Challenging Cases from Front to Back
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Disclosure
• Presenter is on speakers panel of Alcon, Allergan, AMO, Bausch + Lomb, Akorn, Tear Lab, Reichert, Lunovus
• President of MRB Eye Consultants
• Past-President of the Optometric Council on Refractive Technology (OCRT)
• OSSO Board Member
• Presenter has NO financial interest in any products mentioned except:

DISCLOSURES
• Ferrucci
  – Speakers bureau/advisory board for
    • Alcon
    • B&L
    • CenterVue
    • Genentech
    • Heidelberg
    • Macula Risk
    • Maculogix
    • Science Based Health

Patient FS
• 48 yo hispanic male
• Sent from primary care with sudden decreased vision right eye x 1 day
• Oc hx: unremarkable, last exam 3-4 years ago
• Med hx: HTN
• Meds: amlodipine

Exam
• VA 20/200 OD, 20/20 OS
• 1+ APD OD
• EOMs: No pain, no diplopia, no restriction
• FCF: full OU
• SLE: mild arcus OU

Patient FS
• Macula edema
• ONH edema temporarily with leakage on FA
• Focal choroiditis
Patient FS

- Tentative Diagnosis: Neuroretinitis OD
- Additional questions
  - Has flu-like symptoms x 1 mos, feeling better last few days
  - No camping
  - No cough
  - No travel out of country
  - 2 cats. Doesn’t recall being scratched

- Neuroretinitis
  - Cat scratch
  - Syphilis
  - TB
  - Lyme Disease
  - Toxoplasmosis

Lab Tests

- RPR/PPAT-TP: Syphilis
- Lyme Titer
- Quantiferon Gold: TB
- Bartonella Titters: Cat Scratch
  - Henselae
  - Quintana
- Retina Consult
- Infectious disease consult

Retina Consult

- Concur with findings
- Bartonella most likely (Cat Scratch)
  - Start Doxycycline 100 mg bid
- Refer to Infectious disease

Lab Tests

- Labs ordered
  - RPR/PPAT-TP: negative
  - Lyme Titer: negative
  - Quantiferon Gold: negative
  - Bartonella Titters
    - Henselae: 1:1024 (reference 1:64)
    - Quintana: negative
- Added by Infectious diseases
  - HIV testing: negative
  - ACE (Sarcoid): negative
  - HLA-B27 (Reiters): negative
  - Histoplasmosis Abs: negative
  - Toxoplasmosis Abs: negative

Neuroretinitis

- Inflammation of the optic nerve head and surrounding retina
- Not involved with Multiple Sclerosis
- Vitreous cells may be present
- RAPD is present
Neuroretinitis: Retinal Findings

- Serous Retinal Detachment extending to the fovea
- Macular star of hard exudates
- Optic nerve head swelling
- Multifocal retinitis

I felt like something was stabbing my eye

- “This happened another time and the doctor told me to use ointment at night”
- Type II Diabetes
- NKMA

“I REALLY HURTS”
Epithelial Basement Membrane Dystrophy: Map Dot Fingerprint

87% of all RCE occurs in what part of the cornea?

**Inferior Cornea**

46% of all patients in this study had EBMD
- The remainder had trauma induced causes
  - Fingernail
  - Paper cut, etc.
Diagnosis:

- **Recurrent Corneal Erosion**
- **EBMD**

Treatment:

- **What medications should be avoided?**
- **Bland Artificial Tear Ointments**


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**TREATMENT OF RCE**

- **DEBRIDE**
- **DOXYCYCLINE**
  - 50 mg qid
  - 100 mg bid

Long Term Treatment Regimen Recalcitrant RCE

- FreshKote TID x 2 months
- Lotemax QID x 2 weeks then BID x 6 weeks
- Doxy (20 or 50mg) BID x 2 months
TREATMENT

- PROKERA

3 MONTH FOLLOW-UP

- SLIT-LAMP: CLEAR CORNEA
- DX:

PCE (PREVENTED CORNEAL EROSION)
TX: CPM (RESTASIS)

Case SS

- 71 year old retired Military and secret service agent
- Hasn’t had eyes checked in a few years
- VA 20/20 OU with low hyperopic/astigmatic RX
- SLE: mild bleph, trace NSC
- Posterior pole:
Case SS

- A: HH plaque OS
- P: refer for carotid doppler
  - Labs
  - refer to PCP for management of other risk factors
  - Vascular clinic dependent on carotid study

Case SS: Labs

- Labs
  - BP: 134/88
  - Weight: 236
  - BMI: 38.2
  - A1c: 9.9 (H)
  - Triglycerides: 173 (H)
  - HDL: 31.2 (L)
- PCP: diet, education, start insulin

Case SS

- Carotid:
  - Right: non hemodynamically significant soft calcific plaque at left carotid bifurcation
  - Left: 50-69% ICA Stenosis
- Vascular clinic:
  - Monitor left carotid q 6 mos as no symptoms in last year
  - Start ASA therapy

Retinal Plaques

- Several different types of plaques can often be visualized in the retinal vasculature
- Pt is typically elderly, has HTN, CAD, hypercholesterolemia/hyperlipidemia, and/or atherosclerotic disease
- Often totally asymptomatic and found on routine exam

RISK FACTORS

- Age
- HTN
- Vascular disease
- Past vascular surgery
- SMOKING
- High TOTAL cholesterol
- Men> women

Prevalence

- Beaver Dam Eye Study: 1.3%
  - smoking, HTN and DM
  - 9x more likely after age 75 vs. 43-54
  - after 75, 3.1% prevalence
  - Equates to 1.2 million people with emboli 43-86
  - 450,000 are 75-86
  - Fatal stroke 3x as likely over 8 years in pts with emboli, adjusting for other factors
- OD>OS
- Bilateral very infrequently
Prevalence

- Blue Mountain Eye Study: 1.4%
  - HTN, smoking, Vascular disease
- LA Latino Eye Study: 0.4%
  - Smoking, CAD, h/o MI, HTN
- Singapore Eye Study: 0.6%
  - Smoking, high cholesterol, h/o angina

Retinal Plaques

- May present with amaurosis fugax, transient episodes of monocular blindness
- Rarely, may report transient ischemic attack (TIA), which is above with hemiparesis, paraesthesia or aphasia

Retinal plaques

- Three different types of plaques, but all share strong association to significant cardiovascular disease
  - HH 80% > fibrino-platelet 14% > calcific 6%

Retinal Plaques

- Cholesterol (Hollenhorst) plaque
  - Most common
  - Shiny yellow-orange in appearance
  - From plaque in the ipsilateral carotid artery
  - Rarely causes occlusion, unless multiple
  - Typically occurs at bifurcations
  - Mobile in nature

Retinal Plaques

- Fibrino-platelet
  - Appear as dull white to gray, long plugs
  - Typically within arterioles, not at bifurcations
  - May break-up and dissolve with time
  - May lead to BRAO or CRAO
  - Often associated with carotid disease or mitral valve insufficiency

Retinal Plaques

- Calcific
  - Appears more whitish than HH
  - Dull, non-reflective, white
  - Classically within arteriole, not at bifurcation
  - Typically immobile
  - Most dangerous, often cause BRAO
  - Often from cardiac atheromas of heart valves
### Retinal plaques

- **Talc retinopathy**
  - Represents an exogenous plaques as opposed to others
  - Appears typically as multiple shiny yellow plaques within capillaries in posterior pole
  - Typically smaller than other plaques
  - Typically seen in IV drug users
  - Rarely cause complications, but reported cases of associated NV and occlusions

### Retinal plaques

- No direct management of plaques is needed
- Management is aimed at discovering source of embolus to decrease risk of other emboli, occlusion, or stroke
- Pts need referral to internist for complete physical

