TOPICS

- Glaucoma nuts and bolts
- Secondary Glaucoma
  - Blood induced glaucoma
  - All other secondary glaucomas
- If time allows
  - Angle Closure Glaucomas
  - Glaucoma Pearls
  - Surgery videos
  - Medical Management
DISCLOSURES

- None!
GLAUCOMA NUTS AND BOLTS

- Etiology of Glaucoma
- Glaucoma Evaluation
- Following Glaucoma
  - Detecting glaucoma and progression
WHAT IS GLAUCOMA?

Glaucoma is an illness of the optic nerve, the nerve that carries the electrical signals for sight from the retina to the brain.
WHAT IS GLAUCOMA?

The exact cause is not known, but usually glaucoma is associated with a higher than average pressure (low teens-low 20s) inside the eye, which damages the retinal ganglion cell axons. This damage is seen at the "optic nerve head".
WHAT DAMAGES THE OPTIC NERVE IN GLAUCOMA?

- Pattern of Loss tells us the site of damage is at the optic nervehead

- Likely mechanism in high pressure glaucoma is a pressure-dependent blockade of axoplasmic transport

  Douglas Anderson 1974

  Blockade of material (white) at the optic nervehead in a monkey with high eye pressure
WHAT IS GLAUCOMA?

- There are many types of glaucoma, each named for special features.
  - POAG or Primary Open Angle Glaucoma is a diagnosis of exclusion (i.e. look for other causes before labeling it POAG).

- Most are chronic conditions which can be treated but not cured, by lowering the eye pressure to prevent additional loss of sight.
GLAUCOMA EVALUATION

- Current Medications
- Compliance
- Vision
- Pressure (Applanation is gold standard)
- Pachymetry
- Gonioscopy
- Anterior segment exam
- Optic Nerve cupping – structure
- Visual Field (with BCVA) - function
- Imaging (OCT or equivalent) - structure
GLAUCOMA EVALUATION

- **Gonio LOTS of normal angles**, not just narrow ones.
- You need to know what does NORMAL looks like
- Look for narrow angles.

“You can’t see what you’re not looking for”
ANGLE EVALUATION: SCREENING

- Van Herick Method
- Distance between anterior iris/posterior cornea.
- Light illuminating at 60 degrees

**TABLE 1. VAN HERICK SYSTEM**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>PAC Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Angle is wide open</td>
<td>PAC &gt; CT</td>
</tr>
<tr>
<td>3</td>
<td>Angle is narrow</td>
<td>PAC = 1/4 - 1/2 CT</td>
</tr>
<tr>
<td>2</td>
<td>Angle is dangerously narrow</td>
<td>PAC = 1/4 CT</td>
</tr>
<tr>
<td>1</td>
<td>Angle is dangerously narrow or closed</td>
<td>PAC &lt; CT</td>
</tr>
</tbody>
</table>

* Compares peripheral anterior chamber (PAC) depth to corneal thickness (CT).
ANGLE EVALUATION: GONIOSCOPY

- Gonioscopy
  - **Direct (surgical):** Koeppe, Barkan, Wurst, Richardson
  - **Indirect:** Zeiss, Goldman, Sussman

Total internal reflection — makes direct evaluation impossible
SURGICAL ANGLE EVALUATION

- Direct: Koeppe, Barkan, Wurst, Richardson, Franklin
ANGLE EVALUATION

- Indirect: Zeiss, Goldman, Sussman, Possner
  gonio prism
GOLDMANN THREE MIRROR LENS -
COUPLING AGENT REQUIRED BUT BEST VIEW OF ANGLE
ANGLE EVALUATION: SHAFFER SYSTEM

- Shaffer System
  - Angle between iris/TM
  - Visible angle structures
  - TM pigmentation
## Angle evaluation: Shaffer System

<table>
<thead>
<tr>
<th>Grade</th>
<th>Angle Range</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 4</td>
<td>45° to 35° angle</td>
<td>Wide open</td>
</tr>
<tr>
<td>Grade 3</td>
<td>35° to 20° angle</td>
<td>Wide open</td>
</tr>
<tr>
<td>Grade 2</td>
<td>20° angle</td>
<td>Narrow</td>
</tr>
<tr>
<td>Grade 1</td>
<td>≤ 10° angle</td>
<td>Extremely narrow</td>
</tr>
<tr>
<td>Slit</td>
<td>0° angle</td>
<td>Narrowed to slit</td>
</tr>
</tbody>
</table>

