Cystoid macular edema related to intraocular lens dislocation and iris perforation by lens haptic

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ABSTRACT

A 64-year-old man who underwent a secondary 3-piece intraocular lens implantation (IOL) 2 years ago applied with decreased vision. There was a partial IOL capture and one haptic had perforated iris at two sites and was seen anterior to the iris surface. Fundus examination revealed macular edema. Topical nepafenac was initiated, however there was no improvement. Macular edema resolved following repositioning of the IOL and visual acuity improved. Lens dislocation and iris perforation by haptic can result in severe complications on the macula.

Key words: 3-piece intraocular lens, dislocation, haptic, iris perforation, macular edema.

INTRODUCTION

Cystoid macular edema (CME) is mostly associated with cataract surgery, but can also develop following secondary intraocular lens (IOL) implantations. It can be subclinical or sometimes results in permanent vision loss.[1-4] The incidence of CME ranges from 0% to 28%.[5]

Cystoid macular edema has been reported with all types of IOLs with the highest rate in 10-0 prolene iris-sutured IOLs (28%), but this study included accompanying penetrating keratoplasty.[6] A comparison study by Brunin et al. found the rate of CME in iris-fixated IOLs to be 23%.[7] Compared with anterior chamber IOL, CME was higher in 10-0 polypropylene iris-sutured IOL and 8-0 polypropylene scleral-sutured IOL.[6] Additionally, 8-0 prolene scleral-sutured IOLs (13.2%) reached a slightly higher rate of CME than 10-0 prolene scleral-sutured IOLs (0%–10.4%).[6]

Proper selection of secondary IOL type is very important to avoid CME. A small anterior chamber IOL may lead to increased movement within the anterior chamber or a large lens may lead to damage of angle structures resulting in secondary CME.[7] With iris-fixated IOLs, direct manipulation of the uveal tissue can incite additional intraocular inflammation.[8] The key mechanism seems to be surgically induced anterior segment inflammation that results in the release of endogenous mediators.[9]

Here, we present a case of severe CME secondary to 3-piece IOL dislocation and iris perforation.

CASE REPORT

A 64-year-old male patient applied to us for decreased vision in the right eye. He received a secondary sulcus fixated intraocular lens implantation surgery 2 years ago. However, he felt that his right eye vision did not improve significantly. There was no history of trauma or any other surgical intervention. On admission, best corrected visual acuity was 1/10 (Snellen) on the right and 10/10 (Snellen) on the left side. Intraocular pressure measurements were in normal limits.

Ocular examination revealed a sulcus fixated foldable

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hydrophilic acrylic 3-piece IOL. The IOL had a partial pupillary capture in which half of the optic was anterior to the iris and the haptic on the captured side had perforated the iris tissue at 9 o'clock from the anterior, went on behind the iris and came out again on the iris at 7 o'clock. (Figure-1) Conjunctiva, sclera and cornea were normal. Anterior chamber was quiet. There was no vitreous prolapse. Fundus examination revealed macular edema while optic nerve and other parts of the retina were normal. Optical coherence tomography (OCT) (Heidelberg Engineering, Germany) confirmed the diagnosis of cystoid macular edema (Figure-2a). Foveal macular thickness on OCT was 570 microns. There was no vitreoretinal traction or epiretinal membrane. Foveal macular thickness in the left eye was 260 microns.

