AN OVERVIEW OF CLINICAL OPHTHALMOLOGY FOR FAMILY DOCTORS

Andrew and Stephanie Palmier

Eyelid pathology

Dry eye syndrome and lacrimal disorders

Conjunctivital and scleral disorders

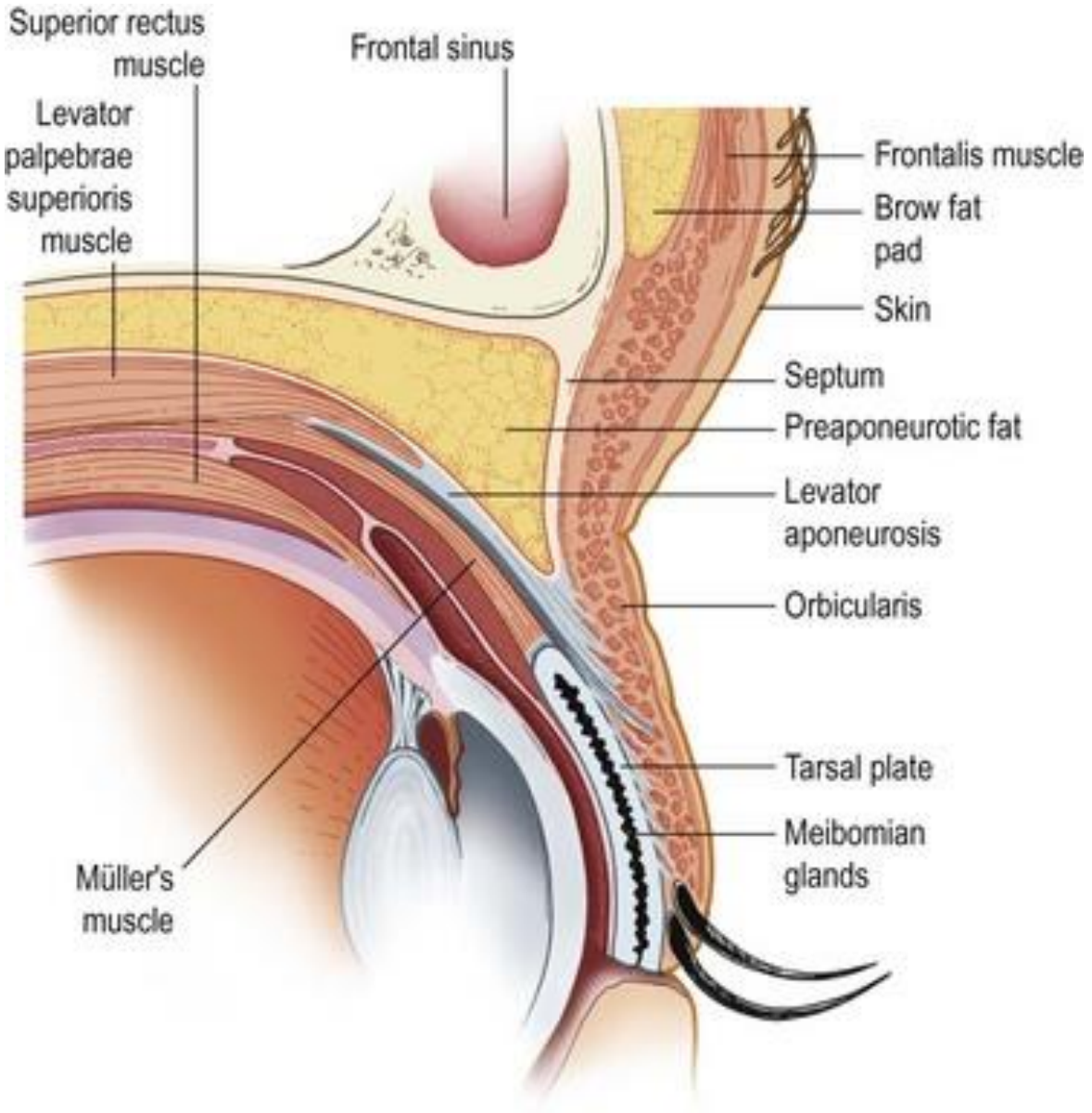
Corneal pathology

Trauma

Thyroid eye disease

EYELID PATHOLOGY

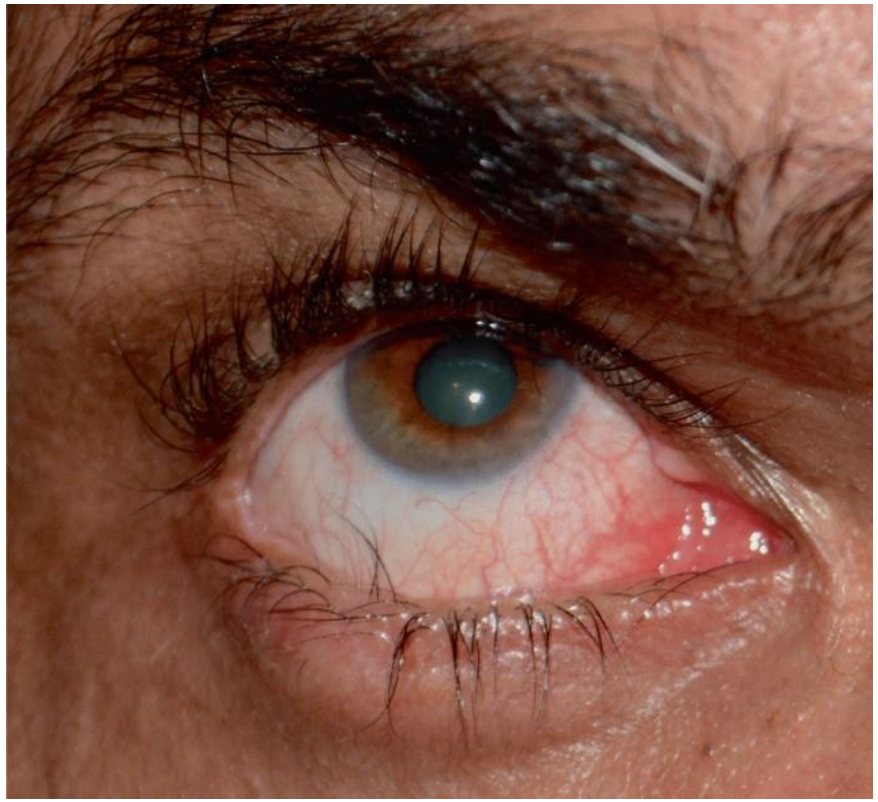
EYELID ANATOMY

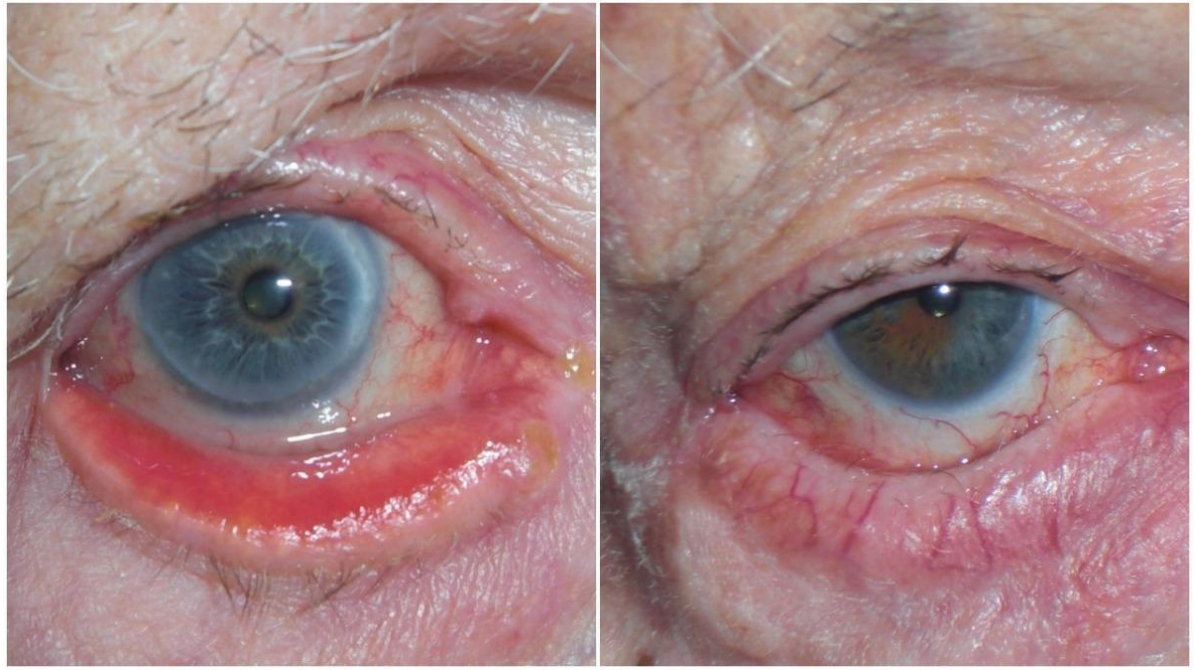


SOME EYELID CONDITIONS A GP MAY ENCOUNTER... HOW TO MANAGE?

- Trichiasis (ingrown lashes)
- Entropion vs ectropion- both require referral for surgical correction
- Blepharospasm, hemi-facial spasm, eyelid myokymia
- Ptosis- to determine the cause assess the pupil for;
    - dilatation (III CN palsy),
    - constriction (Horner's syndrome)
    - normal (congenital, age, MG, myopathy, muscular dystrophy)

SOME EYELID CONDITIONS A GP MAY ENCOUNTER... HOW TO MANAGE?





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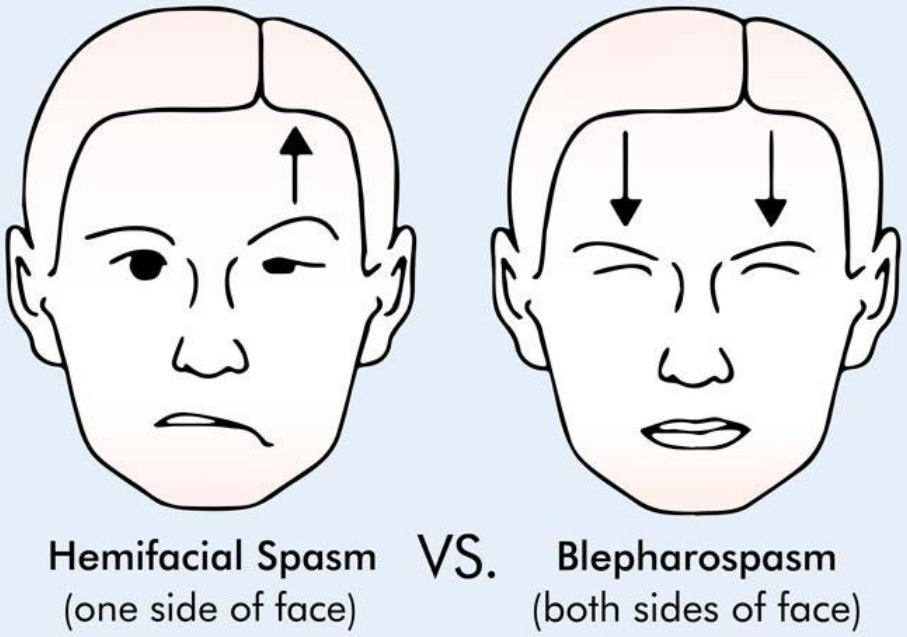
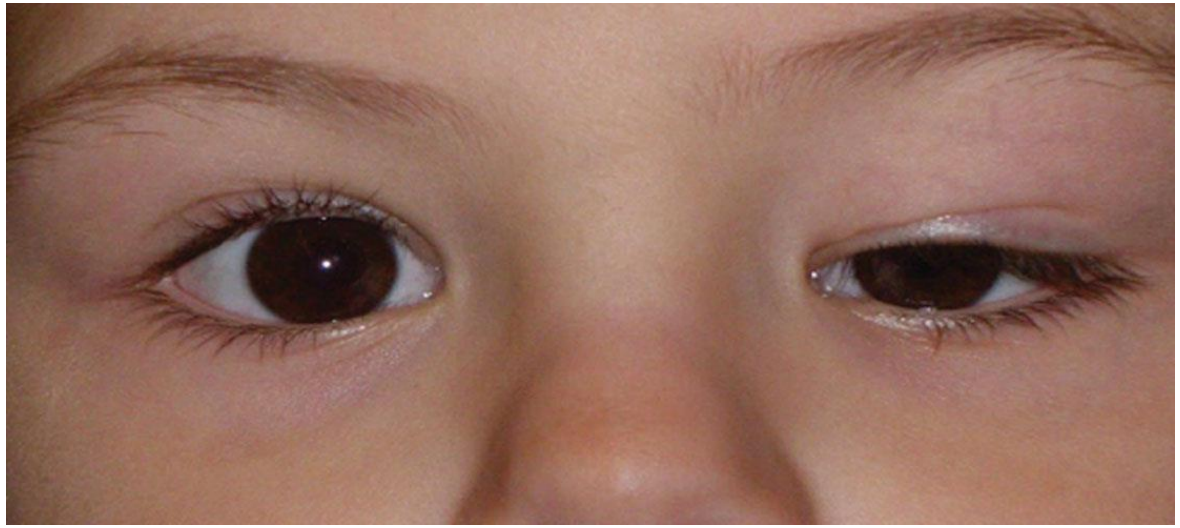
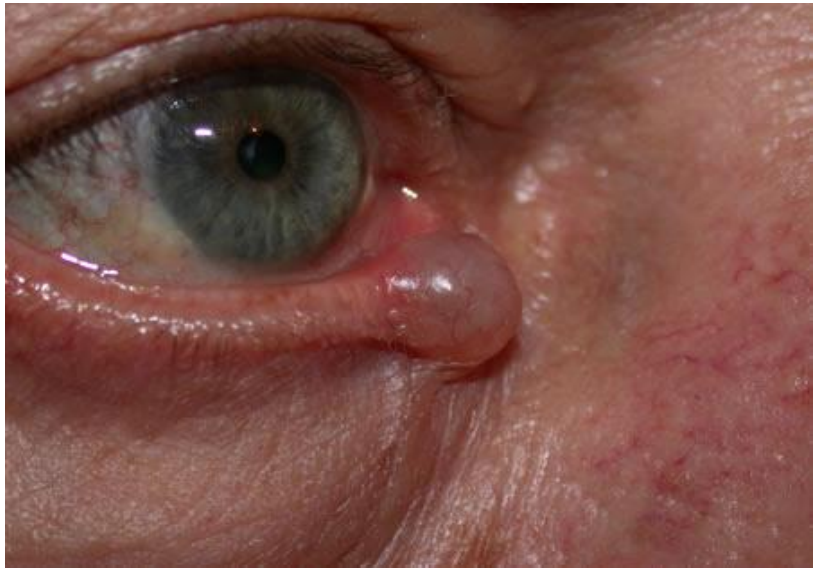


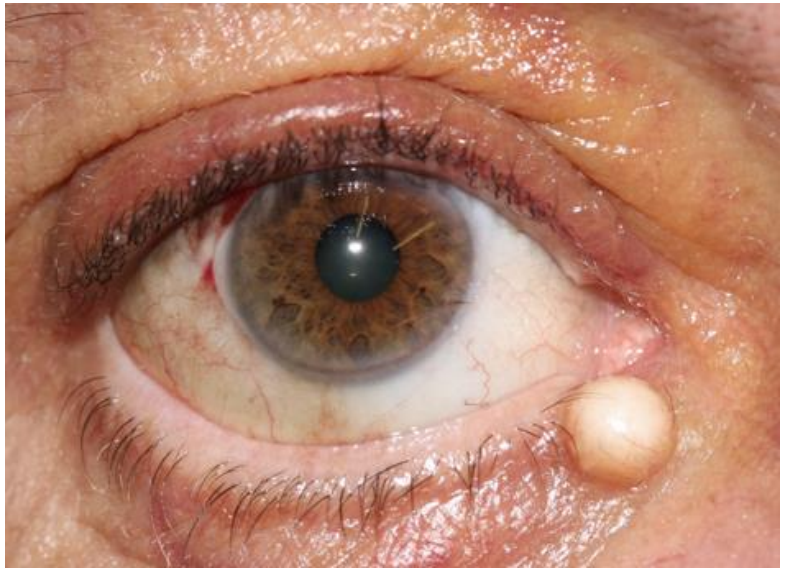


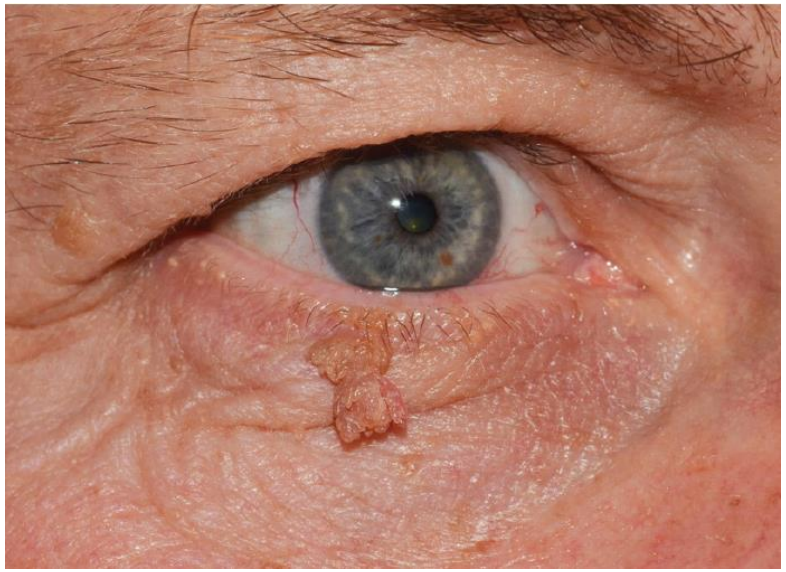
SOME EYELID CONDITIONS A GP MAY ENCOUNTER... HOW TO MANAGE?

