April 1, 2024

INSIGHT

AU/UGA Medical Partnership
Ophthalmology SIG Primer

What's Inside?

The Ophthalmology Student Interest Group is creating a primer featuring common diseases and basic "know-hows" in ophthalmology. The purpose is to give students more exposure to ophthalmology, facilitating more interest and learning in the field. The primer will be updated bimonthly, on the first and third Monday of each month.

Contact us if you're interested in writing a piece!
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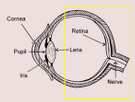
Central Retinal Artery Occlusion

By: Erik Rosas



Central Retinal Artery Occlusion (CRAO): A form of **acute ischemic stroke** resulting in compromised perfusion to the inner layers of the retina.

Epidemiology: Incidence of 1/100K. Avg. age of presentation >60 years.

Risk factors:

- **Similar to other thromboembolic diseases:** HTN, smoking, hyperlipidemia, diabetes, hyper-coagulable states, male gender.
- 33% of patients have clinically significant ipsilateral carotid artery stenosis.
- 5-10% of cases associated with giant cell arteritis.

Pathophysiology:

- Occlusion of the **central retinal artery** by embolism (#1), thrombus, vasculitis, or spasms.
- Acute ischemia leads to retinal edema and pyknosis (cell shrinkage) resulting in **pale** nerve fiber layer appearance.
- **Cherry-red spot** appears due to preserved choroidal circulation to the thin fovea.
- **Irreversible damage** to retina occurs **90 - 120 minutes** after occlusion.

Symptoms:

- Unilateral, painless vision loss occurring within seconds.
- History of amaurosis fugax (transient vision loss).
- VA is often dcreased to only light perception (more common) or count fingers.

Diagnosis: Clinical picture and confirmation with a dilated funduscopic exam or fundus photos.

- **Exam:** 80% of patients have visual acuity of **20/400** or worse. Afferent pupillary defect is common in the affected eye.
- **DFE:** Diffusely **pale retina** with a **cherry red spot**. Narrowing of arterioles (**box-carring**) due to slow segmental blood flow may also be appreciated.

Evaluation:

- **Initial blood work:** POC glucose, A1C, CBC, coagulation assays (PT/INR, PTT)
- **Rule out GCA:** ESR, CRP, and platelets. If history + labs are consistent with GCA then start high-dose steroids immediately.
- **Symptom onset < 6 hours:** CT head w/o contrast to rule out intracranial hemorrhage & determine candidacy for tPA.
- **Others based on individual history:** Carotid artery duplex, ecg, echo, OCT, IVFA.

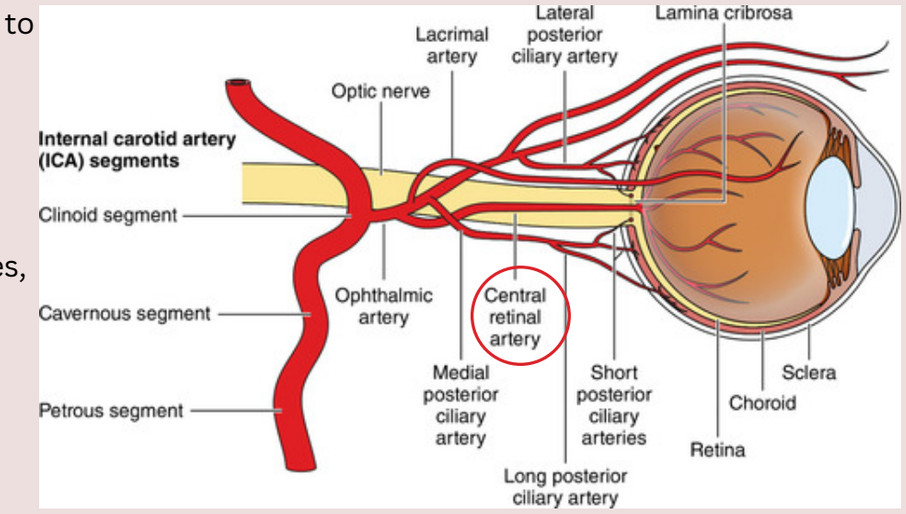
Treatment: **Treat like acute stroke and send the patient to the ED immediately!**

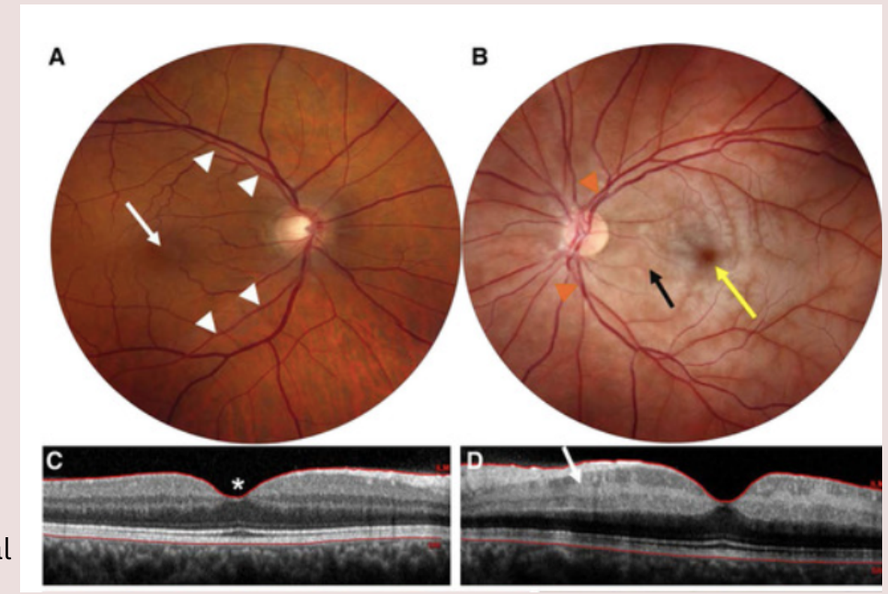
Goal: Preserve remaining sight + prevent occurrence in other eye.

- There is little consensus on the standard of care for CRAO. Mixed results in the current literature regarding tPA and conservative therapies.
- **Early administration of tPA (<4.5h from sx onset):**
  - As of 2021, the AHA recommends shared-decision making when considering the use of IV-tPA for treatment of CRAO. 3 European RCT are currently underway (as of 2024).
- **Conservative therapies:** Anterior chamber paracentesis (<24h) or ocular massage, hyperventilation, and supplemental oxygen. For patients who are not candidates for thrombolysis.
- **Stroke management:** Aspirin and clopidogrel if stroke is not caused by heart embolism.
- **Secondary prevention:** Manage co-morbid conditions such as HLD, DM, HTN, smoking & weight loss.

Prognosis: Vision loss is typically severe and correlated with the amount of retinal edema.

- Even with treatment, improvement in visual acuity is rare. **<20% regain functional visual acuity**.
- Associated with increased risk of recurrent vascular events.
- Life expectancy of patients with CRAO is 5.5 years.





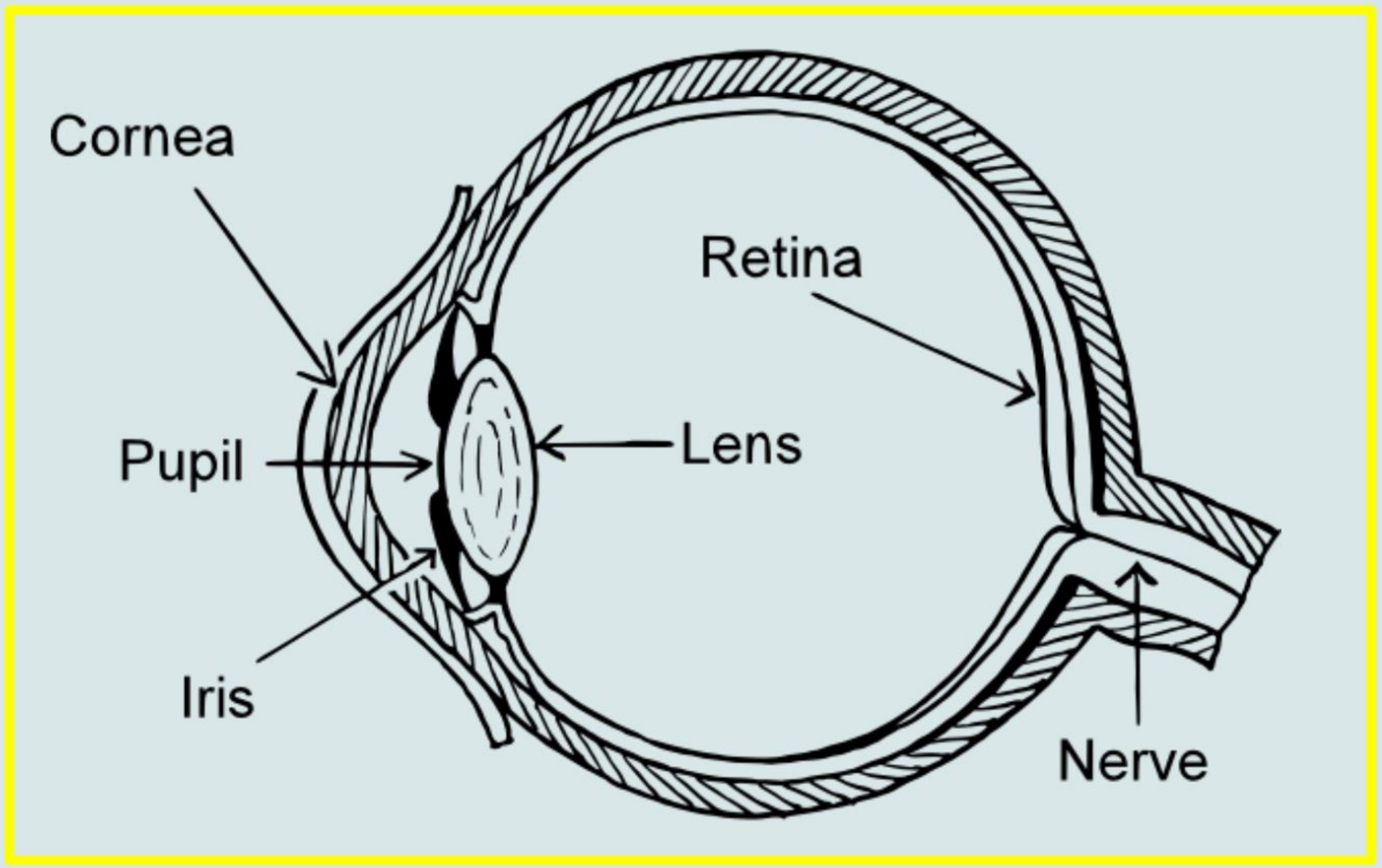
Examination findings in acute CRAO (image B). Note the pale fundal appearance (black arrow), cherry red spot (yellow arrow), and boxcarring vessels (orange triangle). Note acute retinal edema on OCT (image D).

Clinical Pearls:
The ipsilateral carotid artery is often the source of CRAO so patients need a carotid u/s.

Clinical Pearls:
12% of all strokes are preceded by TIA.

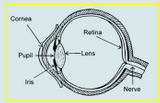
Clinical Pearls:
DDX of Cherry-Red Spot includes: CRAO, Tay-Sachs, Niemann-Pick, GM1 gangliosidosis, mucolipidosis.

FUNDAMENTALS



THE BASICS

By: Kara Ye



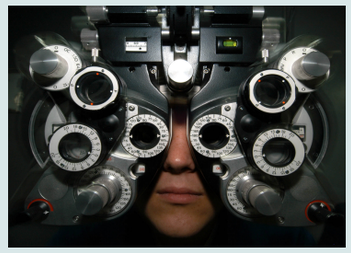
Commonly used abbreviations:

- AMD = age related macular degeneration
- APD = afferent pupillary defect
- CDR = cup to disc ratio
- DFE = Dilated fundus exam
- DR = diabetic retinopathy
- IOP = intraocular pressure
- OD = right eye
- OS = left eye
- PCO = posterior capsular opacity
- PCIOL = posterior chamber intraocular lens
- RNFL = retinal nerve fiber layer
- RPE = retinal pigment epithelium
- SLT = selective laser trabeculoplasty
- LPI = laser peripheral iridotomy
- VA =visual acuity

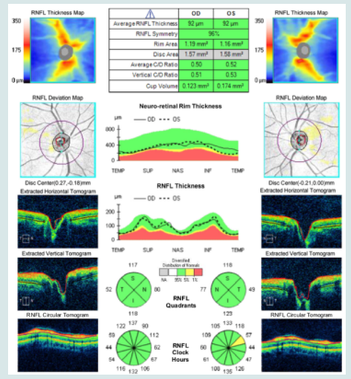
Normal ranges:

- IOP = 10-21 mmHg
- Corneal thickness = 540-560 micrometers
- CDR = 0.3
- Macula thickness = 350 micrometers (250 micrometers for fovea)

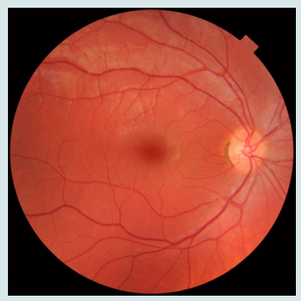
Commonly Used Tests:



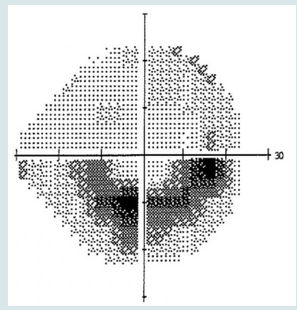
- Phoropter - used to correct refractive error and prescribe glasses prescription.



- Optical Coherence Tomography (OCT)- noninvasive test used to visualized the retina and provide treatment guidance for glaucoma and retinal disease (diabetic retinopathy, AMD, macular edema).



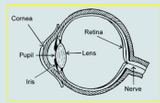
- Fundus Photography - noninvasive test used to take picture of the retina.



- Humphrey Visual Field (HVF) - noninvasive test used to map a person's visual field and assess vision loss, mainly in the evaluation and management of glaucoma.

