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Ocular Manifestations of Obstructive Sleep Apnea  
Dina Erickson, OD, FAAO

Outline:

Background:  
Obstructive sleep apnea (OSA) occurs when the muscles in the back of the throat relax to a level where they obstruct normal breathing. When the airway narrows or closes, breathing may stop for a few seconds, which lowers the blood oxygen level. The brain senses this and briefly rouses the person in order to re-start breathing and reopen the airway. This awakening tends to be very brief and the individual usually doesn’t remember it. This could happen a few times per night, which maybe normal. In OSA patients this event can happen several times per hour depending on the severity of OSA. OSA often goes undiagnosed and can lead to other serious ocular and systemic side effects.

Risk Factors:

- Overweight:  
  - Approximately 50% of OSA patients are overweight  
  - Larger waist  
  - Not all overweight people have sleep apnea  
  - Sleep apnea can occur in normal weight people

- Large neck size  
  - >17 inches for men  
  - >16 inches for women  
    - Increases risk

- Age:  
  - Between 18-60

- Narrow airway
- Family Hx of sleep apnea
- Smoking
- Alcohol use
- Chronic nasal congestion
- Gender:
- Race:
- Systemic conditions:  
  - HTN  
  - DM

OSA treatment:  
- Continuous positive airway pressure (CPAP) remains the most effective treatment option
Associates Systemic conditions:
- OSA is considered a serious medical problem:
  - Complications include:
    - Cardiovascular disease:
      - A large number develop HTN; a risk for heart dz
    - The more severe the OSA the greater risk for developing:
      - Coronary artery dz
      - Heart attack
      - Heart failure
      - Stroke
      - Arrhythmias
      - Sudden death from a cardiac event
  - Daytime fatigue:
    - Repeated awakenings make normal restorative sleep impossible
  - OSA patients tend to suffer from
    - Daytime drowsiness
    - Fatigue
    - Irritability
    - Poor concentration
    - Falling asleep during regular daily tasks
    - Complications with medications and surgery
    - Sleep-deprived partners
  - Symptoms may also include:
    - Memory problems
    - Morning headaches
    - Mood swings
    - Depression
    - Frequent night urination

Ocular manifestations:
- Floppy eyelid syndrome:
  - The most common ocular disorder associated with OSA
  - A relatively uncommon condition characterized by loose, easily everted upper eyelids. Floppy eyelid syndrome (FES) is often seen in overweight, middle-aged males
  - Symptoms consist of:
    - Ocular injection, irritation, itching and stringy mucous discharge, particularly upon awakening. The symptoms may appear unilaterally or asymmetrically.
  - Patients may complain of erratic sleep patterns, chronic sleepiness and morning headaches
  - Ocular signs:
    - Chronic papillary conjunctivitis
    - Mild to moderate bulbar hyperemia
- Punctate corneal epitheliopathy
- Stringy mucous discharge
- Lid ptosis
- Odd "rubbery" consistency to the lids

- Past Ocular Hx may include:
  - MGD
  - Hordeolum or chalazia
  - Keratoconus
  - Seasonal allergic conjunctivitis

- Treatment of FES includes:
  - Artificial tears
  - Antibiotic
  - Using tape or an eye shield during sleep
    - May help prevent spontaneous lid eversion

- **Glaucoma:**
  - Studies have shown increased incidence of POAG and NTG in OSA pts
  - Severity of glaucoma appears to be related to both the frequency and duration of apneic episodes.
  - Mechanism:
    - ONH damage is thought to be due to the ischemia caused by the apneic episodes
    - OSA is thought to cause poor auto regulation of the ON:
      - Causing further ischemic damage
  - Systemic workup if continuous progression despite adequate or aggressive treatment of glaucoma
  - Controversy over benefits of treatment of OSA in glaucoma patients

- **Papilledema:**
  - Several case studies have shown an association between papilledema and untreated OSA
  - Mechanism:
    - OSA results in forced inhalation against a closed airway.
    - This leads to an increase in venous pressure and impaired venous return
    - Which results in an increase in intracranial pressure.
    - Patients with non traumatic papilledema & normal neuroimaging, should be suspected for OSA
  - Continuous positive airway pressure (CPAP) use in patient with papilledema
    - Has been shown to improve or resolve papilledema

- **Nonarteritic Anterior Ischemic Optic Neuropathy (NAION)**
  - Is a result of impaired perfusion to the optic nerve head, or optic disc.
  - Two types
    - Nonarteritic anterior ischemic optic neuropathy is associated with 2.5 times higher prevalence in OSA patients than in controls
  - Mechanism of action
Non adherence to CPAP therapy can lead to second eye involvement

- **Retinal Vein Occlusion**
  - One of the most common causes of blindness not associated with DM
  - Usually occurs at night
  - Patients tend to:
    - Be older
    - Have HTN, DM and atherosclerosis
  - Studies show a strong potential for association
  - Is there a cause and effect?

