Clinical Cases in
GLAUCOMA
An Evidence-based Approach

Shibal Bhartiya • Parul Ichhpujani

JAYPEE

Contents

Chapter 3

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

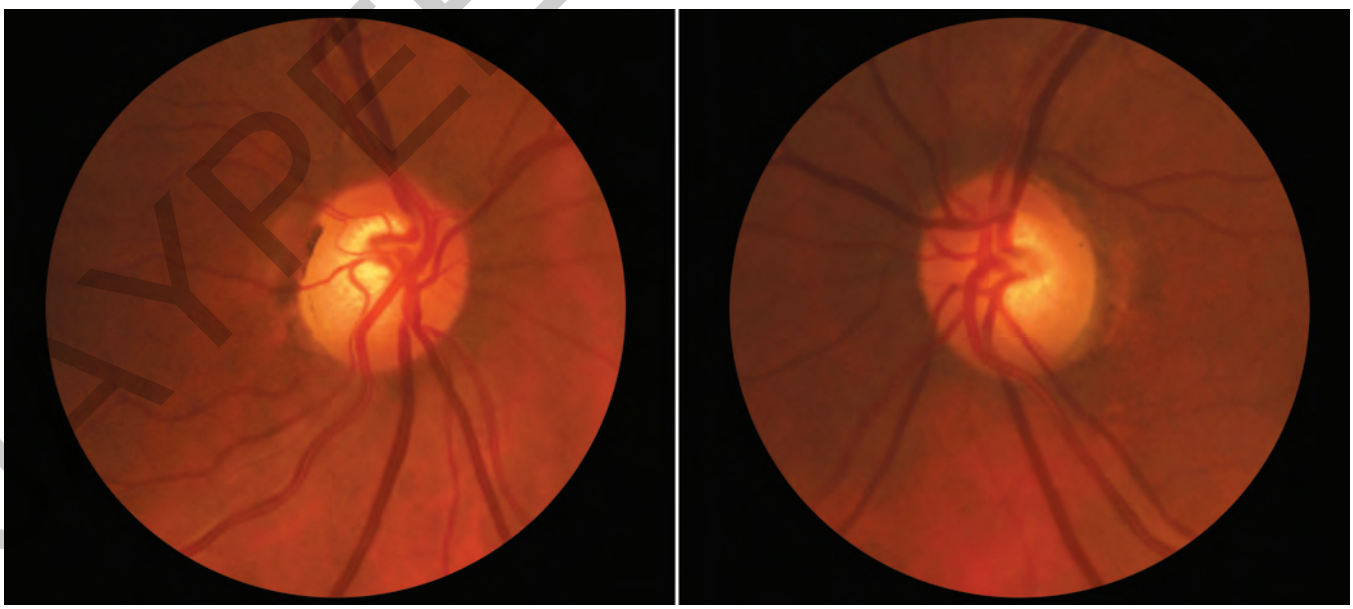

Fig. 3.1: Vertical cup-to-disc ratio of 0.5:1 and 0.45:1, with slightly eccentric cup with ISNT rule maintained.

