Poster Presentation：Glomerular Diseases
Poster No.：B0207
Abstract Submission No.：APCN20250115

IgA-dominant Lupus Nephritis: A case of coexistent glomerulopathies in a 28-year old female

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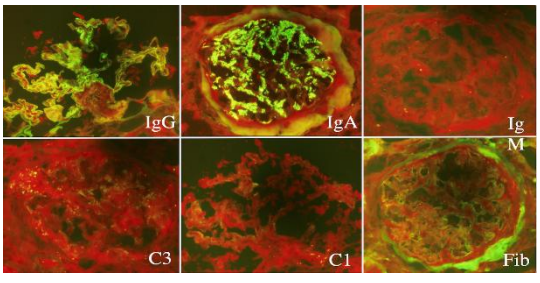
Abstract

Background: Lupus Nephritis (LN) is one of the serious manifestation of systemic lupus erythematosus (SLE) characterized by the classic immune complex-mediated glomerulonephritis with a 'full house" immunofluorescence staining pattern. In contrast, IgA nephropathy (IgAN), the most common primary glomerular disease, is characterized by mesangial IgA-dominant deposits. The coexistence of LN and IgAN is rare and presents a diagnostic challenge with therapeutic and prognostic implications to distinguishing between these two conditions.

Case: A 28 year- old Filipino female presented with a 3 month history of edema and frothy progressing to tea-colored urine. Concurrently, there is also associated pleuritic chest pain leading to worsening dyspnea. Serologic tests showed positive ANA (1:160) and high anti-dsDNA (144 IU/mL) and hypocomplementemia (C3), refractory sub-nephrotic range proteinuria (2+), UPCR 550 mg/gram , 24-hr urine protein:1080 mg/day), hematuria and RBC casts along with pleural effusion and pericardial effusion- confirming SLE with severe activity. A renal biopsy was performed.

Results: The light microscopy analysis showed diffuse proliferative glomerulonephritis with mesangial and endocapillary hypercellularity along with segmental sclerosis and occasional cellular crescents classified as Class IV lupus nephritis. The immunofluorescence analysis revealed predominant mesangial IgA deposition at 3+ intensity along with weaker IgG (1+) and trace levels of C3 and C1q deviating to the classic immunofluorescence picture of standard LN findings (Figure 1) . Electron microscopy confirmed mesangial electron-dense deposits, further sub-classifying the patient as having IgA predominant Class IV lupus nephritis. The patient received high-dose glucocorticoid 1mg/kg/day), mycophenolate mofetil (1.5g/day) hydroxychloroquine (400mg/day) and ACE inhibitor (Enalapril 2.5mg/day). She showed clinical improvement in her lupus disease management after 6 months follow-up with reduction of proteinuria (proteinuria from 2+ to trace, UPCR decreasing levels), stable renal function (CrCl: 90mL/m2) and resolution of edema and serositis (pleural effusion,pericardial effusion) .

Conclusion: The benefit of renal biopsy to address the diagnostic challenge of IgA-dominant glomerulonephritis in lupus patients in combination with the clinical and serologic result is essential for prognostication, short term and long term management. The identification of IgA nephropathy within lupus nephritis as either a separate condition or overlapping disorder remains essential as it may influence both treatment choices and future kidney outcomes. Additional research is necessary to determine the pathophysiology of this coexistence and eventually pave breakthroughs to new treatment approaches for this rare autoimmune overlap syndrome.



Keywords：glomerulonephritis, lupus nephritis, IgA nephropathy

Poster Presentation：Glomerular Diseases
Poster No.：B0208
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Albuminuria-induced Podocyte-derived Extracellular Vesicles Regulate Endoplasmic Reticulum Stress Expression

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Abstract
Introduction: Extracellular vesicles (EVs) play a significant role in glomerular diseases. Urinary EVs are found in diabetic nephropathy, focal segmental glomerulosclerosis and IgA nephropathy. The study was to investigate whether albuminuria could regulate EVs release from podocytes. Then, the podocyte-derived EVs could regulate ER stress.
Methods: Podocytes were exposed to medium alone or in high concentrations of delipidated, endotoxin-free human serum albumin (HSA, 10 mg/ml). An antioxidant N-acetyl-L-cysteine (NAC) and ER stress inhibitors (salibrinal: salu or 4-phenylbutyrate: 4-PBA) were used to treat podocytes after HAS treatment. The P-EVs were isolated and detected by centrifuged method and flow cytometry. The ER stress biomarker (GRP78 and CHOP) expression was analyzed by Western blotting. Intracellular ROS generation was measured using the fluorescent indicator 20, 70-dichlorofluorescin diacetate (DCF-DA).
Results: After HSA treatment, intracellular ROS generation and ER stress biomarkers (GRP78 and CHOP) were increased, NAC down-regulated ROS generation and ER stress biomarkers (GRP78 and CHOP).The quantities of P-EVs were significantly decreased ($p<0.05$) after HSA stimulation, but were significantly increased ($P<0.05$) after the stimulation of HSA+NAC, HSA+salu or HSA+4-PBA. The quantities of P-EVs were decreased ($p<0.05$) after the stimulated by P-EVs derived from HAS treatment, but were increased significantly ($P<0.05$) after the stimulated by P-EVs derived from the treatment of HSA+NAC, HSA+salu or HSA+4-PBA. The levels of GRP78 protein expression were down-regulated by P-EVs derived from HAS treatment, but were up-regulated by P-EVs derived from the treatment of HSA+NAC, HSA+salu or HSA+4-PBA.
Conclusion: The quantities of P-EVs were modulated by albumin stimulation through ROS-ER stress pathways. The P-EVs derived from albumin stimulation can regulate ER stress expression.

Keywords：Extracellular Vesicles; Podocytes; Endoplasmic Reticulum Stress; Albuminuria

Poster Presentation：Glomerular Diseases

Poster No.：B0210

Abstract Submission No.：APCN20250320

Successful use of Rituximab as early therapy to treat collapsing focal segmental glomerulosclerosis

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Abstract

Collapsing focal segmental glomerulosclerosis (cFSGS) is associated with severe nephrotic syndrome (NS) and rapid kidney failure. Treatment options include corticosteroids and calcineurin inhibitors (CNI), but low remission rates remain a concern. The role of rituximab in cFSGS management is not established but has shown promise in isolated cases. We report successful early-line rituximab use in an HIV-negative cFSGS with relative contraindications to standard oral immunosuppressants.

A 59-year-old Malay man presented to nephrology service with new-onset NS and acute kidney injury (AKI). Autoimmune and viral markers including parvovirus and cytomegalovirus, and medication history were unremarkable. Coincidentally, a computed tomography scan revealed a large pelvic tumour. Paraneoplastic NS was initially presumed, and patient underwent early debulking surgery. Histopathology of resected mass subsequently confirmed an inflammatory pseudotumour instead of malignancy. Persistent NS and worsening dialysis-requiring AKI led to a kidney biopsy, revealing cFSGS with 90% podocyte effacement and substantial acute tubular injury, without immune complex deposition. High dose prednisolone (1mg/kg) was initiated but adherence was poor, and the patient was readmitted with anasarca, uncontrolled hypertension and missed dialysis. Rituximab (2x1g) was administered to induce remission and spare steroids. Prednisolone was stopped prematurely by the patient after discharge. Partial remission was achieved in 6 weeks and serum albumin normalised by 12 weeks. At 5 months, relapse with observed with CD 19/20 reconstitution, prompting retreatment with prednisolone and Rituximab (2x1g). Prednisolone was again stopped prematurely, but remission was sustained for 6 months, with pre-emptive Rituximab (1g) administered thereafter. No adverse events were observed.

Evidence to support the use of Rituximab in primary, immune-mediated FSGS is still emerging, while data in cFSGS remains notably scarce. In the largest reported series (n=8), Rituximab achieved and maintained remission in 60% of patients, typically as a later-line option in patients with multi-drug dependent or resistant disease. Its role as front-line therapy in FSGS is less explored but may be clarified by future trials. However, published data and our experience suggest Rituximab can be considered in the initial management of de novo primary cFSGS to induce remission more rapidly, particularly as a steroid or CNI-sparer and when treatment adherence is of concern. Remission despite abrupt steroid cessation in our case suggests that Rituximab played a central role in inducing response in cFSGS. The optimal duration of Rituximab maintenance is unknown, underscoring the need for individualised strategies. Future research should clarify its role across FSGS subtypes and disease phases.

