Novel treatment in IgA nephropathy

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Introduction

- IgA nephropathy (IgAN) is the most common cause of primary glomerulonephritis worldwide.
- The diagnostic hallmark of IgAN is the presence of mesangial depositions of IgA, either predominant or codominant with IgG and/or IgM, which are demonstrated by immunofluorescence microscopy .
- Despite this histopathologic hallmark, IgAN is a heterogenous disease not only in terms of epidemiology or clinical presentation but also in long-term renal disease progression and outcome.
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Clinical presentation

- Clinical presentation of patients with IgAN may vary from asymptomatic microscopic hematuria to macroscopic hematuria with or without proteinuria, acute kidney injury, nephrotic syndrome, or rapidly progressive glomerulonephritis .

- Approximately half of the patients with IgAN present with one or more recurrent episodes of macroscopic hematuria, often accompanying an upper respiratory or gastrointestinal infection

Pathogenesis of IgA nephropathy

- Mesangial deposition of IgA is the cornerstone in the pathogenesis of IgAN.
- IgA is predominantly polymeric IgA of the IgA1 subclass (polymeric IgA1).
- Dysregulated synthesis and metabolism of IgA (resulting in IgA immune complexes with characteristics that favor mesangial deposition) and the mesangial cell reaction to mesangial IgA accumulation are thought to be part of the pathogenetic puzzle.

Pathogenesis of IgA nephropathy

- The autoantigens are a specific set of IgA1 O-glycoforms displaying poor O-linked galactosylation of the IgA1 hinge region, which result in the generation of hinge glycan-specific IgA and immunoglobulin G (IgG) autoantibodies in susceptible individuals.
- As a result, some triggers such as mucosal infection or food antigens may drive the production and release of pathogenic IgA into the circulation.
- There it has the propensity to deposit within the mesangium and trigger glomerular injury .

Pathogenesis of IgA nephropathy

- Although IgAN's pathogenicity is not fully understood, mucosal biopsies from patients with IgA show significantly reduced numbers of polymeric IgA-secreting plasma cells when compared to healthy individuals.
- Another theory suggests that mesangial IgA is derived from systemically located plasma cells in bone marrow sites.
- Cytomegalovirus, Hemophilus parainfluenza, Staphylococcus aureus, Streptococcus, toxoplasmosis, and SARS-CoV-2 have been also implicated .
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Pathogenesis of IgA nephropathy

- Genetic factors influence the pathogenesis of IgAN as well.
- A genetic predisposition of this heterogenous disease has been suggested since its presence among certain families has been well described.
- It is presumed that it does not have classic Mendelian inheritance attributable to a single gene locus but serves as a complex polygenic heterogenous disease.
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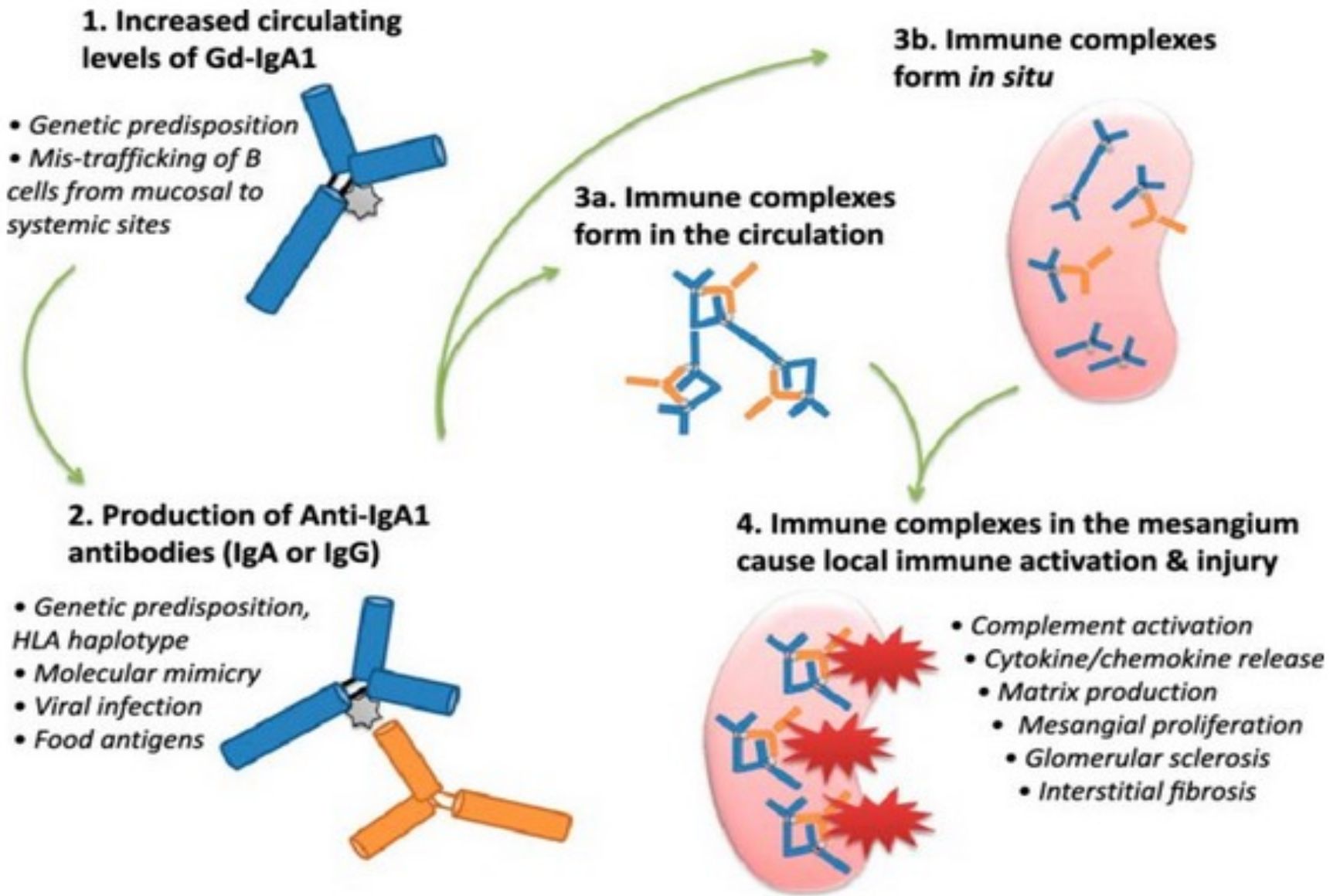
Pathogenesis of IgA nephropathy

