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EDITORIAL

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Lessons from IgA nephropathy: reevaluating the prognosis and management of class I and class II lupus nephritis

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KEY POINTS

What is already known?

- Current KDIGO and EULAR guidelines generally classify Class I and II lupus nephritis (LN) as usually nonproliferative lesions with favorable short-term renal findings („benign"), but uncertain long-term histological stability.
- Kidney-directed immunological therapy is specified for nephrotic-range proteinuria in these classes, leaving a "gray area" of subnephrotic involvement where the necessity of this kind of treatment remains poorly defined.
- The 0.5 g/day proteinuria threshold should serves as an individualized clinical guide beside clinical and serologic factors; however, if Class I/II LN is perceived as lacking therapeutic consequences, this proteinuria threshold risks becoming a rigid cutoff, inadvertently limiting the use of biopsy in patients with lower levels of proteinuria.

What this viewpoint adds?

- Histological instability: Retrospective data indicates that Class II LN is often not a stable endpoint but a precursor, with 70% of cases eventually transforming into more aggressive proliferative (Class III/IV) nephritis.
- The IgA Nephropathy analogy: This article highlights the shared mesangioproliferative pathology between Class I/II LN and IgA nephropathy (IgAN), suggesting that early intervention strategies proven in IgAN should be considered for LN as well.
- Economic impact: The transition to advanced CKD stages in SLE patients results in an exponential increase in healthcare costs (from $36,417 in Stage 1 to $152,169 in Stage 5).
- Proactive management: It advocates for a paradigm shift from a "wait-and-watch" approach toward early targeted intervention (e.g., BAFF or dual BAFF/APRIL inhibition), fostering a more individualized use of the proteinuria threshold and renal biopsy.

Introduction: the problem - current complacency

According to the latest KDIGO guidelines, specific kidney-directed immunological therapy for Class I (minimal mesangial) and Class II (mesangial proliferative) lupus nephritis (LN) is specified for cases presenting with significant, nephrotic-range proteinuria. In such cases, to treat 'lupus podocytopathy,' the initiation of therapy is recommended based on the analogy of minimal change disease and focal segmental glomerular sclerosis treatment protocols [1], (Figure 1). The EULAR guideline for LN, revised in 2025, does not address Class I/II LN. The position statement issued in 2019 declares that immunological treatment is generally not required, adding that 'the presence of significant proteinuria should prompt histological reassessment to detect proliferative changes that may have been overlooked' [2].

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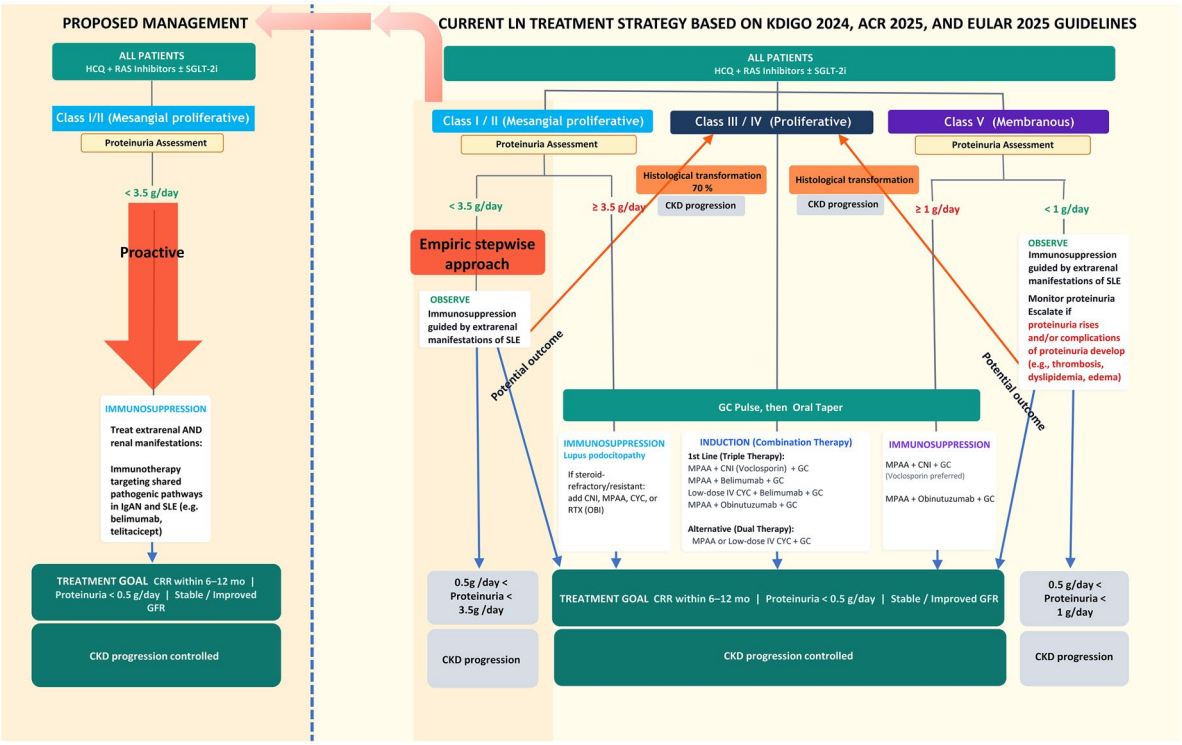