Keywords：Collapsing focal segmental glomerulosclerosis, Rituximab

Poster Presentation：Glomerular Diseases
Poster No.：B0211
Abstract Submission No.：APCN20250333

Patient Response and Outcomes of Protocol Directed Management Therapy Among Filipino Patients With Iga Nephropathy Seen In The Outpatient Department of A Tertiary Hospital From 2021-2023

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Abstract

Background and Objective

In 2021, the National Kidney and Transplant Institute (NKTI) released the second edition of its glomerulonephritis handbook to standardize the treatment of IgA nephropathy (IgAN), a common primary glomerular disease in Asia. This study aimed to assess patient outcomes and treatment response following the protocol-directed management of IgAN between 2021 and 2023.

Methods

A retrospective observational study was conducted involving 242 patients diagnosed with IgAN. Binary logistic regression was used to identify factors associated with therapy response and patient outcomes. The study also examined the relationships between treatment response, risk classification, and IgAN variants, with statistical significance set at $\alpha = 0.05$.

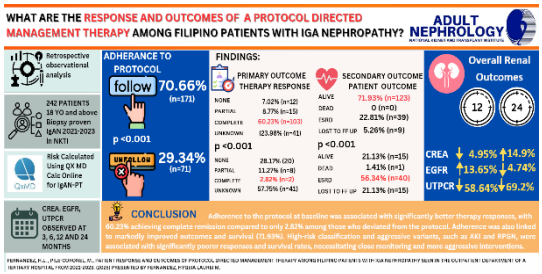
Results

Among the 242 patients, 70.66% adhered to the protocol at baseline, while 29.34% did not. At 12 months, there was a 4.95% decrease in median serum creatinine, suggesting early disease control or minimal progression. However, a 14.9% increase from baseline at 24 months may indicate disease progression, treatment resistance, or non-adherence. Despite this, overall eGFR remained relatively stable over two years, suggesting modest progression.

Significant improvement in proteinuria was observed, with the urine protein-to-creatinine ratio (UPCR) decreasing by 58.46% at 12 months and 69.23% at 24 months. Patients with acute kidney injury (AKI) had poorer outcomes than those without AKI. Notably, 60.23% of patients adhering to the protocol achieved complete remission, compared to only 2.82% among those who did not. Protocol adherence was also associated with improved survival (71.93% alive at follow-up). Conversely, the intermediate-risk group showed the highest progression to end-stage renal disease (ESRD) at 24 months (47.37%) and the lowest survival at 12 months (39.47%), indicating substantial disease progression despite treatment.

Conclusion

Although IgAN typically follows a slowly progressive course, a subset of patients may present acutely with AKI, requiring prompt intervention. The study highlights a clear trend of risk progression, with many patients shifting into higher-risk categories over time. Adherence to the NKTI treatment protocol significantly improves rates of complete remission, survival, and reduces progression to ESRD. These findings underscore the importance of early recognition, risk stratification, and strict compliance with standardized treatment protocols to optimize outcomes in IgAN management.



Keywords：IgA Glomerulonephritis, treatment protocol, patient outcome, proteinuria

Poster Presentation：Glomerular Diseases
Poster No.：B0212
Abstract Submission No.：APCN20250336

Investigating the association of collagen 1α1 expression in podocyte and glomerular disease severity

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Abstract
Background and Aims:
Glomerular diseases are significant contributors to both acute kidney injury (AKI) and chronic kidney disease (CKD). The glomerular filtration barrier is composed of three key structures: the endothelium, the glomerular basement membrane (GBM), and podocytes. Injury to any of these components compromises the filtration function of the kidney, potentially resulting in either nephritic or nephrotic syndrome. Podocytes are known to produce type IV collagen, a major structural component of the GBM. However, it remains unclear whether podocytes also express other types of collagen, such as collagen type I alpha 1 (collagen 1α1), and what functional role this protein might play in podocyte-related glomerular pathology. This study aims to explore the expression and potential role of collagen 1α1 in podocytes and its implications in glomerular disease.
Method:
To identify collagen-producing cells, we utilized Col1a1-GFPTg reporter mice. To investigate the functional role of collagen type I alpha 1 (Col1α1) in podocytes, we employed both constitutive (Podocin-CreTg; Col1α1 floxed/floxed) and inducible (Wt1CreERT2/+; Col1α1 floxed/floxed) podocyte-specific knockout mouse models. Ischemia-reperfusion injury (IRI) was induced as a model of acute kidney injury (AKI). We then compared plasma blood urea nitrogen (BUN), creatinine levels, and urinary protein excretion between Col1α1-deficient mice and their littermate controls under baseline conditions and at day 7 following IRI.
Results:
Our findings indicate that podocytes are potent producers of collagen in both healthy kidneys and those affected by ischemia-reperfusion injury (IRI). In Podocin-CreTg; Col1α1 floxed/floxed mice, deletion of the Col1α1 gene specifically in podocytes did not lead to abnormal plasma levels of BUN or creatinine, nor did it cause increased proteinuria under baseline conditions when compared to littermate controls. In contrast, Col1α1 deletion in Wt1CreERT2/+; Col1α1 floxed/floxed mice resulted in a significant increase in proteinuria relative to controls. Furthermore, in the IRI model, Podocin-CreTg; Col1α1 floxed/floxed mice exhibited higher proteinuria at day 7 post-injury compared to their littermate counterparts.
Conclusion:
In Podocin-CreTg; Col1α1 floxed/floxed mice, podocyte-specific deletion of Col1α1 did not induce significant proteinuria under baseline conditions, but resulted in increased proteinuria following ischemia-reperfusion injury (IRI). In contrast, Col1α1 ablation in Wt1CreERT2/+; Col1α1 floxed/floxed mice led to elevated proteinuria even in the absence of external stressors. This observation suggests a potential compromise of the glomerular filtration barrier, possibly due to podocyte injury or functional impairment. The precise mechanisms underlying the increased proteinuria observed in Wt1CreERT2/+; Col1α1 floxed/floxed mice remain to be fully elucidated and warrant further investigation.

Keywords：Podocyte, Col1a1, Glomerular disease

Poster Presentation：Glomerular Diseases
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Distinct Breath Volatile Organic Compound Signatures Differentiate IgA Nephropathy from Non-IgA Chronic Kidney Disease: A Novel Non-Invasive Diagnostic Approach

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Abstract

Introduction: IgA nephropathy (IgAN) is the most common form of primary glomerulonephritis, yet current diagnosis still depends on invasive kidney biopsy. Non-invasive biomarkers that can distinguish IgAN from other chronic kidney diseases (CKD) remain urgently needed. Breath volatile organic compounds (VOCs) reflect metabolic changes and have emerged as promising diagnostic indicators in systemic diseases. Previous studies had demonstrated the applicability of urine or breath VOCs in monitoring CKD progression, which raises our interest in exploring the role of VOCs in glomerulonephritis. Prior research also suggested the urinary volatile organic compounds (VOCs) of different types of glomerulonephritis may exhibit different patterns. This study investigates whether VOC profiling can discriminate IgAN from non-IgA CKD and healthy controls.

Methods: Breath samples were collected from 161 participants, including healthy controls (n=38), non-IgA CKD stage 2 (n=33) and stage 3 (n=50) patients, and IgAN CKD stage 2 (n=20) and stage 3 (n=20) patients. VOCs were analyzed using gas chromatography after subtraction of background air values. One-way ANOVA identified VOCs with significant differences across groups.

