

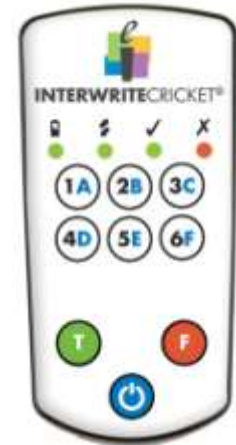
Diagnosing and Managing Ocular Emergencies and Urgencies.

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Disclosures and Special Request

- Paid consultant for:
 - Carl Zeiss Meditec
 - Ista Pharmaceuticals
 - Alcon Pharmaceuticals
- Special Request:
 - Interactive remotes don't work on your TV, so please don't take them home! :)
 - At the end of the lecture, I would ask that you turn them off and please place in a foam tray at the room entrance.
 - Thank You!! Let the Learning Begin!!!



What Classifies an Emergency?

- Any condition in which the patient has the potential for:
 - vision loss,
 - currently experiencing vision loss,
 - permanent structural damage,
 - pain or discomfort,
 - or is an “emergency” for the patient.
- It is important to be able to triage a walk-in patient and, more importantly, a call-in patient.

What questions to ask?

Onset	suddenly noticed or sudden onset?
Visual Loss	any loss of vision? loss vs. blurry vision one eye or both part of visual field or all transient vs. permanent
Pain	is there pain? constant? scale (1-10)
Redness	is there any redness? location?
Associated Factors	contact lens wear? trauma? discharge? photophobia? medical history (eg. DM)

Common Types of Ocular Emergencies

- Vision Loss:
 - Gradual vs. sudden onset
 - Vision loss with or without pain
- Trauma
- Red eyes

Visual Loss

- Visual loss varies greatly in meaning from Pt to Pt
 - ranging from blur to complete blindness and may affect one or both eyes
- Components include:
 - acuity,
 - visual field,
 - color and brightness may be affected jointly or separately
- Detailed history and extent of vision loss crucial

Profound Loss of Vision

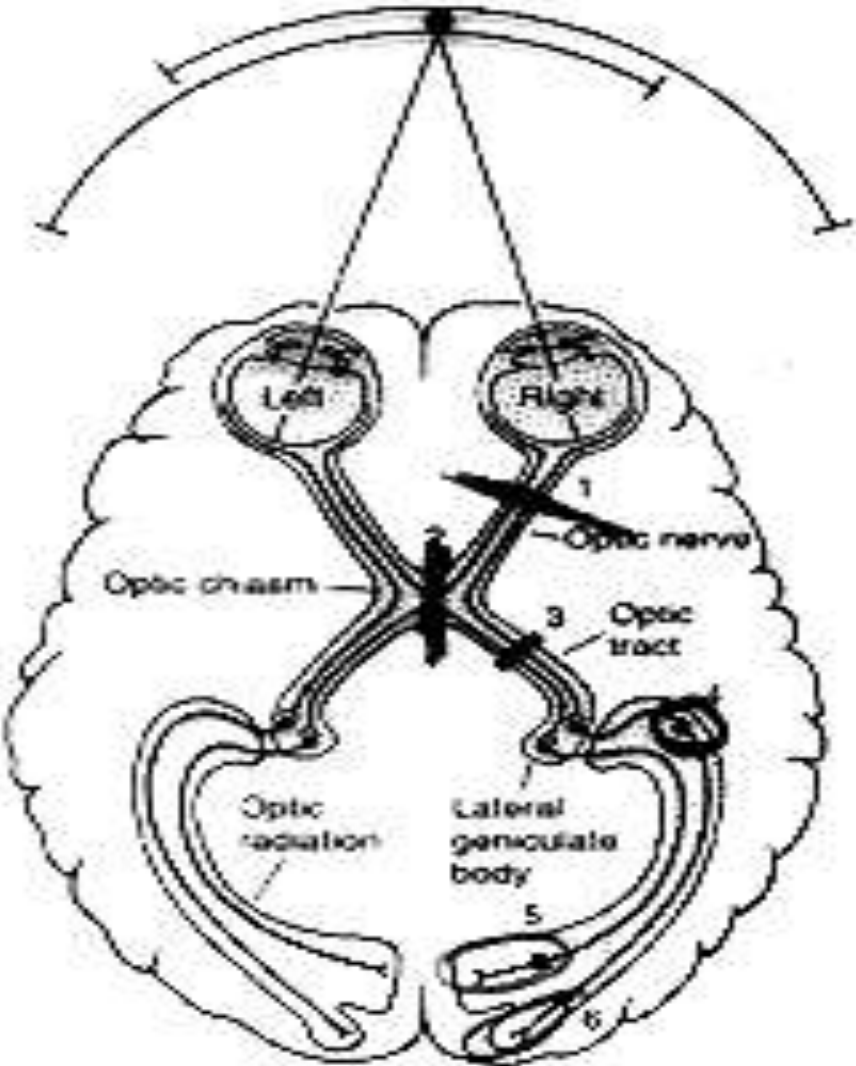
- Referring to a complete or greatly diminished vision affecting the whole field
- Common causes of severe vision loss:

Vascular	central retinal vein occlusion, central retinal artery occlusion, vitreous heme
Inflammatory	optic neuritis
Infiltrative	optic neuropathy
Mechanical	retinal detachment

Monocular vs. Binocular

- Ocular or optic nerve pathology causes monocular vision loss
- lesion at or posterior to chiasm causes binocular vision loss
 - VF defects become more congruous the further back in the visual pathway
 - Homonymous VF defects noted posterior to chiasm
- Difference between mono vs. bino usually straightforward, keeping the following in mind:
 - Patients occasionally mistake homonymous hemianopsia (similar loss of visual field in both eyes) for a monocular loss

Visual Defects



Defects in visual field of

	Left eye	Right eye	
1			Nerve.
2			
3			tract
4			
5			}
6			

Monocular

- Differentiate between eyes that have lost all useful vision and those that have blurred vision
- Blurring of vision is not localized and may be caused by pathology anywhere from cornea to optic nerve
- Need to get anatomical diagnosis first before considering the cause

History

- Onset-gradual vs. sudden onset
- Gradual onset-
 - Cornea and lens-refraction, cataracts
 - Anterior chamber and vitreous-clouding (inflammation)
 - Retinal disease-macular edema
 - Optic nerve-swelling, pallor, atrophy
- Sudden onset-
 - e.g. vitreous heme and vascular occlusive disease

General Appearance

- Level of consciousness
 - When introducing yourself be aware of the patient's gross level of consciousness?
 - Is the patient awake, alert and responsive?
- Personal Hygiene and Dress
 - Is it appropriate for the environment, temperature, age and social status of the patient?
 - Is the patient malodorous or disheveled?