Figure 1. Flowchart comparing current and proposed Class I/II lupus nephritis (LN) management strategies, detailing assessment and therapy pathways.
CKD, Chronic kidney disease (KDIGO 2024: eGFR ≤ 60mL/min/1,73m2 and/or albuminuria ≥ 30mg/24 h and/or urine sediment abnormalities); LN, Lupus Nephritis; IgAN, IgA Nephropathy; Nefecon, targeted-release formulation of the corticosteroid budesonide; GC, Glucocorticoids; RAS-inhibitors, renin-angiotensin system inhibitor; SGLT-2i, sodium-glucose cotransporter-2 inhibitor; HCQ, hydroxychloroquine; MPAA, Mycophenolic Acid Analogs; CNI, Calcineurin Inhibitor; CYC, Cyclophosphamide; OBI, obinutuzumab; RTX, rituximab; CRR, Complete Renal Response.

Overall, the current guidelines classify Class I/II LN as a 'benign' lesion, recommending kidney-directed therapy only for nephrotic-range proteinuria, thereby representing a 'wait-and-watch' approach from a nephrological perspective [3].

Regarding kidney biopsy indication, besides the unexplained decrease in glomerular filtration rate (GFR), both the KDIGO and EULAR recommendations have maintained the 0.5 g/day proteinuria threshold [1,2]. Although the explicitly stated reason for this threshold is to make the indications for a kidney biopsy more deliberate, it creates a potential issue: if low-grade proteinuria is detected alongside a normal GFR, as lower levels of proteinuria are typically associated with low-class LN not requiring therapy intensification, biopsies may not be performed even in equivocal cases [4]. Such equivocal cases encompass not only a concomitant active urinary sediment (e.g., microscopic hematuria, cellular casts) but also clinical profiles carrying an inherently higher risk of severe LN incidence and progressive disease, such as younger age at onset (pediatric and adult patients aged <30 years), male sex, non-White populations, and persistent serologic activity characterized by hypocomplementemia, and/or elevated anti-dsDNA titers, and/or anti-C1q antibodies [1–3,5].

Progression to higher class: the evidence of histological instability and lack of standardized treatment

While large, prospective, randomized controlled trials have not been conducted in patients with Class I/II LN, retrospective studies - based on repeat biopsy findings - indicate a clinically significant probability of histological transformation to a higher class in these patients: an average of 70% if a second biopsy was performed for every Class II LN patient, or an average of 75% if the biopsy was performed according to the clinical course. [3,6–15; Table 1].

The progression from Class I to Class II lupus nephritis marks a definitive shift from minimal mesangial involvement to overt mesangial hypercellularity. In Class I disease immune complexes are strictly confined to the mesangium, usually accompanied by the 'full house' pattern with deposition of multiple immunoglobulin classes (IgG, IgM, IgA) and complement components (C3, C1q), but still failing to induce a significant inflammatory response or structural alterations detectable by light microscopy. As the pathology advances to Class II, the increased accumulation of these deposits triggers both a proliferative response in the mesangial

Table 1. Biopsy-proven histological transformation of Class II lupus nephritis (LN) across various studies.