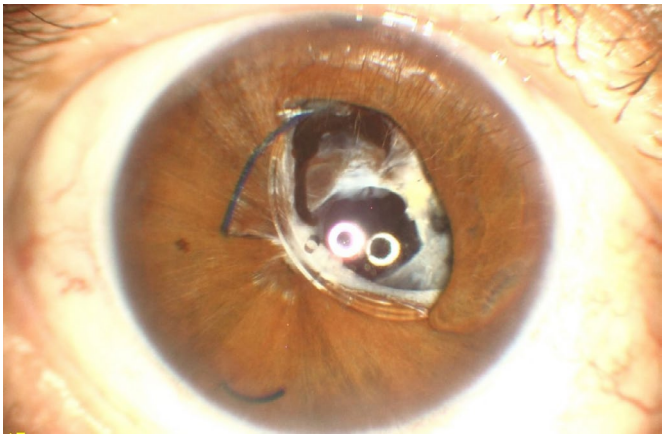

Figure-1: *Partially captured intraocular lens haptic double perforating iris.*

Topical nepafenac (Nevanac 0.1%, Alcon, USA) was initiated at a dose of three times/day for 4 weeks, however there was no improvement during the follow up. Lack of improvement led us repositioning the IOL. The surgery was performed under topical and intracameral anesthesia. After making a temporal main corneal incision and two side port incisions, anterior chamber was filled with viscoelastic. All posterior synechiae were released by a cyclodialysis spatula and pupillary area was expanded in order to check the condition of capsule and IOL. After reforming the posterior chamber with viscoelastic, the IOL was carefully rotated clockwise using a Sinsky hook, releasing it from the the iris. The IOL and haptic was replaced in the ciliary sulcus 3 clock hours away from the initial position. Viscoelastic was aspirated from the eye and anterior chamber was maintained with balanced salt solution. Finally, intracameral carbachol (Miostat 0.01%, Alcon, USA) was administered to keep the IOL away from the iris. The procedure was uncomplicated without anterior vitrectomy. Macular edema resolved rapidly after surgery and visual acuity improved to 20/30 at about 4 weeks. (Figure-2b).

CONCLUSION

There are two mechanisms that can cause CME. One is vitreous traction and the other is intraocular inflammation. With vitreous traction, there is an abnormal connection between the vitreous gel and the macula. Intraocular inflammation remains the most commonly accepted contributor to the development of postoperative CME.[9]

Inflammation can be caused by ocular trauma, surgery or a number of other retinal and uveal diseases. Surgically induced trauma to the iris, ciliary body and lens epithelium disrupts the blood-aqueous barrier resulting in release of prostaglandins, vascular endothelial growth factor, insulin-like growth factor-1 and other endogenous inflammatory mediators.[10] Prostaglandins and other pro-inflammatory mediators, released by the anterior uvea, enter the vitreous and increase the permeability of perifoveal capillaries, resulting in the intraretinal fluid accumulation with cystic changes in the outer plexiform layer and inner nuclear layer of the macula and retinal thickening.[11] A critical threshold of inflammatory mediators in the aqueous is likely required for identifiable macular edema.

Topical non-steroidal anti-inflammatory drugs (NSAIDs) and topical steroids are frequently used as the first-line treatment of pseudophakic CME. In the majority of cases, CME resolves with this regimen and other treatment modalities are not needed. For pseudophakic CME unresponsive to topical treatment, sub-Tenon or subconjunctival steroids provide more sustained drug release and a rapid improvement. Intravitreal triamcinolone acetonide, dexamethasone implant and fluocinolone acetonide implant have been used in refractory pseudophakic CME. Anti-VEGF injection may be a reasonable option in chronic CME patients who are intolerant to steroid treatment. When medical treatment is ineffective in resolving pseudophakic CME, surgical intervention is often the next step.

As with any procedure, each secondary IOL implantation technique has its own advantages, as well as its potential

