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Glaucoma suspects: A practical approach

Syed Shoeb Ahmad

Abstract:

Glaucoma suspects are controversial clinical dilemmas. These individuals harbor certain risk factors or demonstrate some clinical features suggestive of an increased probability to develop glaucomatous optic atrophy in the future. These characteristics range from high intraocular pressure; optic disc, visual field, or retinal nerve fiber layer abnormalities; or abnormal angles to a positive family history of glaucoma and other risk factors. Individuals having these characteristics should be assessed diligently before a diagnosis of glaucoma is made. Glaucoma is a chronic, lifelong condition, having a negative impact on the quality of life, with an increased risk of medication-related side-effects, adverse economic impacts, and the need for lifestyle changes in the patient. Overdiagnosis and unnecessary treatment of such individuals is bereft of any advantage. This review aims to provide a practical blueprint for the proper diagnosis and management of such glaucoma suspects.

Keywords:

Glaucoma, intraocular pressure, ocular hypertension, visual fields

Introduction

Glaucoma constitutes a broad group of diseases characterized by a common attribute of optic nerve degeneration and retinal ganglion cell (RGC) loss. Due to the multiple etiologies and varying presentations of glaucoma, it is occasionally difficult to identify the disease at an early stage.

Glaucoma may remain undiagnosed in communities to the extent of 50%–90%.[1] Poor access to health-care facilities, lack of knowledge among patients, and physician-related factors such as improper training, lack of experience, and instrumentation lead to a failure to diagnose glaucoma. Conversely, studies have found that 15%–50% of glaucoma patients, who were started on antiglaucoma medications, did not meet the criteria of glaucoma. A number of factors are responsible for overdiagnosis. A fear that the patient may lose vision in the future due to glaucoma or the patient could be diagnosed by someone else, with a consequence of medicolegal suits, often forces ophthalmologists to initiate treatment in the absence of characteristic features of glaucoma. It is therefore essential to eliminate both under- and over-diagnosis by proper evaluation of all glaucoma suspects.[1-3]

A glaucoma suspect is defined as a person who has one or more clinical features and/or risk factors which increase the possibility of developing glaucomatous optic nerve degeneration (GOND) and visual deficiency in the future.[4-6] The objective of this review article is to present a practical approach to the diagnosis and management of such glaucoma suspects.

Clinical Features

Glaucoma suspects can be identified during routine screening of individuals for ocular or nonocular conditions. Red flags may come up during the assessment of patients who are referred for screening of

Department of Ophthalmology, Queen Elizabeth Hospital, Kota Kinabalu, Malaysia

Address for correspondence:
Dr. Syed Shoeb Ahmad, Department of Ophthalmology, Queen Elizabeth Hospital, 88587 Kota Kinabalu, Malaysia.
E-mail: syedshoebahmad@yahoo.com

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diabetic retinopathy or those on medications, such as hydroxychloroquine, ethambutol, steroids, and others, to rule out drug-induced toxicities. They may also come to attention during screening of families with glaucoma or population-based studies related to glaucoma.

Individuals are usually regarded as a glaucoma suspect due to the presence of any of these following characteristics:[4]

1. Elevated intraocular pressure (IOP)
2. Optic nerve head (ONH) or retinal nerve fiber layer (RNFL) appearance suggestive of glaucomatous damage
3. Unexplained visual field (VF) defect consistent with glaucoma
4. Abnormal angles
5. Strong family history of severe glaucoma and other risk factors.

Glaucoma suspect with elevated intraocular pressure

The most common cause for individuals to be included in the category of glaucoma suspects is elevated IOP alone in the absence of other features of glaucoma. Sources of error during IOP measurement, such as uncooperative patients, tight neckties, uncalibrated tonometers, and other confounding factors should be excluded.[7] Ocular hypertension (OHT) is defined as IOP >22 mmHg (2 standard deviations above the mean), but without any other abnormal features in the optic discs, VFs, or RNFL.[4] In the absence of other features of glaucoma, if IOP is found to be consistently high on 3 consecutive examinations then, a diagnosis of OHT can be made. Therefore, the diagnosis of OHT is one of exclusion after ruling out any features suggestive of primary open-angle glaucoma (POAG).

