Glaucoma Grand Rounds 2021

Elliot M. Kirstein, OD, FAAO Harper's Point Eye Associates Glaucoma and Diabetes Eye Institute Cincinnati, Ohio

Financial Interests

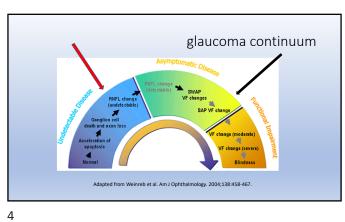
- Optovue speakers alliance
- Reichert speakers alliance
- Haag Streit speakers alliance
- Ocuflow consultant

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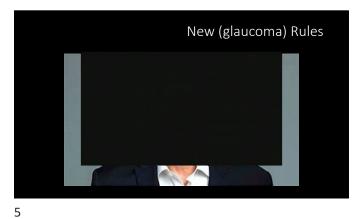
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Agenda

- POAG Pigmentary
- NTG
- Drance Hemorrhage LASIK
- Steroid induced
- Narrow AnglePseudoexfoliation
- Uveitic
- IOP emergency



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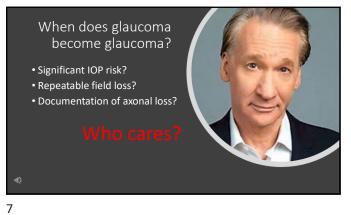


Novel Classification Where is the glaucoma coming from?

- Anterior segment glaucoma etiology
- Posterior segment glaucoma etiology
- Combined glaucoma etiology

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Glaucoma Progression

- Glaucoma is a bilateral disease
- Glaucoma progression is not symmetrical
- Glaucoma progression is not linear

Treat

Risk of PROGRESSION

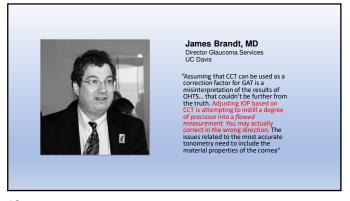


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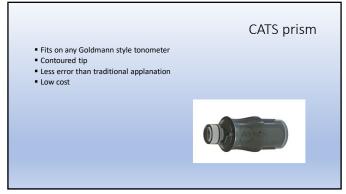
Correction Value (mm Hg) CCT (µm) **Corneal Thickness** & IOP 465 +6 485 +5 Calculation based on data of Ehlers et al (1975) Modified from Stodtmeister (1998) 505 +3 525 +1 545 0 Arithmetic mean of corneal thickness in healthy subjects: 545 µm (Doughty and Zaman 2000) 565 -1 585 -3 605 -4 625 -6

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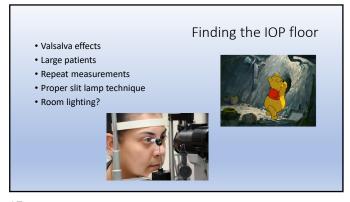






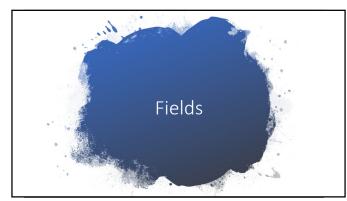


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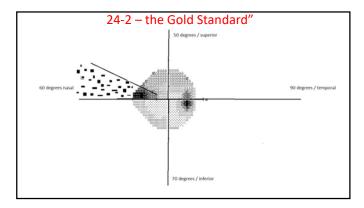


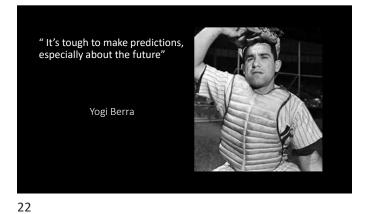


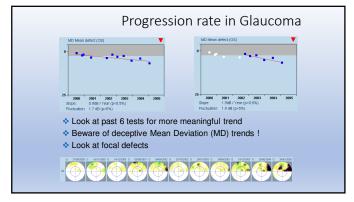
What Changes First? Structure damage often precedes functional damage in glaucoma¹ RNFL loss often precedes optic disc changes² Can GCC change before RNFL? Sommer A. et al. Clinically detected nerve fiber atrophy precedes the onset of glaucomatous field loss. Arch Ophthalmol 1991; 109-77-83. Coulgley HA, Katz J et al. Ophthalmology 1992; 99: 19-28.