- Stye- external vs internal hordeolum
- Chalazion/Meibomian cyst
- Papilloma
- Cyst of Moll and of Zeiss
- Sebaceous cyst
- Dermoid cyst (upper inner and outer angles of the orbit)
- Xanthelasma
- Tumours e.g. BCC at lid margins

BENIGN EYELID LESIONS







EYELID TUMOURS

Examination of the lesion includes assessment for:

-General appearance of the lesion and periocular skin

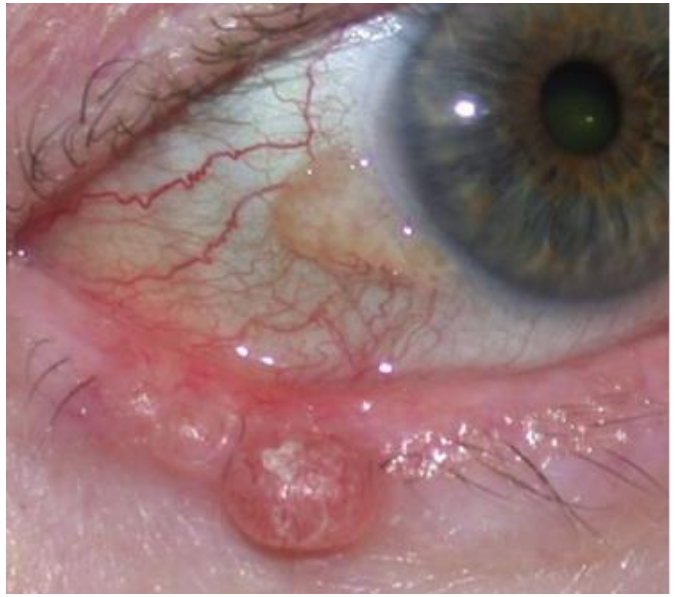
-Distortion of eyelid architecture or eyelid malposition

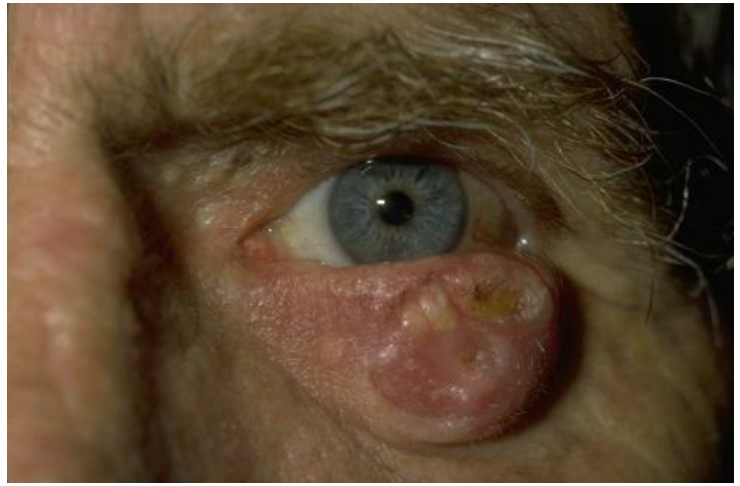
-Presence of skin ulceration

-Madarosis(loss of eyelashes)

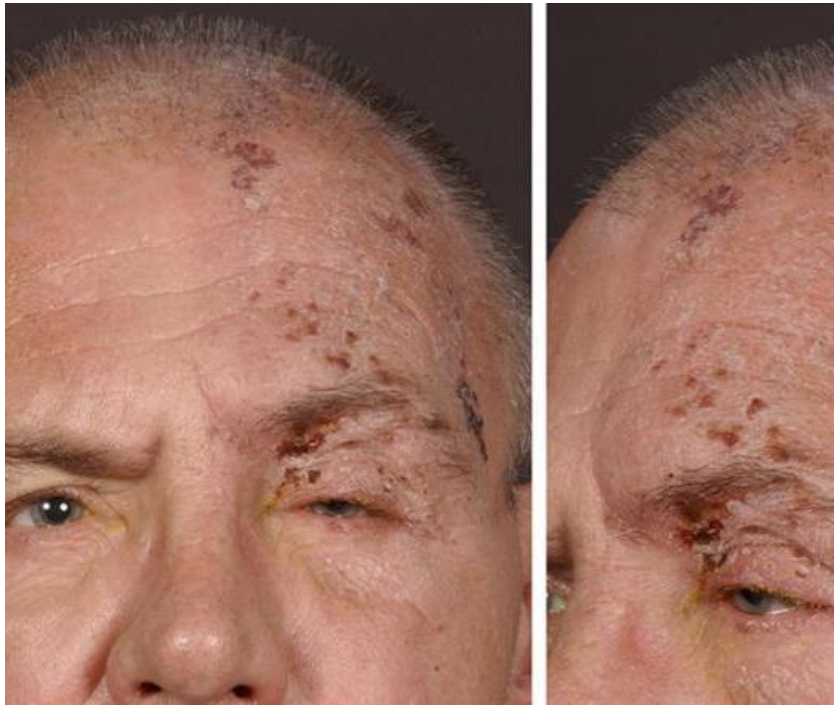
-Telangiectasias

BCC is the most common eyelid malignancy, accounting for over 90% of malignant eyelid neoplasms. One of the known risk factors of BCC is intense exposure to ultraviolet radiation. More than 50% of BCCs of the eyelid initially occur on the lower lid.





INFECTIONS INVOLVING THE EYELIDS AND ADNEXAE



•Herpes zoster ophthalmicus- distribution over V1, typically vesicular rash.

•Hutchinson's sign- strong indicator of ocular involvement.

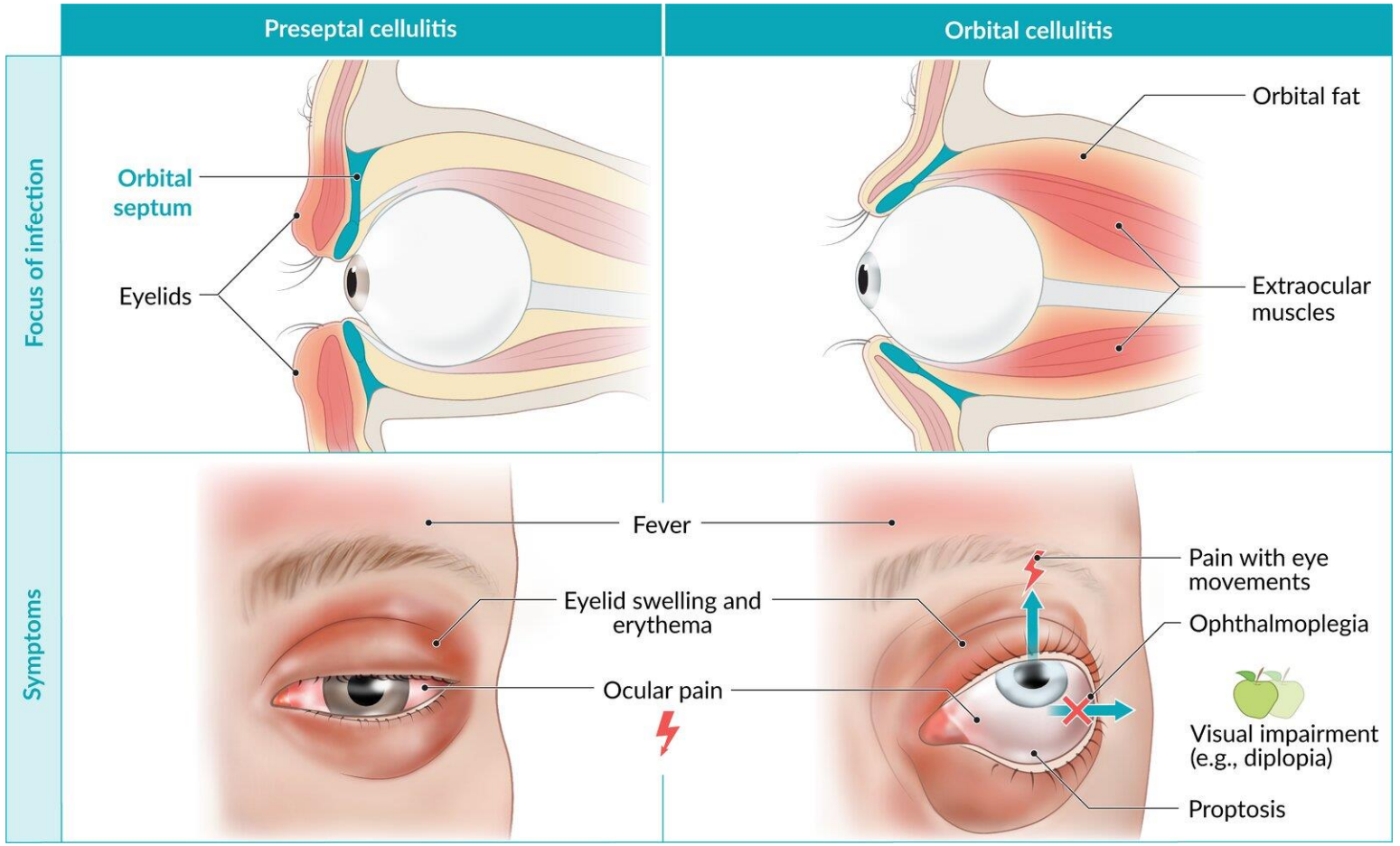
•Treat with oral Aciclovir 800mg 5x daily x 7-10 days. Refer to ophthalmologist to exclude ocular manifestations of the disease, especially if any visual symptoms/signs of epiphora and eye redness.

INFECTIONS INVOLVING THE EYELIDS AND ADNEXAE

Pre-septal cellulitis vs orbital

- Infection isolated anterior to the orbital septum is considered to be pre-septal cellulitis. Causes: insect bites, infected stye
- Orbital cellulitis is an inflammation of the soft tissues of the eye socket behind the orbital septum, a thin tissue which divides the eyelid from the eye socket.

INFECTIONS INVOLVING THE EYELIDS AND ADNEXAE



FEATURES OF ORBITAL CELLULITIS

- Ophthalmic signs most frequently seen with orbital cellulitis are limited ocular motility, proptosis, chemosis, and conjunctival hyperaemia.
- Fever and leucocytosis are also suggestive of an orbital infection.
- Vision loss and an afferent pupillary defect may occur due to severe orbital congestion and optic nerve involvement. Exposure keratopathy may also contribute to diminished vision because of disruption of corneal integrity, microbial keratitis, and stromal opacification.
- Delayed management may result in significant morbidity, including orbital apex syndrome (internal and external ophthalmoplegia, blepharoptosis, diminished corneal sensation, and vision loss) and blindness. Cavernous sinus thrombosis, cranial nerve palsies, meningitis, intracranial abscess formation, and even death can occur without prompt aggressive treatment.

CAUSES AND RISK FACTORS

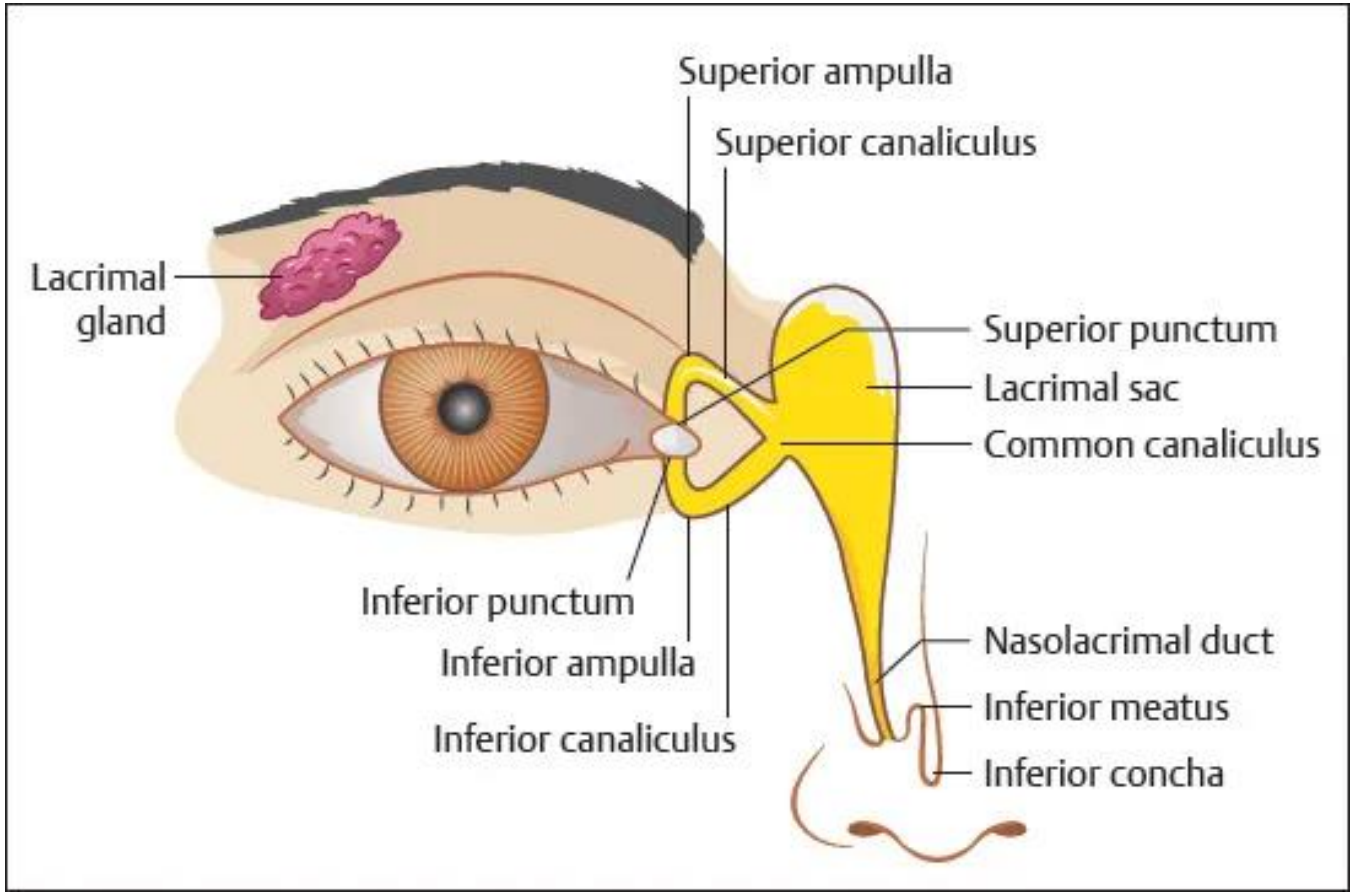
1. More than 90% of all orbital infections are the result of underlying sinus disease.
2. Although sinusitis occurs more frequently in the adult population, orbital cellulitis secondary to sinus disease is seen more commonly in young adults and children.

MANAGEMENT

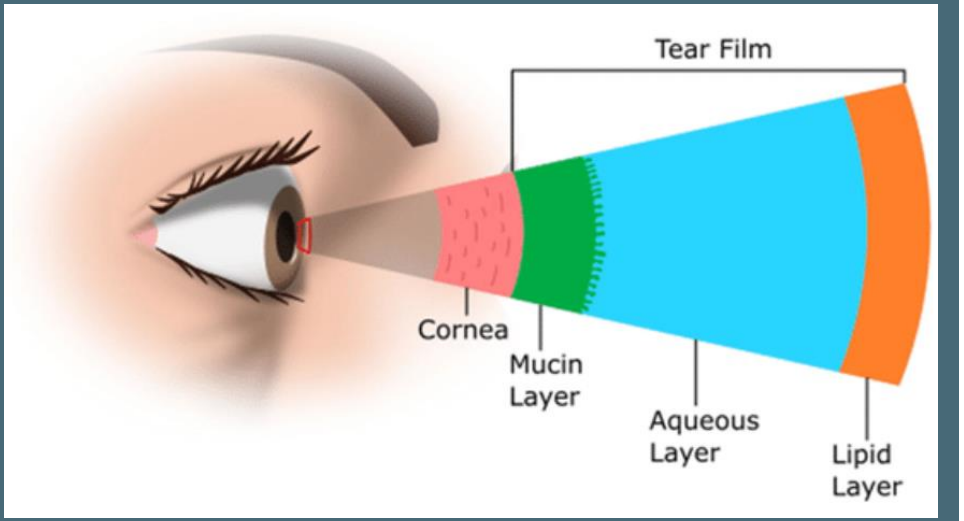
1. Pre-septal cellulitis can be treated with broad spectrum oral antibiotics.
2. Severe pre-septal cellulitis and orbital cellulitis require referral for blood investigations, imaging and IV antibiotics +/- surgical management.

DRY EYE SYNDROME AND OTHER LACRIMAL DISORDERS

ANATOMY OF THE LACRIMAL SYSTEM



CAUSES, CLINICAL PRESENTATION AND MANAGEMENT



- Tears have a similar concentration of electrolytes as in plasma, richer in proteins especially IgA, lysozyme and β-lysin
- Have a lipid layer, an aqueous layer and a mucin layer
- Parasympathetic reflex induced secretions come from the lacrimal duct
- Causes:

  -**decreased tear film production**: old age, Sjogren's, RA, lymphoma, leukaemia, sarcoidosis etc.

  -**excess evaporation of tears**: post-exposure keratitis

  -**mucin deficiency in tears**: Vitamin A def, chemical burns etc.

CAUSES, CLINICAL PRESENTATION AND MANAGEMENT

- Presentation: excess tearing, sensation of FB in eyes, conjunctival injection on examination, distinct pattern of fluorescein uptake on blue light illumination and low tear film break up time.

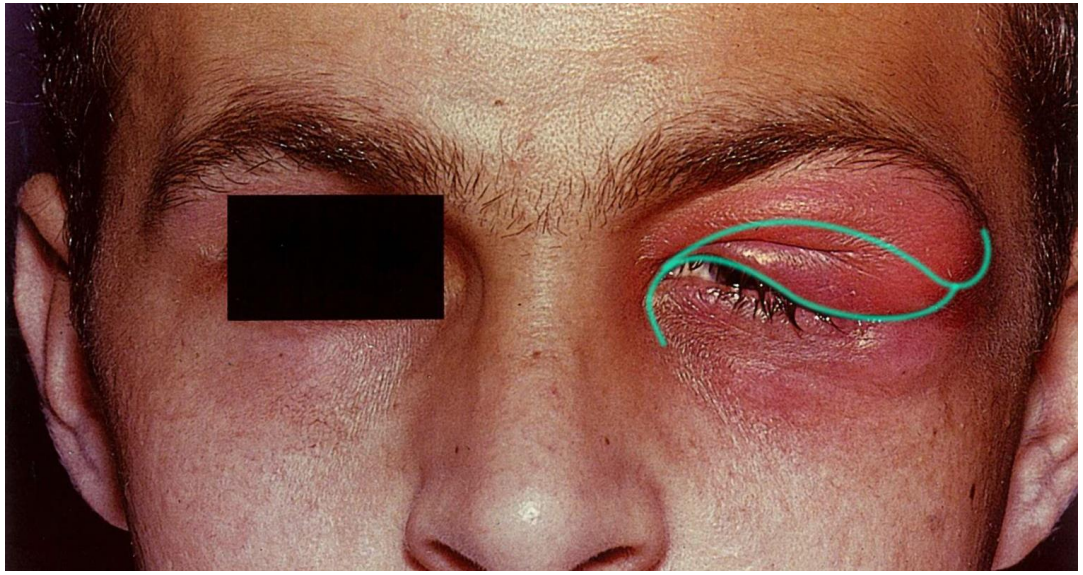
- Management: lifestyle measures, lubricant drops & ointment, topical ciclosporin in unrefractory cases. Punctal plugs are considered in severe cases of keratoconjunctivitis sicca.

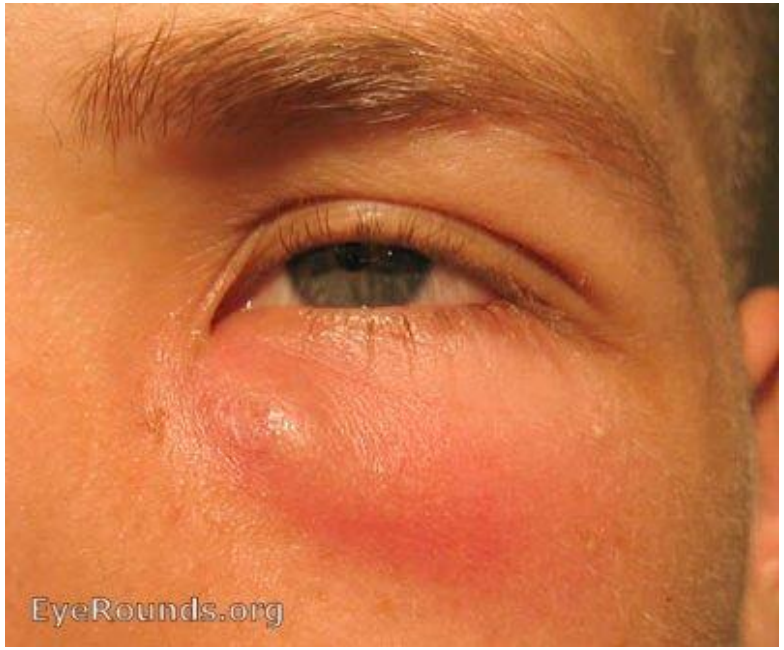
OTHER LACRIMAL SYSTEM DISORDERS

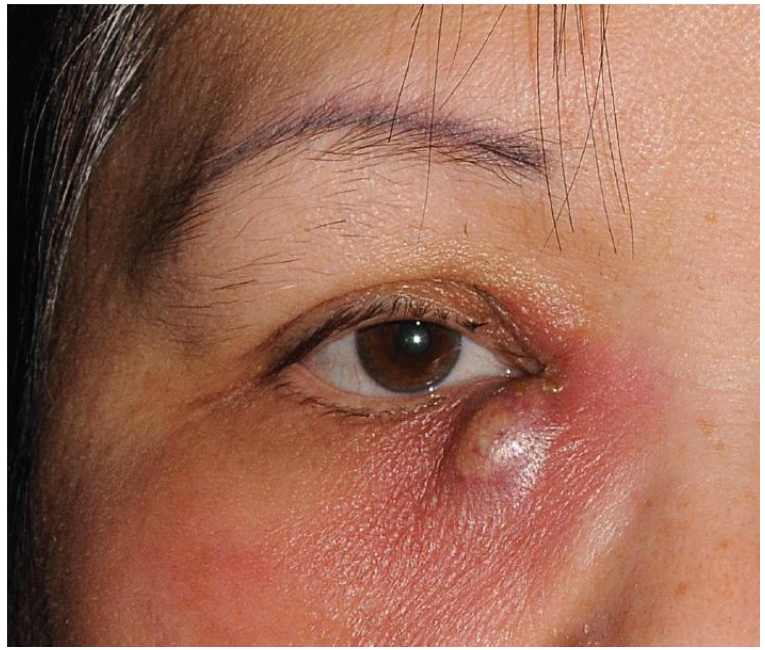
- Nasolacrimal duct non-canalization: child with persistent watery eyes, prone to conjunctivitis. May require probing of the duct under GA.
- Acute vs chronic dacrocystitis- treat with warm compresses, topical AND oral antibiotics. Ophthalmology referral required for assessment and potential surgical management.
- Dacroadenitis
- Epiphora- normal volume with insufficient drainage to the inferior meatus of the nose. Causes include ectropions and draining system blockage (idiopathic or tumours)
- Excess lacrimation can also be secondary to surface eye pathology- corneal abrasions, foreign body, entropion, conjunctivitis, and inflammatory causes.

