MANAGING EYE URGENCIES AND EMERGENCIES: OPTOMETRY'S ROLE IN TRIAGE AND IMMEDIATE CARE

KELLI SHAON OD, FAAO
TAIA DELANO OD, FAAO

NO FINANCIAL DISCLOSURES

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TOPIC OVERVIEW

- Swollen Eyelid –Cellulitis/inflammation
- Acute Vision loss (in a seemingly health eye)
- Orbital fractures
- Chemical burns
- Corneal ulcers
- Uveitis
- Hyphema
- Corneal abrasions
- Swollen optic nerve (s)
- Herpes Zoster

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SWOLLEN EYELID - DDX

- Infectious
- Allergic
- Inflammatory/Autoimmune
- Traumatic
- Systemic/Vascular
- Neoplastic

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SWOLLEN EYELID - DDX

Infectious
- Preseptal cellulitis
- Orbital cellulitis
- Hordeolum/Chalazion
- Viral – VZV, HSV, Adenovirus*

Allergic
- Contact dermatitis
- Seasonal allergies

Trauma
- Insect bites
- Direct trauma
- Post surgical

Inflammatory/Autoimmune
- Orbital inflammation (IOIS)
- Scleritis
- Blepharitis
- Thyroid eye disease

Systemic/Vascular
- Heart Failure
- Kidney disease

Neoplastic
- Orbital Tumors
- Eyelid tumors

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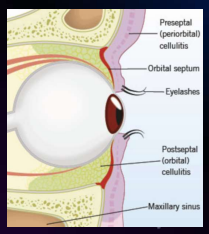
PRESEPTAL CELLULITIS

Definition: Infection the eyelid and periorbital soft tissues anterior to the orbital septum

Etiology
- **Local trauma** (insect bite, scratch, surgery)
- **Spread from nearby infections/inflammation** (hordeolum, chalazion)
- **Skin infections** (impetigo, infected dermatitis)
- **Ocular surface disease** (blepharitis, ocular rosacea)

Clinical Features
- Eyelid redness, warmth, and swelling
- Tenderness of the eyelid
- No proptosis, no pain with eye movement
- No ophthalmoplegia or visual changes
- Usually unilateral
- Rarely fever associated



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PRESEPTAL CELLULITIS

Management/Treatment
- Warm compresses
- Close follow-up – 24-48 hours
- Empiric Oral Antibiotics:
  - Amoxicillin-clavulanate:
    - Adults: 875 mg/125 mg twice daily
    - Children: 20–40 mg/kg/day (amoxicillin component), divided BID
  - Clindamycin: 300–450 mg TID (for penicillin allergy or concern for MRSA)
- If severe case, young children/infant or significant immunocompromised or suppressed – consider hospital admission and IV antibiotics

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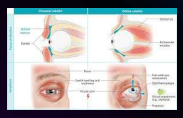
ORBITAL CELLULITIS

URGENT CONDTION!!!

- Definition: Infectious spread post orbital septum
- Vision threatening and can be life threatening
- APD is possible if associated with CON
- Diplopia and Ophthalmoplegia (motility deficits) common
- Pain with eye movements
- Chemosis and Proptosis are common
- Fever and Malaise
- Imaging is needed ASAP
- Etiology: Source of infection??



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ORBITAL CELLULITIS

Management/Treatment

- Requires inpatient care due to the risk of:
  - Vision loss
  - Cavernous sinus thrombosis
  - Intracranial extension (meningitis, abscess)
  - Needs multi-disciplinary care team
- IV Antibiotic Therapy – often broad spectrum
- Urgent CT with contrast of orbits
  - Confirms diagnosis and evaluate for:
    - Subperiosteal abscess, Orbital abscess, Sinusitis

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ORBITAL CELLULITIS

Management/Treatment

- Consulting services: Medicine team, Infectious disease (if available), Ophthalmology/Optometry (monitor vision, APD, IOP) – Monitor q12-24 hours, ENT (sinusitis mgmt) and/or Oculoplastics (Abscess drainage)
- Duration of treatment
  - **IV antibiotics** for at least **3–5 days**, depending on response
  - Transition to **oral antibiotics** (e.g., amoxicillin-clavulanate or clindamycin) to complete **10–14 days** total
- Surgical intervention/drainage indications
  - No signs of improvement in 24-28 hours
  - Orbital or subperiosteal abscess
  - Large abscess on imaging (especially medial >10 mm or inferior >4 mm)
  - Vision loss or optic nerve compression

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ORBITAL INFLAMMATION (IOIS)

Definition: A non-infectious, non-neoplastic inflammatory process involving any part of the orbit. Diagnosis of exclusion - must rule out infection, malignancy, thyroid eye disease

Etiologies

- **Idiopathic**: no isolated cause, but may be immune-mediated (IOIS)
- **Autoimmune conditions**: May be associated with certain conditions
  - Sarcoidosis, lupus, granulomatosis with polyangiitis
- **Viral trigger:** Has been documented in some cases following a vital illness

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ORBITAL INFLAMMATION

Clinical Features

- Acute or subacute onset
- Painful proptosis
- Eyelid edema
- Redness, chemosis
- Ophthalmoplegia (restricted and painful eye movements
- ± Vision changes (if optic nerve involved)
- Usually unilateral, but bilateral cases can
- Can present as
  - Diffuse orbital inflammation
  - Myositis – extraocular muscles inflammation (including tendons)
  - Lacrimal gland involvement – dacryoadenitis

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ORBITAL INFLAMMATION

Imaging Findings (CT/MRI)

- Enlargement of extraocular muscles, including tendon insertions
- Lacrimal gland swelling (if involved)
- Diffuse orbital fat stranding
- No abscess or sinusitis (helps distinguish from orbital cellulitis)
- Contrast enhancement of inflamed tissues

Diagnosis

- Clinical + radiologic diagnosis
- If atypical or unresponsive to treatment, orbital tissue biopsy often required

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ORBITAL INFLAMMATION

Treatment/Management

- Close follow-up initially q1-2 days, until showing improvement
- NSAIDS can be used in milder cases
  - Ibuprofen 600–800 mg **every 6–8 hours (Max daily dose** 3200 mg/day)
  - **Naproxen 500mg BID (Max daily dose** 1000–1500 mg/day)
- High-dose corticosteroids (oral or IV depending on severity)
  - Dramatic and rapid response is typical
  - PO Prednisone – 1-1.5mg.kg day
  - Vision threatening – IV Methlypred – 500-100mg IV x3 day, followed by PO 1mg/kg daily
- Steroid-sparing agents (e.g., methotrexate, azathioprine), if recurrent or chronic

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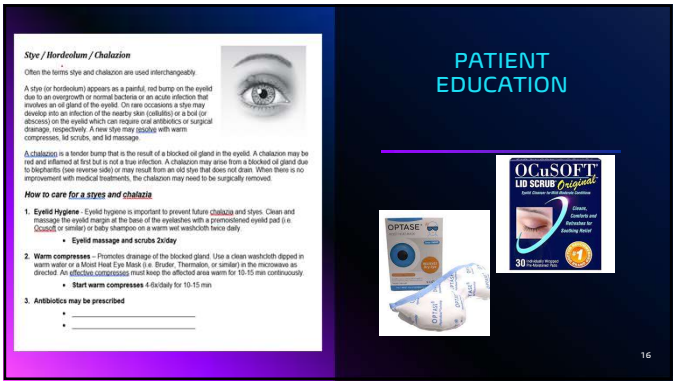

PATIENT EDUCATION

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CASE #1A SWOLLEN EYELID

- 76 year old female presents to our triage clinic as an Urgent care follow-up/referral due to a 4-day history of swelling of his right upper eyelid
- Denies any vision changes or pain with eye movement but does have some pain and tenderness to the eyelid, worse when touched and some crusting of the eyes in the mornings
- Had used warm compresses 1-2x/day since seen in UC 3 days prior
- Also has tried the Erythromycin ung BID like prescribed – has not helped and that makes vision blurry
- Denies fevers, chills, or generalized malaise

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CASE #1B SWOLLEN EYELID

60 year old female presents to our clinic as an outside primary care referral for a recurrence of RUL swelling, she had earlier in the month that improved for a couple of weeks but returned after completing 10 day course of 875-125mg Augmentin. Outside CT facial bones was completed, outside read "preseptal" cellulitis

Medical history: Rheumatoid Arthritis, Hidradenitis Suppurativa, Obstructive Sleep Apnea, Hypertension, Hyperlipidemia

Additional history: 3 weeks prior was diagnosed with maxillary sinusitis, dental abscess (RU molar) and had mild RUL edema. All initial reasons for the Augmentin, symptoms improved. 1 week later had dental extraction. Now over the past 3 days RUL has become red, swollen and not improving with new course of Augmentin

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CASE #1A: SWOLLEN EYELID

What is your working diagnosis?

What testing is needed?

Treatment of condition?

Appropriate follow-up?

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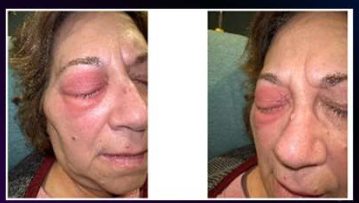
CASE #1B: SWOLLEN EYELID

+ Lid swelling and redness
+ Difficulty opening lid
+ Headache
- No vision changes
- No diplopia
- No fevers/chills

External photos attached



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CASE #1B SWOLLEN EYELID

Lab testing (from visit earlier today): + Leukocytosis = 14.95 (H)

Outside CT facial bones (Radiology overread)

IMPRESSION:
1. Findings consistent with right preseptal cellulitis. No focal abscess.
2. Unrelated right maxillary/ethmoid sinus disease.

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CASE #1B SWOLLEN EYELID

EXAM

VA (sc)

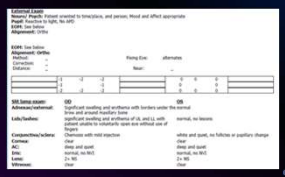
OD: 20/50 ph 20/25+2

OS: 20/20-2

IOP (Tp)

OD: 19

OS: 20



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CASE #1B SWOLLEN EYELID

What do we do next?

What is your working diagnosis?

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CASE #1B SWOLLEN EYELID

1 Day follow-up after broad spectrum IV antibiotics

- Showed improvement in upper lid erythema, redness but inability to elevate RUL
- Conjunctival redness and chemosis was improved,
- Vision stable, IOP Stable
- Leukocytosis slightly improved
- Afebrile
- EOMS: unable to supraduct right eye (- 4 deficit)
- Patient presentation** - Flat affect/Personality Change

What are we thinking?? What next?

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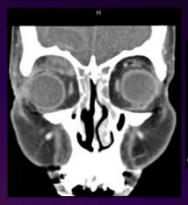
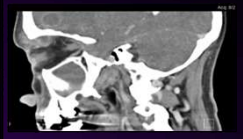
CASE #1B SWOLLEN EYELID





CT results:

Right frontal lobe intracranial abscess (32 mm x 22 mm)

Enhanced superior rectus muscle (21 mm x 9 mm) determined to be abscess

Break in right lamina papyracea at level of posterior ethmoid cells

Communication between right maxillary molar base and maxillary sinus

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CASE #1B SWOLLEN EYELID - UPDATE

- Combined surgical drainage of intracranial abscess and orbital abscess was completed in combined case with oculoplastic surgeon and neurosurgery
- Infectious disease was involved due to extreme nature of case and dental origin of infection
- Patient completed long course of IV antibiotics later followed additional duration of PO
- Last seen in clinic 2 month ago – Patient doing great! EOMs fully intact, complete lid function, and no cognitive issues

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Feature	Preseptal Cellulitis	Orbital Cellulitis	Orbital Inflammation (IOIS)
Definition	Infection anterior to orbital septum (eyelid, skin)	Infection of orbital contents behind orbital septum	Non-infectious inflammation of orbital tissues
Etiology	Trauma, insect bites, skin infection, hordeolum	Sinusitis, trauma, dental infection	Idiopathic, autoimmune, post-viral
Onset	Acute	Acute	Acute to Subacute
Pain	Mild to Moderate	Moderate to severe (esp. with eye mvmt)	Moderate to severe (esp. with eye mvmt)
Vision change	None	Blurred vision, diplopia not uncommon	Possible vision changes
Motility	None	Restricted and painful	Restricted and painful
Chemosis	Uncommon	Common	Common
Proptosis	Absent	Present	Present
Systemic	Usually mild fever or none	Fever, malaise common	Low fever may be present
APD	Absent	May be present	May be present
Imaging	Soft tissue swelling anterior to septum	Orbital fat stranding, abscess, sinusitis, dental concerns possible	Muscle enlargement incl. tendons, no abscess
Treatment	Oral antibiotics, outpatient f/u	IV antibiotics, possible surgery, inpatient	Steroids (after ruling out infection)

Swollen eyelid differentials

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ACUTE VISION LOSS (IN A SEEMINGLY HEALTHY EYE) - DDX

Neurologic

Vascular

Metabolic/Toxic

Genetic/Inherited

Functional (Non-organic)

Infectious/Inflammatory

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ACUTE VISION LOSS (IN A SEEMINGLY HEALTHY EYE) - DDX

Neurologic Causes:
- Optic neuritis
- Occipital lobe stroke
- Multiple sclerosis
- Migraine aura (without headache)
- Leber's Hereditary Optic Neuropathy (LHON)

Vascular Causes:
- Amaurosis fugax
- Posterior circulation stroke
- Giant Cell Arteritis
- Central retinal artery occlusion (early)

Metabolic/Toxic Causes:
- Tobacco-alcohol toxicity
- Ethambutol or isoniazid toxicity

Genetic/Inherited Causes:
- Dominant optic atrophy
- Stargardt disease (early phase)

Functional (Non-organic) Causes:
- Functional vision loss
- Malingering

Infectious/Inflammatory Causes:
- Neurosyphilis or Lyme disease
- Sarcoidosis

Other Considerations:
- Papilledema (early)
- Chiasmal lesions (e.g., pituitary adenoma)
- Retinal dystrophies (early)

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OPTIC NEURITIS

Definition:
- Inflammation of the optic nerve, often associated with demyelinating diseases like multiple sclerosis.

