

Ocular Urgencies and Emergencies



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Financial Disclosures

Lindsey Bull	Jessilin Quint
<ul style="list-style-type: none"> Abbvie-Consultant, Speaker Viartis – Consultant Lenz Therapeutics- Consultant, Speaker 	<ul style="list-style-type: none"> Alcon-Consultant, Speaker Abbvie-Consultant, Speaker Bausch & Lomb-Consultant, Speaker Dompe-Consultant Tarsus-Consultant Ocuphire-Consultant Viartis-Consultant Eyenovia-Consultant Orasis-Consultant Lenz Therapeutics- Consultant, Speaker

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What classifies an urgency or emergency?



- Ocular complaints
- Vision complaints
- Systemic complaints

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Optometrists & Emergencies

- How many people visit urgent care/ER for ocular problems?
- Optometrists are best suited to handle eye emergencies
 - ❖ Urban/suburban setting
 - ❖ Rural setting
 - ❖ Going to urgent care vs optometrist
 - ❖ Integrated health care model

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Office protocols of urgencies and emergencies

- Triage training
 - Same day/asap appointments
 - Within 24 hours
 - At earliest convenience
 - At a future date
- Document, Document, Document
- Importance

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Taking call as an optometrist

- Required by state?
- Required by insurance panels?
- Value to the patient



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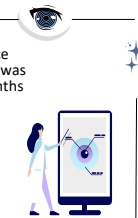
Let's get to some cases!



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34YOM patient **without** medical insurance presents in office with complaint that he was hit in the eye with a piece of metal 2 months ago

Reports that he went to the ER immediately and was told that there was a "possible scratch." Sent home with instructions to take ibuprofen and use erythromycin QID.

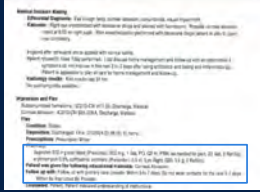


Case #1

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Complete care =


1. Obtaining previous medical records.
2. Important for establishing baseline!



Medical History
Chief Complaint: Eye injury and vision loss since trauma to the right eye.
History of Present Illness: The patient reports a traumatic injury to the right eye while working on a construction site. The injury occurred approximately 2 weeks ago. The patient describes a sharp pain and a sensation of something in the eye. Following the injury, the patient noticed a significant decrease in vision and experienced frequent headaches. The patient has been unable to return to work and is seeking medical attention.
Review of Systems: No other significant findings.
Physical Examination: Visual acuity: 20/40 in the right eye (OS) and 20/20 in the left eye (OD). Pupils: Right pupil is 4 mm, round, and reactive to light and accommodation. Left pupil is 3 mm, round, and reactive. Intraocular pressure (IOP): 18 mmHg in the right eye and 14 mmHg in the left eye. Anterior chamber: No cells or flare. Lens: No significant findings. Posterior segment: No significant findings.
Diagnosis: Traumatic eye injury, suspected intraocular hemorrhage.
Plan: Monitor vision and IOP. Refer to ophthalmology for further evaluation and management.

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Case #1



• Patient complains that this eye has been painful and light sensitive since the suspected injury.

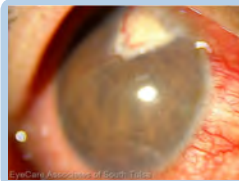
1. Reports significant vision loss
2. Flashes that started 2 weeks ago
3. (+) Headache

"Dr. Bull, can you help me test pupils? I cannot find his right pupil."

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I think we might have a problem...

So what are we thinking now?



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Exam Findings

Anterior segment

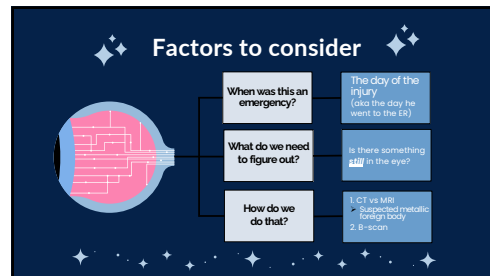
- UCVA OD LP (barely)
- IOP OD 3mmHg with iCare
- 3+ hyperemia
- Corneal scar with iris incarceration
- Corneal neovascularization
- 1+ AC cell

Posterior segment

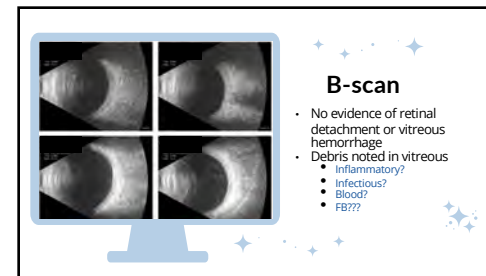
... Good luck ...



