Glaucoma Diagnosis and Treatment

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Glaucoma Update

➤ References
➤ Epidemiology / Definition
➤ Risk Factors
➤ Clinical Evaluation
➤ Visual Fields / Scanning Technology
➤ Treatment Goals
➤ Medications
➤ Cases

Excerpts From:
The International Glaucoma Review,
World Glaucoma Congress

The “International Glaucoma Review: The Journal of the World Glaucoma Association” (www.glaucom.com) is published every four months. This expert publication reviews the world glaucoma literature from the previous four months and provides abstracts and reviews of the most salient information from that time period in a single publication. We are pleased to provide for you, our colleagues in optometry, selected quotes (or in-context paraphrases), and our commentaries.

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Glaucoma Today - FREE

Website: www.glaucomatoday.com
Telephone: 484-581-1830
Glaucoma - Epidemiology

- One of the leading causes of blindness in the world (2nd)
- 67 million people in the world have glaucoma (10% blind)
- 3 million in U.S. have disease (half undiagnosed)
- Estimated 120,000 Americans blind from glaucoma
- Leading cause of irreversible blindness among patients of African descent
- 10-20 million glaucoma suspects
- Over 8 million office visits per year

Underdiagnosis of POAG

- Population studies suggest over half of all glaucoma patients have not been diagnosed
- From the Baltimore Eye Study: One-half of all people who were found to have glaucoma had seen an eye doctor within the past year and were unaware they had glaucoma!

Risk Factors For POAG

- Suspicious ONH cupping
- Elevated or increasing IOP
- Subnormal central corneal thickness (CCT)
- Advancing age (particularly after 50)
- African or Hispanic origin
  - Onset earlier (about 10 years), damage more severe, treatment less successful
- Positive family history (age at Dx?)
- Diurnal fluctuation?
- High myopia

“Chase the Family”

“First-degree relatives of identified OAG patients should be evaluated with optic disc and visual field testing.”

“Among existing OAG patients, 1 in 8 has a living relative with undiagnosed glaucoma. We must be more aggressive in recommending examinations for family members of OAG patients.”

“Screening in vans in the community may make us feel good but chasing family members is a more cost-effective method to find some of the 50% of OAG patients who are presently undiagnosed. We must not only 'take a family history,' but take the initiative in following up on family members.”

Harry A. Quigley, M.D. Archives of Ophthalmology, July 2006
Is Diabetes a Risk Factor For Glaucoma?

- “In this study (in the Netherlands), no association between diabetes and glaucoma was detected. This was in line with two other prospective studies.”
- “If there is any effect of diabetes, then it will be small, and protection is at least as likely as a negative influence.”

Reference: Ophthalmology, October, 2006

Is IOP Fluctuation a Risk Factor For the Development of Glaucoma?

“Long-term IOP fluctuations do not appear to be significantly associated with the risk of developing glaucoma in untreated ocular hypertensive subjects.”


OHTS Summary of Practice Implications

- Risk for progression of ocular hypertension to POAG can be assessed
  - Age, IOP, vertical C/D ratio, CCT
- CCT should be measured in all patients with ocular hypertension and all glaucoma suspects
- Patients at high risk should be treated
- Therapy should be selected based on efficacy, tolerability, and likelihood of patient compliance

Perspective on Central Corneal Thickness (CCT)

- CCT has become “standard-of-care” in the POAG (or suspect) work-up
- Thinner corneas are a strong risk factor for POAG because true IOP is actually higher than the measured IOP.
- Some patients with measured ocular hypertension may simply have a thicker CCT, thus reducing POAG risk because the true IOP is actually less than the measured IOP
- CPT-4 Code 76514
**Risk of POAG in Observation Group by CCT and Baseline IOP**

![Bar chart showing the risk of POAG based on CCT and baseline IOP.](chart1)

*The number of participants varies in each observation group.*


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**Risk of POAG in Observation Group by CCT and Baseline Vertical C/ D Ratio**

![Bar chart showing the risk of POAG based on CCT and vertical C/D ratio.](chart2)

*The number of participants varies in each observation group.*


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**Role of CCT and Glaucoma**

"Thinner CCT may be a significant, independent risk factor for open-angle glaucoma among persons with ocular hypertension."

"It is unclear whether the impact of CCT as a risk factor for glaucoma is mediated largely through its role in determining measured IOP, or whether the thickness of the cornea is a surrogate for greater susceptibility of the eye to damage."

