Glaucoma: Clinical Pearls for Challenging Cases

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Case
- 34 yo, white, male
- Ant Segment:
  - K-Spindle, + ITD, 3+ TM pigment
- GAT = 28/31 mmHg OD/OS
- Pachymetry = 595 µ

Disclosure
- Michael Chaglasian has the following disclosures:
  1. Advisory Board: Allergan, Inc., Alcon Labs, Carl Zeiss Meditec
- The content of this presentation is in no manner influenced by any of the aforementioned parties or companies

Agenda
- Common Secondary Glaucomas
- Ocular Perfusion Pressure
- Ocular Hypertension
- Adjunctive Therapy Options
- Suspicious Optic Nerves

Gonio and Slit Lamp Photos
Clinical Pearls for Challenging Cases

PIGMENTARY GLAUCOMA

- see clinical triad of:
  - Krukenberg's spindle
  - Iris transillumination defects
  - heavy TM pigmentation
- PDS refers to those with these clinical findings but without any glaucomatous changes to the optic nerve
- glaucoma may develop in up to 50% of patients with PDS
  - Typically only 10-15%, literature shows wide variation

Case

- Assessment
  - PDS, High IOP, thick Pach
  - Normal ONH, Normal VF, Normal OCTs

- Plan / Discussion
  - Other Tests?
  - Treatment Trial: ______________
  - Other considerations: ________________
Mechanism
- Photogrammetric and histological studies have shown that a concave posture (posteriorly) of the peripheral iris allows for rubbing against the lens zonules
- **Reverse Pupillary Block** concept

Exercise-induced pigment liberation:
- Pharmacologic pupillary dilation may result in marked pigment liberation accompanied by a rise in IOP.
- The same phenomenon may occur in some patients with PDS during strenuous exercise, particularly exercise involving jarring movements, such as jogging or basketball.
- Pretreatment with low-dose pilocarpine prior to exercise can limit both the pigment liberation and the IOP spike.
Pigmentary Glaucoma

- The glaucoma part of this condition has the same requirements as POAG, that is ONH damage and or VF loss

Management

Management of PG has two strategies:

1. Control of elevated IOP and prevent glaucomatous damage (primary)
2. Eliminate irido-zonular contact
   - If this is possible, it can reduce pigment liberation and over time allow the TM to get rid of the pigment granules

Laser Trabeculoplasty

- Argon or SLT
- Both work well at first,
  - With up to 25% decrease in IOP, but then quickly lose treatment effect, often within 2-3 years

Laser Iridotomy

- Is another treatment option for PDS/PG.
  - It eliminates the "reverse pupillary block"
  - By allowing direct aqueous flow into the AC, an equilibration of pressure is obtained which may help to move the peripheral iris off the lens zonules.
- Only for active PDS, not for older patients.
- Medications are tried first

PIGMENTARY GLAUCOMA

- Prostaglandins
  - Primary, first line therapy, best overall
- Alternatives:
  - Brimonidine, CAI, Beta-Blocker, Fixed-Combination
- Use these and look for a significant decrease in IOP.
  - Still monitor pigment liberation via slit lamp and gonio.
- Many patients require multiple meds

PIGMENTARY GLAUCOMA

- MIOTICS are a theoretical drug of choice in PG because of the mechanics:
  - Lifting the peripheral iris iris off the zonules
  - However their ocular side effects limit their success:
    - Low % (0.5) Pilocarpine solution
- Since they are poorly tolerated in young patients, they often cannot be used
  - Only, helpful in pigment liberation stage (early)
Clinical Pearls for Challenging Cases

Prognosis = Caution

- Some patients with PG:
  - Have a severe, difficult to treat form, of the disease and end up with severe vision loss at an early age
  - Are diagnosed at a late stage because of low suspicion of glaucoma in younger patients and high fluctuation in IOP (patients seen with normal IOP)

Case

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  - Other Tests?
  - Treatment Trial: ______________
  - Other considerations: ______________

Case JB

- 54 yo WF
- Referred in for Glaucoma Suspect
- No significant Medical History
- GAT = 24 OD and 23 OS
- Pachs = 560 and 565
- Gonio =
  - Open angle with moderate pigment

There's a Facebook page for Pigmentary Dispersion Syndrome??

