1 **Challenging Cases from Front to Back**

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2 **Disclosure**

- Dr Bloomenstein is on speakers panel of Alcon, Allergan, AMO, Bausch + Lomb, Akorn, Tear Lab, OcuSoft, 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:

3 **Disclosures-Dr. Fingeret**

- Aerie
- Alcon
- Allergan
- Bausch + Lomb
- Carl Zeiss Meditec
- Glaukos
- Heidelberg
- Sensimed, inc.
- Topcon

4 **Me Love STARBUCKS!**

5 **Case**

"What is that bump?"

6 **"That is Gross"**

- 53 y.o. Male
- No visual complaints
- "My eye had a small spot on the lid that is larger

- "People think there is something wrong with me?"
- NKMA
- NKMD

7. **"I vomited in my mouth"**
8. **What could this be?**
   - Hordeolum
   - Basal Cell Carcinoma
   - Sebaceous Cell Carcinoma
   - Squamos Cell Carcinoma
9. **Clinical Signs**
   - Painless growth
   - Bleeding, crusting, ulceration
   - Pigmentary changes
   - Destruction of normal architecture
   - Central ulceration
   - Telangectasia
   - Loss of cutaneous wrinkles
10. **Horedeolum**
    - Acute inflammation
      - Meibomian gland
        - internal
      - Sebaceous gland
        - external
    - Dense inflammation
    - Painful
    - Non-involuting
11. **Basal Cell Carcinoma**
    - 90-95% of eyelid malignancies
    - Most common malignant tumor of the eyes
    - Arise from hair-bearing skin
    - Cystic type resemble a benign inclusion cyst

  - –Fibrosing difficult to Dx
    - •Lie beneath and lose lashes
    - •Entropian/ectropian
    - •Lid notch/retraction/chalazia
    - •Chronic blepharitis

12 **Basal Cell Carcinoma**

- Location
  - –LL: 50-60%
  - –MC: 25-30%
  - –UL: 15%
  - –LC: 5%
- Hx: fair skin, sun exposure, smoking, prior BCC
- Forms: nodular, morpheaform
- Rarely metastasize
  - –Recurrent or neglected may invade orbit and need exenteration

13 **Basal Cell Carcinoma**

14 **Basal Cell Carcinoma**

15 **Basal Cell Carcinoma**

16 **Basal Cell Carcinoma**

17 **Slit Lamp Exam**

18

19 **Treatment of BCC**

- Biopsy
- Surgical excision, histological control of margins
- MC BCCs more likely infiltrative
- Cryotherapy not recommended
- Radiation therapy (XRT) palliative
  - –Use of ionizing radiation to damage cells DNA
    - •Malignant cells are less likely to repair DNA damage

- Extensive cicatrix

20 **Squamous Cell Carcinoma**

- 40x less common than BCC for the lid
- More common epithelial malignancy of the conjunctiva
- More aggressive
- Solar injury
- In areas of actinic keratosis,

21 **Squamous Cell Carcinoma**

- Potentiated by immunodeficiency
- May resemble various benign inflammatory lesions
  - –Pseudoepitheliomatous hyperplasia
  - –Inverted follicular keratosis
  - –Keratocanthoma

- Metastasis by lymphatic, blood, direct extension (along nerves)

22 **Squamos Cell Carcinoma**

23 **Squamos Cell Carcinoma**

24 **Squamos Cell Carcinoma**

25 **Squamos Cell Carcinoma**

26 **Treatment of SCC**

- Aggressive surgical excision
- Recurrences may require orbital exenteration

27 **Sebaceous Adenocarcinoma**

- Rare malignancy
  - –1-5.5% of eyelid malignancies in U.S.
  - –33% of eyelid malignancies in China
- Highly malignant, lethal

- Sebaceous gland origin: meibomian, Zeis, caruncle, eyebrow or facial skin

28 **Sebaceous Adenocarcinoma**

- F>M
- UL>LL
- Multicentric origin common

29 **Sebaceous Adenocarcinoma**

30 **Sebaceous Adenocarcinoma**

31 **Presentation**

- Patients commonly > 50 y/o
- Yellowish coloration
- Often masquerades as chalazion, chronic blepharitis, SLK, pannus associated w/ adult inclusion conjunctivitis
  - –Misdiagnosis is delayed by average of 3 yrs.