Study	No. of SLE patients who had biopsy/ No. of Class I/II LN patients	No. Class II LN patients with repeat biopsy	Patients with histological transformation (HT)	No. of HT to Class III	No. of HT to Class IV	No. of HT to Class III/IV+V	No. of HT to Class V	No. of HT to Class VI	HT from Class II to proliferative (Class III/IV) lesions (%) if repeat biopsy biopsy was performed
Pagni et al. (2013)	**142/18**	**18**	**13**	**1**	**9**	**2**	**1**		**66.6**
Alsuwaida et al. (2014)	**11/3**	**3**	**3**	**1**	**1**	**1**			**100**
Greloni et al. (2014)	**45/5**	**5**	**5**	**1**	**2**		**2**		**60**
Yang et al. (2015)	1814/127	33	29	8	18	NA	3		89.6
Collado et al. (2016)	41/41	18	17	4	10		3		77.7
Narváez et al. (2017)	**54/9**	**9**	**7**	**2**	**3**		**2**		**55.5**
Alsuwaida et al. (2018)	32/32	17	11	5	4			2	52.9
Pakozdi et al. (2018)	238/28	9	7	1	4	1		1	66.6
Wang et al. (2022)	104/104	16	14	3	3	6	2		87.5
Summary of available data on Class II LN patients	2481/367	128	106	26	54	10	13	3	

Studies marked in bold indicate that re-biopsy was performed for all Class II LN cases regardless of the clinical course.
LN, Lupus nephritis; HT, histological transformation; No., number.

cells and an expansion of the surrounding matrix [16–18]. If the underlying systemic inflammation remains uncontrolled, a further transition from Class II to Class III (focal) and IV (diffuse) LN may occur [3,4,19,20].

According to the limited repeat-biopsy literature neither proteinuria nor serological markers (anti-dsDNA and C3/C4) could reliably predict histological transformation from Class I/II LN to proliferative forms [8,9].

The incidence of transformation together with the lack of validated markers to identify this change should raise our awareness of this condition and lead us to question whether the aforementioned liberal approach and patience toward treating Class I/II LN with subnephrotic proteinuria – which reflects an underlying 'smoldering' mesangial proliferation in the absence of overt podocytopathy – is appropriate.

In addition, a fundamental inconsistency in the guidelines arises when a kidney biopsy confirms Class I or II LN, when proteinuria is detected above 0.5 g/day, while still below the nephrotic range. In such cases, where kidney-directed immunosuppressive treatment is not required, it remains unclear how to interpret these findings and achieve the proteinuria-based complete remission targets defined for Class III/IV/V LN (proteinuria <0.5 g/day). Are we to assume that immunosuppression administered for extrarenal symptoms, combined with adjunctive treatment, will resolve this?

Further uncertainty arises regarding the management of cases that relapse in Class II form after the treatment of Class III or IV lupus nephritis. Could such an outcome be interpreted either as the unsuccessful treatment of a proliferative form, indicating a need for revision of therapy or as a "benign" residual lesion?

Taken together, these factors represent a 'gray area' and an avenue that should be addressed to shift our current paradigms in Class I/II LN and to initiate prospective clinical trials in this patient cohort, focusing on long-term kidney outcomes, protocol or risk-triggered repeat biopsy and biomarkers.

The analogy: learning from IgA nephropathy (IgAN) - can we prevent progression?

Among the primary renal diseases, IgA nephropathy (IgAN) is considered the classic form of mesangioproliferative glomerulonephritis. On light and electron microscopy IgAN and Class II LN share nearly identical features, such as mesangial cell proliferation, although immunofluorescence reveals predominant IgA deposition in the former and the 'full house pattern' in the latter [16–20; Table 2]. Evidence from IgAN shows that mesangioproliferative lesions can carry a poor prognosis on their own, even without the progression to severe proliferative forms that may occur in LN.

Analogous to LN, the proteinuria serves as the primary surrogate endpoint in IgAN, from which long-term outcomes are inferred [21–23].

Table 2. Comparison of Class I/II lupus nephritis and IgA nephropathy.