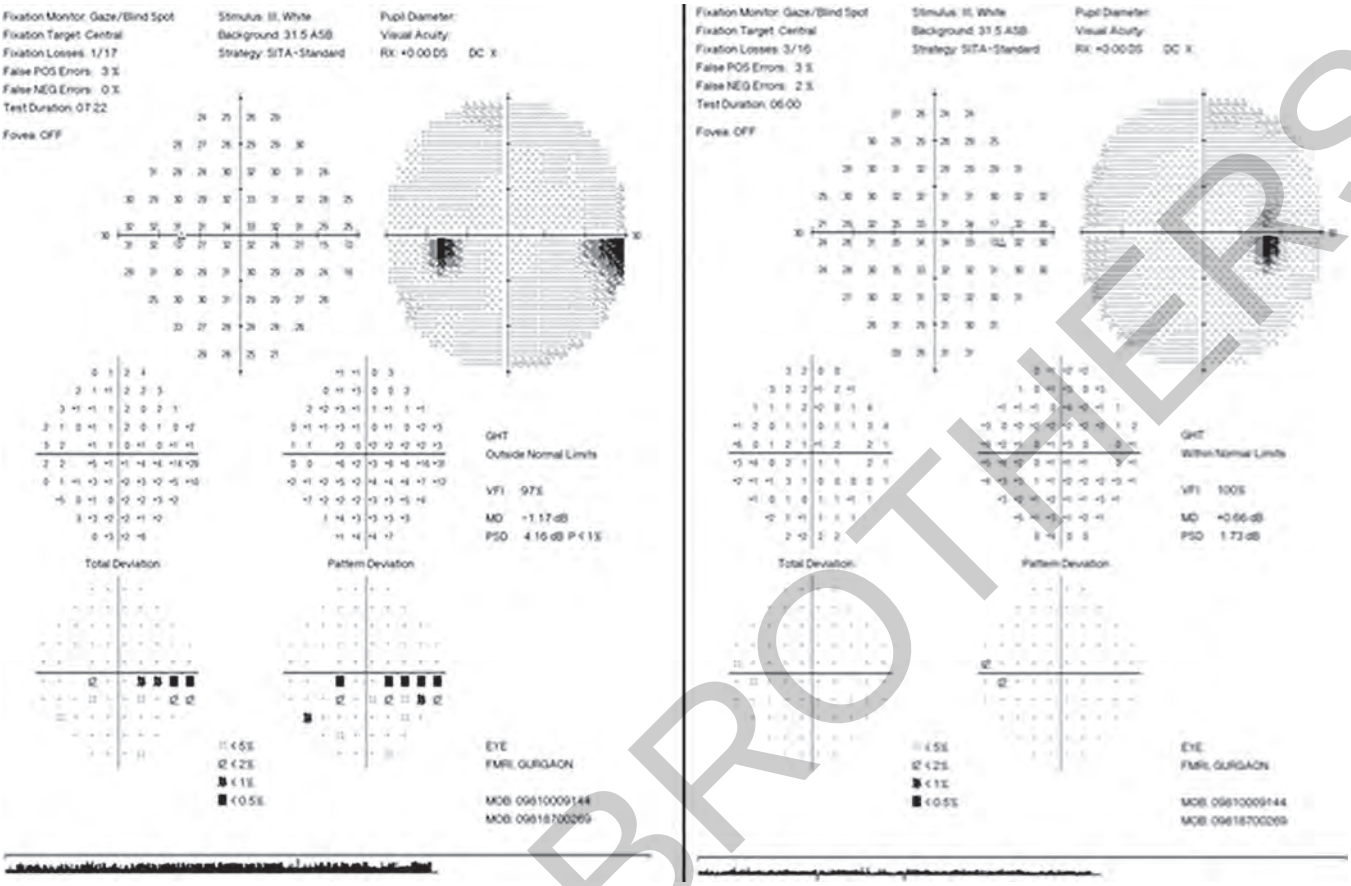

Fig. 3.2: Early glaucomatous damage in left eye.

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.

