Late bacterial keratitis after implantation of intrastromal corneal ring segments

Tristan Bourcier, MD, PhD, Vincent Borderie, MD, PhD, Laurent Laroche, MD

We report a case of bacterial keratitis that occurred after implantation of intrastromal corneal ring segments (Intacs). The patient presented with decreased vision, inflammation, and stromal infiltrates localized at the extremity of an Intacs channel 3 months after surgery. Culture was positive for Clostridium perfringens and Staphylococcus epidermidis. The infiltrates progressed despite treatment with topical fortified and systemic antibiotics. The Intacs were removed. The keratitis slowly resolved, and the patient recovered a best corrected visual acuity of 20/20.


Intrastromal corneal ring segments (Intacs, Keravision) consist of 2 hexagonal poly(methyl methacrylate) segments, each 150 degrees in arc length. They are placed in the midperipheral cornea between the stromal lamellae in a semicircular channel at two-thirds stromal depth. This device reduces low to moderate myopia and has been proposed to correct keratoconus by flattening the central corneal curvature. This procedure spares the center of the cornea, has the potential for reversal and adjustment, and preserves a prolate corneal shape.

Ocular infection is a serious complication of refractive surgery and may lead to a significant reduction in visual acuity. There are several reports of infections (keratitis or endophthalmitis) after incisional or laser refractive surgery. The incidence of microbial keratitis after Intacs implantation is extremely low, and channel infections have been infrequently reported. Although infections usually appear within 3 weeks after surgery, late bacterial keratitis has not been reported.

We present a patient who developed culture-proven bacterial keratitis 3 months after Intacs implantation.

Case Report

A 32-year-old woman had Intacs implantation in the left eye a few weeks after having successful laser-assisted subepithelial keratectomy in the right eye performed in Finland. The preoperative refractive error was −3.00 diopters in both eyes. The postoperative period was uneventful. The patient was treated with topical antibiotics and corticoid eyedrops for 2 weeks.

Three months after surgery, the patient reported pain in the left eye and she was referred to our department. Refraction in the left eye was −0.75 −0.75 × 90 with a best corrected visual acuity (BCVA) of 20/40. The right eye was plano. Slit-lamp examination of the left eye showed white stromal infiltrates at the extremity of the channel of the temporal ring (Figure 1). There was a small overlying epithelial defect. The anterior chamber showed an inflammatory cellular reaction. A fundus examination was normal. Corneal scrapings were obtained for gram and Giemsa stains and bacterial and fungal cultures. The patient was hospitalized, and treatment was started with fortified ticarcillin (6 mg/mL) combined with fortified gentamicin (15 mg/mL) and vancomycin (50 mg/mL) every hour.

Cultures grew Clostridium perfringens and Staphylococcus epidermidis. Gentamycin eyedrops were replaced by rifampicin because the Staphylococcus was resistant to aminoglycosides. The Intacs were removed 48 hours after admission, the temporal channel was irrigated with balanced salt solution (BSS®) for the collection of microbiological samples and then by rifampicin. The entry of both channels was sutured with 10-0 nylon stitches. Clostridium perfringens and S epidermidis were...
also isolated from the channel. Fortified antibiotics were continued every 2 hours during the first week postoperatively. Subconjunctival injections of dexamethasone (4 mg/day) were performed during the next 3 days.

The anterior chamber reaction resolved after 5 days. Over the next 3 weeks, the corneal infiltrate slowly regressed, leaving a cicatricial opacity at the extremity of the stromal channel (Figure 2). The BCVA was 20/20. The antibiotic eyedrops were discontinued.

Discussion

*Clostridium perfringens* is an anaerobic gram-positive spore-forming bacillus. Like other *Clostridium* species, it is widely distributed in the environment (soil) and may be part of the normal flora of the large bowel. It could transiently contaminate the skin of the perianal area and other skin surfaces. Ocular infections caused by *C perfringens* are rare. They include endogenous postoperative or posttraumatic panophthalmitis and gas gangrene of the orbit, causing rapid destruction of ocular tissues. *Clostridium perfringens* has also been reported as the causative organism in anaerobic keratitis. To our knowledge, there are no previously reported cases of clostridial keratitis after refractive surgery.

Keratitis caused by anaerobic bacteria is infrequently encountered, probably because of the aerobic environment of the cornea. However, in the present case, prior corneal surgery and anaerobic conditions in the stromal channels could have predisposed to the development of the infection. Moreover, keratitis caused by anaerobes is frequently associated with the use of topical corticosteroids. Finally, most deep-seated abscesses and necrotizing lesions involving anaerobes are polymicrobial and may include obligate aerobes such as *Staphylococcus* as concomitant organisms. These microorganisms, acting in concert with trauma or tissue necrosis, lower the oxygen tension and the oxidation-reduction potential in tissues and provide favorable conditions for obligate anaerobes to multiply.

The recent results of a European multicenter study of Intacs reported 1 case of channel infection that was observed 3 weeks after Intacs implantation. No microorganisms were identified in that case, and the infection resolved with high doses of topical antibiotics. The segments were not removed. Results of phase II and III of U.S. Food and Drug Administration trials report 1 case of infectious keratitis among 449 eye with Intacs. Infectious keratitis should be differentiated from deposits of extracellular intrastromal substance, which accumulate in the lamellar channel around the segments. These deposits consist of disorganized convoluted of collagenous lamellae and proteoglycan macromolecules. It should also be differentiated from channel haze caused by the physical separation of stromal lamellae when the lamellae are dissected for placing Intacs.

Several mechanisms can help explain the presentation of channel infection after Intacs. The incisional keratotomy to create the channel puts the wound perpendicular to the corneal plane, which tends to heal
slowly. The presence of an intrastromal foreign body carries an additional risk of postoperative infection because of the possible adhesion of cells, proteins, or microorganisms to the surface of the biomaterial.

Finally, the stroma is exposed during surgery to infectious agents from the eyelids, eyelashes, conjunctiva, drapes, atmosphere, and blade. However, we could not exclude that the temporal segment was implanted in the superficial cornea and not as usual at two-thirds depth. Shallow implantation could thus explain the presence of an epithelial defect in the area of inflammation, and it seems possible that the infection started after breakdown of the epithelium. The explanation for the delayed onset of the infection is more complex. 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 the infection, which persisted in the deeper layers of the cornea. Although most infections are apparent within 2 weeks after refractive surgery, late bacterial keratitis after laser in situ keratomileusis or incisional keratotomy procedures has been reported up to several years after surgery.20

Our case illustrates the risk of microbial keratitis even months after Intacs implantation. It highlights the need for long-term postoperative vigilance by both patient and physician and provides evidence of the reversibility of the procedure even after an infectious complication.

References