ORIGINAL ARTICLE

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Risk factors for primary open-angle glaucoma in Filipinos

ABSTRACT

Objective

This study determined the risk factors for primary open-angle glaucoma (POAG) among Filipinos.

Methods

This is a case-control study of POAG cases and controls recruited from the University of the Philippines-Philippine General Hospital (UP-PGH). All underwent a comprehensive eye examination consisting of best-corrected visual acuity, applanation tonometry, slitlamp biomicroscopy, gonioscopy, fundus evaluation, automated perimetry, optic-disc photography, and a standardized questionnaire interview of medical history and family history of glaucoma and other systemic diseases. Visual-field results and optic-disc photos were graded by 2 glaucoma experts as to the presence or absence of glaucomatous optic neuropathy. Risk factors studied were subjected to multiple logistic regression.

Results

A total of 365 participants (164 males, 201 females), mean age of 58.7 years, were subdivided into 193 controls (no glaucoma) and 172 POAG cases. The participants with glaucoma were older, had poorer visual acuity, larger optic cupping, and worse global indices in the visual field. Those 60 years and over had an increased risk of POAG. There was a threefold increased association with POAG for each unit decrease in visual acuity. Enlarged vertical cupping of the optic disc (0.7 or greater) showed 5 times increased risk of developing POAG. There was a 29% and 45% increased risk for POAG for every decibel increase in mean defect and pattern standard deviation respectively. Of the systemic diseases studied, including family history of major medical conditions, only family history of hypertension was strongly associated with POAG, with 2.5 odds of increased association with glaucoma.

Conclusion

The causes of POAG are multiple and complex. Older age, poorer visual acuity, large vertical disc cupping, worse visual-field global indices, and family history of hypertension were associated with POAG in this study.

GLAUCOMA afflicts more than 67 million people worldwide, of whom about 10% are estimated to be blind.[1] It is the leading cause of irreversible blindness worldwide and is second to cataract as the most common cause of blindness in the developing world.[2] Even though economic data on the cost of glaucoma are limited, it is believed that the social and economic impact of glaucoma is enormous. With increasing costs of medication, consultation, and an aging population, the impact of glaucoma is likely to increase and become a public-health problem.

The Third National Survey on Blindness in the Philippines showed that glaucoma is the third major cause of visual impairment, after cataract and refractive error, with a prevalence of 3%.[3] This translates to approximately 2.38 million Filipinos who have visual impairment from glaucoma in at least one eye.

To reduce the incidence of blindness from glaucoma, screening large population has been suggested, but it is costly and likely to yield false-positive and false-negative errors. This is because the diagnosis of glaucoma in its early stage is not straightforward and requires correlation with structural changes in the optic-nerve head (ONH) and functional defects in the visual field. Documenting both structural and functional changes in a large population requires expensive special equipment and time-consuming procedures. Obtaining a single intraocular-pressure (IOP) measurement has only moderate sensitivity and low specificity. In the Baltimore Eye Survey (BES), less than half of those with diagnosed glaucoma had IOP greater than 21.[4] Thus, using IOP measurement as a screening tool is not sensitive enough to detect majority of those at risk of developing glaucoma.

There are many factors that can affect the causation of glaucoma. The literature has identified age,[5-10] elevated IOP,[11-17] race,[6,18-21] and family history[22-27] with strong supporting evidence as risk factors for primary open-angle glaucoma (POAG). Increasing age is associated with a higher incidence of the disease as shown in most population-based studies. Clinical and experimental studies have also shown that elevated IOP beyond a critical level also leads to glaucomatous optic-nerve damage. An individual's susceptibility to the disease is influenced by his genetic makeup, i.e. family history of the disease, as supported by studies on twins and siblings. But the expression of the susceptibility varies. Many glaucoma patients deny any family history. This implies that there are other factors that influence this susceptibility.

Other factors that can influence the development of glaucoma are vascular in nature, supporting the contention that ischemia is another mechanism by which glaucoma damage can occur. Such conditions are systemic hypertension,[28-34] diabetes mellitus,[35-42] thyroid diseases,[43-47]

and migraine.[48-53] Literature review of these factors presents conflicting data.

Myopia, specifically high myopia, has also been shown to have an increased prevalence of POAG, possibly due to the altered rigidity of the sclera at the level of the lamina cribrosa[54-57] seen in an elongated eyeball. The relationship, however, is confusing.

