Optometric Education Consultants

Second Thoughts on Secondary Glaucomas

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DISCLOSURE:

- *Joseph Sowka, OD* is/ has been a Consultant/ Speaker Bureau/ Advisory Board member for Novartis, Allergan, Glaukos, Zeiss, Aerie, Oculat Therapeutix, and B&L. Dr. Sowka has no direct financial interest in any of the diseases, products or instrumentation mentioned in this presentation. He is a co-owner of Optometric Education Consultants

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Disclosures- Greg Caldwell, OD, FAAO

- Will mention many products, instruments and companies during our discussion
  - I don't have any financial interest in any of these products, instruments or companies
- Pennsylvania Optometric Association –President 2010
  - POA Board of Directors 2006-2011
- American Optometric Association, Trustee 2013-2016
  - Thank you to the members and those who join
- I never used or will use my volunteer positions to further my lecturing career
- Lectured for: Shire, BioTissue, Optovue
- Advisory Board: Allergan
- Envolve: PA Medical Director, Credential Committee

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- He is a co-owner of Optometric Education Consultants

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Why Secondary?

- *Secondary* to another condition
  - Inflammation, neovascular disease, neoplastic disease, cataract, etc.
- Potentially curable if underlying cause is treatable
- Approximately 1/3 of all glaucoma cases
- Patient is typically younger than POAG patient
- Angle may be closed or open depending upon type of glaucoma
- ***Knowledge of angle status is very important as this will dictate management***

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Common Secondary Glaucomas

- Pigmentary (angle open)
- Pseudo-exfoliative (angle open nearly always, rarely closed, but possible)
- Early and late traumatic (angle open or closed)
- Steroid induced (angle open)
- Lens induced (angle open or closed)
- Neovascular (angle closed)
- Inflammatory (angle open or closed)

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Best of the Rest

- Miscellaneous causes (angle open or closed)
  - Post retinal detachment surgery
  - Systemic disease related
  - Drug induced
  - Elevated episcleral venous pressure
    - Idiopathic, Sturge Weber syndrome, cavernous sinus fistula
  - Siderosis
  - Post penetrating injury
  - Numerous other potential causes

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The Case of the Resident Misdirection

- 60-year-old Hispanic male presents for CEE
- BCVA 20/20 OD, OS
- Past Ocular History: hx of ocular trauma at 7yo
  - Vague and forced by resident
- IOP: 15 mm OD, 38 mm OS
- Gonio: funky angle- very deep
- Diagnosis: angle recession glaucoma OS

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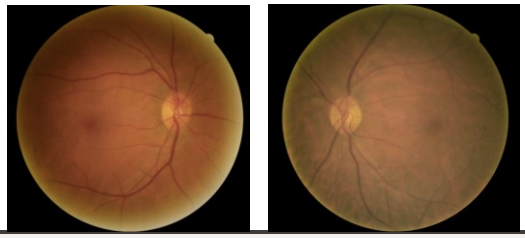
Ancillary Testing – Fundus Photos



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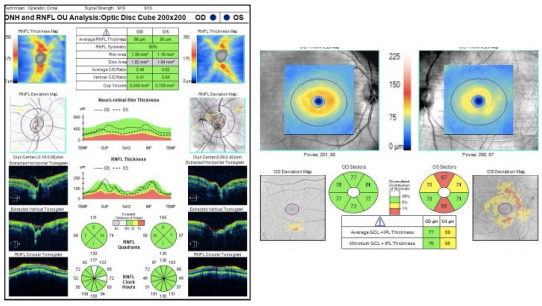

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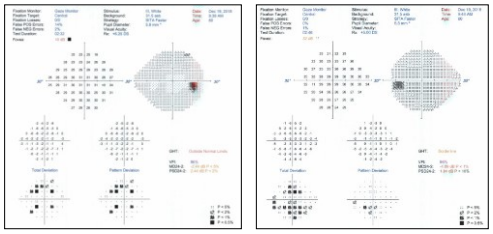

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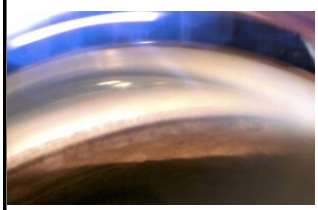

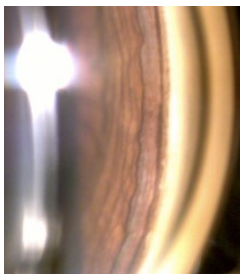

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Polling Question 1

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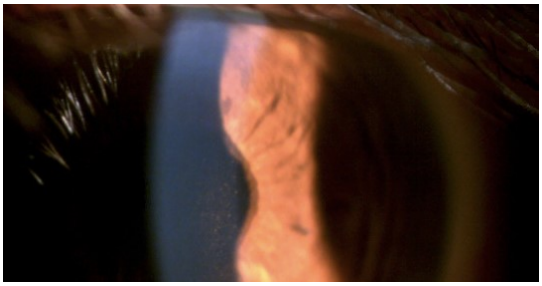

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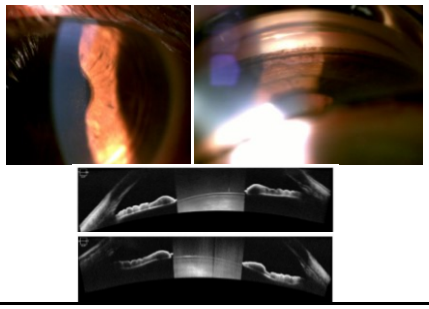

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Pigmentary Glaucoma

- *Secondary open angle mechanism*
- Younger, myopic, white males
  - Does appear in women as well
- Men are often the ones that develop true glaucoma
- Can occur in patients of African descent
  - Often middle-aged women of color aged 45-53 years
  - Distinctly different appearance
  - Planar iris, iris TIDs rare or not present, minimal corneal pigment

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Pigmentary Glaucoma

- Bilateral, but may be asymmetric
- Pigment dispersion syndrome (PDS) is the precursor
- About 50% conversion rate to pigmentary glaucoma over lifetime
- High diurnal IOP fluctuations- IOP spike can easily be missed on single exam

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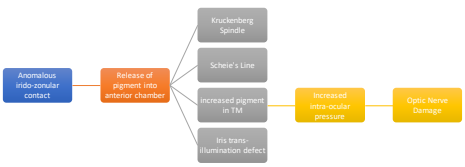
Mechanism of Action Pigment Dispersion Syndrome



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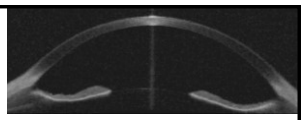
Pigmentary Glaucoma



