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ORIGINAL RESEARCH

Efficacy and Safety of Telitacicept in IgA Nephropathy and IgA Vasculitis-Associated Nephritis in Children: A Retrospective Single-Centre Study

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Background: Childhood IgA nephropathy (IgAN) and IgA Vasculitis-associated nephritis (IgAVN) are same in pathogenesis and still a challenge to pediatricians.

Purpose: To evaluate the efficacy and safety of Telitacicept in pediatric patients with IgAN or IgAVN.

Patients and Methods: This was a retrospective single-centre study of pediatric IgAN or IgAVN with urine protein-to-creatinine ratio (UPCR) over 500 μg/mg who were treated with subcutaneous administration of Telitacicept and followed for at least 6 months. The effects on induction of remission and side effects were evaluated.

Results: 13 pediatric patients including 7 with IgAN and 6 with IgAVN were enrolled in the study. After 6-month treatment, 6 of 13 (46.15%) patients achieved remission, 4 (30.77%) patient achieved complete remission. The median percent reduction in UPCR was 90.74% (84.38%, 94.76%). The median percent reduction in urine red blood cells count was 96.21% (75.33%, 98.99%). Compared with baseline, serum albumin increased significantly [$P < 0.001$, 95% CI (−10.30, −4.04)], and estimated glomerular filtration rate (eGFR) remained stable and within the normal range. Significant reductions were observed in levels of IgA [$P < 0.001$; 95% CI (1.15, 2.49)], IgM [$P = 0.002$; 95% CI (0.53, 1.08)], as well as CD19+ lymphocytes [$P = 0.012$; 95% CI (6.63, 34.67)]. The average follow-up was 6.5 ± 0.8 months, 5 patients achieved glucocorticoid discontinuation. No severe adverse events were observed.

Conclusion: Telitacicept was safe in pediatric IgAN/IgAVN patients and could significantly induce renal remission through reducing proteinuria and hematuria.

Keywords: telitacicept, IgA nephropathy, IgA vasculitis-associated nephritis, pediatric, efficacy, safety

Introduction

IgA nephropathy (IgAN) and IgA vasculitis-associated nephritis (IgAVN) are the most common autoimmune glomerular diseases.[1–3] Both disease pose a risk of progression to end-stage renal disease (ESRD) despite current treatment strategies. In particular, IgAN is the most typical primary glomerular disease globally, with up to 40% of patients eventually developing ESRD.[4,5]

In the current clinical practice for the treatment of these diseases drugs primarily include corticosteroids and immunosuppressants, in addition to symptomatic and supportive treatment. Despite their efficacy in reducing inflammation, these treatments are often limited by their poor curative potential and significant side effects.[6]

A well-established pathogenic link exists between IgAN and IgAVN, centred on aberrantly glycosylated IgA1 molecules, specifically galactose-deficient IgA1 (Gd-IgA1).[3,7,8] This shared mechanism operates through a defined pathogenic cascade, which consists of the production of Gd-IgA1, generation of IgG autoantibodies against Gd-IgA1, formation of pathogenic immune complexes, and their deposition in the kidneys, leading to glomerular injury.[9] This four-

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step hypothesis was originally proposed for IgAN, which has been extended to IgAVN, providing unified mechanistic insights into both diseases.[10]

B cell dysregulation and the overproduction of pathogenic Gd-IgA1 are mediated by the cytokines B-cell activating factor (BAFF) and a proliferation-inducing ligand (APRIL).[11,12] Telitacicept is a fusion protein comprising a recombinant transmembrane activator and calcium modulator and cyclophilin ligand interactor receptor fused to the fragment domain of human IgG. Telitacicept binds BAFF and APRIL, thereby inhibiting the development and survival of plasma cells and mature B cells.[13,14] Clinical trials in adults with IgAN[15] and systemic lupus erythematosus (SLE)[16] have shown that Telitacicept significantly reduces proteinuria with a favorable safety profile.

These results collectively suggest that Telitacicept may represent a novel therapeutic strategy for IgAN and IgAVN. Pediatric patients confront distinct challenges, including a higher risk of relapse, unique growth and developmental concerns, and a lack of clinical trial data specific to this population. In this clinical practice, Telitacicept was administered to pediatric patients with IgAN and IgAVN to evaluate its therapeutic efficacy and safety.