*Based on the angular width of the angle recess.*
ANGLE EVALUATION: SCHEIE SYSTEM 1957
## ANGLE EVALUATION: SCHEIE SYSTEM 1957

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wide open:</td>
<td>All structures visible</td>
</tr>
<tr>
<td>Grade I:</td>
<td>Iris root visible</td>
</tr>
<tr>
<td>Grade II:</td>
<td>Ciliary body obscured</td>
</tr>
<tr>
<td>Grade III:</td>
<td>Posterior trabeculum obscured</td>
</tr>
<tr>
<td>Grade IV:</td>
<td>Only Schwalbe’s line visible</td>
</tr>
</tbody>
</table>

*Angle depth system based on structures visualized.*
ANGLE EVALUATION: SPAETH SYSTEM

- Spaeth System
  - Iris/cornea angle
  - Configuration
  - Iris insertion
  - Pigmentation
# Angle Evaluation: Spaeth System

<table>
<thead>
<tr>
<th>Iris Insertion</th>
<th>Angular Approach</th>
<th>Peripheral Iris</th>
<th>Pigmentation of Trabecular Meshwork</th>
</tr>
</thead>
<tbody>
<tr>
<td>A Anterior to Schwalbe’s line</td>
<td>0° to 50°</td>
<td>r regular</td>
<td>f flat</td>
</tr>
<tr>
<td>B Between Schwalbe’s line and scleral spur</td>
<td>s steep</td>
<td>b bowed anteriorly</td>
<td>1+ minimal</td>
</tr>
<tr>
<td>C Scleral spur visible</td>
<td></td>
<td>p plateau iris</td>
<td>2+ mild</td>
</tr>
<tr>
<td>D Deep with ciliary body visible</td>
<td>q queer</td>
<td>c concave</td>
<td>3+ moderate</td>
</tr>
<tr>
<td>E Extremely deep with &gt;1 mm of ciliary body visible</td>
<td></td>
<td></td>
<td>4+ intense</td>
</tr>
</tbody>
</table>

*Evaluating iris insertion, angular approach, peripheral iris configuration, and degree of trabecular meshwork pigmentation.*
**ANGULAR APPROACH**
**(NOT IRIS RECESS)**

- Assess by means of tangential lines
- One line is tangential to the inner surface of the trabecular meshwork
- The other line is tangential to the middle third of the anterior iris surface.
- The angle formed by these two lines defines the angular approach 0-50 degrees
- This angle does not identify the angle of the iris recess itself, but rather the angular approach of the iris to this recess.
ANGLE EVALUATION: STATE WHAT YOU SEE

- SL
- ATM
- PTM
- SS
- CB
- Iris config
- Pigment
Examine the least narrow or the eye without pathology first.
  - Why? To know what normal is!

Start with the Inferior Angle (superior mirror)
  - The most open - best way to see all structures
  - Most pigmented TM - easily identified structures
GONIOSCOPY
PEARLS/PITFALLS

- Iris Contour
  - Steep insertion (aka narrow approach)
    - Have the patient look into the examining mirror or tip the lens towards the angle being examined to look over the hill and into the angle.
    - In general, if one can readily see over the hill and into the angle without indenting, the angle is not occludable. But if unsure, LPI or re-gonio in the future after discussing angle closure warnings. Which are…?
  - Regular
  - Concave – Queer formation – not politically correct.

- Corneal Wedge technique helps to identify Schwalbe’s line, in lightly pigmented eyes, young people
GLAUCOMA EVALUATION

- Structure – objective measurement
  - Optic nerve head (cupping, thinning, notching)
  - follow the vessel curve, NOT the color of the rim.
  - Nerve fiber layer

**Goal!**
Differentiate structural changes secondary to glaucoma FROM retinal and other optic nerve pathology
GLAUCOMA EVALUATION

- Function – subjective measurement
  - What does the patient see?

- Measuring function… is more difficult.

