Grand Rounds for the Full Scope Optometrist

Joe Munsell, O.D. Dipl. ABO, FAAOMS
jmunsell25@gmail.com
Cheyenne Vision Clinic, Laramie Vision Clinic

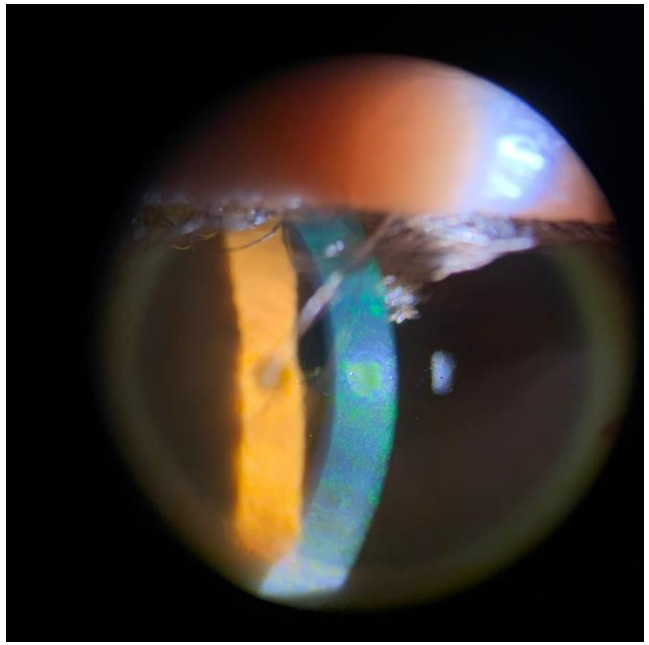
- No Financial Disclosures

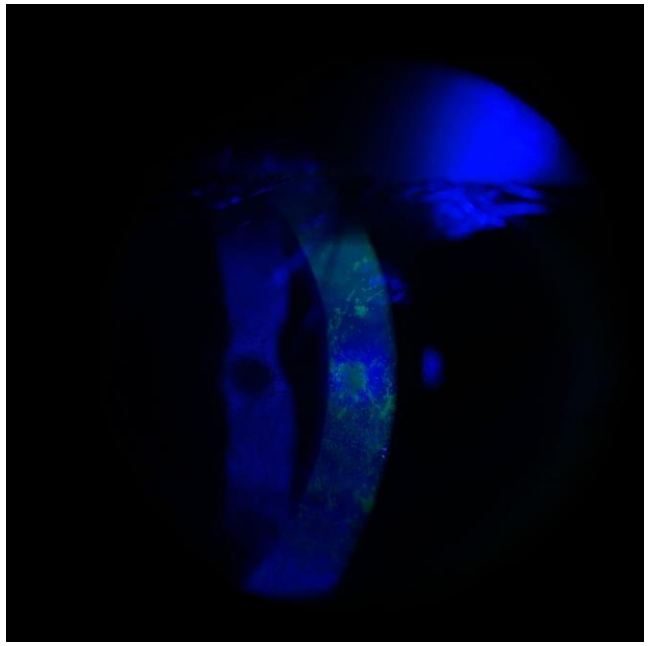
62 YO Male Patient

- Treated by urgent care for a corneal abrasion 3 days ago
- Rxd erythromycin ointment
- Slight irritation and blur at all distances
- Not much improvement per patient

62 YO Male

- 20/60 BCVA OS
- IOP Normal
- 1+ bulbar injection
- Abrasion, 2x2 mm
- 2+ Diffuse SPK





Case Hx

- BCL put in place
- Started Vigamox QID
- PF ATS Q2H

2 day follow up

- No improvement
- Abrasion as large as previous visit
- Pain minimal

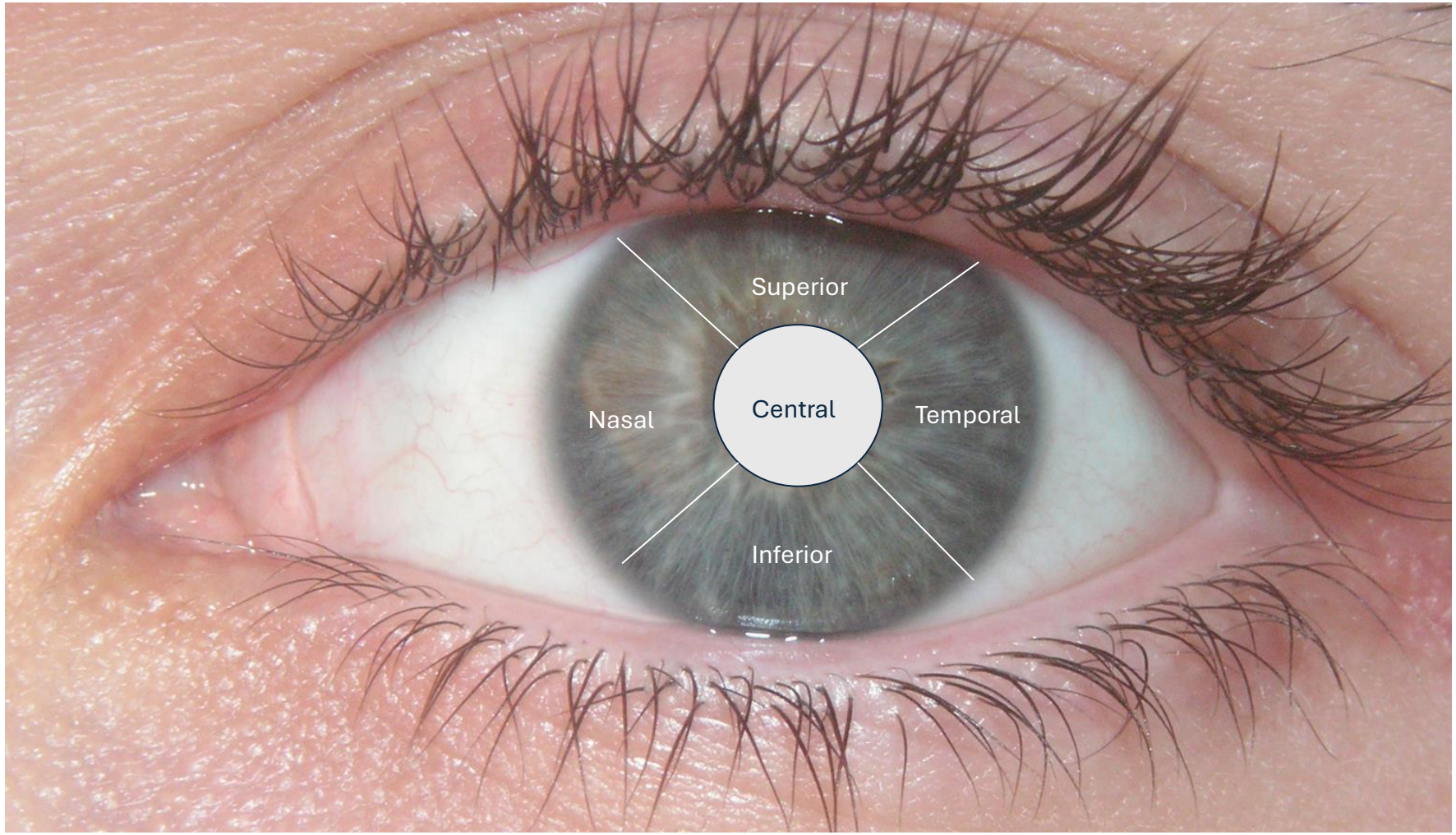
- Further questioning the patient mentioned they had a stroke 6 months prior
- Blink testing showed an incomplete lid closure and an exposure keratitis
- We then performed corneal sensitivity testing and patient had reduced sensitivity
- New Dx: corneal hypoesthesia, neurotrophic keratitis (ulcer)
- Started process of prescribing Oxervate

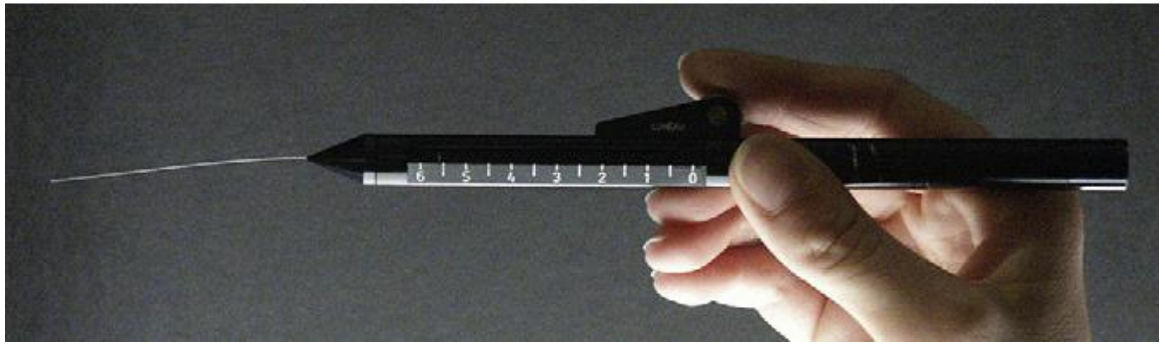
Causes of NK

- Herpetic infections
  - 6% of HSV keratitis, 12.8% of Zoster keratitis
- Physical injury
- Contact lens wear
- Medication induced (BAK)
  - Glaucoma meds, NSAIDS
- Refractive surgery (LASIK)
- Cataract Surgery
- PRP
- RD repair
- Corneal Dystrophies (lattice and granular)

- Systemic Disease
  - Diabetes, MS, Stroke, Vitamin A deficiency
- Masses- Acoustic Neuroma
- Jaw Fracture Reductions and trigeminal neuralgia ablation
- Rare in children
  - Dysautonomia, goldenhair-gobar, Mobius syndrome, familial corneal hypoesthesia

Corneal Sensitivity Testing

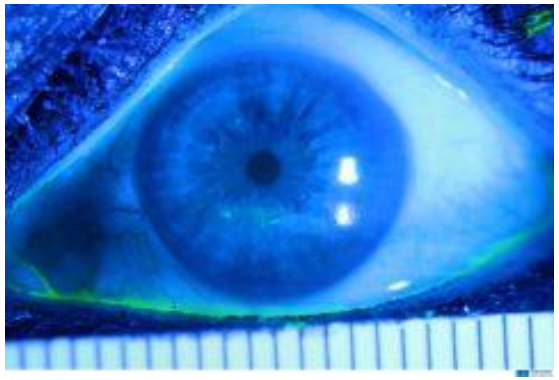




Stages of NK- Mackie Classification

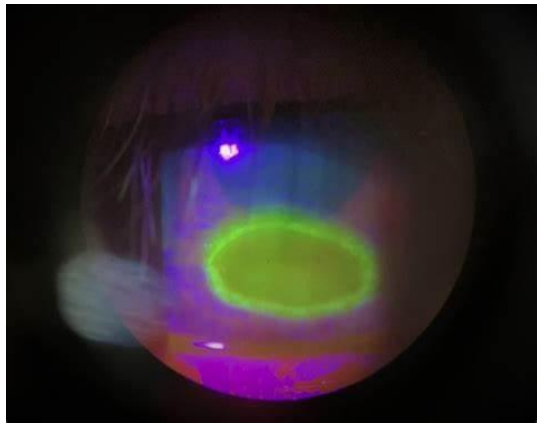
Stage 1:

- corneal epithelial changes with dry and cloudy corneal epithelium
- superficial punctate keratopathy
- corneal edema.



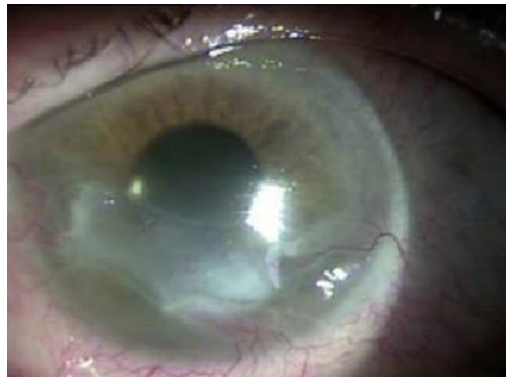
Stage 2:

- recurrent and/or persistent epithelial defects with an oval or circular shape,
- Commonly in superior half of the cornea.



Stage 3:

- corneal ulcer with stromal involvement
- stromal melting
- corneal perforation.



Treatment

- Preservative artificial tears
- Topical antibiotics
- Matrix metalloproteinase inhibitors to reduce risk of corneal melt
- Autologous Serum
  - (Matsumoto, 2004) showed substantial healing in PED's in 7-28 days

Results: The epithelial disorders healed completely in all eyes within 6 to 32 days (mean, 17.1+/-8.0 days), with a decrease in corneal scarring. The mean pretreatment corneal sensitivity was 11.8+/-11.6 mm, which increased to 30.0+/-22.9 mm after treatment at the last follow-up. Five eyes attained normal corneal sensitivity with treatment. The BCVA improved by >2 Landolt lines in 78.6% of the

Treatment

- Amniotic Membranes
- Topical Insulin
  - 1 unit per ml 2-3 x daily
- Oxervate
- Surgical procedures
  - Corneal Neurotization

Use of Topical Insulin to Treat Refractory Neurotrophic Corneal Ulcers

Angeline L Wang [1], Eric Weinlander, Brandon M Metcalf, Neal P Barney, David M Gamm, Sarah M Nehls, Michael C Struck

Affiliations + expand

PMID: 28742619 PMCID: PMC5633504 DOI: 10.1097/ICO.0000000000001297

Abstract

Purpose: To report the clinical course of 6 patients with refractory neurotrophic corneal ulcers that were treated with topical insulin drops.

