Review Article:

Management of Subepithelial Infiltrates Following Viral Keratoconjunctivitis: A Narrative Review

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Abstract:

Viral keratoconjunctivitis is one of the most common contagious ocular conditions encountered in ophthalmic practice. Multifocal subepithelial infiltrates (SEIs) represent its most frequently observed and clinically significant complication, with an estimated incidence ranging from 49.1% to 80%. SEIs are believed to result from cellular immunological reactions triggered by adenoviral replication in the corneal stroma beneath Bowman's membrane. This narrative review examined the evidence for medical and surgical management strategies for SEIs associated with viral keratoconjunctivitis, using PubMed, Google Scholar, and Web of Science databases guided by PRISMA methodology, including 66 relevant articles published after 1990. Corticosteroids provide short-term symptom relief but carry risks of recurrence upon discontinuation and serious adverse effects including cataracts, glaucoma, and enhanced viral replication. Corticosteroid-sparing agents -- cyclosporine A and tacrolimus -- demonstrate superior efficacy with lower recurrence rates and a more favourable safety profile, though tacrolimus may cause burning and foreign-body sensation. Surgical approaches, particularly phototherapeutic keratectomy (PTK) and photorefractive keratectomy (PRK), yield significant long-term improvements in visual acuity and corneal transparency. The adjunctive use of mitomycin C (MMC) with PTK or PRK substantially reduces postoperative haze and recurrence risk. In conclusion, corticosteroid-sparing agents and excimer laser procedures with MMC offer superior long-term outcomes. Controlled comparative studies with extended follow-up periods are required to validate these results and further define recurrence rates.

Keywords:

Viral keratoconjunctivitis; Subepithelial infiltrates; Adenovirus; Corticosteroid-sparing agents; Phototherapeutic keratectomy; Photorefractive keratectomy

Introduction:

Viral conjunctivitis is one of the most common contagious disorders encountered in general and ophthalmic primary care practice. Adenovirus is the most frequent causative agent, accounting for 15% to 70% of all cases worldwide; far less common causes include herpes simplex virus, varicella-zoster virus, influenza virus, picornavirus, and Epstein-Barr virus.[1-4] Clinical features include conjunctival hyperemia, chemosis, engorgement of blood vessels, ocular discharge, and pain. The estimated rate of transmission has been reported to range from 10% to 50%, necessitating preventive measures such as isolation of infected individuals, hand washing, and strict sterilization.[5-7] The prevalence of viral conjunctivitis varies with patient age and season. Diagnosis relies primarily on clinical features.[8-13]

Complications include pseudomembrane formation, occasional bacterial superinfection, and -- most importantly in clinical practice -- multifocal subepithelial infiltrates (SEIs).[14,15] This review aims to comprehensively examine the pathogenesis and management of SEIs complicating viral keratoconjunctivitis, to guide clinical decision-making and identify areas requiring further research.



Fig. 1: Ocular signs of inflammation in the left eye showing conjunctival hyperemia with dilated and engorged blood vessels, and visible ocular discharge.