32 **Key Point**

- Beware of the “chalazion” that later causes loss of eyelashes and destruction of meibomian gland orifices

33 **Basal Cell**

- 90-95% of malignancies
- Hair bearing skin
- M=F
- LL>MC>UL>LC
- Rarely metastasizes
- Recurrence is common

34 **Squamos Cell**

- Common Conjunctiva
  - Rarely involves eyelid
- Aggressive
- Solar injury
  - In areas of actinic keratosis
- 40 X less common than BCC

35

36 **Sebaceous Adenocarcinoma Cell**

- Commonly > 50 y.o.
- Yellowish
- Masquerades
  - Bleph
  - SLK
  - Chalazion
    - Delays DX by 3 yrs.

37

38 **Keratocanthoma**

- Pseudo carcinoma
- Relatively common
- Skin colored or slightly red
  - Rapid enlargement
    - Weeks-months
    - Involutes spontaneously

  - Sun Damage?
  - M>F
  - > 45 y.o.
  - Starts as small boil

39

40 **Keratocanthoma (KA)**

- Relatively common
- Considered low grade malignancy
- Originates in pilosebaceous gland
- Some argue it is variant of invasive SCC
  - SCC, KA-type

41 **Treatment of KA**

- Excision
- Freezing
- Curettage
- Cautery
- Radiotherapy

42 **Diagnosing the Malignancy**

- Most eyelid lesions epidermal origin
- Early detection of eyelid malignancy important
- Clinical examination important, but not 100% accurate
- Biopsy if not sure

43 **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:

44 **Case**

45 **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

46 **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

47 **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

48 **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

49 **RISK FACTORS**

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

50 **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

51 **Prevalence**
- Blue Mountain Eye Study1.4%
  - 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

52 **Retinal Plaques**
- May present with amarosis fugax, transient episodes of monocular blindness
- Rarely, may report transient ischemic attack (TIA) , which is above with hemiparesis, parasthesia or aphasia

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

54 **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

55 **Cholesterol Plaques**

56

57

58 **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

59 **Fibrino-platelet Plaques**

60 **Retinal Plaques**

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

61 **Calcific Plaques**

62 **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

63 **Talc Retinopathy**

64 **Others**

- Tamoxifen Maculopathy (Nolvadex)

65 **Canthanxine Maculopathy**

66 **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

67 **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

68 **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

69 **Retinal Plaques**

1 <50-60% occlusion

2 • ORAL TREATMENT
  –Anti-Platelet
    •ASA
  –Anti-coagulation
    •Comadin, platelet
  –Cholesterol meds

3 >70-99%

4 • SURGICAL TREATMNET
  –Carotid edarterectomy
  –Angioplasty

  –Reduces risk of future stroke!

70 **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
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71 **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

72 **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

73 **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

74 **THE "PREVENTED" CORNEAL EROSION**

PCE

75 **"IT REALLY HURTS"**

1 43 yo female

2 –"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

76 **THE RECURRENT EROSION**

VA: 20/100 PH 20/30

SLIT-LAMP:
SUPERFICIAL SUPERIOR ABRASION
+ STAINING

DX: CORNEAL EROSION

77

78 **What are some leading causes of RCE?**

79 **EBMD**

80

81

82

83

84 **87% of all RCE occurs in what part of the cornea?**

Inferior Cornea

Reidy JJ, Pauli MP et al. *Cornea* 2000 Nov.

85 **46% of all patients in this study had EBMD**

- James Reidy et al. Recurrent erosions of the cornea: epidemiology and treatment. *Cornea* 2000 Nov; 19(6): 767-71
- The remainder had trauma induced causes
  - *–Fingernail*
  - *–Paper cut, etc.*

86 **Non-Treatment:**

- What medications should be avoided?

- Bland Artificial Tear Ointments

Eke T, et al. Recurrent symptoms following traumatic corneal abrasion. *Eye* 1999 June.

87 **Passive Treatment Of RCE**

- DEBRIDE
  - –Weck-Cel Spear
  - –Alger brush
- Bandage Lens
- Broad-spectrum AB

88

89 **NON-HEALING ABRASION**

Active Treatment….Dry or Wet??

