

Glaucoma in general practice: An updated framework for Australian general practitioners

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Background

Glaucoma is a leading cause of irreversible blindness in Australia, with over half of cases undiagnosed. General practitioners (GPs) have a central role in early recognition, risk stratification and coordination of care.

Objective

This article aims to provide a practical framework for GPs to identify glaucoma risk, recognise red-flag presentations and support long-term management by providing a narrative review of contemporary guidelines, systematic review and key clinical trials relevant to primary care, with emphasis on referral pathways and shared-care models.

Discussion

Glaucoma is best conceptualised as a progressive optic neuropathy, with intraocular pressure a modifiable risk factor. Acute angle-closure glaucoma requires same-day referral, while most open-angle disease is asymptomatic and detected through opportunistic screening. Risk-based referral thresholds and medication considerations are outlined. Timely referral, reinforcement of treatment adherence and shared care with optometry and ophthalmology are critical to preventing avoidable vision loss in general practice.

GLAUCOMA IS THE SECOND LEADING CAUSE

of irreversible blindness in Australia in people aged over 55 years, with prevalence increasing as the population ages, but over 50–60% of cases in Australia remain undiagnosed.^{1,2} The economic impact of glaucoma to Australia is significant and rising, with \$4.3 billion in 2025 (up from \$1.9 billion in 2005) in direct and indirect healthcare system costs and lost wellbeing.³ Glaucoma is best understood as a progressive optic neuropathy in which intraocular pressure (IOP) is a modifiable risk factor, not the disease itself; optimal management focuses on slowing disease progression tailored to stage, age and comorbidities.¹

Progression in glaucoma is confirmed with formal visual field (VF) testing and optical coherence tomography (OCT), both of which are outside the scope of general practice and instead performed by optometrists and ophthalmologists.^{4–6} This underscores the key role of general practitioners (GPs) in care coordination rather than direct diagnosis or monitoring, with optometry-led VF testing and optic nerve imaging now central to early community detection and ongoing surveillance.^{4,6} Decisions on glaucoma management, including diagnosis confirmation and progression assessment, should remain under ophthalmologist supervision, especially for patients showing progression, advanced disease or high-risk features.^{5,6}



Figure 1 conceptualises key investigations in glaucoma assessment for GPs, illustrating how VF testing and OCT imaging, while typically conducted by eye specialists, are integral for confirming disease progression.

Early vision preservation in general practice hinges on recognising high-risk patients, referring to eye care specialists and ensuring strict adherence to prescribed glaucoma therapy through monitoring drop adherence and checking that side effects are tolerable.

Classification

Glaucoma is broadly classified by its mechanism and urgency.⁷ Angle-closure glaucoma (ACG) occurs when aqueous outflow is blocked by apposition of the iris to the trabecular meshwork (Figure 2).⁸ Acute attacks cause severe ocular pain, haloes, blurred vision, headache and nausea, and require same-day ophthalmology referral.⁷ Primary open-angle glaucoma (POAG) is chronic and typically asymptomatic, presenting with optic-nerve cupping and field loss, sometimes despite normal IOP (normal-tension glaucoma).⁹ Secondary glaucomas arise from corticosteroid use, uveitis, trauma or vascular disease, with referral urgency determined by the underlying cause.⁷

Figure 3(A) shows a patient history of haloes requiring consideration of ACG. An examination is essential as part of the

