

# PRIMARY OPEN ANGLE GLAUCOMA



**Eye Learn**

All about the Eye

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## Primary open angle glaucoma

1. Describe modern methods for the diagnosis and management of primary open angle glaucoma. J2009
2. Disc changes in open angle glaucoma. D2009
3. What are the minimum diagnostic criteria for Primary Open Angle Glaucoma (POAG)? Give severity classification of POAG with concept of target pressure? (3+7) D2012, J2013
4. Define open angle glaucoma suspect. Discuss the management options and follow up. What are the Global Indices in automated perimetry? 2+4+4 (D2013)
5. Pathology of GOA. (2004)
6. Risk Factors for Open Angle Glaucoma. (2007)
7. Target pressure in management of glaucoma. (2001)
8. POAG factors that help decide target intra ocular changes. (2005)
9. Medical vs surgical management of open angle glaucoma. (2002)

**Definition-** Primary open-angle glaucoma (POAG) is typically a chronic, slowly progressive optic neuropathy with characteristic patterns of optic nerve damage and visual field loss.

Multifactorial	
Main Factors	Other factors
<ol style="list-style-type: none"> <li>1. Elevated intraocular pressure (IOP),</li> <li>2. Advanced age,</li> <li>3. Race,</li> <li>4. Thin central cornea,</li> <li>5. A positive family history of glaucoma</li> </ol>	<ol style="list-style-type: none"> <li>6. Corneal hysteresis,</li> <li>7. Low ocular perfusion pressure,</li> <li>8. Low cerebrospinal fluid pressure,</li> <li>9. Abnormalities of axonal or ganglion cell metabolism,</li> <li>10. Disorders of the extracellular matrix of the lamina cribrosa.</li> </ol>

### Clinical Features

- POAG is typically insidious in onset, slowly progressive, and painless.
- It is usually bilateral but can be quite asymmetric.
- Patients may seem relatively asymptomatic until the later stages of the disease, when central vision is affected.
- POAG is diagnosed based on findings from the assessment of the optic nerve and nerve fiber layer and the results of visual field testing.

### Gonioscopic findings

- To establish a diagnosis of POAG, the clinician must verify that the anterior chamber angle is open.
- The angles can be narrow, but there can be no peripheral anterior synechiae (unless caused by prior laser treatment or surgery),
- No apposition between the iris and the trabecular meshwork, and no developmental abnormalities of the angle.
- Moderate pigmentation of the meshwork is often present in proportion to the patient's age and race.
- Heavy pigmentation is suggestive of other disorders, including pigmentary glaucoma, exfoliative syndrome, trauma, and uveitis
- Gonioscopy should be performed on all patients evaluated for glaucoma and should be repeated periodically in patients with established OAG to monitor for

<ol style="list-style-type: none"> <li>1. As AC becomes shallow with age</li> <li>2. Progressive angle closure caused by lens-induced changes,</li> <li>3. Particularly in patients with hyperopia.</li> </ol>	<ol style="list-style-type: none"> <li>4. When strong miotics are prescribed;</li> <li>5. After argon laser trabeculoplasty or laser peripheral iridotomy;</li> <li>6. Or when IOP increases</li> </ol>
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### Optic nerve head appearance and visual fields

- Although elevated IOP is an important risk factor for OAG, diagnosis of this disease is based primarily on the appearance of the optic nerve head, or optic disc, and on the results of visual field testing.
- Visual field loss should correlate with the appearance of the optic nerve; significant discrepancies between the pattern of visual field loss and optic nerve appearance warrant additional investigation

### Risk Factors

#### 1. Intraocular pressure

- It is known that IOP in the general population is not represented by a Gaussian distribution but rather is skewed toward higher pressures
- Thus, IOPs of 22 mm Hg and above may not necessarily be abnormal from a statistical standpoint.
- Several studies indicate that as many as 30%–50% of individuals in the general population with glaucomatous optic neuropathy and/or visual field loss have initial IOP measurements below 22 mm Hg.
- Elevations of IOP may occur only intermittently in some glaucomatous eyes, with as many as one-third of these elevated measurements due to normal circadian fluctuation.
- In patients with glaucoma, IOP may vary considerably—by 10 mm Hg or more—over a 24-hour period. In contrast, most patients without glaucoma manifest a diurnal range of 2–6 mm Hg.
- Also, in most healthy subjects and glaucoma patients, IOP has a distinct circadian rhythm, with peak pressures often occurring during sleep, particularly in the early-morning hours
- About two-thirds of patients reach peak IOPs during non- office hours.