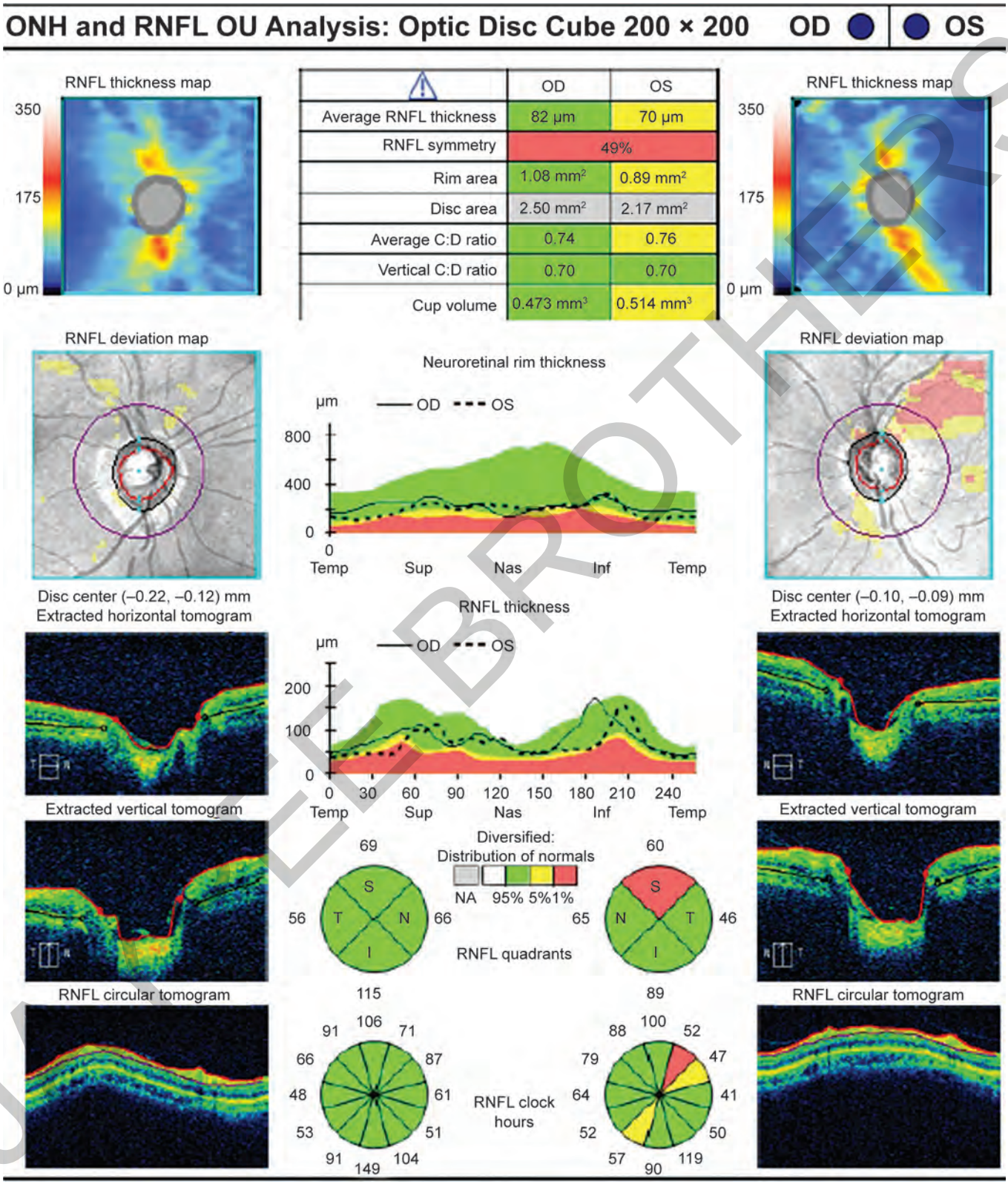

Fig. 3.3: Early RNFL thinning in the left eye on SDOCT.
(ONH: Optic nerve head; RNFL: Retinal nerve fiber layer; SDOCT: Spectral domain optical coherence tomography).