Screening those at risk for developing glaucoma is likely to be more cost-effective. Hence, we determined the risk factors (probability of an individual at risk of developing the disease) for POAG in Filipinos. We evaluated whether the following parameters are risk factors: (1) age, (2) sex, (3) refractive error, (4) diabetes mellitus, (5) systemic hypertension and other cardiovascular disorder, (6) thyroid diseases, (7) migraine, (8) family history of glaucoma, (9) smoking, (10) alcohol consumption.

METHODOLOGY

Glaucoma patients seen at the Glaucoma Clinic of the Department of Ophthalmology, University of the Philippines-Philippine General Hospital (UP-PGH) were recruited to participate in the study after informed consent was obtained. They were recruited as they come, consecutive and purposive. They were defined as having POAG based on the following inclusion criteria: (1) 40 years of age and older; (2) open angles on gonioscopy, and (3) definitive glaucomatous optic neuropathy (GON) based on the presence of at least one visual field and one optic-disc criteria in at least one eye after ophthalmologic exclusion on narrow angles, other types of glaucoma, and other possible causes.

The visual-field criteria consisted of the following: (1) distinctive glaucomatous visual field defects on 24–2 or 30–2 threshold test of the Humphrey perimeter; (2) if visual-field threshold test could not be performed, other test strategy such as full-field, 120-degree suprathreshold test or kinetic perimetry may be done and there was evidence of glaucomatous visual-field loss such as blindness or severe visual impairment.

The optic-disc criteria consisted of the following: (1) at least 2 signs of optic-disc damage present in fundus photographs and/or ophthalmologic examination, such as notching with associated neuroretinal-rim narrowing, nonconformity of the ISNT[58] rule, prominent β-zone peripapillary atrophy associated with neuroretinal-rim thinning, disc hemorrhage, focal retinal-nerve-fiber-layer (RNFL) defect, and asymmetry in cup-disc (CD) ratios between eyes of more than 0.2; (2) if photographs were not available, the ophthalmologic examination or other clinical records documenting GON.

The ophthalmologic criteria consisted of the following: (1) clinical POAG diagnosis after examination by the ophthalmologist; the diagnosis was based on the

assessment of disc and field changes, not on IOP, after excluding other causes; (2) confirmation of previous clinical POAG diagnosis and treatment through record review for participants who did not complete the ophthalmologic examination.

The following were excluded: less than 40 years of age; narrow or closed angle on gonioscopy; secondary types of glaucoma, such as pseudoexfoliation, pigmentary, uveitic, pseudophakic, aphakic, hemolytic, steroid-induced, angle recession or traumatic glaucoma; angle-closure glaucoma; congenital, developmental, or juvenile glaucoma.

The study protocol was reviewed and approved by the Ethics Review Board of UP-PGH.

Recruitment of cases and controls

Glaucoma patients seen at the Glaucoma Clinic of UP-PGH were recruited. On the day of their visit to the clinic, their medical records consisting of prior eye evaluations and previous automated perimetry were reviewed to determine eligibility. This initial screening established the presence or absence of POAG and inclusion as cases in the study. Patients with either eye satisfying the inclusion criteria were included in the study.

Control subjects were recruited at UP-PGH. They consisted largely of companions ("bantays") of patients seen at the eye department.

Controls were defined based on the following: 40 years or older, open angle on gonioscopy with no evidence of glaucoma of any type and normal visual-field and optic-nerve-head findings. Those with narrow or closed angle on gonioscopy, and less than 40 years of age, and "bantay" of cases were excluded.

Informed consent was obtained from all eligible participants following the tenets of the Declaration of Helsinki. The risks and benefits of the study and the procedures to be done were explained.

Each underwent a comprehensive eye examination consisting of the following: determination of best-corrected visual acuity with refraction, applanation tonometry, slitlamp biomicroscopy, gonioscopy, and fundus evaluation. In addition, a questionnaire interview was performed by the same interviewer who was masked as to the status of the eye findings and diagnosis. All participants also underwent automated perimetry with the Humphrey Field Analyzer (Carl Zeiss, San Dublin, CA, USA) testing the threshold of the central 30-degree field. The pupils were dilated for optic-disc photography.

