Scientific Research Publishing

International Journal of Clinical Medicine, 2026, 17(6), 181-200
https://www.scirp.org/journal/ijcm
ISSN Online: 2158-2882
ISSN Print: 2158-284X

The Emirates Medical Association of Nephrology and Transplantation Society Consensus Statements on Optimizing Clinical Management of IgA Nephropathy: The Urgent Need for a Paradigm Shift in the UAE

Ayman Almadani[1*], Dastoor Hormaz[1], Harith AlJubori[2], Ramzi Abou Ayache[3], Ahmed Ewaida[4], Muriel Ghosn[5], Amna Al Hadari[6], Emad Khater[7], Dana Kidder[8], Suhail Al-Salam[9], Shaikha Ali[10,11], Mona Al Rukhaimi[11,12]

[1]SEHA Kidney Care, Abu Dhabi, UAE
[2]Nephrology Department, Al Qassimi Hospital, Emirates Health Services, Sharjah, UAE
[3]Mediclinic City Hospital Dubai, Dubai, UAE
[4]Al Zahra Hospital, Dubai, UAE
[5]Nephrology Department, Cleveland Clinic Abu Dhabi, Abu Dhabi, UAE
[6]Dubai Hospital, Dubai Health, Dubai, UAE
[7]SEHA Kidney Care, Sheikh Khalifa Medical City, Abu Dhabi, UAE
[8]SEHA Kidney Care, Al Ain, UAE
[9]College of Medicine & Health Sciences, UAE University, Al Ain, UAE
[10]Sheikh Khalifa Medical City, Ajman, UAE
[11]Emirates Medical Association of Nephrology and Transplantation Society (EMAN-T), Dubai, UAE
[12]Dubai Medical College, Dubai, UAE
Email: *aymanalmadani@me.com

How to cite this paper: Almadani, A., Hormaz, D., AlJubori, H., Ayache, R.A., Ewaida, A., Ghosn, M., Al Hadari, A., Khater, E., Kidder, D., Al-Salam, S., Ali, S. and Al Rukhaimi, M. (2026) The Emirates Medical Association of Nephrology and Transplantation Society Consensus Statements on Optimizing Clinical Management of IgA Nephropathy: The Urgent Need for a Paradigm Shift in the UAE. *International Journal of Clinical Medicine*, **17**, 181-200.
https://doi.org/10.4236/ijcm.2026.176014

Abstract

Immunoglobulin A nephropathy (IgAN) is the most common form of primary glomerular disease worldwide. IgAN leads to end stage kidney disease in up to 40% - 50% of all patients and is associated with increased mortality, leading to a clear need for increased awareness and early treatment of the disease. In the United Arab Emirates (UAE), the number of IgAN diagnoses has steadily risen over several decades, putting increased pressure on primary care physicians and nephrologists to find nationwide solutions to optimize its clinical management. In recognition of this growing need, the Emirates Medical Association Nephrology and Transplantation Society (EMAN-T) convened a panel of 12 experts with recognized seniority in IgAN clinical management,

Received: May 5, 2026
Accepted: June 9, 2026
Published: June 12, 2026

who identified challenges and opportunities for improving and standardizing national IgAN care pathways. A limitation was a lack of patient or payer representation on the consensus panel. This expert clinical consensus article provides 6 consensus position statements that together set out a vision for optimal IgAN clinical management in the UAE. The statements are intended to serve as a first step towards optimizing IgAN awareness, diagnosis, risk assessment, treatment and ongoing clinical management, with the ultimate goal of improving care pathways for IgAN across the UAE.

Keywords

Immunoglobulin A Nephropathy, IgAN, UAE, Management, Consensus

1. Introduction

Immunoglobulin A nephropathy (IgAN) is the most common form of primary glomerular disease globally that, left untreated, can lead to end stage kidney disease (ESKD) [1]. Diagnosis of IgAN is established based on detection of dominant or co-dominant galactose-deficient IgA1 deposits (Gd-IgA1) in the glomeruli, resulting in damage of the basement membrane and manifesting as hematuria, proteinuria, and ultimately renal failure [2] [3]. IgAN is associated with increased mortality [4] [5], with average life expectancy estimated to be shortened by 6 - 10 years in international studies [6] [7]. Remarkably, up to 50% of patients with IgAN develop ESKD and require dialysis within 20 years of diagnosis [8], meaning there is an unmet need for increased recognition and early treatment of the disease, particularly in the United Arab Emirates (UAE) where current clinical management pathways are suboptimal and non-standardized.

The prevalence of IgAN varies by global region, and is highest in Asia Pacific and lowest across Southern Africa [9]. While not a replacement for robust prevalence data, insights into the regional variations in IgAN frequency come from biopsy studies showing that IgAN diagnoses comprise 50% of all renal biopsies in East Asia (specifically Cambodia, Malaysia, Philippines, Singapore, Thailand, and Vietnam) [9]. Compared with 13% - 20% in Europe, 12% in the USA, and 1% in Southern African nations [9]. In the Middle East, the proportions of biopsies that are IgAN-positive across Gulf nations including Kuwait [10], Oman [11], Bahrain [12], and Saudia Arabia [13], range from ~8% - 14%. In the UAE, there has been a notable increase in reported IgAN from historic levels of ~6% of biopsies (between 1978-1996) [14] to more recent reports ranging from ~11% (between 2005-2014) [15] up to ~23% (between 2010-2015) of all glomerulonephritis biopsies [15] [16]. This appears to be driven, at least in part, by an influx of expatriate populations including high proportions of Filipino and Indian nationals [16], who are at increased risk of IgAN [9]. However, determining the burden of IgAN is hindered by several factors, including the high variability in IgAN presentation [3] as well as the requirement for biopsy in order to make a diagnosis [2]. As such,

it is possible that the burden of IgAN in the UAE is higher than previously reported, due to factors such as access to performing kidney biopsy in private healthcare settings, general reluctance in performing an invasive procedure for confirming the diagnosis and the unknown quantity of patients with mild disease who are not likely to undergo biopsy [9].

Here, we have developed an expert clinical consensus article with the goal of improving care pathways for IgAN across the UAE. We envision a standardized approach to care that contains sufficient scope for personalized treatment plans tailored to individual patient profiles. The consensus was initiated to provide a framework for recognizing IgAN as a common, but treatable, cause of progressive kidney disease, that has a wide range of presentations and may lead to ESKD in patients. In this consensus article, we have developed six position statements to highlight the need for a standardized approach to the diagnosis, risk assessment, and treatment of patients with IgAN in the UAE. We also address the need for wider disease awareness among primary care physicians (PCPs) and patients, including the generation of patient-friendly educational materials to aid increased health-consciousness among affected individuals.

