CASE: THE WORLD’S BEST CONJUNCTIVITIS

CC: 63 YOWF - Referred for “non-specific conjunctivitis”
- The best conjunctivitis that she ever had!

Medical Hx: Unremarkable

Conjunctivitis treated successfully by Attending & Resident:
- Concern over funny lid positioning
- “Consider MG evaluation"

Key Finding: Pictured

CASE: 63 YEAR OLD WHITE FEMALE

What questions do you want to ask?
What tests do you want to perform?

CASE: 63 YEAR OLD WHITE FEMALE

Do you ever have double vision? Yes!
- In extreme gaze up, down, right, and left

Ocular motility findings:
- Abduction, adduction, elevation, and depression deficits

Forced duction testing: Equivocal

“This is not a boating accident!”
- And it isn’t myasthenia gravis either!

Preliminary diagnosis?
What tests do you want to order?
CASE: 63 YEAR OLD WHITE FEMALE

- Presumptive diagnosis:
  Primary aberrant regeneration of CN III from lesion in cavernous sinus
- Plan:
  Refer for MRI of orbits and chiasm with detail to cavernous sinus/parasellar area

CN III PALSY: ABERRANT REGENERATION

- Damage to CN III results in resprouting and miscommunication of nerves to muscles
  - Inferior rectus and medial rectus communicates with levator
  - Medial rectus communicates with pupil
- Clinical picture:
  - Patient looks medial: lid elevates
  - Patient looks lateral: lid lowers
  - Patient looks down: lid elevates (Pseudo-Von Graefe’s)
  - Patient looks medial: pupil constricts

CN III PALSY: ABERRANT REGENERATION

Primary: Occurs independent of antecedent CN III Palsy. Caused by aneurysm or meningioma within cavernous sinus
  - Slow growing with subclinical compression and regeneration concurrently
Secondary: Occurs after an antecedent CN III palsy. Causes:
  - Aneurysm, trauma, tumor, inflammation
  - NEVER DIABETES!!
    - If cause of CN III palsy is determined to be ischemic vascular and then the eye undergoes aberrant regeneration, the initial diagnosis is wrong. You must re-examine for tumor or aneurysm within ipsilateral cavernous sinus.

CASE: 63 YEAR OLD WHITE FEMALE

- MRI results: Cavernous sinus “pristine and perfect”
  - HOWEVER, soft tissue mass seen in orbit.
  - CN III aberrant regeneration? No!
  - Diagnosis: probable orbital malignancy
  - Primary care evaluation: Breast carcinoma
  - Orbital biopsy: Metastatic carcinoma

OCULAR/ORBITAL METASTASIS

Metastatic cancer – spread from one system to another via blood or lymphatic channels

Most common primary tumor sites:
  - Breast > Lung > GU tract > GI tract > Skin

Most common ocular metastasis sites:
  - Choroid > Orbit > Iris > Lids > Optic nerve

The discovery of ocular metastasis is an exceedingly poor prognostic indicator…
MANAGEMENT OF OCULAR METASTASIS

- Treatment is palliative
- Modalities include:
  - Concurrent chemotherapy
  - Irradiation
  - Local excision
  - Enucleation / exenteration
- Despite therapy, average survival is 7-9 mos.
- Outcome of this patient?

Case 2:

A 37 year-old female presented with blurry vision in her left eye after being struck to the side of her left globe while playing with her 6 year-old son 2 days prior
- She denied any redness, pain or nausea.
- Her medical history was negative and she was taking no medications.
- She occasionally wore reading glasses.

Case

- Pupils, motilities and visual fields were all unremarkable
- Uncorrected VA OD: 20/20
  - OS: CF@4Ft
- BVA OD: 20/20 +0.50-0.50x170
  - OS: 20/20

Case

- Pupils, motilities and visual fields were all unremarkable
- Uncorrected VA OD: 20/20
  - OS: CF@4Ft
- BVA OD: 20/20 +0.50-0.50x170
  - OS: 20/20 -5.50-0.75x185
Case

- No ecchymosis was present
- No evidence of subconjunctival hemorrhage or corneal injury
- No cell, flare or hyphema present
- Anterior chamber was deep and quiet OD
- OS anterior chamber:

Case

- IOP: OD: 13 mmHg
  OS: 5 mmHg
- Seidel test was negative
- There was no evidence of iridodialysis, iridodonesis or phakodonesis OS.
- Gonioscopy??
- Should we Dilate?

