

IMMUNOMODULATORY DRUGS IN OPHTHALMOLOGY: A COMPREHENSIVE REVIEW OF INDICATIONS, CONTRAINDICATIONS AND EMERGING EVIDENCE

Dr. Vandana Jain*

Assistant Professor, Dept. of Ophthalmology, Index Medical College Indore, India.

Article Received: 07 August 2026 | Article Revised: 28 August 2026 | Article Accepted: 17 September 2026

*Corresponding Author: Dr. Vandana Jain

Assistant Professor Dept of Ophthalmology, Index Medical College Indore, India.

DOI: <https://doi.org/10.5281/zenodo.23053030>

How to cite this Article: Dr. Vandana Jain (2026) IMMUNOMODULATORY DRUGS IN OPHTHALMOLOGY: A COMPREHENSIVE REVIEW OF INDICATIONS, CONTRAINDICATIONS AND EMERGING EVIDENCE. World Journal of Pharmaceutical Science and Research, 5(10), 46-54.



Copyright © 2026 Dr. Vandana Jain | World Journal of Pharmaceutical Science and Research.

This work is licensed under creative Commons Attribution-NonCommercial 4.0 International license (CC BY-NC 4.0).

ABSTRACT

Background: Ocular inflammatory and autoimmune diseases represent a heterogeneous group of conditions that, when inadequately controlled, lead to irreversible structural damage and visual impairment. Conventional corticosteroids, while effective for acute suppression, are associated with a well-documented constellation of sight-threatening and systemic adverse effects with prolonged use, necessitating the integration of steroid-sparing immunomodulatory therapy (IMT). **Methods:** A comprehensive narrative review of peer-reviewed literature published between 2015 and 2025 was conducted using PubMed, MEDLINE, and Cochrane databases, encompassing randomized controlled trials, systematic reviews, meta-analyses, and landmark observational cohort studies. **Results:** Immunomodulatory drugs spanning antimetabolites, calcineurin inhibitors, alkylating agents, and biologic agents demonstrate variable but clinically meaningful efficacy across uveitis, scleritis, ocular mucous membrane pemphigoid, thyroid eye disease, and other immune-mediated ocular conditions. Biologic therapies—particularly anti-TNF agents, IL-6 inhibitors, and IL-17 pathway antagonists—have substantially expanded therapeutic options. **Conclusion:** A systematic, evidence-based approach to IMT selection, guided by disease phenotype, systemic associations, and patient-specific factors, is essential for optimizing visual outcomes while minimizing treatment-related morbidity.

KEYWORDS: immunomodulatory therapy; uveitis; biologics; methotrexate; mycophenolate mofetil; anti-TNF; thyroid eye disease; scleritis; ocular inflammation.

INTRODUCTION

Immune-mediated ocular diseases encompass a broad spectrum of conditions involving the uvea, sclera, cornea, orbit, and adnexa. Uveitis alone accounts for approximately 10–15% of legal blindness in developed nations and carries a disproportionate burden in working-age individuals, with an estimated global prevalence of 115 per 100,000 population.^[1] Despite the central role of corticosteroids in acute disease suppression, long-term use is constrained by well-documented complications, including posterior subcapsular cataract, secondary glaucoma, osteoporosis and adrenal suppression.^[2] The imperative to control inflammation while simultaneously achieving corticosteroid dose reduction has driven increasing reliance on immunomodulatory drugs (IMDs) across ophthalmic subspecialties.

Immunomodulatory therapy encompasses a diverse pharmacological group of agents that modulate pathological immune activation through distinct but often complementary mechanisms. These include classical antimetabolites (methotrexate, mycophenolate mofetil, azathioprine), calcineurin inhibitors (cyclosporine A, tacrolimus), alkylating agents (cyclophosphamide, chlorambucil) and a rapidly expanding armamentarium of targeted biologic agents.^[3] The First-Line Antimetabolites as Steroid-Sparing Treatment (FAST) uveitis trial and subsequent guidelines from the American Uveitis Society and the European League Against Rheumatism (EULAR) have substantially refined the evidence base for IMT in ocular inflammation.^[4,5] This review synthesizes contemporary evidence regarding indications, contraindications, monitoring requirements and emerging therapeutic applications of IMDs in ophthalmology.

