US EYE
BETTER TOGETHER

PHONE A FRIEND: THE GREATEST CASES THAT I *NEVER* SAW

Joseph Sowka, OD, FAAO, Diplomate

DISCLOSURE:

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- *Joseph Sowka, OD* is/ has been a Consultant/ Speaker Bureau/ Advisory Board member for B&L. Dr. Sowka has no direct financial interest in any of the diseases, products or instrumentation mentioned in this presentation. All disclosures have been mitigated. He is a co-owner of Optometric Education Consultants (www.optometricedu.com)

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Further Disclosures

- I work in a large medical-surgical practice, not an academic referral center.
- I book 25-30 patients per day, including primary care, glaucoma, cornea, emergencies, etc.
- I function much as everyone here today.
- I don't have 2 hours to do a neuro-op evaluation.
- Images presented here may be of low quality and information may be scant, but it is what I had to work with… and it was enough.

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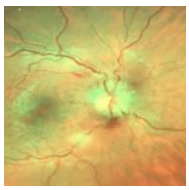
Case 1

- 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



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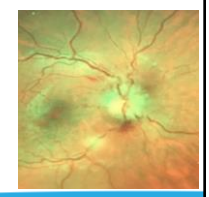
Polling Question 1: What is the likely diagnosis?

- Demyelinating optic neuritis
- Non-arteritic anterior ischemic optic neuropathy
- Arteritic ischemic optic neuropathy
- Compressive optic neuropathy
- Infectious optic neuropathy
- Hereditary optic neuropathy
- Infiltrative optic neuropathy
- Perineuritis
- Papilledema
- Help! I don't know. That's why I am here



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Case 1

- 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.

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Other Considerations

- MOG antibody disease (MOGAD) is a neurological, immune-mediated disorder in which there is inflammation in the optic nerve, spinal cord and/or brain. Myelin oligodendrocyte glycoprotein (MOG) is a protein that is located on the surface of myelin sheaths in the central nervous system.
  - Anti-MOG antibodies
- Neuromyelitis optica spectrum disorder is a rare inflammatory disease that most often affects the optic nerves and spinal cord. Less often, it affects the brain. It often leads to sudden vision loss, paralysis or both. Symptoms after a first attack usually improve.
  - Aquaporin-4 (AQP4) antibodies

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Diagnosis: Infectious Optic Neuropathy

- MRI findings showed optic nerve enhancement possibly consistent with infectious, autoimmune, or granulomatous disease with no evidence of demyelination.
  - Not bilateral, longitudinally extensive, or chiasmal
    - So not MOG or NMOSD
  - "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.

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INFECTIOUS OPTIC NEUROPATHY

- Syphilis
  - Retrobulbar, papillopathy, neuroretinitis, perineuritis
  - Retrobular, bulbar: severe vision reduction
  - Perineuritis has normal vision, MRI optic sheath enhancement
- Lyme - mimics syphilitic optic neuropathy
  - Bite of mammalian deer tick- can cross react with syphilis
- Toxoplasmosis, HIV/AIDS, CMV
  - Destructive to vision
- Neuroretinitis
  - Typically benign lymphoreticulosis (cat scratch disease)

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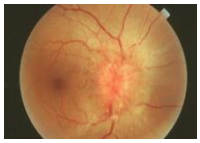
Neuroretinitis

- 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



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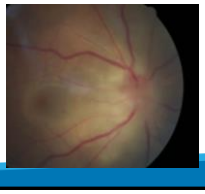
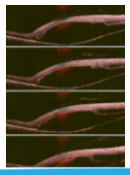
62 YOF

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





Neuroretinitis/ Infectious Optic Neuropathy

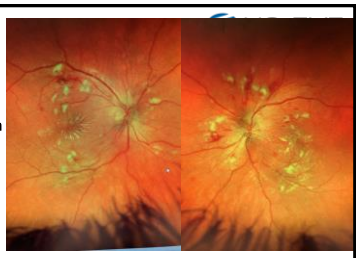
- Many potential etiologies
  - Toxoplasmosis, toxocariasis, measles, syphilis, Lyme disease, herpes simplex and zoster, E-B-V mumps, tuberculosis, malignant hypertension, ischemic optic neuropathy, and leptospirosis, *bartonella* (most common). Fleas are vectors, thus no need for actual scratch.
- Prognosis 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.

Pertinent Information

- Red herrings: Young female with painful vision loss from an optic neuropathy
  - Makes one think ON and MS

- Important information:
  - Papillitis
  - Disc hemorrhage doesn't happen in optic neuritis

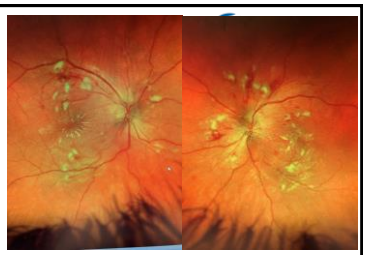
- 22-year-old female
- First eye exam- blurred vision
- 20/40 OD; 20/70 OS
- No medical history
- Thin build



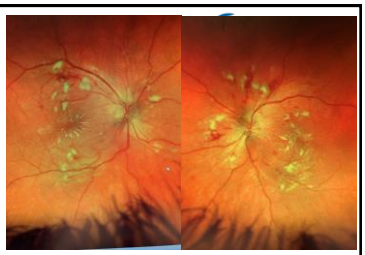
Polling Question 2: What is the likely diagnosis and plan?