Feature	Class I/II lupus nephritis (LN)	IgA Nephropathy (Berger's Disease)
Underlying Disease	Systemic Lupus Erythematosus (SLE)	Primary kidney disease (or Henoch-Schönlein)
Dominant Deposit	IgG, IgM, IgA, C3, C1q ("Full-house")	IgA1 (dominant or co-dominant)
Complement Levels	Often low (C3, C4)	Usually normal
Histology / Light Microscopy (LM)	I: Normal; II: Mesangial hypercellularity and/or mesangial matrix expansion	Mesangial hypercellularity and matrix expansion, endocapillary hypercellularity can occur; Some cases histologically normal (~10%)
Histology / Immunofluorescence (IF)	Positive for all immunoglobulins and C1q, C3, C4	Strong, diffuse mesangial IgA and C3 staining (~90%)
Histology / Electron Microscopy (EM)	I: Mesangial immune (dense) deposits II: Mesangial immune (dense) deposits There may be a few isolated subepithelial or subendothelial deposits visible by IF or EM but not by LM	Mesangial and paramesangial immune (dense) deposits; subepithelial and subendothelial deposits can also be seen
Clinical Symptoms	Microscopic hematuria, mild proteinuria; Creatinine normal	Microscopic hematuria, with or without proteinuria (usually <2g/day); sometimes macroscopic hematuria Creatinine normal or elevated
Targeted biologic treatments: approved, under investigation, or supported by case reports	For SLE: BAFF inhibitors: • Belimumab APRIL and BAFF inhibitors: • Telitacicept • Atacicept Plasma cell inhibitors: • Bortezomib • Daratumumab Complement inhibitors: • Iptacopan	For IgAN: BAFF inhibitors: • Belimumab APRIL inhibitors: • Sibeprenlimab • Zigakibart APRIL and BAFF inhibitors: • Telitacicept • Atacicept • Povetacicept Plasma cell inhibitors: • Bortezomib • Felzartamab • Mezagitamab Complement inhibitors: • Iptacopan

EM, Electron Microscopy; IF, Immunofluorescence; LM, Light Microscopy; APRIL, cytokine A proliferation-inducing ligand; BAFF, B-cell activating factor.

Although proteinuria is an imperfect follow-up marker in LN, as it is nonspecific for ongoing inflammatory activity, it remains a well-established, reliable prognostic factor [21]. Several novel biomarker candidates show promise in reflecting intraglomerular processes, none are currently validated for routine clinical practice. Furthermore, while repeat kidney biopsy remains the gold standard, its invasiveness can limit its use in routine follow-up. Therefore, in LN, while a holistic monitoring approach – integrating kidney function (eGFR), urinary sediment, complement levels (C3/C4), and serological activity (such as anti-dsDNA and anti-C1q antibodies) – remains irreplaceable, current clinical guidelines continue to define proteinuria-based response targets [1–3,5].

Based on outcome data from large-scale studies in IgAN, improving long-term renal outcomes has required a revision of proteinuria goals, which should now be kept as low as possible to prevent progression to advanced chronic kidney disease (CKD) and end-stage renal disease (ESRD) [22]. While LN guidelines remain liberal, recent KDIGO guidelines for IgAN have tightened proteinuria targets, stating that protein excretion 'should be maintained at a minimum of less than 0.5 g/day (or equivalent) and ideally at less than 0.3 g/day (or equivalent)' [22].

Considering the pathological similarities between the mesangial forms of SLE kidney involvement and IgAN – and given that proteinuria is a shared surrogate marker – reducing protein excretion through the control of the underlying immune process, besides conservative nephroprotective care, can be a key to delaying renal failure and preventing transformation.

In recent years, the immunological therapies targeting the mesangioproliferative process of IgAN, have evolved significantly. These emerging therapies target pathways already utilized in SLE treatment, such as B-cell-activating factor (BAFF) inhibition, dual BAFF and a proliferation-induced ligand (APRIL) inhibition, complement inhibition and plasma cell inhibition [14,20,23–25, Table 2].