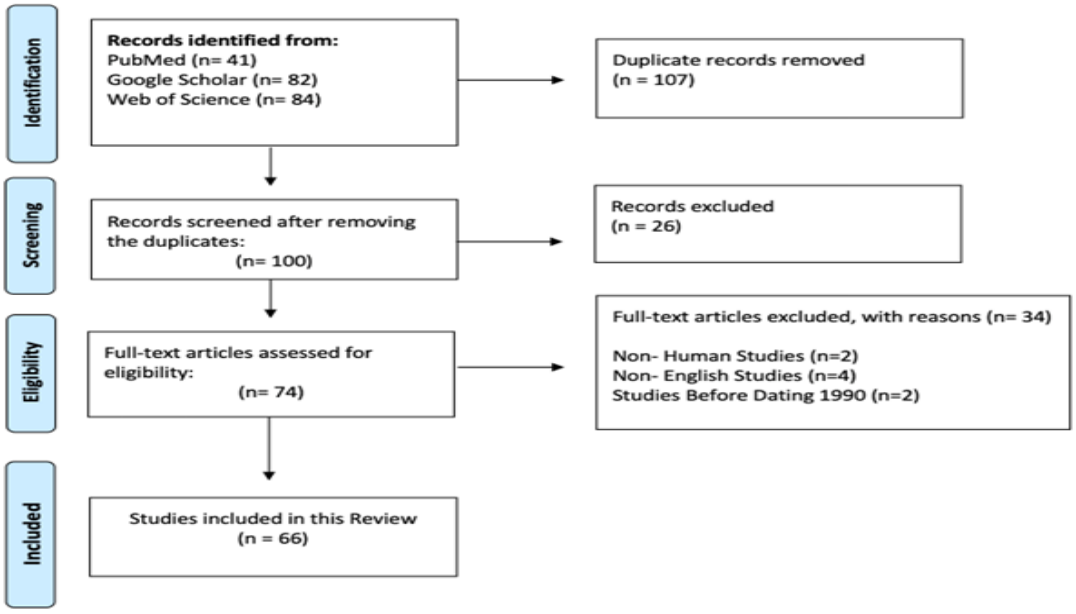


Fig. 2: PRISMA flowchart illustrating the study selection process, outlining the number of studies identified, screened, assessed for eligibility, and included in the review, along with reasons for exclusion at each stage.

CORNEAL SUBEPITHELIAL INFILTRATES:

Corneal SEIs are strongly associated with adenoviral conjunctivitis and are considered pathognomonic for adenoviral infection, typically appearing two to three weeks after onset of infection.[16] The incidence of SEIs is estimated to range from 49.1% to 80%.[17,18] The pathogenesis is believed to involve cellular immunological reactions triggered by viral replication in the corneal stroma beneath Bowman's membrane (BM).[16] This hypothesis is supported by studies demonstrating an early role of innate immune dendritic cells located at the level of Bowman's layer, with histopathological features including accumulation of lymphocytes, macrophages/monocytes, and activated fibroblasts.[19,20]

While SEIs are considered pathognomonic for adenoviral conjunctivitis, other reported causes include brimonidine tartrate use and acanthamoeba keratitis.[21,22] SEIs may persist for years, causing glare, reduced vision when infiltrates obscure the optical axis, foreign body sensation, dry eye syndrome, and progressive corneal scarring.[10,21,23] Slit-lamp examination reveals diffuse, multifocal, round-to-oval, greyish-white, pinhead-sized lesions typically located centrally but potentially involving the entire corneal surface.[16,23-25]

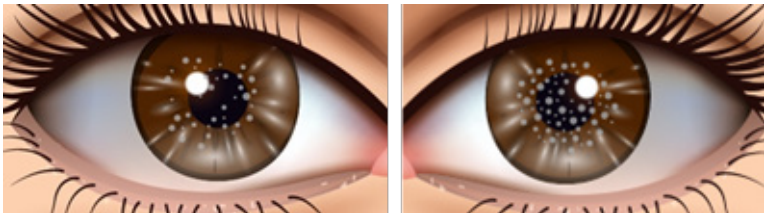


Fig. 3: Slit-lamp examination of corneal subepithelial infiltrates (SEIs) in both eyes, showing multiple greyish-white, pin-head-sized lesions distributed across the cornea.

The infiltrates typically resolve over time without impact on the cornea or visual pathway; however, they may progress to permanent corneal scarring. Optical coherence tomography (OCT) can differentiate reversible from irreversible scars, with Bowman's membrane serving as a key anatomical landmark.[26,27] Prevention through rigorous decontamination, use of disposable tonometer heads, and early application of povidone-iodine (PVP-I) 2% -- which reduces viral load and thereby limits the severity of complications -- is an important strategy.[28,29] A combination of PVP-I 1.0% and dexamethasone 0.1% has also been reported to prevent the development of SEIs when applied early.[30]

MEDICAL MANAGEMENT:

Corticosteroids

Topical corticosteroids are advised

only for severe cases of acute adenoviral keratoconjunctivitis (AAK) with chemosis, lid swelling, or membranous conjunctivitis, primarily to prevent scarring, or for symptomatic relief.[31] When compared with supportive care, both topical corticosteroids and cyclosporine were found to produce fewer serious symptoms and shorter symptom duration. When administered in the acute phase, steroids inhibit the formation of corneal SEIs.[31]

Three landmark reports have examined corticosteroid efficacy. The first identified 53 patients with culture-confirmed adenovirus type 8 infection; a 6-week course of steroid-neomycin combination drops produced no impact on conjunctivitis course or SEI formation compared with lubricants.[32] The second studied 74 patients during a large 1988 outbreak at a US military base in the Philippines in a double-masked trial comparing dexamethasone with artificial tears, and found no significant difference in manifestations or disease duration between groups.[33] The third, a double-blind study of 20 patients without established SEIs at baseline, demonstrated that topical corticosteroids prevented new infiltrate formation compared with placebo, but that SEIs recurred upon discontinuation and cleared again when steroids were resumed.[34]

