KEY ISSUES IN
Glaucoma Management

A **CONTINUING EDUCATION** REVIEW FOR OPTOMETRISTS FROM THE NEW ENGLAND COLLEGE OF OPTOMETRY ISSUE 5

Primary Angle-closure Glaucoma: An Update for Optometrists

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Primary angle-closure glaucoma (PACG) is a sight-threatening but treatable disease. Greater awareness of PACG and more assertive use of gonioscopy for visualizing the iridocorneal angle could help preserve vision for a significant number of at-risk individuals.

Primary angle-closure glaucoma (PACG) affects 16 million people and is a major cause of blindness worldwide. Throughout Asia, PACG affects 0.75% of those over 40 years old.[1] The prevalence of PACG is higher than previously thought among populations of European descent, affecting about 0.4% of individuals with European ancestry.[2] Recent estimates suggest that PACG affects about 1.6 million people in Europe and 581,000 people of European descent in the US.[2] Among patients of African descent, PACG prevalence is lower, but the severity is often worse.[3]

As the global population expands and ages, the number of individuals affected by PACG is expected to reach 23 million by 2020 and 32 million by 2040.[4] The disease can have significant morbidity: about 25% of patients with PACG are blind in both eyes, a higher rate than is associated with primary open-angle forms of glaucoma.[5]

Gonioscopy, the most important diagnostic tool for assessing PACG risk, is vastly underused, resulting in a great many cases of PACG going undetected or being misdiagnosed as open-angle glaucoma. In a chart review of patients in treatment for primary open-angle glaucoma (POAG) in a community-based setting, only half had undergone gonioscopy to rule out a narrow angle at their initial visit.[6] This is unfortunate, since every undetected narrow angle is a missed opportunity to potentially treat the underlying glaucoma cause.

Optometrists see a large number of patients with glaucoma, many who have not yet undergone baseline gonioscopy and may have undetected angle closure. Optometrists play a vital role to identify patients with narrow angles, refer to specialists in a timely manner, manage acute episodes of angle closure, and co-manage patients with established chronic angle closure (see Box, "Optometrists' Role...").

CATEGORIZATION

Angle closure refers to appositional or synechial closure of the anterior chamber angle resulting in obstruction of aqueous fluid outflow. Patients with angle closure are at a significant risk for development of elevated intra-

OPTOMETRISTS' ROLE IN PREVENTING AND MANAGING ANGLE-CLOSURE GLAUCOMA

- Evaluate patient risk for PACG
- Identify and accurately diagnose patients with PACS, PAC, and PACG
- Counsel patients on visual complications of PACG and availability of treatment
- Counsel patients regarding symptoms of acute angle-closure crisis
- Make appropriate referrals to surgical specialists or patient's primary care practitioner
- Manage acute attack of PACG
- Monitor and co-manage patients in treatment for PAC and PACG
- Improve the quality of care rendered to patients with PACG

Source: Care of the Patient with Primary Angle Closure Glaucoma: Optometric Clinical Practice Guidelines. http://www.aoa.org/documents/optometrists/CPG-5.pdf. Accessed May 6, 2016.

More INSIDE:
Secondary Open-angle Glaucoma Detection and Management
Daniel K. Roberts, OD, PhD

TARGET AUDIENCE This educational activity is intended for optometrists.

LEARNING OBJECTIVES Upon completion of this activity, participants will be able to:

1. Diagnose and classify angle closure, angle-closure suspect, and PACG.
2. Provide better care to patients at risk for angle closure.
3. Review key clinical features of common types of secondary open-angle glaucoma.
4. Improve detection of secondary open-angle glaucoma based on history and clinical examination.

Key Issues in Glaucoma Management: A Review for Optometrists is sponsored by the New England College of Optometry and supported by an unrestricted educational grant from Bausch + Lomb, Inc. This publication is administered by an independent editorial committee.

Supported by an unrestricted educational grant from Bausch + Lomb, Inc.

ocular pressure (IOP)—either acute or chronic—and resultant glaucomatous damage to the optic nerve.[7] According to recent schemes, patients with angle closure may be categorized as: (1) primary angle-closure suspect (PACS), (2) primary angle closure (PAC), or (3) primary angle-closure glaucoma (PACG).[8] Proper categorization of the type and degree of angle closure is necessary for choosing the appropriate treatment path (Table I).[7]

PACS describes an individual whose trabecular meshwork is not visible for at least 180 degrees on gonioscopy, indicating at least 180 degrees of irido-trabecular contact. These patients are said to have a narrow angle. PACS patients show no evidence of peripheral anterior synechiae (PAS), which are the result of repeated irido-trabecular contact and long-term flow obstruction; nor do they have elevated IOP or evidence of optic nerve damage.[7,8] Many such patients will not go on to develop angle closure; therefore, management of the PACS patient typically involves careful IOP monitoring and serial gonioscopy to asses any changes in the angle.[7]

