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ORIGINAL RESEARCH

Efficacy and Safety of Rituximab in Treating Adult Patients with Minimal Change Disease and Focal Segmental Glomerulosclerosis: A Prospective Study Compared with Glucocorticoids

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Background: Minimal Change Disease (MCD) / Focal Segmental Glomerulosclerosis (FSGS) are leading causes of adult nephrotic syndrome. Roughly half of patients need long-term immunosuppression for steroid dependence or relapse, but traditional drugs carry substantial adverse effects. Rituximab (RTX) depletes CD20+ B cells and reduces anti-podocyte antibodies; pediatric data are encouraging, yet direct adult evidence—especially between treatment-naïve and relapsed patients—remains scarce.

Methods: This study enrolled 82 patients with MCD/FSGS diagnosed between 2020 and 2023, divided into the RTX group (24 patients, 9 treatment-naïve and 15 relapsed) and the glucocorticoid group (58 patients). The RTX group received standard-dose RTX (375 mg/m^2 weekly for 4 weeks), while the glucocorticoid group was treated with prednisone (1 mg/kg/day). Outcomes were compared using t-tests, χ^2/Fisher, logistic regression, and Kaplan–Meier analyses.

Results: The overall remission rates were 100% in the RTX group and 98.3% in the glucocorticoid group (P=0.876), but the RTX group had a significantly higher eGFR at the last follow-up (124.25 vs 109.00 mL/min/1.73 m^2, P=0.019). A statistically significant intergroup difference was also observed, with complete remission achieved in 89.5% of MCD patients versus 40% of FSGS patients (P< 0.05). In relapsed patients treated with RTX, prednisone dosage decreased from 34.0±15.7 mg/day to 7.7±7.8 mg/day (P< 0.001), annual relapse frequency dropped from 1.0 to 0 episodes/year (P=0.001), and 40% of patients completely discontinued glucocorticoids. The Complete Remission rate in treatment-naïve patients (88.9%) was higher than in relapsed patients (73.3%), but the difference was not statistically significant. No independent predictors of RTX efficacy were identified, and no severe infections or allergic reactions were observed.

Conclusion: RTX equals glucocorticoids in podocytopathy, cuts steroid use and relapse, improves long-term kidney survival, and is safe. Treatment-naïve patients may choose RTX upfront to avoid steroid side effects; relapsed patients can taper and stop steroids. However, due to the limited sample size, these results should be interpreted with caution. Larger trials must confirm its long-term efficacy and ESRD protection.

Keywords: rituximab, minimal change disease, focal segmental glomerulosclerosis, therapeutic effects, nephrotic syndrome

Introduction

Minimal Change Disease (MCD) and Focal Segmental Glomerulosclerosis (FSGS) are common causes of Nephrotic Syndrome (NS), accounting for more than 60% of adult NS cases. The prevalence of chronic kidney disease (CKD) in China is as high as 10.8%-13%, and the proportion of MCD and FSGS in primary patients glomerular disease is increasing year by year.[1] Glucocorticoids (GC), as the first-line treatment for NS, are effective in inducing remission in MCD and FSGS.[2] However, Sustained clinical implementation faces three barriers: glucocorticoid-induced metabolic derangement, progressive bone density reduction, and doubled infection risk, profoundly limiting treatment continuity.[3,4] More critically, up to 50% of patients with MCD and FSGS who achieve remission exhibit steroid dependence or suffer

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from frequent relapses.[2,5,6] This forces clinicians to rely on second-line immunosuppressive agents (eg, cyclosporine A, tacrolimus). However, these agents also have limitations, such as nephrotoxicity and myelosuppression, which seriously affect patient prognosis.[7–9] To improve patient prognosis, the B-cell depleting agent Rituximab (RTX) - a monoclonal antibody targeting CD20 - has provided a new direction in the treatment of podocytopathies in recent years. It stabilizes the glomerular filtration barrier by removing $CD20^+$ B cells,[10–12] and reducing the production of anti-podocyte autoantibodies[13] (eg, Anti-nephrin Antibodies[14]). However, there is still a gap in the evidence for RTX in adults with podocytopathies: available studies have focused on membranous nephropathy (MN), eg, the MENTOR study showed that 2-year remission rates for RTX in MN were significantly better than those for cyclosporine A (60% vs 20%).[15] Although RTX has targeted therapeutic advantages: first, it acts directly on B-cell-mediated immune abnormalities, avoiding non-specific immunosuppression by GCs; second, Steroid-sparing potential: Pediatric data suggest RTX significantly reduces GC doses and related complications;[16,17] and its cost-effectiveness: The advantages of RTX are that it can reduce the number of recurrent hospitalizations, and the long-term medical cost may be better than the traditional regimen.[18,19] However, most of the current studies on MCD/FSGS in adults are small-sample retrospective analyses. Currently, some studies have indicated that rituximab (RTX) can effectively alleviate the condition, maintain long-term remission, and significantly reduce the side effects of glucocorticoids and immunosuppressants in adult patients with glucocorticoid-dependent and/or frequently relapsing minimal change disease (MCD) and focal segmental glomerulosclerosis (FSGS).[20,21] However, there is still a lack of high-quality evidence on the efficacy and safety of treatment-naïve patients of RTX in adults with podocytopathies and no systematic comparison between treatment-naïve patients and relapsed patients. This prospective study is the first to concurrently enroll both treatment-naïve and relapsed adult patients with MCD/FSGS, directly comparing the two cohorts to address the following core questions: Can RTX replace glucocorticoids in treatment-naïve patients to avoid steroid-related toxicity?Can RTX achieve steroid-free remission and sustain long-term disease control in relapsed nephrotic syndrome? We seek to establish evidence-driven foundations for revolutionizing podocytopathy treatment paradigms, redefining RTX's role from salvage option in refractory cases to primary frontline therapy.

Research

Inclusion of Participants

This prospective cohort study enrolled 82 patients diagnosed with minimal change disease (MCD) or focal segmental glomerulosclerosis (FSGS) at the Department of Nephrology of our institution from January 2020 to December 2023. Inclusion criteria included (1) confirmed diagnosis of NS (urinary protein >3.5 g/24-h and serum albumin <30 g/L); (2) all patients with a renal-biopsy diagnosis of podocytopathy were eligible, irrespective of baseline estimated glomerular filtration rate (eGFR) and (3) patients with minimal change disease (MCD) and/or focal segmental glomerulosclerosis (FSGS) who are either treatment-naïve or have frequent relapses and/or steroid dependence. Exclusion criteria covered secondary FSGS, active infections, comorbidities with other glomerular diseases or malignancies, hepatitis B virus/ hepatitis C virus/HIV infections, and those who had received prior RTX therapy.

Observational Indicators and Adverse Event Records

Demographic characteristics (age, gender, BMI), pathology type (MCD/FSGS), laboratory parameters 24-hour urinary protein quantification (24-hup), serum albumin (ALB), serum creatinine (SCr), estimated Glomerular Filtration Rate (eGFR), and immune parameters (CD19+ B-cell count, IgG level). Pre- and post-treatment regimens, duration of maintenance of remission, and number of relapses. Anti-nephrin antibodies were not included in the analysis because only 2 of the screened patients tested positive, yielding an insufficient event rate for meaningful statistical evaluation. Adverse events associated with Rituximab (RTX) during follow-up were documented and assessed.