Antimetabolite Agents

Methotrexate

Methotrexate (MTX) is a folate antagonist inhibiting dihydrofolate reductase, suppresses T-lymphocyte proliferation and modulates cytokine networks integral to ocular inflammation. It remains one of the most widely prescribed IMDs in uveitis management, particularly for pediatric uveitis associated with juvenile idiopathic arthritis (JIA-U), sarcoid uveitis, and intermediate uveitis. Doses range from 7.5 to 25 mg weekly with concurrent folic acid supplementation (1–5 mg/day on non-MTX days) to mitigate mucosal and hepatotoxic effects.^[6]

The Systemic Immunosuppressive Therapy for Eye Diseases (SITE) cohort—a multicenter study enrolling over 8,000 patients established MTX as achieving corticosteroid-sparing success in approximately 44% of uveitis patients at 1 year.^[7] Subsequently, the FAST randomized clinical trial demonstrated non-inferiority of mycophenolate mofetil compared to MTX in achieving treatment success at 6 months, with fewer hepatotoxic events in the MMF arm, thus providing comparative efficacy data for the two leading antimetabolites.^[4]

Indications: Non-infectious anterior, intermediate, posterior and panuveitis; JIA-associated uveitis; ocular cicatricial pemphigoid; and inflammatory scleritis refractory to NSAIDs. **Absolute contraindications:** Pregnancy (Category X; teratogenic), active hepatic disease, renal impairment (eGFR <30 mL/min/1.73m²), bone marrow suppression and active serious infection. Relative contraindications include significant alcohol use, hepatitis B or C co-infection and pulmonary fibrosis. Mandatory monitoring includes periodic complete blood counts, liver function tests, and renal biochemistry. High cumulative doses (>3.5–4 g) may warrant hepatic biopsy in at-risk patients.^[8]

Mycophenolate Mofetil

Mycophenolate mofetil (MMF) selectively inhibits inosine monophosphate dehydrogenase (IMPDH), curtailing de novo purine synthesis in lymphocytes and conferring a lymphocyte-selective immunosuppressive effect. Administered at 1–3 g/day in divided doses, MMF has emerged as a preferred first-line antimetabolite at many tertiary uveitis centers, valued for its tolerability and clinical versatility across multiple ocular inflammatory phenotypes.^[9]

Beyond uveitis, MMF demonstrates efficacy in ocular mucous membrane pemphigoid (MMP), where a prospective cohort study demonstrated superior mucosal disease control when combined with dapsone compared to dapsone monotherapy.^[10] MMF is additionally used in sympathetic ophthalmia and uveitis associated with systemic lupus erythematosus and inflammatory bowel disease. Its principal absolute contraindication is pregnancy associated with established embryopathy syndrome characterized by fetal facial malformations and cardiac defects. Relative contraindications include severe neutropenia and significant renal impairment, the latter requiring dose adjustment.

Azathioprine

Azathioprine (AZA), a purine analogue converted intracellularly to 6-thioguanine nucleotides, impairs both T- and B-lymphocyte proliferation. Dosed at 1–2.5 mg/kg/day, AZA retains important utility in Behçet's disease uveitis, where a controlled trial by Yazici et al. demonstrated significant reduction in ocular attack frequency and visual acuity loss compared to placebo—evidence that established AZA as the standard of care for this condition over three decades.^[11]

Thiopurine methyltransferase (TPMT) genotyping or phenotyping prior to AZA initiation is recommended, as patients with deficient enzyme activity are at heightened risk for life-threatening myelosuppression.^[12] Concurrent allopurinol use constitutes an absolute contraindication (unless AZA dose is reduced by 75%) due to xanthine oxidase inhibition potentiating thioguanine nucleotide accumulation.