- **Goal** - Differentiate decreased visual function secondary to glaucoma from:
  - Retinal problems – RP, prior RD, ARMD, pattern dystrophy
  - Optic nerve – optic nerve tilt, optic pit, hypoplastic nerve, prior trauma, Graves Dz, pituitary tumor,
  - Optic tract, lateral geniculate body, optic radiation, visual cortex and cognitive pathology.
GLAUCOMA EVALUATION – FOLLOW UP

- Which to follow…Structure or Function?

or Both?
IN ESTABLISHED GLAUCOMA WHICH DEMONSTRATES PROGRESSION FIRST, LOSS OF STRUCTURE OR OF LOSS OF FUNCTION?

Why? Possibilities...

Structural changes 1st but no functional changes – perhaps in the nerve fiber layer ‘area of loss’ there are ‘redundant cells’ covering the same area allowing function in that area to continue.

Functional changes 1st but no structural changes – perhaps the nerves are ‘malfunctioning’ prior to actually dying and undergoing apoptosis. Maybe the variability in HVF is not only patient performance. Maybe it is the intermitent functioning of the cells.

Table 2: First detectable structural and functional change in the OHTS, EGPS and EMGT

<table>
<thead>
<tr>
<th></th>
<th>OHTS10</th>
<th>EGPS11</th>
<th>EMGT12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total number of endpoints</td>
<td>125</td>
<td>106</td>
<td>136</td>
</tr>
<tr>
<td>Structural change first</td>
<td>69 (55%)</td>
<td>42 (40%)</td>
<td>1 (1%)</td>
</tr>
<tr>
<td>Functional change first</td>
<td>44 (35%)</td>
<td>64 (60%)</td>
<td>117 (86%)</td>
</tr>
<tr>
<td>Structural and functional change at the same time</td>
<td>12 (10%)</td>
<td>0</td>
<td>18 (13%)</td>
</tr>
<tr>
<td>Length of follow-up</td>
<td>78 months (median)</td>
<td>60 months</td>
<td>72 months (median)</td>
</tr>
<tr>
<td>Cumulative probability of developing an endpoint (treated/observation)</td>
<td>4.4%/9.5%</td>
<td>13.4%/14.1%</td>
<td>45%/62%</td>
</tr>
</tbody>
</table>

EVALUATING STRUCTURE

Obsolete way:

Optic disc drawings—inaaccurate, not done 50% of time

Optic disc photography—very qualitative, only semi-quantitative

Optical Coherence Tomography (laser interferometer)

true structure - 5 micron resolution (Cirrus OCT)
(Technique offers the huge advantage that it can also be used to monitor macular disease)

GDx (Laser Polarimetry)

optical rotation, generally proportional to structure

Heidelberg Retinal Tomography (confocal reflectance from surface), measures shape rather than tissue
EVALUATING FUNCTION

Obsolete ways:
  Tangent screen
  Goldmann Kinetic Perimetry

Standard Automated Perimetry
  Humphery Visual Field

Less Common ways:
  Frequency Doubling Perimetry (FDP)
  Short-wavelength Perimetry (SWAP)
  Pattern Electro Retinogram (ERG)
  High Pass Resolution Perimetry (HRP)
FUNCTIONAL LOSS = PROGRESSION
FOLLOWING GLAUCOMA

Central questions:

- Is the patient compliant with current regimen?
- Is the glaucoma stable?
- Is more therapy needed?

Central Answers:

- Compliance can’t be measured but we know its BAD!!
- IOP increasing – make sure to use goldmann applanation
- VF worsening – repeat to verify loss versus bad test day.
- Optic nerve worsening
  - OCT – repeat because there is some inter-visit variability
  - Photos – OCT attempts to measure the ONH, cupping, etc. but it can interpret to data wrong, especially in abnormal nerves.
Secondary Glaucomas

- Blood induced glaucomas
- Pseudoexfoliation
- Pigmentary
- Phacolytic
- Lens Particle
- Intraocular Tumors
- Fuchs Heterochromic iridocyclitis
- Possner-Schlazmanns Syndrome
- Uvietic
- Elevated EVP
- Trauma
- Neovascular
- Corneal endothelial
- Angle closure glaucomas
Blood Induced Secondary Glaucoma
Blunt Trauma

