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Expert consensus on the likelihood and treatment of high- and low-inflammatory activity Immunoglobulin A nephropathy

Jai Radhakrishnan¹ · Sanjiv Anand² · Mark Haas³ · Leal Herlitz⁴ · Richard Lafayette⁵ · Tingting Li⁶ · Arvind Madan⁷ · Ali Mehdi⁸ · Sayna Norouzi⁹ · Brad Rovin¹⁰ · Shikha Wadhvani¹¹ · Cynthia Campos¹² · Justin Weiss¹² · Michael S. Broder¹² · Irina Yermilov¹²

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Abstract

Purpose Immunoglobulin A nephropathy (IgAN) is characterized by glomerular deposition of immunoglobulin A, with current treatments focused on controlling disease activity and slowing progression. In the context of expanding therapeutic options, this expert panel aimed to provide guidance on defining and managing high- and low-inflammatory activity IgAN phenotypes.

Methods A modified Delphi panel was conducted with eleven US-based nephrologists and pathologists. Participants completed two rounds of online surveys followed by an in-person meeting. Clinical scenarios were rated and analyzed using the RAND/UCLA Appropriateness Method and each were classified as appropriate, inappropriate, or uncertain. Outcomes included consensus on the likelihood of high- versus low-inflammatory activity IgAN, appropriateness of therapies, and the timing of complement inhibitor initiation relative to meningococcal B vaccination.

Results The panel reached agreement on 92% of scenarios. High-inflammatory activity IgAN was characterized by hematuria, moderate- to high-grade proteinuria, and inflammatory histology, whereas low-inflammatory activity IgAN was characterized by absence of hematuria, low-grade proteinuria, stable eGFR, and inactive histology. Panelists more frequently supported adding therapies beyond RAAS±SGLT2 inhibitors for high- versus low-inflammatory activity IgAN. Complement inhibitor initiation was considered appropriate at any point during the meningococcal B vaccination series when antibacterial prophylaxis was administered concurrently.

Conclusions This guidance offers practical recommendations for managing patients with high- and low-inflammatory activity IgAN. As therapeutic options continue to expand, ongoing research and updated consensus efforts will be essential to support decision-making. In light of new KDIGO guidelines, we believe these phenotypes can assist clinicians until more direct biomarkers are identified.

Data Sharing All data are included in the manuscript and/or supporting information.

Keywords Delphi panel · IgA nephropathy · Inflammatory activity · Treatment decision-making · Glomerular disease · Phenotype stratification

Introduction

Immunoglobulin A nephropathy (IgAN) is the most common primary glomerular disease in the world, with an estimated incidence of 2.5 per 100,000 person-years [1–3]. In IgAN, immune complexes with immunoglobulin A (IgA) as the dominant immunoglobulin are deposited in the glomeruli, causing inflammation that can ultimately impair their

filtration function [4]. IgAN presents with a wide spectrum of clinical manifestations and disease trajectories, ranging from isolated microscopic hematuria, episodic gross hematuria, asymptomatic proteinuria and hematuria to rapidly progressive glomerulonephritis and progression to end-stage kidney disease [5, 6]. Proteinuria is associated with loss of kidney function and reduction of proteinuria has been demonstrated to decrease rates of end-stage kidney

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disease (ESKD) [7]. In the United States (US), rates of ESKD range from 12.5% to 23% over a period of 3 to 3.9 years, and increase to 53% over 19 years of observation [8]. At IgAN diagnosis, 50% of adult patients have already reached chronic kidney disease (CKD) stage 3 or greater [9], highlighting the importance of minimizing the risk of developing ESKD by reducing proteinuria early on in IgAN disease management.

There is currently no cure for IgAN, although available therapies focus on targeting immunopathogenesis, reducing renal inflammation, and slowing disease progression. First-line treatment includes renin-angiotensin-aldosterone system inhibitors (RAASi) and possibly sodium-glucose cotransporter 2 (SGLT2) inhibitors, aimed at reducing proteinuria and slowing estimated glomerular filtration rate (eGFR) decline [10, 11]. Additional therapeutic options have emerged for patients at risk of disease progression. Targeted-release budesonide, a corticosteroid formulation, modulates mucosal immunity and effectively reduces proteinuria [12, 13]. Systemic corticosteroids such as prednisone or prednisolone are used less frequently given concerns regarding severe adverse events such as severe or fatal infection, new-onset or worsening diabetes mellitus, and osteonecrosis [14, 15]. More recently, alternative pathway complement inhibitors (e.g., iptacopan) and endothelin receptor antagonists (ERAs) (e.g., sparsentan, atrasentan) have entered clinical use, providing promising additional therapeutic options to the existing treatment landscape [16–19].

This study aims to build on the October 2025 KDIGO guidelines, *Clinical Practice Guideline for the Management of Immunoglobulin A nephropathy (IgAN) and Immunoglobulin A vasculitis (IgAV)* [20] by developing expert-informed guidance to define and manage the high- and low-inflammatory activity phenotypes of IgAN in the context of expanding therapeutic options. Specifically, this study attempted to provide guidance to treating clinicians on what constitutes these two phenotypes of IgAN and assess preferred treatment options for each. The recently released final version of the KDIGO guidelines [20] replaced the traditional term, “glomerular inflammation,” with “IgA-containing immune complex (IgA-IC)-mediated glomerular injury,” emphasizing that kidney damage in IgAN arises from IgA-IC deposition and the subsequent biological responses, including but not limited to, inflammation.

