In-Office Care of the Emergency Patient

Blunt Trauma
Janice McMahon, OD
March 13, 2016

Statistics

- 90% of the 1 million eye injuries seen in the ER per year in the United States are preventable
- 2,000 workplace eye injuries per day
- 1,500 firework injuries per year
- 40,000 sports-related eye injuries per year

Penetrating injury

Blunt ocular trauma
Blunt Orbital Trauma - Fractures

- A fracture of the orbital floor or nasal wall may force air from the sinuses into the orbit; normally there is no connection
- Signs include proptosis, subconjunctival air, and crepitus
- Avoid coughing and do not blow nose
- Treat with ice packs, nasal decongestants, and broad-spectrum antibiotics

Blow-out Fracture

- Due to blunt trauma to inferior rim, compressing the globe within the orbit
- Most common sign is restriction of vertical motility
- Requires x-ray or coronal CT scan
- Surgical repair if diplopia persists or cosmetically unacceptable enophthalmos
Medial Wall Fracture

- Blow to the nose can fracture thin medial wall
- Signs include orbital emphysema, epistaxis, enophthalmos, and depressed nose bridge
- Entrapment of medial rectus is rare
- May have damage to lacrimal system
- Treat with oral antibiotics, ice packs, and nasal decongestants
- Most heal without significant sequelae

Superior Wall Fracture

- Less common, but can be life-threatening
- Potential complications of brain abscess and infectious meningitis
- Signs include rhinorrhea, traumatic ptosis, frontal sinus mucocele, and optic nerve injury
Tripod Fracture

- Involves zygomatic arch which forms lateral & inferior orbital rim
- Signs of blow-out fracture may exist
- Limitation of mandibular movement
- Surgery needed within two weeks to wire bones

Fracture Follow Up

- Check at one and two weeks post-trauma for diplopia and enophthalmos
- Perform gonioscopy and scleral depression at 3-4 weeks
- Education patient on signs and symptoms of orbital cellulitis and retinal detachment

Blunt trauma

- Damage by both compressive and concussive forces
- Ocular structures may become displaced
- Full extent of injury may not be initially apparent, follow up for late complications is important
- Potential impact on all ocular structures

Ecchymosis

- Blunt trauma causes soft tissue damage
- “black eye”
- Look for fractures and anterior segment damage
- Evaluate optic nerve head and peripheral retina - DILATE
- Treat with cold compresses first 24 hours and warm thereafter
**Blunt trauma to conjunctiva**

- Subconjunctival hemorrhage
- Conjunctival edema
- Conjunctival laceration
- Occult scleral rupture – refer if penetrating globe

**Blunt trauma to cornea**

- Endothelial stress
- Post-traumatic keratopathy
- Descemet’s rupture
- Corneal rupture
**Blunt trauma to iris**

- Sphincter muscle tear
- Traumatic iritis
- Iridodialysis

**Hyphema**

- Tear of anterior ciliary body
  - Blood into anterior chamber
  - Potentially serious
- Child vs. adult
- Traumatic vs. spontaneous
Grading a Hyphema

- Microscopic: Red blood cells not settling
- I: <33% AC filled with blood
- II: 33-50%
- III: 50-95%
- IV: 100%, total or eight ball hyphema

Hyphema Treatment

- Hospitalization for children and elderly
- Bed rest vs. limited activity
- Limited ocular motility
- Patch
- Cycloplege
- Steroids
- Antifibrinolytic
Hyphema Rebleed

- Usually occurs day 2-5 after initial injury
- Higher incidence of morbidity: increased IOP, corneal staining, prolonged clot
- 9-38% have complications

Hyphema Follow Up

- If no rebleed, taper steroid and cycloplegic on day 6
- No strenuous exercise for 2 weeks
- Wear eye shield at night
- Normal activity at 1 month
- DFE with gonioscopy and scleral depression at 1 month

Lens Dislocation

- Usually traumatic but can occur from systemic or congenital diseases such as Marfan’s
- Total (luxated) or partial (subluxated) rupture of zonules
- Symptoms of reduced or fluctuating vision, glare, monocular diplopia
- Visualize zonules, vitreous, shallow AC, iridodonesis, phacodonesis
Contusion Cataract

- Rupture of lens capsule
- Rapidly forming cataract
- May not be progressive, observe

Choroidal damage from blunt trauma

- Choroid is weaker than either the sclera or retina and more likely to suffer damage after blunt trauma
- Choroidal rupture is usually from a contrecoup (indirect) injury
  - Concentric to the disc
- SRNVM may be a late complication
Blunt trauma to macula

