Ocular Emergencies for the Primary Care Optometrist

Michael Chaglasian, OD, FAAO
Associate Professor
Illinois Eye Institute
Illinois College of Optometry
mchaglas@ico.edu

Disclosure Statement

- **Honorarium, Speaker, Consultant, Research Grant:**
  - **Aerie, Alcon, Allergan, B+L, Carl Zeiss, Glaukos, Heidelberg, Novartis, Topcon,**

What is a "True" Emergency?

- Pain (vs. discomfort)
- Current or potential for:
  - Vision loss
  - Structural damage
  - Needs immediate (same day) attention

"True" Emergency

- History is key to differentiating emergency versus ***urgency***
  - Phone or in person
  - Proper triage is essential
  - After hours protocol
    - Your office and your specialists
  - Medico-legal implications

History

- Vision
  - One or both eyes?
  - Visual field
  - Sudden or gradual
    - Blurred or lost?
  - Diplopia?
    - Mono or Bino
- Pain
- Redness
- Onset
- Trauma
- Contact lenses
- Recent ocular disease or surgery
- Other diseases
  - cardiac, vascular, or autoimmune
  - viruses
- Medications or recent changes to medications
- Nausea/vomiting

Emergency Exam

- Acuity
- Visual fields
- Pupils
- Ocular Motility
- External examination
- SLE
- IOP
- Fundus exam

Emergency Kit

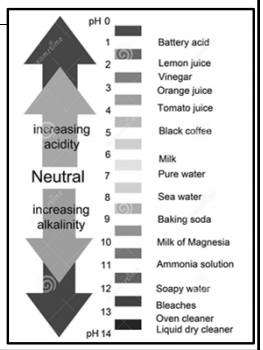
- Eye shield
- Pressure patch
- Sterile eye wash
- Alger brush
- Forceps
- Golf spud
- pH paper
- Bandage CL's
- Diamox
- Topical drops
  - Antibiotics
  - NSAID's
  - Steroids
  - Cycloplegics

"True" Emergency

- Chemical Burns
  - Alkaline
- Central Retinal Artery Occlusion
- Both have extremely high risk of severe and permanent vision loss ***which can be prevented via immediate intervention and treatment***

Chemical Trauma

- Acid exposure
  - Only penetrate through epithelium
    - car battery, vinegar, and some refrigerants
- Alkaline exposure
  - *Penetrates tissues more easily and have a prolonged effect*
    - lime, (plaster, cement) lye, ammonia found in household cleaners, fertilizers, and pesticides
- Have patient bring agent if possible



Chemical Burns

- Copious irrigation
  - anesthetic
  - speculum
  - sterile saline v tap water
- Contacts can be removed after irrigation
- Sweep fornices – repeatedly
- Examination after irrigation and neutralization of pH
- Debride necrotic tissue
- **Want neutral pH of 7.0** – check q15min with pH paper

Chemical Burns

- Determine area of involvement
  - Assessed by extent of FL staining
  - Assess depth of conjunctival penetration
    - Vascular ischemia, necrosis of limbal and bulbar conjunctiva
      - Loss of limbal vascular plexus is key indicator to severity of permanent vision loss
- Check IOP
  - Tonopen
  - IOP meds

Roper Hall Classification of Ocular Burns

Grade	Prognosis	Limbal Ischemia	Corneal Damage
1	Good	None	Epithelial Damage
2	Good	Less than 1/3	Haze but iris detail visible
3	Guarded	1/3-1/2	Total haze that obscures iris
4	Poor	>1/2	Opaque cornea

Alkali Chemical Burn

- 50 yo CM bricklayer
- Mortar splashed in eyes 1 day prior
- Went to ER, eyes rinsed, given drops and Augmentin
- c/o FBS, OD; OS feels fine
- VA: OD **HM**; OS 20/20-2
- pH: 10 OD, 7.4 OS

Alkali Chemical Burn

- Needs more irrigation and debridement
- Emergency amniotic membrane transplant (AMT)

Chemical Burn Management

- Cycloplegic
- AB or AB/steroid combo ung
- Pain control
  - Cyclo, steroids, NSAIDs
- Artificial tears
- Pressure patch
  - BCL not tolerated
- Oral narcotics/NSAID
- Topical sodium ascorbate 10% and Citrate Oral
- Doxycycline
- Diamox
- Tarsorraphy
  - Glue/permanent
- AMT
- Limbal stem cell transplant
  - Uninjured eye - autograft
  - Close relative – allograft
- Corneal transplant
- Artificial corneas

