Biologics: Targets and Therapy

Dovepress
Taylor & Francis Group

Open Access Full Text Article

CASE REPORT

# The Potential of Telitacicept in Treating Lupus erythematosus panniculitis: A Case Report

Xinying Xie[1,*], Shuibo Tang[1,*], Ma Huang[2]

[1]Rheumatology and Immunology Department, Hunan University of Medicine General Hospital, Huaihua, Hunan, People's Republic of China; [2]Department of Intensive Care Medicine, Hunan University of Medicine General Hospital, Huaihua, Hunan, People's Republic of China

*These authors contributed equally to this work

Correspondence: Ma Huang, Email huangma19910219@163.com



**Abstract:** Lupus erythematosus panniculitis (LEP) is a rare and serious skin lesion associated with systemic lupus erythematosus (SLE). In the past, the management of LEP has followed the treatment guidelines for SLE, which is based on the administration of high doses of hormones together with immunosuppressants, with no treatment plans specific for LEP. However, the long-term use of high hormone doses and immunosuppressants can exacerbate skin breakage and calcification. Telitacicep is an approved biological agent demonstrating significant therapeutic efficacy in immune system disorders. We report a case of lupus erythematosus panniculitis in which the patient's clinical symptoms and laboratory test results improved after treatment with a combination of steroids, cyclophosphamide, and the biologic agent Telitacicept. The condition was significantly controlled and alleviated. This report highlights the successful application of Telitacicept in the treatment of lupus panniculitis, providing valuable insights for future management of this condition.

**Keywords:** Telitacicept, biologicals, Lupus erythematosus panniculitis

## Introduction

Systemic lupus erythematosus (SLE) can cause skin damage. This damage falls into two categories, namely, disease-specific and disease-non-specific skin lesions. Apart from disease associations, skin damage can also be secondary to medication use. There are nine major non-specific skin lesions associated with SLE, including reticular cyanosis, purpura, photosensitivity, alopecia, urticarial-like skin lesions (wheals), mucosal ulcers, skin sclerosis, melanin deposition, and hormone lines {Abdelrahman, 2023 #193}[1] Lupus erythematosus panniculitis (LEP), also known as deep erythema lupus, is a specific form of skin lupus in which the skin lesions often present as atrophic erythema and subcutaneous nodules, accompanied by highly specific histopathological manifestations. The condition is rarely seen clinically and has an incidence of only 2%. It is more common in middle-aged women and is associated with a markedly high rate of misdiagnosis. The treatment is difficult and the disability rate is extremely high. To date, there have been few reported cases in the domestic and international literature.[2] Lupus erythematosus panniculitis is associated with immune responses, wherein antigen-antibody complexes may induce local immune reactions, leading to severe manifestations in the skin and subcutaneous fat. External factors may trigger LEP, activating innate and adaptive immunity. Genes encoding pro-inflammatory cytokines participate in apoptosis, leukocyte migration, the type I interferon pathway, the complement cascade, antigen presentation, and antibody production.[3] These external factors can alter the morphology and function of keratinocytes, inducing the release of pro-inflammatory cytokines and apoptosis. Increased inflammatory cytokines and cell death release cellular debris that triggers the aggregation of lymphocytes and plasmacytoid dendritic cells (pDCs), thereby activating the immune system. However, the capture of this debris by pDCs constitutes an autoantigen reservoir for reactive B and T lymphocytes, subsequently generating a cascade of immune-mediated damage.[4] Medications may also exacerbate cutaneous lesions in systemic lupus erythematosus. Examples include penicillin, sulphonamides, phenylbutazone, procainamide, and chlorpromazine. Upon entering the body, these drugs can trigger allergic reactions, thereby worsening skin damage.[5,6] LEP is a seasonal skin disorder representing a relatively

Received: 9 July 2025
Accepted: 25 September 2025
Published: 7 October 2025

uncommon clinical manifestation of systemic lupus erythematosus. Lesions typically manifest on the face, neck, shoulders, chest, back, and buttocks, presenting as asymmetrical nodular skin lesions located subcutaneously, in the mid-dermis, or deeper dermal layers. Typical manifestations include firm, mobile subcutaneous nodules or plaques, often with central depression, presenting as erythematous or hyperpigmented lesions. Overlying skin may exhibit atrophy, hyperkeratosis, telangiectasia, and characteristic discoid erythema lesions.[7–9] Current treatment approaches for LEP have long been guided by systemic lupus erythematosus management protocols,[10,11] focusing on controlling inflammation, alleviating symptoms, and preventing recurrence. Primary methods include photoprotection, topical care, dietary adjustments, antimalarials, and oral corticosteroids.Severe cases may require combination therapy with immunosuppressants for intensive treatment. Overall efficacy remains poor, and specialised treatment protocols are lacking for certain refractory cases.[12] Therefore, it is urgent to identify more effective single or combined treatment options.