Results: Out of 51 VOCs with significant intergroup differences ($p<0.05$), 29 compounds—such as pyrrole, 1-octen-3-ol, and 2-hexen-1-ol—showed elevated levels in non-IgA CKD but were significantly suppressed in IgAN. Conversely, 5 VOCs—including acetic anhydride and acrylonitrile—were uniquely elevated in IgAN patients. Nitric oxide, a marker of vascular and immune status, showed a marked reduction in IgAN compared to both non-IgA CKD and controls. These findings suggest a distinct breathprint for IgAN that is metabolically divergent from other CKD etiologies.

Conclusion: Breath VOC profiling reveals disease-specific metabolic signatures that effectively differentiate IgAN from non-IgA CKD and healthy individuals. This novel, non-invasive tool holds strong potential for screening and early detection of IgA nephropathy, potentially reducing the need for diagnostic biopsy in selected cases. Further studies are needed to verify whether our results can be generalized to patients with advanced CKD, and whether VOCs can predict renal function deterioration in IgAN patients.

Keywords：IgA nephropathy, Chronic kidney disease, Breath volatile organic compounds

Poster Presentation：Glomerular Diseases
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Abstract Submission No.：APCN20250420

Comparative Analysis of Glomerular Structural Damage Induced by E-Cigarette Vapor and Cigarette Smoke in High-Fat Diet-Fed Rats

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Abstract

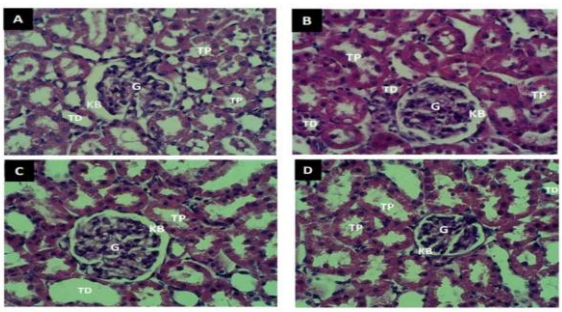
Background: Kidney disease, such as kidney failure, is a condition where the kidneys gradually lose their ability to filter blood properly, resulting in the need for renal replacement therapy such as dialysis or transplantation. There are currently over 1.4 million patients receiving renal replacement therapy worldwide. Internal and external factors could be the risk factors that improve kidney failure, such as race, gender, age, family history, diabetes, obesity, hypertension, and smoking. This study aims to compare the difference in glomerular structural damage induced by e-cigarette vapor and cigarette smoke in high-fat diet-fed rats.

Methods: This study was conducted using a true experimental method with a post-test-only randomized control group design. 28 male Wistar rats aged 1-2 months were randomly divided into four groups, namely normal (group A), high-fat diet (group B), high-fat diet and cigarette smoke (group C), and high-fat diet and e-cigarette vapor (group D). High-fat diet used in this study was the mixture of egg yolk and oxidized palm oil with a ratio of 2:3. It was given 2 mg/day. The intervention of conventional and electronic cigarettes was using a smoke exposure chamber. The dose of nicotine used in the group C and D was 12 mg/day. Renal histological changes were observed after seven weeks of intervention.

Results: The observation using a microscope showed no differences between normal and intervention groups. All of the groups showed histology of normal glomerulus without sclerosis. The renal tubules showed dense epithelial cells without degeneration or necrosis. In addition, in the glomerulus and tubules, there was no inflammation found. The observed condition may be attributed to the study's duration, which might have been insufficient to induce changes in the renal structure.

Conclusion: There was no different effect of cigarette smoke and e-cigarette vapor with a nicotine dose of 12 mg/day after seven weeks of intervention on the renal histology of high-fat diet-fed rats.

Keywords：Kidney Failure, E-cigarette vapor, Cigarette Smoke, Glomerular Structure



Poster Presentation：Glomerular Diseases
Poster No.：B0215
Abstract Submission No.：APCN20250530

Impact of ethnicity on comorbidity burden and clinical outcomes among Southeast Asians with lupus nephritis: a retrospective study

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Abstract

Introduction: Asians have higher lupus nephritis (LN) prevalence and severity compared to Caucasians. However, data on LN prognosis within the diverse Asian ethnicities outside of East Asia is scarce. We aim to evaluate the comorbidity burden and clinical outcomes of LN among different ethnicities within a multi-ethnic Southeast Asian population.

Methods: We performed a single-center, retrospective cohort study of 340 patients with biopsy-proven LN diagnosed between 2011 and 2023 at the Singapore General Hospital. Data was retrieved from electronic medical records at kidney biopsy. The outcome was kidney failure and/or death during follow-up. Univariable and multi-variable logistic regression analyses were performed to evaluate the impact of ethnicity on the outcome.

Results: Among the Chinese (252 patients), Malay (57 patients), Indian (11 patients) and other ethnicities (20 patients), the median age was 42.3 years (interquartile range 31.4–52.8). More Chinese were older (>40 years old) at diagnosis (p=0.002). In pairwise comparisons, Malays were less likely to be older than 40 years old than Chinese (odds ratio [OR] 0.33, 95% confidence interval [CI] 0.18-0.61). Diabetes mellitus was more frequent in Malays (OR 3.05, 95% CI 1.04-8.97) and Indians (OR 5.65, 95% CI 1.06-30.06) than Chinese. Compared to the Chinese and Malays, other ethnicities were less likely to have hypertension and renin-angiotensin system blocker (OR 0.27, 95% CI 0.08-0.96 and OR 0.20, 95% CI 0.06-0.69, respectively). There were no ethnic differences in other comorbidities such as hyperlipidemia, ischemic heart disease, LN histological classes, and type of immunosuppressant for induction treatment.

During the median follow-up of 50 (38, 74) months, 49 patients (14.4%) developed kidney failure and/or died. Incident kidney failure and death occurred in 7.6% and 12.6%, respectively. There was no significant difference in kidney failure and/or death between the ethnic groups. In multi-variable logistic regression models that evaluated race with and without traditional cardiometabolic risk factors (age >40 years, male sex, eGFR <60 ml/min/1.73 m2, diabetes mellitus, hypertension, hyperlipidemia and IHD), race was not significantly associated with patient and kidney survival, but reduced kidney function at diagnosis (adjusted OR 5.77, 95% CI 2.77-12.01) was independently associated with incident kidney failure and/or death.

Conclusion: Future studies should include under-represented ethnic groups to better understand ethnic-specific risk profiles and effects on LN outcomes.

Keywords：Lupus Nephritis, Ethnicity

Table. Univariable odds ratio and 95% confidence for factors compared between races