- 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
- Help! I don't know. That's why I'm here

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



- Red herrings
  - Young
  - Thin build
  - No medical history
- Important findings
  - Degree of retinal ischemia



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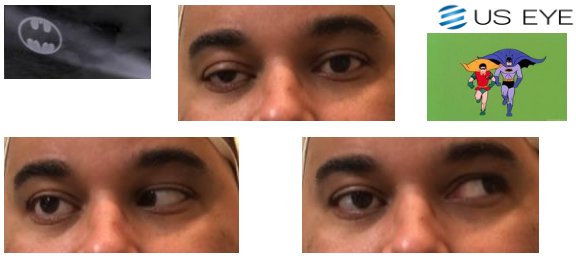
COVID Lockdown

- 39 YOM: Seen by former resident
- Previous history of migraine developed a new and worsening headache with lethargy.
- He presented to a hospital emergency room where he underwent a non-contrast enhanced computed tomography (CT) and magnetic resonance imaging (MRI) which were subsequently interpreted as normal.
  - His headache was attributed to migraine, and he was medicated as such and discharged.
- Three days later, he developed horizontal and vertical diplopia



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COVID Lockdown

- His visual acuity and visual fields were normal.
- He manifested a right pupil-sparing, external partial cranial nerve three palsy and concurrent right sixth nerve palsy. He also complained of worsening headache and lethargy.
- Where is the lesion?

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Polling question 3: What is the problem?

- An intracranial aneurysm
- Migraine
- A brain tumor
- I don't know, that's why I am here

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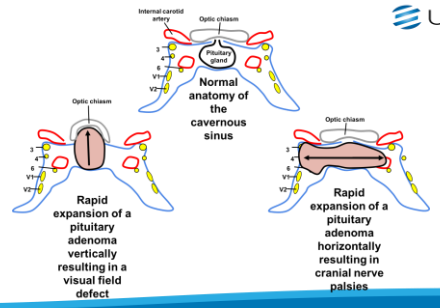
COVID Lockdown

- Let's contact the radiologist for a second reading…
- He was immediately sent for repeat imaging to include contrast-enhanced MRI of the parasellar area and MRA to rule out intracavernous aneurysm and pituitary apoplexy.
- Imaging revealed a pituitary macroadenoma with intratumor hemorrhage consistent with pituitary apoplexy.
- Lateral spread into the right cavernous sinus and possible spread into the left cavernous sinus as well.
- No mass effect on the optic chiasm or prechiasmal intracranial portion of the optic nerve.
  - Hence normal acuity and fields
- The patient was immediately admitted for endocrinological and neurosurgical evaluation

Pituitary apoplexy

- Pituitary apoplexy is a severe and potentially fatal medical condition complicating 2-12% of pituitary adenomas and characterized by the variable association of headache, vomiting, visual impairment, ophthalmoplegia, altered mental state and consciousness, lethargy, and panhypopituitarism.
- Hemodynamic instability may be result from adrenocorticotrophic hormone deficiency, which can be fatal.
- Occurs due to a rapid expansion, mainly caused by hemorrhage or infarction of a preexisting (known or unknown) adenoma



Pituitary apoplexy

- Most common presenting symptom occurring in 90% of patients is sudden onset of severe headache
  - Commonly described as frontal or retro-orbital.
    - Pituitary apoplexy is often overlooked as a possible cause of "thunderclap headache" where diagnostic evaluations tend to direct to more common causes of this presentation including subarachnoid hemorrhage, cerebral venous sinus thrombosis, and cervical artery dissection.
- Approximately 50% have visual abnormalities
  - Blurred vision
- Cranial nerve palsy (CN III) or palsies
  - Cranial nerve VI most common, followed by CN III
- Visual field defects
  - Bitemporal hemianopsia
- Facial weakness

Pituitary apoplexy

- Most symptomatic patients undergo CT scanning in an emergency setting due to the clinical suspicion of acute intracranial hemorrhage
- Acute hemorrhagic infarct may be seen on CT
  - Non-hemorrhagic infarcts will usually show no abnormalities without intravenous contrast
- MRI with contrast is the most effective imaging in cases of suspected pituitary apoplexy
  - MRI is superior to CT

Pituitary apoplexy

- Positive outcome in most cases
  - Conservative medical treatment
  - Stabilize and replace diminished pituitary hormones
- Surgical decompression
  - Trans-sphenoidal or subfrontal transcranial approach
  - Patients with visual impairment and neuro-ophthalmic dysfunction will be selected for surgery.
- Patient was medically stabilized, and surgery delayed due to COVID lock down
- Ultimately underwent successful surgical decompression

Pertinent Information

- Red herrings:
  - No field loss and no vision loss
    - Pituitary apoplexy typically has new onset bitemporal defect…but not always
  - Normal CT and MRI
  - Had been "worked up" in hospital
  - Already diagnosed with migraine

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Pertinent Information

- Worsening headache
  - Rapidly expanding mass
- Lethargy
  - panhypopituitarism
- Multiple cranial neuropathies
  - Localizes to cavernous sinus or posterior orbit
- Normal acuity
  - Optic nerve is in posterior orbit, so normal acuity rules that out
  - Must be cavernous sinus

