REVIEW ARTICLE

Glaucoma in Adults-diagnosis, Management, and Prediagnosis to End-stage, Categorizing Glaucoma's Stages: A Review

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Abstract

Importance: Most frequent worldwide cause of permanent blindness is glaucoma. Early in the course of the disease, glaucoma affects many patients without any symptoms. In order to examine for indications of glaucoma and to ascertain whether systemic illnesses or drugs can raise a patient's risk of developing glaucoma, primary care practitioners should be aware of which patients to send to an eye care specialist. A review of the pathogenesis, risk factors, screening, disease monitoring, and treatment options for open-angle and narrow-angle glaucoma are included.

Observations: The optic nerve and retinal nerve fiber layer (rNFL) are damaged in glaucoma, a chronic, progressive optic neuropathy that can result in a permanent loss of peripheral or central vision. The only risk factor that is known to be controllable is intraocular pressure (IOP). A family history of glaucoma, older age, and non-white race are additional significant risk factors. Numerous systemic diseases and drugs, such as corticosteroids, anticholinergics, certain antidepressants, and topiramate, can put people at risk of developing glaucoma. Open-angle and angle-closure glaucoma are the two main types of disease. Measurement of IOP, perimetry, and optical coherence tomography are diagnostic procedures to evaluate glaucoma and track the course of the condition. In order to treat glaucoma, IOP must be decreased. This is possible with a variety of glaucoma medication classes, laser surgery, and incisional surgery.

Verdicts and relevance: By identifying systemic illnesses and drugs that raise a patient's chance of developing glaucoma and referring high-risk individuals for a thorough ophthalmologic examination, vision loss from glaucoma can be reduced. Clinicians should make sure that patients continue taking their glaucoma drugs as prescribed and should keep an eye out for any negative side effects from any medical or surgical procedures used to treat glaucoma.

Keywords: Advanced glaucoma intervention study, Collaborative initial glaucoma treatment study, Closed angle glaucoma, Glaucoma, Glaucoma scrutility scale, Open-angle glaucoma.

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Introduction

The term "glaucoma," which would be derived from the Greek word "glaukos," is an ambiguous term that refers to IOP.[1]

It is a major contributor to irreversible blindness. As reported by the World Health Organization, 5.1 million individuals are mutually blind from glaucoma.[2]

The optic nerve head getting covered and impairment of the visual field are two symptoms of the diverse group of diseases known as glaucoma. The majority of the time, it is the reason for irreversible blindness worldwide. Progression often stops when IOP has fallen by 30–50% from baseline (Fig. 1).[3]

Degeneration and progressive loss of the optic nerve, together with the loss of retinal ganglion cells, progressive excavation of the optic disc, and thinning of the rNFL, are common symptoms of glaucoma.[1]

It is a notable public health issue because of its high fatality rate and ubiquity. It is important that medical professionals who have a family history of this disease are not often linked with the signs and symptoms that would alert the patient or the doctor to its presence because it is a treatable ailment.[2]

Around 3.5% of the population is 40 years of age or older, according to global standards. Chronic glaucoma forms have no pain and take time to develop symptoms, such as visual field abnormalities.[3]

Glaucoma has become a growing public health issue as the population rises and the number of people swells (Fig. 2).[4]

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Elevated IOP, African heritage, a supportive family, and advanced age are risk factors. Glaucoma is treated with surgery, laser therapy, or a local or systemic IOP -lowering medication.[2]

Utilizing ophthalmoscopy, tonometry, and perimetry, glaucoma is diagnosed.[3]

The examples of glaucoma range from mechanical angle closure of outflow structures in angle closure glaucoma patients,

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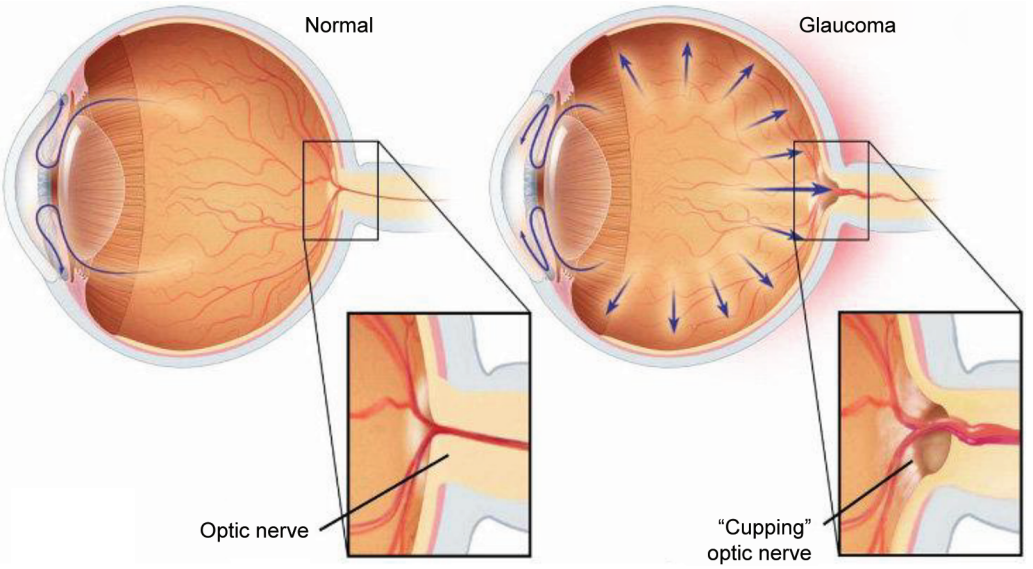