2. Methodology

The Emirates Medical Association Nephrology and Transplantation Society (EMAN-T) recognized the lack of national protocols for IgAN management and the need to optimize IgAN clinical management protocols across the UAE. This unmet need led to the initiation of this consensus article by convening a group of 12 experts (comprising 11 nephrologists and 1 pathologist) from the UAE. The expert panel was selected based on the recognized seniority of all experts in the clinical management of IgAN in the UAE, the experts represented four of the seven emirates of the UAE and represented a variety of healthcare institutions within the UAE. The experts highlighted perspectives on challenges and opportunities for improving and standardizing IgAN care across the UAE. The lead author in collaboration with the chair of the EMAN-T society drafted six statements that reflected the panel perspectives and could guide IgAN clinical management protocols and treatment pathways on a national level. Using the Delphi method, these statements were circulated to all panel members for anonymous voting, whereupon ≥80% agreement was considered a consensus. Statements with <80% agreement were revised and re-circulated for a second round of voting. Only one round of voting was required for all statements, with a 100% response rate achieved, and all statements scoring >80% of agreement at first round. **Table 1** summarizes the agreement percentage for each final statement. All agreed consensus statements are subsequently included herein.

3. Results

3.1. IgAN Diagnosis and Risk Stratification

Consensus statement 1: *Establishing uniform diagnostic criteria is crucial to im-*

plementing structured IgAN care across the UAE

Evidence label: Guideline based

Current diagnostic pathways for IgAN across the UAE are non-standardized. Given >90% of IgAN cases are idiopathic [2] and the average age of an IgAN diagnosis is 30 - 40 years [17], a high degree of clinical suspicion is required to achieve a diagnosis. Clinical suspicion for IgAN should arise in the presence of acute kidney injury, hematuria or proteinuria, often coexisting with hypertension, and prompt referral to nephrology should take place upon discovery of any combination of these signs [18]-[20]. While routine screening for IgAN is common in countries with high disease prevalence (e.g., China and Japan) [18], awareness of IgAN is low among general practitioners, internal medicine, and family medicine physicians in the UAE. As such, when evaluating patients for chronic kidney disease (CKD) the inclusion of a work-up for glomerulonephritis, including IgAN, may be warranted given that IgAN remains the most common glomerular disorder identified through biopsy in the UAE [16].

Table 1. Delphi voting results on final consensus statements.

Statement	Agreement (%)
Establishing uniform diagnostic criteria is crucial to implementing structured IgAN care across the UAE	**100%**
Kidney biopsy is an essential component of IgAN diagnosis, and we recommend enhancing its access across the UAE	**100%**
Standard risk stratification frameworks should be widely adopted to inform prognosis and help create a personalized clinical management plan	**100%**
IgAN management and treatment goals should be clearly defined and standardized across the UAE, including a defined treatment algorithm with treatment escalation strategies	**82%**
Treatment algorithms should be reviewed and updated annually to facilitate the incorporation of emerging therapies into care pathways	**91%**
Ongoing education on best practice and advances in IgAN management should be a major goal, due to the potential to improve care pathways and ensure an aligned approach among all relevant stakeholders in the UAE	**100%**

IgAN, immunoglobulin A nephropathy; UAE, United Arab Emirates.

Other than estimated glomerular filtration rate (eGFR) and proteinuria, no validated prognostic serum or urine biomarkers for IgAN exist [17], meaning greater education and awareness is needed among PCPs. In addition, the incorporation of urine analysis into routine checkups is warranted, as urine analyses are not often conducted unless the patient reports issues like urinary problems or pain. We highly recommend undertaking microscopic urine examination upon a positive hematuria result (strongly favored over dipstick tests or machine cytometry), as this plays a crucial role in the early detection of glomerular diseases including IgAN. Once patients have been referred to nephrology in the UAE, some nephrologists advocate proceeding immediately to biopsy in patients with unexplained hematuria, while others only proceed based on detection of proteinuria of >1

g/day. However, as proteinuria is an internationally recognized negative prognostic factor for IgAN [17], we advocate that nephrologists in the UAE adopt a more proactive and timely approach regarding when to proceed to biopsy. We recommend a kidney biopsy should be considered (unless contraindicated) in all adults with proteinuria ≥ 0.5 g/day (or equivalent) in whom IgAN is a possible diagnosis, in line with the latest KDIGO 2025 international guidelines [17].

Red flags for diagnosis include recurrent gross hematuria, particularly during, or shortly after, upper respiratory infections, persistent microscopic hematuria, unexplained proteinuria ≥ 0.3 g/day, early-onset hypertension, and otherwise unexplained reductions in kidney function. Synpharyngitic hematuria and a family history of IgAN or IgA vasculitis provide additional clues (**Table 2**). The presence of these red flags should trigger prompt referral to nephrology and consideration of kidney biopsy. If red flags are present in a patient, additional consideration should be given to ethnicity, particularly given the increased risk of IgAN in patients of South and Southeast Asian ethnicity who comprise a high number of expatriates to the UAE.

Table 2. Red flags for IgAN diagnosis.

Clinicians should remain aware of the following clinical clues for IgAN
Recurrent gross hematuria during, or shortly after, upper respiratory tract infections
Persistent microscopic hematuria
Unexplained proteinuria ≥ 0.3 g/day
Early-onset hypertension
Otherwise unexplained reductions in kidney function
Synpharyngitic hematuria
Family history of IgAN or IgA vasculitis

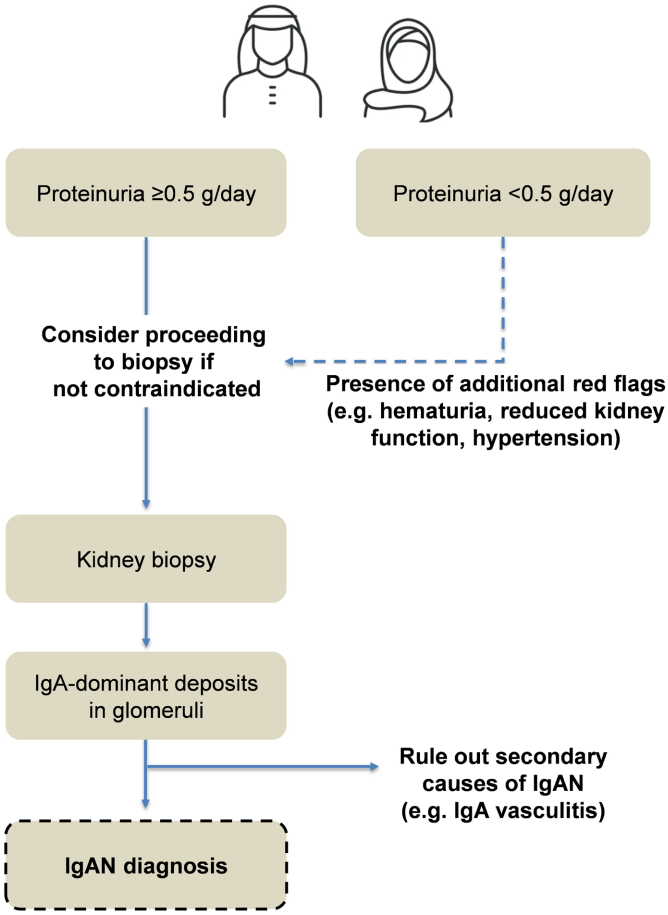
IgA, immunoglobulin A; IgAN, IgA nephropathy.

