Executive summary of the KDIGO 2025 Clinical Practice Guideline for the Management of Immunoglobulin A Nephropathy (IgAN) and Immunoglobulin A Vasculitis (IgAV)

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The Immunoglobulin A nephropathy (IgAN) and Immunoglobulin A vasculitis (IgAV) management guideline was last updated and published as part of the Kidney Disease: Improving Global Outcomes (KDIGO) 2021 Clinical Practice Guideline for the Management of Glomerular Diseases. Based on new developments in IgAN assessment and therapy, a major update of the guideline was necessary in 2024. Compared with the last version, the KDIGO 2025 IgAN guideline now encourages a more liberal kidney biopsy policy and suggests aiming for stricter proteinuria control, with a goal of <0.5 g/d, ideally <0.3 g/d, and a stable estimated glomerular filtration rate. A major new concept in the 2025 guideline is to initiate treatment with (i) therapies that prevent or reduce pathogenic IgA production and IgA/IgA and IgA/IgG immune complex formation along with (ii) therapies to manage the consequences of existing IgAN-induced nephron loss. Approaches to achieve the first aim are currently limited to targeted-release budesonide (Nefecon) or reduced-dose systemic corticosteroid therapy and, in Chinese patients, mycophenolate mofetil. Approaches to the more generic second aim include healthy lifestyle education, renin-angiotensin system blockers, sodium-glucose cotransporter-2 inhibitors, and/or dual endothelin angiotensin receptor blockers. Little has changed for special situations of IgA-dominant immune complex glomerular diseases such as nephrotic syndrome, acute kidney injury, rapidly progressive glomerulonephritis, and pregnancy in IgAN, or children with IgAN or IgAV, given the lack of major clinical trials in these patient populations. Here, we provide an executive summary of the most important changes in the KDIGO 2025 IgAN and IgAV guideline as a quick reference.

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The Kidney Disease: Improving Global Outcomes (KDIGO) organization published a major revision of the Clinical Practice Guideline for the Management of Glomerular Diseases in 2021.[1] In the case of IgA nephropathy (IgAN), a number of important new insights and successful major randomized controlled clinical trials were published after 2021, prompting the present guideline update (Supplementary Table S1).

For the first time, the nephrology community has novel and approved therapies for IgAN, with several more demonstrating benefit in advanced phase clinical trials. However, it should be clear that the trials thus far have not shown how best to use and, in particular, how best to combine these new tools for our patients. In one sense,

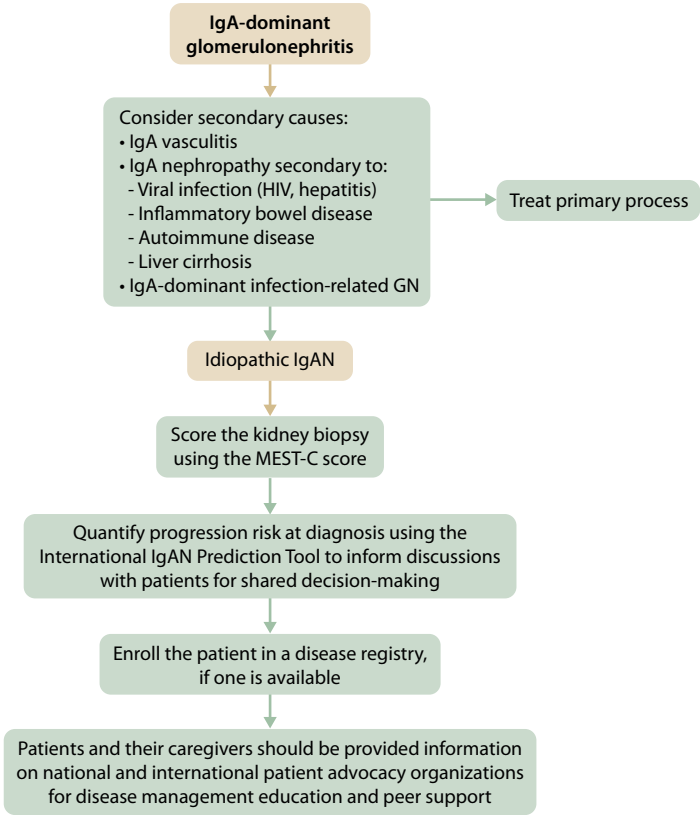

Figure 1 | Initial assessment and management of the patient with immunoglobulin A nephropathy (IgAN). GN, glomerulonephritis; HIV, human immunodeficiency virus; MEST-C, mesangial (M) and endocapillary (E) hypercellularity, segmental sclerosis (S), interstitial fibrosis/tubular atrophy (T), and crescents (C).

