

Delayed Onset Bacterial Keratitis after Implantation of Intrastromal Corneal Ring Segments

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Abstract

The incidence of microbial keratitis after Intacs implantation is extremely low and channel infection has been infrequently reported. We present a 23-year-old woman with keratoconus of both eyes who underwent implantation of two intrastromal corneal ring segments (Intacs, KeraVision, Inc., Fremont, CA, USA) in the right eye. After 2.5 months, the patient presented with decreased vision, inflammation, and stromal infiltration at the lower channel site of Intacs. Culture was positive for *Escherichia coli*. The patient was treated with removing of lower segment of Intacs, and subconjunctival and topical antibiotics. The keratitis resolved, leaving opacity and thinning and mild neovascularization in the lower channel site. Our case report illustrates the risk of microbial keratitis even months after Intacs implantation. It shows need for long-term postoperative attention by both patient and physician.

Keywords: bacterial keratitis, delayed onset, Intacs, keratoconus

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Introduction

Intrastromal corneal ring segments (Intacs) are polymethyl methacrylate segments, placed in semicircular channels between the stromal lamellae at two thirds stromal depth. This device reduces low to moderate myopia and has been proposed to correct keratoconus by flattening central corneal curvature.^{1,2} This procedure has the potential for reversal and adjustment, and leaves the optical center of the cornea untouched.

Infectious complications after Intacs implantation have been infrequently reported, but they are serious and sight threatening when they occur.^{3,4} Infectious usually appear within 3 weeks after surgery.⁵ The incidence of late bacterial keratitis is extremely low.⁵ Herein we report a case of keratoconus who developed unusual bacterial keratitis 2.5

months after undergoing implantation of two Intacs in the right eye. This is an unusual and unique complication of Intacs implantation and as we know this case is the first report of such complication in the literature.

Case Report

A 23-year-old woman presented with keratoconus and intolerance of hard contact lens in both eyes. The left eye was candidate for lamellar keratoplasty due to advanced keratoconus, and right eye for Intacs implantation due to moderate severity of the disease. Before surgery, uncorrected visual acuity (UCVA) of right eye was $\frac{2}{10}$ with manifest refraction; -3.00 – 4.50×40°, and mean keratometry; 47.00 diopters.

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To reduce irregular astigmatism induced by keratoconus and associated myopia, 2 rings were inserted on right eye in July 2005 in the inferior and superior tunnel to flatten the affected area of bulging cornea. Thickness of ring was 0.45 mm inferiorly, and 0.35 mm superiorly. The location of the incision was temporal.

A small corneal incision of about 1.8 mm in length was made 1 mm from the limbus with depth of 70% of the corneal thickness at the incision site. Corneal thickness as measured by ultrasound pachymetry was 455 μm in corneal center and 580 μm at the incision site. The depth of implantation site was 400 μm in our patient. A glide blade was placed into the incision to assess incision length and to verify the adequacy of the pocket. The vacuum centering guide (VCG) was placed along the procedure marker on the cornea. The VCG and procedure marker were aligned with the geometric center of the cornea. The vacuum system was started on low and then shifted to the high setting. The clockwise and counterclockwise separators were used to create the intrastromal tunnels and then the vacuum system was turned off. The VCG was removed and then Intacs (KeraVision, Inc., Fremont, CA, USA) were implanted in the tunnels. The wound was sutured with a 10-0 nylon suture and the knot was buried. Postoperatively, topical ciprofloxacin 0.3% and betamethasone 0.1% were applied four times daily for 2 weeks. The patient was instructed to avoid rubbing her eyes and to use artificial tears frequently. The sutures were removed 6 weeks after surgery.

One week after surgery, UCVA was $8/10$. Manifest refraction was $+1.00-6.75 \times 6^\circ$, and mean keratometry was 45.00 diopters. After one month, UCVA was $8/10$ with manifest refraction; $+1.50-5.50 \times 7^\circ$, and mean keratometry; 45.62 diopters. In slit lamp examination, good wound healing was seen in incision site and locations of Intacs were proper.

Seventy five days after surgery, the patient returned with pain and redness of right eye that was started from 3 days ago. She had not any history of ocular trauma, ocular rubbing, and contact lens usage. Her right eye VA was $1/20$. On slit lamp examination, the right eye had severe injection, infiltration around inferior tunnel, and corneal melting in roof of the

inferior tunnel that caused exposed temporal side of inferior ring, severe anterior chamber reaction with 0.5 mm hypopion (Figure 1).

The patient was hospitalized, and inferior ring was removed and was sent for culture. Corneal scrapings and conjunctival swab were obtained for staining and culture.