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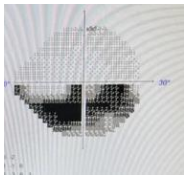
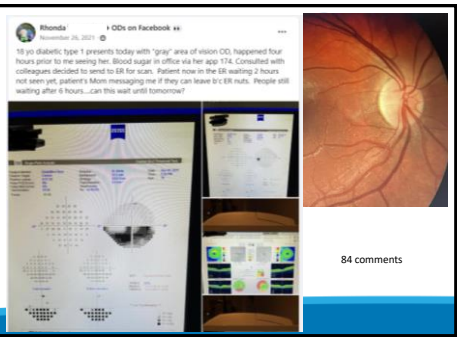
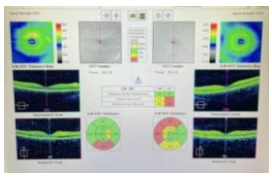
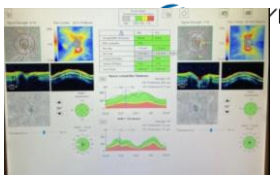
Case From ODs on Facebook School of Medicine





84 comments





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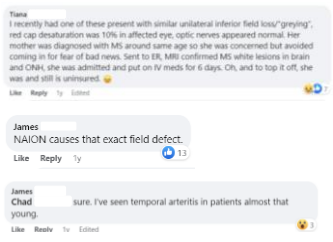

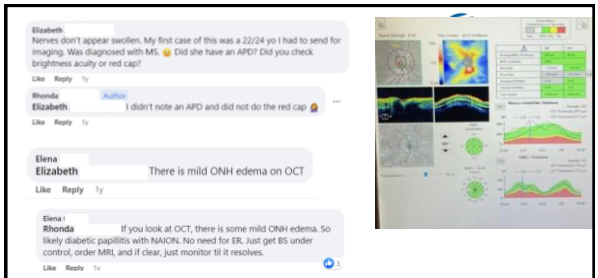

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Paul
Yes it can wait until tomorrow
Like Reply 1y

Marc
Is she on birth control..... NAION
Like Reply 1y 3

Josh
I would think a diabetic papillitis is the most likely cause based on the information given. Careful recheck of AFD maybe confirm with red cap. I'd also want a fundus photo. I would probably boot to retina and let them sort it.
Like Reply 1y Edited

Jay
Josh hat would be a variation of NAION, and ONH would be swollen with fundus eval
Like Reply 1y

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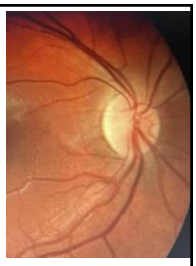
Josh
Rhonda r this may be a bit of a stretch but is there a bit of a blurred margin at 12 o'clock on the nerve?
Like Reply 1y 1

Jill
Young female most likely optic neuritis. Rule out/in MS with mri brain and orbits with and without contrast with Flair sequences. Could have been done outpatient including IV steroids but considering the holidays (and T1DM) probably best they admit given steroids and issues this will create with BS. Hopefully neurologist will get involved in the ER
Like Reply 1y 1

Dorothy Group expert
AiON
Like Reply 1y



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Polling question 4: What is the likely diagnosis?

- Optic neuritis/ MS
- Diabetic papillitis
- Temporal arteritis
- NAAION
- None of the above

- If you choose "None of the above", what do you think that it is?
  - Put it in the chat box

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NO GOOD SAMARITAN HERE

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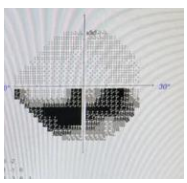
Sunday morning in my email inbox ☹



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Hello Greg,

Can you please help me with this patient? I saw her yesterday at my office, she is my patient, I want to high school with her Mother. She is an 18 year old type I diabetic. She presented yesterday saying that she couldn't see the bottom half of her vision (OD since finishing basketball practice yesterday. At the past she would mention intermittent areas of "gray vision" that would come and go but not lasting more than 15 minutes. I thought these intermittent episodes were possibly ocular migraines secondary to lasting 15 minutes or so. She and her Mother admit that her blood sugar hasn't been the best lately, in office yesterday via her app her blood sugar was 174. Currently she is on an insulin pump. Currently she is not taking BCP, she tried it in the past and hasn't been it for two months.

Entering uncorrected acuities were 20/40 out of each eye, which is consistent with past exams. I did not refract her. I did not note an AFD but was thinking there might likely was one present after seeing her VF. IOP was normal. I did not do color vision testing or the red cap test. You will see her VF attached. There is a hemianopsia OD. I've also attached her fundus photos and OCT. I consulted with an OMD yesterday and her advised me to send her to the ER for a scan secondary to vision loss. I should have known better, I realize this now, as the ER was a complete nightmare, but I was hoping Dr. ____ the OMD on call would have seen her. He did not. The only doctor that seen her was the "drama doctor" and he seemed annoyed that she was seen at the ER.

They did do a CT scan as well as some blood work. I have attached the results from the patient portal that her Mother sent to me via a screen shot. I put this out on FB and some Doctors had similar presentations and it turned out to be NAION and MS. Some were saying they saw a little ONH edema and this could be early diabetic ONH resolving. I was also getting criticized for sending her to the ER. However, in the past I did have patients with ischemic optic neuropathies and some responded well to IV steroids some were left untreated and had vision loss for the rest of their lives. And due to the sudden onset and lasting vision loss I felt it needed attention ASAP.