90 **TREATMENT**

STANDARD PROTOCOL:

- BCL
- ANTI-BIOTIC
- ANTI-INFLAMMATORY
- RTC
- NEW REGIMENT
- AMNIOTIC MEMBRANE
- LATERAL TAPE TARSORAPHY
- PATIENT EDUCATION
- RTC 5 DAYS

91 **Amniotic Membrane**

- Amniotic membrane is the inner most lining of the placenta (amnion) and shares the same cell origin as the fetus
- Contains cytokines and growth factors
  - Anti-Inflammatory (protease inhibitors)
  - Anti-Angiogenic

- Aids in rapid wound healing and re-epithelialization
- Anti-Scarring

92

Cryopreserved amniotic membrane is a biologic therapy that can:

- Promote regenerative healing
- Reduce inflammation
- Minimize scar formation
- Inhibit angiogenesis
- Minimize pain

93

94

95

96 **Long Term TX Regimen Recalcitrant RCE**

- FreshKote TID x 2 months
- Lotemax Gel QID x 2 weeks then BID x 6 weeks
- Doxy (20 or 50mg) BID x 2 months
- Restasis Bid!

97 **5 DAY FOLLOW-UP**

"THE PAIN IS GONE"

- REMOVEDPROKERA
- VASC: 20/15

SLIT-LAMP:
CORNEAL CLEAR

TX: RESTASIS BID

RTC 4-6 WEEKS

98 **3 MONTH FOLLOW-UP**

- "MY EYE FEELS GREAT"
- VASC: 20/15
- SLIT-LAMP: CLEAR CORNEA
- DX:

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

99

100 **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

101 **SF CASE**

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

102 **SF CASE**

103 **SF CASE**

- Assessment:
  - –Acute PVD OD
- Plan:
  - –Pt education
    - Signs/symptoms of RD
  - –RTC when?

104 **SF CASE**

- Really no consensus
- Symptomatic PVD without retinal break
  - –AOA:1-2 weeks
  - – AAO: depending on symptoms, risk factors and clinical finings:
    - 1-6 weeks
    - Then 6 mos to 1 year
  - –Cleveland Clinic: 4-6 Weeks
  - –Others: if no heme or other issues, very low risk so no need to see to back

105 **PVD**

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

106 **Hello….Mr. Wiess**

107 **The Vitreous Humor**

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

108 **Physiologic Changes**

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

109 **Physiologic Changes**

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

110 **Incidence of PVD**

- 65%>65 HAVE A PVD

111 **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

112 **PVDs**

1 • Good News:

–Retinal Tears/Breaks *Relatively* uncommon
- One study: only 7-15% of symptomatic PVDs have a retinal break

–8-26% acute PVDs have an associated RB/RD at the time they present (Ophthalmology AAO 2014)

2 • Bad news:
–7-15% have a retinal break
–The chances of RB there after is <2-5%

113 **Risk Factors**
- Pigment
  –Schaeffer's Sign
    - Indicates break is possible
- Hemorhage
  –90% have break
- Inflammatory cells

114 **My recommendations**
- DFE WITH SCLERAL DEPRESSION
- DISCUSS SIGNS/SYMPTOMS OF RD
- RTC 6 WEEKS
- SEE UNTIL FLASHES SUBSIDE
- IF RISK FACTORS, CONSIDER REFERALL TO RETINA
  –Vitreous heme
  –Pt is Lawyer/father-in-law, etc
  –Just doesn't feel right

115 **Move….**

116 **Case**

117 **48 YO HF**

- Diagnosed with POAG 1995
- Diagnosis made by ophthalmologist in Minnesota
- Relocated to Phoenix, I assume care
- Untreated peak IOP
  - –OD=27mm Hg
  - –OS=29mm Hg

118 **48 YO HF**

Treatment History

- Initial therapy Timolol 0.5%
  - –Discontinued after 2 months
  - –Side effects of bradycardia & fatigue

- Current Medical Regimen:
  - –Xalatan ou qPm x 2 yrs

119 **48 YO HF**

- Since starting Xalatan IOP readings:
  - –OD=17-19mm Hg
  - –OS=18-20mm Hg

- Previous doctor felt that patient was being “safely” treated at this IOP level.

120 ***48 YO HF***

VF 2 yrs ago

2

121 **48 YO HF**

Initial Exam in Phoenix

- BCVA 20/20 OU
- - RAPD
- IOP OD=17mm Hg OS=18mm Hg

- Subjectively, the patient

–Reports excellent compliance
–Denies any side-effects

122 **48 YO HF**
Left Eye: Phoenix ancillary tests

123

Visual Field Progression
in upper teens

124 ***Disc hemorrhage suggests***
Upper teens are NOT low enough

125 **WHAT WOULD YOU DO NEXT?**

126 **MORE TESTING IS NEEDED**
ORA Reichert

127

128

129

130 **Bioengineering of the Eye**

- Viscoelastic tissue with complex, interconnected microstructure[1]

