Ocular Urgencies & Emergencies ~ Are You Prepared?

Richard B. Mangan, OD FAAO
Scott Hauswirth, OD FAAO
Assistant Professors, Dept of Ophthalmology
University of Colorado School of Medicine

eyeam4uk@gmail.com

Subject Line: The Mangan Awards
True Ocular Urgencies or Emergencies

- Lid Laceration
- Orbital Cellulitis
- Retinal Detachment
- Chemical Burn
- Microbial Keratitis
- Acute Angle Closure Glaucoma
- Retinal Vein Occlusion
- Central Retinal Artery Occlusion
- Retina Detachment
- Endophthalmitis
- Giant Cell Arteritis
- Cavernous Sinus Thrombosis
- Horner’s Syndrome
- Third Nerve Palsy
- Hypopyon (Hypopyon Histoematoma)

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:

- Immediate: Within 1-2 hours
- Urgent: Within 24 hours
- Soon: Within 1 week
- Elective: Within 6 weeks to months

I-USE
- 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>&lt; 24 hours</th>
<th>Within 1-2 weeks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chemical Burn</td>
<td>Corneal Abrasion</td>
<td>Recent Onset Diplopia</td>
<td>Dry, Itchy, Watering</td>
</tr>
<tr>
<td>Acute Ocular Trauma</td>
<td>Foreign Body + Vision change</td>
<td>Recent Onset Psychotic</td>
<td>Broken Glasses</td>
</tr>
<tr>
<td>Sudden Vision Loss</td>
<td>Floaters &amp; Flashes</td>
<td>Distance Vision for &gt; 2 weeks</td>
<td>Broken Glasses</td>
</tr>
<tr>
<td>Severe Pain</td>
<td>Monocular Patient</td>
<td>Photophobia</td>
<td>Mild Blepharitis</td>
</tr>
<tr>
<td>Broken Glass + Vomiting</td>
<td>Post-operative patients</td>
<td>Mild Pain &gt; 2-3 Days</td>
<td>Lumps &amp; bumps</td>
</tr>
</tbody>
</table>

Central Retinal Artery Occlusion

- Disruption of vascular perfusion in the central retinal artery leading to global retinal ischemia.
- Mainly due to Emboli or Thrombus at the lamina cribrosa.

- 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 thrombus.
- 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.

Central Retinal Artery Occlusion

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

### Table 1. Options available in treatment of central retinal artery occlusion

<table>
<thead>
<tr>
<th>Group of drugs</th>
<th>Mechanism of action</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Intravenous</td>
<td>Decrease the blood oxygen content</td>
</tr>
<tr>
<td>2. Ocular massage</td>
<td>Increase the blood oxygen content</td>
</tr>
<tr>
<td>3. Intravenous medication</td>
<td>Reduce the intracranial pressure and hence increase the retinal artery perfusion or help disaggregate the embolus</td>
</tr>
<tr>
<td>4. Intravenous thrombolysis</td>
<td>Reduction of retinal edema</td>
</tr>
<tr>
<td>5. YAG Laser photocoagulation</td>
<td>Increase the blood oxygen content</td>
</tr>
</tbody>
</table>

### 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 Prevalence
- 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
  - Electrocadiography (ECG)
  - Echocardiography
  - Possibly, Holter Monitoring
- Co-manage with retinal specialist:
  - Consider IVFA & ERG (diminished B-wave) 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
- Consider IVFA & ERG (diminished B-wave) 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

- 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.
**Acid or Base?**

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfuric acid</td>
<td>Battery acid, industrial cleaner</td>
</tr>
<tr>
<td>Acetic acid</td>
<td>Vinegar, glacial acetic acid</td>
</tr>
<tr>
<td>Hydrochloric acid</td>
<td>Chemical laboratories</td>
</tr>
<tr>
<td>Sulfurous acid</td>
<td>Bleach, Refrigerant, fruit and vegetable</td>
</tr>
<tr>
<td>Hydrofluoric acid</td>
<td>Glass polishing, gasoline alkylates, silicone</td>
</tr>
<tr>
<td>Ammonia</td>
<td>Fertilizers, refrigerants</td>
</tr>
<tr>
<td>Lye</td>
<td>Drain cleaner</td>
</tr>
<tr>
<td>Lime</td>
<td>Plastic, mortar, cement, whitewash</td>
</tr>
<tr>
<td>Potassium hydroxide</td>
<td>Caustic potash</td>
</tr>
<tr>
<td>Magnesium hydroxide</td>
<td>Sparklers, incendiary devices</td>
</tr>
</tbody>
</table>

---

**Epidemiology**

- Chemical burns constitute 7.7% to 18% of all ocular trauma.
  - 23% in young males
  - 29% by Alkali

- 80% of chemical injuries occur in workplace accidents; 30% occur at home. 10% are the result of an assault.
- 20% of chemical injuries result in significant visual and cosmetic disability.
- Only 15% of patients with severe chemical injuries achieve functional visual rehabilitation.