**Table 4-1 Potential Reasons for Undetected High-Tension Glaucoma**

Primary open-angle glaucoma with diurnal IOP fluctuation
Intermittent IOP elevation
Nonacute angle-closure glaucoma
Glaucomatocyclitic crisis
Secondary glaucoma (eg, pigmentary, pseudoexfoliation, uveitic)
Normalized IOP in an eye with previously elevated IOP (eg, corticosteroid-induced, uveitic, pigmentary, hyphema)
Use of medications that may lower IOP (eg, systemic $\beta$ -blocker)
Inaccurate IOP measurement (due to thin central cornea, reduced scleral rigidity, uncalibrated device, poor examiner technique)

#### 2. CCT

- A thinner cornea is an important risk factor for disease progression in individuals with POAG (with higher baseline IOPs) and for the development of glaucoma in individuals with ocular hypertension
- Thicker corneas resist the deformation inherent in most methods of tonometry, resulting in an overestimation of IOP. In contrast, tonometry in eyes with thin corneas underestimates the IOP.
- People with thin corneas have less support tissue in the optic nerve making it more liable to pressure-induced and/or vascular damage.

- The average CCT in adult eyes determined by ultrasonic pachymetry, ranges between 540 and 550  $\mu\text{m}$  and varies with race and ethnicity.
- thin corneas may be a biomarker for disease susceptibility.( African thin cornea, high VCDR)

### 3. Older age is an independent risk factor for POAG

q	Found that the prevalence of glaucoma increases with age, particularly among individuals of African descent, whose prevalence > 11% in >80 years.
Collaborative Initial Glaucoma Treatment Study	visual field defects were 7 times more likely to progress in patients 60 years or older than in those younger than 40 years
The Ocular Hypertension Treatment Study	found an increased risk of progression to OAG with age (per decade): 43% in the univariate analysis and 22% in the multivariate analysis

- The effect of age on the prevalence of POAG holds true even after compensating for the relationship between increasing age and increasing IOP.

### 4. Race

- The prevalence of POAG in the USA is 3–4 times greater in individuals of African descent or Hispanic ethnicity than in primarily European-derived populations.
- Blindness from glaucoma is at least 4 times more common in blacks than in whites.
- In addition, glaucoma is more likely to be diagnosed in black patients at a younger age and at a more advanced stage than it is in white patients.
- It is estimated that the incidence and the prevalence of blindness from glaucoma are 8–10 times higher in black patients than in white patients in the United States.
- Some have proposed that optic nerve ischemia from sickle cell anemia contributes to the high prevalence of POAG in blacks. However, this theory was not supported by one study, which found that only 2 of 40 black patients requiring filtering surgery had a positive test for sickle cell trait.
- In the OHTS, glaucoma was 59% more likely to develop in black patients with OHT than in white patients with ocular hypertension, in a univariate analysis.
- This relationship was not present after controlling for corneal thickness and baseline vertical cup–disc ratio in a multivariate analysis

### 5. Gender

- Conflicting information exists about the effect of gender on the prevalence of POAG.
- In several studies, males had a higher prevalence of glaucoma.
- In the Barbados study, POAG was associated with older men, high IOP, positive family history, lean body mass, and low blood pressure to IOP ratio.

### 6. Positive family history

- In the Baltimore Eye Survey, the relative risk of POAG increased approximately 3.7-fold for individuals who had a sibling with POAG.

### 7. Heredity

- AD, AR , Sex linked recessive
- The Rotterdam study found that relatives of patients with POAG were 10 times more likely to have or develop glaucoma than relatives of those without glaucoma.



- A monozygotic and dizygotic twin study estimated the heritability to be 13%

GLC1A located on chromosome 1 in the q23–25 region	Associated with juvenile-onset open-angle glaucoma 3–4% cases of POAG in adults
GLC1B located on chromosome 2	Associated with adult-onset open-angle glaucoma associated with POAG in adults seem to be related to an early-onset type.
Mutation on chromosome 15	17% of early, but not childhood-onset, glaucoma.
Polymorphisms in the methylenetetrahydrofolate reductase gene	in POAG patients
Novel gene abnormality on chromosome 3	Large Tasmanian family with early-onset open-angle glaucoma, one-third of whom have mutations in the myocilin gene and others with glaucoma show mutations on chromosome 3
Endothelial nitric oxide synthase gene	open-angle glaucoma accompanied by migraine.

## 8. Myopia

Beaver Dam Eye Study	Myopia ( $\leq -1$ D spherical equivalent) was significantly associated with a diagnosis of glaucoma.
Rotterdam follow-up study	high myopia ( $\leq -4$ D spherical equivalent) was associated with an increased risk (2.31 times higher) of development of glaucoma
OHTS	did not find an association between myopia and the incidence of glaucoma

The concurrence of POAG and myopia may complicate diagnosis and management in several ways.