### Retinal Plaques

- Assess risk factors with PCP
  - DN, HTN, lipid panels
- Carotid ultrasound
- MRA: non-invasive image with 2D/3D
- TEE: invasive, probe into esophagus to image heat valves
  - Helpful with calcific
- CTA: CT scan of arteries construct 3D images

### Carotid Ultrasound

- First line screening test
- ORDER WITHIN TWO WEEKS!!
- Identifies flow rate and % stenosis
- Common, internal, and external
- Only ≈20% of asymptomatic emboli will have significant carotid stenosis

### Retinal Plaques

- <50-60% occlusion
- **ORAL TREATMENT**
  - Anti-Platelet
    - ASA
  - Anti-coagulation
    - Comadin, platelet
  - Cholesterol meds
- **>70-99%**
  - **SURGICAL TREATMENT**
    - Carotid endarterectomy
    - Angioplasty
  - Reduces risk of future stroke!

### Retinal Plaques

- After ruling out underlying etiology, see patient regularly, q 6 -12 mos, to evaluate for additional plaques or other disease associated with vascular disease
  - BRVO/CRVO
  - BRAO/CRAO
  - NTG
Is it worth working up these patients?

- 18% of pts with retinal emboli had internal or common carotid stenosis >75% 
- Higher incidence of stroke
  - 8.5% with emboli vs 0.8% w/o per year
- Pts with cholesterol HH emboli have 15% mortality at 1 yr, 29% by year 3, and 54% by 7 years

Secondary Glaucomas

- Elevated IOP related to a specific cause
- Some types may be prevented or treated
- Unilateral
- Elevated IOP leads to typical glaucomatous changes
  - ONH changes and VF loss

Secondary Glaucomas

- Pigmentary
- Pseudoexfoliation
- Traumatic
- Neovascular
- Steroid
- Uvetic

PIGMENTARY GLAUCOMA

Pigment Dispersion (PDS) and Pigmentary Glaucoma

- Inherited
- Abnormal Irido zonular contact
  - Exaggerated by physiological pupillary movement
- Disruption of iris pigment
- Deposition of dispersed pigment in anterior segment
- Pigment deposited in angle
- Transient > Permanent IOP elevation
PDS and Pigmentary Glaucoma

- Diagnostic Triad
  - Corneal endothelial deposition (Krukenberg spindle)
  - Slit-like, radial, mid peripheral iris transillumination defects
  - Dense trabecular pigmentaion
- Iris insertion typically posterior
- Peripheral iris tends to be concave

PDS and Pigmentary Glaucoma Treatment

- Begin therapy early to prevent development of glaucoma
  - Prevent progression of disease
  - Difficult decision when to initiate therapy
    - IOP elevated at any time
    - Monitor transillumination defects
    - Do gonio
- Treatment
  - Miotics
  - Laser iridotomy
  - Argon/Selective Laser Trabeculoplasty

PXE

- Ocular and systemic condition
  - Unknown etiology
- Excessive presence of extracellular material
- Intraocular signs most obvious
- Involves all structures in anterior segment

PXE

- 15-40% probability of developing glaucoma
- 66% present unilaterally
- 17% odds of glaucoma in contralateral eye in 10 years
- Compares unfavorably to POAG
- Cataracts more likely, CE more complicated
Clinical Features:
- Lens: central deposition of white material, a clear zone, and a peripheral zone
- Iris: loss of ruff, sphincter transillumination
- Cornea: clumps of white material pigment
- Angle: splotchy pigmentation and Sampaolesi line
- Zonules: loose with white deposits

Case
- Chief Complaint
  - 68 y.o. Caucasian male
  - Complains of photophobia and blurred vision
  - As well as a headache over right eye for 2 days
- VA 20/80
- PH: 20/20-
- Pupils (- APD)
- EOM’S Normal
- IOP: 43 OD, 14 OS

Slit lamp exam:
- Grade 2- injection:
- Irregular SPK and staining
- AC: grade 3 cell & flare
SLE corneal findings:

Diagnosis??