DACROADENITIS

DACROCYSTITIS





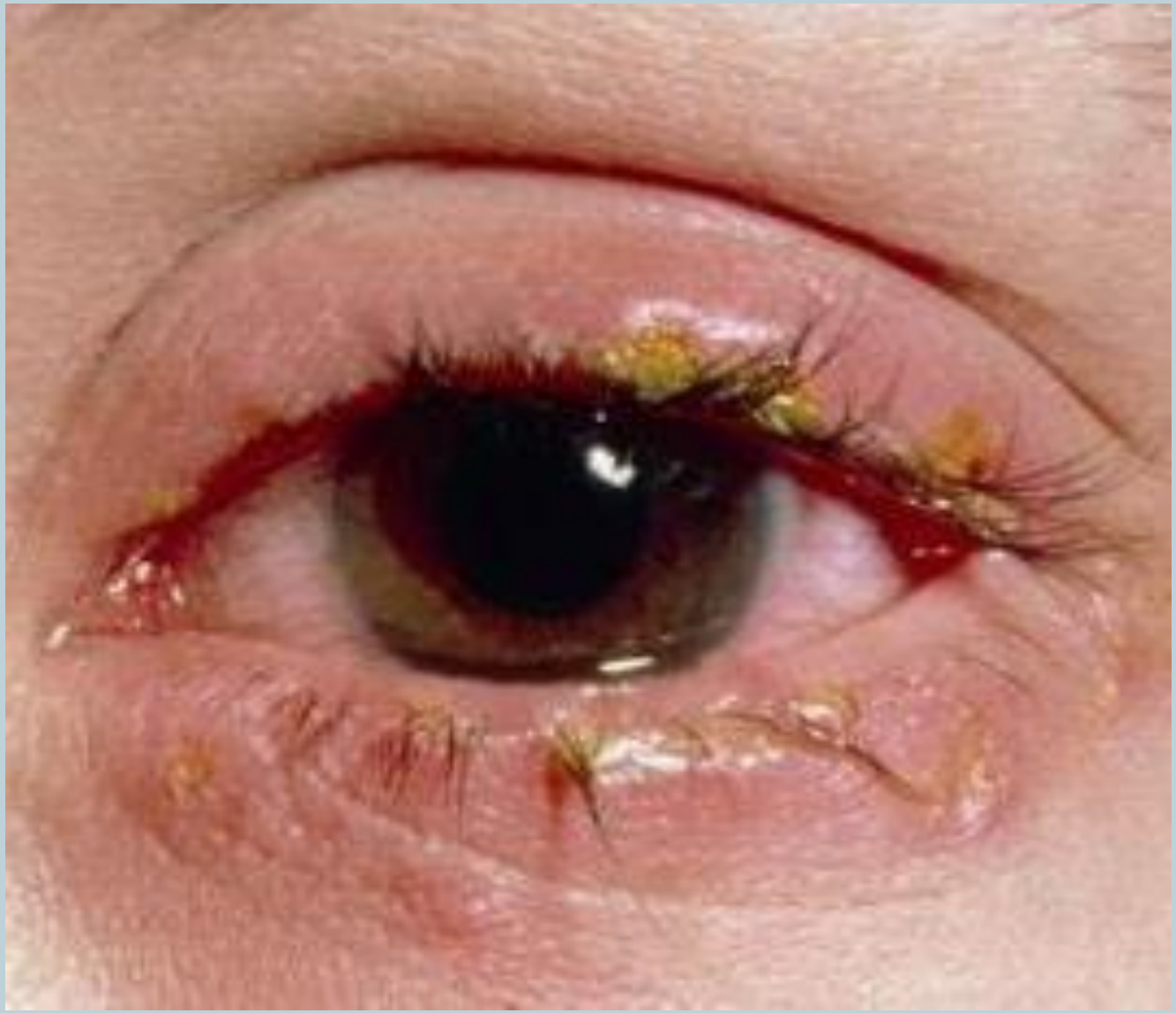


BLEPHARITIS

CAUSES, CLINICAL PRESENTATION AND MANAGEMENT

- Blepharitis is caused by chronic inflammation of the meibomian glands along the lid margin
- Present with long standing eye irritation, erythematous lid margins and scales on the eyelashes. Inflamed meibomian glands may be seen on elevation of the upper eyelid margin
- Can be associated with ingrowing lashes, dry eye syndrome, ectropions and entropions.

BLEPHARITIS

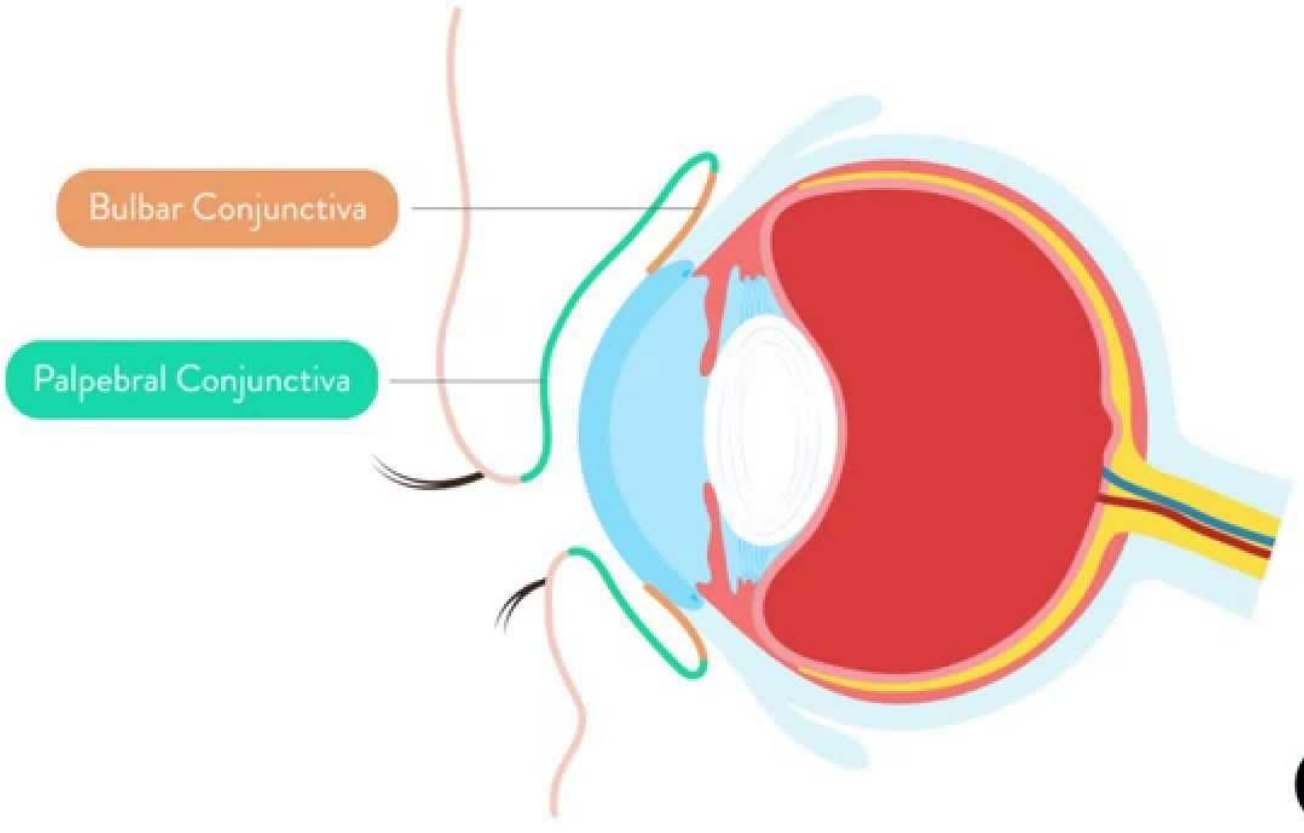


CAUSES, CLINICAL PRESENTATION AND MANAGEMENT

- Long-term management with regular eye hygiene
- May not see an immediate improvement for up to 2 weeks after initiating treatment
- Application of warm, moist flannel + eyelid massage with cotton buds, eyelid wipes, lipid-layer targeting lubricant drops and anti-septic ointments
- Treat any infective exacerbation with topical antibiotic ointments over base of eyelashes
- Topical steroids may also be used in resistant cases, however, it would be best to refer for specialist advice.

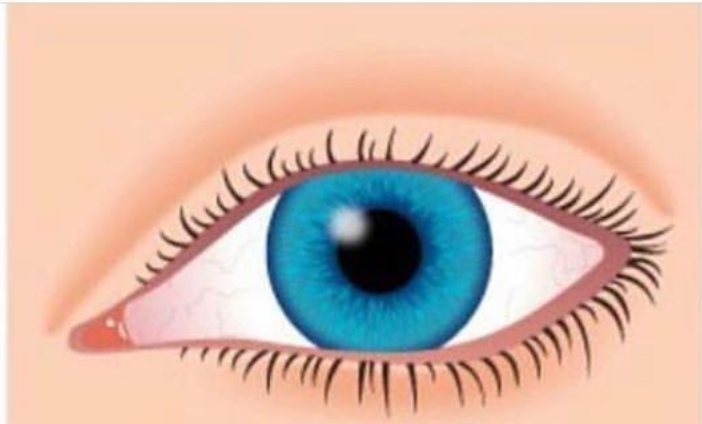

CONJUNCTIVITIS

ANATOMY OF THE CONJUNCTIVA



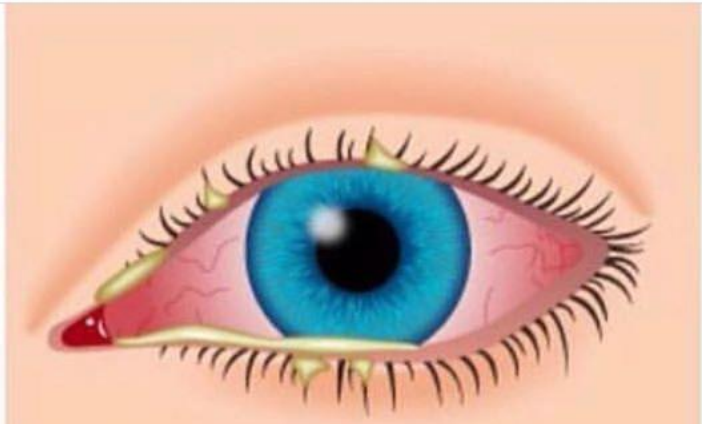
CAUSES, CLINICAL PRESENTATION AND MANAGEMENT

- Infective causes are commonly viral & bacterial
- It is not always easy to distinguish clinically; all present with an acute red eye, usually unilateral then spreading to affect both eyes. Associated with discharge, sometimes crusted and sticky eyelids.
- Viral culprit: mainly adenovirus
- Both bacterial and viral conjunctivitis may be associated with a viral URTI.
- Management: Viral infections are usually self-limiting with symptoms settling within days with good hygiene. However, consider topical antibiotics and anti-inflammatory drops.



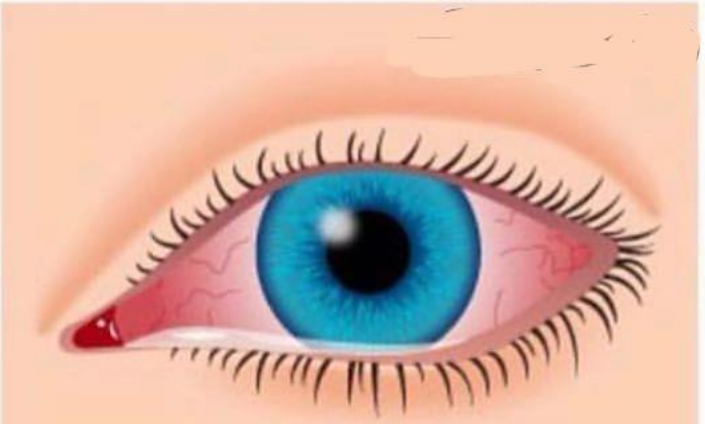
Normal eye

In a healthy eye, the sclera is essentially white with only a few small blood vessels visible. There is an adequate tear film, with no significant discharge or watering.



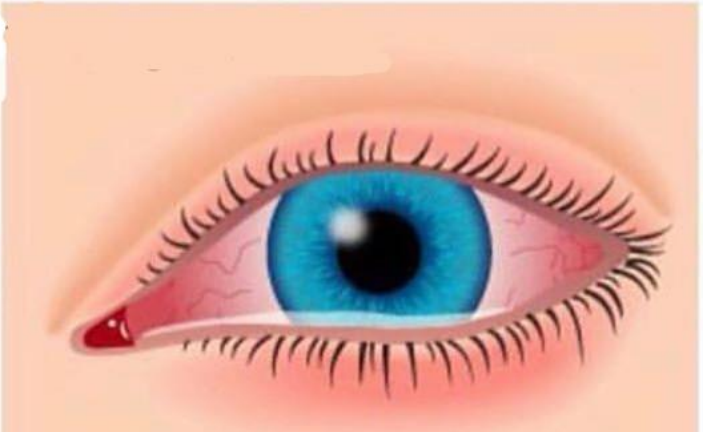
Bacterial conjunctivitis

A red eye with a sticky yellow or yellow/green discharge. Eyelids may be stuck together upon waking. Can affect one or both eyes. Usually spread by direct contact only.



Viral conjunctivitis

The type of conjunctivitis most commonly associated with the term "pink eye." Appearance: red, itchy, watery eye. Can affect one or both eyes. Highly contagious.



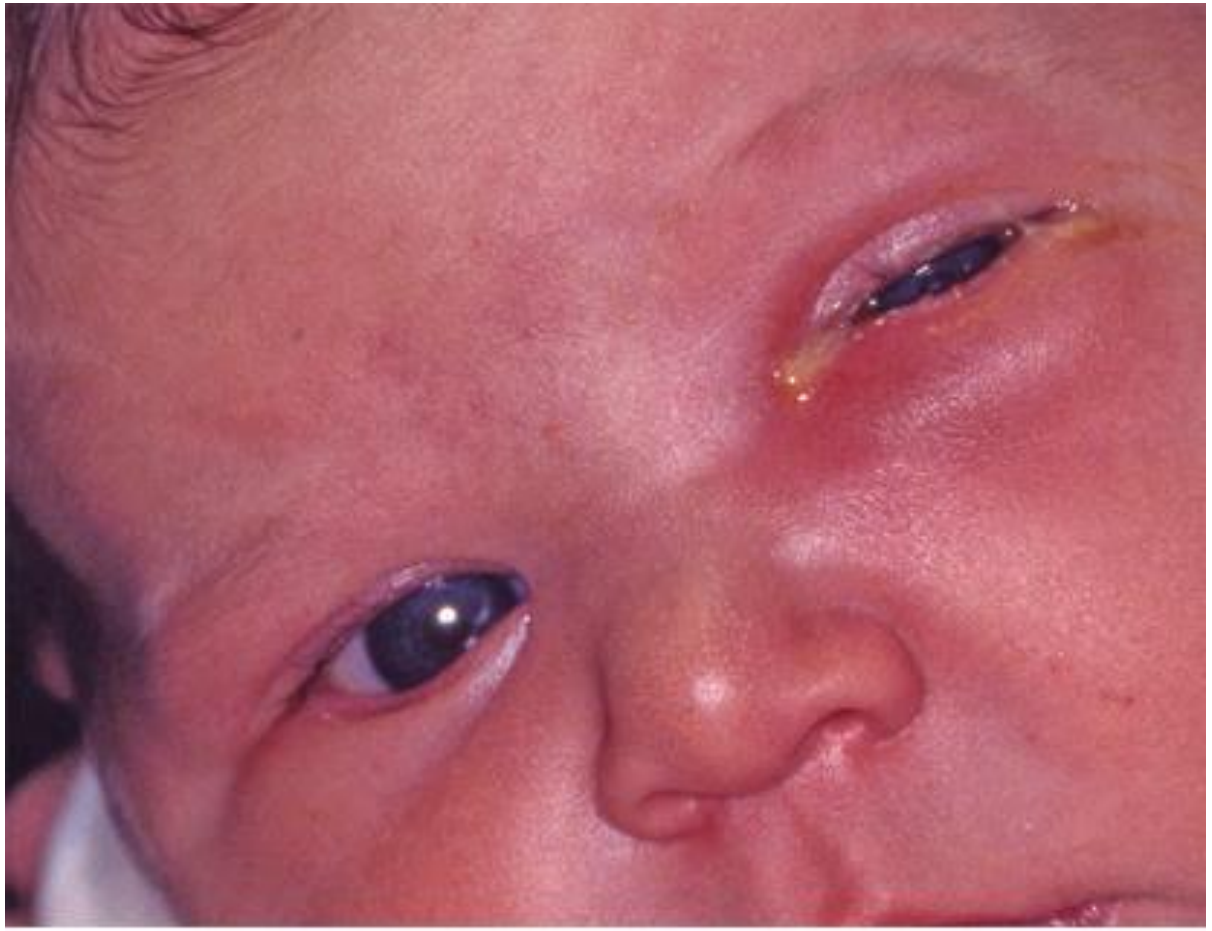
Allergic conjunctivitis

Very similar in appearance to viral conjunctivitis, but accompanied by nasal congestion, sneezing, eyelid swelling and sensitivity to light. Both eyes are affected. Not contagious.

NEONATAL CONJUNCTIVITIS

- Occurs in first 30 days of life
- Require swabs for MC&S and PCR to exclude Chlamydia, Gonorrhoea and herpetic infections and urgent specialist review.
- Enquire for history of maternal STDs
- Not all neonatal conjunctivitis are caused by the above pathogens.
- Broad spectrum topical antibiotics until swab results. Will require liaison with pediatricians and ophthalmologists in the case of positive result for Chlamydia/Gonorrhea/HSV.