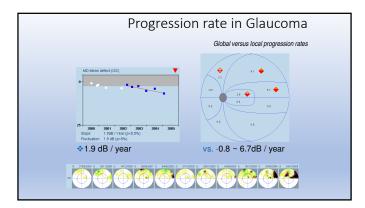


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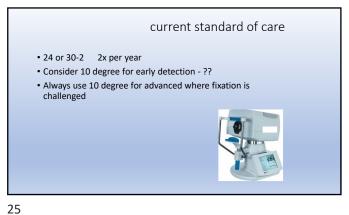


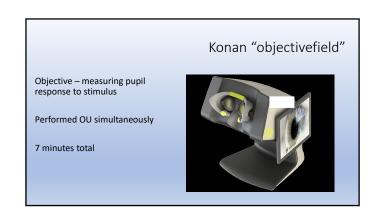


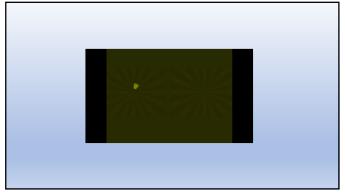


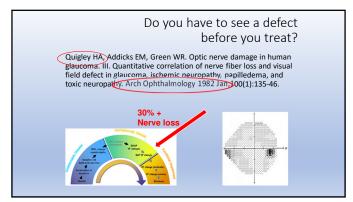


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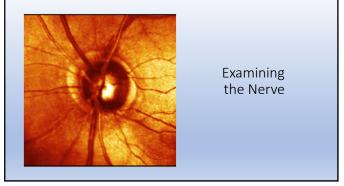




Ophthalmoscopy / Photography



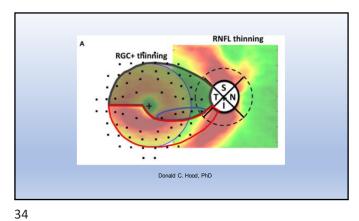
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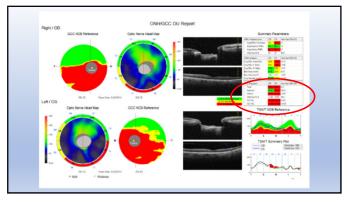




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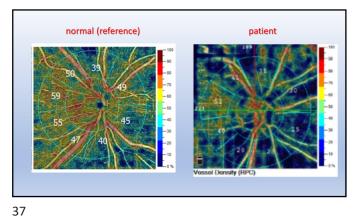


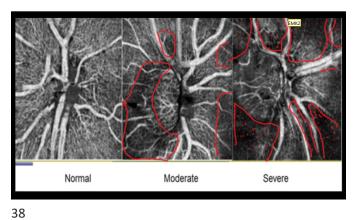




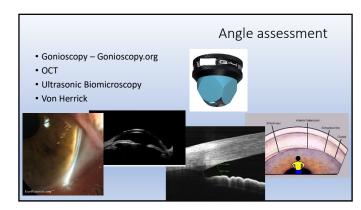


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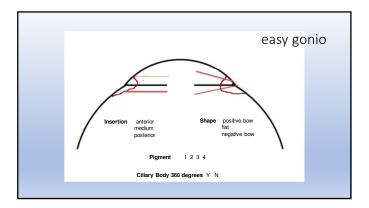


