EYE EMERGENCIES

OCULAR INFECTIONS:

PRESEPTAL (PERIORBITAL) AND POSTSEPTAL (ORBITAL) CELLULITIS:

- Preseptal or periorbital cellulitis is an infection of the eyelids and periocular tissues that is ANTERIOR TO THE ORBITAL SEPTUM
 - o It is generally benign
- Postseptal or ORBITAL CELLULITIS, is an infection of the orbital soft tissues POSTERIOR TO THE ORBITAL SEPTUM → LIFE AND VISION THREATENING → must be treated as an inpatient with IV antibiotics
- The OUTWARD APPEARANCE OF BOTH CONDITIONS ARE VERY SIMILAR → excessive tearing, fever, erythema, oedema, warmth and tenderness to palpation
- CT differentiates the two

Table 236-3 Differential Diagnosis of Preseptal and Postseptal Cellulitis
Preseptal cellulitis
Postseptal cellulitis
Subperiosteal abscess
Orbital abscess
Cavernous sinus thrombosis
Dacryoadenitis
Dacryocystitis
Hordeolum
Bacterial and viral conjunctivitis
Contact dermatitis
Herpes zoster
Herpes simplex

PRESEPTAL CELLULITIS:

- o Usually associated with URTI, especially paranasal cellulitis
- o Primarily a disease of childhood (<10)
- THE EYE ITSELF IS **NOT INVOLVED**:
 - VISUAL ACUITY AND PUPILLIARY REACTION ARE MAINTAINED
- o Full painless, ocular motility is PRESERVED
- o CT scan indicated when there is decreased ocular motility or other signs of orbital involvement or in the child when exam is unreliable
- o TREATMENT:
 - In the nontoxic or adult patient → as an outpatient with oral antibiotics
 - If more ill, treat as per orbital cellulitis (see below)

In children less than 5 years, the major pathogens are Staphylococcus aureus, Streptococcus pneumoniae, Streptococcus anginosus/milleri group, or Haemophilus influenzae type b (Hib) in the unvaccinated. If the child is well, use:

1 amoxycillin+clavulanate 22.5+3.2 mg/kg orally, 12-hourly for 7 days 2 cephalexin 12.5 mg/kg orally, 6-hourly for 7 days. In adults and older children, particularly those vaccinated against Hib, or with a local lesion such as a stye, dacryocystitis, impetigo or a wound, staphylococcal infection is more di/flucloxacillin 500 mg (child: 12.5 mg/kg up to 500 mg) orally, 6-hourly for 7 days. For patients hypersensitive to penicillin (excluding immediate hypersensitivity, see Table 2.2), use cephalexin 500 mg (child: 12.5 mg/kg up to 500 mg) orally, 6-hourly for 7 days.

For patients with immediate penicillin hypersensitivity (see Table 2.2), use:

clindamycin 450 mg (child: 10 mg/kg up to 450 mg) orally, 8-hourly for 7 days.

In the severely ill, take blood cultures and a computerised tomography (CT) scan to exclude sinusitis-associated infection. Treat empirically as for orbital cellulitis.

POSTSEPTAL OR ORBITAL CELLULITIS:

- o Occurs most frequently from spread of paranasal sinusitis → ethmoid sinus most frequently implicated
 - Trauma, intraorbital foreign body, spread of periorbital skin infection, ocular surgery and bacteraemia are also risk factors
- INFECTION IS POLYMICROBIAL
 - Consider MUCORMYCOSIS IN DIABETICS, Haemophilus in unimmunized kids
- Insidious onset
- PAIN ON EYE MOVEMENT
- PROPTOSIS
- Decrease in visual acuity
- **EXAMINATION FINDINGS:**
 - Limitation of extraocular muscles
 - Chemosis
 - **Proptosis**
 - Abnormal pupillary response
 - Decreased visual acuity
 - Involvement of CRANIAL NERVES 3,4 OR 6 SUGGESTS **CAVERNOUS SINUS THROMBOSIS**
- May lead to vision loss and needs aggressive treatment and investigation (CT SCAN) → urgent OPHTHALMOLOGY CONSULT

Orbital (postseptal) cellulitis

Orbital (postseptal) cellulitis is a less common but more serious condition, generally arising from infection of paranasal sinuses (especially untreated) or orbital trauma. Clinical symptoms are more pronounced and the following may be present: reduced vision, limited or painful extra-ocular movement, or proptosis. If suspected, blood cultures and a CT scan of sinuses are essential, as delay in diagnosis may result in serious consequences. Urgent surgical drainage of the sinuses or of an orbital, subperiosteal or intracranial abscess

Causative pathogens include Haemophilus influenzae, Streptococcus pneumoniae, Streptococcus anginosus/milleri group, Staphylococcus aureus, aerobic Gram-negative bacteria and anaerobes. It may be caused by fungi in the immunocompromised or patients with diabetes

Use IV therapy initially (duration may vary from 3 days to 2 weeks), then change to oral therapy for a further 10 days. While awaiting the results of investigations, suitable empirical regimens include

1 cefotaxime 2 g (child: 50 mg/kg up to 2 g) IV, 8-hourly OR THE COMBINATION OF 1 ceftriaxone 2 g (child: 50 mg/kg up to 2 g) IV, daily

PLUS

di/flucloxacillin 2 g (child: 50 mg/kg up to 2 g) IV, 6-hourly

amoxycillin+clavulanate 875+125 mg (child: 22.5+3.2 mg/kg up to 875+125 mg) orally, 12-hourly for a further 10 days.