1. Evaluation of the optic nerve head is particularly challenging in highly myopic eyes that have tilted discs broad, shallow optic cups with less distinct margins or posterior staphylomas.
2. Myopic refractive error may cause magnification of the optic nerve, further complicating accurate optic nerve assessment.
3. Baring of the blind spot or other refractive scotomata on visual field testing
4. Myopia-related retinal degeneration or anomalies can cause visual field abnormalities that are difficult to distinguish from those caused by glaucoma.
5. Patients who are highly myopic may have difficulty performing accurately on visual field tests, making interpretation of visual field abnormalities more challenging.
6. Low ocular rigidity, which makes Schiötz tonometer readings inaccurate
7. Thin corneas and sclera which may give falsely low readings on Goldmann tonometry

## Associated Disorders

1. **Diabetes mellitus** -There is controversy as to whether diabetes mellitus is a risk factor for glaucoma.

The Beaver Dam Eye Study, The Blue Mountains Eye Study, The Los Angeles Latino Eye Study	found an association between diabetes and OAG
Framingham Study, The Baltimore Eye Survey, The Barbados Eye Study Revised analysis of the Rotterdam Study	did not find a significant association
OHTS	Diabetes was associated with a reduced risk of developing glaucoma. Cohort of diabetic patients was skewed, because the presence of retinopathy was an exclusion criterion for this study

- The explanation for this relationship remains obscure, but some investigators have proposed that diabetes affects the small blood vessels supplying the optic nerve, thereby rendering it more susceptible to glaucomatous damage



## 2. Hypertension

Baltimore Eye Survey	<ol style="list-style-type: none"><li>1. HTN was associated with a lower risk of glaucoma in younger (&lt;65 years) subjects and a higher risk of glaucoma in older subjects</li><li>2. The hypothesis is that younger individuals with high BP may have better perfusion of the optic nerve</li><li>3. As these patients age, their chronic hypertension may have adverse effects on the microcirculation of the optic nerve and increase its susceptibility to glaucomatous optic neuropathy</li></ol>
Barbados Eye Study	Relative risk of developing glaucoma among subjects with systemic hypertension was less than 1.0 in all age groups, including those aged 70 years and older.

## 3. Lower ocular perfusion pressure

- (OPP; often defined as diastolic blood pressure + 1/3 systolic blood pressure – IOP)
- Definition of OPP oversimplifies actual ocular blood flow, several factors, including autoregulatory mechanisms in central nervous system perfusion, make the association between OPP and glaucoma intriguing.
- The overtreatment of systemic hypertension may contribute to glaucoma progression in some cases( NTG)
- Blood pressure to be measured before starting a  $\beta$ -adrenergic blocking medication.

## 4. Retinal vein occlusion

- In CRVO elevated IOP due to neovascularization of the angle may progress to glaucoma.
- Also, susceptible individuals with elevated IOP (OHT) are at risk of developing CRVO.
- Consideration should be given to treating elevated IOP in patients with a history of CRVO in order to reduce the risk of CRVO ( HemiRVO similar pathophysiology) in the fellow eye.

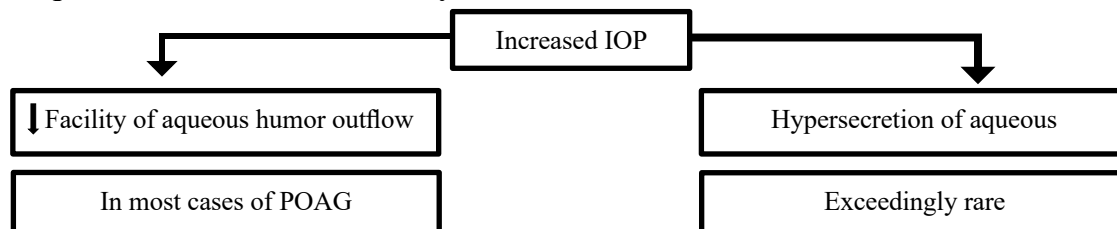
## 5. Other associated conditions

Thyroid disorders	In one study, OAG was associated with chronic thyroid orbitopathy. A more recent study confirmed the association of Graves' disease with POAG, NTG,(IOP can be raised with restrictive muscle conditions). Association was found between males with OAG and hypothyroidism.
Migraine headaches	The Blue Mountains Eye Study suggests an association between OAG and migraine.
Hypercholesterolemia	long-term oral statin or other anticholesterol use is associated with a lower risk of open-angle glaucoma
Sleep apnea	respiratory disturbance leading to transient nocturnal episodes of hypoxia, which may increase the propensity of the optic nerve to damage
Low CSF Pressure	Corneal hysteresis
Raynaud phenomenon	Rotterdam study produced an unexpected and as yet unexplained association between <b>early menopause and glaucoma</b>

## Pathophysiology of POAG

- A detailed discussion of POAG must address two fundamental issues:
  - the mechanism(s) of IOP elevation,
  - the mechanism(s) of progressive optic nerve cupping and atrophy

### Diminished aqueous humor outflow facility



- In enucleated normal human eyes, Grant demonstrated that incising the entire trabecular meshwork reduced the resistance to outflow by 75%.
- ↓resistance to outflow seen in glaucoma must also lie between the anterior chamber and the lumen of Schlemm's canal.