Increasing evidence highlights the benefits of early, effective disease control in SLE [26,27]. Lupus nephritis represents an organ manifestation of SLE rather than an independent disease entity, as emphasized by the updated 2025 EULAR guidelines on SLE with kidney involvement [28]. Taken together, these considerations provide a clear rationale for selecting from the above-mentioned, effective therapies in SLE when Class I/II LN is detected, on the assumption that they can simultaneously control extrarenal manifestations and the underlying mesangial process.

While targeted biologic therapies represent a higher upfront investment, their use in early-stage LN may be cost-effective by preventing insidious damage or histological transformation to proliferative classes. The

financial impact is profound: according to US data from Edgecomb et al. (2026), annual healthcare costs for SLE patients with CKD are nearly double those without renal involvement ($59,956 vs. $31,652), and expenditures escalate exponentially with advancing CKD stage, rising from $36,417 in Stage 1 to $152,169 annually in Stage 5 [29]. A paradigm shift toward „proactive" treatment could significantly reduce the long-term healthcare expenditure associated with CKD progression.

This highlights a growing need to reevaluate the diagnostic approach and treatment of Class I/II LN.

Summary

'Time is kidney': based on data from retrospective studies indicate that mesangioproliferative forms of LN progress to more severe stages in a significant proportion of cases (Table 1, Figure 1). Furthermore, according to data from kidney and cardiovascular outcome trials, addressing even low levels of proteinuria is crucial, as reducing protein excretion is key to delaying CKD progression and reducing cardiovascular risk [22,30].

Coupled with this, the therapeutic successes achieved in IgAN – which shares a similar mesangial pathology – support the premise that arresting the mesangioproliferative process significantly improves long term renal outcomes. Targeted therapies addressing pathways identical to those in IgAN are already part of clinical practice in SLE and carry a 'rational risk' relative to the expected clinical benefits and long-term cost-effectiveness [31–33].

Recent advances in immunotherapies (e.g., belimumab, telitacicept) enable the selection of personalized and effective immunosuppressive therapy tailored to the patient's clinical profile (extrarenal activity, serological profile, degree of proteinuria), balanced with an acceptable safety profile and, in particular, manageable infectious risk [31–33].

In summary, we should be „proactive" in our therapeutic approach to Class I/II LN with subnephrotic-range proteinuria – given that guidelines already recommend specific immunological therapy for nephrotic-range cases – and consider initiating targeted treatment guided by histopathology, proteinuria levels and the patient's clinical SLE phenotype (Figure 1).

Moreover, although both the 2025 EULAR and 2024 KDIGO LN guidelines emphasize that the 0.5 g/day proteinuria threshold is intended only as a clinical guide for intervention [1,28], this threshold easily becomes a rigid cutoff if lower-class LN is perceived as lacking therapeutic consequences [4]. In contrast by adopting the view that targeted treatments are available and more stringent treatment targets are achievable even in lower-class LN, the proteinuria threshold can more easily evolve into a truly individualized approach.

Therefore, there is a pressing need to collect more data on Class I/II LN and to enroll patients with these classes into prospective studies focusing on both the natural history of the disease and the efficacy of conventional synthetic and targeted immunosuppressive therapies (e.g. B-cell-directed agents, complement inhibitors, and drugs targeting intracellular signaling cascades).

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Data availability statement

Data sharing is not applicable to this article as no new datasets were generated or analyzed during the current study. Any inquiries regarding the literature selection may be directed to the corresponding author upon reasonable request.