- Herpes Zoster Ophthalmicus

Herpes Zoster

- Nearly 1 Million Americans develop herpes zoster each year
- Herpes Zoster Ophthalmicus (HZO) accounts for up to 25% of presenting cases
- Over 50% incur ocular damage

Hutchinson’s Sign:

- Lesion on the tip of the nose
- Naso-ciliary branch of ophthalmic division of trigeminal nerve (V)
- Nasal means possibly ciliary (ocular) involvement
Ocular findings:
- Conjunctivitis/Scleritis
- Pseudodendrites
- Neurotrophic keratitis
- Iritis
- Glaucoma
- ION, vein or artery occlusion
- Nerve Palsy

Herpes Zoster Ophthalmicus

Iridocyclitis and HZO
- Most common and most often overlooked ocular complication (43%)
- Highly elevated IOP
  - Study by Thean, Hall & Stawall - clinical Ophthalmology Dec 2001
  - 56% of patients developed glaucoma!!

Treatment: Iridocyclitis
- Pred Acetate 1% q1h or q2h
- Durezol (Difluprednate) 0.05% QID
- Lotemax Long term
- Cycloplegia
  - Homatropine 5% bid
  - Cyclopentolate 1% bid

Also added medication to lower the IOP
- Diamox 500 mg (non-sequels)
  - after asking about sulfa allergies and kidney problems
- Beta-blocker gtts
  - after asking about heart rate and breathing problems
- Iopidine/Alphagan

Treatment of HZO:
- Acyclovir 800 mg 5x/day
- Famvir 500 mg 3x/day or Valacyclovir 1000 mg 3x/day
- Advantages:
  - Easier to take 3x Vs. 5x
  - Decreased post-herpetic neuralgia, faster resolution of patient (Ormrod - Drugs June 2000)
Treatment:

- **When should you begin therapy?**
  - Prior to 72 hours proven for Acyclovir (HE Kaufman)
  - Not as critical for Valacyclovir or Famvir* (Ormrod)

Duration?

- 7 days for most patients although newer studies (Zaal - Am J or Ophthal. Jan 2001) suggest
  - 10 days for patients over age 66 due to shedding

New Vaccine: Zostavax

- Live attenuated zoster vaccine
- Indicated for patients above age 60 who had chicken box as a child but have not had shingles
- Doesn’t work in 100% of cases and decreased effect with age

New Vaccine: Zostavax

- In the Shingles Prevention Study 38,000 patients 60 and older were enrolled
  - 51.3% reduction of herpes zoster
  - 61.1% reduction in the severity of herpes zoster
  - 66.5% reduction in the incidence of post-herpetic neuralgia

Case LBV

- 38 y.o. African American Female
- Complaint of decreased vision for about 1 week
- Longstanding contact lens wearer
- Vision seems to be getting worse over last few days
- No significant pain
- No corneal staining
Herpes Simplex Keratitis is a Conundrum in of Itself

HSV Dendritic Keratitis vs. HZO Pseudodendrites

- Simplex
  - True ulceration (as opposed to elevation)
  - Ulceration picks up staining
  - More obvious terminal end bulbs
  - Younger patients
- Varicella Zoster
  - Elevated or superficial
  - Some portions of pseudo-dendrite may pick up dye but not all
Much more symptomatic
- DEEP vascularization and infiltration
- No clear zone near limbus
- Stain with lissamine - often see conjunctival dendrites
- Significant pain and inflammation

Immune Stromal Keratitis
- Stromal haze
- Neovascularization
- Immune ring
- Intact epithelium

Testing corneal sensitivity
- Cotton Wisp or Dental Floss

Treatment: Epithelial Involvement
- Topical Zirgan 5 x per day until dendrite resolves and then TID x 1 week
- PF artificial tears
- Oral antivirals (Valacyclovir 500mg TID)
- Follow-up next day, day 3-4, day 7-10
Treatment: Stromal & Endothelial-
No epithelial involvement
• Corticosteroids
• Prophylaxis with P.O. Valtex or Acyclovir (or topical Zirgan?)
• Cycloplege