In recent years, extensive research on the pathogenesis and genetic basis of SLE has led to the development of biologics for treating the disease. These have the advantages of being more precise and less toxic, and have thus shown positive effects.[13] Telitacicept is a novel recombinant fusion protein, and is the first biologic to target a proliferation-inducing ligand (APRIL) +B-lymphocyte stimulator (BLyS). It is derived from a fusion of a gene fragment encoding the TAC1 protein, a typical ligand of the BlyS and value-added-inducing ligands, with a crystallizable Fc fragment of human IgG, and is capable of simultaneously blocking the binding of BLys to transmembrane activator and CAML interactor (TACI), inhibiting B lymphocyte differentiation and survival.[14–16] Unlike belimumab, Telitacicept can interact with both BLyS and APRIL, a member of the also known as tumor necrosis factor superfamily. Both BlyS and APRIL are key regulators of lymphocyte development and maturation, and due to this dual targeting, Telitacicept can inhibit immune activity and reduce the systemic immune-inflammatory response, thus achieving an effective immunotherapeutic effect.[17] In clinical practice, Telitacicept has shown reliable efficacy and safety in the treatment of SLE and its complications. The 2020 and 2023 EULAR guidelines for the diagnosis and treatment of SLE recommend the use of biologics for the treatment of refractory SLE, and the 2023 guidelines for treating refractory cutaneous lupus lesions also recommend the combination of biologics, including belimumab, rituximab, and Telitacicept.[16,18] However, despite these recommendations for the use of biologics for treating lupus skin lesions, there have been few clinical applications and reports on their use for LEP. Among these drugs, Telitacicept was independently developed and is currently manufactured in China, and was conditionally approved for marketing in China in 2021 based on the findings of its Phase II clinical trial.[16] In November 2023, Telitacicept received full approval from the State Drug Administration of China (SDA).[19] Based on the results of its Phase II randomized controlled trial, the indications for Telitacicept use are the same as those of belimumab for adults with SLE, ie, adult patients with SLE who still have high disease activity and are autoantibody-positive on the basis of SoC. In the phase IIb trial, the SLE Response Index-4 (SRI-4) response rate at week 48 was higher in the Telitacicept (80, 160, 240 mg) group than in the placebo group, and the proportion of patients with improvements of ≥4 in the SELENASLEDAI score was significantly higher in the tetracilib-treated groups relative to the placebo group.[20] In the Phase III clinical trial, after 52 weeks of treatment, the SRI-4 response rate was higher in the Telitacicept group than in the placebo group, with significant improvements observed in patients with cutaneous mucosal, musculoskeletal, and hematological involvement at baseline.[21] Thus, Telitacicept has strong therapeutic effects on lupus erythematosus panniculitis.As far as we know, this case represents the first reported use of Telitacicept for the treatment of lupus erythematosus panniculitis, offering valuable insights for subsequent management of lupus panniculitis and the development of clinical guidelines.

## Case Description

The patient was a 20-year-old male. Past medical history: Physically fit. Denies history of infectious diseases such as tuberculosis, hepatitis, or malaria, or close contact with such cases. Denies history of hypertension, diabetes, heart disease, or cerebrovascular disease. No history of blood transfusions, blood donation, or surgery.Six years before presentation, he developed recurrent fever, joint pain, and skin nodules on the lower limbs accompanied by ulcers, with no obvious cause. He was diagnosed with SLE at another hospital and was treated with intermittent cyclophosphamide for six months. The prednisone acetate dose was reduced gradually to two tablets per day for maintenance treatment. In August 2020, the patient's original rash worsened, with the appearance of long strip-shaped (approximately 15–20 cm long and 3–4 cm wide) purplish-red lines on his back (Figure 1), accompanied by intermittent fever. After self-medication, the fever recovered and the symptoms improved.

**Figure 1** Abdominal skin damage.

In March 2021, the patient again developed fever and worsening of the rash, accompanied by low back and leg pain, numbness in both lower limbs, and an inability to walk, which could not be relieved by self-medication with cold and flu medicines. This led to his first admission to our department. Laboratory tests on admission showed urinary protein (+), ESR 58 mm/h, IgG 47.00 g/L, C3 0.18 g/L, C4 0.20 g/L, anti-U1RNP (+), anti-Sm antibody (+) anti-ribosomal P protein antibody (+), anti-Ro-52 (+), anti-SSA (+), anti-ds-DNA (-), and ANA1:1000 positivity (cytoplasmic type). Computed tomography (CT) indicated X bone mineral density in the right patella, right lower femur, and upper tibia, with multiple dense shadows in the soft tissue of the pelvic wall and marked calcification of the skin of the lower limbs. Skin biopsy showed infiltration of a few plasma cells, lymphocytes, and histiocytes around small blood vessels in the superficial-deep dermis (Figure 2). The primary diagnoses were: 1. Systemic lupus erythematosus (SLEDAI score 29) neuropsychiatric lupus, lupus-associated cutaneous vasculitis, and secondary hyperglobulinemia; and 2. Ischemic necrosis of the bilateral femoral heads. The patient was treated with the following regimen: methylprednisolone 500 mg/day for 3 consecutive days, which was then changed to prednisone 50 mg/day for maintenance; dexamethasone 5 mg+methotrexate 10 mg intrathecal injection, administered twice; Mycophenolate Mofetil tablets 0.75 g twice daily, and hydroxychloroquine sulfate 0.4 g/day.