Patients and Methods

A retrospective analysis was conducted on pediatric patients diagnosed with IgAN or IgAVN between January 2023 and December 2024 in the Department of Pediatrics at Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology, who received Telitacicept treatment. The criteria for inclusion were as follows: (1) a diagnosis of IgAN or IgAVN confirmed by renal biopsy; (2) urine protein-to-creatinine ratio (UPCR) > 500 μg/mg; (3) received Telitacicept treatment. The criteria for exclusion were as follows: (1) secondary IgAN or IgAVN; (2) age older than 18 years; (3) patients who received rituximab or other biologics within 6–12 months; (4) UPCR ≦ 500 μg/mg.

All patients were administered weekly subcutaneous injections of Telitacicept, with a treatment plan consisting of either 80mg (weighing ≦ 40 kg) or 160mg (weighing > 40 kg) doses. Patients continued to receive ACE inhibitors/ARBs, corticosteroids, or other immunosuppressants throughout Telitacicept treatment, and tapering within the permitted range was also allowed. The follow-up period after Telitacicept treatment was 6 months. For remission was characterized by the resolution of proteinuria (UPCR <200 μg/mg) coupled with normal estimated glomerular filtration rate (eGFR)≥90 mL/min/1.73 m^2 or stable renal function. Complete remission additionally requires the resolution of hematuria, defined as <5 RBCs/HPF or a negative urine dipstick.[17]

Observational Indicator

The primary observational indicators were the changes in UPCR, eGFR, serum albumin, and urine red blood cells counts after treatment compared with baseline. eGFR was calculated using the Schwarz equation for children, with the formula: eGFR=0.413 × height (cm) / serum creatinine (mg/dL).[18] The secondary observational indicators were the changes serum urea, serum uric acid, serum immunoglobulins (IgA, IgG, IgM), complement (C3, C4) and lymphocyte subsets. Adverse events (AEs) were systematically recorded and graded according to Common Terminology Criteria for Adverse Events Version 5.0.

Statistical Methods

The normality of distribution for all continuous variables is assessed using the Shapiro–Wilk test. For normally distributed data, results are expressed as mean ± standard deviation (SD). For non-normally distributed data, results are reported as median with interquartile range (IQR). Matched groups were compared using the paired *t*-test or the Wilcoxon matched-pairs signed rank test, depending on the results of a normality test for data distribution. Analyses are performed using SPSS v26.0 (IBM Corp) and R programming language (version 4.5.1). Statistical significance was defined as a P-value < 0.05.

Results

Baseline Characteristics of Patients

This study enrolled 13 pediatric patients (7 with IgAN and 6 with IgAVN), comprising 6 females (46.2%) and 7 males (53.8%). Baseline clinical characteristics before Telitacicept initiation were summarized in Table 1. Prior to Telitacicept

Table 1 Baseline Characteristics, Treatment Regimens, and Clinical Outcomes of Pediatric Patients with IgAN or IgAVN Treated with Telitacicept

NO	Sex	Age at Initiation of Telitacicept (Years)	Disease Course and Treatment History (Months)	Pathological Diagnosis	Telitacicept (mg/wk)	UPCR (μg/mg)			Urine RBC Count (μL)			P Tapering Post-Treatment	AEs
						Before Starting Treatment	3m	6m	Before Starting Treatment	3m	6m		
1	Female	11.8	Supportive care for 0.2m	IgAN (WHO Class III)	160	538.76	90.42	78.90	208.3	38.9	36.0	2.5mg/d at 6m	No
2	Male	13.8	UPCR 2677.69 μg/mg; P/ CTX for 6m; P/ TAC/MMF for 6m	IgAN (WHO Class V)	160	1058.57	475.69	247.88	13.8	8.1	5.0	12.5mg/d at 6m	No
3	Male	5.0	24h UP: 5.4 g; P/CTX for 1m	IgAN (Lee's III)	80	1465.12	154.42	120.71	2892.7	134.5	36.8	Stopping at 6m	Respiratory tract infection
4	Female	14.3	24h UP: 1.27g; P/TAC for 3m	IgAN (WHO Class III)	160	966.86	NA	83.22	166.8	16.3	53.5	2.5mg/d at 6m	No
5	Male	12.6	Supportive care for 0.2m	IgAN (Lee's IV)	160	569.7	106.56	93.77	6883.7	117.2	11.2	2.5mg/d at 6m	No
6	Male	12.3	24h UP: 4.76g; P/CTX for 2.5m	IgAN (Lee's V)	160	4160.82	113.97	112.75	9793.8	1923.6	74.4	2.5mg/d at 6m	No
7	Female	10.6	UPCR 708.71μg/mg; P/MMF for 10m	IgAN (WHO Class III)	80	749.39	NA	82.64	1449.2	111.2	33	Stopping at 6m	No
8	Female	11.0	UPCR 286.68 μg/mg; P/TAC for 6m	IgAVN (ISKDC IIIa)	160	1179.09	261.42	109.18	138.1	17.4	117.7	Stopping at 6m	Local injection site reactions
9	Male	11.3	UPCR 172.68 μg/mg; P for 5m	IgAVN (ISKDC IIa)	160	841.54	129.89	124.36	194.8	283.0	9.5	Stopping at 6m	No