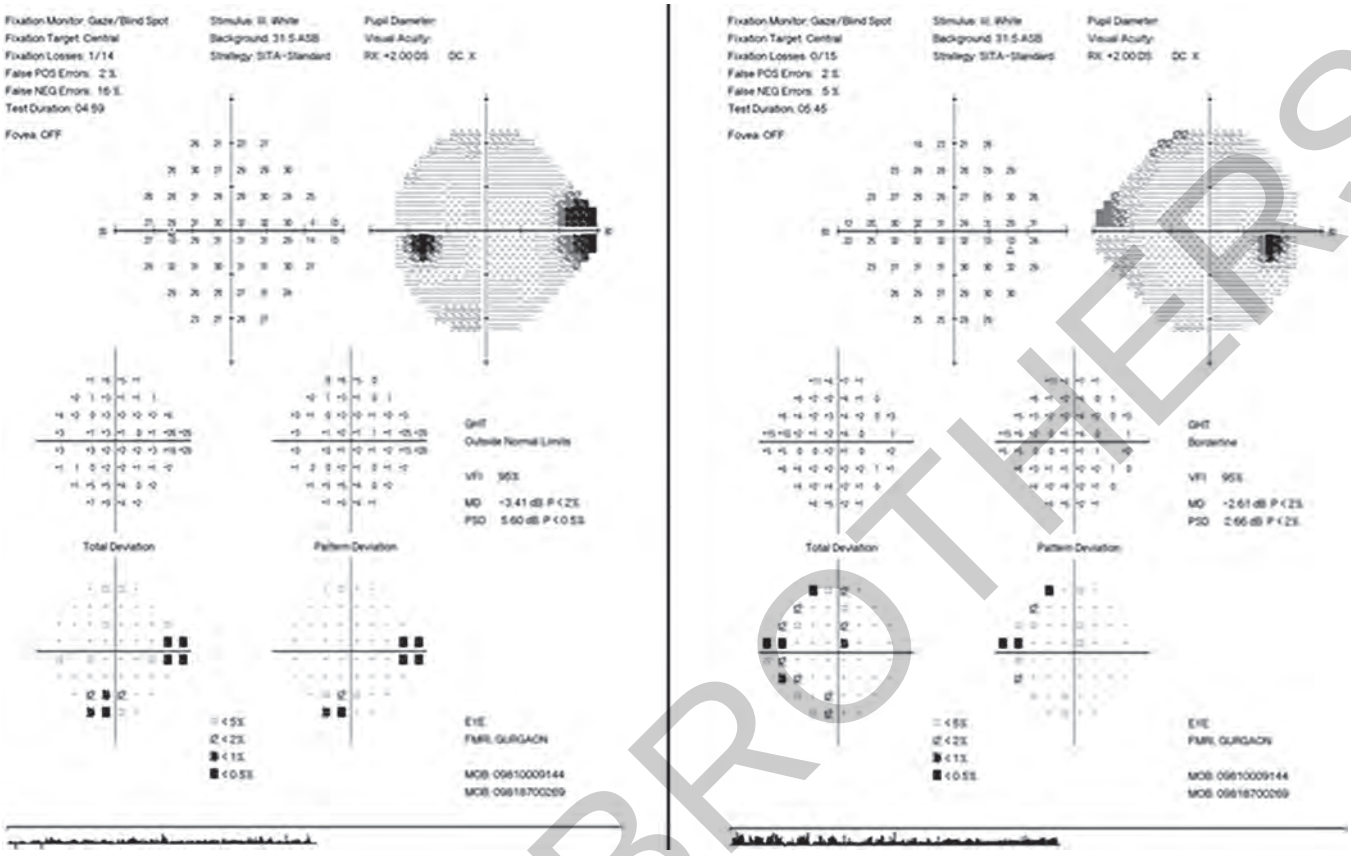

Fig. 3.4: Early glaucomatous visual field defects.

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.