- Macular holes can follow commotio retinae, choroidal ruptures, or subretinal hemorrhages
- Can also occur with whiplash injury
- May present years after trauma
- BIO and FANG differentiate full from partial thickness holes
Macular Hole Treatment

- Vitrectomy removes vitreous and any remaining vitreal strands attached to the retina at the posterior pole
- Air bubble is injected into the eye and the patient must maintain a head-down position for a minimum of 2-3 weeks
- Most optimistic for holes present <6 months
Blunt trauma to retina

- Blunt injury causes traction at vitreous base
- Most frequently inferotemporally and superonasally
- Pre-existing retinal degeneration increases risk for tears, breaks, and retinal detachment

Focal laser

Barrier grid laser
Commotio Retinae

- Swelling of the retina after trauma
- White opacification with ill-defined borders
- Resolves in days to weeks
- Also called Berlin's edema when macular
Neuro-Ophthalmic Emergencies

Katherine B. Lynch, OD, FAAO

Stay calm – ask your questions!

- Is this true binocular diplopia
  - Does it disappear when one eye is covered
  - If yes, are the images horizontal or vertical worse at distance or near, left or right gaze
- A good case history is imperative
  - Frequency
  - Onset
  - Duration
  - Eye involved
  - Systemic history (DM, HTN, h/o Cancer?)
  - Associated symptoms?

The exam

- Vision
- Pupils
- EOMS
  - Versions
    - Ductions – required if versions are not full
      - Hering’s Law
    - If deviation is present in both → true paresis or mechanical restriction
- Cover test
  - Phoria or tropia?

Is it comitant?

- Comitancy= the same deviation in all positions of gaze. (within 4 pd)
- How do you know?
  Maddox Rod in 9 positions
- Is there a suspicion of a hyper deviation? Don’t forget to tilt the head and remember your Park’s 3 Step

Parks Three Step – It’s a March

- In primary gaze which eye appears hyper? LEFT
- Is the hyper exacerbated in left or right gaze? RIGHT
- When you tilt the head right or left, is the hyper more pronounced. LEFT

The deviation is comitant

How do you know?

- Versions = normal
- Maddox rod = within 4 pd in all positions of gaze

→ On to Von Graefe measurements/binocular testing!
Things that give us comitant deviations

- Decompensated phorias
- Loss of bifoveal fixation (AMD)
- Longstanding paralysis/paresis
- Childhood strabismus

... to name a few

→ Comitant deviations should give you peace of mind.

What if it is not comitant?

Identify your emergencies!

- Was this acute? Constant?
- Is the pupil involved?
- Do multiple muscles/nerves appear to be involved?
- Is it painful?
- Do you suspect increased intracranial pressure?
- GCA symptoms?

→ Emergent work up, referral to the ER

Not all non-comitant deviations are emergencies, but all of them require more care

- Based on your measurements, observations, you can make inferences about the lesion
  - Horizontal or vertical restriction?
  - Worse at the end of the day?
  - Forced duction
  - Associated symptoms: nystagmus, ptosis, etc.

→ These will require coordination with the PCP, likely neurology, and imaging

Restrictive= Problem is in the orbit!

- Thyroid Eye Disease
- Orbital pseudotumor
- Space occupying lesion
- Entrapment

Neurologic: Vascular, Neuromuscular, Neural

- Diabetes
- Hypertension
- Myasthenia Gravis (a great mimic)
- INO (nystagmus)
- 1 ½ Syndrome (nystagmus)
WHAT MIGHT SOME OF THESE EMERGENCIES LOOK LIKE?

Famous Friends...

Third Nerve Palsy: When you are really feeling down and out...
- Textbook: Large ptosis on the affected side, eye involved appears to be down and out.

- Classic causes: Aneurysm or Diabetes
- Complete (all muscles of the 3rd) vs. Incomplete (all or some of the 3rd to varying extent)
- "Rule of the pupil" -- Pupil involved vs. sparing (where do pupil fibers live? = outside)
- Painful (PCA) vs. non painful
- 3rd nerve palsy is an emergency until proven otherwise

Fourth Nerve Palsy: It’s traumatic being fourth...
- Textbook: Hyper deviation, relief with head tilt away from the affected side

- Classic causes: – History of trauma!
  – Congenital (but you wouldn’t complain then)
- No trauma and it’s new, they’ve got place to go -> Imaging!

