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CASE SERIES

Telitacicept: A New Therapy for the Treatment of Optic Neuromyelitis Spectrum Disease Associated with Other Autoimmune Disorders

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Abstract: Neuromyelitis optica spectrum disorders (NMOSD) are primarily autoimmune diseases mediated by B cells and AQP4-IgG antibodies, typically affecting the optic nerves and spinal cord, and are characterised by high relapse rates and significant disability. We present two cases of NMOSD patients who also had systemic lupus erythematosus (SLE), with one case additionally complicated by myasthenia gravis (MG). Both patients initially received first-line treatment with corticosteroids; however, no clinical improvement was observed; As a result, the treatment was switched to the dual-target biologic agent, Telitacicept. Following the administration of Telitacicept, both patients demonstrated significant improvements in clinical symptoms, daily functional abilities, and imaging findings. This report highlights the successful use of Telitacicept in treating NMOSD complicated by other autoimmune diseases, which may serve as an important reference for the management of NMOSD.

Keywords: neuromyelitis optica spectrum disorders, Telitacicept, BLyS, APRIL

Introduction

Neuromyelitis optica spectrum disorders (NMOSD) constitute a group of autoimmune-mediated, inflammatory demyelinating diseases affecting the central nervous system (CNS), predominantly involving the optic nerves and spinal cord.[1,2] The clinical spectrum typically encompasses optic neuritis (ON) and longitudinally extensive transverse myelitis (LETM), with potential brainstem involvement manifesting as area postrema syndrome (APS).[3] NMOSD is characterised by a notably high rate of relapse and disability.[4] Current therapeutic strategies for NMOSD involve a tiered immunotherapy approach. First-line acute management typically includes high-dose intravenous methyprednisolon (IVMP),[5] plasma exchange (PE),[6] or intravenous immunoglobulin (IVIG).[7] Commonly used immunosuppressive agents during the remission period include mycophenolate mofetil (MMF),[8] azathioprine (AZA),[9] rituximab (RTX),[10] ofatumumab.[11]

The pathogenesis of Neuromyelitis Optica Spectrum Disorder (NMOSD) is closely linked to the differentiation of B cells into plasmablasts, which produce pathogenic aquaporin-4 antibodies (AQP4-IgG).[12,13] MG is primarily an autoimmune disease caused by the aberrant activation of B cells and the production of pathogenic antibodies.[14] Systemic Lupus Erythematosus (SLE), another autoimmune condition, is primarily driven by an amplified B cell immune response and the production of autoantibodies.[15] Thus, B cells play a crucial role in the autoimmune diseases. B lymphocyte stimulating factor (BLyS), also known as B lymphocyte activation factor (BAFF), and proliferation-inducing ligand (APRIL) play important roles in the development, proliferation and survival of B cells by binding to the BAFF receptor (BAFF-R),[16] transmembrane activator and calcium regulator and cyclophilin ligand interaction (TACI),[17] and the B-cell maturation antigen (BCMA).[18]

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Telitacicept is a novel recombinant fusion protein comprising the transmembrane activator and calcium modulator and cyclophilin ligand interactor (TACI) receptor and the crystallisable fragment (Fc) of human immunoglobulin G (IgG).[19] It simultaneously targets the two cytokines, BLyS/BAFF and APRIL, thereby indirectly inhibiting B cell proliferation and survival, and reducing the production of pathogenic antibodies and immunoglobulins.[20] In 2021, Telitacicept was approved by the China National Medical Products Administration for the treatment of patients with SLE.[19] A single-centre, single-arm, open-label study involving eight AQP4-IgG-positive patients with recurrent NMOSD demonstrated favourable clinical outcomes following PE combined with Telitacicept treatment.[21] A multicentre, randomised, open-label, Phase 2 clinical study investigating the use of Telitacicept in MG demonstrated that, after 24 weeks of follow-up, Telitacicept exhibited favourable clinical efficacy.[22] Currently, Telitacicept is also widely used in the treatment of other B-cell-mediated autoimmune diseases, such as rheumatoid arthritis.[23]

