

1 **Ocular Urgencies & Emergencies**

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2 3 4 5 6 7 8 **Case #1**

- You are escorting a patient to the front desk to check out. While there, you over hear 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 patients 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).

9 **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?

10 **Case #1 ~ Ocular Chemical Burn**

Urgency or Emergency?

11 **Case #1 ~ Ocular Chemical Burn**

- 1 • 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.

12 13 14 **Acid or Base?**15 16 17 18 **Case #1 ~ Did the Receptionist Make The Right Decision?**19 **Telephone Triage**

- 1 • 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.
- 2 • 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.

20 **Telephone Triage**

- 1 • 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.
- 2 • 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.

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24 **Nasal Canullar Irrigation**

25 **Clinical Examination**

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

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28 **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

29 **Treatment Options ~ Grade 2 Chemical Burn**

- Topical antibiotic (e.g., fluoroquinolone) QID
- 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

30 **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
-

31 **Treatment Options ~ Grade 3 Chemical Burn**32 33 **Treatment Options ~ Grade 4 Chemical Burn**

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

34 **Keratoprosthesis**35 **Case #1 ~ Pearls**

- 1 • Take the time to educate your receptionist(s) on how to telephone triage the ocular chemical burn patient.
 - Keep a triage checklist posted at the front desk.
- 2 • 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.
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36 37 38 39 **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.

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- 1 • 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.
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41 **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.

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42 **Giant Cell Arteritis (GCA)**

43 **Temporal Arteritis (GCA)**

- An occlusive inflammatory process causing ischemic disease. Affects medium & large-sized arteries.
- HA (New onset, localized, progressive) is the major feature in 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 eastern European decent. Northern US.
- 2:1 female > male

44 **Causes**

- Overall, etiology is unknown.
- Genetic – persons of northern European decent.
- Infectious – *Chlamydia* and parvovirus may be impetus for destructive inflammation.

45 **Causes**

- Autoimmune – (cellular and humoral). The granulomatous histopathology of GCA has suggested the presence of a cell-mediated immune reaction directed at antigens in or near elastic tissue in the arterial walls.

46 **Differentials**

- Polymyalgia rheumatica
- Transient ischemic attack
- Systemic infections
- Amyloidosis with prominent vascular involvement
- Neoplasms
- Arteriosclerotic vascular disease
- Arteriovenous fistulas
- Other forms of vasculitis

47 **Pathophysiology**

- Typically involves inflammation of the aortic arch and its branches.
- Almost any artery, as well as veins, may be affected occasionally.
- Likely determinant of arterial susceptibility to GCA is the presence and/or quantity of internal elastic lamina within the vessel wall.

48 **Pathophysiology**

- Most commonly involved arteries:
 - Extracranial vertebral arteries
 - Superficial temporal arteries
 - Posterior ciliary arteries
 - Ophthalmic arteries

49 **Systemic History**

- May 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.

50 **Temporal Arteritis (GCA)**

- Signs & Symptoms:
 - Superficial Temporal Artery swelling, erythema, tenderness & pulselessness.

51 **Temporal Arteritis (GCA)**

- Signs & Symptoms:
 - Superficial Temporal Artery swelling, erythema, tenderness & pulselessness.
 - Scalp tenderness, jaw claudication, & pain in the throat, neck, teeth, gums, or eye.
 - Neurologic = transient visual loss, diplopia, mental sluggishness, & rarely, stroke. (R/O AION)
 - Systemic = fever, weight loss, anorexia, malaise, myalgias (Polymyalgia Rheumatica), sweating, & chills.
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52 **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.

53 **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.

54 **Ophthalmic History**

- The most common cause of vision loss is anterior ischemic optic neuropathy (AION).
- Results from ischemia of the optic nerve head, supplied mainly by the posterior ciliary arteries.
- Most AION is nonarteritic (87-91%).
- History of sudden painless loss of vision frequently accompanies the patient with AION.

55 **Temporal Arteritis (GCA)**

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.

56 **Ophthalmic Exam**

- Most common cause of vision loss is AION.
- Examination of the fundus may reveal optic disc edema, with or without splinter hemorrhages along the disc margin.

- Arteritic AION, as in GCA, typically presents with a chalky white edematous optic disc.
- Automated visual field testing reveals an inferior altitudinal defect, inferior nasal sectorial defect, or central scotoma.

