Herpes Simplex Keratitis Following Excimer Laser Application

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ABSTRACT

PURPOSE: To report two cases of herpes simplex keratitis following excimer laser application.

METHODS: Two immunocompetent patients with no history of ocular viral infection developed ulcers after LASIK and phototherapeutic keratectomy (PTK), respectively.

RESULTS: Antiviral treatment was administered, and the lesions healed within 14 days.

CONCLUSIONS: These two cases suggest that herpes simplex virus was associated with the use of the excimer laser. [*J Refract Surg.* 2006;22:509-511.]

Herpes simplex keratitis following excimer laser application, such as LASIK,[1-4] photorefractive keratectomy (PRK),[5] and phototherapeutic keratectomy (PTK),[6,7] has been demonstrated in animal experiments but few cases have been reported in humans. We present two cases of herpes simplex keratitis following excimer laser treatment.

CASE REPORTS

CASE 1

A 44-year-old woman presented with injected conjunctiva and decreased best spectacle-corrected visual acuity (BSCVA) of 20/25 in the right eye. Medical history indicated an immunocompetent woman without previous cold sore, blistering rash, atopic disease, dry eye, ocular trauma, inflammation, or ocular virus infection. She denied use of immunosuppressant medication.

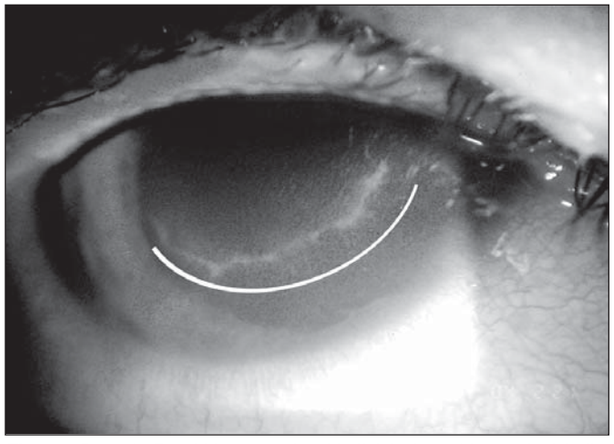

Figure. *Case 1.* Dendritic corneal epithelial lesion with terminal bulbs found at the lower part of the LASIK corneal flap in the right eye using slit-lamp microscopy. Flap margin is indicated by the white curve.

Uneventful LASIK for correction of myopia was performed at a private clinic. A Hansatome (Bausch & Lomb, Rochester, NY) was used to create a superior 8.0-mm hinged, 180-µm thick lamellar flap and the Keracor 117C (Bausch & Lomb) was used for excimer laser corneal ablation. Uncorrected visual acuity (UCVA) was −4.50 diopters (D) in the right eye and −5.00 D in the left eye. One week after LASIK, UCVA was 20/20 in both eyes. Mild topical corticosteroid was used for 2 weeks postoperatively. One month after LASIK, the patient complained of stinging pain and blurred vision in the right eye. Preservative-free artificial tears and prophylactic topical antibiotics were prescribed by the surgeon who performed the procedure for the diagnosis of superficial punctate keratitis associated with postoperative LASIK dry eye. Her symptoms became worse after 3 days of treatment and she was referred for evalution.

Ocular examination revealed a dendritic epithelial ulcer with terminal bulbs at the lower part of the corneal flap of LASIK between 4 and 8 o'clock in the right eye (Fig 1). Herpes simplex keratitis was considered, and 3% acyclovir ointment 5 times per day was prescribed. Preservative-free 0.3% sodium hyaluronate eye drops every 2 hours were given for both eyes. The lesion healed with only mild punctate epithelial defects remaining after 1 week of treatment, but a dendritic corneal ulcer appeared on the LASIK flap between 4 and 6 o'clock in the left eye. Polymerase chain reaction test of tear samples of both eyes revealed herpes simplex virus DNA. Topical acyclovir for both eyes, as

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well as oral acyclovir 200 mg 5 times a day was prescribed for 10 days following appearance of the second ulcer. The dendritic lesion in the left eye healed after 1 week of treatment. Best spectacle-corrected visual acuity was 20/20 in both eyes 1 month later. At 1-year follow-up, there was no evidence of herpes simplex keratitis recurrence.

