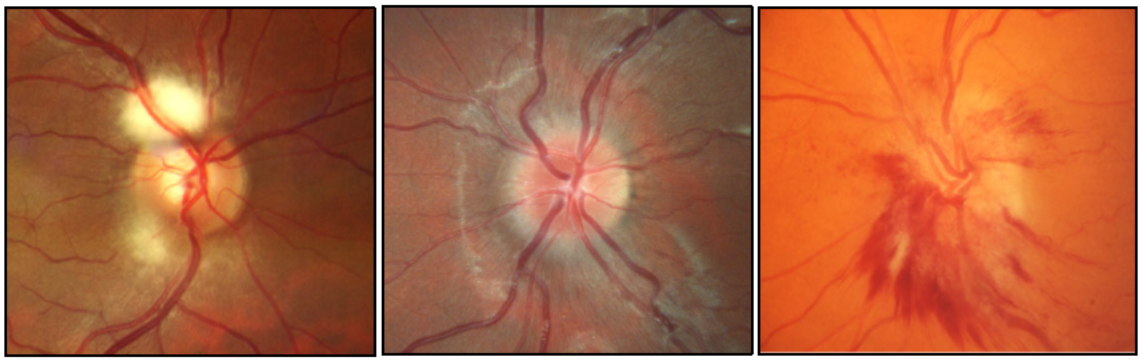


DIAGNOSE THIS DISC EDEMA!

Carolyn Majcher, OD, FAAO, FORS
Oklahoma College of Optometry

1

Contact:
- majcher@nsuok.edu
- 918-444-4155

Disclosures:
- Paid consultant/speaker for:
  - Carl Zeiss Meditec
  - Regeneron Pharmaceuticals
  - Iveric Bio (Astellas)
  - Optomed
  - Apellis Pharmaceuticals
- Paid advisory board member for LENZ Therapeutics, Notal Vision, Topcon, Tarsus, Genentech

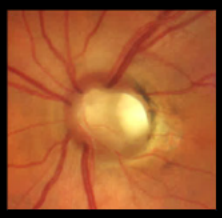
All relevant relationships have been mitigated

2

OPTIC NEUROPATHY

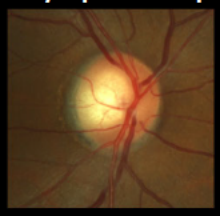
Can break down neuropathies into 4 diff diagnostic grps based on disc appearance:

Anomalous



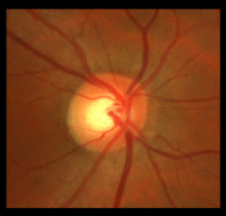
- ON hypoplasia
- ON coloboma
- Morning Glory
- Optic disc pit
- Tilted discs
- Myelinated NFL
- Megalopapilla
- Posterior staphyloma

Pale/Optic Atrophy



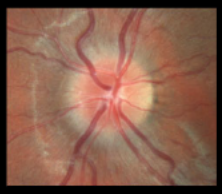
- Compressive optic neuropathy (may also be swollen or normal)
- Nutritional/toxic optic neuropathy (may also be swollen)
- Dominant/recessive optic atrophy
- Assoc with neurodegenerative disease
- Assoc with retinal dystrophies

Normal



- Optic neuritis (1/3rd swollen)
- Posterior ischemic optic neuropathy
- Traumatic optic neuropathy (acute mild swelling possible)

Swollen



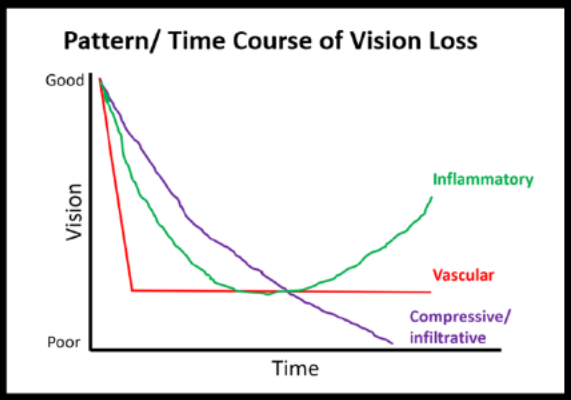
- Anterior ischemic optic neuropathy
- Papilledema
- Malignant HTN
- Diabetic papillopathy
- Inflammatory/infectious optic neuropathies (may also be pale)
- Radiation optic neuropathy (may also be normal)
- Disc drusen (pseudoedema)
- Leber's Hereditary Optic Neuropathy (pseudoedema)

* DISCLAIMER: there is considerable overlap among these 4 groups

3

NEURO-OPHTHALMIC EXAM

- **History:** Used as a guide along with age, gender, underlying illnesses, and disease risk factors shape the rest of the exam to confirm or eliminate each differential
- 3 most crucial qualifiers in determining etiology of VL
  1) Laterality
  2) Time course
  3) Associated symptoms



- Trauma/head injury?
- Previous surgeries/blood loss?
- Alcohol/drug use?
- Nutrition?
- Family history?
- Previous neuroimaging?
- Occupation?

CHECK VITALS (ESP BP)!!

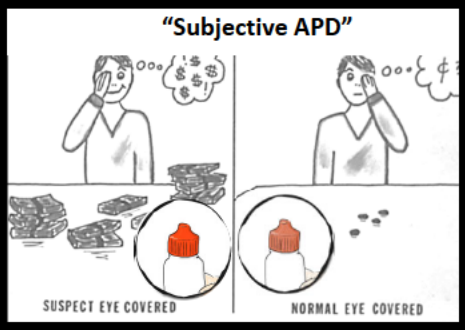
4

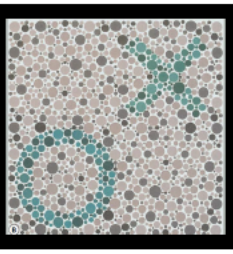
NEURO-OPHTHALMIC EXAM

Abnormal Tests in Neuropathy (Afferent Visual Function)

- BCVA (PHNI if neuropathy)
- RAPD
- Red desaturation & brightness comparison
- Color vision
- Contrast sensitivity
- Visual field
- Optic nerve head examination

Diagnostic dilemma: Is VL due to refractive error, amblyopia, media opacity, retinal lesion, ON or chiasmal disease???





5

Clinical Distinction Between Optic Neuropathy vs Maculopathy

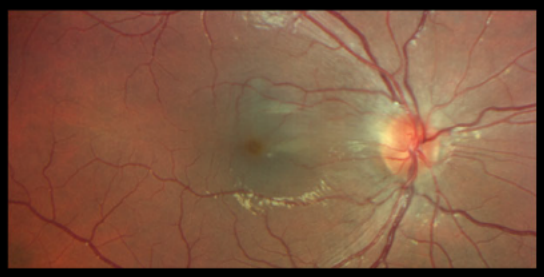
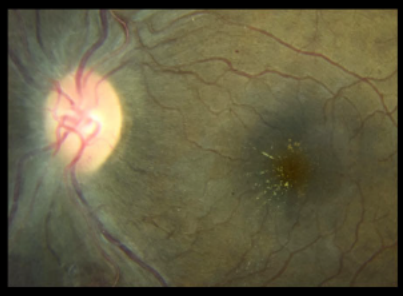
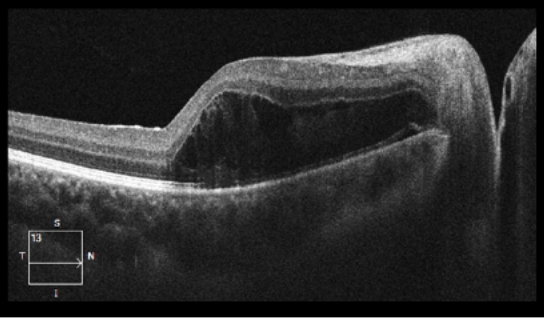
Symptom, Sign, or Test	Optic Neuropathy	Maculopathy
Metamorphopsia	Rare	Common
Darkening of vision	Common	Rare
Photopsia	Rare	Common
Pain	Common in optic neuritis, rare in other neuropathies	Rare
Dyschromatopsia	Severe	Mild
RAPD	Common	Rare (unless severe & asymmetric)
ERG (esp mfERG)	Normal	Abnormal usually
VEP	Large latency delay	Small latency delay

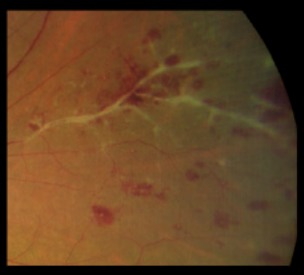
Diagnostic dilemma: Is VL due to refractive error, amblyopia, media opacity, retinal lesion, ON or chiasmal disease???

6

NEURO-OPHTHALMIC EXAM

Don’t forget abut the macula and the periphery!!

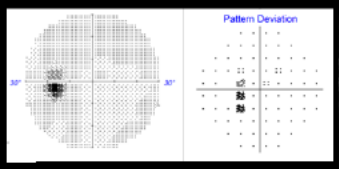


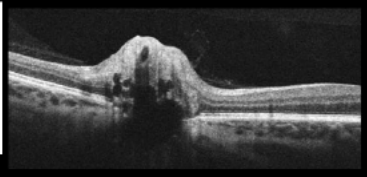
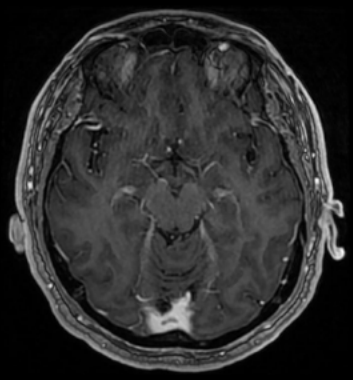
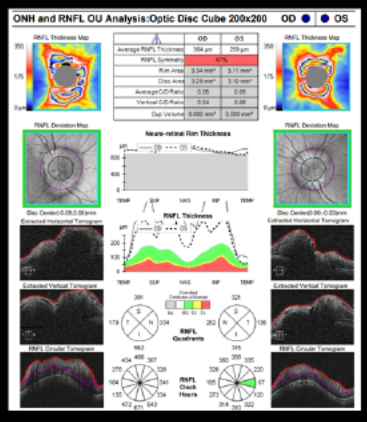

7

NEURO-OPHTHALMIC EXAM

Ancillary Tests:

- Photography- color/FAF
- OCT (ONH/NFL & Macular/GCC)
- Ultrasound (B-scan/doppler)
- Neuroimaging
- Labs/TAB
- IV fluorescein angiography
- Potential acuity meter
- VEP/ERG
- Photostress recovery test



8

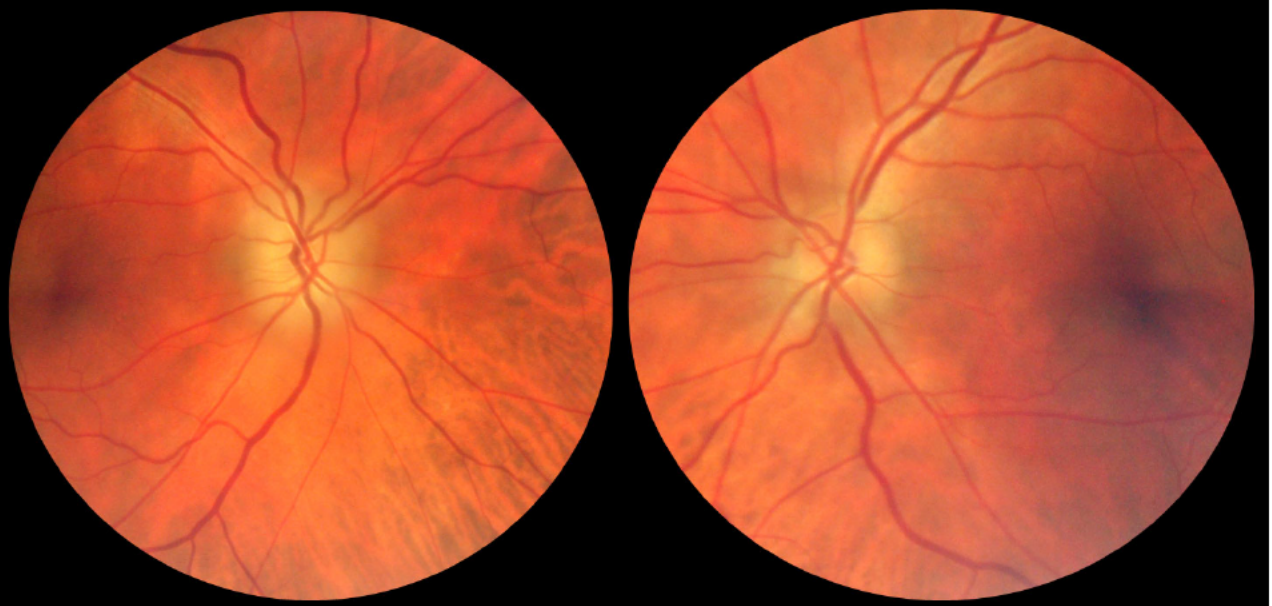
DOUBLE TROUBLE

58yo Native American male – ER visit, sudden painless VL OD x 12 hrs (midnight last night while watching TV). No improvement since onset.

- Oc Hx: Unremarkable
- Med Hx:
  - **HTN, A fib, heart disease, hyperlipidemia**, gout, psoriatic arthropathy,
- Medications: Eliquis, Diltiazem, iron, Allopurinol, Buspirone, Carvedilol, Digoxin, Colchicine, Metoprolol, Vitamin D3, Entresto, Fenofibrate, Gabapentin, Lasix
- VAs @dist:
  - **OD HM PHNI**
  - OS $20/25^{+2}$ PHNI
- Entrance testing: Generalized constriction of VF OD, **3-4+ APD OD**
- SLE: trace NS cat OU
- IOPs: OD 11/ OS 15 mmHg
- BP: 115/62

9

DOUBLE TROUBLE



Any additional history questions you want to ask now?

10

Systemic symptoms/signs of GCA

- **Headache/scalp tenderness 56%**
- **Anorexia/weight loss 31-52%**
- **Polymyalgia rheumatica (PMR) 22-50%**
- **Jaw claudication 53%**
- **Malaise/weakness 22-38%**
- **Myalgia 29%**
- **Temporal artery tenderness/pain, prominent, pulseless 20%**
- **Low grade fever 11-26%**
- **Scalp tenderness/necrosis 11-18%**
- Neck pain 16%
- Anemia 13%
- Tongue pain/sores or necrosis

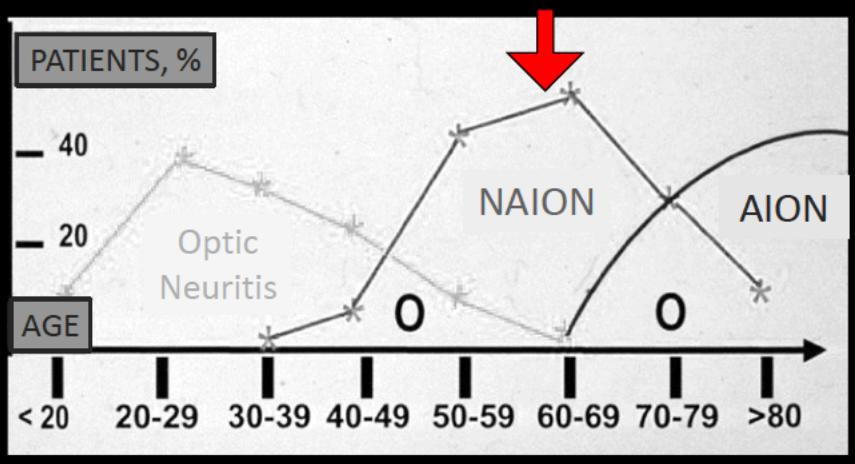
~20% OF GCA CASES WITH VL ARE OCCULT (OCULAR MANIFESTATIONS WITH NO SYSTEMIC SIGNS/SYMPTOMS)!!!!

Hayreh SS. Occult GCA: ocular manifestations. AJO 1998

11

DOUBLE TROUBLE

Differential Diagnoses for Unilateral Disc edema
Overlapping Clinical Profiles

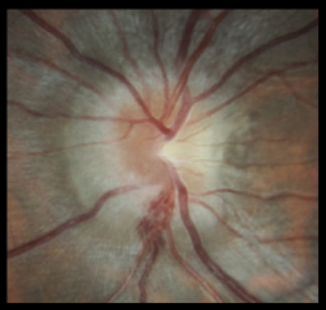


12

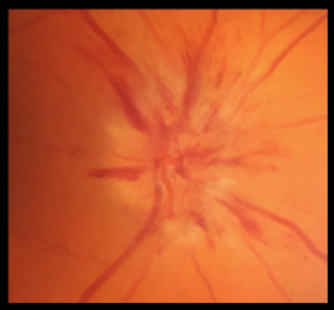
OPTIC DISC SWELLING DIFF DX

- **Diagnostic dilemma**: Papilledema, anterior ischemic optic neuropathy, optic neuritis, pseudopapilledema, etc.?

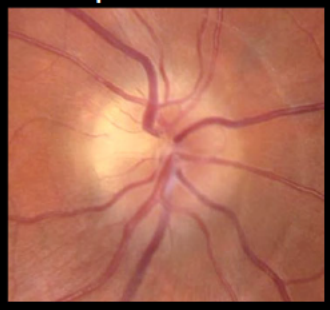
Papilledema



NA-AION



Optic Neuritis



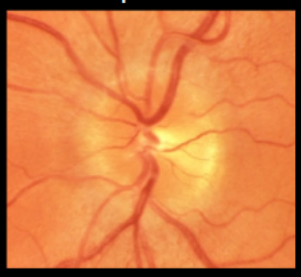
DISC APPEARANCE AND PATTERNS OF VF LOSS MAY BE SIMILAR!!

13

DOUBLE TROUBLE

Differential Diagnoses for Unilateral Disc edema

Optic Neuritis

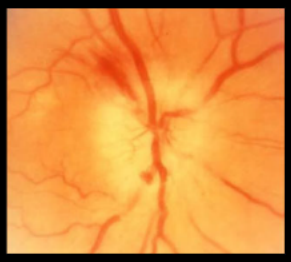


Pain on eye movt 92%
Presenting VA variable
Central/diffuse VF
2/3rds Retrobulbar
Disc hemorrhages rare
77% female, 85% white
MRI ON sheath enhancement, WMLs

NA-AION

Painless 90%
20/60 or better
VL loss can progress for days
Altitudinal VF loss
Disc edema often **sectoral**
Disc is **hyperemic** "luxury perfusion"
Fellow eye disc at risk
Associated with vasculopathic risk factors (HTN etc), sleep apnea, Viagra & Amiodarone

A-AION

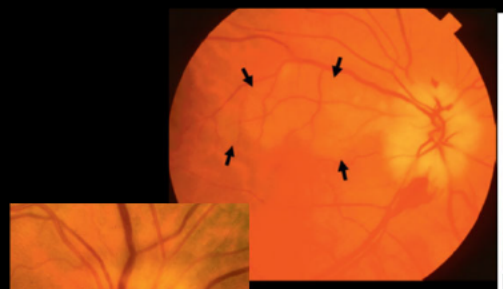


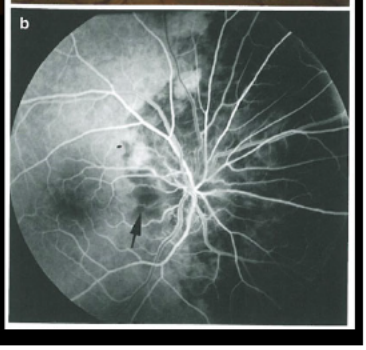
Painful VL 79%
CF or worse
Nadir in minutes
Preceding TMVL possible
Disc edema is diffuse & PALLID
30% simultaneous bilat involvement
80% have systemic GCA symptoms

14

DOUBLE TROUBLE

Differential Diagnoses for Unilateral Disc edema





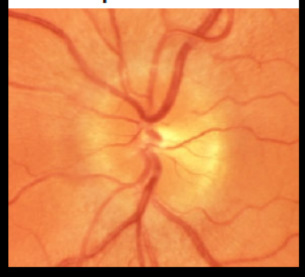
CONCURRENT POST SEG ISCHEMIA (CWS, CILIORETINAL ARTERY OCCLUSION, CHOROIDAL OR INNER RETINAL INFARCTION = A-AION UNTIL PROVEN OTHERWISE!!

15

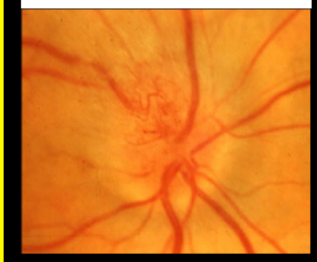
DOUBLE TROUBLE

Differential Diagnoses for Unilateral Disc Edema

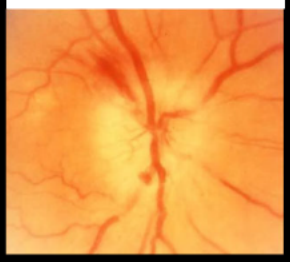
Optic Neuritis



NA-AION



A-AION



What systemic testing is needed to differentiate between these diagnoses?

