

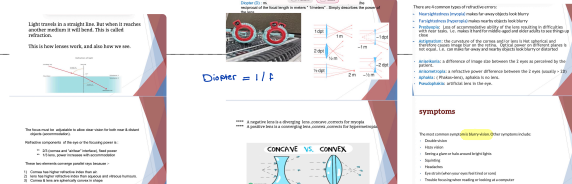
OPHTHALMOLOGY

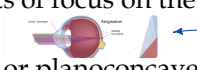
SUMMARY

Done by: Shahed Atiyat

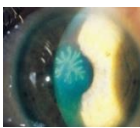
"Strabismus, glaucoma and uveitis are not included in this summary"


Optic and refraction




<p>Hypermetropia (Long-sightedness)</p>	<ul style="list-style-type: none"> Rays of light focus behind the retina causing blurry vision of near objects ± distant objects (low optical power) <i>↳ young children are born with it but compensate with strong accommodation with risk of Esotropia.</i> Causes: normal development of the eye (physiologic), short eyeball, ectopia lentis, post-operative aphakia, +ve family history. <i>= no lens</i> <i>↳ light moves far away</i> Most them born with it, but it may not cause vision problems until they get older. Complication: angle-closure glaucoma & strabismus or amblyopia (in children). <i>Esotropia</i> Corrected by convex lens (converging lens), contact lens or refractive surgery. <i>+ve lens</i> <p><i>* Clinical: - no blurry vision but struggle seeing near objects.</i></p>
<p>Myopia (Short-sightedness)</p>	<ul style="list-style-type: none"> Light focuses in front of retina causing blurry vision of distant objects (high optical power). Usually presents in 1st or 2nd decades, rarely begins after 25, except in DM or cataract. <i>Stops at 20</i> Causes: Keratoconus, cataract, DM, long eyeball. Complication: retinal tear or detachment, macular hole, open-angle glaucoma. Corrected by concave lens (diverging lens), contact lens or refractive surgery. <i>-ve lens</i>
<p>Astigmatism</p>	<ul style="list-style-type: none"> The optical power in different plans is not equal due to non-spherical cornea or lens. Non-spherical cornea or lens >> different points of focus on the retina >> difficult to see fine details, either close up or from a distance.  Corrected with a cylindrical lens (planoconvex or planoconcave) or refractive eye surgery.
<p>Presbyopia <i>aging of the lens after 45.</i> <u>+30 D</u></p>	<ul style="list-style-type: none"> A normal aging process (usually after age 45), due to reduced accommodation ability. The focus behind the retina >> trouble seeing things up close (specifically reading). Needing to hold reading materials farther away to focus on them. Presbyopia gets worse over time, but it usually stops getting worse after age 65. Occur earlier in hypermetropes than myopes.
<ul style="list-style-type: none"> Emmetropia (normal vision): light from distant object focus on the retina with the eye at rest "not accommodating". The visible part of the EM spectrum lies between the wavelengths 390nm & 760nm. Refractive component of the eye (focusing power): 2/3 cornea, 1/3 lens (increase with accommodation). The most common symptom in refractive errors is blurry vision. Aniseikonia: different image size between the 2 eyes as perceived by the patient. Anisometropia: different refractive power between the 2 eyes (usually > 2 Diopter). 	

Cornea & sclera

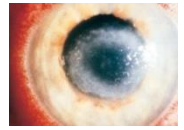
<p>Herpes simplex keratitis</p> <ul style="list-style-type: none"> HSV 1 is the most common cause. Primary infection usually acquired in childhood (may as conjunctivitis) >> resolution & latency of the virus in the trigeminal ganglion. Recurrent infection >> reactivation of latent virus (risk increases if the patient is debilitated (systemic illnesses, immunosuppression) and with stress. Pathognomonic: Dendritic ulcer, heals without scar (seen by fluorescein dye with blue light in slit lamp). May progress to stromal keratitis in severe cases (disciform keratitis) >> corneal edema & permanent scarring >> corneal graft may be needed. Treatment: Topical antiviral (Aciclovir), avoid topical steroid; it causes extensive ulceration. 	
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<p>Herpes zoster ophthalmicus</p> <ul style="list-style-type: none"> Caused by HZV which is responsible for chickenpox. Ocular manifestation usually preceded by pain and vesicles in the distribution of ophthalmic division of trig. nerve. Ocular problems (lid swelling, keratitis, iritis, secondary glaucoma) are more likely if the nasociliary branch is affected (signaled by vesicles at the root of the nose) >> Hutchinson's sign Treatment: Oral aciclovir is effective in reducing post-infected neuralgia if is given within 3 days of vesicles eruption, topical steroid for ocular disease and antibacterials to cover secondary infection. 	
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<p>Bacterial keratitis (90% of corneal infection)</p> <ul style="list-style-type: none"> Prime pathogens: staph.epidermis, staph.aureus, strep.pneumoniae. S&S: severe pain, purulent discharge, ciliary injection, visual loss, hypopyon (WBC in anterior chamber), white corneal opacity. Risk factors: keratoconjunctivitis sicca, breach in the corneal epithelium, contact lens, prolonged use of topical steroids. Treatment: topical broad-spectrum antibiotics (Fluorquinolones), or combined antibiotics. 	
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Acanthamoeba keratitis

- Caused by protozoa.
- Risk factor: **contact lens use (even in immunocompetent)**.
- Cause extremely painful keratitis & ring-shaped infiltration or abscess.
- Treatment: Topical chlorhexidine and propamidine for months, may end with corneal graft.

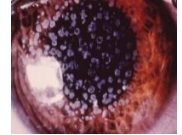


Fungal keratitis

- More common in warmer climates.
- Should be considered in lack of response to antibacterial treatment, trauma by plants, prolonged steroid use.
- Cause corneal opacity that appear fluffy.
- Treatment: Topical and systemic anti-fungal drops (natamycin).

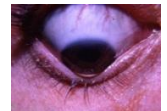
Corneal dystrophies

- Rare inherited disorder, but relatively common in Jordan due to high consanguinity rates.
- Various substances build up in the cornea affecting corneal transparency.
 - ✓ Anterior dystrophy: involves epithelium, cause recurrent corneal erosion.
 - ✓ Stromal dystrophy: cause visual loss due to corneal opacities.
 - ✓ Posterior dystrophy: involves endothelium, cause gradual visual loss due to corneal edema.
- Treatment: no treatment in asymptomatic, **keratoplasty** in symptomatic severe cases.



Keratoconus

- Sporadic disorder, thinning of the center of cornea leading to ectasia and cone-shaped cornea (painless).
- Keratoconus causes visual loss secondary to **progressive irregular myopia & astigmatism**.
- Diagnosed by corneal topography.
- Munson's sign: V-shape lower eyelid.
- Modalities of treatment for KC:
 - ✓ Spectacles or contact lenses.
 - ✓ **Corneal cross linking** (linking of the stromal collagen by exposing UV radiation), stops KC progression.
 - ✓ Intra stromal corneal rings (reduce astigmatism).
 - ✓ Corneal graft
 - Avascular host cornea provides an immune-privileged site for grafting, success rate 80%
 - No need for HLA matching, extracted within 24h after death, can be grafted within 2 weeks from extraction.
 - Use non-absorbable sutures (16 sutures) for 1 year.
 - Topical steroids eye drops are used post-operation to prevent graft rejection.



