

## CLINICAL GRAND ROUNDS: PERSPECTIVES FROM AN OPTOMETRIST AND AN INTERNIST

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## DISCLOSURE

Joseph Sowka, OD is/ has been a Consultant/ Speaker Bureau/ Advisory Board member for Novartis, Allergan, Glaukos, and B&L. Dr. Sowka has no direct financial interest in any of the diseases, products or instrumentation mentioned in this presentation. He is a co-owner of Optometric Education Consultants ([www.optometricedu.com](http://www.optometricedu.com))

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### WORLD'S BEST DISC HEMORRHAGE

- 33 YOWM
- Occipital HA x 4 mos
  - Visual aura with HA
- Worsens when standing after sitting
- Relieved by sleep
- Denies vision loss, nausea, diplopia, pain on eye movement, behavioral changes
- Recent age-appropriate physical normal

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### WORLD'S BEST DISC HEMORRHAGE

- 20/20 OD, OS with myopic correction
- Pupils, EOMs, conf fields normal OU
- Biomicroscopy normal OU
- IOP 12 mm Hg OU
- Nasally obliquely inserted nerves

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**Form: 07-15 (Revised) Test**

Patient Number: 0000000000    Date: 07-15-2008  
 Patient Target Center:    Strategy: 07-A-Fast    Age: 33  
 Patient Lenses: 01-10-00    Rx: -1.00 DS    DC: X  
 Patient HED Error: 0.0    Test Method: 02-15  
 Test Method Error: 0.0  
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 Patient: 0000

\*\*\* Low Test Reliability \*\*\*  
 MD: -1.00 dB  
 MS: -1.00 dB  
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Now what?

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## WORLD'S BEST DISC HEMORRHAGE

- Co-manage
- MRI w and w/o contrast of brain and orbits
- Complete blood work blood work up including FTA-ABS/RPR ; Lyme titer; CBC w/differential
- Rule out mass lesion, infections, collagen vascular and autoimmune etiology.

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## WORLD'S BEST DISC HEMORRHAGE

- Serology normal
- MRI: Large intracranial mass was identified in fronto/parietal region more toward right side
- Outcome?

What are some of the 'problem headaches'?

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**ARE THERE ANY**  
  
**QUESTIONS?**

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## 74 YOWM

- Presents with 'worst headache of his life'
  - Sees: PA, ER physician; cardiologist; NP; 3 ODs
    - *3 week period*
  - Histories: Eye ache; jaw pain, scalp pain, facial pain, somnolence; malaise; jaw claudication
  - Diagnoses: TMJ; Lyme disease
  - "vasculitis such as temporal arteritis highly unlikely", "Not GCA"
    - *However, ESR and CRP ordered and elevated- never reviewed*
  - End result?



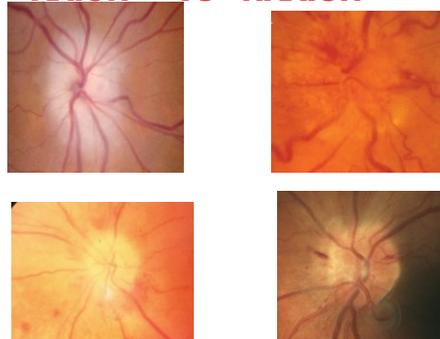
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## AAION

- Pallid optic nerve swelling with flame hemorrhages, arteriole attenuation and NFL infarcts
- Optic nerve dysfunction
- Visual field defects
- Pallid swollen nerve
- Giant cell arteritis/ PMR risk factors
- Typically 70s, uncommon under 60
- High risk bilateral involvement

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## AAION VS NAAION



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## GIANT CELL ARTERITIS/ TEMPORAL ARTERITIS

- A systemic disorder affecting primarily the elderly and characterized by granulomatous inflammation of large- and medium-sized arteries
- Most patients are over age 60 and the majority are over age 70
- Men and women are equally affected
- Inflammatory cells produce cytotoxic enzymes and reactive oxygen species that destroy vascular tissue and obstruct vessel lumen

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## GIANT CELL ARTERITIS/ TEMPORAL ARTERITIS

- Headache (present in over 90%)
- Scalp tenderness
- Jaw claudication (almost diagnostic)
- Ear pain
- Arthralgias
- Temple pain and/or tenderness
- Malaise
- Intermittent fevers
- • \*MUST ASK PATIENT DIRECTLY

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## OCULAR MANIFESTATIONS

- Loss of vision
  - Transient (eg, amaurosis fugax; TIA)
  - Persistent (AAION; CRAO)
- Double vision
- Eye pain
- Red eye