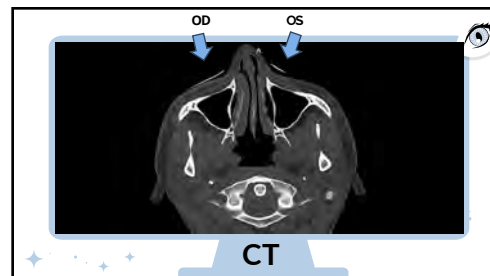
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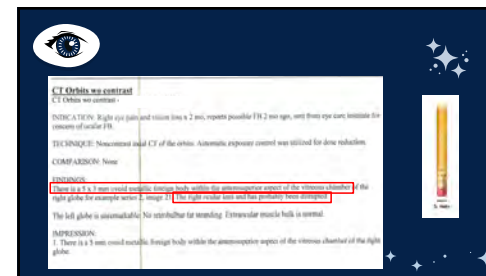
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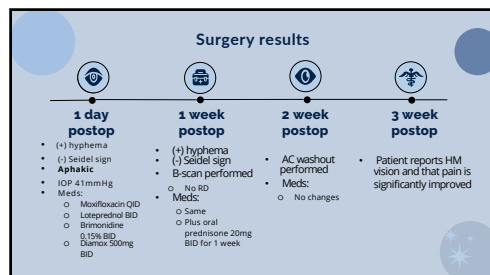
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56 year old male with complaints of his right eye hurting

- Began 1-2 week ago
- Thinks he scratched eye after removing contact lenses

(+) pain 2/10 severity
(+) photophobia
(+) blurry vision
(+) watering

Case #2



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History

- Medical history:** Anxiety, Depression, HTN, neuropathy (feet)
- Medications:** lisinopril, Effexor, Xanax
- Allergies:** NKDA
- Ocular history:** unremarkable
LEE 5 years ago, Monthly MF contact lenses
- Social history:** 1-2 drinks/week, non-smoker

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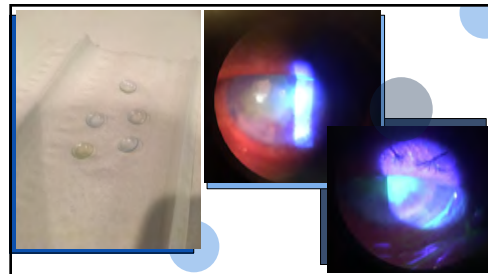
Entrance Testing

- **BCVA:** HM @ 4ft OD NIPH; 20/30 OS
- **Pupils:** PERRLA, (-)APD
- **Confrontational VF:** grossly full OU
- **EOMs:** Full & Smooth OU, (-)nystagmus
- **IOP:** (iCare) 17 mmHG OD, 16 mmHG OS

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Slit Lamp Findings		OD	OS
	Lids & Lashes	Normal	Normal
	Conjunctiva/Sclera	3+ injection	Trace Injection
	Cornea	Contact Lens Diffuse edema Central epi defect Neovascularization (0.5mm I & N)	Contact Lens
	A/C	Hazy View	Deep & Quiet
	Iris	Brown, Grossly normal	Brown, WNL
	Lens	Trace NS	Trace NS

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Posterior Pole Findings		
	OD (Hazy View)	OS
Vitreous	Quiet-no cells	Quiet-no cells
Optic nerve	Pink, healthy rim 0.3/0.3 C/D ratio	Pink, healthy rim 0.3/0.3 C/D ratio
Macula	Flat & clear	Flat & clear
Retina	No breaks/tears	No breaks/tears

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Diagnosis

What's your diagnosis?

1. Corneal abrasion?
2. Neurotrophic Keratitis?
3. Corneal ulcer secondary to CL overwear?




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Sterile vs Infectious Infiltrate	
Sterile	Infectious
Smaller lesion (<1mm)	Larger lesion (>1mm)
Peripheral location	Central location
Minimal epithelial damage	Significant epithelial defect
No mucous discharge	Mucopurulent discharge
Less pain or photophobia	Pain & photophobia
No or minimal A/C reaction	Anterior chamber reaction
No lid involvement	Lid edema, hypopyon

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Infectious Infiltrates



Contact Lens patient = treat as infectious until proven otherwise

- Viral**
- Fungal**
- Bacterial**
Staphylococcus, Streptococcus, and Pseudomonas
-Contact Lenses: *Pseudomonas aeruginosa*
- *Staphylococcus aureus*
- Protozoan**
Acanthamoeba

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Non-infectious infiltrates

01	02	03	04
Marginal corneal infiltrates	Contact lens-induced acute red eye (CLARE)	Contact lens-induced peripheral ulcer (CLPU)	Infiltrative keratitis

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Culturing

When to culture:

- Large, central ulcer
- Unresponsive to treatment
- Post-surgical, monocular, or immunocompromised
- 3-2-1 Guideline: 3mm size, 2+ ulcers, 1mm visual axis

"Quick culture" = sterile swab placed in prepared (thioglycolate) broth and sent to lab to be placed on nutrient plates

Best to perform culture before initiating treatment

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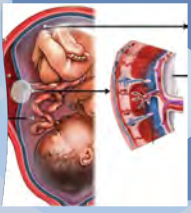
Treatment

