Laser iridotomy to treat uveitis-glaucoma-hyphema syndrome secondary to reverse pupillary block in sulcus-placed intraocular lenses: Case series

Harmanjit Singh, MD, FRCSC, Milad Modabber, MD, MSc, Steven G. Safran, MD, PA, Iqbal Ike K. Ahmed, MD, FRCSC

PURPOSE: To present cases of uveitis-glaucoma-hyphema (UGH) syndrome due to reverse pupillary block in sulcus-placed posterior chamber intraocular lenses (PC IOLs) that were managed with laser peripheral iridotomy (LPI).

SETTING: Community-based subspecialty clinics.

DESIGN: Retrospective interventional case series.

METHODS: A chart review of patients with a sulcus-placed PC IOLs presenting with UGH syndrome and reverse pupillary block with posterior iris bowing as diagnosed by gonioscopy and anterior segment optical coherence tomography was carried out. Laser peripheral iridotomy was performed in the eyes included in the study. The main outcome measure was clinical resolution of UGH syndrome.

RESULTS: The study included 6 eyes of 6 patients with a mean age of 59.8 years (range 43.0 to 66.0 years) who presented with unilateral UGH syndrome a mean of 28.7 months (range 0.3 to 84.0 months) after PC IOL implantation. All patients were previously myopic, with 5 (83.3%) having a history of vitrectomy. The mean axial length was 27.0 mm ± 1.4 (SD). An LPI was used to treat the reverse pupillary block with resultant improvement in iris profile and resolution of UGH syndrome in all eyes. The mean intraocular pressure decreased from 30.5 ± 10.0 mm Hg on 0.5 ± 0.8 glaucoma medications to 15.5 ± 3.2 mm Hg postoperatively on 0.7 ± 1.2 medications.

CONCLUSIONS: The UGH syndrome due to reverse pupillary block occurred after sulcus-placed PC IOLs in susceptible patients, those with axial myopia, and post-vitrectomized eyes. The cases were managed with LPIs.

Financial Disclosure: Dr. Ahmed is a consultant to Alcon Laboratories, Inc. and Abbott Medical Optics, Inc. No author has a financial or proprietary interest in any material or method mentioned.

J Cataract Refract Surg 2015; 41:2215–2223 © 2015 ASCRS and ESCRS

Reverse pupillary block was described by Campbell[1] in his work on pigment dispersion syndrome (PDS). Potash et al.[2] performed ultrasound biomicroscopy in PDS patients to support this mechanism. Reverse pupillary block has been proposed as a principal causative mechanism for PDS in phakic patients.[3–6] In reverse pupillary block, the iris functions as a flap valve, allowing aqueous to pass from the posterior chamber to the anterior chamber but not in the opposite direction. The trapped aqueous within the anterior chamber causes posterior bowing of the peripheral iris, leading to mechanical friction between the posterior iris pigmented epithelium and the zonular bundles, causing pigment release.[1,5] The liberated pigment accumulates within the trabecular meshwork after aqueous convection currents. This may impair aqueous outflow, resulting in increased intraocular pressure (IOP) and possibly pigmentary glaucoma.[5,7]

Pigment dispersion syndrome in phakic patients often presents in young persons with myopia, with a

male predominance and a higher preponderance for individuals of white ethnicity.[8] Clinically, PDS presents with slit-like radial midperipheral iris transillumination defects; Krukenberg spindles; deposition of pigment on the anterior and posterior lens capsule, zonular fibers, iris, cornea, and the trabecular meshwork; as well as a posteriorly inserted iris that is concave in configuration.[3,5,8] The increased anterior chamber depth and the posterior bowing of the iris are of key importance in the etiology of PDS.[1–3,9] In such cases, relative pupillary block is further precipitated by exercise, accommodation, and blinking.[7,10] Each factor influences the iridozonular distance and may play a role in the disease process.[8]

Although classically described in phakic patients, reverse pupillary block has also been reported in pseudophakic eyes. Blumenthal and Chen[11] described reverse pupillary block during extracapsular cataract extraction. Secondary reverse pupillary block has also been described after cataract surgery and intraocular lens (IOL) implantation.[6,12–15] This was first reported by Karickhoff[6] after IOL implantation in the ciliary sulcus causing pigment dispersion. Since this initial observation, 2 case reports of pseudophakic reverse pupillary block with PDS have been reported after in-the-bag implantation of an IOL.[13,14] Rare cases of reverse pupillary block have also been described in scleral-sutured IOLs, yet without any sign of PDS.[12,15] Medical treatment in these cases were suboptimum.

Karickhoff[6] proposed laser peripheral iridotomy (LPI) as a potential treatment for reverse pupillary block. Subsequent reports of LPI for the treatment of reverse pupillary block with PDS for in-the-bag[13,14] posterior chamber IOLs (PC IOLs) and treatment of reverse pupillary block for sulcus-placed[12,15] PC IOLs without PDS have also been described.

