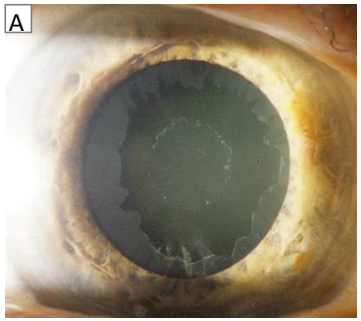
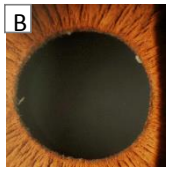
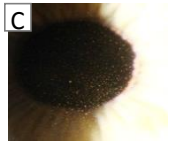
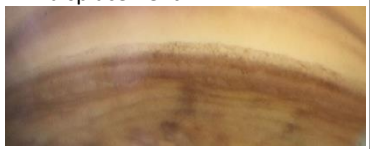
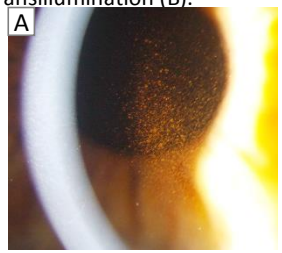
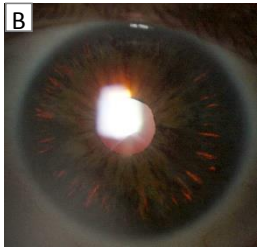
Centre for Eye Health

CHAIR-SIDE REFERENCE: SECONDARY GLAUCOMAS

Secondary glaucomas occur when there is an ***identifiable*** cause of impaired trabecular meshwork outflow, other than apposition of the iris to the posterior cornea, causing periodic or constant intraocular pressure (IOP) elevation. Where there is suspicion for glaucoma, it is important to closely investigate for signs or history suggestive of secondary glaucomas, as this will guide appropriate management. For more information about assessing the anterior chamber angle, please see the **gonioscopy and anterior chamber angle chair-side reference.**
This chair-side reference is not exhaustive for all types of secondary glaucomas. Please note that the anterior eye signs below may not always present with glaucomatous optic nerve head changes, in which case these would indicate a relatively higher risk of developing glaucoma in future.

TRABECULAR MESHWORK OUTFLOW OBSTRUCTED (BY PARTICLES OR DEGENERATION)

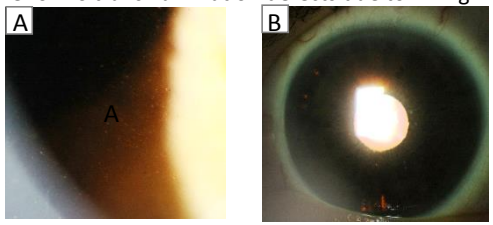
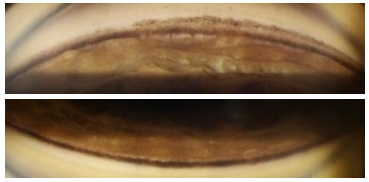
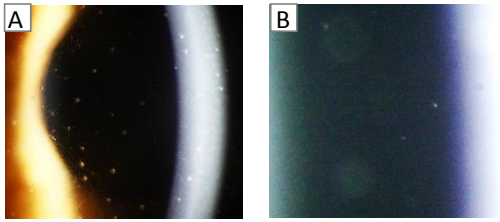
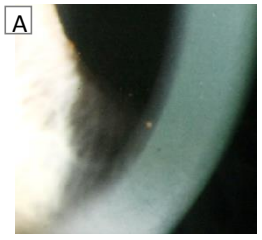
Slitlamp Findings	Gonioscopy Findings	IOP Findings	Description and Clinical Considerations
Pseudoexfoliation Syndrome (PXF)			
White dandruff-like deposits at pupillary margin and on the anterior lens (A). May have associated pupillary ruff atrophy and transillumination (B) and scattered pigment and/or PXF deposits on endothelium (C)   	• Patchy trabecular meshwork pigmentation • Pigmentation at Schwalbe's line also common (Sampaolesi's line) • May also have dandruff-like deposits in the angle • Angles tend to be narrower due to weakened zonules and anterior crystalline lens displacement 	• Within normal range or elevated • Asymmetric IOPs possible with unilateral presentation: PXF eye will have higher IOP	• Deposition of PXF material and iris pigment (released due to interaction between pupillary ruff and anterior lens capsule) within the trabecular meshwork increases IOP • Bilateral condition but may have asymmetric manifestation • Dilation often required for diagnosis, however pupil reactions may be sluggish and dilate poorly • Consider earlier cataract referral • Encourage routine cardiovascular reviews with GP • Increased prevalence with age. Higher prevalence in Scandinavian and Greek populations *No glaucoma → ongoing monitoring as glaucoma suspect (6-monthly IOP checks. Consider IOP phasing with borderline or asymmetric IOPs* *Glaucoma present, IOP ≥24mmHg and/or IOP fluctuations >4mmHg with applanation tonometry → treatment likely indicated*
Pigment Dispersion Syndrome (PDS)			
Krukenberg's spindle: dense endothelial pigment in a vertical pattern inferonasal (A). Radial spoke-like mid-peripheral iris transillumination (B).  	Homogeneously dense trabecular meshwork pigmentation Posteriorly bowed or 'concave' mid-peripheral iris  	• Within normal range or elevated • Strenuous exercise may cause IOP spikes	• Deposition of iris pigment, released due to interaction with posteriorly displaced iris and lens zonules, within the trabecular meshwork increases IOP • Bilateral but may be asymmetric • Patients tend to be younger (20s to 40s). Associations with myopia and Caucasian ethnicity. *No glaucoma → ongoing monitoring as glaucoma suspect required. Consider IOP phasing with borderline or asymmetric IOPs* *Glaucoma present, IOP ≥24mmHg and/or IOP fluctuations >4mmHg with applanation tonometry → treatment likely indicated*