- 70% less than 20 years old, Male 3:1
- Sports injuries up to 60%
- Increased IOP, secondary hemorrhages 4-35%
- Corneal blood staining 2%
- Poor visual acuity with rebleed and increased IOP
Blunt Trauma Associations

- Hyphema
- Iris sphincter tear
- Iridodialysis
- Cyclodialysis
- Trabecular Tear
- Inflammation
- Zonular rupture
- Ruptured Globe
Hyphema occurs from injury to vessels of peripheral iris or anterior ciliary body.
Hyphema Grading

- I - Layered blood less than 1/3 AC (50 to 60%)
- II - 1/3 to 1/2 AC (20 to 30%)
- III - 1/2 to less than total hyphema (15%)
- IV - Total or “Eight-ball” hyphema (5 to 10%)
- Microhyphemas - detectable by slit lamp
- Easier to just measure in ‘mm’
Complications- Secondary Glaucoma

- Leads to corneal blood staining
- Optic Atrophy
- Mean duration- 6 days
- Greater than 24 mmHg in 32% of patients
- Higher IOP and longer duration with total or near total hyphemas
Glaucoma - Mechanism

- Mechanical obstruction of TM with RBCs and fibrin
- +/- pupillary block from clot
- Long term- damage to TM/ Outflow system
Corneal Blood Staining

• Stromal infiltration with hemosiderin

• Tiny yellowish granules appear in posterior 1/3 of corneal stroma (Precedes gross staining by 24-36 hrs)

• Usually result of prolonged IOP elevation

• Clearing occurs peripherally- Amblyopia
Secondary Hemorrhage

- Clot lysis and retraction 2 to 5 days after injury
- Maximal rebleed in this period
- Highest incidence in grade III and IV, children less than age 6, blacks (24% vs 4% for whites)
- May be more severe than initial bleed
- Incidence of rebleed 4 to 35%
- Worse visual prognosis
Hyphema Complications

- Angle recession 30 to 85% - Gonioscopy
- 6-10% angle recession glaucoma
- Posterior synechiae
- Peripheral Anterior synechiae
- Optic atrophy
- Traumatic mydriasis
Hyphema Management

- Most can be managed as outpatients
- Consider admission in high risk cases or uncooperative patients
**Patient Orders**

- Quiet activities, may walk in hall
- Unilateral shield, no patch
- No reading, TV okay
- Elevate HOB 30 degrees
- Tylenol prn pain, no Aspirin
- Atropine 1% tid, +/- Pred Forte qid
- Sickle cell test if Black or Mediterranean
- If hospitalized consider Amicar – fibrinolysis inhibitor (very controversial)
Glaucoma Management

• Non sickle cell disease/trait:
  • Beta Blockers
  • Alpha agonists
  • Topical CAIs
  • Oral CAIs
  • IV Mannitol
  • Surgery

• Avoid miotics – increased inflammation
Glaucoma Management

- Sickle cell disease or trait:
  - Start with beta blockers
    - Use caution with all other agents
      - Topical and oral CAIs may reduce AC pH and induce sickling
      - Alpha agonists may affect (constrict) iris vasculature
      - Consider surgery earlier
After Discharge

- 3 to 4 weeks
  - Gonioscopy
  - Retinal exam
- Yearly follow up due to late glaucoma
- Glaucoma more frequent with > 270 degree angle recession and is uncommon with less than 180 degrees
Possible Surgical Criteria?
(controversial)

- IOP > 60mmHg x 48 hrs
- IOP > 50mmHg x 3 days
- IOP > 35mmHG x 7 days
- IOP > 24 x 6 days with >50% hyphema
- Total hyphema x 4 days
- Sickle trait or disease IOP >35 for >24 hours
Surgical Techniques

- Limbal paracentesis
- Limbal irrigation/aspiration
- Clot extraction
- Vitrector methods –
- Avoid instrumentation in AC if possible
Sickle Cell - Vicious Cycles

- Increased IOP --> decreased perfusion of nerve and retina --> sludging/sickling --> infarction of neuronal tissue
- Decreased perfusion of anterior segment --> hypoxia in AC --> further sickling --> TM obstruction --> increased IOP
- Hyperosmotics --> hemoconcentration --> more sludging/sickling
- Diamox --> decreased pH --> more sickling
Hyphema after surgery

