

# American Academy of Optometry: Case Report 1

## **Capsular Block Syndrome Causing Reduced Vision Years After Cataract Surgery**

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### **Abstract:**

Late-postoperative capsular block syndrome is a treatable complication presenting months to years after cataract surgery employing continuous curvilinear anterior capsulorhexis and a posterior chamber intraocular lens implantation. Patients with late-postoperative capsular block syndrome may present with blurry vision months to years following cataract surgery performed with continuous curvilinear capsulorhexis and posterior capsule intraocular lens implantation. The defining clinical sign is entrapment of turbid fluid between the intraocular lens and posterior capsule that may result in reduced vision or refractive error shift. Late-postoperative capsular block syndrome is distinguishable from other late complications of cataract surgery, including delayed endophthalmitis or phacoantigenic uveitis, by lack of concurrent robust ocular inflammation. Successful treatment is often achieved with laser capsulotomy.

**Keywords:** *cataract surgery complications, capsular block, intraocular lens, postoperative complication, pseudophakia*

## **Introduction**

The introduction of anterior capsulorhexis by Gimbel and Neuhann in the mid-1980s revolutionized modern cataract surgery by creating a window in the anterior capsule with smooth edges, leaving the posterior capsule intact to support the intraocular lens implant.<sup>1</sup> Various capsulectomy techniques that preceded capsulorhexis left ragged edges in the anterior capsule rim that were predisposed to tears, increasing complication rates.<sup>1</sup> However, capsulorhexis is not free from its own unique problems. One such adverse event is late-postoperative capsular block syndrome, an uncommon, often visually significant, complication of cataract surgery performed with anterior continuous curvilinear capsulorhexis and implantation of a posterior chamber in-the-bag intraocular lens.<sup>2,3</sup> Arising months to years postoperatively, capsular block syndrome occurs secondary to capsule fibrosis that adheres the remaining rim of the anterior capsule to the anterior surface of the intraocular lens. This leads to a sealed compartment between the lens implant and the posterior capsule that then fills with turbid fluid.<sup>2-6</sup> Patients may present with symptoms of blurred or hazy vision, while others lack symptoms.<sup>2,3</sup> Successful treatment in symptomatic patients has been demonstrated with Neodymium-doped Yttrium Aluminum Garnet (Nd:YAG) capsulotomy.<sup>2-9</sup> This case elucidates the defining clinical characteristics and management of one case of late-postoperative capsular block syndrome.

## **Case Report**

### **Initial Visit**

A 67 year-old male presented on 17 August 2018 for an urgent exam with a chief complaint of a 3-month history of gradually worsening hazy vision in the right eye. He denied pain, photophobia, ocular redness, or discharge. His ocular history was significant for pseudophakia in both eyes status post uncomplicated cataract extraction performed with continuous curvilinear capsulorhexis, phacoemulsification, and posterior chamber in-the-bag intraocular lens implantation performed seven years prior in the right eye and twelve years prior

in the left eye. Specifically, the implanted intraocular lens in the right eye was a one-piece Acrysof IQ® (Alcon, Ft. Worth, TX) monofocal posterior chamber lenses. The left eye had a one-piece intraocular lens implant determined clinically, however the exact model was unknown as the left eye surgery was performed by an outside provider. The patient was also noted to have low myopia and presbyopia at previous exams. The patient's medical history was notable for hypertension and hyperlipidemia for which he was taking lisinopril and atorvastatin. The patient reported no known allergies to drugs. The patient's family ocular history was unremarkable.

His best corrected distance visual acuity was 20/30 OD and 20/20 OS with a refractive error of -1.00 +0.50 x 180 OD and -1.25 +0.50 x 180 OS. Pinhole testing did not improve the vision in the right eye. Near visual acuity was 0.4/0.8 M OD and 0.4/0.4M OS with +2.00 add over the manifest refraction. Pupils were equally round and briskly reactive to light without a relative afferent pupillary defect OU. Extraocular muscle motilities were full OU. The patient was found to be orthophoric at distance and near with cover test. Confrontation visual fields were full OD and OS.