Treatment Program and Follow-Up

RTX Group: Treatment-naïve patients (no prior immunosuppressive therapy): received intravenous rituximab 375 mg/m^2 once weekly for four infusions only; no concomitant glucocorticoids were used. Relapsed patients (steroid-dependent or

steroid-resistant): intravenous rituximab 375 mg/m^2 once weekly for four infusions was added to ongoing oral glucocorticoids, which were subsequently tapered and discontinued according to clinical response. Glucocorticoid (GC) group: received standard Prednisone therapy (1 mg/kg/d, maximum 60 mg/day), tapered to a maintenance dose (5–10 mg/day) after 4 weeks. Initial treatment was Rituximab (RTX)1–4 times weekly or 1 g, 1–2 times every 2 weeks. B-cell depletion was defined as a peripheral B-lymphocyte count < 5 cells/mm^3. Rituximab (RTX) dosage was adjusted during follow-up according to individual patient characteristics, such as renal function, urinary protein quantification and B-lymphocyte count. Renin-angiotensin-aldosterone system (RAAS) inhibitors were titrated according to blood pressure. Follow-up cycles were after one course of Rituximab (RTX) and at months 1, 3, 6 and every 3–6 months thereafter until the endpoint. The follow-up schedule included outpatient visits at baseline and 3/6/9/12 months after treatment, with urine protein, blood biochemistry, and immune markers tested at each visit. The study endpoint was set as the development of End-Stage Kidney Disease (ESKD) or selection of another type of immunosuppressive therapy.

Treatment Response and Renal Outcome

Determination of therapeutic efficacy: (1) Complete Remission (CR): 24-h urine protein quantification< 0.3 g, serum albumin > 3.5 g/L, and serum creatinine normal; (2) Partial Remission (PR): 24-h urine protein quantification of urinary protein 0.3–3.5 g/d and with a reduction of >50% from baseline, serum albumin concentration improved or returned to normal, and serum creatinine stabilized or elevated< 30%; (3) No Remission (PR) refers to the failure to achieve the above criteria. (4) Relapse: urine protein> 3.5 g/24 h after a period of remission. Frequent relapses were defined as ≥2 every 6 months or ≥4 every 12 months. Steroid dependence: Therapeutic non-response defined as relapse concurrent with GC administration or within 14 days post-therapy.[22] Primary endpoint: failure to achieve complete or partial remission (ie, treatment failure) within 12 months. Key secondary composite endpoint: sustained decline in eGFR ≥40% or initiation of renal replacement therapy.

Statistical Analysis

All data were managed and analyzed using SPSS 26.0. Continuous variables were first tested for normality with the Shapiro–Wilk test. Variables that were normally distributed and met the assumption of homogeneity of variance are presented as mean ± standard deviation ($\overline{x} \pm s$). Within-group comparisons used the paired-sample *t*-test; between-group comparisons used the independent-sample *t*-test. Continuous variables that were not normally distributed are expressed as median (interquartile range). Within-group comparisons used the Wilcoxon signed-rank test; between-group comparisons used the Mann–Whitney *U*-test. Multiple comparisons were corrected with the Bonferroni test. Categorical variables are reported as frequency (percentage) and compared between groups with the χ^2-test or Fisher's exact test.

Associations were examined with Spearman correlation analysis. Risk-factor analysis was performed with logistic regression. Variables with P < 0.20 in univariate analysis were entered into the multivariate model. Multicollinearity was assessed by the variance inflation factor (VIF); VIF < 10 indicated no significant collinearity. Model goodness-of-fit was evaluated with the Hosmer–Lemeshow test (P > 0.05 indicates adequate fit). Relapse-free survival was analyzed by the Kaplan–Meier method, with differences assessed by the Log-rank (Mantel-Cox) test. A two-sided P-value < 0.05 was considered statistically significant.

Outcomes

Comparison of Basic Data of Patients in RTX and Glucocorticoid Groups

Comparison of Baseline Characteristics and Treatment Outcomes

The Mann–Whitney U and chi-square tests confirmed the comparability of baseline profiles between the RTX and glucocorticoid cohorts, including demographic data, pathological characteristics, and laboratory parameters (gender, age, BMI, pathological classification, comorbidity burden, relapse history, baseline 24-hour urinary protein, serum albumin, serum creatinine, and eGFR; all P > 0.05). However, patients in the control group (glucocorticoids) had a significantly longer follow-up duration after treatment initiation than those in the RTX group (P < 0.05). For detailed data, see Table 1.

Table 1 Comparison of General Information and Baseline Clinical Characteristics Between the RTX and Glucocorticoid Groups

Parameter	RTX (n=24)	Glucocorticoids (n=58)	χ^2/z value	P-value
Gender, n (%)			0.068	0.795
Male	15 (62.5%)	38 (65.5%)		
Female	9 (37.5%)	20 (34.5%)		
Age (years)	31.50 (20.25, 40.00)	27.00 (18.00, 44.00)	−0.592	0.554
BMI (kg/m^2)	25.08 (22.35, 27.34)	24.46 (22.16, 27.52)	−0.153	0.878
Pathologic diagnosis, n (%)			0.832	0.362
FSGS	5 (20.8%)	6 (10.3%)		
MCD	19 (79.2%)	52 (89.7%)		
Number of relapses (times)*	0 (0, 0)	0 (0, 0)	−0.201	0.841
Follow-up duration (years)	1.58 (1.27, 2.42)	2.46 (1.65, 3.23)	−2.505	0.012
Annualized relapse rate after medication (times/year)	0 (0, 0)	0 (0, 0)	−0.345	0.730
24-h urine protein quantification (g/24 h)	4.28 (3.20, 9.72)	5.00 (3.49, 8.56)	<0.001	1.000
Serum Albumin (g/L)	20.85 (17.63, 25.73)	20.05 (17.88, 25.50)	−0.326	0.744
Serum Creatinine (μmol/L)	69.50 (61.00, 95.25)	72.50 (57.75, 91.00)	−0.031	0.976
eGFR (mL/min/1.73 m^2)	103.00 (96.25, 120.00)	101.50 (80.25, 117.25)	−0.856	0.392

Notes: *Data are presented as the total number of relapses prior to enrollment.

Analysis of the final clinical outcomes revealed no statistically significant differences between the two groups in the final 24-hour urinary protein quantification, serum albumin, or serum creatinine levels (all $P > 0.05$). Notably, the final eGFR was significantly lower in the glucocorticoid group compared to the RTX group ($P < 0.05$).

Comparison of Treatment Response and Final Clinical Outcomes

Analysis of therapeutic efficacy revealed no statistically significant difference in the overall remission rate between the RTX and glucocorticoid groups (100% vs 98.3%, $P = 0.876$). Within the RTX group, patients with MCD achieved a significantly higher complete remission rate than those with FSGS (89.5% vs 40.0%, $P < 0.05$).

