Poster Presentation：Glomerular Diseases
Poster No.：C0434
Abstract Submission No.：APCN20250003

Bridging Clinical Practice Gaps: Insights from Chinese Physicians on the Diagnosis and Management of Immunoglobulin A Nephropathy

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Abstract

Background and Aims

Immunoglobulin A nephropathy (IgAN) is the most common primary glomerular disease globally[1]. Its incidence is notably higher in Asia than in North America and Europe[2].

The goal of this activity was for Chinese learners to better understand the burden of IgAN, the immunopathogenesis of IgAN and recently approved therapies.

Methods

A 26-question, online, CPD-accredited survey was developed, encompassing knowledge, case-based (competence), and confidence questions. Evidence-based feedback and peer responses highlighted correct responses. Activity launched 17 May 2024, data collected 6 September 2024.

Results

151 Chinese physicians completed all questions in the timeframe

- 38% saw 1 to 10 patients with IgAN per month, and 33% saw 11 to 20 per month
- Only 26% correctly recognized that the RaDar study showed that IgAN can be progressive even in patients considered to be at low risk (proteinuria < 1 g/d)
- 48% correctly chose asymptomatic microscopic hematuria and proteinuria as the most common clinical presentation of IgAN
- 36% correctly identified the presence of tubular atrophy (T) in the MEST-C score as the most significant predictor of worse outcomes
- 60% could not identify the correct order of the multi-hit hypothesis
- Only 15% correctly identified the production of Gd-IgA1 as a potential effect of APRIL and BAFF in the pathogenesis of IgAN
- Only 28% knew that the 2021 KDIGO guidelines recommend considering clinical trial enrollment as the best next step for managing high-risk IgAN patients who are not improving with supportive care
- 72% failed to recognize that reduced proteinuria is the surrogate endpoint currently used in clinical trials to assess treatment efficacy
- Only 18% correctly identified that in adults with biopsy-proven IgAN with high risk for progression in the PROTECT trial, sparsentan showed a 40% reduction in UACR and a significantly slower decline in eGFR at 2 years vs irbesartan
- 56% correctly identified that sibeprenlimab in the phase 2 ENVISION trial significantly reduced UPCR vs placebo at 12 months in patients with progressive IgAN
- After completing the activity, <20% reported feeling mostly/very confident in their understanding of the burden of IgAN and of emerging treatments

Conclusions
This activity revealed significant gaps in Chinese physician's knowledge and confidence regarding the burden, diagnosis, and management of IgAN, highlighting the need for targeted educational initiatives to improve clinical decision-making.

Reference
1. Zhang Z, et al. IgA Nephropathy: A Chinese Perspective. Glomerular Dis. 2021;2(1):30-41
2. Zaidi O, et al. A targeted literature review of prevalence and treatment patterns of IgA nephropathy in mainland China, Taiwan, and South Korea. Kidney Int Rep. 2022;7(2)

Keywords：IgAN, education

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Rare Coexistence of Alport Syndrome and C3 Glomerulopathy in a Young Male: Insights from Longitudinal Clinical, Genetic, and Histopathological Data

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Abstract

Alport syndrome, caused by COL4A3, COL4A4, or COL4A5 mutations, and C3 glomerulopathy (C3G), driven by complement dysregulation, rarely coexist, posing diagnostic and therapeutic challenges. We report a 24-year-old male with both conditions, presenting longitudinal clinical, genetic, and histopathological findings.

The patient, a student from Uttarakhand, reported a 15-year history of migraine-like headaches (unilateral, with nausea, photophobia, phonophobia) and bilateral sensorineural hearing loss since age 9, noticed by parents. For 6 months, he experienced frothy urine and generalized weakness. Laboratory data over 12 months (03/24–03/25) showed progressive renal dysfunction: serum creatinine rose from 2.39 to 4.55 mg/dL, estimated glomerular filtration rate (eGFR) declined from 29.5 to 17 mL/min/1.73 m² (Stage 4 CKD), and 24-hour urine protein decreased from 4.41 to 2.56 g/day, with urine albumin-creatinine ratio (ACR) consistently in the A3 category (e.g., 4165.8 mg/g, 10/24). Complement levels (C3 122 mg/dL, C4 21 mg/dL) were normal, and ANA was negative. Ocular examination was normal.

A renal biopsy (25/04/24) revealed focal segmental glomerulosclerosis (FSGS), dominant C3 deposits suggestive of C3G, and a chronicity score of 6 (moderate chronic changes). Electron microscopy from prior reports noted minimal GBM contouring and mesangial electron-dense deposits, with preserved trilaminar architecture, atypical for Alport syndrome. Clinical exome sequencing identified compound heterozygous COL4A3 mutations (c.1411G>T, p.Glu471*; c.1967dup, p.Pro658Thrfs*34), confirming autosomal recessive Alport syndrome 3B. Parental carrier screening verified the inheritance pattern, with the father and mother carrying mutations in exons 27 and 23, respectively.

Management included losartan for renoprotection, mycophenolate mofetil, and prednisolone for immunosuppression, with supportive care (calcitriol, sodium bicarbonate, erythropoietin). Eculizumab was considered but not initiated for cost reasons. Despite treatment, renal function deteriorated, highlighting the challenge of managing dual pathologies.

This case illustrates the complexity of overlapping glomerular diseases. The preserved GBM architecture despite COL4A3 mutations suggests an atypical Alport presentation, while normal complement levels complicate the C3G diagnosis, possibly indicating a variant form. Structural GBM defects may trigger complement activation, though the exact mechanism remains unclear. This rare coexistence underscores the need for integrated genetic and immunological evaluations in atypical glomerulopathies. Further research is essential to elucidate the interplay between these conditions and explore targeted therapies, such as complement inhibitors, in such cases.

Keywords：Alport syndrome, C3 glomerulopathy, COL4A3, nephrotic syndrome, complement dysregulation

Poster Presentation：Glomerular Diseases
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Beyond Nine Months: Real-World Efficacy and Safety of Extended Nefecon Therapy in IgA Nephropathy

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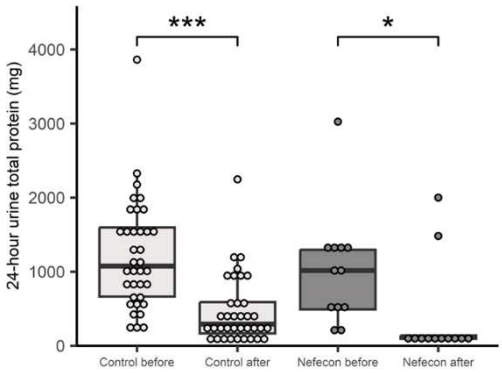
Introduction: IgA nephropathy (IgAN) is the most common primary glomerulonephritis in Asia. Epidemiological data show that approximately 60% of Chinese IgAN patients progress to end-stage kidney disease (ESKD) within 15 years. Nefecon, a targeted-release formulation of budesonide, reduces galactose-deficient IgA1 by acting on Peyer's patches in the distal ileum. Although the NefIgArd trial demonstrated its efficacy over 9 months, evidence for longer treatment duration remains limited.

Methods: We conducted a retrospective, propensity score-matched study at XXX Hospital. Twelve patients with primary IgAN received Nefecon 16 mg/day for 12 months. Thirty-six matched controls received conventional supportive therapy, mostly combined with corticosteroids or immunosuppressants. Matching was based on age, sex, serum creatinine, eGFR, and 24-hour urine protein. Clinical and laboratory data were collected at baseline and after 12 months. Primary outcomes included changes in 24-hour urine total protein and eGFR slope.

Results: In the Nefecon group, median 24-hour urine protein significantly decreased from 1016 [490, 1296] mg to 114 [96, 139] mg ($p = 0.037$), while in the control group, it decreased from 1074 [663, 1597] mg to 291 [167, 589] mg ($p < 0.001$). Post-treatment, proteinuria was significantly lower in the Nefecon group than in controls ($p = 0.01$). The eGFR slope was higher in the Nefecon group (5.4 [1.6, 11.6] vs. –3.4 [–12.9, 8.2] mL/min/1.73 m²/year, $p = 0.032$). No severe infections occurred in the Nefecon group. Mild adverse events included changes in bowel habits, sleep disturbances, and menstrual irregularities.

Conclusion: Twelve-month Nefecon treatment significantly reduced proteinuria and preserved renal function in patients with IgAN, with better safety and tolerability compared to conventional therapy.

Keywords： IgA nephropathy, Nefecon, Extended therapy, Real-world study, Proteinuria, Renal function, Safety



Poster Presentation：Glomerular Diseases
Poster No.：C0437
Abstract Submission No.：APCN20250060

A Study of Clinical Presentation and Correlative Histopathological Patterns in Membranous Nephropathy

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Abstract

Background

The aim of this study was to investigate the correlation between kidney dysfunction and pathological features in the kidney tissues of patients with primary membranous nephropathy (MN) diagnosed by kidney biopsy.

Method

A retrospective study was conducted on cases of MN diagnosed in kidney biopsies performed at the First Central Hospital of Mongolia (FCHM) over a period of 12 years. The clinical presentations of patients were assessed by chief complaints and laboratory data at the time of performing renal biopsy. The pathologic findings in light microscopy (LM) and immunofluorescence microscopy (IF) in patients diagnosed with MN will be summarized in the kidney biopsy analysis. Statistical analysis was performed using SPSS and STATA 15.0 software.

Results

A total of 305 kidney biopsies performed at the FCHM between 2011 and 2023 resulted in the diagnosis of 51 cases of primary MN. The mean age of patients with membranous nephropathy was 40.6±9.3 years, with the oldest age of 65 and the youngest of 22 years, and 36 (70.59%) were male and 15 (29.41%) were female. In the kidney biopsy, 33.3% showed global sclerosis, 94.12% showed thickening of the glomerular basement membrane (GBM), 31.2% showed double counter, 88.24% showed holes in the GBM, and 54.9% showed spike-like changes by LM. IF showed IgG 3+ in 37.3%, 2+ in 39.2%, 1+ in 13.7%, and trace staining in 9.8%, while 74.5% of the cases were positive for C3, 93.1% for kappa, and 79.5% for lambda. LM showed thickening of the GBM (OR 23.5, 95% CI 0.093-0.53, p value= 0.007) and interstitial fibrosis (95% CI 6.98-31.07, p value= 0.003) contributing to the decrease in eGFR. Patients who underwent biopsy later (months) after the diagnosis of chronic glomerulonephritis had a higher incidence of interstitial fibrosis (74.6±98.43, p value=0.002). Overall, globally, sclerosis was associated with increased systolic blood pressure (p value 0.01, 95% CI [0.092, 0.601]) and diastolic blood pressure (p value 0.0084, 95% CI [0.104, 0.623] OR 6.25).

Conclusions

This study shows that the pathological changes in membranous nephropathy confirmed by kidney biopsy, including interstitial fibrosis, contribute to the decrease in glomerular filtration rate.

Keywords：Membranous nephropathy, light microscopy, immunofluorescence, kidney biopsy

Poster Presentation：Glomerular Diseases
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Study of Rituximab/Mycophenolate Combination Therapy in Paediatric Patients with Calcineurin Inhibitor-Resistant Focal Segmental Glomerulosclerosis

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Abstract

Introduction: Calcineurin Inhibitors like tacrolimus or cyclosporine have proven efficacy in children with focal segmental glomerulosclerosis (FSGS) with a steroid dependent (SD) or steroid resistant (SR) course. However, there is a paucity of data and therapeutic options for calcineurin inhibitor (CNI) resistant forms of FSGS in children. The objective of the study was to evaluate a novel therapeutic option in CNI resistant FSGS by using the dual therapy of rituximab and mycophenolate to maintain remission.

Methods: This was a prospective study that included children in a state of non-remission despite 6 months of continuous therapy with CNIs or two or more relapses while on CNIs or while maintaining adequate drug trough levels or those with CNI toxicities which mandated discontinuation of CNI. All patients were followed up every 3 months to evaluate remission status and monitor the adverse effects of drugs. The clinical, therapeutic profile, and treatment outcomes like sustained remission versus no remission, in all subjects were who received dual rituximab + mycophenolate as maintenance therapy for a minimum of 1 year were recorded and analyse.

Results: The age of all thirteen patients were between 2.4 to 17.6 years with median age of presentation was 7.8 years with M:F ratio of 2.2. Ten (76.9%) of them had an SD course and three (23.1%) had an SR course. Four (30.7%) had evidence of acute/chronic CNI toxicity, and the remaining nine (69.3%) showed no response to CNI therapy despite adequate trough levels. Post dual therapy, 11 (84.6%) had sustained remission for at 1 year and two (15.4%) children did not show remission. None reported adverse reactions or infections, and all had preserved renal functions.

Conclusion: Combination therapy with rituximab and mycophenolate among children with CNI resistant FSGS can emerge as a promising and efficacious treatment strategy to ensure sustained remission in this subset of patients.

Keywords：Resistant focal segmental glomerulosclerosis; Rituximab + Mycophenolate Combination Therapy; Paediatric patients

Table: Outcome of dual therapy with rituximab and mycophenolate in children with CNI-resistant Focal Segmental GlomeruloSclerosis

Case no	Total doses of RTX	Response	Total duration of follow-up post RTX/MMF	No of relapses	Adverse effects/ infections during therapy
1.	2	CR	2 years 6 months	None	None
2.	4	CR	4 years	1 R after 2 years	None
3.	2	CR	1 year 9 months	None	None
4.	2	CR	2 years	None	None
5.	2	CR	1 year 9 months	None	None
6.	2	CR	1 year 6 months	None	None
7.	2	CR	1 year 6 months	None	Mild LRTI
8.	2	CR	1 year 3 months	None	Mild LRTI
9.	2	CR	1 year 8 months	None	None
10.	2	No response	1 year	-	None
11.	4	CR	1 year	1 R after 1 year	None
12.	2	CR	1 year	None	None
13.	2	No response	1 year	-	None

RTX- Rituximab, MMF- mycophenolate, CR- complete remission, R- relapse

Poster Presentation：Glomerular Diseases
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Abstract Submission No.：APCN20250087

Complement Inhibition with Iptacopan in Refractory MPGN: Taiwan's First Case Using Iptacopan with Clinical Benefit

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Abstract

Background

Membranoproliferative glomerulonephritis (MPGN) is a rare glomerular disease characterized by hypercellularity, mesangial matrix expansion and duplication of the glomerular basement membrane. There is no specific therapy for MPGN and prognosis is unfavourable. Conventional treatment usually comprise inhibition of renin-angiotensin system and immunosuppressants, including corticosteroids, cyclophosphamide, cyclosporine, and mycophenolate mofetil, are often ineffective. Emerging targeted therapies such as complement factor B inhibitors (e.g., iptacopan) offer a potential new approach for difficult cases. We presented a conventional treatment failure MPGN case, experienced improving of proteinuria and stabilized estimated glomerular filtration rate (eGFR) slope after iptacopan treatment.

Methods

We reviewed the clinical course of a 67-year-old cirrhotic man with histology-proven MPGN that developed following a ruptured colonic diverticulitis in November 2020. Longitudinal data for kidney function (eGFR) and proteinuria (urine protein-to-creatinine ratio, UPCR) from 2020 to 2025 were collected from medical records. The patient received inhibition of renin-angiotensin system and multi-agent immunosuppressive therapy during 2021–2022, including high-dose prednisolone, cyclophosphamide, cyclosporine, and mycophenolic acid. Due to continued disease progression, iptacopan (200 mg twice daily) was initiated in June 2024 and continued through mid-2025. We correlated the timing of treatments with changes in eGFR and UPCR.

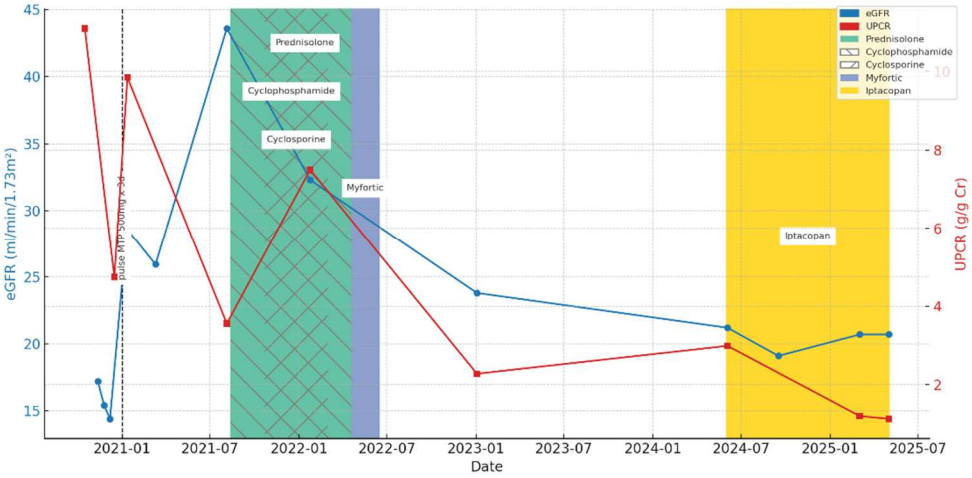
Results

Despite intensive immunosuppression in 2021–2022, the patient's proteinuria remained in the nephrotic range (UPCR ~3–4 g/g) and his eGFR showed a steady decline. By late 2024, eGFR had fallen to around 20 mL/min/1.73 m² with persistent heavy proteinuria. After starting iptacopan, a marked improvement in renal parameters was observed. Over the ensuing 12 months, UPCR decreased to 0.82 g/g and the decline in eGFR halted, stabilizing at roughly 22.4 mL/min/1.73 m² in June 2025. The temporal association suggests that previous immunosuppressive regimens had minimal impact on the disease trajectory, whereas complement inhibition coincided with significant reduction in proteinuria and stabilization of kidney function.

Conclusion

In this refractory MPGN case, targeted complement pathway inhibition with iptacopan dramatically reduced proteinuria and stabilized renal function after conventional immunosuppressants failed to halt disease progression. This case highlights the potential of complement inhibition therapy as an effective strategy in managing MPGN.

Keywords：MPGN; complement inhibition; iptacopan


eGFR
UPCR
Prednisolone
Cyclophosphamide
Cyclosporine
Myfortic
Iptacopan
Prednisolone
Cyclophosphamide
Cyclosporine
Myfortic
Iptacopan
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eGFR (ml/min/1.73m²)
UPCR (g/g Cr)
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Poster Presentation：Glomerular Diseases
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Determining the Association of Glomerular C3 Staining in the Histopathology Results and Renal Outcomes amongst Filipinos with IgA Nephropathy

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Abstract

Introduction: IgA nephropathy (IgAN) is a leading cause of chronic kidney disease (CKD), particularly in Southeast Asia, where it presents predominantly among younger populations. The disease is pathologically defined by glomerular deposition of immunoglobulin A (IgA), often accompanied by complement component C3. Recent studies suggest that both the degree of glomerular IgA and C3 deposition may correlate with disease severity and outcomes. However, the independent prognostic value of glomerular C3 staining remains unclear. This study aimed to determine the association between glomerular C3 staining intensity and renal outcomes in Filipino patients with IgAN.

Methods: A retrospective cohort analytical study was conducted at the National Kidney and Transplant Institute (NKTI), including 90 biopsy-proven IgAN patients from 2021 to 2023. Inclusion criteria encompassed adults ≥18 years old with ≥8 glomeruli per biopsy and no prior immunosuppressive therapy. Demographic, clinical, histopathologic, and laboratory data were extracted. IgA and C3 staining intensities were evaluated via immunofluorescence. Outcomes included renal function indicators (eGFR, serum creatinine, proteinuria) and histological features (mesangial hypercellularity, segmental sclerosis, endocapillary hypercellularity, crescents). Descriptive statistics, Chi-square, Fisher's exact, and Kruskal-Wallis tests were used for analysis at a 0.05 significance level.

Results: The cohort had a median age of 32 years, with 57.78% females. Most patients demonstrated high IgA staining (53.33% with 3+), while C3 deposition was predominantly mild (48.89% with 1+; 33.33% trace). Mesangial hypercellularity was present in 27.78%, segmental sclerosis in 72.22%, and endocapillary hypercellularity in only 8.89%. A significant association was found between higher IgA staining and mesangial hypercellularity ($p = 0.0334$), with residual analysis confirming that 3+ IgA was strongly associated with M1 lesions. However, no significant associations were found between C3 staining and renal function (serum creatinine $p = 0.1760$; UPCR $p = 0.1644$) or key histopathologic markers including mesangial hypercellularity ($p = 0.0854$) and segmental sclerosis ($p = 0.4389$).

Conclusion: In this Filipino cohort with early-to-moderate IgAN, higher IgA staining was significantly associated with mesangial hypercellularity, reinforcing its role in active glomerular injury. Conversely, glomerular C3 staining was not significantly associated with renal dysfunction or histological severity, suggesting it may play a complementary rather than independent prognostic role. These findings highlight the importance of incorporating IgA intensity into histopathologic assessment while emphasizing the need for a multidimensional approach to risk stratification that integrates clinical and immunopathologic parameters.

Keywords：IgA Nephropathy, Glomerular C3 Deposition, Mesangial Hypercellularity, Renal Histopathology, Filipino Cohort

Poster Presentation：Glomerular Diseases
Poster No.：C0442
Abstract Submission No.：APCN20250134

How MicroRNA Expression and Target Prediction help to manage Nephrotic Syndrome in children?

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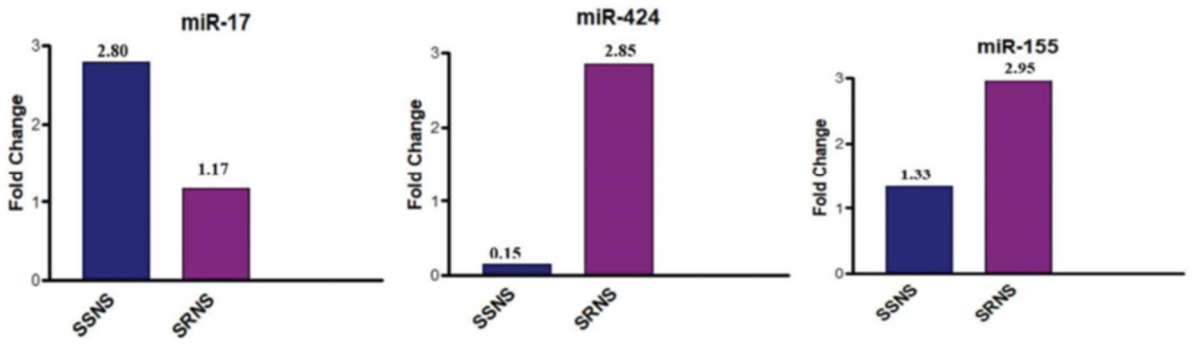
Abstract
Background: miRNAs have been shown to act as non-invasive biomarkers in serum and urine for a number of illnesses, including kidney disease. It has been found that miR-155 has a major role in kidney disease. The expression patterns of the microRNAs miR-17-5P, miR-155p, and miR-424-5p were examined in this study in both healthy subjects and children with steroid-sensitive nephrotic syndrome (SSNS) and steroid-resistant nephrotic syndrome (SRNS).
Materials and Methods: Total RNA was isolated from the urine samples from the three groups (SSNS n = 100, SRNS n = 100, and healthy control group n = 100). Bioinformatics tools such as miRWalk and miR-Tar link were used in predicting targets for the microRNAs. Online database and g profiler software are used to evaluate the targets based on the biological functions. The expression pattern for the candidate microRNAs was carried out using quantitative real time polymerase chain reaction (RT-PCR) equipment.
Results: MiR-155p, miR-424-5p, and miR-17-5P have been discovered to have stronger predictions related to the pathophysiology of illnesses, according to the miRtarlink. While miR-17 was down-regulated in the SRNS group and upregulated in the SSNS group, miR-424 and miR-155 were up-regulated in the SRNS group. In the SRNS group, miR-17-5p was downregulated, but miR-424-5p and miR-155p were upregulated.
Conclusion: The podocyte junction of the actin cytoskeleton is significantly influenced by the microRNA expression pattern. In the SRNS group, miR-17-5p is downregulated and miR-424-5p and miR-155p are increased. The pathophysiology of pediatric nephrotic syndrome can be better understood and managed by combining gene expression analysis with the study of potential microRNAs.

Keywords：Nephrotic syndrome; microRNAs Expression analysis; miRWalk



Poster Presentation：Glomerular Diseases
Poster No.：C0443
Abstract Submission No.： APCN20250168

The Associations Among the Use of Sodium-Glucose Cotransporter 2 Inhibitor, Histopathology and Clinical Outcome in Patients With Immunoglobulin A Nephropathy

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Abstract

Introduction：The effect of sodium-glucose cotransporter 2 inhibitor(SGLT2i) in retarding immunoglobulin A
Nephropathy (IgAN) progression was inconsistent. This study examined the associations among SGLL2i, pathology classification of IgAN, and clinical outcome.

Methods：Biopsy-proven IgAN adults from 2021 to 2024 were enrolled. Oxford classification, the use of SGLT2i, and laboratory data were assessed during the baseline period and followed till death, dialysis, or administrative censor. Severe GFR decline was defined as eGFR MDRD ≥ 50% decline from baseline. Propensity score matching (PSM) analysis was conducted to compare those with and without SGLT2i. Adjusted hazard ratios (aHRs) were evaluated for composite outcome using Cox hazards model. Linear mixed model was used to test pathology score, SGLT2i and eGFR change over time.

Results：

From a total of 142 subjects and 43 matched pairs, the incidence of dialysis and severe GFR decline were 5.8 vs. 8.5 per 1000 patient-month and 5.1 vs. 8.8 per 1000 patient-month. SGLT2i users had significantly lower risk of composite outcome after adjusting multivariate adjustment in PSM (aHR 0.62, 95% CI: 0.25–0.95; $P < 0.05$); whereas the association was insignificant after adjusting pathology classification (aHR 0.71, 95% CI: 0.20–1.71; $p = 0.222$). In linear mixed model, SGLT2i user had a trendy fashion in retarding eGFR decline (0.9 [–0.2, 2.4] mL/min/1.73 m²/year; $p = 0.089$). It was less benefit in Oxford pathology T2 and C2 lesions (–3.4 and –5.7 mL/min/1.73 m²/year, respectively; both $p < 0.05$).

Conclusion：The benefit of SGLT2i in retarding eGFR decline in IgAN was not convincing, especially in those with T2 and C2 lesions.

Keywords：Dialysis, MDRD, Immunoglobulin A nephropathy, linear mixed model, propensity score matching

Poster Presentation：Glomerular Diseases
Poster No.：C0444
Abstract Submission No.：APCN20250171

Preventive Effects of Nicorandil Against Contrast-Induced Nephropathy in Patients With Renal Insufficiency Undergoing Percutaneous Coronary Intervention: A Meta-Analysis of Randomized Trials

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Abstract

Background: Contrast-associated nephropathy (CIN) is a severe complication after percutaneous coronary intervention (PCI). Nicorandil, a K-ATP channel activator, has been widely used to treat angina pectoris and heart failure. Several studies have reported that nicorandil can improve renal perfusion. But its effect on CIN prevention is still inconsistent. This study aimed to assess the effect of nicorandil against CIN in patients with renal insufficiency undergoing PCI.

Methods: A literature search was performed in PubMed, BMC, and Embase databases within the past ten years. We have systematically searched electronic databases up to December, 2024 to find randomized controlled trials (RCTs) which assessed the effect of nicorandil on CIN, including the incidence of CIN and renal function (SCr and eGFR). Review Manager 5.4.1 software was used for the data analysis. Results were reported as mean differences (MD) for continuous variables and odds ratio (OR) for dichotomous variables with a 95% confidence interval (CI) using a random-effects model.

Results: A total of 630 participants from 3 RCTs were included. The incidence of CIN was significantly decreased in the nicorandil group compared to the control group (OR: 0.36; 95% CI: 0.18 to 0.72; p = 0.004; I2: 4%). Compared with the control group, patients receiving nicorandil had significantly lower SCr levels (-7.30 mmol/L; 95% CI: -11.33 to -3.27; p = 0.0004). The amount of eGFR also significantly decreased in the nicorandil group, but the decline was significantly higher in the control group (-3.22 mL/min/1.73 m2; 95% CI: -4.64 to -1.81; p = <0.00001) at 72 hours after the procedure.

Conclusions: Nicorandil may be preventative against the incidence of CIN in patients with renal insufficiency undergoing PCI. However, further studies with larger scale and better design are needed to confirm this result.

Keywords：Nicorandil, CIN, PCI, meta-analysis

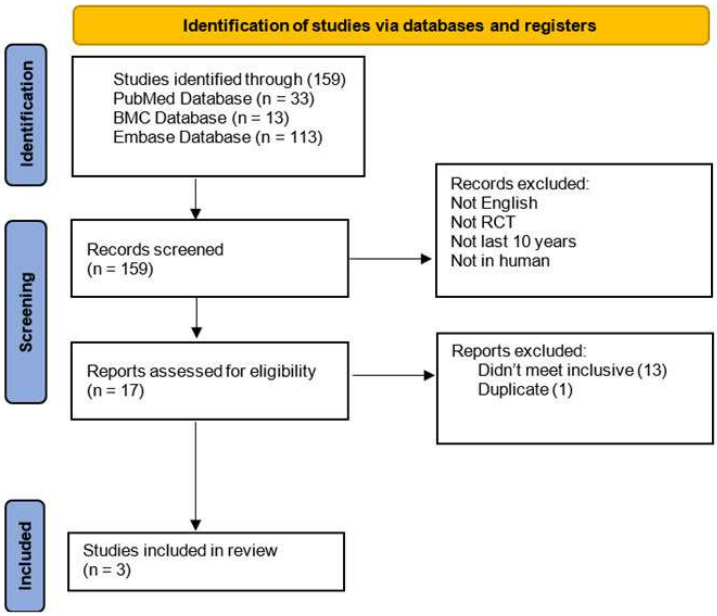

Figure 1. Systematic review flow chart study inclusion

Poster Presentation：Glomerular Diseases
Poster No.：C0445
Abstract Submission No.：APCN20250186

Milk in the Chest: Chylothorax Revealing Hidden Complications in Paediatric Nephrotic Syndrome

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Abstract

Introduction: Chylothorax is a rare and diagnostically challenging condition in the paediatric population, particularly when it manifests as an initial indicator of occult central venous thrombosis in the context of steroid-resistant nephrotic syndrome (SRNS). The interplay between hypercoagulability, lymphatic disruption, and immunosuppressive therapy adds complexity to clinical management.

Case Presentation: We report the case of a 10-year-old Malay boy with SRNS secondary to biopsy-confirmed minimal change disease (MCD), who developed a chylothorax in association with extensive central venous thrombosis. Despite immunosuppressive treatment with corticosteroids, cyclosporine, and rituximab, the patient showed no remission and experienced recurrent hospitalisations for edema control. He later presented with worsening edema, isolated left upper limb swelling, and radiological evidence of a large left pleural effusion. Chest tube thoracostomy yielded milky pleural fluid, and biochemical analysis confirmed the diagnosis of chylothorax. Contrast-enhanced computed tomography (CECT) of the thorax delineated a long-segment thrombus involving the left internal jugular, left subclavian, and left brachiocephalic veins. Laboratory tests showed no evidence of infection but revealed acute kidney injury and reduced levels of protein S and anti-thrombin activity on thrombophilia screening. The patient was managed with chest drainage, dietary modifications consisting of a low-fat diet rich in medium chain triglycerides (MCTs), and anticoagulation using subcutaneous fondaparinux. Immunosuppression was switched from cyclosporine to mycophenolate mofetil due to acute kidney injury and ongoing proteinuria. Follow-up imaging at three months showed complete resolution of left brachiocephalic vein thrombosis and improvement in the remaining venous segments.

Conclusion: This case highlights the complex relationship between nephrotic syndrome, thrombotic risk, and lymphatic dysfunction. Clinicians should consider central venous thrombosis in nephrotic patients presenting with chylothorax. Early recognition and multidisciplinary care are key to favourable outcomes.

Keywords：Chylothorax; Steroid-resistant nephrotic syndrome (SRNS); Minimal change disease (MCD); Central venous thrombosis; Hypercoagulability; Fondaparinux; Lymphatic disruption

Poster Presentation：Glomerular Diseases
Poster No.：C0446
Abstract Submission No.：APCN20250204

Case report: Nephrotic syndrome induced by lenvatinib treatment in a patient with von Hippel-Lindau syndrome

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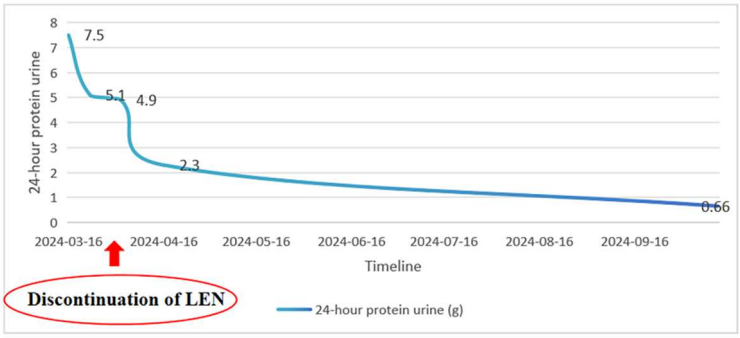
Abstract

Introduction: Von Hippel-Lindau (VHL) syndrome is an autosomal dominant hereditary disease characterized with mutiple organ tumors. Tyrosine kinase inhibitor (TKI) is one of the targetd treatment for VHL syndrome. Lenvatinib (LEN), an oral small-molecule multiple TKI, and proteinuria is one of the most common adverse events associated with LEN. This is the first report about LEN-induced TMA and FSGS-like lesion in a VHL syndrome patient.

Case description: A 50-year-old male was admitted due to the foaming in his urine and lower limbs edema since two months ago. Massive urine protein, hypoalbuminemia, and hypercholesterolemia were observed in Table 1. The urinary Bence-Jones protein, serum tumor markers, ANCA, ANA, PLA2R antibodies and complement were negative. In 2008, he was diagnosed with VHL syndrome via genetic testing, presenting with spinal hemangioblastoma, cerebellar hemangioblastoma, and pheochromocytoma. The patient underwent three surgeries to remove the tumors. Three years ago, he was diagnosed with pNET and treated with octreotide 20mg every month for 6 times (the last dose was two years ago). In January 2022, the patient was treated with LEN at 12mg/day. The patient has a history of hypertension and hypothyroidism for 2 years and diabetes for 3 months. Three months ago, he was diagnosed with acute cerebral infarction with no symptoms of movement and sensation abnormality and treated with mannitol for dehydration. Three days ago, the patient had a fever, accompanied by cough and expectoration and took oral ibuprofen, then the symptoms relieved. The patient's father died at the age of 50 (cause unknown), and his son and daughter have been diagnosed with VHL syndrome.

Discussion: In view of the nephrotic syndrome presenting in a VHL syndrome patient, we excluded other possible causes, tumor-related nephropathy and drug-related nephrotoxicity were finally considered. Then the renal biopsy was proved with thrombotic microangiopathy (TMA) and focal segmental glomerulosclerosis (FSGS)-like pattern (Figure 1). Based on the above analysis and renal pathology, the etiology of nephrotic syndrome in the patient was due to LEN-induced kidney damage. With the multidisciplinary advice of oncology department and the pNET was stable, lenvatinib was discontinued. The patient was followed up in the outpatient clinic for six months. After discontinuation of LEN and continuous supportive therapy, proteinuria slowly decreased to 0.66 g/day with stable renal function (Figure 2). Drug-induced kidney injury deserves further attention.