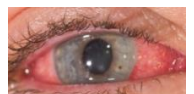
Note: The conjunctiva & cornea are protected against infection by:

- Blinking.
- Washing away of debris by the flow of tears.
- The antibacterial properties of the tears.
- Entrapment of the foreign particles by mucus.
- The barrier function of the corneal epithelium. **Neisseria gonorrhoeae** is the only organism that can penetrate the **intact epithelium**

Indications of corneal graft (keratoplasty): keratoconus, cataract, traumatic scar, herpes infection, corneal dystrophy, decompensated cornea in old age.

Complication of corneal graft: rejection (most common, type IV hypersensitivity reaction), recurrence, cataract, astigmatism, endophthalmitis.

Episcleritis	Scleritis
Inflammation of superficial layer of the sclera causing mild discomfort with segmental redness of the eye	Cause intense ocular pain with generalized redness and swelling of the sclera
Rarely associated with systemic diseases	Usually associated with collagen vascular diseases, most commonly RA
Usually self-limiting	Complication: Scleral thinning (scleromalacia), sometime with perforation/ Keratitis/ Uveitis/ Cataract/ Glaucoma
Topical anti-inflammatory if symptom persist	Mild: Topical steroid & oral NSAID Moderate to severe: High doses of systemic steroids or cytotoxics



General info:
- Densest part: equator, avascular, highly elastic.

Histology of the Lens

- The lens fibers align parallel to the equator.
- Concentrating lens fibers will have their nuclei, but as they age, they become more densely packed and their cytoplasm is filled with protein called crystallins.
- The more lens fibers lose their nuclei and become densely packed to produce a single transparent structure.

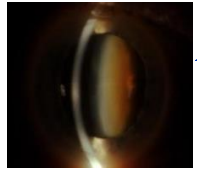
Lens

Other causes of cataract - atopic dermatitis

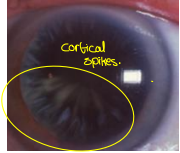
- Anterior subcapsular cataract:
 - Causes: Diabetes in 30%, trauma in 10%, infection in 10%, frequently because mature.
 - Waxes in anterior capsule.
- Posterior subcapsular cataract:
 - Causes: Diabetes in 30%, trauma in 10%, infection in 10%, frequently because mature.
 - Waxes in posterior capsule.

Cataract

- It is the opacification of the lens of the eye (becomes cloudy causing hazy image).
- The commonest cause of treatable blindness worldwide.
- Types:**
 - Nuclear:** the most common type, cloudiness in the center of the lens. *→ nucleus becomes hard due to condensation of lens nucleus and deposition of brown pigment within the lens.* presents with myopia while reading is less affected.
 - Cortical:** opacification of cortex of the lens, opaque cortical spokes (fissuring).
 - Posterior subcapsular:** involve the back of the lens adjacent to the capsule, **RF: prolonged topical steroid.**
- Causes:**
 - Age-related cataract:** the most common cause (senile cataract); due to cumulative exposure to smoking, UV radiation, elevated blood sugar.
 - Ocular conditions: trauma, topical steroids, uveitis, high myopia, ocular surgery or tumor.
 - Systemic diseases: DM, galactocemia, hypocalcemia, fabry disease, atopic dermatitis, myotonic dystrophies, radiation, congenital cataract, TORCH infection, Down syndrome, Lowe syndrome, systemic drugs (steroids, chlorpromazine).
- Symptoms & signs:** *Bilateral*
 - Painless progressive loss of vision, change in refraction (myopia), glare, amblyopia in infant.
 - Decreased visual acuity & lens opacity seen on slit-lamp.
 - Cataract appears black against red reflex (especially if mature).
 - Leukocoria like in congenital cataract, may lead to amblyopia in children.
- Type of surgery:** *The only definitive treatment.*
 - Indication: cataract reducing the quality of life, no need to wait for cataract ripening
 - Phacoemulsification:** preferable, 2 small incisions at the limbus, **no sutures**, emulsify the lens into liquid by the energy of ultrasound wave, early rehabilitation (recovery). *the IOL is placed posteriorly*
 - Extra-capsular cataract extraction (ECCE): extended incision at the limbus and must be sutured, useful when dealing with very hard cataract.
 - Intra-capsular cataract extraction (ICCE): larger incision, high rate of complications.
- Post-operative care:**
 - Short course of steroid and antibiotic drop.
 - Spectacle or multifocal IOL for near vision since the patient cannot accommodate.
- Complication of cataract surgery:**
 - Posterior capsule opacification:** most common (20%), Tx: ndYAG laser. *(out-patient)*
 - Vitreous loss** (risk of glaucoma and retinal traction), Tx: vitrectomy at operation time and delay of IOL placement.
 - Iris prolapse:** due to inadequate suturing or inappropriate management of vitreous loss, Tx: excise prolapsed iris & resuture incision.
 - Endophthalmitis:** rare (0.3%) but **most serious**, within few days, presents with painful red eye & reduced visual acuity & hypopyon, Tx: intravitreal broad-spectrum antibiotic.
 - Staph.epidermidis, staph.ureus, pseudomonas. *Source: 1) Patient own external flora 2) environmental flora 3) instruments*
 - Astigmatism** by tight sutures.
 - Macular edema:** Tx: topical NSAIDs & steroids
 - Retinal detachment**



Nuclear cataract



Cortical cataract



Post. subcapsular



Mature cataract

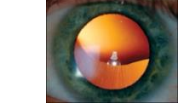


Immature cataract



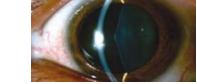
Iris prolapse

Ectopia lentis



- Weakness of the zonules causes lens displacement.
- The lens become rounder and myopic.
- Etiology: trauma (most common), homocystinuria (displaced downward), Marfan syndrome (displaced upward).

Anterior lenticonus

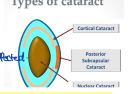


- Abnormal lens shape, the anterior curvature of the anterior part is increased centrally.
- Seen in Alport's syndrome (AR, deafness & nephropathy)

Leukocori



- Abnormal white reflection from the pupil (instead of red).
- Seen in retinoblastomas, congenital cataracts, retinal detachment, glaucoma & corneal dystrophy.



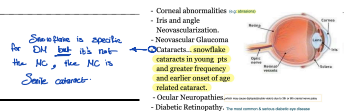
Types of cataract

Phacoemulsification

Extra Capsular Cataract Extraction

Intracapsular cataract extraction

Other causes of cataract - atopic dermatitis



Retina → the most common Microangiopathy in both DM1/2.
 Causes: vision loss/diplopia/pain

Diabetic retinopathy

- It is a **progressive** microangiopathy of the **retinal blood vessels** caused by chronic hyperglycemia.
- The most common cause of moderate to severe vision loss between ages 25 and 74 years.
- Retinal blood supply: **central retinal artery and choroidal circulation.**
- Risk factors: **duration of dm, poor control of dm, HTN, hyperlipidemia, nephropathy, obesity, pregnancy, smoking.**
- 2 stages/types:

* Note: NL optic nerve
 1) pink 2) cup/disk ratio <0.5 3) well circumscribed



Note: Microangiopathy has leakage and obstruction:
 1) leakage → loss of pericytes that leads to microaneurysms and inter blood retinal barrier disruption leads to plasma leakage into retina causing retinal edema + hard exudates.
 2) occlusion → deformed RBCs and platelet stickiness ⊕ VEGF is produced by hypoxic retina = stimulates stunting and growth of new vessels.