Evaluation of final clinical parameters showed no significant intergroup differences in 24-hour urinary protein (0.16 vs 0.12 g/24 h, $P = 0.170$), serum albumin (45.05 vs 46.00 g/L, $P = 0.099$), or serum creatinine levels (60.00 vs 66.50 μmol/L, $P = 0.178$). However, the final estimated glomerular filtration rate (eGFR) was significantly higher in the RTX group compared to the glucocorticoid group (124.25 vs 109.00 mL/min/1.73 m^2, $P = 0.019$). For detailed results, see Table 2.

Table 2 Treatment Response and Final Clinical Outcomes in the RTX and Glucocorticoid Groups

Parameter	RTX (n=24)	Glucocorticoids (n=58)	χ^2/z Value	P-Value
Treatment Response, n (%)			−0.156	0.876
Complete Remission (CR) (n)	19*	47		
Partial Remission (PR) (n)	5	10		
No response to treatment (n)	0	1		
Remission rate (%)	100	98.3		
Final Clinical Characteristics				
24-h urine protein quantification (g/24 h)	0.16 (0.11, 0.29)	0.12 (0.08, 0.24)	−1.372	0.170
Serum Albumin (g/L)	45.05 (40.80, 47.28)	46.00 (42.08, 48.75)	−1.651	0.099
Serum Creatinine (μmol/L)	60.00 (53.00, 71.00)	66.50 (57.50, 76.50)	−1.346	0.178
eGFR (mL/min/1.73 m^2)	124.25 (116.05, 133.50)	109.00 (93.75, 125.25)	−2.350	0.019

Notes: The results were adjusted for sex, age, BMI, and post-treatment time. *Of the 19 patients who achieved complete remission in the RTX group, 17 were diagnosed with MCD (89.5%) and 2 with FSGS (40.0%).

Table 3 Comparison of General Information Between the RTX Group Population, Treatment-Naïve Patients and Relapsed Patients

Parameter	Treatment-Naïve (n=9)	Relapsed (n=15)	χ^2/t/z Value	P-Value
Gender, n (%)			0.960	0.327
Male	4 (44.4%)	11 (73.3%)		
Female	5 (55.6%)	4 (26.7%)		
Age (years)	27.00 (21.00, 35.00)	36.00 (19.00, 48.00)	−0.567	0.599
BMI (kg/m^2)	24.34 (22.16, 26.99)	26.42 (22.59, 27.99)	−0.745	0.456
Pathologic diagnosis, n (%)			-	0.118
FSGS	0 (0%)	5 (33.3%)		
MCD	9 (100.0%)	10 (66.7%)		
Steroid therapy at the moment of RTX infusion, n (%)	0 (0%)	15 (100%)		
RTX infusion with glucocorticoids, n (%)	0 (0%)	15 (100%)		
Number of RTX infusions	3.11± 1.76	3.53± 1.81	−0.559	0.582
Total RTX dose (g)	2.24± 0.87	2.38± 0.74	−0.406	0.689

Comparison of Basic Data of RTX Group Population, Treatment-Naïve Patients and Relapsed Patients

Comparison of General Information Between the RTX Group Population, Treatment-Naïve Patients and Relapsed Patients

The Mann–Whitney *U*-test, two independent samples *t*-test, chi-square test and Fisher's exact test revealed that in the RTX group population, there was no statistically significant difference in the comparison of gender, age, BMI, pathological diagnosis, past medical history, number of RTX infusions and total RTX dose between the primaries and relapsed patients ($P> 0.05$). See Table 3.

Comparison of Efficacy Profiles in the RTX Group Population, Treatment-Naïve Patients and Relapsed Patients

The Mann–Whitney *U*-test, chi-square test and Fisher's exact test found that In the RTX group, differences in remission rates, number of relapses after treatment, time since treatment, frequency of relapses in the year after treatment, improvement in relapse frequency, time to first relapse, and average duration of remission maintenance were compared between treatment-naïve and relapsed patients ($P>0.05$). The percentage of discontinued glucocorticoids was lower in relapsed patients than in first-treatment patients ($P< 0.05$). The number of relapses before RTX initiation, the time before medication, and the frequency of relapses in the year before medication were higher in the relapsed patients than in the treatment-naïve patients ($P< 0.05$). See Table 4.

Table 4 Comparison of Efficacy Profiles in the RTX Group Population, Treatment-Naïve Patients and Relapsed Patients

Parameter	Treatment-Naïve (n= 9)	Relapsed (n=15)	χ^2/t/z Values	P-Value
Remission, n (%)			-	0.615
Partial Remission (PR)	1 (11.1%)	4 (26.7%)		
Complete Remission (CR)	8 (88.9%)	11 (73.3%)		
Steroid withdrawal, n (%)	9 (100.0%)	6 (40.0%)	6.270	0.012
Number of relapses before RTX initiation (times)	0 (0, 0)	2.00 (1.00, 4.00)	−3.923	<0.001
Time before medication (years)*	0 (0, 0)	2.67 (0.67, 3.33)	−3.902	<0.001
Frequency of relapses in the year before medication (times/year)	0 (0, 0)	1.00 (0.57, 1.82)	−3.900	<0.001
Number of relapses after medication (times)	0 (0, 0)	0 (0, 1.00)	−1.656	0.290
Time since medication (years)**	2.08 (1.46, 2.38)	1.42 (0.92, 2.42)	−0.658	0.519
Annualized relapse rate after medication (times/year)	0 (0, 0)	0 (0, 0.29)	−1.653	0.290
Frequency improvement	1.00 (1.00, 1.00)	1.00 (0.88, 1.00)	−1.653	0.290

(*Continued*)

Table 4 (Continued).

Parameter	Treatment-Naïve (n= 9)	Relapsed (n=15)	χ^2/t/z Values	P-Value
Time to response***	1.00 (1.00,1.50)	1.00 (1.0,4.0)	−0.581	0.561
Time to first relapse (months)	0 (0, 0)	0 (0, 0.33)	−1.654	0.290
Average duration of maintenance of remission (months)	22.56±6.65	19.07±11.24	0.842	0.409

Notes: *Time before medication is defined as the interval from initial disease onset to the first RTX infusion. **Time since medication is defined as the duration of follow-up after the first RTX infusion. ***Time to response was defined as the time from treatment initiation to initial clinical response, comprising either complete or partial remission.

Comparison of Initial and Final Clinical Data for the RTX Group Population, Treatment-Naïve Patients and Relapsed Patients

The Mann–Whitney *U*-test found that in the RTX group population, the 24-h urine protein quantification (final), serum albumin (initial), serum albumin (final), serum creatinine (initial), serum creatinine (final), eGFR (initial), eGFR (final), T (initial), T (final), $CD4^+$T (initial), $CD4^+$T (final), $CD8^+$T (initial), $CD8^+$T (final), $CD3^+CD4^+CD8^+$T cells (initial), $CD3^+CD4^+CD8^+$T cells (final), B (initial), B (final), NK (initial), NK (final), CD45 (initial), CD45 (final), IgG (final), IgA (initial), IgA (final), IgM (initial), IgM (final), C3 (initial), C3 (final), C4 (initial) and C4 (final) levels were compared, and the differences were not statistically significant ($P > 0.05$). The 24-h urine protein quantitative (initial) levels were lower in the relapsed patients than in the treatment-naïve patients ($P < 0.05$). The IgG (initial) levels of the relapsed patients receiving RTX retreatment were lower than those of the treatment-naïve patients ($P < 0.05$). See Tables 5 and 6.