Juxtacanalicular tissue	TM prolapse into SC	Intrasccleral collector channels
It may be the main site of resistance to outflow, It has greatest concentration of mucopolysaccharides It has greatest phagocytic activity	outflow facility↓ because the TM prolapses into SC, thus occluding the lumen and preventing circumferential flow of aqueous humor to the collector channels.	↓outflow facility in glaucoma has also been ascribed to an obstruction of the intrasccleral collector channels by accumulation of glycosaminoglycans in adjacent sclera
confirmed by careful microcannulation and pressure measurements at various locations within the TM Resistance was 7–14mm internal to the inner wall of Schlemm's canal.	SC only collapses at very high levels of IOP. No evidence shows that the canal is occluded when IOP 25–35mmHg (mc in POAG)	Accounts for approximately 50% of POAG cases (Krasnov) This theory was partially refuted by experiments that demonstrated that unroofing SC did not ↓resistance to outflow in glaucomatous eyes until the canal was entered; that is, no scleral blockage was noted.

- If the hypothesis is accepted that the trabecular meshwork or the endothelium of Schlemm's canal is the site of the increased resistance to outflow in POAG, the question of what process interferes with normal aqueous elimination must be asked.
- Several theories have been proposed to explain this phenomenon, including those that follow:

#### 1. An obstruction of the trabecular meshwork by foreign material.

- ✓ Several investigators have noted the accumulation of foreign material in the trabecular meshwork and juxtacanalicular tissue, including pigment, red blood cells, glycosaminoglycans, amorphous material, extracellular lysosomes, plaque-like material, and protein.
- ✓ Lütjen-Drecol and Rohen have postulated that the electron-dense material consists of collagen and elastin and that these materials are responsible for the increased resistance to aqueous outflow.



- ✓ It is also possible that a normal constituent that is catabolized insufficiently or synthesized excessively obstructs the meshwork.

## 2. A loss of trabecular endothelial cells.

- ✓ Glaucomatous eyes have fewer endothelial cells than normal eyes, although the rate of decline is similar.
- ✓ This suggests a premature aging process in glaucomatous eyes.
- ✓ A loss of endothelial cells would interfere with various important trabecular functions, including phagocytosis and synthesis and degradation of macromolecules.
- ✓ The lack of a complete endothelial covering could allow the trabecular beams to fuse.

## 3. A reduction in pore density and size in the inner wall endothelium of Schlemm's canal.

- ✓ The endothelium lining the inner wall of Schlemm's canal accounts for 10–20% of the total resistance.
- ✓ Ultramicroscopic pores can be found in the endothelium of the inner wall of Schlemm's canal, and they seem to be reduced in both size and density in open-angle glaucoma.

## 4. A loss of giant vacuoles in the inner wall endothelium of Schlemm's canal.

- ✓ They may play a crucial role in moving fluid from the meshwork into the lumen of Schlemm's canal.
- ✓ A reduction in the number and size of these microstructures is seen in glaucoma.
- ✓ Alvarado and Murphy found a reduction in the area of 'cul-de-sacs' in the juxtacanalicular tissue in glaucomatous eyes; this reduction could account for the increased resistance to outflow.

## 5. A loss of normal phagocytic activity.

- ✓ Phagocytosis occurs in the trabecular meshwork continuously and represents the self-cleaning filter of the meshwork.
- ✓ It has been postulated that the trabecular endothelial cells lose their normal phagocytic activity or are overwhelmed by foreign material, which leads to cell death or migration from the beams.

## 6. Disturbance of neurologic feedback mechanisms.

- ✓ Nerves, whose function is unknown, have been found in the trabecular meshwork.
- ✓ Nerve endings, some of which could be mechanoreceptors, located in the scleral spur of humans.
- ✓ It has been speculated that these nerves could function to slow down aqueous formation or speed outflow when IOP is elevated.
- ✓ Theoretically, some interference with this feedback mechanism could lead to unchecked elevation of IOP.