- Nonproliferative diabetic retinopathy NPDR (microvascular leakage):**
 Hyperglycemia >> loss of pericytes >> weak retinal vessels leading to:
 - Microaneurysms** this is the first sign. → First place is fovea and macula.
 - Increase vascular permeability >> lipid & protein leakage >> **hard exudate & retinal edema.**
 - Other signs: **dot & blot hemorrhage** (microaneurysms rupture in deep layers), **flame-shaped hemorrhage** (retinal capillaries rupture in superficial layers), **cotton-wool spots** (accumulation of dead nerve cells, fluorescein angio shows no capillary perfusion), **venous changes (heaving and looping)**, **Intraretinal microvascular abnormalities IRMAs** (like abnormal branching adjacent to the areas with non perfusion).

- Proliferative diabetic retinopathy PDR (microvascular occlusion):**
 Basement membrane thickening, endothelial cell damage, deformed RBCs, platelet stickiness & aggregation >> vessels occlusion >> retinal hypoxia >> **VEGF release >> new vessel formation (neovascularization) "hallmark"**
 - These new vessels form at or near the optic disc (NVDs), anywhere in the retina (NVEs) or at iris (NVI)
 - Other signs: **Vitreous hemorrhages, per-retinal & subhyloid hemorrhages** (breaking of the new vessels), **traction retinal detachment** (due to contraction of the fibrous component of the new vessels), **Neovascular glaucoma.**

* Note: we can't differentiate between microaneurysms & dot hemorrhage. clinically we need fluorescein

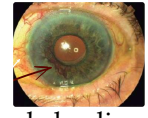
hyper-fluorescent & early filling
 hypo-fluorescent & no filling

- Classification based on severity:
 - Mild NPDR:** microaneurysms only.
 - Moderate NPDR:** Microaneurysms and/or dot and blot hemorrhages in more than 1 quadrant, soft exudate, Cotton wool spots or venous beading in one quadrant.
 - Severe NPDR:** microaneurysms in 4 quadrants or venous changes in 2 quadrants or IRMA in one quadrant.
 - PDR:** neovascularization or Vitreous or pre-retinal hemorrhage.
 - Macular edema:** intraretinal fluid (edema) and retinal thickening involving the macula, diagnosed by OCT.



- Treatment:**
 - Mild & moderately NPDR: Good diabetic control & control associated diseases & regular follow-up (9 months for mild and 6 months for moderate).
 - Significant macular edema: anti-VEGF & localized laser photocoagulation at the point of leakage.
 - Sever NPDR: pan-retinal photocoagulation & anti-VEGF & close follow-up every 4 months.
 - PDR: pan-retinal laser photocoagulation & anti-VEGF.

- Complications:**
 - Vitreous hemorrhage.
 - Tractional retinal detachment.
 - Rubeosis iridis:** Neovascularization of the **iris** and drainage angle leading to increased IOP and progressive glaucoma (angle-closure).
 - Sudden visual loss (due to macular edema [mcc] or ischemia, vitreous hemorrhage, retinal detachment).



Symptoms of diabetic retinopathy may include:
 - Blurred vision
 - The impairment of color vision
 - Flares, or transparent and colorless spots and dark strings that float in the patient's field of vision
 - Streaks or streaks that block the person's vision
 - Poor night vision
 - Sudden and total loss of vision

Investigations:
 IFA, Ind eye
 OCT to assess the thickness of retina to diagnose macular edema
 Fluorescein angiography

Complications of Diabetic Retinopathy:
 - Vitreous hemorrhage
 - Tractional retinal detachment
 - Rubeosis iridis
 - Glaucoma
 - Blindness

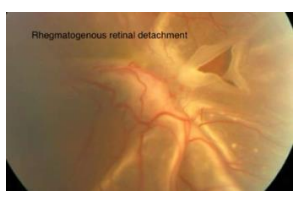
Neovascular Glaucoma:
 - Complication of rubeosis iridis
 - New vessels cause angle closure
 - Mechanical obstruction to aqueous outflow
 - Intra ocular pressure rises
 - Pupil gets distorted as iris gets pulled
 - Eye becomes painful and red
 - Loss of vision

Blindness:
 - Non-clearing vitreous hemorrhage
 - Neovascular glaucoma
 - Tractional retinal detachment
 - Macular ischemia

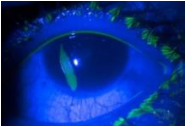
Prevention of Complications:
 1- By early institution of appropriate treatment
 2- Early detection of DM in its asymptomatic/traceable condition
 3- Routine regular examination of all diabetes (at least yearly)
 4- Appropriate referral to ophthalmologist

Retinal detachment

- Detachment of the neurosensory retina from the retinal pigment epithelium, types:
 - Rhegmatogenous retinal detachment** is the most common:
 - Due to retinal **tear** > retinal fluid seeps into subretinal space > detachment.
 - RF: trauma, intraocular surgery, myopia, increased age.
 - Traction retinal detachment:**
 - By **contracting fibrous tissue** on retinal surface like in proliferative diabetic retinopathy.
 - Exudative retinal detachment:**
 - Subretinal **fluid exudate accumulation** without retinal tears
- Symptoms: Prodromal symptoms (light flashes, floaters, no pain or red eye, scotoma, then sudden painless loss of vision).
- Management: surgical repair of the retina, vitrectomy, photocoagulation may be used.





Eye injuries

<p>Corneal abrasions</p> 	<ul style="list-style-type: none"> • Loss of epithelial layer of the cornea. • Etiology: direct injury, prolonged contact lens wear, foreign body under the eyelid, ... • Symptoms: severe eye pain, photophobia, foreign body sensation, epiphora, normal visual acuity, normal pupillary reflex. <i>↳ the nerve endings are exposed</i> • Diagnosis: green staining defect on fluorescein examination. <i>↳ unless the middle part of cornea is affected.</i>
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Vision-threatening conditions:


- Ocular chemical burns ■ Orbital compartment syndrome ■ Open globe injury ■ Traumatic hyphema
- Vitreous hemorrhage ■ Retinal trauma ■ Optic nerve injury ■ Periocular injuries that threaten vision

<p>Ocular chemical burn</p> 	<ul style="list-style-type: none"> • Eye contact with acid or alkali (alkali cause more severe damage than acid). • Symptoms: decreased vision, moderate to severe eye pain, photophobia, conjunctival redness, blepharospasm (inability to open the eye), symblepharon (lid attached to the globe). <i>↳ comes at late complications.</i> • In severe cases of alkali exposure, opacification of the cornea happens with white conjunctiva due to ischemia. <i>of the conjunctiva and scleral vessels.</i> • Management: continuous irrigation (30-60 min) with water or saline, morgan lens can be used, steroids & cyclopentolate. <i>* irrigation should be less forceful if a concomitant globe rupture is suspected.</i>
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<p>Orbital compartment syndrome</p>  <p><i>* immediate intervention. * Dx: clinically</i></p>	<ul style="list-style-type: none"> • Orbital compartment pressure increased causing compressive optic neuropathy and ischemia. • Symptoms: proptosis, afferent pupillary defect, peri-orbital swelling/hemorrhage, eye pain, acute onset of decreased vision, decrease retrobulbar, chemosis, subconjunctival hemorrhage. • Most commonly due to retrobulbar hemorrhage caused by trauma. • May cause irreversible vision loss within 1-2 hours. • Tonometry shows increase in IOP, fundoscopy shows papilloedema. • Management: <ul style="list-style-type: none"> ○ Lateral canthotomy and inferior cantholysis (do not delay the decompression). ○ Elevation of the head of the bed to at least 45 degrees & pain control. ○ Correction of any coagulopathy and/or cessation of anticoagulant therapy. ○ Prevent sudden increased IOP by cough suppression, Antiemetics therapy, stool softeners. ○ Hospitalization for definitive management of the underlying cause.
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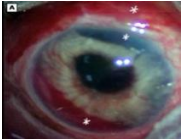
Penetrating injury to the eye *Full thickness.*