This reference is designed a guide to aid diagnosis and management decisions however individual cases must be assessed in the context of all available clinical data. For personalised clinical support or advice, please make a free telehealth appointment with one of the CFEH Senior Staff Optometrists.

Centre for Eye Health

CHAIR-SIDE REFERENCE: SECONDARY GLAUCOMAS

TRABECULAR MESHWORK OUTFLOW OBSTRUCTED (BY PARTICLES OR DEGENERATION)

Slitlamp Findings	Gonioscopy Findings	IOP Findings	Description and Clinical Considerations
Burnt-Out Pigment Dispersion Syndrome (PDS)			
May be as per active PDS or pigment less dense (A) Fewer or no transillumination defects due to 'filling in' (B) 	Pigment reversal sign: superior angle shows heavier pigmentation than inferior angle 	Within normal range	• Inactive version of PDS where there is no pigment liberation • Typically seen in older patients (>50 years old) due to increased lens thickness with age. • If glaucoma is present, less likely to demonstrate progression as IOP spikes unlikely *Ongoing monitoring as glaucoma suspect required if not already on treatment*
Acute Uveitic Glaucoma			
Keratic precipitates at the corneal endothelium Anterior chamber cells and/or flare 	Peripheral anterior synechiae and increased trabecular meshwork pigmentation may be visible 	Elevated	• Deposition of inflammatory cells into the trabecular meshwork and/or acute trabeculitis increases IOP • Typically occurs in herpetic and toxoplasmosis-related acute anterior uveitis • Unilateral or bilateral • Asymptomatic or displays uveitis symptoms (severity dependent) including sore, painful red eye with associated photophobia and blurred vision *Treat underlying uveitis in conjunction with topical glaucoma treatment (not prostaglandin analogues as pro-inflammatory)* *Referral to or collaboration with ophthalmologist required*
Chronic Uveitic Glaucomas, including Fuchs' Uveitis Syndrome (FUS) and Posner-Schlossman Syndrome (PSS)			
Mild anterior uveitis signs - A/C cells and flare, few keratic precipitates (A). FUS (also known as Fuchs' heterochromic iridocyclitis) shows iris heterochromia. May also show iris atrophy and patchy transillumination 	Open, unremarkable trabecular meshwork findings	FUS: within normal range or mildly elevated PSS: elevated to >40mmHg during an acute attack, IOP typically normal in between episode	• Recurrent attacks of acute trabeculitis associated with mild anterior uveitis acutely raises IOP • PSS is unilateral, FUS is typically unilateral (13% bilateral) • Will display mild uveitis symptoms during attack (as above) • Glaucoma development more common with chronic uveitis compared to acute uveitis *Treat underlying uveitis in conjunction with topical glaucoma treatment (not prostaglandin analogues as pro-inflammatory)* *Referral to or collaboration with ophthalmologist required*

