True Ocular Urgencies or Emergencies

- Self-assessment
- Orbital Contusion
- Retinal Detachment
- Retinal Hemorrhage
- Malaria
- Glaucoma
- Acute Angle Closure Glaucoma
- Neovascular Glaucoma
- Intraocular Foreign Body
- Central Retinal Vein Occlusion
- Retinal Vein Occlusion
- Retinal Detachment
- Endophthalmitis
- Giant Cell Arteritis
- Cavernous Sinus Thrombosis
- Horner's Syndrome
- Acute Angle Closure Glaucoma
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- Glaucoma

Triaging Ocular Urgencies & Emergencies

- Triage => is defined as the sorting of patients according to the urgency of their need for care. It comes from the French verb trier, meaning to separate, sift, or select.

The goal of triage is to establish a level of urgency based on these common definitions:

- I-USE

  - Immediate: Within 1-2 hours
  - Urgent: Within 24 hours
  - Soon: Within 1 week
  - Elective: Within 1-2 weeks

  - I => 1 hour
  - U => 1 day
  - S => 1 week
  - E => 1 month

For Example

<table>
<thead>
<tr>
<th>ASAP</th>
<th>Within 1-2 hrs</th>
<th>Within 24 hrs</th>
<th>Within 1-2 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chemical Burns</td>
<td>Corneal Abrasion</td>
<td>Recent Onset Diplopia</td>
<td>Dry, itchy, redness</td>
</tr>
<tr>
<td>Acute Ocular Trauma</td>
<td>Foreign Body</td>
<td>Recent Onset Diplopia</td>
<td>Dry, itchy, redness</td>
</tr>
<tr>
<td>Retinal Detachment</td>
<td>Flashes &amp; Floaters</td>
<td>+ Vision change</td>
<td>Flashes &amp; Floaters</td>
</tr>
<tr>
<td>Sudden Vision Loss</td>
<td>Binocular Vision Loss</td>
<td>Vision change</td>
<td>Flashes &amp; Floaters</td>
</tr>
<tr>
<td>Severe Pain</td>
<td>Unilateral Pain</td>
<td>Pain + Vision change</td>
<td>Flashes &amp; Floaters</td>
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Central Retinal Artery Oclusion

- Disruption of vascular perfusion in the central retinal artery leading to global retinal ischemia.

- Mainly due to Emboli or Thrombus at the lamina cribrosa.

Central Retinal Artery Oclusion

- Cholesterol Emboli (HHP): Yellow-Orange (Refractile) often originating from carotid arteries. Visible only 20% of the time.

- Calcific Emboli: White & usually cause a distal retinal infarction. Typically arise from cardiac valves
Artery Occlusions

- Platelet Fibrin Emboli: Dull white and most commonly arise from a carotid thrombosis.
- Talc, Tumor, Septic, and Fat emboli are less common.

Central Retinal Artery Occlusion

- Symptoms: Unilateral, painless, acute vision loss
- 94% CF to LP vision
- +/- Amaurosis Fugax
- If VA is LP or worse, strongly consider Ophthalmic Artery occlusion

Central Retinal Artery Occlusion

- Signs: Superficial opacification or whitening of the retina in the posterior pole with a cherry-red spot in the center of the macula (which may be subtle)
- Absence of a cherry red spot likely indicated involvement of the ophthalmic artery.

Central Retinal Artery Occlusion

- Additional signs:
  - Marked RAPD
  - Boxcarring or Segmentation of the blood vessels.

Retinal Tolerance Time to Ischemia


38 rhesus monkeys with HTN, atherosclerosis with clamping of CRA at entry into nerve:
- Occlusion < 97 min: no retinal damage
- Occlusion 105 min - 240 min: variable degree of damage
- Occlusion > 240 min: total optic nerve atrophy and nerve fiber damage
Timing is Critical

<table>
<thead>
<tr>
<th>Initiation of Tx for CRAO</th>
<th>Prognosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Within 90 Minutes</td>
<td>Fair (Complete)</td>
</tr>
<tr>
<td>Within 4 Hours</td>
<td>Fair (Partial)</td>
</tr>
<tr>
<td>Within 6 Hours</td>
<td>Guarded</td>
</tr>
<tr>
<td>After 6 Hours</td>
<td>Poor</td>
</tr>
</tbody>
</table>

- Treat IMMEDIATELY before starting work-up:
- Digital Ocular Massage
- Systemic acetazolamide (500mg)
- Topical B-Blocker q15min X2
- Breathing into paper bag (respiratory acidosis) - vasodilation
- Anterior Chamber Paracentesis

Digital Massage for CRAO

Before Trans-lumenal Yag Embolectomy

After Translumenal Yag Embolectomy (TLE)
Risk Factors for CRAO

- DM (33%)
- HTN (67%)
- CAD (25%)
- CVA
- TIA's
- Higher Smoking Prevelance
- 30% patients had ICA stenosis (> 50%)
- 70% patients had ICA plaques
- 50% patients had abnormal echo with a source of embolus
- 30% patients had ICA stenosis (> 50%)
- 70% patients had ICA plaques
- 50% patients had abnormal echo with a source of embolus