*Reference: AJO, May, 2006*

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**Perspective on Pachymetry**

- “The World Glaucoma Association panel considered ways to measure IOP accurately. In particular, the measurement of central corneal thickness was identified as crucial.” (Review of Ophthalmology, Aug. 2007)

- “Almost 50% of OHTS participants had corrected IOP’s below the recruitment threshold, suggesting that many of the participants may never have been at much risk of developing glaucoma.” (Ophthalmology, Nov. 2007)

- “CCT is the most heritable aspect of ocular structure (more than refraction, axial length, or optic disc size), suggesting that it is under exquisite genetic control.” (Ophthalmology, Nov. 2007)
The general clinical evaluation of a new glaucoma suspect / patient

- This clinical evaluation builds upon a careful family history, personal medical history, current health status, and medication(s)
- Best corrected vision
- Document pupil size and reactivity
- Careful slit lamp biomicroscopy noting A/C depth, any iris abnormalities such as pigment dispersion, retroillumination defects, pseudoexfoliation, corneal guttata, etc.
- Applanation tonometry, noting time
- Pachymetry to determine CCT

Glaucoma Work-Up (continued)

- Baseline gonioscopy (4-mirror preferred) looking for PAS, angle recession, angle pigmentation, and the anatomic patterns of the angle anatomy
- Thorough BIO to r/o any peripheral pathology
- Stereoscopic evaluation of the optic nerve heads (60D, 78D, or Hruby lens); glaucoma detected most often through dilated pupils
- Baseline static threshold visual fields
- Image analyzer of optic nerve head (GDX-VCC/OCT)
- Optic disc photographic documentation

Breakthrough on Gonioscopic Training

- A most wonderful website exists to help teach superb gonioscopic anatomy and technique
- Please seek and study:
  www.gonioscopy.org

Optic Nerve Head Evaluation

- Cup depth is critical - Stereopsis!
- Are cup walls steep or sloping?
- Note rim translucency and vertical elongation of the cup
- Is the cup concentric with the disc, or is the cup displaced?
- Is the neuroretinal rim thinned more at certain clock hours than others? Especially look for any accentuated erosion of the inferotemporal or superotemporal regions.
- Is the disc generally pink, yellowish, or pale?
ISN’T

- Helpful diagnostic observation in ONH evaluation
- Normal neuroretinal rim anatomy follows the ISN’T rule
  - Inferior rim should be thickest
  - Superior rim is slightly less thick
  - Nasal rim is slightly less thick
  - Temporal rim should be the thinnest
- Most ONH’s are round or slightly vertically oval
- ISN’T rule may not hold if ONH horizontally oval

Optic Disc Size and Glaucoma

- Bergtson (25 yrs ago)
  - Normal small discs have small cups
  - Normal large discs have large cups.
- Average disc diameter 1.5 mm

<table>
<thead>
<tr>
<th>Disc Diameter</th>
<th>Mean C/D</th>
<th>Upper Limit</th>
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</thead>
<tbody>
<tr>
<td>Small</td>
<td>1.0-1.3mm</td>
<td>.35</td>
</tr>
<tr>
<td>Medium</td>
<td>1.4-1.7mm</td>
<td>.45</td>
</tr>
<tr>
<td>Large</td>
<td>1.8-2.0mm</td>
<td>.55</td>
</tr>
</tbody>
</table>

- Implications for glaucoma diagnosis and management
  - A high ratio may not be pathologic
  - C/D’s for large discs change by a smaller amount
  - C/D changes caused by glaucoma occur more slowly in large discs than in small discs (baseline photos large discs especially important)
  - C/D asymmetry is not always pathological

Sizing the Optic Nerve Head

- There is poor agreement between slit lamp ophthalmoscopy, HRT, and OCT in classifying disk size as small, average, or large.
- Jonas proposed that in routine practice, the clinician conduct “a quick, crude estimate of whether the disk in question is average-sized (medium), smaller-than-average, or larger-than-average.”