Fig. 1: Comparison between normal and glaucoma eyes

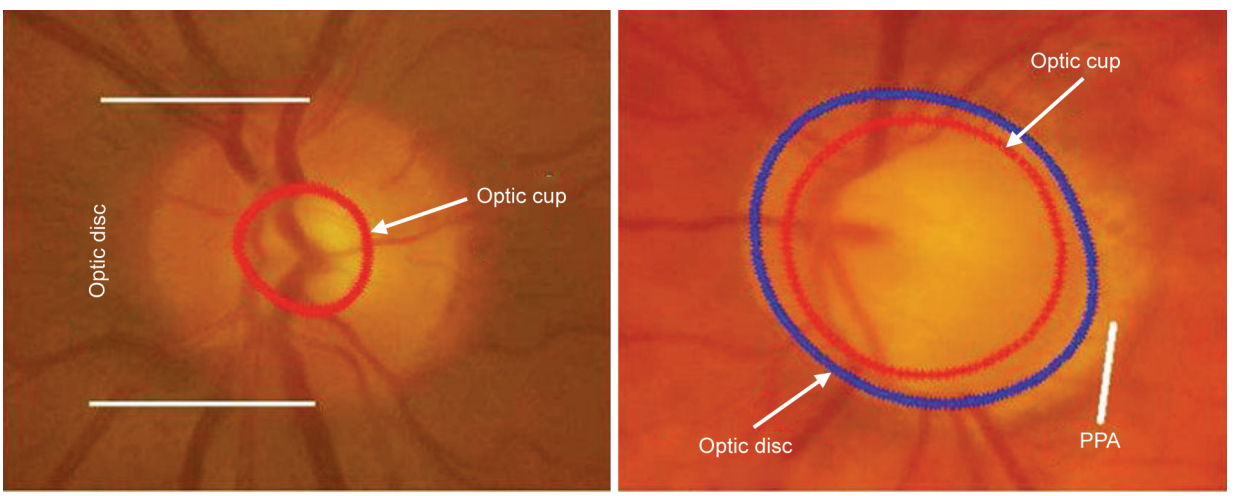


Fig. 2: Comparison of the optic disc area of the normal and glaucomatous image

who frequently present with acute visual loss and ocular pain, to increased or extended outflow resistance in patients with open-angle glaucoma (OAG), who are frequently asymptomatic.[5]

Glaucomatous Morphology of the Optic Nerve

The rNFL, which is made-up of ganglion cells and their associated axons, is harmed by glaucoma. This causes gradual and asymmetric alterations in the optic cup, along with a corresponding decrease in the visual field. Structural alterations often occur prior to functional loss. Up to 40% of the retinal nerve fibers may be damaged prior to the onset of visible alterations in the visual field. These rNFL defects have morphologies that are consistent with the rNFL pattern structure discovered in the retina. In its consistent position, the NFL had a striated appearance that extended from the optic disc and was thicker in the superior and inferior poles than in the temporal and nasal poles (Fig. 3).

Attributed to the reason that glaucoma tends to affect the inferior and superior fiber preferentially, localized loss is frequently found in these regions. Glaucomatous alterations can also be present as diffuse loss of the striations in the NFL.

Changes in discs have a wide range of characteristic patterns.