EYE DROP COLOR GUIDE

By: Kara Ye



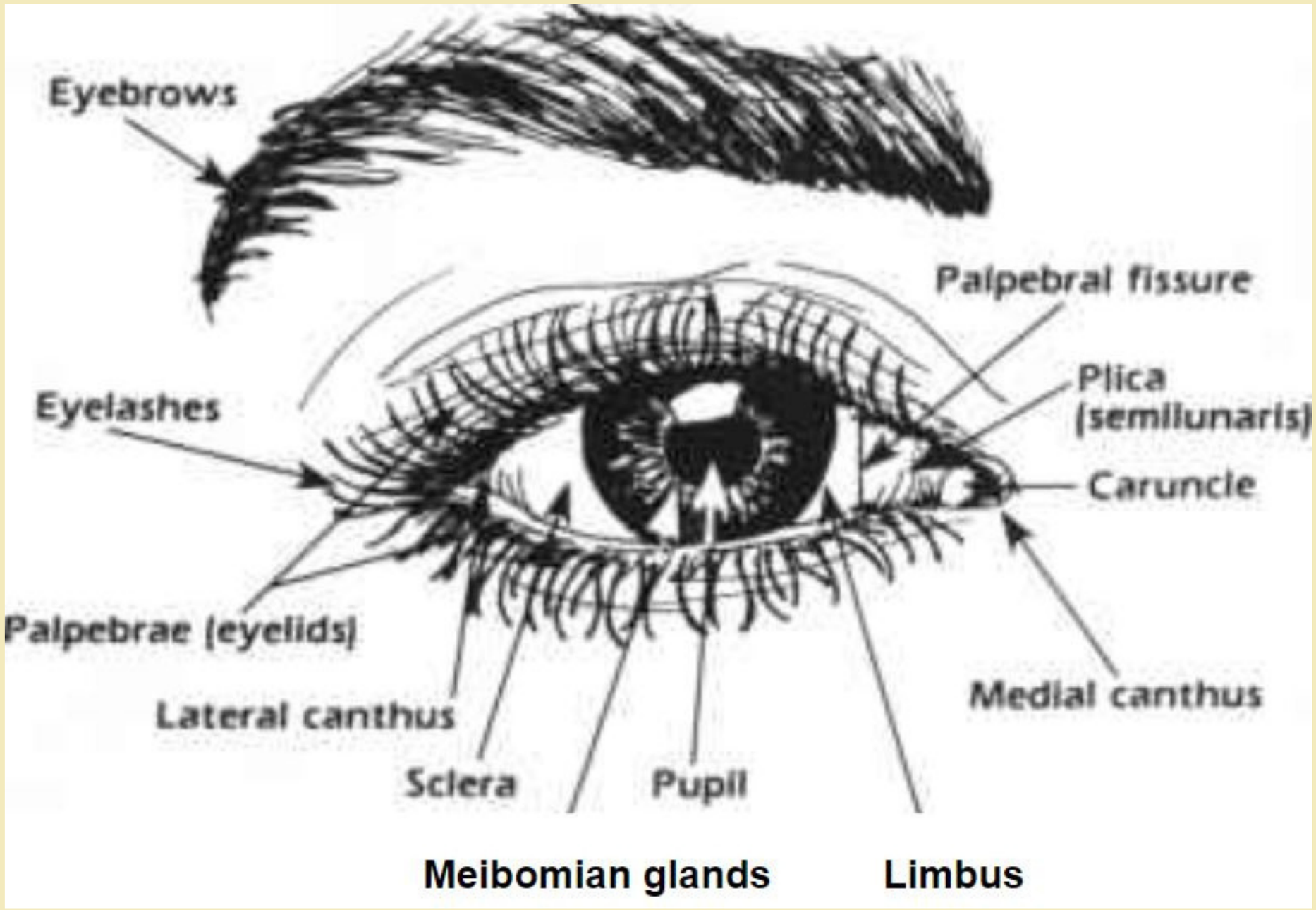
Commonly Used Drops			
Color	**Class**	**Examples**	**Uses**
Red	Dilating	Atropine (strongest, 7-10 day dilation), Cyclopentolate (24hrs), Tropicamide (6hrs)	Accommodative esotropia, amblyopia, blind painful eye, hyphema, uveitis, DFE
White, Clear, or Black	Antibiotic	Azithromycin (Azasite), Gentamicin, Tobramycin-Dexamethasone (Tobradex), Polymyxin B-Trimethoprim (Polytrim), Neomycin-Polymyxin (Neocidin), Neomycin-Polymyxin B-Dexamethasone (Maxitrol), Bacitracin is black	Superficial bacterial ocular infection, prophylaxis
Tan/ Brown	Antibiotic	Fluoroquinolones (Gatifloxacin, Levofloxacin, Ciprofloxacin, Ofloxacin, Moxifloxacin)	Bacterial conjunctivitis, corneal ulcer
Gray	NSAID	Ketorolac, Diclofenac, Prolensa, Ilevro	Intraocular inflammation, macular edema
Pink or White	Steroid	Prednisolone Acetate (Pred Forte), Prednisolone Phosphate, FML, Durezol, Dexamethasone, Loteprednol-Lotemax	Intraocular inflammation, macular edema
Dark Green	Cholinergic	Pilocarpine, Carbachol	AACG, intraoperative miosis

IOP Lowering Drops			
Color	**Class**	**Examples**	**Uses**
Teal	Prostaglandin analog	Latanoprost (Xalatan), Bimatoprost (Lumigan), Travoprost (Travatan Z), Tafluprost (Zioptan), Vyzulta	Once a night (QHS) *Tafluprost is preservative free
Orange	Carbonic anhydrase Inhibitor	Dorzolamide (Trusopt), Brinzolamide (Azopt), Acetazolamide	TID
Yellow	Beta blocker	Timolol (Timoptic), Betimol, Istalol)	BID
Purple	Alpha agonist	Brimonidine (Alphagan P)	TID
Dark Blue	Combos with timolol	Combigan (brimonidine + timolol), Cosopt (dorzolamide+timolol)	BID
Light Green	alpha-agonist combo	Simbrinza (brimonidine + brinzolamide)	TID
White	Rho kinase inhibitor	Netarsudil (Rhopressa)	QHS

Typically the teal drop is the first drop to be started (or SLT if patient elects laser procedure). If the IOP remains uncontrolled, step up the therapy by adding an orange, yellow, or purple drop. The combo drops can be used for patient convenience and compliance if on multiple drops. Rhopressa is usually added last because it is very expensive.

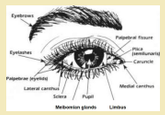
Gels/ointments	
Muro sodium chloride	Used to treat corneal edema
Erythromycin, Neomycin-Polymyxin B-Bacitracin (Neosporin), Bacitracin-Polymyxin, Tobramycin-Dexamethasone (TobraDex), Neomycin-Polymyxin B-Dexamethasone (Maxitrol), Timethoprim-polymyxin B (Polytrim)	Used to treat inflammation and superficial bacterial ocular infections (ex: blepharitis)

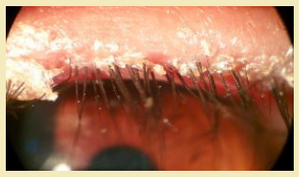
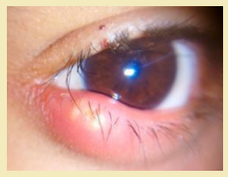
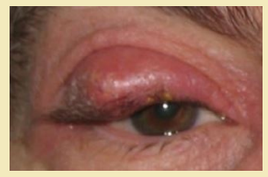
OCULAR ADNEXA



EYELID HYGIENE

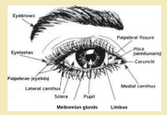
By: Kara Ye, Sarah Ye

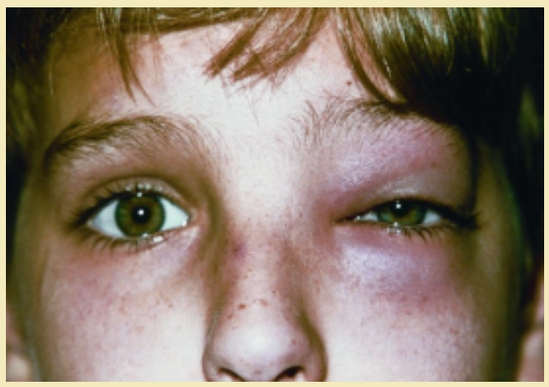


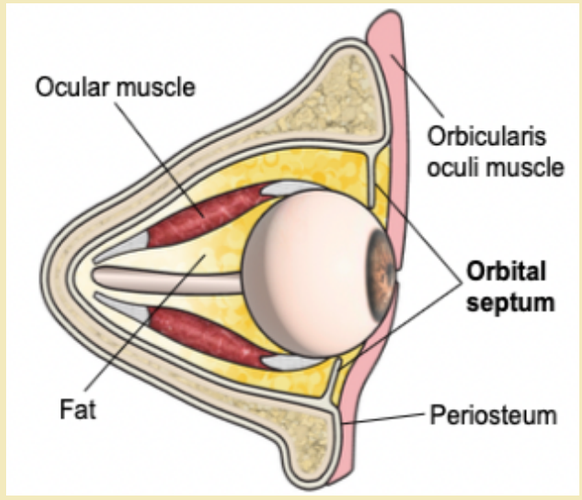
Common Eyelid Conditions			
	Blepharitis	**Hordeolum/Stye**	**Chalazion**
Causes	• Inflammatory condition of eyelid margin • Unclear etiology. May be due to bacteria (***staph aureus***), mites, or hormonal changes • **Anterior blepharitis**: affects eyelid skin, base of eyelashes, eyelash follicles • **Posterior blepharitis**: meibomian gland	• Acute inflammation of oil glands of the eyelid due to **bacteria** or mites (demodex) ◦ Like acne on your eyelid • **External**: originates from glands of Zeiss or Moll • **Internal**: originates from meibomian gland	• Chronic **sterile** obstruction of meibomian gland in tarsal plate, lipogranuloma development
Image	• Scales of anterior blepharitis 	• External hordeolum 	• Chalazion 
Risk Factors	• Dry eye • Postmenopausal female (low estrogen decreases tear production) • Acne rosacea • Demodicosis • Old age	• Chronic blepharitis • ***Staph aureus*** infection • Previous styes • Poor diet • Stress	• Blepharitis • Rosacea • Prior chalazion • Meibomian gland dysfunction • Demodex • Smoking • Bortezomib
Symptoms + Slit Lamp Exam	• Eyelid redness, burning, and irritation • Tearing • Eyelid crusting and sticking • Eyelash sticking together ("tepee sign") 	• **Tender** to the touch • Red swollen nodule at eyelashes or mid eyelid • Eyelid edema • May have multiple at one time	• Typically **painless** solitary eyelid nodule • Evert eyelid for better visualization
Treatment **eye drops color coded according to Eye Drop Color Guide (p. 7)*	• **Eyelid hygiene** (warm compresses for 5 minutes BID-TID, lid scrubs BID, remove face makeup daily) • Antibiotics: oral doxycycline, erythromycin, or bacitracin ointment • Steroid drops: **FML**, **loteprednol etabonate** • Steroid-antibiotic drops: **tobradex**, **maxitrol**, **neomycin-polymyxin**, etc. • Lipiflow for Meibomian gland dysfunction	• **Eyelid hygiene** • Antibiotics: erythromycin or bacitracin ointment • Monitor for progression to periorbital cellulitis (rare but serious) • Recurrence is common if eyelid hygiene is not maintained	• Mild: **Eyelid hygiene** • Severe: Steroid injection (triamcinolone), oral doxycycline, erythromycin ointment, or steroid-antibiotic drops • Persistent: incision and drainage
Clinical Pearls	• Patients can make their own warm compresses by microwaving a sock full of dry rice for 10 seconds • Do not reuse lid scrubs • Sebaceous carcinoma or sebaceous hyperplasia can appear similar to a chalazion. Make sure to consider this in persistent chalazions		

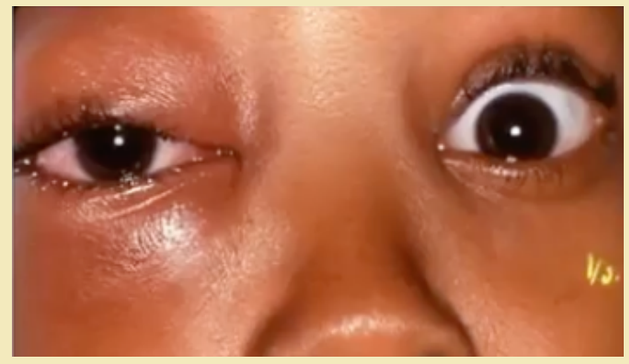
Preseptal vs Orbital Cellulitis

By: Kara Ye, Sarah Ye

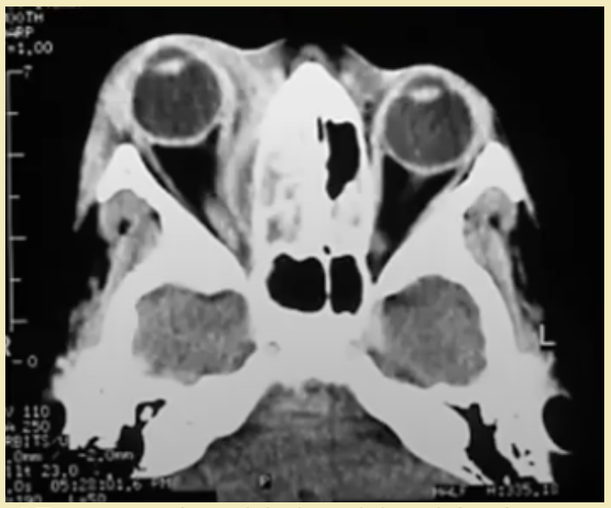


	Preseptal Cellulitis: Acute infection **anterior** to the orbital septum.	**Orbital Cellulitis (aka postseptal)**: Acute infection **posterior** to the orbital septum.
Cause	• Scratch or insect bite near eye leading to skin infection • Commonly **gram + bacteria** (**Staph**. and **Strep**. species)  *Preseptal cellulitis OS*	• 90% due to adjacent sinusitis (commonly ethmoid). • the orbit is surrounded by sinuses so infection can easily spread! • Infectious spread from other nearby sites (dacryocystitis, dental/ear/face infection, endophthalmitis, periorbital cellulitis). Bacteremia with septic emboli. • Children <9: **gram + bacteria** (**Staph**. and **Strep**. species) • Children >9 and adults: polymicrobial (aerobes + anaerobes) • Immunocompromised and diabetic pts: polymicrobial, fungal (mucor and **aspergillus**)
Symptoms/ Exam Findings	• +/-Fever • Moderate lid edema/erythema • Preserved EOM • NO proptosis • Moderate chemosis • Preserved VA • Preserved pupils	• **Fever** and leukocytosis • Marked lid edema/erythema • **Painful and restricted EOM** • **Ophthalmoplegia with diplopia** • **Proptosis** • **Chemosis** • **Reduced VA** • **Pupil defect with ON involvement** • **Black eschar suggests fungal infections**
Treatment	• Afebrile, Mild: oral amoxicillin-clavulanic (augmentin) 875mg BID or cephalexin (Keflex) 500mg BID x 7 days or azithromycin (z-pack) x 5 days. • Moderate/severe: IV cefuroxime or ceftriazone • Add TMP-SMX or vancomycin if MRSA risk factors are present • Pt needs to be sent to the hospital and get a CT scan if there is minimal improvement after 48 hour.	• Hospitalize patient and give IV antibiotics (triple therapy with vancomycin, ceftriaxone, metronidazole) b/c orbital cellulitis is commonly polymicrobial. Narrow the spectrum once blood cultures return. • Surgical drainage if abscess is present. • Monitor for complications: severe exposure keratopathy, ulcerative keratitis, secondary glaucoma, septic uveitis, exudative retinal detachment, optic neuropathy, panophthalmitis, cranial nerve palsies, CRAO, CRVO, cavernous sinus thrombosis, meningitis, brain abscess, death.