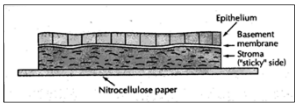
Chemical Burn - Ocular Sequelae

- Advanced glaucoma
- Eyelid destruction
- Symblepharon
- Cicatricial Entropion - months
- Trichiasis
- Ectropion
- Ankyloblepharon
- Descemet membrane detachment
- Retinal detachments
- Chronic Dry Eye
- Persistent epithelial defects/ RCE
- Infectious keratitis
- Stromalysis
- Perforation
- Irreversible intraocular damage
- Phthisis bulbi
- Hypopyon

Amniotic Membrane Transplant (not Bandage)

- Used as a surgical graft, where the tissue is integrated into the host. Amniotic membrane provides the scaffold for re-epithelialization.
- Amniotic membrane is the innermost layer of the placenta, with 3 layers:
  - Epithelium
  - thick basement membrane: supports epi call migration and inhibits apoptosis
  - Avascular stromal matrix: rich in growth factors & protease inhibitors

AM applied with epithelial-basement side facing UP, stroma DOWN
Acts as basement membrane for corneal epithelium to grow over



16. Sippel KC, et al. Amniotic membrane surgery. Curr Opin Ophthalmol 2001;12:269-281

AMT

- When used as a graft, AM is typically glued or sutured in place, and epithelium is expected to grow over it.
- AMT has an low immune response, so few rejections
- Shown to:
  1. ↑ epithelialization
  2. ↓ fibrosis, ↓ inflammation, ↓ scarring
  3. provides an anti-inflammatory and anti-scarring effect that minimizes the buildup of scar tissue
- Other uses:
  - Stevens-Johnson syndrome
  - Bullous /band keratopathy, pterygia
  - Prevent post-op adhesion of conj/sclera in trabeculectomy

CRAO

- Sudden, painless, unilateral loss of vision
- (+) APD
- Swollen, pale posterior pole
- "Cherry red" spot
- Vascular attenuation &/or boxcarring of flow in arteries & veins
- May see an embolus in vessel on optic nerve

CRAO

- 47yoCF with 3 day h/o blur, OS, with severe loss yesterday
- Had been seen twice in previous week
  - 1 MD, 1 OD
  - Dx: Dry eyes
  - Tx: Artificial tears
  - Spoke to tech yesterday
- Casual about coming in

Patient findings

OD		OS
20/20	**Distance Acuity**	**CF 1'**
		(+) APD
12	**TA**	**12**
	BP 128/79	
WNL	**SLE**	**WNL**

CRAO

- In office AC paracentesis & digital massage
  - ASA
- To Cardiologist
  - Echocardiogram
- To ER
  - Blood work
    - ESR: rule out Temporal Arteritis
- To PCP
  - Hematology workup
- To Retina
  - Brimonidine

Vision Never Improved

CRAO

- Echocardiogram
  - Dilated aortic root
    - associated with connective tissue dx
- Blood work:
  - ESR: minimally elevated
  - Elevated homocysteine level
    - amino acid in blood derived from the digestion of protein-rich foods
  - Elevated PTT (Partial thromboplastin test)
  - Other hypercoagulability factors negative
  - Negative CT, MRI, MRA

CRAO Causes

- Emboli
  - 74% cholesterol
  - 10.5% calcific
  - 15.5% platelet-fibrin
- Thrombosis
- Vasculitis
  - GCA
- Post-trauma

- In patients of 70 years of age and older, giant cell arteritis is more likely to be the cause than in younger patients.

CRAO

- Incidence
  - 3.5/10,000 in general population
- 92% of patients have </= CF acuity
  - Only ~ 8% will experience VA improvement
- Two thirds of patients experience 20/400 vision while only one in six will experience 20/40 vision or better.
- Risk Factors
  - Age >40
  - Systemic conditions
    - HTN, DM, CV dx, prior MI, stroke, hypercholesterol, hypercoagulable state
  - Social/Medication history
    - Smoking

CRAO Testing

- ESR & CRP
  - r/o GCA
- CBC/lipid profile
- Cardiology referral
  - risk of MI
- Carotid Doppler – r/o emboli from carotid
- Auto-immune
- Inflammatory
- Hematology workup
  - Coagulopathies
    - Younger pts

CRAO

- Retina suffers no detectable damage up to **97 minutes**
- BUT sustains massive, ***irreversible damage after 4 hours***

  - *Hayreh SS, Zimmerman MB, Kimura A, et al. Central retinal artery occlusion. Retinal survival time. Exp Eye Res 2004;78:723-36.*

CRAO & Mortality

- Patients with emboli have a 56% mortality rate over 9 years, compared to 27% for an age-matched population without emboli
- Life expectancy of patients with CRAO is 5.5 years compared to 15.4 years for an age-matched population without CRAO

CRAO – Acute treatment

- Lower IOP
  - **Anterior chamber paracentesis**
    - remove 0.1 to 0.4 ml of aqueous fluid within the first 24 hours.
  - Digital ocular massage
    - Some use 3-Mirror Lens
  - IOP lowering agents

CRAO – Acute treatment

- Move occlusion
  - Carbon/carbogen inhalation
    - CO_2 dilates arterioles & increases O_2 delivery to ischemic tissues
  - ***Paper bag – dilate retinal vessel***
- Remove embolus
  - Local intraarterial fibrinolysis (LIF) was associated with similar visual outcomes but increased rate of adverse reactions when compared to observation in the EAGLE study.
  - The study did not recommend LIF for acute CRAO.