- Mucosal tissue (especially in the gastrointestinal tract) constitutes a physical barrier against invaders.
- The host's immune system, microbiota, and pathogens are the three main players.
- With technological advances in genotyping, genetic studies, in particular hypothesis-free genome-wide association studies (GWASs), have documented significant associations of IgAN with several single-nucleotide polymorphisms within or near immune-related genes such as major histocompatibility complex (MHC) loci, thereby highlighting the immune component of this disease.
- The exact pathogenesis of IgAN remains unclear since genetic, environmental and autoimmune pathways interact with each other.

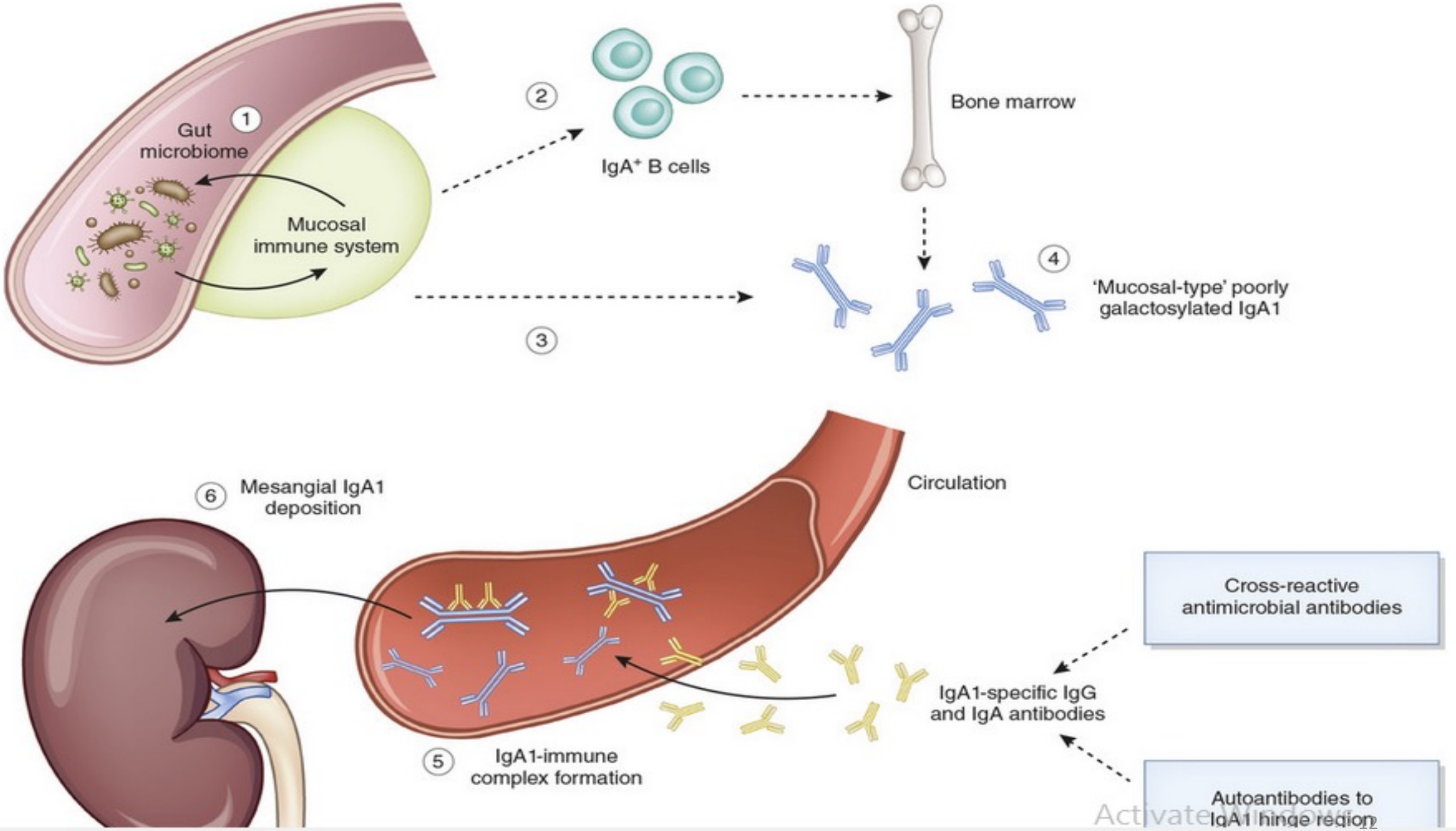
Pathogenesis of IgA nephropathy- (“fourth hit)

- “First hit” refers to the production of aberrant galactose-deficient IgA1 (Gd-IgA1) by plasma cells,
- leading to the synthesis of autoantibodies directed against the aberrant, Gd-IgA1 (“second hit”).
- “Third hit” refers to the formation of pathogenic immune complexes circulating in the bloodstream after the binding of autoantibodies to the Gd-IgA1.
- As a result, circulating immune complexes deposit at the points of filtration (mesangial cells located between the glomerular basement membrane and fenestrated endothelium of the kidney, leading to deposition of local immune activation, inflammation, and glomerular injury (“fourth hit”) .


