Συμπεράσματα από τη θεραπευτική στρατηγική των τελευταίων ετών στον ΣΕΛ. Νέες τάσεις, τι ακολουθεί.

Γεώργιος Μπερτσιάς

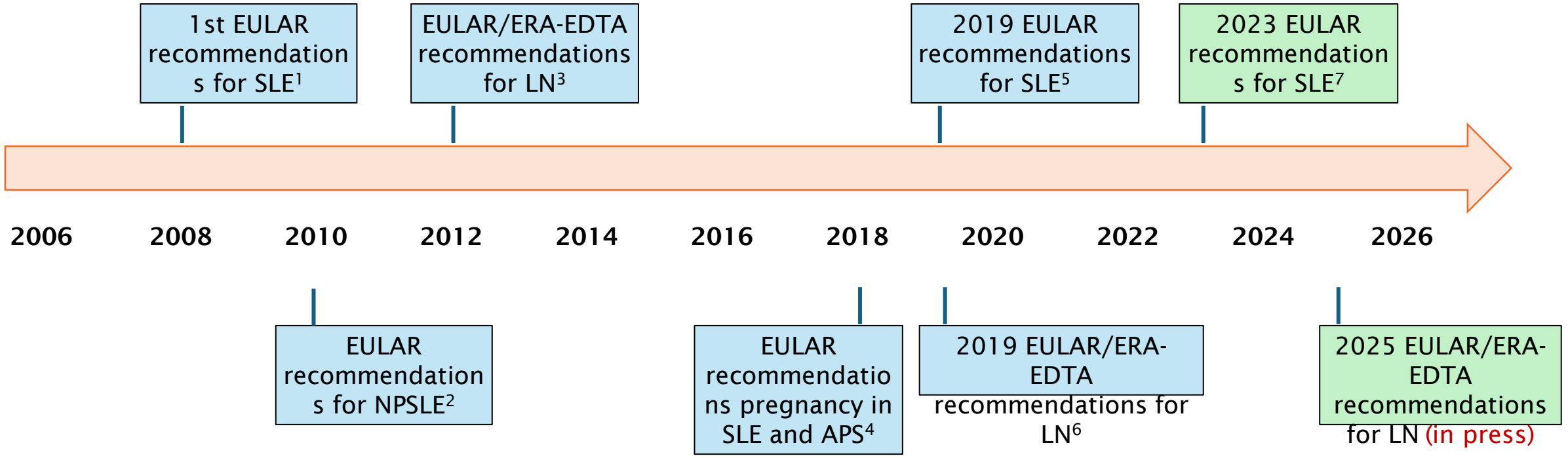
Κλινική Ρευματολογίας και Κλινικής Ανοσολογίας, ΠαΓΝΗ και Ιατρική Σχολή Πανεπιστημίου Κρήτης

03-10-2025

Σύγκρουση συμφερόντων

- **Speaker/consultancy fees:** Otsuka, Lilly, Pfizer, GSK, AstraZeneca, Novartis, AbbVie, Argenx

EULAR recommendations on SLE: counting >15 years



APS, antiphospholipid syndrome; ERA-EDTA, European Renal Association; EULAR, European Alliance of Associations for Rheumatology; LN, lupus nephritis; NPSLE, neuropsychiatric systemic lupus erythematosus; SLE, systemic lupus erythematosus.

1. Bertsias G et al. Ann Rheum Dis 2008;67:195-205; 2. Bertsias GK et al. Ann Rheum Dis 2010;69:2074-82; 3. Bertsias GK et al. Ann Rheum Dis 2012;71:1771-82;
4. Andreoli L et al. Ann Rheum Dis 2017;76:476-85; 5. Fanouriakis A et al. Ann Rheum Dis 2019;78:736-45; 6. Fanouriakis A et al. Ann Rheum Dis 2020;79:713-723.
7. Fanouriakis A et al. Ann Rheum Dis 2024;83(1):15-29

What prompted the 2023 update?

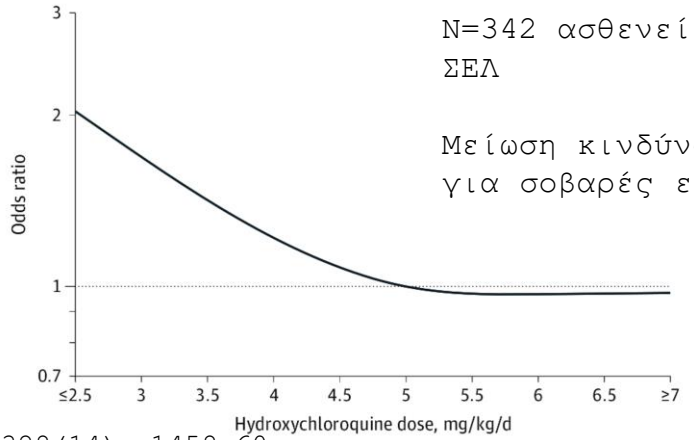
- Need for early/prompt diagnosis and treatment and the role of new classification systems
- Disease activity instruments and their implementation in clinical care
- **New agents approved (belimumab, anifrolumab, voclosporin)[1]**
- **Therapeutic strategy of SLE:**
  - Treat-to-target concept
  - Patient subgrouping/stratification based on sociodemographic, clinical and biological data,
  - Combination therapies[1]
  - Glucocorticoid tapering, withdrawal of immunosuppressive treatment
- **Improved strategies for the prevention of flares**
- **Assessment of the risk for infections (e.g., SARS-Cov2) and mitigation strategies (e.g. immunisations)**

SARS-Cov2, severe acute respiratory syndrome coronavirus 2; SLE, systemic lupus erythematosus.
Fanouriakis A et al. Ann Rheum Dis. 2024;83(1):15–29 and speakers own opinion (Dr Bertsias is part of the EULAR Task Force for the SLE and Lupus Nephritis recommendations [2008–2023])

General SLE recommendations

Recommendation[1]	LoA mean (SD)
HCQ is recommended for all patients (1b/A), unless contraindicated, at a target dose of 5 mg/kg real body weight/day (2b/B) but individualised based on risk for flare (2b/B) and retinal toxicity	9.21 (1.35)

Figure. Estimated Odds Ratio for Lupus Flares According to the Average Weight-Based Hydroxychloroquine Dose



N=342 ασθενείς με ΣΕΛ

Μείωση κινδύνου και για σοβαρές εξάρσεις

JAMA. 2022; 328(14): 1458-60

Χρήση του HCQ

- 200 → 400 mg/ημέρα (αρχική δόση): διατήρηση τουλάχιστον 3-6 μήνες, έως σταθεροποίησης νόσου
- Νόσος υπό έλεγχο: τιτλοποίηση προς τα 5 mg/kg/ημέρα

GFR, glomerular filtration rate; HQC, hydroxychloroquine; LoA, level of agreement; LN, lupus nephritis; SD, standard deviation; SLE, systemic lupus erythematosus.
Fanouriakis A *et al. Ann Rheum Dis.* 2024;83(1):15-29; 2. Speaker's own opinion.