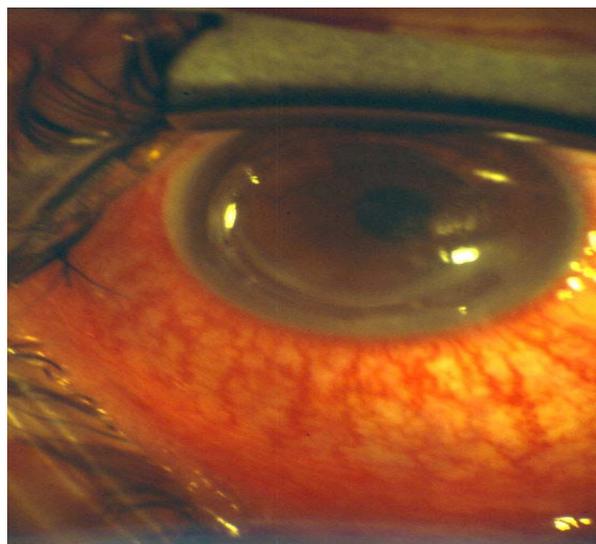


Figure 1. The right eye had severe injection, infiltration around inferior tunnel, and corneal melting in the roof of the inferior tunnel with exposed temporal side of inferior ring.

Subconjunctival injection of gentamicin (40 mg/mL) and vancomycin (50 mg/mL) was performed. The patient was treated with a combination of topical fortified amikacin (20 mg/mL) and vancomycin (25 mg/mL) drops hourly. Smears were negative for bacteria and fungus, but cultures of ring segment and corneal scraping were positive for *Escherichia coli* (*E. coli*) that were sensitive to amikacin and gentamicin in antibiogram. Corneal infiltration showed regression (Figure 2), and the patient was discharged after one week. Fortified antibiotics were continued with lower frequency for 3 weeks.

Six months later, UCVA was $4/10$ and refraction; $-5.50 - 5.00 \times 5^\circ$, and mean keratometry; 49.25 diopters. In slit lamp examination, infiltration was resolved, although corneal thinning, fibrous tissue and mild vascularization were present in the inferior tunnel site (Figure 3).



Figure 2. One week after explanation of Intacs and fortified antibiotic therapy; infiltration decreased. Melting of roof of inferior tunnel and thinning and injection were present.

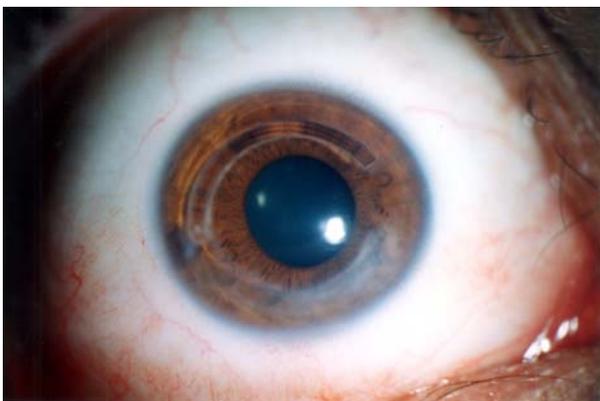


Figure 3. Six months after treatment, corneal thinning, scar, and mild vascularization were present in the inferior tunnel site.

Discussion

Bacterial keratitis after Intacs implantation has been reported infrequently.^{3-5,9} Majority of infections occur within 2 weeks after this procedure, although late bacterial keratitis after laser in situ keratomileusis or incisional keratotomy procedures has been reported up to several years after surgery.⁶

E. coli is a species of coli form bacteria of the family of Enterobacteriaceae. The organisms in this group are all gram-negative rods of moderate size. Members of the family of Enterobacteriaceae are widely distributed in the environment. Although bacilli in the enteric group are part of the normal human flora, they can sometimes be pathogenic. Enterics of ocular significance include *Escherichia*, *Shigella*, *Klebsiella*, *Serratia*, *Proteus*, and *Enterobacter*.⁷ *Escherichia*, *Shigella*, *Klebsiella*, *Proteus*, and *Enterobacter* have been isolated from corneal ulcers.⁸

European Multicenter Study of Intacs reported one case of channel infection in 159 eyes that had Intacs implantation. The infection was observed 3 weeks after Intacs implantation. No microorganism was identified in that case, and the infection resolved with high doses of topical antibiotics.³ Results of phase II and III of U.S. Food and Drug Administration trials report one case of infectious keratitis among 449 eyes with Intacs implantation.⁴

Several mechanisms can help explain the presentation of channel infection after Intacs. Although Intacs implantation does not remove tissue from cornea, this procedure is disruptive to the corneal integrity and potentially exposes the stroma to infectious organisms. The incisional keratotomy to create the channel presents a port of entry for microorganism and puts the wound perpendicular to corneal plan, which tends to heal slowly. The presence of an intrastromal foreign body and postoperative use of topical steroids carry an additional risk for infection.^{5,9}

The explanation for late onset of the infection is more difficult. Because microorganisms were present at the extremity of the stromal channel, they were probably introduced at the time of surgery. Postoperative antibiotics may have delayed the development of infection, which persisted in the deeper layers of the cornea.

We think that once an organism is established in the stromal channel after Intacs implantation, the infection may be extremely difficult to eradicate because the nidus of infection is sequestered from the ocular surface defenses and because the epithelium serves as a relative barrier to penetration of antimicrobial agents. Due to the above reasons, post-operative corneas in Intacs implantation might be considered compromised because of lack of ocular surface protection in the interface.

Conclusion

Our case report illustrates the risk of microbial keratitis even months after Intacs implantation. It shows the need for long-term postoperative follow-up for such procedures. As we know such complication after more than 2 months after implantation of Intacs has not been reported previously.

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