Consensus statement 2: *Kidney biopsy is an essential component of IgAN diagnosis, and we recommend enhancing its access across the UAE*

Evidence label: Guideline based

Obtaining a kidney biopsy is an essential requirement for an IgAN diagnosis. Early biopsy is crucial to ensure timely treatment initiation and prevention or limitation of potential further kidney damage. In line with KDIGO 2025 Guidelines, we recommend a kidney biopsy should be considered (unless contraindicated) in all adults with proteinuria ≥ 0.5 g/day (or equivalent) in whom IgAN is a possible diagnosis [17]. In cases where there is a clinical suspicion for IgAN, for example, when patients co-present with hematuria and reduced kidney function, we recommend the threshold for performing biopsy should be lowered to proteinuria < 0.5 g/day. The diagnosis of IgAN is made by demonstrating the presence of dominant or codominant IgA deposits in the mesangium or capillary loops (accompanied by cell proliferation and expansion) [18]. Next, secondary causes must be

excluded before the diagnosis of primary IgAN can be confirmed. These include IgA vasculitis, IgAN secondary to viral infections (e.g., HIV or hepatitis), IgA-dominant post-infectious glomerulonephritis, inflammatory bowel disease, autoimmune disease, and liver cirrhosis [17] [18]. A summary of our recommendations for standardized IgAN diagnostic criteria to be adopted throughout the UAE are summarized in Figure 1.



IgA, immunoglobulin A; IgAN, IgA nephropathy; UAE, United Arab Emirates.

Figure 1. Recommendations for IgAN diagnosis in the UAE.

Given the central role of kidney biopsy in achieving an IgAN diagnosis, this needs to be more firmly integrated into clinical practice across the UAE to ensure disparities in access and reimbursement are not an obstacle to diagnosis. A single biopsy is usually sufficient to achieve diagnosis if a thorough risk assessment is carried out, for example, the quality of the biopsy should be rapidly confirmed by assessing the number of glomeruli. An exception is cases of suspected sampling error (e.g., <8 glomeruli) or discordance between clinical presentation and biopsy findings, where repeat biopsy may be considered. Once a diagnosis has been made, patients should ideally be enrolled in a registry, and EMAN-T has begun work on

establishing a kidney biopsy registry with external stakeholders. A patient registry is an organized system that uses observational study methods to collect uniform data (clinical and other) to evaluate specified outcomes for a population defined by a particular disease, condition, or exposure, and that serves one or more predetermined scientific, clinical, or policy purposes [21]. Benefits of a national registry include defining incidence and prevalence, guiding strategic direction, operational standards, and stakeholder collaboration. A registry will provide a more accurate picture of IgAN epidemiology within the UAE's multiethnic population. It will also enable long-term tracking of kidney outcomes and disease progression and facilitate benchmarking and comparison of clinical performance across UAE centers. The registry can inform clinical pathways and support the development of standardized national protocols, it has the potential to accelerate early adoption and evaluation of emerging therapies through real-world evidence, and support health technology assessments and reimbursement decisions for new treatments. Registries can generate local evidence to address gaps not covered by international cohorts, which is of importance when considering the UAE's unique ethnic distribution and biopsy practices.

However, there are challenges when establishing a national registry, including necessity of strict compliance with UAE data privacy laws, requirements for robust governance frameworks, secure data management processes, and clear patient consent procedures. There are logistical complexities of standardizing data fields, definitions, and formats across various hospital systems and variability in electronic medical record platforms and documentation practices.

There are some specific challenges and opportunities to improve biopsy access throughout the UAE. These include a lack of recognition that biopsy is a lifesaving procedure in patients with rapid progressive glomerulonephritis or unexplained rapid decline in eGFR. There should be increased clarity in healthcare institutions as to whether interventional radiology or nephrology departments are responsible for biopsy. The biopsy instrument and needle size should be standardized, as well as standardized training provided for the designated department. Identification of Centers of Excellence for biopsy should be considered. These Centers would be regularly audited, including maintenance of logbook for operators, formal training, certification and recertification for operators.

Consensus statement 3: *Standard risk stratification frameworks should be widely adopted to inform prognosis and help create a personalized clinical management plan*

Evidence label: Guideline based

At the time of diagnosis, over 50% of patients already have CKD stage 3 - 5 [20], indicating a substantial loss of functional nephrons. This stark observation underlines the need for early action to prevent or limit further kidney decline. As such, early quantification of risk of progression is needed to inform discussions with patients for shared decision-making on the initiation of appropriate therapy as soon as possible. The International IgAN Prediction Tool is the primary method

that is currently used in a prognostic manner for risk prediction in patients with IgAN [17]. The tool was designed to predict the risk of 50% decline in eGFR or ESKD following biopsy by integrating clinical and histological factors, including eGFR, blood pressure, proteinuria, age, ethnicity, medication use (angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, and immunosuppressants) and MESTC scores (based on the kidney biopsy and incorporating mesangial hypercellularity [M], endocapillary hypercellularity [E], segmental sclerosis [S], tubular atrophy/interstitial fibrosis [T], and crescents [C]) [17] [18].

It is important to note that ethnicity has the potential to impact risk stratification due to differences in MESTC scores. For example, Chinese and Japanese ethnicities yield a greater 5-year ESKD progression risk than Caucasian ethnicity [17] [18]. The International IgAN Prediction Tool has not been externally validated in the UAE's multi-ethnic population and local validation is urgently needed. The importance of ethnicity is well illustrated when comparing the rate of eGFR decline in the control arms of the phase 3 STOP-IgAN [22] [23] and TESTING [24] trials, which included predominantly White and Asian patients, respectively, and were 1.6 mL/min/1.73m^2 per year for White patients compared with 4.97 mL/min/1.73m^2 per year in Asian patients.

There are some drawbacks to the International IgAN Prediction Tool, including the exclusion of more modern therapies in its stratification score [18], as well as its limitation of being able to fully capture longer-term risks [25]. As such, there is scope for discovering alternative risk stratification tools that go beyond current markers (proteinuria and eGFR), which are later indicators of disease, and that can be adjusted to incorporate clinical changes over time [25]. Implementation and validation of non-invasive methods to visualize kidney inflammation in IgAN is also an important future goal, and there have been some advances in the use of molecular imaging techniques including positron electron tomography- and magnetic resonance imaging-based methods in this regard [26] [27].

A future risk stratification tool should be optimized for use in the UAE by integrating longitudinal variables such as time-averaged proteinuria, and eGFR slope, and adding validated noninvasive biomarkers, when available. Additional ethnicity-specific predictors could be considered to be a factor in the tool. In future, a local validation study is recommended to ensure the risk model accurately reflects the variable access to biopsy and the multiethnic population in the UAE. Ideally risk stratification would have the potential to contribute to a personalized treatment plan for patients.