Calcineurin Inhibitors

Cyclosporine A

Cyclosporine A (CsA) inhibits calcineurin-mediated dephosphorylation of NFAT transcription factors, suppressing IL-2 transcription and T-helper cell activation. In ophthalmology, CsA occupies a unique dual role encompassing systemic therapy for severe uveitis and topical formulations for ocular surface disease.^[13]

Systemic CsA (2.5–5 mg/kg/day) is indicated for Behçet's disease uveitis, Vogt-Koyanagi-Harada disease and sight-threatening uveitis refractory to antimetabolites. A controlled trial demonstrated superior ocular inflammation control in Behçet's uveitis with CsA versus colchicine. Nephrotoxicity and hypertension are the principal dose-limiting adverse effects requiring regular monitoring of serum creatinine, blood pressure and trough drug levels. Absolute contraindications include uncontrolled hypertension, pre-existing renal impairment (eGFR <30 mL/min) and active malignancy.^[13]

Topical CsA 0.05% (Restasis) and 0.1% cationic emulsion (Ikervis) are approved for moderate-to-severe dry eye disease (DED). The pivotal SANSIKA trial demonstrated statistically significant improvement in corneal fluorescein staining scores and Schirmer test values with 0.1% CsA compared to vehicle at 6 months.^[14] Topical CsA is further employed for vernal keratoconjunctivitis (VKC), atopic keratoconjunctivitis (AKC) and steroid-sparing management of corneal graft rejection.

Tacrolimus

Tacrolimus (FK506), acting via the FKBP-12 immunophilin with 10–100-fold greater potency than CsA, is established as a topical agent (0.03–0.1% ointment) for AKC and VKC. A randomized comparison of topical tacrolimus versus betamethasone in VKC reported equivalent efficacy with markedly fewer steroid-related ocular complications.¹⁵ Systemic tacrolimus (0.05–0.15 mg/kg/day) is employed in selected cases of refractory uveitis, particularly post-transplant uveitis and Behçet's disease unresponsive to CsA, leveraging its distinct immunophilin-binding profile.

Alkylating Agents

Cyclophosphamide

A nitrogen mustard prodrug generating alkylating metabolites that cross-link DNA, is reserved for the most severe ophthalmic inflammatory conditions given its significant toxicity profile. Principal indications include necrotizing scleritis associated with ANCA-associated vasculitis (granulomatosis with polyangiitis, GPA), rapidly progressive ocular MMP and orbital inflammatory disease refractory to conventional immunosuppression. Pulse intravenous cyclophosphamide (500–1000 mg/m²) offers equivalent efficacy to daily oral therapy with a reduced risk of urothelial toxicity; concurrent mesna administration and adequate hydration are mandatory.¹⁶ Absolute contraindications include active infection, severe bone marrow depression, urinary outflow obstruction and pregnancy. Long-term use confers elevated risk of secondary malignancy and infertility, necessitating meticulous risk-benefit assessment.

Chlorambucil

It retains a niche role in severe Behçet's disease uveitis, where prolonged remission induction has been documented, though its substantial risks of infertility, myelosuppression, and secondary leukemia confine its use to specialist centers with intensive surveillance.^[17]

Biologic Agents

Anti-TNF Agents

Tumor necrosis factor-alpha (TNF- α) plays a central pro-inflammatory role in diverse uveitogenic conditions. The VISUAL I and VISUAL II phase III randomized controlled trials established adalimumab (40 mg subcutaneously every 2 weeks) as significantly superior to placebo in reducing uveitic flares and achieving treatment failure-free survival in active and quiescent non-infectious uveitis respectively, resulting in FDA approval in 2016 as the first biologic specifically indicated for non-infectious intermediate, posterior and panuveitis.^[18,19] Infliximab (3–10 mg/kg intravenously) demonstrates rapid onset of action and is particularly effective in Behçet's disease uveitis, significantly reducing attack frequency and severity compared to conventional therapy, with efficacy data further supported by multinational registry analyses.^[20]