Methods: Retrospective chart review of patients who had neurotrophic corneal ulcers or epithelial defects refractory to standard medical and surgical treatment. Insulin drops, prepared by mixing regular insulin in artificial tears with a polyethylene glycol and propylene glycol base at a concentration of 1 unit per milliliter, were prescribed 2 to 3 times daily.

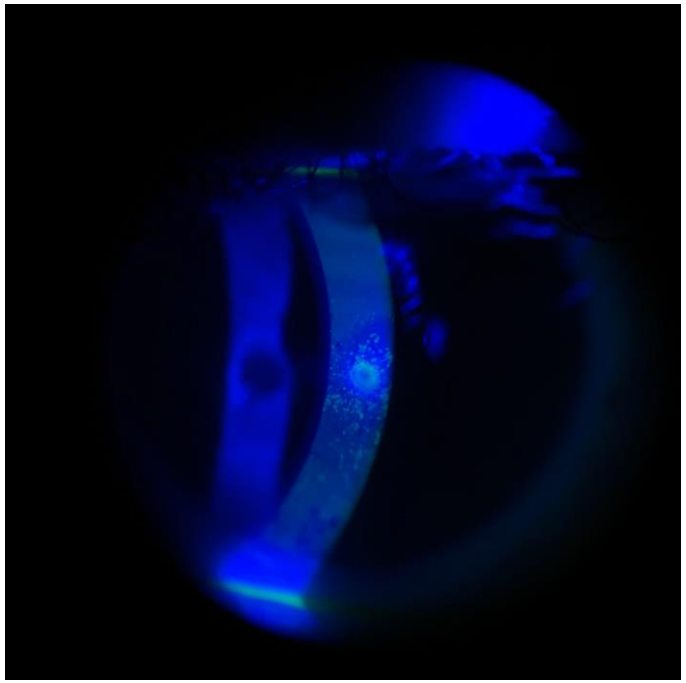
Results: Six patients, aged 2 to 73 years, developed neurotrophic corneal ulcers refractory to a range of medical and surgical treatments, including bandage contact lens, amniotic membrane grafting, and permanent tarsorrhaphy. Each patient was administered topical insulin drops with complete corneal reepithelialization within 7 to 25 days.

Oxervate

- Cenegermin (recombinant human growth factor)
- First FDA approved treatment of NK (2018)
- 1 drop 6 x a day for 8 weeks
- “two eight-week, randomized controlled multi-center, double-masked studies.Across both studies, complete corneal healing in eight weeks was demonstrated in 70 percent of patients treated with Oxervate compared to 28 percent of patients treated without cenegermin”

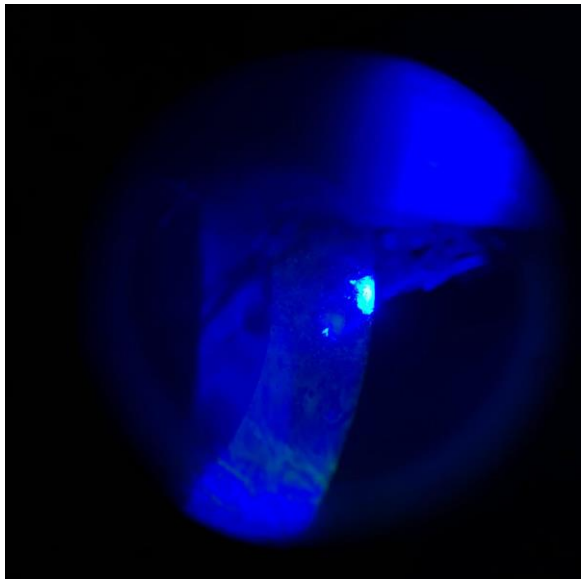
1 week follow up

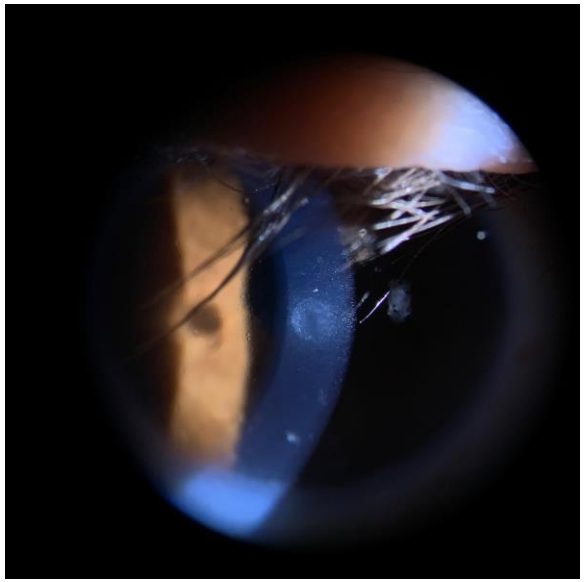
- 20/50 BCVA
- 30% less staining



6 week follow up

- 20/30 BCVA
- 70% resolution





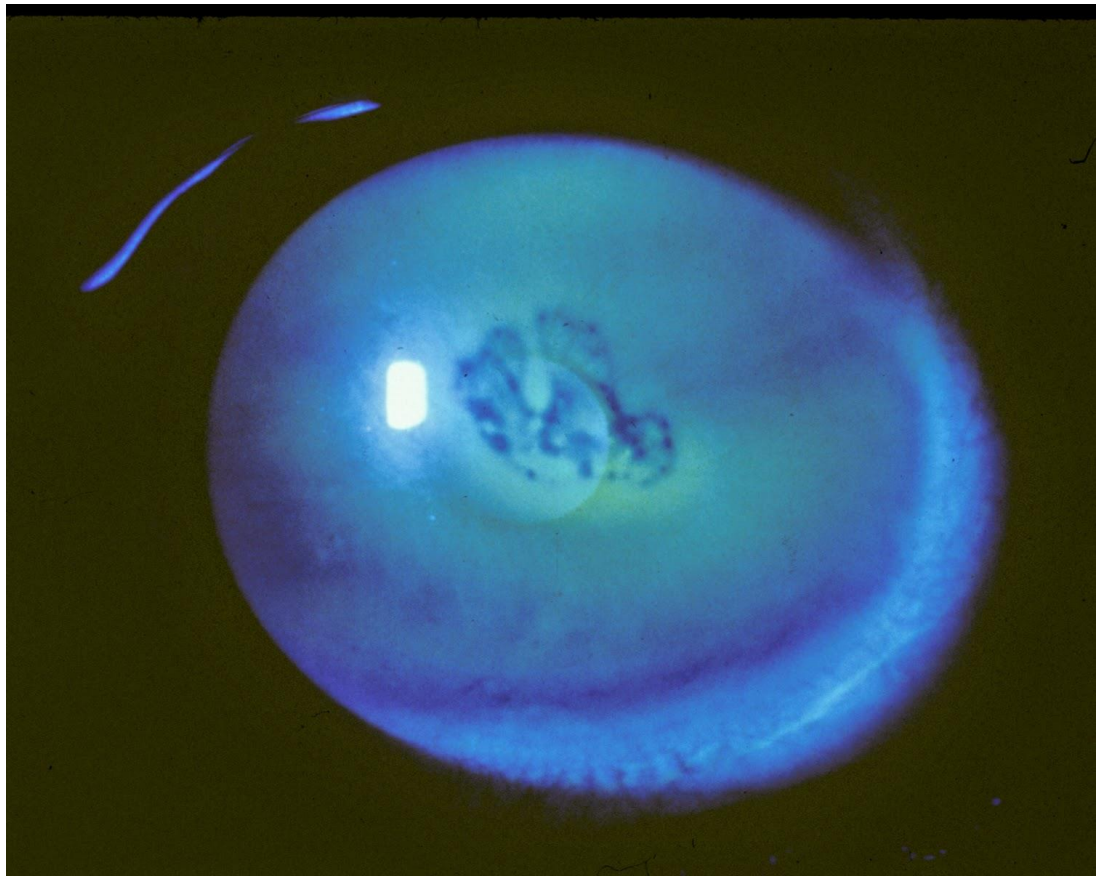
NK Poll Question

48 YO White Female

- Woke up at 2 am with 7/10 pain
- BCVA 20/20 Od 20/50 OS
- IOP normal
- Ocular history of EBMD, Fuchs
- Ocular medications: using Muro 128 at night and Pf ATS throughout day

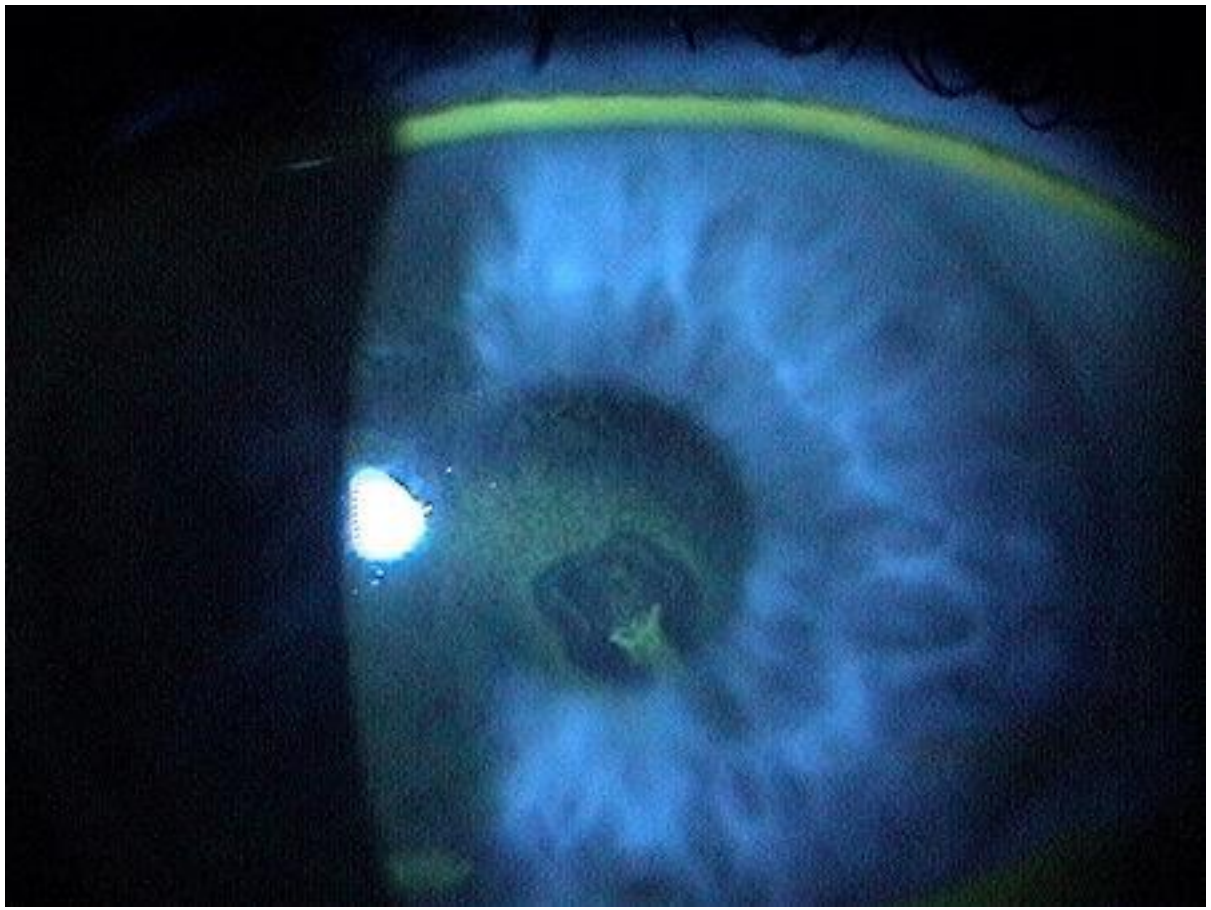
Recurrent Corneal Erosions

- Most RCE's occur in anterior ⅓ of cornea
- Large erosions easier to manage
- Micro erosions more difficult- small break in AM then re-epirheliaize by the time they present in office
- Pain, redness, tearing
- Associated with corneal dystrophies, history of trauma or infection, DED, diabetes, blepharitis, rosacea



Acute Treatment

- Constant lubrication
- Muro 128
- Antibiotics
- Bandage Contact Lens
- Environment
  - Ceiling fans



Recurrence treatment

Debridement

Doxycycline

- 50 mg BID
- Prevents breakdown of collagen and hemidesmosomes
- Down regulates production of lipase