CASE 2

A 76-year-old immunocompetent woman was referred due to pain in the left eye of 4 months' duration. She had a history of glaucoma in both eyes and underwent trabeculectomy and cataract surgery in the left eye 10 years prior to presentation. Macular edema developed 5 years postoperatively and she underwent unsuccessful grid laser photocoagulation. There was no history of cold sore, blistering rash, atopic disease, dry eye, ocular trauma, inflammation, or ocular viral infection. She denied use of immunosuppressant medications.

Ocular examination revealed severe corneal edema with bullous formation and cornea epithelial defect in the left eye. Best spectacle-corrected visual acuity in the left eye was count-fingers at 30 cm. The diagnosis was pseudophakic bullous keratopathy with recurrent corneal erosion. Phototherapeutic keratectomy was performed, in addition to therapeutic contact lens treatment, to relieve recurrent corneal epithelium erosion. Chloramphenical 0.25% eye drops 4 times a day and preservative-free lubricant every 2 hours were prescribed to the left eye postoperatively. Three weeks after PTK, the patient presented with irritation and conjunctival injection in the left eye. Slit-lamp microscopy revealed a dendritic epithelial ulcer with terminal bulbs at the paracentral cornea between 5 and 9 o'clock in the left eye. Polymerase chain reaction test of the tear sample of the left eye revealed herpes simplex virus DNA. Herpes simplex keratitis following PTK was considered and 3% acyclovir ointment 5 times a day was prescribed immediately. After 1 week of treatment, the corneal ulcer healed and only mild superficial punctate keratitis was seen. During 6-month follow-up, herpes simplex keratitis did not recur.

DISCUSSION

In the cases presented, typical dendritic lesions of herpes simplex keratitis occurred within 1 month after excimer laser surgery. Polymerase chain reaction test demonstrated herpes simplex virus DNA in tear samples of the lesion eyes. Symptomotology worsened following preservative-free lubricant treatment and resolved with antiviral treatment. Thus, herpes simplex keratitis associated with LASIK and PTK procedures was suggested.

Many people without clinical herpes simplex infection have latent virus. Worldwide, 60% to 90% of the adult population is positive for HSV-1 antibody and only 1% to 6% of primary infections are clinically recognized.[8] Asymptomatic people can shed herpes simplex virus in tears.[8] This report suggests that herpes simplex virus infection in individuals without clinical signs and symptoms was activated by excimer laser surgery.

The association of excimer laser corneal ablation and activation of herpes simplex virus has been demonstrated in rabbits and mice,[4,6] but only a few human cases of herpes simplex keratitis associated with LASIK and PRK have been reported.[1-3,5] These patients had a history of herpes simplex keratitis, systemic herpes simplex virus infection, or multiple corneal surgeries before PRK was performed.[1-3] Some patients were treated with bilateral LASIK, but postoperative herpes simplex keratitits occurred only in one eye. We present the first case with bilateral herpes simplex keratitis after bilateral LASIK without previous corneal surgery or history of herpes simplex.

Systemic prophylactic antiviral agents in patients with a history of herpes simplex keratitis before PRK or LASIK has been suggested.[1,3,5,9] Factors such as preoperative emotional stress, postoperative tear dysfunction, ultraviolet radiation exposure, and surgical trauma (eg, lamellar keratoplasty) have been related to reactivation of herpes simplex virus. Further, herpes simplex keratitis may be secondary to postoperative use of topical corticosteroid (case 1). Although few cases have been reported, the relationship between herpes simplex keratitis and LASIK, PRK, or PTK cannot be excluded and patients should be informed of this possibility before surgery.

We report a case of herpes simplex keratitis associated with bilateral LASIK in a healthy woman and a patient with herpes simplex keratitis after PTK for treatment of pseudophakic bullous keratopathy. We suggest the importance of obtaining a detailed patient history before surgery and administering prophylactic antiviral treatment in patients with previous herpes simplex virus infection.

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Dry Eye After Photorefractive Keratectomy With Adjuvant Mitomycin C

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ABSTRACT

PURPOSE: To report a patient with dry eye after bilateral photorefractive keratectomy (PRK) with mitomycin C treatment in one eye.

METHODS: A 29-year-old woman underwent PRK for moderate myopia. The left eye was randomly assigned and intraoperative topical mitomycin C was administered. The right (control) eye was treated with intraoperative corticosteroid only.

RESULTS: The patient developed dry eye symptoms and superficial punctuate keratopathy in the eye treated with mitomycin C. Fifteen months after surgery no improvement was noted.