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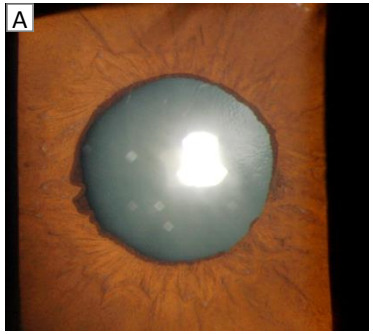
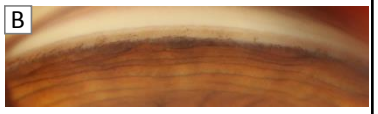
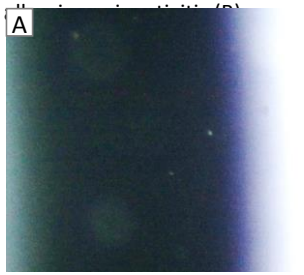
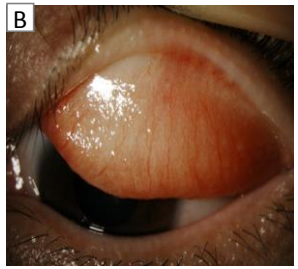
CHAIR-SIDE REFERENCE: SECONDARY GLAUCOMAS

TRABECULAR MESHWORK OUTFLOW OBSTRUCTED BY PARTICLES OR DEGENERATION

Slitlamp Findings	Gonioscopy Findings	IOP Findings	Description and Clinical Considerations
Angle Recession			

Unremarkable or signs of previous blunt trauma
Distortion of pupil shape and irregular pupillary ruff (A)
 
A
	Irregular widening of the ciliary body. Record extent of recession. May show mottled pigmentation at trabecular meshwork and/or Schwalbe's line
	 
	B
	 
	C
		Within normal range or elevated	• Previous blunt trauma causes ciliary body rupture, and associated trabecular meshwork degeneration reduces aqueous outflow
			• Unilateral, affecting the eye with previous blunt trauma, however up to 50% will develop glaucoma in uninjured fellow eye
			• Peak incidences of angle recession glaucoma are 3 to ≥10 years after blunt trauma
			• Risk factors for development of angle recession glaucoma
			  • Presence of hyphema at time of ocular injury
			  • Angle recession >180°
			  • Lens displacement
			  • Increase trabecular meshwork pigmentation
			
			No glaucoma → ongoing monitoring as glaucoma suspect required
			Glaucoma present and/or IOP ≥24mmHg → Referral to or collaboration with ophthalmologist required

Steroid-Induced Glaucoma			

Unremarkable unless associated anterior or posterior eye pathology requiring topical steroid use
Associated conditions include Anterior uveitis (A) and
 
A
 
B
	Unremarkable or related to anterior eye pathology requiring topical steroid use	Elevated, responses can be stratified by increase in IOP:
		• Low: <6mmHg
		• Moderate: 6-15mmHg
		• High: >15mmHg
			• Steroid medications causing increased resistance to outflow within the trabecular meshwork -> IOP elevation.
			• Likelihood of IOP spike associated with:
			  1. *Administration route:* Intravitreal > periocular (subconjunctival sub-tennon or retrobulbar) > topical > systemic
			  2. *Steroid potency*: Strongest association with Dexamethasone > Prednisolone > fluoromethalone
			  3. *Dosage*
			  4. *Duration*
			• Often observed days to weeks after commencing steroid treatment, however rare cases showed delayed IOP increase
			• Increased risk of steroid response if existing glaucoma/OHTN, FOHx of glaucoma, high myopia, younger age and connective tissue disease (e.g.rheumatoid arthritis)
			