TABLE I ANGLE CLOSURE CLASSIFICATION

	Iridotrabecular contact (≥180°)	Elevated IOP	PAS	Glaucomatous optic neuropathy
PACS	Present	Absent	Absent	Absent
PAC	Present	At least 1 present		Absent
PACG	Present	At least 1 present		Present

IOP: intraocular pressure; PAC: primary angle closure; PACG: primary angle-closure glaucoma; PACS: primary angle-closure suspect; PAS: peripheral anterior synechiae

Sources: Emanuel ME, et al. *Curr Opin Ophthalmol*. 2014;25:89-92.; Foster PJ, et al. *Br J Ophthalmol*. 2002;86:238-42.

Patients with PAC have a closed angle and evidence of chronic damage in the form of PAS, elevated IOP, or both; alternatively, they may have a history of acute angle closure that resolved without observable damage to the optic nerve.[8] Treatment of PAC aims to prevent progression to glaucoma; laser peripheral iridotomy (LPI)—a hole placed in the iris for fluid to bypass pupil block—is generally indicated, but it must be weighed against associated risks, including increased IOP, corneal decompensation, cataract progression, and visual disturbance.[7,9]

The third and most critical category is PACG, in which there is a closed angle on gonioscopy and either evidence of damage to the optic nerve, visual field abnormality, or both. Such patients may need either LPI, iridectomy, or lens extraction to open the angle; they may also require medical or surgical management of their glaucoma (Figure 1).[7] There is increasing support for the use of lens extraction—either clear lens or cataract—as a means for anatomical opening of the irido-trabecular angle, increasing anterior chamber depth, and as an alternative to iridectomy in patients with PAC or PACG for reducing IOP.[10,11]

The above categorization scheme pertains to primary, chronic angle closure. "Primary" refers to the observation that no discernible cause has been identified; by contrast, "secondary" refers to angle closure that is due to a known ocular pathologic triggering event, such as inflammation, neovascularization, trauma, or a lens-related disorder.[8] Angle closure may also be cat-

KEY ISSUES IN GLAUCOMA MANAGEMENT — Issue 5

STATEMENT OF NEED

Glaucoma, a group of ocular diseases characterized by progressive damage to the optic nerve, is the second leading cause of blindness worldwide. It affects a significant and growing portion of the US population.[1,2]

As primary eyecare providers, medical optometrists are well positioned to identify patients at risk and to diagnose, monitor, and treat glaucoma. However, given that the expanded scope of practice incorporating glaucoma treatment is relatively new, many optometrists lack confidence in their ability to treat this potentially blinding disease. In order to instill confidence and help optometrists make sound clinical judgments about the care of glaucoma patients, *Key Issues in Glaucoma Management* will help optometrists better understand the various aspects and nuances of the disease, including our current understanding of the role of intraocular pressure (IOP) in glaucomatous optic nerve damage. Course content will also include current rationale on glaucoma diagnosis and evidence-based strategies for reducing IOP.

Each installment of *Key Issues in Glaucoma Management* will look at an important topic in glaucoma diagnosis or therapy. Each issue will build from a basic level to instill understanding and confidence in medical optometrists. *Key Issues in Glaucoma Management* aims to support optometrists' clinical reasoning and decision-making abilities and help them turn medical management of glaucoma into a vital segment of their practices.

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2. Eye Diseases Prevalence Research Group. Prevalence of open-angle glaucoma among adults in the United States. *Arch Ophthalmol*. 2004;122:532-8.

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DATE OF ORIGINAL RELEASE

June 2016. Approved for a period of 24 months.

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egorized as either acute or chronic, both of which can lead to permanent damage to the optic nerve and vision loss at different rates. Acute angle closure, or acute angle-closure crisis, is a sudden or severe closure of the irido-trabecular angle characterized by a dramatic IOP spike as well as ocular and systemic symptoms, which may include decreased or blurred vision, halos around lights, pain, redness, tearing, photophobia, headache, nausea, and vomiting.[8,12]

Chronic angle closure is more common, accounting for 70% to 80% of angle-closure cases, and is more insidious.[5] It is associated with milder fluctuations in IOP and no symptoms, so it commonly goes undetected until the development of ocular hypertension and/or frank glaucoma.