Sixth Nerve palsy: A lot of pressure...
- Textbook: Unilateral esotropia worse at distance and when attempting to look laterally toward the affected side
• Classic causes
  – Viral in children
  – Recent illness?
  – Ischemic in adults
  – h/o hypertension/DM
• BUT be careful here, because of its’ course, it is vulnerable to compression
  – Look for s/s of ICP

If there are multiple nerves involved, think further back...
• The middle school dance of the Cavernous Sinus
  – Wallflowers (run in the wall)
    • CN III
    • CN IV
    • CN V
      – VI
      – VII
  – Out on the dance floor (through the center of the sinus)
    • CN VI
    • Internal Carotid w/the sympathetic plexus

Signs and Syndromes of the CS
• Ophthalmoplegia
• Sensory loss
• Abnormal pupils
• Classic causes:
  – Tolosa-Hunt
  – Pituitary Tumors
  – Carotid Cavernous Fistula
  – Infection

Increased Intracranial Pressure
• Classic causes: Hemorrhage, infection, space occupying lesions, many others...
• Signs: Headache, changes in vision, DIPLOPIA, tinnitus, nausea, changes in consciousness/behavior
• How do the optic nerves look?
  – Remember Papilledema, by definition, is bilateral optic nerve swelling secondary to increased intracranial pressure
• Imaging prior to LP

Papilledema and your OCT
• Anomalous nerves, ONHD or early papilledema?
  – Shape of RPE/Bruch’s Membrane
  – RNFL thickness
  – Hyper-reflective spaces (?)
  – Consider HVF (enlarged blind spot vs. others areas of scotoma), Bscan

Giant Cell Arteritis
• Eye pain over age 55 \( \rightarrow \) Always ask about scalp tenderness, jaw claudication, recent malaise
• GCA has a predilection for the Ophthalmic Artery
• Changes in vision
• Diplopia
• ESR, CRP, Platelets to be ordered TODAY
• Can lose vision in the fellow eye in a short time frame, so high dose steroids are often started before temporal biopsy is completed and GCA is confirmed
Unilateral Nerve Swelling

- NAION
- Diabetic Papillitis
- Vein occlusion
- Uveitis
- Optic Neuritis
- Infiltrative disease
- Compression
Corneal Urgencies and Emergencies

Overview

- Corneal Abrasions
- Corneal Erosions
- Chemical Injuries
- Corneal Lacerations
- Corneal Foreign Bodies
- Corneal Ulcers

Format for Today’s Discussion

- Presentation of Case
- Review Etiology of Condition
- Discuss Treatment Options

Triage

- Emergent Situation
  - Requires immediate action
  - Patient is seen same day

- Urgent Situation
  - Requires patient be seen within 24-36 hours

- Routine Situation
  - Requires patient to be seen within a few days to a week

Symptoms can point you in the right direction...

- Redness
- Discharge
- Foreign body sensation
- Itching
- Burning
- Lid edema
- Photophobia
- Pain
- Changes in vision

Defense Mechanisms of the External Eye

- Ocular Adnexa
- Lymphoid Tissues
- Eyelid Blinking
- Tear Turnover
65 y/o WM

Corneal Abrasion Treatment

- Antibiotic bid to q2hr
- Ointment qid
- Copious Lubrication
- NSAID
- Cycloplegic
- Bandage CL
- Pressure Patch
- Amniotic Membranes
- Follow up every 24 hours

Potential Complications

- Corneal scarring
- Infectious keratitis
- Decreased vision
- Recurrent erosions

90 y/o AAF
Recurrent Corneal Erosion (RCE) Etiology and Pathophysiology

- Previous traumatic corneal abrasion
  - Fingernail, tree branch, paper
- Corneal dystrophies
  - Anterior basement membrane, Lattice, Granular
- Disturbance to Bowman’s layer

- Age: 24-73 (highest prevalence between 3rd and 4th decade)
- M=F (slightly higher female)
- Interval between initial abrasion and first recurrence: 2 days to 16 years
- 10% cases bilateral

RCE Acute Treatment

- Similar to treatment for abrasions
  - Antibiotics
  - Copious Lubrication
  - Cycloplegics
  - NSAIDs (topical vs oral)
  - Bandage CL
- May need to debride
  - If not healing in 24-48 hours

RCE Prophylactic/Long-term Treatment

- Hypertonic therapy
  - Muro 128 drops or ung qid
  - Doxycycline (50-100 mg bid)
  - Vitamin C
  - Azasite
  - Restasis and punctal plugs
  - Autologous Serum
- Bandage and/or scleral lenses
- Amniotic membranes
- Topical corticosteroids
  - Lotemax
- Surgical
  - Anterior stromal puncture
  - Phototherapeutic keratectomy (PTK)
  - Superficial keratectomy

