CLINICAL CASES IN GLAUCOMA
An Evidence-based Approach

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The Health Sciences Publisher
New Delhi | London | Panama
Dedicated
To those, who will read this book
And to Aradhya, who will only ever read the dedication.
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Preface

There is some stuff that we love to read, and then there is some stuff that we have to read: to remain relevant in our clinical practice, to be responsible citizens, to be who we want to be. The *Journal of Current Glaucoma Practice, Journal of Glaucoma, Survey of Ophthalmology, American Journal of Ophthalmology, British Journal of Ophthalmology, Annals of Ophthalmology, National Geographic, New York Times, Guardian, Times of India, Leo Tolstoy, Harry Potter, Orson Scott Card, Calvin and Hobbes, Vogue*...The list is not half complete, we have not mentioned even one of the zillion prescribed textbooks, and we already have three hundred hours of reading, to be crammed into a twenty-four hour day, if we read every day. A twenty-four hour day that also needs us to work, sleep, and go about the general business of living.

So what we have done, in this beautiful little book, is to condense all of the current published glaucoma evidence into tiny bite-sized pieces, relevant to your everyday glaucoma practice. Some of the best minds in glaucoma who have been reading and writing glaucoma for very many years, have got together and picked up representative cases from their own clinics, and delineated the preferred practice pattern in the light of the available evidence-base. We have sifted the information on glaucoma that is currently available, chosen the one which actually stands up to scrutiny, removed the statistical jargon and concentrated on the lowest common denominator: evidence-based clinical practice of glaucoma.

*Clinical Cases in Glaucoma: An Evidence-based Approach* is therefore an easy read, and gives you a real world feel of how the early manifest glaucoma trial (EMGT) or the tube versus trabeculectomy (TVT) or the ocular hypertension treatment study (OHTS) actually translate into the last patient you saw on Thursday. Reading the book will help you make better clinical judgments, if you are a veteran clinician-surgeon. It will help you understand the science of glaucoma better, if you are a researcher, and it will help you take care of your patients better, if you are just stepping into clinical practice.

All of us who have worked on this book together have at various points in our career missed having a book such as this in the front pockets of our lab coats. We are, therefore, delighted to have actually put it together for you, and for ourselves, when the fingerprints of the newly available minimally invasive glaucoma surgery (MIGS) escape us, as we scratch our heads over a 52-year-old patient who keeps forgetting to use his glaucoma medication.

Happy reading, and our best wishes for taking care of your glaucoma patients better. Each day.

Shibal Bhartiya MS
Parul Ichhpujani MS
We would like to acknowledge the efforts of all our authors and contributors. Without their continued support and dedication, this book would not have been possible.

We would also like to thank our publishers, Jaypee Brothers Medical Publishers (P) Ltd for their valued support and faith in us. A special word of gratitude for Ms Chetna Malhotra Vohra (Associate Director—Content Strategy) and our extremely competent and capable Development Editor, Ms Nedup Denka Bhutia.

It would have impossible to take on this project without the continued and unwavering support of our family, friends and colleagues. A very special thanks to them too.
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Primary Open-Angle Glaucoma

Shibal Bhartiya, Parul Ichhpujani

INTRODUCTION

Primary open-angle glaucoma (POAG) is a chronic progressive optic neuropathy defined by an open, normal appearing anterior chamber angle and raised intraocular pressure (IOP), with no other underlying disease, in the presence of the characteristic cupping of the optic disc with corresponding visual field defects, due to retinal ganglion cell loss. If there is an identifiable underlying cause for raised IOP, this is termed secondary glaucoma. If the IOP is within normal limits, this is termed normal-tension glaucoma (NTG) or low-tension glaucoma (LTG).

Case 1: No treatment required for early primary open-angle glaucoma

Mr X, a 72-year-old gentleman, was found to have persistently elevated IOPs on three visits [oculus uterque (OU) 23, 24, 24 mm Hg]. The cup-to-disc (C:D) ratio was 0.5:1 and 0.45:1 with slightly eccentric cup with inferior > superior > nasal > temporal (ISNT) maintained (Fig. 3.1). The visual field [humphrey visual field (HVF) 30-2, Swedish Interactive Threshold Algorithms standard] showed early changes suggestive of glaucomatous damage (Fig. 3.2). The central corneal thickness (CCT) was 531 microns and

Fig. 3.1: Vertical cup-to-disc ratio of 0.5:1 and 0.45:1, with slightly eccentric cup with ISNT rule maintained.
522 microns for the right and left eyes, respectively. The retinal nerve fiber layer (RNFL) optical coherence tomography (OCT) (CIRRUS) did show early RNFL thinning (Fig. 3.3), and gonioscopy showed wide open angles with no pigmentation. He had a coronary artery bypass surgery 6 years ago and was a hypertensive on medication. There was no family history of glaucoma or blindness.

