Complement inhibitors and B cell–modifying agents for IgA nephropathy—a Kidney Disease: Improving Global Outcomes (KDIGO) commentary

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Kidney Disease: Improving Global Outcomes (KDIGO) updated its clinical practice guideline for the management of glomerular diseases in 2021, more than a decade after the first glomerular diseases guideline was published, reflecting slow progress in drug development. But since then, novel therapies for several glomerular diseases have been successfully tested and approved by regulatory agencies, none more so than IgA nephropathy (IgAN). To keep pace with new therapies, the IgAN guideline was updated again in 2025. After this revision came to press, 3 additional IgAN treatments received accelerated approval by the US Food and Drug Administration. Because the presumptive mechanisms of action of 2 of these new therapies are mechanistically different from those of previously approved drugs, the KDIGO IgAN Work Group felt that a brief commentary outlining where the new therapies may fit into the overall IgAN treatment strategy was warranted in lieu of a full guideline update, pending additional evidence for these and other therapies.

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Kidney Disease: Improving Global Outcomes (KDIGO) updated its clinical practice guideline for the management of glomerular diseases in 2021, more than a decade after the first glomerular diseases guideline was published,[1] reflecting slow progress in drug development. But since then, novel therapies for several glomerular diseases have been successfully tested and approved by regulatory agencies, none more so than IgA nephropathy (IgAN). To keep pace with new therapies, the IgAN guideline was updated again in 2025.[2] After this revision came to press, 3 additional IgAN treatments received accelerated approval by the US Food and Drug Administration. Because the presumptive mechanisms of action of 2 of these new therapies are mechanistically different from those of previously approved drugs, the KDIGO IgAN Work Group felt that a brief commentary outlining where the new therapies may fit into the overall IgAN treatment strategy was warranted in lieu of a full guideline update, pending additional evidence for these and other therapies. Beyond the drugs discussed in this update, there are several additional drugs in phase III clinical trials. These include B-cell modulators: povetacicept, telitacicept, and zigakibart; plasma cell depleters: felzartamab and mezagitamab; complement inhibitors: ravulizumab and sefaxersen; an endothelin receptor antagonist, zibotentan; a nonsteroidal mineralocorticoid receptor antagonist, finerenone; and an aldosterone synthase inhibitor, vicadrostat.

Shortly after the 2025 IgAN update was published, atrasentan was granted accelerated approval for the treatment of adults with primary IgAN at risk of rapid disease progression, generally considered as urine protein-to-

creatinine ratio (uPCR) ≥ 1.5 g/g. Approval was based on the results of the phase 3 ALIGN trial (Atrasentan in Patients With IgA Nephropathy, ClinicalTrials.gov Identifier: NCT04573478).[3] Like sparsentan, atrasentan is an endothelin type A receptor antagonist. Unlike sparsentan, atrasentan does not block the angiotensin receptor, and as such, in ALIGN it was added on top of a renin-angiotensin system inhibitor. After 36 weeks of atrasentan treatment, participants receiving the treatment demonstrated a 38.1% reduction in uPCR from baseline compared with a 3.1% reduction in uPCR in participants receiving placebo, a significant treatment difference of 36.1% ($P < 0.001$) favoring atrasentan.[3] Except for mildly increased fluid retention in patients on atrasentan, adverse events did not differ substantially between the 2 groups. The ALIGN trial is ongoing to determine whether atrasentan will also slow the decline in estimated glomerular filtration rate (eGFR) of people with IgAN compared with a renin-angiotensin system inhibitor alone. Atrasentan will fit into the IgAN treatment framework at a similar position as sparsentan (Figure 1).

Considerable evidence suggests a role for the complement system, especially the alternative pathway, in mediating inflammatory glomerular damage in IgAN, but complement may also be important for chronic progressive loss of kidney function through profibrotic activities in the tubulointerstitial compartment.[4] A role of complement in IgAN was tested in the phase 3 APPLAUSE-IgAN trial (Study of Efficacy and Safety of LNP023 in Primary IgA Nephropathy Patients, ClinicalTrials.gov Identifier: NCT04578834) using iptacopan, an oral complement factor B inhibitor that blocks the alternative complement pathway.[5] After 36 weeks of treatment, participants receiving iptacopan had uPCR 38.3% lower than that of participants treated with placebo ($P < 0.001$).[5] This led to the accelerated approval of iptacopan for adults with primary IgAN at risk of rapid disease progression in the United States. Very recently, the completed APPLAUSE-IgAN trial was