Presentation

Case 1

A 40-year-old female patient was admitted to our department in January 2024, presenting with symptoms of numbness in both lower limbs, weakness of the left limb, urinary dysfunction, and ptosis. She had a medical history of myasthenia gravis (MG) and NMOSD. She had been diagnosed with MG 10 years prior, and a computed tomography (CT) scan of the chest revealed an anterior mediastinal mass (histopathologically diagnosed as thymic hyperplasia). She underwent PE and thymectomy and was treated with pyridostigmine (90 mg orally, three times daily). She reported adequate symptom control and discontinued the medication independently after two and a half years. Six months ago, the patient developed skin pruritus, joint pain, and elevated blood pressure (140/95 mmHg), subsequently, the patient went to a local hospital for medical consultation. An autoimmune antibody profile revealed an elevated level of anti-double-stranded DNA antibody (dsDNA) at 15 IU/mL (reference range: 0–10 IU/mL). However, the patient stated that no medications were taken to manage these symptoms. Three months prior, she sought medical attention at a local hospital due to numbness in the left limb. Magnetic resonance imaging (MRI) revealed linear and patchy areas of hyperintense signal on T2-weighted imaging within the cervical spinal cord, alongside patchy regions of hyperintense T2 signal observed in the thoracic spinal cord, post-contrast imaging demonstrated significant enhancement in these lesions and serological immunity tests indicated AQP4-IgG positivity, supporting a diagnosis of NMOSD. She had been regularly taking prednisone and MMF outside the hospital. Upon this admission, physical examination revealed a malar rash on the face, oral ulcers, a positive fatigue test, muscle strength of IV+ in the right lower limb, IV–in the left lower limb, and decreased sensation below the waist. The Expanded Disability Status Scale (EDSS) score was 6 points. MRI showed abnormal signals in the cervical and thoracic spinal cord. Repetitive nerve stimulation showed a low-frequency decrement exceeding 10%. Cell-based assay (CBA) with indirect immunofluorescence tests indicated positivity for AQP4-IgG in both serum and cerebrospinal fluid (CSF) (serum titer 1:10, CSF titer 1:10). Enzyme-linked immunosorbent assay (ELISA) tests indicated serum anti-acetylcholine receptor antibody (AchR-Ab) was 210.4 pg/mL (reference value <4.5 pg/mL). Residual positive test results included anti-double-stranded DNA antibody (dsDNA) 68.81 IU/mL (reference range 0–10IU/mL) and 24-hour urinary protein quantifications showing total urine volume of 2950mL/24hours. The final diagnosis was neuromyelitis optica spectrum disorder (NMOSD), myasthenia gravis (MG), and systemic lupus erythematosus (SLE).

After confirming the diagnosis, the patient received high-dose methylprednisolone pulse therapy during the acute phase, which involved intravenous infusion of methylprednisolone succinate at doses of 1000mg, 500mg, 250mg, 120mg sequentially, once daily, each for three consecutive days, followed by oral administration. However, on the second day of the 500 mg infusion, the patient's condition deteriorated, with weakness developing in the right limb. Physical examination revealed muscle strength of grade III in the affected limb, and the Expanded Disability Status Scale (EDSS) score increased to 6.5 points. Despite this, the patient reported no subjective discomfort, and no new lesions were observed on MRI. The deterioration was considered to be related to acute-phase exacerbation, and the use of intravenous immunoglobulin (IVIG) was recommended. However, the patient declined this option due to cost concerns. Methylprednisolone pulse therapy was continued, and by the end of the pulse therapy, the muscle strength of the right upper limb had improved to grade III+, with the patient reporting a subjective sense of well-being, however, there was no

significant change in the EDSS score. This necessitated a more cautious approach in selecting subsequent treatment options. Given the coexistence of multiple immune diseases and the high risk of relapse, we opted for a multi-targeted biologic agent capable of precise immune modulation, which could address multiple immune diseases simultaneously while demonstrating long-term clinical efficacy, safety, and a reduced risk of relapse. Therefore, we chose the biologic agent Telitacicept. Following a thorough discussion with the patient, treatment with Telitacicept was initiated (240 mg, subcutaneous injection, once weekly for a total of 24 injections) alongside oral prednisone (48 mg/day). Two weeks after the first injection, the patient showed clinical improvement, with symptoms of numbness and weakness in both lower limbs slightly alleviated, and she reported improved urination, allowing for catheter removal. By the eighth injection (the 7th week after the initial injection), the patient continued to improve, enabling her to walk slowly without assistance, and the EDSS score decreased to 5.5. The prednisone reduced to 20mg/day. MRI revealed linear and patchy areas of hyperintense signal on T2-weighted imaging within the cervical spinal cord, as well as patchy regions of hyperintense T2 signal in the thoracic spinal cord. Post-contrast imaging demonstrated significant enhancement in these lesions. At the 3rd month follow-up, to our surprise, the patient was able to walk independently and perform simple daily activities without assistance. MRI showed that the range of cervical and thoracic lesions was reduced, with the EDSS score improving to 5.0 points, and prednisone was completely discontinued. At the 6th month, the patient reported being able to walk freely and independently perform most daily activities, with the condition remaining stable MRI demonstrated linear and patchy areas of hyperintense signal on T2-weighted imaging within the cervical spinal cord, as well as patchy regions of hyperintense T2 signal in the thoracic spinal cord. Post-contrast imaging revealed significant enhancement in these lesions, which showed marked reduction and improvement compared to previous scans. The EDSS score significantly decreased to 2.0 points, but the AQP4-IgG test was positive. Two months after the final injection, the patient's condition remained stable The results of EDSS score, modified Rankin Scale (mRS) score, and Hamilton Anxiety Scale (HAMA) score are shown in (Figure 1).