Methods

We used the RAND Corporation/University of California, Los Angeles (UCLA) modified Delphi panel method to systematically assess expert perspectives. The process included selecting expert clinicians, performing a focused literature

review, preparing detailed rating materials, administering an initial survey (Round 1), convening an in-person meeting to discuss divergent responses, distributing a follow-up survey (Round 2), and analyzing agreement among panelists (Fig. 1).

Our panel included 11 experts (9 nephrologists, 2 medical kidney pathologists) with an average of 22 years (range 6–39) of clinical experience managing IgAN and seeing an average of 46 IgAN patients or biopsies per year. All panelists were from the US (Table 1). The panelists received honoraria from the study sponsor, Novartis Pharmaceutical Corporation. The sponsor had no role in the study methodology or interpretation of the results and was blinded to all individual panelist ratings; only group medians were provided. Modified Delphi panels do not involve human subjects as defined in 45 Code of Federal Regulations part 46, thus institutional review board approval was not required.

Panelists reviewed a literature summary on the multi-hit hypothesis, mechanistic pathophysiology, and treatment options for IgAN compiled in September 2024 and informed by 57 published studies. A reference list of all studies included in the literature review is provided in the Supplementary Materials.

To design the rating form survey, all panelists participated in two individual video calls over four months. The rating form was updated through an iterative process, incorporating clinical insights and real-world experiences after each panelist call. The rating form included five core clinical characteristics to assess the likelihood of a scenario describing either the high- or low-inflammatory activity phenotype. Clinical characteristics included: current presence or absence of hematuria; current degree of proteinuria; current eGFR trajectory; timing of the most recent kidney biopsy; and histologic findings of the most recent kidney biopsy (Table 2).

In each of the clinical scenarios, panelists rated either the likelihood of high-inflammatory activity IgAN or the appropriateness of four treatment classes used as either mono- or combination therapy. Treatment classes included: systemic corticosteroids (e.g., prednisone, prednisolone), targeted-release budesonide, ERA-based therapies (e.g., sparsentan, atrasentan), and alternative pathway complement inhibitors (e.g., iptacopan). Although atrasentan is listed, the in-person meeting occurred prior to its approval by the FDA in April 2025. The final rating form is presented in the **Supplementary Materials**. Additionally, for each scenario, panelists were asked to assume patients had been optimized on RAASi±SGLT2i. Panelists were not asked to rate the likelihood that exclusively inflammatory lesions on a more recent biopsy is high-inflammatory activity IgAN, since a recent biopsy showing inflammatory lesions is already a strong indicator of active inflammation; adding “no hematuria”

Fig. 1 Overview of modified Delphi panel process**Table 1** Panelists' demographic and clinical experience

Characteristic	<i>n</i>
Total number of panelists	11
Sex	
Male	7 (63.6%)
Female	4 (36.4%)
Race ^a	
White	5 (45.5%)
Asian	5 (45.5%)
Middle Eastern/North African	2 (18.2%)
US geographic region	
Northeast	1 (9.1%)
South	2 (18.2%)
Midwest	4 (36.4%)
West	4 (36.4%)
Practice setting	
Academic	10 (90.9%)
Non-academic	1 (9.1%)
Median (range) years in primary specialty	22 (6–39)

^aPanelists could choose multiple races

would not have been an informative discriminator. Panelists were asked to assume that patients had access to all recommended therapies. They were instructed to base their ratings solely on the best clinical choice informed by available

evidence, without considering insurance coverage, cost, or other external constraints that may vary by patient.

In response to the late 2024 Advisory Committee on Immunization Practices (ACIP) update, which aligned the MenB-4 C dosing schedule with that of MenB-FHbp [21], panelists also evaluated the appropriateness of initiating complement inhibitor after each meningococcal vaccine dose (MenB and MenACWY), with or without concurrent antibacterial prophylaxis.

Each panelist independently completed the rating form in January 2025, where they rated the appropriateness of various treatments as well as the likelihood that different hypothetical scenarios reflected high-inflammatory activity disease. All 11 panelists completed the final rating form consisting of 396 scenarios, each rated on a nine-point scale from 1 to 9. For example, when evaluating treatments, a score of 1 indicated the intervention was inappropriate, 5 denoted uncertainty or balanced risks and benefits, and 9 indicated it was appropriate. The central tendency and spread of responses were summarized using median and range.

During an in-person meeting in February 2025, panelists were provided with a document showing their own first-round ratings, the group median, and the range of ratings. Disagreement in a scenario was defined as at least two

Table 2 Clinical characteristics considered by panelists

Clinical characteristics included in patient scenarios	Categories	Definitions
Current hematuria status	Absent	Negative hematuria on an adequately concentrated urine sample [dipstick] or below ULN on microscopic exam
	Present	Positive dipstick or above ULN on microscopic exam, excluding extraglomerular causes of hematuria
Current degree of proteinuria	Low grade	<0.5 g/24 h
	Moderate grade	0.5–1.5 g/24 h
	High grade	>1.5 g/24 h
Current eGFR trajectory	Stable	0–10% decrease in last 6 months
	Declining	>10% decrease in last 6 months
Timing of the most recent kidney biopsy	More recent	Obtained at 0–3 months
	Less recent	Obtained at around 12 months
Histologic findings of most recent kidney biopsy	Inflammatory lesions	A biopsy with inflammatory features only (e.g., E, C)
	Fibrotic lesions	A biopsy with fibrotic features only (e.g., S, T), excludes T2 lesions
	Mixed lesions	A biopsy with both inflammatory and fibrotic lesions

ULN=upper limit of normal, eGFR=estimated glomerular filtration rate, E=endocapillary hypercellularity, C=cellular/fibrocellular crescents, S=segmental glomerulosclerosis, T0-T1=tubular atrophy/interstitial fibrosis, T2= $\geq 50\%$ tubular atrophy/interstitial fibrosis

ratings in the 1–3 range and two ratings in the 7–9 range. Given this definition of agreement, it was possible to have agreement on statements where as many as two of the panelists disagreed with the rest of the panel.