Table 5 Comparison of Initial Clinical Data for the RTX Group Population, Treatment-Naïve Patients and Relapsed Patients

Parameter	Treatment-Naïve (n=9)	Relapsed (n=15)	*Z* Value	P-Value
24-h urine protein quantification (g/24 h)	10.11 (4.07, 18.04)	3.30 (2.56, 7.26)	−2.296	0.021
Serum Albumin (g/L)	20.00 (17.15, 22.00)	24.20 (17.60, 36.20)	−1.372	0.174
Serum Creatinine (μmol/L)	69.00 (62.00, 73.50)	77.00 (55.00, 104.00)	−0.507	0.640
eGFR (mL/min/1.73 m^2)	102.00 (98.00, 123.50)	104.00 (73.00, 120.00)	−0.955	0.347
T (initial) (cells/μL)	1126.40 (931.40, 1126.40)	2115.50 (1086.85, 2834.40)	−1.202	0.282
$CD4^+$T (initial) (cells/μL)	612.10 (593.50, 612.10)	1013.50 (678.55, 1314.10)	−1.389	0.209
$CD8^+$T (initial) (cells/μL)	455.60 (255.10, 455.60)	835.10 (311.30, 1468.70)	−0.832	0.482
$CD3^+CD4^+CD8^+$T (initial) (cells/μL)	4.50 (1.90, 4.50)	8.90 (4.05, 12.85)	−0.647	0.600
B (initial) (cells/μL)	161.22 (119.15, 263.95)	298.22 (181.18, 390.95)	−1.960	0.055
NK (initial) (cells/μL)	148.70 (116.60, 148.70)	204.70 (73.65, 658.85)	−0.277	0.864
CD45 (initial) (cells/μL)	1476.70 (1214.40, 1476.70)	2594.30 (1619.70, 3462.75)	−1.572	0.145
IgG (g/L)	3.16 (2.35, 5.24)	6.57 (4.02, 9.36)	−2.037	0.043
IgA (g/L)	1.62 (1.18, 2.42)	1.81 (1.03, 2.18)	−0.067	0.948
IgM (g/L)	1.30 (0.93, 1.63)	1.01 (0.76, 1.80)	−1.102	0.292
C3 (g/L)	0.91 (0.87, 1.09)	0.95 (0.83, 1.07)	−0.033	1.000
C4 (g/L)	0.28 (0.25, 0.34)	0.23 (0.22, 0.28)	−1.870	0.060

Table 6 Comparison of Final Clinical Data for the RTX Group Population, Treatment-Naïve Patients and Relapsed Patients

Parameter	Treatment-Naïve (n=9)	Relapsed (n=15)	z Value	P-Value
24-h urine protein quantification (g/24 h)	0.15 (0.07, 0.21)	0.17 (0.11, 2.20)	−0.926	0.379
Serum Albumin (g/L)	45.40 (43.05, 46.20)	44.90 (37.40, 47.60)	−0.507	0.640
Serum Creatinine (μmol/L)	59.00 (56.50, 61.50)	62.00 (49.00, 101.00)	−0.627	0.558
eGFR (mL/min/1.73 m^2)	126.50 (116.10, 132.00)	122.00 (98.70, 135.00)	−0.746	0.482

(*Continued*)

Table 6 (Continued).

Parameter	Treatment-Naïve (n=9)	Relapsed (n=15)	z Value	P-Value
T (final) (cells/μL)	1240.65 (1009.85, 2005.30)	1856.00 (1745.40, 2183.33)	−1.780	0.083
$CD4^+T$ (final) (cells/μL)	784.80 (514.70, 1160.20)	1078.05 (789.28, 1246.68)	−1.405	0.180
$CD8^+T$ (final) (cells/μL)	440.80 (336.18, 766.88)	822.60 (522.78, 996.40)	−1.592	0.125
$CD3^+CD4^+CD8^+T$ (final) (cells/μL)	7.10 (5.20, 11.30)	9.10 (6.38, 15.25)	−0.984	0.335
B (final) (cells/μL)	3.85 (1.40, 35.70)	5.75 (1.73, 20.48)	−0.037	0.971
NK (final) (cells/μL)	258.35 (110.10, 409.15)	173.10 (99.35, 466.35)	−0.187	0.892
CD45 (final) (cells/μL)	1588.10 (1364.98, 2401.78)	2216.70 (1933.28, 2722.60)	−1.592	0.125
IgG (g/L)	6.44 (3.91, 8.07)	5.55 (3.42, 8.17)	−0.263	0.831
IgA (g/L)	1.24 (1.08, 1.52)	1.44 (0.82, 2.06)	−0.351	0.765
IgM (g/L)	1.10 (0.90, 1.51)	0.77 (0.50, 1.21)	−1.491	0.152
C3 (g/L)	1.02 (0.82, 1.06)	0.81 (0.66, 0.94)	−1.228	0.244
C4 (g/L)	0.26 (0.23, 0.32)	0.24 (0.17, 0.37)	−0.439	0.701

RTX Group Population, Analysis of Factors Affecting Complete Remission (CR) in Treatment-Naïve Patients and Relapsed Patients

Using remission status as the dependent variable (complete remission = 1, partial remission = 0), we performed logistic regression analysis with treatment type, sex, age, pathological diagnosis, BMI, initial 24-h urinary protein, baseline albumin, baseline creatinine, baseline eGFR, baseline B-cell count, baseline IgG, number of RTX infusions, and total RTX dose as independent variables. Univariate regression analysis did not identify any variable that significantly influenced complete remission in the RTX group ($P > 0.05$). Multivariate regression analysis, including variables with $P < 0.2$ from the univariate analysis, also did not identify any independent factor that significantly influenced complete remission ($P > 0.05$). Variance inflation factors (VIF) for the variables in the model ranged from 1.706 to 7.876, indicating no multicollinearity among the independent variables. The Hosmer–Lemeshow goodness-of-fit test showed no significant difference between the observed and predicted probabilities of complete remission ($\chi^2 = 7.749$, df = 8, $P = 0.458$), $P > 0.05$, indicating a good model fit. See Table 7.

Table 7 RTX Group Population, Analysis of Factors Affecting Complete Remission (CR) in Treatment-Naïve Patients and Relapsed Patients

Parameter	Univariate Analysis				Multivariate Analysis			
	P	*OR*	95% CI		*P*	*OR*	95% CI	
			Lower Limit	Upper Limit			Lower Limit	Upper Limit
Type of treatment								
Treatment-naïve	Reference							
Relapsed	0.378	0.344	0.032	3.688				
Sex								
Male	Reference							
Female	0.999	807,737,507.4	<0.001	<0.001				
Age (years)	0.249	0.957	0.888	1.031				
Pathological diagnosis								
FSGS	Reference							
MCD	0.253	3.556	0.405	31.233				
BMI (kg/m^2)	0.888	0.982	0.766	1.26				
Baseline 24-h urine protein (g/24 h)	0.810	1.02	0.87	1.194				

(*Continued*)

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Table 7 (Continued).