All data were entered into a standard data collection form by the examining ophthalmologist and the questionnaire interviewer. All forms were further reviewed by a research associate for consistency and completion. Coding was performed for each parameter and data were entered into MS Excel (Microsoft Corporation, Redmond, WA, USA).

The optic-disc photographs were graded by two glaucoma specialists as to the presence or absence of GON. The visual fields were assessed by the same specialists as to the presence or absence of definite glaucomatous field defect, following the minimum criteria set in the American Academy of Ophthalmology Preferred Practice Pattern.[59]

The following risk factors were included in the questionnaire interview adopted from the BES[22] and the Beaver Dam Eye Study:[60]

1. *Family history of glaucoma in parents, siblings, children.* Participants were asked to identify their first-degree relatives and whether each had a history of glaucoma. Only positive responses were accepted as a positive history in the relative. Negative answers or "don't know" responses were considered negative.

2. *Smoking history.* Participants were asked in the interview if they currently smoke or had ever smoked greater than 100 cigarettes, and if so, how many cigarettes were smoked daily, for how many months and date of stopping. For categorical analyses, participants who smoked were classified as past or current smokers.

3. *Alcohol use* was determined by subdividing participants into current drinkers (any positive response for drinking in the past year) and current nondrinkers for each alcohol type (beer, wine, and liquor).

4. *Refractive error* for each participant was based on best-corrected spectacle correction using the spherical part of the prescription.

5. *Cardiovascular diseases* such as essential hypertension, ischemic heart disease, carotid-artery stenosis were considered present if the participant was being treated for such conditions with specific medications or had specific laboratory exams that were positive, or based on medical records or confirmation with the medical doctor. Current systemic hypertension was defined as blood pressure taken on two separate determinations greater than or equal to 140 mm Hg systolic or 90 mm Hg diastolic with or without medications.

6. *Diabetes mellitus* was defined as either a history of diabetes treated with insulin or oral hypoglycemic agents and/or diet, or glycosylated hemoglobin level greater than two standard deviations above the mean of the relevant age-sex group and a random blood-sugar level greater than 11.1 mmol/l.

7. *Thyroid disease* was checked from the interview questionnaire and confirmed by the treating physician. Any "don't know" or "not sure" responses that could not be verified by the treating physician were considered negative.

8. *Migraine headaches* were obtained from the questionnaire interview asking for specific questions as any recurrent, severe or pulsating headaches that is unilateral in character and may be associated with nausea, vomiting,

photophobia, and/or sometimes visual disturbance, occurring in the past. A history of migraine headaches was considered positive if it occurred more than 1 year from the initial diagnosis of glaucoma to differentiate associated ocular pain from elevated IOP of glaucoma.

Statistical methods

To determine the risk factors of POAG, multivariate logistic regression analysis was performed with Stata Release 6 (Stata Corporation, TX, USA) to identify the effects of each variable adjusting for the effects of other variables. Significance of the variables to be retained or removed from the model was judged using the Likelihood Ratio Test (LRT).

RESULTS

A total of 365 participants (164 males, 201 females) examined from June 2001 to July 2005 were included in the study. Mean age was 58.7 years. There were 193 controls (no glaucoma) and 172 cases with confirmed POAG. The participants with glaucoma were older, with a mean age of 62.1 years. Both sexes were equally affected. Those with glaucoma also had poorer visual acuity, with mean of 0.82 (20/25). More myopia was seen in those with glaucoma, with a wider spread toward the higher myopic refraction. Larger optic cupping, with a mean of 0.68, and higher values (or worse) for the global indices (mean defect and corrected pattern standard deviation) in the visual field were seen in the glaucoma group, indicating GON (Table 1). The IOPs in both groups were similar.

Univariate analysis showed that older age, worse visual acuity, larger vertical cupping, and worse visual-field mean defect were associated with POAG (Table 2). Those 60 years of age and over have an increased risk of having POAG than those less than 60 years. For every increase in age after 65 years, there is a 6% increase in risk per year for developing POAG. For each unit decrease in visual acuity as measured using the decimal system, there was an almost 3 times increased association with POAG. Enlarged vertical cupping of the optic disc, specifically 0.7 or greater, showed 5 times increased risk of association with POAG. Likewise, for every decibel increase in mean defect in the visual field, there was a 29% risk of association with POAG.

