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CASE SERIES

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Single-dose telitacicept therapy for refractory idiopathic membranous nephropathy: A case series

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Key Clinical Message

We report three cases of IMN from our center, where patients received a single dose of telitacicept after showing no response to conventional treatments. Although one case did not respond, the other two cases achieved complete or partial remission of proteinuria. These cases illustrates the telitacicept may offer new hope for the treatment of IMN.

Abstract

Despite the variety of treatment options available, effective therapies for refractory membranous nephropathy remain lacking. Recently, some reports have suggested that telitacicept is a new therapeutic option. However, only a few published studies have documented the use of telitacicept for treating idiopathic membranous nephropathy (IMN). We present three cases of IMN from our center, where patients received a single dose of telitacicept after showing no response to conventional treatments, including glucocorticoids, tacrolimus, mycophenolate mofetil, cyclophosphamide, cyclosporine, and rituximab. Although one case did not respond, the other two cases achieved complete or partial remission of proteinuria. Thus, telitacicept may offer new hope for the treatment of refractory membranous nephropathy.

KEYWORDS

case series, refractory membranous nephropathy, telitacicept, therapy

1 | INTRODUCTION

Idiopathic membranous nephropathy (IMN) is a common pathological type of glomerular disease, ranking second among primary glomerular diseases, with an increasing incidence rate. The histological hallmark of IMN is the deposition of immune complexes in the subepithelial space of the glomerular filtration barrier. Untreated IMN patients have a 10-year renal survival rate of 60%–80%.[1] Over the past decade, there has been

significant progress in understanding the pathogenesis of IMN. IMN is now considered an autoimmune disease.[2] Unlike other autoimmune kidney diseases, pathogenic circulating autoantibodies against the M-type phospholipase A2 receptor (PLA2R1) and thrombospondin type-1 domain-containing 7A (THSD7A) on podocytes are considered the main factors leading to IMN.[3] The 2021 Kidney Disease Improving Global Outcomes (KDIGO) guidelines emphasize selecting appropriate treatment based on clinical risk assessment of progressive renal function loss.[4] Clinical studies on IMN have confirmed remission rates of 57%–89% with rituximab treatment.[5,6] The remission rates with glucocorticoid combined with cyclophosphamide (CYC) and glucocorticoid combined with tacrolimus are approximately 88% and 53%, respectively.[7] Conventional therapies consisting of corticosteroids and immunosuppressants can have significant side effects and are not effective for all patients. Regardless of the treatment regimen, remission rates remain limited. Identifying new therapeutic options is a major challenge when these treatments fail. Considering the pathogenesis of IMN, telitacicept offers comprehensive inhibition and regulation of lymphocytes, including CD20-positive B cells, plasma cells, and T cells, significantly reducing the risk of forming circulating and in-situ immune complexes, thus achieving therapeutic effects. To date, only a few IMN cases have been treated with telitacicept; hence, its efficacy in Asian populations has yet to be confirmed. In this case report, we present three adult patients with refractory IMN treated with telitacicept. The application of telitacicept in the treatment of membranous nephropathy warrants further attention.

2 | CASE REPORTS

2.1 | Case 1

A 40-year-old man was admitted to the respiratory department with a diagnosis of pulmonary embolism. His blood pressure was 120/68 mmHg, and he weighed 61 kg. Physical examination revealed no edema in the extremities. Laboratory tests showed a serum creatinine (SC) level of 71 μmol/L, total serum protein of 39.6 g/L, and serum albumin (SA) of 19.8 g/L. Urinalysis indicated a 24-h urinary protein quantification (24 h-P) of 14.768 g/day, with a urine protein-to-creatinine ratio of 9.34 g/g creatinine. No anti-PLA2R antibodies were detected. The patient was diagnosed with nephrotic syndrome (NS). His family history did not indicate any hereditary diseases.