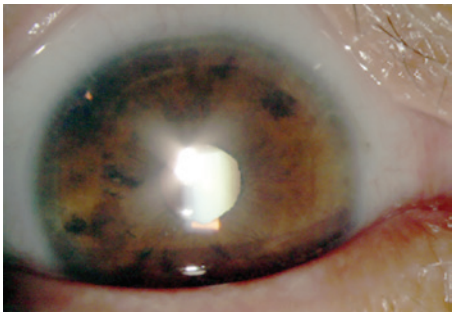

FIGURE 1 LPI may be effective by equilibrating pressure on either side of the iris. *(Source: Image courtesy Dr. Hoguet.)*

RISK FACTORS

Patients of advanced age, female sex, and East Asian ethnicity have higher rates of angle closure compared with other populations.[13,14] Anatomical risk factors include shorter axial length, shallower anterior chamber, and relatively large or anteriorly positioned lens.[5,14]

Angle closure is generally considered to occur by one of two mechanisms: pupillary block or plateau iris. Pupillary block, the more common form, involves an anterior bowing of the pupillary portion of the iris at mid-dilation, closing the angle and leading to buildup of aqueous in the posterior chamber.[14] Patients with a shallow anterior chamber or thicker than average lens are predisposed to pupillary block. Plateau iris is thought to occur due to a more anterior positioning of the ciliary body, which infringes upon the angle and causes a block. It is associated with emmetropia and is more common among young, female patients.

Most individuals with demographic and/or anatomic risk factors do not develop angle closure, an observation that has prompted investigation into what else may contribute to the development of PACG. It may be that the pathophysiology of primary angle closure is more dynamic and complex than previously appreciated. For example, dysfunction of the iris (eg, tendency toward increased iris volume) and choroidal tissues (eg, dysregulation of choroidal expansion) may play significant roles in angle closure.[5] A "spongier" iris, ie, one that retains higher than average fluid volume on pupillary dilation, is a common feature among individuals of Asian descent and may contribute to their higher incidence of angle-closure disorders.[15]

As PACG is a major cause of blindness, and since population-based studies suggest familial- and ethnicity-based predisposition, there is a lot of interest in uncovering the genetic underpinnings of PACG.[16,17] Conceivably, finding the gene or genes responsible would facilitate greater understanding the disease itself and pave the way for genetic risk assessment (for family members of affected individuals or other at-risk patients) or novel treatments. Studies to date suggest a complex pattern of inheritance for PACG rather than a single gene. Researchers recently identified five new genes and confirmed three others that may play a role in PACG, including a gene encoding choline acetyltransferase (the enzyme that catalyzes synthesis of pupil-constricting neurotransmitter acetylcholine), two that may play a role in cell–cell adhesion, and one that may be activated by the female hormone estradiol.[18]

DIAGNOSIS

Early detection of angle closure prior to the development of PACG is crucial to preventing vision loss. Ideally, patients at risk for PACG (and those believed to have POAG or are open-angle glaucoma suspects) should undergo examination to assess the angle using indentation gonioscopy, the gold standard for detection of angle closure and identification of PAS. Other tools, such as ultrasound biomicroscopy or optical coherence tomography, are increasingly used to evaluate anterior segment structures and contributing factors such as iris cysts or plateau iris, but these tools should not be relied upon to make the diagnosis of angle closure.

Patients meeting the gonioscopic criteria for PACS—drainage structure that cannot be visualized for at least 180 degrees—should be referred to a specialist for further evaluation of narrow angle. Patients with PAC or PACG should also be referred for further evaluation

CORE CONCEPTS

- PACG, a leading cause of blindness in the world, is underdiagnosed, and prevalence is expected to rise rapidly in upcoming decades.
- Optometrists play a vital role in diagnosing glaucoma, identifying patients with narrow angles, making appropriate and timely referrals, managing acute angle closure, and co-managing patients in treatment for chronic angle closure.
- Risk factors for angle closure include Asian ethnicity, female sex, advanced age, family history, and crowding of anatomic features within the anterior segment.
- Genetics likely plays a role in PACG etiology.
- Most patients with risk factors for PACG, including those considered PACS, do not develop advanced disease; however, progression occurs in a substantial minority who go untreated.
- Patients with glaucoma and glaucoma suspects should undergo gonioscopy to rule out angle closure. Don't assume the angle is open in patients with glaucoma: always check.

and surgical treatment. Before leaving the office, all narrow-angle and closed-angle patients should be counseled about signs and symptoms of acute angle closure and advised to seek medical care immediately should they occur.

CHRONIC ANGLE CLOSURE

When caught early, most cases of angle closure can be essentially "cured" via iridotomy, and optic nerve damage can be prevented (Figure 2). Medical therapy is of limited value in the management of patients with PAC. Attempts to open the angle using topical pilocarpine are often complicated by poor tolerability, low patient compliance, and risk for retinal detachment.