Patients suspected of OHT can be assessed according to the following paradigm:

Establish a baseline

An individual with elevated IOP should be evaluated as any other glaucoma patient. Certain routine initial investigations which should be performed include serial IOP measurements; gonioscopy; optic disc photos (if possible, stereoscopic); central corneal thickness (CCT); and VF and RNFL thickness (RNFLT) assessments. Other investigations, such as ultrasound biomicroscopy, scanning laser polarimetry, and confocal scanning laser ophthalmoscopy, can be individually customized depending on their availability or indication. IOP should be checked on three different days at different times of the day so that any IOP spikes are not missed. The time should be recorded duly in the medical records to determine any diurnal fluctuation of IOP. Attempts should be made to obtain at least 3 VFs during the 1st year of assessment.[8] Optic disc photos are also important since they are less likely to show change compared to VFs which might not always be reliable. RNFLT assessment is becoming common with the availability of optical coherence tomography (OCT) machines. OCT allows quantitative RNFLT measurements, as well as evaluation of ONH topography and macular thickness. Among these, RNFLT assessment remains the most reliable, even as a stand-alone parameter. In a study to determine and compare the diagnostic performance of OCT, stereoscopic disc photos, and VFs among a group of glaucoma specialists, it was found that OCT had better internal agreement as well as better agreement with the consensus of clinicians.[9] Certainly, OCT is still far from perfect due to the acquisition and biological factors which can affect the interpretation of the findings. Whenever possible, both structural (OCT) and functional (VF) changes should be analyzed since one could precede the other.[10] A combined evaluation of these parameters (Bayesian method) is more advantageous compared to individual analyses for the detection of glaucoma. Other imaging techniques for glaucoma are also available. They include confocal scanning laser ophthalmoscopy (e.g., Heidelberg retinal tomography) and scanning laser polarimetry (e.g., GDx Nerve Fiber Analyzer). Now, newer imaging technologies are on the horizon which could identify the presence or absence of glaucoma at the cellular level. One such technique is detection of apoptosing retinal cells technology which is being studied to diagnose the appearance of apoptosis as a primary event in glaucoma, before OCT or VF changes.[11] A number of other imaging techniques such as swept-source OCT and OCT-angiography are being evaluated to improve the early diagnosis of glaucoma.

Determine the potential risk of glaucomatous optic nerve degeneration

While managing patients with elevated IOP, it is vital to distinguish between individuals who will probably not progress to GOND versus those who might. Certain factors are found to be associated with an increased risk of conversion to POAG.[4] These include:

i. High initial IOP
ii. Positive family history of glaucoma
iii. History of retinal vein occlusion (RVO)
iv. Disc hemorrhage
v. High myopia (>6 D)
vi. Increasing age (>70 years).

IOP is an important parameter in the management of glaucoma since it is the only modifiable risk factor at present. The OHT treatment study (OHTS) found that high IOP alone can be a risk factor for the development of glaucoma. The OHTS reported that a 1 mmHg increase in IOP was associated with a 10% increase in relative risk of conversion to POAG. The study also found that a 22.5% decrease in IOP in treatment arm (vs. 4% in control arm)

was associated with a reduction in the development of POAG from 9.5% in controls to 4.4% in the treatment group at 60 months of follow-up.[12] Therefore, there is unequivocal evidence of IOP being an important risk factor for the development of glaucoma. It is also a common factor responsible for the overdiagnosis of glaucoma. Due to the perceived risks of high IOP and fear of lawsuits, individuals are often started on antiglaucoma medications, despite the absence of other features of glaucoma.[1]

Family history of glaucoma should always be inquired in cases of glaucoma-suspects. Epidemiologic and genetic studies have found a positive linkage of glaucoma with family history. The Baltimore Eye Survey reported that after considering age-adjusted associations of POAG with a family history of glaucoma, there is a 3.69 times higher risk of development of POAG in siblings than in parents (odds ratio = 2.17) or children (odds ratio = 1.12).[13] Genome-wide association studies have also found a number of genes responsible for congenital and familial glaucoma, while no such consistent genetic association could be found in sporadic glaucoma.[14]