2.1.1 | Diagnostic workup

After stabilizing his pulmonary embolism, the patient was transferred to our department for further evaluation. A renal biopsy was performed for an accurate diagnosis (Figure 1a). Light microscopy of 38 glomeruli showed no global or segmental sclerosis. Mild mesangial cell and matrix proliferation were observed, with open capillary loops and thickened, rigid basement membranes, displaying numerous spike-like structures. There was no significant deposition of Congo red-positive material in the mesangial or subendothelial regions, and subepithelial deposits were observed. There was no mesangial interposition or double contour formation, and no crescents were seen. Tubular epithelial cells exhibited granular and vacuolar degeneration without significant atrophy. The interstitium showed scattered inflammatory cell infiltration without noticeable fibrosis. Small arterial walls were unremarkable. Immunofluorescence staining revealed granular deposits of IgG (3+), C3 (+/−), and IgM (1+) along the capillary loops, with no significant staining for IgA, C1q, Fib, or Alb. Tubular reabsorption droplets were positive for Alb, and C3 was positive in small arterial walls. Additional immunofluorescence showed PLA2R (3+) and THSD7A (−). Electron microscopy revealed diffuse podocyte foot process effacement and numerous subepithelial and intramembranous electron-dense deposits. Renal pathology confirmed stage III membranous nephropathy. Serological tests for HBsAg, HCV antibodies, ANA, ANCA, anti-GBM antibodies, and rheumatoid factor were negative. Serum immunoelectrophoresis did not detect monoclonal proteins, and there was no evidence of hypocomplementemia. We detected anti-tumor-associated antigen (TAA) antibodies in serum to excluded malignancies.

2.1.2 | Initial treatment

The patient was initially treated with an angiotensin II receptor blocker (ARB), anticoagulants, prednisone (PSL; 60 mg/day), and cyclosporine (CyA; 150 mg/day). After 8 weeks, the patient remained in NS status (CyA valley concentration: 134 ng/mL). CyA was discontinued, and dapagliflozin was added to the treatment regimen. Subsequently, a single intravenous dose of rituximab (375 mg/m^{2}for 4 weeks) was administered, with premedication consisting of loratadine (10 mg) and acetaminophen (0.5 g). The infusion was well-tolerated, and the PSL dose was gradually tapered off. However, after 4 months, there was no significant clinical or laboratory improvement.