Etiology
- Multiple sclerosis (MS)
- Post-viral or idiopathic
- Neuromyelitis optica
- Infections (e.g., syphilis, Lyme disease)

Clinical Features
- Pain with eye movement
- Decreased vision and color vision
- Relative afferent pupillary defect (RAPD)
- Disc may be swollen (papillitis) or normal (retrobulbar)

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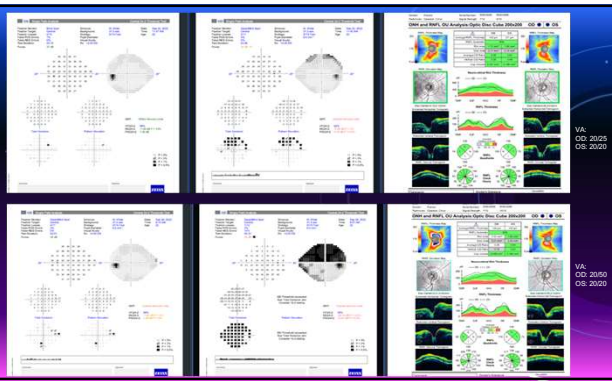

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OPTIC NEURITIS

Management/Treatment
- MRI/MRV brain and orbits w/ and w/o contrast, orbital cuts, and fat suppression
- If MRI shows at least one typical area of demyelination: start methylprednisolone 1 g IV QD x 3 days followed by oral prednisone 1 mg/kg PO QD x 10 days and then taper
- recommend CBC, ESR, CRP, RPR, FTA-ABS, Quantiferon Gold, Lyme titers, ACE, CXR
- Neurology referral for MS evaluation
- **The Optic Neuritis Treatment Trial (ONTT) found steroid treatment reduced initial progression to clinically definite multiple sclerosis (CDMS) for 3 years. Steroid therapy only increases the rapidity of visual return but does not improve final visual outcome.

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AMAUROSIS FUGAX

Definition:
- Transient monocular vision loss due to retinal ischemia, often described as a 'curtain coming down' over the eye.

Etiology
- Emboli from carotid artery disease
- Cardiac emboli (e.g., atrial fibrillation)
- Giant cell arteritis
- Hypercoagulable states

Clinical Features
- Sudden, painless vision loss in one eye
- Typically lasts seconds to minutes
- Normal ocular exam between episodes

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AMAUROSIS FUGAX

Management/Treatment
- Amaurosis fugax is considered by the American Heart Association to be a form of TIA
- MRI
- Urgent carotid ultrasound and cardiac evaluation(including echocardiogram)
- ESR/CRP to rule out giant cell arteritis
- Antiplatelet therapy (aspirin)
- Carotid endarterectomy if high-grade stenosis
- CBC with differential, fasting blood sugar, hemoglobin A1c, and lipid profile (to rule out polycythemia, thrombocytosis, diabetes, and hyperlipidemia)

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FUNCTIONAL VISION LOSS

Definition:
- Vision loss without an identifiable organic cause; often psychogenic or malingering.

Etiology
- Conversion disorder
- Malingering (secondary gain)
- Psychological stress/trauma

Clinical Features
- Normal ocular exam and imaging
- Inconsistent visual responses
- Normal optokinetic nystagmus, mirror test, etc.
- May involve tunnel vision

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TESTING FOR MONOCULAR VISION LOSS

Test Name	Principle	How it works
Red-Green Duochrome test	Red and green filters isolate vision to each eye. Seeing letters through a filter indicated vision in that eye	Patient wears red/green glasses and reads colored letters. If they read letters only visible through the "Blind" eye's filter, they are using that eye
Stereopsis (Depth Perception) Testing (e.g., Randot Stereo Tests)	Stereopsis requires good vision in both eyes and binocular fusion. If a patient claims monocular blindness but demonstrates good stereopsis, it indicates they are using both eyes.	Patient wears 3D glasses and views stero images. Seeing 3D shapes suggest both eyes are working.
Fogging Lens test	Blurs the "good" eye with a strong plus lens. Only the "bad" eye remains optically clear.	If the patient reads clearly despite the "good" eye being fogged, then they must be using the "bad eye."
Vertical prism dissociation test	Prism in front of only one eye should induce vertical diplopia if both eyes have vision.	A 4-prism diopter lens is placed base-down in front of the good/unaffected eye. A 20/20 or larger size Snellen is projected. If the patient is able to see two letters of equal clarity, it establishes good vision in the affected eye
"20/400" at 10 feet test	Vision should improve as distance decreases. Fixed complaints at all distances are inconsistent	A patient who reads the same line at all distances (e.g., 20/400 at 20ft and at 5 ft) is likely exaggerating or feigning vision oss.

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RELATIONSHIP OF VISUAL ACUITY AND STEREOPSIS

Visual acuity	Average Stereopsis (seconds of arc)	Titmus stereopsis
20/20	40	9/9 circles
20/25	43	8/9 circles
20/30	52	8/9 circles
20/40	61	7/9 circles
20/50	78	6/7 circles
20/70	94	5/9 circles
20/100	124	3/3 animals or 4/9 circles
20/200	160	3/9 circles or 2/3 animals

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TESTING FOR BINOCULAR OCULAR VISION LOSS

Test	Description	Expected in True Vision Loss	Findings in Functional Vision Loss	Interpretation
Optokinetic Nystagmus (OKN)	Patient views a rotating striped drum or tape	No response if truly blind	Normal OKN present	Indicates vision is intact subconsciously
Mirror Test	Patient asked to look at their own eyes in a mirror	No fixation if vision impaired	Patient fixes gaze normally	Requires functioning binocular vision
Fingertip touching	Patient touches fingertips of two hands	Still able to touch finger tips	Patients with functional binocular blindness will often claim they are unable to do this.	Indicates deception since this task is possible even with vision loss
Tangent Screen (Peripheral Fields)	Maps visual field at close and far distances	Peripheral field expands with distance	Same field size at all distances	Field constriction in tubular shape is non-physiologic
Visual Evoked Potentials (VEP)	Measures electrical response in visual cortex	Reduced amplitude in true loss	Normal VEP with claimed blindness	Objective confirmation of intact visual pathway
Stereoacuity Testing (e.g., Titmus Fly)	Assesses depth perception	Absent in true binocular loss	Normal stereopsis reported	Indicates functional use of both eyes

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CASE #2A

13 YEAR OLD CAUCASIAN FEMALE PRESENTS TO TRIAGE CLINIC FOR SUDDEN ONSET BLURRED VISION OD. PATIENT STATES THIS STARTED FRIDAY AFTER SCHOOL SUDDENLY BUT HAS WORSENED SINCE THAT TIME. PATIENT STATES VISION IS BLURRY IN ALL FIELDS IN THE EYE.

DENIES EYE PAIN WITH MOVEMENT. NO EXTREMITY WEAKNESS, NUMBNESS, TINGLING, ANTECEDENT FLU-LIKE VIRAL SYNDROME.

DOES ENDORSE RECENT RASH ON CHEST BELIEVED TO BE RELATED TO USING FAKE EYELASHES

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TANGENT SCREEN

•The patient sits about three feet away from a flat, black fabric screen with a central target.
•One eye is covered while the patient focuses on the central target with the other eye.
•The examiner moves a small target (like a pin on a wand) into the patient's side vision from the periphery of the screen inwards.
•The patient indicates when they first see the target, and the examiner marks this point on the screen.
•This process is repeated at various points around the central target to create a map of the patient's visual field.

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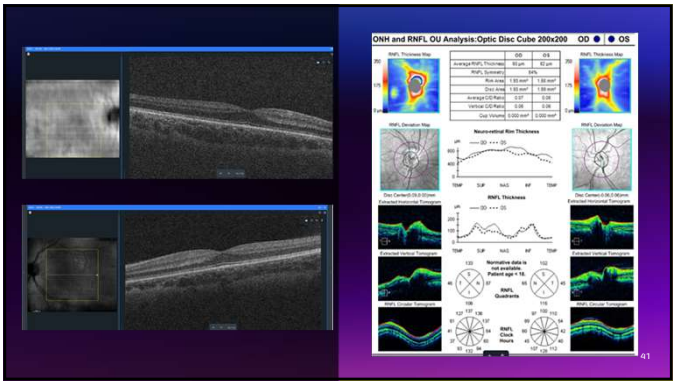

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FUNCTIONAL VISION LOSS

Management/Treatment

- Reassurance and education
- Avoid unnecessary invasive testing
- Psychiatric evaluation if indicated
- Follow-up to monitor for organic pathology

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PHYSICAL EXAM

VISUAL ACUITY:
OD: CF @ 4FT
OS: 20/20-2

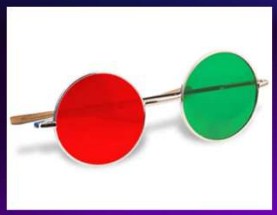
EOMS:
FROM OU

RED/GREEN TEST: FAILED

PUPILS:
3+ APD OD

SLIT LAMP EXAM:
WNL OU

FUNDUS EXAM:
NERVE:
TR DISC EDEMA SUPERIOR OD,WNL OS
OTHERWISE WNL OU



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ASSESSMENT/PLAN:

Optic Neuritis of Right Eye
- Pt with symptoms suggestive of optic neuritis including unilateral decrease in vision, red desaturation OD.
- No extremity weakness, numbness, tingling, antecedent flu-like viral syndrome
- Patient sent to emergency department for further evaluation and management:
- Recommend MRI/MRV brain and orbits w/ and w/o contrast, orbital cuts, and fat suppression. If MRI shows optic nerve inflammation suggestive of optic neuritis, recommend workup below:
  - neurology consult and can consider starting methylprednisolone with appropriate pediatric dosage.
  - Lab work: CBC, ANA/ANCA, ESR/CRP, ACE, Sjogren's - B12, B1, Folate, lyme antiboidies, toxo, bartonella, HIV, RPR, HSV, QuantiFERON Gold

*NOTE: NEVER use oral prednisone as a primary treatment because of increased risk of recurrence found in ONTT.

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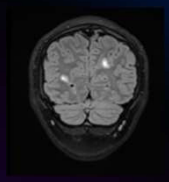
FINDINGS:

- MRI BRAIN: "There are multiple T2/FLAIR enhancing foci in the supratentorial periventricular and subcortical white matter of the bilateral cerebral hemispheres without corresponding restricted diffusion. AdditionalT2/FLAIR enhancing foci seen in the bilateral cerebellum and pons. Some of these lesions demonstrate incomplete enhancement."



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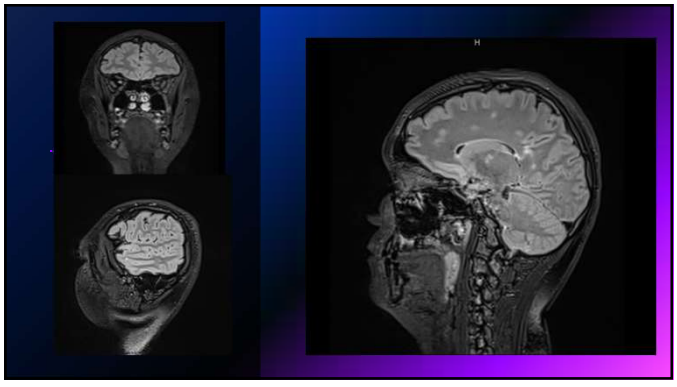

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NEUROLOGY CONSULT ASSESSMENT/PLAN:

1. Demyelinating changes in brain

13 year old female with progressive right eye pain and blurred vision and vision loss for 1 week and MRI showed multiple enhancing lesions in the supra and infratentorial brain including pons suggestive of active demyelinating disease, possible etiologies include MS, NMO and MOGAD.

Recommendations:

1) Solu-Medrol 1 gram IV per day for 5 days
2) GI prophylaxis
3) follow up CSF MS panel, NMO and MOGAD panels

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CASE #2B

61YO FEMALE PRESENTS WITH CONCERN OVER FLASHES IN HER RIGHT EYE AND A FILM OVER IT FOR THE PAST MONTH. SHE ENDORSES EPISODES OF COMPLETE WHITENING OF VISION IN HER RIGHT EYE LASTING 3-5 MINUTES OVER THE PAST 4 WEEKS.

(+) INTERMITTENT PHOTOPHOBIA

TYPE II DM, LAST A1C 11.5% (02/2023)

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PHYSICAL EXAM

VISUAL ACUITY: OD: 20/30 OS: 20/30

EOMS: FROM OU CVF: FULL

PUPILS: PERRL (-)APD

SLIT LAMP 2+ NS OU, OTHERWISE WNL OU

FUNDUS EXAM:

OPTIC NERVES:

OD 0.50 OS 0.55, SHARP MARGINS OU, (-)PALLOR/EDEMA OU

MACULA: OU: CLEAR

VESSELS: NORMAL OD; HOLLENHORST PLAQUE SUPERIOR TEMP ARCADE

PERIPHERY: WHITE WITHOUT PRESSURE SUPERIOR TEMP OS, INFERIOR OD: 360 SCATTERED MILD BLOT HEMORRHAGES OD

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ASSESSMENT/PLAN:

1. amaurosis fugax OD
   - with episode of complete whitening of vision lasting 3-5 minutes over the past 4 weeks
   - with concurrent findings of impending CRVO as well as Hollenhorst plaque in fellow eye
   - patient deferred ambulance transportation and opted to have her driver that is here with her today take her to the ED
   - Discussed with Pete in ED and recommend full stroke work-up : recommend ESR, CRP, CBC, carotid doppler MRA or CTA and cardiac evaluation with echocardiogram
2. impending CRVO OD
   - with unilateral mid-peripheral blot hemorrhages
   - DDx: includes OIS
3. Hollenhorst plaque OS
   - superior temporal arcade
4. NIDDM without ocular manifestations
   - Patient educated on importance of BS control. Monitor yearly.

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FINDINGS:

1. HEAD CT: No evidence of acute intracranial abnormality.
2. HEAD CTA: No evidence of significant intracranial arterial abnormality. The intracavernous right internal carotid artery is occluded.
1. NECK CTA: right internal carotid artery complete occlusion due to presence of calcified and noncalcified atheromatous plaque at its origin.