Glaucoma-suspects having a history of RVO should be regarded to have an additional risk factor. In a study by David *et al*., it was found that patients with increased IOP and/or glaucoma have a higher prevalence of RVO than persons with no history of elevated IOP.[15] Occasionally, only splinter hemorrhages can be seen on the optic disc. In the absence of other factors such as hypertension or diabetes, these so-called Drance hemorrhages signify the appearance of focal disc damage and VF loss.[5] Nonetheless, splinter hemorrhages can occur in other conditions such as posterior vitreous detachment, optic disc drusen, vascular occlusive retinal diseases, nonglaucomatous optic neuropathy, leukemia, and lupus. In the presence of disc hemorrhages in a patient with OHT, the risk of conversion to POAG increases by 6 times (by univariate analysis) and 4 times (by multivariate analysis). The Asia Pacific Glaucoma Guidelines mention that compared to a single episode of disc hemorrhage, recurrent hemorrhages increase the risk of optic nerve damage by 3–4 times.[16]

Refractive errors should be assessed in all glaucoma suspects. A number of epidemiological studies such as the Blue Mountains Eye Study, Barbados Eye Study, Beaver Dam Eye Study, Singapore Malay Eye Study, and others have shown a positive association of POAG with the increase in the degree of myopic refraction.[17-20] Conversely, other studies such as the OHTS did not show a significant relationship between the two conditions.[19] In a review of high myopia as a risk factor for POAG, Chen reported that the link between myopia and increased susceptibility or progression to glaucoma remains controversial.[21] Regarding hypermetropia, the Beaver Dam Eye Study had reported that such individuals have a 40% more likelihood to have OHT compared to emmetropic individuals at baseline. No such association was reported in myopic individuals.[19] Studies have shown a higher prevalence of primary angle closure glaucoma (PACG) in hyperopic individuals, especially when above 2D.[22,23] However, von Romunde did not find any statistically significant correlation between refractive error and PACG.[24]

Increasing age as a risk factor for the development of glaucoma has been identified in a number of studies. The Barbados Eye Study and Blue Mountains Eye Study found age to be a major risk factor for the development of POAG.[16,17] The Advanced Glaucoma Intervention Study also reported a 30% increase in the odds for VF progression for every 5 years' increment of age.[25] Since age is positively associated with a higher prevalence of glaucoma, it is recommended to screen all possible persons above 40 years of age for this disease.

CCT was found to be a powerful predictor for the development of POAG in OHTS. The relative risk of POAG increased 81% for every 40 μ thinning of cornea.[12] Both OHTS and the European Glaucoma Prevention Study found that the risk of developing POAG was greater in eyes with CCT <555 μ compared with eyes having CCT of 588 μ or greater.[26] Another study has reported that patients with thinner corneas tend to have more severe glaucoma even on initial examination and have a higher risk of progression.[27] The actual IOP can be overestimated on Goldman applanation tonometry in eyes with thicker corneas, whereas an underestimation may happen in eyes with less than average CCT. Refractive surgery can alter the corneal biomechanics and corneal thickness, thus resulting in falsely low IOP readings.[6] However, in the presence of corneal edema IOP tends to be underestimated and is overestimated in over corneal scars due to the increased rigidity of fibrous tissue.[28] It is therefore mandatory that all glaucoma suspects undergo pachymetry so that their IOP can be assessed in the proper perspective. It has not been verified if CCT is regarded as a risk factor because of its effect on IOP measurements or an independent risk factor unrelated to IOP. To overcome any errors, a number of algorithms have been developed to provide correction factors between IOP and CCT.[29] Currently, no linear correction formula for the two parameters is available.[6] In the absence of a universally acceptable formula, the World Glaucoma Association Consensus on IOP suggested that correction factors should not be used in individual patients.[30] Furthermore, a study of East Asian individuals did not find any evidence of thinner corneas being an independent risk factor for the development of glaucoma.[31] If possible, CCTs should be

assessed for all glaucoma suspects, at least on the first visit, to obtain a baseline.