Keywords：Drug-Induced Kidney Injury, Nephrotic syndrome, von Hippel-Lindau syndrome, Tyrosine kinase inhibitor, Lenvatinib



Poster Presentation：Glomerular Diseases
Poster No.：C0447
Abstract Submission No.：APCN20250210

Biomarkers in Lupus Nephritis

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Abstract

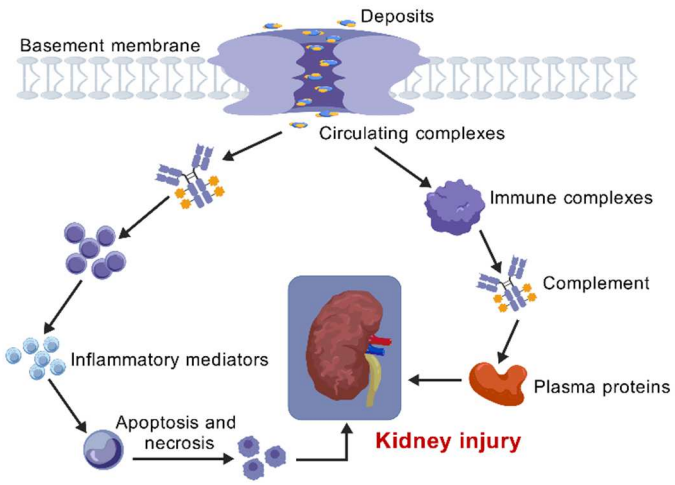
Introduction: Renal involvement is one of the most serious manifestations of systemic lupus erythematosus (SLE). While kidney biopsy is the gold standard for diagnosing lupus nephritis (LN), its invasiveness limits clinical application. Traditional markers such as serum creatinine, proteinuria, anti-dsDNA antibodies, and complement levels lack sufficient sensitivity and specificity for early LN detection. Novel biomarkers are urgently needed to improve diagnostic accuracy, guide prognosis, monitor therapeutic response, and detect early renal flares.

Methods: We conducted a comprehensive literature review across multiple databases including CNKI, Wanfang, CMA, and PubMed. This review summarizes recent findings on emerging LN biomarkers, including anti-C1q antibodies, monocyte chemoattractant protein-1 (MCP-1), neutrophil gelatinase-associated lipocalin (NGAL), and immunoglobulin-binding protein 1 (IGBP1).

Results: Anti-C1q antibodies, when combined with complement C3 and C4, significantly improve LN diagnostic and predictive performance. The combined negativity of anti-C1q, anti-dsDNA, C3, and C4 shows strong negative predictive value for renal involvement. Urinary MCP-1 levels correlate with disease activity and treatment response; persistently high levels are associated with poor response and chronic kidney damage, including fibrous crescents and tubular atrophy. Elevated urinary IGBP1 is linked to tubulointerstitial inflammation and may assist in histological differentiation of LN.

Conclusion: Although multiple novel biomarkers have been identified, none have yet been validated in large, multi-ethnic longitudinal cohorts. A single biomarker is unlikely to replace conventional parameters. Future research should focus on combining novel biomarkers with clinical indicators to enhance accuracy in monitoring disease progression, predicting renal flares, and tailoring treatment strategies in LN.

Keywords：Lupus Nephritis; Biomarkers; Anti-C1q Antibodies; MCP-1; Disease Monitoring



Poster Presentation：Glomerular Diseases
Poster No.：C0448
Abstract Submission No.：APCN20250239

Shared Molecular Networks and Potential Therapeutic Targets Between Lupus Nephritis and Anti-Neutrophil Cytoplasmic Antibody-Associated Nephritis: A Multidimensional Integrative Analysis

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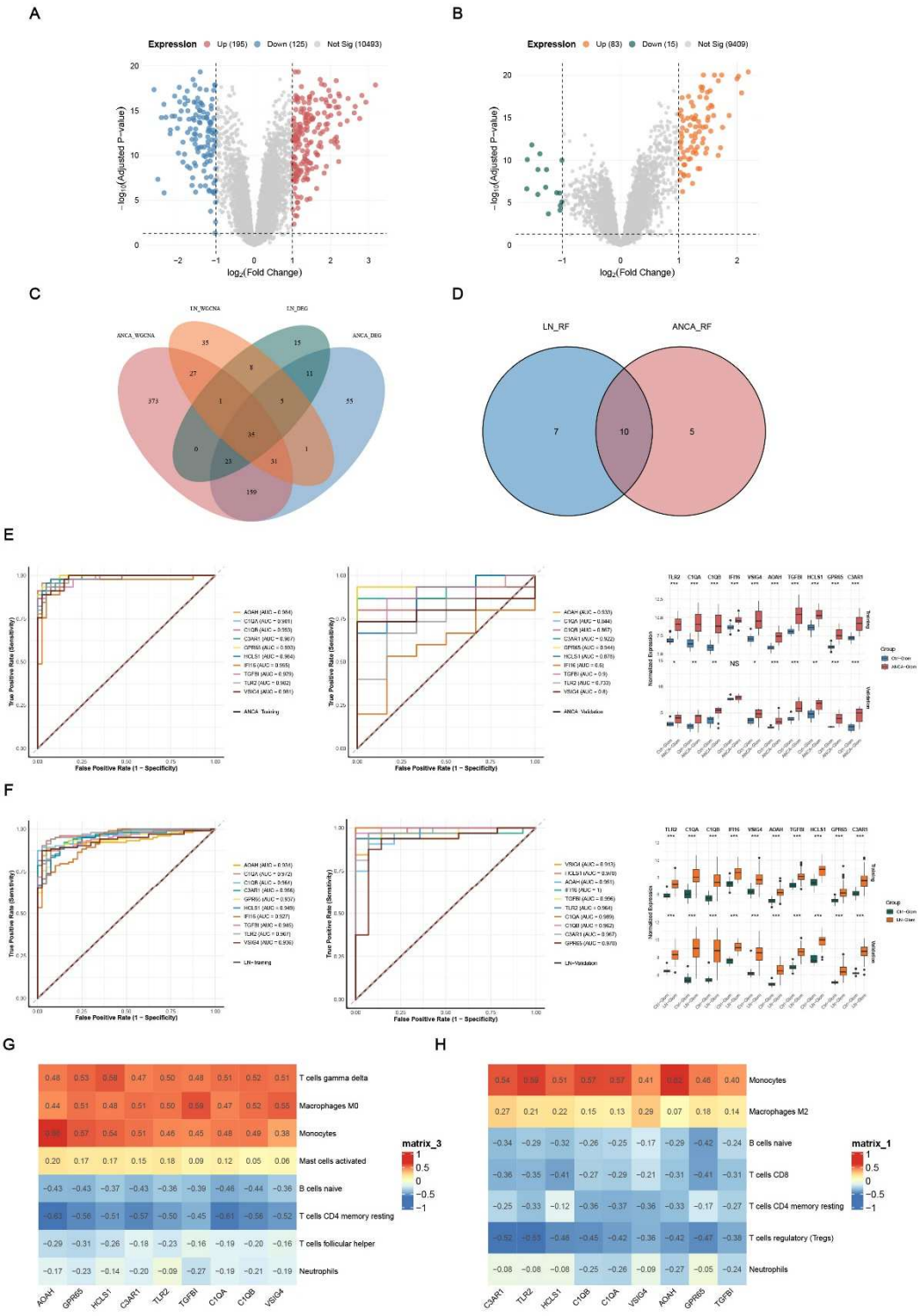
Abstract
Introduction: Renal involvement in systemic lupus erythematosus (SLE) and antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) manifests as lupus nephritis (LN) and ANCA-associated glomerulonephritis (AAGN), respectively. Both diseases are characterized by immune-mediated renal injury, and a significant proportion of lupus nephritis (LN) patients exhibit concomitant ANCA positivity in clinical practice. However, the shared molecular mechanisms underlying renal injury remain unclear.
Methods: We integrated datasets from the GEO database (three AAGN datasets; four LN datasets) and performed differential gene analysis, weighted gene co-expression network analysis (WGCNA), and random forest modeling to identify shared pathogenic genes. CIBERSORT-based immune infiltration analysis, consensus clustering, and targeted drug prediction (via DSigDB, molecular docking, and molecular dynamics simulation) were employed to elucidate function and clinical value.
Results: Nine core shared genes (TLR2, C1QA, C1QB, VSIG4, AOAH, TGFBI, HCLS1, GPR65, C3AR1) were identified, functionally enriched in complement activation, macrophage polarization, and metabolic-immune interaction pathways. A multi-gene diagnostic model based on these genes demonstrated high predictive efficacy in both training and external validation cohorts. All genes exhibited significant negative correlations with glomerular filtration rate (GFR). Immune infiltration analysis revealed that resting CD4+ memory T cells and naïve B cells were negatively correlated with all shared genes in AAGN, while regulatory T cells (Tregs) showed negative correlations in LN. Monocytes were positively correlated with all shared genes in both diseases. Consensus clustering and subsequent immune infiltration analysis further delineated distinct subtypes associated with heterogeneous immune states and clinical phenotypes. By prediction, Ephedrone and Pentachlorobiphenyl were the 2 most significant drugs, both associated with C1QA and C1QB.
Conclusion: AAGN and LN may share molecular mechanisms involving complement activation, macrophage polarization, and metabolic-immune crosstalk. The identified shared genes could serve as potential biomarkers and therapeutic targets for ANCA-positive LN.

Keywords：Lupus nephritis, ANCA-associated glomerulonephritis, Bioinformatics analysis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B
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−log10(Adjusted P−value)
log2(Fold Change)
C
LN_WGCNA
LN_DEG
ANCA_WGCNA
ANCA_DEG
D
LN_RF
ANCA_RF
7
10
5
E
True Positive Rate (Sensitivity)
False Positive Rate (1 − Specificity)
ANCA Training
ANCA Validation
Normalized Expression
Group
Ctr−Ctrn
ANCA−Ctrn
NS
F
True Positive Rate (Sensitivity)
False Positive Rate (1 − Specificity)
LN−Training
LN−Validation
Normalized Expression
Group
Ctr−Ctrn
LN−Ctrn
G
T cells gamma delta
Macrophages M0
Monocytes
Mast cells activated
B cells naive
T cells CD4 memory resting
T cells follicular helper
Neutrophils
AOAH GPR65 HCLS1 CSAR1 TLR2 TGFBI C1QA C1QB VSIG4
matrix_3
H
Monocytes
Macrophages M2
B cells naive
T cells CD8
T cells CD4 memory resting
T cells regulatory (Tregs)
Neutrophils
CSAR1 TLR2 HCLS1 C1QB C1QA VSIG4 AOAH GPR65 TGFBI
matrix_1

Poster Presentation：Glomerular Diseases
Poster No.：C0449
Abstract Submission No.：APCN20250247

Suppression of complement alternative pathway by monthly dosing of sefaxersen, a novel antisense oligonucleotide therapy in development for IgAN

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Abstract

Introduction: Increased activity of the complement alternative pathway (AP) contributes to the pathogenesis of IgA nephropathy (IgAN). Sefaxersen (IONIS-FB-LRx, RO7434656), an antisense oligonucleotide (ASO) against Factor B (FB), is the first mRNA-targeting therapy in late-stage development for IgAN. Here, we describe the PK profile and mechanism of action of sefaxersen using data from Phase 2 clinical studies in IgAN and geographic atrophy (GA).

Methods: Sefaxersen is a second-generation GalNAc-conjugated ASO for enhanced stability and targeted delivery to the liver. Sefaxersen 40, 70, or 100 mg was administered Q4W in 223 participants with GA (NCT03815825), and 70 mg in 23 participants with IgAN (NCT04014335). An additional dose was administered 2 weeks after the first dose in the majority of participants. Complement proteins were serially measured in plasma (FB, Bb) and urine (Factor Ba, IgAN patients only). Classical pathway and AP functional activity in serum were measured by Wieslab WCP and WAP assays, respectively.

Results: The plasma concentration profile of sefaxersen following SC injection shows rapid absorption, sharp decline post-Cmax due to tissue distribution, and slower terminal elimination phase driven by re-distribution from tissue, with apparent half-lives ranging from approximately 5-8 weeks.

Q4W dosing with sefaxersen rapidly and durably reduced complement proteins FB and Bb in plasma, and Ba in urine. Near maximum suppression of FB, Bb, and Ba levels was reached by earliest assessments at Week 5 and Week 9 and maintained over the dosing intervals in GA and IgAN patients, respectively. Plasma levels of FB and Bb were reduced by 69% and 73%, respectively, from baseline to

inferred steady-state in patients with GA at the 70 mg dose. Plasma FB levels were reduced by a mean of 69%, Factor Bb by 79%, and urine Factor Ba levels by 78% from baseline to inferred steady-state in IgAN.

The AP activity was reduced by 34% at inferred steady-state in GA at the 70 mg dose, and reduced by 39% after 9 weeks in IgAN, whereas classical pathway activity was maintained.

Conclusion: Sefaxersen demonstrates a pharmacokinetic profile supporting Q4W subcutaneous administration and provides durable reduction in AP complement components. Substantial reduction in AP functional activity was achieved while complement classical pathway activity was maintained, potentially maintaining host defense against pathogens.

This abstract was also submitted for the ERA 2025 congress. By submitting the abstract to APCNxTSN 2025, abstract authors declare that re-submitting the abstract is permitted by the organizers of the previous congress.

Keywords：sefaxersen, IgAN, complement, alternative pathway, ASO, IMAgINATION, Factor B

Poster Presentation：Glomerular Diseases
Poster No.：C0450
Abstract Submission No.：APCN20250256

High Serum IgA/C3 Ratio Predicts Disease Progression in IgA Nephropathy: A Retrospective Cohort Study

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[7] Division of clinical informatics, department of digital medicine, Taichung Veterans General Hospital

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Abstract

Background:

IgA nephropathy (IgAN) is the most common form of primary glomerulonephritis globally, characterized by mesangial deposition of IgA-containing immune complexes and complement activation. Identifying reliable biomarkers for early risk stratification remains critical for improving long-term outcomes. This study investigated the prognostic value of the serum IgA/C3 ratio at diagnosis in predicting disease progression in IgAN.

Methods:

We retrospectively analyzed 330 patients with biopsy-proven IgAN. Receiver operating characteristic (ROC) curve analysis identified an optimal IgA/C3 ratio cutoff of 3.386 for risk stratification. Patients were grouped based on this threshold (<3.386 vs. ≥3.386), and the longitudinal decline in urine protein-to-creatinine ratio (UPCR) was compared. Kaplan–Meier analysis and log-rank testing were used to assess differences in proteinuria trajectories.

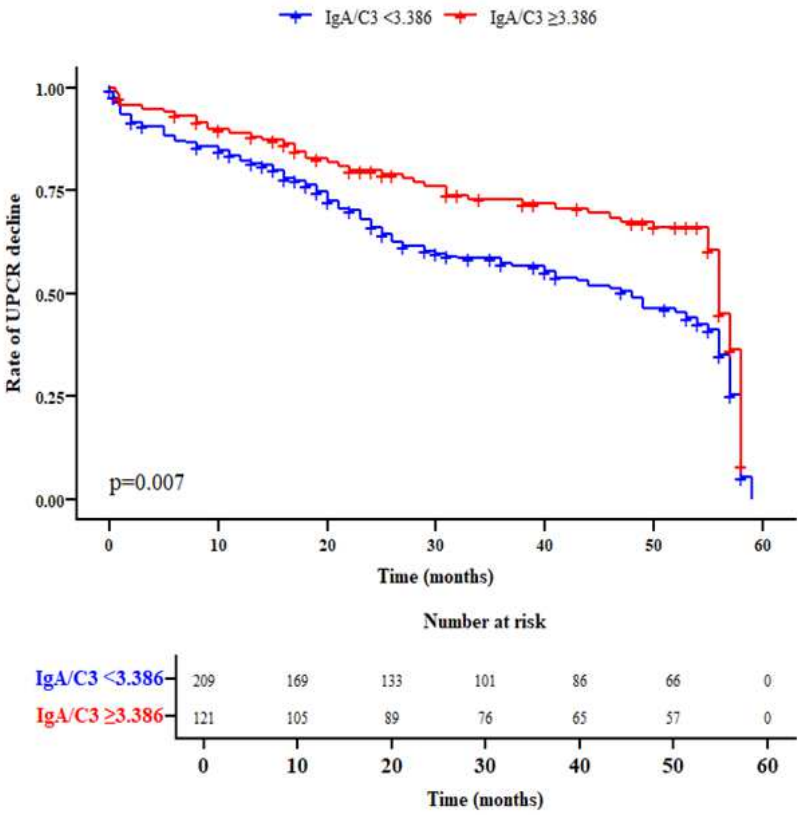
Results:

Kaplan–Meier analysis revealed a significant difference in UPCR decline between the two groups. Patients with an IgA/C3 ratio ≥3.386 exhibited a significantly slower reduction in proteinuria compared to those with a ratio <3.386 (log-rank test, $p = 0.007$). A higher IgA/C3 ratio at disease onset was associated with less favorable proteinuria control over time.

Conclusions:

The serum IgA/C3 ratio is a readily accessible and significant prognostic biomarker for disease progression in IgAN. Early identification of high-risk patients using this ratio may facilitate more aggressive monitoring and tailored therapeutic strategies.

Keywords：IgA nephropathy, Serum IgA/C3 ratio


IgA/C3 <3.386
IgA/C3 ≥3.386
Rate of UPCR decline
1.00
0.75
0.50
0.25
0.00
p=0.007
0
10
20
30
40
50
60
Time (months)
Number at risk
IgA/C3 <3.386
209
169
133
101
86
66
0
IgA/C3 ≥3.386
121
105
89
76
65
57
0
0
10
20
30
40
50
60
Time (months)

Poster Presentation：Glomerular Diseases
Poster No.：C0451
Abstract Submission No.：APCN20250258

Long-Term Prognosis in Japanese Patients with Lupus Nephritis: Impact of Remission Failure and Relapse

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Abstract

Introduction: While pathological classification and therapeutic response are known to influence the prognosis of lupus nephritis, limited data on the long-term outcomes are available based on these factors. This study aimed to elucidate the long-term prognosis of Japanese patients with lupus nephritis, specifically focusing on pathological classification and treatment strategies.

Methods: We retrospectively analyzed 54 patients diagnosed with lupus nephritis via renal biopsy at our institution between April 2002 and March 2024. Inclusion criteria were urinary protein excretion ≥0.5 g/gCr, estimated glomerular filtration rate (eGFR) ≥15.0 mL/min/1.73 m², and follow-up duration exceeding 6 months. Primary endpoints were time to remission—defined as urinary protein <0.5 g/gCr without a sustained ≥40% decline in eGFR—and time to composite events, including sustained serum creatinine increase ≥50%, renal death, or all-cause mortality.

Results: The mean age at diagnosis was 43 yr, and 42 patients (78%) were female. Baseline eGFR averaged 74.2 mL/min/1.73 m², and median proteinuria was 4.0 g/gCr. The mean initial prednisolone dose was 41 mg/day. Methylprednisolone pulse therapy was administered in 23 patients (43%), and 18 patients (33%) received hydroxychloroquine. Immunosuppressive therapy included a single agent in 31 patients (57%) and dual agents in 16 patients (30%). No patients received biologics during the initial remission induction phase. Pathological classification revealed five patients with Class II (two with coexisting Class V), 12 with Class III (six with Class V), 21 with Class IV (five with Class V), and 16 with isolated Class V disease. Of the total cohort, 50 patients (93%) achieved remission; however, nine (18%) of these experienced relapse, defined as proteinuria ≥1 g/gCr. Remission and relapse rates were not significantly associated with pathological classification or treatment modality. A total of 12 composite events were recorded: 10 cases of sustained serum creatinine elevation ≥50% and two deaths. Among patients who achieved remission without relapse (n = 41), only one event occurred. In contrast, three events occurred in four patients who failed to achieve remission, and eight events were observed among the nine patients who relapsed. The 10-yr overall survival rate was 76%, with no significant differences across pathological classifications or treatment regimens.

Conclusion: Our findings indicate that in Japanese patients with lupus nephritis, long-term prognosis is more strongly associated with remission status and relapse occurrence than with pathological classification or treatment type. Therefore, therapeutic strategies should prioritize achieving and maintaining remission to improve long-term renal and overall outcomes.

Keywords：Japanese patients, long-term prognosis, lupus nephritis, relapse, remission

Poster Presentation：Glomerular Diseases
Poster No.：C0452
Abstract Submission No.：APCN20250260

The Correlation of Proteinuria, Hematuria, and Serum Creatinine with Pathology Severity Index in Glomerulonephritis Patients (Longitudinal Study: Development of Clinico-Pathological Scoring in Glomerulonephritis Patients)

Gede Wira Mahadita[1,2,3]; I Gde Raka Widiana[1,2,3]; Reny Prasetya Duarsa[2]; Yenny Kandarini[1,2,3]; Ni Wayan Winarti[2]; Nyoman Paramita Ayu[1,2,3]

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Abstract

Introduction: The incidence of Chronic Kidney Disease (CKD) increases annually in Indonesia and globally. GN is an etiology of CKD that often goes undetected and has a poor prognosis. The diagnosis of GN should be established by biopsy, and further prognosis assessment also requires scoring. It is imperative to conduct additional research to develop a score for all forms of GN and its correlation with clinical parameters such as serum creatinine, hematuria, and proteinuria.
Objective: To determine the relationship of pathological severity index (PSI) scoring to proteinuria, hematuria, and SC in GN patients.
Methods: This study used an observational longitudinal study design from 2016-2024. Inclusion criteria: age 12-65 years and undergoing kidney biopsy procedures at Prof. Dr. IGNG Ngoerah Hospital; the number of glomeruli in the biopsy core 4. Proteinuria and hematuria were taken from pre-biopsy data. PSI is a semiquantitative renal histopathologic score, which is based on the injury pattern found in each renal compartment: glomerular (Glomerular Index), tubular (Tubular Index), interstitial (Interstitial Index), and vascular (Vascular Index). The glomerular index score consists of the sum of the abnormality score+activity+severity+distribution of lesions in the glomerulus. Tubular index score: consists of the sum of Abnormality + Activity + Distribution + Cast in tubules. The interstitial index score consists of Abnormality + Activity + Distribution. Vascular index score consists of vascular abnormality+activity score. Data analysis included a bivariate Pearson/Spearman correlation test with a significance limit of $p<0.05$ and multivariate analysis with linear regression.
Results: 150 subjects [male/female=72 (48%)/78 (52%); mean age=28.37 12.22 years] who met the inclusion-exclusion criteria were included in this study. The most significant proportion of consecutive post-biopsy GN diagnoses: Lupus Nephritis (LN) 58 people (38.7%), FSGS 29 people (19.3%); IgA Nephropathy 17 people (11.3%); Minimal Change Disease 16 people (10.7%); MPGN 10 (6.7%); Membranous 4 people (2.7%); and the remaining 3% with other diagnoses. The average PSI score and the components of each index are as follows: PSI score=10.13 of 4.56; Glomerular Index score=4.49 of 2.32, Tubular Index score=2.9 of 1.56; Interstitial index score=2.36 of 1.78, and Vascular Index=0.41 0.88. PSI was significantly positively correlated with proteinuria ($r=0.299$; $p<0.01$). PSI also correlated with hematuria ($r=0.168$; $p=0.04$) and SC ($r=0.255$; $p=0.002$). From multivariate linear regression analysis, only proteinuria and SC influenced PSI [(PSI=7.57+0.845*Proteinuria-Urinalysis+0.471*SC)] with adjusted R2= 11.6% ($p<0.01$).
Conclusion: PSI score associated with hematuria, proteinuria, and SC in GN patients

Keywords：Pathology Severity Index, proteinuria, hematuria, and glomerulonephritis

Poster Presentation：Glomerular Diseases
Poster No.：C0453
Abstract Submission No.：APCN20250264

Retrospective Analysis of the Outcomes and Predictors of Primary IgA Nephropathy –A South Indian Experience

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Abstract

Introduction

IgA nephropathy is the most prevalent primary glomerulonephritis worldwide. Indian IgAN population exhibit relatively rapid decline of kidney function.Hypertension, nephrotic range proteinuria, and kidney biopsy findings including the degree of interstitial fibrosis and sclerosed glomeruli associate with poor prognosis.

Methods

We retrospectively analysed biopsy proven cases of IgAN at AIIMS, Mangalagiri between March 2022 and March 2025. Of 72 cases, after excluding secondary, stage 5 CKD and irregular follow up, 27 adult patients (age>18 years) were studied. The outcomes were response to treatment at 6 months and 12 months in terms of proteinuria reduction(response- at least 50% reduction from baseline proteinuria) and 5mL/min/1.73m2 eGFR decline from baseline. Baseline demographic characteristics, laboratory and kidney biopsy findings were compared between responders and non-responders. Response to treatment approaches like oral prednisolone, targeted-release budesonide(TRB) & Mycophenolate Mofetil (MMF)were assessed.

Results

The mean age was 35±10 years,59% males. Majority presented with nephrotic syndrome (NS) (33%), followed by sub-nephrotic proteinuria (25.9%), nephritic syndrome (18.5%) & rapidly progressive renal failure (14.8%). 3.7% had asymptomatic microscopic haematuria and 3.7% had gross haematuria. The mean BP at baseline was 142± 24 / 93± 17 mmHg. Mean 24hour protein, serum albumin, serum creatinine was 2452±2632 mg, 3.7±0.4g/dL, 1.8±0.8mg/dL respectively. Responders at 6 months had significantly lower baseline systolic BP(135.5 ±12.97mm Hg vs 155.44±35.97mm Hg, p= 0.04), lower mean serum creatinine (1.46±0.56 mg/dL vs 2.46 ± 0.80 mg/dL , p= 0.001) & higher eGFR (63.88 ± 28.22 vs 35.58 ± 20.86 mL/min/1.73m2,p =0.014). 44% (12) had 5ml/kg/1.73m2 decline in eGFR at 6 months. 3(11%) had progressed to stage V CKD at 1 year. Patients with more than 5 ml/min eGFR decline at 6 months had significantly higher mean 24 hr urine protein compared to those with less rapid progression (1828 mg ±1245 mg vs 894±868 mg, p=0.046). Haas's score of 2 and lesser correlated with better response to treatment(p=0.019). 52%(14) patients were on TRB, while 41%(11) were on oral prednisolone.20% of those on prednisolone,25% percent of those on budesonide and 50% of those on prednisolone with MMF had significant decline in eGFR at 6 months, while none did in the TRB with MMF group.

Conclusion

In our cohort, NS was the most common presentation of IgAN. systolic blood pressure and serum creatinine were independent predictors of treatment response. Higher 24hour proteinuria correlated with rapid eGFR decline. Targeted-release budesonide combined with MMF showed promising results in preserving kidney function.

Keywords：IgAN, predictors of treatment outcome

TABLE 1. BASELINE CHARACTERISTICS OF TREATMENT RESPONDERS AND NON-RESPONDERS AT 6 MONTHS

BASELINE INDICES	RESPONDERS AT 6 MONTHS		NON-RESPONDERS AT 6 MONTHS		
	MEAN	SD	MEAN	SD	P VALUE
AGE (years)	34.78	10.38	36.33	10.10	0.714
SBP (mm Hg)	135.5	12.97	155.44	35.97	0.044
DBP (mm Hg)	89.94	10.10	100.44	25.78	0.138
HB(g/dL)	12.08	2.67	12.24	1.68	0.875
TLC (per microlitre)	11151	4687	9575	2868	0.366
PLATELET (lakhs)	3.10	0.78	2.72	0.38	0.180
24 HR URINE PROTEIN (mg)	2452	2632	2900	1648	0.646
S. ALBUMIN(g/dL)	3.81	0.42	3.53	0.37	0.112
CHOLESTEROL (mg/dL)	203	85	208	36	0.880
TRIGLYCERIDES (mg/dL)	157	73	206	105	0.167
HDL (mg/dL)	43	7.7	39	15.7	0.352
LDL (mg/dL)	110	35	110	46	0.977
CREATININE (mg/dL)	1.46	0.56	2.46	0.80	0.001
eGFR(mL/min/1.73m2)	63.88	28.22	35.58	20.86	0.014

Poster Presentation：Glomerular Diseases
Poster No.：C0454
Abstract Submission No.：APCN20250269

A 1-year-old Boy With MIRAGE Syndrome And Nephrotic Syndrome, Whose Kidney Histopathology Revealed Membranous Nephropathy-like Findings: A Case Report

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Abstract

Introduction:

MIRAGE (myelodysplasia, infection, restriction of growth, adrenal hypoplasia, genital phenotypes and enteropathy) syndrome is a severe congenital immunodeficiency disorder, which was identified as a monogenic disease. The SAMD9 gene on chromosome 7, which is the disease-causing gene for MIRAGE syndrome, undergoes mutations (gain-of-function) that lead to excessive formation of late endosomes. Histopathological confirmation has been reported in fewer than 10 cases, with findings consistent with interstitial nephritis or suggestive of glomerular diseases like focal segmental glomerulosclerosis, however, there are no reports of membranous nephropathy-like findings.

Case:

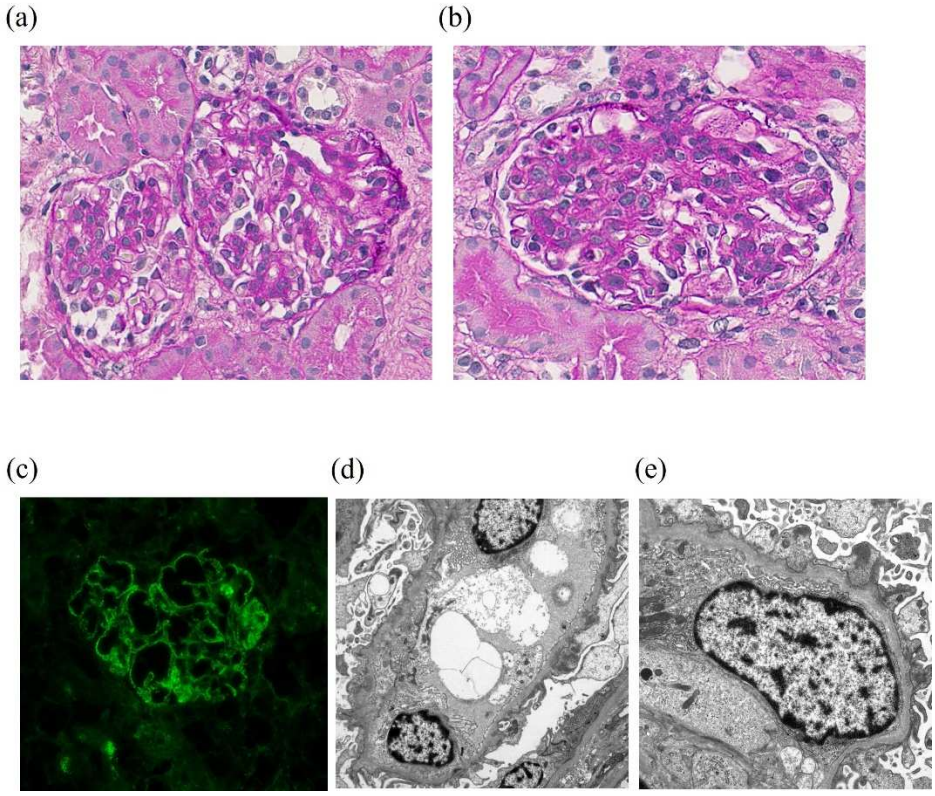
A male neonate was delivered at 35 weeks of gestation with birthweight of 1492 g. He had low platelet counts and anemia, recurrent infection, growth restriction, high level of adrenocorticotropic hormone, low level of cortisol and no identification of the adrenal glands, hypospadias and intractable diarrhea. A known variant (c.3877C>T, p.R1293W) in SAMD9 was identified in a heterozygous state by whole exome sequencing, leading to a genetically confirmed diagnosis of MIRAGE syndrome at the age of 3 months. He had repeated increases in cortisone stress doses due to recurrent infections, and developed hypertension. At 16 months of age, he presented with microscopic hematuria. Over time, there was progressive increase in proteinuria, which met the criteria for pediatric idiopathic nephrotic syndrome (NS). He developed systemic edema, meaning he was unable to discontinue regular albumin infusions. Blood examination revealed hypoalbuminemia (1.8 g/dL), normal kidney function, and normal complement activity. His titer of antineutrophil cytoplasmic antibody, anti-DNA antibody, anti-glomerular basement membrane (GBM) antibody and antinuclear antibody were normal. Urine examination revealed proteinuria (urine protein/creatinine ratio; 42 g/gCr) and microscopic hematuria. At 18 months, he underwent a kidney biopsy. Light microscopic findings revealed diffuse mesangial proliferation and partial segmental glomerular lobulation with neither crescentic legion nor sclerotic glomeruli(Fig.a,b). Immunofluorescence staining demonstrated diffuse granular deposition of IgG along the GBM and dominant deposition of IgG in mesangial matrix (Fig.c). Electron microscopy revealed foot process effacement and diffuse subepithelial electron-dense deposits (Fig.1d,e). Although there were membranous nephropathy-like findings, chronic changes were scarce. After 4 weeks of prednisolone therapy following a pattern similar to pediatric idiopathic NS, proteinuria persisted, leading to diagnosis of steroid-resistant NS. At 21 months of age, the patient died of multi-organ failure as a result of severe gastrointestinal infection and necrotizing enterocolitis.

Conclusion:

This is the first case with histologically membranous nephropathy-like findings and marked

mesangial proliferation in an child with MIRAGE syndrome with NS.

Keywords：Membranous nephropathy, MIRAGE syndrome, Nephrotic syndrome, Steroid resistant



Poster Presentation：Glomerular Diseases
Poster No.：C0455
Abstract Submission No.：APCN20250288

A Management Dilemma: Immunosuppressive Therapy in a Case of ANCA Associated Vasculitis and Tuberculosis Lymphadenitis

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Abstract

Background and Aims:

The anti-neutrophil cytoplasmic antibody (ANCA) associated vasculitis (AAV) is a rare systemic autoimmune disease caused by the inflammation of small sized blood vessels, and can lead to organ threatening or life threatening complications without prompt treatment with immunosuppressive therapy. However, immunosuppressive therapy increases infective risks, making the treatment particularly challenging in patients with concurrent infections. Here we present the management of a patient who was diagnosed with concomitant AAV and active tuberculosis (TB) lymphadenitis.

Case Presentation:

A 78 year-old male patient presented for rapidly declining kidney function, with creatinine rising to 218 umol/L from his baseline of 77 umol/L 1 year ago. This was associated with microscopic hematuria (756 urinary red blood cells (RBCs) per uL, 28% dysmorphic) and sub-nephrotic range proteinuria (urine protein/creatinine ratio (uPCR) 1.232 g/g). Autoimmune markers were positive for anti-nuclear antibody and anti-myeloperoxidase (MPO) antibody. His kidney biopsy revealed pauci-immune crescentic glomerulonephritis, demonstrating 16% fibrocellular/cellular crescents, 36% global sclerosis and 25% tubular atrophy with interstitial fibrosis. He had cervical lymphadenopathy concurrently, for which an excisional biopsy showed suppurative necrotizing granulomatous inflammation with polymerase chain reaction confirming TB. He was commenced on anti TB treatment and initiated on induction immunosuppressive therapy with dose attenuated pulse intravenous (IV) methylprednisolone 200 mg (4.22 mg/kg) for 2 doses and rituximab 375 mg/m2 for 4 doses, followed by a rapid tapering of oral prednisolone. The choice of corticosteroid regimen was based on the common use of adjuvant IV dexamethasone (0.3 mg/kg for 7 days) in TB meningitis, and we hypothesized that an equivalent dose of methylprednisolone would be safe in our patient. Twenty weeks post treatment initiation, his renal function improved to a creatinine of 170 umol/L, corresponding to an eGFR of 35 mL/min/1.73 m2 (Figure 1a). His AAV disease activity, as evidenced by microscopic hematuria and proteinuria, has also improved significantly (Figure 1b).

Conclusion:

Acute AAV with concurrent active TB infection at presentation is rare and represents a treatment conundrum, for which various immunosuppressive regimens have been explored, ranging from methylprednisolone alone to more intensive regimens including corticosteroid and cyclophosphamide. Our case has demonstrated that it is safe and effective to use an attenuated regimen of corticosteroids combined with rituximab as induction therapy to manage patients requiring immunosuppression for AAV while undergoing anti TB treatment. Further large-scale case studies are required to validate these findings and determine the optimal treatment strategy.