**Most require combination therapy**

61 y/o AAF
### Chemical Injuries

- Potentially vision threatening!
- No matter how busy... Chemical burns are seen FIRST!
- By-pass VA, quickly check pH, then begin immediate irrigation
  
  **IRRIGATE, IRRIGATE, IRRIGATE!!**

- Time and action are critical
- Severity is related to:
  - Properties of the chemical
  - Area of affected surface
  - Duration of exposure

### Etiology

- **Eye injuries account for 4-7% of workplace injuries**
  - 84% are chemical burns

- **Incidence**
  - 30 per 10,000
  - 82-91% men (16-45 y/o)
  - 90% accidental
  - Alkali 2x more common
  - 2/3 occur at work
  - 10% Intentional
  - Assault

### Chemical Injuries

- **Acidic Burns**
  - Sulfuric (battery acid), hydrofluoric, hydrochloric
  - Usually a self-limiting burn
  - Low pH: 6.9 or lower

- **Alkali Burns**
  - Lime, ammonia, sodium hydroxide
  - Very damaging- can easily penetrate all ocular layers
  - High pH: 7.1 or greater
Grading of Severity

- Grade I
  - Involves corneal epithelium only
  - Limbal stem cells spared
  - No limbal ischemia
  - Cornea remains clear
  - Prognosis: Excellent

- Grade II
  - Partial loss of limbal stem cells
  - Focal limbal ischemia
    - <1/3 of limbus
  - Hazy cornea
  - Anterior segment structures are visible
  - Prognosis: Good

- Grade III
  - Total loss of corneal epithelium
  - Loss of most limbal stem cells
  - Stromal haze
  - Extensive limbal ischemia
    - 1/3 to ½ of limbus
  - Prognosis: Guarded

- Grade IV
  - Complete loss of corneal epithelium and limbal stem cells
  - Opaque cornea
  - No view of iris or pupil
  - “Porcelainization”
    - > ½ Limbal ischemia
  - Prognosis: Extremely poor

Grading

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Chemical Injuries Treatment

- Depends upon severity of burn
  - Severe burns (aka significant ischemia, open globe, periocular open wounds) = immediate external referral
  - Mild to moderate = treat the problem

  - Copious irrigation with normal saline for at least 30 min and repeat every 30 minutes until neutral pH is reached

Medical Treatment

- Double eversion of the upper eyelid
- Debridement of necrotic epithelium
- Artificial tears
- Antibiotic ointment
- Cycloplegic agents

  Goals of Treatment: Reduce inflammation, promote epithelial regeneration, and prevent corneal ulceration

- Steroids
- BCL
- Sclerals
- Ascorbic acid (Vitamin C)
- Citric acid
- Doxycycline
- Punctal plugs

Surgical Treatment

- Limbal stem cell transplantation
- Amniotic membrane grafting
- Division of conjunctival bands
- Correction of eyelid deformities
- Tarsorrhaphy
- Penetrating Keratoplasty
- Keratoprosthesis

Goal: Promote revascularization of the limbus, restore limbal stem cells, re-establish fornices

Follow up every 24 hours!
54 y/o CM

Potential Complications

- Corneal Opacification/Scarring
- Symblepharon
- Dry Eye
- Ocular Ischemia
- Corneal Neovascularization
- Loss of limbal stem cells – irregular corneal surface

- Punctal stenosis or occlusion
- Limbal stem cell deficiency
- Pannus formation
- Cataracts
- Glaucoma
Corneal Lacerations

- Etiology:
  - Industrial accidents
  - Traffic accidents
  - Home accidents
  - Assault
  - Traumatic wound rupture

- Immediate Referral
- Small, partial-thickness lacerations may generally be treated as abrasions with patching therapy
- Larger wounds often require immediate surgery
- No ointment should be applied to the eye
- Seidel Test
  - Positive = full thickness
  - Negative = partial thickness
- Fox shield over eye for protection
  - Need to keep anterior chamber intact

29 y/o CM

Corneal Foreign Body Removal

- ***Accurate assessment of depth of penetration before removal***
  - Pupil irregularities, iris tears, transillumination defects, lens opacities, hyphema, shallow A/C, low IOP
  - Irrigation
  - Sterile cotton swab or sponge
  - Spud
  - Jeweler’s forceps
  - Needles
  - Alger brush
  - May need to remove deeper rust rings at a later date after they have migrated to the surface
Corneal Foreign Body Treatment