General Appearance

- Posture and Motor control
 - What posture does patient assume while sitting in the exam chair
 - Are there any signs of involuntary motor activity such as tremors
 - E.g. damage to the cerebellum may produce a tremor that usually worsens with movement of the affected limb

General Appearance/Vital Signs

- Height, Weight and Build
 - Note general body proportions and look for any gross deformities
- Vital signs
 - These include:
 - Blood pressure
 - Pulse
 - Respiratory rate
 - Temperature

EMR, CMS and MU

- Physicians and hospitals which implement an electronic medical record and meet the specified requirements will receive up to five years of Medicare or Medicaid incentive payments.
- http://www.cms.gov/ehrincentiveprograms/01_overview.asp
- 2012 is the last year that a physician can start to receive incentives and obtain the full five years of payments.

EMR, CMS and MU

- The implementation of this policy will be in three steps over the course of the next five years.
 - Stage 1 sets the baseline for electronic data capture and information sharing.
 - Stage 2 (2013) and Stage 3 (2015) will continue to expand on this baseline and be developed through future rule making.
 - CMS expects that Medicare incentive payments will begin in May 2011.
 - Registration for the EHR incentive program will begin in January 2011.

EMR, CMS and MU

- Stage 1: Meaningful use (2011 and 2012) includes meeting both a core set and a menu set of objectives.
 - There are 25 meaningful-use objectives, and 20 must be completed to qualify for an incentive payment.
 - Fifteen are required core objectives, and the remaining five may be chosen from the list of 10 menu set objectives.

Meaningful Use Objectives

- 8. Record and chart changes in the following vital signs for more than 50 percent of all patients age 2 and over:
 - height; weight; blood pressure; calculate and display body mass index (BMI); and plot and display growth charts for children 2-20 years, including BMI.

Case Example

- 48 yr old white female presented for diabetic eye exam on referral from her PCP
 - She was scheduled 2 weeks previously but had fallen and was unable to make that appointment
 - She reports that her vision in her right eye seems to be getting worse over the past several weeks.
 - Upon presentation she was finger count in her right eye and 20/40 in her left with a significant VF defect noted in the left eye.

Case Example

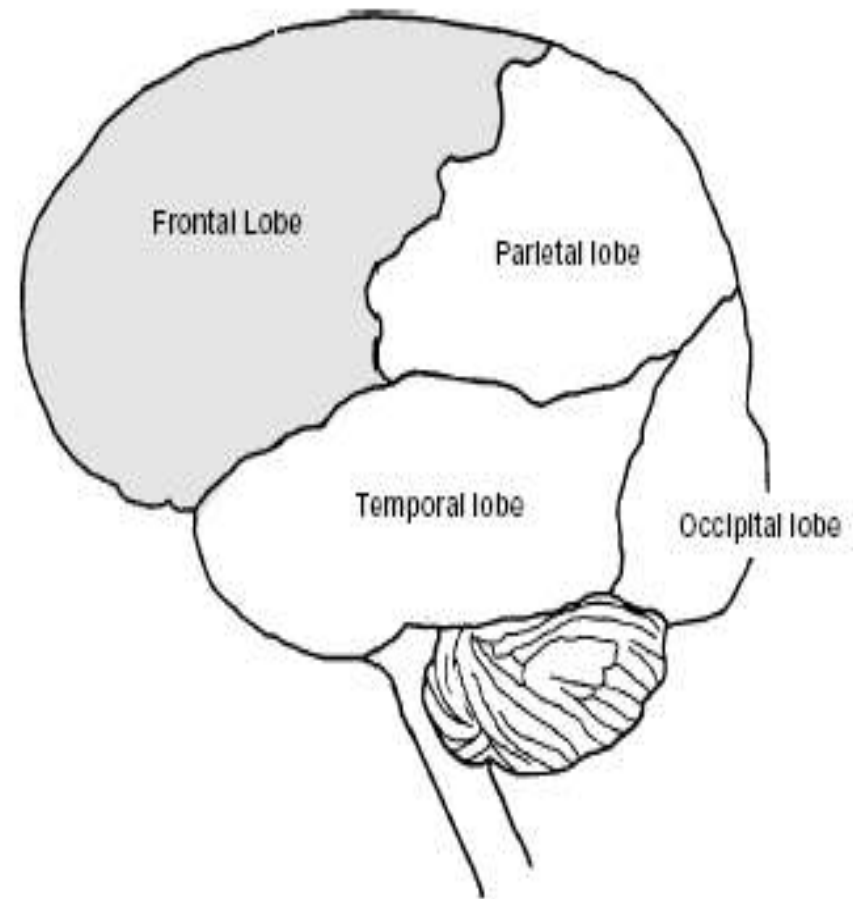
- Upon entering the room I noted that her right hand was twitching
 - I asked her how long that had been going on and she said about 2-3 weeks
 - I asked her if she experienced headaches, to which she said she had bad headaches that even woke her up at night
- Fundus eval revealed bilateral swollen optic nerve heads

Case Example

- Contacted her PCP who told me that she had examined the patient 3 weeks prior and had not noted any of these findings
- Referred the patient for an immediate MRI which wasn't able to be scheduled until the next day
 - MRI revealed large mass in her brain
 - She was referred for immediate brain surgery
 - Neurosurgeon reported that she removed a tangerine sized **Craniopharyngioma**, which was the largest tumor she has ever removed