Reference: AJO, September, 2006, pp. 375-379

ONH Hemorrhage

- Highly specific for glaucoma
  - Commonly inferotemporal in POAG
  - Commonly superotemporal in NTG
- Prevalence higher in NTG (20-35%)
- Disc hemorrhages may precede a VF defect or a change in nerve head
- Ominous sign in glaucoma patients
- Associated with aspirin use and diabetes

(Ophthalmology 09/04)
Glaucoma - Visual Fields

- Program Strategies
- Perspectives on Perimetry
- Visual Field Interpretation
  - Foundational guidelines
  - Catch trials
  - Grey scale
  - Total and Pattern deviation
  - Glaucoma hemifield test
  - Global indices
  - Summary
- Plaquenil Visual Field Testing

Short Wavelength Automated Perimetry (SWAP) (SITA-SWAP)

- Uses blue target on bright yellow background
- Theorized to detect functional loss earlier than white on white
- Can be used with an HVF-II units only
- Advise to look for:
  - localized color change (from yellow to violet)
  - an achromatic spot
- If patient has 20/30 or worse NS, use W on W
- If patient is felt to have moderate to advanced glaucoma, use white on white
- SITA-SWAP-much enhanced

SITA-SWAP VS SITA-FAST

- “Surprisingly, there was no significant difference between conventional SAP and the 2 SWAP programs in diagnostic sensitivity.”
- “SITA-Fast has previously been reported to be able to identify at least as much significant glaucomatous field loss as SITA Standard . . . and to have a diagnostic sensitivity of more than 90%.”
- “Our results indicate that there may be some differences in sensitivity between SWAP and SAP, but that those differences are probably smaller than what has been believed previously.”
- SWAP 12 min - - SITA-SWAP 4.1 min - - SITA-Fast 3.5 min.

Humphrey Matrix

- 2nd generation frequency doubling technology
- Original FDT: tests 17 points with 10 degree targets
- Matrix 24-2 equivalent: tests 55 points with 5 degree targets
- Has 5 threshold tests: N-30, 24-2, 30-2, 10-2, and macular test
- Has small footprint, easy to operate, floppy & CD drives
- May well be best perimeter available and relatively inexpensive
**SITA vs Matrix Study**

- No significant difference in outcomes
- “Each visual field test tended to identify different subrsets of eyes with glaucomatous-appearing optic discs as abnormal.”
- Functional changes in glaucoma detection may differ significantly between individuals
- Matrix may not detect more extensive damage as compared to SITA

**Ultrasummary**

- A combined cerebral assessment of:
  - Pattern Deviation probability plots as compared to Total Deviation probability plots
  - Pattern Standard Deviation probability values
- These probability plots give the greatest VF data guidance to the functional status of the patient’s optic nerves
- **Remember:** ALWAYS CORRELATE THE CLINICAL FINDINGS WITH THE VISUAL FIELD STUDIES!

**Perspective on Progression**

“Because fluctuation in visual sensitivity is a confounding factor, if a follow-up visual field test discloses progression, it must be repeated at least once – preferably twice – before you conclude that the deterioration is indicative of true progression. Most clinicians know from experience that patients often exhibit apparent visual field progression on a single test, only to have the next test clearly demonstrate stability relative to the baseline visual fields, and multiple clinical trials have confirmed this.”

Rev of Ophthal, January 2008

**Glaucoma Progression Analysis**

- New software program for Humphrey perimeters
- Statistically analyses all points for progression
- Can be immensely helpful in detecting VF progression
- Can be done both retrospectively and prospectively
- A new standard in detecting progression
- Available from Carl Zeiss-Meditec
Optic Nerve Head Image Analyzers
- GDX-VCC, OCT-3, HRT, RTA, etc.
- Can be helpful in early diagnosis
- Limited value in advanced glaucoma
- Excellent for detection of progression
- A COMPONENT of the glaucoma evaluation
- Not a "litmus test" for glaucoma

Update on Scanning Devices
- "The OCT, GDx, and HRT performed as well as, but not better than, qualitative evaluation of optic disk stereo photographs for detection of early perimetric glaucoma."
- "The three imaging devices performed as well as, but not better than, the evaluation of optic disk photographs by glaucoma specialists."

AJO, November 2007

Glaucoma vs Suspect
- SH 46 yowf referred by an optometrist for 2nd opinion regarding POAG
- Only child – no family hx glaucoma – 2nd eye exam
- BVA 20/20 20/20
- Grade I V-H angles
- IOP by TA 19mmHg 4:40 pm
- 9OD oph: 0.75 c/d with mild IT thinning OU
- CCT: 599 OD 565 OS
- HVF: A few spotty scotomas inferiorly – OU
- GDX-VCC: Thick NFL, NFI 23/26, all parameters WNL-OU

Treatment Goals For POAG
- Set target IOP range when initiating therapy
- General guideline: reduce IOP by the % of the pretreatment baseline IOP. (Example, if the initial IOP is 30 mmHg, try to reduce IOP by 30%, yielding a target IOP of approximately 20 mmHg.)
- Modify target IOP according to the stage and severity of the disease
- Re-evaluate and adjust over time (years)
Target Range of IOP

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Expert Perspective on “Target IOP”

“Estimation of target pressure is based on a patient’s risk factors for progression, the level of IOP that caused damage, the severity of disease, and longevity. There is, of course, no way to determine in advance which IOP will be safe. There is no evidence that setting a variable target has clinical value for most patients with chronic glaucoma.”