- Early - Usually clears quickly
- Late - Look for vessels on inner aspect of incision
Ghost Cell
Glaucoma
Etiology

• Obstruction to aqueous outflow secondary to degenerated RBCs
• Occurs weeks to months after hemorrhage
Clinical

• Clearing hyphema, increasing IOP 40 to 70 mmHg
• Khaki colored cells in aqueous
• Pseudohypopyon, khaki colored TM
Histology

- Degenerating RBCs with Heinz bodies
- Aggregates of hemoglobin on vitreous strands
- Ghost cells are less pliable than fresh RBCs
Differential Diagnosis

- Uveitis
- Endophthalmitis
- No KPs seen with ghost cells
Management

- Medical
- AC washout - may need to be repeated
- Vitrectomy – these cells usually come from old vitreous hemorrhages
Hemolytic Glaucoma

- Clinical - Similar to Ghost cell but with reddish-brown appearance
- Mechanism - Free hemoglobin, RBCs and macrophages filled with debris that blocks the TM
Hemosiderotic Glaucoma

- Clinical- prolonged history of recurrent intraocular hemorrhage
- Slow onset- weeks to years later
- Associated with retinal degeneration, cataract, heterochromia, iron staining of cornea, ciliary body degeneration
Histology

- Breakdown of hemoglobin to globin, bilirubin and iron
- Iron causes degenerative changes in TM
Spontaneous Hemorrhage

• Intraocular tumors
  • JXG
  • Melanoma
  • Retinoblastoma
  • Other

• Neovascularization of the iris

• Blood dyscrasias
Secondary Open-Angle Glaucoma

- Pseudoexfoliation
- Pigment dispersion
- Lens induced
- Tumor related
- Uveitis
- Elevated episcleral venous pressure
Secondary OAG
(Pseudoexfoliation)

**Vignette:** 50 y/o white (Scandanavian) female referred optometry for asymmetric IOP or c/d  **OR** 50 y/o with dense 4+ cataract, small pupil, phacodonesis, and end-stage glaucomatous optic neuropathy referred by your previous resident colleague for cataract extraction
Pseudoexfoliation
(Ophthalmic Features)

- **EXAM:**
  - Angle: Heavily pigmented TM in variegated pattern, Sampaesi’s line
  - Shallow AC
  - Peri-pupillary TI defects with poorly dilating pupil
  - Bulls-eye pattern on anterior lens capsule
  - Phaco/Iridodonesis
Pseudoexfoliation (Ophthalmic Features)

**EXAM:**

- **Angle:** Heavily pigmented TM in variegated pattern, Sampoelesi’s line
- **Shallow AC**
- **Peri-pupillary TI defects with poorly dilating pupil**
- **Bulls-eye pattern on anterior lens capsule**
- **Phaco/Iridodonesis**
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  - Peri-pupillary TI defects with poorly dilating pupil
  - Bulls-eye pattern on anterior lens capsule
  - **Phaco/Iridodonesis**
Pseudoexfoliation
(Ophthalmic Features)

- **Cause:** Distinctive fibrillar material deposition through body (angle → TM damage/obstruction, endothelium, lens capsule, ZONULES)

- **Common in:** Strongly age related (>50 yrs), Scandanavian, unilateral or bilateral (asymmetric),

- **Treatment:**
  - Higher IOP than POAG, worse pronosis
  - ALT very effective, but short lived (PXE>PDS>POAG>NTG)
  - May need TAE (increased inflammation)
  - Higher rate of vitreous loss with cataract surgery due to zonular weakness
Laser Trabeculoplasty

Argon Laser Trabeculoplasty

- Placement of laser burns at junction of pigmented/non-pigmented TM
- Effective at reducing IOP in PXE, PDS, POAG, NTG
- Contraindicated in inflammatory glaucoma, angle recession, lack of effect in fellow eye
- GLT: As effective as Timolol at 2 years

Selective Laser Trabeculoplasty

- Non-destructive laser procedure
- Energy directed at pigmented cells of TM
- Less energy, broader field of treatment
Secondary OAG
(Pigment Dispersion Syndrome)

