Uveitis-Associated Rheumatic Disease: For Non-Ophthalmologists

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Abstract

Uveitis is a leading cause of vision loss worldwide. Its etiology is classified into infectious, non-infectious, and idiopathic categories. Among non-infectious cases, uveitis associated with rheumatic diseases is common. Effective management of uveitis and identification of underlying rheumatic conditions are crucial. Therefore, interdisciplinary collaboration between ophthalmologists and internists or rheumatologists is essential. To achieve this, non-ophthalmologists need a solid understanding of uveitis, including ocular anatomy, classification, clinical features, rheumatic disease associations, and current treatment strategies. These aspects will be addressed in this review.

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Introduction

The eye is composed of three main layers and three chambers. The uvea, part of the middle layer, includes the iris, ciliary body, and choroid, as illustrated in Figure 1.[1-3]

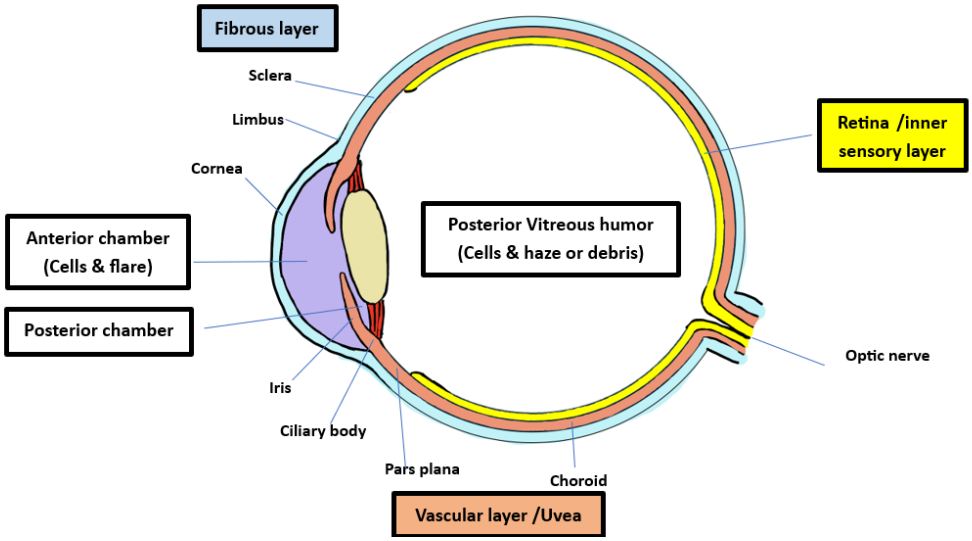

Figure 1 Sagittal section of the eye showing the anatomical structure of its three layers and chambers.
Adapted from Riordan-Eva P. Anatomy & Embryology of the Eye. In: Riordan-Eva P, Augsburger JJ, editors. Vaughan & Asbury's General Ophthalmology, 19e. New York, NY: McGraw-Hill Education 2017[1,4] Hochberg MC, Gravallese EM, Smolen JS, Van der Heijde D, Weinblatt ME, Weisman MH. Rheumatology: Elsevier.; 2023[2]

The outer layer or fibrous layer, includes the sclera, a tough connective tissue that maintains the eye's shape, and the cornea, a transparent, curved surface that refracts light. The middle layer or uvea, consists of the iris, which regulates pupil size; the ciliary body, which produces aqueous humor and adjusts lens focus; and the choroid, a vascular layer that nourishes the retina.[5] The inner layer, the retina, contains photoreceptors that convert light into electrical signals transmitted to the brain via the optic nerve. The eye also contains three chambers. The anterior and posterior chambers, located in front of the lens, are filled with aqueous humor, a clear fluid that maintains intraocular pressure and nourishes surrounding tissues. Inflammatory conditions can disrupt its clarity. The vitreous chamber, located behind the lens and surrounded by the retina, contains the vitreous body, a gel-like substance that helps maintain the eye's shape and supports the retina.[6]

Ocular Immune Response

The body normally mounts an immune response to eliminate foreign antigens, but some organs—known as immune-privileged sites—suppress this response to prevent tissue damage. These sites include the eye, brain, spinal cord, testes, placenta, and fetus, which possess unique anatomical and physiological features that tightly regulate immune activity. The eye is a key example because inflammation could impair vision. Its immune privilege is maintained through several mechanisms: the internal ocular structures lack lymphatic vessels, avascular structures like the cornea and lens that restrict immune cell access, immunosuppressive molecules in the aqueous humor (such as transforming growth factor-β and α-melanocyte-stimulating hormone) help dampen immune responses, and ocular tissues express apoptosis-inducing ligands like Fas ligand and TRAIL (TNF-related apoptosis-inducing ligand) that eliminate