Risks and benefits of initiating glaucoma therapy were discussed with Mr X, keeping in mind the following:
- Life expectancy
- Early field defects
- Slightly elevated eye pressures
- Pre-existing dry eye due to old age which would probably get exacerbated with antiglaucoma medication.

He agreed that deferring treatment was better than initiating treatment immediately on diagnosis. He was asked to follow-up every 4–6 months for 2 years. His visual fields did not show any significant change on serial monitoring. The visual field evaluation was thereafter scheduled for once a year.

Case 2: Treatment for early primary open-angle glaucoma required depending on age

Ms A, a 53-year-old lady, was found to have persistently elevated IOPs on three visits (OU 28, 24, 25 mm Hg) with a diurnal fluctuation of 8 mm Hg in both the eyes. The cup: disc ratio was 0.6:1 and 0.55:1 with a focal neuroretinal rim thinning. The visual field showed early changes suggestive of glaucomatous damage (Fig. 3.4). The CCT was 532 and 526 for the right and left eyes, respectively. The RNFL OCT (Cirrus) showed RNFL thinning (Fig. 3.5), and gonioscopy showed wide open angles. She had no comorbidities. There was no family history of glaucoma or blindness.

Risks and benefits of initiating glaucoma therapy were discussed with Ms A, keeping in mind the following:
- Long-life expectancy
- Early field defects
- Slightly elevated eye pressures.
Fig. 3.3: Early RNFL thinning in the left eye on SDOCT.

(ONH: Optic nerve head; RNFL: Retinal nerve fiber layer; SDOCT: Spectral domain optical coherence tomography).
She agreed that it was better to initiate treatment immediately on diagnosis. She was prescribed travoprost eye drops, one drop each eye, once at bedtime in both eyes and asked to follow-up after 6 weeks. The IOP was found to be 18 mm Hg OU with a diurnal fluctuation of 4 mm Hg and 5 mm Hg, respectively. She was advised to repeat fields after 6 months, and no significant change on serial monitoring was noted for over 2 years. She was advised to continue drops as prescribed.

**Case 3: Treatment for early primary open-angle glaucoma required depending on central corneal thickness**

Mrs X, a 68-year-old lady was found to have persistently elevated IOPs on three visits (OU 24, 25, 25 mm Hg), with a diurnal fluctuation of 9 mm Hg and 8 mm Hg, respectively. The cup: disc ratio was 0.7 and 0.65 with corresponding early changes on visual fields suggestive of glaucomatous damage. The CCT was 472 μ and 482 μ for the right and left eyes, respectively. The RNFL OCT (CIRRUS) showed early RNFL thinning (Fig. 3.6), and gonioscopy showed wide open angles. She had no comorbidities. There was no family history of glaucoma or blindness.

Risks and benefits of initiating glaucoma therapy were discussed with Ms X, keeping in mind the following:
- Life expectancy
- Early field defects
- Elevated eye pressures
- Increased chances of progression in CCT less than 520 microns.

She agreed that it was better to initiate treatment immediately on diagnosis. She was prescribed bimatoprost eye drops, one drop each eye, once at bedtime in both eyes and asked to follow-up after 6 weeks. The IOP was found to be 16 OU with a diurnal fluctuation of 4 mm Hg and 3 mm Hg, respectively. She was advised to repeat fields after 6 months and no significant change on serial monitoring for over 2 years. She complained of dryness in both eyes and was prescribed carboxymethylcellulose eye drops, thrice a day, which obviated her symptoms. She was advised to continue drops as prescribed, and the visual field evaluation was thereafter scheduled for once a year.
Fig. 3.5: Flattening of retinal nerve fiber layer humps on SDOCT. (ONH: Optic nerve head; RNFL: Retinal nerve fiber layer; SDOCT: Spectral domain optical coherence tomography).
Fig. 3.6: SDOCT showing thinning of retinal nerve fibre layer in right eye.
(ONH: Optic nerve head; RNFL: Retinal nerve fiber layer; SD OCT: Spectral domain optical coherence tomography).
Case 4: Treatment for moderate primary open-angle glaucoma requiring more than one antiglaucoma medication

Mr X, a 67-year-old gentleman, was diagnosed with moderate POAG with persistently elevated IOPs on two visits (OD and OS: 34, 35 mm Hg) with a diurnal fluctuation of 8 mm Hg OU. The C:D ratio was 0.85 and 0.7 with broken ISNT rule and corresponding moderate glaucomatous damage on visual fields (Fig. 3.7). The CCT was 522 and 512 for the right and left eyes, respectively. The RNFL OCT also showed flattening of RNFL humps and gonioscopy showed wide open angles.

