Abstract: Glaucoma suspect is a diagnosis reserved for individuals who do not definitively have glaucoma at the present time but have characteristics suggesting that they are at high risk of developing the disease in the future based on a variety of factors. This review provides a practical approach to individuals classified as glaucoma suspects caused by one or more of the following risk factors or indicators of disease: ocular hypertension, optic nerve features suggestive of glaucoma, visual field abnormalities, and other characteristics placing them at greater risk than the average population. In addition to diagnostic considerations, this overview provides information on therapeutic approaches to the glaucoma suspect.

Key Words: glaucoma suspect, diagnosis, therapy, review

Glaucoma Suspect: Diagnosis and Management

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Glaucoma is a slowly progressive disease with characteristic optic nerve abnormalities caused by accelerated retinal ganglion cell (RGC) loss and concomitant retinal nerve fiber layer (RNFL) thinning. Normally, individuals have enough ganglion cells to preserve vision for a lifetime, but in those with glaucoma, the rate of RGC death may be too fast to allow visual function to remain normal. The result is progressive peripheral and/or central visual defects, which can, in some individuals, lead to complete vision loss. Modern treatment slows the rate of vision loss, but even treated individuals may still go blind depending on when the disease is detected. Of note, the exact structure-function relationship for any given stage of glaucoma in a particular patient is still not predictable. Eventually, in vivo imaging will aid in providing noninvasive neural cell counts, at which time a link between cellular pathophysiology and clinical manifestations will be better characterized.1

Currently, glaucoma is the leading cause of irreversible blindness worldwide. In 2013, an extensive meta-analysis of 50 population-based studies from around the world estimated the glaucoma prevalence estimate for 2010 projected by Quigley (2016;5: 32–37) or about 64.3 million afflicted.2 This was similar to the glaucoma prevalence estimate for 2010 projected by Quigley and Broman3 in 2006. Therefore, although routine glaucoma screening may be controversial from the perspective of the primary care provider, certainly identifying glaucoma is an important task among vision care specialists. Glaucoma suspects are the key group to identify and, in some cases, treat with intraocular pressure (IOP)–lowering therapy in an effort to prevent blindness. It is more compelling to screen high-risk populations once every 12 months, including diabetics, individuals with a family history of glaucoma, African Americans older than age 50, and Hispanic Americans aged 65 years and older.2

RISK FACTORS

The term “glaucoma” encompasses many diseases, some of which can be secondary to a variety of causes resulting in subcategories such as neovascular, pseudoxfoliative, pigmentary, uveitic, steroid induced, traumatic angle recession, post-surgical, and so on. Other categories include congenital, primary open-angle (POAG), and primary angle-closure (PACG), all of which are generally associated with higher than average IOP as the primary risk factor. Thus, the term “glaucoma suspects” may be any of these types (predisesease), but generally, individuals are labeled “glaucoma suspects” when they have at least 1 clinical feature of the disease, including an elevated IOP, a suspicious optic nerve appearance commonly referred to as “cupping,” a repeatable visual field abnormality consistent with optic nerve damage, or a strong family history of glaucomatous disease. From the Baltimore Eye Survey, age-adjusted associations of POAG with a family history of glaucoma were 3.69 times higher in siblings and 2.17 times higher in parents relative to those with no family history of disease.5 However, the genetic inheritance of glaucoma has not been simple to elucidate and is multifactorial. It has been difficult to find a single biomarker for glaucoma. Other risk factors derived from large prospective cohort studies include increased age, ethnicity, selected medication, myopia, and a thin cornea.6 Finally, some specific risk factors may be associated with normal- or low-tension glaucoma, such as migraine headaches, disc hemorrhages, vascular dysregulation, and sleep apnea.7,8 But the hallmark sign separating true pathology from a glaucoma suspect, regardless of IOP, is progressive glaucomatous visual field loss distinct from neurologic field defects or nonprogressive defects that can mimic glaucoma.