activated immune cells. Additionally, the eye demonstrates anterior chamber–associated immune deviation (ACAID), where antigens trigger a regulatory rather than inflammatory immune response. This suppresses T-cell and B-cell activation, promoting immune tolerance and minimizing tissue damage. This immune privilege explains why corneal transplantation often succeeds without systemic immunosuppression, although failure of these protective mechanisms or when autoimmune responses target ocular tissues, immune-mediated inflammation such as uveitis can occur.[6-8] Uveitis is a significant cause of visual impairment globally.[9] It can be idiopathic, infectious, or associated with systemic, particularly rheumatic conditions. For non-ophthalmologists, recognizing and managing uveitis, as well as, identifying rheumatic disease is crucial for preventing vision loss and systemic complications.

Epidemiology

The prevalence of uveitis varies globally, and is influenced by geographic, genetic, environmental, socioeconomic, and healthcare-related factors. In the United States, estimates range from 200 to 234 cases per 100,000 people.[10-12] In contrast, prevalence rates can be significantly higher in developing countries and parts of Asia, such as India, where they may reach up to 730 cases per 100,000 population.[13-15] Uveitis is a major cause of visual impairment, accounting for approximately 10% of blindness in the United States and up to 20–25% of blindness worldwide.[10-12] It is the fifth leading cause of vision loss among working-age adults in developed countries.[16]

In the Asia-Pacific region, the prevalence of non-infectious and idiopathic uveitis ranges from 11-83% and 10-65%, respectively.[15] Specific types of uveitis in this region included HLA-B27-associated uveitis (0-45%), Behcet's disease (0-28%), Vogt-Koyanagi-Harada (VKH) syndrome (0-21%), and sarcoidosis (0-11%).[15]

In Thailand, the prevalence of non-infectious and idiopathic uveitis ranges from 38-44% and 13-48%, respectively and uveitis associated with systemic disease accounts for approximately 24-36% of all cases.[15, 17-20] Common associated rheumatic conditions include Juvenile Idiopathic Arthritis (JIA) with a prevalence of 12.4–12.8%[17, 21]; Behçet's Disease (7.1–15.4%[20, 21]); VKH Syndrome (6.8–13.5%[17, 20, 21]); HLA-B27/Spondyloarthritis (SpA)-Associated Uveitis (5.2-12.4%[17-20, 22]); sarcoidosis (0-9%)[17-20, 22]; and Systemic Lupus Erythematosus (SLE) (1.4%[21]).

Classification of Uveitis

Uveitis is classified using the Standardization of Uveitis Nomenclature (SUN) system[23] based on the anatomical site of inflammation, which includes anterior, intermediate, posterior, or panuveitis.

It is also categorized by disease onset, duration, and course. Onset can be sudden (abrupt symptoms) or insidious (gradual development), while duration is limited if it lasts up to three months or persistent if it lasts longer. The course of disease includes acute uveitis (sudden and resolves within three months), recurrent (repeated episodes of inflammation, separated by inactive periods without treatment for more than three months), or chronic (persistent inflammation with relapse within three months after discontinuing treatment). Clinically, uveitis is further classified as granulomatous or nongranulomatous based on slit-lamp findings. [1,5] Granulomatous inflammation shows large, clumped keratic precipitates (deposits of inflammatory cells) typically located on the inferior corneal endothelium, In contrast, nongranulomatous inflammation presents with small, diffusedly distributed keratic precipitates. Uveitis may also extend to adjacent ocular structures, such as cornea (keratouveitis) and often involves the vitreous humor, retina, or optic nerve. A hallmark feature of uveitis is the presence of inflammatory cells in the aqueous and/or vitreous humor, often accompanied by retinal vasculitis or macular edema, both of which can significantly impair visual function

Symptoms and Signs of Uveitis According to Anatomical Location

Anterior uveitis involves inflammation of the iris and/or ciliary body. Symptoms may range from asymptomatic to a sudden onset of ocular redness, pain, and photophobia, with pain often caused by ciliary muscle spasm or increased intraocular pressure. Slit-lamp examination typically shows inflammatory cells and aqueous flare in the anterior chamber, and the severity of inflammation is graded according to the SUN classification system. In severe cases, hypopyon may develop.[1, 2, 5, 23]

Intermediate uveitis primarily affects the vitreous, posterior chamber or posterior portion of the ciliary body and peripheral retina. It commonly presents with floaters and blurred vision due to inflammatory cells in vitreous or macular edema. On examination reveals vitreous cells and vitreous haze, which are graded based on the degree of fundus visibility. [1, 2, 5, 23]