CRAO

- Risk of neovascularization
  - NVD, NVI, NVG
  - Reports from 2.5-35%
  - 18.2% @ an average of 8.5 weeks post CRAO

- *Ocular neovascularization following central retinal artery occlusion: prevalence and timing of onset. Eur J Ophthalmol. 2010 Nov-Dec;20(6):1042-6.*

Urgencies

- Giant Cell Arteritis
- Endophthalmitis
- CRVO
- Papilledema
- Optic neuritis
- Corneal abrasion
- Iritis
- Corneal ulcer
- Penetrating injury
- Ruptured globe
- Lacerations
- Corneal foreign body
- Retinal detachment
- Orbital cellulitis

Giant Cell Arteritis / Temporal Arteritis

Background on GCA

- Giant cell arteritis (GCA) is the most common primary vasculitis in adults.
- Histopathologically, GCA is marked by generalized granulomatous inflammation of medium- to large-sized vessels that occurs in the elderly.
- **Patients commonly note associated symptoms such as headache, jaw claudication, diplopia, myalgias, and constitutional symptoms.**
- **The most commonly feared sequela of GCA is permanent visual loss secondary to arteritic anterior ischemic optic neuropathy (AAION).**
- Since the vision loss from AAION can progress rapidly, and can involve the fellow eye within a matter of days, GCA is considered an ophthalmologic emergency.

Giant Cell Arteritis / Temporal Arteritis

- Inflammation of medium & large sized arteries of the body, restricting blood flow & causing pain
- VA loss usually unilateral, but risk of loss in other eye if not treated within 1 week
- Elderly (>60), white, female
- Scalp tenderness, jaw claudication, HA, loss of appetite, weight loss, fatigue
- Need stat ESR & temporal artery biopsy
  - Normal Sed Rate:
    - F= (age+10)/2
    - M= age/2

GCA Exam Findings

- CRAO
- AION
- Flame hemes, cotton wool spots
- Pale, swollen disc
- 6th nerve palsy

Treatment

- High dose systemic steroids
  - 60-100mg qd
  - IV methylprednisone
    - 250 mg q6h x 3 days
- Aspirin

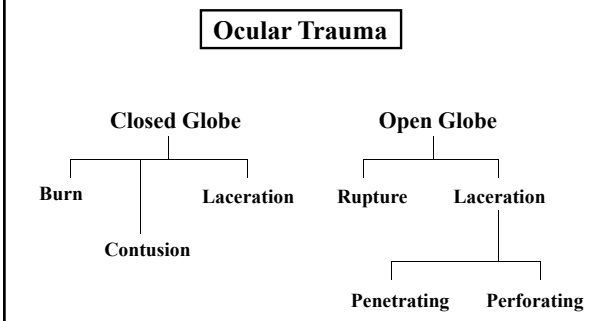
- Referrals: Neurology, Rheumatology, Ophthalmology
  - Temporal Artery Biospy
  - Color duplex ultrasonography

Ocular Urgencies

- Trauma
  - Penetrating/non-penetrating
    - Hyphema
    - Blowout fractures
    - Ruptured Globe
    - Lacerations
    - Corneal Abrasions
    - Foreign Bodies
- Retinal detachment
- Orbital Cellulitis
- Iritis
- Corneal ulcer
- Optic neuritis
- Acute exophthalmus

Trauma

- 2,500,000 traumatic eye injuries /yr in US
  - 40,000-60,000 lead to visual loss
- 40% of all new cases of monocular blindness
- 80% occur in men
- 75% are work related
- Average age 30



Hyphema Grading

- Grade 1 - Layered blood occupying <1/3 of the anterior chamber
  - >50%
- Grade 2 - 1/3-1/2 of the anterior chamber
- Grade 3 - Layered blood filling 1/2 to less than total of the anterior chamber
- Grade 4 - Total (100%) clotted blood, aka 8-ball hyphema
  - <10%

Traumatic hyphema

- r/o rupture
- Fox shield
- Restrict activity/head elevation
  - Hospitalization?
- D/C blood thinners
  - ASA, NSAID's
- Atropine
- Pred Forte qid
- ~30% have elevated IOP
  - Beta blocker
  - Gonio ~ 1 month later
- r/o sickle/sickle trait
- 10-20% re-bleed rate
  - More common w/Gr. 3-4
  - corneal staining, glaucoma, optic atrophy
  - Systemic aminocaproic acid (ACA)

Angle Recession

- Blunt trauma which causes a hyphema is more likely to also cause an angle recession
- Between 2 and 20% of patients with angle recession will go on to develop glaucoma

Traumatic Glaucoma

- patients with damage to greater than **two-thirds** of angle are also more likely to develop glaucoma
- glaucoma associated with angle recession may develop months to years following the injury
- Glaucoma is related to magnitude of hyphema,
  - Only 13% get when hyphema is less than half of AC
- Bed rest (head elevated) or very limited activity along with patching are usually recommended.