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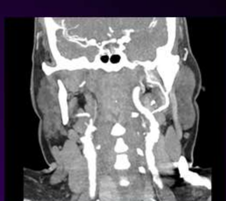
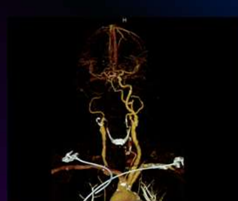
FINDINGS:





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VASCULAR SURGERY CONSULT :

HPI:
61 yof with a hx of CAD s/p PCI, uncontrolled DM A1C 11, who presented to ophthalmology office with a 1 month hx of right vision brightness "as if someone is shining a light in my right eye". She has known floaters for many years due to her DM. Patient denies any hx of stroke or TIAs. What she describes is not at all consistent with amaurosis fugax. She denies any temporary vision loss. She currently takes asa, ambulates daily, denies any one sided weakness, difficulty with speech or vision. CTA demonstrated occlusion of the right ICA. There are no older studies to compare this to.

Assessment/Plan:
- 61 yof with asymptomatic right ICA occlusion.
- no intervention from a vascular surgery standpoint
- she should f/u with vascular surgery clinic in a few months for f/u of left ICA stenosis <50% with a duplex US

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CASE #2C

35-YEAR-OLD FEMALE WITH NO SIGNIFICANT PAST MEDICAL HISTORY PRESENTS AFTER BEING SEEN IN THE EMERGENCY DEPARTMENT YESTERDAY WITH A RIGHT HOMONYMOUS HEMIANOPSIA VISUAL FIELD DEFECT.

ORIGINALLY, SHE WOKE UP ON 12/23/21 WITH EYE PAIN AND WAS FOLLOWED BY AN OUTSIDE OPTOMETRIST FOR A CORNEAL ABRASION. ONCE THIS WAS RESOLVED, SHE WAS FOLLOWED UP FOR HER SUSPICIOUS OPTIC NERVES AND A HVF WAS PERFORMED. AT THAT VISIT, THE PATIENT WAS FOUND TO HAVE A RIGHT HOMONYMOUS HEMIANOPSIA. SHE WAS SENT TO THE ED YESTERDAY AND HAD AN MRI AND MRA WHICH WERE BOTH FOUND TO BE UNREMARKABLE.

OF NOTE, SHE ALSO DESCRIBES STOMACH PAIN, NAUSEA, DIARRHEA, AND VOMITING ON-AND-OFF WHICH STARTED AROUND THANKSGIVING AS WELL AS AN ACUTE EPISODE OF SHORTNESS OF BREATH WHICH SHE WAS SEEN IN THE ED FOR. HER WORK-UPS FOR THIS WERE ALSO DEEMED NORMAL. PRIOR TO THIS SHE HAD ANXIETY STEMMED FROM AN INABILITY TO LOSE WEIGHT BUT HAS SINCE HAD SOLB WEIGHT LOSS SINCE 11/2021. NO RECENT FEVERS, NO RECENT JAW PAIN. TEMPORAL PAIN W/ HEADACHES. SOME RIGHT EYE PAIN.

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PHYSICAL EXAM

VISUAL ACUITY: OD: 20/30 OS: 20/25

EOMS: FROM OU CVF: FULL

PUPILS: PERRL (-)APD

IOP: 22/19

SLIT LAMP: 1+ NS OU, OTHERWISE WNL OU

FUNDUS EXAM:

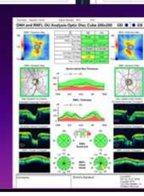
OPTIC NERVES:

OD 0.60 OS:0.55, SHARP MARGINS OU, (-)PALLOR/EDEMA OU

MACULA: OU: CLEAR

VESSELS: NORMAL OU

PERIPHERY: FLAT AND ATTACHED 360 OU



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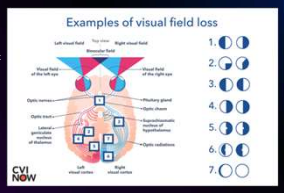
ASSESSMENT/PLAN:

1. Right Homonymous Hemianopsia
 - Respecting midline and more dense in right lower quadrant in both eyes
 - Patient noticed right peripheral field defect in both eyes since December 23, 2021
 - Repeat VF today shows dense right defect w/ more density in lower right quadrant in both eyes, concerning for post-chiasmal/post-LGN cranial abnormality in left parietal cortex
 - MRI and MRV negative for any acute abnormalities, and called neuroradiology again today who confirmed that MRI an MRV did not show any acute stroke or chronic stroke or left parietal lobe abnormalities seen
 - platelets WNL, CRP and ESR slightly elevated
 - No jaw claudication, young age, no recent fever
 - No optic disc edema or retinal detachments seen in either eye
- Patient has had anxiety, GI issues w/ no obvious source, SOB w/ no diagnosis at ED – high suspicion for psychosomatic/functional vision loss
 - Recommend patient f/u w/ neuro-oph next week



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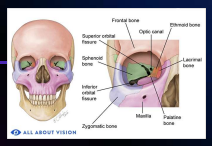
ORBITAL FRACTURES



Where ORBITAL fractures can occur

- Floor – Maxilla (roof of maxillary sinus) and Palatine bone
  - Infraorbital groove and foramen – in a fracture can result in infraorbital nerve impingement/damage
    - Can results in Sensory disturbance
- Medial wall– ethmoid, lacrimal, maxillary and sphenoid bones
- Lateral wall – comprised of Zygomatic (anteriorly) and Greater wing of Sphenoid bone (posteriorly)
- Roof – Frontal bone and small posterior portion is Lesser wing of Sphenoid bone
  - This requires Skull based surgeon to evaluate (often Neuro-surgery)

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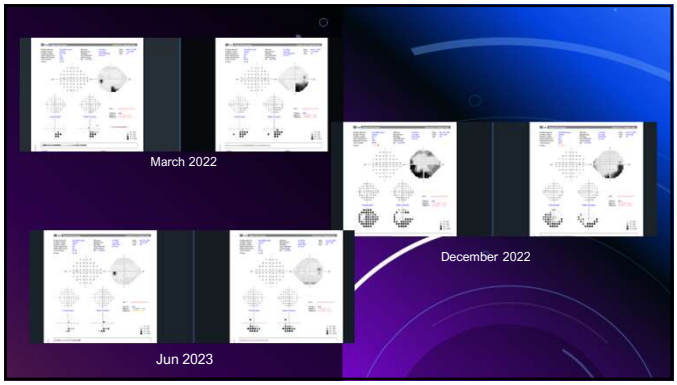

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ORBITAL FRACTURES

Definition: A break in one or more of the bones that make up orbit, often caused by blunt trauma to the globe or periorbital region.

With a fracture of the orbital floor, the orbital contents (fat and muscle) may herniate into the maxillary sinus, which can result in soft tissue entrapment and possible muscle dysfunction. If nasal wall, fracture fibers of medial rectus can also get trapped in ethmoid

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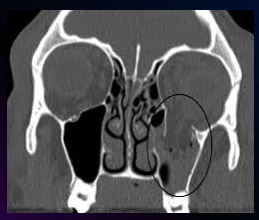
ORBITAL FRACTURES



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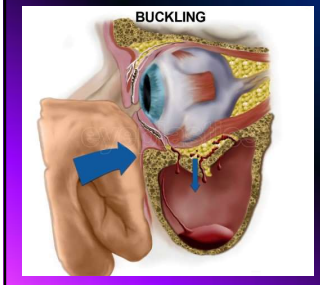

ORBITAL FRACTURES

Case History to Obtain

- What was mechanism of injury (e.g., fist, ball, MVC)?
- Diplopia present (especially on upgaze)?
- Presence of orbital pain or pressure?
- Numbness in cheek/upper lip (infraorbital nerve)?

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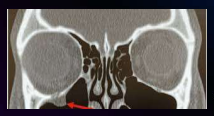
ORBITAL FRACTURES

CT imaging of orbits/facial bones (non-contrast):

- Verify location of bone fracture
- Assess for concern of muscle entrapment
- Presence or absence of orbital emphysema
- Is there herniation of orbital contents



** ALWAYS READ YOUR OWN IMAGES – if you need help reach out to Radiology

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ORBITAL FRACTURES

External/ Ophthalmologic Evaluation

- Periorbital swelling, ecchymosis
- **Enophthalmos** - Hertel/exophthalmometry (perform if able at baseline but every follow-up)
- **Motility/EOMS**
- **Check for restricted eye movements**, especially upward gaze (inferior rectus entrapment)
- **Diplopia** in upward/lateral gaze
- **Subconjunctival hemorrhage**
- Hypoesthesia of infraorbital region
- Positive forced duction test (if necessary)
- External photos -9 gaze + Worm's eye
- Visual acuity
- Pupils (check for RAPD)
- Extraocular movements
- Intraocular pressure
- Fundoscopic exam

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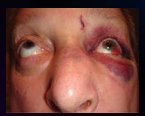
ORBITAL FRACTURES

MANAGEMENT

Most small fractures can be managed conservatively unless complicated.

- **Avoid nose blowing**, sneezing (may cause orbital emphysema)
- **Nasal decongestants** (e.g., oxymetazoline for 3–5 days)
- **Oral antibiotics** (controversial; used in large fractures or sinus involvement):
  - Amoxicillin-clavulanate or clindamycin
- **Cold compresses** in first 24–48 hrs
- **Head elevation** to reduce swelling
- **Analgesia** for pain control – often APAP



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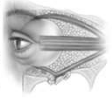
Orbital "Blow Out" Fractures

The orbit, or eye socket, is a bony opening that contains the eyeball and the muscles, blood vessels, nerves and fat that help support it. Blunt force trauma to the head or around the eye can break the bones of the orbit, leading to a "blow-out" fracture. The areas along the inside wall (the wall between the eye and the nose) and floor are the thinnest and fractures are more likely to occur here. A CT scan is usually obtained to confirm the presence and exact location of the broken bone(s). Soft tissue may sometimes be trapped in the fracture site. Symptoms of an orbital fracture may include pain, swelling, bruising, double vision, nausea, numbness of the cheek or upper teeth. After swelling subsides, the eye can appear sunken. It is important that the eyeball is carefully examined, as it can also be damaged as a result of the trauma.

Treatment

Not all broken orbit bones need to be fixed. If the fracture site is not too big, if there is no bothersome double vision and if the eye doesn't look sunken, many patients can be allowed to heal without the need for surgery. Right after the injury, it is not always clear if a patient will need surgery. Your surgeon will follow you closely and may prescribe **cold compresses**, **antibiotics or a short course of anti-inflammatory pills**. **During this time you should avoid sneezing or blowing your nose and should not fly in an airplane or go deep-sea diving.** These activities may allow air to enter the orbit, causing further discomfort and damage.



PATIENT EDUCATION

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ORBITAL FRACTURES

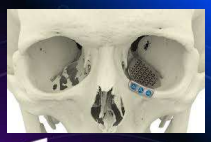
Surgical intervention

Non-Urgent surgery, typically within 1–2 weeks (if indicated)

- Non-resolving diplopia with positive forced duction
- Enophthalmos >2 mm or large orbital floor defect (>50% or >2 cm²)
- Globe malposition (Hypoglobus) with cosmetic deformity

Urgent Surgery (<24–48 hrs):

- **Oculocardiac reflex** (bradycardia, nausea, syncope from muscle entrapment)
- **Muscle entrapment** (especially in children – "white-eye blowout fracture")



Repair material:
Titanium mesh
Porous polyethylene (Medpor)
Autologous graft (e.g., calvarial bone)

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CASE #3A: ORBITAL FRACTURE

66 yo female referred from outside Urgent care after she had a ground floor fall 3 days prior. She tripped over something that resulted in her hitting the left side of her face. She has mild tenderness around the eye but numbness inferonasal under the eye.

VA sc : OD 20/40 OS 20/40

IOP 10/12

EOMS: full OD, -0.5 limitation in supraduction OS – with mild pain with gaze

Pupils: ERRL - APD

Color plates: 11/11 OD/OS

Lids: WNL OD, Edema/ecchymosis LUL/LLL

Anterior seg: Gr 2 NS OU; SCH OS, otherwise WNL

Post Seg: WNL

Hertel: Base 97 19OD 17OS

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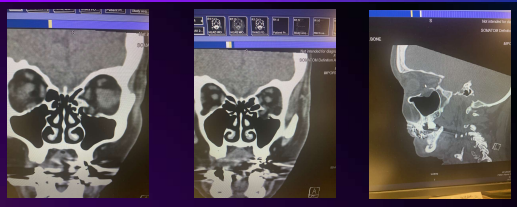
CASE #3A: ORBITAL FRACTURE



- CT scan personally reviewed and shows left orbital floor blowout fracture with inferior displacement of the fracture fragment and herniation of intraorbital fat into the left maxillary sinus. There is no obvious extraocular muscle entrapment.

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CASE #3B: ORBITAL FRACTURE –

35 yo female presents to clinic after being seen the night before in local ED with documented left orbital floor fracture.

- What extra testing do I want to do specifically for fractures??

VA sc : OD 20/20 OS 20/20

IOP 16/18

EOMS: full OD/OS

Pupils: ERRL - APD

Color plates: 11/11 OD/OS

Lids: WNL OD, Edema/ecchymosis LUL/LLL

Anterior seg: WNL except noted in photo

Post Seg: WNL

Hertel: Base 102 OD 19 OS 19

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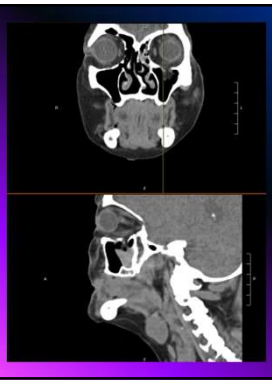
CASE #3B: ORBITAL FRACTURE



IMPRESSION:
Bilateral nasal bone fractures and nasal septal fracture with fracture also
present in the left medial orbital wall. Adjacent soft tissue hematoma in the
left infraorbital region.

