Novel maneuver for the alleviation of pseudophakic optic capture and consequent pupillary block: A case report and technique

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Abstract

Posterior chamber pseudophakic pupillary block (PPB) due to anterior optic capture is a rare condition with possible grave consequences. It can lead to extremely elevated intraocular pressure (IOP) and absence of anterior chamber space which may preclude the use of standard treatment methods. We describe a novel technique for its alleviation. An 80-year-old woman presented to a tertiary medical center with the left pupillary block 3 months after implantation of an intraocular lens (IOL) in the ciliary sulcus and 3 days after routine evaluation for age-related macular degeneration with tropicamide 0.5%. By exerting manual pressure on the corneal surface, the inferior half of the IOL optic was reduced into the posterior chamber, creating sufficient space for immediate Neodymium:Yttrium-Aluminum-Garnet (Nd:YAG) peripheral iridotomy. After IOP dropped to 20 mmHg, the upper half of the IOL optic was reduced into the posterior chamber using a Sinskey hook inserted through a single limbal paracentesis, and the anterior chamber was filled with an ophthalmic viscoelastic device. The device was subsequently aspirated and 1% acetylcholine was injected to constrict the pupil above the IOL optic. There were no operative complications. The corneal edema resolved, with normal cupping of the optic nerve head. At 3 months, the IOP was stable at 14–18 mmHg.

The maneuver described to allow for laser treatment of PPB is technically simple and non-invasive and was highly successful in an 80-year-old patient. Repositioning the pseudophakic IOL optic may be planned as a subsequent, non-urgent procedure.

Key words: Non-invasive maneuver, optic capture, pseudophakic pupillary block

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Introduction

Posterior chamber intraocular lens (PCIOL) optic capture is a complication of cataract surgery. It may lead to pseudophakic pupillary block (PPB) due to the inhibition of aqueous flow through the pupil. This, in turn, causes extremely high intraocular pressure (IOP) which poses a risk of grave visual consequences.1,2 PPB following intraocular lens (IOL) implantation is more common in cataract surgery with anterior chamber IOL implantation, especially when peripheral iridectomy has not been performed or is non-patent.3,4 PPB due to the presence of synechiae between the PCIOL and the posterior iris surface is rare after cataract surgery with PCIOL implantation,5,6 although it has been reported relatively more often in patients with uveitis or toxic anterior segment syndrome.5,6

In the past, when “can opener” capsulorrhesis, extracapsular cataract extraction, and J-loop sulcus-fixed IOLs were in common use, PCIOL optic capture, with or without pupillary block, was a more frequent finding.6-9 In the current era of continuous curvilinear capsulorrhesis, phacoemulsification, and foldable PCIOLs, optic capture is rare, and the number of cases of PPB with ocular hypertension has considerably decreased.10-12

Available options for the alleviation of PPB include Nd:YAG iridotomy, local and systemic osmotic agents, or other local and systemic IOP-lowering medications. In severe unresponsive cases, surgical reduction of the IOL optic into the posterior chamber and even pars plana vitrectomy may be used.

We describe a non-invasive maneuver for the alleviation of PPB due to PCIOL optic capture.
Case Report

An 80-year-old woman was admitted to the ophthalmic emergency service of a tertiary medical center with complaints of reduced visual acuity and severe pain in the left eye which had started 3 days previously. Ophthalmic history was significant for dry age-related macular degeneration (AMD) in both eyes and phacoemulsification with tear of the posterior capsule. The patient had undergone implantation of a foldable, three-piece IOL (MA60AC; Alcon Laboratories, Inc., Fort Worth, TX, USA) in the ciliary sulcus of the left eye 3 months before the present admission. Review of the surgical records revealed that there was no vitreous prolapse to the anterior chamber, and anterior vitrectomy had not been done. Findings at the 1-week and 1-month follow-up evaluations were unremarkable. The documented best-corrected visual acuity (BCVA) was 20/30.

Physical examination of the patient at admission yielded no abnormalities with the exception of nausea and slightly elevated blood pressure. Ophthalmic evaluation revealed a visual acuity of 20/30 in the right eye, which had a mild nuclear cataract and macular drusen but was otherwise normal. Findings in the left eye were as follows: Visual acuity – hand motion, IOP 76 mmHg (by Goldmann applanation tonometry), ciliary injection, corneal epithelial microcystic edema, completely absent anterior chamber, and complete contact of the IOL optic (which was located anteriorly to the iris) with the corneal endothelium. The entire iris was pressed against the corneal endothelium as well. The IOL haptics were located behind the iris, and the pupil was immotile and in the mid-dilated position [Figure 1a]. The fundus was hardly visible, but the retina could be seen to be attached.

Further, anamnesis was performed, and the patient disclosed that she had been examined by a retina specialist 3 days before admission for routine AMD follow-up. Tropicamide 0.5% (Mydramide; Fischer Pharmaceuticals, Ltd., Tel Aviv, Israel) was used during this examination. The patient noted that the pain in her left eye had started a few hours after the examination and gradually worsened over the next 2 days, at which time she sought medical treatment.