The multivariate analysis showed the persistent effects of global indices or higher mean defect and pattern standard deviation of the visual field, indicating strong association with POAG (Table 3). For every increase in decibel of the pattern standard deviation, there was a 45% increased risk of association with glaucoma.

Of the systemic diseases studied, including family history of major medical conditions, only family history

Table 1. **Demographic and visual characteristics of the study population.**

Variables	Controls	Cases	*p*
Age (years)			
Mean	56.2 ± 10.09	62.1 ± 10.36	< 0.001
Range	40 to 80	40 to 84	
Male: Female Ratio	34 : 61	30 : 40	0.19
Visual Acuity			
Mean	0.93 ± 0.17	0.82 ± 0.25	0.001
Range	0.33 to + 1.00	0.00 to +1.00	
Refraction (Spherical Equivalent)			
Mean	0.32 ±1 .63	-0.48 ± 2.17	0.008
Range	-7.50 to +3.0	-6.50 to +2.75	
IOP (mm Hg)			
Mean	12.6 ± 2.84	13.0 ± 3.18	0.40
Range	6 to 20	6 to 24	
Vertical Cupping			
Mean	0.54 ± 0.13	0.68 ± 0.18	< 0.001
Range	0.25 to 0.7	0.5 to 1.0	
Visual Field Mean Defect (Decibels)			
Mean	1.24 ± 3.11	8.53 ± 7.24	< 0.001
Range	-9.13 to 11.30	-0.74 to 33.0	
Visual Field Corrected Pattern SD (Decibels)			
Mean	3.42 ± 2.49	7.97 ± 3.67	< 0.001
Range	0.0 to 11.65	0.73 to 19.5	

Table 2. **Association of age, sex, and visual parameters with primary open-angle glaucoma (univariate analysis).**

Parameter	Coefficient	Odds Ratio	95% CI[a]	*p*
Age	0.06	1.06	1.03 – 1.10	< 0.001
Sex	-0.24	0.79	0.40 – 1.53	0.48
Visual Acuity	-2.89	0.06	0.01 – 0.30	0.001
Refraction	-0.17	0.84	0.68 – 1.04	0.12
Intraocular Pressure	0.05	1.05	0.94 – 1.16	0.38
Vertical Cupping	5.15	171.94	5.51 – 5369.58	0.003
Visual-Field Mean Defect	0.29	1.34	1.16 – 1.55	< 0.001
Visual-Field Corrected Pattern Standard Deviation	0.12	1.13	0.58 – 2.18	0.72

[a]Confidence interval

of hypertension was strongly associated with POAG, with 2.5 odds of increased association with glaucoma. Smoking and alcohol consumption were not associated with POAG.

DISCUSSION

Many chronic diseases, including POAG, are associated with aging. This study showed that those over 60 years of age are more likely to have glaucoma than those less than 60. For every increase in age after 65 years, there is a 6% increase in the risk per year for developing POAG. All population-based studies on prevalence and incidence consistently show a steady increase with age,[5-8] with a

Table 3. **Association of age, sex, and visual parameters with primary open-angle glaucoma (multivariate analysis).**

Parameter	Coefficient	Odds Ratio	95% CI[a]	p
Age	0.05	1.05	0.99 – 1.11	0.12
Sex	0.14	1.15	0.41 – 3.22	0.79
Visual Acuity	0.42	1.52	0.06 – 39.98	0.80
Refraction	-0.26	0.77	0.54 – 1.10	0.15
Intraocular Pressure	0.09	1.10	0.92 – 1.32	0.31
Vertical cupping	3.78	43.65	0.18 – 10885.67	0.18
Visual-Field Mean Defect	0.18	1.19	1.01 – 1.41	0.04
Visual-Field Corrected Pattern Standard Deviation	0.45	1.56	1.24 – 1.97	< 0.001

[a]Confidence interval

Table 4. **Association of systemic diseases and family history with primary open-angle glaucoma.**