Management

- Early Phase Glaucoma and Acute Trauma
  - Acute Inflammation / Iritis
  - Hyphema
    - red blood cells in the anterior chamber
    - results from a tear in the angle that breaks a blood vessel
    - rebleeds occur up to 7 days after trauma

Hyphema

- Measure the percent of hyphema in AC
- Measure the IOP
  - may have very high IOP
- NO gonioscopy,
  - must wait 7 days to prevent causing a rebleed
- Check sickle cell status
  - Those with SC may need AC wash out

Medications: Early Phase

- For Inflammation:
  - Cycloplegia:
    - w/ atropine 1% BID, Homatropine 5%
  - Topical steriods:
    - prednisolone acetate 1% QID
  - NO aspirin products: rebleed

Elevated IOP from Hyphema

- PGAs:
  - Not an option for early phase
  - Good option b/c uveoscleral outflow
- Beta Blockers
  - Timoptic XE, Betagan, etc.
- Alphagan
- Topical or Oral CAI's
- Cosopt or Combigan= good option
- The usual duration of an uncomplicated hyphema is 6 days
- **Anterior Chamber "Wash Out"**

Trauma - Penetrating Injury

If rupture- STOP exam

- Fox shield and to hospital for exam under anesthesia
- CT if foreign body can't be seen
- Systemic antibiotics
- Compromise protective outer layers, increasing the risk of infection
  - Risk of endophthalmitis
- Intraocular contents may be outside the globe

Trauma - Laceration

- Corneal
- Conjunctival
- Lid

- Be alert with high velocity injuries for intraocular foreign bodies

- Seidel's sign: (+) full thickness laceration

Corneal Lacerations

- Full thickness
  - protect w/ shield & immediate referral
- Partial thickness
  - treat as abrasion

Corneal Laceration

- 41yoCM- construction foreman
- Nail ricocheted off a wall and hit his OD 1 day prior – felt OK
- Today, 5/10 pain, photophobia, FBS
- h/o metallic FB 10 ya
- Recent tetatnus shot

Corneal Laceration

- Va OD: 20/20; OS: 20/25
- (+) epi defect
- (+) AC rxn
- (-) Seidel's sign
- TA 9 mmHg, OU
- Lid eversion negative
- Fundus exam WNL

- + Ocuflox
- + Cyclogyl

- **Get CT to r/o intraocular foreign body**

Corneal Laceration

- 1 day follow up, feels 50% better
- Va 20/30
- No epithelial defect
- + stromal haze

- Decrease Ocuflox
- Start Pred-Forte

Conjunctival Lacerations

- SLE
  - Subconj heme
  - (+) Fl stain
- DFE
  - **Mild**
    - **Majority are self-limiting**
    - **Antibiotic, patch, follow up 1 day**
  - Severe
    - Antibiotic, patch & refer

Blow Out Fracture

- Direct trauma to the globe causes an increase in intra-orbital pressure & decompression via orbital floor fracture
  - Maxillary bone & posterior medial wall
- Periorbital swelling, ecchymosis & restricted eye movement
- Diplopia, hypesthesia of cheek & teeth, orbital pain
- Most common sign is limited upward or downward gaze due to entrapment of IR or IO
- Crepitus
  - palpate the bony rim of the orbit or lid
    - small bubbles of air "pop" when compressed.

Management

- ***When should surgical repair of blowout fractures be considered?***
  - Fractures with persistent symptoms (typically double vision or pain) are usually candidates for surgical repair. Timing of the repair varies, but most often is within two weeks of the injury. Initial repair may consist of any of the following:
  - • exploration of fracture site and repositioning of bone
    • release of trapped tissue from fracture site
    • covering of fracture site with synthetic material
- ***What long-term problems may develop following blowout fractures?***
  - Most fractures heal without long-term effects. However, strabismus surgery (eye muscle surgery) is sometimes necessary for persistent double vision. Occasionally, persistent double vision can be treated with non-surgical methods (prism glasses or botulinum toxin injections)