Optometrists play an important role in co-managing patients following iridotomy, iridectomy, or other surgical interventions. Follow-up may include IOP monitoring and repeated gonioscopy to assess the angle. Patients with concerning IOP elevations should be referred back to a specialist for advanced medical management of their glaucoma.

ACUTE ANGLE CLOSURE

Patients who present with acute angle closure need immediate attention to open the angle and lower IOP. Sometimes gonioscopy itself may be therapeutic in breaking the episode; applying light pressure to the globe with placement of the gonial lens can mechanically open up the angle and should be attempted. Acute medical management may include topical cholinergic agents (pilocarpine 1% to 2%), β-adrenergic antagonists, α2-adrenergic agonists, or prostaglandin analogs; oral, topical, or intravenous carbonic anhydrase inhibitors and/or a hyperosmotic agent may be administered orally or intravenously, avoiding any patient-specific contraindications.[12,19] More specific guidelines for emergency management of acute angle-closure crisis are available at and AOA.org and AAO.org.

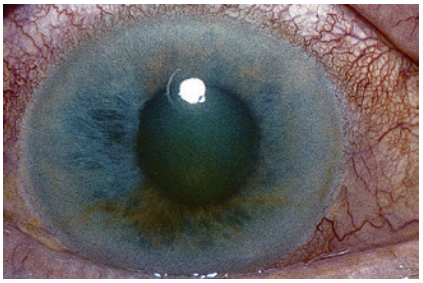

FIGURE 2 Optometrists should counsel patients at risk regarding the signs and symptoms of acute angle-closure crisis and be prepared to treat in case of emergency. *©2016 American Academy of Ophthalmology*

If IOP reduction is achieved, the eye should be evaluated for laser or surgical iridectomy, which is typically delayed for several days to allow for inflammation associated with the acute episode to subside. If the IOP does not respond to medical therapy, immediate iridectomy may be necessary. A thorough evaluation of the unaffected eye is also important; prophylactic iridectomy may be indicated since narrow angles tend to occur bilaterally.

CONCLUSION

By 2040, PACG will affect 32 million people worldwide and be responsible for half of cases of blindness-inducing glaucoma. Treatment is surgical and distinct from POAG treatment. The need for more aggressive use of gonioscopy to detect PACG and at-risk patients cannot be overstated.

Ambika Hoguet, MD, is an attending physician at Massachusetts Eye and Ear and an instructor of ophthalmology at Harvard Medical School. Dr. Hoguet has no relevant disclosures. Medical writer Noelle Lake, MD, assisted in the preparation of this manuscript.

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Secondary Open-angle Glaucoma Detection and Management

DANIEL K. ROBERTS, OD, PHD

The first and perhaps most important step in managing secondary open-angle glaucoma is to recognize the disease—despite its various forms and often subtle signs.

While the most common form of glaucoma is primary open-angle glaucoma (POAG), in which the cause is unknown, there is also a large group of secondary glaucomas resulting from an underlying condition that reduces the outflow of the aqueous, causing elevated intraocular pressure (IOP) in the presence of an open anterior chamber angle. Two common causes of secondary open-angle glaucoma are pigment dispersion syndrome and exfoliation syndrome (also known as pseudoexfoliation syndrome). Other secondary glaucomas that are likely to be encountered in a general optometric practice include those secondary to trauma, Fuchs' uveitis syndrome (ie, Fuchs' heterochromic iridocyclitis), and glaucomatocyclitic crisis (ie, Posner-Schlossman syndrome).

As first-line eye care providers, optometrists are in the perfect position to screen for glaucoma and its underlying causes. Identifying secondary open-angle glaucoma is crucial, both for appropriate treatment of the underlying condition but also because POAG is a diagnosis of exclusion. Secondary open-angle glaucoma often presents with characteristic signs, but these can be subtle and even shared between different secondary glaucoma types. To avoid misdiagnosis and delay in treatment, it is important to remain mindful

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of the distinctive forms of secondary open-angle glaucoma while carefully performing clinical examinations.

EXFOLIATION SYNDROME VS PIGMENT DISPERSION SYNDROME

Both exfoliation syndrome and pigment dispersion syndrome may cause pigment dispersion in the anterior chamber, but they must be distinguished from each other. Exfoliation syndrome, which does not always lead to elevated IOP, typically occurs in individuals older than 65 years of age.[1] The condition is characterized by deposition of dandruff-like white material on the structures in the anterior segment including the back of the cornea, the chamber angle, and the lens. The small granular flakes may first be noticed along the pupil border prior to pupil dilation. On the anterior lens capsule, the deposits commonly assume a target-like or "bull's eye" pattern that is seen with pupil dilation. Increased patchy, irregular pigmentation of the trabecular meshwork is typical in patients with exfoliation syndrome, which is caused by rubbing of the back of the iris against the rather coarse anterior lens surface created by deposits of the proteinaceous granular amyloid material.