Parameter	Univariate Analysis				Multivariate Analysis			
	P	*OR*	95% CI		*P*	*OR*	95% CI	
			Lower Limit	Upper Limit			Lower Limit	Upper Limit
Baseline serum albumin (g/L)	0.328	1.102	0.907	1.34				
Baseline serum creatinine (μmol/L)	0.061	0.971	0.942	1.001	0.660	0.969	0.842	1.115
Baseline eGFR (mL/min/1.73 m^2)	0.169	1.026	0.989	1.064	0.900	1.015	0.799	1.291
Baseline B (cells/μL)	0.637	1.003	0.992	1.014				
Baseline IgG (g/L)	0.079	0.636	0.383	1.054	0.716	0.860	0.381	1.939
Number of RTX infusions	0.971	0.99	0.561	1.744				
Total RTX dose (g)	0.532	0.666	0.187	2.38				

RTX Group Population, Analysis of Factors Influencing Relapse in Treatment-Naïve Patients and Relapsed Patients

Using relapse as the dependent variable (relapse = 1, no relapse = 0), we performed logistic regression analysis with 13 variables, including treatment type, sex, and age. Univariate regression analysis did not identify any factors that significantly influenced relapse in the RTX group (P > 0.05). Multivariate regression analysis also did not identify any independent factors that significantly influenced relapse (P > 0.05). The variance inflation factors (VIF) were 1.167–1.170, indicating no multicollinearity. The Hosmer–Lemeshow test showed no significant difference between the observed and predicted probabilities of complete remission ($\chi^2 = 3.510$, df = 8, P = 0.898), P > 0.05, indicating a good model fit. See Table 8.

Table 8 RTX Group Population, Analysis of Factors Influencing Relapse in Treatment-Naïve Patients and Relapsed Patients

Parameter	Univariate Analysis				Multivariate Analysis			
	P	*OR*	95% CI		*P*	*OR*	95% CI	
			Lower Limit	Upper Limit			Lower Limit	Upper Limit
Type of treatment								
Treatment-naïve	Reference							
Relapsed	0.999	587,445,280.2	<0.001	<0.001				
Sex								
Male	Reference							
Female	0.577	0.500	0.044	5.700				
Age (years)	0.357	0.956	0.868	1.052				
Pathological diagnosis								
FSGS	Reference							
MCD	0.823	0.75	0.061	9.270				
BMI (kg/m^2)	0.124	0.718	0.471	1.095	0.378	0.817	0.520	1.281
Baseline 24-h urine protein (g/24 h)	0.192	0.689	0.394	1.205	0.349	0.746	0.404	1.377
Baseline serum albumin (g/L)	0.794	1.018	0.893	1.160				
Baseline serum creatinine (μmol/L)	0.516	1.008	0.984	1.033				
Baseline eGFR (mL/min/1.73 m^2)	0.536	1.012	0.975	1.051				
Baseline B (cells/μL)	0.601	0.996	0.982	1.011				
Baseline IgG (g/L)	0.485	1.156	0.769	1.738				
Number of RTX infusions	0.438	1.253	0.709	2.215				
Total RTX dose (g)	0.576	1.481	0.374	5.856				

Table 9 Comparison of Relapses Before and After Re-Treatment with RTX

Parameter	Pre-Medication (n=15)	Post-Medication (n=15)	z/t Value	P-Value
Glucocorticoid use dosage (Prednisone mg/day)	34.00±15.69	7.67±7.76	7.854	<0.001
Frequency of recurrence (times/year)	1.00 (0.57, 1.82)	0 (0, 0.29)	−3.297	0.001
Time to first relapse (months)	0 (0, 0.33)	0 (0, 4.00)	−1.841	0.066
Average duration of maintenance of remission (months)	12.33±10.44	19.07±11.24	−1.700	0.111

Notes: Pre-medication is defined as the period before RTX re-treatment in patients undergoing repeat therapy; post-medication is defined as the period after RTX re-treatment.

Comparison of Relapses Before and After Re-Treatment with RTX

The paired samples *t*-test and Wilcoxon signed rank test found that in the retreatment population using RTX, the differences in time to first relapse and mean time to maintenance of remission were not statistically significant when comparing patients before and after medication ($P > 0.05$). The amount of glucocorticoid use and the frequency of relapses were lower in patients after medication than before medication ($P < 0.05$). See Table 9.

RTX Group Population, Survival Analysis of Relapse Prognosis in Treatment-Naïve Patients and Relapsed Patients

The Log-Rank (Mantel-Cox) test using the Kaplan-Meier method found no statistically significant difference in relapse rates between treatment-naïve and relapsed patients in the RTX group (χ^2=1.627, P=0.202), with a hazard ratio (HR) of 54.994 (95% CI: 0.001–5,521,742.802). In the RTX group population, the treatment-naïve patients did not experience any relapse, and the median time to relapse for relapsed patients was 42 months (95% CI: 22.38–61.62). See Figure 1.

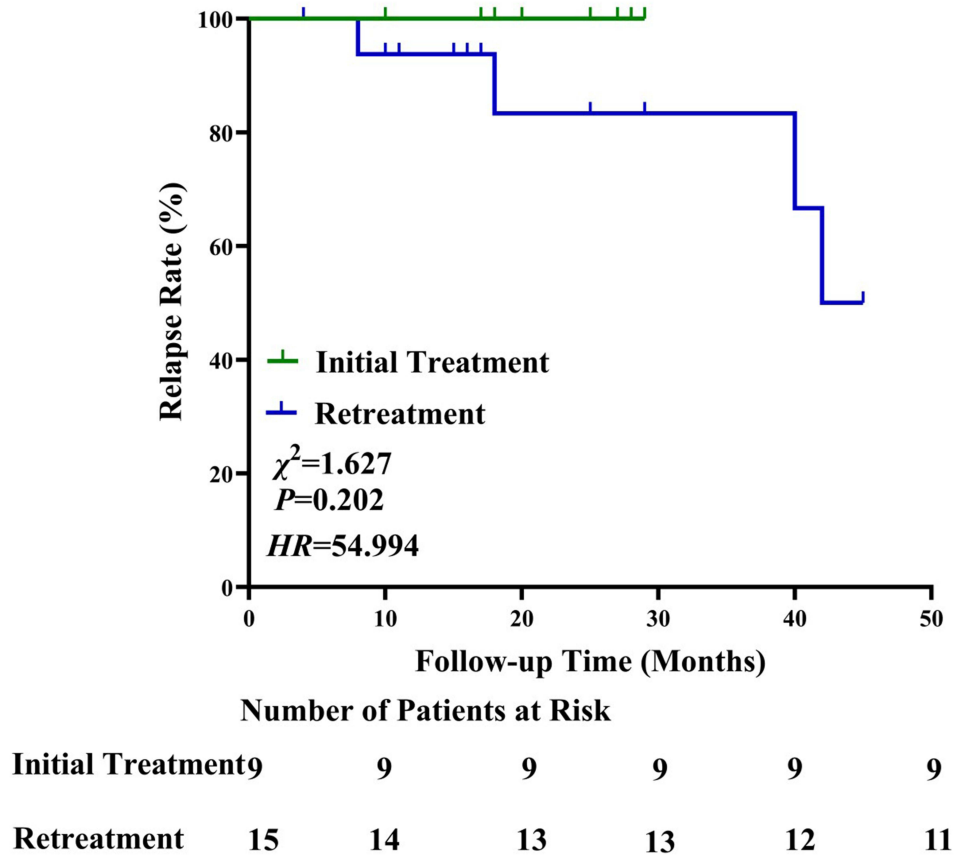

Figure 1 Survival Curves for Relapse Prognosis in Treatment-Naïve and Relapsed Patients of the RTX Group.
Notes: Due to the exceedingly Limited number of events, the HR estimate is rendered imprecise.