CRAO Work-Up

Stat ESR, CRP, and Platelets in any patient 50 years old or older:
- Check Blood Pressure
- CBC with Diff
- Fasting Glucose
- Glycosylated hemoglobin
- PT / PTT

With systemic risk factors:
- Lipid Profile
- ANA, Rheumatoid Factor
- FTA-ABS
- Serum Protein Electrophoresis
- Hemoglobin Electrophoresis
- Antiphospholipid Antibodies

CRAO Work-Up

- Refer to internist for a complete work-up:
  - Carotid Doppler Ultrasound
  - Electrocardiography (EKG)
  - Echocardiography
  - Possibly, Holter Monitoring
  - Co-manage with retinal specialist
    - Consider IVFA & ERG to confirm diagnosis
    - NVI / NVD / NVE occur in up to 20% of patients with a mean onset of 4 weeks. PRP & Anti-VEGF treatments may be warranted.

Case #1 ~ Ocular Chemical Burn

NVI / NVD / NVE occur in up to 20% of patients with a mean onset of 4 weeks. PRP & Anti-VEGF treatments may be warranted.
Case #1

- You are escorting a patient to the front desk to check out. While there, you overhear your receptionist tell a patient to “bring him in immediately…we will work him right in!”
- When you inquire as to what the urgency is, your receptionist relays to you that your established patient’s husband accidentally got splashed in the eye with a chemical he uses for fertilizing his crops. He is in severe pain and can barely open his eye(s).

Case #1 ~ Ocular Chemical Burn

- True Ocular Emergency!!!
- Whether by gas, liquid, or solid, an acid or alkaline burn can cause irreversible damage to the eye and adnexa if urgent action is not taken.

Case #1

- Was this the right decision?
- Did your receptionist gather enough information?
- Are you prepared to handle such a problem?
- Where do we go from here?

Case #1 ~ Ocular Chemical Burn

- More severe
- Penetrate rapidly
- They combine with all proteinaceous tissues, denaturing and dissolving collagen, resulting in destruction of cells and necrosis of the tissues.
- They render the iris and adnexa useless, which enhances chances of vision loss
- They resist removal of conjunctiva or lids
- “Caustic fumes” the conjunctiva appears cloudy white and opaque.
Johns Hopkins University Epidemiologic Study
JAMA Ophthalmology (August 2016)

• Nationwide emergency room visits over 4 full years (2010-2013)
• Total OCB: 144,149
• Highest incidence rates for individual years:
  • 1 & 2 year olds; then
  • 24 year old
Case #1 ~ Did the Receptionist Make The Right Decision?

Telephone Triage

1. Upon hearing of a chemical splash injury, make sure that the preliminary irrigation process begins on site before the patient seeks care.

2. If the chemical splash occurred outside of the workplace, remind the caller that the shower or an outdoor hose is an adequate option.

3. Attempt to determine time lapse between burn event and when irrigation started.

4. Attempt to determine the type of chemical that entered the eye(s).

5. Attempt to determine if the patient is wearing contact lenses. Irrigation should not stop in an effort to remove contact lenses.

6. Irrigation should take place for a minimum of 20 to 30 minutes before the patient is brought to the office or emergency room.

7. When the patient is ready to make the trip to the ER or office, remind them to bring the container that held the offending chemical. Important information may be obtained from the labeling.

8. If the injury occurred in the workplace, ask the patient to bring the MSDS (material safety data sheet) if available.

9. If the injury occurred where there is no or limited access to water for irrigation, refer them to the nearest emergency room or your office, whichever is closer.

10. Assist with dispatching emergency services as needed.

Special Situation

If contamination with metallic lithium (i.e., lithium ion batteries), sodium, potassium, or magnesium has occurred, irrigation with water can result in a chemical reaction that causes burns to worsen. In these situations, the area should be covered with mineral oil and the metallic pieces should be removed with forceps and placed in mineral oil. If forceps are not available, soak the area with mineral oil and cover it with gauze soaked in mineral oil.
Nasal Canullar Irrigation

Clinical Examination
- Re-check pH
- Case History
- Chief Complaint:
  - Pain, photophobia, epiphora, blepharospasm, reduced vision
  - External & Slit Lamp Examination
  - IOP assessment

Treatment Options ~ Grade 1 Chemical Burn
- Prednisolone acetate 1% QID
- Topical antibiotic ung (e.g., erythromycin) QHS to QID
- Preservative-free artificial tears PRN
- Cycloplegic for pain PRN

Treatment Options ~ Grade 2 Chemical Burn
- Prednisolone acetate 1% Q1H while awake with rapid taper between days 10-14
- Long-acting cycloplegic (i.e., atropine 1%)
- Oral pain medication PRN
- IOP Lowering agent(s) PRN

- Oral doxycycline to reduce risk of corneal melting through MMP inhibition
- Oral vitamin C (1,000mg to 2,000mg) QID
- Sodium ascorbate drops (10%) Q1H while awake
- Debridement of necrotic tissue, using tissue adhesive as needed
Treatment Options ~ Grade 3 Chemical Burn

