

Case report

Transient crystalline lens deposits following the insertion of a phakic sulcus-fixated collamer intraocular lens in a hyperopic eye



Lama El Khatib^a, Ahmad K. Hatoum^b, Hassan M. Moukhadder^a, Nasrine Anais El Salloukh^a, Shady T. Awwad^{a,*}

^a Department of Ophthalmology, American University of Beirut Medical Center, Beirut, Lebanon

^b Department of Ophthalmology, Jordan University of Science and Technology, Irbid, Jordan

ARTICLE INFO

Keywords:

ICL
Implantable collamer lens
Deposits
Crystalline lens
Iridocyclitis

ABSTRACT

Purpose: The purpose of this study was to report crystalline lens deposit formation following ICL implantation for the correction of hyperopia.

Observations: A 23-year-old male presented at the American University of Beirut Medical Center in 2008 seeking refractive surgery for hyperopia. His cycloplegic refraction was $+7-1.25 \times 115^\circ$ and $+7-1.00 \times 115^\circ$ in the right and left eyes, respectively, yielding a vision of 20/20 bilaterally. The patient underwent right eye insertion of a non-toric phakic sulcus-fixated collamer lens 2 weeks after undergoing peripheral iridotomies. The early postoperative course was complicated by anterior chamber inflammation and the appearance of diffuse whitish precipitates on the anterior surface of the crystalline lens, hypotony, and a mid-dilated mildly reactive pupil. With the prompt administration of topical and systemic steroids, the anterior chamber reaction subsided, and the anterior capsular deposits gradually resolved peripherally with some remaining centrally over the course of several weeks. The patient's visual acuity at 6 months was 20/20.

Conclusions and importance: Adequate viscoelastic removal and minimal iris stimulation seem to be essential to avoid this condition in hyperopic implants that lack a central port. Additionally, prompt treatment can minimize visual impairment and hasten visual recovery.

1. Introduction

With the limitations of corneal refractive surgery for the correction of high refractive errors, and with the subsequent FDA approval of phakic intraocular lenses (PIOLs), the latter gained wide acceptance in the ophthalmic community. Posterior chamber PIOLs were found to lead to significantly less corneal endothelial loss than angle-supported anterior chamber IOLs and anterior chamber iris-supported IOLs.¹ The Visian implantable collamer lens (ICL, STAAR Surgical, Monrovia, CA, USA) is the most commonly implanted posterior chamber PIOL.² It is a ciliary sulcus-based foldable IOL requiring sulcus to sulcus diameter prediction directly through high-frequency ultrasonography or indirectly via white to white measurement to prevent IOL-crystalline lens contact or excessive anterior vaulting.³ The Visian ICL is not currently FDA approved for the treatment of hyperopia in the US. We present a case of Visian ICL implantation for the correction of hyperopia that was complicated in the early postoperative period by anterior chamber reaction and the subsequent deposition of whitish precipitates on the surface of the crystalline lens.

2. Case report

A 23-year-old male presented in November 2008 at the American University of Beirut Medical Center seeking refractive surgery. His manifest refraction was $+5.75-1.00 \times 015^\circ$ and $+5.75-1.00 \times 180^\circ$, and his cycloplegic refraction $+7.00-1.00 \times 015^\circ$ and $+7.00-1.00 \times 180^\circ$ in the right and left eyes, respectively, yielding a vision of 20/20 in either eye. His eye exam was essentially negative. His external anterior chamber depth of 3.0 mm bilaterally was deemed sufficient for Visian ICL implantation. The patient underwent neodymium:YAG laser peripheral iridotomies (PIs) to the right eye at 10 and 2 o'clock. Two weeks later, a spherical (non-toric) ICL was implanted in the right eye under topical anesthesia and conscious sedation. The ICL was successfully inserted into the sulcus albeit with some excessive manipulation that took a few minutes to counter a sub-optimally dilated and slowly constricting pupil, while additional viscoelastic (HEALON OVD, Abbott Vision, Santa Ana, CA, USA) injection was used to widen it. The anterior lens capsule was not touched.

On postoperative examination 3 hours later, the patient had severe

* Corresponding author.

E-mail address: sawwad@gmail.com (S.T. Awwad).