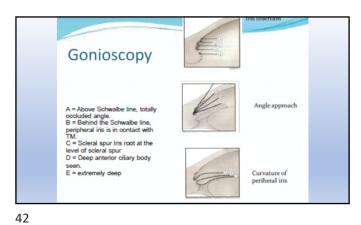






EMK2 Dr. Elliot M. Kirstein, 12/21/2019









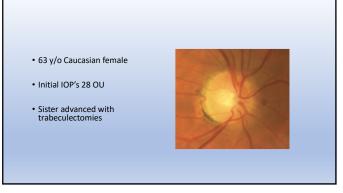
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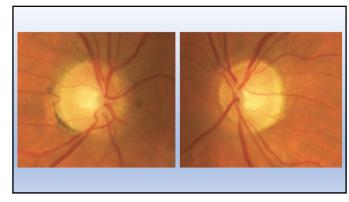


Primary Open Angle Glaucoma

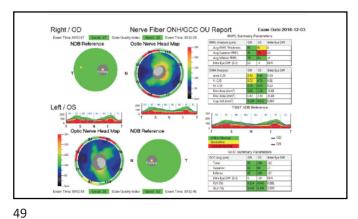
- Most common
- Highly hereditary
- IOP seldom over low 30's
- No apparent angle anomaly
- First changes seen mostly structural

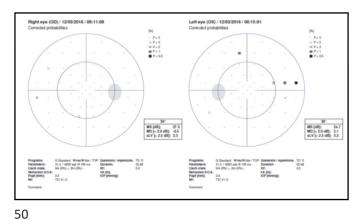
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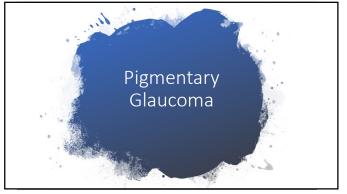




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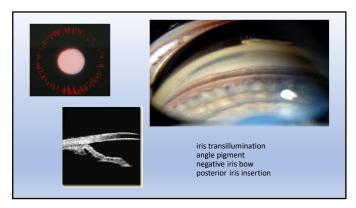






48 y/o white male routine exam Monday, 2/25/2005, 2:00 pm • Post RK 15 years, "no family Hx of glaucoma" – High plus – high Cylinder • Gat 24 / 22 • PAK 638µm / 632µm "corrected IOP" 21 / 18 Pigment dispersion, OU mild (no endothelial pigment, negative bowed iridies, 2+ angle pigment, slight iris trans illumination No family History • WHATS NEXT? Work-up or wait until next year?

51 52



Thursday, 2/28/2005 9:00 AM Follow-up Visit

- Excellent fields
- Excellent HRT (small nerves)
- + "mother had glaucoma"
- DCT = 37.4 / 32.8 (GAT 28 / 22)
- Rx qpm OU Travataprost / RTO 14 days
- WHAT'S NEXT?

53 54

Thursday, 1:00 pm phone call (same day)

• My right eye is "foggy" & "red"

"come on in!"

Thursday, 2:00 pm Re-check

- V/A = 20/50, 20/30
- Cornea cloudy OD>OS, 2+ injection OU
- Pupils equal & reactive
- A/C 2+ cells (Pigment) OU
- Crisp new Krukenberg spindles OU
- DCT 50.2 / 44.8

treatment?

55 56

8/3/2021



Thursday, 3:30 PM

V/A = 20/25+, 20/25+•DCT = 19, 14

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NTG patient profile

- IOP not over low 20's
- Low blood pressure profusion pressure (DPP)
- High myopia
- Race
- Migraine
- Gender

diastolic perfusion pressure (DPP)

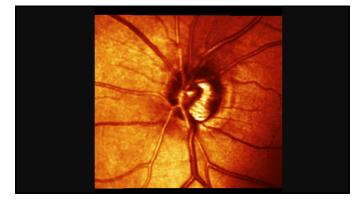
DPP = diastolic BP - IOP

Barbados and Baltimore Eye Studies
Increased risk for low DPP (below 55)

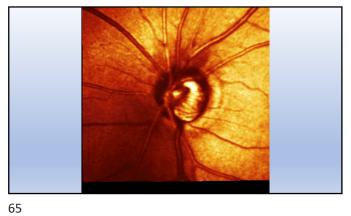
This means that the highest risk is a combination of high IOP and low diastolic BP