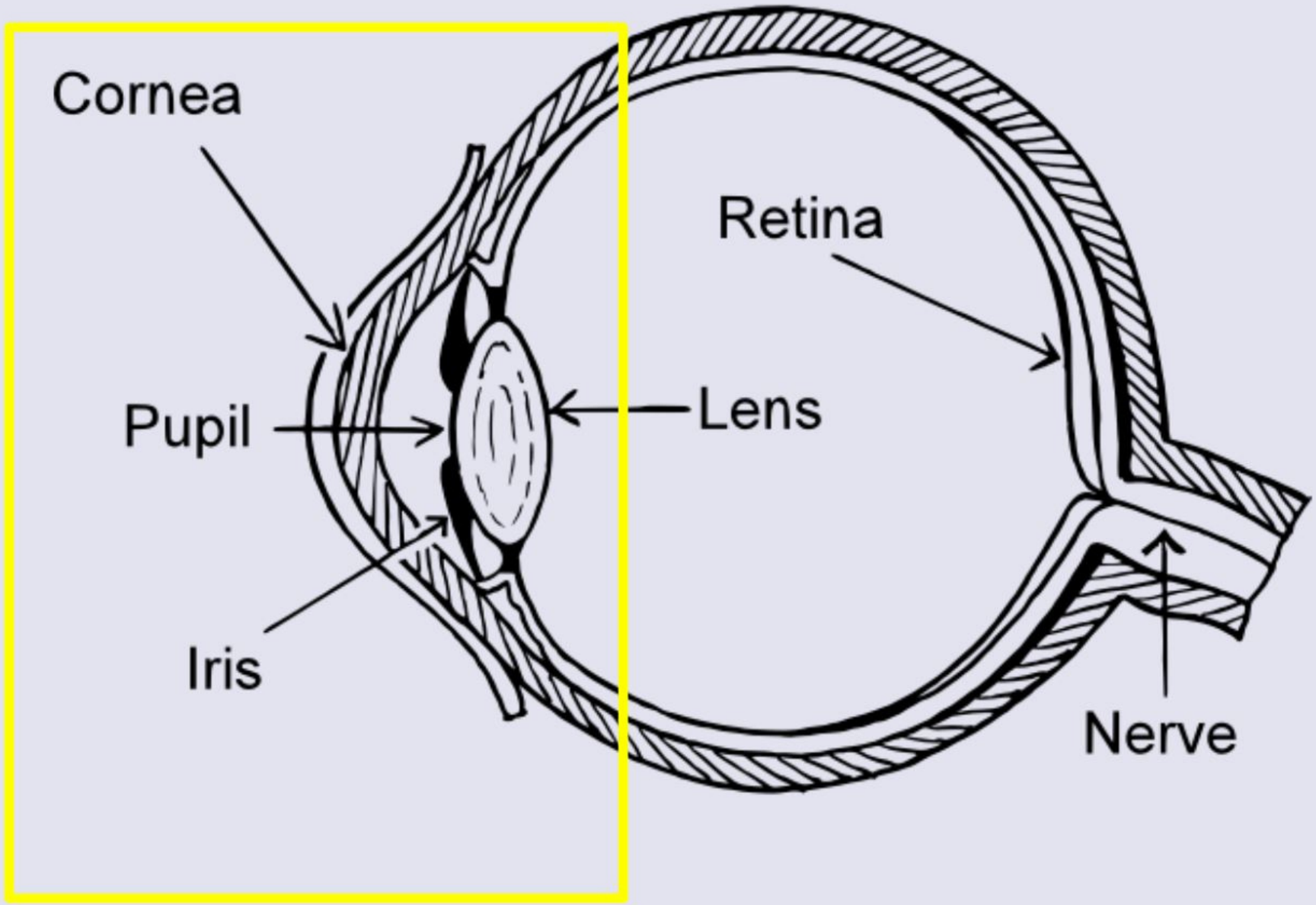
Orbital cellulitis OD



CT scan: ethmoid sinusitis with abscess leading to orbital cellulitis OD

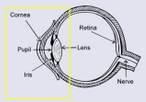
Clinical Pearls:
Preseptal cellulitis seldom leads to serious complications; however, orbital cellulitis is vision and life-threatening. Suspect subperiosteal or orbital abscess (10% prevalence) if orbital cellulitis does not improve after 48 hours of IV antibiotics; a CT scan with contrast is needed to look for abscess formation.
Venous drainage of the eyelid and orbit goes to the cavernous sinus so watch out for cavernous sinus thrombosis (90% mortality)!

ANTERIOR SEGMENT



Primary Open Angle Glaucoma

By: Kara Ye



Glaucoma: Loss of retinal ganglion cells leads to thinning of the retinal nerve fiber layer (RNFL) and increased cup to disc ratio (CDR). There is progressive peripheral visual field loss to the point where patients develop tunnel vision. Glaucoma is associated with high intraocular pressure (IOP), but high IOP doesn't cause glaucoma (*low tension glaucoma exists!).

Types of Glaucoma: Primary open angle glaucoma (POAG), secondary glaucoma (neovascular, phacomorphic, traumatic, steroid induced, pseudoexfoliation syndrome), acute angle closure glaucoma (AACG), congenital glaucoma.

In POAG the angle (drainage channel) is open, but aqueous humor is being overproduced or not being drained by the trabecular meshwork.

Risk factors: FHx, African American, age > 60, myopia, diabetes, thin corneas, suspicious nerves, high IOP.

Workup: Obtain history for risk factors. Then perform pachymetry, gonioscopy, and IOP check with Goldmann applanation. Then DFE with fundus photos and OCT RNFL. Humphrey visual field tests (24-2) can be performed at a follow-up visit because those take time to complete.

Signs: Elevated IOP >21, Gonioscopy shows open angles and no peripheral anterior synechiae (PAS), loss of rim tissue on optic nerve, splinter hemorrhages that cross disc margins, cup/disc asymmetry >0.2, bayoneting of blood vessels, large CDR >0.6, characteristic visual field loss (nasal step, paracentral scotoma, arcuate scotoma that extends from blind spot nasally).

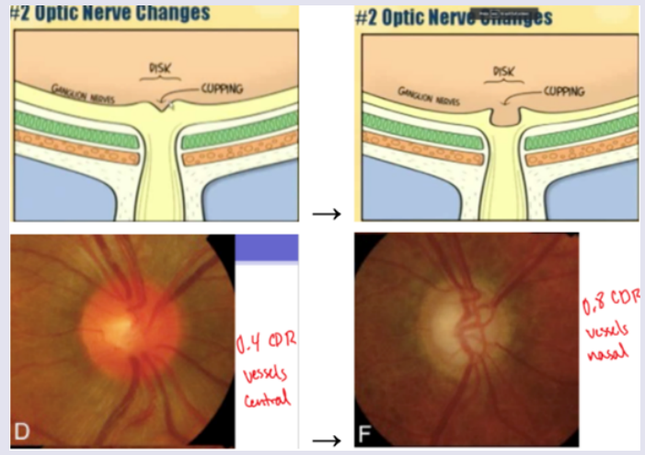
Treatment: There is no cure, but there is management.

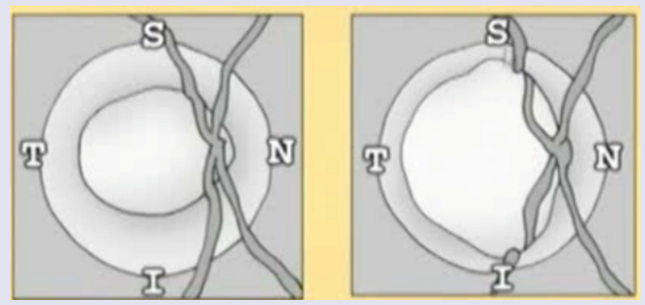
Medications:

1. Increase uveal scleral outflow: Prostaglandin analog (**latanoprost**) or selective laser trabeculoplasty (SLT) are both good initial treatment options. If these do not control IOP to target number then consider adding more eye drop options (see below). Target is 30% reduction from max IOP
2. Aqueous suppressants: beta blocker (**timolol**), alpha-2 agonist (**brimonidine**), carbonic anhydrase inhibitors (**acetazolamide**).
    a. Be careful when using beta blockers because they can worsen bronchospasm, bradycardia, depression, confusion, impotence, and myasthenia gravis.

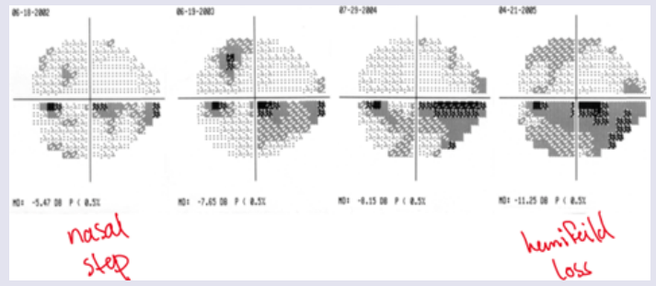
Procedures/Surgery

1. Selective Laser trabeculoplasty (SLT): outpatient laser procedure that takes 5 minutes. May need to repeat every 2-3 years.
2. Cataract surgery with minimally invasive glaucoma surgery (Phaco IOL with kahook or omni iStent)
3. Filter Surgery (trabeculectomy)
4. Tube Shunt implant (Ahmed tube)
5. Cyclophotocoagulation (ECP) → decreases ciliary body aqueous humor production.





ISNT rule: glaucoma goes against ISNT rule.
Normally I has thickest rim>S>N>T
In glc, I is thinnest, then S, then T and N

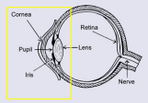


Visual field loss in glaucoma.

Clinical Pearls:
Digital punctal occlusion after instilling drops helps absorption. High IOP isn't the cause of glaucoma. Patients with thin corneas have IOP higher than measured.

Acute Primary Angle Closure Glaucoma

By: Kara Ye



Acute Angle Closure Glaucoma (AACG): The angle is blocked so aqueous humor cannot drain into the trabecular meshwork. There is primary and secondary angle closure glaucoma.

Causes:

- Pupillary block: occurs when the lens pushes against the iris, leading to blockage of normal aqueous humor flow from the posterior to the anterior chamber. This leads to increased posterior chamber pressure which causes the iris to be pushed forward, leading to blockage of the trabecular meshwork.
- Abnormal iris anatomy, such as in plateau iris and high peripheral iris roll.

Risk Factors: Shallow chambers (seen in high hyperopia), large cataract, elderly, FHx, female, Inuit or East Asian, diabetes.

- May be precipitated by factors that cause eye dilation such as topical mydriatic drops (red tops: **atropine**, **cyclopentolate**), anticholinergic medications, accommodation (reading), and dim light (night, movies).

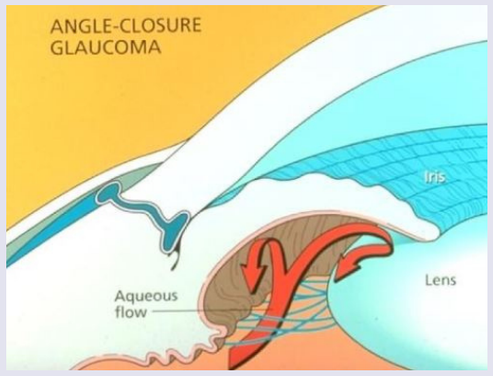
Symptoms: Severe eye pain, blurred vision, halos, headache, nausea, vomiting.

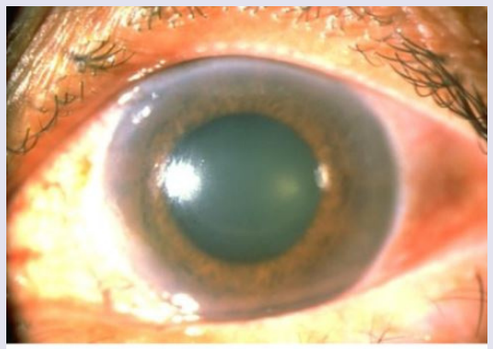
- Symptoms are worse in dark rooms and at night due to pupil dilation.