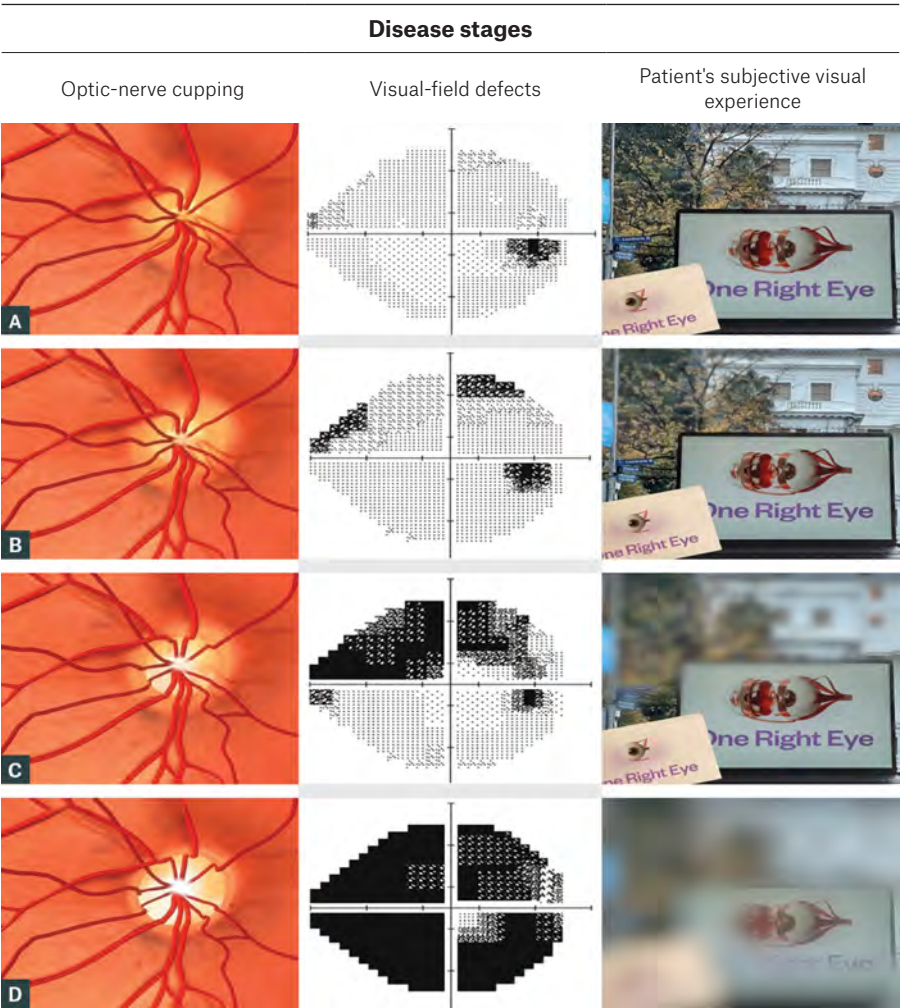


Figure 1. Progressive optic-nerve damage and visual-field loss in glaucoma. Figure shows the relationship between optic-nerve cupping, corresponding visual-field defects, and the patient’s subjective visual experience across disease stages. **(A)** No nerve damage (normal). **(B)** Nerve damage (early glaucoma). **(C)** Glaucoma. **(D)** Advanced glaucoma. Adapted from One Right Eye. One Right Eye, updated 2021 (www.onerighteye.com), with permission from One Right Eye.

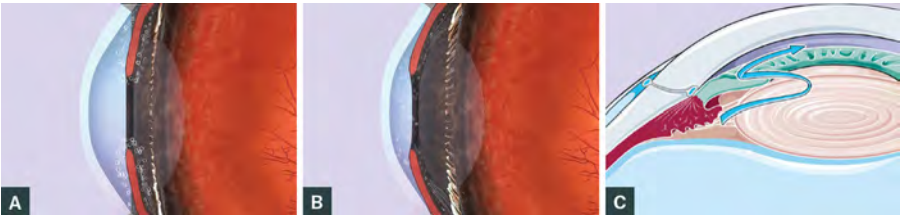


Figure 2. Open and closed anterior chamber angles in glaucoma. Schematic cross-sections illustrating aqueous humour dynamics. **(A)** Shows normal open angle configuration. **(B)** Shows normal closed-angle configuration. **(C)** Shows blockage of the trabecular meshwork and pupillary block mechanism in acute angle-closure glaucoma. **(A)** and **(B)** Adapted from One Right Eye. One Right Eye, updated 2021 (www.onerighteye.com), with permission from One Right Eye. **(C)** Adapted from Wikimedia Commons in accordance with the Creative Commons license (CC-BY-SA-3.0).

GP consultation, with ACG features shown in Figure 3(B). Although Figure 3(B) is reflective of a slit lamp examination, a GP will be able to identify conjunctival infection, corneal oedema and a mid-dilated fixed pupil on physical examination.