Despite providing symptom relief, corticosteroids carry substantial risks: enhanced adenovirus replication, prolonged viral shedding, cataracts, glaucoma, opportunistic infection, and delayed corneal healing.[25] Current consensus recommends restricting corticosteroid use to patients with pseudomembranes or bothersome SEIs and avoiding prophylactic use early in infection. A stepladder approach -- beginning with an immunosuppressant such as tacrolimus and adding steroids only in recalcitrant cases -- has been proposed.[63]

Antivirals

No FDA-approved antiviral therapy currently exists that reliably reduces the duration of adenoviral keratoconjunctivitis, limits viral replication, or inhibits SEI formation. Ribavirin demonstrated limited in vitro effectiveness and was inadequate against the three most common EKC-associated adenovirus serotypes (Ad8, Ad19, Ad37).[35,36]

Ganciclovir has shown significant dose-dependent inhibitory activity against EKC-associated serotypes by PCR-based evaluation,[37] and a study in 18 patients in Saudi Arabia demonstrated that ganciclovir 0.15% ophthalmic ointment resolved conjunctivitis in 7.7 days versus 18.5 days with artificial tears.[38] Topical cidofovir 0.2% was well tolerated without local or systemic adverse effects; however, it produced no statistically significant benefit on the acute or chronic phase of adenoviral

keratoconjunctivitis, nor on the frequency of SEIs at 21 days.[39]

Corticosteroid-Sparing Agents

Cyclosporine A (CsA)

Cyclosporine A (CsA) is an immunomodulator that has demonstrated both efficacy and safety in the treatment of SEIs. A study of 18 symptomatic eyes from 13 patients who had previously received topical steroids for post-EKC SEIs used topical 0.05% CsA four times daily combined with loteprednol etabonate (LE) three times daily for three weeks, followed by tapering LE and continuing CsA for six months. Substantial symptom reduction (pain, halo/glare, photophobia) was achieved in 76.9%, 100%, and 100% of patients respectively.[40]

A further trial administered four drops of CsA daily to 70 eyes (48 patients) with post-AAK SEIs, initiated a median of 18.5 months after the acute phase. Of 70 eyes, 40 responded favourably, 16 did not respond, 4 discontinued due to ocular burning, and 10 had insufficient follow-up.[41] CsA 1% administered four times daily for 21 days during acute infection produced early subjective improvement and a trend toward reduced corneal opacity incidence.[39] Shifting from topical steroids to CsA reduced the total number of medications required, particularly in steroid responders requiring IOP-lowering agents.[42] A comparative study of low-potency corticosteroid versus 0.5% CsA reported complete SEI resolution in 70% of steroid-treated cases at six months, with a lower recurrence rate than previously reported in the literature.[64] Recurrence rates with CsA (5-9%) are markedly lower than with corticosteroids (up to 88%).[34,64,65]

Tacrolimus

Tacrolimus exerts its immunosuppressive and anti-inflammatory effects by inhibiting calcineurin, thereby suppressing T-cell proliferation and activation.[42] FDA-approved concentrations for systemic use (0.1% and 0.03%) are available; topical tacrolimus 0.03% has proven beneficial for giant papillary conjunctivitis and vernal keratoconjunctivitis.[43,44]

Bhargava et al. demonstrated that tacrolimus 0.03% ointment was more effective than dexamethasone 0.05% ointment for post-AK SEIs, with a lower recurrence rate and no significant IOP elevation, although burning and foreign-body sensation were reported.[25] A prospective non-controlled interventional case series in patients who had failed or been intolerant of topical corticosteroids administered tacrolimus 0.03% ointment twice daily for 22 weeks, achieving significant objective clinical improvement in all patients without severe SEIs at endpoint; one case of extreme dizziness

was documented as an unusual adverse effect.[45] A separate report of seven steroid-unresponsive patients found treatment success in 6 of 7 patients (85.7%), with satisfactory control of SEIs and relief of ophthalmological complaints.[46] Recent evidence has confirmed tacrolimus efficacy against persistent SEIs in resistant cases.[66]

SURGICAL MANAGEMENT:

When medical management with corticosteroids or antivirals fails to restore corneal transparency, or when long-term medication use becomes intolerable, surgical intervention is considered. SEIs may persist for months to years after EKC, and the long-term use of corticosteroids carries risks of cataract, glaucoma,[23] and corneal stromal thinning.[24]