Apart from CCT, other biomechanical parameters such as corneal hysteresis (CH) have been identified which could be related to the development and progression of glaucoma. Hysteresis is defined as the difference between the pressure at which the cornea bends inward from an airjet applanation and the pressure at which it rebounds again. This difference is used to assess a biomechanical property of the cornea related to its elasticity.[32] CH was found to be lower in glaucomatous, compared to nonglaucomatous eyes. It was also reported that lower CH values were associated with progression of glaucomatous VFs, independent of CCT.[33] Thus, CH may play a role in the assessment of glaucoma suspects, although so far its importance remains to be established.[34]

Optic nerve head or retinal nerve fiber layer abnormalities

Optic nerve head

The ONH should be evaluated on slit-lamp biomicroscopy using appropriate lenses. The size of the disc is also a factor to be considered in the assessment of cupping. Large discs appear to have larger cups. When examining optic discs, the magnification factor of the condensing lens used for slit-lamp biomicroscopy should be taken into account.[35] Abnormal cupping or an increase in the cup-to-disc ratio (CDR) is frequently associated with glaucoma suspects. Stereoscopic evaluation of the optic discs has shown a Gaussian distribution of the mean CDR at 0.4 with only an approximately 5% normal population having CDRs of 0.7 or more. A difference of 0.2 between the two eyes should be viewed with suspicion. Such a finding is present in only 1% of the normal population.[36] Studies have shown that both cup and pallor enlarge slightly with age. However, the age-related enlargement of optic cup is gradual, compared with the more rapid progression of GOND. While in physiologic cupping, despite a high CDR, the sizes of the cups bilaterally are nearly symmetrical.

Particular attention should be paid to the neuroretinal rim (NRR). The rim is broadest inferiorly among all quadrants, followed by the superior, nasal, and temporal rims (ISNT-rule). The loss of inferior or superior NRR leads to a vertical elongation of the cup and loss of the ISNT-rule leading to a suspicion of glaucoma.[37,38] It should, however, be kept in mind that the ISNT-rule applies to normal sized discs only. In large optic discs, a large cup might be confused for GOND. In nonglaucomatous optic discs, despite a large cup, the NRR remains stable and healthy. On an average, non-glaucomatous black individuals African-Americans have larger disc areas and large CDR compared to whites Caucasians, although, a substantial overlap is present although a substantial overlap is present. Increased size of the physiologic cup may also occur as a familial trait or seen in conditions such as high myopia.[5]

While glaucoma is associated with an increase in CDR, it can also lead to increased pallor, but sparing the remaining NRR. Vascular causes are commonly associated with disc pallor. In the absence of other significant histories, previous episodes of high IOP may also cause disc pallor. Therefore, a history of acute momentary visual obscuration, redness, and pain in the eye has to be enquired.[39]

Glaucomatous optic atrophy shows certain characteristic features which help to differentiate it from physiologic cupping. These mechanical and vascular signs seen in glaucomatous discs are given in Table 1.

Stereophotographs of the optic discs are ideal to preserve the records of the patient for future reference.[5] However, computerized digital images or even hand drawings may suffice, with the relevant details marked in the drawing. The mechanical and vascular signs mentioned in Table 1 can be incorporated while recording the findings in the case notes.