Guess how many “friends”?
Clinical Pearls for Challenging Cases

Slit Lamp

Summary / Questions

- Does this patient have glaucoma?
- If not, how high is the risk for developing glaucoma?
- What other tests need to be done?
- When do you see this patient back?
- When/How do you start treatment?
- What is the prognosis for this patient?

Terminology

- Exfoliation Syndrome – XFS
- Exfoliation Glaucoma – XFG

- “Psuedoxfoliation” term is still used by some → “PEX”, “PXS”, “PXG”

Introduction and Background

- Exfoliation syndrome (XFS) is the most common identifiable cause of open-angle glaucoma worldwide.
- It is characterized by excess synthesis and progressive accumulation of abnormal extracellular fibrillar material
  → The continuous accumulation of exfoliation material (XFM) and pigment in the outflow system leads to elevated intraocular pressure (IOP) and eventually to the development of exfoliative glaucoma (XFG)
### Introduction and Background

- It has been estimated that the worldwide number of individuals with XFS is between 60 and 70 million, or equal to the total number of people in the world with glaucoma.
- About 25% of persons with XFS have elevated IOP, and one-third of these have glaucoma.
- Taking all epidemiological studies as a whole, XFG appears to account for about 15–20% of open-angle glaucoma in Caucasians.

### XFS in the Eye

- Classic Three Zones
- XFM frequently found at the pupillary border
- Pigment loss from pupillary ruff and deposition in TM
  - “Sandpaper” effect
  - Iris transillumination at margin
- Increased IOP post dilation
  - Via pigment liberation

### Genetics = LOXL1

- **Lysyl oxidase-like 1 gene (LOXL1)**
  - genomewide association study of 16,000 Icelandic and Swedish participants,
  - two mutation of single base differences in the sequence of the LOXL1 gene
  - accounted for 99% of all cases of exfoliative glaucoma (although not everyone with them gets glaucoma)
  - Same findings found in a recent US study
- lysyl oxidase family of genes is necessary for the formation and maintenance of elastic tissue, and the Extra Cellular Matrix (ECM)

LOXL1

- the effect of the genetic variants now detected in the LOXL1 gene seems to be to **lower the production rate of the protein it specifies**.

- slightly lower production rate of LOXL1 may be harmless during most of a lifetime, the deficit may accumulate through many decades, explaining why exfoliative glaucoma is most common among the elderly.


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Genetic Testing

Our genetic health scans cover an ever growing range of conditions.

How?

http://www.decodeme.com

How Much $$$?

http://www.decodeme.com
XFS in the Eye

- XFG patients exhibit a greater fluctuation in the 24-hour IOP curve than patients with POAG
- lens subluxation, phacodonesis
  - weakened lens zonules
  - Potential complications in cataract extraction
- cataract formation
- poor dilation and posterior synechia
- narrow angles/angle closure

Elevated Homocysteine Levels

- Highly cytotoxic amino acid derived from methionine metabolism.
  - It is found in plasma, aqueous humor, and tear fluid in XFS with and without glaucoma
- Elevated HC levels are a recognized cardiovascular risk factor, get levels!!!!!
- HC levels are inversely associated with intake of folate and vitamins B2, B6, and B12
  - Potential treatment recommendation

Plasma Levels Of Homocysteine in XFG

Elevated levels (>14 µg/l) in up to 43% of patients with XFS/XFG.

Elevated Homocysteine Levels

Treatment for Hyperhomocysteinemia

- Potential treatment recommendation:
  - FOLTX™ tablets:
    » Folacin (Folic Acid) 2.5 mg
    » Pyridoxine (B6) 25 mg
    » Cyanocobalamin (B12) 2 mg

Does FOLTX have a proven benefit?

- Treatment benefits have not been demonstrated yet.
  - Folic Acid Treatment on High Risk Women. JAMA 2008
- So?
  - Does a Normal, Healthy patient with a new diagnosis of XFS/XFG need labs and work up?
    - Unclear at this time
    - With Risk Factors = YES!