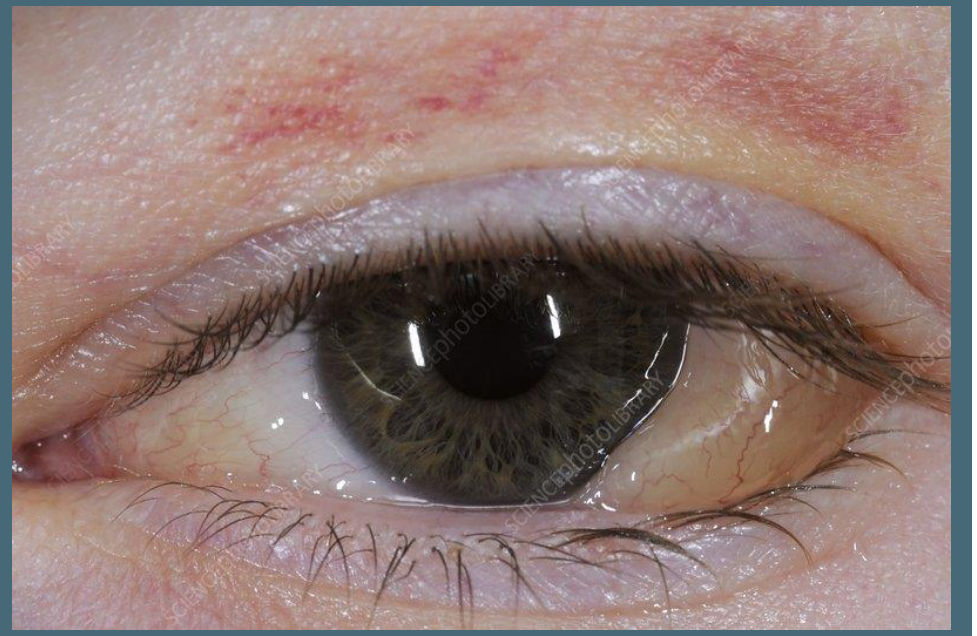
CHLAMYDIA CONJUNCTIVITIS



Source: K.J. Knoop, L.B. Stack, A.B. Storrow, R.J. Thurman:
The Atlas of Emergency Medicine, 4th Edition,
www.accessemergencymedicine

ALLERGIC CONJUNCTIVITIS

CAUSES, CLINICAL PRESENTATION AND MANAGEMENT

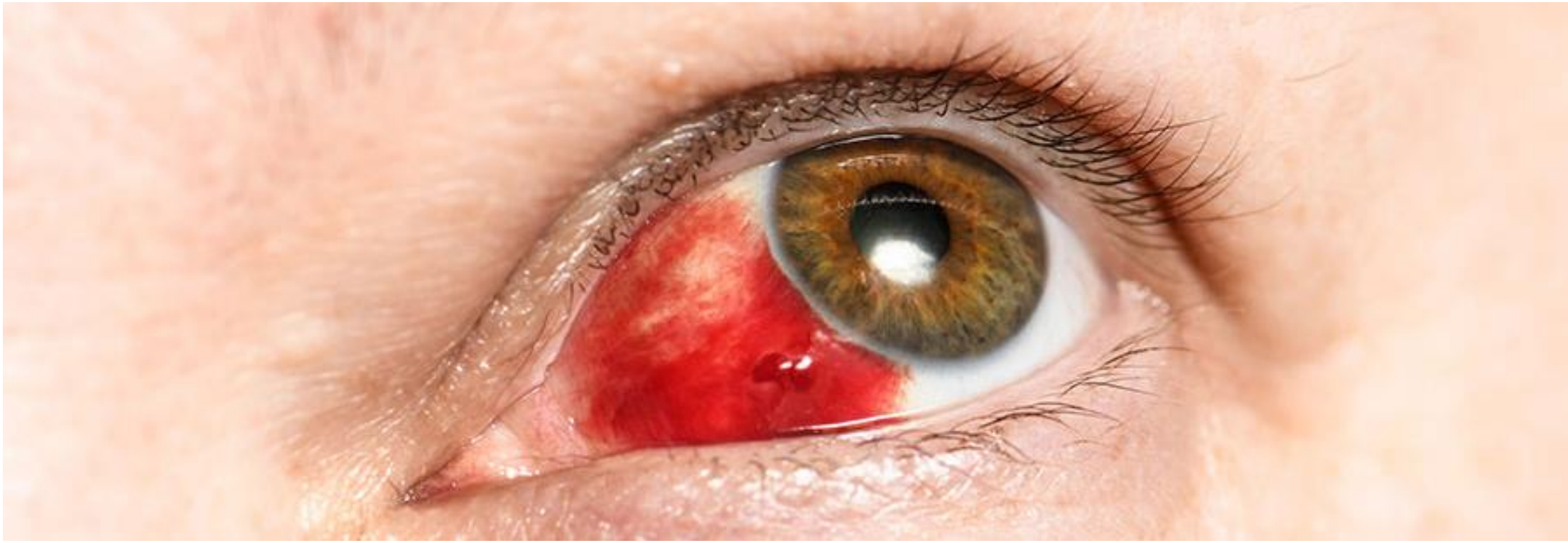


- Seasonal vs atopic
- Often present with bilateral symptoms, red and watery eyes a/w itching and possible photophobia
- Identify cobblestone appearance over upper sub-tarsal area, follicles over lower sub-tarsal area. Chemosis and conjunctival injection may be present.
- Management includes topical lubricants, anti-histamines, mast cell stabilizers, occasionally requiring topical steroid

SUBCONJUNCTIVAL HAEMORRHAGE

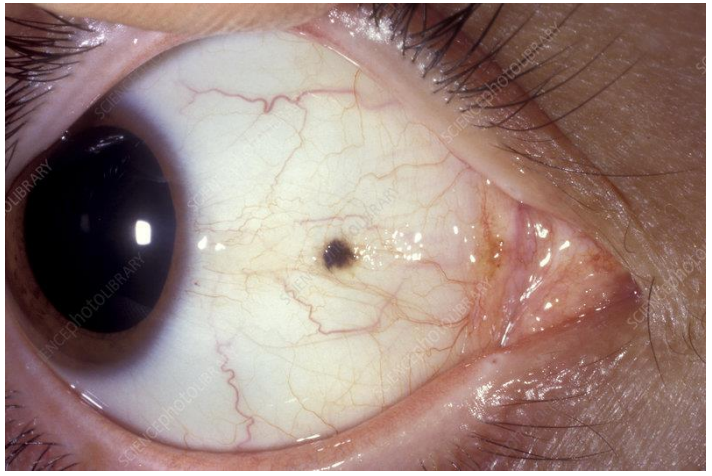
- Appears as a bright red stain over the conjunctiva, usually alarming to patients despite its benign nature.
- What to assess: history of anti-coagulant use, trauma, recent Valsalva manoeuvres, high BP.
- Management: reassurance, will fade in colour slowly over days. Prescribe lubricant drops due to tendency of tear film disruption.
- No need for ophthalmic assessment unless secondary to blunt force trauma and a/w visual symptoms.

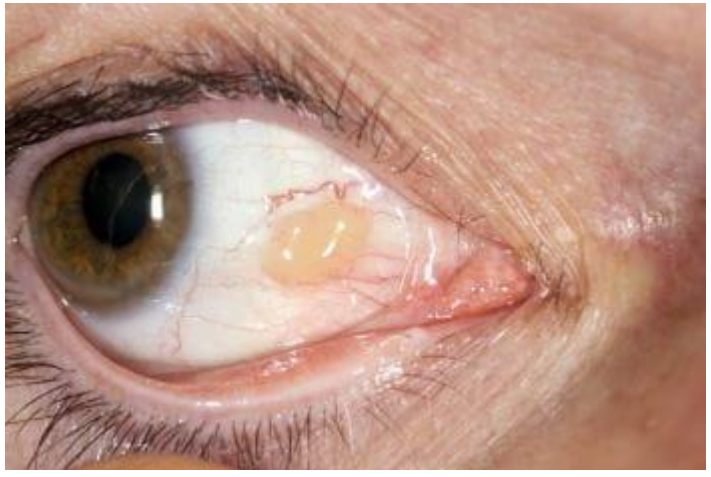
SUBCONJUNCTIVAL HAEMORRHAGE

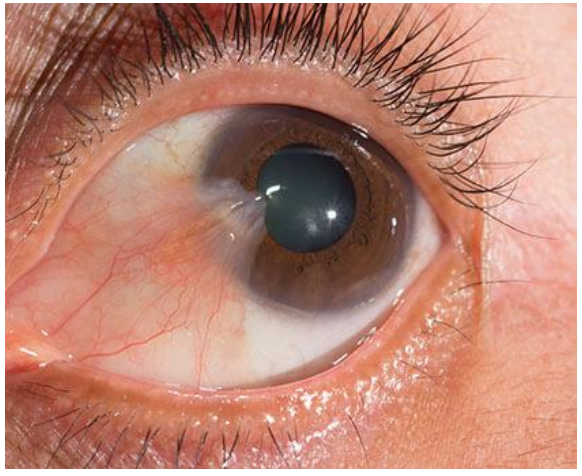


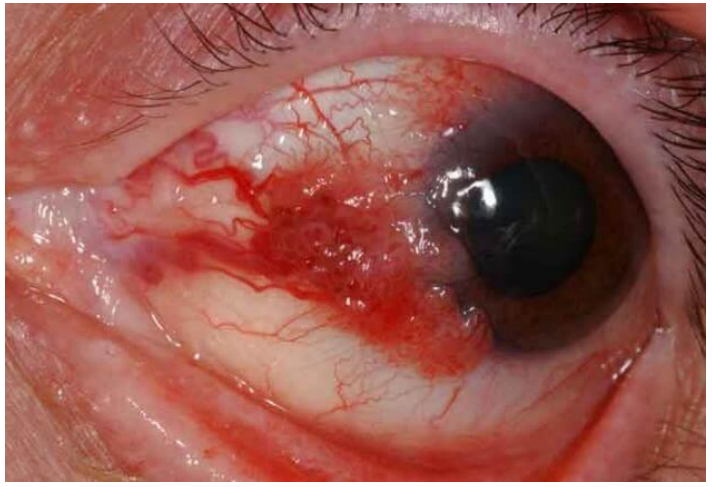
OTHER CONJUNCTIVAL DISORDERS

- PINGUECULUM
- PTERYGIUM
- CONJUNCTIVAL NAEVI AND CYSTS
- CONJUNCTIVAL TUMOURS E.G. SCC



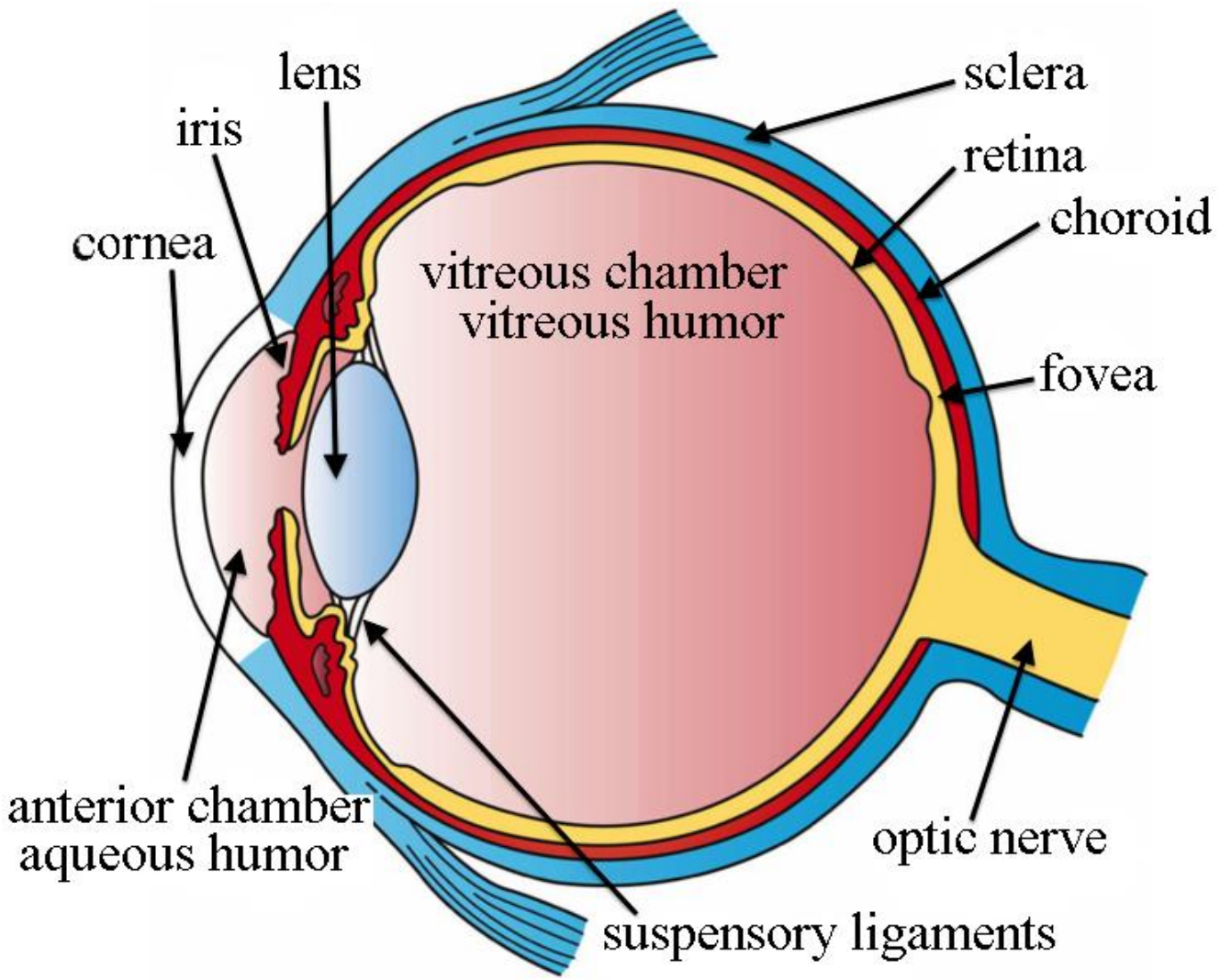






SCLERAL PATHOLOGY

ANATOMY OF THE SCLERA



EPISCLERITIS VS SCLERITIS

Episcleritis

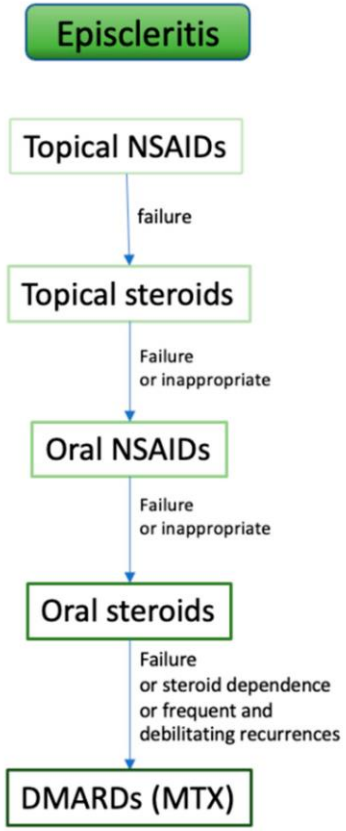
a. usually unilateral
b. Diffuse vs segmental
c. Symptoms include localised/sectoral eye redness and ocular discomfort
d. Treat with topical NSAIDs
e. a/w RA, SLE and UC, however 70% are idiopathic.

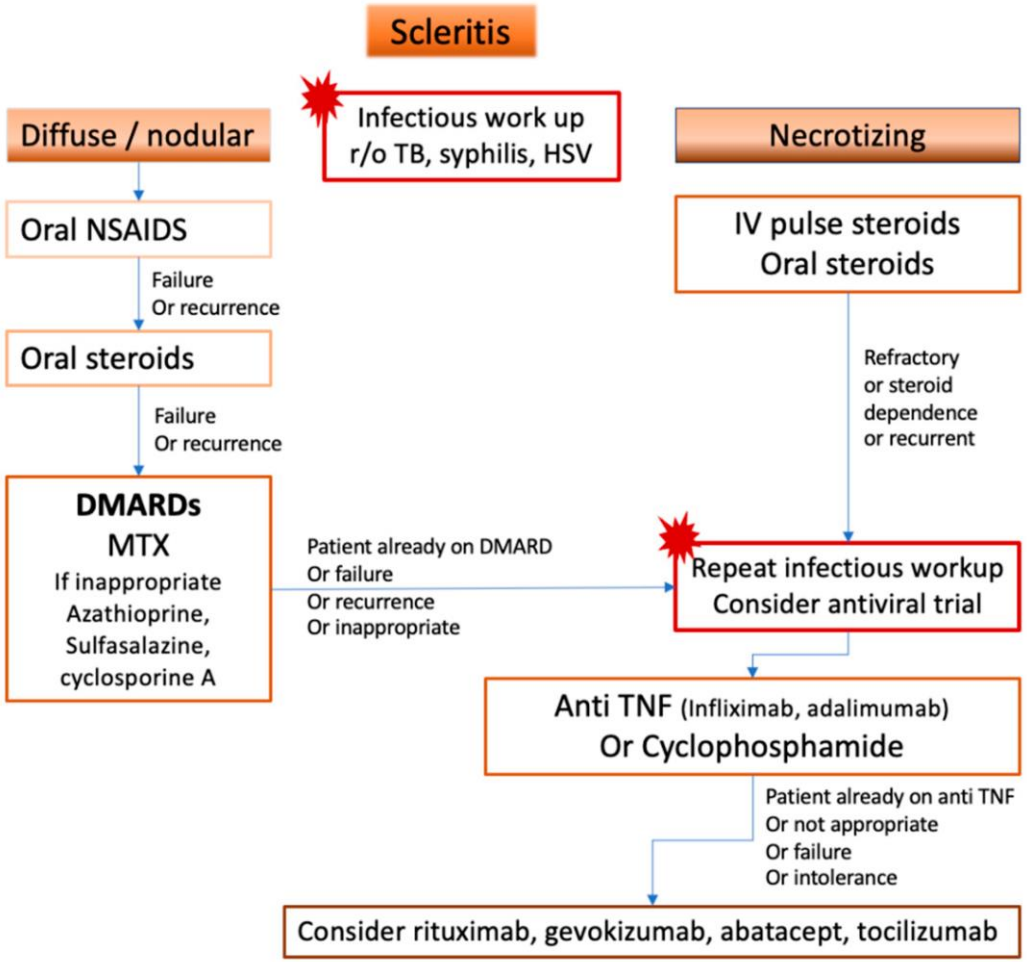
EPISCLERITIS VS SCLERITIS

Scleritis

- Painful red eye +/- visual loss
- Diffuse vs nodular vs necrotizing (rare), anterior vs posterior.
- Treat with oral NSAIDs +/- topical steroids- urgent referral to ophthalmology.
- More associated with AI disease/systemic conditions in 50% of cases. Commoner in women. Needs a full work up.

TREATMENT


Episcleritis
Topical NSAIDs
failure
Topical steroids
Failure
or inappropriate
Oral NSAIDs
Failure
or inappropriate
Oral steroids
Failure
or steroid dependence
or frequent and
debilitating recurrences
DMARDs (MTX)

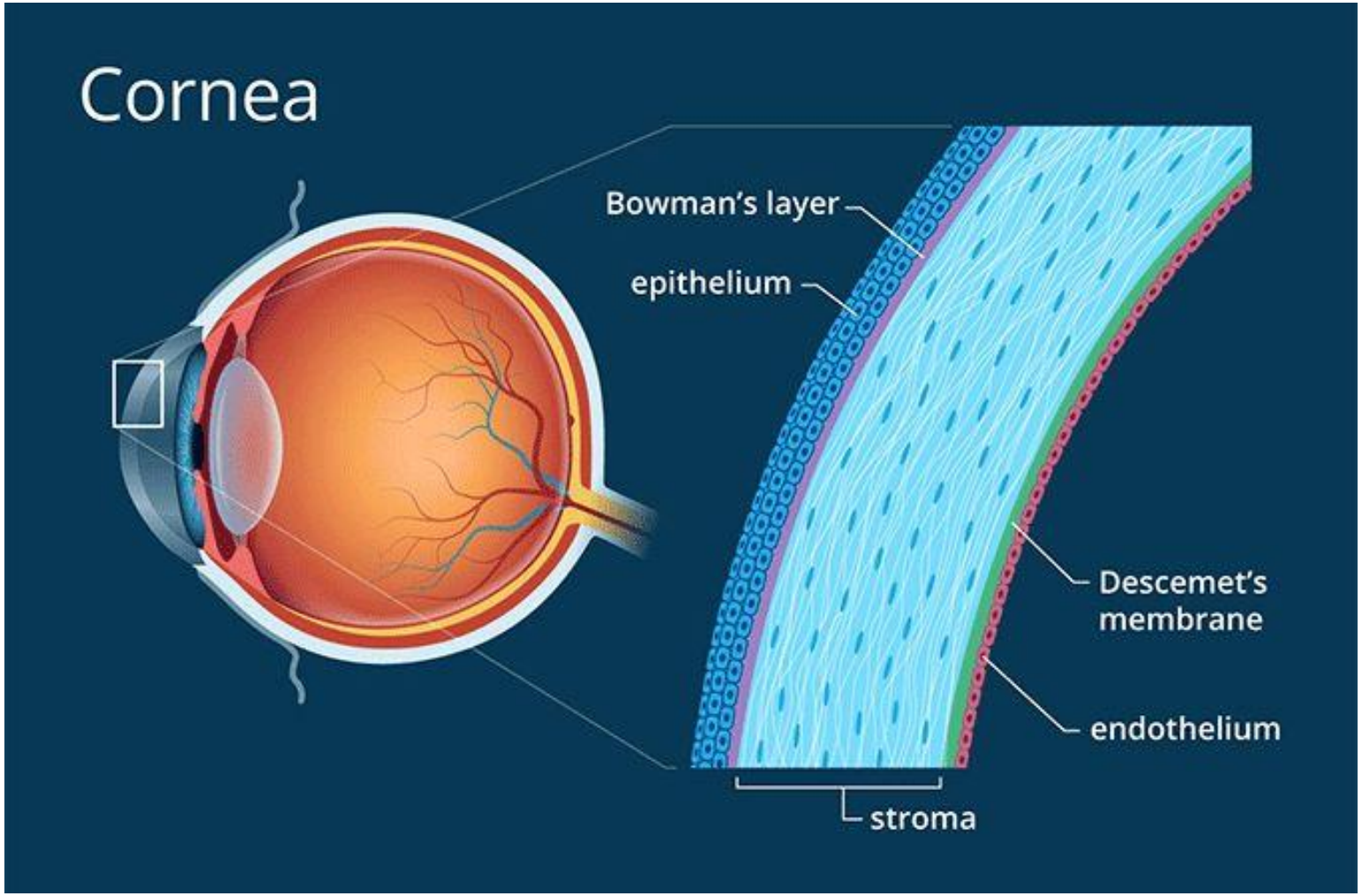

Scleritis
Diffuse / nodular
Infectious work up
r/o TB, syphilis, HSV
Necrotizing
Oral NSAIDS
Failure
Or recurrence
Oral steroids
Failure
Or recurrence
DMARDs
MTX
If inappropriate
Azathioprine,
Sulfasalazine,
cyclosporine A
Patient already on DMARD
Or failure
Or recurrence
Or inappropriate
IV pulse steroids
Oral steroids
Refractory
or steroid
dependence
or recurrent
Repeat infectious workup
Consider antiviral trial
Anti TNF (Infliximab, adalimumab)
Or Cyclophosphamide
Patient already on anti TNF
Or not appropriate
Or failure
Or intolerance
Consider rituximab, gevokizumab, abatacept, tocilizumab

CLINICALLY DISTINGUISHING BOTH

- In order to help distinguish clinically between both conditions, phenylephrine eye drops may be used.
- Phenylephrine blanches the conjunctival and episcleral vessels but has no effect on scleral vessels.
- Therefore, if eye redness improves after phenylephrine installation, a diagnosis of episcleritis can be made.