	Compared to Chinese			Compared to Malay	
	Malay	Indian	Other races	Indian	Other races
Comorbidity burden					
Age >40 years, yes versus no	**0.33 (0.18, 0.61)**	0.41 (0.12, 1.43)	0.71 (0.9, 1.78)	1.24 (0.32, 4.77)	2.17 (0.78, 6.13)
Male sex, yes versus no	1.13 (0.54, 2.36)	1.77 (0.45, 6.95)	0.53 (0.12, 2.35)	1.57 (0.36, 6.89)	0.47 (0.09, 2.31)
Diabetes mellitus, yes versus no	**3.05 (1.04, 8.97)**	**5.65 (1.06, 30.06)**	-	1.85 (0.32, 10.66)	-
Glucose-lowering agent, yes versus no	1.50 (0.39, 5.73)	2.70 (0.31, 23.43)	1.42 (0.17, 11.82)	1.80 (0.17, 19.10)	0.95 (0.09, 9.67)
Fasting glucose >7.0 mmol/L, yes versus no	1.49 (0.59, 3.77)	3.07 (0.56, 16.82)	0.55 (0.07, 4.38)	2.06 (0.33, 12.81)	0.37 (0.04, 3.26)
HbA1c >7%, yes versus no	2.52 (0.52, 12.14)	-	-	-	-
Hypertension, yes versus no	1.3 (0.56, 1.88)	0.72 (0.18, 2.96)	**0.27 (0.08, 0.96)**	0.71 (0.16, 3.13)	0.26 (0.07, 1.02)
RAS blocker, yes versus no	1.85 (0.99, 3.43)	1.04 (0.27, 3.96)	0.37 (0.12, 1.16)	0.56 (0.13, 2.34)	**0.20 (0.06, 0.69)**
Systolic BP >130 mmHg, yes versus no	1.03 (0.55, 1.92)	2.61 (0.66, 10.35)	0.40 (0.14, 1.15)	2.53 (0.59, 10.99)	0.39 (0.12, 1.25)
Hyperlipidemia, yes versus no	1.17 (0.57, 2.41)	3.20 (0.83, 12.40)	0.47 (0.11, 2.11)	2.73 (0.63, 11.81)	0.40 (0.08, 1.99)
Fasting LDL-cholesterol >2.6 mmol/L, yes versus no	1.70 (0.72, 3.40)	0.73 (0.0.12, 4.50)	5.82 (0.74, 46.07)	0.43 (0.06, 3.03)	3.43 (0.39, 30.52)
Statin, yes versus no	1.75 (0.94, 3.23)	2.69 (0.63, 11.53)	1.61 (0.62, 4.22)	1.54 (0.33, 1.06)	0.92 (0.31, 2.71)
Ischemic heart disease, yes versus no	0.70 (0.08, 5.93)	4.54 (0.49, 42.30)	2.02 (0.23, 17.69)	6.50 (0.37,114.64)	2.89 (0.17, 48.62)
Systemic lupus erythematosus, yes versus no	1.56 (0.70, 3.49)	1.29 (0.35, 4.76)	0.74 (0.24, 2.30)	0.83 (0.19, 3.57)	0.47 (0.13, 1.76)
eGFR <60 ml/min/1.73 m^2, yes versus no	1.60 (0.86, 2.98)	1.20 (0.31, 4.67)	-	0.75 (0.18, 3.16)	-
UPCR >3 g/g, yes versus no	1.43 (0.77, 2.64)	0.87 (0.23, 3.32)	1.36 (0.52, 3.56)	0.61 (0.15, 2.53)	0.95 (0.32, 2.80)
Class III, yes versus no	1.05 (0.48, 2.31)	2.11 (0.62, 7.21)	1.10 (0.34, 3.51)	2.00 (0.50, 8.00)	1.04 (0.28, 3.92)
Class IV, yes versus no	1.07 (0.46, 2.50)	0.61 (0.13, 2.93)	1.22 (0.26, 4.16)	0.57 (0.10, 3.16)	1.14 (0.28, 4.64)
Class V, yes versus no	2.07 (0.94, 4.58)	1.73 (0.48, 6.22)	0.91 (0.24, 3.47)	0.84 (0.20, 3.44)	0.44 (0.10, 1.91)
Isolated V, yes versus no	2.05 (0.68, 6.23)	1.12 (0.13, 9.35)	2.01 (0.40, 10.08)	0.54 (0.06, 5.21)	0.98 (0.17, 5.84)
Mixed III /IV and V, yes versus no	1.55 (0.60, 3.99)	2.08 (0.51, 8.40)	0.46 (0.06, 3.72)	1.34 (0.28, 6.43)	0.30 (0.03, 2.70)
Patient and kidney outcomes					
Kidney failure and/or death, yes versus no	1.44 (0.68, 3.03)	1.33 (0.28,6.42)	-	0.93 (0.18, 4.92)	-
Kidney failure, yes versus no	1.94 (0.76, 4.91)	3.07 (0.61, 15.36)	-	1.59 (0.28, 8.90)	-
Death, yes versus no	1.03 (0.39, 2.70)	1.34 (0.15, 11.98)	-	1.30 (0.13, 13.13)	-

BP, blood pressure; eGFR, estimated glomerular filtration rate; LDL, low-density lipoprotein; RAS, renin-angiotensin system; UPCR, urine protein-to-creatinine ratio.

Poster Presentation：Glomerular Diseases
Poster No.：B0216
Abstract Submission No.：APCN20250586

SPAK Deficiency Exacerbates Podocyte Injury and Metabolic Reprogramming in FSGS Mouse Model

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Abstract

Background:

Sterile 20/SPS1-related proline/alanine-rich kinase (SPAK) regulates ion transport and cellular homeostasis in the kidney, yet its role in glomerular injury remains poorly defined. This study aimed to investigate the impact of SPAK deficiency in a murine focal segmental glomerulosclerosis (FSGS) model.

Methods:

SPAK-knockout (KO) and wild-type (WT) mice were subjected to adriamycin-induced FSGS. Renal tissues were analyzed for podocyte markers (Nephrin and Podocin), mitochondrial biogenesis (PGC1α, ATP5A1) and dynamics (FIS1, DRP1, MFN1, OPA1), mitophagy-related proteins (PINK1, Parkin), and glycolytic enzymes (LDHA, phosphorylated PDHB) via Western blotting.

Results:

Compared to WT-FSGS mice, SPAK KO-FSGS mice exhibited further reduction in Nephrin and Podocin, indicating exacerbated podocyte injury. Mitochondrial biogenesis markers were downregulated, accompanied by altered mitochondrial dynamics, with increased fission markers (FIS1, DRP1) and decreased fusion markers (MFN1, OPA1). Mitophagy markers (PINK1 and Parkin) appeared modestly upregulated, possibly reflecting compensatory responses to enhanced mitochondrial stress. Concurrently, LDHA and P-PDHB levels were elevated, suggesting a metabolic shift toward glycolysis and impaired mitochondrial oxidative function.

Conclusion:

SPAK deficiency was associated with greater podocyte injury and altered mitochondrial homeostasis in FSGS, along with metabolic reprogramming indicative of a glycolytic shift. These findings suggest that SPAK may influence glomerular responses under pathological stress and warrants further investigation as a potential regulator of mitochondrial and metabolic function in kidney disease.

Keywords：SPAK, focal segmental glomerulosclerosis, mitochondria, metabolic reprogramming

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Urine-Derived Stem Cells Attenuate Podocyte Injury and Renal Fibrosis in Adriamycin-Induced Nephropathy via Anti-Apoptotic and Anti-Inflammatory Mechanisms

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Abstract

Background:

Chronic kidney disease (CKD) involves progressive damage to glomerular structures, often initiated by podocyte loss and apoptosis. Adriamycin (ADR)-induced nephropathy serves as a well-established model for mimicking this pathological process. Urine-derived stem cells (UDSCs), with their renal lineage potential and immunomodulatory capacity, have emerged as a potential therapeutic option. This study aimed to evaluate the protective effects of UDSCs on podocyte injury and renal dysfunction in ADR-induced CKD models.

Methods:

Human podocytes were treated with ADR to induce cellular damage. UDSCs were co-cultured using a transwell system to assess paracrine-mediated protective effects. Inflammatory and apoptotic proteins such as NF-κB, IL-1β, NLRP3, p53, Caspase-3, and cytochrome C were analyzed by western blot. Podocyte cytoskeletal integrity and nephrin expression were visualized by immunofluorescence staining. In vivo, mice with ADR-induced nephropathy received either single or multiple injections of UDSCs. Renal function was evaluated using urinary albumin/creatinine ratio, and tissue-level changes were assessed by histological staining and transmission electron microscopy.

Results:

ADR stimulation activated inflammatory and apoptotic pathways in a time-dependent manner, leading to podocyte structural disruption and nephrin downregulation. Co-culture with UDSCs suppressed pro-inflammatory and apoptotic markers and preserved cytoskeletal organization. In the in vivo model, UDSC treatment improved renal function, reduced albuminuria, and alleviated glomerular injury. Repeated UDSC administration showed greater efficacy in reducing fibrosis and restoring podocyte foot process morphology compared to single treatment. Overall, transcriptomic analysis supported these findings, indicating downregulation of genes related to ferroptosis, oxidative stress, and apoptosis following UDSC therapy.

Conclusion:

UDSCs offer substantial protection against ADR-induced renal injury by attenuating inflammation, inhibiting apoptosis, and preserving podocyte architecture. These results suggest that UDSCs may serve as a promising regenerative strategy for CKD, with enhanced therapeutic potential through repeated administration.

