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Review Article

Use of corticosteroids in treating non-infectious uveitis: an update

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ABSTRACT

Uveitis is characterized by the inflammation of the uveal tract, the vascular membrane of the ocular globe. Its etiologies are divided into two main categories: infectious and non-infectious. Corticosteroids are an important class of medications that have been used in the treatment of uveitis for decades. They can be administered either systemically (orally or intravenously) or locally (topical drops, periorbital injection, intravitreal injection, suprachoroidal injection, or intravitreal implant). This review updates and describes each of the currently available corticosteroid options for treating uveitis, along with their respective advantages and disadvantages. The main advantage of corticosteroids in treating uveitis is their ability to quickly and effectively control the inflammatory response. However, their use may result in various side effects, either local (cataract, elevated intraocular pressure) or systemic (osteonecrosis, adrenal insufficiency, Cushing syndrome). Overall, corticosteroids are not an appropriate option for long-term treatment of uveitis.

Key words: corticosteroid, uveitis, non-infectious, implant, suprachoroidal

INTRODUCTION

Uveitis is defined as inflammation of any component of the uveal tract: the iris, ciliary body, or choroid[1]. Clinical symptoms include ocular redness, eye pain, photophobia, decreased visual acuity, visual distortion, or floaters. Objective signs include ciliary flush, keratic precipitates, inflammatory cells, flare in the anterior chamber, lens exudation, posterior synechiae, vitreous cells and flare, snowballs and snowbank, choroidal lesions, and choroidal thickening[1,2].

Uveitis is typically classified based on the anatomical site of inflammation into anterior uveitis (involving the iris and ciliary body), intermediate uveitis (involving the pars plana of the ciliary body), posterior uveitis (involving the choroid), or panuveitis (involving all three structures). It may also be classified based on disease onset as acute uveitis (duration $\leq$ 3 months) or chronic uveitis (duration > 3 months). Etiologically, uveitis can be categorized as infectious, non-infectious, or malignant. Common infectious causes include *Toxoplasma gondii*, *Toxocara spp.*, *Mycobacterium tuberculosis*, *Treponema pallidum* (syphilis), and HIV. Malignancy-related causes must be ruled out, particularly in cases with initial onset in elderly patients over 60 years old or marked vitreous cells. The most common non-infectious causes are autoimmune in nature and may occur without associated systemic disease[2,3].

Uncontrolled uveitis can lead to various sight-threatening complications, including band keratopathy, secondary cataract, secondary glaucoma, cystoid macular edema, retinal vasculitis, and subretinal fluid accumulation[1–3].

THE ROLE OF CORTICOSTEROIDS

At present, corticosteroids remain the primary form of uveitis treatment. These steroid molecules are used to prevent or suppress inflammatory effects as well as perform immunosuppressive actions. They affect various cell types, including lymphocytes, macrophages, polymorphonuclear leukocytes, vascular endothelial cells, and fibroblasts. Corticosteroid molecules penetrate cell membranes and bind to soluble receptors in the cytosol. The resulting complexes then translocate to nuclear binding sites responsible for gene transcription, inducing or suppressing the transcription of certain mRNAs. These reactions lead to the downregulation of pro-inflammatory protein expression (prostaglandins, leukotrienes, and thromboxanes) by blocking enzymes that convert phospholipids into arachidonic acid, which is later converted into these proteins. Simultaneously, the expression of various cytokines is also downregulated. In addition, corticosteroids exert non-genomic, rapid actions independent of gene transcription regulation, including inhibition of prostaglandin E2 and blockade of T-cell receptor signaling[4–6].

Side effects of corticosteroids depend on their route of administration. While local administration may in-

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crease intraocular pressure (IOP) and cause cataracts as well as fibrosis of the oculomotor muscles and periorbital tissue, systemic administration can lead to serious complications such as hyperglycemia, hypertension, osteoporosis, Cushing syndrome, and adrenal insufficiency [5,6].