CONCLUSIONS: Photorefractive keratectomy with mitomycin C treatment could induce or exacerbate dry eye. [*J Refract Surg.* 2006;22:511-513.]

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During the past several years, changes have been made in refractive surgery trends. Although LASIK is a widely used treatment technique, there is increasing interest in excimer surface ablation procedures such as photorefractive keratectomy (PRK), laser subepithelial keratomileusis (LASEK), and epi-LASIK.

Recently, encouraging results have been reported in reducing haze after PRK by administering a single intraoperative application of mitomycin C.[1] These studies suggest that the prophylactic use of a diluted mitomycin C 0.02% solution produced lower haze rates, better uncorrected visual acuity (UCVA) and best spectacle-corrected visual acuity (BSCVA) results, and more accurate refractive outcomes than those achieved in the control group.[1,2] Despite these results, there is considerable evidence of mitomycin C toxicity in the literature and concern is increasing regarding the side effects and long-term complications.[3,4] Although, mitomycin C use in refractive surgery differs from pterygium and glaucoma surgery, there is increased awareness of its possible complications. We report a patient who developed dry eye symptoms in the left eye after PRK with mitomycin C application, whereas the fellow eye treated with PRK and corticosteroids did not develop similar symptoms.

CASE REPORT

A 29-year-old woman with no history of dry eye underwent bilateral PRK for moderate myopia. Preoperative examination was normal. She was informed of possible intra- and postoperative complications, and was included in a prospective, double-masked, randomized clinical trial for the safety and efficacy of topical mitomycin C use after PRK in myopic eyes. One eye was randomly assigned to receive intraoperative topical mitomycin C application. Randomization was performed by opening a sealed envelope that contained the treatment information. The patient gave written informed consent in accordance with institutional guidelines and the Declaration of Helsinki. Preoperative corneal sensitivity (Cochet-Bonnet esthesiometer) and Schirmer tests were not performed, as they were not included in the initial study protocol design.

Preoperative corneal thickness was 500 µm in both eyes. Epithelium was removed using a rotating soft brush at the 8-mm zone. Ablation was performed using the Allegretto Wavelight (WaveLight AG Technologie, Erlangen, Germany) 200 Hz excimer laser system. Attempted correction was -5.50 -0.50×170 in the left eye and -5.00 -1.00×180 in the right eye (mitomycin C eye and control eye, respectively). Immediately after laser treatment, the left eye received a single topi-

cal application of mitomycin C 0.02% diluted in balanced salt solution. This was administered by placing a mitomycin C–soaked sponge (7 mm in diameter) over the ablated stroma. The sponge was kept in place for 2 minutes, then the corneal surface and entire conjunctival fornix were vigorously irrigated with 20 cc of balanced salt solution to remove residual mitomycin C. The right eye received a sponge soaked in balanced salt solution applied in the same manner. The procedure was completed without complication. At the end of surgery, a combination of steroids and antibiotic drops (Tobradex; SA Alcon, Couvreur NV, Belgium) was administered in both eyes 4 times daily, and a bandage soft contact lens was applied until full corneal re-epithelialization occurred. After re-epithelialization, the left eye received a placebo solution (artificial tears) whereas the control eye was treated with fluorometholone sodium 2% (FML; Allergan, Westport, County Mayo, Ireland) 4 times daily for 2 weeks. The dosage of corticosteroid and placebo was tapered one drop every second week. No additional steroids were used during follow-up.

The postoperative period was uneventful. At 1 month, the patient reported dryness in both eyes treated with frequent preservative-free artificial tears. Uncorrected visual acuity (UCVA) was 20/32 in both eyes. Examination revealed mild superficial punctate keratopathy of both corneas. Three months after surgery, the patient reported that the sensation of dryness improved in the control eye but remained stable in the mitomycin C eye. Superficial punctuate keratopathy did not improve in the mitomycin C eye whereas no evidence was noted in the control eye, except for mild haze. Uncorrected visual acuity was 20/20 in both eyes. Fifteen months postoperatively, UCVA was 20/20 in both eyes with an improvement in haze (trace) formation in the control eye. Slit-lamp microscopy of the left eye revealed no improvement of superficial punctate keratopathy and the patient reported ocular irritation and foreign body sensation. Tear film status tests (Schirmer test without anesthetic eyedrops [Schirmer I] and with anesthetic eyedrops [Schirmer II]) were performed, revealing an excessive decrease in tear secretion in the left eye (Schirmer I and II, 3 mm and 2 mm, respectively) in comparison with the control eye (Schirmer I and II, 8 mm and 6 mm, respectively). Preservative-free artificial tears (Vismed; TRB Chemedica AG, Haar/München, Germany) and ointment (Dacrio Gel, SA Alcon) were administered.