1. Increased circulating levels of Gd-IgA1
• Genetic predisposition
• Mis-trafficking of B cells from mucosal to systemic sites
2. Production of Anti-IgA1 antibodies (IgA or IgG)
• Genetic predisposition, HLA haplotype
• Molecular mimicry
• Viral infection
• Food antigens
3a. Immune complexes form in the circulation
3b. Immune complexes form in situ
4. Immune complexes in the mesangium cause local immune activation & injury
• Complement activation
• Cytokine/chemokine release
• Matrix production
• Mesangial proliferation
• Glomerular sclerosis
• Interstitial fibrosis

Pathogenesis of IgA nephropathy….

- The pathogenesis of IgA nephropathy provides a theoretical basis for the locally targeted treatment of mucosal immune system disorders.
- Therefore, reducing Gd-IgA1 production by inhibiting mucosal B-lymphocyte activation and Peyer plaque proliferation can also become a new way to treat primary IgA nephropathy

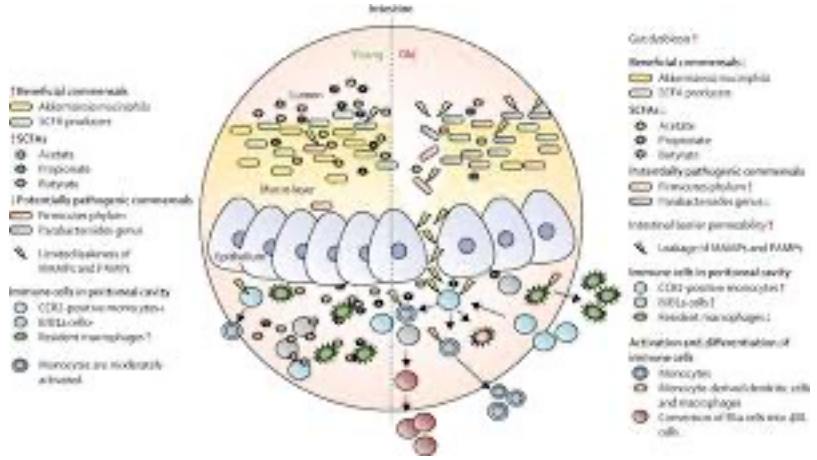


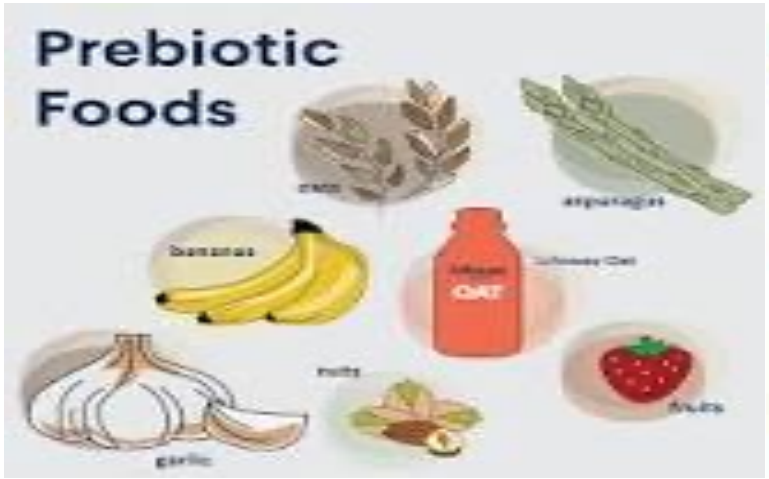
Roles of microbiota in the progression of the IgAN

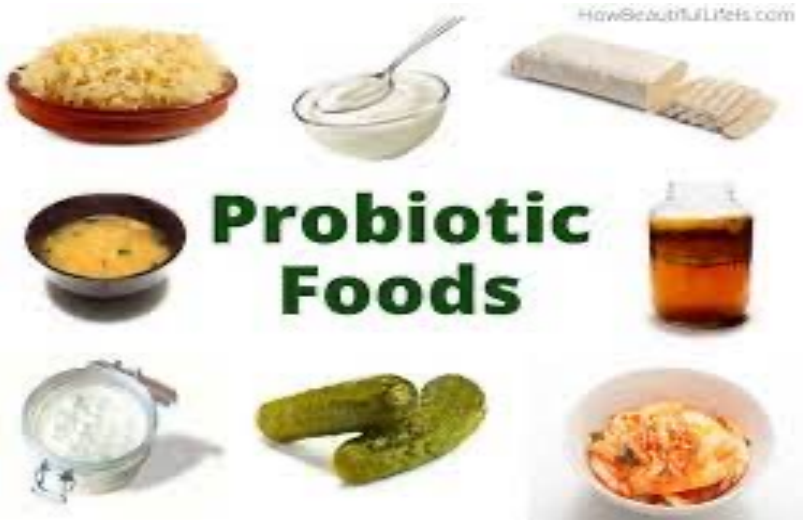
- The gut-kidney axis may play a key role in the development of IgAN.
- Human IgAN was suggested to be a maladaptive host response to the microbiota.
- Moreover, intestinal microbiota and its metabolites play a key role in IgA-immune responses. .
- Microbial infections can stimulate the differentiation of B-cells into IgA-secreting plasma cells.
- In the development of IgAN, an aberrant mucosal immune response to commensal microbiota, which is triggered by the hyperactivation of IgA-promoting cytokines.