- Irido-zonular contact and friction
- Posterior bowing of mid-peripheral iris (reverse pupil block)
- Aqueous is trapped in anterior chamber
- Valve-effect is created
- Possible relationship with blink and/or accommodation
- Constant rub between iris and lens zonules
- Pigment release

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Pigmentary Glaucoma

- Development of Krukenberg's spindle (KS)
  - Doesn't always have to be a spindle formation
  - May be diffuse pigment
- The presence of Krukenberg's spindle or endothelial pigment should lead you to transilluminate the eye and do gonio.
- Transillumination defects (radially located in mid-peripheral iris)
- The presence of transillumination defects should lead you to perform gonioscopy
  - Transillumination defects not always present
  - Dependent upon iris thickness
- Not directly related to IOP

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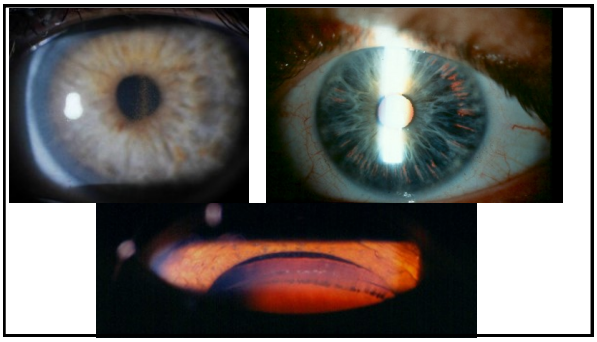

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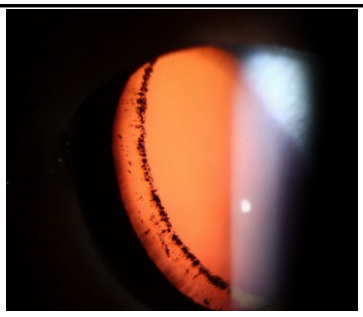

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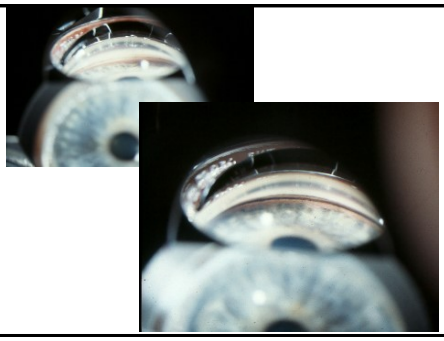

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Pigmentary Glaucoma

- The TM endothelial cells phagocytize pigment.
- Eventually, digested pigment as well as the increased activity breaks down the TM cells which lift off the trabecular beams. The overall result is a breakdown of the TM secondary to having to process the pigment. The subsequent inability to process aqueous causes IOP elevations.
- Physical blockade is only a minimal part of the reason for the pressure rise. Trabecular meshwork may have pigment deposition w/o IOP increase- depends on the ability of TM to process and phagocytize pigment

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Historical Factoid:

- There exists a situation where patients complain of blurred vision when exercising.
- This relates to pigment dispersion patients who release greater amount of pigment from jarring exercise with subsequent trabecular meshwork accumulation, aqueous flow impedance, acute rise in IOP, shutdown of the sodium-potassium pump on the endothelium and development of corneal edema causing blurred vision.

- Virtually every eye care clinician knows this and can recite the mechanism and cause. What they don't know is that this all stems from a single case report in the 1960s and researchers have never been able to duplicate it in any clinical trial. While you need to know this in order to be conversant with the misguided, realize that it is likely an urban legend.

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Polling Question 2

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Pigmentary Glaucoma

- Treat PDS as a risk factor for glaucoma development. Initial fields, disc, and NFL analysis is indicated to assess what status of damage may have already occurred. Diagnosis can be missed.
- Tx similar to POAG when glaucoma develops
  - Beta blockers, CAI, adrenergic agonist, prostaglandins, ROCK (?)
- There is an argument that because prostaglandins increase the size of the pigment cells, it may exacerbate the blockage. This concept is unproven, however and many patients have been successfully managed with these medications

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Pigmentary Glaucoma

- Pilocarpine 1% or 2% is theoretically useful for relieving the irido-zonular friction but is not clinically or practically reasonable due to adverse medical effects.
- Patients with pigment dispersion syndrome/ pigmentary glaucoma have a higher incidence of retinal pathology such as lattice degeneration and retinal detachment
- SLT
  - Works well but paradoxical IOP spike can happen
- LPI?

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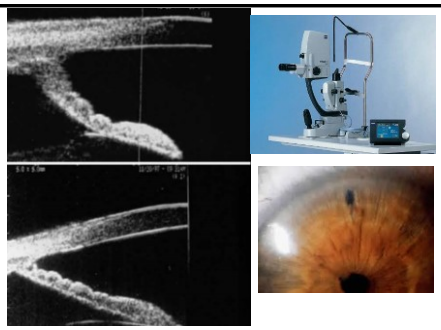

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The Case of the Bad Prognosis?

- 78 YOWF
- Average IOP (1 yr x5); 22 mm OD, 20 mm OS
- CCT: 517 OD, 527 OS
- PXE material OU
- Gonio open OU with moderate pigment

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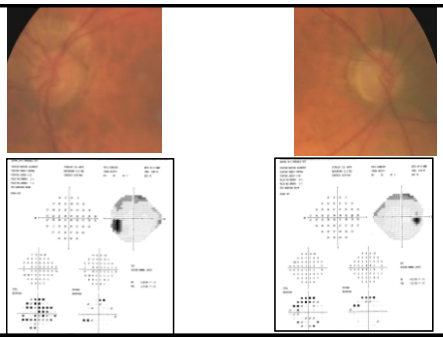

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Bad Prognosis?

- PXE glaucoma diagnosed
- Considerations:
  - Mild field loss
  - Older age
  - Lower initial baseline IOP
  - PXE
- Can this patient be monitored, or should she be treated?

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Bad Prognosis?

- Pt answers the question- declines treatment
- Bad experience with treatment suggested by doctors in past
  - more afraid of treatment than glaucoma
  - Wants to see change or other conclusive proof of need for treatment.
- However, everything says she will do poorly
  - Peak IOP: *34 mm Hg OD, 37 mm Hg OS*

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Pseudo-Exfoliative/ Exfoliative Glaucoma

- Exfoliation- pseudoexfoliation- true exfoliation?
- Age-related generalized disorder of the extracellular matrix characterized by the deposition of abnormal basement membrane (fibrillar extracellular material) on anterior lens capsule, iris, and in trabecular meshwork.
- Abnormal basement membrane comes from lens, iris, ciliary body, and uvea. In that true "exfoliation" is clinically very rare, pseudoexfoliation syndrome and pseudoexfoliative glaucoma are often termed "exfoliation"
- Exfoliation is probably the best term because issues arise when this material is rubbed off.