BCL

Amniotic Membranes

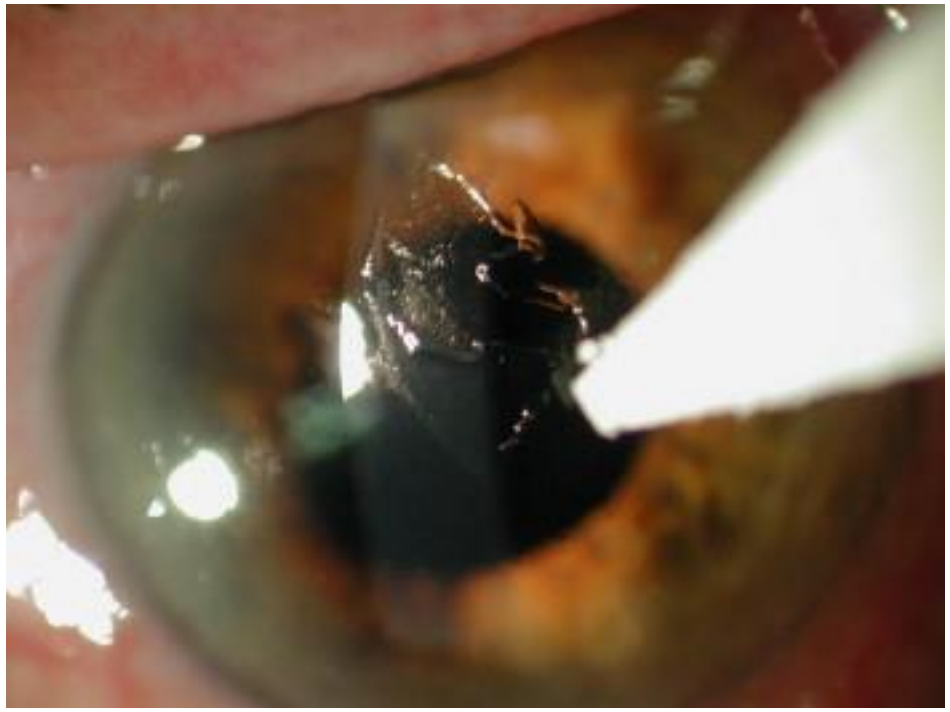
Surgical Interventions

- Stromal puncture, PTK

Corneal Epithelial Debridement

What I do:

- Soften epithelium
  - Topical anesthetic- 3-4 drops over course of 2 minutes
- Weccell sponge to debride a larger radius than defect
  - Sponge, forceps, CTA, spud
- Pull flaps straight and tight
  - Not up and out
  - Treatment rate ~ 80 %



Via ScienceDirect

Gold Standard debridement

- Diamond Burr polishing
- Long term corneal haze
- Reduce with topical steroid

Amniotic Membranes

- Innermost lining of the placenta (amnion)
- Regenerative platform that possess natural growth factors with scaffolding properties within a complex extracellular matrix that has:
  - Anti-inflammatory
  - Anti angiogenic
  - Antimicrobial
  - Anti scarring
- Promotes:
  - Stem cell expansion
  - Pain suppression
  - Cellular migration
  - Expedient recovery

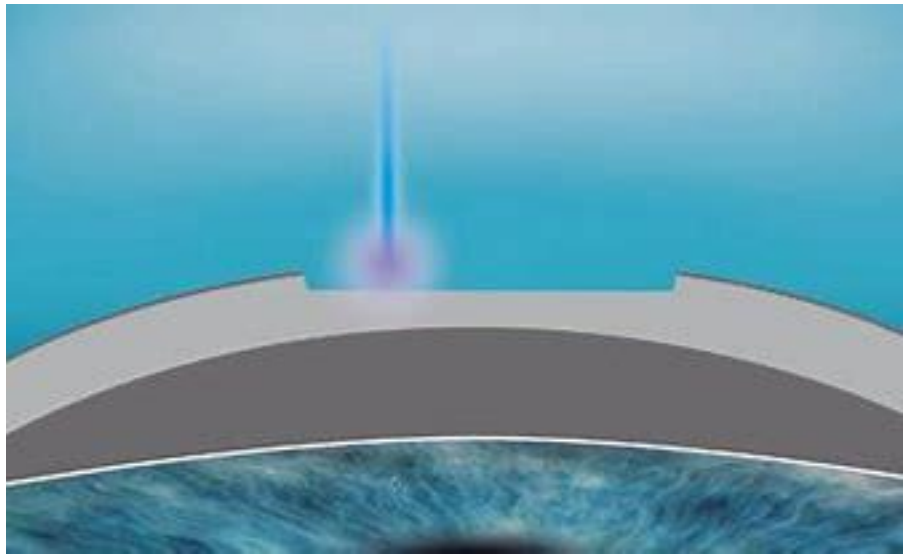
Amniotic Membranes

- Biotissue
  - Prokera, Prokera Slim, Prokera clear, Amniograft, Amnio Guard
- IOP ophthalmics
- Acellular membrane
  - ACELLFX, Apollo

- Follow up 3 day and 5 day

PTK

- Focal or large diameter
- Epithelium removed or transepithelial
- Option if > 1 episode a month



RCE our patient

- 3 day follow up
- More discomfort
- Developed sterile infiltrates
  - BCL too tight
- Left in place and rxd pred forte QID
- 5 day follow up
- Membrane completely integrated and infiltrates resolved

Case: 34 year old, male, landscaper

CC: “My eye’s been killing me since something hit it two days ago.”

History

- Hit in the eye when working outside
- Initially “felt scratched,” used OTC redness drops
- Went to urgent care yesterday: given oflaxicin QID
- Today: Worse pain, more light sensitivity, vision blurrier
- No contact lenses, no prior eye surgery

Symptoms

- Deep, aching pain
- Photophobia
- Tearing
- Feels “swollen” but not particularly itchy

- VA: 20/60 OD, 20/20 OS
- Conjunctival injection
- Cornea (OD):
  - Paracentral infiltrate
  - Feathery borders, irregular margins
  - Early satellite lesions around main infiltrate
  - Epithelial defect smaller than infiltrate footprint
- AC: 1–2+ cell; tiny hypopyon possible
- IOP: Normal
- No obvious foreign body remaining

Fungal Keratitis

- In tropical / subtropical regions, fungal keratitis can be 20–60% of all microbial keratitis
- In the U.S., estimates range from 5–20% of infectious keratitis, higher in southern states
- Trauma with vegetative matter is the single strongest predictor for filamentous fungi (esp. Fusarium, Aspergillus)
- CL wear is a major risk factor for yeast keratitis (Candida) and trauma-independent cases.

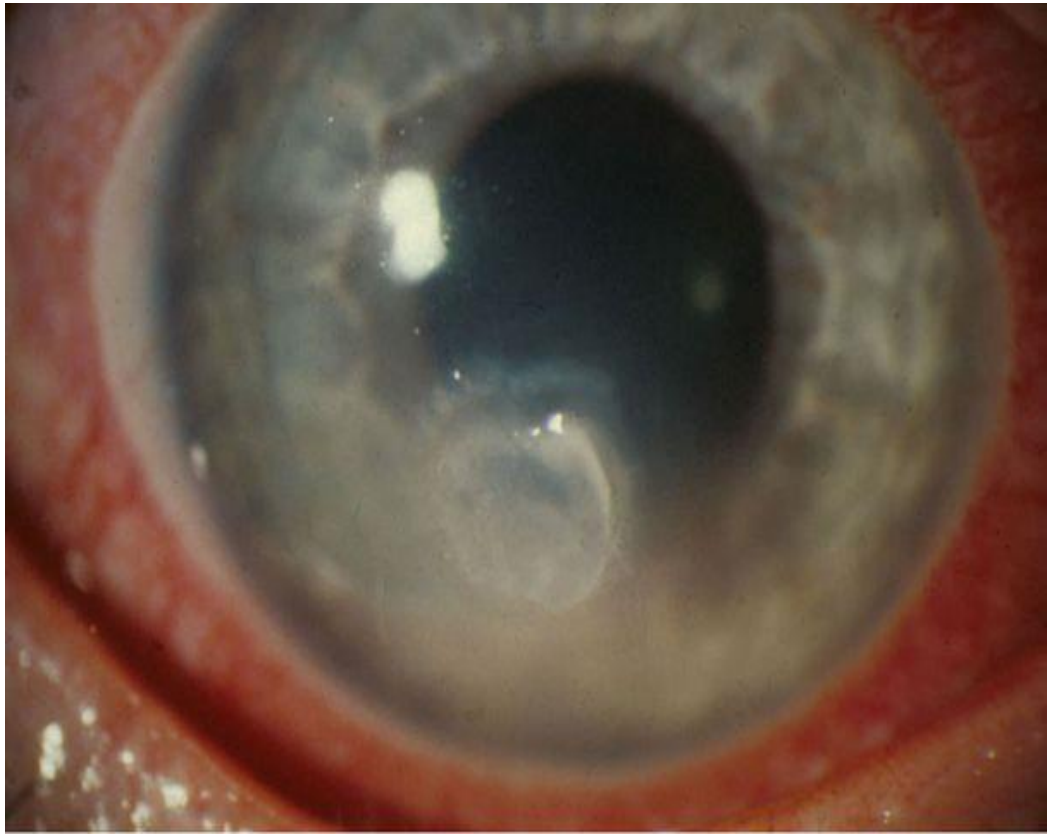
Fungal Keratitis- Testing

- Gram stain (may be negative or non-specific)
- **KOH / Calcofluor white stain:**
  - Fast, sensitive (often >80–90% for detection of hyphae).
- Cultures: **Sabouraud dextrose agar (gold standard for fungi)**
- Blood/chocolate for co-infection
- In Vivo Confocal Microscopy (IVCM) (where available)
  - Sensitivity for detecting fungal filaments often >85–90%.
  - Shows hyperreflective, branching, filamentous structures in stroma.

Sabouraud Dextrose Agar (SDA)

FK Key Features

- **Feathery, fluffy, or “cottony” margins**
  - Classic for filamentous fungi (Fusarium, Aspergillus).
  - Bacterial ulcers tend to have more well-defined, dense borders.
- **Satellite lesions**
  - Small, secondary infiltrates around a main lesion.
  - Strong association with fungal keratitis; reported in **~70%** of Fusarium cases.
- **Epithelial defect smaller than stromal infiltrate**
  - Bacteria usually produce a defect that matches or exceeds infiltrate extent.
  - Fungi can travel in stroma under relatively intact epithelium.



Fungal Keratitis Key Features

- **Trauma with organic matter**
  - Plant, soil, tree branches = classic antecedent for filamentous fungi.
  - In many series, **>70%** of filamentous fungal ulcers follow vegetative trauma.
- **Pain vs appearance mismatch**
  - Pain can be **less** than you'd expect from the severity (due to early nerve damage).
  - Bacterial ulcers often produce "angrier" symptoms.
- **Slower onset**
  - Bacterial ulcers: 24–48 hours explosive.
  - Fungal: often smoldering, worsens over days.

DJO Digital Journal of Ophthalmology
www.djo.harvard.edu

Fungal Treatment

- Natamycin 5%
  - First-line therapy for filamentous fungal keratitis, particularly Fusarium.
- Dosage:
  - Q1H for first 48–72 hours
  - Then taper to Q2H while awake as improvement begins
  - Total course often 4–6 weeks or more.
- MUTT (Mycotic Ulcer Treatment Trial):
  - Natamycin superior to topical voriconazole 1% for Fusarium ulcers:
    - Better visual acuity outcomes
    - Lower risk of perforation
    - Lower need for PKP

Mycotic Ulcer Treatment Trial I (MUTT I)

ClinicalTrials.gov ID NCT00996736

Sponsor University of California, San Francisco

Information provided by Thomas M. Lietman, University of California, San Francisco (Responsible Party)

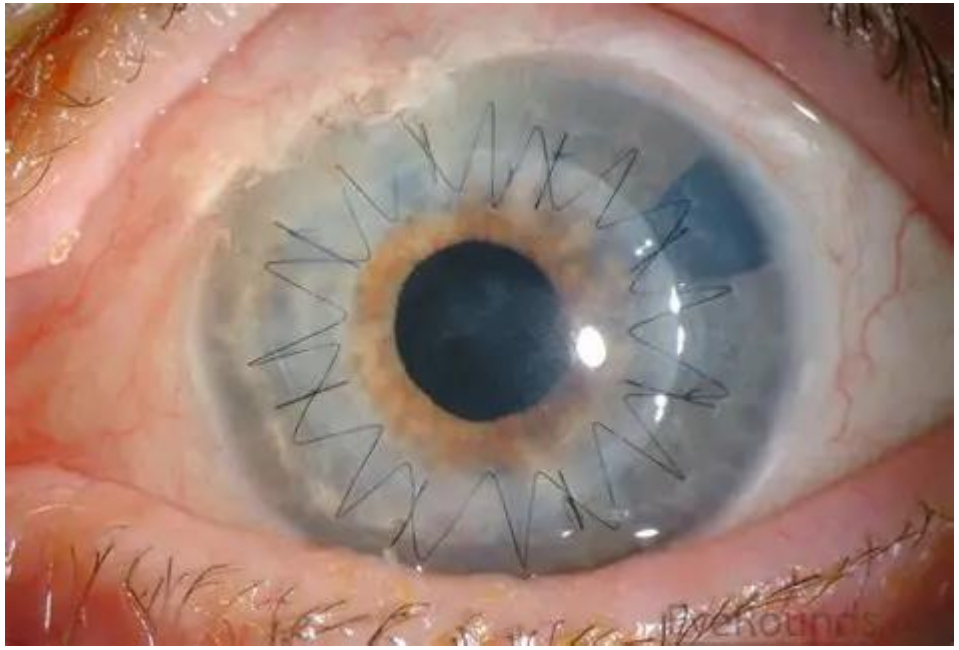
Last Update Posted 2018-08-01

Fungal Treatment

- VORICONAZOLE
  - Better activity against some yeasts and non-Fusarium species.
  - Can be used as:
    - Topical 1% Q1–2H (where available)
    - Oral 400mg BID for 24 hours then 200mh BID
- Adjunctive Therapies
  - Cycloplegic (atropine or cyclopentolate)
- No steroids initially
  - NEVER start steroids until:
    - Organism identified and under control
- Consider CXL (Corneal Cross-Linking) — emerging evidence
  - Riboflavin + UVA :microbial killing + collagen strengthening.
  - Studies show improved outcomes in refractory fungal infections, but data is still evolving.