On April 25, 2022, the patient was readmitted to our department due to fever and the development of a new ulcer on his right thigh. Physical examination revealed a large number of red patches on both hands, some of which were ulcerated and scabbed, while the skin on the outer side of the right thigh was damaged, with a lesion measuring approximately 2*2 cm with purulent secretion. There were scattered red patches, brown macules, subcutaneous nodules, and hard swellings (irregularly shaped with a soybean-like appearance) of varying sizes, with some of the hard nodules showing skin erythema and erosion. There were also symmetrical, oblique-shaped purplish-red streaks on both sides of the waist. Both knee joints were swollen, with high skin temperatures, and the knee joints were flexed and unable to straighten. The skin on the front of the right knee joint showed extensive red coloration and swelling, with yellowish crusts (Figure 3). The laboratory test results showed ESR 154 mm/h, anti-Sm (+), anti-U1RNP (+), anti-SS-A (+), anti-Ro52 antibody (+), anti-dsDNA antibody (±), anti-nuclear antibody 1:1000 positive (cytoplasmic type), C3 0.31 g/L, IgG 65.90 g/L, C-ANCA (+-), and 24-h urine protein quantification 1065.84 mg/24 h. CT examinations indicated multiple punctate and strip-shaped dense shadows in the soft tissues around the knee joints. The bone densities of the right patella, the lower segment of the right femur, and the upper segment of the right tibia were uneven, with patchy and slight high-density shadows. Multiple punctate and strip-shaped dense shadows were seen in the soft tissues of the