Retinal nerve fiber layer thickness

The RNFL consists of bundles of axons of the RGCs. They can be observed using red-free (green) light on slit-lamp biomicroscopy or by capturing the image photographically. In healthy retinas, the nerve fiber bundles appear as fine, bright striations which follow an arcuate distribution from the ONH, an area where they are best visible. The RNFL is easiest to identify in the inferotemporal quadrant, followed by the superotemporal, superonasal, and inferonasal quadrants. The RNFL striations should be compared above and below the horizontal meridian to look for any differences. Age-related changes may decrease the visibility of the RNFL, while in glaucoma patients, RNFL loss may appear as generalized attrition, slit defects, or wedge-shaped defects.[40-42]

Table 1: Ophthalmoscopic signs suggestive of glaucomatous optic nerve degeneration

Mechanical signs	Vascular signs
Large optic cup	Disc hemorrhage
Asymmetrical cups	Nasal displacement of vessels
Progressive enlargement of cup	Baring of circumlinear vessels
Narrowing/notching of rim	Tortuosity of retinal vessels on the disc
Vertical elongation of cup	
RNFL loss	
Exposed lamina cribrosa (laminar dot sign)	
Peripapillary crescent	

RNFL=Retinal nerve fiber layer

A number of imaging techniques have been developed to assess the RNFL. These include the confocal scanning laser ophthalmoscope, scanning laser polarimetry, and OCT. The last mentioned technique is most commonly used. It is a noncontact, noninvasive *in vivo* tool which provides high-resolution images of the RNFL, ONH, and macula. Glaucoma patients show thinning of the peripapillary RNFL, providing structural evidence of glaucomatous damage. Assessment of RNFL has been found to be extremely useful in glaucoma suspects, especially when differentiation from myopia or physiologic cupping is required.[43] Changes seen on OCT can also be used as a pointer to anatomical changes in optic discs. A more diligent repeat examination of the ONH focused on OCT findings can reveal changes missed on an earlier inspection of the optic disc. However, in certain cases, measurement of RNFLT and ganglion cell complex may provide abnormal data which leads to a suspicion of glaucoma in the absence of other clinical signs of the disease. It should be remembered that false-positive findings can occur on OCT, a feature known as "red disease."[44] This is due to a lack of the acquired data in the normative database of the machine (e.g., in high myopia). Similarly, the machine may only assess normal areas, miss out the damaged areas of the retina and provide erroneously normal outputs in glaucoma patients. This is known as "green disease."[45] Therefore, single measurements of these parameters should not be regarded as diagnostic. The tests should be repeated over time to look for definitive changes from baseline indicative of glaucoma. Certain software in the OCT machine, such as the Glaucoma Progression Analysis, can be utilized to assess progressive disease.[4] Progression analyses (progression event detection) are advantageous as they do not require normative databases needed for interpretation of OCT findings. It also obviates other confounding factors such as racial differences and differentiation from high myopes. Localized progression events which are repeated in two or more successive images are compared to baseline images (red events) to provide better diagnostic accuracy.[46] Newer parameters such as the Bruch's membrane opening-minimal rim width have been found to have high association with glaucomatous functional changes seen also on VFs, and thus, have a better ability to detect early glaucoma.[47] Consequently, it needs to be highlighted that undue reliance on newer glaucoma diagnostics leads to overdiagnosis of glaucoma when interpreted in isolation and without considering the complete clinical picture.[1]

Suspicious visual fields

VF assessment has traditionally been performed by automated static threshold perimetry utilizing the 30-2 or 24-2 programs. However, De Moraes *et al*. have recently reported that some glaucoma suspects may reveal central defects on 10-2 tests which are missed on 24-2 programs.[48] Traynis *et al*. have also reported that as much as 16% of eyes with a normal 24-2 VF result show significant abnormalities on the 10-2 test.[49] Macular VF defects found in glaucoma can range from arcuate scotomas to papillofoveal horizontal step ("pistol-barrel") defects.[50] In a symptomatic patient, the appearance of a VF defect should direct the examiner to the onset (date, circumstance, and associated complaints) and subsequent course of the symptoms. A VF defect which appears suddenly is unlikely glaucoma. In such cases, the examiner should rule out conditions such as anterior ischemic optic neuropathy. A visually significant symptom which was noted by the patient some time back and which seems to have worsened could be due to glaucoma, cataract, or a slow-growing compressive lesion.[4]

