AOA’s definition of Optometry approved Sept 2012

Doctors of optometry (ODs) are the independent primary health care professionals for the eye. Optometrists examine, diagnose, treat, and manage diseases, injuries, and disorders of the visual system, the eye, and associated structures as well as identify related systemic conditions affecting the eye.

PREVENTION
WELLNESS
TREATING THE WHOLE PATIENT
MEDICAL OPTOMETRY

…..where do we fit in?

Not just this…
But also this…
• 52 Caucasian male
• Never had an eye exam
• No regular health care
• Vision goes “out” when he turns his head

• ≥70% blockage before ocular manifestations

• 5 year mortality rate – 40%
  • MI is mc
  • 4/5 strokes are causes by atherosclerosis at carotid bifurcation

Vascular Supply Systems to Brain

1. Internal Carotid system
   • Supplies anterior and lateral portions of brain
   • Unilateral visual disturbances

2. Vertebrobasilar system
   • Provides posterior brain
   • Bilateral visual symptoms

• X 2 hours ago
• 62 year old white male
• Heavy smoker, hx hypercholesterolemia, +HTN

“Vision went out, but now it’s back”

Ocular signs of carotid artery disease

1. Amaurosis Fugax
2. CRAO
3. Hollenhorst Plaque
4. Ocular Hypoperfusion

Only 10% of emboli from ICA end up in OA. !
Intra-arteriolar emboli

- Increased risk of stroke, mortality, co-morbidity
  - 25% have carotid stenosis >40%
- Symptoms?
  - Often transient – plaques are pliable
  - Correlated with degree of occlusion?
  - Predictive of future events?
- Doppler
- EKG/Angiography

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81 Caucasian female

- Wants new glasses before a trip to Paris
- PMHx:
  - Atrial fibrillation
  - Recent falls – due to TIA
  - VA 20/30 due to cataracts
  - DFE – retinal heme and intra-arteriolar plaque

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Atrial Fibrillation

- Most common cardiac arrhythmia
- Increased risk of mortality by 40-90%
  - TIA, stroke (x5) and MI
- Screen for with RAO patients
  - 1 risk of stroke
  - 1 need for anticoagulant

"Blood work-up"....tests driven by differentials

- CBC with differential
- Chem 7
- Lipid Profile
- ESR
- C-Reactive Protein
Chem 7 / Basic Metabolic Panel

1. Creatinine
2. Blood urea nitrogen (BUN)
3. Glucose
4. Carbon dioxide
5. Chloride
6. Sodium
7. Potassium
8. (Sometimes Calcium)

• Screens for
  • Kidney disease
  • Liver Disease
  • Diabetes and other blood sugar disorders

End Stage Renal Disease

76 Caucasian male

- Hx severe anemia secondary to ESRD
- 30% carotid occlusion

Bilateral blot hemes, all 4 quadrants
- disc edema, -tortuosity
- artery attenuation

Test Ordered | Results
--- | ---
FRS | 107 mg/dl
AIC | 5.6
CBC | Hb: 10 g/dl
McV normal
Hct: 32%
Platelets: 100,000 cmm

Anemia
Thrombocytopenia
Normocytic
Normocytic
All other values normal

Carotid Doppler | 30% Carotid Occlusion both sides
Also Consider: Common Coagulopathies

<table>
<thead>
<tr>
<th>Condition</th>
<th>Name of Lab Test</th>
</tr>
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<tbody>
<tr>
<td>Antiphospholipid Antibody /</td>
<td>Antiphospholipid Antibody Panel</td>
</tr>
<tr>
<td>Antiphospholipid Syndrome</td>
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<tr>
<td>Protein C and/or S deficiency</td>
<td>Protein C and S Activity with Reflex to Protein C and/or S Antigen</td>
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<tr>
<td>Antithrombin III deficiency</td>
<td>Antithrombin III Panel</td>
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<tr>
<td>Elevation of platelet factor 4</td>
<td>Platelet Factor 4</td>
</tr>
<tr>
<td>Factor V Leiden</td>
<td>Factor (V) Leiden Mutation Analysis</td>
</tr>
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</table>

Factor V Leiden??? What’s that?!!