I am really bothered by the lack of concern and support and I am worried about her vision.

What would you do next? MRI on Monday?? I really need help in managing this patient and it seems I am not getting anywhere. I spoke to her Mom this morning and her vision is still the same.

Thank you for your help.

Miranda

From: Greg Caldwell <gnubold@gmail.com>
Date: November 27, 2021 at 4:11:04 PM EST
To: Joe Sowka <joesowka@gmail.com>
Subject: Fwd: BB info

I was going to suggest she send to me, will you help me?

Dr. Greg Caldwell
Fellow of the American Academy of Optometry and Diplomate, American Board of Optometry
614-931-2030 mobile #

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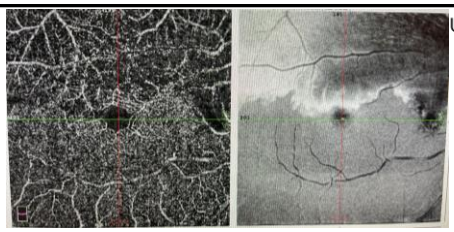
GREG, USE EVERYTHING FROM FB AS THE "ANTI-DIFFERENTIAL DIAGNOSIS" AS THEY ARE ALL WRONG.
IT IS RETINA.
USE OCT AND OCTA

US EYE

US EYE



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Red herrings:

- Fundus and disc normal
  - This is how acute artery occlusion present
- Young
  - Not the typical profile
- Multiple opinions where the "findings were made to fit the diagnosis"
  - NAAION
    - Too young and disc not swollen
  - AAION
    - Are you high?
  - Diabetic papillitis
    - No vision loss in DP
  - Optic neuritis
    - Painless

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Pertinent Information

- Acute, painless loss of vision
- Just happened
- Fundus and disc normal

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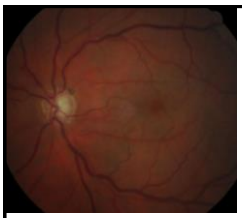
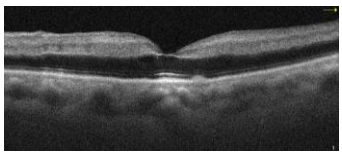




Guidelines

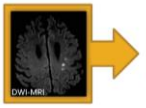
- Any patient with suspected TIA or those with acute retinal ischemia should be evaluated urgently in order to identify those at high risk of immediate cerebral infarction and cardiac ischemia

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 2011; 42: 227-276

Adapted from Drs. Nancy Newman and Valerie Biousse; 2015

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 Valerie Biousse; 2015

DWI in Acute Retinal TIA/Ischemia

- DWI-MRI identifies subgroup of patients at very high risk of major stroke
- DWI-MRI needs to be performed within 24/48 hours of visual loss to allow for effective prevention of recurrent stroke

Adapted from Drs. Nancy Newman and Valerie Biousse; 2015

TIA/ Acute Retinal Arterial Ischemia

- 10-15% of patients with TIA/ acute retinal arterial ischemia with have disabling stroke within 3 months and half will occur within 48 hours.
- DWI-MRI within 48 hrs
- CTA of cervical/ intracranial arteries
- EKG/ Echocardiogram
- Labs:
  - CBC with platelets
  - Coagulation assessment
  - lipids

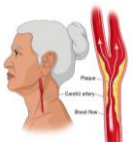
Tell the patient:

- "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 Valerie Biousse; 2015

Polling question 5: A patient with a TIA/ RAO has mild carotid artery stenosis (<50%): The issue is therefore not caused by carotid artery disease

- Agree
- Disagree



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Finding the Etiology- New Thoughts

- Carotid origins of stroke rely upon degree of stenosis
- Low degree of stenosis (<50%) leads to dismissal as cause
  - Not 'bad disease'
- Advanced MRI methods can identify high-risk features of carotid plaques
  - Symptomatic nonstenotic (<50%) carotid disease (SyNC)
- Carotid atherosclerosis on the ipsilateral side (symptomatic) to recent ischemic event (within the last 7 days) shows significantly higher rates of *high-risk features* such as intraplaque hemorrhage, fibrous cap ulceration, and intramural hematoma.

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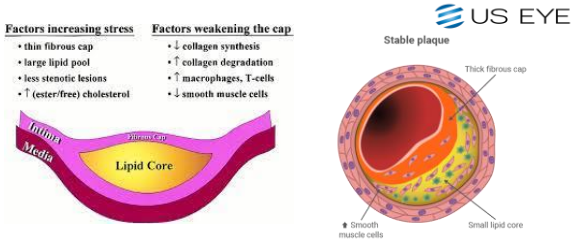

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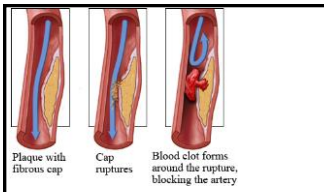
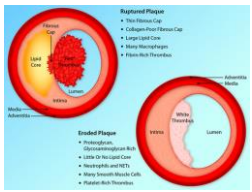