For patients with hypersensitivity to penicillin (see Table 2.2) or if methicillin-resistant S. aureus (MRSA) is suspected or cultured, seek expert advice

 COMPLICATIONS → Raised intraocular pressure (lateral canthotomy), cavernous sinus thrombosis, frontal bone osteomyelitis, meningitis, subdural empyema, epidural abscess and brain abscess

PROBLEMS RELATED TO THE LIDS:

STYE (EXTERNAL HORDEOLUM):

• An acute bacterial infection (usually staph) of the follicle of an eyelash and adjacent sebaceous glands → has the appearance of a small pustule at the margin of the eyelid



- Warm compresses and erythromycin ointment is usually sufficient treatment
 - o Systemic antibiotics if there is surrounding cellulitis

CHALAZION:

• Acute or chronic inflammation of the eyelid secondary to blockage of one of the MEIBOMIAN oil glands



- Usually a painless lump, treat with warm compresses
- If chronic or recurrent → may require injection of corticosteroids into the lesion by ophthalmology

BLEPHARITIS:

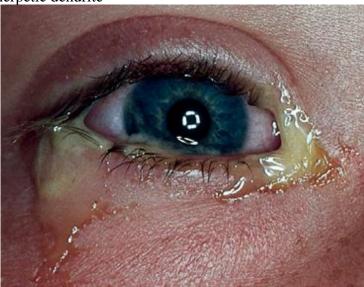
- Common cause of prolonged red eye **→** inflammation of the eyelash follicles
- Associated with seborrhoeic dermatitis and atopic dermatitis
- Symptoms → conjunctival injection, crusting, swollen, pruritic eyelids,
- Daily cleansing of the eyelids and eyelashes

CONJUNCTIVITIS:

- AN INFLAMMATORY CONDITION OF THE CONJUNCTIVA
- USUALLY VIRAL IN AETIOLOGY
- The main aim in ED is to discern the occasional case of SERIOUS BACTERIAL CONJUNCTIVITIS or CORNEAL HERPETIC INVOLVEMENT → both of these result in vision loss without aggressive treatment
- KERATOCONJUNCTIVITIS → delineates corneal involvement

BACTERIAL CONJUNCTIVITIS:

- Painless, MUCOPURULENT DISCHARGE (unilateral or bilateral)
 FREQUENTLY CAUSING ADAHERENCE OF THE EYELIDS ON AWAKENING
- Chemosis is common
- FLUORESCEIN STAINING CRUCIAL → avoid missing a corneal abrasion, ulcer or herpetic dendrite

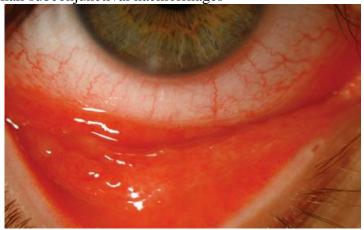


In severe cases, use:

- 1 chloramphenicol 0.5% eye drops 1 to 2 drops, every 2 hours for the first 24 hours, then decreasing to 6-hourly until discharge resolves, for up to 7 days. Chloramphenicol 1% eye ointment may be used at bedtime
 OR
- 2 framycetin 0.5% eye drops 1 to 2 drops, every 1 to 2 hours for the first 24 hours, then decreasing to 8-hourly until discharge resolves, for up to 7 days.

- Consider tobramycin eyedrops in contact lens wearers for pseudomonas coverage **VIRAL CONJUNCTIVITIS:**
 - The most common aetiology of viral conjunctivitis is ADENOVIRUS
 - Generally resolves spontaneously → influenza, measles and mumps can all cause it
 - HERPES SIMPLEX AND ZOSTER CAN BOTH CAUSE CORNEAL SCARRING AND LOSS OF VISION IF NOT TREATED
 - Typical viral conjunctivitis is preceded by an URTI and complaints are mainly of watery discharge and a painless RED EYE

 Examination is of bilateral conjunctival injection, occasional chemosis and small subconjunctival haemorrhages



- Important to examine the cornea with fluorescein to avoid missing A HERPETIC DENDRITE
- Treatment consists of cool compresses, decongestants, artificial tears for 5-7 days

EPIDEMIC KERATOCONJUNCTIVITIS:

- An adenovirus that is highly contagious and tends to occur in epidemics
- Patient complains of marked eye redness, photophobia, FB sensation and excessive tearing (EPIPHORA).
- Virus is viable for FIVE WEEKS and is resistant to standard disinfectants
 EXTREMELY CONTAGIOUS

ALLERGIC CONJUNCTIVITIS:

- Allergens can cause a watery discharge, redness and ITCHING
- Causes an oedematous conjunctivae with PAPILLAE (irregular mounds) on the inferior conjunctival fornix
- OFFENDING ALLERGEN SHOULD BE SOUGHT AND ELIMINATED
- Moderate cases → antihistamines
- Severe cases (with ophthalmology guidance) → steroid eye drops → ophthal to start