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Because only two relapses occurred, the HR confidence interval is extremely wide and should be interpreted with caution.

Survival Analysis of Prognosis for Relapse Before and After Medication in a Retreatment Population Using RTX

The Log-Rank (Mantel-Cox) test, using the Kaplan-Meier method, found that in the retreatment population using RTX, our data revealed statistically insignificant variation in relapse frequency pre- versus post-medication (χ^2= 1.448, *P*= 0.229), with a hazard ratio (HR) of 1.979 (95% CI: 0.618–6.340). In the retreatment population with RTX, the median time to relapse was 32 (18.555, 45.445) months for patients before medication and 42 (22.378, 61.622) months for patients after medication. See Figure 2.

Adverse Events in Adults with MCD or FSGS Treated with Rituximab

A total of six adverse events were recorded in six patients (FSGS, n = 3; MCD, n = 3). No infusion-related reactions —such as rash or fever—were observed. During follow-up, the most frequent adverse events were infections: two upper-respiratory-tract infections and three pulmonary infections. No severe or fatal adverse events occurred. See Table 10.

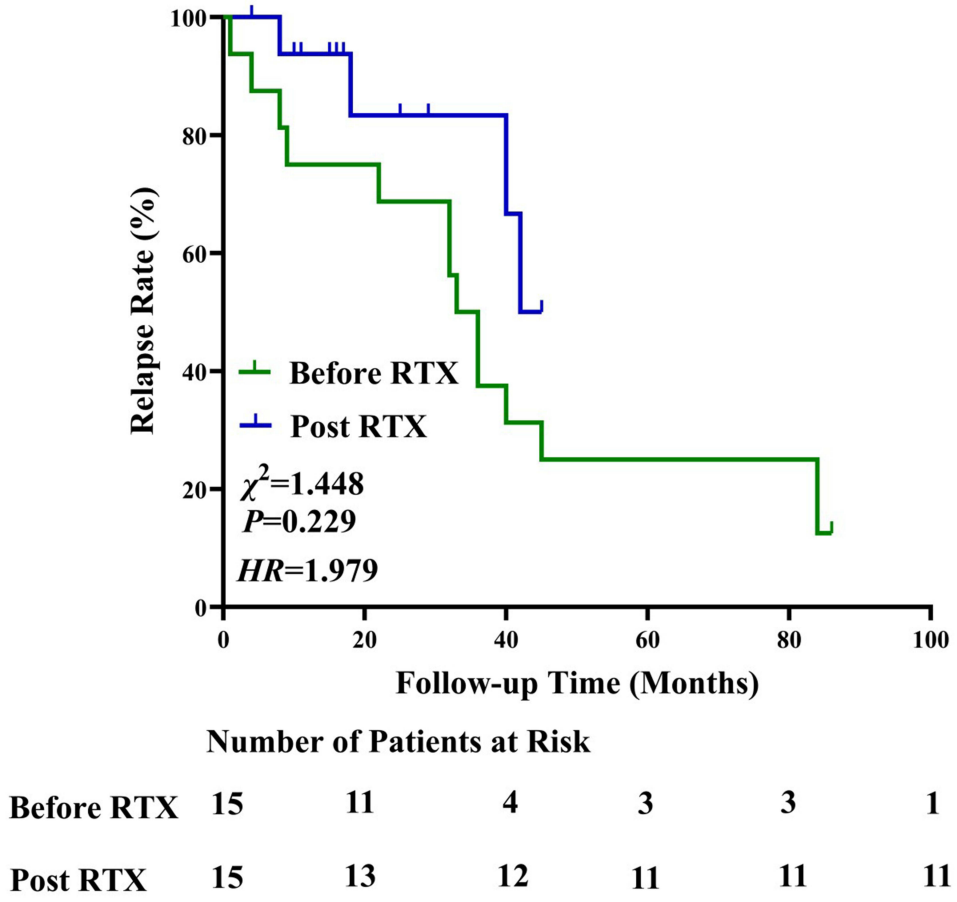

Figure 2 Survival curves for relapse prognosis before and after RTX treatment in relapsed patients.

Table 10 Adverse Events in Adults with MCD or FSGS Treated with Rituximab

Adverse Event	MCD (n=19)	FSGS (n=5)
Patients with adverse events	3	3
Times of adverse events	3	3
Fatal	0	0
Nonfatal		
Anemia	0	0
Herpes zoster	0	0
Skin rash	0	0
Chest tightness	0	0
Upper respiratory tract infection	1	1
Pulmonary infection	1	2
Pneumocystis pneumonia	0	0
Abnormal liver function	1	0
Urinary tract infection	0	0
Hair loss	0	0

Discussion

This study enrolled 82 adult patients with podocytopathies (minimal change disease [MCD] or focal segmental glomerulosclerosis [FSGS]) who visited the Department of Nephrology at our institution between January 2020 and December 2023. Participants were treated with either rituximab (RTX) or glucocorticoids to evaluate the efficacy and safety of RTX in managing podocytopathies, while also conducting a preliminary exploration of therapeutic regimens.

Effectiveness of RTX Treatment

In the study, we found no significant difference between the RTX group and the glucocorticoid (GC) treatment group in terms of remission rates, including Complete Remission (CR) and Partial Remission (PR). This result not only confirms the first-line status of glucocorticoids in the treatment of podocytopathies but also highlights the efficacy of RTX in adult patients, with a remission rate of 100%.[22–25] Additionally, the RTX group had significantly higher eGFR at the last follow-up than the glucocorticoid group, suggesting that RTX may be more protective of renal function. Given the higher proportion of FSGS and the shorter follow-up duration in the RTX cohort, the true efficacy of rituximab may have been underestimated. This finding is consistent with the 2018 randomized controlled trial in pediatric patients by Basu[26] This study imposed no lower limit on baseline renal function and therefore included two adult patients with podocytopathy whose eGFR was < 60 mL/min/1.73 m^2 (49 and 30 mL/min/1.73 m^2, respectively). Although the sample is small, neither patient progressed to ESKD during follow-up, further supporting the renal safety of RTX in individuals with reduced eGFR. Larger studies are warranted to confirm its efficacy and risk profile in patients with pre-existing renal impairment. Proteinuria selectivity index was not measured in the present study; however, the adult MCD/FSGS cohort reported by Allinovi et al suggests that a higher index is associated with an increased rate of complete remission following RTX.[27] If this finding is confirmed in future prospective studies, the index could serve as an auxiliary tool for identifying patients most likely to benefit from first-line RTX therapy. In the RTX group, there was no significant difference in remission rates between treatment-naïve and relapsed patients, indicating that RTX is equally effective in relapsed patients. Notably, baseline 24-hour urinary protein was higher in treatment-naïve than in relapsed patients (10.11 ± 5.47 vs 3.30 ± 2.18 g/24 h, P = 0.021). After including this variable in a multivariable logistic regression, baseline proteinuria did not significantly affect either complete remission (P = 0.143) or relapse (P = 0.217), suggesting that this imbalance did not materially alter the efficacy assessment. Nonetheless, residual confounding cannot be excluded and should be addressed in larger, prospective studies with appropriate matching techniques. Although the Complete Remission rate in treatment-naïve patients (88.9%) was not significantly higher than that in relapsed patients (73.3%) (P>0.05), we observed a higher complete remission rate in the treatment-naïve group. This difference may be related to less severe