CORNEAL PATHOLOGY

ANATOMY OF THE CORNEA



CORNEAL ECTASIAS

- **Keratoconus**
  - Mainly affecting young adults
  - May be precipitated by chronic eye rubbing
  - Symptoms of keratoconus include:
    - Monocular diplopia, blurry vision with haloes, photosensitivity.
  - A/w myopia and characterised by high degree astigmatism and corneal thinning.
  - Treatment: hard/hybrid contact lenses, corneal cross-linking for progressive disease and corneal grafting in severe cases.
  - Other ectasias include pellucid marginal degeneration and keratoglobus.

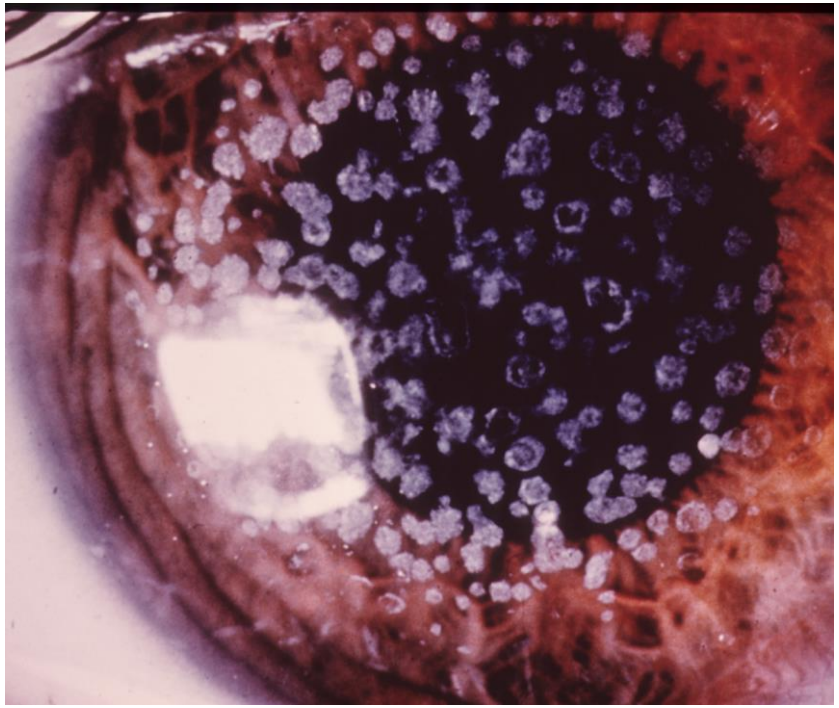
KERATOCONUS

CORNEAL DYSTROPHIES

TABLE 1: THE CLASSIFICATION OF CORNEAL DYSTROPHIES BASED ON THE ANATOMICAL LAYER CHIEFLY AFFECTED.	
Epithelial / Subepithelial	Epithelial basement membrane dystrophy Epithelial recurrent erosion dystrophy Meesman dystrophy Lisch epithelial dystrophy Gelatinous drop like dystrophy
Bowman's layer	Reis Buckler dystrophy Thiel Behnke dystrophy
Stromal	Lattice dystrophy Granular dystrophy Macular dystrophy Schnyder crystalline dystrophy Central cloudy dystrophy of Francois
Descemet's membrane / Endothelial	Fuchs endothelial dystrophy Posterior polymorphous dystrophy Congenital hereditary endothelial dystrophy X linked endothelial dystrophy

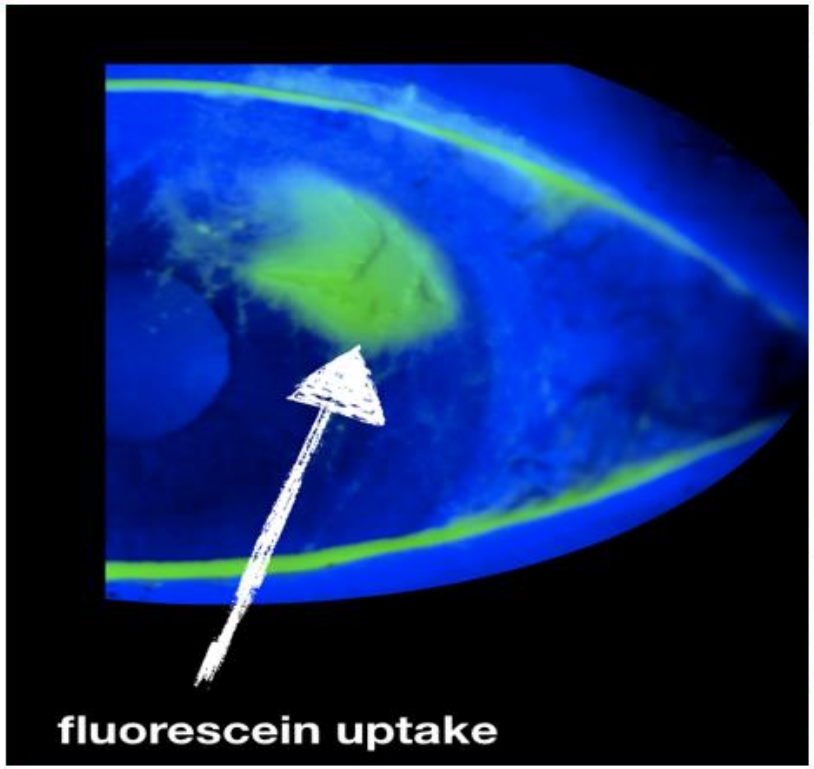
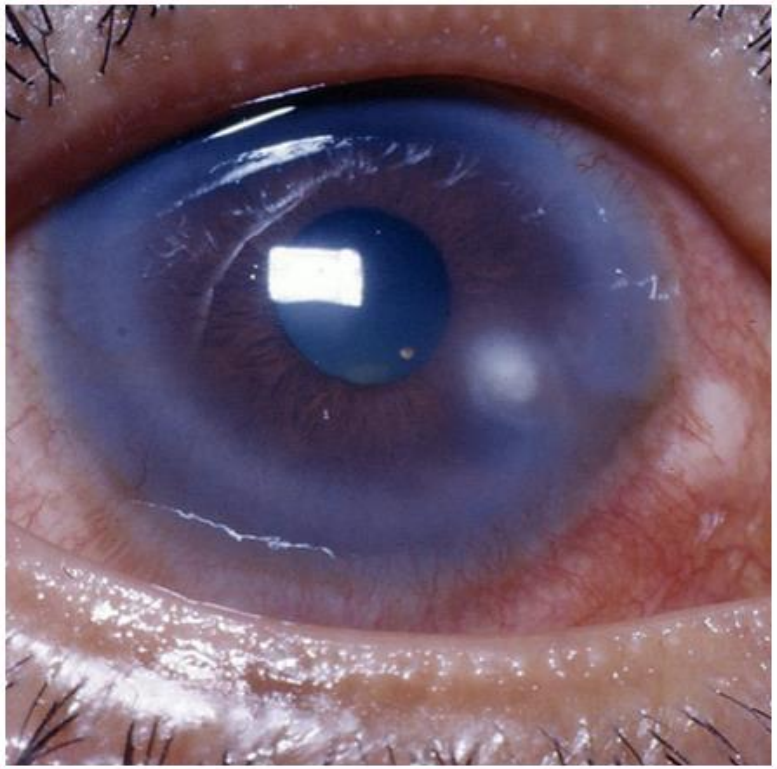
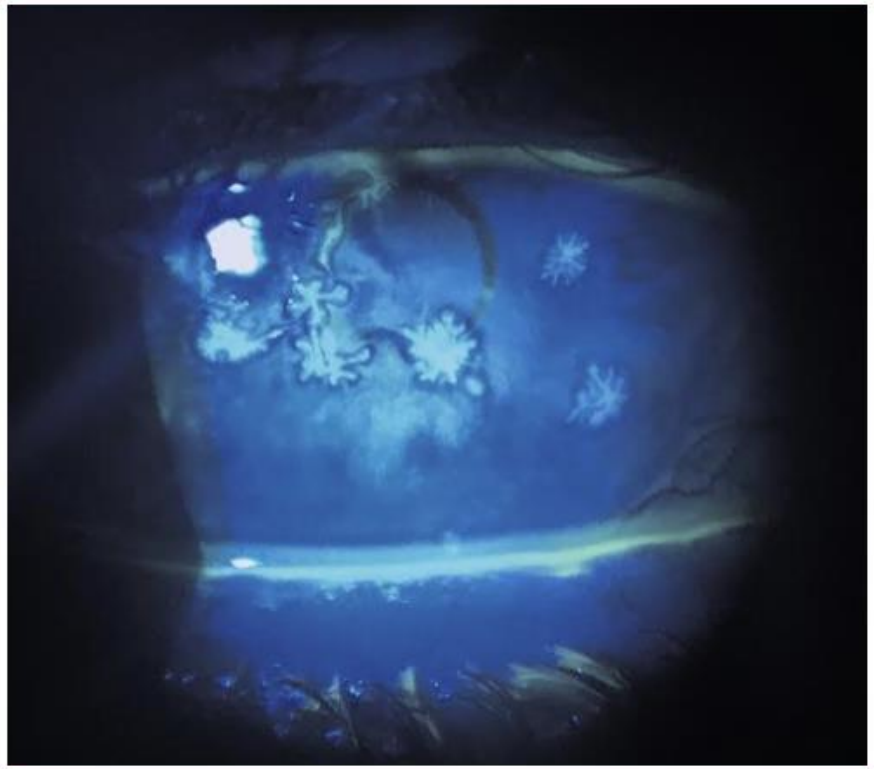
Recurrent corneal erosion syndrome can follow a history of corneal trauma or can be secondary to an underlying epithelial/anterior stromal corneal dystrophy.

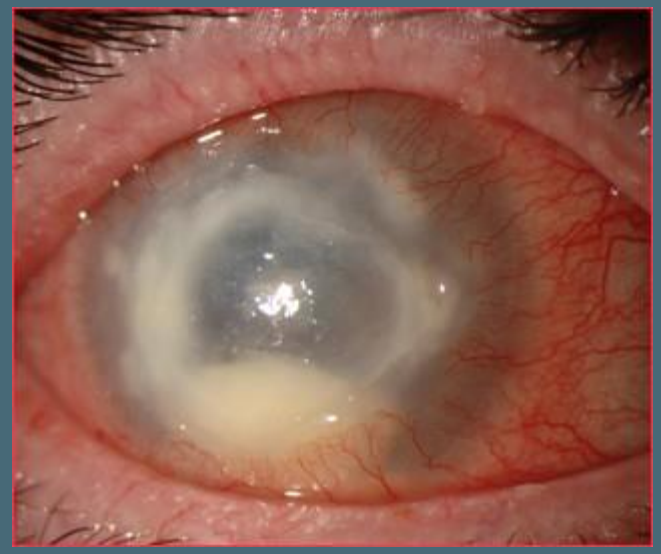
CORNEAL DYSTROPHIES



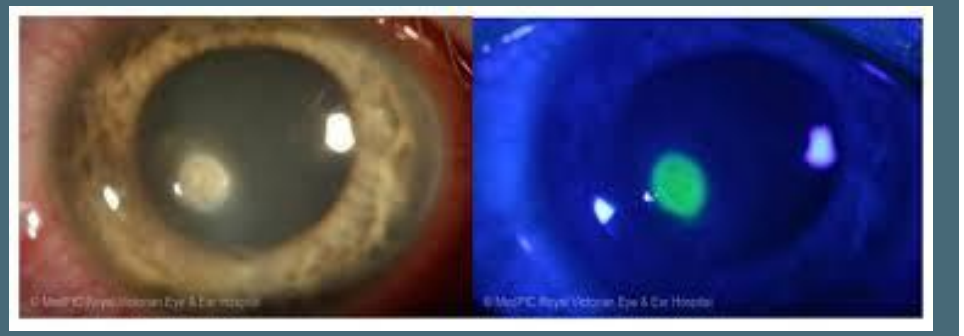
DISTINGUISHING BETWEEN CORNEAL ULCERS FROM CORNEAL EROSIONS

- Corneal abrasion/erosion vs keratitis/corneal ulcers:
    - Demonstrated on fluorescein stain on blue light illumination.
    - Present with an acute red eye – diffuse conjunctival injection, photophobia, foreign body sensation, epiphora and blurred vision.
    - Erosions/abrasions are superficial epithelial defects, without surrounding inflammation and usually heal within 24 hours.
    - Ulcerations are epithelial defects with surrounding inflammation- infective vs sterile. Inflammation is visible as greyish clouding of the cornea.
    - Infiltrations: white/yellowish discolouration due to leukocytes.
    - Severe corneal ulcers may result in a hypopyon.

CORNEAL ABRASION	CORNEAL ULCER	HERPES KERATOCONJUNCTIVITIS
		

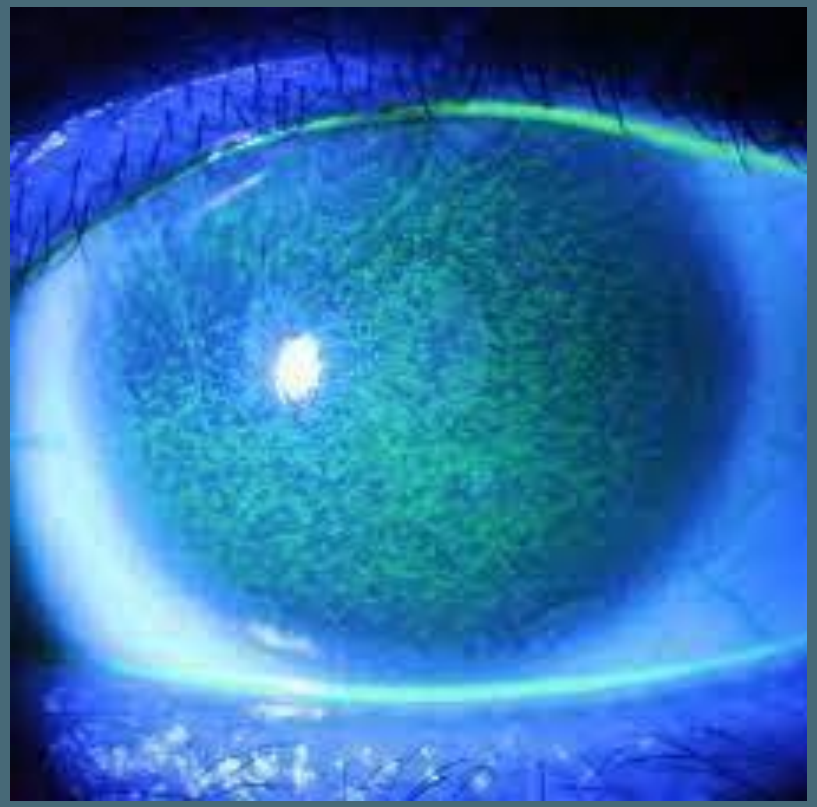


INFECTIVE KERATITIS



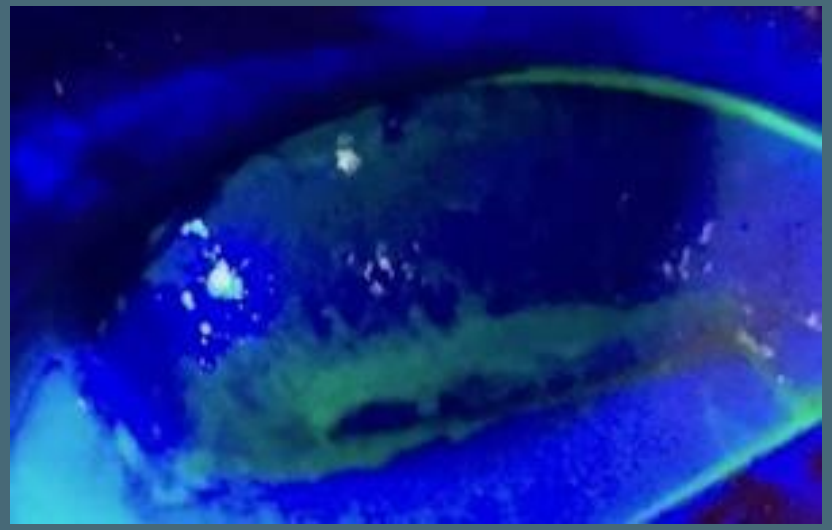
- Bacterial keratitis AKA 'corneal ulcer' is a serious bacterial infection of the cornea which can, in severe cases, cause loss of vision.
- Both Gram positive and Gram negative organisms are implicated as causative agents. About 80 % of bacterial keratitis is caused by Staphylococcus, Streptococcus and Pseudomonas species.
- Infectious ulcers may also be due to fungi, viruses and protozoa (Acanthamoeba).
- These cases require referral to A&E for Ophthalmic assessment and corneal scrapings. Treatment involves frequent administration of topical antibiotics, cycloplegia and admission for close monitoring and IV Abx in severe cases.
- Contact lens wearers: high risk for more severe infection with Acanthamoeba resulting severe sight threatening disease. Send contact lens and case for MC&S. Importance of education re good contact lens use and hygiene.
- Herpes simplex keratoconjunctivitis: Dendritic ulcer on fluorescein staining. Treat with topical acyclovir. AVOID TOPICAL STEROIDS. Refer for specialist review.

UV KERATOPATHY



- Ultraviolet keratopathy is caused by the toxic effects of acute high-dose ultraviolet radiation.
- Symptoms include ocular pain, tearing, conjunctival chemosis, blepharospasm, and blurred vision typically several hours after exposure.
- Treatment: topical lubricants +/- topical antibiotics/ointments. No need for topical cycloplegics/mydratics. AVOID dispensing topical anaesthetics.
- No need to patch the eye. Wearing sunglasses with UV protection is recommended.

EXPOSURE KERATOPATHY



Superficial punctate keratopathy of the middle to inferior cornea which can result in more extensive and deeper epithelial erosions if left untreated.

Can be secondary to severe ectropion, VII CN palsy, diminished corneal sensation due to CN V neuropathy or herpetic infections, eyelid retraction in TED.

Manage with regular lubrication, ointments, eye taping. May require surgical correction.