- After removal of foreign body
  - Measure size of resultant epithelial defect
  - Treat as for corneal abrasion
- Cycloplegic
- Antibiotic
- Bandage contact lens
- Corticosteroids
  - After re-epithelialization to reduce scarring
- Follow up
  - 24 hours

Look for signs of intraocular FB:
- Corneal laceration, iris tear, lens opacity, collapsed anterior chamber, low IOP
Corneal Ulcer

- **INFECTION** of the cornea by microbes
- Characterized by excavation of the corneal epithelium, Bowman's, and stroma
- Infiltration
- Necrosis of tissue
- Ideally all cases should be cultured
- Realistically...
  - >2mm in size
  - <3mm from visual axis
  - >1/4 corneal depth

Corneal Ulcer Etiology

- Bacterial
  - S. aureus
  - S. pneumoniae
  - M. lacunata
  - P. aeruginosa

- Fungal
  - Fusarium
  - Aspergillus
  - Candida

- Acanthamoeba

Corneal Ulcer Risk Factors

- Eyelid disorders
- Chronic corneal disease
- Refractive surgery
- Blepharitis
- Chronic lacrimal drainage obstruction
- Immunosuppression
- Trauma

- Contact lenses
  - Poor personal hygiene
  - Contaminated solutions
  - Lens surface deposits
  - Non-compliance with disinfection
  - Lens manipulation
  - Corneal hypoxia with extended wear

Corneal Ulcer Signs/Symptoms

- Clinical manifestations
  - Foreign body sensation
  - followed by increasing pain and photophobia
  - Decreased VA
  - Marked conjunctival hyperemia and inflammation
  - Ciliary flush
  - Pupillary constriction
  - Mucopurulent discharge
  - Intense anterior chamber reaction, with or without hypopyon
  - Ragged, irregular epithelial ulceration with underlying necrotic stromal infiltration and surrounding epithelial edema

Signs in chronological order

- Infiltrate
  - Circum-corneal injection
  - Stromal edema
  - Small hypopyon

- Enlargement of infiltrate
  - Enlarging hypopyon
  - Severe infiltration

- Progressive infiltration
  - Corneal perforation
  - Endophthalmitis

Ulcer Vs. Infiltrate
Corneal Ulcer Treatment

- Discontinue CL wear
- Culture
- Broad spectrum topical antibiotic therapy
  - Consider loading dose of antibiotic
  - One drop q 5 min for 15-30 min
  - One drop q 30-60 min for 24 hrs
- Fortified subconjunctival or IV antibiotics
  - Cefazolin, vancomycin, gentamycin, tobramycin
- Cycloplegic
- Topical corticosteroids
  - SCUT study
- Amniotic Membrane
- PKP
- CXL?

Fungal Keratitis Treatment

- Filamentous fungi: Natamycin 5% (Natacyn) q1hr (including during sleep)
- Yeast: Amphotericin B 0.15% q5min x 1 hour, then q1hr
  - Fluocytosine orally 150mg/kg/day or topically 1% 1gt q30mins to inhibit fungal growth
- No Steroids
- Consider hospitalization
- Voriconazole

**Natamycin is the only commercially available antifungal for ophthalmic use in the US**

Acanthamoeba Keratitis Treatment

- Topical treatment often includes a combination of agents:
  - **Loading dose** (first 1-3 days)
    - Chlorhexidine 0.02% and/or PHMB (polyhexamethylene biguanide) 0.02%
    - AND
    - Propamidine isethionate 0.1% (BroleneTM)
  - **POSSIBLY INCLUDE**
    - Neomycin solution or fluoroquinolone
  - All meds q1hr (also during sleep)
  - Each drug given at the same interval separated by 5 minutes

Corneal Ulcer

- Follow up every 24 hours
- Pain level, inflammation, and size of defect should decrease continually
- Taper medications as needed
- Refer non-compliant patients and worsening ulcers
Summary

- Reviewed Conditions:
  - Corneal Abrasions
  - Corneal Erosions
  - Chemical Injuries
  - Corneal Lacerations
  - Corneal Foreign Bodies
  - Corneal Ulcers
- Case Presentation
- Etiology Review
- Treatment Discussion

Clinical Pearls

- Thorough history
- Symptoms can point you in the right direction
- Careful examination
- Treat each case individually
  - Consider newer approaches
- Follow up appropriately
- Refer as necessary
Retinal Urgencies and Emergencies

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Assistant Professor
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March 13, 2016