Blow Out Fracture

- Ice packs 24-48 hours, no nose blowing
- Recovery can be spontaneous - 2-3 days
- Systemic antibiotics
- Can trigger orbital cellulitis, angle recession glaucoma, & RD.
- Prompt surgery if poor cosmesis, persistent diplopia, enophthalmus, pediatric patient with entrapment

Corneal Abrasion

- Acute pain & photophobia
- May need anesthetic to perform exam
- Debride loose epi if necessary
- Antibiotic/cycloplegic/NSAID
- Pressure patch v. Bandage CL
- RCE issues

Corneal Abrasion

- 44yoCM
- CC: Pocked in OS with belt buckle 1 day prior
- : S/P PRK OU
- (+) FBS, photophobia, redness

  - Antibiotics
  - Cycloplegic
  - Treat any associated anterior chamber reaction
  - BCL?
  - Debridement of loose tissue
  - RCE issues
    - Fingernail, paper, fingernail, pre-existing EBMD

Corneal/Conjunctival Foreign Body

- History important
  - ? object
  - High speed?
  - DFE
- SLE
- Lid eversion

1) Removal: irrigation, moistened cotton swab, spud, needle, forceps, Alger brush
2) Antibiotic/cyclo/patch/BCL
3) Steroids for scar modulation after defect healed

Rhegamatogenous Retinal Detachment

- Symptoms:
  - Flashes
    - Vitreous separates from the retinal pigment epithelium
  - Floaters
    - Separation at retinal vessel may leak blood into the vitreous
  - Decreased VA from macular involvement can lead to severe, permanent vision loss
- Separation of neurosensory retina from the underlying RPE, by fluid from the vitreous cavity and into the sub-retinal space.
- Time course of retinal break or defect to time of detachment is highly variable and unpredictable

Rhegamatogenous RD

- Develop from Retinal Defect:
  - Hole or Break in sensory retina
  - Frequently from Posterior Vitreous Detachment (PVD)
- Other Forms of Retinal Detachments:
  - Exudative
  - Tractional

Retinal Detachment

- 51yo AAM presents to Urgent Care @ IEI
- CC: Sudden blurry vision superiorly OS
  - x1 week, painless & progressive
    (-) trauma
    (-) flashes
- POH: Cataract extraction, OU
- LEE: 2 years ago at IEI, no SRx
- PMH: HTN, NIDDM (controlled)

Entrance Examination

- VA sc: OD 20/60+1 PH: 20/60+2
- OS 20/50-1 PH: 20/40+2
- Pupils: PERRL 1+APD(OS)
- EOMs: FROM OU
- CVF: FTFC OD
  - Constricted superiorly OS

Slit-Lamp Examination

	OD	OS
Adnexa/Lids:	1+ MGD	1+ MGD
Conjunctiva/Sclera:	W&Q	W&Q
Cornea:	Unremarkable	Unremarkable
Angles:	4+T/4+N	4+N/4+T
Anterior Chamber:	D&Q	D&Q
Iris:	Unremarkable, (-)NVI	Unremarkable, (-)NVI
Lens:	PCIOL in place, intact, clear	PCIOL in place, intact, clear
IOP (Goldmann):	14	12
Anterior Vitreous:	Unremarkable	(+)Shafer's sign

Rhegmatogenous Retinal Detachment

- Patient education:
  - risk of permanent vision, need for IMMEDIATE retinal referral.
- Patient Treatment and Outcome:

RRD Statistics

Prevalence: RRD 10-15/100,000
- 70%: Non-Traumatic
  - 50% myopic
    - 1-3D: 4x risk, >3D: 10x risk
  - 10% increased risk in fellow eye
- 10%: Trauma
- 20%: Post-Cataract Surgery
  - S/P Cataract Extraction
    - 10 year risk: 6x risk
  - S/P Refractive lens exchange
    - 2-8% RRD within 1 month
- 20 - 30% have lattice degeneration

SYMPTOMATOLOGY

- Often asymptomatic and thus detected on routine examination
- Inferior and temporal location from atrophic holes
  - Patients less aware of these slowly progressive RDs as superior and nasal fields of view are less sensitive
- Flashes/Floaters. Yes or No?
  - Not noticed while sleeping
  - Unilateral symptoms often masked
  - Not affecting central vision
  - Slow progression/change
  - Frequent "Red Herring"
    - TIA, acephalgic migraine, PVD w/o detachment
      - PVD Follow Up: 6-8 weeks of exam detection or with increased symptoms

Key Findings for Referral

- Finding a retinal break
- Finding subretinal fluid
  - May not find break, but it's likely there as that's how the fluid got in
- Other Signs:
  - Acute PVD
  - Pigment cells in vitreous or vitreal heme
- Techniques to help:
  - Scleral depression and 3-Mirror retinal exam
  - OCT