- Same as Grades II & III
- For significant necrosis, a tenonplasty can help re-establish limbal vascularity
- Stem cell transplantation
- Penetrating keratoplasty
- Keratoprosthesis

Treatment Options ~ Grade 4 Chemical Burn

Case #1 ~ Pearls

- Take the time to educate your receptionist(s) and technicians on how to telephone triage the ocular chemical burn patient.
- Make sure you have pH strips on hand that are not expired.
- Keep a triage checklist posted at the front desk.
- Normal pH 24-48 hours before does not mean pH will be normal today.
- Do not forget to check IOP, especially in the presence of an Alkaline burn.
Case #2

- An 84 year old woman develops a constant head and neck ache. She calls her HMO internist, who told her to take Motrin. Although she was a woman not given to complaining, she called the doctor again when the pain persisted. The internist told her that neck pain from arthritis is common at her age and not to worry, but the patient insisted on coming in to the doctor's office.

Case #2

- The internist nurse practitioner took her blood pressure and gave her Relafin without drawing any tests. 2 weeks later she went blind in one eye. She was referred to the HMO ophthalmologist, who then referred her to a neuro-ophthalmologist. Unfortunately, 24 hours after the HMO ophthalmologist saw her, she went blind in the other eye.

Case #2

- It was later determined that she possessed many of the “classic” symptoms of GCA. The failure to recognize temporal arteritis had turned a functioning, independent woman into a blind person confined to home.

Giant Cell Arteritis (GCA)
Temporal Arteritis (GCA)

- HA (New onset, localized, progressive) is the major feature in 65-90% of cases. Otherwise, patients may be vague with their symptoms.
- Age of Onset: >50 (incident increases w/ age)
- Incidence: 20 per 100,000; Prev: 200 / 100,000; Higher incidence in Caucasians and those of Scandinavian or Northern / Eastern European decent. Northern US.
- 4:1 female > male

Unfortunately, if they are sitting in your chair, they most likely have already experienced:
Sudden, painless, non-progressive visual loss (count fingers) in at least one eye.

• Decreased VA => a “True” ophthalmic emergency

Systemic History

- 50% of GCA patients begin with symptoms of anorexia, fever, malaise, myalgia, night sweats, and weight loss.
- Prodromal symptoms may occur for a few days or may even stretch out to weeks.
- Hallmark symptom of GCA is its new-onset localized headache.
- Localized to the temporal or occipital area, and may be occasionally may be diffuse or bilateral.
- 5-10% are ASYMPTOMATIC

Signs & Symptoms:
- Superficial Temporal Artery swelling, erythema, tenderness & pulselessness.
- Scalp tenderness, jaw claudication (50% of patients), & pain in the throat, neck, teeth, gums, or eye.

AAION

- + APD
- Pale, swollen “chock white” disc, + / - flame hemorrhage
- Altitudinal VF defect
- Occasionally:
  - Vaso-occlusive disease
  - Neuro-ophthalmic disease
r3  Do search to see about auscultation of temporal artery with stethoscope.
rbm, 6/4/2008
Sequelae of GCA

Temporal Arteritis (Dx)

- Lab Tests:
  - CBC w/ Differential
  - Anemia
  - Elevated platelet count
  - ESR (Westergren): 50-95 mm/h
  - Men: (age / 2) + 10
  - Women: (age + 10 / 2)
  - >40 is suspicious
  - C-Reactive Protein (99%)
  - Fibrinogen
  - Temporal Artery Biopsy

Procedures

- Superficial temporal artery biopsy (TAB) shows focal granulomatous arteritis, often with giant cells with skip areas within normal arterial walls.

Histopathology

- Reveals inflammatory infiltrate surrounding a fragmented internal elastic lamina within the media of an arterial wall.
- Infiltrate consists predominantly of mononuclear cells with giant cell formation.
- Mechanism is believed to be related to dysfunction of cellular immunity, but etiology is unknown.
What is the normal range for Fibrinogen?

Look-up Color Duplex Ultrasonography of Temporal Arteries.

rbm, 6/4/2008
False Negatives

- Not all patients with GCA will have abnormal labs. 15-30% with + TAB have a normal ESR.
- TAB has a false negative rate (Skip lesions) of 5-9%.

The American College of Rheumatology 5 point scoring system

- Age more than 50 years.
- A Westergren ESR greater than 50.
- Temporal artery tenderness or abnormality on exam.
- New onset headache.
- Positive temporal artery biopsy

Treatment

- The universally accepted treatment for GCA is high-dose corticosteroid therapy.
- Goals of treatment are to reverse the disease and to prevent further progression. This is of utmost importance especially in the ophthalmic arteries to prevent blindness.

- First-line acute therapy without visual signs / symptoms => oral prednisone 40-60mg of prednisone ~or~ 1-2mg/kg/day.
- In the presence of visual or neurological symptoms => 80 to 100mg/d.
- Ranitidine 150mg BID PO (histamine type 2 receptor blocker) – or – Prevacid (Proton Pump Inhibitor) for GI protection.
- Fosamax Plus D – 10mg daily in the prevention of steroid induced osteoporosis.
- Baby ASA (81mg) – decreases cranial ischemic complications.