- **Retinal Thickness**
  - Some studies have shown an inverse relationship between OSA and RNFL thickness
  - Not all studies agree on this

- **Retinal Sensitivity**:
  - Studies have shown that OSA patients have a diffuse decrease in retinal sensitivity
    - Without localized VF defects typical of glc etc
  - Pathogenesis is not clear

- **Keratoconus (KC):**
  - Strong association between FES and KC
  - KC patients have a higher risk of developing OSA
  - Patients with higher risk of developing OSA have a more severe KC
  - Proposed mechanisms:
    - Mechanical
    - Ischemia-reperfusion
    - Poor lid apposition
    - Decreased elastin fibers
    - MGD
    - Common inflammatory pathway
    - Genetic predisposition

- **Dry Eye and corneal abrasions**
  - Air leakage from the CPAP mask can lead to severe dry eyes
  - This in turn can lead to corneal abrasion and other corneal problems if left untreated or uncorrected.

- **Central Corneal Thickness**:
  - A recent study showed that CCT was significantly decreased in patients with OSA compared to the control group
  - CCT was found to be inversely correlated with the severity of OSA
  - Possible mechanism.
    - Hypoxia
    - Stromal changes include:
      - Edema
      - Stromal acidosis
• NV
• Changes in corneal shape

○ Conclusion:
  ▪ CCT tends to become thinner over time
  ▪ CCT should be measured regularly on OSA patients
  ▪ CCT thinning should be taken into consideration when measuring IOP and following glaucoma
What’s What? Diagnosis and Treatment of Contact Lens Complications

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Course Description:
The prompt diagnosis and management of contact lens related complications is important in every optometric practice to both promote good ocular health and to maximize patient comfort. This course will discuss soft contact lens complications, including diagnosis and management options. The characteristic of each complication will be reviewed as well as the etiology and differential diagnosis.

Course Learning Objectives:
After participating in this course, the participant should be able to:
1. Be familiar with the treatment and management of common contact lens associated complications.
2. Describe basic corneal physiology and its relationship to the etiology of contact lens induced complications.
3. Be familiar with current research in the area of complications.
4. Relationship of modality and replacement schedule to complications rates
5. Signs and symptoms of serious and significant complications and their incidence rates

Outline:
I. Lids and Lashes
   A. Meibomian Gland Dysfunction
      i. Results
         1. Correlation between length of wear and decrease in function of M-glands
         2. 30 year old CL wearer had M-score of ~65 year old non-CL wearer
         3. No significant difference between lens materials
      ii. Treatment
         1. Warm compresses
         2. Lid hygiene
         3. Therapeutics?
         4. Lipoflow Thermal Pulsation
   II. Tear film
       A. Dry Eye
          i. Nearly 40% of Americans experience dry eyes
          ii. 8% women 45-84 years old have a clinical diagnosis of dry eye
          iii. Evaporative
              1. Lipid layer
          iv. Aqueous deficient
              1. Aqueous layer
          v. CL drop out
             1. Discontinued lens wear
             2. “Successful” wearers report similar comfort issues
          vi. CL split the tear film
1. Reduced tear volume
2. Considerations
   a. Allergies
   b. Age
   c. Gender
   d. Medications
vii. Subjective
   1. FBS, burning, gritty, itchy, light sensitivity, tearing
viii. Objective
   1. Hyperemia, chemosis, lid involvement, corneal involvement
ix. DEQ – 5
x. Treatment
   1. CL parameter change
   2. Lid intervention
   3. Ophthalmic
   4. Oral
   5. Supplements
   6. Management of associated disease
   7. Scleral CL
   8. B. Mucin Balls
      i. 10-100 um discrete balls of mucin
      ii. Trapped between the CL and the cornea
      iii. Asymptomatic
         1. Not related to age, gender or Rx
      iv. Higher incidence in steep corneas, high modulus materials, CW and not use
         of rewetting drops
      v. Protective or not?
III. Conjunctiva
A. Limbal epithelial hypertrophy
   i. Asymptomatic
   ii. Observed in EW, HEMA wearers
   iii. Possible precursor to neovascularization
   iv. Must be viewed with Nafl
   v. Resolved in 3-5 days
      1. Decrease wearing time from EW to DW
B. CL Papillary Conjunctivitis (CLPC)
   i. GPC vs. CLPC
   ii. Mucus discharge, itching after lens removal, decreased WT
   iii. Stages 1-4
   iv. Immune reaction and/or mechanical
   v. Average blink per minute 12.55
   vi. Management
      1. Rule out mechanical cuase
      2. Manage deposits
      3. Lens care system
      4. Lubricaition
      5. Pharmacologic intervention
      6. Topical steroids
      7. Consider GP
C. CL Acute Red Eye (CLARE)  
   i. Response to gram (-) endotoxins on ens  
   ii. Resolution is rapid and complete – no known association w/ MK  
   iii. Signs  
      1. Bulbar hyperemia  
      2. Infiltrates  
   iv. Management  
      1. D/C lens ewar  
      2. Lubricate and cycloplege?  
      3. Re-establish DW first  
      4. Recurrence is possible  

