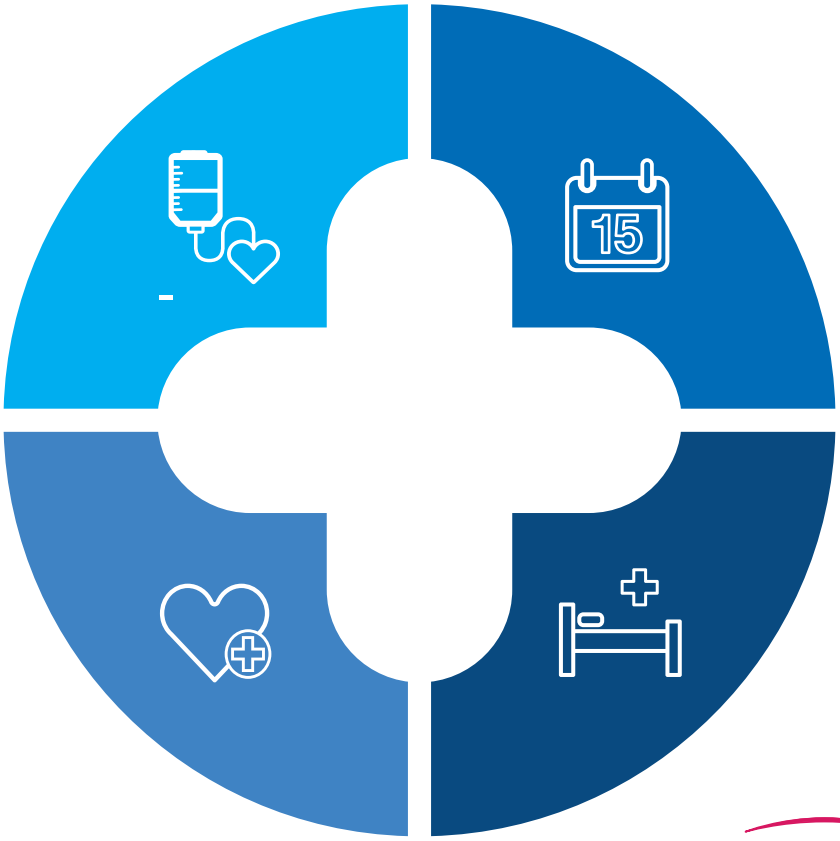
Treating immunoglobulin A nephropathy (IgAN): a guide



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UK-KINPE-214 | February 2026

This guide outlines up-to-date approaches to identifying, managing and modifying the underlying drivers of immunoglobulin A nephropathy, a progressive immune-mediated kidney disease, including the role of targeted-release budesonide.

Immunoglobulin A nephropathy (IgAN) is the most prevalent primary glomerular disease globally, and it is a leading cause of chronic kidney disease (CKD) and kidney failure in young and middle-aged adults (Schena and Nistor, 2018). The estimated incidence is 2.5 per 100 000 adults per year (Zaidi et al, 2024). However, the true global incidence and prevalence remain unknown given subclinical disease, international variation in screening and biopsy practices, and limitations in data recording accuracy.

Current understanding of IgAN pathophysiology is summarised by a multi-hit hypothesis (Berger and Hinglais, 1968). The hypothesis presents that an excess of poorly O-galactosylated IgA1 in the circulation leads to the formation of IgA-immune complexes, which are deposited within the glomerular mesangium and drive glomerular inflammation and fibrosis.

Although once considered a predominantly benign condition, recent registry data demonstrate that, without early and effective disease control, most patients progress to kidney failure over their lifetime (Pitcher et al, 2023). Landmark data from the UK National Registry of Rare Kidney Diseases (RaDaR) showed that among people with IgAN, median kidney survival was 11.4 years, with a mean age at kidney failure or death of just 48 years (Pitcher et al, 2023). Cohorts from Sweden and the USA report reductions in life expectancy of 6 and 10 years respectively, mainly attributed to complications of kidney failure (Hastings et al, 2018; Jarrick et al, 2019).

Patient identification

IgAN arises from a complex interplay of genetic and environmental factors. Patients may experience intermittent episodes of visible haematuria following mucosal infections, which is thought to reflect a dysregulated mucosal immune response to encountered antigens (Suzuki et al, 2011).