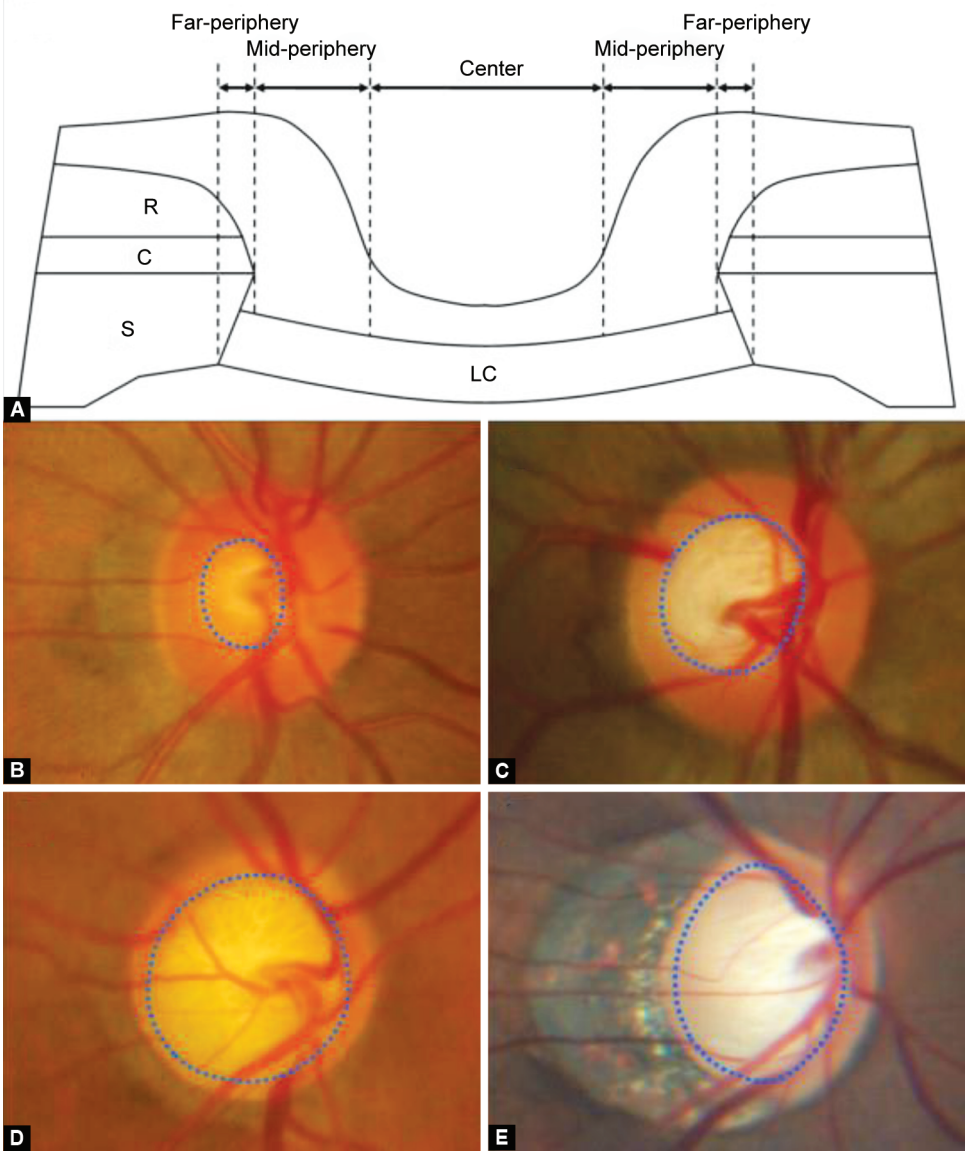
The neural rim thins as ganglion cells and their axons are destroyed. Typically, localized thinning in early glaucoma might result in focal notching or focal atrophy of the neural rim. This tends to happen in the inferotemporal area of the optic nerve due to preferential loss of the inferior nerve fibers. The optic cup often enlarges in a vertical or oblique form, and the temporal rim becomes implicated as a glaucomatous process advances, and this is followed by focal neuronal loss and atrophy in the superotemporal region to a lesser extent. The nasal quadrant is the final area to be affected.[5]

Various Glaucoma Forms

OAG

Open-angle glaucoma (OAG) may arise from any degree of 10P ocular nerve injury.

Either a sluggish or a fast pace of advancement is possible. When 10P increased, patients may begin to suffer abnormalities to the optic disc or visual field much later (Fig. 4).[6] Secondly, primary OAG.



Figs 3A to E: (A) Schematic diagram of a cross-section of the optic nerve head shows the microstructures that obscure the lamina cribrose from a clinical view; (B) The clinically visible portion of the lamina cribrose (B–E, blue dotted circles); (C) Generally increases with increasing optic disc size; (D) Glaucomatous damage; (E) Degree of optic disc tilting (C, choroid; LC, lamina cribrose; R, retina; S, sclera)

Pathophysiology

Glaucoma pathogens are not well known. The proportion of IOP has a consequence on the mortality of positively disposed cells. The interplay of aqueous humor secretion and drainage by the ciliary body through several channels, as well as the determination of IOP by the trabecular meshwork and uveoscleral outflow pathway. Patients with OAG experience greater resistance to aqueous outflow *via* the trabecular meshwork. IOP can produce mechanical stress and strain on the posterior structure of the eyes, particularly the lamina cribrose and surrounding tissue. Where the optic nerve fibers (retinal ganglion cell axons) exit the eye, the sclera is where the lamina is accomplished. The lamina is the weakest part of the pressurized eye's wall, and stress and strain brought on by IOP can cause it to compress, deform, and remodel. This mechanical axonal damage interferes with the retrograde delivery of vital trophic factors to retinal ganglion cells from their brainstem target. Individuals may develop glaucomatous optic neuropathy with IOP within the usual range and abnormally low cerebrospinal fluid levels.