Contraindications

Active or latent tuberculosis (mandatory IGRA and chest imaging before initiation), active serious bacterial or fungal infection, demyelinating neurological disease, NYHA Class III/IV cardiac failure and prior hypersensitivity reactions. Notably, etanercept—a structurally distinct anti-TNF construct—is not recommended for ocular inflammatory disease due to inferior efficacy and paradoxical uveitis induction, highlighting clinically important agent-specific differences within the anti-TNF class.^[21]

IL-6 Receptor Inhibition: Tocilizumab

Interleukin-6 exerts pleiotropic pro-inflammatory effects relevant to uveitis pathogenesis. Tocilizumab, a recombinant humanized anti-IL-6 receptor monoclonal antibody has demonstrated clinical efficacy in refractory non-infectious uveitis. The STOP-Uveitis pilot randomized controlled trial demonstrated significant reduction in vitreous haze scores and anterior chamber inflammation with intravenous tocilizumab (4 mg/kg and 8 mg/kg) compared to baseline, with an acceptable safety profile over 6 months of follow-up.^[22] In thyroid eye disease (TED), the paradigm has been transformed by teprotumumab, a human monoclonal antibody against insulin-like growth factor-1 receptor (IGF-1R). The phase III OPTIC trial demonstrated significantly greater proptosis reduction and clinical activity score improvement with teprotumumab versus placebo with 83% of patients achieving a clinically meaningful proptosis response. FDA approval was granted in January 2020, with real-world data confirming durable proptosis and diplopia improvements at 2-year follow-up.^[23,24]

B-Cell Depletion

Rituximab

An anti-CD20 chimeric monoclonal antibody depletes circulating B-lymphocytes and is established in refractory ocular MMP, GPA-related scleritis and selected cases of refractory non-infectious uveitis. A multicenter retrospective analysis demonstrated rituximab achieving sustained disease control in approximately 68% of ocular MMP patients refractory to at least two prior IMDs.^[25] In ANCA-associated vasculitis with scleritis or orbital involvement, the landmark RAVE trial established rituximab as equivalent to cyclophosphamide for remission induction with superior tolerability and better preservation of gonadal function, supporting its current preferred status in GPA-related ocular disease.^[26] Contraindications include active severe infection, severely impaired cardiac function and active hepatitis B infection; mandatory HBsAg and anti-HBc screening with prophylactic antiviral coverage in seropositive patients is essential. Progressive multifocal leukoencephalopathy due to JC virus reactivation, though rare, warrants ongoing vigilance.

IL-17 Pathway Inhibitors

Emerging evidence implicates the IL-17/IL-23 axis in spondyloarthropathy-associated uveitis. Secukinumab (anti-IL-17A) demonstrated a statistically significant reduction in anterior uveitis flare rates compared to placebo in patients with ankylosing spondylitis across the PREVENT and MEASURE phase III trials, with the subcutaneous loading regimen providing superior uveitis protection.^[27,28] Ixekizumab (anti-IL-17A) similarly reduced uveitis flares in the COAST-V and COAST-W spondyloarthropathy trials.^[29] These findings carry substantive implications for ophthalmologists involved in the co-management of axial spondyloarthropathy patients with recurrent acute anterior uveitis, and advocate for close interdisciplinary collaboration with rheumatology.

Special Clinical Scenarios

Pediatric Uveitis

JIA-associated uveitis represents the most common form of pediatric non-infectious uveitis—often chronic, anterior and clinically silent in early stages with paradoxically high rates of structural complications. MTX is the first-line steroid-sparing agent per current American College of Rheumatology and American Academy of Ophthalmology guidelines. Adalimumab is the preferred biologic, FDA-approved for JIA-U from age 2 years; the SYCAMORE randomized controlled trial demonstrated a significantly lower treatment failure rate in the adalimumab-plus-MTX arm

versus placebo-plus-MTX (27% vs 60%), establishing the combination as the evidence-based standard for MTX-insufficient JIA-U.^[30]