- Geometrical attributes are not a surrogate for biomechanical properties[1]
  –eg: CCT does not describe viscoelasticity

- The eye appears to be a mechanical structural continuum[2]

– Tissue properties may provide additional diagnostic information[3]

131 **Hysteresis: Not a New Concept**

- A measurement that characterizes response to application and removal of force (load/unload)[1]
  – Found in materials or systems that do not instantly follow forces applied to them but react slowly, *or dissipate a portion of the applied energy* [1]

- More than 7500 papers published on hysteresis in a variety of medical fields[2]
  – Various tissues and structures (tendon, lung, arteries, etc)
  – The importance of Corneal visco-elasticity had been discussed and explored (*EX-VIVO)* prior to the ORA[3]

132 **Basic Parameters**

133

134 **Corneal Hysteresis: A New Ocular Parameter**

135

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141

142 **The Cornea, IOP, and Glaucoma**

143

144

145

146 **The Cornea and Glaucoma**

147

148 **Define & Describe IOPcc**
Corneal-Compensated Intraocular Pressure

149 **Background**

150

151 **IOPCC vs CCT 184 Normals**

152 **28 eyes Pre/Post LASIK IOPcc**

153 **IOPcc in Normal Tension Glaucoma**

154

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156

157

158

159

160 **48 YO HF**

- CH OD: 7.3 OS: 6.9

  - –IOP g OD: 19 mm Hg IOPcc OD: 25 mm Hg
  - –IOP g OS: 17 mm Hg IOPcc OS: 2
- Patient switched from Xal to Lumigan
- IOP at 2 wks after switch
  - –IOPcc OD=12mm Hg
  - –IOPcc OS=13mm Hg
- IOP 3 months after switch
  - –IOPcc OD=09mm Hg
  - –IOPcc OS=11mm Hg

161 **48 YO HF**

Summary

- AGIS 7 asserts that IOP reduction correlates with visual field preservation.
- Low teens preserve visual field better than upper teens
- Fewer medications improve compliance

162 **NEXT …**

163 **Case Report -Slit Lamp View**

164 **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…

165 **Case Report**

1 • 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

2 • 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

166 **Anterior Uveitis**

1 • 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

167 **Anterior Uveitis**

• Reiter's syndrome-HLA-B27
• Ankylosing spondylitis-HLA-B27
• Juvenile rheumatoid-HLA- B27
• Behcet's- HLA-B5
• Tuberculosis
• Syphilis

168 **Signs and Symptoms**

• Painful
• Photophobic

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

169 **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*

170 **Anterior Uveitis Treatment**

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

171 **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

172 **Anterior Uveitis Treatment**

- Topical Steroid-Suspension

–*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

173 **Durezol**

Difluprednate 0.05% ophthalmic emulsion
- 5 ml bottle

Developed as an emulsion
- No shaking required
- BAK-freee
  - Uses sorbic acid as a preservative

174 **Difluprednate Molecule**

Prednisilone molecule modified to increase:Potency PenetrationPower

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

176 **What about my patient the lawyer?**

Do you know what contraception is for an attorney?

177 **Case Report-Uveitis "With a Twist"**
- Posner-Schlossman Syndrome (glaucomatocyclitis crisis)

–Unilateral
–Occurs in patients 20-50 y.o.
–Blurred vision
–Pain
–Mild injection of cornea
–Increased IOP
–Anterior uveitis

178 **Posner-Schlossman's**

1 • Anterior chamber reactions could include KP's
• Normal ONH
• No visual field loss

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

179 **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

180 **Secondary Glaucomas**

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

181 **Secondary Glaucomas**

• Pigmentary
• Pseudoexfoliation

- Traumatic
- Neovascular
- Steroid
- Uvetic

182 **Gonioscopy…**

You have to evaluate the angle!