Diagnosis

- Diagnosis made essentially as it is for POAG.
- Understand that the patient is a higher risk and thus treatment may be started earlier,
  but ALL patients with XFS do NOT need treatment.
**Medical Therapy**

- **Prostaglandins**
  - In a recent parallel diurnal study, latanoprost was slightly more effective than timolol in lowering IOP in eyes with XFG.
  - There was a trend for better diurnal IOP control with latanoprost and a significantly lower diurnal fluctuation and mean peak IOP with this therapy.
- **Beta Blockers**
  - Reports are mixed on the success of this class of medications.
- **CAI’s**
  - Dorzolamide has had mixed success in several studies.
- **Cholinergics**
  - 1% pilocarpine at bedtime!!!, stop the pupil from moving.

**Laser Therapy**

- ALT is particularly effective, at least early on, in eyes with XFG.
- The initial drop in IOP is greater in XFG.
  - Several studies, however, have reported a gradual reduction in success over time, with long-term success drops to approximately 35-55% at 3-6 years.
- Selective laser trabeculoplasty (SLT) may be an effective and safe alternative to ALT in the treatment of XFG.

**Surgical Therapy**

- The results of trabeculectomy in XFG are comparable to those with POAG.
- However, surgical complications are more common in patients with XFS.
  - Weakened zonular support may allow intraoperative lens movement or, in extreme cases, subluxation.
- The most important and the most difficult choice for treating XFG is still the decision for timely surgery.

**Key Points**

- Careful slit lamp exam pre/post dilation
- Frequent IOP checks for patient with the syndrome
- Work with PCP on Homocysteine levels
- Treat glaucoma more aggressively
- Note findings for cataract surgeon

**Case JB**

**Dx:**
1. Exfoliation Syndrome/Glaucoma
   - High Risk but No evidence of glaucoma damage at present

**Plan:**
1. IOP recheck 1-2wks, diurnal flux
2. Then start PGA qhs OU
3. Repeat testing in ~9 months
4. Patient to discuss with PCP to check homocysteine level at next visit

**Case WS**

- 75 yo male
- + HTN w/ multiple BP meds x 20+ yrs
  - 105/68 in office
  - 5’ 5”, 142 lbs
- CCT= 532µ
- Initial IOP 23 mmHg
  - Now repeatedly 11-13 mmHg over 5+ years
- Current Medication:
  - PGA
- Good compliance and follow up
9 Years Later. Can you see the change?

Stereo Photos

- Obtain Baseline Photographs
  - Stereo is preferred
    - Screen-Vu Stereo Viewer™
      - www.berzin.com
  - Read, Review, and Document in record
  - Repeat periodically, or when change is suspected

Guided Progression Analysis™ (GPA™)

Photos
Case WS
- Q: What is the Explanation?
  - Progression with IOP in low teens.
- Compliance?

Other Potential Risk Factors:
- 24 Hour IOP
  - Highest IOP in Nocturnal Period (midnight-5AM)
- DOPP
  - Diastolic Ocular Perfusion Pressure

Ocular Perfusion Pressure: Risk Factor for Glaucoma

Ocular Perfusion Pressure
- The differential between arterial BP and IOP
  - Ocular perfusion is regulated to maintain constant blood flow to the optic nerve despite fluctuating blood pressure and IOP
  - The major cause of reduced blood flow is thought to be secondary to vascular dysregulation in susceptible patients, resulting from abnormal/insufficient auto-regulation.

Ocular Perfusion Pressure (OPP) = BP - IOP
(BP is mean arterial pressure, diastolic BP, or systolic BP)

OPP and Glaucoma: Hemodynamics
- SPP = SBP – IOP
- DPP = DBP – IOP Diastolic Measure
  - easiest to use, best current evidence
- MPP = 2/3 mean arterial pressure – IOP
  - Arterial Pressure = DBP + 1/3 (SBP – DBP)
  - May best reflect perfusion physiology

Ocular Perfusion Pressure and Glaucoma

Lower Diastolic, Systolic, or Mean Pressure Reduces Perfusion Pressure
Higher IOP Negatively Impacts Perfusion Pressure
Perfusion Pressure Is a Result of A Delicate Balance Between IOP and Blood Pressure
Lower Perfusion Pressure Is Associated with Increased Risk for Open Angle Glaucoma

Low ocular perfusion pressure has been shown to be strongly associated with the prevalence of glaucoma progression in multiple population-based surveys:

- Baltimore Eye Survey (AA and Caucasian)\(^1\)
  - 6x excess of POAG in subjects with lowest category of Ocular Perfusion Pressure (OPP)
- Egna-Numarkt Study (Caucasian)\(^2\)
  - Lower Diastolic Ocular Perfusion Pressure (DOPP) associated with marked, progressive increase in frequency of POAG
- Barbados 4 yr Eye Study (African-Caribbean)\(^3\)
  - 4-year risk of developing glaucoma increased dramatically at lower perfusion pressure
- Proyecto Ver (Hispanic)\(^4\)
  - Found lower Diastolic Perfusion Pressure (DPP) associated with increased risk of POAG

POAG Risk Factors at year 9
Barbados Eye Study

OPP and Glaucoma Progression: Population Studies

- Lower IOP improves OPP
- Measure blood pressure on your patients
- Higher systemic BP improves OPP, but you do not necessarily want to raise BP:
  - Stroke #3 cause of death in US behind CVD & CA!
  - Avoid drugs that lower systemic BP beyond patient’s desired systemic control.
  - Communicate with PCP
  - Look for nocturnal hypotension.

Los Angeles Latino Eye Study

- Cross-sectional study of 6,357 Latinos, >40 years in Los Angeles, CA.
- Persons with low diastolic and systolic perfusion pressures had a higher risk of POAG.
- DOPP <50 mmHg, the prevalence of glaucoma rapidly increases linearly.

Clinical Control of OPP

- Lower IOP improves OPP
- Remains number 1 goal!!
- Measure blood pressure on your patients

Studies Summary

- These large studies provide strong evidence among different populations for the relationship between vascular deficits and the prevalence, incidence and progression of glaucoma
- Some Limitations,
  - no direct measure of ocular blood flow
  - Varied definitions of hypertension
Nocturnal Hypotension and OPP

- Low blood pressure (BP) at night, coupled with high IOP in supine position, compromise OPP.
  - ? Up to 50% of patients with HTN
  - Using systemic BP meds in the AM to minimize nocturnal hypotension makes sense.

- Using IOP lowering drugs that lower IOP while sleeping makes sense.
  - Avoiding IOP meds that LOWER systemic BP at night (beta blockers, alpha agonists) makes sense.

24 hr IOP Measure via SCL
SENSIMED Triggerfish® - Continuous IOP Monitoring

Back to Case

24 Hour Blood Pressure

Holter Monitor

Case WS

- DOPP=
  - DBP of 68 mmHg @ 2PM and IOP of 12 mmHg
  - Gives 56 mmHg
- Nocturnal BP with Holter Monitor
  - DBP @ 2AM = 58
- Nocturnal IOP (estimate)
  - IOP of 12 mmHg @ 2PM = ?? @ 2AM ~ 18 mmHg
- Nocturnal DOPP
  - 58 - 18 = **40 mmHg**, potentially a high risk
Case WS

- Is there anything else that can be done?

**Possibly:**
- Offer Nocturnal IOP control
- Offer Improved DOPP

Add a CAI  BID

Letter to PCP, explain OPP and Low BP related Risk

? Adjust BP Meds

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CASE AC

- 45 yo, woman
- Myopia, no sig. medical history
- + family history glaucoma
- GAT= 27 OD 25 OS
- Pachs = 565 µ

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Summary

- IOP Fluctuation
- Increased Nocturnal IOP
- Low Nocturnal Blood Pressure
  equals
- Low Diastolic Perfusion Pressure
  equals

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24-hour habitual IOP Azopt vs. timolol add-on efficacy

CASE AC

![Eye images](Image)

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VFVs
What do you do now?