Fungal Keratitis - Surgical Tx

- If progressive despite maximal medical therapy:
  - Therapeutic **penetrating keratoplasty** (TPK)
    - Goal is to remove infected tissue before perforation
    - Perforation risk in severe fungal ulcers can reach **20–30%.**
    - Adjunct with cyclosporin to prevent graft rejection
    - 80% of grafts remained clear of infection at follow up



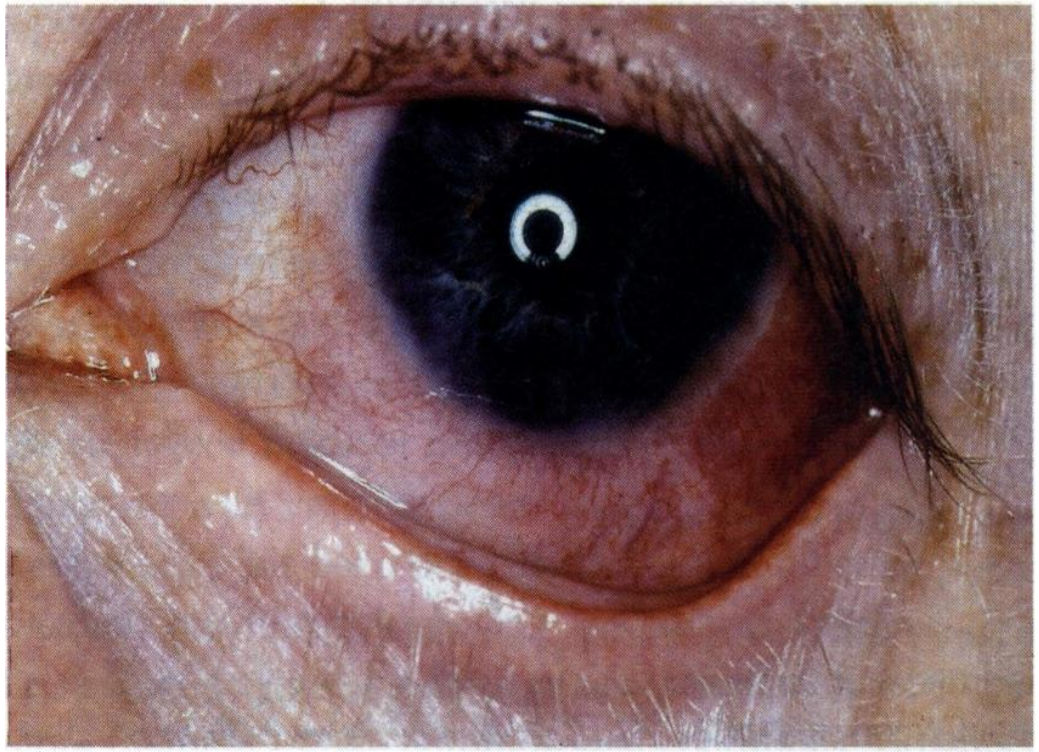
Case: 81 yo, male

- **CC:** “My eye is very red and hurts.”
  Symptoms:
  - No discharge
  - Pain wakes him up at night
  - Feels like pressure behind the eye
  - Mild photophobia
  - No trauma
  - No CL wear

Case

Exam:

- Sectoral violaceous injection
- Pain when pressing globe
- Pain with eye movements
- Clear cornea
- No AC reaction
- No mucous discharge
- No tenderness of eyelid margin
- Partial blanching with 2.5% phenylephrine



Scleritis

- Pain is Key
  - Scleritis pain = deep, boring, often radiates to brow, temple, ear
  - Worsens with eye movements
  - Wakes the patient at night
  - Episcleritis pain = mild irritation, NEVER wakes them
- Color
  - Episcleritis = bright red / salmon pink
  - Scleritis = blue-violet hue (deep vascular congestion)
    Caused by engorged deep episcleral plexus.

Scleritis

- Pheylephrine Blanching (helpful but not diagnostic)
  - Blanching = episcleritis
  - Partial blanching = 70% likelihood scleritis
  - No blanch = scleritis
- Sensitivity ≈ 80%

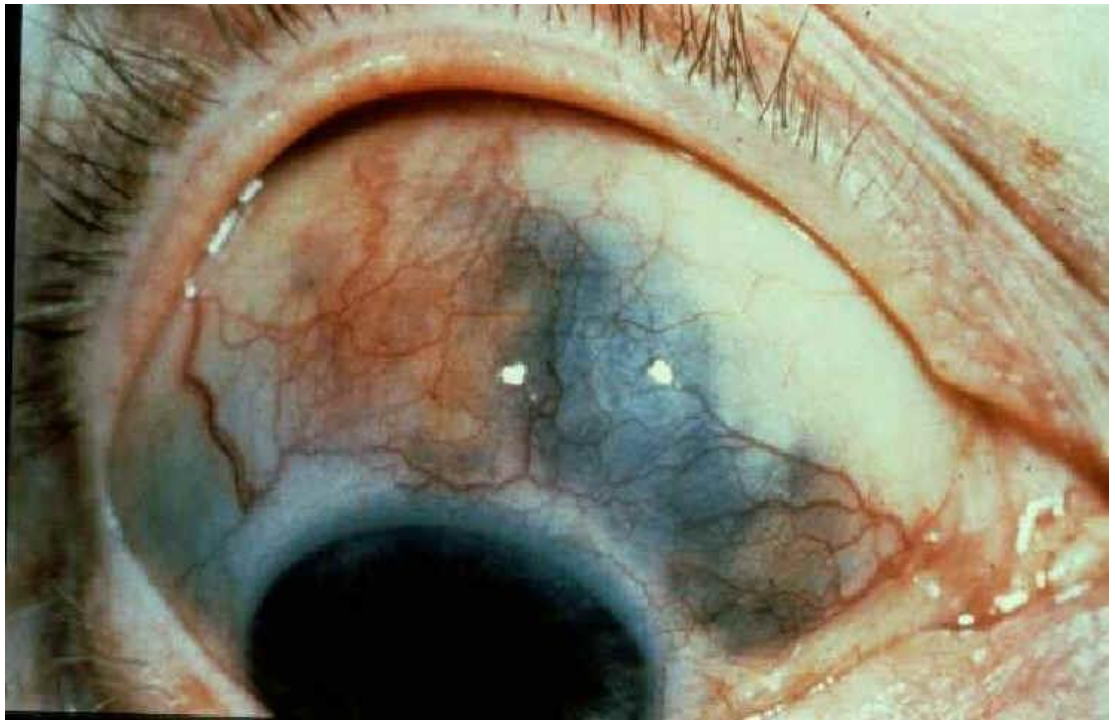
  You CANNOT rely solely on this test.

Scleritis

1. **Systemic symptoms often precede eye symptoms**
    1. Ask about:
        1. Morning stiffness
        2. Joint pain
        3. Diarrhea/abdominal cramps (IBD)
        4. Chronic sinusitis (GPA risk)
        5. Skin rashes (psoriasis, lupus)

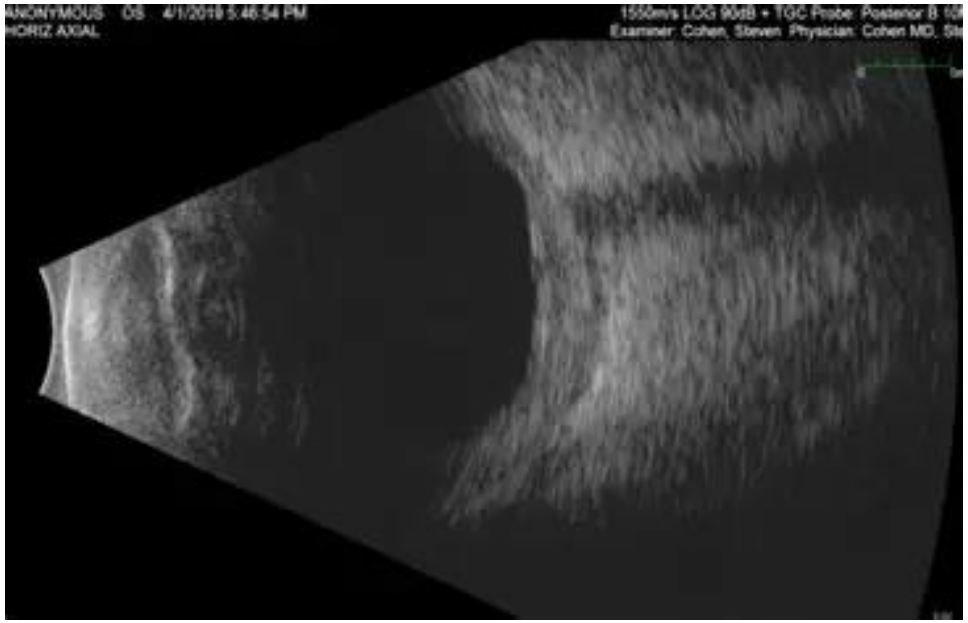
Scleritis

- Necrotizing Scleritis Clues
  - Scleral thinning
  - Bluish uveal show
  - Severe, lancinating pain
  - Often bilateral
  - Possibly associated with GPA or RA
- Necrotizing scleritis has ~74% risk of vision loss if untreated.



Posterior Scleritis

- Posterior Scleritis Clues Pain worse on eye movement
  - Proptosis
  - Choroidal folds
  - Macular edema
  - Optic disc edema
  - Ultrasonography: “T-sign” (fluid in Tenon’s space)
  - Posterior scleritis misdiagnosis rate = 50–70%



Systemic Correlations

- Up to 50% of scleritis cases are tied to systemic disease.
- Top Associations
  - Rheumatoid arthritis (18–33%)
  - Granulomatosis with polyangiitis – GPA (15–20%)
  - Inflammatory bowel disease – Crohn's/UC (4–10%)
  - Psoriasis/Psoriatic arthritis (3–9%)
  - Systemic lupus erythematosus (2–6%)
  - Relapsing polychondritis (up to 30% have ocular involvement)

Systemic Correlations

- Infectious (4–10%):
  - HSV
  - VZV
  - Syphilis
  - TB
  - Lyme
  - Pseudomonas (especially post-surgical)

Scleritis- Work up

- Every patient with suspected scleritis should get:
  - Autoimmune Labs (High Yield):
    - ANA
    - RF
    - Anti-CCP (RA-specific, very useful)
    - ESR + CRP
    - ANCA (c-ANCA = GPA hallmark)
    - HLA-B27 (if uveitis history)
  - Infectious Panel:
    - Syphilis: RPR / VDRL + FTA-ABS
    - Lyme: ELISA
    - TB: QuantiFERON Gold
    - HSV / VZV PCR if keratitis suspicion
    - CBC, CMP baseline

Scleritis Treatment

- First-Line (for NON-necrotizing, non-infectious)
  - Oral NSAIDs:
    - Indomethacin 50 mg TID (most effective)
    - Naproxen 500 mg BID
    - Ibuprofen 600–800 mg TID
  - Success rate: 37–50% with NSAIDs alone.

- If no improvement in 3–5 days: Oral Steroids
  - Prednisone 40–60 mg daily
  - Taper over 4–6 weeks
    Success rate: 70–80%.

