LETTER TO THE EDITOR

Acanthamoeba keratitis associated with intracorneal hydrogel inlay

Kératite amibienne associée à un inlay intracornéen

Introduction

Several surgical techniques have been proposed for correcting hyperopia, including laser in situ keratomileusis (LASIK), photorefractive keratectomy, and conductive keratoplasty [1]. Additive refractive keratoplasty refers to a procedure in which a biomaterial (intracorneal inlay or implant) is added to the corneal tissue to modify the optical power of the cornea. This method is potentially reversible as the implant may be removed if necessary. It has been investigated for nearly half a century and it was first suggested by Barraquer in 1949 [2].

Despite the excellent initial tolerance of intracorneal inlay [3,4], severe complications were reported (perilentic deposits, corneal haze, decentration, increase of corneal aberrations, decrease of the optical performance of the cornea, and induced astigmatism) [5–7], hydrogel lenses were soon superseded by laser refractive surgery and their use declined. To the best of our knowledge, infectious keratitis associated with intracorneal hydrogel inlay has not been reported previously. We report a case of Acanthamoeba keratitis associated with intracorneal lens implantation for the correction of low hyperopia.

Case report

In 2007, a 51-year-old healthy woman underwent bilateral intracorneal inlay implantation (Permavision, Anamed Inc., Lake Forest, California) for correction of low hyperopia (+3.75 D in right eye and +3.50 D in left eye). Postoperative uncorrected visual acuity was 20/25 in both eyes.

Figure 1. Slit-lamp examination of right (A, B and C) and left (D) eye. A. Opacities on the inlay surface, anterior stromal and epithelial necrosis, and ulcer over the superior edge of the inlay. B. Corneal section: epithelial ulcer and anterior stroma necrosis. C. Fluorescein staining: ulceration at the superior edge of the inlay. D. Opacities on the inlay surface.
Seven years after surgery, the patient was referred to our Department for decreased vision in right eye, associated with worsening pain and redness over the last 48 hours. She reported projection of superficial foreign bodies (dust) in right eye before the onset of symptoms. On presentation, best-corrected visual acuity (BCVA) was 20/400. Slit lamp examination of right eye revealed opacities on the inlay surface and corneal ulcer with anterior stromal and epithelial necrosis over the superior edge of the inlay (Fig. 1A, B and C). The left eye revealed mild perilimbal corneal opacity (Fig. 1D). Uncorrected visual acuity was 20/25 in left eye. Anterior segment optical coherence tomography (AS-OCT) scans of right eye showed ulceration of the corneal epithelium and anterior stroma over the intracorneal inlay, but no portion of the inlay was directly exposed. The inlay appeared as a hyporeflective structure, localized at a depth of 150 μm in the stroma, surrounded by hyperreflectivity (Fig. 2A and B). In left eye, a thin hyperreflectivity surrounded the hyporeflective inlay while stroma reflectivity appeared normal (Fig. 2C).

Diagnosis of infectious keratitis was assumed. Corneal scrapings were obtained for gram stain, cultures for bacteria and fungi, and polymerase chain reaction (PCR) for Acanthamoeba, Herpes Simplex virus 1 and 2, Varicella Zoster virus, cytomegalovirus, and adenovirus. The patient was hospitalized and intensive treatment was set up with topical ticarcilline 6 mg/mL, gentamycine 15 mg/mL, and vancomycine 50 mg/mL every hour. Cultures and PCR were all negative. Lack of improvement within 48 hours of admission led us to perform in vivo confocal microscopy (Heidelberg Retinal Tomograph III-Rostock Cornea Module®, Heidelberg engineering, Heidelberg, Germany) of the right cornea. Diagnosis of Acanthamoeba keratitis was made on identification of highly reflective, round images with lower reflective borders evoking Acanthamoeba cyst-like images. An intensive specific treatment with topical polyhexamethylene biguanide (PHMB) and desomedin every hour was then introduced and topical antibiotics were lowered to six times a day. The corneal ulcer healed 5 days after introduction of specific treatment. However, 3 months later, BCVA remained...
low (20/200) because of a central corneal opacity. AS-OCT revealed hyperreflectivity around but also within the inlay (Fig. 3A and B). Surgical removal of the intracorneal inlay was performed. As no cleavage plane was found over the inlay, the inlay was removed with the anterior stromal flap and corneal epithelium. Histopathologic analysis showed a thin stromal layer, surrounded by 2 epithelia: one lying on an intact Bowman’s membrane corresponding to the normal corneal epithelium, and another corresponding to an epithelial ingrowth. The hydrogel inlay was not identified on histopathological sections (the inlay was dissolved during histopathologic process) (Fig. 4A and B). Corneal thickness was reduced by 150 μm and a hyperreflective zone remained in the central anterior stroma (Fig. 5A and B). Slit-lamp examination showed corneal haze around the center of the cornea, in the former area of the inlay (Fig. 5C). Two months after inlay removal, the patient’s BCVA was of 20/25 with +6.50 (−1.75 × 115°).