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## DIAGNOSIS

- Careful history: Must ask about nonvisual symptoms
- Examination
- Laboratory studies
  - Erythrocyte sedimentation rate
    - Lowered by statins and NSAIDS
  - C-reactive protein
    - Not affected by statins and NSAIDS
  - Elevated platelet count

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## TREATMENT FOR AAION / GCA

When vision loss ensues, patient recommended receive 1-2 mg IV methylprednisolone for several days, followed by high dose (80-120 mg) daily oral prednisone

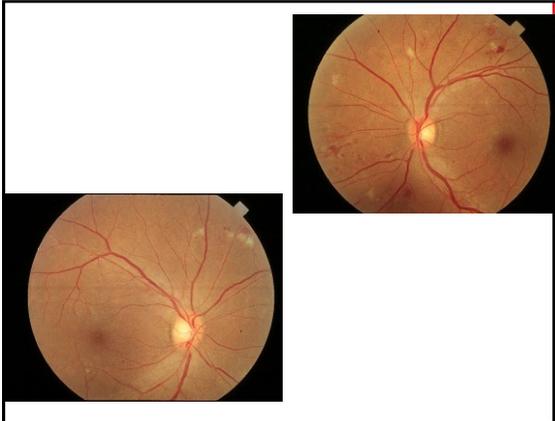
- Issues with steroid treatment long term?
- Actemra?

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## THE MEDICINE MAKES ME SICK

- 52 YOWF
- Medical history: hypertension x 10 years; NIDDM x 2 yrs
  - Medicines unknown
  - Poorly controlled
  - Pt non-compliant
- BP: 157/109 RAS

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## HYPERTENSIVE RETINOPATHY

- **Arteriosclerotic vessel changes**
  - Some classification schemes include vessel changes in hypertensive retinopathy and others don't
- **Elschnig's spots – subtle choroidal infarcts**
- **CWS**
- **Flame shaped hemorrhages**
- **Macular edema (rare)**
- **Macular star/ ring of exudates**
- **Disc edema**

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## NOW A TWIST

- **47 YO BM**
- **Obese**
  - 400 lbs (and that's being kind)
- **Headaches x 3 months**
- **Vision reduction x 2 months**
  - 20/50 OU
- **BP: 212/155 RAS**

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So, What do you think  
now?

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**Hypertensive  
EMERGENCIES  
&  
Hypertensive URGENCIES**



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# ARE THERE ANY QUESTIONS?



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## CASE: 20 YEAR OLD WHITE FEMALE

- **CC:** Intermittent itching and irritation OU x 2 months
  - Worse after showers
  - Eyelids red and swollen all the time
  - Lid scrubs not helpful
- **Medical Hx:** non-contributory
- **BVA** 20/20 OD, OS

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## 20 YEAR OLD WHITE FEMALE

- Significant erythema OU
- Thick crusting about lashes
- IOP normal OU
- Fundus unremarkable



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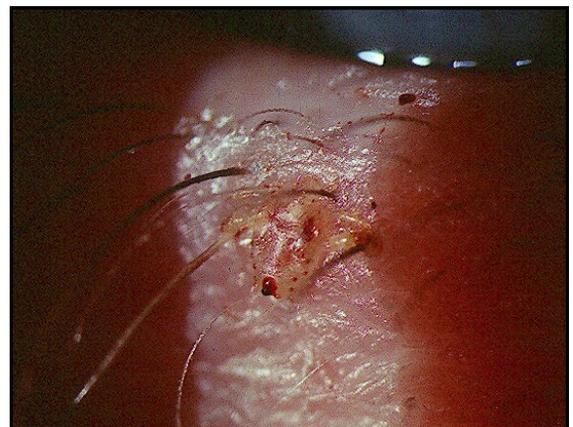
## 20 YEAR OLD WHITE FEMALE

- Can I get some more detail?

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## CRAB LOUSE INFECTION

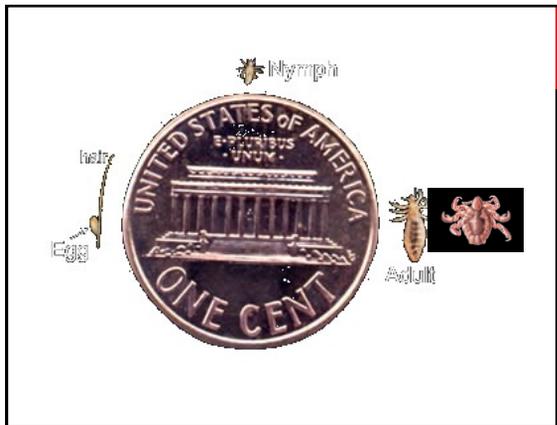
- Pediculosis refers to infestation by *Pediculus humanus corporis* (body) or *capitus* (head).
- Phthiriasis refers to eyelid infestation by *Phthirus pubis* (pubic louse).
  - Eyelid infestation is almost always *Phthirus pubis*.
- *Phthirus* organisms are 2 mm long with a broad-shaped, crab-like body
- Thick, clawed legs make it less mobile than *Pediculus* species