OCT for RD

- Use Wide-field Mode
- Helpful in Differential diagnosis
- Helpful in determining macular status
  - "on or off"

Treatment

- Laser
  - Slit lamp or Laser Indirect Ophthalmoscopy
    - LIO for more peripheral breaks
  - For subclinical holes and breaks with limited SRF and areas of contact between the retina and RPE remain
  - Laser is applied at the border of the attached and detached retina, the photocoagulation creates "seal" that prevent further progression of the break/detachment
- Monitor Asymptomatic Breaks?
  - Low (~5%) risk for progression

Treatment

- Pneumatic Retinopexy (62-78%)
  - Oil/Gas bubble with laser
- Pars Plana Vitrectomy: small incision
  - Fluid-Air, air-gas exchange, silicone oil injection with laser
- Scleral Buckle: for larger RDs
  - Thin scleral band placed under conjunctiva to indent sclera towards retina followed by laser or cryotherapy
  - Decreased in utilization in favor of PPV due to numerous SEs

Clinical Pearls - Urgency

Macula On Status	Timeframe
Progressive/large	Immediate/next day
Stable/small/inferior	<5 days

Macula Off Duration	Mean Postoperative VA†
≤10 days	20/41
11 days to 6 weeks	20/121
>6 weeks	20/178

†Scleral buckle repairs

Endophthalmitis

- Incidence 0.13%
  - (review of 215 articles from 1963-2003)
- Symptoms:
  - Pain, decreased vision, photophobia, redness
- Signs:
  - Corneal edema, hypopyon, vitritis,

Endophthalmitis Vitrectomy Study (1995)

- 420 patients < 6 weeks post CE or 2° IOL c s/s of endophthalmitis
- Randomized to +/- systemic antibiotics
- Randomized to ParsPlanaVitrectomy (PPV) v AC tap & vitreous injection with antibiotics
- Results:
  - Patients with > LP: Tap and inject (no need for immediate PPV)
  - Patients with < LP: **Immediate PPV**, and inject
    - 3 fold increase in the frequency of achieving 20/40 or better acuity

Acute Angle Closure Glaucoma

- Classic Symptoms:
  - Unilateral, painful eye
  - Blurred vision, halos
  - Nausea, vomiting
- Characteristics:
  - Older, white, hyperopic females age > 30 yrs
    - Peak age 55-70
  - First-degree relative with ACG
  - History - previous episodes, medications, surgeries, etc.

When can the angle close?

TABLE 1 Shaffer System of Angle Classification

Numerical grade	Angle grade	Angle width	Clinical interpretation
4	Wide open angle, ciliary body seen	35°-45°	Closure impossible
3	Wide open angle, scleral spur seen	25°-35°	Closure impossible
2	Narrow angle-moderate, only the trabeculum identified	20°	Closure possible
1	Narrow angle-extreme, only Schwalbe's line and top of the trabeculum seen	10°	Eventual closure probable
0	Narrow angle-complete or partial closure, iridocorneal contact, apex of corneal wedge not identified	0°	Closure present or imminent

Acute Angle Closure Glaucoma

- Symptoms: Pain, halos around lights, redness, nausea, vomiting
- Signs: Corneal edema, fixed,oval mid-dilated pupil, ciliary flush, AC inflammation, glaukomflecken
- Extremely elevated IOP
- Gonioscopy
  - Indentation
  - Both eyes

CLINICAL FEATURES:

- Acute Angle Closure Attack:
  - diffuse conjunctival hyperemia
  - cloudy, edematous cornea
  - fixed, oval, mid-dilated pupil
  - VA often significantly reduced
  - IOP ranges 40-70+ mm Hg
  - inflammation / aqueous cell/flare

"CLASSIC" PATIENT SYMPTOMS:

- These symptoms are associated with "Acute" Angle Closure
  - deep, aching pain (high IOP)
  - redness, tearing, photophobia
  - blurred vision, halos (edema)
  - nausea, vomiting (pain)
  - Headache

Note:
- Many patients do NOT have these symptoms

GONIOSCOPY

Look for PAS (peripheral anterior synechia) as evidence of angle closure

Gonioscopy of both eyes to confirm a narrow angle approach (symmetry)

Only Indentation gonioscopy shows this, not three mirror.

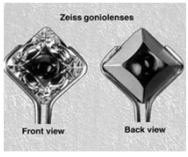
Gonioscopy Lenses

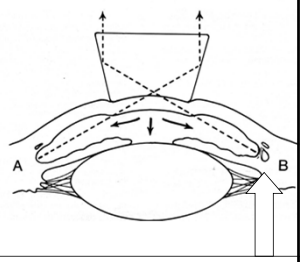
Posner 4 mirror
» **Handle**

Sussman 4 mirror
» **No handle**

» **www.ocular-instruments.com**

Indentation Gonioscopy

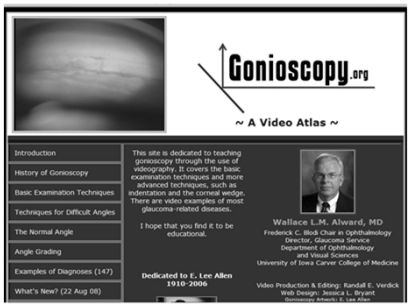




A. = Appositional angle closure

B. = Synechial angle closure

Gonioscopy on the Web!



