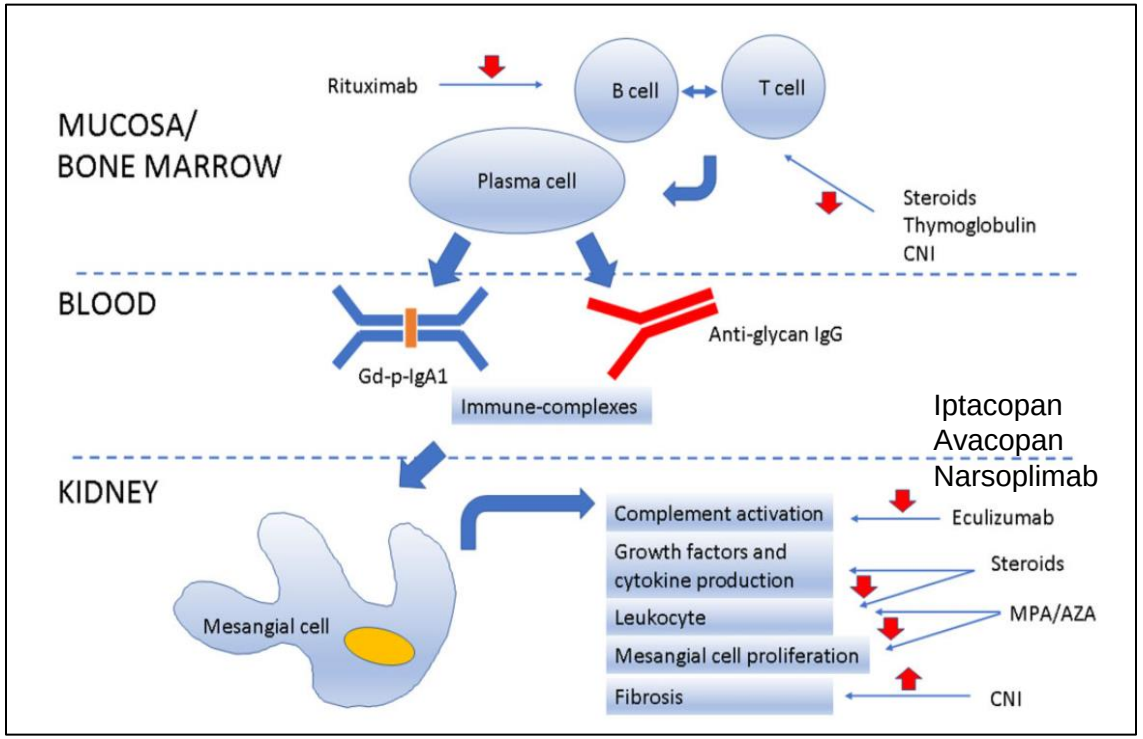
Class I evidence ♦ – treatment(s) is recommended based on current evidence/consensus; Class II evidence ◊ – treatment(s) should be considered based on current evidence/consensus; Class III evidence ◊ ♦ – treatment(s) is not recommended based on current evidence/consensus. [a]Continue with or restart/initiate calcineurin-inhibitor (either cyclosporine or tacrolimus). GN, glomerulonephritis.



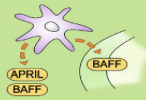
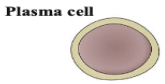
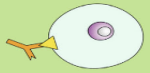
Lim et als. Frontiers in Inmunology. 2019 Vol 10 Art 1944
Reich et al. CJASN 17: 1243–1246, 2022

CORTICOIDES EN NEFROPATÍA IGA

Estudio	Medicación	Dosis inicial	Desescalada	Exposición total
TESTING 2022	Metilprednisolona	0,4 mg/kg/d(máx 32 mg/d) 2 m	Reducción 4 mg/mes 4 meses	6 m
TESTING 2017	Metilprednisolona	0.6-0.8 mg/kg/d(máx 48 mg/d) 2 m	Reducción 8 mg/mes 4 meses	6 m
Manno et al	Prednisona	1 mg/kg/día 2 m	Reducción 0,2 mg/kg cada mes 4 m	6 m
Lv et al	Prednisona	0,8-1 mg/kg/d 2 m	Reducir 5-10 mg/14 días 4 m	6 m
Pozzi et al Stop-IgA	Metilprednisolona iv +prednisona oral	Metilprednisolona 1 gr iv 3 días mes 1,3,5 y prednisona oral 0,5 mg/kg/48 h 6 m	no	6 m
NEFIGARD	TRF- Budesonida	16 mg/d 9 m	Reducir dosis 8 mg/semana 2 semanas, suspender	9m



Soporte: **sparsentan(PROTECT TRIAL)**, espironolactona, finerenona

New prospective therapy targeting the gut-mucosal immune system		
	Target	**Prospective therapy**
	Mucosal hyperresponsiveness	Gluten free diet
	Microbiota	Antibiotics/Prebiotics
	Microbiota	Fecal microbiota transplantation
 Enteric budesonide	B-cell activation and proliferation; intestinal and local inflammation	Enteric budesonide
 HCQ	Toll-like receptors and cytokine production	Hydroxychloroquine
 Atacicept Blisibimod VIS649	BAFF/APRIL inhibitor	Atacicept Blisibimod VIS649
 Bortezomib	Plasma cell proteasome inhibitor	Bortezomib
 Rituximab Obinutuzumab	B-cell depletion therapy	Rituximab Obinutuzumab

Gesualdo et al. Seminars in Immunopathology (2021) 43:657–668
Infante et al. Clinical Kidney Journal, 2020, vol. 13, no. 5, 758–767

ORGANIZA:

SGAN
sociedad gallega de nefrología

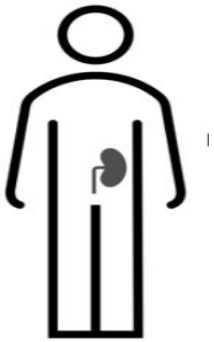
GEFS

No AF de Enfermedad renal

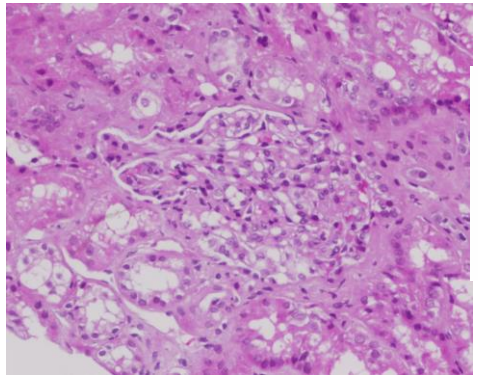
2001: Crp 2 mg/dl Prot 11 g/d→ GEFS

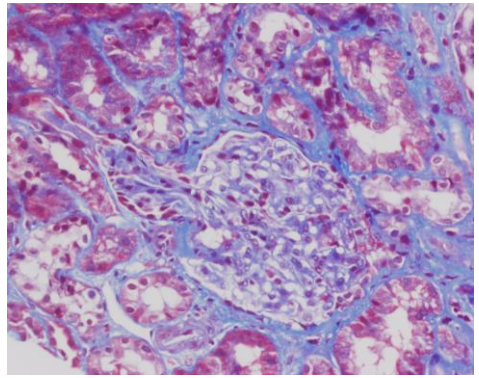
HD 2003

TRDF 2007 Retraso función injerto. 1er mes Crp 3 mg/dl , Prot 5 g/d



38 a





PF (10 sesiones)