- Preferred treatment for AAION:
  - Pulse Therapy: 5 day course of IV Methylprednisolone induction therapy of 15mg/kg/d (about 1000mg/d).
  - 22 fold increased chance of improving visual acuity if started within the first day.
  - Less Cumulative Dose of Steroids overall compared to oral Tx.
  - May reduce remission rates.

- Prognosis is poor if treatment is initiated > 48 hours.
- 30% of patients will continue to show a decline in vision despite aggressive steroid therapy.

- Once the signs of clinical inflammation are suppressed and the ESR is maintained at a low level, corticosteroids may be tapered slowly.
- No agreement exists as the length of treatment with corticosteroids for GCA.
- It may be reasonable to maintain the patient on treatment for 2 years to lessen the chances for relapses, although relapses have been reported.
Case #3

- A 31 year old caucasion male presented for follow-up after being diagnosed with preseptal cellulitis OS. He claims compliance with his treatment regimen (Bactrim DS BID PO), but feels that his left eyelid has become more swollen and tender. He feels his vision has remained unchanged.

Case #3

- Entrance BCVA was 20/20 each eye. While EOMs were full OU, the patient reported a pulling sensation OS when looking superior temporal. The left eye appeared slightly proptotic. Retropulsion assessment was inconclusive. Confrontational fields and pupil testing were unremarkable. Dilated fundoscopic exam noted no apparent pathology of the posterior pole. Optic nerves were healthy in appearance with CD ratios of 0.3/0.3 OD, OS.

Case #3

- So despite being treated with oral antibiotics, his condition appears to be worsening. Could this be a case of antibiotic resistance? Is this pulling sensation suggestive of early stage orbital cellulitis? What is the next step in management given what we know?
Periorbital and Orbital Cellulitis

Pathogenesis

- Sinusitis
- Extension of external ocular infection (ie hordeolum, dacryocystitis/dacroadenitis)
- Dental abscess
- Superficial break in the skin (ie infected bug bite, acne, eczema, periocular surgery or direct penetrating trauma)
- Hematogenous spread

Organisms

**Children**
- Haemophilus influenza type b (before Hib vaccine in 1985)

**Adults**
- Gram (+)
  - Staphylococcus aureus (including MRSA)
  - S. epidermidis
  - Streptococcus pyogenes
  - Enterococcus

**Gram (-)**
- Pseudomonas (especially trauma)
- Klebsiella

**Fungal**
- Mucormycosis, Aspergillus species

Epidemiology

- Increased incidence during the winter due to the increased incidence of sinusitis
- No ethnic preferences
- Blindness occurs in up to 11% of cases
- In children, twice as common in males
- More common in children than adults: mean age 7-12 years old
History

- Onset rapid or slow?
- Pain or tenderness to palpation?
- Recent fever, chills, rash or other systemic symptoms?
- Recent contact with other infected persons?
- Recent surgery (including eye or dental), hospitalization or trauma?
- Stiff neck or change in mental status?
- History of cancer, diabetes, HIV, organ transplantation, pulmonary or renal disease?

Clinical Signs and Symptoms

- Unilateral erythema of eyelid
- Swelling of eyelid
- Warmth of eyelid
- Tenderness of eyelid
- Blurred vision
- Ophthalmoplegia
- Proptosis
- Chemosis

Physical Exam

- Observe for degree of ocular swelling
- Assess extraocular movement
- Evaluate for foreign body
- Assess visual acuity

Preseptal Cellulitis

Axial contrast-enhanced CT image of the orbit shows soft tissue thickening of the right preseptal region (between arrows). The retroorbital fat is normal (arrowheads).
Orbital Cellulitis due to Ethmoid Sinusitis

Orbital cellulitis secondary to ethmoid sinusitis. Axial contrast-enhanced fat-suppressed T1-weighted MR image obtained in a 40-year-old man demonstrates right exophthalmos and heterogeneous enhancement of orbital (arrows) and periorbital (arrowhead) fat.

Subperiosteal Abscess due to Ethmoid Sinusitis

Imaging: Indications
- Eyelid edema that makes a complete examination impossible
- Presence of CNS involvement (ie, seizures, focal neurologic deficits, or altered mental status)
- Deteriorated visual acuity or color vision
- Proptosis
- Ophthalmoplegia
- Clinical worsening or no improvement after 24-48 hours

Differential Diagnosis
- Allergic reaction
- Edema from hypoproteinemia
- Orbital wall infection
- Subperiosteal hematoma
- Orbital pseudotumor
- Orbital myositis
- Retinoblastoma
- Metastatic carcinoma
- Exophthalmos secondary to thyroid dysfunction

Admission Criteria
- Patients with orbital cellulitis presenting with:
  - Eyelid edema
  - Diplopia
  - Reduced visual acuity
  - Abnormal light reflection
  - Ophthalmoplegia
  - Proptosis
- Appears toxic
- Eye exam is unable to be completely performed
- Signs of CNS involvement:
  - Lethargy
  - Vomiting
  - Seizures
  - Headache
  - Cranial nerve deficits
Management

- Depends on the patient's appearance, ability to take oral medications, compliance and clinical progression of the disease.
- Empiric antibiotics should cover Staphylococcus and Streptococcus species, particularly MRSA.
- Treat for 7-10 days for periorbital cellulitis.
- Treat for 10-14 days for orbital cellulitis.
- If no improvement in 24-48 hours consider consulting Infectious Disease, ENT and/or neurosurgery.