the updated guideline could have been very brief; at this time, it is reasonable to say that any of the approved therapies can be used in any patient with IgAN who has proteinuria and is at risk of progressive disease. We believe that this is a nihilistic point of view. These drugs have presumptive mechanisms of action that we think we understand, at least in part; so, in an effort to bring some order to the choice of therapies, this IgAN and IgA vasculitis (IgAV) guideline update takes some license to propose what we consider rational ways to use the new medications. We also propose that suggested combinations of medications targeting different points in the IgAN pathogenic pathways may be more effective and that maintenance immunologic treatment, as used in most other immune-mediated glomerular diseases, may be required. The idea of combination and maintenance therapies is reasonable because we have already seen that none of the newly approved drugs achieve the proteinuria or glomerular filtration rate goals that seem to be needed to prevent dialysis over the lifetime of a patient with IgAN. In addition, trial experience to date shows that when some medications are stopped, proteinuria quickly returns. As more data are gathered, this IgAN guideline will be appropriately updated, ideally moving toward a fully evidence-based set of recommendations.

DIAGNOSIS OF IgAN

As in the 2021 guideline, the 2025 IgAN and IgAV guideline notes that IgAN can only be diagnosed with a kidney biopsy, that there are no validated diagnostic serum or urine biomarkers for IgAN, that the mesangial (M) and endocapillary (E) hypercellularity, segmental sclerosis (S), interstitial fibrosis/tubular atrophy (T), and crescents (C) (MEST-C) score should be determined, and that secondary causes should be excluded (Figure 1). However, the 2025 guideline encourages a more liberal biopsy policy, noting that to ensure an early diagnosis and prompt treatment of IgAN, a kidney biopsy should be considered in all adults with proteinuria $\geq$0.5 g/d (or equivalent) in whom IgAN is suspected.

PROGNOSIS OF IgAN

Clinical and histologic data at the time of biopsy should be used to risk stratify patients by the International IgAN Prediction Tools, which are now available in different versions, depending on age (adult vs. children) and the time elapsed since the kidney biopsy. In addition to the lack of diagnostic biomarkers for IgAN, we do not have novel validated biomarkers for IgAN prognosis, except for estimated glomerular filtration rate (eGFR) and proteinuria.

The updated 2025 guideline again states that the International IgAN Prediction Tools and the Oxford Classification

MEST-C score have not been evaluated as a means of determining the likely impact of any particular treatment regimen and, at present, should not be used to decide on a specific treatment therapy. Histopathologic features, in particular crescents, must be interpreted in the context of clinical features, most importantly, the rate of change in eGFR.

TREATMENT OF IgAN

Aims of treatment and interventions for all patients with IgAN

The goal of treatment is to reduce the rate of kidney function loss and return it to the physiological state (i.e., <1 ml/min per year for most adults) for the rest of the patient's life. The only currently validated early biomarker to help guide clinical decision-making is urine protein excretion. Although the 2021 guideline recommended a treatment goal of proteinuria reduction to <1 g/d, there is now considerable evidence that lower levels of proteinuria or albuminuria are associated with a significant lifetime risk of kidney failure.[2–4] The proteinuria goal was therefore lowered to <0.5 g/d while on or off treatment of IgAN, ideally <0.3 g/d (or equivalent).

At the time of diagnosis, most patients with IgAN already present with more or less advanced, but certainly established, chronic kidney disease (CKD). A major new concept in the 2025 guideline is therefore a dual and concordant focus on treatments that aim to (i) prevent or reduce IgA-containing immune complex (IgA-IC) formation and IgA-IC–mediated glomerular injury and (ii) manage the consequences of existing IgAN-induced nephron loss (Figure 2). While it is currently unknown whether treating the immune aspect of IgAN requires lifelong or intermittent therapy, the CKD aspect will likely need lifelong treatment. Treating the immune aspect of IgAN should incorporate treatments that prevent or reduce IgA-IC formation with (or without) treatments that block IgA-IC–mediated glomerular injury.