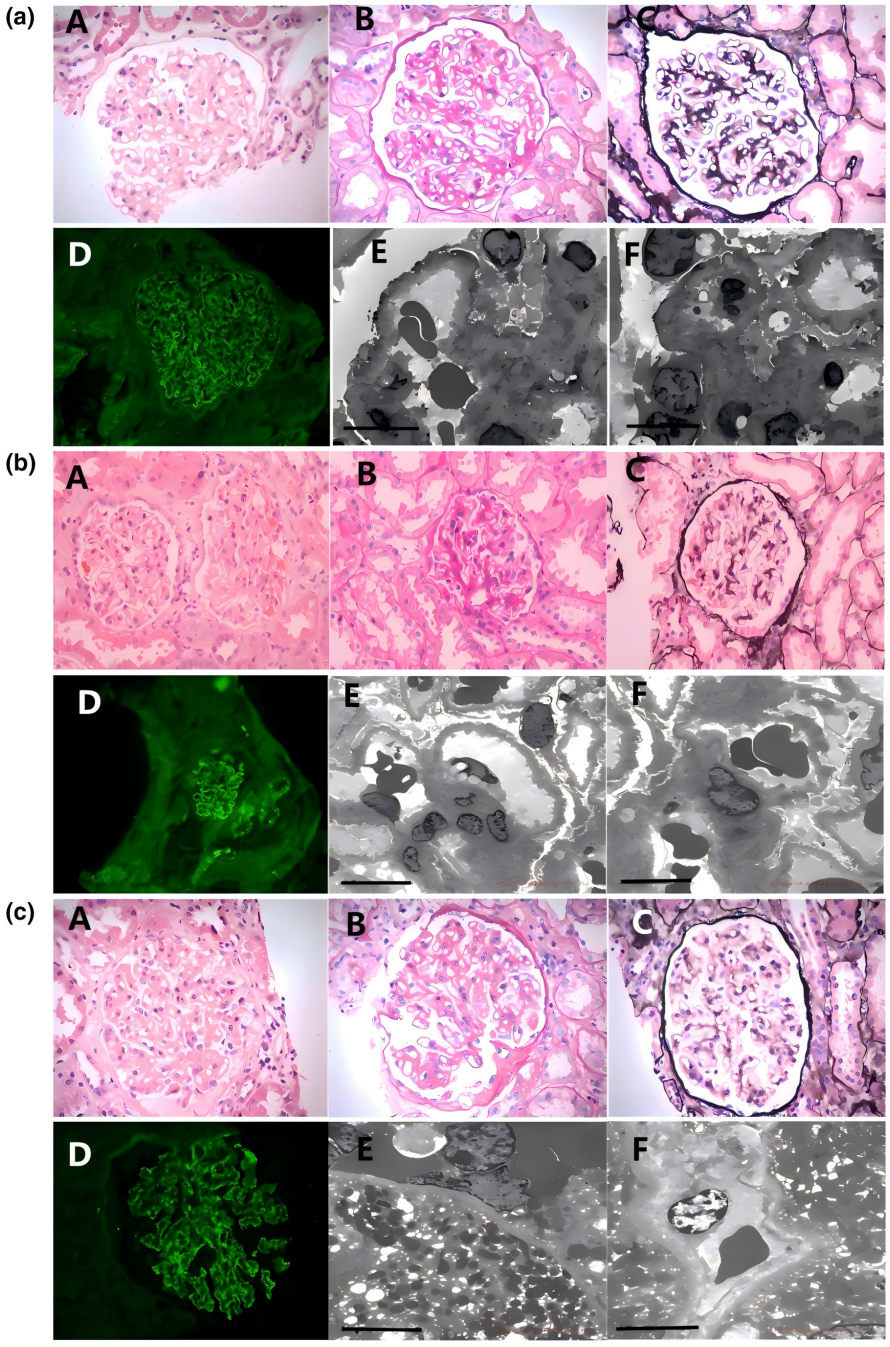

FIGURE 1 (a) Biopsy findings of case1. (A) Hematoxylin–eosin staining (×400); (B) periodic-acid silver methenamine staining (×400); (C) periodic acid-silver metheramine (PASM, ×400); (D) immunofluorescence staining for IgG (×400); (E, F) electron microscopy (EM, 2 kx), bar = 5 μm. (b) Biopsy findings of case 2. (A) Hematoxylin–eosin staining (×400); (B) periodic-acid silver methenamine staining (×400); (C) periodic acid-silver metheramine(PASM, ×400); (D) immunofluorescence staining for IgG (×400); (E) electron microscopy (EM, 2 kx), bar = 10 μm; (F) electron microscopy (EM, 3 kx), bar = 5 μm. (c) Biopsy findings of case 3. (A) Hematoxylin–eosin staining (×400); (B) periodic-acid silver methenamine staining (×400); (C) periodic acid-silver metheramine (PASM, ×400); (D) immunofluorescence staining for IgG (×400); (E, F) electron microscopy (EM, 4 kx), bar = 5 μm.

2.1.3 | Additional treatment

Despite various immunosuppressive treatments over 6 months, the patient remained in a persistent NS state. Additional rituximab treatment was recommended, but the patient lost confidence in rituximab. Considering treatment resistance, rituximab treatment efficacy is typically assessed 3 months post-administration. In case of failure, CYC or alternative protocols based on individual center experiences are suggested. Due to the patient's fertility concerns, he refused the CYC regimen.