D. Superior Limbic Keratoconjunctivitis (aka Thimerosal hypersensitivity)  
   i. Not to be confused with Theodore’s SLK  
   ii. Variable onset 2 months to 2 years  
   iii. Management  
      1. D/C SCL wear, eliminate exposure  
      2. Resolution is slow  

IV. Cornea – Epithelium  
A. Superior Epithelial Arcuate Lesion (SEAL)  
   i. Epithelial splitting superiorly, usually unilateral  
   ii. Lesion 2-5mm in length  
   iii. Little or no involvement of the bulbar conjunctiva  
   iv. Mechanical in nature  
   v. Management  
      1. D/C CL wear, lubricate  
      2. Change in BC or OAD and/or material  
B. Inferior Arcuate Corneal Staining  
   i. Course punctate epithelial disruption in the inferior cornea  
   ii. Appears independent of water content, lens thickness or refractive error  
   iii. Toxicity to debris accumulation  
   iv. Modulus dependant? Lens removal?  
C. Solution Induced Corneal Staining (SICS)  
   i. Corneal staining attributed to solution sensitivity  
   ii. Ocular inflammation of the palpebral conjunctiva  
   iii. Management  
      1. Change lens care product  
      2. Daily disposable  
      3. Continuous wear  
   iv. Topping off  
      1. Loss of efficacy of the lens care product  
   v. Rub vs. No-rub  
      1. Rubbing - Most effective way of removing surface deposits and pathogens  
   vi. Staining studies  
      1. Andrasko Grid  
         a. Observation after 2 hours of lens wear  
         b. Observed average percentage of corneal staining  
      2. IER Matrix Study  
         a. Observation over 3 months  
         b. Observed percentage of patients with corneal staining
D. Preservative-Associated Transient Hyperfluorescence (PATH)
   i. Nafl is binding to the preservative on the epithelium
      1. Peak binding of Polyquad/Aldox is 30 minutes
      2. Peak binding of PHMB is 2 hours
   ii. Noted after lens application
   iii. Diffuse corneal staining – epithelium unaffected
   iv. Resolution is 6-8 hours post lens removal
E. Microcysts
   i. Asymptomatic
   ii. 15-50 microns translucent or gray consisting of cellular debris
   iii. Do not stain until they break through the surface epithelium
   iv. Best seen with reversed illumination
   v. Thought to be from metabolic stress
   vi. Management
      1. Decrease wearing time
      2. Higher Dk material – rebound

V. Cornea – stroma
A. Vascularization
   i. Limbal hyperemia
      1. Increased blood flow at the limbal arcades
   ii. Neovascularization
      1. Vasostimulation Theory
         a. CL induced epithelial trauma results in a release of enzymes
         b. Inflammatory cells migrate to the site and release
            vasostimulating agents that cause vessels to grow in the
            direction.
      2. Hypoxia Theory
         a. Tissue hypoxia results in an increased production of lactic
            acid which may result in venous drainage
         b. Chronic edema results in stromal softening or loss of
            compactness reducing the physical barrier to vessel
            penetration
            i. Generally thought that hypoxia alone cannot cause
               vascularization
      3. Oxygen permeability
         a. Dk vs. water content / silicone
      4. Management
         a. D/C lens wear
         b. Treat any underlying pathology
         c. Minimize physical insult
      5. Corneal edema
         a. Epithelial vacuoles
            i. 10% of non-wearers, unknown etiology
            ii. 5-30 microns spherical fluid or gas filled vacuoles
            iii. Unreversed illumination
            iv. Generally asymptomatic
         b. Epithelial Bullae
            i. Low prevalence in CL wearers
            ii. Chronic epithelial edema
iii. 5-30 microns irregular shaped that may coalesce in the central cornea
iv. Generally asymptomatic
   1. In severe cases, the bullae may break through the epithelial surface = pain!