---

**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

---

**COMMON ALKALI SUBSTANCE AT HOME**

<table>
<thead>
<tr>
<th>Compound</th>
<th>Common names</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ammonia</td>
<td>Muriatic acid, water treatment, soil treatment</td>
</tr>
<tr>
<td>Lye (NaOH)</td>
<td>Soap, lye</td>
</tr>
<tr>
<td>Baking soda</td>
<td>Sodium bicarbonate</td>
</tr>
<tr>
<td>Bleach</td>
<td>Sodium hypochlorite</td>
</tr>
<tr>
<td>Soap</td>
<td>Sodium stearate</td>
</tr>
<tr>
<td>Limewater</td>
<td>Sodium carbonate</td>
</tr>
</tbody>
</table>

---

**Epidemiological Trends of Chemical Ocular Burns in the United States - JAMA Ophthalmology**
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.

Management of Ocular Chemical Injury

1. Irrigate the injured eye under running water for 15 to 20 minutes using a balanced electrolyte solution.
2. Remove any contact lenses if present.
3. If the eye is closed, gently pull the lower lid to expose the cornea.
4. After irrigation, continue with any appropriate medical treatment as prescribed by the healthcare provider.
5. If the patient’s vision is affected, refer them to an ophthalmologist for further evaluation and treatment.
Nasal Canular Irrigation

Clinical Examination

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

Roper Hall Classification

<table>
<thead>
<tr>
<th>Grade</th>
<th>Graded</th>
<th>Local Treatment</th>
<th>Current Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Good</td>
<td>None</td>
<td>Epithelial damage</td>
</tr>
<tr>
<td>II</td>
<td>Good</td>
<td>0.5%</td>
<td>None</td>
</tr>
<tr>
<td>III</td>
<td>Severe</td>
<td>1%</td>
<td>None</td>
</tr>
<tr>
<td>IV</td>
<td>Poor</td>
<td>1%</td>
<td>Corneal grafting</td>
</tr>
</tbody>
</table>

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
- Topical antibiotic (e.g., fluoroquinolone) QID
- Prednisolone acetate 1% QIH 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

### Treatment Options ~ Grade 2 Chemical Burn
- 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
- Same as Grades II & III
Case #1 ~ Pearls

- Take the time to educate your receptionist(s) and technicians on how to telephone triage the ocular chemical burn patient.
- Keep a triage checklist posted at the front desk.
- Make sure you have pH strips on hand that are not expired.
- 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 internists 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.

Giant Cell Arteritis (GCA)

- 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.

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

Mortality / Morbidity

- Visual symptoms are present in about 33% of patients.
  - 40-50% are transient
  - 50-60% are permanent
- One of the more serious complication is the onset of blindness from involvement of the ophthalmic artery.
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 diffuse or bilateral.
- 5-10% are ASYMPTOMATIC.

Temporal Arteritis (GCA)

- 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.

Ophthalmic History

- 50% of patients with GCA eventually experience visual symptoms (transient visual blurring, diplopia, eye pain, sudden loss of vision).
- Transient repeated episodes are usually reversible.
- Sudden loss of vision is ominous and almost always permanent.

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

<table>
<thead>
<tr>
<th>AXONI</th>
<th>NAION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>Mean age, 9th decade</td>
</tr>
<tr>
<td>Gender</td>
<td>Female-Male</td>
</tr>
<tr>
<td>Associated symptoms</td>
<td>Juxtaocular, headache, scalp tenderness, myalgia, constitutional symptoms</td>
</tr>
<tr>
<td>Associated systemic conditions</td>
<td>Peripheral neuropathy</td>
</tr>
<tr>
<td>Visual acuity</td>
<td>Often worse than 20/200</td>
</tr>
<tr>
<td>Laboratory evaluation</td>
<td>Abnormal ESR, CRP</td>
</tr>
</tbody>
</table>

Temporal Arteritis (Dx)

- Lab Tests:
  - CBC w/ Differential
  - Anemia
  - Elevated white count
  - ESR (Wasserman) 90-95%
  - Morn. sig. / 2
  - Women (age + 10 / 2)
  - >40 is suspicious
  - C-Reactive Protein (99%)
  - Temporal Artery Biopsy