Starting from April 6, 2023, the patient received 240 mg telitacicept weekly (qw) for 12 weeks, along with oral prednisone 20 mg daily (qd). By May 2023, the 24 h-P reduced to 6.640 g/day. In June 2023, 24 h-P further decreased to 2.790 g/day, SC to 60.2 μmol/L, and SA to 27.7 g/L. By July 2023, 24 h-P was 0.56 g/day, SC was 65 μmol/L, and SA was 35.2 g/L. From July 28, 2023, telitacicept dose was adjusted to 160 mg qw for 8 weeks. By August 2023, 24 h-P was 0.26 g/day, SC was 73 μmol/L, and SA was 39.6 g/L. After 8 weeks of treatment, telitacicept dose was reduced to 80 mg qw. Proteinuria remained in complete remission (CR), and prednisone

was tapered to a maintenance dose of 5 mg daily by September 12, 2023.

2.1.4 | Conclusion

This case highlights the potential effectiveness of Telitacicept in treating refractory primary stage III membranous nephropathy (PLA2R antibody mediated). Despite the lack of response to conventional therapies, the patient achieved significant remission with telitacicept, suggesting it may be a valuable alternative for refractory IMN treatment. Further research and clinical trials are necessary to confirm these findings and establish optimal treatment protocols.

2.2 | Case 2

A 63-year-old woman with a history of hypertension and hyperuricemia was admitted due to severe lower extremity edema and a weight gain of 7 kg. Urinalysis revealed significant proteinuria and hematuria, with a urinary protein-to-creatinine ratio of 7.40 g/g creatinine and a 24 h-P of 7.132 g/day. Her SC level was 89 μmol/L, and SA was 21 g/L. The patient was diagnosed with NS. There was no significant family history of hereditary diseases. The informed consent was obtained from the patient for publication of this case report and any accompanying images.

2.2.1 | Diagnostic workup

A week after admission, a renal biopsy was performed for histological diagnosis (Figure 1b). Light microscopy of 28 glomeruli showed global sclerosis in 4 glomeruli and no segmental sclerosis in the others. The remaining glomeruli exhibited mild mesangial cell and matrix proliferation, thickened basement membranes, and occasional spike-like structures without mesangial interposition or double contour formation. Immunofluorescence staining revealed granular deposits of IgG (3+), C3 (2+), and IgM (1+) along the capillary loops, with negative results for IgA, C1q, Fib, and Alb. Tubular reabsorption droplets were positive for Alb, and small arterial walls were positive for C3. Further immunofluorescence showed IgG1 (1+), IgG2 (1+), IgG3 (−), IgG4 (3+), PLA2R (−), and THSD7A (−).

Electron microscopy of the biopsy specimen showed irregular thickening of the basement membrane, diffuse podocyte foot process effacement, numerous subepithelial and intramembranous electron-dense deposits, and basement membrane proliferation around some deposits. Mild mesangial cell and matrix proliferation was also noted. The renal pathology confirmed membranous nephropathy (MN). Serological tests for HBsAg, HCV antibodies, anti-HAV antibodies, anti-GBM antibodies, and rheumatoid factor were negative. The patient's antinuclear antibody (ANA) was positive, but no autoimmune disease was identified. Serum and urine immunoelectrophoresis did not detect monoclonal bands, and serum complement levels were within the normal range. Malignancy was ruled out as a cause of secondary MN.

2.2.2 | Initial treatment

The patient was initially treated with prednisone (PSL) at 25 mg/day and tacrolimus (TAC; 1 mg twice daily, increased to 2.5 mg twice daily based on blood levels; TAC valley concentration: 6 ng/mL), along with optimization of baseline treatments. After 8 weeks, her SC increased to 118 μmol/L, SA remained at 20 g/L, and there was no significant improvement in lower extremity edema. Proteinuria remained high (6.40 g/g creatinine). The PSL dose was increased to 40 mg/day, and mizoribine (MZR) was added at 100 mg/day, increased to 200 mg/day based on blood levels. However, these treatments were ineffective. Given the negative serum PLA2R and THSD7A results, we recommended rituximab or CYC. Due to financial concerns and convenience, the patient opted for CYC.