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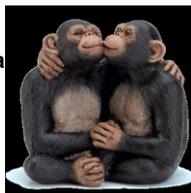
## CRAB LOUSE INFECTION

- Infest areas where the adjacent hairs are within its grasp (eyelashes, beard, axillary region, pubic region).
  - Rarely do they infest the scalp.
- Ocular signs and symptoms:
  - visible organisms
  - reddish brown deposits (louse feces)
  - 2° blepharitis with preauricular adenopathy
  - follicular conjunctivitis
  - bilateral ocular itching and irritation

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## CRAB LOUSE INFECTION: TREATMENT

- *Pediculus* organisms possess good mobility and can be passed from person to person by either close contact with an infested individual or by contact with contaminated bedding.
- *Phthiriasis* are slow moving organisms that cannot typically be passed unless cilia is brought into close proximity with infested cilia.



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## CRAB LOUSE INFECTION: TREATMENT

- Forceps removal of all visible organisms and nits
  - Removed debris should be placed into an alcohol wipe and discarded
- Pediculocidal medicated shampoo
  - Lidane 1% (gamma benzene hexachloride)
  - Kwell®, Nix® or Rid®, which is a safe, effective, nonprescription pediculocide
    - NOT for ocular use



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## CRAB LOUSE INFECTION: TREATMENT

- Topical therapy may include:
  - smothering lice & nits with petroleum jelly (or other bland ointment) X tid
  - 1% yellow mercuric oxide or 3% ammoniated mercuric oxide X bid
  - cholinesterase inhibitors (e.g. physostigmine)
- Typically, nits survive a single application of these agents.

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## CRAB LOUSE INFECTION: TREATMENT

- Daily follow for 7 - 10 days
  - nits hatch q7-10 days
- Thoroughly wash all clothing and linens that may have been exposed.
- Patients should refrain from “interpersonal contact” until the disease is 100% resolved.
- Educate exposed partners to report for examination and evaluation.

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## CONSIDERATIONS IN STD TESTING AND REPORTING

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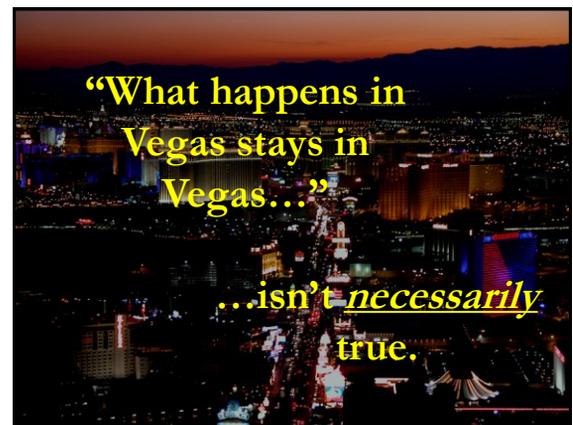
## “I’M NOT GOING BACK IN THERE!”

- OD-4 Student examines older male patient
- “I’m not going back in there. There are worms!”
- “I think that I am going to pass out”
- Nothing really to set up
- Social History: Recently returned from trip to Las Vegas

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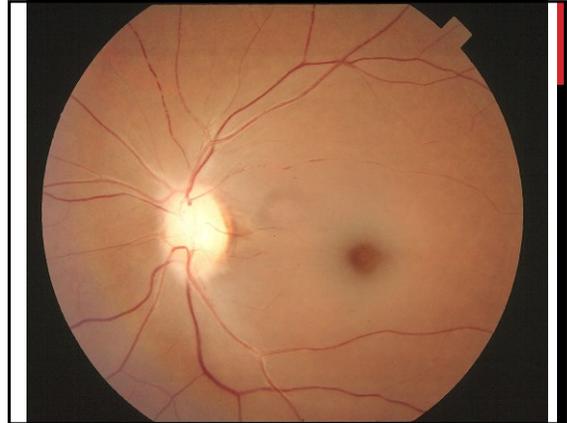


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## CASE: I (DON'T) FEEL GOOD!