<table>
<thead>
<tr>
<th>ARTERITIC AXON</th>
<th>NON ARTERITIC AXON</th>
</tr>
</thead>
<tbody>
<tr>
<td>MALE</td>
<td>FEMALE</td>
</tr>
<tr>
<td>30 years</td>
<td>30 years</td>
</tr>
<tr>
<td>MALE</td>
<td>FEMALE</td>
</tr>
<tr>
<td>50 years</td>
<td>50 years</td>
</tr>
<tr>
<td>ASSOCIATED FEATURES</td>
<td>Non-arteritic</td>
</tr>
<tr>
<td>Visual Acuity</td>
<td>Rapid onset</td>
</tr>
<tr>
<td>DIPPE' SYMPTOMS</td>
<td>Pain may be less than typical suboccipital headache</td>
</tr>
<tr>
<td>DSA</td>
<td>&gt;90% right sided</td>
</tr>
<tr>
<td>TIA</td>
<td>Occasional 2-5 brief</td>
</tr>
<tr>
<td>MENTAL</td>
<td>Poor prognosis</td>
</tr>
<tr>
<td>IMPAIRMENT</td>
<td>Hypoactivity in about 40%</td>
</tr>
<tr>
<td>TREATMENT</td>
<td>Urgent administration of corticosteroids</td>
</tr>
</tbody>
</table>

Temporal Artery Biopsy

OCULOPLASTICS.INFO
TEMPORAL ARTERY BIOPSY
RICHARD C. LOUIE, M.D.
What is the normal range for Fibrinogen?

Look-up Color Duplex Ultrasonography of Temporal Arteries.

rbm, 6/4/2008
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.

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 or prednisone 0.5-1mg/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.
Treatment

- Preferred treatment for AAION:
  - Pulse Therapy: 3 day course of IV Solu-Medrol (Methylprednisone) 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 relapse, although relapses have been reported.

Case #3

• A 31 year old caucasian 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 EOM's 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 funduscopic 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.

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 influenzae type b (before Hib vaccine in 1985)
  - Staphylococcus aureus (including MRSA)
  - S. epidermidis
  - Streptococcus pyogenes
  - Enterococcus

- **Adults**
  - 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

**Table 1: Differences Between Preseptal and Orbital Cellulitis**

<table>
<thead>
<tr>
<th>Finding</th>
<th>Preseptal Cellulitis</th>
<th>Orbital Cellulitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Puffiness</td>
<td>Moderate to severe</td>
<td>Moderate or marked</td>
</tr>
<tr>
<td>Chemosis</td>
<td>Absent or mild</td>
<td>Absent</td>
</tr>
<tr>
<td>Proptosis</td>
<td>Rare</td>
<td>Rare</td>
</tr>
<tr>
<td>Decreased</td>
<td>Vitamin deficiency</td>
<td>Vitamin deficiency</td>
</tr>
<tr>
<td>Normal Mobility</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Intraocular Pressure</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Abscess</td>
<td>Abscess</td>
<td>Abscess</td>
</tr>
<tr>
<td>Hemorrhage</td>
<td>Hemorrhage</td>
<td>Hemorrhage</td>
</tr>
<tr>
<td>Eyelid motility</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Chemosis</td>
<td>Chemosis</td>
<td>Chemosis</td>
</tr>
</tbody>
</table>

**Note:**

- This table provides a comparison between preseptal and orbital cellulitis, highlighting differences in signs and symptoms.
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).

Preseptal Cellulitis

Axial contrast enhanced CT image, obtained in a 22-year-old man with swelling around the left eye, demonstrates left peri-orbital soft-tissue edema (arrow) without orbital abnormality.

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 reflex
  • Ophthalmoplegia
  • Proptosis
• Appears toxic
• Eye exam is unable to be completely performed
• Signs of CNS involvement:
  • Lethargy
  • Vomiting
  • Seizures
  • Headache
  • Cranial nerve deficit

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.

Case #4:

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 palpable 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 in each eye.
Case #4

- 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.

Review of Anatomy

- Iris sphincter
- Iris dilator
- Parasympathetic pathway
- Sympathetic pathway

Anatomy of the Sympathetic Pathway to the Eye

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

Dilation Lag

Horner’s Syndrome:
Clinical Features

F. Miosis, more noticeable in dim illumination. Note: Pupil reacts to light and near are normal.
G. Anhidrosis 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

Horner's Syndrome:
Clinical Features…Lastly

Horner's: Localization of Lesion

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

Horner's Secondary to Pancoast Tumor

Horner's pupil (OS)

- Dark
- Light

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% tropine will dilate a Horner’s pupil after 30-45 minutes but will not dilate a normal pupil. 0.5% works also
- 1% hydroxycamphetamine 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.
### Most Common Causes of Horner's Syndrome

<table>
<thead>
<tr>
<th>1st order</th>
<th>2nd order</th>
<th>3rd order</th>
</tr>
</thead>
<tbody>
<tr>
<td>Horner</td>
<td>Horner</td>
<td>Horner</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Hypertension</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Infection</td>
<td>Infection</td>
<td>Infection</td>
</tr>
<tr>
<td>Benign neoplasms</td>
<td>Benign neoplasms</td>
<td>Benign neoplasms</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td>Myasthenia gravis</td>
<td>Myasthenia gravis</td>
</tr>
<tr>
<td>Trauma</td>
<td>Trauma</td>
<td>Trauma</td>
</tr>
</tbody>
</table>