
Treatment of interface keratitis with oral corticosteroids

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ABSTRACT

Purpose: To describe the results of treating interface keratitis using a combination of intensive topical and oral corticosteroids.

Setting: Casey Eye Institute, Portland, Oregon, USA.

Methods: Thirteen eyes treated for grade 2 to 3 interface keratitis using an oral corticosteroid (prednisone 60 to 80 mg) as well as an hourly topical corticosteroid were retrospectively reviewed. The best corrected visual acuity (BCVA) was used as an objective guide of whether to treat with intense topical and oral corticosteroids, flap irrigation, or both. Predisposing factors such as intraoperative epithelial defects or a history of severe allergies or atopy were also looked for.

Results: All 13 eyes responded favorably to the combination of intensive topical and oral corticosteroids and had a BCVA of 20/20 after the keratitis resolved. In 6 eyes (46%), the patients had a history of severe seasonal allergies. One day postoperatively, 3 eyes (23%) had an epithelial defect and 2 eyes (15%), lint particles or debris embedded in the interface. With oral corticosteroid use, 3 patients (23%) noted mild stomach irritation and 2 (15%) noted nervousness. All 5 side effects resolved without sequelae. No patient developed a serious side effect.

Conclusion: A short, intense course of an oral corticosteroid was an effective treatment in patients with grade 2 or higher interface keratitis when combined with a topical corticosteroid administered hourly. The BCVA is a helpful objective measure of the severity of interface keratitis and can be used to guide the clinician in the therapeutic strategy.
J Cataract Refract Surg 2002; 28:454–461 © 2002 ASCRS and ESCRS

Interface keratitis or diffuse lamellar keratitis (DLK) can be a serious complication after laser in situ keratomileusis (LASIK).^{1–5} Its onset is often insidious and the etiology, thought to be multifactorial, is unclear.^{6–16} While there have been many theories about the cause of interface keratitis, there are a limited number of treatment strategies. Currently, the 2 main strategies involve relieving the flap and irrigating the flap interface to re-

move inflammatory cells and any antigenic stimulus that may be provoking the inflammation and using intensive topical corticosteroid drops to suppress the inflammation locally. We propose a third strategy that includes the use of a high-dose oral corticosteroid. We also identify several risk factors that may predispose patients to interface keratitis.¹⁷

Patients and Methods

The cases comprised 13 eyes of 13 patients who developed interface keratitis (DLK) after LASIK (Table 1). They occurred from April 1998 to October 2000. The cases were identified by a retrospective review of the

Accepted for publication September 18, 2001.

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medical history and surgical records. During this period, all eyes that developed grade 3 or higher interface keratitis (Linebarger classification)¹⁸ were treated with a high-dose oral corticosteroid (prednisone).

Patients were questioned about past medical conditions to exclude a history of tuberculosis, fungal infections, amebiasis, peptic ulcer disease, or diabetes, which can be exacerbated by oral corticosteroid use. The high-dose oral corticosteroid dosage was 60 to 80 mg per day in a single dose. For more severe interface keratitis, 60 to 80 mg were given on the first day and divided into 30 to 40 mg 2 times daily on subsequent days to increase the potency of the antiinflammatory treatment as needed until the interface keratitis improved on slitlamp examination.

The patients were also treated with hourly maximum-potency topical corticosteroid drops (prednisone acetate 1%) while awake. If the patient had an epithelial defect, the topical corticosteroid was tapered to 4 times a day after the first 24 hours of treatment. The oral corticosteroid was gradually tapered over a 1-week to 10-day period once the inflammation was controlled. To avoid adrenal suppression and a rebound inflammatory reaction, the oral corticosteroid was usually tapered by 20 mg every other day to 20 mg and then the oral corticosteroid was reduced to 10 mg for 1 to 2 days before it was discontinued entirely. The topical corticosteroid was also tapered gradually over 1 to 2 weeks. To minimize the risk of ocular hypertension, an attempt was made to completely stop oral and topical corticosteroid use within 10 days of initiating therapy. Topical prophylactic antibiotic agents were given 4 times a day and also tapered as the inflammation subsided.

Before surgery, patients received a complete eye examination including manifest and cycloplegic refractions as well as a medical history and a detailed history of their allergies, skin conditions, and inflammatory diseases. Patients were routinely asked whether they had a history of childhood asthma, chronic bronchitis or sinusitis, contact dermatitis, or a family history of severe allergies or atopy.