3.2. Treatment Pathways for IgAN

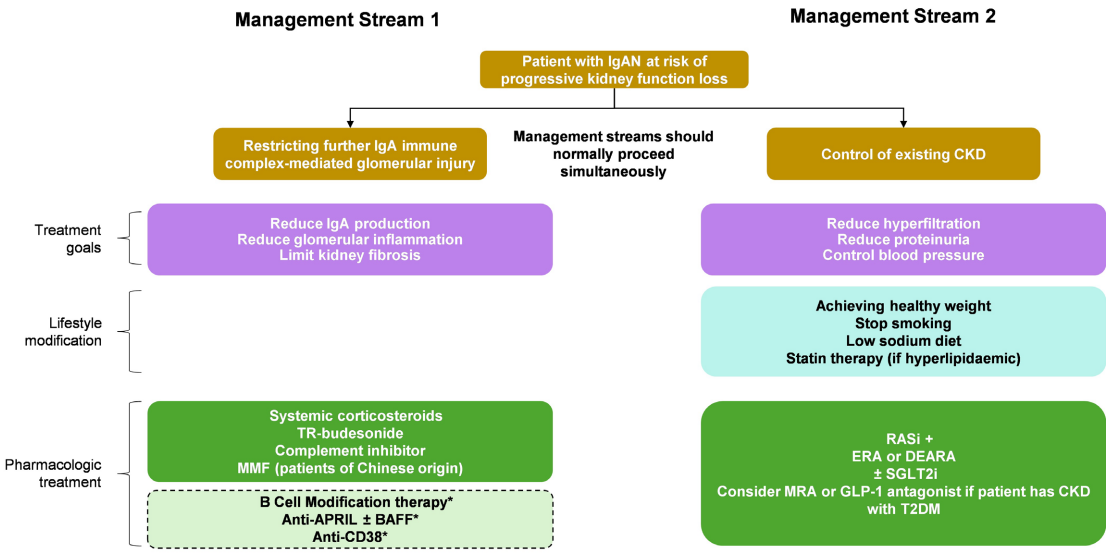
Consensus statement 4: *IgAN management and treatment goals should be clearly defined and standardized across the UAE, including a defined treatment algorithm with treatment escalation strategies*

Evidence label: Guideline based

The ultimate treatment goal for IgAN is to prevent progression to ESKD. Spe-

cifically, the rate of kidney function decline should be reduced to <1 mL/min per year for rest of patient's life [17] [18]. Currently, the only biomarker to guide treatment decisions is proteinuria, which tracks well with kidney outcomes, and should be maintained at a minimum of <0.5 g/day (ideally < 0.3 g/day). Achievement of this may require multiple pharmacological and non-pharmacological strategies, particularly in patients with extensive kidney scarring [17] [18]. Extent of microhematuria is associated with IgAN progression, and is emerging as a potential future therapeutic goal in IgAN management [25]. Ideally, in future, treatment should be personalized where possible, matching treatment options to specific pathophysiological profiles, whereby clinical context encompassing all available data should be utilized. Key factors affecting disease risk, and therefore treatment decisions, include ethnicity and age at presentation, as well as current rate of disease progression.

As a starting point for standardizing IgAN management across the UAE, and in line with KDIGO international guidelines [17], lifestyle modification is recommended in all patients including the achievement of a healthy weight, cessation of smoking, and adaptation of a low sodium diet. From this point, clinical management of IgAN can be broadly split into two main management streams: 1) prevention or minimization of further IgA immune complex-mediated glomerular injury, with the treatment paradigm aimed at slowing disease, inflammation and fibrosis and 2) universal supportive care to provide control of existing CKD (managing the generic response to nephron loss, which is present in newly diagnosed cases at ~50% [20]) (**Figure 2**).



*When approved for use in the UAE. APRIL, a proliferation-inducing ligand; BAFF, B-cell activating factor; CKD, chronic kidney disease; DEARA, dual endothelin angiotensin receptor antagonists; ERA, endothelin receptor antagonist; GLP-1, glucagon-like peptide-1 IgA, immunoglobulin A; IgAN, IgA nephropathy; MRA, mineralocorticoid receptor antagonist; RASi, renin-angiotensin system inhibitor; SGLT2i, sodium-glucose cotransporter 2 inhibitor; T2DM, type 2 diabetes mellitus; TR, targeted release.

Figure 2. Proposed IgAN treatment algorithm for the UAE.

3.2.1. Preventing or Minimizing Further IgA Immune Complex-Mediated Glomerular Injury

Targeting immunological activity is the most important treatment goal in IgAN management and has the ability to target multiple pathological mechanisms simultaneously to combat disease heterogeneity and achieve effective disease control (Management stream 1; **Figure 2**). The "four-hit hypothesis" of IgAN pathogenesis provides a useful framework with which to consider immunological treatment interventions and is pertinent for higher-risk or persistent-proteinuria groups. Hit 1 involves increased production of pathogenic Gd-IgA1, followed by Hit 2, which is the production of auto-antibodies against Gd-IgA1 [28]. The third and fourth hits involve, respectively, the generation of and subsequent deposition of circulating immune complexes in the glomerular mesangium, leading to inflammation and fibrosis [28]. Overall, therapies targeting any stage of this four-Hit immunological response are aimed at halting glomerular inflammation, reducing the production of Gd-IgA1, and stopping pro-fibrotic signals in the kidney [28]. While most of these stages are well covered by new and emerging treatments, interventions to directly stop pro-fibrotic signals are lagging behind on the treatment horizon [28].

The STOP-IgAN [22] and TESTING [24] trials provided the first robust randomized clinical trial evidence that on-treatment systemic glucocorticoids were effective at mitigating glomerular inflammation in IgAN. However, long-term use of corticosteroids is controversial due to short- and long-term toxicity, and current KDIGO IgAN treatment guidelines have strict recommendations for their use, with targeted release (TR)-budesonide now recommended ahead of methyl prednisone in patients at risk of progressive loss of kidney function [17]. TR-budesonide is designed to target the gut-associated lymphoid tissue of the terminal ileum to reduce the production of pathogenic Gd-IgA1 ("Hit 1"), and was found to lower urine protein-to-creatinine ratio and limit eGFR loss compared with placebo after 9 months of treatment in the phase 3 NefigArd trial [29] [30].

Strategies to target the production of auto-antibodies against IgA1 ("Hit 2") include B-cell depleting and modification therapies [18] [28] [31] [32]. B-cell depletion therapies utilizing anti-CD20 agents such as rituximab have so far shown limited efficacy [28], while other strategies including CD38 depletion are currently being tested, as are B-cell modification strategies such as inhibition of B-cell activating factor (BAFF) and cytokine A proliferation-inducing ligand (APRIL) (discussed in later section) [18] [28] [31] [32]. Strategies to target the inflammatory pathways in IgAN (driven by Hits 3 and 4) are another major focus of IgAN treatment, with inhibition of complement activation seen by some physicians as an efficacious and safer alternative to systemic corticosteroids in controlling kidney inflammation in IgAN [28]. A summary of our recommended treatment algorithm for standardizing the clinical management of patients with IgAN across the UAE is provided in **Figure 2**.