The Monocular Trial

- “To determine the effectiveness of topical therapy, it is necessary to distinguish between the therapeutic impact of an agent on IOP and ordinary background fluctuations of IOP. When starting a new topical agent, it may be useful to begin by treating only one eye and comparing, at follow-up visits, the relative change of the IOP in the two eyes. If a drug fails to reduce IOP, it should be discontinued.”
- Schedule monocular trial f/u visit about same time of day as initial visit to control for diurnal variation

Glaucoma Treatment Options

- Prostaglandin Analogs
- Beta-Adrenergic Blockers
- Prostaglandin / Beta-Blocker combinations
- Adrenergic Agonists
- Adrenergic Agonist / Beta-Blocker combination
- Carbonic Anhydrase Inhibitors (CAI’s)
- CAI / Beta-Blocker combination
- Pilocarpine derivatives
- Epinephrine derivatives
- Laser Trabeculoplasty
- Surgical Trabeculectomy
Prostaglandin Receptor Agonists

- Latanoprost (Xalatan) 0.005%
- Travoprost (Travatan) 0.004%
- Travoprost (Travatan Z) 0.004%
- Bimatoprost (Lumigan) 0.03%

Prostaglandins

- 12 hour delay in maximum effectivity therefore best to instill between the evening meal and bedtime, but time of instillation not critical
- Maximum overall effectivity: 2 weeks. Do monocular trial and recheck in 2 or 3 weeks
- Iris color does not affect clinical effectivity
- BAK preserved: Guard against SPK, especially in patients with dry eyes
- Fully compatible with all others, except pilocarpine
- 90 drops per 2.5 ml (4-5 weeks of therapy)


Switching Drugs Within A Class

“Switching from one drug to another within the same class makes little sense unless the adverse effect profile has been proven to be different.”

“If you are thinking of switching drugs in the same class for IOP control, you should think again . . . Switching for decreased adverse effects, when IOP is controlled, is a reasonable approach. If a change in therapy is indicated, always consider a one-eye trial to avoid erroneous result.”

Dan Eisenberg, M.D., Ophthalmology Times, May 15, 2002

Combining the Prostaglandins

“The combination of bimatoprost and latanoprost in POAG increases the IOP and should not be considered as a therapeutic option.”

Lumigan-Induced Periocular Skin Changes

- Time to onset: 3 to 15 months
- Time to resolution following discontinuation: 3-9 months
- “Reversibility of prostaglandin-induced periocular hyperpigmentation is in contrast to the irreversible or very slow reversible nature of prostaglandin-induced iris hyperpigmentation.”
- Mechanistic explanation: dermal melanocytes are continent relative to melanin granules, whereas iris melanocytes are incontinent
- Switching to another prostaglandin may or may not evoke a lessened expression

Reference: Ophthalmology, November 2006

Latisse

- Formulation same as Lumigan
- Mechanism: Latisse takes a lash in the telogen (resting) phase and moves it into the anagen (growth) phase.
- The cycle of lash follicles is about 5 months. Small changes are seen at about 2 months.
- Comes with sterile single use brushes to apply the drop along the upper lash line
- Can use Latisse for contact lens patients when lenses out
- May be used on chemo patients
- Available by Allergan - 3 ml bottle in kit with 60 disposable brushes

Improvements in Eyelash Length

Mean change in eyelash length from baseline

Week 1 4 8 12 16 20

LATISSE

Vehicle

_mean change in length

Bimatoprost-treated subject representing mean change in length

Mean Change in Eyelash Length from Baseline (mm)

* * ** p<0.001

GI Problems with Prostaglandins

- Acute GU distress/gastric burning can occur with the prostaglandins
- Usually occurs shortly after initiation of treatment
- Symptoms rapidly self-limit upon discontinuation
- Thought to involve GI smooth muscle activity
- “Although apparently a rare effect, GI distress with PG use should be a consideration for patients given these medications and returning with such complaints”

Archives of Ophthamology, May 2008
**Topical Beta-Andrenergic Receptor-Blocking Drugs**