16

DOUBLE TROUBLE

Same day workup:

- Labs:
  - **ESR: 79 mm/hr (lab norm 2-10)**
  - **CRP: 49 mg/L (lab norm 0-10)**
  - **Platelets: 423 x 10^3/mcL (lab norm 150-450)**
  - PT/INR: high
  - Glucose 99 mg/dL
  - FTA-abs, RPR, ANA, ACE, RMSF, tick panel, Bartonella – all WNLs
- CT head w/o contrast: Unremarkable
- Carotid Doppler US: Bilateral ICA plaque but <50% stenosis (not hemodynamically significant)

ASSESSMENT: Very high suspicion for A-AION

PLAN:

- IV methyl Pred 250mg q6hrs for 12 doses followed by 80-100mg PO prednisone daily
- Scheduled TAB rt side in 2 days
- Refer to neuro-ophthalmology

17

GCA Work-up

- **ESR**
  - A nonspecific marker of inflammation and/or neoplastic disease.
  - **High end of normal for men = age/2**
  - **High end of normal for females = age +10/2**
  - **Hayreh >46 (mean of 85 in cases of + TAB)**
- **CRP**
  - An acute phase protein produced by hepatocytes
  - **Normal is <2.54 mg/dL**
  - **Hayreh >2.45 (mean of 6.6 in cases of + TAB)**
- **CBC with diff and platelet count**
  - Thrombocytosis = **↑ platelet count >400 x10^3/L**
- **TAB**
  - Most specific test for GCA (gold standard)
  - Test ipsilateral side of ocular involvement
  - Still perform if ESR/CRP are norm if suspicion is high
- **Temporal artery Doppler US**

Giant Cell Arteritis:
Validity and Reliability
of Various Diagnostic Criteria

SOHAN SINGH HAYREH, MD, PHD, DSC, PATRICIA A. PODHAJSKY, BSN, REMA RAMAN, MSC, AND BRIDGET ZIMMERMAN, PHD

The odds of a positive TAB was:
2.0 times greater with ESR of 47 - 107 mm/hour
3.2 times greater with CRP > 2.45 mg/dl

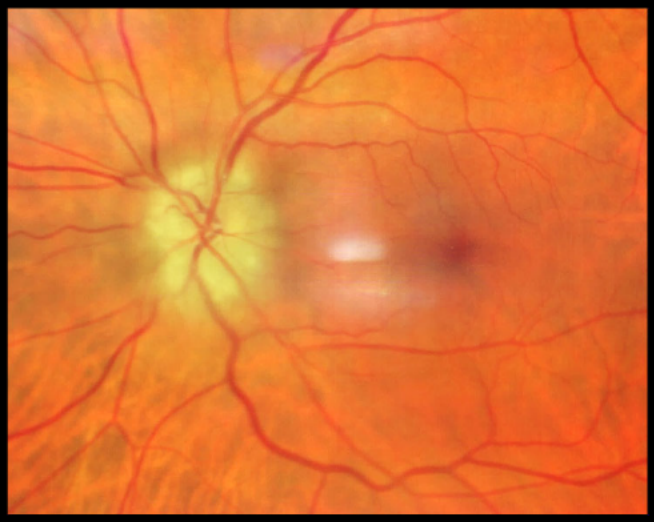
"Elevated ESR and CRP = 97% specific for GCA"

18

DOUBLE TROUBLE

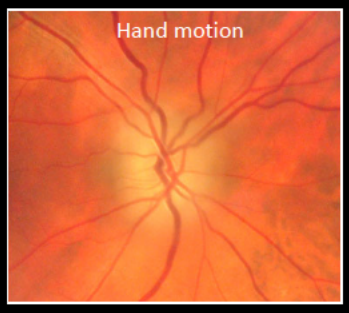
3 Day Optometry FU

- Pt on last day of IV methyl pred
- Had TAB yesterday - inadequate sample for evaluation of GCA (repeat TAB left side with neuro-ophthalmologist in 1 week)
- States his vision OS has been decreased since coming out of recovery from TAB
- VAs @dist:
  - OD HM PHNI
  - **OS 20/800 PHNI**

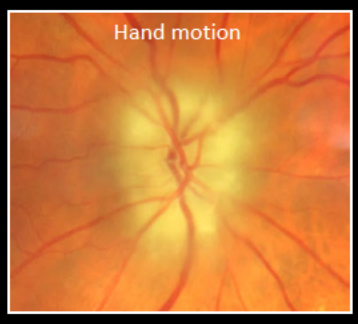


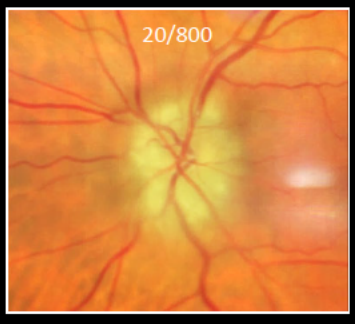
19

BASELINE

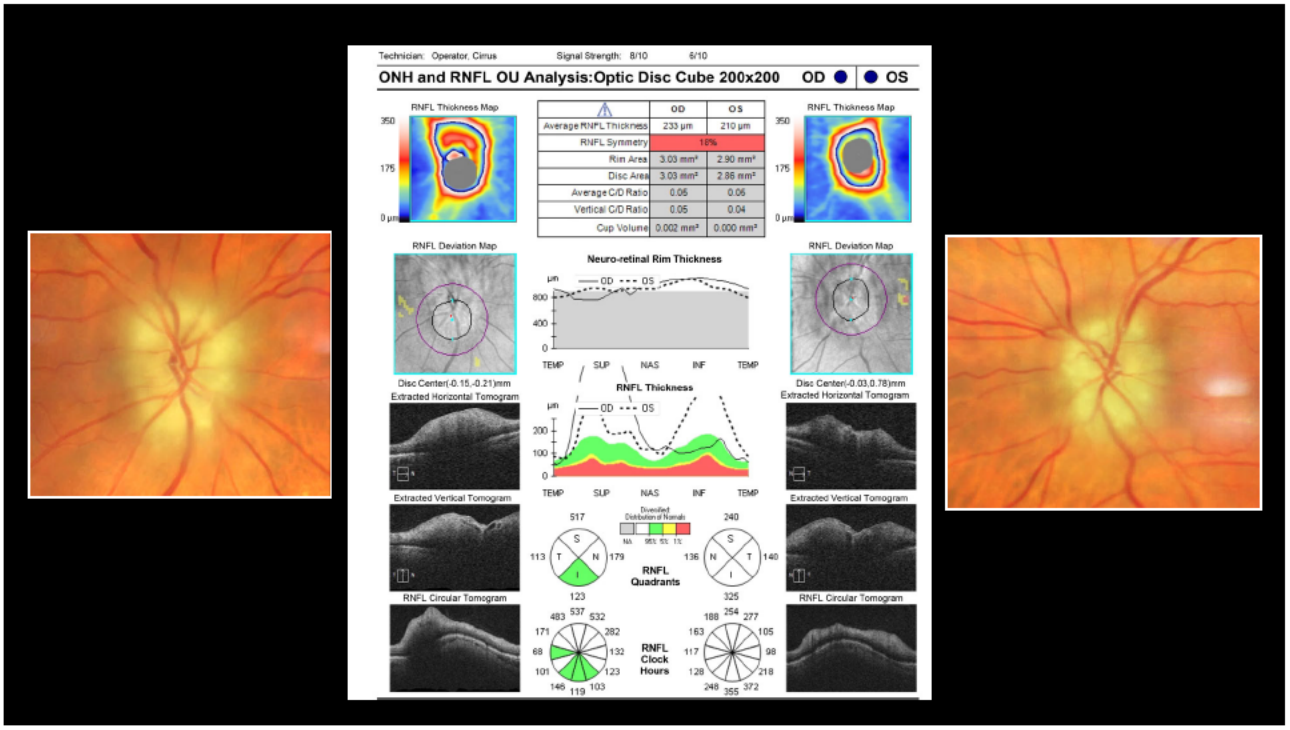


3 DAYS LATER....





20



21

DOUBLE TROUBLE

ASSESSMENT:

- **Still Highly Suspect A-AION**
- **Now bilateral involvement** despite emergently starting IV steroids

X 3!!

PLAN:

- **Repeat TAB left side** with neuro-ophthalmologist in 1 week
- **Finish out course of steroids** (IV methyl Pred 250mg q6hrs for 12 doses followed by 80-100mg PO prednisone daily)

Left side TAB & another rt side biopsy- both negative for GCA

Neuro-ophthalmology still highly suspects A-AION but cannot rule out sequential NA-ION due to cardiovascular risk factors esp unstable A-fib

22

DOUBLE TROUBLE

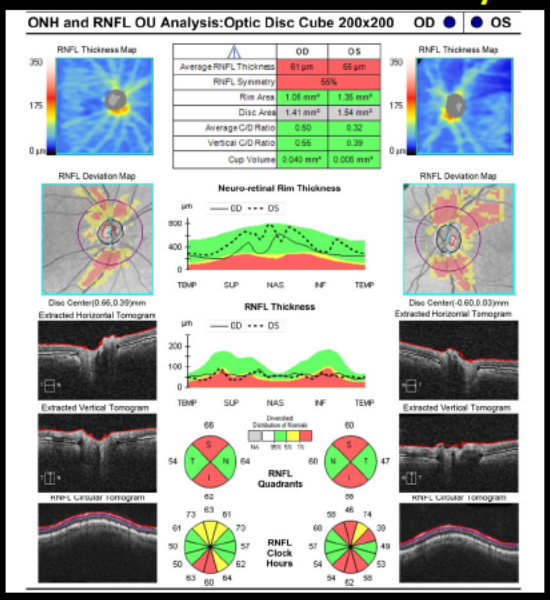
1 year later……

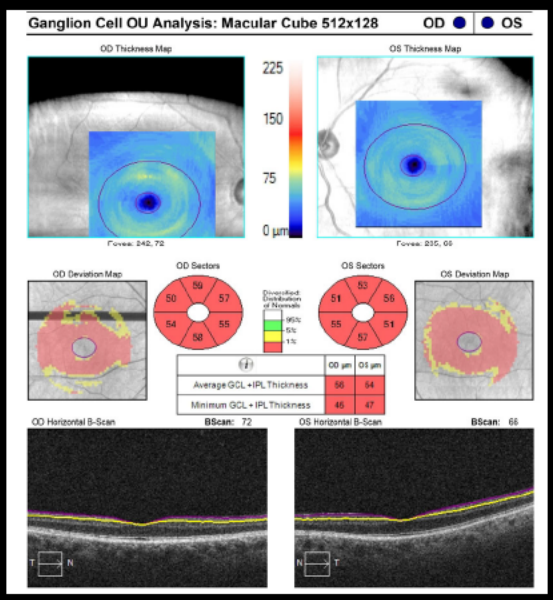
OD: 20/50 **OS: 20/100**

23

DOUBLE TROUBLE

1 year later……



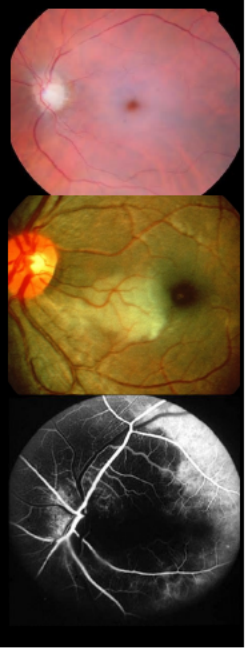


24

OCULAR MANIFESTATIONS OF GCA

30-50% OF PTS WITH GCA PRESENT WITH OCULAR INVOLVEMENT!!

- **Arteritic anterior ischemic optic neuropathy (A-AION, 80-90%)**
  - **30% bilateral at presentation**
  - **75% will become bilateral if left untreated**
    - **Can occur within days, ~50% within 1 week**
- **CRAO 5%**
- **Cilioretinal artery occlusion 22%**
- BRAO 5%??
- CWSs (present in approx 30% with acute vision loss)
- IVFA evidence of SPCA occlusion (choroidal or disc vascular insufficiency/ischemia) ~100%



SUSPECT GCA IN ANY ACUTE ONSET NEUROLOGIC DEFICIT IN A PT OVER 50 YEARS OF AGE!!!

Hayreh SS et al. Ocular manifestations of giant cell arteritis. Am J Ophthalmol. 1998 Apr;125(4):509-20.

25

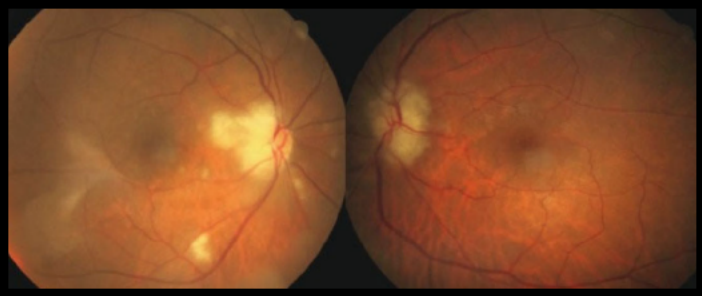
GCA TREATMENT

HIT HARD AND HIT FAST TO PREVENT BILATERAL BLINDNESS!

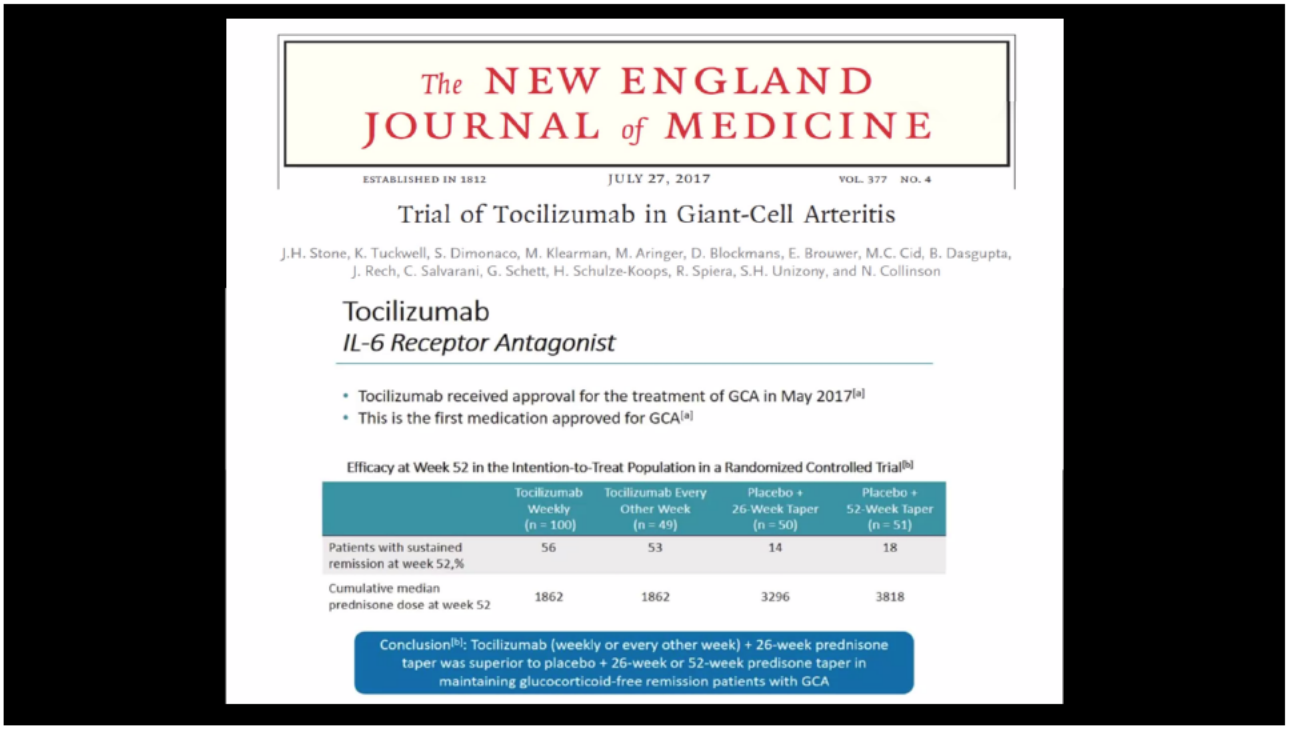
- When GCA is highly suspected do not wait for TAB results to begin treatment
- IV methyl pred 1g per day x 3 days (250 mg q6h for 12 doses), then switch to prednisone 80 to 100 mg PO qd (or 1 mg/kg per day) day x 1 mo then taped over 6–12 months while confirming normal ESR and no systemic symptoms
- Vitamin D 2,000 units daily + calcium
- Low dose ASA

Rates of vision improvement:

- **58% improved if steroid administered with 24hr**
- 6% improved if administered after 24hrs



26



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 JULY 27, 2017 VOL. 377 NO. 4

Trial of Tocilizumab in Giant-Cell Arteritis

J.H. Stone, K. Tuckwell, S. Dimonaco, M. Klearman, M. Aringer, D. Blockmans, E. Brouwer, M.C. Cid, B. Dasgupta, J. Rech, C. Salvarani, G. Schett, H. Schulze-Koops, R. Spiera, S.H. Unizony, and N. Collinson

Tocilizumab

IL-6 Receptor Antagonist

- Tocilizumab received approval for the treatment of GCA in May 2017[a]
- This is the first medication approved for GCA[a]

Efficacy at Week 52 in the Intention-to-Treat Population in a Randomized Controlled Trial[b]

	Tocilizumab Weekly (n = 100)	Tocilizumab Every Other Week (n = 49)	Placebo + 26-Week Taper (n = 50)	Placebo + 52-Week Taper (n = 51)
Patients with sustained remission at week 52,%	56	53	14	18
Cumulative median prednisone dose at week 52	1862	1862	3296	3818

Conclusion[b]: Tocilizumab (weekly or every other week) + 26-week prednisone taper was superior to placebo + 26-week or 52-week predisone taper in maintaining glucocorticoid-free remission patients with GCA

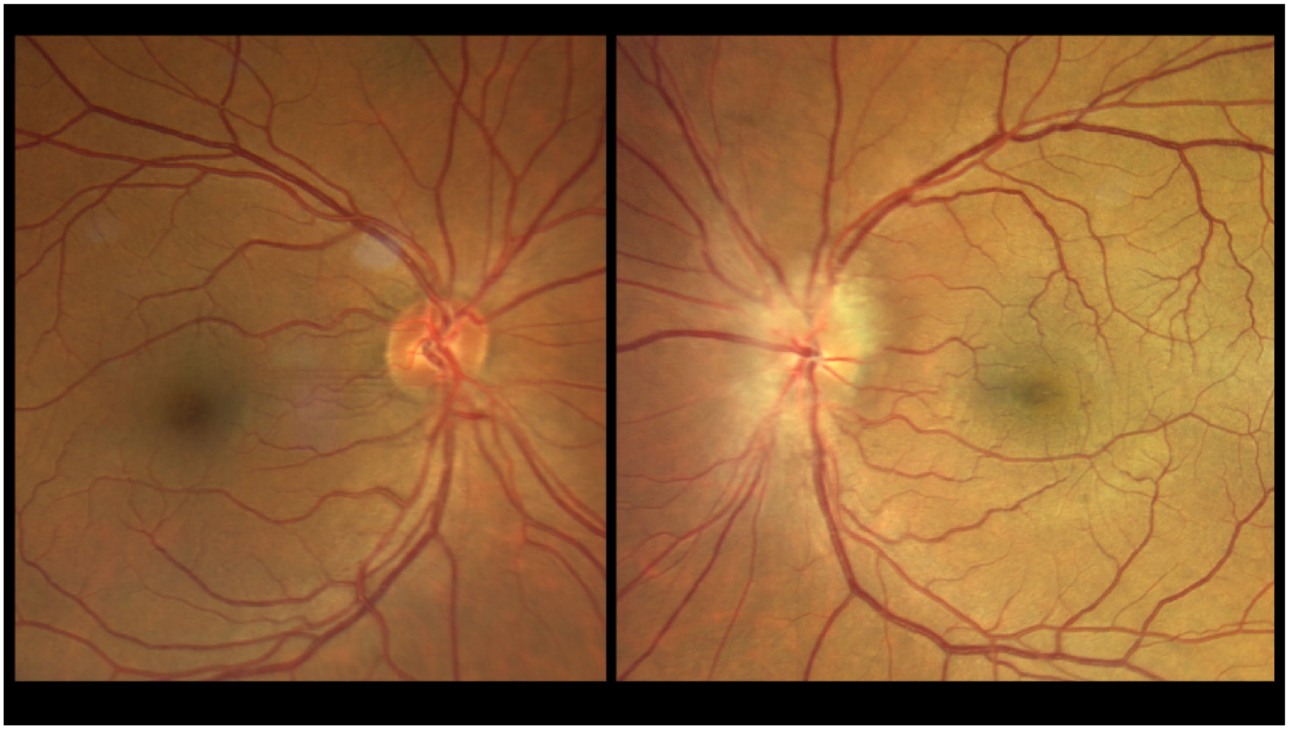
27

THE CASE OF THE CURIOUSLY SWOLLEN DISCS

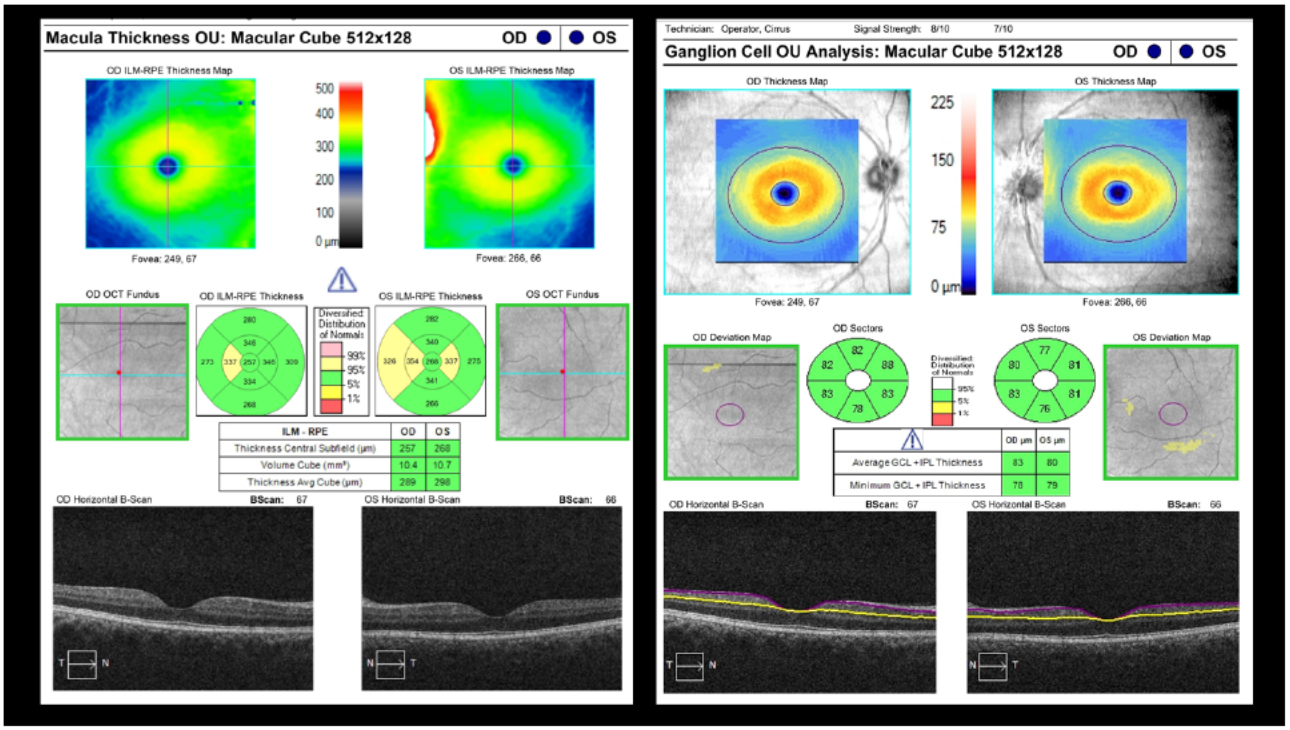
45yo male – Walk-in NP complaining of missing vision inf field OS x 1 week, first noticed upon waking, (-) pain