US EYE

Finding the Etiology- New Thoughts

- Symptomatic nonstenotic carotid disease (SyNC), seem to cause strokes
- Catheter angiography allows for assessment of stenosis degree but is unable to depict intrinsic plaque features.
  - Same for ultrasound Doppler
- Dedicated MRI sequences to detect plaque features such as intraplaque hemorrhage, a lipid-rich necrotic core, and fibrous cap rupture/irregularity identify lesions prone to causing infarct without being overly stenosed/ occluded

US EYE

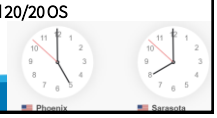
Finding the Etiology- New Thoughts

- Requires a dedicated MRI protocol
  - May not be feasible in many centers
- CTA has now become standard imaging for possible acute ischemic stroke and is performed in almost all patients with AIS.
- Next step- establish imaging markers using CTA.
- No single reliable CTA plaque imaging biomarker has been identified so far.
- This is why we need to work with professionals
- New information coming

US EYE

Help, I think I have a 3rd!

- 75-year-old Caucasian male
- Longstanding complaints of diplopia
- He denied any acute episodes of diplopia in the past and was never diagnosed with any cranial neuropathy.
- Malignant, nonmetastatic intestinal cancer, which was diagnosed 7 years ago and for which he underwent surgery to remove
- Best corrected visual acuity was 20/100 OD and 20/20 OS
  - Lamellar macular hole OD
- PERRL (-) RAPD



US EYE

- Right adduction deficit, as well as limited supraduction and infraduction.
- *No Ptosis*
- Pupil normal and reactive- *no constriction with attempted adduction*



US EYE

US EYE

Polling Question 6: Is this a 3rd nerve palsy?

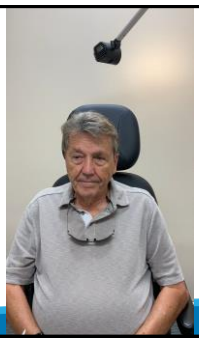
- Yes
- No
- Kind-of, sorta, maybe-ish

US EYE

Polling Question 7: What is the problem?

- He has a 3rd nerve palsy from an aneurysm
- He has a third nerve palsy from a tumor
- He has myasthenia gravis
- He has primary aberrant regeneration from a cavernous sinus mass

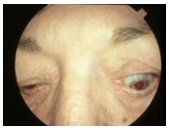
US EYE

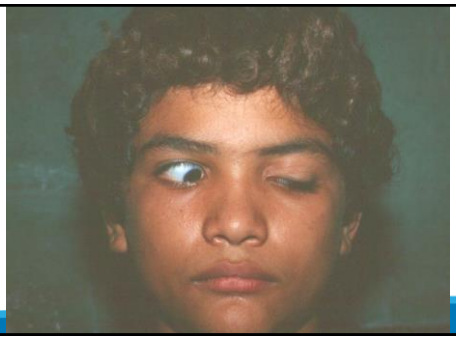


US EYE

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)
    - Most characteristic
  - Patient looks medial: pupil constricts





Pseudo-Von Graefe's sign
Secondary aberrant regeneration
Never diabetes

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.

Outcome

- MRI obtained with attention to orbit, parasellar area, cavernous sinus, and MRA Circle of Willis.
- MRI showed soft tissue mass in right cavernous sinus encompassing ICA and consistent with meningioma or lymphoma
- Contacted oncologist who ordered PET scan to rule out lymphoma
- Treatment options: surgery, radiotherapy, observation
  - Observation

Dissecting the pertinent issues

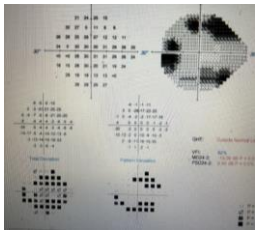
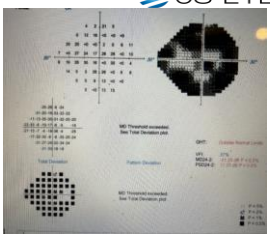
- 3rd nerve dysfunction, but no ptosis
- Chronic
- Most prominent feature of aberrant regeneration not present

- Key feature was lid synkinesis
  - Lid lowers with abduction and elevated with attempted adduction

Help!

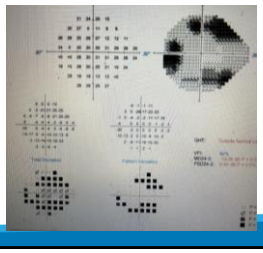
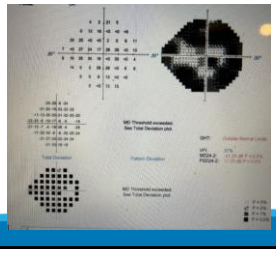
- 30 YOM
- White "looks weird" OD x 1 day
- 20/20 OD, OS
- PERRL (-) RAPD
- Dilated exam normal
- Feels worse next day
- Med hx: unremarkable?
- PCN x 10 days for sinus infection
- Fields:

US EYE

US EYE

HAVE YOU EVER HAD A BILATERALLY CONSTRICTED FIELD PATIENT OF UNKNOWN ETIOLOGY? - CHAT

US EYE

- CT and CTA contrast- normal
- Difficulty getting MRI in the area
- So, what will cause bilateral sudden field constriction in a 30 year old with normal imaging?
  - Intracranial disease?
  - Something ingested?
  - Bilateral optic neuropathy?

