Common Urgencies and Emergencies

1. Trauma:
   1. Chemical burns
   2. Open globe trauma
2. Infectious:
   1. Endophthalmitis
   2. Orbital cellulitis
3. Neurological:
   1. Acute third nerve palsy (rule out intracranial aneurysm)
4. Glaucomatous:
   1. Angle closure glaucoma
5. Retinal:
   1. Macula on rhegmatogenous retinal detachment
6. Vascular:
   1. Central retinal artery occlusion
   2. Ischemic optic neuropathy

Case Presentation

- Splash of drano in right eye about 15 min ago.
  - Started washing eye since then
- Gross examination:
  - Cornea: sloughing of epithelium, edema
  - Scattered limbal ischemia
- Why just a gross examination at this point?
Chemical Burns

- Range from mild epithelial defects to complete opacification and destruction of tissue
- Alkali injuries are typically more severe than acid injuries
- Incidence: 7.7-18% of all ocular traumas
  - > young males and > at work
  - The severity of the chemical injury depends on the chemical substance
  - pH
  - Toxicity of the chemical (degree of penetration and cell injury)
  - Duration of contact

Alkali Burns

- Cause saponification of the fatty acids in cell membrane.
- Subsequently, destruction of the proteoglycan ground substance and collagen fibers of the stromal matrix.
- Stronger alkali substances may penetrate into the anterior chamber in 15 minutes.
  - Secondary conditions: glaucoma, cataracts, and uveitis.

Acid Burns

- Limited to superficial layers of the cornea causing protein coagulation.
- Coagulated proteins can act as a barrier to further acid penetration
- Hydrogen molecules damage the ocular surface by altering the pH.
- Anions cause protein denaturation, precipitation, and coagulation

Classification

- Modified Hughes/Roper Hall classification
  - Based on the degree of corneal involvement
  - Limbal ischemia
- Dua classification
  - Limbal involvement (in clock hours)
  - Percentage of conjunctival involvement
Chemical Burns

**Immediate Management:**
- Copious irrigation with sterile isotonic solution until pH is neutral.
- Tap water, although hypotonic, is an appropriate substitute.
- Instill topical anesthetic (proparacaine or lidocaine 4%) to facilitate irrigation and relieve pain.
- Irrigate under eyelids to remove chemical residue completely.
- In cases of acid burns, debridement of devitalized corneal epithelium should be done.

**Medical Management:**

**Inflammation:**
- Steroids: Pred Forte every 1hr or Durezol every 2hrs then taper
- Recommended only for the acute phase
- Usage should be guarded and followed closely

**Infection:**
- Broad spectrum antibiotic

**Pain:**
- Topical anesthetic or preferred anesthetics (e.g., proparacaine, lidocaine) and follow with topical corticosteroids

**Bandage contact lens**
- Amniotic membrane

**Surgical therapy**
- Limbal stem cell transplantation
- Corneal transplantation

Case Presentation

**Presentation:**
- 102F fever
- Painful eye movement, OS
- MHx: Previous sinusitis infection
- Restricted ocular motility, OS
- BCVA: 20/20 OD; 20/50 OS
- Chemosis
- Proptosis
Orbital Cellulitis

Life threatening infection of the orbital soft tissues behind the orbital septum

Pathogenesis:
- Extension from periorbital structures
  - Paranasal sinus
  - Face and eyelids
  - Lacrimal sac (dacryocystitis)
  - Teeth (dental infection)
  - Extension of pre-septal cellulitis
- Exogenous causes:
  - Trauma - within 72 hours of injury that penetrates the septum
  - Surgery
- Endogenous causes:
  - Bacteremia with septic embolization
  - Intrabulbar causes:
    - Endophthalmitis
    - Dacryoadenitis
- Sinusitis:
  - H. influenzae (children)
  - S. pneumoniae
  - S. aureus
  - Other streptococci
  - Moraxella catarhalis
  - Anaerobic
  - Post trauma/post-surgical:
    - S. aureus
    - Anaerobic

Presentation:
- Fever (102°F or greater)
- Painful eye movement
- Restricted ocular motility
- Decrease in vision
- Proptosis/globe displacement
- Raised intraocular pressure
- Eyelid edema (absence of eyelid crease)
- Conjunctival chemosis
- Severe malaise and headache
Orbital Cellulitis

**Complications:**
- Subperiosteal/orbital abscess
- ONH damage and blindness
- Cavernous sinus thrombosis
- Meningitis
- Septicemia (blood infection)
- Death

- Prognosis depends on the organism and the extent of disease at the time of presentation

---

Case Presentation

- 65 yo Asian female
- Ocular pain
- Blurred vision: in one eye
- Halos around lights
- With nausea and one episode of vomiting
- Headache

- BCVA: 20/20 OD, 20/50 OS
- IOP: 20 mmHg/63 mmHg
- Mid-dilated OS pupil
- Closed angles, OS; narrowed angle, OD

---

Acute Angle Closure Glaucoma

- Characterized by a rapid and large increase in the intraocular pressure (IOP), resulting from a sudden blockage of the trabecular meshwork by the iris.