TRADITIONAL USES OF CORTICOSTEROID

Depending on the specific clinical presentation of uveitis, corticosteroids may be administered either systemically or locally. Examples of local administration include topical eye drops, subconjunctival injections, sub-Tenon or peribulbar injections, and intravitreal injections. Systemic routes typically involve oral or intravenous administration (Figure 1).

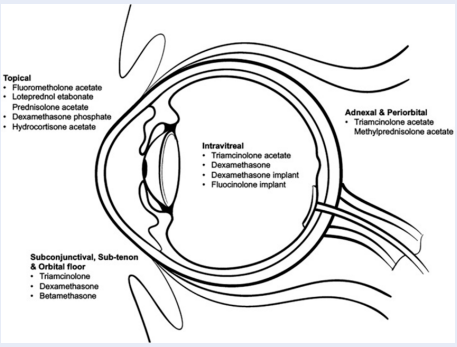

Figure 1: The possible local administrations of corticosteroids [7]

Topical eye drops

Topical corticosteroids have been the first-line treatment for anterior uveitis since the 1950s. The most commonly used topical corticosteroids are listed in Table 1. Topical corticosteroids are indicated exclusively for anterior uveitis, as they are generally ineffective for intermediate or posterior uveitis due to limited drug penetration into the posterior segment [8].

Different corticosteroids vary in potency and corneal penetration, which are the two key factors that determine clinical efficacy. These properties are governed by the molecular structure of the drug. Specifically, the fluorine atom enhances the binding affinity of corticosteroid molecules to their receptors [9] while the acetate group promotes corneal penetration. Loteprednol contains an ester group, while most others possess a ketone group, a structural feature that facilitates faster drug inactivation and results in a lower risk of IOP elevation [9].

Since corneal penetration is critical to therapeutic effectiveness, the clinical anti-inflammatory activity of topical corticosteroids may differ from their pharmacologic potency (Table 1). Fluorometholone and loteprednol, though associated with fewer side effects, have weaker anti-inflammatory effects, making them unsuitable for managing uveitis; instead, they are more appropriate for treating ocular surface inflammation. The most commonly used agents for anterior uveitis treatment are prednisolone acetate 1% and dexamethasone phosphate 0.1% [10].

In patients with active anterior uveitis, topical corticosteroids are typically initiated at a dosing frequency of one drop every 1–2 hours [11]. Once the inflammatory response is controlled (defined as resolution of anterior chamber cells or reduction to 1+), the dosage should be gradually tapered over 1–2 months [2]. IOP should be monitored weekly for the first two weeks, and patients should be monitored periodically thereafter, as delayed-onset complications may arise several months after the initiation of therapy [12].

In addition to secondary glaucoma and cataract formation, potential complications include corneal epithelial toxicity, corneal ulceration, crystalline keratopathy, delayed epithelial healing, and systemic absorption (particularly in pediatric patients) [8].

Periocular injections

Periocular injections are commonly indicated for unilateral intermediate or posterior uveitis, for patients who cannot undergo systemic therapy (e.g., pregnancy, lactation, or peptic ulceration), and for anterior uveitis with keratic precipitates or persistent anterior chamber cells that are unresponsive to topical treatment [12–14]. Commonly used agents include 0.5–1 mL of dexamethasone, triamcinolone, hydrocortisone, or betamethasone, which vary in formulation and their duration of action [15]. While the effects of dexamethasone phosphate last 1–2 days, triamcinolone acetonide effects may persist for more than 3 months (Table 2).

The mechanism by which corticosteroids penetrate intraocular tissues is not fully understood [16]; however, these drugs can persist in ocular tissues for several days, with the highest concentrations found in the choroid and retinal pigment epithelium. Drug crystals deposited in periocular tissues enable sustained drug release, maintaining high intraocular concentrations while keeping systemic concentrations relatively low [17].