Management

- Obtain blood culture in younger patients or those that appear systemically ill.
- Culture ocular discharge.
- Obtain orbital, epidural abscess or sinus fluid if patient requires surgery.

Surgical Management

- Consider if:
  - > 9 years old.
  - Frontal sinusitis.
  - Non medial location of the subperiosteal abscess.
  - Large subperiosteal abscess.
  - Presence of gas in the abscess on CT suggesting an anaerobic etiology.
  - Recurrent episode of subperiosteal abscess.
  - Nasal polyps which suggest chronic sinusitis.
  - Evidence of acute optic neuropathy.
  - Dental infection (likely an anaerobic infection).

Summary

- Orbital cellulitis is an emergency that requires prompt diagnosis and evaluation.
- Periorbital cellulitis and orbital cellulitis have distinct differences that can be elicited by careful history and physical examination.
- If the physical exam cannot be fully completed for any reason, radiologic imaging is required.
- Patients with systemic illness or evidence of orbital cellulitis or neurologic involvement require inpatient admission.
- Improvement should occur within 24-48 hours with antibiotics.
A 36-year-old white male presents to your office with a chief complaint of intermittent pain on the right side of his head and orbital area along with redness and foreign body sensation in his right eye. He works in construction and is concerned that he may have gotten something in his eye.

External examination shows a mild ptosis (1.5mm) with minimal palpebral injection and no lid edema in the right eye. No foreign body of the cornea or bulbar conjunctiva is noted on a slit-lamp examination; nor is any evident on lid eversion. The cornea is clear, the anterior chamber is well formed and quiet. His unaided visual acuity is 20/20 each eye.

Pupil testing reveals anisocoria greatest under dim illumination and most noticeable during the first few seconds after the lights were turned down.

The right pupil showed a delay in dilation consistent with “dilation lag” found in Horner’s syndrome.

Anhidrosis is not evident and not reported by the patient.

- Iris sphincter
- Iris dilator
- Parasympathetic pathway
- Sympathetic pathway
Horner's Syndrome:
Clinical Features

A. Moderate Ptosis (2-3mm) due to paralysis of Muller's muscle
B. “Upside Down Ptosis” - Mild elevation of the lower lid due to paralysis of the smooth muscle attached to the inferior tarsal plate.
C. Apparent Enophthalmos due to A & B above
D. + Dilation Lag (classic finding)
E. Decreased IOP on affected side

F. Miosis, more noticeable in dim illumination. Note: Pupil reacts to light and near are normal.
G. Anhydrosis on Ipsilateral side of face if lesion is below the Superior Cervical Ganglion => Not a 3rd order neuron.
H. Increase in Amplitude of Accommodation due to unopposed action of the parasympathetic.
Horner's Syndrome: Clinical Features…

Lastly

Horner's: Localization of Lesion

- 4% Cocaine
  - + Test => Anisocoria will increase
- Hydroxyamphetamine (Paradrine 1%)
  - Preganglionic lesion => YES dilation
  - Postganglionic lesion => No dilation
- If suspect pre-ganglionic lesion => Chest CT or MRI.

Horner's

- Testing: 4% cocaine will dilate a normal pupil by blocking the re-uptake of epinephrine but will not dilate the Horner’s pupil. Shelf life of only six months if preserved and cost of $90
- More practical: 1% Iopidine will dilate a Horner’s pupil after 30-45 minutes but will not dilate a normal pupil. 0.5% works also
- 1% hydroxyamphetamine will dilate a first or second order Horner’s but not a third by releasing NE from postganglionic synapses. Must wait one hour to check and need 72 hour washout if cocaine was used
- Ptosis only patients will get lid elevation with Naphazoline. Little pupillary mydriasis.

Horner's Secondary to Pancoast Tumor

Most Common Causes of Horner's Syndrome
Flashes & Floaters ~ How urgent is URGENCY?

Case #5

- It's Saturday night and you're getting ready to sit down and watch a movie with your family. The answering service calls with a patient on the other line. They tell you that the patient is complaining of an increase in floaters after having a single bright flash of light. How many times have we all gotten this phone call, right?! How many times have we gone into the office after hours or on weekends, only to dilate the patient to diagnose an uncomplicated PVD?

Case #5

- What do you tell the answering service? Do you simply agree to forego the movie and see the patient at your office in an hour? Do you ask them to call back if the floaters worsen before an early morning appointment on Monday? Do you ask them to be NPO after midnight and see them the next morning "just in case"? Or, do you need more information?