Neurological Screening: Cerebrum

- Frontal lobe
 - Emotions, drive, affect, self-awareness, and responses related to emotional states
 - Motor cortex associated with voluntary skeletal movement and speech formation (Broca)



Mental Status

- Primarily the function of the Frontal lobe
 - Consciousness
 - How we initiate activity in response to our environment
 - Judgments we make about what occurs in our daily life
 - Controls our emotional response, our expressive language
 - Assigns meaning to words, word association
 - Memory of habits and motor activities
 - “oriented to time and place”

Evaluation of Mental Status

Evaluation of Mental Status
Consciousness/Alertness
Orientation
Memory
Speech
Appropriate affect
Object Recognition
Praxis

Consciousness/Alertness

- The clinician first evaluates alertness, usually while taking the patient's history.
 - Does the patient seem alert and aware?
 - Does she/he seem confused?
 - Is she/he acting in a rational manner
- Decreased alertness indicates dysfunction of both cerebral hemispheres or of the reticular activating system of the brainstem.
- If alertness is sufficient, then it is appropriate to examine and interpret other aspects of the patient's mental status.

Orientation/Memory

- To test for orientation and memory, ask for the patient's full name, the location, and the date
 - often noted as "Alert and oriented to person, place, and time"
 - note the exact response.
- Presenting a set of three common words to the patient and asking him/her to repeat them minutes later can test for short-term memory loss.

Appropriate Affect/Praxis

- Is the patient exhibiting inappropriate emotions, such as laughter or crying?
 - this type of emotional display may be due to bilateral cerebral damage.
- Can the patient understand simple questions and commands?
 - inability to carry out simple instructions is called dyspraxia and may be due to a deep frontal lobe lesion.

Object Recognition

- Ask the patient to name some easy objects (e.g., pen, watch, tie) and some more difficult ones (e.g., fingernail, belt buckle).
- Can the patient close his/her eyes and identify an object by touching it?
 - Inability to do so can indicate a lesion in the non-dominant parietal lobe.

Speech

- Is the patient speaking clearly and using vocabulary appropriately?
- Can the patient repeat single words and sentences (a standard is "No ifs, ands, or buts")?
- These, as well as other kinds of language abnormalities, are usually caused by lesions involving:
 - the dominant (usually left) frontal lobe (Broca's Area),
 - and/or the temporoparietal lobe (Wernicke's Area).
- Multiple deficiencies can signal a global disorder whereas an individual deficit is more likely to signal a more localized lesion.

Mental Status

- For patients with compromised mental status, it is important to document specifically the questions that they were asked and how they were answered.
 - This is the only way to detect changes in mental status when different doctors are following the patient.

Case History: Mental Status

- Inability to take meds properly: dementia
- Attention slipping at work or business
- Grooming and personal hygiene may deteriorate in depression, schizophrenia and dementia

Gradual Onset: Refractive Error

- Unlikely to present acutely but patient may self refer if suddenly noticed decreased acuity
- Signs and symptoms (S&S) include:
 - monocular or binocular blurring of vision without distortion and
 - visual acuity improves with pinhole.

Gradual Onset: Cataracts

- Most common non-refractive cause of visual impairment
- S&S include:
 - misting/blurring of vision,
 - glare,
 - change in refractive error (typically myopic shifts)
- Most common are age related (though congenital, metabolic and traumatic possible)

Gradual Onset: Cataracts

- The decreased acuity must correlate with the severity of the cataract...
 - ie if cataract doesn't correlate with the amount of vision loss (or afferent pupillary defect present) then you need to find another reason for the vision (or other test results)

Signs and Symptoms of Bacterial Conjunctivitis

Clinical presentation – uni- / bi-lateral

Signs:

- Bulbar conjunctival injection
- Purulent discharge
- Morning matting of eyelashes
- Chemosis

Symptoms:

- Photophobia
- Blurred vision
- Tearing



Hyperemia



Chemosis



Purulent discharge

Treatment/Management

- Topical antibiotic therapy
 - Vigamox TID for 7-10 days
 - Moxeza BID for 7 days
 - Category C
 - Zymar q 2 hours for Days 1 and 2, then QID for Days 3-7 (being discontinued)
 - Zymaxid q 2 hours for Day 1, then BID-QID Days 2-7
 - Category C
 - Azasite BID for 2 days then qd for next 7-10 days
 - Category B
 - Besivance TID for 7-10 days
 - Category C
 - Tobramycin/Gentamicin QID for 7-10 days
 - Category B
 - Polytrim q3hrs (max 6x/day) for 7-10 days
 - approved to age of 2 months
 - Category C

Signs and Symptoms of Allergic Conjunctivitis

Clinical presentation – bilateral

Signs:

- Conjunctival edema
- Conjunctival hyperemia
- Chemosis
- Lid edema
- Watery discharge

Symptoms:

- Itching
- Burning
- Photophobia
- Foreign body sensation
- Blurred vision



Lid edema and bilateral hyperemia



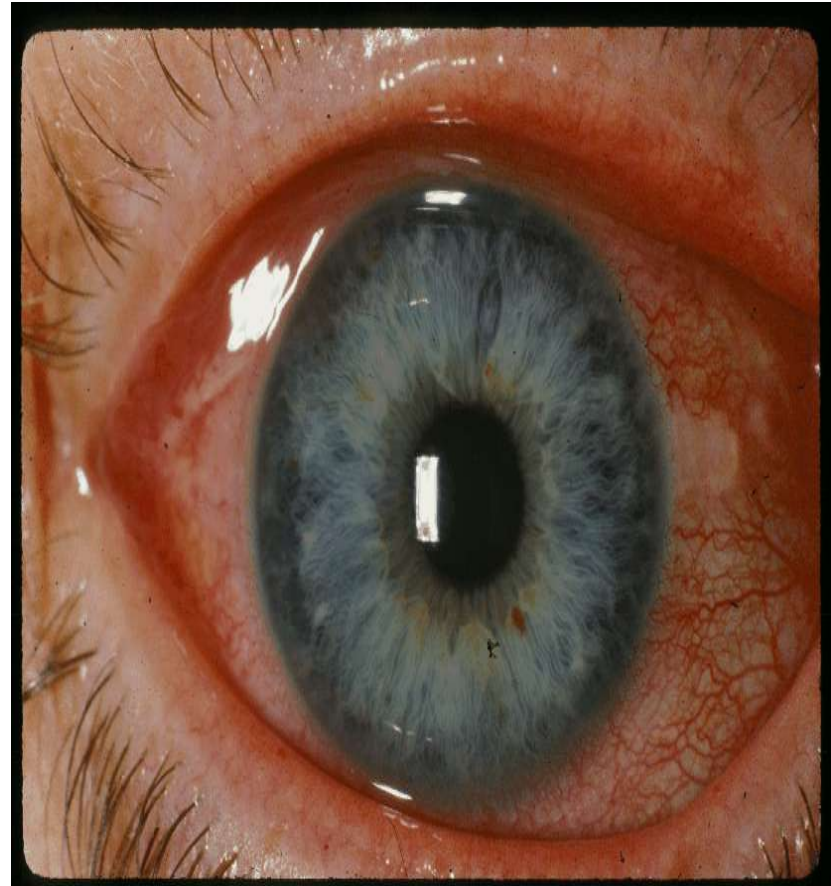
Hyperemia



Chemosis

Viral Conjunctivitis

- Most common infectious keratitis presenting on emergent basis
- 62% caused by adenovirus
- Two major types:
 - Pharyngoconjunctival fever
 - Epidemic keratoconjunctivitis



Viral Conjunctivitis

- PCF: history of recent/current upper respiratory infection
- EKC: highly contagious with a history of coming in contact with someone having a red eye.
 - Adenovirus 8 common variant leading to “rule of 8’s”
 - First 8 days red eye with fine SPK
 - Next 8 days deeper focal epithelial lesions
 - Following 8 potential development of infiltrates
 - Resolution
- RPS Adeno Detector available to use for adenoviral confirmation.

Viral Conjunctivitis: Signs and Symptoms

- Gritty sensation
 - Watery discharge
 - sticky in mornings
 - follicular response
 - Chemosis
 - injection
 - SPK
 - Infiltrates possible
 - Positive lymph nodes
- Pseudomembranes in severe cases
 - Subconjunctival hemes



Management

- Consider the use of anti-inflammatory treatment to relieve patient symptoms and improve comfort
 - Tobradex QID OU
 - Lotemax QID OU
- EKC patients are typically very uncomfortable and would benefit from anti-inflammatory treatment
 - especially if infiltrates or pseudomembrane present

Management

- Betadine (Melton-Thomas Protocol):
 - Proparacaine
 - 1 or 2 drops of NSAID
 - 4-5 drops of Betadine 5%
 - Get patient to close eye and gently roll them around
 - After one minute, lavage the eye
 - Instill another drop of NSAID
 - Lotemax 4 times a day for 4 days

Management

- Antivirals used in HSV keratitis are ineffective in treatment of viral conjunctivitis
 - New Update: in conversation with several colleagues, Zirgan 4-5 times/day has shown significant improvement in patients over a 7-10 time period.
- Important to stress limited contact with others, frequent hand washing, not sharing of towels, etc.

Case History

- 38 black male, complaining that the vision in his right eye is blurry.
 - Got the current Rx 3 weeks previously, and started out good but in last couple of days OD vision has become blurry
- Medical Hx: no current health concerns and no medications

Entrance Skills

- Va's: OD: 20/25, OS: 20/20
- Pupils: PERRL
- CVF: full to finger count
- EOM's: FROM
- Amsler: central metamorphopsia OD
- HVF: 10-2 (see VF)

SINGLE FIELD ANALYSIS

EYE: RIGHT

NAME: ██████████

ID: 110724

DOB: 03-08-1969

CENTRAL 10-2 THRESHOLD TEST

FIXATION MONITOR: GAZE-BLINDSPOT

STIMULUS: III, WHITE

PUPIL DIAMETER: 5.5 MM

DATE: 05-19-2007

FIXATION TARGET: CENTRAL

BACKGROUND: 31.5 ASB

VISUAL ACUITY:

TIME: 10:07 AM

FIXATION LOSSES: 2/15

STRATEGY: SITA-STANDARD

RX: +4.00 DS -2.00 DC X 115

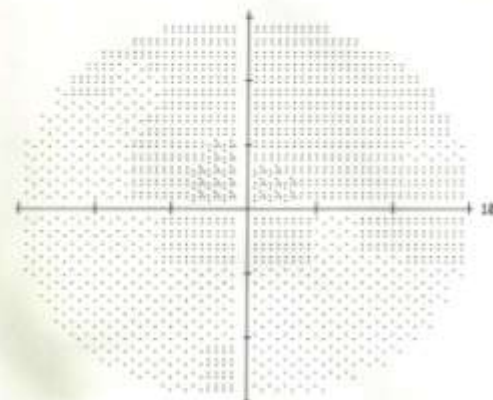
AGE: 38

FALSE POS ERRORS: 0 %

FALSE NEG ERRORS: 0 %

TEST DURATION: 00:32

FOVER: OFF



TOTAL
DEVIATION



11 < 5L
11 < 2L
11 < 1X



PATTERN
DEVIATION



MD -3.12 DS P < 2L
PFD 2.40 DS P < 1X

PACIFIC UNIVERSITY FAMILY EYE CENTER
511 SW 10TH AVE
PORTLAND, OR



Case

- 65 year old Caucasian patient presents with sudden onset loss/blurring of vision in the right eye
- PMHx: HTN for 15 years, takes “water pill”
- VA’s: 20/60 OD, 20/25 OS
- Pupils: PERRL –APD
- CVF: Inferior defect right eye, no defects noted in the left eye