- Decrease visual acuity & afferent pupillary defect.
- Teardrop pupil, Seidel sign, decreased IOP.
- Uveal tissue and aqueous prolapse through the wound.
- Give prophylaxis antibiotic and tetanus prophylaxis, keep NPO.
- Rapid primary closure within 24 hours.



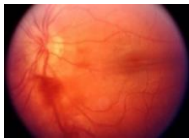
Blunt trauma to the eye

- Subconjunctival hemorrhage superiorly.
- Conjunctival laceration.
- Hyphema
- Iridodialysis superiorly (separation of the iris from the ciliary body due to blunt trauma).



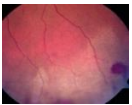
Vitreous hemorrhage

- It is extravasation of blood within or around vitreous body.
- Causes: PDR, retinal vein occlusion, retinal tear or detachment.
- Management: vitrectomy.



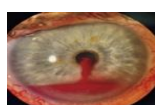
Comotio retinae

- Retinal edema after blunt closed globe injury.
- Asymptomatic or decrease visual acuity.
- Ophthalmoscopy shows retinal whitening with normal vessel +/- retinal hemorrhage.
- Exclude the presence of retinal break or detachment.
- Typically resolve without intervention (observation).



Hyphema *↳ Macroscopic: Gross eye
Microscopic: SLE should used* ** look at the next page*

- Blood in anterior chamber results from a tears in the vessels of the ciliary body or iris.
- Pain, decreased visual acuity, photophobia, and anisocoria, increase IOP.
- MCC is traumatic, Rubiosis iridis is the mcc of non-traumatic.
- Management: monitoring of IOP, eye shield, limitation of activity, cycloplegia to manage pain, glucocorticoid eye drops to lower the risk of rebleeding. Or Surgical clot evacuation if it was: large & persistent or any complication happens.
- Complication: rebleeding, uncontrolled IOP, glaucoma, corneal staining blood.



Optic nerve injury (traumatic optic neuropathy)

- Causes decrease vision, red color desaturation, decrease color vision, afferent pupillary defect.
- Direct injury vs indirect injury (most common).

Periocular injuries that threaten vision

Head trauma can cause a carotid cavernous sinus fistula.

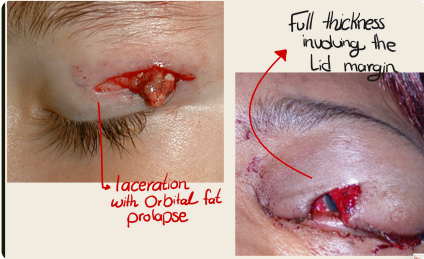
- Retinal vessel distention and high intraocular pressure.
- Mild proptosis and engorgement of the episcleral vessels.
- Chemosis.

Serious condition associated with red eye: scleritis, angle-closure glaucoma, bacterial/viral keratitis, hyphema, hypopyon.

Laceration

1 Eyelid lacerations

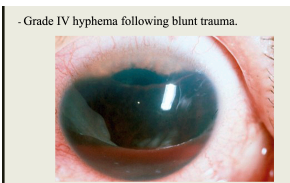
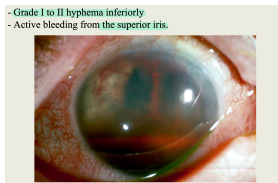
- Ocular injury may accompany eyelid laceration in up to two-thirds of cases.
- Types of eyelid lacerations:**
 - Full-thickness laceration of the eyelid
 - Lacerations with orbital fat prolapse
 - Lacerations through the lid margin
 - Lacerations involving the tear drainage system. *→ at the inner corner of the eye*
 - Lacerations with poor alignment and/or avulsion



Hyphema

Traumatic hyphema: Grading and prognosis

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



Management:

- Surgical clot evacuation:** *surgical aspiration*

Indications:

- Large persistent hyphemas (≥grade III for >10 days)
- Early corneal blood staining
- Uncontrolled intraocular pressure (ie, >50 mmHg for >5 days) despite maximal medical therapy

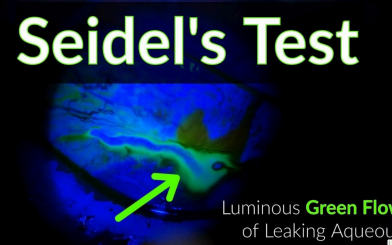
3 Open globe injuries:

open globe injury

- Open globe rupture:** Occurs following blunt eye injury.
- Open globe laceration:** Refers to a penetrating injury to the eye by a sharp object.
 - penetrating (entry wound but no exit wound)
 - perforating (entry and exit wounds)
 - Corneal laceration
 - Corneal-scleral laceration
 - Scleral laceration

Signs & Symptoms:

- Markedly decreased visual acuity
- Relative afferent pupillary defect *+ve RAPD*
- Eccentric or teardrop pupil. *the pupil is malaligned*
- Extrusion of vitreous *= Open globe injury*
- External prolapse of the uvea (iris, ciliary body, or choroid) or other internal ocular structures
- Tenting of the cornea or sclera at the site of globe puncture
- Low intraocular pressure (checked by an ophthalmologist only) *leakage of aqueous humor*
- Seidel sign *Seidel Sign: What it means: Fluorescein dye test shows aqueous humor leaking from the eye, seen as a clear stream through the dye. Why it matters: Confirms active wound leakage - diagnostic for full-thickness perforation.*



Red eye

DDx of Red eye: Benign conditions

- Stye (hordeolum)
- Chalazion
- Blepharitis
- Subconjunctival hemorrhage
- Conjunctivitis
 - Bacterial
 - Viral
 - Allergic
- Corneal abrasion (urgent follow-up if not better in 24 to 48 hours)
- Corneal foreign body (urgent follow-up if not better in 24 to 48 hours)
- Contact lens overwear (urgent follow-up if not better in 24 to 48 hours)
- Dry eye syndrome

Hypopyon

- Leukocytic exudate seen in the anterior chamber.



Acute angle-closure glaucoma

- The conjunctival vessels are dilated, especially near the cornea (ciliary flush).
- The cornea is slightly hazy (edematous).