Slit lamp examination of the anterior segment revealed clean lids and lashes, clear and quiet palpebral and bulbar conjunctiva, and white sclera OU. A temporally-located clear corneal incision scar was noted in each cornea. The anterior chamber was found to be deep and without cell or flare OU. The iris was flat and blue OU. Fluorescein Sodium was instilled in each eye. No corneal staining was observed, and the tear break up time was approximately 8 seconds in each eye. One drop containing Fluorescein Sodium 0.25% and Benoxinate Hydrochloride 0.4% was instilled in each eye in order to measure intraocular pressures by Goldmann applanation tonometry. The intraocular pressures were 15 mmHg OD and 13 mmHg OS.

The patient was dilated using one drop of 1% Tropicamide and one drop of 2.5% Phenylephrine OU. When the patient was fully dilated, posterior segment examination was performed using a slit lamp, 90 diopter lens, then a 20 diopter lens with a binocular indirect ophthalmoscope. Examination of the right intraocular lens revealed a well-centered one-piece

lens implants with 360 anterior capsule fibrosis located peripheral to the visual axis as well as posterior capsule opacification inferior to the visual axis. The left one-piece intraocular lens was clear and centered. No anterior capsule fibrosis nor posterior capsule opacification was noted OS. In the right eye, the posterior capsular bag was distended posteriorly and filled with hazy, brunescient fluid between the intraocular lens and posterior capsule (**Figure 1a**). Bilateral Weiss rings were visible and no vitreous cell was observed. The optic nerves had distinct margins and pink, healthy rim tissue. The cupping was graded to be 0.4 horizontally and vertically OU. The macula was flat and even OU with a healthy appearance to the posterior pole OU. The blood vessels were of normal caliber and contour OU. The peripheral retina was flat and intact 360 OU.

Anterior segment optical coherence tomography(OCT) using the Zeiss Cirrus and ultrasound biomicroscopy(UBM) using the VueMax were performed on the right eye to confirm the presence of a fluid-filled and posteriorly distended capsular bag. Anterior segment OCT revealed a well-formed and quiet anterior chamber and flat iris. The intraocular lens was clearly visible with a hyperreflective, well-defined body extending posteriorly (**Figure 2a**), consistent with the milky fluid contained within the capsular bag noted with slit lamp examination. Similarly, ultrasound biomicroscopy showed an ultrasound-hollow well-demarcated posterior extension of the posterior capsule, consistent with fluid contained within the capsular bag (**Figure 3a**).

The following differential diagnoses were considered:

1. Posterior capsule opacification
  - a. Posterior capsule opacification is a common late postoperative complication of extracapsular cataract extraction that can arise months to years postoperatively secondary to proliferation and migration of residual lens epithelial cells.<sup>27</sup> If cell migration involves the visual axis, patients may

experience reduced or hazy vision. Posterior capsule opacification was observed in this patient, however this was noted inferior to the visual axis (**Figure 1**) which does not explain the patient's reduced visual acuity and symptoms of hazy vision.

## 2. Phacoantigenic uveitis

- a. Phacoantigenic uveitis was included in the differential as it is a late complication of cataract surgery. The term "phacoantigenic uveitis" includes three separate clinical entities – phacoanaphylactic endophthalmitis, phacogenic nongranulomatous uveitis, and phacolytic glaucoma – that involve intraocular inflammation occurring secondary to an immune reaction to lens proteins.<sup>26</sup> Phacoanaphylactic endophthalmitis and phacogenic nongranulomatous uveitis both result from surgical or traumatic lens capsule rupture. Phacoanaphylactic endophthalmitis presents as an acute granulomatous anterior uveitis days to months postoperatively, whereas phacogenic nongranulomatous uveitis is a chronic, nongranulomatous anterior uveitis arising weeks postoperatively. While both of these entities occur somewhat later in the postoperative course, they demonstrate some degree of intraocular inflammation, of which this patient had none. Additionally, patients with phacoantigenic uveitis are often symptomatic for pain, photophobia, or redness, whereas this patient denied any of these symptoms. Therefore, phacoantigenic uveitis was deemed an unlikely diagnosis.