When to use Oral Antivirals?
• Valtrex 500 mg TID or 1000mg qd
• Toxicity of Viroptic required lower dosing and introduction of oral antivirals
• Patients with Hx of stromal keratitis
  • Children
    – Primary HSV (acyclovir)
  • Surgical prophylaxis

Oral Antivirals
• Inhibit viral DNA polymerase without inhibiting normal cellular activity
• Works best if treatment initiated within 72 hours
• Pregnancy category B
• Caution in patients with renal disease

<table>
<thead>
<tr>
<th>Antiviral Drug</th>
<th>HSV</th>
<th>HZO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acyclovir</td>
<td>400 mg 5x/day for 1 week</td>
<td>800 mg 5x/day for 1 week</td>
</tr>
<tr>
<td>Valacyclovir</td>
<td>500 mg TID for 1 week</td>
<td>1000 mg TID for 1 week</td>
</tr>
<tr>
<td>Famciclovir</td>
<td>250 mg TID for 1 week</td>
<td>500 mg TID for 1 week</td>
</tr>
</tbody>
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Herpetic Eye Disease Study I
• Herpes Stromal Keratitis, Not on Steroid Trial
  – Pred Phosphate faster resolution and fewer treatment failures
  – Delaying treatment did not affect outcome
• Herpes Stromal Keratitis, on Steroid Treatment
  – No apparent benefit in the addition of oral acyclovir to the treatment of topical corticosteroid and topical antiviral
  • HSV Iridocyclitis, Receiving Topical Steroids
    – Trend in the results suggests benefit in adding oral acyclovir

Herpetic Eye Disease Study II
• HSV Epithelial Keratitis Trial
  – No benefit from oral ACV with topical trifluridine in preventing the development of stromal keratitis / iritis
• Acyclovir Prevention Trial
  – Reduced by 41% the probability of recurrence
  – 50% reduction in the rate of return of the more severe form
• Ocular HSV Recurrence Factor Study
  – No results available

Case Report
• 37 y.o. attorney presents with complaints of intermittent mild discomfort in the right eye
• “The eye seems to have blurred vision during attacks”
• “The pupil seems larger sometimes in the eye”
• Friends tell the patient he looks like he has been drinking
• Differential diagnosis please...
SF CASE

- 68 year old male
- Presents with c/o flashes floaters OD x 2 days
  - No pain
  - No change in acuity
- Med hx: Type 2 DM x 2 years, well controlled; HTN; ED
- Meds: Metformin, HCTZ, Lipitor, Viagra
- Oc Hx: Unremarkable

SF CASE

- Entering VA: 20/25 OU
- SLE: WNL
- IOP 14 mm OU
- DFE:

SF CASE

- Assessment:
  - Acute PVD OD

- Plan:
  - Pt education
    - Signs/symptoms of RD
  - RTC when?

The Vitreous Humor

- Floaters typically most common symptom
  - Cobwebs
  - Files
  - Hairs
- Flashes
  - Indicative of traction on retina, but not necessarily a tear or break

- Vitreous attached most firmly at
  - Macula
    - VMT
  - Vitreous base
  - Around optic nerve head
    - Weiss' Ring
  - Also, some traction on blood vessels
    - Vit heme

The vitreous is composed of water, hyaluronic acid, hyalocytes, and type II collagen.
Physiologic Changes

- With age, liquifaction due to reduction in hyaluronic acid causes loss of support.
- This process is referred to as synchesis.

Physiologic Changes

- Vitreous shrinkage, contraction and collapse can cause traction.
- This process is referred to as syneresis.