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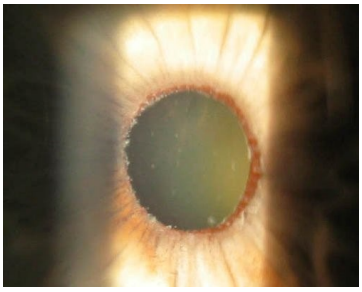

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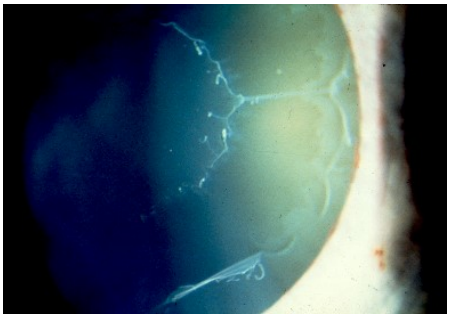

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Pseudo-Exfoliative/ Exfoliative Glaucoma

- Mechanism of glaucoma is nearly always *secondary open angle*, however in some uncommon cases, the PXE material may lead to lens dislocation with pupil block angle closure
- High prevalence in northern Europeans
- High altitude, northern climate, exposure to UV radiation are risk factors
  - In the United States, people who live in the northern tier (above 42° latitude) have the highest incidence of exfoliative syndrome. Those in the southern tier (below 37°) have the lowest incidence.

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Pseudo-Exfoliative/ Exfoliative Glaucoma

- Peripupillary transillumination (may be seen in absence of clinically detectable pseudo-exfoliative material)
  - The presence or development of peripupillary TID is a very important indicator of PXE- PXE suspects
- Abnormal basement membrane
  - Deposited on anterior lens capsule, not from lens
- Pigment released from pupil border
- Posterior synechia
- Radial pigment deposition

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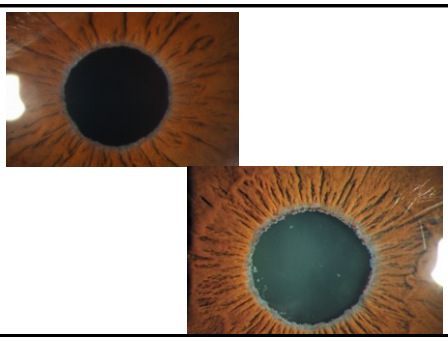

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Pseudo-Exfoliative/ Exfoliative Glaucoma

- Heavy pigment (and exfoliative material) found in trabecular meshwork and may block trabecular meshwork, but the mechanism is not well understood
- Essentially functions the same as pigmentary glaucoma
- PXE is a significant complicating factor in cataract extraction- loss of lens
  - Lensectomy is not curative-material will deposit on IOL as well as remaining anterior capsule
- Now recognized as a generalized systemic disorder of the extracellular matrix
  - Exfoliation material is present in the walls of posterior ciliary arteries, vortex veins, and central retinal vessels as well as in the heart, lung, liver, kidney, gall bladder, and cerebral meninges
    - **Associated with central retinal vein occlusion (CRVO)**

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Pseudo-Exfoliative/ Exfoliative Glaucoma

- Systemic associations include TIA's, stroke, Alzheimer's disease, hearing loss, hyperhomocysteinemia, and heart disease
- Polymorphisms of the lysyl oxidase-like 1 (LOXL 1) gene on chromosome 15 are specifically associated with syndrome and glaucoma
- LOXYL 1 enzymes are essential for the formation, stabilization, maintenance, and remodeling of elastic fibers and prevent age-related loss of elasticity of tissues
- LOXYL 1 protein is a major component of the exfoliation deposits

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Pseudo-Exfoliative/ Exfoliative Glaucoma

- Overall, about 40% likelihood of developing glaucoma throughout life
- When glaucoma develops, IOP is usually higher than in POAG
  - More rapid progression than POAG
  - IOP very labile
  - Difficult to control
  - More likely to need surgery
  - More complications with cataract surgery
- Highest IOP is often occurring outside normal office hours.
- IOP may transiently rise after dilation due to pigment liberation

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Pseudo-Exfoliative/ Exfoliative Glaucoma

- Treat as POAG
  - Beta blockers
  - Prostaglandins
  - Adrenergic agonists
  - CAI's
  - Rock inhibitors?
- ALT/ SLT - good modality
- Trabeculectomy/tube, maybe MIGS but disease is usually too severe

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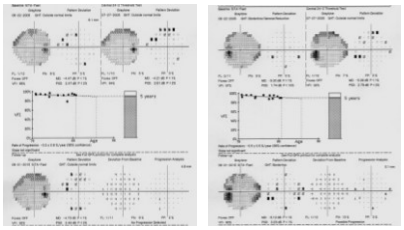
Back to Velois: 2005-2015



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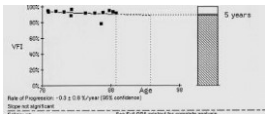
2005-2015



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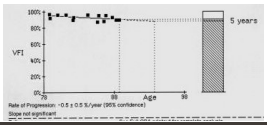

OS

Risk factors: PXE, High IOP, older age: yet no progression?! Now she shares a new concern. Final outcome?

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Traumatic Glaucoma: Angle Recession

- Cleavage of ciliary body muscles
- Widening and deepening of angle
- Fellow eye comparison is necessary because this is not obvious
- Etiology is thought to be trabecular meshwork scarring/sclerosis
- Problems occur years after antecedent trauma
- Secondary open angle

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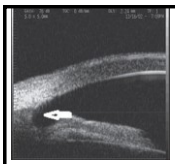

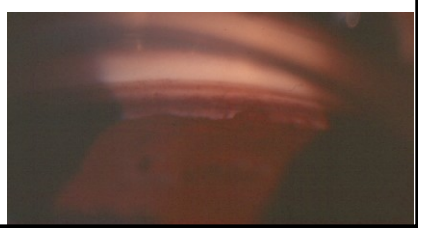

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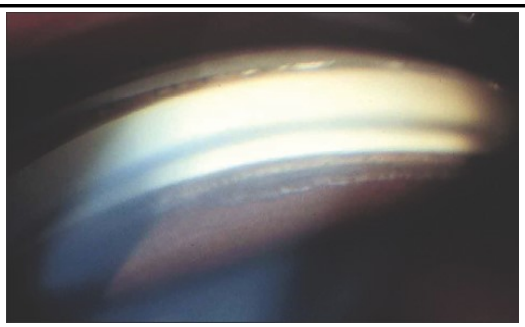

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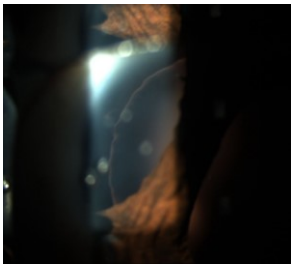
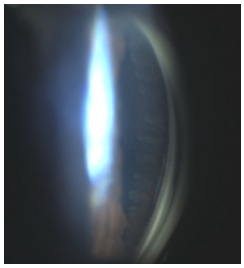

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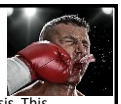
Traumatic Glaucoma: Angle Recession



- The trauma damages the meshwork, causing scarring and sclerosis. This may not be gonioscopically apparent and initially may not affect IOP. However, as trabecular function declines with age, there is an unmasking of this traumatic dysfunction.
- This should be your first thought when encountering unilateral glaucoma
- 10-20% of angle recession pts. develop secondary glaucoma
- Severity of glaucoma often, but not always, related to extent of recession
- The trabecular damage is not limited solely to the extent of the recessed angle. The entire meshwork is usually damaged to some degree.