**Pathophysiology:**
- Pupillary block
- Excessive iris-lens apposition impedes the flow of aqueous from PC to AC, elevating PC aqueous pressure
- Secondary forward bowing of peripheral iris results in occlusion of the TM
- Alternative mechanisms: Plateau iris, ciliary block.
Acute Angle Closure Glaucoma

Risk Factors:
- Age > 40 yo (lens thickness, cataracts)
- Female gender (2x > male gender)
- Family history (first degree relatives)
- Hyperopic individuals
- Anterior chamber depth < 2.5mm
- Race: Inuit 5% > Asian 1.4% > White 0.6% > Black 0.2%
- Certain medications e.g. Topamax

Acute Angle Closure Glaucoma

Acute Symptoms:
- Ocular pain
- Blurred vision
- Haloes around lights
- Nausea and vomiting
- Headache (frontal or supraorbital)

Acute Signs:
- Elevated IOP
- Closed/narrow angle
- Corneal edema
- Conjunctival injection
- Mid-dilated sluggish moving and irregularly shaped pupil
- Iris bombe (typically)
- Glaucometeker and sectoral iris atrophy – indicators of previous episodes of AACG
- Optic nerve head damage
Acute Angle Closure Glaucoma

Tests:
- Gonioscopy
  - To examine the iridocorneal angle
  - R/O other causes:
    - Glaucomatocyclitic crisis, NV glaucoma, malignant glaucoma, angle mass, iridocorneal endothelial syndrome (ICE)
- Dynamic gonioscopy
  - Reversible (appositional closure)
  - Irreversible (synechial closure).
- A scan, ultrasound biomicroscopy, anterior segment OCT

Acute Angle Closure Glaucoma

Treatment:
- Topical drops:
  - Beta adrenergic antagonists
  - Alpha2 adrenergic agonists
  - CAI: topical and oral.
  - Mistica 1-2% Pilocarpine after IOP starts to normalize
  - Hypersmotics
  - Topical corticosteroids
- Miotics 1-2% Pilocarpine after IOP starts to normalize
- Globe compression and dynamic gonioscopy

Acute Angle Closure Glaucoma

Surgical Treatment:
- Laser peripheral iridotomy (LPI)
- If laser iridotomy cannot be performed:
  - Surgical iridectomy
  - Peripheral iris may be flattened with a laser iridoplasty
  - Pupillary block may be relieved with a laser pupilloplasty.

The fellow eye shares the anatomic predisposition for increased pupillary block:
- Recommended to do a peripheral iridotomy.
- 50% chance of developing an acute attack in the untreated fellow eye within 5-10 years.

The image contains photographs and diagrams related to eye structures and procedures.
Case Presentation

Symptoms:
- Double vision (vertical and horizontal)
- Severe ptosis, OD
- Ipsilateral frontal headache
- Dilated pupil, OD
- Down and out position, OD

Pupil Involving Third Nerve Palsy

Isolated CN3 palsy with pupil involvement should be considered an ophthalmic and neurosurgical emergency (95-97% is caused by an aneurysm in PCA)

Pupil Involving Third Nerve Palsy

Imaging:
- Computed tomography angiography (CTA)
- Magnetic resonance angiography (MRA)
- Common catheter angiography (CCA)

Highly and equally sensitive in diagnosing of 95% of 3 mm intracranial aneurysm or larger

Early intervention can improve return of neural function and avoid a catastrophic rupture, which carries a significantly high rate of mortality
Case Presentation

- 54 yo female.
- Flashes of light and floaters since yesterday, OS
- Darkness over one half of visual field OS

- BCVA: 20/20 OD, OS
- Shaffer’s sign “tobacco dust” pigmented cells in the vitreous
- And:

Rhegmatogenous Retinal Detachment

- Full thickness retinal defect caused by liquefied vitreous passing through a retinal break into a potential epithelioretinal interspace between the sensory retina and the RPE.
- ”rhegma” – break
- Most common retinal detachment