Keywords：ANCA associated vasculitis, immunosuppressive therapy

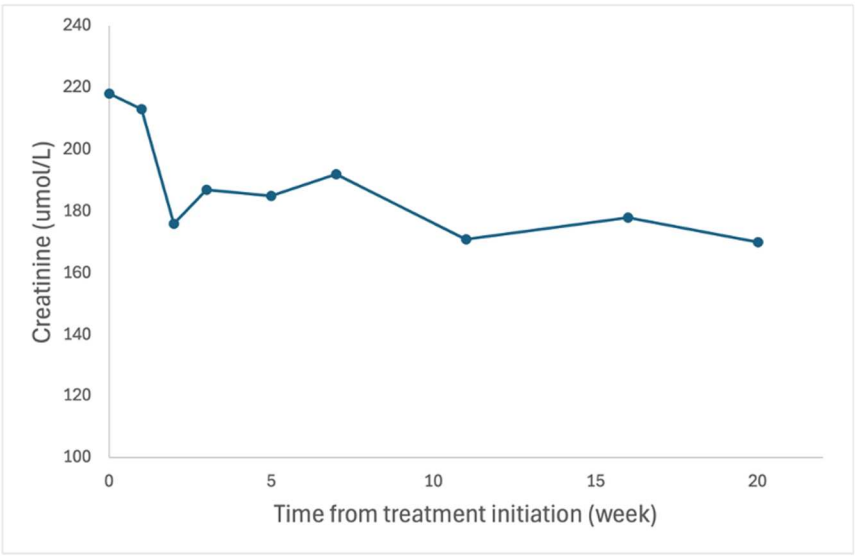

Figure 1a Renal function trend following initiation of immunosuppressive therapy

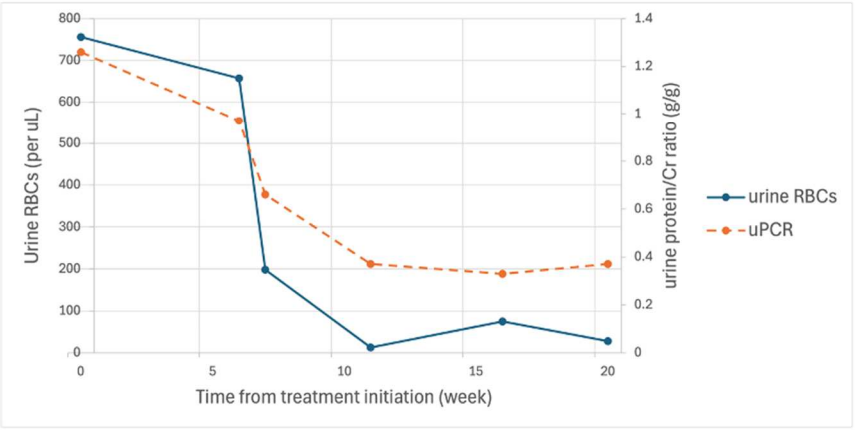

Figure 1b Degree of hematuria and proteinuria following initiation of immunosuppressive therapy

Poster Presentation：Glomerular Diseases
Poster No.：C0456
Abstract Submission No.：APCN20250299

Clinical Predictors of Relapse after Rituximab Discontinuation in Adult Patients with Steroid-Dependent Nephrotic Syndrome Achieving Glucocorticoid-Free Remission

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Abstract

Introduction: Repeated administration of rituximab often induces glucocorticoid-free remission in most patients with steroid-dependent nephrotic syndrome (SDNS). However, predictors of relapse after rituximab discontinuation are not well characterized. This study aimed to identify clinical predictors of relapse following rituximab discontinuation in adult SDNS patients.

Methods: We screened 56 adult patients (≥18 years) with SDNS who received rituximab at our institution between December 2014 and March 2024. Of these, 33 patients who achieved glucocorticoid-free remission and subsequently discontinued rituximab were included. Rituximab (500 mg fixed dose) was administered every six months and discontinued after a median of six infusions (range: 4–12). All patients were followed for at least six months of follow-up after rituximab discontinuation. Associations between relapse and 18 clinical variables—including rituximab dose per body surface area (RTX/BSA), age at disease onset, sex, peak proteinuria, and number of rituximab infusions—were retrospectively evaluated using Kaplan–Meier analysis and Cox proportional hazards models.

Results: The mean age at disease onset and rituximab discontinuation was 29 and 43 years, respectively; 55% of the cohort were female. During the 2 years preceding rituximab initiation, a total of 45 relapses occurred among the 33 patients, and median peak proteinuria was 6.67 g/gCr. Importantly, no patient relapsed while receiving rituximab. During a median follow-up of 784 days after rituximab discontinuation, 12 of 33 patients (36%) relapsed. The 2- and 5-year cumulative relapse rates were 32% and 55%, respectively. In univariate Cox regression analysis, RTX/BSA was inversely associated with relapse risk. However, neither RTX/BSA nor other factors were retained as significant independent predictors in the multivariate model. Using the minimum p-value approach based on the log-rank test, the optimal RTX/BSA cutoff was identified as 274.2 mg/m². Restricted cubic spline modeling showed an upward trend in relapse risk when RTX/BSA fell below approximately 270–300 mg/m². Kaplan–Meier analysis using the 274.2 mg/m² cutoff revealed that the high-dose group (n = 27) had a significantly lower relapse rate than the low-dose group (n = 6) ($p = 0.018$), with 2-year relapse rates of 26% and 58%, respectively.

Conclusion: An RTX/BSA below 274.2 mg/m² may be a clinical predictor of relapse after rituximab discontinuation in adult SDNS patients. Individualized rituximab regimens, such as shortening treatment intervals or using multidose protocols, may be considered for patients with larger BSAs to maintain sustained remission.

Keywords：Japanese patients, clinical predictor, rituximab, steroid-dependent nephrotic syndrome, body surface area

Poster Presentation：Glomerular Diseases
Poster No.：C0457
Abstract Submission No.：APCN20250300

Urinary Remission Following Tonsillectomy and Steroid Pulse Therapy in a Taiwanese Patient with IgA Nephropathy

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Abstract
Case Presentation:
A 45-year-old man initially developed high fever and haematuria at the age of 30, leading to a diagnosis of nephritis. Antihypertensive therapy was initiated but discontinued after approximately two years. Thereafter, annual health check-ups consistently revealed haematuria and proteinuria, which remained untreated. At the age of 43, prior to relocating to Japan, a renal biopsy in Taiwan confirmed a diagnosis of IgA nephropathy, and steroid therapy was initiated. Upon his arrival in Japan at age 44, he continued oral medications (valsartan 320 mg, benzbromarone 50 mg, rosuvastatin 10 mg, and prednisolone 5 mg) sent from Taiwan every three months. At age 45, a workplace health screening revealed haematuria, proteinuria, and impaired renal function, prompting referral by an occupational physician. At the initial evaluation, serum creatinine was 1.47 mg/dL, urinalysis showed proteinuria (3+, 1.3 g/gCr), and haematuria (U-RBC 3+), indicating active urinary findings. Following a detailed explanation of the standard Japanese treatment approach—tonsillectomy combined with steroid pulse therapy—and after obtaining informed consent, the patient underwent the procedure. One year after treatment, urinary abnormalities had resolved, and serum creatinine improved to 1.32 mg/dL.
Discussion:
Although renal dysfunction likely attributable to chronic histologic changes persisted, urinary abnormalities resolved. Tonsillectomy combined with steroid pulse therapy is considered a "Japan-specific treatment" according to the KDIGO international guidelines and is not recommended as a global standard. Nonetheless, this case suggests that the approach may be beneficial in non-Japanese patients, warranting further investigation through additional case studies.

Keywords：IgA nephropathy, Steroid pulse therapy combined with tonsillectomy, Urinary remission

Poster Presentation：Glomerular Diseases
Poster No.：C0458
Abstract Submission No.：APCN20250309

Single-Cell Spatial Profiling of IgA Nephropathy with CODEX Technology

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Abstract

Background:

Kidney diseases involve complex microenvironments shaped by diverse cellular interactions and molecular networks. Understanding the spatial architecture of these microenvironments is critical for elucidating disease mechanisms and discovering novel therapeutic targets. While conventional methods like immunofluorescence (IF) and immunohistochemistry (IHC) enable spatial visualization, they are limited in multiplex capacity. High-dimensional techniques such as mass cytometry and single-cell RNA sequencing (scRNA-seq) lack spatial context, restricting their utility in tissue-level analyses.

Objective:

This study aims to apply CODEX (CO-Detection by Indexing), a high-dimensional multiplexed spatial proteomics technology, to human kidney tissue for detailed microenvironmental analysis in IgA nephropathy (IgAN), a common form of chronic glomerulonephritis lacking curative therapy. Despite extensive research over the past five decades, current pathological evaluations remain focused on macroscopic changes, providing limited insight into cellular dynamics and microenvironmental complexity.

Methods:

Using the PhenoCycler-Fusion system (Akoya Biosciences), we conducted multiplexed immunofluorescence staining on formalin-fixed, paraffin-embedded (FFPE) renal biopsy samples from IgAN patients. The CODEX platform enabled simultaneous detection of over 40 protein markers at single-cell resolution. To enhance tissue segmentation and spatial quantification, we integrated AI-based image analysis using DenseNet (Indica Labs) to delineate glomerular and tubular regions, followed by HighPlex FL-based quantitative analysis.

Results:

Our approach allowed comprehensive spatial mapping of disease-associated cell types, including mesangial cells, immune infiltrates, and tubular components. Distinct spatial patterns and activation states were observed between patients with severe versus mild vascular involvement, suggesting potential markers for disease stratification. The integration of CODEX with AI-assisted image analysis offered higher-dimensional insight beyond traditional qualitative pathology.

Conclusion:

This study demonstrates the feasibility and utility of CODEX for spatial single-cell analysis in renal pathology. By uncovering previously unresolvable microenvironmental heterogeneity in IgAN, this approach offers a powerful platform for identifying disease mechanisms and potential therapeutic targets. Moreover, its compatibility with FFPE tissues enables retrospective analysis without requiring additional biopsies, enhancing its clinical applicability.

Keywords：IgAN, CODEX

Poster Presentation：Glomerular Diseases
Poster No.：C0459
Abstract Submission No.：APCN20250313

The Spectrum of Renal Involvement in Thalassemia: A Cross-Sectional Study on Ferritin, Hematuria, Proteinuria, and Kidney Function

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[2] Division of Hematology, Department of Internal Medicine, Universitas Indonesia Hospital, Depok, Indonesia

Abstract

Background

Thalassemia is a widespread health issue in Southeast Asia. Despite its prevalence, there's a lack of comprehensive data on renal involvement in thalassemia patients. Research into kidney function abnormalities in this population is now crucial, especially given the increased use of deferasirox and the improved life expectancy of these patients. A major concern for individuals receiving regular blood transfusions is the unavoidable risk of iron overload in various bodily tissues, including the kidneys, which can lead to organ dysfunction. Given the scarcity of studies exploring kidney function in iron-overloaded patients and its correlation with kidney function, this study aims to analyse the spectrum of renal involvement in thalassemia patients referred to the Thalassemia Clinic at Universitas Indonesia Hospital in 2025.

Methods

This cross-sectional study was conducted in Thalassemia Clinic of Universitas Indonesia Hospital, Indonesia in 2025. Blood sample was obtained to measure creatinine, glomerular filtration rate, haemoglobin and ferritin. In addition, urine specimen was collected to determine proteinuria and haematuria. Twenty-three subjects with diagnosis of thalassemia requiring blood transfusion and chelation therapy were included in the study. Subjects with known primary kidney disease, systemic disease affecting kidney functions and acute infections were excluded in this study. Statistical analysis was performed using Statistical Package for Social Science (SPSS) version 27. Correlation between serum ferritin level and proteinuria along with haematuria were determined by Pearson's Correlation, while correlations of blood transfusion and ferritin levels with creatinine and glomerular filtration rate were determined by Spearman's coefficient.

Results

There was no significant correlation between severity of anemia and kidney function. There were significant correlations between frequency and total volume of blood transfusion and glomerular filtration rate ($p<0.05$). Significant correlations were also found between serum ferritin levels and proteinuria, hematuria, and lower kidney function ($p<0.05$).

Conclusion

Blood transfusion and higher serum ferritin levels (mean 5534.9 ng/mL) are correlated with lower kidney function and proteinuria. As this study found that higher serum ferritin levels and frequent blood transfusion might be linked with lower kidney function, future longitudinal studies should be done to assess ferritin levels as risk factors for renal involvement in thalassemia.

Keywords：Thalassemia, proteinuria, ferritin

Poster Presentation：Glomerular Diseases
Poster No.：C0460
Abstract Submission No.：APCN20250315

Frasier syndrome in the practice of a pediatric nephrologist in Kazakhstan: from diagnosis to treatment. A clinical case.

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Abstract

Case Study:

Introduction: Frasier syndrome is a rare inherited diseases characterized by steroid resistant nephrotic syndrome, which leads to renal failure, gonadal tumor: most commonly gonadoblastoma or dysgerminoma, and pseudohermaphroditism: female external genitalia with sex chromosomes XY. Which is commonly caused by a splice site mutation of Wilms tumor-suppressor gene 1 (WT1).

Case Report:

Patient N. is a 3 years old female presenting with a sore throat, swelling of the eyelids, headaches, oliguria, hypercholesterolemia, hypoproteinemia, hypoalbuminemia, proteinuria 13.2g/l. Treatment: Prednisone 35 mg/day - 6 weeks + 40 mg/day for another 2 weeks + 3 pulse injections of Solu-Medrol, proteinuria remains 3.3g/l – 0.99g/l and steroid resistance is established. Nephrobiopsy result: FSGS (segmental vascular loop sclerosis – 75%, focal sclerosis – 25%). Cyclosporine was added to therapy, after a year of cyclosporine therapy: proteinuria 7.2g/day – 1.55g/day, urea 13.40mmol/L, GFR 69.67ml/min. Considering cyclosporine resistance, a genetic analysis was performed, the result: Gene WT1 Chr11:g.32413513C>T, ENST00000332351.3:c1432+5G>A female external genitalia with chromosomes XY. At the age of 9 the terminal stage of CKD was reached and peritoneal dialysis is performed for renal replacement therapy. For poetic reasons gonadectomy was performed at the age of 9 years. The result of histopathology showed gonadoblastoma of the right gonad. Streak testis with foci of undifferentiated gonadal tissue. Discussed by oncologists, given the biopsy result no chemotherapy was prescribed.

Conclusion:

There are two patients with Fraser syndrome in Kazakhstan. The first patient was diagnosed with end-stage CKD on the background of nephrotic syndrome, steriodresistant variant. Receiving renal replacement therapy: peritoneal dialysis. During the examination, a mass was found in the pelvis, and a Pfannenstiel laparotomy was performed. Adnexectomy on the right + polychemotherapy. As the result, an algorithm has been developed for the diagnosis of nephrotic syndrom, multi-resistant variant, followed by determination of the karyotype and a molecular genetic study. In the second patient, the result of early detection of Frasier syndrome and gonadectomy was achieved and no chemotherapy was prescribed. Patient was achieved and receives peritoneal dialysis. Put on the waiting list for kidney transplantation from cadaverous donation.

Keywords：Frasier syndrome, nephrotic syndrome, gonadoblastoma , pseudohermaphroditism, gonadectomy

Poster Presentation：Glomerular Diseases
Poster No.：C0461
Abstract Submission No.：APCN20250316

"RESULT" Trial to Simultaneously Evaluate Three Immunologic Investigational Therapeutics in Primary Focal Segmental Glomerulosclerosis/Minimal Change Disease

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Abstract

Background:

Primary focal segmental glomerulosclerosis (FSGS) and minimal change disease (MCD) continuum is a major cause of nephrotic syndrome among adult and pediatric patients. There is a lack of safe and effective therapies for FSGS/MCD, which is considered to be immune-mediated. We describe the protocol and operational feasibility for the Renal Efficacy Signaling UmbrelLa Trial (RESULT).

Methods:

RESULT (NCT06500702) is an innovative global Phase 2a randomized, placebo-controlled umbrella trial that simultaneously evaluates the safety and efficacy of 3 novel investigational therapies targeting immunological pathways implicated in primary FSGS/MCD: an anti-CD40L monoclonal antibody, an anti-OX40L and anti-TNF bispecific, and a BTK-inhibitor. Patients 16-75 years with biopsy-confirmed primary FSGS/MCD, eGFR 45 mL/min/1.73 m2 and UPCR 3 g/g will enter a 12-week double blind period followed by a 12-week open-label extension. Primary endpoint is UPCR reduction from baseline to Week 12 for each investigational medicinal product vs pooled placebo. Operational feasibility has been conducted in 132 study sites across 23 countries.

Results:

Majority (83%) of Investigator responders expressed high interest in the trial, with top reasons being scientific importance (62%) and medical interest for patients (67%). Overall, 79% viewed burden of study procedures as appropriate, and 87% perceived remote study visits as safe. Interviews conducted with 11 affected adults, adolescents, and caregivers confirmed the high burden of disease, need for better therapies, and suitability of trial protocol.

Conclusions:

This global RESULT Phase 2a trial utilizes an innovative efficacy signal-seeking master protocol to simultaneously evaluate 3 immunologically active investigational therapies in FSGS/MCD and is the first umbrella trial in nephrology. Operational feasibility and stakeholder feedback demonstrate high scientific and medical interest, as well as appropriateness of trial design and assessments. The study is currently recruiting (https://clinicaltrials.gov/study/NCT06500702).

Funding Sources/Commercial Support:
Sanofi

Presenter: Ethen Cheng, on behalf of the RESULT study group

Keywords：Primary focal segmental glomerulosclerosis, Minimal change disease, Nephrotic syndrome, clinical trial

Poster Presentation：Glomerular Diseases
Poster No.：C0462
Abstract Submission No.：APCN20250317

Hansen's Disease Associated with Mesangioproliferative IgA Nephropathy: A Case Report

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Abstract

Introduction

Leprosy, a chronic bacterial illness, can cause renal involvement in various forms, but mesangioproliferative IgA nephropathy is not commonly recognized because of leprosy. This is not due to the mycobacterium but an immune complex interaction during active infection and erythema nodosum leprosum episodes. Anti-leprotic drugs can also contribute to renal injury.

Case report

An 18-year-old male with a history of leprosy presented with body swelling, shortness of breath, and weakness, along with decreased urine output. He completed 18 months of multidrug therapy (MDT), including dapsone, rifampicin, and clofazimine, following Nepal's national leprosy guidelines. He also had multiple nodules on the ears (Figure 1).

Urine analysis showed nephrotic range albuminuria, a progressive increase in creatinine, and elevated urea with positive antinuclear antibody (ANA) results.

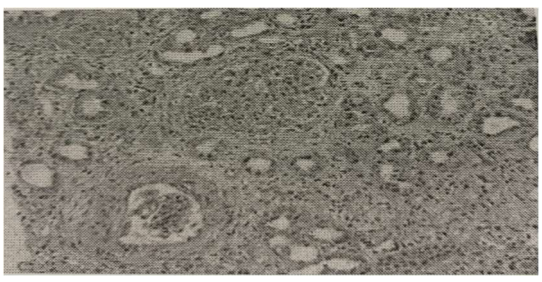
The skin slit smear showed multibacillary lesions. The kidney histopathology showed mesangioproliferative IgA nephropathy (Figure 2). Diffuse immunofluorescence (DIF) showed IgA (3+) with negative IgG and IgM, C3 (3+) granular positivity over mesangium, negative C1q, kappa and lambda light chain (2+) granular positivity over mesangium. Transthoracic echocardiogram (TTE) revealed heart failure with mid-range ejection fraction (HFmrEF), with an ejection fraction of 45-50% and Grade III left ventricular diastolic dysfunction (LVDD).

An arteriovenous fistula (AVF) creation was planned for hemodialysis access. His treatment included sodium bicarbonate, ranitidine, calcium acetate, vitamin D, prazopressin, carvedilol, nifedipine, diltiazem, iron, folic acid, methylcobalamin, dapsone, rifampicin, and clofazimine, along with steroids and maintenance hemodialysis. His skin lesions responded well to treatment, but he continued to be dialysis dependent.

Conclusion

The widespread use of renal biopsy has disclosed an ever-increasing prevalence of glomerulonephritis in patients with leprosy. In summary, this case highlights the complex presentation of a young patient with leprosy, complicated by renal involvement leading to nephropathy. Prompt diagnosis and management of infectious and renal complications are crucial in improving patient outcomes.

Keywords： Glomerulonephritis, Lepsrosy, Mesangioproliferative IgA nephropathy



Poster Presentation：Glomerular Diseases
Poster No.：C0463
Abstract Submission No.：APCN20250324

The correlation between PLA2R-Ab levels and clinical characteristics of primary membranous nephropathy, and the exploration of its application value in treatment selection

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Abstract

Objective：This study aimed to retrospectively analyze the clinical and pathological differences among primary membranous nephropathy (PMN) patients with varying PLA2R-Ab titers, and explore the relationship between PLA2R-Ab titers and disease activity. It also sought to investigate the differences in initial treatment efficacy and adverse reactions among PMN patients, and identify the influencing factors for effective remission.

Method：PMN patients who underwent definitive diagnosis by renal puncture biopsy at the First Affiliated Hospital of Xinjiang Medical University from July 2015 to January 2024 were selected. Based on PLA2R-Ab titer levels, they were divided into three groups: PLA2R-Ab-negative, low-titre, and high-titre. Subsequently, patients with regular follow-up for at least 12 months were further screened and categorized into five treatment groups, namely hormone+CTX, hormone+TAC, hormone+CsA, RTX, and supportive therapy groups. Intergroup data were compared, and treatment efficacy rates at 3, 6, 9, and 12 months were analyzed. Multifactorial COX regression was employed to determine independent risk factors.

Result：Significant differences were observed in total cholesterol, albumin, serum IgG, serum κ and λ light chains, 24h urine protein quantification, and tubulointerstitial damage degree among the three groups. Lymphocyte counts differed significantly between the negative and high-titre groups. At 1-year follow-up, the negative group had a higher effective remission rate. Hormone + CTX and hormone + TAC groups showed superior 1-year efficacy. Adverse reaction incidences were comparable among groups. Cox analysis indicated that baseline PLA2R-Ab positivity, severity of baseline 24h urine protein, and comorbid diabetes predicted 1-year effective remission.

Conclusion：Serum PLA2R-Ab status was associated with proteinuria severity and tubulointerstitial damage. PLA2R-Ab-negative patients had a higher remission rate. Baseline PLA2R-Ab positivity, 24h urine protein severity, and diabetes were independent risk factors for 1-year effective remission in PMN patients.

Keywords：PLA2R-Ab，primary membranous nephropathy

Poster Presentation：Glomerular Diseases
Poster No.：C0464
Abstract Submission No.：APCN20250905

CREBBP Gene Mutation in a Child with Steroid-Resistant Nephrotic Syndrome: A Rare Association

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Abstract
Introduction:
Steroid-resistant nephrotic syndrome (SRNS) is a heterogeneous disease with multiple genetic associations. Mutations in the CREBBP gene, typically linked to Rubinstein-Taybi syndrome (RTS), have not been widely reported in isolated SRNS cases. We present a rare case of SRNS in a child found to have a CREBBP gene mutation without classical RTS features.

Method:
A 2-year-old male presented with nephrotic syndrome at 1 year of age. He was born full-term via normal vaginal delivery to non-consanguineous parents, with no family history of similar illness. Clinical evaluation revealed no hypertension, normal serum creatinine, and bland urine sediment. Notably, there were no skeletal abnormalities, dysmorphic features, or cognitive delays. Initial treatment with oral prednisolone (2 mg/kg/day) failed to induce remission after 6 weeks, classifying him as SRNS. A kidney biopsy showed mesangioproliferative glomerulonephritis without immune deposits. The child was treated with cyclosporine-A (5 mg/kg/day) and enalapril (up to 0.5 mg/kg/day), with steroid tapering. However, no clinical improvement was observed after 6 months of therapy.

Results:
Genetic testing, performed due to the early onset of disease, identified a heterozygous missense mutation in the CREBBP gene on chromosome 16. This mutation is typically associated with RTS, a syndrome characterized by distinctive facial features, broad thumbs and toes, growth deficiency, and intellectual disability—none of which were present in this child.

Conclusion:
This case highlights a novel association between CREBBP gene mutation and SRNS in the absence of classical RTS features. The role of CREBBP in podocyte function and glomerular integrity warrants further investigation. Genetic testing in early-onset SRNS remains crucial for diagnosis and management, even in the absence of syndromic features.

Keywords：Nephrotic syndrome, CREBBP Gene, Rubinstein-Taybi syndrome

Poster Presentation：Glomerular Diseases
Poster No.：C0466
Abstract Submission No.：APCN20250337

A Case Report of Mild Mesangial Proliferative Glomerulonephritis Complicated by Lower Limb Arterial Thrombosis

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Abstract
Objective: This case report examines the clinical features and management challenges of mild mesangial proliferative glomerulonephritis complicated by recurrent right lower limb arterial thrombosis, culminating in amputation. The findings aim to improve recognition of arterial thrombosis in nephrotic syndrome (NS) with this pathological subtype and mitigate severe treatment complications.

Methods: We present a case of mild mesangial proliferative glomerulonephritis with two episodes of right lower limb arterial thrombosis leading to amputation. Clinical, imaging, and histopathological data were analyzed and contextualized with existing literature.

Results: A 33-year-old male developed NS in July 2023 (ALB 23.5 g/L, urine protein 30.24 g/24h, TG 5.68 mmol/L, LDL-C 4.32 mmol/L). Renal biopsy confirmed mild mesangial proliferation; electron microscopy demonstrated diffuse foot process effacement and microvillous transformation without basement membrane thickening, consistent with podocytopathy. Initial therapy included steroids and indobufen. By August 2023, albumin improved to 43.9 g/L, though proteinuria persisted (3+). After indobufen discontinuation in September 2023, acute right lower limb arterial thrombosis occurred (ALB 23.6 g/L), successfully treated with thrombolysis. NS recurred in July 2024 (ALB 19.8 g/L), followed by another arterial thrombosis requiring amputation on August 1, 2024 after failed conservative management.

Conclusion: Thromboembolism in NS most frequently involves renal veins (20%–45%), deep veins (25%), or pulmonary arteries (15%–20%). Arterial thrombosis is rare (1.8%–5.0%), predominantly affecting males at single sites. Mild mesangial proliferative glomerulonephritis seldom accompanies arterial thrombosis. This case reinforces the literature: thrombosis in NS may arise irrespective of histological subtype or disease stage.
Nephrotic Syndrome；Arterial Thrombosi；Mild Mesangial Proliferative Glomerulonephritis；Antiplatelet Therapy；Amputation

Keywords：Nephrotic Syndrome；Arterial Thrombosi；Mild Mesangial Proliferative Glomerulonephritis； Antiplatelet Therapy；Amputation

Poster Presentation：Glomerular Diseases
Poster No.：C0467
Abstract Submission No.：APCN20250343

Clinicopathological Features of MPGN-like IgA Nephropathy

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Abstract
Introduction:
IgA nephropathy is known to show a variety of histological patterns. Among these, only a small proportion of cases present a membranoproliferative glomerulonephritis (MPGN)-like histological pattern. We aimed to elucidate the clinicopathological characteristics of MPGN-like IgA nephropathy.

Methods:
A total of 735 patients diagnosed with IgA nephropathy at the Nagasaki University Hospital and its affiliated facilities between 1996 and 2014, with available electron microscopy findings, were included.

Results:
Twenty-nine cases (3.9%) were identified as MPGN-like IgA nephropathy. Of these, nine cases were associated with liver disease, and two cases had a history of infections. The remaining 18 cases had no notable underlying conditions. Compared to non-MPGN cases, patients with MPGN-like IgA nephropathy were older, had lower serum total protein and albumin levels, and showed higher levels of proteinuria. No significant differences were observed in complement levels (C3, C4). According to the Oxford classification, the rate of E1 was higher, and S1 was lower in patients with MPGN-like IgA nephropathy. Electron microscopy findings of MPGN-like IgA nephropathy were electron-dense deposits in both subepithelial and subendothelial regions, along with mesangial interposition. In four cases, a repeat biopsy was performed: three maintained the MPGN-like pattern, but one case changed to a mesangial proliferative glomerulonephritis pattern with steroid therapy.

Conclusion:
A portion of MPGN-like IgA nephropathy cases were associated with liver disease, suggesting the possibility of secondary forms. However, no notable background factors were identified in the majority of cases. The pathogenesis in these cases may differ from that of typical IgA nephropathy, and detailed history-taking and clinical follow-up are essential for elucidating the underlying causes.

Keywords：IgA nephropathy, Membranoproliferative glomerulonephritis, Liver disease

Poster Presentation：Glomerular Diseases
Poster No.：C0468
Abstract Submission No.：APCN20250344

Real-world Study: Efficacy and Safety of Telitacicept in IgA Nephropathy without Hormones or immunosuppressants

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Abstract
Objective
Telitacicept, a dual APRIL/BLyS inhibitor, demonstrates a significant proteinuria lowering effect in IgAN patients. To assess the efficacy and safety of telitacicept in patients with IgA nephropathy.
Method
In this study, 14 biopsy proven IgAN patients were recruited, with proteinuria excretion more than 2g/d. Patients treated with 160mg telitacicept weekly for at least 6 months with optimized supportive therapy (ACEI/ARB). We collected the changes of urine protein quantification, urine red blood cell , serum creatinine, eGFR, serum albumin, total cholesterol and other indicators were analyzed.
Results
At 4 weeks of treatment with telitacicept，the average value of 24-hour urine protein quantification decreased from 4.27±2.63g/d at baseline to 2.61±1.92 (P=0.04). At 24 weeks of treatment, the mean value of proteinuria decreased to 1.28±0.75 (P=0.002).After 4 weeks of telitacicept treatment, the average serum creatinine decreased from 104.53±35.20umol/L at baseline to 99.29±33.01 (P=0.92)，and the mean serum creatinine decreased to 98.07±30.37 (P=0.63) at 24 weeks. The average eGFR of patients increased from 74.42±28.77 at baseline to 79.45±31.56 (P=0.70) at 4 weeks, while the mean eGFR of patients increased to 77.11±24.62 (P=0.68) at 24 weeks of treatment. After 4 weeks of telitacicept treatment, the average serum albumin level increased from 38.92±5.03 g/L at baseline to 43.25±4.40 (P=0.01). And at 24 weeks of treatment, the average serum albumin of patients increased to 47.29±8.17 (P=0.002). The average value of urine red blood cells decreased from 57.11±101.18/HPF at baseline to 20.74±31.83 (p=0.01) at 4 weeks, and the mean value of urine red blood cells decreased to 5.53±7.33 (P=0.002) at 24 weeks of telitacicept treatment. The average cholesterol level of the patients decreased from 5.26±1.34mmol/L at baseline to 5.57±1.73 (P=0.59) at 4 weeks of treatment. While at 24 weeks of treatment, the mean value of urine red blood cells decreased to 4.69±0.81 (P=0.11).In terms of safety, none of the 14 patients had adverse reactions such as infection, allergic reactions at the injection site during treatment with telitacicept. All IgA patients were not treated with corticosteroids or immunosuppressants during the treatment of telitacicept. During the 24-week follow-up, no patient had a recurrence of the disease.
Conclusion
For patients with IgA nephropathy, telitacicept can reduce urine protein, urine red blood cell production, and increase body albumin, effectively improve kidney function. It has a relatively high safety profile, providing a new treatment option for patients with IgA nephropathy.

Keywords：IgA nephropathy , telitacicept, efficacy, safety

Poster Presentation：Glomerular Diseases
Poster No.：C0469
Abstract Submission No.：APCN20250352

Multi-targeted therapy concluding Nefecon, Mycophenolate Mofetil and Ambrisentan for IgA nephropathy: a case report

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Abstract

IgA nephropathy is the most common glomerulonephritis across the world, and effective treatment strategies are still being explored. This case report describes a patient with IgA nephropathy who received multi-targeted therapy consisting of Nefecon, Mycophenolate Mofetil (MMF), and Ambrisentan. The treatment regimen was carefully designed based on the patient's condition and the potential mechanisms of each drug. During the treatment course, the patient's clinical symptoms gradually improved, with a significant reduction in proteinuria and a stabilization of renal function. No severe adverse reactions were observed, and the patient showed good compliance with the treatment. This case demonstrates the potential effectiveness and safety of the multi-targeted therapy combining Nefecon, MMF, and Ambrisentan for IgA nephropathy. Although it is only a single case report, it provides valuable insights and may inspire further clinical research and exploration of new treatment strategies for IgA nephropathy.

Keywords：IgA nephropathy; Nefecon; Mycophenolate Mofetil; Ambrisentan; multi-targeted therapy

Poster Presentation：Glomerular Diseases
Poster No.：C0470
Abstract Submission No.：APCN20250357

Nefecon for IgA Nephropathy with Tonsillar lesions: A Case Report

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Abstract

Introduction

IgA nephropathy (IgAN), which is characterized by IgA deposition in the glomerular mesangium, progresses to end-stage renal disease in up to 50% of patients within 20 years, imposing a substantial disease burden[1]. Studies have shown that galactose-deficient IgA1 (Gd-IgA1) plays an important role in the occurrence and development of IgAN. Although previous studies have implicated the tonsillar mucosal immune system in the pathogenesis of IgAN[2], recent evidence suggests that gut-associated lymphoid tissue, particularly Peyer's patches in the terminal ileum, serves as the primary source of Gd-IgA1 [3-5]. However, under conditions of pathogen infection or immune activation, activated B cells from tonsillar mucosa-associated lymphoid tissue can migrate via efferent lymphatic vessels into the systemic circulation and subsequently translocate to the gastrointestinal mucosa[6], which not only indicating the existence of a shared mucosal immune system between the tonsils and the gut but highlighting the interrelationship between their mucosal membranes. Nefecon is a novel targeted-release formulation of the locally acting corticosteroid budesonide, designed to suppress Gd-IgA1 production in the distal ileal lymphoid tissue. Nevertheless, whether nefecon also has a therapeutic effect on IgAN patients with tonsillar lesions is still limited.

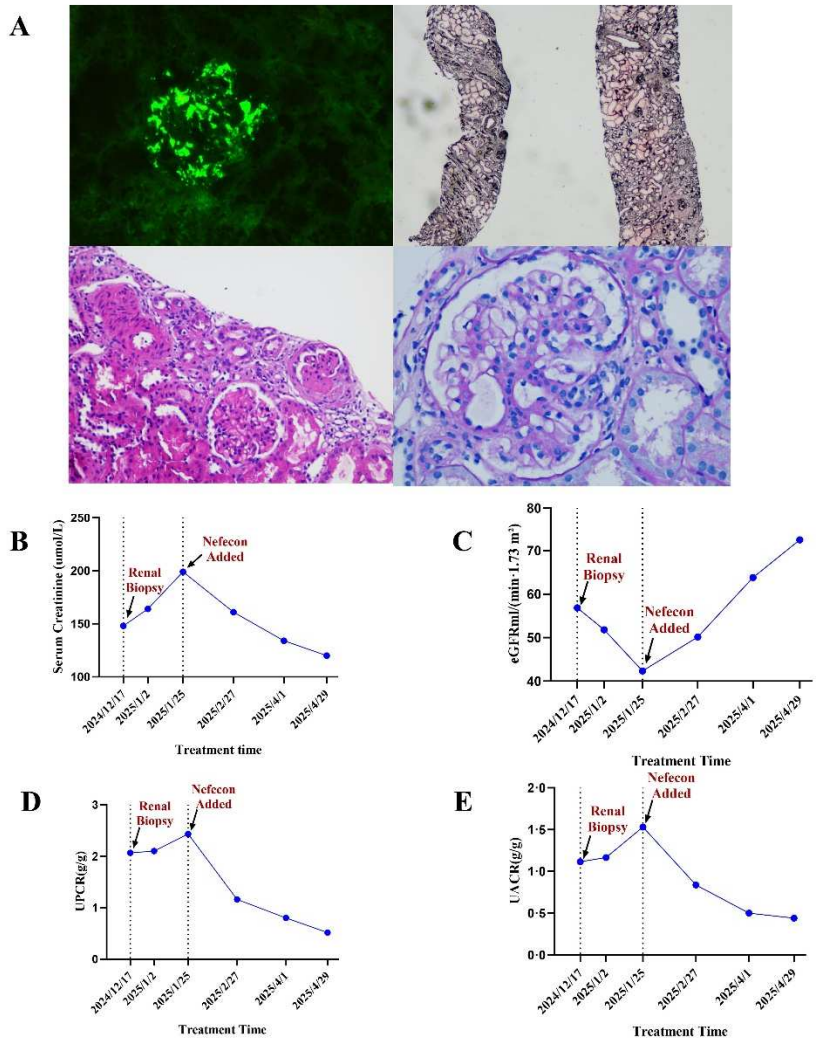
Case Description

A case of young male patient with IgAN and recurrent chronic tonsillitis was treated with nefecon. At baseline, he exhibited significant proteinuria (UPCR 2.433 g/g; UACR 1.53 g/g), hematuria (urinary red blood cells 27500/μL), and reduced renal function (eGFR nadir 42.35 mL/min/1.73m²). Renal biopsy confirmed IgAN (proliferative sclerosis type; Oxford classification M1E0S1T3C0) with 62.9% glomerulosclerosis (Fig. 1A). Initial treatment with ACEI, hydroxychloroquine and leflunomide for one month was ineffective. Therapy was then escalated to nefecon combined with standard-dose hydroxychloroquine and mycophenolate mofetil. After 3 months, his hematuria disappeared. Renal function improved significantly. eGFR increased by 71.1% to 72.48 mL/min/1.73m². Proteinuria markedly decreased, with UPCR falling 78.8% to 0.516 g/g and UACR declining 71.2% to 0.441 g/g (Fig. 1B-1E). These results suggest nefecon may reduce proteinuria and improve eGFR in IgAN patients with tonsillar involvement and advanced glomerulosclerosis.