## 3. Delayed-onset postoperative endophthalmitis

- a. Another uncommon postoperative complication of cataract surgery is delayed endophthalmitis, which is characterized by a slowly progressing intraocular inflammation.<sup>28</sup> Clinical signs include a white intracapsular plaque,

conjunctival congestion, hypopyon, and vitreous inflammation. However, the appearance of the opalescent material within the capsule did not resemble that of a capsular plaque and there was no evidence of intraocular inflammation, making this differential diagnosis less likely

4. Late-postoperative capsular block syndrome (Final Diagnosis)

- a. Late-postoperative capsular block syndrome is a rare complication of cataract surgery employing anterior curvilinear capsulorhexis and posterior chamber-in-the-bag intraocular lens placement. It is characterized by 360 fibrosis of the anterior capsule edges, milky fluid posterior to the intraocular lens and contained within the posterior capsule, and the minimal to absent intraocular inflammation that arises months to years postoperatively. This patient's clinical presentation aligned well with this clinical description and the diagnosis of late-postoperative capsular block syndrome was made.

It was discussed with the patient that the likely cause of his visual symptoms was the entrapment of cloudy fluid in the physiologic bag that contained the lens implant in his right eye. It was explained that this is an uncommon complication that can occur many months to years following uneventful cataract surgery, and is often easily treated with Nd:YAG laser treatment to open the bag and release the trapped fluid. It was discussed with the patient that complications of infection or inflammation secondary to Nd:YAG laser capsulotomy have not been described in the literature when used to treat this specific condition, however when used to treat a similar condition (posterior capsule opacification), Nd:YAG laser capsulotomy has been associated with subsequent mild intraocular inflammation, swelling of the macula, retinal tear or detachment, and an increase in intraocular pressure. The patient was referred to ophthalmology for treatment with Nd:YAG capsulotomy.

## **Follow Up #1**

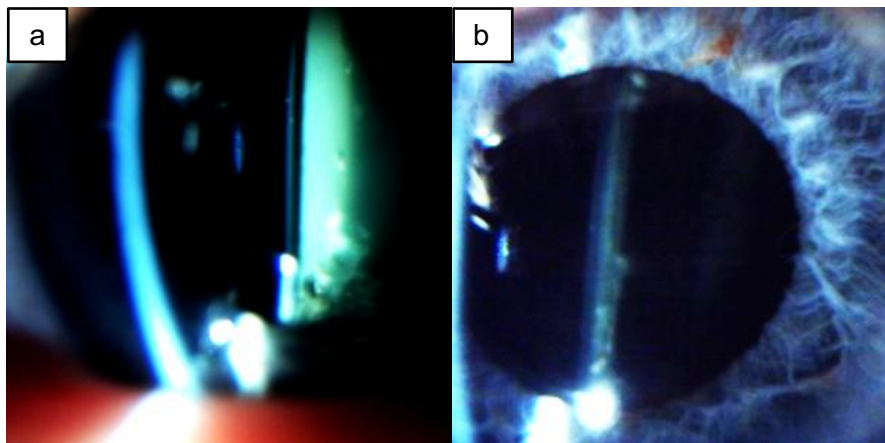
The patient returned to clinic on 21 September 2018 for the scheduled capsulotomy with ophthalmology. The patient reported stable hazy vision in the right eye and best corrected visual acuity was unchanged. The right eye intraocular pressure was measured by a technician to be 19 mmHg using Tonopen after instillation of one drop of Fluress. The technician instilled 1% Tropicamide and one drop of 2.5% Phenylephrine OU to dilate the patient. A posterior capsulotomy OD was performed using a Nd:YAG laser, resulting in the release of the cloudy fluid into the vitreous. A dilated fundus exam was performed following the procedure, which noted the posterior segment findings to be stable and within normal limits. The patient was prophylactically started on prednisolone acetate 1% QID for 7 days and timolol 0.5% BID for 5 days to prevent inflammation and intraocular pressure spike following the procedure. The patient was instructed by to follow up in 1 week to monitor for complications related to Nd:YAG.