DDx of Red eye: Serious conditions

Angle-closure glaucoma	Emergency
Hyphema	Emergency
Hypopyon	Emergency
Iritis	Urgent
Infectious keratitis	
Bacterial	Emergency
Viral	Urgent
Scleritis	Urgent

Eyelid & lacrimal system

→ patient presents due to cosmetic / vision impairment

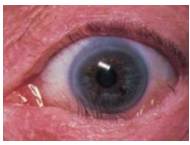
Ptosis



- Abnormally low position of upper eyelid, normally upper lid covers 1-2mm of upper limbus.
- Causes:
 - ① ○ Mechanical; lid edema, lid scarring, lid lumps (hemangioma, tumor, infection, hematoma)
 - ② ○ Disinsertion of levator muscle aponeurosis from its insertion on tarsus (mcc of acquired ptosis).
 → eye might be out and down.
 - ③ ○ Neurological; 3rd nerve palsy, Horner syndrome.
 - ④ ○ Myogenic; myasthenia gravis, muscular dystrophy. / Botulism.
 - Congenital; mc is malformation of the levator palpebrae superioris muscle.
- Signs:
 - ① ○ Palpebral aperture size reduction.
 - ② ○ Lid creases are absent or in abnormal position. (high).
 - ③ ○ Horner's (miosis, anhidrosis), 3rd nerve palsy (mydriasis, diplopia), myasthenia gravis (fatigue after repeated movement).
- Management: manage the underlying cause, **otherwise do surgery.**
- **In children if visual axis is covered >> amblyopia (lazy eye).**

* Note: Management of MG:
1) Neostigmine
2) Thymectomy.

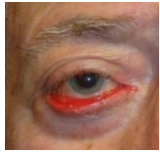
Entropion



Friction of lashes against cornea causing trichiasis. chronic irritation.

- Inturning of the lid margin and lashes into the globe, usually lower lid.
- ① • Spasmodic Entropion: tightly closing the eye. due to pain / irritation.
- ③ • ^{Related to age.} Involutional Entropion: in elderly due to weakening of the lower lid retractors, so the preseptal part of the orbicularis muscle override the pretarsal muscle.
- Cicatricial Entropion: due to conjunctival scarring. from trauma / infection / Stevens-Johnson.
- Management:
 - Taping of lower lid and lubrication (short-term treatment).
 - Botulinum toxin into the palpebral part of orbicularis muscle.
 - Surgery. → Suture eversion of the lid, indications: 1) Recurrent erosions 2) Recurrent infra 3) uncontrolled eye irritation.

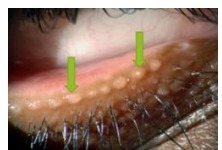
Ectropion



- Eversion of the lid away from the globe.
- Causes:
 - Age-related orbicularis muscle laxity. (Acquired ectropion).
 - 7th nerve palsy. (paralytic ectropion)
 - Scarring of the periorbital skin. (Cicatricial ectropion) * (Congenital ectropion) → Down / Buphthalmos Syndrome.
- **Symptoms: epiphora due to decreased tear drainage, dehydration, irritable eye.**
- Surgical management is an effective treatment & lubrication.

clinical features: 1) conjunctival hyperemia
2) blurry vision
3) photophobia.

Blepharitis



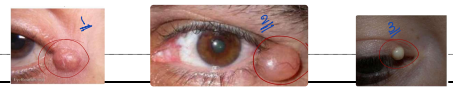
- Chronic inflammation of the lid margins, need long-term treatment.
- Associated with atopic eczema, seborrheic dermatitis & rosacea.
- Cause red, sore & itchy eye, **worse in morning.**
- **Anterior blepharitis:** (Skin / eyelash / follicles).
 - Inflammation concentrated along the eyelashes.
 - Associated with Staphylococcus overgrowth which can cause marginal keratitis (ulceration of the peripheral cornea).
 - Signs:
 - ≡ Scaling and redness of the lid margin.
 - ≡ Ulceration (Staph infection).
 - ≡ Collarettes formation (cylindrical dandruff)
 - ≡ Reduction in the eyelashes number.
 - Management:
 - Lid toilet with bicarbonate solution or baby shampoo; to remove the squamous debris.
 - Topical steroid.
 - Topical antibiotic (fusidic acid gel) in case of staph. infection.
- **Posterior blepharitis:** (Meibomian gland).
 - Caused by meibomian gland dysfunction due to obstruction by squamous debris.
 - Signs:
 - Plugging of meibomian orifices.
 - Thickened, cloudy meibomian secretion.
 - Injection of the lid margin and conjunctiva.
 - Management:
 - Lid massage after hot bathing & artificial tear in case of dry eye.
 - Topical azithromycin drops.
 - Oral doxycycline or tetracycline.



Benign lumps

Malignant

Note: we have 3 types of cysts:
 ① Sebaceous: oblique + excised for cosmetic causes
 ② Cyst of Moll: Translucent due to sweat gland obstruction
 ③ Cyst of Zeis: oblique due to accessory sebaceous gland obstruction



lashes abnormality

Trichiasis



- Eyelashes are directed towards the globe with normal lid margin >> irritation and abrasion.
 - Associated with trachoma (chlamydia trachomatis) in developing world. * Patients complain of irritation and foreign body sensation.
 - Treatment: epilation of the offending lashes, cryotherapy or electrolysis for recurrent cases.
- RF: 1) MC: chronic blepharitis 2) Trachoma 3) Trauma 4) Idiopathic.

Chalazion

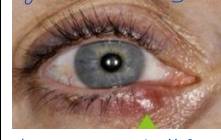


* Note: involvement of Zeis gland is sup chalazion vs Meibomian is deep chalazion.

- Lipo-granuloma within the tarsal plate due to meibomian gland obstruction.
- Painless lid swelling usually resolves within 6 months.
- If persistent >> incised and drainage.

Hordeolum

↳ common acute inflammation of the sebaceous glands around the eyelid follicle (caused by staph aureus).



↳ According to location
 1) External: Zeis gland on ant eyelid
 2) Internal: meibomian gland on post eyelid.

- Internal hordeolum (abscess): painful abscess within meibomian gland.
 - Managed by drainage and topical antibiotics.
- External hordeolum (stye): very painful abscess of eyelash follicle.
 - Managed by eyelash removal, hot compressor and topical antibiotic.

Molluscum contagiosum



- Umbilicated lesion found at the lid margin caused by DNA pox virus. often clustered.
- Most common in childhood (<5 years) & early adolescence, male > female.
- It causes irritation of the eye and small elevation of lymphoid tissue (follicular conjunctivitis).
- Transmission: Direct skin contact, auto-inoculation, fomites.
- Risk factors: immunosuppression, active atopic dermatitis, warm & humid climates.
- Treatment: excision the lesion. ↳ 20% of HIV have this.

Squamous cell papilloma (skin tags)



- Caused by HPV.
- Fibrovascular core and thickened squamous epithelium. ↳ pedunculated.
- Excised for cosmetic reasons.

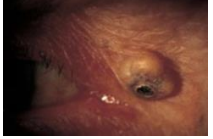
Xanthelasma



- Lipid-containing lesion.
- Usually bilateral.
- Hypercholesterolemia.
- Check blood cholesterol.
- Excised for cosmetic reasons.

Keratocanthoma

↳ well differentiated squamoproliferative keratotic lesion that is now considered a low risk histological variant of SCC.



- Brownish fast-growing (3-6 weeks) lesion with a central crater, filled with keratin.
- Do histology; may have a malignant feature.
- Excision; spontaneous regression led to scar.

