Secondary Glaucomas

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Learning Objectives

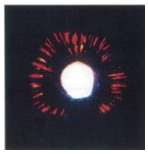
- Classify secondary glaucomas by their cause
- Outline the components of evaluation for secondary glaucomas
- Distinguishes the epidemiology, characteristics, clinical findings and management of pigment dispersion syndrome and pigmentary glaucoma
- Distinguishes the epidemiology, characteristics, clinical findings and management of pseudoexfoliation syndrome and pseudoexfoliation glaucoma
- Distinguishes the epidemiology, characteristics, clinical findings and management of steroid-induced glaucoma
- Distinguishes the epidemiology, characteristics, clinical findings and management of traumatic glaucoma
- Distinguishes the epidemiology, characteristics, clinical findings and management of neovascular glaucoma
- Distinguishes the including epidemiology, characteristics, clinical findings and management of post-operative glaucoma
- Understand the characteristics, clinical findings and management: inflammatory glaucoma, Possner Shlossman glaucoma, phacolytic glaucoma, ghost cell glaucoma, secondary angle closure glaucoma, iridocorneal endothelial syndromes

What is "Secondary Glaucoma"

- Glaucoma that occurs as a result of another condition
- Diagnosis of Inclusion
  - Primary open angle glaucoma is diagnosis of EXclusion-you have excluded all of the secondary glaucomas and other conditions
- Almost entirely anterior segment conditions
  - →elevate IOP

In this lecture:

- Epidemiology
- Characteristics
- Clinical findings
- Management
- Focus on specific conditions:
  - Pigmentary
  - Pseudoexfoliation
  - Steroid induced
  - Traumatic
  - Neovascular
  - Post-operative
- Brief info on other conditions



Epidemiology

- Secondary glaucomas (as a whole) account for 7-35% of all glaucomas*
- On average, lower age of onset
- Dallas study 2011 (Kooner et al)
  - Post-surgical most common
- India study 2008 (Gadia et al)
  - Causes: 1. post-vitrectomy 2. trauma 3. corneal pathology 4. aphakia 5. neovascular 6. pseudophakia 7. steroid-induced 8. uveitic

*Gadia et al. 2008 (22%), Das et al. 2001 (7%), Teikari and O'Donnell 1989 (33%), Qureshi et al. 2006 (35%), Kooner et al 2011 (7%-US)

Secondary Glaucomas

- Glaucomas associated with intraocular tumors
  - Malignant melanoma
  - Retinoblastoma
  - Metastatic carcinoma
  - Leukemias and lymphomas
  - Benign tumors
- Glaucomas associated with elevated episcleral venous pressure
- ***Glaucomas associated with inflammation***
  - Glaucomas associated with uveitis
  - Glaucomas associated with keratitis, episcleritis, scleritis
- ***Steroid-induced glaucoma*** **
- ***Glaucomas associated with ocular trauma*** **
- ***Glaucomas associated with hemorrhage***
- Glaucomas following intraocular surgery
  - ***Ciliary block (malignant) glaucoma***
  - Glaucomas in pseudophakia and aphakia
  - Epithelial, fibrous, and endothelial proliferation
  - Glaucomas associated with corneal surgery
  - Glaucomas associated with vitreoretinal surgery

Courtesy of Jasmine Yumori, OD.

Secondary Glaucomas

- Glaucomas associated with disorders of the corneal endothelium
  - ***Iridocorneal endothelial syndrome***
  - Posterior polymorphous dystrophy
  - Fuchs' endothelial dystrophy
- Glaucomas associated with disorders of the iris and ciliary body
  - ***Pigmentary glaucoma*** **
  - Iridoschisis
  - Iris and ciliary body cysts
  - Aniridia
- Glaucomas associated with disorders of the lens
  - ***Pseudoexfoliation syndrome*** **
  - Phacolytic
  - Lens particle
- Glaucomas associated with disorders of the retina, choroid, and vitreous
  - ***Neovacular glaucoma*** **
  - Glaucomas associated with retinal detachment and vitreoretinal abnormalities

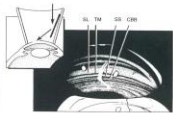
Courtesy of Jasmine Yumori, OD.