Roles of microbiota in the progression of the IgAN

- Since microbiota dysbiosis was found to be associated with the incidence and progression of IgAN , antibiotics could be used to treat or prevent IgAN development or progression.
- Moreover, gut microbiota manipulation might be a new option for the therapy of IgAN, including dietary interventions, prebiotics, and probiotics, or through fecal microbiota transplantation.







Supportive Care in All Patients

- In the absence of variant forms of IgAN (e.g., IgAN with AKI or RPGN) and secondary causes (e.g., autoimmune disease, liver cirrhosis, inflammatory bowel disease, HIV, hepatitis, IgA vasculitis), optimized supportive care is initially suggested.
- Optimized supportive care focuses on blood pressure management, reduction of proteinuria, lifestyle modification, and total cardiovascular risk addressing .
- High-quality data support the benefit of blood pressure (BP) control and reduction of proteinuria to delay kidney disease progression in all chronic kidney disease (CKD) populations .
- Control of BP involves initially lifestyle modifications, such as salt restriction, dietary modification, weight reduction, smoking cessation, lipid lowering, and physical exercise as part of a holistic approach.

Supportive Care in All Patients—The Role of SGLT2 Inhibitors

- Sodium-Glucose Cotransporters (SGLTs) are proteins that occur primarily in the kidneys and play an important role in maintaining glucose balance in the blood.
- SGLT1 and SGLT2 are the two most known SGLTs of this family.
- Sodium-glucose Cotransporter 2 (SGLT2) is located in the proximal tubule of the nephron and causes dynamic reabsorption of 90% of filtered glucose together with sodium.

Supportive Care in All Patients—The Role of SGLT2 Inhibitors

- Their last function, combined with the reduction of blood pressure caused by natriuresis, results in their nephroprotective effect .
- SGLT2 inhibitors are also indicated in maximal supportive care of patients with IgAN and proteinuria.

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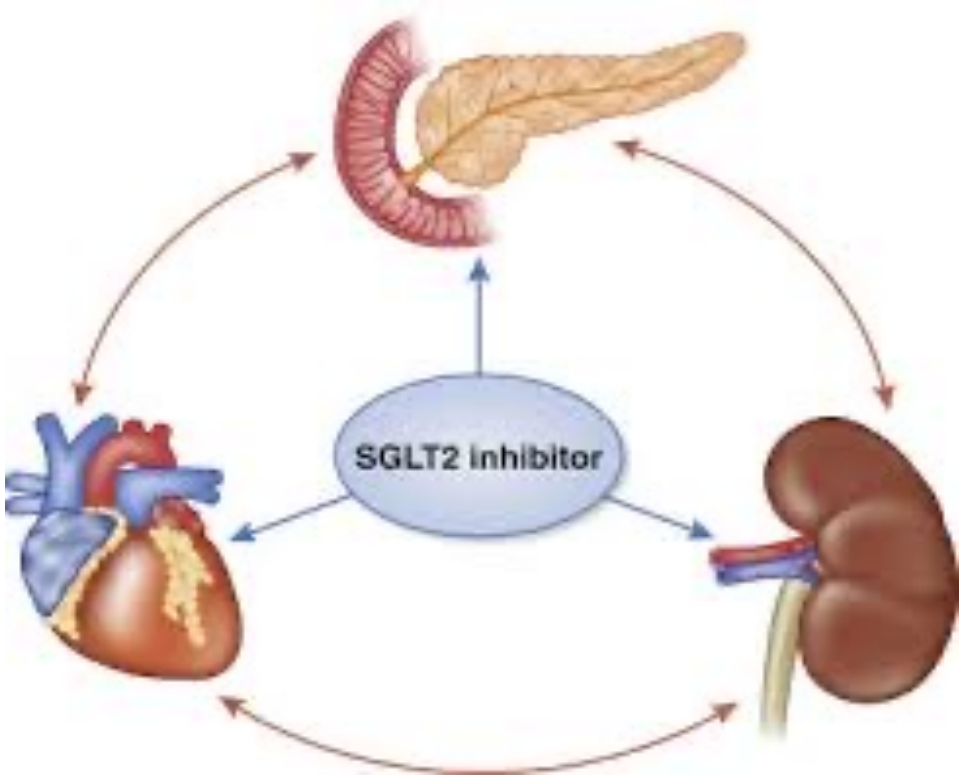


