

# Ophthalmology

## Glaucoma

### Introduction / Epidemiology

- Glaucoma is a group of progressive optic neuropathies characterized by optic nerve damage and visual field loss, often associated with elevated intraocular pressure (IOP), but not always.
- It's a chronic, often asymptomatic condition until advanced stages, and is a leading cause of irreversible blindness globally.

### Epidemiology:

- **Global prevalence:** Over 76 million people worldwide (as of 2020), projected to increase to 111.8 million by 2040.
- Leading cause of irreversible blindness worldwide.
- **Types:**
  - Primary Open-Angle Glaucoma (POAG): ~70–90% of cases in Western countries.
  - Primary Angle-Closure Glaucoma (PACG): more prevalent in Asia.
  - Secondary Glaucomas: due to other ocular/systemic diseases.
- **Age-related:** Most common in individuals **>40 years**.
- **Risk factors:** Family history, African ancestry (especially for POAG), Asian ancestry (PACG), diabetes, hypertension, corticosteroid use, high myopia, trauma.

### Etiopathogenesis

#### Pathophysiology:

- Optic neuropathy is the final common pathway—characterized by retinal ganglion cell death, optic disc cupping, and visual field defects.
- Intraocular Pressure (IOP): Elevated IOP is the most significant modifiable risk factor.
  - IOP increases due to imbalance between aqueous humor production and outflow.
  - Aqueous humor is produced by the ciliary body and drains via:
    - Trabecular Meshwork (conventional pathway) – impaired in POAG.
    - Uveoscleral outflow (unconventional pathway) – influenced by prostaglandin analogs.

#### Types:

#### 1. Primary Open-Angle Glaucoma (POAG):

- Open anterior chamber angle.
- Chronic, slow progression.

- Pathogenesis: dysfunction in trabecular meshwork → increased IOP → optic nerve damage.

## 2. Primary Angle-Closure Glaucoma (PACG):

- Narrow anterior chamber angle → obstruction of aqueous outflow.
- Can be **acute** (medical emergency) or **chronic**.
- Precipitating factors: pupillary block, shallow anterior chamber, hyperopia.

## 3. Normal-Tension Glaucoma:

- Optic neuropathy despite normal IOP.
- May involve vascular dysregulation, systemic hypotension.

## 4. Secondary Glaucomas:

- Causes: trauma, uveitis, steroid use, neovascularization (e.g., in diabetic retinopathy), pseudoexfoliation syndrome.

## Clinical Features / Symptoms

### Open-Angle Glaucoma (POAG):

- Asymptomatic in early stages.
- Gradual, bilateral **peripheral vision loss** (tunnel vision).
- Central vision remains intact until late stages.
- **No pain**, no redness.
- Late symptoms: severe visual field loss, blindness.

### Angle-Closure Glaucoma (ACG):

- **Acute ACG:**
  - Sudden onset severe eye pain.
  - Headache, nausea, vomiting.
  - Blurred vision, halos around lights.
  - Red eye, mid-dilated unreactive pupil.
  - Hard globe (on palpation).
  - Medical emergency.
- **Chronic ACG:** May be asymptomatic like POAG or have intermittent symptoms.

## Diagnosis & Investigations

### Screening:

- Indicated in high-risk populations (e.g., age >40, family history, African/Asian descent).

### Key Diagnostic Tools:

## 1. Tonometry: Measures IOP.

- Goldmann applanation tonometry is gold standard.
- Normal IOP: 10–21 mmHg.
- Elevated in POAG and ACG, though not always.

## 2. Ophthalmoscopy / Fundoscopy:

- Optic disc evaluation:
  - Increased cup-to-disc ratio ( $>0.6$ ).
  - Notching of neuroretinal rim.
  - Disc hemorrhages.

## 3. Visual Field Testing:

- Perimetry (e.g., Humphrey Field Analyzer).
- Early changes: nasal step, arcuate scotomas.
- Advanced: tunnel vision, central sparing.

## 4. Gonioscopy:

- Evaluates angle status (open vs closed).

## 5. Optical Coherence Tomography (OCT):

- Measures retinal nerve fiber layer (RNFL) thinning.