 https://doi.org/10.2147/BTT.S551578 

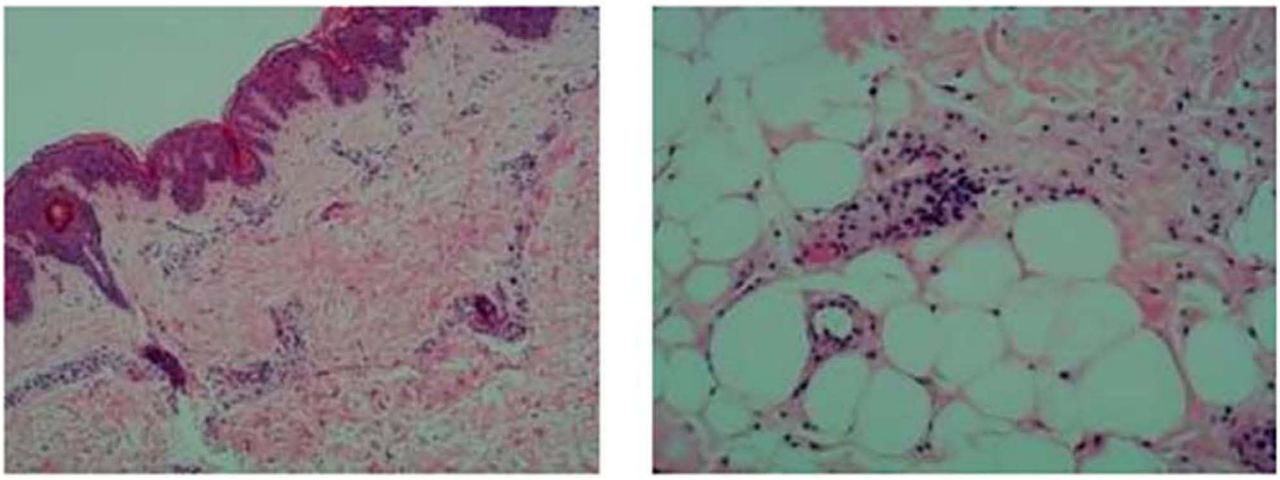

**Figure 2** Skin biopsy for pathological examination (2021.3.23).

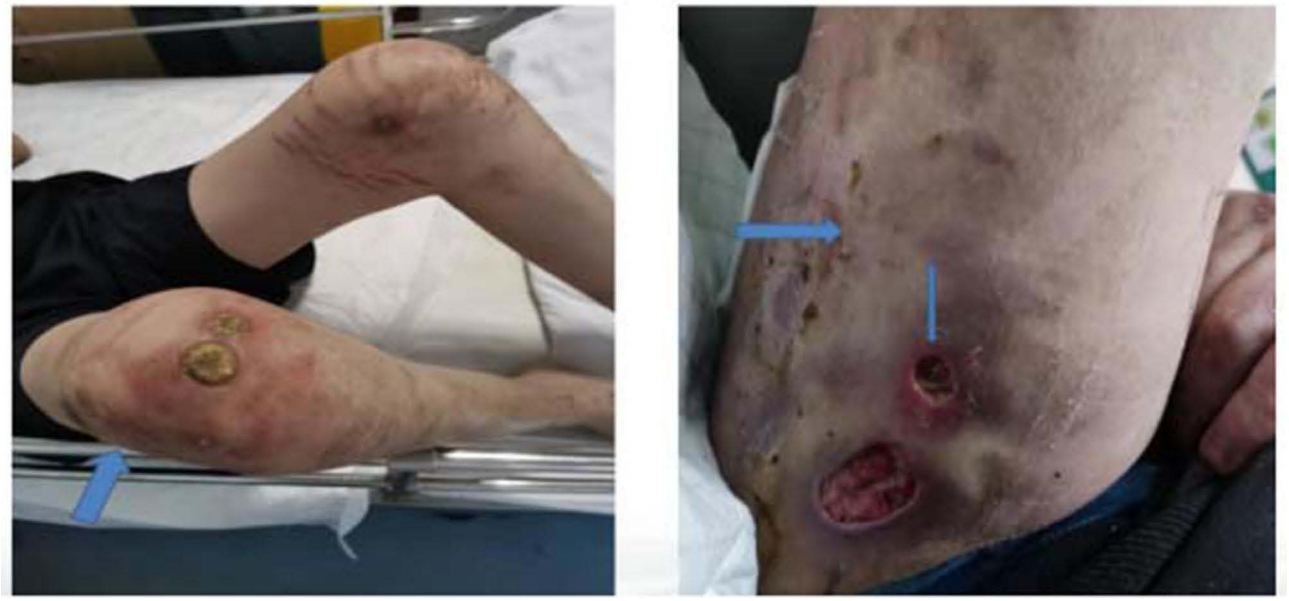

**Figure 3** Damage to the lower limb skin (Blue arrows indicate indurated necrotic ulceration).

pelvic wall. Biopsy of the right thigh indicated a small amount of degraded calcified tissue in the sample, while the skin of the right forearm, subcutaneous fat tissue, and epidermis were essentially normal (Figure 4). Greater infiltration of plasma cells, with sparse lymphocytes and neutrophil infiltration was visible around the dermal vessels and sweat glands, while subcutaneous fat lobules were necrotic with greater deposition of calcium salts. Considering the significant increase in skin nodules and the presence of ulcers, increased urine protein, elevated globulin and IgG levels, and reduced amounts of complement C3, the biopsy examination did not support a diagnosis of infection or tumor, and there was also a tendency toward panniculitis and calcification. The treatment plan was adjusted as follows: methylprednisolone 80 mg/day for 5 consecutive days, changed to prednisone tablets 50 mg/day for maintenance; cyclophosphamide injection 1.0 g * 1 dose; sulfamethoxazole (SMZ) for infection control, wound dressing, and other supportive treatments. This treatment led to significant reductions in the wound secretions and improvements in joint pain, while the rash became lighter with no fever and decreased levels of globulin (IgG 23.5 g/L) on re-examination on May 17, 2022, and the patient was discharged.

In June 2022, the patient again experienced fever, with worsening lower-limb ulceration, and he was admitted to our department for the third time. Upon admission, a CT scan revealed that, compared to the scan performed on May 5, 2022, there

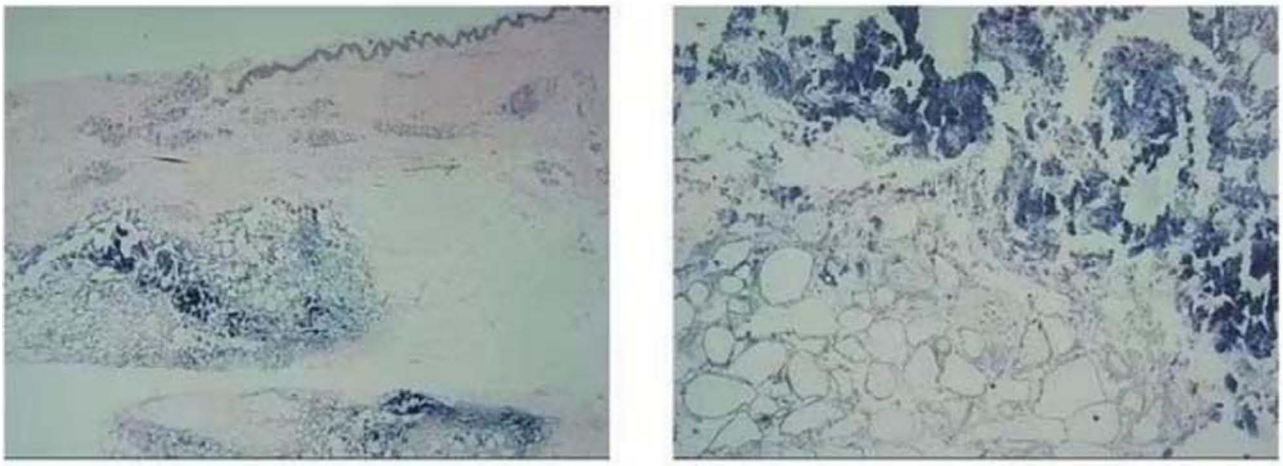

**Figure 4** Skin biopsy of the right lower limb (2022.04.28).

were patchy and faintly patchy shadows of slightly higher density in the right patella, lower segment of the right femur, and upper segment of the tibia, similar to the previous findings. We reconsidered the situation. Despite conventional and repeated use of corticosteroids and immunosuppressive agents, the patient's condition remained unresponsive, with a tendency to worsen. According to the 2019 ACR guidelines for the diagnosis and treatment of SLE, treatment with biologics (2B) may be considered for patients with SLE who respond poorly to, are intolerant to, or relapse after corticosteroid and/or immunosuppressive therapy. We actively discussed this with both the patient and his family, who provided written informed consent. The treatment plan was adjusted as follows: Telitacicept 160 mg once weekly; methylprednisolone 80 mg/day for 5 days, then switched to prednisone 50 mg/day (with a weekly reduction of 1 to 2 tablets per day for maintenance); cyclophosphamide injection 0.8 g once; immunadsorption (H280×4, DNA adsorption once).

The fourth laboratory examination in August 2022, showed a 24-h urine protein quantification of 709.87 mg, CRP level of 9.95 mg/L, ESR of 42 mm/h, complement C3 level of 0.78 g/L, and IgG of 27.91 g/L. The 24-h urine protein quantification, CRP, ESR, C3, and IgG levels were all improved compared to before. Due to the slow action of biologic agents, we continued to increase the dose of methylprednisolone to 40 mg/day (reducing one tablet every two weeks to a dose of 2 tablets/day), combined with a single 0.8 g dose of co-trimoxazole (CTX), and Telitacicept treatment. The patient's condition improved compared to the previous examination.