VI. Cornea – Keratitis
A. Infiltrate – focal accumulation of cells or tissue within the anterior stroma
   i. PMD leukocytes
   ii. Mononuclear cells
   iii. Infectious or sterile
   iv. 1 week to 20 years following initial fitting
   v. Physical factors
      1. Limbal redness, prior inflammatory event, corneal staining
   vi. Response
      1. Topical or systemic medications
      2. Preserved lens care systems
      3. Immune reaction
      4. Bacterial infection
      5. Viral infection
      6. Staph hypersensitivity
      7. Corneal hypoxia
      8. Dystrophy
      9. Exposure
   vii. Infiltrates will appear with almost any chronic irritation to the cornea
B. Infiltrative keratitis
   i. Inflammatory reaction of the cornea
   ii. Mild to moderate irritation, mild/moderate irritation, redness, occasional discharge
   iii. Signs
      1. Anterior stromal infiltration
      2. With or without epithelial involvement
      3. A/C reaction rare
      4. Can be bilateral
      5. No lid edema
      6. Moderate redness
      7. VA may or may not be affected
   iv. Etiology
      1. FB entrapment
      2. Mechanical trauma
      3. Bacterial toxins
      4. MPS reaction
   v. Risk factors
      1. CL wear
   vi. Differential Diagnosis
      1. Viral keratoconjunctivitis, CLARE, CLPU
   vii. Management
      1. D/C CL wear
      2. Steroid if moderate symptoms or decreased vision
      3. Lubricate
      4. Recurrence is possible – especially if toxic reaction
5. Switch to single use or preservative-free lens care
viii. Rarely scars vs. CLPU (Bulls’ eye scar)
C. CL Peripheral Ulcer (CLPU)
i. Signs
1. Peripheral location
2. Regularity of borders
3. Absence of photophobia, no visual involvement, no A/C reaction
4. Rapid resolution
5. Infiltrate
6. 
ii. Symptoms
1. Discomfort – moderate to severe FB sensation to asymptomatic
2. Redness – slight
3. Tearing
iii. Etiology
1. Inflammatory reaction to gram (+) exotoxin
   a. Released by S. aureus
   b. Bacteria rare in cultures
iv. Management
1. Anti-infective agent
2. Cycloplege (if A/C reaction – rare)
3. Steroids after re-epithelialization
4. Monitor closely
D. Microbial Keratitis
i. Focal defect or excavation of the sub-epithelial corneal surface
   1. Produced by sloughing of necrotic inflammatory tissue
   2. Must have an acute inflammatory infiltrate of the epithelium & stroma in the presence of an infectious microorganism
   3. Infection of the corneal surface cannot occurs without initial bacterial attachment or binding to epithelial cells
      a. Normal cornea binds few bacteria so spontaneous infection is rare
      b. Traditional EW – epithelium becomes edematous – increased attachment of microorganisms – compromised epithelium
   4. Infiltrate
      a. Central or paracentral, large & irregular, satellite lesions, anterior stroma to full thickness, corneal edema, epithelial loss, A/C reaction, lid involvement, bulbar and limbal redness, unilateral, hypopyon
ii. Symptoms (mild to severe)
   1. Pain, photophobia, tearing, blepharospasm, red eye, floaters, AM lid crusting, purulent discharge
iii. Risk factors
   1. Trauma, surface disease, smoking, age, high ametropia, lens replacement, years of wear, CL material, CL case care, water exposure, illness and sleeping in CL
   2. Extended wear
      a. Schein & Poggio – incidence rate
         i. 1/500 for EW and 1/2500 in DW
b. Cheng
   i. 1/500 in EW and 1/2857 in DW

   c. Schein & McNally - Silicone hydrogel
      i. 0.3 to 3.6 per 10,000

   d. Morgan & Efron
      i. Higher risk of severe keratitis in EW
         ii. EW in silicone hydrogel carry 5x less risk of severe keratitis

   e. Keay & Stapleton
      i. Principle risk factor of MK is EW

   f. Stapleton – Annualized incidence per 10,000
      i. Overnight wear - the risk of MK and associated vision loss is the highest.