• Factor V – clotting protein
  • genetic mutation: ↑clotting in veins
  • Mc prothrombotic gene mutation in caucasians – 5% population
  • Caucasians of European descent
• Often undiagnosed, however....
  • deep vein thrombosis
  • pulmonary embolisms
  • CRVO
  • 11% of ocular vasc occlusions assoc with FVL (Schockman 2015; Fegan 2002)

57 Caucasian female with “borderline” HTN and Factor V Leiden

85 WM, HTN, Smoker

• Black circle over vision x 2 days
• FB vision
• Pupils normal

Red-Free
39 WM with progressive, relapsing MS

- Resistant to tx due to SEs
- 20/60 OD, +APD
- RNFL and GCC thinning both eyes
Optic Neuritis Treatment Trial, circa 1992

- IV steroids over oral
  - 1000mg IV methylpred x 3 days
  - Speeds recovery of vision; reduces recurrences
- Alternative? Considerations?
  - Bioequivalent Oral Steroid—
    - oral prednisone (1250-mg over 3 days) vs. high dose IV over 3 days
    - No significant differences in VA, Contrast Sensitivity, VEP at 1 month or 6 months
  - Steroid “Smoothie”

1 month later – after steroid infusion

- 20/40
- (-)APD
- VF improved

- Head injury 3 months ago
  - Imaging in ER all negative
- Vertical diplopia
  - Worse in down gaze
  - Right head tilt
SO Palsy

- **Etiology**
  - Trauma
  - Decompensated congenital – slow onset
  - Least likely of EOM palsies to have underlying etiology, BUT...
    - Microvascular disease
    - Brain abnormality

- **Treatment**
  - Prism, surgery, botox

EOM palsies: Do not assume......

1. Vasculopathic
   - 16.5% thought to be ischemic had another cause (neoplasm, MS, GCA)
   
2. True isolation

And don’t forget about...

- GCA
- Carotid Artery Dissection
- Aneurysm
- ...
76 year old male with double vision

- Left-sided headache
- Denies Trauma

If you want to find something wrong, do an MRI......

- Brain aneurysm: incidental finding?
- Incidence
- Size, anatomical location
- Likelihood of relation to problem?

Concussion – what to look for, our role?

- Eye exam findings
- How we can help
Giant cell arteritis

- Clinical presentations
  - Arteritic ION
  - CN palsies
  - CRAO
- Labs - if suspected, send to ER and order all of the following:
  - ESR – 13% are normal
  - C-reactive protein – 1% are normal
  - Platelet count, CBC with differential (anemia often present)
- Biopsy indicated for anyone who is suspected, regardless of ESR

Patient Labs

<table>
<thead>
<tr>
<th>Test</th>
<th>Result</th>
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<tbody>
<tr>
<td>CBC</td>
<td>all normal</td>
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<tr>
<td>ESR</td>
<td>15</td>
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<tr>
<td>CRP</td>
<td>&lt; 1</td>
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</table>

- 68 Caucasian male
- Blurry vision
- VA 20/200
- +APD

Myasthenia suspicion... ?

- Tensilon Test – low sensitivity
- Icepack Test
- Sustained upgaze x 2 min

- Bloodwork – high false negatives
  - ACH, thymic panel, ANA, DM
- Imaging – 70% have thymic hyperplasia (mediastinal imaging)

Chronic double vision, imbalance

- Loves BI prism
- Glasses have never been quite right
Carotid Artery Dissection → Painful Horner’s

• 48 year old male presents with a big pupil in the left eye.
  • ROS: right-sided neck pain, headache

• Exam
  • Right eye – miosis, ptosis
  • Dilates with 0.5% apraclonidine

• Horner’s – 3rd order neuron defect along sympathetic pathway

Carotid Artery Dissection

• Traumatic or spontaneous
• Cause of 2.5% of strokes
  • 10-25% of ischemic events in patients <45
  Rao, J Vasc Surg 2011