Parameter	Coefficient	Odds Ratio	95% CI[a]	p
Aspirin use	0.40	1.49	0.44 – 5.01	0.52
Steroid use	1.32	3.73	0.23 – 59.54	0.35
Hypertension	0.12	1.12	0.43 – 2.91	0.81
Diabetes mellitus	-0.56	0.57	0.16 – 2.01	0.38
Heart disease	0.32	1.37	0.30 – 6.32	0.69
Thyroid disease	-1.25	0.29	0.04 – 2.25	0.24
Migraine	0.58	1.78	0.09 – 34.14	0.70
Family History				
Glaucoma	0.25	1.28	0.47 – 3.51	0.63
Hypertension	0.95	2.58	1.26 – 5.32	0.01
Diabetes	-0.29	0.75	0.36 – 1.55	0.44
Heart disease	-0.05	0.95	0.48 – 1.87	0.88
Thyroid disease	0.05	1.05	0.42 – 2.62	0.91
Migraine	-1.07	0.34	0.05 – 2.54	0.30

[a]Confidence interval

Table 5. **Association of cigarette smoking and alcohol consumption with primary open-angle glaucoma.**

Parameter	Coefficient	Odds Ratio	95% CI[a]	p
Cigarette smoking[1]	-0.46	0.63	0.60 – 6.61	0.70
Years smoked[2]	0.04	1.04	0.98 – 1.10	0.20
Current smoker[3]	-0.98	0.37	0.06 – 2.24	0.28
Alcohol drinker[4]	-0.09	0.92	0.27 – 3.10	0.89
Alcohol years[5]	-0.12	0.89	0.41 – 1.92	0.76

[a]Confidence interval
[1]Smoked cigarette regularly for more than 3 months.
[2]Number of years of smoking cigarette regularly.
[3]Smoked cigarette at least for the past year.
[4]Drinked alcoholic beverages regularly (at least 1 glass a day) for more than 3 months.
[5]Number of years of drinking alcoholic beverages regularly.

doubling of the prevalence per decade. The effect of age seemingly disappeared when other factors such as worse visual-field mean defect and pattern standard deviation were included in the model, both of which showed much stronger association with POAG. The relatively younger age (mean 62.5, median 64.5) of the glaucoma group in

this study could have resulted in a relatively weaker effect of age on the risk of developing glaucoma compared to other studies. Nevertheless, the effect of age in this population was highly significant in the univariate analysis (Table 2).

There was no predilection of POAG for either sex in this study even though more females participated in both groups. Among the population-based surveys done in the last two decades, the Barbados Eye Study showed male gender to be a major risk factor for POAG among its predominantly black population.[29]

Poorer visual acuity had almost 3 times the risk of association with POAG. Many in the glaucoma group had advanced glaucoma associated with poorer vision (less than 20/40) and large visual-field defects.

The type of refractive error was not associated with POAG even though there were more participants with myopia in the glaucoma group. Distribution of the refractive errors in both groups showed the same spread of refraction, with majority of those in the control group having 20/20 (6/6) vision and those in the glaucoma group having low myopic refraction. Several case series[54-55] and case-control studies[56-57] have reported an association of POAG with myopia, particularly high myopia, which is supported by several population-based prevalence surveys[61-63] reporting a prevalence of POAG in those with myopia ranging from 48 to 70%. However, individuals with myopia were not found to have a higher incidence or progression of glaucoma in the OHTS[64] or EMGT[42] respectively. A possible explanation for the difference is that studies reporting associations have a higher prevalence of high myopia ($\geq$ 6.0D). The participants in the glaucoma group in this study have low myopia ($\leq$ 3.0D) with a mean of only –0.48D.

A vertical cupping greater than 0.7 was associated with five times increased risk of developing POAG, significantly shown in the univariate analysis (Table 2). This effect was not seen in the multivariate analysis as the influence of visual-field defect was much stronger. Moreover, there were many in the normal group with large optic discs which were associated with larger optic cups. More than half in the normal group had vertical cupping between 0.5 to 0.7 with a mean of 0.54. In the glaucoma group, the mean was approximately 0.7 with majority greater than or equal to 0.7. Numerous studies have reported increased incidence of glaucomatous visual-field defects among those with larger CD ratios.[64-67] More recently, the OHTS showed a 1.4-fold increase in the incidence of POAG among ocular-hypertensive patients for every 0.1 unit increase in the baseline CD ratio.[64] Caution, however, should be exercised in the interpretation of studies that use ONH parameters (i.e., CD ratio, vertical-disc diameter, disc area) to define glaucoma. The estimates may be

inflated because the criteria for defining POAG may be inherent in the parameter itself, such as defining glaucoma as those with C/D ratio greater than 0.7.