Slit Lamp Exam: Red eye, fixed mildly dilated pupil, ↑↑IOP on tonometry (usually above 30, but can go up to 60-70s!), corneal edema (because high IOP presses water into cornea), occluded angles on gonioscopy, narrow/occludable angle in the fellow eye, posterior synechiae, posterior exam showing spontaneous arterial pulsations (which indicates elevated IOP).

Treatment: EMERGENCY

1. Compression gonioscopy can determine if the trabecular blockage is reversible.
2. Max out drops (**timolol**, **brimonidine**, **latanoprost**, **dorzolamide**) and start acetazolamide 500 mg PO or IV. Paracentesis with 30-gauge needle at 6 o'clock if needed. IV mannitol is also an option but not really used in clinic. **Pilocarpine** (constricts pupils so it opens the angle) has fallen out of favor due to adverse side effects (headache accommodative spasm). If the IOP does not decrease after two courses of max treatment then proceed to laser peripheral iridotomy.
3. Laser peripheral iridotomy (LPI) is the definitive treatment. If the affected eye is too inflamed then perform LPI on the fellow eye due to 40-80% chance of AACG in 5-10 years.





Acute angle-closure glaucoma

Clinical Pearls:
Don't dilate patients with narrow angles! They might go into AACG.

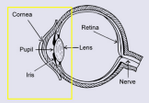
Giving **atropine** for bradycardia can cause AACG in a patient with narrow angles.

Consider testing renal function before giving **acetazolamide** or mannitol.

Avoid **acetazolamide** in sickle cell disease as it can induce more red blood cell sickling and precipitate sickle cell crisis. **Acetazolamide** can also cause sulfonamide-induced myopia and worsen angle closure.

Dry Eye Syndrome (DES)

By: Kara Ye



Dry Eye Syndome (DES) is one of the most common ocular conditions. DES is when the natural tear film, which functions to protect the cornea and conjunctiva from air damage as well as provide focusing power for vision, begins the breakdown. Therefore, DES can lead to not only eye irritation but also intermittent blurry vision.

Causes: most common causes is age and hormone changes. Aging causes decreased tear quality and quantity.

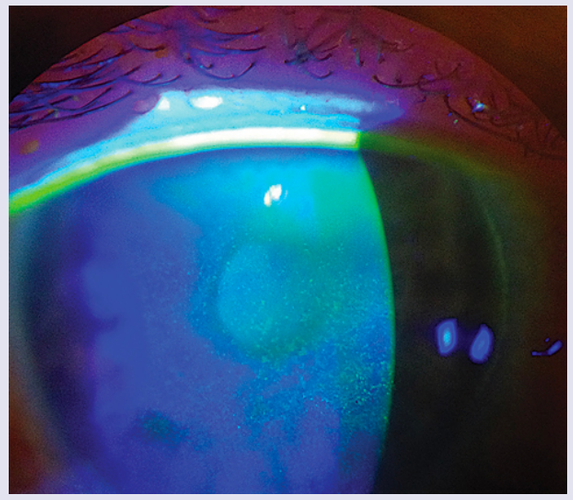
- Decreased tear quality occurs in conditions such as blepharitis or meibomian gland dysfunction, which cause the lipid layer of the tear film to be deficient -> faster tear film evaporation -> symptoms (blurry vision, burning) worse in the AM.
- Decreased tear quantity causes a deficiency in the aqueous layer of the tear film -> worse symptoms in PM
- Decreased tear quality and quantity often occur together, and mucin layer deficits can also occur.

Other causes: lifestyle/environment (allergies, dry climate, sleeping with fan, air vent blowing toward eyes, decreased blinking which often occurs when reading), systemic disease (Sjrogens, SLE, RA, GPA, sarcoidosis), conjunctival/corneal damage (LASIK, cataract surgery), drugs (OCPs, antihistamine, TCAs, chemotherapy), and many more.
Risk Factors: menopausal/postmenopausal women, sleeping with fan, air vent blowing towards face, not blinking enough, smoking, dry climate, systemic disease above.

Symptoms: blurry vision that improves with blinking, burning, excessive tearing, redness, foreign body sensation in eye.
Slit Lamp Exam: decreased tear meniscus (<0.5mm) seen at inferior lid margin (normal tear meniscus will have a convex shape), decreased tear breakup time (<10s), punctate corneal erosions in random location (best seen with fluorescein), mucus or debris or filaments in the cornea, parallel conjunctival fold lines.

Treatment: Mild DES can be treated with OTC artificial tears (AT's) 3-4 times a day, preferably preservative-free and in vials, such as refresh, systane, or theratears. Moderate/severe DES can be treated with: lubricating eye ointment at bedtime, preservative-free AT's every 1-2 hours, humidifiers, smoking cessation, cyclosporine eye drops (Restasis), Lifitegrast eye drops (Xiidra), Verinacline nasal spray (Tyrvaya), punctal plugs, omega-3 supplements, autologous serum tears, topical vitamin A, and bandage soft contact lens. Lateral tarsorrhaphy can be considered as a last resort.

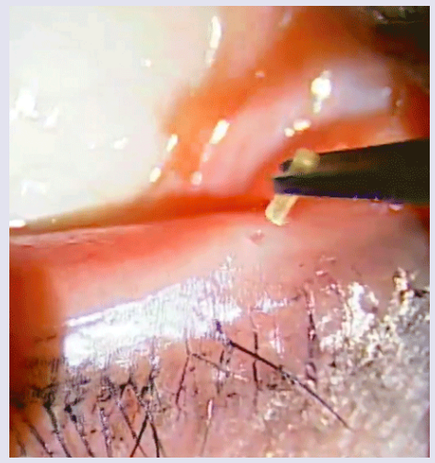
Clinical Pearls: tear film defects in an isolated area may indicate a focal corneal surface irregularity. Always use preservative-free AT's if usage is > 4 times a day, as preservatives can irritate the eye. Treat concurrent blepharitis and meibomian gland dysfunction with warm compresses and lid scrubs. Consider workup for connective tissue disease if patient history is suggestive. Patients with significant DES should be discouraged from corneal refractive surgery (LASIK, SMILE, PRK) & contact lens wear.



Punctate epithelial erosions

Preservative-free artificial tear vials

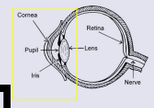
Never ever ever use visine because it contains tetrahydrazoline and naphazolin, which temporarily constricts blood vessels to help eye redness but chronic use leads to rebound vessel dilation = make redness worse (conjunctivitis medicamentosa).



Punctal Plugs

Ocular Emergency: Hyphema

By: Sarah Ye



What is it: A hyphema is the accumulation of red blood cells in the anterior chamber.

Causes: Common etiologies include blunt ocular trauma, intraocular surgeries, and the use of substances that impact coagulation factors or platelet functions (ethanol, aspirin, warfarin).

Spontaneous hyphemas may also occur in the absence of an identifiable cause and are associated with iris neovascularization, uveitis, and blood dyscrasia.

Risk Factors: past medical and family history (hemoglobinopathies, hemophilia, von Willebrand's disease), current medication use (antiplatelets, anticoagulants), and previous eye trauma.

Symptoms: blurry vision, visual distortion, pain, headache, and photophobia in the setting of trauma or high IOP secondary to hyphema

Exam: Initial evaluation includes an ophthalmic examination and complete history detailing events before the onset of the hyphema.

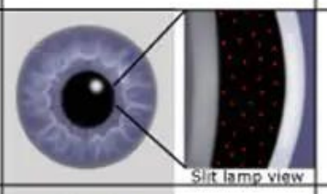
Hyphemas can be graded by the percent of the AC that is filled with blood. Grade 0 (microhyphema) hyphema will have circulating RBCs in the AC without a layering of blood. Grade I is less than 33% of the AC filled with blood; grade II is 33%-50% filling; grade III is greater than 50% but less than total filling, and grade IV "eight-ball " hyphema is total filling of the AC with blood.

Labs: Patients with unknown family or medical history may be screened with PT and PTT.

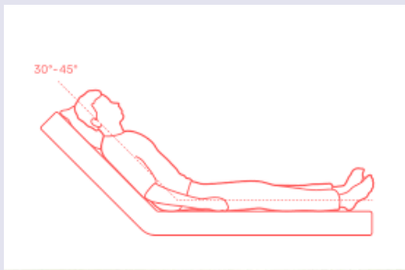
Treatment: Initial management includes

- elevation of the head above 30 degrees to allow blood to settle below the visual axis and topical analgesics for pain control
- Topical cycloplegics such as **cyclopentolate 1%** once acute glaucoma has been ruled out. It dilates the eye to prevent iris muscle spasms (reduce pain and risk for re-bleed)
- topical steroid therapy with **prednisolone acetate 1%** has been used to decrease inflammation and synechiae formation
- Daily IOP measurements and slit lamp exams are recommended to monitor for complications such as elevated IOP, re-bleed, and corneal blood staining

Prognosis: Grade I hyphemas are the most common with a good prognosis while grade IV is the least common with only a 20-50% of return to a vision of 20/50 or better.

Grade	Anterior chamber filling	Diagram	Best prognosis for 20/50 vision or better
Microhyphema	Circulating red blood cells by slit lamp exam only		90 percent
I	<33 percent		90 percent
II	33-50 percent		70 percent
III	>50 percent		50 percent
IV	100 percent		50 percent

http://blog.clinicalmonster.com/2016/01/04/morning-report-traumatic-hyphema-2/



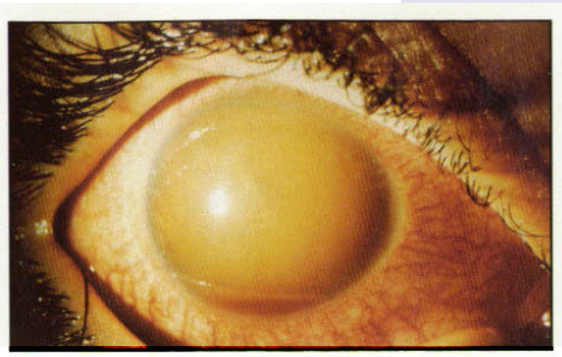
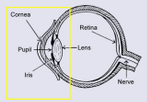


Figure 5: Corneal blood staining in a 5-year-old boy. Note the active bleeding site at 9 o'clock.

.Crouch ER Jr, Crouch ER. Management of traumatic hyphema: therapeutic options.

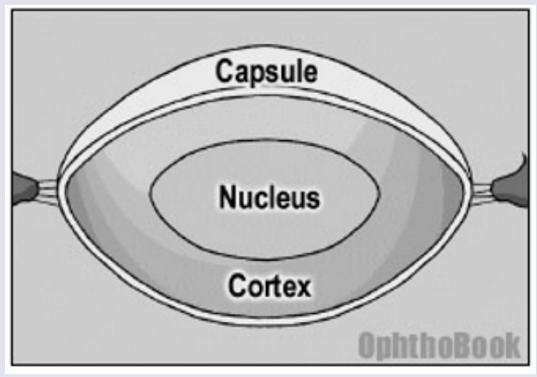
It is important to watch for corneal blood staining in children to prevent development of amblyopia. Visual pathway is not mature until around age 8 y/o!

Cataracts

By: Forrest Clark 

Lens Anatomy: To understand cataracts, it is imperative that you first grasp some fundamental aspects of lens anatomy. The lens is positioned behind the iris and has three main components, which can be likened to the structure of a peanut M&M:

1. Capsule (Candy Coating): a thin, transparent hyaline membrane enveloping the lens.
2. Cortex (Chocolate layer): Surrounding the nucleus, the cortex is made up of younger lens fibers.
3. Nucleus (Peanut): Situated at the center, the nucleus contains the oldest fibers of the lens.



What are Cataracts?
For us to have clear vision, the structures of the lens must maintain transparency. A cataract occurs when any part of the lens becomes opaque or cloudy, leading to a loss of transparency.

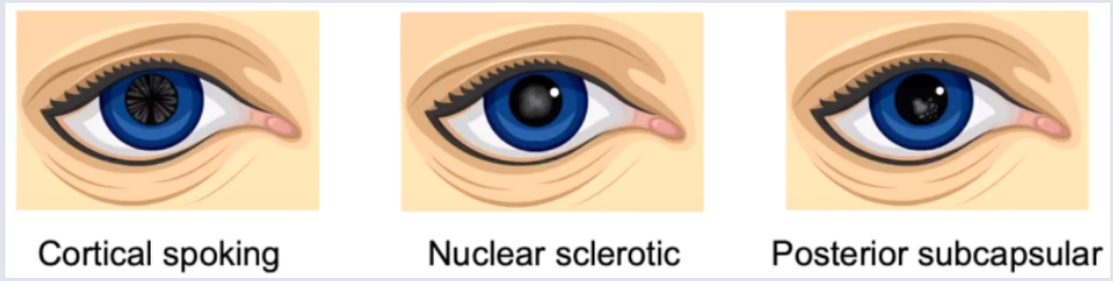
Pathophysiology:
Cataract formation is a natural process that occurs as the eye ages, and it arises from various factors. As the lens ages, its metabolic function declines, leading to the hydration (edema) and denaturation of lens proteins and fibers, resulting in the development of opacities. This process is akin to how an egg's clear albumin turns white when cooked.
While age-related cataracts are the most prevalent, they can also be congenital or arise due to factors such as trauma, toxicity, or as a secondary effect of metabolic diseases like Wilson's disease, among others.

Risk Factors:
Advanced age, sex (females>males), UV exposure, smoking.

Signs and Symptoms:
Cataracts usually present with slowly progressive vision loss or blurring over months to years. Glare, especially to bright sunlight or oncoming headlights while driving at night and altered color perception may also be present.

Types of Cataracts:
There are many advanced subtypes of cataracts, but the three types to be aware of are:

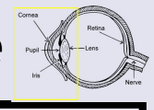
1. ***Nuclear Sclerotic Cataract (NSC)***: The most common form of cataract; develops as the lens' nucleus hardens due to aging.
   a. Slit lamp exam (SLE): characterized by opacification of the central lens, which presents with a brunescent (yellow-brown) appearance.
2. ***Posterior Subcapsular Cataract (PSC)***: a plaque-like opacity that develops in the posterior portion of the lens; associated with ocular inflammation and steroid use.
   a. SLE: presents with a granular, smudge-like appearance in the posterior portion of the lens.
3. ***Cortical Spoking Cataract (CSC***): Pathophysiology similar to that of NSCs
   a. SLE: presents with opaque, white tendrils appearing in the periphery (often pop up in the inferonasal quadrant first).