- Timolol (Timoptic and Timoptic XE / Betimol) 0.25% and 0.5%; (Istalol) 0.5%
- Levobunolol (Betagan) 0.25% and 0.5%
- Metipranolol (Optipranolol) 0.3%
- Carteolol (Ocupress) 1.0%
- Betaxolol (Betoptic-S 0.25%)
  - Have longer half-lives than other beta-blockers

**Topical Beta-Blockers**

- Decrease aqueous production
- Reduces IOP 25%; no response 15%
- R/O asthma
- Recommend monocular trial with lowest concentration once daily
- Possible diminished effect if used with systemic beta-blockers
- Monitor resting heart rate at baseline and follow-up

**Timolol Hemihydrate**

- Unique form of timolol
- Clinically equivalent to timolol maleate
- Available in 0.25% and 0.5%
- Can be used once daily for most patients (a.m. instillation)
- Relatively inexpensive beta-blocker
- Not generically substitutable
- Betimol ophthalmic solution by Vistakon Pharmaceuticals

**Meta-analysis of Drug Efficacy**

“The results of this study show that prostamide or prostaglandin analogs are most effective for lowering IOP by monotherapy in POAG or OH patients. However, the beta-blocker Timolol is almost as effective and, thereby still a good treatment option. The beta blocker betaxolol, alpha 2-adrenergic agent brimonidine, carbonic anhydrase inhibitors, brinzolamide, and dorzolamide are clearly less effective.”

**Adrenergic Receptor Agonists**

- Brimonidine
- Apraclonidine
- Dipivefrin
- Epinephrine

**Brimonidine Tartrate**

- Alpha-2 adrenergic agonist; tid FDA approval
- Acts by reducing aqueous production with some enhancement of uveoscleral outflow
- Reduces IOP similar to timolol 0.5% bid
- Side effects: fatigue and dry mouth most common side; uveitis reported; may reduce systolic BP 10 mmHg
- Less tachyphylaxis or allergy development than the other alpha-2 agonists
- Neuro-protective potential unknown
- Alphagan (0.2%) by Allergan, generic Alphagan P (0.15%) by Allergan and Alphagan P (0.1%) by Allergan

**Combigan Ophthalmic Solution**

- Combination of 0.2% brimonidine and 0.5% timolol
- Approved by FDA in 2007
- Indicated in patients with glaucoma or ocular hypertension who require adjunctive or replacement therapy due to inadequately controlled IOP
- Combigan effects on IOP:
  - Reduced IOP up to 33% (7.6 mmHg) from baseline
  - Up to 29% (6.9 mmHg) from baseline when added to a prostaglandin
  - Up to 25% (5.7% mmHg) when added to a beta-blocker
- Ocular allergy rate 5.2%
- Available in 5, 10, and 15 ml bottle

**Philosophy on Combination Drugs**

- Never initiate therapy with a combination
- Always try one of the ingredient drugs first
- Add a second drug via a combination product if initial single drug failed to meet target pressure…
- …, or could also try the other ingredient drug as a therapeutic trial (if the initially selected ingredient drug was inadequate in achieving target pressure range)
Carbonic Anhydrase Inhibitors (Topical and Systemic)

- Dorzolamide (Trusopt)
- Brinzolamide (Azopt)
- Acetazolamide (Diamox)
- Methazolamide (Neptazane)

Topical CAI’s

- Dorzolamide 2% sol. and Brinzolamide 1% susp.
- Mechanism: decreases aqueous humor secretion
- Reduces IOP approximately 15%
- FDA dosage: tid, practical dosage bid
- Contraindications: Allergy to sulfa and/or history of blood dyscrasias
- Side effects: minimal; some burning, bitter taste, rare allergic reaction
- Most all patients controlled with oral acetazolamide were successfully controlled with a topical CAI
- Azopt 1% susp-Alcon; Trusopt 2% sol-Merck

Dorzolamide Hydrochloride 2% - Timolol Maleate .5% (Cosopt)

- Both components decrease IOP by reducing aqueous humor secretion
- Because of the CAI, must be used bid, which results in excessive beta-blocker therapy
- Contraindications: patients with asthma, heart disease or COPD; allergy to sulfa drugs
- Ocular side effects: burning/stinging and perversion in taste
- Marketed as Cosopt by Merck and generically available October 08

Contemporary Glaucoma Medication Flow

1st Tier: Prostaglandin qd (Lumigan, Travatan Z, or Xalatañ)
Beta-Blocker 0.25% or 0.5% qd

2nd Tier: CAI or Brimonidine

3rd Tier: Cosopt or Combigan

4th Tier: Pilocarpine (preferably Pilopine HS Gel)
Dipivefrin
Oral CAI (preferably Methazolamide)
Laser Trabeculoplasty

- SLT targets pigmented trabecular meshwork
- SLT requires $60,000-$80,000 investment
- ALT / SLT are equal in efficacy and side-effects
- “There is currently no data to support that retreatment with SLT is superior to ALT.”