LoA: Level of agreement

General SLE recommendations

Recommendation[1]	LoA mean (SD)
GCs, if needed, are dosed based upon the type and severity of organ involvement (2b/C) and should be reduced to maintenance dose of ≤5 mg/day (prednisone equivalent) (2a/B) and, when possible, withdrawn; in patients with moderate to severe disease, pulses of IV MP (125–1000 mg per day, for 1–3 days) (3b/C) can be considered	9.57 (0.77)

Points to consider

- 'Acceptable' threshold of daily GC dose for maintenance treatment reduced to maximum 5 mg/day prednisone equivalent (*vs.* 7.5 mg/day in the 2019 recommendations)
- Extrapolation from observational data linking GC dose >5 mg/day to exaggerated risk for organ damage[2]

GC, glucocorticoids; IV, intravenous; LoA, level of agreement; LN, lupus nephritis; MP, mycophenolate; SD, standard deviation; SLE, systemic lupus erythematosus.

1. Fanouriakis A *et al. Ann Rheum Dis*. 2024;83(1):15-29.; 2. Speaker's own opinion.

Ann Rheum Dis. 2024; 83: 15–29

Χρήση κορτικοστεροειδών: «…more art than science…»

Patients with mild SLE

Timing	GC dosage
Week	**Oral prednisone equivalent (mg/day)**
1	10–15
2	5–10
3	2.5–5
4	0 if lupus is controlled

Patients with moderate SLE

Timing	GC dosage
Optional day 1–3	**With or without*** IV MP pulses 0.125–0.5 g/day (for up to 3 days as initial treatment)
Week	**Oral prednisone equivalent (mg/day)**
1	20–30
2	20–25
3	15–20
4	15–20
5	10–15
6	10–15
7	5–10
8	5–10
9	2.5–7.5
10	2.5–7.5
11	0–5
12	0–5
13	0–2.5
14	0–2.5
>15	0 if lupus is controlled

Patients with severe non-renal SLE or active lupus nephritis†‡

Timing	GC dosage
Day 1–3	IV MP pulses 0.25–0.5 g/day (for up to 3 days as initial treatment)
Week	**Oral prednisone equivalent (mg/day)**
0–2	0.5–0.6 mg/kg (up to 40)
3–4	0.3–0.4 mg/kg
5–6	15
7–8	10
9–10	7.5
11–12	5
13–14	2.5
15–16	2.5
17–18	2.5
19–20	2.5
21–24	2.5
>25	<2.5

APLAR 2025
(abstract)

- Ώσεις μεθυλπρεδνιζολόνης:
  - Σε «απειλητική» νόσο (π.χ. μεγάλη περικαρδιακή συλλογή με αρχόμενο cardiac tamponate, θρομβοπενία <30.000/μL, κυψελιδίτιδα)
  - Όταν σκεφτείς τη χορήγηση ≥30-40 mg/ημέρα ισοδύναμο πρεδνιζόνης
  - Για την «επιτάχυνση» του tapering στεροειδών
  - Δοσολογία: 125 – 1000 mg, x1-3 ώσεις

General SLE recommendations

Recommendation	LoA mean (SD)
In patients not responding to HCQ (+/- GC) or patients unable to reduce GC below doses acceptable for chronic use, addition of immunomodulating/immunosuppressive agents (methotrexate [**1b/B**], azathioprine [**2b/C**] or mycophenolate [**2a/B**]) and/or biologic agents (**belimumab** [1a/A] or **anifrolumab** [**1a/A**]) should be considered	9.32 (0.91)

Points to consider

- RCTs and LTE support the superiority of biologics (over SoC) in reducing SLE activity, flares and GC dose
- Not a prerequisite to try ≥1 csDMARDs/ISTs prior to biologics
- No preference between the two currently approved biologics (for general SLE)
- **Emphasis on the prompt initiation of csDMARDs/ISTs or biologics** to expedite disease control and GC reduction

csDMARD, conventional synthetic disease-modifying antirheumatic drug; GC, glucocorticoids; HCQ, hydroxychloroquine; IST, immunosuppressive therapy; LoA, level of agreement; LN, lupus nephritis; LTE, long-term extension; RCT, randomised controlled trials; SD, standard deviation; SLE, systemic lupus erythematosus; SoC, standard-of-care.

Fanouriakis A *et al. Ann Rheum Dis*. 2024;83(1):15-29

Μυκοφαινολικό: αποτελεσματικότητα στο ΣΕΛ

Table 2. Improvement of clinical and laboratory manifestations in 6 and 12 months. Values are n (%) unless otherwise specified.

Variables	Time	Nonrenal, n = 72	Renal, n = 108
CNS	Baseline	11	7
	6 mos	8 (72.7)	3 (42.9)
	12 mos	8 (72.7)	6 (85.7)
Vasculitis	Baseline	2	6
	6 mos	2 (100)	6 (100)
	12 mos	1 (50)	6 (100)
MSK manifestations	Baseline	19	11
	6 mos	11 (57.9)	10 (90.9)
	12 mos	14 (73.7)	11 (100)
Renal	Baseline	0	105
	6 mos	0 (0)	20 (19)
	12 mos	0 (0)	29 (27.6)
Skin disease	Baseline	27	30
	6 mos	7 (25.9)	10 (33.3)
	12 mos	11 (40.7)	16 (53.3)
Serositis	Baseline	8	7
	6 mos	6 (75)	4 (57.1)
	12 mos	5 (62.5)	7 (100)
Immunological abnormalities	Baseline	45	85
	6 mos	11 (24.4)	15 (17.6)
	12 mos	12 (26.7)	15 (17.6)
Hematological abnormalities	Baseline	10	3
	6 mos	8 (80)	2 (66.7)
	12 mos	6 (60)	2 (66.7)

CNS: central nervous system; MSK: musculoskeletal.

- Δεδομένα από σειρές ασθενών (σε συνδυασμό με μέτριες/υψηλές δόσεις στεροειδών) & μία ελεγχόμενη μελέτη (MMF vs. AZA)

- Αποτελεσματικότητα σε:
  - ✓ **Υποξύ δερματικό λύκο**
  - ✓ **Αιμολυτική αναιμία**
  - ✓ **Θρομβοπενία**
  - ✓ **Αγγειίτιδα**
  - ✓ **Νευρολογικές εκδηλώσεις**

- Κλινικά **σημαντικό αποτέλεσμα στους 3–4 μήνες** ➔ ύφεση ≈ 40–50% στο 1 έτος

- **Υποτροπές ≈ 15–20%**

- **Κύρια ένδειξη: μετρίως σοβαρές ή σοβαρές εκδηλώσεις ΣΕΛ, αστοχία ή τοξικότητα σε AZA, αποφυγή χρήσης CYC**

Tselios K, et al. *J Rheumatol.* 2016; 43:552-8; Conti F, et al. *Immunol Res*. 2014; 60:270-6; Posalski JD, et al. *Lupus*. 2009; 18:516-21

Βιολογικοί παράγοντες & τροποποίηση της φυσικής πορείας της νόσου

Table 1 Application of the proposed matrix for non-renal disease activity and organ damage disease modification criteria

Product	Disease modification potential		Disease modification confirmed (beyond 5 years)
	Outcomes: year 1	Outcomes: years 2–5	Outcomes: year 5 or later
	❶ Significant reduction in disease activity measured using a validated tool (ie, SELENA-SLEDAI, BILAG, SRI-4) ❷ Significant reduction in severe flare measured using a validated tool (ie, SFI or BILAG) ❸ Reduction in use of steroids* and/or immunosuppressants	❶ Sustained improvement in multiple organ domains/no worsening in multiple organ domains ❷ Prevention of severe flares ❸ Continued reduction in use of steroids* and/or immunosuppressants	No change in SDI or delayed progression
Anifrolumab	1 2 3	1 2 3	●
Belimumab	1 2 3	1 2 3	●

(εξωνεφρικός ΣΕΛ)

Διευκόλυνση επίτευξης των θεραπευτικών στόχων (treat-to-target)

Askanase AD, et al. Lupus Science & Medicine 2024;11:e001124; Ann Rheum Dis. 2025 May;84(5):767-776.