<https://doi.org/10.1016/j.ajoc.2020.100598>

Received 24 March 2019; Received in revised form 9 January 2020; Accepted 9 January 2020

Available online 16 January 2020

2451-9936/© 2020 Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

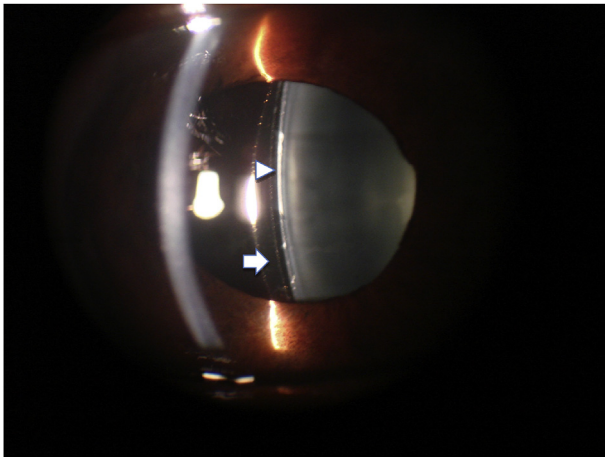


Fig. 1. A slit lamp photo of the right eye on postoperative day 3 after ICL insertion. There is a florid anterior chamber reaction and diffuse whitish deposits on the anterior surface of the crystalline lens.

nausea and vomiting, but no eye pain. The anterior chamber was deep, there was good ICL vaulting estimated at 1.5 central corneal thickness (CCT), around 750 μm , and the intraocular pressure (IOP) was 37 mmHg, so he was given oral acetazolamide and intravenous ondansetron for nausea. Follow-up on the second day showed a florid anterior chamber reaction and 4 + pigmented cells with an IOP of 5 mmHg. No leak was detected and the ICL vaulting had decreased to 3/4 CCT (approximately 400 μm). Hourly topical steroids were subsequently initiated and acetazolamide was discontinued. The patient presented the next day with diffuse whitish deposits appearing on the anterior surface of the crystalline lens (Fig. 1). His IOP was 5 mmHg and the anterior chamber reaction was still present. Oral prednisone at a dose of 1 mg/kg was added to the regimen. A mid-dilated pupil with a sluggish reaction to light and a small notch nasally, most probably iatrogenic in origin, was detected on exam, but was not clinically significant as to disrupt pupillary sphincter function.

Over the course of the next few days, the patient's IOP gradually increased to 9 mmHg, and the anterior chamber cells decreased in density with a slow decrease in the surface area of the crystalline lens deposits, mainly peripherally. On day 9, there was obvious clearing superiorly and at the peripheral areas (Fig. 2), followed later by central patchy deposit regression. His IOP reached 15 mmHg, and the ICL vaulting remained approximately 400 μm . Oral steroids were tapered

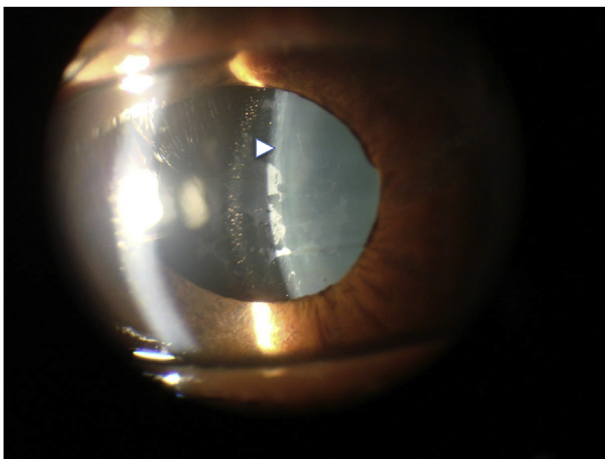


Fig. 2. A slit lamp photo of the right eye on postoperative day 9 after ICL insertion. There is clearing of the whitish deposits superiorly and at the peripheral areas.

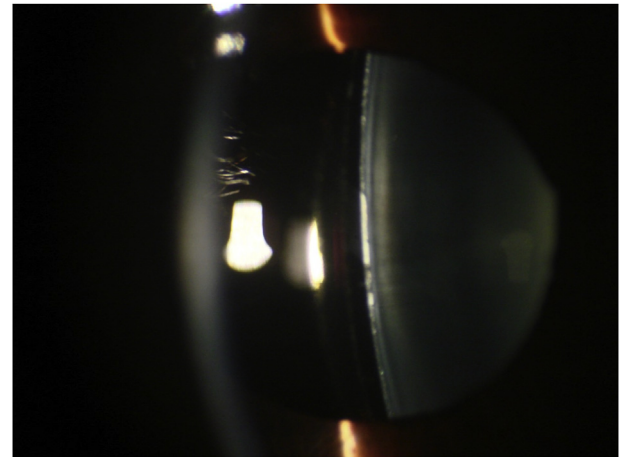


Fig. 3. A slit lamp photo of the right eye 6 months after ICL insertion. Most peripheral opacities on the crystalline lens have cleared and only some very faint central opacities can be viewed.

gradually over the course of 3 weeks and topical steroids over the course of 10 weeks. At the 6-month follow up, peripheral clearing was complete, but a few central patchy opacities remained (Fig. 3). The IOP was 15 mmHg in both eyes, and the ICL vaulting was approximately 1 CCT (500 μm). Uncorrected visual acuity was 20/25, and best-corrected vision was 20/20 with a manifest plano-refraction of -1.00×015 . The pupil diameter was 3.5 mm, and the patient was not disturbed by bright light.