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CASE #3C: ORBITAL FRACTURE

29 yo male presented to the ED after being hit in the right side of face and orbit with a football 24-30 hours prior. He noted pain and swelling of the eyelids on that side. Was endorsing mild blurry vision some difficulty focusing with right eye

VA sc : OD 20/20-1 OS 20/20

IOP 21/20

Pupils: ERRL - APD

EOMS: full OD/OS – with exception of mild blurring of images in inferior gaze OD

Color plates: 11/11 OD/OS

Anterior seg: WNL except noted in photo

Post Seg: WNL
Hertel: Base 104 OD 16mm OS 17mm

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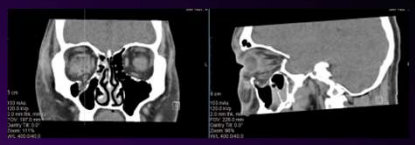
CASE #3C: ORBITAL FRACTURE



IMPRESSION:
1. Small right lamina papyracea and right orbital floor blowout fracture with associated edema sinus. No imaging signs of entrapment.
2. Right-sided retrobulbar hematoma and associated proptosis with suboptimal evaluation of the optic nerve, possibly injured.
3. Right periorbital contusion/hematoma.

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CASE COMPARISION: ORBITAL FRACTURES

Based off initial history/exam and CT…Which of these 3 cases – need surgery, if any?

When would you see these patients back?

What examination information is required?

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CHEMICAL BURNS

Definition:
- injury to the eye from exposure to acidic or alkaline substances

Emergency!!
- Time and vision sensitive- severity is based on the agent, duration and penetration depth

Clinical Presentation:
- Immediate pain and tearing
- Redness, photophobia, blurred vision
- Lid swelling, conjunctival chemosis
- Corneal haze or opacification
- Elevated IOP (in severe cases)

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CHEMICAL BURNS- TYPES OF CHEMICAL ANGENTS

Alkali Burns (more severe)
- Examples: Ammonia, Lye (sodium hydroxide), lime, bleach
- Mechanism: saponifies cell membranes, penetrates rapidly

Acid Burns:
- Examples: Sulfuric acid (battery acid), hydrochloric acid
- Mechanism: coagulation necrosis, usually limited depth

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CHEMICAL BURNS

Common sources of exposure:
- Household cleaners
- Industrial chemicals
- Fertilizers, pesticides
- Car batteries
- Airbags

Signs:
- Corneal epithelial defects range from scattered supericial punctate keratopathy to loss of the entire epithelium.
- perilimbal ischemia
- Chemosis
- Conjunctival blanching
- Corneal edema/opacification

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CHEMICAL BURNS- INITIAL EMERGENCY MANAGEMENT

1. Irrigation (most critical!!)
   - Begin immediately, even before arriving in clinic
   - Use normal saline, but advise patient to start flushing immediately with whatever they have available
   - Continue for at least 30 minutes or until pH is 7.0-7.3

2. Remove any particulate matter
   - Evert lids
   - Use cotton swab or forceps to sweep

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CHEMICAL BURNS

Ongoing medical care:
- Consider cycloplegic (e.g., cyclopentolate 1% or 2%, homatropine 5% b.i.d. to t.i.d.) if significant photophobia, pain, or AC inflammation. If limbal ischemia is suspected, avoid phenylephrine because of its vasoconstrictive properties.
- Frequent (e.g., q1h while awake) use of preservative-free artificial tears.
- Consider topical steroids (i.e., prednisolone acetate 1% q.i.d.) as adjunctive treatment with topical antibiotic for a week even if epithelial defect is present, especially if alkali injury.
- Oral pain medication (e.g., acetaminophen) as needed.
- If IOP is elevated, acetazolamide 250 mg p.o. q.i.d, may be given. Electrolytes, especially potassium, should be monitored in patients on these medications. Add a topical beta-blocker (e.g., timolol 0.5% b.i.d.) if additional IOP control is required. Alpha-agonists should be avoided because of their vasoconstrictive properties, especially if limbal ischemia is present.
- Consider oral vitamin C
- Consider oral doxycycline

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CHEMICAL BURNS- VITAMIN C

Mechanism
- Essential for collagen synthesis and stromal repair

Indication:
- Moderate to severe ocular chemical burns; supports corneal healing and prevents melting

Dosage:
- 500mg-2g daily (orally)

Why:
- Reduces risk or corneal ulceration and promotes epithelial regeneration

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VITAMIN C LITERATURE

- Grobe GM, Reichl S. Characterization of vitamin C-induced cell sheets formed from primary and immortalized human corneal stromal cells for tissue engineering applications. Cells Tissues Organs. 2013;197:283–97. 7
- Lee MY, Chung SK. Treatment of corneal neovascularization by topical application of ascorbic acid in the rabbit model. Cornea. 2012;31:1165–9.
- Chen J, Lan J, Liu D, Backman LJ, Zhang W, Zhou Q, et al. Ascorbic acid promotes the stemness of corneal epithelial stem/ progenitor cells and accelerates epithelial wound healing in the cornea. Stem Cells Transl Med. 2017;6:1356–65.
- Cho Y-W, Yoo W-S, Kim S-J, Chung I-Y, Seo S-W, Yoo J-M. Efficacy of systemic vitamin C supplementation in reducing corneal opacity resulting from infectious keratitis. Medicine. 2014;93:e125.

- "Vitamin C neutralizes oxygen free radicals and inhibits damage to neighboring epithelial and stromal tissues surrounding a wound with protective effects on the cornea."
- Leads to "generation of healthy keratocyte tissue, enhancement of collagen synthesis, and suppression of corneal neovascularization"
- "ascorbic acid accelerates corneal epithelial wound healing by enhancing and upregulating corneal epithelial stem cells"
- "systemic vitamin C supplementation reduced corneal opacity size
- "This finding may be of particular interest in infectious keratitis in the context of corneal injury. As mentioned previously, the use of corticosteroids continues to be controversial due to concerns in delaying wound healing and prolonging infection. As such, vitamin C supplementation may be an effective means to decrease unwanted corneal opacity without incurring potential side effects of steroids.
- "vitamin C may additionally be useful in cases of exuberant ocular surface inflammation associated with corneal thinning and risk of perforation, such as may occur in the setting of chemical and/or thermal injury.

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CHEMICAL BURNS- DOXYCYCLINE

Mechanism
- Inhibits matrix metalloproteinases (MMPs), reducing corneal stromal degradation and inflammation

Indication:
- Severe corneal abrasions and chemical burns at risk for stromal melting

Dosage:
- 50-100mg orally BID

Evidence:
- Effective in preventing corneal melting and promoting healing

80

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DOXYCYCLINE LITERATURE

- Perry HD, Hodes LW, Seador JA, Donnenfeld ED, McNamara TF, Golub LM. Effect of doxycycline hydate on corneal epithelial wound healing in the rabbit alkali-burn model. Preliminary observations. Cornea. 1993;12(5):379–382.
- Dursun D, Kim MC, Solomon A, Pflugfelder SC. Treatment of recalcitrant recurrent corneal erosions with inhibitors of matrix metalloproteinase-9, doxycycline, and corticosteroids. Am J Ophthalmol. 2001;132(1):8–13

- "A possible cause for the pathologic changes observed in corneal erosion is degradation of the epithelial attachment complexes by matrix degrading enzymes."
- "Increased levels and/or activity of several members of the matrix metalloproteinase enzyme family have been reported in patients with recurrent corneal erosions."
- "oral doxycycline administration also decreased the collagenase activity in corneas obtained from these animals."
- "systematically administered doxycycline, promotes corneal reepithelialization in the rabbit alkali-burn model, a result, perhaps, of the drug's ability to inhibit excessive collagenase activity.

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CHEMICAL BURNS

Ongoing medical care, continued....
- Consider amniotic membrane in severe cases
- May require:
  - Conjunctival grafts
  - Limbal stem cell transplantation
  - Corneal transplant

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CHEMICAL BURNS

Prognostic factors:

Worse Prognosis:
- Limbal ischemia
- Corneal opacification
- Delayed irrigation

Better Prognosis:
- Rapid and prolonged irrigation
- minimal limbal involvement
- Early medical therapy

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CHEMICAL BURNS

Grading systems for prognosis:

(Thank you Laura Palazzolo, MD

Table 2. Roper Hall Classification for Ocular Chemical Injury

Grade	Prognosis	Cornea	Limbal Ischemia
I	Very good	Corneal epithelial damage	None
II	Good	Corneal epithelial damage	<33 percent
III	Guarded	Total epithelial loss, stromal haze, iris details obscured	33 to 50 percent
IV	Poor	Cornea opaque, iris and pupil obscured	>50 percent

REVIEW OF OPHTHALMOLOGY

Table 1. Dua Classification for Ocular Chemical Injury

Grade	Prognosis	Clinical Findings	Conjunctival Involvement	Analogue Scale
I	Very good	0 clock hours of limbal involvement	0 percent	0/0 percent
II	Good	<3 clock hours of limbal involvement	<30 percent	0.1 to 3/1 to 29.9 percent
III	Good	3 to 6 clock hours of limbal involvement	30 to 50 percent	3.1 to 6/31 to 50 percent
IV	Good to guarded	6 to 9 clock hours of limbal involvement	50 to 75 percent	6.1 to 9/51 to 75 percent
V	Guarded to poor	9 to <12 clock hours of limbal involvement	75 to <100 percent	9.1 to 11.9/75.1 to 99.9 percent
VI	Very poor	Total (12 clock hours) limbal involvement	100 percent	12/100 percent

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CASE #4A

59YO MALE PRESENTS AFTER BEING STRUCK IN THE EYE WITH THE CAP TO ANHYDROUS AMMONIA WHILE WORKING WITH IT

BELIEVES HE GOT SOME OF THE CHEMICAL IN HIS EYES AS WELL

REPORTS IMMEDIATE ONSET OF REDNESS, PAIN, BLURRED VISION.

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1-DAY FOLLOW UP: PHYSICAL EXAM

Visual acuity: OD: 20/25 OS: 20/CF

Pupils: RRL OD, dilated and poorly reactive to light OS

IOP (Tp)

OD: 11

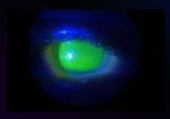
OS: 10-

SLIT LAMP EXAM:

Conjunctiva/sclera ; white and quiet OU

Cornea: 2+ K edema. Epi defect spanning entirety of the cornea sparing with growth in superonasal quadrant with rolled edges around the limbus. No blanching of limbal vessels.

Anterior chamber: 2-3+ RBCs



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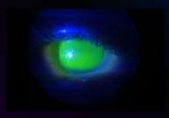
PHYSICAL EXAM

- pH OS- 8. 3 10CC flushes used to irrigate left eye and fornices swept with moistened cotton-tip applicator. pH checked 5 minutes later- 7
- Visual acuity: OD: 20/25 OS: 20/80
- Pupil: RRL OD, dilated and poorly reactive to light OS
- IOP (Tp)
- OD: 16
- OS: 32-- One drop Timolol instilled- IOP remained 32. One drop Timolol/Brim instilled-- IOP 31.
- SLIT LAMP EXAM:
- Conjunctiva/sclera: diffuse injection and chemosis, no peri-limbal ischemia
- Cornea: Epithelial defect spanning near whole surface of cornea, rolled epithelial edges near limbus
- Anterior chamber: deep with 0.5mm inferior layered hyphema and circulating 2-3+ mixed cell



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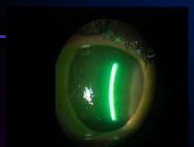
ASSESSMENT/PLAN:

1. Alkaline Injury, Left Eye; Large corneal epithelial defect, left eye
- Secondary to anhydrous ammonia 06/11/2025
- Fornices swept and copious irrigation applied until pH normalized
- Loose epi debrided at SL 06/11/2025
- No evidence of limbal ischemia
- Prokera placed today. See clinic procedure note.
- Continue Pred Forte QID OS
- Continue Vigamox QID OS
- Frequent use of artificial tears (q1 hour)

2. Ocular Hypertension, Left Eye
- Refractory to 2 rounds of topical therapy. Likely secondary to inflammation/bleed
- started Diamox 06/11/2025-- 500mg immediate release given in clinic 06/11/2025
- IOP much improved at follow up 06/12/2025, can decrease Diamox to 250mg BID, if IOP improved at follow come off Diamox.

3. Traumatic Mydriasis, Left Eye

4. Hyphema, Left Eye
- Minimal layered hyphema, likely secondary to blunt trauma from ammonia cap
- continue Cyclopentolate BID OS
- Protective eye shield provided to be worn while sleeping/walking. Instructed to limit bending/straining
- RTC tomorrow for AC check



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ASSESSMENT/PLAN:

1. Alkaline Injury, Left Eye; Corneal Abrasion, Left Eye
- Secondary to anhydrous ammonia earlier today, 06/11/2025
- Fornices swept and copious irrigation applied until pH normalized
- Loose epi debrided at SL today, 06/11/2025
- Start Pred Forte QID OS
- Start Vigamox QID OS
- Frequent use of artificial tears (q1 hour), sample provided today

2. Intraocular Hypertension, Left Eye
- Refractory to 2 rounds of topical therapy. Likely secondary to inflammation/bleed
- Will start Diamox-- 500mg immediate release given in clinic today, 06/11/2025
- Start Diamox 250mg QID until follow up tomorrow

3. Traumatic Mydriasis, Left Eye

4. Hyphema, Left Eye
- Minimal layered hyphema, likely secondary to blunt trauma from ammonia cap
- Start Cyclopentolate BID OS
- Protective eye shield provided to be worn while sleeping/walking. Instructed to limit bending/straining
- RTC tomorrow for AC check

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SUBSEQUENT FOLLOW UP:

06/16/2025: Prokera removed from eye due to symblepharon formation. Large epi-defect. Add BCL. Switch to Durezol q1-2 ours while awake. Add doxycycline 100mg BID

06/18/2025: removed BCL. Significant improvement in epi-defect. Add Maxitrol TID, taper Durezol to QID. Continue Vigamox and Doxy.

06/23/2025: Placed Prokera to wear for 1 week, significant improvement in epi-defect- D/C Maxitrol.