59 year Retrospective Meta-analysis
Hollands and Colleagues

- Found and reviewed 17 relevant studies looking at the association between flashes & floaters, PVD, and likelihood of retinal tear.
- If patients report flashes & floaters: 14% had a retinal break or tear.
- Flashes but no floaters: 13.7%
- Floaters but no flashes: 13.5%
- Add in "Subjective Visual Complaints" => Prevalence increases to 45%
Case #5

- You have the answering service connect you directly with the patient. You determine that the patient is a 50 yo high myope who had an onset of flashes & floaters 10 days earlier. She was seen by her optometrist same day and diagnosed with an uncomplicated PVD.

- She was educated on the warning signs of RD and instructed to call the office immediately should there be any worsening or change in her symptoms.

Case #5

- Hence her call. She reports an increase in floaters and feels that the lower part of her vision has become affected. Based on her symptoms, you ask her to meet you at your office this evening. Surprisingly, she asks if this could wait until tomorrow. She lives by herself in a rural town 45 – 50 minutes away. She does not feel comfortable driving herself and she is not sure she can manage a ride this evening. You explain to the patient that you are concerned that she may have a loose or torn retina and that the longer we wait in addressing it, the greater the risk of macular involvement and the potential for permanent vision loss. She said she would make some calls and call you back.

59 year Retrospective Meta-analysis

Hollands and Colleagues

- Patients previously diagnosed with an UNCOMPPLICATED PVD have a 3.4% risk of developing a retinal tear within 6 weeks.

- Predictive symptoms for retinal tear in this subset of patients:
  - Sudden increase in floaters (>10 floaters), and / or
  - Subjective Vision Loss

- So do all retinal tears lead to detachment if undiagnosed?

Do all Retinal Tears lead to Detachment?

- NO

- Shea & Colleagues: 33-50% of all tears lead to detachment.

- 81 yo asymptomatic patient with self-sealing supero-temporal Horseshoe retinal tear, OS

Mechanism of RRD

- Size of retinal break
- Location of retinal break
- Residual vitreous traction
- Degree of vitreal syneresis (liquefied vitreous)
Case #5

- Your patient calls you back and indicates she has found a ride and will meet you at your office in about an hour. Soon after she arrives, you determine that she has preserved central vision (BCVA 20/20- each eye), but has reduced confrontation fields inferiorly and slight asymmetry in her intra-ocular pressures (12mmHg OD, 16mmHg OS).

- After she is dilated, slit-lamp exam of the vitreous increases your suspicion of retinal detachment after you notice some pigmentary “dusting”.

- Indirect ophthalmoscopy revealed a large, superiorly located retinal break with a secondary “macula-on” rhegmatogenous retinal detachment (Picture 2).

- After communicating your findings to the patient, you stress the importance of having this detachment “addressed” urgently. But what does that mean, really? Should this be surgically addressed that same evening? Is it OK to order NPO (no food or drink) after midnight with the goal of evaluating and surgically repairing the next day? Could it even wait until Monday so the main surgical team with an expertise in retinal surgery could be available to insure a more seamless procedure?

59 year Retrospective Meta-analysis
Hollands and Colleagues

- 12 Studies related findings on ocular examination to the presence of retinal tear. The presence of two slit-lamp findings, together or in isolation, was highly suggestive of retinal tear:
  - Vitreous Heme => 62% probability of retinal tear
  - Pigment Dusting => 88% probability of retinal tear.

Timing of Foveal Sparing RRD Surgical Repair: The Great Debate

Wynhoff et al:
199 patients underwent scleral buckling for Mac-On detachments:
52% were surgically repaired within 24 hours,
27% within 1-3 days,
18% within 3-7 days,
Rest: > 1 week

Erlitch et al:
14 patients had small gauge vitrectomy for Mac-On detachments:
Timing ranged from 1 to 5 days

Lai et al:
66 eyes that had scleral buckle, vitrectomy, or both. Range of 0.8 hours to 5.5 days.
Timing of Foveal Sparing RRD Surgical Repair:
The Great Debate

- Prospective Analysis: Ho et al.
  - 82 Mac-On RRD’s
  - 87% showed no progression from presentation to surgery
  - Remaining 13% showed an average rate of progression of 1.8 disc diameters per day!

R/B Ratio of delaying surgery

- Pros
  - Optimal surgical conditions
  - Most experienced staff
  - Less cost for patient (surgery performed after hours costs 25% more)

- Cons
  - Every eye is different. Some have progressed to Mac-off in as quickly as 4-8 hours.
  - What if there is further delay from unforeseen events?

Case #6: Is HELP on the way?

- A 60-year-old African American male presented complaining of a red, irritated right eye with associated brow ache, and he felt his vision had decreased slightly in that eye.

Slit-lamp examination revealed a superiorly located opalescent bleb surrounded by 3+ conjunctival injection. There appeared to have a mucopurulent infiltrate within the bleb in the right eye. The anterior chamber was deep, showing trace cell and flare, right eye.