Nevus (mole)



- Derived from naevus cell (altered melanocytes)
- Can be pigmented or not.
- No treatment is necessary. ↳ May be congenital or acquired.

Basal cell carcinoma



- The most common malignancy of the eyelid (90%).
- Slowly growing, locally invasive, non-metastasizing.
- Painless nodule may be nodular, scaly or ulcerative (rodent ulcer). ↳ Fitzpatrick types 1-2
- RF: fair-skinned individuals, Hx of prolonged sun exposure, UV radiation.
- Treatment: excision with safe margins, Mohs surgery, cryotherapy or radiotherapy.
- Good prognosis.

Squamous cell carcinoma



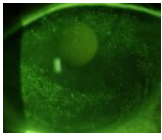
- More malignant & faster growing, metastasize to regional lymph nodes.
- Hard nodule or scaly patch.
- It can arise de novo or from actinic keratosis (pre-malignant lesion).
- UV exposure is important risk factor.
- Treatment: excision with safety margins.

Lacrimal system

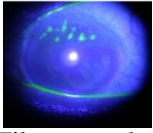
Lacrimal diseases

- *Abnormalities of the lacrimal gland and system:
1. Tear flow and evaporation. ↳ leads to surface hyperosmolarity = ocular damage + visual loss (Keratoconjunctivitis Sicca).
 - Aqueous deficient - dry eye (deficient lacrimal gland secretions), Causes:
 1. Normal with age.
 2. Sjögren's syndrome. ↳ associated with dry mouth and meibomian gland dysfunction. ↳ with SLE, RA, Scleroderma etc: decreased lacrimal functions, T cell infiltrates in minor salivary gland tissue, Anti-Rib and Anti-La Abs.
 - Inadequate mucous production, causes:
 1. loss of goblet cells.
 2. Drainage of tears.
 2. Disorders of the tear drainage
 - Obstruction of tear drainage (most common site is nasolacrimal duct)
 - Infection - Dacryocystitis

Aqueous deficient - dry eye



Punctate staining



Filamentary keratitis

- Symptoms:
 - Grittiness, burning, photophobia, heaviness in the lids and ocular fatigue.
 - Symptoms worse in the evening
 - In severe cases: reduced visual acuity due to corneal damage.
- Signs with fluorescein stain:
 - Early break up of the tear film after 5 seconds of blink suppression
 - Punctate staining of the eye with fluorescence will show small dots of fluorescein over the exposed corneal or conjunctival surface
 - Filamentary keratitis (tags of abnormal mucus).
- Treatment:
 - Artificial tears, but if was severe:
 - Shielded spectacles to create humid environment around the eye.
 - Surgery: to occlude the puncta either temporarily or permanently, or to correct the position of the eye lids.



Sjogren's syndrome

- Chronic autoimmune diseases lead to deficient lacrimal gland secretions.
- More common in older women.
 - Primary >> idiopathic
 - Secondary >> associated with other autoimmune disease like RA, SLE, scleroderma.
- Diagnosis:
 - Decreased salivary and lacrimal functions.
 - T-cell infiltrates in minor salivary gland.
 - Anti-Rho and anti-La titers.
- Management: artificial tears, shielded spectacles, puncta plugs or surgical occlusion, topical anti-inflammatory (cyclosporine).

Evaporative dry eye

- A. Inadequate Meibomian oil delivery (MGD):**
- Extensive Meibomian gland obstruction > deficient tear film lipid layer > water loss from eyes.
 - Treatment as in posterior blepharitis.
- B. Malposition of the globe or lid Margin:**
- Ectropion
 - Lagophthalmos: Incomplete lid closure as in 7th CN palsy.
 - Proptosis
 - Infrequent blinking (Parkinson's)
- Management:**
- Correction of lid deformity
 - Artificial tears and lubricants
 - Local Botulinum toxin injection into the levator muscle in case of incomplete closure
 - Lateral Tarsorrhaphy

Infantile Drainage Obstruction

- The nasolacrimal system canalizes and become patent near term period.
- Watery eyes will result from failure of the distal end of the nasolacrimal duct to canalize.
- Obstructed canaliculi may result in mucocele or dacryocystitis.
- The conjunctiva is not inflamed.
- Dx: pressure over lacrimal sac >> discharge from puncta
- Treatment: mostly resolves spontaneously in the 1st year of life, If not > probing to perforate the occluded membrane through the lacrimal duct

Adult Drainage Obstruction

- Most common site of obstruction is the nasolacrimal duct.
- Causes: infections, direct trauma, fractures, topical drugs.
- Present with watery eyes, stickiness and white eyes, worse in cold or windy weather.
- Stenosed punctum can be seen on slit lamp.
- Patency of the Nasolacrimal system can be assessed by:
 - Syringing normal saline into the canaliculi.
 - Fluorescein dye disappearance after 5 minutes.
 - Dacryocystogram: radio-opaque dye followed by X-ray.
 - Dacryoscintigram: Radioactiveisotope followed by gamma camera
- Management: Dacryocystorhinostomy.

Dacryocystitis




- Infection of the lacrimal sac, secondary to obstruction of the nasolacrimal duct at the junction of the lacrimal sac.
- Presentation: pain, redness, swelling over the inner aspect of the lower eyelid and epiphora
- Mostly due to staph (step. aureus) or strep
- Management: admission, systemic antibiotic, incision and drainage, dacryocystorhinostomy.

The Orbit

- Size is 30cc, 4 sided pyramid shaped

- The orbit consists of 7 bones:
 1. Frontal bone.
 2. Ethmoid bone.
 3. Lacrimal bone.
 4. Sphenoid bone.
 5. Maxillary bone.
 6. Palatine bone.
 7. Zygomatic bone.

- The major nerves and vessels enter through:
 - ✓ **Optic canal:**
 - **At the posterior apex of the orbit.**
 - **Transmits optic nerve.**
 - ✓ **The superior and inferior orbital fissures:**
 - Transmits blood vessels and cranial nerves from the brain to the orbit (and from face and venous drainage back to the brain).



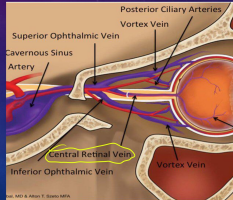
Optic canal • Runs obliquely through the lesser wing of the sphenoid bone

- **Optic nerve (CN II)**
- **Ophthalmic artery**

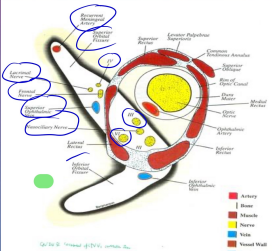
• **The optic canal:**

- At the apex of the orbit within the sphenoid bone.
- The structures that enter through it:
 1. Optic nerve.
 2. Ophthalmic artery.
 3. Central retinal vein.