RTX (2 d)

Crp 3 mg/dl Prot 5 g/d

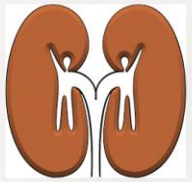
Reinicio HD 2012

Éxitus 2013 por HSA

BIOMARCADORES

- suPAR sérico
- AutoAc CD40
- Ac antiAT1R

PRETR



Aumentan en recurrencia:
Apolipoproteína A1-b urinaria,
alfa 1 antitripsina, CLC-1

- suPAR urinario

POSTR

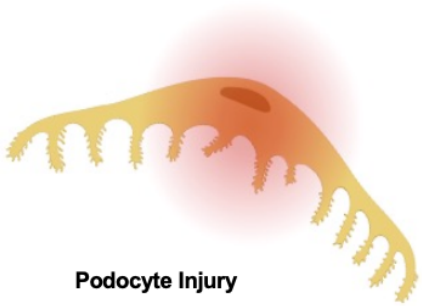
Lim et al. Frontiers in Inmunology. 2019 Vol 10 Art 1944

Riesgo de recurrencia bajo

Genetic FSGS
High penetrance/low frequency
(e.g., nephrin mutation, *COL4A3/A4/A5*)
Low penetrance/high frequency
(e.g., *APOL1* high-risk variants)

Medication-associated FSGS
Direct podocyte toxicity
(e.g., pamidronate, lithium, mTOR inhibitors)

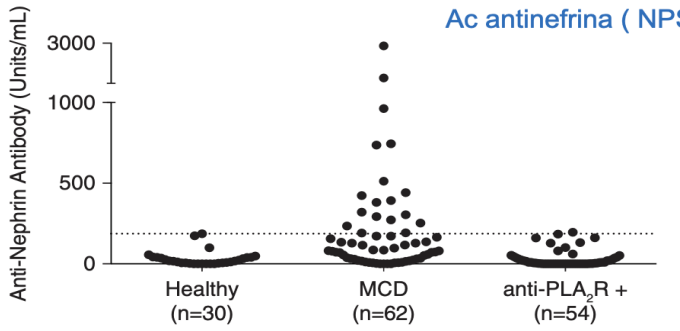
Adaptive FSGS/Hyperfiltration
Overload/stress injury
Risk factors:
- Hypertension
- Small kidney mass
- Obesity
- Recurrent AKI

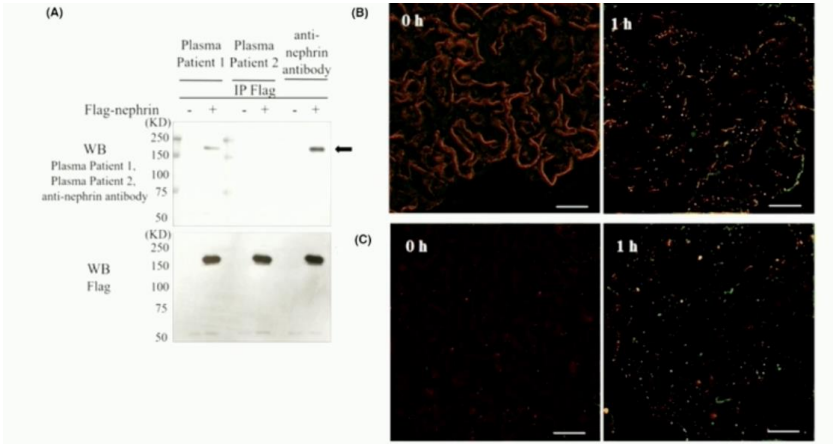


Podocyte Injury

A

Ac antinefrina (NPSH1)





Halleke et al.August 2022 | ASN Kidney News | 25
Watts et al. JASN 33: 238–252, 2022
Hattori et al. AJT.Vol22, Issue 10

RECURRENCIA GEFS

- Precoz (media 1,5 meses)
- Tasa recurrencia 30 %
- Pérdida injerto hasta un 30-50 % a 5 a
- Factores de riesgo: variables en distintos estudios (*TRDV, no asociación con histología, jóvenes, enfermedad agresiva en RN (sin sd. nefrótico poco riesgo*), *recurrencia previa 80%*

11742 TR
176 GEFS
idiopática

- Raza blanca
- > Edad al dx RN
- Bajo IMC
- Nefrectomía RN pretrasplante
- Recurrencia en injerto previo → 45 %; 100 % 2º injerto

Uffing et al. CJASN 15: 247–256, 2020
Uffing et al. CJASN 16: 1730–1742, 2021

Cómo trataría la recurrencia

Empírico
No ensayos clínicos
La > parte de las series régimen de plasmaféresis + RTX

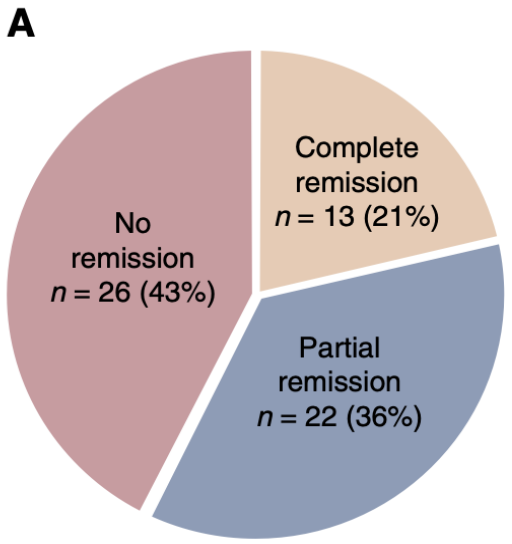
Refractariedad?: LDL aféresis, adalimumab, abatacept, galactosa oral, ACTH (case reports)