06/30/2025: Prokera removed, OS: 20/60- begin Durezol taper -> 3x/day for 1 week, 2x/day for 1 week, 1x/day for 1 week then STOP ; stop Vigamox QID OS; continue doxycycline 100mg BID for 1 mo total

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CORNEAL ULCERS

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CORNEAL ULCERS - INFECTIOUS

Bacterial Ulcer Treatments

- For mild and small ulcers, a fluoroquinolone (moxifloxacin) can provide adequate gram-positive and gram-negative coverage.
- For central, large or with robust anterior chamber reaction, fortified antibiotics should be started. Ensure you have both gram-positive and gram-negative (Pseudomonas)
- Meds: Fortified Tobramycin 15 mg/mL (Gram -) and Fortified Vancomycin 25mg/mL (Gram +)
- Dosing:
  - Drops should be given initially every one to two hours, 24 hours per day (even overnight), and spaced out five minutes between drops or alternating q 1 hour between types. After improvement, the frequency can be decreased to allow for sleep

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CORNEAL ULCERS DDX

INFECTIOUS
- BACTERIAL
- FUNGAL
- VIRAL
- ACANTHAMOEBA

STERILE/NON-INFECTIOUS
- NEUROTROPHIC
- PUK
- EXPOSURE

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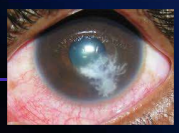
CORNEAL ULCERS - INFECTIOUS



FUNGAL

- **Common Causes:** *Fusarium, Aspergillus, Candida*
- **Presentation**: Gradual onset, feathery-edged infiltrate, satellite lesions, dry appearance
- **Risk Factors**: Trauma with vegetative matter, chronic steroid use, agriculture workers
- **Management:** Corneal Scrapings (taken from deep within the lesion) – Can take several weeks for results
- **Treatments:** Corneal specialist referral, Debridement **AND** Natamycin 5% drops (especially for filamentous fungi) or amphotericin B 0.15% drops (especially for Candida), or topical fortified voriconazole 1% initially q1–2h around the clock, then taper over 4 to 6 weeks

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CORNEAL ULCERS - INFECTIOUS

Bacterial

- **Most common type**
- **Common Causes:** *Staphylococcus*, *Streptococcus*, *Pseudomonas* (esp. in contact lens users)
- **Presentation:** Pain, redness, purulent discharge, focal stromal infiltrate, hypopyon
- **Risk Factors**: Contact lens wear or abuse, trauma, dry eye, ocular surface disease
- **Management:**
  - Corneal cultures/sensitivities, topical antibiotics, cycloplegics, shield (if thinning and risk of perforation)
  - Consider culturing contact lenses/case if available

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CORNEAL ULCERS - INFECTIOUS

VIRAL

- **Common Causes: HSV, VZV**
- **Presentation**: HSV - Dendritic or geographic ulcers (staining with fluorescein), decreased corneal sensation; VZV – recent skin lesions (V1/V2) or history of HZO in the past, Pseudodendrites are smaller in size, elevated without central ulceration, do not have terminal bulbs
- **Risk factors:** aging, increased stress, immunocompromised
- Recurrent disease can cause stromal scarring and vision loss

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CORNEAL ULCERS - INFECTIOUS

VIRAL

Management: VZV Ophthalmiscus

- Treat systemic infection with Acyclovir 800mg 5x/day OR valacyclovir 1000mg TID
- Cornea findings with frequent PF tears, ointment, steroids only needed if AC reaction

Management: HSV Keratitis

- **Cycloplegics if AC reaction**
- **Topical Antivirals:**
  - Trifluridine (1% drops) 9x/day or ganciclovir gel (0.15%) 5x/day are frequently used.
- **Oral Antivirals:**
  - Acyclovir 400mg 5x/day OR valacyclovir 500mg TID may be prescribed, especially if topical treatment is not tolerated or in cases of severe infection

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CORNEAL ULCERS - INFECTIOUS

Acanthamoeba Keratitis

Rare but serious; often misdiagnosed early

Presentation: Severe pain out of proportion to clinical findings, ring infiltrate, photophobia; worsens over a period of weeks

Risk Factors: Contact lens wear (especially with poor hygiene or swimming)

Management: Cornea specialist referral – Corneal scrapings or biopsy needed

Very complex treatments – not easily available

- Polyhexamethylene biguanide 0.02% (PHMB) drops q1h or chlorhexidine 0.02% drops q1h
- Often takes many weeks to month for treatment
- Need for eventual corneal transplant is not uncommon

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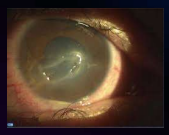
CORNEAL ULCERS – NON-INFECTIOUS

Neurotrophic Ulcer

Due to loss of corneal sensation (e.g., HSV, diabetes, CN V damage)

Presentation: Chronic or poor healing, persistent epithelial defect

Painless, non-healing ulcer



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CORNEAL ULCERS – NON-INFECTIOUS

Neurotrophic Ulcer Management:

- Stage 1 - Persistent punctate staining: Preservative-free artificial tears q2–4h and artificial tear ointment q.h.s.
- Stage 2 - Small corneal epithelial defect:
  - Antibiotic ointment (e.g., erythromycin or bacitracin q.i.d. to q1–2h) for 3 to 5 days or until resolved. Usually requires prolonged artificial tear treatment.

  Consider placement of a bandage contact lens with prophylactic antibiotic along with frequent preservative-free artificial tears (q1–2h) OR Amniotic membranes
- Stage 3 - Ulceration : antibiotic ointment q2h, amniotic membrane, Oral doxycycline 100 mg BID, Vitamin C 1 to 2 g daily may promote collagen synthesis and reduce the level of ulceration.
  - Tarsorrhaphy – temporary
  - Oxervate – Use if drop is 6x/day for 8 weeks

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CORNEAL ULCERS – NON-INFECTIOUS

PUK (Peripheral Ulcerative Keratitis)

Presentation: Crescentic peripheral thinning with adjacent scleritis

Management: look for underlying systemic etiology (History and Lab testing); **Requires systemic immunosuppression

- Associated with **rheumatoid arthritis**, granulomatosis with polyangiitis (GPA), SLE

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CORNEAL ULCERS – NON-INFECTIOUS

PUK (Peripheral Ulcerative Keratitis): Corneal Management

- Preservative free artificial tears and ointments and closure of puncta with plugs or cautry
- Topical cyclosporine to decrease inflammation.
- Oral doxycycline may be used due to its anti-collagenase activity along with Vitamin C
- Cyanoacrylate adhesive applied to the ulcer bed to limit ulceration with BCL (if impending perforation)
  - May also prevent influx of white blood cells from the tear film

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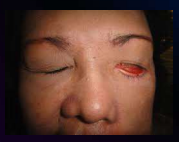
CORNEAL ULCERS – NON-INFECTIOUS

Exposure Keratopathy

Definition: Corneal breakdown due incomplete eyelid closure

- Leads to desiccation and epithelial breakdown, usually inferiorly



Management

- Frequent PF artificial tears, lubricating ointments, Moisture googles/taping/Tegaderm
- Treat the etiology if able (CN VII palsy, TED, lid positioning)
- Scleral lenses
- Surgical correction, when/if able
- Close follow-up

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TYPES OF CORNEAL ULCERS

Type	Pain	Onset	Features	Risk Factors
Bacterial	Moderate	Acute	Focal white infiltrate, discharge	Contact lens, trauma
Fungal	Mild	Subacute	Feathery edges, satellite lesions	Trauma with plant matter
Viral (HSV)	Mild	Recurrent	Dendritic pattern, ↓ sensation	History of HSV, immunosuppressed
Acanthamoeba	Severe	Chronic	Ring infiltrate, severe pain	Contact lens + water exposure
Neurotrophic	None	Chronic	Non-healing ulcer	CN V palsy, diabetes
PUK	Variable	Chronic	Peripheral thinning, scleritis	Autoimmune disease

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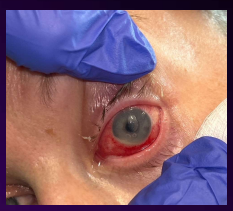
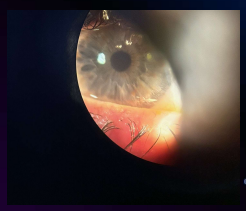
CASE #5 – CORNEAL ULCER(S)





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CASE #5A – CORNEAL ULCER(S)

Patient #1 – 50 yo female, in-patient x2 weeks, admitted due to complications from diffuse CNS lymphoma. Noted to have a red eye by nursing staff and realized patient had been wearing soft contact lens since admission.

Diagnosis: (See Photo)

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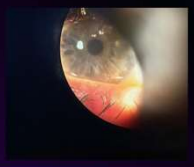
CASE #5B – CORNEAL ULCER(S)

Patient #2: 26 female, contact lens wearer, occasionally sleeps in lenses but had not in the past week or two, presents to triage clinic with intermittent irritation to her left eye for the past 1-2 days. Vision is stable but does note mild light sensitivity.

Diagnosis: (see photo)



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CASE #5 – CORNEAL ULCER

Treatment:

- Is this the same treatment for both cases?
- If not, how do you treat differently and why?

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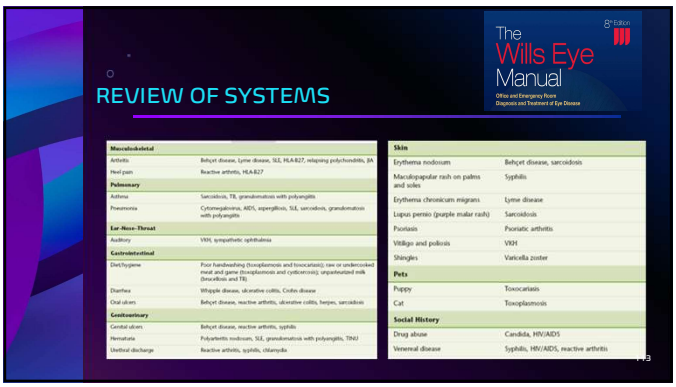

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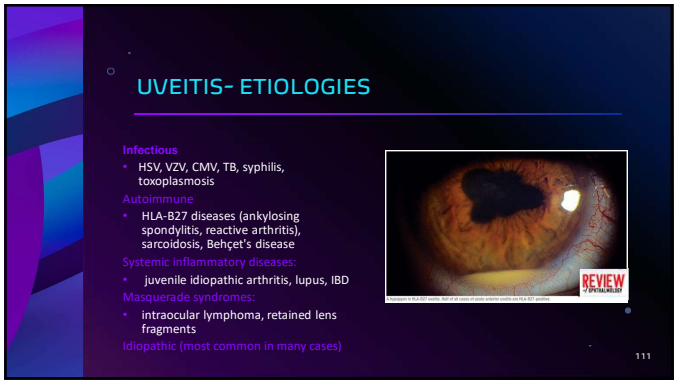

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UVEITIS

Complications

- Posterior synechiae: adhesions between iris and lens
- Cataract: from chronic inflammation or steroid use
- Glaucoma: due to inflammation or steroid response
- Macular edema: one of biggest threats to vision
- Retinal detachment: especially in severe posterior uveitis
- Optic nerve damage: from inflammation or elevated IOP
- Band keratopathy: calcium deposition in cornea

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CASE #6A

40 YEAR OLD MALE WITH HISTORY OF CTL WEAR PRESENTS TO TRIAGE CLINIC FOR 3 DAY FOLLOW UP FOR BLURRY VISION OD

WAS TOLD AT PREVIOUS EXAM THREE DAYS AGO THAT HIS BLURRY VISION WAS SECONDARY TO HIS REFRACTIVE ERROR AND FROM SWITCHING TO HIS GLASSES COMPARED TO HIS CONTACTS.

HERE FOR CTL EXAM TODAY.

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PHYSICAL EXAM

VISUAL ACUITY: OD: 20/70 OS: 20/20

EOMS: FROM OU CVF: FULL

PUPILS: PERRL (-)APD

SLIT LAMP EXAM:

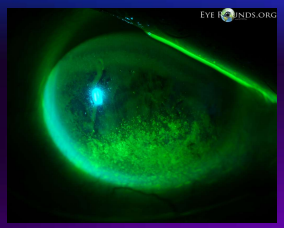
CORNEA:

STIPPLED APPEARANCE, DIFFUSE PEE ; CLEAR, NO GUTTAE OS

ANTERIOR CHAMBER:

DEEP AND 3+ CELL OD, DEEP AND 2+ CELL OS

FUNDUS: WNL OU



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ASSESSMENT/PLAN:

1. Anterior nongranulomatous bilateral uveitis
- first occurrence
- denies systemic issues, but given bilateral nature will order labs
- Cell grade: 3+ (26-50 per 1x1 field) OD and 2+ OS
- hesitant to start topical steroids given cornea involvement/issues, given suspicious of underlying Herpetic etiology will start acyclovir 400mg 5x a day and FU x 4 days prior to starting topical steroids
- started cyclopentolate 1% BID OU.

2. Diffuse PEE, right > left eyes
- ddx herpetic etiology give, #1
- will start treating with acyclovir prior to starting steroids as discussed above
- RTC on monday for re-evaluation and steroid initiation

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PHYSICAL EXAM

VISUAL ACUITY: OD: 20/40 OS: 20/20

EOMS: FROM OU CVF: FULL

PUPILS: PERRL (-)APD

SLIT LAMP EXAM:

CORNEA:

TR DIFFUSE PEE ; CLEAR, NO GUTTAE OS

ANTERIOR CHAMBER: TR CELL OD, TR CELL OS

FUNDUS: WNL OU

International Centre for Eye Health London School of Hygiene & Tropical Medicine

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4 DAY FU ASSESSMENT/PLAN:

1. Anterior nongranulomatous bilateral uveitis
- first occurrence
- denies systemic issues, labs unremarkable
- previous Cell grade: 3+ (26-50 per 1x1 field) OD and 2+ OS; now tr OU on Acyclovir alone so very suspicious of underlying herpetic etiology
- will go ahead and start Pred Q2H given improvement of cornea today, but strong precautions discussed
- Pred Q2H and continue Acyclovir 400mg 5/day

2. Diffuse PEE, right > left eyes
- ddx herpetic etiology give, #1- continue to believe this underlying etiology given normal labs and improvement on Acyclovir and AT alone
- continue Refresh and acyclovir
- RTC on Thursday, sooner with any worsening of symptoms

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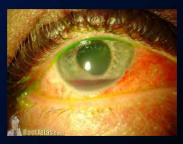
HYPHEMA



Definition: the accumulation and settling of red blood cells within the anterior chamber.