US EYE

Let's Talk About Toxic Optic Neuropathy

- Toxic optic neuropathy is characterized by bilateral, usually symmetric vision loss, papillomacular bundle damage, central or cecocentral scotoma, and reduced color vision.
- Nutritional deficits, including the vitamins thiamine (B1), riboflavin (B2), niacin (B3), pyridoxine (B6), cobalamin (B12), folic acid, and proteins with sulfur-containing amino acids that can trigger or enhance toxic optic neuropathy
- The use of systemic medications in high doses or for a prolonged duration
- Exposure to a toxic substance in the environment

US EYE

Toxic Optic Neuropathy

- Alcohols: Commercial alcohol, Methanol, Ethylene glycol
- Antibiotics: Chloramphenicol, Sulfonamides, Linezolid
- Antimalarials: *Hydroxychloroquine*, Chloroquine, Quinine
- Antitubercular: Isoniazid, *Ethambutol*, Streptomycin
- Antiarrhythmic: Digitalis, *Amiodarone*
- Anticancer: Vincristine, Methotrexate, Tamoxifen
- Anti-seizure: Vigabatrin
- PDE inhibitors: Sildenafil
- Heavy metals: Lead, Mercury, Thallium
- Other: Carbon Monoxide, Tobacco

US EYE

Symptoms

- Loss of central or paracentral visual acuity
- Reduced contrast perception
- General loss of color perception, particularly red
- Photophobia
- Poor dark adaptation

US EYE

Exam

- No RAPD, when bilateral disease
- Normal, swollen, or hyperemic optic disc in early disease
- Temporal optic disc pallor in late disease
- Centrocecal scotoma in visual fields with preservation of the peripheral field.
- Decreased visual acuity
- Photophobia
- Abnormal Visual Evoked Potentials (VEP) or Electroretinogram (ERG)

US EYE

Lab Testing

- Complete blood culture and urinalysis to screen for specific toxins
- Heavy metal screening if heavy metal toxicity is suspected
- Serum B-12 and folate levels if the patient presents with bilateral central scotomas
- Neuroimaging

US EYE

GREAT INFORMATION, BUT IT HAS *NOTHING* TO DO WITH THIS CASE

US EYE

10 Causes of Tunnel Visual Fields/ Bilaterally Constricted Fields

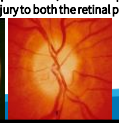
- 5 optic nerve causes
- 2 retinal causes
- 1 intracranial cause
- 1 psychophysical cause: Bad testing (fatigue, artifact, inattention, poor instructions)
- 1 default cause: Malingering/ Psychological/ Non-organic

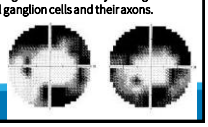
US EYE

5 Optic Nerve Causes of Bilaterally Constricted Visual Fields

- End-stage glaucoma
- End-stage papilledema (post-papilledema optic atrophy)
- Optic nerve drusen (asymptomatic)
- Optic nerve hypoplasia (asymptomatic)
- Vigabatrin optic neuropathy
  - Anticonvulsant for refractory focal epilepsy in children 2 years of age or older
  - May cause permanent, concentric peripheral visual field loss, thought to be secondary to drug-induced injury to both the retinal photoreceptors and the retinal ganglion cells and their axons.



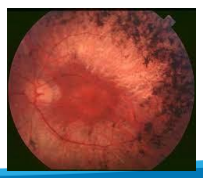
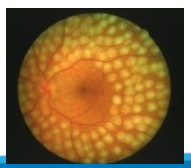





US EYE

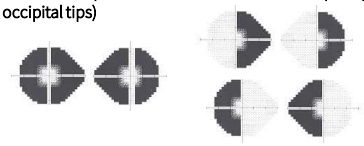
2 Retinal Causes of Bilaterally Constricted Visual Fields

- Retinitis pigmentosa and RP-like conditions
- Peripheral retinal scarring and degeneration (includes PRP scarring)

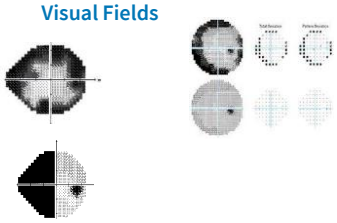
1 Intracranial cause of Bilaterally Constricted Visual Fields

- Bilateral occipital lobe infarcts with "macular sparing" (sparing of occipital tips)



1 Psychophysical Cause of Bilaterally Constricted Visual Fields

- Bad Testing
  - Fatigue, Inattention
  - Artifact
  - Poor instruction



1 Default Cause of Bilaterally Constricted Visual Fields

- Psychological/ Malingering/ Non-organic

Outcome

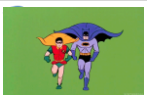
- Neuroimaging normal
- Serology normal except for low B12 levels
- *ONE* B12 pill solved everything.
- = Non-organic vision loss

Pertinent Information

- Acute simultaneous bilateral constricted field loss
- Not respecting vertical or horizontal midline
- Fundus and disc normal
- Initial imaging normal
- Strongly consider toxicity

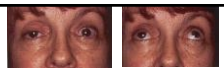
- Red herrings:
  - Constricted fields, not cecocentral loss