OCT

Thoughts?
**Blunt Trauma**
- Common complications of Blunt Trauma to internal anterior segment
  - Traumatic hyphema
  - Iritis
  - Hypotony
  - Mydriasis
  - Iridodialysis
  - Phakodonesis
  - Lens subluxation
  - Cyclodialysis
  - Angle recession

**Cyclodialysis Cleft**
- Separation of the Ciliary Muscle from the Scleral Spur
  - Aqueous drains into the suprachoroidal space
    - Chronic Hypotony
  - Clinical observation (gonio)
    - UBM
    - Anterior segment OCT
- In contrast – Angle recession is a tear in the ciliary muscle between the circular and longitudinal layers but the longitudinal muscle is still attached

**Angle Recession**
- Longitudinal fibers still attached to the scleral spur

**Cyclodialysis Cleft**
What Develops?

- Ciliary effusion
  - Abnormal collection of fluid in the subarachnoid space
    - May be medication induced (likely bilateral)
      - Topamax (topiramate)
    - Typically caused by Hypotony
    - May develop choroidal folds
  - Aqueous production is reduced
    - Perpetuates Hypotony
      - Uveal scleral outflow is enhanced
        - Prostaglandin release?
  - “A soft eye is a sick eye”

Ciliary Effusion

- Anterior Rotation of the Ciliary Body
  - Reduces tension on the zonules
    - Lens Thickening
      - Induces myopia
    - Iris-Lens diaphragm shifts anteriorly
      - Induces myopia by changing effectivity
        - $P = \frac{F}{1+dF}$
    - Shallowing of Anterior Chamber
      - Potential for angle closure

Ciliary Effusion

- Diagnostic Management
  - UBM Preferred
  - Anterior segment OCT
  - Clinical observation
- Treatment
  - Cycloplegics
    - Atropine
  - Anti-inflammatories
    - Steroids? If cleft present
      - Topical
      - Oral
    - NSAIDS

Treatment

- Treated with Atropine 1% BID OS and Prednisolone Acetate 1% QID OS and was reappointed the next day.
- OS: $-1.25 - 0.75 \times 165$ 20/20
  - Exhibiting a 4 diopter hyperopic shift in 1 day
  - Anterior chamber less shallow
- IOP: OS 5 mmHg
Day 1 Follow-up

Treatment
- Taper began at 6 weeks
- Final refraction and BVA
  - OD: +0.50-0.50x170 20/20
  - OS: +0.50-0.75x165 20/20
- IOP was 14 mmHg OU.

Final Follow-up

Any Questions???????
Case History

- Hispanic female, 2nd decade of life
- CC: Loss of vision in the right eye x 1-2 months
- Systemic Hx: Unremarkable

Case 3
My patient has an APD. Now what?

Pertinent findings

- VA OD 20/100 OS 20/20
- PUPILS + APD OD
- MOTILITIES Full and smooth OU (-) pain
- CF: FTFC OU

What next?

Pupillary Light Reflex Pathway

Afferent Pathway
Communication
Efferent Pathway

Healthy Eye

— Afferent Pathway
— Communication
— Efferent Pathway

Construction
Less or no constriction
Unilateral severe damage:

- Optic neuropathy
  - Unilateral
    - Bilateral AND Significantly Asymmetrical
- Severe retinal defect
  - eg. RD, vascular occlusion, large macular scar
- Severe amblyopia
  - mild RAPD
  - Dx of exclusion
- Severe cataract
  - Inverse RAPD
  - Dx of exclusion

Findings

Photos

- ONH:
  Borders distinct, pink, flat
  (-) pallor (-)edema
  Healthy rim tissue

- Macula:
  Flat and healthy

- Retina:
  Flat x 360 deg, no RD

Photos

- ONH:
  Borders distinct, pink, flat
  (-) pallor (-)edema
  Healthy rim tissue

- Macula:
  Flat and healthy

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  - Dx of exclusion

Visual Fields

Where is the lesion most likely located?

Retina
- Unilateral
- Bilateral

Neurological
- Retrobulbar?