CORNEAL DISORDERS:

HERPES SIMPLEX VIRUS:

- CLINICAL FEATURES:
 - o Can affect eyelids, conjunctiva and cornea. Can give history of cold sores
 - o Complains of red eye and decreased vision
 - o Infection tends to be unilateral
 - Fluorescein staining has CLASSICAL LINEAR BRANCHING WITH TERMINAL BULBS (DENDRITIC ULCER)



- HSV KERATITIS CAN PROGRESS TO CORNEAL SCARRING AND REQUIRES PROMPT TREATMENT WITH TOPICAL ANTIVIRAL AGENTS
 aciclovir 3% eye ointment topically, 5 times daily for 14 days or for at least 3 days after healing.
 - DO NOT PRESCRIBE STEROIDS AND REFER TO AN OPHTHALMOLOGIST WITHIN 24-48 HOURS

HERPES ZOSTER OPHTHALMICUS:

- Shingles typically in the FIRST DIVISION OF THE TRIGEMINAL NERVE WITH OCULAR INVOLVEMENT
- The rash does NOT CROSS THE MIDLINE and involves only the upper eyelid
- Involvement of the nasociliary nerve is associated with cutaneous lesions on the TIP OF THE NOSE (HUTCHINSON SIGN) → predicts high likelihood of ocular involvement
- Eye involvement may take the form of \rightarrow keratitis, uveitis, retinitis, chorioiditis
- 1 famciclovir 250 mg orally, 8-hourly for 7 days or famciclovir 500 mg orally, 8-hourly for 10 days (in immunocompromised patients)
- 1 valaciclovir 1 g orally, 8-hourly for 7 days

OR

- 2 aciclovir 800 mg (child: 20 mg/kg up to 800 mg) orally, 5 times daily for 7 days (aciclovir is preferred in children and in pregnancy, seek expert advice).
 - Pain reduction can be achieved with topical cycloplegic agents (CYCLOPENTOLATE 1%)

Consider admission if the patient is immunocompromised or systemically ill
 IV acyclovir

CORNEAL ULCER:

- A serious infection involving multiple layers of the cornea and is a major cause of impaired vision and blindness worldwide
- Initial disruption can be due to desquamation, trauma or direct microbial invasion
- Widespread use of contacts has led to a rise of PSEUDOMONAS INFECTION
 incidence rises dramatically in those who use extended-wear lenses and those who sleep with them in situ
- Patients complain of discharge, ocular pain/FB sensation, photophobia or blurred vision. DECREASED VISUAL ACUITY IF IN THE CENTRAL AXIS
 - Slit lamp reveals flare and cell form iritis and occasionally a HYPOPYON



CORNEAL ULCER SEEN AT FIVE O'CLOCK POSITION

- Need to be treated aggressively with topical antibiotics (consider ciprofloxacin if contact wearer)
- STEROID EYE DROPS SHOULD NEVER BE INITIATED IN ED
- DO NOT PATCH THE EYE BECAUSE OF THE RISK OF PSEUDOMONAS INFECIOTN, WHICH CAN CAUSE RAPID, AGGRESSIVE ULCERATION WITH CORNEAL PERFORATION AND MELTING
- **COMPLICATIONS** corneal scarring, corneal perforation, anterior and posterior synechiae, glaucoma and cataracts

UVEAL TRACT PROBLEMS:

UVEITIS/IRITIS:

- Inflammation of the anterior segment of the uveal tract
- It not a true ocular emergency but does require follow up
- Pain is caused by irritation of the ciliary nerves and ciliary muscle spasm
- WBC release from the uveal vessels may be seen in the anterior chamber with slit lamp appearance being that of "cells" → "snowflakes in the headlights"
- CLINICAL FEATURES:

- Unilateral pain, can be bilateral in systemic disease (associated in these cases with arthritis, GI symptoms, urethritis)
- o CONSENSUAL PHOTOPHOBIA (shine light on the unaffected eye causes pain in affected eye) → highly suggestive or iritis
- o Diffuse conjunctival injection
- Hypopyon in severe disease
- DIFFERENTIAL DIAGNOSIS:

Table 236-5 Differential Diagnosis of Iritis					
Systemic diseases	Malignancies				
Juvenile rheumatoid arthritis	Leukemia				
Ankylosing spondylitis	Lymphoma				
Ulcerative colitis	Malignant melanoma				
Reiter syndrome	Trauma/environmental				
Behçet syndrome	Corneal foreign body				
Sarcoidosis	Post-traumatic (blunt trauma)				
Infectious	Ultraviolet keratitis				
Tuberculosis					
Lyme disease					
Herpes simplex					
Toxoplasmosis					
Varicella zoster					
Syphilis					
Adenovirus					

• TREATMENT → aimed at reducing inflammation and preventing complications such as posterior synechiae → early ophthalmologic consultation. Use LONG-ACTING CYCLOPLEGIC TO BLOCK THE PUPILLARY SPHINCTER (homatropine lasts 2-4 days or tropicamide lasts 24 hours)

DISORDERS OF THE VITREOUS:

ENDOPHTHALMITIS:

- Inflammation (usually infection) of the aqueous or vitreous humeur that frequently leads to loss of vision
- MOST FREQUENTLY IT IS POST-SURGICAL
 - Other causes → penetrating ocular injury, and rarely by haematogenous spread
- Symptoms → headache, eye pain, photophobia, vision los and ocular discharge
- Examination \rightarrow scleral injection, chemosis, hypopyon, evidence of uveitis
- TREATMENT → aspiration of the vitreous or pars plana vitrectomy and administration of INTRAVITREAL ANTIBIOTICS AND STEROIDS

• DO NOT USE TOPICAL ANTIBIOTICS AS THEY ARE TOXIC TO INTRAOCULAR CONTENTS

If there is to be significant delay before obtaining advice or admitting the patient to a specialised unit, use:

ciprofloxacin 750 mg (child: 20 mg/kg up to 750 mg) orally, as a single dose

PLUS

vancomycin 25 mg/kg up to 1.5 g (child less than 12 years: 30 mg/kg up to 1.5 g) IV, as a single dose (slow infusion required, see <u>Dosing and monitoring of vancomycin</u>).

Alternatively, use

gentamicin (adult and child) 5 mg/kg IV, as a single dose

PI IIS

cephazolin 2 g (child: 50 mg/kg up to 2 g) IV, as a single dose.

Do not use topical antibiotics as preservatives are toxic to intraocular contents.

VITREOUS HAEMORRHAGE:

- The vitreous is avascular and firmly attached to the eye → traction at these sites of attachment can cause haemorrhage
- Commonest causes are PROLIFERATIVE DIABETIC NEPHROPATHY, POSTERIOR VITREOUS DETACHMENT (ELDERLY), OCULAR TRAUMA (SHAKEN BABY)
- Unusual cause → subhyaloid haemorrhage associated with SAH
- History may include sudden painless vision loss and sudden appearance of black spots or hazy vision
- Check INR, platelets and withhold antiplatelets
- Ocular US useful in diagnosis and excluding retinal detachment (see later)

SUBCONJUNCTIVAL HAEMORRHAGE:

- The fragile conjunctival vessels can rupture from trauma, sudden Valsalva, HT or spontaneously
- Eye exam is normal other than presence of haemorrhage
- REASSURANCE IS KEY → usually resolves in 2 weeks



CONJUNCTIVAL ABRASION, LACERATION AND FOREIGN BODY:

- The conjunctiva has less innervation than the cornea, so abrasions are far less symptomatic than corneal abrasions
- Vision should not be affected
- Perform SEIDEL TEST to exclude perforation of the globe → consider in high velocity injuries → can be negative if a full-thickness laceration is small or has spontaneously closed
- Superficial lacerations without other ocular injury only require topical erythromycin or chloramphenicol ointment
- Any suspicion of globe laceration requires immediate ophthalmologic referral

CORNEAL ABRASION, LACERATION AND FOREIGN BODY:

- The corneal epithelium is fragile and easily damage
 - o It is richly innervated, therefore very painful when injury occurs
 - o Epithelium regenerates rapidly, so healing time is short (24-48 hours)
- Damaged epithelium is a portal of entry for bacteria, viruses and fungi

CORNEAL ABRASION:

- Injury causes intense pain that may be delayed several hours
- Ask about foreign body sensation and mechanism of injury (if known)
- Blepharospasm may be present with severe pain → relief of pain with topical anaesthesia IS VIRTUALLY DIAGNOSTIC OF CORNEAL ABRASION
- Decreased visual acuity may occur if the abrasion is in the central visual axis or if there is associated iritis
- Seidel test should be negative
- TREATMENT → reliving pain and preventing infection are cornerstones. PATCHING THE EYE DOES NOT PROMOTE HEALING, but some patients feel better. DO NOT PATCH AFTER FINGERNAIL SCRATCHES, VEGETABLE MATTER OR CONTACT LENS → high risk for infection
- Can use topical NSAIDs (ketolorac, does not impair healing), cycloplegics (not atropine as its effect lasts 2 weeks) and topical antibiotics
 - DO NOT EVER PRESCRIBE TOPICAL ANAESTHETICS, AS THEY INHIBIT CORNEAL HEALING AND OBLITERAL NORMAL CORNEAL PROTECTIVE MECHANISMS (I.E. BLINKING)
- For those with large abrasions or abrasions in the central visual axis → follow up with ophthalmology in 24 hours

CORNEAL LACERATION:

• Full thickness corneal lacerations can be characterised by a misshapen iris, macro or micro-hyphaemia, decrease in visual acuity and shallow anterior chamber

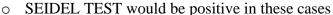
- SEIDEL TEST WILL BE POSITIVE → small lacerations can close spontaneously and Seidel will be negative, and there may be no gross distortion of anatomy
- Pain out of proportion to physical findings, decreased in visual acuity or other unexpected ocular symptoms may be the only findings
- MAINTAIN HIGH INDEX OF SUSPICION → prompt referral as patients can quickly develop endophthalmitis or traumatic cataract → vision-threatening

ULTRAVIOLET KERATITIS:

- AKA SNOW BLINDNESS or "welder's flash"
- Effects are cumulative
- Corneal cells do not die immediately → delay of up to 6-12 hours then severe pain and photophobia
- Slit lamp punctate corneal oedema, and abrasions
- Treatment → double patching, use of cycloplegics, topical antibiotics and oral analgesia

