



Optometric  
Education  
Consultants



## Non-Stop Neuro-Op

Joseph Sowka, OD, FAAO, Diplomate  
Greg Caldwell, OD, FAAO  
Joseph Pizzimenti, OD, FAAO

1



## 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))



The ideas, concepts, conclusions and perspectives presented herein reflect the opinions of the speaker; he has not been paid, coerced, extorted or otherwise influenced by any third party individual or entity to present information that conflicts with his professional viewpoints.

2

## Disclosures- Greg Caldwell, OD, FAAO

- The content of this activity was prepared independently by me - Dr. Caldwell
- Lectured for: Alcon, Allergan, Aerie, BioTissue, Kala, Maculogix, Optovue
- Advisory Board: Allergan, Sun, Alcon, Maculogix, Dompe
- Envolve: PA Medical Director, Credential Committee
- Healthcare Registries – Chairman of Advisory Council for the Diabetes Registry
- I have no direct financial or proprietary interest in any companies, products or services mentioned in this presentation
- The content and format of this course is presented without commercial bias and does not claim superiority of any commercial product or service.
- Optometric Education Consultants - Scottsdale, Minneapolis, Florida (Ponte Vedra Beach), Mackinac Island, MI, Nashville, and Quebec City - Owner



OptometricEdu.com/webinars

3



## DISCLOSURE

- *Joseph Pizzimenti, OD* is a Consultant for Zeiss, Notal Vision, Maculogix, and ZeaVision. He has no direct financial interest in any of the diseases, products or instrumentation mentioned in this presentation.

The ideas, concepts, conclusions and perspectives presented herein reflect the opinions of the speaker; he has not been paid, coerced, extorted or otherwise influenced by any third party individual or entity to present information that conflicts with his professional viewpoints.

4

## Case

- A 27-year-old woman presents urgently complaining of painful vision loss in her right eye.
- She has no known medical history
- She has an edematous optic nerve with hemorrhaging, an afferent pupil defect, superior arcuate scotoma, pain when she moves her eye, and 20/70 visual acuity. Fellow eye is normal



OptometricEdu.com/webinars

5

## Differential Diagnosis

- Demyelinating optic neuritis
- Non-arteritic anterior ischemic optic neuropathy
- Arteritic ischemic optic neuropathy
- Infectious optic neuropathy
- Hereditary optic neuropathy
- Infiltrative optic neuropathy
- Perineuritis
- Papilledema

OptometricEdu.com/webinars

6

## Case

- Infectious optic neuropathy needs to be strongly investigated in a case like this. Neuroretinitis, often associated with cat-scratch disease, will present with a macular star of exudates, but this finding may be missing early in the disease. However, optical coherence tomography (OCT) will show a serous macular detachment early in the disease course.
- In this case, contrast-enhanced MRI of the orbits/chiasm and brain should be ordered to rule out demyelination and neural sheath swelling in perineuritis.
  - Contrast enhanced MRI orbits and chiasm with fat suppression
  - Contrast enhanced brain MRI looking for white matter lesions
- The patient should also be tested for numerous infectious agents including Bartonella, syphilis, Lyme, tuberculosis, herpes, Epstein-Barr, and rickettsioses, to name a few.
  - This can best be done in concert with the patient's primary care physician or an infectious disease specialist.

OptometricEdu.com/webinars

7

## Diagnosis: Infectious Optic Neuropathy

- MRI findings showed optic nerve enhancement possibly consistent with infectious, autoimmune, or granulomatous disease with no evidence of demyelination.
  - "Basically says there is something wrong with the optic nerve"
- Serological testing subsequently revealed very high titers of Epstein Barr Nuclear Antibody IgG and Epstein Barr Capsid Antibody IgG.
- The pathogenesis of infectious optic neuropathies may involve direct involvement of the optic nerve by a pathogen and /or indirect involvement with inflammatory, degenerative, or vascular mechanisms.