3. Contact Lens Assessment in Youth (CLAY)
   a. SCL complications are related to age
      i. 15-25 years old at the most risk
         1. More likely to nap in SCL
         2. More likely to sleep in their lenses in different situations
         3. More water exposure

4. Comparing risk - LASIK
   a. Vision loss of two or more lines
      i. 0.5 to 1.4% of individuals during the intraoperative and early post-operative
      ii. 1 per 2500 late post-operative
         1. Primarily from post-surgical ectasia

b. “…equivalent to the risk following 20 years of EW hydrogel wear where lenses are used for 6 nights continuously or silicone hydrogel contact lens use where lenses are used for 30 nights continuously”. (Stapleton OVS 2007)

iv. Bacterial (Pseudomonas, Serratia, Staph, Strep)
   1. Pseudomonas – one of the most common isolated in CL related MK
      a. Liquefactive necrosis – perforation in 48 hours
      b. Semi-opaque ground glass appearance

   2. Invasion of an intact epithelium
      a. Corynebacterium diphtheria
      b. Listeria
      c. Haemophilus

   3. Culture
      a. Less than 1 mm from the pupil, 3 or more infiltrates, 3 mm or greater in size

4. Treatment
   a. Broad spectrum antibiotic

v. Protozoa (Acanthamoeba)
   1. Appear dendritic or patchy stromal infiltrates
   2. Symptoms disproportionate to signs
      a. Early – lots of pain, not a lot going on
   3. Risk factors
      a. CL wear (90% of cases)
      b. Injuries from vegetative matter
c. Hot tubbing

4. Treatment
   a. Brolene 1%, Neosporin, Chlorhexadine, oral itraconazole
   b. Penetrating keratoplasty

vi. Fungal (Aspergillus, Candida, Fusarium)
   1. Large white infiltrate with fluffy or branching margins
   2. Significant edema
   3. Fusarium
      a. Found in soil, vegetation and water
   4. High risk of loss of BCVA
   5. Treatment (NO STEROIDS)
      a. Natamycin 5% and/or Amphotericin B 0.15%, oral itraconozol and cycloplete

vii. Viral
   1. Simplex
      a. Course punctate staining w/ linear branching and terminal end bulbs
      b. Geographic appearance
      c. Pseudodendrite (epithelial heaping)
      d. Treatment
         i. Zirgan gel, Vira-A ung, Viroptic, acyclovir (or famciclovir or valacyclovir)
   2. Zoster
      a. No terminal end bulb on pseudodentdrites (raised mucous plaques)
      b. Treatment (most effective within the first 3 days)
         i. Lubrication, topical steroid, cycloplege, acyclovir (or famciclovir or valacyclovir)

viii. Treatment
   1. Progressively worsens without treatment
   2. Discontinue CL wear immediately
   3. Corneal scraping and antimicrobial therapy
   4. Referral to a conreal specialist if in visual axis or not responding to therapy

E. Wearing modality and material
   i. Daily disposable – convenient, decrease deposit formation, decrease incidence of complications
   ii. Silicone hydrogel – increased oxygen permeability, equal risk of infiltrative event

VII. Summary of CL associated serious and significant events
A. Rare
B. Absolute risk has remained constant for DW and EW respectively
C. Occurrence
   i. 1 in 10,000 for GP
   ii. 3-4 in 10,000 for DW SCL
   iii. 10-20 in 10,000 for EW SCL
D. Vision loss with CL related to MK
   i. 0.3 to 3.6 in 10,000
E. Sterile keratitis
   i. 1-7% of SCL wearers
F. Principle risk factor for MK is overnight wear

VIII. Cornea – Endothelium
A. Endothelial Bedewing
   i. Deposits of unknown etiology in patients who are CL intolerant
      1. Not necessarily induced by CL wear
   ii. Idiopathic – 20% occurrence in non-CL wearers
B. Endothelial Blebs
   i. Black, non-reflecting areas
   ii. 100% prevalence in CL wearers
   iii. Rapid onset – 10 minutes after application
   iv. Rapid resolution – 2 minutes post removal
   v. Adaptation to endothelium
      1. Blebs peak at 20-30 minutes then decrease after ~1 hour
      2. Diurnal fluctuations and decrease over length of wear
   vi. Asymptomatic
   vii. Relatively minimal clinical significance
C. Polymegathism
   i. Change in cell size
D. Pleomorphism
   i. Change in cell shape

IX. Put it into practice
A. Evidence based care
B. Identifying those at higher risk
   i. 15-25 year olds
   ii. Extended wear
C. Customize prescribing habits to decrease risk
D. Customize patient education to decrease risk
E. Practitioner resources
   i. Efron Grading Scales
   ii. IER / LVPEI Guide – differential between MK and CLPU
F. Patient resources
   i. Association of CL Educators
   ii. FDA
   iii. IER (Brien Holden Vision Institute)
   iv. Industry
G. Targeted education
   i. University of Waterloo
   ii. CDC
Current Trends in Uveitis Management
Dina Erickson, OD, FAAO

Outline:

Background:
- A group of inflammatory disease that affect the highly vascularized uveal tissue.
- A collection of pathological conditions with similar clinically observable signs.
- May be associated with systemic disease.
- May lead to blindness if left undetected or untreated.
  - Early detection and treatment is essential.