On the day of surgery, patients were prepared with povidone—iodine (Betadine®) and the eye was draped to remove lashes from the operative field. A lid speculum was then placed and the microkeratome pass performed using a Hansatome® (Bausch & Lomb). The patients were treated with 1 of 4 excimer lasers: Summit Apex

Plus, VISX Star 2, Nidek EC-5000, or Bausch & Lomb Technolas® 217. The excimer laser treatments were based on the patient's functional requirements and the surgeon's judgment. The flap interface was routinely irrigated with balanced salt solution (BSS®) after the laser treatment. The patients were given ofloxacin (Ocuflox®) a minimum of 3 times postoperatively after the flap was repositioned and allowed to adhere for 3 minutes. A corticosteroid was not routinely given in the first 12 to 24 hours postoperatively. Thus, none of the eyes with interface keratitis received a topical corticosteroid in the first 12 to 24 hours.

Patients with epithelial defects were treated with a therapeutic soft contact lens if the epithelial defect involved more than 30% to 40% of the corneal flap surface. The soft lens was removed once the epithelium healed. Postoperatively, eyes were routinely treated with topical tobramycin 0.3% and dexamethasone 0.1% (TobraDex®) 4 times a day starting 12 to 24 hours after surgery. If the patient had interface keratitis, the TobraDex was continued 4 times per day in addition to the intensive topical corticosteroid until the inflammation subsided.

Interface Keratitis Management

Once patients were diagnosed with interface keratitis, they were treated with the following regimen:

1. Hourly prednisolone acetate 1% for interface keratitis greater than grade 2.
2. Topical TobraDex continued 4 times a day concurrently.
3. Treatment with high-dose oral prednisone (60 to 80 mg) daily for grade 3 interface keratitis in which the best corrected visual acuity (BCVA) was 20/30 or worse. The tapering regimen is described above.
4. Treatment with flap relifting and irrigation of the flap interface and inflammatory cells if the BCVA was 20/50 or worse.

The BCVA was an important objective guide to the severity of the interface keratitis.

Results

Thirteen eyes of 13 patients developed grade 2 to 3 interface keratitis. The clinical data are summarized in Table 1. Two patients developed bilateral interface

Table 1. Oral corticosteroid use with interface keratitis.

Patient	Age (Years)	Sex	Allergy History (Oral)	Day of Onset and Postoperative Treatment	Oral Prednisone Dose
1	41	F	Penicillin	Day 1	60 mg, 1-week taper
2	38	F	Severe atopic, asthma GPC	Day 1	60 mg, 1-week taper
3	43	F	PCW, aspirin, rosacea, meibomian gland dysfunction	Day 1	60 mg
4	39	F	Atopic dermatitis, seasonal allergies, childhood asthma, eczema, bronchitis	Day 1 postop; OU IK noted; corneal erosion noted OD	60 mg, lifted and irrigated
5	41	M	Contact dermatitis, asthma, allergies (dust mites, mold, etc.)	Day 1	80 mg initially; 40 mg PO 2× day × 5 days then taper
6	38	F	Drug allergy only; sulfa	Day 1 mild grade 1/2; day 4 Grade 3 IK, flap reflected and irrigated	60 mg pred; 20 mg PO 3×/day for 5 days then taper over 10 days
7	43	M	Tape adhesives	Day 1 epithelial defect; day 4 grade 3 IK	60 mg, then 40 mg, then 20 mg
8	51	F	Atopic, eczema, penicillin allergy	Day 1 20/30; day 2 20/70 grade 3	20/60 (6-line loss)
9	47	F	Darvon, TB test allergy	Day 1 20/25; day 2 20/30 blurry	1 week taper; day 2 60 mg
10	53	F	Atopic individual, eczema, childhood asthma	Day 1 20/20 ⁻¹ ; day 2 20/30 ⁻²	60 mg then 20 mg 3×/day tapered over 1 week
11	50	M	Nonatopic but had PRK haze reaction in other eye previously; no allergies	Day 1 20/30 BCVA; day 2 20/60 BCVA	60 mg, hourly topical steroids; 20 mg 3×/day
12	32	M	Childhood allergies	Day 1	80 mg, tapered over 10 days
13	41	M	None	Day 1	60 mg

BCVA = best corrected visual acuity; Epi = epithelial; GPC = glant papillary conjunctivitis; PCN = penicillin; IK = interface keratitis; inf = inferior

keratitis, but the contralateral eye was less severe than grade 3 interface keratitis. In all eyes, the interface keratitis was recognizable on the first day postoperatively. All eyes responded to treatment with a combination of intensive full-strength topical and oral corticosteroids. In 3 severely affected eyes (23%), the flap was lifted and the flap interface irrigated.