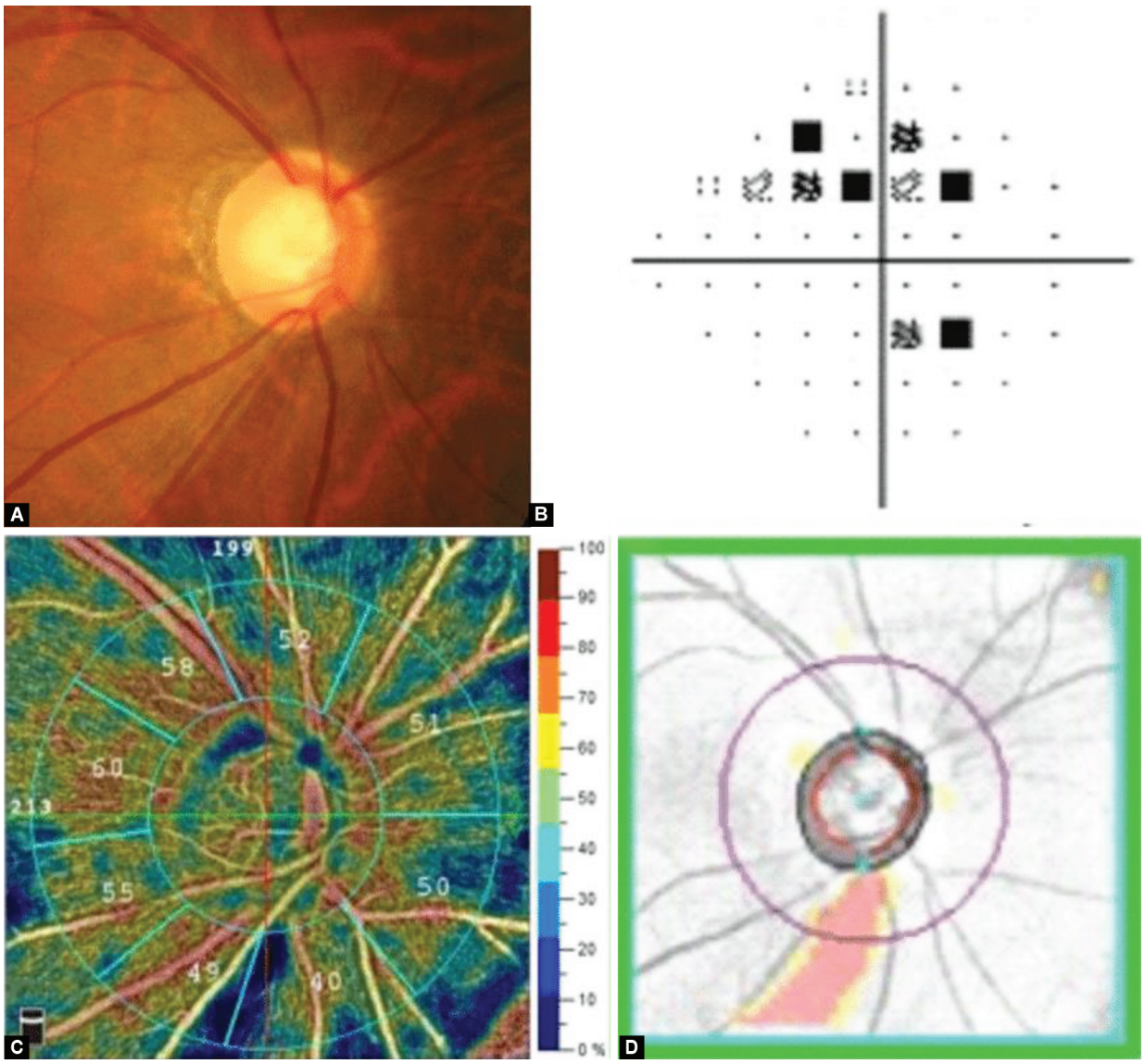
In these cases, fluid causing a significant pressure gradient across the lamina of the optic nerve may also be a contributing factor to glaucoma, along with compromised immunology, excitotoxicity, and oxidative stress.[7]

By changing their environment and making other retinal neurons and cells in the central visual pathway more susceptible to injury, primary neural pathogenic processes may cause secondary neurodegeneration.[7]

Closed-angle Glaucoma

Trabecular meshwork may result in closed-angle glaucoma because of a physical obstruction.

Then there is OAG, which might present more suddenly. IOP >40 mm Hg can result in optic nerve injury as well as irreversible nerve damage (>60 mm Hg) (Fig. 5).[4]



Figs 4A to D: Primary OAG

Primary Closed-angle Glaucoma

The main distinction between primary closed-angle glaucoma and primary OAG is the obstruction of the angle by apposition, which results in an anatomically closed angle (defined as having at least 27% of the angle occluded).

Iris, lens, and retrolenticular structural disorders are the main causes of primary closed-angle glaucoma. Pupillary block, the most frequent cause of angle closure, results from resistance to aqueous humor passage through the pupil from the posterior to anterior chambers. Angle-closure is caused by the aqueous humor behind the iris increasing its convexity. For a sizable amount of angle closure in Asian patients, non-pupil block mechanisms such as plateau-like arrangement must be at fault. Closed-angle glaucoma may also result from dynamic physiological processes that increase iris volume together with pupil dilation and choroidal effusion.[7]

Medicine-induced Glaucoma

Due to increased IOP brought on by various medicines, based on their mechanism of action and patients' predisposition, the medicine may worsen preexisting glaucoma or induce glaucoma.[4]