Posterior uveitis involves inflammation of the retina and choroid and usually causes painless blurred vision, floaters, flashes of light (photopsias), scotomas, distorted vision (metamorphopsia), and poor night vision. On ophthalmic examination may reveal, retinal or choroidal inflammatory infiltrates, vascular sheathing, hemorrhages, retinal detachment, retinal pigment epithelium changes, optic nerve involvement. [1,2, 5, 23]

Causes of Uveitis

Uveitis can be categorized into three main groups: infectious, non-infectious, and idiopathic[9, 24-27]

1. **Infectious Causes (11-50%)** These include bacteria, viruses, parasites, and fungal infections
2. **Non-infectious Causes (58-79%)**
   - **Medication-associated uveitis (1%):** Drugs linked to anterior uveitis include cidofovir, prostaglandin analogues, bisphosphonates, fluoroquinolones, sulfonamides. Intermediate uveitis may be triggered by anti-vascular endothelial growth factor agents. Uveitis at any location has been reported with rifabutin, protein kinase inhibitors, immune checkpoint inhibitors, and certain vaccines.
   - **Systemic disease-associated uveitis:** These include HLA-B27-associated uveitis and spondyloarthritis, Behçet's disease, sarcoidosis, juvenile idiopathic arthritis, Vogt-Koyanagi-Harada disease, systemic lupus erythematosus, systemic vasculitis (e.g., Kawasaki disease, polyarteritis nodosa, granulomatosis with polyangiitis, giant cell arteritis), inflammatory bowel disease (IBD), IgG4-related disease, multiple sclerosis, etc.
   - **Local ocular disease-associated uveitis:** Uveitis can arise as the sole inflammatory site as a response to trauma, local inflammation/infection or reaction to intraocular malignancies. These include posttraumatic uveitis, Fuchs heterochromic iridocyclitis, primary ocular lymphoma, white dot syndromes**,** and sympathetic ophthalmia—a bilateral diffuse granulomatous uveitis following trauma or surgery to one eye.
3. **Idiopathic (27-51%)**

Uveitis and Rheumatic Diseases

Uveitis is a recognized manifestation of various rheumatic diseases. The eye and joint tissues share structural similarities, including the presence of hyaluronic acid, type II collagen, and aggrecan—a proteoglycan found in both tissues. These shared components may explain why patients often experience symptoms of arthritis and uveitis concurrently (mimicry).[28]

1. HLA-B27/Spondyloarthritis-Associated Uveitis

HLA-B27 positivity is found in about 33–67%[29] of patients with acute anterior uveitis (AAU). This form of uveitis typically affects young adults aged 20–40 years, with a male predominance. It usually presents with a sudden onset of unilateral anterior uveitis with severe inflammation, sometimes accompanied by hypopyon. The condition is often recurrent, with episodes lasting 4–6 weeks and sometimes alternating between eyes. If untreated, complications such as posterior synechiae, secondary glaucoma, vitritis, vasculitis, papillitis, and macular edema may develop. [5, 30-32] In contrast, HLA-B27–negative uveitis is more often bilateral, chronic, and less recurrent, and is less commonly associated with systemic rheumatic diseases. HLA-B27-associated AAU is strongly linked to spondyloarthritis (SpA), with prevalence rates of 50–65% among affected individuals.[30] Among patients with AAU without a prior diagnosis of SpA, newly diagnosed SpA accounted for approximately 47% with axial spondyloarthritis comprising 50% and peripheral spondyloarthritis 18%.[33]

1.1 Axial Spondyloarthritis

Radiographic axial spondyloarthritis, also known as ankylosing spondylitis (AS), commonly presents with acute anterior uveitis and is strongly associated with HLA-B27 positively. Uveitis occurs in approximately 23–40%[34, 35]of patients with AS and in 15.9% of patients with non-radiographic axial spondyloarthritis.[35] Risk factors for developing AAU include longer disease duration, male gender, and HLA-B27 positivity.[35] A Thai study reported a uveitis prevalence of 24% among patients with axial spondyloarthritis, with associations to HLA-B27 positively and delayed diagnosis of the axial spondyloarthritis.[36]

1.2 Reactive Arthritis (formerly Reiter's Syndrome)

Symptoms typically develop 2-4 weeks following infections with organisms such as *Ureaplasma urealyticum*, *Chlamydia*, *Shigella*, *Salmonella*, or *Yersinia*.[37] The most common ocular manifestation is conjunctivitis.[37] AAU is uncommon in the early stages (5–20%) but may occur in up to 50% of patients in later stages. Uveitis symptoms are similar to those seen in AS.[5]

1.3 Psoriatic Arthritis

Ocular findings include anterior uveitis in approximately 5 % of cases.[38] Clinical presentations vary, with reports of both predominantly unilateral[38] and bilateral uveitis[39], and may include posterior segment involvement**.** Risk factors for uveitis in psoriatic arthritis include HLA-B27 positivity, sacroiliitis on MRI, and poorer physical function.[38]

