Pseudoexfoliation and Pigmentary Glaucomas

Kimberly V. Miller, MD

Open-angle glaucoma secondary to pseudoexfoliation or pigment dispersion syndrome is often challenging to diagnose and treat. With timely detection and appropriate management, however, these types of glaucoma can be effectively controlled to prevent vision loss.

Pseudoexfoliation (PXF) syndrome and pigment dispersion syndrome are two conditions that are frequently associated with open-angle glaucoma. PXF syndrome, in particular, is considered the most common cause of secondary open-angle glaucoma. First described in 1917 in a Finnish population, PXF syndrome is prevalent in Scandinavian countries. In the US population, the prevalence of PXF syndrome is about 2.6% for ages 65 to 74 years and 5.0% for ages 75 to 85 years.

The rates of glaucoma associated with PXF or pigment dispersion syndrome vary depending on the location of a practice and patient demographics. In some practices depending on the ethnic backgrounds of the patients, PXF glaucoma can account for nearly 30% of glaucoma cases, and pigmentary glaucoma accounts for about 1%. Both PXF and pigmentary glaucomas are insidious in nature; they share many similarities in regard to disease mechanism and ocular manifestations. When managing patients suspected of glaucoma, it is important to be on the lookout for both of the conditions and distinguish them from each other.

MECHANISMS OF IOP ELEVATION

Both PXF and pigment dispersion syndrome are late-onset genetic disorders. PXF is a systemic disease of the basement membrane without a clear inheritance pattern. It has recently been strongly associated with the lysyl oxidase-like 1 gene (LOXL1). In the eye, where the condition is most easily diagnosed, the epithelial cells of the ciliary body, iris, and lens capsule slough off their basement membranes as the result of physiological rubbing between the tissues during pupillary movement. After entering the current of the aqueous humor, tiny clusters of the basement membrane make their way to the angle and accumulate in the trabecular meshwork, leading to mechanical congestion of aqueous outflow, ocular hypertension, and ultimately glaucoma.

Pigment dispersion syndrome has an autosomal dominant inheritance, and the presence of a concave iris contour is thought to be the underlying mechanism of pigment shedding. The posterior bowing of the mid-peripheral iris—called reverse pupillary block—results in mechanical rubbing between the posterior iris surface and anterior lens zonules, causing the release of melanin pigment granules into the anterior chamber from disrupted iris pigment epithelial cells. As with basement membrane material in PXF syndrome, these pigment particles can clog the trabecular meshwork, leading to increased intraocular pressure and ultimately glaucoma.

See INSIDE for:

Heads Up: Lesser-known Glaucoma Risk Factors and Therapeutic Targets

by Robert Ritch, MD
Peripheral transillumination defects, which are distinct from the peripapillary iris transillumination defects seen in PXF syndrome.

THE DIAGNOSTIC WORKUP

Although PXF and pigment dispersion syndrome have a distinctive constellation of clinical features, an accurate diagnosis requires that clinicians be mindful of both conditions and vigorously look for the signs of each. When I see a patient who comes in for evaluation of glaucoma, I always ask myself these questions: Is there a Krukenberg spindle? Is there an abnormal pupillary border or, on the dilated exam, an abnormal appearance of the anterior capsule? What does the gonioscopy look like? How much pigment is there?

For patients who show signs of either PXF or pigment dispersion syn-

### 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, affecting a significant and growing portion of the US population. Much remains to be understood about the pathophysiology of glaucoma, but high intraocular pressure (IOP) has been identified as a key risk factor for progression. Medical and surgical therapies for the disease are primarily directed at reducing IOP. Recent years have seen significant innovation in the treatment of glaucoma, including gentler and more effective topical drugs, less invasive surgical techniques, and new molecules and mechanisms of action. As pharmaceutical and surgical treatments for glaucoma rapidly advance—and as research continues to provide insights about the disease's neurologic underpinnings—comprehensive ophthalmologists are challenged to remain up-to-date.

To give their glaucoma patients the full benefit of treatment advances, clinicians require clear, actionable insights from subspecialists and researchers. Topics in Glaucoma will present current research in the context of comprehensive care, providing non-specialists with clearly presented, evidence-based clinical judgments from experts in the field.