Risk stratification

Recent systematic reviews show that the risk of glaucoma increases steadily with age, rising by approximately 7% for each additional year.^{10–12} Individuals aged 60–69 years exhibit a three-fold higher risk compared with those aged 40–49 years.^{10,11} Having a first-degree relative with glaucoma confers an estimated two-point-five-fold elevation in risk.^{10–12}

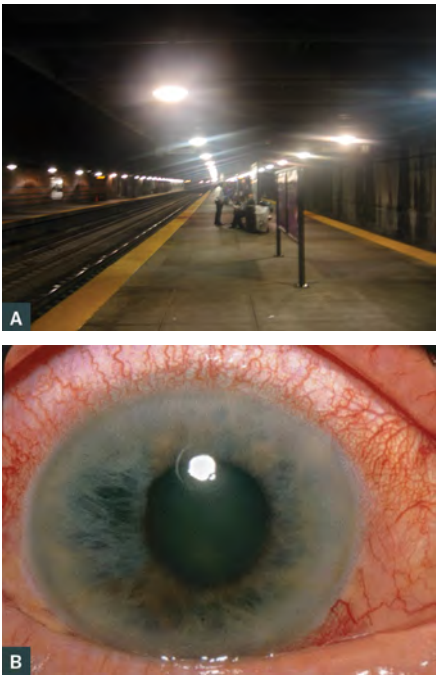


Figure 3. **(A)** Simulated patient view of haloes around streetlights, representing light diffraction from corneal oedema in acute angle-closure (AAC) or markedly elevated intraocular pressure. While not the most common presentation of glaucoma, this symptom warrants urgent ophthalmology referral. **(B)** Slit-lamp photograph showing acute angle-closure glaucoma with conjunctival injection, corneal oedema, and a fixed mid-dilated pupil due to pupillary block of the trabecular meshwork. Adapted from Wikimedia Commons and EyeWiki in accordance with the Creative Commons license (CC-BY-SA-3.0).

Glaucoma incidence is disproportionately elevated among individuals of African and Asian ancestry compared with those of European descent.^{10–12} High myopia and marked hypermetropia also contribute to increased susceptibility for both open-angle and angle-closure subtypes.^{10–12} Diabetes mellitus and chronic corticosteroid use have been identified as significant risk factors in cohort analyses.^{10–12} Furthermore, a history of ocular trauma is associated with higher odds of developing secondary glaucoma.^{10,12} While not always present, ocular hypertension remains the single strongest modifiable clinical risk factor and is useful for screening, with risk rising for each 1 mmHg increment.^{10–12} Male sex is associated with higher risk of POAG and ACG is more frequent in females.^{11,12} Hypertension and barriers to accessing care, such as lower socioeconomic status and limited eye screening, might also contribute to increased risk in some populations.^{10,12}

When to suspect and when to act

Acute angle-closure (AAC) should be suspected in any patient with sudden ocular pain, haloes, blurred vision, nausea or vomiting, and a red eye with a mid-dilated fixed pupil or corneal haze (Table 1). The GP should take a detailed history and perform an examination looking for the described signs. Bilateral involvement is rare in an acute case.⁵

Urgency is key and the GP and clinic team should coordinate same-day ophthalmology or an emergency department referral (Table 2).^{4–6}

Intermittent (prodromal) angle-closure attacks are specifically distinguished by occurrence in dim light and might be accompanied by headaches or periorbital discomfort.^{4,5,13} Like AAC, a history and examination is required as well as considering secondary causes such as trauma, lens-induced, or uveitic angle closure. Referral to same-day ophthalmology or an emergency department is required.⁷

Sudden vision loss, new field defects, or optic-disc haemorrhage signal rapidly progressive open-angle disease and require urgent referral.^{4,5} Do not delay referral in cases presenting with atypical or ambiguous features.⁴

GPs can work collaboratively with community-based optometrists who can measure IOP when this is not available to the GP. In primary care, asymptomatic patients might present to optometry with IOP >35 mmHg, or 25–34 mmHg with suspicious discs or reproducible field defects. These patients should be reviewed by an ophthalmologist within 48 hours.⁴ High-risk individuals with borderline IOP or normal pressure but strong risk factors should still be referred within 1–3 months.⁴ A GP might be required to coordinate care with their community optometry colleagues, including completing the referral to the ophthalmologist.