DISCUSSION

Photorefractive keratectomy can induce or exacerbate dry eye after surgery. The cause involves decreased corneal sensation, resulting in reduced tear production. In this report, because preoperative measurement of tear flow was not available, the presentation of increased dry eye symptoms and postoperative decreased tear flow confirmed by Schirmer tests, which were worse in the left eye compared to the control eye, indicates that adjuvant mitomycin C application in PRK may affect tear production. A possible explanation of this complication may be the increased extent and duration of corneal hypesthesia associated with mitomycin C application, in addition to the PRK ablation damage to the corneal nerve plexus, which did not resolve after 15 months of follow-up (nerve plexus usually recovers completely after 12 months[5]).

Furthermore, a direct decrease in aqueous tear production, increased tear osmolarity and evaporation, and toxic effect in lacrimal glands and goblet cell density[6] could be additional factors that contribute to mitomycin C–induced dry eye. We suggest that the adverse effect of mitomycin C to the surrounding tissue could be minimized by eliminating application to the ablation site. Surgical experience with LASEK indicates it is feasible to expose only part of the cornea to a liquid substance (ie, alcohol). Similarly, irrigation could be performed using a cone- or cylinder-like container that seals against the corneal surface and uses an aspiration port to safely remove the excessive mitomycin C from the corneal surface.

It is possible that all of these explanations are partly correct, and each may contribute to the symptoms experienced postoperatively. Although dry eye could be more common and more severe in patients with preexisting tear flow abnormality, preoperative evaluation of tear film status is necessary to identify patients with tear film deficiency.

Photorefractive keratectomy with mitomycin C application may induce tear deficiency or exacerbate preexisting dry eye disease. This side effect can be present up to 15 months after surgery. Candidates for surgery should be screened preoperatively so that patients with borderline tear secretion can be advised of possible mitomycin C–related side effects.

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Decentration and Cataract Formation 10 Years Following Posterior Chamber Silicone Phakic Intraocular Lens Implantation

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ABSTRACT

PURPOSE: To report a 10-year follow-up for bilateral implantation of a Chiron Adatomed silicone posterior chamber phakic intraocular lens (PIOL).

METHODS: A 32-year-old man presented with bilateral blurred vision and monocular diplopia in the left eye of 2 years' duration.

RESULTS: Slit-lamp microscopy showed bilateral anterior subcapsular cataract and temporal PIOL decentration, and no visible space between the PIOL and crystalline lens in the right eye. After explantation of the posterior chamber PIOL, lens aspiration, and IOL implantation, uncorrected visual acuity improved to 20/15 in the right eye. Scanning electron microscopy examination showed denser deposits on the central portion of the back surface when compared with the edges.

CONCLUSIONS: Long-term follow up of certain designs of posterior chamber PIOLs may reveal late occurrence of complications. Cataract formation may be related to direct contact between the implanted and crystalline lenses. [*J Refract Surg.* 2006;22:513-515.]

In 1986, Fyodorov introduced the idea of implanting a negative silicone intraocular lens (IOL) in the posterior chamber, just anterior to the surface of the crystalline lens for the correction of myopia in phakic patients.[1,2] Chiron Adatomed GmbH (Ratingen, Germany) produced another silicone posterior chamber phakic intraocular lens (PIOL) in 1992 that was first implanted in Germany.[2,3] Since then, various designs of silicone myopic PIOLs have been reported— Fyodorov, Russian plate (Mikof Company, Russia),[1] and top-hat style.[3,4] However, production of these lenses was soon abandoned because of high complication rate.[2]

One hundred twenty-five implanted silicone-generation Chiron Adatomed posterior chamber PIOLs have been reported in three studies.[3,5,6] We report the longest follow-up for this design.

CASE REPORT

A 32-year-old man presented with complaints of bilateral gradual blurring of vision and monocular diplopia in the left eye of 2 years' duration. Ten years earlier, he had bilateral implantation of posterior chamber PIOLs for the correction of high myopia. He reported no history of ocular disease.