References

[1] Kidney Disease: improving Global Outcomes (KDIGO) Lupus Nephritis Work Group. KDIGO 2024 Clinical Practice Guideline for the management of LUPUS NEPHRITIS. Kidney Int. 2024;105(1S):S1–S69. doi: 10.1016/j.kint.2023.09.002.
[2] Fanouriakis A, Kostopoulou M, Cheema K, et al. 2019 Update of the Joint European League Against Rheumatism and European Renal Association-European Dialysis and Transplant Association (EULAR/ERA-EDTA) recommendations for the management of lupus nephritis. Ann Rheum Dis. 2020;79(6):713–723. doi: 10.1136/annrheumdis-2020-216924.
[3] Norouzi S, Contreras G. KDOQI US Commentary on the KDIGO 2024 Clinical Practice Guideline for the Management of Lupus Nephritis. Am J Kidney Dis. 2026;88(2):179–195. doi: 10.1053/j.ajkd.2026.05.002.
[4] Beck LH, Jr, Ayoub I, Caster D, et al. KDOQI US Commentary on the 2021 KDIGO Clinical Practice Guideline for the Management of Glomerular Diseases. Am J Kidney Dis. 2023;82(2):121–175. doi: 10.1053/j.ajkd.2023.02.003.
[5] Calatroni M, Conte E, Stella M, et al. Clinical and immunological biomarkers can identify proliferative changes and predict renal flares in lupus nephritis. Arthritis Res Ther. 2025;27(1):72. doi: 10.1186/s13075-025-03536-5.
[6] Bomback AS. Nonproliferative forms of lupus nephritis: an overview. Rheum Dis Clin North Am. 2018;44(4):561–569. doi: 10.1016/j.rdc.2018.06.003.
[7] Collado MV, Dorado E, Rausch S, et al. Long-term outcome of lupus nephritis class II in argentine patients: an open retrospective analysis. J Clin Rheumatol. 2016;22(6):299–306. doi: 10.1097/RHU.0000000000000395.
[8] Pakozdi A, Pyne D, Sheaff M, et al. Utility of a repeat renal biopsy in lupus nephritis: a single centre experience. Nephrol Dial Transplant. 2018;33(3):507–513. doi: 10.1093/ndt/gfx019.
[9] Narváez J, Ricse M, Gomà M, et al. The value of repeat biopsy in lupus nephritis flares. Medicine (Baltimore). 2017;96(24):e7099. doi: 10.1097/MD.0000000000007099.
[10] Pagni F, Galimberti S, Goffredo P, et al. The value of repeat biopsy in the management of lupus nephritis: an international multicentre study in a large cohort of patients. Nephrol Dial Transplant. 2013;28(12):3014–3023. doi: 10.1093/ndt/gft272.
[11] Alsuwaida AO. The clinical significance of serial kidney biopsies in lupus nephritis. Mod Rheumatol. 2014;24(3):453–456. doi: 10.3109/14397595.2013.844293.
[12] Greloni G, Scolnik M, Marin J, et al. Value of repeat biopsy in lupus nephritis flares. Lupus Sci Med. 2014;1(1):e000004. doi: 10.1136/lupus-2013-000004.
[13] Alsuwaida AO, Bakhit AA, Alsuwaida FA, et al. The long-term outcomes and histological transformation in class II lupus nephritis. Saudi Med J. 2018;39(10):990–993. doi: 10.15537/smj.2018.10.22435.
[14] Yang J, Liang D, Zhang H, et al. Long-term renal outcomes in a cohort of 1814 Chinese patients with biopsy-proven lupus nephritis. Lupus. 2015;24(14):1468–1478. doi: 10.1177/0961203315593166.
[15] Wang S, Chen D, Zuo K, et al. Long-term renal outcomes of mesangial proliferative lupus nephritis in Chinese patients. Clin Rheumatol. 2022;41(2):429–436. doi: 10.1007/s10067-021-05909-y.
[16] Feehally J, Floege J. Immunoglobulin A nephropathy and IgA vasculitis (Henoch-Schönlein Purpura). In: Johnson RJ, Feehally J, Floege J, Tonelli M, editors. Comprehensive Clinical Nephrology. 6 th ed. Philadelphia (PA): Elsevier; 2018. p. 273–275.
[17] Haas M. IgA nephropathy and IgA Vasculitis (Henoch-Schönlein Purpura) Nephritis. In: Jennette JC, Olson JL, Silva FG, D'Agati VD. editors. Heptinstall's Pathology of the kidney. 7th ed. Philadelphia (PA): Lippincott Williams & Wilkins; 2014. p. 854–884.
[18] Bajema IM, Wilhelmus S, Alpers CE, et al. Revision of the International Society of Nephrology/Renal Pathology Society classification for lupus nephritis: clarification of definitions, and modified National Institutes of Health activity and chronicity indices. Kidney Int. 2018;93(4):789–796. doi: 10.1016/j.kint.2017.11.023.
[19] Sethi S, De Vriese AS, Fervenza FC. Acute glomerulonephritis. Lancet. 2022;399(10335):1646–1663. doi: 10.1016/S0140-6736(22)00461-5.
[20] van Schaik M, van Kooten C, Arnaud L, European Renal Association (ERA) Immunonephrology Working Group (IWG)., et al. Disease modification in lupus nephritis: towards a pathophysiology-based treatment paradigm. Nat Rev Nephrol. 2026;22(10):684–703. Jul 23. Epub ahead of print. doi: 10.1038/s41581-026-01103-y.
[21] Zhang S, Magder L, Goldman D, et al. Clinical, histologic, and serological predictors of renal function loss in lupus nephritis. Arthritis Care Res (Hoboken). 2026; Apr 28. Epub ahead of print. doi: 10.1002/acr.80070.