Organ-specific recommendations

Skin involvement in SLE	LoA mean (SD)
Treatment of active skin disease should include **topical agents** (GC, calcineurin inhibitors) (2b/B), antimalarials (HCQ, CQ) (1a/A), and/or systemic glucocorticoids (4/C) as needed, with **methotrexate** (1b/B), **mycophenolate** (4/C), **anifrolumab** (1a/A), or **belimumab** (1a/B), considered as second-line therapy	9.35 (1.06)

Points to consider

- Anifrolumab rated with LoE: A due to the CLASI data included in phase II/III trials
- **Real-world evidence supports its superior efficacy especially in refractory mucocutaneous involvement**

Organ-specific recommendations

Hematological involvement in SLE	LoA mean (SD)
For acute treatment of severe autoimmune thrombocytopenia, **high-dose GC** (including pulses of intravenous methylprednisolone) (4/C), with or without **intravenous immunoglobulin G** (4/C), and/or **rituximab (2b/B)**, and/or high-dose intravenous cyclophosphamide (4/C), followed by maintenance therapy with rituximab (2b/B), azathioprine (2b/C), mycophenolate (2b/C), or cyclosporine (4/C), should be considered.	9.35 (1.06)

General SLE recommendations

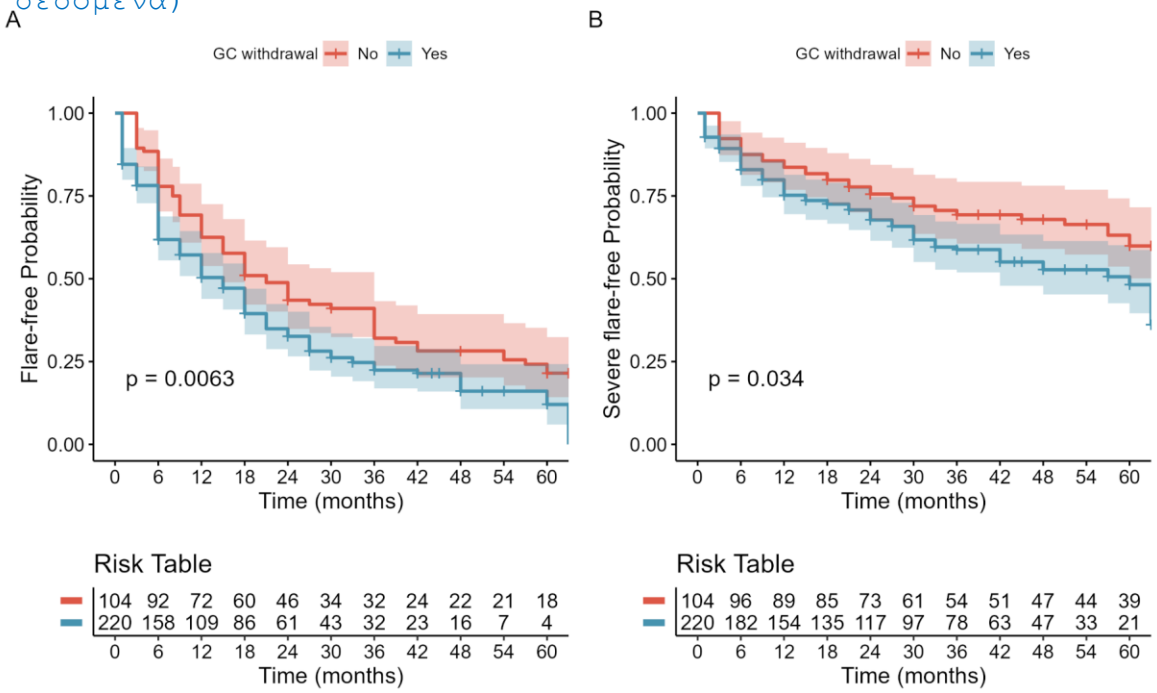
Recommendation[1]	LoA, mean (SD)[1]
In SLE patients achieving sustained remission, **gradual tapering** of treatment should be considered, with **withdrawal of GC first** (2a/B)	9.89 (0.38)

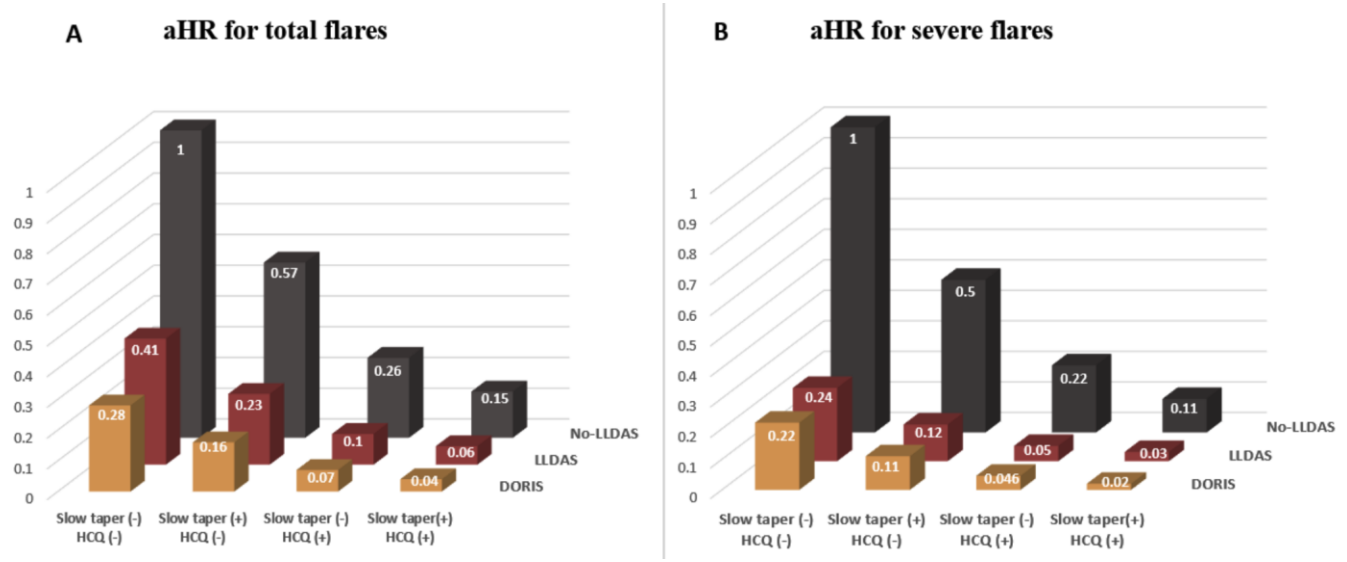
GC, glucocorticoids; IST, immunosuppressive therapy; LN, lupus nephritis; LoA, level of agreement; M0, month zero; M24, 24 months; SLE, systemic lupus erythematosus.

1. Fanouriakis A *et al. Ann Rheum Dis.* 2024;83(1):15–29. 2. Jourde-Chiche N *et al. Ann Rheum Dis.* 2022;81(10):1420–1427; 3. Jourde-Chiche N *et al. Nephrol Dial Transplant.* 2022; 37 (Suppl. 3): i818–i822 (Abstract FC053); 4. Speaker's own opinion.