			Discontinuation of steroid medication if appropriate
			• *Liaise with prescribing practitioner as required*
			Referral or collaboration with ophthalmologist required if
			• *Steroid medication cannot be ceased immediately*
			• *IOP >24mmHg or Glaucoma present*

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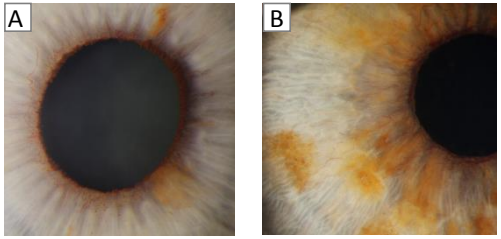
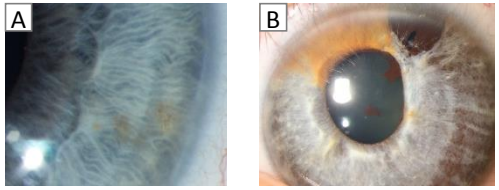
CHAIR-SIDE REFERENCE: SECONDARY GLAUCOMAS

TRABECULAR MESHWORK OUTFLOW OBSTRUCTED BY MEMBRANE (PRE-TRABECULAR)

Slitlamp Findings	Gonioscopy Findings	IOP Findings	Description and Clinical Considerations
Iris Neovascularisation (Rubeosis Irides)			

Early: neovascularisation on superficial pupillary margin (A)
Later: spreads radially towards angle (B)
 
	• Early angle neovascularisation visible as fine blood vessels traversing across the angle
	• Peripheral anterior synechiae in later stages
		Normal in early stages
		
		Elevated in later stages with circumferential angle closure
			• Retinal ischaemia causes release of VEGF into the vitreous, inducing development of new vessels at the superficial iris
			  • Most commonly associated with ischaemic retinal vein occlusions and proliferative diabetic retinopathy. Also referred to as "90 day glaucoma"
			  • Abnormal iris vessel growth can occur weeks to years after the initial ischaemic event
			• Asymptomatic in early stages, but in later stages high IOP causes symptoms (sore, painful red eye, blurred vision if associated corneal oedema)
			
			Prompt referral to ophthalmologist required. Ideally, management of ischaemia at the initial episode to help reduce the risk of iris neovasculisation.

Axenfeld-Rieger Syndrome			
Prominent posterior embryotoxon, peripheral iris strands may be visible (A). Rieger anomaly: additional iris atrophy, abnormal pupil shape and ectropian uveae (B) 	• Axenfeld anomaly: peripheral iris strands adherent to posterior cornea • Peripheral anterior synechiae may also be present	Within normal range or elevated	• Congenital spectrum of conditions where anomalies in angle structure and/or adhesions of peripheral iris to angle result in IOP elevation • Rieger syndrome: associated with developmental dental (few, small teeth) and facial (maxillary hypoplasia, broad nasal bridge, telecanthus and hypotelorism) anomalies • Positive family history often present due to autosomal dominant inheritance • Bilateral but can present asymmetrically *Referral to ophthalmologist required*

INCREASE IN IOP RELATED TO THE CRYSTALLINE LENS (LENS-RELATED GLAUCOMA)

Phacolytic Glaucoma			
Hypermature cataract, large white particles floating in the anterior chamber, pseudohypopyon, conjunctival hyperaemia. Corneal oedema.	• Open with absence of peripheral anterior synechiae	Typically very elevated	• **Open-angle** glaucoma. Trabecular meshwork obstructed due to leakage of lens proteins through the lens capsule of a hypermature cataract • Acute onset of pain and poor vision. *Urgent referral to ophthalmologist*
Phacomorphic Glaucoma			
Similar clinical appearance to angle closure, however with a dense white cataract, Corneal oedema.	• Closed angle	Very elevated	• Secondary **closed-angle glaucoma. S**wollen or intumescent mature cataract causes pupillary block and iris bombe -> severe pain, blur, photophobia. • Management similar to acute angle closure *Urgent referral to ophthalmologist*