3. Discussion

The postoperative course and sequence of events point toward florid iridocyclitis developing within 24 hours postoperatively. The sudden drop in the ICL vault by day 1 postoperatively suggests retained viscoelastic material, and the accompanying drop in the IOP in the presence of a well-formed anterior chamber and absence of leak, with a subsequent slow increase to baseline over several days suggests possible ciliary body (CB) shut down in addition to medication overtreatment. The combination of retained viscoelastic material, stasis from the low IOP, and iridocyclitis might have led to the crystalline lens capsular deposits.

Iritis or iridocyclitis has been rarely reported in phakic lens implantation, probably due to its generally benign nature.^{3,4} In a retrospective study by Zhou et al. acute iridocyclitis was reported in 1 eye out of 993 eyes (0.1%).⁵ However, it is interesting that deposits due to anterior chamber reactions have been historically and routinely described to be over the endothelium (keratic precipitates) and even over polymethyl methacrylate (PMMA), silicone, and acrylic intraocular lenses, but never over the crystalline lens capsule in the absence of friction.⁶

It is plausible to assume that the entrapped viscoelastic material under the ICL, which did not have a central port as it was a hyperopia correcting lens, reacted with the inflammatory substrates and deposited over the lens capsule or trapped the former. The very cause of the iridocyclitis could have been the result of undue intraoperative mechanical iris stimulation or a reaction to impurities in the drugs injected intracamerally. It is unclear whether the use of a different viscoelastic material, such as methyl cellulose, which is human based, as opposed to the animal-based product used, would have prevented such deposits.

The literature has reported an IOL-directed immune reaction characterized by deposits of whitish precipitates on synthetic IOLs.⁷ However, in our case, the ICL was not clinically affected by this deposition, ruling out the possibility of this inflammatory or immune reaction being directed against the ICL material (a porcine/HEMA copolymer).

Maldonado et al. described fleck-like opacities on the anterior capsule associated with a tight implant adherence with the crystalline lens at this site. This was not the case in our patient as the vaulting was approximately 750 μm when the opacities appeared.⁶ Finally, possible intraoperative trauma to the anterior lens capsule could also have caused capsular opacities. However, some parts of the opacities regressed over time, which refutes this hypothesis.

4. Conclusion

In summary, the formation of anterior capsular deposits after ICL insertion is very rare but possible, especially in hyperopic ICL implants with no central port and with excess residual viscoelastic material. Proper selection and adequate removal of the viscoelastic material along with minimal iris stimulation are recommended to avoid this condition. In addition, early identification and prompt treatment are essential to minimize visual impairment and hasten visual recovery.

Patient consent

The patient consented to the publication of the case. This report does not contain any personal information that could lead to the identification of the patient.

Funding

No funding or grant support.

Authorship

All of the authors attest that they meet the current ICMJE criteria for

authorship.

Declaration of competing interest

Shady Awwad is a consultant for STAAR Surgical, the manufacturer of the ICL. All of the other authors have no financial disclosures pertaining to this work.

Acknowledgements

None.

References

1. Kohnen T, Kook D, Morral M, Güell JL. Phakic Intraocular Lenses part 2: results and complications. *J Cataract Refract Surg*. 2010;36(12):2168–2194.
2. Pineda 2nd R, Chauhan T. Phakic intraocular lenses and their special indications. *J Ophthalmic Vis Res*. 2016;11(4):422.
3. Packer M. Meta-analysis and review: effectiveness, safety, and central port design of the intraocular collamer lens. *Clin Ophthalmol (Auckland, NZ)*. 2016;10:1059.
4. Fernandes P, González-Méijome JM, Madrid-Costa D, Ferrer-Blasco T, Jorge J, Montés-Micó R. Implantable collamer posterior chamber intraocular lenses: a review of potential complications. *J Refract Surg*. 2011;27(10):765–776.
5. Zhou TA, Shen Y, Wang Y, Xia JH. Mid-long term follow up results in the correction of extreme myopia by posterior chamber phakic intraocular lens. *Zhonghua Yan Ke Za Zhi*. 2012;48(4):307–311.
6. Maldonado MJ, García-Feijóo J, Benítez Del Castillo JM, Teutsch P. Cataractous changes due to posterior chamber flattening with a posterior chamber phakic intraocular lens secondary to the administration of pilocarpine. *Ophthalmology*. 2006;113(8):1283–1288.
7. Werner L. Biocompatibility of intraocular lens materials. *Curr Opin Ophthalmol*. 2008;19(1):41–49.