OptometricEdu.com/webinars

8

## Neuroretinitis

- Mild RAPD compared to vision loss
  - Vision loss more retinal than optic nerve
- Serous macular RD
  - OCT shows subretinal fluid between disc and macula in cases with disc edema only
- Macular star late finding

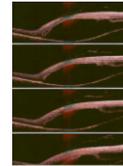


OptometricEdu.com/webinars

10

## 62 YOF

- 'Strep throat'
- CF @ 8' OD, 20/25 OS – antibiotics x 1 day
- RAPD OD
- Black spot and blurry vision 3 days



OptometricEdu.com/webinars

11

## Neuroretinitis/ Infectious Optic Neuropathy

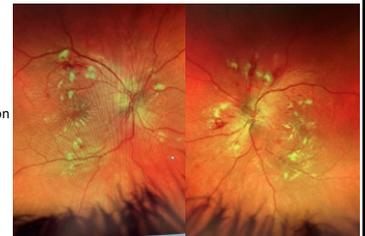
- Many potential etiologies
  - Toxoplasmosis, toxocariasis, measles, syphilis, Lyme disease, herpes simplex and zoster, E-B-V mumps, tuberculosis, malignant hypertension, ischemic optic neuropathy, and leptospirosis, *Bartonella* (most common). Fleas are vectors, thus no need for actual scratch.
- Prognosis for visual recovery excellent, especially if the cause is cat scratch disease. Other causes need treatment
  - Most patients will have a return to normal or near normal vision without
  - Antimicrobial therapy may be used to hasten recovery.
    - Rifampin, ciprofloxacin, doxycycline, sulfamethoxazole; doxycycline 100 mg PO BID for one month. Intravitreal injection of Avastin™ (Bevacizumab) has been shown to improve both visual acuity as well as decrease macular edema. However, the overall good prognosis of neuroretinitis may not justify this treatment, especially since this information comes from case reports and not controlled clinical trials.
  - While antibiotics are frequently used for cat scratch disease neuroretinitis, there are no controlled clinical trials that indicate a better clinical outcome from this therapy. The same can be said for the use of oral steroids and intravitreal anti-angiogenic medications.

OptometricEdu.com/webinars

12

## Case

- 22-year-old female
- First eye exam- blurred vision
- 20/40 OD; 20/70 OS
- No medical history
- Thin build



OptometricEdu.com/webinars

13

## Diagnosis and Plan?

- Bilateral CRVO- coagulopathy workup
- Idiopathic intracranial hypertension- prescribe diamox and weight loss
- Brain tumor- immediate MRI
- Malignant hypertension- BP assessment and ER referral
- Infectious optic neuropathy because that was what the last case was

OptometricEdu.com/webinars

14

## Case 2

- BP 180/144
- Likely diagnosis- malignant hypertension
  - But it *could* be other things



OptometricEdu.com/webinars

15

## Hypertensive Crises



# Hypertensive EMERGENCIES & Hypertensive URGENCIES

OptometricEdu.com/webinars

16

## Hypertensive EMERGENCY

- Severe Hypertension + *End-Organ Damage*
  - Examples of end-organ damage... *hypertensive encephalopathy, intracerebral hemorrhage, acute myocardial infarction, left ventricular failure with pulmonary edema, acute coronary syndrome, dissecting aortic aneurysm, or eclampsia*
- Hypertensive EMERGENCIES require immediate BP reduction (not necessarily to normal ranges) to prevent or limit organ damage.
  - Patients with hypertensive emergencies are often admitted through the ER for aggressive treatment.

OptometricEdu.com/webinars

17

## Hypertensive URGENCY

- Severe Hypertension + NO End-Organ Damage
  - Typically identified during **routine** evaluation
  - Usually represents chronic hypertension, nonadherence to drug therapy or inadequate treatment by the PCP.



OptometricEdu.com/webinars

18

## Hypertensive URGENCY

- DOES NOT warrant aggressive BP reduction, as rapid reduction of BP may be associated with significant MORBIDITY:
  - Causes a rightward shift in the pressure/flow autoregulatory curve in critical arterial beds (cerebral, coronary, renal).
  - Can result in marked reduction in perfusion, causing ischemia and infarction; BP must be reduced in a SLOW and CONTROLLED fashion.
  - Patients with hypertensive urgencies are usually treated with oral medications and followed over several days to weeks to evaluate their response to therapy.