- Oc Hx: approx. -1D to 2D myope, LEE 7mo ago
- Med Hx: **DM type 2 (last a1c 9.6%), HTN, hypercholesterolemia, hx of alcoholism**
- Meds: ASA 81mg, simvastatin, **sildenafil**, lisinopril, insulin, metformin
- VAs @dist:
  - OD 20/20
  - **OS 20/20**
- Entrance testing: **Inf nasal constriction of VF OS, trace APD OS**
- SLE: WNLs OU
- IOPs: 17 mmHg OD/OS
- **BP: 144/92**

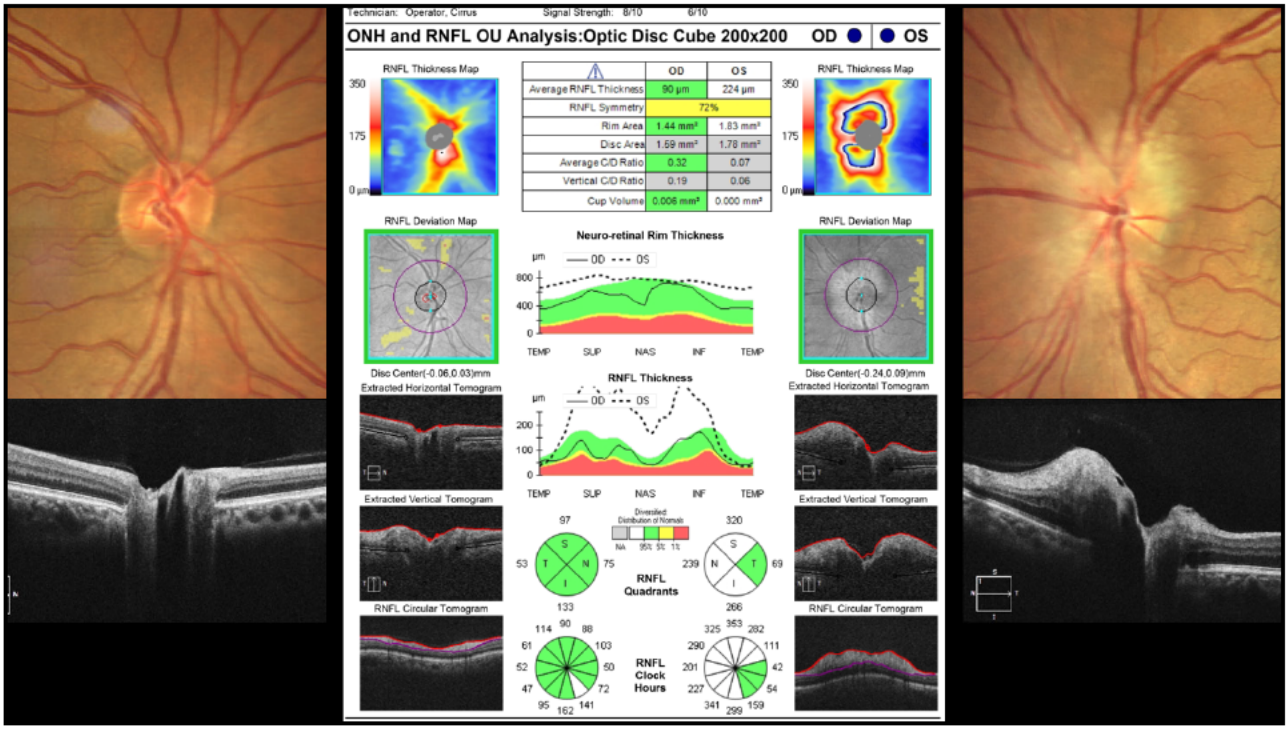
28



29

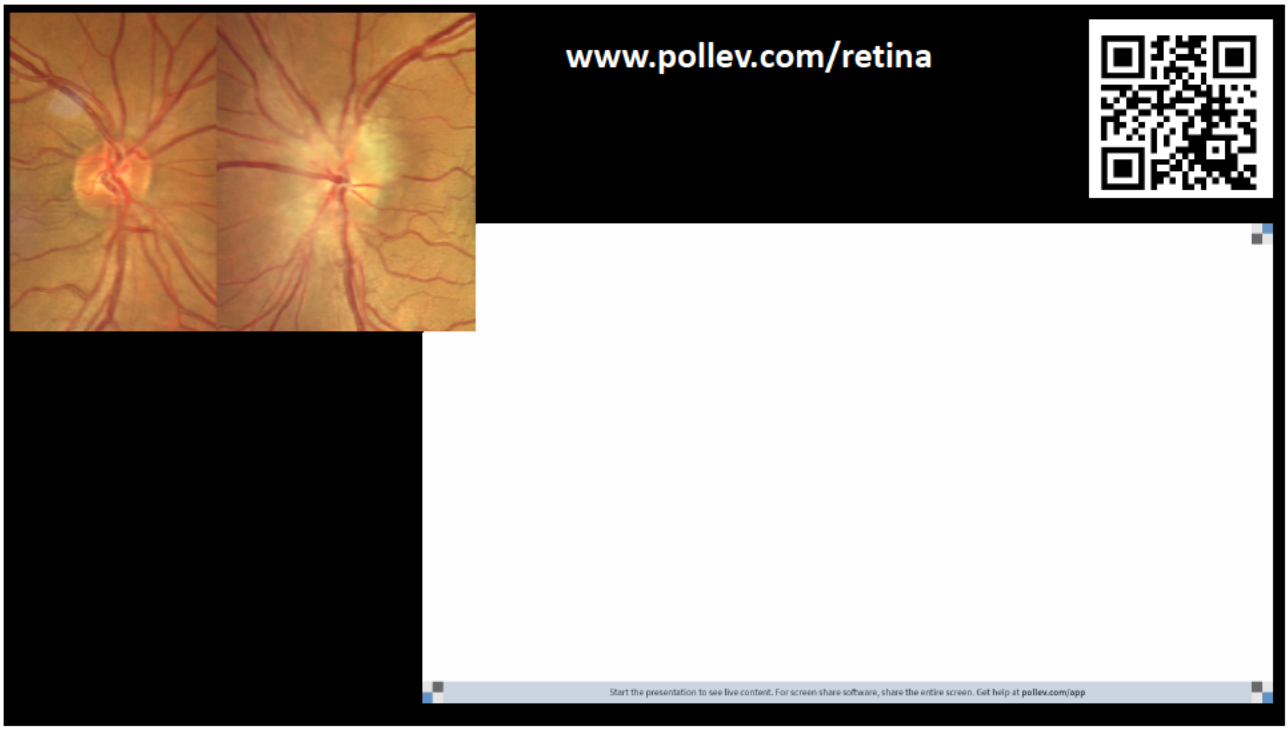

Macula Thickness OU: Macular Cube 512x128 OD OS
OD ILM-RPE Thickness Map
OS ILM-RPE Thickness Map
500
400
300
200
100
0 µm
Fovea: 249, 67
Fovea: 266, 66
OD OCT Fundus
OD ILM-RPE Thickness
Diversified Distribution of Normals
99%
95%
5%
1%
OS ILM-RPE Thickness
OS OCT Fundus
ILM - RPE
OD
OS
Thickness Central Subfield (µm)
257
268
Volume Cube (mm³)
10.4
10.7
Thickness Avg Cube (µm)
289
298
OD Horizontal B-Scan
BScan: 67
OS Horizontal B-Scan
BScan: 66
Technician: Operator, Cirrus
Signal Strength: 8/10 7/10
Ganglion Cell OU Analysis: Macular Cube 512x128 OD OS
OD Thickness Map
OS Thickness Map
225
150
75
0 µm
Fovea: 249, 67
Fovea: 266, 66
OD Deviation Map
OD Sectors
82
88
83
78
OS Sectors
77
81
80
81
83
76
OS Deviation Map
OD µm
OS µm
Average GCL + IPL Thickness
83
80
Minimum GCL + IPL Thickness
78
79
OD Horizontal B-Scan
BScan: 67
OS Horizontal B-Scan
BScan: 66

30

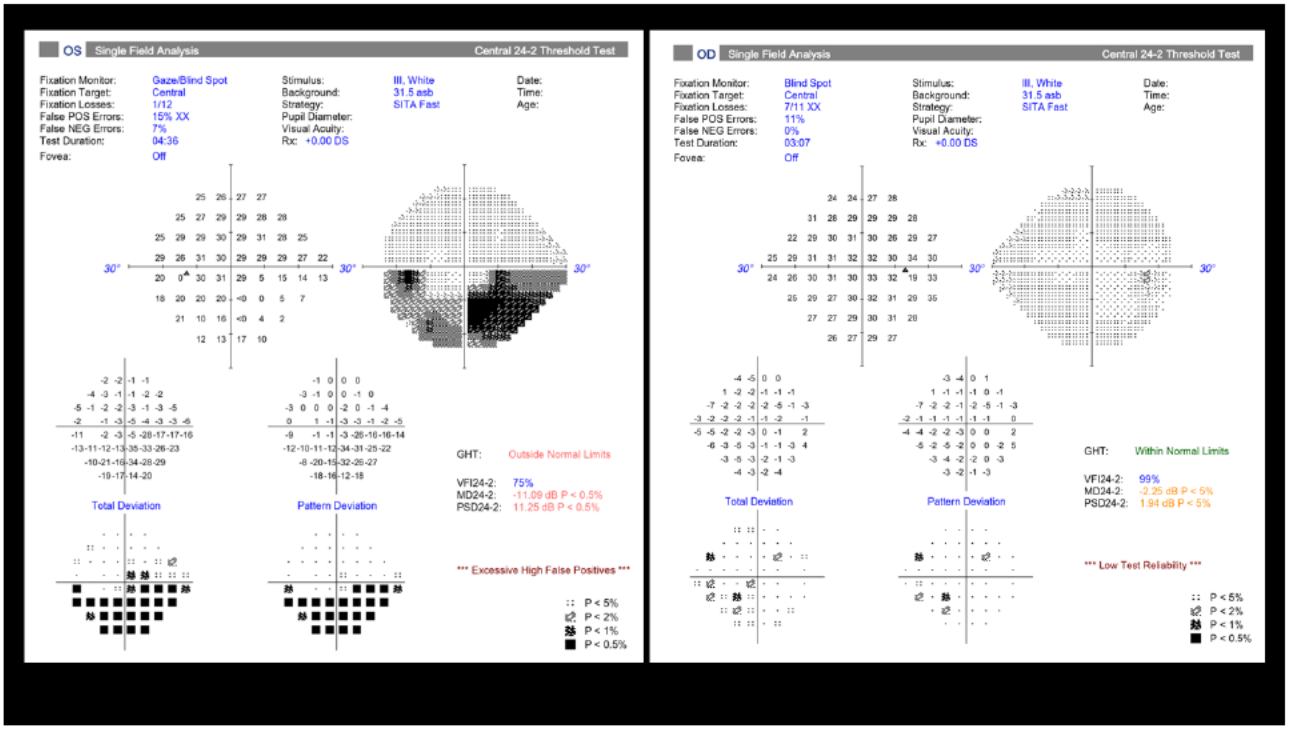


	OD	OS
Average RNFL Thickness	90 µm	224 µm
RNFL Symmetry	72%	
Rim Area	1.44 mm²	1.83 mm²
Disc Area	1.59 mm²	1.78 mm²
Average C/D Ratio	0.32	0.07
Vertical C/D Ratio	0.19	0.06
Cup Volume	0.006 mm³	0.000 mm²

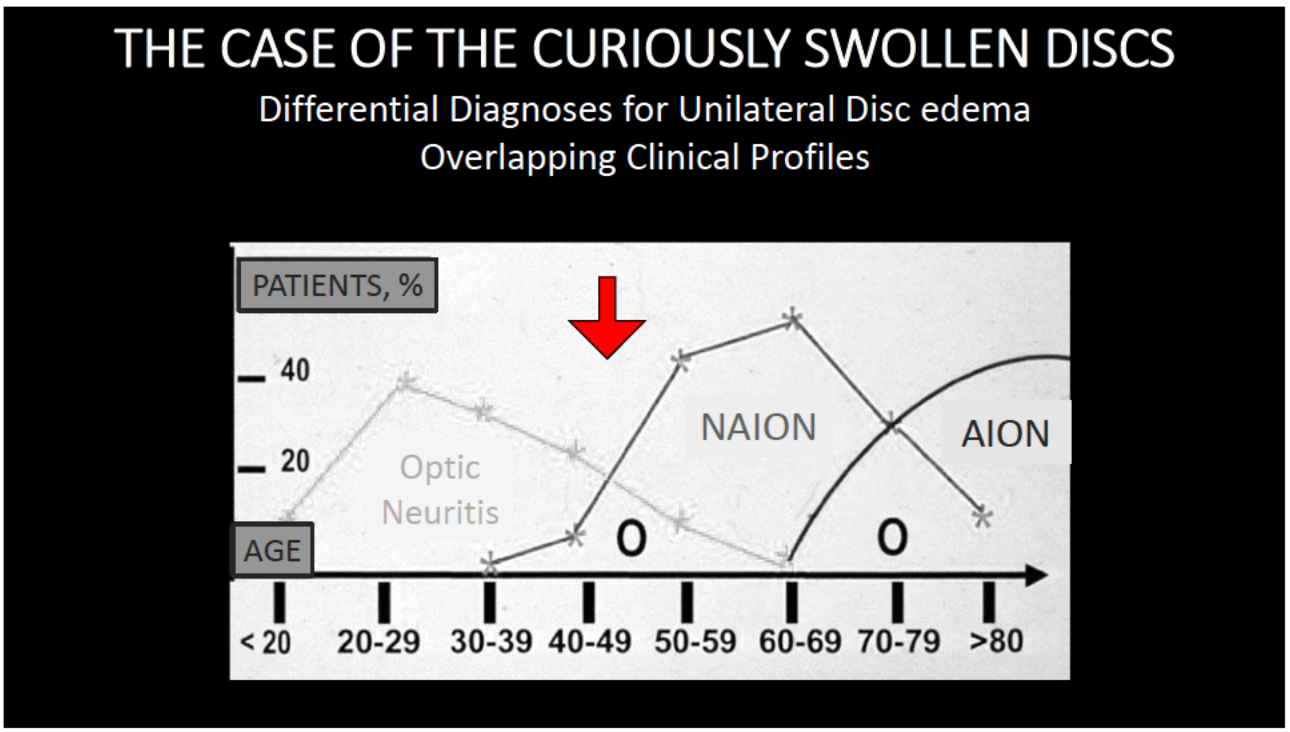
31



32


OS Single Field Analysis Central 24-2 Threshold Test
Fixation Monitor: Gaze/Blind Spot
Fixation Target: Central
Fixation Losses: 1/12
False POS Errors: 15% XX
False NEG Errors: 7%
Test Duration: 04:36
Fovea: Off
Stimulus: III, White
Background: 31.5 asb
Strategy: SITA Fast
Date:
Time:
Age:
Total Deviation
Pattern Deviation
GHT: Outside Normal Limits
VFI24-2: 75%
MD24-2: -11.09 dB P < 0.5%
PSD24-2: 11.25 dB P < 0.5%
*** Excessive High False Positives ***
P < 5%
P < 2%
P < 1%
P < 0.5%
OD Single Field Analysis Central 24-2 Threshold Test
Fixation Monitor: Blind Spot
Fixation Target: Central
Fixation Losses: 7/11 XX
False POS Errors: 11%
False NEG Errors: 0%
Test Duration: 03:07
Fovea: Off
Stimulus: III, White
Background: 31.5 asb
Strategy: SITA Fast
Date:
Time:
Age:
Total Deviation
Pattern Deviation
GHT: Within Normal Limits
VFI24-2: 99%
MD24-2: -2.25 dB P < 5%
PSD24-2: 1.94 dB P < 5%
*** Low Test Reliability ***
P < 5%
P < 2%
P < 1%
P < 0.5%

33

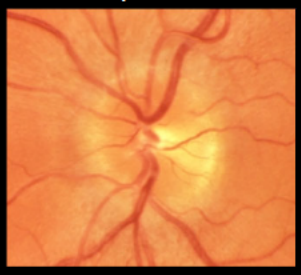

THE CASE OF THE CURIOUSLY SWOLLEN DISCS
Differential Diagnoses for Unilateral Disc edema
Overlapping Clinical Profiles
PATIENTS, %
40
20
AGE
Optic Neuritis
NAION
AION
< 20
20-29
30-39
40-49
50-59
60-69
70-79
>80

34

THE CASE OF THE CURIOUSLY SWOLLEN DISCS

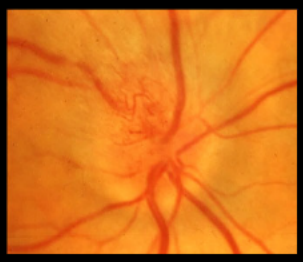
Differential Diagnoses for Unilateral Disc edema

Optic Neuritis



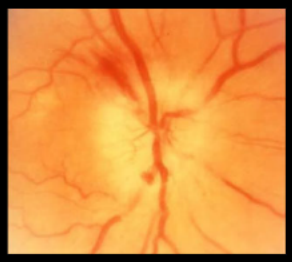
Pain on eye movt 92%
Presenting VA variable
Central/diffuse VF
2/3rds Retrobulbar
Disc hemorrhages rare
77% female, 85% white
MRI ON sheath enhancement, WMLs

NA-AION



Painless 90%
20/60 or better
VL loss can progress for days
Altitudinal VF loss
Disc edema often **sectoral**
Disc is **hyperemic** "luxury perfusion"
Fellow eye disc at risk
Associated with vasculopathic risk factors (HTN etc), sleep apnea, Viagra & Amiodarone

A-AION



Painful VL 79%
CF or worse
Nadir in minutes
Preceding TMVL possible
Disc edema is diffuse & PALLID
30% simultaneous bilat involvement
80% have systemic GCA symptoms

35

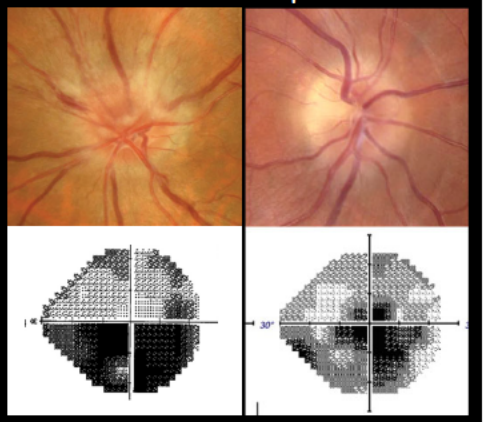
NAION vs Optic Neuritis (ON)

- Age? >50 vs <40yo
- **Pain? 10% vs 92% esp on eye movt**
- Optic disc edema? 100% vs 35%
  - Altitudinal/sectoral swelling, arterial attenuation, disc surface capillary dilation aka "luxury perfusion" more common in NAION
- **Hemorrhage? 72% vs 6%**
- **MRI -contrast enhancement of the ON rare in NAION**
- VF defect? inf altitudinal vs central/diffuse (variable)
- IVFA- delayed disc filling in NAION?

NOT helpful: lab tests (not helpful to differentiate NAION vs ON but can rule out infectious/inflammatory etiologies), VA, gender

Optic neuritis diagnosis will always be a clinical one!

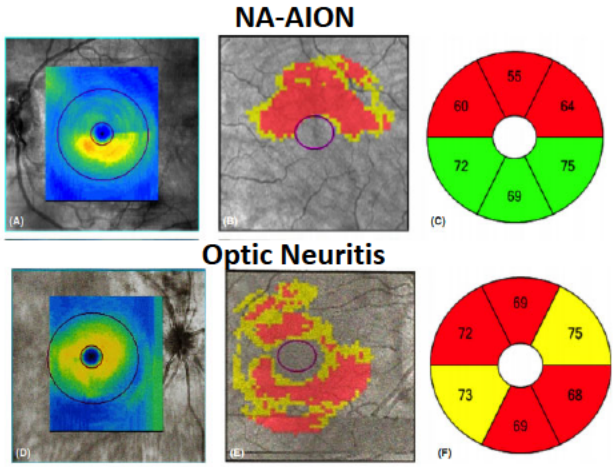
NAION | Optic Neuritis



36

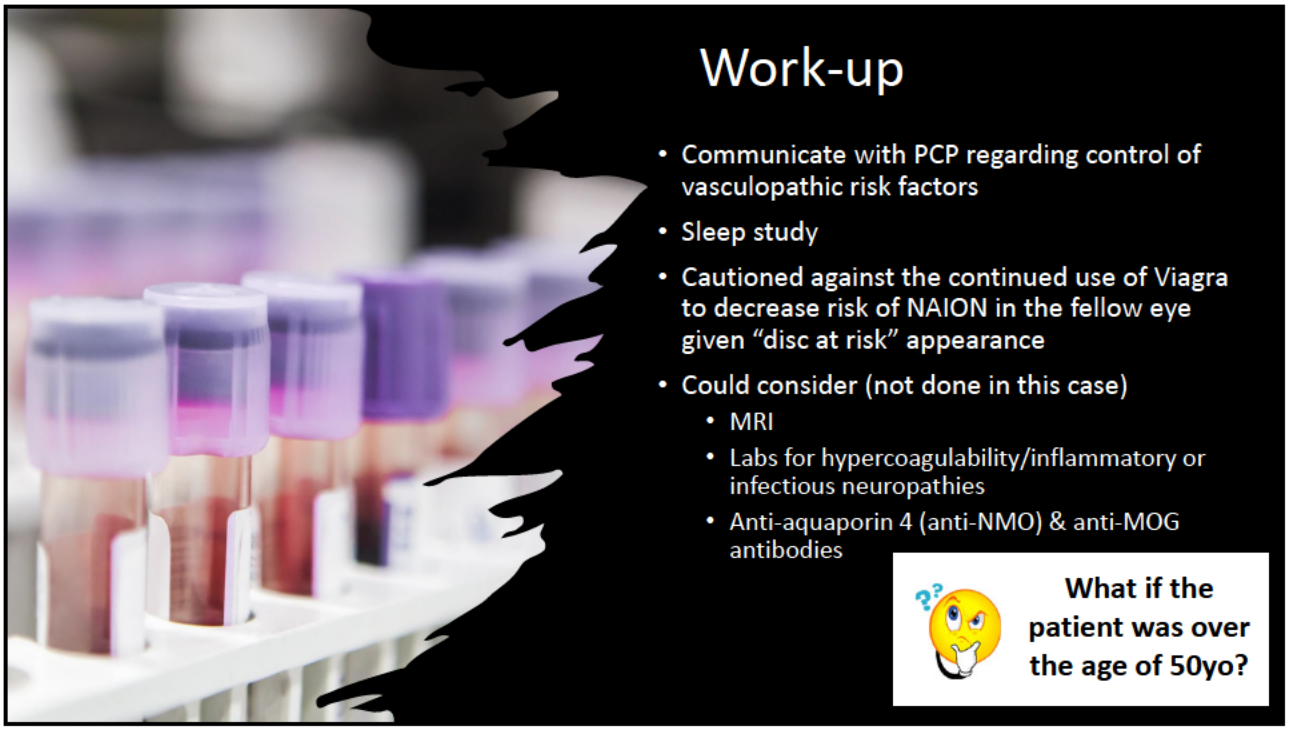
NAION vs Optic Neuritis (ON) – Late GCC Findings

- GCC Analysis: greater hemispheric difference in GCC thickness in NAION than optic neuritis



Erlich-Malona et al. Distinguishing ION from ON by ganglion cell analysis. Acta Ophthal. 2016.