183 **Four Mirror**

- Advantages
  - –Couping material NOT required
  - –Easy
  - –All 4 quadrants visible at same time
  - –Indentation gonioscopy can be performed
  - –Fundus visible
- Disadvantages
  - –No globe stabilization
  - –May artificially open the angle

184 **THE ANGLE**

185 **Views from the Lens**

186 **PIGMENTARY GLAUCOMA**

187 **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

188 **PDS and Pigmentary Glaucoma**

- Diagnostic Triad
  - –Corneal endothelial deposition (Krukenberg spindle)

  - –Slit-like, radial, mid peripheral iris transillumination defects
  - –Dense trabecular pigmentation
- Iris insertion typically posterior
- Peripheral iris tends to be concave

189

190 **PDS and Pigmentary Glaucoma Treatment**

- Begin therapy early to prevent development of glaucoma
  - –Prevent progression of disease
    - Therapy is not just to lower IOP
  - –Difficult decision when to initiate therapy
    - IOP elevated at any time
    - Monitor transillumination defects
    - Do gonio
  - –Treatment
    - Miotics
    - Laser iridotomy
    - Argon/Selective Laser Trabeculplasy

191 **PSEUDOEXFOLIATION SYNDROME**

PXE

192 **PXE**

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

193 **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

194

- 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*

195

196

197 **Xen (Allergan, Ca)**

Not FDA Approved

198 **TIME FOR 1 MORE…**

199 **Case Study**

- 67 year old WM
- "My vision is not good…I have blurred vision. My eyes cry a lot too. They cry all the time."
- +NIDDM (diet controlled 15 years)
- NKMA
- History of skin lesion removed from cheek

200 **Case Study**

- VACC
  - –20/30 (PH-20/20) OD
  - –20/100 (PH-20/70) OS
- SLEX
  - –2+ guttata-OU
  - –Mild pigment on endo-OU
  - –1/2+ NSC/Tr PSC-OD

–2+NSC/2+ PSC-OS

201

202 **Cataract Surgery OS**

- VASC
  - –20/100
  - –2-3+ Striae
  - –3+ POME
  - –1+ cells
  - –Lens centered
  - –IOP
    - 14 mmHg (ORA)

203 **Fuch's Dystrophy**

- Autosomal dominant inheritance
- Bilateral / Asymetry
- Late onset > 50 y.o.
- Females affected 3 times more than males
  - –5.7 % develop edema
- Characterized
  - –Corneal guttata
    - Excessive accumulation of abnormal endothelial secretions
    - Appears in 30-40th year of life

204 **Fuch's Dystrophy**

- Characterized
  - –Corneal Guttata
    - Small refractile "drops" on corneal endothelium
    - Affects the "pump" action of the endothelium
  - –Edema
    - Greater in the AM

- Desiccates as day goes on
- Long standing edema may lead to corneal scarring
- RCE's common

205 **Fuch's Dystrophy**

- Symptoms vary with degree of guttata and compromise of the endothelial tissue
- Moderate guttata
  - –May affect visual function
  - –May induce mild-moderate edema
    - Halos around lights
    - Hazy vision > a.m.
- Severe guttata
  - –Vision decreases
  - –Possible bullous develops

206 **FUCH'S DYSTROPHY**

207 **Fuch's Dystrophy**

- Treatment
  - –Early stages of disease
    - Increase artificial tears
    - Hyperosmotics qhs
    - BCL used if Bullous is present
    - EDUCATION!
  - –Visual function is significantly compromised
    - Penetrating keratoplasty
    - Deep Lamellar endothelial keratoplasty (DLEK)
    - Descemet stripping automated endothelial keratoplasty (DSAEK)

208 **Fuch's Dystrophy**

209 **Fuch's Dystrophy**

- DLEK
  - –Recipient cornea is stripped of Descemet's membrane and endothelium
  - –Transplantation of donor cornea through small incision
  - –Results in
    - Improves endothelial function, corneal clarity and restores vision
  - –Minimally affects refraction
  - –Can provide rapid visual recovery
  - –Maintains structural integrity of the cornea

210 **Case Study**

- 6 months PO
  - –VACC
    - 20/30-OS
    - Slex
      - –Well centered lens
      - –3+ guttata
      - –Mild pigment endo
    - IOP
      - –15 mmHg (ORA)

211 **Case Study**

- 13 months later
  - –Patient calls….
  - –"Sorry to bother you on a Sunday, but my eye is blurry today and it hurts alittle."
  - –"I have seen the corneal surgeon recently and he said my cornea looked good."
  - –"I was 20/30. Today it is not so great.."
  - –SO…. I head into see patient…

212 **CASE STUDY**

- VASC
  - –LP-OS
  - –IOP

- 22 mmHg (ORA)

213

214

215 **Case**

I am seeing RED

216 **Red Eye Day**

1 Case 2

2 • 52 yo female
- Contact lens wearer
- Red eye x 3 hours
- No discomfort, but feels dry
- VA unaffected

- Good health

3 Case 3

4 • 45 yo female
- Uncomfortable eye
- On and off x 8 months
- Positive allergy history
- No systemic meds other then OTC allergy