Rhegmatogenous Retinal Detachment

Risk Factors
- Posterior vitreous detachment
- Axial myopia (> -3 D increases the danger 10 fold)
- Surgery (Post cataract surgery or Yag laser capsulotomy)
- Lattice degeneration: (Up to 30% of all RRD.)
- The lifetime risk of RD in a patient with lattice is less than 1%.
- RD in one eye
  - Risk in 2nd eye is 5.8% (1st year) and 10% within 4 years.
- Fam hx of RD
- Ocular trauma
- Genetic disorders: Marfans syndrome, sticklers syndrome
Rhegmatogenous Retinal Detachment

Treatment depends upon the type and size of retinal detachment:

- Small & peripheral:
  - Laser photocoagulation (or cryotherapy) around the break
  - Takes 3 - 14 days for maximal retinal adhesion to be achieved.

- Larger RRD:
  - Vitrectomy
  - Scleral buckle
  - Pneumatic retinopexy
  - CF6 or C3F8 "air/gas bubble" as a tamponade
  - Silicone oil

Time frame for management:

- The status of the macula is the primary determinant of the emergency.
  - If "Mac on" the repair should be performed within 24 hours.
  - If "Mac-off" the repair is less critical and can be performed within 7 days.

Case Presentation

- 72 yr WM
- Sudden, severe, painless loss of vision in right eye
- MHx: left carotid stent (3 weeks ago)
- Meds: Plavix, Aspirin, Multivitamin
- BCVA:
  - CF, OD
  - 20/30, OS
  - APD
Central Retinal Artery Occlusion

Presentation:
- Sudden, painless, monocular, severe loss of vision.
- May have had previous episodes of amaurosis fugax.
- >50 years of age
- Vision acuity from 20/40 to CF
- Retinal appearance:
  - Opaque, edematous
  - Cherry red spot
  - Narrowed retinal arterioles
  - Pallor of ONH and sometimes disc edema.
  - Laser ablation: optic nerve atrophy and neovascularization (controversial)

Central Retinal Artery Occlusion

Etiology:
- Arterial emboli (cholesterol, fibro-platelet, calcific)
- Thrombotic
- Arteritis: GCA, polyarteritis nodosa.
- Hypercoagulation disorders, collagen vascular, and inflammatory disorders such as lupus.
- Ocular conditions associated with retinal arterial obstruction (ONH drusen)

Embolus is the most common cause of CRAO, accounting for over 2/3 of all cases. The carotid artery and the heart are the most common sources.

Central Retinal Artery Occlusion

Treatment:
- To dislodge the embolus:
  - Ocular massage
  - Artery dilation to up to 16% due to autoregulation
  - Blood flow increases to up to 86%
  - Then, lowering IOP
  - Arterial chemoreceptor parasympathetic
  - IV urokinase
  - Vasodilation (inhaled CO2)
  - Intra-arterial thrombolysis
  - Hyperbaric oxygen therapy
### Central Retinal Artery Occlusion

#### Systemic evaluation:
- Cardiovascular evaluation
- Hyperviscosity evaluation
- Hypercoaguable evaluation
- Hemoglobinopathies

<table>
<thead>
<tr>
<th>Vascular work up suggested</th>
<th>Systemic evaluation:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exclude arteritic cause</td>
<td>Cardiovascular</td>
</tr>
<tr>
<td>Common vascular risk factors</td>
<td>Blood pressure, fasting cholesterol and profile, fasting blood sugar level</td>
</tr>
<tr>
<td>Investigating for embolic sources</td>
<td>Duplex carotid ultrasound, echocardiogram</td>
</tr>
<tr>
<td>Young patients (&lt;50yo) and/or vascular risk factors</td>
<td>Vascular screen (ANA, ENA, ANCA, ACE), hypercoaguable screens (antiphospholipid antibody, factor V Leiden, protein C &amp; S), myeloproliferative or sickle cell disease (blood film, homocysteine)</td>
</tr>
</tbody>
</table>

#### Systemic and ocular conditions related to retinal artery occlusions:

| Coagulopathies | Antiphospholipid antibodies, Protein C deficiency, Protein S deficiency, Antithrombin III deficiency, Elevation of platelet factor 4, Sickle cell anemia, Homocysteine. |
| Systemic vasculitis | Polyarteritis nodosa, temporal arteritis, Kawasaki's syndrome, Wegener's granulomatosis, Sjogren's disease, systemic lupus erythematosus. |
| Osteologic | Metastatic tumors, leukemia, lymphoma. |
| Infective disease | Syphilis, HIV. |
| Trauma | Direct ocular compression, penetrating injury, retrobulbar injection, orbital trauma, Pneumothorax. |
| Ocular conditions | Premature arterial loops, optic nerve drusen, necrotizing herpetic retinitis, toxoplasmosis. |
| Other causes | Oral contraceptives, pregnancy, drug abuse, migraine. |