Format for Today’s Lecture

- Case Presentation
- Review of Condition
  - Sequelae and appropriate follow-ups
- Treatment and Referral Discussion

Classification of Systemic Hypertension

<table>
<thead>
<tr>
<th>Stage of HTN</th>
<th>BP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Systolic: &lt;120 OR Diastolic: &lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>Systolic: 120-139 OR Diastolic 80-89</td>
</tr>
<tr>
<td>Stage 1 HTN</td>
<td>Systolic: 140-159 OR Diastolic: 90-99</td>
</tr>
<tr>
<td>Stage 2 HTN</td>
<td>Systolic: &gt;160 OR Diastolic: &gt;100</td>
</tr>
</tbody>
</table>

Goal is to maintain blood pressure <140 / 90 mm Hg

Symptoms of Target Organ Damage

- Shortness of breath
- Headache (often severe, bilateral, pulsating frontal or occipital)
- Dizziness
- Chest pain (myocardial infarction symptoms)
- Paresis/paresthesias (TIA/CVA symptoms)
- Acute ischemic or hemorrhagic stroke
- Acute myocardial infarction
- Acute heart failure
  * Papilledema
  * Cotton wool spots, hemorrhages, exudates
  * Vitreous hemorrhage
  * Progressive TOD: ONH edema

Bilateral swollen nerves...What other findings present?

Hypertensive Crisis

- Upper level of Stage II HTN (>180/120)

Dilate vs not??

Hypertensive Urgencies
- >180/120 (-) progressive TOD

Hypertensive Emergencies
- >180/120 (+) progressive TOD
Hypertensive Crisis Management by OD’s

<table>
<thead>
<tr>
<th>BP</th>
<th>Clinical Findings</th>
<th>Referral</th>
<th>Timeline</th>
<th>Ocular F/U</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe HTN</td>
<td>No acute TOD</td>
<td>PCP</td>
<td>3-7 days</td>
<td>1-3 m</td>
</tr>
<tr>
<td>HTN Urgency</td>
<td>(+) Mild HTN ret</td>
<td>PCP</td>
<td>24-72 hrs</td>
<td>1 m</td>
</tr>
<tr>
<td>HTN Emergency</td>
<td>(+) Mod HTN ret</td>
<td>ER or call 911</td>
<td>Immediate, decreased 15-25% within 2 hours</td>
<td>1 m after hospital release</td>
</tr>
</tbody>
</table>

Hypertensive Retinopathy Classifications: Older

<table>
<thead>
<tr>
<th>Grade</th>
<th>Scheie (1953)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Broadening of light reflex with mild arteriole narrowing</td>
</tr>
<tr>
<td>II</td>
<td>More pronounced narrowing, focal constriction, AV crossing changes</td>
</tr>
<tr>
<td>III</td>
<td>Grade 2 + hemorrhages, exudates, copper wire appearance of arterioles</td>
</tr>
<tr>
<td>IV</td>
<td>Grade 3 + papilledema, silver wire appearance of arterioles</td>
</tr>
</tbody>
</table>

Three year survival rate: 22% for Grade III and 6% Grade IV

Hypertensive Retinopathy Classifications: Newer

<table>
<thead>
<tr>
<th>Grade</th>
<th>Wong (2004)</th>
<th>Systemic Associations</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>No detectable signs</td>
<td>Modest association with risk of stroke, heart disease, and death</td>
</tr>
<tr>
<td>Mild</td>
<td>Generalized arteriolar narrowing, focal arteriolar narrowing, arteriovenous nicking, or a combination</td>
<td>Strong association with risk of stroke, death from cardiovascular disease</td>
</tr>
<tr>
<td>Mod.</td>
<td>Presence of blot, or flame-shaped hemorrhage, microaneurysm, soft exudates, or a combination of these signs</td>
<td>Strongest association with death</td>
</tr>
<tr>
<td>Severe</td>
<td>Presence of moderate hypertensive retinopathy signs; and optic disc swelling</td>
<td></td>
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CRVO (Central Retinal Vein Occlusion)

<table>
<thead>
<tr>
<th>Ischemic (non-perfused) CRVO</th>
<th>Non-ischemic (perfused) CRVO</th>
</tr>
</thead>
<tbody>
<tr>
<td>% of CRVO</td>
<td></td>
</tr>
<tr>
<td>RAPD?</td>
<td></td>
</tr>
<tr>
<td>FA</td>
<td></td>
</tr>
<tr>
<td>BCVA</td>
<td></td>
</tr>
</tbody>
</table>