Διακοπή των κορτικοστεροειδών στο ΣΕΛ

Ασθενείς με μέτριο/σοβαρό ΣΕΛ (προοπτικά δεδομένα)



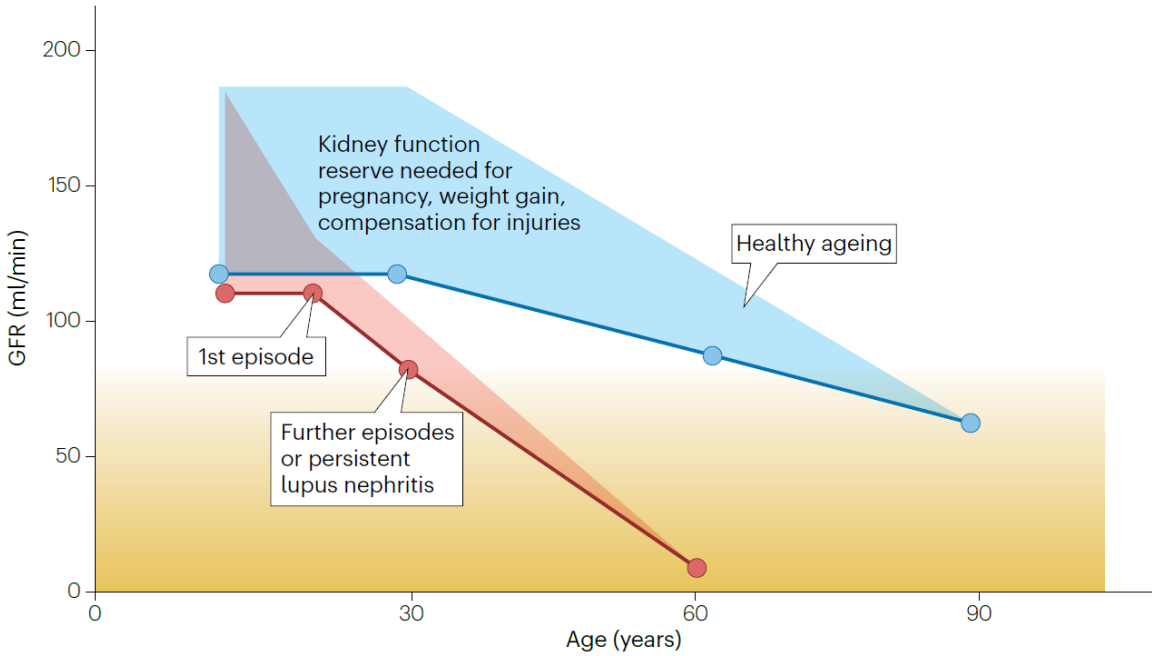


1. Slow GC tapering (6 months) until complete withdrawal
2. Use of HCQ >50% of time
3. DORIS or LLDAS (at the time of complete withdrawal)

Katechis S, et al. RMD Open. 2025; 11(1): e005118.

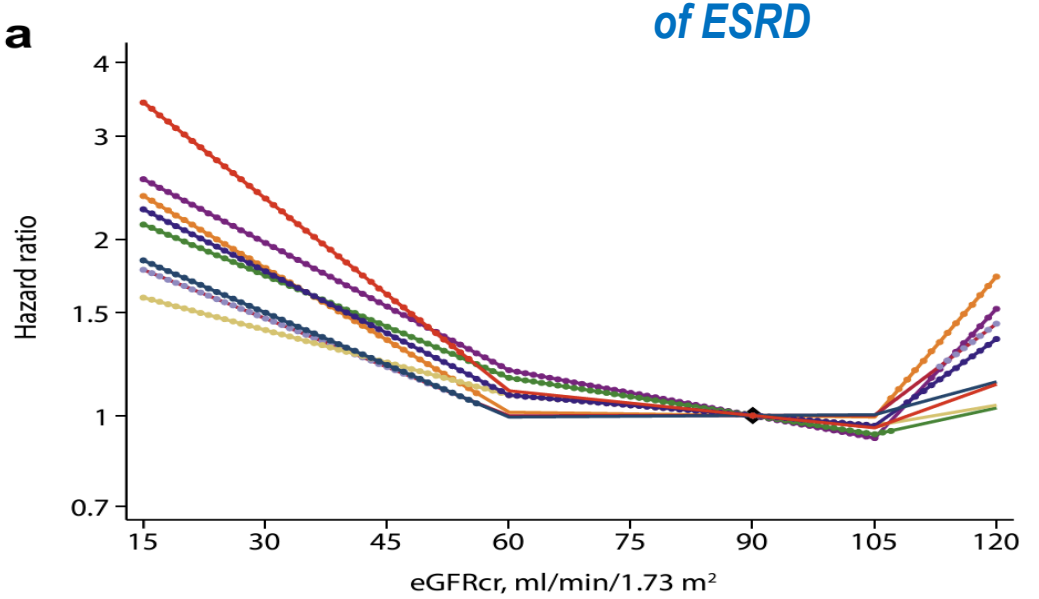
ΣΕΛ με νεφρική προσβολή

Functional reserve and its compromise by each flare of lupus nephritis



Nat Rev Rheumatol. 2024 Nov;20(11):699-711

The multiple morbidities and significant mortality of ESRD



The 60 ml/min GFR threshold

EULAR Recommendations for SLE with Kidney Involvement (2025)

Diagnosis/Targets

- **Kidney biopsy**

  Indispensable; repeat if clinical uncertainty

- **Target - Prevention of**

  - Chronic kidney disease

  - Flares

- **Milestones**

  - Kidney function: preservation or improvement by 3 months

  - Proteinuria:
  * Reduction by 25% at 3 mo
  * Reduction by 50% by 6 mo
  * UPCR < 700 mg/g by 12 mo

Immune treatment

- **Early combination therapy**

  Hydroxychroloquine and Glucocorticoids **with** immunosuppressive **and** CNI **or** biologic

- **Glucocorticoids**

  - Start with pulses

  - Continue with 0.3-0.7 mg/kg/day prednisone

  - Taper to ≤ 5 mg/day by 4-6 months and withdraw, when possible

- **Immunosuppressives**

- MMF, low-dose CY

- **CNI**

  - Voclosporin or TAC (CsA)

- **Biologics**

  Belimumab, Obinutuzumab

Non-immune treatment

- **Nephroprotection**

- Low salt (less than 5 g/day)

- Control blood pressure (RAAS blockade 1st choice)

- SGLT2-inhibitors (in stable disease, if residual proteinuria after 12 mo)

- **Dyslipidaemia**

- **Vaccinations**

Influenza, COVID-19, HZV, S. pneumoniae

- **Bone health**

Severe or Refractory

- **Severe**

Consider high-dose IV-CY plus pulse IV-MP

- **Refractory**

  - Combination of IV-CY with B cell depletion

  - Addition of a CNI if heavy proteinuria

  - Experimental therapies in the context of a protocol

- **Thrombotic microangiopathy**

- Plasma exchange

- Complement inhibitors

- Anti-vWf (caplacizumab)

Recommendation 4: Immunosuppressive treatment (1)