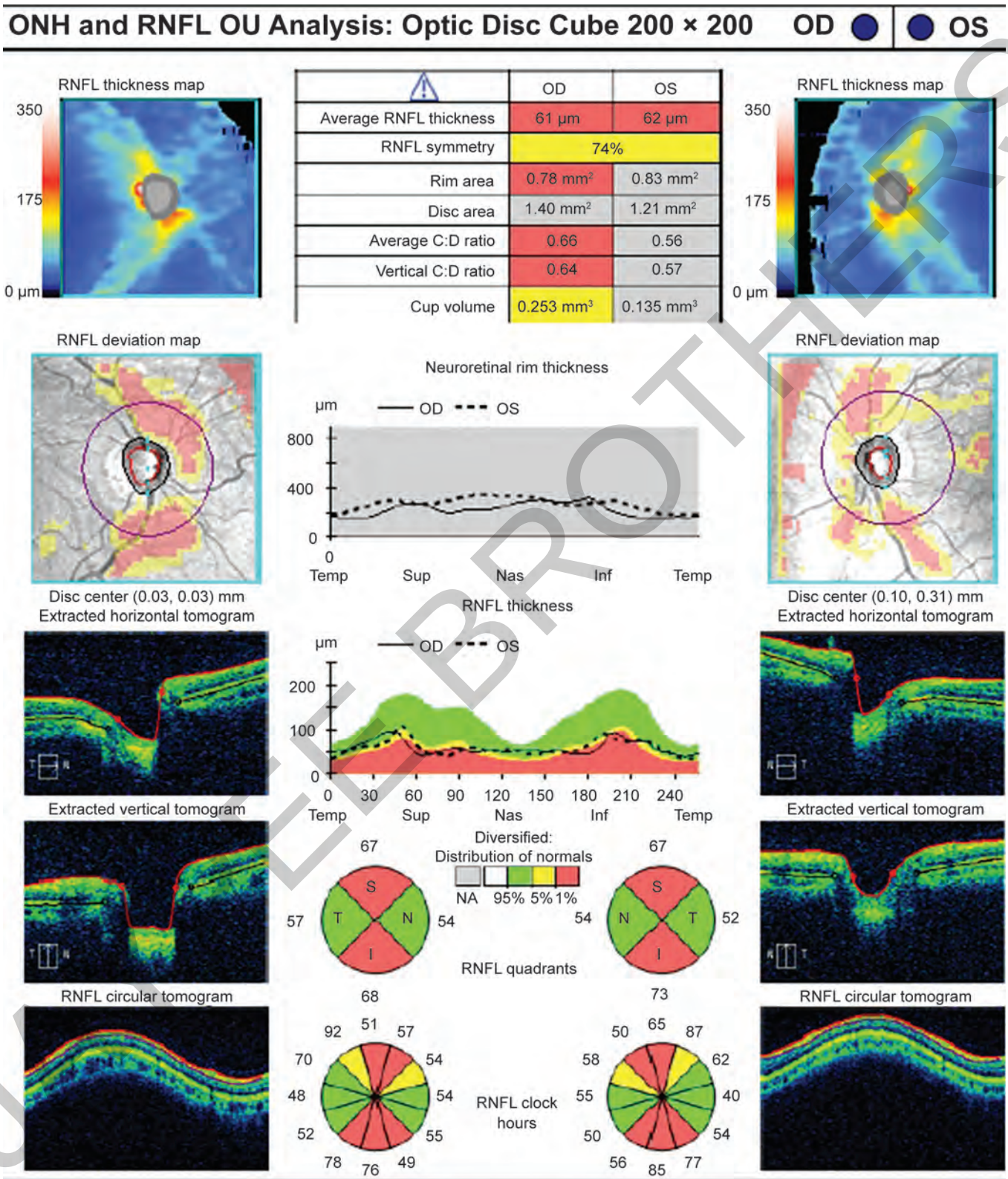

Fig. 3.5: Flattening of retinal nerve fiber layer humps on SDOCT.
(ONH: Optic nerve head; RNFL: Retinal nerve fiber layer; SDOCT: Spectral domain optical coherence tomography).