Where consensus was reached, ratings were grouped into one of three categories: appropriate, uncertain, or inappropriate. During the meeting, panelists shared their rationale for their ratings and discussed definitions and categories used in the scenarios. In March 2025, panelists completed the ratings a second time. The panelists then drafted

consensus statements based on their ratings (Fig. 2). Panel consensus is summarized in the results below.

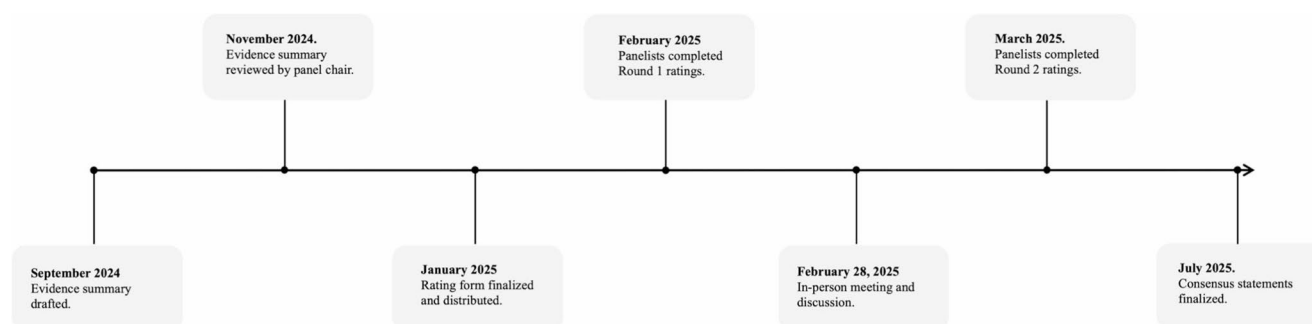
Results

The panel agreed on 92% of the second-round ratings, compared to 66% in the first round. Agreement that a scenario was appropriate or likely was defined as all but two panelists rating it 7–9; agreement that it was inappropriate or unlikely was defined as all but two rating it 1–3. The guidelines presented in Figs. 3, 4 and 5 summarize the panel's recommendations on the likelihood and treatment of high- and low-inflammatory activity IgAN, the appropriateness of various treatment options, and the appropriateness of initiating complement inhibitor after each dose of the meningococcal vaccine. Figure 3 includes recommendations for the classification of the high- and low-inflammatory activity phenotypes, Fig. 4 presents treatment recommendations for high-inflammatory activity IgAN, and Fig. 5 includes treatment recommendations for low-inflammatory activity IgAN.

If unspecified in the figure, refers to both more recent and less recent biopsies.

Treatment guidance is recommended for adult patients (≥ 18 years) with primary IgAN only and is not intended for patients with secondary IgAN or other variants such as IgAN with minimal change disease, IgAN with acute kidney injury, IgAN with rapidly progressive glomerulonephritis, active IgAN vasculitis, recurrent IgAN in a transplanted kidney, or IgAN superimposed on diabetic nephropathy. Patients were assumed not to have severe and irreversible kidney damage, acute illness, any relative contraindications for the medications of interest, or to be pregnant.

Every clinical scenario is different, with its own set of complex characteristics. In practice, many other clinical and non-clinical factors, beyond those addressed below, can influence how high- and low-inflammatory activity IgAN are classified and treated. For instance, we assume that