Managing the consequences of existing IgAN-induced nephron loss includes nonpharmacologic interventions, where appropriate; all patients should receive extensive lifestyle advice on smoking and vaping cessation, weight control, dietary sodium restriction (<2 g/d), and regular exercise. Blood pressure should be controlled to a target of ≤120/70 mm Hg. Measures to reduce glomerular hyperfiltration and the impact of proteinuria on the tubulointerstitium include renin-angiotensin system (RAS) blockade or dual endothelin angiotensin receptor antagonism, with or without sodium-glucose cotransporter-2 inhibition.

Factors to consider when choosing a treatment and/or treatment combinations for patients with IgAN at risk of progressive loss of kidney function are presented in Table 1. Newly approved treatments are unlikely to be used or even available in resource-limited settings where cheaper and more easily resourced drugs have been and will continue to be used. A cardiovascular risk assessment should also be undertaken and interventions commenced as per local guidelines.

Where possible, enrollment in a clinical trial should be considered, realizing that trial design, particularly a placebo-controlled trial, will have to be changed in light of newly approved treatments for IgAN.

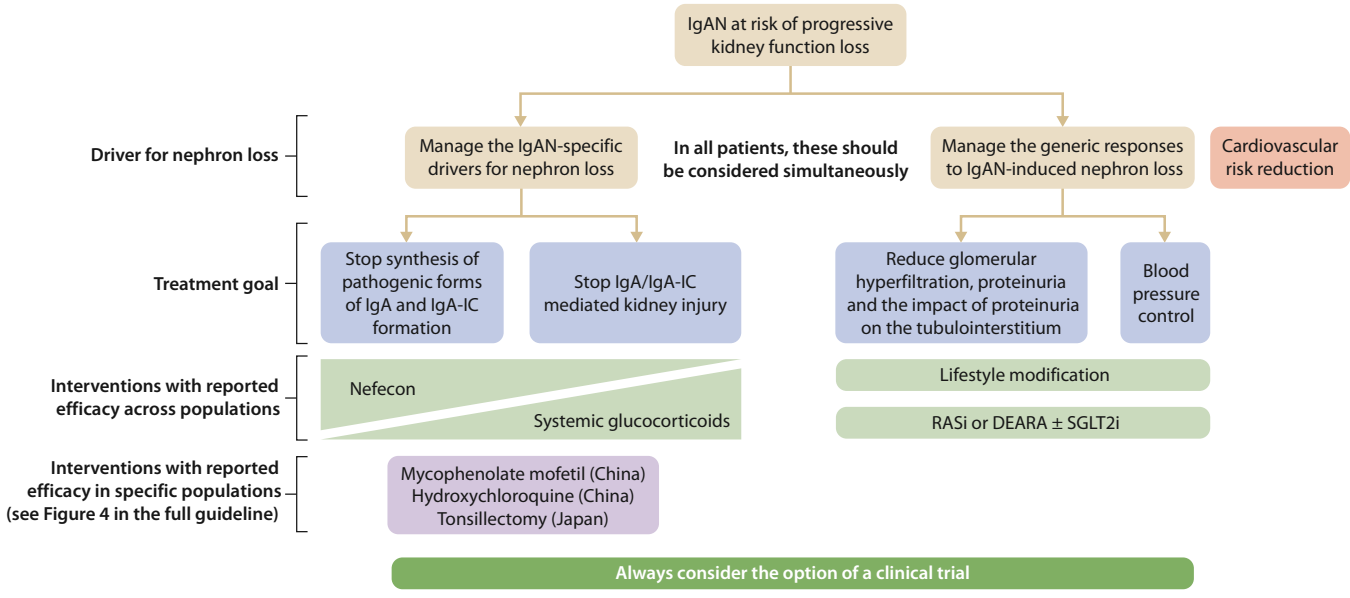

Figure 2 | Treatment targets in immunoglobulin A nephropathy (IgAN) and the positioning of drugs included in this guideline. Reflecting current understanding, Nefecon is shown as having a predominant effect on the production of pathogenic forms of IgA and IgA-containing immune complexes (IgA-ICs), with an undetermined direct effect of systemically absorbed budesonide on the kidneys. Systemic glucocorticoids have a well-documented anti-inflammatory effect within the kidneys and an undetermined direct effect on the production of pathogenic forms of IgA and IgA-ICs. Strategies to manage the generic response to IgAN-induced nephron loss may also include the use of 3-hydroxy-3-methyl-glutaryl-coenzyme A reductase inhibitors in selected patients. DEARA, dual endothelin angiotensin receptor antagonist; RASi, renin-angiotensin system inhibitor; SGLT2i, sodium-glucose cotransporter-2 inhibitor.