Scleritis Treatment

- If STILL no improvement :Immunosuppressants
  - Methotrexate 15–25 mg weekly
  - Azathioprine 1–2 mg/kg
  - Mycophenolate mofetil
  - Cyclophosphamide (necrotizing disease)
  - Biologics
    - Infliximab
    - Rituximab
  - Rituximab success in refractory scleritis: 72–80%

Scleritis Treatment

- If infectious: do NOT use steroids
- Culture-guided antibiotics
  - TB → 4-drug therapy
  - Syphilis → IV penicillin
  - HSV → Acyclovir/Valacyclovir
  - VZV → Antivirals + steroids only after viral control

Scleritis Referral

Urgent Rheumatology Referral:

- ANY necrotizing scleritis
- Positive ANCA
- Recurrent disease
- Bilateral
- Posterior scleritis
- Treatment failure after NSAIDs + steroids

Urgent Ocular Specialist Referral:

- Scleral thinning
- Vision-threatening inflammation
- Posterior scleritis
- Suspected infectious scleritis

Emergency Referral:

- Severe pain + bluish sclera
- Proptosis
- Decreased VA
- Suspicion of orbital extension

Case 53 yo female

- CC: “The left side of my head and eye really hurts.”
- History
  - Onset: 2–3 days of burning, stabbing pain on the left side
  - New light sensitivity and tearing OS
  - Tried OTC artificial tears that lead to no real improvement
  - Past medical: mild HTN, possibly prediabetes; no known immunosuppression

Case

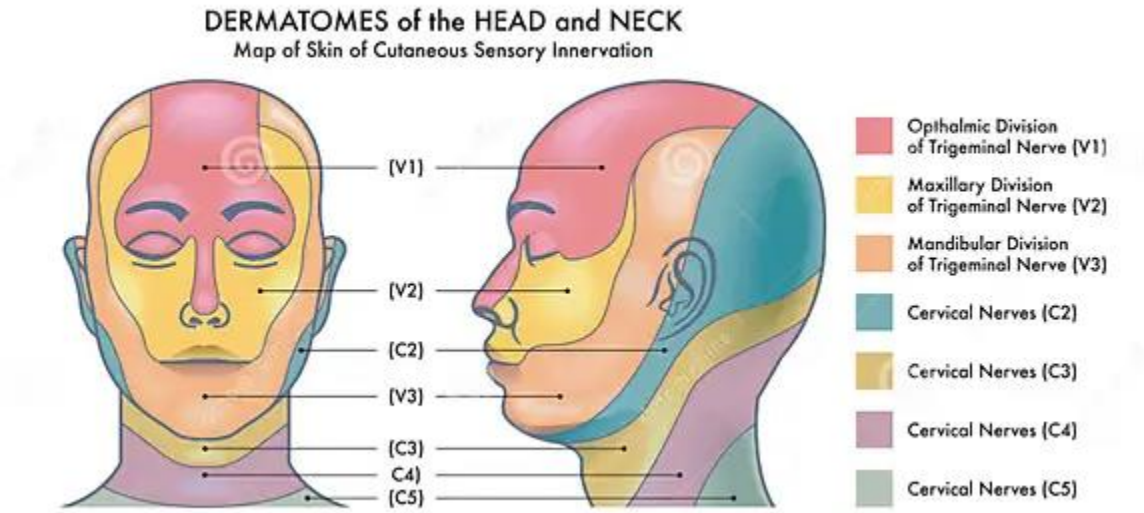
- Symptoms
  - Pain in forehead
  - Mild redness, no discharge
  - Denies double vision or vision loss
- Exam
  - VA: 20/20 OU
  - Lids: maybe subtle erythema
  - Conj: mild injection
  - Cornea: trace SPK or normal
  - AC: quiet
  - IOP: normal
  - Fundus: normal

Herpes Zoster Opthalmicus

- Lifetime risk of herpes zoster: **20–30%**, rising to **>50%** by age 85.
- HZO represents **10–20%** of all zoster cases.
- Peak incidence: adults >50 years, but increasing in younger adults with immunosuppression.
- Risk factors:
    - Age > 50
    - Immunocompromised (HIV, chemo, steroids, biologics)
    - Autoimmune disease
    - Cancer
    - Psychological stress, trauma (less robust data but often reported)

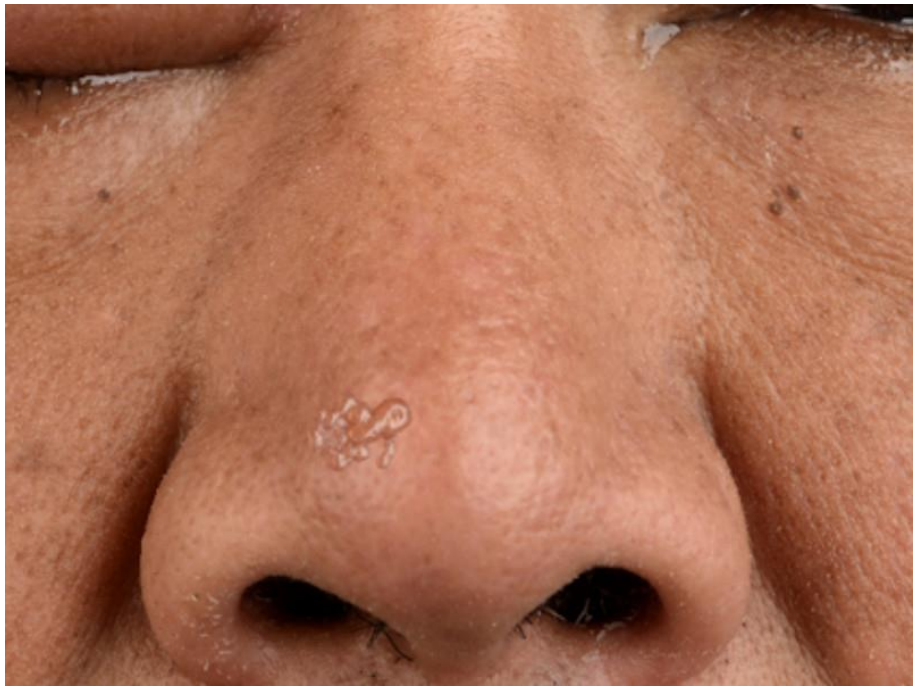
HZO

- Dermatomal pain PRECEDES rash in 60–70% of patients
- Burning, shooting, or stabbing pain in V1 distribution
- Neurocutaneous clue: hyperesthesia/allodynia
  - Light touch (even air, hair, or glasses frame) causes disproportionate discomfort.
  - This is a hallmark of zoster neuropathic pain, not simple sinus pain or tension headache.



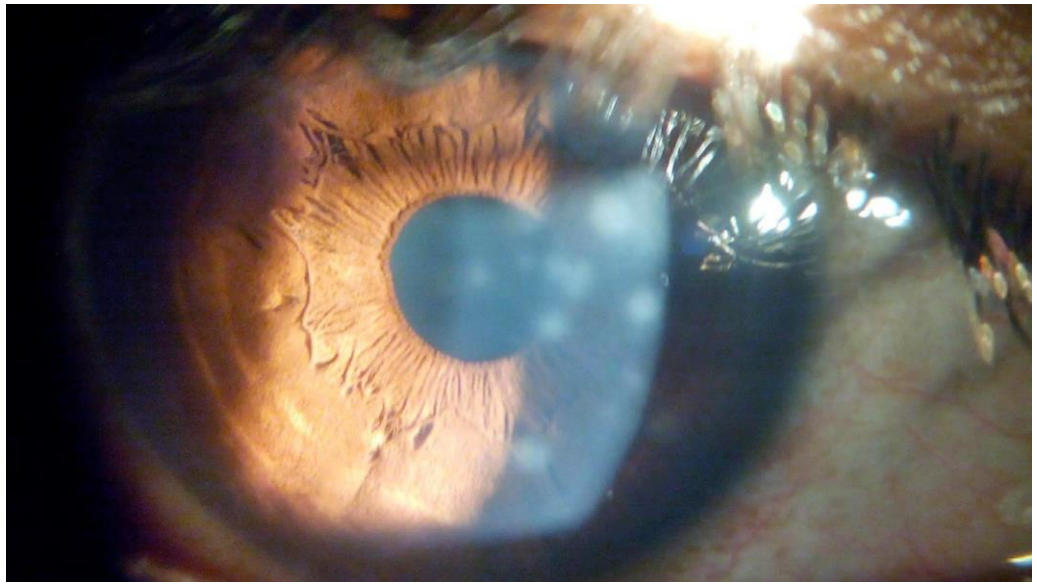
HZO

- Rash is NOT required to start antivirals
- By the time classic vesicles appear, viral replication is already underway.
- Hutchinson’s sign
  - Indicates Naso ciliary nerve involvement → higher chance of ocular involvement.
  - absence of Hutchinson’s sign does not rule out serious eye disease.
- Pain out of proportion to ocular findings
  - Mild SPK + mild redness + intense V1 pain should scream HZO in your mind



HZO Ocular Manifestations

- Lids:
  - vesicles, ulceration, scarring, cicatricial changes
- Conjunctiva:
  - follicular conjunctivitis, pseudomembranes
- Cornea:
  - Epithelial keratitis (pseudodendrites)
  - Stromal keratitis (nummular, interstitial)
  - Neurotrophic keratopathy
- Uvea:
  - anterior uveitis (often with elevated IOP)
- Sclera:
  - scleritis/episcleritis
- Retina:
  - acute retinal necrosis (rare but devastating)
- Nerves:
  - cranial nerve palsies (III, IV, VI), optic neuritis



Uveitis

- Uveitis occurs in ~40–60% of documented HZO cases.
- Most appears within 1–2 weeks of rash onset but uveitis can appear before vesicles, with rash, or weeks–months later.
- Frequently granulomatous
- Often hypertensive (30–40%)
- Can occur before, during, or after rash
- Recurs in 20–30%

HZO Treatment

- For immunocompetent adults:
  - Valacyclovir 1,000 mg TID x 7–10 days
  - Famciclovir 500 mg TID x 7–10 days
  - Acyclovir 800 mg 5×/day x 7–10 days
  - Valacyclovir tends to be preferred due to TID dosing and similar efficacy.
- Timing:
  - Best if started within 72 hours of rash onset.
- BENEFITS
  - Shortens duration of acute rash and pain
  - Reduces risk and severity of PHN (post-herpetic neuralgia)
  - Lowers incidence of ocular complications (keratitis, uveitis)
  - Some studies suggest antiviral therapy reduces PHN risk by ~50% if initiated early.

HZO Treatment

- Epithelial keratitis
  - Lubrication
  - Zirgan 5 x a day- potential
- Stromal keratitis or uveitis:
  - Topical corticosteroids (e.g., Pred Forte QID)
  - WITH concurrent systemic antivirals
  - Cycloplegia (e.g., homatropine BID)
- Neurotrophic keratopathy:
  - Aggressive lubrication
  - Bandage CL or scleral lens
  - AMT (amniotic membrane transplant) if persistent epithelial defect
  - Oxervate (cenegermin) where appropriate
- IOP elevation:
  - Aqueous suppressants (beta-blockers, CAIs)
  - Avoid prostaglandins in active inflammation

Post herpetic neuralgia

- NSAIDs for mild pain
- Gabapentin or pregabalin for neuropathic pain
  - 300mg PO qhs
- Sometimes TCAs (e.g., nortriptyline) in coordination with PCP
  - 10–25 mg qHS → titrate to 50–75 mg as tolerated
- Goal: reduce central sensitization and long-term PHN.
  - PHN (pain >3 months after healed rash) risk:
  - Increases with age (up to 50% in >60–70 years)
  - More severe, prolonged acute pain = higher risk

Post Herpetic Neuralgia

- The Acyclovir Study Group (NEJM)
  - Acyclovir within 72 hours of rash onset:
- Topical Lidocaine Patch
- Capsaicin 8%
- Nerve Block
- Experimental Tx
  - Neuromodulation
  - Botox (Botulinum toxin A)
  - Duloxetine
    - Strong evidence in diabetic neuropathy
  - Low-Level Light Therapy (LLLT)
    - Early studies suggest nerve-regenerative effects

Long Term Antiviral Suppression

- ZEDS
  - 2025 update
  - 12 months of low dose valacyclovir reduced new or worsening eye disease by 26%

HZO

- **Shingrix** (recombinant zoster vaccine) reduces herpes zoster risk by **>90%** in adults ≥50.
- Also significantly reduces HZO incidence and PHN.
- You can recommend patients over 50 talk to PCP about vaccination—especially after an HZO episode (once acute disease resolves).

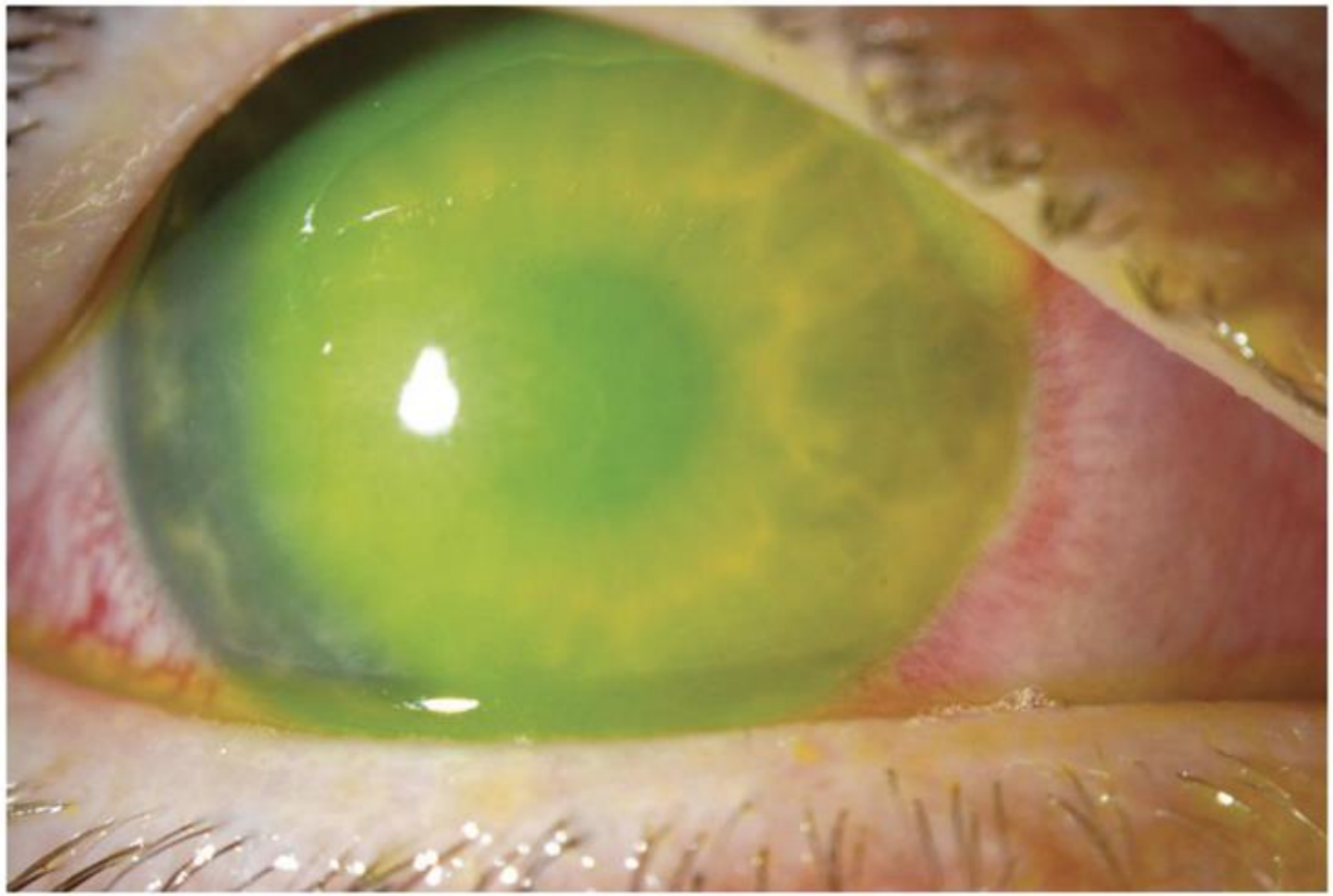
Case: 37yo white female

- Patient was in such extreme pain she was crying in waiting room and couldn't walk so a wheelchair was utilized
- CC: extreme pain, light sensitivity, redness, and burning after putting contacts 2 hours prior
- Patient reported that 1 hour after inserting contacts, they fell out and the pain started
- Patient was camping with friends
- No past history of ocular conditions
- Flexeril