Treatment:
Treatment of cataracts is usually operative and cataract extraction is the signature surgery of ophthalmology. This surgery involves using a specialized ultrasound technique called phacoemulsification to break up and remove the cloudy lens. Subsequently, an artificial intraocular lens (IOL) is implanted in its place to restore vision. (For more detailed information on cataract surgery, click here.)

Ocular Emergency: Open Globe Injury

By: Sarah Ye, Kara Ye



Open Globe Injury: Full thickness injury that ruptures the eye.

Etiology and Pathophysiology: Open globe injuries are classified as secondary to

- Blunt trauma (ex: being hit in the eye, motor vehicle accident, elderly patient falling and hitting eye): Site of rupture commonly occurs where the sclera is thinnest and weakest (behind the insertion of the rectus muscles, the limbus, insertion of the optic nerve, and sites of prior eye surgery).
- Laceration with a sharp object (ie. being cut in with a knife, nail in the eye, etc).

Epidemiology: women were more likely to be elderly (median age 73) and injured from falls while men were younger (median age 36) and likely injured from projectiles at work or home improvement

Risk factors: prior eye surgery (ie: penetrating keratoplasty, radial keratotomy), elderly, working jobs that involve projectiles (metalworking, construction, home improvement)

Symptoms: pain, decreased vision, loss of fluid from the eye (positive seidel test). History of trauma, fall, or sharp object entering the eye.

DDx:

- Traumatic hyphema
- Orbital fracture
- Closed-globe injury
- Orbital compartment syndrome

Avoid excess eye manipulation or placing pressure on the globe!

Clinical Findings:

- clinical diagnosis based on history and physical examination. Exam is done by **penlight, indirect ophthalmoscope, or slit lamp**
- **Critical findings:**
  - Full thickness scleral or corneal laceration (use **seidel test**)
  - A **peaked** or irregular pupil
  - **Bullous** subconjunctival hemorrhage (involving 360° of the bulbar conjunctival because the thinnest part of the sclera/most likely area to rupture is just posterior to the rectus muscle insertion sites)
  - A deeper shallow anterior chamber (AC) compared to the fellow eye
  - Iris transillumination defects
  - Lens material or vitreous in the AC, foreign body, extraocular motility limitations (will be greatest in the direction of rupture)
  - intraocular contents outside of the globe (ex: **iris prolapse**)

Diagnostic Testing: despite imaging, a ruptured globe remains a **clinical diagnosis.**

- If globe rupture is suspected, many recommend proceeding directly to the operating room for exploration and skipping contact B-scan ultrasonography for concern for iatrogenic expulsion of globe contents
- CT orbits has an accuracy of 81%, sensitivity of 76%, and specificity of 85%, so while CT is useful in cases of ocular trauma it can not be solely relied on due to potentially undiagnosed globe rupture

Management:

- Initial Management in the ED:
  - protect the eye with a hard shield but do not patch
  - prophylactic antibiotic treatment: vancomycin or cefazoline and moxifloxacin to cover organisms commonly associated with posttraumatic endophthalmitis
  - antiemetics (ondansetron) to prevent nausea and vomiting
  - Tetanus prophylaxis
- Consults ophthalmology for immediate surgery

Anesthesia must be aware not to use high dose ketamine or succinylcholine, as these medications can increase IOP and cause expulsion of intraocular contents.

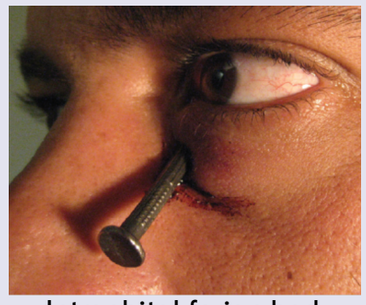
Prognosis:

The Ocular Trauma Score (OTS) is used to predict visual outcome

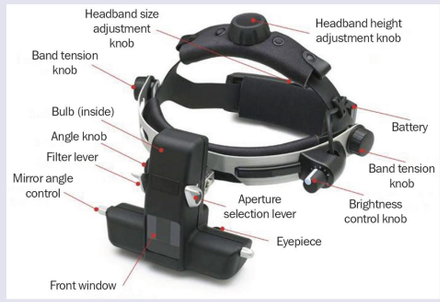
- ranges from 1 (most severe injury) to 5 (least severe injury)
- first determine initial visual acuity (VA) after injury, then subtract each associated diagnosis/complication
- correspond the raw points/OTS to the estimated probability of attaining a specific VA 6 month follow-up (see clincial vignette)

Clinical Vignette: During my ophthalmology rotation at Grady, a 30 y/o man came in with bilateral penetrating open globe injuries from an unsuccessful suicide attempt. The gun was aimed too anteriorly so the bullet went through one eye, scraped his nasal bridge, and out the other eye missing his brain entirely. He was rushed to surgery.

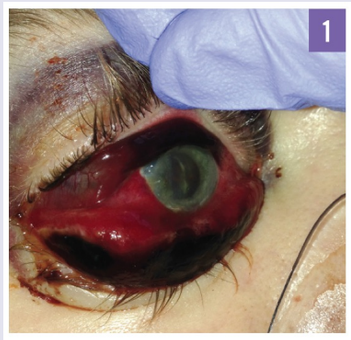
For his OTS he came in NLP (60), had ruptured globes (-23), perforating injury (-14), and retinal detachments (-11). The sum of his raw points is therefore 60-23-14-11 = 12. His OTS is 1 meaning at 6 months he has a 74% chance of being NLP, 15% of being able to perceive light/hand motion, 7% chance of seeing between 1/200-19/200, 3% chance of seeing between 20/200-20/50, and 1% chance of seeing better than 20/40. He ended up getting bilateral enucleations so he is NLP since both eyes were removed.



Intraobital forign body open globe injury

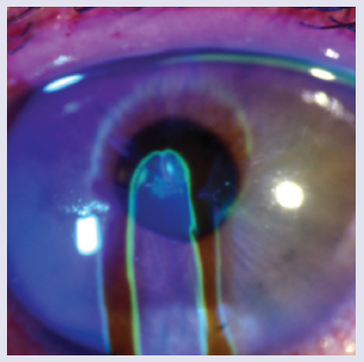


indirect ophthalmascope goes on your head

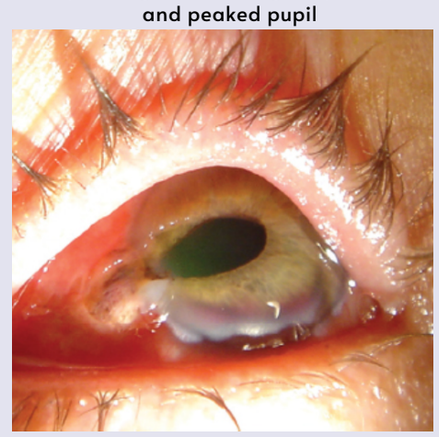


The patient had a 4-mm irregular fixed pupil, a cloudy cornea, and a circumferential subconjunctival hemorrhage with prolapse.

Ruptured globe showing iris prolapse, and peaked pupil



Positive seidel test indicating there is a full thickness cornel laceration



Ruptured globe showing flat AC, iris prolapse, and peaked pupil.

Shield the eye with a metal fox shield (right eye) is preferable but if there isn't one in the ED a styrofoam cup (left eye) can come in handy

TABLE 3.14.1 Calculating the OTS

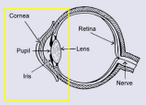
Variable	Raw Points
Initial Vision	
NLP	60
LP/HM	70
1/200 to 19/200	80
20/200 to 20/50	90
>20/40	100
Rupture	–23
Endophthalmitis	–17
Perforating injury	–14
Retinal detachment	–11
Afferent pupillary defect	–10

LP/HM, light perception/hand motion; NLP, no light perception.

TABLE 3.14.2 Visual Prognosis per OTS

Sum of Raw Points	OTS	No Light Perception (%)	Light Perception/ Hand Motion (%)	1/200 to 19/200 (%)	20/200 to 20/50 (%)	>20/40 (%)
0 to 44	1	74	15	7	3	1
45 to 65	2	27	26	18	15	15
66 to 80	3	2	11	15	31	41
81 to 91	4	1	2	3	22	73
92 to 100	5	0	1	1	5	94

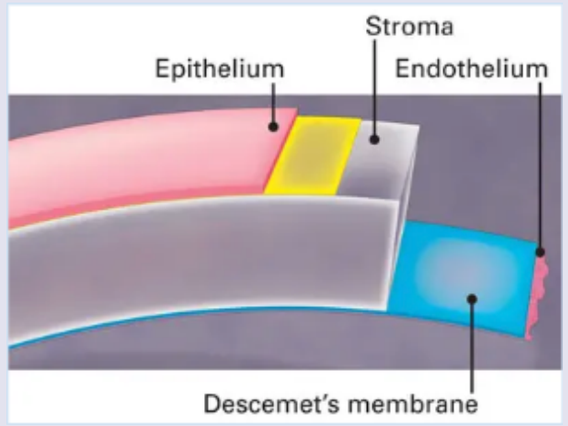
Fuch's Endothelial Corneal Dystrophy

By: Kara Ye 

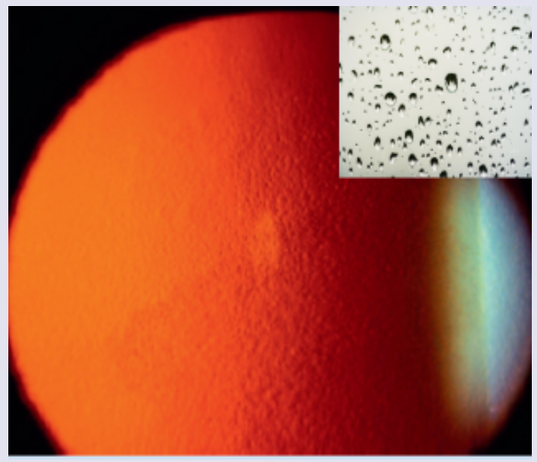
Fuch's endothelial corneal dystrohy is the most common posterior corneal dystrophy and involves the endothelial layer of the cornea. Endothelial cell loss/damage causes corneal swelling and decreased vision. It has autosomal dominant inheritance but can also occur sporadically.

Causes: autosomal dominant inheritance
- associated with CTG trinucleotide repeat on TCF4 gene
- More recent studies identified additional genetic variants at KANK4, LAMC1, and LINC00970/ATP1B1 genes.
- Early onset Fuchs is associated with COL8A2 mutation.

Risk Factors:
- CTG trinucleotide repeat on TCF4 gene
- smoking (doubles risk of corneal guttae!)
- low BMI
- female
- age over 50

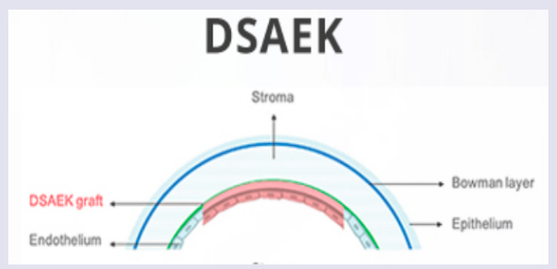


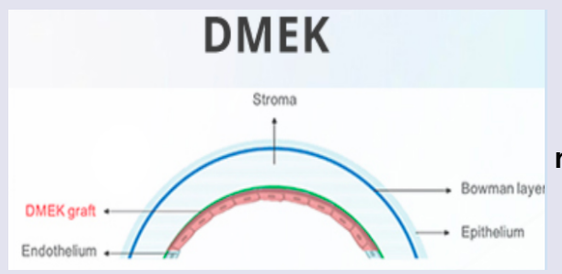
Layers of the cornea. Fuchs affects the endothelium



Corneal guttae seen by retroillumination, resembling dew drops.

Symptoms: blurry vision that is worse after waking up (due to fluid buildup from closed eyes). Symptoms are usually bilateral and develops in 50-60s. Pain if bullae rupture.
Slit Lamp Exam: Corneal guttae with stromal edema bilaterally. Bullae, descemet's folds, subepithelial haze, and scarring/pannus may be present in more severe cases.
Other workup tests to support diagnosis:
- History for previous surgery? If yes then consider post surgical corneal edema.
- Measure IOP: if IOP is elevated then consider glaucoma or edema from elevated IOP.
- Corneal pachymetry: thickness>600 is highly suggestive of fuchs.
- Specular microscopy (images endothelium) and corneal tomography (to monitor progression of fuch's) can be helpful if available.