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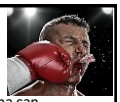
Traumatic Glaucoma: Angle Recession



- Observation if IOP, discs normal. Always remember that glaucoma can develop years later due to declining trabecular function and these patients are forever at risk.
- Aqueous suppressants
  - Beta blockers, CAI's, Alphagan
  - Miotics, ROCK inhibitors very questionable due to changes in meshwork
  - ***Prostaglandin analogs seem to work very well***
- Laser trabeculoplasty -poor response if recession > 180
- Trabeculectomy/ tubes work well
- MIGS questionable

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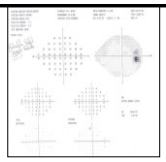
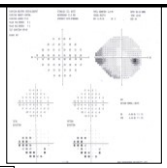
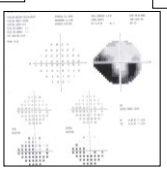

Angle recession glaucoma:
IOP 47 mm
Pt non-compliant and declines surgery

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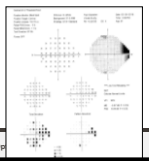
Miss Daisy, Driving?

- ***Angle recession OS*** from blunt trauma several years ago
- Peak IOP 47 mm- corneal edema
- Hyperemia with Travatan Z- sampled with Zioptan
  - Much better than Travatan, but still a little hyperemia. Scheduled for 3 week f/u and presents promptly 11 months later- only used 3 drops of Zioptan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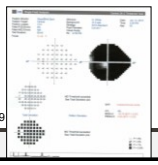
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Likes to try to drive with OS to see if she can do it

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Sowkaism: The trauma that causes angle recession also seems to cause brain damage

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The Case of the Snowy Referral

- 21 year old Hispanic female
- Chief Complaint: referral for elevated intraocular pressure – no referral noted
- Pt c/o snowy vision for the last few months, that is getting worse
- Past Ocular History: unremarkable
- Past medical history: Asthma dx 2017
- VA: OD 20/70, PH 20/40 OD, OS
- PERRLA OD, OS; -APD OD, OS
- Gonio: open to CB x360 without abnormalities OD, OS
- Pachymetry: 639 OD, 640 OS

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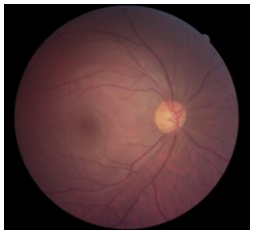

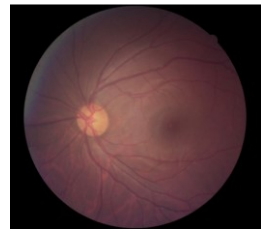

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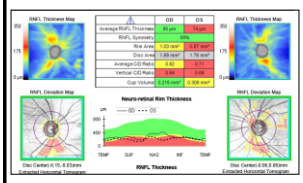

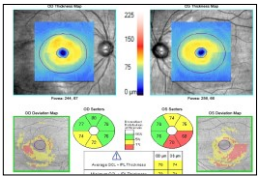

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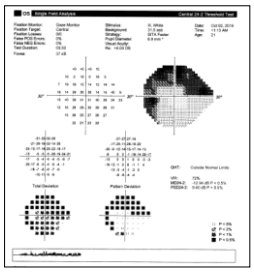
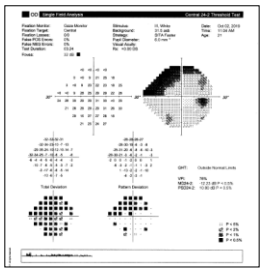




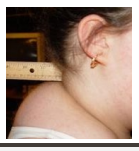
BTW, I forgot to mention that her IOP is 72 mm Hg OU

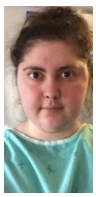
59

60

So, How Are You Treating That Asthma Again?

- Lens: 2+ PSC OD, OS (snowy vision)
- IOP: 72 mm OD, OS
- Self medicating with IM dexamethasone 2-3 times per week
- Weight gain
- Hair loss
- Diabetes
- Steroid induced glaucoma
- Iatrogenic Cushing syndrome





61

Steroid Induced Glaucoma

- Secondary open angle
- Outflow difficulty- steroids are thought to change the TM ability to process aqueous.
  - Glycoaminoglycan (GAG) accumulation is thought to be the underlying difficulty
- TM endothelium decreases phagocytotic ability
  - Steroids may prevent release of enzymes that normally depolymerize gags and prevents TM endothelial cells from keeping TM properly cleaned up and healthy.
- Increased difficulty of outflow

62

Steroid Induced Glaucoma

- Steroids commonly affecting IOP are prednisolone, dexamethasone, betamethasone, and difluprednate
  - Difluprednate often affects IOP faster and more significantly, but not to a greater prevalence than prednisolone
- Any steroid can elevate IOP
  - Loteprednol is considered a soft steroid with less propensity to elevate IOP, but it can cause glaucoma like any steroid

63

Steroid Induced Glaucoma

- Response is dependent upon:
- Frequency of application
- Dose
- Duration
- Genetic predisposition
- Genetic relationship - TIGR/Myocillin gene
  - The incidence points to an autosomal recessive inheritance pattern
- 2/3rds are steroid responders- few get into trouble

64

Steroid Induced Glaucoma

- Those at risk include:
  - Myopes, Pts. with POAG, Children
- Treatment:
  - D/C steroids
- After prolonged use, IOP may not lower with medication cessation
  - Aqueous suppressants, Prostaglandins (depending upon the amount of inflammation and route of steroid)
  - Anything designed to enhance trabecular outflow (Trabeculoplasty, miotics, Rock inhibitors) will have a poor effect; trabeculectomy works better

65

Outcome

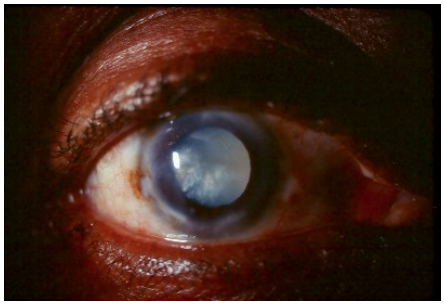
- Multiple meds used- insufficient effect
- Risk of adrenal insufficiency and adrenal crisis
- Referred to endocrinologist immediately for oral steroid taper.