Evaluation of Secondary Glaucomas

- History
  - OCULAR SURGERY
  - TRAUMA
  - Ocular pathology
  - Steroid use
- Eye health exam
  - Specifically look at:
    - Cornea (Krukenberg spindle)
    - Anterior chamber (cells/flare)
    - Iris (transillumination defects)
    - Lens (double ring sign)
    - Optic nerve head

Evaluation of Secondary Glaucomas

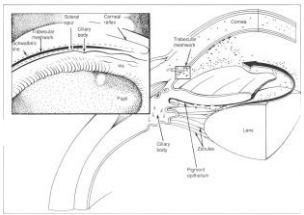
- Gonioscopy
  - Crucial for most secondary glaucor
  - Pigment in angle
  - Neovascularization
  - Recession
  - Lens particles



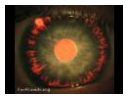
PIGMENTARY GLAUCOMA

Epidemiology

- 1-2.5% prevalence
- Bilateral and asymmetric
- Onset 20-40 years
- Not genetic
- Association with
  - Myopia
  - Males
  - Caucasians



Characteristics

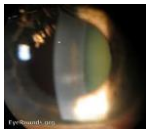


- Posterior part of iris rubs on zonules
- Release of pigment from the iris
  - Pigment is then carried through the aqueous to the anterior chamber→deposits on corneal endothelium and trabecular meshwork
  - Midperipheral transillumination defects
  - Pigment also deposits on lens capsule and zonules
- Doesn't always form glaucoma-high risk
- May report IOP spike after exercise/exertion

PIGMENTARY GLAUCOMA

Clinical findings

- Hallmark:
  - Iris transilliumination
  - Pigment on cornea (Krukenberg spindle)
  - Pigment on lens capsule
  - Deep anterior chamber
  - Pigment in angle on gonioscopy
  - Large IOP fluctuations
- Occasional:
  - Pigment dusting on iris



PIGMENTARY GLAUCOMA

PDS vs. Pigmentary glaucoma

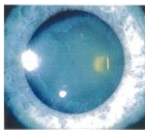
- Pigment dispersion syndrome:
  - Shows clinical findings specific to pigment dispersion
  - No signs of glaucoma
    - IOP not elevated
    - C/D normal
    - No VF defects

PIGMENTARY GLAUCOMA

Management

- Generally, same as POAG
- Respond well to SLT/ALT

PSEUDOEXFOLIATION



PSEUDOEXFOLIATION GLAUCOMA

Epidemiology

- 3-10% of glaucoma in US, 75% in Sweden
- Onset 50-70 years
- Starts unilateral, then bilateral
- Probably partially genetic
- Association with
  - Slightly more common in females
  - Caucasians (though lots in Peru)

PSEUDOEXFOLIATION GLAUCOMA

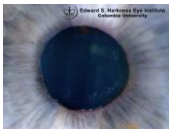
Characteristics

- Protein like material accumulates in the eye
- Material is deposited on the lens, iris ciliary epithelium and trabecular meshwork
  - Obstructs outflow
- Doesn't always form glaucoma-high risk

PSEUDOEXFOLIATION GLAUCOMA

Clinical findings

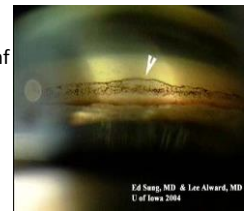
- Hallmark:
  - Double ring sign on lens
  - White flakey material on iris pupillary frill
  - Iris transilliumination at pupillary frill
  - Pigment in angle
  - Increased IOP (often high)
    - Greater diurnal fluctuations
- Occasional:
  - White flakey material on cornea
  - Narrow anterior chamber



PSEUDOEXFOLIATION GLAUCOMA

Clinical findings

- Gonioscopy:
  - Pigment in angle
    - Sampaolesi line: anterior to Schwalbe's line, especially inf
    - TM: especially inf
  - Narrow angles-especially inf