- 66 year old Black male
- CC: sudden, painless blurring OS x 2 days
- No previous eye or medical care
- BVA OD 20/30, OS HM
- Pupils: ERRL (+) RAPD OS
- Good appetite, poor diet

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## CRAO: TREATMENT ?

- Paracentesis
- Carbogen
- Digital massage
- Hyperventilation
- Urokinase/streptokinase
- 1-24 hr window of opportunity
- Does anything work?



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## CRAO: SYSTEMIC CONSIDERATIONS

### Atherosclerosis

Carotid artery disease

### GCA

Antiphospholipids ABS

Infectious endocarditis

Vasospastic disease

Cardiac arrhythmia

Clotting factor abnormalities

### Hypertension

Diabetes

Cardiac valve disease

Cardiovascular disease

Hyperlipidemia

Disc drusen

Mural thrombosis

Hyperviscosity syndromes

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## Guidelines

- Any patient with suspected TIA or those with acute retinal ischemia should be evaluated urgently in order to identify those at high risk of immediate cerebral infarction and cardiac ischemia

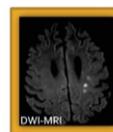
Guidelines for the prevention of stroke in patients with stroke or transient ischemic attack: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2013; 44: 527-526

Adapted from Drs. Nancy Newman and Biousse; 2015

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## All Patients with Acute Retinal Arterial Ischemia

- MUST have immediate brain imaging
  - Brain MRI with DWI >>> Head CT
- Including patients with transient visual loss (presumed of vascular origin)



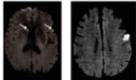
Presence of cerebral ischemia portends higher risk of stroke

Adapted from Drs. Nancy Newman and Biousse; 2015

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### Concurrent Acute Brain Infarcts in Patients with Monocular Visual Loss

- ¼ with acute retinal ischemia had acute brain infarction (anywhere) on brain DWI-MRI
  - Infarctions often small, multiple, ipsilateral to retinal ischemia, asymptomatic
- DWI-MRI abnormal in:
  - 33% with CRAO/BRAO vs 18% with TVL
  - 28% with embolic vs 8% non-embolic retinal ischemia



Adapted from Drs. Nancy Newman and Biousse; 2015

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### Tell the patient:

- "Go to the Emergency Department"
- "Tell them you had a retinal stroke"
- Do not send these patients to their PCP, cardiologist, neurologist, neuro-ophthalmologist
- Do not try to obtain the workup yourself



Adapted from Drs. Nancy Newman and Biousse; 2015

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### THE CASE OF THE COLORED FLASHING LIGHTS

- 45 YOHF presented with colored "map-like" phosphenes and small black flashing spots OD x two weeks
- Noted that she had to "look between the lights" to see out of her right eye.
  - 20/20 OD, OS
- Medical history was unremarkable except for treated migraines
- Lost 1 pregnancy

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So,  
What's your diagnosis?



Management...?



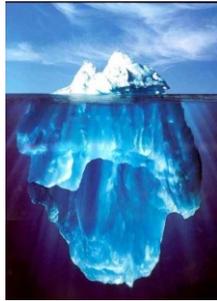
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### CASE CONTINUED

- She returned four days later complaining of decreased vision in the right eye, which had reduced to counting fingers at ten feet.
  - Macular edema, more extensive hemorrhaging, cotton wool spots, disc edema and dilated vessels
- Underwent IV injections and showed improved vision of 20/70 OD during follow up examinations.
  - Released by retinal specialist
  - No medical evaluation

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## Now What?



Are there any tests that you would like to order?

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## CRVO: SYSTEMIC CONSIDERATIONS

- |                             |                          |
|-----------------------------|--------------------------|
| ■ <i>Hypertension</i>       | ■ <i>Diabetes</i>        |
| ■ <i>Hyperviscosity</i>     | ■ Syphilis               |
| ■ CV disease                | ■ Cardiovascular disease |
| ■ Sickle                    | ■ Leukemia               |
| ■ Polycythemia              | ■ Carotid artery disease |
| ■ Hyperlipidemia            | ■ Sarcoid                |
| ■ <i>Autoimmune factors</i> | ■ Clotting abnormalities |
| ■ <i>Homocysteine</i>       |                          |

Coagulopathy evaluation vs. Atherosclerotic evaluation?

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## TREATMENT & MANAGEMENT

- Referred blood work through PCP
  - DM, HTN, hypercoag, ANA, antiphospholipid antibodies, anticardiolipin, PT, PTT, ESR, CBC with diff
- Elevated erythrocyte sedimentation rate
- Mildly elevated cholesterol level.
- Elevated anti-cardiolipin IgM antibodies
  - Suggestive of antiphospholipid antibody syndrome
  - She was recommended for long term anti-coagulant therapy to prevent future thrombotic events, but patient never followed through.