66

The Case of…28 Days Later

- 54 YOF- CEE
- 20/200 OD; 20/40 OS
  - Cataract OD > OS
- Cataract consult
  - 20/400 OD, 20/50 OS
  - Not good candidate due to corneal and retinal rubella scarring
- 28 days later…pt calls complaining of rapidly decreasing vision OD and pain
  - LP OD, 32 mm Hg OD; + A/C reaction; angles open

67



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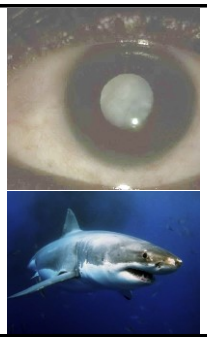

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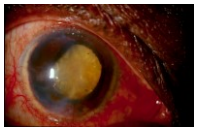
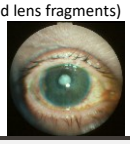
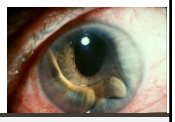
Lens Induced Glaucomas



- Phacolytic
- Phacomorphic
- Lens particle
- Phacoanaphylactic (retained lens fragments)
- Ectopia lentis





70

Phacolytic Glaucoma

- Uveitis and elevated IOP in association with hypermature cataract
- Predominately secondary open angle
- Acute onset of pain and redness in an eye that is non-seeing
  - Vision typically is in light perception range
- Hypermature cataract- lens leaks out internal proteins, which are antigenic. Capsule ruptures and extrudes lens proteins into anterior chamber
- Antigen/antibody reaction and subsequent A/C reaction

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Phacolytic Glaucoma

- Provokes macrophage response
- Heavy molecular weight proteins become soluble
- Proteins can leak out through an intact capsule
- Liquefaction of lens cortex and attenuation of lens capsule
- White flocculent material in chamber and on lens surface
- Bloated macrophages with lens material within them found in anterior chamber
  - PMN's, plasma cells, and lymphocytes are typically absent

72

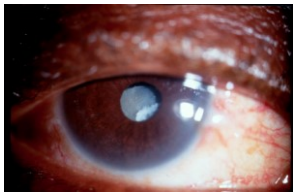
Phacolytic Glaucoma

- Cured by lensectomy and possibly vitrectomy
- Possibility of capsular rupture with subsequent vitrectomy required
- Medical therapy initially to temporize IOP and quell inflammation
  - Corticosteroids Q1-2H, depending upon severity
  - Cycloplegia (unless there is zonular damage and danger of subluxation): homatropine 5%, atropine 1%
  - Beta blockers, alpha adrenergic agonists, CAI's
  - Avoid prostaglandins and miotics; rho-kinase inhibitors?

73

The Case of the 'Safe to Dilate?' Patient

- 65 YOF
- Pain and poor vision- LP
- IOP 35 mm



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The Case of the "You're Wrong", NO, "You're Wrong"

- 65 YOF seen with dense cataract
- Referred for consult- cataract surgery deferred
- Returns 1 year later for CEE- sees resident
- Resident and patient get into "Spirited Debate"
- Resident issue: The patient insists that she never had cataract surgery, but she has no lens
  - Both insist that they are right

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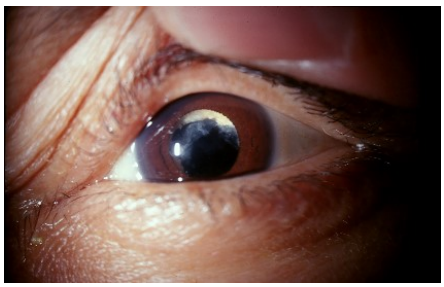

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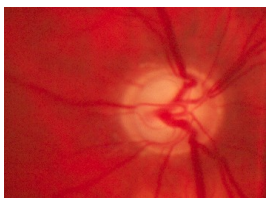
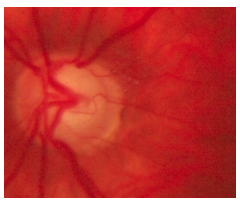
The Case of the Non-Routine Routine Eye Exam

- 50 YOM- CEE
- 20/40 OD, OS
- Rx: (-) 18.00 – 2.50 x 180 OU
- IOP: 42 mm Hg OU
- Constricted visual fields and advanced glaucomatous disc damage OU

77

What Next?





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The Case of the Non-Routine Routine Eye Exam

- Gonioscopy: Chronic angle closure OU
- Non-myopic fundus
- Lens protrudes slightly into A/C
- Diagnosis: Chronic angle closure secondary to phacomorphic glaucoma secondary to isolated microspherophakia
- Management: LPI OU followed by topical glaucoma meds
  - Then things got complicated…

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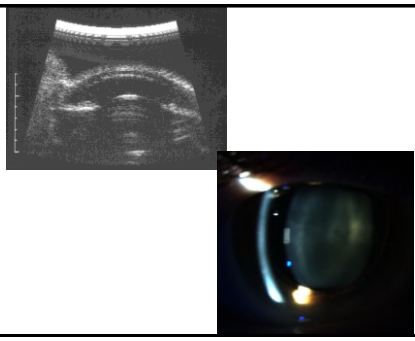

80

Phacomorphic Glaucoma

- Phaco=lens; morph=shape
- Secondary angle closure with pupil block
- Most common lens-induced glaucoma
- Unilateral or asymmetric cataract associated with asymmetric shallowing of the anterior chamber not explained by other factors
- Difficult to differentiate from primary angle closure
- Acute to intermittent red, painful eye, typically at night
- May present asymptomatically with chronic angle closure

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Phacomorphic Glaucoma

- Typically, vision is greatly reduced (<20/400) from the cataract
- Due to increasing lens thickness: irido-lenticular apposition from growth of the lens cortex and intumescence of the lens.
- May be associated with short globe axial length
- Occasionally, phacomorphic glaucoma will occur not due to mature cataract formation, but due to microspherophakia (often associated with Weill-Marchesani syndrome)
  - Presents as acute or chronic angle closure in eyes with high myopia.