CORNEAL DEGENERATIVE DISEASE

Bullous keratopathy

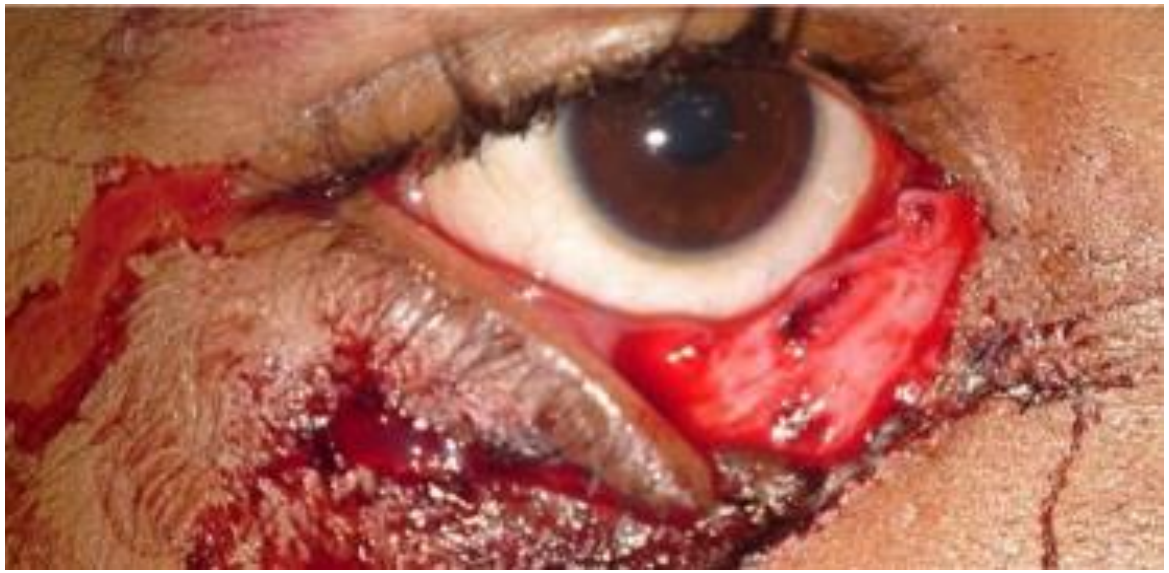
- Due to accumulation of fluid within corneal storma due to epithelial dysfunction.
- Results in haziness of the cornea, corneal bullae and corneal erosion.
- Causes include Fuch’s corneal dystrophy, elevated IOPs, herpetic keratitis, cataract surgery and collagen vascular diseases.

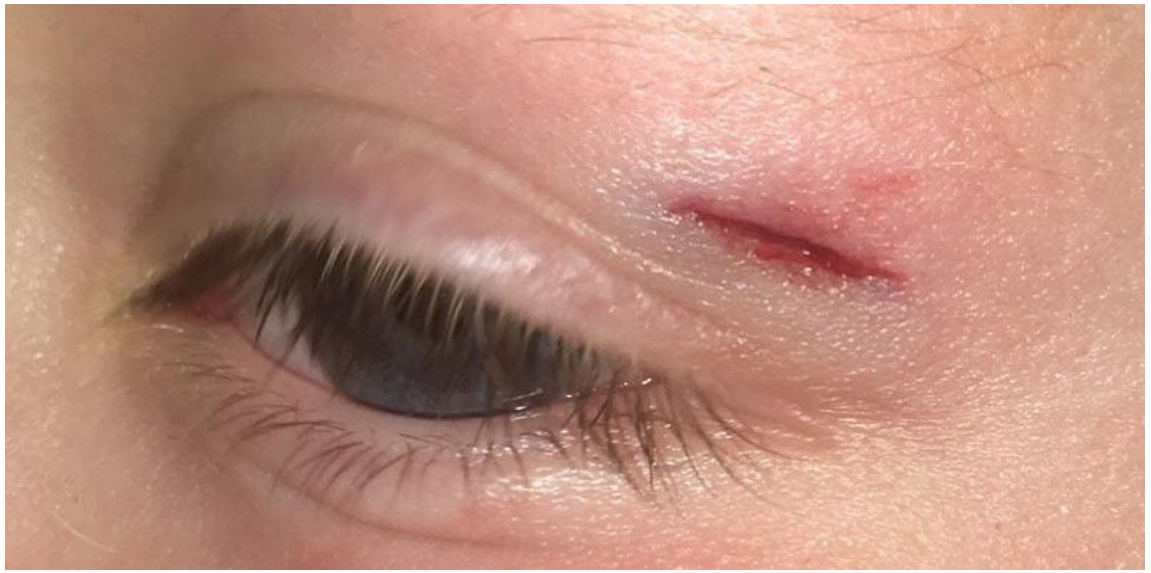
Band keratopathy

Corneal calcification secondary to chronic inflammatory ocular disease, chemical trauma and systemic hypercalcemia.


TRAUMA

EYELID LACERATIONS





CONJUNCTIVAL AND CORNEAL TRAUMA

Foreign bodies: assess sub-tarsal area for hidden FB. Remove non-embedded FB with swab on stick.

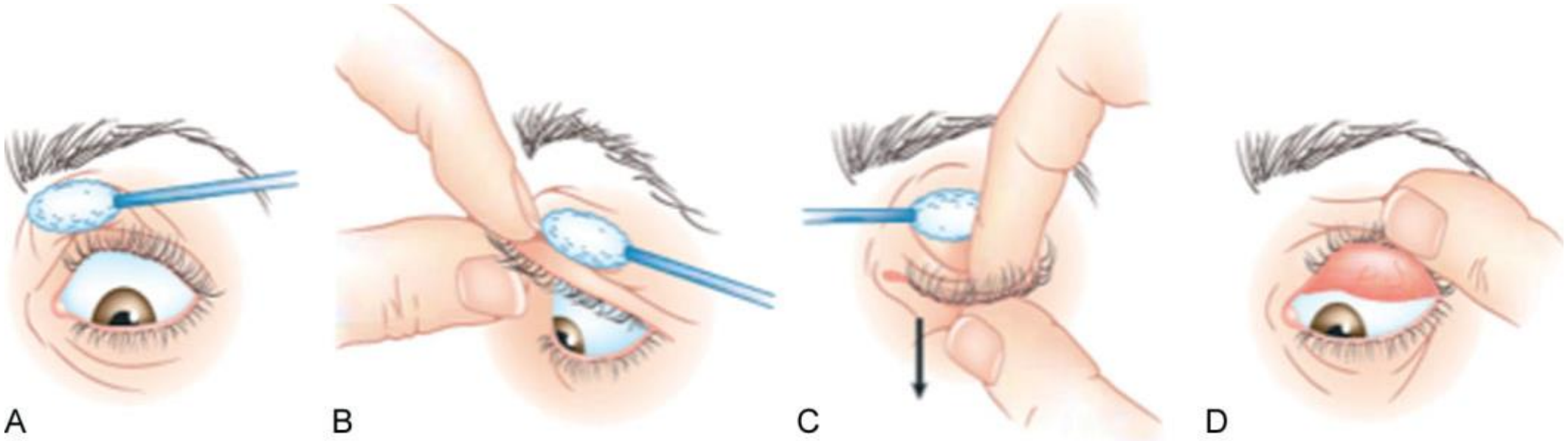
Conjunctival scratches and lacerations: may require referral for suturing and assessment for possible penetrating injury if deep.

Cover with topical antibiotic ointment +/- lubricants prior to discharging patients.

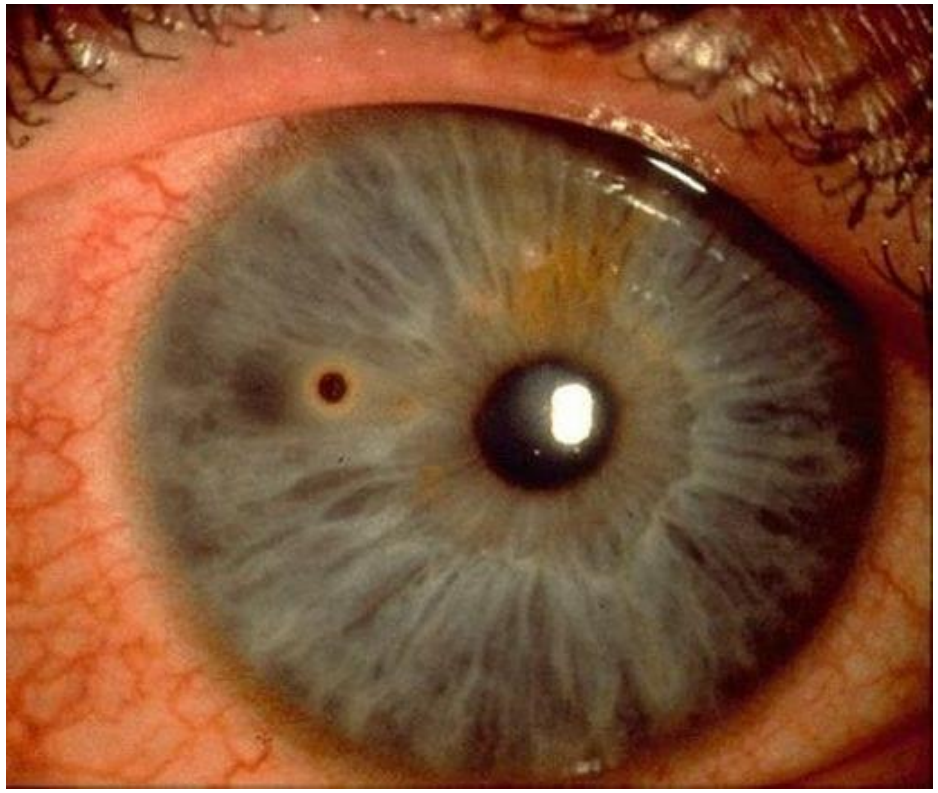
Refer to ophthalmology if no facilities/experience for removing embedded corneal foreign bodies (e.g. metal deposits)

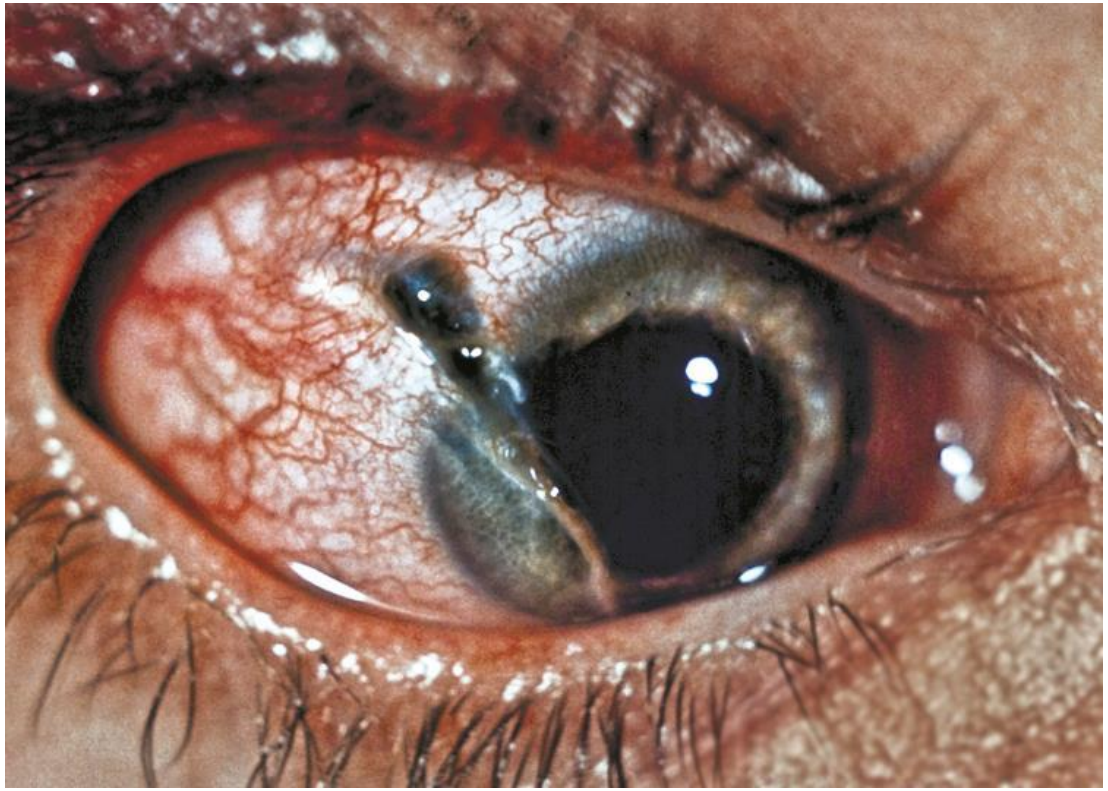
Abrasions secondary to organic causes e.g. leaves/fingernail scratches etc require cover with topical fluoroquinolone.

EYELID EVERSION TECHNIQUE

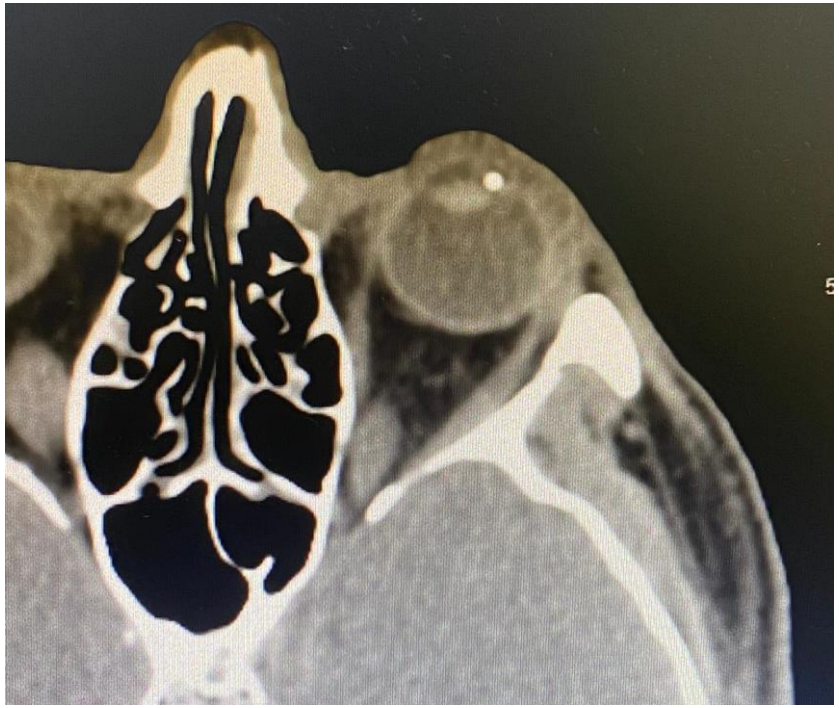


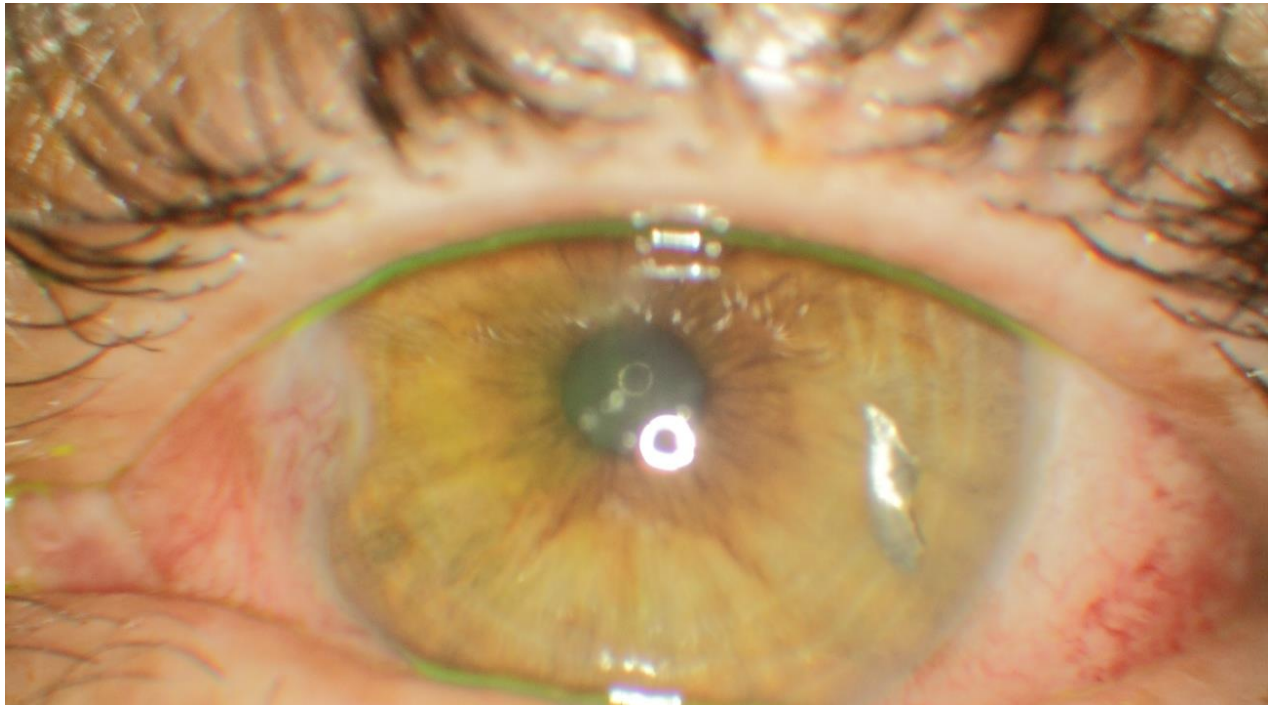
SUPERFICIAL CORNEAL FB VS PENETRATING INJURY





FB PENETRATING INJURY





CHEMICAL INJURIES

- Chemical (alkali and acid) injury of the conjunctiva and cornea is a true ocular emergency and requires immediate intervention.

- Chemical injuries to the eye can produce extensive damage to the ocular surface and anterior segment leading to visual impairment and disfigurement.

- Alkali agents are lipophilic and therefore penetrate tissues more rapidly than acids.

CHEMICAL INJURIES

HISTORY

- The severity of ocular injury depends on four factors: the toxicity of the chemical, how long the chemical is in contact with the eye, the depth of penetration, and the area of involvement.

- The patient should be asked when the injury occurred, whether they rinsed their eyes afterwards and for how long, the mechanism of injury (was the chemical under high pressure?), the type of chemical that splashed in the eye, and whether they were wearing eye protection. If available, it is helpful to obtain the packaging of the chemical.

CHEMICAL INJURIES

EXAMINATION

- Prior to a full ophthalmic exam, the pH of both eyes should be checked. If the pH is not in physiologic range, then the eye must be irrigated to bring the pH to an appropriate range (between 7 and 7.2). It is recommended to wait at least five minutes after irrigation before checking the pH to ensure that the pH does not rise or fall secondary to retained particulate matter.

- The palpebral fissures should be checked and the fornices should be swept during the initial exam. Both the palpebral and bulbar conjunctiva should be examined with fluorescein under a cobalt blue light. As above, retained particulate matter can cause persistent damage, despite irrigation.

Roper Hall Classification for Ocular Surface Burns			
Grade	**Prognosis**	**Cornea**	**Conjunctiva/Limbus**
I	Good	Corneal epithelial damage	No limbal ischemia
II	Good	Corneal haze, iris details visible	<1/3 limbal ischemia
III	Guarded	Total epithelial loss, stromal haze, iris details obscured	1/3-1/2 limbal ischemia
IV	Poor	Cornea opaque, iris and pupil obscured	>1/2 limbal ischemia

Dua Classification for Ocular Surface Burns				
Grade	**Prognosis**	**Clinical findings**	**Conjunctiva Involvement**	**Analogue Scale***
I	Very good	0 clock hours of limbal involvement	0%	0/0%
II	Good	< 3 clock hours of limbal involvement	< 30%	0.1-3/1-29.9%
III	Good	Between 3-6 clock hours of limbal involvement	30-50%	3.1-6/31-50%
IV	Good to guarded	Between 6-9 clock hours of limbal involvement	50-75%	6.1-9/51-75
V	Guarded to poor	Between 9 and 12 clock hours of limbal involvement	75-100%	9.1-11.9/75.1-99.9%
VI	Very poor	Total limbus (12 clock hours) involved	Total conjunctiva (100%) involved	12/100%

*The analogue scale records the amount of limbal involvement in clock hours of affected limbus/percentage of conjunctival involvement. The conjunctival involvement should be calculated only for the bulbar conjunctiva, up to including the conjunctival fornices.