## **Follow Up #2**

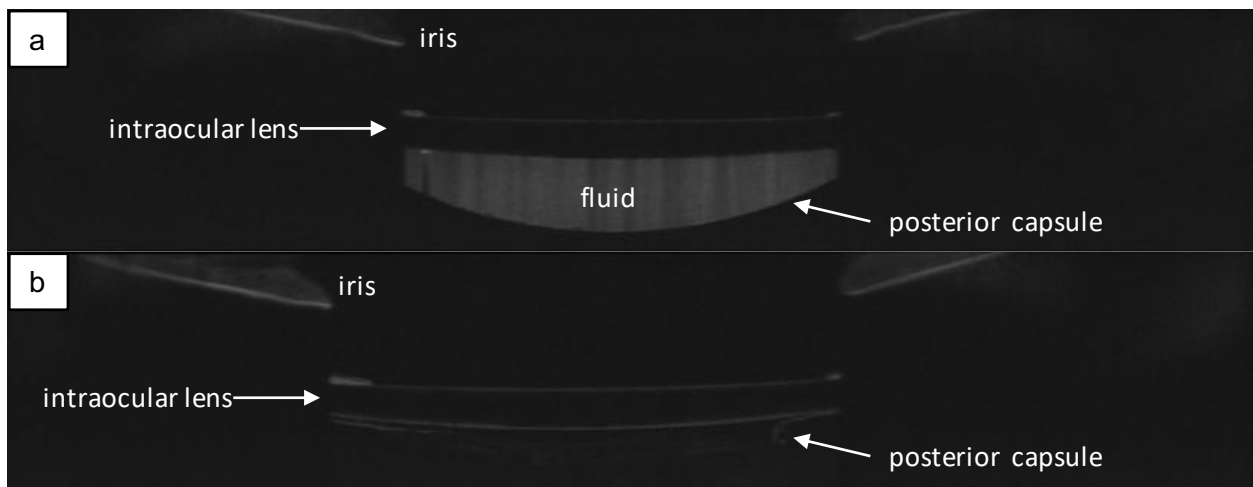
The patient presented to ophthalmology for scheduled follow up on 28 September 2018. The patient reported marked improvement in vision and complete resolution of haze OD. The patient reported completion of the 7-day course of prednisolone acetate 1% course and 5-day course of timolol 0.5%. He denied pain, photophobia, flashes, or floaters. Best corrected vision in the right eye now measured 20/20. Intraocular pressure OD was 16 mmHg using Goldmann applanation tonometry, following installation of one drop of Fluress. The ophthalmologist reported no evidence of intraocular inflammation. The patient was dilated OD with one drop of Tropicamide 1% and one drop of Phenylephrine 2.5%. The dilated fundus exam demonstrated a flat and intact retina. The patient was instructed to follow up annually for routine examination and to return stat with any acute changes to vision, flashes, floaters, onset of ocular pain, photophobia, or discharge.

The patient was returned to optometry clinic for repeat imaging on the same day following YAG capsulotomy. Anterior segment OCT and UBM were repeated, which demonstrated that the posterior capsule was appositional to the intraocular lens without residual retained fluid in the bag (**Figures 1b, Figure 2b, Figure 3b**).