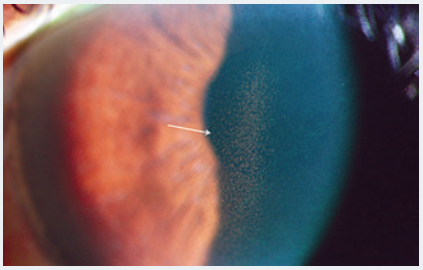

FIGURE 1 Krukenberg spindle (arrow) occurring with pigment dispersion syndrome. *(Image courtesy of Dr. Roberts.)*

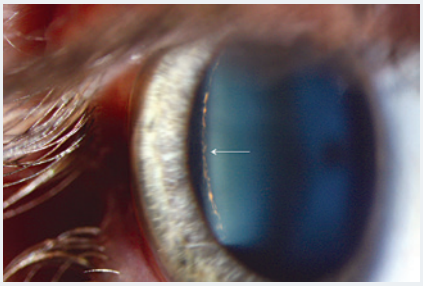

FIGURE 2 Pigment deposition (arrow) along the equatorial/posterior lens capsule (Scheie line) in pigment dispersion syndrome. *(Image courtesy of Dr. Roberts.)*

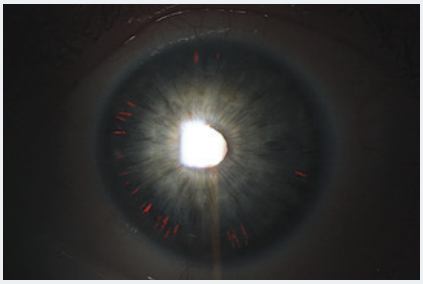

FIGURE 3 Slit-like iris transillumination defects in pigment dispersion syndrome, seen with conventional slit-lamp examination. *(Image courtesy of Dr. Roberts.)*

Patients with pigment dispersion syndrome are very frequently young or middle-aged male adults who have myopia. Because primary pigment dispersion syndrome may be inherited in an autosomal dominant fashion, these patients often have a family member that is also affected.[2] Classic-presenting pigment dispersion syndrome is more common in young myopic males, is generally bilateral, and is characterized by Krukenberg spindles—a fine pigment deposition in a vertical spindle shape on the central corneal endothelium (Figure 1). Increased, somewhat more homogenous pigmentation of the trabecular meshwork is a classic finding of pigment dispersion syndrome and a phenomenon that may be associated with the mechanism of IOP elevation in pigmentary glaucoma. One often overlooked yet pathognomonic sign for pigment dispersion syndrome is a Scheie line or Zentmayer's ring, a pattern of pigment deposits along the equatorial/posterior lens surface where zonular fibers attach and where the anterior hyaloid face attaches to the posterior lens surface (Figure 2).[3] Another hallmark sign of pigment dispersion syndrome is iris transillumination defects (Figure 3). These slit-like transillumination defects can be extremely subtle; in dark brown irides, in particular, it is likely that they can only be observed with special techniques such as infrared iris imaging (Figure 4).[4,5]

Pigment dispersion syndrome can be associated with long anterior zonules, a trait possibly caused by gene mutation.[6,7] Long anterior zonule-associated pigment dispersion is a relatively common but less well-known condition that may be confused with the "classic" variety of pigment dispersion syndrome.[8,9] The condition is not well documented in the literature; but in clinical practice, the long anterior zonule trait may be the most likely cause of Krukenberg spindles.[10] In cases where Krukenberg spindles are present, therefore, one should dilate the pupil and carefully examine the anterior surface of the lens for radially oriented fibers that represent longer-than-normal lens zonules (Figure 5). Additionally, post-dilation pressure spiking resulting from increased liberation of pigment following iris movement has been reported. Therefore, the clinician should re-measure IOP following dilation before dismissing patients with pigment dispersion syndrome.[11] Exercise induced

CORE CONCEPTS

- Secondary open-angle glaucoma is less common than POAG, but the possibility of secondary glaucoma must be ruled out before making the diagnosis of POAG.
- There are a range of secondary causes of open-angle glaucoma, the common ones being exfoliation syndrome, pigment dispersion, trauma, Fuchs' uveitis, and Posner-Schlossman syndrome.
- Secondary open-angle glaucoma often has characteristic yet hidden signs. A thorough history and ocular examination is vital in differentiating between the primary and secondary forms of glaucoma and telling different secondary glaucoma types apart.
- All glaucoma suspects should receive gonioscopy. A main goal of this basic examination technique is not just to differentiate between open- and narrow-angle glaucoma, but to identify any potential signs of secondary glaucoma.
- Medical therapy is the initial treatment for most secondary open-angle glaucomas. Results of the treatment, however, may vary depending upon the type and course of glaucoma.