The adjusted immunosuppressive regimen included prednisone (30 mg daily) and CYC (0.4 g IV twice monthly). By June 2023, after 10 IV doses of CYC (total dose 4.1 g), her 24 h-P was 4.13 g/day, SA was 22.1 g/L, and SC was 85.6 μmol/L. Serum PLA2R and THSD7A remained negative. We suggested rituximab or rituximab combined with tacrolimus, but the patient lacked confidence in tacrolimus and was concerned about the high costs and hospitalization associated with rituximab. We continued oral prednisone (20 mg daily), stopped CYC, and started telitacicept at 240 mg weekly.

2.2.3 | Follow-up and response

After 12 weeks of telitacicept treatment, the patient's 24 h-P decreased to 2.652 g/day, indicating partial remission (ICR). We then adjusted the dose to 160 mg weekly. By week 20 of treatment, her 24 h-P further decreased to 1.15 g/day, SC was 71 μmol/L, and SA significantly increased to 41 g/L. Telitacicept was then reduced to 80 mg weekly. To date, the patient's serum albumin has remained above 39 g/L, and 24 h-P has

remained between 1.134–2.769 g/day. No signs of NS have been observed, and her SC has remained stable (65–79 μmol/L). The PSL dose has been gradually tapered to a maintenance dose of 5 mg daily without the use of additional immunosuppressants.

2.2.4 | Conclusion

This case demonstrates the potential effectiveness of telitacicept in treating refractory IMN. Despite initial resistance to conventional therapies, the patient achieved significant remission with telitacicept, suggesting it may be a valuable treatment option for refractory IMN. Further research and clinical trials are necessary to confirm these findings and establish optimal treatment protocols.

2.3 | Case 3

A 79-year-old male was diagnosed with NS after being found to have a urinary protein excretion of 7.665 g/g creatinine while receiving treatment for a bleeding gastric ulcer at another institution. His SC level was 175 μmol/L, with total serum protein and albumin levels of 42.7 and 23 g/L, respectively. Upon admission, his blood pressure was 158/96 mmHg, and his weight was 69.5 kg. He had a 3-year history of hypertension but denied any family history of hereditary diseases. The written informed consent was obtained from the patient for publication of this case report and any accompanying images.

2.3.1 | Diagnostic workup

A renal biopsy (Figure 1c) performed in our department revealed 14 glomeruli, of which 5 showed global sclerosis and 2 exhibited segmental sclerosis. Light microscopy indicated the presence of spike-like structures without mesangial interposition or double contour formation, and no crescents were observed. Immunofluorescence staining demonstrated granular deposits of IgG (3+), IgM (1+), C3 (1+), and IgA (1+) along the capillary loops, with positive staining in the small arterial walls for C3. Additional staining revealed IgG1 (+), IgG2 (+/−), IgG3 (−), IgG4 (3+), PLA2R (−), and THSD7A (−) (Figure 1c). The histological diagnosis was membranous nephropathy. Tests for hepatitis B and C, ANAs, and anti-glomerular basement membrane (GBM) antibodies were negative. Serum and urine immunofixation electrophoresis did not detect monoclonal proteins, and serum complement levels were within normal limits. Malignancy was ruled out as a secondary cause of IMN.

2.3.2 | Treatment and follow-up

Given the patient's history of gastric ulcer bleeding, corticosteroid use was limited. The patient was advised to use rituximab or a combination of rituximab and tacrolimus. Due to concerns about his age and compromised immunity, the patient and his family opted for rituximab monotherapy.