The variations in laboratory test results included memory B cells (defined as CD45+CD19+CD27+CD38- cells), plasma blasts (defined as CD45+CD19+CD27+CD38+ cells), plasma cells (defined as CD45+CD19+CD27+CD38 +CD138+ cells), as well as levels of IgG, BCMA, BLyS, APRIL, AchR-Ab, and dsDNA, summarized in the figures below (Figures 2A–C and 3). At one month post-treatment, the number of memory B cells decreased, followed by an increase at week seven, after which it declined again. In contrast, the number of plasmablasts and plasma cells increased at one month but decreased by week seven, continuing to decline thereafter (Figure 2A). Serum levels of BLyS, APRIL, IgG, BCMA, AchR-Ab, and dsDNA demonstrated a decrease at the one-month mark after initial injection, increased at week seven, and subsequently showed a declining trend (Figures 2B, C and 3).

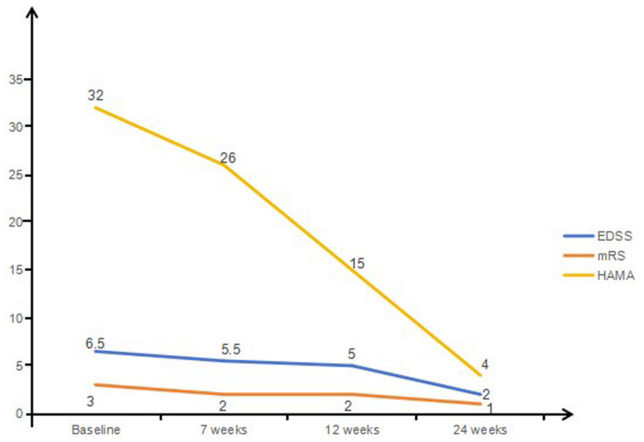

Figure 1 Evolutionof clinical severity of NMOSD in the patient, assessed through EDSS score, mRS score, HAMA score.