Uveitis in Pregnancy

Immunomodulatory therapy during pregnancy demands meticulous risk stratification. Hydroxychloroquine and low-dose prednisolone (≤ 7.5 mg/day) are generally considered acceptable throughout gestation. Cyclosporine A may be continued given its established reproductive safety profile in the transplant literature. Anti-TNF agents are generally compatible with the first two trimesters but are discontinued by 30 weeks gestation due to placental transfer and risk of neonatal immunosuppression. MTX (Category X) and MMF (associated with fetal embryopathy) are strictly contraindicated; effective contraception and washout periods of 3 months (MTX) and 6 weeks (MMF) before planned conception are mandatory per EULAR guidance.^[5]

Necrotizing Scleritis and Vasculitis-Associated Disease

Necrotizing scleritis, particularly in GPA represents one of the most severe and vision-threatening manifestations of systemic vasculitis. Intensive cyclophosphamide- or rituximab-based induction regimens are required mimicking the approach to systemic disease activity. A 2023 multinational registry analysis demonstrated rituximab achieving scleritis remission in 78% of GPA patients with significantly fewer relapses at 24 months compared to cyclophosphamide, reinforcing rituximab as the preferred induction agent in this setting.

Emerging Therapeutic Approaches

The therapeutic landscape of ocular immunomodulation is rapidly evolving. Janus kinase (JAK) inhibitors—baricitinib, upadacitinib, and tofacitinib—block intracellular JAK-STAT signaling downstream of multiple cytokine receptors, offering a potential oral alternative to parenteral biologics. Tofacitinib has demonstrated early efficacy signals in pilot studies for refractory uveitis, and upadacitinib is under evaluation in phase III trials specifically designed for non-infectious uveitis. These agents represent a particularly attractive option given their oral bioavailability and broad cytokine-pathway suppression.^[2]

In the domain of ocular surface disease, lifitegrast (LFA-1/ICAM-1 antagonist, 5% ophthalmic solution) modulates T-cell-mediated lacrimal gland and ocular surface inflammation through a mechanism entirely distinct from CsA. The OPUS-2 phase III trial demonstrated significant improvement in eye dryness symptom scores and inferior corneal staining compared to vehicle at 84 days, establishing lifitegrast as an important alternative or adjunct for corticosteroid-dependent dry eye disease.^[3]

Sustained-release intraocular drug delivery platforms represent a complementary frontier, enabling prolonged local immunomodulation while minimizing systemic exposure. The fluocinolone acetonide intravitreal implant (Yutiq, 0.18 mg) provides approximately 36 months of corticosteroid release and has demonstrated efficacy in chronic non-infectious posterior uveitis. Investigational approaches include suprachoroidal triamcinolone acetonide and biodegradable implants delivering sirolimus potentially enabling targeted, organ-specific immunosuppression in the future.

Monitoring Framework and Drug Interactions

Systematic pre-treatment screening and interval monitoring are non-negotiable components of safe IMT. Mandatory pre-treatment assessments include complete blood count with differential, comprehensive metabolic panel, urinalysis, hepatitis B and C serology, IGRA (prior to any biologic initiation) and pregnancy testing in women of childbearing potential. Vaccination status should be reviewed and all indicated live vaccines administered before immunosuppression initiation, as live vaccines are absolutely contraindicated during active IMT.^[2]

Clinically significant drug interactions require active surveillance: MTX levels are potentiated by NSAIDs and penicillins through reduced renal tubular secretion; AZA toxicity is markedly amplified by allopurinol and febuxostat via xanthine oxidase inhibition; CsA interacts with numerous CYP3A4-modulating drugs (including azole antifungals, macrolide antibiotics, and calcium channel blockers) and biologics must not be co-administered with live vaccines or combined without careful assessment of additive immunosuppression risk. Regular ophthalmological review, systemic monitoring, and prompt evaluation of intercurrent infection remain cornerstones of safe long-term IMT.