In January 2023, the patient received an intravenous infusion of rituximab 1.0 g on Days 1 and 15. In February 2023, follow-up tests showed a 24 h-P of 8.41 g/day, SA of 25.4 g/L, SC of 163 μmol/L, and a CD20 count of 2 cells/μL. Blood tests for PLA2R and THSD7A remained negative. The patient received a second infusion of rituximab 1.0 g in February 2023. By April 2023, his 24 h-P was 7.65 g/day, SA was 26.4 g/L, SC was 143.3 μmol/L, and CD20 count was 0 cells/μL. Despite this treatment, the patient remained at moderate to high risk, and further rituximab treatment was recommended.

The patient lost confidence in rituximab, and with his consent, we initiated telitacicept at a dose of 240 mg weekly for 12 weeks, starting on May 19, 2023. Follow-up urinalysis in July 2023 showed a urinary protein excretion of 7.44 g/g creatinine. By October 2023, his 24 h-P had decreased to 5.41 g/day, SA had increased to 28 g/L, and SC had decreased to 140 μmol/L. Despite treatment with telitacicept, significant proteinuria and hypoalbuminemia persisted. No adverse events were observed during the course of telitacicept treatment.

2.3.3 | Conclusion

This case highlights the challenges in managing refractory IMN in elderly patients with comorbid conditions. Despite initial treatments with rituximab and subsequent telitacicept, the patient continued to exhibit significant proteinuria and hypoalbuminemia. Telitacicept may offer some benefit, but further research is needed to establish its efficacy and safety in this patient population.

3 | DISCUSSION

The 2021 KDIGO guidelines suggest that for patients with refractory membranous nephropathy (IMN) unresponsive to rituximab or CYC, expert consultation should be sought, and treatments such as bortezomib, anti-CD38 therapy, and belimumab should be considered.[4] In this report, we discuss three cases of refractory IMN treated with telitacicept. Baseline characteristics of these patients are summarized in Table 1. All patients provided informed consent for the publication of their cases.

TABLE 1 Main clinical and laboratory characteristics of patients at baseline.

Characteristics	Case 1	Case 2	Case 3
Age (years)	40	63	79
Gender (male/female)	Male	Female	Male
Clinical parameters			
Body weight (kg)	61	64	69.5
Systolic blood pressure (mmHg)	120	137	158
Diastolic blood pressure (mmHg)	68	77	96
Laboratory parameters			
Total protein (g/L) (normal ranges: 60–83)	39.6	41	42.7
Serum albumin (g/L) (normal ranges: 35–55)	19.8	21	23
Serum creatinine (μmol/L) (normal ranges: 44–133)	71	89	175
eGFR(CKD-EPI) (mL/min/1.73 m^2)	111.57	59.63	31.22
24 h proteinuria (g/day) (normal ranges: 0.028–0.141)	14.768	7.132	9.652
Urinary protein excretion (g/g Cre) (normal ranges: 0.10–0.028)	9.34	7.4	7.66
Anti-PLA2R antibody	Negative	Negative	Positive
Treatment before telitacicept			
PSL (mg/day)	60	25	Unused
CyA (mg/day)	150	Unused	Unused
TAC (mg/day)	Unused	5	Unused
MZR (mg/day)	Unused	200	Unused
IVCY	Unused	4.1	Unused
Rituximab	375 mg/m^2	Unused	1 g
SGLT2 inhibitor	10 mg	10 mg	Unused
RAS inhibitor agent	Losartan 25 mg	Valsartan 160 mg	Losartan 50 mg

Abbreviations: Cre, creatinine; CyA, cyclosporine; eGFR, estimated glomerular filtration rate; IVCY, intravenous cyclophosphamide; MZR, mizoribine; PLA2R, phospholipase A2 receptor; PSL, prednisolone; RAS, renin-angiotensin system; SGLT2, Sodium-d dependent glucose transporters 2; TAC, Tacrolimus.