SD-OCT

What next?
Ocular motilities

Pain when looking to the left, and when looking up

Right Eye

Left Eye

The superior and medial recti muscles are much more closely attached to the dural sheath of the optic nerve

Characteristic pain in extreme eye movements in retrobulbar neuritis, especially in adduction and superior gaze

Unilateral severe damage:

- Optic neuropathy
  - Unilateral
  - Bilateral AND Significantly Asymmetrical

- Severe retinal defect
  - e.g. RD, vascular occlusion, large macular scar

- Severe amblyopia
  - mild RAPD
  - Dx of exclusion

- Severe cataract
  - - Intense RAPD
  - - Dx of exclusion

SD-OCT

Ganglion Cell Complex (GCC)

GCC at the Macula

Has over 50% of all retinal ganglion cells

GCC (all three layers) becomes thinner as Ganglion Cells die
What ARE the rnfl thickness findings?

Isolated ON  
MSON

RNFL Thickness  
Isolated ON

NFL Thinning: Rate of progression

RNFL Thickness  
MSON  
- Loss of RNFL thickness

DDX  
Isolated ON vs MSON ??  
- NOT according to extent of thinning

MS without ON

MSON  
- NO prior H/O ON  
RNFL thinning in both eyes

DDX  
Isolated ON vs MSON  
- RNFL thinning in both eyes

H/O ON 3 years before
Correlation between RNFL thinning and GCIP thinning

Threshold of 75 µm of RNFL thickness
Corresponding Decrease in Visual Recovery

1 - Asymptomatic episodes of subclinical ON
Mechanism
2 - Retrograde trans-synaptical degeneration

Mechanism
3 - Primary neurodegeneration in the absence of inflammation

MULTIPLE SCLEROSIS
Potential Use:
1) Establish a diagnosis in symptomatic patients
2) Screening in asymptomatic patients
3) Provide prognostic data in established patients
4) Monitor efficacy of therapy
5) Monitor progression of disease w/ or w/o therapy
6) More insight in structure - function relations & more understanding in pathophysiology

Impression/Plan
• Retrobulbar Neuritis
  • Referred to neuro-ophthalmology
  • Confirmed dx
  • MRI of brain
  • Result: Demyelinating disease (likely MS)

Case 4:
35 YEAR OLD MAN
• Wants another opinion due to “hemorrhage on my right eye”
• Happened 3 days ago after vomiting
  - Claims food poisoning from chicken Caesar salad
  - Still feels a little nauseated
• Saw ophthalmologist 3 days ago, told he had a bruise on his eye and it should go away in 1-2 weeks
35 YEAR OLD MAN

BVA 20/100 OD, 20/70 OS
- Hx of amblyopia OD
- Current Rx OD +5.50 OS +4.50

Any concerns?

Patient noticed blurry vision OS
- Started 2 weeks ago
- Did not mention because he is more concerned about the blood on his right eye

Headaches for 2 weeks, decrease if patient stands up

ROS: unremarkable

Decide to dilate OU

RETINAL FINDINGS

DISCUSSION

DIFFERENTIAL DIAGNOSIS

- Hypertensive retinopathy
- Blood dyscrasia
- Terson’s syndrome
- Valsalva retinopathy
- Purtscher’s retinopathy
- Shaken baby syndrome

TERSON’S SYNDROME

- Terson’s syndrome originally was defined by the occurrence of vitreous hemorrhage in association with subarachnoid hemorrhage.
- Terson’s syndrome now encompasses any intraocular hemorrhage associated with intracranial hemorrhage and elevated intracranial pressures.
- Intraocular hemorrhage includes the development of subretinal, retinal, subhyaloidal, or vitreal blood.
- The classic presentation is in the subhyaloidal space.

TREATMENT

- Emergency referral to neurologist due to high suspicion of intracranial hemorrhage and elevated intracranial pressure
- Intracranial hemorrhage confirmed with MRI
- Patient later diagnosed with Hairy Cell Leukemia and cryptococcal meningitis

Case 5:
47 YEAR FEMALE

CC: Horizontal double vision in far left gaze
BVA: 20/20 OD, OS
Medical Hx: newly diagnosed diabetes
Left abduction deficit in far left gaze
• Negative forced duction test
Mild ocular injection OS
IOP: 14 mm Hg OD, 16 mm Hg OS
Fundus: normal OU

Thoughts?