OptometricEdu.com/webinars

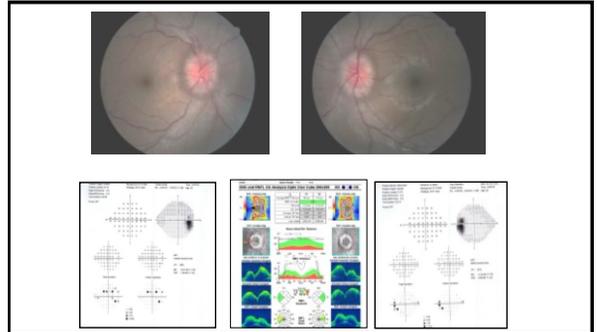
19

## Case

- 33-year-old-female
- Horizontal diplopia- CN 6 palsy OS
- Headache
- TVOs 20/day
- Denies OCP, tetracyclines, vitamin A
- Lost 10 lbs- headaches improved
- 118/72
- 5'5"; 160lbs; BMI 26.62

OptometricEdu.com/webinars

20



21

## What is the most likely diagnosis?

- Pseudotumor cerebri
- Idiopathic intracranial hypertension (IIH)
- Malignant hypertension
- Intracranial mass lesion

OptometricEdu.com/webinars

22

## PTC vs. IIH

- Pseudotumor Cerebri (PTC)
  - Increased intracranial pressure in the absence of an intracranial mass lesion
  - Many causative agents have been identified
    - Tetracyclines, vitamin A, contraceptives, venous sinus thrombosis (thus need for MRV)
- Idiopathic Intracranial Hypertension (IIH)
  - Increased intracranial pressure without an identifiable cause
  - Young, obese females are at risk
- Primary PTC
  - IIH
- Poor CFS drainage

OptometricEdu.com/webinars

23

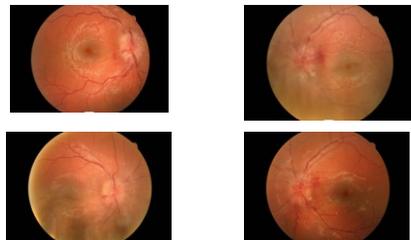
## Case

- 16-year-old-male
- Vision has been fluctuating for 6 weeks
- PCP feels it's normal growth spurt
- Mom feels it's migraines as there is a strong family history
  - Still wants eyes checked
- VA 20/20 OD/OS uncorrected
- Externals: normal
- Meds
  - Inhaler for asthma PRN
  - Minocycline 50 mg BID PO for acne

OptometricEdu.com/webinars

26

## IIH: "It's not rare if it's in your chair"



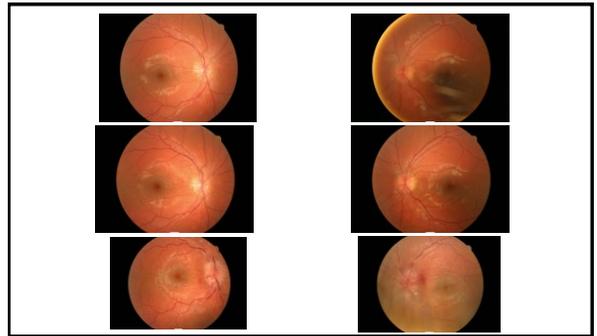
27

### What is the most likely diagnosis?

- Pseudotumor cerebri
- Idiopathic intracranial hypertension (IIH)
- Malignant hypertension
- Intracranial mass lesion

OptometricEdu.com/webinars

28



29

### Precautions With Oral Tetracycline Analogs

- Enhanced photosensitivity
- Avoid in children and pregnancy (Category D)
- Can enhance Coumadin
- Can enhance the action of digoxin
- ?Long term use with increase risk of breast cancer?
  - 1 paper/study, not regarded as highly reliable study
  - Further investigation discredited the association
- Benign intracranial hypertension, reported cases
  - 17 cases from 1978-2002

OptometricEdu.com/webinars

30



31

### 6 Month Later

OptometricEdu.com/webinars

32

### 1 Year Later

OptometricEdu.com/webinars

33

## Case

- A 25-year-old woman was involved in a minor automobile accident where she was hit by another driver. The accident was reportedly minor, with no initial injury to either driver, and both cars were able to be driven away. She felt that she experienced only a mild-to-moderate bump during the accident with no head trauma or loss of consciousness. However, immediately upon waking the next morning, though she had no physical pain, she experienced profound double vision.

OptometricEdu.com/webinars

34

## What is the likely cause?

- A subarachnoid hemorrhage
- A third nerve palsy
- Orbital fracture
- Fourth nerve palsy
- Sixth nerve palsy

OptometricEdu.com/webinars

35

## Case

- She described the diplopia as vertical and worse at near. She had a distinct right hyper deviation which, on alternate cover test, worsened in left gaze and right head tilt. This was a signature motility of a cranial IV (trochlear) palsy.



