Late-onset capsular bag distension syndrome following antihistamine administration

Ilyse D. Haberman, MD, Irene M. Rusu, MD, Jonathan B. Kahn, MD

A 51-year-old woman presented with acute myopic shift 3 years after uneventful cataract extraction in both eyes; recent medical history was remarkable for administration of intravenous meclizine 2 weeks prior to presentation. Examination of each eye revealed anterior chamber crowding and a posterior chamber intraocular lens (IOL) with distension of the capsular bag; clear fluid with a green hue was present between the IOL and the posterior capsule. We postulate a mechanism for the development of late-onset capsular bag distension syndrome. To our knowledge, no medication-induced incidences have been reported in the literature.

Financial Disclosure: No author has a financial or proprietary interest in any material or method mentioned.

JCRS Online Case Reports 2014; 2:58–60 © 2014 ASCRS and ESCRS

Capsular block is a rare complication of cataract surgery with posterior chamber intraocular lens (PC IOL) implantation following a continuous curvilinear capsulorhexis (CCC). Capsular block is traditionally classified by its time of onset: intraoperative, early postoperative, or late postoperative. The mechanism of late postoperative capsular block involves the accumulation of particulate matter posterior to the IOL, which draws aqueous humor posterior to the IOL via osmosis. We describe a unique case of late-onset capsular block secondary to a systemic antihistamine and hypothesize a mechanism for the development of capsular block.

CASE REPORT

A 51-year-old Asian woman presented complaining of acutely decreased vision at distance and improved vision at near. The ocular history was significant for uneventful cataract extraction with bilateral implantation of a 1-piece acrylic PC IOL 3 years previously. On examination 3 months before presentation, the uncorrected distance visual acuity was 20/20 in both eyes; dilated fundoscopy was unremarkable, with monofocal acrylic 1-piece IOLs positioned in the capsular bag; and there was no anterior capsule fibrosis or fluid accumulation behind the IOLs. The recent medical history was remarkable for vertigo, for which the patient had been admitted to the hospital and administered intravenous meclizine 2 weeks previously.

On examination, the corrected distance visual acuity (CDVA) was 20/20 with a manifest refraction of $-1.50 \cdot -1.00 \times 165$ in the right eye and 20/20 with a manifest refraction of $-1.50 \cdot -0.25 \times 15$ in the left eye. Slitlamp examination was remarkable for anterior chamber crowding in both eyes. Gonioscopy revealed a 360-degree grade III ciliary body in both eyes with mild plateau iris configuration. Intraocular pressure was 15 mm Hg in the right eye and 17 mm Hg in the left eye. Dilated examination was remarkable for a PC IOL in each eye, with appreciable distension of the capsular bags and clear fluid with a green hue posterior to both IOLs (Figure 1).

Humphrey visual field testing was within normal limits in both eyes, as was optical coherence tomography (OCT) of the macula. Optical coherence tomography of the angle showed slight anterior displacement of the iris in both eyes. Anterior segment OCT of the lens–capsule complex demonstrated capsule distension with hyper-reflective material between the IOL and the posterior capsule in both eyes (Figure 2).

Meclizine was discontinued, and prednisolone acetate 1.0% eyedrops were started. The medication was self-discontinued after 2 weeks because there was no improvement in distance vision. On follow-up examination 1 month later, the myopic shift was slightly improved. The CDVA was still 20/20 in both eyes, with a manifest refraction of $-0.75 \cdot -0.50 \times 135$ in the right eye and $-1.25 \cdot -0.75 \times 5$ in the left eye. Repeat gonioscopy revealed a 360-degree grade III ciliary body in both eyes with normal iris insertion. The distension of the capsular bag was stable, with a green-hued fluid behind the IOL in both eyes. A treatment plan

Submitted: March 2, 2014.
Final revision submitted: April 15, 2014.
Accepted: April 15, 2014.

From New York University, Langone Medical Center, New York, New York, USA.

Corresponding author: Jonathan B. Kahn, MD, 161 Madison Avenue, Suite 5-SE, New York, New York 10016, USA. E-mail: jonathankahnmd@gmail.com.