1. Antibiotic
 - Fluoroquinolones: Gram - & +
 - Aminoglycosides: Gram -
 - Polymixin-B: Gram -
 - Other: erythromycin (G+, some G-), bacitracin (G+), azithromycin (G + & -)
2. Steroid
3. Amniotic Membranes

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Amniotic Membranes

- Derived from placentas
- Amniotic membrane (AM)=inner layer of the fetus membranes
- AM contains: structural proteins, specialized proteins, cytokines, growth factors
- MOA poorly understood
- Faster healing, less pain, less scarring, less inflammation
- Ocular history




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Amniotic membrane therapeutic benefits

01. Anti-inflammatory Suppresses pro-inflammatory cytokines	02. Anti-microbial 1. Produces anti-microbial cytokines 2. Serves as physical barrier on the wound surface	03. Anti-scarring 1. Reduce protease activity 2. Prevents adhesion of injured surfaces to each other
04. Analgesic Effective covering of exposed nerve endings	05. Anti-angiogenic Prevents formation of new blood vessels	06. Promotes re-epithelialization Growth factors assist in cell proliferation and differentiation


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Types of amniotic membranes




Dehydrated

- Dehydrated by using a low-temp vacuum process to remove moisture
- Shelf stable
- Must use BCL or shield to hold in place
- Thought that dehydrating may impact structural integrity
- Lower cost



Cryopreserved

- Tissue is sterilized and frozen
- Must be stored in freezer (-112° to 39 2p°)
- Plastic ring vs collagen shield/BCL
- GyroTek vs SteriTek
- High level of structural integrity
- Higher cost




Lyophilized

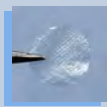
- Type of "dehydrated" membrane
- 350 micron thickness
- Dehydrated through a process similar to "freeze drying"
- Must use BCL to hold in place
- Possibly preserves the membrane's structure better than standard dehydration
- Lower cost


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Types of amniotic membranes





Dehydrated






Cryopreserved





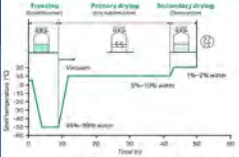
Lyophilized



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Lyophilization

- Low temperature
- Minimizes impact on proteins



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Corneal sensitivity testing



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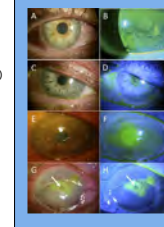
Neurotrophic keratitis (NK)

- "Stain without Pain" → significant discrepancy between clinical findings & symptoms
- Degenerative corneal condition due to reduced neural innervation
- Risk Factors: infection, injury, or inflammation
→ trauma, tumors, inflammatory lesions, herpetic infections, chronic corneal exposure, surgical, damage to trigeminal nerve
- Disease progression often unnoticed by the patient

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Neurotrophic Keratitis Stages

- Stages I – III
 - Stage 1= General epithelial alterations (A & B)
 - Stage 2= Persistent epithelial defects (C & D)
 - Stage 3= Corneal ulceration (E & F)
- Perforation (G & H)



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NK treatment options



- Amniotic Membranes
- Topical Insulin (1UL/ml)
- Cenergem-bkbj (0.02%)- Oxervate
- Matrix Therapy Agent
- Surgical Management

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Oxervate (cenegermin-bkbj)

- FDA approved in 2018
- cenegermin-bkbj is structurally identical to human Nerve Growth Factor protein made in ocular tissue
- It is a recombinant nerve growth factor (protein) → this protein activates receptors that allow for differentiation & maintenance of neurons that support the innervation of the cornea
- Dosing: 6x/day (2hr Intervals) for 8 weeks
- Apply 1-1.5 ul if using ung/gel after
- Wait 15 minutes for CL insertion
- Can do another round if needed

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Case 2: 56YOM corneal ulcer

Treatment

1. 0.3% ciprofloxacin q30min
2. 1% cycloplegic in office
3. Prokera Slim Amniotic Membrane
4. RTC 1 day




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Follow-up

1 day	4 day	6 day	10 day
<ul style="list-style-type: none"> - Prokera Slim 80% dissolved - Replaced with new Prokera - Continue topical antibiotic q30min - RTC 1 day 	<ul style="list-style-type: none"> - 2nd Prokera dissolved - Resolved infiltrate - 2+ SPK cornea - Taper topical antibiotic to qid - Start 1% pred acetate q2hr - Copious PFATs - RTC 2 days 	<ul style="list-style-type: none"> - 1+ SPK - D/C topical antibiotic - Decrease 1% pred acetate qid & increase PFATs - RTC 3 day 	<ul style="list-style-type: none"> - Trace SPK - BCVA 20/25- - IOP stable - Small central epithelial scar

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Case #3



24YOM new patient arrives as an emergency work in with the complaint that he was "cleaning a tire approximately an hour ago and it exploded on him."

- 10/10 pain OU
- Cannot open eyes
- UCVA OD 20/50 OS 20/100
- IOP ??

