Bilateral Ring-Shaped Marginal Keratitis after Photorefractive Keratectomy: A Case Report

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Abstract

***Purpose*:** To report a case of bilateral dense ring-shaped marginal keratitis after photorefractive Keratectomy (PRK)

***Case report*:** A 44-year-old man developed bilateral dense ring-shaped marginal keratitis after PRK. The patient had moderate to severe meibomian gland dysfunction preoperatively that was treated incompletely. He was treated with mild topical antibiotics, topical and systemic steroids and systemic doxycycline. The condition was controlled with faint peripheral scarring.

***Conclusion*:** This case suggests the role of blepharitis and meibomian gland dysfunction in producing marginal keratitis after excimer laser for which complete preoperative treatment is recommended.

***Keywords*:** Photorefractive Keratectomy, Marginal Keratitis, Meibomian Gland Dysfunction

Iranian Journal of Ophthalmology 2011;23(1):71-74 © 2011 by the Iranian Society of Ophthalmology

Introduction

Photorefractive keratectomy (PRK) has been used to correct refractive errors. The application of anti-metabolites has decreased the incidence of corneal haze after PRK in recent years.[1] Therefore, many ophthalmologists are interested in performing PRK as their refractive procedure of choice. As any surgical procedure, complications can arise after PRK. Corneal infiltrate is one of the complications of the surgery that must be diagnosed and treated appropriately. Corneal infiltrates after PRK can be infectious or sterile. Sterile infiltrates at and adjacent to the site of the laser ablation caused by the use of topical nonsteroidal anti-inflammatory drugs (NSAIDs), autoimmune process, and blepharitis have been described.[2-5]

Herein, we report a case of bilateral ring-shaped marginal keratitis of presumed sterile etiology after myopic PRK. We believe that recognition of this entity will allow the surgeon to select a conservative approach, avoiding the aggressive treatment.

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Received: August 1, 2010
Accepted: 24 February 2011

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We state that our only interest is academic and we have no financial interest in this publication.

Case report

A 44-year-old man presented to refractive surgery ward of Farabi Eye Hospital for correction of myopic astigmatism in 2009. His past medical and past ocular history was unremarkable. Cycloplegic refraction was -4.75-1.5x180 OD and -4.5-175x165 OS. Best corrected visual acuity (BCVA) was $^{20}/_{20}$ OU. Slit-lamp examination revealed meibomian gland dysfunction bilaterally. The remainder of examination was normal. Ultrasonic pachymetry and Orbscan were within normal limits.

The patient was ordered to have lid scrubs for one week and then underwent bilateral PRK simultaneously. Epithelial removal was performed with use of 20% ethanol and ablation was performed with the Technolas 217Z using Zyoptix Tissue Saving. The ablation profile was OD -4.6-1.5x180 and OS -4.35-1.75x165. Mitomycin C (MMC) 0.02% was applied topically for 20 seconds. Bandage contact lens was placed on ablated cornea at the end of surgery. Chloramphenicol and diclofenac eye drops were started four times a day.

On the first postoperative day, the patient presented with marked lid swelling and conjunctival hyperemia. On slit-lamp examination, there was dense ring-shaped marginal keratitis, bilaterally (Figure 1). A lucid interval was seen between the lesion and limbus. The infiltration was outside the epithelial removed zone. Contact lenses were removed; smear and cultures were obtained bilaterally. Fortified amikacin (20 mg/ml) and vancomycin (50 mg/ml) every 60 minutes were started (with suspicion of bacterial ulcer) in addition to systemic prednisolone 75 mg, preservative free artificial tear every four hour, systemic vitamin C 500 mg four times a day and doxycycline 100 mg twice a day were applied. Work-up started to rule out any systemic or inflammatory disease. As smear and cultures of corneal scraping revealed to be negative, fortified antibiotics were rapidly tapered and replaced by topical ciprofloxacin every four hours at fifth day after surgery (Figure 2). At that time topical steroid (prednisolone acetate), six times a day was added to therapy since the epithelial healing was noted. The epithelium was completely healed by eighth postoperative day and significant decrease in density of infiltration was noted. Therefore, ciprofloxacin was discontinued and systemic steroid was tapered. Vitamin C and doxcycycline were discontinued at the end of third postoperative week. At the end of third postoperative month slit-lamp exam revealed faint peripheral scar and mild haze formation bilaterally (Figure 3).

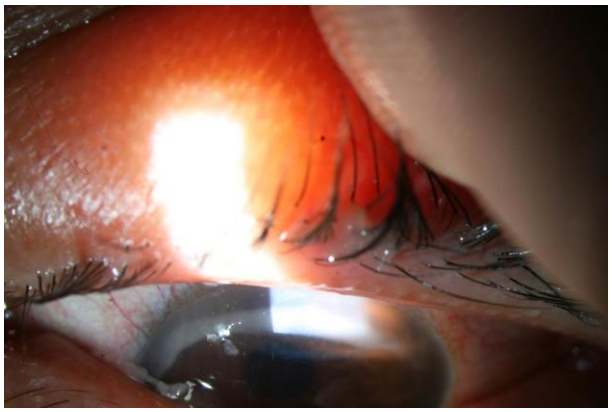

Figure 1. Dense marginal infiltration at first postoperative day

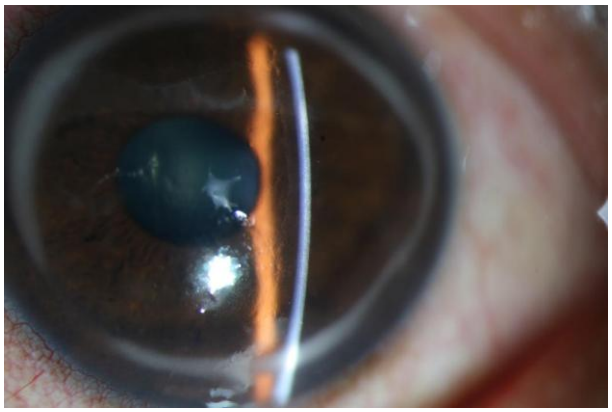

Figure 2. Ring-shaped marginal infiltration at 5th postoperative day

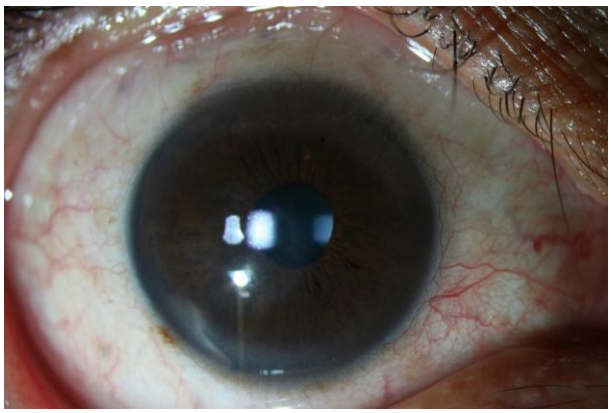

Figure 3. Faint peripheral scar and mild haze formation at third postoperative month

At the final visit; refraction was -0.75-0.25x175 OD and -1-0.25x35 OS with $^{20}/_{20}$ BCVA bilaterally.

Discussion

Peripheral corneal infiltrates after PRK are nonspecific accumulation of inflammatory cells that can be divided in two main categories: infectious keratitis and sterile keratitis.

Infectious etiology seems unlikely in this case because of the immediate bilateral involvement with dense infiltration, presence of infiltration ring outside the epithelial removed zone and negative smear and culture.

Sterile infiltrates have been reported after topical NSAIDs use.[2,3] Block of cyclo-oxygenase pathway of arachidonic acid metabolism by these agents leads to accumulation of lipo-oxygenase pathway products and results in leukocyte accumulation in peripheral cornea. It is believed that these infiltrates are related to the use of topical NSAIDs without concomitant use of topical steroids.[2] In our case, this etiology seems unlikely because of concomitant use of topical steroid.

Peripheral sterile keratitis can be seen after ocular surgery in patients with some types of autoimmune disease such as rheumatoid arthritis[5-7] but work-up for such etiologies was negative in our case.

Contact-lens-induced hypoxia is also thought to contribute to the formation of sterile infiltrates. Again, use of topical steroids, along with NSAIDs has reduced the probability of this entity in our patient.[3]

Blepharitis and meibomian gland dysfunction has been reported as a cause of sterile peripheral infiltration after excimer laser[5,8,9] that can be the case in our patient. Marginal keratitis in patients with blepharitis is usually caused by hypersensivity reaction to toxins produced by bacteria colonizing eyelid margins. It has been proposed that excimer laser treatment of cornea can result in production of cytokines and inflammatory chemokines.[10] Ambrósio et al reported two cases of peripheral corneal infiltration in early postoperative period after laser in situ keratomileusis (LASIK) in patients with moderate to severe chronic meibomian gland dysfunction but the infiltration was seen in peripheral cornea between hours 5 to 7 and not in the shape of a complete ring as in our case. They hypothesized that blepharitis and meibomian gland dysfunction produce an inflammatory condition which predisposes the peripheral cornea to increased cellular migration because of chemokines produced in response to excimer laser.[5]

In addition, Rao et al reported subepithelial corneal infiltrates after PRK resembling staphylococcal-immune infiltrates. The condition responded to treatment with topical diluted steroids and antibiotics as our case.[9]

In summary, incomplete treatment of blepharitis preoperatively (for one week), increased expression of the meibomian secretions with lid manipulation intraoperatively,[9] and good response to steroid therapy are reasons in the favor of immune etiology due to blepharitis in our case.

Conclusion

Our case suggests the role of blepharitis and meibomian gland dysfunction in producing marginal keratitis after excimer laser for which complete preoperative treatment is recommended.

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