Uncorrected visual acuity (UCVA) was 20/30 in the right eye and 20/40 in the left eye, improving with pinhole to 20/25. Refraction was $+0.25\ -1.50 \times 91$ in the right eye and $-2.27\ -1.37 \times 62$ in the left eye. Intraocular pressure was normal in both eyes. Slit-lamp microscopy showed bilateral superior limbal conjunctival scarring, patent bilateral superior surgical peripheral iridectomy, and bilateral anterior subcapsular opacification. When compared with the left eye, the right eye had denser cataract, no visible space between the PIOL and crystalline lens, and no visible nasal optical edge prior to dilation. After dilation, moderate temporal decentration was seen in the left eye. Funduscopy revealed bilateral myopic changes and a cup-to-disc ratio of 0.5. Axial length was 26.58 mm in the right eye and 26.26 mm in the left eye.

The Chiron Adatomed PIOL was explanted from the right eye using a 5.5-mm clear corneal temporal approach in which it was dislocated into the anterior chamber, followed by lens aspiration using a Sovereign system with whiteStar power modulation (Advanced Medical Optics, Santa Ana, Calif) . It was a single, boat-shaped piece with an overall diameter of 12.0 mm. The optic had a biconcave configuration with a diameter of 5.25 mm. Scanning electron microscopy examination showed diffuse deposits of unknown origin over the entire front surface. It was denser on the central portion of the back surface when compared with the edges (Figs 1 and 2). A +10.0-diopter (D), foldable, three-piece AcrySof posterior chamber IOL (MA60BM; Alcon Laboratories Inc, Ft Worth, Tex), optic diameter 6.0 mm,

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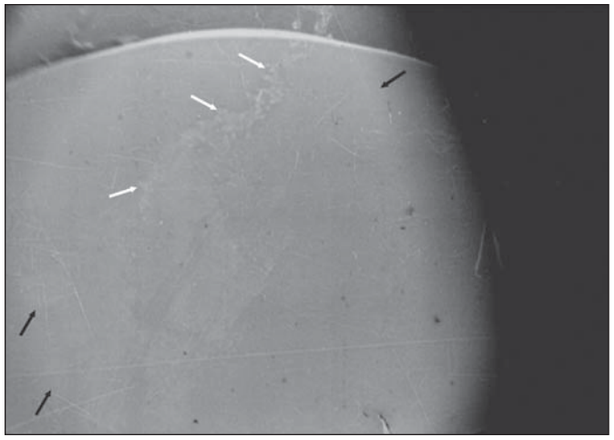

Figure 1. Explanted IOL (scanning electron microscopy, original magnification ×11) with diffuse opacities on the front surface (white arrows). These opacities were denser at the area of contact between the concave back surface of the IOL and crystalline lens centrally (black arrows). Two surgically induced cracks during explantation are located at the inferior margin.

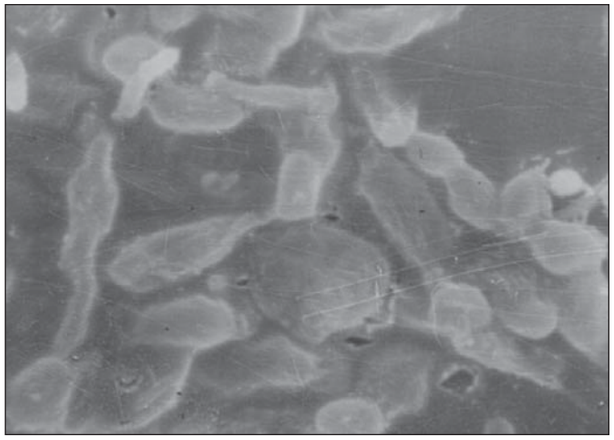

Figure 2. Higher magnification of the abnormal opacity deposits (scanning electron microscopy, original magnification ×5500). These deposits are of unknown etiology and may represent dead cells or fiber accumulation.

was implanted. The Holladay formula[7] was used to calculate IOL power, aiming for emmetropia. At 2 months postoperatively, UCVA improved to 20/15 and refraction was +0.25 −3.0 × 96.