Treatment was aimed at rapidly reducing the extremely high IOP. It consisted of intravenous administration of 500 mg acetazolamide (Diamox; Mercury Pharmaceuticals, Ltd., London, UK) and oral glycerol 60 mg (according to 1 mg/kg body weight), timolol 0.5%/dorzolamide 2% (Cosopt, Merck & Co., Inc., Kenilworth, NJ, USA), apraclonidine 0.5% (Iopidine Alcon N.V., Puurs, Belgium), and travoprost 0.004% (Travatan, Alcon N.V.).

One hour later, IOP dropped to 68 mmHg. Tropicamide 0.5% (Mydramide, Fischer Pharmaceuticals Ltd.) and phenylephrine 10% (Efrin-10; Fischer Pharmaceuticals Ltd.) were then instilled to dilate the pupil and resolve the PPB. However, after 30 min, the pupil seemed to be immotile and the IOP did not decrease.

Surgical technique

At this point, we introduced a novel maneuver. A glass rod was used to manually exert pressure on the anterior surface of the cornea at the area corresponding to the optic-haptic IOL junction, where the optic edge was closest to the pupillary margin [Figure 1b]. This resulted in a reduction of the inferotemporal quarter of the IOL optic into the posterior chamber. We then pressed on the cornea the 2nd time, reducing the whole inferior half of the optic into the posterior chamber [Figure 1c]. A shallow inferior anterior chamber was then formed which allowed sufficient space for immediate Nd:YAG peripheral iridotomy at the 6 O’clock position [Figure 1d]. An immediate gush of aqueous fluid was noted flowing anteriorly through the iridotomy. The IOP dropped to 20 mmHg within 20 min.

Two hours later, the IOP was stable at 20 mmHg, and the patient was taken to the operating room. The upper half of the IOL optic was reduced into the posterior chamber using a Sinskey hook inserted through a single limbal paracentesis, while the anterior chamber was filled with an ophthalmic viscoelastic device. The device was subsequently aspirated, and 1% acetylcholine was injected to constrict the pupil above the IOL optic. No intraoperative difficulties or complications were noted.

Patient follow-up

The corneal epithelial edema resolved on the 1st post-operative day, and the corneal stromal edema that was noted at admission

![Figure 1](image-url)
resolved 1 week later [Figure 2]. The optic nerve head had normal color and cupping, and the retina looked normal. By the 3-month follow-up, BCVA had improved to 20/50. The IOP remained stable at 14–18 mmHg.

Discussion

Although PPB following PCIOL pupillary capture is rare, it may have serious consequences due to extremely high IOP. We present a novel, simple, non-invasive maneuver to reduce the IOL optic into the posterior chamber, thereby creating enough space in the anterior chamber for Nd:YAG peripheral iridotomy with resolution of the PPB. Repositioning of the PCIOL optic may be planned as a subsequent, non-urgent procedure.

It should be borne in mind that exerting pressure on the IOL optic through the cornea to reduce it to the posterior chamber may damage some endothelial cells. However, in the risk assessment process in the present case, given the generally favorable outcome of endothelial keratoplasty, we concluded that sacrifice of the endothelial cells was preferable to irreversible retinal or optic nerve head damage.

Several other options were considered when local and systemic medication failed to lower the IOP within 1 h. The first was to attempt Nd:YAG peripheral iridotomy on the iris while the iris was still attached to the corneal endothelium. This maneuver has never been published to the best of our knowledge, and it might have failed to resolve the PPB due to the complete absence of anterior chamber space. Nevertheless, it would have been our next choice had manual reduction of the IOL optic failed.

Another possibility was pars plana vitrectomy. However, summoning a vitreoretinal surgeon and operating room personnel would have taken too long, increasing the chances of irreversible ocular damage in this urgent setting. Moreover, performing sclerotomies in the presence of extremely high IOP pose a high risk of severe surgical complications relative to sclerotomies in eyes with normal IOP.

Medical treatment with other osmotic compounds (such as intravenous mannitol and oral sorbitol) was also contemplated. However, the case was urgent, and a quicker, more definitive solution was needed.

The exact process leading to this clinical picture of anterior PPB in our patient is unknown. We speculate that following pupillary dilation during the patient’s fundus examination 3 days before her hospitalization – probably a few hours after the mydriatic effect subsided – the IOL optic moved forward, and the pupil constricted behind it, leading to optic capture and, consequently, PPB. The right-side up position of the IOL ruled out an angulated upside-down IOL in the ciliary sulcus as the reason for the anterior movement of the optic, and the normal ocular axial length (22.4 mm) ruled out a short eye as the reason for the high posterior vitreous pressure. Capsular fibrosis and contraction may also explain anterior movement of the PCIOL optic, but this mechanism is more likely in cases of “in the bag” PCIOL, whereas in our patient, the PCIOL was placed in the ciliary sulcus.

The main limitation of the present work is lack of real-time images due to the urgent nature of this case. Representative artist’s impressions were added to clarify the clinical picture and the novel maneuver described.

Conclusions

PCIOL-associated optic capture and consequent PPB is a rare but severe and urgent clinical presentation that poses a treatment dilemma and necessitates careful risk management analysis to prevent a grave, irreversible outcome. In cases of absent anterior chamber space, we suggest a simple and technically easy maneuver consisting of the use of a glass rod to place pressure on the anterior surface of the cornea. This reduces the IOL into the posterior chamber, thereby allowing sufficient space for Nd:YAG peripheral iridotomy with resolution of the PPB.

References