OptometricEdu.com/webinars

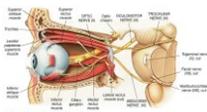
36



37

## CN IV Anatomy

- Exits the midbrain posteriorly and decussates
- Longest course
- Travels around tentorium, through cavernous sinus, through SOF
- Most prone to trauma



OptometricEdu.com/webinars

38

## CN IV Palsy

- Longstanding CN IV palsy may present with diplopia from decompensation
  - Observe old photos for head tilt (*Facebook Tomography*)
- Rule of 40-30-20-10
  - 40% traumatic
  - 30% idiopathic
  - 20% vascular
  - 10% CNS lesion



OptometricEdu.com/webinars

39

## CN IV Management

- Isolated, non-traumatic:
  - Evaluate for ischemic diseases
  - Non-ischemic causes of non-traumatic, isolated CN IV palsy rare
- Look for longstanding decompensation
  - Increased vertical vergences
  - Old photos

OptometricEdu.com/webinars

40

## 66 YOF

- OD disc edema- mild pallor, no hemorrhages or telangiectasia
- OS disc- small, crowded disc at risk; C/D < 0.2
- VA: 20/400 (longstanding macular scar)
- Noticed inferior vision loss x 1 day
  - Inferior arcuate scotoma
- Mild headache- relieved by OTC
- Malaise and loss of appetite- lost 7 lbs
- No jaw claudication or temporal head pain
- What to do?

OptometricEdu.com/webinars

41

## NAAION

- Risk factors:
  - Hypertension, diabetes, atherosclerotic disease, small optic nerves
- Inferior field defects
- Hyperemic swollen nerve- disc at risk
- Progressive moderate vision loss with potential recovery
- Late 30s/ early 40s and beyond
- Painless



OptometricEdu.com/webinars

42

## AAION

- Pallid optic nerve swelling with flame hemorrhages, arteriole attenuation and NFL infarcts
- Pain (of some sort)
- Severe optic nerve dysfunction
- Visual field defects
- Giant cell arteritis/ PMR- risk factors
- Typically 70s, uncommon under 60
- High risk bilateral involvement



OptometricEdu.com/webinars

43

## Diagnosis

- Careful history: Must directly ask about nonvisual symptoms
  - Headache (present in over 90%), scalp tenderness, jaw claudication (almost diagnostic), ear pain, arthralgias, temple pain and/or tenderness, malaise, intermittent fevers
- Examination
- Laboratory studies
  - Erythrocyte sedimentation rate
    - Lowered by statins and NSAIDS
  - C-reactive protein
    - Not affected by statins and NSAIDS
  - Elevated platelet count

OptometricEdu.com/webinars

44

## AAION

### Diagnosis

- Prodrome, GCA symptoms
- Elevated ESR/CRP (combination of the two gives high specificity [97%])
- Elevated platelet count (acute phase reactant)
- Ophthalmoscopy
- Fluorescein angiography
- Temporal artery Biopsy

### Treatment

- Prompt steroids and hydration
- Consider IV when vision loss present
  - Very effective in prevention of second eye
  - Occasionally restores vision
  - Actemra?

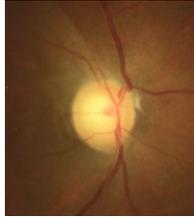
Sudden vision loss in the elderly is GCA until proven otherwise

OptometricEdu.com/webinars

45

## Case 6

- A 46-year-old woman
- She had a history of breast cancer 5 years earlier and was using tamoxifen.
- She reported that she got some cleaning fluid in her right eye 4 weeks earlier.
  - She said that she had some moderate pain and visual blur which subsided, but then several hours later her vision significantly diminished in this eye. She attributed this to the chemical getting in her eye.
  - She was now 20/400 OD (previously she was 20/20).
  - RAPD OD
  - Pale optic disc with attenuated retinal arterioles
  - OCT showed profound retinal thinning.



OptometricEdu.com/webinars

46

## What is the likely diagnosis?

- Arteritic anterior ischemic optic neuropathy
- Non-arteritic anterior ischemic optic neuropathy
- Central retinal artery occlusion
- Chemical injury to the optic nerve
- Orbital tumor
- Optic neuritis

OptometricEdu.com/webinars

47

## Case 6

- Based upon her history of sudden, painless vision loss, a pale disc, attenuated retinal vessels, and profound retinal thinning on OCT, she was diagnosed with central retinal artery occlusion (CRAO). The chemical exposure had no bearing.
- Potential causes:
  - Heart disease, cardiovascular disease, giant cell arteritis, smoking, obesity, carotid artery disease, bacterial endocarditis.
  - Emboli from various sources travel through the vascular system becoming lodged inside the central retinal artery obstructing the flow of blood to distal tissues.
    - Calcific emboli are most likely to cause retinal artery occlusion and are often cardiac in origin.
  - Etiologies related to malfunctioning clotting factors in blood such as antiphospholipid disease, factor VIII abnormality along with protein S and C alteration are also possible etiologies.