57 **AION**

- + APD
- Pale, swollen disc, + / - flame hemorrhage
- Altitudinal VF defect
- Occasionally:
 - Vaso-occlusive disease
 - Neuro-ophthalmic disease

58 59 60 61 62 63 **Ophthalmic Exam**

- Other presentations include:
 - Posterior ischemic optic neuropathy
 - Central retinal artery occlusion
 - Branch retinal artery occlusion
 - Choroidal ischemia
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64 65 **Ophthalmic Exam**

- Neuro-ophthalmic manifestations include:
 - Diplopia
 - Ptosis
 - Nystagmus
 - Internuclear ophthalmoplegia
 - Pupillary abnormalities

66 **Temporal Arteritis (Dx)**

- Lab Tests:
 - CBC w/ Differential
 - Anemia
 - Elevated platelet count
 - ESR (Westergren) (90-95%)
 - Men (age / 2)
 - Women (age + 10 / 2)
 - >40 is suspicious
 - C-Reactive Protein (99%)

- Fibrinogen
- Temporal Artery Biopsy

67 **Procedures**

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

68 **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.

69 **Medical Care**

- 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.

70 **Medical Care**

- Using constitutional symptoms, vascular symptoms, and the ESR findings as guides, physicians usually are able to gradually taper steroids to a maintenance dose for 2 years.
- Initially, high doses of corticosteroids may be given at 1-2 mg/kg/day until the disease activity is suppressed adequately.

71 **Medical Care**

- 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.

72 **In/Out Patient Medications**

- Corticosteroids are the mainstay of therapy. In some steroid-resistant cases, a recipe of cyclosporin-azathioprine or cyclosporin-methotrexate may be used.

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76 **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.

77 **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 funduscopy 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.

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78 **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?

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79 **Periorbital and Orbital Cellulitis**

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82 **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

83 **Organisms**

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

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84 **History**

- Past sinus disease?
- Past dental disease?
- Previous eye surgery?
- History of trauma?

85 **Physical Exam**

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

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87 **Clinical Signs and Symptoms**

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

- Chemosis
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88 89 90 **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
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91 92 **Differential Diagnosis**

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

93 **Admission Criteria**

- 1 • Patients with orbital cellulitis presenting with:
 - Eyelid edema
 - Diplopia
 - Reduced visual acuity
 - Abnormal light reflexes
 - Ophthalmoplegia
 - Proptosis
- 2 • Appears toxic
 - Eye exam is unable to be completely performed
 - Signs of CNS involvement:
 - Lethargy
 - Vomiting
 - Seizures
 - Headache
 - Cranial nerve deficit

94 **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, ophthalmology, ENT and/or neurosurgery

95 **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
- Include a sepsis evaluation if the patient appears toxic or has neurologic involvement

96 **Complications**

- Local abscess formation
- Orbital cellulitis
- Intracranial extension of infection (eg subdural empyema, intracerebral abscess, extradural abscess and meningitis)
- Cavernous venous sinus thrombosis
- Septic emboli of the optic nerve
- Optic nerve ischemia (due to compression) may result in visual loss

97 **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

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100 **Case #4:**

101 **Case #4**

102 **Case #4**

103 **Case #4**

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- 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.

104 **Review of Anatomy**

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

105 **Anatomy of the Sympathetic Pathway to the Eye**

106 **Horner's Syndrome:
Clinical Features**

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

107 **Dilation Lag**

108 **Horner's Syndrome:
Clinical Features**

- Miosis, more noticeable in dim illumination. Note: Pupil rxns to light and near are normal.
- Anhydrosis on Ipsilateral side of face if lesion is below the Superior Cervical Ganglion => Not a 3rd order neuron.
- Increase in Amplitude of Accommodation due to unopposed action of the parasympathetic.

109 **Horner's Syndrome**

110 **Horner's pupil (OS)**

111 **Horner's Syndrome:
Clinical Features...*Lastly***

112 **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.

113 **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.

114 **Horner's Secondary to Pancoast Tumor**115 **Most Common Causes of Horner's Syndrome**116 **Flashes & Floaters ~ How urgent is URGENT?**117 **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?

118 **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?

119 **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%

120 **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.

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- 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.
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122 **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.

123 **59 year Retrospective Meta-analysis
Hollands and Colleagues**

- Patients previously diagnosed with an UNCOMPLICATED 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?

124 **Do all SUPERIOR 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

125 **Mechanism of RRD**

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- Size of retinal break
- Location of retinal break
- Residual vitreous traction
- Degree of vitreal syneresis (liquefied vitreous)
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126 **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 (which eye) and slight asymmetry in her intra-ocular pressures (16mmHg OD, 12mmHg OS).

127 **Case #5**

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

128 **Case #5**

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

129 **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.
-

130 **Case #5**

- 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?

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131 **Timing of Foveal Sparing RRD Surgical Repair: The Great Debate**

1 Wykoff et al:

199 patients underwent scleral buckling for Mac-On detachments:

52% were surgically repaired within 24 hours.