**Fig. 2** Study timeline

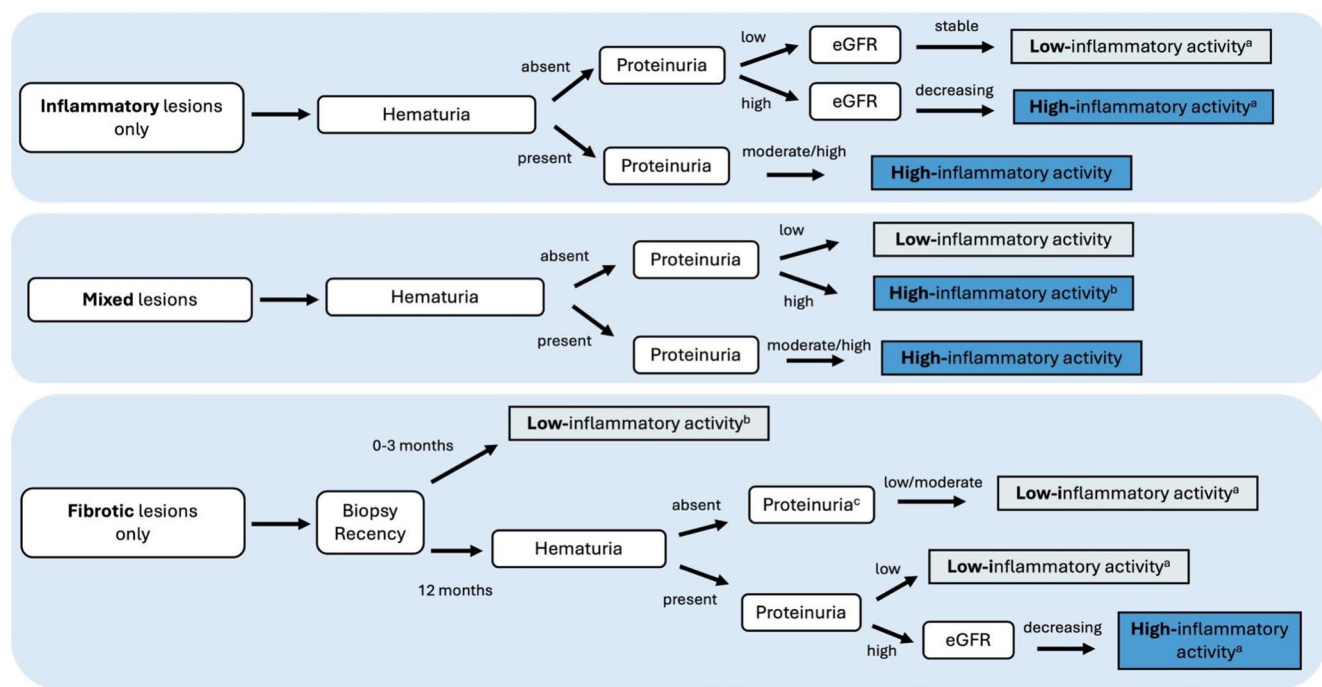


Fig. 3 IgAN by inflammatory phenotype. eGFR: estimated glomerular filtration rate. ^a Less recent biopsy only. ^b More recent biopsy only

patients are adherent to their care plan and are not subject to health insurance restrictions or financial limitations.

IgAN phenotypes

While IgAN is inherently an inflammatory disease, “high-inflammatory activity phenotype” is intended to distinguish a subset of patients with a heightened degree of activity or inflammation, and thus a higher likelihood of disease progression, who would benefit most from immunomodulatory interventions. The high-inflammatory activity phenotype is often associated with hematuria, moderate- to high-grade proteinuria, and inflammatory biopsy features including endocapillary hypercellularity and cellular/fibrocellular crescents.

The “low-inflammatory activity phenotype” refers to patients with histologic and clinical features suggestive of limited or no inflammation – individuals less likely to benefit from intensive immunomodulatory activity. The low-inflammatory activity phenotype is often associated with an absence of hematuria, low-grade proteinuria, relatively stable eGFR, and inactive or often fibrotic biopsy findings, including segmental glomerulosclerosis, tubular atrophy and interstitial fibrosis. More details on the phenotypes can be found in Fig. 3. Scenarios for which the panel did not reach consensus are not included in the figure. For example, in some scenarios, the panel did not reach consensus on how to categorize patients with moderate proteinuria in

combination with other features, and such cases were therefore excluded from the table.

High-inflammatory activity phenotype treatment recommendations

The panel supports the use of targeted-release budesonide or alternative pathway complement inhibitors as monotherapies, as well as ERA-based therapies in combination with targeted-release budesonide, for patients with this phenotype. For patients with inflammatory or mixed (inflammatory and fibrotic) lesions, the panel also supports combination therapy with alternative pathway complement inhibitors and ERA-based therapies, particularly in those with high-grade proteinuria. Additional treatment recommendations on how to treat this phenotype are provided in Fig. 4.

Low-inflammatory activity phenotype treatment recommendations

In patients with the low-inflammatory phenotype, panelists generally do not support the use of additional treatments beyond RAASi±SGLT2i. However, ERA monotherapy is recommended for patients with the following two combinations of characteristics: (1) fibrotic lesions, absent hematuria, and moderate- to high-grade proteinuria, and (2) fibrotic lesions, hematuria, moderate proteinuria, and stable eGFR. We provide further recommendations on how to treat this phenotype in Fig. 5. Illustrative examples of patient