Where possible, documentation of visual acuity and pupil reactions should be recorded in the referral. If a GP is completing a referral, any optometry recorded information should be provided as part of the referral.^{4,13}

Finally, an important practice point of care for GPs is that if AAC is suspected and no contraindication exists, topical β -blocker (eg Timolol) and oral acetazolamide might be administered while arranging transfer.⁶

Practical examination of the eye in general practice

Clinical assessment should include visual acuity testing and a brief history covering family history of glaucoma, corticosteroid exposure, ocular trauma and prior surgery.^{4,5} Direct ophthalmoscopy might allow a gross assessment of the optic disc, but accurate estimation of the cup-to-disc ratio is often challenging in undilated pupils and should be interpreted cautiously.^{5,13} Findings such as marked asymmetry between eyes or a clearly enlarged cup might still warrant referral. Tonometry is not routinely available in primary care and is not required to guide referral decisions; where prior IOP measurements are available from optometry or hospital records, they might provide supportive context.^{5,6} The oblique flashlight test can be used as a simple screening tool for a shallow anterior chamber and risk of angle closure.^{4,6} While low-cost fundus cameras and smartphone adaptors might facilitate image capture in some settings,

Table 1. Glaucoma classification for general practitioners

Quick reference classification system using contemporary terminology, organised by anatomical mechanism and clinical urgency.

Group	Subtype	Typical presentation	Urgency
Angle-closure glaucoma (ACG)	Acute angle closure (AAC)	Painful red eye, haloes, nausea/vomiting, mid-dilated pupil, IOP >30 mmHg	Same-day ED/ophthalmology
	Primary angle-closure glaucoma (PACG)	Progressive peripheral field loss, shallow anterior chamber	Routine ophthalmology
Primary open-angle glaucoma (POAG)	–	Insidious, asymptomatic, cupping $\pm$ raised IOP	Routine ophthalmology
Normal-tension glaucoma (NTG)	–	Glaucomatous disc/field change with normal IOP	Routine ophthalmology
Secondary glaucomas	Steroid-induced, pseudo-exfoliative, uveitic, pigmentary, traumatic, neovascular	Context-specific features	As per scenario

ED, emergency department; GP, general practitioner; IOP, intraocular pressure.

their availability and accuracy are variable and they require training; they should be viewed as adjuncts rather than standard GP tools.^{4,6} Artificial intelligence-based disc analysis remains investigational and is not recommended for routine clinical use.¹

Coordinating care

GPs have a central role in maintaining continuity of glaucoma care (Box 1). Ensuring uninterrupted access and correct use of topical therapy is essential, as intervals for review with an ophthalmologist might be annual, especially as Pharmaceutical Benefits Scheme (PBS) prescribing now allows up to 12 months' supply for some glaucoma medications under the 60-day dispensing model. Reinforcing drop technique, monitoring side effects and timely script renewal help prevent avoidable disease progression. Common systemic adverse effects include bradycardia, hypotension and bronchospasm with topical β -blockers, particularly in patients with underlying cardiac or respiratory disease; fatigue, dry mouth, drowsiness and delayed allergic conjunctivitis with α -agonists; and paraesthesia, gastrointestinal upset, electrolyte disturbance and metabolic acidosis with carbonic anhydrase inhibitors, especially when oral agents are used. Awareness of these effects will allow GPs to identify intolerance, manage comorbidities safely and prompt timely review with an ophthalmologist.⁶

Box 1. How a general practitioner can assist in caring for patients with glaucoma

- Coordinate and manage – ensure continuity of prescribed topical therapy, address systemic comorbidities and arrange timely review
- Refer rather than defer – any patient with suspected glaucoma (elevated IOP, suspicious disc, relevant symptoms or risk factors) should be referred to an eye care specialist for baseline assessment (ophthalmologist or optometrist), or directly to an ophthalmologist when red-flag criteria are met
- Shared records – copy referral correspondence to both optometrist and ophthalmologist where possible; this facilitates the emerging team-based model and avoids fragmented care

IOP, intraocular pressure.

Shared-care pathways should always retain ophthalmologist oversight, particularly for patients with progression, advanced disease or high-risk profiles. Optometry-performed VF testing serves a screening function but does not replace specialist evaluation.⁴ Reference to The Royal Australian and New Zealand College of Ophthalmologists (RANZCO) Glaucoma Referral Pathway supports appropriate triage and follow-up planning. Clear documentation of diagnosis, referral urgency and recall intervals in correspondence, and liaison with the treating ophthalmologist prior to elective procedures, assists optimisation of cardiovascular and anaesthetic risk factors (Box 2).¹⁴

Finally, patients with a diagnosis of glaucoma will require long-term GP care and follow-up. Care coordination is key and completion of a GP Comprehensive Care Management Plan (GPCCMP) should be considered by the GP and clinic team. Completion of such

a plan will ensure a patient will be provided with optimal, holistic care and ensure comorbidities, medication side effects and interactions are reviewed annually (Figure 4). Patient education is essential in glaucoma care. Use of reputable, freely available visual or digital resources might assist patient understanding, reinforce adherence and support shared decision making in general practice.