Risks and benefits of initiating glaucoma therapy were discussed with him, keeping in mind the following:
- Life expectancy
- Field defects
- Elevated eye pressures
- Increased chances of progression in CCT less than 520 microns.

He agreed that it was better to initiate treatment immediately on diagnosis and to try a single medication for efficacy and safety rather than combination therapy. He was prescribed bimatoprost eye drops, one drop each eye, once at bedtime in both eyes, and asked to follow-up after 2 days. The IOP was found to be 24 mm Hg OU, and he was thereafter asked to report for a water drinking test (WDT) after 4 weeks. The IOP was found to be 22 mm Hg OU with a diurnal fluctuation of 4 mm Hg and 6 mm Hg, respectively.

Since the target IOP was not reached with one drug, a fixed dose combination of bimatoprost and timolol was advised, once at bedtime. The fact that the efficacy of timolol is less at night was weighed against the chances of reduced compliance with addition of a second bottle. This was discussed with the patient and he preferred to use a fixed-dose combination (FDC) in view of convenience of use. At 4 weeks follow-up, the IOP was found to be 16 mm Hg OU with a diurnal fluctuation of 3 mm Hg and 5 mm Hg, respectively.

He was advised to repeat fields after 4 months and no significant change was observed on serial monitoring for over 2 years. He was advised to continue drops as prescribed and the visual field evaluation was thereafter rescheduled for once every 6 months, with a RNFL OCT performed annually.

Case 5: Treatment of severe primary open-angle glaucoma requiring surgery since not controlled on maximal tolerable medical therapy

Mr X, a 69-year-old pseudophakic gentleman, was on treatment for advanced POAG over the last 6 years with persistently elevated IOPs on two visits (OD and OS: 26, 25)
with a diurnal fluctuation of 8 mm Hg OU on treatment [bimatoprost harmonized system (HS), brimonidine + timolol FDC BD, brinzolamide BD]. The C:D ratio was 0.9 OU with a marked concentric neuroretinal rim (NRR) loss (Fig. 3.8). The visual field could not be performed to poor visual acuity. The CCT was 532 and 541 for the right and left eyes, respectively. Risks and benefits of glaucoma surgery were discussed with him and he agreed that it was better to go ahead with surgery since even maximal topical therapy was insufficient to control his IOP.

He was advised trabeculectomy augmented with mitomycin C, for the right eye first, followed by the left eye. After surgery his IOP was 16 and 18, respectively, without any medication, despite release of the releasable sutures in the early postoperative period. He required the addition of a prostaglandin analog (bimatoprost HS, OU) to achieve target pressure of 11 mm Hg and 12 mm Hg, respectively. He attained the target IOP and then he was advised to continue drops as prescribed.

Case 6: Treatment of severe primary open-angle glaucoma requiring surgery at first diagnosis due to severe visual field loss

Mrs X, a 65-year-old pseudophakic lady with diabetes, presented to the outpatients clinic with IOP of 32 both eyes, and a near total optic atrophy in the right eye, and a C:D ratio of 0.85 OD and 0.9:1 OS. Her best-corrected visual acuity in right eye was 6/60 and 3/60 in the left eye. A 24-2 HVF OD was predictably showing a severe visual field loss encroaching fixation and a 10-2 test was there after advised, which showed split fixation. Visual field in left eye could not be performed due to poor vision.

The possibility of imminent visual loss was discussed with her and the risks and benefits of primary surgery were also discussed with the patient and her family. Given the advanced stage of glaucomatous damage, high IOP, it was decided to perform a primary trabeculectomy on both eyes at an interval of 4 weeks. In the interim, she was referred to an internist for euglycemic control and prescribed bimatoprost HS, brimonidine + timolol FDC BD and brinzolamide BD eye drops for both eyes. After surgery, her IOP was 12 and 11, respectively, without any medication following release of the releasable sutures in the early postoperative period for the left eye only. A repeat field was ordered after 4 months for the right eye and no significant change was observed on serial monitoring for over a year.