IOP elevation has also been reported in patients with pigment dispersion.[12]

TRAUMATIC GLAUCOMA

Traumatic glaucoma is typically unilateral. A history of blunt ocular trauma is an important piece of diagnostic history, but the contributing injury may be forgotten by the patient, and it is not uncommon for patients to deny previous trauma in the presence of overt clinical evidence that indicates otherwise. Notably, many patients with traumatic glaucoma may go on to develop open-angle glaucoma in the contralateral eye.[13] It is likely that these are patients who are predisposed to the disease.

When examining the patient, one sign of previous trauma may be an irregular pupil or notching of the pupillary border, which signals a tear in the iris sphincter muscle. Another possible lead is the presence of traumatic cataract. Classic trauma-caused cataract has a distinctive rosette or petalloid shape and a grainy or iridescent appearance. The anterior cortex of the lens is often involved, and though the cataract often only involves a sector of the lens, they may occupy the entire circumference of the lens.

Another indication of traumatic glaucoma is recession of the anterior chamber angle, which usually occurs as a result of a tear between the longitudinal and circular ciliary muscle fibers. With gonioscopy, angle recession may appear as a wider than normal ciliary body band. Some patients may also show an iridodialysis, a tearing away of the peripheral iris root from the ciliary body (Figure 6). An acute angle recession is often accompanied by hyphema, and while not a requisite for angle recession, hyphema should be viewed as an important correlate. After an acute angle recession, some patients may eventually demonstrate peripheral anterior synechia in the affected region, which may obscure a recession and make accurate diagnosis more difficult. Angle recession can sometimes be very subtle and easy to overlook, especially if it occurred years ago. In suspicious cases, where an angle recession may not be as readily apparent, it is important to carefully compare the iridocorneal angle of the affected eye to the corresponding angle in the fellow eye—ie, the nasal angle of the affected eye would need to be compared to the nasal angle of the other eye.

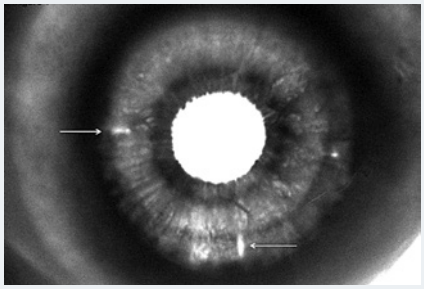

FIGURE 4 Iris transillumination defects (arrows) in a dark-brown iris, which could be visualized only with infrared examination. *(Image courtesy of Dr. Roberts.)*

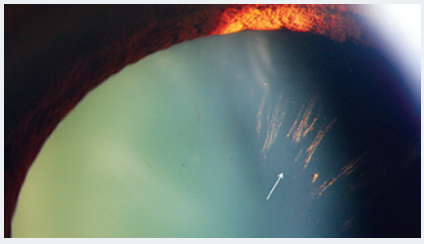

FIGURE 5 Long anterior lens zonules (arrow) on the anterior lens surface may rub against the back of the iris, becoming pigmented and also causing pigment dispersion signs within the anterior segment. *(Image courtesy of Dr. Roberts.)*

FIGURE 6 Gonioscopic view of a traumatic angle recession and an associated iridodialysis where the iris root has torn away from the ciliary body. Ciliary processes can be seen through the opening. The arrow points to the ciliary body band of the iridocorneal angle. *(Image courtesy of Dr. Roberts.)*

OTHER SECONDARY OPEN-ANGLE GLAUCOMAS

Fuchs' heterochromic iridocyclitis, ie, Fuchs' uveitis syndrome, is a chronic form of iridocyclitis that usually affects young to middle-aged adults.[14] The condition is typically unilateral, and, as its name suggests, the involved eye can have a different color than the opposite one due to iris stromal atrophy and loss of pigment. In reality, the heterochromia of the iris may be difficult to detect even with careful observation. Iris anterior stromal atrophy, instead, may be easier to identify; thus, the iris stromal architecture in the affected eye should be carefully compared to the fellow eye under high magnification. Patients with Fuchs' uveitis typically have mild anterior chamber reaction, with fine, diffusely scattered stellate keratic precipitates. Some may present with posterior subcapsular cataract and/or secondary glaucoma. Both complications eventually occur in a high percentage of people with Fuchs'.