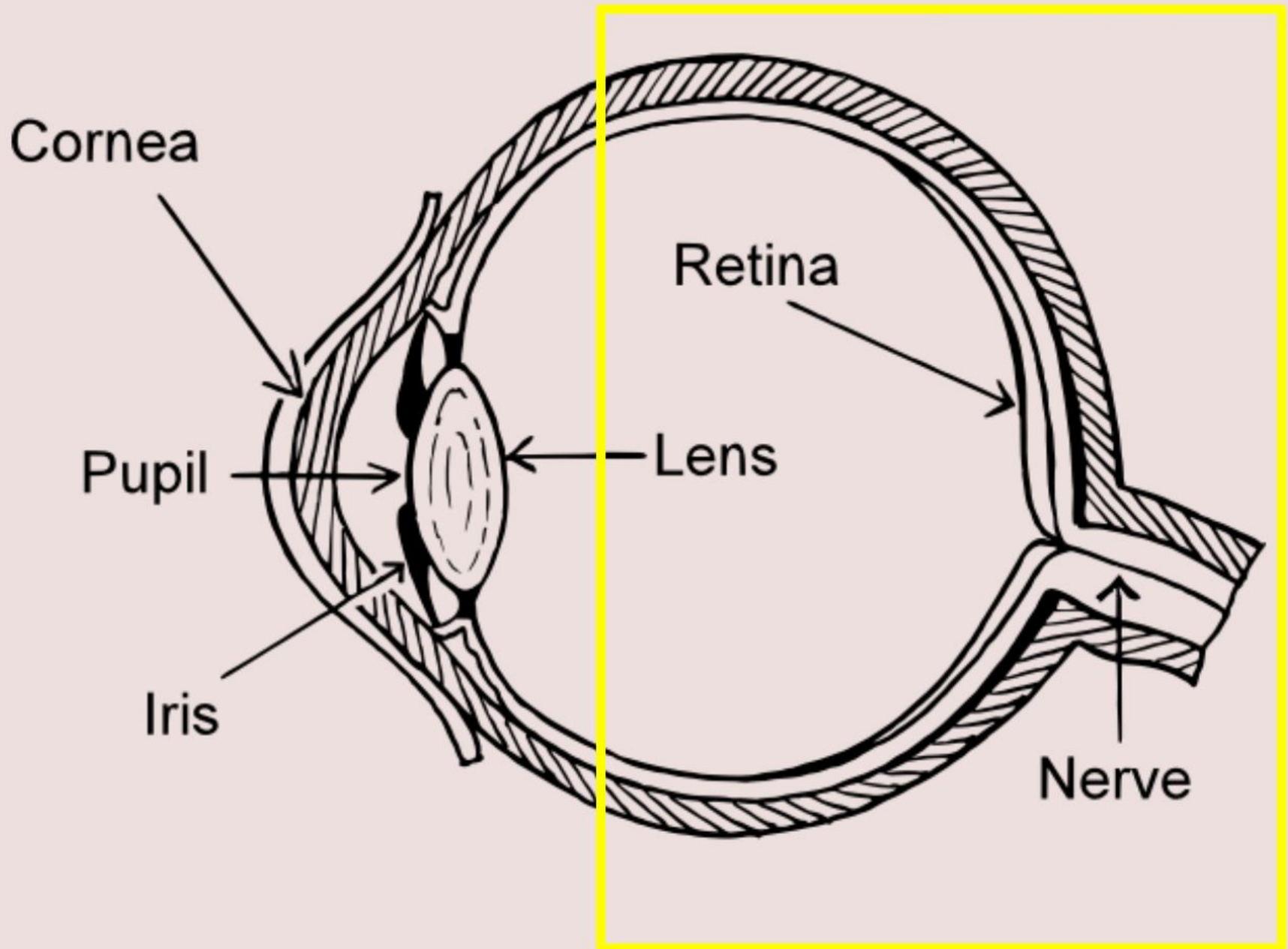
DSAEK/DSEK: descemet membrane endothelial keratoplasty replaces endothelium, descemet's membrane, and some stroma.

DMEK: descemet membrane endothelial keratoplasty replaces endothelium and descemet's membrane.

Treatment:
Medical: **Muro sodium chloride 5% drops** QID or ointment QHS to dehydrate cornea. Gently blow dry or fan eye at arm's length in the morning. IOP reduction if needed. Monitor every 3-12 months for corneal edema or scarring.
Surgical: Endothelial keratoplasty (DSEK/DSAEK, DMEK, DSO), which is a type of partial thickness corneal transplant in most cases if medical management fails to improve VA. Penetrating keratoplasty which is full thickness corneal transplant is there is anterior stromal scarring.
Corneal endothelial cell therapy is currently in clinical trials and on the horizon!

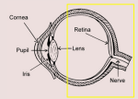
*If there is only corneal guttae without stromal edema, then it is endothelial dystrophy, which progresses to fuch's once stromal edema develops.
*Guttae are best seen using retroillumination.
*Guttae is latin for "drop". Guttae in Fuch's look like drops on the cornea.

POSTERIOR SEGMENT



MULTIPLE EVANESCENT WHITE DOT SYNDROME (MEWDS)

By: Sarah Ye



MEWDS: A rare inflammatory chorioretinopathy part of a group of inflammatory disorders (called White Dot Syndromes) which share the presence of discrete, multiple, well-circumscribed, yellow-white lesions at the level of the retina, outer retina, RPE, choriocapillaris, and/or choroid

Causes: Currently the etiology is unknown, but MEWDS commonly develops after a viral illness which suggests direct viral infection or immune-mediated damage of the retina and choroid

Risk factors: More common in young (<50 years old) females; recent viral illness

Symptoms: Unilateral acute onset central scotoma, floaters, photopsias, decreased VA, decreased night vision

Slit Lamp Exam: Well-circumscribed yellow-white dots are seen on retinal exam *[black arrows]*; granularity around fovea *[blue arrow]*

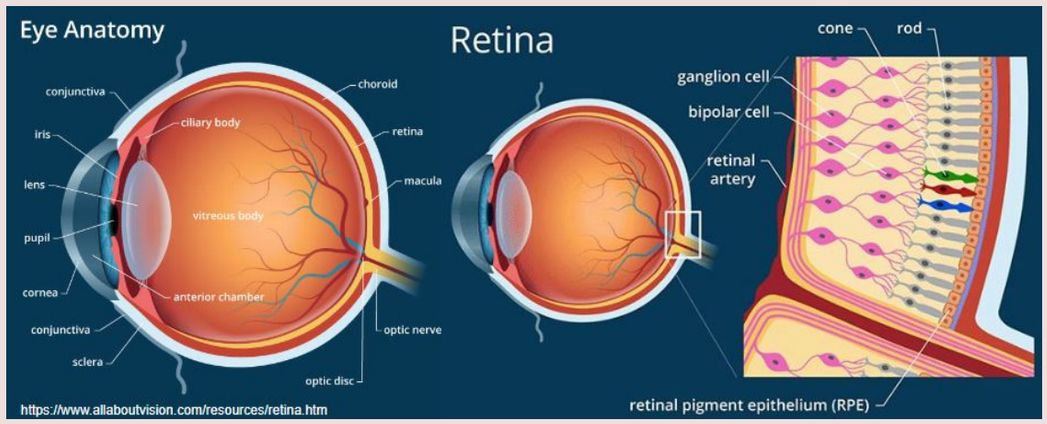
Imaging: Imaging aids in monitoring retinal spots.

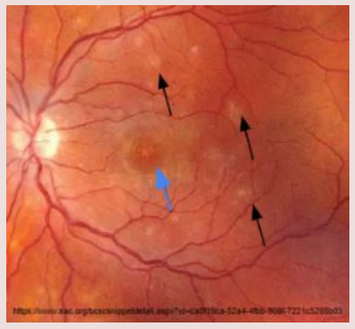
- Fluorescein angiography: lesions will be bright and surround the fovea. This is called the “wreath sign” since it looks like a wreath
- Indocyanine green angiography: lesions will be dark *[red arrows]* and may represent hypoperfusion to the choroid
- OCT shows dark areas at the level of the photoreceptors (rods, cones)

Labs: BMP, CBC w/ diff, ESR, FTA-ab, QuantiFERON Gold to rule out systemic inflammatory or infectious etiologies (syphilis, tuberculosis)

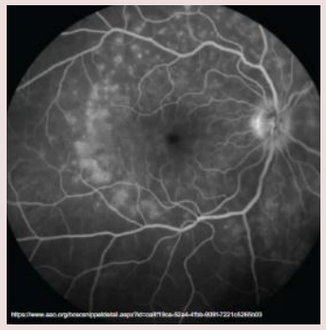
Treatment: Close observation with dilated fundus exams. A few case reports have shown that steroid therapy is effective in shortening the course of MEWDS.

Prognosis: Vision returns to baseline over 4-8 weeks. Complications (persistent scotoma, choroidal neovascularization, cystoid macular edema) are rare.

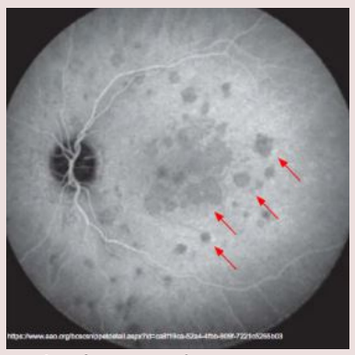




Slit lamp exam



Fluorescein angiography: "wreath sign"



Indocyanine green angiography

Is this the left or right eye?

Hint: Whichever side the optic disc is on

Answer: Left eye

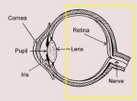
Clinical Pearls:

When presented with unilateral acute vision loss, rule out emergent situations like trauma, retinal detachment, giant cell arteritis, central retinal artery occlusion, central retinal vein occlusion, and infections before proceeding with more benign conditions such as MEWDS.

Abbreviations:
RPE: retinal pigment epithelium; BMP: basic metabolic panel; CBC: complete blood count; ESR erythrocyte sedimentation rate; FTA-ab: Fluorescent treponemal antibody absorption test

Retinopathy of Prematurity

By: Erik Rosas, Kara Ye



Retinopathy of Prematurity (ROP): Potentially blinding eye disease that can occur in premature (<31 weeks gestation) and low birth weight infants (<2.8 lbs). Leading cause of childhood blindness in the U.S.

Pathophysiology: Vascular development of the retina in the nasal and temporal zones is not complete until weeks 32 and 40 respectively. This is a hypoxic process in-utero driven by IGF-1 and VEGF. A premature infant is prematurely exposed to higher oxygen levels after birth which decreases VEGF. This causes delayed and then abnormal retinal vascularization.

Risk factors:

- Birth before 31 weeks gestation; birth weight < 1250g (2.8 lbs); supplemental oxygen therapy use
- Other potential maternal, perinatal, and infant factors under investigation:
  - Maternal → Diabetes, HTN, Smoking.
  - Infant → Race, Gender, Cardiac abnormalities.

Clinical Manifestations: Likely no clinical features present in the premature infant. Thus, screening infants meeting risk factors is important.

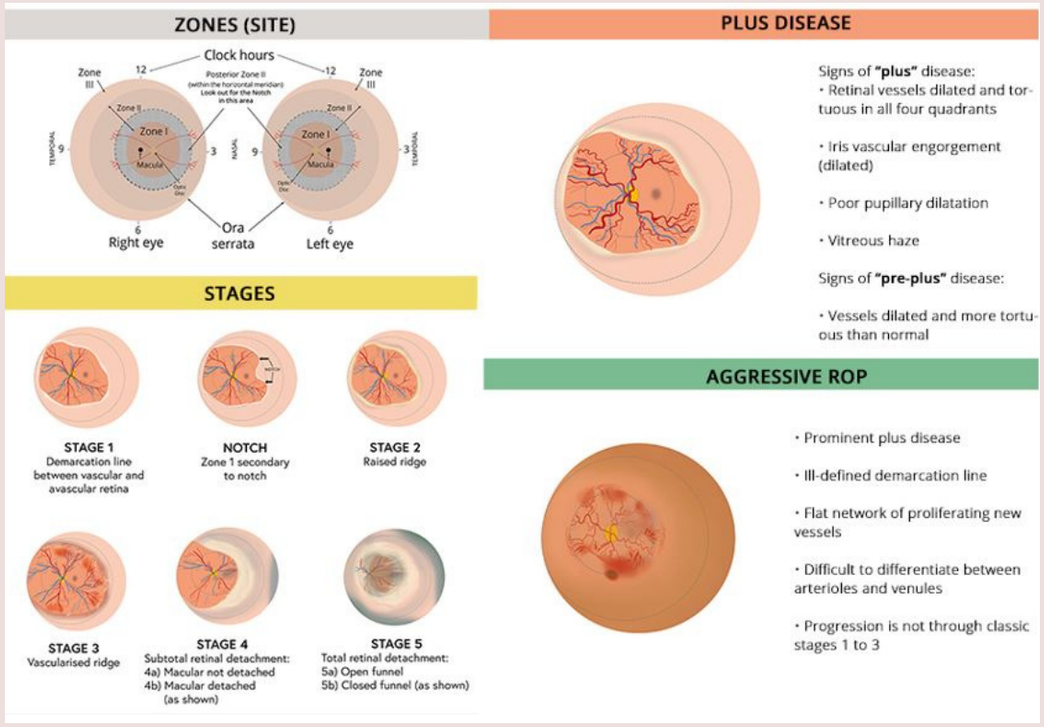
- **Leukocoria**: white reflection instead of red reflex. Very rare to be the initial presentation of ROP.

Diagnosis: Dilated fundus exam at 4 weeks postnatal age or at 31 weeks gestational age. Whichever comes latest.

- Guidelines from the AAO and AAP: screen all infants born ≤30w GA or ≤1500g (3.3lbs) birth weight.
  - Larger infants (1500g-2000g) may benefit from screening as well if risk factors mentioned above are present.

Prognosis:

- Milder cases: disease improves and leaves no permanent damage; resolves spontaneously (90% of all infants). Follow up beyond the first decade of life is typical for untreated patients.
- More severe disease: can lead to impaired vision or even blindness. Treated patients will typically have long-term, regular follow ups.
  - Long-term visual impairment in 7-15% of children with moderate to severe ROP.

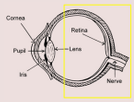


Treatment		
Stage	**Description**	**Management**
0	Normal retina	No ROP specific follow-up needed.
1	Mildly abnormal blood vessel growth	Typically do not require treatment and the disease spontaneously resolves.
2	Moderately abnormal blood vessel growth	
3	Severely abnormal blood vessel growth	May not require treatment: laser photocoagulation &/or intravitreal anti-VEGF agents.
4	Partial retinal detachment • Stage 4A: without macular involvement • Stage 4B: with macular involvement	Surgical intervention attempted to promote reattachment of the retina. • Surgical options: scleral buckling or vitrectomy
5	Total retinal detachment	

Clinical Pearls:

- Very rare for leukocoria to be the initial presentation of ROP → If present, leading DDX is retinoblastoma.
- Scheduling exams early in the morning after the infant's nap and feeding time will ensure better cooperation and a less irritable baby!