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What kind of injury are we looking for?

How do we rank these in order of what we need to manage/rule out first?

Foreign body
Chemical injury
Abrasion

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Concern #1 : Chemical burn

What chemical was being used?
Ask for specific name/photo of the product

Common tire cleaner components	pH
Water	7.0
Ethylene glycol butyl	5.5-8.0
Sodium lauryl ether sulfate	7.5
Ethanol	7.33
C10-16 alcohol	4.9
Sodium C14-16	8.0-10.0
Sodium Xylene Sulfonate	11.96


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Concern #1 : Chemical burn

Start flushing!

- o Ideally have patient begin flush before they leave to come to the office
- o Sterile saline
- o Morgan lens
- o Constant
- o Recommended 15-20 minutes
- o Until pH is 7.0-7.5
- o pH strips in office

After 1st flush patient's pH was WNL



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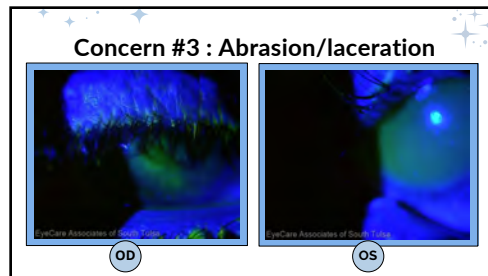
Concern #2 : Foreign body

Tire explosion

01 Location Corneal? Conjunctival? Under the lids? Adnexa?	02 Depth Must rule out penetrating injury! Seidel sign?
03 Material Know your object! Tire made of rubber, steel, and fabric	04 Removal Is this something that can be done in office or is a surgeon required?

Slit lamp exam showed no evidence of foreign bodies and no penetrating injury

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Concern #3 : Abrasion

Anterior segment

- OD: approximately 15+ small, scattered abrasions
- OS: Complete corneal epithelial loss
- 3+ conjunctival hyperemia OU
- AC 1-2+ cell OU

Posterior segment

- Blunt force injury!

MUST DOCUMENT INTERNAL EXAM

Important to note: size and location of injuries-
Due to chemical burn or impact of foreign object?

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How do we treat each eye?

OD

1. Lesser injury
2. Better visual acuity

Small abrasions: 1-3 days

OS

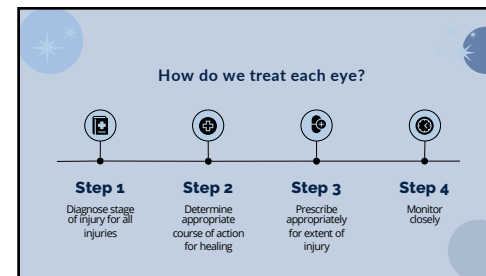
1. Greater injury
2. Poorer visual acuity

Large abrasions: up to 14 days

Should we treat each eye the same or does the severity/extent of the injuries allow us to make individual decisions for each eye?





1. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6122156/pdf/1732879.pdf>

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Stages of ocular chemical burn⁵

Grade 1  Corneal epithelial loss only = good prognosis	Grade 2  Grade 1 + corneal edema and haze, 1/3 of limbus with conjunctival ischemia = some permanent scarring possible
Grade 3  Grades 1 & 2 + significant corneal haziness, less than 1/3 of limbus affected by ischemia = possible visual impairment (prognosis is variable)	Grade 4  Grades 1-3 + cornea is opaque, more than 1/3 of the limbus is affected by ischemia = possibility of perforation (prognosis is poor)

5. Bazzin M, Yusef A, & Ahmed S. An update on chemical eye burns. Eye. 2013; 1362-1377 (2013). <https://doi.org/10.1038/s41433-013-0458-6>

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Treating each eye

OD:

- Bandage contact lens

OS:

- Debridement of loose epithelial tissue performed in office
 - Wick-cell and forceps
- Amniotic membrane placed
 - Hydrated membrane used

BCL vs Amniotic membrane:

- Amniotic membrane thought to decrease risk of RCE⁶ and antimicrobial properties?
- Amniotic membrane = large visual disturbance
- Cost to patient
 - Insurance

6. Frost CL, Smith, Jessica. "My Patient Has Recurrent Corneal Erosion. Now What?" April 2016. Review of Optometry. www.reviewofoptometry.com/article/040616my-patient-has-recurrent-corneal-erosion-what
7. Maheshwari C, Jain RK, Wield J. Transplants. 2014;425:111-112.

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What to prescribe?