1.4 Inflammatory Bowel Disease (IBD)

Approximately 5% of patients with chronic IBD develop uveitis, with a female predominance (82%). HLA-B27 positivity is reported in about 46% of cases. Uveitis in IBD tends to be bilateral, predominantly affecting the posterior segment, with an insidious onset and chronic course. However, some patients may also experience acute, recurrent unilateral anterior uveitis.[40]

2. Sarcoidosis

Ocular involvement and uveitis in sarcoidosis are more commonly observed in individuals aged 50–65 years.[44] Sarcoidosis can affect any part of the eye, including the orbit and adnexa, and may present as granulomas on the eyelids or conjunctiva, or as lacrimal gland infiltration. Uveitis is a common manifestation, with anterior uveitis being the most common, occurring in approximately 71% of cases.[44] There are four distinct patterns of ocular sarcoidosis:[44] (1) acute anterior uveitis; (2) chronic granulomatous anterior uveitis, which is characterized by mutton-fat keratic precipitates, Koeppe nodules at the pupillary margin, Busacca nodules on the iris; and vitritis with white cell clumps in the anterior vitreous; (3) posterior uveitis, seen in about 25% of cases, often involves retinal vasculitis and presents with yellowish-gray nodular lesions along the choroid or retina, along with "snowball" opacities in the vitreous; and (4) panuveitis, which accounts for 6–33% of cases and is associated with a poorer visual prognosis.[41]

3. Behçet's Disease

Uveitis in Behçet's disease is typically bilateral and nongranulomatous, most commonly presenting as posterior uveitis or panuveitis, which affect approximately 50%–80% of patients.[45] The inflammation can significantly impair vision, and recurrent episodes may lead to incomplete visual

recovery or even blindness due to optic nerve ischemia. Common ocular signs include multifocal chalky-white retinal infiltrates, retinal hemorrhages, vitritis, artery and venous occlusions, macular edema, and retinal or optic nerve atrophy. Anterior uveitis and hypopyon may also occur during acute attacks.[45] Hypopyon in Behcet's disease is typically non-fibrinous and mobile, often shifting position rapidly in response to gravity.

4. Antinuclear Antibody-Associated Disease

These autoimmune disorders including SLE, mixed connective tissue disease, scleroderma, and Sjögren's syndrome, more commonly cause ocular manifestations such as keratoconjunctivitis sicca, retinal vasculitis, or optic neuritis than uveitis, with uveitis occurring in approximately 0.1–4.8% of cases[5,46]

When present, uveitis is more often posterior uveitis associated with retinal vasculitis and optic neuritis rather than isolated anterior uveitis. In patients with SLE, autoantibodies may also trigger lupus choroidopathy (choroiditis), which is considered part of posterior uveitis. Panuveitis and choroiditis are rare but indicate severe disease and may reflect active systemic vasculitis.

5. ANCA-Associated Vasculitis (AAV)

Ocular manifestations occur in approximately 20-60% of patients with AAV, with the highest prevalence seen in Granulomatosis with Polyangiitis (up to 36-50%), followed by Eosinophilic Granulomatosis with Polyangiitis (around 18%) and Microscopic Polyangiitis (about 7%)[47] Common ocular features include diffuse anterior scleritis—often necrotizing—and peripheral ulcerative keratitis. Uveitis, which may be anterior, intermediate, or posterior, occurs in approximately 2.8-11% of cases.[5, 48]

Strategy for Diagnosing Rheumatic Diseases Associated with Uveitis

There is no universal international consensus on the optimal diagnostic approach for uveitis, but several accepted strategies exist to identify systemic diseases associated with uveitis. One widely recommended method is the standardized diagnostic approach from the ULISSE study[49], which showed that a standardized diagnostic approach is more cost-effective and clinically efficient than open-ended strategy. This approach start with the initial investigations include complete blood count, erythrocyte sedimentation rate, C-reactive protein, tuberculin skin test, syphilis serology, and chest radiograph, followed by additional tests guided by the anatomic type of uveitis, clinical findings, and patient history.

The optimal diagnostic approach for rheumatic diseases associated with non-infectious uveitis should depend on the clinical presentation and patient context. For internists, after excluding medication-associated uveitis, a systematic clinical review and physical examination for common rheumatic diseases (see Table 1) should be performed to identify evidence of a specific rheumatic disease. Investigations should be conducted based on clinical indications.

If no evidence is found through clinical review, the diagnostic work-up should be guided by the anatomic type, onset, duration, and severity of uveitis (see Table 1).[25, 49, 50] Conversely, patients with systemic rheumatologic diseases should also be screened for ocular involvement, including uveitis. If there is evidence from history or physical examination, referral to an ophthalmologist is recommended for further evaluation.