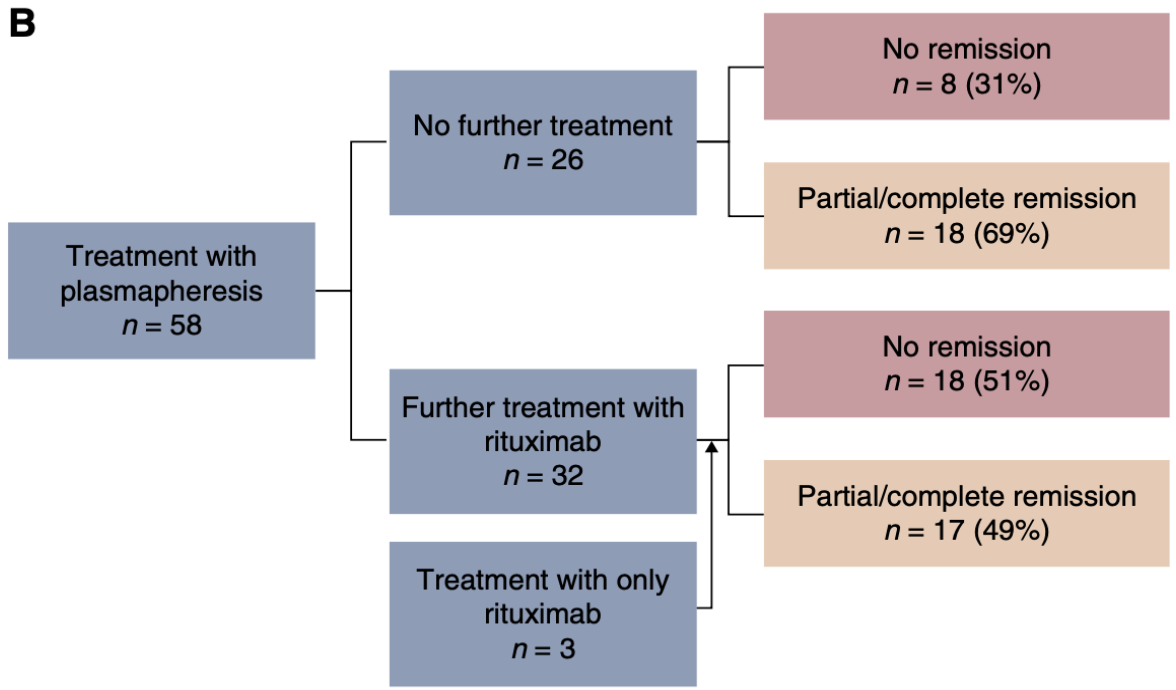
Treatment	Study	Population	Design	Total (*n*)	Dosage	Response Rate Complete Remission+ Partia Remission [a]/No Remission (%)	Comments
Plasmapheresis	Ponticelli *et al.* 2010 (82)	Children and adults	Review of case series and case reports	144	Variable	98 out of 144 (68%)	Review of case reports, therefore publication bias
	Gonzalez *et al.* 2011 (78)	Children	Retrospective, single center	17	Unknown	15 out of 17 (88%)	Treatment of recurrent FSGS not described in methods
	Schachter *et al.* 2010 (83)	Children and adults	Retrospective, single center	12	PP: 4–48 sessions	8 out of 12 (75%)	
	Mansur *et al.* 2019 (84)	Children and adults	Retrospective, single center	61	PP: median 20 sessions	22 out of 61 (36%)	Patients also received high dose steroids (70%) Some patients also received RTX (16%)
	Francis *et al.* 2018 (85)	Children	Retrospective, multicenter	20	PP: 10–92 sessions	15 out of 20 (75%)	Many other treatments used: iv CsA, CP, RTX, high dose steroids, ABT, galactose
Plasmapheresis + rituximab	Alasfar *et al.* 2018 (80)	Adults	Prospective single center	40	PP: >10 sessions RTX: 1–2 doses (375 mg/m^2)	35 out of 40 (87%)	Not all participants received RTX (50%) No definition of recurrent FSGS
	Uffing *et al.* 2020 (5)	Adults	Retrospective, multicenter	61	Variable	35 out of 61 (57%)	Large differences between treatment regimen between patients Not all patients received RTX (57%)
	Garrouste *et al.* 2017 (86)	Adults	Retrospective, multicenter	19	PP: unknown RTX: 1–4 doses (375 mg/m^2)	12 out of 19 (63%)	Some patients also received iv CsA (26%)
	Alachkar *et al.* 2013 (87)	Adults	Retrospective, single center	24	PP: median 15 sessions RTX: 1–2 doses (375 mg/m^2)	19 out of 24 (79%)	Not all patients received RTX (54%)
	Staeck *et al.* 2015 (88)	Adults	Retrospective, single center	12	PP: median 11 sessions RTX: unknown	11 out of 12 (92%)	Not all patients received RTX (50%) Other treatments used: iv CsA, high dose steroids
Immunoadsorption	Allard *et al.* 2018 (25)	Children	Retrospective, multicenter	12	IA: median 129 sessions	10 out of 12 (83%)	Many other treatments used: PP, iv CsA, RTX, ABT, BTZ, CP, saquinavir, galactose
Plasmapheresis + iv cyclosporine	Canaud *et al.* 2010 (89)	Children and adults	Prospective, single center	10	PP: 25–39 sessions CsA iv: 14 days (target level 200–400)	10 out of 10 (100%)	All patients also received high dose oral steroids
Oral cyclosporine	Shishido *et al.* 2013 (90)	Children	Prospective, single center	10	CsA oral: target level 4500–5500 ng^{a}h/ml	9 out of 10 (90%)	All patients also received high dose iv steroids
ACTH gel	Grafals *et al.* 2019 (91)	Adults	Retrospective, two centers	14	ACTH: 80 units twice a week	5 out of 14 (36%)	Many other treatments used: PP, high-dose steroids, ABT, Bela, RTX ACTH used as "last resort." In patients without PP, ACTH did not result in response Study sponsored by pharmaceutical company
	Alhamad *et al.* 2019 (92)	Adults	Retrospective, two centers	20	ACTH: 40–80 units twice a week	10 out of 20 (50%)	ACTH used as "last resort" if PP and RTX did not work. Divergent definition of CR and PR Researcher funded by pharmaceutical company

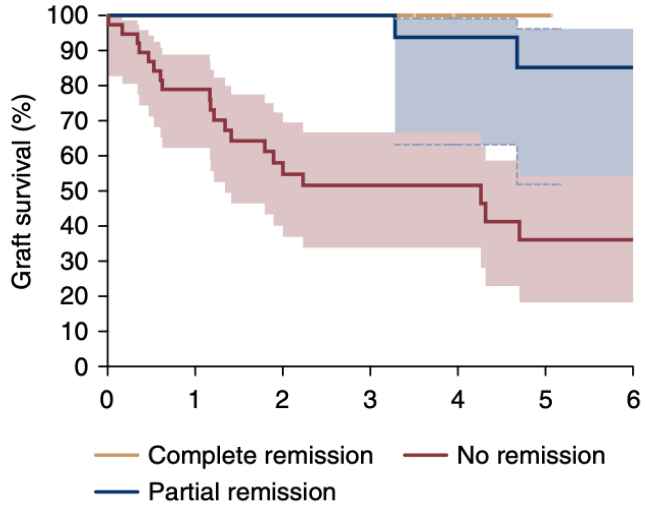
Uffing et al. CJASN 16: 1730–1742, 2021

THE TANGO STUDY
Post-Transplant Glomerular Diseases

176 pac GEFS idiopática
56 pac recurren (32 %)