Incidence of PVD

<table>
<thead>
<tr>
<th>Age</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;30</td>
<td>Rare</td>
</tr>
<tr>
<td>30-59</td>
<td>10%</td>
</tr>
<tr>
<td>60-69</td>
<td>27%</td>
</tr>
<tr>
<td>&gt;70</td>
<td>63%</td>
</tr>
<tr>
<td>&gt;80</td>
<td>75%</td>
</tr>
</tbody>
</table>

- 65% >65 have a PVD

Incidence of PVD

- Incidence may be accelerated by
  - Myopia
  - Trauma
  - Prior vitreoretinal disease
  - Surgery
  - Inflammation
- Symmetrical 90% of the time
- Happens to second eye with 1-2 years

PVDs

- Good News:
  - Retinal Tears/Breaks Relatively uncommon
    - One study: only 7-15% of symptomatic PVDs have a retinal break
- Bad news:
  - 7-15% have a retinal break

Risk Factors

- Pigment
  - Schaeffer’s Sign
    - Indicates break is possible
- Hemorrhage
  - 90% have break
- Inflammatory cells
Case

- 67 yo white male
- +DM Type 2 x 5-6 years, HTN
- Here for diabetic check
- Reports good vision
- “other health issues not related to eyes”
- 20/20 OU

Roth Spot

- White centered hemorrhage
- Hemorrhagic CWS
- First described by Moritz Roth, MD in 1872
- Seen in patients with bacteremia

Roth Spot

- CLASSICALLY associated with:
  - Bacterial Endocarditis
  - Leukemia
  - Pernicious Anemia

Roth Spot: Associations

<table>
<thead>
<tr>
<th>COMMON</th>
<th>LESS COMMON</th>
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<tbody>
<tr>
<td>DIABETIC RETINOPATHY</td>
<td>TRAUMA</td>
</tr>
<tr>
<td>HYPERTENSIVE RETINOPATHY</td>
<td>SHAKEN BABY SYNDROME</td>
</tr>
<tr>
<td>HIV</td>
<td>MULTIPLE MYELOMA</td>
</tr>
<tr>
<td>ISCHEMIA</td>
<td>ARETROVENOUS MALFORMATION</td>
</tr>
<tr>
<td>COLLAGEN VASCULAR DISEASE</td>
<td>CARBON MONOXIDE POISONING</td>
</tr>
</tbody>
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Roth Spots: Work Up

- Most common
  - HTN: Check BP (<120/80)
  - DM: A1c (<6.5) or FBS (<126)
  - CBC with white cell differential
    - Anemias, polycythemia, leukemias
  - Less common
    - PT/INR: for clotting disorders
    - Older pts: ESR/CRP to r/o GCA
    - Younger pts (<40): lipids, antiphospholipids, ANA (lupus)

Our patient: Acute myeloid leukemia
- Anemia
- Infection
- Hyperlipidemia
- Prostate cancer
- CAD
- Hypercholesteremia

Cotton Wool spots

- Fluffy, cloud like, whitish deposits in retina
- Typically associated with ischemia
- Differential diagnosis
  - Exudates
  - Drusen
  - Myelinated nerve fiber layer
Cotton wool spots

- DM and HTN most likely cause
- Typically, associated with systemic disease
- One study, pt with CWS and no known medical history
  - Elevated BS (DM) in 20%
  - Elevated BP (HTN) in 50% of pts

Cotton wool spots

- Neoplastic
  - Lymphoma, leukemia, metastatic carcinoma
- Medication induced
  - Interferon, chemotherapeutic agents
- Miscellaneous
  - Trauma, high-altitude retinopathy, Purtscher Retinopathy
- Idiopathic

Cotton wool spots workup

- Most common
  - HTN: Check BP (<120/80)
  - DM: A1c (<6.5) or FBS (<126)
  - CBC with white cell differential
    - Anemias, polycythemia, leukemias, bleeding disorders
- Less common
  - PT/INR: for clotting disorders
  - Older pts: ESR/CRP to r/o GCA
  - RF
  - Cardiac Risk factors

Case Report

- Slit Lamp findings of R.E.
  - Normal lids and lashes
  - 1+ Conjunctival injection
  - 1+ cells/minimal flare
  - Round and responsive iris
  - Clear lens
  - IOP with Tonopen-24 mm Hg