Immunosuppressive Therapy in High-Risk Patients

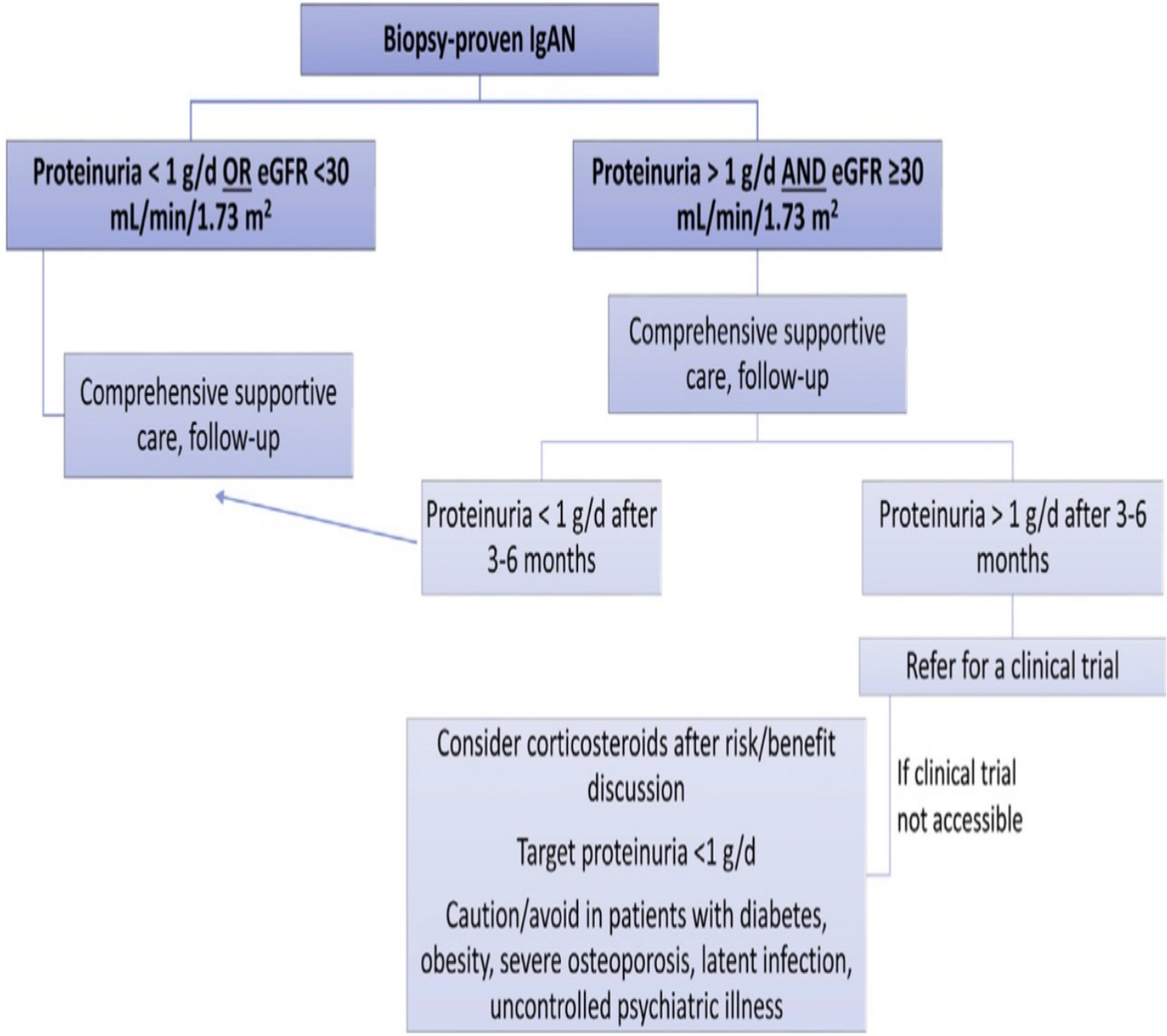
- Multiple studies demonstrate that proteinuria is the most powerful predictor of long-term renal outcome .
- Proteinuria reduction <1 g/day is a reasonable treatment target .
- Improvement of hematuria and stabilization of renal function are also goals of therapy.
- Patients with IgAN and persistent proteinuria ≥1 g/day, despite three to six months of optimized supportive care, are deemed at high risk of disease progression and may be considered eligible for immunosuppressive therapy.
- Immunosuppressive therapy is contraindicated in patients with evidence of severe and irreversible kidney damage (eGFR < 30 mL/min/1.73 m2 for more than 3 months, small echogenic kidneys, interstitial fibrosis, tubular atrophy, or severe glomerulosclerosis on kidney biopsy) since they usually tend to have an increased risk of treatment-emergent toxicity with no benefit.

Immunosuppressive Therapy in High-Risk Patients

- Several immunosuppressive treatment regimens have been studied but documented no evidence of efficacy in IgAN (cyclophosphamide, azathioprine, calcineurin inhibitors, rituximab) .
- There are few data to support the efficacy of mycophenolate mofetil (MMF) as a first-line treatment for IgAN in the past .

Immunosuppressive Treatment: Corticosteroids

- Corticosteroid is the only currently available immunosuppressive agent with evidence supporting its efficacy.
- The 2012 KDIGO clinical practice guideline on GN suggests a 6-month course of steroids therapy in patients who have over 1g/d of proteinuria and eGFR > 50 mL/min/1.73 m2.
- The best available evidence therefore points toward the potential benefits of corticosteroids in patients with over 2g/d of proteinuria.
- The potential morbidity and mortality must be acknowledged and carefully reviewed with patients before proceeding with this therapy.
- Patients at particularly high risk of toxicity include those with pre-existing obesity, diabetes, and latent infections.
- Combination of cyclophosphamide and steroids is recommended in IgAN only for patients presenting with RPGN.