Uffing et al. CJASN 16: 1730–1742, 2021

Cómo prevenir la recurrencia

Treatment	Study	Population	Design	Total (*n*)	Genetic Testing	Dosage	Recurrence Rate Per Group Recurrence/No Remission (%)	Comments
Plasmapheresis	Ohta *et al.* 2001 (75)	Children	Retrospective PPP versus none	21	No info	2–3 sessions PP pre-transplant	PPP: 5 out of 15 (33%) None: 4 out of 6 (67%)	No information on exclusion of genetic FSGS Multiple allografts per patient
	Gohh *et al.* 2005 (76)	Adults + 1 child	Retrospective Single group (PPP)	10	No info	8 sessions PP peri-Tx	PPP: 3 out of 10 (30%)	No control group Large differences in time point PP was started
	Hickson *et al.* 2009 (77)	Adults + children	Retrospective PPP versus none	30	Not performed Familial FSGS excluded	1 or more sessions PP pre-Tx	PPP: 6 out of 7 (86%) None: 7 out of 23 (30%)	Study was designed to define patients with high-risk for FSGS recurrence, not to assess effects of PPP
	Gonzalez *et al.* 2011 (78)	Children	Retrospective PPP versus none	34	NPHS2 tested in 10 patients	1–10 sessions PP pre-Tx	PPP: 9 out of 17 (53%) None: 10/17 (59%)	1 patient had a heterozygous NPHS2 mutation
	Verghese *et al.* 2018 (79)	Children	Retrospective PPP versus none (historical cohort)	51	NPHS2 tested in PPP group	1–3 sessions PP pre-Tx, 5 sessions post-Tx	PPP: 7 out of 26 (27%) None: 8 out of 31 (26%)	Historical control group, significant differences between groups No genetic testing in control group
Plasmapheresis + rituximab	Alasfar *et al.* 2018 (80)	Adults	Prospective PPP+PRTX and PRTX versus none	66	Genetic FSGS excluded, no info on number of patients tested	3–10 sessions PP peri-Tx 1–2 doses RTX	PRTX and PPP+PRTX: 23 out of 37 (62%) None: 14 out of 27 (52%)	Prophylactic treatment on the basis of high/low risk No differentiation between PRTX only and combined PRTX+PPP
Rituximab	Fornoni *et al.* 2011 (23)	Children	Retrospective PRTX versus none (historical cohort)	41	No info	1 dose RTX (375 mg/m^2)	PRTX: 8 out of 27 (30%)[a] None: 10 out of 14 (71%)	Study was designed to investigate mechanisms of rituximab, therefore limited clinical data and significant differences between groups
LDL-apheresis + rituximab	Sannomiya *et al.* 2018 (81)	Adults	Retrospective Single group (RTX + LDL-apheresis)	5	No info	1 dose RTX (100 mg) and 2 sessions LPL-apheresis pre-Tx	PRTX+LDL: 0 out of 5 (0%)	No control group Exclusion of secondary FSGS not mentioned

PPP, prophylactic plasmapheresis; PP, plasmapheresis; Tx, transplant; PRTX, prophylactic rituximab.
[a]No clear definition of FSGS recurrence; numbers are on the basis of treatment with plasmapheresis within 1 month after transplant.

Ningún estudio ha demostrado eficacia PF/RTX profiláctica (pequeño tamaño, retrospectivos, no randomizados)
Monitorización proteinuria precozmente posTR

ONGOING TRIALS
- PRI-VENT FSGS (PF+RTX)
- BLESELUMAB (ANTICD40)
- ACTH

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Nefropatía membranosa

Sd. Nefrótico 2º a GNM (27 a). PLA2 R desconocido → Tto tacrolimus → ERC progresiva →DP 2a → TRDF 2014 PLA2 R neg → Injerto inicialmente funcionante (Crp1,4 mg/dl)

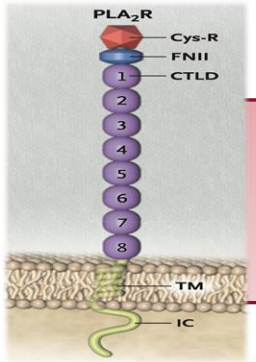
2 a: Crp 2,17 mg Prot 1,8 gr/24 h. DSA neg → BR: Recidiva GNM PLA2 R, THSD7A neg → Tto soporte→ Crp 1,8 mg/dl Prot 0,4 g/24 h → 2018: Crp 6,5 mg/dl→ RAH+ Recidiva GNM (PLA2 R positivos).FIAT intensa. Inicia HD

Actualmente en HD, en LE re TR

38 a

Prevalencia: 10-50 %
Pérdida injerto: 10-50%

RECURRENCIA GNM



PRETRASPLANTE | TRASPLANTE | POSTRASPLANTE

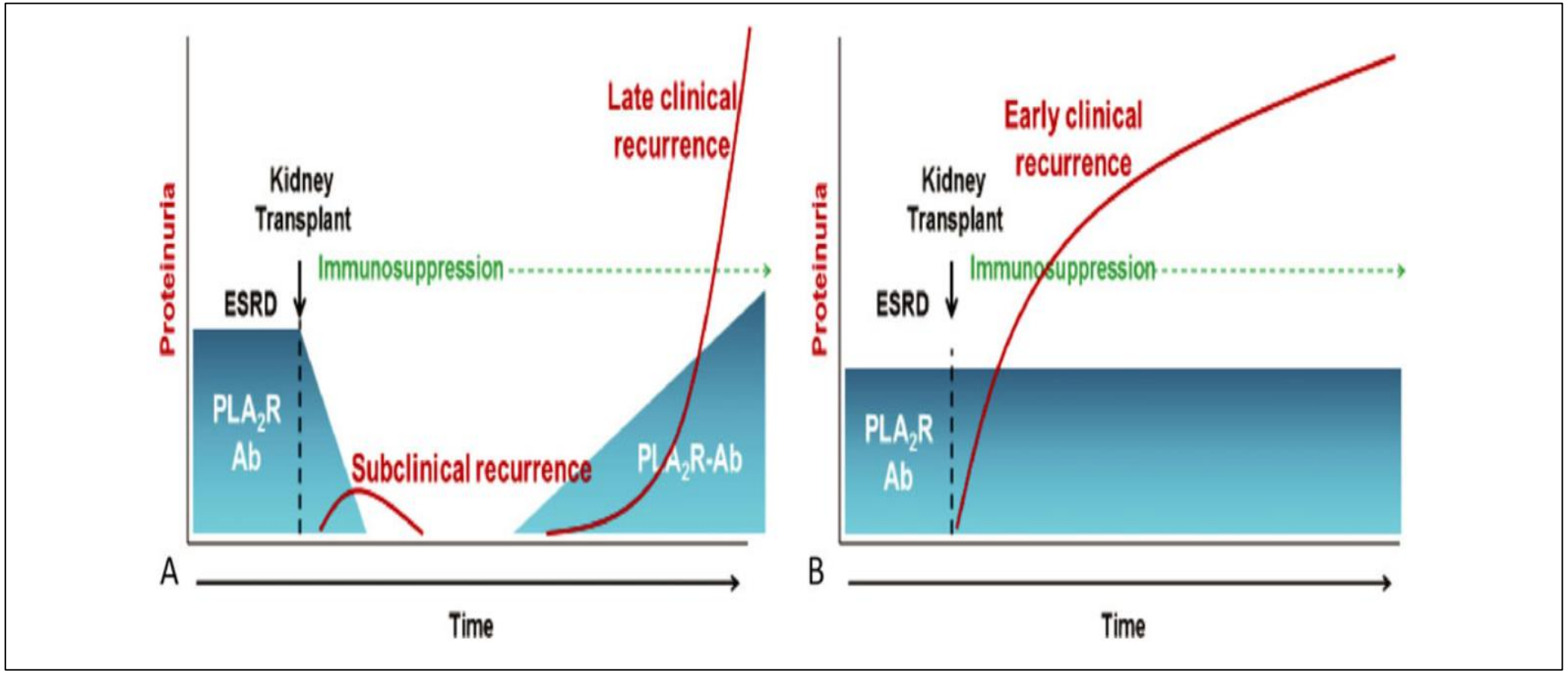
TÍTULO AC ANTIPLA2 R
IS con anticalcineurínicos

RIESGO DE RECURRENCIA ELEVADO

- AC ANTIPLA2 R ELEVADOS (>29 RU/ml, 45 RU/ml)
- CURSO AGRESIVO RN
- RECURRENCIA PREVIA
- Polimorfismos riesgo recidiva locus HLA-D y PLA2R1 : RES9271188 (intergenic between HLA-DRB1 AND HLA-DQA2) at the HLA-D locus along with rs6726925 and rs13018963 at the PLA2R1 locus)

- MONITORIZACIÓN AC PLA2 R(3-6 M)
- PROTEINURIA
- BIOPSIA SI POSITIVIZACIÓN PLA2R?