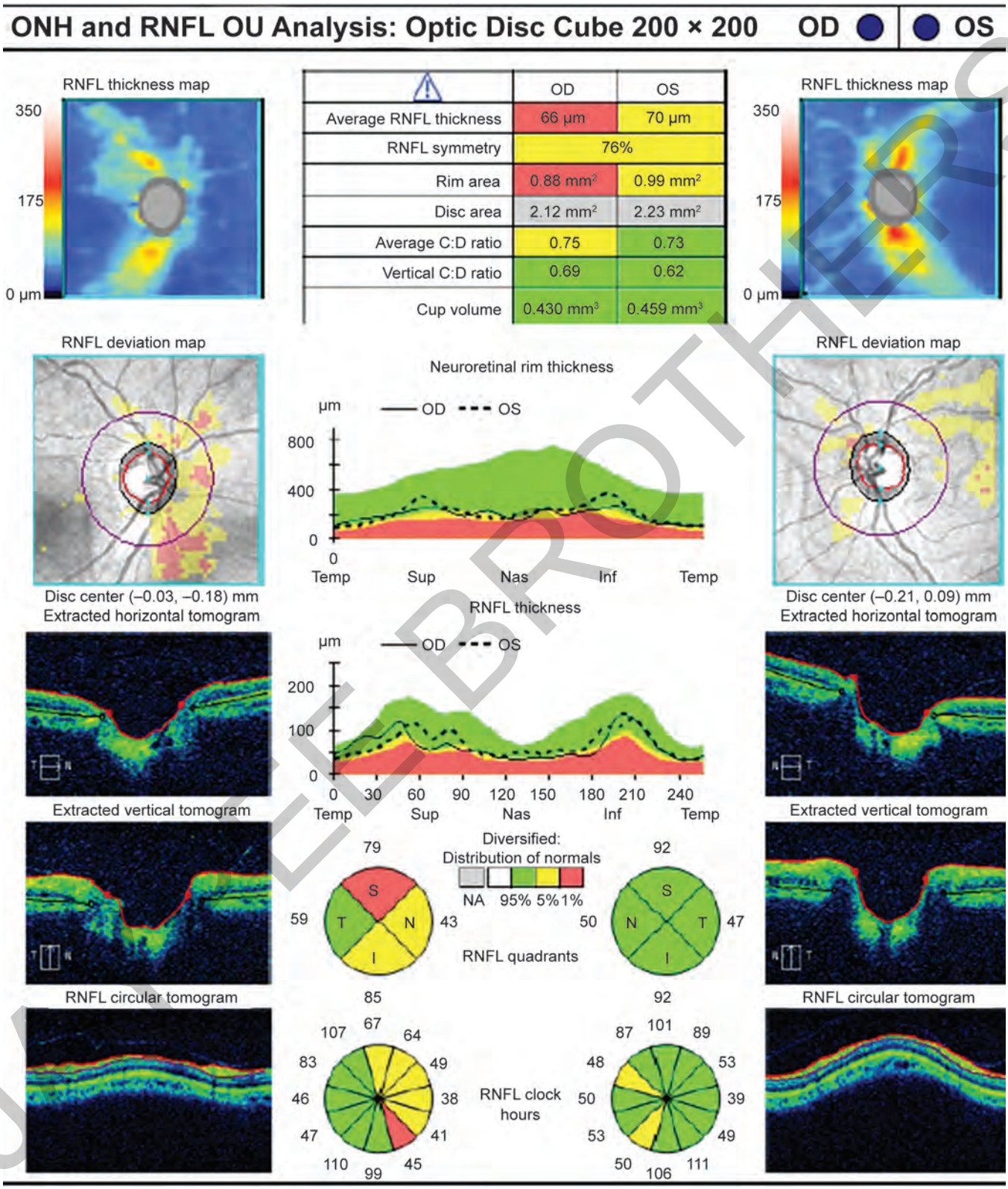

Fig. 3.6: SDOCT showing thinning of retinal nerve fibre layer in right eye.
(ONH: Optic nerve head; RNFL: Retinal nerve fiber layer; SD OCT: Spectral domain optical coherence tomography).