3.2.2. Control of Existing CKD

The initial treatment response to nephron loss is aimed at reducing hyperfiltra-

tion, proteinuria, and controlling blood pressure with supportive care. Here, in Management stream 2 (**Figure 2**), maximally titrated renin-angiotensin system (RAS) inhibitors, endothelin receptor antagonists (ERAs), dual endothelin angiotensin receptor antagonists (DEARAs), and sodium-glucose cotransporter 2 (SGLT2) inhibitors—including dual inhibitors are used [17] [18]. The PROTECT trial showed that 400 mg of the DEARA sparsentan taken once daily reduced proteinuria and preserved kidney function compared with maximally titrated irbesartan in patients with IgAN [33]. Based on this, sparsentan is recommended by the KDIGO 2025 guidelines [17] in patients with IgAN who are at risk of progressive loss of kidney function. Likewise, KDIGO 2025 guidelines recommend the use of SGLT2 inhibitors based on pre-specified analyses of trials of DAPA-CKD (dapagliflozin) [34] and EMPA-KIDNEY (empagliflozin) [35] studies, which were conducted in a broad range of patients with CKD, including those with IgAN. The pre-specified interim analysis of the ALIGN trial showed that 0.75 mg of the ERA atrasentan per day resulted in significantly and clinically reduced proteinuria versus placebo in patients with biopsy-proven IgAN [36], and its recent approval in the UAE (preceded by the approval in the US by the U.S. Food and Drug Administration), allows its incorporation into local treatment algorithms. Additional molecules under investigation for controlling existing CKD in IgAN include the ERA SC0062, and the mineralocorticoid receptor antagonist finerenone, which is currently used in patients with CKD associated with type 2 diabetes [31].

We propose an initial standardized approach across the UAE to control existing CKD in IgAN by using maximally tolerated RAS inhibitors with or without an ERA or DEARA, and consideration of adding an SGLT2 inhibitor (**Figure 2**). It is important to note, however, that therapy restricted solely to managing CKD alone should only be considered as a short-term option for low-risk patients in a "wait and watch" approach, because patients restricted to these therapies do not tend to do well longer term, and it is anticipated that these agents may need to be used in conjunction with immune-targeting agents.

Consensus statement 5: *Treatment algorithms should be reviewed and updated annually to facilitate the incorporation of emerging therapies into care pathways*

Evidence label: *Trial supported*

The number of treatment options for IgAN was limited until relatively recently. There are now a range of treatments across different classes that are recently FDA approved (or expected soon) or undergoing evaluation in clinical trials, with the potential for a dramatic expansion in the treatment landscape in the coming years. As such, it is important that treatment algorithms in the UAE are reviewed every year to ensure that the latest available treatments are incorporated into the algorithm. This will ensure treatments can be personalized wherever possible to suit individual patient profiles.

One area that is undergoing substantial clinical evaluation is the complement pathway. This plays a key role in IgA pathogenesis, driving both glomerular inflammation and mesangial injury, and deposition of C3 is a hallmark of IgAN [18]

[28] [31] [32]. Iptacopan specifically inhibits the alternative complement pathway by targeting factor B, and an oral dose of 200 mg twice daily was shown to reduce proteinuria in patients with biopsy-proven IgAN in the APPLAUSE-IgAN trial [37], resulting in its approval for use in the UAE. Several other complement inhibitors are also undergoing evaluation in Phase 2 and Phase 3 trials, including antisense inhibitors of complement B (sefaxersen; RO7434656) and C5 (cemdisiran), inhibitors of complement D (vemircopan), complement C3 (pegce tacoplan), complement C5 (ravulizumab) and complement C5a (avacopan) [18] [28] [31] [32]. Complement inhibitors serve as a useful alternative to corticosteroids, particularly in patients resistant to corticosteroids or in whom a steroid-sparing regimen is required, and will broaden the treatment armamentarium to facilitate a more personalized approach to IgAN treatment [18].

B-cell modification therapies are another class of treatments that are undergoing extensive evaluation in patients with IgaN [18] [28] [31] [32]. The APRIL and BAFF cytokines stimulate B-cell activation, maturation and proliferation, thus ultimately promoting the secretion of immunoglobulins including Gd-IgA1. Sibeprenlimab is an APRIL inhibitor that has recently been evaluated in the Phase 3 VISIONARY trial [38]. That study showed that 400 mg subcutaneous sibeprenlimab every 4 weeks resulted in a significant reduction in 24-hour urinary protein-to-creatinine ratio versus placebo after 9 months of treatment [38], and resulted in its approval by the FDA for the treatment of primary IgAN. A second B-cell modification therapy, the APRIL/BAFF dual inhibitor atacicept, has recently received FDA Breakthrough Therapy Designation for the treatment of IgAN (as of January 2026) based on the results of the ORIGIN 3 trial [39]. That trial showed that a once-weekly subcutaneous injection of atacicept was superior to placebo in reducing proteinuria after the 36-week interim analysis [39].

Additional B-cell modification therapies under evaluation in Phase 2 and Phase 3 trials include the APRIL inhibitor zigakibart and the APRIL/BAFF dual inhibitors telitacicept and povetacicept, with Phase 3 data suggesting promising reductions in Gd-IgA1, proteinuria and slowing of kidney function loss in patients with IgAN [18] [28] [31] [32]. Other B-cell modifying therapies include CD38-depleting monoclonal antibodies (felzartamab, mezagitamab), as CD38 is thought to be closely involved in the formation of Gd-IgA1, and the proteasome inhibitor bortezomib, which results in plasma cell depletion [18] [28] [31] [32]. Identification of which patients are most likely to respond to B-cell modification therapies will be key to incorporation of these agents into treatment algorithms upon first approval.

When considering how to personalize the treatment algorithm for IgAN, supportive care with RASi, SGLT2 inhibitor, and lifestyle modifications should be recommended for all patients regardless of risk category (**Figure 2**). TR-budesonide should be used in patients with persistent proteinuria (≥0.5 - 1 g/day) despite optimized supportive care and evidence of active mucosal-driven disease and can be useful when systemic steroid sparing is desired. Systemic glucocorticoids can also

be used in high-risk patients when TR-budesonide or targeted therapy are unavailable or contraindicated. Mycophenolate mofetil is potentially useful as a corticosteroid-free immunosuppressant, particularly in patients of Chinese origin [17]. A complement inhibitor may be used in patients with high-grade proteinuria, rapid EGFR decline, or biopsy evidence of complement activation or patients who are steroid-resistant or steroid-intolerant.

An overview of ongoing molecules being evaluated in IgAN Phase 2 and 3 clinical trials is summarized in **Table 3**. The potential for new available treatments underpins our recommendation for an annual review of the treatment algorithm; however, it is acknowledged that this may be logistically challenging. As such, we then advocate a review of the treatment algorithm every two years at a minimum.

The proposed treatment algorithm is endorsed by EMAN-T and accordingly should be incorporated into care pathways in UAE. This will involve endorsement and adoption by the ministries of health and major private healthcare providers. The algorithm should be integrated into electronic medical records and supported by clinical decision prompts to standardize practice across centers. If feasible, an early engagement with the pharmaceuticals and therapeutics committee will streamline formal approval and reimbursement of therapy. To encourage adoption of the national treatment algorithm, CME-accredited education should be provided to nephrologists, pharmacists and PCPs to ensure correct patient selection and monitoring. The treatment algorithm should be piloted through designated Centers of Excellence to refine implementation and collect national data on the therapy. Investment in such centers may also facilitate enrollment in clinical trials, enabling new treatments to reach patients in a timely manner. It is important to ensure equitable access to the treatment algorithm and avoid unnecessary delays.