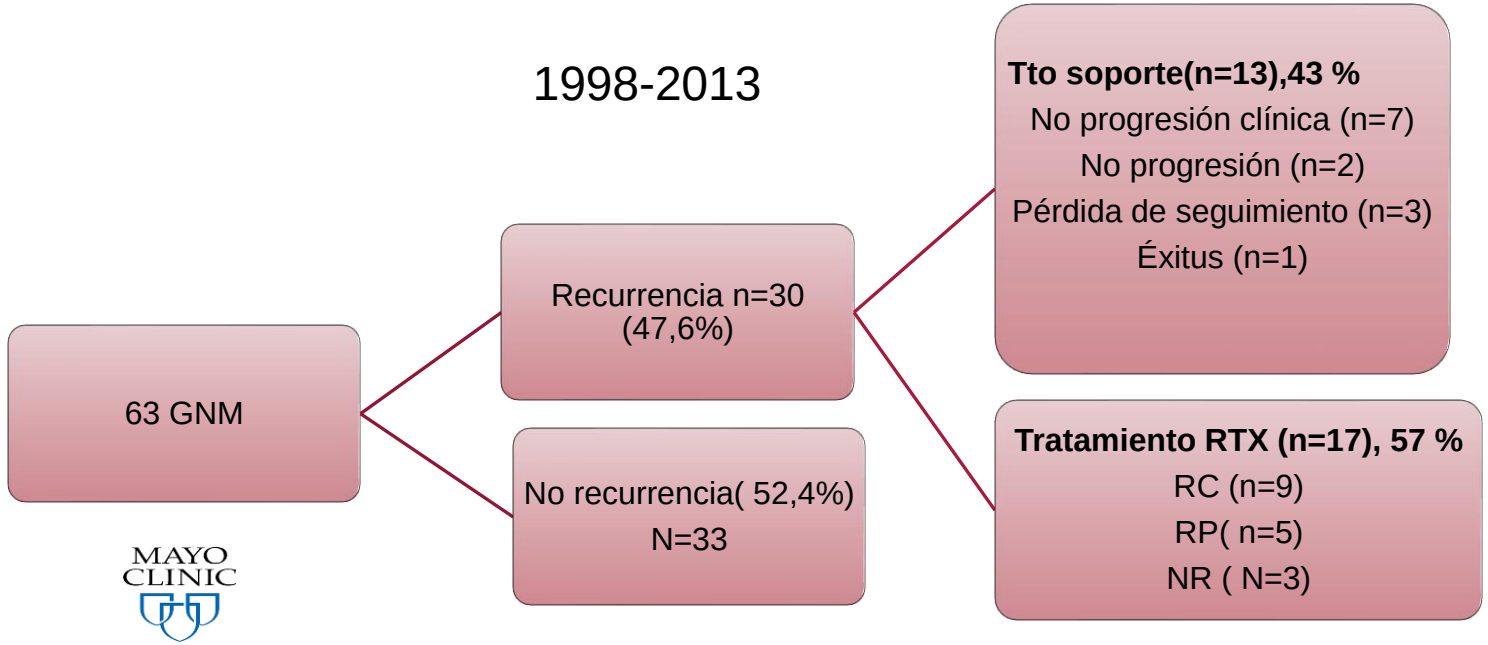
Berchtold et al. Kidney International (2021) 99, 671–685
Uffing et al. CJASN 16: 1730–1742, 2021
Gupta et al. Clin Transplant 2016: 30: 461–469
Quintana et al. Transplantation 2015; 99:1709-1714



Gupta et al. Clin Transplant 2016: 30: 461–469

CÓMO TRATARÍA LA RECURRENCIA

- Series de casos, estudios retrospectivos unicéntricos
- Antiproteinúricos
- Deterioro fx renal, síndrome nefrótico, evento trombótico o > 1 gramo tras medidas antiproteinúricas ?-->IS
- Rituximab de elección (*dosis MENTOR*)
- Refractariedad: bortezomib, obinutuzumab, ofatumumab (case reports)
- No tratamiento preventivo (salvo si recidiva en TR previo con PLA2 R elevados)



82 % éxito terapéutico
40 % resolución histológica

Grupper et al. Transplantation 2016;100: 2710–2716

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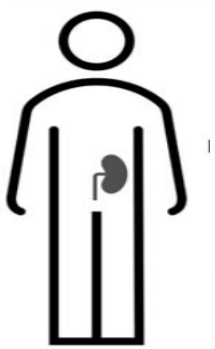
GNMP/GNC3



61a

GNMP tipo I
DP 2 a
TRDF 2004

Recidiva 1 año posTR
(revisada C3)
Tto soporte+esteroides
Pérdida injerto 2008

TRDF 2015

Injerto funcionante Crp
1,5 Prot 0,2

2017: Crp 1,9, prot 1,7
gr/día

BR:GNMP
IF C3+++

Estudio genético c' sin
alteraciones
Elevación c5b9

Eculizumab 4 años

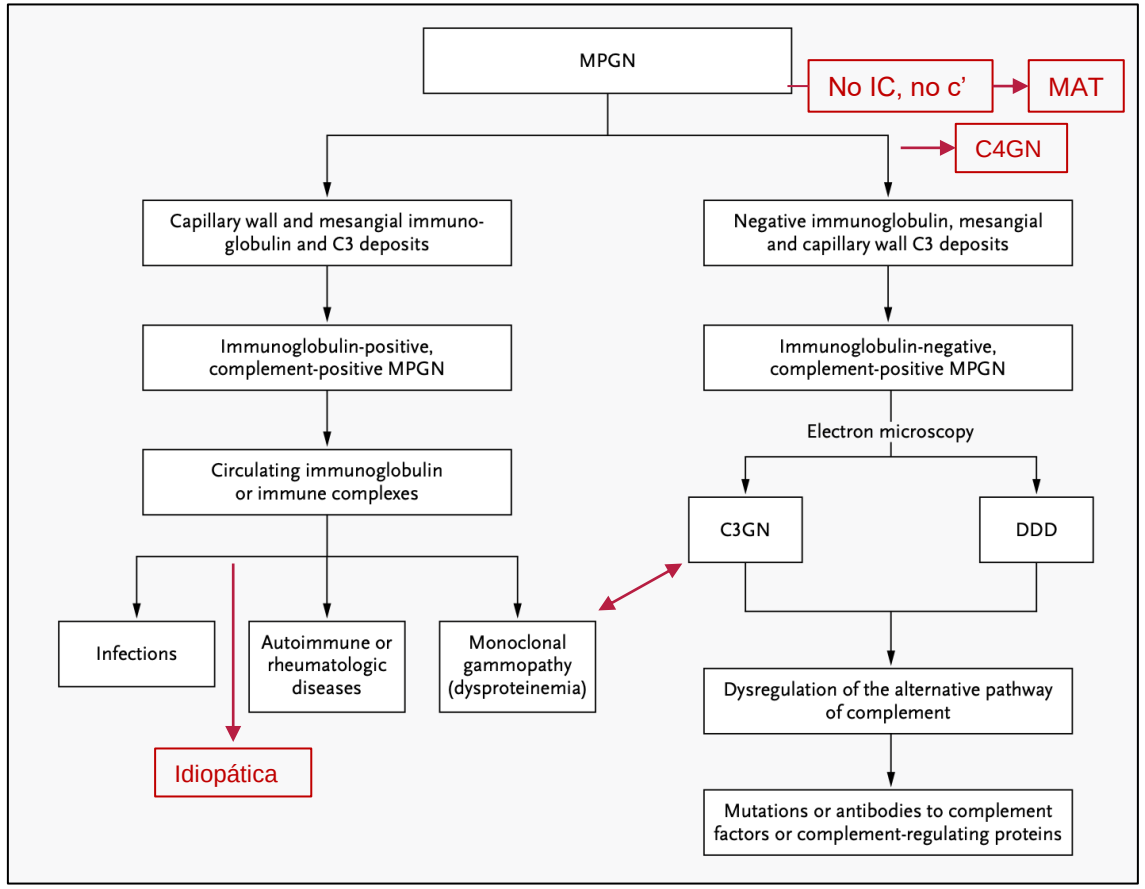
Pérdida injerto 2021
(disfunción crónica+
agudización tras cx
cardíaca)