Table 1 Common Clinical Manifestations of Rheumatic Diseases Associated with uveitis

Type	Characteristic features	Rheumatic diseases	History taking with physical examination	Suggested Investigations*
Acute Anterior uveitis	Unilateral recurrent or bilateral alternating	AS (40%)	IBP, arthritis, OTW, TTW, Chest expansion, Schober test	Pelvic radiograph, HLA-B27, and other investigation according to manifestation

		Reactive arthritis (5-20%)	Hx GI,GU infection, arthritis, rash†	
		PsA (5-10%)	Psoriasis, psoriasis nail, arthritis, family history PsA	
		IBD-associated arthritis (2-12%)	chronic diarrhea, arthritis	
	Symmetrical bilateral involvement with/without hypopyon	Behçet's Disease	Recurrent oral/genital ulcer, EN, thrombosis	Pathergy test, and other investigation according to manifestation
	Unilateral/bilateral granulomatous (mutton-fat, keratic precipitates)	Sarcoidosis (most common)	Fever, arthritis, EN, hilar lymphadenopathy	CXR, serum ACE, histology, and other investigation according to manifestation
	Non specific	SLE	Fever, malar rash, DLE, arthritis, LN, AIHA	CBC, UA, CXR, ANA, and other investigation according to manifestation
Chronic Anterior uveitis	Bilateral non-granulomatous	JIA	Children, arthritis, unilateral uveitis (uncommon)	ANA, RF
		Behcet's disease	See Behcet's disease above	See Behcet's disease above
	Granulomatous	Sarcoidosis	See sarcoidosis above	See sarcoidosis above
		VKH	Headache, tinnitus, neck stiffness, hearing loss, poliosis, vitiligo, alopecia, recurrent uveitis up to 50-70%	Lumbar puncture and spinal fluid investigation and other investigation according to manifestation
Intermediate uveitis		Sarcoidosis	See sarcoidosis above	See sarcoidosis above
		Behçet's Disease	See Behcet's disease above	See Behcet's disease above
Posterior uveitis	Acute non-granulomatous	Behcet' disease	See Behcet's disease above, retinal vasculitis	See Behcet's disease above
		HLA-B27-associated uveitis (rare)	See AS, PsA, and IBD-associated arthritis above	See AS, PsA, and IBD-associated arthritis above
	Chronic bilateral non-granulomatous	Behcet' disease	See Behcet's disease above, retinal vasculitis	See Behcet's disease above
		VKH	See VKH above	See VKH above
		Sarcoidosis	See sarcoidosis above	See sarcoidosis above
	Acute/chronic granulomatous	Sarcoidosis (less frequent)	See sarcoidosis above	See sarcoidosis above
		VKH	See VKH above	See VKH above
Panuveitis	Acute/chronic bilateral non-granulomatous	Behcet's disease	See Behcet's disease above, retinal vasculitis	See Behcet's disease above

	Acute / chronic bilateral granulomatous	VKH	See VKH above	See VKH above
		Sarcoidosis	See sarcoidosis above	See sarcoidosis above

*Investigation should be guided by clinical manifestation. If no signs suggest a rheumatic disease, further investigations should be performed based on the following factors: anatomical location of uveitis, granulomatous vs. non-granulomatous nature, onset, and duration.

Abbreviation: ACE, angiotensin-converting enzyme; AIHA, autoimmune hemolytic anemia; ANA, anti-nuclear antibodies; AS, Ankylosing spondylitis; CBC, complete blood count; CRP, C-reactive protein; CXR, chest X-RAY; DLE, discoid lupus erythematosus; ESR, erythrocyte sedimentation rate; EN, erythema nodosum; HLA , human leucocyte antigen; IBD, Inflammatory bowel disease; JIA, Juvenile idiopathic arthritis; LN, lupus nephritis; OTW, occiput to wall; PsA, Psoriatic arthritis; SpA, spondyloarthritis; TTW, tragus to wall; †rash, keratoderma blennorrhagicum and/or circinate balanitis

Treatment of Uveitis-Associated Rheumatic Disease

The primary goals are to prevent vision loss by achieving remission and preventing relapse, to control both ocular and systemic inflammation, and to minimize treatment-related adverse effects. Treatment strategies are tailored based on disease severity, anatomical location, and associated systemic conditions. A fundamental principle is regular monitoring to assess disease activity and treatment side effects.[51]