Keywords：Urine-derived stem cells (UDSCs) Podocyte injury Adriamycin nephropathy Chronic kidney disease

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Breaking The Norm: Minimal Change Disease in an Adult

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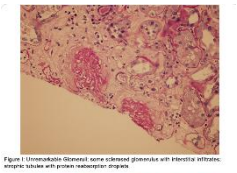
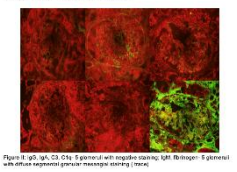
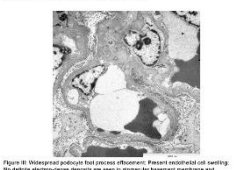
Abstract
Minimal change disease (MCD) is a common cause of nephrotic syndrome in children. It is characterized by an increase in renal membrane permeability and loss of protein due to damage to the glomerular filtration barrier. However, in adults, it is not a common disease and the exact incidence is not known. We present a case of a 68-year-old male, known hypertensive, not known chronic kidney disease who came in due to Bipedal Edema. He had a 2-week history of progressing bipedal edema and bubbly urine after he self-medicated with an antibiotic for sore throat. Upon consult, the patient was seen awake, not in acute cardiorespiratory distress with stable vital signs and normal anthropometrics. The patient had bipedal edema +2 up until the thigh with scrotal edema. The following pertinent laboratories revealed elevated creatinine at 3.25, BUN 66, normal electrolytes, hypercholesterolemia (427 mg/dL), hypertriglyceridemia (277 mg/dL), hypoalbuminemia (2.64 mg/dL), normal ASO titer at <200, normal C3, and non-reactive to hepatitis B infection but with previous hepatitis A infection. Kidney biopsy was done which revealed widespread podocyte foot process effacement. This was signed out as Minimal change disease. Patient was initially subjected to hemodialysis and started on steroid therapy. He eventually had renal recovery with improvement of creatinine to 0.94 mg/dL (eGFR 81.9). Prednisone 50mg/tab 1 tab once daily was initially maintained but eventually tapered off. Although the patient responded to steroid therapy, Unsampled focal segmental glomerulosclerosis (FSGS) should still be included in the consideration since this condition can also present with symptoms being manifested in minimal change disease and the degree of foot process effacement does not differentiate between unsampled primary FSGS and MCD. Some evidence suggests a common etiology between the pathogenesis of MCD and idiopathic FSGS should be studied together. However, this is still under study at the moment.

Keywords：Glomerulonephritis, Minimal change disease adult onset, Nephrotic syndrome







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Histopathological Pattern and Characteristics of Patients with Kidney Biopsy in Surabaya: A Single Center Study

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Abstract
Background: Renal biopsy is crucial while evaluating for the diagnosis of glomerular, vascular, tubulointerstitial, and genetic diseases. It gives vital information which helps in estimating the disease prognosis, progression, and management. While being gold standard for diagnosing many renal diseases, the data of renal biopsy and the characteristic of the patients and the disease are still rare in Indonesia.

Aim: We aim to document and analyse the data of renal biopsy information and characteristics in Soetomo General Academic Hospital, Surabaya, Indonesia. With the data, we hope to have better understanding of epidemiology and clinical manifestations of renal disease of the patient, so we can provide better management in the future.

Methods: We conducted a retrospective study on all kidney biopsy reports from patients over the age of 18, from October 2023 to June 2025. Descriptive statistics was used to summarise demographic characteristics. Categorical values were expressed as absolute frequencies and percentages.

Results: There were 37 renal biopsies included in the study. 20 (53%) patients were female; mean age was 29.16 ± 11.03 (18 to 55) years old; the clinical manifestations were mostly (25; 65.79%) nephrotic syndrome, 9 (23.68%) patients lupus nephritis, 3 (7.89%) patients CKD, 1 (2.63%) non-nephrotic proteinuria. The most common pathological findings were lupus nephritis and focal segmental glomerulonephritis (11; 28,94%). From the lupus nephritis patients, mostly were class IV (5; 9%).

Conclusion: Focal segmental glomerulonephritis and lupus nephritis were the most common findings in our community. Mostly young adult female with clinical characteristics of nephrotic syndrome. We need to optimize the use of this diagnostic modality to have more data and understanding of glomerulonephritis in our community and improve the management both the diagnosis and treatment of the diseases.

Keywords：Kidney biopsy, renal, histopathology, characteristics, glomerulonephritis

Poster Presentation：Glomerular Diseases
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Molecular Alterations of Podocytes and Parietal Epithelial Cells in Primary Focal Segmental Glomerulosclerosis vs IgA Nephropathy in CKD Stages 1-3

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Abstract

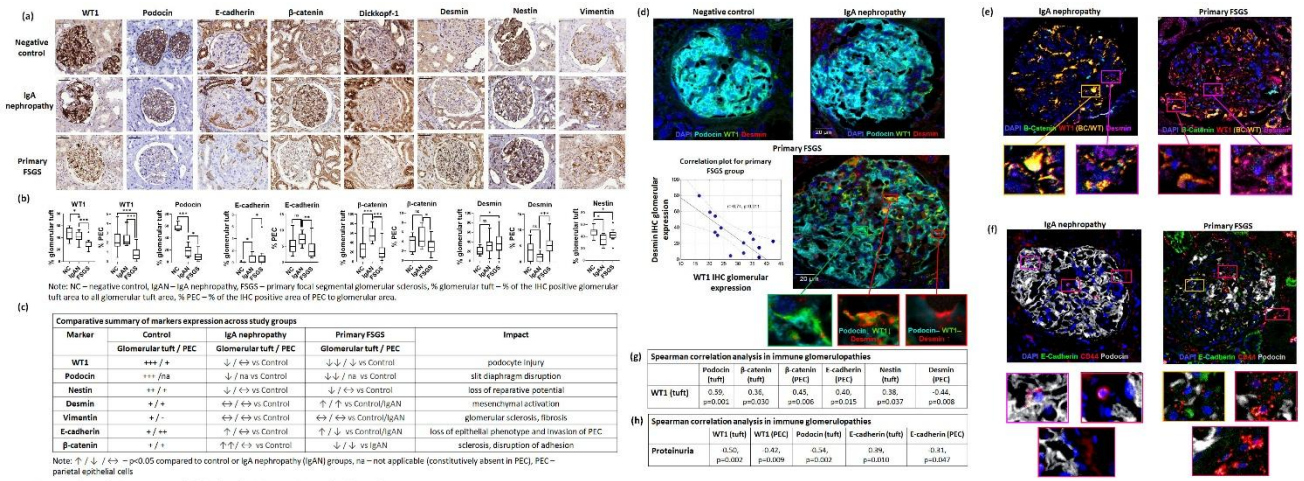
Introduction: Primary focal segmental glomerulosclerosis (pFSGS) and IgA nephropathy (IgAN) exhibit distinct clinical courses, yet both involve podocyte injury. We previously identified WT1 loss and desmin upregulation in pFSGS as early hallmarks of podocyte dysfunction vs. IgAN. Here, we deepen this analysis by comparing molecular phenotypes of podocytes and parietal epithelial cells (PEC), focusing on WT1 related epithelial, mesenchymal markers and reparative potential.

Methods: Quantitative immunomorphology and confocal microscopy assessed glomerular expression of WT1, podocin, β-catenin, dickkopf-1, E-cadherin, PAX8, CD44, Ki67, nestin, desmin, and vimentin in kidney samples of patients with morphologically confirmed pFSGS (n=16), IgAN (n=14) with CKD stages 1-3. Controls (n=16) were non-proteinuric nephrectomy samples. Confocal microscopy were conducted at the Center for Collective Use by Pavlov Institute of Physiology Russian Academy of Sciences.