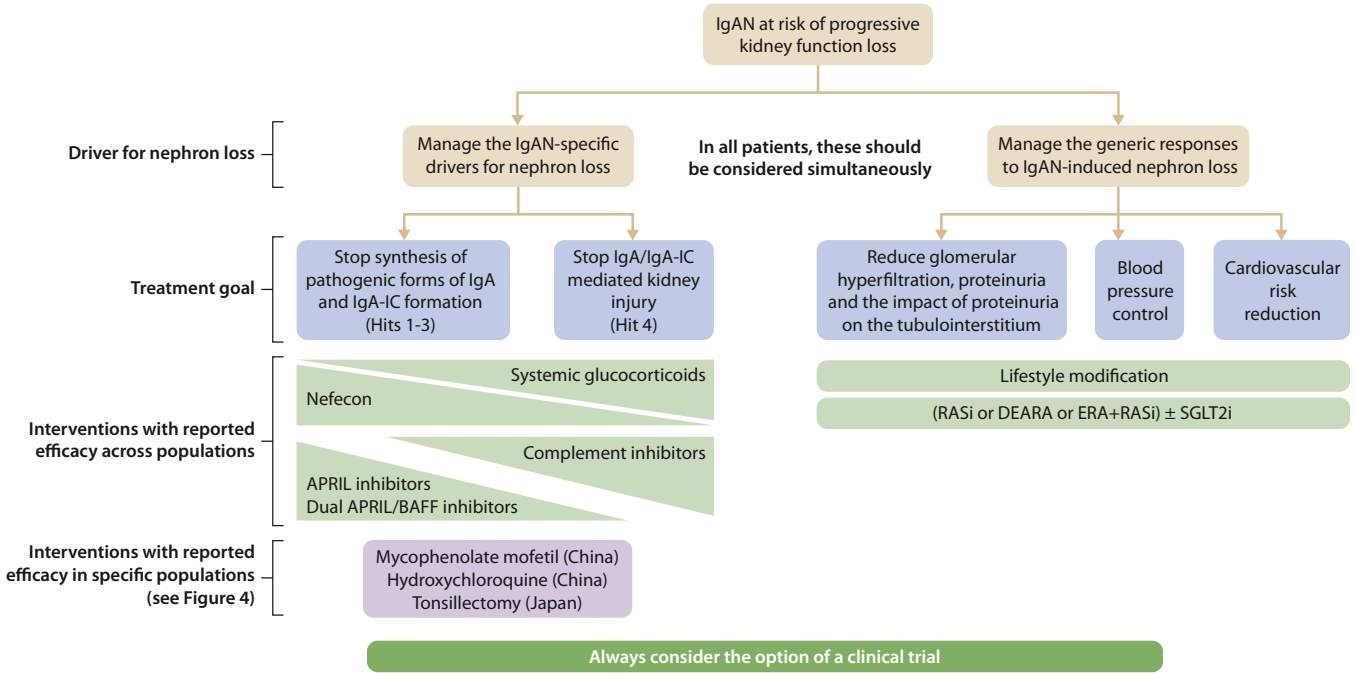

Figure 1 | Therapeutic targets and positioning of treatments in IgA nephropathy (IgAN). Reflecting our latest understanding, treatments should target IgAN-specific drivers for nephron loss (hits 1–3 and hit 4) and generic response to IgAN-induced nephron loss. Drugs targeting the IgAN-specific drivers for nephron loss likely act on different mechanisms of the 4-hit hypothesis. Triangles denote that a drug class most likely exerts its key actions on either hits 1–3 or hit 4 but may target all hits to a variable extent, as indicated by their lengths of the triangle. Nefecon, APRIL (proliferation-inducing ligand) inhibitors, and dual APRIL/BAFF (B-cell activating factor) inhibitors are shown as having a predominant effect on the production of pathogenic forms of IgA and IgA-containing immune complexes (IgA-ICs) (hits 1–3), whereas systemic glucocorticoids and complement inhibitors are shown as having a predominant anti-inflammatory effect on IgA-IC–mediated kidney injury (hit 4). Interventions to manage the generic response to IgAN-induced nephron loss include lifestyle modification and renin-angiotensin system inhibitor (RASi), dual endothelin angiotensin receptor antagonist (DEARA), or a combination of endothelin type A receptor antagonist (ERA) and RASi, with or without sodium-glucose cotransporter-2 inhibitor (SGLT2i). Additional strategies may also include the use of 3-hydroxy-3-methyl-glutaryl-coenzyme A reductase inhibitors in select patients. The order of drugs listed here does not imply a recommended ranking or delivery sequence.

presented, and iptacopan was found to decrease annual eGFR loss from 6.12 ml/min per 1.73 m^2 to 3.10 ml/min per 1.73 m^2 ($P < 0.001$).[6] Consideration for full regulatory approval is underway. One of the major concerns with complement inhibition is increased susceptibility to infections with encapsulated bacteria, thus mandating vaccination or prophylactic antibiotics before iptacopan administration. This occurred in fewer than 0.5% of participants in the trial, and all recovered with appropriate antibiotic treatment.[5,6] Treatment with iptacopan did not result in an imbalance of safety relative to placebo.