Vignette: 32 year-old ophthalmology residents presents c/o mild eye pain in OD during exercise.
Pigment Dispersion Syndrome
(Ophthalmic Features)

**EXAM:**

- Krukenberg spindles
- Heavily pigmented TM (Homogenous)
- Mid-peripheral iris TI defects
- Anterior/Posterior lens deposits
Pigment Dispersion Syndrome (Ophthalmic Features)

EXAM:

- Krukenberg spindles
- Heavily pigmented TM (Homogenous)
- Mid-peripheral iris TI defects
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(Ophthalmic Features)

- **EXAM:**
  - Krukenberg spindles
  - Heavily pigmented TM (Homogenous)
  - Mid-peripheral iris TI defects
  - Anterior/Posterior lens deposits
Pigment Dispersion Syndrome (Ophthalmic Features)

- **Cause:** Liberation of iris pigment due to a “reverse pupillary block”
- **Occurs in:** Young, myopic, type A males
  - IOP may increase with exercise
- **Treatment:**
  - LPI to eliminate iris/zonular touch
  - May need chronic IOP meds/TAE (20-50% progress to pigmentary glaucoma)
Pigment Dispersion Syndrome (Ophthalmic Features)

- Ultrasound biomicroscopy pre/post LPI
Secondary OAG (Lens Induced—Phacolytic)

- **Vignette:** 65 y/o with h/o poor Va OD x 5 years (painless, progressive) presents with acute onset of decreased vision, pain, photophobia.
Secondary OAG
(Lens Induced—Phacolytic)

- **EXAM:**
  - IOP normal to high
  - Microcystic edema without KP
  - AC cell with clumps, possible hypopyon
  - Mature cataract with wrinkled anterior capsule
Secondary OAG (Lens Induced—Phacolytic)

**EXAM:**

- IOP normal to high
- Microcystic edema without KP
- AC cell with clumps, possible hypopyon
- Mature cataract with wrinkled anterior capsule
Secondary OAG
(Lens Induced—Phacolytic)

- **Cause:**
  - Leakage of HMW lens protein through capsule of hypermature cataract
  - Engorged Mø engulfed w/ HMWP clog TM

- **Treatment:**
  - Treat with IOP agents, topical steroids
  - Definitive treatment cataract extraction
Secondary OAG
(Lens-Particle Glaucoma)

- **Vignette:** 45 y/o s/p phaco with IOL POW#1 with c/o pain, photophobia, headache, nausea, vomiting, and decreased vision. Cataract extraction was uneventful but a significant amount of sub-incisional cortex was left in capsular bag.
Lens Particle Glaucoma
(Ophthalmic Features)

- **EXAM**
- IOP 45
- Microcystic edema
- Moderate AC reaction
- Cortical material in AC
Lens Particle Glaucoma (Ophthalmic Features)

- **EXAM**
  - IOP 45
  - Microcystic edema
  - Moderate AC reaction
  - Cortical material in AC
Lens Particle Glaucoma (Ophthalmic Features)

- **Cause:**
  - Incomplete removal of lens material
  - Obstruction of TM by lens material (IOP rise dependent on TM function)

- **Occurs in:** Post cataract patients following CE, capsulotomy, trauma (weeks to years).

- **Treatment:**
  - IOP control (topical/systemic), topical steroids initially
  - AC washout (+/- tube shunt)
Lens Particle Glaucoma (Ophthalmic Features)

*Phacoanaphylaxis* rare variant of lens induced uveitis/glaucoma. Caused by sensitization of lens protein after surgery/trauma with secondary granulomatous uveitis (has KP)
Secondary OAC (Intraocular tumors)

- Causes of glaucoma:
  - Direct invasion of angle—most common
  - Deposition of tumor cells/debris/WBC on TM
  - Intraocular hemorrhage
  - Angle closure (CB rotation, Iris neovascularization)

- Adult tumors: Melanoma, metastatic, lymphoma, leukemia

- Childhood tumors: Rb, JXG, Dyktioma
Secondary OAG
(Uveitic Glaucoma--Fuchks)