Pharmacologic Management

1. **Corticosteroids** remain the cornerstone of initial therapy.
   - **For Non-sight-threatening disease,** a stepwise approach is recommended.
     1. Local corticosteroids are first-line for active, non-severe uveitis that does not significantly affect daily life. For anterior uveitis, topical corticosteroids such as 1% prednisolone acetate are typically used, with peak therapeutic effect within 3–7 days and full response by one month.[52] For unilateral intermediate, posterior, or panuveitis, local corticosteroid injections or implants are considered. Triamcinolone injections typically peaks within one month and lasts 3–6 months. Dexamethasone implants peak at two months and last up to six months, while fluocinolone acetonide implants, FDA-approved for chronic non-infectious posterior uveitis, can last up to 2.5 years.[53] The Multicenter Uveitis Steroid Treatment (MUST) Trial showed comparable visual outcomes between systemic corticosteroids and fluocinolone implants over five years, but by year seven, systemic therapy yielded better visual acuity and fewer ocular complications, including lower rates of glaucoma and elevated intraocular pressure.[54]
     2. Systemic corticosteroids are recommended for active, severe uveitis that affects daily life, bilateral intermediate, posterior, or panuveitis, and cases with incomplete response to local therapy, chronic or recurrent uveitis, or systemic manifestations (e.g., sarcoidosis, Behçet's disease).[55] Prednisolone is typically prescribed at 0.5–1 mg/kg/day, with high-dose therapy limited to one month to avoid complications. Follow-up within 1–2 weeks is essential to assess response. If no improvement is seen within 2–4 weeks, or if systemic corticosteroids are needed beyond three months, immunosuppressive agents should be considered.[41]
   - **For sight-threatening or severe inflammatory ocular disease**—such as Behçet's disease, VKH syndrome, optic nerve or macular involvement, or fulminant inflammation unresponsive to standard corticosteroids—requires aggressive treatment. Intravenous pulse methylprednisolone

500–1000 mg/day for 1–3 days,[56,57] are initiated, followed by rapid tapering of oral corticosteroids and escalation to systemic immunosuppressive therapy if needed.[56,57]

2. Immunosuppressive therapy

Indications are sight-threatening, bilateral, recurrent, or chronic uveitis; inadequate response to corticosteroids after 2–4 weeks; need for corticosteroid-sparing therapy; prevention of recurrence in high-risk conditions (e.g., HLA-B27-associated uveitis, Behçet's disease); and systemic rheumatic disease requiring immunosuppression.[55] Drug selection should be individualized based on uveitis severity, systemic comorbidities, and patient-specific factors.[41,55,58] (Table 2). All immunosuppressive agents carry an increased risk of infection, necessitating vigilant surveillance.

2.1 Antimetabolites, typically for less severe or corticosteroid-dependent uveitis

- Methotrexate, often the first-line agent for anterior, posterior, and panuveitis, is favored for its safety profile and lower risk of adverse effects. It has shown superior efficacy over mycophenolate mofetil and azathioprine in sarcoidosis with uveitis.[59]
- Azathioprine is effective in severe uveitis associated with Behcet's disease,[60] intermediate uveitis[61] and JIA-related iridocyclitis.
- Mycophenolate mofetil has demonstrated efficacy in posterior uveitis, scleritis, and JIA-associated uveitis unresponsive to methotrexate[62]

2.2 Alkylating Agents, reserved for severe, sight-threatening uveitis or when antimetabolites fail.

- Cyclophosphamide is effective in granulomatosis with polyangiitis, polyarteritis nodosa, chronic lupus-related, and Crohn's disease.[63]

2.3 Calcineurin Inhibitors (CNIs) are used in corticosteroid-resistant or severe uveitis.

- Cyclosporine is commonly used but contraindicated in Behçet's disease with neurological involvement due to potential central nervous system adverse effect from mild tremors and headaches to seizures, cortical blindness, and coma.[64]
- Tacrolimus has shown efficacy in intermediate and posterior uveitis and may offer a more favorable side effect profile compared to cyclosporine.[65]

3. Biologic therapy is considered in cases of refractory or relapsing non-infectious uveitis, where disease activity persists despite adequate trials of conventional immunosuppressants. Or cases with persistent inflammation or recurrent attacks.[4] All biologics are associated with an increased risk of infection; therefore, regular surveillance for infections is necessary.