Discussion

In this report, we present a case of young male IgAN patient with recurrent chronic tonsillitis who achieved long and stable remission following nefecon treatment. To our knowledge, this is the first report of nefecon use in IgAN patients with concomitant tonsillar lesions. This case may provide evidence for a safe, targeted, and effective therapeutic option for IgAN patients with tonsillar lesions, potentially offering an alternative to tonsillectomy and thereby reducing patient burden and associated costs.

Keywords：IgA nephropathy; Nefecon; Tonsillar lesions; Shared mucosal immune system



Poster Presentation：Glomerular Diseases
Poster No.：C0471
Abstract Submission No.：APCN20250379

Association Between Age-adjusted Global Glomerulosclerosis With Progression Of Kidney Disease In Patients With Idiopathic Membranous Nephropathy

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Abstract

Introduction

Idiopathic membranous nephropathy is the major cause of nephrotic syndrome in older adults. This study aims to identify the association between age-adjusted global glomerulosclerosis and kidney disease progression and treatment response in patients with idiopathic membranous nephropathy.

Methods

This retrospective study included 220 patients diagnosed with idiopathic membranous nephropathy between 2015 and 2023. Patients were categorized into three groups based on the age-related thresholds of global glomerulosclerosis: no glomerulosclerosis, glomerulosclerosis normal for age, glomerulosclerosis abnormal for age. Kidney disease progression was defined as a ≥30% decline in estimated glomerular filtration rate (eGFR) from baseline. Complete and partial remission were assessed based on the degree of proteinuria reduction and normalization of serum albumin levels.

Results

The median follow-up duration was 31 months (interquartile range 13–58). Kidney disease progression occurred in 60 individuals (27.3%), while 41 (18.6%) and 153 (69.5%) achieved complete and overall remission at 6 months, respectively. In multivariable analysis, global glomerulosclerosis abnormal for age was independently associated with an increased risk of kidney disease progression compared with no global glomerulosclerosis (hazard ratio 3.01; 95% CI 1.52–5.98; $P < 0.01$). However, both global glomerulosclerosis normal for age and global glomerulosclerosis abnormal for age groups showed no significant
association with achieving complete remission at 6 months compared with no global

glomerulosclerosis group in multivariable analysis. Over the 24-month follow-up, eGFR remained significantly lower in the glomerulosclerosis abnormal for age group compared to the other groups ($P < 0.01$). The decline in proteinuria was more marked in the no GSG and GSG normal for age groups than in the GSG abnormal for age group during the initial 6 months ($P = 0.04$), but afterwards, there were no significant differences in proteinuria among the three groups.

Conclusion

This study highlights that age-adjusted global glomerulosclerosis is associated with kidney disease progression but not with treatment response regarding remission in patients with idiopathic membranous nephropathy.

Keywords：Membranous nephropathy, Global glomerulosclerosis, Kidney disease progression, Complete remission

Poster Presentation：Glomerular Diseases
Poster No.：C0472
Abstract Submission No.：APCN20250381

Evaluation of the Combined Use of Nefecon and Hydroxychloroquine in the Management of IgA Nephropathy

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Abstract

Background：IgA nephropathy (IgAN) is an immune-related kidney condition, predominantly affecting those of East Asian heritage. Nefecon, an innovative oral formulation of budesonide, is engineered for targeted release to block the creation of galactose-deficient IgA1, which underlies IgAN. This study presents findings on the efficacy of Nefecon in combination with hydroxychloroquine for the treatment of IgAN in a real-world setting.

Methods: The study included three patients with biopsy-confirmed IgAN. All participants received a treatment regimen consisting of Nefecon combined with hydroxychloroquine. The primary outcome measure was the change in proteinuria from baseline over the follow-up period.

Results: Baseline characteristics of the three patients are detailed in the Figure .The maximum follow-up duration was 10 months.The mean estimated glomerular filtration rate (eGFR) was 50.77 mL/min/1.73 m², and the median urine protein-to-creatinine ratio (PCR) was 0.99. By the end of the 8-month follow-up, the combination therapy resulted in a reduction of median proteinuria to 0.39, representing a 44.9% decrease from baseline. Initially, the eGFR experienced a modest increase to 63.86 ml/min/1.73 m² after three months, followed by a further rise to 85.5 ml/min/1.73 m² at eight months. The eGFR level demonstrated an elevation of 35.5 ml/min/1.73 m² (24%) compared to baseline after six months of combination therapy. No serious adverse events were reported throughout the follow-up period.

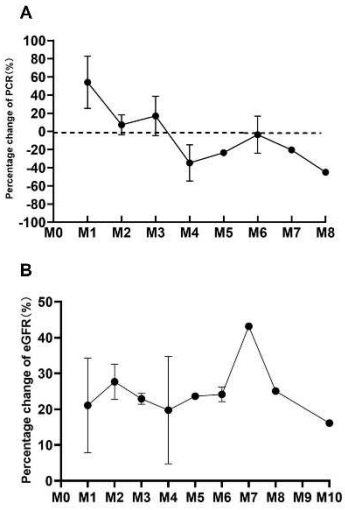
Conclusions: In patients with IgAN, the combination of Nefecon and hydroxychloroquine treatment can decrease proteinuria and has shown a positive safety profile.

Keywords：IgA nephropathy, Nefecon, hydroxychloroquine

tabel1.Baseline Clinical and Pathological Characteristics of IgA Nephropathy Patients at Starting Combination Treatment

Patient No.	Case1	Case2	Case3
follow-up time.M	7	5	10
Age of renal biopsy.Yrs	38	25	39
Age of Nefeccon initiationg.Yrs	38	25	39
Sex	Male	Male	Male
SBP（mmhg）	139	146	136
DBP（mmhg）	93	97	83
eGFR(ml/min/1.73m^2)	47.5	15.27	89.55
Cr(umol/l)	156	430	91.8
PCR(mg/g)	0.54	1.72	0.715
ACR(mg/g)	342.31	1407.69	401.08
Alb(g/l)	43.5	41.1	40.6
UA(unol/l)	642	436	502
Hb(g/l)	133	110	136
OXFORD score	NA	M1E0S1T2C1	M1E0S1T0C1
Initial treatment or recurrence treatment	RT	RT	RT
MRA			
SGLT2i	√		√
Previous GC/IS medication	HCQ	GC+HCQ	NA
Combination therapy with Nefecon	Nefecon+HCQ	Nefecon+HCQ	Nefecon+HCQ

Alb, albumin; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; GC, glucocorticoids; Hb, hemoglobin; IS, immunosuppressants; HCQ,Hydroxychloroquine; MRA, mineralocorticoid receptor antagonist; NA, not available; Oxford-C, crescents; Oxford-E, endocapillary proliferation; Oxford-M, mesangial hypercelluiarity; Oxford-S,segmental glomerulosclerosis; Oxford-T, tubular atrophy and interstitial fibrosis; SBP, systolic blood pressure; SGLT2i, sodium-glucose cotransporter 2 inhibitors; UA, uric acid;



Poster Presentation：Glomerular Diseases
Poster No.：C0473
Abstract Submission No.：APCN20250397

Comparison of the efficacy of rituximab and tacrolimus in high-titer PLA2R antibody-associated membranous nephropathy patients

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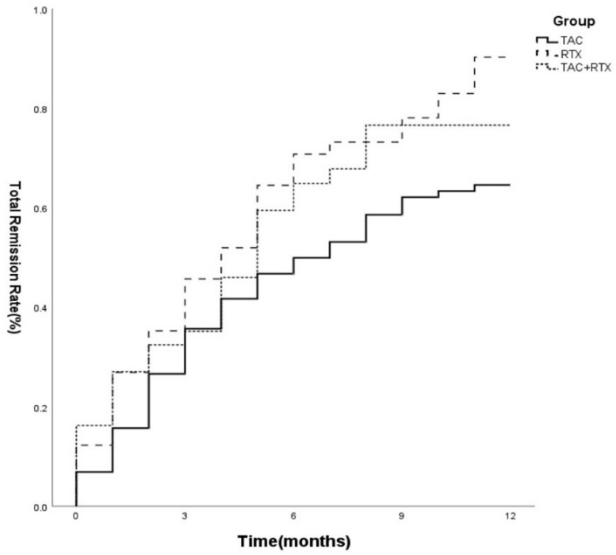
Abstract
Background: This study aimed to analyze the remission rate of rituximab and tacrolimus in patients with high-
titer anti-PLA2R antibodies associated with membranous nephropathy.
Methods: Patients diagnosed with membranous nephropathy by positive anti-PLA2R antibodies and the level
>100 RU/ml at diagnosis, with or without renal biopsy, from January 2018 to April 2023, were enrolled. A total
of 188 patients were enrolled according to different treatment regimens and were divided into the rituximab
group (RTX group, n = 49), the tacrolimus group (TAC group, n = 102), and the rituximab combined with
tacrolimus group (RTX+TAC group, n = 37). The clinical responses of the patients were analyzed.
Results: After 12 months of follow-up, 85.7% of patients in the RTX group, 75.7% of patients in the RTX+TAC group, and 61.8% of patients in the TAC group achieved total remission. During follow-up, 14.7% of patients in the TAC group developed end-stage renal disease, and survival curves showed significant differences in total and
partial remission rates among the three groups (p=0.015)
Conclusion: Rituximab was superior to tacrolimus regarding total remission rate in patients with high-titer
PLA2R antibody. These findings reinforce RTX's position as a first-line therapy for iMN, particularly
advantageous for patients at high risk of disease progression.

Keywords：membranous nephropathy, rituximab, tacrolimus



Poster Presentation：Glomerular Diseases
Poster No.：C0474
Abstract Submission No.：APCN20250399

A case of proliferative glomerulonephritis with monoclonal IgG deposits (PGNMID) with crescent formation that responded favorably to low-dose daratumumab

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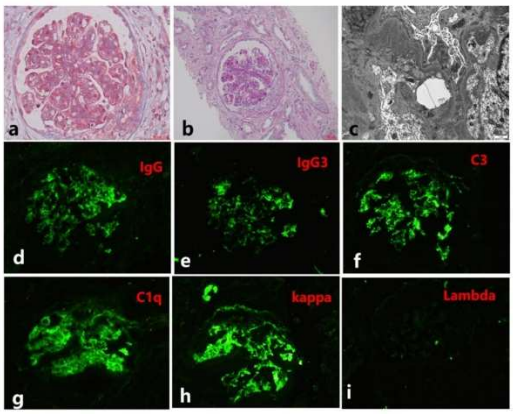
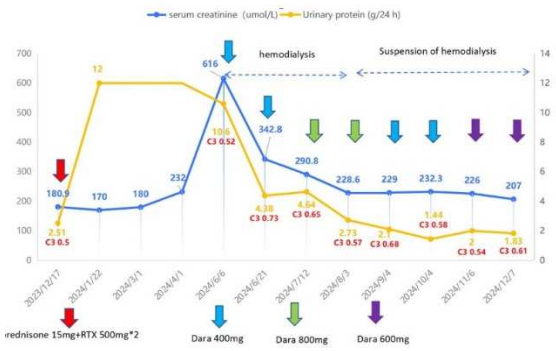
Abstract
Introduction: Proliferative glomerulonephritis with monoclonal immunoglobulin deposits (PGNMID) is a rare renal disease without standardized treatment modalities. Daratumumab is a human IgG monoclonal anti-CD38 antibody that has been demonstrated to be highly effective and safe in treating PGNMID.
Case Description: The patient is a middle-aged woman whose first kidney biopsy suggested proliferative glomerulonephritis. Despite receiving treatment with glucocorticoids, glucocorticoids combined with immunosuppressants, and rituximab, her condition continued to progress. Upon a second kidney biopsy, she was diagnosed with proliferative glomerulonephritis associated with monoclonal immunoglobulin deposition (IgG3 κ). The patient received a total dose of 400-800 mg of daratumumab every two weeks. After the first dose of daratumumab, her 24-hour proteinuria decreased from 12g to 4.38g, and serum creatinine levels dropped from 616umol/L to 342.8umol/L. Two months later, she was able to discontinue hemodialysis. After six months of follow-up, the kidney disease achieved complete remission, with no serious adverse reaction.
Discussion: In conclusion, this case underlines the potential of low-dose daratumumab as a viable option for PGNMID, even in severe cases presenting as rapidly progressive glomerulonephritis.

Keywords：PGNMID, Daratumumab





Poster Presentation：Glomerular Diseases
Poster No.：C0475
Abstract Submission No.：APCN20250406

MAJESTY: A Phase III, Randomized, Open-Label, Active Comparator–Controlled, Multicenter Study Evaluating the Efficacy and Safety of Obinutuzumab in Patients With Primary Membranous Nephropathy

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Abstract

Introduction

The MENTOR study demonstrated that rituximab was superior to cyclosporine in maintaining long-term remission in primary membranous nephropathy (pMN). Obinutuzumab, a type II anti-CD20 monoclonal antibody, depletes B cells more effectively than rituximab. The MAJESTY study (NCT04629248) assesses the efficacy and safety of obinutuzumab versus tacrolimus among patients with pMN.

Methods

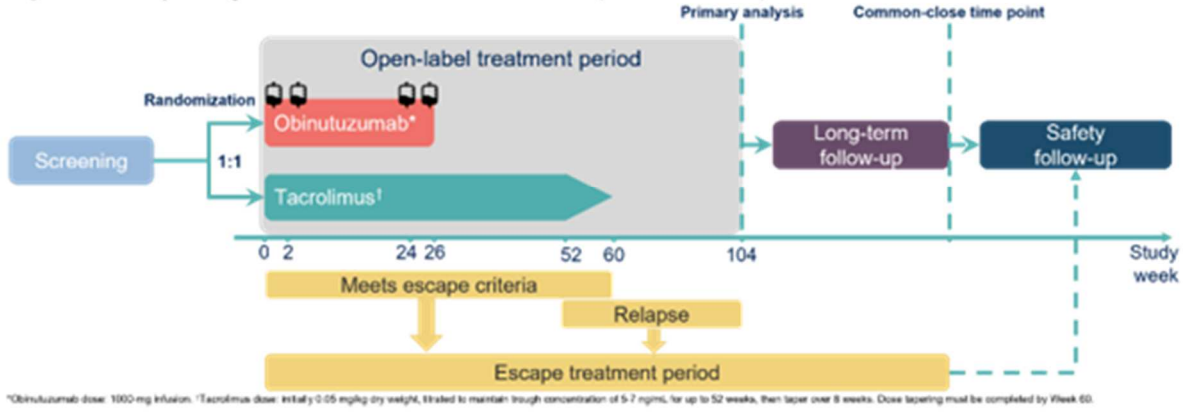
Participants were randomized 1:1 to receive obinutuzumab or tacrolimus (Figure 1). Obinutuzumab (1000 mg) is dosed on Day 1 and Weeks (Wks) 2, 24, and 26; tacrolimus (0.5 mg/kg) is given twice daily. Participants enter escape treatment with obinutuzumab if any of the following criteria are met: 1) Wk 24 urine protein-to-creatinine ratio (UPCR) >3.5 g/g and <25% decrease from baseline; 2) Wk 52 UPCR>3.5 g/g and <50% decrease from baseline; 3) Wk 52-104 UPCR >3.5 g/g after previously achieving proteinuric complete or partial remission (CR/PR); 4) tacrolimus discontinuation criteria. The primary endpoint is the proportion of patients with CR, defined as UPCR ≤0.3 g/g with a stable estimated glomerular filtration rate (eGFR) at Wk 104. Secondary endpoints include the proportion of participants who achieve CR or PR (UPCR >0.3 and ≤3.5 g/g and ≥50% decrease from baseline) at Wk 104 and the proportion who achieve CR at Wk 76.

Results
MAJESTY is fully enrolled with 142 participants across 86 sites and 11 countries. The study population comprises 31% women, and 25% Asian, 3% Black and 70% White participants; 29% received prior immunosuppressive therapy. At baseline, mean (SD) age was 51.2 (11.1) years and albumin was 2.9 (0.6) g/dL; median (IQR) eGFR was 87.0 (62.5-104.5) mL/min/1.73 m2 and 24 hours UPCR was 6.8 (5.2-9.0) g/g. Among 110 (78%) participants with anti-PLA2R antibody >14 RU/mL, median (IQR) titer was 147 (52-287) RU/mL.

Conclusion
Full results of the MAJESTY study will be available upon completion.

Keywords：Membranous nephropathy, clinical trial

Figure 1. Study Design of the Phase III MAJESTY trial (NCT04629248)



Poster Presentation：Glomerular Diseases
Poster No.：C0477
Abstract Submission No.：APCN20250421

Treatment Results of IgA Nephropathy And Some Factors Affecting Treatment Response

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Abstract
Background: Treatment of patients with IgA nephropathy remains difficult due to lack of consensus in treatment regimen, especially immunosuppressants.
Objectives: to evaluate the treatment results of patients with IgA nephropathy after 6, 12 months and investigate some factors affecting the treatment results.
Subjects and method: descriptive, prospective study; 62 IgA nephropathy patients in Thong Nhat hospital, HCM city, from October 2012 to December 2024.
Inclusion criteria: patients ≥18 years old, were diagnosed IgA nephropathy based on kidney biopsy.
Exclusion criteria: patients that lost of follow up treatment, a kidney biopsy sample with <6 glomeruli.
Results: men were 52.4%, the mean age was 39.7±15.7, mean eGFR was 58.9±35.1ml/min/1.73m2. Complete remission, partial remission and non-response after 6 months was 35.7%, 33.3% and 31%, respectively; after 12 months was 45.9%, 20.8% and 33.3%, respectively.
After 6 months, 100% patients with eGFR ≥60ml/min/1.73m2 had remission, 43.5% patients with eGFR <60ml/min/1.73m2 had remission ($p<0.001$); 82.8% patients with proteinuria <3.5g/24h had remission, 38.5% patients with proteinuria ≥3.5g/24h had remission ($p<0.009$). Patients with S1, T1, T2 histologic patterns of IgA nephropathy had poor response to treatment than the others.
After 12 months, patients with eGFR ≥60ml/min/1.73m2, proteinuria <3.5g/24h had better response to treatment than the others ($p<0.05$).
Conclusion: the response rate to treatment of IgA nephropathy patients after 6 months and 12 months were 69% and 66.7%, respectively. Factors related to treatment response include eGFR<60ml/min/1.73m2, proteinuria ≥3.5g/24h at the time of diagnosis and histopathological lesions S1, T1, and T2.

Keywords：IgA nephropathy, treatment, related factors

Poster Presentation：Glomerular Diseases
Poster No.：C0478
Abstract Submission No.：APCN20250425

Ranking New Therapies for IgA Nephropathy: A Network Meta-Analysis of Efficacy and Safety

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Abstract
Objectives: Immunoglobulin A nephropathy (IgAN) is a leading cause of progressive renal dysfunction, necessitating targeted therapies to improve outcomes. While multiple novel treatments have emerged, direct comparative data is limited. This systematic review and network meta-analysis evaluates the relative efficacy and safety of new IgAN therapies to guide evidence-based clinical decision-making.

Methods: A systematic literature search was conducted in PubMed, Embase, and Cochrane Central for randomized controlled trials (RCTs) published in the past 12 years, evaluating the efficacy of novel IgAN therapies. A Bayesian network meta-analysis compared treatment effects on urine protein-to-creatinine ratio (UPCR) reduction and estimated glomerular filtration rate (eGFR) preservation, using mean differences (MD) and Surface Under the Cumulative Ranking (SUCRA) scores to assess relative effectiveness.

Results: A total of 12 RCTs involving 912 IgAN patients were included. For UPCR reduction, Mycophenolate (SUCRA: 0.761) and Atacicept (SUCRA: 0.731) ranked highest, demonstrating significant proteinuria reduction [MD −0.62, 95% CrI: −1.8, 0.52] and [MD −0.55, 95% CrI: −1.5, 0.40], respectively. Hydroxychloroquine followed (SUCRA: 0.701), while Budesonide (SUCRA: 0.532) and Iptacopan (SUCRA: 0.478) showed more modest effects.For eGFR preservation, Telitacicept (SUCRA: 0.748) (MD 8.5; 95% CrI −6.2, 21.5) and Mycophenolate (SUCRA: 0.726) (MD 7.9; 95% CrI −7.1, 22.0) ranked highest, followed by Budesonide (SUCRA: 0.693) (MD 6.3; 95% CrI −1.0, 14.8). Atacicept (SUCRA: 0.545) (MD 4.2; 95% CrI −6.8, 16.5) showed moderate effectiveness, while Hydroxychloroquine, Iptacopan, and Fluticasone ranked lowest, with placebo demonstrating minimal benefit.

Conclusions: This network meta-analysis suggests that Mycophenolate and Telitacicept provide the most favorable outcomes in proteinuria reduction and eGFR preservation among IgAN therapies. Given the variability in treatment responses, further head-to-head trials are warranted to refine personalized treatment strategies for IgAN patients.

Keywords：IgA Nephropathy, Therapeutic Benefit, Ranking Efficacy

Poster Presentation：Glomerular Diseases
Poster No.：C0479
Abstract Submission No.：APCN20250480

Successful Treatment of Steroid-Dependent Mesangial Proliferative Glomerulonephritis with Telitacicept: A Case Report

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Abstract

Introduction: To evaluate the efficacy of telitacicept combined with corticosteroid therapy for mesangial proliferative glomerulonephritis (MPGN).

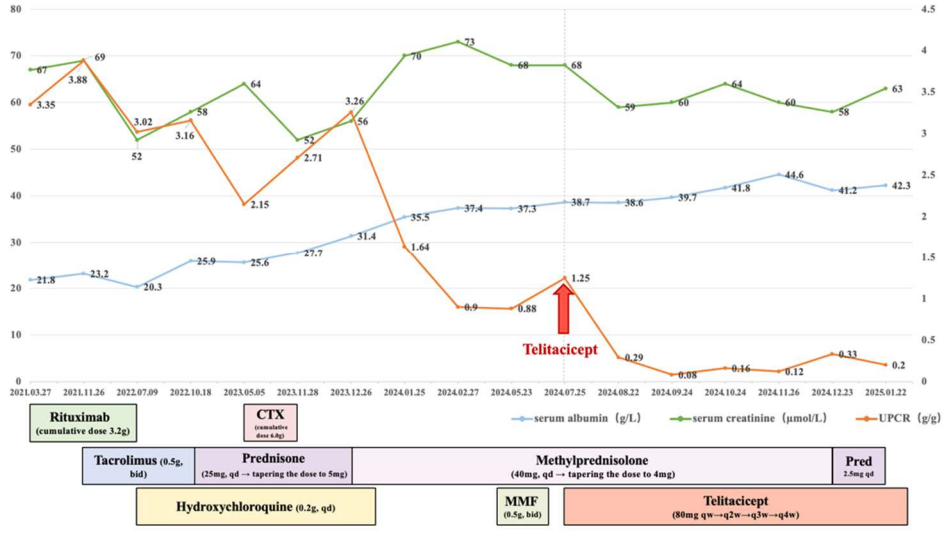
Method: We report a case of a 46-year-old male presenting with nephrotic syndrome. In January 2021, his urine protein-to-creatinine ratio (UPCR) was 5.49 g/g and serum albumin was 26.3g/L. Renal biopsy confirmed MPGN with diffuse mesangial electron-dense deposits and 35% foot process fusion. Sequential immunosuppressive therapies including rituximab(cumulative dose 3.2g), tacrolimus, cyclophosphamide (CTX) (cumulative dose 6.0g) and mycophenolate mofetil(MMF) showed limited efficacy. Full-dose methylprednisolone reduced proteinuria (the lowest UPCR 0.88 g/g) but tapering steroids resulted in proteinuria rebound (UPCR 1.25 g/g), indicating steroid dependence. Additionally, prelonged steroid use led to steroid-induce diabetes. Innovative combination therapy with telitacicept 80 mg weekly and methylprednisolone 20 mg daily was initiated in July 2024. Serial monitoring of proteinuria, serological markers, immunological profiles, and adverse events was performed.

Results: After eight weeks of treatment, the patient's UPCR dropped to 0.08 g/g. Although IgG levels decreased at the initial treatment, they remained within the relatively safe range after dose adjustment. Currently, glucocorticoid was successfully discontinued and the telitacicept dose is subsequently reduced to 80 mg every 4 weeks. The combined therapy achieved to a rapid and sustained remission, with UPCR remaining at a low level. Importantly, no serious infections or disease relapses occurred during glucocorticoid tapering.

Conclusion: This is the first report of telitacicept combined with glucocorticoid treatment for steroid-dependent MPGN, effectively alleviating clinical symptoms , reducing proteinuria and enabling a rapid glucocorticoid taper with minimal adverse effects. These results highlight telitacicept as a promising treatment option for steroid-dependent MPGN, warranting further randomized trials.

Keywords：mesangial proliferative glomerulonephritis, telitacicept

Figure 1 The treatment process received by the patient and changes in indicators



Poster Presentation：Glomerular Diseases
Poster No.：C0480
Abstract Submission No.：APCN20250481

Clinical Efficacy of Telitacicept in the Treatment of IgA Nephropathy complicated by Diabetes Mellitus: A Case Report

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Abstract
Introduction: Immunoglobulin A nephropathy (IgAN) is one of the most prevalent primary glomerulonephritis globally, characterized by mesangial IgA deposition and driven by immune dysregulation. Traditional therapies involving glucocorticoids with or without immunosuppressive increase the risk of infection, osteoporosis, and glucose metabolism disturbances. Telitacicept, a novel biologic targeting both B lymphocyte stimulator(BLyS) and a proliferation-inducing ligand(APRIL), may mitigate disease progression by suppressing pathogenic IgA1 production. This case report from Xiangya Hospital of Central South University demonstrates the potential efficacy of telitacicept monotherapy in managing IgAN with comorbid diabetes mellitus (DM), a population at high risk for steroid-related complications.
Method: We observed a 49-year-old male with poorly controlled DM(diagnosed over 10 years, complicated by diabetic retinopathy and peripheral neuropathy) presented with massive proteinuria, with the 24-hour urinary protein excretion (24-h UPE) of 9 g. The initial clinical diagnosis was diabetic nephropathy. Initial treatment with renin-angiotensin system inhibitors (allisartan isoproxil), sodium-glucose co-transporter 2 inhibitor (dapagliflozin) and nonsteroidal mineralocorticoid antagonist (finerenone) failed to reduce proteinuria (24-h UPE escalated to 29 g). Renal biopsy confirmed the diagnosis as IgAN (Lee's classification: Grade III; Oxford classification: M1E1S1T0C1). To avoid glucocorticoid-induced hyperglycemia, telitacicept monotherapy was initiated at 240 mg subcutaneously weekly, later tapered to 160 mg weekly. Proteinuria, serum albumin, and safety markers were monitored monthly.
Results: After 9 months of telitacicept treatment, the patient's 24-h UPE decreased from 29 g to 0.75 g, and serum albumin normalized from 24.1 g/L to 42.4 g/L. Immunoglobulin G levels remained within normal limits, and no infections, cytopenias, or glycemic exacerbations were observed.
Conclusion: This case highlights telitacicept's dual advantages in IgAN with DM: significant proteinuria reduction without compromising glycemic control or increasing infection risk, offering a promising therapeutic option for such patients. Further randomized trials are warranted to validate its role as a steroid-sparing agent in this high-risk population.
Keywords：Immunoglobulin A nephropathy, telitacicept

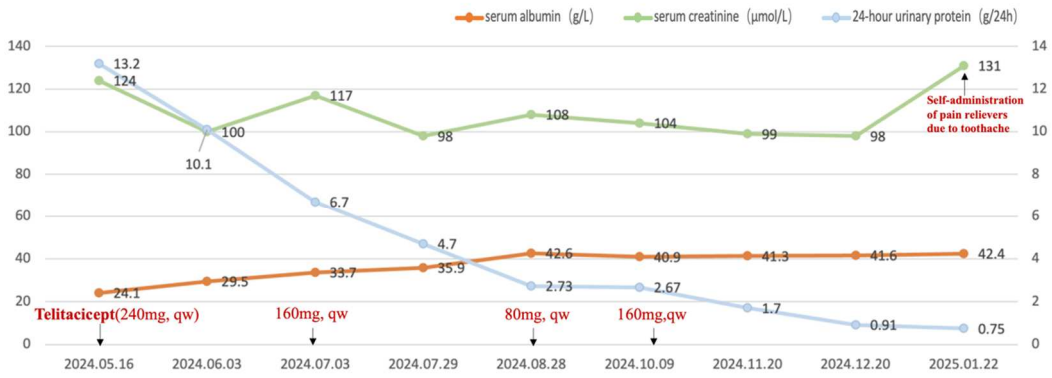

Figure 2 Changes in serum albumin , creatinine and 24-hour urinary protein during treatment

Poster Presentation：Glomerular Diseases
Poster No.：C0481
Abstract Submission No.：APCN20250487

A Rare Coexistence of C3 Glomerulopathy And Systemic Lupus Erythematosus In A Young Female

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Abstract
Introduction:
C3 glomerulopathy (C3G) is a rare kidney disease caused by complement cascade dysregulation. Systemic lupus erythematosus (SLE) is an autoimmune disease with multiorgan involvement. Very few reports exist in literature about the co-existence of both diseases, with no reported case yet in the Philippines. This report explores the clinical presentation and therapeutic considerations of a patient diagnosed with both diseases.

Methods:
We report an unusual case of a 29-year-old woman diagnosed with SLE by fulfillment of the 2019 EULAR/ACR and 2012 SLICC criteria, and with C3G through renal biopsy and immunofluorescence. Furthermore, we review the available literature published from January 2010 to March 2025 on the clinical features and management of C3G in the setting of SLE.

Results:
In addition to our case, very few reports exist in literature regarding the fortuitous association of SLE in association with C3G. The coexistence of both diseases is a rare clinical scenario, posing therapeutic challenges especially in low-income countries. There is no established optimal treatment for C3G, and while newer studies show that eculizumab suggests benefit for both diseases, it was not available locally. Similar to some of the previous reports, our patient was treated with methylprednisolone pulse therapy, hydroxychloroquine, mycophenolate mofetil, and enalapril, which resulted in low disease activity. There was improvement in proteinuria and stable creatinine during follow-up.

Conclusion:
This rare case of SLE and C3G emphasizes the need for personalized treatment strategies, especially in resource-limited settings. It highlights the importance of understanding the underlying mechanisms of these conditions and the need for ongoing research to improve patient management and outcomes in these unique cases.

Disclaimer: This abstract was also submitted for the Asia-Pacific League of Associations for Rheumatology (APLAR) congress. By submitting the abstract to APCN x TSN 2025, abstract authors declare that re-submitting the abstract is permitted by the organizers of the previous meeting."

Keywords：C3G, SLE, immunology

Poster Presentation：Glomerular Diseases
Poster No.：C0482
Abstract Submission No.：APCN20250834

Histological Pattern of Glomerulonephritis (GN) in Sylhet: A Single Centre Study

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Abstract

Introduction:
Glomerulonephritis (GN) encompasses a wide spectrum of renal pathologies with varying clinical presentations and histological patterns. Regional data is essential to understand the local disease burden and guide management. This study aims to identify the histological patterns of GN in patients undergoing renal biopsy in a tertiary center in Sylhet.

Methods:
This observational study was conducted using retrospective data collected from medical records. A total of 52 patients who underwent renal biopsy between 1st July 2023 and 31st December 2024 at a tertiary center in Sylhet were included. Patients aged between 11 and 70 years were studied. Demographic data, biopsy results, and renal function (serum creatinine) were analyzed.

Results:
Among 52 patients, 25% were male (13) and 75% female (39). The most frequent histological diagnosis was Lupus Nephritis Class IV (27%, 14 patients), followed by Focal Segmental Glomerulosclerosis (FSGS) (23%, 12 patients), Membranous Nephropathy (13.5%, 7), Lupus Nephritis Class III (5.8%, 3), and Class II (3.8%, 2). Other findings included Diffuse Membranoproliferative GN (7.7%, 4), IgA Nephropathy (5.8%, 3), Immune Complex-Mediated GN (3.8%, 2), Crescentic GN (5.8%, 3), Minimal Change Disease (1.9%, 1), and Renal Amyloidosis (1.9%, 1). 13.5% (7 patients) had elevated serum creatinine, including cases of Amyloid Kidney (1.9%), FSGS (1.9%), Immune Complex GN (1.9%), IgA Nephropathy (1.9%), and Crescentic GN (5.8%, 3 patients).

Conclusion:
Lupus nephritis, particularly Class IV, was the most prevalent GN pattern in this study, with a female predominance. FSGS and membranous nephropathy were also common. Notably, a significant proportion of patients exhibited elevated serum creatinine, particularly those with Crescentic GN, indicating advanced renal dysfunction. The high prevalence of Lupus Nephritis underscores the need for targeted screening and early intervention in this region. These findings provide crucial local epidemiological data, emphasizing the importance of renal biopsy in guiding diagnosis and management. Further large-scale studies are recommended to explore etiological factors, optimize treatment strategies, and improve renal outcomes this region.

Keywords：Glomerulonephritis (GN), Focal Segmental Glomerulosclerosis (FSGS)

Poster Presentation：Glomerular Diseases
Poster No.：C0483
Abstract Submission No.：APCN20250498

Early Endothelial Dysfunction in Lupus Nephritis: A Cross-Sectional Evaluation Using Flow-Mediated Dilation and Carotid Intima-Media Thickness

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Abstract
Background:
Systemic lupus erythematosus (SLE) is associated with accelerated atherosclerosis, especially in patients with lupus nephritis (LN). Chronic inflammation, endothelial damage, and renal dysfunction synergistically increase cardiovascular risk. However, conventional risk calculators often fail to detect early vascular damage in young SLE patients. This study aimed to evaluate endothelial function and structural vascular changes in SLE patients with and without nephritis using flow-mediated dilation (FMD) and carotid intima-media thickness (CIMT), and to correlate these findings with disease activity and renal markers.
Methods:
This single-center cross-sectional study enrolled 100 adult SLE patients (95% female, mean age 29.88 ± 6.53 years), fulfilling ACR 1997 classification criteria and having disease duration ≥2 years. Fifty had biopsy-confirmed lupus nephritis of ≥6 months. All participants underwent high-resolution ultrasound to measure CIMT and FMD (via brachial artery occlusion and release). Clinical data including SLE Disease Activity Index (SLEDAI), disease duration, albuminuria, and standard renal function markers were recorded.
Results:
CIMT positively correlated with age ($\rho = 0.209$, $p = 0.037$), while FMD was inversely correlated ($\rho = -0.252$, $p = 0.011$). Patients ≥25 years had significantly more deranged CIMT and FMD ($p < 0.05$). Compared to non-nephritis patients, those with LN had significantly impaired FMD and higher SLEDAI scores ($p < 0.05$), though CIMT did not differ significantly. In LN patients, age positively correlated with CIMT ($\rho = 0.441$, $p = 0.001$) and inversely with FMD ($\rho = -0.312$, $p = 0.028$). In non-nephritis patients, FMD inversely correlated with disease duration ($\rho = -0.288$, $p = 0.043$) and albuminuria ($\rho = -0.285$, $p = 0.045$).
Conclusion:
Lupus nephritis patients demonstrate significantly greater endothelial dysfunction despite similar CIMT to non-nephritis SLE patients, suggesting that functional vascular injury precedes structural changes. FMD is a sensitive, non-invasive marker that may serve as a valuable screening tool in nephrology clinics to detect early cardiovascular risk in LN patients. Integrating FMD into routine follow-up may enable timely intervention and improve long-term cardiovascular and renal outcomes.