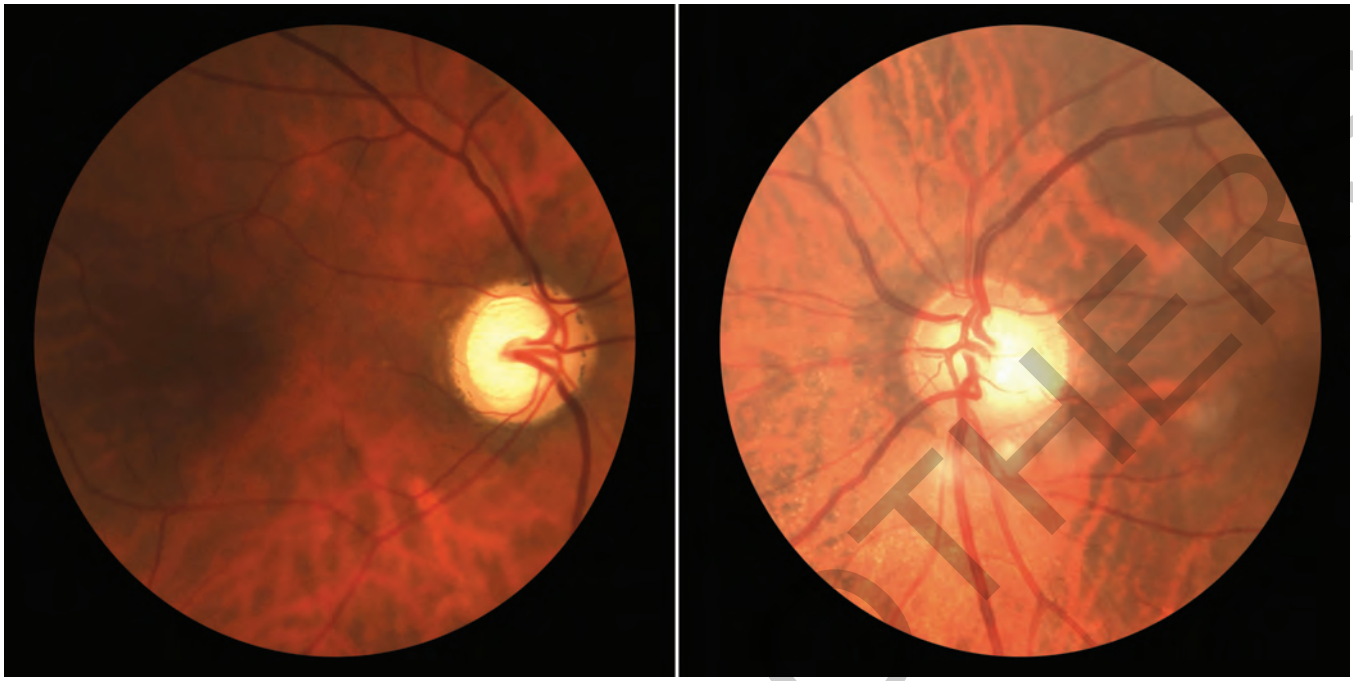

Fig. 3.7: Vertical cup-to-disc ratio of 0.85 and 0.7, with broken ISNT rule.

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)

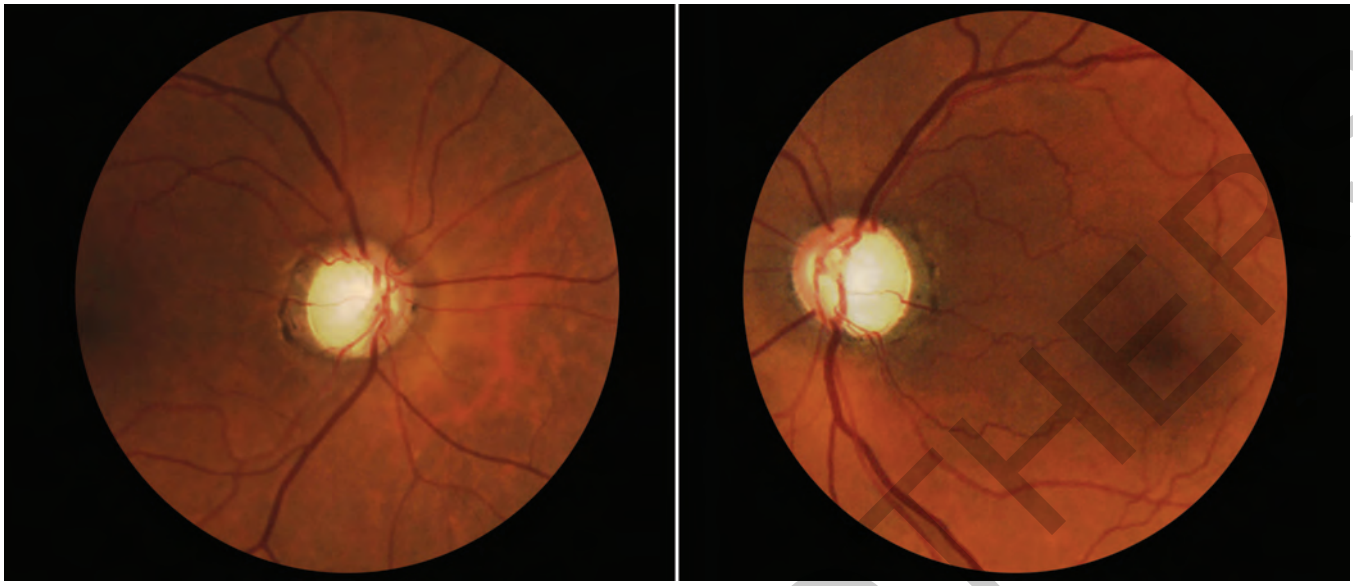

Fig. 3.8: Vertical cup-to-disc ratio of 0.9 OU with a marked concentric neuroretinal rim loss.

