

Lens vacuoles during phakic intraocular lens implantation

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ABSTRACT: Lens damage during phakic intraocular lens implantation is a major complication. A case of a patient who developed lens vacuoles during toric implantable collamer lens (ICL) removal from her right eye and insertion of a new non-toric ICL due to persistent rotation of the former is presented in this work. As this event is very uncommon when implanting phakic intraocular lenses, the performed surgical procedure, the appearance of the vacuoles and the hypothesis on the origin of the vacuoles is described.

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Refractive surgery with phakic intraocular lenses (pIOL) is a solution for young patients with clear lenses. The development of significant lens opacities after the insertion of these lenses are a complication that should be kept in mind. The incidence of cataractogenesis described after the implantation of posterior chamber pIOL ranges between 25.7% and 3.6% depending on the model of pIOL¹. The suggested etiology for the development of cataracts are: intermittent contact between pIOL and the lens, operative trauma, insufficient lens nutrition and chronic subclinical inflammation. Anterior subcapsular cataracts are the most common type of cataract. These cataracts may either remain stable over time or lead to a reduction in visual acuity, and as such, these patients should undergo closer follow-up².

The present work describes the case of a patient scheduled for the removal of a toric implantable collamer lens (ICL) from her right eye and the insertion of a new non-toric ICL due to the persistent rotation of the former. Reposition in accordance with the correct astigmatism axis was attempted twice, however, after both procedures, the ICL appeared to be misaligned in the days following. During the fourth surgical procedure, when the toric

ICL was removed, vacuoles appeared on the surface of the patient's lens. The surgical procedure performed and the evolution of the vacuoles is also described.

CASE REPORT

A 35-year-old caucasian female with no previous medical nor ocular history of interest received a consult for refractive correction. Best-corrected visual acuity was 0 logMAR in her right eye and 0.09 in her left eye, with a manifest refraction of $-4.25 -2.25 \times 5^\circ$ and $-6 -1.75 \times 180^\circ$ right and left eye respectively. Corneal topography (WaveLight® Oculyzer™ II, Alcon Cusi, S.A., El Masnou, Spain) showed regular astigmatism, with no signs of corneal ectasia, and included normal Belin-Ambrosio Enhanced Ectasia Display. In both eyes the pupil diameter was 5.5 mm, the anterior chamber depth was 3.4 mm, the central corneal thickness was 556 microns (right eye) and 550 microns (left eye) with a white to white distance of 11.5 mm. No endothelial defects were found using specular microscopy and the lens was clear in both eyes.

The inserted lens was an ICL (Visian® ICL toric V4c design, Staar Surgical Co, Monrovia, California, USA) measuring 12.6 mm with an expected residual spherical equivalent of +0.18 diopters. The recommended axis on which the lens should be positioned was 0-180 degrees.

The surgical procedure and postoperative period in the left eye of the patient was uneventful. The following events refer only to the right eye.

The surgical procedure was as follows:

Under topical anesthesia after pupil dilatation, the surgeon (LAR) performed a paracentesis of 1 mm through which a cohesive viscoelastic solution

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(PROVISC® Solution, sodium hyaluronate 10 mg/ml, Alcon Cusi, S.A., El Masnou, Barcelona, Spain) was injected and a 3.2 mm valved clear-corneal temporal incision was then made. The toric ICL was inserted using the ICL injector (MSI-TF MicroSTAAR injector, Staar Surgical Co, Monrovia, California, USA) and positioned on the correct axis as recommended in the surgical plan supplied by the lens provider. After that, the viscoelastic solution was removed using an irrigation/aspiration probe connected to an INFINITI® Vision System (Alcon Cusi, S.A., El Masnou, Spain) and acetylcholine 1% was injected. After hydration of the corneal incisions, the final step was the injection of 0.05 ml of moxifloxacin 0.5% (VIGAMOX® Solution, Alcon Cusi, S.A., El Masnou, Spain) and 0.05 ml of actocortine 125 mg/ml. Postoperatively, antibiotic and steroid drops were prescribed.