## Figures

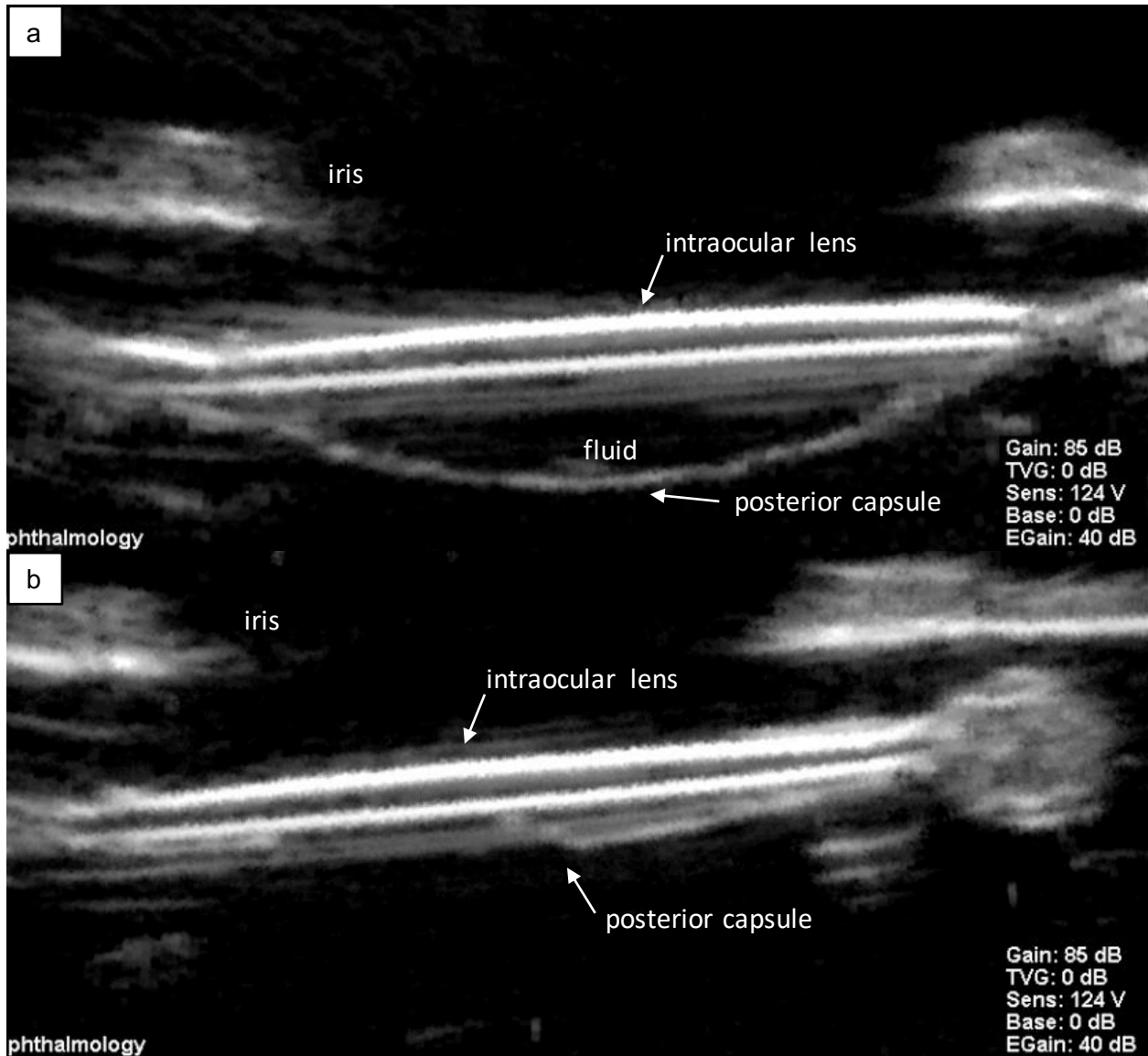


**Figure 1:** Slit lamp photographs before (a) and after (b) laser posterior capsulotomy. (a) Turbid fluid distends the capsule posteriorly. Posterior capsular opacification can be seen inferiorly. (b) Following laser posterior capsulotomy, no fluid is evident posterior to the intraocular lens.