**Histopathologic study** of the conventional aqueous drainage system from patients with POAG reveals a number of abnormalities, including those that follow

1. Alterations in the trabecular beams, including fragmentation of collagen, increased curly and long-spacing collagen, and coiling of fiber bundles	5. Decreased number of trabecular endothelial cells
2. Thickened basement membranes.	6. Reduced actin filaments
3. Narrowed intertrabecular spaces	7. Accumulation of foreign material
4. Fused trabecular beams	8. Decreased number of giant vacuoles
	9. Narrowing of collector channels
	10. Closure of Schlemm's canal
	11. Thickened scleral spur.



- Evidence favors the trabecular meshwork or the endothelium of Schlemm's canal as the site of the increased resistance.
- If we accept this hypothesis, we must still ask why outflow facility is reduced in POAG.
- Various investigators have linked **the increased resistance to outflow** with altered corticosteroid metabolism, dysfunctional adrenergic control, abnormal immunologic processes, and oxidative damage.

### 1. Altered corticosteroid metabolism

- ✓ POAG patients had increased sensitivity to respond to topical steroid having similar pattern of inheritance as POAG.
- ✓ Various investigators noted patients with POAG had

1. Increased plasma levels of cortisol; 2. Increased suppression of plasma cortisol with different doses of exogenous dexamethasone; 3. Disturbed pituitary adrenal axis function,	4. Continued suppression of plasma cortisol by dexamethasone despite concomitant administration of diphenylhydantoin (phenytoin); 5. Increased inhibition of mitogen-stimulated lymphocyte transformation by glucocorticoids.
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- ✓ Researchers postulated that endogenous corticosteroids affected trabecular function by

✓ Altering prostaglandin metabolism, ✓ Glycosaminoglycan catabolism, ✓ Release of lysosomal enzymes,	✓ Synthesis of cyclic adenosine monophosphate ✓ Or inhibition of phagocytosis
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### 2. Dysfunctional adrenergic control

- ✓ In analogous fashion to the corticosteroid theory, others have proposed that the diminished outflow facility in patients with POAG could be explained by an increased sensitivity to adrenergic agonists. Various reports indicated that patients with POAG had
  - A greater IOP reduction after the administration of topical adrenaline (epinephrine);
  - A greater response to adrenaline (epinephrine) or theophylline in inhibiting mitogen-stimulated lymphocyte transformation,
  - More frequent premature ventricular contractions after topical administration of adrenaline (epinephrine).
- ✓ Furthermore, ocular hypertensive subjects who demonstrated a fall in IOP greater than 5mmHg after topical adrenaline (epinephrine) administration had a higher rate of developing visual field loss.
- ✓ However, additional studies have generally failed to confirm an increased sensitivity to adrenergic agonists in patients with POAG.

### 3. Abnormal immunologic processes

- ✓ Other investigators have explained the diminished aqueous humor outflow in POAG by abnormal immune responses.
- ✓ Increased levels of gamma-globulin and plasma cells have been detected in the TM of POAG patients.
- ✓ Glaucoma patients were noted to have a high prevalence of antinuclear antibodies.
- ✓ Endothelin-like immunoreactivity has been noted to be increased in the aqueous of glaucoma patients, suggesting a role for this molecule in IOP regulation.
- ✓ Antibodies to heat shock protein, an indicator of cell stress, have been noted to be increased in the serum of glaucoma patients.
- ✓ An association between POAG and certain HLA was reported and then refuted by multiple studies.

- ✓ Evidence for immunologic factors in open-angle glaucoma, especially in the retinal ganglion cell layer, have led some to propose vaccination as a potential neuroprotecting treatment in glaucoma

#### 4. Oxidative damage

- ✓ Interest has developed in the question of whether the trabecular meshwork could be damaged by oxidative insult.
- ✓ The meshwork contains glutathione, which may protect the endothelial cells from the effects of hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>) and other oxidants.
- ✓ This interesting hypothesis is still the subject of active research.

#### 5. Other toxic influences

- ✓ Lütjen-Drecoll has postulated that transforming growth factor (TGF) beta2 may be involved in the pathogenesis of open-angle glaucoma.
- ✓ Dan and co-workers have shown that there is a threefold increase in the levels of plasminogen activator inhibitor in the aqueous humor of glaucoma patients compared to cataract patients without glaucoma.
- ✓ These findings suggest that this protein may play some role in the pathogenesis of increased IOP.

#### Conclusion

- ✓ In summary, the cause of the trabecular dysfunction in POAG is unclear at present. To date, no single theory explains the pathophysiology.