Stepwise escalation of glaucoma treatment

Glaucoma is managed in a stepwise fashion, escalating from drops to selective laser trabeculoplasty (SLT), minimally invasive glaucoma surgery (MIGS) and, in advanced cases, incisional procedures (trabeculectomy or tube shunt).^{15–17}

After glaucoma surgery, patients remain under close ophthalmology review, but GPs contribute by reinforcing adherence to

Table 2. When to refer on a patient with suspected glaucoma

Symptom/sign (any one is sufficient)	Likely condition	Action
Severe peri-ocular pain, red eye, haloes, blurred vision, nausea/vomiting PLUS one of steamy cornea, ciliary flush, fixed mid-dilated pupil, IOP ≥ 30 mmHg	Acute angle-closure glaucoma (AACG) ^a	Same-day ophthalmology/ED
Transient brow ache or blurred vision in dim light; haloes around light, nausea/vomiting episodes, shallow anterior chamber on oblique flashlight test	Intermittent angle closure	Urgent referral ≤ 1 week
Sudden drop in VA or new field loss OR fresh optic-disc (Drance) haemorrhage OR vertical CDR ≥ 0.8 or asymmetry ≥ 0.3	Rapidly progressive open-angle glaucoma	Urgent referral ≤ 1 week
IOP ≥ 35 mmHg (even if asymptomatic) OR IOP 25–34 mmHg with suspicious disc or reproducible VF defect	Sight-threatening ocular hypertension	Urgent referral ≤ 48 h
IOP 22–25 mmHg without other risk factors; high-risk patient (age > 60 years, FHx, diabetes, steroid use) even if IOP normal	Possible early POAG/NTG	Routine referral 1–3 months

^aThe criteria combine patient-reported symptoms and exam signs that mandate escalation. Use with The Royal Australian and New Zealand College of Ophthalmologists Glaucoma Referral Pathway;¹⁴ thresholds reflect common consensus triggers for GP action.
CDR, cup-to-disc ratio; ED, emergency department; FHx, family history; GP, general practitioner; IOP, intraocular pressure; NTG, normal-tension glaucoma; POAG, primary open-angle glaucoma; VA, visual acuity; VF, visual field.

Common glaucoma medications – GP quick reference

PBS-listed medications, monitoring requirements and key side effects

PBS listed (general/streamlined)

β-blockers (first-line) - Timolol 0.25%, 0.5% Timoptol BD dosing (or daily with XE formulation) - Betaxolol 0.5% Betoptic BD dosing Local effects - Stinging/burning on instillation - Dry eyes - Corneal anaesthesia Systemic effects - Bradycardia - Hypotension - Fatigue - Depression Contraindications - Asthma - COPD - Heart block - Bradycardia <60 bpm - Decompensated heart failure GP monitoring - Check pulse and BP at routine visits - Review respiratory symptoms - Watch for mood changes Prostaglandin analogues (first-line) - Latanoprost 0.005% Xalatan Once daily (evening) - Bimatoprost 0.03% Lumigan Once daily (evening) - Travoprost 0.004% Travatan Once daily (evening) - Tafuprost 0.0015% Saflutan Once daily (preservative-free)	Local effects - Conjunctival hyperaemia (red eyes) - Eyelash growth/darkening - Iris pigmentation (permanent) - Peri-orbital fat atrophy Systemic effects - Minimal systemic effects - Rare: headache - Rare: flu-like symptoms GP monitoring - Warn patients about potential permanent iris colour change (especially if treating one eye only) and cosmetic changes to eyelashes Alpha-2 agonists - Brimonidine 0.15%, 0.2% Alphagan BD-TDS dosing - Apraclonidine 0.5%, 1% Iopidine TDS (short-term use) Local effects - Allergic conjunctivitis (common) - Follicular conjunctivitis - Ocular dryness - Burning sensation Systemic effects - Dry mouth (very common) - Fatigue/drowsiness - Headache - Hypotension Contraindications - MAO inhibitor use - Infants/young children (risk of CNS depression) - Severe cardiovascular disease GP monitoring - High rate of allergic reactions (up to 25% with brimonidine) – may develop after months of use	Carbonic anhydrase inhibitors - Brinzolamide 1% Azopt BD-TDS dosing - Dorzolamide 2% Trusopt TDS dosing - Acetazolamide (oral) Diamox 250 mg BD-QID (emergency use) Local effects (topical) - Stinging/burning - Bitter taste - Blurred vision - Superficial punctate keratitis Systemic effects (oral > topical) - Paraesthesia (tingling fingers/toes) - Metallic taste - Fatigue/malaise - Hypokalaemia - Metabolic acidosis - Kidney stones Contraindications - Sulfonamide allergy - Severe renal/hepatic impairment - Hypokalaemia - Hyperchloraemic acidosis	GP monitoring (for oral acetazolamide) - Check electrolytes (especially K+) and creatinine if prolonged therapy - Monitor for acidosis symptoms Combination therapies - Dorzolamide/Timolol Cosopt BD dosing - Brinzolamide/Timolol Azarga BD dosing - Brimonidine/Timolol Combigan BD dosing - Latanoprost/Timolol Xalacom Once daily (morning) - Bimatoprost/Timolol Ganfort Once daily - Travoprost/Timolol Duotrav Once daily Important notes - Combines side effects and contraindications of both components - Improves compliance (fewer bottles, less frequent dosing) - Generally reserved for patients not controlled on monotherapy - Cost-effective under PBS when multiple agents needed Key GP reminders - Always check pulse/BP with β-blockers - Warn patients about iris colour changes with prostaglandins - Monitor for allergy with brimonidine - Check sulfa allergy before CAIs - PBS authority required: most glaucoma medications require authority prescription for continuing therapy - Preservative-free options: available for patients with preservative sensitivity (eg Saflutan, Monopost)
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Figure 4. Common glaucoma medications quick reference chart. PBS-listed topical and oral glaucoma medications organised by drug class, with key monitoring requirements and contraindications for general practice. Note the importance of checking for respiratory disease before prescribing β-blockers, warning patients about cosmetic changes with prostaglandin analogues, and monitoring for delayed allergic reactions with α-agonists. Combination therapies improve adherence but carry cumulative side effects from both components.⁶