Results: Patients with pFSGS exhibited nephrotic syndrome and typical glomerular alterations on light and electron microscopy, while IgAN showed milder proteinuria with higher glomerular sclerosis. Compared to control, both demonstrated common molecular pattern of podocyte injury – WT1↓/Podocin↓/Nestin↓/E-cadherin↑(Fig 1 a-c), likely indicating slit diaphragm disruption, loss of epithelial features and reparative potential. In pFSGS, podocyte alterations were more pronounced and accompanied by desmin↑ (Fig 1 a-c), which inversely correlated with WT1 expression, and predominantly localized in WT1↓- podocytes (Fig 1 d). IgAN characterized by higher β-catenin expression in podocytes (Fig 1 a-c, e). In FSGS vs IgAN, β-catenin was significantly lower both in podocytes and PEC without obvious differences in dickkopf-1 expression (Fig 1 a-c). Besides lower β-catenin, pFSGS demonstrated other molecular alterations in PEC – WT1↓/E-cadherin↓/Desmin↑, expression of CD44, PAX8 (including glomerular tuft area), likely related to the loss of epithelial phenotype, PEC activation and glomerular invasion (Fig 1 a-c, f). In the pooled pFSGS/IgAN group, (i) WT1 glomerular expression directly correlated with epithelial markers, and negatively – with desmin (Fig 1 g); (ii) proteinuria was associated with WT1, podocin, and E-cadherin glomerular expression (Fig 1 h).

Conclusion: Primary FSGS exhibits severe dual podocyte-PEC molecular shifts characterised by the lose of epithelial identity and acquire mesenchymal features in podocytes with PEC pathologic activation and impaired reparative potential, determining the structural and functional disorders of these cells.

Keywords：Podocytopathy; Parietal Epithelial Cells; Primary focal segmental glomerulosclerosis; IgA nephropathy; Biomarkers; WT-1; Podocin; β-catenin; E-cadherin; Desmin



Note: NC – negative control, IgAN – IgA nephropathy, FSGS – primary focal segmental glomerular sclerosis, % glomerular tuft – % of the IHC positive glomerular tuft area to all glomerular tuft area, % PEC – % of the IHC positive area of PEC to glomerular area.

(c)

Comparative summary of markers expression across study groups				
Marker	**Control**	**IgA nephropathy**	**Primary FSGS**	**Impact**
	Glomerular tuft / PEC	**Glomerular tuft / PEC**	**Glomerular tuft / PEC**	
WT1	+++ / +	↓ / ↔ vs Control	↓↓ / ↓ vs Control	podocyte injury
Podocin	+++ /na	↓ / na vs Control	↓↓ / na vs Control	slit diaphragm disruption
Nestin	++ / +	↓ / ↔ vs Control	↓ / ↔ vs Control	loss of reparative potential
Desmin	+ / +	↔ / ↔ vs Control	↑ / ↑ vs Control/IgAN	mesenchymal activation
Vimentin	+ / -	↔ / ↔ vs Control	↔ / ↔ vs Control/IgAN	glomerular sclerosis, fibrosis
E-cadherin	+ / ++	↑ / ↔ vs Control	↑ / ↓ vs Control/IgAN	loss of epithelial phenotype and invasion of PEC
β-catenin	+ / +	↑↑ / ↔ vs Control	↓ / ↓ vs IgAN	sclerosis, disruption of adhesion

Note: ↑ / ↓ / ↔ – p<0.05 compared to control or IgA nephropathy (IgAN) groups, na – not applicable (constitutively absent in PEC), PEC – parietal epithelial cells

(g) Spearman correlation analysis in immune glomerulopathies

	Podocin (tuft)	β-catenin (tuft)	β-catenin (PEC)	E-cadherin (PEC)	Nestin (tuft)	Desmin (PEC)
WT1 (tuft)	0.59, p=0.001	0.36, p=0.030	0.45, p=0.006	0.40, p=0.015	0.38, p=0.037	-0.44, p=0.008

(h) Spearman correlation analysis in immune glomerulopathies

	WT1 (tuft)	WT1 (PEC)	Podocin (tuft)	E-cadherin (tuft)	E-cadherin (PEC)
Proteinuria	0.50, p=0.002	0.42, p=0.009	0.54, p=0.002	0.39, p=0.010	0.31, p=0.047

Figure 1. Glomerular immunomorphological analysis in experimental and control groups:
(a) Representative immunohistochemical staining patterns of key markers; (b) Quantitative morphometric analysis of IHC expression levels; (c) Comparative summary of markers IHC expression across study groups; (d) Confocal microscopy demonstrating cellular localization and co-expression of Podocin, WT1 and desmin; (e) WT1, beta-catenin and desmin; (f) Podocin, E-cadherin and CD44; (g) Spearman correlation analysis between WT1 glomerular expression and related epithelial and mesenchymal markers in glomerular tuft and PEC, and (h) between glomerular marker expression and proteinuria severity in immune glomerulopathies.

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Abstract Submission No.：APCN20251109

Exploring the Mechanism of Quercetin in the Treatment of IgA Nephropathy Based on Network Pharmacology and Molecular Docking

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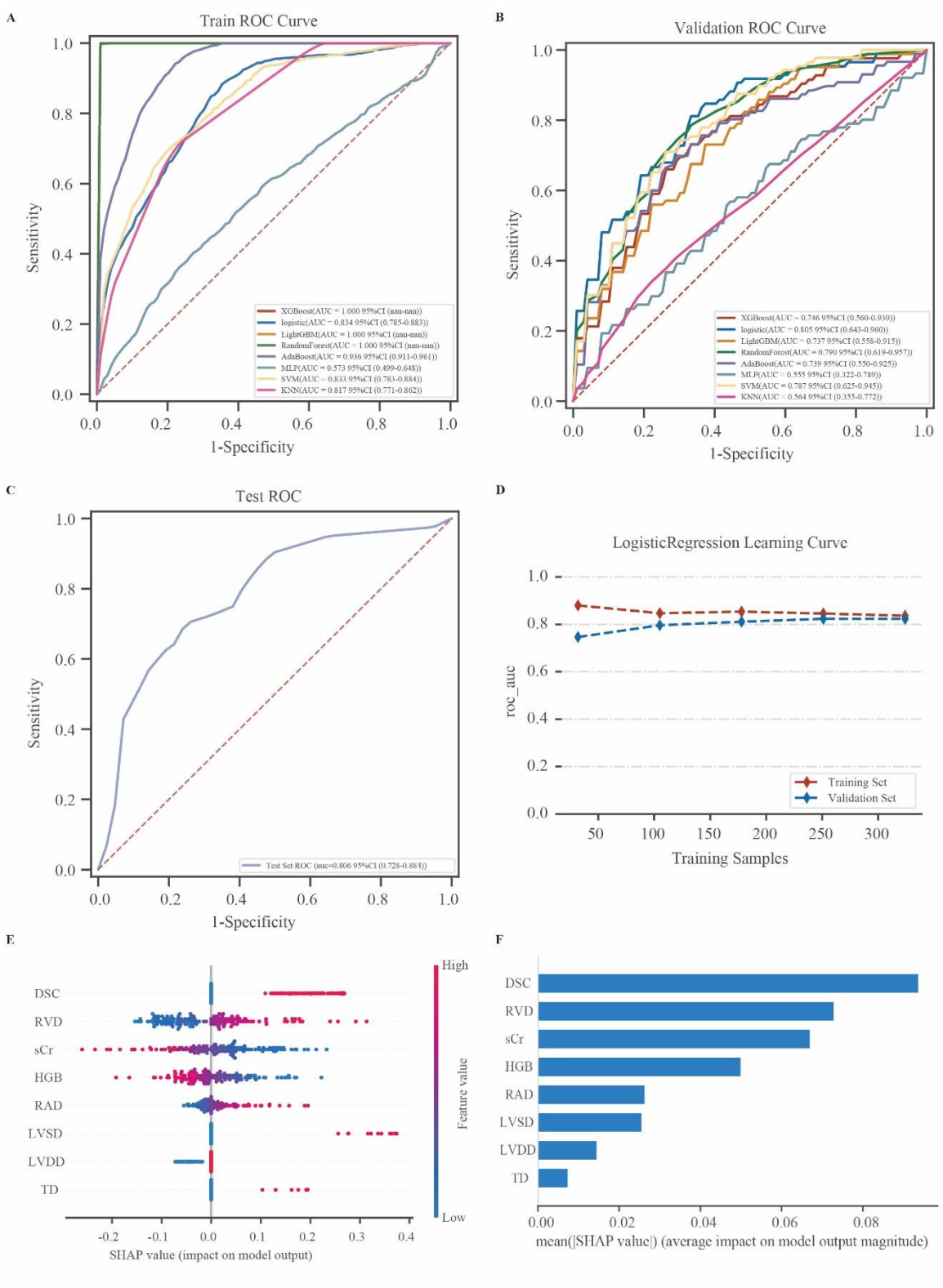
Abstract
Introduction
IgA nephropathy (IgAN) is the most common primary glomerulonephritis worldwide, with a particularly high prevalence in China, where it accounts for 45–60% of primary glomerular diseases and is a leading cause of end-stage renal disease (ESRD). Approximately 30–40% of patients progress to ESRD within 20–30 years of initial clinical presentation, resulting in significant physical, psychological, and economic burdens. Current management strategies focus on supportive therapy, including lifestyle modification, blood pressure control, and proteinuria reduction, but therapeutic options remain limited.
Quercetin, a natural flavonoid compound, has demonstrated therapeutic potential in various diseases due to its anti-inflammatory, antioxidant, anti-fibrotic, and anti-apoptotic properties, and its potential role in the treatment of IgAN has attracted increasing attention. This study aimed to investigate the potential mechanisms of quercetin in the treatment of IgAN using network pharmacology and molecular docking approaches.
Methods
The pharmacokinetic properties (ADME) of quercetin were evaluated using the Traditional Chinese Medicine Systems Pharmacology Database (TCMSP), with OB ≥ 30% and DL ≥ 0.18 as thresholds. Potential targets of quercetin were predicted via TCMSP, and IgAN-related targets were retrieved from GeneCards, OMIM, and DrugBank databases, followed by deduplication and standardization using the UniProt database.
A Venn diagram was generated to identify intersecting targets between quercetin and IgAN. The intersecting targets were imported into the STRING database to extract PPI data, and the PPI network was constructed using Cytoscape. Core targets were identified using the MNC algorithm in the CytoHubba plugin. GO and KEGG enrichment analyses were performed using Metascape with thresholds of $p < 0.01$, count > 3, and enrichment factor > 1.5, followed by visualization. Molecular docking was conducted using AutoDock Tools, with protein structures obtained from the PDB database, and docking results were visualized using PyMOL and Discovery Studio.
Results
Core targets identified included MMP9, TNF, IL-1β, EGFR, and AKT1. Enrichment analysis indicated that quercetin may exert its therapeutic effects through pathways including TNF signaling, IL-17 signaling, and atherosclerosis-related pathways. Molecular docking results demonstrated strong binding affinities between quercetin and these key targets.
Conclusion
Quercetin shows potential as a therapeutic agent for IgAN by acting on multiple targets and pathways. It may exert renal protective effects by regulating MMP9-mediated extracellular matrix metabolism, inhibiting mesangial cell proliferation and fibrosis, downregulating TNF and IL-1β to suppress inflammatory pathways and glomerular inflammation, and downregulating AKT1 and EGFR to prevent abnormal mesangial cell proliferation.