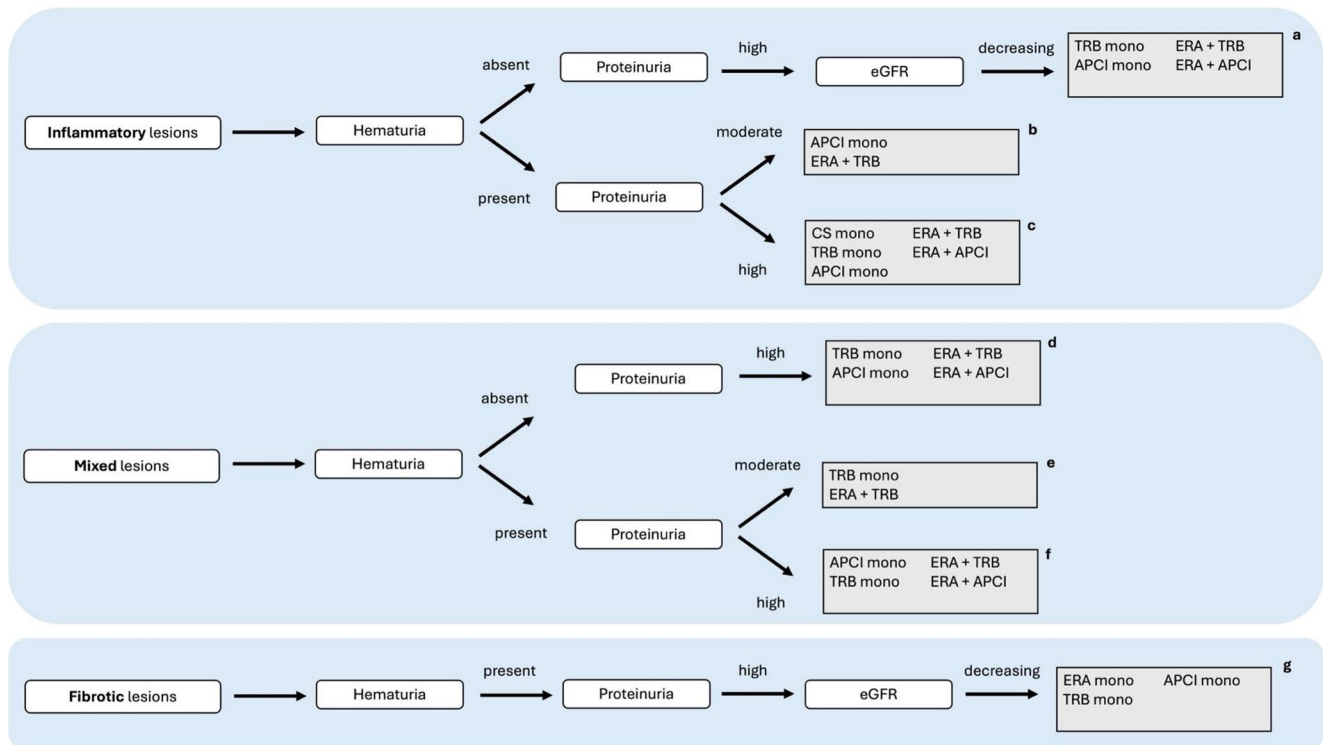


Fig. 4 Consensus treatment recommendations for the high-inflammatory activity phenotype. APCI: alternative pathway complement inhibitors, CS: systemic corticosteroids, eGFR: estimated glomerular filtration rate, ERA: endothelin receptor antagonists, mono: monotherapy, TRB: targeted-release budesonide. ^a If eGFR is decreasing, TRB monotherapy is also supported. ^b If eGFR is decreasing, TRB+APCI is also supported. ^c If eGFR is decreasing, APCI monotherapy and ERA+APCI are also supported; if eGFR is stable, ERA monotherapy is also supported. ^d If eGFR is decreasing, ERA+CS and TRB+APCI are also supported; if eGFR is stable, ERA monotherapy is also sup-

ported. ^e This scenario is only considered part of the high-inflammatory activity phenotype when the biopsy is older (~12 months ago). ^f This scenario is only considered part of the high-inflammatory activity phenotype when the biopsy is recent (0–3 months). All other scenarios are considered inflammatory regardless of biopsy timing (e.g., within the past 0–3 months or ≥12 months ago). ^g This scenario is only considered part of the inflammatory IgAN phenotype when the biopsy is less recent (>12 months ago). Panelists did not consider more recent biopsies (0–3 months) with inflammatory lesions and absent hematuria because that was considered incongruent with real-world data.

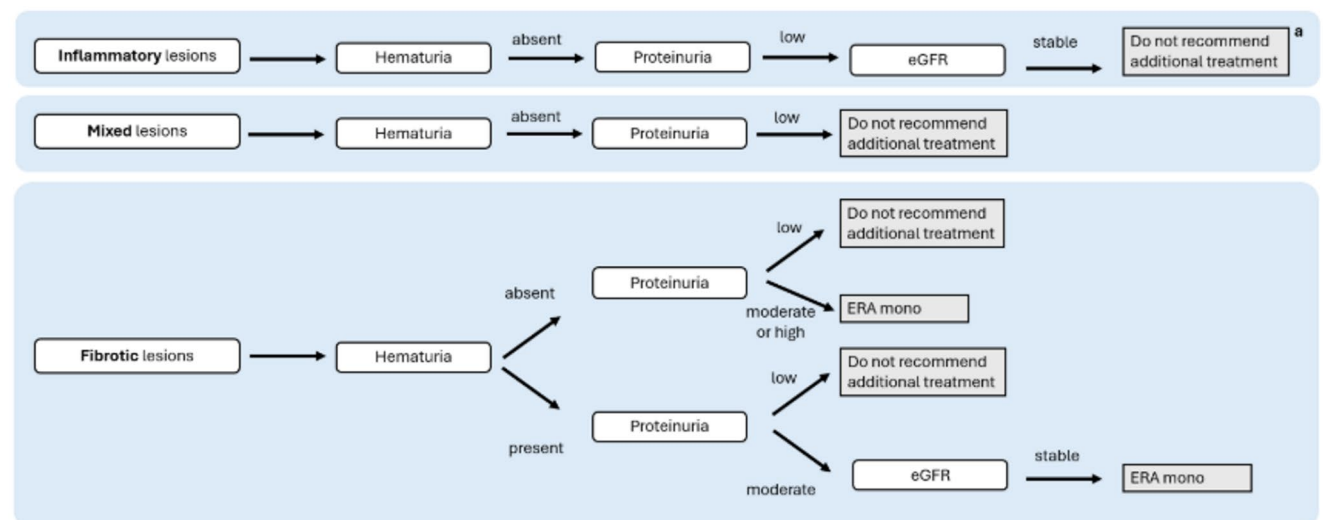


Fig. 5 Consensus treatment recommendations for the low-inflammatory activity phenotype. eGFR: estimated glomerular filtration rate, ERA: endothelin receptor antagonist, mono: monotherapy. ^a This scenario is only considered part of the fibrotic or low-inflammatory activ-

ity phenotype when the biopsy is older (~12 months). All other scenarios are considered fibrotic or low-inflammatory activity regardless of biopsy timing (e.g., within the past 0–3 months or ~12 months ago).