Table 1 | Factors to consider when choosing a treatment and/or treatment combinations for patients with IgAN at risk of progressive loss of kidney function

Question	Considerations
Is the clinical trial population in which the drug was tested representative of the patient being treated (see Table 2 in the full guideline)?	**Age:** In the trials of SGLT2i, patients were on average 6–8 years older than those recruited into the NefIgArd and PROTECT trials and 15–17 years older than those recruited into the STOP-IgAN and TESTING studies.
	Race: The TESTING study was almost exclusively conducted in Asian patients. STOP-IgAN was exclusively conducted in Caucasians. In the NefIgArd and PROTECT studies, Asian patients were relatively underrepresented compared with those in trials of SGLT2i and systemic glucocorticoids.
	eGFR: In the trials of SGLT2i, the average eGFR at inclusion was 12–14 ml/min per 1.73 m^2 lower than that of patients included in the NefIgArd, PROTECT, STOP-IgAN, and TESTING studies.
	Concomitant medications: In all recent studies in IgAN, patients were required to be on a stable optimized dose of RASi for 90 days prior to enrollment. An optimized maximally tolerated dose was not required in the trials of SGLT2i.
	Optimization of RAS blockade: The only trial to formally uptitrate RASi was the PROTECT trial. In the NefIgArd and TESTING studies, participants were required to be on locally physician-attested, optimized, maximally tolerated dose of RASi.
What is the labeled indication for the drug?	With the new drug approval pathway for IgAN, labeled indications may vary depending on the country and whether the drug has an accelerated approval or conditional market authorization, where assessment of efficacy has been made on the basis of proteinuria, or full approval, based on its effect on the rate of loss of kidney function.
What are the key advantages of the available treatment options?	Nefecon is the only treatment to date proven to reduce the levels of pathogenic forms of IgA and IgA-containing immune complexes (IgA-ICs).
	Systemic glucocorticoids are highly effective anti-inflammatory drugs but have no proven impact on levels of pathogenic forms of IgA or IgA-ICs at the doses recommended in this guideline.
	SGLT2i have been shown to not only reduce the rate of progressive loss of kidney function but also reduce the incidence of adverse cardiovascular events, particularly in people with diabetes. They are also generally well tolerated.
	The DEARA sparsentan is the only drug to have shown efficacy beyond the in-trial uptitrated RASi. Of note, more patients were included in the PROTECT trial than in all the trials of RASi in IgAN combined.
	RASi effectively reduce proteinuria and have extensive efficacy and safety data in CKD and cardiovascular disease.
What are the key risks of the available treatment options?	As there is some systemic absorption of budesonide, patients and healthcare providers should be aware of the possibility of some systemic glucocorticoid–related side effects with Nefecon. These are usually mild and reversible upon treatment cessation.
	When using systemic glucocorticoids, a reduced-dose protocol should be followed. Antimicrobial prophylaxis against *Pneumocystis jirovecii* and antiviral prophylaxis in hepatitis B carriers should be used, and the patient should be made aware of the risks of gastrointestinal bleeding, infection, and metabolic, cosmetic, and neuropsychiatric side effects, alongside the potential impact on bone health.
	As with all endothelin receptor antagonists, there is a significant risk of embryofetal toxicity, and women of childbearing potential must use a reliable form of contraception and undergo monthly pregnancy testing.

CKD, chronic kidney disease; DEARA, dual endothelin angiotensin receptor antagonist; eGFR, estimated glomerular filtration rate; IgAN, immunoglobulin A nephropathy; NefIgArd, Efficacy and Safety of Nefecon in Patients With Primary IgA Nephropathy; PROTECT, A Study of the Effect and Safety of Sparsentan in the Treatment of Patients With IgA Nephropathy; RAS, renin-angiotensin system; RASi, renin-angiotensin system inhibitor(s); SGLT2i, sodium-glucose cotransporter-2 inhibitor(s); STOP-IgAN, Supportive Versus Immunosuppressive Therapy for the Treatment of Progressive IgA Nephropathy; TESTING, Therapeutic Effects of Steroids in IgA Nephropathy Global.