All eyes recovered a BCVA of 20/20 or better after treatment (Table 1). In all patients, the oral and topical corticosteroids were discontinued by 2 weeks.

Predisposing Factors

Six patients (46%) had a history of severe seasonal allergies, atopy, and adult or childhood asthma. On the

first day postoperatively, 3 eyes (23%) had an epithelial defect and 2 eyes (15%) had lint particles or debris embedded in the interface.

Complications

Three patients (23%) noted mild stomach irritation that was relieved by antacids. Two patients (15%) noted anxiety and nervousness: This resolved spontaneously in 1 patient and subsided in the second after the oral steroid was tapered from 60 mg/d to 40 mg/d after 2 days of treatment. No patient developed serious side effects as a result of the oral corticosteroid therapy. No patient was noted to have an elevation in intraocular pressure (IOP) or signs of glaucoma.

Table 1. (cont.)

Maximum Acute Reduction BCVA	Interface Keratitis	Final BCVA 20/20	Comments
20/60	Grade 3	20/20	Epi defect; LASIK retreatment over PRK; flap lifted and irrigated
20/40	Grade 3	20/20 OU 6 mo postop	Took Alomide FML intermittently over first 6 months for itching allergic conjunctivitis
20/200	Grade +3	20/20 2 mo postop	Rosacea, aspirin allergy
20/60 (6-line loss)	Grade +3 OD	20/20 5 mo postop	Severe atopic with ABM dystrophy; epithelial defect day postop; flap lifted and irrigated day 2
20/40	Grade 3	20/20 ⁻¹	Other eye had PRK with haze previously
20/25 BCVA day 1; 20/80 day 4	Grade 3	20/20 ⁻¹	Flap lifted and irrigated on day 4 postop
20/40 (3-line loss)	Grade 3	20/20 OS 6 mo postop	Possible hypersensitivity to tape; epithelial defect
20/70	Grade 3 OD; grade 1 OS	20/20 OU	Presumed allergy to Dexacidin; bilateral IK other eye grade 1
20/30 day 2 (2-line loss)	Grade 3	20/20	Superior interface lint; local IK superiorly
20/30 (3-line loss) from 20/15 postoperatively	Grade +2-3 OD; grade 2 OS	20/20 +1 3 mo postop	Quick response to oral steroids; bilateral IK other eye grade 2
20/20 to 20/60 (5-line loss)	Grade +3	20/20 ⁻²	Other eye had severe haze response to PRK
BCVA dropped 2 lines from 20/15 to 20/25	Grade 2-3	20/15	Moderate allergic history; no other predisposing factors
20/40	Grade +3	20/20	Lint in interface inf. temp; flap lifted and lint removed on day 2 postoperatively

Discussion

Our management of interface keratitis or DLK includes a third strategy besides the use of frequent (hourly) topical corticosteroid drops and lifting the flap and irrigating the interface in severe cases.^{1,5-7,14,17,18} We found the use of an intense, short-term oral corticosteroid helpful in controlling interface keratitis when it affects vision. Within 24 to 48 hours, the interface keratitis usually began to resolve and the vision slowly improved. The oral corticosteroid dosage was based on a methylprednisolone plasma half life of 78 to 188 minutes and a biologic half life of 18 to 36 hours.¹⁹

We began using an oral corticosteroid after discussions about the mechanism of interface keratitis with

several dermatologists and an ocular immunologist. Two of the dermatologists noted the similarity in the time course of interface keratitis and poison oak contact dermatitis. They used a combined approach of topical and oral corticosteroids (60 to 80 mg/d) to control the swelling and inflammatory damage caused by the contact dermatitis associated with poison oak as well as atopic dermatitis.^{20,21}

We found the use of an oral corticosteroid to treat interface keratitis advantageous for several reasons. First, an oral corticosteroid taken once or twice daily is more likely to be properly carried out (compared with an hourly topical corticosteroid that requires more diligent compliance throughout the day). Second, an oral corticosteroid provides around-the-clock antiinflammatory

treatment when the patient is sleeping. An hourly topical corticosteroid is typically not given while the patient is sleeping or if attempted, proper compliance may be difficult. Third, an oral corticosteroid can be used in a complementary fashion with the topical corticosteroid. This allows both local and systemic suppression of the immune response to minimize interface inflammation. If this aggressive corticosteroid combination is used before the inflammation progresses to grade 4 interface keratitis, intrastromal scarring may be avoided. Fourth, in eyes with large epithelial defects, the use of intense topical corticosteroid drops and their preservatives may inhibit reepithelialization and, in some instances, cause the epithelium to slough entirely.²² The use of an oral corticosteroid helps reduce the inflammatory response without causing significant local epithelial toxicity or reducing reepithelialization. In eyes with severe epithelial defects, we used an intense oral corticosteroid dose, 60 to 80 mg, and reduced the topical corticosteroid from hourly to 4 to 6 times per day maximally to encourage reepithelialization while controlling inflammation.