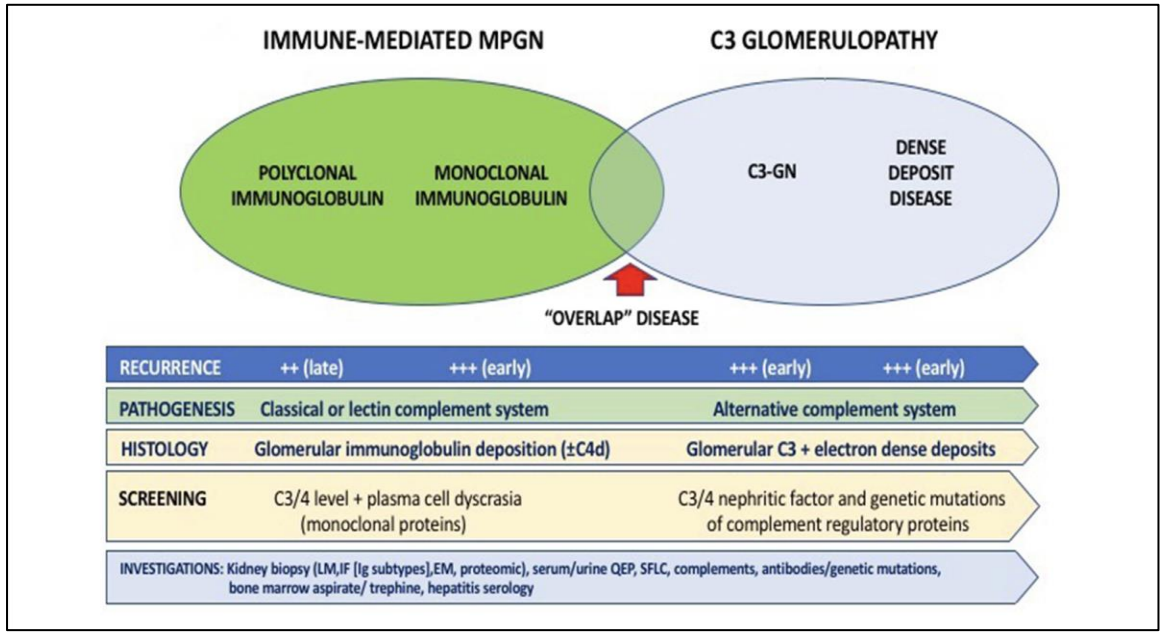
HD en la actualidad

Membranoproliferative Glomerulonephritis — A New Look at an Old Entity

Sanjeev Sethi, M.D., Ph.D., and Fernando C. Fervenza, M.D., Ph.D.

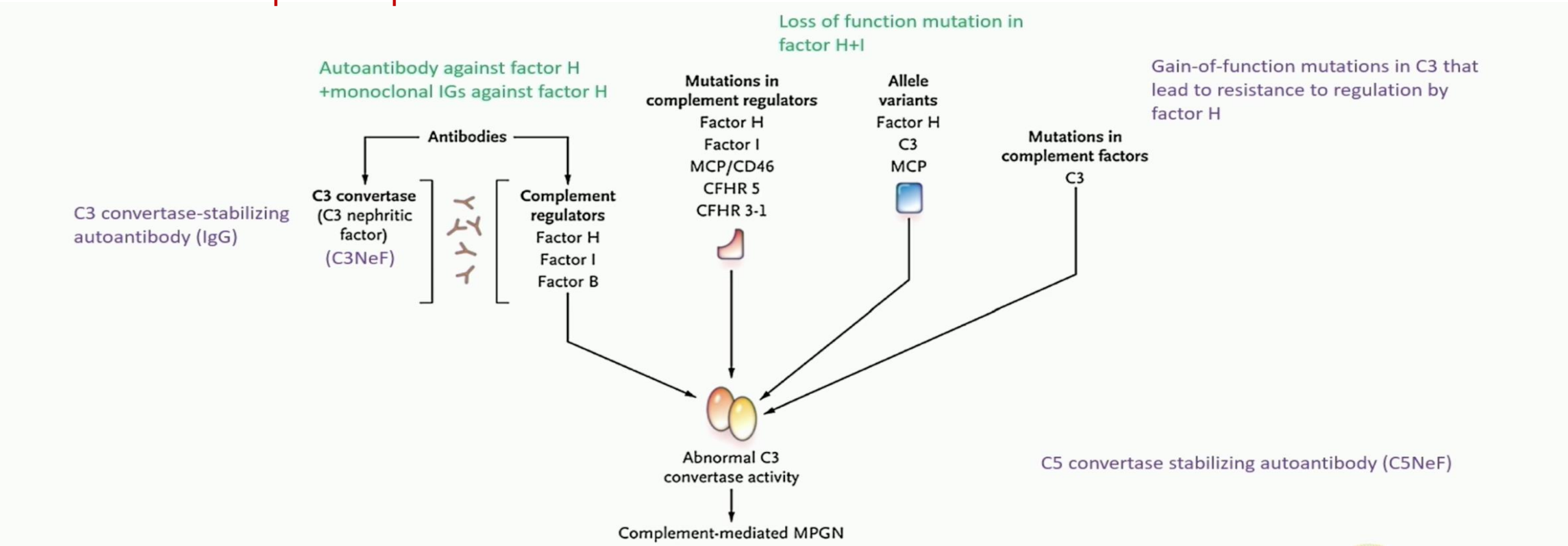
N ENGL J MED 366;12 NEJM.ORG MARCH 22, 2012





Lim et al. Frontiers in Inmunology. 2019 Vol 10 Art 1944

GNMP mediadas por complemento



Barbour et al.. Am J Transplant. 2015 Feb; 15(2):312-9.