DISCUSSION

The main advantages of posterior chamber PIOL use for correction of high myopia are that surgery is relatively simple, reversible, carries no risk of corneal endothelium contact, and does not depend on the vagaries of corneal wound healing or sacrifice of the crystalline lens and its accommodative ability.[8,9]

The case reported demonstrates three unique findings. First, it presents the longest follow-up (10 years) for the Chiron Adatomed IOL design compared to other studies in which follow-up ranged from 6 to 32 months (mean 17.2 months)[3] and 3 to 24 months.[5,6] The longest follow-up reported for older-design silicone-generation PIOLs was 7 years.[4] Second, it demonstrates late occurrence of cataract formation 10 years postoperatively when compared with the Fechner et al[3] and Brauweiler et al[6] reports. In the former, the incidence of cataract was 18% and onset ranged from 12 to 24 months postoperatively, whereas in the latter, the incidence was 81.1% with onset ranging from 3 to 24 months. Third, it highlights the first scanning electron microscopy examination of an explanted Chiron Adatomed IOL.

Several potential pathophysiological mechanisms for cataractogenesis have been proposed. The fact that an anterior subcapsular cataract is the most dominant pattern of cataract in all cases, and the observation of its formation as early as 3 months postoperatively in two patients of the Brauweiler et al series,[6] would strongly suggest direct trauma to the crystalline lens as a mechanism of cataractogenesis. Fechner et al[3] observed that the space between the IOL posterior surface and crystalline lens, defined as the lens vault, may be advantageous, as none of the opacified crystalline lenses had a visible space. In contrast, the crystalline lens in eyes with forward-buckled IOLs have remained clear. In 1999, Fechner stated that the Chiron Adatomed IOL used in his study was not constructed to provide enough space.[10] In fact, 58% of 40 eyes reported had implantations of small IOLs ⩾0.5 mm longer than the horizontal corneal diameter white-to-white distance.[3] Others claim the predictable measurement of the posterior chamber width is the ciliary sulcus diameter rather than the white-to-white distance because the latter has an irregular contour causing internal diameter variations and may be obtained using variable techniques (surgical caliber versus topography-based calibers).[10] Furthermore, small optical diameter was claimed by Wiechens to induce cataract 7 years after implanting a top-hat style IOL. Thus, constant or intermittent contact from increased crystalline lens curvature, either during accommodation or gradual enlargement of anterior-posterior lens diameter, could explain cataract formation.[3] This rationale was supported by the finding of a circular contact zone of an acellular substance on the back surface of IOLs by scanning electron microscopy.[4] Fechner recommended altering the lens vault by implanting oversized IOLs (adding 1.0 mm to the white-to-white diameter), rationalizing that a larger IOL will be forced to buckle forward, thereby increasing the lens vault, or by shortening the posterior IOL surface

radius to lift the center away from the anterior surface of the crystalline lens.[3,10]

In contrast, the report by Marinho et al[5] did not support the hypothesis of a short IOL diameter causing cataract formation as none of their patients developed cataract up to 24 months of follow-up, although 13% had IOL implantations 1.0 mm shorter than the horizontal white-to-white diameter. However, this study reported shorter follow-up (86.8% and 76.5% eyes, mean ≤12 and ≤6 months, respectively)[5] when compared with the report by Fechner et al (15.9% eyes, mean ≤6 months).[3]

Another mechanism of cataractogenesis is that a posterior chamber PIOL can induce metabolic changes in the crystalline lens by altering its oxygen transport and nutrition transmission, either through a decrease in the lens vault from constant or intermittent trauma or through subclinical inflammation.[2,9,11] However, the composition of new generation IOLs has undergone change, incorporating new materials, such as collagen, to increase their hydrophilia, oxygen permeability, and biocompatibility.[8] Four designs of IOLs (ICM V1 to V4 and ICH V1 to V4, minus and plus power, respectively) have been developed by STAAR Surgical AG (Nidau, Switzerland/Monrovia, Calif). Variation in the incidence of cataract formation following implantation may be due to deviation in definition of cataract or opacity, follow-up period, surgical technique, and lens design.[12] The highest incidence (16.6%) was observed with a non-vaulted flat V3 design. It is no longer used and is replaced by a better vaulted design, V4. Cataracts were of non-progressive, anterior subcapsular type with the V4 and were more likely to be associated with age >50 years, progressive corneal endothelial cell loss, intraoperative trauma, and low-central implantable contact lens vault.[8,9] Chiron Adatomed IOL decentration has been reported in only one series (5.8%) necessitating explantation of the small diameter (10.5 mm) IOL.[3] Combined optic decentration and cataract formation in our patient strongly suggests a slightly undersized IOL diameter, relative to the white-to-white diameter, was implanted.

Our findings of late complication occurrence support long-term follow-up in patients with PIOLs. Use of ultrasound biomicroscopy or optical coherence tomography is helpful in choosing the appropriate IOL size to reduce or eliminate complications.

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