(*Continued*)

Table 1 (Continued).

NO	Sex	Age at Initiation of Telitacicept (Years)	Disease Course and Treatment History (Months)	Pathological Diagnosis	Telitacicept (mg/wk)	UPCR (μg/mg)			Urine RBC Count (μL)			P Tapering Post-Treatment	AEs
						Before Starting Treatment	3m	6m	Before Starting Treatment	3m	6m		
10	Male	3.7	Urine protein 3+; P/CTX for 2m	IgAVN (ISKDC V)	80	2384.63	351.23	940.52	1065.4	52.4	25.4	Stopping at 4m	Local injection site reactions
11	Female	11.6	Urine protein 2+; P for 4m	IgAVN (ISKDC V)	160	3838.28	355.82	200.45	2666.7	628.5	152.2	10mg/d at 6m	No
12	Male	10.0	UPCR 2020.20 μg/mg; P/MMF for 1m	IgAVN (ISKDC IIIa)	160	1534.9	69.11	78.89	1534.94	47.7	8.4	2.5mg/d at 6m	No
13	Female	12.4	UPCR 2164.18 μg/mg; P/MMF for 1m	IgAVN (ISKDC IIIa)	160	1443.11	99.59	75.84	3150.3	298.0	119.3	2.5mg/d at 6m	No

Note: NA indicates that the UPCR was not assessed.

Abbreviations: UPCR, urine protein-to-creatinine ratio; RBC, red blood cells; 24h UP, 24 hour urinary protein excretion; IgAN, IgA nephropathy; IgAVN, IgA vasculitis-associated nephritis; CTX, cyclophosphamide pulse; P, prednisone; MMF, mycophenolate mofetil; TAC, tacrolimus; AEs, adverse events; m, month ;d, day.

initiation, 11 patients received the following immunosuppressive therapies: methylprednisolone pulse (3/13), oral glucocorticoids (11/13), cyclophosphamide pulse (4/13), mycophenolate mofetil (4/13), and tacrolimus (3/13).

Efficacy

The initial dosage of Telitacicept treatment was 160 mg per week in 10 patients (76.9%) and 80 mg per week in 3 patients (23.1%). A significant improvement in UPCR was observed at 2 months following Telitacicept treatment (Figure 1A). The level of UPCR decreased from 1179.0 (795.5, 1960.0) to 278.0 (123.9, 450.2) μg/mg (P = 0.002), and the median reduction in UPCR reached 81.93% (75.62%, 86.26%) at 2 months (Figure 1B). After 6-month treatment, the level of UPCR decreased from 1179.0 (795.5, 1960.0) to 109.2 (80.77, 162.4) μg/mg (P = 0.001), as shown in Table 2, with a median percent reduction in UPCR was 90.74% (84.38%, 94.76%).

A significant improvement in urine red blood cells count was observed at 1-month follow-up (Figure 1C). The level of urine red blood cells count decreased from 1449.2 (180.8, 3021.5) to 336.8 (85.10, 981.5) μL (P = 0.01), and the median reduction in urine red blood cells count reached 71.62% (41.08%, 91.97%) at 1 month (Figure 1D). After 6-month treatment, the level of urine red blood cells count decreased from 1449.2 (180.8, 3021.5) to 36.0 (10.35, 96.05) μL (P = 0.001), as shown in Table 2, with a median percent reduction in urine red blood cells count decreased was 96.21% (75.33%, 98.99%). Significant improvement in serum albumin (Figure 1E and F) and serum urea (Figure 1G and H) at follow-up were noted. The uric acid (Figure 1I and J) and eGFR (Figure 1K and L) level remained stable during the entire follow-up period, detailed results were presented in Table 2.