Other ONH parameters, such as the presence of β-zone peripapillary atrophy and disc hemorrhages, have received attention in the glaucoma literature. In this study, none in the glaucoma group was noted to have disc hemorrhage, either examined clinically or in the disc photographs. The prevalence of disc hemorrhage in glaucoma was reported to be less than 30%[68] and more common in normal-tension than high-tension glaucoma. Disc hemorrhages are evanescent and may easily be overlooked unless specifically looked for. Moreover, its presence is a sign of active progression of the disease. All glaucoma patients in this study were already on glaucoma treatment or had filtering surgery. The mean IOP in the glaucoma group was 13.0 versus 12.6 mm Hg for control, with slightly higher standard deviation in the glaucoma group but with similar range. With IOP less than 25 mm Hg in the glaucoma group, the presence of disc hemorrhage was unlikely. Observation for β-zone peripapillary atrophy was not done in this study.

Worse global indices (higher mean defects and pattern standard deviations) used to characterize the visual-field defects were seen in the glaucoma group. The standard deviations were wide depicting the range of field defects in this group—from early to the advanced stage. Some of the changes in the early stage may be due to short-term fluctuations that were also present in the control group. One of the limitations in this study was that a single field was obtained. For a reliable diagnosis, a series of fields should be obtained to eliminate the effect of learning curve.

Many studies in the past have shown consistent association between IOP and glaucoma but it was only recently that a strong dose-response relationship has been shown in prevalence surveys[4-7, 29, 32, 36, 39, 69-70] and in longitudinal studies of incidence[17, 20, 25, 71] and progression.[71] The most decisive new evidence was the demonstration by randomized clinical trials that IOP lowering decreased the incidence[17] and progression of glaucoma[15-16] compared to no treatment. In addition, there is support for at least one biologic mechanism that links elevated IOP to apoptosis of neurons of the optic nerve through blockage of retrograde axonal transport.[72-73] Thus, IOP is both a risk factor for and a cause of glaucoma. This study was not able to demonstrate IOP as a risk factor primarily because the participants in the glaucoma group had already been treated either medically or surgically, such that the IOP range is similar to that of the control group.

Other risk factors considered were systemic diseases such as diabetes, hypertension, thyroid disease, migraine, use of specific medications, and family history of the systemic diseases mentioned earlier. None had significant association except for family history of hypertension (OR=2.58). A major limitation of questionnaire interview is the relative inaccuracy of the participants' responses. Studies have shown that recall by patients versus actual tests or confirmations by the primary-care physicians have large discrepancies. There were attempts to contact the attending doctors but the response rate was rather low, with many patients not having a permanent one.

Family history of glaucoma did not show any significant association with POAG in this study. Other studies have reported a relative risk between 2 and 4 for first-degree relatives.[22, 74-76] The odds ratio was higher if based on patients with previously diagnosed glaucoma than if based on newly detected cases,[76] suggesting that having a diagnosis of POAG leads to a greater awareness of glaucoma in the family. The population in this study was from the lower socioeconomic group who were most likely not as knowledgeable about their general health and that of their family. Moreover, they were less likely to seek medical consultation for an asymptomatic condition until late in the course of the disease. Hence, the accuracy of patients' responses in the questionnaire interview may be less reliable. Requesting examination of first-degree relatives of participants is preferred but time-consuming. Genetic-linkage studies are also more accurate but expensive.

Limitations of this study include biases inherent in a case-control study, such as the selection of cases and controls. Both were drawn from the same population—those who went to PGH for their health needs. Companions ("bantays") were recruited as controls since several ophthalmic factors were studied in association with POAG and those seeking eye care were more likely to have a higher prevalence of eye problems. Attempt at age matching was done initially at the start of the study to remove the effect of aging on systemic diseases and glaucoma, but there was difficulty recruiting older controls, especially those greater than 70 years with mild to no cataract and no other ocular problems. The recruitment of controls was done consecutively as they come, similar to the glaucoma group, and the result was a much younger control. For studying relatively rare diseases such as glaucoma, a case-control study design is most practical and economical and the information gathered could provide the basis for future population studies.

In summary, the causes of POAG are multiple and complex. Determining the risk factors for the disease is necessary to identify the target population that needed to be screened, such as the elderly (those older than 60), those with poorer visual acuity, elevated IOP, worse global indices on visual-field tests, large vertical disc cupping, and family history of hypertension to prevent irreversible blindness.

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