Phototherapeutic keratectomy (PTK) and photorefractive keratectomy (PRK) use an argon fluoride 193-nm excimer laser to ablate corneal tissue and are established as safe and effective modalities for anterior corneal disorders.[47,48] In the context of EKC, removal of subepithelial and stromal tissue reduces immunogenic stimulation and thereby lowers the risk of recurrence.[49] Mitomycin C (MMC), an alkylating antitumour antibiotic that decreases cell proliferation analogously to radiation, is used adjunctively in PTK and PRK to prevent subepithelial fibrosis, reduce corneal haze, and enhance wound healing.[50,51]

Phototherapeutic Keratectomy (PTK)

Multiple studies have confirmed the efficacy of PTK for SEIs, demonstrating significant visual improvement.[24,52,53] PTK also smoothens the irregular corneal surface associated with subepithelial opacity, further improving vision quality.[54] Yamazaki et al. reported successful SEI removal with PTK plus low-dose MMC in all patients, with significant improvement in contrast sensitivity and patient-reported visual acuity and quality.[24] Elhamaky conducted a comparative study of transepithelial PTK with MMC versus topical tacrolimus in post-adenoviral SEIs, finding significant improvement in best-corrected visual acuity, OSDI score, corneal densitometry, and higher-order aberrations in both active treatment groups but not in controls.[52] Oliveira et al. confirmed significantly improved visual acuity in the PTK group compared with controls.[55] Preoperative assessment of lesion depth using pentacam or OCT is recommended to guide the extent of ablation.[24,52]

Photorefractive Keratectomy (PRK)

Several reports confirm the efficacy and safety of PRK for SEI removal.[23,49,56] PRK without MMC has successfully corrected myopia, eliminated central SEIs, and prevented central recurrence, with only peripheral asymptomatic infiltrates observed at follow-up.[22,48] The topical use

of MMC with PRK provides satisfactory clinical and refractive results without postoperative complications.[23,56] PRK is preferred over PTK in patients with pre-existing myopia because it offers greater refractive accuracy.[23,57]

COMPLICATIONS:

A well-documented complication of both PTK and PRK is a hyperopic shift resulting from ablation and central corneal flattening.[58,59] The magnitude of this shift is variable but largely proportional to ablation depth.[58,60] Minimal tissue ablation produces less hyperopic shift and better visual quality.[61] Strategies to minimise hyperopic shift include moving the eye in a circular fashion during laser application to maintain a convex corneal shape,[24] and employing a two-step PTK procedure to achieve shallower ablation.[52,58,62]

Additional complications of both procedures include corneal haze formation, postoperative pain, and recurrence of the initial corneal pathology.[23,56] All these risks, including hyperopic shift, can be significantly reduced by intraoperative MMC, shallow ablation, and enlarging the treatment zone to 8-9 mm.[50,52,62]

RECURRENCE:

Recurrence rates following treatment of SEIs vary substantially depending on the management modality. Bhargava et al. reported recurrence rates of 7.5% with tacrolimus and 17.5% with corticosteroids.[24] PTK has demonstrated a recurrence rate of 9.6%, which was observed concurrently with steroid discontinuation.[16,24,25] In the study by Oliveira et al., PRK with MMC yielded a recurrence rate of 11.7% in the treatment group compared with 77.7% in controls.[16,24,25] Elhamaky's comparative study found SEI persistence at one year significantly lower in the transepithelial PTK group (9%) and tacrolimus group (23.8%) compared with controls (85%).[52]

PRK without MMC prevents central recurrence but not peripheral infiltrates in some reports, while central recurrence has been observed in others.[22,48] The combination of MMC with PRK appears to prevent recurrence effectively, with no recurrence reported in two studies over more than one year of follow-up.[23,56]

Conclusion:

Corticosteroid-sparing agents -- particularly cyclosporine A and tacrolimus -- demonstrate superior efficacy against SEIs compared with corticosteroids, with lower recurrence rates and a more favourable adverse effect profile. Burning and foreign-body sensation remain the primary side effects associated with tacrolimus. Surgical management with PTK or PRK provides better long-term outcomes and lower recurrence rates than medical management alone. The adjunctive

use of MMC with PTK or PRK reduces the risk of recurrence and significantly decreases postoperative complications such as haze formation. Additional long-term, controlled comparative studies are needed to assess the persistence of outcomes and long-term recurrence risks, as most published studies have follow-up intervals of less than two years. Larger comparative studies are also required to define optimal treatment selection between PTK, PRK, and medical management.

DECLARATIONS:

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