Biopsy-proven IgAN
Proteinuria < 1 g/d OR eGFR <30 mL/min/1.73 m²
Proteinuria > 1 g/d AND eGFR ≥30 mL/min/1.73 m²
Comprehensive supportive care, follow-up
Comprehensive supportive care, follow-up
Proteinuria < 1 g/d after 3-6 months
Proteinuria > 1 g/d after 3-6 months
Refer for a clinical trial
If clinical trial not accessible
Consider corticosteroids after risk/benefit discussion
Target proteinuria <1 g/d
Caution/avoid in patients with diabetes, obesity, severe osteoporosis, latent infection, uncontrolled psychiatric illness

Immunosuppression: Beyond Corticosteroids

- The combination of mycophenolate mofetil (MMF) at 1.5 g/d with low-dose prednisone for a total course of 6 months was not inferior to full-dose prednisone in inducing proteinuric remission at month 6 and 12 with fewer adverse events such as diabetes
- Hydroxychloroquine is proposed to have immunomodulatory effects. Recently, a small randomized trial demonstrated a greater reduction in proteinuria compared with placebo at 6 months in Chinese patients who have persistent proteinuria of 0.75-3.5 g/d despite RAS blockade
- Tonsillectomy has been associated with a reduction in proteinuria and improved kidney survival in Japanese populations.
- In one randomized controlled trial conducted in Japan, tonsillectomy combined with pulse steroids therapy did not have benefits over steroids alone

Targeted-Release Budesonide

- A targeted-release formula of budesonide (Nefecon) was developed for use in IgAN.
- The rationale for design of this compound was to release drug at the distal ileum where the largest site of Gd-IgA1 secreting cells is located: namely, the mucosal-associated lymphoid tissue.
- This is hypothesized to result in a reduction in production of Gd- IgA1 by cells originating from these sites or by mucosal- derived cells that migrate from the intestinal lymphoid tissue to other sites such as the bone marrow.
- Any absorbed drug is hypothesized to primarily undergo first- pass metabolism, thereby limiting systemic steroid exposure.

Complement Inhibitors

- Currently, there are several ongoing clinical trials of complement inhibitors being tested with various eGFR inclusion criteria:
- eGFR > 30 mL/min/1.73 m2 for OMS721 (Narsoplimab, a monoclonal antibody against MASP-2), LNP023 (a complement factor B inhibitor), Cemdisiran (small-interfering RNAs directed against C5), and APL-2 (a C3 inhibitor);
- eGFR > 40 mL/min/1.73 m2 for IONIS-FB-LRx (an antisense inhibitor of complement factor B);
- and eGFR > 60 mL/min/1.73 m2 for CCX168 (Avacopan; a C5a receptor antagonist).

Other Investigational Agents
Inhibition of Immune Complex-Activated Complement Activity

- Topical complement activation has a role in the pathogenesis of IgAN, as pathogenic immune complexes of galactose deficient IgA1 can activate both the alternative and lectin pathways, leading to the generation of the membrane attack complex C5b-9, inducing mesangial cell apoptosis and glomerular inflammation via IL-6 and TGF-β1 production .
-
- **Glomerular C3 deposition in IgAN predicts more severe clinical features, worsen histopathological characteristics, and long-term poor renal survival .**

- Factors under investigation that target the inhibition of C5a activation are avacopan, an anti-C5a receptor antagonist, which showed an improvement in the slope of the UPCR in a short-term pilot study (NCT02384317) , ravulizumab, an eculizumab-derived long-acting C5-blocking antibody (SANCTUARY study, NCT04564339), and cemdisiran, a small interfering RNA-targeting C5 (NCT03841448).

Other Investigational Agents
Inhibition of Immune Complex-Activated Complement Activity

- In addition, agents that inhibit the complement activation pathway are being tested in phase II trials such as APL-2 (NCT03453619) and iptacopan (NCT03373461).
- Iptacopan was well tolerated and led to a continuous reduction in proteinuria at 6 months and will be further evaluated in the ongoing Phase III APPLAUSE-IgAN trial (NCT04578834) .
- IONIS-FB-LRx is an antisense inhibitor of complement factor B messenger ribonucleic acid (CFB mRNA), which is under phase II clinical trial and the result is pending (NCT04014335).
- Finally, Narsoplimab, which is a human monoclonal antibody against mannan-associated lectin-binding serine protease-2 (MASP-2), inhibits lectin complement pathway activation.
- Interim analysis from a phase II clinical trial suggests that narsoplimab treatment reduced proteinuria and preserved kidney function .

Inhibition of BAFF/APRIL Signaling

- BAFF (B-cell activating factor) and APRIL (a proliferation-inducing ligand) are tumor necrosis factor family ligands involved in B cell and plasma cell function and survival and the pathogenesis of several autoimmune diseases, including IgAN .
- In patients with IgAN, there is increased expression of APRIL, which is associated with increased expression of Gd-IgA1 antibodies .
- Thus, targeting APRIL and BAFF may reduce Gd-IgA1 antibody levels.
- The phase II/III BRIGHT-SC study (NCT02062684), which studied blisibimod, a monoclonal antibody against both soluble and membrane BAFF, showed a reduction of proteinuria compared to placebo.
- Anti-APRIL antibodies sibeprenlimab (NCT05248646) and BION-1301 (NCT03945318) are in phase III and II clinical trials, respectively, to test efficacy and safety in patients with IgAN .
- Atacicept, a soluble TACI-Immunoglobin fusion protein, which targets both BAFF and APRIL, showed a reduction in Gd-IgA1 antibody levels and proteinuria when evaluated in the randomized phase II JANUS study (NCT02808429) in 16 patients with IgAN .
- Finally, telitacicept, another BAFF/APRIL inhibitor, showed proteinuria reduction in 44 patients with IgAN in a phase II clinical trial (NCT04905212)

Plasma Cell and B Cell Depletion

- Rituximab, a chimeric monoclonal antibody targeted against CD20, shows lack of efficacy in a randomized, controlled clinical trial .
- The promising felzartamab, a fully human IgG1 monoclonal antibody designed to deplete CD38+ plasma cells, is in a phase II clinical trial for patients with IgAN and an increased risk of disease progression (NCT05065970).
- Finally, bortezomib, a proteasome inhibitor that depletes plasma cells and is approved for the treatment of multiple myeloma, showed complete remission of proteinuria in 4 out of 8 patients in the first year of follow-up in a pilot trial (NCT01103778) .

