

Second to None: A Deep Dive into Secondary Glaucomas

1 hour

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Description

This talk provides a focused overview of secondary glaucomas, highlighting their diverse etiologies, pathophysiology, and clinical management. Attendees will gain insight into diagnostic challenges and tailored treatment approaches for glaucomas secondary to pseudoexfoliation, pigment, trauma, inflammation, and neovascularization. Case-based discussions will illustrate key decision-making strategies.

Objectives

- Identify/differentiate the types and underlying causes of secondary glaucomas including pseudoexfoliative, pigmentary, uveitic, steroid-induced, neovascular and trauma
- Understand the pathophysiology and systemic associations underlying various forms of secondary glaucoma to enhance interdisciplinary care
- Determine appropriate treatment and when to refer for co-management or surgical intervention

I. Introduction

- Brief overview of primary vs. secondary glaucomas
- Importance of recognizing secondary glaucomas in routine optometric care
- Goals of early detection and collaborative management

II. Pseudoexfoliative Glaucoma

Pathophysiology

- Accumulation of fibrillar pseudoexfoliative material in anterior segment structure
- Associated with increased outflow resistance in the trabecular meshwork

Clinical Pearls

- Look for flaky deposits on lens capsule, pupillary ruff, and angle
- Often unilateral/asymmetric in early stages

Treatment

- Often more aggressive course than POAG
- IOP-lowering therapy: prostaglandins, beta-blockers, laser trabeculoplasty (high success)
- Monitor for rapid progression and poor pupil dilation during surgery

III. Pigmentary Glaucoma

Pathophysiology

- Posterior bowing of the iris rubs pigment off the posterior iris surface
- Pigment disperses into aqueous, clogging trabecular meshwork

Clinical Pearls

- Mid-peripheral iris transillumination defects
- Dense trabecular meshwork pigmentation
- Krukenberg spindle

Treatment

- Miotics (e.g., pilocarpine) to reduce iris-zonule contact (limited by side effects)
- Prostaglandin analogs and beta-blockers
- Consider laser peripheral iridotomy in younger patients to flatten iris configuration

IV. Uveitic Glaucoma

Pathophysiology

- Inflammatory cells and debris block trabecular outflow
- Possible synechiae formation, steroid-induced IOP spikes

Clinical Pearls

- Chronic or recurrent anterior uveitis
- May present with synechiae, flare, keratic precipitates, and fluctuating IOP

Treatment

- Treat underlying inflammation (topical/systemic corticosteroids, immunomodulators)
- IOP control: aqueous suppressants preferred (avoid prostaglandins if active inflammation)
- Careful steroid use to avoid worsening IOP

V. Neovascular Glaucoma

Pathophysiology

- Retinal ischemia (e.g., diabetic retinopathy, CRVO) → VEGF release → neovascularization of iris and angle → synechial angle closure

Clinical Pearls

- Early signs: rubeosis iridis at pupillary margin
- Gonioscopy: new vessels crossing scleral spur, fibrovascular membranes

Treatment

- Urgent retinal evaluation and anti-VEGF therapy
- Panretinal photocoagulation (PRP)
- IOP control: cycloplegics, aqueous suppressants, surgical options (tube shunt, cyclodestruction)

VI. Traumatic Glaucoma

Pathophysiology

- Blunt trauma: angle recession, hyphema, trabecular damage
- Penetrating trauma: scarring, inflammation

Clinical Pearls

- Look for angle recession on gonioscopy
- Monitor for delayed IOP elevation months to years post-trauma

Treatment

- Aqueous suppressants
- Avoid prostaglandins in active hyphema
- Long-term follow-up for late-onset glaucoma

VII. Steroid-Induced Glaucoma

Pathophysiology

- Corticosteroids reduce aqueous outflow via ECM changes in trabecular meshwork
- May mimic POAG in appearance

Clinical Pearls

- Can occur with topical, periocular, inhaled, or systemic steroids
- IOP elevation may begin within weeks

Treatment

- Discontinue or taper steroid if possible
- Use steroid-sparing alternatives (e.g., loteprednol)
- IOP-lowering therapy: topical aqueous suppressants, laser trabeculoplasty if persistent

VIII. Questions and Discussion