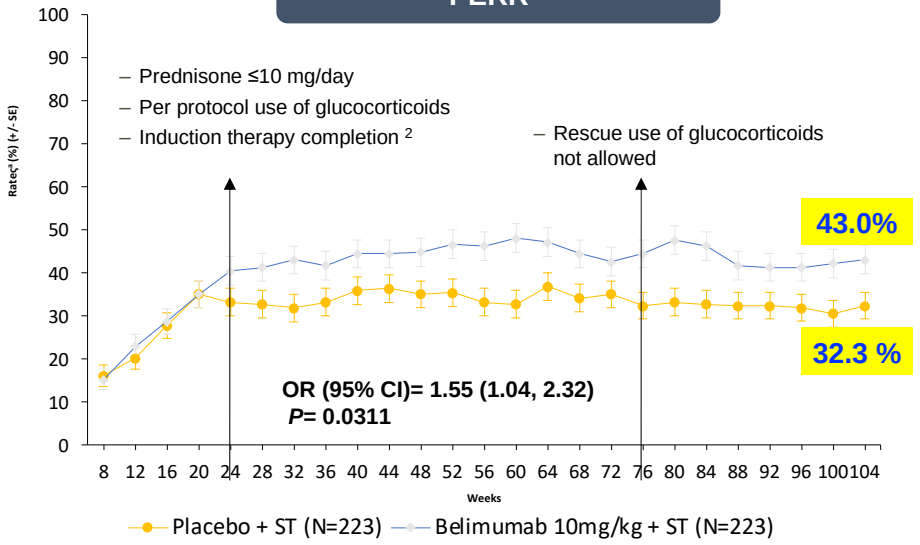
Recommendation	LoA, mean (SD)
For patients with active lupus nephritis, *especially those with poor prognostic factors*, **we recommend combination therapy** of: **a)** mycophenolate or low-dose intravenous cyclophosphamide with belimumab (**1b/A**), or **b)** mycophenolate with a calcineurin inhibitor (voclosporin or tacrolimus) (**1b/A**), or **c)** mycophenolate with obinutuzumab (**1b/A**). Alternative regimens include single-agent therapy with either mycophenolate (**1a/A**) or low-dose intravenous cyclophosphamide (**1a/A**).	**9.59 (0.78)**

LoA: Level of agreement

Targeting B-cells: belimumab (add-on) versus standard therapy in active lupus nephritis (BLISS-LN)

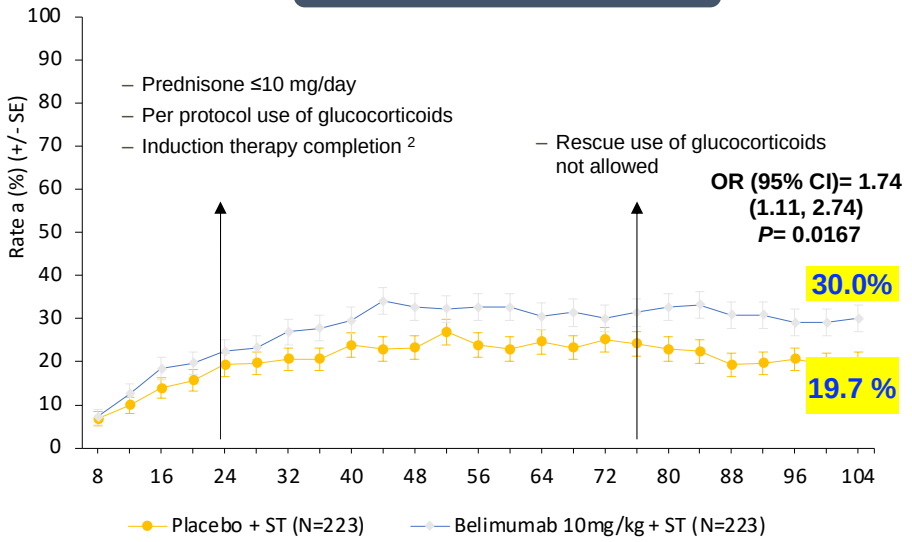
55% greater odds to meet the primary endpoint of renal response

PERR [1]



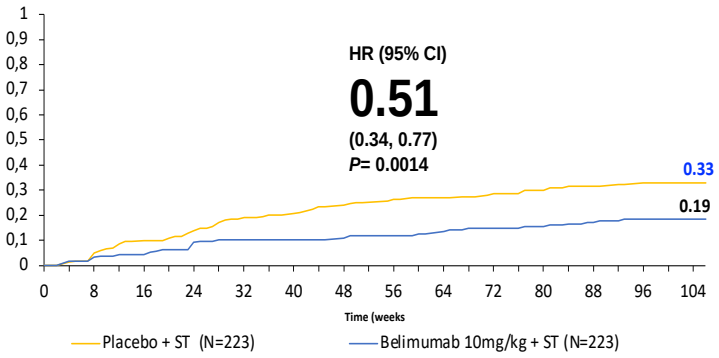
74% greater odds to achieve complete renal response

CRR



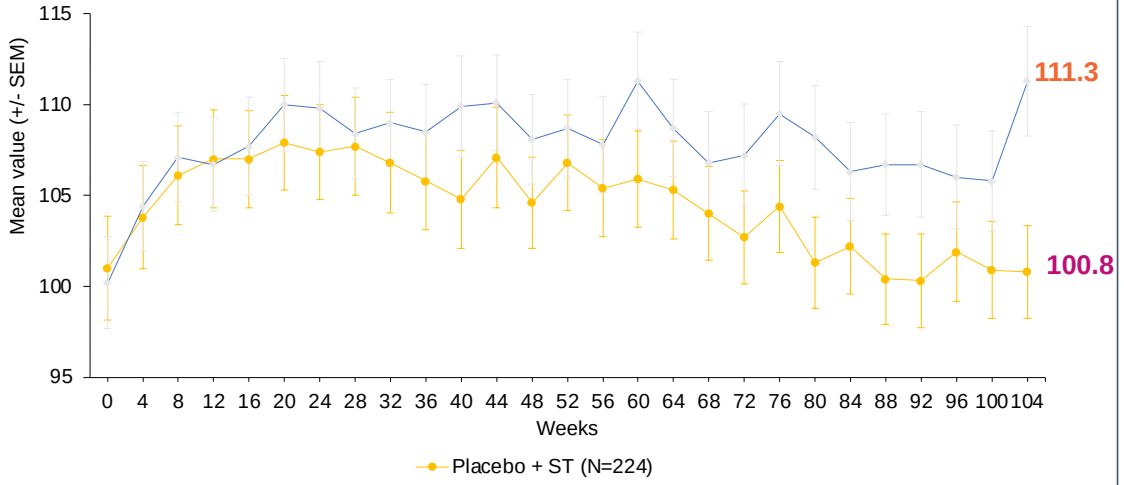
Furie R, et al, N Engl J Med. 2020;383: 1117-28

49% lower odds for renal adverse event or death in LN patients treated with belimumab