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.

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

Severity	*Description*
Mild	GON + normal visual field on SAP
Moderate	GON + VFD in one hemifield, but not within 5° of fixation on SAP
Severe	GON + VFD in both hemifields ± loss within 5° of fixation in at least one hemifield on SAP

(GON: Glaucomatous optic neuropathy; VFD: Visual field defect; SAP: Standard automated perimetry).
Source: American Academy of Ophthalmology Glaucoma Panel. (2010). Preferred practice pattern guidelines. Primary open-angle glaucoma. [online] Available from www.aao.org/ppp. [Accessed July, 2016].

- Based on the above investigations POAG severity can be graded as mentioned in Table 3.1.

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 antiglaucoma 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.

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.

Trial	*No. of patients*	*Follow-up years*	*Study*	*Intervention*	*Outcome*
OHTS	1,636	10	Eyes without POAG and IOP 24–32 mm Hg 20% IOPR	Medications	POAG risk: 4.4% treated vs. 9% untreated 10% increased risk for every mm Hg increase
CNTGS	230	7	POAG in eyes with IOP< 24 mm Hg 30% IOPR	Medications and surgery	POAG progression: 12% treated vs. 35% untreated
EMGT	255	7–11	Newly diagnosed early stage POAG. IOPR protocol driven	Betaxolol and laser trabeculoplasty or no treatment	POAG progression: 45% treated vs. 62% untreated
CIGTS	607	5+	Newly diagnosed POAG. IOPR protocol driven	Medications and surgery	No significant difference in visual field loss with initial trabeculectomy (−46%) vs. medical therapy (−38%)
AGIS	591	10–13	Advanced POAG. IOPR protocol driven	Argon laser trabeculoplasty (A) and trabeculectomy (T): ATT and TAT sequences	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.< 14.0 mm Hg.

(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 trabeculoplasty followed by trabeculectomy followed by trabeculectomy; TAT: Trabeculectomy followed by argon trabeculoplasty 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

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

*Risk category**	*Description*	*Treatment targets*
High	• 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	≥40% IOPR or 1–2 SD below population mean (9–12 mm Hg)
Moderate	• Mild GON with early VFD • Mild-moderate GON with low IOP • Younger age	>30% IOP reduction or population mean
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

(GON: Glaucomatous optic neuropathy; VFD: Visual field defect; IOP: Intraocular pressure; IOPR: Intraocular pressure reduction; OH: Ocular hypertension; CCT: Central corneal thikness; GLC: Glaucoma; POAG: Primary open-angle glaucoma).
Source: Adapted from Asia-Pacific Glaucoma Guidelines, 2nd edition. 2008.

- *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 50 mm Hg may alter blood flow to the ONH and systolic OPP (systolic BP – IOP) less than or equal to 125 mm Hg is known to have a higher risk of POAG progression.
- *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.

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

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Clinical Cases in

Glaucoma

An Evidence-based Approach

Clinical Cases in Glaucoma: An Evidence-based Approach is an easy-to-read, comprehensive overview of the available evidence in current glaucoma practice. Representative cases with diagrams and pictures, as also flowcharts and algorithms for patient management, form the core structure of the manual.

The book aims to enable the student/practitioner in approaching each patient in a logical and scientific manner. It will also aid in preparing for clinical case discussions at various fora and exams. The review of literature and accompanying commentary provide useful insight and a holistic, multidisciplinary overview of glaucoma.

In the current paradigm of disease management, which is becoming increasingly focused on the evidence pyramid, *Clinical Cases in Glaucoma: An Evidence-based Approach* will be an invaluable handbook for understanding, teaching and practicing glaucoma.

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An avid educator and researcher, she has been responsible for the design and execution of many clinical trials in both clinical and basic research. Dr. Bhartiya has lectured at, and chaired various sessions in regional and international meetings. She serves as a Reviewer for many ophthalmology journals, and is the Executive Editor of the *Journal of Current Glaucoma Practice*: The Official Journal of the International Society of Glaucoma Surgery.

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