#### Optic nerve cupping and atrophy

- Cupping consists of backward bowing of the lamina cribrosa, elongation of the laminar beams, and loss of the ganglion cell axons in the rim of neural tissue.
- Histologic studies indicate that optic nerve cupping includes the loss of all three elements of the disc – axons, blood vessels, and glial cells.
- A recent study of the submicroscopic histopathology and immunohistochemistry of the optic nerve showed fibrosis, arteriosclerotic changes and loss of capillaries in glaucomatous optic nerves compared to non-glaucomatous ones
- The lamina is a relatively rigid structure that surrounds the densely packed axons. It is the tissue that divides the higher IOP space from the lower subarachnoid pressure space. Early in glaucoma, the lamina is compressed. In the later stages of the disease, the laminar sheets become fused, and the entire lamina bows backward.
- It is commonly accepted that increased IOP either directly or indirectly causes optic nerve cupping. The evidence for this can be summarized as follows:
  1. Most patients with POAG have increased IOP, which generally predates by years the development of cupping and visual field loss.
  2. Elevated IOP is a major risk factor for the development of POAG in glaucoma suspects.
  3. Elevated IOP is the only known common element to a wide variety of secondary glaucomas.
  4. Even in normal-pressure glaucoma, in which IOPs do not exceed the statistically 'normal' range, the degree of cupping is related to the level of IOP.
  5. In all animal models of glaucoma, elevated IOP precedes optic nerve damage and visual loss.<sup>248</sup>

6. Mechanical changes in the topography of the optic nerve and in the lamina cribrosa are seen early in experimental glaucoma with elevated IOP in monkeys and are not seen in other forms of optic nerve damage.

## Investigation

- Diurnal IOP measurements may be useful in some situations, including diagnosing POAG, explaining progressive damage despite apparent good pressure control, evaluating the efficacy of therapy, and distinguishing normal-tension glaucoma from POAG.
- When pressure is higher in one eye, that eye usually has a larger cup and a more damaged visual field than the fellow eye.
- Marked differences in IOPs between the two eyes should raise suspicion of Exfoliative syndrome or another form of secondary glaucoma.
- An afferent pupillary defect can be seen in patients with asymmetric or unilateral glaucoma. This finding, which is also referred to as Marcus Gunn's sign, is elicited by the swinging flashlight test. It has even been noted in patients with asymmetric cupping and normal kinetic visual fields

## Differential Diagnosis

<p><b>I Secondary/Developmental glaucoma</b> Exfoliative syndrome, Pigmentary dispersion, Trauma, Anterior segment inflammation, Subacute or chronic angle closure, Elevated episcleral venous pressure, Axenfeld's and Rieger's syndromes, Corticosteroid administration.</p> <p><b>II Non- glaucomatous ON</b> AION NAAION TON Nutritional/toxic ON Compressive optic nerve lesions.</p> <p><b>III ONH anaomalies</b> ONH pits ONH colobomas</p>	<p><b>IV Arcuate scotoma</b> <u>Chorioretinal lesions</u> Myopic degeneration Atypical retinitis pigmentosa Photoreceptor degeneration Branch vein occlusion Branch artery occlusion Juxtapapillary chorioretinitis</p> <p><u>Optic disc lesions</u> Drusen Pits Colobomas Papillitis Chronic papilledema</p> <p><u>Optic nerve lesions</u> Arteritic and non-arteritic ischemic optic neuropathy Retrobulbar neuritis Exophthalmos Pituitary tumors Meningiomas Aneurysms Chiasmatic arachnoiditis</p>
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## Treatment

### Indications- Patient has

1. Classic glaucoma triad of visual field loss, optic nerve cupping, and elevated IOP, or is at high risk of developing them.( <b>main</b> )	5. Vascular occlusion associated with increased IOP.
2. Progressive cupping without detectable visual field loss	6. In patients with asymmetric POAG (i.e., bilateral elevated IOP with unilateral optic nerve cupping and unilateral visual field loss),
3. Development of visual field loss,	7. The other eye usually is treated aggressively because it has at least a 40% chance of developing visual field loss over a 5-year period.
4. Episodes of corneal edema caused by elevated IOP,	

### Goals

- The major goal of glaucoma treatment is to preserve good visual function for the patient's lifetime and prevent interference, in so far as is possible, with the quality of life.
- This is accomplished by lowering the IOP (until a better treatment comes along) to a level that will stop, or at least slow, the progression of optic nerve damage and its consequent vision loss.
- The treatment should maximize good visual function and comfort, as well as preserve a reasonable quality of life for the patient by minimizing the side effects from the treatment itself, the risk of vision loss and, in many cases, the costs associated with treatment.
- we have many choices of pressure-lowering therapy, including medications, laser surgery and incisional surgery, no curative therapy exists for POAG, so one can only aim at controlling the disease.
- We can lower the IOP but as yet cannot directly protect the optic nerve or enable regeneration of damaged or dead ganglion cells. Although some improvement in optic nerve parameters can be expected in a minority of patients treated for glaucoma