Vision Loss Without Pain: Diabetes/Diabetic Retinopathy

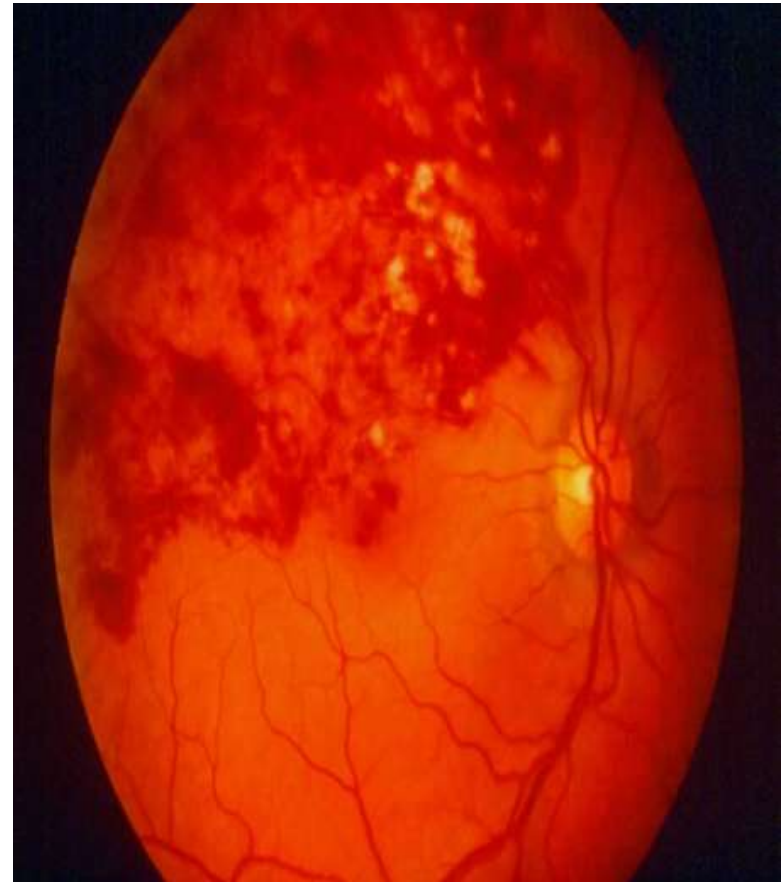
- Microvascular complications resulting in capillary closure & abnormal permeability
- S&S include;
 - blurring of vision (maculopathy and refractive error shifts),
 - sudden drop in vision (vitreous heme),
 - dot and blot hemes,
 - exudate,
 - cotton wool spots,
 - neovascularization (iris, retina and disc)

Vision Loss Without Pain: Vein Occlusion

- Associated with:
 - hypertension,
 - coronary artery disease,
 - DM and
 - peripheral vascular disease.
- Usually seen in elderly patients (60-70), slight male and hyperopic predilection.
- Second most common vascular disease after diabetic retinopathy.

Branch Retinal Vein Occlusion: Signs/Symptoms

- BRVO: sudden, painless, visual field defect.
 - patients may have normal vision.
 - quadrantic VF defect,
 - dilated tortuous retinal veins with superficial hemes and CWS
 - typically occurs at A/V crossing (sup/temp)



BRVO

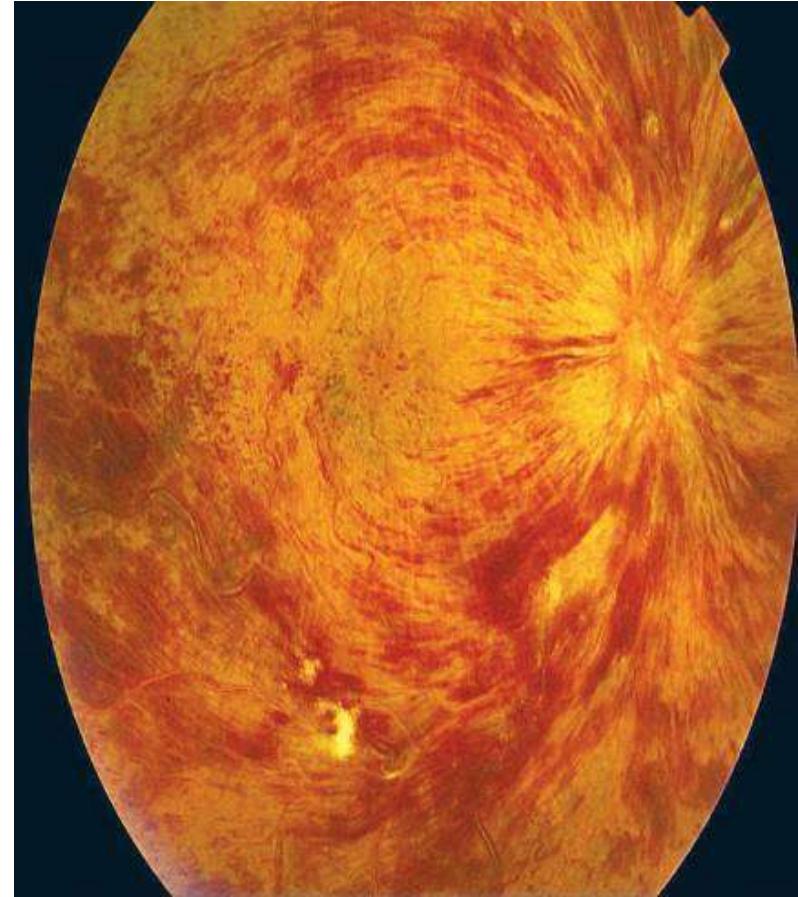
- BRVO more common than CRVO and has more favorable prognosis
 - Overall 50-60% of BRVO patients will maintain VA of 20/40 or better
- Visual loss results from:
 - Macular edema
 - Foveal hemorrhage
 - Vitreous heme
 - Epiretinal membrane
 - RD
 - Macular ischemia
 - Neovascularization complications

BRVO: Treatments

Macular grid laser	can result in improved VA in patients with chronic macular edema secondary to BRVO
Intravitreal corticosteroids	SCORE (Standard Care vs. Corticosteroid for Retinal Vein Occlusion) showed improvement equivalent to laser however more complications
Anti-VEGF	has shown beneficial effects in resolving macular edema and Lucentis is currently approved for treatment