The most recent addition to the therapeutic choices for IgAN is sibeprenlimab, a humanized monoclonal antibody against the B-cell survival factor, "a proliferation inducing ligand" (APRIL). Sibeprenlimab received accelerated approval by the US Food and Drug Administration for adults with primary IgAN at risk of disease progression (no uPCR criteria) in light of the results of the phase 3 VISIONARY trial (Phase 3 Trial of Sibeprenlimab in Immunoglobulin A Nephropathy, ClinicalTrials.gov Identifier: NCT05248646).[7] At 40 weeks, participants treated with sibeprenlimab had a 50.2% reduction in uPCR compared to a 2.1% increase in the placebo group. The effect size favoring sibeprenlimab was a 51.2% decline in proteinuria ($P < 0.001$).[7] Anti-APRIL, as a therapy acting directly on B cells, was expected to reduce the production of pathogenic forms of IgA. Pharmacodynamic studies in VISIONARY demonstrated that galactose-deficient IgA1 (Gd-IgA1) fell by close to 70% in participants treated with sibeprenlimab and mirrored an 68.8% fall in total IgA. IgM fell by 74.5% and IgG by 35.0%.[7] Adverse events were similar between placebo and sibeprenlimab. About 8% of participants treated with sibeprenlimab developed antidrug antibodies that had neutralizing capability. Proteinuria reduction in participants who developed anti-drug antibodies was slightly lower than in those who did not develop antidrug antibodies (−42% vs. −53%), but whether that is clinically relevant remains to be determined. The VISIONARY trial is ongoing to determine the effect of sibeprenlimab on eGFR decline.

Another phase 3 trial using a B-cell therapeutic, atacicept, was also recently completed and is undergoing US Food and Drug Administration review. Atacicept is a fusion protein of the transmembrane activator and calcium modulator and cyclophilin ligand interactor receptor and IgG. Transmembrane activator and calcium modulator and cyclophilin ligand interactor is a B-cell surface receptor for both APRIL and B-cell activating factor (BAFF), so atacicept blocks the activity of both these B-cell survival factors. APRIL is important in plasma cell and plasmablast differentiation, whereas BAFF mediates B-cell survival and maturation. Blocking APRIL selectively reduces IgA production (relative to IgG) and blocking BAFF reduces total immunoglobulins. Therapeutically targeting both BAFF and APRIL was postulated to be more effective for treating IgAN than blocking APRIL alone. To this end, atacicept was tested in the ORIGIN 3 study (Atacicept in Subjects With IgA Nephropathy, ClinicalTrials.gov Identifier: NCT04716231) and reduced uPCR by 45.7% at week 36 compared with 6.8% in participants who were given placebo, with an overall effect size favoring atacicept of 41.8% ($P < 0.001$).[8] Gd-IgA1 fell by 68% at 36 weeks in the atacicept arm, consistent with the fall in total IgA by 63.5%, IgM by 74.6%, and IgG by 35.5%.[8] Adverse events were similar between placebo and atacicept. The ORIGIN trial is ongoing to determine the effect of atacicept on eGFR decline. Nonetheless, at present, it is difficult to differentiate between selective APRIL and dual BAFF/APRIL antagonists in the efficacy and safety of IgAN treatment.

When considering all the drugs available for IgAN, it is difficult to choose a first-line therapy and determine its optimal treatment duration. All the approved drugs reduce proteinuria and mitigate eGFR decline significantly better than a renin-angiotensin system inhibitor alone (Table 1), but without head-to-head comparisons, it is difficult to rank therapies by the extent of proteinuria reduction or eGFR preservation, and as such, Table 1 is not intended to suggest that the different trial results should be directly compared. Phase 3 data for the effects of atrasentan and sibeprenlimab on eGFR are pending, but phase 2 trial data for sibeprenlimab and atacicept suggest that these stabilize eGFR very well.[9,10] Other factors that will drive choice of therapy for IgAN will be availability and cost. But some general concepts on approaching treatment can be articulated. Starting with the 4-hit hypothesis of IgAN pathogenesis, it is reasonable to match drugs by their presumptive mechanisms of action to the hits they target (Figure 1). Hits 1–3 describe the origin of circulating pathogenic IgA, the formation of IgA-containing immune

Table 1 | Phase 3 proteinuria and eGFR data for available and emerging IgAN therapeutics (as of April 15, 2026)