No incidents were recorded during the procedure however the epicrystalline intraocular lens underwent rotation that required reorientation of the lens in the operation room on two separate occasions - at two days, and then ten days after the first procedure.

The same procedure was performed on both occasions in order to rotate the intraocular collamer lens. Under topical anesthesia, the surgeon (LAR) injected 0.1 ml of bupivacaine 0.50% plus adrenaline 1/200,000 in the anterior chamber with a 32 gauge needle, repositioning the lens with the tip of the needle. At the end of the procedure, acetylcholine, followed by antibiotic and steroids, were injected into the anterior chamber.

As the ICL rotated again after the second realignment, the decision was made to remove the lens and address the refractive error with a combined procedure: the insertion of a spherical ICL and the correction of the residual astigmatism using photorefractive keratectomy (PRK).

The removal of the toric ICL and the implantation of the new spherical ICL was performed in a single procedure under topical anesthesia. After pupil dilatation, the surgeon used a blunt cannula to reopen the temporal corneal incision and filled the anterior chamber with chondroitin sulphate 40 mg/ml + sodium hyaluronate (VISCOAT®, Alcon Cusi, S.A., El Masnou, Spain). The extraction of the toric ICL and the insertion of the new spherical ICL was performed without any kind of contact between the ICLs nor the surgical instruments with the cornea or the patient's lens, but once the viscoelastic solution was removed, we observed the presence of several small vacuoles on the crystalline surface (Figure 1). These vacuoles were located mainly at the periphery of the lens, but some of them were close to the visual axis. The procedure ended with the injection of acetylcholine 1%, antibiotics and steroids as described previously, and the hydration of the corneal incisions.

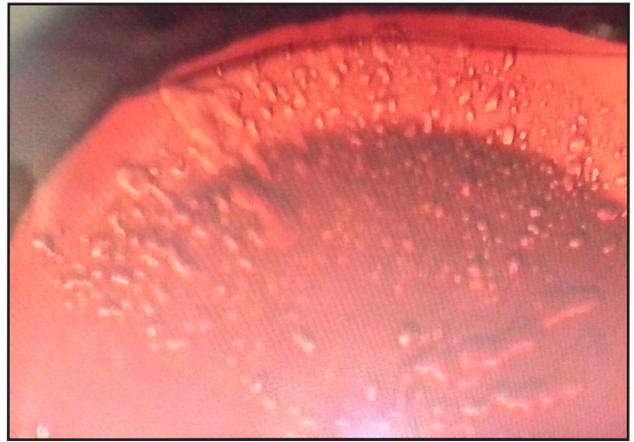


Figure 1. Lens vacuoles observed intraoperatively after the extraction of the ophthalmic viscosurgical device.

Twenty-four hours later the patient came in for evaluation and her vision was found to be 0.09 logMAR with mild corneal edema and refraction of $-0.75 \times 180^\circ$. The vacuoles persisted, however they did not affect the visual axis, as had been observed the day before.

A week later the vacuoles could be seen only after pupillary dilation (Figure 2) and visual acuity was 0.04 logMAR with the following refraction: $+0.50 -4.25 \times 180^\circ$.

A month after surgery the vacuoles remained stable, with a visual acuity of 0.04 logMAR with a refraction of $+0.75 -3 \times 180^\circ$. At this point she was scheduled for LASIK correction of the residual astigmatism.

Four months after the first procedure and three months after the LASIK was performed, the patient's visual acuity was 0.04 logMAR with no residual refraction. As we can observe in Figure 3, most of the vacuoles disappeared during these four months, and they did not influence the final visual outcome.