podocyte injury (mainly foot process effacement)[28] and higher B cell activity in treatment-naïve patients, while relapsed patients may have progressed to focal sclerosis or fibrosis, thereby limiting the repair effects of RTX. Notably, the subgroup of treatment-naïve patients who received rituximab monotherapy represents a particularly interesting population. In this study, these patients achieved a high complete remission rate (88.9%) without the concomitant use of glucocorticoids, demonstrating the efficacy of RTX as a standalone initial treatment. This finding is of significant clinical relevance as it suggests that rituximab monotherapy could potentially serve as a first-line option for selected adult patients with podocytopathies, aiming to avoid steroid-related toxicities from the outset. The literature on this specific treatment strategy in adults remains scarce, making our observations a valuable contribution to the field.

Furthermore, treatment-naïve patients had lower baseline IgG levels (3.16 g/L), which significantly increased to 6.44 g/L after treatment, while relapsed patients maintained higher IgG levels both before and after treatment (baseline 6.57 g/L, last follow-up ≥5 g/L), possibly related to dynamic B cell homeostasis reconstruction. The long-term remission in relapsed patients may be associated with pathogenic antibody clearance: after B cell depletion, transitional B cells differentiate into new plasma cells during the recovery period (approximately 6–12 months), leading to IgG rebound, or long-lived plasma cells continuously secrete protective antibodies to maintain high titers.[29,30] This phenomenon is similar to the results of rituximab treatment in autoimmune diseases (such as lupus), where although B cell counts significantly decrease, protective antibody titers do not decline.[31] In addition treatment-naïve patients who have not experienced long-term glucocorticoids or immunosuppressive therapy and whose immune function is not suppressed[32] may have higher B cell activity and inflammatory factor (such as NF-κB) levels,[33,34] which may enhance the targeted therapeutic effect of RTX.

The Efficacy of Rituximab (RTX) in Sustaining Remission

To evaluate the efficacy of rituximab (RTX) in sustaining remission of podocytopathies, we conducted a comparative study between RTX and glucocorticoid therapy. Our findings revealed no statistically significant differences in relapse frequency or annualized relapse rates between the RTX and glucocorticoid groups. Furthermore, treatment-naïve patients receiving RTX demonstrated comparable effectiveness to relapsed/refractory patients in maintaining remission and reducing relapses, underscoring RTX's robustness as a therapeutic strategy.[24,35] Compared with previous studies that included only steroid-resistant MCD cases and had a smaller sample size (n = 17),[23] our study simultaneously enrolled both treatment-naïve and relapsed/refractory patients, thereby expanding the representativeness of the study population. These results align with recent randomized controlled trial (RCT) data from 2023, reinforcing RTX's role in long-term disease control.[20] Although the mean duration of maintained remission did not differ significantly between relapsed/refractory and treatment-naïve patients, relapsed/refractory patients treated with RTX achieved a dramatic reduction in glucocorticoid dosage from 34.00 ± 15.69 mg/day to 7.67 ± 7.76 mg/day ($P < 0.001$), with 40% (6/15) attaining complete steroid cessation. Furthermore, the annual relapse frequency in relapsed/refractory patients declined from 1.00 to 0 episodes/year ($P = 0.001$). These outcomes are in alignment with pediatric studies (eg, Fujinaga), where RTX enabled 82% steroid withdrawal and an 80% reduction in relapses within 2 years.[36] The recent RITERM multicentre long-term follow-up study has further confirmed that, in adult patients with MCD/FSGS (n = 183, 36-month follow-up), RTX treatment led to sustained relapse-free survival in 61% of participants and allowed discontinuation of all concomitant immunosuppressive agents. Rituximab thus facilitated both initial and long-term responses in the majority of adults with refractory glomerular disease while markedly reducing the need for other immunosuppressants.[37] In this study, relapsed/refractory patients treated with RTX showed an increase in the mean duration of maintained remission from 12.33 ± 10.44 months pre-treatment to 19.07 ± 11.24 months post-treatment, consistent with relapse-free survival rates and remission durations reported in prior studies.[19,38,39] This effect may be attributed to RTX's sustained depletion of B cells,[40] which effectively eliminates pathogenic B cells responsible for generating autoantibodies such as anti-annexin A2,[41] anti-UCHL1,[42] and anti-nephrin antibodies,[14] thereby stabilizing clinical remission. The trend toward steroid-free remission in maintaining remission after RTX treatment demonstrated by the relapsed patients in this study further support the therapeutic universality of RTX across age groups. This result not only implies that patients are free from the adverse effects of long-term steroid-related adverse effects, such as bone density loss, metabolic disorders and the risk of infection[3,4,43] but also reveals the excellence of RTX in maintaining remission in podocytopathies. Survival

and prognostic analyses revealed no statistically significant differences in relapse rates between treatment-naïve and relapsed/refractory patients within the RTX group (χ^2 = 1.627, P = 0.202). Similarly, comparisons of relapse patterns before and after RTX treatment in relapsed/refractory patients also showed no significant differences. Although statistical significance was not reached in the survival analyses, RTX effectively maintained remission in both cohorts while reducing glucocorticoid maintenance doses, with relapsed/refractory patients achieving an annual relapse frequency of zero episodes/year. The steroid-sparing effect of RTX mitigates long-term glucocorticoid-related complications (eg, metabolic disorders, infections), thereby improving patient outcomes. These findings align with a 2015 study demonstrating RTX's ability to reduce adverse drug reactions in steroid-dependent nephrotic syndrome.[44] Future research should expand sample sizes, dynamically monitor immune parameters, and extend follow-up durations to identify prognostic factors influencing survival outcomes under RTX therapy, ultimately guiding personalized treatment strategies.