#### Central Retinal Artery Occlusion
- The only true emergency would be if the patient comes within 97 min of the event and to rule out giant cell arteritis in patients older than 50 years.
- If GCA, steroid therapy is recommended immediately and scheduled for temporal artery biopsy.
- Otherwise, etiology evaluation is generally recommended as an outpatient basis.
- Time is tissue!!!
- Prognosis:
  - Life expectancy: 5.5 years
**Case Presentation**

- 78 yo WF
- Sudden and painless decrease in vision for 2 days, OD
- New onset of headaches for three days that gets worse after combing hair
- Joint pain
- General malaise
- RAPD
- BCVA: 20/300 OD, 20/30 OS
- After DFE and HVF:

**Arteritic Anterior Ischemic Optic Neuropathy**

- Inflammatory and thrombotic involvement of the short posterior ciliary arteries (SPCAs) with resultant optic nerve head occlusion and infarct of retina
- Caused by Giant cell arteritis (GCA)
- Chronic medium and large sized arteries vasculitis that affect the superficial temporal arteries and ophthalmic arteries
- Approximately 0.5-27 cases per 100,000 people aged 70 years or older
- Incidence is higher for Caucasians and European descent
- Females 3x > males

**Systemic presentation:**

- Headaches
  - New onset headaches
  - Most common systemic complaint
  - Localized to the temporal side of the head
- Jaw claudication
- Scalp tenderness (combing hair)
- Night sweats
- Fever
- Arthralgia
- ≥ 60 yo
  - 90% of affected individuals >65 yo
Arteritic Anterior Ischemic Optic Neuropathy

Ophthalmic presentation:
- Amaurosis fugax (30%)
- Visual field defect (commonly altitudinal)
- APD
- Pale swollen disc often with flame-shaped hemorrhages
- Optic cupping and atrophy occur later as the edema resolves

Management
- Laboratory testing:
  - Erythrocyte sedimentation rate (ESR)
  - ≥ 47 mm/h
  - Normal in 13% of GCA patients
  - C-reactive protein (CRP)
  - ≥ 2.45 mg/dl
  - CBC with differential = anemia of chronic inflammation

ESR + CRP → 99% sensitivity, 97% specificity for GCA

Treatment
- Systemic corticosteroids
  - Immediate IV steroid therapy
  - 2 weeks or until symptoms resolve and ESR, CRP normalize
  - Slow taper of oral prednisone for ~ 24 months.
  - Temporal artery biopsy (gold standard)
  - Once visual loss occurs, it is rarely recovered.
Case Presentation:
- Blowout Fracture

Case Presentation
- Penetrating Ocular Injury

Case Presentation
- Perforating Ocular Injury
Risk Factors for Open Globe Trauma

Take Home Message
- Have a clear pre-determined plan of action
- Be prepared with an emergency kit:
  - Litmus strips
  - IOP lowering medications
  - BP kit
  - Thermometer for suspected cellulitis
  - Fox shield and tape
  - CPR responder pack
  - Epinephrine pen
- Calm demeanor in the face of emergency is critical because patients feel worse and loses confidence if the doctor appears anxious

Take Home Message
- Knowledge of local professionals who can help and know how to refer:
  - Ophthalmologist with specialty
  - Emergency room
  - Internal medicine
  - Neurology
  - Radiology
  - Infectious disease specialist
- Document, document, document
  - If it is not in the chart, it was not done
- Appropriate follow up care
  - Phone calls to patient as well as specialty doctors will enhance the quality of care
LASIK Emergency - Presentation

- ER physician calls about 29 yo AF post op week #2 after LASIK OU with a 20/20 result OU whose 5 year old child had struck her in the eye with a "piece of metal"
- Vision on presentation: 20/CF at 2' Symptons: significant pain and photophobia

LASIK Emergency – Physical Exam

- Corneal tissue appeared heaped up over visual axis

LASIK Emergency - Management

Cleaning Beneath Flap
LASIK Emergency – Follow Up

- Presentation 20/CF
- Post reposition, cleaning, smoothing, and epithelial debridement
- 2 Days post op
- UCDVA 20/20

PROMPT repositioning and smoothing critical to prevent permanent tissue folds

References


9. Mario Bradvica, Tvrtka Benašić and Maja Vinković (2012). Retinal Vascular Occlusions, Advances in Ophthalmology, Dr Shimon Rumelt (Ed), InTech, Available from:


