Triaging Ocular Urgencies and Emergencies

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What Classifies an Emergency?

• Any condition in which the patient has:
  – the potential for visual loss,
  – currently experiencing vision loss,
  – permanent structural damage,
  – pain or discomfort, or
  – is an “emergency” for the patient.

• It is important to be able to triage a walk-in patient and, more importantly, a call-in patient.
## What questions to ask?

<table>
<thead>
<tr>
<th>Onset</th>
<th>suddenly noticed or sudden onset?</th>
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<tbody>
<tr>
<td><strong>Visual Loss</strong></td>
<td>any loss of vision?</td>
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<tr>
<td></td>
<td>loss vs. blurry vision</td>
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<tr>
<td></td>
<td>one eye or both</td>
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<tr>
<td></td>
<td>part of visual field or all</td>
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<tr>
<td></td>
<td>transient vs. permanent</td>
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<tr>
<td><strong>Pain</strong></td>
<td>is there pain? constant? scale (1-10)</td>
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<tr>
<td><strong>Redness</strong></td>
<td>is there any redness? location?</td>
</tr>
<tr>
<td><strong>Associated Factors</strong></td>
<td>contact lens wear? trauma?</td>
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<tr>
<td></td>
<td>discharge?</td>
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<td>photophobilia? medical history (eg. DM)</td>
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</table>
Common Types of Ocular Emergencies

- Vision Loss:
  - Gradual vs. sudden onset
  - Vision loss with or without pain
- Trauma
- Red eyes
Visual Loss

• Visual loss varies greatly in meaning from Pt to Pt
  – ranging from blur to complete blindness and may affect one or both eyes

• Components include:
  – acuity,
  – visual field,
  – color and brightness may be affected jointly or separately

• Detailed history and extent of vision loss crucial
Profound Loss of Vision

- Referring to a complete or greatly diminished vision affecting the whole field
- Common causes of severe vision loss:

<table>
<thead>
<tr>
<th>Vascular</th>
<th>central retinal vein occlusion, central retinal artery occlusion, vitreous heme</th>
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<tbody>
<tr>
<td>Inflammatory</td>
<td>optic neuritis</td>
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<tr>
<td>Infiltrative</td>
<td>optic neuropathy</td>
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<tr>
<td>Mechanical</td>
<td>retinal detachment</td>
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</table>
Monocular vs. Binocular

- Ocular or optic nerve pathology causes monocular vision loss
- Lesion at or posterior to chiasm causes binocular vision loss
  - VF defects become more congruous the further back in the visual pathway
  - Homonymous VF defects noted posterior to chiasm
- Difference between mono vs. bino usually straightforward, keeping the following in mind:
  - Patients occasionally mistake homonymous hemianopsia (similar loss of visual field in both eyes) for a monocular loss
Visual Defects
Monocular

- Differentiate between eyes that have lost all useful vision and those that have blurred vision
- Blurring of vision is not localized and may be caused by pathology anywhere from cornea to optic nerve
- Need to get anatomical diagnosis first before considering the cause
History

• Onset-gradual vs. sudden onset
• Gradual onset-
  – Cornea and lens-refraction, cataracts
  – Anterior chamber and vitreous-clouding (inflammation)
  – Retinal disease-macular edema
  – Optic nerve-swelling, pallor, atrophy
• Sudden onset-
  – e.g. vitreous heme and vascular occlusive disease
Refractive Error

- Unlikely to present acutely but patient may self refer if suddenly noticed decreased acuity
- Signs and symptoms (S&S) include monocular or binocular blurring of vision without distortion and visual acuity improves with pinhole.
Cataracts

- Most common non-refractive cause of visual impairment
- S&S include misting/blurring of vision, glare, change in refractive error (typically myopic shifts)
- Most common are age related (though congenital, metabolic and traumatic possible)
- The decreased acuity must correlate with the severity of the cataract...
  - ie if cataract doesn’t correlate with the amount of vision loss (or afferent pupillary defect present) then you need to find another reason for the vision (or other test results)
Flashes and Floaters

- Patients often present complaining of “spots” or “cobwebs” in front of their eyes.
- Causes of floaters include: posterior vitreous detachment (PVD), retinal tear, vitreous heme, uveitis.
- Since PVD and retinal tears present the same way, a RT has to be eliminated.
- Ask the patient whether spots move with eye and continue to move after the eye has stopped.
- Large spots could be blood clots.
Flashes and Floaters

- Sudden onset typically means a PVD, retinal tear or heme
- If the spots appear after flashing light, then retinal tear must be eliminated
- Myopes tend to have floaters and will notice them for a long time
- Key is to rule out potentially sight threatening condition for the floaters, ie retinal tear.
- Patients with retinal condition such as lattice degeneration and myopes need to be educated about S&S of RD (flashed and floaters)
Red Eyes

- Red eyes are most common reason patient seeks emergent care
- Quite often red eye has accompanying symptomatology
  - eg. pain, soreness, decreased VA
- Conditions giving rise to redness can be broadly classified anatomically
Red Eye Exam

- Case history is crucial!
  - Key symptoms such as pain, decreased VA, photophobia and discharge
- Discharge?
  - mucopurulent or watery
- Duration?
  - acute or chronic
- Previous history?
  - Is this a recurrence of a previous condition eg iritis?
- Extraocular symptoms?
  - Is this a manifestation of a systemic condition or conversely is the severity of the condition causing systemic upsets (nausea and vomiting)
Pattern of Redness

- Examples:
  - Maximal redness in the visible white part in the corners-
    - conjunctivitis
  - Segmental-
    - episcleritis
  - Limbal (around the colored part)-
    - iritis, glaucoma
  - Brawny red-
    - scleritis
  - Interpalpebral (between the lids)-
    - dry eyes
  - Deep crimson red-
    - subconjunctival heme
Case

• 27 year old pharmacy student presents to the clinic on emergent basis
  – complains about red/painful eyes for the past 2 days
  – started OD then transferred to OS
  – reports a watery discharge, no itching, and is not a contact lens wearer
  – reports that others in his class have had a similar red eye
  – no seasonal, food or drug allergies
  – has taken Visine 4-5 times/day since eyes became red but hasn’t helped much
Conjunctivitis

Bacterial Conjunctivitis

Allergic Conjunctivitis

Viral Conjunctivitis

Blepharo-conjunctivitis
Viral Conjunctivitis

• Most common infectious keratitis presenting on emergent basis
• 62% caused by adenovirus
• Two major types:
  – Pharyngoconjunctival fever
  – Epidemic keratoconjunctivitis
Viral Conjunctivitis

- PCF: history of recent/current upper respiratory infection
- EKC: highly contagious with a history of coming in contact with someone having a red eye.
  - Adenovirus 8 common variant leading to “rule of 8’s”
    - First 8 days red eye with fine SPK
    - Next 8 days deeper focal epithelial lesions
    - Following 8 potential development of infiltrates
    - Resolution
- RPS AdenoPlus available to use for adenoviral confirmation.
AdenoPlus

• Have you heard about this?
Interpreting the Results

NEGATIVE RESULT
• Only a **BLUE** line appears in the control zone.
  – A negative result is indicative of an absence of Adenovirus Antigens.

POSITIVE RESULT
• The presence of both a **BLUE** line in the control zone and a **RED** line in the result zone indicates a positive result.
• Even if the **RED** line is faint in color, incomplete over the width of the test strip, or uneven in color, it must be interpreted as positive.
• A positive result indicates the presence of Adenovirus antigens.
Interpreting the results

Invalid Result

• If a **BLUE** line does not appear, the test may be invalid.
  – Reimmerse the absorbent tip into the buffer vial for an additional 10 seconds.
  – If a **BLUE** line still does not appear after 10 minutes, the test must be discarded and the subject retested by resampling the eye using a new AdenoPlus test kit.
Patient has “Red Eye”

Front Office IDs & Isolate the “Red Eye”

Patient is taken to “Red Eye Room”

“Red Eye” Patient history & work up

Tech performs AdenoPlus™ test to rule out Adenovirus

Dr. starts clinical evaluation with Adenoviral conjunctivitis confirmed, or rule out

Dr. proceeds with evidence based treatment
History Signs Symptoms

- Pink eye exposure, spread from one eye to the other, recent upper respiratory symptoms
- Itching, burning, foreign body sensation, tearing, discharge, eyelash matting
- Pre-auricular adenopathy, chemosis
- No significant pain, light sensitivity, or visual loss

AdenoPlus

POSITIVE

- Education: hygiene and hand washing
- Supportive care: artificial tears, cool compresses and antihistamines
- Antiviral medication
- No antibiotics

NEGATIVE

- Consider topical antibiotics or antihistamines
Viral Conjunctivitis: Signs and Symptoms

- Gritty sensation
- Watery discharge
- Sticky in mornings
- Follicular response
- Chemosis
- Injection
- SPK
- Infiltrates possible
- Positive lymph nodes

- Pseudomembranes in severe cases
- Subconjunctival hemes
Management

• Consider the use of anti-inflammatory treatment to relieve patient symptoms and improve comfort
  – FML QID OU
  – Lotemax QID OU
    – (there is a new Lotemax gel that has a higher viscosity and increases contact time)
• EKC patients are typically very uncomfortable and would benefit from anti-inflammatory treatment
  – especially if infiltrates or pseudomembrane present
Management

• Betadine (Melton-Thomas Protocol):
  – Proparacaine
  – 4-5 drops of Betadine 5%
    • Get patient to close eye and gently roll them around
    – After one minute, lavage the eye
    – Lotemax 4 times a day for 4 days
• Alternative: Betadine swabsticks.
  – 5% Betadine solution only comes in 30 ml bottles cost $14.00.
  – Case of 200 Betadine swabsticks apprx. 45 dollars.
Management

- Antivirals used in HSV keratitis are ineffective in treatment of viral conjunctivitis
  - New Update: in conversation with several colleagues, Zirgan 4-5 times/day has shown significant improvement in patients over a 7-10 time period.
- Important to stress limited contact with others, frequent hand washing, not sharing of towels, etc.
Bacterial Conjunctivitis
Signs and Symptoms of Bacterial Conjunctivitis

Clinical presentation – uni-/bi-lateral

**Signs:**
- Bulbar conjunctival injection
- Purulent discharge
- Morning matting of eyelashes

**Symptoms:**
- Photophobia
- Blurred vision
- Tearing

- Hyperemia
- Chemosis
- Purulent discharge
Treatment/Management

- Topical antibiotic therapy
  - Vigamox TID for 7-10 days
  - Moxeza BID for 7 days
    - Category C
  - Zymaxid q 2 hours for Day 1, then BID-QID Days 2-7
    - Category C
  - Azasite BID for 2 days then qd for next 7-10 days
    - Category B
  - Besivance TID for 7-10 days
    - Category C
  - Tobramycin/Gentamicin QID for 7-10 days
    - Category B
  - Polytrim q3hrs (max 6x/day) for 7-10 days
    - approved to age of 2 months
    - Category C
Allergic Conjunctivitis

- Allergic Conjunctivitis
- Dry Eye
- Blepharitis
- Bacterial Conjunctivitis
Prevalence of Allergic Conjunctivitis

- Allergies affect as many as 40 to 50 million Americans
- Incidence and prevalence of allergic conjunctivitis has been rising over the last 40 years
Seasonal Allergic Conjunctivitis (SAC)

- Occurs during peak allergy seasons: (spring & fall)
- Primarily caused by outdoor allergens – pollen (ragweed, mountain cedar), grasses
- Produces hallmark signs and symptoms such as itching, redness, chemosis, tearing and lid swelling
Perennial Allergic Conjunctivitis (PAC)

- Milder than SAC
- Occurs year round
- Primarily an indoor disease
  - Environmental controls can be effective
- Can become more severe with higher pollen counts
Hallmark Signs and Symptoms

- Hyperemia
- Chemosis
- Lid edema
- Tearing
Signs and Symptoms of Allergic Conjunctivitis

Clinical presentation – bilateral

**Signs:**
- Conjunctival edema
- Conjunctival hyperemia
- Chemosis
- Lid edema
- Watery discharge

**Symptoms:**
- Itching
- Burning
- Photophobia
- Foreign body sensation
- Blurred vision
Mast Cell Cascade
Treatment

• Ocular allergy sufferers need;
  – fast relief of signs and symptoms,
  – long-lasting therapeutic effects,
  – comfortable and safe topical drugs,
  – convenient treatment regimen

• Therapeutic focus is mostly confined to the suppression of mast cells, their degranulation and the effects of histamine and other mast-cell derived mediators.
Treatment of Ocular Allergy

Medications:
• Topical OTC drops
• Oral antihistamines (prescription and OTC)
• Topical NSAID drops
• Topical antihistamines
• Topical mast cell stabilizers
• Topical steroid drops
• Topical dual-action drugs (antihistamine/mast cell stabilizers)
# Ocular Allergy Medication Options

<table>
<thead>
<tr>
<th>Medication Type</th>
<th>Medication Names</th>
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<tbody>
<tr>
<td>Tetrahydrazoline HCl</td>
<td>VISINE*, MURINE* Plus</td>
</tr>
<tr>
<td>Naphazoline HCl</td>
<td>NAPHCONE® eye drops, VASOCON*</td>
</tr>
<tr>
<td>Phenylephrine HCl</td>
<td>PREFRIN*</td>
</tr>
<tr>
<td>Oxymetazoline HCl</td>
<td>VISINE L.R.*</td>
</tr>
<tr>
<td>Naphazoline/Antazoline</td>
<td>VASOCON*-A</td>
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<tr>
<td>Naphazoline/Pheniramine</td>
<td>NAPHCONE® eye drops</td>
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<tr>
<td>Ketorolac</td>
<td>ACULAR*</td>
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<tr>
<td>Suprofen</td>
<td>PROFENAL® solution</td>
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<tr>
<td>Diclofenac</td>
<td>VOLTAREN*</td>
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<tr>
<td>Levocabastine</td>
<td>LIVOSTIN*</td>
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<tr>
<td>Emedastine</td>
<td>EMADINE® solution</td>
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<tr>
<td>Loteprednol</td>
<td>ALREX*</td>
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<tr>
<td>Rimexolone</td>
<td>VEXOL** suspension</td>
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<tr>
<td>Cromolyn</td>
<td>CROLOM*, MAXICROM™ solution</td>
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<tr>
<td>Lodoxamide</td>
<td>ALOMIDE® solution</td>
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<tr>
<td>Nedocromol</td>
<td>ALOCRIL*, TILAVIST*</td>
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<tr>
<td>Pemirolast</td>
<td>ALAMAST*</td>
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<tr>
<td>Azelastine</td>
<td>OPTIVAR*, LASTIN*</td>
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<tr>
<td>Ketotifen</td>
<td>ZADITOR*, ALAWAY*, ZYRTEC, CLARITIN</td>
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<tr>
<td>Epinastine</td>
<td>ELESTAT*</td>
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<tr>
<td>Olopatadine</td>
<td>PATANOL® PATADAY</td>
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<tr>
<td>Pazeo</td>
<td>PATANOL® PATADAY</td>
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<tr>
<td>Bepotastine</td>
<td>BEPREVE</td>
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<tr>
<td>Alcaftadine</td>
<td>LASTACAFT</td>
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</table>

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**Vexol is a trademark of N.V. Organon
Subconjunctival Hemorrhage

- Subconjunctival heme:
  - typically happen spontaneously (usually after valsalva),
  - painless with possible mild discomfort,
  - blood red patch on eye.
  - Resolve on own in couple of weeks,
  - AT for comfort.
Corneal Ulcers

- Infective bacterial and fungal corneal lesions cause severe pain and loss of vision
- S and S:
  - Pain, photophobia, tearing
  - Mucopurulent discharge with generalized conjunctival injection
  - Decreased vision (esp if on visual axis)
  - Possible anterior chamber reaction and hypopyon
  - Dense infiltrate
  - Satellite lesions around main lesion may indicate fungal infection
Associated Factors

- Contact lens wear, especially soft and extended wear lens
- Recent history of corneal trauma
- Topical steroid use
- History of exposure to vegetative matter (fungal etiology)
Management

• Infective ulcers need to be cultured!
• If contact lens wearer, consider culture of contact lens
• Intensive topical antibiotic regimen,
  – e.g. loading dose of Vigamox/Zymar 2gtts q 15 min x 1 hour, 1gt q 30 min x 6 hours, 1 gt q 1 hr until f/u in 24 hours
  – Cycloplegic (e.g Homatropine 5% BID)
• Consider steroid after several days treatment with antibiotics to eliminate underlying infiltrate.
Marginal Corneal Ulcers

- Marginal corneal ulcers-
  - pain, photophobia,
  - watering,
  - tend to be recurrent,
  - peripherally discrete infiltrate and ulcer,
  - associated with eyelid margin disease (blepharitis).

- Treat as any ulcer,
- i.e. loading dose of Vigamox/Zymar and follow up in 24 hours. After 48-72 hours consider adding steroid (Pred Forte) QID to eliminate infiltrate.
Corneal Abrasion

- Commonest cause of injury is:
  - direct contact (e.g., poking in eye),
- followed by:
  - foreign body,
  - contact lens related and
  - no trauma recalled
Corneal Abrasion: Treatment

- Remove any loose or jagged tissue
- Topical antibiotic:
  - e.g. Viagmox, Zymar QID for prophylactic coverage
- Consider bandage (therapeutic) CL for comfort and healing:
  - e.g. N&D, PureVision, Oasys
- Pain management:
  - Homatropine qd-bid or
  - topical NSAIDs: Acular, Voltaren tid-qid
- Follow up in 1 day, educate on RCE
Case

• 20 year old male presents with a red painful eye
  – complains about red/painful right eye
  – Started that morning when he woke up
  – reports a watery discharge, no itching, and is not a contact lens wearer

• SLE:
  – See attached image with NaFl stain
Herpes Simplex Keratitis: Clinical Features

- Characterized by primary outbreak and subsequent reactivation
- Primary outbreak is typically mild or subclinical
- After primary infection, the virus becomes latent in the trigeminal ganglion or cornea
- Stress, UV radiation, and hormonal changes can reactivate the virus
- Lesions are common in the immunocompromised (i.e. recent organ transplant or HIV patients)
Herpes Simplex Keratitis

• Tx:
  – Viroptic (trifluridine) q 2h
    • then taper down for 10-14 days.
    • Viroptic is toxic to the cornea.
  – Zirgan available, use 5 times a day
• Oral acyclovir (2 g/day) has been reported to be as effective as topical antivirals without the toxicity
  – Valtrex (valcyclovir) 500 mg TID for 7-10 days
  – Famvir (famciclovir) 250 mg TID for 7-10 days
Herpes Simplex Keratitis

- Consider prophylaxis of 400 mg acyclovir BID for 1 year to decrease recurrence
  - Valtrex 500 mg qd

- If stomal keratitis present, after epi defect has healed, add Pred Forte QID until inflammation reduced and then slowly taper.
Dendritic ulceration before treatment with Zirgan

Cornea after treatment with Zirgan
Thermal Burns

- Typically eye is protected by the lids and blink
- Usually more injury to lids
- Thermal burns are classified the same as chemical burns
- Severe burns may result in necrosis of lower lid, cornea and sclera
  - maybe relatively painless due to extensive destruction of nerve endings
- Thermal injuries tend to penetrate deep
  - have to address deeper tissue inflammation and damage.
Superglue

- Pain, lids stuck together
- Fracture glue with forceps
- Treat like an abrasion
- May need to cut eyelashes to get lids open
Corneal Foreign Body

• Common work related injury
• Important to understand circumstance of injury (e.g. high speed projectile? Possible intraocular FB?)
• Metallic FB will leave rust ring
• Exam may have to be done with anaesthetic (to obtain VA’s, pupils, SLE)
• Evert upper lid to ensure no FB present
Painful Red Eyes with Loss of Vision

- **Causes:**
  - Acute congestive glaucoma
    - angle closure (blockage of drainage)
  - Severe Inflammation
    - severe acute anterior uveitis (inflammation of iris),
    - endophthalmitis (inner eye infection)
  - Central corneal lesions
    - keratitis (corneal inflammation),
    - corneal abscess (ulcer)
Acute Glaucoma

• Most are angle closure but can also be secondary (due to other eye diseases)
• Signs and symptoms:
  – Unilateral (typically)
  – HA, nausea, vomiting
  – Haloes around lights
  – Decreased VA
  – Edematous and cloudy cornea
  – Anterior chamber looks shallow and pupil is oval, fixed and mid-dilated
  – More common in hyperopes and elderly females
Acute Angle Closure - corneal edema and redness (circumlimbal)
Management of Angle Closure

- Untreated, can cause severe visual loss and eventual blindness
- Immediate treatment includes oral acetazolamide (Diamox)
- Topical glaucoma drops (any other glaucoma meds you have available!) q 15 min X 2 and then BID.
- Topical steroid
- Pilocarpine
- Serial tonometry (eye pressure checks) over next couple of hours, if no response consider use of hyperosmotic (eg oral glycerol)
- Definitive treatment is LPI (laser peripheral iridotomy) and performed in both eyes
Acute Anterior Uveitis

- **Signs and Symptoms:**
  - Unilateral (typically), pain, photophobia (sensitivity to light) and tearing
  - Redness maximal at limbus (ciliary flush)
  - VA affected to varying degree
  - Most cases are idiopathic, but may also be associated with systemic disease or infection (eg rheumatoid arthritis, herpes zoster)
Keratic Precipitates (KP)

[Image of Keratic Precipitates]
Hypopyon
Management

- Cycloplegic (dilation of the pupil)-
  - relief of pain,
  - prevention of synechiae, and
  - reduction of inflammation (eg. Homatropine 5% BID)

- Steroid treatment-
  - reduction of inflammation utilizing topical steroids (eg Pred Forte 1% q 1-4 hrs) and use of orals (e.g 40 mg/day with taper to 10-15 mg/day) if vitritis present

- Consider systemic evaluation if bilateral, granulomatous or recurrent.
Chemical Burns

- Chemical burns account for a small but significant fraction of ocular trauma.
- Majority of victims are young and exposure occurs at home, work and vehicle accidents (air bags)
- Damage dependent upon nature of chemical and how rapidly irrigation is started
  - Speed of initial irrigation has the greatest influence on the prognosis and outcome of eye burns
- Severity of ocular injury related to type of chemical, volume and pH of solution and the duration of exposure.
Alkali Chemical Burns

- Occur more frequently than acid injuries
- Tend to cause more severe injury than acids because the hydroxlion (OH) saponifies the fatty acids of cell membranes resulting in cell disruption and cell death. The alkali radical rapidly penetrates deeper into tissue of eye.
Alkali Chemical Burns

- Most common agents are:
  - Calcium hydroxide (lime) [plaster, mortar]
  - Potassium hydroxide [Nair]
  - Sodium hydroxide (lye) [Drano]
  - Ammonia [household cleaners]

- Penetration rate increases from calcium hydroxide (slowest) to ammonium hydroxide (fastest).

- Irreversible damage occurs at a pH above 11.5.
Alkali Chemical Burns

- Alkali exposure results in:
  - Loss of corneal and conjunctival epi, stromal keratocytes and endothelium
  - Loss of clarity is secondary to stromal hydration
  - Damage to the vascular endothelium of conjunctival and episcleral vessels
  - Intraocular structures such as iris, lens and ciliary body are rapidly damaged if alkali penetrates cornea.
Acidic Chemical Burns

- Epithelium provides effective barrier to weak acids. Stronger acids cause protein precipitation in epithelium and stroma which creates a barrier to further penetration.
- Very strong acids penetrate as quickly as alkalis
Clinical Classification of Chemical Burns

- Clinical course and prognosis correlates with:
  - extent of limbal ischemia,
  - extent of damage to conjunctival and episcleral tissue and
  - damage to intraocular structures.

- Eye burns are classified in 4 grades.
Chemical Burn Treatment

- Immediate irrigation is of paramount importance
- Most patients are disabled by severe blepharospasm and disorientation so require assistance away from harm and to initiate irrigation.
- Make sure to remove any solid particulate matter prior to beginning irrigation
- Minimum of 15 minutes constant irrigation (some recommend 30 minutes)
Chemical Burn Treatment

- Water is commonly recommended however it is hypotonic to corneal tissue and can result in increased water intake into the corneal and subsequent diffusion of corrosive materials deeper into cornea.
- Recommend fluids of higher osmolarity such as sterile lactated Ringers and balanced saline solution.
Vision Loss with no Pain: Vascular Abnormalities

- Diabetes/diabetic retinopathy
- Central/branch retinal vein occlusion
- Central/branch retinal artery occlusion
Diabetes

• Microvascular complications resulting in capillary closure & abnormal permeability
• S&S include blurring of vision (maculopathy and refractive error shifts), sudden drop in vision (vitreous heme),
• Fluctuations in sugar levels result in shifting refractive errors..
  – typically myopic shifts with increased blood sugar
• Untreated diabetic maculopathy and neovascularization are potentially sight threatening
Lenticular Changes-Dynamic
Vein Occlusion

- Associated with:
  - hypertension,
  - coronary artery disease,
  - DM and peripheral vascular disease.
- Usually seen in elderly patients (60-70), slight male and hyperopic predilection.
- Second most common vascular disease after diabetic retinopathy.
- BRVO: typically occurs at A/V crossing (sup/temp)
- CRVO: thrombus occurring at lamina
Vein Occlusion: Signs/Symptoms

- **BRVO:**
  - sudden, painless, visual field defect.
  - Patients may have normal vision.
  - Quadrantic VF defect,
  - dilated tortuous retinal veins with superficial hemes and CWS.
CRVO

- CRVO:
  - sudden, painless unilateral loss of vision.
  - Decreased vision ranging from near normal to hand motion with majority in 20/200 range.
  - Dilated tortuous vessels, with numerous superficial retinal hemes and CWS.
Artery Occlusion

- Primarily from:
  - embolism from cholesterol, calcifications, plaques.
- Usually occurs in elderly associated with
  - hypertension (67%),
  - carotid occlusive disease (25%),
  - DM (33%) and cardiac valvular disease.
- CRAO more common.
- Sudden loss of unilateral, painless vision (defect dependent upon location of occlusion).
BRAO

- BRAO typically temporal retinal bifurcations.
CRAO

- CRAO has profound vision loss with history of amaurosis fugax (transient vision loss).
- Vision is usually CF (count fingers) to LP (light perception) with positive APD.
- Diffuse retinal whitening with arteriole constriction, cherry red macula.
Ophthalmic Emergency

• Treatment is controversial due to poor prognosis and questionable benefit.
• Treat immediately before workup, if patient present within 24 hours of visual loss.
  – Digital ocular massage,
  – systemic acetozolamide (500 mg IV or po),
  – topical ocular hypertensive drops (Iopidine, B-blocker),
  – anterior chamber paracentesis,
  – consider admission to hospital for carbogen Tx (93% oxygen, 7% carbon dioxide)
Vision Loss Without Pain: TIA/TMB/Amaurosis Fugax

- Refers to temporary visual impairment of variable duration (seconds to hours)
  - TIA: transient ischemic attack—can be cerebral or retinal
  - TMB: transient monocular blindness secondary to a retinal TIA
  - Amaurosis Fugax: same as TMB
- Abrupt onset, progression to involve all or part of visual field, sight usually returns
- Within affected area, visual acuity maybe dimmed or completely lost
TIA’s