Discussion

Infectious keratitis after intracorneal implantation of hydrogel inlay is an uncommon complication, but as with all biomedical implants, infection remains a risk that should not be ignored. Conversely, several cases and series of infectious keratitis have been reported after intrastromal corneal ring segment (ICRS) implantation for correction of keratoconus or low myopia, since their use is much more common [8—11]. Although most cases occurred in the first postoperative months, other cases occurred later, up to 22 months after surgery [10]. All reported cases concerned bacterial keratitis and no Acanthamoeba keratitis was reported.

In the literature, in many cases of infectious keratitis after ICRS implantation, control of infection required removal of the ICRS, and in the most severe ones, penetrating keratoplasty was necessary [10]. The occurrence of infection after ICRS implantation may be enhanced by trauma or breakdown of the overlying epithelium or

Figure 3. Anterior segment optical coherence tomography scans of right eye (A: horizontal scan, B: vertical scan) after treatment showing hyperreflectivity around and within the inlay and resolution of corneal ulcer. (RTVue©, Optovue Inc, Fremont, CA).

Figure 4. Histopathologic study (A: hematoxylin eosin staining, B: Masson trichrome staining) showing a thin stromal layer surrounded by 2 epithelia: one lying on an intact Bowman’s membrane corresponding to the normal corneal epithelium and another corresponding to an epithelial ingrowth.
extrusion of the ring from the channel [10, 12]. In the present case, late onset of infection 7 years after implantation is too long a time for the Acanthamoeba organism to persist on the implant without causing disease, so it must have gained entry only recently. This may have occurred during the local trivial trauma causing epithelial defect over the intracorneal inlay. By slit lamp examination, AS-OCT and histology, it appears that extensive epithelial ingrowth had developed around the implant. This epithelial ingrowth was present for a long time and was not associated with the trauma, as it was also found in the fellow eye. The epithelial ingrowth may have created an epithelial pathway into the implant space, and may have been the means by which the amoeba gained access to the interior of the cornea. Another explanation for late infection could have been spontaneous melting over the area of epithelial ingrowth and subsequent superinfection unrelated to the recent foreign body trauma. This scenario has been reported after Lasik [13]. Indeed, in the current case, besides the infectious complication, another complication of intracorneal inlay implantation has occurred: the bilateral diffuse perilimbal opacification, corresponding to epithelial ingrowth. Although infection is an infrequent complication after intracorneal inlay implantation, it may be serious and sight-threatening when it occurs. A minor ocular trauma, even long after the initial surgery, may be an access for infection, furthermore facilitated by perilimbal epithelial ingrowth.

Disclosure of interest

The authors declare that they have no conflict of interest concerning this article.

References


Figure 5. A, B. Postoperative anterior segment optical coherence tomography of right eye showing persistent hyperreflectivity in the central anterior stroma (RTVue©, Optovue Inc, Fremont, CA). C. Slit-lamp examination of right eye, 1 month postoperatively showing corneal opacity around the central cornea.
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