82

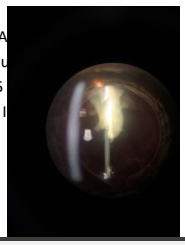
Phacomorphic Glaucoma

- Beta-blockers, alpha-2 adrenergic agonists, topical corticosteroids, topical or oral carbonic anhydrase inhibitors may be all systematically employed.
- An exceptional effect of prostaglandin analogs in managing the IOP of patients with chronic angle closure glaucoma both before and following LPI has been reported.
- Pilocarpine 2% and corticosteroids can also be used.
- RhoKinase inhibitors questionable.
- LPI
- Lens extraction

83

The Case of The Found Dinosaur

- 63 YOM c/o veiling over OD for past 2 days; VA
- Hx of lasered retinal tear- always worried abou
- Hx cataract removal with YAG capsulotomy 15
- Initial inspection reveals opacification behind I
  - But what about that YAG history?
- Grade 2 anterior chamber reaction
- IOP 32 mm OD, 15 mm OS



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Phacoanaphylactic Uveitis/ Retained Lens Fragment

- Inflammatory secondary glaucoma usually due to antigenic lens materials inadvertently left in the eye.
- Autoimmunity to lens antigens, which may be left in anterior chamber following procedure.
- Occurs as a severe uveitis following cataract extraction- may be confused with endophthalmitis.
- In post-surgical cases, there will be either lens cortex or nucleus material (which may not be readily observable) that was not completely removed during the operation. When this happens, it is termed, “retained lens fragment”. Should penetrating lens trauma be the inciting factor, then the term lens particle glaucoma is used.

85

Phacoanaphylactic Uveitis/ Retained Lens Fragment

- Retained lens fragments may hide between IOL and posterior capsule and be protected until later.
- Initiates an open angle glaucoma without pupil block
- Nuclear lens fragments are much more likely than cortical fragments to induce this response.
- Initial inclination to increase/use steroids
  - Rarely effective in providing a cure. Short term only
  - Aqueous suppressants can be used but the material should be removed
  - Pt was placed on topical steroids and Combigan until the fragment was YAGed

86

The Case of the Disappearing Diabetic

- 82-year-old Hispanic male presents for IOP check; LEE: 2 yrs ago – lost to follow-up; POAG OS, severe stage
- Pseudophakia w/ PCIOL OU 2010; YAG Posterior Capsulotomy OS 2010
- Diabetes Mellitus Type 2 x1998
- Medications
  - Latanoprost qhs OU
  - Glyburide 5mg Tablet QD po
- Chief complaint: Pt reports ocular eye-pain and redness of left eye that started 15 days ago

87

The Case of the Disappearing Diabetic

- 20/25 OD; NLP OS
- PERRL (+)RAPD OS
- 3+ diffuse injection OS
- Microcystic corneal edema OD
- Diffuse NVI at the pupil margin OS
- Anterior Chamber: deep & quiet OD; 1/10th hyphema with RBCs in anterior chamber OS
- Lens: PCIOL in good position OD; limited views OS
- IOP 23 mm OD, 62 mm OS
- Gonio: PAS and hyphema OS

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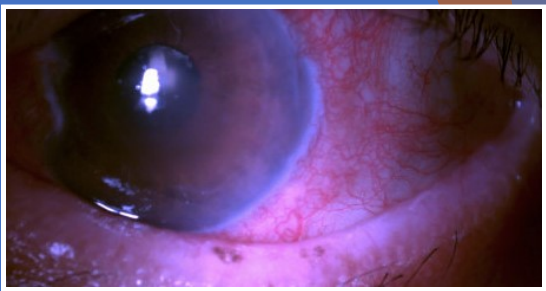

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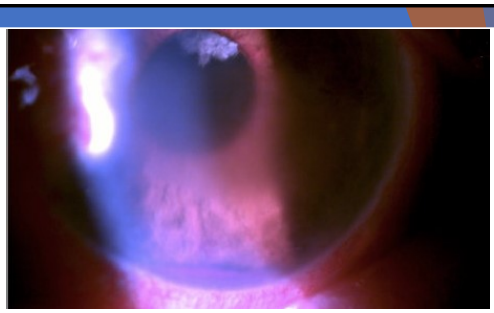

90

The Case of the Disappearing Diabetic

- Management:
  - Combigan BID, atropine BID, pred forte QID, continue latanoprost
- Referred for retinal intervention
- IOP 26 mm OS, old CRVO, cornea clear, better fundus view
- IV avastin and PRP

91

Neovascular Glaucoma

- Neovascularization of the iris and angle (NVI/NVA)
- Mechanism is *secondary angle closure without pupil block*
- Many possible causes
- Mechanism of action
- Inflammation and high IOP
  - Poor prognosis
  - Poorly responsive to medical treatment
- Called the 90 day glaucoma- usually occurs within 90 days of antecedent vascular occlusion
  - Don't be fooled. It can and does happen a lot sooner in many cases.

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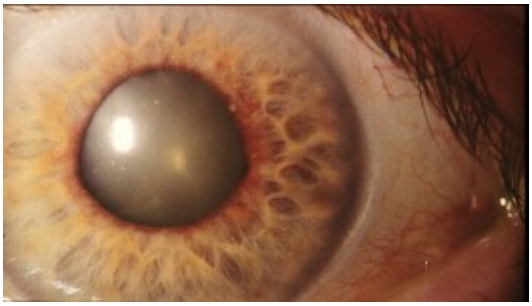

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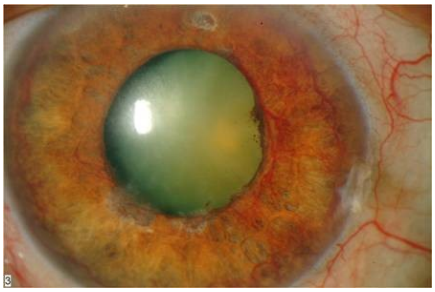

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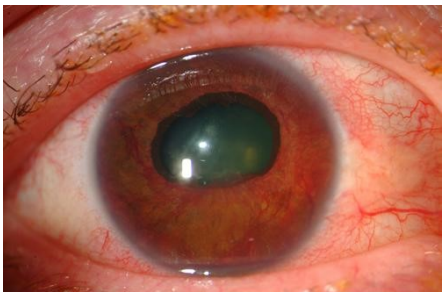

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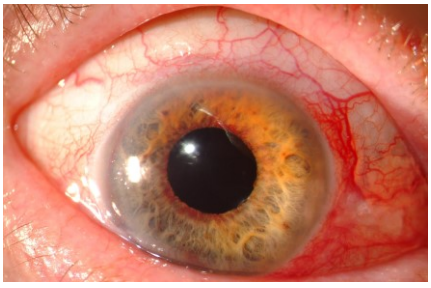

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97

Neovascular Glaucoma

- Initial medical tx: cycloplegia (atropine) and Pred forte used for inflammatory component. May also temporarily use aqueous suppressants until more definitive treatment can be done.
- Generally, you do not chronically medically treat this type of glaucoma.
- Trabeculectomy if not too much of the angle is compromised- high likelihood of failure
- Pan-retinal photocoagulation (PRP) to destroy the ischemic retina and reduce the vasoproliferative substance and induce regression of neovascular vessels. Generally successful (90% success) in diabetic retinopathy if <270 degrees of closure. Much less successful in ocular ischemic syndrome. Cryotherapy may be used in place of PRP.