Case: PT SK

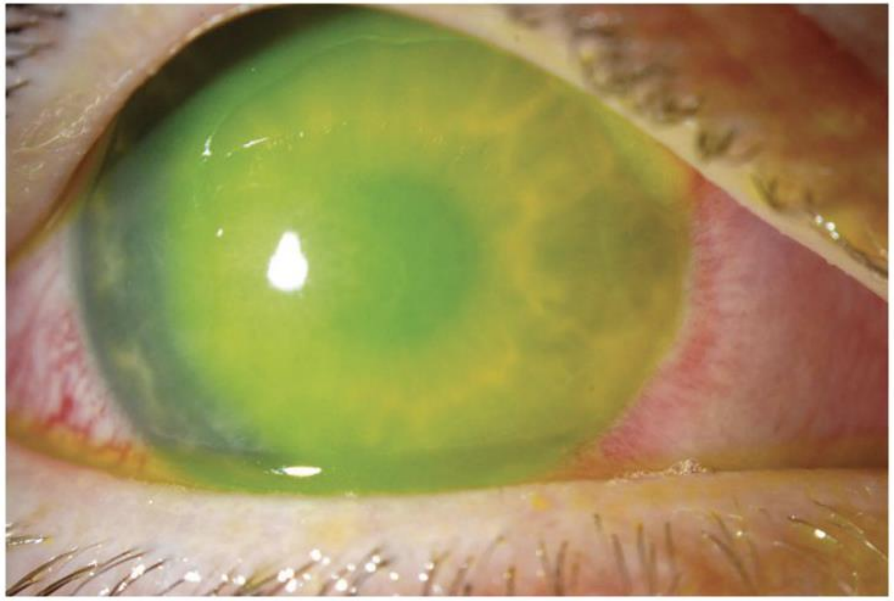
- VA: 20/70 OD,OS no improvement with pinhole
- Patient refused iop due to pain and reported pain on EOM on all gazes
- Pupils: PERRLA
- Instilled proparacaine OU so we could examine eyes

Case: PT SK



Case: PT SK

- PH tested tear film with Hydrion strips: 9!
- Immediately irrigated with sterile saline over the course of 10 minutes until pH was 7
- Debrided necrotic tissue with wescot sponge
- Instilled 1% atropine in office
- Rxd Vigamox Q2H, Pred Forte BID, ATs Q1H
- INstilled AO N&D bandage CL
- Tylenol #3 1 pill PO q12hrs
- RTC 1 day and requested she bring in her CL case



Chemical Burns

- Acidic burns: tend to be less severe
  - Most commonly from sulfuric acid
    - Hydrochloric acid, nitric acid, pigments and dyes, explosives
- Alkali Burns
  - Concerning
  - Severity correlated to duration of exposure and pH of solution
  - Common agents:
    - Lyes, ammonia, hair products, non phosphate detergents, dishwasher soaps, disk batteries

Chemical Burns

- 61% are industrial accidents, 37% home based
- Most common cause of alkali injury of the eye is assault
- May take 72 hours following injury to assess damage to ocular structures
- Prognosis depends on
  - Total area of corneal defect
  - Number of clock hours or degrees of limbal blanching
  - Area and density of corneal opacity
  - Evidence of increased IOP
  - Loss of lens clarity

Chemical Burns

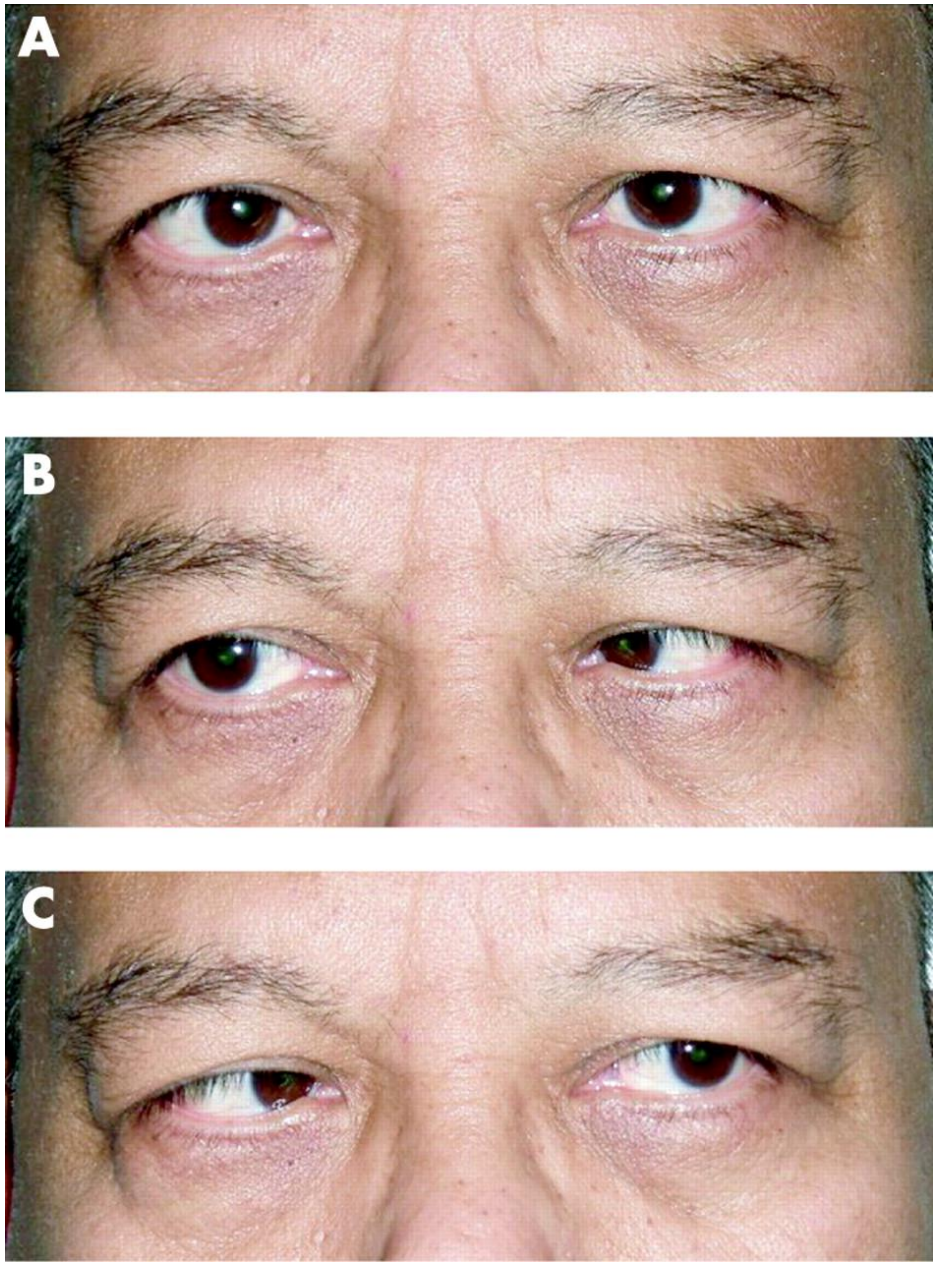
- Treatment:
  - Copious irrigation with isotonic saline or lactate ringer
  - Goals: reestablish maintenance of intact and corneal epithelium and control balance between collagen synthesis and collagenolysis, and minimize sequela
  - Acute phase:
    - Broad spectrum antibiotic
    - Cycloplegia
    - Promote epithelialization with bandage SCL
      - Retinoic acid? fibronectin?
    - Corticosteroids to reduce inflammation
    - Glaucoma meds
    - Ascorbate- reduced incident of corneal ulceration- 2g/day or 10% solution
    - If injured eye not reepithelialized by 21 days may use tissue adhesives, PK, or amniotic membranes

PT SK

- PT returned next day with CL case
- Smelled of ammonia
  - Suspected someone she was camping with put ammonia in her contact lens case
  - CLs acted as a depot for the ammonia
  - When CLs fell out they took sloughed epithelium with them
- Slowly tapered Pred forte and vigamox over course of three weeks, supplemented with ATS Q1H
- 3 week follow up patient was 20/20 BCVA with no corneal scarring

Case 46 year old male

CC: “Double vision and a terrible headache.”



Case

History:

- Onset: 3 days of worsening retro-orbital headache, now constant and severe
- New horizontal diplopia that’s worse at distance
- Reports numbness of forehead
- Mild fever
- Recent history: “Really bad sinus infection” 1–2 weeks ago

Case

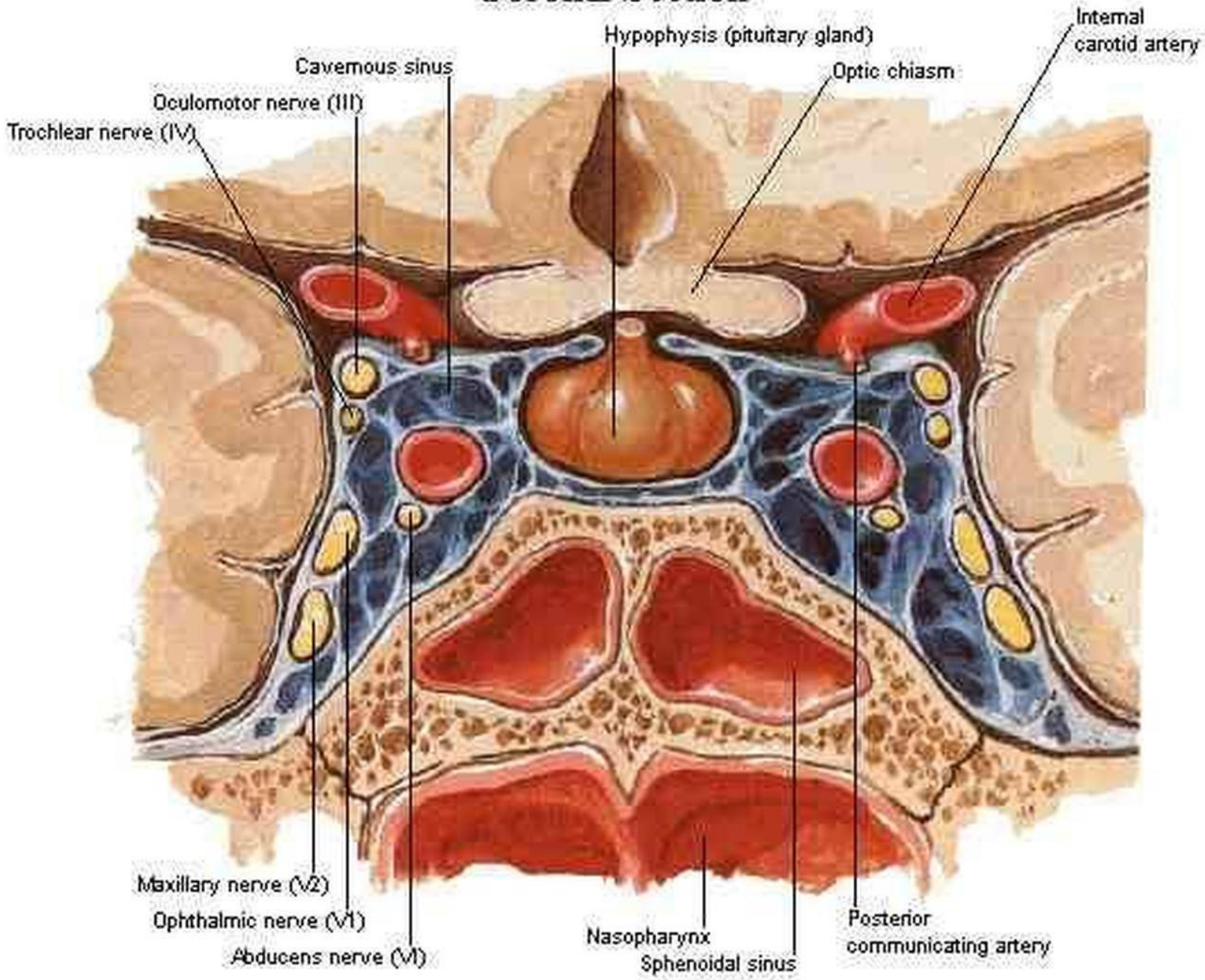
- VA: 20/25 OU
- Pupils: trace anisocoria, sluggish response on affected side
- EOMs: Limited abduction
- Mild ptosis
- Decreased V1 sensation (forehead / upper lid)
- Cornea: clear
- AC: quiet
- Fundus: normal
- Mild proptosis and chemosis on the affected side

Cavernous Sinus Thrombosis

- Cavernous sinus thrombosis (CST) used to have a **20–30% mortality**, now still around **10–15%** even with modern management, and significant morbidity (cranial nerve deficits, vision loss, stroke) in up to **30–50%** of survivors.