At the 6-month follow-up, Telitacicept treatment led to a significant reduction in serum levels of immunoglobulins, with a particularly marked decrease in IgA (2.60 ± 1.16 vs 0.78 ± 0.49 g/L, $P < 0.001$) and IgM [0.85 (0.74, 1.13) vs 0.19 (0.12, 0.27) g/L, P = 0.002]. The longitudinal changes in IgA (Figure 2A and B), IgM (Figure 2C and D), and IgG (Figure 2E and F) were shown in Figure 2. A significant reduction was also observed in the CD19+ B lymphocyte subset [20.19 (11.69, 33.27) vs 7.13 (3.24, 11.41)%, P = 0.012], this change was shown in Figure 2G and H. In contrast, complement C3 levels (Figure 2I and J), and complement C4 levels (Figure 2K and L) remained unchanged.

At the final follow-up, the mean duration of follow-up was 6.5 ± 0.8 months. Corticosteroids had been discontinued in 5 patients (Table 1). Among 6 of 13 (46.15%) patients attained remission and 4 (30.77%) attained complete remission, remission and complete remission rates at each time point were presented in Supplementary Table 1.

AEs

No severe AEs were reported. Among the 13 patients, 3 AEs were observed (Table 1). 1 cases of respiratory tract infection were relieved upon antibiotic treatment, and 2 cases of local injection site reactions were resolved spontaneously without treatment.

Discussion

Currently, limited clinical reports and data exist on the use of Telitacicept in IgAN[19–21] and IgAVN.[22] Therefore, our study collected clinical data from nine patients with IgAN and IgAVN treated with Telitacicept at a single center. By analyzing the treatment process, laboratory data before and after treatment, and AEs, we aimed to evaluate the efficacy and safety of Telitacicept in this patient population.

This study enrolled 13 patients with severe proteinuria prior to Telitacicept therapy. The UPCR showed a significant decrease at the 2-month follow-up after treatment, which is consistent with the results reported in both single-centre and multicentre studies.[20,22] Additionally, improvements were observed in serum albumin, and urinary red blood cell counts compared with the baseline levels before Telitacicept treatment. A study focusing on refractory childhood IgAVN revealed that Telitacicept administration led to a significant reduction in proteinuria levels among the majority of participants. This single-center, retrospective observational study included seven children who had previously been treated with glucocorticoids and at least one immunosuppressants therapy but continued to exhibit proteinuria. The results indicated that six out of the seven children experienced a reduction in proteinuria, with two achieving complete remission characterized. No serious adverse reactions were reported, underscoring the safety profile of Telitacicept in this