<u>Fatores genéticos</u>:
25% portan variantes raras en los genes relacionado con el complemento
- Genes convertasa **C3**: Codifica factor 3 del complemento
- Genes **CFB**: Codifica el factor B del complemento
- Genes **CFH Y CPI**: Codifica factores de H e I
- Genes **CFHR5**: Codifica proteína 5 relacionada con el factor H

<u>Factores adquiridos</u>:
Autoanticuerpos: **Factores nefríticos C3**. + freq contra C3bBb. 80% DDD, 50%C3GN.
Factores nefríticos C5. + C3GN
Factores nefríticos C4, Ac-Factor H, AC-Factor B: 10%

GRUPO DE TRABAJO
Sociedad Española de Nefrología
SENTRA
SENTRA
Grupo de Trasplante Renal

GLOMEN

Recurrence of immune complex and complement-mediated membranoproliferative glomerulonephritis in kidney transplantation

Fernando Caravaca-Fontán[1,2], Natalia Polanco[3], Blanca Villacorta[4], Anna Buxeda[5], Armando Coca[6], Ana Ávila[7], Rocío Martínez-Gallardo[8], Cristina Galeano[9], Rosalía Valero[10], Natalia Ramos[11], Natalia Allende[12], Leónidas Cruzado-Vega[13], María José Pérez-Sáez[5], Ángel Sevillano[3], Esther González[3], Ana Hernández[3], Emilio Rodrigo[10], Mario Fernández-Ruiz[1,14], José María Aguado[1,14], Miguel Ángel Pérez Valdivia[4], Julio Pascuall[5], Amado Andrés[1,2,3] and Manuel Praga[1,2,3], the Spanish Group for the Study of Glomerular Diseases and the Spanish Group of Kidney Transplant

[1]Instituto de Investigación Hospital 12 de Octubre (imas12), Madrid, Spain, [2]Department of Medicine, Universidad Complutense de Madrid, Madrid, Spain, [3]Department of Nephrology, Hospital Universitario 12 de Octubre, Madrid, Spain, [4]Department of Nephrology, Hospital Universitario Virgen del Rocío, Sevilla, Spain, [5]Department of Nephrology, Hospital del Mar, Institut Mar for Medical Research, Barcelona, Spain, [6]Department of Nephrology, Hospital Clínico Universitario de Valladolid, Valladolid, Spain, [7]Department of Nephrology, Hospital Universitario Doctor Peset, Valencia, Spain, [8]Department of Nephrology, Hospital Universitario de Badajoz, Badajoz, Spain, [9]Department of Nephrology, Hospital Universitario Ramón y Cajal, Madrid, Spain, [10]Department of Nephrology, Hospital Universitario Marqués de Valdecilla, Santander, Spain, [11]Department of Nephrology, Hospital Universitario Vall d'Hebron, Barcelona, Spain, [12]Department of Nephrology, Hospital Universitario Son Espases, Palma de Mallorca, Spain, [13]Department of Nephrology, Hospital General Universitario de Elche, Elche, Spain and [14]Unit of Infectious Diseases, Hospital Universitario 12 de Octubre, Madrid, Spain

- Proteinuria, hematuria, HTA, deterioro FG
- C3 bajo en mediadas por c', suele preceder manifestación clínica
- Puede parecerse a glomerulopatía del trasplante (dobles contornos)

33/54 que recurren (61%) fallo injerto

Recurrence of immune complex and complement-mediated membranoproliferative glomerulonephritis in kidney transplantation

Background

Membranoproliferative glomerulonephritis (MPGN) represents a histologic pattern of glomerular injury which may be due to several etiologies.

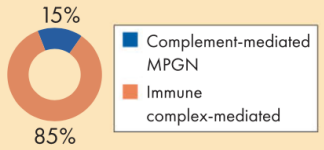
Few studies have analyzed post-transplant recurrence of MPGN according to current classification.

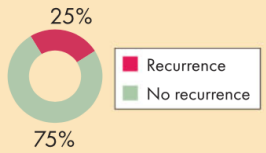
Methods

Retrospective cohort
- 11 hospitals
- 1981–2021
- Median follow-up 79 months

220 kidney graft recipients
- Biopsy-proven native disease due to MPGN
- Assessed recurrence

Results





37% Kidney failure — The main predictors were development of **rejection** and **recurrence**

25% Disease recurrence — Over a median of **16 months** after kidney transplantation

Higher recurrence — Dysproteinemia (67%) Complement-mediated (62%)

Remission determinants — **Early time to recurrence:** eGFR < 30 ml/min/1.73 m² Serum albumin < 3.5 g/dl

Conclusion

One-fourth of patients developed recurrence in the allograft, especially in cases with complement-mediated disease or in those associated with dysproteinemia.

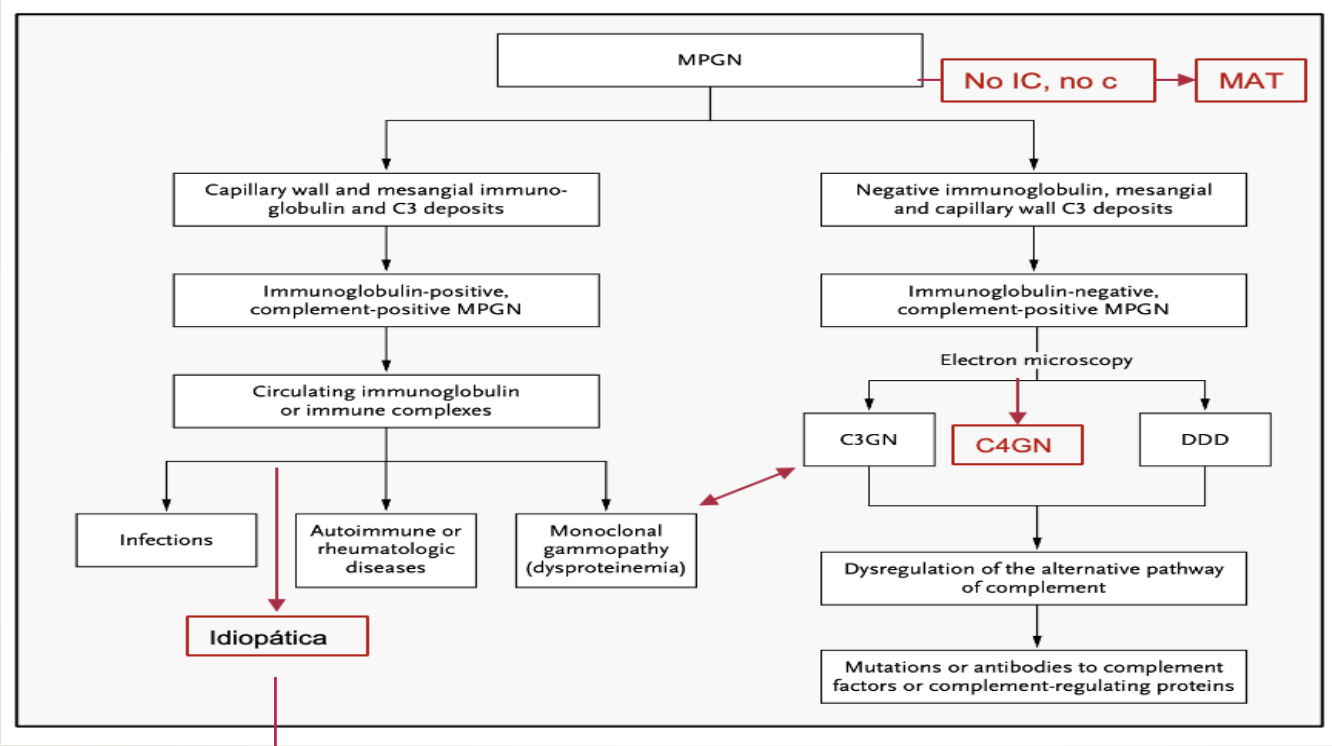
NEPHROLOGY DIALYSIS TRANSPLANTATION

Caravaca-Fontán F., et al. NDT (2022)
@NDTSocial

Caravaca-Fontán et al. NDT (2023) 38; 222-235

COMO TRATARÍA LA RECURRENCIA

TTO SOPORTE



TTO ETIOLOGICO

Moderada: aumento prednisona 1mg/kg. Aumento MMF
Severa: ?? RTX/PF/MP/CF??