Elevated IOP is a well-known strong risk factor for glaucoma, yet not all individuals with high pressure develop glaucoma. The Barabados Eye Studies revealed in more than 9 years of follow-up that, although persons of African origin with a baseline IOP more than 25 mm Hg had a 13-fold relative risk of developing POAG, most cases arose with a lower baseline IOP.5 Because many individuals with glaucoma may not always have a high IOP and the majority with a higher-than-average IOP do not develop glaucoma, relying on IOP to diagnose glaucoma is not advisable. Two landmark 5-year clinical trials were undertaken to determine the benefit of treating ocular hypertensive glaucoma suspects: the Ocular Hypertension Treatment Study (OHTS)10 completed in 2002 and the European Glaucoma Prevention Study (EGPS).11 The former study included 1636 participants with IOP between 24 and 32 mm Hg, with 9.5% of untreated subjects converting to POAG across 5 years versus only 4.4% of treated subjects. Its other breakthrough finding was that a thinner central corneal thickness (CCT) was an independent risk factor for developing glaucoma. The same study also found that a thicker CCT was protective, but there is no validated linear correlation correction formula for IOP and CCT, as corneal biomechanics can also affect IOP readings gathered indirectly through the cornea. By combining the longitudinal data from the OHTS and EGPS, a validated glaucoma progression risk calculator was created using the inputs of age, IOP, CCT, vertical cup-to-disc (C/D) ratio, and pattern standard deviation of visual field test (http://ohts.wustl.edu/risk/calculator.html).12 Unfortunately, the risk factors in the calculator are essentially not modifiable except IOP, the vertical C/D ratio is very subjective and, along with
the pattern standard deviation, is used to define the disease—all points that make the calculator a bit less useful. Nonetheless, when the point system estimates greater than 15% chance of conversion to glaucoma in the next 5 years, the prediction model provides evidence of when to initiate therapy selectively instead of treating every case of high IOP automatically.

Besides increased IOP, the other most common reason for suspected glaucoma is “cupping” or an enlarged C/D ratio visualized on routine funduscopic examination. The distinction between physiologic cupping and pathologic cupping is not always easy, and accurate classification can be quite subjective even among experts. Pathological optic disc cupping is most often caused by glaucoma but may also be seen with several less common neuro-ophthalmic conditions. Typically, glaucomatous cupping may involve disc excavation with neuroretinal rim loss, focal notching, bayoneting or nasalization of blood vessels as the rim thins, baring of the lamina cribrosa (laminar dots), beta zone peripapillary atrophy, splinter hemorrhages, and enlarging RNFL defects. Traditional teaching states that a healthy neuroretinal rim tends to have the thickest nerve fiber layer inferiorly, then superiorly, then nasally, and then temporally (ISNT rule). However, disc excavation has also been described in those with optic nerve hypoplasia, optic nerve head drusen, morning glory syndrome, septo-optic dysplasia, optic nerve compression, traumatic optic neuropathy, anterior ischemic optic neuropathy or systemic shock (low perfusion), radiation optic neuropathy, Leber hereditary optic neuropathy, and dominant optic atrophy.

Thus, these conditions should be ruled out in cases with a large C/D ratio.

High myopia has long been known to be associated with glaucoma. The Blue Mountains Eye Study found a 2- to 3-fold increase in glaucoma prevalence related to myopia, and this was confirmed by multiple subsequent studies even after incorporating axial length and adjusting for cataract. The challenge is that anomalous optic nerves with RNFL bundle defects may be confused with the appearance of a glucosomatous optic nerve, particularly in cases with high myopia, nerve tilt or oblique insertion, staphyloma, coloboma, optic pit, and large peripapillary atrophy. Many of these diagnostic dilemmas may or may not be seen concomitantly with high IOP, and oftentimes assessing carefully for glaucomatous visual field progression is the primary method of teasing out a glaucoma diagnosis in the setting of an anomalous-appearing uninterpretable optic nerve.

Physiologic cupping is typically characterized by bilaterally round, symmetrically enlarged optic cups without focal RNFL thinning. The disc area may or may not be larger than average, but clinicians are more suspicious of glaucoma with large optic disc areas, particularly when greater than 3.0 mm. However, a large intact cup within a large disc often represents physiologic cupping, as the same normal amount of annular neuroretinal rim is spread over a larger area. Also, superotemporal and inferotemporal RNFL bundles tend to vary in location and converge more temporally with increasing myopia, which, although appearing abnormal, may be normal for a given individual. Individuals of African descent have a higher prevalence of open-angle glaucoma relative to whites but they also may have large disc areas, so it is important to measure the disc size in all glaucoma suspects to aid in distinguishing physiologic cupping.