BD, twice a day; BP, blood pressure; CAIs, carbonic anhydrase inhibitors; CNS, central nervous system; COPD, chronic obstructive pulmonary disease; GP, general practitioner; PBS, Pharmaceutical Benefits Scheme; QID, four times a day; TDS, three times a day; XE, gel-forming solution.

Box 2. Common terms and abbreviations used in ophthalmology

AACG	Acute angle closure glaucoma	NTG	Normal tension glaucoma
ACG	Angle closure glaucoma	OCT	Optical coherence tomography
CACG	Chronic angle closure glaucoma	PACG	Primary angle closure glaucoma
CCT	Central corneal thickness	PBS	Pharmaceutical Benefits Scheme
CDR	Cup to disc ratio	POAG	Primary open angle glaucoma
ED	Emergency department	RANZCO	Royal Australian and New Zealand College of Ophthalmologists
FHx	Family history	RNFL	Retinal nerve fiber layer
GP	General practitioner	RVEEH	Royal Victorian Eye and Ear Hospital
IOP	Intraocular pressure	VA	Visual acuity
IV	Intravenous		

prescribed post-operative drops, reviewing systemic medications that might influence IOP, and recognising warning symptoms such as ocular pain, redness or blurred vision that require urgent re-referral. While SLT and MIGS have short recovery periods, trabeculectomy and tube procedures demand longer follow-up; GPs can confirm ongoing use of prescribed eye drops, support management of comorbidities such as diabetes and hypertension, and ensure timely communication with the treating ophthalmologist.

Conclusion

Glaucoma remains a leading yet under-recognised cause of irreversible vision loss in Australia, and general practice is uniquely positioned to shift that trajectory. The GP’s contribution lies less in diagnosis than in vigilance, coordination and continuity of care. Reinforcing adherence to topical therapy, ensuring uninterrupted PBS-supported supply, monitoring for systemic side effects and maintaining a low threshold for re-referral are practical, high-yield actions. Embedded within a shared-care framework, these steps offer the most realistic path to preventing avoidable blindness in the Australian community.

Key points

- Recognise and act on angle-closure red flags immediately.
- Think progression, not pressure: IOP is a surrogate marker.
- Maintain continuous medication supply: renewal of scripts every 6 months is critical for stable annual-review patients.

- Screen for steroid exposure (all routes) and history of ocular trauma.
- Support drop adherence: prescribe, teach technique and monitor side-effects.
- GPs should consider having an established relationship with an ophthalmologist in their region so that urgent referrals and advice can be sought for eye conditions including glaucoma.

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