INVESTIGATIONS

Every patient of glaucoma requires a careful and comprehensive eye examination. Mandatory tests include:

- **Visual Acuity and Refraction**
- **Tonometry (Applanation)**

On at least two different occasions, at different times of the day, with IOP more than 21 mm Hg is mandatory for diagnosing POAG. IOP less than 21 mm Hg does not rule out glaucoma.
Chapter 3: Primary Open-Angle Glaucoma

- **Slit Lamp Examination**
  A through slit lamp evaluation is mandatory to rule out any secondary reasons for elevated IOP.

- **Gonioscopy**
  Anatomically normal and open angles are mandatory for diagnosing POAG.

- **Optic Nerve Assessment**
  A dilated assessment of the optic nerve head (ONH) is essential together with a red-free evaluation of the peripapillary RNFL. The ONH can be documented using a hand-drawn, labeled diagram (special emphasis on cup/disc ratio, notching, RNFL defects and/or hemorrhage) and/or a clinical picture. A color photo and a red-free photo of the ONH are essential for serial follow-up.

- **Visual Field Testing**
  Reliable visual fields provide a baseline for future follow-up. The first visual fields are usually discarded as unreliable or having a learning curve.

- **Central Corneal Thickness (Pachymetry)**
  It is an adjunct that helps to make therapeutic decisions.

- **Imaging of the Optic Nerve with Retinal Nerve Fiber Layer Analysis**
  Imaging of the optic nerve with RNFL analysis (ocular coherence tomography, Heidelberg retinal tomography or scanning laser polarimetry) provides a statistical comparison with the normative database, thereby providing additional objective information for subsequent management.

  In addition, the following tests, if performed, help in managing the condition better in case the facilities exist and are affordable to the patient.

- **Diurnal Variation of Intraocular Pressure**
  A 24-hour diurnal variation of IOP includes IOP recording every 2 hours, preferably using the same Goldmann applanation tonometry, by the same observer, whenever possible. A diurnal variation of more than 8 mm over 24 hours is considered indicative of glaucoma. Diurnal variation of IOP curve may provide additional information and influence treatment protocol.

- **Water Drinking Test**
  A WDT with 10 mL/kg body weight of water over 5 minutes may be performed as a surrogate for diurnal variation of IOP to provide a rough idea of IOP peaks and fluctuation.

- **Stereo-optic disc photographs to confirm normal optic nerve parameters and document baseline.**

<table>
<thead>
<tr>
<th>Severity</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>GON + normal visual field on SAP</td>
</tr>
<tr>
<td>Moderate</td>
<td>GON + VFD in one hemifield, but not within 5° of fixation on SAP</td>
</tr>
<tr>
<td>Severe</td>
<td>GON + VFD in both hemifields ± loss within 5° of fixation in at least one hemifield on SAP</td>
</tr>
</tbody>
</table>

(GON: Glaucomatous optic neuropathy; VFD: Visual field defect; SAP: Standard automated perimetry).

**Table 3.1: Severity of primary open-angle glaucoma.**

**FOLLOW-UP PROTOCOL**

Follow-up protocol is to be customized to the individual patient depending on the risk of developing glaucoma, risk factors present and whether treatment has been initiated or not. Initially, a follow-up may be scheduled after 4–6 weeks for safety and efficacy checks after initiating topical anti-glaucoma therapy. Six fields, done over 2 years, are required for establishing the rate of progression.

Repeat visual field and optic nerve testing may be performed annually or sooner if changes are suspected.

**WHAT IS THE NATURAL COURSE OF THE DISEASE?**

- Of approximately 1.2 million RGC at birth, about 25% are naturally lost over 75 years.

- With POAG retinal ganglion cell loss is accelerated with a generally slow, but variable rate of loss.

- Up to 40% of optic nerve fibers need to be lost before a visual field defect appears on automated perimetry usually progressing from paracentral or mid-peripheral defect in the earlier stages to temporal visual field loss and loss of central fixation points in advanced disease.