Carotid Artery Dissection

• Presentation
  • Headache – up to 69% most common presenting symptom
  • Unilateral neck pain – up to 49%
  • Ipsilateral Horner’s – up to 60%
  • Visual manifestations associated with artery dissections
    • Photopsia
    • VF defect

• Mgmnt
  • Immediate Imaging: CT/CTA, MRI/A, T1W with contrast and fat suppression, Doppler
  • Anti-platelets, anti-coagulant tx

Big pupil problem → think 3rd N

• Parasympathetic pupillary fibers are located superficially along 3rd CN
  • Likely to be affected early with compressive lesion

• Anatomically at risk for aneurysm
  • 85% are within anterior Circle of Willis
  • 30-35% are adjacent to 3rd CN
  • PCA – most common

• Imaging Considerations
3rd Nerve – a nice overview


1400 personally examined patients – 37 years

- Presentation
  - bilateral in 11%
  - complete in 33%
  - isolated in 56%
- Etiology
  - trauma (26%)  
  - diabetes (11%) 
  - aneurysm (10%) 
  - surgery (9%) 
  - infection (5%)

- Of 234 patients with diabetes
  - 23 due to microvascular ischemia
  - 85% had pupillary involvement—often bilateral
  - 5 had aneurysms
  - Only 2% of aneurysms spared the pupil.

- Painful onset 
  - 96% of aneurysms 
  - 88% of diabetic cases.

- Of 234 patients with diabetes
  - 2/3 due to microvascular ischemia

- 53% had pupillary involvement—often bilateral
- 5 had aneurysms
- Only 2% of aneurysms spared the pupil.

- Painful onset
  - 96% of aneurysms
  - 88% of diabetic cases.


73 WF, unexplained APD OS

- 20/20
- Recent history of vertigo
- MRI revealed small, insignicant meningioma
44 WM with bilateral ptosis

- POH and PMH: unremarkable when questioned
- FOH:
  - Ptosis
  - Cataracts: Father and sister
  - “maybe” macular degeneration
- Exam:
  - Colorful nuclear opacities
  - Macular stippling OS

Ptosis -- DDx

- 3rd Nerve Palsy
- Horner’s Syndrome
- Congenital ptosis
- Levator Dehiscence
- Myasthenia Gravis
- Less commonly
  - Chronic progressive external ophthalmoplegia (CPEO)
  - Kearns Sayre syndrome
  - Ocularpharyngeal muscular dystrophy
  - Myotonic Dystrophy

Myotonic Dystrophy

- AD w/variable penetrance
- 1 in 8000 (presenting age 20-30)
- Myotonia:
  - ↑ muscle contraction with slow relaxation
  - Distal muscles of limbs, face, neck
- Multiple systems Endo, Resp, C/V
- ↓ intelligence, MH
- Later involvement of larynx, vocal cords, pharynx
**Back to case history...**

- **Family History:**
  - Sister had “muscular dystrophy” but died at age 45 before definitive dx
  - Father and both sisters also had droopy eyelids

- **Patient history:**
  - Admits having difficulty writing, gripping and releasing his grip after handshakes

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**Ocular manifestations**

- Ptosis (80%)
- Ocular motility disturbances
- Orbicularis weakness
- Hypotony (as low as 4mmHg)
- “Christmas Tree Cataracts”
- Peripheral retinal changes—up to 50%
- Macular involvement—20%
  - Granular pigment changes with stellate pattern

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**Myotonia - Dx and Mgmt**

- **Dx**
  - Family history, clinical presentation
  - Creatine kinase (CK) levels
  - Electromyography (EMG)
  - Abnormal ERG (↓dark adaptation)
  - Muscle biopsy
  - DNA testing

- **Treatment is palliative (Heat, cold avoidance, quinine)**
- Rarely, anti-myotonic drugs are used
- Genetic counseling