As perimetry remains a highly subjective test, on an average, 3 VF assessments should be done in the 1st year to detect an overall change in mean deviation of 4 dB over 2 years in a patient with average VF variability. Progressive VF loss is the hallmark sign which separates a true pathology from a glaucoma suspect.[6] The VFs should be scanned for atypical features such as central scotomas (examine the foveal threshold value) and asymmetry across the vertical midline. Such abnormalities are usually not seen in glaucoma.[4] The central scotomas can be assessed by Amsler chart testing and OCT should be done, if available. This will rule out any macular pathology. Slit-lamp examination should be done to look for trauma and conditions causing intermittent IOP elevations, for example, pigment dispersion or narrow angles. A dilated fundus examination should be done to rule out any retinal or macular scars which could produce VF defects. Other abnormalities such as subtle staphylomas or signs of previous vascular occlusion, for example, vascular attenuation, shunt vessels, and peripheral hemorrhages should also be looked for.[4]

In case a suspicious VF result is obtained on consecutive tests, which does not appear glaucomatous, certain medical conditions such as hypertension, hypotension, sleep apnea, and cerebrovascular accidents should be ruled out to exclude the possibility of microvasculopathy induced optic nerve damage. Furthermore, neurosurgical conditions such as space-occupying lesions should be excluded by appropriate imaging techniques. A neuroradiologic examination, with a computerized tomography or magnetic resonance imaging scan, is indicated in the presence of following features:[4]

i. Unexplained decrease in visual acuity (e.g., an unexplained central scotoma)

ii. Unusual VF loss (field loss other than nerve fiber bundle defects or paracentral scotomas)
iii. Bitemporal or homonymous hemianopic defects
iv. Pallor disproportionate to cupping unless the pallor is clearly confined to either the upper or lower half of the nerve (likely related to a vascular event)
v. Uniocular field loss not explained by obvious asymmetry or concurrent unilateral disease.

Abnormal angles

Certain gonioscopic findings often lead to a suspicion of glaucoma.[4] The following abnormal characteristics which lead to a suspicion of this condition include:

1. Narrow angle (Iridotrabecular contact over >3 quadrants)
2. Congenital anomalies of the angle, for example., Axenfeld–Reiger syndrome
3. Angle recession.

Patients with narrow angles are prone for ACG. However, not all individuals with narrow angles develop such glaucomas. Unfortunately, provocative tests and population-based studies have not been able to accurately diagnose eyes susceptible to develop ACG. Patients with angle anomalies and angle recession should undergo annual reviews with measurement of IOP, gonioscopy, and VF assessment; to rule out any suspicion of development of glaucoma.[4] Improper gonioscopy techniques can lead to misdiagnosis of the condition. Thus, the procedure should be practiced and mastered to avoid any procedural errors. A comprehensive review of the procedure is presented elsewhere.[51] The management of patients with narrow angles is discussed further in the treatment section.

Strong family history of severe glaucoma and other risk factors

Normotensive individuals with a family history of severe glaucoma but without any personal features suggestive of glaucoma can be followed up yearly with all routine investigations. Optic disc photos are useful because nonglaucomatous causes are less likely to produce changes in the photos compared to VFs. Patients may also have poor response to perimetric tests sometimes, especially with increasing age and development of cataract. Thus, optic disc photos provide more robust comparison on follow-up. In case there is abnormal rise in IOP or changes in the optic discs, patients require more extensive evaluation to confirm progression to glaucoma.[4] History of other conditions such as migraine, peripheral vasospasm, and sleep apnea is also contributory in the assessment and follow-up of patients suspected of normal tension glaucoma. A history of steroid use in any form is also pertinent as steroid-induced glaucoma is liable to be missed at times, unless specifically asked for.[52,53]

Treatment of Glaucoma Suspects

At present, the treatment of glaucoma is limited only to control of IOP. However, in glaucoma suspects, occasionally measures can be undertaken to prevent the development of glaucoma. Depending on the risk category, IOP reduction can be considered, after discussing the condition with the patient. It is imperative to consider the possibility of progressive visual loss against the expected life expectancy, treatment-related side-effects, financial impact, and negative effects on the quality of life of the individual. Patients with low risk may opt for yearly monitoring of their condition. Conversely, those in the medium-to-high-risk category can be encouraged to have their review every 6 monthly or less, depending on clinical judgment.[54] A grading scale of glaucoma patients is given in Table 2. Treatment can be started with a prostaglandin analog as they have to be instilled only once per day, have an effective IOP lowering capability and are devoid of significant side effects.[6] Another possible option is to use brimonidine as it could have a neuroprotective capability.[55]