We report our experience with 6 pseudophakic patients with sulcus-placed PC IOLs who presented with reverse pupillary block and uveitis-glaucoma-hyphema (UGH) syndrome that was treated with LPI. To our knowledge, this is the largest case series on the use of LPI in pseudophakic patients with sulcus-placed PC IOLs who presented with reverse pupillary block and UGH syndrome.

Submitted: December 6, 2014.
Final revision submitted: February 23, 2015.
Accepted: February 23, 2015.

From the Department of Ophthalmology and Vision Sciences (Singh, Ahmed), University of Toronto, Toronto, the Department of Ophthalmology (Modabber), McGill University, Montreal, Quebec, and Trillium Health Partners (Ahmed) and Credit Valley EyeCare (Ahmed), Mississauga, Ontario, Canada; Capital Health System (Safran), Pennington, New Jersey, USA.

Presented at the annual meeting of the Canadian Ophthalmological Society, Halifax, Nova Scotia, Canada, June 2014.

Corresponding author: Iqbal Ike K. Ahmed, MD, FRCSC, Credit Valley Eye Care, 3200 Erin Mills Parkway, Mississauga, Ontario L5L 1W8, Canada. E-mail: ikeahmed@mac.com.

PATIENTS AND METHODS

From 2 centers (Credit Valley Eye Care, Mississauga Ontario, Canada, and Capital Health System, Pennington, New Jersey, USA), consecutive cases of pseudophakic patients who presented with UGH syndrome in the presence of a sulcus-placed PC IOL and were found to have posterior iris bowing with reverse pupillary block were included in this retrospective study. Patients with in-the bag or bag-sulcus position PC IOLs were excluded.

Uveitis-glaucoma-hyphema syndrome was defined as evidence of uveitis, pigment dispersion, iris transillumination defects, hyphema, and/or elevated IOP in the presence of reverse pupillary block findings with a sulcus-placed PC IOL. Reverse pupillary block was identified using gonioscopy and anterior segment optical coherence tomography (AS-OCT) (Spectralis, Heidelberg Engineering, Inc.).

All eyes diagnosed with UGH syndrome and reverse pupillary block were treated with a neodymium:YAG LPI. Data collected included demographic data, visual acuity, IOP, glaucoma medications, and ocular examination findings before and after LPI treatment. Anterior segment OCT was performed before and after treatment. The main outcome measure was resolution of UGH syndrome.

Statistical analyses were performed using SPSS software (version 20, SPSS, Inc.). A Wilcoxon signed-rank test was used to compare logMAR corrected distance visual acuity (CDVA), IOP, and number of glaucoma medication classes before and after iridotomy. Data are reported as the mean $\pm$ standard deviation; a P value of 0.05 or less was considered statistically significant.

RESULTS

Six eyes of 6 patients were included in this study. A brief summary of each case is described below.

Case 1

A 64-year-old man was referred for a dislocated IOL–bag complex in the left eye; the axial length (AL) was 26.01 mm. He had phacoemulsification with in-the-bag implantation of a poly(methyl methacrylate) (PMMA) IOL 13 years previously with subsequent retinal detachment, requiring a scleral buckle and vitrectomy. The patient had explantation of the IOL–bag complex combined with a 20-gauge pars plana vitrectomy. A sulcus scleral-sutured PC IOL (16.5 diopter [D] P366UV, Bausch & Lomb) was uneventfully implanted in the eye.

One day postoperatively, the patient's uncorrected distance visual acuity (UDVA) was 20/25 and the IOP was 15 mm Hg. At 10 days, he presented with ocular pain and blurred vision and his vision had

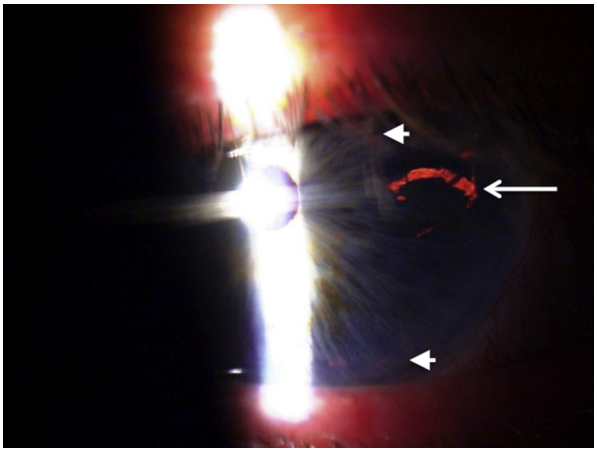


Figure 1. Various types of iris transillumination defects of the left eye: diffuse peripheral defects (*arrowheads*) and fingerlike defect (*arrow*).

decreased to 20/100 with an IOP of 43 mm Hg. Severe pigment dispersion syndrome was present with 3+ pigment in the anterior chamber, fingerlike iris transillumination defects (Figure 1), microcystic edema, and broad contact between the concave iris and the sulcus IOL, as confirmed on AS-OCT, indicating the presence of reverse pupillary block (Figure 2). An LPI was performed the same day after instillation of topical antiglaucoma medications. Backward flow of pigment was observed when perforation of the iris was complete, with associated resolution of reverse pupillary block (Figure 3). The day after, the patient's vision was 20/30 and IOP was 23 mm Hg. By 1 month, the IOP had normalized to 15 mm Hg without the use of glaucoma medications and his CDVA was 20/20. At last follow-up at 36 months, his CDVA was 20/20 with an IOP of 16 mm Hg without antiglaucoma medication, with no recurrence of UGH syndrome.