JCRS Online Case Reports 2014; 2:58–60 © 2014 ASCRS and ESCRS

58 © 2014 ASCRS and ESCRS Published by Elsevier Inc.
was formulated to perform neodymium:YAG (Nd:YAG) laser capsulotomy to release the fluid into the vitreous cavity and to relieve the capsular block.

DISCUSSION
Capsular block, or capsular bag distension syndrome, is a rare complication of cataract surgery with PC IOL after CCC. First described by Davison, capsular bag distension syndrome is now classified according to the time of onset as intraoperative, early-onset postoperative, or late postoperative (1 month or later after surgery). Early capsular bag distension syndrome is characterized by capsule hyperextension, anterior displacement of the IOL, anterior chamber shallowing, and postoperative myopic shift. The classic mechanism of distension is thought to be blockage by the ophthalmic viscosurgical device (early-onset) or particulate matter (late-onset) within the capsular bag, which allows an osmotic shift of aqueous fluid into the capsular bag. In early capsular bag distension syndrome, posterior pressure created by the accumulation of fluid behind the IOL pushes the IOL–capsule complex forward. Movement of the IOL anteriorly in the eye brings the focal point in front of the retina and is responsible for the acute myopic shift.

Late postoperative capsular bag distention syndrome is less common and typically presents with accumulation of turbid fluid between the IOL and the posterior capsule. Analysis of this substrate has demonstrated the presence of α-crystallins, suggesting the substance is derived from residual lens epithelial cells. The late-onset syndrome presents with decreased visual acuity but only variably with refractive changes, suggesting IOL displacement is not a consistent feature. It is possible that the amount of posterior pressure created, as well as the elastic properties of the patients’ respective capsules, determines the amount of IOL displacement.

In this case, the accumulated fluid behind the IOLs was clear with a green hue, perhaps secondary to the filtering effect of a yellow-colored (light-filtering) IOL. The accepted treatment of capsular block distension syndrome is Nd:YAG posterior capsulotomy, which allows the accumulated fluid to escape into the vitreous cavity.

According to the time course, this case would be considered late onset (3 years after cataract extraction). The examination findings, however, are more consistent with early onset as there was anterior chamber narrowing and an acute myopic shift suggestive of anteriorization of the IOL complex. We postulate a unique mechanism for capsular block in this case in which the myopic shift was temporally linked to the administration of intravenous meclizine.
Meclizine (a piperazine derivative) is a first-generation histamine H1 antagonist used as an antiemetic for motion sickness and vertigo. First-generation H1 antagonists have poor selectivity and demonstrate anti-muscarinic, anti-α-adrenergic, and anti-serotonin effects. Both histamine H1 receptors and cholinergic muscarinic receptors are found in the ciliary body and mediate the contraction and relaxation of the ciliary body–zonule–lens complex, which may alter the position of the IOL and result in acute myopia. Cases of acute myopia secondary to topiramate are similarly well-described in the literature. This sulfa-based drug, via an unknown mechanism, causes ciliary body edema and anterior rotation of the ciliary body–lens complex. The phenomenon has also been described secondary to promethazine, which is a first-generation H1 antihistamine with properties similar to those of meclizine.

It is plausible that the administration of meclizine caused an acute change in the morphology and/or position of the ciliary body, changing the tension of the zonular fibers and the relative position of the capsular bag to the IOL. Given the patient’s young age at the time of surgery, it is possible that the capsule may be more elastic and distensible; lack of anterior capsule fibrosis supports the possibility that the anterior capsule may not be completely adherent to the IOL optic. Thus, ciliary body rotation could change the tension on the capsular bag, creating small gaps between the anterior capsule and the IOL. The negative pressure created by the potential space between the IOL and the posterior capsule would draw aqueous humor into the capsule complex and allow it to accumulate posterior to the IOL. As the effects of the antihistaminergic/anticholinergic medication wears off, the ciliary body would revert to its normal position, sealing off the previously created small gaps. Fluid would be trapped inside the capsular bag posterior to the IOL, causing capsular block. The posterior pressure from the fluid would remain, causing anterior displacement of the IOL, shallowing of the anterior chamber, and acute myopia, a clinical picture indistinguishable from typical early-onset capsular bag distension syndrome.

To our knowledge, this is the first report of medication-induced capsular block. As the mechanism for capsular block in this case is unique, it cannot be classified according to traditional nomenclature.

REFERENCES