Depending on whether the disease predominantly affects the anterior or posterior segment, corticos-

Table 1: Common topical corticosteroids and their anti-inflammatory potencies

Medication	Peak anterior chamber concentration (ng/mL)	Relative anti-inflammatory potency	Effective anterior chamber anti-inflammatory effect
Prednisolone acetate 1% [6]	669.9-1130.0	1.00	31.3-53.0
Dexamethasone alcohol 0.1% [6,7]	31.0	6.25	9.3
Dexamethasone phosphate 0.1% [6,7]	30.5	6.25	9.2
Betamethasone phosphate 0.1% [6,9]	20.3	6.25	4.5
Betamethasone phosphate 0.5% [6,9]	7.7	6.25	1.8
Prednisolone sodium phosphate 0.5% [6,9]	25.6	1.00	1.0

Table 2: Injectable preparations of corticosteroid

Medication	Concentration	Potency (equivalent to cortisone)	Duration of action
Dexamethasone phosphate [13]	4 mg/mL	5	1-3 days
Triamcinolone acetonide [13]	40 mg/mL	1	2-4 months
Betamethasone phosphate [14]	3 mg/mL	5	1-2 days
Betamethasone phosphate/acetate [14]	3.6 mg/mL	5	7-10 days
Methylprednisolone acetate [15]	80 mg/mL	1	>3 months

teroids can be administered via subconjunctival, sub-Tenon, or transseptal injection. Subconjunctival injection is often selected for anterior uveitis, while sub-Tenon or transseptal injection may be used for intermediate or posterior uveitis. In cases with macular edema, the injection technique should aim to deliver the drug as close to the macular region as possible [13,15].

The most commonly used transseptal injection technique involves administering the drug through the inferior eyelid into the orbital floor. In sub-Tenon injection techniques, corticosteroids may be administered superotemporally or inferiorly. In addition, sub-Tenon injections may be performed in the posterior Tenon capsule—where the depot is invisible on clinical examination—or in the anterior Tenon capsule, where the depot remains visible and can be surgically removed if necessary [13,15].

In addition to elevated IOP and cataract formation, complications of periocular injection vary depending on the technique used. Subconjunctival injections may lead to subconjunctival hemorrhage [13], while Sub-Tenon injections may cause ptosis if performed superiorly [13]. Transseptal injection carries risks of orbital fat prolapse through the septum, and—in the most severe cases—globe penetration, which may result in retinal breaks or vitreous hemorrhage [13].

Intravitreal injection

Intravitreal corticosteroids are commonly indicated for patients with non-infectious uveitis who exhibit an inadequate response to systemic corticosteroids, cannot tolerate systemic therapy, or present severe, vision-threatening intraocular inflammation (e.g., sympathetic ophthalmia) or cystoid macular edema [18–21]. This route of administration is also employed in uveitis patients associated with systemic diseases, including Behçet's disease, particularly as a bridging therapy to long-term immunosuppressive treatment [21].

The most frequently used agent is preservative-free triamcinolone acetonide at a concentration of 40 mg/mL, typically administered at a dose of 4 mg/0.1 mL with a 27-gauge needle. The injection site is located 4 mm posterior to the limbus in phakic eyes and 3.5 mm in aphakic eyes, avoiding the 3, 6, 9, and

12 o'clock meridians. Peak therapeutic efficacy occurs approximately one month post-injection, with effects generally sustained for up to three months [18]. Numerous studies have demonstrated that intravitreal triamcinolone significantly reduces macular edema and decreases the need for systemic corticosteroids and immunosuppressive agents [21–25]. A randomized controlled trial comparing intravitreal triamcinolone with sham injections combined with systemic corticosteroids in patients with uveitic cystoid macular edema also reported superior anatomical and angiographic outcomes in the triamcinolone group, including greater resolution of edema and reduced fluorescein leakage on fluorescein angiography [25].

Despite its efficacy, the primary limitation of intravitreal triamcinolone is its relatively short duration of action, which may necessitate repeated injections [26,27]. Consequently, some authors advocate its use primarily in cases refractory to other treatment modalities to minimize injection frequency. The most significant adverse effect is elevated IOP; secondary glaucoma has been reported in 25–50% of cases. However, up to 99% of these cases respond to topical IOP-lowering therapy, and only 1% of patients require surgery to lower IOP.[26] In addition, intravitreal triamcinolone is associated with a fivefold increased risk of secondary cataract formation. Other potential complications include vitreous hemorrhage, retinal tears, and endophthalmitis [27].