- Microhyphema - small amount of blood that is only evident under close
- 8-ball – full AC/Total hyphema

Etiology

- Blunt force or lacerating trauma
- After intraocular surgery
- Spontaneously in conditions such as rubeosis iridis, juvenile xanthogranuloma, iris melanoma, keratouveitis, leukemia, hemophilia,
- Use of substances that alter platelet or thrombin function (eg, ethanol, aspirin, warfarin)

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HYPHEMA

EVALUATION

- Must rule out an open globe!!
- Visual acuity
- Slit lamp exam - Height and color of the hyphema should be documented. Height can be measured in millimeters from the inferior corneal limbus
- Pupil evaluation – Presence of APD??
- IOP
- DFE (if possible)
- Consider CT orbits in concern for penetrating injury or orbital fracture

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HYPHEMA

Grading

- **Grade 0:** There is no visible layering, but there are red blood cells within the AC (microhyphema)
- **Grade I:** Layered blood occupies less than 1/3 of the anterior chamber
- **Grade II:** Blood fills 1/3 to 1/2 of the anterior chamber
- **Grade III:** Layered blood fills 1/2 to less than total of the anterior chamber
- **Grade IV:** Total filling of the anterior chamber with blood
  - Total hyphema – total AC fill of Red blood
  - 8-ball – total AC fill of Red-black blood
    - Black color is suggestive of impaired aqueous circulation and decreased oxygen concentration. (Increase risk of pupillary block and secondary angle closure)

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HYPHEMA

MANAGEMENT

- Very close follow-up – watch for rebleed
- Eye shield, limited activity, and head elevation (45 degrees)
- APAP use only
- Avoid ASA and NSAID use
- Assess Risk for sickle cell disease – obtain lab testing if any concerns

MEDICATION MANAGEMENT

Topical steroids (prednisolone 1% QID to q1h)

Cycloplegics (cyclopentolate 1% TID or Atropine BID)

In the setting of IOP elevation, topical aqueous suppressants are used 1st line

Acetazolamide or mannitol may be required if topical management fails to control the pressure.

Surgical intervention, if can't control

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HYPHEMA

SURGICAL MANAGEMENT

- Majority of hyphemas will resolve with medical management alone
- Approximately 5% of patients with a traumatic hyphema require surgery
- Sx required if uncontrolled glaucoma, corneal blood staining, the persistence of a large or total hyphema, and active bleeding in the anterior chamber
- AC washout with irrigation and aspiration is commonly performed, other procedures based on glaucoma, pupil block, etc

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CASE #7A

45 yo male presents to clinic for follow-up after being seen the day prior in a local ED, after being hit in the left eye with a piece of flying rock/concrete

VA sc OD 20/25, OS 20/40

Pupils – RRL – APD but sluggish OS

EOMS – full OU

IOP (app) 16/15

Anterior Segment: see photo

Posterior segment: unremarkable

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CASE #7A

Treatment

- Start topical prednisolone 1% 6x/day OS
- Start cyclopentolate 1% TID OS
- Clear fox shield wear
- Head elevated 45 degrees when sleeping
- No IOP drops required at this time
- Follow-up every 1-2 days

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CASE #7B

63 yo female presents to triage clinic with sudden onset tiny brown floaters making vision blurry in the right eye. No triggering factors, just noticed after getting out of shower in the AM

VA sc OD 20/40-1, OS 20/25

Pupils – RRL – APD but OD (see photo)

EOMS – full OU

IOP (app) 18/18

Anterior Segment: see photo, mild NS both eyes, otherwise unremarkable

Posterior segment: unremarkable

MHx: HTN, no history of other cardiovascular disease, no Diabetes, no history of ocular disease or Sx

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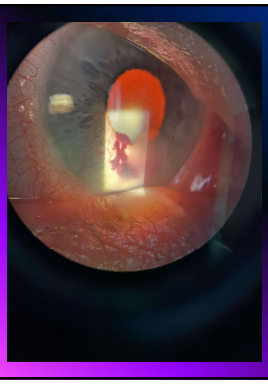

CASE #7B

Diagnosis?

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CASE #7B

Diagnosis: Iris capillary hemangioma with spontaneous hyphema

Treatment:

- Cyclopentolate 1% TID - attempt to immobilize iris movement
- Topical Prednisolone drops q4 hours
- Head elevated at 45 degrees, minimal activity, avoid ASA/NSAIDs
- Once bleeding is stable – consider Nd:YAG laser photocoagulation, if rebleeds or difficult to control bleeding

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CORNEAL ABRASIONS - DDX

Exposure Keratopathy
Corneal ulcer
Recurrent Corneal Erosion
Neurotrophic Keratopathy
Corneal Foreign Body
Chemical burn
UV Burn (Photokeratitis)
Herpes Simplex Keratitis

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CORNEAL ABRASIONS

Definition:
- Scratch/scrape on corneal epithelium from non-penetrating trauma

Etiology
- Superficial trauma (fingernails, branches, foreign bodies, makeup brushes, paper, sports equipment). Contact lens misuse.

Clinical Features
- Sudden onset: foreign body sensation, pain (can be severe), redness, tearing, blurry/hazy vision, photophobia, excessive squinting. History of trauma.

Treatment
- Topical antibiotics (erythromycin, fluoroquinolone/aminoglycoside for contact lens wearers). Cycloplegics for photophobia (e.g., cyclopentolate). Lubricating eye drops/ointments. Oral/topical NSAIDs for pain; Bandage contact lens (optional).

Clues for differentiation
- Epi-defect without underlying infiltrate; known trauma

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EXPOSURE KERATOPATHY

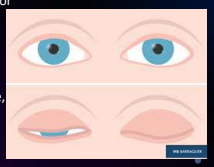
Definition:
- Damage to the cornea from incomplete eyelid closure or reduced blinking.

Etiology
- Facial nerve palsy, lagophthalmos, sedation, exophthalmos

Clinical Features
- Punctate epithelial defects inferiorly, dry corneal surface, redness, foreign body sensation

Treatment
- Lubrication (artificial tears/ointments), moisture chambers, taping lids, tarsorrhaphy if severe.

Clues for differentiation
- Inferior staining, lagophthalmos or Bell's



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RECURRENT CORNEAL EROSION

Definition:
- Repeated breakdown of the corneal epithelium due to poor epithelial adhesion.

Etiology

Previous trauma or corneal dystrophy (e.g., EBMD).

Clinical Features
- Sudden onset pain upon waking, tearing, photophobia, recurrent episodes.

Treatment
- cyclo BID-TID, Erythromycin ung QID, consider hypertonic saline ointment(5% sodium chloride), PFAT, bandage contact lens, epithelial debridement, anterior stromal puncture.

Clues for differentiation
- Pain on waking, normal findings between episodes

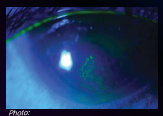

Photo: Rebecca Royes, OD

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PHOTOKERATITIS (UV BURN)

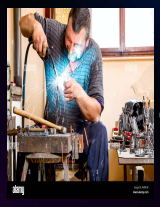
Definition:
- Corneal epithelial damage due to excessive UV light exposure.

Etiology
- Welding without eye protection, sunlamp exposure, snow blindness.

Clinical Features
- Severe eye pain, photophobia, redness, tearing, usually bilateral.

Treatment
- Lubrication, cold compresses, topical NSAIDs, oral analgesics; avoid anesthetic drops.

Clues for differentiation
- Bilateral pain, delayed onset, punctate staining



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NEUROTROPHIC KERATITIS

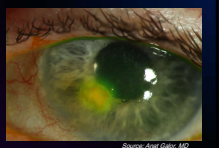
Definition:
- Degenerative disease of the cornea caused by impaired trigeminal innervation which leads to decreased corneal sensation resulting in poor epithelial healing and risk of corneal ulceration

Etiology
- Herpes Simplex or Zoster, DM, neurosurgical procedures (involving trigeminal nerve), intracranial tumors (acoustic neuromas), chronic ctl wear, long-term medication toxicity, corneal surgery

Clinical Features
- Decreased or absent corneal sensitivity, non-healing epi defects, minimal symptoms despite significant damage

Treatment
- Manage underlying causes, D/C toxic drops, PFATs, Therapeutic contact lens or amniotic membrane, Autologous serum eye drops, Tarsorrhaphy for non-healing ulcers

Clues for differentiation
- Lack of pain despite large epithelial defects, Poor healing even with standard therapy, Reduced corneal sensation on testing, Minimal injection compared to severity, Often no trauma history, unlike mechanical abrasions



Source: Anat Galor, MD

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CASE #8A

22YO MALE WITH HX OF BILATERAL VESTIBULAR SCHWANNOMA , BELLS PALSY OS, COMPLAINS OF NEW RIGHT SIDED FACIAL NUMBNESS AND DROOPING.

SEEN IN THE ED 5/2/22 WITH RIGHT SIDED FACIAL NUMBNESS AND DROOP, DIAGNOSED WITH RIGHT SIDED BELLS PALSY.

HE HAS BEEN USING ERYTHROMYCIN OINTMENT OS. USED A COUPLE TIMES OD BUT STOPPED DUE TO BURNING. HE DENIES USE OF OTHER DROPS.

MOTHER REPORTS NO OTHER NEW CONCERNS AT THIS TIME, JUST WANTED TO GET RIGHT EYE CHECKED OUT.

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PHYSICAL EXAM

VISUAL ACUITY: OD: 20/30 OS: 20/40

EOMS: FROM OU CVF: FULL

PUPILS: PERRL (-)APD

CN5: DECREASED SENSATION V1-V3 BILATERALLY

CN7: BILATERAL FACIAL DROOP WITH NEAR COMPLETE WEAKNESS OF FACIAL MUSCLES BILATERALLY

SLIT LAMP EXAM:

LIDS/LASHES: NORMAL OD, 5 MM SCLERAL SHOW OD AND 2MM OS WITH GOOD BELLS OU WITH INCOMPLETE CORNEAL COVERAGE OD ON BLINK

CORNEA: MODERATE INFERIOR PEE OU, OD>OS

CONJUNCTIVA: MODERATE INJECTION OU

AC: DEEP AND QUIET OU

IRIS: NORMAL, (-)NVI OU

LENS: OU: CLEAR

(05/02/2022 20:43 CDT MRI Brain)

1. No evidence of significant intracranial abnormality.
2. Stable postsurgical changes of right translabyrinthine resection with similar small residual irregular enhancement of the right CPA. A left CPA enhancing mass is unchanged.
3. Unchanged enhancement of the bilateral trigeminal nerves.
4. Tiny right tentorial meningioma.

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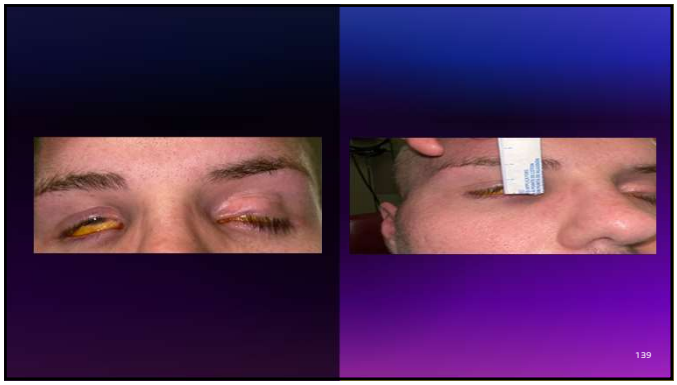

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CORNEA ASSESSMENT/PLAN:

1. Exposure keratopathy, both eyes (OD>OS)
2. Neurotrophic Keratitis OU
- No sensation to cotton wisp on exam previously. Poor/absent response today OU (OD worse than left. Assessing on blink response. Patient deaf.)
- no dendrites seen on exam today
- Increase PFATs to TID OU
- Continue nightly PF ung OU
- Buy moisture chamber to use at night while sleeping

RTC: as needed with cornea; as scheduled with ENT and oculoplastics

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ASSESSMENT/PLAN:

1. Neurofibromatosis Type II
2. Cranial Nerve V & VII paresis, right side
   - Almost no facial movement of right and left face. No sensation to touch of right and left side of the face in V1-V3 distributions.
   - with some concern over cerebellopontine angle given acute involvement of both V and VII (chronic hearing loss on both right and left so unable to evaluate CN VIII), but per MRI no changes since last scan 03/2022
   - already scheduled with Dr. Carr next Tuesday, recommend keeping appointment; also recommend keeping ENT appointment in June
3. Exposure keratoconjunctivitis OD, OS
   - chronic OS, acute OD –

OD: New right sided facial numbness and facial droop that started around 4/28/2022; with 5mm lagophthalmos, moderate inferior PEE
   - Evaluated in the ED with unremarkable CT head, CTA head and neck
   - Start lubrication schedule - PF AT Q2hrs

OS: status post V VII nerve transfer and radiation vs. bells palsy, onset may 2020; s/p gold weight placement in the left upper eyelid on 08/14/2020
   - with moderate inferior PEE today - continue with current lubrication schedule at night--does not respond well to blurry vision with ointment given deafness during the day.
   - Continue moisture chamber at night as patient tolerates

RTC 2 months oculoplastics to re-eval potential need for gold weight

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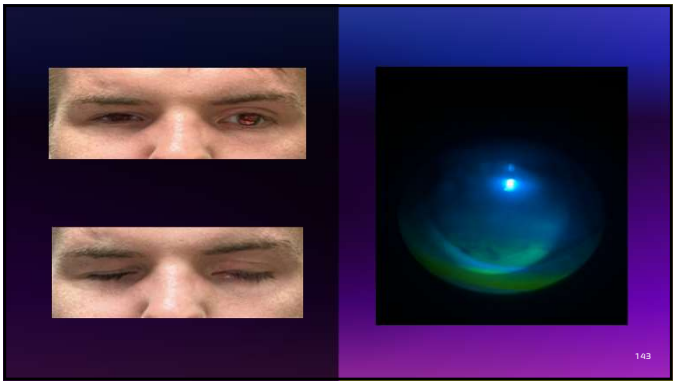

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OCULOPLASTICS ASSESSMENT/PLAN:

1. New onset R facial paresis, 4/28/2022
- With Paralytic lagophthalmos OD>OS and Exposure keratopathy -- weakness slowly improving
- evaluated by neurosurgery 5/2022, stable MRI without surgical disease
- Improvement of lagophthalmos OD and improvement of right side mouth movements since last visit
- Recommend monitor lagophthalmos for continued improvement, and if no improvement in 2 months when RTC, consider gold weight

2. Dendritic Keratitis OS
- Inferior dendritiform staining OS
- concern for HSV keratitis, no recent rashes or cold sores

3. Neurotrophic Keratitis OU
- recommend continued PFATs and ung as taking
- moisture goggle qhs if able

RTC within 2 weeks consult cornea re neurotrophic keratopathy and new dendrite OS & 2 months oculoplastics to re-eval potential need for gold weight

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CASE #8B

67F PRESENTS TO TRIAGE CLINIC WITH LEFT EYE PAIN AND FBS.