98

Neovascular Glaucoma

- A newer modality to manage refractory NVG involves trans-scleral diode laser cyclophoto-coagulation. This reduces aqueous production through the laser-induced ablation of the ciliary processes.
- A still newer modality (used in conjunction with methods mentioned above) involves ocular injection of Avastin or Lucentis, which are anti-VEGF drugs
- Not definitive treatment though temporarily very effective.
- Must be accompanied by PRP- otherwise vessels will return.

99

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Neovascular Glaucoma- Here's What To Do

- Atropine 1% BID (yes, its needed)
- Pred forte QID (or equivalent)
- Aqueous suppressants (Combigan, Simbrinza, Alphagan, Timolol, Cosopt, et.)
- PGAs- Meh. Avoid miotics
- Get retina consult for anti-VEGF and PRP
- Done

102

Uveitic Glaucoma

- Glaucoma secondary to uveitis may occur by one or by a combination of several different pathophysiological mechanisms
- Clinical Appearance: Two Types
  - "Hot" eye with pronounced episcleral injection, profuse anterior chamber reaction, high IOP, and variable patient discomfort (sometimes excruciating agony).
  - Quiet and insidious IOP elevation in patients with chronic iridocyclitis
    - More likely to cause glaucoma
- The increased prevalence of glaucoma in chronic uveitis reflects the cumulative effects of inflammation and steroid use. In older patients, minimal amounts of inflammation may overcome a trabecular meshwork with declining function. In younger patients, severe inflammation is usually necessary to overcome a healthy, functional trabecular meshwork.

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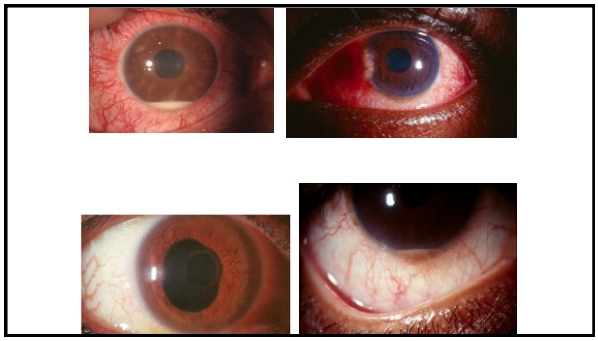

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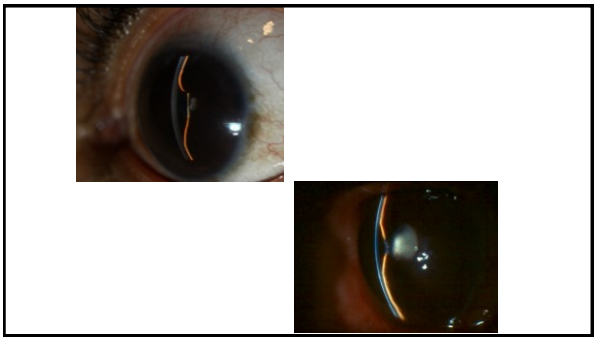

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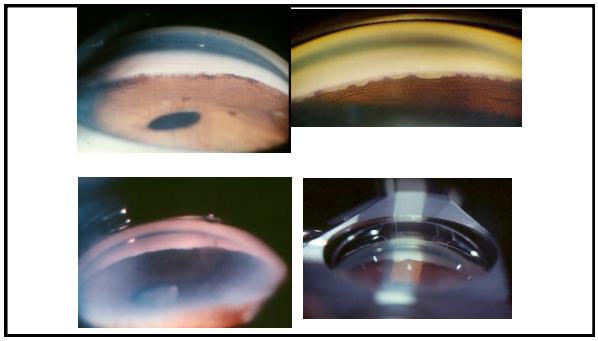

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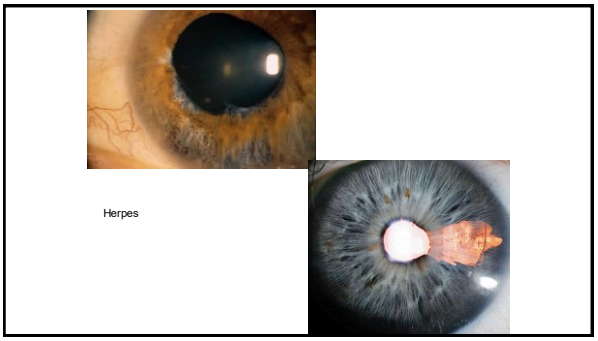

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Dynamics of IOP in Uveitis

Lowering IOP	Elevating IOP
Secretory hypotony from inflamed ciliary body	Posterior synechia with pupil block and angle closure
Prostaglandin induction leading to increased uveoscleral outflow	Peripheral anterior synechia impeding aqueous outflow
	Direct trabecular inflammation decreasing aqueous processing ability
	Accumulation of inflammatory cells impeding aqueous outflow through meshwork
	Flare causing increased aqueous viscosity, impeding trabecular outflow
	Steroid induced alteration in trabecular meshwork

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Uveitic Glaucoma

- In most cases of uveitic glaucoma, there will be a combination of factors causing pressure elevation.
- Chronic low-grade inflammation is more likely to cause glaucoma and more likely to occur in older patients. In younger patients, greater degree of inflammation (such as in acute anterior uveitis) is necessary to cause glaucoma.
- Secondary angle closure can occur in uveitic glaucoma from posterior synechiae and pupil block, extensive PAS, choroidal/ciliary effusions, and neovascularization.