37



Work-up

- Communicate with PCP regarding control of vasculopathic risk factors
- Sleep study
- Cautioned against the continued use of Viagra to decrease risk of NAION in the fellow eye given "disc at risk" appearance
- Could consider (not done in this case)
  - MRI
  - Labs for hypercoagulability/inflammatory or infectious neuropathies
  - Anti-aquaporin 4 (anti-NMO) & anti-MOG antibodies

What if the patient was over the age of 50yo?

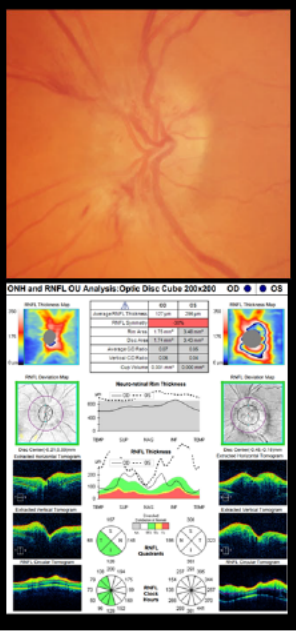
38

NON-ARTERITIC ISCHEMIC OPTIC NEUROPATHY (NAION)

- **Most common cause of unilateral disc edema/optic neuropathy in adults > 50yo**
- **Accounts for 90-95% of ant ION cases**

Pathology/Mechanism

- Not well understood, but likely some combo of:
  - **ON infarction from SPCA insufficiency** (exacerbated by microvascular arteriosclerotic disease or dysfunctional ONH vasc autoregulation?)
  - **"Compartment syndrome"** phenomenon, axonal crowding at the level of the LC (more vulnerable to ischemia)
  - **Embolic etiology is thought to be RARE** (unless concurrent retinal/choroidal infarction is present)

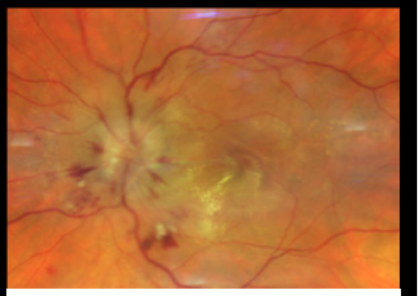


39

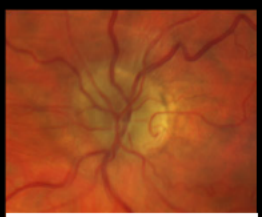
NON-ARTERITIC ISCHEMIC OPTIC NEUROPATHY (NAION)

Fairly well-established risk factors/associated conditions

- **HTN (47%, may also be assoc with acute HTN crisis)**
- **DM (24-34%%)**
- **Hypercholesterolemia (70%)**
- **Disc at risk (small crowded ONH or disc drusen)**
- **Sleep apnea**
- **Ischemic heart disease (21%, 11% previous MI)**
- **Major blood loss**
- **Perioperative spine and cardiac surgery (incidence ~0.1% in high-risk surgeries)**
- Hypercoagulable states (antiphospholipid antibodies, ↑homocysteine)
- Anemia
- Migraine (esp pts <50yo)



NAION + acute HTN crisis



Small, crowded disc

40

NON-ARTERITIC ISCHEMIC OPTIC NEUROPATHY (NAION)

Questionable risk factors/associated conditions

Nocturnal hypotension????

- **Perhaps a contributor in eyes with progressive VL**
- Sig association btw progressive VF loss and nocturnal hypotension in pts with HTN taking PO hypotensives (Hyreh 1994 AJO)

New evidence against nocturnal hypotension hypothesis:

24-Hour Blood Pressure Monitoring in Patients With Anterior Ischemic Optic Neuropathy

Klara Landau, MD; Jacqueline M. S. Winterkorn, PhD, MD; Lionel U. Mailloux, MD; et al
Arch Ophthalmol. 1996;114(5):570-575.

- **No difference in nighttime diastolic nadir** compared to controls
- Pts with NAION consistently had a lower mean daytime BP than controls and a **lag in the usual rise in BP in the morning** to meet increasing daytime demands for perfusion

ESC European Society of Cardiology — European Heart Journal (2020) **41**, 4565–4576 doi:10.1093/eurheartj/ehz754

CLINICAL RESEARCH
Hypertension

Bedtime hypertension treatment improves cardiovascular risk reduction: the Hygia Chronotherapy Trial

- 19,084 pts followed for 6yrs
- Half took BP meds in AM, half at bedtime
- **Sig lower risk of CVD, MI, cardiac failure, or stroke with bedtime dosing**

41

Non-Arteritic Ischemic Optic Neuropathy (NAION)

Questionable risk factors/associated conditions

- **Smoking (49%)?**
- **Certain meds: phosphodiesterase-5 (PDE-5) inhibitors, amiodarone,** oxymetazoline, phenylephrine?
- **Cataract surgery?**
- Hyperopia?
- Papillary vitreous detachment?
- **NAION eyes do NOT have increased incidence of ipsilateral carotid disease**
-

Viagra (Sildenafil) FDA Package Insert

WARNINGS AND PRECAUTIONS

- Patients should stop VIAGRA and seek medical care if a sudden loss of vision occurs in one or both eyes, which could be a sign of non arteritic anterior ischemic optic neuropathy (NAION). VIAGRA should be used with caution, and only when the anticipated benefits outweigh the risks, in patients with a history of NAION. Patients with a "crowded" optic disc may also be at an increased risk of NAION. (5.3)

Cordarone (amiodarone) FDA Package Insert

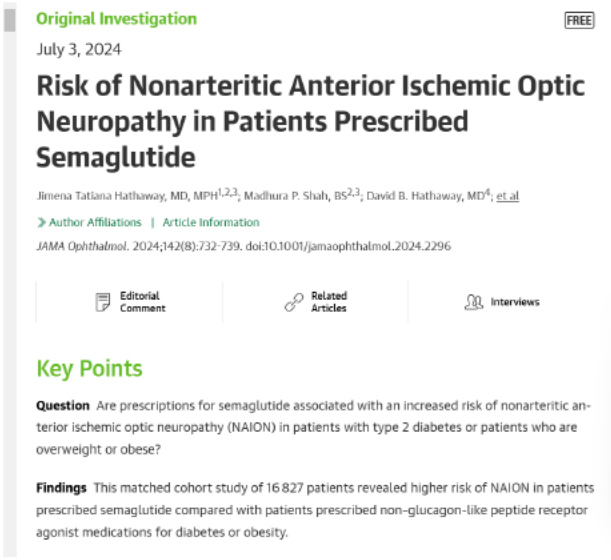
Loss of Vision

Cases of optic neuropathy and/or optic neuritis, usually resulting in visual impairment, have been reported in patients treated with amiodarone. In some cases, visual impairment has progressed to permanent blindness. Optic neuropathy and/or neuritis may occur at any time following initiation of therapy. A causal relationship to the drug has not been clearly established. If symptoms of visual impairment appear, such as changes in visual acuity and decreases in peripheral vision, prompt ophthalmic examination is recommended. Appearance of optic neuropathy and/or neuritis calls for re-evaluation of Cordarone therapy. The risks and complications of antiarrhythmic therapy with Cordarone must be weighed against its benefits in patients whose lives are threatened by cardiac arrhythmias. Regular ophthalmic examination, including funduscopy and slit-lamp examination, is recommended during administration of Cordarone (See "ADVERSE REACTIONS").

42

Non-Arteritic Ischemic Optic Neuropathy (NAION)

- **The findings suggest a potential risk of NAION associated with prescriptions for semaglutide**
- The cumulative incidence of NAION for the semaglutide and non–GLP-1 RA cohorts over 3 years was 8.9% (95% CI, 4.5%-13.1%) and 1.8% (95% CI, 0%-3.5%), respectively.



Original Investigation FREE

July 3, 2024

Risk of Nonarteritic Anterior Ischemic Optic Neuropathy in Patients Prescribed Semaglutide

Jimena Tatiana Hathaway, MD, MPH[1,2,3]; Madhura P. Shah, BS[2,3]; David B. Hathaway, MD[4]; et al

Author Affiliations | Article Information

JAMA Ophthalmol. 2024;142(8):732-739. doi:10.1001/jamaophthalmol.2024.2296

Editorial Comment | Related Articles | Interviews

Key Points

Question Are prescriptions for semaglutide associated with an increased risk of nonarteritic anterior ischemic optic neuropathy (NAION) in patients with type 2 diabetes or patients who are overweight or obese?

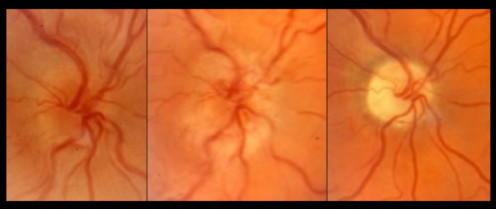
Findings This matched cohort study of 16 827 patients revealed higher risk of NAION in patients prescribed semaglutide compared with patients prescribed non–glucagon-like peptide receptor agonist medications for diabetes or obesity.

43

NON-ARTERITIC ISCHEMIC OPTIC NEUROPATHY (NAION)

Prognosis/Clinical course

- **Nonprogressive (~75%) and progressive forms exist**
  - If progressive, usually occurs within the first month
- **VA stable or improves in most without tx** (IONDT)
  - 31% gained ≥3 lines at 2 years
- Repeated attacks in the same eye are unusual (6.4%)



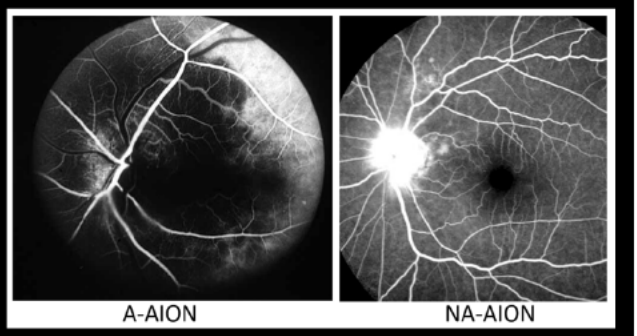
- Fellow eye risk/bilateral involvement
  - **5 year risk of 2nd eye involvement ~15%**
  - **Lifetime risk may be as high as 30-40%**
  - Greater risk in **younger pts (<50yo)** those with DM/HTN, and worse VA

44

NON-ARTERITIC ISCHEMIC OPTIC NEUROPATHY (NAION)

Work up

- **Patients ≥ 50yo: ESR/CRP/CBC with diff and platelet count; maybe also IVFA to R/O GCA**
  - **Neuroimaging is usually unnecessary** in older pts with classically presenting NA-AION
- Younger pts: Labs for hypercoagulability/inflammatory or infectious neuropathies, MRI to exclude optic neuritis/compressive or infiltrative conditions
- **All pts: Investigate for DM, HTN, chol, cardiovascular disease, sleep apnea**
- **Embolic WU (carotid eval) unnecessary** unless concurrent retinal/choroidal ischemia/retinal emboli



A-AION NA-AION

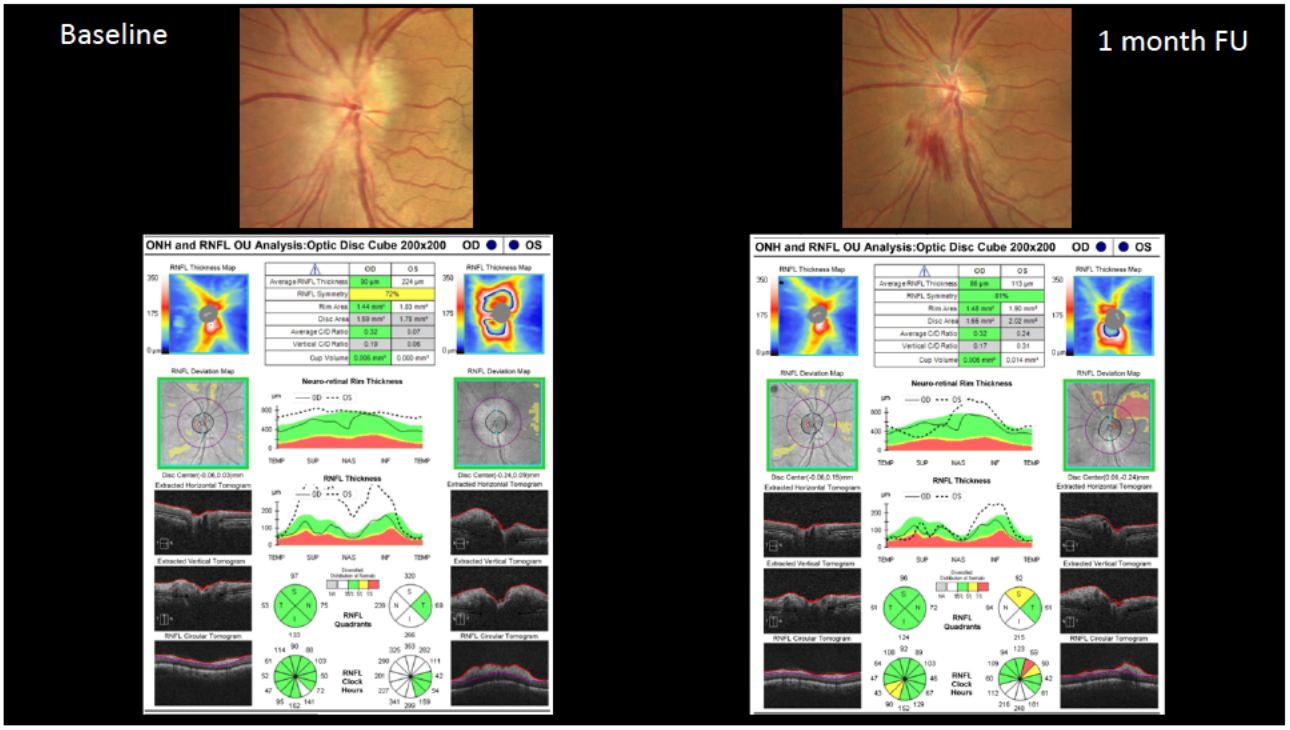
45

NON-ARTERITIC ISCHEMIC OPTIC NEUROPATHY (NAION)

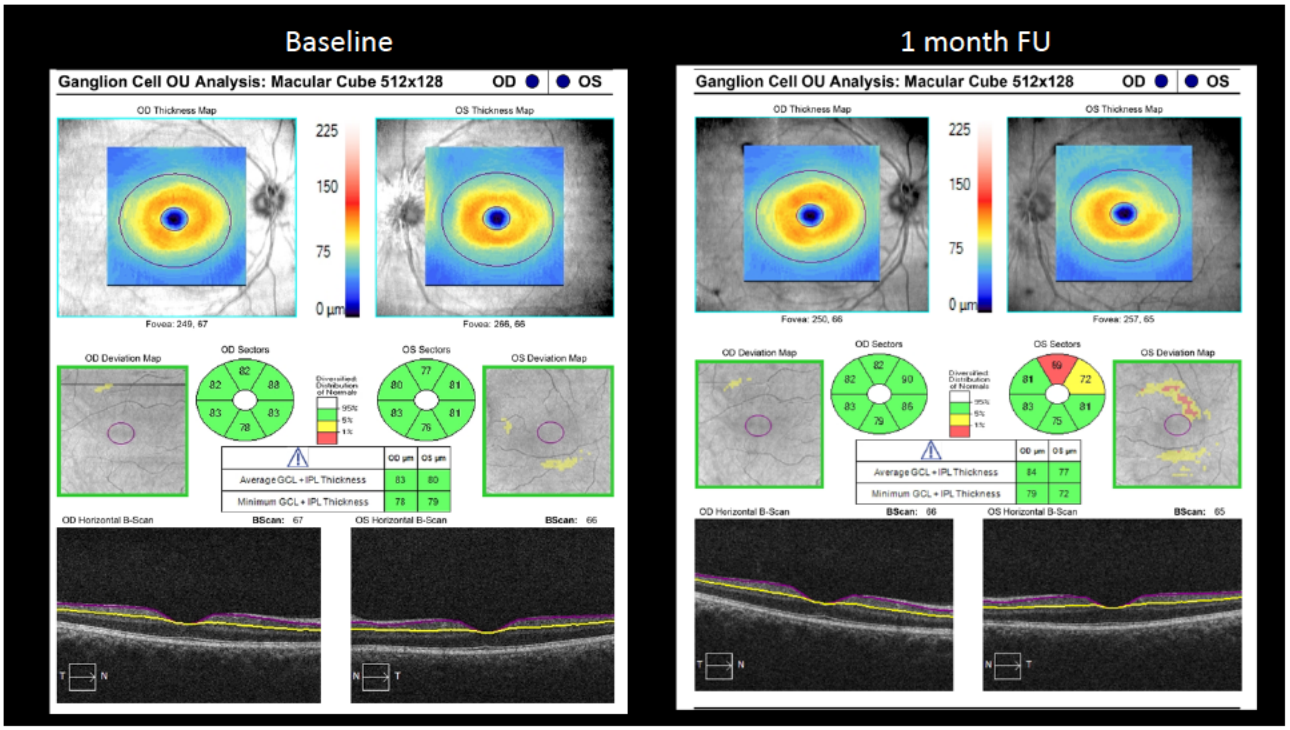
Management

- **No proven therapy**
- **Control systemic predisposing risk factors/cardiovascular risk factor modification**
- **D/C smoking**
- **Caution against use of high-risk meds (erectile dysfunction drugs, amiodarone) in individuals with history of NA-AION and "discs at risk"**
- **81mg ASA?** -does not work as prophylaxis for the fellow eye but may reduce risk of secondary stroke
- Avoid over treating HTN?
- Levodopa and carbidopa? Benefit in one study, non-reproducible

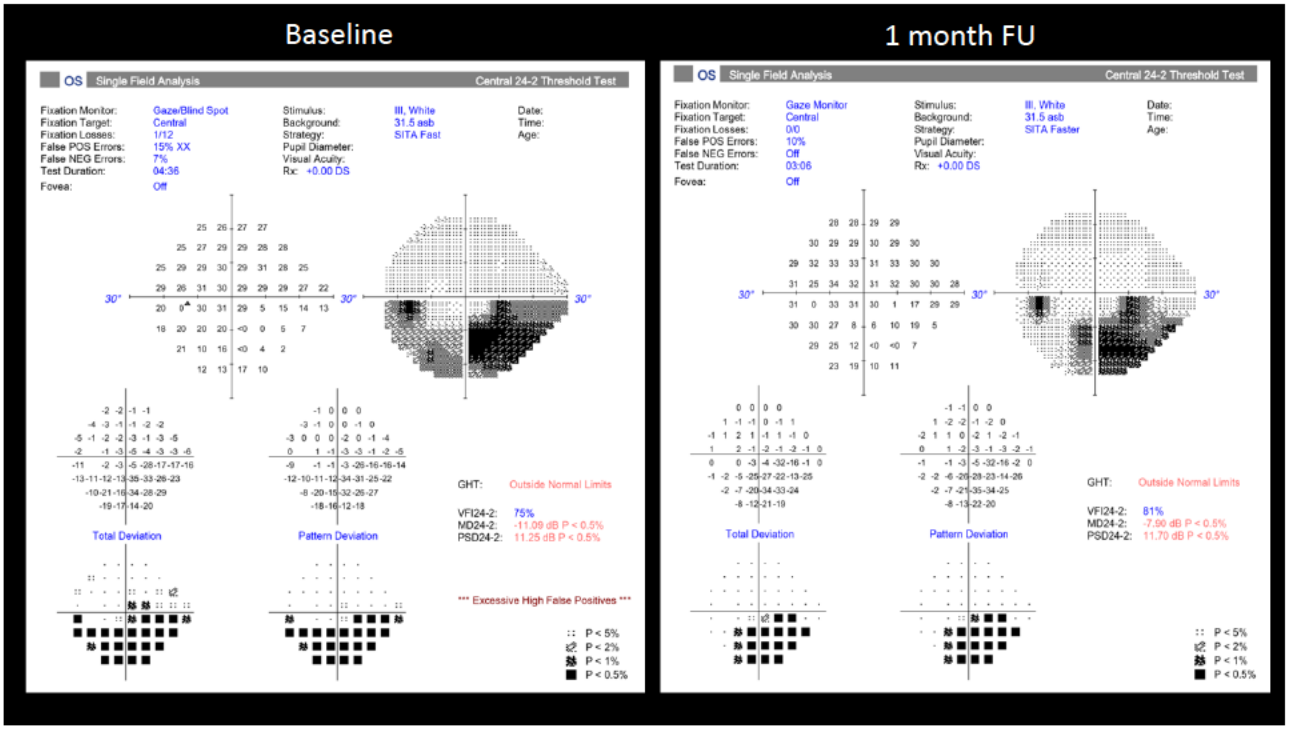
46


Baseline
1 month FU
ONH and RNFL OU Analysis:Optic Disc Cube 200x200 OD OS
ONH and RNFL OU Analysis:Optic Disc Cube 200x200 OD OS

47


Baseline
1 month FU
Ganglion Cell OU Analysis: Macular Cube 512x128 OD OS
Ganglion Cell OU Analysis: Macular Cube 512x128 OD OS
OD Thickness Map
OS Thickness Map
OD Deviation Map
OD Sectors
OS Sectors
OS Deviation Map
OD Horizontal B-Scan
OS Horizontal B-Scan
Average GCL + IPL Thickness
Minimum GCL + IPL Thickness

48



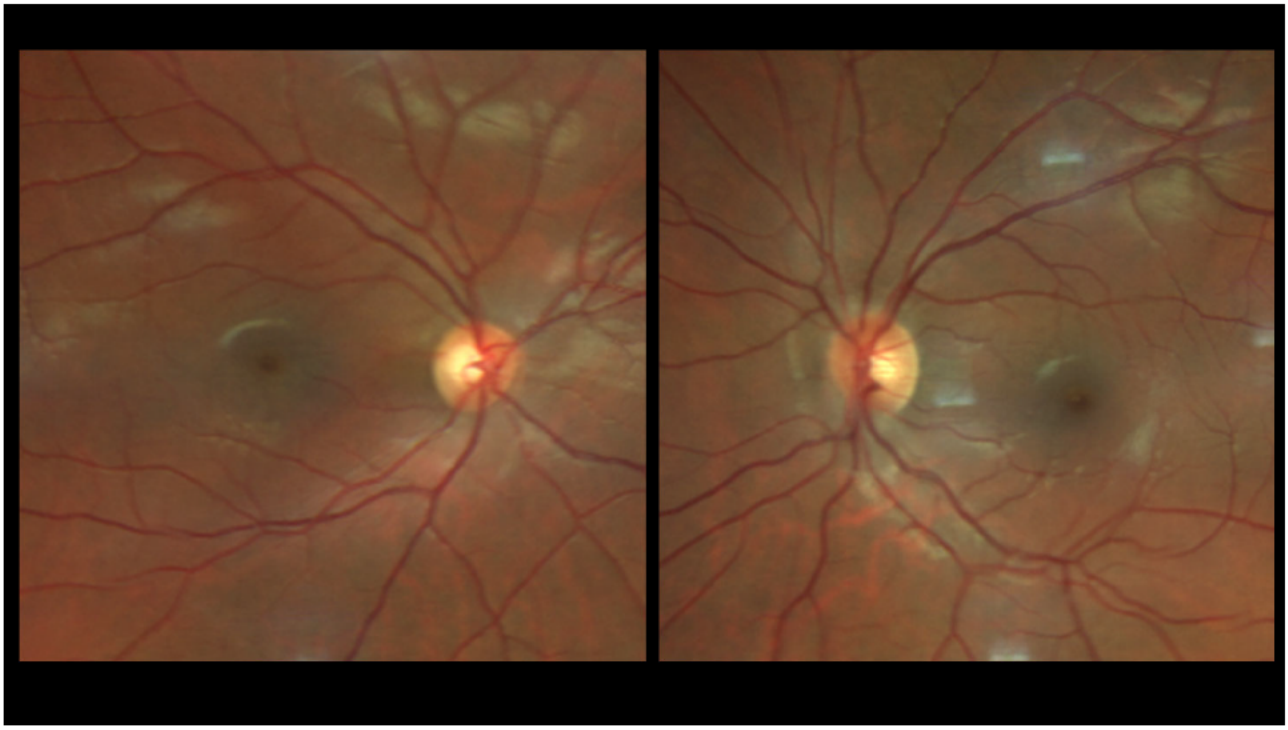
49

WHEN THE LITTLE THINGS MATTER MOST....