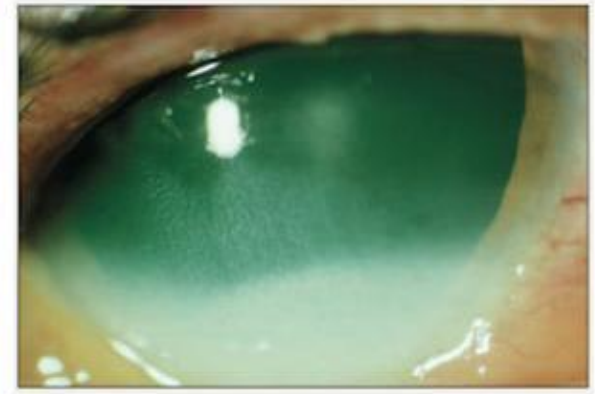
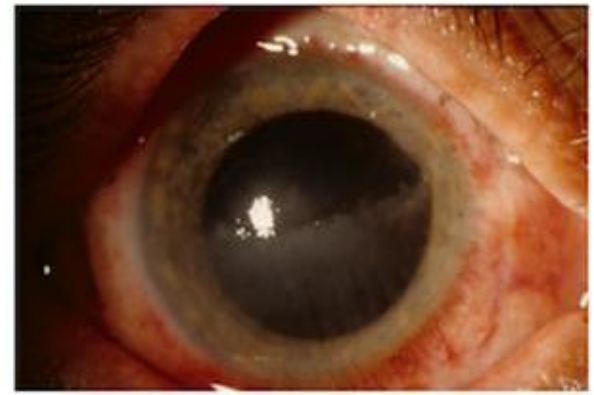


Figure A (Above) B (Below)



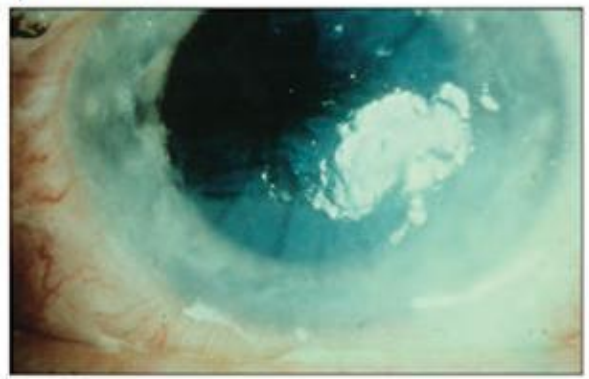


Figure C (Above)

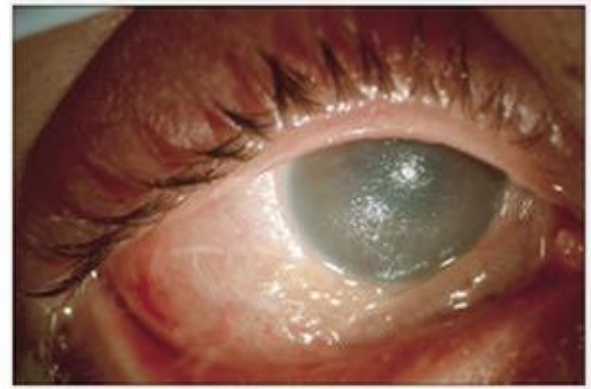


Figure D (Above)

Image Legend

Figure A – Acute Grade II burn. **Figure B-** Grade II burn one week after presentation. **Figure C-** Acute grade III burn with corneal haze. Involvement of approximately 6 clock hours. **Figure D-** Acute grade IV burn (Roper Hall) and grade VI (Dua). Images courtesy of Dr. Kathryn Colby and Dr. James Chodosh (Massachusetts Eye and Ear Infirmary).

CHEMICAL INJURIES

MANAGEMENT

- Early irrigation is critical in limiting the duration of chemical exposure. The goal of irrigation is to remove the offending substance and restore the physiologic pH.
- To position the patient for irrigation, the patient should ideally be seated upright with their head supported and tilted toward the affected side. Irrigation may require manual opening of the eyelid of the affected eye to combat blepharospasm. The irrigating fluid should be administered nasal to lateral, poured away from the non-affected eye to prevent injury to that eye. Covering the uninjured eye with a shield may help prevent additional chemical injury from irrigation spills. During irrigation, the patient should be asked to blink frequently. Additionally, during irrigation, the patient should be asked to look in all directions to ensure that the conjunctival sacs are irrigated.

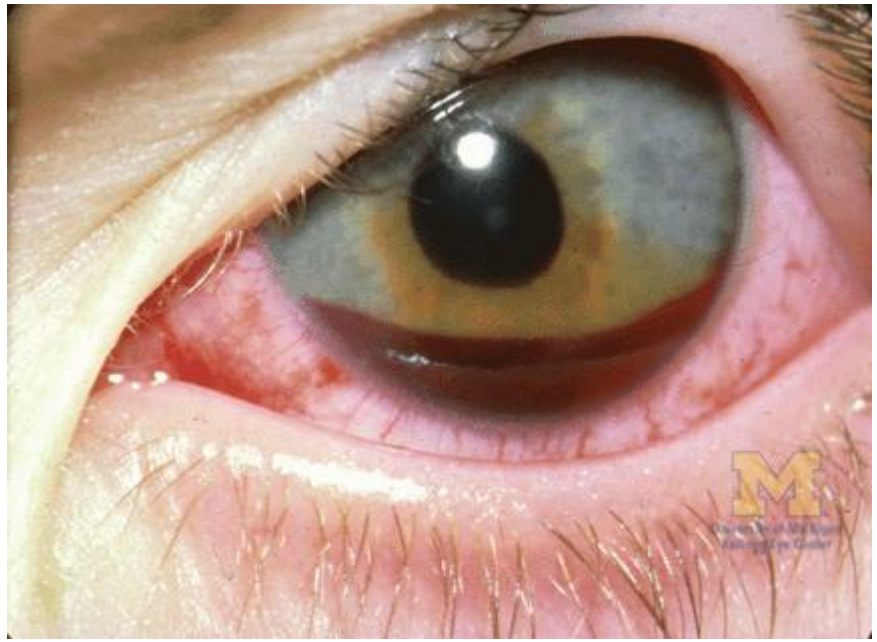
CHEMICAL INJURIES

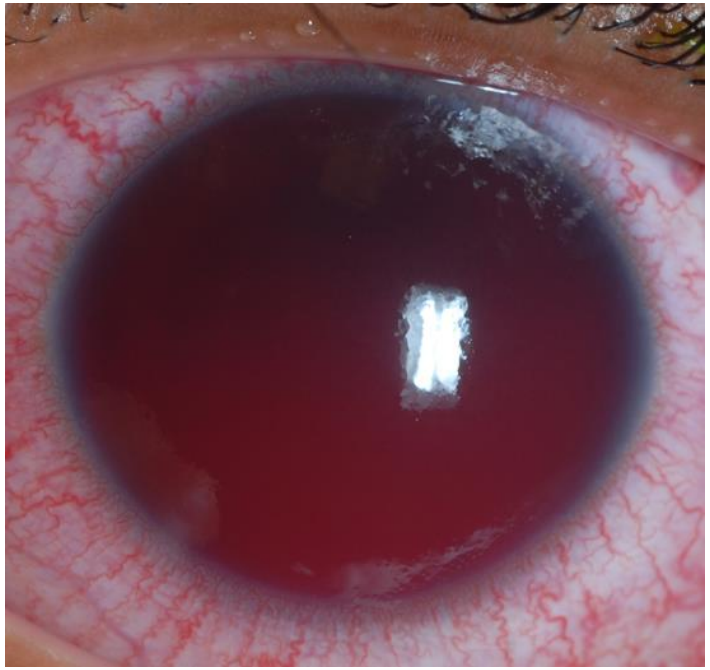
MANAGEMENT

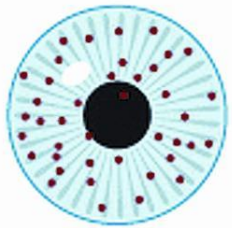
- Topical antibiotics
- Cycloplegic agents
- Artificial tears
- Steroid drops
- Oral vitamin C supplementation +/- Doxycycline

BLUNT TRAUMA

- It is important to ask for a detailed history, mechanism of injury and force of trauma.
- Ocular signs of blunt trauma include:
  - Subconjunctival haemorrhage
  - Traumatic mydriasis (fixed dilated pupil)
  - Hyphaema (blood in the anterior chamber)
  - Iridodialysis
  - Traumatic iritis and cataract
- Refer to ophthalmologist urgently for assessment if presenting with h/o high impact trauma and visual symptoms, to exclude trauma to the globe and retinal pathology like commotio retinae, retinal tears and vitreous haemorrhage.





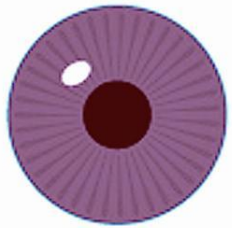


Microhyphema
Circulating red blood cells

Grade I
≤ 1/3 anterior chamber vol.

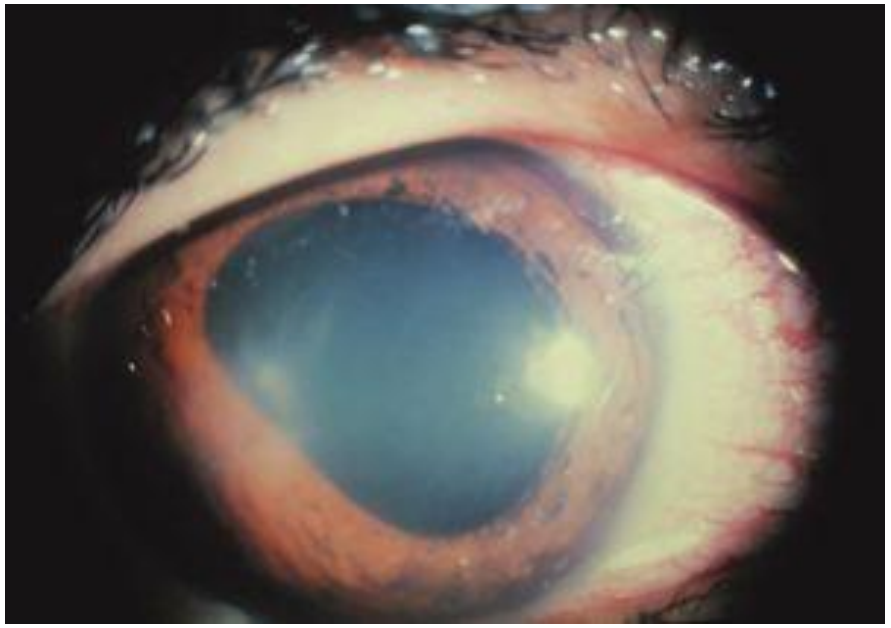
Grade II
1/3 - 1/2 anterior chamber vol.

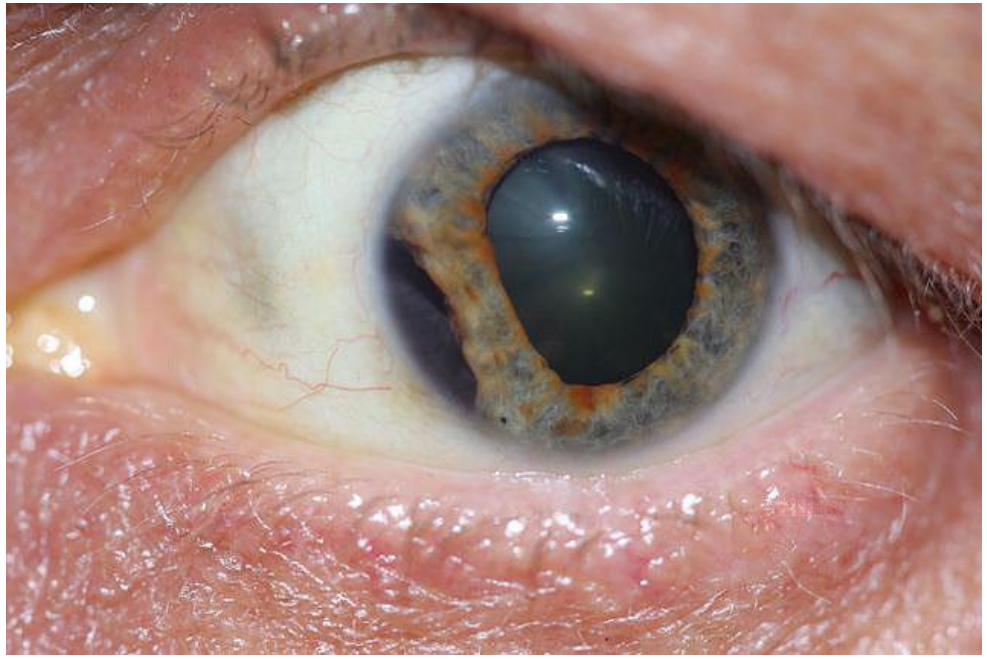
Grade III
> 1/2 anterior chamber vol.



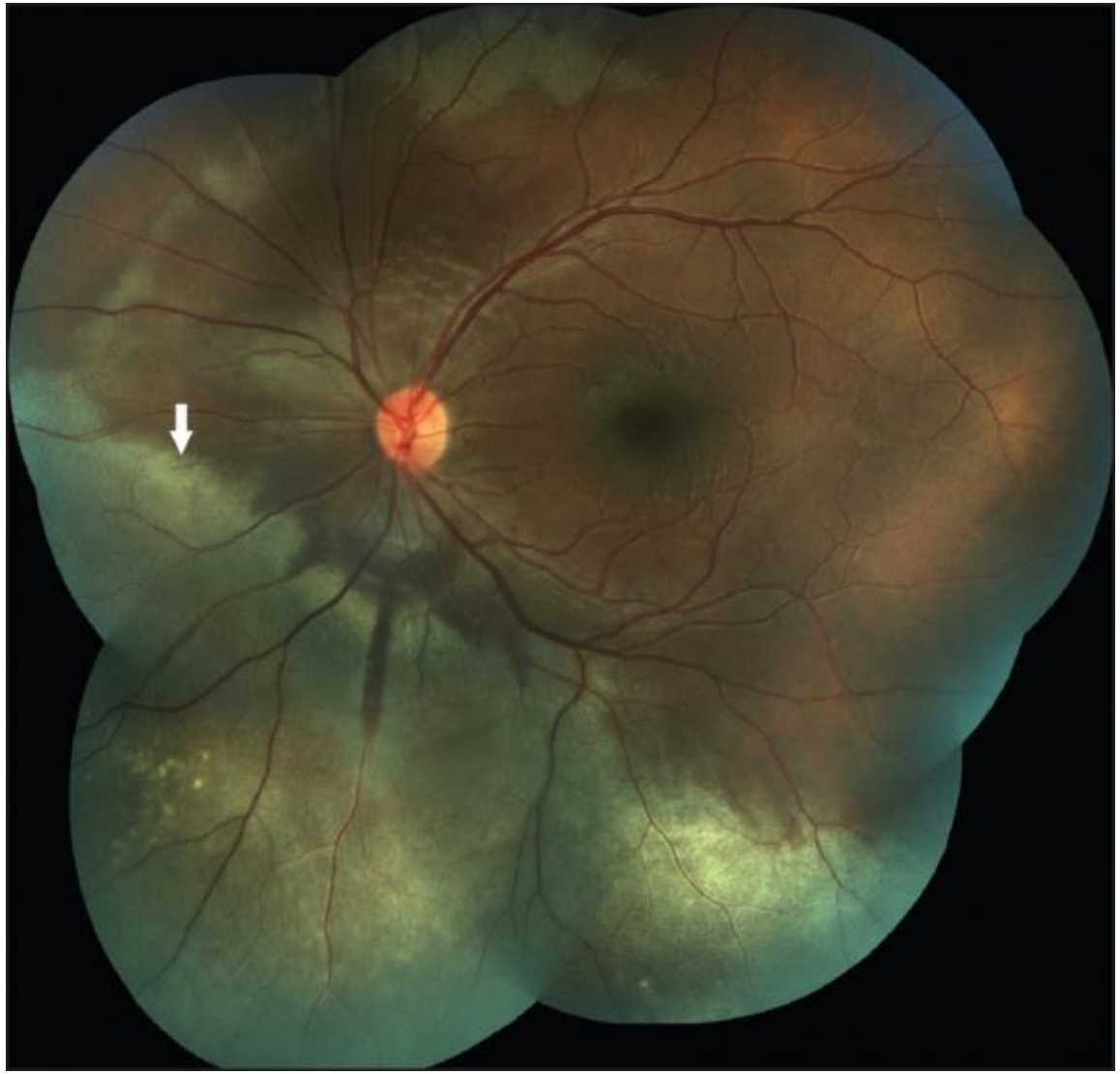
Grade IV
Total anterior chamber vol.
"eight ball hyphema"

TRAUMATIC MYDRIASIS AND IRIDODIALYSIS





COMMOTIO RETINAE

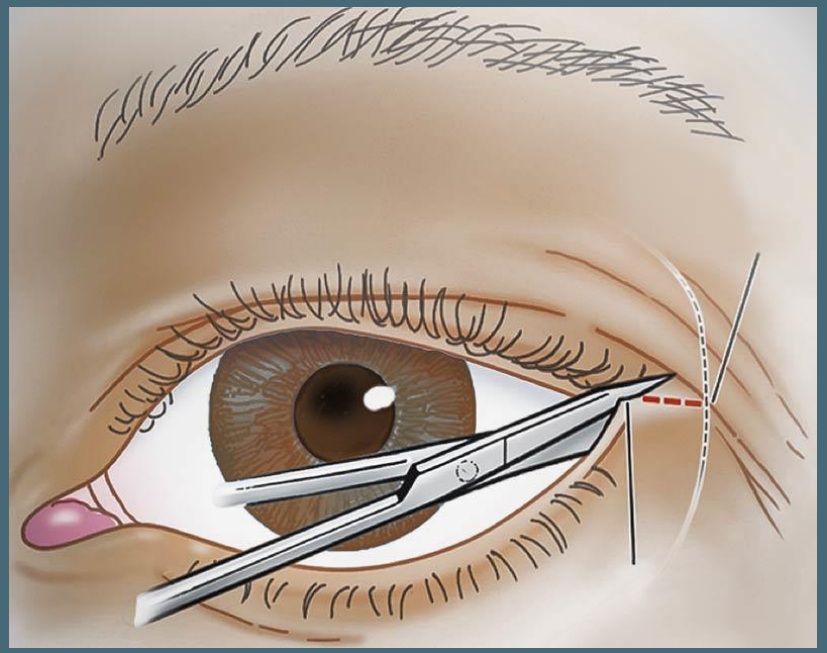


ORBITAL COMPARTMENT SYNDROME

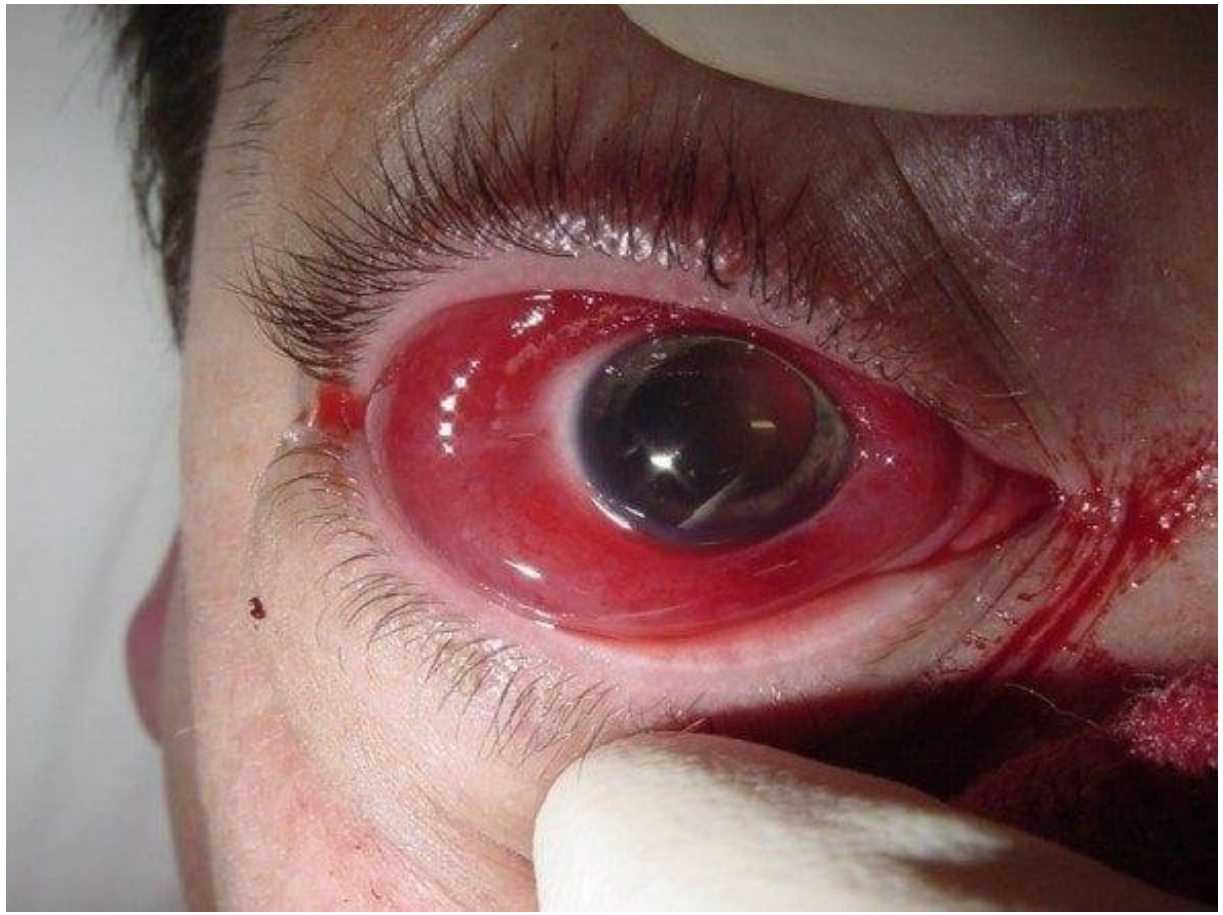
- One of the few true Ophthalmic emergencies that require prompt identification of the signs and urgent treatment.