Intraocular pressure (IOP) was 5mm Hg OD and 9mm Hg OS. NaFl strip application revealed a subtle leak from the infero-lateral edge of the bleb in the right eye. Dilated examination revealed advanced cupping in both eyes. The periphery was intact without choroidal effusions. Vitreous cells were not evident on slit-lamp exam or under binocular indirect ophthalmoscopy. B-scan ultrasound was acoustically normal in both eyes.
Blebitis Acronyms to Remember

- **HELP**
  - **H** = Hypotony
  - **E** = Endophthalmitis
  - **L** = Leak
  - **P** = Pain

- **RSVP**
  - **R** = Redness
  - **S** = Sensitivity to Light
  - **V** = Vision Loss
  - **P** = Pain

Managing Late Bleb Leaks

- **Conservative Approach**
  - Supress Aqueous Production
  - Antibiotic Prophylaxis
  - Keep the eye lubricated
  - Protection from trauma or mechanical rubbing

- **Surgical Revision**
  - Needling the Bleb
  - Autologous Blood Patching
  - Compression Suturing
  - Amnion Membrane Grafting

Seidel Test

Christopher Teng, MD

Large Diameter BCL 4 Late Bleb Leaks

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<thead>
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Differential DX of Bleb related infections

**Blebitis**

- Defined as mucopurulent infiltrate within a bleb
- If no anterior chamber response => Aggressive Topical (PQ+QH) or Fortified DMSO + Oral (Moxifloxacin)
- Monitoring Daily
- Timely treatment Prognostic Good

**Blebitis related Endophthalmitis**

- Defined as hypopyon, cells in the anterior vitreous, or hypopyon posterior corneal or vitreous opacities
- B-Scan may confirm
- Refer to retinal and/or glaucoma specialist for infection control and bleb revision
- Time Treatment Prognosis: expect a 4 line drop in acuity with potential for CF or LP vision
Topic #7: Third Nerve Palsy

Parasympathetic Pathway

- Four neuron arc:
  - Retina to the pretectal nucleus in the midbrain (1)
  - Pretectal nucleus to the EW nucleus (2)
  - EW nucleus to the ciliary ganglion (3)
  - Ciliary ganglion to the iris sphincter with short ciliary nerves (4)

Isolated Third N. Palsy w/ Pupil Involvement

- Sudden Onset Unilateral Ptosis with Eye or Head Pain
- Acuity is Typically Unaffected unless damage is in Superior Orbital Fissure
- Eye is in non-comitant exotropic & hypotropic position (“down & out”)
Isolated Third N. Palsy w/ Pupil Involvement

- Posterior Communicating Artery Aneurysm (Most Common)
- Tumor or Trauma
- HZO
- Leukemia
- Venous lnfiltration Syndrome
- Space Occupying Lesion
- Ischemic Vascular Disease
- Uncal Herniation Syndrome
- Subdural Hematoma
- Pituitary Apoplexy
- Ischemic Vascular Disease (Rare)

Pupil sparing / Pupil involving

- Rule of thumb: Pupil sparing third nerve palsies tend to be ischemic while those involving the pupil tend to be due to aneurysms or tumors
- Not a firm rule
- Pupil sparing may become pupil involving so follow very closely

Pupil involving vs. pupil sparing

Third Nerve Management

- Immediate Gad enhanced MRI / MRA if any question of aneurysmal involvement. Patient may complain of a severe headache and will often have other neurological signs
- If patient is diabetic or hypertensive and the pupil is not involved they can be followed closely without imaging studies

Third Nerve Management

- Patient education and reassurance a must
- Diplopia relief with patching
- Most ischemic palsies resolve over several months
Case #8

Patient History

- 68 year old white female.
- Ocular History:
- Medical history:
  - Diabetes
  - Renal Problems.

Recent Exam Findings

- July 2004
  - VA- 20/25, OU.
  - Cup-to-disc: 4/4, OU.
- July 2005
  - VA- 20/25, OD, 20/60, OS.
  - Cup-to-disc: 6/6, OD.
  - 9/9, OS.

Present Exam Findings

- VA- OD- 20/25
  - OS- NLP
- PERRL + APD, OS.
- TA- 16, OD
  - 67, OS

Observations, OS

Neovascular Glaucoma

- Elevation of IOP.
- Painful red eye.
- Closure of anterior chamber angle from fibrovascular membrane formation.
Causes

- Central retinal vein & artery occlusion.
  40-60% of NVG cases.
- Diabetic retinopathy.
- Carotid artery occlusive disease.
- Chronic retinal detachments
- Usually occurs within 90 days of antecedent vascular occlusion.

Signs/Symptoms

- Acute onset of redness, pain, and blurred vision.
- Circumciliary injection.
- Corneal edema.
- Deep anterior chamber with moderate flare.
- NV/NVA.

Pathophysiology

- Stimulus = Lack of Oxygen.
- Hypoxic retinal tissue results in the release of vasoproliferative factors, i.e. VEGF.
- VEGF acts upon endothelial cells of viable capillaries to stimulate the formation of a new vessels.
- Once released, the angiogenic factors diffuse through the vitreous and posterior chamber into the aqueous and the anterior segment.