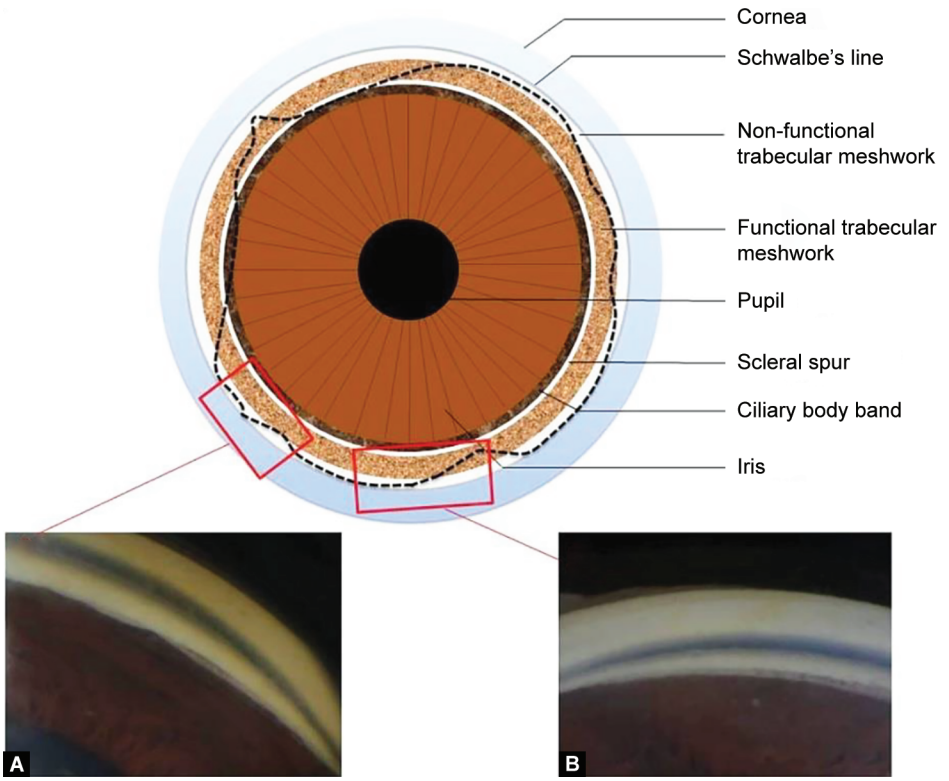
OAG Risk Factors

The main glaucoma risk factors are:

- Older in years.[21–23]
- Increased IOP.[21–23]
- Myopia is prominent.
- There must be a positive glaucoma family history.

A major risk factor is ethnicity. Evacuation of the optic disc is very challenging to assess in eyes with extreme myopia. Myopia-related optic disc enlargement may increase the risk of glaucoma and its associated cribrose. Another potential pathogenic factor has been identified for the lamina cribrose in people with severely myopic (long) eyes, which is induced by eye movements.[1]

The only modifiable factor for OAG that has been discovered so far is elevated translaminar pressure gradient or elevated IOP. According to research on randomized controlled ocular



Figs 5A and B: Primary closed-angle glaucoma

hypertension (OHT), lowering increased IOP (21 mm Hg) by 22.5% can reduce the probability of developing OAG over the course of 5 years from 9.5 to 44%.[1]

Factor Associated with Closed-angle Glaucoma

Asian ethnicity, female sex, and senior age are risk factors for angle closure. Eyes and angle closure seem to be shared biometric traits. Small eyes with a crowded anterior segment, shallow central anterior chamber depth, thicker and more anteriorly positioned lenses, and short axial length of the eye are the main risk factors for angle closure.

Another anatomical risk factor for angle closure with anterior segment optical coherence tomography is smaller anterior chamber width area and volume, broader circles with more iris curvature, and a greater lens vault.[7]

Methods

- Preliminary investigation.
- The advanced glaucoma intervention study (AGIS).
- Collaborative initial glaucoma treatment study (CIGTS).
- Esterman binocular scale.
- Hodapp-Parrish-Anderson Bascom Palmer glaucoma scrutility scale (GSS).
- Draft GSS development.
- Glaucoma scrutility scale.
- Six-point formula.

Preliminary Investigation

A literature review was done to look at previously created GSSs. The CIGTS and AGIS both used the Esterman binocular scale as their tagging system, which contained targeted GSSs. Due to the existence of various GSSs, the "seed system" was thought to have the potential to be modified. Ophthalmology GSS was also considered by American Academy, but as it could only be applied to three stages (no field loss, moderate field loss, and severe field loss), it was not put to use.[6]

AGIS

The Humphrey field analyzer report was used by AGIS to calculate a patient's visual field score using the central 24–2 total deviation. The superior, inferior, and nasal portions of the visual field comprised the study's three scoring sectors. Depending on the field location, it was regarded abnormal when the threshold value visual field deviated by five to nine decibels (dB) from the normal values as expressed in dB. Depressions in the superior field and inferior field must be greater than each other, and peripheral depressions must be greater than central depressions in order to be considered abnormal. Scores were assigned based on the dB depression measured in the various locations. A score of zero indicates no loss of the visual field. The maximum score was 20, though. A stage of field advancement was arbitrarily defined as beginning with a score increase of four.[6]

CIGTS

Similar to how the AGIS uses it. The scoring methodology was employed by the CIGTS staging system. In this study, glaucoma staging was likewise carried out using the Humphrey field analyzer based on the central 24–2 program, but AGIS was substituted in favor of categories of probability dB deviation plot. Marks ranging from 0 to 20. The patient was deemed to have advanced when the CIGTS score rose by three points or higher.[6]