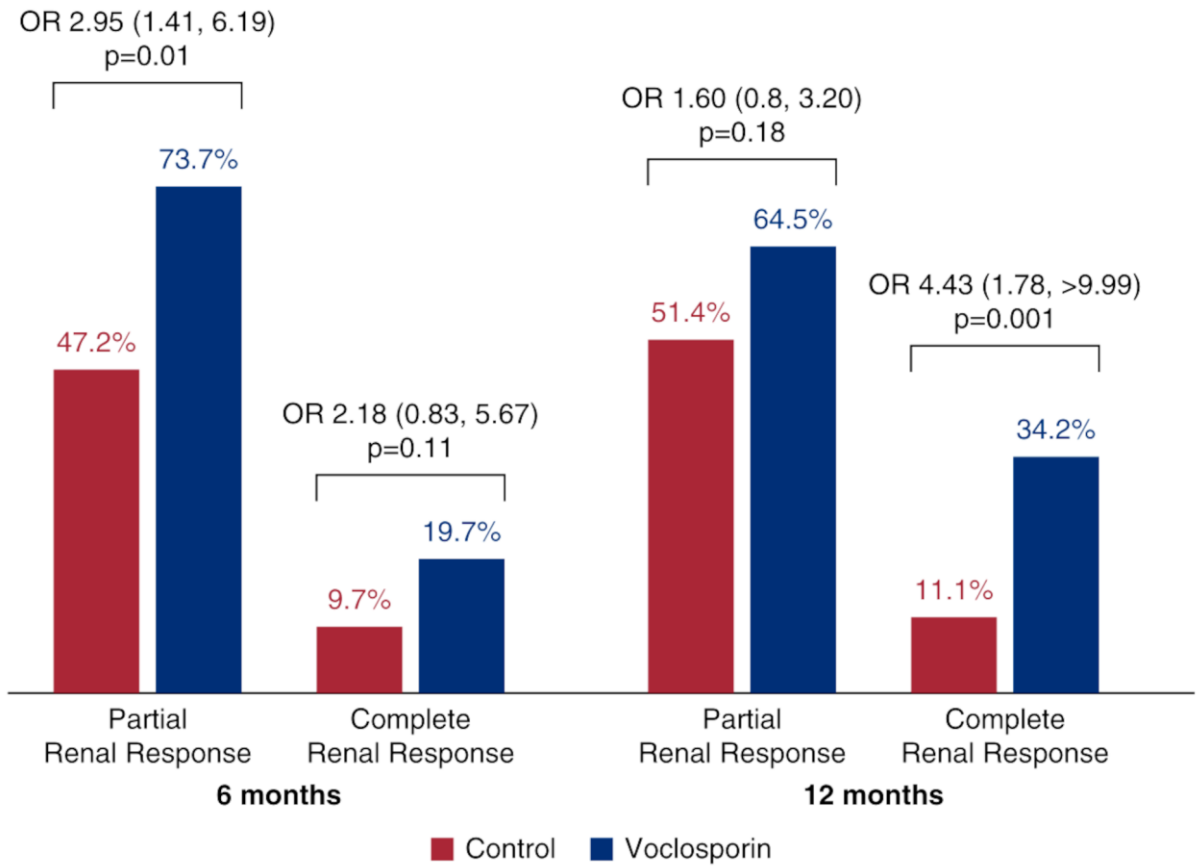
	Placebo + ST N=223	Belimumab 10mg/kg + ST N=223
Events [a], n	63	35
Renal worsening[b]	39	17
Treatment failure due to renal disease[c]	20	16
Doubling of serum creatinine	1	1
ESRD	1	0
Patient death	2	1

Average GFR was higher in belimumab-treated LN patients



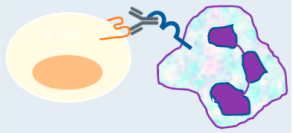
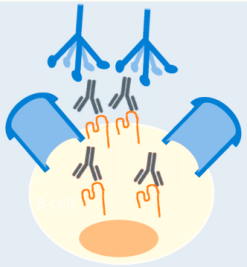
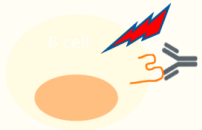
Efficacy of Voclosporin in Proliferative Lupus Nephritis with High Levels of Proteinuria

Hanni Menn-Josephy,[1] Lucy S. Hodge,[2] Vanessa Birardi,[2] and Henry Leher[2]



CJASN. 2024; 19: 309–318

Obinutuzimab (type II anti-CD20 mAb) depletes >93% of peripheral B-cells in SLE

Effector mechanism of B cell depletion by anti CD20 mAb		Type I anti-CD20 mAb	Type II anti-CD20 mAb **Obinutuzumab**
ADCC and ADCP FcGR dependent *Via Neutrophil*		++	++++
Via NK cell		++	++++
Internalisation and complement dependent cytotoxicity		+++	+
Direct Cell Death FcGR independent		+ Apoptosis	++ Nonapoptotic lysosome-mediated cell death

- Less dependent on complement-mediatd cytotoxicity
- Less internalized from the cell membrane
- Presumambly induces higher depletion of tissue infiltrating B-cells (kidneys)
- *In vitro* studies suggest less dependency of BAFF concentration

Reddy VR, et al. Rheumatology. 2022; 61: 2894–2904
Reddy VR, et al. Rheumatology. 2017; 56:1227–1237
Marinov AD, et al. Arthritis Rheumatol. 2021; 73: 826–836
Clin J Am Soc Nephrol. 2018;13(10):1502-9;
J Autoimmun. 2022; 102873; Biologicals. 2021; 69: 1–14
Ann Rheum Dis. 2022; 81: 100–7

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Efficacy and Safety of Obinutuzumab in Active Lupus Nephritis

R.A. Furie,[1] B.H. Rovin,[2] J.P. Garg,[3] M.B. Santiago,[4,5,6] G. Aroca-Martínez,[7,8] A.E. Zuta Santillán,[9] D. Alvarez,[11] C. Navarro Sandoval,[10] A.M. Lila,[13] J.A. Tumlin,[14] A. Saxena,[15] F. Irazoque Palazuelos,[16] H. Raghu,[3] B. Yoo,[3] I. Hassan,[17] E. Martins,[18] H. Sehgal,[18] P. Kirchner,[18] J. Ross Terres,[3] T.A. Omachi,[3] T. Schindler,[18] W.F. Pendergraft III,[3] and A. Malvar,[12] for the REGENCY Trial Investigators*

Table 2. Primary and Secondary End Point Results in the Intention-to-Treat Population.*