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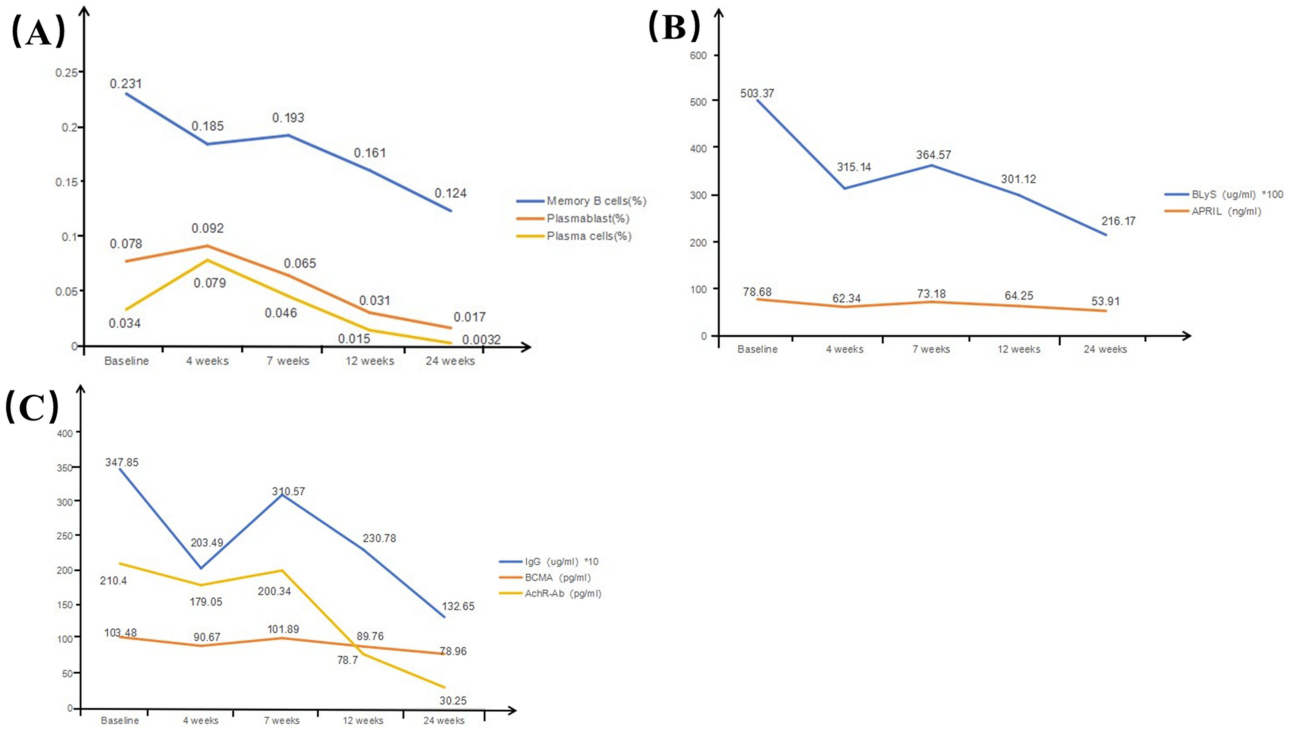

Figure 2 (**A**) The changes in frequency of memory B cells, plasmablast, and plasma cells in the patient. (**B**) The changes of levels of serum BLyS and APRIL in the patient. (**C**) The changes of levels serum IgG, BCMA, and AChR-Ab in the patient.

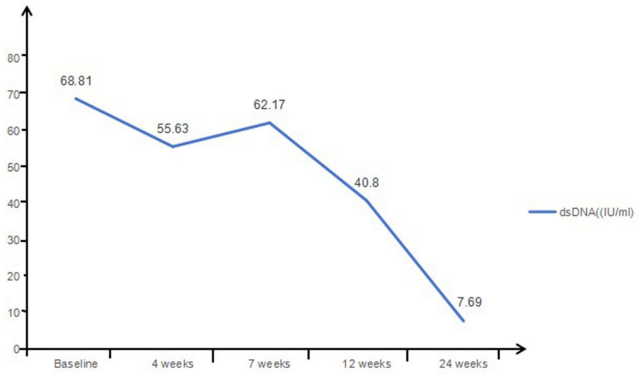

Figure 3 The changes of levels dsDNA in the patient.

Case 2

A 16-year-old female student initially presented to the gastroenterology department of a local hospital with symptoms of nausea and vomiting. During the course of treatment, she developed dizziness and gait instability, which necessitated a referral to the neurology department. Laboratory investigations revealed a serum AQP4-IgG titer of 1:100. MRI demonstrated abnormal signal changes in the dorsal medulla and at the atlantoaxial level of the cervical spinal cord, leading to a diagnosis of NMOSD. She was treated with corticosteroids and immunoglobulin therapy, followed by regular outpatient administration of prednisone (48 mg/ day, reducing by 4mg per week) and MMF (0.5g/ day).

Two months later, she experienced a recurrence of gait instability and was admitted to the neurology department of our hospital. Upon admission, clinical examination revealed a butterfly-shaped facial rash, heightened sensitivity to bright light, muscle strength in both lower limbs graded at IV+, a positive Romberg test, gait instability characterized by a “tandem walk”, and instability on the heel-knee-shin test. The EDSS score at admission was 6.0 points. Cranial MRI

again showed abnormal signal changes in the dorsal medulla and at the atlantoaxial level, suggestive of inflammatory lesions. CBA testing demonstrated positive AQP4-IgG in both serum and cerebrospinal fluid (CSF) (serum titer 1:100, CSF titer 1:10). Additionally, anti-double-stranded DNA (dsDNA) antibody testing was 56.17 RU/mL, and a 24-hour urine protein quantification revealed a total urinary output of 3000 mL/24h. Ultimately, she was diagnosed with NMOSD accompanied by SLE.