- On comparing the mean age at presentation of patients with early relative visual field loss to those with absolute field loss within 5° of fixation, the estimated average time for untreated early disease to progress to end-stage blindness substratified by IOP levels 21–25 mm Hg, 25–30 mm Hg, and more than 30 mm Hg was 14.4 years, 6.5 years and 2.9 years, respectively.
Clinical Cases in Glaucoma: An Evidence-based Approach

**Table 3.2: Randomized clinical trials in primary open angle glaucoma establishing the role of intraocular pressure lowering in reducing development and progression of disease.**

<table>
<thead>
<tr>
<th>Trial</th>
<th>No. of patients</th>
<th>Follow-up years</th>
<th>Study</th>
<th>Intervention</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>OHTS</td>
<td>1,636</td>
<td>10</td>
<td>Eyes without POAG and IOP 24–32 mm Hg 20% IOPR</td>
<td>Medications</td>
<td>POAG risk: 4.4% treated vs. 9% untreated 10% increased risk for every mm Hg increase</td>
</tr>
<tr>
<td>CNTGS</td>
<td>230</td>
<td>7</td>
<td>POAG in eyes with IOP&lt; 24 mm Hg 30% IOPR</td>
<td>Medications and surgery</td>
<td>POAG progression: 12% treated vs. 35% untreated</td>
</tr>
<tr>
<td>EMGT</td>
<td>255</td>
<td>7–11</td>
<td>Newly diagnosed early stage POAG. IOPR protocol driven</td>
<td>Betaxolol and laser trabeculectomy or no treatment</td>
<td>POAG progression: 45% treated vs. 62% untreated</td>
</tr>
<tr>
<td>CIGTS</td>
<td>607</td>
<td>5+</td>
<td>Newly diagnosed POAG. IOPR protocol driven</td>
<td>Medications and surgery</td>
<td>No significant difference in visual field loss with initial trabeculectomy (~46%) vs. medical therapy (~36%)</td>
</tr>
<tr>
<td>AGIS</td>
<td>591</td>
<td>10–13</td>
<td>Advanced POAG. IOPR protocol driven</td>
<td>Argon laser trabeculectoplasty (A) and trabeculectomy (T): ATT and TAT sequences</td>
<td>Visual function outcomes better with ATT in blacks and TAT in whites. Mean visual field loss 3 times greater when IOP 14.0–17.5 mm Hg vs.&lt; 14.0 mm Hg.</td>
</tr>
</tbody>
</table>

(POAG: Primary open-angle glaucoma; OHTS: Ocular hypertension treatment study; IOPR: Intraocular pressure reduction; CNTGS: Collaborative normal-tension glaucoma study; EMGT: Early manifest glaucoma trial; CIGTS: Collaborative initial glaucoma treatment study; AGIS: Advanced glaucoma intervention study; ATT: Argon trabeculectomy followed by trabeculectomy followed by trabeculectomy; TAT: Trabeculectomy followed by argon trabeculectomy followed by trabeculectomy.)

- Early manifest glaucoma trial (EMGT) has shown that progression was faster in older than in younger patients (p = 0.002), and those with newly diagnosed untreated pseudoexfoliative glaucoma (PXFG) (93%) compared with high-tension glaucoma (HTG) (74%) or NTG (56%) (p = 0.012) over 5 years. Median time to progression also differed considerably among groups; 19.5 months in pseudoexfoliation glaucoma, 44.8 months in HTG and 61.1 months in NTG (p less than 0.0001).
- Table 3.2 enlists the trials, which show the beneficial effect of IOP lowering in reducing the disease progression.

**BROAD GUIDELINES FOR MANAGEMENT**

The goals of treatment in POAG are to control IOP in a target range and to maintain stable optic nerves, RNFL and visual fields. The target IOP is different for each patient and is the pressure at which it is thought that the patient will not sustain further damage. Table 3.3 enlists the risk categories, which guide treatment targets.

**POINTS TO REMEMBER**

1. Intraocular pressure must be measured two or more times on separate occasions before labeling a patient as having elevated eye pressures. The risk for ONH damage increases 10 times when IOP more than or equal to 24 mm Hg, more than 40 times when IOP more than 30 mm Hg.
2. A gonioscopy must be performed to rule out angle closure and a slit lamp biomicroscopy, and/or imaging studies of the ONH must be performed to document optic nerve damage. A reliable visual field is essential for diagnosing glaucoma.
3. A pachymetry is required to give an indication of the eyes ability to withstand higher pressures. There are no validated nomograms for IOP correction on the basis of CCT. IOP corrected for corneal thickness, therefore, does not provide a valid basis for initiating or not initiating therapy.
4. The threshold for starting treatment and establishing target IOP for POAG must be lower for patients with increased risk factors. These include:
   - **Race:** West Africans, Afro-Caribbeans and Hispanics have the highest predilection for disease as well as blindness
   - **Family history:** Family history of glaucoma, or glaucoma-induced blindness: a first-degree relative with POAG increases the risk 9 times, and increases the risk of disease to and 23%.
   - **Age:** Younger patients
Chapter 3: Primary Open-Angle Glaucoma

- **Patients with myopia:** Myopic eyes may have weaker scleral support, thus becoming more susceptible to damage, with an additional familial link between the two diseases.