Medications:

- Moxifloxacin- QID until follow-up
- Prednisolone- QID until follow-up
- Bromfenac- BID until follow-up
- Atropine- 1gtt instilled in office
 - Decision made to continue dosing in office to cut patient costs
- Ativan (lorazepam)*- Patient given Rx for two 1mg tablets
- Gabapentin**- One 300mg capsule TID for 4 days
- PF tears- prn

Important to note:

- *Lorazepam is a schedule IV medication
- **Gabapentin is a schedule V medication in some states

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Follow-ups

Day 1 post injury

OD:

- UCVA 20/100
- Corneal abrasions significantly improved
- BCL replaced

OS:

- UCVA 20/400
- Amniotic membrane removed for evaluation
- Epithelium beginning to heal with significant 8.5x8.5mm abrasion noted
- Amniotic membrane reinserted in eye

Continue all meds as previously instructed

RTC 3 days- post weekend

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Entrance Testing

- **BCVA:** NLP OD; 20/200 NIPH OS
- **Pupils:** fixed, miotic OD; round, minimal reactivity OS
- **Confrontational VF:** I & T constriction OS
- **FOMs:** Full & Smooth OU

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Slit Lamp Findings

	OD	OS
Cornea	WNL	Edema 3+ guttata inferior KPs
A/C	Quiet irido-corneal touch	4+ cells/3+ flare (-)hypopyon
Iris	Atrophy	I, IT, ST synechiae
Lens	Displaced w/PS	4+ pigment AC
Vitreous	No view	(+)cells-hazy view of post pole

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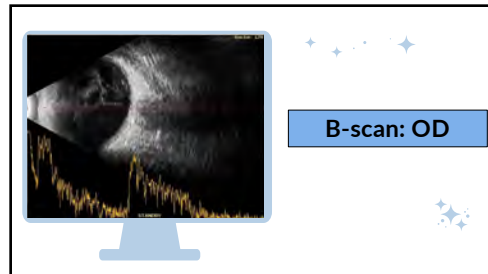
Clinical Exam

- **IOP:** 7 OD/10 OS (mmHG)
- **Gonioscopy:** PAS OS
- **DFF:**
 - OD: no view (dense cataract)
 - OS: photo

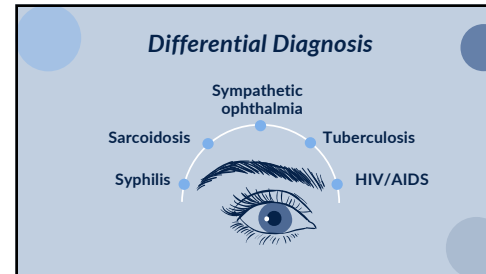
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Case Management

1. 1% PA Q1hr & 1% Atropine BID OS
2. Lab Work-up
3. Uveitis specialist referral

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Diagnostic Testing

- **Labs:**
 - FTA-ABS (inconclusive)

Diagnosis: Syphilitic Panuveitis

- Unremarkable CSF & CXR

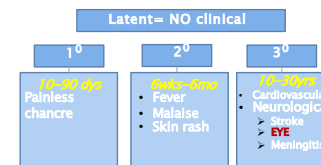
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Treatment

- Hospitalized
- 60mg prednisone PO QD
- 24 million units/day IV aqueous PCN X 10 days

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Acquired Syphilis Stages:



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Neurosyphilis Tx (CDC Guidelines)

Ocular disease = Neurosyphilis

1. Aqueous PCN G 18-24 million units/day IV x 10-14 days
 - Alt: Procaine PCN 2.4 million units/day IM x 10-14 days **PLUS** PO Probenecid 500 mg QID x 10-14 days
2. CSF examination & HIV testing
3. Repeat LP Q6mo X 2 yrs

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Case 4: Post-Ab treatment

- VA 20/100, PH 20/50 OS
- Essential resolution of uveitis and vitritis
- Follow-up on going



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Case #5

15YOM new patient presents in office with complaint that he woke up with "no vision" OS this morning.

Reports wavy vision and eye pain OS before going to bed last night. Throbbing eye pain has continued.

- o (+) history of migraines
- o Had slight headache at bedtime
- o (+) "fender bender" last week

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Case #5

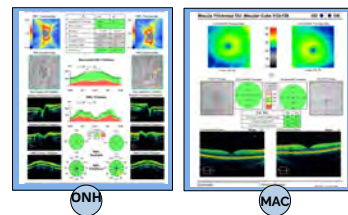
Exam findings:

- UCVA: OD 20/20 OS NLP
- IOP with iCare: OD 19 OS 14
- PERRL APD
- No pain on eye movement with full range of motion
- Anterior segment OU with slit lamp: unremarkable
- Posterior segment OU with slit lamp: unremarkable

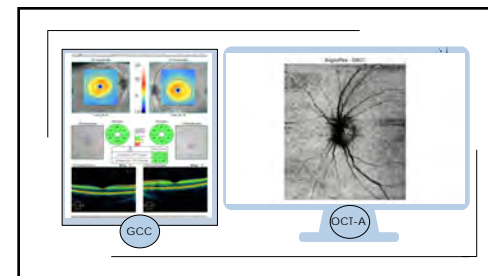
Where do we go from here?
What testing do we need?

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OCT



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OS: Attempt made but patient unable to perform

So now what?