HAD LID LINER TATTOO DONE TODAY AND SYMPTOM ONSET WAS WITHIN 30 MINUTES OF PROCEDURE.

NO APPAREN EYE CONTACT DURING PROCEDURE, PER PATIENT, HOWEVER TOPICAL LIDOCAINE APPLIED TO LIDS.

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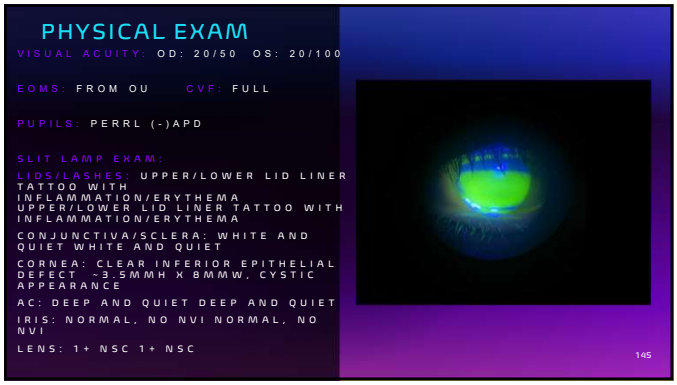

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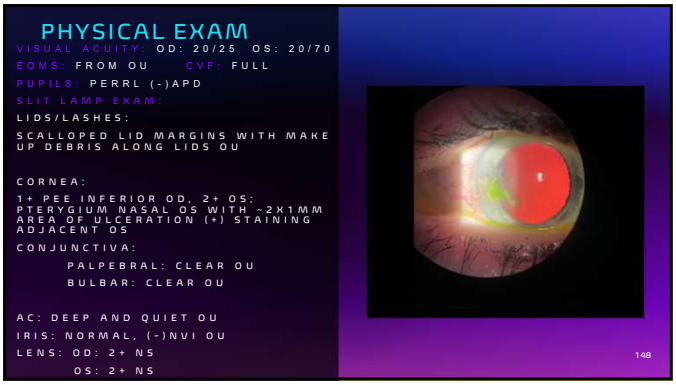

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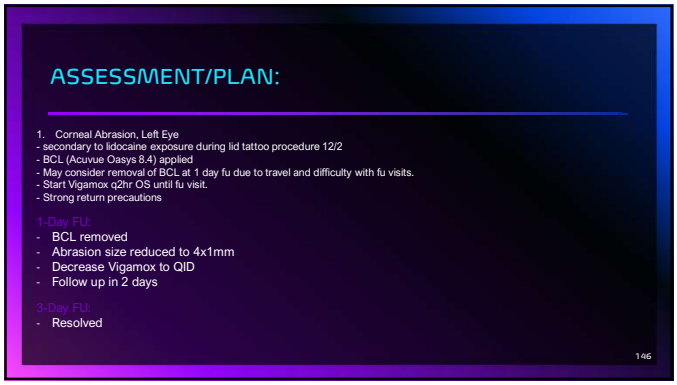

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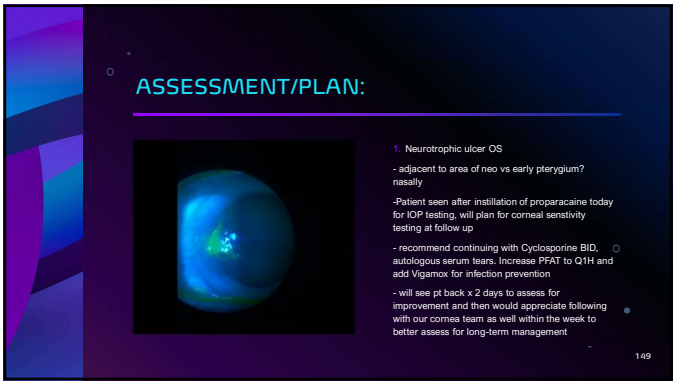

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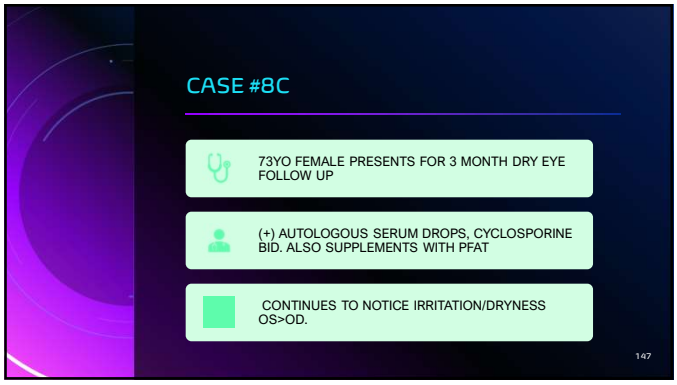

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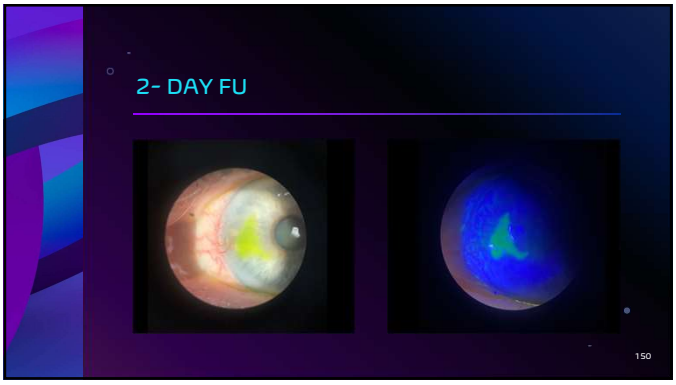

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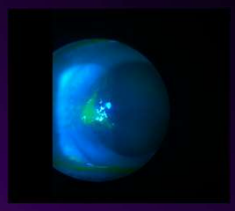
ASSESSMENT/PLAN:



1. Neurotrophic ulcer OS
- appears desensitized although reports equal sensation, however was able to easily tolerate viral swab without anesthetic
- adjacent to pterygium vs neo nasally (HSV?)
- recommend continuing with Cyclosporine BID, autologous serum tears. Increase PFAT to Q1H and add Vigamox for infection prevention
- Start acyclovir 500 mg TID due to lack of improvement with PFAT and antibiotic coverage as well as suspicion for HSV otherwise due to possible neo nasally
- Given lack of improvement x 2 days, would appreciate following with corneal service prior to the weekend

- History of LASIK of in 2021

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	Corneal Abrasion	Recurrent Corneal Erosion (RCE)	UV Keratopathy (Photokeratitis)	Exposure Keratopathy	Neurotrophic ulcer
Definition	Scratch/scrape on corneal epithelium from non-penetrating trauma	Repeated breakdown of corneal surface due to abnormal epithelial adhesion to basement membrane.	Corneal burn from acute UV radiation exposure, primarily damaging epithelium.	Corneal damage from prolonged exposure to external environment	Degenerative disease of the cornea caused by impaired trigeminal innervation which leads to decreased corneal sensation resulting in poor epithelial healing
Underlying Etiologies	Superficial trauma (fingernails, branches, foreign bodies, makeup brushes, paper, sports equipment). Contact lens misuse.	Previous trauma/abrasion (most common, 45-64% of cases). Corneal dystrophies (EMBD, Reis-Buckler, lattice, macular, granular, Fuchs endothelial) (19-29% of cases). Dry eye disease, diabetes, ocular surgery, Meibomian gland dysfunction, band keratopathy, nocturnal lagophthalmos	Artificial sources (arc welding, tanning beds, UV lamps, metal halide lights). Natural sources (snow blindness, solar eclipse viewing, prolonged sun exposure at high altitude).	Lagophthalmos (facial nerve palsy, eyelid dysfunction, cicatricial, nocturnal, medication-induced, ICU stay). Proptosis (thyroid eye disease, orbital tumors). Neurologic/neurotrophic diseases (impaired blink/sensation). Lid malposition (ectropion, entropion, coloboma). Other infectious keratitis, corneal thinning, sleep apnea, floppy eyelid syndrome.	Herpes Simplex or Zoster, DM, neurosurgical procedures (involving trigeminal nerve), intracranial tumors (acoustic neuromas), chronic cl wear, long-term medication toxicity, corneal surgery
Clinical Presentation and Symptoms	Sudden onset: foreign body sensation, pain (can be severe), redness, tearing, blurry/hazy vision, photophobia, excessive squinting. History of trauma.	Sudden onset of sharp pain, often upon awakening. Blurred vision, excessive tearing (lacrimation), photophobia, redness, foreign body sensation. Recurrent episodes.	Delayed onset (0.5-12 hrs post-exposure).Bilateral pain (often severe), foreign body sensation (gritty), marked photophobia, excessive tearing.	Blurred vision (may fluctuate), eye irritation, redness, dry eyes, foreign body sensation, burning, increased tearing. Symptoms worse in morning if nocturnal lagophthalmos. Pain and photophobia can be present.	Decreased or absent corneal sensitivity, non-healing epi defects, minimal symptoms despite significant damage
Slit Lamp Findings	Corneal defect staining with fluorescein (green under cobalt blue light). Traumatic: linear/geographic shapes. Contact lens–related: multiple punctate lesions coalescing into round, central defect. Vertical linear lesions (inner eyelid foreign body). Conjunctival injection, sub-edema, pupillary meiosis.	Epithelial microcysts, loose epithelium, or epithelial defects staining with fluorescein. "Negative staining" (non-staining lesions protruding through tear film, common in EMBD). Often bilateral/symmetrical (dystrophy) or corresponds to prior injury. Lower half of cornea most frequently affected.	Diffuse punctate epithelial erosions (SPK) staining with fluorescein. Punctate defects may coalesce in severe cases. Conjunctival injection, chemosis. Corneal swelling/edema, mild anterior chamber inflammation (cell/flare). Erythema of upper/lower eyelids.	Superficial punctate epithelial staining, most commonly inferior one-third. Punctate and macro epithelial erosion, stromal opaqueness (whitening) with epithelial defects, stromal scars (frequently in lower cornea). Incomplete blink, lagophthalmos, Conjunctival injection or chemosis.	Mild: Punctate epithelial erosions, dry, hzy cornea, reduced corneal sensation, decrease ear meniscus, mild conjunctival injection Modrate: persistent epithelial defect, no underlying stromal inflammation, staining Severe: corneal ulcer with stromal melting, perforation, hypopyon
Treatment Protocols	Topical anesthetics (for exam). Topical antibiotics (erythromycin, fluoroquinolone/aminoglycoside for contact lens wearers). Cycloplegics for photophobia (e.g., cyclopentolate). Lubricating eye drops/ointments. Oral/topical NSAIDs for pain; oral opioids for severe pain. Bandage contact lens (optional). Foreign body removal (swab, irrigation, needle). Avoid topical corticosteroids. Eye patching generally not recommended.	Conservative: Preservative-free artificial tears, nightly lubricating/hypertonic saline ointments (6-12 months). Topical antibiotics. Cycloplegics, temporary eye patching (acute). Oral pain relievers (ibuprofen). Oral doxycycline + topical corticosteroids (MMP-9 inhibitor). Topical cyclosporine 0.05%. Autologous serum drops. Bandage contact lens (with prophylactic antibiotics). Punctal occlusion. Surgical: Epithelial debridement, Anterior Stromal Punctura (ASP), Phototherapeutic Keratectomy (PTK), amniotic membrane transplantation.	Avoid further UV exposure. Supportive care: frequent artificial tears, lubricating eye drops/ointment (erythromycin) Pain management: oral NSAIDs (ibuprofen, acetaminophen), topical NSAIDs. Discontinue contact lens use (several weeks). Home remedies: rest with eyes closed, cold washcloth, sunglasses.	Address underlying cause. Interim/supportive: frequent artificial tears (preservative-free), nightly lubricating ointment (Lacri-lube). Punctal plugs. Eyelid closure promotion: passive closure, taping, patching, temporary tarsorrhaphy, moisture chambers goggles, Bandage/scleral contact lenses (with prophylactic antibiotics). Surgical (for refractory cases/underlying pathophysiology): temporary/permanent tarsorrhaphy, gold weight implantation, eyelid reconstruction, orbital decompression, amniotic membrane	Manage underlying causes, D/C toxic drops, PFATs, Therapeutic contact lens or amniotic membrane, Autologous serum eye drops, Tarsorrhaphy for non-healing ulcers

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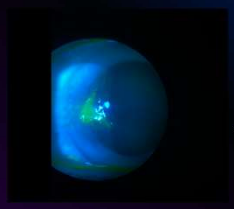
CORNEA CLINIC: ASSESSMENT/PLAN:

1. Neurotrophic ulcer OS
- possibly neurotrophic due to prior Lasik vs herpetic
- ulceration appears sterile today without infiltrate
- Recommend starting lubricating eye ointment Q1h OS while awake
- Decrease moxifloxacin to BID OS
- Decrease acyclovir to 500mg BID

RTC 1 week for follow up of neurotrophic ulcer OS



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OPTIC DISC EDEMA – "SWOLLEN OPTIC NERVE(S)"

- Unilateral vs. Bilateral?
- **True Papilledema** is bilateral and due to raised intracranial pressure vs **optic disc edema** can be from variety of other causes (unilateral or bilateral)
- Case history matters

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CORNEA CLINIC: 1 WEEK FU

1. Neurotrophic ulcer OS
- improved
- possibly neurotrophic due to prior Lasik vs VZV
- ulceration appears sterile today without infiltrate
- Decrease lubricating ointment use to nightly OS
- Stop moxifloxacin drops OS
- Continue acyclovir to 500mg BID

RTC 3 months

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UNILATERAL DISC EDEMA - DDX

Optic Neuritis
- Demyelinating (e.g., multiple sclerosis)
- Infectious (e.g., syphilis, Lyme disease)
- Parainfectious (post-viral)

Ischemic Optic Neuropathy
- Anterior ischemic optic neuropathy (AION)
  - Non-arteritic (NAION)
  - Arteritic (usually due to giant cell arteritis)

Compressive Optic Neuropathy
- Tumors (e.g., meningioma, optic glioma)
- Orbital mass or lesion

Trauma - Orbital or optic nerve trauma

Inflammatory and Autoimmune Disorders
- Sarcoidosis
- Lupus
- GPA

Infectious Optic Neuropathy
- Herpes zoster
- Tuberculosis
- Toxoplasmosis

Infiltrative Diseases
- Leukemia
- Lymphoma

Papillophlebitis

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BILATERAL DISC EDEMA - DDX

Papilledema (due to raised intracranial pressure)
- Brain tumor or abscess
- Hydrocephalus
- IIH
- Cerebral venous sinus thrombosis
- Meningitis or encephalitis

Toxic/Nutritional Optic Neuropathy
- Methanol
- Ethambutol
- Vitamin B12 deficiency
- Tobacco-alcohol related

Malignant Hypertension

Infiltrative or Inflammatory Conditions
- Sarcoidosis
- Neurosyphilis
- Tuberculosis
- Autoimmune - NMO/MOG (can be Unilateral or Bilateral)

Hereditary Optic Neuropathies
- Leber's Hereditary Optic Neuropathy (LHON)
- Dominant optic atrophy

Pseudopapilledema
- Optic disc drusen

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OPTIC DISC EDEMA

EXAMINATION: Obtain Detailed Case History
- Recent headaches, changes in vision, transient visual disturbances (TVOs), pulsatile tinnitus, and symptoms suggestive of increased intracranial pressure (ICP).
- Review medical history for autoimmune diseases, cancer, recent infections (e.g., tick bites, rashes), and inflammatory conditions like giant cell arteritis (GCA).
- Obtain a comprehensive list of medications, including prescription drugs, over-the-counter medications, supplements, topical applications, and recently discontinued agents.