### Target pressure

- The current practice is to estimate the pressure level (range) below which further damage to the optic nerve is unlikely to occur (target pressure) and then aim to keep the IOPs consistently below this level or, at least, within the estimated range.
- The target pressure is estimated by
  - i. Noting the untreated level of IOP;
  - ii. The degree of optic nerve cupping and visual field loss;
  - iii. The family history of glaucoma;
  - iv. The presence of any other aggravating conditions such as diabetes mellitus or arteriosclerotic vascular disease,
  - v. And the rate of progression if known.
- In the average patient, the clinician should aim for a pressure 20–30% below the initial untreated pressure.
- With greater optic nerve damage, the target pressure should be lowered.
  - i. 0.8 disc diameter cupping or more),
  - ii. Increasing age,
  - iii. And more risk factors,
- The target pressure should be reassessed periodically and lowered if
  - i. Progression,
  - ii. Optic nerve hemorrhage,

iii. Or increase in risk factors occurs

- One should also keep in mind that in the AGIS, not only was lack of progression associated with a low average IOP but also with no IOPs exceeding 18mmHg during the entire 6 years of the study.
- So, maintaining the IOP consistently below 18mmHg in the average glaucoma patient and lower yet in the patient with advanced disease seems like a reasonable goal.
- While lowering pressure definitely slows or stops progression in most patients, it does not do so in all patients and it is difficult to identify those who will progress or reliably determine target pressure from any baseline characteristics.

## Treatment

- The usual progression of treatment in POAG is medical therapy, followed by laser trabeculoplasty, then filtering surgery.

### A. Medical therapy

- 90% of patients can be expected to be controlled with medications over their lifetime
- For the typical glaucoma patient, two or three medications would be the maximum suggested before resorting to laser or incisional surgery.
- At present, maximum medical therapy consists of a prostaglandin-like agent, beta-adrenergic antagonist, a topical carbonic anhydrase inhibitor, and an alpha -agonist.
- Systemic carbonic anhydrase inhibitors are rarely used unless surgery is not feasible or has failed.
- The **failure of medical treatment** is usually judged by
  - i. Inadequate control of IOP,
  - ii. Progressive visual field loss or optic nerve cupping,
  - iii. The appearance of an optic nerve hemorrhage,
  - iv. Intolerable side effects of medication,
  - v. Or demonstrated (or admitted) poor compliance with therapy.

### B. When medical treatment fails or when at least two topical agents have been found wanting addition

- Argon or selective laser trabeculoplasty is the next therapeutic option for most individuals with POAG,
- Primary treatment for those unable or unlikely to use medical therapy.
- Laser trabeculoplasty seems to be as effective as timolol for initial treatment of glaucoma.
- Most ophthalmologists recommend laser trabeculoplasty before resorting to miotics (except in aphakic or pseudophakic eyes),
- This technique reduces IOP substantially in 70–80% of patients.
- Most individuals continue to require at least some medical therapy after laser trabeculoplasty; it is possible to reduce the number of medications in a significant percentage of patients.
- Unfortunately, in many patients, IOP rises again months to years after laser treatment.
- There seems little difference in the long-term outcome between argon and selective laser trabeculoplasty
- AGIS showed that whites responded better to filtering surgery first compared to blacks who responded better to argon laser trabeculoplasty first.

### C. If medical treatment and laser surgery are inadequate to control POAG,

- Filtering surgery is the next appropriate step.
- Filtering surgery controls IOP in approximately 80–90% of patients with POAG

- Filtering surgery is best of the three modalities. but is associated with a greater loss of visual acuity and a higher incidence of cataract( AGM and glaucoma itself may cause cataract)