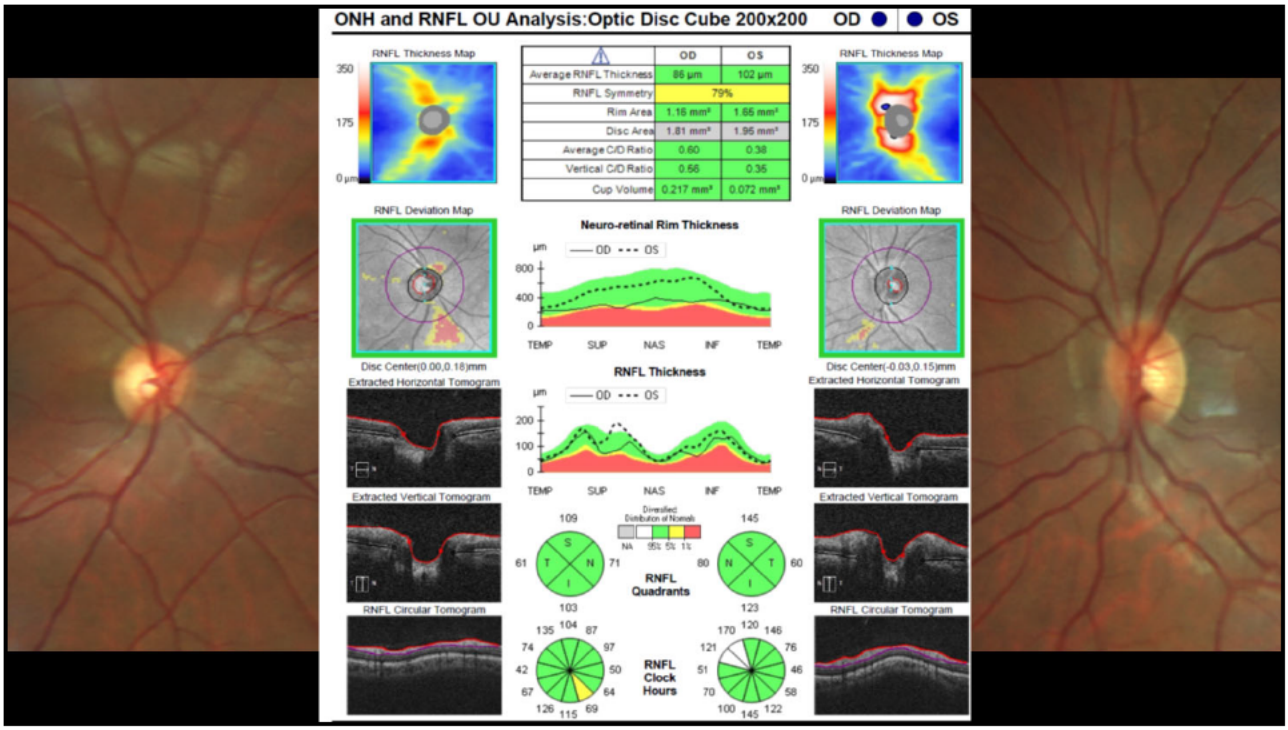
27yo Native American female – Sent over from urgent care, sudden central vision loss OS 6 days ago that has been worsening , headache and pain (7/10) worse on eye movt

- Oc Hx: Unremarkable
- Med Hx: depression, anxiety, asthma, eczema, bipolar disorder, smoker
- Meds: Albuterol/Ipratropium inhaler
- VAs @dist:
  - OD 20/25
  - **OS LP (PHNI)**
- Entrance testing:
  - CVF: **Central/inferior constriction of VF OS**
  - Pupils: Sluggish direct response OS, **2+ APD OS**
  - EOMs: Full but with **pain OS**
- SLE: WNLs OU
- IOPs: 13 OD/OS mmHg
- BP: 111/76

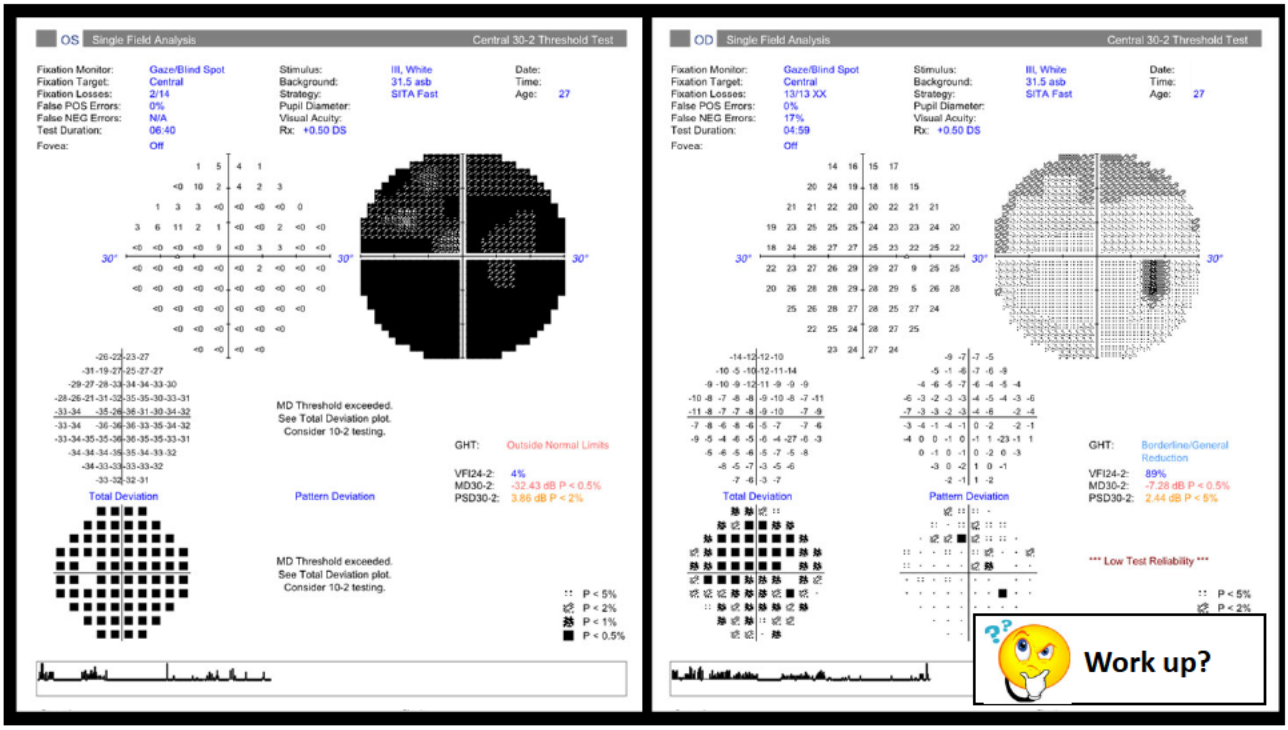
50



51

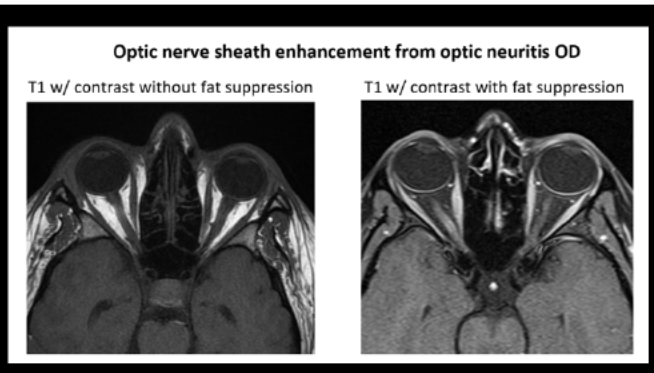

ONH and RNFL OU Analysis:Optic Disc Cube 200x200
OD
OS
RNFL Thickness Map
RNFL Deviation Map
Neuro-retinal Rim Thickness
RNFL Thickness
Extracted Horizontal Tomogram
Extracted Vertical Tomogram
RNFL Circular Tomogram
RNFL Quadrants
RNFL Clock Hours
TEMP
SUP
NAS
INF

52



53

Work-up

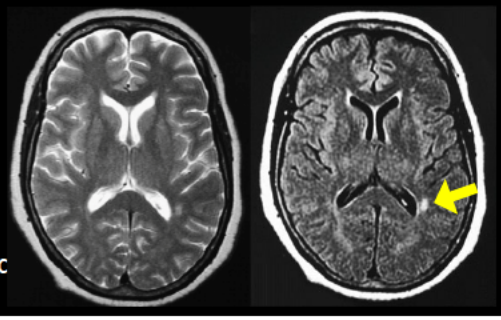


Neuroimaging (MRI brain and orbits)

- With & w/o contrast enhancement
- + orbital fat suppression
- T2 with FLAIR

- Labs?
  - Inflammatory / infectious / hypercoagulability
  - Anti-aquaporin 4 (anti-NMO) & anti-MOG antibo

FLAIR (fluid level attenuated inversion recovery)



54

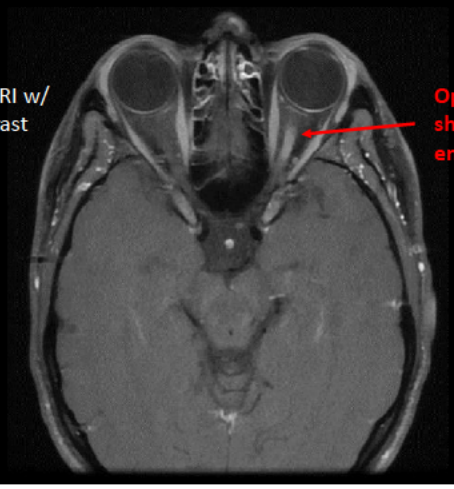
Neuroimaging

What is this patient's risk of CDMS over the next 15 years?

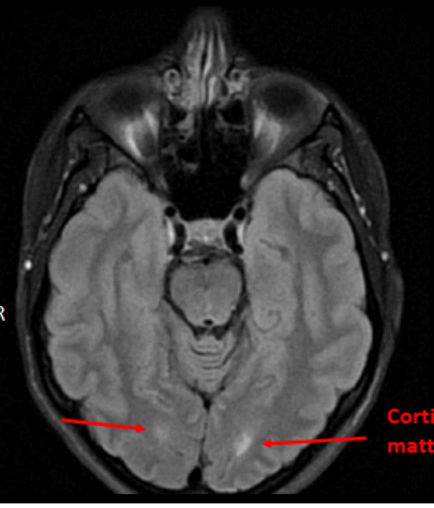
- **MRI of brain and orbits w/ & w/out contrast with fat suppression and FLAIR**

T1 MRI w/ contrast

Optic nerve sheath enhancement



T2 MRI with FLAIR



Cortical white matter lesions

55

www.pollev.com/retina

When poll is active, respond at **pollev.com/retina**
Text **RETINA** to **37607** once to join

What treatment option is MOST APPROPRIATE for this patient?

Observation
Oral steroid alone
IV steroid followed by oral taper
Oral acetazolamide

Start the presentation to see live content. For screen share software, share the entire screen. Get help at **pollev.com/app**

56

What treatment option is MOST APPROPRIATE for this patient?

A. Observation
B. Oral steroid alone
C. IV steroid followed by oral taper
D. Oral acetazolamide

57

Diagnosis & Plan

Retrobulbar optic neuritis OS

- Tx: **IV methylprednisolone** 1g/day x 3 days + 60 mg oral prednisone x 11 days with taper of 20 mg on day 1, followed by 10 mg on days 2 to 4
- Refer to **neurology for full neurologic exam & consideration of immunomodulatory tx (started on diroximel fumarate)**
- Vit D level

58

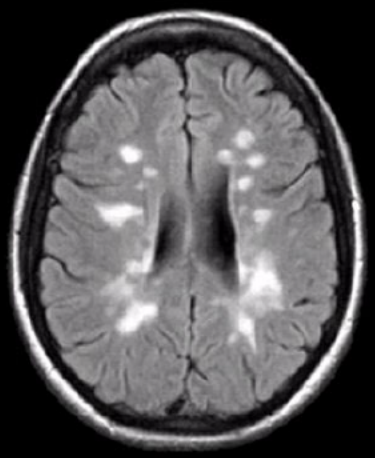
OPTIC NEURITIS (ON)

Prognosis/clinical course

- Recovery should begin within 1 month
- 72% expected to be 20/20 after 15 years
- Similar prognosis at 1 year regardless of treatment

Development of MS

- One episode of ON does not = MS
- **The most reliable predictor for the future development of MS is the presence of MRI white matter lesions**
- ONTT 15 year FU
  - **Overall, 50% developed CDMS**
    - **Positive MRI- 72%**
    - Negative MRI- 25%
- Other predictive tests- CSF oligoclonal bands in pts with normal MRI



59

OPTIC NEURITIS (ON)

- **Atypical ON cases work-up!**- MRI (brain & spinal cord), CBC, FTA-abs, RPR, tick panel, ACE, CXR, ESR/CRP, ANA, anti-AQP4 (NMO), anti-MOG antibodies, CSF analysis, bartonella ab panel, B12 & folate, copper, genetic mitochondrial analysis, ERG prn
- Atypical ON features:
  - NLP (NMO)
  - Progression of VL after 1-2 weeks
  - Simultaneous bilateral VL (NMO, lebers, toxic)
  - Disc/retinal hemorrhages (NA-ION, syph)
  - Severe disc swelling
  - Macular/retinal exudates (neuroretinitis)
  - Vitritis/uveitis (sarcoid, syph)
  - Retinal vein sheathing (sarcoid, SLE)
  - Lack of partial recovery by 1 month
  - Absence of pain
  - Persistent pain

60

OPTIC NEURITIS (ON)

Treatment

Scenario #1: First episode of typical acute ON, MRI white matter lesions & no prior history of MS

- **Consider IV corticosteroids followed by PO pred taper**
  - Reduces the risk of CDMS for the first 2 years
  - Hastens visual recovery
  - **Do NOT give PO steroids alone!!!**
- **Refer to neurologist/neuro-ophthalmologist for possible treatment with immunomodulatory med to reduce the risk of developing CDMS**
  - CHAPS, ETOMS, BENEFIT, PreCISe
- Vit D supplementation is levels are low

Scenario #2: First episode of typical acute ON, MRI negative, and no prior history of MS

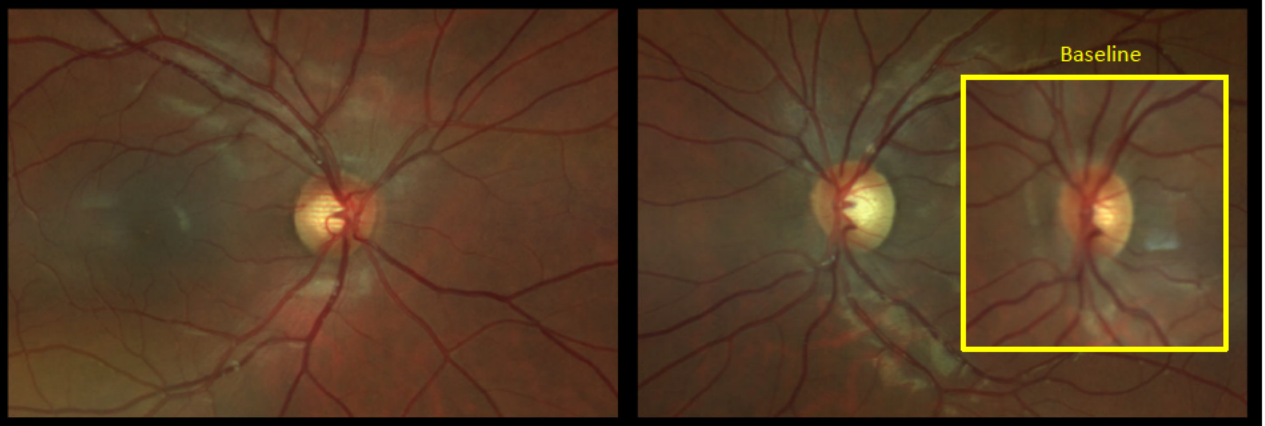
- **Observation vs IV corticosteroids followed by PO pred taper**
- Repeat MRI at 3, 6, 9, 12 months then yearly

Scenario #3: Recurrent bouts of ON with prior dx of MS

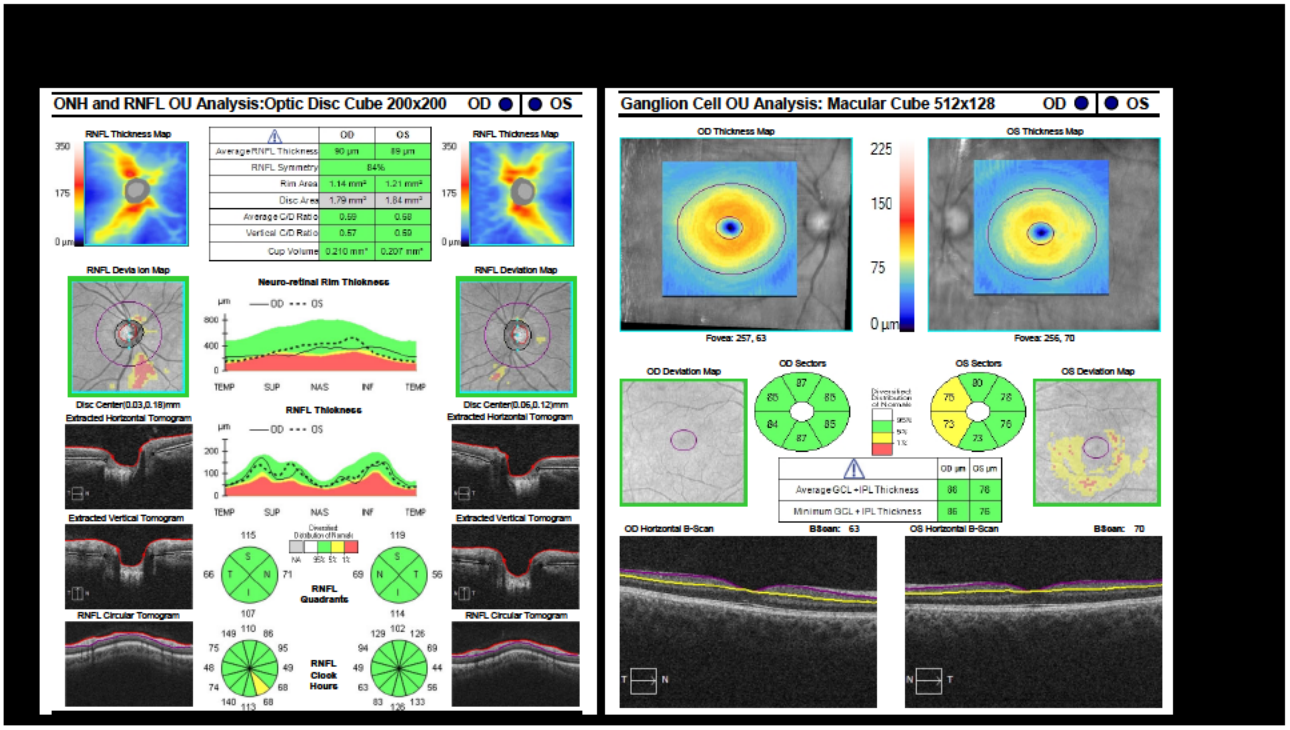
- Discuss with neurologist/neuro-ophthalmologist