Systemic administration

Systemic corticosteroids are indicated for patients with bilateral intermediate or posterior uveitis, or uveitis associated with systemic diseases. They are also recommended for cases unresponsive to local therapy, or in patients for whom local corticosteroids are contraindicated, such as those with ocular surface inflammation or uncontrolled elevated IOP, or when the inflammatory response is severe and vision-threatening. In pediatric patients, systemic corticosteroids are often preferred due to the challenges of administering local therapy, which may require sedation or general anesthesia [12,28].

Oral prednisone is the most commonly used systemic corticosteroid, typically initiated at a dose of 1–1.5 mg/kg/day. To minimize the risk of complications such as osteonecrosis, the total daily dose should ideally not exceed 60 mg. Once adequate inflammatory control is achieved, the dose should be gradually tapered to the lowest effective maintenance dose, which is generally recommended not to exceed 0.1 mg/kg/day or 7.5 mg/day. Maintenance therapy should be continued for at least three months following treatment initiation [29].

Before initiating systemic corticosteroids, patients should receive comprehensive counseling regarding their potential adverse effects. Baseline investigations are essential to exclude infectious causes of uveitis—including tuberculosis, syphilis, *Toxoplasma gondii*, and *Toxocara spp.*—as well as other systemic infections. Additional assessments should include chest radiography, skeletal imaging, electrocardiograms (ECG), blood pressure and blood glucose measurement, and an evaluation of body weight and height. In female patients of reproductive age, a pregnancy test should be performed before treatment begins. For patients requiring long-term corticosteroid therapy or those at elevated risk of osteoporosis, concurrent supplementation with calcium and vitamin D is advised [30,31].

Intravenous corticosteroids are typically reserved for severe, vision-threatening inflammation associated with systemic diseases, such as Behçet's disease, Vogt-Koyanagi-Harada syndrome, sympathetic ophthalmia, serpiginous choroiditis, or uveitis linked to multiple sclerosis. In these cases, intravenous methylprednisolone is usually administered at a dose of 1 g/day for three consecutive days, or at a lower dose (250–500 mg/day) for five consecutive days to reduce potential adverse effects. This is followed by oral prednisone at 1 mg/kg/day, with a gradual taper based on clinical response. Hospitalization is recommended during intravenous corticosteroid administration to monitor for rare but serious complications, including seizures, anaphylaxis, cardiac arrhythmias, and sudden cardiac death. These potential adverse events should be thoroughly discussed with patients and their families before treatment begins [12].

Systemic corticosteroids are associated with a range of adverse effects, including peptic ulceration, weight gain, psychological disturbances, osteoporosis, hyperglycemia, hypertension, Cushing's syndrome, adrenal insufficiency, and growth suppression in children. These risks should be carefully weighed and clearly communicated to patients and caregivers. Follow-up evaluations should include monitoring of blood glucose, blood pressure, height, weight, and repeat skeletal imaging when indicated, to rule out osteoporosis [12,29].

NOVEL CORTICOSTEROID ADMINISTRATIONS

Intravitreal implants

Corticosteroid implants are inserted into either the anterior or posterior chamber to treat uveitis, prevent

postoperative inflammation, or manage other retinal disorders with inflammatory components. These implants gradually release the drug at an optimal concentration, ensuring long-term therapeutic efficacy while minimizing adverse effects. Depending on their composition, implants may be biodegradable or non-biodegradable; the latter require surgical removal after drug depletion. A variety of implants containing different corticosteroids at varying concentrations are available and can be selected based on clinical presentation and therapeutic goals.