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OPTIC DISC EDEMA

EXAMINATION: Physical exam
- Measure blood pressure, to rule out malignant hypertension
- Assess visual acuity, color vision, motility, and pupils (RAPD)
- Anterior segment exam and IOP
- Visual field testing, OCT, Fundus photos (if avaliable)
- DFE - evaluating the optic disc for elevation, blurring of margins, hyperemia, presence of hemorrhages, exudates, or CWS
- Conduct a thorough neurological examination to assess for other focal neurological deficits
  - TEST Cranial nerves!!

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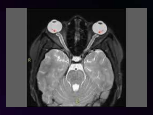
OPTIC DISC EDEMA

Imaging Studies
- An MRI of the brain and orbits with and without contrast, including fat suppression sequences, is the preferred study to rule out intracranial masses, tumors, or inflammation affecting the optic nerves
- MRV (Magnetic Resonance Venography):
  - Recommended in suspected cases of papilledema to evaluate the dural venous sinuses for thrombosis
- CT scan: May be used in acute settings or if MRI is contraindicated
- MRI/CT must be done prior to any lumbar puncture to rule out mass lesion



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OPTIC DISC EDEMA

Lumbar puncture (if indicated)
- If neuroimaging is normal, a lumbar puncture should be performed to measure the opening pressure and analyze the cerebrospinal fluid for signs o infection or inflammation (e.g., cell count, protein, glucose) - CSF analysis

Laboratory testing

Depending on the suspected etiology and if unilateral or bilateral involvement
- Inflammatory/Autoimmune: ESR, CRP, CBC, ANA, RF, ACE, Aquaporin-4 (for NMO), anti-MOG
- Infectious: Lyme titer, RPR/FTA-ABS (for syphilis), Bartonella, etc.
- Other: HbA1C (diabetes), serum folate, B12

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CASE #9: OPTIC DISC EDEMA

37 yo female, presents to triage clinic with almost a week of significantly diminished vision in left eye and floaters in the right eye.
- Reports headaches that has increased in intensity and frequency over the last few months. Headache is worse when she lays flat.
- Positive transient visual obscurations that have happened multiple times in the past week Wednesday. Occur sporadically, particularly when she bends down and stands back up, and noted in both eyes Left eye worse than right.
- Also does report "whooshing/whirling" sound left ear > right ear.
- Endorses >100 lb weight gain since the beginning of the pandemic.
- It has been >5 years since she has been to the doctor.

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CASE #9: OPTIC DISC EDEMA

Entering VA cc
OD 20/30
OS Hand motion

EOMS: full OD/OS
IOP (app) 14/16
Pupils: ERRL – APD
Visual fields: see formal HVF
Anterior segment: normal limits OU
Posterior segment: see photos

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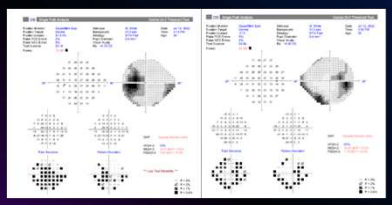
CASE #9: OPTIC DISC EDEMA



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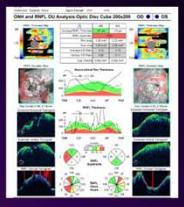
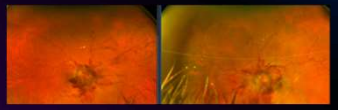
CASE #9: OPTIC DISC EDEMA





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CASE #9: OPTIC DISC EDEMA

What's next?

- If you work in an outpatient setting?
- If you work in a hospital based clinical setting?

- What testing should be ordered/requested?

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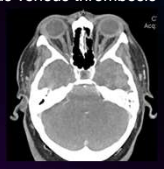
CASE #9: OPTIC DISC EDEMA

Additional testing:

- CT and CVA/CTA scan - partially empty sella turcica OU, posterior globe flattening, without cerebral mass noted; no dural sinus venous thrombosis (DSVT)
- LP - opening pressure 45



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CASE #9: OPTIC DISC EDEMA

DIAGNOSIS: IIH (Idiopathic Intracranial Hypertension)

- Idiopathic intracranial hypertension is a disorder producing symptoms and signs of increased intracranial pressure in the absence of an alternative cause
- Most common in child-bearing age females
- Obesity or more recent large weight gain
- Recent use of new medications
  - Vitamin A, birth control, steroids, Tetracyclines

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MANAGEMENT FOR IIH

- Weight loss – lifestyle, diet with goal of minimum of 10-15% of body weight
- Medications
  - Acetazolamide dosage is 250 mg BID, titrated according to benefits and tolerability (max of 4 g per day)
  - Topiramate - 25 mg once daily and increased by 25 mg every couple of weeks until achieving maintenance dose of 50 mg BID
  - GLP-1 agonist – facilitate weight loss but also show reduction in CSF reduction
- Surgical
  - **For vision threatening or s**evere/uncontrolled cases
    - Ventriculoperitoneal shunts to divert CSF
    - Transverse sinus stenting
    - Optic nerve sheath fenestration – while can decrease papilledema and improve CRVO, carries 1-2.6% risk of severe vision loss and has high failure rate

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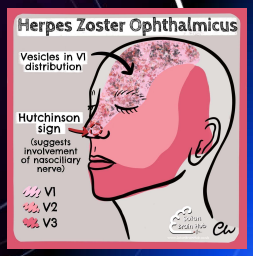
HERPES ZOSTER OPHTHALMICUS

ETIOLOGY

- REACTIVATION OF VARICELLA-ZOSTER VIRUS (VZV) IN THE OPHTHALMIC BRANCH (V1) OF THE TRIGEMINAL NERVE.

RISK FACTORS

- AGE >50 YEARS
- IMMUNOSUPPRESSED (CANCER, HIV, STEROIDS)
- PHYSICAL/EMOTIONAL STRESS



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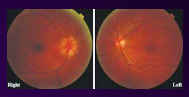
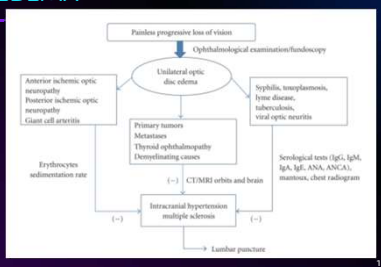
UNILATERAL DISC EDEMA





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CLINICAL FEATURES

Ocular Structure	Signs	Onset	Treatment
Adnexa	Cutaneous Rash with vesicular eruption swelling, ptosis, lagophthalmos	0 days	Palliative with cool compresses and topical lubrication. Surgical correction of Ptosis and Lagophthlamos may be indicated with chronic damage
Cornea	Punctate epithelial Keratitis and Pseudodendrites	1-6 days	Topical lubrications, Debridement or none
	Anterior Stromal Keratitis	1-2 weeks	Topical steroids
	Discform Keratitis	1 month- years	Topical steroids
	Neurotrophic Keratopathy	months- years	Topical lubrication, topical antibiotics for secondary infections
Iris	Iritis	2 weeks-years	Topical steroids, oral steroids, oral acyclovir
Conjunctiva, Episclera, Sclera	Papillary or follicular conjunctivitis	2-3 days	Palliative with cool compresses and topical lubrication, broad spectrum antibiotic for secondary bacterial infection
	Episcleritis/Scleritis	1 week	Topical NSAIDS and/or topical steroids
Retina	Acute Retinal Necrosis/Progressive outer retinal necrosis	varied	Intravenous antivirals for 7-10 days followed by oral antivirals for 14 weeks
Neuro-ophthalmic	Oculomotor nerve palsy	varied	Oral and/or intravenous antivirals and oral steroids
	Optic Neuritis	varied	Intravenous antivirals and oral steroids

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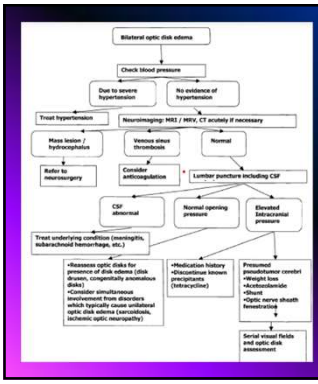
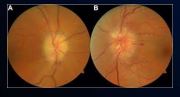


BILATERAL DISC EDEMA



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MANAGEMENT/TREATMENT

- Systemic antivirals (start within 72 hours):
  - Acyclovir 800 mg 5x/day OR Valacyclovir 1 g TID for 7–10 days.
- Ocular treatment:
  - Topical corticosteroids (if uveitis/stromal keratitis): four to eight times per day and adjusted according to clinical response.
  - Cycloplegics for pain/spasm and when uveitis is present
  - Ophthalmic antibiotic ointment (erythromycin) to skin lesions b.i.d or if SPK present
  - IOP lowering therapies if secondary IOP elevation
  - Admission with IV antivirals if concern over: Retinitis, choroiditis, optic neuritis, or cranial nerve pals
- Pain management:
  - Topical lidocaine, topical capsaicin
  - cool compresses
  - NSAIDs
  - Consider pain management specialist for gabapentin, opioids (if needed) and long-term management for postherpetic neuralgia.

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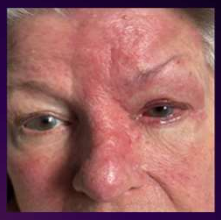
CASE #10A



86 Y/O FEMALE PRESENTED WITH A FEW DAY HISTORY OF MIGRAINE AND ONE DAY HISTORY OF LEFT FACIAL RASH AND LEFT EYE PAIN.

PATIENT HAS HAD A MIGRAINE HEADACHE THIS WEEK STARTING 2 DAYS AGO. SHE WENT TO THE DOCTOR WHO GAVE HER A SHOT FOR HER MIGRAINE WHICH DIDN'T HELP (THE SHOT TYPICALLY IMPROVES HER MIGRAINES).

PATIENT WORKED IN THE YARD YESTERDAY AROUND WEEDS. SHE WOKE UP THIS MORNING WITH A RASH ON THE LEFT SIDE OF THE FACE AND PAIN AND BLURRY VISION IN THE LEFT EYE.

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FOLLOW-UP VISITS.

Slow resolution of anterior uveitis over the course of 6 weeks and 4 visits:

At the time of resolution, the following plan was implemented:
- Stop topical cyclopentolate 1% BID to left eye
- Taper topical prednisolone 1% 6x daily for 1 week, QID daily for 1 week, BID x 1 week, then Q day x 1 week before D/C

Over her subsequent FU visits, she began having signs of post-herpetic neuralgia left side
- offered neurology consult for help in pain management, but pt prefers to start with her internist
- started on gabapentin 100 mg three times a day.

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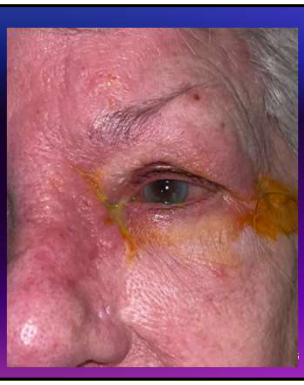
PHYSICAL EXAM

VISUAL ACUITY: OD: 20/20 OS: 20/100
EOMS: FROM OU CVF: FULL
PUPILS: PERRL (-)APD
IOP: 16/19
SLIT LAMP EXAM:
LIDS/LASHES: ERYTHEMATOUS RASH OF LEFT FOREHEAD AND LEFT SIDE OF NOSE (SPARING TIP OF NOSE)

CONJUNCTIVA/SCLERA: +2-3 CILIARY FLUSH OS
CORNEA: CLEAR OU

AC: DEEP AND QUIET DEEP AND QUIET OD, +2 AC CELL OS

IRIS: NORMAL, NO NVI NORMAL, NO NVI
LENS: PCIOL OU
FUNDUS: WNL OU



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QUESTIONS?

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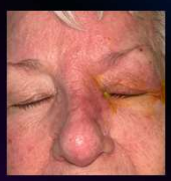
ASSESSMENT/PLAN:

1. Herpes Zoster Ophthalmicus with Anterior Uveitis, Left Eye
- onset of symptoms 4/24/25
- Topical cyclopentolate 1% BID to left eye
- Topical prednisolone 1% 6x/day to left eye
- Valtrex 1,000 mg TID for 7 days
- Erythromycin ointment BID to skin lesions as develop blistering
- Cool compresses TID to left periocular skin as needed



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THANK YOU!

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