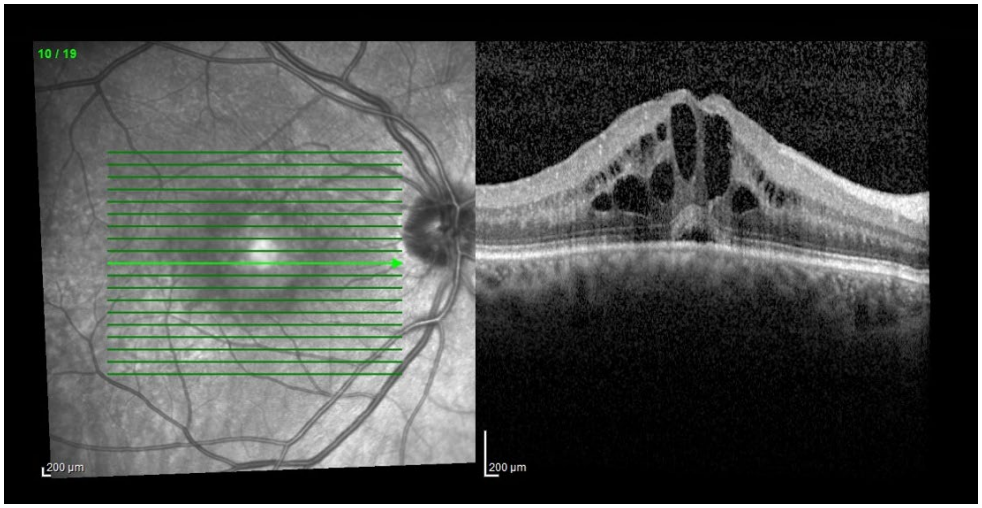

Figure-2a: *Optical coherence tomography image showing cystoid macular edema before treatment.*

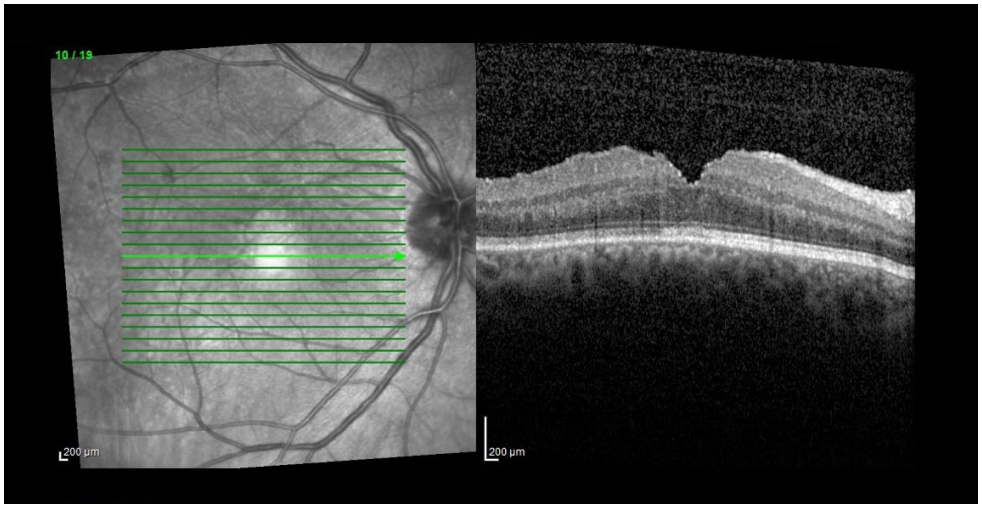

Figure-2b: *Resolution of cystoid macular edema following IOL repositioning.*

complications, including IOL tilt or dislocation, pupillary capture or block, postoperative uveitis, endophthalmitis, glaucoma, retinal breaks or detachments, vitreous hemorrhage and suture/haptic exposure or erosion.[7,12-14]

Surgical treatment is not necessary in all cases of dislocated IOLs. In cases with normal visual acuity, observation alone may be a viable option. Surgery is mostly indicated in cases of decreased vision, glaucoma, IOL dislocation into the vitreous cavity, CME and retinal detachment.[15] In this case, CME did not respond to topical NSAID treatment. It was likely that uveal irritation caused by the dislocated IOL and iris perforation induced a persistent and chronic inflammatory stimulus for CME, which was resistant to medical intervention. Therefore, instead of switching to peri/intraocular steroid or bevacizumab injections, we decided to perform an IOL repositioning.

To conclude, this is an interesting case of postoperative CME due to dislocated 3-piece IOL and iris perforation by IOL haptic with a rapid resolution following surgical correction. This case also shows the significant relationship between the anterior and posterior segments of the eye. Even in a quiet eye, IOL dislocation with perforation of iris tissue by a highly bio-compatible material can cause severe consequences in the posterior segment.

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