Lastly, 2 other strong risk factors frequently mentioned in association with glaucoma in individuals with average IOPs are reduced ocular perfusion pressure and disc hemorrhages (originally Drance hemorrhages). But these typical flame-shaped hemorrhages can also occur near the disc edge in association with other conditions such as posterior vitreous detachment, optic disc drusen, vascular occlusive retinal diseases, nonglaucomatous optic neuropathy, and systemic conditions such as diabetes, hypertension, leukemia, and lupus.

### Diagnosis

Among all glaucoma types, POAG is classified as a neurodegenerative disease, and POAG suspects display a constellation of risk factors or clinical findings that indicate a pretest probability of disease. Primary open-angle glaucoma suspects have open angles and subjective structural optic disc or nerve fiber layer changes. Primary angle-closure glaucoma subjects have variable amounts of closed angle, and the current nomenclature divides the closed-angle group into primary angle-closure suspect (PACS), primary angle closure (PAC), and PACG. The PAC designation refers to closed angle with peripheral anterior synchiae and/or elevated IOP but no structural nerve damage, whereas PACG has definite nerve damage. In PACS, then, a shallow peripheral angle with iridotrabecular contact in at least 3 quadrants is seen without peripheral anterior synchiae but with otherwise normal IOP and optic nerve. Glaucoma can be divided into preperimetric and perimetric disease based on the absence or presence of reproducible glaucomatous visual field defects. Generally, glaucoma suspects have converted to true glaucoma once classic field defects, including arcuate bundle defect, nasal step, paracentral scotoma, and altitudinal defect, appear and are not attributable to any other optic neuropathies. An initial glaucoma evaluation includes a history and physical examination and a comprehensive adult medical eye evaluation, including IOP measurement, CCT, gonioscopy, anterior and posterior segment evaluation with optic nerve head imaging, and perimetry testing. Oftentimes, a diagnosis of an open- or closed-angle glaucoma suspect is made on a single visit, but serial follow-up testing moves the suspicion level either higher or lower.

### Intraocular Pressure

Intraocular pressure is an important part of every glaucoma suspect evaluation because there is an exponential increase in glaucoma risk with a progressively higher IOP. Whereas the normal pressure range for a white population is 15 to 16 mm Hg with a standard deviation of 3 mm Hg, up to 61% of patients with POAG have a single screening IOP less than 21 mm Hg, making abnormal IOP as a diagnostic criterion less than optimal. Further, it can be difficult to obtain accurate noninvasive readings, as isolated office visit measurements do not reflect the full range of IOP changes during a 24-hour period. To complicate matters further, the long-standing gold standard method for checking IOP is the Goldmann applanation tonometer (GAT), which has known flaws in terms of estimating the true manometric pressure of the eye because GAT is an indirect measurement through the cornea. For example, a history of refractive surgery can alter corneal biomechanics and corneal thickness, resulting in falsely low IOP readings. Also, the GAT reference standard assumes a specific CCT, which varies widely in all populations. At present, there is no proven formula to adjust IOP for CCT. However, most of the evidence-based clinical trials have used GAT IOP as an outcome measure, and thus it is difficult to interpret the relevance of newer methods such as noncontact tonometry, dynamic contour tonometry, rebound tonometry, or ocular response analyzer. All noncontact tonometers have their own limitations, especially if used to obtain a single isolated value of a physiologic parameter that is quite variable with every heartbeat and eye movement. External and internal 24-hour IOP-monitoring devices are under study to record peak IOP as well as short- and long-term IOP fluctuation, and it remains unclear whether IOP fluctuation also plays an independent role in glaucoma progression. Knowing how IOP behaves in low-pressure glaucoma and while on various glaucoma therapies would be interesting as well. The most exciting potential of 24-hour IOP devices is the possibility that circadian IOP patterns may predict future
glaucoma progression, and such devices may be able to help obtain such clinically useful information. There is 1 specific situation when IOP is used to make the diagnosis of glaucoma, and that is when it is not possible to visualize the optic nerve and field testing is impossible. Then, if the visual acuity is diminished and the IOP is elevated, a diagnosis may be assumed.