Types of uveitis:
- Anterior uveitis
- Intermediate uveitis
- Posterior uveitis
- Panuveitis uveitis

Effective Uveitis management overview:
- Check IOP:
- Check for any previous ocular surgery
- Evaluate severity
- Consider systemic involvement
- Rule out corneal causes
- Treat aggressively and restore the blood aqueous barrier
- Examine the post seg

Check IOP:
- Usually goes down initially due to CB inflammation
- Can then go up with increased cells and debris in the CB
- Certain etiologies can cause IOP to go up
  - Angle closure
  - HZO

Ocular surgery:
- Uveitis after ocular surgery can mean endophthalmitis
- After cataract surgery
  - Can be up to 10 days after surgery
    - According to 2009 & 2010 Surveys
- After Anti VEGF tx
• After Blebs
• Other ocular surgery

**Evaluate Severity:**
• Determine if a systemic workup is necessary
• Signs to look for:
  - Keratic Precipitates (KPs)
  - Bilaterality
  - Recurrence
  - Hypopyon
  - PAS
  - Synechae

**Systemic involvement:**
• Initial tests to run:
  - Check lymph nodes
  - CBC with diff
  - HLS-B27 antibody (+in ~50% of iritis pts)
  - SED rate
  - ANA (antinuclear antibody)
  - FTA_ABS (fluorescent treponemal antibody absorption)
  - ACE (angiotensin converting enzyme)

**Rule out corneal causes:**
• Corneal trauma/abrasion
• Corneal infection

**Treat aggressively:**
• “Hit it hard”
• Start with at least q2h or q1h.
  - PF brand or generic?
  - Availability
• Consider using a stronger steroid
  - Durezol QID
    - Can be used at ½ the frequency of PF for equal efficacy
    - BAK free
    - Emulsion so no shaking needed
      - Makes it more bioavailable
    - Comparison to PF
    - FDA approved 2008
  - Lotemax ung qhs
    - Benefits of using Lotemax ung
      - Doesn’t increase IOP
      - Non-preserved
      - Effective in post-op inflammation and pain
• Remember that ophthalmic steroids are contraindicated in most virus induced corneal and conj diseases.
• Can steroids be used for corneal ulcers:
  o Studies regarding steroid use with corneal ulcers
  o Take home message
• Don't forget Cycloplegia
  o Homatropine 2%, 5%
  o Cyclopentolate 1%, 2%
• Why do we cycloplege?
  o Restablishes vasc permeability
  o Prevent synechia
  o Manages pain
  o Stabalize the blood-aqueous barrier and help prevent further protein leakage (flare)
  o Secondary benefit:
    ▪ Dilation
• Consider the risk of relapse

The Multicenter Uveitis Steroid Treatment Trial
• To compare relative effectiveness of
  o Systemic corticosteroids plus immunosuppression when indicated
  Versus
  o Fluocinolone acetonide implant (implant therapy) FOR
  o Noninfectious uveitis

Outcomes:
• Substantial VA improvement in both groups
• The implant group achieved inflammatory control both faster and more often
• Complications:

Examine the post seg:
• Need to rule out systemic involvement
• Look for post uveitis
Course Outline

I. Keratoconus
   A. 23 year old female presents with a chief concern of decreasing vision OD>OS for past 2 years
      1. Rarely wears spectacles
      2. Previous eye exam 5 years ago
      3. POH
         a. LASIK consult 6 months prior
   B. Exam
      1. Reduced best corrected spectacle acuity
      2. Keratometry
      3. Retinoscopy and MR
      4. Anterior segment
         a. Unremarkable
         b. Differential Fleisher’s Ring, Kayser-Fleischer Ring, Hudson-Stahli line
      5. Topography
         a. Inferior steepening with superior/temporal flattening
   C. Treatment options
      1. Spectacles
      2. Corneal GP
      3. SCL / Customs SCL
      4. Scleral CL
      5. Implantable intracorneal corneal ring segments
      6. Corneal cross-linking
         a. Riboflavin
         b. Patient selection