There are many proposed causes of interface keratitis.^{5-8,10,12,14,23} We think the most compelling theory is that an antigenic endotoxin on the gram-negative cell wall surface is capable of inciting an intense neutrophilic response.^{2,24} This lipopolysaccharide (LPS) is stable for short cycles of steam sterilization used with most LASIK instruments. Holland et al.² suggest that the sterilizer water reservoirs may breed bacteria if not drained after use. The bacteria are killed during sterilization, but their biofilm excites an inflammatory reaction. This includes debris from the cell wall such as the LPS (endotoxin from gram-negative bacteria), and the peptidoglycan (gram-positive bacteria) may deposit on the surgical instruments during the sterilization process. The surgical instruments introduce this foreign debris into the interface during surgery.

The use of an intensive hourly topical steroid or oral corticosteroid should be approached with caution because it may aggravate an infection if a bacterial inoculum is the cause of the keratitis. Reports of infectious keratitis after LASIK are uncommon but do exist.^{11,12}

Ocular hypertension can be particularly difficult to detect in post-LASIK eyes because a small cleft or pseudochamber may form in the stromal interface at the level of the flap, causing an artificially low or normal IOP. (The actual IOP may be 40 mm Hg or greater

when measured on the peripheral cornea away from the cleft.)

There are 2 recent reports of glaucoma or ocular hypertension, which is difficult to detect and may be associated with atypical DLK after the use of an intensive topical corticosteroid. In the case reported by Najman-Vainer and coauthors,³ a fluid-filled cavity developed in the interface. This led to an erroneous Goldmann IOP measurement of 3 mm Hg over the central cornea, which had a cystic cleft in the interface. The actual IOP, measured by a TonoPen® on the peripheral cornea, was 38 mm Hg. The retinal examination revealed severe glaucomatous cupping with a 1.0 cup-to-disc ratio 6 months postoperatively. In the second case, Lyle and Jin⁴ noted interface fluid accumulation associated with intensive topical corticosteroid use and epithelial ingrowth in the interface. This was also associated with ocular hypertension, which was detected after drainage of the interface fluids. The interface fluid resolved after repeated surgical scraping of the interface and discontinuation of the topical corticosteroid.

If possible, the topical and oral corticosteroid treatment should be limited to 10 to 14 days because of the risk of corticosteroid-induced ocular hypertension and glaucoma. We believe it is prudent to limit intensive topical and oral corticosteroid use to the first 10 days after surgery. If corticosteroids are used subsequently, the IOP should be monitored by methods that avoid the central cornea if there is any evidence of interface cyst formation.

In our series, we noted host factors that may play a role in the development of interface keratitis. Six eyes (46%) that developed interface keratitis were in patients with a history of seasonal environmental allergies, atopic disease, childhood asthma, or chronic bronchitis or sinusitis. The allergic tendency in patients with these conditions may predispose the patients to develop a more dramatic inflammatory response than occurs in individuals with fewer allergic tendencies. The patients responded well to therapy with aggressive topical and oral short-term corticosteroid treatment. A short course of a high-dose corticosteroid is well tolerated in a healthy individual and is used safely in dermatologic conditions. In patients with atopic dermatitis, a short course (1 week) of an oral corticosteroid (60 to 80 mg/d) has been recommended to treat atopic dermatitis exacerbation.^{20,21} A similar

strategy is used to treat acute asthmatic exacerbation with an oral high-dose corticosteroid.

Three eyes (23%) that had epithelial defects developed grade 3 interface keratitis. Interface keratitis associated with an epithelial defect was noted after corneal scraping without lifting the flap by Steinert and coauthors.¹⁵ Similarly, Haw and Manche¹⁴ noted late interface keratitis in 6 eyes that were 2 to 12 months post LASIK. Eyes that have an epithelial defect at the time of surgery or later may have as much as a 24-fold increase in the risk of developing interface keratitis.¹³ The prophylactic use of oral corticosteroids in a patient who has a large epithelial defect may be considered. We have treated 10 patients using this strategy with good results.