- Overall good health

217 **What do these have in common?**

218 **Conjunctival Redundancy**
Conjunctivochalasis

- Defined as a redundant loose nonedematous inferior conjunctiva
- Located between the globe and inferior eyelid

  - –Conjunctival folds lying along the inferior lid margin
- Some proposed *causes*:
  - –Aging
  - –Ocular surface inflammation
  - –Delayed tear clearance (cause or symptom?)
  - –More common in females
  - –More common in blepharitis patients
  - –Co-existing pinguecula
- Patients are mostly asymptomatic; however correct diagnosis crucial in symptomatic patients

219 **Redundant Conjunctival Folds**

220 **Conjunctival Redundancy**
Conjunctivochalasis

221 **Conjunctival Redundancy**
Conjunctivochalasis

222 **Conjunctival Redundancy**
Conjunctivochalasis

223 **ALL YOU SEE IS THE TIP OF THE ICEBERG**

224

225 **Go to the Source:**
Hydrodynamics (Water Movement) that Hydrates the Ocular Surface

226 **Conjunctival Redundancy**
Conjunctivochalasis

- Symptoms:
  - –Tearing/epiphora-especially with nasal folds due to blockage of punctum
  - –Dryness*
  - –FBS
  - –Redness

–Eye pain
–Blurry vision; especially in down gaze

227

228

229

230 **How Is Tear Flow Interfered by CCh?**

•CCh blocks tear flow into the punctum to cause delayed tear clearance

231 **What Are Unique Clinical Features of Dry Eye Caused by CCh?**

232 **Conjunctival Redundancy**
Conjunctivochalasis

233 **Conjunctival Redundancy**
Conjunctivochalasis

234 **Conjunctival Redundancy**
Conjunctivochalasis

• Treatment:
–Lubricants
–Antihistamines
–Topical Steroids
–Surgical resection of the conjunctiva

235 **Why Is CCh Refractory to Conventional Dry Eye Treatments?**

• Fornix Obliteration by CCh
  • Cannot hold patient's own tears
  • Seldom benefit from artificial tears
  • Easy to generate "overflow"
  • Restasis™ is not helpful

236

237 **Conclusion**

- CCh is an overlooked cause of dry eye.
- CCh dry eye differs from ATD dry eye in diurnal variation, symptoms changed by gazes and blinking.
- Symptomatic CCh is due to (1) interference of tear meniscus, (2) punctal drainage and (3) interference of tear flow from the fornix to the meniscus.
- Surgical treatment should be directed to deepening the fornix with AMT.

238 **Key Surgical Steps**

239 **DONE?**

240 **Case Study**

- Hypopion with Bullous
  - –Treatment
    - Durezol qid
    - Besivance qid
    - Homatropine 5% tid
    - Fresh Kote qid
    - Lotemax Ung-qhs
    - Sleep elevated….

241 **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

242

243 **Roth Spot**

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

244

245 **Roth Spot**

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

246 **Roth Spot: Associations**

1 COMMON

2 –DIABETIC RETINOPATHY
–HYPERTENSIVE RETINOPATHY
–HIV
–ISCHEMIA
–COLLAGEN VASCULAR DISEASE

3 LESS COMMON

4 –TRAUMA
–SHAKEN BABY SYNDROME
–MULTIPLE MYELOMA
–ARETRIOVENOUS MALFORMATION
–CARBON MONOXIDE POISOINING

247 **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, bleeding disorders
- Less common
  - –PT/INR: for clotting disorders
  - –Older pts: ESR/CRP to r/o GCA
  - –Younger pts (<40): lipids, aniphospholipids, ANA (lupus)

248

249 **Cotton Wool spots**

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

250 **MYELINATED NERVE FIBER**

251 **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

252 **Cotton Wool Spots**

- Ischemic
  - –VO, OIS, anemia, hyperviscosity/hypercoagulation, acute blood loss, radiation
- Immune
  - –SLE, scleroderma, polyaretrirtis nodosa, GCA
- Infectious

  - –HIV, cat scratch, Rocky Mountain Spotted Fever, leptospirosis, onchocerciasis, bacteremia/fungemia
- Embolic
  - –Carotid/cardiac emboli, endocarditis, deep venous emboli

253 **Cotton Wool spots**

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

254 **INTERFERON RETINOPATHY**

255 **INTERFERON RETINOPATHY**

256 **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

257 **Thanks!**