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When can the angle close?

TABLE 1 Shaffer System of Angle Classification

Numerical grade	Angle grade	Angle width	Clinical interpretation
4	Wide open angle, ciliary body seen	35°-45°	Closure impossible
3	Wide open angle, scleral spur seen	25°-35°	Closure impossible
2	Narrow angle-moderate, only the trabeculum identified	20°	Closure possible
1	Narrow angle-extreme, only Schwalbe's line and top of the trabeculum seen	10°	Eventual closure probable
0	Narrow angle-complete or partial closure, iridocorneal contact, apex of corneal wedge not identified	0°	Closure present or imminent

Management - Acute Angle Closure Glaucoma

- Oral CAI's (250 - 500 mg Diamox)
  - Tablet form only, not Sequel form
    - Sequel takes too long to act
- Topical fixed combination:
  - Combigan / Cosopt
  - Iopidine (if available)
- Topical steroids
- When IOP < 45, add 1-2% pilocarpine

- Definitive Treatment: Laser Peripheral Iridotomy (LPI)
  - Eliminates pupillary block
  - Prophylactic LPI of fellow eye

Corneal Ulcer

- Peripheral vs Central
- Infection
  - Staph, strep, moraxella, pseudomonas
- Pain, redness, photophobia
- CL wear
- Chronic eyelid disease
- SLE: focal accummulation of inflammatory cells in excavation with overlying epi defect

Corneal Ulcer

- 15yoCF
- Being treated elsewhere for Corneal ulcer, OS
  - > 1 week with fortified antibiotics, oral AB's
- SCL wearer
  - Fell asleep on a road trip
- VA sc: OD 20/20, OS 20/400 PH NI

Differential Dx

- Infiltrate
- Ulcer
  - Pseudomonas
  - Fungal
  - Acanthamoeba

Corneal Ulcer Treatment

- Discontinue CL wear
- Antibiotic
  - BESIVANCE (**besifloxacin**) 0.6%
  - 1 gtt q15minX 2 hrs, then hourly while awake
- Cycloplegic
- Culture if available, if central, if >3mm

Corneal Ulcer Treatment

- Fortified agents?
  - subconjunctival loading dose injection of 0.3 cc gentamicin mixed with 0.3 cc lidocaine at the time of presentation
- Steroids when defect is closed to modulate scarring
  - The SCUT study (Steroids for Corneal Ulcers Trial), a large series involving 500 patients that was published in the *Archives of Ophthalmology*, found no overall difference in three-month BSCVA and no safety concerns with adjunctive corticosteroid therapy for bacterial corneal ulcers

If NI, consider fungal or acanthamoeba….

Optic Neuritis

- **Unilateral, acute loss of vision**
- **Women, 20-45yo, AA**
- **Strong association with MS**
- The patient's history may reveal the following signs and symptoms of optic neuritis:
  - Preceding viral illness
  - Rapidly developing impairment of vision in 1 eye or, less commonly, both eyes: During an acute attack
  - Dyschromatopsia in the affected eye: Occasionally may be more prominent than the decreased vision [4]
  - Retro-orbital or ocular pain:
    - In association with the vision changes and usually exacerbated by eye movement; the pain may precede vision loss
  - Uhthoff phenomenon, in which vision loss is exacerbated by heat or exercise

Optic Neuritis: Signs and Symptoms

- Varying degrees of vision reduction:
  - Ranging from a mild decrease in visual acuity to complete vision loss, which is often monocular but can be binocular
  - scotomas, ocular or orbital pain, worse on EOM's
- (+) APD, color vision & VF loss
  - Abnormal contrast sensitivity and color vision:
    - In almost all patients with adult optic neuritis who have decreased visual acuity

Optic Neuritis: Signs and Symptoms

- **Field defects:**
  - May include altitudinal, arcuate, nasal step, central scotoma, cecocentral scotoma
- Initially, the optic nerve head may appear normal.
  - Papillitis (swollen disc) may be seen in one third of patients with optic neuritis