- Patients with poor access to repeat glaucoma investigations.

- **Thin CCT:** A CCT of less than or equal to 555 μm increased the risk three times as compared with a CCT more than 588 μm. For every 40 μm decrease in CCT, the relative risk of developing POAG is 1.71.

- **Optic nerve head hemorrhage:** Disc hemorrhage increases risk of POAG 3.7 times, although most eyes with the hemorrhage (87%) may not develop POAG over 5 years (OHTS).

- **Low Ocular Perfusion Pressure:** Diastolic ocular perfusion pressure (OPP) [diastolic blood pressure (BP) – IOP] less than or equal to 125 mm Hg may alter blood flow to the ONH and systolic OPP (systolic BP – IOP)

- **Ancillary risk factors:**
  - Genetic: Myocilin gene (MYOC) on chromosome 1 (3–4% of POAG)
  - Vasospasm: Migraine, Raynaud’s disease
  - Long-term steroid use
  - Obstructive sleep apnea.

- **Glaucoma suspect with moderate risk**
  - Fellow eye of established GON: excluding secondary unilateral glaucoma
  - OH with multiple risk factors: thin CCT, high IOP, suspicious discs
  - GLC gene mutations associated with severe POAG
  - Recurrent disc hemorrhages
  - Pseudoexfoliation
  - Younger age

  Monitor closely for change or treat depending on risk and patient preferences
  Treat if risk(s) increase(s) with ≥20% IOP reduction or 1 SD above population mean

- **Glaucoma suspect with low risk**
  - OH
  - Older age
  - Pigment dispersion with normal IOP
  - Glaucoma suspect disc, including disc asymmetry
  - Glaucoma family history
  - Less important:
    - Steroid responder
    - Myopia
    - β-peripapillary atrophy
    - Diabetes mellitus
    - Uveitis
    - Systemic hypertension

  Monitor

### Table 3.3: Risk categories to guide treatment targets for primary open angle glaucoma.

<table>
<thead>
<tr>
<th>Risk category*</th>
<th>Description</th>
<th>Treatment targets</th>
</tr>
</thead>
<tbody>
<tr>
<td>High</td>
<td>Moderate-advanced GON with VFD+ Higher IOP Rapid progression Bilateral VFD Pigmentary or pseudoexfoliative glaucoma Advanced VFD or fixation threat Glaucoma-related visual disability Younger age</td>
<td>≥40% IOPR or 1–2 SD below population mean (9–12 mm Hg)</td>
</tr>
<tr>
<td>Moderate</td>
<td>Mild GON with early VFD Mild-moderate GON with low IOP Younger age</td>
<td>&gt;30% IOP reduction or population mean</td>
</tr>
<tr>
<td>Glaucoma suspect with moderate risk</td>
<td>Fellow eye of established GON: excluding secondary unilateral glaucoma OH with multiple risk factors: thin CCT, high IOP, suspicious discs GLC gene mutations associated with severe POAG Recurrent disc hemorrhages Pseudoexfoliation Younger age</td>
<td>Monitor closely for change or treat depending on risk and patient preferences Treat if risk(s) increase(s) with ≥20% IOP reduction or 1 SD above population mean</td>
</tr>
<tr>
<td>Glaucoma suspect with low risk</td>
<td>OH Older age Pigment dispersion with normal IOP Glaucoma suspect disc, including disc asymmetry Glaucoma family history</td>
<td>Monitor</td>
</tr>
</tbody>
</table>

(GON: Glaucomatous optic neuropathy; VFD: Visual field defect; IOP: Intraocular pressure; IOPR: Intraocular pressure reduction; OH: Ocular hypertension; CCT: Central corneal thickness; GLC: Glaucoma; POAG: Primary open-angle glaucoma).


5. It is important to consider the economics of glaucoma therapy as also compliance issues. Quality of life costs of treatment versus no treatment must be weighed for the individual patient.

6. The risks and benefits of selective laser trabeculoplasty versus topical glaucoma therapy must be discussed with the patient.

7. Advanced visual field damage must be addressed surgically whenever required. Indications for surgery are discussed later in the book.
SUGGESTED READING