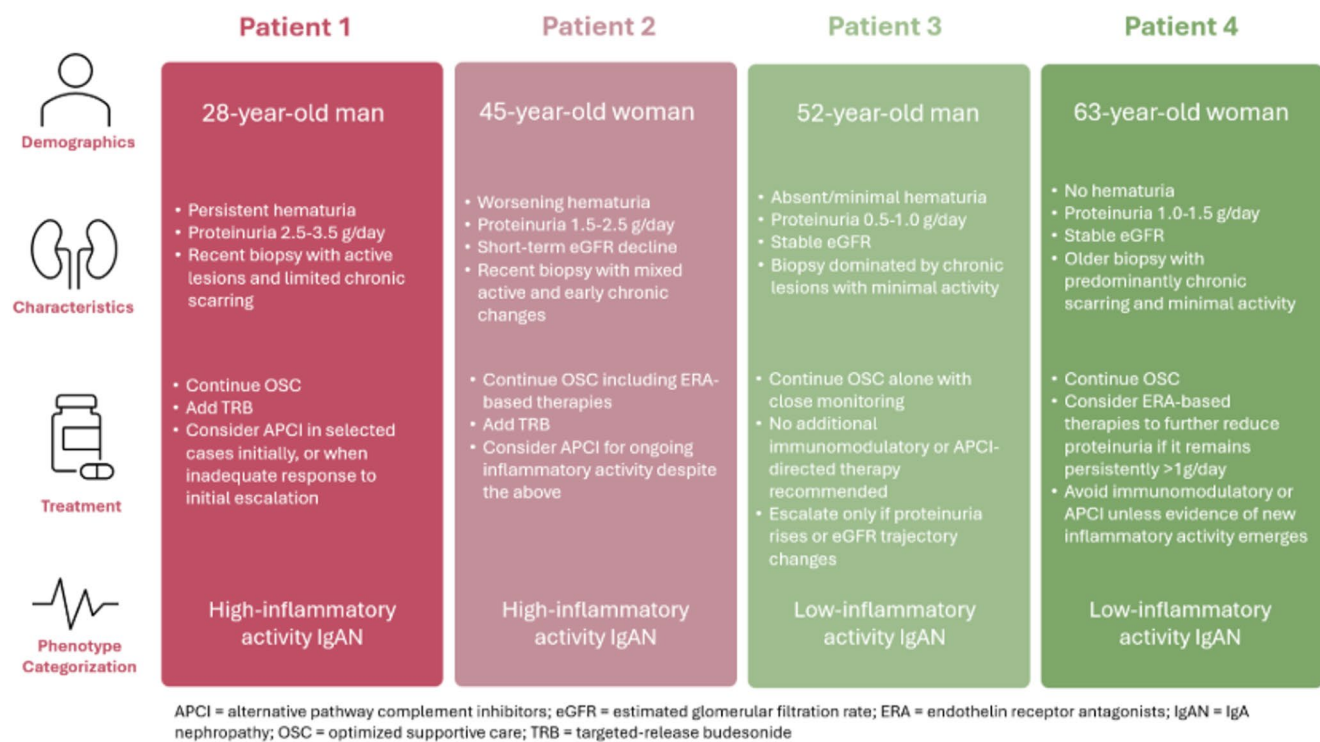


Fig. 6 Examples of patient categorizations

categorizations into either high- or low-inflammatory IgAN can be found in Fig. 6.

Timing of meningococcal serogroup B vaccination when initiating complement inhibition

Panelists also assessed the appropriateness of initiating complement inhibitor at various points during the meningococcal vaccination series (MenB and MenACWY). Panelists agreed that vaccination decisions should follow the Fall 2024 recommendations of the Advisory Committee on Immunization Practices (ACIP) for patients initiating complement inhibitor. They further recommended that complement inhibitor initiation may be considered at any time during the meningococcal B vaccination series, provided that antibacterial prophylaxis is administered concurrently. The panel was unable to come to consensus on the need for long-term antibacterial prophylaxis. Some panelists favored discontinuing antibacterial prophylaxis after the third dose, while others preferred to extend antibiotic coverage beyond the third dose to ensure full protection. However, panelists agreed that patients should remain on antibiotics through at least the second meningococcal vaccine administration.

Discussion

Recognition of the evolving therapeutic landscape for IgAN is critical for optimizing patient care. As multiple new therapeutic classes emerge, clinicians must increasingly make treatment decisions in the absence of phenotype-stratified or head-to-head comparative trial evidence. This consensus was developed to provide a structured, clinically grounded IgAN phenotype framework to support decision-making by organizing patients according to patterns of disease activity and progression risk.