Keywords：Lupus Nephritis,Endothelial Dysfunction,Cardiovascular Risk

Parameter	Non-Nephritis (Mean ± SD)	Nephritis (Mean ± SD)	p-value
FMD (%)	8.2 ± 1.9	5.9 ± 2.1	< 0.05
CIMT (mm)	0.52 ± 0.07	0.54 ± 0.08	NS
SLEDAI Score	4.1 ± 1.2	7.6 ± 2.4	< 0.01
Age (years)	28.4 ± 5.7	31.1 ± 6.2	< 0.05
Disease Duration (years)	3.4 ± 1.1	3.7 ± 1.3	NS
Urine Albumin (mg/dL)	15 ± 8	45 ± 20	< 0.05

Poster Presentation：Glomerular Diseases
Poster No.：C0484
Abstract Submission No.： APCN20250500

Thymoma-Related Glomerular Diseases: Clinical Observations from a Case Series of Eight Patients from Two Medical Centers in Taiwan

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Abstract

Background:
Thymoma is a rare neoplasm frequently associated with autoimmune disorders, including glomerulopathies. However, the clinicopathologic spectrum and outcomes of thymoma-associated glomerular diseases remain poorly defined.

Methods:
We conducted a retrospective case series of eight patients diagnosed with glomerulopathy in the context of thymoma at two tertiary centers in Taiwan over the past two decades. Clinical presentation, renal pathology, thymoma staging, treatment, and outcomes were analyzed.

Results:
The cohort (5 females, 3 males; mean age 59.7 years) exhibited a diverse range of renal pathologies, including minimal change disease (MCD; n = 3), membranous nephropathy (n = 2, including one with anti-THSD7A positivity), FSGS (n = 2), and lupus nephritis (n = 1). Five patients developed glomerulopathy after thymectomy, while three were diagnosed parathymically. Six patients had co-existing myasthenia gravis. Despite immunosuppressive therapy or thymectomy, most patients showed poor renal response, and five progressed to end-stage kidney disease (ESKD). Three patients died within three years after renal diagnosis, while two were lost to follow-up.

Conclusion:
Thymoma-associated glomerulopathies are heterogeneous in pathology and clinical course. Post-thymectomy
onset does not preclude autoimmune renal involvement. Early recognition and multidisciplinary cares are essential, although prognosis remains guarded.

Keywords：Thymoma, Glomerulopathy, Paraneoplastic syndrome

Poster Presentation：Glomerular Diseases
Poster No.：C0485
Abstract Submission No.：APCN20250501

Clinical Prognosis and Outcome Determinants in Anti-Neutrophil Cytoplasmic Antibodies (ANCA)-Associated Pauci-Immune Crescentic Glomerulonephritis: Insights from a Single-Center Study in Taiwan

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Abstract

Background:

Anti-neutrophil cytoplasmic antibody (ANCA)-associated pauci-immune crescentic glomerulonephritis (PICGN) exhibits with variable renal outcomes. This study aims to assess the clinical characteristics, treatment modalities, and prognostic factors affecting renal survival and dialysis independence in patients treated at Chang Gung Memorial Hospital (CGMH), a tertiary medical center in Taiwan.

Methods:

We retrospectively analyzed 65 patients diagnosed with ANCA-associated PICGN, categorized into microscopic polyangiitis (MPA), granulomatosis with polyangiitis (GPA), and renal-limited vasculitis (RLV). Clinical parameters, treatment regimens including plasmapheresis, and histopathological classifications were evaluated. Kaplan-Meier survival analysis and log-rank tests were used to assess renal and overall survival, stratified by initial dialysis requirement, creatinine levels, and histological class.

Results:

The majority of patients were MPO-ANCA positive (81.5%), with a mean serum creatinine of 7.73 ±4.16 mg/dL at presentation. Initial dialysis was required in 58.5% of patients, with a significantly lower rate of successful dialysis withdrawal observed in the MPA subgroup ($p = 0.011$). Plasmapheresis did not significantly improve renal survival in patients with initial creatinine >3.4 mg/dL ($p = 0.899$) or >5.7 mg/dL ($p = 0.965$), nor in those requiring dialysis at presentation ($p = 0.134$). The need for dialysis within the first month was associated with a significantly worse renal outcome (log-rank $p < 0.001$). Histopathological classification was not a significant predictor of renal survival (log-rank $p = 0.168$).

Conclusion:

In this CGMH cohort, early dialysis requirement within one month was a strong predictor of poor renal prognosis in ANCA-associated PICGN. Plasmapheresis did not confer survival benefits across stratified patient groups. Although histopathological classification was not a significant prognostic factor, its potential value warrants further investigation. These findings underscore the importance of early diagnosis and timely intervention in improving renal outcomes.

Keywords：Anti-neutrophil cytoplasmic antibodies, vasculitis, crescentic glomerulonephritis, RPGN

Poster Presentation：Glomerular Diseases
Poster No.：C0486
Abstract Submission No.：APCN20250529

The Set Up of a Rare Kidney Disease Service in Shenzhen - Early Report from a Single Centre

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Abstract

Introduction

Rare kidney diseases have been largely neglected until the past few years. With increased understanding of the pathomechanism of these rare diseases, availability of more investigations including genetic tests, and availability of orphan drugs, renal rare diseases have gradually emerged and began to get recognition globally and in China. Our Hospital in Shenzhen is a fully accredited 3A comprehensive hospital with an annual outpatients of 1.6 million. In 2023, our Hospital was awarded a rare disease grant to 1) set up local rare disease registry; 2) provide training; 3) offer multi-disciplinary care to needed patients; and 4) act as a hub to collaborate with other institutions in clinical research. We have reported some of our early results in this report.

Methods

The Chinese National Health Commission produced its first list of 121 rare diseases in 2018, and a further 86 diseases in its second list in 2024. About 10 of them are linked to renal disorders. The list has been set up in our Hospital IT and all clinicians are encouraged to register their patients in the Hospital IT system from January 2023. Rare diseases related to the kidneys include atypical HUS, Alport's, Erdheim Chester Disease, Fabry, Gitelman, IgG4 related disease, POEMS Syndrome, ANCA vasculitis and Von Hippel-Lindau. The entry of rare renal diseases have been retrieved from the hospital IT and presented in Table 1.

Results

TIO is tumour induced osteomalacia. The 41 year-old man presented with multiple fractures and hypophosphatemia. He was diagnosed to have a phosphaturic mesenchymal tumour with elevated FGF23 eighteen months later.

We also encountered a case of Erdheim-Chester Disease. There was a 20-year interval between the onset of disease and the diagnosis in this 45 year-old female. She developed CKD3-4 and responded to trametinib.

Conclusions

Many rare renal diseases were never diagnosed or misdiagnosed or diagnosed late. Many may lead to end stage kidney disease. Further to dialysis cost, the impact of such diseases on individual and family is extensive. In China, there is a genuine move to provide better care and equity of care for these patients. Shenzhen is the 4th biggest city in China. Therefore, its participation in the Chinese national rare disease collaborative network is crucial.

Acknowledgement

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(2) Sanming Project of Medicine in Shenzhen (No.SZSM202311022)
(3) Translational Medicine Research Center of HKU-Shenzhen Hospital

Keywords：rare kidney disease, tumour induced osteomalacia, Erdheim-Chester Disease

Types of Rare Diseases	No. of cases
Alport's	24
ANCA vasculitis	62
aHUS	5
Erdheim–Chester Disease	1
Fabry	8
IgG4 related disease	10
POEMS syndrome	1
TIO	1
Gitelman	3

Poster Presentation：Glomerular Diseases
Poster No.：C0487
Abstract Submission No.：APCN20250595

Remission and Relapses in lupus nephritis among multi-racial Southeast Asians: a single center retrospective study

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Abstract

Lupus nephritis has a relapsing/remitting course that can progress to kidney failure. This study aims to assess treatment outcomes and factors associated with remission among multi-racial Southeast Asians treated for lupus nephritis.

This was a single-center, retrospective cohort study of adults with biopsy-proven lupus nephritis diagnosed between 2018 and 2020 at Singapore General Hospital. Clinical, laboratory and pharmacotherapy data were collected from electronic health records every 6 months ("visits") for three years from diagnosis. We excluded visits with incomplete data for immunosuppressive therapy and remission status.

Primary outcomes were remission and relapses during follow-up. To account for variability across patients and repeated measures over time, mixed effects logistic regression analyses were used to examine factors associated with remission. The mixed effects model included fixed effects for (1) follow-up time point alone; and (2) follow-up time point and factors listed in Table 1 determined a priori. The sensitivity analysis added factors that were significantly associated with remission after adjustment for follow-up duration.

We included 73 patients with 301 visits. Remission and complete remission occurred in 63.0% and 39.7% respectively at Month 6. Remission rates increased to 82.9% at Month 24 but were lower at Month 36. The complete remission rate increased to 75.0% at Month 30 and was lower at Month 36.

Nephritic relapses occurred in 1.4% at Month 6, increased to 5-7% at Month 12-24, and increased to 11% in the third year of follow-up.

Table 1 shows the clinical and histological characteristics at follow-up. After adjusting for the follow-up duration, known SLE at kidney biopsy, hyperlipidemia, and acute kidney disease at presentation were associated with reduced likelihood of remission, while higher eGFR was associated with higher likelihood of remission.

The mixed effects logistic regression found that follow-up at Month 18 and 24 (OR 3.478 [95% CI: 1.015-11.912] and 8.620 [95% CI: 2.008-36.995] respectively, compared to Month 6), higher eGFR (OR 1.032 [95% CI: 1.012-1.053]), and higher C4 (OR 1.662 [95% CI: 1.011-2.732]) levels were independently associated with remission. Higher C3 (OR 0.043 [95% CI: 0.005-0.364]) levels at kidney biopsy and use of multitarget therapy within 6 months before follow-up (OR 0.172 [95% CI: 0.035-0.838]) were inversely associated with remission. The sensitivity analysis (addition of SLE and hyperlipidemia) revealed SLE was inversely associated with remission (OR 0.127 [95% CI: 0.037-0.435]).

Future studies should further elucidate the relationship between different induction regimens including multitarget therapy and clinical outcomes.

Keywords：Lupus Nephritis

Table 1. Factors associated with remission during follow-up, after adjusting for follow-up time point.

	All N = 301	No remission N = 86	Remission N = 215	OR (95% CI)*	P value
At diagnosis					
Age, years	38.8 (30.9, 52.4)	45.2 (32.2, 52.1)	37.8 (28.8, 52.7)	0.993 (0.955-1.032)	0.72
Male sex, n (%)	64 (21.3)	20 (23.3)	44 (20.5)	0.837 (0.237-2.937)	0.78
Race, n (%)					
- Chinese	232 (77.1)	73 (84.9)	159 (74.0)	1.00 (reference)	-
- Malay	35 (11.6)	9 (10.5)	26 (12.1)	1.548 (0.320-7.485)	0.586
- Indian	10 (3.3)	3 (3.5)	7 (3.3)	0.902 (0.062-12.180)	0.940
- Others	24 (8.0)	1 (1.2)	23 (10.7)	10.869 (0.749-157.765)	0.08
SLE before biopsy, n (%)	175 (58.1)	72 (83.7)	103 (47.9)	0.115 (0.039-0.340)	<0.001
Diabetes mellitus, n (%)	7 (2.3)	7 (8.1)	0	Data imbalance	
Hypertension n (%)	83 (27.6)	34 (39.5)	49 (22.8)	0.372 (0.119-1.158)	0.088
Systolic BP, mmHg	126 (110, 143)	131 (117, 146)	121 (108, 138)	0.974 (0.948-1.001)	0.059
Diastolic BP, mmHg	78 (65, 84)	77 (67, 90)	72 (64, 82)	0.980 (0.945- 1.016)	0.270
Hyperlipidemia, n (%)	57 (18.9)	28 (32.6)	29 (13.5)	0.215 (0.062-0.754)	0.017
BMI, kg/m²	23.8 (20.6, 28.7)	24.0 (20.8, 28.9)	23.9 (20.6, 28.5)	0.956 (0.905-1.009)	0.102
Ischemic heart disease, n (%)	6 (2.0)	1 (1.2)	5 (2.3)	2.833 (0.124-67.195)	0.509
Acute kidney disease, n (%)	106 (35.2)	43 (50.0)	63 (29.3)	0.309 (0.106, 0.902)	0.032
- Rapidly progressive GN, n (%)	48 (15.9)	26 (30.2)	22 (10.2)	0.171 (0.043-0.685)	0.013
- Required KRT, n (%)	12 (4.0)	3 (3.5)	9 (4.2)	1.255 (0.061-25.798)	0.883
Serum creatinine, µmol/L	74 (59, 137)	99 (65, 193)	70 (57, 103)	0.990 (0.982-0.997)	0.009
eGFR, ml/min/1.73 m²	107.0 (74.4, 122.7)	88.6 (38.4, 115.6)	108.7 (91.9, 124.2)	1.023 (0.008-1.038)	0.003
Serum albumin, g/L	30 (24, 35)	30 (27, 36)	30 (24, 35)	0.959 (0.890- 1.034)	0.275
UPCR, g/g	3.28 (1.51, 7.47)	3.43 (1.65, 8.24)	2.56 (1.35, 5.93)	1.025 (0.964-1.089)	0.430
Hematuria present, n (%)	281 (93.4)	75 (87.2)	206 (95.8)	3.937 (0.650- 23.831)	0.135
Urine red blood cells, number per hpf	28 (5, 138)	14 (32, 78)	33 (8, 175)	1.001 (1.00, 1.002)	0.141
Complement C3	0.54 (0.46, 0.66)	0.62 (0.48, 0.92)	0.54 (0.45, 0.64)	0.824 (0.367-1.846)	0.636
Complement C4	0.08 (0.05, 0.14)	0.15 (0.07, 0.21)	0.08 (0.04, 0.10)	1.094 (0.831-1.439)	0.641
Histology					
- Class V, n (%)	11 (3.7)	5 (5.8)	6 (2.8)	0.318 (0.029-3.491)	0.347
- Crescents, n (%)	13 (4.3)	2 (2.3)	11 (5.1)	2.067 (0.151-28.339)	0.586
- Focal segmental glomerulosclerosis, n (%)	91 (30.2)	35 (40.7)	56 (26.0)	0.457 (0.150-1.393)	0.168
- Hypertensive arteriolosclerosis, n (%)	32 (10.6)	14 (16.3)	18 (8.4)	0.395 (0.073-2.126)	0.278
- Thrombotic microangiopathy, n (%)	24 (8.0)	12 (14.0)	12 (5.6)	0.210 (0.026-1.668)	0.139
Within 6 months before follow-up					
RAS inhibitors, n (%)	195 (64.8)	55 (64.0)	140 (65.1)	0.806 (0.329-1.974)	0.636
Immunosuppressants, n (%)	301 (100)	86 (100)	215 (100)		
- Prednisolone, n (%)	291 (96.7)	84 (97.7)	207 (96.3)	0.945 (0.078-11.444)	0.964
- Prednisolone dose, mg/day	9 (5, 26)	10 (8, 30)	7 (5, 20)	1.001 (0.973-1.029)	0.966
- Methylprednisolone, n (%)	36 (12.0)	11 (12.8)	25 (11.6)	1.196 (0.341-4.192)	0.779
- MMF or Myfortic, n (%)	254 (84.4)	64 (74.4)	190 (88.4)	1.968 (0.646-5.999)	0.233
- Cyclophosphamide, n (%)	34 (11.3)	11 (12.8)	23 (10.7)	1.129 (0.357-3.572)	0.836
- Azathioprine, n (%)	22 (7.3)	7 (8.1)	15 (7.0)	1.298 (0.288-5.888)	0.735
- Hydroxychloroquine, n (%)	276 (91.7)	73 (84.9)	203 (94.4)	4.295 (0.776-23.774)	0.095
- Multi-target therapy, n (%)	26 (8.6)	12 (14.0)	14 (6.5)	0.285 (0.066-1.230)	0.092
Serum creatinine, µmol/L	67 (55, 89)	86 (63, 225)	64 (54, 77)	0.979 (0.969-0.989)	<0.001
UPCR, g/g	0.32 (0.12, 1.23)	1.68 (0.92, 4.60)	0.21 (0.10, 0.49)	0.939 (0.886-0.996)	0.035
Statin, n (%)	75 (24.9)	35 (40.7)	40 (18.6)	0.463 (0.180-1.190)	0.110
Recent hospitalization					
- Any, n (%)	47 (15.6)	22 (25.6)	25 (11.6)	0.454 (0.167-1.229)	0.120
- Infection, n (%)	24 (11.2)	11 (15.5)	13 (9.0)	0.749 (0.220-2.548)	0.642

BP, blood pressure; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; RAS, renin-angiotensin system; UPCR, urine protein-to-creatinine ratio.
Categorical variables presented as number (column percentage). Continuous variables presented as median (25th centile, 75th centile).
*Adjusted for follow up time point using mixed effects logistic regression.

Poster Presentation：Glomerular Diseases
Poster No.：C0488
Abstract Submission No.：APCN20250613

Unmasking a Hidden Storm: Fatal Thrombosis in Steroid-Responsive Nephrotic Syndrome Revealing an Occult Myeloproliferative Neoplasm

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Abstract

Background:

Steroid-responsive nephrotic syndrome (NS) is commonly considered a benign, immune-mediated condition with favorable prognosis. However, rare but fatal complications may occur when underlying systemic processes remain undiagnosed. We report a case of fatal thrombotic events during clinical remission of NS, ultimately revealing features suggestive of an occult myeloproliferative neoplasm (MPN).

Case Presentation:

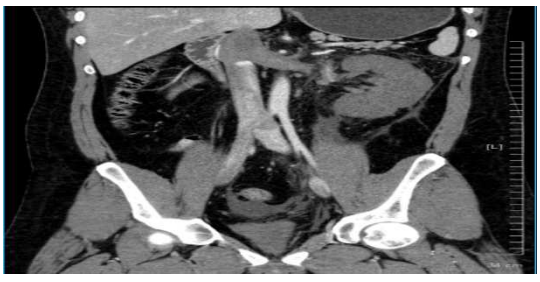
A 31-year-old previously healthy Thai male presented with generalized edema and nephrotic-range proteinuria (UPCI 11 g/g). Initial labs showed normal renal function and unremarkable blood counts. He responded dramatically to corticosteroids, with complete resolution of edema and UPCI reduction to 0.11 within three weeks. Kidney biopsy was deferred due to the rapid and complete response.

Twelve weeks later, during steroid tapering, the patient developed acute abdominal pain and gross hematuria. Notably, there was no recurrence of edema or nephrotic activity, suggesting the thrombotic event occurred during a quiescent phase. CT imaging revealed extensive thrombosis involving the inferior vena cava, bilateral renal veins, iliac veins, and total occlusion of the left renal artery with renal infarction. Repeat CBC showed hemoglobin 21.2 g/dL, hematocrit 65.5%, WBC 30,800/mm³, and platelet count 180,000/μL. These findings raised high concern for a latent myeloproliferative disorder, such as polycythemia vera. Unfortunately, the patient died within 24 hours before JAK2 mutation testing or bone marrow biopsy could be performed.

Conclusion:

This case highlights the critical importance of continuous vigilance even in seemingly well-controlled NS. Unexpected thrombosis and hematologic shifts may reflect a hidden clonal driver rather than a typical steroid course complication. Routine follow-up with attention to evolving blood profiles may offer early clues to life-threatening systemic disease. Clinicians should remain aware that not all immunologic remissions guarantee long-term safety.

Keywords：nephrotic syndrome, thrombosis, polycythemia vera, JAK2 mutation, myeloproliferative neoplasm, kidney infarction



Poster Presentation：Glomerular Diseases
Poster No.：C0489
Abstract Submission No.：APCN20250650

Telitacicept improves renal function in patients with lupus nephritis

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Abstract
Objective: This study aimed to assess the efficacy of Telitacicept in treating lupus nephritis patients with renal impairment while evaluating its impact on renal function.
Methods: A lupus nephritis patient confirmed by renal biopsy received treatment with 160 mg Telitacicept in conjunction with glucocorticoids and mycophenolate mofetil. Lupus-specific markers, 24-hour proteinuria, serum creatinine, and estimated glomerular filtration rate (eGFR) were assessed at baseline, 2 months, and 3 months post-treatment.
Results: The combined therapy of Telitacicept with glucocorticoids promptly decreased proteinuria, maintained stably serum creatinine and eGFR levels,
Conclusion: The combination of Telitacicept with glucocorticoids and other medications can rapidly reduce urinary protein, maintain stable levels of serum creatinine and eGFR, and decrease glucocorticoids dosage.

Keywords：Telitacicept, lupus nephritis, glucocorticoids dosage reduction

Poster Presentation：Glomerular Diseases
Poster No.：C0490
Abstract Submission No.：APCN20250673

A Rare Case of Pediatric Systemic Amyloidosis Secondary to Leprosy and Tuberculosis with Severe Renal Involvement

Widdy Winarta[1]; Eka Laksmi Hidayati[1]; Rizqi Amalia[2]; Sandra Widaty[3]; Meilania Saraswati[4]; Monik Ediana Miranda[4]; Ening Krisnuhoni[4]; Henny Adriani Puspitasari[1]; Reza Fahlevi[1]; Wahyuni Indawati[5]; Oryza Gryagus Prabu[6]; Maruhum Bonar[6]; Himawan Aulia Rahman[7]; Pramita Gayatri[7]; Hanifah Oswari[7]; Sudung O. Pardede[1]

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Abstract

Background:

Systemic amyloidosis in children is rare, particularly in association with chronic infectious diseases. This case highlights a 17-year-old male with systemic amyloidosis and severe renal involvement, likely secondary to relapsing leprosy and intestinal tuberculosis.

Case:

The patient initially presented with chronic diarrhea, oral candidiasis, 15 kg weight loss, and a history of leprosy. Further evaluation led to a diagnosis of relapsing leprosy with first degree disability, erythema nodosum leprosum, intestinal TB (IGRA positive), and severe malnutrition. Initial diagnostic workup included esophagogastroduodenoscopy and colonoscopy. Amyloidosis was suspected due to the presence of amorphous deposits, and Congo red staining confirmed amyloid deposition in tissues from the esophagus to the colon. Following the confirmation of gastrointestinal amyloidosis, the possibility of systemic involvement was considered. Given the presence of persistent nephrotic-range proteinuria and hypoalbuminemia, renal amyloidosis was suspected. A kidney biopsy was subsequently performed, and its findings were consistent with those of the gastrointestinal tract biopsies, confirming the presence of amyloid deposits in the renal glomeruli, vascular walls, and peritubular areas.

The patient was treated with anti-tuberculosis and anti-leprosy medications, high-dose corticosteroids (for leprosy reaction), and ACE inhibitors. Diarrhea improves following initiation of therapy. Inflammatory markers and infection-related indices showed significant improvement over six months: CRP decreased from 88 to 6 mg/L, and both the morphological and bacteriological indices of leprosy improved. Despite these improvements in infectious and inflammatory status, renal

function continue to deteriorate. The patient remained in nephrotic-range proteinuria, and serum creatinine rose progressively from 0.5 to 7.26 mg/dL over six months. Hemoglobin dropped to 7.6 g/dL, and ureum peaked at 261 mg/dL. The patient ultimately required hemodialysis following an episode of acute uremic encephalopathy.
Whole-exome sequencing revealed a heterozygous NPHS1 variant, classified as VUS (variance of unknown significance). Due to local limitations, amyloid typing (e.g., AA vs. AL) and serum amyloid A concentration could not be performed.

Conclusion:
This is a rare pediatric case of systemic amyloidosis with extensive gastrointestinal and renal involvement, likely secondary to chronic infectious disease. Early progression to end-stage kidney disease highlights the importance of timely diagnosis and intervention. There is a pressing need for accessible amyloid typing and guidance on the use of newer therapies such as SGLT-2 inhibitors in pediatric amyloidosis. This case underscores the diagnostic and therapeutic challenges in managing systemic amyloidosis in resource-limited settings and supports the call for international collaboration in such complex cases.

Keywords：Pediatric amyloidosis, nephrotic syndrome, leprosy, tuberculosis, renal failure, congo red

Poster Presentation：Glomerular Diseases
Poster No.：C0491
Abstract Submission No.：APCN20250677

Glomerular hyperfiltration and long-term health outcomes across different populations: a systematic review and meta-analysis.

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Abstract

Background: Glomerular hyperfiltration (GHF), defined as supraphysiological elevation of estimated glomerular filtration rate (eGFR), has been considered as a preclinical indicator of substantial kidney damage. However, the evidence is yet conclusive regarding the prognostic role of GHF.

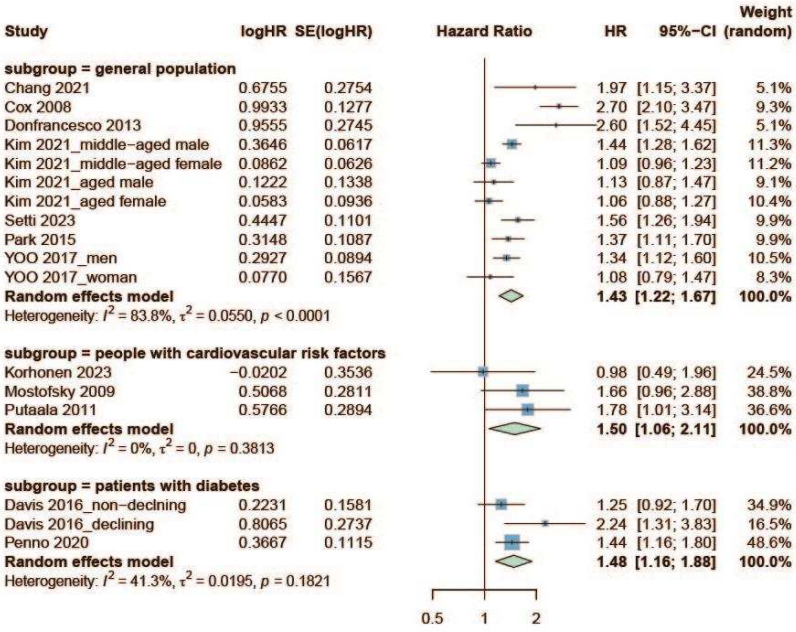
Methods: Databases including Pubmed, Embase and Cochrane library were rigorously searched for cohort studies from the inception until March, 2025. Cross-sectional studies and a public database were employed for estimation of GHF cut-off value. To investigate effect of GHF on long-term health outcomes, mortality, cardiovascular disease (CVD), albuminuria and rapid decline of eGFR were considered as major endpoints.

Results: 30 cohorts were included in systemic review and 24 of them were further allocated for meta-analysis. GHF 95th percentile ranged from 94.7 to 146.7 mL/min/1.73 m2 in sex and age-specific categories. Meta-analysis demonstrated significant association between GHF and mortality in general population [Hazard ratio (HR) 1.43, 95% confidential interval (CI), 1.22 – 1.67], individuals of cardiovascular risks (HR 1.50, 95%CI, 1.06 – 2.11) and diabetic population (HR, 1.48, 95%CI, 1.16 – 1.88). Significant CVD was observed in general population (HR, 1.45, 95%CI, 1.02 – 2.07), while no significance was noted for albuminuria. In diabetic patients, GHF was considered to intensify eGFR decline (HR 3.62, 95%CI, 2.45 – 5.36). Subgroup analysis revealed that study region, confounders and GHF definition might be sources of heterogeneity.

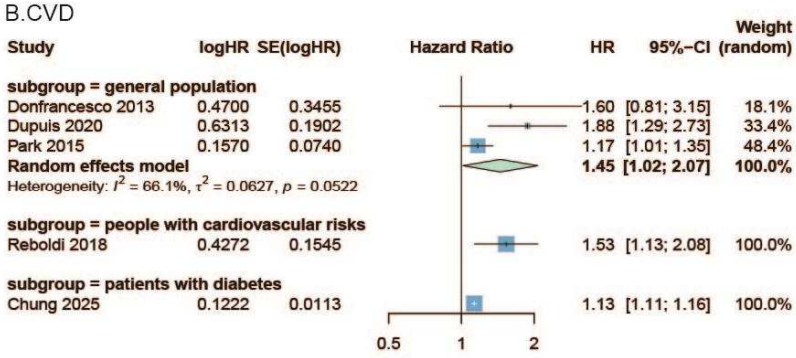
Conclusion: GHF is an independent risk factor for long-term mortality, cardiovascular disease and renal function decline across various populations. Substantial variations in 95th percentile eGFR values across age, sex, and racial groups was observed, highlighting requirement for precise, population-specific diagnostic criteria for GHF.

Keywords：glomerular hyperfiltration, mortality, cardiovascular disease, albuminuria, meta-analysis.

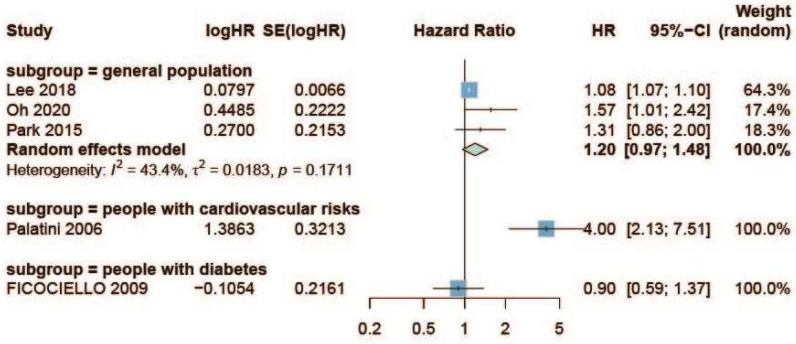
A.Mortality



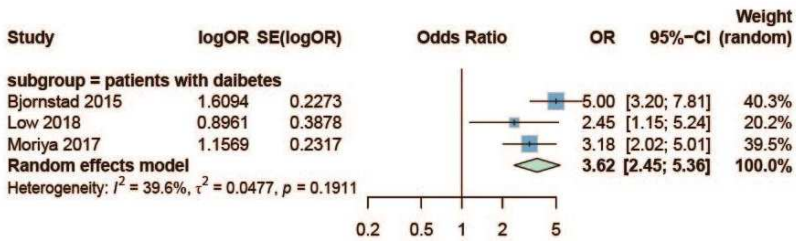
B.CVD



C.Albuminuria



D.Rapid eGFR decline



Poster Presentation：Glomerular Diseases
Poster No.：C0492
Abstract Submission No.：APCN20250680

Renal Thrombotic Microangiopathy Associated with Malignant Hypertension: A Retrospective Study from a Single Center in Taiwan

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Abstract

Background:
Malignant hypertension (MHTN) is a life-threatening condition that can lead to renal thrombotic microangiopathy (TMA), a pathological process that may mimic atypical hemolytic uremic syndrome (aHUS). The clinical features and treatment responses—particularly to plasma exchange (PLEX)—in this setting remain under-characterized.

Methods:
We retrospectively reviewed 18 patients diagnosed with malignant hypertension and biopsy-confirmed renal TMA at a tertiary center. Clinical presentations, laboratory parameters, renal histology, complement profiles, genetic findings, and responses to PLEX and anti-C5 therapy were analyzed. Two representative cases were described in detail.

Results:
All patients presented with severe hypertension (mean BP 224/138 mmHg) and acute kidney injury (mean serum creatinine 7.6 mg/dL). No patients had severe ADAMTS13 deficiency. Kidney biopsy revealed TMA features predominantly involving arterioles and glomeruli. Complement gene variants were identified in three patients.
Among the 11 patients who received PLEX, eight derived clinical benefit: two had marked hematologic and renal recovery; three improved further after subsequent anti-C5 therapy; and three showed partial responses without progression to ESRD. The remaining three patients had no significant response. Among the seven patients who did not receive PLEX, four of them had ESRD later, two of them had chronic kidney disease, and the remaining one patient was lost to follow-up.

Conclusions:
Renal TMA associated with malignant hypertension presents with variable clinical outcomes and may closely resemble complement-mediated aHUS. While PLEX may be beneficial in selected cases, responses are heterogeneous. Early renal biopsy and complement evaluation, with consideration of targeted therapy such as anti-C5 agents, are essential for individualized management.

Keywords：malignant hypertension, thrombotic microangiopathy, plasma exchange

Poster Presentation：Glomerular Diseases
Poster No.：C0493
Abstract Submission No.：APCN20250690

Rapidly Progressive Glomerulonephritis in Lupus Nephritis Complicated by Infective Endocarditis: a Therapeutic Challenge

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Abstract

Introduction:

Rapidly progressive glomerulonephritis in lupus nephritis represents a severe form of renal involvement in systemic lupus erythematosus (SLE). Management often requires aggressive immunosuppression, which increases susceptibility to serious infections, including infective endocarditis (IE). We report a case of a 33-year-old woman with lupus-related rapidly progressive glomerulonephritis who developed infective endocarditis following the initiation of hemodialysis and immunosuppressive therapy.

Case Illustration:

A 33-year-old female with a 4-year history of lupus nephritis presented with progressive dyspnea, abdominal distension, and oliguria. She had been previously treated for biopsy-proven ISN/RPS Class IV + V lupus nephritis with pulse-dose methylprednisolone and cyclophosphamide. At presentation, clinical and laboratory findings were consistent with acute on chronic kidney disease, fluid overload, and refractory hyperkalemia, necessitating supportive hemodialysis.

Physical examination revealed alopecia, pallor, ascites, and bilateral pitting edema in the lower extremities. Laboratory result revealed anemia (Hb 8 g/dL), elevated urea (252 mg/dL) and creatinine (1.7 mg/dL), hyponatremia (132 mmol/L), hyperkalemia (6 mmol/L), hypoalbuminemia (2.1 g/dL), low complement levels (C3:20 mg/dL, C4:4 mg/dL), and elevated anti-dsDNA level (341.2 IU/mL). Blood cultures were initially negative, but repeated cultures and echocardiography revealed large vegetations 60.5 mm × 34.2 mm on the tricuspid valve with severe regurgitation. Chest X-Ray and echocardiography also showed cardiomegaly and pericardial effusion. The patient underwent tricuspid valve repair and evacuation of vegetation and received broad spectrum antibiotics. While early postoperative recovery was initially stable, her condition rapidly deteriorated due to hospital-acquired pneumonia, septic shock, and gastrointestinal bleeding. Despite optimal medical management, the patient succumbed during hospitalization.

Discussion:

RPGN associated with LN requires prompt and aggressive immunosuppression to prevent irreversible kidney damage and progression to end-stage renal disease (ESRD). However, such therapy increases the risk of severe infections. In this case, infective endocarditis (IE) was suspected to be catheter-related and compounded by immunosuppressive therapy. Large valvular vegetations (>10 mm) increase the risk of embolization, and require surgical intervention. This case emphasizes the delicate balance between aggressive immunosuppression and mitigating infection risk, especially in

patients requiring hemodialysis.

Conclusion:
This case highlights the complexity of managing RPGN in the context of immunosuppression and highlights the importance of early detection, cautious immunosuppressive use, prompt antibiotic initiation, and multidisciplinary care to reduce mortality.