Cavernous Sinus

Coronal Section



CST Red Flags

- **Multiple cranial nerves involved on the same side**
  - Microvascular CN VI palsy: isolated, painless, usually in older diabetic patient
  - Cavernous sinus: often **III + IV + VI + V1**, sometimes V2
- **Pain that's severe and persistent**
  - Painful ophthalmoplegia is a hallmark
  - Worsening over days, not brief spikes
- **Fever or systemic signs**
  - Especially if there's recent sinusitis, facial cellulitis, or dental infection
- **Proptosis and chemosis**
  - Venous congestion from impaired cavernous sinus/outflow via superior ophthalmic vein
- **Bilateral progression**
  - Cavernous sinus on both sides communicates → disease often becomes bilateral

Etiology

- **Septic Cavernous Sinus Thrombosis:**
  - Most commonly from:
    - **Paranasal sinusitis** (esp. sphenoid, ethmoid)
    - Facial infections (furuncles, cellulitis)
    - Dental abscess
    - Orbital cellulitis
  - **Microbiology:**
    - Staph aureus (including MRSA) → ~60–70%
    - Streptococci
    - Anaerobes
    - Gram-negative rods occasionally

Etiology

- **Aseptic/Non-infectious Cavernous Sinus Syndromes:**
  - Tolosa–Hunt syndrome (idiopathic granulomatous inflammation; painful ophthalmoplegia responsive to steroids)
  - Carotid-cavernous fistula
  - Metastatic tumors
  - Pituitary apoplexy with lateral extension
  - Thrombophilia-related CST (prothrombin gene mutation, factor V Leiden, etc.)

Work up

- Immediate action
  - **Check vitals** (especially fever, BP, HR)
  - Quick basic neuro screen:
    - CN III, IV, VI motility
    - V1 (forehead) + V2 (cheek) sensation
    - Pupils (anisocoria? sluggish reaction?)
    - Corneal sensation
  - If pattern fits: **no “monitoring” period**.

    This is now an **emergent imaging case.**

Work up

- **Emergent Imaging (ER)**
  - They should obtain:
    - **MRI brain + orbits with contrast**
    - **MRV (MR venography)** → to evaluate venous thrombosis
    - CT of paranasal sinuses (if not already done)
    - MRI/MRV is more sensitive than CT for early CST and inflammatory lesions.
  - CBC with differential (look for leukocytosis)
  - Blood cultures
  - ESR
  - CRP
  - Coagulation panel
  - Hypercoagulable workup
  - Systemic infectious workup (if septic CST suspected)

CST Treatment

- For Septic Cavernous Sinus Thrombosis:
  - IV Broad-Spectrum Antibiotics (aggressive, high dose)
    - Vancomycin (MRSA coverage)
    - 3rd/4th gen cephalosporin (e.g., ceftriaxone, cefepime)
    - ± Metronidazole (anaerobic coverage)
  - Anticoagulation (controversial, but increasingly used)
    - Heparin or LMWH
  - Surgical drainage of:
    - Paranasal sinus infection and Orbital abscess if present
  - Steroids
    - Only considered after adequate coverage and source control

CST Treatment

- Non-infectious Cavernous Sinus Syndromes
  - Diagnostic criteria include:
  - Unilateral pain
  - Cranial nerve III, IV, and/or VI palsies
  - MRI evidence of cavernous sinus inflammation
  - Dramatic response to steroids within 48–72 hours
  - Treatment:
    - High-dose oral or IV steroids
    - Very close imaging + neuro follow-up

Case: PT JF 9 yo white male

- CC: pain in left eye for a few days
- Described a pinching feeling in front and behind of the eye
- Foster mother reported what looked like a bug bite on lid
- Patient revealed severe headache with same onset
- No past ocular or medical history
- No medication

Case: PT JF

- VAs sc 20/50 OD, 20/80 OS, ph 20/20 OD, 20/30 OS
- IOP: OD 14, OS 17
- PERRLA
- EOM testing showed pain upon abduction OS
- Ishihara: OD 14/14. OS 4/14
- SLE:
  - Swollen adnexa, erythema, tenderness on left side of face extending from brow to lower cheek
  - 2+ chemosis
  - Trace proptosis
- Fundus Photos


[redacted]
2012
Eye Care Clinic, Cheyenne, WY
OPTOS, P200DTx
Laterality: R
Red: 50%
Green: 50%
Zoom: 4.91
Presentation: Multiple
1/2
4


[redacted]
Eye Care Clinic, Cheyenne, WY
OPTOS, P200DTx
Laterality: L
Red: 50%
Green: 50%
Zoom: 4.91
Presentation: Multiple
1/1
4

Differentiating: Preseptal vs Orbital Cellulitis

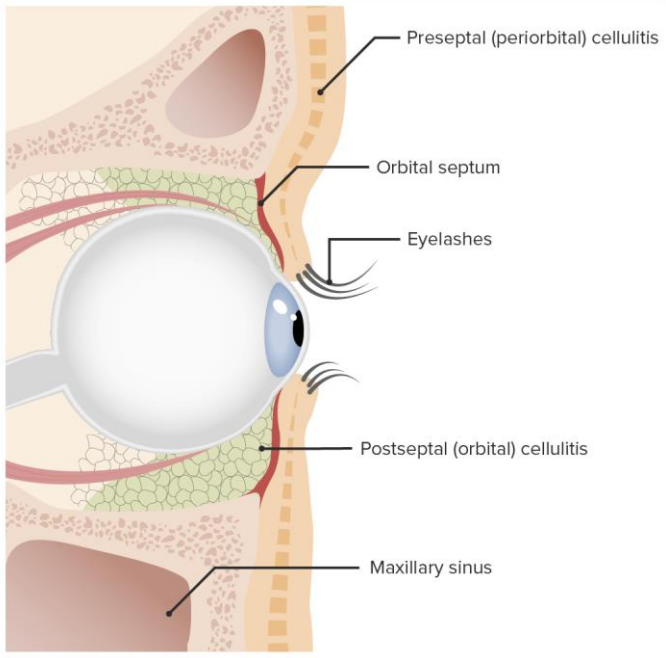
FINDING	ORBITAL	PRESEPTAL
Visual Acuity	Decreased	Normal
Proptosis	Marked	Absent
Chemosis and Hyperemia	Marked	Rare/Mild
Pupils	RAPD	Normal
Pain and Motility	Restricted and Painful	Normal
IOP		Normal
Temperature	102 - 104	Normal/mild elevation
HA and Assoc. Symptoms	Common	Absent

Preseptal Cellulitis

- Infection and inflammation anterior to orbital septum that is limited to superficial tissue

- Typically following hordeolum or sinus infection

Rit Radiology



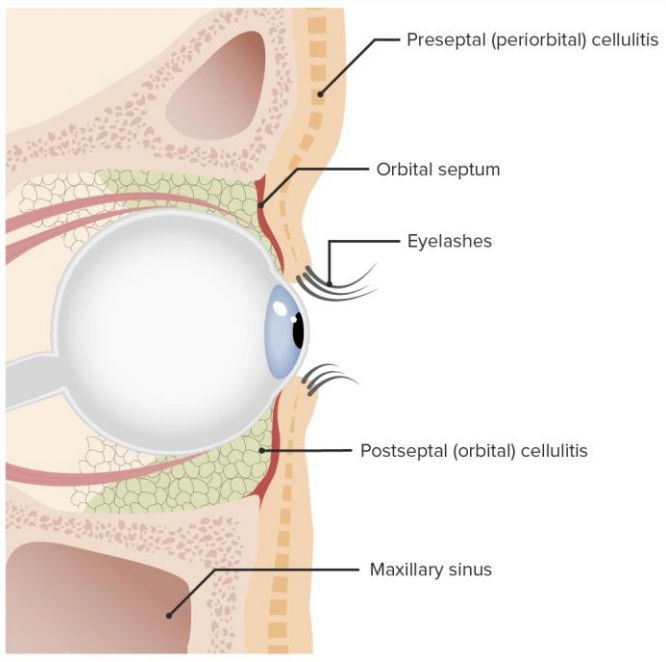
Poll Question

If it was determined that our 9 year old patient had preseptal cellulitis that was secondary to a MRSA infection, how would you treat?

a) Levaquin (levofloxacin)
b) Keflex (cephalexin)
c) Bactrim (trimethoprim-sulfamethoxazole)
d) Augmentin (amoxicillin- Clavulanic acid)

Preseptal Cellulitis

- Treatment:
  - Keflex 500mg BID-TID x 5-7 days
  - Augmentin 500 mg BID-TID x 5-7 days
  - If possible MRSA infection consider Bactrim/ Septra



Augmentin

- Amoxicillin + potassium clavulanate
  - Clavulanate is a B-lactamase inhibitor which inactivates penicillinase
- Adult dosing - take with food and 2 hours later take a probiotic
  - 250-500 mg tablets q8- q12 hours
  - Or 875 mg q 12 hours
- Pediatrics
  - 45-90mg/kg/day q 12 hours
  - Less than 3 months: 30mg/kg/day
- Effective for skin infections such as dacryocystitis, internal hordeolum, preseptal cellulitis, otitis media, sinusitis, URI's
- PCN Allergy?

Keflex (cephalexin)

- 1st generation cephalosporin
- Closely related structurally and functionally to penicillins
  - More resistance to B-lactamases
  - Cross sensitivity was stated to be ~10% closer to 1%
- Adult Dosing
  - 500 mg BID to TID
- Pediatric Dosing
  - 25-100mg.kg/day divided in 8 hours
- Effective against most gram positive pathogens and great for skin and soft tissue infections, GI, otitis media

MRSA

- Healthcare associated (HA-MRSA) is severe, invasive disease in hospitalized patients
  - Risk factors: antibiotics, prolonged hospital stay, intensive care, invasive devices, proximity to others with MRSA
- Community associated (CA-MRSA) most commonly in soft tissue infections in young healthy patients with no hospitalization.
  - Risk factors: skin trauma (lacerations, tattoos, abrasions), body shaving, HIV, shared equipment

MRSA

- Treatment
  - Clindamycin 300- 450 mg q6-8 hours
  - Bactrim (trimethoprim-sulfamethoxazole)
    - 80mg/400mg QID
    - Double Strength
      - 160/800 BID
    - Broader spectrum of action compared to sulfa medications alone
    - Effective in treating UTIs, URI's, and skin infections
    - Never Rx in a pt taking methotrexate- folic acid inhibitors- can cause bone marrow suppression!!
  - Doxycycline
    - 100mg BID

Orbital Cellulitis

- Infection and inflammation within the orbital cavity
- Most commonly secondary to ethmoid sinusitis or direct trauma
- Reduced VA, pain, HA, diplopia, bulging eye, APD, EOM restriction, lid swelling fever, ON compression, leukocytosis in 75% of cases, and lethargy

Orbital Cellulitis

- Causative agents: Staph. Aureus, Strep pyogenes, H. influenza (peds)
- Recent dental work? Diabetes?
  - Mucormycosis

Orbital Cellulitis

- Refer for CT scan of orbits and paranasal sinuses, axial and coronal with contrast
  - MRI helpful if concomitant cavernous sinus thrombosis is suspected
- CBC
- Blood culture- helpful in pediatric patients
- Neurology consult if suspected meningitis
- Consider ENT consult if paranasal sinusitis is present

Orbital Cellulitis

- Treatment:
  - Broad spectrum IV antibiotics for 48- 72 hours followed by oral for at least 1 week
  - Given the high prevalence of CA-MRSA:
    - Vancomycin 15 mg/kg every 12 hours for adults
    - Vancomycin 40 mg/kg/day in children
    - Plus (in combination with) one of the following
      - Ampicillin-sulbactam 3g IV every 6 hours
      - Ceftriaxone 2 g IV every 24 hrs
      - Cefotaxime 2 g every 4 hours
    - After 72 hours oral antibiotics
      - Clindamycin, Bactrim, Augmentin, Cefpodoxime
  - Surgery in cases of progression of orbital signs with antibiotic treatment
    - Concurrent Subperiosteal or orbital abscess 10% of cases

Case: PT JF

- Referred for immediate CT of the orbit, CBC, and blood culture at local hospital
- ER determined severe orbital cellulitis and patient was transferred to Denver's children and remained inpatient for 1 week and treated with IV antibiotics and oral for 2 weeks following
- 1 month follow up
    - All signs and symptoms completely resolved
    - BCVA 20/20