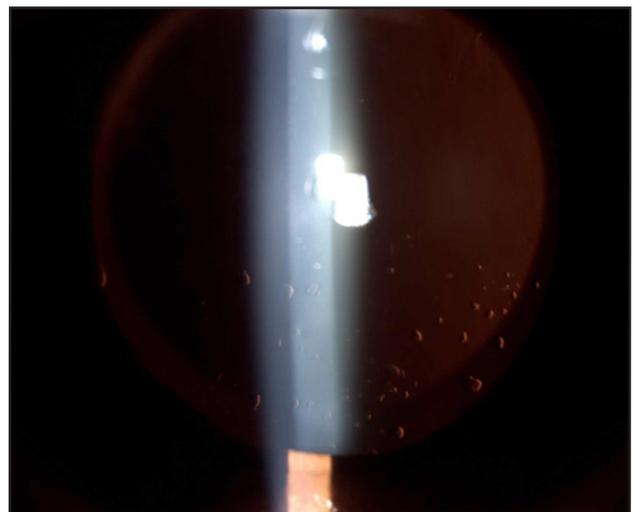


Figure 2. Lens vacuoles observed one week after the surgical procedure using biomicroscope.

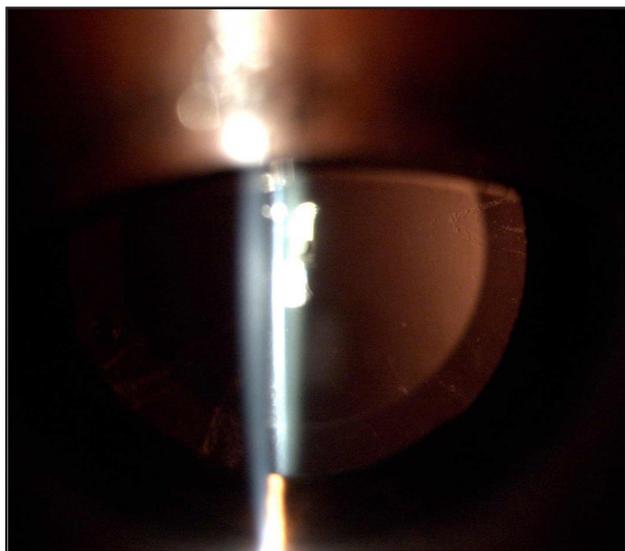


Figure 3. Lens image using biomicroscope four months after the vacuoles developed.

DISCUSSION

The spectrum of complications after the implantation of posterior chamber phakic intraocular lenses ranges from endothelial cell loss to retinal detachment, as has been described in other works². The progressive development of lens opacities (cataractogenesis) is one of these complications and are a stable phenomenon, with little impact on visual acuity, rarely require surgery, and develop after surgery. The vacuoles that were observed in our case, appeared intraoperatively and disappeared during follow up, and therefore their features did not match those of cataracts which developed after the insertion of an ICL.

To the best of our knowledge, this is the first published case on lens vacuoles which developed during the insertion of an ICL.

The surgical procedures performed were highly reproducible, all uneventful, and the only element which distinguished the last surgery (where the vacuoles were observed) from the others was the use of VISCOAT®. As a result, we consider that this is the reason for the development of the lens alteration.

Although the toxicity of VISCOAT® on lens epithelial cells has been described by Budo et al.³ and Khaled et al.⁴, their findings are limited to light or electron microscope view during capsulorhexis. Despite this fact, VISCOAT®

is known to be a good endothelium protector and for this reason we decided on its use in the ICL exchange in order to attempt to protect the intraocular structures that had already been subjected to three recent procedures. The development of lens vacuoles was highly unexpected, although fortunately they proved to resolve spontaneously and did not affect visual function.

Although we highly discourage our colleagues from using VISCOAT® for phakic intraocular procedures, even in difficult cases such as this one, the behavior of these vacuoles in the present case was benign.

We are not able to explain the mechanism by which the lens vacuoles disappeared, but in our opinion, it may be related to the lens epithelial regeneration phenomena described by Saika et al.⁵ who described lens capsule structure regeneration after cataract surgery in rabbits.

As we were not able to find similar publications which included a greater number of eyes, other etiological mechanisms related to repeated surgery that lead to epithelial lens dysfunction should not be dismissed (i.e. epithelial oedema secondary to repeated irrigation/aspiration maneuvers).

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