61

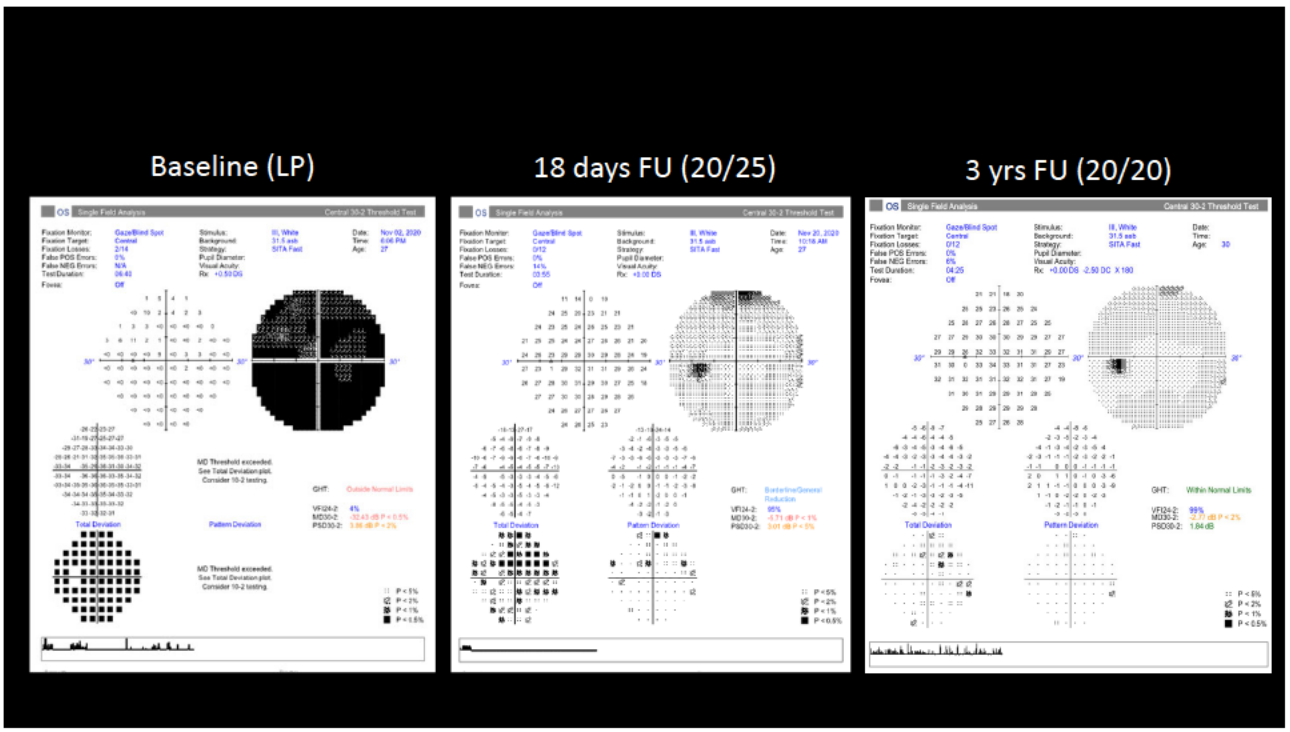
18 days FU



62


ONH and RNFL OU Analysis:Optic Disc Cube 200x200 OD OS
RNFL Thickness Map
RNFL Deviation Map
Neuro-retinal Rim Thickness
RNFL Thickness
RNFL Quadrants
RNFL Clock Hours
Disc Center(0.03,0.18)mm
Disc Center(0.06,0.12)mm
Extracted Horizontal Tomogram
Extracted Vertical Tomogram
RNFL Circular Tomogram
Ganglion Cell OU Analysis: Macular Cube 512x128 OD OS
OD Thickness Map
OS Thickness Map
OD Deviation Map
OS Deviation Map
OD Sectors
OS Sectors
OD Horizontal B-Scan
OS Horizontal B-Scan

63


Baseline (LP)
18 days FU (20/25)
3 yrs FU (20/20)

64

DIGGING DEEP

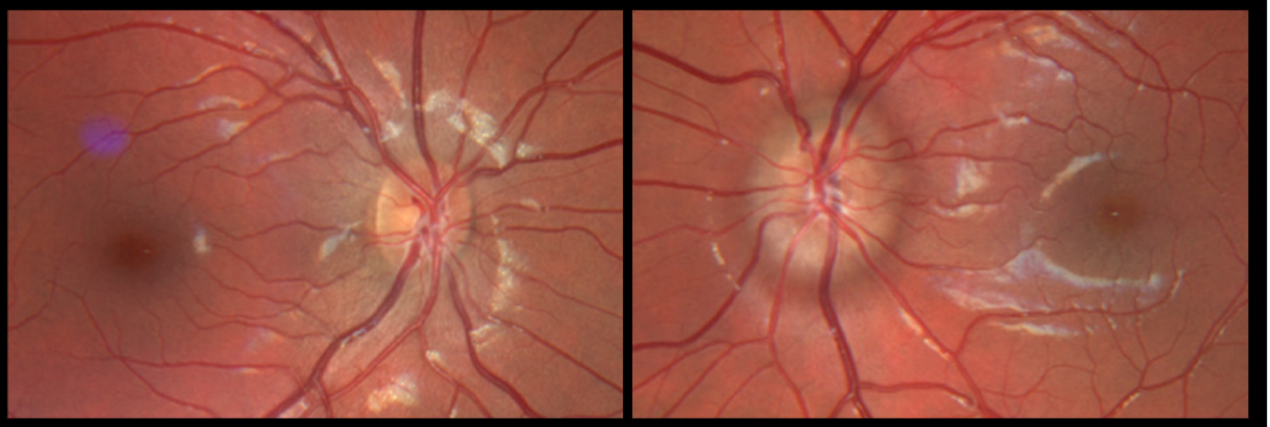
17yo female – Routine exam, complains of blur at distance x 1-2yrs

- Oc Hx: Unremarkable
- Med Hx: WNLs, no meds

- BCVAs @dist:
  - OD -1.00 sph $20/20^{-2}$
  - OS -1.00 -0.25 x 075 $20/20^{-1}$
- Entrance testing: **Nasal constriction of VF OS, 1+ APD OS**
- SLE: WNLs OU
- IOPs: OD 12/ OS 11 mmHg
- BP: 117/82

65

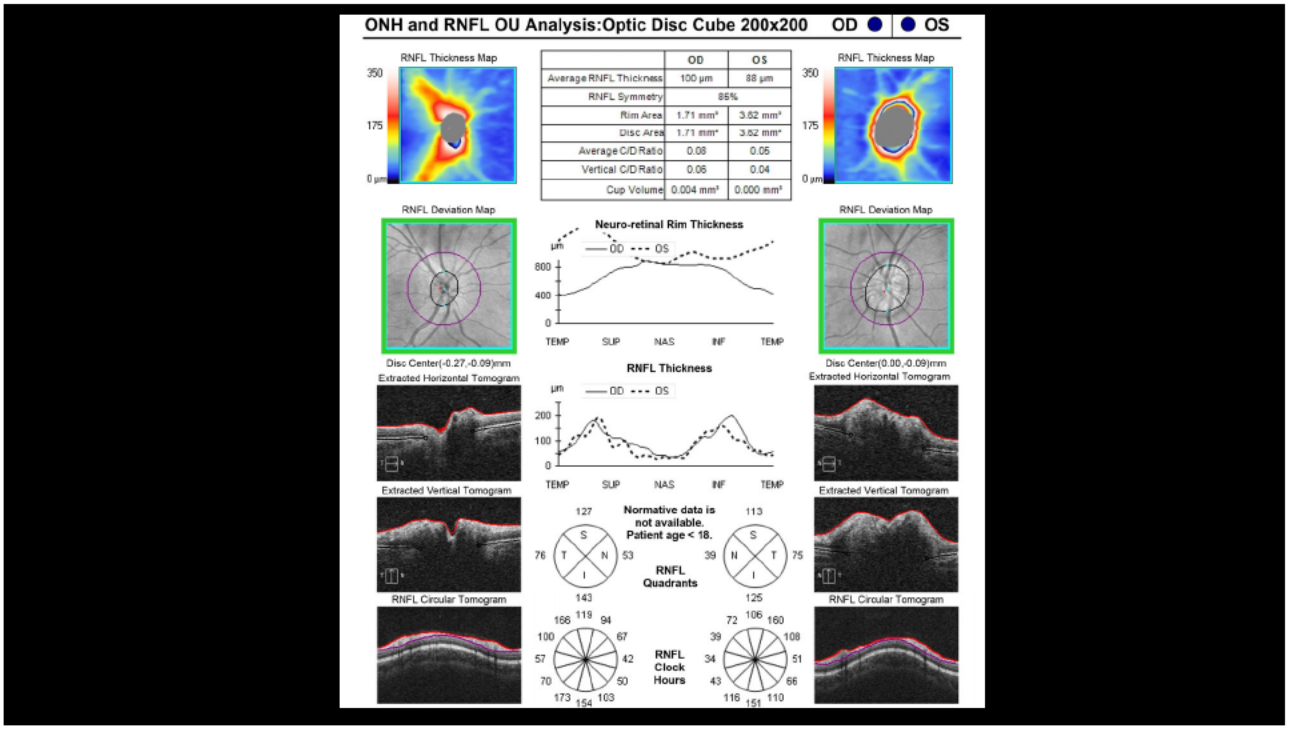
DIGGING DEEP



Are there features of TRUE papilledema present?

What additional ancillary testing is needed?

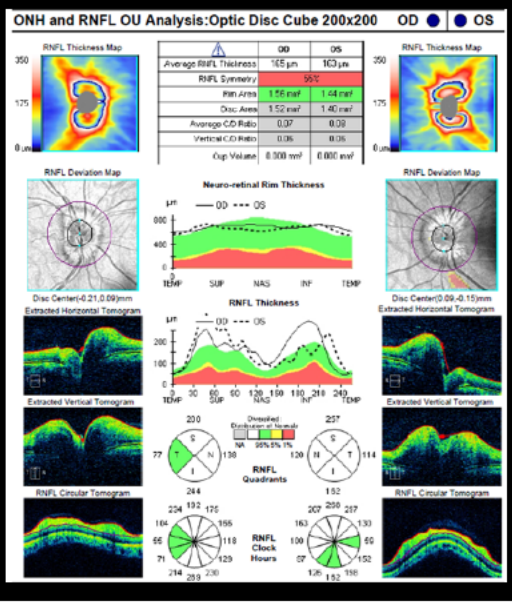
66



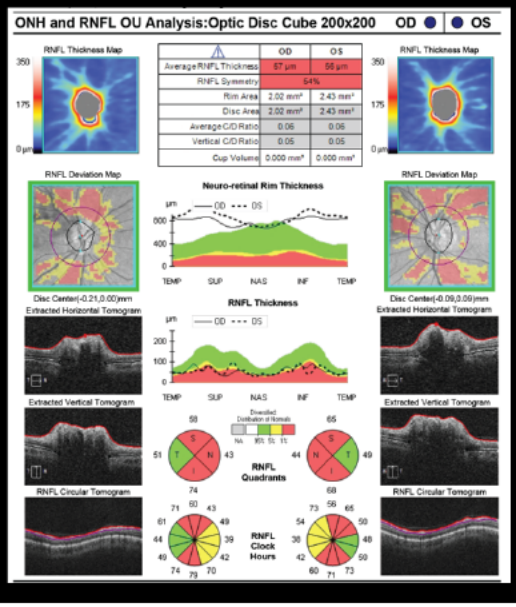
67

Papilledema vs Disc Drusen (NFL OCT)

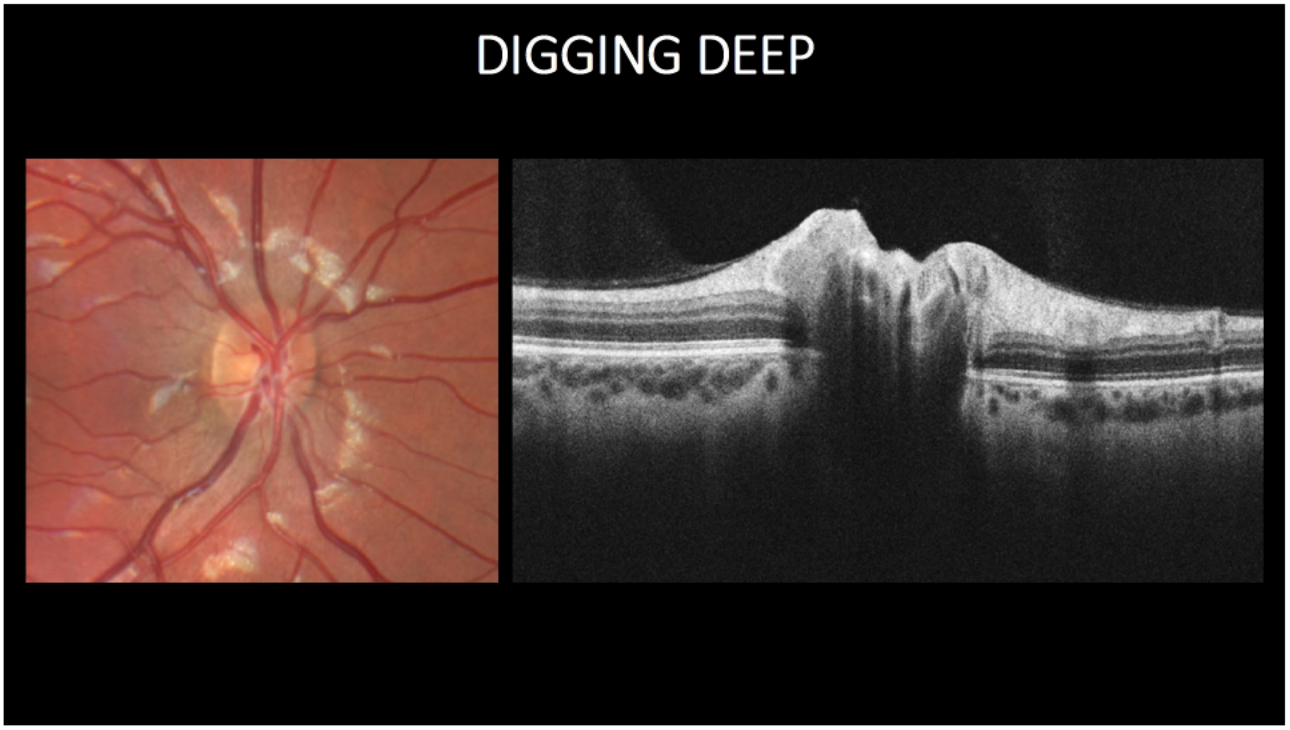
Papilledema



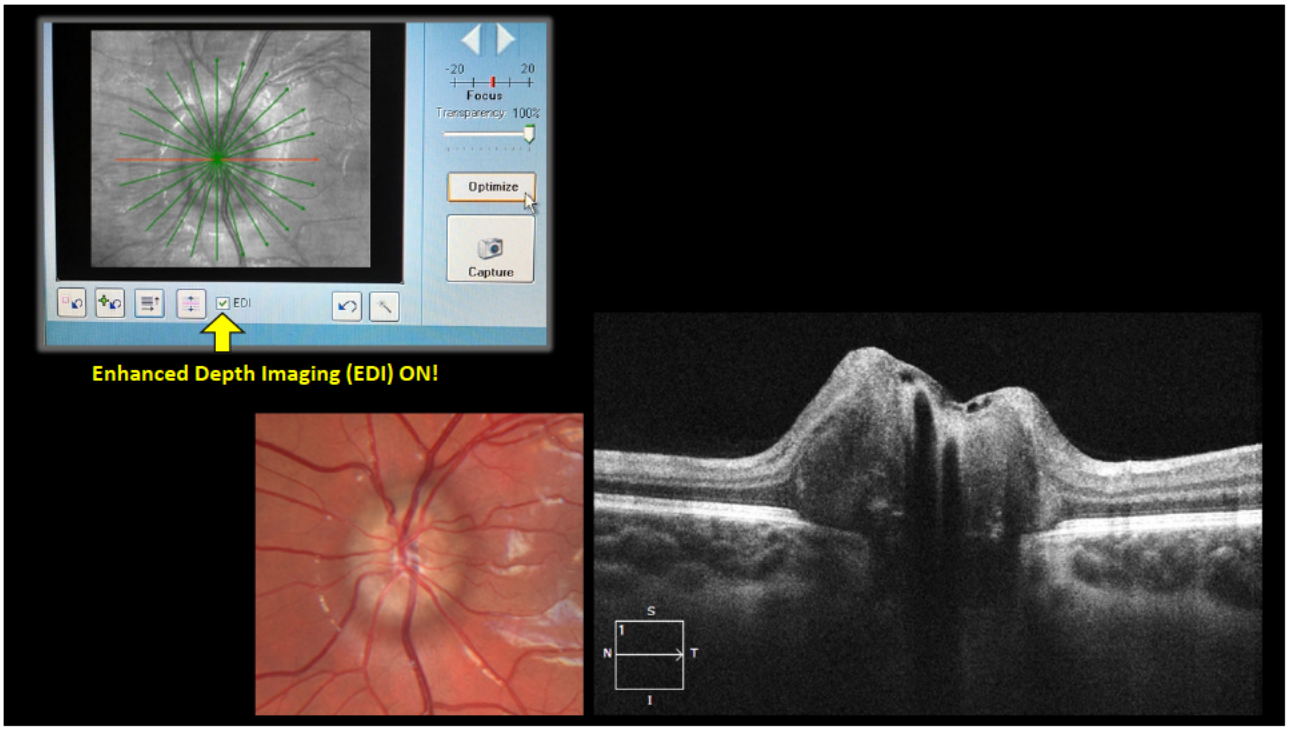
Disc drusen



68

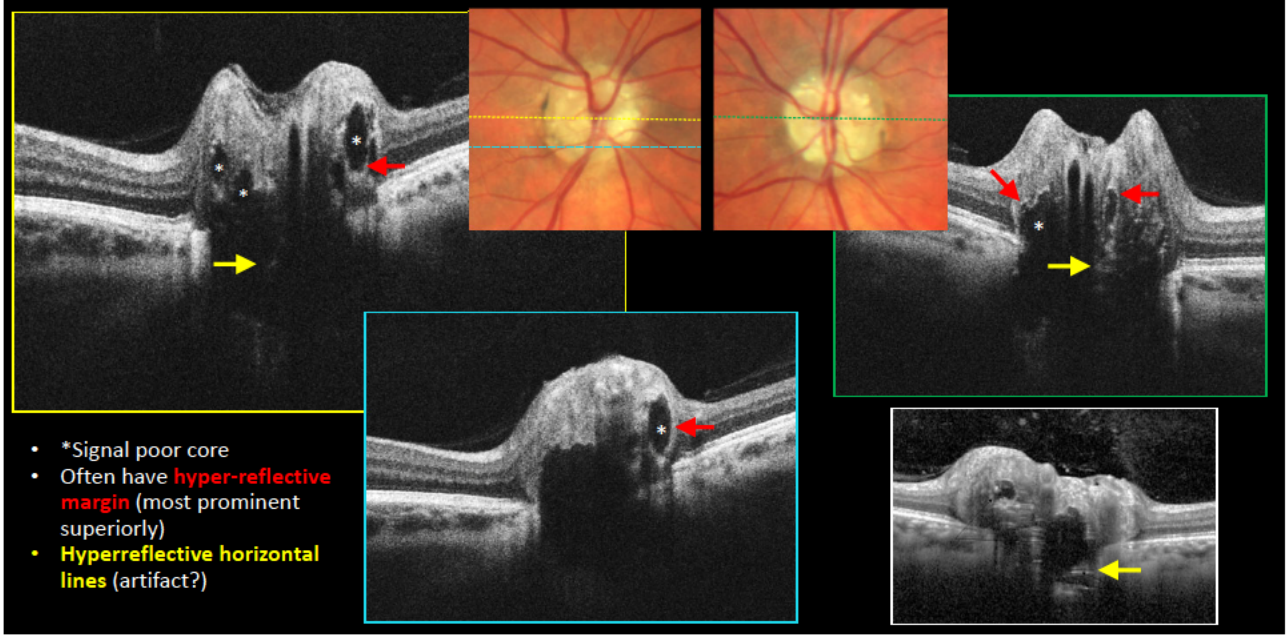

DIGGING DEEP

69


-20
20
Focus
Transparency 100%
Optimize
Capture
EDI
Enhanced Depth Imaging (EDI) ON!
S
N
T
I

70

OCT APPEARANCE OF DISC DRUSEN

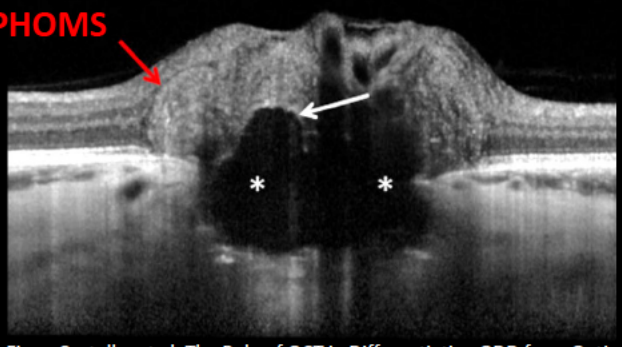


- *Signal poor core
- Often have **hyper-reflective margin** (most prominent superiorly)
- **Hyperreflective horizontal lines** (artifact?)