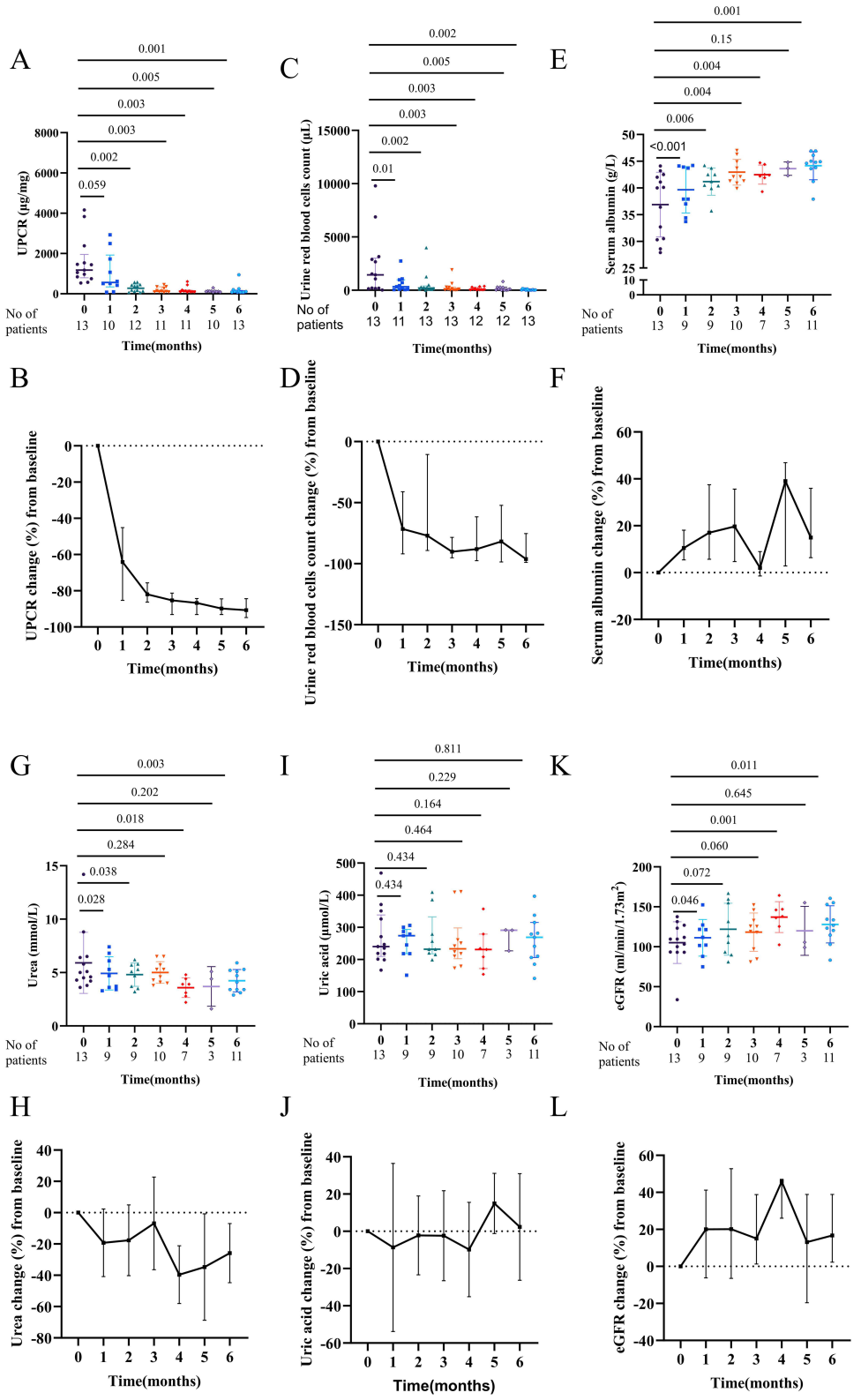

Figure 1 UPCR, urine red blood cells count and kidney function results during follow-up. (**A**) UPCR; (**B**) UPCR change (%) from baseline; (**C**) urine red blood cells count; (**D**) urine red blood cells change (%) from baseline; (**E**) serum albumin; (**F**) serum albumin change (%) from baseline; (**G**) serum urea; (**H**) serum urea change (%) from baseline;(**I**) serum uric acid; (**J**) serum uric acid change (%) from baseline; (**K**) eGFR;(**L**) eGFR change (%) from baseline. *P*-value, compare to baseline.
Abbreviations: UPCR, urine protein-to-creatinine ratio; eGFR, estimated glomerular filtration rate.

Table 2 Comparison of Pre-Treatment and Post-Treatment Laboratory Test Outcomes Following Telitacicept Therapy

Laboratory Index	Pre-Treatment	Post-Treatment	*P*	95% CI
UPCR (μg/mg)	1179.0 (795.5, 1960.0)	109.2 (80.77, 162.4)	0.001	772.92, 2262.0
Urine red blood cells count (μL)	1449.2 (180.8, 3021.5)	36.0 (10.35, 96.05)	0.001	576.65, 4199.52
Serum albumin (g/L)	36.88 ± 6.0	44.05 ± 2.47	<0.001	−10.30, −4.04
eGFR (mL/min/1.73m^2)	109.5 (93.27, 125.1)	133.5 (117.5, 149.9)	0.003	−40.42, −10.99
Serum urea (mmol/L)	5.00 (4.25, 6.45)	3.4 (3.10, 5.15)	0.001	0.75, 2.4
Serum uric acid (μmol/L)	270.3 ± 85.57	253.5 ± 73.80	0.425	−27.49, 61.09
Serum IgA (g/L)	2.60 ± 1.16	0.78 ± 0.49	<0.001	1.15, 2.49
Serum IgM (g/L)	0.85 (0.74, 1.13)	0.19 (0.12, 0.27)	0.002	0.53, 1.08
Serum IgG (g/L)	6.27 ± 3.28	4.97 ± 1.37	0.111	−0.35, 2.95
Complement C3 (g/L)	0.98 ± 0.17	0.85 ± 0.13	0.045	0.0035, 0.26
Complement C4 (g/L)	0.22 ± 0.06	0.19 ± 0.06	0.067	−0.0026, 0.068
CD19$^+$B lymphocyte subset (%)	20.19 (11.69, 33.27)	7.13 (3.24, 11.41)	0.012	6.63, 34.67