47 YEAR OLD BLACK FEMALE

• Presumptive diagnosis: Left vasculogenic CN VI palsy - monitor
• Returns 1 week with marked worsening of injection, diplopia and ophthalmoplegia
• IOP: 16 mm Hg, 26 mm Hg
• Fundus disc congestion and vascular tortuosity OS

What does she look like NOW?
What do you want to do NOW?

47 YEAR OLD BLACK FEMALE

CT scan:

What do you think NOW?

CAROTID CAVERNOUS SINUS FISTULA

Cavernous sinus...
• Trabeculated venous cavern
• Houses CN III, IV, VI, V1, oculosympathetics, and ICA
• Drains eye and Adnexa via inferior and superior ophthalmic veins to petrosal sinuses and jugular vein

Fistula...
• Rupture of ICA or meningeal branches within sinus
  • Meningeohypophyseal, McConnell’s Capsular, Inferior Cavernous
  • Mixing of arterial blood in venous system
CAROTID CAVERNOUS SINUS FISTULA

Hemodynamic
- High flow vs low flow

Angiographic
- ICA vs meningeal branches

Etiology
- spontaneous vs traumatic

- Increased venous pressure
- Orbital congestion
- Proptosis (pulsatile)
- Corneal exposure
- Arteriolization
- Orbital bruit
- Myopathies and cranial neuropathies with diplopia
- Secondary glaucoma
CAROTID CAVERNOUS SINUS FISTULA

Vision threatening – not life threatening
Spontaneous etiology – spontaneous resolution
  • ICA compression with contralateral hand
Traumatic – clipping and ligation
Balloon or particulate embolization
Manage glaucoma aggressively
  • Prostaglandin analogs

RULE: BEWARE THE CHRONIC RED EYE

- Dilated & tortuous episcleral vessels that go to the limbus and back (omega loops)
- Intervening “clear conjunctiva”
- Red eye that doesn’t respond to any topical treatments
  - Bag-o-Meds
- Other non-red eye findings: Chemosis, IOP elevation, proptosis, ophthalmoplegia, ptosis, lid edema

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ODE TO A FISTULA

Beware the chronic red eye
It isn’t infected, inflamed, or dry.
When corkscrew vessels makes the eye reds
And the patient has bag-o-meds.
The problem is deep
And arterial blood has begun to seep.
Your first fistula you will always miss
But on your second case you will never be remiss

Joseph Sowka, OD

Case 6:

Case

A 52 year-old Hispanic male complained of a blind spot in his left eye for 3 weeks
PMHx (+) GERD for which he was recently prescribed Omeprazole
• Hernia repair in 2010
• General health was unremarkable with no recent illness or fever
• No recent travel reported
• POHx non-contributory

Case

BVA OD: 20/20
OS: 20/20 -3 (patient stated only letters on the right of the line were visible)
Pupils: Isocoric with 1+ APD OS
VF: OD: FTFD
OS: Temporal Constriction
HVF OS: Enlarged Blindspot
Amsler Grid: OD: Normal
OS: Temporal scotoma noted

Case

BP 115/70 mmHg
Color Vision was normal in each eye
Anterior segment was unremarkable
• No Cells or flare present in A/C OU
IOP OD: 15 mmHg
OS: 16 mmHg
Case

- Posterior Segment:
  OD: Unremarkable
  OS: Elevated ONH with hemorrhages
  Retinal Folds were visible
  No Macular Star formation present
  No Vitritis Present

Neuroretinitis
Elevated Optic Nerve with intraretinal hemorrhages. Note the retinal striations representing serous fluid. There is no macular star lipid formation present.

Initial Presenting VF, note the enlarged blind spot in the left eye

OCT illustrating an edematous optic nerve head

Thoughts?
Patient reported exposure to a new litter of kittens in his house
Diagnosis
Neuroretinitis secondary to Bartonella henselae
(Cat Scratch Disease)

Background
Cat Scratch disease is a systemic infection by the gram-negative Bartonella bacillus. A detailed medical history including recent travel, animal exposure, skin lesions and general health is necessary to facilitate the diagnosis.

Neuroretinitis
Neuroretinitis is an inflammation of the optic nerve, leading to optic nerve swelling, and surrounding retina resulting in a serous detachment that typically involves lipid deposition resulting in a macular star formation.