Inhibition of Endothelin A Receptor and Angiotensin II Subtype 1 Receptor

- Endothelin-1 (ET-1), largely through activation of endothelin A receptors, and angiotensin II have a role in kidney function decline by contributing to inflammation and fibrosis in the kidney, changes to the shape of podocytes, podocyte loss, mesangial cell proliferation and increased permeability of the glomerular filtration barrier.
- Both are also causing vasoconstriction, leading to increase glomerular pressure .
- Sparsentan is a dual-acting antagonist of both endothelin type A (ETA) and angiotensin II subtype 1 (AT1) receptors.
- Atrasentan is a potent and selective endothelin A receptor antagonist with the potential to provide benefits in IgA nephropathy and other proteinuric glomerular diseases by reducing proteinuria.
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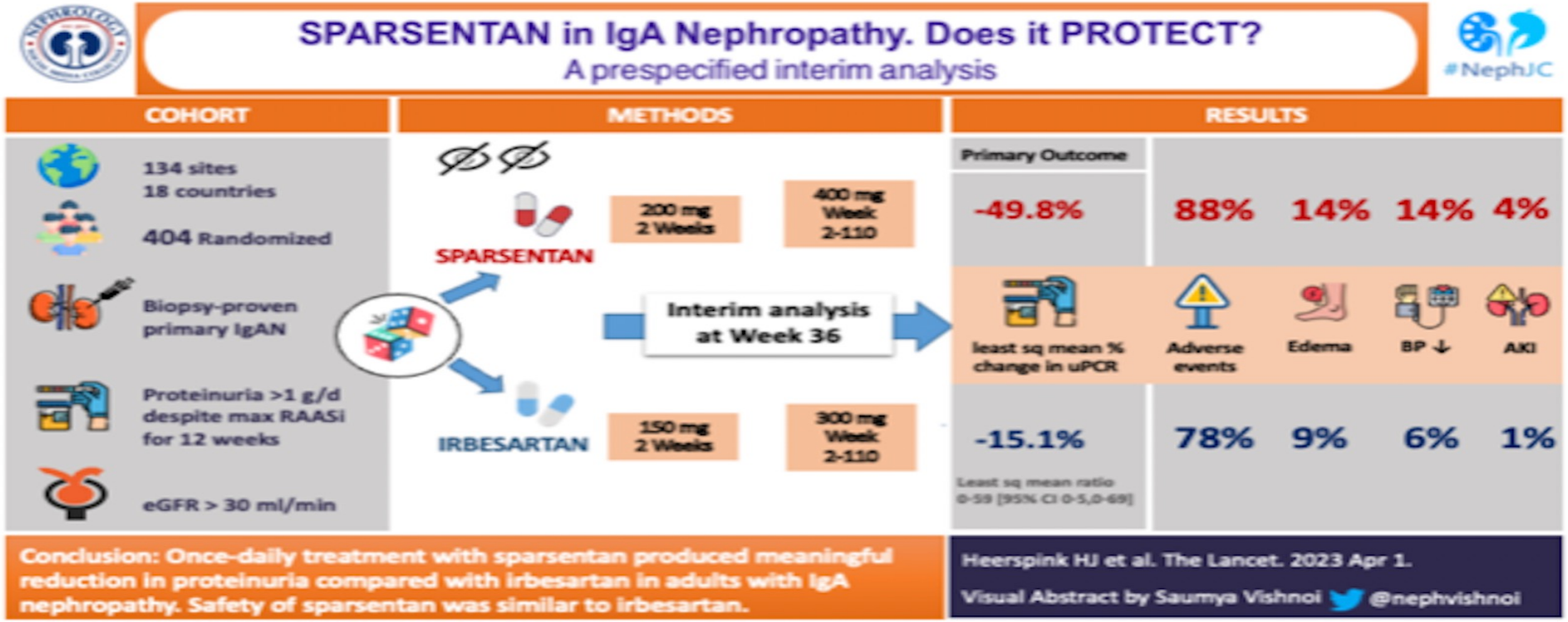
Is Sparsentan effective and safe for patients with IgAN on maximal Raas blockade? Check out the VA by NSMC intern Saumya Vishnoi



Findings

Between Dec 20, 2018, and May 26, 2021, 404 participants were randomly assigned to sparsentan (n=202) or irbesartan (n=202) and received treatment. At week 36, the geometric least squares mean percent change from baseline in urine protein–creatinine ratio was statistically significantly greater in the sparsentan group (–49·8%) than the irbesartan group (–15·1%), resulting in a between-group relative reduction of 41% (least squares mean ratio=0·59; 95% CI 0·51–0·69; p<0·0001). TEAEs with sparsentan were similar to irbesartan. There were no cases of severe oedema, heart failure, hepatotoxicity, or oedema-related discontinuations. Bodyweight changes from baseline were not different between the sparsentan and irbesartan groups.