PSEUDOEXFOLIATION GLAUCOMA

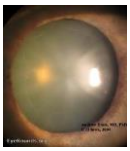
PXS vs. Pseudoexfoliation glaucoma

- Pseudoexfoliation syndrome
  - Shows characteristics of pseudoexfoliation:
    - Double ring sign on lens
    - White flakey material on iris pupillary frill
    - Iris transilliumination at pupillary frill
    - Pigment in angle
    - Narrow angle
  - No signs of glaucoma
    - IOP not elevated
    - C/D normal
    - No VF defects

PSEUDOEXFOLIATION GLAUCOMA

Management

- Generally, same as POAG
  - SLT/ALT very successful, but for shorter time
- Prognosis often worse than POAG



STEROID INDUCED

STEROID INDUCED GLAUCOMA

Epidemiology

- Significant use of corticosteroids
- Onset varies
  - Rarely within hours, sometimes after years of use
- Starts unilateral, then bilateral
- Genetic predisposition to response
- Association with
  - Patients with POAG are more likely to have steroid response
  - Patients with diabetes
  - High myopes

STEROID INDUCED GLAUCOMA

Characteristics

- Mechanism unknown
  - Increased resistance to trabecular outflow→outflow facility is lowered
- Doesn't always cause glaucoma-high risk
- Depends on steroid administration
  - Ophthalmic, Dermatologic, Intranasal, Inhaled, Oral, IV
  - Proportional to the type of administration, dosage, potency and duration
  - Low liklihood with:
    - flurometholone, rimexolone, medrysone or loteprednol

STEROID INDUCED GLAUCOMA

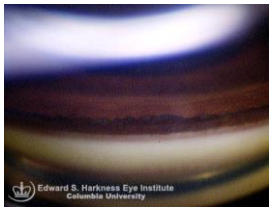
Clinical findings

- Hallmark:
  - History of steroid use
- Otherwise, looks like POAG
- Which came first? POAG or steroid use…

STEROID INDUCED GLAUCOMA

Management

- Stop steroid or decrease if possible
- Switch to "safer" steroid/non-steroidal
- Communicate with prescribing physician
- Treat as POAG
- Laser trabeculectomy is less effective



TRAUMATIC GLAUCOMA

TRAUMATIC GLAUCOMA

Characteristics-Types

- Early:
  - Hyphemic glaucoma
  - Acute trabecular injury
- Late:
  - Angle Recession glaucoma
- Unilateral (unless trauma to both eyes)

TRAUMATIC GLAUCOMA

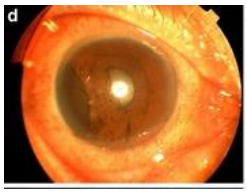
Early Traumatic Glaucoma

- Hypemia
  - Can occur up to 7 days after injury
  - Glaucoma most linked to rebleeding
  - Glaucoma from RBC accumulation in trab
- Acute trabecular injury
  - Iris sphincter tear
  - Cyclodialysis
  - Rare glaucoma

TRAUMATIC GLAUCOMA

Iridodialysis

- Disinsertion of the iris from the scleral spur
  - Can lead to
    - →damage to the TM
    - →Formation of peripheral anterior synechia
- S: asymptomatic, glare, monocular diplopia
- Evaluation:
  - Slit lamp exam
  - IOP check
- Management:
  - Monitor for glaucoma
  - Colored contacts



Late Traumatic Glaucoma

- Angle Recession
  - Sloooow onset: 10-20 years after injury
  - Glaucoma more likely with 2/3-3/4 of angle damage
- Other than gonioscopic appearance, presents like POAG
  - Treatment differences:
    - Miotics contraindicated
    - ALT rarely effective