OptometricEdu.com/webinars

48

## Case 6

- 46-year-old patient, conditions such as giant cell arteritis, atherosclerosis, hypertension and diabetes are not likely
- Tamoxifen is a selective estrogen receptor
- Tamoxifen has been noted to cause a crystalline retinopathy and should be in the differential of possible causes. However, this patient had a unilateral occurrence and no crystalline maculopathy, thus a direct cause is unlikely.
- Tamoxifen associated with thromboembolic events
- CRAO can be caused by inherited or acquired thrombophilia, especially homocysteinemia. Thrombophilia is also caused by exogenous estrogens (oral contraceptives, tamoxifen).
  - Breast cancer create a pro-thrombotic state with an increased risk of thromboembolism.
- Tamoxifen-associated thrombophilia likely precipitated her CRAO
  - As the occlusion was over a month old, urgent referral to a stroke unit stroke was not required. Communication was made with the patient's primary care physician to inform of the retinal vascular event and initiate investigation for a pro-thrombotic state and discuss the possible association with tamoxifen use and prevention of future thrombotic events.

OptometricEdu.com/webinars

49

## Case 7: 78 YOF

- Sudden onset of ptosis OS
- Immediately following parathyroid surgery
- Headache and eye pain
- Dilation lag and positive iodipine test



OptometricEdu.com/webinars

50

## Discussion

- What is Horner's Syndrome?
  - a triad of clinical signs arising from disruption of sympathetic innervation to the eye and ipsilateral face that causes *miosis*, upper lid *ptosis*, mild elevation of the lower lid, and *anhidrosis* of the facial skin.

51

## What is the most likely cause?

- Lung cancer
- Carotid dissection
- Direct surgical trauma to the nerve
- Migraine

52

## Horner's Syndrome: Etiologies

- First-order neuron disorder: Stroke (e.g., vertebralbasilar artery insufficiency or infarct); tumor; multiple sclerosis (MS), and, rarely, severe osteoarthritis of the neck with bony spurs.
- Second-order neuron disorder: Tumor (e.g., lung carcinoma, metastasis, thyroid adenoma, neurofibroma). Patients with pain in the arm or scapular region should be suspected of having a Pancoast tumor. In children, consider neuroblastoma, lymphoma, or metastasis.



53

## Horner's Syndrome: Etiologies

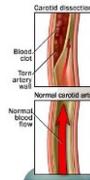
- Third-order neuron disorder: Headache syndrome (e.g., cluster, migraine, Raeder paratrigeminal syndrome), internal carotid dissection, herpes zoster virus, otitis media, Tolosa-Hunt syndrome, neck trauma/tumor/inflammation, prolactinoma.
- Congenital Horner syndrome: Trauma (e.g., during delivery).
  - **Facebook tomography**
- Other rare causes: Cervical paraganglioma, ectopic cervical thymus



54

## Carotid Dissection

Ⓢ A 3rd-order Horner's and ipsilateral head, eye, or neck pain of acute onset should be considered diagnostic of internal carotid dissection unless proven otherwise.



55

## Carotid Dissection

- Carotid artery dissection presents with the sudden or gradual onset of ipsilateral neck or hemicranial pain, including eye or face pain
- Often associated with other neurologic findings including an ipsilateral Horner's syndrome, TIA, stroke, anterior ischemic optic neuropathy, subarachnoid hemorrhage, or lower cranial nerve palsies
- 52% with ocular or hemispheric stroke with 6 days
  - 67% within first week; 89% within 2 weeks; none after 31 days
- Horner's from suspected carotid dissection should go to ER