### REFERENCES


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**FACULTY AND DISCLOSURE STATEMENTS**

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drome on the slit lamp exam, a complete diagnostic workup for glaucoma is warranted: full history including family history, measurement of IOP, gonioscopy, dilated fundus examination, visual field testing, and measurement of the retinal nerve fiber layer and ganglion cell layer via optical coherence tomography (OCT). If the tests are normal, patients should be followed as glaucoma suspects every 6 months to 1 year.

In some cases, the development of glaucoma can precede the classic slit lamp findings, especially with PXF syndrome. Patients who show no signs of PXF at the initial diagnosis of glaucoma may be found to develop features that are characteristic of the condition while being followed for glaucoma.

TREATMENT CHALLENGES

Not all patients with PXF or pigment dispersion syndrome will develop glaucoma, but predicting which patients will go on to develop optic nerve damage and loss of visual function is nearly impossible. PXF glaucoma tends to increase with age; in eyes with PXF syndrome, the probability of converting to glaucoma is about 30 to 40% in 10 years.8 For patients with pigment dispersion syndrome, the conversion rate is 10% at 5 years and 15% at 15 years according to one community-based study of 113 patients; young, myopic men have the highest risk of conversion.8

Because patients with PXF or pigment dispersion syndrome are at risk of developing glaucoma, I usually initiate therapy earlier in comparison to patients with suspected primary open-angle glaucoma, especially in cases where there are already suggestive signs of glaucomatous damage, such as suspicious optic disc cupping, characteristic visual field changes, or a slightly thinner retinal nerve fiber layer.

Compared with primary open-angle glaucoma, PXF glaucoma and, to a certain extent, pigmentary glaucoma can be more difficult to control with medical therapy. PXF glaucoma is often bilateral yet clinically asymmetric; topical IOP-lowering medications may be effective initially but have a high failure rate. For pigmentary glaucoma, patients are generally younger at the age of onset. They are more difficult to manage in that they require longer-term treatment to prevent glaucomatous damage. The average age of diagnosis is over 60 years for primary open-angle glaucoma and between 20 and 40 years for pigmentary glaucoma. That gives at least 20 additional years during which the typical patient with pigmentary glaucoma needs to manage the disease.

Since medical management often falls short, patients with PXF or pigmentary glaucoma are more likely to undergo laser or surgical therapy. They respond better to argon or selective laser trabeculoplasty than patients with primary open-angle glaucoma, probably because of greater absorption of laser energy by the pigmented trabecular meshwork. On the other hand, the power of the laser must be kept low in order to avoid IOP spikes.

When medical and laser treatments prove inadequate, surgical intervention—typically trabeculectomy—becomes necessary and is usually effective in controlling IOP. Patients with PXF glaucoma, however, may present a greater risk for surgical complications. Because PXF syndrome is associated with iris vascular leak and an impaired blood-aqueous barrier, these patients are more prone to postoperative inflammation and pressure spikes.10 In cataract surgery, in particular, PXF syndrome poses a unique challenge. In addition to an increased postoperative inflammatory response, the risk of having intraoperative complications is heightened because of weak zonules, poor pupil dilation, and compromised aqueous outflow.

NEWER OPTIONS

One controversial procedure in laser treatment of pigmentary glaucoma is laser peripheral iridotomy (LPI). It is speculated that LPI may be particularly beneficial in eyes with pigmentary glaucoma because, theoretically, creating a small hole in the peripheral iris can equalize the pressure between the anterior and posterior chamber, relieve reverse pupillary block, and flatten the iris. However, large prospective studies are scarce, and there is no convincing evidence to either support or disprove the procedure as an effective treatment.

One retrospective study conducted by members of the American Glaucoma Society suggests that iridotomy has no significant effect on the course of pigmentary glaucoma,11 whereas a randomized controlled study showed that LPI decreased the risk of developing glaucoma in 10 years in high-risk patients.12 Most recently, a Cochrane systematic review concludes that there is insufficient high quality evidence to establish the long-term effects of LPI.13

Apart from the traditional surgical
approaches such as trabeculectomy, surgical options for glaucoma patients now include a newer group of angle-based procedures including ab-interno trabeculectomy and trabecular micro-bypass stent implantation. Seeking to recover the natural trabecular outflow pathway, these minimally invasive procedures have the potential to work even better than in primary open-angle glaucoma. Thus far, no studies have looked at the comparative effectiveness of angle-based procedures in different types of glaucoma.