Globally, IgAN prevalence varies substantially (Schena and Nistor, 2018). It is more common and exhibits a more severe phenotype with faster disease progression in Asian than Western populations (Schena and Nistor, 2018), possibly due to systemic mass screening in some Asian countries, late referrals in Western countries and adoption of different indications for kidney biopsy. Similar patterns in migrant populations implicate genetic factors (Barbour et al, 2013). Studies have identified more than 30 independent genome-wide significant risk loci, accounting for approximately 11% of disease risk (Kiryluk et al, 2014; 2023; Li et al, 2020; Sanchez-Rodriguez et al, 2021).

Clinically, IgAN presents across a wide spectrum, from asymptomatic haematuria and/or proteinuria to episodic visible haematuria (*Box 1*). Most patients experience gradual loss of kidney function over years to decades. Fewer than 5% present with nephrotic syndrome or rapidly progressive glomerulonephritis (Cheung et al, 2025). Population-level screening using urinary dipstick testing is used in some Asian countries with higher IgAN prevalence (Lee

Box 1. Patient risk stratification

People with immunoglobulin A nephropathy are at increased risk of progression if they exhibit one or more of the following features:

- Persistent proteinuria ≥1.0 g/day
- Declining estimated glomerular filtration rate or rising serum creatinine
- Hypertension
- High-risk histological features (e.g. active MEST-C lesions)
- Younger age at presentation with significant proteinuria

et al, 2023). However, diagnosis can only be confirmed through kidney biopsy demonstrating diffuse, dominant mesangial IgA staining (Lee et al, 2023).

Management

With the introduction of new therapies and increasing recognition of the high lifetime risk of kidney failure, simultaneous targeting of IgA immune complex–mediated kidney damage and the general maladaptive response to nephron loss are now recommended.

Addressing maladaptive responses to nephron loss

Poorly controlled proteinuria and hypertension are established risk factors for kidney failure and death in IgAN (Berthoux et al, 2011). The Kidney Disease Improving Global Outcomes (KDIGO) IgAN and IgAV Work Group recommends strict targets (Rovin et al, 2025):

- Proteinuria <0.5 g/day, ideally <0.3 g/day
- Blood pressure ≤120/70 mmHg.

Renin–angiotensin system inhibition (RASi) is a long-established therapy that slows CKD progression by reducing blood pressure and proteinuria (Cheng et al, 2009). A meta-analysis of randomised controlled trials demonstrated that RASi slowed estimated glomerular filtration rate (eGFR) decline and reduced proteinuria in people with IgAN (Cheng et al, 2009). KDIGO recommends treatment with an optimised, maximally tolerated dose of an angiotensin-converting enzyme inhibitor (ACEi) or angiotensin II receptor blocker (ARB) for all patients with IgAN (Rovin et al, 2025).

The endothelin system interacts closely with the renin–angiotensin system and contributes to glomerulosclerosis and tubulointerstitial fibrosis in various kidney diseases (Komers and Plotkin, 2016). Sparsentan, a dual endothelin type A receptor antagonist and ARB, was evaluated in the phase 3 PROTECT trial (Rovin et al, 2023). Compared with irbesartan, sparsentan improved proteinuria, chronic eGFR slope and absolute eGFR change at 2 years (Heerspink et al, 2023; Rovin et al, 2023). Sparsentan is now approved for IgAN in several countries, and KDIGO suggests its use in patients at risk of disease progression (Rovin et al, 2025). Atrasentan, a selective endothelin type A receptor antagonist, has received accelerated approval from the US Food and Drug Administration for adults with primary IgAN at risk of rapid progression (Heerspink et al, 2025). The drug is being evaluated in the phase 3 ALIGN trial (Heerspink et al, 2025).

Sodium–glucose co-transporter 2 (SGLT2) inhibitors have demonstrated significant reductions in kidney disease progression and mortality in IgAN and are recommended in patients at risk of progression. The DAPA-CKD (Wheeler et al, 2021) and EMPA-KIDNEY trials (Baigent et al, 2022) demonstrated that SGLT2 inhibitors slowed kidney disease progression and improved survival in patients with IgAN when compared with placebo. KDIGO suggests the use of SGLT2 in patients with IgaN at risk of progression (KDIGO IgAN and IgAV Work Group, 2025).

CKD is frequently complicated by dyslipidaemia and accelerated cardiovascular disease (Schiffrin et al, 2007). Therefore, statin treatment is recommended for all patients with CKD aged ≥50 years and for those aged 18–49 years with additional cardiovascular disease risk factors (KDIGO Lipid Work Group, 2013).