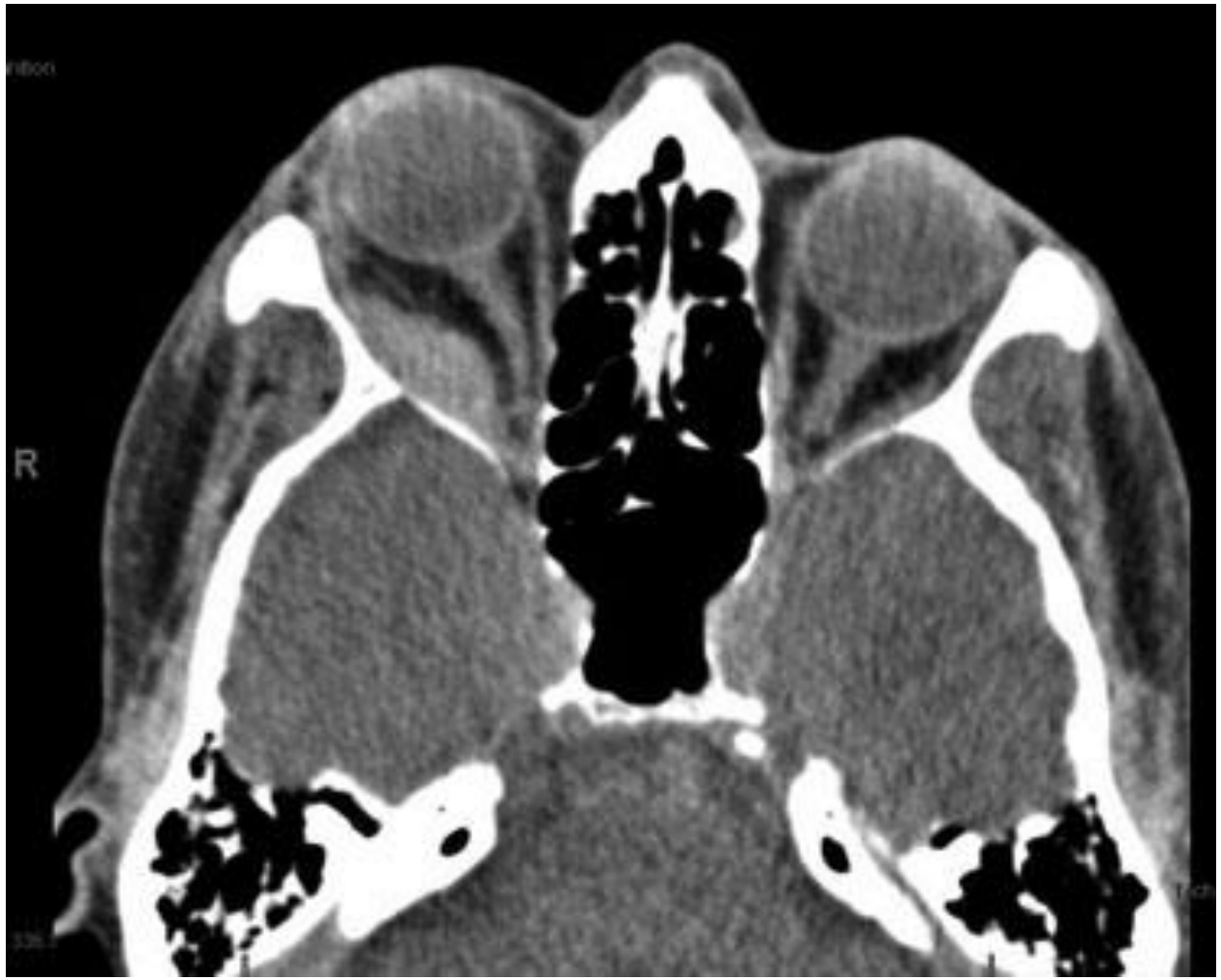
- OCS is a vision threatening elevation of intra-orbital pressure that exceeds the vascular perfusion pressure of the ophthalmic artery. It can result in ischemia and irreversible vision loss if not corrected emergently. It is most observed after trauma.

ORBITAL COMPARTMENT SYNDROME



- Important physical exam findings include
  - periorbital haemorrhage and subconjunctival haemorrhage
  - decreased visual acuity in the affected eye
  - relative afferent pupillary defect
  - elevated IOP
  - tense orbital haematoma
  - limited motility
  - proptosis and resistance to retropulsion
  - colour vision may be affected, particularly with red desaturation.
- OCS is first and foremost a clinical diagnosis. No radiographic test should ever pre-empt treatment if clinical suspicion is high, especially if there is a decrease or loss of vision.
- Treatment is with Lateral Canthotomy and Inferior Cantholysis

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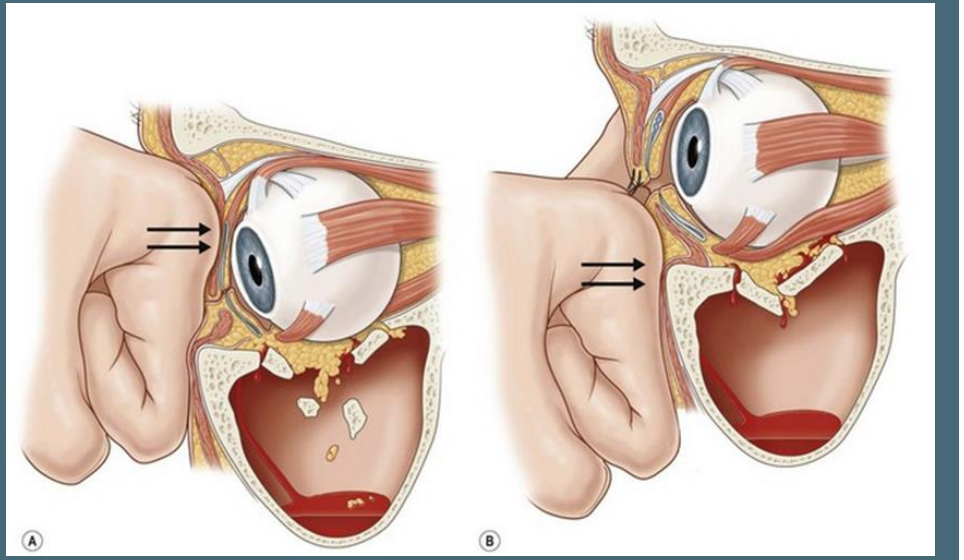
ORBITAL FRACTURES



- It is estimated that 30-40% of all facial fractures involve the orbit.
- Most orbital fractures occur in males in their second decade of life. In adults, MVAs and assault are the most common mechanisms of injury. However, in paediatric patients falls and sports-related injuries are more common.
- Fractures of the orbit are common and challenging to manage. They deserve special consideration because both surgical and observational management may result in compromise to vision and/or globe position.
- Most orbital fractures require a multi-disciplinary approach to management including ophthalmologists, orthoptists, ENT, maxillofacial surgeons and neurosurgeons.

ORBITAL FRACTURES

ASSESSMENT

- **History**

  Mechanism of injury

  Diplopia, areas of numbness, pain, epistaxis, visual symptoms

- **Clinical examination**

  Periorbital ecchymoses/oedema, surgical emphysema, globe position (exo-/ enopthalmos, dystopia), globe pulsation, pupillary responses/ RAPD, resistance to retropulsion, ocular motility, SCH, orbital rim discontinuity. Associated ocular trauma. Potential cervical/ head injury, collapse may be due to oculocardiac reflex (EOM entrapment)

ORBITAL FRACTURES

INVESTIGATIONS

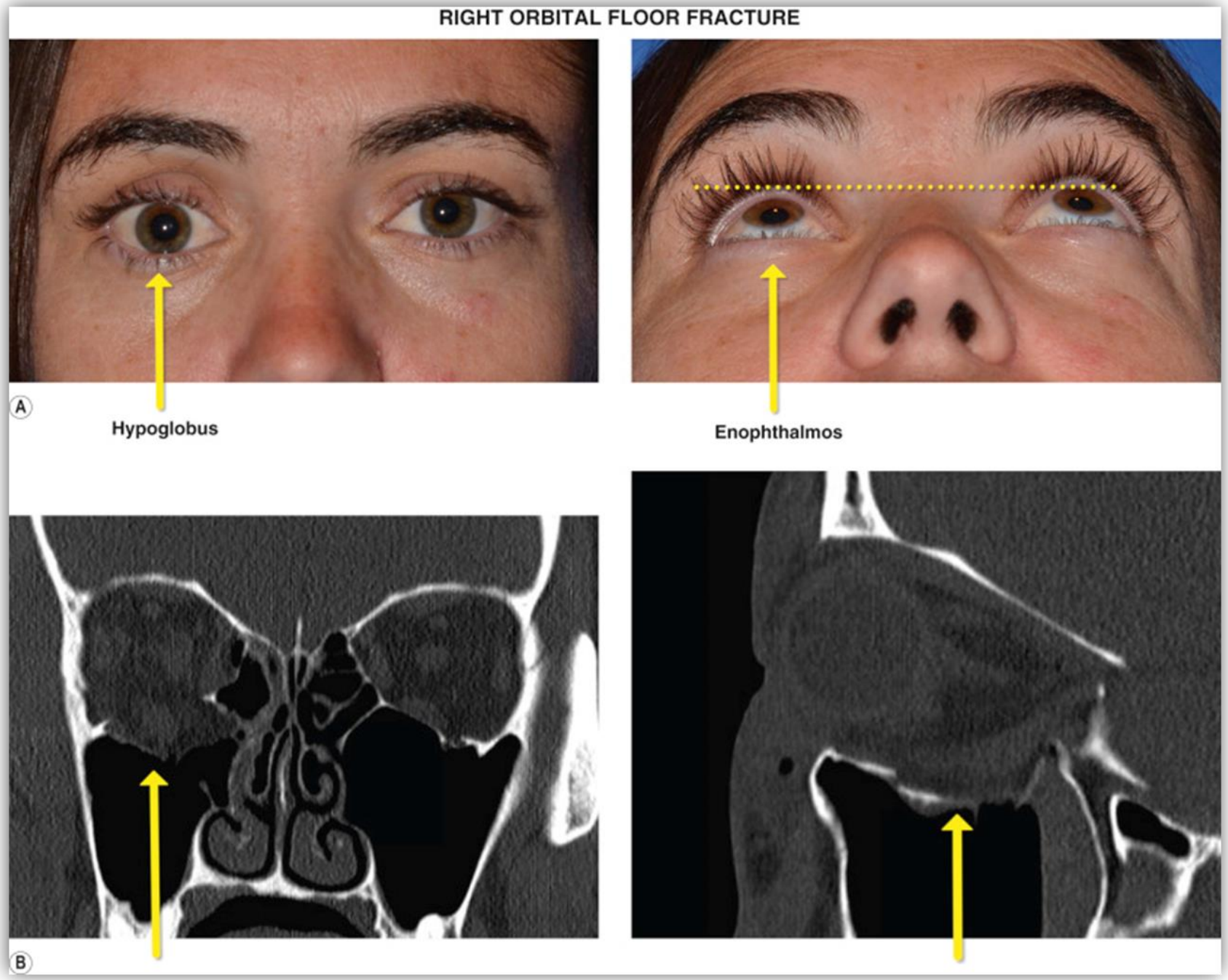
- CT (2mm axial and coronal slices) – to identify fractures, prolapsed orbital fat/EOM and haemorrhage
- Facial Xrays
- Hess tests – useful for mechanical restrictive patterns and monitory recovery

ORBITAL FRACTURES

MEDICAL MANAGEMENT

- Patients with dysmotility may benefit from a short (5-7 day) course of steroids (0.75 – 1.0 mg/kg per day of prednisone) in the acute setting if there are no contraindications. Such treatment may help periorbital and extraocular muscle oedema to subside more quickly to determine if the patient's dysmotility is transient or if surgery is necessary.

- Antibiotics may be prescribed in the acute setting. Patients should be informed that nose-blowing could lead to orbital or soft tissue emphysema and is discouraged for 4-6 weeks after the injury.


RIGHT ORBITAL FLOOR FRACTURE
A
Hypoglobus
Enophthalmos
B


THYROID EYE DISEASE

THYROID EYE DISEASE

Typically, hyperthyroid patients present with proptosis (exophthalmos), tearing, lid retraction, FB sensation, retroocular pain, peri-orbital oedema and symptoms of double vision/restrictive eye movements.

'I'M SLOw': an acronym to remember which orbital muscles are involved first due to anti-TPO and anti-TG antibodies resulting in inflammation of the orbital muscles and swelling of retro-orbital fat tissue.

Treatment: mild disease may require only lubricants and Se supplementation vs severe which may require high dose steroids and immunosuppressants. Severe cases may require orbital decompression. Advise smoking cessation as this is imperative for resolution.

EUGOGO GUIDELINES

Assessment of activity by the clinical activity score (CAS)*. CAS < 3 = inactive GO; CAS ≥ 3 = active GO. A ten-item CAS, including an increase in exophthalmos of ≥2 mm, a decrease in eye motility of ≥8° or a decrease in visual acuity in the last 1–3 months, is useful to assess progression of GO after the first visit.

Assessment of activity	
1.	Spontaneous retrobulbar pain
2.	Pain on attempted upward or downward gaze
3.	Redness of eyelids
4.	Redness of conjunctiva
5.	Swelling of caruncle or plica
6.	Swelling of eyelids
7.	Swelling of conjunctiva (chemosis)

MILD GO

General recommendations

- Refrain from smoking
- Treat thyroid dysfunction (preferably with antithyroid drugs, especially if risk factors for deterioration/progression of GO are present (see below)
- Avoid iatrogenic hypothyroidism in treating patients with GD/GO
- Referral to thyroid-eye clinics if risk factors present (active GO, smoker, high TSHR-Ab, unstable / severe hyperthyroidism)
- Search for dry eye syndrome

Management

Local treatment

- Artificial tears, especially when dry eye present
- Ophthalmic gels (cornea protection during the night)

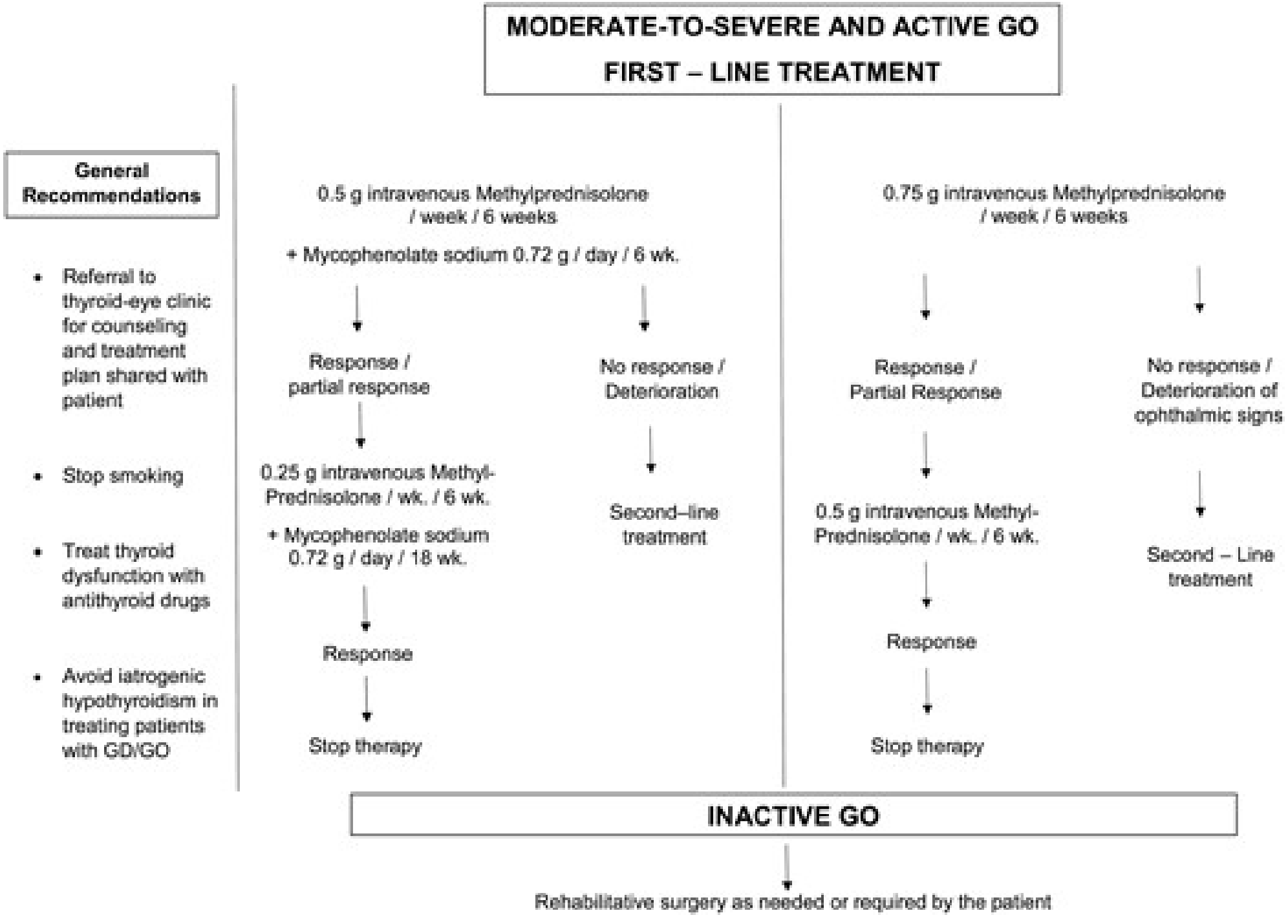
Systemic adjunct therapy for active GO

- Selenium supplementation for six months (fasting intake)

Quality of life markedly impaired

↓

Discuss low dose immunomodulatory (active GO) or rehabilitative surgery (inactive GO) following extensive counseling and shared decision


MODERATE-TO-SEVERE AND ACTIVE GO
FIRST – LINE TREATMENT
General Recommendations
• Referral to thyroid-eye clinic for counseling and treatment plan shared with patient
• Stop smoking
• Treat thyroid dysfunction with antithyroid drugs
• Avoid iatrogenic hypothyroidism in treating patients with GD/GO
0.5 g intravenous Methylprednisolone / week / 6 weeks
+ Mycophenolate sodium 0.72 g / day / 6 wk.
Response / partial response
0.25 g intravenous Methyl-Prednisolone / wk. / 6 wk.
+ Mycophenolate sodium 0.72 g / day / 18 wk.
Response
Stop therapy
No response / Deterioration
Second–line treatment
0.75 g intravenous Methylprednisolone / week / 6 weeks
Response / Partial Response
0.5 g intravenous Methyl-Prednisolone / wk. / 6 wk.
Response
Stop therapy
No response / Deterioration of ophthalmic signs
Second – Line treatment
INACTIVE GO
Rehabilitative surgery as needed or required by the patient

THANK YOU

Question time ☺