Keywords：Quercetin; IgA nephropathy; Network pharmacology; Molecular docking; Mechanism study



Poster Presentation：Glomerular Diseases
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Abstract Submission No.：E_APCN20251262

Exosome-like Particles Derived from *Chlorella sorokiniana* Mitigate Interferon-alpha-induced Renal Injury

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Introduction: Lupus nephritis (LN), a severe complication of systemic lupus erythematosus (SLE), involves immune complex deposition and chronic renal inflammation. Central to LN pathogenesis is the dysregulated activity of autoreactive $CD4^+$ helper T cells, which secrete pro-inflammatory cytokines such as Interferon-alpha (IFN-α), Interleukin-17 (IL-17), and Tumor Necrosis Factor-alpha (TNF-α), promoting macrophage activation and tissue damage. Recently, plant-derived exosomes, particularly from microalgae, have demonstrated significant immunomodulatory and anti-inflammatory capabilities, offering potential therapeutic benefits in inflammatory diseases.
Materials and Methods: IFN-α-induced renal injury models were established in vitro using rat renal mesangial cells (RMC) and porcine renal proximal tubular cells (LLC-pk1). Cells were cultured in Dulbecco's Modified Eagle Medium/Nutrient Mixture F-12 (DMEM/F-12) supplemented with 10% fetal bovine serum (FBS) and 1% penicillin/streptomycin at 37°C, 5% CO_2. Upon reaching 70–80% confluence, cells were passaged using 0.25% trypsin-EDTA and seeded at 2×10^5 cells/well in 6-well plates. After 24 hours of adherence, cells underwent pre-treatment with IFN-α (100, 200, or 500 IU/mL), followed by exposure to *Chlorella sorokiniana*-derived exosome-like particles (mean size: 72.8 nm; concentrations: 10^7, 10^8, or 10^9 particles/mL). Cell viability was assessed using Methylthiazolyl Tetrazolium (MTT) assays, and cytokine levels (IL-6, TNF-α) were quantified by Enzyme-Linked Immunosorbent Assay (ELISA).
Results: *Chlorella sorokiniana*-derived exosome-like particles significantly reduced IFN-α-induced cytotoxicity in both RMC and LLC-pk1 cells. Additionally, treatment downregulated nuclear factor-kappa B (NF-κB) signaling and diminished IL-6 and TNF-α secretion. These particles demonstrated high biocompatibility and effective modulation of inflammatory pathways.
Conclusions: *Chlorella sorokiniana*-derived exosomes-like particles effectively ameliorate IFN-α-mediated renal inflammation and cellular injury, representing a promising therapeutic strategy for LN.

Key words: Exosome-like particles, *Chlorella sorokiniana*, Interferon-alpha, Lupus nephritis, Immunomodulation

Poster Presentation：Glomerular Diseases
Poster No.：B0224
Abstract Submission No.：E_APCN20251265

Proteomics Unveils Molecular Profiles and Potential Pathogenic Mechanisms in IgA Nephropathy

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Introduction: IgA nephropathy (IgAN) is the most common primary glomerular disease globally, characterized by IgA deposition in the glomeruli. Its pathogenesis remains incompletely understood, highlighting the need for comprehensive molecular profiling to identify key proteins and pathways involved.
Aims: This study aims to identify differentially expressed proteins between IgAN patients and healthy controls (HC) using proteomics, and to elucidate potential pathogenic mechanisms and biomarkers for IgAN.
Materials and Methods: Plasma samples from 80 IgAN patients and 80 HC were analyzed using protein corona proteomics with the Proteograph™ Assay Kit. Differential proteins were identified based on P-VALUE < 0.05 and FOLD CHANGE ≤ 0.5 or ≥ 2. Further analysis included principal component analysis (PCA), protein-protein interaction (PPI) network analysis, KEGG pathway enrichment, Gene Ontology (GO) analysis, and Cluster of Orthologous Groups (COG) classification.
Results: PCA revealed significant differences between IgAN and HC groups ($P<0.001$). A total of 216 differentially expressed proteins were identified, with 121 upregulated and 95 downregulated in IgAN. Notably, CHMP4A, NOG, FBL, TNFRSF11B, and CAPN3 were the top 5 upregulated proteins, while CD63, IGKV1D-16, VDAC2, DOCK5, and PDIA6 were the top 5 downregulated proteins. PPI analysis showed that ACTB had the highest number of interacting proteins (46, $P = 0.024$). KEGG analysis highlighted the proteasome pathway (hsa03050) as the most significantly altered, while GO analysis identified the organic nitrogen compound metabolic process as a key affected pathway. COG analysis revealed 265 proteins involved in post-translational modification and protein turnover.
Conclusion: This study provides novel insights into the molecular landscape of IgAN, identifying key proteins and pathways that may contribute to its pathogenesis. These findings could serve as potential biomarkers and therapeutic targets for IgAN, warranting further validation in future studies.