- Slit Lamp findings of L.E.
  - Normal lids and lashes
  - Trace conjunctival injection
  - No cells/flare
  - Round and responsive iris
  - Clear lens
  - IOP with Tonopen-14 mm Hg
Anterior Uveitis

- Described as an inflammation of the iris
  - Iritis
- Anterior uveitis is 4x’s more common than posterior
- Peak prevalence is 20-50 y.o.
- Unilateral
- Bilateral may be indicative of more serious etiology
- Witnessed in patients with systemic conditions

Signs and Symptoms

- Painful
- Photophobic
- Ciliary flush-circumlimbal
- Miotic pupil
- Reduced IOP (often seen)
- Posterior synechiae
- Presence of keratic precipitates on the endothelium

Signs and Symptoms of Ant. Uveitis

- Pathognomonic Sign-Cells and Flare
  - Cells and flare are best detected in dark with a slit beam (not conical)
  - Grading Scale
    - Trace: 1-3 cells noted No Flare
    - 1+: 4-8 cells noted Faint haze just detectable
    - 2+: 9-15 cells noted Moderate haze
    - 3+: Too many to count Marked haze iris detail fuzzy
    - 4+: Appears like a snow Plastic iris, fibrin clot storm

Anterior Uveitis Treatment

- Objectives:
  - Reduce severity
  - Prevent posterior synechiae
  - Prevent damage to iris blood vessels
  - Reduce frequency of attacks

Anterior Uveitis Treatment

- Start with cycloplegia
  - Places ciliary body in rest-closes blood vessels down
  - Cyclopentolate 1% (BID)-good for mild reaction
  - Homatropine 5% (QID)-intermediate reactions
  - Atropine 1% (BID)- severe reactions
Anterior Uveitis Treatment

- **Topical Steroid**
  - Prednisolone Acetate 1% (Pred Forte, Allergan)
  - Acetate penetrates the cornea and the anterior chamber
  - Do not substitute
  - *Shake rattle and roll*
  - Dose Q1H for severe inflammations or Q2h for moderate inflammations
  - QID most often prescribed—not the best though
  - Patients should be monitored every 1-2 weeks

Anterior Uveitis Treatment

- **Econopred Plus 1% (Alcon)**
  - Prednisolone acetate suspension
  - Similar production to Pred Forte
  - Needs to be shaken
  - Penetrates into the cornea and anterior chamber

Durezol

- Difluprednate 0.05% ophthalmic emulsion
  - 5 ml bottle
  - Developed as an emulsion
  - No shaking required
  - BAK-free
  - Uses sorbic acid as a preservative

Difluprednate Molecule

Prednisolon e molecule modified to increase:
- Potency
- Penetration
- Power

Anterior Uveitis

- **Chronic cases**
  - Require low dosage of steroid-qd
  - Rebound iritis from cataract surgery
  - RBO discussed with patient
- **Chlorambucil**
  - Immunosuppressive drug
  - TOO many side-effects
- **Cyclosporine**
  - Immunosuppressive drug
  - Restasis (cyclosporine 0.05%, Allergan)

What about my patient the lawyer?

Do you know what contraception is for an attorney?
Case Report-Uveitis “With a Twist”

- Posner-Schlossman Syndrome (glaucomatocyclitic crisis)
  - Unilateral
  - Occurs in patients 20-50 y.o.
  - Blurred vision
  - Pain
  - Mild injection of cornea
  - Increased IOP
  - Anterior uveitis

Posner-Schlossman’s

- Anterior chamber reactions could include KP’s
- Normal ONH
- No visual field loss

- Treat the iritis and the IOP
  - Pred 1% qid or Durezol
  - Alphagan P bid or Betimol bid
  - Avoid prostaglandins and miotics

Posner-Schlossman’s

- Prolonged antiglaucoma agent and steroids should be avoided
- Surgery is generally ineffective
- Self-limiting condition no long term treatment is indicated
- May experience multiple attacks