Central Retinal Vein Occlusion: Signs/Symptoms

- CRVO: thrombus occurring at lamina is classical theory but new evidence indicates that the occlusion is typically in the optic nerve posterior to the lamina cribrosa
 - decreased VA ranging from near normal to hand motion with majority 20/200 range
 - dilated tortuous vessels, with numerous retinal hemes and CWS



Central Retinal Vein Occlusion

- Visual morbidity and blindness are primarily from:
 - persistent macular edema,
 - macular ischemia and
 - neovascular glaucoma

Central Retinal Vein Occlusion

- CRVO's can be ischemic or non.
 - Classical definition of ischemic is 10-disc area of non-perfusion found on angiography
 - RAPD and ERG maybe better predictor
 - VA's typically worse in ischemic
 - Increased number of cotton wool spots with decreased VA maybe predictive

Central Retinal Vein Occlusion

- Ischemic CRVO may lead to iris neovascularization and neovascular glaucoma
 - Estimated apprx 20% of CRVO's are ischemic with 45% of those developing neo
- Regular examinations (1-2 wks) to monitor for ischemia or neo development
 - should include gonio as angle neo can precede iris rubeosis

CRVO Treatments

PRP	indicated for iris neo (>90% have neo regression and lowers risk of neo glaucoma to 1%)
Grid Macular Laser	not effective in improving VA in eyes with macular edema though is effective in BRVO macular edema
Hemodilution	remains inconclusive
Medical Treatment	oral or systemic anticoagulant or rheological agents have shown limited evidence of improvement with potential serious side effects

CRVO Treatments

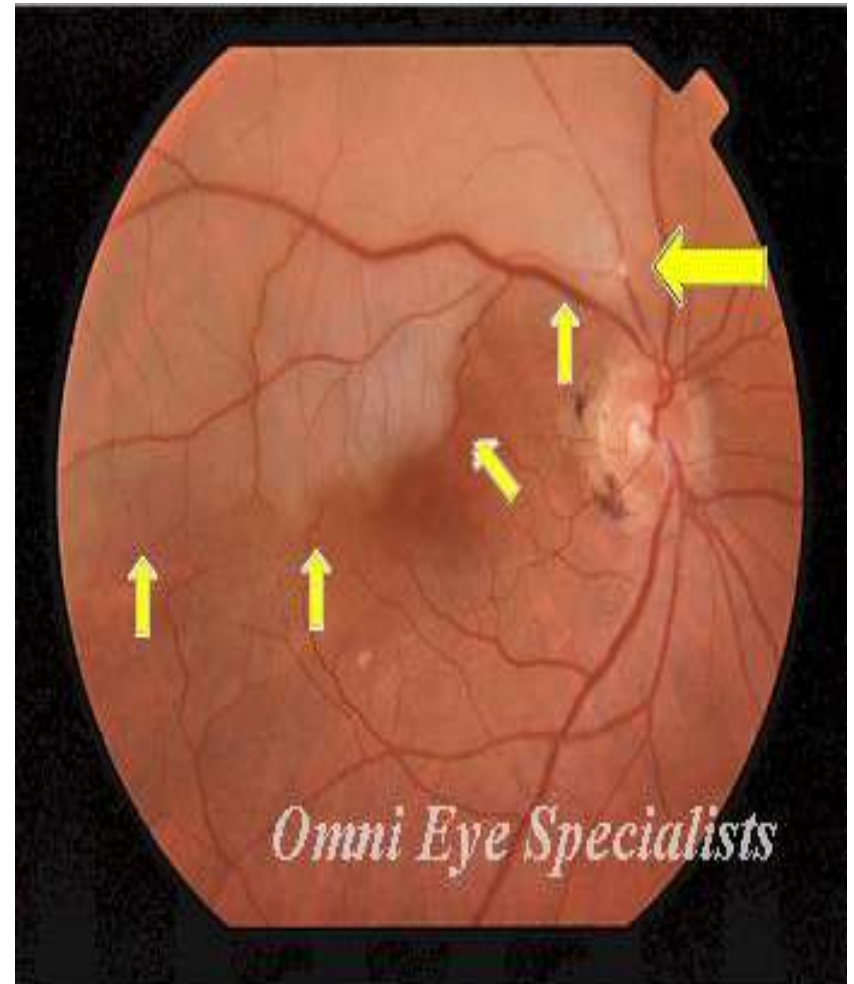
Intravitreal corticosteroids	<p>SCORE (Standard Care vs Corticosteroid for Retinal Vein Occlusion Study)</p> <ul style="list-style-type: none">- showed significant improvement long term for both CRVO and BRVO associated edema though potentially more complications
Anti-VEGF	<p>has shown promising for both macular edema and iris neo</p> <ul style="list-style-type: none">- Found beneficial for CRVO macular edema- RAVE (Rubeosis Anti-VEGF) trial is looking at complete VEGF blockage in preventing neo glaucoma in ischemic CRVO
RON (Radial Optic Neurotomy)	<p>debate on mechanism of action and efficacy</p> <p>Currently insufficient evidence to recommend</p>

Vision Loss Without Pain: Artery Occlusion

- Primarily embolic in nature from cholesterol, calcifications, plaques.
- Usually occurs in elderly associated with:
 - hypertension (67%),
 - carotid occlusive disease (25%),
 - DM (33%) and
 - cardiac valvular disease.
- Sudden loss of unilateral, painless vision
 - defect dependent upon location of occlusion