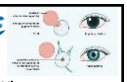
When getting worse is good



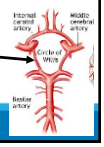
- 71 YOF
- Is 4 days cataract PO OD and 7 days PO OS
  - Has significant headache and normal PO appearances
- 6 days later still has headache/ retro-orbital ache and develops horizontal and vertical diplopia.
- Call and text from worried colleague

US EYE





- Pupil involved CN III palsy is posterior communicating (PCOM) aneurysm until proven otherwise- emergency
  - Needs ER STAT and admission
- 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
  - Aneurysms are always painful
  - Ischemic palsies are painful 90% of the time



US EYE

One day later

US EYE

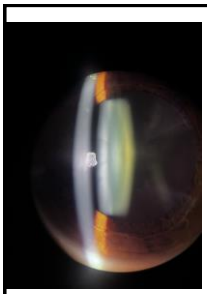
Three days later

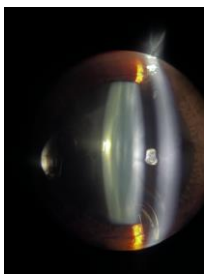
US EYE

Progressive painless vision loss

- 39 YOF- former student
- Calls with concerns about painless progressive vision loss OD for 1.5 years- 20/50 OD, 20/20 OS
- Corneal topo, pupils, color, OCT, fields- normal OU
- Has seen cataract, retina- normal
- Has had 2 MRIs- normal
- Has seen renowned neuro-ophthalmologist at prestigious university- no diagnosis
  - Refers to more renowned neuro-ophthalmologist at more prestigious university
- Wants to order "antibodies against optic nerve"
  - MOGAD/ NMOSD
- Pertinent information- PH better than refraction, loss of accommodation OD only,
  - -1.00 shift but not able to read

US EYE



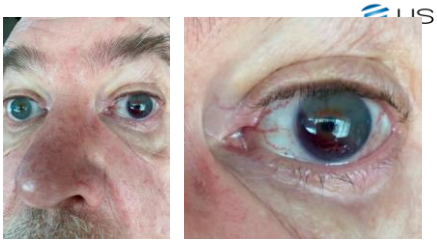


US EYE

POST-OP RED EYE

- 73 YOM- cataract and mild glaucoma
- Underwent cataract extraction with iStent the day before
- On-Call emergency call- 6:30 am
- "Woke up and my eye was all red"
- "Can't really see"

US EYE

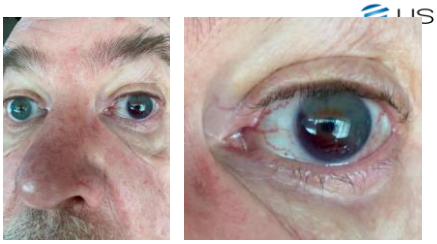


US EYE

Polling question 8

- Not a problem because hyphema commonly occurs after iStent
- Not a problem because blurred vision is from corneal edema
- Problem because the IOL dislocated
- Problem but I'm not sure why

US EYE



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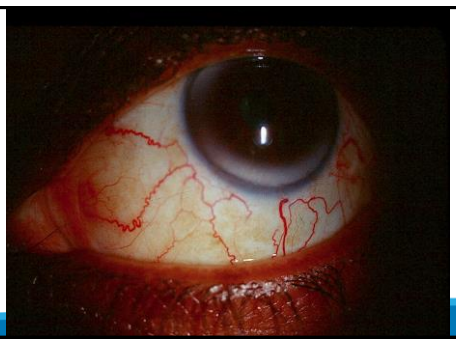

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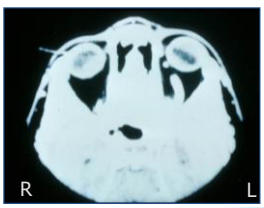
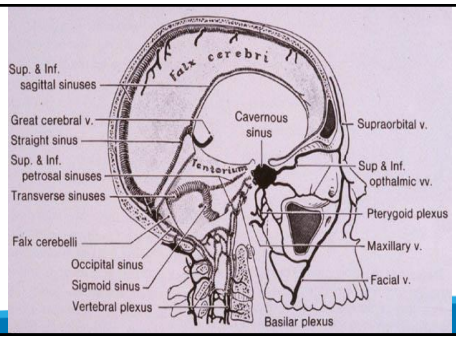
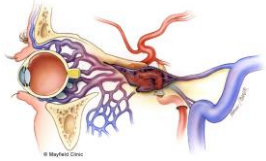
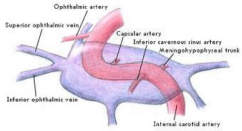


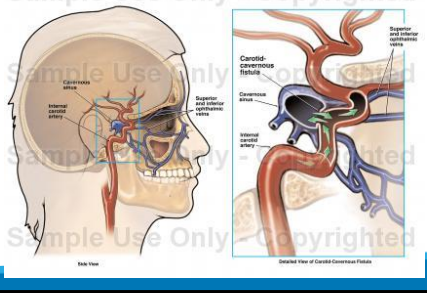
CAROTID CAVERNOUS SINUS FISTULA

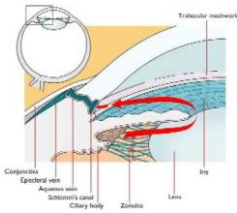
- Cavernous sinus…
  - Trabeculated venous cavern
  - Houses CN III, IV, VI, V1, oculosympathetics, and ICA
  - Drains eye and Adnexa via inferior and superior ophthalmic veins to petrosal sinuses and jugular vein
- Fistula…
  - Rupture of ICA or meningeal branches within sinus
    - Meningeohypophyseal, McConnell's Capsular, Inferior Cavernous
  - Mixing of arterial blood in venous system