Glaucomatocyclitic crisis is another form of inflammatory glaucoma that typically occurs in middle-aged individuals.[15] Patients may present with recurrent attacks characterized by mild unilateral eye pain, parilimbal conjunctival and episcleral injection, mild anterior chamber cell and flare, and elevated IOP. The increase in IOP can vary markedly in magnitude, with pressures frequently reaching 40 mm Hg or even higher. Corneal edema may be present with very high IOPs. When clinical findings point to glaucomatocyclitic crisis, it is important to inquire about the patient's history of previous episodes of increased pressure. Treatment includes topical steroids, topical antiglaucoma drops, or, sometimes, topical or oral NSAIDS. Recurrent attacks of the condition may increase the risk of chronic open-angle glaucoma—it is possible that those who develop a chronic glaucoma secondarily are predisposed to the disease.

THE ROLE OF GONIOSCOPY

Diagnosing secondary open-angle glaucoma can be challenging, and it is important to obtain a detailed history and to perform a comprehensive dilated eye exam. One should ask the patient about factors that might contribute to glaucoma, such as previous ocular trauma or inflammation and history of topical or oral corticosteroid use. Given that POAG is a bilateral condition (albeit asymmetric in some patients) and certain secondary glaucomas are more likely monocular, the clinician should especially consider possible secondary causes any time a patient presents with unilateral glaucoma.

Gonioscopy is especially important in cases of secondary open-angle glau-

coma because it can provide valuable diagnostic information. Clinicians should perform the exam routinely when evaluating patients suspected of glaucoma. In reality, though, the technique is severely underused: studies have shown that barely half of patients with open-angle glaucoma had gonioscopy performed at their initial presentation.[16,17] Many practitioners think of performing gonioscopy primarily to determine whether the anterior chamber angle is open or not, when in fact it is also very useful in differentiating between primary open-angle glaucoma and other secondary open-angle forms of glaucoma. It is true of course that gonioscopy must also be performed to rule out a narrow angle, even when not anticipated based on the van Herick test, but most patients with wide chamber angles measured with the van Herick test will also have open angles when viewed gonioscopically. Nonetheless, even when one is fairly certain that the anterior chamber is wide open based on standard slit-lamp assessment using the van Herick test, gonioscopy should still be performed when indicated, not only to definitively rule out a narrow angle but to help rule out secondary forms of open-angle glaucoma.

Occasionally, unusual findings may be encountered by the clinician when performing gonioscopy. Thus, in addition to verifying an open iridocorneal angle with gonioscopy, the clinician should therefore put at least equal emphasis on looking for signs of secondary open-angle glaucoma, including the presence of angle recession, abnormal pigmentation, peripheral anterior synechia, goniodysgenesis, or other congenital abnormalities. This is especially true in the presence of elevated IOP in the presence of an open angle.

MANAGEMENT

Overall, secondary open-angle glaucoma is treated similar to primary open-angle glaucoma, with the goal of preventing optic nerve damage through IOP reduction. Classes of ocular hypertensive medications that are commonly used include prostaglandins, beta-blockers, and carbonic anhydrase inhibitors. That said, open-angle glaucoma can be secondary to a number of conditions, and treatment strategies may vary depending on the primary disease. In some cases, treating the primary disease eliminates or reduces the secondary glaucomatous response.

EXFOLIATIVE AND PIGMENTARY GLAUCOMA

Exfoliative glaucoma can be difficult to treat.[18] Many patients with the condition require aggressive medical therapy with multiple drug classes; some will progress to a point where surgical intervention becomes necessary.

Medical therapy is often successful in controlling pigmentary glaucoma. Some patients, however, may become recalcitrant to therapy. Usually these are patients who have severe forms of pigmentary glaucoma or have had pigment dispersion for a long period of time. These patients begin to develop trabecular meshwork scarring, which leads to collapse of the intra-trabecular spaces and thus obstruction of aqueous outflow. When much of the trabecular meshwork function is lost to scarring, pigmentary glaucoma becomes extremely difficult to control.

One potential but still understudied treatment for pigmentary glaucoma is laser iridotomy.[19] The procedure may help relieve reverse pupillary block, a characteristic feature of eyes with pigmentary glaucoma whereby a higher pressure in the anterior chamber relative to the posterior chamber causes the iris to bow backward.[20] This posterior bowing of the peripheral iris may contribute to rubbing of lens zonules against the posterior iris surface and hence dispersion of pigment granules.[21] It seems reasonable to believe that a laser iridotomy should be helpful for at least some patients affected by pigmentary glaucoma, but more clinical studies are needed to clearly establish its long-term effectiveness when applied to diverse types of patients with variable disease presentations.