It is anticipated that multiple new treatments in the IgAN landscape have the potential to shift care from supportive and systemic steroid treatment to mechanism-based target therapy which will improve precision and may lead to a better outcome. There will be an increased ability to reduce proteinuria, potentially slow eGFR decline, and delay ESKD in more patients, with the additional potential to reduce long-term dialysis. However, new therapies will introduce greater complexity in clinical decision-making, requiring patient stratification and a standardized treatment algorithm. Investing in developing specialized centers for the care of patients with IgAN, and other glomerular disorders, that enroll patients to local and international clinical trials will be an important step in improving patient access to new therapies. Once approved, new treatments can incur health economic considerations and may have an impact on hospital budgets; studies should be conducted to analyze this impact and recognize the benefits of increased patient health to national healthcare capacity. Lastly, as with all new treatments, they should be monitored to evaluate efficacy, safety and cost-effectiveness as they are introduced into clinical use.

Table 3. Ongoing IgAN clinical trials that may impact future treatment algorithms in the UAE.

Treatment	Mechanism of Action	Phase	Clinical Trial Identifier
SC0062	ERA	Ph 3	NCT06819826
Finerenone	MRA	Ph 3	NCT05047263
Sefaxersen	Complement B inhibitor (antisense)	Ph 3	NCT05797610
Cemdisiran	Complement C5 inhibitor (antisense)	Ph 2	NCT03841448
Vemircopan	Complement D inhibitor	Ph 2	NCT05097989
Pegcetacoplan	Complement C3 inhibitor	Ph 2	NCT03453619
Ravulizumab	Complement C5 inhibitor	Ph 3	NCT06291376
Avacopan	Complement C5a inhibitor	Ph 2	NCT02384317
Sibeprenlimab	APRIL inhibitor	Ph 3	NCT05248646
Zigakibart	APRIL inhibitor	Ph 3	NCT05852938
Atacicept	APRIL/BAFF dual inhibitor	Ph 3	NCT04716231
Telitacicept	APRIL/BAFF dual inhibitor	Ph 3	NCT05799287
Povetacicept	APRIL/BAFF dual inhibitor	Ph 3	NCT06564142
Felzartamab	CD38-depleting agent	Ph 3	NCT06935357
Mezagitamab	CD38-depleting agent	Ph 3	NCT06963827
Bortezomib	Plasma cell-depleting agent	Ph 4	NCT01103778

APRIL, a proliferation-inducing ligand; BAFF, B-cell activating factor; ERA, endothelin receptor antagonist; IgAN, immunoglobulin A nephropathy; MRA, mineralocorticoid receptor antagonist; Ph, phase; UAE, United Arab Emirates.

3.3. Improving Disease Awareness and Education

Consensus statement 6: *Ongoing education on best practice and advances in IgAN management should be a major goal, due to the potential to improve care pathways and ensure an aligned approach among all relevant stakeholders in the UAE*

Evidence label: Expert opinion

Awareness of IgAN pathophysiology is generally low among PCPs and nephrologists in the UAE. We have outlined key areas from diagnosis through ongoing management that we believe are important focus points for targeted educational activities to achieve maximum effect with regard to optimizing IgAN care pathways.

Achieving an initial diagnosis is often the first hurdle in the IgAN pathway, with many nephrologists unaware that the threshold for proceeding to biopsy is lowered for suspected IgAN. As such, education is needed on the importance of early biopsy and timely treatment so that these can be more firmly integrated into clinical practice. Similarly, a greater awareness of the most at-risk profiles is needed, including recognizing the increased risk among South Asian expatriate populations compared with UAE nationals. As PCPs are often first to encounter patients with IgAN, they play a large role in early recognition and diagnosis of the disease and accordingly should be targeted in educational programs. The key role of PCPs

is even more relevant considering that there are only around 100 nephrologists in the UAE, which is insufficient to diagnose a disease accounting for upwards of 20% of all glomerular nephritis cases [16]. Integral to this, widespread education on the importance of routine urine analysis needs to be more firmly established in primary care, and future implementation of mass screening programs would be beneficial given high prevalence of IgAN.

At the prognosis stage, education is needed to overcome the common misconception among nephrologists that IgAN is not a severe disease, and the risks of progression to ESKD and associated mortality risks need to be more firmly appreciated. In particular, the importance of proteinuria thresholds should be integrated into daily clinical practice, specifically involving greater utilization of proteinuria as a prognostic marker and a shift from the old mindset that levels < 1 g/day indicate a mild disease. In addition to increased awareness of the importance of such prognostic tools, a greater appreciation of the heterogenous nature of disease should be encouraged. IgAN must be recognized among nephrologists as a broad-spectrum disorder, and greater clarity on how to classify and manage diverse patient profiles would be beneficial. Importantly, a greater focus should be placed on early treatment to prevent rapid progression, with a clear understanding of personalized medicine approaches to IgAN with regard to targeting specific pathophysiological profiles. Historically, the lack of biomarkers has led to an overreliance on repeat kidney biopsy to guide treatment, but this approach is not sustainable and additional measures of disease are needed.

To achieve some of these educational goals, we advocate for the development of a practical toolkit, or pocket guide, to aid nephrologists from diagnosis through prognosis and treatment. To facilitate a standardized care pathway across the UAE, a greater establishment of networks is needed between care centers, as well as regular meetings to discuss IgAN management and to share case studies. Current virtual meeting series organized by SEHA Kidney Care and physical and virtual meetings organized by EMAN-T (including the annual glomerulonephritis conference) should be further advertised and could be opened to the wider nephrology network to encourage broader participation, and this could be underpinned by greater collaboration between societies and hospitals.

At the patient level, societies are an excellent way to provide patient educational materials as well as to increase patient voice, for example leaflets in multiple languages to highlight "Understanding IgAN". These can also have a major impact on policymakers, which can increase awareness of disease for reimbursement reasons. Often, new treatments are expensive, and insurance companies may be reluctant to approve the medication for reimbursement purposes. Studies highlighting cost-effectiveness are often needed, and particularly when placed in the context of future potential dialysis costs, would generally convince payers on the benefits of shorter-term high-cost medications if dialysis could be delayed by 10 years. However, this becomes less justifiable with expatriate patients, who may not remain in the UAE indefinitely. Local guidelines and endorsements from national

bodies (e.g., DHA, MoH, Abu Dhabi regulators) would be important to validate the importance of reimbursing treatment costs among policymakers, making it difficult for insurers to deny coverage, and the EMAN-T Society will be critical in this process.