**FUNDUS EXAMINATION**

A stereoscopic view of the optic nerve through a dilated pupil when feasible is the preferred examination technique. There is evidence that glaucomatous changes can be detected at the level of the optic nerve head and RNFL before any findings on tests such as standard automated perimeter (SAP) in regions of the world where perimeters, fundus cameras, and optical coherence tomography (OCT) machines are easily accessible, baseline testing is performed to support or refute a glaucoma diagnosis and to document findings for future comparison. This is critical to glaucoma suspect monitoring and follow-up, as glaucoma progression can be slowed but is currently not curable or reversible, and thus it is ideal to identify rapid progressors early. Color stereophotography has long been the accepted method for documenting optic nerve head appearance, especially as the technology required to perform such tests is unlikely to be available over time and sometimes glaucoma takes years to develop. Precision medicine emphasizes customized therapy to each individual’s rate of disease development, and serial high-resolution OCT is a rapidly emerging area for individualized early detection of change. Repeat automated structural testing at a very high reproducibility allows each individual to have his or her own “normative” data or change from baseline rather than relying on population norms.

**VISUAL FIELDS**

Perimetry has long been the mainstay test for managing glaucoma suspects and detecting glaucoma deterioration because it is the main visual outcome measure. The preferred technique is automated static threshold perimeter, commonly with the central 24- or 30-degree program. Both trend and event analyses are used for establishing change within a series of visual field tests. Because fields can be highly subjective based on patient cooperation, repeat confirmatory visual field examinations are strongly recommended. On average, 3 fields are needed in 1 year to detect an overall change in mean deviation of 4 dB over 2 years in a patient with average visual field variability. One way to think about fields in glaucoma suspects is to realize that they can help differentiate between rapid and slow progressors, which can affect the choice of therapeutic intervention. With FORUM image management software (Carl Zeiss Meditec, Dublin, Calif), clinicians can easily see the Visual Field Index plotted over time and look at the Glaucoma Progression Analysis for localized event changes simultaneously with Visual Field Index trend analysis. In the OHTS trial, when the 2 study end points of visual field progression or optic disc change were compared, both were found to be important, as either could be the first to demonstrate the initial evidence of glaucomatous damage.

If identifying preperimetric glaucoma using progression on OCT accurately predicts perimetric vision loss, then it makes sense to initiate or increase therapy sooner instead of waiting for confirmatory visual field changes. If a clinician only has access to 1 abnormal field, however, then repeat examinations are needed, as the question is whether the field is stable or still worsening. Some glaucoma suspects may have normal fields and abnormal OCTs, and still others may have normal fields and normal OCTs but elevated IOPs. Given these different glaucoma suspect situations, clinicians need to assimilate multiple inputs of age, vision, IOP, nerve status, and visual field reserve to slow further vision loss until restorative therapies become available in the future.

**OPTICAL COHERENCE TOMOGRAPHY**

Optical coherence tomography, currently spectral-domain OCT (SD-OCT), is a powerful, rapidly evolving, computer-based imaging tool that complements fundus photography. Using good-quality images without artifacts, OCT provides quantitative information about the RNFL, optic nerve head, and ganglion cell layer with high reproducibility. Numerous studies have demonstrated the sensitivity and specificity of OCT to distinguish between normal and glaucomatous eyes with an area under the receiver operating characteristic curve value of approximately 0.9 (best with more advanced glaucoma). The RNFL thickness is the primary parameter of interest, although the ganglion cell layer with inner plexiform layer and the ganglion cell complex (ganglion cell complex = ganglion cell layer with inner plexiform layer + RNFL) offers additional information. One reason to obtain both macular and optic nerve cube scans is that macular thickness maps can be especially useful in high myopes when the RNFL appears abnormal because of tilted discs. Event and trend analyses are applied to OCT parameters just as they are for visual fields; however, some unresolved issues are whether all eyes have a linear rate of structural loss, if age-related loss varies as a function of baseline RNFL, and how to accommodate old data when there are so many different machines with nonshareable data. With Cirrus SD-OCT (Carl Zeiss Meditec), clinicians have the RNFL thickness and deviation maps to view localized event changes combined with the Guided Progression Analysis trend analysis tool. As SD-OCT is highly reproducible with a resolution of approximately 5 μm, the trend analysis is a powerful way to align each image using blood vessel registration and compare the same spot every time to detect subtle changes in early disease when all the scans are of good quality and similar signal strength. One downside of OCT is that glaucoma tends to progress slowly through the years (and sometimes, the technology changes faster than the disease).