**Figure 2:** Anterior segment optical coherence tomography before (a) and after (b) laser posterior capsulotomy. (a) Distention of the capsular bag is represented by the well-defined hyperreflectivity posterior to the intraocular lens. (b) Following laser capsulotomy, the remaining peripheral posterior capsule can be seen posterior to the lens.



**Figure 3:** Ultrasound biomicroscopy before (a) and after (b) laser posterior capsulotomy. (a) The posterior capsule can be seen as bowing posteriorly. The turbid fluid is represented by a hyporeflective space between the posterior capsule and the lens. (b) Following laser capsulotomy, the remaining posterior capsule can be seen closely abutting the lens.

## Discussion

Capsular block syndrome was first described by Davison in 1990 and Holtz in 1992 as a complication of continuous curvilinear capsulorhexis in which remnants of the anterior capsule become sealed to the anterior surface of the lens implant.<sup>8,9</sup> The subsequent collection of posterior intracapsular fluid can result in all or some of the following clinical signs: anterior lens displacement, shallowing of the anterior chamber, myopic shift, elevated intraocular pressure, and accumulation of hazy fluid within the capsular bag.<sup>8,9</sup> This type of capsular block syndrome was later deemed early-postoperative capsular block syndrome by Miyake et al in 1998, who subdivided capsular block syndrome into three distinct entities: intraoperative, early-postoperative, and late-postoperative capsular block syndrome.<sup>2</sup> All three types have in common the occlusion of the anterior capsular opening created by capsulorhexis with subsequent fluid accumulation within the closed capsule. However, these entities differ in time of onset and intracapsular contents.<sup>2</sup>

Intraoperative capsular block syndrome occurs during surgery as the bag swells with balanced salt solution during rapid hydrodissection.<sup>2</sup> Early-postoperative capsular block syndrome arises within one day to several weeks postoperatively and is thought to be secondary to retained viscoelastic within the bag.<sup>2,10</sup> Late-postoperative capsular block syndrome, as seen in this case report, arises months to years postoperatively secondary to fibrosis between the lens implant and the edges of the anterior capsule initially created by capsulorhexis.<sup>3-6</sup> In late-postoperative capsular block syndrome, fibrosis occurs along the entirety of the anterior capsule opening and creates a closed compartment between the lens

implant and the capsule, within which milky, turbid, or white fluid collects.<sup>3-6</sup> As the capsule fills with fluid, it bows posteriorly,<sup>3</sup> lending to descriptions such as capsular bag distension syndrome, liquefied aftercataract, and capsulorhexis-related lacteocruemnesia. Some authors have argued that liquefied aftercataract is a separate entity from late-postoperative capsular block syndrome that is defined by a lack of intraocular lens anterior displacement and intraocular pressures spike. However, most agree that these complications are clinically indistinguishable and both are treated with laser capsulotomy.<sup>11,12</sup>

In contrast to intraoperative and early-postoperative capsular block syndrome, a shallow anterior chamber or increased intraocular pressure are rarely seen in late-postoperative capsular block syndrome. In late-postoperative capsular block syndrome there may be a hyperopic shift secondary to a concave lens effect created by accumulated fluid,<sup>6</sup> myopic shift due to anterior displacement of the lens<sup>10</sup> or light scattering by the turbid fluid,<sup>11</sup> or no refractive error shift at all.<sup>4,13</sup> Patients may complain of blurry or hazy vision years after uncomplicated cataract surgery. In a study of 44 eyes with late-postoperative capsular block syndrome, the average time of onset was 3.8 years after surgery, but ranged from two months to 6 years.<sup>3</sup> The longest reported duration between surgery and presentation of capsular block syndrome was 20 years.<sup>14</sup> This patient fits within this reported range, with symptom onset seven years postoperatively. A lack of robust intraocular inflammation distinguishes late-postoperative capsular block syndrome from other late complications of cataract extraction, such as delayed endophthalmitis and phacoantigenic uveitis.<sup>3,15</sup>