Evaluation and Workup

- **Magnetic resonance imaging (MRI)** is highly sensitive for and specific in the assessment of inflammatory changes in the optic nerves, and for central nervous system white matter lesions.
  - MRI also helps to rule out structural lesions.
- Labs. Only if other forms of neuritis are possible
  - Erythrocyte sedimentation rate
  - Thyroid function tests
  - Antinuclear antibodies
  - Angiotensin-converting enzyme
  - Rapid plasma reagin
  - Mitochondrial deoxyribonucleic acid (DNA) mutation studies

Management

- Visual function begins to improve 1 week to several weeks after onset, even without any treatment
- Permanent residual deficits in color vision and contrast and brightness sensitivity are common
- Pharmacologic therapy in optic neuritis (ON) is directed at ameliorating the acute symptoms of pain and decreased vision caused by demyelinating inflammation of the nerve; varying regimens of corticosteroids have been used for this purpose.
- IV steroids do little to affect the ultimate visual acuity in patients with optic neuritis, but they do speed the rate of recovery;
  - Some clinicians advocate IV steroids in patients with either severe or bilateral vision loss

Optic Neuritis Treatment Trial

- 448 pts
  - Oral placebo, IV steroids with placebo orals, or oral steroids
  - The primary goal was to determine whether oral or IV steroids altered the visual outcome in patients with acute optic neuritis.
    - **IV steroids accelerated recovery of VA initially, but provides no longterm benefit.**
    - **Oral steroids provided no visual benefit and was associated with a higher rate of recurrence.**
  - **Initial MRI result, if abnormal with white matter abnormalities, was the single most important predictor of the future risk of MS**
    - Routine blood tests of no value

If Possible MS

- For patients with optic neuritis whose brain lesions on MRI indicate a high risk of developing clinically definite MS, treatment with immunomodulators:
  - interferon [INF] beta-1a,
  - INF beta-1b,
  - glatiramer acetate

  (may be considered)

Papilledema

- Bilateral optic nerve head edema, secondary to increased ICP
- Decreased acuity may not be presenting symptom
  - Headache
- Need imaging to rule out space occupying lesion
- Lumbar tap/CSF studies
- Pseudotumor Cerebri - Dx. of exclusion

Central Retinal Vein Occlusion

- Non-ischemic v. Ischemic
  - Ischemic – less common, more severe with poorer prognosis
    - NVG/NVI
      - q1month x 6months, q2-3 months x 6 months, q4-6 months x 3 years
- Men > 50
- <40: Needs systemic workup
  - antiphospholipid antibody syndrome
- Poor acuity, possible APD
- Hemes, dilated vessels, CWS, retinal & macula edema
- Lower IOP

Neovasculogenesis:

- Chronic retinal hypoxia is theorized to initiate the production of an angiogenic substance / vasoproliferative substance which elicits new blood vessel growth.
  - Vascular Endothelial Growth Factor (VEGF)
- Iris tissue is the primary site for developing neovascularization.
  - Progresses through several stages:

Angle Closure Stage

- Time frame for development to this stage:
  - can be variable, from days to months
- NVG is known as the:
  - "Ninety day glaucoma", because of its rapid onset
- Features:
  - pain, photophobia
  - decreased Va
  - increased IOP, high
  - conj. congestion
  - iritis
  - corneal edema
  - rubeosis iridis
  - ectropian uvea
  - synechial angle closure

First Step= Medical treatment.

- Medical therapy is directed at lowering the IOP, reducing the anterior segment inflammation and making the patient more comfortable.
  - Note the similarities and ***differences*** to the management of primary acute angle closure

Rapid IOP Reduction:

- Acetazolamide: 250 mg x 2
- Beta blocker and Alphagan
- Anti-inflammatory:
  - prednisolone acetate 1%
- Atropine 1%
  - For pain and inflammation
- Referral for Avastin and PRP

Central Retinal Vein Occlusion

- Prompt referral to PCP
  - FBS, ESR, CBC, lipid profile, FTA-ABS
- Strong association with HTN, DM, POAG
  - Check BP and IOP in office
- Hyperlipidemia, hyperviscosity
- Macula edema treatment:
  - Avastin
  - Ozurdex
  - Kenalog
  - PRP

Orbital Cellulitis

- Usually results from spread of infection from paranasal sinuses
  - CT
  - ENT consult
- Tx with IV antibiotics
- Must distinguish from Preseptal
  - Both present with lid edema, pain, redness
  - FEVER
  - PROPTOSIS
  - PRECIPITATING EVENT
  - ANY AGE, SEX, RACE

Patient "Emergencies"

- Ocular Migraine
- Redness/itching/burning/discharge
- Subconjunctival hemorrhage
- Gradual change in visual acuity
- Lid bumps (non- painful)
- Refractive changes

Take Home Message

- Be prepared
  - Have appropriate equipment/ referrals in place
- Take the time to train your staff
  - You are ultimately responsible
- Be methodical in your thinking & your examination
- Know when it's OK to treat, and when you must refer

Thank you!