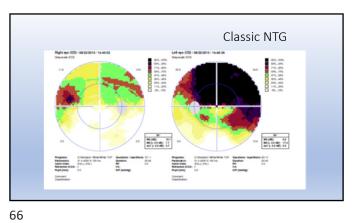
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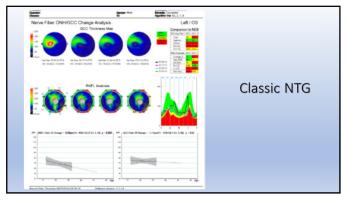
Classic NTG IOP OD = 20 / 21 69 y / o Caucasian male OPA = .9mm Hg OU No Family Hx Tall slender stature Open angles – light pigment CCT – 540 / 535 Para central defects OS > OD Poor tolerance to beta blockers

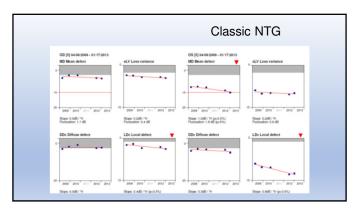


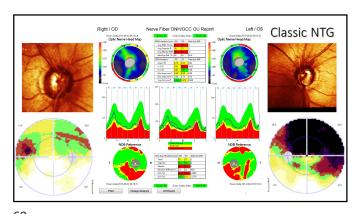
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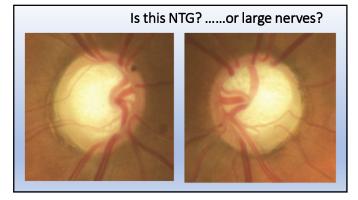


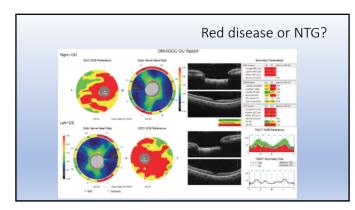


53 y/o Caucasian female

- "They've always told me that something way funny in the back of my eye, but the ophthalmologist said I didn't have glaucoma"
- IOP's High teens
- 550 micron pachymetry
- BP 135/85
- High body mass index
- No known family history of glaucoma

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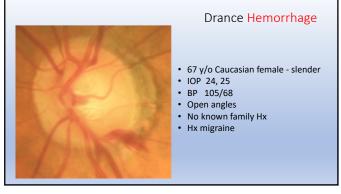


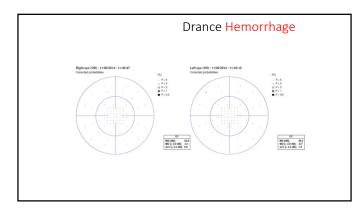
Drance Hemorrhage

- 13% POAG / 20% NTG
- 84% are missed
- 100% with 2 disc hemorrhages will have field loss
- 81% with 1 disc hemorrhage will have field loss
- 3 fold progression risk even under treatment

Jeffrey M Liebmann, MD - New York Eye & Ear Infirmary 3.2015

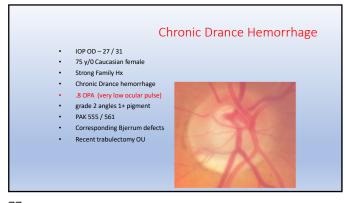
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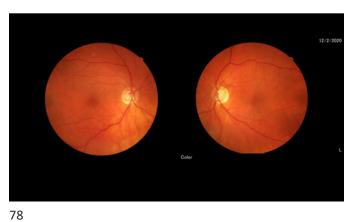




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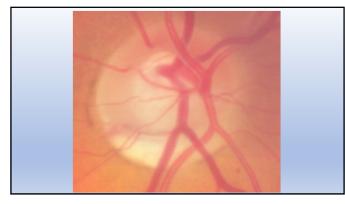
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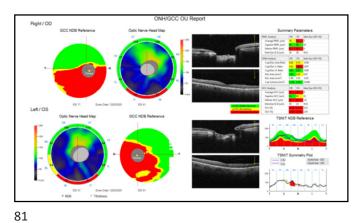


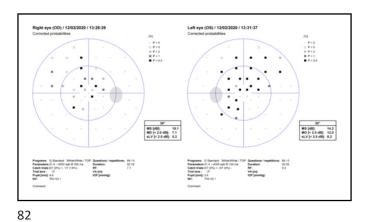
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LASIK •15,000,000 Americans •Severe effect on applanation tonometry • Masks elevated IOP!!!