56

## Case 8: 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

57

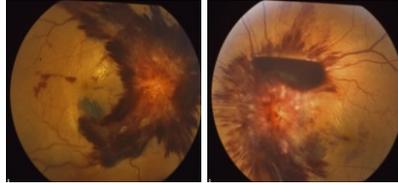
## 35 year old man

- 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



58

## Retinal Findings Discussion



OptometricEdu.com/Webinars

59

## Differential Diagnosis

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

OptometricEdu.com/webinars

60

## 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.

OptometricEdu.com/webinars

61

## 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

OptometricEdu.com/webinars

62

## Case 9: 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

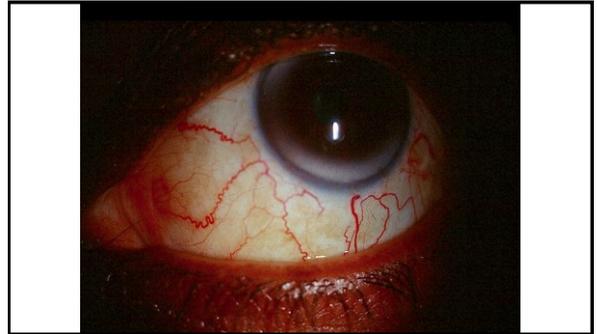
63

### 47 Year Old 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?*

64



65

### 47 Year Old Female

- CT scan:

*What do you think NOW?*

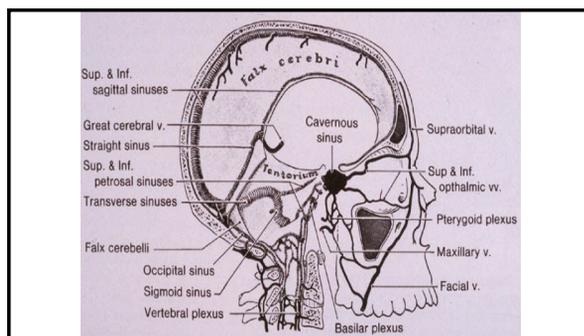


66

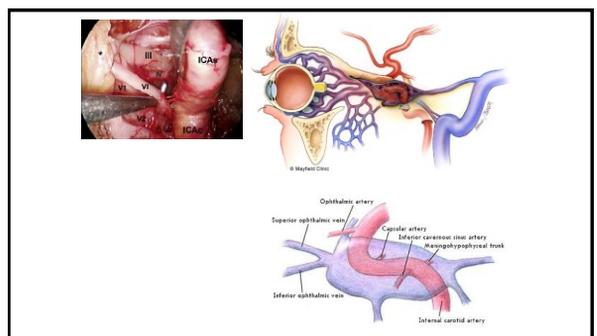
### Carotid Cavernous Sinus Fistula

- 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

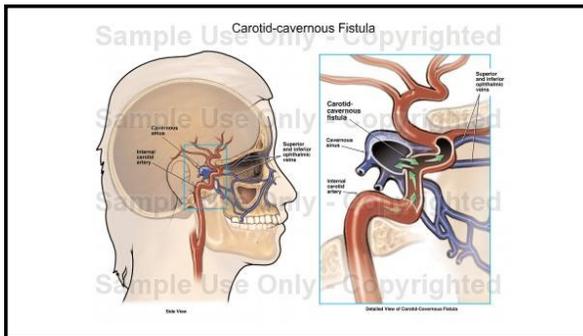
67



68



69



70

## Carotid Cavernous Sinus Fistula

- Hemodynamic
  - High flow vs low flow
- Angiographic
  - ICA vs meningeal branches
- Etiology
  - spontaneous vs traumatic

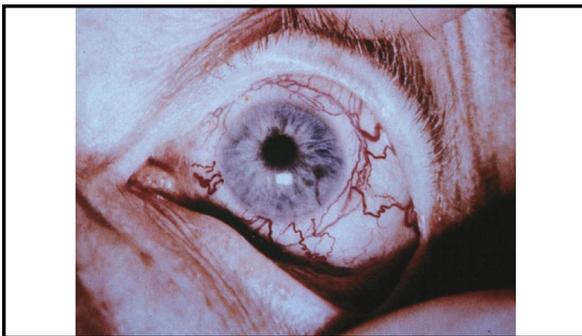
71



72



73



74



75

### Carotid Cavernous Sinus Fistula

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

76

### 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

77



78

### Rule: Beware the Chronic Red Eye

- Dilated & tortuous episcleral vessels that go to the limbus and back (omega loops)  $\Omega$
- 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

 OptometricEdu.com/webinars

79

### Case 10: 23 Year Old White Female

- CC: Sudden onset pupil dilation with ipsilateral headache
- Medical Hx: normal
- BVA: 20/20 OD, OS
- Pupils:
  - 3 mm anisocoria, OS larger, anisocoria greater in bright illumination. Previously isocoric. (-) RAPD, (+) Accom
- Remainder of exam normal
- Similar incident 2 days antecedent, resolved within hours
- What does she look like?