Lifestyle changes underpin holistic care in CKD. Patients with IgAN are recommended to (Rovin et al, 2025):

- Restrict dietary sodium (<2 g/day)
- Cessate smoking and vaping
- Control weight
- Conduct endurance exercise.

Addressing condition-specific drivers of nephron loss

Although general CKD management strategies reduce glomerular hypertension and modulate

the tubulointerstitial response to proteinuria, they do not address upstream IgAN-specific drivers of nephron loss (Rovin et al, 2025). Therefore, treatments must minimise IgA immune complex formation and IgA immune complex-mediated glomerular injury. These concurrent treatment strategies must be initiated early to reduce lifetime risk of kidney failure (Rovin et al, 2025).

Historically, systemic glucocorticoids were the only treatment available to target IgAN-specific drivers of nephron loss. The TESTING study is the largest randomised controlled trial of systemic glucocorticoids in IgAN, in which a reduced-dose regimen of methylprednisolone (0.4 mg/kg/day) significantly reduced the frequency of 40% eGFR decline, kidney failure or death due to kidney disease compared with placebo (Lv et al, 2022). However, the beneficial effects on proteinuria and eGFR were not sustained in the 3 years following glucocorticoid cessation. A greater number of serious adverse events, including death, were also observed in the methylprednisolone group, despite reducing the dose from the higher 0.6–0.8 mg/kg/day regimen before the trial was stopped early due to safety concerns (Lv et al, 2017).

Current evidence suggests that systemic glucocorticoids offer only short-term benefit and carry substantial risks of treatment-related toxicity. If used, KDIGO recommends a reduced-dose regimen of methylprednisolone (or equivalent) at 0.4 mg/kg/day (maximum 32 mg/day) for 2 months, followed by tapering by 4 mg/day each month for a total of 6–9 months (Rovin et al, 2025). Treatment should be combined with antimicrobial prophylaxis and antiviral prophylaxis in those carrying hepatitis B, along with gastroprotection and bone protection (Rovin et al, 2025).

The therapeutic landscape in IgAN treatment is rapidly changing, with multiple new therapies now approved or in advanced-stage clinical trials. A targeted-release formulation of budesonide is the first drug to be approved for IgAN.

Targeted-release budesonide

Targeted-release budesonide (Kinpeygo®▼) is designed to suppress pathogenic IgA production at its source. Growing evidence implicates gut-associated lymphoid tissue (GALT) as a major source of circulating galactose-deficient IgA1 in IgAN (Coppo, 2018). The capsule dissolves in the terminal ileum, delivering budesonide directly to the GALT, with extensive first-pass hepatic metabolism minimising systemic steroid exposure (Barratt et al, 2020).

The phase 3 NefIgArd trial evaluated targeted-release budesonide in adults with IgAN (Lafayette et al, 2023). Targeted-release budesonide significantly improved kidney outcomes compared with placebo, lowering the risk of kidney failure, reducing the risk of a 30% eGFR decline and achieving sustained proteinuria reduction over 2 years (Lafayette et al, 2023). Following the NefIgArd trial, targeted-release budesonide became the first drug approved for the treatment of IgAN.

In the phase 2 NEFIGAN trial, targeted-release budesonide significantly reduced levels of galactose-deficient IgA1 and IgA immune complexes, providing mechanistic support for its disease-modifying effects (Wimbury et al, 2024). Given the importance of targeting the underlying immunological drivers of IgAN, the latest KDIGO guideline (Rovin et al, 2025) recommends that patients at risk of disease progression are treated with a 9-month course of targeted-release budesonide as first-line therapy when available. Targeted-release budesonide is recommended over systemic glucocorticoids, with reduced-dose systemic glucocorticoids suggested only when targeted-release budesonide is unavailable (Rovin et al, 2025).

National Institute for Health and Care Excellence (NICE) guidance found that targeted-release budesonide in combination with standard care is cost effective compared with standard care alone for people with IgAN and a baseline urine protein-to-creatine ratio ≥90 mg/mmol or a

protein excretion of ≥1.0 g/day (NICE, 2026). An incremental cost-effectiveness ratio of £1950 per quality-adjusted life year (QALY) gained was estimated. However, the committee noted there was uncertainty in the modelling of standard care, given that sparsentan and SGLT2 inhibitors were not included as part of standard care in the clinical trial (NICE, 2026). Nonetheless, the committee concluded that the most likely cost-effectiveness estimate for targeted-release budesonide plus standard care compared with standard care alone was likely to be under £20 000 per QALY gained, representing an acceptable use of NHS resources.