Keywords：Lupus Nephritis, Rapidly Progressive Glomerulonephritis, Infective Endocarditis

Figure 1. Echocardiography

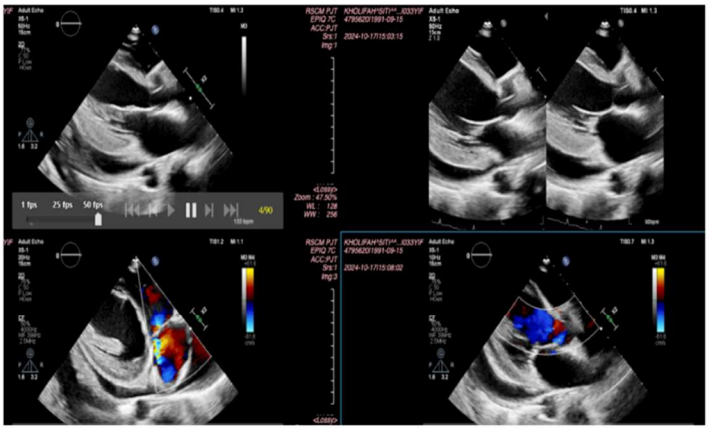
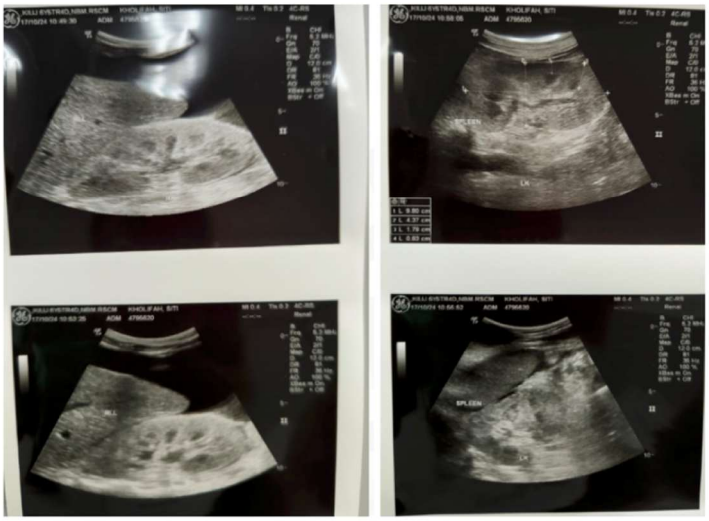


Figure 2.Renal Ultrasound



Poster Presentation：Glomerular Diseases
Poster No.：C0494
Abstract Submission No.：APCN20250712

Nefecon Therapy Mitigates Renal Function Decline and Reduces Proteinuria in Steroid-Resistant IgA Nephropathy: A Case Report

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[2] Alberta Institute,Wenzhou Medical University

Abstract

Introduction: IgA nephropathy (IgAN), the most prevalent form of glomerulonephritis worldwide, is characterized by hematuria, proteinuria, and progressive renal function decline. Novel therapeutic approaches are warranted for patients who experience disease progression despite maximal supportive care and conventional immunosuppression.

Case Description: A 28-year-old female initially presented with bilateral lower limb edema six years ago following an upper respiratory tract infection. Routine urinalysis one year later revealed proteinuria. She subsequently sought medical attention due to worsening edema. Although her presentation suggested nephrotic syndrome, renal biopsy confirmed IgAN, classified as Oxford M1E1S1T0C2 (Fig. 1). Initial therapy comprised high-dose intravenous corticosteroids and tacrolimus, transitioning to a tapering course of oral prednisone. However, upon prednisone dose reduction to 30 mg/day, persistent edema indicated steroid resistance. Consequently, she received three courses of intravenous rituximab over six months combined with oral mycophenolate mofetil (MMF). This regimen significantly improved her edema and reduced the Urinary Protein-to-Creatinine Ratio (UPCR) to <1 g/g. Over the subsequent three years, maintenance therapy included an SGLT2 inhibitor, an angiotensin receptor blocker (ARB), MMF, and hydroxychloroquine. During this period, her estimated Glomerular Filtration Rate (eGFR) gradually declined from 120 to 82.9 mL/min/1.73m², while UPCR fluctuated between 0.18 and 1.43 g/g. In February 2025, nefecon (16 mg/day) was added to her existing regimen without dosage adjustment. After three months, her UPCR decreased to 0.48 g/g, and her eGFR improved to 90.2 mL/min/1.73m² (Fig. 2).

Discussion: This case demonstrates that nefecon therapy may effectively mitigate renal function decline and reduce proteinuria in steroid-resistant IgAN, potentially attributable to its distinct mechanism of action compared to systemic corticosteroids. Immunosuppressive agents can be used concurrently with nefecon in progressive disease. However, the long-term prognostic impact of nefecon requires further investigation.

Keywords：IgA Steroid-Resistant

(Fig.1)

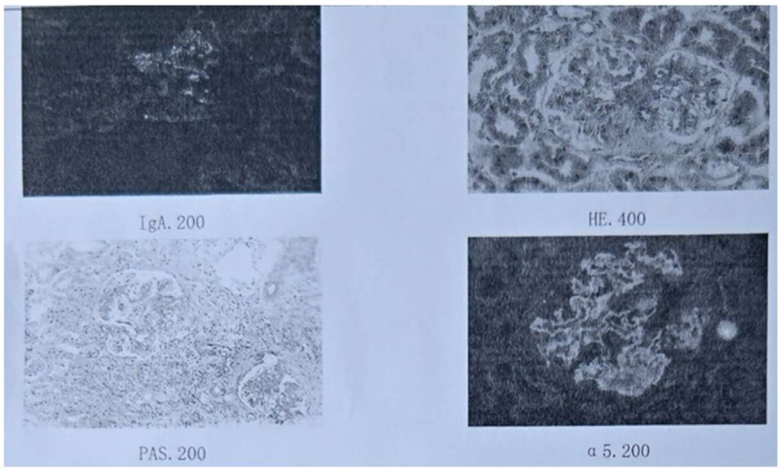
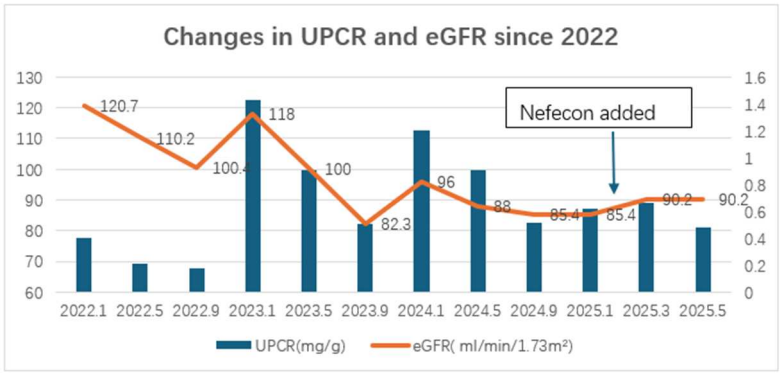

IgA. 200
HE. 400
PAS. 200
α 5. 200

Fig.2


Changes in UPCR and eGFR since 2022
Nefecon added
120.7
110.2
100.4
118
100
82.3
96
88
85.4
85.4
90.2
90.2
2022.1
2022.5
2022.9
2023.1
2023.5
2023.9
2024.1
2024.5
2024.9
2025.1
2025.3
2025.5
UPCR(mg/g)
eGFR(ml/min/1.73m²)

Poster Presentation：Glomerular Diseases
Poster No.：C0495
Abstract Submission No.：APCN20250714

Clinicopathological features of membranous nephropathy with glomerular tip lesions

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Abstract

Introduction:

Membranous nephropathy (MN) is a major cause of nephrotic syndrome in adults. While some patients experience spontaneous remission, others progress to end-stage renal disease. Segmental sclerosis is occasionally observed in MN and has been associated with poor prognosis, although its clinical significance remains unclear. Glomerular tip lesion (GTL), a localized form of segmental sclerosis at the tubular pole of the glomerulus, is typically observed in the tip variant of focal segmental glomerulosclerosis (FSGS) and generally associated with favorable outcomes. Although GTL is also seen in other glomerular diseases including MN, detailed investigations in this context are limited. This study aimed to clarify the clinicopathological features of MN cases with GTL.

Methods:

Among 180 patients diagnosed with MN by renal biopsy at Nippon Medical School between 2010 and 2020, six cases with GTL were identified. Their clinical and pathological characteristics were retrospectively analyzed.

Results:

All six patients were male, aged 43 to 70 years, with serum creatinine levels ranging from 0.94 to 1.29 mg/dL, indicating mildly reduced renal function. All presented with nephrotic-range proteinuria and hematuria. Phospholipase A2 receptor staining was positive in three cases, and none had autoimmune diseases or malignancies, consistent with primary MN. Urinary protein selectivity was generally high, and all patients achieved either complete or partial remission following treatment. GTL was identified in 6% to 20% of glomeruli, in addition to typical features of membranous nephropathy. Most cases were classified as Ehrenreich–Churg stage I or II, with minimal interstitial injury. Immunohistochemical analysis demonstrated cytokeratin 7 (CK7)-positive parietal epithelial cells (PECs) within the segmental sclerotic lesions in four of the six cases. Serial section analysis confirmed co-localization of CK7 and annexin A3, indicating the presence of a subset of activated PECs potentially involved in GTL formation, resembling mechanisms proposed for tip lesions in FSGS.

Conclusion:

All MN cases with GTL presented with nephrotic syndrome and responded well to therapy, showing a trend toward favorable prognosis. GTL may represent a distinct morphological variant within MN with potential prognostic implications.

Keywords：membranous nephropathy, glomerular tip lesions, parietal epithelial cells

Poster Presentation：Glomerular Diseases
Poster No.：C0496
Abstract Submission No.：APCN20250720

Clinical Case Summary Severe Postpartum Flare of Systemic Lupus Erythematosus (SLE) and Lupus Nephritis: A Case Report

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Abstract
Case Presentation
The patient was previously diagnosed with glomerulonephritis in May 2023. On April 9, 2025, at 29–30 weeks of gestation, she delivered vaginally at the National Center for Maternal and Child Health due to severe preeclampsia superimposed on chronic glomerulonephritis. She was admitted to the Nephrology Center of the First Central Hospital on April 21, 2025 (26 days postpartum), with complaints of shortness of breath, generalized edema, abdominal distension, and decreased urine output.

Physical examination:
Oxygen supplementation was required (SpO_2: 94% on 5L/min). Shallow breathing, respiratory rate: 22/min, BP: 145/110 mmHg, HR: 91 bpm. Urinary catheter output: ~700 mL/day. Peripheral edema in the lower limbs and back. Body weight: 94 kg. Abdominal girth: 112 cm.
Laboratory findings: CBC: WBC 11.8 $\times 10^9$/L, HGB 8.3 g/dL, HCT 24.2%, PLT 275 $\times 10^9$/L
Biochemistry: Albumin 17 g/L, Total protein 45.7 g/L, CRP 0.78 mg/L, Creatinine 0.83 mg/dL, Cholesterol 10.7 mmol/L, Urea 33.4 mg/dL
Immunology: ANA detect 8.9 (↑), ANA profile: SS-B 1.24 (↑), SS-A52 1.69 (↑), SS-A60 1.65 (↑), Anti-dsDNA 22.5 (↑), p-ANCA 0.5, c-ANCA 0.9, C4 12.2 mg/dL, C3 32.5 mg/dL (↓)
Urinalysis: RBCs 0.75 mg/dL, Protein 3.0 mg/dL, Protein/Creatinine ratio 2+, Albumin/Creatinine ratio 2+
24-hour urine protein: 7.8 g/day
Abdominal ultrasound: Both kidneys 13.1 cm, with increased echogenicity and preserved cortical thickness. Significant ascites observed.
Chest X-ray: Bilateral pleural effusions below the 6th rib; cardiomegaly.
Echocardiogram (04/24): Left ventricular hypertrophy, EF 80%, normal PASP, pericardial effusion (1–2 cm).
Treatment and Outcome: Initial therapy included pulse methylprednisolone (500 mg x 3), followed by oral (28 mg), mycophenolate mofetil (2000 mg), hydroxychloroquine (400 mg), ARB, anticoagulants, antiplatelets, statins, and supportive therapies. Twelve days after initiation of pulse therapy, her symptoms had resolved. Body weight decreased to 54 kg Repeat labs: CBC: WBC 17.8, HGB 11.8, HCT 35.0, PLT 403
Biochemistry: Albumin 25 g/L, Total protein 48.9 g/L, CRP 0.57 mg/L, Creatinine 0.46 mg/dL, Cholesterol 13.8 mmol/L, Urea 32 mg/dL Urinalysis: No RBCs, Protein 3.0 mg/dL Imaging: Resolution of pleural and pericardial effusions

Discussion
SLE poses a significant risk of multiorgan involvement during flares, particularly affecting kidneys, lungs, heart, and the nervous system. In this case, the postpartum period may have triggered immune dysregulation and subsequent disease flare. Timely and comprehensive management led to favorable outcomes. The patient responded well to combined glucocorticoid and immunosuppressive therapy.

Conclusion
Postpartum flares of SLE can present as severe, multisystem illness. Early diagnosis and aggressive treatment are crucial to preserving organ function and preventing progression to chronic kidney disease.

Keywords： Keywords: Systemic lupus erythematosus, lupus nephritis, anti-dsDNA, ANA, nephrotic syndrome

Poster Presentation：Glomerular Diseases
Poster No.：C0497
Abstract Submission No.：APCN20250739

Clinicopathological Features of Unclassified Immune Complex Glomerulonephritis: A Retrospective Study from a Single-Center in Taiwan

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Abstract

Background:

Immune complex glomerulonephritis (IC-GN) encompasses a broad range of disorders, many of which are well-defined by underlying etiologies such as lupus nephritis, infection-related GN, or monoclonal gammopathy-associated GN. However, a subset of patients presents with immune complex deposition without fulfilling the diagnostic criteria for any established glomerular disease entity. These unclassified cases remain poorly understood.

Methods:

We retrospectively reviewed renal biopsy cases from 2016 to 2024 at a tertiary hospital in Taiwan. Among them, 28 patients were identified as having immune complex glomerulonephritis that did not fulfill the diagnostic criteria for any established glomerular disease entity. Clinical data, laboratory findings, pathological features, and outcomes were analyzed. Patients were further stratified by the presence or absence of a membranoproliferative glomerulonephritis (MPGN) pattern.

Results:

Among the 28 cases, potential associations included autoimmune features (28.6%), paraproteinemia (35.7%), hematologic malignancy (14.3%), solid organ tumors (3.6%), and chronic infections (17.9%). Patients with MPGN had significantly higher systolic and diastolic blood pressure at presentation ($p = 0.001$ and $p < 0.001$, respectively). Although not statistically significant, the MPGN group tended to have higher proteinuria (mean 4.31 vs. 2.32 g/day), lower serum albumin, and worse renal function.

Overall, renal outcomes were poor across both groups: nearly 40% of patients progressed to dialysis-dependent kidney failure, with several requiring dialysis at the time of diagnosis or shortly thereafter. In addition, mortality during follow-up was notable, highlighting the severity and progressive nature of this condition.

Conclusion:

A small but distinct group of patients with immune complex GN lacks an identifiable underlying etiology yet exhibits substantial renal injury and unsatisfactory clinical outcomes. Although these cases cannot be clearly classified into existing glomerular disease entities, many are associated with specific clinical contexts—such as autoimmune diseases, paraproteinemia, hematologic disorders, solid organ tumors, or chronic infections—which may offer diagnostic and therapeutic insights.

Keywords：immune complex GN; autoimmunity; paraproteinemia; malignancy; chronic infection

Poster Presentation：Glomerular Diseases
Poster No.：C0498
Abstract Submission No.：APCN20250778

Longer Follow-Up of Povetacicept Shows Potential for Treatment of Primary Membranous Nephropathy (RUBY-3 Study)

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Abstract

Introduction: pMN is caused by pathogenic B-cells that produce autoantibodies, such as anti-PLA2R, which attach to autoantigens on podocytes, resulting in glomerular injury. BAFF and APRIL are central to pMN pathogenesis by promoting survival and maturation of pathogenic transitional and naïve B-cells, T-cell-independent B-cell responses to certain antigens, B-cell regulation, and Ig class-switch recombination. Povetacicept, a dual inhibitor of BAFF and APRIL, represents a significant therapeutic advancement by targeting the root cause of disease. Updated interim data for participants dosed with povetacicept in the RUBY-3 study are provided and longer-term data will be provided in the presentation.

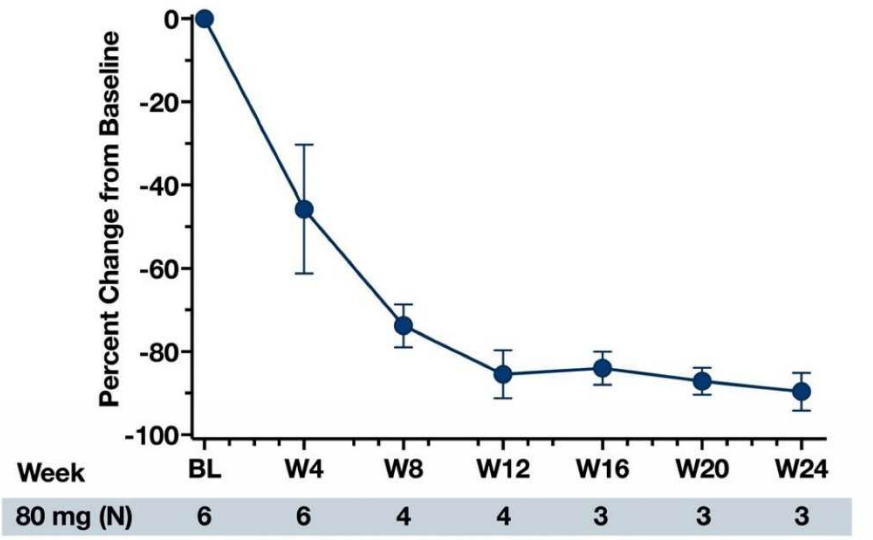
Methods: RUBY-3 is an ongoing Phase 1/2, open-label study in adults with pMN, receiving povetacicept 80 mg subcutaneously administered Q4W (n=10 dosed; n=5 at 24 weeks). Primary objective: evaluation of the safety of povetacicept. Secondary objectives: efficacy, PK, and biomarker changes with povetacicept treatment.

Results: Data at 24 weeks (n=5) indicate mean 24-hour UPCR decreased 57% (from 3.8 g/g to 1.6 g/g) and eGFR was stable. By Week 24, 80% (4/5) of participants achieved immunologic remission, 60% (3/5) of participants achieved partial clinical remission, and mean anti-PLA2R autoantibody declined from baseline by 78%. In the subset of 3 participants at moderate to high risk of disease progression defined by baseline 24-hour UPCR >3.5 g/g, at Week 24, mean 24 hour UPCR decreased by 62% (from 5.0 g/g to 1.9 g/g); eGFR was stable; 67% (2/3) participants achieved immunologic remission and partial clinical remission; and anti-PLA2R autoantibodies declined 90%. Povetacicept was generally safe and well tolerated.

Conclusion: Povetacicept was generally safe and well tolerated in adults with pMN through 24 weeks and resulted in substantial reductions in UPCR and anti-PLA2R autoantibody and stable eGFR including in subjects at high risk of disease progression. These updated data reinforce the potential of povetacicept as therapy for pMN.

Keywords：pMN, primary membranous nephropathy, povetacicept, RUBY-3, PLA2R, UPCR, glomerular nephritis, eGFR, BAFF, APRIL, B cell

RUBY-3 Study, Anti-PLA2R Autoantibody Mean Percentage Change from Baseline to Week 24



Poster Presentation：Glomerular Diseases
Poster No.：C0499
Abstract Submission No.：APCN20250792

Rapid Renal Recovery with Telitacicept and Nefecon Combination in Advanced IgA Nephropathy: A Case Report

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Abstract

Introduction

Immunoglobulin A nephropathy (IgAN) with chronic kidney disease (CKD) stage G4 often progresses despite standard therapy. We present the first case demonstrating efficacy of telitacicept (BLyS/APRIL inhibitor) combined with nefecon (gut-targeted budesonide) in treatment-resistant IgAN.

Methods

A 60-year-old male with biopsy-proven IgAN (Oxford M1E0S1T1C0, Lee grade IV, CKD G4) showed:
Baseline (August 2023): Serum creatinine (Scr) 278 μmol/L, 24-hour urine protein (24h-UP) 4742 mg
Therapeutic interventions:
1.Initial: Hydroxychloroquine + sacubitril/valsartan (August 2023)
2.Intensified: Telitacicept 160 mg/week + dapagliflozin (September 2023)
3.Rescue: Finerenone (February 2024)
4.Definitive: Telitacicept 80mg/week+ nefecon 16 mg/day (October 2024)

Results

After 9 months of combination therapy (June 2025):
Proteinuria: 24h-UP decreased 93.8% (4742 → 294 mg/day)
Renal function: Scr reduced 20.3% (300 → 239 μmol/L)
eGFR increased >30%
Safety: No infections, hyperkalemia, or hepatotoxicity

Conclusion

Telitacicept-nefecon therapy achieved:
1.Near-complete proteinuria remission (24h-UP <300 mg/day).
2.Significant renal function improvement despite advanced chronicity.
This dual-target strategy warrants prospective validation for high-risk IgAN.
3.Safety: No infections, hyperkalemia, or liver injury observed.

Keywords：Immunoglobulin A nephropathy,Telitacicept,Nefecon

Poster Presentation：Glomerular Diseases
Poster No.：C0500
Abstract Submission No.：APCN20250800

Crescentic IgAN and anti-GBM nephritis without linear GBM immunofluorescence: A first report case

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Abstract

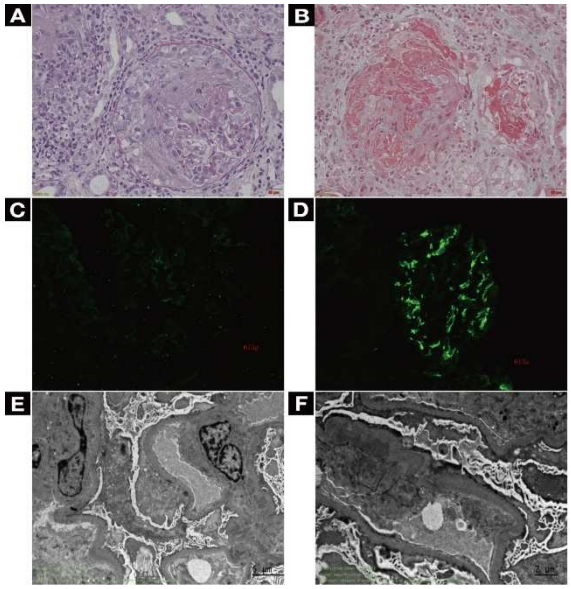
Introduction: Anti-glomerular basement membrane (anti-GBM) disease is instigated by circulating pathogenic autoantibodies that specifically target autoantigens expressed in the basement membranes of organs. When the kidneys are affected, it can lead to crescent formation and glomerular necrosis, with immunofluorescence typically showing linear deposition of IgG and C3 along the GBM. Although rare, a pauci-immune renal presentation can occur. Crescentic IgA nephropathy (IgAN) is a distinct type of IgAN characterized by rapid clinical progression and poor prognosis. The co-existence of anti-GBM nephritis crescentic and IgAN without GBM deposition has not been previously reported. This study presents a unique case that contributes to the understanding of such complex renal diseases.

Methods: We describe the case of a 34-year-old male with chief complaints of gross hematuria accompanied by fever for half a month and increasing serum creatinine（Scr）for 2 days. The serum anti-GBM antibody was positive, and the renal biopsy suggested crescentic glomerulonephritis. However, there was no immunoglobulin （Ig） deposition along the GBM, while immunoglobulin A（IgA）and C3 were lumpy-stained in the mesangial area. Therefore, crescentic IgAN complicated with anti-GBM nephritis was considered. He received a pulse dose of intravenous methylprednisolone and cyclophosphamide, with plasmapheresis for 22 sessions during hospitalization, until the anti-GBM antibody turned negative.

Results: The level of SCr did not decline, and the patient had to remain on dialysis.

Discussion: This case represents a novel combination of crescentic IgAN and anti-GBM nephritis without typical GBM deposition. The lack of response to treatment in terms of SCr reduction and the need for continuous dialysis highlight the complexity and severity of this co-existing condition. Further research is warranted to better understand the underlying mechanisms and develop more effective treatment strategies for such rare and challenging renal diseases.

Keywords：rescentic IgA nephropathy; anti-GBM disease; rapidly progressive glomerulonephritis（RPGN）; crescent formation; immunofluorescence



Poster Presentation：Glomerular Diseases
Poster No.：C0501
Abstract Submission No.：APCN20250809

Rising Burden of Chronic Kidney Disease Due to Glomerulonephritis Attributable to High Sodium Diet in Indonesia: GBD 2017–2021 Analysis

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Abstract

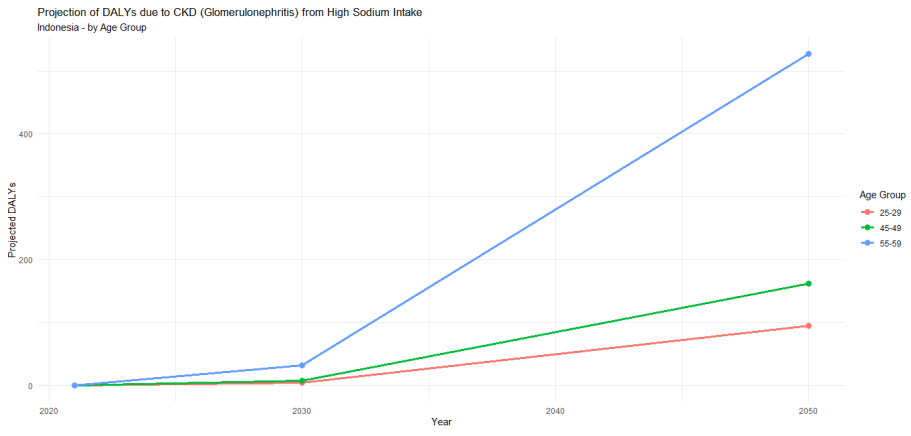
A high-sodium diet is a well-established risk factor for hypertension and progressive kidney damage, particularly through glomerular injury. However, age-stratified estimates of chronic kidney disease (CKD) attributable to sodium intake due to glomerulonephritis are scarce in Indonesia. This study aims to quantify the age-specific burden of CKD linked to glomerulonephritis and high sodium intake between 2017 and 2021.

Data were obtained from the Global Burden of Disease (GBD) study for the years 2017–2021. We analyzed disability-adjusted life years (DALYs) attributable to high sodium intake in relation to CKD caused by glomerulonephritis in three adult age groups (25–29, 45–49, and 55–59 years). The compound annual growth rate (CAGR) was calculated to assess trends over time.

The 55–59 age group bore the highest burden, with DALYs increasing from 75 in 2017 to 95 in 2021 (CAGR: +6.08%). The 45–49 group showed a modest increase from 3 to 4 DALYs (CAGR: +7.46%). Notably, there were no DALYs recorded for the 25–29 age group during the entire period, suggesting either delayed onset or under-recognition of sodium-attributable glomerular disease in younger adults.

The rising burden of glomerulonephritis-related CKD attributable to dietary sodium among older Indonesian adults underscores the need for age-targeted interventions. These findings support the implementation of national sodium reduction policies and kidney health education to mitigate long-term renal complications.

Keywords：Chronic kidney disease (CKD), Glomerulonephritis, Sodium intake



Poster Presentation：Glomerular Diseases
Poster No.：C0503
Abstract Submission No.：APCN20250878

Correlation between crescentic formation causing rapidly progressive glomerulonephritis and renal outcomes in Thai lupus nephritis

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Abstract

Background

Usually, more than 50% crescentic formation associated with idiopathic rapidly progressive glomerulonephritis (RPGN). The amount of crescentic formation in lupus nephritis (LN) that causes RPGN is inconclusive. This study aimed to determine the significant number of crescents in LN leading to RPGN, and predicting for clinical outcomes including renal remission, renal flare, doubling of serum creatinine (SCr), end stage renal disease (ESRD), and mortality outcome.

Methods

Patients performed kidney biopsy between 2010 - and 2015 were retrieved from medical records. Inclusion criteria were: 1) age 2 18 years 2) biopsy-proven LN. This study excluded transplanted kidneys. Patients with RPGN were identified by clinical syndrome of rapid loss of renal function without definite cause in glomerular disease. Baseline demographic data included age, gender, CBC, SCr, estimated glomerular filtration rate (eGFR), albumin, urine protein creatinine ratio (UPCR), % of glomerular sclerosis, crescent, interstitial fibrosis, tubular atrophy, activity index (Al: cellular/fibrocellular crescent, endocapillary hypercellularity, leukocyte infiltration, fibrinoid necrosis, hyaline thrombi, interstitial inflammation) , chronicity index (Cl: glomerular sclerosis, fibrous crescent, tubular atrophy, interstitial fibrosis), and renal outcomes were recorded.

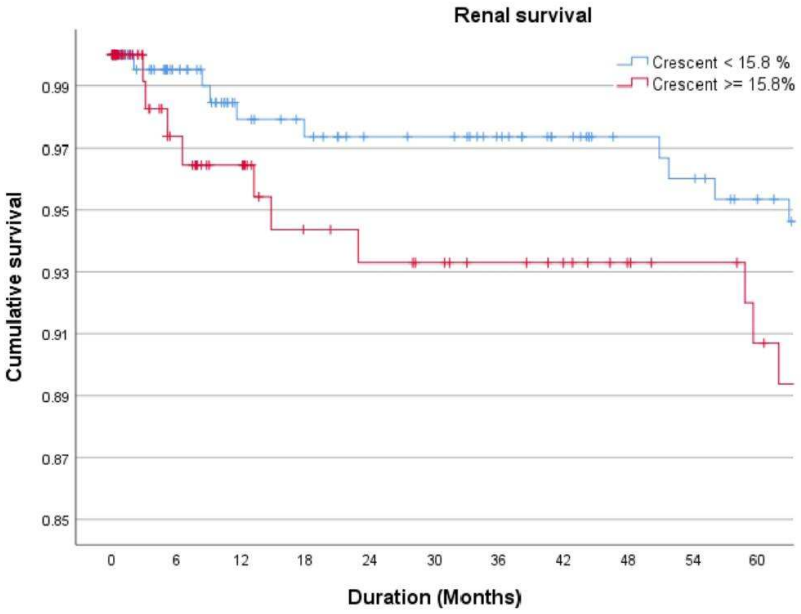
Results

A total of 406 patients (155 patients with crescents 2 15. 8%). Median age, SCr, serum albumin, UPCR in crescents 2 15.8 % group (/!V compared to crescent < 15.8% group (B) were 30 and 31 years old, 1.5 and 0.9 mg/dl, 2.59 and 2.7 g/dl, 4.78 and 4.14 gig.Cr, respectively. Crescents 2 15.8% associated with RPGN with the odds ratio 3.76 [2.45 - 5.77, p < 0.001]. After adjusting with Al and Cl score, this cutoff value was not demonstrated a significant correlation. Hazard ratio for predicting renal flare was 0.57 [0.38-0.85, p 0.006]. This cut-off value had a tendency to predict renal remission, doubling of serum creatinine, ESRD, and death.

Conclusions

From our study, the cutoff point of crescentic formation affecting kidney function potentially RPGN was 15. 8% that persuaded the proper treatment. Crescents < 15. 8% predicted more renal flare. However, this cutoff was not significantly predicting renal remission, doubling of serum creatinine, end stage renal disease and death.

Keywords：Crescentic glomerulonephritis, Rapidly progressive glomerulonephritis, Lupus nephritis


Renal survival
Crescent < 15.8 %
Crescent >= 15.8%
Cumulative survival
0.99
0.97
0.95
0.93
0.91
0.89
0.87
0.85
0
6
12
18
24
30
36
42
48
54
60
Duration (Months)

Poster Presentation：Glomerular Diseases
Poster No.：C0504
Abstract Submission No.：APCN20250890

Studying The Effects Of Rituximab Treatment In Pediatric Steroid-Dependent Nephrotic Syndrome In Mongolia

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Abstract

Background: The annual incidence of nephrotic syndrome is 1.15-16.9 new cases per 100.000 children worldwide. 85-90% of patients have complete remission of proteinuria within 4-6 weeks of steroid therapy and there are steroid-sensitive.
Therefore, we aimed to use rituximab therapy in children with steroid-dependent nephrotic syndrome and study the results.

Material and methods
The study was conducted from March to June 2025, based on the National Center for Maternal and Child Health, using a retrospective approach and selecting research designs appropriate for each objective. The statistical processing of the study was performed using Excel, IBM-SPSS 27, and Rev Man 5.0 programs.

Results
The mean age of the study participants was 12.5±3.9, the mean age at diagnosis was 8.3±3.8, and 86.4% (n=19) were male, and 59.1% (n=13) were from Ulaanbaatar. In the clinical case series, the mean eGFR at the start of rituximab treatment was 90.3±14.1, which increased to 106.4±19.3 after six courses of treatment. There were also no relapses after treatment. When comparing the test results at the beginning of treatment and after six courses of treatment in the rituximab-treated cases, there were statistically significant differences in the levels of cholesterol (p=0.04; CI: 0.01-3.1) and albumin (p=0.02; CI: -3.7-0.1). There was no statistically significant difference in the number of leukocytes, neutrophils, or lymphocytes in the assessment of B-cell regeneration (p=0.102, p=0.158, p=0.287). For Objective 2, there was a statistically significant difference in the eGFR between the rituximab-treated and non-rituximab-treated groups (p=0.04; CI: 1-23).
There was also a statistically significant difference in disease relapse (p=0.03). A total of 7 international studies were included in Objective 3, and rituximab treatment increased survival by 69% in children with steroid-dependent, prequently relapsing nephrotic syndrome (HR=0.31, CI 0.16-0.54).

Conclusion
1. Rituximab treatment is effective in children with steroid-dependent, prequently relapsing nephrotic syndrome, as demonstrated by clinical data. Long-term follow-up studies are needed.
2. The results of rituximab treatment in children with steroid-dependent, prequently relapsing nephrotic syndrome are shown in the form of increased glomerular filtration rate, improved renal function, and reduced number of disease relapse compared to the control group.
3. International studies have shown that rituximab treatment is effective in children with steroid-dependent, prequently relapsing nephrotic syndrome and increases patient survival.

Keywords：SDNS, pediatric nephrotic syndrome, rituximab

Poster Presentation：Glomerular Diseases
Poster No.：C0505
Abstract Submission No.：APCN20250896

Case report: A Rare Case of IgA Nephropathy Associated with Type II Abernethy Malformation

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Abstract
Background: Abernethy malformation (AM), a rare congenital portosystemic shunt, is exceptionally rarely associated with glomerulonephritis. The causal link and therapeutic implications of portosystemic shunting in the pathogenesis of IgA nephropathy (IgAN) remain poorly defined, especially in Type II AM preceding advanced cirrhosis.

Case Presentation: A 30-year-old male presented with nephrotic syndrome and biopsy-proven mesangial proliferative IgAN (Lee III, Oxford M1E0S1T0-C1). Concurrently diagnosed cirrhosis (CT: nodularity, splenomegaly, varices) and subsequent recurrent hepatic encephalopathy led to angiography, revealing a definitive Type II AM with a high-flow portocaval shunt draining into the inferior vena cava, attenuated portal flow, and anomalous vessels. This represents one of fewer than ten reported cases globally linking AM with IgAN.

Intervention: Due to hemorrhage risk upon trial clamping, laparoscopic partial occlusion (80% reduction) of the portosystemic shunt was performed (May 2023).

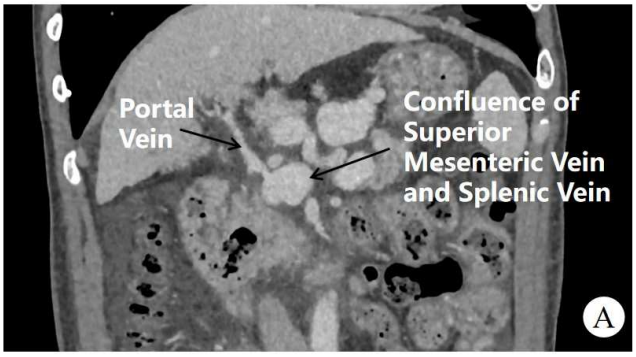
Outcomes: Post-operatively, portal flow velocity increased (10-12 cm/s vs. 6-8 cm/s baseline). Crucially, proteinuria decreased from a pre-intervention baseline of 0.8-2.1 g/24 h to 0.19-0.29 g/24 h at 2 months postoperatively. Hepatic encephalopathy episodes significantly reduced during 10-month follow-up.