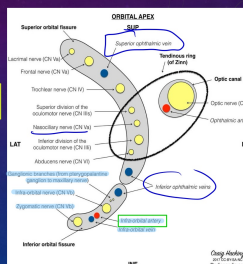
Ophthalmic Artery (maxillary branch)



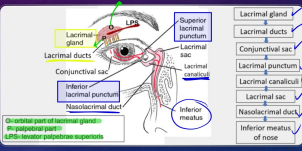
- **The superior orbital fissure:**
- Bounded by the lesser and greater wings of the sphenoid.
- The structures that pass through it:
 1. Cranial nerves (CN) (3, 4, 6)
 2. **Lacrimal nerve**
 3. Frontal nerve
 4. **Nasociliary nerve**
 5. Superior ophthalmic vein
 6. Orbital branch of middle meningeal artery.
 7. Recurrent branch of lacrimal artery
 8. Superior orbital vein



- **The inferior orbital fissure:**
- Formed by the greater wing of the sphenoid, the maxilla and the palatine bone.
- The structures that enter through it:
 1. Infraorbital nerve.
 2. Zygomatic nerve
 3. Parasympathetic fibers (to lacrimal gland)
 4. Infraorbital artery (from maxillary artery).
 5. **Infraorbital vein.**
 6. Inferior ophthalmic vein.

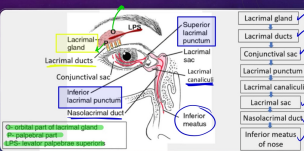


- **The lacrimal gland:**
- Lies anteriorly in the superolateral aspect of the orbit.
- Lacrimal sac lies On the anterior part of the medial wall.



Lacrimal gland	Lacrimal gland
Lacrimal ducts	Lacrimal ducts
Conjunctival sac	Conjunctival sac
Lacrimal sac	Lacrimal sac
Lacrimal canaliculi	Lacrimal canaliculi
Lacrimal punctum	Lacrimal punctum
Nasolacrimal duct	Nasolacrimal duct
Inferior meatus	Inferior meatus

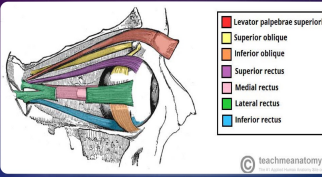
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Lacrimal canaliculi	Lacrimal canaliculi
Lacrimal punctum	Lacrimal punctum
Nasolacrimal duct	Nasolacrimal duct
Inferior meatus	Inferior meatus

- **Function of the orbit?**
- Protection of the **globe.**
- Provides attachment of the muscles which stabilize ocular movement.
- (The 6 ocular muscles originate at the apex around the optic nerve and insert into the globe.)
- A conduit for the transmission of nerves and blood vessels.

origin apex

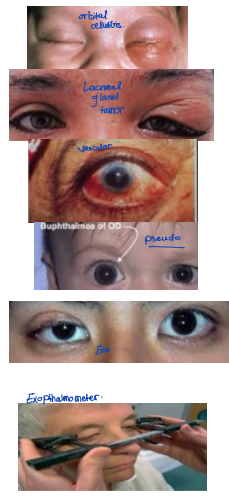


Levator palpebrae superioris
Superior oblique
Inferior oblique
Superior rectus
Medial rectus
Lateral rectus
Inferior rectus

The orbit

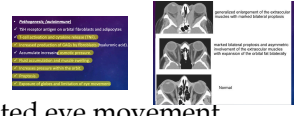
Proptosis

- Some causes:
 - Orbital cellulitis:** mcc of unilateral proptosis in children. MC is a spread from Rhinosinusitis.
 - Thyroidopathy: mcc for proptosis in adults is **Graves' disease** [exophthalmos].
 - Neoplasm:**
 - Intra-canal lesion: eye is displaced forward, like **meningioma**.
 - Extra-canal lesions: eye is displaced to one side, like **lacrimal gland tumor** (to nasal side).
 - In children, rapidly developing Proptosis; **rhabdomyosarcoma**. (MC malignant tumor in children (vs. capillary hemangiomas as a benign)).
 - Orbital venous varix:** **transient** proptosis induced when increasing cephalic venous pressure (Valsalva maneuver).
 - Pseudoproptosis** (buphthalmos, contralateral enophthalmos, contralateral blepharoptosis, ipsilateral lid retraction, axial myopia).
 - Traumatic**, orbital/facial fracture. * Note: Acute fracture → Proptosis, Late fracture → Enophthalmos.
- How to measure proptosis? By **hertel exophthalmometer** (distant from the lateral orbital rim to the apex of the cornea), normal range 10-21 mm.
 - Mild: 21-23 mm, moderate: 24-27 mm, severe: 28 mm or more.
 - A difference of more than 2 mm between the two eyes is significant.



Thyroid eye disease

- MCC of unilateral or bilateral proptosis in adults** (mainly hyperthyroidism, may be hypo).
- Inflammation >> extra-ocular muscles and soft tissue swelling.
- Signs & symptoms of Graves ophthalmopathy:
 - Painful eye, peri-orbital edema, proptosis, chemosis, diplopia.
 - Reduced visual acuity, conjunctivitis, lid lag, lid retraction, restricted eye movement.
- Complication: **corneal ulcer and perforation, compressive optic neuropathy** (vision loss).
- Tx of urgent complications:** systemic steroids, radiotherapy, surgical orbital decompression.
- Assessment of severity by **Werner's classification (NO SPECS)**
 - 0: No signs or symptoms
 - 1: Only signs
 - 2: Soft tissue involvement
 - 3: Proptosis
 - 4: Extra-ocular muscle involvement
 - 5: Corneal involvement
 - 6: Sight loss (optic nerve inv.)



Enophthalmos

Seen in:

- Orbital fractures (blowout fracture).
- Horner's syndrome (pseudoenophthalmos).
- Orbital fat atrophy. (FD).
- Congenital abnormality.
- Metastatic disease. (Breast cancer).



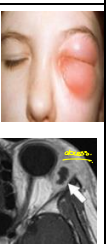
Preseptal cellulitis

- Inflammation of soft tissue **anterior to orbital septum**.
- Etiology: periorbital trauma or dermal infection
- Unilateral painful, erythematous, swollen lid, **no proptosis, normal extra-ocular muscle & visual acuity**.
- The globe is not affected.
- Clinical diagnosis.
- Tx: oral antibiotics amoxicillin with clavulanic acid
- If severe or child <1 year give IV antibiotics. it may turn to orbital cellulitis quickly.



Orbital cellulitis *Emerg*

- Inflammation of soft tissue **posterior to orbital septum**. *Staph/strep infection*
- Etiology: bacterial rhinosinusitis, preseptal cellulitis.
- Unilateral painful, erythematous, swollen eye, fever, **proptosis, ophthalmoplegia, decreased visual acuity**.
- Dx: MRI/ CT (cathartone + varco) for 1 week.
- Tx: Admission, IV antibiotics, drainage of abscess if present, optic nerve decompression if it's compromised.
- Complication: optic nerve inflammation, cavernous sinus thrombosis, meningitis, brain abscess, vision loss. / optic neuropathy (RAPD etc.)



Orbital tumor

- Lacrimal gland tumors:** malignant tumor has poor prognosis, benign tumor has risk of malignant transformation.
- Optic nerve gliomas:** may be associated with neurofibromatosis
- Meningioma:** arising from the middle cranial fossa, difficult to excise, may spread by optic canal to the orbit.
- Lymphoma**
- Rhabdomyosarcoma:** It's the **commonest malignant primary** orbital tumor in **children**, rapidly growing, chemotherapy is effective if it is localized.
- Capillary hemangiomas** as the **most common benign** orbital tumors affecting **children**
- Metastasis:**
 - Children:** Most commonly from **Neuroblastomas**, Ewing sarcoma, Wilms tumor, and leukemias.
 - Adults:** Breast, lung, prostate, or GI tract



- check the picture

Dermoid cyst



- Congenital lesions are caused by the continued growth of ectodermal tissue beneath the surface, which may present in the medial or lateral aspect of the superior orbit.
- Excision is usually performed for cosmetic reasons and to avoid traumatic rupture, which may cause scarring.
- Some may be attached deeply by a stalk, and a before surgery to identify this deeper CT scan may be necessary.