- Stroke is 3\textsuperscript{rd} leading cause of mortality in developed countries and most common cause of neurological disability

- 15-20\% of patients with stroke have a preceding TIA, though guidelines for referral and evaluation are debated
  
  - Traditional guidelines suggested that assessment should be complete within 1 week of TIA
TIA’s

- Risk of stroke after TIA has traditionally been considered relatively low, but
  - new studies indicate that the risk is much higher than previously thought and the time window for prevention is short.
- Effective secondary prevention depends on reliable identification of those at high risk and targeting treatment.
TIA’s: High Risk Factors

- Five (5) risk factors are associated with a high risk (30%) of recurrent stroke at 3 months:
  - Age over 60
  - Symptom duration greater than 10 minutes
  - Motor weakness
  - Speech impairment
  - Diabetes

- Isolated sensory or visual symptoms were associated with low risk of stroke!
TIA: Early Treatment

- Several treatments are likely to be effective in preventing stroke in the acute phase after a TIA:
  - Aspirin
  - Anticoagulants
  - Statins
  - Endarterectomy (for >50% carotid stenosis)
- Further research needed for:
  - Lowering blood pressure acutely after TIA
  - Prophylactic use of neuroprotective drugs
Amaurosis Fugax: TMB

- Most common cause is:
  - thromboembolic disease (e.g., carotid artery disease throwing emboli) or
  - vasospasm
- Described as “curtain falling over vision”
- Risk of stroke or death is about 3-5%,
  - which is significantly lower than for a cerebral TIA (15-20%)
- Px still require work-up to determine cause:
  - e.g., carotid doppler
Trauma

- Major cause of visual impairment
- Unilateral vision loss secondary to trauma common
- Children and young adults most at risk with males>females
- Detailed history is important outlining specifics of the trauma

Classification:
- Non-penetrating: blunt trauma, surface injury
- Penetrating: no foreign body(fb) and retained fb
- Burns: chemical, thermal, radiation
Airbags

- Airbags have decreased the incidence of severe morbidity and mortality related to motor vehicle crashes
- Can cause serious eye injuries and significant visual morbidity that vary from mild to severe
Airbag Ocular Injuries

- Eyelid lacerations
- Periorbital fractures
- Corneal abrasions
- Hyphemas
- Lens dislocations
- Angle recessions

- Corneal-scleral lacerations
- Alkaline corneal burns
- Cyclodialysis
- Retinal detachments
- Retinal hemorrhages
- Vitreous hemorrhages
- Traumatic macular holes
Airbag Ocular Injuries

- Bilateral eye injuries are common
- Have to assess protection and possible trauma secondary to eyeglasses
- Most common injuries include:
  - Corneal abrasions
  - Eyelid trauma
  - Hyphemas
- Vision threatening secondary to:
  - RD, scleral rupture, lens dislocation
Blunt Trauma

- Causes injury by distorting the globe and abrading tissue
- A blow to the cornea causes compression of the globe and a corresponding stretching of ocular and orbital tissues
- S&S include:
  - pain and tearing if cornea involved
  - Decreased VA if central cornea
  - Subconjunctival heme
  - TIA possible
  - Aching pain-iritis
Blow Out Fracture

- Medial wall and floor commonly affected
- Pain in direction of action of muscle, bruised or entrapped
- Diplopia (double vision) with limitation of muscle movement
- Numbness, crepitus, enophthalamos
- If hyphema is present, force of injury maybe sufficient to cause fracture
- May cause prolapse of muscle or surrounding fibrous septa which tethers inferior or medial rectus causing diplopia and restricted movement
- Requires x-ray, antibiotics (oral augmentin 250-500 mg po tid for 10 days) and possible reconstruction
Exophthalmometry
Normal Anatomy

- levator palpebrae superioris
- lacrimal gland
- superior rectus
- superior oblique
- lateral rectus
- globe
- medial rectus
- inferior oblique
- inferior rectus
- connective tissue
- maxillary sinus

Blow-Out Fracture