A recent longitudinal study estimating the diagnostic accuracy and lead time of OCT detection of glaucoma before the development of field defects found that the receiver operating characteristic curve areas were 0.87, 0.77, and 0.65 at 0, 4, and 8 years before visual field loss, respectively. At 95% specificity, up to 35% of eyes had abnormal average RNFL thickness 4 years before the development of visual field loss, demonstrating the powerful use of OCT in glaucoma suspects to detect glaucoma before fields. In advanced glaucoma, checking OCT is less useful because there is no RNFL left to measure, only residual glial and blood vessels. In those cases, serial visual field testing is more useful. In an important study to determine the RNFL thickness at which visual field damage becomes detectable, Wollstein et al found that the mean SD-OCT RNFL thickness threshold for visual field loss was 75.3 μm (95% confidence interval: 68.8–81.8) in their study population of 74 normal and 40 glaucoma subjects. Thus, one of the main strengths of OCT in glaucoma suspects is that a normal RNFL scan reveals with a high likelihood that glaucoma is not present. Serial OCT RNFL typically detects conversion to glaucoma in one of 3 ways: 1) expansion of RNFL defect, 2) development of new defect, or 3) deepening of existing defect. The defects are most commonly located at the inferotemporal meridian and should be studied carefully on the RNFL thickness maps. Any suspected new change should be assessed in terms of consistency with other clinical features.
PROMISING GLAUCOMA TESTING MODALITIES

Because glaucoma pathogenesis begins at the ganglion cell level, an ideal imaging technique to diagnose and monitor glaucoma would quantify RGCs noninvasively with high specificity and sensitivity in vivo. Such a test would optimally label the unhealthy cells before inevitable apoptosis occurs, opening the doorway to neuroprotection or other regenerative therapies. Current cellular markers under study in vivo are fluorescent labels, including CapQ and Annexin-A5. However, these probes are used mostly in animal models and are just starting to be used in phase I trials. The difference between the proportion of apoptotic cells in healthy eyes compared with glaucomatous eyes is not yet clear. As glaucoma is not limited to the loss of RGCs but also extends to the posterior visual pathway, there is a potential to use advanced morphological and functional magnetic resonance techniques to detect glaucomatous optic neuropathy. Whereas most of the techniques (morphometry, diffusion tensor imaging, arterial spin labeling, and functional connectivity) can only detect qualitative changes in advanced glaucoma, diffusion tensor imaging has demonstrated some promise in identifying early glaucoma. In patients with glaucoma, diffusion tensor imaging identified an increase in mean diffusivity and a decrease in fractional anisotropy more evident at the proximal site of the optic nerve. This suggests that studying the entire optic pathway in glaucoma with magnetic resonance may yield a new imaging biomarker.

Perimetry has used different stimulus size, color (blue on yellow) short wavelength automated perimetry, and contrast gratings (achromatic vertical sine wave grating of low spatial frequency at a high temporal frequency = frequency doubling technology) all in an attempt to identify earlier glaucoma damage. An emerging approach to neuroprotection or other regenerative therapies. Current approaches on microperimetry in glaucoma have focused on evaluating the techniques (morphometry, diffusion tensor imaging, arterial spin labeling, and functional connectivity) can only detect qualitative changes in advanced glaucoma, diffusion tensor imaging has demonstrated some promise in identifying early glaucoma. In patients with glaucoma, diffusion tensor imaging identified an increase in mean diffusivity and a decrease in fractional anisotropy more evident at the proximal site of the optic nerve. This suggests that studying the entire optic pathway in glaucoma with magnetic resonance may yield a new imaging biomarker.

For early detection of glaucoma, an adjunctive therapy.47 Selective laser trabeculoplasty (SLT) is considered, with selective laser trabeculoplasty (SLT) sometimes being a first-line therapy.49 Alternatively, other topical glaucoma medication classes, including beta-blockers, carbonic anhydrase inhibitors, or fixed-combination ocular hypotensive agents, may be considered as adjuncts to prostaglandins. Most of the time, with each additional medicine given, the IOP-lowering effect is diminished compared with monotherapy. Factors influencing selection include efficacy, cost, side effects, dosage, and frequency, and the effect on the diurnal IOP curve.50 A meta-analysis of 10 studies looking at the additive effect of the above 3 medication classes when combined with PGAs revealed a statistically similar reduction in mean diurnal IOP ranging from approximately 2.3 to 3.0 mm Hg.51 As a reference comparison, initial monotherapy with PGAs was found to lower IOP by 8 to 8.6 mm Hg from an elevated baseline in the XLT study.52 One other reason for considering PGAs as first-line therapy is that these agents have been shown to lower nocturnal IOP better than other classes. Unfortunately, because glaucoma suspects are asymptomatic, adherence to medication can be poor, as demonstrated in 1 electronic dosing aid study where nearly 45% of patients took fewer than 75% of their prescribed doses.53 LASER TRABECULOPLASTY