109

Glaucoma Secondary to Chronic Iridocyclitis:

- Likely due to trabeculitis and increased aqueous viscosity due to flare and not angle closure or inflammatory cell accumulation within meshwork
- Vision may be variable due to increased inflammation and protein in the aqueous
  - Biomicroscopic appreciation of changes in inflammation difficult
- Typically occurs when steroids are tapered or disease in not being well controlled
  - Needs increased steroid dosage

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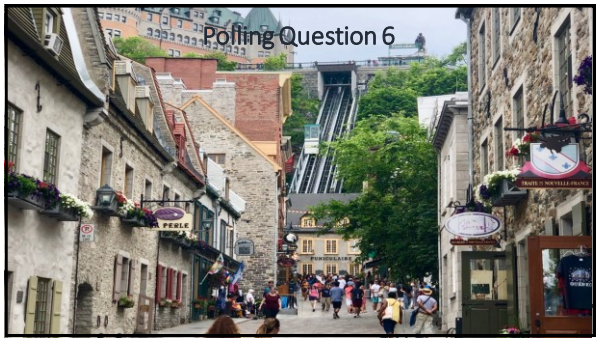

111

Uveitic Glaucoma

- Aggressively reduce inflammation
  - E.g. Pred forte (prednisolone acetate 1%) Q15min x 6H, then Q1H while awake, or difluprednate 0.05% QID
  - May require steroid injections in very severe cases
  - This is absolutely essential to prevent disaster. Do not worry about steroid response glaucoma. Under-treating inflammation due to concerns about steroid impact on pressure is false economy. *THE* MOST IMPORTANT MEDICATION.
- Cycloplegia:
  - Atropine 1% BID
    - Prevent PAS with aggressive anti-inflammatory therapy above
    - Break/prevent posterior synechia
    - In the early stage, prevent or break posterior synechia with potent cycloplegics
    - Steroids help dissolve fibrin and adhesions

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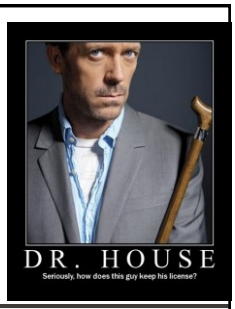
Uveitic Glaucoma

- Lower IOP
  - Beta-blockers, alpha-2 adrenergic agonists, ROCK and CAI's (topical or oral)
- Avoid miotics - exacerbate the condition because it causes movement to a damaged tissue, leading to further breakdown of the blood-aqueous barrier and an increase in inflammation and inflammatory cell in the anterior chamber. This can lead to disastrous consequences
- Leave prostaglandin analogs as last medical alternative
  - There will be high levels of prostaglandins already in the anterior chamber that mitigate the inflammatory reaction. More likely to not help rather than hurt
  - PGAs have been shown to be effective in managing uveitic glaucoma, but just not an optimal choice

113

The Case of *It just isn't clear*

- 24 YOBF
- CC: Blurred vision OS
- Happens twice a year since age 7
- BVA 20/15 OD, 20/20 OS
- PERRL (-) RAPD
- CF: FTFC OD, OS
- Medical history unremarkable

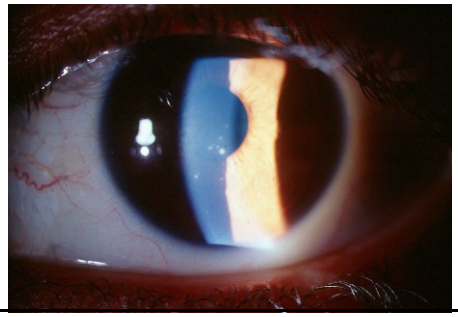


114

Case: *It just isn't clear*

- Conjunctiva clear OU
- Cornea: steamy edema, KP's
- A/C deep
- IOP: 21 mm Hg OD, *70* mm Hg OS

115



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MANAGEMENT
This Patient

- In Office: Pred Forte, Timoptic 0.5%, Alphagan, Trusopt (i gt. each, separated by 5 min)
- After 30 min: IOP 50 mm Hg; edema completely gone!
  - ***"Now everything is perfect. Can I go now?"***
- Repeat regimen:
  - After 30 min: IOP 35 mm Hg → Send patient home with Pred Forte Q2H; Alphagan TID
  - F/U 24 Hrs: IOP 10 mm Hg
  - Threshold fields, OCT: Normal OD, OS

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Points to Ponder

Is GCC a truly benign disease?

Is GCC a real diagnosis or a variant of uveitic glaucoma?

Is GCC an herpetic variant?

Needs steroids- avoid miotics

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The Best of the Rest

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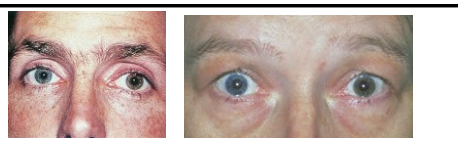

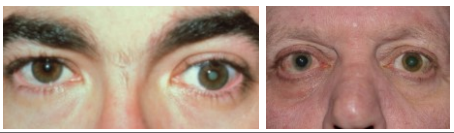

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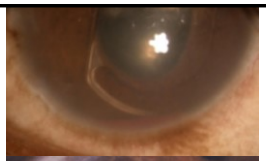
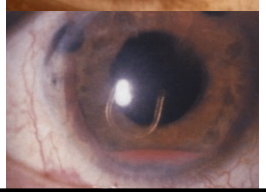
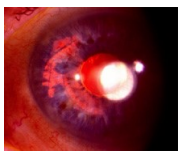




UGH



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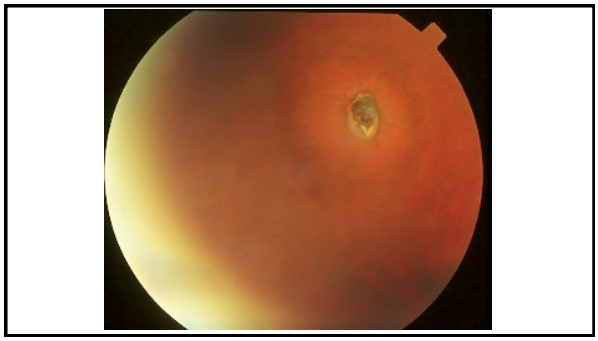

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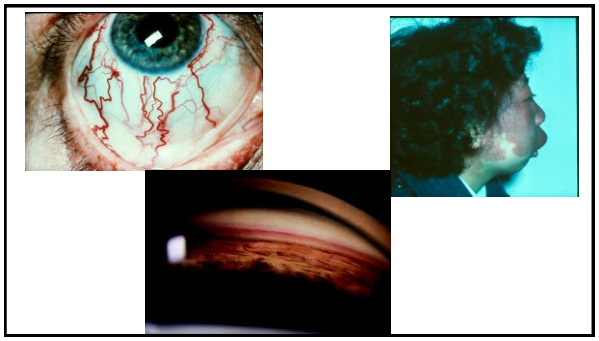

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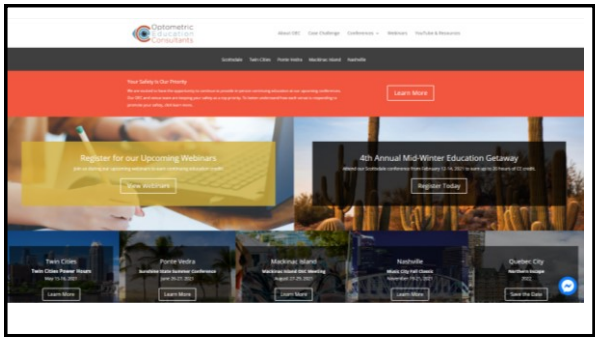
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