Surprisingly, despite the local COVID-19 outbreak, the patient not only did not contract COVID-19 but also showed significant improvement in his condition. On 27 January 2023, he was admitted for the fifth time for a follow-up examination, and it was noted that the rash had improved significantly. The follow-up examination showed IgG 20.22 g/L, IgM 0.58 g/L, CRP 11.22 mg/L, 24-h urine protein quantification: 1235.79 mg; an ENA profile of anti-Sm antibody positive (+), anti-U1RNP antibody positive (+), anti-SS-A (Ro) antibody positive (+), anti-Ro52 antibody positive (+), anti-nuclear antibody 1:320 positive (cytoplasmic type), with no significant abnormalities in complement C3 and C4 levels. Overall, both the clinical symptoms and laboratory test results showed marked improvements relative to the previous findings. The current treatment regimen involves prednisolone acetate tablets 8 mg once daily and telitacicept 80 mg once weekly by subcutaneous injection; a total of 6 cycles of CTX have been administered in this phase, with subsequent CTX maintenance therapy recommended every three months to sustain remission.

In June 2023, the patient was re-admitted to hospital for a sixth follow-up visit. The rash had improved significantly compared to the previous visit (Figure 5). The follow-up examination showed IgG 25.70 g/L, with normal levels of immunoglobulin A/M and complement C3/C4, and a 24-h urine protein quantification of 505.50 mg.

The seventh hospitalization for follow-up examination took place in July 2024, with no discomfort reported. The laboratory results showed IgM 0.29↓g/L, while IgA, IgG, C3, and C4 were all negative and the 24-h urine protein quantification was 394.25 mg/24 h. According to the SLEDAI 2000 scoring table, the score was 0 points, indicating that the patient's lupus was inactive.

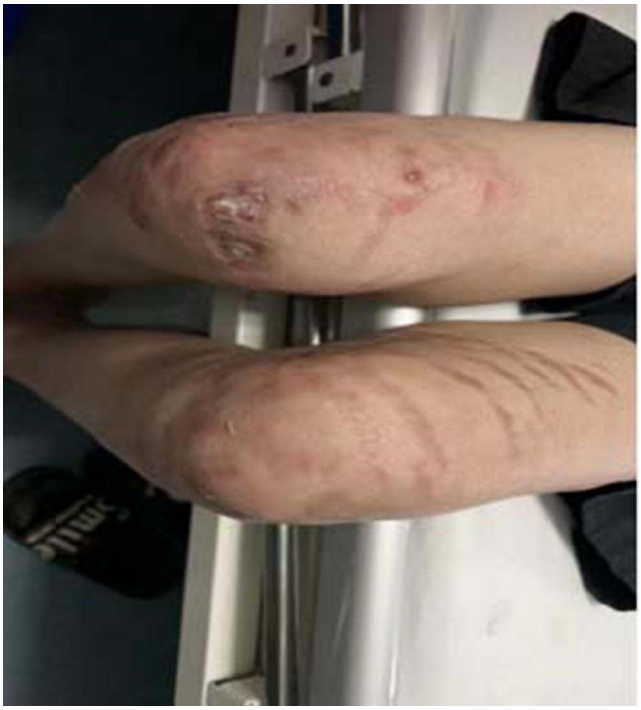

**Figure 5** The condition of skin damage on the lower limbs after treatment with Telitacicept.

Since this follow-up examination, the patient's symptoms have significantly improved, and all laboratory tests are within normal ranges, indicating a stable condition (Figure 6A–C). The current treatment plan involves the biologic agent Telitacicept, still adhering to an injection of 80 mg every week, prednisone acetate 5 mg per day, hydroxychloroquine sulfate 0.2 g per day, and cyclophosphamide 0.8 g once every six months.