4. Summary and Conclusion

The UAE has seen a steady rise in the number of IgAN diagnoses over the last several decades, increasing the urgency with which solutions are needed to optimize clinical management pathways. We have presented a multi-pronged approach that outlines the vision of EMAN-T to address this need. We highlight red flags and IgAN patient profiles that should raise immediate clinical suspicion and offer specific recommendations to enable a more rapid recognition and diagnosis of IgAN across the UAE. Implementation of these recommendations should be accompanied by quality improvement metrics (e.g., time from referral to biopsy, percentage of patients achieving proteinuria <0.5 g/day at 12 months). A limitation of this publication should be acknowledged that the expert panel has no patient or payer representation and is comprised of only twelve nephrologist experts. Future iterations of this consensus should include patient advocacy group input and health economics expertise. With increased education and awareness among PCPs and nephrologists, we suggest early and effective risk stratification and treatment is an area with substantial potential to improve outcomes by limiting the number of patients progressing to ESKD. With an increasing number of effective treatments now available and more approvals expected in the near future, it is more important than ever for clinicians to keep abreast of the evolving IgAN treatment landscape. As such, we strongly favor a national treatment algorithm with regular reviews to ensure the latest treatments are included as appropriate among ongoing clinical management options. The generalizability of our algorithm and recommendations to other Gulf nations remains to be seen. However, we believe that adopting these recommendations, along with a national focus on improving disease awareness and education among clinicians and patients, will serve to improve the care and ultimately extend lives of patients with IgAN in the UAE.

Acknowledgements

This consensus article was initiated by the EMAN-T society. The authors had full control of the content, and the article reflects their opinions. The authors acknowledge John Bett and Laura D'Castro from Astraeus Medical Ltd for editorial support in the preparation of this manuscript, with funding for medical writing support provided by Novartis. All authors met the International Committee of Medical Journal Editors authorship criteria.

Funding Statement

The authors declare financial support was provided by Novartis for editorial sup-

port in the development of this article. The funder was not involved in the statement drafting, voting, the writing of this article, or the decision to submit it for publication.

Author Contributions

All authors made a significant contribution to the work reported, whether that was in the conception, acquisition of data, analysis and interpretation, in all these areas, took part in drafting, revising, critically reviewing the article, gave final approval of the version to be published, have agreed on the journal to which the article has been submitted, and agree to be accountable for all aspects of the work.

Conflicts of Interest

Dr. Dana Kidder declares payment of honoraria from Novartis, Sobi, GSK, and AstraZeneca, and support for attending meetings from Sob. Dr. Shaikha Ali declares payment of honoraria, expert testimony, and support for attending meetings from Novartis. No other authors have any conflicts to declare.

References

[1] Pitcher, D., Braddon, F., Hendry, B., Mercer, A., Osmaston, K., Saleem, M.A., *et al.* (2023) Long-Term Outcomes in IgA Nephropathy. *Clinical Journal of the American Society of Nephrology*, **18**, 727-738. https://doi.org/10.2215/cjn.0000000000000135

[2] Rout, P. and Afzal, M. (2025) IgA Nephropathy (Berger Disease). StatPearls Publishing.

[3] Willey, C.J., Coppo, R., Schaefer, F., Mizerska-Wasiak, M., Mathur, M. and Schultz, M.J. (2023) The Incidence and Prevalence of IgA Nephropathy in Europe. *Nephrology Dialysis Transplantation*, **38**, 2340-2349. https://doi.org/10.1093/ndt/gfad082

[4] Lee, H., Kim, D.K., Oh, K., Joo, K.W., Kim, Y.S., Chae, D., *et al.* (2012) Mortality of IgA Nephropathy Patients: A Single Center Experience over 30 Years. *PLOS ONE*, **7**, e51225. https://doi.org/10.1371/journal.pone.0051225

[5] Knoop, T., Vikse, B.E., Svarstad, E., Leh, S., Reisæter, A.V. and Bjørneklett, R. (2013) Mortality in Patients with IgA Nephropathy. *American Journal of Kidney Diseases*, **62**, 883-890. https://doi.org/10.1053/j.ajkd.2013.04.019

[6] Hastings, M.C., Bursac, Z., Julian, B.A., Villa Baca, E., Featherston, J., Woodford, S.Y., *et al.* (2018) Life Expectancy for Patients from the Southeastern United States with IgA Nephropathy. *Kidney International Reports*, **3**, 99-104. https://doi.org/10.1016/j.ekir.2017.08.008

[7] Jarrick, S., Lundberg, S., Welander, A., Carrero, J., Höijer, J., Bottai, M., *et al.* (2019) Mortality in IgA Nephropathy: A Nationwide Population-Based Cohort Study. *Journal of the American Society of Nephrology*, **30**, 866-876. https://doi.org/10.1681/asn.2018101017

[8] Hassler, J.R. (2020) IgA Nephropathy: A Brief Review. *Seminars in Diagnostic Pathology*, **37**, 143-147. https://doi.org/10.1053/j.semdp.2020.03.001

[9] Stamellou, E., Seikrit, C., Tang, S.C.W., Boor, P., Tesař, V., Floege, J., *et al.* (2023) IgA Nephropathy. *Nature Reviews Disease Primers*, **9**, Article No. 67.

https://doi.org/10.1038/s41572-023-00476-9

[10] Ghani, A.A., Al Waheeb, S., Al Homoud, E., Al Helal, B. and Hussain, N. (2011) Clinical and Histopathological Spectrum of IgA Nephropathy in Kuwait. *Annals of Saudi Medicine*, **31**, 152-157. https://doi.org/10.4103/0256-4947.77491

[11] Alwahaibi, N., Al-Khazimi, O. and Al-Riyami, M. (2022) Histopathological Spectrum of Glomerular Diseases in Oman: A Five-Year Study. *Saudi Journal of Kidney Diseases and Transplantation*, **33**, 425-431. https://doi.org/10.4103/1319-2442.385966

[12] Arrayed, A.A., Shariff, S. and Maamari, M.M.A. (2007) Kidney Disease in Bahrain: A Biopsy-Based Epidemiological Study. *Transplantation Proceedings*, **39**, 875-878. https://doi.org/10.1016/j.transproceed.2007.03.079

[13] Mitwalli, A.H. (2000) Glomerulonephritis in Saudi Arabia: A Review. *Saudi Journal of Kidney Diseases and Transplantation*, **11**, 567-576.

[14] Yahya, T.M., Pingle, A., Boobes, Y. and Pingle, S. (1998) Analysis of 490 Kidney Biopsies: Data from the United Arab Emirates Renal Diseases Registry. *Journal of Nephrology*, **11**, 148-150.

[15] Alhadari, A.K., Alalawi, F.J., Seddik, A.A., Zahra, K., Kumar, D., Yousif, H., *et al.* (2017) Pattern of Acute Glomerulonephritis in Adult Population in Dubai: A Single-Center Experience. *Saudi Journal of Kidney Diseases and Transplantation*, **28**, 571-578. https://doi.org/10.4103/1319-2442.206443

[16] Al Shamsi, H., El Tahrawi, R., Hindawy, B., Khater, E. and Hassan, M. (2020) Multicenter Review of Glomerular Diseases in the Emirates of Abu Dhabi: Six Years' Experience. *Saudi Journal of Kidney Diseases and Transplantation*, **31**, 1069-1077. https://doi.org/10.4103/1319-2442.301173

[17] Rovin, B.H., Barratt, J., Cook, H.T., Noronha, I.L., Reich, H.N., Suzuki, Y., *et al.* (2025) KDIGO 2025 Clinical Practice Guideline for the Management of Immunoglobulin A Nephropathy (IgAN) and Immunoglobulin A Vasculitis (IgAV). *Kidney International*, **108**, S1-S71. https://doi.org/10.1016/j.kint.2025.04.004

[18] Ramsawak, S., Cohen, S., Linares, A. and Cavanaugh, C. (2025) IgA Nephropathy: Update on Pathogenesis and Treatment. *Cleveland Clinic Journal of Medicine*, **92**, 373-383. https://doi.org/10.3949/ccjm.92a.24105

[19] Aseem, S. and Zheng, S. (2024) Immunoglobulin A Nephropathy: A Review. *The Permanente Journal*, **28**, 87-94. https://doi.org/10.7812/tpp/24.089

[20] Wyatt, R.J. and Julian, B.A. (2013) IgA Nephropathy. *New England Journal of Medicine*, **368**, 2402-2414. https://doi.org/10.1056/nejmra1206793

[21] Gliklich, R.E., Dreyer, N.A. and Leavy, M.B. (2014) Patient Registries. Registries for Evaluating Patient Outcomes: A User's Guide. 3rd Edition, Agency for Healthcare Research and Quality (US).