Complementary and Alternative Medicine for Glaucoma

- “Mega doses of vitamins do not benefit patients with glaucoma”
- Bilberry does not “improve night vision or contrast sensitivity”
- “The effects of Chinese herbs on human glaucoma are unknown”
- Marijuana’s systemic toxicity and short half-life make it a “poor treatment option”
- Ginkgo has not been proven beneficial (antiplatelet effect)
- Diet or body type has no influence on glaucoma
- Exercise “can be mildly effective in lowering IOP or visual field.”
- “There is no evidence that relaxation techniques lower IOP.”
- Summary: “There is little evidence to support prescribing complementary and alternative medicine for the treatment of glaucoma.”

Source: Glaucoma Today, Step-Oct 2006

Glaucoma During Pregnancy

- “It is well documented that IOP decreases during pregnancy in healthy women.” (More pronounced during 2nd & 3rd trimesters.)
- Brimonidine – Cat. B; all others Cat. C.
- “In our experience, obstetricians are most comfortable with the use of beta-blockers because this class of medications is used to control hypertension during pregnancy.”
- Avoid prostaglandins because of potential (probably very low) to induce labor.
- All care must be individualized.

Reference: Archives of Ophthalmology, August 2006

Angle Closure Management Options

- 500 mg of Diamox* in tablet form
- Alphagan q 15 min x 2
- A beta-blocker, q 15 min x 2
- Pilocarpine 2% once above steps completed
- Potent steroid q 1h if there is much associated inflammation
- YAG photoiridotomy once control is achieved
Sulfa Allergy Perspective

“This study shows that although there is an association between hypersensitivity after receipt of sulfonamide antibiotics and a subsequent allergic reaction after receipt of a sulfonamide nonantibiotic, the association appears to be due to a predisposition to allergic reactions rather than to cross-reactivity between sulfonamide antibiotics and sulfonamide nonantibiotics.”


Alert on Topiramate (Topamax)

- Approved 12-96 for seizure disorders
- Unapproved: Migraine HA, weight loss, depression, bipolar disorder
- Mechanism of action is unknown
- At least 115 reported cases of acute, bilateral, secondary angle-closure glaucoma
- Onset usually within first 2 weeks of therapy
- Most common presenting symptom: blurred vision
- Exact mechanism of increased IOP is unknown
- Tx: Stat consult with prescribing physician to begin to reduce topiramate dosage; then aqueous suppressants, oral CAI, cycloplegia (retracts ciliary body) - no miotics
- IOP normalizes in 1-4 days, no laser treatment indicated

The Secondary Glaucomas

- Pigmentary Glaucoma
- Pseudoexfoliation
- Glaucmatocyclitis Crisis

Pigment Dispersion Syndrome (PDS)
(Pigmentary Glaucoma)

- Predominately affects young, myopic men
- About 50% of cases progress to pigmentary glaucoma
- Concave iris rubs zonules releasing pigmentation particles
- Pigmented debris can overwhelm trabecular meshwork
- Gonioscopy reveals dense trabecular pigment band
- Iris retroillumination defects is hallmark finding
- Laser photoiridotomy is helpful with PDS, but plays little or no role once pigmentary glaucoma has occurred
Pseudoexfoliation Syndrome

- Increases in older age – represents about 5%+ of all the glaucomas
- If unilateral, will become bilateral over 5-10 years
- When glaucoma is present, IOP tends to be higher and more difficult to control
- Whitish flakes at pupil border and lens face
- Gonioscopy – looking for trabecular pigmentation (Sampaolesis’s Line)
- No systemic association – not inherited

Glaucmatocyclitic Crisis (Posner-Schlossman Syndrome)

- Acute (recurrent), unilateral, open-angle glaucoma associated with mild uveitis in a white eye
- Most commonly occurs in patients age 20-50 years
- Self-limited disease
- Symptoms: none to mild discomfort
- Signs: IOP usually 40-60 mmHg, few cells in anterior chamber - some small KP corneal edema (haloes), none to mild redness
- Treatment: steroid, beta blocker, alpha adrenergic, cycloplegia