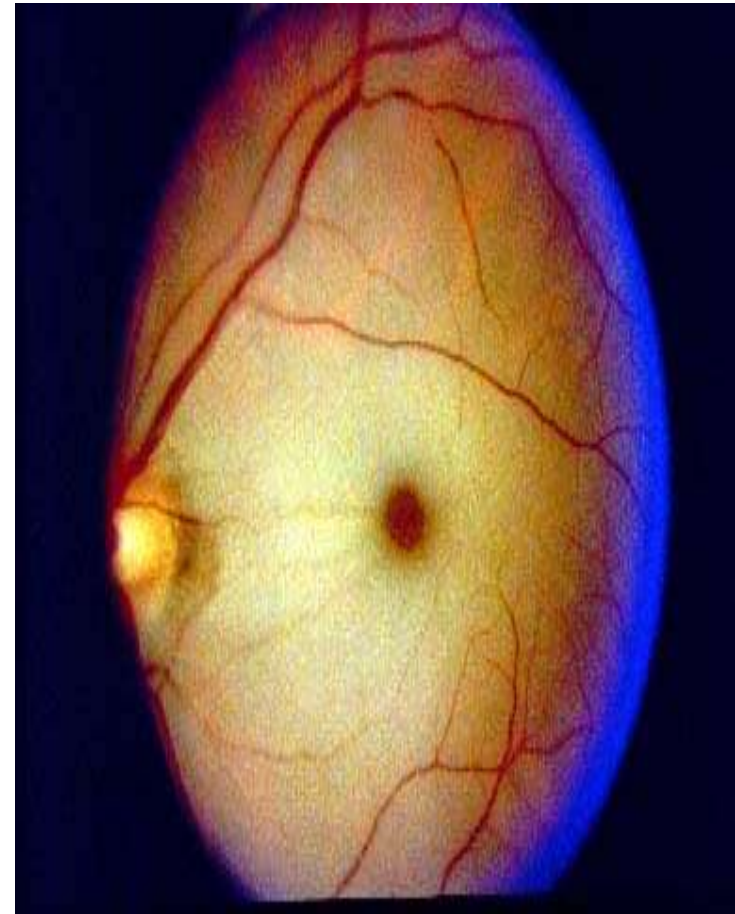
Vision Loss Without Pain: Artery Occlusion

- BRAO typically located in temporal retinal bifurcations.



CRAO

- CRAO has profound vision loss with history of amaurosis fugax.
 - Vision is usually CF(count fingers) to LP (light perception) with positive APD.
 - Diffuse retinal whitening with arteriole constriction, cherry red macula.



Ophthalmic Emergency

- Treatment is controversial due to poor prognosis and questionable benefit.
- Treat immediately before workup, if patient presents within 24 hours of visual loss:
 - Digital ocular massage,
 - systemic acetazolamide (500 mg IV or po),
 - topical ocular hypertensive drops (lopidine, B-blocker),
 - anterior chamber paracentesis,
 - consider admission to hospital for carbogen Tx (high carbon dioxide)

Vision Loss Without Pain: TIA/TMB/Amaurosis Fugax

- Refers to temporary visual impairment of variable duration (seconds to hours)
 - TIA: transient ischemic attack-can be cerebral or retinal
 - TMB: transient monocular blindness secondary to a retinal TIA
 - Amaurosis Fugax: same as TMB
- Abrupt onset, progression to involve all or part of visual field, sight usually returns
- Within affected area, visual acuity maybe dimmed or completely lost

TIA's

- Stroke is 3rd leading cause of mortality in developed countries and most common cause of neurological disability
- 15-20% of patients with stroke have a preceding TIA, though guidelines for referral and evaluation are debated
 - Traditional guidelines suggested that assessment should be complete within 1 week of TIA

TIA's

- Risk of stroke after TIA has traditionally been considered relatively low, but
 - new studies indicate that the risk is much higher than previously thought and the time window for prevention is short.
- Effective secondary prevention depends on reliable identification of those at high risk and targeting treatment.

TIA's: High Risk Factors

- Five (5) risk factors are associated with a high risk (30%) of recurrent stroke at 3 months:
 - Age over 60
 - Symptom duration greater than 10 minutes
 - Motor weakness
 - Speech impairment
 - Diabetes
- Isolated sensory or visual symptoms were associated with low risk of stroke!

TIA: Early Treatment

- Several treatments are likely to be effective in preventing stroke in the acute phase after a TIA:
 - Aspirin
 - Anticoagulants
 - Statins
 - Endarterectomy (for >50% carotid stenosis)
 - Further research needed for:
 - Lowering blood pressure acutely after TIA
 - Prophylactic use of neuroprotective drugs

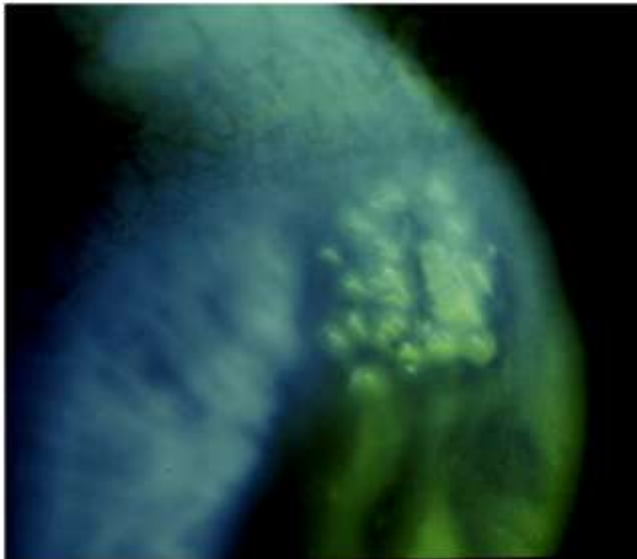
Amaurosis Fugax:TMB

- Most common cause is:
 - thromboembolic disease (eg carotid artery disease throwing emboli) or
 - vasospasm
- Described as “curtain falling over vision”
- Risk of stroke or death is about 3-5%,
 - which is significantly lower than for a cerebral TIA (15-20%)
- Px still require work-up to determine cause:
 - e.g. carotid doppler

Recurrent Corneal Erosion: Treatment

- If severe enough to cause vision loss or repeated episodes:
 - oral doxycycline with/without topical corticosteroid
 - Doxy 50 mg bid and FML tid for 4-8 weeks
 - both meds inhibit key metalloproteinases important in disease pathogenesis
 - Azasite (topical azithromycin)
 - debridement,
 - stromal puncture, or
 - PTK