Most of the current IOP-lowering agents work by increasing uveoscleral outflow (prostaglandins) or decreasing aqueous production (beta blockers, alpha agonists, carbonic anhydrase inhibitors). Several new classes of glaucoma medications with novel mechanisms of action targeting the trabecular meshwork are anticipated to become available in the near future. While having more therapeutic options is always a good thing, whether or not these new agents will enhance our ability to medically manage PXF and pigmented glaucomas remain to be seen.

Kimberly V. Miller, MD, is director of the glaucoma service and clinical assistant professor of surgery (ophthalmology) at Warren Alpert Medical School of Brown University in Providence, RI. She states that in the past 12 months, she has not had a financial relationship with any commercial organization that produces, markets, resells, or distributes healthcare goods or services consumed by or used on patients relevant to this manuscript. Medical writer Ying Guo, MBBS, assisted in the preparation of this manuscript.

REFERENCES
1. Lindberg JG: Kliniska undersökningar over depigmenteringen av pupillarranden och genomlysbarheten av iris vid fall av aldersstarr samt i normala ogon hos gamla personer. [Clinical studies of depigmentation of the pupillary margin and transillumination of the iris in cases of senile cataract and also in normal eyes in the aged] [Ph.D. Thesis]. 1917, Helsinki, Finland: Helsinki University.
Heads Up: Lesser-known Glaucoma Risk Factors and Therapeutic Targets

Robert Ritch, MD

Intraocular pressure (IOP) is a dynamic variable influenced by multiple factors, including posture, blood pressure, sleep disorders, exercise, intracranial pressure, and others. It is important that eye care providers identify factors and disorders that might contribute to raised IOP and/or glaucoma progression and make appropriate recommendations and referrals.

Glucoma and intraocular pressure (IOP) are so intimately connected—in pathophysiology, diagnosis, and treatment—that the two can easily merge into a single entity in our minds.

Of course, they are not: glaucoma is a degenerative disease of the optic nerve, and IOP is one important contributing factor in glaucoma. Many individuals with elevated IOP never develop glaucoma; conversely, a significant number of glaucoma patients have normal IOP, a condition known as normal-tension glaucoma (NTG). Furthermore, we know that reducing IOP slows but may not halt glaucoma progression. That glaucoma remains a leading cause of blindness worldwide is a testament to the fact that our current understanding of this devastating disease is far from complete.

Clinicians hoping to deliver optimal care to glaucoma patients would do well to consider IOP in the context of the broader physiologic picture, taking into account factors that may be contributing to IOP elevation or acting on the optic nerve by mechanisms not reflected in IOP. This article covers several such factors (eg, vascular, sleep-related, even exercise-related) that are not commonly discussed but that may be relevant to clinical practice.

SLEEP APNEA

OSAS is characterized by repeated episodes of partial or complete upper airway obstruction lasting 10 seconds or longer during sleep. It affects 9% of women and 24% of men between ages 30 and 60 years.

Several studies have shown that patients with primary open-angle glaucoma (especially normal-tension glaucoma) have been found to have higher-than-expected rates of OSAS or sleep-disordered breathing. Conversely, patients with OSAS have been shown to have significantly higher rates of hypertensive and normotensive glaucoma than the general population. In addition, OSAS has been associated with floppy eyelid syndrome, non-arteritic anterior ischemic optic neuropathy (NAION), and papilledema.

Studies have shown correlation between severity of OSAS and severity of glaucoma. For example, Lin and colleagues showed that patients with OSAS had increased risk for normotensive glaucoma and that nighttime oxygen saturation positively correlated with retinal nerve fiber layer thickness, suggesting that impaired oxygen delivery to the retinal nerve contributed to its degeneration.

In OSAS, a disturbance of the normal autoregulatory mechanisms that occur during sleep leads to hypoxia and sympathetic activation, which may lead to hypoxia-induced blood vessel damage and altered blood flow to the optic nerve head. Eventually, this cascade, alone or in combination with other predisposing factors, can reduce ocular perfusion pressure and either directly damage the optic nerve or increase its susceptibility to injury from other insults.