The single most important factor in the diagnosis of glaucoma is progression in the parameters. OHTS reported that 9.5% of the patients in the observation-only group progressed. Thus, 90.5% did not. Therefore, not all patients need to be treated.[5] A 20% decrease in IOP should be tried first in the medium-to-high-risk individuals. Any patient with IOP above 40 mmHg should be considered at high risk and started on treatment to lower the IOP.[4] Others consider baseline IOP of >30 mmHg an indication to start treatment. Patients with an IOP of >28 mmHg and CDR of >0.6 require treatment due to the high risk of developing glaucoma. However, in patients with IOP between 22–30 mmHg, other factors may need to be taken into account before

Table 2: Grading of glaucoma severity

Stage	Characteristic feature
Mild- or early-stage glaucoma	Optic nerve abnormalities consistent with glaucoma but no visual field abnormalities on any white-on-white visual field test or abnormalities present only on short-wavelength automated perimetry or frequency doubling perimetry
Moderate-stage glaucoma	Optic nerve abnormalities consistent with glaucoma and glaucomatous visual field abnormalities in one hemifield and not within 5° of fixation
Severe-stage glaucoma, advanced-stage glaucoma, end-stage glaucoma	Optic nerve abnormalities consistent with glaucoma and glaucomatous visual field abnormalities in both hemifields and/or loss within 5° of fixation in at least one hemifield

starting treatment.[51] Apart from topical treatment, argon laser trabeculoplasty (ALT) or selective laser trabeculoplasty can be used as alternative or additional therapy. The Glaucoma Laser Trial reported the effectiveness of ALT to be equivalent to that of treatment with timolol.[56] Some studies have shown that cataract extraction alone may cause lowering of IOP. Cataract surgery can be combined with microincisional glaucoma surgery (MIGS) implantation to further reduce IOP. The advent of MIGS has provided another option to treat patients while conserving the conjunctiva for further incisional procedures if required in the future. Cataract surgery helps in an improved assessment of the optic nerve and RNFL. It also obviates certain errors associated with VF testing. As the duration between the cataract and a future glaucoma filtering surgery is prolonged, it reduces the risk of trabeculectomy failures.

Other indications to start or modify treatment on review are the appearance of the following features:[4]

i. Disc hemorrhage
ii. Increased cupping
iii. Glaucomatous VF defect.

Increased cupping should be documented on serial examinations. To confirm the deepening/widening of old scotomas or appearance of new ones, repeat VF testing is required.

Individuals with narrow angles can be considered for prophylactic peripheral iridotomy. The indications for this procedure are given in Table 3.

Conclusion

Glaucoma suspects form a diverse group of individuals who pose a diagnostic dilemma. As the diagnosis of glaucoma is a life-changing condition, which can lead to depression and affect the quality of life of the individuals, caution should be exercised before labeling an individual with this condition.[57] This article provides a feasible blueprint for the diagnosis and management of such glaucoma suspects.

Table 3: Indications for prophylactic peripheral iridectomy

ACG in the contralateral eye
History of transient episodes of blurring of vision, colored halos, or pain (usually self-limited)
Elevation of IOP associated with crowding of the angle on pupillary dilatation
Transportation or economic difficulties for the individual in case of emergency
Significant anxiety in the individual regarding risk of ACG

ACG=Angle-closure glaucoma, IOP=Intraocular pressure

Ethical approval

The study was conducted in accordance with the Declaration of Helsinki and was approved by the local ethics committee of the institute. Informed written consent was obtained from all patients prior to their enrollment in this study.

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Conflicts of interest

The authors declare that there are no conflicts of interests of this paper.

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