Pachymetry

Correction Values

<table>
<thead>
<tr>
<th>Corneal Thickness (µm)</th>
<th>Correction Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>405</td>
<td>7</td>
</tr>
<tr>
<td>425</td>
<td>6</td>
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<tr>
<td>445</td>
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<td>465</td>
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<tr>
<td>685</td>
<td>-7</td>
</tr>
<tr>
<td>705</td>
<td>-8</td>
</tr>
</tbody>
</table>

Correction values according to corneal thickness of 545 µm

Conversion Charts: don't really work

NOT VALID!
Clinical Pearls for Challenging Cases

IOP and CCT

“Assuming that CCT can be used as a correction factor for GAT is a misinterpretation of the results of OHTS... that couldn't be further from the truth. Adjusting IOP based on CCT is attempting to instill a degree of precision into a flawed measurement. You may actually correct in the wrong direction. The issues related to the most accurate tonometry need to include the material properties of the cornea”

James Brandt, MD, Director, Glaucoma Services, UC Davis

Pachymetry: 3 Outcomes

- Thin: <555 µ High Risk
- Average: 555-588 µ No change in Risk
- Thick: >588 µ Low Risk

Applied to patients with ocular hypertension

Risk Calculator

Risk Calculator Outcomes:
Recommendation Guide to Patient Management (by expert panel)

5-Year Risk for Progression of OHTN → Glaucoma

<table>
<thead>
<tr>
<th>Risk Level</th>
<th>Range</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>&lt;5%</td>
<td>Monitor</td>
</tr>
<tr>
<td>Moderate</td>
<td>5%-15%</td>
<td>Consider treatment</td>
</tr>
<tr>
<td>High</td>
<td>&gt;15%</td>
<td>Treatment</td>
</tr>
</tbody>
</table>

The predictions derived using these methods are designed to aid but not to replace clinical judgment.

OHTS – RC Limitations?

- A number of factors described as predictive in previous studies either did not add to the explanatory power of the OHTS–EGPS pooled model or were not assessed in this study. These include:
  1. Family History
  2. Life Expectancy
  3. Diabetes (?)
  4. Race (?)

http://ohts.wustl.edu/risk/calculator.html

Also iPhone App
Case EG
- 67 yo, AA male, Retired school teacher
- Good health, no medications
- + Family History of glaucoma
- OHTN/Early Glaucoma
- CCT= 567, 571
- Pre-Tx IOP ~ 30 mmHg OD, OS
- With PGA:
  - Always 20-23 mmHg x 5+yrs
  - Good Compliance
Can you see the change?

Case EG Discussion
- Is this progression?
- Other things you’d like to see/do?
- Options for adjunctive treatment?

SECOND LINE AGENTS
Options for Adjunctive Therapy

**Products:**

<table>
<thead>
<tr>
<th>Product</th>
<th>Additional Mean IOP Reduction when Added to a PGA (at 3 months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed Combinations</td>
<td></td>
</tr>
<tr>
<td>Combigan®</td>
<td>6.9 mm Hg&lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>Cosopt®</td>
<td>5.2 mm Hg&lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td>Alpha-agonists</td>
<td></td>
</tr>
<tr>
<td>ALPHAGAN® P</td>
<td>3.3 mm Hg&lt;sup&gt;2&lt;/sup&gt;</td>
</tr>
<tr>
<td>Carbonic anhydrase inhibitors (CAIs)</td>
<td></td>
</tr>
<tr>
<td>Azopt&lt;sup&gt;2&lt;/sup&gt;</td>
<td>2.1 mm Hg&lt;sup&gt;2&lt;/sup&gt;</td>
</tr>
<tr>
<td>Trusopt&lt;sup&gt;3&lt;/sup&gt;</td>
<td>1.2 mm Hg&lt;sup&gt;2&lt;/sup&gt;</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td></td>
</tr>
<tr>
<td>Timolol</td>
<td>2.7 mm Hg&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
</tbody>
</table>


Criteria for the Choice of Adjunctive Therapy

- Incremental efficacy
  - The main reason for changing initial monotherapy is the need for additional IOP lowering<sup>1</sup>
  - The purpose of adjunctive therapy is to obtain target IOP<sup>2</sup>
- Other considerations
  - Compliance
  - Tolerability
  - Safety


Alpha Agonists

- Alphagan-P 0.1% (Allergan)
  - ↓ BAK → Purite (↓ toxicity)
  - Less ocular allergy
- Aqueous suppressant and:
  - ↑ uveoscleral outflow
  - ? Neuroprotection?
- Bid vs. Tid dosing
- NO Neuroprotection!