Pathophysiology, II

- Vasogenic factors interact with vascular structures where the greatest aqueous-tissue contact occurs.
- The result is new vessel growth at the pupillary border and iris surface and over the iris angle.
- Ultimately leading to formation of fibrovascular membranes.

Pathophysiology, III

- The neovascularization, along with its fibrovascular support membrane, acts to both physically block the angle and bridge the angle.
- The vessels pull the iris and come into apposition, thus blocking the trabecular meshwerk.

Stage I, Early

- Small, dilated capillaries at pupillary margin.
- Vessel arborization onto iris near pupil.
- Normal IOP.
Stage II, Mid-Phase

- Involvement of anterior chamber angle.
- Radial vessel progression.
- Hyphema.
- Increase in IOP.

Stage III, Late

- Contraction of the fibrovascular membrane.
- 360° angle closure.
- Ectropion uvea.
- Significant anterior chamber reaction.

Management

- Medically treating neovascular glaucoma is challenging at best.
- Medical consult to rule out systemic disease.
- Duplex/Doppler scans to rule out carotid occlusive disease.

Medical Management

- If there is any degree of inflammation and ocular pain, prescribe a topical cycloplegic such as atropine 1% b.i.d. as well as a topical steroid such as Pred Forte.

IOP Control

- Medical therapy with topical β-adrenergic antagonists, a-2 agonists, and topical or oral carbonic inhibitors lower IOP. However, chronic medical therapy is not indicated for neovascular glaucoma.
- Aqueous suppressants may be used in order to temporarily reduce IOP. However, chronic medical therapy is not indicated for neovascular glaucoma.
- Aqueous suppressants will temporize IOP and angle closure will continue.

Medical Management, II

- Ultimate management of NVG involves eradication of the vessels with PRP or cryo.
- Goal: destroy ischemic retina, minimize oxygen demand of the eye, and reduce the amount of VEGF being released.
- If a significant amount of the angle is in permanent synechial closure, filtering surgery must then be employed.
However…

• What if the patient is, like ours, blind?
• The primary goal of treatment in this stage is pain control.
• For blind, painful eyes with uncontrollable IOP, options include continued medical therapy, cyclodestruction, retrobulbar alcohol injection, or enucleation.

But…

• Our patient was also not in pain.
• Plan of action:
  Retinal consult.
  Possible PRP to save cornea from decompensation.

Future Possibilities

• Anti-VEGF therapy.
• VEGF appears to produce its effect partly by being proinflammatory, leading to leukocyte adhesion and inflammation.
• VEGF can induce injury to the endothelium, cause fenestrations in endothelial cells, and cause breakdown of tight junctions.

Pointers…

• Retinal artery occlusions develop NVG in only 17 percent of cases and typically within four weeks post-occlusion.
• Miotics are contraindicated in any case where there is active inflammation. Prostaglandin analogs should likewise be avoided.
An 18-year-old male with a right eye injury presented with entrance acuity at the level of hand motion (HM) and an intraocular pressure (IOP) of 17mm Hg. An anterior segment examination revealed right upper lid edema and bruising.

A slit-lamp examination confirmed corneal edema with mild folds centrally, a subconjunctival hemorrhage with chemosis and 3+ cell and flare with a 2mm hyphema in an adequately formed anterior chamber.

The cornea and conjunctiva were seidel negative. The right pupil was 5mm with poor reactivity. The lens appeared centered, but it was difficult to assess due to poor clarity.

Funduscopic exam was limited due to severe vitreous hemorrhage. A B-scan ultrasound did not suggest retinal detachment. The patient was referred to our staff retinal specialist the next day. A biometry was repeated and, once again, did not suggest retinal detachment.

Risk Factors for Open Globe:
- Penetrating Lid Injury
- Hypotony + Hyphema
- Positive Seidel
- Peaked Pupil and/or APD
- Shallowed or absent anterior chamber
- 360 degree bullous sub-conjunctival hemorrhage
- Lens dislocation
- Iridealalysis
Case #9: War Paint

- If there is any question regarding the integrity of the globe, consider a CT scan of the eye and orbit, or refer.

Case #9: War Paint

- ASTM International investigation into ocular paintball injuries between 2001 & 2010 (11,827 eye injuries):
  - 3.8% (0-9yo)
  - 68.1% (10-19 yo)
  - 18.1% (20-29 yo)
  - 10% (30yo+)

- Locale:
  - Home: 27.6%
  - Public Property: 5.0%
  - Recreation or Sports Venue: 22.3%
  - Street or Highway: 0.2%
  - Not recorded: 44.9%

Eye Injuries From Airsoft Guns Up Over 500 Percent

- 3,000 children were treated in U.S. emergency departments in 2012 for eye injuries related to paintball guns, airsoft guns, BB guns and pellet guns - but the big increase was in airsoft guns.

- Investigators from the Stanford University School of Medicine found that the rates for eye injuries from non-bullet guns increased by 511% between 2010 and 2012, reaching 8.4 per million children and the rise was almost exclusively due to air guns related injuries, which parallels their growth in popularity.

Case #10: Darwineyes