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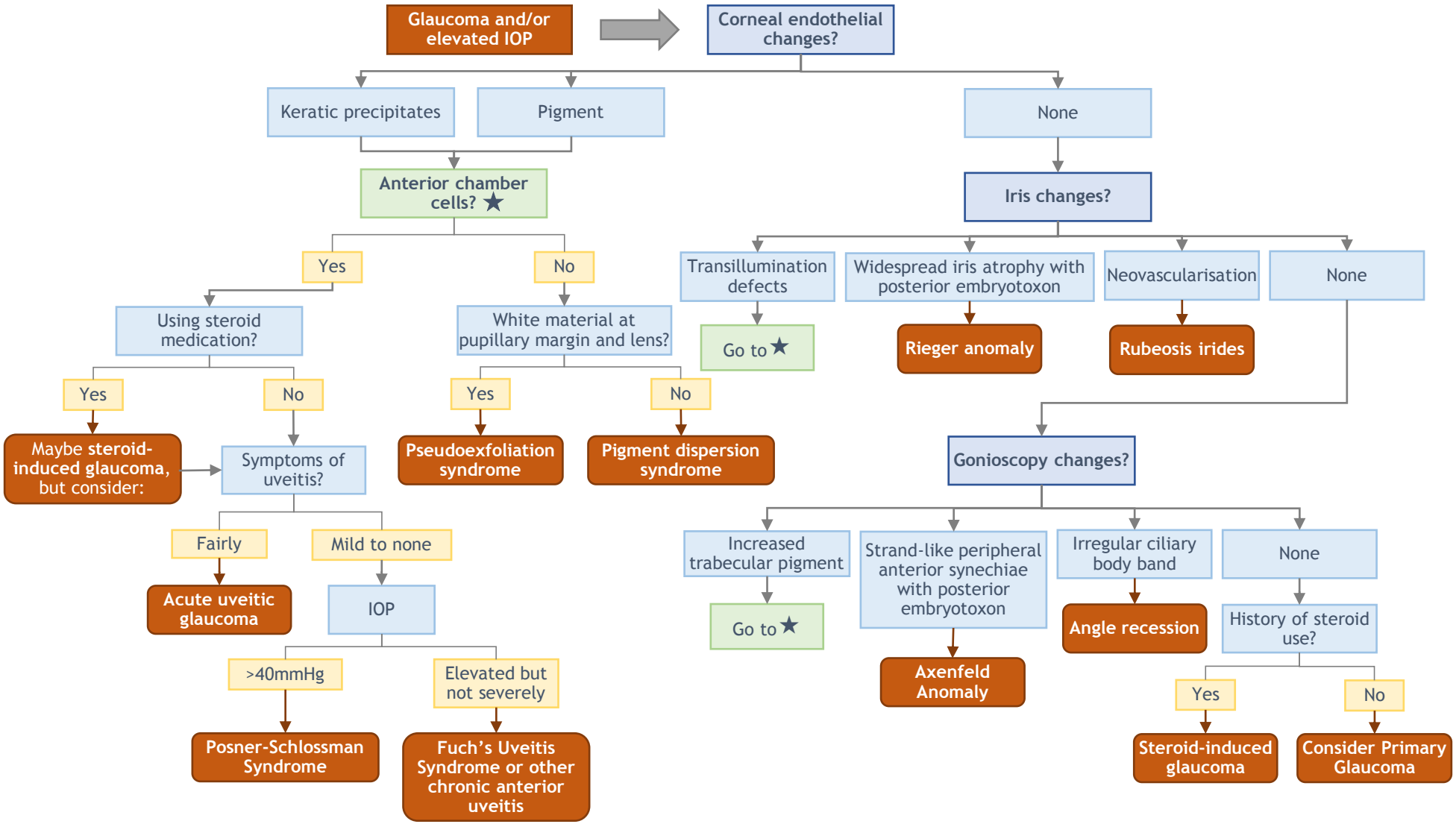
Differential Diagnosis Flowchart



This reference is based on the current literature and evidence at the time of writing. This reference is designed a guide to aid diagnosis and management decisions however individual cases must be assessed in the context of all available clinical data.