CORNEAL FOREIGN BODIES:

- Usually superficial and benign, but penetration of a foreign body into the globe can cause loss of vision
- Visual acuity should not be decreased unless in visual axis
- Presence of hyphaema suggests globe perforation





- Metallic foreign bodies can produce rust rings that are toxic to the corneal tissue
 - o If a rust ring is present, an ophthalmic burr can remove superficial rust, but it often reaccumulates in the next day → discharge on ointment to follow up with ophthalmology for additional burring if needed
 - NO ED DRILL BURRING IF THE RUST RING IS PRESENT IN THE VISUAL AXIS
 - The deeper the stromal involvement, the greater the risk of stromal scarring



RUST RING

- FOREIGN BODY REMOVAL:
 - o Can be done with moistened cotton applicator
 - Can also use 25g needle (bevel up) to remove superficial foreign bodies using slit lamp to guide

LID LACERATIONS:

- AUTOMATIC REFERRAL FOR SURGICAL REPAIR IF:
 - o Involvement of the lid margin
 - o Those within 6-8mm of the medial canthus or involving the lacrimal duct or sac
 - o Those involving thee inner surface of the lid
 - o Those associated with PTOSIS
 - Those involving the tarsal plate or levator palpebrae muscle (suspect with a horizontal lacerations with ptosis or when orbital fat is seen)
- Deep lacerations medial to the punctum potentially can transect the canalicular system → need urgent opthalmological assessment
 - Instillation of fluorescein into the eye with subsequent appearance in the wound indicates loss of canalicular integrity
 - o Need to go to OT within 24-36 hours
 - o Discharge on oral cephalexin with follow up arranged



- Consider the possibility of corneal laceration and globe rupture with full thickness lid lacerations
- Very small lacerations at the lid edge (<1cm) do not need suturing and can heal spontaneously

BLUNT EYE TRAUMA:

- FIRST STEPS OF ASSESSMENT → VISUAL ACUITY, ANTERIOR CHAMBER AND INTEGRITY OF THE GLOBE
- Need adequate inspection → can fashion a bent paperclip to work as an eyelid speculum
- If the anterior chamber is flat, a ruptured glboe is certain
- HYPHAEMA IS ALSO EVIDENCE OF SIGNIFICANT TRAUMA → metal shield over the eye and consult ophthalmology (do not manipulate the eye further if this is suspected)

HYPHAEMA:

 Blood or blood clots in the anterior chamber and in the setting of trauma, usually results from bleeding from a RUPTURED IRIS ROOT VESSEL (can occur spontaneously in sickle cell disease)



OCCASIONALLY HYPHAEMA WILL FORM CLOTS AND NOT LAYER OUT

- TREATMENT → ALL HYPHAEMA SHOULD BE SEEN BY OPHTHALMOLOGY
 - Elevate patients head to promote settling of suspended RBCs inferiorly to prevent occlusion of the trabecular meshwork
 - o Dilate the pupil to prevent "PUPILLARY PLAY", which can stretch iris vessels and worsen bleeding
 - o Control of intraocular pressure \rightarrow topical beta-blockers, IV mannitor, topical α -agonists (apraclonidine), carbonic anhydrase inhibitors
 - Do NOT GIVE CARBONIC ANHYDRASE INHIBITORS TO PEOPLE WITH SICKLE CELL DISEASE as it will lower aqueous pH and thus cause RBC to sickle, further clogging outflow through the trabecular meshwork
 - o Re-bleeding can occur 3-5 days later in up to 30% cases → may need anterior chamber "washouts" by ophthalmology

BLOW-OUT FRACTURES:

- Most frequent sites are through the INFERIOR WALL (maxillary sinus) and MEDIAL WALL (ethmoid sinus)
 - o Fractures of medial wall are associated with subcutaneous emphysema (exacerbated by sneezing)
 - Fractures of the inferior wall with ENTRAPMENT OF THE INFERIOR RECTUS CAN CAUSE RESTRICTION OF UPGAZE AND DIPLOPIA



- Isolated blow-out fractures do not require immediate surgery (with or without entrapment) and can be referred to maxillofacial or ophthalmology.
- All blow-out fractures with normal initial eye exam should be referred to an ophthalmologist for an outpatient full dilated examination to rule out any unidentified retinal tears or detachments

RUPTURED GLOBE:

- A VISION-THREATENING EMERGENCY THAT MAY BE EASILY MISSED
 - A penetrating wound of the cornea caused by a tiny piece of metal launched form a grinder may easily be overlooked and requires a high index of suspicion
- MECHANISM OF INJURY:
 - o The globe tends to rupture at the thinnest points of the sclera → the limbus, and at the insertion of extraocular muscles
 - o Any projectile has the potential to penetrate the eye and suspect globe penetration with any puncture o laceration of the eyelid or periorbital area
 - The smaller the diameter the offending object, the higher is the likelihood of occult injury → especially if under high velocity (gun, lawnmower, grinders, hammering against metal)

• CLINICAL FEATURES:

- o Patient may have eye pain but no change in visual acuity
- Whenever globe rupture is suspected, cover the eye with a metal eye shield and consult ophthalmology immediately
- Examination → decreased VA, irregular or tear-drop-shaped pupil, an afferent pupillary defect, hyphaema, positive Seidel test and lens dislocation

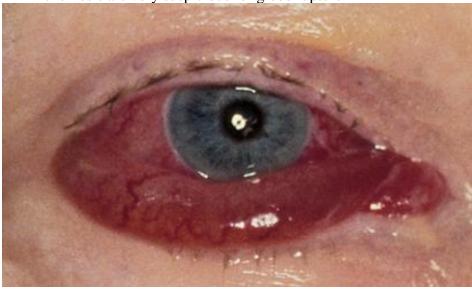


Uveal prolapse with teardrop pupil



Corneal laceration with positive Seidel's, small hyphaema.