OTHERS

Patients with glaucomatocyclitic crisis usually only need to use IOP-lowering drops temporarily in conjunction with concurrent topical antiinflammatory drops to address the underlying inflammatory etiology. Inflammatory episodes will in fact usually resolve without treatment, with IOP returning to normal on its own. Treatment, however, greatly hastens recovery and helps protect the optic nerve against high IOP that may have cumulative effects after repeated episodes of the inflammatory-induced attacks.

For patients with Fuchs' uveitis, antiinflammatory therapy may be effective in some cases depending on the degree of associated inflammation, but long-term corticosteroid treatment may actually hasten the development of glaucoma and posterior subcapsular cataract. For this reason, patients who have Fuchs' uveitic syndrome are often simply observed, especially if they are asymptomatic. Some patients may require treatment only for periods of time depending on the severity of symptoms, which include pain and discomfort. If the patient develops secondary glaucoma, then topical antiglaucoma drops can be used to control IOP. Since prostaglandins could potentially exacerbate intraocular inflammation, the medical treatment usually starts with aqueous suppressants such as topical beta-blockers.[22]

Because secondary open-angle glaucoma is not as common as POAG, exact etiologic diagnosis is often more elusive and may be clinically challenging. Although there is anecdotal evidence that one class of IOP-lowering agents may be better than another, we do not have sufficient evidence-based data to support which drug class is always best for which disease. It is anticipated that new classes of glaucoma medications will become available in the near future. Because these agents have novel and often combined mechanisms of action, they would be a great addition to the therapeutic options for glaucoma patients. However, as with the current medications, their actual benefit in the treatment of secondary open-angle glaucoma may very much depend on the specific type of the disease.

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Turn to page 9 for ROBERTS REFERENCES.

EXAMINATION ANSWER SHEET — KEY ISSUES IN GLAUCOMA MANAGEMENT — Issue 5

This CE activity is sponsored by the New England College of Optometry and is supported by an unrestricted educational grant from Bausch + Lomb, Inc. Submit your answers to the below test online by visiting **http://www.neco.edu/academics/continuing-education/online-ce/key-issues-glaucoma**. You may also access the online test by scanning the QR code on the right. The New England College of Optometry designates this activity for a maximum of 1 hour of COPE-approved continuing education credit. There is no fee to participate in this activity. In order to receive CE credit, participants should read the report and then take the posttest. A score of 70% is required to qualify for CE credit. Estimated time to complete the activity is 60 minutes. CE exam expires May 31, 2018.

COPE ID#: 49801-GL
Event #: 111558

1. Which of the following diagnostic tools is most important to help differentiate between primary open-angle glaucoma and secondary open-angle glaucoma according to Dr. Roberts?
   A. Visual field testing
   B. Gonioscopy
   C. OCT
   D. Corneal pachymetry

2. Which of the following is the gold standard for detecting a narrow angle?
   A. UBM
   B. OCT
   C. Gonioscopy
   D. Point-of-care genetic testing

3. Genetic studies in PACG may be useful for:
   A. Providing clues to pathophysiologic mechanisms
   B. Identifying risk factors
   C. Both A and B
   D. None of the above

4. What is the most likely diagnosis for a patient with high IOP, optic nerve damage, and 155 degrees of iridotrabecular contact on gonioscopy?
   A. POAG
   B. PACG
   C. PAC
   D. PACS

5. Which of the following findings does NOT support the diagnosis of traumatic glaucoma?
   A. Angle recession
   B. "Notching" of the pupil border
   C. Persistent pupillary strands and membranes
   D. Rosette cataract

6. Which of the following is NOT a risk factor for PACG?
   A. Female sex
   B. Tall stature
   C. East Asian ethnicity
   D. Advanced age

7. Which of the following secondary glaucoma types may be accompanied by low-grade inflammatory reaction in the anterior chamber?
   A. Fuchs' syndrome
   B. Exfoliation syndrome
   C. Possner-Schlossman syndrome
   D. Both A and C

8. All of the following are symptoms of angle closure except:
   A. Haloes around lights
   B. Blepharospasm
   C. Vomiting
   D. Photophobia

9. Which of the following findings is/are characteristic of classic pigment dispersion syndrome?
   A. Scheie line
   B. Krukenberg spindles
   C. Iris transillumination defects
   D. All of the above

10. Which of the following statements is true about the treatment of secondary glaucoma?
   A. Attacks of glaucomatocyclitic crisis may resolve without treatment
   B. Medical treatment of Fuchs' uveitic syndrome starts with aqueous suppressants if IOP is elevated
   C. Traditional IOP-lowering medications may be less effective for exfoliative glaucoma
   D. All of the above

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