NEOVASCULAR GLAUCOMA

NEOVASCULAR GLAUCOMA

Epidemiology

- Predisposing factors:
  - Any condition that have affect on retinal vasculature:
    - Diabetes, hypertension, carotid occlusive disease
- Association with
  - 1/3 Diabetic retinopathy
  - 1/3 Central retinal vein occlusion
    - 30% of CRVO develop neovascular glaucoma
    - Often develops 90 days after event (though range is 2 weeks to 2 years)
  - 1/3 Other

NEOVASCULAR GLAUCOMA

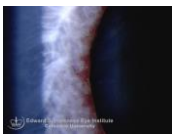
Characteristics

- Hypoxia to the eye causes vasoproliferation
- New blood vessels develop in the iris and trabecular meshwork →form fibrovascular membrane
- Anterior synechia form from contracture of blood vessels in the trab → **secondary angle closure**
- Iris neovascularization does not always develop into neovascular glaucoma
  - Can be treated before glaucoma develops

NEOVASCULAR GLAUCOMA

Clinical findings

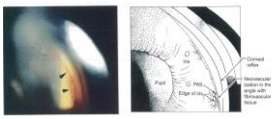
- Hallmark:
  - Neovascularization at iris pupil frill
  - Neovascularization in the angle
- Occasional:
  - Mild anterior cells and flare
  - Hyphema (NOTE: this condition is not hemolytic glaucoma)
  - Corneal edema from extremely high IOP



NEOVASCULAR GLAUCOMA

Management

- Reduce inflammation and pain
  - Topical steroid and cycloplegic
- Reduce IOP
  - Avoid prostaglandins
- URGENT REFERRAL FOR PRP
- Prognosis
  - Often bad
    - Poor vision
    - Painful eye



POST-OPERATIVE

POST-OPERATIVE

Epidemiology

- Increased IOP after surgery
- Actually more post-operative ocular hypertension

POST-OPERATIVE

Etiology

- Likely after retinal surgery
  - Etiology:
    - Inflammation
    - Hyphema
    - Anterior displacement of ocular structures
- After cataract surgery:
  - IOP increases 1 hour after surgery, normal 1 week
  - Etiology:
    - Retained viscoelastic material/lens particles
    - Pupillary block
    - Hyphema
    - Pigment dispersion
    - Inflammation

POST-OPERATIVE

Management

- Treat based on findings
  - Pupil block: PI
  - Inflammation: topical steroid
  - IOP lowering medications
    - Avoid prostaglandin analogs

RARE SECONDARY GLAUCOMAS

RARE

Inflammatory

- IOP increase with inflammation
- Differential:
  - Posner-Schlossman
  - Steroid-response
  - Pigmentary glaucoma
  - Neovascular glaucoma
  - Fuchs heterochromic iridocyclitis

RARE

Inflammatory

- Epidemiology
  - Patients with systemic inflammatory disease
  - Patients with previous corneal disease
  - Post-surgical patients
- Characteristics
  - Glaucoma findings AND inflammatory findings
    - Uveitic=before steroid starts
    - Steroid induced=after steroid starts

RARE

Inflammatory

- Management
  - Treat inflammation
  - Treat glaucoma
    - AVOID prostaglandin analogs

RARE

Possner Shlossman

- AKA Glaucomatocyclitic crisis
- Glaucoma and mild uveitis
- Glaucoma-secondary to trabeculitis
- Recurrent, unilateral

RARE

Possner Shlossman

- Epidemiology
  - Very rare
  - Young adults (male)
  - HLA-B27 positive
- Characteristics
  - Mild to no pain
  - May have halos

RARE

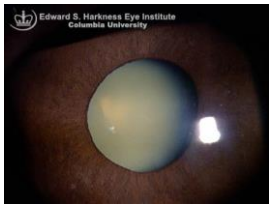
Possner Shlossman

- Clinical Findings
  - Very high IOP (70's)
  - Eye white/minimally injected
  - Mild corneal edema
  - Mild uveitis
- Management
  - Topical steroids/NSAIDS
  - IOP lowering
  - Pain management if needed

RARE

Phacolytic

- Leakage of lens material
- Hallmark: mature cataract, severe pain, white material in anterior chamber