**D. If one drainage procedure fails to control IOP or if the risk factors for failure are high**

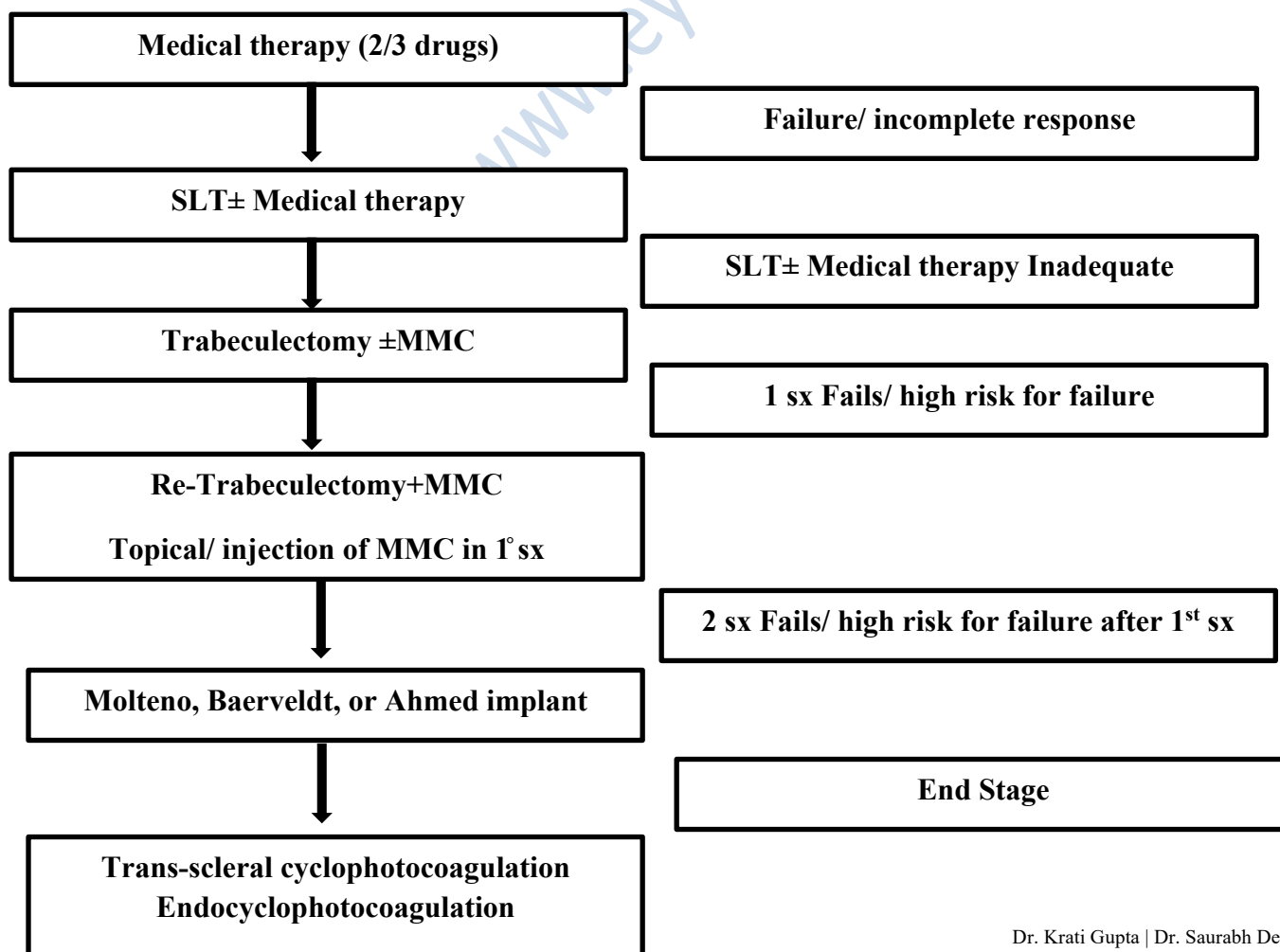
- (e.g., black African ancestry, youth, secondary glaucoma),
- Many ophthalmologists repeat filtering surgery with an inhibitor of wound healing, such as 5-fluorouracil or mitomycin C.
- On the other hand, many use topical application or injection of these agents even in primary filtering operations.

**E. If two or more filtering surgeries have failed despite anti-fibrosis agents or there is a high likelihood of failure after a single filtering operation fails,**

- A tube-shunt (glaucoma drainage) device such as a Molteno, Baerveldt, or Ahmed implant can be used.
- A recently concluded RCT suggests that non-valved implants such as the Molteno or Baerveldt are as good if not somewhat better at controlling IOP at 1 year than a trabeculectomy in a previously operated eye.
- So, some would consider a tube-shunt procedure after the first trabeculectomy (or similar) fails.<sup>3</sup>

**F. End-stage cases.**

- It is also possible to reduce aqueous humor formation by treating the ciliary body with trans-scleral cyclophotocoagulation or endocyclophotocoagulation, although these are usually reserved for end-stage cases.



**Prognosis-** The prognosis in POAG is determined by

The degree of optic nerve damage The vulnerability of the disc tissue The presence of systemic vascular disease	The height of the iop The compliance with treatment The timeliness and appropriateness of treatment
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The patients at greatest risk of blindness are those who present with visual field loss at the time of diagnosis.

cumulative risk of	Unilateral blindness	bilateral blindness
10 years of diagnosis	7.4%	3.4%,
20 years of diagnosis	13.5%	4.3%

**Rehabilitation** referral to

<b>Vision rehabilitation specialist</b>	<b>Orientation and mobility specialists</b>
Can help improve visual function by Optimizing lighting, Enhancing contrast, Reducing glare, Providing adaptations to enhance activities of daily living	Can be consulted and vision substitution strategies (eg, talking books, watches) utilized to improve daily function and quality of life for these patients.

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