5-22% non-ischemic convert to ischemic
CRVO sequelae: NVG

- 33-45% ischemic CRVO's develop NVG
  - NVI very common *look VERY closely!*
  - "90 Day Glaucoma"

- Follow up: monitoring for neo (iris, IOP, Gonio, DFE)
  - q 1 m x 6 m
  - q 3 m x 6-9 m
  - q 4-6 m x 3 years

- Referral to glaucoma: NVG remains a therapeutic challenge
  - Pharmacological tx, PRP, trabeculectomy, drainage implants

CRVO sequelae: Macular Edema

- Treated with Anti-VEGF
- CVOS vs. SCORE vs. CRUISE vs. Ozurdex vs. Copernicus/Galileo

- Referral within 2-4 weeks
  - Focal laser, PRP if ischemia and neo
  - Avastin/Lucentis/Eylea
  - Ozurdex
  - Kenalog
  - Triamcinolone

BRVO

- Ischemic (rare)
  - >4 DD capillary non-perfusion FA
  - At risk to develop neo
    - NVD/NVE more common than NVI

- Non-ischemic (common)

  - Macular edema
    - BVOS vs. SCORE vs. BRAVO
    - Avastin/Lucentis/Eyelea
    - Occasional laser

BRAO

Cilio-retinal artery occlusion
Artery Occlusions and GCA

- CRAO → May be associated with GCA (2-10%)
  - Occlusion of PCA (GCA has predilection to PCA)
  - NOT CRA
  - Share common trunk

- Cilio-retinal artery occlusion → May be associated with GCA

- BRAO → GCA association VERY RARE!!

- Clinical pearl: No embolus seen → Ask GCA history questions
  - Work up if suspected

Systemic pathology: CRAO

- Younger (< 40 years old): coagulopathies, collagen vascular disorders

- Older (> 40 years old): GCA, heart, carotid

What blood work and/or tests to suggest?

- CBC with diff → anemia, polycythemia, and platelet disorders
- ESR and CRP → GCA
- FBS, cholesterol, triglycerides, and lipid panel → atherosclerotic disease
- IN PERSONS <40 YEARS OLD
  - Fibrinogen
  - antiphospholipid antibodies
  - PT/aPTT
  - serum protein electrophoresis
  - ANA, RF, anticitrullinated protein antibodies

- Carotid ultrasound or doppler → atherosclerotic disease (plaques)
- Electrocardiogram (ECG) → arrhythmias
- Echocardiogram → cardiac source of embolism

How many of you had a patient this week that presented with chief complaint of “flashes” and/or “floaters”?

Flashes: vitreous traction on the peripheral retina

Floaters: vitreous opacities i.e. PVD, vitreous collagen condensation, blood

Flashes and Floaters: PVD

- Flashes and/or floaters need DILATED exam that day
  - There are no symptoms that can distinguish PVD alone from PVD with an associated retinal break

- Rule out retinal tear/detachment
  - 8-26% patients with an acute symptomatic PVD have a retinal tear at initial exam
  - If no tear at initial exam, 2-5% chance one will develop within following weeks
Do not forget to look at the anterior vitreous...

Posterior Vitreous Detachment

- Signs suggestive of a peripheral retinal tear (80% with no tear at initial exam, but have at follow-up):
  - Pigment in vitreous (+) Schaeffer’s sign
  - Vitreous hemorrhage
  - New or increase in symptoms

- Tests to help find the tear
  - Scleral depression
  - B-scan

PVD Follow-up

- Symptomatic PVD with no retinal break
  - No high risk factors
    - Within 6 weeks
  - High risk factors
    - Within 24 hrs to 1 week, depending
    - ASAP if increase flashes, floaters, curtain over vision

Complications from PVD

Macular Conditions:
- Epiretinal membrane
- Macular hole
- Vitreo-macular adhesion
- Vitreo-macular traction

Peripheral Conditions:
- Rhegmatogenous retinal detachment
- Operculated retinal break
- Vitreous hemorrhage
- Retinal tear

High Risk Factors:
- Vitreous hemorrhage
- Retinal hemorrhage
- Schaeffer’s sign
- Lattice degeneration/cystic tuft
- Mod High myopia
- h/o tear or RD in other eye
- Pseudophakia
- s/p yag capsulotomy
Retinal Tear

- 25-90% of untreated, symptomatic retinal breaks will progress to RD unless prompt treatment occurs
  - Refer for immediate treatment (barricade laser)
  - After treatment risk decreases to < 5%

- Asymptomatic retinal tear
  - Call retinal specialist, may or may not be treated
Retinal Detachment

