POST LASIK INFECTION

Infection occurring after photorefractive keratectomy (PRK) may be

1. Secondary to the defect in the epithelium as well as the use of therapeutic contact lenses. Unlike photorefractive keratectomy (PRK), the integrity of Bowman's membrane and the corneal epithelium is maintained intact after LASIK, hence the risk for microbial keratitis after LASIK is considered lower than other procedures. Despite this, the occurrence of keratitis after LASIK is a reality and numerous case reports testify this.

2. During surgery, the corneal stroma may come into contact with infectious agents coming from the patient’s own body or from contaminants present on the instruments.

3. The surgeon and the operating room may also act as a source.

4. Breaks in the epithelial barrier and excessive surgical manipulation are other risk factors.

5. Other factors in the post-operative period such as delayed postoperative reepithelialization of the cornea, the use of topical steroids and therapeutic
contact lenses as well as the decreased corneal sensitivity and the dry eye situation may all contribute to post LASIK infections.

**CLINICAL SIGNS AND SYMPTOMS**

Infectious keratitis generally presents later than diffuse lamellar keratitis with which it is often confused. It traditionally presents at least 1 week after surgery and often months later. Fungal keratitis usually has a late onset (2 weeks after surgery), though *S. epidermidis* and *Mycobacterium* may also present late.

<table>
<thead>
<tr>
<th>CLINICAL FEATURES</th>
</tr>
</thead>
<tbody>
<tr>
<td>A focal area of infiltrate associated with diffuse or localized inflammation, which may extend throughout the corneal thickness is generally seen. It may extend into the untreated area of the cornea and outside the flap. The flap may begin to melt. There may be</td>
</tr>
</tbody>
</table>

*Figure 4- Corneal ulcer with hypopyon after Lasik*
associated ciliary congestion, secondary iritis, hypopyon and secondary glaucoma. There is loss in best corrected visual acuity (BCVA) as well as uncorrected visual acuity (UCVA).

Infectious post LASIK keratitis has also got to be differentiated from sterile corneal infiltrates which have been described after PRK and LASIK. Sterile infiltrates also present with symptoms similar to infectious keratitis. Subepithelial white infiltrates which may be associated with immune rings are seen in the first few post-operative days. Smears and cultures are negative, and it responds to topical steroids. It may result in stromal scarring and loss of BCVA. Numerous etiologies have been proposed for this including staphylococcal-immune mediation, secondary to the use of topical NSAIDs without concomitant use of topical steroids and contact-lens-induced hypoxia.

**COMPLICATIONS:**

The infection (Fig 4,5) can spread to involve all the layers of the cornea and can cause flap and stromal melting and scarring, AC reaction, hypopyon, secondary glaucoma, anterior and posterior synechiae, irregular astigmatism, loss of BCVA and UCVA etc.
PREVENTION:

It is important to take every possible measure to prevent this sight threatening complication. Preoperative evaluation of the adnexa and the lacrimal apparatus and treatment of any pre-existing condition should become a routine for all patients just as it is for cataract surgery. Some surgeons do advocate performing surgery in only one eye at a time or using completely different sets for the two eyes in case of simultaneous bilateral procedures. It is highly advisable to maintain rigid asepsis throughout the surgical procedure including the use of sterile drapes etc. Good sterilization techniques is a must. can prevent the use of contaminated instruments. Povidone–iodine solution should be used to paint the lids pre-operatively. All fluids applied to the eye before, during, and after LASIK should be sterile as atypical mycobacteria epidemics have been traced to have originated from the use of nonsterile water used to clean instruments or to the ice used during LASIK.
TREATMENT OF POST LASIK KERAUITIS

Early diagnosis and institution of appropriate therapy is of prime importance in the treatment of post LASIK infections. Any focal infiltrate should be considered infectious until proven otherwise. Flap elevation and culturing should be performed as early as possible in all cases where post-LASIK infectious keratitis is suspected. Smears help in deciding on immediate treatment which is then changed according to the culture and sensitivity reports. Polymerase chain reaction testing is also helpful in diagnosis. A corneal biopsy may be required in some cases. Empiric therapy is not helpful as opportunistic and atypical organisms with unusual antimicrobial sensitivities are common and these do not responsive to conventional therapy.

The ASCRS White Paper recommends elevation of the flap, culture, and irrigation of the stromal bed with antibiotic solution (fortified vancomycin 50 mg/mL for rapid-onset keratitis and fortified amikacin 35 mg/mL for delayed-onset keratitis) for all post LASIK infectious keratitis.
For rapid-onset keratitis, it recommends a fourth-generation topical fluoroquinolone such as gatifloxacin 0.3% or moxifloxacin 0.5% given in a loading dose every 5 minutes for 3 doses and then every 30 minutes, alternating with an antimicrobial that is rapidly bacteriocidal and has increased activity against gram-positive organisms, such as fortified cefazolin 50 mg/mL every 30 minutes. In patients working in a hospital environment, with added risk for methicillin-resistant *Staphylococcus aureus* (MRSA), it recommends the substitution of fortified vancomycin 50 mg/mL for cefazolin every 30 minutes to provide more effective therapy against MRSA. Oral doxycycline 100 mg twice a day to inhibit collagenase production and discontinuation of corticosteroids is also advised. Treatment should be modified according to culture and sensitivity reports.

For delayed-onset keratitis, which is commonly due to atypical mycobacteria, nocardia, and fungi, the ASCRS White Paper recommends beginning therapy with amikacin 35 mg/mL every 30 minutes, alternating with a fourth-generation fluoroquinolone (gatifloxacin 0.3% or moxifloxacin 0.5%) every 30 minutes along with oral doxycycline 100 mg twice a day, and discontinuation of corticosteroids.

This treatment is ineffective for fungal infections which often presents late with more extensive keratitis. Appropriate anti-fungal agents should be started and
modified according to sensitivity reports. Fungal infections are often difficult to treat because of the lack of potent antifungal agents, low penetration through intact corneal epithelium, ocular toxicity and decreased solubility. The flap may often need to be amputated, for better penetration of the antifungal agents. In unresponsive cases with extensive involvement of the cornea, a penetrating keratoplasty may often become necessary. The polymerase chain reaction testing can be used to diagnose the causative organism, especially in cases with limited availability of samples. Confocal microscopy can also be made of diagnostic use.

The development of corneal ectasia is a well-recognized complication of LASIK and amongst other contributory factors, unrecognized pre-operative forme fruste keratoconus is also an important one. Patients with this disorder are poor candidates for refractive surgery because of the possibility of exacerbating keratectasia. It is known that posterior corneal elevation $^{1-3}$ is an early presenting sign in keratoconus and hence it is imperative to evaluate posterior corneal curvature (PCC) in every LASIK candidate.