[22] Rauen, T., Eitner, F., Fitzner, C., Sommerer, C., Zeier, M., Otte, B., *et al.* (2015) Intensive Supportive Care Plus Immunosuppression in IgA Nephropathy. *New England Journal of Medicine*, **373**, 2225-2236. https://doi.org/10.1056/nejmoa1415463

[23] Rauen, T., Wied, S., Fitzner, C., Eitner, F., Sommerer, C., Zeier, M., *et al.* (2020) After Ten Years of Follow-Up, No Difference between Supportive Care Plus Immunosuppression and Supportive Care Alone in IgA Nephropathy. *Kidney International*, **98**, 1044-1052. https://doi.org/10.1016/j.kint.2020.04.046

[24] Lv, J., Wong, M.G., Hladunewich, M.A., Jha, V., Hooi, L.S., Monaghan, H., *et al.* (2022) Effect of Oral Methylprednisolone on Decline in Kidney Function or Kidney Failure in Patients with IgA Nephropathy: The TESTING Randomized Clinical Trial. *JAMA*, **327**, 1888-1898. https://doi.org/10.1001/jama.2022.5368

[25] Stamellou, E., Bruchfeld, A., Caravaca-Fontan, F., Frangou, E., Kronbichler, A., Mirioglu, S., *et al.* (2025) The 2025 KDIGO IgA Nephropathy Guideline Update: An ERA Immunonephrology Working Group Perspective. *Clinical Kidney Journal*, **18**, sfaf324. https://doi.org/10.1093/ckj/sfaf324

[26] Wang, H., He, L., Feng, L., Zhang, W., Liu, N. and Zhang, W. (2024) Non-Invasive Assessment of IgA Nephropathy Severity with [18F]AlF-NOTA-FAPI-04 PET/CT Imaging. *Clinical Kidney Journal*, **17**, sfae340. https://doi.org/10.1093/ckj/sfae340

[27] Klinkhammer, B.M., Lammers, T., Mottaghy, F.M., Kiessling, F., Floege, J. and Boor, P. (2021) Non-Invasive Molecular Imaging of Kidney Diseases. *Nature Reviews Nephrology*, **17**, 688-703. https://doi.org/10.1038/s41581-021-00440-4

[28] Barratt, J., Lafayette, R.A. and Floege, J. (2024) Therapy of IgA Nephropathy: Time for a Paradigm Change. *Frontiers in Medicine*, **11**, Article ID: 1461879. https://doi.org/10.3389/fmed.2024.1461879

[29] Lafayette, R., Kristensen, J., Stone, A., Floege, J., Tesař, V., Trimarchi, H., *et al.* (2023) Efficacy and Safety of a Targeted-Release Formulation of Budesonide in Patients with Primary IgA Nephropathy (NefIgArd): 2-Year Results from a Randomised Phase 3 Trial. *The Lancet*, **402**, 859-870. https://doi.org/10.1016/s0140-6736(23)01554-4

[30] Barratt, J., Lafayette, R., Kristensen, J., Stone, A., Cattran, D., Floege, J., *et al.* (2023) Results from Part a of the Multi-Center, Double-Blind, Randomized, Placebo-Controlled NefIgArd Trial, Which Evaluated Targeted-Release Formulation of Budesonide for the Treatment of Primary Immunoglobulin a Nephropathy. *Kidney International*, **103**, 391-402. https://doi.org/10.1016/j.kint.2022.09.017

[31] Yang, Y. and Xu, G. (2025) Opportunities and Challenges in the Treatment of IgA Nephropathy. *Frontiers in Pharmacology*, **16**, Article ID: 1559593. https://doi.org/10.3389/fphar.2025.1559593

[32] Coppo, R. (2025) Treating IgA Nephropathy: Looking at the Future without Forgetting the Past. *Journal of Clinical Medicine*, **14**, Article No. 4045. https://doi.org/10.3390/jcm14124045

[33] Rovin, B.H., Barratt, J., Heerspink, H.J.L., Alpers, C.E., Bieler, S., Chae, D., *et al.* (2023) Efficacy and Safety of Sparsentan versus Irbesartan in Patients with IgA Nephropathy (PROTECT): 2-Year Results from a Randomised, Active-Controlled, Phase 3 Trial. *The Lancet*, **402**, 2077-2090. https://doi.org/10.1016/s0140-6736(23)02302-4

[34] Heerspink, H.J.L., Stefánsson, B.V., Correa-Rotter, R., Chertow, G.M., Greene, T., Hou, F., *et al.* (2020) Dapagliflozin in Patients with Chronic Kidney Disease. *New England Journal of Medicine*, **383**, 1436-1446. https://doi.org/10.1056/nejmoa2024816

[35] The EMPA-KIDNEY Collaborative Group (2023) Empagliflozin in Patients with Chronic Kidney Disease. *New England Journal of Medicine*, **388**, 117-127. https://doi.org/10.1056/nejmoa2204233

[36] Heerspink, H.J.L., Jardine, M., Kohan, D.E., Lafayette, R.A., Levin, A., Liew, A., *et al.* (2025) Atrasentan in Patients with IgA Nephropathy. *New England Journal of Medicine*, **392**, 544-554. https://doi.org/10.1056/nejmoa2409415

[37] Perkovic, V., Barratt, J., Rovin, B., Kashihara, N., Maes, B., Zhang, H., *et al.* (2025) Alternative Complement Pathway Inhibition with Iptacopan in IgA Nephropathy. *New England Journal of Medicine*, **392**, 531-543. https://doi.org/10.1056/nejmoa2410316

[38] Perkovic, V., Trimarchi, H., Tesar, V., Lafayette, R., Wong, M.G., Barratt, J., *et al.* (2026) Sibeprenlimab in IgA Nephropathy—Interim Analysis of a Phase 3 Trial. *New*

England Journal of Medicine, **394**, 635-646. https://doi.org/10.1056/nejmoa2512133

[39] Lafayette, R., Barbour, S.J., Brenner, R.M., Campbell, K.N., Doan, T., Eren, N., *et al.* (2026) A Phase 3 Trial of Atacicept in Patients with IgA Nephropathy. *New England Journal of Medicine*, **394**, 647-657. https://doi.org/10.1056/nejmoa2510198