VF

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Referrals

What to order:

Questions to ask:

- Vascular risk factor?
- Stress?
- Home/school environments
- Migraine history

Additional testing:

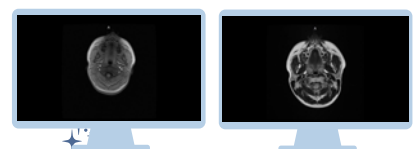
- Brain/orbit imaging
- Bloodwork- hypercoagulability panel
- VEP/ERG
- Follow-up/repeat OCT and VF

Who to refer to:

- STAT imaging/bloodwork
 - Patient's mother given my cell number with instructions to have physician call me
- Pediatrician
- Neuro-ophthalmologist

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MRI



- Brain and orbits unremarkable
- No significant intracranial abnormality
- Bloodwork WNL

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Electrodiagnostics

Test shows good reliability

- Normal low and high contrast amplitude and latency
- No apparent defects within the visual pathway

VEP

ML-ERG and FL-ERG:

- Tests show good reliability
- Both WNL:
 - No abnormalities in the photoreceptors

ERG

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Case #5

Diagnosis:

- Functional vision loss triggered by transient vision loss in the left eye from vasospasm/migraine
 - Studies show patients with visual aura migraines have vision that remains abnormal 7+ days post migraine⁸

Recommendations:

- Continuous monitoring
- Pediatric headache neurologist
- Patient to start nortriptyline



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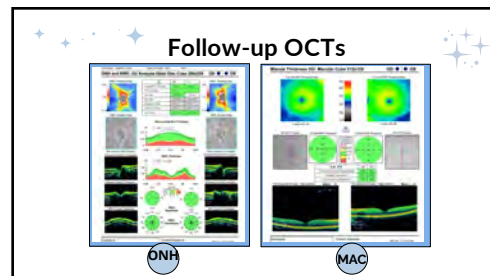
1 week follow-up

- Pt states vision has been improving OS
- States when he covers OD, OS vision will appear doubled, with both eyes open, double vision goes away.
- Pt mentions headache prior to VA starting to improve.

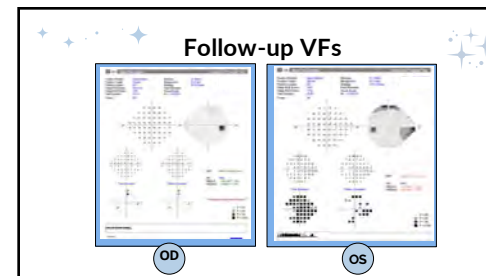
Exam findings:

- UCVA: OD 20/20 OS 20/300
- PERRL-APD
- Anterior segment: unremarkable
- Posterior segment: unremarkable

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Continued follow-ups

3 weeks later:
➤ UCVA OS: 20/100

1 month later:
➤ Patient wakes up that morning with "vision back to normal"
➤ UCVA OS: 20/20

Total time for visual acuity resolution: **2 months**
➤ Patient now under care of pediatric neurologist for migraine control
➤ No additional incidences of vision loss since March

1 week follow-up

1 month follow-up

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What is functional vision loss/nonorganic vision loss? (FVL) (NOVL)

FVL/NOVL:

- Presence of abnormal vision and/or visual fields without organic pathology⁹
- Visual impairment characterized by a disparity between the patient's self-reported visual symptoms and clinical findings¹⁰
- Lacks causative identifiable organic pathology
- Cannot be explained by any identifiable organic pathology in the eye
 - Vision loss not caused by the eye itself but caused by a psychological factor

DIAGNOSIS OF EXCLUSION!

9. Kiki A, Theodoroulopoulos M, Southall P. Nonorganic Vision Loss. European Ophthalmic Review. 2020;15(1):45-52. <https://doi.org/10.1016/j.eur.2020.04.001>
10. Merton DA, Sheehy J. Nonorganic Vision Loss. Updated 2020. Jan 10. In: StatPearls. StatPearls Publishing; 2020. <https://www.ncbi.nlm.nih.gov/books/NBK535550/>

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Functional vision loss

- Functional vision loss is most common in teenagers
 - Typically bilateral
 - More common in females
- According to studies: 20% of functional vision loss patients had migraine or eye/facial pain at time of diagnosis¹¹
- Psychiatric disease was twice as likely in adults compared to children¹¹
- The prognosis is good with a spontaneous recovery in the majority of patients¹²

11. Smith A, Theodoroulopoulos M, Southall P. Functional vision loss in adults and children: clinical presentation, management, and outcomes. Ophthalmology. 2020;127(10):2611-6. doi: 10.1016/j.ophtha.2020.03.040. PMID: 32145382
12. Smith A, Theodoroulopoulos M, Southall P. Functional vision loss in adults and children: clinical presentation, management, and outcomes. Ophthalmology. 2020;127(10):2611-6. doi: 10.1016/j.ophtha.2020.03.040. PMID: 32145382

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Case #5

Cloverleaf defect

Tunnel vs funnel
 Tunnel: Visual field will stay the same as patient moves back
 Funnel: Visual field will expand as patient moves back

11. Linn S, Nils A, Rasmussen, Dornik. (2024). Functional Vision Loss. In: 5772 In: Ophthalmology. 134(20).
12. Rasmussen, A, Dornik, D. (2024). Functional Vision Loss. In: 5772 In: Ophthalmology. 134(20).