3.1 | Challenges and treatment considerations

One challenge with these cases was the negative status for serum PLA2R and THSD7A antibodies, which precluded using antibody titers as a basis for therapy. Therefore, treatment adjustments were guided by 24-h urinary protein excretion and serum albumin levels. After conventional therapies, including angiotensin receptor blockers (ARBs), sodium-glucose cotransporter-2 (SGLT2) inhibitors, corticosteroids, tacrolimus, mycophenolate mofetil (MMF), CYC, cyclosporine, and rituximab, proved ineffective, we considered continuing rituximab or switching to another B-cell-targeted therapy. A review of IMN pathogenesis and the mechanisms of current therapies informed our decision-making process.

IMN's immune mechanism involves the formation and deposition of immune complexes containing immunoglobulins and complement. Activated B cells and plasma cells are the primary sources of antibody secretion. When stimulated by antigens, plasma cells can produce large quantities of antibodies, leading to podocyte damage and alterations in the glomerular basement membrane, resulting in proteinuria.[8] Rituximab has shown potential as a treatment for IMN, with evidence suggesting it maintains proteinuria remission for up to 24 months better than cyclosporine.[9] Other therapies like obinutuzumab, ofatumumab, and belimumab also target CD20-positive B cells but not plasma cells.[10] Proteasome inhibitors (bortezomib) and anti-CD38 therapies (daratumumab and felzartamab) target plasma cells but not CD20-positive B cells.[11] CYC and calcineurin inhibitors (tacrolimus and cyclosporine) inhibit T cells and their cytokines but lack effective B-cell suppression.[12]

3.2 | Telitacicept mechanism and application

Telitacicept is a recombinant fusion protein combining the extracellular domain of the transmembrane activator and calcium modulator and cyclophilin ligand interactor (TACI) with the human IgG1 Fc fragment. TACI receptor

TABLE 2 Comparison of clinical data of patients with MN before and after treatment.

Clinical indicators	Case 1		Case 2		Case 3	
	Pre-treatment	Post-telitacicept	Pre-treatment	Post-telitacicept	Pre-treatment	Post-telitacicept
Total protein (g/L)	39.6	68	41	67	42.7	53.9
Serum albumin (g/L)	19.8	39.6	21	41	23	28
Serum creatinine (μmol/L)	71	73	89	79	175	140
eGFR (CKD-EPI)	111.57	116.32	59.63	68.75	31.22	39.46
24 h proteinuria (g/day)	14.768	0.26	7.132	2.652	9.652	5.41
Urinary protein/creatinine ratio	9.34	0.12	7.4	1.19	7.66	4.63

has a high affinity for B lymphocyte stimulator (BLyS) and a proliferation-inducing ligand (APRIL). Telitacicept interferes with abnormal B cell and plasma cell activation by antagonizing the interaction between BLyS or APRIL and their receptors on B lymphocytes.[13] By blocking BLyS, telitacicept inhibits the maturation of immature B cells, potentially controlling disease progression. Blocking APRIL inhibits the differentiation of mature B cells into plasma cells, affecting the secretion of autoantibodies by autoreactive plasma cells, thus better controlling disease activity.[14,15] Telitacicept can also inhibit T cell activation due to TACI receptors on T cells.

Currently, telitacicept is mainly used for rheumatoid arthritis, systemic lupus erythematosus (SLE), multiple sclerosis, and IgA nephropathy. In pediatric patients aged 5–18 with active SLE, telitacicept combined with standard therapy significantly improved treatment efficacy, reduced corticosteroid dosage, and showed efficacy in lupus nephritis.[16] Clinical studies in adult primary Sjögren's syndrome (pSS) have demonstrated good clinical efficacy, tolerability, and safety of telitacicept.[17] In primary glomerular disease treatment, current reports mainly focus on phase II clinical studies of telitacicept in IgA nephropathy, showing that it can effectively reduce urinary protein levels.[18] Zhang et al. first reported the application of telitacicept in IMN treatment in 2023.[19] Given the pathogenesis of autoimmune kidney diseases and telitacicept's mechanism of action, there is reason to believe telitacicept has broad prospects in treating autoimmune kidney diseases.