- Macula ON vs. OFF
- Location

<table>
<thead>
<tr>
<th>Location/Macula</th>
<th>Referral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Superior + macula ON</td>
<td>Emergent/Immediate</td>
</tr>
<tr>
<td>Inferior, nasal, temporal + macula ON</td>
<td>Emergent</td>
</tr>
<tr>
<td>Macula OFF</td>
<td>Call Retinal specialist</td>
</tr>
</tbody>
</table>

Types of Retinal Detachments

Rhegmatogenous
- Caused by a tear in retina
- Fluid detaches the neurosensory retina from RPE
- If non-traumatic detachment in one eye ➔ 10% increased risk of developing in other eye

Non-rhegmatogenous
- Not caused by tear
- Tractional: proliferative diabetic retinopathy, Sickle cell
- Serous/Exudative: AMD, vascular disease, tumors

Operculated Hole
Atrophic Retinal Holes In/Adjacent to Lattice

Vitreous Hemorrhage

- What is the source of the bleed?
  - Neovascularization: DM, Sickle Cell, CRVO, etc.
  - PVD/Retinal tear/Retinal detachment
  - Adjacent source? Macroneurysms, tumors, CNVM

- DFE of contralateral eye to give clues to etiology

- B-scan

Vitreous Hemorrhage

- Referral: Retina specialist
  - Retinal detachment or break → Urgent
    - Call surgeon
  - NVA or NVI → Urgent
  - Type I diabetes → One month
  - Type II diabetes → 2-3 months
  - Non-clearing in 2-3 months
**CSME (Clinically Significant Macular Edema)**

- Aka Diabetic macular edema (DME)

- CSME:
  1. Edema within 1/3 DD (500 um) of center of fovea
  2. Hard exudates within 1/3 DD, with associated/adjacent edema
  3. Edema ≥ 1 DD (1,500um), that is within 1 DD of the center of the fovea

- Recall: Can be 20/20 and have CSME

**CSME ETDRS**

- Referral: within 2-4 weeks

- Treatment:
  - Not center macula = focal laser
  - Center of macula = laser, anti-VEGF, intravitreal steroids, vitrectomy
  - Educate on adequate glycemic (<7.0 A1C), blood pressure, cholesterol control

**CSME**

- No standard treatment algorithm

- Guidelines:
  - Center of macula involved?
    - Yes: Anti-VEGF x 6m
    - No (adjacent microaneurysm): Focal/grid laser
  - No resolution – taut posterior hyaloid?
    - Yes: PPV/ILM peel
    - No: Focal/grid laser, Anti-VEGF, Triamcinolone q4-6m, Scatter Targeted Peripheral PRP, Dexamethasone, Vitrectomy
27 y/o WF CL dispense

CNVM (Choroidal Neovascular Membrane)

- Etiologies
  - AMD (10% exudative)
  - High myopia (> -6.00D)
  - Angioid streaks (PEPSI!)
  - Histoplasmosis
  - Trauma/Choroidal rupture
  - Inflammatory disorders
  - Polypoidal choroidal vasculopathy
  - Infectious: toxoplasmosis, toxocara canis, tuberculosis, viral retinopathies

CNVM

- Treatment:
  - Anti-VEGF: Avastin/Lucentis/Eyelea

- Referral: Call the retina specialist
  - If **macula involved** (i.e. AMD, myopic degeneration) ➔ **immediate** (24-72 hours)
  - If macula not involved (i.e. choroidal rupture) ➔ **within 1 week**

Analyzing CNVM with OCT

CNVM from Myopic Degeneration
CNVM from AMD

CNVM from Histoplasmosis

ANGIOD STREAKS

Traumatic Choroidal Rupture (nasal)

Traumatic Choroidal Rupture (temporal) with CNVM

CNVM from Polypoidal choroidal vasculopathy (PCV)
Summary
1. Hypertensive crisis
2. Vein Occlusions
3. Artery Occlusions
4. Flashes/floaters
   ▪ PVD
   ▪ Retinal tears
   ▪ Retinal detachments
   ▪ Retinal holes
5. Vitreous hemorrhage
6. CSME
7. CNVM

Clinical Pearls

▪ See ASAP in your office:
  ▪ Flashes and/or Floaters
  ▪ Sudden decrease vision
  ▪ Sudden metamorphopsia or scotoma
▪ Remember not only visual threats but systemic threats
▪ Communication and Referral
  ▪ Specialist
  ▪ Patient
▪ Thorough chart documentation
▪ Patient education

Thank you!
Questions?