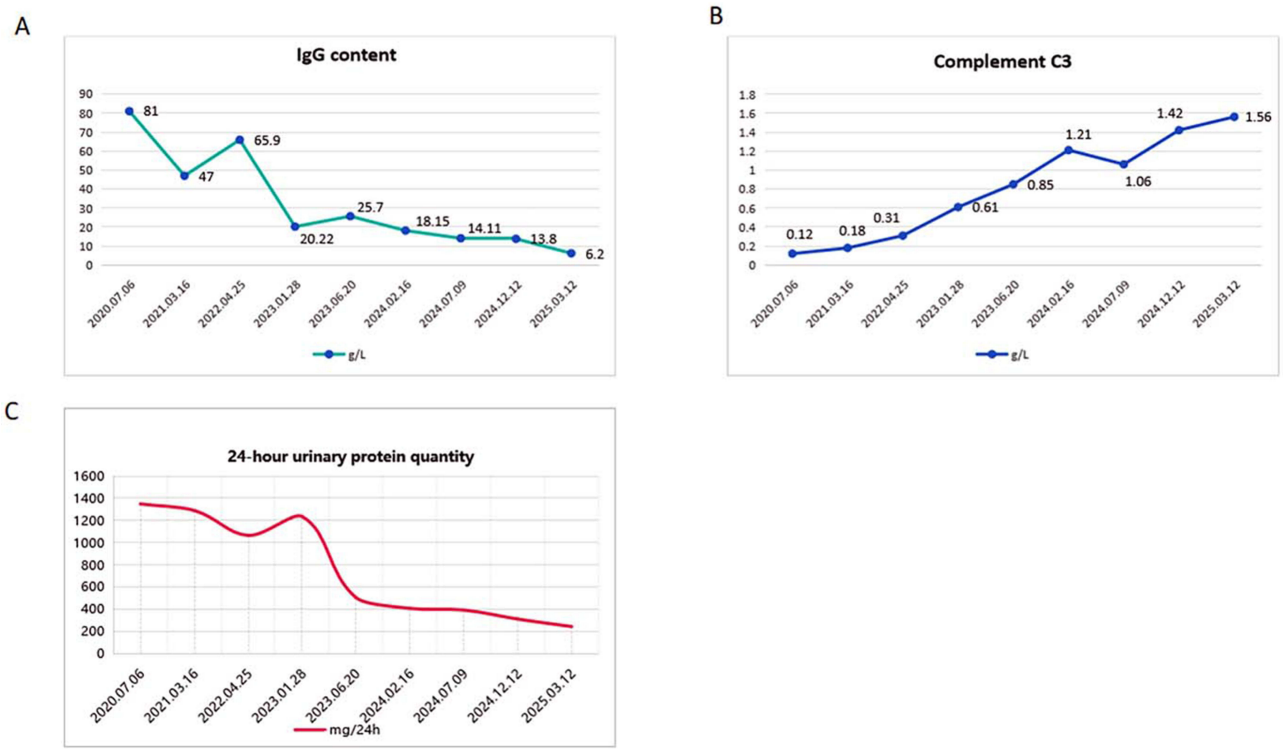


**Figure 6** Changes in various indicators following the use of Telitacicep. (**A**) The changes in IgG levels after treatment with Telitacicept. (**B**) The changes in 24-hour urine protein quantification after treatment with Telitacicept. (**C**) The changes in complement C3 after treatment with Telitacicept.

The summary of this case: The patient suffered from LEP combined with a high immune inflammatory response and elevated globulin levels. Telitacicept demonstrated a favorable therapeutic effect on this patient: 1. It assisted in the long-term remission of SLE; 2. It reduced the doses of hormones and immunosuppressants, avoiding the effects of these drugs on lupus itself; 3. Telitacicept has an excellent therapeutic effect on lupus panniculitis with calcification.

## Discussion

SLE can present concurrently or successively with damage to the bones, joints, muscles, kidneys, heart, lungs, central nervous system, blood, eyes, hearing, and skin. In terms of skin lesions, SLE often leads to lower limb ulcers, which are more common in areas below the knee where the skin barrier is damaged. This is caused primarily by vasculitis, antiphospholipid antibodies, gangrenous pustular dermatosis, or cutaneous calcinosis.[19] Cutaneous calcinosis is rarely seen in SLE patients and usually occurs in female patients.[22] It typically appears during the later stages of the disease, and occasionally can also present as skin ulcers.[23,24]

Lupus erythematosus panniculitis is a rare subtype of SLE, and in the early stages of the disease, skin calcification is emphasized as an important histopathological finding.[25] Panniculitis occurs in approximately 2–5% of SLE patients, with a higher prevalence in women. Due to the rarity of LEP, there are limited data on the sex distribution and the specific factors contributing to sex differences in incidence.[23] Consequently, treatment for LEP is more restricted. Currently, LEP treatment follows the same protocols as that for SLE, including antimalarial drugs, corticosteroids, and immunosuppressants. For patients with skin calcification and ulcers, the primary treatment approach involves multidisciplinary collaboration, including consultation with specialists in nephrology, dermatology, wound care, nutrition, and pain management. Wound care represents the foundation of treatment, and focuses on removing necrotic tissue, promoting wound healing, preventing infection, and addressing the severe pain caused by the condition itself. However, the overall treatment outcomes remain suboptimal.[26] In the present case, despite conventional treatment with corticosteroids combined with cyclophosphamide and regular wound care, the ulcers on the patient's lower limbs failed to heal, and globulin levels, instead of decreasing, increased, with the lupus activity remaining severe. However, after the addition of Telitacicept, the patient's overall condition improved significantly. This is consistent with the findings of Gupta et al on two cases of refractory LEP, where treatment with rituximab yielded excellent results, effectively controlling and alleviating the patients' conditions. Therefore, biologics play a significant role in the treatment of lupus panniculitis.

Telitacicept is a novel dual-targeted biologic agent with immunosuppressive properties, and has shown significant efficacy in treating disorders of the immune system and thus holds significant therapeutic promise. In the present study, we observed significant improvements in a patient with LEP in terms of symptoms, signs, laboratory findings, imaging, and quality of life following treatment with Telitacicept. This finding not only provides robust clinical evidence and experience supporting the use of Telitacicept for treating LEP but also offers valuable insights into its advantages over other biologics and treatment modalities, thereby providing important guidance for the future standardization of LEP therapy. From the pharmacological perspective, Telitacicept is a novel fusion protein that significantly inhibits the activity of BLyS and APRIL, thereby suppressing the development and survival of plasma cells and mature B cells, with consequent good therapeutic effects on autoimmune diseases.[27] Multiple clinical studies have also confirmed that combination therapies including Telitacicept for SLE can reduce disease activity while also decreasing the dosages of oral medications, including corticosteroids. In terms of efficacy and safety, Telitacicept shows excellent safety. For example, in a case of SLE with hepatitis, the patient was heavily dependent on corticosteroids. After switching to belimumab therapy, the liver function did not improve significantly and showed a trend toward worsening. However, after switching to Telitacicept combination therapy, the patient's liver function improved markedly and eventually returned to normal, allowing gradual reductions in the corticosteroid dosage. In a controlled study of 15 patients with refractory cSLE who were intolerant to glucocorticoids, regular treatment with Telitacicept resulted in significantly improved renal function, control of proteinuria, a significant increase in SRI-4 scores, and a gradual reduction in glucocorticoid dosage.[28] Mild to moderate adverse reactions were manageable. In another study on Schnitzler syndrome, patients who received 16 doses of belimumab showed slight improvements but continued to experience relapses. After switching to treatment with Telitacicept, the conditions of the patients stabilized, their blood counts returned to normal,

the SLEDAI scores were 0, and no relapses occurred during a three-year follow-up period.[29] Therefore, compared to the the single targeting of BlyS by belimunab, Telitacicept demonstrates a clear advantage.