71

Peripapillary Hyperreflective Ovoid Mass-like Structures (PHOMS)

- A **nonspecific OCT sign** of prelaminar peripapillary axonal distension/herniation and crowding that **can occur in acquired and dysplastic ONH anomalies** (optic disc drusen, papilledema, myopic tilted discs, etc.)
- **PHOMS** are **NOT optic disc drusen!!!**; & NOT an optic disc drusen precursor



* Disc drusen hyporeflective core

White arrow = Disc drusen hyperreflective margin or "hat"

Fiona Costello, et al. The Role of OCT in Differentiating ODD from Optic Disc Edema. Asia-Pac J Ophthalmology 2018

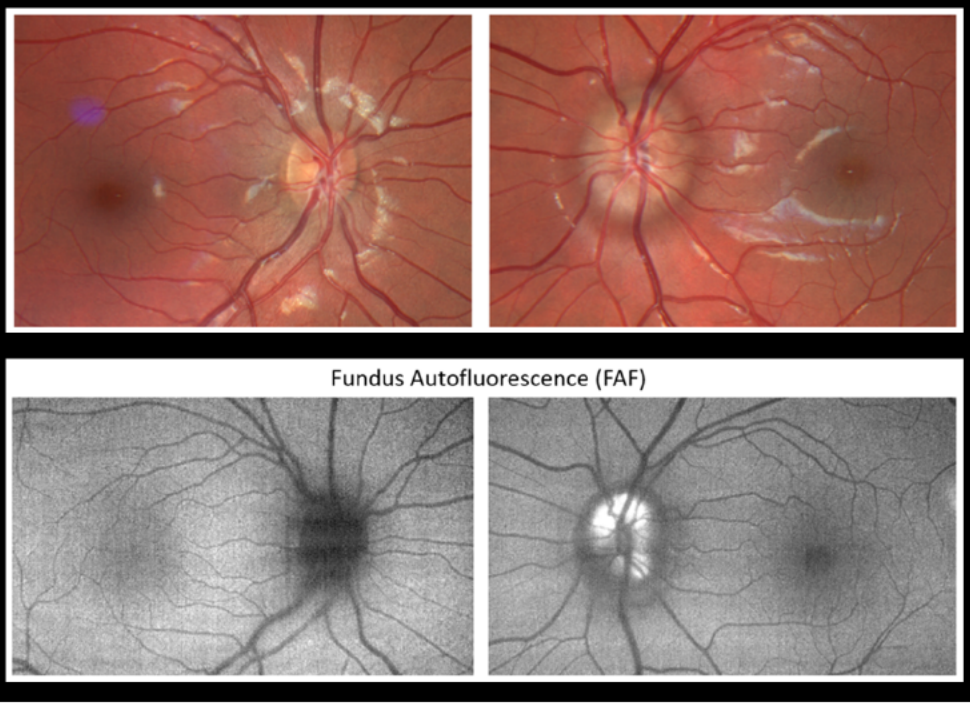
72

Peripapillary Hyperreflective Ovoid Mass-like Structures (PHOMS)

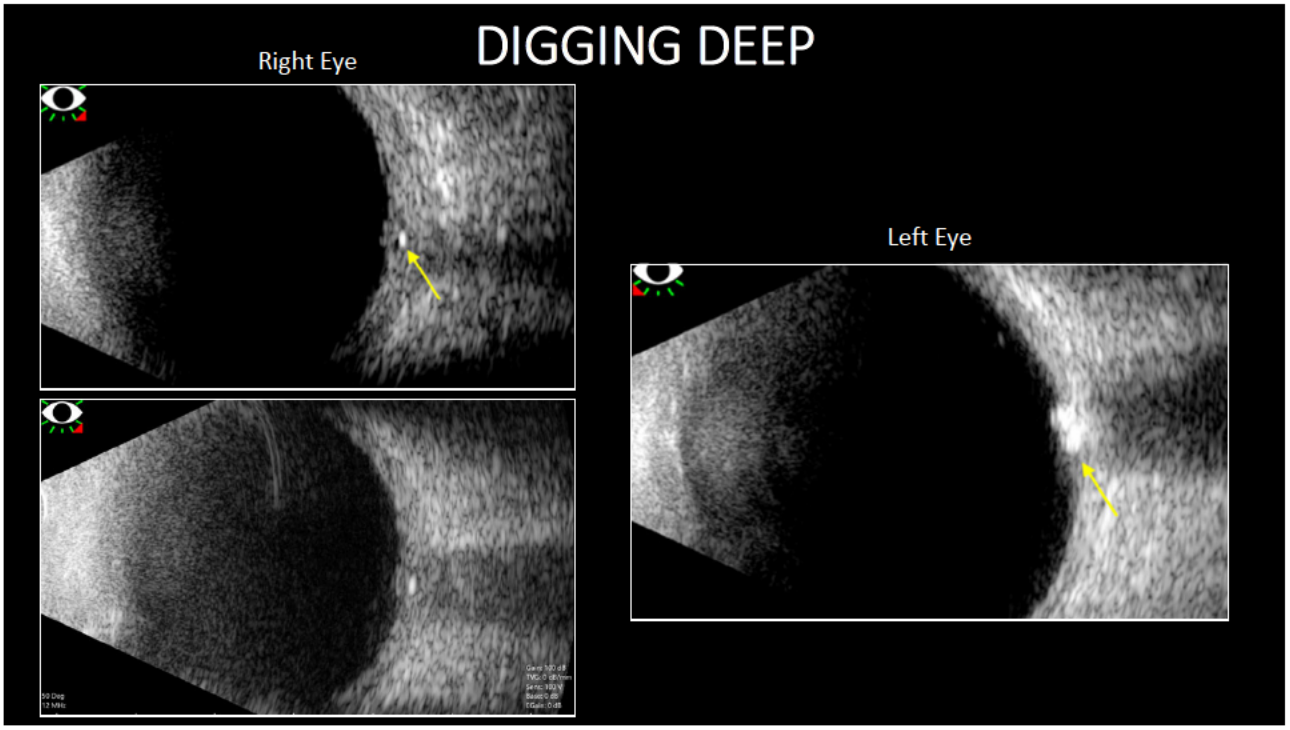
- **PHOMS** are **NOT optic disc drusen!!!**

Fiona Costello, et al. The Role of OCT in Differentiating ODD from Optic Disc Edema. Asia-Pac J Ophthalmology 2018

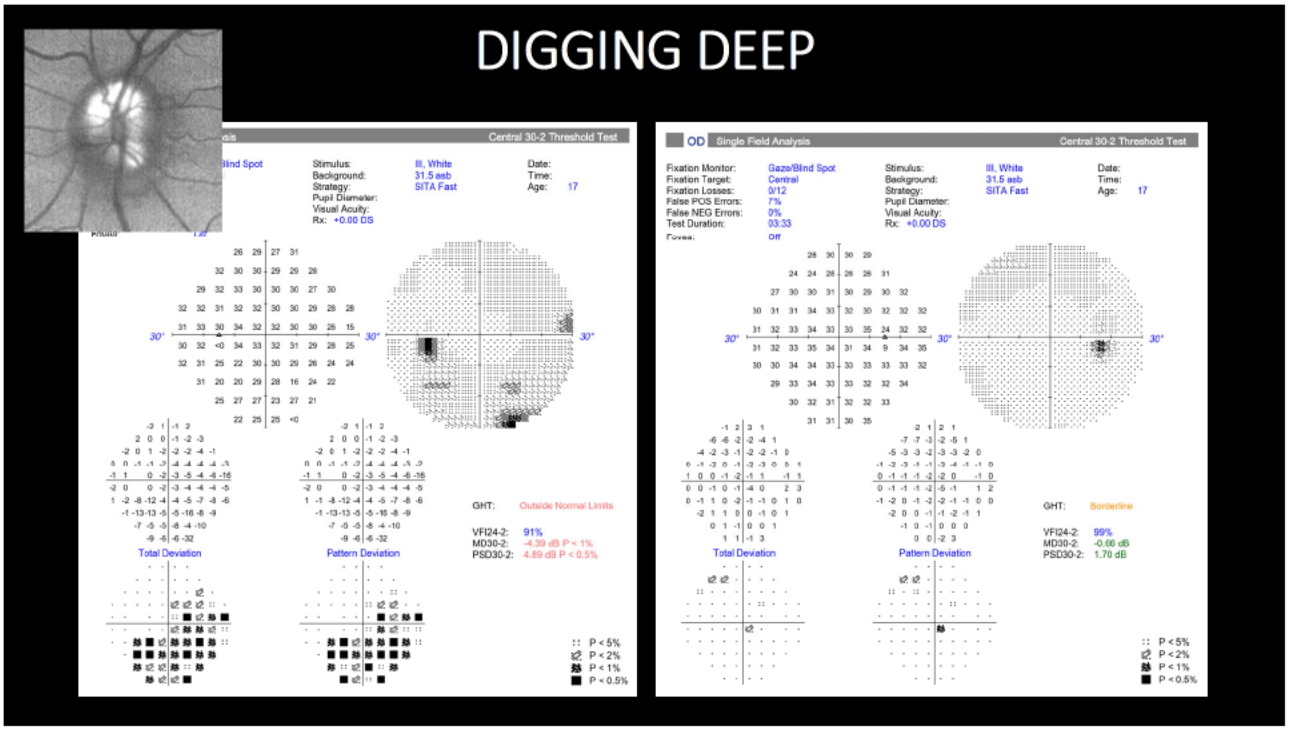
73



74


DIGGING DEEP
Right Eye
Left Eye

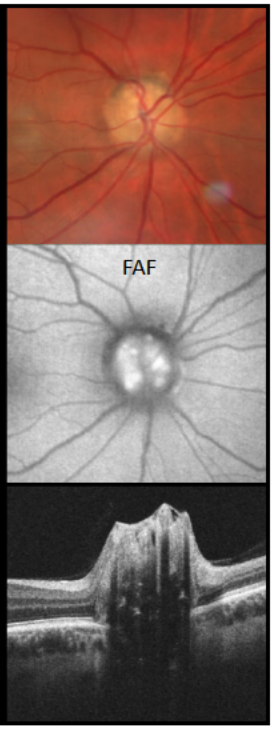
75


DIGGING DEEP
Central 30-2 Threshold Test
Total Deviation
Pattern Deviation
OD Single Field Analysis
Central 30-2 Threshold Test
Total Deviation
Pattern Deviation

76

OPTIC DISC DRUSEN

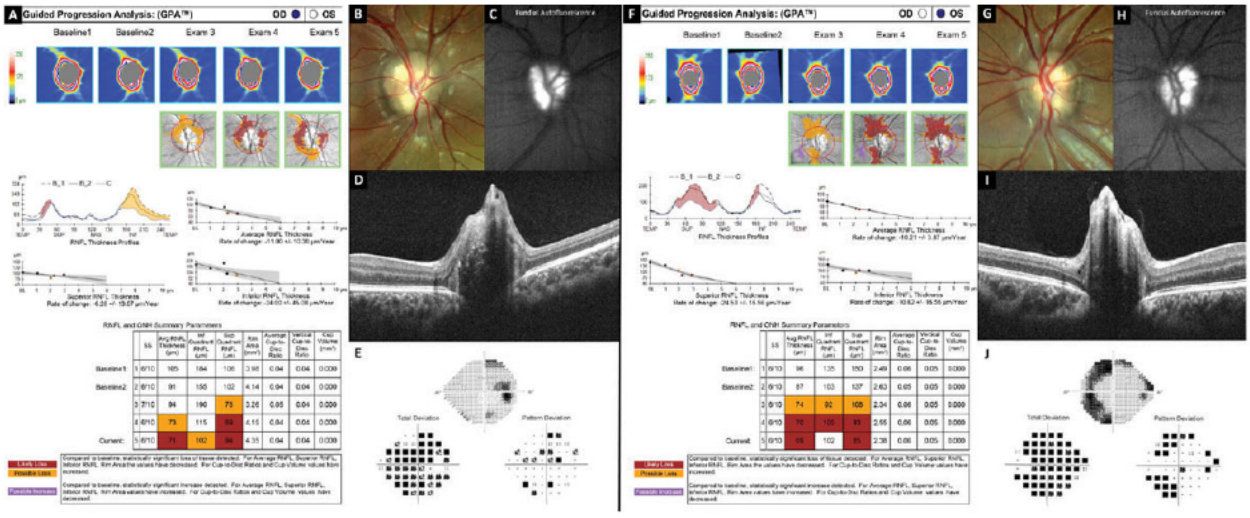
- Colloid bodies within substance of ONH **anterior to the lamina cribrosa**
- Extracellular deposition of axoplasmic material with ultimate calcification (owing to narrow posterior scleral foramen/Bruch membrane opening)
- VL can occur but usually **long-standing, painless, and asymptomatic**
- **Stability demonstrated by serial examination may be key** in confirming a diagnosis of pseudopapilledema
- Signs: irregular (scalloped) disc margin, loss of central cup, anomalous vasculature (trifurcations), variable NFL & VF loss, no IVFA leakage, + SVP in most



77

Progressive NFL thinning and VF loss possible

Ex. severe disc drusen in a 16yo with progressive NFL thinning and VF loss



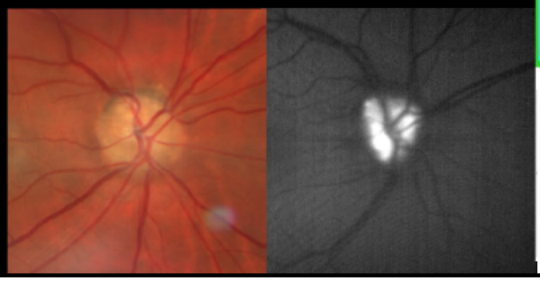
https://www.reviewofoptometry.com/article/take-oct-to-the-next-level

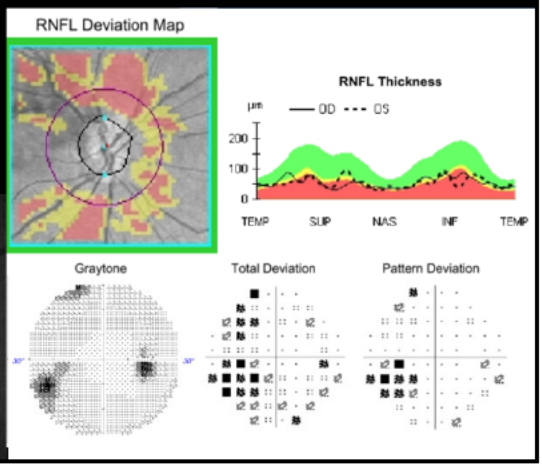
78

OPTIC DISC DRUSEN

Management:

- **Monitor for CNVM, NA-ION, CRVO**
- **No SOC tx**
  - **Lower IOP (esp if elevated)?**



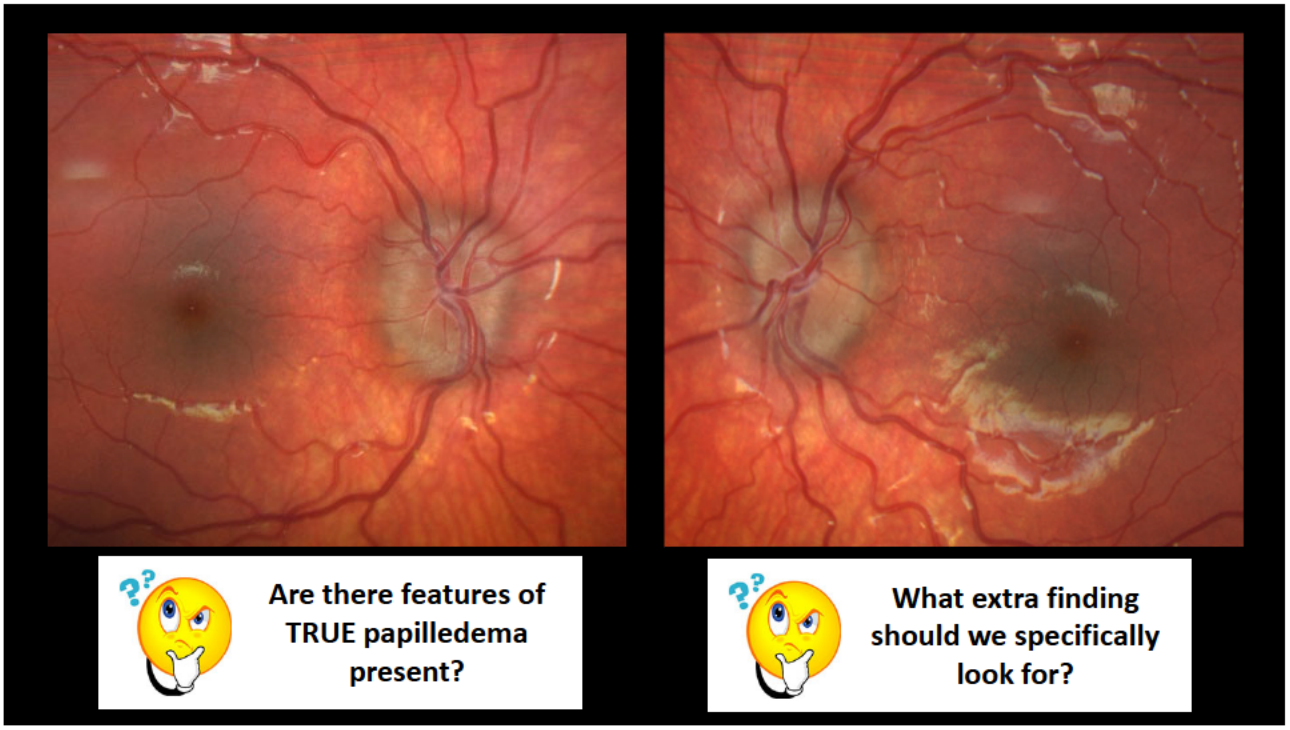


79

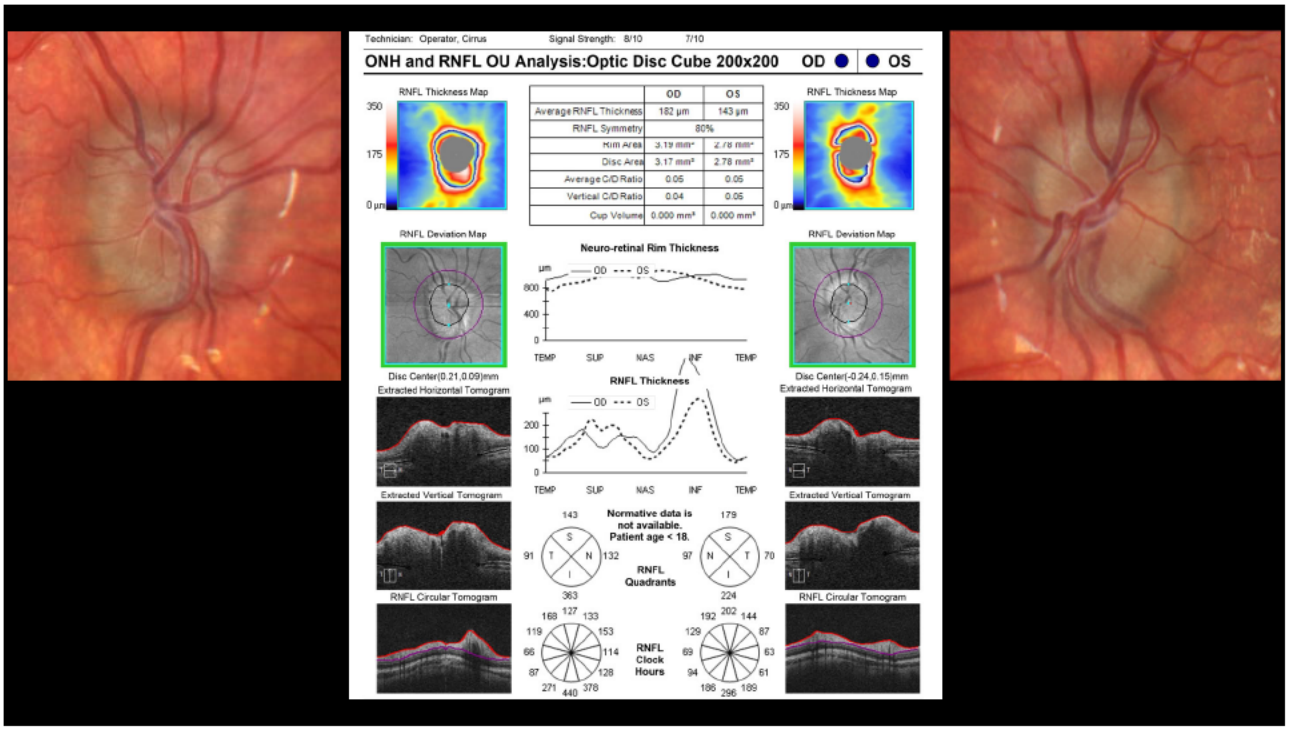
6yo white female

- CC: First eye exam. No ocular or systemic complaints per patient and mother.
- Oc Hx: unremarkable
- Medical Hx: unremarkable
- Meds: none
- Final Refraction
- OD: +1.00 -1.25x176: $20/20^{-3}$
- OS: +1.50 -1.75x002: $20/20^{-2}$
- Entrance testing and SLE of ant seg: Unremarkable OU
- IOPs 21/20mmHg
- BP: 98/66

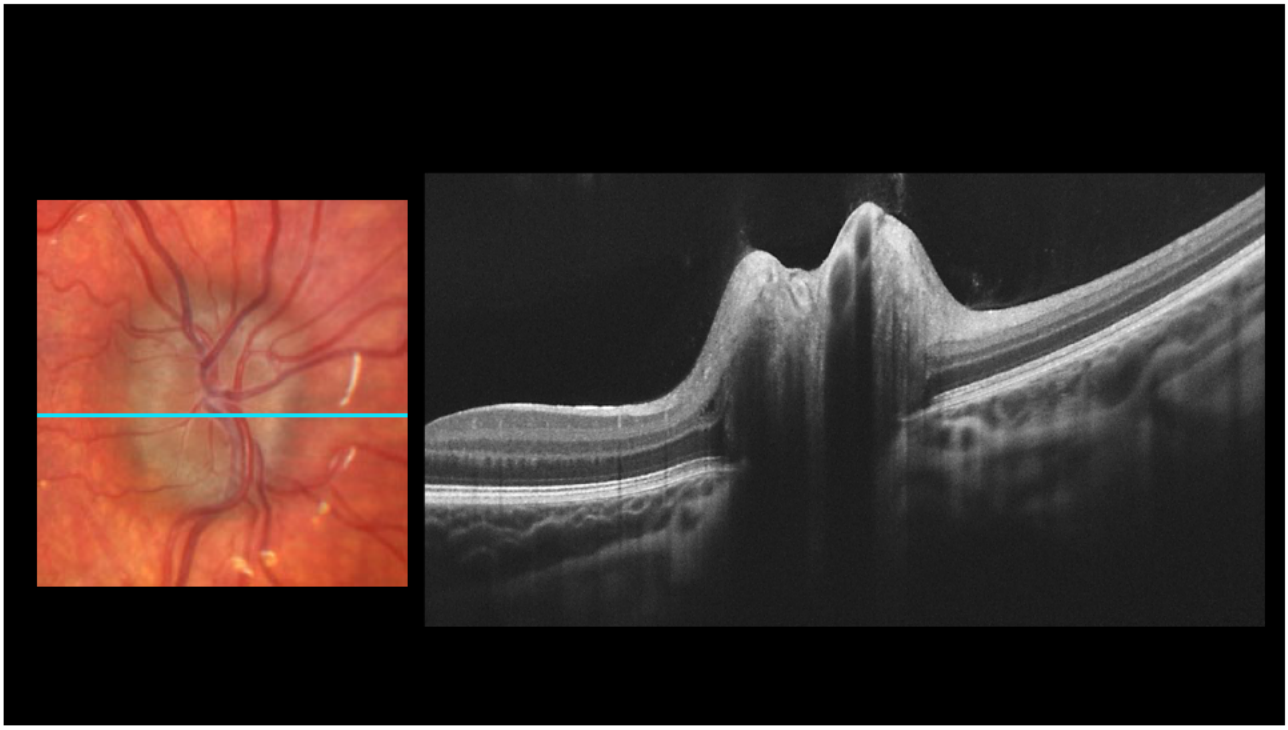
80


Are there features of TRUE papilledema present?
What extra finding should we specifically look for?