[22] Kidney Disease: improving Global Outcomes (KDIGO) IgAN and IgAV Work Group; Rovin BH, Barratt J, Cook HT, et al. KDIGO 2025 Clinical Practice Guideline for the Management of Immunoglobulin A Nephropathy (IgAN) and Immunoglobulin A Vasculitis (IgAV). Kidney Int. 2025;108(4S):S1–S71. doi: 10.1016/j.kint.2025.04.004.
[23] Rajasekaran A, Rizk DV, Campbell KN. Immunoglobulin A nephropathy: new treatment options. Annu Rev Med. 2026;77(1):267–279. doi: 10.1146/annurev-med-043024-015024.
[24] Wu M, Cheng Y, Zhao X, et al. Efficacy and safety of belimumab in IgA nephropathy: real-world evidence. Nephrol Dial Transplant. 2026;Mar 23:gfag067. Epub ahead of print. doi: 10.1093/ndt/gfag067.
[25] Desai NB, Gashti C, Whittier WL. "A Change Is Gonna Come" to Treatment of Lupus Nephritis: a Review. Am J Kidney Dis. 2026;87(3):422–432. doi: 10.1053/j.ajkd.2025.08.016.
[26] Ugarte-Gil MF, Hanly J, Urowitz M, et al. Remission and low disease activity (LDA) prevent damage accrual in patients with systemic lupus erythematosus: results from the Systemic Lupus International Collaborating Clinics (SLICC) inception cohort. Ann Rheum Dis. 2022;81(11):1541–1548. doi: 10.1136/ard-2022-222487.
[27] Rua-Figueroa Fernández de Larrinoa Í, Lozano MJC, Fernández-Cid CM, et al. Preventing organ damage in systemic lupus erythematosus: the impact of early biological treatment. Expert Opin Biol Ther. 2022;22(7):821–829. doi: 10.1080/14712598.2022.2096406.
[28] Fanouriakis A, Kostopoulou M, Anders HJ, et al. EULAR recommendations for the management of systemic lupus erythematosus with kidney involvement: 2025 update. Ann Rheum Dis. 2026;85(1):75–90. doi: 10.1016/j.ard.2025.09.007.
[29] Edgecomb AG, Huang SP, Averell C, et al. Disease severity and healthcare costs associated with chronic kidney disease in patients with systemic lupus erythematosus. Clin Ther. 2026;48(2):138–147. doi: 10.1016/j.clinthera.2025.12.015.
[30] Kidney Disease: improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 Clinical Practice Guideline for the evaluation and management of chronic kidney disease. Kidney Int. 2024;105(4S):S117–S314. doi: 10.1016/j.kint.2023.10.018.
[31] Yu Z, Lin Y. Risk of infection for different B-cell targeting agents in treating systemic lupus erythematosus: A systematic review and network meta-analysis. Autoimmun Rev. 2026;25(2):103987. doi: 10.1016/j.autrev.2026.103987.
[32] Zhao Y, Chi G, Xia S, et al. Infectious complications of Belimumab with standard care in systemic lupus erythematosus: a systematic review and meta-analysis. Semin Arthritis Rheum. 2025;73:152754. doi: 10.1016/j.semarthrit.2025.152754.
[33] Ma X, Wang Y, Liang Y, et al. Efficacy and safety of telitacicept in systemic lupus erythematosus. Curr Pharm Des. 2026;May 19. Epub ahead of print. doi: 10.2174/0113816128420479251218010807.