Case 2

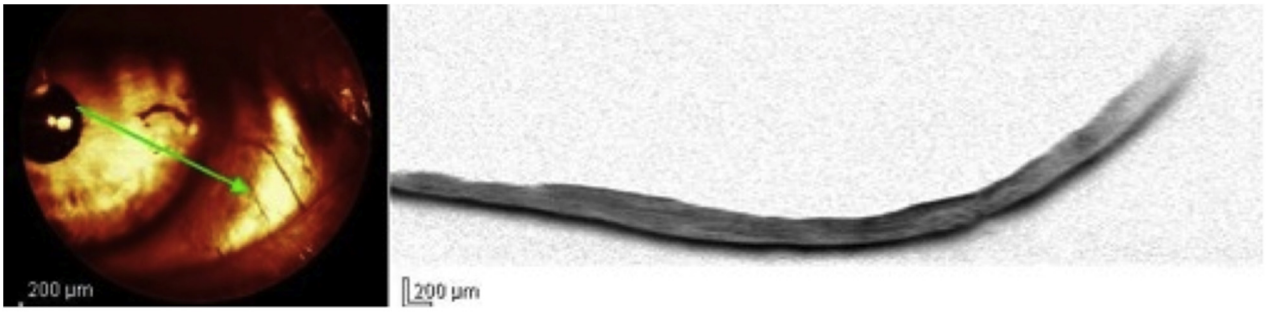
A 43-year-old man was referred for left-eye UGH syndrome. Five years prior, he had a 3-piece silicone IOL (7.0 D Tecnis Z9002, Abbott Medical Optics, Inc.) placed in the sulcus in that eye, with an AL of 28.46 mm. He also had a history of vitrectomy for a left retinal detachment. On examination, the patient's vision was counting fingers, with an IOP of 38 mm Hg. Slitlamp biomicroscopy examination revealed 2+ cells and pigment in the anterior chamber with the presence of hyphema and corneal edema. There was also a posterior bowing of the iris with fingerlike iris transillumination defects, confirmed on AS-OCT (Figure 4). There was deposition of pigment and red blood cells in the angle (Figure 5). These findings were in keeping with reverse pupillary block, and LPI was performed.

After LPI, gonioscopy revealed flattening of the iris (Figure 6). One hour after LPI, the IOP was measured at 24 mm Hg and vision was 20/400. The patient was placed on a steroid taper for a few weeks. No glaucoma medications were used. One day after LPI, his vision was 20/60 and IOP was 11 mm Hg. The iris appeared flat with no posterior bowing. At the 2-week follow-up, vision had improved further to 20/30, with an IOP of 17 mm Hg. There was no recurrence of UGH syndrome over 2 years of follow-up.

Case 3

A 66-year-old woman was referred for PDS in the right eye; the AL was 25.84 mm. She had a history of right phacoemulsification and IOL implantation, vitrectomy for retinal detachment, and 2 IOL exchanges for recurrent dislocated IOLs. The most recent IOL implanted was a sulcus scleral-fixated PMMA IOL (15.5 D P366UV, Bausch & Lomb).

Seven months postoperatively, the patient presented with evidence of irido-IOL contact, pigment dispersion syndrome, and fingerlike iris transillumination defects. Her CDVA was 20/30 with an elevated IOP of 35 mm Hg. This was recognized as reverse pupillary block, and LPI was performed. The iris immediately shifted forward and away from the



A *B*

Figure 2. *A*: Shadow of concave iris (*arrowhead*) raised peripherally and deep centrally. Iris fingerlike transillumination defect (*arrow*) can also be appreciated. *B*: Corresponding AS-OCT of the same eye showing posterior bowing of the iris.