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LASIK

• LASIK

• Age 58 / male

• OD = 20/70 OS = 20/25

• Never had IOP over 21

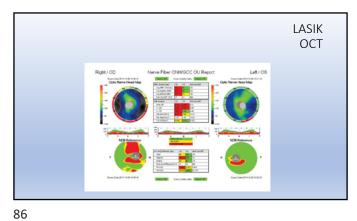
• Late diagnosis

• Retinal tear

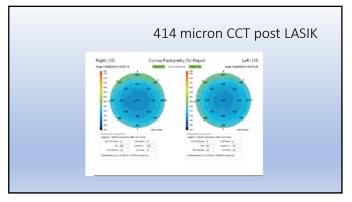
• Epi Retinal Membrane with peel

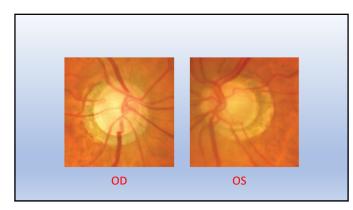
• Low blood pressure

• Advanced Normal Tension Glaucoma

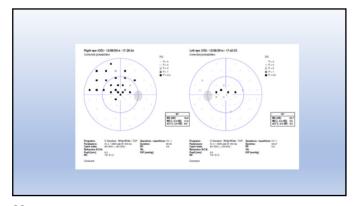


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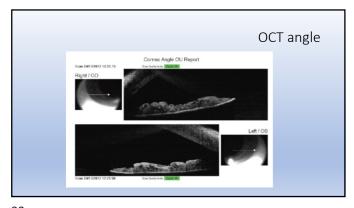


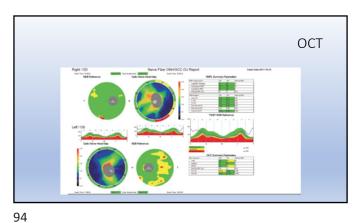


67-year-old Caucasian male

- \bullet Initial treatment with IOP risk at 30 mm OU (2 visits) good nerves and fields
- No family history of glaucoma
- Takes statins and aspirin for cardiovascular risk
- Denies other medications

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IOP

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- January 2016 with latanoprost monotherapy 19 mm OU
- April 2016 43 mm Hg OU
- Add Combigan BID OU and IOP mid 20's
- June 2016 22 mm Hg OU
- Sept 2019 IOP mid 40's

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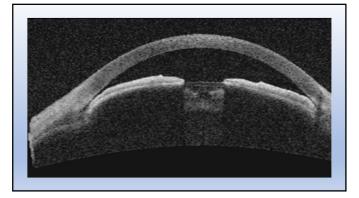
• Surgical consult – SLT's – similar post Tx fluctuation

October 2016

• Patient reports that he has had a recent steroid injection of dermatologic issues and has had several in past few years.







LPI vs. No LPI • Err on side of caution • LPI not totally benign • Preventing potential blindness • Cataract acceleration • Prevention of angle closure • Photophobia • Fairly benign

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Long Term Studies

- Recent Chinese Study 6 out of 485 patients closed
- 1993 Chicago Study 8 out of 129 patients closed
- India recent study 1 out of 48 patients closed

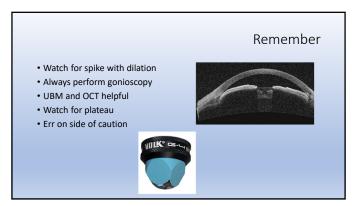
Closure with dilation

- Singapore 3 / 471
- Rotterdam 2 /149
- Baltimore 0 / 38

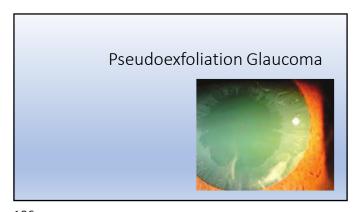
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Stages / Classifications