80



81

## 23 Year Old White Female

*What questions do you want to ask?*

*What tests do you want to order?*

82

## 23 Year Old White Female

- Additional questions to ask:
  - *Any double vision?* No!
  - *Any use of ophthalmic pharmaceuticals?* No!
  - *Any history of migraine headaches?* Maybe...

### **Differential diagnosis?**

Aneurysmal compression on CN III? **No**  
Pharmacological misadventure? **No**

83

## Benign Episodic Pupillary Mydriasis

- Episodic unilateral mydriasis
  - Lasts minutes to weeks
- Accompanied by blurred vision and headache
- Young, healthy females (*may have migraine history*)
- Peculiar sensations about affected eye
  - Often progresses to headache
  - Not typical migraine
- Defective accommodation
- Lid and motility defects not present
- Extensive medical testing unremarkable

84

## Benign Episodic Pupillary Mydriasis

- Anisocoria greater in bright than dim
  - Parasympathetic dysfunction
    - Not an aneurysm
    - Edinger-Westphall lesion?
- Migraine variant – most likely etiology
- Treatment – none except to avoid unnecessary testing

85

## Pupil Rules

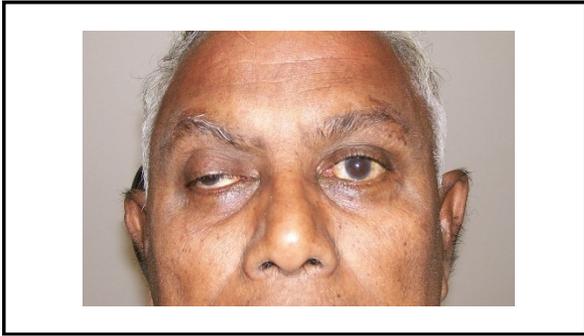
- Anisocoria greater in dim = sympathetic dysfunction
  - Horner's syndrome- look for dilation lag
  - Miotic use
- Anisocoria greater in light = parasympathetic dysfunction
  - CN 3 palsy
  - Tonic pupil
  - Pharmacologic or traumatic pupil
    - No reactivity?

86

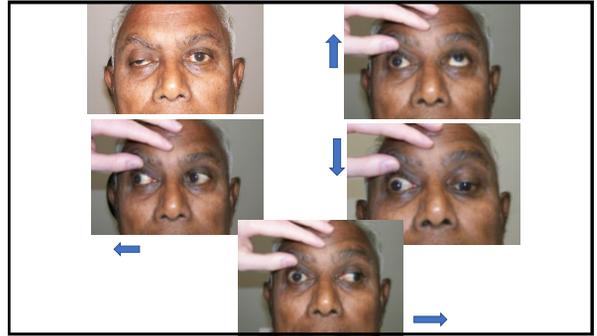
## 63 YOIM

- Long standing glaucoma patient
- Sudden onset of orbital pain x 3 days
- + DM; +HTN
- On coumadin
- Pacemaker
- No vision change
- Presents as walk-in emergency glaucoma eval

87



88



89



90

**63 YOIM**

- Pupil involved CN III palsy
- 3 days duration at least
- Most likely cause: intracranial aneurysm
- Sent to ED with detailed notes and recommendations
- Endovascular therapy with coils
- Hospitalized 23 days

OptometricEdu.com/webinars

91



92

**CN III Palsy Clinical Picture**

- An eye that is down and out with a ptosis
- Adduction, elevation, depression deficits
- Isocoric or anisocoric

OptometricEdu.com/webinars

93



94

### CN III Anatomy

- Vulnerable to compression from aneurysm in subarachnoid space
  - Posterior communicating artery (PCOM)
  - Junction PCOM and ICA
  - Tip of basilar artery

OptometricEdu.com/webinars

95

### Still More Clues

- Pupil involved CN III palsy is PCOM aneurysm until proven otherwise
- Incomplete palsy is PCOM aneurysm until proven otherwise
  - Regardless of pupil
- 30% of CN III palsy are caused by aneurysm**
- Pain is pain
  - Only helpful when not present
- Vasculopathic CN III will resolve in time
- Life threatening posterior communicating aneurysm will rupture in time