27% within 1-3 days

10% within 3-7 days

Rest: > 1 week

2 Erlich et al:

114 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 5.9 days.

132 **Timing of Foveal Sparing RRD Surgical Repair: The Great Debate**

1 • 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!

133 **R/B Ratio of delaying surgery**

1 • Pro's

• Optimal surgical conditions

• Most experienced staff

• Less cost for patient (surgery performed after hours costs 25% more)

2 • Cons

• Every eye is different. Some have progressed to Mac-off in as quickly as 8 hours.

• What if there is further delay from unforeseen events?

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140 **Case #6: Is HELP on the way?**

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• 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.

141 **Case #6: Is HELP on the way?**

- He reported a history of glaucoma surgery (trabeculectomy) in both eyes approximately five years earlier and was not on any topical glaucoma medication currently.
- His uncorrected distance visual acuity was 20/50 OD and 20/25 OS, and pinhole acuity was 20/30 OD.

142 **Case #6: Is HELP on the way?**

- 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.

143 **Case #6: Is HELP on the way?**

- 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.

144 **Blebitis Acronyms to Remember**

- 1 • HELP
 - H = Hypotony
 - E = Endophthalmitis
 - L = Leak
 - P = Pain
- 2 • RSVP
 - R = Redness
 - S = Sensitivity to Light
 - V = Vision Loss
 - P = Pain

145 146 **Managing Late Bleb Leaks**

- 1 • Conservative Approach
 - Suppress Aqueous Production
 - Antibiotic Prophylaxis
 - Keep the eye lubricated
 - Protection from trauma or mechanical rubbing
 - 2 • Surgical Revision
 - Needling the Bleb
 - Autologous Blood Patching
 - Compression Suturing
 - Amniotic Membrane Grafting
-

147 **Large Diameter BCL 4 Late Bleb Leaks**

148 **Blebitis**

149 **Differential DX of Bleb related infections**

1 Blebitis

2 • Defined as mucopurulent infiltrate within a bleb

• If no anterior chamber response => Aggressive Topical (FQ Q1H or Fortified Abs) + Oral (Moxifloxacin)

• Monitoring Daily

• Timely treatment Prognosis: Good

3 Bleb-related Endophthalmitis

4 • Defined as hypopyon, cells in the anterior vitreous, or culture positive aqueous or vitreous tap.

• 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.

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151 **Topic #7: Third Nerve Palsy**

152 **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")

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155 **Isolated Third N. Palsy**

156 **3rd N w/ Pupil Involvement OS**

157 **Isolated Third N. Palsy w/ Pupil Involvement**

• Posterior Communicating Artery Aneurysm (*Most Common*)

• Tumor or Trauma

• HZO

• Leukemia

• Uncal Herniation Syndrome

• Space Occupying Lesion

• Subdural Hematoma

• Pituitary Apoplexy

• Ischemic Vascular Disease (Rare)

158 **Pupil sparing / Pupil involving**

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• 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

159 **Pupil involving vs. pupil sparing**

160 **Pupil involving vs. pupil sparing**

161 **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

162 **Third Nerve Management**

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

163 **Case #8**

164 **Patient History**

- 68 year old white female.
- Ocular History:
CRAO, 2003.
- Medical history:
Diabetes
Renal Problems.

165 **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.

166 **Present Exam Findings**

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

167 **Observations, OS**

168 **Neovascular Glaucoma**

- Elevation of IOP.

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

169 **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.

170 **Signs/Symptoms**

- Acute onset of redness, pain, and blurred vision.
- Circumcilliary injection.
- Corneal edema.
- Deep anterior chamber with moderate flare.
- NVI/NVA.
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171 **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.

172 **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.

173 **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 cornea into apposition, thus blocking the trabecular meshwork.

174 **Stage I, Early**

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

175 **Stage II, Mid-Phase**

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

176 **Stage III, Late**

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

177 **Management**

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

178 **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.

179 **IOP Control**

- Medical therapy with topical β -adrenergic antagonists, α -2 agonists, and topical or oral carbonic inhibitors lower IOP.
- 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.
-

180 **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.

181 **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, retro bulbar alcohol injection, or enucleation.

182 **But...**

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

183 **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.

184 **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.

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188 **Case #9: War Paint**

- 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.

189 **Case #9: War Paint**

- 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.

190 **Case #9: War Paint**

- Funduscopy 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.

191 **Case #9: War Paint**

192 **Case #9: War Paint**

- Risk Factors for Open Globe:
 - Penetrating Lid Injury
 - Hypotony + Hyphema
 - Positive Seidel
 - Peaked Pupil and / or APD
 - Shallow or absent anterior chamber
 - 360 degree bullous sub-conj heme
 - Lens dislocation
 - Iridodialysis

193 **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.