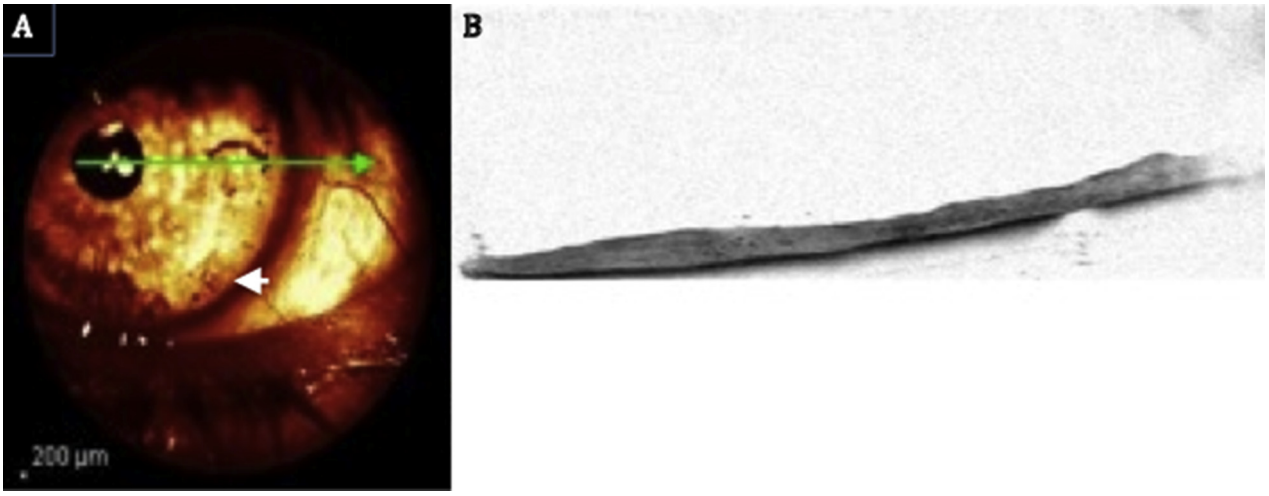


Figure 3. *A*: Post-laser peripheral iridotomy showing iris flattening and absence of the peripheral iris shadow (*arrowhead*). *B*: Corresponding AS-OCT image of the same eye also shows marked iris flattening.

IOL, with resolution of the reverse pupillary block. She was placed on a topical prostaglandin for IOP control. Two days postoperatively, the patient's vision was 20/20 and her IOP had normalized to 12 mm Hg. At the 4-year follow-up, her CDVA remained 20/20 with an IOP of 18 mm Hg on a topical carbonic anhydrase inhibitor.

Case 4

A 65-year-old man was referred for new-onset distortion of vision in the left eye; the AL was 25.39 mm. His previous ophthalmologic history included a bilateral myopic laser in situ keratomileusis procedure and cataract surgery more than 7 years ago with sulcus IOL placement complicated by vitreous loss in the left eye and subsequent retinal detachment requiring vitrectomy and scleral buckle. On examination, the patient's CDVA was 20/25. He had signs of PDS, and his IOP was elevated at 28 mm Hg in the left eye. He was placed on a topical prostaglandin and a β-blocker.

At the 3-month follow-up, he was found to have fingerlike iris transillumination defects, pigment deposition at the angle, and persistently elevated IOP of 29 mm Hg. Reverse pupillary block was diagnosed, and he was treated with LPI. At the 1 day postoperative examination, his CDVA was 20/20 and IOP had improved to 20 mm Hg. At the 2-week follow-up, the patient's IOP was 17 mm Hg. Four years after LPI, he remained stable with a CDVA of 20/20 and an IOP of 12 mm Hg on a topical prostaglandin and β-blocker.

Case 5

A 65-year-old man was referred for partial IOL optic capture through the pupil and secondary uveitis. He had a history of traumatic aphakia

Figure 4. Iris fingerlike transillumination defects (*arrow*) correlating with IOL haptics or optic contact. Corresponding AS-OCT image suggestive of acute reverse pupillary block with posterior bowing of the iris.

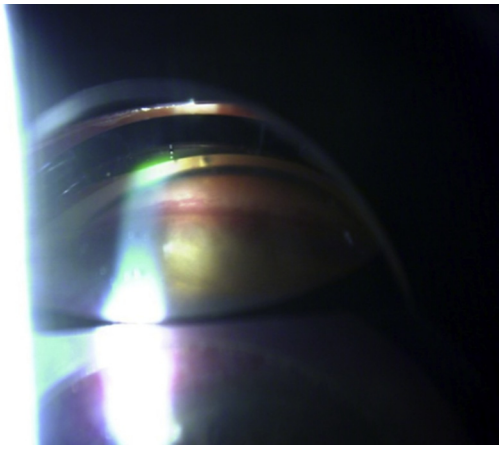


Figure 5. Gonioscopy image of the iridocorneal angle before iridotomy. The iris appears concave and deep with a thin layer of hyphema and pigmentation of the trabecular meshwork.

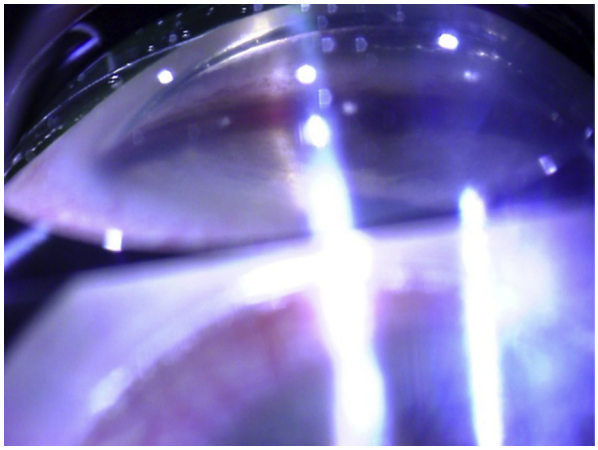


Figure 6. Gonioscopy image of the iridocorneal angle after iridotomy showing the flattened iris with resolution of reverse pupil block and iris concavity.