The patient commenced high-dose methylprednisolone pulse therapy during the acute phase. However, her symptoms did not improve significantly; Instead, she developed weakness in both lower limbs, resulting in an increase in her EDSS score to 6.5 points. Nevertheless, the patient reported no significant discomfort, and no new lesions were observed on MRI. Given the patient's young age at onset, rapid disease progression, and considerations regarding prognosis, it was necessary to select a treatment that could swiftly control disease progression, improve outcomes, and ensure long-term safety and efficacy while extending the interval between relapses. As the patient was under 18 years of age, after thorough discussion with the family, treatment with Telitacicept was initiated (240 mg, administered via subcutaneous injection once weekly for 24 weeks). Similar to the first patient, clinical symptoms began to improve around the second week of treatment. The EDSS score decreased to 4.5 by the third month and further improved to 2.0 by the sixth month. Prednisone was discontinued by the third month, although AQP4-IgG remained positive. Two months after the final injection, the patient's symptoms continued to improve, and her condition remained stable.

Discussion

As a novel dual-target biologic agent, Telitacicept has demonstrated promising potential in the treatment of NMOSD complicated with other autoimmune diseases. In this study, two NMOSD patients with concurrent SLE, one of whom also had MG, showed significant improvements in clinical symptoms, daily living abilities, and imaging findings following Telitacicept treatment. These findings not only provide clinical evidence supporting the application of Telitacicept in the treatment of such complex autoimmune disorders, but also prompt further investigation into its mechanism of action, its distinctions from conventional therapies, and its potential therapeutic value across various disease contexts. From a mechanistic perspective, Telitacicept, as an antibody fusion protein molecule, demonstrates dual targeting capability towards the BLyS/BAFF-APRIL system, which plays a crucial role in maintaining B-cell homeostasis.[19] BLyS/BAFF regulates the differentiation and maturation of immature B cells through its binding to BAFF-R, TACI, and BCMA,[16,20,24] while simultaneously supporting B cell proliferation, plasma cell survival, and class switch recombination. Similarly, APRIL modulates plasma cell function and survival via its interaction with TACI and BCMA.[20,24] Telitacicept exerts its therapeutic effects through dual mechanisms: firstly, by targeting BLyS /BAFF on B cell surfaces, it impedes the transition from pro-B cells to pre-B cells and subsequently to mature B cells, thereby reducing the generation of abnormal B cells. Secondly, through its targeting of APRIL on B cell surfaces, it disrupts the differentiation cascade from B cells to plasma cells and antibody production, consequently suppressing B cell proliferation and survival. This dual action ultimately leads to a reduction in pathogenic antibody and immunoglobulin production. Telitacicept exhibits a distinct mechanism of action compared to currently available monoclonal antibody therapies. Conventional monoclonal antibodies, such as rituximab and inebilizumab, exert their immunomodulatory effects through direct interaction with B cells, inducing B cell apoptosis.[25,26] In contrast, Telitacicept's unique dual-targeting approach modulates B cell activity through alternative pathways, offering a novel therapeutic strategy in autoimmune disease management. From a safety perspective, Telitacicept demonstrates a favourable profile by selectively targeting abnormally proliferating B cells and plasma cells rather than inducing global B cell depletion. Through its competitive binding to BLyS and APRIL, it effectively inhibits the proliferation of aberrant B lymphocytes and antibody production by plasma cells, while preserving the function of normal B lymphocytes and plasma cells. This selective mechanism allows for the maintenance of essential immune functions, thereby reducing the risk of infections.[27,28] Consequently, Telitacicept exhibits superior safety compared to both B cell-depleting agents and conventional immunosuppressants. Regarding therapeutic indications, both patients in this study presented with multiple autoimmune comorbidities, and Telitacicept demonstrated favourable efficacy in these complex clinical scenarios. However, concerning its potential application in isolated NMOSD cases, theoretical considerations suggest that Telitacicept's mechanism of action remains relevant, given the established pathogenic role of B cell-derived AQP4-IgG antibodies in NMOSD development. The B cell-