Beta Blockers as Adjunctive Therapy to a Prostaglandin Analogue

- Brinzolamide (Azopt)
- Dorzolamide (Trusopt)
  - Generic availability

- Consistent, moderate, monotherapy IOP reductions
  - (15-20%, 4 to 6 mm Hg)
- FDA Labeled as TID agents
Clinical Pearls for Challenging Cases

Post Dose: IOP Change from Baseline

Best Adjunctive Therapy?

Overall = CAI

Advantages of Fixed Combinations
- Dosing—1 drop vs 2 drops
- Convenience
- Potential to improve compliance
- No risk of washout from second drug
  - Washout impedes absorption, thereby reducing efficacy
- Possible cost savings
  - Only 1 copay


Adjunctive Therapy Conclusions
1) PGAs are often not enough
2) BB don’t work at night if measuring in the supine habitual position
3) An Expert panel Suggests:
   - Adding CAIs is an agreeable option to a PGA
   - Adding an alpha agonist is less agreeable
   - Adding Fixed Combination should be reserved for failure of adding a single agent ???

Timolol Fixed Combinations
- Cosopt®
  - Dorzolamide hydrochloride/timolol maleate solution
- Generic dorzolamide / timolol maleate ophthalmic solution

Fixed Combination: Combigan
- Combigan (Allergan)
  - Brimonidine 0.2% and timolol 0.5%
  - BID dosing
- Less allergy than brimonidine alone
  - timolol is a buffer

Introducing... SIMBRINZA™ Suspension
A fixed-dose combination of brinzolamide 1% and brimonidine 0.2%

- Additional 1-3 mm Hg IOP-lowering compared to the individual components\(^1\)\(^2\)\(^3\)
- Delivers 21-35% IOP-lowering efficacy\(^1\)\(^2\)\(^3\)
- Only fixed-combination without a beta blocker\(^1\)\(^2\)\(^3\)
- Adverse events profile consistent with those of its individual components\(^1\)\(^2\)\(^3\)
- Creates new treatment possibilities for lowering IOP

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Case GS
- 55 yo C, F, Seeking second opinion
- History of being treated for glaucoma
  - Currently on PGA and beta blocker
  - Was recommended to have laser trabeculoplasty
- No insurance currently
- IOP= 17 mmHg
  - Reports PreTx IOP around 21mmHg
- CCT= 555µ

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Photos

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Three-Month Randomized Trial of Fixed-Combination Brinzolamide, 1%, and Brimonidine, 0.2%

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Left SAP VFVs Right

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GDx
Case GS

- More aggressive treatment was recommended, including:
  - Adding Alphagan
  - Adding CAI
  - Switching to Cosopt (Combigan not available yet)
  - ALT/SLT

- Patient wanted a third opinion
Clinical Pearls for Challenging Cases

Next Steps
- Treatment Options
- Longer Term Considerations
  - What is the right target pressure?

Clinical Pearls
Optic Nerve Evaluation
“The 5Rs”
- Ring, Rim, RNFL, Region, Retinal disc heme

Adapted from FORGE program
R. Weinreb, F. Medeiros, R. Susanna

Scleral Ring and Disc Size
- Scleral Ring = Outer Disc Margin
  - First Step in Determining Disc Size

Scleral Ring and Disc Size
- At the Slit Lamp
  - Volk Lenses:
    - 60D = x 1.0
    - 78D = x 1.1
    - 90D = x 1.3

Scleral Ring and Disc Size
- Small
- Average
- Large
  - Disc/Cup Size ≠ Risk

http://www.optometryjaoa.com
Rule #2: Size of Neuroretinal Rim

- Rim Width = Distance between outside border of disc and bending of blood vessel on inner rim
- ISN'T Rule: Inferior > Superior > Nasal > Temporal
- Localized Notching / Thinning
- Color of Rim

Width of the NRR around the disc

Neuroretinal Rim

- Rim Width:
  Distance between border of disc and position of blood vessel bending

CASE EB

- 57 yo, woman
- Annual Exam
- Normal Slit Lamp
- Open Angles
- GAT= 21 OD 21 OS
- Pachs = 540 µ

Localized Rim Thinning/Notching

Case EB
Clinical Pearls for Challenging Cases

Five years later

Case EB
Questions / Discussion

mchaglas@ico.edu