(AL 27.87 mm) in the left eye, with subsequent vitrectomy and secondary intrascleral haptic fixation of a 3-piece acrylic PC IOL 3 months prior to presentation. On examination, his CDVA was 20/80 and IOP was 20 mm Hg. Posterior bowing of the iris, 2+ pigment cells, iris transillumination defects, and uveitis were suggestive of reverse pupillary block.

The patient was treated with LPI, with resolution of the UGH syndrome and normalization of the iris position relative to the PC IOL. His CDVA was 20/50 and the IOP 15 mm Hg without antiglaucoma medication 18 months after LPI.

Case 6

A 56-year-old man was referred for UGH syndrome in the right eye; the AL was 28.21 mm. Pertinent history included phacoemulsification 18 months prior to presentation, complicated by posterior capsule rupture, and a 3-piece acrylic PC IOL placed in the sulcus. On presentation, he had a posteriorly displaced iris with pseudophakodonesis, iris transillumination defects, 2+ pigment cells, and uveitis. The patient's CDVA was 20/40, with an IOP of 18 mm Hg on 1 glaucoma medication class.

On diagnosis of reverse pupillary block, LPI was performed, with a resultant improvement in the iris profile. Twelve months after LPI, the patient's CDVA was 20/40 and the IOP 20 mm Hg without adjunctive antiglaucoma medication.

All Cases

Table 1 summarizes the demographic and clinical characteristics of study participants. The mean age of patients included in this case series was 59.8 years (range 43.0 to 66.0 years). They presented with unilateral UGH syndrome a mean of 28.7 months (range 0.3 to 84.0 months) after sulcus PC IOL implantation. All patients were myopic, and 5

Table 1. Patient demographics and clinical characteristics.

Pt	Sex	Age (Y)	Axial Length (mm)	Time Between Surgery and LPI (Mo)	Clinical Presentation
1	M	64	26.01	0.3	TID, PDS, concave iris bowing, elevated IOP, increased pigment deposition at the angle, ocular pain, blurred vision
2	M	43	28.46	60.0	TID, PDS, concave iris bowing, hyphema, elevated IOP, increased pigment deposition at the angle
3	F	66	25.84	7.0	TID, PDS, concave iris bowing, elevated IOP, increased pigment deposition at the angle
4	M	65	25.39	84.0	TID, PDS, concave iris bowing, elevated IOP, increased pigment deposition at the angle
5	M	65	27.87	3.0	TID, PDS, concave iris bowing
6	M	56	28.21	18.0	TID, PDS, concave iris bowing, elevated IOP, pseudophakodonesis

IOP = intraocular pressure; LPI = laser peripheral iridotomy; PDS = pigment dispersion syndrome; Pt = patient; TID = transillumination iris defect

(83.3%) had a history of retinal detachment requiring vitrectomy. Clinical presenting signs included posterior iris bowing (6 eyes; 100%), pigment dispersion and uveitis (6 eyes; 100%), iris transillumination defects (6 eyes; 100%), elevated IOP (5 eyes; 83.3%), and increased pigment deposition at the angle (4 eyes; 67.7%). One case of pseudophakodonesis was observed. Krukenberg spindles were absent. In each case, reverse pupillary block was diagnosed clinically with gonioscopy and confirmed with AS-OCT. Laser peripheral iridotomy was used to treat the reverse pupil block in all 6 eyes, with rapid improvement in reverse pupillary block and resolution of UGH syndrome in all cases. The mean follow-up was 31.0 months (range 12.0 to 48.0 months).

Table 2 compares the CDVA, IOP, and medication requirement before and after LPI. Two patients required ongoing glaucoma medication postoperatively.

DISCUSSION

Reverse pupillary block is a rare postoperative complication of cataract surgery that was first reported by Karickhoff[6] after IOL implantation in the ciliary sulcus. Herein, we presented 6 cases of pseudophakic reverse pupillary block causing UGH syndrome that were associated with elevated IOP and pigment dispersion and successfully treated with laser iridotomy. In our cases, posterior bowing of the iris and iris–IOL contact were primarily responsible for causing UGH syndrome. Contact between the square edge of the IOL optic or haptics in the sulcus and the posterior iris pigment epithelium led to pigment release or hyphema. This contributed to impaired aqueous flow through the trabecular meshwork and a subsequent IOP rise.