modulating properties of telitacicept may therefore prove equally effective in isolated NMOSD cases. Supporting this notion, research conducted by Guan Yangtai et al suggests that Telitacicept administration following plasma exchange could emerge as a novel therapeutic strategy for relapsing NMOSD patients.[21] Nevertheless, large-scale, multicentre randomised controlled trials are warranted to further validate the efficacy and safety profile of telitacicept in treating isolated NMOSD cases. In this study, Telitacicept demonstrated significant therapeutic efficacy in two patients who had failed to respond to conventional first-line treatments, including corticosteroids. The treatment outcomes were particularly noteworthy, with patients exhibiting symptomatic relief, remarkable reduction in lesion size as evidenced by MRI findings, and substantial improvement in EDSS scores. However, when compared with established second-line therapies for NMOSD or multiple immune-mediated disorders, the current clinical evidence remains insufficient to substantiate Telitacicept's superiority in terms of long-term efficacy, safety profile, and relapse rates. Therefore, large-scale, multicentre clinical trials are warranted to further validate these preliminary findings and establish its therapeutic position in the management of such complex conditions. This study, through the analysis of two cases of NMOSD complicated with other autoimmune disorders, has demonstrated the remarkable therapeutic potential of Telitacicept in the management of autoimmune diseases. Although the findings warrant preliminary conclusions, they provide compelling evidence for a novel alternative therapeutic strategy in NMOSD and related autoimmune conditions. The observed clinical outcomes suggest that this approach may offer superior therapeutic prospects, meriting further investigation in subsequent phases of clinical research.

Consent for Publication

Informed consents were obtained from the patients. (The minor has received the consent of the guardian).

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.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Kitley J, Leite MI, Nakashima I, et al. Prognostic factors and disease course in aquaporin-4 antibody-positive patients with neuromyelitis optica spectrum disorder from the United Kingdom and Japan. *Brain*. 2012;135(Pt 6):1834–1849. doi:10.1093/brain/aws109
2. Klein da Costa B, Brant de Souza Melo R, Passos GRD, et al. Unraveling B lymphocytes in CNS inflammatory diseases: distinct mechanisms and treatment targets. *Neurology*. 2020;95(16):733–744. doi:10.1212/wnl.0000000000010789
3. Wingerchuk DM, Banwell B, Bennett JL, et al. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. *Neurology*. 2015;85(2):177–189. doi:10.1212/wnl.0000000000001729
4. Palace J, Lin DY, Zeng D, et al. Outcome prediction models in AQP4-IgG positive neuromyelitis optica spectrum disorders. *Brain*. 2019;142(5):1310–1323. doi:10.1093/brain/awz054
5. Wang S, Xue M, Wang J, et al. Effects of intravenous pulse methylprednisolone in neuromyelitis optica during the acute phase. *Ann Clin Transl Neurol*. 2024;11(10):2731–2744. doi:10.1002/acn3.52188
6. Bonnan M, Valentino R, Olindo S, Mehdaoui H, Smadja D, Cabre P. Plasma exchange in severe spinal attacks associated with neuromyelitis optica spectrum disorder. *Multi sclero*. 2009;15(4):487–492. doi:10.1177/1352458508100837
7. Schwab I, Nimmerjahn F. Intravenous immunoglobulin therapy: how does IgG modulate the immune system?. *Nat Rev Immunol*. 2013;13(3):176–189. doi:10.1038/nri3401
8. Akatani R, Chihara N, Koto S, et al. Efficacy and safety of mycophenolate mofetil for steroid reduction in neuromyelitis optica spectrum disorder: a prospective cohort study. *Immunol Med*. 2024;47(2):85–92. doi:10.1080/25785826.2024.2304364
9. Tang Q, Yao M, Huang Y, Bian J, Wang Y, Ji W. A comparison of the efficacy of tocilizumab versus azathioprine for neuromyelitis optica spectrum disorder: a study protocol for systematic review and meta-analysis. *Medicine*. 2023;102(4):e32748. doi:10.1097/md.0000000000032748
10. Dong GY, Meng YH, Xiao XJ. A meta-analysis on efficacy and safety of rituximab for neuromyelitis optica spectrum disorders. *Medicine*. 2022;101(36):e30347. doi:10.1097/md.0000000000030347
11. Zhan Y, Zhao M, Li X, et al. A meaningful exploration of ofatumumab in refractory NMOSD: a case report. *Front Immunol*. 2023;14:1208017. doi:10.3389/fimmu.2023.1208017