Cat Scratch Disease is the most common associated etiology of infectious neuroretinitis

Cat Scratch Disease (CSD)
- Epidemiology
  - Caused by the Bartonella gram-negative bacillus
    - Typically Bartonella henselae
  - CSD is found worldwide and associated with domestic and feral cats
  - About 40% of cats may be infected
  - Typically age 18 and younger with slightly greater incidence in males
  - Hospital admissions more common in adults
  - Seasonality peak incidence in Fall-Winter in US

Clinical Manifestations (CSD)
- Typically self-limiting and benign infection
- Linear scratch abrasion developing a pustule lesion
- Lymphoreticulosis - Lymphadenopathy is most common involving node closest to drainage of lesion
- Flu-like symptoms
  - Malaise
  - Fatigue
  - Fever
- Immuno-compromised patients are at great risk of systemic and ocular involvement
Ocular Manifestations (CSD)
Anterior Segment
- Parinaud’s Oculoglandular Syndrome
- Anterior uveitis might be present

Ocular Manifestations (CSD)
Posterior Segment
- Neuroretinitis
  - Sudden Painless Vision Loss
  - Typically unilateral but may be bilateral
  - ONH Swelling
    - Intra retinal hemorrhages may be present
    - Macular exudative star may form within first 3 weeks
      - Lipid rich fluid leakage has been demonstrated to originate from an optic disc vessel accumulating in a star or radial pattern in the outer plexiform layer
  - VA can range from 20/20 to LP
  - Mild to moderate Vitritis may be present

Less Common Posterior Segment
- White Dot Syndrome has been associated
- Venous and Arterial occlusions have been reported

Lipid-rich fluid flows from an optic disc vessel and accumulates in the outer plexiform layer forming the star or radial pattern
Making the Diagnosis

- Thorough case history
  - Inquire about pet exposure
    - Cats and kittens
  - Suspicious skin lesions
    - Flea bites
  - Recent travel

- Clinical Presentation
  - Presence of neuroretinitis is highly suspicious of CSD
  - OCT is helpful in detecting early serous elevation

- Lab Testing
  - Bartonella henselae serum antibody titer
    - B. henselae IgG
    - B. henselae IgM

Top Differential Diagnosis

The following should be considered in the work-up:

- Toxoplasmosis
- Lyme Disease
- Syphilis
- Sarcoid
- TB
- Malignant hypertension if bilateral

Lab Tests

- Bartonella henselae antibody titer
  - B. henselae IgG - Positive >1:256 dilutions
    - A positive IgG (titer >1:128) suggests a current or previous infection. Increases in IgG titers in serial specimens would indicate an active infection.
  - B. henselae IgM - Negative

- Toxoplasmosis antibody titer
  - IgG – Negative
  - IgM – Negative

- ANA – Negative

- RPR – Negative

- ACE – 79 (slightly elevated)

Treatment

- Since CSD is typically self-limiting and benign, antibiotic treatment is optional in immunocompetent patients

- For immunocompromised patients or those patients where antibiotic therapy is desired the following antibiotics have been reported to be efficacious:
  - Azithromycin
  - Ciprofloxacin
  - Rifampin
  - Gentamicin, Trimethoprim Sulfaemethoxazole
  - Penicillins, cephalosporins, tetracyclines, erythromycin

- There is not a general consensus in the literature on the most efficacious antibiotic.
**Case Treatment**

- **Doxycycline 100 mg bid**
- **RTC 1 month**
  - VF on return
- **Patient returned in 1 month for follow-up**
  - ONH edema was greatly reduced
  - OS VF retained the enlarged blind spot
  - Doxycycline was continued for 4 more weeks

---

**Clinical Pearls**

- CSD the most common infectious etiology associated with neuroretinitis
- A thorough history including past animal exposure, presence of skin lesions and travel is paramount
- The condition is self-limiting and the prognosis for complete resolution with acuity returning to normal is very good
- Lab testing should include Bartonella henselae IgG and IgM titers
- When antibiotic therapy is employed, doxycycline 100mg bid for 6-8 weeks appears to be effective.