II. Steven’s Johnson Syndrome (SJS)
   A. 31 year old male with SJS since 3 years old suspected triggered from anticonvulsant medication
      1. Hospitalized
      2. Copious lubrication
      3. POH
         a. Severe dry eye
         b. Trichiasis
         c. Cicatricial entropion
         d. Symblepharon
         e. Foreshortened fornix
         f. Corneal thinning and stromal fibrosis
B. Exam
   1. Significantly reduced best corrected spectacle acuity
   2. Medications
      a. Liquigel QID OU, Lacri-Lube QHS OU
   3. Anterior segment
      a. Trichiasis, symblepharon, ectropion OS, corneal thinning/scarring, punctate staining
C. Life and sight threatening disease
   1. Hypersensitivity to drugs or food
   2. Rarely occurs after 35 years old
   3. Involves all bodies mucous membranes
   4. Acute episode with chronic effects
D. SJS by the numbers
   1. Incidence is 0.4 to 1 case per million
   2. Mortality 1-5%
   3. Ocular complication 50%
   4. Erythema Muliforme (mild) to Toxic Epidermal Necrolysis (severe)
      a. Varying locations and severity
   5. Unknown mechanism
      a. Genetic component?
      b. Triggers
E. Treatment of long-term ocular complications
   1. Lubrication
   2. Contact lenses
III. Scleral contact lenses
   A. Moisture chamber – vault the entire cornea
      1. What is the best solution?
      2. Therapeutics?
   B. Fitting
      1. Goals
      2. Sagittal height
      3. Parameters
         a. Central zone
         b. Limbal zone
         c. Scleral zone
      4. Materials
   C. Application / removal
      1. DMV
      2. Two/three finger techniques
      3. Lens Inserter
   D. Complications
      1. Deposits / poor wetting
      2. Conjunctival prolapse
      3. Lens Fogging
Preseptal cellulitis:

**History:**

- Called to the medical side clinic for an emergency consult. MD is panicking about a patient’s eye and wants our opinion.
- A 53 year old Hispanic female with a painful puffy left eye (Spanish speaking)
- Started a week prior with a small nodule on the superior left lid that she picked at
- Got much worse by Sunday so she went to urgent care
- She was given Cephalexin at urgent care that she has been taking
- Yellow discharge last 2 days but none today
- Had a similar episode 6 years prior and the lesion had to be “cut out and drained”
- LEE was 1 yr prior at Kaiser
- Her systemic health was unremarkable except she had a cold 2 weeks prior
- What would you do next?
- What other tests would you want to do?
- What other questions might you want to ask?

**Objective findings:**

- VAs 20/30-2 OD, 20/25-1 OS
- Ant seg exam:
  - OD unremarkable
  - OS: See photo of lids, otherwise conj, cornea and AC unremarkable
- **DDX?**
  - Preseptal cellulitis
  - Orbital cellulitis
- How did we make the diagnosis

**Preceptal cellulitis:**

**Overview:**

- A relatively common eyelid infection that involves the periorbital tissue anterior to the orbital septum
- Acute eye lid edema and erythema

**Causes:**

- Local spread of URI:
  - Usually the cause, if no evidence of trauma or local lid infection is found.
- Spread of eyelid infection
- Following trauma to the eyelids
- Spread of a sinusitis or dacryocystitis
- Following an insect bite to the eyelids

**Offending Pathogens:**

- Most common ones;
  - Staphylococcus aureus
  - Staphylococcus epidermidis
  - Streptococcus species
  - Haemophilus influenzae
- You MUST Rule out Orbital Cellulitis!!

**Typical Objective findings:**

- Acute eyelid erythema,
- Conjunctival injection
- Tenderness to touch
- Pain (lid pain)
- Eyelid edema
- NO APD
- NO PROPTOSIS
- NO PAIN ON EOMs
- NO EOM restriction
- Usually Normal VAs
- Orbital CT is necessary if the eyelids cannot be separated to evaluate for:
  - Proptosis
  - Limited ocular motility
  - VA loss

**Treatment:**

Mild preseptal cellulitis:

Empiric Tx for adults and older children:

- Amoxicillin/clavulanate (Augmentin)
- Mild to moderate: 500/125 mg PO q12hr or 250/125 mg PO q8hr for 10 days
- Severe: 875/125 mg PO q12hr or 500/125 mg PO q8hr or 2000 mg (2 extended-release tabs) PO q12hr for 7-10 days
- For children older than 5 (<40Kg): 25 to 45 mg/kg/day p.o. in two divided doses,
- Maximum daily dose of 90 mg/kg/day
Other antibiotic options:

- Cephalosporin such as cefaclor (Ceclor) can also be used.
  - 250-500 mg, by mouth, three times per day for 10 days (for adults and older children)
  - 20-40 mg/kg/day by mouth, in three divided doses with a maximum of 1g/day for 10 days (for younger children).