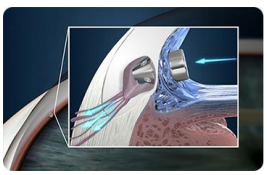






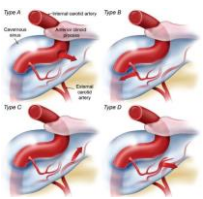


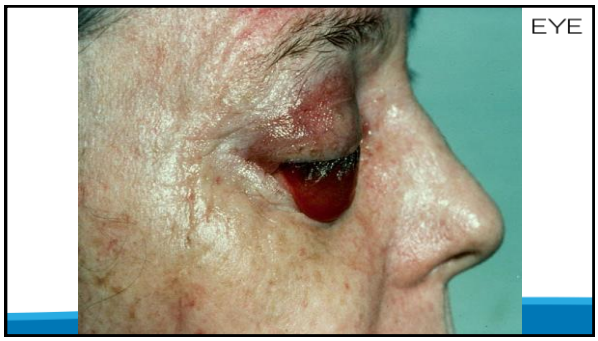


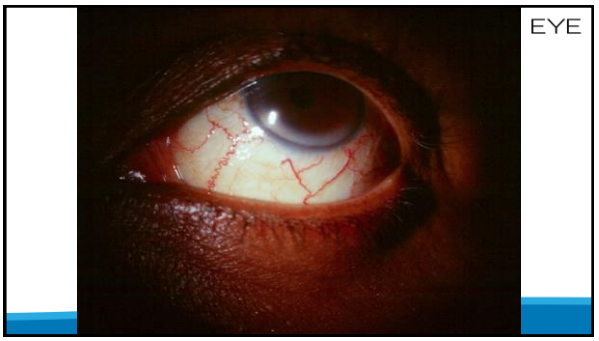


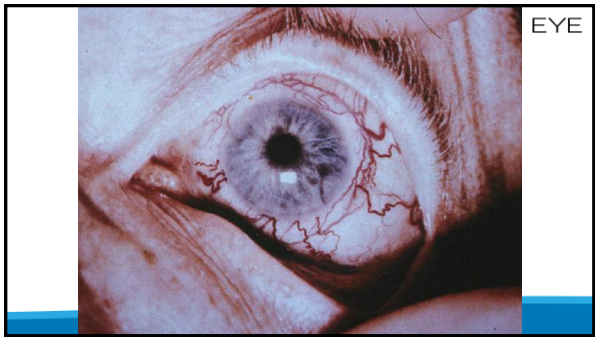
CAROTID CAVERNOUS SINUS FISTULA

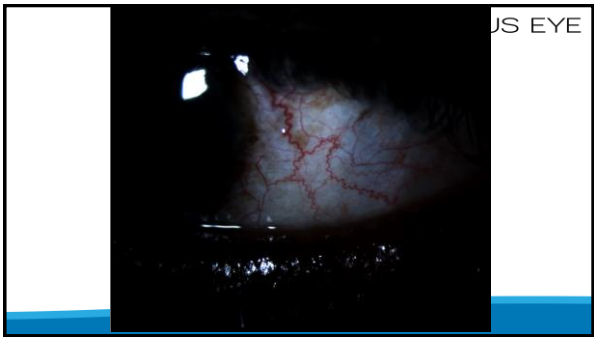
- Hemodynamic
  - High flow vs low flow
- Angiographic
  - ICA vs meningeal branches
- Etiology
  - spontaneous vs traumatic











US EYE

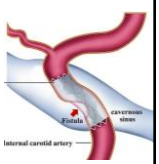
CAROTID CAVERNOUS SINUS FISTULA

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

US EYE

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



US EYE

Rule: Beware the Chronic Red Eye

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

US EYE

Non-ischemic CN VI

- 68 YOM:
- Pt develops isolated CN 6 Palsy OS
- Med hx: treated hypertension; rare salivary gland *cancer* treated 2 years previously.
- Consult on possible ischemic event
- Recommend imaging- pt had malignancy in cavernous sinus
- Outcome

US EYE

CN VI Demographic Groups

- Older adults (usually not bad)
  - Vascular disease common- resolves-3mos
    - Consider GCA over 60 yrs
- Children (may be bad)
  - Presumed viral illness, trauma, malignancy (50%)
- Young adults (usually bad)
  - Vascular disease (4%) and idiopathic (13%) uncommon
  - Usually complicated CN VI palsy (hemiparesis, Horner syndrome, facial paresis)
    - Cerebrovascular accidents involving the pons, aneurysm (typically within the cavernous sinus) or neoplasm (33%-cavernous sinus, pons), MS (24%).

US EYE

NEVER DIAGNOSE IDIOPATHIC (OR ISCHEMIC) ANYTHING IN A PATIENT WITH A HISTORY OF CANCER

US EYE

WHEN LOOKING FOR MASS CAUSE OF CN VI PALSY, THE BASE OF THE PONS AND CAVERNOUS SINUS ARE TWO COMMON HIDING SPOTS