The UGH syndrome secondary to sulcus-placed IOLs, particularly single-piece acrylic IOLs, has been reported.[16] It is unclear whether pupil block was present in those cases, but the primary mechanism of UGH syndrome in those cases appears to be chafing of the posterior iris surface by the bulky haptics and the acrylic tacky surface of the IOL. The treatment in those cases required surgical intervention.

Table 2. Mean CDVA, IOP, and number of medication classes before and after LPI.

Parameter	Mean ± SD		*P* Value
	Pre-LPI	Post-LPI	
CDVA (logMAR)	0.6 ± 0.5	0.2 ± 0.2	.04*
IOP (mm Hg)	30.5 ± 10.0	15.5 ± 3.2	.04*
No. of medication classes	0.5 ± 0.8	0.7 ± 1.2	.564

CDVA = corrected distance visual acuity; IOP = intraocular pressure; LPI = laser peripheral iridectomy
*Significant at the 5% level

However, UGH syndrome in sulcus-placed IOLs can also present in conjunction with reverse pupillary block. To our knowledge, only rare cases of reverse pupillary block after sulcus-placed PC IOL implantation have been described.[6,12,15] Karickhoff[6] reported 2 cases of sulcus PC IOL that resulted in postoperative reverse pupillary block and pigment dispersion. One of the 2 cases included elevated IOP to 27 mm Hg. Both eyes were treated with short-term mydriatic agents that relieved the reverse pupillary block. One case required ongoing steroid therapy for 2 months. Laser iridotomy was not performed in those 2 cases. Moreover, Higashide et al.[12] described the use of AS-OCT for identifying the concave iris configuration in reverse pupillary block in a small case series of pupil capture of scleral-fixated PC IOLs. No description of PDS or iris transillumination was found in any of their cases. In contrast, all of our patients had evidence of UGH syndrome with increased IOP and pigment dispersion.

Reverse pupillary block after in-the-bag IOL implantation has also been described.[13,14] In a case report by Rhéaume et al.,[14] a patient developed secondary pigment dispersion syndrome associated with intermittent reverse pupillary block diagnosed 1 year after in-the-bag IOL implantation. The authors postulated that the large capsulorhexis exposed the IOL optic to come in contact with the iris pigment epithelium, where it was not covered by the anterior capsule. An LPI was performed, resulting in significantly diminished iris concavity. Itagaki et al.[13] also presented a case of reverse pupillary block diagnosed 1 day after in-the-bag IOL implantation with elevated IOP that persisted until laser iridotomy was performed to alleviate posterior bowing of the iris. In contrast to these 2 case reports describing reverse pupillary block after in-the-bag IOL implantation, all of our patients presented with reverse pupillary block and UGH syndrome after sulcus-placed PC IOL implantation.

In our case series, 3 patients (50%) had a scleral-fixated PC IOL. Scleral-fixated IOLs may be prone to IOL tilt and variable postoperative axial position, particularly more anteriorly than expected.[15,17–23] This can potentially increase the risk for iris contact, reverse pupil block, and UGH syndrome.

In classic phakic reverse pupillary block and PDS, anatomic risk factors, such as posterior iris insertion, a deeper anterior chamber, and large irides

relative to the anterior segment size, have been proposed as factors leading to increased iridolenticular contact.[9,24] However, using AS-OCT, Aptel et al.[25] analyzed the physiologic features of the iris in reverse pupillary block in patients with PDS and found no evidence of abnormally enlarged irides in susceptible eyes. They postulated that the increased predisposition of iridolenticular contact in PDS eyes was secondary to a more flaccid iris and to reverse pressure gradients across the anterior and posterior chamber.[25] For reverse pupillary block after in-the-bag IOL implantation, Itagaki et al.[13] also postulated the possible role of a flaccid iris that is prone to contact with the lens in PDS. A similar mechanism may be involved in reverse pupillary block after a sulcus-placed PC IOL, in which the flaccid iris acts as a valve preventing aqueous humor movement from the anterior to posterior chamber. This may be precipitated in myopic vitrectomized eyes.

In phakic PDS, risk factors include high myopia, young age, and male sex.[8,26] Notably, in the present study, all the patients were highly myopic and all except 1 had a history of vitrectomy, suggestive as major risk factors for PDS and reverse pupillary block in patients after sulcus-placed PC IOLs. Most of our patients (83.3%) were men, as seen in phakic PDS. Clinically, in our cases, many presenting features were similar to those seen in phakic PDS, such as iris transillumination defects and deposition of pigment on the trabecular meshwork. All patients had fingerlike transillumination defects. These can be described as resembling the "finger of God" sign due to their similarity to the popular painting "The Creation of Adam" by Michelangelo (Figure 7).[A] The finger of God sign can be considered iris stretch marks due to posterior distensive forces during episodes of reverse pupillary block that stretch the semielastic iris tissue, possibly along its anatomic planes. Iris contact with the IOL optic or haptics may also play a role in their appearance (Figure 4).