Hospitalize for i.v. antibiotics IF:

- Moderate to severe cellulitis
- Patient is toxic
- Patient may be noncompliant
- Child younger than 5 years
- No noticeable improvement or worsening after a few days with Tx.

Central Retinal Artery Occlusion

History:

82 year old Caucasian female complaining of “no vision” in the right eye since Nov and wants to know if there is anything we can do for her. She was seen by the local ophthalmologist and now wants a second opinion. Doesn’t want to tell us what he found so we can come up with our own diagnosis.

Review of systems is positive for:

- Carotid artery blockage with repair 2 years prior
- Type 2 DM
- HTN
- Ovarian cancer stage 1
- Seasonal allergies
- GI issues

Meds:

- Lisinopril
- Metformin
- Simvastatin
- Gabapentin
- Tricor
- Lasix

Objective findings:
VAs:
- OD: HM, OS 20/25

Pupils:
- Positive APD OD

Ant Seg
- Unremarkable

IOP:
- GAT: 9 mmHg OD & OS

Post Seg:
- Mid peripheral hemes OD
- Hollenhorst plaque superior temporal to disc.
- OCT and fundus photos taken. See slides

Central Retinal Artery Occlusion

Overview:
- Sudden, painless and severe loss of vision
- Vision loss occurs due to loss of blood supply to the inner layer of the retina.
- Acutely, obstruction of the central retinal artery results in inner layer edema and pyknosis of the ganglion cell nuclei.
- The retina becomes opacified and yellow-white in appearance due to the ischemic necrosis
- A cherry red spot is seen in the fovea due to:
  - Intact RPE and choroid underlying the fovea
  - The fovea is nourished by the choriocapillaris

Mortality:
- Life expectancy of patients with CRAO is 5.5 years compared to 15.4 years for an age-matched population without CRAO

Causes:
- Can vary depending on the patients’ age and comorbid diseases present.
- Atherosclerotic changes:
  - The LEADING cause of CRAO in pts 40-60 years of age
  - Seen in 45% of cases
  - 20% of cases have 60% or greater stenosis
- HTN tends to be present in 2/3 of pts
- DM
Cardiac abnormalities
Emboli:
  - Can be cholesterol, calcific or talc
  - Heart emboli are the most common cause in patients younger than 40
  - Associated with worse VAs.
  - Associated with higher morbidity and mortality rates

Work up:
- CBC
- ESR
- Fasting BG
- Blood cultures to evaluate for bacterial endocarditis and septic emboli
- Imaging:
  - Carotid ultrasound
  - FA
- OCT findings:

Treatment:
- Ocular massage
- Lower IOP
- Anterior chamber paracentesis
- Hyperbaric oxygen

Macular Hole:

History:
- A 39 year old Caucasian female presented with sudden painless vision loss in her left eye.
- No improvement with refraction
- Photos
- OCT

Macular hole review:
- Pathophysiology
- Stages of macular hole
- Risk factors
  - Age
  - Gender
  - Elevated plasma fibrinogen
- Impact on quality of life
- Treatment options:
o Observation
o Vitrectomy
  ▪ Advantages
  ▪ Disadvantages
o Ocriplasmin: Jetrea FDA approved Oct 2012 for the Tx of VMT
  ▪ How it works
  ▪ Patient selection
  ▪ Simulates surgery
  ▪ Clinical efficacy
  ▪ Safety

Future potential

IV. Wrap-up
Tulalip CE
Sunday, Sept 20, 2015
Tulalip Resort Casino,
Tulalip, Washington
6 hours, $250
Dina Erickson & Beth Kinoshita

Homecoming CE
Jefferson Hall, Pacific University
Saturday, October 3, 2015
6 hours, $100 (special homecoming rate)

GLAUCOMA SYMPOSIUM
Saturday, January 9, 2016
Willows Lodge, Woodinville, Washington
7 hours with Howard Barnebey & Murray Fingeret
For more information contact FREDERIM@pacificu.edu

2016 ISLAND EYES CONFERENCE
January 17 - 23, 2016
Sheraton Maui Resort
Up to 29 hours of OD Education $700 - $800
Nate Lighthizer, Leo Skorin, Denise Goodwin, Stanley Teplick,
and featured speakers from Waterloo Class of 1994

Coeur d’Alene CE
April 29 & 30, 2016
The Coeur d’Alene Resort, Idaho
10 hours $350

2016 VICTORIA CONFERENCE
July 21 – 24, 2016
Delta Ocean Pointe, Victoria, BC
20 hours of education $450 - $550

For more conference Information contact: JEANNE@pacificu.edu