 Large subconctival haemorrhage involving entire sclera or haemorrhagic chemosis are very suspicious for globe rupture



Haemorrhagic chemosis

- NEVER MEASURE INTRAOCULAR PRESSURE
- UNFORTUNATELY, THE EXAMINATION MAY BE NEARLY NORMAL AFTER GLOBE RUPTURE FROM A TINY HIGH-SPEED PROJECTILE
- DIAGNOSIS AND TREATMENT:
 - Diagnosis is made based on a combination of history, physical exam and CT scanning
 - o Once globe rupture is suspected, do NOT MANIPULATE THE EYE
 - o Elevate the bed and place a protective metal eye shield
 - o Broad spectrum IV antibiotics and ADT as appropriate
 - o OPHTHALMOLOGY URGENTLY

RETROBULBAR HAEMATOMA:

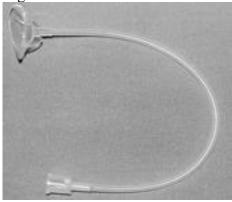
- Severe blunt trauma to the orbit can occasionally cause a retrobulbar haematoma due to an abrupt increase in intraocular pressure
- The conscious patient will complain of pain, proptosis and decreasing vision
- CT scan
- Measure intraocular pressure **→** if above 40mmHg, emergency lateral canthotomy indicated

CHEMICAL OCULAR INJURY:

- CHEMICAL BURNS TO THE EYE ARE TRUE OCULAR EMERGENCIES
 → complications include scarring of the cornea with permanent loss of vision and loss of the eye due to corneal perforation
- IMMEDIATE IRRIGATION WITH 1-2L NORMAL SALINE

ALKALI AND ACID INJURIES:

- Alkali injuries occur more frequently and tend to be more serious as they cause LIQUEFACTIVE NECROSIS, characterised by denaturing of proteins and saponification and thus DEEP PENETRATION INTO THE EYE
 - Acids cause COAGULATION NECROSIS → acts as a barrier to further penetration
- Immediate treatment should begin at the scene and include thorough irrigation until the pH is <7.4
 - o Can use a Morgan lens



 Severe chemical burns can cause scleral whitening, secondary to ischaemia and blood vessel injury



- Alkaline substance with pH<12 or acidic substances with pH >2 are though not to cause serious injury, but duration of exposure can increase severity
- Obtain ophthalmology consult for all but minor burns

CYANOACRYLATE (SUPERGLUE):

- Can cause adherence of the lids but there is rarely permanent damage to the eye
- To remove → instill generous amounts of chlorsig ointment on the eyelids to moisten, lubricate and provide antibiotic cover

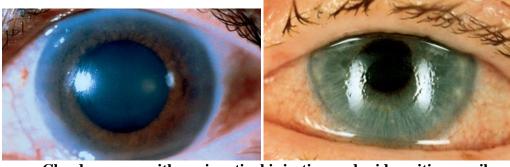
ACUTE AND PAINFUL VISION REDUCTION OR LOSS

ACUTE ANGLE-CLOSURE GLAUCOMA:

- PATHOPHYSIOLOGY:
 - Glaucoma represents a group of ocular disorders characterised by increased intraocular pressure causing optic neuropathy and vision loss if left untreated
 - o Intraocular pressure increases because the outflow of aqueous humeur is impaired due to a variety of mechanisms
 - Obstruction to aqueous outflow is the basic underlying problemn in glaucoma
 - In acute angle-closure glaucoma, the lens or peripheral iris blocks the trabecular meshwork, obstructing outflow of the aqueous
 - An acute attack is usually precipitated by pupillary dilatation
 - Intraocular pressure eventually exceeds the capacity of the corneal pump, causing the cornea to become oedematous and less transparent, thus explaining the foggy vision or halos

• CLINICAL FEATURES

- o ABRUPT ONSET, PAINFUL AND MAY RESULT IN SEVERE VISUAL IMPAIRMENT IF NOT TREATED QUICKLY
- o Complaints → severe pain in the affected eye, frontal/supraorbital headache, blurred vision, nausea and vomiting
- o Examination → FIXED, MIDPOSITION PUPIL. Hazy cornea with conjunctival injection, rock-hard globe



Cloudy cornea with conjucatival injection and mid-position pupil

- Easy to misdiagnose as a symptom complex due to prominence of other symptoms
- TREATMENT:
 - TREATMENT IS LOWERING THE INTRAOCULAR PRESSURE BY BLOCKING PRODUCTION OF AQUEOUS HUMEUR

pilocarpine 4% eye drops, 1 drop every 5 minutes for first hour [Note 1]