- Primary angle closure suspect ("occludable")
- Primary angle closure
- Primary angle closure glaucoma





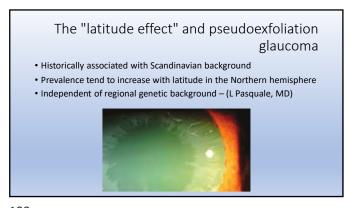


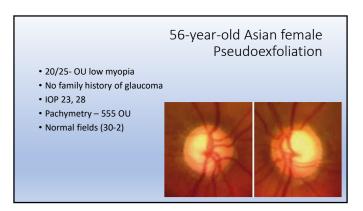
pseudoexfoliation glaucoma

- Systemic disorder caused by progressive accumulation of extracellular material over various tissues.
- Weakness of the zonules cause of complication during cataract
- Angle changes common.
- Pigment and flecks of pseudoexfoliative material in angle "Sampaolesi's line"
- Mostly bilateral and asymmetric
- Historically associated with Scandinavian background
- Prevalence tend to increase with latitude in the Northern hemisphere

pseudoexfoliation glaucoma – big rule..... DILATE! Or you WILL MISS IT!

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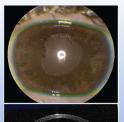




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• Uveitic Glaucoma

- Common complication of uveitis affecting some 20% of patients
- Inflammatory debris obstructs trabecular meshwork
- Secondary angle-closure glaucoma can result – iris bombé
- In the long-term, inflammation can also cause scar tissue that further obstructs outflow
- Usually treated with corticosteroids
- Watch for High IOP with iritis!
- Always suspect herpetic component and consider herpetic prophylaxis





Fuchs' Heterochromic Iridocyclitis

- Rare, chronic form of iridocyclitis characterized by iris heterochromia, lowgrade anterior chamber reaction with small stellate keratic precipitates, posterior subcapsular cataract and secondary open-angle glaucoma.
- Usually unilateral, affecting the hypochromic eye, and affects men and women equally in the third to fourth decade.
- Open angle without synechiae
- Does not respond to corticosteroid therapy, and corticosteroids may worsen the IOP elevation.
- Treatment is initiated with medical therapy.
- Frequently fails to be controlled medically and often requires filtration.

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Glaucomatocyclitic crisis or Posner-Schlossman syndrome

- Recurrent attacks of mild anterior uveitis with marked elevations of IOP
- Affects young to middle-aged adults, who present with blurred vision and eye pain.
- Resolves spontaneously within a few weeks.
- IOP is usually markedly elevated (in the 40- to 60-mmHg range)
- Returns to normal between attacks.
- Chronic secondary glaucoma may develop.
- Gonioscopy may reveal keratic precipitates on the trabecular meshwork, suggesting trabeculitis as the etiology of the elevated IOP. Another theory suggests that increased levels of aqueous prostaglandins may increase aqueous production.
- Treatment consists of corticosteroids and antiglaucoma medications during episodes.

60 mm Hg OU

- 38 y/o female African American
- Grade 4 angles
- .4 c/d OU
- 1+ chamber reaction
- Schlemms canal injected

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Juvenile rheumatoid arthritis (JRA)

- Most known cause of childhood uveitis
- Frequent cause of uveitic glaucoma
- Glaucoma is reported to occur in up to 44 percent of patients
- Often require surgical intervention

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IOP Emergency IOP 35+, 45+, 50+

- Angle Closure
- Acute steroid response
- Pigmentary storm
- Uveitic Spike
- Combigan q 30 minutes
- Prostaglandin 1X
- Diamox 500 MG then 250 BID
- Dilation and q4h pred 1% with uveitic
- Pilocarpine 1% q 30 minutes with closure
- Probable surgical referral

Thank You!

Elliot M. Kirstein, OD, FAAO

Harper's Point Eye Associates Glaucoma and Diabetes Eye Institute

Cincinnati, Ohio