Esterman Binocular Scale

Used by Mills and Drance, the Dicon A2000 perimeter's copper vision diagnostics automated binocular visual field exam yielded the Esterman visual function score (CooperVision, Fairport, New York). The scoring system was used for both the central and peripheral visual fields, and it was weighted according to the functional significance of the various visual field regions. The American Medical Association acknowledged the Esterman rating system as a capability in 1984. Patients with advanced glaucoma provided answers to a 15-item quality of life questionnaire to assess the difficulties they experienced with daily activities as a result of their visual field loss. The authors did not attempt to apply the Esterman test in order to standardize the stage of illness progression, though.[6]

HODAPP-ANDERSON-PARRISH Bascom Palmer GSS

Similar to the CIGTS and AGIS systems, stage assignments are possible using the system based on Humphrey visual field (HVF) testing, making a multicenter test an easily adaptable review of a chart in the past. Using this staging technique, patients with glaucoma at various stages of the illness based on the combination of the mean defect (MD) and the following and a score probability of pattern deviation (distortion from a normalized) plot score, the dB plot (stages II–IV), the visual field pattern, or, for either corrected pattern standard deviation (CPSD) in stage I or glaucoma, or the pattern standard deviation (PSD) results of the Hemifield test despite the system's usefulness based on the fact that it uses visual field loss as a sign during pilot testing, it was discovered that this GSS fails to capture the entire spectrum of glaucoma development stages, from individuals who had early, mild visual field abnormalities to individuals with terminal illnesses who are blind.

After reviewing the literature on current GSSs, a team of experts was gathered, comprising four glaucoma experts. Taking advantage of this, we have utilized expert groups to produce guidelines. There are 20–24 different medical research fields, including ophthalmology. 25. The experts consult the common medical literature. Combining initial algorithms with final draughts algorithms forms a standard, usually for the treatment of prudence. Among the GSSs now in use, the Bascom Palmer expert group determined that GSS was the most suited because it made systematic severity stage assignment possible. Mostly based on the HVF parameters in a way that is comparatively distilled. The CIGTS and AGIS scoring systems call for more intricate calculations and are considered more prone to scoring errors as a result. Furthermore, none of those rating methods has been applied to any appreciable extent in clinical practice. When compared, clinically, the Bascom Palmer GSS has been utilized for routine medical attention. Despite the lack of any comparisons compared to other systems of the Bascom Palmer GSS, the AGIS and CIGTS were discovered by Katz and collaborators to be techniques of grading that varied greatly in their evaluation of visual field development. Range of glaucoma severity during development, from early and little visual field degeneration leading to blindness from end-stage illness. Results of a visual acuity test were added for deciding how to categorize the end-stage. Then, secondary changes were made to guarantee that the visual field parameter selections and, within each level, the corresponding threshold values were in line with the regular progression patterns of the visual field. Visual acuity, in particular, was discovered to be a crucial addition to the end-stage classification assignment to make things easier—GSS use, the creation of staging tables, and were tailored for specific HVF kinds.

Validation of the GSS

At Duke University in Durham, Durham, North Carolina, the GSS was pretested by reviewing the charts of 30 patients. All HVF s in the 30 charts were retrospectively given stage using the GSS approach. The GSS parameters and decision rules were appropriately modified to address any ambiguities that emerged in the stage assignment during the pretest. The GSS was then completed and further assessed for its capacity to independently and unmistakably stage a fresh batch of 68 glaucoma charts at six American centers. The worst-eye visual field score was used to stage the patients. The tool was able to categorize every patient with glaucoma, from OHT through end-stage illness.

Six Point Method

Stage 0 (normal visual field), stage I (early), stage II (moderate), stage III (advanced), stage IV (severe), and stage V make up the six phases of the most recent GSS (end-stage). Staging criteria are mostly based on the HVF, with MD serving as the key measure. There are three subdomains for modification based on CPSD/PSD and Hemifield test results for stages II through IV. Based on inadequate visual acuity and a severe loss of field in stage V classifications that prevent performing visual field-testing.[8]

Diagnostic Evaluation

Symptoms of Glaucoma

With pain radiating from the eye, visual impairment, conjunctival hyperemia, and sometimes nausea and vomiting with a tense, rock-hard globe, acute angle closure manifests itself. An emergency that demands immediate treatment to prevent severe ocular damage and blindness is termed "ophthalmological." In an advanced stage, the open angle generally has no symptoms, and most of them are unaware that they have the condition.

One-third of people in an advanced or late stage in at least one eye at the time of diagnosis already have this condition. At the time of presentations at the, Gramer et al. reported that 10–20% of patients were already unable to derive a vehicle at the clinic because of binocular visual field defects.[9]

Early Detection of Glaucoma

Only after the condition has advanced to that point does it become sympathomimetic. The German Ophthalmological Association advises routine screening exams for early detection beginning at age 40. Any positive discovery must be followed by additional testing because the test's low sensitivity and specificity (e1 and e2) and the disorder's low prevalence result in a significant rate of false positives (>65%, and much greater in younger individuals). Regular examination is especially crucial for disorders with higher incidence and prevalence in risk groups so they can be identified and treated early in their progression. There haven't been any random, controlled trials on this subject yet. There are no population-wide periodic for glaucoma screenings in Germany or other European nations, nor are glaucoma screening exams reimbursed by the statutory health insurance carriers.[15–17]