Fortunately, OSAS is treatable with nasal or oral continuous positive airway pressure (CPAP); but it remains marked-

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TOPICS IN GLAUCOMA 5
ly underdiagnosed (Figure 1).\textsuperscript{1} Suspect OSAS in patients with obesity, increased neck girth, upper airway abnormalities, snoring, daytime sleepiness, or insomnia, although it may also be present without these findings.\textsuperscript{11}

**FLAMMER SYNDROME**

Flammer syndrome is a cluster of findings in otherwise healthy individuals that is associated with vascular dysregulation including an increased incidence of normal-tension glaucoma.\textsuperscript{3} Patients are generally thin, myopic females with cold hands (Raynaud’s phenomenon) and low blood pressure.

Flammer syndrome should be on the radar of eye care providers who treat NTG. Proposed vascular stabilizing treatments include magnesium supplementation, Gingko biloba, and very low dose calcium channel blockers, as Dr. Flammer feels that 3 mg nifedipine dissolved in a liter of water and sipped throughout the day improves ocular blood flow without lowering blood pressure. Other measures include lifestyle practices such as yoga (but not with inverted posture positions, see below), stress avoidance (which needs to be proven in controlled studies), supplemental omega-3-fatty acids (the triglyceride form, as the acid and alcohol forms are largely ineffective), and sufficient caloric intake to maintain adequate weight, as the development of glaucoma has been suggested to correlate inversely with body mass index (BMI).

Twenty-four-hour blood pressure monitoring reveals that most healthy people experience a minor dip in blood pressure (about 10% below mean arterial pressure) at night. Those who do not dip are prone to developing strokes. However, overdipping (eg, a 20% to 30% reduction) is a risk factor for progression of glaucoma, particularly NTG, and this can lead to decreased ocular perfusion pressure (OPP), which can contribute to ocular ischemia. A recent major prospective study showed that the magnitude and depth of nocturnal blood pressure dip predicted progression of visual field loss among patients with glaucoma.\textsuperscript{4}

**SLEEP POSITION**

We spend up to one-third of our lives asleep; and supine IOP increases may play a role in progression of glaucoma.\textsuperscript{12} In healthy subjects, being recumbent increases IOP compared with sitting or standing; and sleeping in the lateral decubitus position (side sleeping) or prone (face-down) with head turned has been shown to increase IOP in the lower eye.\textsuperscript{13}

Usually the increase in IOP due to recumbency is minor, such as 1 mmHg to 2 mmHg, but some patients may experience increases of as high as 10 mmHg. Patients with vascular dysregulation or elevated IOP may experience even sharper IOP increases in prone and lateral decubitus postures. One study showed that a significant majority of patients with bilateral NTG or high-tension glaucoma and asymmetric visual field loss reported sleeping on the more affected side.\textsuperscript{14} Ocular compression from side sleeping with the lower eye pressed against a pillow or one’s hand can increase IOP markedly.

In a prospective study by Buys and colleagues, glaucoma patients with well-controlled IOP and a new disc hemorrhage (N = 17) underwent IOP and blood-pressure monitoring every two hours while sleeping in one of two positions: flat supine position (night one) or on a sloping wedge pillow that raised the upper torso and head 20° to 30° (night two). Sleeping with the head elevated on the wedge pillow was associated with mean 3.18 mmHg lower IOP (without affecting OPP) compared with sleeping flat.\textsuperscript{12} More than one third of patients in the study experienced at least 20% IOP reduction in the head-up position.

Glaucoma patients should be encouraged to sleep on their back (presuming they do not have OSAS) or the side of the less affected eye. Specially designed belts that discourage rolling or eye shields that protect against physical pressure on the eye during sleep may be advised.

**YOGA**

Yoga, which has become enormously popular in the West in recent decades, is a mind-body-breath integration practice
that involves assuming and sometimes holding a wide range of postures known as asanas. Some asanas involve lowering the head below the level of the waist (eg, downward-facing dog, forward bend) and/or feet (eg, plow pose, legs up the wall), inducing a transient elevation in IOP. Headstand (“sirsasana”) represents the most extreme inversion; IOP has been shown to at least double in healthy patients and those with glaucoma while in the pose (Figure 2).15

I once encountered a yoga practitioner who was nearly blind from glaucoma, diagnosed as NTG, with IOPs of 15 mmHg, who stood on her head for 20 minutes a day for 20 years. When we tested her in this position, her IOP rose to 60 mmHg. And there are other reports of glaucoma progression related to sustained sirsasana practice, at least one of whom experienced full recovery of visual field deficit within several months of stopping.16,17

Several colleagues and I recently conducted a study looking at IOP changes induced by four common head-down yoga postures among patients with and without primary open-angle glaucoma. Each pose was held for 2 minutes; IOP was measured before, during and after each pose using a Reichert Model 30 pneumotonometer. We found that all participants experienced an IOP increase during each of the poses (generally between 6 mmHg and 11 mmHg rise; average maximal difference of up to 12.6 mmHg or 79% over baseline), with IOP returning to baseline within two minutes of ending the pose.18 Yoga-induced IOP change was not significantly different between the normal and glaucoma groups.