194 **Case #9: War Paint**

- 1 • 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+)

2 • Locale:

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

195 **Eye Injuries From Airsoft Guns Up Over 500 Percent**

- 1 • 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.
- 2 • 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 gun related injuries, which parallels their growth in popularity.

196 **Case #10: Darwineyes**

197 **Acute Glaucoma Conditions**

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199 **Classification**

Primary angle closure ± glaucoma
Secondary angle closure ± glaucoma
Acute angle closure crisis

Primary open angle glaucoma
Secondary open angle glaucoma

200 **Primary Angle Closure**

Drainage angle occludable from primary mechanism (iris trabecular meshwork contact in three or more quadrants), with either raised IOP and/or primary peripheral anterior synechiae and if optic nerve damage = glaucoma

201 **picture of pupil block**

202

203 **Secondary Angle Closure**

Similar clinical findings in angle/AC though caused by "posterior pushing mechanism":

- Secondary pupil block from uveitis (seclusio pupillae and iris bombe)
- Lens induced - phacomorphic, anterior subluxation, traumatic lens dislocation
- Malignant glaucoma

204 **Secondary Angle Closure**

- Retinopathy of prematurity / PHPV
- Choroidal expansion from: drugs, posterior scleritis, AIDS, PRP, arteriovenous fistulas
- Anterior neovascularization

205 **Acute Angle Closure Crisis**

Complete occlusion of drainage angle resulting in sudden elevation of intraocular pressure (IOP)

Clinical signs: hazy cornea, fixed mid-dilated pupil, IOP >21mmHg, shallow anterior chamber

Symptoms: blurred vision, nausea & vomiting, headache

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207 **Acute Angle Closure Crisis**

- Examination: VA, IOP, slit-lamp examination, gonioscopy, undilated optic disc viewing, fellow eye assessment
- Think about and look for the mechanism of closure during the examination

208 **Acute Angle Closure Crisis**

Medical management

- Eyedrops: beta blockers, alpha agonists, carbonic anhydrase inhibitors and pilocarpine
- Oral or intravenous acetazolamide
- Oral hyperosmotic agent (glycerol) if safe for patient

209 **Acute Angle Closure Crisis**

Medical Management

Intravenous hyperosmotic agents (mannitol) it safe for patient

topical glycerin/glycerol eyedrops to improve the view

topical steroids

210 **Acute Angle Closure Crisis**

Surgical treatment

peripheral iridoplasty early if IOP not lowering

AC paracentesis for temporary IOP lowering - usually reserved for cases where other treatment has failed

Laser peripheral iridotomy: YAG, argon/diode. Treat fellow eye also!

211 **Acute Angle Closure Crisis**

Surgical treatment

- Lens extraction - often effective in refractory cases and/or if significant cataract present
- Trabeculectomy not usually performed (50% failure rate)

212 **Acute Angle Closure Crisis**

If you are sure of a secondary non pupil block mechanism (especially if it is atypical and asymmetric) then use atropine

213 **Case NM**

81 yr old woman referred from nursing home with three week history of red aching eyes and reduced vision bilaterally

VA HM, 20/800; IOP 66, 54 mmHg, hazy cornea and shallow ACs

diagnosed with acute angle closure crisis: topical timolol, brimonidine, pred forte and oral diamox 250mg administered

214 **Case NM**

arrived at GCC A&E: IOP 54, 48.
 Bilateral YAG Laser peripheral iridotomy performed
 IOPs one hour later were 18, 14
 discharged home on combigan BD and pred forte Q2hrly OU

215 **Case NM**

returned for review 2 days later: IOP 46,23
 corneae a little clearer, difficult view of disc as very dense cataracts
 drainage angle closed synechially in 3/4 quadrants OD

216 **Case NM**

phaco/PCIOL OD 2 days later, subTenon's anaesthesia
 generalised zonule weakness noted intraoperatively so capsular tension ring inserted
 corneal oedema gradually settled and UAVA 6/9
 optic nerve OD 0.9

217 **Case NM**

IOPs 17, 14 on Latanoprost nocte, Combigan BD OU
 planning phaco/PCIOL OD in the next month or two - VA 6/18, angle very narrow but open in 2
 quadrants, hazy view of disc 0.3

218 **Open Angle Glaucoma Emergencies**

If IOP > 40mmHg
 Primary: not common, but possible (younger)
 Secondary: trauma-related, steroid response, hyphaema, pigment dispersion syndrome,
 pseudoexfoliation, Posner-Schlossman syndrome, phacolytic glaucoma, ghost cell glaucoma

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