---

**Case Treatment**

- **On Final follow-up - 3 months from the patient’s initial presenting symptoms**
  - Patient completed antibiotic therapy
  - VA OD: 20/20
  - OS: 20/20
  - HVF OS: Enlarged blind spot
  - Neuroretinitis completely resolved
  - Duration of Doxycycline treatment was 8 weeks

---

**Any Questions??????**
Case 7
Help! My patient can't adduct!

Case History
- AA female, 2nd decade of life
- CC: double vision when looking to the left x a couple of weeks
- Systemic Hx: Numbness in the right leg x about 1 month
- Otherwise unremarkable

Motilities

Differential Diagnosis of Limited Adduction
- Internuclear Ophthalmoplegia
- Partial III CN palsy
  - involving the medial rectus only
  - extremely rare
  - convergence would be affected
- Myasthenia Gravis
  - variable with fatigue
  - Cogan's lid twitch
  - ptosis on sustained superior gaze
- Grave's

Forced Ductions Test

Negative Forced Ductions

Neurological Etiology
- Mechanical Etiology
Differential Diagnosis of Limited Adduction
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Fatiguability on Sustained Superior Gaze
- Fatiguability
- Myasthenia Gravis

Cogan’s Lid Twitch
- Overshoot of lid in a twitch
- Myasthenia Gravis

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Internuclear Ophthalmoplegia
- Disorder of conjugate lateral gaze
  - coupled, ie connecting both eyes

Internuclear Ophthalmoplegia
- Ipsilateral Eye: Impairment of adduction
- Contralateral Eye: Abducts with nystagmus

Internuclear Ophthalmoplegia
- Disorder of conjugate lateral gaze
  - coupled, ie connecting both eyes
Medial Longitudinal Fasciculus (MLF)
- fiber tracts (bundles of axons)
- composed of both ascending and descending fibers
- one on each side of the brainstem
- crossed
- main central connection b/w III, IV, VI

Conjugate Left Gaze

Internuclear Ophthalmoplegia

Internuclear Ophthalmoplegia
- Exotropia in primary gaze except for mild cases
- Convergence: Intact/better

Convergence
Symptoms of INO

- Diplopia
- Oscillopsia
- May have other symptoms of brainstem disease
  - Vertigo, Limb Numbness, Weakness

Causes of INO

- Stroke (38%) older patients
- MS (34%) younger patients
- Other causes (28%)
  - Tumors
  - Hemorrhage
  - Infection
  - Trauma

What next?

- Thinning NFL/GCC in contralateral eye
  - OCT NFL + GCC
    - No h/o of Optic Neuritis, no evidence of glaucoma
    - Multiple Sclerosis

Impression/Plan

- INO, high suspicion of MS
- Referred to neuro-ophthalmology
  - MRI brain w/ and w/o
  - Confirmed dx

80 YEAR OLD MAN

- Reports a sudden loss of vision OD
- Vision is count fingers at 2 feet OD and 20/25 OS
- APD OD grade 4
- Fundus photos OU
CRAO TREATMENT/WORK-UP/FOLLOW-UP?

- Anterior chamber paracentesis (less than 24 hours)
- STAT blood work
  - 2-10% of CRAOs are caused by thrombosis from Giant Cell Arteritis (GCA)
  - Sed-rate
  - C-reactive protein
    - Qualitative or quantitative?
  - CBC with differential
- Monitor for neovascularization, every 3-6 weeks

CRAO, BRAO, TIA (AMAUROSIS FUGAX)

- Acute Stroke Ready Hospital
  - Certification recognizes hospitals that meet standards to support better outcomes for stroke care as part of a stroke system of care
  - Developed in collaboration with the Joint Commission (JSC), eligibility standards include:
    - Dedicated stroke-focused program
    - Staffing by qualified medical professionals trained in stroke care
    - Relationship with local emergency management systems (EMSs) that encourage timely in-hospital assessment tools and communication with the hospital prior to bringing a patient with a stroke to the emergency department
    - Access to stroke expertise 24 hours a day, 7 days a week (in person or via telemedicine) and transfer agreements with facilities that provide primary or comprehensive stroke services.
    - 24/7 ability to perform rapid diagnostic imaging and laboratory testing to facilitate the administration of IV thrombolytics in eligible patients
    - Streamlined flow of patient information while protecting patient rights, security and privacy
    - Use of data to assess and continually improve quality of care for stroke patients
  - Warn hospital is suspicion for GCA
  - 20% of stroke or heart attack within 3 years
  - However of those who experienced CVA or MI
    - 80% were within 24-48 hours; those remaining
    - 50% occurred in 2 weeks
    - Majority within the next 90 days
  - Not PCP, not retinologist, just the Acute Stroke Ready Hospital!