An overview of treatments that lower the IOP in OAG[1] (Table 1):

Table 1: Treatment for early detection of glaucoma

Treatment category	*Treatment type*		
Drugs	Local application	Prostaglandin analogues β blockers A β2-adrenergic agonist Carbonic anhydrase inhibitors Miotic agents	Improved uveoscleral and trabecular outflow. Reduced aqueous humor production. Reduced aqueous humor production and increased uveoscleral outflow. Reduced aqueous humor production. Widening of the chamber angle
	Systemic intake	Carbonic anhydrase inhibitors Osmotically active substances (mannitol IV)	Reduced aqueous humor production Osmotic removal of water from the globe
Operative interventions	Laser therapy	Laser trabeculoplasty	Increased outflow of aqueous humor *via* the canal of Schlemm
		Cyclophotocoagulation	Reduced aqueous humor production
	Surgery	Cyclocryocoagulation	Reduced aqueous humor production. A minimally
		Invasive procedure	For example, implantation of a stent in the canal of Schlemm to lessen the outflow resistance of the trabecular meshwork
		Filtering procedure	For example, deep sclerotomy: widening of the outflow pathways without incising the eye
		Filtering procedure	For example, trabeculectomy: creation of an accessory pathway for the aqueous humor to flow out of the eye under the conjunctiva

Current Status of Glaucoma Practice with Graphical Representation

Therapy for Glaucoma

The only method of treatment that has been proven to be effective, producing positive results, and widely acknowledged for the prevention of glaucoma is the reduction of IOP. Regular application of eye drops helps lower the IOP in people with OAG. The main objective is to reach a target pressure that is individually determined and below which it is expected that glaucoma will not advance and where this lack of progression can be observed and documented.

The target pressure is established for each patient based on the degree of glaucomatous damage already present, the rate of structural and functional progression, the IOP at the time, and any additional risk factors that may exist.[10–14]

Laser Therapy

Supplemental laser therapy may be investigated if local treatment is unsuccessful in lowering IOP or fails to achieve the desired IOP results in a slight intraocular reduction. Pressure laser therapy typically produces increased aqueous humor following laser trabeculoplasty or decreased aqueous humor following cyclophotocoagulation (Fig. 6). The latter lowers the IOP by at least 20 in 47 % of the treated eyes, even after taking into account potential side effects. Although their efficacy hasn't been fully tested, both are applicable for use with the micropulse laser technique. Supplemental laser therapy may be investigated if local treatment is unsuccessful in lowering IOP or fails to achieve the desired IOP results in a slight intraocular reduction. Pressure laser therapy typically produces increased aqueous humor following laser trabeculoplasty or decreased aqueous humor following cyclophotocoagulation. The latter lowers the IOP by at least 20 in 47% of the treated eyes, even after taking into account potential side effects. Although their efficacy hasn't been fully tested, both are applicable for use with the micropulse laser technique.[15,16]

Surgical Direction

Similar to primary OAG, surgical intervention is necessary when medication or laser therapy fails to adequately lower IOP or when the optic nerve or visual field impairment is progressing. If the pressure control is still too high after receiving laser and medical treatment, trabeculectomy—either by itself or in conjunction with lens extraction—should be taken into consideration, especially in more severe cases of OAG. When lens-related mechanisms are predominant, lens extraction is also done, especially when a large cataract is obstructing vision.[8,9,17]

Result and Conclusion

The purpose of the GSS decision rules was to determine which criteria ought to be applied when categorizing patients from stages 0 through V. Stages 0 through IV were assigned primarily using this measure, with three extra standards for stage changes based on levels 0 on the CPSD/PSD and Hemifield test dB plot for stages II through IV. The decision-making process included additional criteria rules that govern both the staging table and the GSS stage definitions. The definition of these choice guidelines is that if a patient passes the MD requirements for a certain stage (stages I through IV), but falls short of one of if the patient meets the additional requirements for that level, then the patient is classified at the stage before; if a patient meets the criteria and the MD requirements for a specific stage, meets any stage (stages I–IV), and at least one of the extra requirements for a previous stage, in addition to one or more of the requirements for the next stage, then the patient classification in the initial stage is based on MD standards.

Glaucoma is a chronic progressive optic neuropathy that can lead to permanent loss of peripheral or central vision. IOP is the only known modifiable risk factor. Other important risk factors include older age, non-white race, and a family history of glaucoma. Most of the time, glaucoma is the reason for irreversible blindness worldwide. Progression often stops when IOP has fallen by 30–50%.

African heritage and advanced age are risk factors. It is a notable public health issue because of its high fatality rate and ubiquity. A total of 3.5% of the people in the population are 40 years of age or older. Up to 40% of the retinal nerve fibers may be damaged by glaucoma. This causes gradual and asymmetric alterations in the optic cup, along with a corresponding decrease in the visual field.