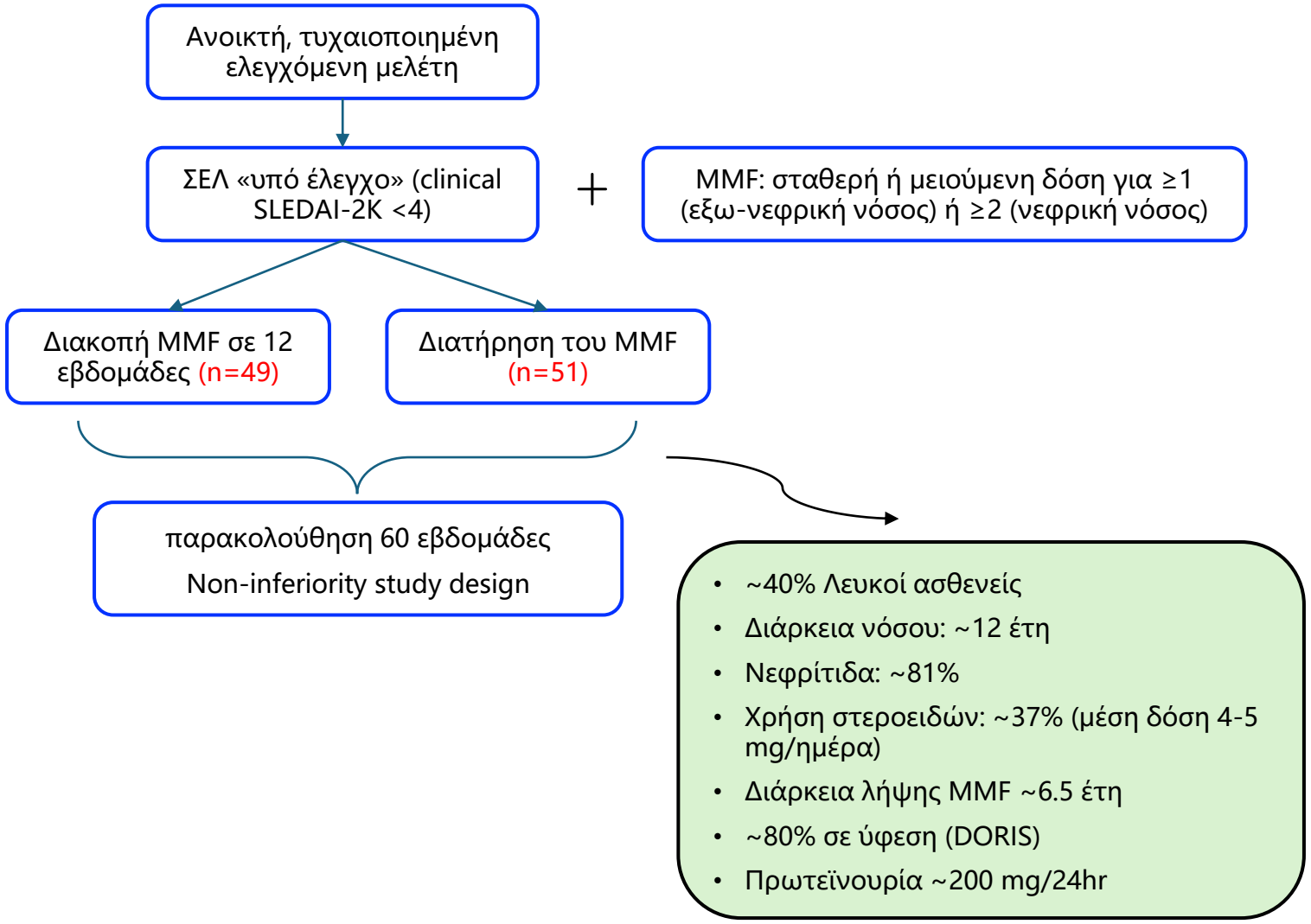
End Point	Obinutuzumab (N=135)	Placebo (N=136)	Adjusted Difference (95% CI)†	P Value‡
Primary end point				
Complete renal response at wk 76 — % (95% CI)§¶‖	46.4 (38.0 to 54.9)	33.1 (25.2 to 41.0)	13.4 (2.0 to 24.8)	0.02
Key secondary end points				
Complete renal response at wk 76 with prednisone taper to ≤7.5 mg/day between wks 64 and 76 — % (95% CI)§¶‖**	42.7 (34.3 to 51.1)	30.9 (23.1 to 38.7)	11.9 (0.6 to 23.2)	0.04
UPCR <0.8 at week 76 with no intercurrent event — % (95% CI)¶‖††	55.5 (47.1 to 64.0)	41.9 (33.6 to 50.2)	13.7 (2.0 to 25.4)	0.02
Change in the eGFR from baseline to wk 76 — ml/min/1.73 m^2‡‡	2.31±2.71	−1.54±2.71	3.84 (−1.83 to 9.51)	
Death or renal-related event through wk 76 — % (95% CI)¶§§	18.9 (12.1 to 25.6)	35.6 (27.5 to 43.8)	−16.8 (−27.4 to −6.2)	
Overall renal response at wk 50 — % (95% CI)¶‖¶¶	59.1 (50.8 to 67.4)	50.7 (42.2 to 59.2)	8.4 (−3.4 to 20.1)	
Change in FACIT-F score from baseline to wk 76 — points‡‡	1.8±1.2	3.1±1.2	−1.4 (−3.9 to 1.2)	

Recommendation 7: Gradual withdrawal of treatment

Recommendation	LoA, mean (SD)
In patients in sustained remission, gradual withdrawal of immunosuppressive and/or biologic therapy should be considered **after three years of therapy following response**, taking into consideration the risk for flare (**2a/B**)	**9.31 (0.97)**

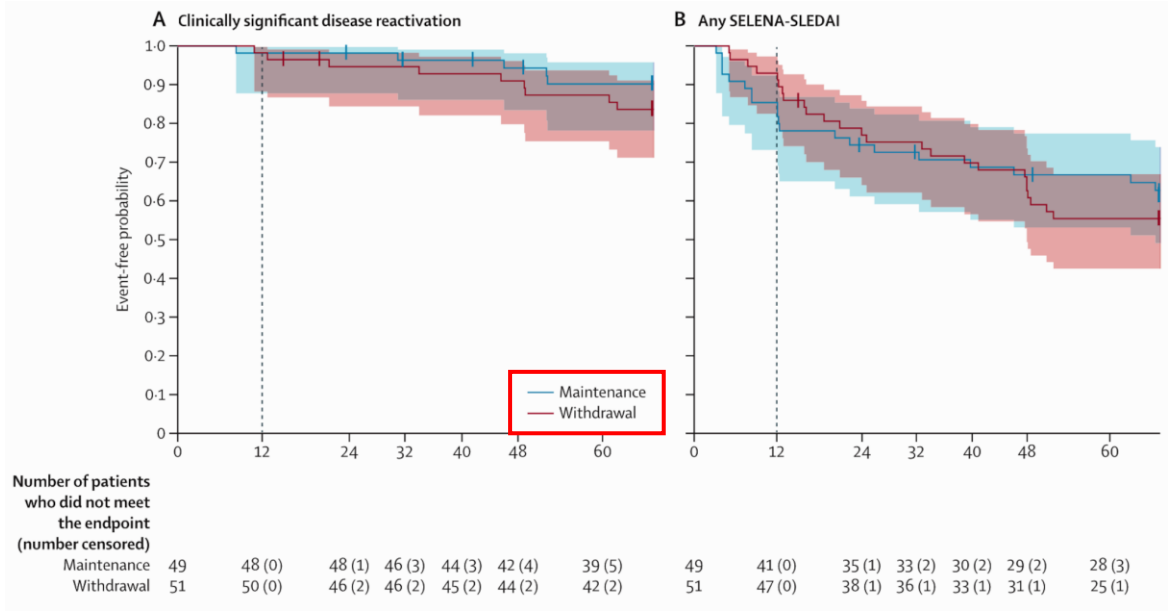
LoA: Level of agreement

Διακοπή του μυκοφαινολικού σε σταθερό, ανενεργό ΣΕΛ



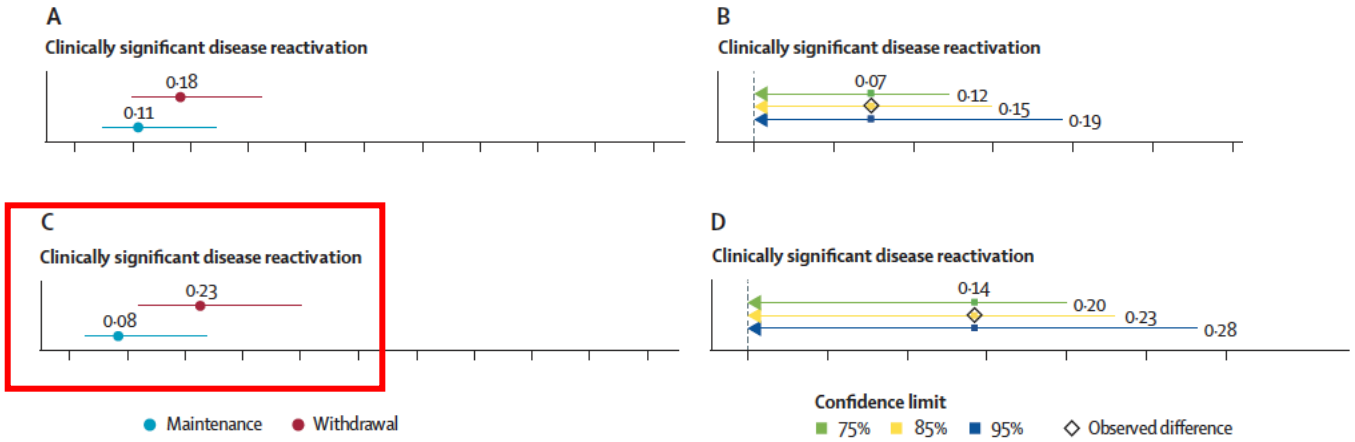
Chakravarty EF, et al. Lancet Rheumatol 2024; 6: e168–77

Διακοπή του μυκοφαινολικού σε σταθερό, ανενεργό ΣΕΛ: προσοχή στη νεφρίτιδα!