The *fluocinolone acetonide 0.59-mg implant* is a non-biodegradable device composed of a polyvinyl acetate and silicone shell enclosing a 0.59-mg fluocinolone acetonide core. This implant is surgically inserted through a pars plana sclerotomy and sutured to the sclera, providing sustained drug release at a rate of 0.3–0.5 μg/day for up to three years. It is approved for treating intermediate, posterior, and panuveitis[32]. The efficacy of the 0.59-mg fluocinolone acetonide implant has been demonstrated in several studies[33–36], most notably the Multicenter Uveitis Steroid Treatment (MUST) trial[33]. This study compared the implant with the combined treatment of systemic corticosteroid and immunosuppressive agents in 479 eyes from 255 patients. At 24 months, 88% of eyes in the implant group achieved inflammation control compared to 71% in the systemic therapy group, representing a statistically significant difference. In terms of safety, systemic therapy was associated with a higher risk of systemic infections requiring antibiotic treatment[37]. In addition to elevated IOP and cataract formation, the most common complications of the fluocinolone implant include hypotony (reported in 25% of patients) and implant dislocation (observed in 5% of cases). In certain cases, surgical removal of the implant was necessary due to scleral suture leakage or migration of the implant into the vitreous cavity[37].

The *dexamethasone 0.7-mg implant* is a biodegradable device administered intravitreally via a single-use applicator through the pars plana; the procedure can be performed in an outpatient setting. The implant consists of a drug core containing 0.7 mg of dexamethasone encased within a poly(lactic-co-glycolic acid) polymer matrix, which gradually degrades into water and carbon dioxide, allowing for sustained drug release[38]. Peak intravitreal concentrations of dexamethasone are maintained for approximately two months, with a therapeutic duration lasting up to six months—typically between three and four months—with no significant difference observed between vitrectomized and non-vitrectomized eyes[39]. This implant is approved for the treatment of non-infectious uveitis, diabetic macular edema, and macular edema secondary to retinal vein occlusion. A randomized clinical trial involving 229 patients with uveitis compared the efficacy of the 0.7-mg dexamethasone implant with a 0.35-mg implant and a sham procedure over a 26-week follow-up period[40]. At week 26, both treatment groups demonstrated statistically significant improvements in visual acuity compared to the sham group. The 0.7-mg implant group achieved a higher rate of vitreous haze resolution at both week 8 and week 26 relative to the other two groups. Notably, 25% of patients showed vitreous haze resolution as early as week 3, indicating the relatively rapid onset of therapeutic effects. Other studies have also demonstrated the efficacy of the 0.7-mg dexamethasone implant in treating uveitic macular edema and uveitis associated with systemic inflammatory diseases[41,42]. One limitation of the implant is its relatively short duration of action—typically three to four months, with a maximum of six months—which may require repeated injections. Although the implant has the potential to migrate into the anterior chamber, its biodegradable nature ensures that such adverse effects are generally transient. Furthermore, while the implant carries the typical risks associated with intravitreal corticosteroid administration, the incidence of elevated IOP and cataract formation is relatively low[40].

The *fluocinolone acetonide 0.18-mg implant* is a non-biodegradable cylindrical polyamide device containing a core of 0.18 mg fluocinolone acetonide. It is administered intravitreally via the pars plana using a specialized applicator and provides sustained drug release at a rate of 0.1–0.2 μg/day, with therapeutic effects lasting up to three years. The implant is approved for preventing recurrence in non-infectious intermediate and posterior uveitis. A phase III clinical trial involving 129 patients with non-infectious uveitis randomized participants into two groups: one receiving the fluocinolone 0.18-mg implant and a sham group. After 12 months of follow-up, the treatment group demonstrated significantly lower recurrence rates compared to the sham group. At 6 months, recurrence rates were 27.6% in the treatment group versus 90.5% in the sham group; at 12 months, these rates were 37.9% and 97.6%, respectively[43,44]. Advantages of the fluocinolone 0.18-mg implant include its long duration of action and ability to be administered under sterile conditions in an outpatient clinic setting. However, the drug concentration released from the implant is suitable only for maintaining remission in stable uveitis and is insufficient for managing acute or recurrent inflammatory episodes. Given

Table 3: The characteristics of corticosteroid implants for treating uveitis

Agent	Bio-degradable	Duration
Flucinolone acetonide 0.59 mg [32]	No	Up to 3 years
Dexamethasone 0.7 mg [38]	Yes	Up to 6 months (normally 3 – 4 months)
Flucinolone acetonide 0.19 mg [43,44]	No	Up to 3 years
Flucinolone acetonide 0.18 mg [45]	No	Up to 3 years

its non-biodegradable nature, the implant may migrate into the anterior chamber, potentially leading to corneal decompensation. Therefore, caution is advised in aphakic patients or pseudophakic patients with compromised posterior capsules [43,44].