3.3 | Case summary and clinical outcomes

In this case report, three patients with refractory IMN, unresponsive to corticosteroids and immunosuppressants (such as CyA, CYC, MZR, MMF, and rituximab), received telitacicept as second-line therapy. Each patient's clinical course is detailed. All three cases were prescribed maximal tolerated ACE inhibitor or ARB therapies. We chose telitacicept 240 mg based on the results of previous efficacy and safety data.[18] CR was defined as proteinuria <0.3 g/day. Partial remission (ICR) was defined as proteinuria between 0.3 and 3.5 g/day. Nonresponse (NR) was defined as persistent nephrotic-range proteinuria (≥3.5 g/day). Treatment efficacy was assessed using 24 h-P and SA levels. If 24-h urine collection was not possible, the spot urine protein-to-creatinine ratio (g/g creatinine) was used.

In cases 1 and 2, telitacicept as a second-line therapy was effective (Table 2). The first patient achieved and maintained CR within 4 months of telitacicept treatment. The second patient, although achieving only ICR, maintained a serum albumin level above 39 g/L without NS symptoms, and his kidney function improved (SC level decreased from 89 to 79 μmol/L). No edema was observed posttreatment. For these reasons, telitacicept was considered effective in the second case.

Cravedi et al.[20] conducted a prospective matched cohort study comparing single-dose rituximab (1 g) with the standard four-week regimen (375 mg/m^2) for treating nephrotic IMN. Results indicated that a single rituximab dose was as effective as the standard regimen in inducing IMN remission. Therefore, we used a single-dose rituximab for IMN treatment. Case 3 did not respond to single rituximab infusions. In cases 2 and 3, renal tissue PLA2R and THSD7A antibodies were negative. Prunotto et al.[21] detected specific anti-aldose reductase and anti-manganese superoxide dismutase (SOD2) IgG4 in the serum of IMN patients, indicating that other autoantibodies might be present in addition to anti-PLA2R antibodies. Conversely, case 3 did not show significant improvement with telitacicept treatment, as his proteinuria and kidney function remained unchanged.

3.4 | Potential explanations and limitations

The variability in telitacicept treatment outcomes in our cases might have several explanations: case 1 showed the

best response to telitacicept. In cases 2 and 3, elevated SC levels at rituximab treatment initiation suggested possible kidney fibrosis, potentially affecting drug targeting. Both patients had a history of hypertension and bilateral kidney atrophy. In this study, no telitacicept-related adverse events, such as infections or bone marrow toxicity, were observed in IMN treatment.

However, this case report has limitations. Given the insufficient current evidence for telitacicept in IMN treatment, our therapeutic approach requires further research and observation. It remains uncertain whether patients will experience relapses or long-term adverse effects in the future, necessitating larger sample sizes to determine telitacicept's therapeutic efficacy and further comparative studies to analyze the efficacy of telitacicept combined with other immunosuppressants and telitacicept monotherapy in the treatment of refractory IMN.

4 | CONCLUSION

Telitacicept shows promise as a second-line therapy for refractory IMN, particularly in cases where conventional treatments fail. Further studies are warranted to confirm its efficacy and safety in this context.

AUTHOR CONTRIBUTIONS

Liping Sun: Conceptualization; project administration; writing – review and editing. **Fuce Chen:** Writing – original draft. **Xinzhou Zhang:** Investigation; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The corresponding author's data supporting this study's findings are available upon reasonable request.

ETHICS STATEMENT

This study protocol was reviewed and approved by the Ethics Committee of Shenzhen People's Hospital, approval number LL-KY-2023023-01. Written participate statements were obtained from the patients.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

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