3.1 Anti-tumor necrotic factors agents

- Adalimumab, a fully human monoclonal antibody, was FDA-approved in 2016–2017 for non-infectious intermediate, posterior, and panuveitis. The VISUAL I, II, and III trials demonstrated its efficacy in reducing treatment failure, preventing visual acuity deterioration and reducing relapse rates.[66,67] It also helped achieve remission and reduced corticosteroid dependence. Adalimumab has shown strong efficacy in JIA-associated chronic anterior uveitis (SYCAMORE study) and Behçet's disease-related ocular inflammation.[68]
- Infliximab, a chimeric monoclonal antibody, is approved in Japan for Behçet's disease with refractory uveoretinitis. Though not FDA-approved for uveitis, it is widely used off-label for JIA, sarcoidosis, VKH, and HLA-B27-associated uveitis. Meta-analyses show comparable efficacy to adalimumab,[69] with slightly higher adverse event rates[70]
- Golimumab, another fully human monoclonal antibody, has shown promise in reducing relapse rates (from 1.73 to 0.62 events per person-year), lowering corticosteroid doses, and decreasing the need for other immunosuppressive drugs in refractory panuveitis[71]
- Etanercept, a soluble TNF receptor fusion protein, has demonstrated inferior efficacy compared to other anti-TNF agents and is not recommended for active uveitis.[72]
- Combination therapy using an anti-metabolite and a biologic agent has been shown to be more effective than anti-metabolite monotherapy in achieving disease quiescence and corticosteroid-sparing success.[73]

3.5 Other Biologic Agents

- Rituximab, an anti-CD20 monoclonal antibody, is effective as a second or third-line agent in refractory NIU, particularly in Behçet's disease, JIA, and VKH.[74]
- Tocilizumab, a humanized monoclonal antibody targeting the IL-6 receptor, is effective and safe in treating severe, refractory uveitis, particularly in JIA cases that are unresponsive to anti-TNF agents.[75]

4. Janus kinase inhibitors—including tofacitinib, baricitinib, upadacitinib, and brepocitinib—are emerging treatment options for refractory non-infectious uveitis, particularly in immune-mediated inflammatory diseases such as JIA and spondyloarthritis. These agents are currently used off-label. Evidence from case reports, multicenter studies, and early-phase clinical trials has demonstrated rapid and sustained improvements in visual acuity and intraocular inflammation. For instance, a recent case series reported long-term remission in patients with isolated non-infectious uveitis treated with baricitinib and upadacitinib, with no serious adverse events over a mean follow-up of 31.6 months. Similarly, retrospective studies of tofacitinib and upadacitinib showed reduced flare frequency and good tolerability, with gastrointestinal upset being the most commonly reported side effect.[77]

Table 2 Summarizes Immunosuppressive Medications and Biological agents for Treating Non-infectious Uveitis, Including Dosages, Side effects, and Follow-up Monitoring.[56, 80-82]

Medication	Assess response	Dose & route	Side effects	Baseline /Monitor side-effect
Methotrexate	6-12 weeks	7.5–25 mg/wk.; PO or SQ; with folic acid ≥ 1 mg./day (practically in Thailand 5 mg/day)	Stomatitis, hair loss, nausea, neutropenia, infections, hepatotoxicity, interstitial pneumonitis, teratogenic effect	CBC, LFT, Cr, CXR, HBV, HCV/ CBC, LFT, Cr, CXR
Azathioprine	8-12 weeks	1–2.5 mg./kg./day; PO	Nausea, hepatotoxicity, marrow suppression	CBC, LFT, Cr, CXR, HBV, HCV / CBC, ALT, Cr
Mycophenolate Mofetil	6-12 weeks	1 g bid; PO	GI upset, diarrhea, leucopenia, teratogenic effect	CBC, LFT, Cr, CXR, HBV, HCV / CBC, ALT, Cr
Cyclophosphamide	4-12 weeks	1-2 mg./kg. PO/ 0.75–1g/ BSA IV q 4 weeks	Marrow suppression, hemorrhagic cystitis, malignancy risk if high cumulative dose, infertility, teratogenic effect	CBC, LFT, Cr, CXR, HBV, HCV, CXR, UA / CBC, ALT, Cr, UA
Cyclosporine A	4-12 weeks	2.5-5 mg./kg/d; PO	Nephrotoxicity, HT, hirsutism, gingival hyperplasia, electrolyte imbalance	BP, CBC, LFT, Cr, electrolytes, HBV, HCV / BP, CBC, ALT, Cr, blood levels (if using high dose)
Tacrolimus	6-12 weeks	0.1-0.15 mg./kg/d; PO	Nephrotoxicity, HT, hypomagnesemia, hyperkalemia, hyperglycemia, dyslipidemia, neurological symptoms (tremor, paresthesia),	BP, CBC, Cr, LFT, FBS, Chol, TG, serum magnesium, serum potassium / BP, Cr, ALT, FBS, blood levels (if using high dose)

Medication	Assess response	Dose & route	Side effects	Baseline /Monitor side-effect