Finally, a *fluocinolone acetonide 0.19-mg implant* has a similar design and drug load to the 0.18-mg implant. The implant is inserted intravitreally through the pars plana using a 25-gauge applicator and releases fluocinolone at a rate of 0.2–0.5 mg/day for up to 3 years [45]. It has been approved for treating non-infectious uveitis in several European countries, but is currently approved only for diabetic macular edema in the USA [46].

For convenience, the characteristics of currently available implants for treating uveitis are summarized in Table 3.

Suprachoroidal injection

The concept of delivering corticosteroids into the suprachoroidal space—located between the choroid and sclera—has been explored since 2006 [47]. Suprachoroidal injection involves a microneedle designed to penetrate precisely 1 mm deep, ensuring accurate drug delivery. The injection site mirrors that used for intravitreal injections, requiring standard aseptic preparation in patients. Triamcinolone acetonide at a concentration of 40 mg/mL serves as the preferred agent, with an injection volume of 0.1 mL [47].

Theoretically, the suprachoroidal route allows corticosteroids to be delivered directly to the target tissues, allowing for higher drug concentrations in the choroid and thereby enhancing therapeutic efficacy. In addition, this route minimizes systemic drug exposure, reducing the risk of systemic side effects. Suprachoroidal injection also helps avoid complications associated with intravitreal injection, such as vitreous hemorrhage, retinal tears or detachment, and endophthalmitis [47].

Several custom-made devices have been developed to facilitate suprachoroidal injection techniques. Recently, specialized commercial devices have been produced for administering triamcinolone into the suprachoroidal space, allowing regulatory-approved clinical trials to evaluate the efficacy of this approach [1].

Phase I and II clinical trials (involving 9 and 22 patients, respectively) have yielded promising results, demonstrating improvements in visual acuity and reductions in macular edema as well as a favorable safety profile [48,49]. The Phase III PEACHTREE trial included 160 patients with macular edema secondary to non-infectious uveitis, randomized into two groups: the treatment group ($n = 96$), who received two suprachoroidal injections of triamcinolone at 12-week intervals; and the control group. The primary endpoint was the proportion of patients achieving a 15-letter or greater gain in best-corrected visual acuity (BCVA) at week 24. Upon completion of the study, the treatment group showed significant improvements in both visual acuity and macular edema compared to the control group [50].

Potential complications of suprachoroidal injections include inadvertent penetration into the vitreous cavity and choroidal hemorrhage.[47]

CONCLUSIONS

In summary, corticosteroids represent a double-edged sword in the management of uveitis. They are used early in treatment due to their rapid and potent anti-inflammatory effects. Various corticosteroids have been administered systemically (orally or intravenously) or locally (topical instillation, periocular injection, intravitreal injection, intravitreal implant, or suprachoroidal injection), each with distinct advantages and risks. However, corticosteroid therapy often causes adverse effects, ranging from local complications, such as elevated intraocular pressure and cataract formation, to systemic effects that include adrenal insufficiency and Cushing's syndrome, especially when high doses are used over prolonged periods. Regardless of the route of administration, corticosteroids are not considered suitable for the long-term management of chronic uveitis.

ABBREVIATIONS

IOP: intraocular pressure

Competing interests

The author(s) declare that they have no competing interests

COMPETING INTERESTS

The authors declare no competing interest.

AUTHORS' CONTRIBUTIONS

Conceptualization: B.H.T and T.C.T.T.N; methodology: T.C.T.T.N; writing – original draft preparation: B.H.T and H.N.L; writing – review and editing: B.H.T and T.C.T.T.N. All authors have read and agreed to the published version of the manuscript.

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