12. Lennon VA, Wingerchuk DM, Kryzer TJ, et al. A serum autoantibody marker of neuromyelitis optica: distinction from multiple sclerosis. *Lancet*. 2004;364(9451):2106–2112. doi:10.1016/s0140-6736(04)17551-x
13. Carnero Contentti E, Correale J. Neuromyelitis optica spectrum disorders: from pathophysiology to therapeutic strategies. *J Neuroinflammation*. 2021;18(1):208. doi:10.1186/s12974-021-02249-1
14. Trinh VB, Fairclough RH. Development of peptide mimics of the human acetylcholine receptor main immunogenic region for treating myasthenia gravis. *Int J mol Sci*. 2024;26(1):229. doi:10.3390/ijms26010229
15. Cooper GS, Dooley MA, Treadwell EL, St Clair EW, Parks CG, Gilkeson GS. Hormonal, environmental, and infectious risk factors for developing systemic lupus erythematosus. *Arthritis Rheum*. 1998;41(10):1714–1724. doi:10.1002/1529-0131(199810)41:10<1714::Aid-art3>3.0.Co;2-u
16. Thompson JS, Bixler SA, Qian F, et al. BAFF-R, a newly identified TNF receptor that specifically interacts with BAFF. *Science*. 2001;293(5537):2108–2111. doi:10.1126/science.1061965
17. Yao X, Ren Y, Zhao Q, et al. Pharmacokinetics analysis based on target-mediated drug distribution for RC18, a novel BLyS/APRIL fusion protein to treat systemic lupus erythematosus and rheumatoid arthritis. *Eur J Pharm Sci*. 2021;159:105704. doi:10.1016/j.ejps.2021.105704
18. Kampa M, Notas G, Stathopoulos EN, Tsapis A, Castanas E. The TNFSF members APRIL and BAFF and their receptors TACI, BCMA, and BAFFR in oncology, with a special focus in breast cancer. *Front Oncol*. 2020;10:827. doi:10.3389/fonc.2020.00827
19. Dhillon S. Telitacicept: first Approval. *Drugs*. 2021;81(14):1671–1675. doi:10.1007/s40265-021-01591-1
20. Rennert P, Schneider P, Cachero TG, et al. A soluble form of B cell maturation antigen, a receptor for the tumor necrosis factor family member APRIL, inhibits tumor cell growth. *J Exp Med*. 2000;192(11):1677–1684. doi:10.1084/jem.192.11.1677
21. Ding J, Jiang X, Cai Y, et al. Telitacicept following plasma exchange in the treatment of subjects with recurrent neuromyelitis optica spectrum disorders: a single-center, single-arm, open-label study. *CNS Neurosci Ther*. 2022;28(10):1613–1623. doi:10.1111/cns.13904
22. Yin J, Zhao M, Xu X, et al. A multicenter, randomized, open-label, phase 2 clinical study of telitacicept in adult patients with generalized myasthenia gravis. *Eur J Neurol*. 2024;31(8):e16322. doi:10.1111/ene.16322
23. 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
24. Wu Y, Bressette D, Carrell JA, et al. Tumor necrosis factor (TNF) receptor superfamily member TACI is a high affinity receptor for TNF family members APRIL and BLyS. *J Biol Chem*. 2000;275(45):35478–35485. doi:10.1074/jbc.M005224200
25. Ongphichetmetha T, Jitprapaikulsan J, Siritho S, Rattanathamsakul N, Detweeratham T, Prayoonwiwat N. Efficacy and safety of rituximab in multiple sclerosis and neuromyelitis optica spectrum disorder. *Sci Rep*. 2024;14(1):3503. doi:10.1038/s41598-024-53838-y
26. Fujihara K, Sato H. [efficacy and safety of inebilizumab, an anti-CD19 monoclonal antibody, for the treatment of neuromyelitis optica spectrum disorder: based on the N-momentum trial]. *Brain and Nerve*. 2024;76(10):1153–1160. doi:10.11477/mf.1416202751
27. Merrill JT, Wallace DJ, Wax S, et al. Efficacy and safety of atacicept in patients with systemic lupus erythematosus: results of a twenty-four-week, multicenter, randomized, double-blind, placebo-controlled, parallel-arm, phase IIb study. *Arthritis Rheumatol*. 2018;70(2):266–276. doi:10.1002/art.40360
28. van Vollenhoven RF, Kinnman N, Vincent E, Wax S, Bathon J. Atacicept in patients with rheumatoid arthritis and an inadequate response to methotrexate: results of a Phase II, randomized, placebo-controlled trial. *Arthritis Rheum*. 2011;63(7):1782–1792. doi:10.1002/art.30372