Treating IgAN

Targeted-release budesonide (Nefecon) and systemic corticosteroid therapy. Treatment of the immunologic aspect of IgAN is necessary to induce remission of the disease (Figure 2). The new Recommendation 1.4.3.1 suggests, where approved, treatment with a 9-month course of Nefecon for patients who are at risk of progressive loss of kidney function with IgAN (*2B*). Given that a 9-month treatment course of Nefecon may not result in a sustained clinical response in terms of proteinuria reduction or eGFR stabilization, data on the safety and efficacy of extended or additional treatment courses are awaited.

In settings where Nefecon is not available, Recommendation 1.4.3.2 suggests that patients who are at risk of progressive loss of kidney function with IgAN be treated with a reduced-dose systemic glucocorticoid regimen combined with antimicrobial prophylaxis (*2B*). Based on the Therapeutic Effects of Steroids in IgA Nephropathy Global (TESTING) trial,[7,8] this therapy consists of methylprednisolone (or equivalent) 0.4 mg/

kg/d (maximum 32 mg/d) for 2 months followed by dose tapering by 4 mg/d each month for a total of 6–9 months. Treatment should be combined with antimicrobial prophylaxis against *Pneumocystis jirovecii* and antiviral prophylaxis in hepatitis B carriers, along with gastroprotection and bone protection according to national guidelines.

As before, the updated guideline notes that the risk of toxicity of systemic glucocorticoids increases in patients with eGFR <30 ml/min per 1.73 m^2, diabetes and prediabetes, obesity, latent infections (e.g., viral hepatitis and tuberculosis), active peptic ulceration, uncontrolled psychiatric illness, osteoporosis, or cataracts.

Other treatments evaluated in patients with IgAN. As in the 2021 guideline, the 2025 guideline does not recommend treatment of IgAN with antiplatelet agents, anticoagulants, azathioprine, cyclophosphamide (other than in the setting of rapidly progressive IgAN), calcineurin inhibitors, rituximab, or fish oil. There is insufficient evidence to support the use of mycophenolate mofetil in non-Chinese patients, whereas in Chinese patients, where trials have been conducted, it can be considered as a glucocorticoid-sparing agent. Similarly, in Chinese patients, hydroxychloroquine may be considered, but there is insufficient evidence to support its use in other ethnicities. Finally, tonsillectomy is recommended in Japan but not in other countries.

Treating IgAN-associated CKD

RAS blockade. As before, blood pressure should be lowered to ≤120/70 mm Hg by using a RAS inhibitor (RASi) as the first-choice drug intervention. More generally, the 2025 guideline recommends that all patients with IgAN be treated with an optimized maximally tolerated dose of either an angiotensin-converting enzyme inhibitor (ACEi) or an angiotensin II receptor blocker (ARB) (Recommendation 1.4.4.1; *1B*). Exceptions include patients with contraindications such as low blood pressure, bilateral renal artery stenosis, or hyperkalemia, especially from advanced CKD. RAS blockade should not preclude the concomitant introduction of therapies that target the drivers of IgAN or glomerular inflammation for patients who will likely benefit from them.

Dual endothelin-1 and angiotensin II receptor blockade. The new Recommendation 1.4.4.2 suggests that, where approved, patients who are at risk of progressive loss of kidney function with IgAN be treated with sparsentan (*2B*), a dual endothelin angiotensin receptor antagonist. Sparsentan should replace, rather than being prescribed together with, a RASi.

Sodium-glucose cotransporter-2 inhibition. The new Recommendation 1.4.4.3 suggests that patients who are at risk of progressive loss of kidney function with IgAN be treated with a sodium-glucose cotransporter-2 inhibitor (SGLT2i) (*2B*). Important caveats include that there was no requirement for patients with IgAN to be on an optimized maximally tolerated dose of RASi for a minimum of 3 months in the 2 landmark trials of SGLT2i in CKD that included significant numbers of patients with IgAN.[5,6] In addition, patients with IgAN in these 2 trials had long-standing disease and low eGFR, resulting in uncertainty of the benefit of SGLT2i, especially in younger patients with IgAN and those with eGFR >60 ml/min per 1.73 m^2.

Special situations

Very little has changed in this section of the guideline compared with the 2021 version (Supplementary Table S1). A brief synopsis of special situations is provided here for completeness. A review of the treatment of recurrent IgAN will be included in the next update as more data become available.