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## CASE CONTINUED

- Seven months later the patient returned with the same signs and symptoms in her right eye.
- At this time, the vision was markedly more decreased with more evidence of ischemia
  - CF @ 6'
- She was referred to a hematologist
- Now on anti-coagulation therapy

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## PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME

- Thrombotic disorder
- Secondary antiphospholipid syndrome
  - Associated several autoimmune diseases but most often systemic lupus erythematosus
- Primary antiphospholipid syndrome is not associated with further systemic disease
- Recurrent vascular thrombosis, pregnancy loss and positive anticardiolipin or lupus anticoagulant are all characteristics of this disorder

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## PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME

- The clinical criteria
  - One or more vascular thrombotic episodes of venous, arterial or small vessel thrombosis in any organ or tissue or spontaneous abortion.
- Laboratory testing must show persistently elevated **anticardiolipin** antibodies, IgG and/or IgM or **lupus anticoagulant** (inhibits the conversion of prothrombin to thrombin) at least six weeks apart

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## PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME

- Phospholipids are identified by the body as "foreign."
  - The antiphospholipid antibodies are produced against the "foreign" antigen.
- These antibodies have an affinity for cell membranes in platelets, vessel endothelial cells and clotting factors.

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## PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME

- The antibodies appear to react with the cell membranes causing irritation or stimulation, thus disrupting the coagulation cascade
- This disruption leads to abnormal blood clotting and inhibits normal phospholipid binding.

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## PRIMARY ANTIPHOSPHOLIPID ANTIBODY SYNDROME

- This abnormal or inhibition of proper phospholipid binding leads to a hypercoagulable state thus causing thrombosis.
- Propensity of clot formation is within the venous and arterial portions of the vascular tree, especially targeting the retinal vessels and placenta

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## JUST A ROUTINE EXAM...

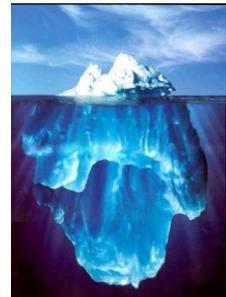
- 72 YOWM
- No visual or ocular complaints
- HTN x 20 years
- Lifetime smoker
- Reasonably compliant with meds

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## Now What?



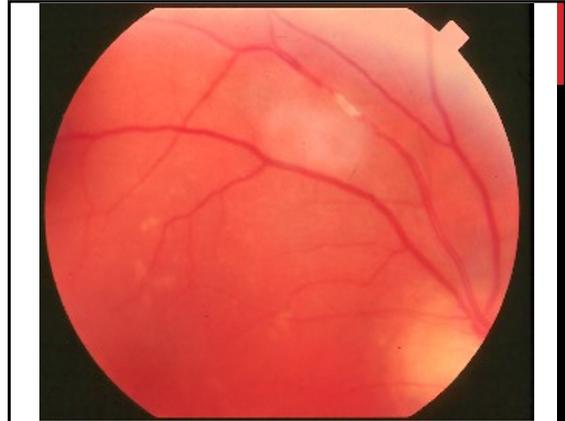
Are there any tests that you would like to order?

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## ASYMPTOMATIC RETINAL EMBOLI

- Cholesterol emboli are the most commonly encountered (80% of emboli) while fibrin-platelet emboli represent 14% of emboli and calcific emboli account for just 6% of visible retinal emboli

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## ASYMPTOMATIC RETINAL EMBOLI

- The proper approach to managing asymptomatic retinal emboli is truly not known.
- The most common associated systemic disease is hypertension, cardiovascular disease and stroke.
- In many studies, the prevailing risk factor for retinal emboli is smoking.

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## ASYMPTOMATIC RETINAL EMBOLI

- Patients should be evaluated by an internist for hypertension, coronary artery disease, diabetes, and carotid artery disease
- A complete physical with stress echocardiogram, fasting glucose, lipid levels, blood chemistry with cardiac enzymes, magnetic resonance angiography, transthoracic and transesophageal echocardiography may be indicated, especially for patients with symptomatic retinal emboli.

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## ASYMPTOMATIC RETINAL EMBOLI

- No consensus on the need for carotid ultrasonography in patients with asymptomatic retinal emboli
  - Majority of these patients do not have high grade carotid stenosis
- There is no clear indication for carotid endarterectomy in patients with asymptomatic retinal emboli, even with concurrent high grade carotid stenosis
  - There does seem to be a benefit to carotid endarterectomy in patients with symptomatic retinal emboli and high grade carotid stenosis

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