Importantly, this framework is intended as a pragmatic clinical tool rather than a mechanistic redefinition of IgAN pathophysiology. Recent *Kidney Disease: Improving Global Outcomes* (KDIGO) guidance has appropriately shifted emphasis from a loosely defined concept of “glomerular inflammation” toward “IgA immune complex-mediated glomerular injury,” reflecting the central role of immune complex deposition and downstream injury pathways [20]. This evolution highlights that IgA immune complexes can drive kidney damage through both inflammatory and non-inflammatory mechanisms, including complement activation, podocyte injury, and predominantly profibrotic pathways leading to nephron loss.

In this context, the high- and low-inflammatory activity categories proposed here should be understood as a clinical simplification of a broader and more complex biological

spectrum. They are not intended to capture all pathogenic mechanisms of IgAN, nor to imply that inflammatory features fully define disease activity or treatment responsiveness. Rather, they provide a structured way to integrate routinely available clinical and histological features, such as hematuria, proteinuria, eGFR trajectory, and biopsy findings, into a practical assessment of short-term risk and perceived disease activity.

The panel recognized that IgAN progression is not exclusively driven by overt inflammatory lesions. Patients with few to no inflammatory features may still experience progressive nephron loss due to persistent immune complex-mediated injury and profibrotic remodeling. This is consistent with KDIGO's emphasis on immune-complex driven glomerular injury as the mechanism of disease [20]. Accordingly, the framework presented here should be interpreted as complementary to, rather than a replacement for, mechanistic models of IgAN pathogenesis. It reflects how clinicians currently synthesize imperfect clinical features when selecting among expanding therapeutic options.

Within this framework, patients with high-inflammatory activity IgAN were characterized by features traditionally associated with active injury, such as hematuria, high-grade proteinuria, and inflammatory biopsy features, while low-inflammatory activity IgAN was characterized by absent hematuria, low-grade proteinuria, stable eGFR, and inactive, often fibrotic histology. These distinctions informed a risk-based prescribing strategy following supportive care (RAAS inhibition $\pm$ SLGT2 inhibition), in which therapy selection was aligned with clinical and histologic risk markers.

For high-inflammatory activity IgAN, the panel supported ERA-based therapies, targeted-release budesonide, or alternative pathway complement inhibitors, alone or in combination depending on disease severity. For low-inflammatory activity IgAN, ERA-based therapies were more frequently recommended, and immunomodulatory therapies were generally discouraged in the absence of features suggesting active disease. Across scenarios, the implicit goal of treatment was reduction of proteinuria and stabilization of kidney function in higher-risk patients, while avoiding overtreatment in lower-risk presentations. It is important to emphasize that currently available randomized controlled trial data do not stratify efficacy by histologic activity, hematuria status, or combined clinical phenotypes in a way that validates phenotype-driven treatment selection. Accordingly, the framework presented here should not be interpreted as evidence that these phenotypes predict differential therapeutic response. Rather, it reflects how clinicians already integrate multiple imperfect markers of disease activity in real-world decision-making, in the context of

expanding therapeutic options and incomplete biological stratification tools.

Our phenotype framework also builds on KDIGO guidance by translating the concept of immune complex-mediated glomerular injury into operational clinical categories that can be applied using routinely available data. While KDIGO appropriately emphasizes mechanistic pathways, clinical practice still requires translation of these concepts into actionable decisions at the patient level. The proposed categories are intended to bridge the gap between these pathways and real-world practice.

These treatment recommendations must be considered in the context of accessibility and affordability, particularly in resource-limited settings. In such circumstances, the panelists acknowledged that systemic corticosteroids may be more readily available and, therefore, may represent an appropriate treatment option. These recommendations are also inherently limited by the available evidence base. At the time of the panel, no head-to-head clinical trials had directly compared the safety and efficacy of corticosteroids with targeted-release budesonide or alternative pathway complement inhibitors. Furthermore, corticosteroid use in patients with IgAN has been associated with an increased risk of adverse events, including osteoporosis, hypertension, and diabetes, and infection rates have been reported to be 2.6 to 3.5 times higher among patients receiving corticosteroids than among those not receiving corticosteroids [22]. Consequently, although corticosteroids may remain an important treatment option in certain settings, panelists expressed some uncertainty regarding the appropriateness of systemic corticosteroid monotherapy or combination therapy, reflecting concerns about their safety profile and lack of direct comparative evidence with newer therapies.

Though panelists were aligned that initiating alternative pathway complement inhibitors was highly appropriate at any point in the vaccination series when patients were taking antibacterial prophylaxis, there was less agreement regarding alternative pathway complement inhibitor initiation in the absence of prophylaxis. For the first and second doses of the MenB vaccine, panelists agreed that initiating alternative pathway complement inhibitors without prophylaxis was inappropriate because patients were unlikely to have developed sufficient vaccine-mediated protection. However, after the third dose, panelists disagreed on the appropriateness of initiating alternative pathway complement inhibitors without prophylaxis. This disagreement largely reflected uncertainty regarding whether completion of the MenB vaccination series alone provides adequate protection against meningococcal infection in patients receiving complement inhibition.

Several limitations should be considered. This framework is based on expert consensus from a US-based panel

and does not reflect global variability in practice patterns or treatment access. In addition, many newer therapeutic classes, including B-cell-targeted and APRIL/BAFF pathway inhibitors such as sibeprenlimab, were not fully incorporated into the current evidence base at the time of panel development, and were therefore not included in this study [23, 24]. Moreover, at the time of the panel meeting, no therapeutic class had prospective evidence demonstrating efficacy based on histologic activity. Finally, as emphasized by KDIGO [20], the absence of a reliably defined “inflammatory glomerulus” underscores the inherent limitations of histology-based categorization, particularly given the contribution of profibrotic immune-complex mediated injury that may occur without overt inflammatory features.