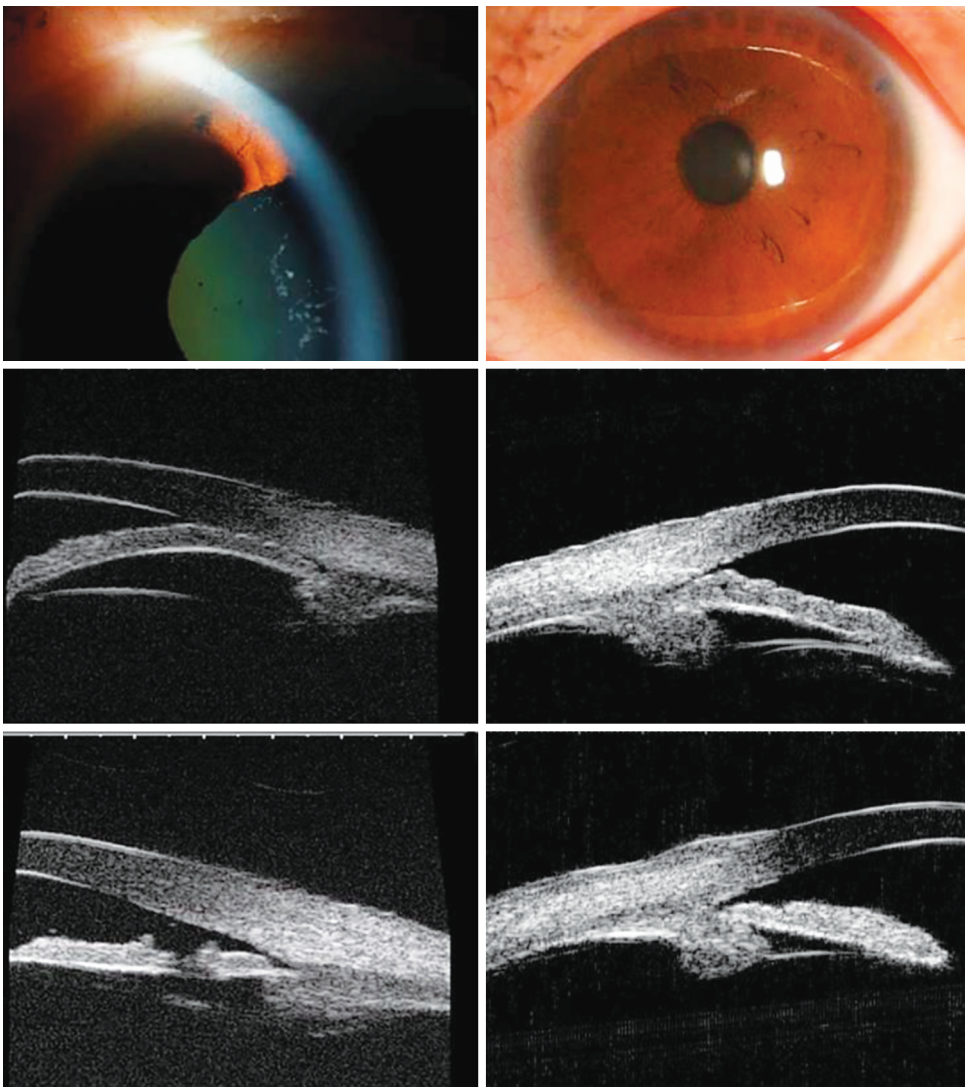


Fig. 6: Laser therapy for glaucoma

The neural rim thins as ganglion cells and their axons are destroyed. Localized thinning or focal atrophy of the neural rim leads to focal neuronal loss and atrophy. IOP can produce mechanical stress and strain on the posterior structure of the eyes, particularly the lamina cribrose. The proportion of IOP has a consequence on the mortality of positively disposed cells. Patients with OAG experience greater resistance to aqueous outflow *via* the trabecular meshwork.

Pupil block is the most frequent cause of angle closure in Asian patients. Increased IOP or translaminar pressure gradient is the only modifiable factor for OAG that has been discovered so far. The risk factors are older in years (21–23) female sex, and senior age are risk factors for angle closure. Ophthalmology GSS was also considered by the American Academy of optics, but as it could only be applied to three stages (no field loss, moderate field loss, and severe field loss), it was not used.

The American Medical Association acknowledged the Esterman rating system as a capability in 1984. During pilot testing, it was discovered that this GSS fails to capture the entire spectrum of glaucoma development stages. Similar to the CIGTS and AGIS systems, stage assignments are possible using the system based on HVF testing, making a multicenter test easily adaptable. The Bascom Palmer GSS is based on the HVF parameters in a way that is comparatively distilled. The CIGTS and AGIS scoring systems, which call for more intricate calculations, were considered to be more prone to scoring errors as a result.

None of those rating methods has been applied to any appreciable extent in clinical practice. Staging criteria are mostly based on the HVF, with MD as the key measure. German Ophthalmological Association advises routine screening exams for early detection beginning at age 40. Any positive discovery must be followed by additional testing because the test's low sensitivity and specificity and the disorder's low prevalence result in a significant rate of false positives.

There are no population-wide periodic glaucoma screenings in Germany or other European nations. Overview of treatments that lower the IOP in OAG. The target pressure is established for each patient based on the degree of damage already present, the rate of progression, and any additional risk factors that may exist. After laser trabeculoplasty, pressure laser therapy typically works in at least 20% of the treated eyes. In a modest intraocular lowering.

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