Interpretation

Once-daily treatment with sparsentan produced meaningful reduction in proteinuria compared with irbesartan in adults with IgA nephropathy. Safety of sparsentan was similar to irbesartan. Future analyses after completion of the 2-year double-blind period will show whether these beneficial effects translate into a long-term nephroprotective potential of sparsentan.

Funding

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  Further information is available at www.filsparirems.com or 1-833-513-1325.
- **Hypotension:** Hypotension has been observed in patients treated with ARBs and ERAs. There was a

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بودزوناید (سیستمیک خوراکی)

پاییز 1402

Indications:

- -بیماری کرون فعال درگیر کننده ایلئوم یا کولون بالارونده (خفیف تا متوسط)
- -نفروپاتی ایمونوگلوبولین A اولیه به جهت کاهش پروتئینوری در بیماران با ریسک بالای پیشرفت بیماری
- -کولیت اولسراتیو
- **- انتروپاتی از دست دهنده پروتئین به دنبال جراحی فونتان**

Contraindication

- حساسیت به بودزوناید یا هر جزئی از فرمولاسیون دارو
- در صورت حساسیت به دیگر کورتیکواستروئید ها، به دلیل شباهت ساختار شیمیایی و عملکرد فارماکولوژیک، ریسک حساسیت متقاطع وجود دارد
- سل فعال
- هرگونه عفونت کنترل نشده موضعی یا سیستمیک باکتریایی، ویروسی و قارچی.
- واکنش ازدیاد حساسیت به سویا، لسیتین و بادام زمینی

Dosing

- **نفروپاتی ایمونوگلوبولین A اولیه (درمان جایگزین):**
- در بیماران با ریسک بالای پیشرفت سریع بیماری (مانند نسبت پروتئین به کراتینین ادراری ≥ 5/1 گرم در گرم) استفاده می شود.
- کپسول با رهش تاخیری، 16 میلی گرم روزانه صبح ها به مدت 9 ماه، سپس کاهش دوز به میزان 8 میلی گرم روزانه به مدت 2 هفته و قطع

conclusion

- IgAN is the most common primary glomerulonephritis worldwide and had different heterogeneity in terms of clinical presentation and risk of progression.
- The current therapeutic options in patients who are at increased risk for ESKD are mainly glucocorticoids, which, however, do not appear to be of long-term benefit and are associated with the appearance of several adverse events.
- In terms of supportive care, SGLT2 inhibitors show that they can reduce proteinuria, with more studies needed to understand their effect on long-term outcomes.
- A FIND-CKD trial is also underway to evaluate the effect of finerenone, a novel non-steroidal mineralocorticoid receptor antagonist (MRA) in the non-diabetic CKD patient population (NCT05047263).
- The combination treatment with SGLT2i may have further applications in patients with IgAN.
- Dual-acting inhibitors of ETA and AT1 receptor are also another future option in terms of supportive care.

IgA Nephropathy: Core Curriculum 2021

Prapa Pattrapornpisut, Carmen Avila-Casado, and Heather N. Reich

Immunoglobulin A nephropathy (IgAN) is the most common primary glomerular disease worldwide. The diagnostic histologic hallmark is dominant or codominant IgA staining on kidney biopsy; however, patients may present with various clinical syndromes ranging from asymptomatic abnormalities noted on urinalysis to rapidly progressive glomerulonephritis. Given substantial heterogeneity in the clinical course of disease, online risk calculators are available that may assist in prognostication and inform discussions with patients. Comprehensive supportive treatment is central in the initial therapy of IgAN; the additive benefit of currently available immunosuppressive agents remains an area of controversy. Although proteinuria is attenuated by the use of corticosteroids, the long-term benefits have been questioned, and the use of corticosteroids is associated with severe adverse effects, notably infection. Recent advances in our understanding of mucosal immunity and the role of the complement system in IgAN pathogenesis are leading to development of novel therapeutic options, which are being evaluated in ongoing clinical trials. In this installment of the *AJKD* Core Curriculum in Nephrology, IgAN pathogenesis, clinical manifestations, histology, prediction tools, and treatment are reviewed, and case examples are presented to illustrate the approach to the management of patients with IgAN.

Complete author and article information provided at end of article.

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Introduction

Immunoglobulin A nephropathy (IgAN) is the most prevalent primary glomerulonephritis (GN) worldwide, with an overall incidence of at least 2.5 per 100,000. Estimation of the true incidence is challenging primarily due to variations in biopsy practice patterns, and it is likely that IgAN is more common than appreciated. Notwithstanding differences in

worldwide: a systematic review of the literature. *Nephrol Dial Transplant*. 2011;26(2):414-430. ★ESSENTIAL READING

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The Core Curriculum

IgA Nephropathy: Current Treatment and New Insights

Dimitra Petrou [1]

, Petros Kalogeropoulos [1,*] , George Liapis [2] and Sophia Lionaki [1,*]

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Abstract: IgA Nephropathy (IgAN) is the most common cause of primary glomerulonephritis world- wide. Despite the histopathologic hallmark of mesangial IgA deposition, IgAN is a heterogenous autoimmune disease not only in terms of

Case Reports > Saudi J Kidney Dis Transpl. 2020 Mar-Apr;31(2):521-523.

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Successful treatment of a patient with posttransplant IgA nephropathy with targeted release formulation of budesonide

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Abstract

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Current knowledge of targeted-release budesonide in immunoglobulin A nephropathy: A comprehensive review

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Immunoglobulin A (IgA) nephropathy is a common autoimmune kidney disease. Accumulating studies showed that IgA nephropathy may be partially


Thank you