Late postoperative capsular block syndrome is rare with few known risk factors. A retrospective chart review of 1100 eyes in 990 patients who underwent phacoemulsification and posterior chamber intraocular lens implantation reported an incidence of 0.46% for early postoperative and 0.27% for late postoperative capsular block syndrome over 37 months.<sup>16</sup> Postoperative capsular block syndrome was more common in eyes with an axial length greater

than 25 millimeters. The authors postulated that longer eyes tended to have large capsular bags, which allows for fluid retention or collection following surgery.

Properties of the intraocular lens may also influence the development of capsular block syndrome. In the study that included 1100 intraocular lenses with both three-piece and one-piece design, no eyes with a three-piece design developed postoperative capsular block syndrome. The Akreos Adapt,<sup>®</sup> a one-piece lens that features a four-point plate haptic design, more commonly resulted in postoperative capsular block compared to the Acrysof MA60BM,<sup>®</sup> which has a C-loop haptic design. Of note, this patient had an AcrySof<sup>®</sup> lens, the same intraocular lens associated with capsular block syndrome in the above study. The authors hypothesized that the four-point non-angulated haptic design of the Akreos Adapt<sup>®</sup> allowed for greater contact area between the intraocular lens and the anterior capsule, thus enabling anterior capsule fibrosis to the lens surface. Similarly, Vock et al found that intraocular lenses with little to no angulation were more likely to result in anterior capsule opacification secondary to reduced space between the lens and anterior capsule.<sup>17</sup> Hydrophilicity of the implant material may also contribute to postoperative capsular block syndrome.<sup>16</sup> Anterior capsule opacification is more common in hydrophobic acrylic than hydrophilic acrylic materials.<sup>17,18</sup> It can be deduced that greater rates of anterior capsule opacification coincide with an increased risk of capsular block syndrome, however the association of anterior capsule opacification and contraction with postoperative capsular block syndrome has yet to be investigated.

There is no consensus as to the contents of the intracapsular fluid. Some have hypothesized that residual lens epithelial cells proliferate and produce collagen and extracellular matrix within the retained fluid, resulting in higher osmolarity within the capsule. The osmotic gradient draws aqueous humor into the capsule leading to the accumulation of turbid fluid.<sup>2,3</sup> Several studies have identified the common lens proteins  $\alpha$ - and  $\beta$ -crystallin in aspirated fluid samples evaluated by electrophoresis, supporting this theory.<sup>11,19,20</sup> However, a study utilizing a similar technique to evaluate the fluid did not identify proteins indicative of collagen,

contradicting the notion that collagen production leads to fluid collection.<sup>15</sup> More recently, the theory was further augmented with the discovery of elevated levels of the proinflammatory cytokines tumor necrosis factor- $\alpha$  and interleukin- $1\beta$  within the closed capsular bag via aspiration and flow cytometry. It was proposed that residual lens epithelial cells release these proinflammatory cytokines that promote anterior capsule fibrosis and contribute to the breakdown of the blood aqueous barrier, ultimately resulting in capsular block syndrome. The absence of gamma globulins that would suggest an antigen-antibody reaction has led to the belief that capsular block syndrome is not the result of an infectious process.<sup>19</sup> Conversely, aspiration of the milky substance in one case of late postoperative capsular block syndrome identified *Propionibacterium acnes* by bacteriological culture and polymerase chain reaction,<sup>21</sup> the same organism known to cause endophthalmitis. The patient in that report was treated with Nd:YAG capsulotomy and topical levofloxacin and topical prednisolone. After a 4 week treatment and steroid taper, full recovery was observed. *Propionibacterium acnes* was also isolated from the fluid in two more reported cases of late capsular block syndrome that were later treated with capsulotomy alone.<sup>22</sup> The authors postulated that endophthalmitis did not develop following release of the capsular fluid due to a low bacterial count or the suboptimal environment provided by the oxygenated vitreous for the typically anaerobic aerotolerant bacterial species. The patient in this case report presented without signs of ocular inflammation, did not receive antibiotic prophylaxis after capsulotomy, and recovered without complication.