Nephrotic syndrome. In the rare patient with IgAN and nephrotic syndrome, it is often unclear whether nephrotic syndrome is a specific variant of IgAN (with a mesangioproliferative pattern of glomerular injury) or the coexistence of minimal change disease (MCD) in a patient with IgAN (mesangial IgA deposition and light and electron microscopy features otherwise consistent with MCD). The former should be treated like other patients with IgAN, and the latter should receive treatment analogous to that of MCD.

Nephrotic-range proteinuria without nephrotic syndrome commonly reflects coexistent secondary focal segmental glomerulosclerosis or the development of extensive global glomerulosclerosis and tubulointerstitial fibrosis (due to, e.g., chronic damage resulting from IgAN, obesity, or uncontrolled hypertension).

Rapidly progressive IgAN. Rapidly progressive IgAN is defined as a ≥50% decline in eGFR over ≤3 months, where other causes of rapidly progressive glomerulonephritis and acute kidney injury have been excluded. A kidney biopsy generally shows extensive glomerular crescent formation. Patients with rapidly progressive IgAN should be offered treatment with cyclophosphamide and systemic glucocorticoids in accordance with the KDIGO 2024 Clinical Practice Guideline for the Management of Antineutrophil Cytoplasmic Antibody (ANCA)–Associated Vasculitis. There is insufficient evidence to support the use of rituximab for the treatment of rapidly progressive IgAN.

Acute kidney injury. Acute kidney injury may occur in rapidly progressing IgAN, but often accompanies disease flares with visible hematuria, in which case treatment should focus on supportive care with consideration for a repeat kidney biopsy if there is no improvement in kidney function within 2 weeks of cessation of hematuria.

Childbearing. IgAN is a disease predominantly of young adults, and all women of childbearing potential should be offered preconception counseling, and discontinue RASi, SGLT2i, sparsentan, and Nefecon before conceiving. Blood pressure control should be optimized with pregnancy-compatible antihypertensive medications prior to conception. In women at risk of progressive loss of kidney function, a trial of treatments to optimally address the immunopathogenesis of IgAN prior to conception may be desirable in an attempt to get the disease under optimal control. Systemic glucocorticoids may be used during pregnancy, but the use of Nefecon in pregnancy is currently not advised.

Pediatric IgAN. In 2025, the International Pediatric Nephrology Association (IPNA) developed clinical practice recommendations for the management of IgAN and IgAV in children.[9] The KDIGO 2025 Guideline for the Management of IgAN and IgAV has harmonized its guidance with that of the IPNA recommendations. It is clear from a review of the pediatric guideline that evidence is derived mostly from retrospective studies and observational data, suggesting a large unmet need for more robust data in pediatric patients with IgAN and IgAV.

IgA vasculitis

While considerable progress has been made in the treatment of IgAN, these drugs have not yet been tested in IgAV. While it is certainly tempting to believe that some or all of the drugs approved or in trials in IgAN may be used for IgAV, this remains to be demonstrated. One important recommendation we have carried forward that is worth repeating is Recommendation 1.9.1.1, which recommends not using systemic glucocorticoids to prevent nephritis in patients with isolated extrarenal IgAV (*1B*).

CONCLUSION

It is now clear that IgAN, once considered a slow-moving glomerular disease with a generally good prognosis, is more often relentlessly progressive and will result in the need for kidney replacement therapy in most patients if not adequately treated. Ideally, treatment should aim to return the lifetime trajectory of kidney function loss to the physiological state. The past several years have seen a surge of new IgAN therapies, either approved or in late-phase clinical trials. These therapies address various steps in the pathogenesis of IgAN and are mechanistically unique, providing the opportunity for synergistic combinations to achieve the newly articulated goals of treatment to preserve kidney function for a patient's lifetime. As more therapies become available for clinical use, the KDIGO Work Group looks forward to further updating this guideline.

DISCLOSURE

The development and publication of this guideline were supported by Kidney Disease: Improving Global Outcomes (KDIGO). The opinions or views expressed in this summary are those of the authors and do not necessarily reflect the opinions or recommendations of the International Society of Nephrology or Elsevier. Dosages, indications, and methods of use for products that are referred to in the supplement by the authors may reflect their clinical experience or may be derived from the professional literature or other clinical sources. Because of the differences between *in vitro* and *in vivo* systems and between laboratory animal models and clinical data in humans, *in vitro* and animal data do not necessarily correlate with clinical results.

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Supplementary material is available online at www.kidney-international.org.

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