Stromal Puncture



PEPSI

5 most common causes of Angiod Streaks:

P: Pseudo-Xanthoma Elasticum (PXE)

E: Ehlers-Danlos

P: Paget's Disease

S: Sickle Cell

I: Idiopathic

Vision Loss without Pain: Anterior Ischemic Optic Neuropathy

- Ischemic infarction of anterior optic nerve due to occlusion of posterior ciliary circulation just behind lamina.
- Two types:
 - Arteritic: giant cell arteritis/temporal arteritis
 - Non-arteritic/idiopathic: associated with hypertension (40%) and diabetes (20%)

Epidemiology

- Nonarteritic: usually seen in younger patients
 - Fellow eye involved in 25-40% of cases
 - Associated with hypertension and diabetes



Epidemiology

- Arteritic: usually seen in >55 yrs old (mostly over 70)
 - fellow eye involved in 75% of cases within 2 weeks without treatment

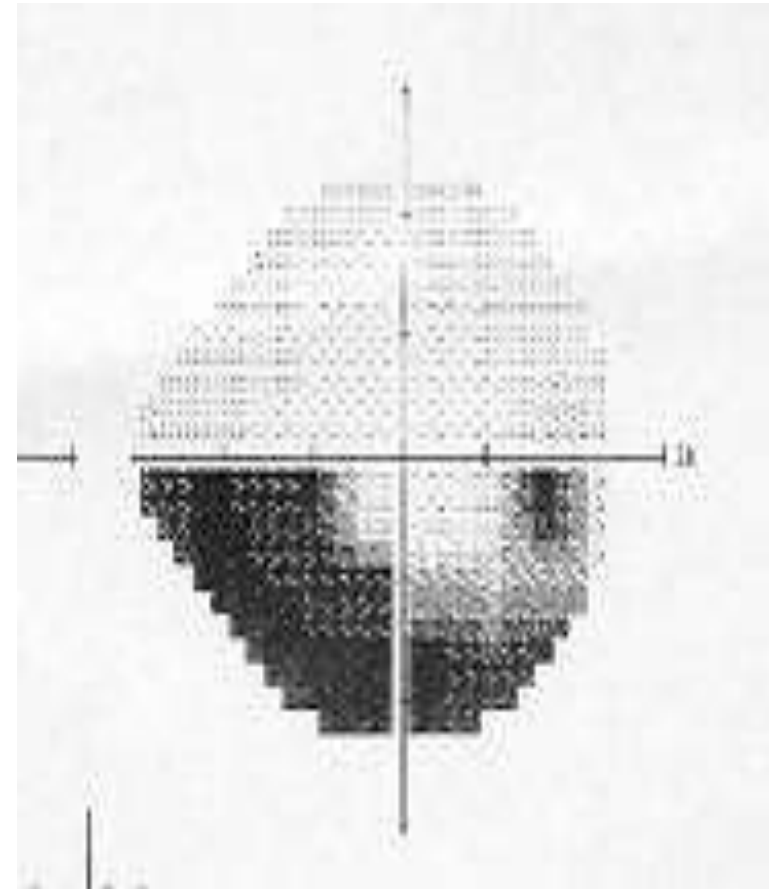


Symptoms

- Acute visual loss (arteritic>non)
- dyschromatopsia
- Arteritic may also have associated:
 - Headache, fever, malaise,
 - weight loss, scalp tenderness, jaw claudication,
 - amaurosis fugax, diplopia, and eye pain.

Ocular Signs

- Sudden, unilateral, painless decreased vision and color vision
- Positive RAPD
- Altitudinal visual field defect (usually inferior and large)
- Swollen optic disc
- Fellow nerve often crowded with small or absent cup (“disc at risk”)



Ocular Signs: Arteritic

- Arteritic may also have:
 - Swollen, tender temporal artery
 - Cotton wool spots
 - BRAO or CRAO
 - Ophthalmic artery occlusion
 - Cranial nerve palsy (primarily VI)
 - Optic disc cupping is seen late

Additional Testing

- Lab tests:
 - STAT ESR (rule out arteritic form)
 - CBC (low hematocrit, high platelets)
 - Fasting blood sugar
 - C reactive protein,
 - VDRL/FTA-ABS
 - ANA
- Check blood pressure

Additional Testing

- Arteritic: consider artery biopsy
 - Will remain positive up to 2 weeks after starting steroid treatment
- Consider FLAN: choroidal non perfusion in arteritic form
- Medical consult

Management

- Arteritic:
 - Systemic steroids to prevent fellow eye involvement
 - methylprednisolone 1 g IV qd in divided doses for 3 days then,
 - prednisone 60-100 mg po qd with a slow taper
 - Check PPD, blood glc and chest radiographs before starting systemic steroids
- Non-arteritic:
 - Consider daily aspirin

Preseptal Cellulitis

- Infection and inflammation located anterior to the orbital septum and limited to the superficial periorbital tissues and eyelids.
- Usually follows periorbital trauma or dermal infection (suspect staph sp in trauma).
- Eyelid swelling, redness, ptosis, pain and low grade fever.
- Tx *Augmentin 500/125 mg TID or 875/125 mg BID for 7-10 days* or if moderate to severe IV Fortaz (ceftazidime) 1-2 g q8h.



Cephalosporin: Ocular Indications

- ***Orbital Cellulitis***: Infection and inflammation within the orbital cavity producing orbital S&S.
 - most commonly secondary to ethmoid sinusitis. Staph and Strept most common isolates.
 - decreased VA, pain, red eye, HA, diplopia, bulging eye, APD, EOM restriction, lid swelling and fever.
- ***IV ceftriaxone (Rocephin) 50 mg/kg/Q12h/day or***
- ***IV cefotaxime (Claphoran)50 mg/kg/Q6h/day***
 - plus IV vancomycin 30 mg/kg/day in 3 doses infused over 90 minutes (penicillin allergy) or
 - IV clindamycin 40 mg/kg/day in 3 doses