Targeted-release budesonide is generally well tolerated, with mostly mild and reversible side effects. The safety profile is consistent with known budesonide effects, reflecting limited systemic absorption. During the 9-month treatment period in the NefIgArd trial (Lafayette et al, 2023), the most common treatment-emergent adverse events were:

- Peripheral oedema
- Hypertension
- Muscle spasms
- Acne
- Headache.

During the 15-month observational follow-up, adverse event rates were similar between the targeted-release budesonide and placebo groups (Lafayette et al, 2025).

Advanced disease management

Despite optimal care, around 50–75% of adults with IgAN are estimated to develop kidney failure within 20 years of diagnosis (Pitcher et al, 2023). As with other causes of kidney failure, kidney transplantation is the gold standard of management and associated with improved survival and quality of life compared with dialysis (Tonelli et al, 2011).

Overall, people with IgAN have favourable transplantation outcomes compared with many other forms of glomerulonephritis (Cosio and Cattran, 2017). However, clinical disease recurrence can be seen in 15–30% of patients (Ponticelli et al, 2004; Cosio and Cattran, 2017).

Although kidney transplantation is the preferred form of kidney replacement therapy, not all patients are suitable candidates. Careful assessment of individual risk factors and comorbidities is essential to determine transplant suitability and expected risk profile. Comprehensive recommendations are available to support clinicians in making these complex decisions (Rovin et al, 2025).

For patients unsuitable for, or awaiting, kidney transplantation, renal replacement therapy is provided by dialysis. Significant variation remains in global practice patterns, but there is increasing recognition of the importance of personalising dialysis care to individual patient needs whenever possible (Chan et al, 2019).

Patient selection

Targeted-release budesonide was given full marketing authorisation from the European Medicines Agency in July 2024. It is indicated for adult (≥18 years) patients with (EMA, 2025):

- Biopsy-proven primary IgAN
- Proteinuria ≥1.0 g/day (or urine protein-to-creatinine ratio ≥0.8 g/g)
- No contraindications.

Absolute contraindications include hypersensitivity to the active substance or excipients and severe liver impairment (Child-Pugh Class C), due to the risk of markedly increased systemic exposure and steroid-related adverse events (European Medicines Agency (EMA), 2025). Additional caution and monitoring are required in patients with the following (EMA, 2025):

- Conditions that increase risk of glucocorticoid-related adverse effects (including active or untreated infections)

- Hypertension
- Diabetes mellitus
- Osteoporosis
- Peptic ulcer disease
- Glaucoma
- Cataracts.

Targeted-release budesonide should not be used during pregnancy unless clinically essential. Limited human data are available, but budesonide has been shown to cause abnormalities in foetal development in pregnant animals (EMA, 2025).

Conclusion

By direct delivery to gut-associated lymphoid tissue, targeted-release budesonide reduces production of galactose deficient IgA1 and thereby addresses an early driver of disease activity. Clinical trial data demonstrate sustained reductions in proteinuria and a favourable effect on kidney function, with a safety profile consistent with limited systemic steroid exposure. As a result, KDIGO now recommends targeted-release budesonide over systemic glucocorticoids for patients at risk of disease progression, with contemporary guidelines prioritising targeted therapies with lower toxicity (Rovin et al, 2025). Its integration into routine care represents a measured advancement in the management of IgAN, supporting earlier intervention and complementing established supportive therapies.

Conflicts of interest

JB has received consulting, speaker and grant support from Alnylam, Argenx, Astellas, BioCryst, Calliditas, Chinook, Dimerix, Galapagos, GlaxoSmithKline, Novartis, Omeros, Travere Therapeutics, Vera Therapeutics, Visterra.

LR has no conflicts of interest to declare.

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Written by **Jonathan Barratt**, Mayer Professor of Renal Medicine, University of Leicester, and **Lydia Roberts**, NIHR Academic Clinical Fellow Paediatrics, University of Leicester.

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IgA, immunoglobulin A; IgAN, IgA nephropathy; UPCR, urine protein-creatinine ratio.

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