PLUS

- 1 acetazolamide 500 mg orally or IV [Note 2], immediately, and 250 mg orally, every 6 hours until surgery [Note 3]
 OR
- 1 mannitol 20% 5 to 10 mL/kg (= 1 to 2 g/kg) IV over 30 minutes
 OR
- 2 glycerol liquid 100 mL orally, with crushed ice

PLUS

- 1 betaxolol 0.25% eye drops, 1 drop every 2 hours for the first 6 hours
 OR
- 1 levobunolol 0.25% eye drops, 1 drop every 2 hours for the first 6 hours [Note 4] OR
- 1 timolol 0.5% eye drops, 1 drop every 2 hours for the first 6 hours

PLUS

- 1 brimonidine 0.2% eye drops, 1 drop every 2 hours for the first 6 hours
- 1 apraclonidine 0.5% eye drops, 1 drop every 2 hours for the first 6 hours

PLUS

- 1 bimatoprost eye drops 0.03%, 1 drop every 2 hours for the first 6 hours
 OR
- 1 latanoprost eye drops 0.005%, 1 drop every 2 hours for the first 6 hours
 OR
- 1 travoprost eye drops 0.004%, 1 drop every 2 hours for the first 6 hours.

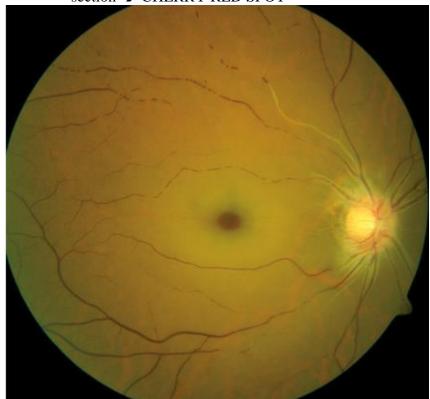
Laser or surgical iridectomy would then be performed as soon as possible.

PAINLESS REDUCTION IN VISION OR VISUAL LOSS:

CENTRAL RETINAL ARTERY OCCLUSION:

PATHOPHYSIOLOGY:

- The first branch off the internal carotid artery is the ophthalmic artery, which supplies the central retinal artery, which in turn supplies the inner retina
 - If the central retinal artery becomes occluded, the inner retina will infarct and become pale, less transparent and oedematous
 - The macula is the thinnest portion of the retina and the intact underlying choroidal circulation remains visible through this section → CHERRY RED SPOT

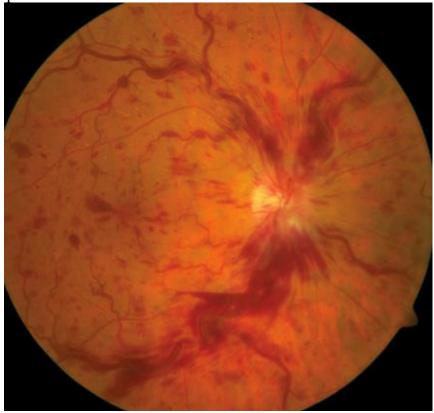


- Causes include → embolus of platelet aggregates, thrombosis, thrombosis, GCA, vasculitis, sickle cell, trauma
- CLINICAL FEATURES:
 - o Sudden, profound, painless monocular visual loss is characteristic
 - o Often preceded by episodes of AMAUROSIS FUGAX
 - o Exam will reveal afferent pupillary defect
 - o Pale retina and cherry red macula
 - o RARE
 - No firm treatment guidelines due to rarity

CENTRAL RETINAL VEIN OCCLUSION:

- Thrombosis of the central retinal veins causes retinal venous stasis, oedema and haemorrhage
 - o RF include DM, HT, CVA, CV disease
- Loss of vision is variable, ranging from vague blurring to rapid, painless and monocular visual loss
- BLOOD AND THUNDER FUNDUS → diffuse retinal haemorrhage and oedema

• No specific treatment



FLASHING LIGHTS AND FLOATERS (RETINAL DETACHMENTS)

- New-onset flashing lights or floaters, if monocular (binocular symptoms are almost intracranial in origin i.e. migraines) make one consider retinal detachment
- Flashes of light, floater or a a dark veil or curtain in the field of vision and decreased visual acuity require dilated indirect ophthalmoscopic evaluation by an ophthalmologist within 24 hours

GIANT CELL ARTERITIS (TEMPORAL ARTERITIS):

- GCA is a systemic vasculitis involving medium-sized arteries in the carotid circulation
- GCA can cause a painless ischaemic optic neuropathy with devastating visual consequences and rapid contralateral involvement if not diagnosed and treated promptly
- Patients are generally >50 with history of PMR
- Symptoms headache (temporal), jaw claudication, myalgias, fatigue, fever, anorexia and scalp tenderness
- Can develop rapid and profound vision loss
- Elevated CRP and ESR (>70, often above 100)
- Treatment consists of high-dose oral or IV steroids.
 - DO NOT DELAY STEROIDS WHILE WAITING FOR BIOPSY TO BE PERFORMED → biopsies will still be positive a week after initiation of steroid therapy

BELL'S PALSY:

- Dysfunction of peripheral cranial nerve VII, commonly of viral origin
 - o It is a palsy of the ipsilateral upper and lower face
- The orbicularis muscles are involved, resulting in incomplete closure of the eyelids on the affected side and leading to corneal exposure keratitis
- ANTIVIRALS PROVIDE NO BENEFIT
- CORTICOSTEROIDS CONFER TREATMENT BENEFIT

Recent randomised trials and a Cochrane review support the use of prednis(oi)one, which should be initiated within 48 hours of onset. Use:

prednis(ol)one 1 mg/kg (up to 100 mg) orally, daily in the morning for 5 days.