Treatment of C3 Glomerulopathy in Adult Kidney Transplant Recipients: A Systematic Review

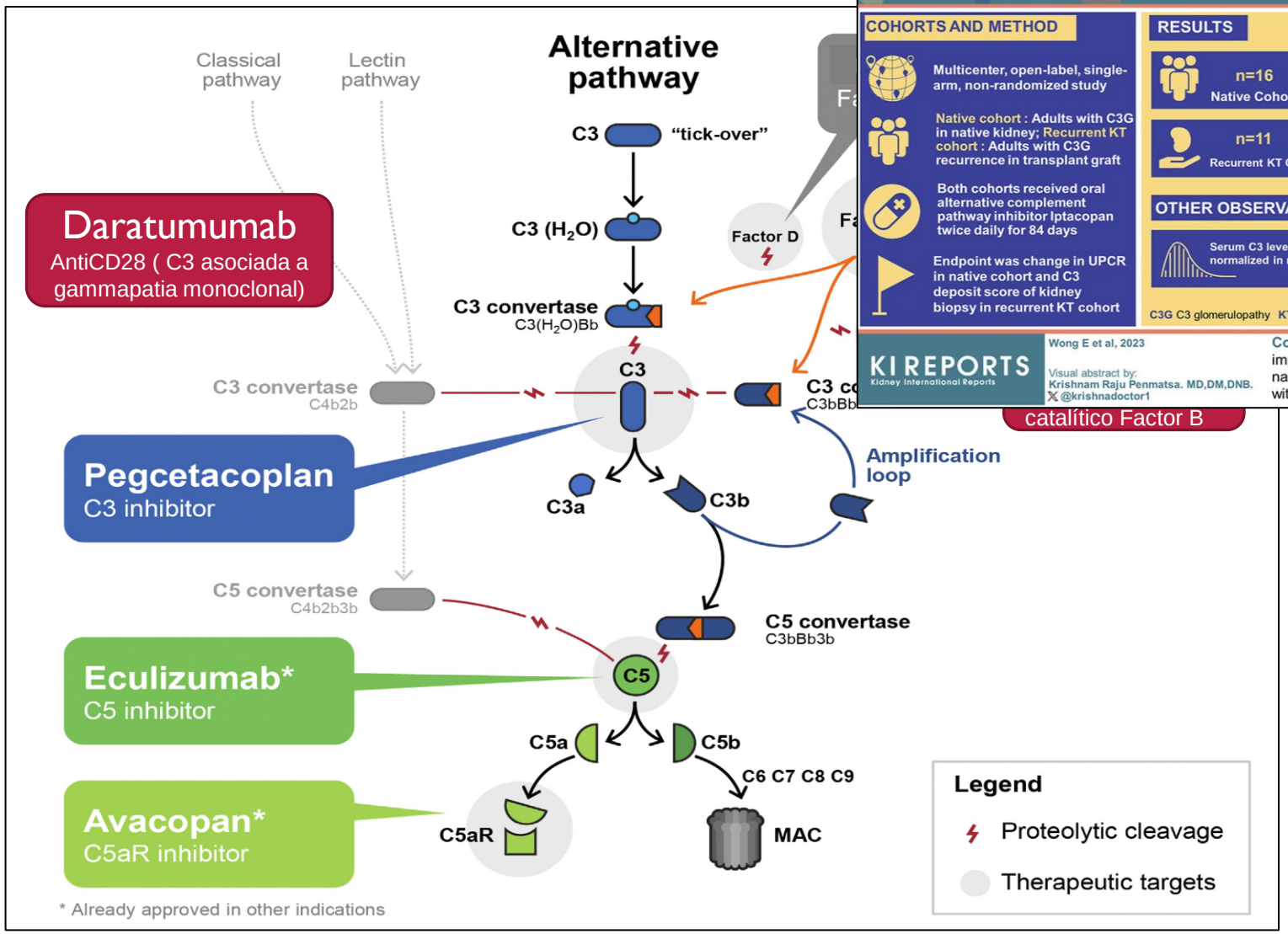
Maria L Gonzalez Suarez [1,2,*], Charat Thongprayoon [2,*], Panupong Hansrivijit [3], Karthik Kovvuru [4], Swetha R Kanduri [4], Narothama R Aeddula [5], Aleksandra I Pivovarova [1], Api Chewcharat [6], Tarun Bathini [7], Michael A Mao [8], Arpita Basu [9] and Wisit Cheungpasitporn [1,2,*]

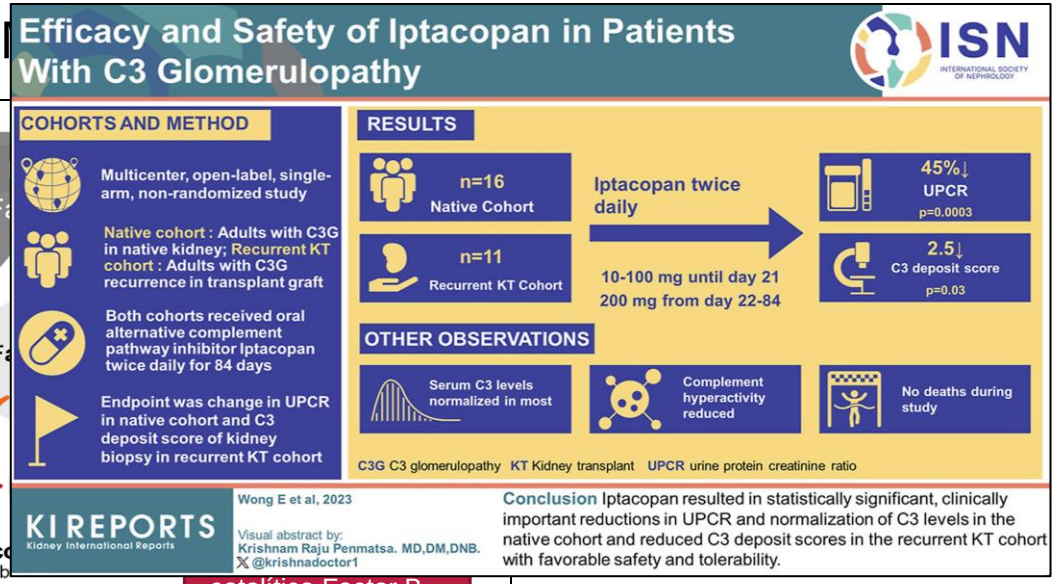
González-Suarez et al. Med Sci, 2020,8,44

	Pérdida injerto C3	Pérdida injerto DDD
Eculizumab	33%	22%
Plasmaféresis	42%	56%
Rituximab	81%	70%
No tto	32%	53%

Niveles C5b-9 predictor de respuesta a tratamiento

Fármacos en desarrollo GN





Bomback et al.Kidney Int Rep (2022) 7, 2150–2159

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CONCLUSIONES

- Recidiva GN posTR es la 3ª causa de pérdida de injerto censurada por muerte
- Evidencia muy baja
- Distintas entidades
  Infradiagnosticadas
- Información epidemiológica, clínica y terapéutica muy variable
- Necesario generar estudios de calidad

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MUCHAS GRACIAS

Andrés López Muñiz
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Servizo de Nefroloxía

@NefroCHUAC

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A Coruña