Proposed mechanisms to explain posture-induced IOP increase include hydrostatic increase in orbital and episcleral vein pressure (causing back pressure against aqueous drainage) and possibly changes in choroidal thickness.18

But there is currently no evidence that brief inversions of a minute or two increase risk for glaucoma progression; it may be that transient IOP spikes are harmless, either because they are short-lived or because a concomitant rise in cerebrospinal fluid pressure might provide an effective counter-pressure against IOP across the lamina cribrosa during brief inversions.18 And theoretical risk must be considered in the context of manifold long-term health benefits associated with carefully conducted yoga practice, including reduced stress, improved parasympathetic tone, and neurohormonal activity.19

CONCLUSION
Vascular disorders that might affect the eye and sleep-related body position merit consideration for all patients in glaucoma care, particularly when glaucoma progression is unexplained.

REFERENCES

Robert Ritch, MD, holds the Shelley and Steven Einhorn Distinguished Chair in Ophthalmology and is Surgeon Director Emeritus and chief of glaucoma services at the New York Eye and Ear Infirmary of Mount Sinai, New York City. He states that in the past 12 months, he has not had a financial relationship with any commercial organization that produces, markets, resells, or distributes healthcare goods or services consumed by or used on patients relevant to this manuscript. Medical writer, Noelle Lake, MD, assisted in the preparation of this manuscript.
1. Which of the following is NOT a typical presentation of PXF syndrome or PXF glaucoma?
   A. Young patients in their 20s
   B. Whitish deposits on the anterior lens capsule
   C. Patchy pigment deposition in the trabecular meshwork
   D. Peripapillary iris transillumination defects

2. Theories regarding IOP rise in inverted positions include:
   A. Increased choroidal thickness
   B. Ocular venous congestion
   C. Increased CSF pressure
   D. A and B

3. The probability of converting from PXF syndrome to PXF glaucoma in 10 years is about:
   A. Less than 5%
   B. 10 to 20%
   C. 30 to 40%
   D. 60% to 70%

4. Flammer syndrome is characterized by which of the following?
   A. Thin build, female sex, cold hands
   B. Obesity, male sex, sleep apnea
   C. Hypertension, insomnia, heightened pain sensitivity
   D. None of the above

5. Which of the following features is associated with PXF syndrome?
   A. Poor pupillary dilation
   B. Weak lens zonular attachments
   C. Compromised blood-aqueous barrier
   D. All of the above

6. Compared with patients with primary open-angle glaucoma, patients with PXF or pigmentary glaucoma respond better to which of the following therapies?
   A. Topical IOP-lowering medications
   B. Laser trabeculoplasty
   C. Laser iridotomy
   D. Trabeculectomy

7. Patients with unilateral visual field loss related to glaucoma should be advised to sleep:
   A. With the affected side lower than the unaffected side
   B. In prone, head-turned position
   C. In a supine, slightly elevated position
   D. During the day

8. OSAS affects an estimated:
   A. 10% of men and women over 50
   B. 24% of women age 30 to 60
   C. 24% of men age 30 to 60
   D. 45% of men over age 50

9. Which of the following is least likely to induce transient rise in IOP?
   A. Downward facing dog pose
   B. Sirsasana (headstand)
   C. Lying supine with feet up 90 degrees (feet against the wall pose)
   D. Sitting meditation

10. Which of the following is LEAST likely to induce transient rise in IOP?
    A. 10% of men and women over 50
    B. 24% of women age 30 to 60
    C. 24% of men age 30 to 60
    D. 45% of men over age 50

ANSWERS:
1. A
2. B
3. C
4. A
5. A
6. B
7. C
8. B
9. C
10. D

EXAMINATION ANSWER SHEET  TOPICS IN GLAUCOMA — ISSUE 10

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