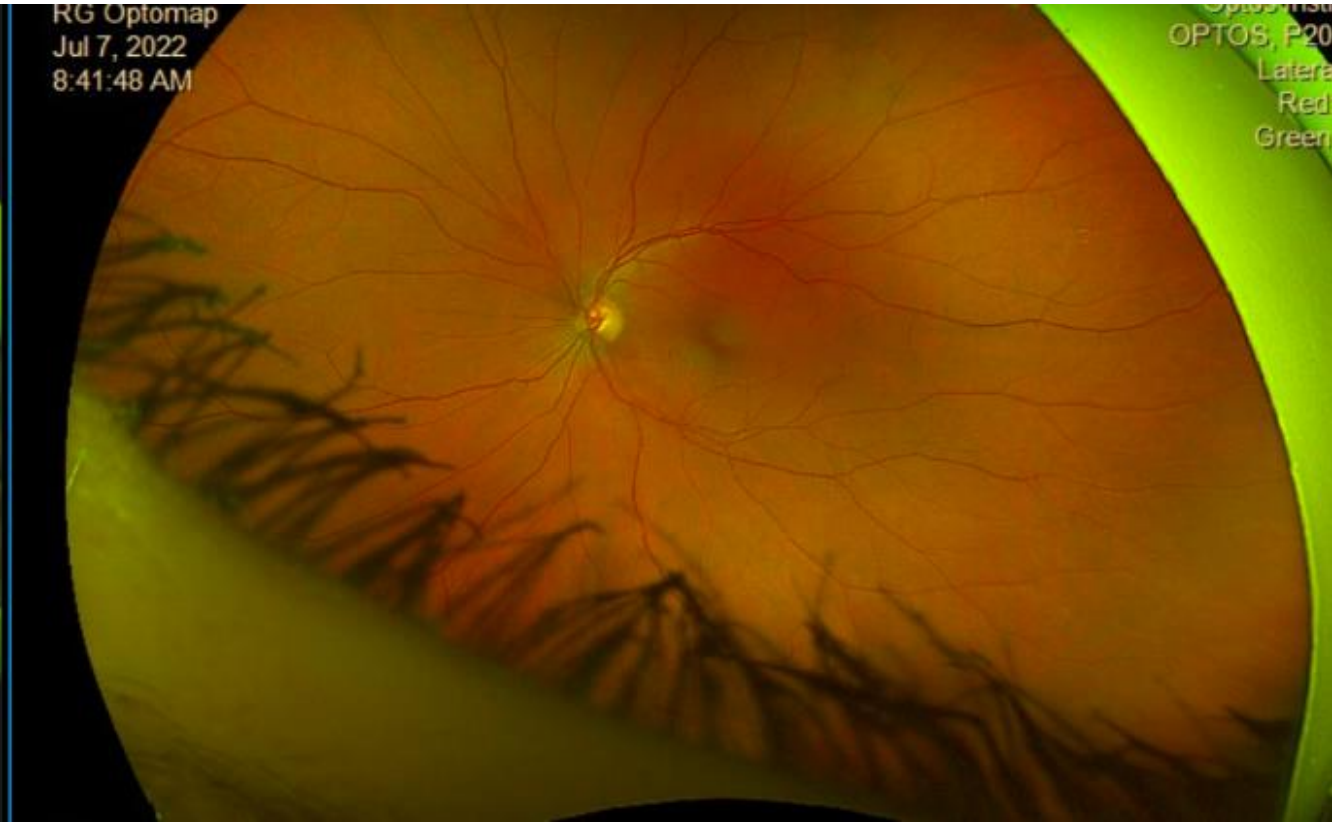
Case: PT MH

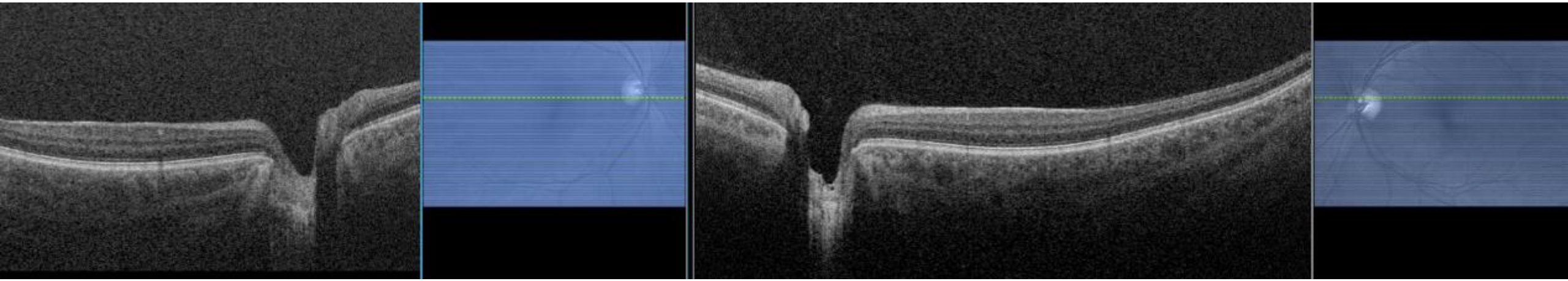
- 46yo White Female
- CC: double vision and headaches
- Started a month ago and has gradually gotten worse- worsens with fatigue
- No systemic or ocular health history
- Taking Mucinex

Case PT MH

- VA: OD 20/70 ph 20/40, OS 20/40 ph 20/20
- IOP: 15/13
- BP: 100/76
- PERRLA
- EOM:
  - OD: restriction on up gaze leading to double vision on anything superior to primary gaze
- VF: clear
- SLE: unremarkable


halvorson, michelle
DOB: Jun 29, 1976
RG Optomap
Jul 7, 2022
8:40:16 AM
Laterality: R
Red: 50%
Green: 50%


RG Optomap
Jul 7, 2022
8:41:48 AM



Case: PT MH

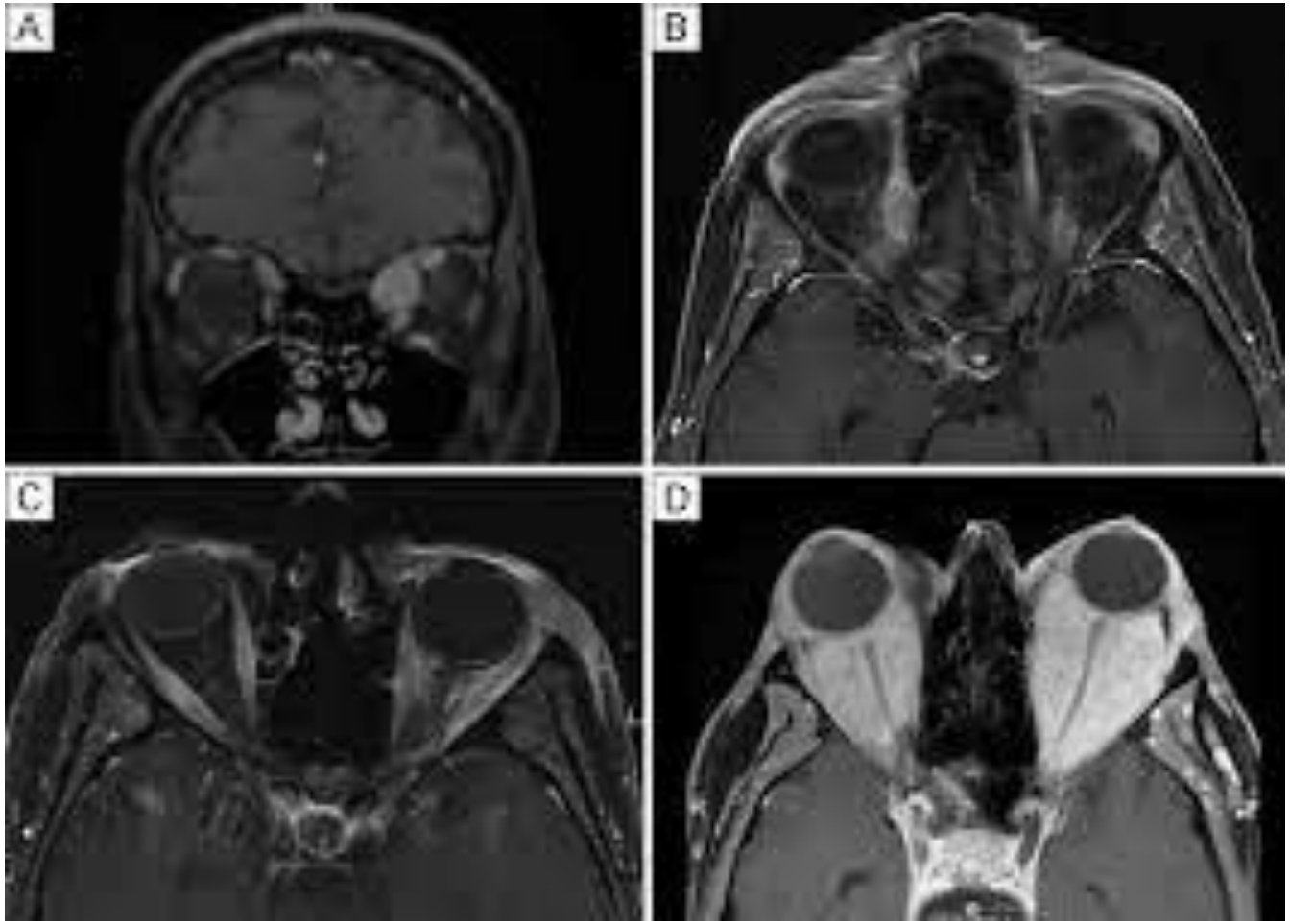
- DDx Diplopia/ CN3 palsy- considering MG or TED
- Sent order for labs
- Referred to Neurology for MRI of brain and orbits and further testing
  - Saw her and diagnosed a partial 3rd nerve palsy and scheduled her for an MRI
- About a month went by and I haven't received any follow up notes or heard from anyone so I called to check in
  - She reported double vision was worse so I had her come in.
  - No one had called her about the results of her labs or MRI

Case: PT MH

- PTs eye was drastically more hypertropic with a measurable proptosis
- We found her lab results after calling the lab and all levels were normal
- Called the neurologist and he agreed this seemed like TED so he scheduled her for MRI of the orbits and a follow up with endocrinology

Shiley Eye Institute

Case: PT MH



Thyroid Eye Disease

- Autoimmune condition in which bodies immune system attacks and causes inflammation to tissues surrounding the eye including the muscles, fat, and connective tissue
- The active inflammatory phase can last from 3 months to 3 year years followed by the stable phase when the inflammation has stabilized
- Typically associated with Graves disease
  - Graves disease typically leads to hyperthyroidism but can cause hypothyroidism as well
  - In rare cases TED is can happen in cases with normal thyroid levels (<3%)

Thyroid Eye Disease

- More common in women
- 16/100000 women, 2.9/100000 men
- Median age of diagnosis is 43
- The eyes are particularly vulnerable to Graves due to proteins that are similar to the immune system of the thyroid gland (thyrotropin receptor on orbital fibroblasts and IGF-1R)
- Smoking greatly increases risk

Thyroid Eye Disease

- Signs and symptoms:
  - redness, pain, bulging, dry eye, increased pressure, double vision headache, decreased vision
- As the EOMs are attacked they lose the ability to stretch resulting in proptosis and restriction
- When the optic nerve becomes compresses irreversible loss of vision can occur
- In rare cases glaucoma can occur

Thyroid Eye Disease

- Treatment
  - Smoking Cessation
  - ATs, lid taping, bcl
  - Selenium and vitamin D supplements?
  - Aloe vera juice?- hypothyroidism
  - Oral prednisone vs IV Methylprednisolone
  - Orbital decompression
  - Radiation
  - Tepezza
- Our patient was started on Tepezza

Thyroid Eye Disease

- Tepezza (2020)
  - Infusion- can be done as a home infusion
  - Inhibits IGF-1R
    - Prevents activation of orbital fibroblasts leading to a reduction in inflammation, preventing muscle and fat tissue remodeling, and preventing tissue expansion

- Eliminated diplopia in 2x more patients than placebo (67% maintained)

Thyroid Eye Disease

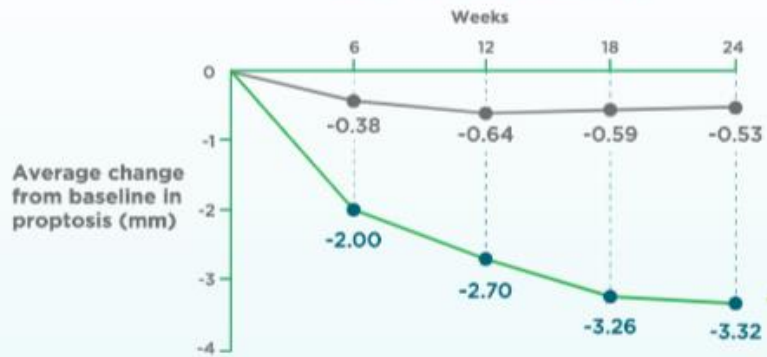
- Tepezza
  - Side Effects?
    - Muscle spasms
    - Nasueua
    - Hair loss
    - Abdominal pain
    - Hyperglycemia
    - Hearing loss- reversable?
    - Taste changes
    - Headaches

Proptosis responders (patients achieving a ≥2 mm reduction in proptosis)[1,2*] (Study 2)

Significantly greater proptosis response rate **TEPEZZA 83%** vs placebo **10%** (Week 24)

***P*<0.01 at Week 24**
TEPEZZA (n=41)
placebo (n=42)

Average change from baseline in proptosis over 24 weeks[1] (Study 2)



TEPEZZA significantly decreased proptosis **-3.3 mm** vs placebo **-0.5 mm** (Week 24)

Thyroid Eye Disease

- Tocilizumab
  - IV

- Rituximab
- Sub Q IGF IR antibodies 0pipeline