OptometricEdu.com/webinars

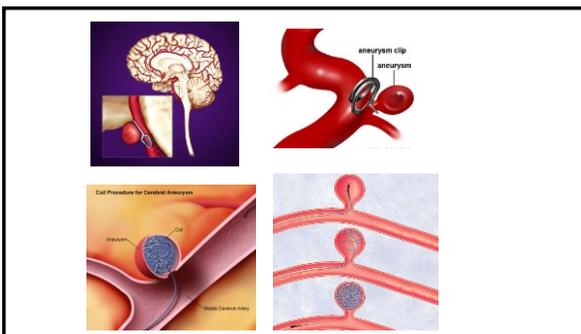
96

### Still More Clues

- CN III palsy caused by aneurysm
  - 20% die within 48 hrs from rupture
  - 50% overall die
  - Average time from onset to rupture – 29 days
    - 80% rupture w/1 29 days
    - Many never make it to hospital
- Ruptured aneurysms
  - 5% surgical mortality
  - 60% functional impairment post-op
- Unruptured aneurysms
  - No mortality; 75% with normal outcomes; 50% with CN III recovery

OptometricEdu.com/webinars

97



98

### Rule

**Never dilate a patient with cranial nerve III palsy**

OptometricEdu.com/webinars

99

### Rules for CN III palsy imaging

- High suspicion of aneurysm: DSA (gold standard)
- CT/CTA is preferred non-invasive imaging for CN III palsy
  - CT for SAH
- CTA requires contrast- renal impairment prefers MRI/MRA
- CTA superior to MRI when patient can't have MRI
  - Pacemaker, claustrophobia
- MRI superior for non-aneurysmal causes (tumor)
  - MRA adds very little time to scan

OptometricEdu.com/webinars

101

OptometricEdu.com/webinars

102

### A Different patient and Prognosis

- 63 YOF
- Diabetes and HTN
- Sudden onset retro-orbital pain

OptometricEdu.com/webinars

103

Complete CN III palsy with pupil sparing and vasculogenic risk factors

OptometricEdu.com/webinars

104

### Which is better? One or two?

Resolves over several weeks

Hospitalized 23 days with 2 neurosurgical procedures

OptometricEdu.com/webinars

105

### Suspect the worst

- Optometrist sees patient with CN III palsy
- Referred to ophthalmologist next day
- Pt dies from SAH before consult

OptometricEdu.com/webinars

106

## Does presence of vasculopathic risk factors help?

- Arteriosclerotic risk factors in elderly favors microvascular etiology but does not rule out aneurysm
- HTN, DM, atherosclerosis, hypercholesterol all common and don't protect against aneurysm
- Answer: no, but makes me very nervous when NOT present

OptometricEdu.com/webinars

107

## Does acuteness of presentation help?

- Ans: Yes and No
- Aneurysm expansion usually produces acute manifestations, but chronic and evolving cases well known
- Acute is more worrisome
- Chronic and improving less worrisome but does not rule out aneurysm
- Resolved without recurrence reassuring

OptometricEdu.com/webinars

108

## Aneurysm Risk Assessment: Isolated CN 3 palsy

- Isolated dilated pupil none
- Complete CN3-normal pupil low
- Partial CN3 – normal pupil high
- Pupil involved CN3 **emergency**

OptometricEdu.com/webinars

109

## Never out of the woods

- Pt develops CN III palsy from aneurysm
- Successfully treated with aneurysm clip
  - All coils are inert and MRI safe; not all clips are MRI safe
- Radiologic tech doesn't verify type of clip
- Pt undergoes F/U MRI with non-MRI safe clip in major medical center
- Clip displaces during MRI
- Patient has fatal hemorrhage during procedure
- Patient survived disease...killed by follow up

OptometricEdu.com/webinars

110

## Pupil Rules

- Fixed and dilated and unresponsive to light or near = pharmacologic or iris trauma



OptometricEdu.com/webinars

111

## Rule: Isolated Dilated Pupil Is Almost NEVER An Aneurysm

**Ambulatory patients with isolated dilated pupil more likely to harbor iris or ganglion (Adie's) lesion or medication misadventure than CN 3 palsy**

**Comatose patient is a different story**

**Risk of angiography is much higher than risk of aneurysm in this setting**

**No imaging needed for isolated dilated pupil**

OptometricEdu.com/webinars

112