On occasion, late-postoperative capsular block syndrome may resolve spontaneously secondary to contraction of the anterior capsule fibrosis creating an opening in the capsule through which fluid may exit,<sup>3</sup> however the majority of cases require treatment. Successful treatment with Nd:YAG capsulotomy of the anterior or posterior capsule has been well-described within the literature for early and late-postoperative capsular block syndrome.<sup>2-9</sup> In Nd:YAG capsulotomy, a solid-state laser (Nd:YAG) of wavelength 1064 nm is focused on the anterior or posterior capsule and pulsed in order to disrupt the capsule tissue via optical

breakdown.<sup>29</sup> In the case of capsular block syndrome, the disruption of the posterior capsule leads to the release of fluid into the vitreous cavity, deflation of the capsular bag, and repositioning of the displaced intraocular lens implant. This results in a reversal of the refractive error shift and restoration of visual acuity. Common complications of Nd:YAG capsulotomy include retinal tear or detachment, cystoid macular edema, increase in intraocular pressure, and uveitis,<sup>30</sup> thus patients should be monitored closely in the post-operative period and should be instructed to return immediately if symptoms of retinal detachment or inflammation occur. In many cases, intraocular pressure increase can occur within hours of the procedure and pressures should be monitored same-day. Patients are often prescribed a short course of topical steroids with or without cycloplegics as prophylaxis to post-laser inflammation as well as intraocular pressure lowering agents to address or prevent postoperative intraocular pressure spike, though this may vary between providers. Patients should be monitored closely while on steroids or intraocular pressure lowering medications.

Posterior capsulotomy may not be a viable treatment approach if severe turbidity of the sequestered fluid or excessive posterior capsule displacement secondary to fluid accumulation or high myopia prevents focusing of the laser on the posterior capsule.<sup>11,23</sup> In cases not treatable with posterior capsulotomy, anterior capsulotomy may be performed peripheral to the lens edge.<sup>10,23</sup> However, late-postoperative capsular block syndrome treated with anterior capsulotomy may have a lower success rate and an increased recurrence rate when compared to those treated with posterior capsulotomy.<sup>24</sup> In response to anterior capsulotomy, the released fluid presumably exits through the trabecular meshwork.

Surgical treatment may be indicated in some cases of late-postoperative capsular block syndrome. In cases with concurrent significant posterior capsular opacification, posterior continuous curvilinear capsulorhexis may be the preferred treatment method to avoid complications associated with the high laser energy required to treat dense posterior capsular opacification.<sup>25</sup> Another technique involving anterior capsule release, opaque fluid aspiration,

and irrigation of the posterior capsule has also been reported to effectively and safely treat postoperative capsular block syndrome.<sup>20</sup>

## **Conclusion**

Late-postoperative capsular block syndrome can occur months to years following uncomplicated cataract surgery utilizing capsulorhexis and a posterior chamber in-the-bag intraocular lens implant. Patients may present to optometric practices due to symptoms of blur or hazy vision. This uncommon complication is characterized by fibrotic adhesion of the anterior capsule edges to the anterior intraocular lens surface with milky fluid accumulation between the lens implant and the posterior capsule. It is distinguishable from other late complications of cataract surgery, including delayed endophthalmitis or phacoantigenic uveitis, by lack of robust concurrent ocular inflammation. Some cases may resolve spontaneously while others may benefit from laser capsulotomy.

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