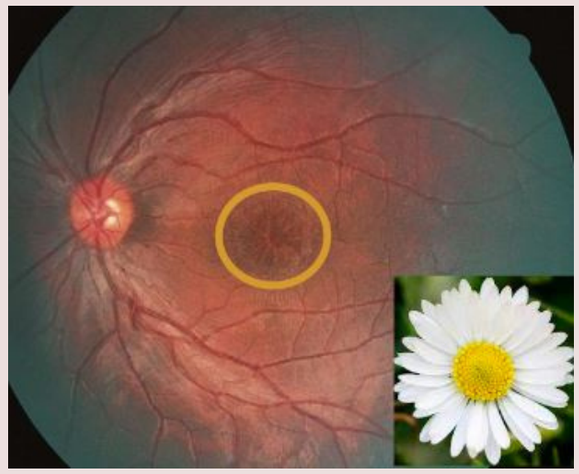
Retinoschisis

By: Kara Ye 

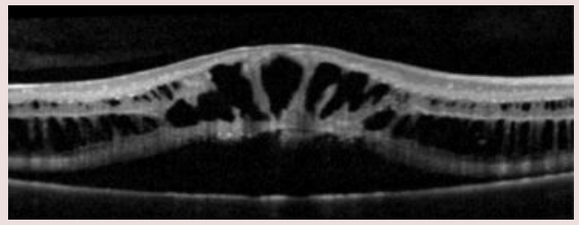
	Juvenile X-linked Retinoschisis	Age-related Degenerative Retinoschisis
Cause	• Separation of the layers of the retina due to **mutations in the RS1 gene** which codes for retinal adhesion proteins. • X-linked recessive so mainly **males** are affected. Usually presents in school age children. • May have **family history** of poor vision.	• Separation of the layers of the retina due to age-related degeneration. • Usually presents in 50s-70s.
Signs/ Eye exam	• Can be asymptomatic. • Decreased VA if macula is involved or if **vitreous hemorrhages** appear. • Cystic **foveal changes** in a radiating pattern (**petaloid appearance**) on fundus exam (98-100% of patients). Peripheral schisis in 50% of patients.	• Usually asymptomatic. • Decreased VA and absolute scotoma may occur. Vitreous hemorrhage is rare. • **Smooth dome shaped** cystoid changes near the ora serrata. Usually located inferotemporally and bilateral. • Absent corrugations and pigment demarcation line (vs retinal detachment where RPE demarcation line and corrugations are present).
Workup	• Ask about family history. • DFE with scleral depression rules out retinal detachment, tears, or holes. • OCT and indocyanine green angiography to visualize layer of schisis and cystoid changes respectively. • IV fluorescein angiography will not show leak, ruling out cystoid macular edema.	• Slit lamp exam will be **negative** for anterior chamber pigment cells (Shafer's sign) or inflammation. • DFE with scleral depression rules out retinal detachment, tears, or holes. • OCT to visualize layer of retinal that is split. • Visual Field test to identify absolute scotoma.
Treatment	1. Eye exams every 6-12 months by a pediatric or neuro-ophthalmologist. 2. Patching for amblyopia. Glasses for hyperopia. 3. Genetic counseling and low vision aids (large print books). 4. **Dorzolamide** drops or **acetazolamide** pills improves cystic areas. 5. Surgery: Vitrectomy for non-clearing vitreous hemorrhages. Scleral buckle for retinal detachment.	1. Monitor if asymptomatic. 2. Eye exams every 6 months as patients are at high risk of retinal detachment. 3. Scleral buckle if retinal detachment develops.

Fun Fact:

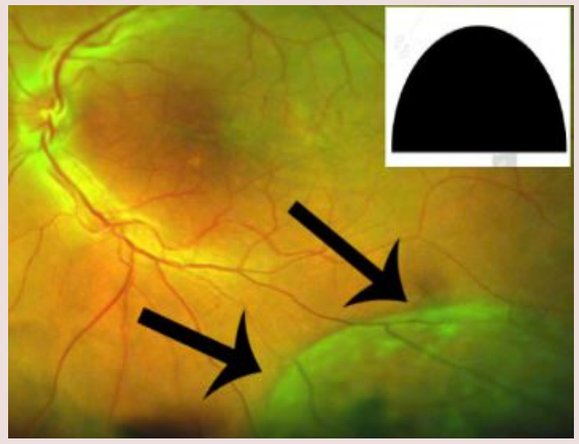
- There are 10 retinal layers! In retinoschisis, retinal splitting usually occurs at the outer plexiform layer.



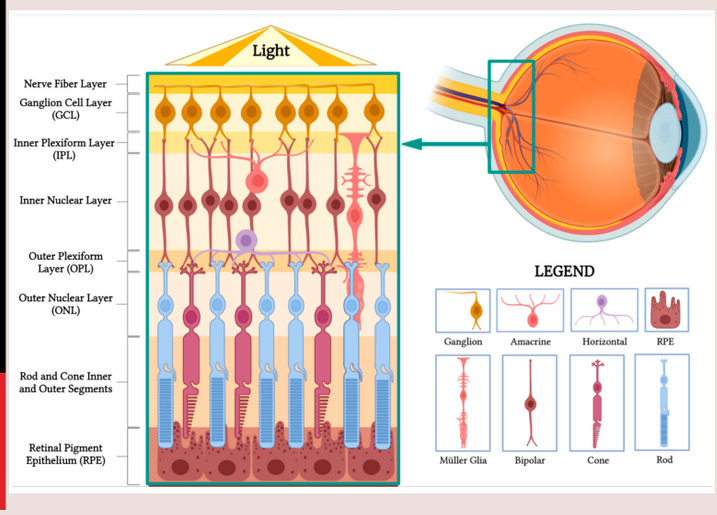
Petaloid foveal changes seen in x-linked retinoschisis



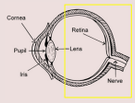
OCT showing cystic separation of retinal layers seen in the fovea x-linked retinoschisis



Dome shaped infratemporal schisis seen in age related retinoschisis



Idiopathic Intracranial Hypertension

By: Sarah Ye 

Idiopathic Intracranial Hypertension (IIH)/Pseudotumor Cerebri is increased ICP of unknown etiology. The primary ocular finding is papilledema.

- **Optic disc edema**: swelling of the optic nerve head due to any etiology (inflammatory, infiltrative, compressive)
- **Papilledema**: swelling of the optic disc caused by increased ICP

Epidemiology: higher prevalence in obese females of childbearing age (1.0:100,000 vs 7.9-20:100,000); mean age 29.0 (±7.4)

Other Risk factors:

- Certain medicines: TCA, vitamin A, lithium, anabolic steroids, OCPs, nalidixic acid, cyclosporin
- Systemic illnesses: OSA, hypothyroidism, anemia, Addison disease, SLE, Behçet's syndrome, PCOS, coagulation disorders, uremia

Hypothesized Pathophysiology:

- Cerebral edema (but not really seen in pts)
- Distal transverse venous sinuses stenosis-> cerebral venous HTN (but not clear if stenosis is the primary cause or is secondary to the elevated ICP)
- Obesity-> increased intrabdominal pressure-> increased cardiac filling pressure-> impedes venous return from brain-> elevated intracranial venous pressure (but IIH is also seen in non-obese pts)
- Microthrombosis in the sagittal sinus-> block CSF absorption in arachnoid granulations (but no hydrocephalus is seen in pts)

Symptoms: headaches (84%), transient vision loss (68%), back pain (53%), pulsatile tinnitus (52%), double vision (18%)

DDx:

- Intracranial mass lesions
- Increased CSF production (ex: choroid plexus papilloma)
- Decreased CSF absorption (ex: adhesions after meningitis, SAH)
- Obstructive hydrocephalus
- Obstruction of venous outflow (ex: venous sinus thrombosis, jugular vein compression)

Dx Testing:

- Slit Lamp Eye Exam: papilledema
- Optic Nerve photos
- Visual field: enlarged blind spot, nasal step
- Neuroimaging: MRI/CT scan, MRA/MRV to rule out ddx
- Lumbar puncture
- Sleep study

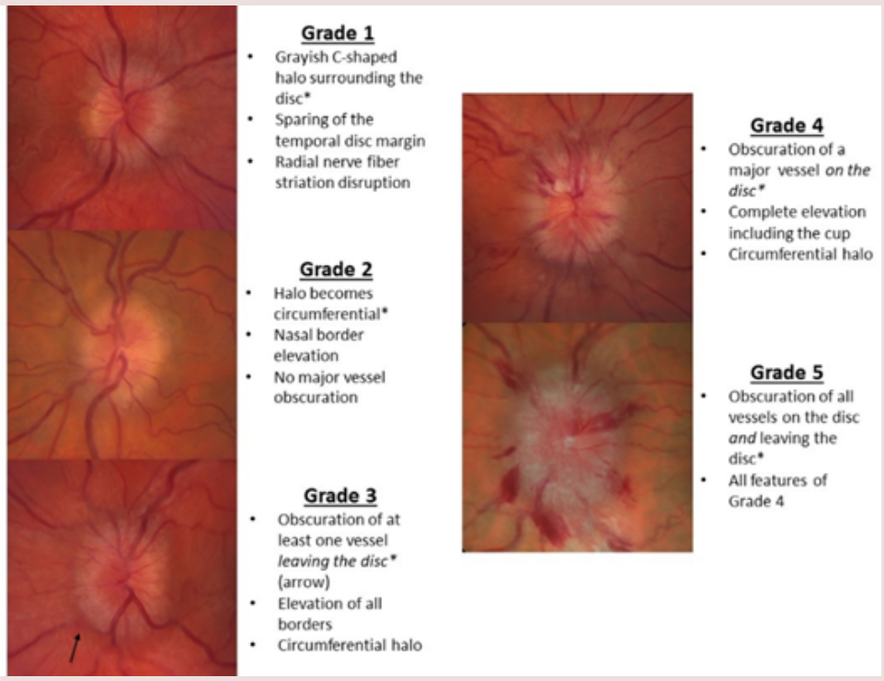
Labs: BMP, CBC w/ diff, ESR, TSH

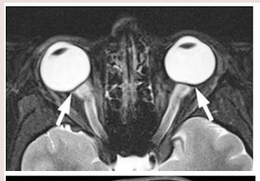
Treatment: no formal guidelines

- Weight loss (5-10%)
- Medication: Acetazolamide carbonic anhydrase inhibitor reduces rate of CSF production. Topiramate carbonic anhydrase inhibitor improves headaches and suppresses appetite to aid weight loss
- Surgery is usually for fullminant IIH: VP Shunt, Optical nerve sheath fenestration, Venous sinus stenting

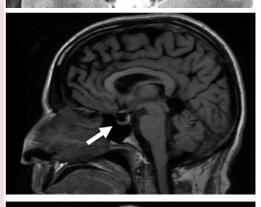
Prognosis:

- course is variable (weeks to years)
- independent factors associated with worse visual outcome: Male, African American, Morbid obesity, Anemia, OSA, Fulminant IIH
- Recurs in 8-38% of patients



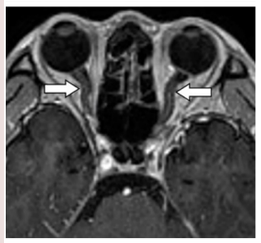


Axial T2 fat-saturated MRI showing flattening of the posterior globes (white arrows)



Sagittal T2 FLAIR showing empty sella in a patient with IIH (white arrow)

https://eyewiki.aao.org/Empty_Sella_Syndrome



Optic nerve tortuosity (white arrows) is a characteristic imaging finding in IIH. Low sensitivity, high specificity (80.8-95.0%)

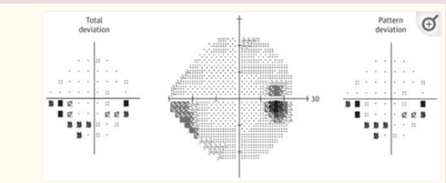


Figure 3

A Typical Example of the Most Common Visual Field Defect

Images show an enlarged blind spot with a partial inferior arcuate nerve fiber bundle defect.

TABLE 5

Revised diagnostic criteria for idiopathic intracranial hypertension[5]

IIH (Typical)
A. Papilledema
B. Normal neurologic examination +/- cranial nerve abnormalities
C. Neuroimaging: Normal brain parenchyma void of hydrocephalus, meningeal abnormalities, lesions as seen on MRI +/- gadolinium and MRV to also rule out venous sinus thrombosis
D. Normal CSF composition
E. Elevated LP opening pressure > 250 mm H_20 (adults); > 280 mm H_20 (children); > 250 mm H_20 if not sedated or obese (children)
IIH without papilledema qualifies if criteria B-E are met and the patient also has unilateral or bilateral abducens nerve palsy; or in the absence of papilledema and abducens nerve palsy, an IIH diagnosis can be suggested if criteria B-E are met and 3 of the following neuroimaging criteria are satisfied:
1. Empty sella turcica
2. Flattening of the posterior aspect of the globe
3. Distension of the perioptic subarachnoid space +/- tortuous optic nerve
4. Transverse venous sinus stenosis

CSF, cerebrospinal fluid; IIH, idiopathic intracranial hypertension; LP, lumbar puncture; MRI, magnetic resonance imaging; MRV, magnetic resonance venography.

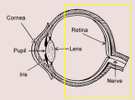
Clinical Pearls:

Patients with papilledema should always be evaluated for an intracranial mass. First step is to perform MRI of the brain before proceeding with a LP!

Always measure the blood pressure to determine if disc swelling is due to a hypertensive emergency!

Attack on Melanocytes!: Vogt-Koyanagi-Harada (VKH) Disease

By: Sarah Ye



VKH is an autoimmune CNS condition that affects the skin, eyes, and ears. It is likely caused by loss of immune tolerance to melanocytes within the meninges, eyes, skin, hair, and inner ear.