Sudden loss of vision

Sudden transient (<24 h)

Vascular:

- Carotid pathology
- Cardioembolic emboli
- GCA
- Vasospasm

Neurogenic:

- Retinal migraine

Ophthalmic:

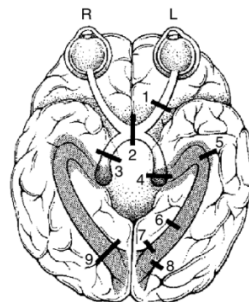
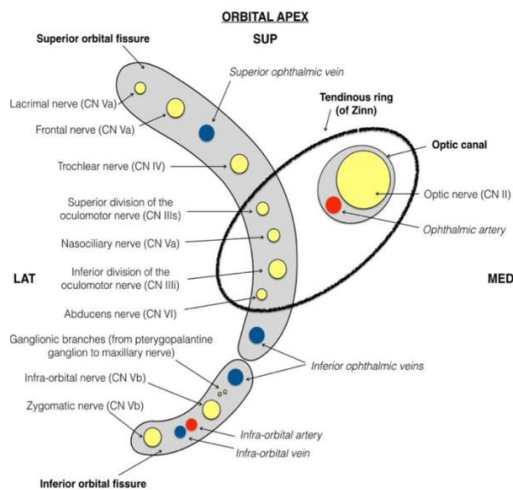
- Papilledema
- Optic disc drusen
- Subacute (intermittent) angle-closure glaucoma

Sudden persistent (>24 h)

- Ischemic optic neuropathy
- Angle-closure glaucoma (painful)
- Microbial keratitis
- Acute anterior uveitis
- Endophthalmitis
- Hyphema
- Vitreous hemorrhage
- Rhegmatogenous retinal detachment (painless)
- Central & branch retinal artery occlusion (painless)
- Central & branch retinal vein occlusion (painless)
- Anterior ischemic optic neuropathy
- Optic neuritis (painful)
- Hydrops Keratoconus (painful)

Gradual loss of vision

- Refractive error
- Keratoconus
- Corneal edema
- Corneal scar
- Corneal infection (Keratitis)
- Corneal dystrophies
- Corneal degeneration
- Cataract
- Ectopia lentis
- Change in shape (Anterior & posterior lenticonus)
- Vitritis (Infection: Toxoplasmosis, endophthalmitis. Autoimmune: Behçet disease, Sarcoidosis)
- Diabetic retinopathy
- Retinal vein occlusion (central and branch)
- Age related macular degeneration (AMD)
- Acquired maculopathies: macular hole, epiretinal membrane
- Posterior & anterior uveitis (painful)
- Retinal dystrophies (Retinitis pigmentosa)
- Macular dystrophies (Stargardt's disease)
- Open-angle glaucoma



Location

Field Defect

	Left Eye	Right Eye	Comment
1 Left Optic Nerve			No light perception left eye
2 Chiasm			Bitemporal hemianopia
3 Right Optic Tract			Incongruous left homonymous hemianopia
4 Left Lateral Geniculate Nucleus			Right homonymous sectoranopia (lateral chorioid artery) - or -
5 Left Temporal Lobe			Incongruous right homonymous hemianopia
6 Left Parietal Lobe			Right homonymous upper quadrant defect ("pie in the sky")
7 Left Occipital Lobe (upper bank)			Right homonymous lower quadrantanopsia (macular sparing)
8 Left Occipital Lobe (lower bank)			Right homonymous upper quadrantanopsia (macular sparing)
9 Right Occipital Lobe			Left homonymous hemianopia (macular sparing)

Notes:

- **Contraindications for LASIK surgery:**
Keratoconus, unstable refractive error, pregnancy, severe dry eye syndrome, autoimmune diseases.
- **Side effects of Acetazolamide:**
Hypokalemia, renal tubular acidosis causing hyperchloremic non-anion gap metabolic acidosis.
- **Side effects cyclopentolate:**
Elevation of IOP, burning, irritation, photophobia, blurred vision, superficial punctate keratitis.
- **Side effects of topical prednisolone:**
Blurry vision, increased IOP, burning sensation, eye irritation.
- **Causes of anisocoria:**
Anterior uveitis, acute angle closure glaucoma, eye trauma, Horner syndrome.
- **Causes of vision loss in graves patient:**
Optic compressive neuropathy, corneal ulceration or infection.
- **Causes of abducens nerve palsy (CN VI):**
Trauma, Psudotumor cerebri, cavernous sinus thrombosis, diabetic retinopathy, giant cell arteritis, vasculitis.
- **Steps of phacoemulsification:**
Anesthesia > corneal incision > Capsulorhexis > phacoemulsification > irrigation & aspiration > lens insertion at posterior chamber.
The final step is to inject salt water in to the corneal wounds to cause the area to swell and seal the incision.
Advantages: no sutures, 2 small incisions.
- **Causes of amblyopia:**
A vision impairment of one eye (also called "lazy eye"), due to abnormal communication between the eye and the brain. May develop as a result of uncorrected strabismus, cataract, or refractive errors.
- **Test to confirm glaucoma:**
Gonioscopy, Tonometry, visual field examination, optic nerve head examination.
- **Normal characteristics of the cornea:**
 - Diameter: 11-12mm, thickness: 0.5 mm.
 - 5 layers: epithelium (stratified squamous non-keratinized, regenerates from the stem cells in the limbus), bowman membrane (acellular layer of collagen) , stroma (90% of corneal thickness), descemet membrane (tough), endothelium (monolayer of non-regenerating cells, actively pump water from the stroma to control hydration of the cornea).
 - Avascular, epithelium and ant.stroma derives its nutrition from ambient air and tear film, post.stroma and endothelium from aqueous humour.
 - Rich in sensory nerve endings originating from the nasociliary branch of trigeminal nerve
- **Normal characteristics of the lens:**
 - Avascular, derived from ectoderm, highly elastic, harden with age.
 - The lens capsule is a thick, homogenous external lamina formed by proteoglycans & collagen IV.
 - The lens epithelium is a single layer of cuboid cells, present only on the anterior surface of the lens.
 - Differentiating lens fibers: elongated nucleated cells filled with crystalline.
 - Mature lens fibers: no nuclei, densely packed to produce a unique transparent structure.
- **The orbit consists of 7 bones:**

Frontal bone, Ethmoid bone, Lacrimal bone, Sphenoid bone, Maxillary bone, Palatine bone, Zygomatic bone.

- **Structure passes through optic canal:**

Optic nerve, Ophthalmic artery, Central retinal vein

- **The lacrimal gland:** Lies anteriorly in the superolateral aspect of the orbit. lacrimal sac lies On the anterior part of the medial wall.
- The 6 ocular muscles originate at the apex around the optic nerve and insert into the globe.

Final past papers: <https://doctor2020.jumedicine.com/wp-content/uploads/sites/12/2024/07/Ophthalmology-final-past-papersedited.pdf>

Best of luck