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Case #6

Patient: 67YOWM

CC: "I can't see out of my right eye, it started 2 days ago."

HP: (-) pain
 (+) headache-right side of head
 (+) blurry vision-right eye only
 (+) fatigue, pain around back of neck X 2 wks, scalp tenderness
 (-) jaw pain/claudection



- Medical history:** unknown, LME 10+ yrs ago
- Medications:** none
- Allergies:** NKDA
- Ocular history:** unremarkable LEE 2yr ago, cataracts
- Social history:** (-)ETOH, non-smoker

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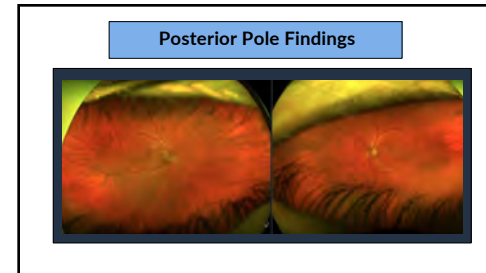
Entrance Testing

- BCVA:** HM @ 4 ft OD, 20/30 OS
- Pupils:** PERRLA, (+)APD OD
- Confrontational VF:** restricted OD, grossly full OS
- FOMS:** Full & Smooth OU, (-)nystagmus
- IOP:** (NCT) 10 mmHG OU

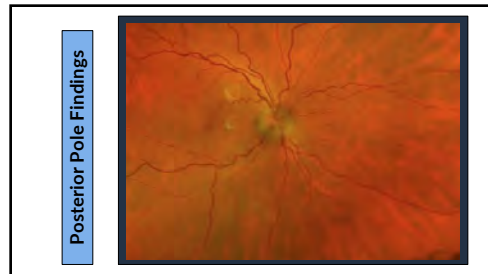
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Slit Lamp Findings		OD	OS
	Lids & Lashes	Normal	Normal
	Conjunctiva/Sclera	Trace injection	Trace Injection
	Cornea	Clear	Clear
	A/C	Deep & Quiet	Deep & Quiet
	Iris	Brown, WNL	Brown, WNL
	Lens	2+ NS	2+ NS

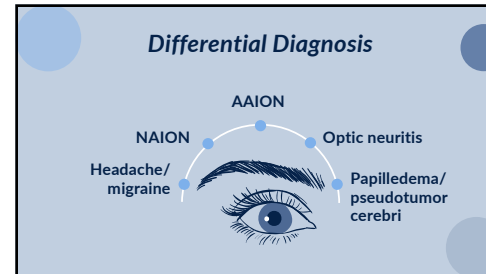
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What's your diagnosis?

- **Diagnosis:** Arteritic ischemic optic neuropathy (AION)- Giant Cell arteritis (GCA)
- **3 Criteria for (American College of Rheumatology) Classification of GCA:**
 - Age of onset >50yrs or older
 - Onset of new headache
 - Temporal artery abnormality (tender or reduced pulsation)
 - Elevated ESR (>50mm/hr Westergren)
 - Abnormal artery biopsy showing necrotizing vasculitis with predominant monocular cell filtration or granulomatous inflammation

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Treatment

- **ER** → CBC, ESR, CRP, FBS, FTA-ABS, ANA
 - ESR >100mm/hr
 - CRP 33mg/L
 - Normal neuroimaging
 - Order Temporal Artery Biopsy
- **Rheumatology consult**
- **Vascular Surgeon** → Temporal Artery Biopsy confirmed GCA
- **Neuroimaging** → rule out intracranial process
- **Steroids** → IV - 1g methylprednisolone sodium succinate X 3 days then 60mg oral prednisone

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Follow-ups


1 week follow-up	2 week follow-up
<ul style="list-style-type: none"> Resolution of headaches, pain, fatigue No change in optic nerve edema Vision decreased to LP Rheumatology for GCA management 	<ul style="list-style-type: none"> Resolved optic nerve edema, improved perfusion VA: NLP OD - no improvement to-date, 20/30 OS

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Giant cell arteritis

- Most common vasculitis adults >50 years
- Incidence 18 per 100,000; Women 4X more likely
- Highest prevalence in Caucasians (Scandinavian or Northern European decent)
- Granulomatous inflammatory vasculopathy affecting medium & large sized arteries
- External carotid branches, ophthalmic, vertebral, distal subclavian & thoracic aorta
- >50 yo, females > males

➤ **Goal: recognize & treat GCA before AION occurs**



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Symptoms

Headache/scalp/temple artery tenderness	Tongue/scalp necrosis
Jaw claudication	Weakness
Neck pain	Fatigue
Weight loss	Unexplained fever