Factors Affecting Remission

To evaluate factors influencing remission in podocytopathies treated with rituximab (RTX), we analyzed multiple clinical and laboratory variables, including treatment type, sex, age, pathological diagnosis, BMI, initial 24-hour urinary protein quantification, baseline serum albumin, baseline serum creatinine, baseline eGFR, baseline B cells, baseline immunoglobulins (IgG), RTX infusion frequency, and total RTX dose. Results from this study revealed no significant associations between these variables and Complete Remission or relapse rates ($P > 0.05$). These findings highlight the broad applicability of RTX in the treatment of podocytopathies, demonstrating consistent efficacy and safety across diverse patient subgroups, regardless of age, pathological subtype, baseline proteinuria, or renal function. This suggests that RTX is not constrained by disease severity and may serve as a first-line therapeutic option for treatment-naïve patients or a safer, more effective alternative for steroid-dependent podocytopathy patients. In contrast, a multicenter retrospective study in 2023 noted that serum albumin, serum creatinine and eGFR at baseline, as well as total lymphocyte count, CD4+ T-cell count, CD8+ T-cell count, and CD56+CD16+ NK-cell count after Rituximab (RTX) treatment, may be associated with the risk of relapse in patients with podocytopathies.[20] This may be different from the results of the present study due to the following factors: (1) The RTX dose selection was different, the present study used a standard dose of RTX (375 mg/m^2 x 4 infusions) whereas this 2023 study chose to give 375 mg/m^2 x 2 full doses of Rituximab (RTX) over 2 weeks, and then analyzed the counts of the lymphocytes in the peripheral blood, including in particular the CD19+ B-cells. B-cell depletion (BCD) was defined as a peripheral blood count of CD19+ B cells below 5 cells/L, as assessed by flow cytometry. If BCD was achieved after two complete doses, the first course of Rituximab (RTX) was completed. Otherwise, additional doses were added every 2 weeks until BCD was achieved; (2) Differences in patient enrollment may account for the differences in outcomes, as this study included patients who were treatment-naïve and did not develop steroid dependency or immunosuppressive-related complications, whereas the 2023 study exclusively involved patients who were steroid-dependent or frequently relapsed, which may affect their risk of podocyte relapse after RTX treatment; (3) The length of follow up Impact: The median follow-up time for patients enrolled in this study was 2.08 years for the RTX treatment-naïve patients group and 1.42 years for relapsed patients, whereas this 2023 study was set at 2 years. Previous KIDGO guidelines noted that especially patients with FSGS and low baseline eGFR have a risk of relapse that may be seen in the longer term; (4) Limited patient enrollment: As a preliminary exploratory study, our research was constrained by the availability of cases, resulting in a relatively small sample size. This limitation likely contributed to the failure of the multivariate analysis to identify any predictive factors for RTX efficacy. These discrepancies may explain the divergent findings. In our cohort, we observed no statistically significant relationship between the duration of B-cell depletion and subsequent relapse; this limitation likely stems from the small sample size and remains one of the unresolved mechanistic questions of this study. Future investigations in larger cohorts are required to further delineate the association between these variables and Rituximab (RTX) efficacy, thereby deepening our understanding of the mechanisms underlying RTX treatment of podocytopathies.

Analysis of Adverse Events

The study found that despite lower baseline IgG levels in treatment-naïve patients within the RTX group (3.16 g/L), RTX therapy did not exacerbate hypogammaglobulinemia. During follow-up, no significant hypogammaglobulinemia was observed, with post-treatment IgG levels in treatment-naïve patients rising to 6.44 g/L. This result is consistent with current consensus on RTX use in immune-mediated kidney diseases. In the RTX treatment process, dynamic immune system reconstruction typically lasts 6–12 months, during which B cell depletion occurs and IgG levels gradually recover.[45] Additionally, RTX reduces pathogenic antibodies (such as anti-nephrin antibodies),[13,14] stabilizing the podocyte barrier and improving podocyte function-related clinical manifestations, such as significant reduction in proteinuria (24-hUP from baseline 4.28 g to last follow-up 0.16 g) and eGFR stabilization (from baseline 103.00 mL/min/1.73m^2 to last follow-up 124.25 mL/min/1.73m^2). Although direct observation of podocyte structural repair requires repeated renal biopsy (clinically limited), improvements in the above clinical indicators indirectly support RTX's repair effect on podocytes. During follow-up, no serious adverse events such as fever, rash, hepatitis B reactivation, thrombocytopenia, or leukopenia occurred in the RTX group. A total of six adverse events were recorded, primarily mild-to-moderate infections (upper respiratory tract infection, n = 2; pulmonary infection, n = 3); no severe or fatal adverse events were observed. All patients completed the full treatment course without discontinuation due to adverse events, indicating a manageable long-term safety profile.[20] Similarly, anti-nephrin antibody data were excluded from the present analysis owing to the very low positivity rate observed in our initial screening.

Limitations of the Study and Future Directions

This study has the following limitations: (1) It is a single-center study with a small sample size (n=24 in the RTX group), which may limit statistical power for subgroup analysis between treatment-naïve and relapsed patients, affecting accurate assessment of intergroup differences. Future studies should consider expanding the sample size to improve statistical power and result reliability. (2) Quality of life-related indicators (such as bone mineral density and lipid levels) were not included, making it difficult to comprehensively evaluate long-term risks and impact on patient quality of life. Future studies should include these indicators to more comprehensively assess RTX's long-term efficacy and safety. (3) Although biomarkers (such as anti-nephrin antibodies, inflammatory factors, etc.) were tested, dynamic monitoring was lacking, limiting in-depth exploration of RTX's mechanism. Future studies should include dynamic biomarker monitoring to better understand RTX's efficacy mechanism and predict individualized treatment responses. (4) This prospective analysis has inherent biases, potentially affecting result reliability and generalizability. Future studies should adopt a randomized controlled trial (RCT) design and conduct multicenter RCTs to reduce bias and improve result credibility. Future studies should adopt the registration model of the RITERM study to conduct a prospective, multicenter RCT for validating the efficacy and safety of RTX in patients across different renal function stages.

Conclusion

In adult patients with podocytopathies, rituximab (RTX) demonstrates efficacy comparable to that of glucocorticoids, significantly improves renal function, reduces steroid dependence, and exhibits a favorable safety profile. Descriptive data showed that treatment-naïve patients receiving RTX experienced no glucocorticoid-related adverse events, whereas relapsed patients exhibiteda statistically significant reduction in steroid dosage and a decreasing trend in relapse frequency after RTX, although the latter did not reach statistical significance. We therefore speculate that RTX may represent an option for patients who require long-term immunosuppression and have no contraindications. For patients who require long-term immunosuppression and have no contraindications, RTX may be a preferred choice. In this study, no severe adverse events were observed during RTX administration or follow-up, preliminarily verifying its safety. Given the small sample size, the findings should be interpreted cautiously. These findings provide high-quality preliminary evidence for RTX's clinical application in podocytopathy treatment. However, due to study limitations (such as its single-center design, small sample size) long-term efficacy and mechanism of RTX need further validation through multicenter randomized controlled trials to comprehensively evaluate its value in podocytopathy treatment.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of the First Affiliated Hospital of Bengbu Medical college (Approval No. [2020]117). Informed consent was obtained from all individual participants included in the study.

Author Contributions

All authors made substantial contributions to the reported work, whether in the conception, study design, execution, data acquisition, analysis, and interpretation, or in all these areas; participated in drafting, revising, or critically reviewing the manuscript; approved the final version to be published; agreed on the target journal for submission; and agreed to be accountable for all aspects of the work.

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Disclosure

The author(s) report no conflicts of interest in this work.

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