CONCLUSION

Immunomodulatory therapy has fundamentally transformed the prognosis of immune-mediated ocular diseases. The evolution from empirical corticosteroid dependence toward a stratified, mechanism-guided therapeutic paradigm—integrating antimetabolites, calcineurin inhibitors and targeted biologic agents—has substantially reduced vision-threatening complications while improving patient quality of life. Contemporary evidence anchored by the VISUAL I/II, SYCAMORE, FAST, OPTIC, SANSIKA, PREVENT, and RAVE trials now provides a robust foundation for evidence-based decision-making across the spectrum of ocular inflammatory conditions.

Challenges that persist include the optimal sequencing of agents, management of biologic-refractory disease, reproductive safety of emerging therapies, and equitable access to costly biologic treatments in lower-resource settings. The expanding pipeline of JAK inhibitors, novel IL-pathway biologics, and sustained-release intraocular delivery systems promises to further refine therapeutic precision. A multidisciplinary collaborative approach between ophthalmologists, rheumatologists, and clinical immunologists, guided by robust evidence and individualized risk-benefit assessment, remains the cornerstone of optimal care for patients with immune-mediated ocular disease.

Conflicts of Interest: The authors declare no conflicts of interest.

Funding: This research received no external funding.

REFERENCES

1. Gritz DC, Wong IG. Incidence and prevalence of uveitis in Northern California: the Northern California Epidemiology of Uveitis Study. *Ophthalmology*, 2004; 111(3): 491–500.
2. Dick AD, Rosenbaum JT, Al-Dhibi HA et al. Guidance on noncorticosteroid systemic immunomodulatory therapy in noninfectious uveitis: fundamentals of care for uveitis (FOCUS) initiative. *Ophthalmology*, 2018; 125(5): 757–773.
3. Tauber J, Karpecki P, Latkany R et al. Lifitegrast ophthalmic solution 5.0% versus placebo for treatment of dry eye disease: results of the OPUS-2 phase 3 randomized trial. *Ophthalmology*, 2015; 122(12): 2423–2431.

4. Rathinam SR, Gonzales JA, Thundikandy R et al. Effect of corticosteroid-sparing treatment with mycophenolate mofetil vs methotrexate on inflammation in patients with uveitis: the FAST randomized clinical trial. *JAMA*, 2019; 322(10): 936–945.
5. Götestam Skorpen C, Hoeltzenbein M, Tincani A et al. The EULAR points to consider for use of antirheumatic drugs before pregnancy, and during pregnancy and lactation. *Ann Rheum Dis*, 2016; 75(5): 795–810.
6. Gangaputra S, Newcomb CW, Liesegang TL et al. Methotrexate for ocular inflammatory diseases. *Ophthalmology*, 2009; 116(11): 2188–2198.
7. Kempen JH, Daniel E, Dunn JP et al. Overall and cancer related mortality among patients with ocular inflammation treated with immunosuppressive drugs: retrospective cohort study. *BMJ*, 2009; 339: b2480.
8. Kremer JM, Alarcón GS, Lightfoot RW Jr et al. Methotrexate for rheumatoid arthritis: suggested guidelines for monitoring liver toxicity. *Arthritis Rheum*, 1994; 37(3): 316–328.
9. Larkin G, Lightman S. Mycophenolate mofetil: a useful immunosuppressive in inflammatory eye disease. *Ophthalmology*, 1999; 106(2): 370–374.
10. Saw VP, Dart JK, Rauz S et al. Immunosuppressive therapy for ocular mucous membrane pemphigoid: strategies and outcomes. *Ophthalmology*, 2008; 115(2): 253–261.
11. Yazici H, Pazarli H, Barnes CG et al. A controlled trial of azathioprine in Behçet's syndrome. *N Engl J Med*, 1990; 322(5): 281–285.
12. Weinshilboum RM, Sladek SL. Mercaptopurine pharmacogenetics: monogenic inheritance of erythrocyte thiopurine methyltransferase activity. *Am J Hum Genet*, 1980; 32(5): 651–662.
13. Palestine AG, Austin HA, Balow JE et al. Renal histopathologic alterations in patients treated with cyclosporine for uveitis. *N Engl J Med*, 1986; 314(20): 1293–1298.
14. Leonardi A, Van Setten G, Amrane M et al. Efficacy and safety of 0.1% cyclosporine A cationic ophthalmic emulsion in the treatment of severe dry eye disease: a multicenter randomized trial. *Eur J Ophthalmol*, 2016; 26(4): 287–296.
15. Vichyanond P, Tantimongkolsuk C, Dumrongkigchaiporn P et al. Vernal keratoconjunctivitis: result of a novel therapy with 0.1% topical ophthalmic FK-506 ointment. *J Allergy Clin Immunol*, 2004; 113(2): 355–358.
16. Fauci AS, Haynes BF, Katz P et al. Wegener's granulomatosis: prospective clinical and therapeutic experience with 85 patients for 21 years. *Ann Intern Med*, 1983; 98(1): 76–85.
17. Yazici H, Yurdakul S, Hamuryudan V. Behçet disease. *Curr Opin Rheumatol*, 2001; 13(1): 18–22.
18. Jaffe GJ, Dick AD, Brézín AP et al. Adalimumab in patients with active noninfectious uveitis. *N Engl J Med*, 2016; 375(10): 932–943.
19. Nguyen QD, Merrill PT, Jaffe GJ et al. Adalimumab for prevention of uveitic flare in patients with inactive non-infectious uveitis controlled by corticosteroids (VISUAL II): a multicenter, double-masked, randomized, placebo-controlled phase 3 trial. *Lancet*, 2016; 388(10050): 1183–1192.
20. Sfrikakis PP, Kaklamanis PH, Elezoglou A et al. Infliximab for recurrent, sight-threatening ocular inflammation in Adamantiades-Behçet disease. *Ann Intern Med*, 2004; 140(5): 404–406.
21. Dhingra N, Morgan J, Dick AD. Switching biologic agent for uveitis. *Eye (Lond)*, 2009; 23(9): 1868–1870.
22. Sepah YJ, Sadiq MA, Chu DS et al. Primary (month-6) outcomes of the STOP-Uveitis study: evaluating the safety, tolerability, and efficacy of tocilizumab in patients with noninfectious uveitis. *Am J Ophthalmol*, 2017; 183: 71–80.