Adalimumab*	4-12 weeks	80 mg. SC then 40 mg. q 2 weeks	injection site reaction, TB, lupus like reaction, demyelinating disorder	CBC, LFT, Cr, CXR, HBV, HCV, CXR, TST/IGRA / CBC, ALT, Cr, TB infection
Infliximab	2-12 weeks	3–5 mg./kg IV at week 0, 2, 6 then q 8 week	Infusion reaction, TB, lupus like reaction, demyelinating disorder	See Adalimumab
Golimumab	4-12 weeks	50 mg. SC monthly	See Adalimumab	See Adalimumab
Tocilizumab	8-12 weeks	4-8 mg/kg. IV q 4 weeks	Allergic reaction, intestinal perforation, autoimmune cytopenia, dyslipidemia	CBC, LFT, Cr, CXR, lipid profile, HBV, HCV / CBC, ALT, Cr, lipid profile
Rituximab	3-6 months	1,000 mg.IV q 2 weeks then repeat q 6 month	Infusion reactions, hepatitis B reactivation	CBC, LFT, Cr, CXR, HBV, HCV, CXR / CBC, ALT, Cr

BP= blood pressure, CBC = complete blood count; HBV, hepatitis B viral profile (HBs Ag, Anti-HBc Ab); HCV, Anti-HCV Ab; LFT = liver function test, PO = per oral, IM = intramuscular, SQ = subcutaneous, Cr = creatinine, CXR = chest X ray, HT = hypertension, PCP = Pneumocystis carinii pneumonia, BSA = body surface area, TST/IGRA = tuberculin skin test/ interferon gamma release assay , *(FDA approved for NIU and JIA)

Adapted from Shahab MA, Mir TA, Zafar S. Optimising drug therapy for non-infectious uveitis. International Ophthalmology.2019 Quartier P, Saadoun D, Belot A, Errera MH, Kaplanski G, Kodjikian L, et al. French recommendations for the management of non-infectious chronic uveitis. Rev Med Interne. 2023;44[5]227-52[83, 84]

Access and Reimbursement for Anti-TNF Agents for Uveitis Treatment in Thailand[77]

In Thailand, at present, adalimumab and infliximab are reimbursable under the Comptroller General's Department with pre-authorization, based on the following criteria:

- Eligible ocular conditions include Behçet's disease with ocular involvement, non-infectious necrotizing scleritis, ocular sarcoidosis, and Vogt-Koyanagi-Harada (VKH) disease in the acute phase.
- Indications for anti-TNF use require that patients have failed or been intolerant to at least two standard immunosuppressive agents, each administered for a minimum of three months at standard doses: methotrexate (0.3 mg/kg/week, max 25 mg/week), azathioprine (2–2.5 mg/kg/day), mycophenolate mofetil (2–3 g/day), cyclosporine A (3–5 mg/kg/day), cyclophosphamide (2 mg/kg/day), and chlorambucil (0.1 mg/kg/day).
- Dosage: Adalimumab is administered as an initial 80 mg subcutaneous injection, followed by 40 mg every two weeks. Infliximab is given intravenously at 5 mg/kg at weeks 0, 2, and 6, then every eight weeks.
- Treatment evaluation should be conducted every three months to assess efficacy and safety.
- Discontinuation criteria include lack of clinical response after six months, best-corrected visual acuity worse than finger counting at one foot, or an allergic reaction to the medication.

Adjunctive Treatment for Uveitis

Adjunctive therapies support primary anti-inflammatory strategies and help alleviate symptoms, prevent complications, and improve patient comfort.

1. **Mydriatic and cycloplegic agents** such as atropine, homatropine, cyclopentolate, and tropicamide—are used in anterior uveitis to relieve pain and photophobia by relaxing the ciliary muscle and prevent posterior synechiae formation[52, 78].

2. **Non-steroidal anti-inflammatory drugs (NSAIDs)**, although not first-line treatments for uveitis, may be beneficial in managing associated conditions such as non-necrotizing anterior scleritis or cystoid macular edema. Options include topical agents like nepafenac and ketorolac, as well as systemic NSAIDs.[52]
3. **Antiglaucoma medications**—including timolol, brimonidine, dorzolamide, and latanoprost—are employed to manage steroid-induced ocular hypertension or secondary glaucoma.[52]
4. **Osteoporosis prevention** is recommended for patients on long-term systemic corticosteroids. Supplementation with calcium and vitamin D, and possibly bisphosphonates, can help mitigate bone loss.[79]

Summary

Among uveitis-associated-rheumatic diseases, HLA-B27-related or spondyloarthritis-associated uveitis most commonly presents as anterior uveitis. Conversely, Behcet's disease, VKH syndrome and sarcoidosis can involve all chambers of the eye. Corticosteroids are the first-line treatment, with the route of administration depending on the location and severity of inflammation. Immunosuppressive medications are indicated for cases with inadequate or incomplete response to corticosteroids, for steroid-sparing purposes, and to prevent relapse. Biologic agents are used when there is insufficient response to immunosuppressive drugs. All these medications increase the risk of infection; therefore, close monitoring of adverse effects is essential to ensure safe and effective management.

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