In our cases, the interface keratitis was controlled with a combination of topical and oral corticosteroids. With oral corticosteroid use, we were able to reduce the topical corticosteroid use to 4 to 6 times per day in some cases 24 hours after initiating an hourly topical corticosteroid. This reduced dosage was less likely to inhibit reepithelialization than an hourly topical corticosteroid, which sometimes leads the entire epithelium to slough and may reduce reepithelialization.²²

In 2 cases in this series, lint particles and debris were noted in the interface on the first postoperative day. Although the particles were away from the visual axis, the interface keratitis reduced the BCVA to 20/40. The vision improved quickly with intense topical and oral corticosteroid use. In 1 case, the flap was lifted and irrigated and the lint removed. In the second case, the lint particle was observed and intensive oral and topical corticosteroids given. The lint particle was resorbed over 6 months. The interface keratitis was more intense around the interface debris, but it did spread out over the visual axis to reduce the vision temporarily. We have had other cases in which lint was lodged in the interface but there was only a minimal interface keratitis reaction, which suggests that some lint is more antigenic. We think it is reasonable to remove any debris that lies directly over the pupil if there is significant inflammation because of the risk of subsequent scarring. In some recent cases, when the lint or debris was peripheral, we have been able to remove lint particles or debris with a jeweler's forceps without relifting the flap.

There was no significant scarring, irregular astigmatism, or BCVA loss in the 13 cases. This may be a result of our aggressive approach to interface keratitis. We used

the BCVA as an objective monitor of interface keratitis severity; it helped guide our treatment regimen. We recommend the aggressive use of topical and oral corticosteroids when there is moderate BCVA loss to 20/40. When the BCVA drops below 20/50, relifting and irrigating the flap should be considered within 1 to 2 days of surgery or as soon as possible. We have seen several patients with severe interface keratitis whose treatment was delayed beyond the first 48 hours and was less aggressive. These patients developed focal scarring that reduced the BCVA below 20/50, and they noted marked distortion of vision from irregular astigmatism. Fraenkel et al.¹ note that the vision in patients with severe interface keratitis improves with time, but in our referred cases, several patients had a slow and disabling visual recovery and several eyes had persistent BCVA loss after 6 months.

Interface keratitis is a sight-threatening condition similar to a corneal or intraocular infection that requires prompt attention within the first 24 to 48 hours.^{5-7,18} If the interface keratitis begins to affect vision, it is critical to treat it promptly (within 24 to 48 hours) and aggressively with topical and oral corticosteroids and, if necessary, flap relifting and irrigation. We believe this early, aggressive treatment minimizes the likelihood of a severe inflammatory reaction, which can result in scarring.

We now treat some patients prophylactically with an oral corticosteroid if they appear to be at high risk for interface keratitis. This includes patients who have severe atopy or allergies or large epithelial defects after LASIK. In retreatments, if a person has had severe interface keratitis, we often treat him or her with an oral corticosteroid preoperatively and postoperatively to minimize inflammation. We also use an oral corticosteroid in patients who have an epithelial debridement associated with treatment for microstriae in which the flap is relifted.

Another strategy to reduce the incidence of interface keratitis is proposed by Peters and coauthors²⁵ in which they apply a topical corticosteroid intrastromally during the procedure. This seems to reduce the incidence of nonspecific DLK. The authors note that it is well tolerated and are encouraged by the results. Further studies of the prevention of interface keratitis are warranted.

This study is a preliminary report, which suggests that the addition of an oral corticosteroid may be helpful in addition to the current regimen of an intensive topical corticosteroid and, in more severe cases, flap relifting

and irrigation. A clinical case series cannot directly answer whether oral steroid use is superior to topical use because of the small and sporadic number of cases and the variation in presentation patterns that we see in our practice. A large multicenter study may be a consideration in the future. Alternatively, the issue of the etiology and optimal management of interface keratitis may be approached using an animal model as recently reported by Peters et al.²⁴ Despite these limitations, we have found the strategy described to be useful.

In summary, we believe it is important for the surgeon to realize that interface keratitis is a sight-threatening condition.^{6,7,12} In this study, we noted that multiple allergies, atopism, an epithelial defect, or significant debris in the interface may be associated with a significant interface keratitis reaction. In our experience, the prompt use of intensive topical and oral corticosteroids is warranted when the BCVA is worse than 20/40. If more severe visual loss occurs, relifting and irrigating the flap may be indicated. The potential risks of the use of intensive topical and oral corticosteroids are not to be minimized. They should be balanced with the need to minimize inflammation and enhance visual recovery after LASIK.

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None of the authors has a financial interest in any product mentioned.