Laser iridotomy has been reported to be effective in relieving reverse pupillary block after PC IOL implantation.[12–14,24] All of the patients in our case series responded immediately to laser iridotomy, with improvements in vision and IOP. Contrary to the characteristic posterior-to-anterior rush of fluid immediately after LPI in traditional relative pupillary block, the breakthrough fluid rush in reverse pupillary block is in the reverse direction. Physiologically, this intraocular compartment syndrome, whereby differential pressures exist between the anterior and posterior chambers, the creation of an iridotomy allows rapid resolution of reverse pupillary block, as the pressures across the anterior and posterior segments then

Figure 7. Finger of God sign, reflecting the fingerlike transillumination defects commonly observed in reverse pupillary block.

freely equilibrate.[4] This restores the planar configuration of the iris, relieving posterior bowing and reverse pupillary block.[4,27–29] This in turn minimizes iridozonular contact and arrests pigment dispersion. Our post-LPI AS-OCT revealed successful flattening of the iris and widening of the space between the posterior iris and IOL. This was noted to be secondary to the forward shift of the iris and the stable IOL position in the posterior chamber after treatment. Laser peripheral iridotomy should be considered the initial therapy to manage UGH syndrome secondary to reverse pupillary block in sulcus-placed PC IOLs. This may be considered prior to surgical intervention, even in single-piece acrylic PC IOLs, although this was not studied in our report. In our series, this therapy was successful in all cases. However, in some cases, ongoing glaucoma medical therapy may be required due to chronic trabecular meshwork dysfunction or underlying primary open-angle glaucoma.

In conclusion, UGH syndrome due to reverse pupillary block can occur after sulcus-placed PC IOLs in susceptible patients, such as those with myopic or post-vitrectomized eyes. Laser peripheral iridotomy can treat the reverse pupillary block, which appears to have resolved the UGH syndrome in this case series

and may reduce the need for surgical intervention. Further studies are needed to address the incidence of UGH syndrome and reverse pupillary block after sulcus-placed PC IOL implantation and to delineate further causative mechanisms.

WHAT WAS KNOWN

- Reverse pupillary block can occur in eyes with sulcus-placed PC IOLs.
- Laser peripheral iridotomy can successfully treat reverse pupillary block.

WHAT THIS PAPER ADDS

- Uveitis-glaucoma-hyphema syndrome in sulcus-placed PC IOLs in certain susceptible eyes (axial myopia, post-vitrectomized) might develop due to reverse pupillary block.
- The fingerlike transillumination defects common to all patients in this case series, hereby termed finger of God sign, can be considered iris stretch marks, likely due to posterior distension forces during episodes of reverse pupillary block or iris contact with the IOL.
- Laser peripheral iridotomy can successfully treat the reverse pupillary block, which in turn appears to have resolved the UGH syndrome in this case series.