Notes: Data are present mean ± SD or median (IQR). Pre-treatment indicates baseline values measured prior to Telitacicept therapy. Post-treatment indicates values measured at the last follow-up after Telitacicept therapy.
Abbreviations: UPCR, urine protein-to-creatinine ratio; eGFR, estimated glomerular filtration rate; 95% CI, 95% confidence interval.

cohort.[23] These findings suggest that Telitacicept may be a promising therapeutic option for pediatric IgAVN, although larger prospective studies are warranted.

The clinical efficacy observed in this study is supported by the immunologic profile changes consistent with Telitacicept's dual-target mechanism. Telitacicept exerts its mechanism of action by simultaneously binding to both BAFF and APRIL, thereby blocking their interactions with the receptors. This dual inhibition effectively suppresses aberrant B-cell activation, leading to reduced production of Gd-IgA1, and also impedes plasma cell differentiation, which subsequently lowers the levels of pathogenic autoantibodies.[24] Consistent with this mechanism, Telitacicept treatment led to significant reductions in IgA, IgM and CD19+ B lymphocyte subset levels, whereas IgG levels did not decrease markedly. These results are largely consistent with previous reports.[22] Telitacicept had a low incidence of AEs, with no severe adverse reactions reported. A case series investigating the safety and efficacy of Telitacicept in children with biopsy-confirmed IgAVN reported significant reductions in 24-hour urinary protein levels, as well as decreases in CD19+ B lymphocyte subset, IgA, and IgM levels. The study also noted improvements in serum albumin and complement C4 levels, further supporting the therapeutic benefits of Telitacicept. The absence of drug reactions in the enrolled patients reinforces its safety and tolerability as an adjunct to standard treatment.[25] At the same time, the importance of immunologic monitoring should be emphasized. When using telitacicept, attention should be given to preventive measures, including avoidance of live-attenuated vaccines in the short term. For patients requiring long-term follow-up, regular reassessment of immunoglobulin levels and lymphocyte subsets is recommended to monitor the degree of immunosuppression and the associated risk of infection.

The 6-month follow-up period allows only the evaluation of short-term efficacy and preliminary safety. Therefore, our study has certain limitations. First, the small sample size and heterogeneous patient population may reduce statistical power and introduce selection bias, necessitating validation in larger, multicenter studies. Second, renal outcomes such as sustained eGFR stabilization or progression to ESRD require longer observation, and the short follow-up is insufficient to determine the long-