Despite these limitations, there remains a clear need for structured clinical approaches to guide treatment selection for IgAN. Until biomarker-driven or prospectively validated phenotypes are established, clinicians must continue to make decisions using incomplete but clinically meaningful signals of disease activity. This consensus provides a pragmatic framework for organizing those signals in a way that is consistent with current practice and complementary to evolving mechanistic models of disease.

Future work should aim to test whether these clinically defined phenotypes are associated with meaningful kidney outcomes, develop quantitative measures that better capture immune complex-driven injury and fibrosis, and identify biomarkers that reflect the full spectrum of IgAN biology beyond inflammatory features alone.

Physicians should continue to apply their clinical expertise and refer to local protocols and guidelines to characterize and manage high-inflammatory and low-inflammatory activity IgAN. We hope this guidance can be a useful reference for healthcare professionals and serve as a foundation for additional research and discussion as new evidence becomes available.

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Data availability All data are included in the manuscript and/or supporting information.

Declarations

Competing interest Jai Radhakrishnan has received consulting fees from Novartis, Calliditas, Vera Therapeutics, Travers, Otsuka, and Alexion/Astra Zeneca. Sanjiv Anand has received payment or honoraria from CareDx, Natera, Calliditas, and Alexion. Mark Haas has received consulting fees from Novartis, AstraZeneca, Argenx, Otsuka, Biogen, Travers, and Vera Therapeutics; and has received research funding from Argenx. Leal Herlitz has received consulting fees from Novartis and Travers; has received payment or honoraria from Otsuka; and has received research funding from National Institutes of Health (NIH). Richard Lafayette has received consulting fees from Amgen, Calliditas, Biogen, Roche, Travers, BeiGene, Novartis, Vera, Alexion, GlaxoSmithKline, Omeros, and Alembic; and has received research funding from NIH and the University of Michigan. Tingting Li has received consulting fees from Novartis, Travers, Calliditas, Amgen, GlaxoSmithKline, and Vera Therapeutics; and has received research funding from Amgen, Vertex, Novartis, Omeros, Travers, Dimerix, and Calliditas. Arvind Madan has received consulting fees from Calliditas, Amgen, Novartis, and Akebia; and has received payment or honoraria from Novo Nordisk and AstraZeneca. Ali Mehdi has received payment or honoraria from Alexion; and has participated in a data safety monitoring board or advisory board for Vera Therapeutics, Calliditas, Novartis, and Alexion. Sayna Norouzi has received consulting fees from Calliditas, Alexion, Otsuka, and Novartis; and payment or honoraria from Boehringer Ingelheim and Sanofi. Brad Rovin has received consulting fees from Alexion, Novartis, Vera Therapeutics, Vertex, Calliditas, Otsuka, Travers, and Biogen; payment or honoraria from Medscape; and research funding from NIH and Lupus Research Association. Shikha Wadhvani has received consulting fees from Calliditas Therapeutics, Novartis Pharmaceuticals, Otsuka, Travers Therapeutics, Vera Therapeutics; payment or honoraria from Calliditas Therapeutics, Novartis Pharmaceuticals, Otsuka, Travers Therapeutics, Vera Therapeutics; support for meetings and/or travel from Boehringer Ingelheim, Calliditas Therapeutics, Novartis Pharmaceuticals, Otsuka, Travers Therapeutics, Vera Therapeutics. Cynthia Campos, Justin Weiss, Michael S Broder, and Irina Yermilov were employees of ADVI Health during the work described in this publication.

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Authors and Affiliations

Jai Radhakrishnan¹ · Sanjiv Anand² · Mark Haas³ · Leal Herlitz⁴ · Richard Lafayette⁵ · Tingting Li⁶ · Arvind Madan⁷ · Ali Mehdi⁸ · Sayna Norouzi⁹ · Brad Rovin¹⁰ · Shikha Wadhvani¹¹ · Cynthia Campos¹² · Justin Weiss¹² · Michael S. Broder¹² · Irina Yermilov¹²

✉ Justin Weiss
Justin.weiss@advi.com

¹ Division of Nephrology, Columbia University Vagelos College of Physicians and Surgeons, New York, NY, USA

² Intermountain Health, Murray, UT, USA

³ Department of Pathology and Laboratory Medicine, Cedars-Sinai Medical Center, Los Angeles, CA, USA

⁴ Department of Anatomic Pathology, Department of Kidney Medicine, Cleveland Clinic, Cleveland, OH, USA

⁵ Division of Nephrology, Stanford University Medical Center, Stanford, CA, USA

⁶ Division of Nephrology, Washington University in St. Louis, St. Louis, MO, USA

⁷ Central Florida Kidney Specialists, Orlando, FL, USA

⁸ Department of Kidney Medicine, Cleveland Clinic, Cleveland, OH, USA

⁹ Division of Nephrology, Loma Linda University, Loma Linda, CA, USA

¹⁰ Division of Nephrology, Department of Medicine, The Ohio State University, Columbus, OH, USA

¹¹ Division of Nephrology, University of Texas Medical Branch, Galveston, TX, USA

¹² ADVI Health, 1000 F Street NW, Suite 650, Washington, D.C 20004, USA