REFERENCES

1. Campbell DG. Pigmentary dispersion and glaucoma; a new theory. Arch Ophthalmol 1979; 97:1667–1672
2. Potash SD, Tello C, Liebmann J, Ritch R. Ultrasound biomicroscopy in pigment dispersion syndrome. Ophthalmology 1994; 101:332–339
3. Sowka J. Pigment dispersion syndrome and pigmentary glaucoma. Optometry 2004; 75:115–122
4. Karickhoff JR. Reverse pupillary block in pigmentary glaucoma: follow up and new developments [letter]. Ophthalmic Surg 1993; 24:562–563
5. Yang JW, Sakiyalak D, Krupin T. Pigmentary glaucoma. J Glaucoma 2001; 10(suppl 1):S30–S32
6. Karickhoff JR. Pigmentary dispersion syndrome and pigmentary glaucoma: a new mechanism concept, a new treatment, and a new technique. Ophthalmic Surg 1992; 23:269–277
7. Liebmann JM, Tello C, Chew S-J, Cohen H, Ritch R. Prevention of blinking alters iris configuration in pigment dispersion syndrome and in normal eyes. Ophthalmology 1995; 102:446–455
8. Niyadurupola N, Broadway DC. Pigment dispersion syndrome and pigmentary glaucoma—a major review. Clin Exp Ophthalmol 2008; 36:868–882
9. Davidson JA, Brubaker RF, Ilstrup DM. Dimensions of the anterior chamber in pigment dispersion syndrome. Arch Ophthalmol 1983; 101:81–83
10. Jensen PK, Nissen O, Kessing SV. Exercise and reversed pupillary block in pigmentary glaucoma. Am J Ophthalmol 1995; 120:110–112
11. Blumenthal M, Chen V. Reversed pupillary block [letter]. Ophthalmic Surg 1991; 22:304
12. Higashide T, Shimizu F, Nishimura A, Sugiyama K. Anterior segment optical coherence tomography findings of reverse pupillary block after scleral-fixated sutured posterior chamber intraocular lens implantation. J Cataract Refract Surg 2009; 35:1540–1547
13. Itagaki H, Kunikata T, Hiratsuka K, Saito J, Oshika T. Reverse pupillary block associated with pigment dispersion syndrome after in-the-bag intraocular lens implantation. J Cataract Refract Surg 2013; 39:1925–1928
14. Rhéaume M-A, Duperré J, Harasymowycz P, Thompson P. Pigment dispersion and recurrent hyphema associated with in-the-bag lens implantation. J Cataract Refract Surg 2009; 35:1464–1467
15. Corder DM, McDermott ML, Shin DH, Parrow KA, Lau-Sickon LK. Measured and predicted anterior chamber depth for transsceral suture-fixated posterior chamber intraocular lenses. Ophthalmic Surg 1993; 24:28–30
16. Chang DF, Masket S, Miller KM, Braga-Mele R, Little BC, Mamalis N, Oetting TA, Packer M; for the ASCRS Cataract Clinical Committee. Complications of sulcus placement of single-piece acrylic intraocular lenses; recommendations for backup IOL implantation following posterior capsule rupture. J Cataract Refract Surg 2009; 35:1445–1458
17. Mura JJ, Pavlin CJ, Condon GP, Belovay GW, Kranemann CF, Ishikawa H, Ahmed K II. Ultrasound biomicroscopic analysis of iris-sutured foldable posterior chamber intraocular lenses. Am J Ophthalmol 2010; 149:245–252
18. Sewelam A, Ismail AM, El Serogy H. Ultrasound biomicroscopy of haptic position after transscleral fixation of posterior chamber intraocular lenses. J Cataract Refract Surg 2001; 27:1418–1422
19. Loya N, Lichter H, Barash D, Goldenberg-Cohen N, Strassmann E, Weinberger D. Posterior chamber intraocular lens implantation after capsular tear: ultrasound biomicroscopy evaluation. J Cataract Refract Surg 2001; 27:1423–1427
20. Manabe S-I, Oh H, Amino K, Hata N, Yamakawa R. Ultrasound biomicroscopic analysis of posterior chamber intraocular lenses with transscleral sulcus suture. Ophthalmology 2000; 107:2172–2178
21. Steiner A, Steinhorst UH, Steiner M, Theischen M, Winter R. Ultraschallbiomikroskopie (UBM) zur Lokalisation der Kunstlinsenhaptik nach transskleraler Nahfixation [Ultrasound biomicroscopy (UBM) for the localization of artificial lens haptics after transscleral suture fixation]. Ophthalmologe 1997; 94:41–44
22. Pavlin CJ, Rootman D, Arshinoff S, Harasiewicz K, Foster FS. Determination of haptic position of transsclerally fixated posterior chamber intraocular lenses by ultrasound biomicroscopy. J Cataract Refract Surg 1993; 19:573–577
23. Kamal AM, Hanafy M, Ehsan A, Tomerak RH. Ultrasound biomicroscopy comparison of ab interno and ab externo scleral fixation of posterior chamber intraocular lenses. J Cataract Refract Surg 2009; 35:881–884
24. Inoue Y, Baba T, Nagayama M, Miyoshi T. [Reverse pupillary block after trans-scleral fixation of intraocular lens]. [Japanese]. Ganka 2004; 46:1899–1903
25. Aptel F, Beccat S, Fortoul V, Denis P. Biometric analysis of pigment dispersion syndrome using anterior segment optical coherence tomography. Ophthalmology 2011; 118:1563–1570
26. Siddiqui Y, Ten Hulzen RD, Cameron JD, Hodge DO, Johnson DH. What is the risk of developing pigmentary glaucoma from pigment dispersion syndrome? Am J Ophthalmol 2003; 135:794–799

27. Carassa RG, Bettin P, Fiori M, Brancato R. Nd:YAG laser iridotomy in pigment dispersion syndrome: an ultrasound biomicroscopic study. Br J Ophthalmol 1998; 82:150–153. Available at: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1722484/pdf/v082p00150.pdf. Accessed August 5, 2015
28. Scott A, Kotecha A, Bunce C, Balidis M, Garway-Heath DF, Miller MH, Wormald R. YAG laser peripheral iridotomy for the prevention of pigment dispersion glaucoma; a prospective, randomized, controlled trial. Ophthalmology 2011; 118:468–473
29. Breingan PJ, Esaki K, Ishikawa H, Liebmann JM, Greenfield DS, Ritch R. Iridolenticular contact decreases following laser iridotomy for pigment dispersion syndrome. Arch Ophthalmol 1999; 117:325–328. Available at: http://archopht.jamanetwork.com/article.aspx?articleid=411623. Accessed August 5, 2015

OTHER CITED MATERIAL

A. Wikipedia. The creation of Adam. Available at: http://en.wikipedia.org/wiki/The_Creation_of_Adam. Accessed August 5, 2015

First author:
Harmanjit Singh, MD, FRCSC

Department of Ophthalmology, Notre-Dame Hospital, Centre Hospitalier de l'Université, Montréal, Montreal, Quebec, Canada