	Mean change (%) from baseline in 24-hr uPCR (g/g) at 9 mo		Annual rate of eGFR decline (ml/min per 1.73 m^2)	
Therapeutic	Treatment arm	Placebo arm	Treatment arm	Placebo arm
Methylprednisolone	−52[a]	−4[a]	−2.5[b]	−4.9[b]
Nefecon	−31	−5	−3.6[b,c]	−5.4[b,c]
Sparsentan	−50	−15	−2.9[b]	−3.9[b]
Atrasentan	−38	−3	NA	NA
Iptacopan	−45	−8	−3.1	−6.1
Sibeprenlimab	−50	2	NA	NA
Atacicept	−46	−7	NA	NA

eGFR, estimated glomerular filtration rate; IgAN, IgA nephropathy; NA, not available; uPCR, urine protein-to-creatinine ratio.
[a]Change from baseline 24-hr urine protein.
[b]Total slope.
[c]Calculated using the primary supportive random coefficient analysis.

complexes or immune aggregates, and the localization of these immune complexes or aggregates to the glomerular mesangium. At present, pathogenic IgA is often equated to Gd-IgA1 but this is likely an over-simplification, and the specific pathogenic forms of IgA still need to be defined. Nonetheless, drugs that decrease the production of Gd-IgA1 or total IgA should be effective in addressing hits 1–3. In contrast, hit 4 describes the effects of pathogenic IgA deposits on the kidney and the myriad of injury pathways activated in response to their accumulation in the mesangium; drugs that decrease glomerular inflammation or block complement should be effective in hit 4. Figure 1, which is a modified version of Figure 3 from the 2025 KDIGO IgAN guideline update, matches drug classes with their expected pathogenic targets. Some caveats regarding Figure 1 should be considered. The matching of drug to target focuses on the predominant effects of a specific therapeutic, but all these drugs have secondary actions that may be beneficial. For example, endothelin type A receptor antagonist/dual endothelin angiotensin receptor antagonist may also have anti-inflammatory properties. Certain individuals may benefit from combinations of the new therapeutics, but so far there are no data on combining these drugs, and as such, combinations are not emphasized in Figure 1. Finally, the order of drugs listed does not imply a recommended ranking or sequence of the delivery of these therapies regarding their efficacy or safety. Many of these IgA drugs will likely be used long-term, either continuously or in cycles. Although long-term BAFF inhibition in immune-mediated diseases is known to be well-tolerated, there are no similar safety data for APRIL or BAFF/APRIL inhibition.[11,12] These considerations also apply to long-term complement inhibition, although chronic complement inhibition, in particular C5 antagonism, has a good safety profile in atypical hemolytic-uremic syndrome, paroxysmal nocturnal hemoglobinuria, and C3 glomerulopathy.[13–15]

Figure 1 also suggests that hits 1–3 and chronic kidney disease (CKD) should be addressed simultaneously. The rationale for treating CKD early in IgAN is that many people with IgAN already have moderately advanced CKD by the time they are diagnosed, but this should be individualized. Given that some of the injury mechanisms in progressive CKD are similar to injury mechanisms initiated in hit 4, the separation of hit 4 from CKD may be somewhat artificial but this is helpful for organizing a therapeutic strategy. A notable finding of many phase 3 trials in IgAN is that, within the confines of a clinical trial, all subgroups of people with IgAN (ethnic, eGFR, proteinuria, and mesangial [M] and endocapillary [E] hypercellularity, segmental sclerosis [S], interstitial fibrosis/tubular atrophy [T], and crescents [C] [MEST-C] score) seemed to benefit equally from the intervention, be it endothelin-1, complement, or B-cell growth factor antagonism.[3,5,7,8] This observation supports the KDIGO recommendation to use biopsy findings for prognostic purposes only and not for therapeutic decision making.

We anticipate that the schema in Figure 1 will evolve and be regularly updated to include new drugs or drug categories that become approved. As more clinical trial, real-world data, and validated biomarker data become available, it may be possible to recommend particular therapies or combinations of therapy as ideal first-line approaches. Finally, the

schema in Figure 1 will become further refined as pathogenic forms of IgA are more precisely defined, perhaps under the umbrella of Gd-IgA1, and as biomarkers become available to track pathogenic hits and the response of the kidneys to injury and treatment.

Given the rapid pace of new development in IgAN therapeutics, this update is provided as a stopgap to a full update to the KDIGO IgAN guideline. Many important issues have not been discussed here, for example, (i) the increasing evidence that low levels of proteinuria are associated with worse prognosis in IgAN, (ii) implications of hematuria, (iii) maintenance therapy, (iv) combination therapy, (v) duration of therapy, and (vi) therapy switching. These issues and more will be tackled in the next formal update of the KDIGO IgAN guideline.

DISCLOSURE

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DISCLAIMER

This commentary purposefully does not make recommendations or include practice points. These will be contained in the next guideline update.

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