Laser therapy such as SLT may benefit high-risk glaucoma suspects as either a primary therapy choice over medicines or as an adjunctive therapy.47,48 Selective laser trabeculoplasty is as effective as argon laser trabeculoplasty or a single ocular hypotensive agent in reducing IOP in glaucoma and ocular hypertension.54 The treatment effect is not equal among all individuals and also decreases over time, but the procedure is repeatable.55
Adverse effects are minimal, such as postlaser intraocular inflammation or transient IOP spikes, which generally resolve quickly. A few isolated cases of transient corneal stromal edema have been reported.\textsuperscript{56} The major advantages of laser trabeculoplasty over medications for glaucoma suspects are not having to worry about compliance, avoiding drop toxicity, and convenience. Positive cost savings have been modeled for SLT as a primary therapy.\textsuperscript{57} Given some patient reluctance to having a procedure performed initially, it is important to present medicine and laser trabeculoplasty equally to avoid bias among newly diagnosed patients. Even though PGAs are associated with a generally safe side effect profile, some individuals have concerns about the iris color and other periocular cosmetic changes, and these patients may be ideal candidates for SLT. One study published a 5-year follow-up of SLT versus medications in 58 Chinese eyes with POAG or ocular hypertension, and SLT showed a similar reduction in IOP compared with medical therapy alone.\textsuperscript{58} Thus, SLT use is associated with short-term evidence and some long-term data supporting its use as a first-line therapy for glaucoma suspects.\textsuperscript{49}

**CATARACT SURGERY**

Surgery is generally not a first-line therapy for glaucoma suspects, but there is mounting evidence that cataract surgery alone lowers IOP. If glaucoma suspects are taking eye drops and are going to have a cataract removed, then there is also an opportunity to combine cataract surgery with minimally invasive glaucoma surgery with strong safety profiles to control IOP without medication. Such an approach is controversial in individuals for whom a glaucoma diagnosis has not been confirmed. There seem to be some promising minimally invasive glaucoma surgery choices on the horizon that are performed in combination with cataract surgery.\textsuperscript{59} In addition, cataract removal can improve imaging of the optic nerve and assist in the interpretation of perimetric testing. One other benefit of cataract surgery before filtering surgery is that it can be advantageous to have already removed the lens both for technical reasons and for trabeculectomy failure rates after surgery.\textsuperscript{59} In addition, cataract removal can improve imaging of the optic nerve and assist in the interpretation of perimetric testing. Such an approach is controversial in individuals for whom a glaucoma diagnosis has not been confirmed. There seem to be some promising minimally invasive glaucoma surgery choices on the horizon that are performed in combination with cataract surgery.\textsuperscript{59} In addition, cataract removal can improve imaging of the optic nerve and assist in the interpretation of perimetric testing. One other benefit of cataract surgery before filtering surgery is that it can be advantageous to have already removed the lens both for technical reasons and for trabeculectomy failure rates after phacoemulsification.\textsuperscript{59} However, the emphasis here is not to convey cataract surgery as a treatment for glaucoma suspects, as there is no current evidence to support this, but to acknowledge the trend of IOP reduction in some patients.

Multiple retrospective studies suggest that cataract extraction produces a significant and sustained IOP reduction in individuals with open-angle glaucoma, ocular hypertension, and angle-closure glaucoma. For ocular hypertensives, analysis of the OHTS fellow eye data revealed that postoperative IOP after cataract surgery had a significant mean decrease of 16.5% for at least 3 years.\textsuperscript{61} In a recent study from India, clear lens extraction led to a significant reduction in IOP from 27 to 13 mm Hg at 12 months in 44 eyes with PACG and persistently raised IOP after laser peripheral iridotomy.\textsuperscript{62} Although that scenario is still controversial, symptomatic cataracts affecting daily life are indicated for removal and may lower IOP in those with narrow angles, hyperopia, and thick anteriorly vaulted lenses.\textsuperscript{63}

In summary, the diagnosis and management of the glaucoma suspect continue to evolve and are aided by advances in technology that allow us to confirm the disease earlier, follow progression more accurately, and treat with better therapeutic options.\textsuperscript{49}

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