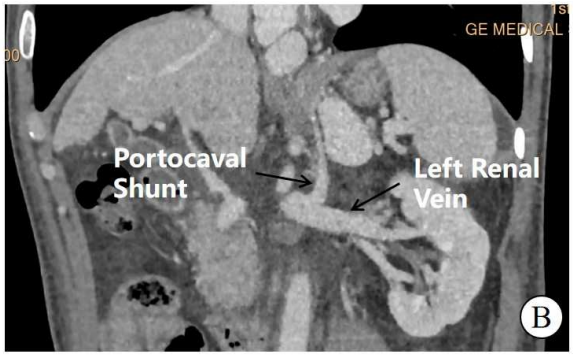
Discussion: This case offers strong clinical support for a direct pathogenic effect of portosystemic shunting in IgAN, distinct from cirrhosis-related mechanisms. The marked and persistent drop in proteinuria after partial shunt correction (from 0.8-2.1 g/24h pre-op to 0.19-0.29 g/24h at 2 months post-op), achieved without increased immunosuppression, points strongly to shunting-induced bypass of hepatic clearance as a key mechanism driving glomerular injury through systemic IgA immune complex accumulation. This improvement occurred even in the presence of established cirrhosis, suggesting the shunt's effect may be separate from, or additive to, cirrhosis itself. These findings support the proposed disease mechanism and are consistent with limited previous observations of renal improvement following shunt correction. Notably, this case shows that significant renal benefit can be gained with partial shunt occlusion when complete closure is not feasible due to safety concerns.

Conclusion: This exceptionally rare case highlights Type II AM as a potential primary etiology for IgAN. The substantial reduction in proteinuria following targeted shunt modification provides convincing clinical evidence that reducing aberrant portosystemic shunting directly influences IgAN disease activity. Early intervention on the shunt could potentially slow progressive glomerular damage. These findings suggest that evaluating for portosystemic shunts should be considered in cases of unexplained IgAN, especially when hepatic abnormalities are present, and that shunt correction might offer a non-immunosuppressive treatment avenue. Multicenter studies are needed to confirm

the long-term renal benefits of this approach.

Keywords： IgA nephropathy, type II Abernethy malformation, portosystemic shunt, congenital extrahepatic shunt disconnection, case report.





Poster Presentation：Glomerular Diseases
Poster No.：C0508
Abstract Submission No.：APCN20250925

Comparison Between Standard of Care Treatment and Dapagliflozin With Standard of Care Treatment In Lupus Nephritis Patients : A Randomized Controlled Trial

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Abstract

Introduction: The exploration of sodium-glucose cotransporter-2 inhibitors (SGLT-2i), particularly Dapagliflozin, presents a novel avenue in treating Lupus Nephritis (LN). Stemming from the intriguing observations in the DAPA-CKD trial, where non-diabetic patients with chronic kidney disease experienced significant cardiovascular and renal benefits, this study investigates the potential advantages of adding Dapagliflozin to the standard of care treatment in Lupus nephritis patients.

Materials and Methods: We conducted a randomised controlled open-label study by enrolling 32 newly diagnosed Lupus nephritis (Class III/IV/V/III+V/IV+V) patients. Among them, 17 received Dapagliflozin in addition to standard care, while 15 received standard care alone. The patients were followed up monthly for 6 months. The primary Outcome was the treatment response after 6 months as per the KDIGO guidelines. The Secondary Outcome included the frequency of urinary tract Infections in both arms, the number of lupus flares in both arms and the number of SLE-related complications and deaths in each arms.

Results: Baseline characteristics revealed no significant demographic differences between the intervention (MMF + steroids + Dapagliflozin) and standard of care (MMF + steroids) groups (Table 1). While both groups exhibited prevalent lupus-related symptoms, the intervention group demonstrated significantly lower mean systolic blood pressure and total protein levels at baseline. At the 6-month follow-up, the clinical response rate in the intervention group was 81.8%, with a numerical trend toward better outcomes compared to the standard of care group (60%) (p value - 0.425). The frequency of urinary tract infections was higher in the dapagliflozin arm, albeit not statistically significant (23.1% vs 6.7% ; p value - 0.31). No significant differences were observed in serum creatinine, proteinuria, or serum albumin between the groups at the 6-month mark. Adverse events, including death and infections, were comparable between the groups.

Conclusion: This pilot study suggests that the addition of Dapagliflozin to standard care may offer a potential avenue for improving clinical responses in LN with acceptable adverse effects. Despite limitations, the results highlight a promising trend that warrants further investigation.

Keywords：lupus nephritis, systemic lupus erythematosus, dapagliflozin, mycophenolate mofetil, steroids, clinical response, urinary tract infections, renal biopsy, SGLT2 inhibitors, DAPA CKD

Characteristic	Intervention group (MMF + steroids + Dapagliflozin) n = 17	Standard of care treatment (MMF + steroids) n = 15	P-value
Demographic characteristics			
Mean Age (in years)	35.71±10.90	33.53±9.16	0.549
M/F (n)	2/15	3/12	0.645
Renal biopsy • Class III LN • Class IV LN • Class V LN • Class III + V LN • Class IV + V LN	 2 (11.8%) 3 (17.6 %) 7 (41.2 %) 4 (23.5%) 2 (11.8%)	 2 (13.3%) 5 (33.3%) 2 (13.3%) 3 (20%) 3 (20%)	 1.000 0.423 0.122 1.000 0.645
Estimated GFR **≥60 ml per minute per 1.73 m2** **≥90 ml per minute per 1.73 m2**	 14 (82.3%) 11 (64.7%)	 12 (80%) 9 (60%)	
SLEDAI-2K score (mean)	24.47±6.93	24.40±6.26	0.976
Biomarkers • Antinuclear antibodies • Low C3 and C4 • Anti ds DNA	 17 (100%) 12 (70.6%) 12 (70.6%)	 15(100%) 13 (86.7%) 12 (80%)	 0.402 0.691
Proteinuria (g/day) (median)	3.9 (2.25 – 6.75)	2.80 (1.53 – 4.7)	0.50
Serum Albumin (g/dl) (mean)	2.67(1.88-3.03)	2.92(2.30-3.37)	0.093
Serum Creatinine(mg/dl) (median)	0.79(0.60-1.14)	0.80(0.70-1.17)	0.705

Abbreviations – MMF – mycophenolate mofetil , M -males , F- females , n – number , LN -Lupus nephritis , SLEDAI -SLE disease activity index

Poster Presentation：Glomerular Diseases
Poster No.：C0509
Abstract Submission No.：APCN20250936

Nefecon (Budesonide Delayed-release Capsules) Achieves Superior Renal Protection In IgA Nephropathy: A Case Study

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Abstract

Background: A 57-year-old male with 7-year history of abnormal urinalysis and 3-year renal dysfunction was admitted on February 27, 2024. Past medical history included right partial nephrectomy for renal cell carcinoma, hypertension, and gout. Baseline serum creatinine was 138 μmol/L (eGFR 48.87 mL/min) with proteinuria (2.3g/24h).

Methods: Renal biopsy confirmed mild mesangial proliferative IgA nephropathy (Lee grade II). Treatment included:

Telitacicept (160 mg/week subcutaneously, February–November 2024)

Nefecon (budesonide delayed-release capsules 16 mg/day, December 2024–present)

Concomitant therapies: irbesartan, febuxostat, and lifestyle modifications.

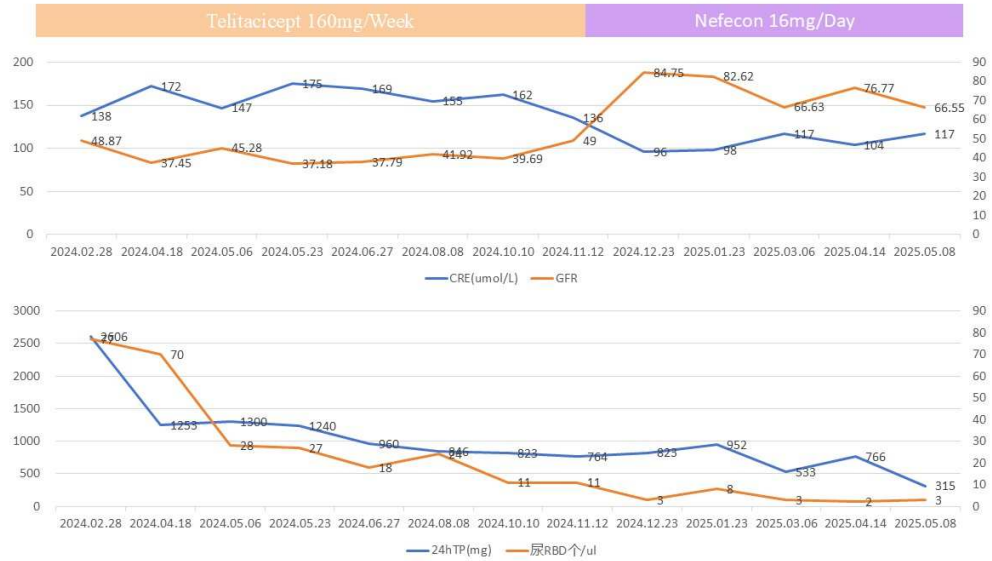
Results:

Telitacicept phase (9 months): Proteinuria decreased from 2.3g to 0.8g/24h, and the serum creatinine has no significant change (138→175→136 μmol/L).

Nefecon phase (6 months): Proteinuria further reduced to 0.3g/24h with significant renal protect (creatinine: 136→96→117 μmol/L). Hematuria resolved, and antihypertensive regimen was simplified. No adverse events occurred. (Figure1)

Conclusion: While telitacicept reduced proteinuria, But the serum creatinine has no significant change, Nefecon achieved sustained proteinuria reduction with superior renal function preservation, highlighting its efficacy in IgA nephropathy management.

Keywords：IgA nephropathy, Telitacicept, Nefecon, Proteinuria, Renal protection



Poster Presentation：Glomerular Diseases
Poster No.：C0510
Abstract Submission No.：APCN20250942

Evaluation Of Pathophysiology Biomarkers In Kidney Biopsies Collected In Imagination, A Phase 3 Study Of Sefaxersen In Patients With IgA Nephropathy

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Abstract

Introduction: The complement alternative pathway (AP) has been implicated in the pathogenesis of IgA nephropathy (IgAN) and increased activity of Factor B (FB), a key mediator of the AP, is associated with poorer kidney outcomes. Mesangial deposition of IgA and complement, including AP proteins, is often observed in the diagnostic biopsy but there is limited understanding of in situ changes during disease progression or treatment. Sefaxersen (RO7434656, IONIS-FB-LRx), an antisense oligonucleotide (ASO) against FB, is the first AP mRNA-targeting therapy in late-stage development for the treatment of IgAN. We previously reported reductions in AP activity and urine protein to creatinine ratio (UPCR) in a Phase 2 IgAN study of sefaxersen (NCT04014335).

Methods: IMAgINATION (NCT05797610) is a Phase 3, randomized, double-blind, placebo-controlled trial evaluating the efficacy and safety of sefaxersen in adults with biopsy-confirmed primary IgAN. The primary endpoint is a change from baseline in 24h UPCR at Week 37. Key secondary endpoints include eGFR slope from baseline at Week 105, time to the composite kidney failure endpoint and patient-reported outcomes. In addition to blood and urine biomarkers, optional kidney biopsy samples will be collected at baseline, Week 37, and Week 105 for histopathology assessments including MEST-C score, complement and immune complex deposition, and exploratory biomarkers of inflammation and fibrosis.

Results: Characterization of baseline and post-treatment kidney tissue biomarkers and their concordance with blood/urine biomarkers and disease progression are expected upon study completion.

Conclusions: The assessment of blood, urine and tissue pathophysiology biomarkers in the placebo-controlled IMAgINATION study of sefaxersen may uniquely support the understanding of systemic and in situ drug mechanism of action, disease modification, and association with disease

progression in patients with IgAN.

“This abstract was also submitted for the ASN Kidney Week 2025 congress. By submitting the abstract to APCN x TSN 2025, abstract authors declare that re-submitting the abstract is permitted by the organizers of the previous meeting”

Keywords：IgAN, complement, Factor B, alternative pathway, kidney biopsy, ASO, IMAgINATION, sefaxersen

Poster Presentation：Glomerular Diseases
Poster No.：C0511
Abstract Submission No.：APCN20250951

Remarkable Renal Recovery with Targeted-Release Budesonide in Treatment-Refractory IgA Nephropathy at eGFR 11.95 mL/min/1.73m²: A Paradigm-Shifting Case

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Abstract
Clinical Profile
A 47-year-old male with biopsy-proven IgA nephropathy (Lee III-IV) presented with progressive renal impairment:
• 8-year disease course
• 2017: Initial biopsy → mesangial proliferative GN
• 2022: Repeat biopsy → IgAN (Lee III-IV), Proteinuria 0.94 g/d, Scr 218 μmol/L
• 2023: Failed conventional therapy (prednisone + Telitacicept)
• Admission (2024): Scr 469 μmol/L, eGFR 11.95 mL/min/1.73m², Proteinuria 0.78 g/d (CKD 4)

Therapeutic Intervention
Table1.Treatment Timeline
Date Therapy Scr (μmol/L)
2017-2022 RAASi Scr 218 μmol/L
May 2023 Prednisone 20mg/day Scr 202 μmol/L
Oct 2023 + Telitacicept 160mg/week Scr 319 μmol/L
Jan 2024 Steroid taper Scr 240 μmol/L
Apr 2024 Therapy discontinuation Scr 443 μmol/L
Jul 2024 Nefecon initiation 16mg/day Scr 450 μmol/L
May 2025 Nefecon maintained Scr 360 μmol/L

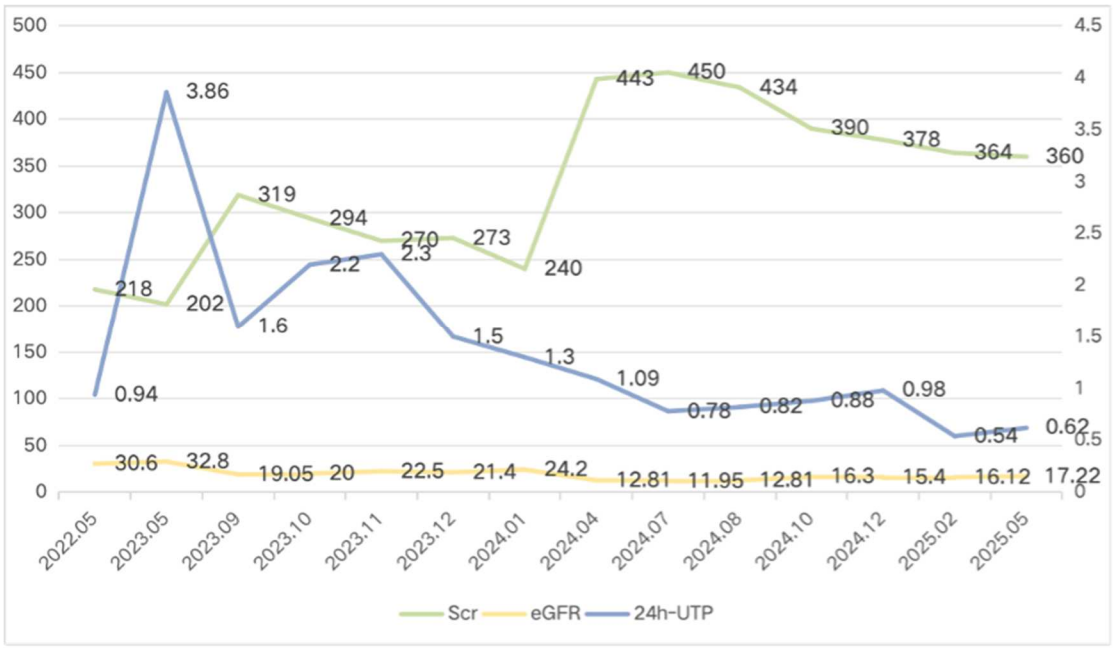
Table2.Treatment Response-(Nefecon 16mg/day): 9-month follow-up
Date Scr (μmol/L) eGFR (mL/min/1.73m²) 24h-UTP (g/d)
Jul 2024 450 11.95 0.78
Aug 2024 434 12.81 0.82
Oct 2024 390 16.30 0.88
Dec 2024 378 15.40 0.98
Feb 2025 364 16.12 0.54
May 2025 360 17.22 0.62

Figure1. Serial Renal Parameters

Conclusion
In this adult patient with primary IgA nephropathy and severe renal insufficiency (eGFR <15 mL/min/1.73m²), the Targeted-Release Budesonide effectively reduced serum creatinine, improved eGFR, and delayed renal function progression over a 9-month period. This case strongly supports the imperative for timely and specific etiological intervention in IgA nephropathy. Commencing

targeted therapies like Targeted-Release Budesonide as early as possible in the disease course, rather than reserving them only for advanced stages, is critical to maximize the potential for preserving renal function and improving long-term outcomes ("treat early, benefit early"). Further investigation into the efficacy and safety of Targeted-Release Budesonide in late-stage IgA nephropathy is warranted.

Keywords：IgA Nephropathy; Targeted-release budesonide; CKD 4; Nefecon; Renal function preservation



Poster Presentation：Glomerular Diseases
Poster No.：C0512
Abstract Submission No.：APCN20250953

Clinical Efficacy of Targeted-Release Budesonide in an Adult with Primary IgA Nephropathy on Optimized Supportive Therapy

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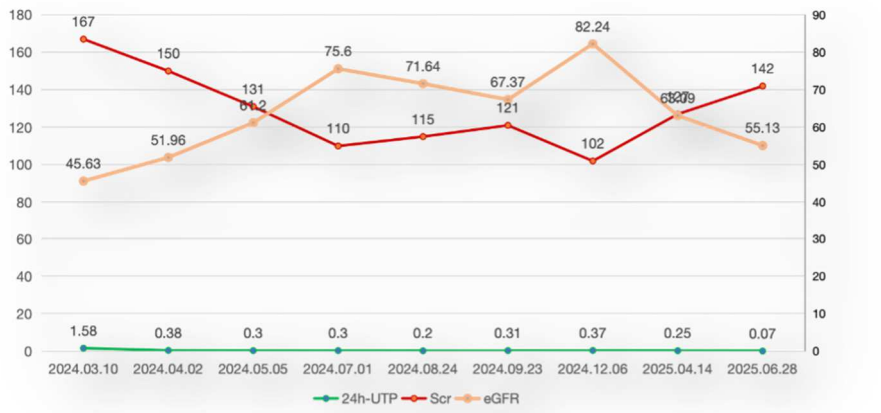
Abstract
This case report examines the renoprotective effect of Targeted-release budesonide (Nefecon) in a patient with progressive primary IgA nephropathy (IgAN) refractory to optimized supportive care.
Clinical Profile
A 33-year-old male presented in March 2024 with hypertension (7 years) and renal dysfunction (6 years).
Disease progression:
• 2017: Incident hypertension (untreated)
• 2018: BP 180/100 mmHg; proteinuria 1+; Scr 142 μmol/L
• 2018-2024: On amlodipine+candesartan (peak Scr 350 μmol/L)
• Feb 2024: Proteinuria 2.70 g/d, Scr 144 μmol/L, eGFR 54.59 mL/min/1.73m²
Baseline characteristics (Mar 2024):
• BP 132/90 mmHg, BMI 31.1 kg/m²
• Lab: Proteinuria 1.58 g/d, Scr 167 μmol/L, eGFR 45.63 mL/min/1.73m²
• Serology: Autoantibodies/ANCA/anti-GBM negative
Renal biopsy:
• 13 glomeruli
• 11 globally sclerosed (84.6%)
• 1 ischemic contraction
• 1 with mild mesangial hypercellularity
Diagnosis: IgAN with advanced chronicity
Therapeutic Intervention
1. Optimized supportive therapy: Perindopril 8 mg/d + dapagliflozin 10 mg/d
2. Add-on targeted therapy: Targeted-release budesonide 16 mg/d
3. Lifestyle: Low-salt/protein diet, weight reduction
Treatment Response
Week 4
• Proteinuria 0.38 g/d, Scr 150 μmol/L, eGFR 51.96 mL/min/1.73m²
Week 36 (Targeted-release budesonide 16mg/d)
• Proteinuria 0.30 g/d, Scr 102 μmol/L, eGFR 82.24 mL/min/1.73m²
Week 37-38 (The targeted-release budesonide was tapered to 8 mg during weeks 37–38 and then discontinued)
• Proteinuria sustained at 0.30 g/d
• Progressive renal impairment: Scr 142 μmol/L, eGFR 55.13 mL/min/1.73m²
Conclusion
In this advanced IgAN case (＞84% glomerulosclerosis), Targeted-release budsonide:
1. Achieved rapid proteinuria remission (>75%) and eGFR recovery within 4 weeks
2. Maintained renal function during 9-month therapy period
3. Post-discontinuation observation：

• Persistent proteinuria control (<0.3 g/d)
• Significant Scr elevation and eGFR decline occurred
4. Clinical implications:
Extended maintenance therapy may be required for persistent renoprotection
More clinical practice guidance is needed for maintenance treatment protocols after 9 months period

Keywords：IgA nephropathy; Proteinuria; Targeted-release budesonide; Renoprotection



Poster Presentation：Glomerular Diseases
Poster No.：C0513
Abstract Submission No.：APCN20250955

Efficacy and Safety of Targeted-Release Budesonide in IgA Nephropathy with Severe Renal Impairment (eGFR <20 mL/min/1.73m²): A Case Report

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Abstract
Clinical Profile
A 43-year-old male with biopsy-proven IgA nephropathy (Lee II) presented with Progressive renal impairment:
• Baseline (2015): Scr 73 μmol/L, eGFR 90 mL/min/1.73m²
• Admission (2024): Scr 363 μmol/L, eGFR 16.64 mL/min/1.73m²(CKD 4) ， Proteinuria 1.73 g/d
• Comorbidities: Renal hypertension (BP 148/97 mmHg), hyperuricemia (586 μmol/L)
Diagnostic Confirmation
• Renal biopsy (2015): Mesangial hypercellularity, IgA dominant deposits
• 2024 Imaging:
Bilateral renal atrophy (right 94×39 mm, left 92×45 mm)
Cortical thinning (loss of corticomedullary differentiation)

Therapeutic Intervention
1. Targeted therapy: Targeted-release budesonide (Nefecon) 16 mg/d
2. Optimized supportive care:
• Nifedipine CR + carvedilol (30mg/d+12.5mg/d)
• Febuxostat (40mg/d)
• Calcium/Vitamin D (100 IU/d)
3. Ancillary: Traditional Chinese adjuvants

Treatment Response (9 Months)

Parameter	Baseline	Month 9	Δ (%)
Proteinuria (g/d)	1.73	0.25	-85.5%
Scr (μmol/L)	363	282	-22.3%
eGFR (mL/min/1.73m²)	16.64	22.42	+34.9%
BP (mmHg)	148/97	128/82	-13%/-15%

Discussion:
1. Unprecedented eGFR improvement:
• 34.9% eGFR increase defies natural decline
• Mechanism: Reduction in Gd-IgA1 production via gut-immune modulation
2. Safety advantages over systemic steroids:
• Targeted delivery: 90% first-pass hepatic metabolism → minimal systemic exposure
• Zero severe AEs: No infections, diabetes, or osteoporotic fractures
3. Clinical implications:
Targeted-release budesonide may bridge the therapeutic gap for advanced IgAN patients ineligible for conventional immunosuppression.

Conclusion

In this severe IgAN case (eGFR 16.6 mL/min/1.73m²)):

1. Efficacy confirmed: Rapid proteinuria remission (＞85%) , Significant eGFR recovery (+34.9%)
2. Safety validated: No systemic steroid toxicity

Keywords：IgA nephropathy; Advanced CKD; Targeted-release budesonide; Proteinuria; Renal function recovery

Figure1. Proteinuria (g/d)

Figure2. Scr (μmol/L)

Figure3. eGFR (mL/min/1.73m²)

Poster Presentation：Glomerular Diseases
Poster No.：C0514
Abstract Submission No.：APCN20250965

Waldenström Macroglobulinemia Mimicking Glomerulonephritis: A Case Report

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Abstract

Background:

Waldenström Macroglobulinemia (WM) is a rare, slow-growing B-cell lymphoma which is characterised by monoclonal IgM production. It is rare and patients usually present with insidious symptoms of fatigue, weight loss and recurrent infections. The high concentration of the large pentameric structure often leads to hyperviscosity syndrome. We described a case of steroid resistant nephrotic syndrome which ultimately turned out to be a case of haematological malignancy

Case presentation:

A 57-year-old gentleman who has type 2 diabetes mellitus for 1 year with fairly good control of diabetes mellitus DM (HbA1c 6.8%) and hypertension, presented with a new onset of nephrotic syndrome. He has profound oedema and ascites on clinical examination. Blood investigations showed acute kidney injury with serum creatinine was 161 umol/L, hypercholesterol, borderline serum corrected calcium 2.46 and hypoalbuminemia. The urinalysis showed haemoproteinuria and urine PCI was 1054.69mg/mmol. His DM eye screening revealed mild non-proliferative diabetic retinopathy. First renal biopsy performed showed focal segmental glomerulosclerosis with full house immunofluorescent staining. Infective screening, ANA and Anti-PLA2R were negative. He was treated as possibly evolving lupus nephritis and thus started on steroids. Subsequent serum electrophoresis results showed IgM Kappa 4.9g/L mid-gamma region with marked immunoparesis. Bone marrow aspiration and trephine biopsy (BMAT) showed 9% of plasma cells. CT scan was done to look for other possible causes especially lymphoma and incidentally showed bilateral renal vein thrombosis. He was diagnosed to have Waldenstrom's macroglobulinaemia. Second renal biopsy with electron microscopy was done as he failed to respond to initial treatment. The electron microscopy showed a diffuse membranous pattern with accompanying focal active & sclerosing lesions. Based on the clinical history, morphological features (focal active and sclerosing lesions), immunofluorescence (C1q positivity) and immunohistochemistry findings(IgG4 negativity), a secondary form of membranous glomerulonephritis was diagnosed. The steroids were tapered and the haematology team decided to start chemotherapy for him.

Discussion

In Waldentrom macroglobulinemia, the monoclonal IgM produced by the malignant B cells not only can infiltrate hematopoietic tissues causing pancytopenia, lymphadeopathy, hepatomegaly and splenomegaly but also can react as an autoantibody and deposit in extracellular spaces of the kidneys, causing extra-haematological symptoms. This case highlights the importance of comprehensive evaluation in nephrotic syndrome of unknown etiology and the needs for repeat renal biopsy in suspicious cases.

Keywords：Nephrotic syndrome, glomerular disease, waldenstrom's macroglobulinaemia, membranous nephropathy, steroid resistant nephrotic syndrome

Poster Presentation：Glomerular Diseases
Poster No.：C0515
Abstract Submission No.：APCN20250969

The Outcome Of Pregnancy In Biopsy Proven Lupus Nephritis. A Retrospective Case Series From A Single Centre.

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Abstract

Introduction

The management of Lupus Nephritis (LN) during pregnancy is challenging. Tailoring treatment strategies that balance disease control and maternal-fetal safety is imperative.

Methods

This is a retrospective case series of two biopsy proven LN in pregnancy from 2024 to 2025 at Hospital Selayang. We report the outcome for both mother and baby until 8 weeks post-partum.

Results

Case 1

30-year-old lady presented with history of malar rashes, photosensitivity and frothy urine 7 years ago. Her initial serum ANA positive, speckled 1:640, low C3 and C4, Urine Protein Creatinine Ratio (PCR) 508mg/mmol. Kidney biopsy revealed LN Class IV with high Activity Index. She received Mycophenolate mofetil following which she only achieved partial remission due to compliance issue. She was then given 6 doses of IV Cyclophosphamide with a cumulative dose of 3g. She conceived after 2 years in complete remission. During booking visit at 8 weeks of pregnancy she was normotensive, full blood count (FBC), liver function, kidney function were normal but her urine PCR was 150mg/mmol. Anti-dsDNA 39 IU/ml, C3 and C4 normal. Anti-Ro, Anti-La were negative. Azathioprine at 1.5mg/kg/day was initiated along with Prednisolone and Hydroxychloroquine. She achieved complete remission a month later. She needed methyldopa for pregnancy induce hypertension (PIH) and kidney function remained static. She delivered at term via emergency caesarean section for fetal distress. Infant birth weight 3.5kg, APGAR score 8/9. No neonatal lupus with appropriate weight gain on breast-milk feeding.

Case 2

29-year-old pregnant lady presented with nephrotic syndrome, skin rashes, joint pains at 26 weeks of pregnancy. FBC and serum creatinine normal range. Serum ANA positive, speckled 1: 560, low C3 and C4, Urine PCR 308mg/mmol. Serum anti-dsDNA 40 IU/ml, C3 and C4 normal range, anti-Ro, anti- RNP, anti-Sm were positive. She was empirically initiated on Tacrolimus, Prednisolone and Hydroxychloroquine. Antenatally she was normotensive and kidney function remain static. She delivered at term via spontaneous vagina delivery. Infant birth weight 2.5kg, APGAR score 9/9. No neonatal lupus. Infant on exclusive breast-milk feeding with appropriate weight gain. Kidney biopsy performed at 6 weeks post partum revealed LN Class V. Azathioprine 1.5mg/kg/day was added at post-partum 6 weeks for LN in partial remission and achieved complete remission in 1 month.

Conclusion

Personalized management strategy is the key in treating pregnant mothers with LN. Azathioprine is effective in achieving full remission for LN within a month during the antenatal and post-natal period.

Keywords：Lupus nephritis, Pregnancy, Kidney biopsy, Maternal, Fetal.

Poster Presentation：Glomerular Diseases
Poster No.：C0516
Abstract Submission No.：APCN20250979

Rituximab for Treatment of Tubulointestitial Nephritis with Uveitis (TINU)

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Abstract
A 17-year-old male with no prior medical history presented with acute renal failure and severe azotemia (creatinine 10.14 mg/dL; BUN 82 mg/dL). Three weeks prior, he had started pantoprazole for diffuse abdominal pain. Hemodialysis was initiated, and a kidney biopsy revealed diffuse tubulointerstitial nephritis with predominant lymphocytic infiltration, sparse plasma cells and segmented leukocytes. Eosinophils and granulomas were absent. Immunofluorescence showed no immune complex deposition, and immunohistochemical stains for polyoma virus, HSV, CMV, and adenovirus were negative. Staining for CD138, IgG, and IgG4 revealed focal areas with up to 10 IgG4-positive plasma cells per high power field, raising concern for IgG4-related tubulointerstitial nephritis. However, storiform fibrosis, hypocomplementemia, and immune complex deposits were absent, and there was no radiologic evidence of systemic IgG4-related disease.
Serologic evaluation revealed an elevated serum IgG4 (416 mg/dL; normal: 2–170) and a small IgG lambda monoclonal spike (0.2 g/dL), without evidence of amyloidosis or lymphoproliferative disease. High-dose prednisone led to partial improvement in renal function, but it was discontinued due to steroid-induced psychosis. The patient was transitioned to mycophenolate. After three months, creatinine remained at 3.0 mg/dL, and signs of tubular dysfunction persisted (hypophosphatemia, hypokalemia, elevated urinary β2-microglobulin >10,000 mcg/L, and proteinuria). Repeat kidney biopsy showed ongoing interstitial nephritis with T-cell–predominant infiltrates and rare eosinophils. Glomeruli were preserved with only focal periglomerular fibrosis.
The patient subsequently developed uveitis. Bone marrow biopsy showed normocellular marrow with rare non-necrotizing granulomas and no clonal plasma cells, raising suspicion for TINU (tubulointerstitial nephritis and uveitis) syndrome. Based on evolving clinical findings, a diagnosis of TINU was established. Rituximab was initiated, resulting in gradual improvement in renal function. After 24 months of treatment, the patient achieved complete remission, with creatinine decreasing to 1.6 mg/dL and creatinine clearance improving to 82 mL/min. He remains clinically stable on weekly methotrexate and folic acid.
This case underscores the diagnostic overlap between IgG4-related disease and TINU. Although early findings raised suspicion for IgG4-related nephropathy, the clinical evolution and development of uveitis supported TINU. To our knowledge, this represents a rare case of successful long-term treatment of TINU with rituximab, which is not a standard therapy for this condition. Further studies may be warranted to evaluate its role in refractory or atypical TINU presentations.

Keywords：TINU, tubulointerstitial nephritis with uveitis, rituximab

Poster Presentation：Glomerular Diseases
Poster No.：C0517
Abstract Submission No.：APCN20250988

Mesangial Expansion as a Predictor of Renal Outcome in IgA Nephropathy

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Abstract

Background:
IgA nephropathy (IgAN) is the most common primary glomerulonephritis worldwide and a leading cause of end-stage renal disease (ESRD). While the Oxford classification has provided key prognostic histologic parameters, the clinical significance of mesangial expansion remains under-explored.

Objective:
To evaluate the association between mesangial expansion and renal outcomes in patients with biopsy-proven IgAN.

Methods:
We conducted a retrospective cohort study in single medical center involving 378 patients diagnosed with IgAN between 2011 and 2023. Mesangial expansion was assessed by light microscopy, defined morphologically as a mesangial area greater than the capillary lumen. Patients were categorized into expansion and non-expansion groups. The primary composite outcome was progression to eGFR <15 mL/min/1.73 m², dialysis, or kidney transplantation. We used Kaplan–Meier, univariable/multivariable logistic regression. Kaplan–Meier survival analysis was performed for subgroups defined by mesangial expansion and M score (M0 vs. M1).

Results:
Among the 378 patients (mean age 42.2 years), 109 (28.8%) demonstrated mesangial expansion. Kaplan-Meier analysis revealed significantly worse renal survival in the expansion group (log-rank $p < 0.01$). In multivariable Cox analysis, mesangial expansion remained an independent predictor of adverse renal outcome (adjusted hazard ratio [aHR]: 2.96; 95% CI: 1.25–7.02; $p = 0.014$). Bedsides, mesangial expansion, especially when combined with M1 lesions, was associated with significantly worse renal survival ($p = 0.007$).

Conclusion:
Mesangial expansion is a robust histopathological predictor of renal prognosis in IgAN. Incorporation of this parameter into routine biopsy evaluation may enhance risk stratification and inform treatment strategies in patients with IgAN.

Keywords：IgAN, Mesangial expansion

Poster Presentation：Glomerular Diseases
Poster No.：C0518
Abstract Submission No.：APCN20251013

Sclerostin in Glomerulonephritis: A New Link Between Bone and Kidney Disease

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Abstract

BACKGROUND: Sclerostin is a protein secreted by osteocytes that inhibits the Wnt/β-catenin signaling pathway, leading to osteoporosis. Due to its proinflammatory cytokine properties, sclerostin may play a role in damage to the glomeruli and tubules. Studies examining the effects of sclerostin in glomerulonephritis patients are limited.

OBJECTİVES: The aim of this study is to measure serum sclerostin levels in glomerulonephritis patients and evaluate its relationship with glomerulonephritis.

DESIGN AND SETTING: Our study is a cross-sectional, case-control study conducted between September 2024 and December 2024. Serum sclerostin levels were measured using enzyme-linked immunosorbent assay (ELISA) in 49 glomerulonephritis patients and 30 healthy participants. The relationship between serum sclerostin levels and demographic features, as well as biochemical data, was analyzed in glomerulonephritis patients.

RESULTS: Serum sclerostin levels were significantly higher in glomerulonephritis patients (10,62 ng/ml; 2,7-22,07) compared to the healthy control group (7,14 ng/ml; 2,36-19,62) ($p<0,001$). Significant correlations were found between sclerostin and estimated glomerular filtration rate (eGFR), creatinine, proteinuria, parathyroid hormone (PTH), vitamin D, serum calcium, and C-reactive protein (CRP).

CONCLUSİONS: This study is a rare investigation showing that serum sclerostin is associated with proteinuria and eGFR in glomerulonephritis patients. It suggests that sclerostin could be a potential biomarker in the diagnosis and treatment of glomerulonephritis, offering new targets for therapeutic strategies.

Keywords：Sclerostin. Glomerulonephritis. Biomarker. Gloerular filtration rate. Proteinuria.

Poster Presentation：Glomerular Diseases
Poster No.：C0519
Abstract Submission No.：APCN20251023

Soluble biomarkers in IgA nephropathy: analysis of baseline data from the phase 2 trial of ravulizumab (SANCTUARY)

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Abstract

Background:
In IgA nephropathy (IgAN), immune complex formation and deposition leads to complement activation, culminating in complement terminal pathway-mediated inflammation and kidney damage. Validated soluble biomarkers of disease activity and pathophysiology are needed in IgAN. This analysis aimed to characterize biomarkers using baseline data from the ph2 trial (NCT04564339) of ravulizumab (RAV) (a complement C5 inhibitor) and from healthy adult normal donors (ND).