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AAION (arteritic anterior ischemic optic neuropathy)

- Most common cause of severe vision loss from GCA
- Infarction of short posterior ciliary arteries that supply optic nerve
- 1 in 5 GCA patients will develop monocular vision loss related to AAION
- 1/3 patients amaurosis fugax present as sign of impending AION
- Vision loss severe & responds poorly to treatment
- If untreated, 50% lose vision in fellow eye within days to weeks of onset
- TRUE OCULAR EMERGENCY**

- Acute phase → ON appear swollen & pale, flame hemorrhages
- Later → no edema, optic atrophy sets in


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Arteritic AION	Non-arteritic AION
"older" patient population	"younger" patient population
Female > male	No relation
HA, scalp tenderness, jaw claudication	Occasional orbital pain
Better VA	Worse VA
FFA: choroidal & disc filling delay	Disc filling delay
Poor prognosis for recovery; fellow eye 95% cases	3 line VA improvement in 43% cases; fellow eye <30% cases
Urgent corticosteroid treatment	Doubtful role of corticosteroids

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ESR

- Measures height of RBC's settling out of plasma per hour
- Male Norm: age/2
- Female Norm: age + 10 /2



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GCA Ocular Manifestations

- Cranial nerve involvement (CN VI)→diplopia
- Cotton wool spots
- Central Retinal artery occlusion (CRAO)
- Visual Field defect (altitudinal, arcuate, cecocentral scotoma)
- Choroidal infarction
- Nystagmus/internuclear ophthalmoplegia
- Rare=anterior segment neovascularization/ocular ischemic syndrome

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GCA

- Actemra (tocilizumab)** =2017 FDA expanded & approved use of subcutaneous Actemra (tocilizumab) to treat adults with GCA
 - subcutaneous
 - First FDA approved therapy specific to this type of vasculitis
- Polymyalgia Rheumatica (PMR)**
 - Systemic autoimmune disease
 - Shoulder & hip girdle pain
 - 50% GCA patients also have PMR
 - Controversy: GCA & PMR separate or different manifestations of same disease

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GCA Clinical Pearls

- **Thorough case history**
- **Prompt treatment—start tx before lab results are back**
 - If aggressive steroid tx initiated within first 24hrs of onset of visual symptoms, 50% chance of vision improvement
 - Temporal biopsy should be done within 1 week of starting steroid tx
 - Beware of normal labs
 - 15-30% patients with (+) temporal artery biopsies have normal ESR
 - Biopsy temporal artery 5-9% false negative rate due to skip lesions

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Case #7

84YOM presents for a cataract preoperative evaluation

- BCVA OD 20/30-1 OS 20/30
- Glare test OD 20/80 OS 20/60
- Patient is taken back for IOL master, topography, OCTs
- Begins to complain that he feels short of breath and asks to pause the testing momentarily so he can go outside for a moment
- Technician agrees and wheels patient (and wife and son) outside for some fresh air

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Case #7: When to call 911

- Technician calls me and asks me to come outside urgently
- Arrive outside to patient with decreased respirations and slow heart rate and consciousness that is in and out

Step 1
Assign someone to call 911

Step 2
Assign someone to retrieve AED/defibrillator

Step 3
Continue to assess breathing and heart rate

- Found that patient's HR and breathing increased when lying vs sitting
- Regained consciousness momentarily

Step 4
Assess need for CPR

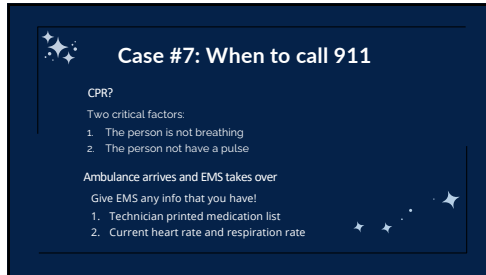
- If needed, begin to perform until AED arrives or EMS arrives and takes over

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AED/Defibrillator DOs and DONTs

When to use:	When NOT to use:	Special circumstances
<ul style="list-style-type: none"> • The person is unconscious • The person is not breathing properly • Absent breath or abnormal • Person's heart rhythm stops due to cardiac arrest <ul style="list-style-type: none"> ◦ Heart attack & cardiac arrest 	<ul style="list-style-type: none"> • The person is conscious • The person is breathing normally • The person has a DNR 	<ul style="list-style-type: none"> • Pacemaker? <ul style="list-style-type: none"> ◦ Typically can still use ◦ Do not apply over where pacemaker was installed • Is the person or the surface wet? • Hairy chest? <ul style="list-style-type: none"> ◦ Must be adhered to skin

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Case #7: When to call 911

CPR?

Two critical factors:

1. The person is not breathing
2. The person not have a pulse

Ambulance arrives and EMS takes over

Give EMS any info that you have!

1. Technician printed medication list
2. Current heart rate and respiration rate

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Thanks!

Do you have any questions?

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