GENU VII BELL'S PALSY:

- o A CVA masquerading as a peripheral seventh nerve palsy
- o It is a stroke involving CN VI and the ipsilateral cranial nerve VII as it "genuflects" around the sixth nerve nucleus
- o This results in a cranial nerve VII palsy identical to a Bell's palsy, with the added finding of the inability to abduct the ipsilateral eye

POSTERIOR COMMUNICATING ARTERY ANEURYSM:

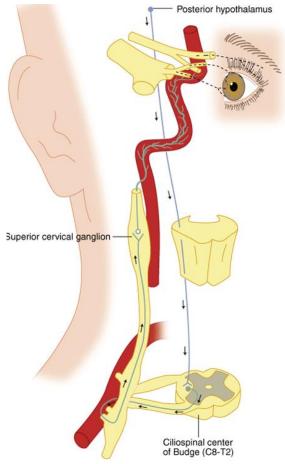
- Acute cranial nerve II palsy with ipsilateral pupillary dilation is a POSTERIOR COMMUNICATING ANEURYSM UNTIL PROVEN OTHERWISE
- Expansion of an aneurysm of the PCOM artery frequently causes compression of the outer fibres of cranial nerve III → dilation on the affected side

HORNER SYNDROME:

 Physical finding of ipsilateral ptosis, miosis and anhydrosis are characteristic of Horner syndrome



• Interruption of the sympathetic supply to the eye can occur anywhere along the pathway from the brainstem to the sympathetic plexus surrounding the carotid artery



- ED evaluation includes CXR< CT brain and cervical region and CTA for carotid dissection.
- Differential diagnosis includes → CVA, tumours, internal carotid artery dissection, herpes zoster, trauma. In kids, think neuroblastoma, lymphoma and metastasis

PAPILLOEDEMA:

- Bilateral oedema of the head of the optic nerve due to increased ICP → common finding in malignant HT, pseudotumour cerebri, intracranial tumours and hydrocephalus
- Any disease process that increases the ICP and thereby inhibits vascular or axoplasmic flow in the optic nerve causes congestion and oedema of the nerve head
- Disk margins are blurred, the cup is diminished and the nerve head is elevated with vascular congestion

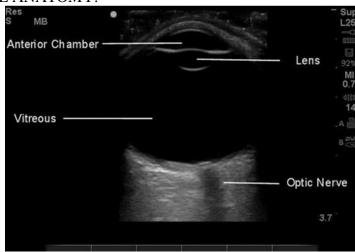


PSEUDOTUMOUR CEREBRI (BENIGN INTRACRANIAL HYPERTENSION):

- Increased ICP, papilloedema and normal CSF with normal CT/MRI charcterise this condition
- Visual field defects
- Consult neurosurgery

OCULAR ULTRASOUND:

- Very useful in diagnosis of a variety of conditions
 - o RETINAL DETACHMENT
 - o RETROBULBAR HAEMATOMA
 - o GLOBE PERFORATION
 - o LENS DISLOCATION
 - o VITREOUS HAEMORRHAGE
 - INTRAOUCLAR FOREIGN BODY
- NORMAL ANATOMY:



• RETINAL DETACHMENT → HYPERECHOIC MEMBRANE IN THE POSTERIOR ASPECT OF THE GLOBE



• FOREIGN BODY → BRIGHT REVERBERATION ARTIFACT



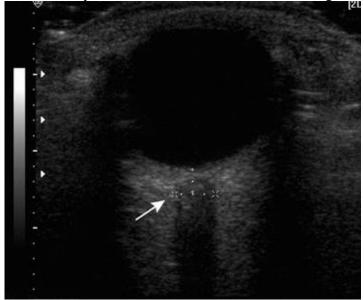
• EXTENSIVE GLOBE RUPTURE → ABNORMAL IRREGULAR SHAPE OF THE EYE WITH OCULAR CONTENTS DISPLACED



 VITREOUS HAEMORRHAGE → BRIGHT ECHOES IN THE POSTERIOR CHAMBER, WITH "SOCKS IN THE WASHING MACHINE" APPEARANCE ON EYE MOVEMENT



• RAISED INTRACRANIAL PRESSURE AND MEASUREMENT OF OPTIC NERVE SHEATH DIAMETER → normal measurements <5mm in adults, <4.5mm in children and <4mm in infants. The ONSD is measured 3mm posterior to the globe fro both eyes and three measurements are averaged



• CAUTION WITH OCULAR ULTRASOUND → if globe rupture is suspected, apply copious amounts of gel so that the transducer doesn't actually have to touch the eyelid as any pressure on the eye could be detrimental