Methods:
Soluble biomarker levels in 60 pts with IgAN at baseline in SANCTUARY and from a validation cohort of up to 25 ND were compared. Biomarkers were measured from spot urine samples: sC5b-9 and factor Ba (complement pathway products), CD163 (macrophage renal infiltration marker), KIM-1 (specific marker of renal proximal tubule injury), and NGAL (marker of renal tubule injury). A stringent sample collection protocol was implemented for all samples and immunoassays were developed and validated to FDA guidance (Bioanalytical Method Validation, 2018). Biomarkers were normalized to urine creatinine (Cr).

Results:
Baseline levels of 4 of 5 urine biomarkers were significantly elevated in pts with IgAN vs ND: sC5b-9/Cr, Ba/Cr, CD163/Cr, and KIM-1/Cr ($p<0.0001$ for all) (Table). These 4 biomarkers were also elevated above the upper limit of normal (observed mean for ND + 2 SD) in >50% of pts with IgAN, at baseline: sC5b-9 elevated in 93% of pts, CD163 in 82%, Ba in 65%, and KIM-1 in 62%.

Conclusion:
Soluble urine biomarkers of complement activation, inflammation, and proximal tubule injury were detected at significantly higher levels in this cohort of patients with IgAN vs ND, and the vast majority (56/60) had elevated terminal complement activation (urine sC5b-9). These results provide insights into the pathophysiology of IgAN and suggest the potential clinical utility of soluble biomarkers in this heterogenous disease.

"This abstract was also submitted for the ASN Kidney Week 2025 congress. By submitting the abstract to APCN x TSN 2025, abstract authors declare that re-submitting the abstract is permitted by the organizers of the previous meeting".

Keywords：IgA nephropathy; Complement; Clinical trial

Table. Soluble urine biomarkers in patients with IgA nephropathy and normal donors

Biomarker	Biomarker levels at baseline[a] median (Q1, Q3)		p-value[b]	Patients with biomarker levels >ULN[c] at baseline n/N (%)
	Patients with IgAN	Normal donors		
sC5b-9/Cr (ng/mg Cr)	n=60 **22.0** (7.5, 42.9)	n=15 **0.5** (0.3, 1.1)	<0.0001	56/60 (93.3)
CD163/Cr (ng/mg Cr)	n=60 **5.3** (3.1, 8.8)	n=25 **0.8** (0.5, 1.1)	<0.0001	49/60 (81.7)
Ba/Cr (ng/mg Cr)	n=60 **178.0** (18.0, 373.4)	n=24 **2.5** (1.6, 6.0)	<0.0001	39/60 (65.0)
KIM-1/Cr (ng/mg Cr)	n=60 **2.0** (1.3, 2.8)	n=25 **0.8** (0.5, 1.1)	<0.0001	37/60 (61.7)
NGAL/Cr (ng/mg Cr)	n=60 **10.0** (6.8, 15.2)	n=25 **6.5** (3.4, 16.3)	0.10	6/60 (10.0)

CI, confidence interval; Cr, creatinine; ULN, upper limit of normal.

[a]Baseline = Day 1 visit (pre-dose); when no Day 1 data available, screening visit timepoint used. [b]p-values calculated by Wilcoxon rank sum test for differences. [c]ULN = Observed mean for normal donors + 2 standard deviations.

Note: All urine samples are spot collection (time of void not known); both normal donors and patient assay values were normalized to respective time-matched urine creatinine values.

Poster Presentation：Glomerular Diseases
Poster No.：C0520
Abstract Submission No.：APCN20251027

Scedosporium apiospermum Infective Endocarditis in a Patient with ANCA-Associated Vasculitis: A Rare and Fatal Opportunistic Infection

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Abstract

Background: ANCA-associated vasculitis (AAV) often requires high-dose immunosuppressive therapy, which can make patients more vulnerable to unusual infections. Among them, Scedosporium apiospermum is a rare but serious fungal pathogen. While this fungus is occasionally seen in immunocompromised individuals, infective endocarditis caused by it is extremely uncommon and challenging to treat.

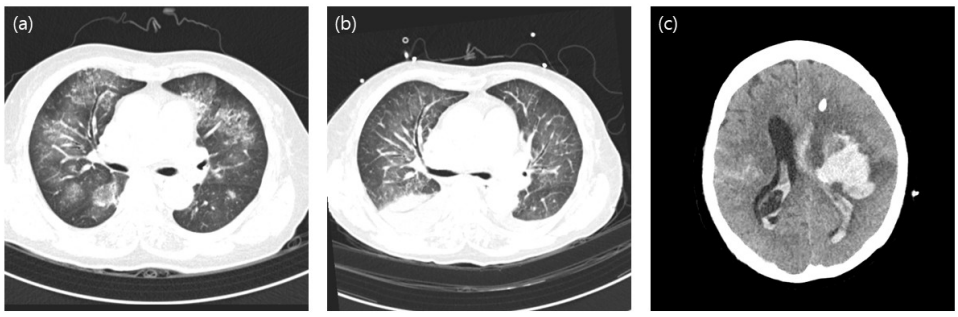
Case Presentation: We describe a 67-year-old woman who came to the hospital with hemoptysis and acute kidney injury. Lab work showed a high p-ANCA level (169 U/mL), proteinuria, and signs of alveolar hemorrhage on chest CT, pointing toward microscopic polyangiitis (Fig1-(a)). She was started on IV steroids, oral cyclophosphamide, and plasma exchange. Her condition initially improved, but three months later, she returned with shortness of breath, worsening kidney function, and pulmonary edema (Fig1-(b)). An echocardiogram revealed a large vegetation on the anterior mitral leaflet, causing severe mitral regurgitation. She underwent emergency mitral valve surgery. Although broad-spectrum antibiotics were initially administered, cultures from the resected valve grew S. apiospermum. Fluconazole was replaced with voriconazole. Unfortunately, her condition worsened: she developed a brain hemorrhage (Fig1-(c)), needed a temporary pacemaker for complete AV block, and progressed to multi-organ failure. She passed away on hospital day 110.

Discussion: This case serves as a reminder of how rare fungal pathogens can cause devastating disease in immunocompromised patients. S. apiospermum often resists standard antifungals, and in the absence of routine susceptibility testing, diagnosis is frequently delayed. When endocarditis is involved, outcomes are generally poor despite aggressive therapy.

Conclusion: In patients receiving immunosuppressive treatment, clinicians should be aware of the risk of rare fungal infections. Early recognition of S. apiospermum endocarditis and use of appropriate

Keywords：AAV, vasculitis, fungal infective endocarditis

Figure 1. Radiologic findings
(a) HRCT findings performed at the time of diagnosis of ANCA vasculitis, which were pulmonary hemorrhage in both lungs.
(b) HRCT taken during the second hospitalization. The diffuse peribronchial consolidation and GGA of both lungs previously seen showed improvement, but new pulmonary edema and pleural effusion were found.
(c) Acute ICH in the left basal ganglia as confirmed by brain CT.



Poster Presentation：Glomerular Diseases
Poster No.：C0521
Abstract Submission No.：APCN20251029

Real-World Analysis of Efficacy and Safety of Nefecon Combined with Low-Dose Corticosteroids in Reducing Proteinuria Over Three Months in High-Risk Progressive IgA Nephropathy

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Abstract
Background:
IgA nephropathy (IgAN) is the most common primary glomerulonephritis, with up to 40% of patients progressing to end-stage kidney disease (ESKD) within 20 years. Proteinuria is a key predictor of disease progression. Nefecon, a targeted-release budesonide formulation, has shown efficacy in reducing proteinuria and preserving renal function in clinical trials. Updated management guidelines prioritize two core objectives: reducing pathogenic IgA and IgA immune complex formation to mitigate glomerular injury, and managing the resultant chronic kidney disease caused by nephron loss. Therapies targeting reduction in galactose-deficient IgA1 (Gd-IgA1), such as Nefecon, are recommended. For patients with severe glomerular inflammation, adjunct glucocorticoid therapy may be considered following a thorough risk-benefit assessment.However, real-world evidence on the efficacy of these approaches in high-risk IgAN patients remains limited.
Objective:
To evaluate the efficacy and safety of Nefecon combined with low-dose glucocorticoids in reducing proteinuria within three months in high-risk IgAN patients under real-world conditions.
Methods:
This single-center, retrospective study included eight biopsy-confirmed IgAN patients with ≥1 g/24 h proteinuria, treated between January 2023 and February 2025. All received Nefecon 16 mg/day plus oral methylprednisolone (initial dose 0.4 mg/kg/day), tapered by 4 mg every two weeks. Efficacy was assessed by changes in proteinuria and eGFR; safety by adverse event monitoring.
Results:
Among eight patients (62.5% male, median age 39 years), all had moderate-to-severe histologic disease (Lee grade III–V). Baseline mean eGFR was 59.93 ± 22.46 mL/min/1.73 m². After a median follow-up of 92 days, eGFR increased to 64.06 ± 19.23 mL/min/1.73 m² ($p = 0.142$). Proteinuria decreased significantly by 49.5%, from 2.93 ± 1.39 g/24 h to 1.48 ± 1.04 g/24 h ($p = 0.029$). No significant changes were observed in serum glucose, calcium, phosphorus, urea, or creatinine. No serious treatment-related adverse events were reported.
Conclusion:
This real-world study demonstrates that a three-month course of budesonide (Nefecon) combined with low-dose corticosteroids significantly reduces proteinuria and maintains stable renal function in high-risk patients with IgA nephropathy (IgAN), with a favorable safety profile. These findings support the efficacy of this budesonide-based combination regimen as a promising therapeutic option for the early management of high-risk IgAN. However, further confirmation in larger-scale studies is warranted.

Keywords：IgA nephropathy · Nefecon · low-dose glucocorticoids

Poster Presentation：Glomerular Diseases
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Abstract Submission No.：APCN20251086

Association Between Glomerulosclerosis Severity and eGFR Decline in IgA Nephropathy

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Abstract
Background:
While blood pressure and proteinuria are well-established prognostic factors in immunoglobulin A nephropathy (IgAN), the relationship between histopathological findings—specifically the extent of glomerulosclerosis—and the rate of estimated glomerular filtration rate (eGFR) decline remains unclear. This study aimed to explore the potential correlation between renal histopathology, assessed through image analysis software, and the slope of eGFR reduction.
Methods:
The mesangial matrix fraction in patients with IgAN was quantified using ImageJ, an image analysis software developed by the National Institutes of Health (Bethesda, MD, USA). We investigated the relationships between the mesangial matrix fraction and various clinical parameters, including the slope of eGFR decline, changes in urinary protein levels, urinary erythrocyte counts, urinary N-acetyl-β-D-glucosaminidase (NAG), and urinary β2-microglobulin (β2MG). All patients were receiving standard treatment for IgAN, including corticosteroids and other supportive therapies.
Results:
A significant inverse correlation was found between the mesangial matrix fraction and eGFR in patients with IgA nephropathy. However, no significant associations were observed between the mesangial matrix fraction and changes in proteinuria, urinary erythrocyte count, urinary NAG, or urinary β2MG.
Conclusions:
Quantitative analysis of the mesangial matrix fraction using imaging software may serve as a useful predictor of eGFR decline in patients with IgAN.

Keywords：IgAN, eGFR, ImageJ, mesangial matrix fraction

Poster Presentation：Glomerular Diseases
Poster No.：C0523
Abstract Submission No.：APCN20251124

Approach to the categorization of C3-dominant glomerulonephritis with MPGN feature: A retrospective clinicopathological observational study

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Abstract
Introduction: Membranoproliferative glomerulonephritis (MPGN) is a morphological diagnosis based on renal pathology, arising from a spectrum of underlying etiologies, including genetic disorders, infections, collagen diseases, and neoplastic conditions. Based on immunofluorescence (IF), predominant of complement 3 (C3) deposition have been distinguished from immunoglobulin-positive MPGN. C3-dominant MPGN is currently classified as C3 glomerulopathy (C3G). Although C3G is often associated with inherited complement regulatory abnormalities, it can occasionally be diagnosed in older patients. Therefore, we comprehensively analyzed MPGN and focused on C3-dominant GN with MPGN feature.
Methods: We conducted a cross-sectional review of adult patients diagnosed as MPGN at our hospital since 2014, and carefully observed clinicopathological findings. Cases with lupus nephritis or IgA vasculitis presenting MPGN were excluded.
Results: During the study period, 28 (2.8%) out of 1001 cases were diagnosed as MPGN based on LM. IF analysis identified 10 cases of C3G. EM in these cases did not reveal dense deposit disease (DDD) patterns, indicating a diagnosis of C3 glomerulonephritis (C3GN). Among C3GN cases, 7 patients exhibited decreased serum C3 levels, with one also showing reduced CH50. Other 3 patients had normal C3 levels; notably, 2 of them had detectable monoclonal immunoglobulins. The remaining 17 cases of MPGN were classified as immune complex–mediated MPGN with IgG deposition, which were further subclassified as follows: cryoglobulinemic GN (6 cases, including one associated with HCV), proliferative GN with monoclonal immunoglobulin deposits (PGNMID) (5 cases), infection-related GN (2 cases), and idiopathic MPGN without underlying disease (4 cases). A significant trend in gender distribution across diagnostic categories was not confirmed. However, age distribution varied: patients with C3GN and cryoglobulinemic GN were mostly under 50, while all PGNMID occurred in patients over 50. Among the C3GN group, nearly all patients under 50 had low C3 levels, except one with monoclonal IgGκ. Interestingly, half of the C3GN patients over 50 had normal C3 levels.
Conclusion: In our analysis, 35% of MPGN cases were classified as C3GN, with age-associated variations in complement levels, suggesting heterogeneity in pathogenesis among patients. Meanwhile, PGNMID presenting with MPGN was observed predominantly in older patients. Notably, recent reports have emphasized cases that were ultimately diagnosed as PGNMID after the detection of masked IgG deposits. Therefore, in cases of C3GN in older patients, it might be critical to consider the possibility of masked IgG and to perform IgG staining on paraffin-embedded tissue to reliably exclude PGNMID.

Keywords：MPGN. C3 glomerulopathy, masked IgG, PGNMID

Poster Presentation：Glomerular Diseases
Poster No.：C0524
Abstract Submission No.：APCN20251131

Adult Podocytopathies: Clinicopathologic Features, Treatment Response, and Outcomes

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Abstract

Background: Podocytopathies, including minimal change disease (MCD) and focal segmental glomerulosclerosis (FSGS), are glomerular disorders characterized by podocyte injury, commonly presenting as nephrotic syndrome. Podocytopathies are well-characterized in children; however, the data on adults, including clinical features, treatment responses, and long-term outcomes, remain limited.

Objectives: To evaluate the clinical profile, histopathological patterns, treatment responses, and outcomes in adult patients with biopsy-proven podocytopathy.

Methods: We conducted a single-center retrospective observational study of patients aged ≥14 years with biopsy-confirmed podocytopathy between January 2019 and December 2024.

Results: Podocytopathy was identified in 218 (28.6%) out of 763 native kidney biopsies, including 73 (33.5%) with MCD and 145 (66.5%) with FSGS. The histologic subtypes of FSGS included not otherwise specified (n=109, 75.2%), tip variant (n=23, 15.9%), perihilar (n=11, 7.6%), and apical lesions (n=2, 1.4%). Electron microscopy, performed in 213 patients (97.7%), exhibited diffuse podocyte foot process effacement.

The mean age at presentation was 29.63±13.86 years, with 136 patients (62.4%) being male. Hypertension was present in 23 patients (10.6%), while one patient each (0.5%) had diabetes with hypertension and diabetes alone. The median duration of edema prior to presentation was 3 months (IQR: 1–5.25 months). Baseline laboratory investigations showed mean eGFR of 90.4±41.0 mL/min/1.73 m², mean serum albumin of 2.15±0.85 g/dL, and mean 24-hour urinary protein excretion of 6.09±3.36 g. Microscopic hematuria was observed in 39 patients (26.9%).

Of the 211 patients (96.8%) who received oral corticosteroids as first-line therapy, 128 (60.7%) achieved complete remission and 49 (23.3%) achieved partial remission. MCD was significantly more likely to achieve remission compared to FSGS (p = 0.013). Relapse occurred in 51 patients (28.8%) following initial remission, with 37 being infrequent relapsers and 14 frequently relapsing or steroid-dependent. Second-line immunosuppressive therapy was administered to 36 patients: cyclophosphamide (n=25), calcineurin inhibitors (CNIs; tacrolimus, n=8; cyclosporine, n=1), and rituximab (n=2).

During a median follow-up of 11 months (IQR: 7-19.5), 191 patients (87.6%) achieved remission (133 complete, 58 partial). A total of 54 patients (24.8%) failed to achieve remission, including 3 who progressed to end-stage renal disease and 1 who died. No clinical or biochemical parameters were significantly associated with non-remission. Adverse events included infections in 23 patients (10.5%), hyperglycemia in 16 (7.3%), and avascular necrosis in 3 (1.4%).

Conclusions: FSGS is the most common podocytopathy in adults, typically presenting with nephrotic syndrome and preserved renal function. Steroids are effective, with high remission rates, though relapses are common. Progression to ESRD is rare within the study period.

Keywords：Podocytopathy, Minimal Change Disease, Focal Segmental Glomerulosclerosis

Poster Presentation：Glomerular Diseases
Poster No.：C0525
Abstract Submission No.：APCN20251151

Projected Burden of CKD due to Glomerulonephritis Attributable to High Sodium Intake in Indonesia: A 2030 and 2050 Forecast

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Abstract

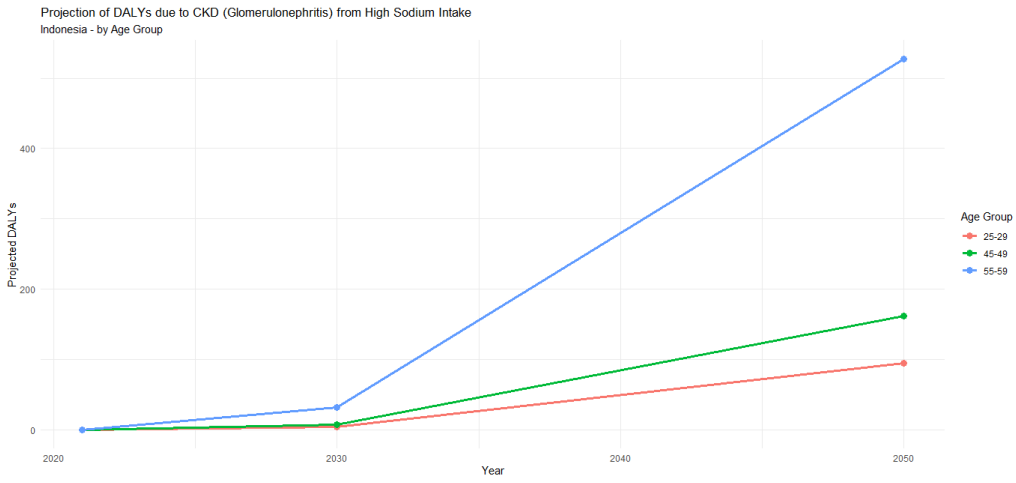
Chronic kidney disease (CKD) due to glomerulonephritis is an emerging health challenge in aging populations. High sodium intake is a modifiable dietary risk factor that contributes to glomerular injury and long-term renal decline. Understanding future disease burden is crucial to guide preventive strategies. To project the future burden of CKD due to glomerulonephritis attributable to high sodium intake in Indonesia through 2030 and 2050, based on recent Global Burden of Disease (GBD) data.

DALYs from the Global Burden of Disease (GBD) Study (2017–2021) served as baseline data. Projection to the years 2030 and 2050 was conducted in R using exponential growth modeling based on compound annual growth rate (CAGR) for each age group (25–29, 45–49, 55–59 years).

In 2021, DALYs attributable to high sodium intake were negligible in the 25–29 age group and modest in the 45–49 group (4 DALYs), but substantially higher in the 55–59 group (95 DALYs). Projections suggest that by 2030, the burden in the 55–59 group could increase to 162 DALYs, and by 2050, reach 527 DALYs—a 5.5-fold rise. For the 45–49 group, DALYs are expected to double to 8 in 2030 and increase eightfold to 32 in 2050. The 25–29 group is projected to remain unaffected unless earlier dietary exposure becomes clinically manifest in later years.

The projected increase in CKD burden attributable to sodium intake among older Indonesians highlights a critical window for intervention. Public health efforts should prioritize sodium reduction and early kidney screening among middle-aged adults to mitigate future renal disease burden.

Keywords：Chronic kidney disease (CKD), Glomerulonephritis, Sodium intake, Disease burden projection, Disability-adjusted life years (DALYs)



Poster Presentation：Glomerular Diseases
Poster No.：C0526
Abstract Submission No.：APCN20251179

IgG Subclass Profiling of Anti-PLA2R1 Antibodies Improves Detection and Monitoring of Membranous Nephropathy

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Abstract

Background:
Anti-PLA2R1 autoantibodies are central to the diagnosis of membranous nephropathy (MN), with IgG4 being the dominant subclass. However, a subclass-based assessment strategy may provide superior sensitivity and clinical relevance, particularly in cases with negative total anti-PLA2R1 IgG results.

Methods:
We analyzed serum samples from 129 patients with biopsy-proven MN, including 76 PLA2R-positive and 38 triple-negative cases (negative for PLA2R, THSD7A, and NELL-1 by glomerular staining). Total anti-PLA2R1 IgG levels were measured using a commercial ELISA, and IgG1, IgG3, and IgG4 subclasses were quantified using in-house assays to explore the added diagnostic value of subclass profiling.

Results:
In PLA2R-positive MN, 80.3% of patients were positive for total anti-PLA2R1 IgG. Subclass analysis increased detection rates substantially: 89.5% for IgG3 and 98.7% for IgG4. Importantly, 93.3% of total IgG-negative patients remained positive for IgG4, while none had detectable IgG1 or IgG3. Among total IgG-positive patients, subclass positivity was 45.9% (IgG1), 96.7% (IgG3), and 100% (IgG4). Overall, 98.7% of PLA2R-positive cases were subclass-positive.

In triple-negative MN, subclass testing identified additional cases missed by total IgG: 18.9% were positive for IgG3 and 29.7% for IgG4, increasing the overall subclass detection rate to 37.8%. Among those negative for total IgG, 32.4% remained subclass-positive, highlighting the diagnostic advantage of this approach.

Conclusion:
A subclass-based assessment strategy using IgG3 and IgG4 detection significantly improves the serological sensitivity for membranous nephropathy. This approach identifies otherwise undetected cases, particularly in seronegative or triple-negative patients, and may serve as a valuable adjunct in both diagnosis and disease monitoring.

Keywords：membranous nephropathy, PLA2R1, IgG subclass, seronegative MN, subclass-based diagnosis, autoimmune kidney disease

Poster Presentation：Glomerular Diseases
Poster No.：C0527
Abstract Submission No.：APCN20251185

Prevalence of Anti-Nephrin Antibodies in Minimal Change Disease: A Biopsy-Cohort Study in Taiwan with Clinical Correlations

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Abstract

Background:
Minimal change disease (MCD) is a leading cause of nephrotic syndrome and is increasingly recognized as an immune-mediated glomerulopathy. Circulating anti-nephrin antibodies have recently been proposed as potential pathogenic and diagnostic biomarkers in a subset of MCD patients. However, data from Asian populations, particularly Taiwan, remain limited.

Methods:
Between August 2016 and July 2024, we prospectively collected serum samples from patients undergoing renal biopsy at Chang Gung Memorial Hospital. Among these, 100 patients with biopsy-confirmed MCD and nephrotic-range proteinuria were enrolled. Circulating anti-nephrin antibodies were detected using immunoprecipitation followed by western blotting. Baseline clinical parameters were compared between antibody-positive and antibody-negative groups.

Results:
Anti-nephrin antibodies were detected in 55% (55/100) of patients. Compared to the antibody-negative group (n = 45), antibody-positive patients had significantly higher serum creatinine levels (1.43 ± 1.46 vs. 0.87 ± 0.72 mg/dL, $p = 0.015$), lower serum albumin levels (1.96 ± 0.36 vs. 2.33 ± 0.58 g/dL, $p < 0.001$), and more severe proteinuria (18.7 ± 11.9 vs. 10.5 ± 9.1 g/day, $p < 0.001$). No significant differences were observed in age, sex, serum cholesterol, or triglyceride levels.

Conclusion:
In this biopsy-based Taiwanese cohort, more than half of MCD patients had detectable circulating anti-nephrin antibodies. Their presence was associated with greater disease severity, including heavier proteinuria, lower serum albumin, and impaired renal function. These findings provide region-specific evidence supporting the clinical relevance of anti-nephrin antibodies as potential biomarkers for disease activity in MCD.

Keywords：minimal change disease, nephrotic syndrome, anti-nephrin antibody, immunoprecipitation, Taiwan, biomarker

Poster Presentation：Glomerular Diseases
Poster No.：C0528
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When Lupus Nephritis Meets The Nervous System: A Case Report of Severe Systemic Lupus Erythematosus Flare Treated Through Multidisciplinary Strategy

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Abstract

Background

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease which can affect multiple vital organs, with lupus nephritis (LN) and neuropsychiatric SLE (NPSLE) as two severe manifestations contribute significantly to morbidity and mortality. Both manifestations indicate high disease activity and require aggressive immunosuppressive treatment. However, the therapeutic approach becomes complex when LN and NPSLE occur simultaneously, due to the limited availability of specific biomarkers for NPSLE, the varying effectiveness of immunosuppressive regimens in different organs, and the increased risk of systemic toxicity. This case report highlights the critical role of a multidisciplinary strategy in addressing these diagnostic and therapeutic dilemmas, emphasizing the importance of individualized care in real-world clinical settings.

Case Illustration

A 24-year-old woman with a known history of SLE was admitted to the emergency room at Moewardi Hospital with a chief complaint of worsening dyspnea, altered mental status, and generalized fatigue. Clinical assessment and laboratory investigations revealed signs of active LN, including massive proteinuria and hematuria, as well as neurological manifestations consistent with NPSLE. Serological tests were strongly positive for ANA, anti-dsDNA, anti-Sm/RNP, and anti-Ro antibodies, with a high MEX-SLEDAI score of 19. The patient was diagnosed with a severe SLE flare involving renal and central nervous system manifestations, complicated by bilateral pneumonia, heart failure (NYHA IV), and impending type 2 respiratory failure. She received high-dose intravenous methylprednisolone pulse therapy, antibiotics, oxygen supplementation, and multidisciplinary supportive care. Gradual clinical improvement was observed.

Discussion

This case highlights the complexity of diagnosing and managing SLE when it presents with simultaneous involvement of the kidneys and central nervous system. The patient exhibited both LN and NPSLE, a rare but life-threatening combination. These overlapping manifestations contributed to acute respiratory failure, likely due to pulmonary edema, bilateral pneumonia, and possible central nervous system involvement. Early recognition and aggressive treatment using high-dose corticosteroids and supportive intensive care were critical to prevent further deterioration. The case underscores the importance of a multidisciplinary approach in managing severe SLE flares, especially those involving vital organs, to improve patient outcomes.

Conclusion
The simultaneous involvement of the kidneys and central nervous system in SLE represents a critical clinical scenario that demands urgent, comprehensive care. In this case, early diagnosis, timely initiation of high-dose immunosuppressive therapy, and a coordinated multidisciplinary approach were essential in stabilizing the patient and improving outcomes.

Keywords：Systemic lupus erythematosus, Lupus nephritis, Neuropsychiatric SLE, Multidisciplinary care, Immunosuppressive therapy

Poster Presentation：Glomerular Diseases
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In Silico Identification of TRPC5 Inhibitors for Focal Segmental Glomerulosclerosis: Molecular Docking and Dynamics Simulation Study

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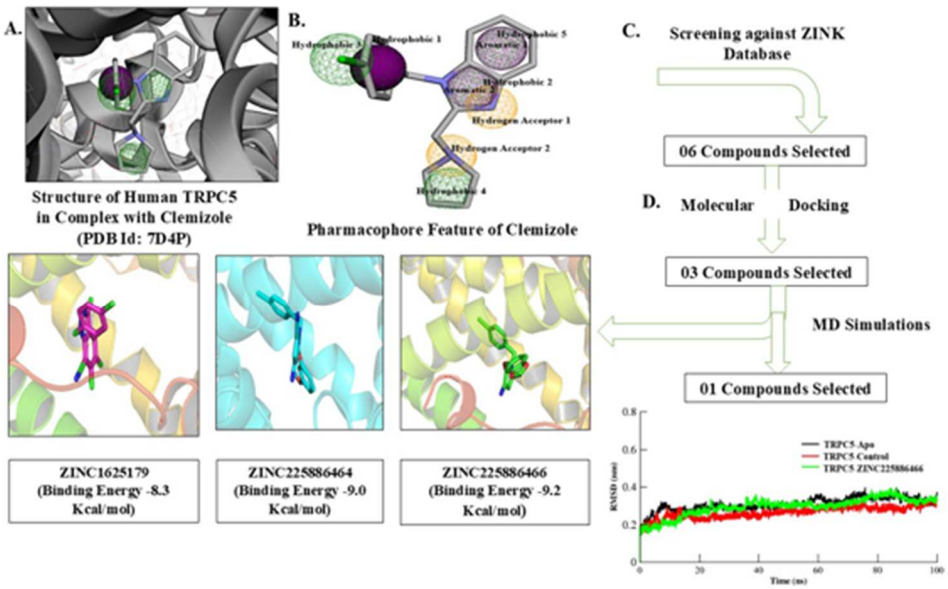
Abstract
Background: Focal segmental glomerulosclerosis (FSGS) is a kidney disease that gets worse with time and is marked by damage to podocytes and proteinuria. The Transient Receptor Potential Cation Channel Subfamily C Member 5 (TRPC5) is very important for podocyte dysfunction, which makes it a good target for treatment.
Methodology: Molecular docking and molecular dynamics (MD) simulations were used to find new TRPC5 inhibitors. We used the crystal structure of human TRPC5 with clemizole (PDB ID: 7D4P) as a model for the structure. A virtual screening of the ZINC database was done based on clemizole's binding site. Six hits that were structurally relevant were then docked into the TRPC5 active site. Based on how well they bind and how they interact with one other, the top three choices were put through 100 ns MD simulations to make sure they were still good.
Results: The three lead compounds, ZINC1625179, ZINC225886464, and ZINC225886466, had strong docking scores of −8.3, −9.0, and −9.2 kcal/mol, respectively. They also replicated clemizole's binding conformation in the TRPC5 binding pocket. ZINC225886466 had the best dynamic stability of the group. The RMSD analysis showed that the protein-ligand conformations stayed stable during the simulation. Other tests, such as radius of gyration (Rg) and hydrogen bonding patterns, backed up the complex's structural integrity and ongoing interactions.
Conclusion: This investigation shows that ZINC225886466 could be a TRPC5 inhibitor with stable binding properties, and more experiments are needed to confirm its use in treating FSGS.
Future Directions: More research is needed, both in the lab and in living things, to confirm that the lead chemicals we found can change how TRPC5 works and stop the progression of FSGS.
Keywords：TRPC5 Inhibition; Focal Segmental Glomerulosclerosis (FSGS); Molecular Docking; Molecular Dynamics Simulation; Structure-Based Drug Design (SBDD)



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Breath VOC Signatures as a Novel Tool to Distinguish IgA Nephropathy from Non-IgA CKD

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Background:
IgA nephropathy (IgAN) is the most prevalent primary glomerulonephritis worldwide, particularly in the Asia-Pacific region. Despite its high burden, definitive diagnosis still relies on invasive kidney biopsy. There is an urgent need for non-invasive biomarkers to differentiate IgAN from other chronic kidney diseases (CKD). Volatile organic compounds (VOCs) in exhaled breath reflect systemic metabolic activity and have shown diagnostic value in various diseases. This study explores the potential of breath VOC profiling as a novel, non-invasive diagnostic approach for IgAN.
Methods:
Breath samples were collected from 161 participants: healthy controls (n=38), non-IgA CKD patients at stage 2 (n=33) and stage 3 (n=50), and biopsy-confirmed IgAN patients at stage 2 (n=20) and stage 3 (n=20). VOCs were analyzed using gas chromatography, with environmental background subtraction. One-way ANOVA identified VOCs that significantly differed across groups ($p<0.05$).
Results:
Among 51 VOCs with significant intergroup differences, 29 compounds (e.g., pyrrole, 1-octen-3-ol, 2-hexen-1-ol) were elevated in non-IgA CKD but suppressed in IgAN. In contrast, 5 VOCs (e.g., acetic anhydride, acrylonitrile) were uniquely elevated in IgAN. Nitric oxide levels, a marker of endothelial and immune function, were significantly reduced in IgAN compared to both non-IgA CKD and healthy controls. These results indicate a distinct VOC "breathprint" for IgAN.
Conclusion:
Breath VOC profiling can differentiate IgAN from other CKD subtypes and healthy individuals based on unique metabolic signatures. This non-invasive, patient-friendly method shows promise for early screening and diagnostic triage in IgAN, with potential to reduce reliance on kidney biopsy, especially in resource-limited settings.

Poster Presentation：Glomerular Diseases
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Rituximab Levels and CD19 Counts at Day 15 and 90 as Early Prognostic Markers in Patients with Membranous Nephropathy

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Introduction: Membranous nephropathy (MN) is the most common cause of primary nephrotic syndrome in adults. Treatment and follow-up are guided by KDIGO risk stratification. Rituximab (Rtx) has emerged as a first-line therapy in recent years; however, data on predictors of Rtx response are limited. A serum Rtx level <2 μg/mL at month 3 has been associated with poor prognosis. We aimed to evaluate the prognostic value of Rtx levels and CD19+ B-cell counts measured on days 15 and 90 in predicting treatment response at months 1, 3, and 6 in patients with primary MN.
Methods: In this prospective observational study, we included 46 primary MN patients classified as moderate or high-risk according to KDIGO criteria. Patients received either Rtx monotherapy or Rtx combined with a calcineurin inhibitor (CNI) and low-dose corticosteroid (CS), in addition to supportive therapy. Appropriate statistical analyses were performed. Due to the limited number of patients, complete and partial remission were analyzed together as treatment response.
Results: Demographic and clinical characteristics of patients are presented in Figure 1. Baseline demographics and clinical features were similar between responders and non-responders at month 6. Patients with day 15 Rtx levels >56 μg/mL had significantly higher response rates compared with those ≤56 μg/mL (Table 1). Patients with undetectable Rtx levels at day 90 had significantly lower response rates at month 6 (p=0.001). CD19+ B-cell depletion did not differ between responders and non-responders. In subgroup analysis, although patients in the Rtx+CNI+CS group had higher proteinuria and higher risk at baseline, Rtx levels were comparable between treatment groups (p=0.95).
Conclusion: Our study demonstrates that day 15 Rtx levels are strongly associated with treatment response at months 1, 3, and 6. Day 90 levels were also predictive of 6-month response, consistent with prior literature. Particularly in patients receiving Rtx monotherapy, monitoring drug levels at day 15 may allow early prediction of response and timely addition of CNI. Secondary findings suggest that combined therapy may be more appropriate in high-risk patients compared with monotherapy. Larger prospective studies are warranted.

Encore Abstract Declaration

This abstract was also submitted for the 42nd National Nephrology Congress. By submitting the abstract to APCN x TSN 2025, the authors declare that re-submitting the abstract is permitted by the organizers of the previous meeting. At the time of submission, no acceptance decision has yet been communicated.

Table 1: Comparison of day 15 rituximab levels with clinical responses at months 1, 3, and 6

	Month 1 ***no response***	**Month 1** ***response***	**p**	**Month 3** ***no response***	**Month 3** ***response***	**p**	**Month 6** ***no response***	**Month 6** ***response***	**p**
Low ≤56 (µg/ml)	13 (86.7%)	2 (13.3%)	**<0.001**	10 (66.7%)	5 (33.3%)	**0.004**	10 (66.7%)	5 (33.3%)	**<0.001**
High >56 (µg/ml)	8 (25.8%)	23 (74.2%)		7 (22.6%)	24 (77.4%)		4 (12.9%)	27 (87.1%)	