RARE

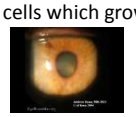
Ghost cell

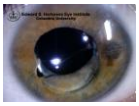
- Intraocular hemorrhage, RBC degenerate and block trabecular meshwork

RARE

Secondary angle closure

- Iridocorneal endothelial
  - 3 conditions: Chandler, essential iris atrophy, iris nevus (Cogan Reese)
  - Abnormal corneal endothelial cells which grow into angle
- Ciliary block
  - Usually post-surgical
  - Shallow AC with patent PI





RARE

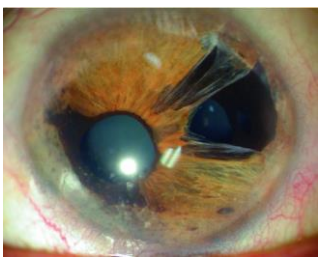
Corneal endothelial disorders

- ICE syndromes (iridocorneal endothelium)
  - Group of disorders
  - Causes variable degree of iris atrophy, corneal edema and secondary angle closure
- Types
  - Chandler syndrome
  - Essential/progressive iris atrophy
  - Iris nevus/ Cogan-Reese syndrome

RARE

ICE Syndrome

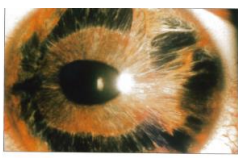
- Characteristics
  - Asymptomatic
  - Irregular pupil
  - Blur
  - Pain
    - Corneal edema
    - Increased IOP



RARE

ICE

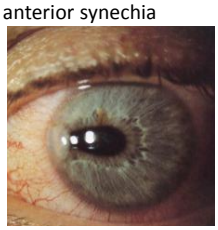
- Clinical findings
  - Corneal endothelial findings (beaten-bronze appearance)
  - Corneal edema
  - Abnormal iris appearance
  - Abnormal gonioscopic appearance



RARE

Progressive iris atrophy-ICE

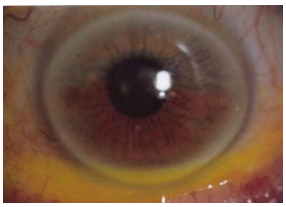
- Clinical findings
  - Iris stromal atrophy
    - Pseudopolycoria
  - Development of peripheral anterior synechia
    - Corectopia
- Prognosis: good



RARE

Cogan-Reese-ICE

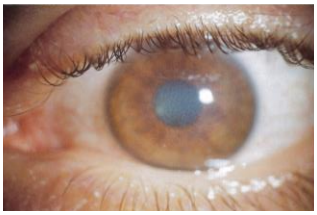
- Clinical findings
  - Diffuse iris nevus
    - May look like heterochromia
  - Pigmented nodules on iris
- Prognosis: poor



RARE

Chandler-ICE

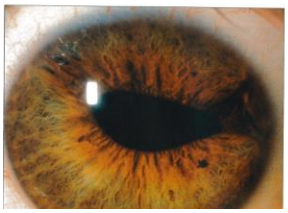
- 50% of ICE
- Clinical findings
  - Most corneal changes of ICE's
  - Iris atrophy
    - Corectopia
- Prognosis: variable



RARE

ICE Management

- Management
  - IOP lowering
  - SLT/ALT/PI ineffective
  - Corneal edema



Key Points

- In all glaucoma patients/suspects:
  - Thorough history
  - Careful anterior segment evaluation
  - Gonioscopy
- All secondary glaucomas
  - Treat primary condition first
- Consider best treatment
  - IOP lowering medications vs. surgical
- Medicolegal implications

Cases

Questions?

References

1. Ehlers JP et al. The Wills Eye Manual. 5th ed. Lippincott Williams & Wilkins; 2008.
2. Stamper RL et al. Becker-Shaffer's Diagnosis and Therapy of the Glaucomas. 8th ed. Mosby Elsevier; 2009.
3. Lewis TL, Fingeret M. Primary Care of the Glaucomas. Appleton & Lange; 1993.