Keywords: IgA nephropathy; Proteomics; Protein corona; Biomarkers; Pathogenic mechanisms

Poster Presentation：Glomerular Diseases
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Abstract Submission No.：E_APCN20251268

AXL Disrupts the Integrity of the Slit Diaphragm by Inhibiting ZO-1 Phase Separation and Aggravates Podocyte Injury in Diabetic Nephropathy

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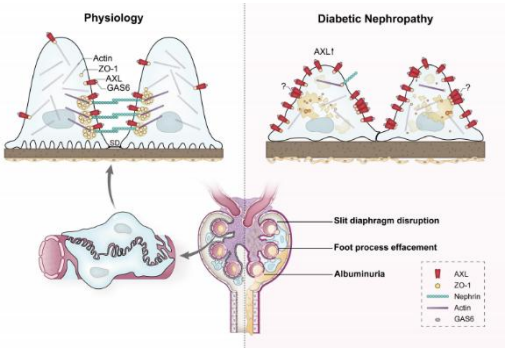
Introduction
Foot cell slit diaphragm injury is an important cause of foot cell lesions and proteinuria in diabetic nephropathy. Zonula occludens-1 (ZO-1) is an important connexin in the SD region and plays a key role in maintaining the normal structure and biological function of foot cells. The aim of this study was to explore the regulatory mechanism of AXL on ZO-1 and provide theoretical and experimental basis for targeted treatment of foot cell injury in diabetic nephropathy.
Methods
We examined the expression level of AXL in renal tissues of diabetic nephropathy patients and diabetic mice by database mining, single-cell sequencing and molecular biology. Immunoelectron microscopy, immunofluorescence and RNAScope were applied to detect the localisation of AXL in renal tissues. The target molecules that AXL may act on were screened and confirmed by mass spectrometry and Co-IP techniques. Apply techniques such as FRAP to detect the regulatory effect of AXL on ZO-1 phase separation. Application of podocyte-specific knockout AXL mice to verify the effect of AXL on diabetic nephropathy phenotype and its role.
Results
Our study found that AXL is expressed in glomerular podocytes and localised at the slit diaphragm. Up-regulation of AXL expression in renal tissues of patients with diabetic nephropathy predicts increased urinary protein and deterioration of renal function. In vivo animal experiments showed that overexpression of AXL aggravated podocyte injury in diabetic nephropathy mice, whereas AXL inhibitors significantly reduced podocyte injury. Podocyte-specific knockout of AXL in diabetic mice showed reduced podocyte damage and improved albuminuria. Further research has revealed that AXL downregulated the membrane expression level of the tight junction protein ZO-1 and altered its membrane localization. ZO-1 forms a membrane-bound phase through liquid-liquid phase separation, thereby participating in the formation of tight junctions. Its PSG domain is the key structural domain for phase separation. In diabetic nephropathy, overexpression of AXL inhibits the phase separation of ZO-1 by binding to the PSG domain of ZO-1, resulting in a reduction of the membrane-bound phase of ZO-1 and a loosening of the tight junction structure.
Conclusions
Our results suggest that AXL affects the integrity of the slit diaphragm by influencing the ZO-1 phase separation, leading to an imbalance in the cellular junction function of the podocytes and thus causing podocyte injury. It is suggested that AXL may serve as a potentially important target for regulating the slit diaphragm of podocytes, providing a new idea for the treatment of diabetic nephropathy.



Poster Presentation：Glomerular Diseases
Poster No.：B0226
Abstract Submission No.：E_APCN20251271

Podocyte Heterogeneity in Diabetic Kidney Disease: Deciphering the Metabolic Subtype and Its Crosstalk with Parietal Epithelial Cells

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Introduction
Podocyte injury critically contributes to diabetic kidney disease (DKD) progression, yet the cellular heterogeneity of podocytes and their distinct contributions to disease progression remains poorly characterized.
Methods
Single-cell RNA sequencing (scRNA-seq) was performed on kidneys from db/db (n=4) and db/m (n=4) mice to identify podocyte subtypes. We analyzed their functional characteristics, key transcription factors, interactions with parietal epithelial cells (PECs), and clinical relevance.
Results
Podocyte heterogeneity was mapped through single-cell RNA sequencing analysis. We identified three distinct subtypes: Cryab^{hi} metabolic subtype (Metab-subtype), Slc6a6^{hi} transitional phenotype (Trans-subtype), Magi2^{hi} adhesion subtype (Differentiated-subtype). Notably, we discover that both the Trans-subtype and Differentiated-subtype originated from the Metab-subtype. Transcription factor analysis suggested that Wt1 may play a key role in subpopulation conversion. Conjoint trajectory analysis of podocytes and PECs revealed impaired differentiation of PECs into Cryab^{hi} podocytes in DKD. The CDKN1A^{hi} subsets of PECs present during the DKD were classified, and CDKN1A^{hi} PECs were suggested as a primary podocyte subset associated with regeneration. We further uncovered intercellular crosstalk between Cryab^{hi} podocytes and PECs, predominantly mediated by the JAG-NOTCH2 and APP-SORL1/VEGFA_NRP2 ligand-receptor pairs. Clinically, the Cryab^{hi} subtype exhibited a negative correlation with proteinuria and serum creatinine but a positive correlation with eGFR, suggesting that its decline contributes to unfavorable prognosis of DKD.
Conclusion
These findings provide novel insights into podocyte heterogeneity and regeneration in DKD, highlighting potential therapeutic targets.
Key word
diabetic kidney disease; scRNA-seq; podocyte heterogeneity; Cryab; Cdkn1a; JAG1-NOTCH2;

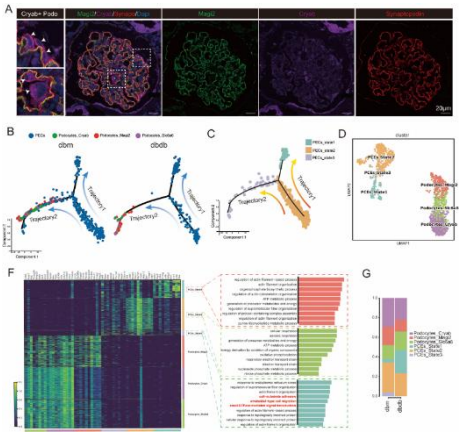

Figure 1. Cellular trajectory profiling reveal dynamic states of CDKN1A^{hi} PECs and Cryab^{hi} podocyte subpopulations in diabetic and control conditions.

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The *CARD9* S12N mutation is associated with an increased risk of IgA nephropathy in Han Chinese

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Abstract
Introduction: *CARD9*, identified as a susceptibility gene for IgA nephropathy (IgAN) in genome-wide association studies, encodes an adaptor protein crucial for pathogen defense and innate immunity. This study aimed to investigate the associations of *CARD9* variants with IgAN and their potential functional implications in disease pathogenesis.

Methods: A total of 3,319 IgAN patients and 6,747 healthy controls were enrolled across three independent cohorts, and 10 candidate single-nucleotide polymorphisms were genotyped. Serum cytokines and galactose-deficient IgA1 (Gd-IgA1) were measured by Luminex technology and ELISA. Molecular dynamics (MD) simulations assessed the residue mutations and dynamic properties of the CARD9 protein. Expression profiling data from the GEO database were used to explore *CARD9* expression and relevant pathways in renal tissues of IgAN patients.

Results: We found that rs4077515-T (S12N mutation) was associated with increased IgAN risk (OR = 1.26, 95% CI = 1.07–1.49, P = 0.006) and greater disease severity. The S12N mutation was significantly correlated with elevated serum levels of Gd-IgA1 and multiple cytokines particularly IL-17, IL-6, IL-1β, IL-8, and TNF-α. MD simulations revealed that the S12N mutation reduced structural stability of CARD9 and altered its interaction with BCL10. Gene expression profiling data suggested *CARD9* expression was upregulated in the renal interstitium of IgAN patients and was linked to Dectin-1, NF-κB expression and B cell-related immune pathways. Interestingly, IgAN patients carrying the S12N mutation exhibited significant alterations in gut microbial composition and taxa abundances, as revealed by in-depth analysis of our previously published 16S rRNA sequencing data.

Conclusions: Our results suggest that *CARD9* S12N mutation confers susceptibility to IgAN, potentially by modulating the CARD9-mediated immunity, particularly via the Dectin/CARD9/IL-17 and Dectin-1/CARD9/NF-κB signaling pathways.