The current standard treatment approach for LEP primarily follows the therapeutic protocols established for SLE, particularly in refractory cases where no specific treatment regimen has yet been developed. There is not a great deal of information on the efficacy of Telitacicept in treating skin damage associated with SLE. Recently, several studies have reported the use of Telitacicept to treat stubborn skin erythema in a patient with SLE, achieving very good results.[30] In the present case, we found that the LEP patient did not achieve satisfactory results with hormone therapy combined with immunosuppressants, while the addition of Telitacicept resulted in significant improvements in the patient's lower limb ulcers, while the levels of various biochemical and immune indicators also improved or even returned to normal. This indicates the potential of using Telitacicept for LEP treatment. Although this study described only a single case, the findings provide new ideas and strategies for LEP treatment, as well as a reference for the formulation of specific treatment strategies for lupus panniculitis.However, to our knowledge, this case also represents the first reported instance of Telitacicept being successfully employed in the treatment of LEP, making it highly instructive.

However, in clinical practice, further extensive research and validation are required, focusing not only on the collection of clinical data collection but also on basic research. Meanwhile, we must be mindful of its adverse reactions, including exacerbation of infections, haematological damage, gastrointestinal reactions, and hepatic impairment.[31,32] This case study demonstrates the therapeutic value of Telitacicept in a single patient with lupus erythematosus panniculitis. However, due to individual variability, it is not known whether the same therapeutic effects can be achieved in other patients or whether unknown adverse reactions may arise. Nevertheless, the success achieved in this case provides a valuable reference for the development of future systematic guidelines for the treatment of LEP, and Telitacicept is a promising candidate for inclusion in such guidelines.

## Ethics Statement

This study does not require institutional approval.

## Patient Consent Form

The patient provided written informed consent for the publication of the patient's clinical data and accompanying images.

## Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

## Funding

This work was supported by grants from the 2022 Guidance Project of Jishou University (grant number:Jdzd22023).

## Disclosure

The authors report no conflicts of interest in this work.

## References

1. Abdelrahman FAF, El-Sayed SA, Abuel-Atta AA, et al. Nephrotoxicity induced by different diameters of sphere gold nanoparticles with special emphasis on the nephroprotective role of quercetin. *Open Vet J*. 2023;13(6):723–731. doi:10.5455/OVJ.2023.v13.i6.7
2. Carsuzaa F, Pierre C, De Jaureguiberry JP, et al. [Panniculitis-fasciitis syndrome disclosing systemic erythematous lupus]. *Ann Dermatol Venereol*. 1996;123(4):259–261.
3. Gondane S, Kothiwala R, Dangi S, et al. Lupus Erythematosus Panniculitis in Pregnancy. *Indian J Dermatol*. 2015;60(6):637. doi:10.4103/0019-5154.169164
4. Rangel LK, Villa-Ruiz C, Lo K, et al. Clinical characteristics of Lupus erythematosus panniculitis/profundus: a retrospective review of 61 patients. *JAMA Dermatol*. 2020;156(11):1264–1266. doi:10.1001/jamadermatol.2020.2797