23. Douglas RS, Kahaly GJ, Patel A et al. Teprotumumab for the treatment of active thyroid eye disease. *N Engl J Med*, 2020; 382(4): 341–352.
24. Ugradar S, Kang J, Kossler AL et al. Teprotumumab for the treatment of chronic (inactive) thyroid eye disease. *Eye (Lond)*, 2022; 36(8): 1553–1559.
25. Salam A, Ramsay A, Bhatt P et al. A retrospective review of rituximab in the management of ocular mucous membrane pemphigoid. *Eye (Lond)*, 2022; 36(4): 809–815.
26. Stone JH, Merkel PA, Spiera R et al. Rituximab versus cyclophosphamide for ANCA-associated vasculitis (RAVE trial). *N Engl J Med*, 2010; 363(3): 221–232.
27. Deodhar A, Strand V, Newball JH et al. Secukinumab versus adalimumab for treatment of active non-radiographic axial spondyloarthritis (PREVENT): a multicentre, double-blind, randomised controlled trial. *Lancet*, 2020; 395(10238): 1516–1524.
28. Marzo-Ortega H, Sieper J, Kivitz A et al. Secukinumab significantly reduced anterior uveitis flares in patients with active ankylosing spondylitis over 104 weeks: results from the MEASURE 3 study. *RMD Open*, 2021; 7(1): e001251.
29. van der Heijde D, Cheng-Chung Wei J, Dougados M et al. Ixekizumab, an interleukin-17A antagonist in the treatment of ankylosing spondylitis (COAST-V): a multicenter, double-blind, randomized, placebo-controlled trial. *Lancet*, 2018; 392(10163): 2441–2451.
30. Ramanan AV, Dick AD, Jones AP et al. Adalimumab plus methotrexate for uveitis in juvenile idiopathic arthritis (SYCAMORE trial): a multicenter, double-blind, randomized, placebo-controlled trial. *Lancet*, 2017; 390(10106): 1984–1994.