- **Vignette:** 45 year-old male referred to you by optometry for evaluation of cataract OS. While taking a history the patient reports some doc treated him in the past with a milky colored drop off and on for a couple of years. He denies ever having significant pain, photophobia, but does report he now has difficult driving at night due to glare.
Fuch’s heterochromic iridocyclitis (Ophthalmic Features)

- **EXAM:**
  - Iris hypochromia
  - Fine Stellate KP with low grade AC reaction
  - Fragile vessels in angle—not associated with PAS
Fuch’s heterochromic iridocyclitis (Ophthalmic Features)

**EXAM:**

- Iris hypochromia
- Fine Stellate KP with low grade AC reaction
- Fragile vessels in angle—not associated with PAS
Fuch’s heterochromic iridocyclitis
(Ophthalmic Features)

- **Cause**: Idiopathic, unilateral, chronic iritis with low-grade AC reaction with small, stellate KP

- **Occurs in**: Middle aged men

- **Treatment**:
  - **Inflammation**: Treat cell not flare (less response to PF)
  - **IOP control**:
    - Possible avoidance of prostaglandin analogs due to association with uveitis. (Note: brimonidine tartate has been reported to induce granulomatous uveitis in the literature)
    - OAG occurs in ~15% of patients—IOP does not correspond to degree of cell.
  - **Cataract**: Eye needs to be quiet x 3 months for CE
Secondary OAG
(Uveitic Glaucoma—Glaucomaocyclitis crisis)

*Vignette*: 45 year-old man with h/o recurrent episodes of unilateral blurred vision with mild eye pain presents c/o similar symptoms. He has never sought treatment for previous episodes due to funding issues. Now insured, he seeks your medical attention immediately with this current episode.
Glaucomatocyclitic crisis (Ophthalmic Features)

**EXAM:**

- IOP: 50 OD, 14 OS
- Iritis with fine KP OD
- Open angle with KP on TM OD
- C/D 0.9 AND 0.25
Glaucotomocyclitic crisis
(Ophthalmic Features)

- **Cause:**
  - Idiopathic (possibly prostalandin-mediated).
  - Iritis generally resolved without treatment and IOP normalizes inbetweenabouts.
  - May develop OAG after increasing number of attacks

- **Common in:** Middle-aged

- **Treatment:** Treat active Dz. May need chronic IOP control
Uveitic Glaucoma
(Iridocyclitis)

- During acute iridocyclitis IOP may be low due to ciliary body shutdown.
- IOP may be high if trabecular function reduced (HZV associated with high IOP).
- Angle may become occluded by fibrin, debris, white cells.
- Angle closure possible from PAS or seclusio pupillae.
- Treat with topical steroids and aqueous suppressants (? Prostaglandins). If filtration required, tube shunt better option. PI for Seclusio pupillae.
Secondary OAG (Elevated EVP)

- **Vignette:** 54 year-old white male referred by family medicine for evaluation of chronic red eye. Patient had a h/o MVA 6 months ago and sustained head trauma. Patient has been treated unsuccessfully with topical anti-histamines, antibiotics, steroids, and vaso-constrictors.
Elevated Episcleral Venous Pressure (Carotid-Cavernous fistula)

**EXAM:**

- IOP 34 OD, 17 OS
- Mild proptosis
- Dilated episcleral veins
- Blood in Schlemm’s canal
- Engorged retinal veins with peripheral hemorrhages
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**Cause:**
- **AV Malformations:** CC fistula, orbital varix, Sturge-Weber Syndrome
- **Venous Obstruction:** Thyroid ophthalmopathy, retrobulbar tumor
- **Superior vena cava syndrome**

**Treatment:**
- Aqueous suppressants better than meds that increase TM outflow
- Filtering surgery (tube-shunt) may be complicated by uveal effusions or suprachoroidal hemorrhage—**sclerostomies**
Secondary Open-Angle Glaucoma (Trauma)

- Trauma can induce elevated IOP in many ways:
  - Hyphema
  - Angle recession
  - TM tears
  - Iritis
  - Ectopia lentis
  - Vitreous hemorrhage
  - Retinal detachment
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Ghost Cell Glaucoma
Hemolytic Glaucoma
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Thank you for your attention