5. Zucchi D, Silvagni E, Elefante E, et al. Systemic lupus erythematosus: one year in review 2023. *Clin Exp Rheumatol*. 2023;41(5):997–1008. doi:10.55563/clinexprheumatol/4uc7e8
6. Allen ME, Rus V, Szeto GL. Leveraging heterogeneity in systemic lupus erythematosus for new therapies. *Trends Mol Med*. 2021;27(2):152–171. doi:10.1016/j.molmed.2020.09.009
7. Bano S, Bombardieri S, Doria A, et al. Lupus erythematosus and the skin. *Clin Exp Rheumatol*. 2006;24(1 Suppl 40):S26–35.
8. Pearce L. Systemic lupus erythematosus. *Nurs Stand*. 2016;30(43):17. doi:10.7748/ns.30.43.17.s21
9. Yokogawa M, Takaishi M, Nakajima K, et al. Epicutaneous application of toll-like receptor 7 agonists leads to systemic autoimmunity in wild-type mice: a new model of systemic Lupus erythematosus. *Arthritis Rheumatol*. 2014;66(3):694–706. doi:10.1002/art.38298
10. Shen SZ, Cai JY, Sun LY, et al. Clinical study on the treatment of patients with systemic lupus erythematosus using telitisib combined with cyclophosphamide. *Chinese Journal of Clinical Pharmacology*. 2025;41(10):1367–1372.
11. Li X, Wu X, Pan T. Clinical study on the treatment of systemic lupus erythematosus with tetrahydrozoline combined with hydroxychloroquine sulfate. *Modern Drugs and Clinical Practice*. 2024;39(21):2871–2876. doi:10.7501/j.issn.1674-5515.2024.11.021
12. Vale E, Garcia LC. Cutaneous lupus erythematosus: a review of etiopathogenic, clinical, diagnostic and therapeutic aspects. *An Bras Dermatol*. 2023;98(3):355–372. doi:10.1016/j.abd.2022.09.005
13. Marinho A, Delgado Alves J, Fortuna J, et al. Biological therapy in systemic lupus erythematosus, antiphospholipid syndrome, and Sjögren's syndrome: evidence- and practice-based guidance. *Front Immunol*. 2023;14:1117699. doi:10.3389/fimmu.2023.1117699
14. Samy E, Wax S, Huard B, et al. Targeting BAFF and April in systemic lupus erythematosus and other antibody-associated diseases. *Int Rev Immunol*. 2017;36(1):3–19. doi:10.1080/08830185.2016.1276903
15. Möckel T, Basta F, Weinmann-Menke J, et al. B cell activating factor (BAFF): structure, functions, autoimmunity and clinical implications in Systemic Lupus Erythematosus (SLE). *Autoimmun Rev*. 2021;20(2):102736. doi:10.1016/j.autrev.2020.102736
16. Zeng L, Yang K, Wu Y, et al. Telitacicept: a novel horizon in targeting autoimmunity and rheumatic diseases. *J Autoimmun*. 2024;148:103291. doi:10.1016/j.jaut.2024.103291
17. Blair HA, Duggan ST. Belimumab: a Review in Systemic Lupus Erythematosus. *Drugs*. 2018;78(3):355–366. doi:10.1007/s40265-018-0872-z
18. Fanouriakis A, Tziolos N, Bertsias G, et al. Update on the diagnosis and management of systemic lupus erythematosus. *Ann Rheum Dis*. 2021;80(1):14–25. doi:10.1136/annrheumdis-2020-218272
19. Pons-Estel BA, Bonfa E, Soriano ER, et al. First Latin American clinical practice guidelines for the treatment of systemic lupus erythematosus: Latin American Group for the Study of Lupus (GLADEL, Grupo Latino Americano de Estudio del Lupus)-Pan-American League of Associations of Rheumatology (PANLAR). *Ann Rheum Dis*. 2018;77(11):1549–1557. doi:10.1136/annrheumdis-2018-213512
20. Jiang WJ. Observation on the efficacy and safety of tixicodin in the treatment of severe active systemic lupus erythematosus. *Jiangxi Medicine*. 2020;55(11):1637–1641. doi:10.3969/j.issn.1006-2238.2020.11.029
21. Jin ZH. A study on the efficacy and safety of tixicodin in the treatment of systemic lupus erythematosus and its impact on age-related B cells. *USTC (University of Science and Technology of China)*. 2024;15:45–78.
22. Deng GM, Tsokos GC. Pathogenesis and targeted treatment of skin injury in SLE. *Nat Rev Rheumatol*. 2015;11(11):663–669. doi:10.1038/nrrheum.2015.106
23. Lederhandler M, Valins W, Zoghbi Z, et al. Leg ulcers in systemic lupus erythematosus associated with underlying dystrophic calcinosis and bone infarcts in the absence of antiphospholipid antibodies. *JAAD Case Rep*. 2016;2(2):164–167. doi:10.1016/j.jdcr.2016.02.009
24. Park YM, Lee SJ, Kang H, et al. Large subcutaneous calcification in systemic lupus erythematosus: treatment with oral aluminum hydroxide administration followed by surgical excision. *J Korean Med Sci*. 1999;14(5):589–592. doi:10.3346/jkms.1999.14.5.589
25. Fraga J, García-Díez A. Lupus erythematosus panniculitis. *Dermatol Clin*. 2008;26(4):453–63,vi. doi:10.1016/j.det.2008.06.002
26. Zhou Y, Chen Y, Yin G, et al. Calciphylaxis and its co-occurrence with connective tissue diseases. *Int Wound J*. 2023;20(4):1316–1327. doi:10.1111/iwj.13972
27. Fan Q, Ji H, Liu Y, et al. Refractory lupus hepatitis successfully treated with telitacicept who failed to belimumab: a case report and literature review. *Lupus*. 2024;33(4):414–419. doi:10.1177/09612033241233021
28. Sun L, Shen Q, Gong Y, et al. Safety and efficacy of telitacicept in refractory childhood-onset systemic lupus erythematosus: a self-controlled before-after trial. *Lupus*. 2022;31(8):998–1006. doi:10.1177/09612033221097812
29. Sun S. Talticept for the treatment of systemic lupus erythematosus combined with Schnitzler syndrome. *Chinese Journal of Integrated Traditional and Western Medicine in Nephrology*. 2023;34(14):1113–1114.
30. Diao L, Tang N, Zhang C, et al. Avermectin induced DNA damage to the apoptosis and autophagy in human lung epithelial A549 cells. *Ecotoxicol Environ Saf*. 2021;215:112129. doi:10.1016/j.ecoenv.2021.112129
31. Jin Y, Zhu J, Sheng A, et al. Telitacicept as a BAFF/April dual inhibitor: efficacy and safety in reducing proteinuria for refractory childhood IgA vasculitis nephritis. *Pediatr Nephrol*. 2025;40(8):2561–2569. doi:10.1007/s00467-025-06769-3
32. Dhillon S. Telitacicept: first approval. *Drugs*. 2021;81(14):1671–1675. doi:10.1007/s40265-021-01591-1