Epidemiology: F>M (2:1); wide age range 3-78 y/o but generally 30-40 y/o; Asian, Latino, Native American, or Asian Indian

Risk factors: Genetics (HLA-DRB4, HLA-DRB1-04*05, and HLA-DRB-04*01)

Pathophysiology:

- patients develop T-cell-mediated immunity against melanocytes following an inciting viral environmental factor (CMV, EBV)
- loss of tolerance of melanocytes→ non-necrotizing granulomas in the eye, the inner ear, the skin, and hair→ depigmentation, hearing loss, uveitis

Symptoms: 4 specific phases

1. **Prodromal:** dizziness, eye pain, photophobia, headache, meningeal signs, fever, recent viral illness
2. **Acute uveitic:** blurry vision and photophobia
3. **Chronic phase:** extraocular physical manifestations
   a. Hearing (tinnitus, dysacusis, and hearing loss)
   b. Depigmentation (skin, eyebrows, eyelashes, scalp, ciliary body)
4. **Recurrent chronic phase:** all the symptoms above

Clinical and Diagnostic Findings:

1. **Prodromal:** very subtle chorioretinal findings seen on ICG-angiography, CSF pleocytosis
2. **Acute uveitic:** chorioretinal inflammation→ edema extending to the retina, the optic nerve, ciliary body→ exudative retinal detachments, papillitis, acute angle closure
3. **Chronic phase:** extraocular physical manifestations
4. **Recurrent chronic phase:** panuveitis, sunset glow fundus, choroidal neovascular membranes, neovascularization of the iris, open- and closed-angle glaucoma, and subretinal fibrosis

DDx:

- Sympathetic ophthalmia
- VKH-like Medication Toxicity (checkpoint inhibitors)
- Choroidal Melanoma
- Infectious Uveitis: Syphilis, TB, endophthalmitis
- Alport and Cogan Syndromes
- Sarcoidosis
- SLE Chorioretinopathy

Diagnosis: clinical but can be supported with diagnostic testing

- 3 classifications of VKH disease:
  a. **Complete VKH disease:** neurologic, dermatologic, and eye findings all are present
  b. **Incomplete VKH disease:** either dermatologic or neurologic findings
  c. **Suspected VKH disease:** eye findings only
- require exclusion of other causes of intraocular findings, including a history of trauma, surgery, or infectious etiologies

Diagnostic Testing:

- No serum markers currently established
- CSF pleocytosis during the prodromal phase
- ICGA: 90 to 100% sensitivity
- FA
- OCT (OCTA possibly)

Management:

- **Systemic treatment:** systemic Immunomodulatory therapy (alkylating agents, antimetabolites, and TNF-a inhibitors), IV pulse corticosteroid
- **Local treatment:** Sub-Tenon triamcinolone injections, Fluocinolone intravitreal implant
- **Surgery:** limited to treating the complications (cataracts, high IOP, macular holes)

Prognosis:

- Positive prognosis if treated early (with both corticosteroid and IMT)
- Poor prognostic indicators: early age at onset, recurrent episodes, sunset glow fundus

Figure 9-57 Vogt-Koyanagi-Harada syndrome. Vitiligo of the upper eyelid and marked poliosis in the convalescent stage.

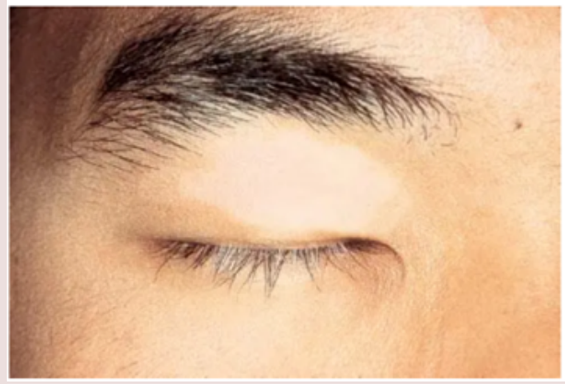
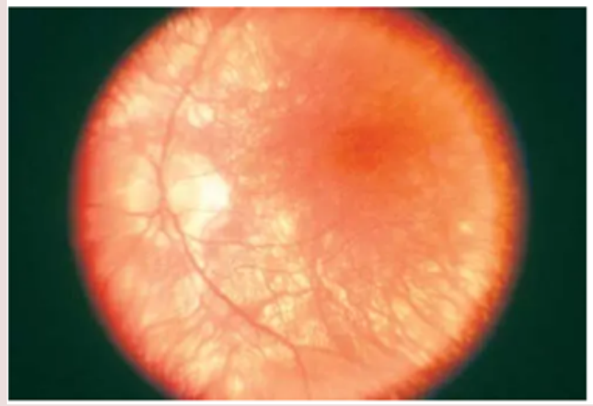
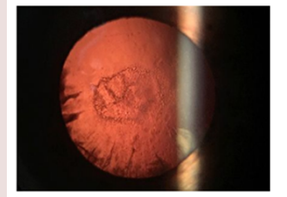


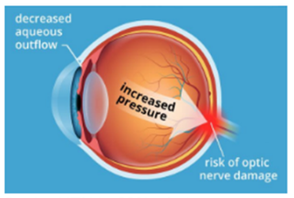
Figure 9-55 Vogt-Koyanagi-Harada syndrome. Fundus photograph showing sunset-glow fundus appearance with juxtapapillary detachment in a Hispanic patient in the convalescent stage.



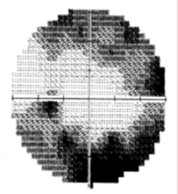
sunset-glow fundus (orange-red fundus + optic disc pallor) from recurrent flare-ups



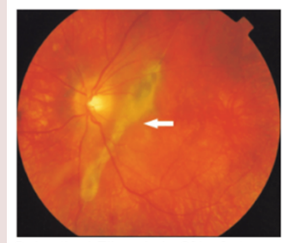
Cataracts in 45% of pts



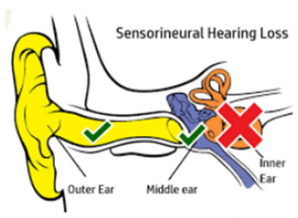
Ocular HTN in 33% of pts



Glaucoma in 18% of pts

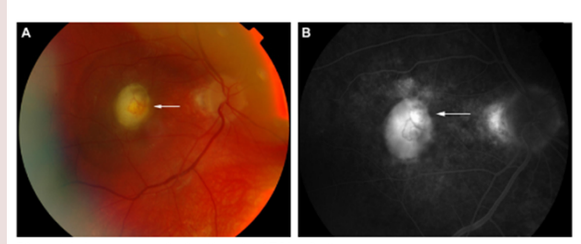


Subretinal Fibrosis in 6% of pts



Sensorineural Hearing loss in 68% of pts

Vertigo in 19% of pts



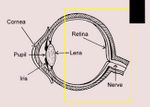
Choroidal neovascularization in 17% of pts

Clinical Pearl:

Remember the affected areas in VKH have melanocytes which are the skin, hair, eyes, and inner ears (who knew the ear had melanocytes!).

Diabetic Retinopathy

By: Erik Rosas



Diabetic Retinopathy (DR): Microvascular disorder of the retina secondary to chronic hyperglycemia. #1 cause of blindness in the American working age group.

Epidemiology: In the U.S there are 9.6 million people diagnosed with DR. At the time of T2DM diagnosis, 20% of patients have DR. **90% and 60%** of patients with T1DM and T2DM will develop DR by **20 years** from diagnosis, respectively.

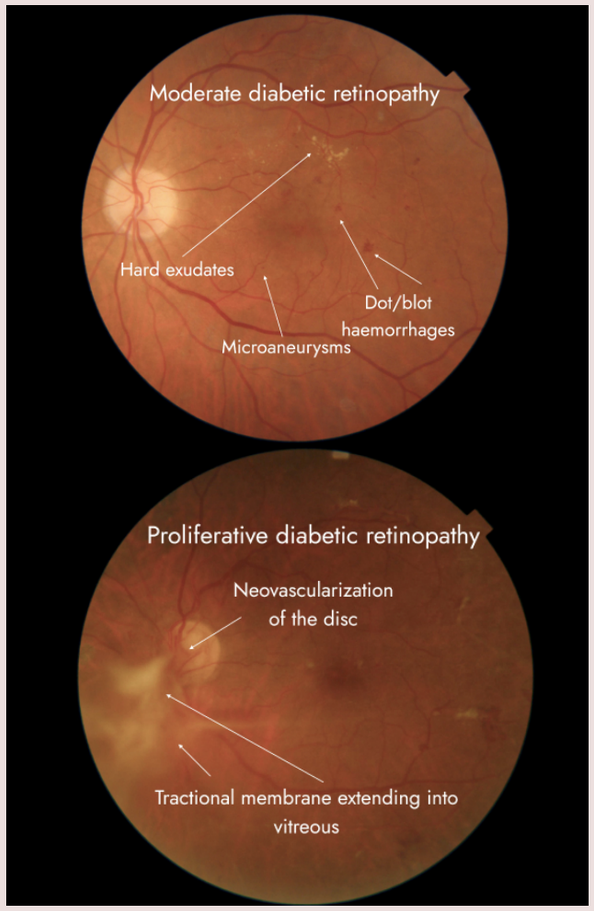
Risk factors:

- **Poor glycemic control + time since diabetes onset** are the two greatest risk factors.
- Male, insulin use, and high systolic BPs, Mexican American, African American.

Pathophysiology:

- Chronic hyperglycemia leads to damage of the retinal micro-vasculature.
- Retinal vessel damage leads to increased vascular permeability, microvascular occlusion, ischemia, and neovascularization.
- **There are two subtypes of DR:**
  - **Non-proliferative diabetic retinopathy (NDPR)**: Includes **micro-aneurysms**, **dot-blot hemorrhages**, and nerve infarcts that manifest as **cotton-wool spots**.
  - **Proliferative retinopathy (PDR):** Involves neovascularization of the retina due to prolonged ischemia and release of **VEGF**.

  Diabetic Macular Edema (DME): a complication of DR. Leakage from blood vessels secondary to inflammation leads to accumulation of fluid under the macula. Remnants of the fluid manifest as yellow, lipid products called **hard exudates.**



Symptoms: Often asymptomatic in early stages, but can progress to visual impairment and blindness.

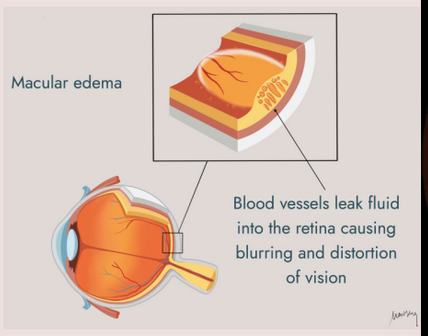
- Symptoms include: blurred vision from lens or macular edema, floaters, flashes of light, and **painless vision loss**.
- Associated complications leading to symptoms: **Macular edema, vitreous hemorrhage, and retinal detachment.**

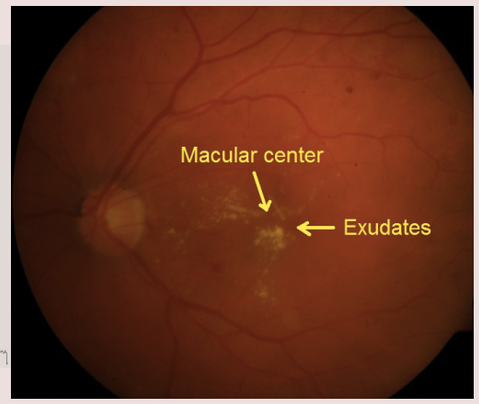
Screening:

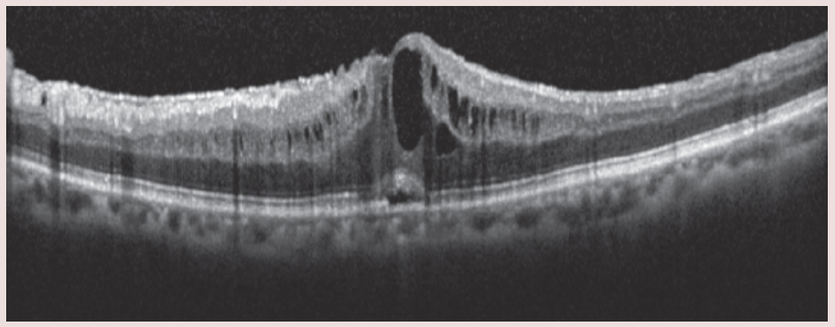
- **T1DM patients:** Within 3-5 years of diagnosis.
- **T2DM patients:** *As soon as diagnosis is made*.
- **Rationale:** DR has a more insidious onset in the early stages of T1DM.
- **Patients with pre-existing diabetes planning pregnancy or pregnant:** Before pregnancy or in 1st trimester.

Diagnosis:

- **Useful tests**: VA, Slit-lamp exam, DFE, OCT, and FA.
- **NPDR:** Mild, moderate, and severe disease.
  - Severe disease (using the **4-2-1 rule**): Any of the following > 4 quadrants of retinal hemorrhages, >/= 2 quadrants of venous bleeding, or 1 or more quadrants of intraretinal microvascular abnormality (IRMA).
- **PDR:** Neovascularization of the retina, vitreous, iris, or disk. Neovascularization of the disk = high-risk for vision loss.
- **DME**: Retinal thickening at or within 500 µm of the center of the fovea, hard exudates at or within 500 µm of the center of the fovea if next to retinal thickening. Seen with OCT macula.







Macular edema seen on OCT

Treatment Goals: Prevent onset, delay progression, preserve and improve vision.

- **General:** Strict glucose control **(HbA1C <7%)** and BP control; may take 4 weeks on adequate treatment for blurry vision to resolve
- **DME:** Intravitreal **Anti-VEGF** agents or laser therapy.
- **PDR:** Pan-retinal photocoagulation (PRP), vitrectomy, or intravitreal **anti-VEGF**.

Prognosis: Severe vision loss can be prevented with appropriate treatment in 90% of cases.

- Increased risk of progression to PDR: higher A1C level, renal impairment, younger age at onset, hyperlipidemia.
- Patients with DR have a higher risk of morbidity/mortality due to increased CV events (eg, MI & Stroke).
- Special populations: Progression of retinopathy is seen in 16-85% of patients who become pregnant.

Clinical Pearls:

Macular edema may develop even months after initial A1c control.
Only 60% of patients with DM receive annual eye exams.
The most common cause of blindness in diabetes is secondary to macular edema.
By 2050, 16.0 million Americans will be DX with DR.

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