## 6. Pachymetry:

- Measures central corneal thickness.
- Thinner corneas associated with underestimated IOP and increased glaucoma risk.

## Treatment Goals / Therapeutic Objectives

- Prevent or slow progression of optic nerve damage.
- Preserve visual function and quality of life.
- Reduce IOP to a target pressure individualized for the patient.
- Minimize treatment side effects, cost, and patient burden.
- Monitor disease progression closely over time.

## Non-Pharmacological Therapy

### 1. Lifestyle:

- Limited role, but some suggest:
  - Avoiding activities that raise IOP (e.g., head-down yoga positions).
  - Aerobic exercise may reduce IOP modestly.

## 2. Laser Therapy:

### A. Laser Trabeculoplasty (ALT or SLT):

- Indicated in POAG.
- Improves outflow via trabecular meshwork.
- May be first-line in some guidelines (e.g., LiGHT trial suggests SLT as initial therapy).

### B. Laser Peripheral Iridotomy (LPI):

- Used in angle-closure glaucoma.
- Creates a hole in peripheral iris to relieve pupillary block.

## 3. Surgery:

- Reserved for failed medical/laser therapy.
- Options:
  - Trabeculectomy (most common).
  - Glaucoma drainage devices.
  - Minimally invasive glaucoma surgeries (MIGS).

## Pharmacological Therapy

### First-Line Drugs (for POAG):

#### 1. Prostaglandin Analogues (PGAs):

- Examples: **Latanoprost, Travoprost, Bimatoprost.**
- **Mechanism:** Increases uveoscleral outflow.
- **Dose:** Once daily at bedtime.
- **Advantages:** Potent IOP-lowering, once daily, well-tolerated.
- **Adverse effects:** Eyelash growth, iris pigmentation, conjunctival hyperemia.

#### 2. Beta-blockers:

- Examples: **Timolol, Betaxolol.**
- **Mechanism:** Decrease aqueous humor production.
- **Dose:** Twice daily.
- **Caution:** Contraindicated in asthma, COPD, bradycardia, heart block.

#### 3. Alpha-2 Adrenergic Agonists:

- Example: **Brimonidine.**
- **Mechanism:** Decrease production and increase uveoscleral outflow.

- **Side effects:** Dry mouth, fatigue, allergy, CNS depression in children.

#### 4. Carbonic Anhydrase Inhibitors (topical):

- Example: **Dorzolamide, Brinzolamide.**
- **Mechanism:** Decrease aqueous humor production.
- **Dose:** 2–3 times/day.
- **Systemic CAIs (e.g., Acetazolamide)** used short-term or in acute settings.

#### 5. Rho Kinase Inhibitors (newer):

- Example: **Netarsudil.**
- **Mechanism:** Increases trabecular outflow.
- **Adverse effects:** Conjunctival hyperemia, corneal verticillata.

#### Therapy Principles:

- Start with **monotherapy.**
- **Assess IOP response** in 4–6 weeks.
- Add or switch drugs if inadequate.
- **Combination therapy** if monotherapy fails.

### Individualized Patient-Specific Therapeutic Plan

Factors influencing drug choice:

| Patient Factor                   | Implication                                                                      |
|----------------------------------|----------------------------------------------------------------------------------|
| <b>Age</b>                       | Older patients may tolerate PGAs better; avoid alpha-agonists in young children. |
| <b>Pregnancy</b>                 | Most topical drugs not recommended; beta-blockers may be used with caution.      |
| <b>Comorbidities</b>             | Asthma/COPD: avoid beta-blockers; cardiovascular disease: monitor HR.            |
| <b>Renal/hepatic dysfunction</b> | Caution with systemic CAIs.                                                      |
| <b>Allergies</b>                 | Preservative-free formulations may be required.                                  |
| <b>Adherence issues</b>          | Once-daily PGAs preferred.                                                       |

### Controversies / Special Considerations

- Normal-Tension Glaucoma: Debate about whether to lower IOP in all cases.
- LiGHT Trial (2019): Suggested SLT as first-line treatment over medications.
- Over-reliance on IOP: Some patients with normal IOP can still progress (need for RNFL/visual field monitoring).



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