Σύνολο ασθενών

Ασθενείς με νεφρίτιδα



Chakravarty EF, et al. Lancet Rheumatol 2024; 6: e168–77

EULAR Recommendations for SLE with Kidney Involvement (2025)

Diagnosis/Targets

- **Kidney biopsy**

  Indispensable; repeat if clinical uncertainty

- **Target - Prevention of**

  - Chronic kidney disease

  - Flares

- **Milestones**

  - Kidney function: preservation or improvement by 3 months

  - Proteinuria:
  * Reduction by 25% at 3 mo
  * Reduction by 50% by 6 mo
  * UPCR < 700 mg/g by 12 mo

Immune treatment

- **Early combination therapy**

  Hydroxychroloquine and Glucocorticoids **with** immunosuppressive **and** CNI **or** biologic

- **Glucocorticoids**

  - Start with pulses

  - Continue with 0.3-0.7 mg/kg/day prednisone

  - Taper to ≤ 5 mg/day by 4-6 months and withdraw, when possible

- **Immunosuppressives**

- MMF, low-dose CY

- **CNI**

  - Voclosporin or TAC (CsA)

- **Biologics**

  Belimumab, Obinutuzumab

Non-immune treatment

- **Nephroprotection**

- Low salt (less than 5 g/day)

- Control blood pressure (RAAS blockade 1st choice)

- SGLT2-inhibitors (in stable disease, if residual proteinuria after 12 mo)

- **Dyslipidaemia**

- **Vaccinations**

Influenza, COVID-19, HZV, S. pneumoniae

- **Bone health**

Severe or Refractory

- **Severe**

Consider high-dose IV-CY plus pulse IV-MP

- **Refractory**

  - Combination of IV-CY with B cell depletion

  - Addition of a CNI if heavy proteinuria

  - Experimental therapies in the context of a protocol

- **Thrombotic microangiopathy**

- Plasma exchange

- Complement inhibitors

- Anti-vWf (caplacizumab)

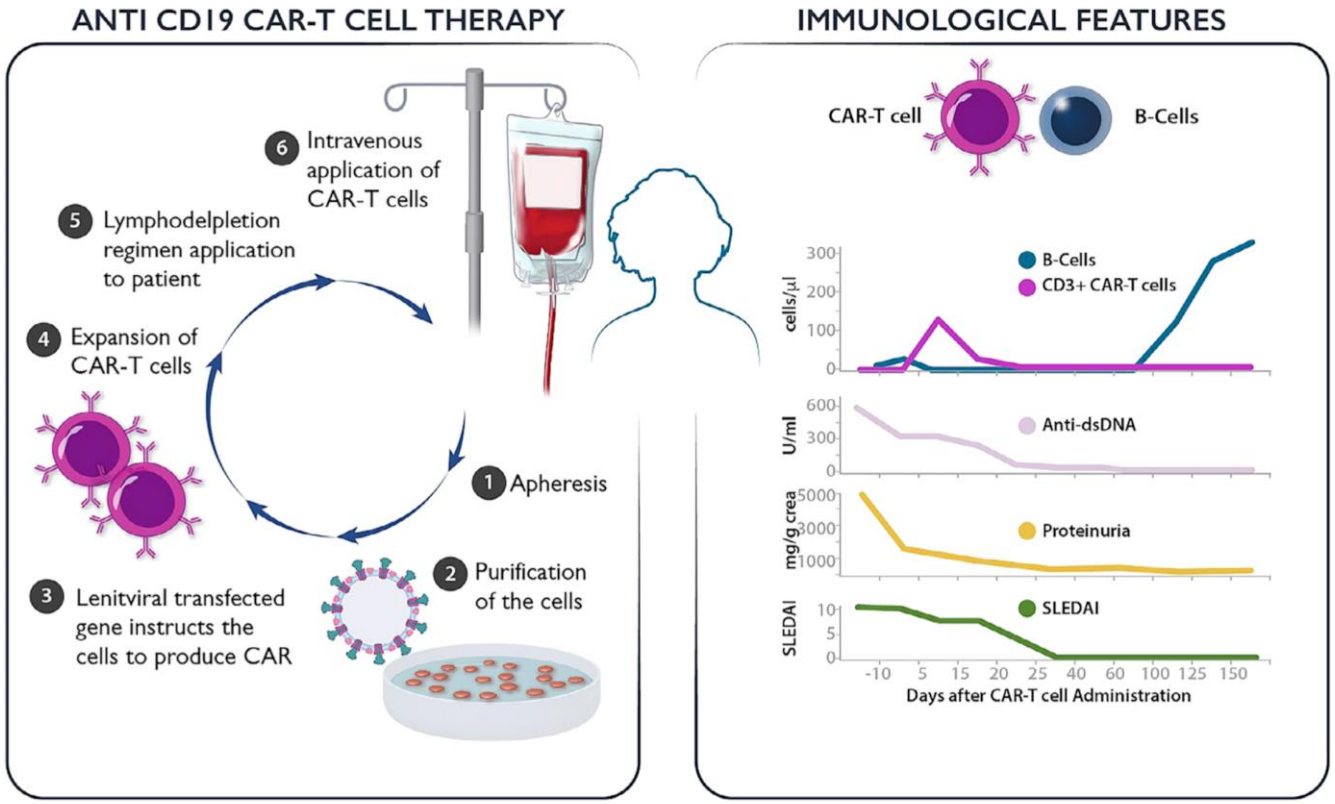
Τι να αναμένουμε;

- Από την ύφεση (DORIS), στην «βαθιά» ύφεση (DORIS+)
- Στρατηγικές ενίσχυσης της αποτελεσματικότητας υφιστάμενων θεραπειών (π.χ., πρώιμη έναρξη, βιοδείκτες)
- Νεφρίτιδα: (επαναληπτική) βιοψία για την εξατομίκευση της θεραπείας
- Βελτίωση αντιμετώπισης συννοσηροτήτων
- Νέοι θεραπευτικοί παράγοντες

Καινοτόμες στοχευμένες θεραπείες στο ΣΕΛ

Promising new agents
Anti-BAFF-receptor
Upadacitinib (JAK1 inhibitor)
Deucravacitinib (TYK2 inhibitor)
“Engagers” (e.g. bispecific CD20/CD3 T-cell engaging antibody)

…allogeneic CD19+ CAR T-cells
BCMA CAR T-cells
CAR NK cells
….CAR T & mAbs



Arthritis Rheumatol. 2024; 76(4):497-504