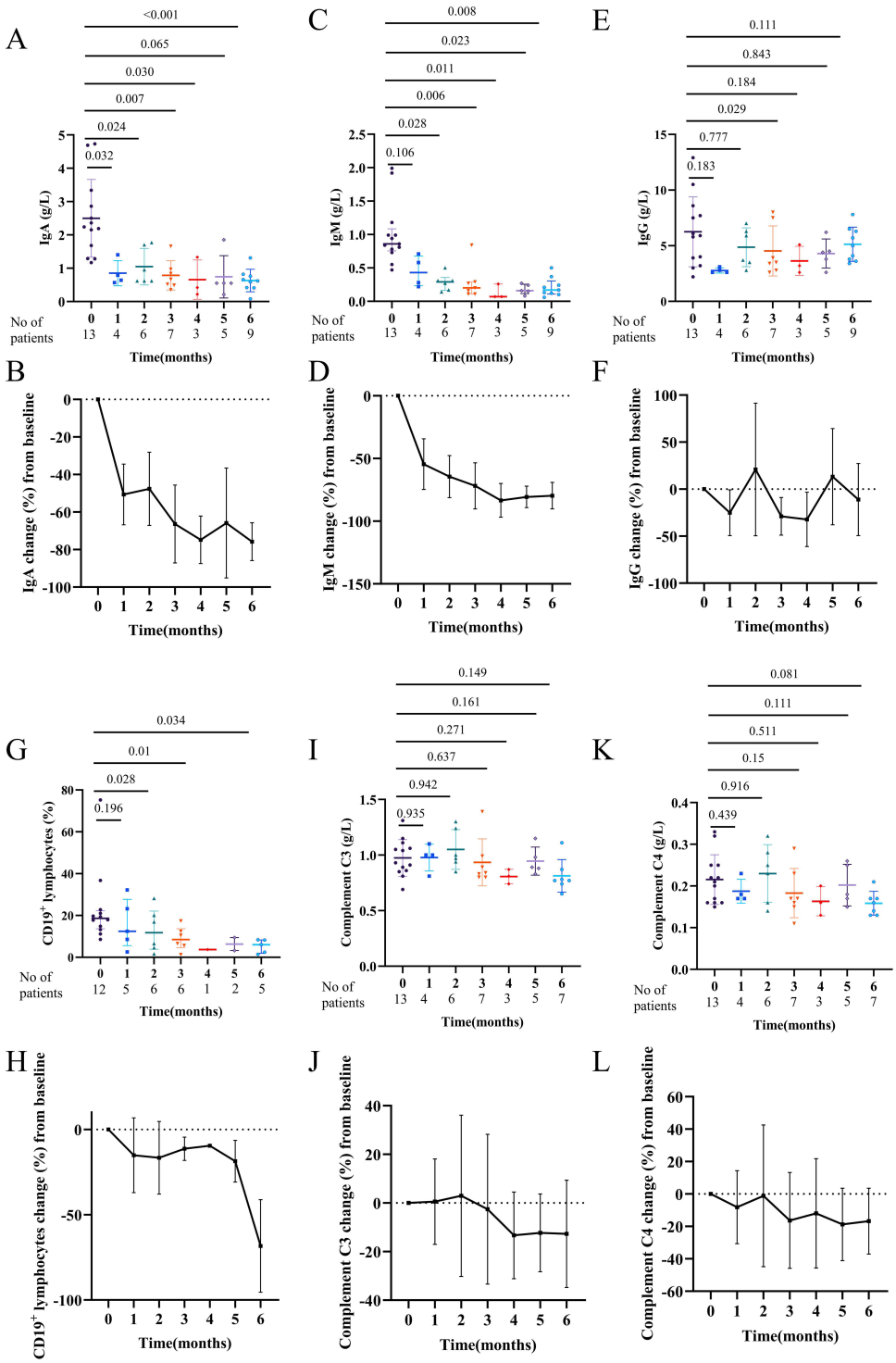

Figure 2 Immune function results during follow-up. (**A**) serum IgA; (**B**) serum IgA change (%) from baseline; (**C**) serum IgM; (**D**) serum IgM change (%) from baseline; (**E**) serum IgG; (**F**) serum IgG change (%) from baseline; (**G**) CD19+B lymphocyte subset; (**H**) CD19+B lymphocyte subset change (%) from baseline; (**I**) complement C3; (**J**) complement C3 change (%) from baseline; (**K**) complement C4; (**L**) complement C4 change (%) from baseline. *P*-value, compare to baseline.

term renal protective effects. Finally, most enrolled children were receiving corticosteroids, other immunosuppressive agents, or ACE inhibitors/ARBs therapy at baseline, so the observed therapeutic effects may partially reflect these concomitant treatments. Future controlled, prospective studies are needed to clarify the independent efficacy and long-term safety of Telitacicept.

Conclusion

Telitacicept showed significant efficacy in reducing proteinuria, hematuria and improving serum albumin while preserving renal function in pediatric IgAN/IgAVN patients, indicating a potential clinical benefit. 5 patients discontinued corticosteroid therapy after Telitacicept treatment. The treatment maintained a favorable safety profile throughout the study. The significant reductions in IgA, IgM, and CD19+ B cells provide mechanistic support for Telitacicept's efficacy. This study is limited by its small sample size, retrospective design, and short follow-up, thus necessitating larger, long-term future studies.

Data Sharing Statement

The datasets generated or analyzed in this study are also available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

This study was approved by The Human Ethics Committees of the Tongji Hospital, Tongji Medical College, Huazhong University of Science and Technology. Approval number: TJ-IRB202506039. The guardians of the patients were fully informed about the purpose of the research, in accordance with the Declaration of Helsinki. Written informed consent was obtained from the parents or legal guardians of all participants. All patient data were handled confidentially.

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Disclosure

The authors report no conflicts of interest in this work.

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