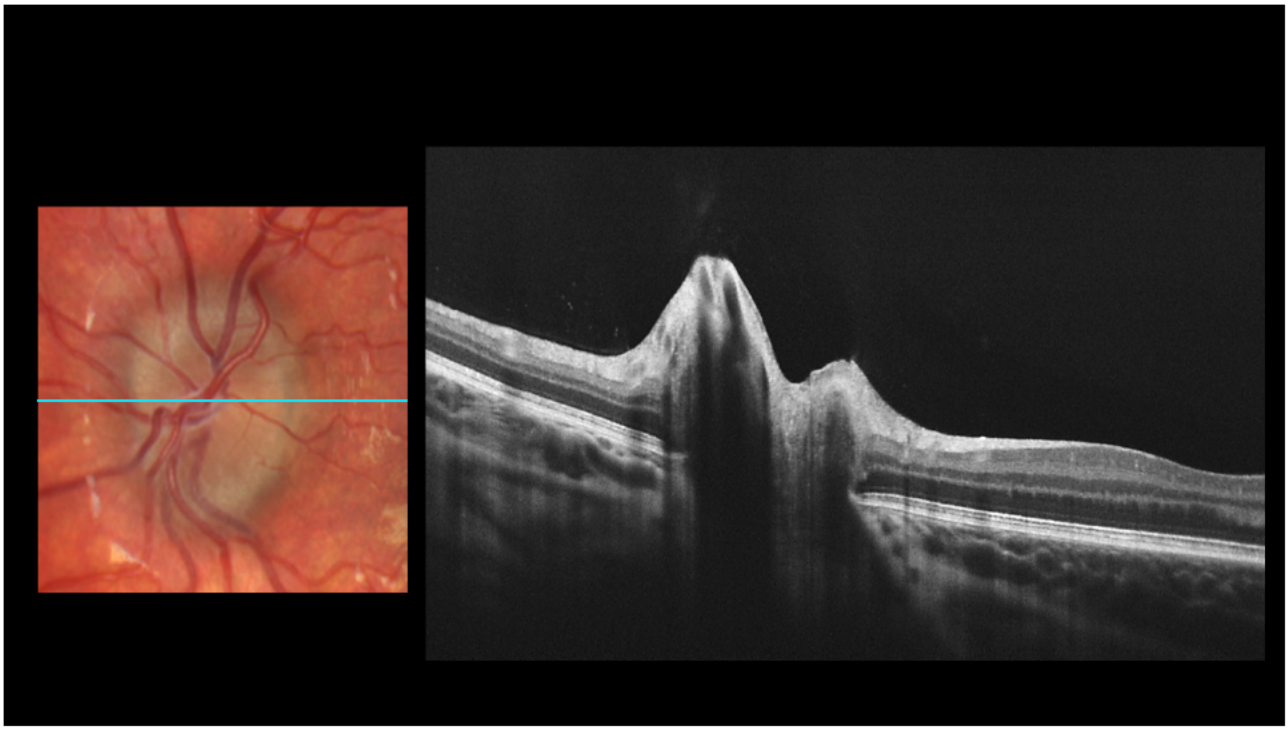
81


Technician: Operator, Cirrus
Signal Strength: 8/10 7/10
ONH and RNFL OU Analysis:Optic Disc Cube 200x200
OD
OS
RNFL Thickness Map
RNFL Deviation Map
Neuro-retinal Rim Thickness
RNFL Thickness
Extracted Horizontal Tomogram
Extracted Vertical Tomogram
RNFL Circular Tomogram
Normative data is not available. Patient age < 18.
RNFL Quadrants
RNFL Clock Hours

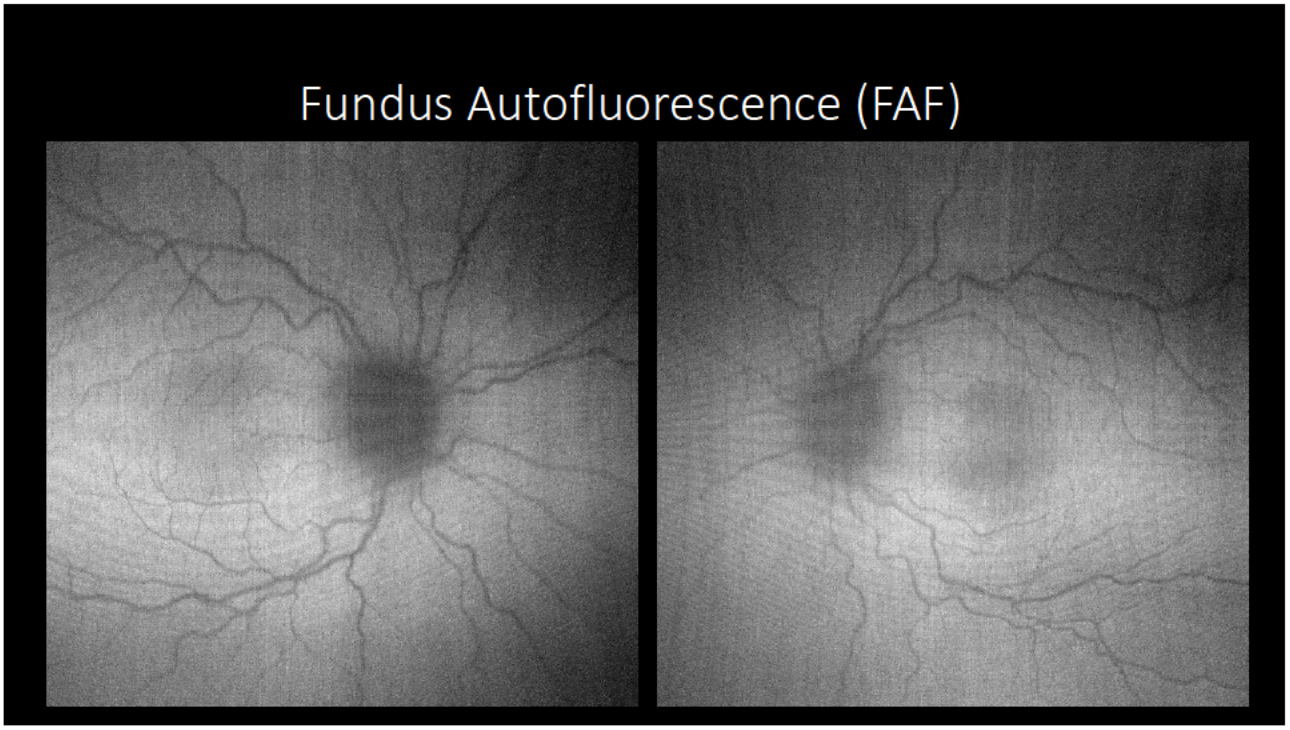
82



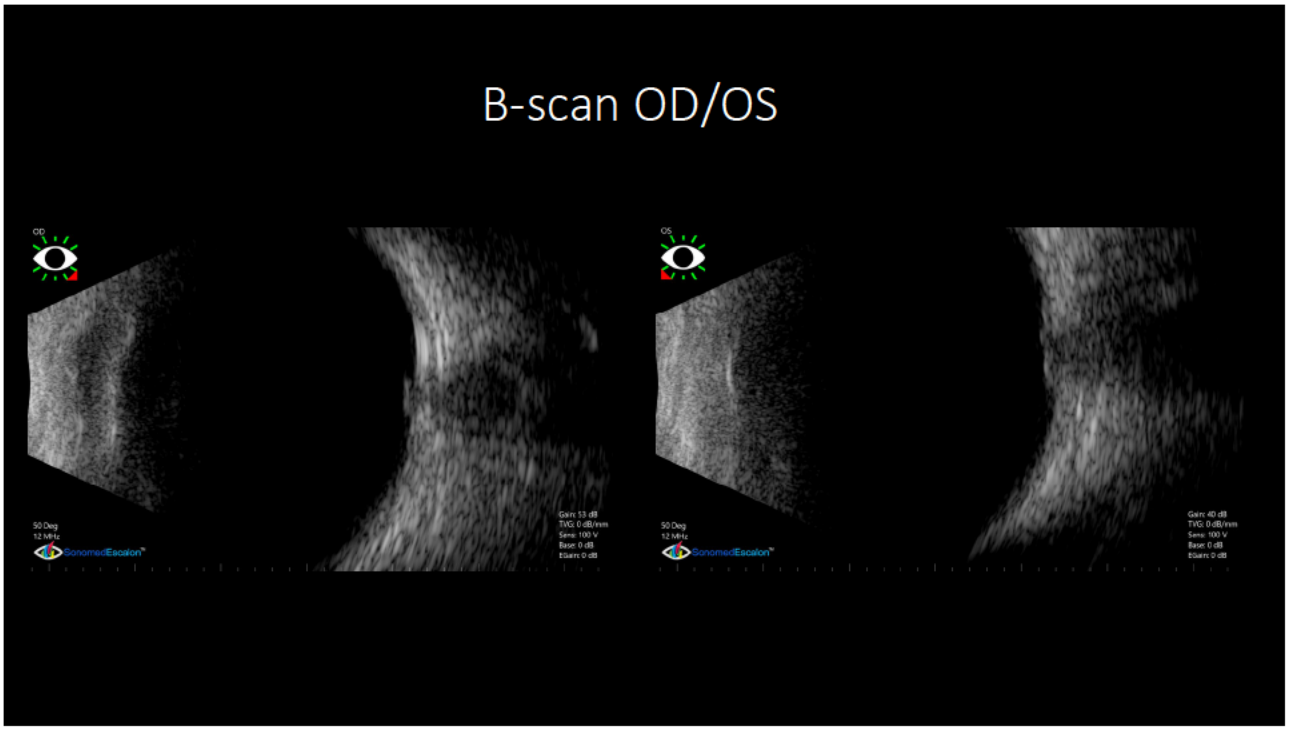
83



84

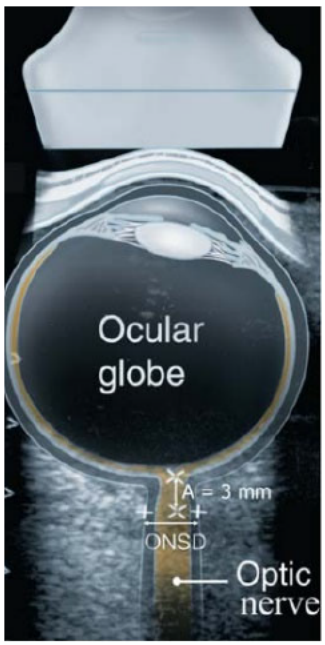

Fundus Autofluorescence (FAF)

85


B-scan OD/OS
OD
OS
50 Deg
12 MHz
SonomedEscalon
50 Deg
12 MHz
SonomedEscalon

86

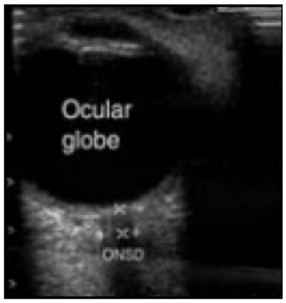
PAPILLEDEMA B-SCAN ONSD



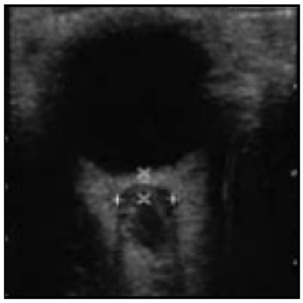
B-scan

- Optic nerve sheath diameter (ONSD) >5 mm is highly suspicious for papilledema
- Need to put into a clinical context

Normal

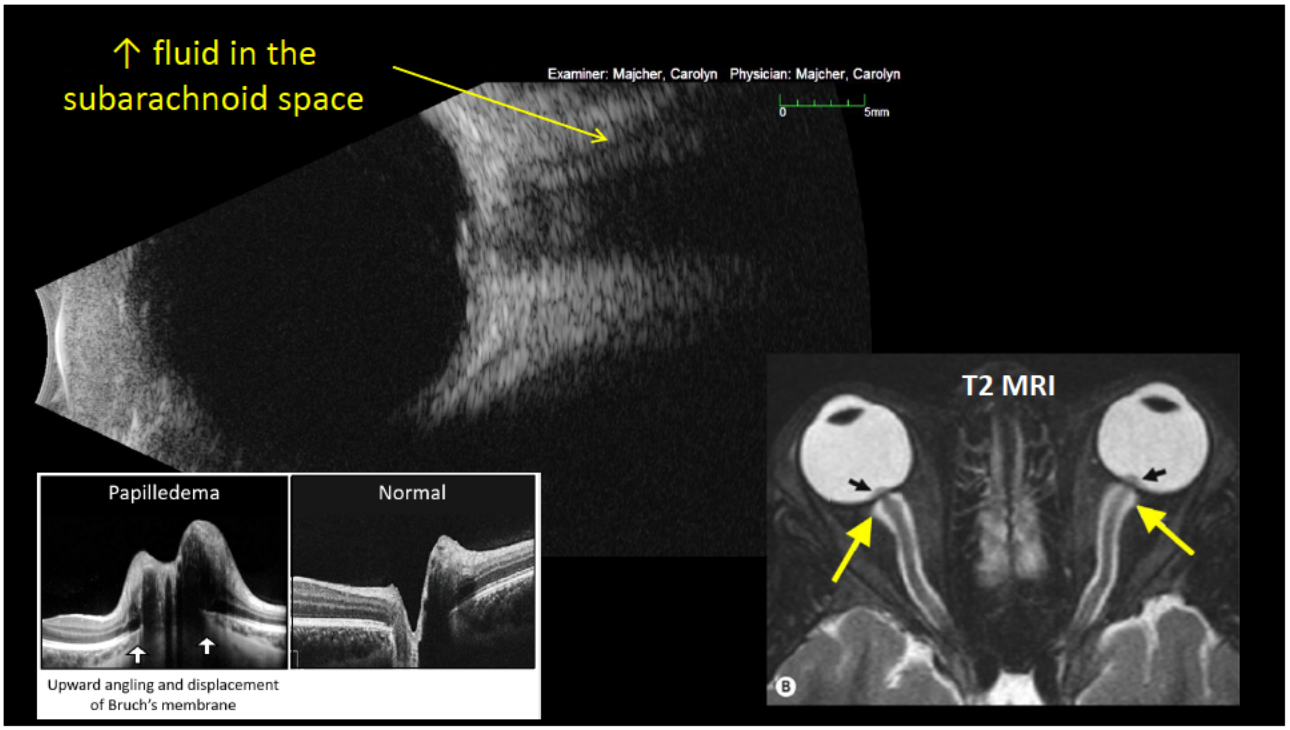


Papilledema

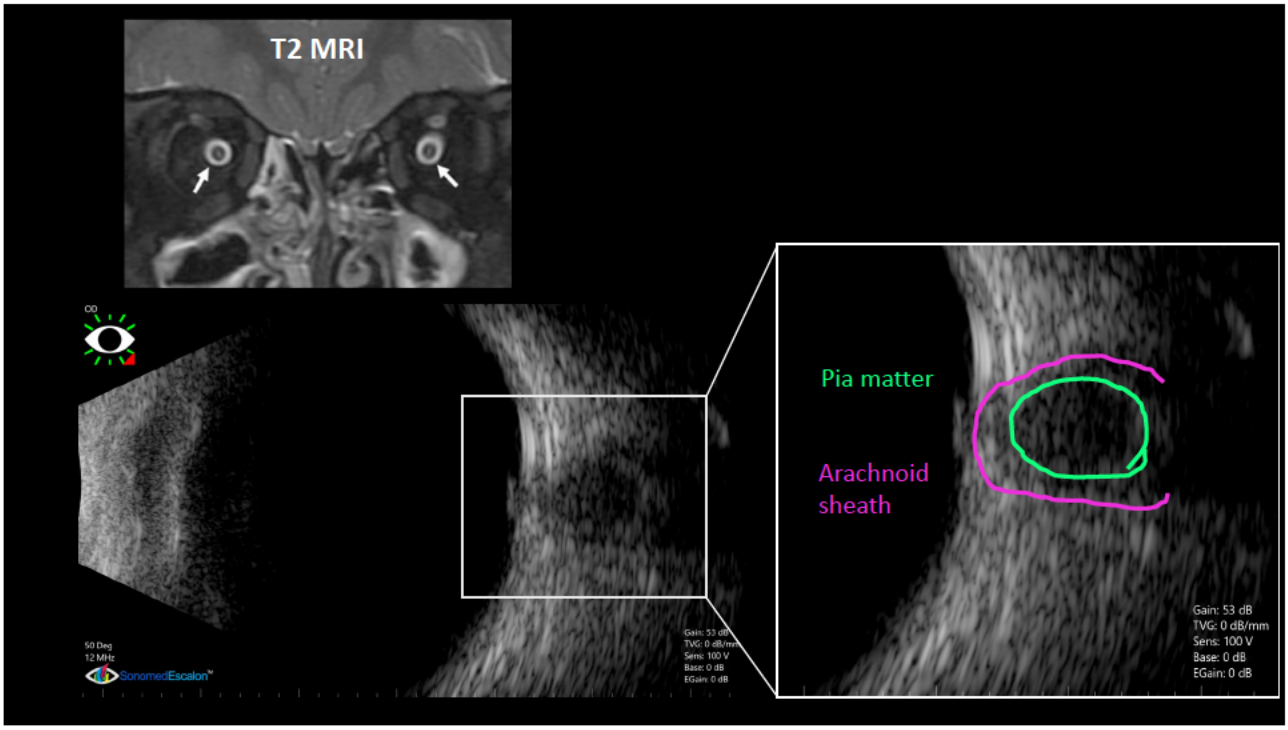


Blaivas M, et al. Elevated ICP detected by bedside emergency ultrasonography of the optic nerve sheath. Academic Emergency Medicine 2003;10:376-81

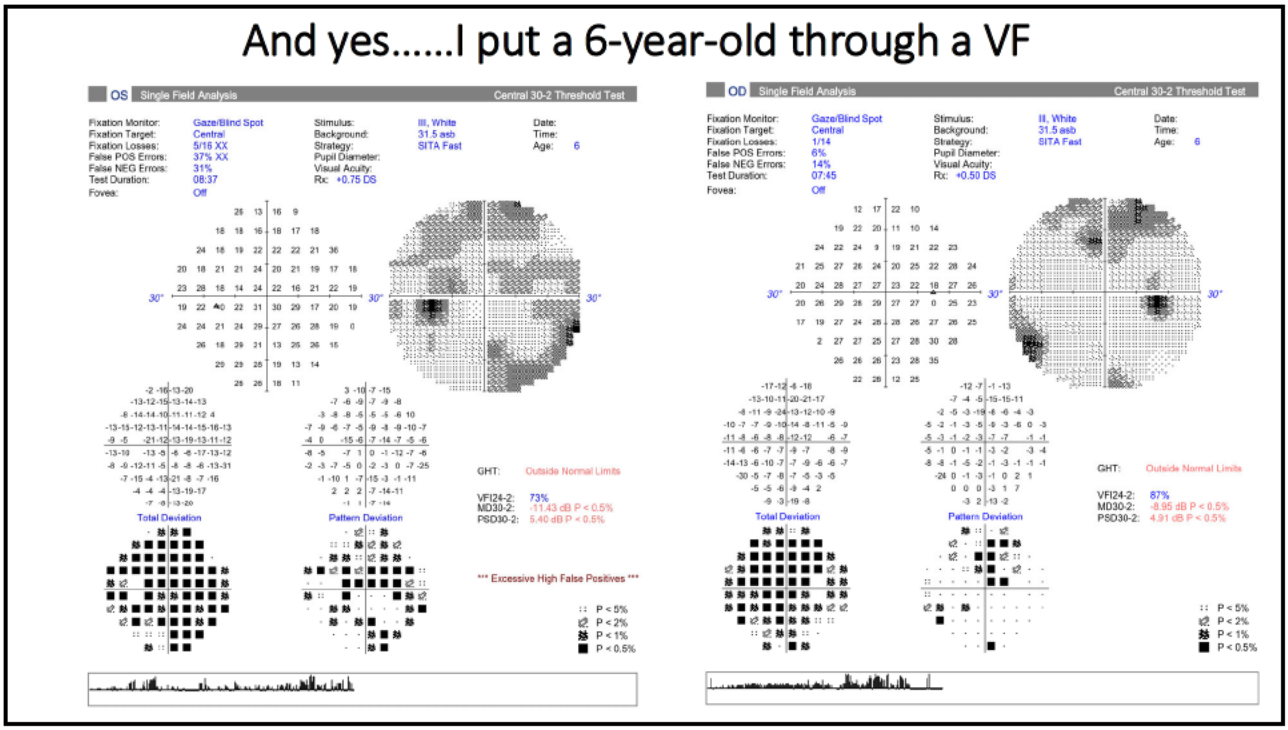
87



88


T2 MRI
OD
Pia matter
Arachnoid sheath
50 Deg
12 MHz
SonomedEscalon™
Gain: 53 dB
TVG: 0 dB/mm
Sens: 100 V
Base: 0 dB
EGain: 0 dB

89

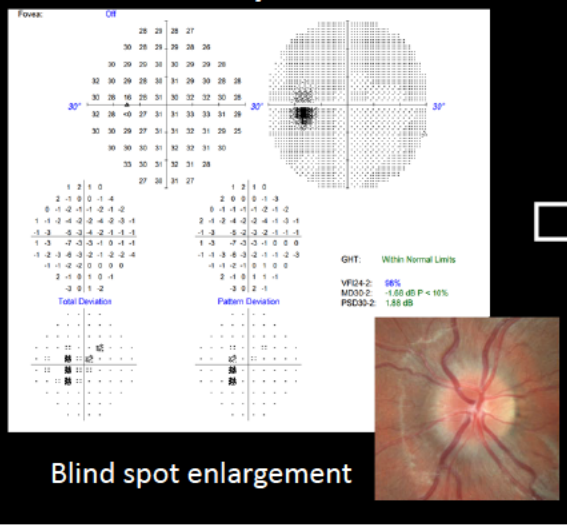

And yes……I put a 6-year-old through a VF
OS Single Field Analysis Central 30-2 Threshold Test
Fixation Monitor: Gaze/Blind Spot
Fixation Target: Central
Fixation Losses: 5/16 XX
False POS Errors: 37% XX
False NEG Errors: 31%
Test Duration: 08:37
Fovea: Off
Stimulus: III, White
Background: 31.5 asb
Strategy: SITA Fast
Rx: +0.75 DS
Date:
Time:
Age: 6
Total Deviation
Pattern Deviation
GHT: Outside Normal Limits
VFI24-2: 73%
MD30-2: -11.43 dB P < 0.5%
PSD30-2: 5.40 dB P < 0.5%
*** Excessive High False Positives ***
P < 5%
P < 2%
P < 1%
P < 0.5%
OD Single Field Analysis Central 30-2 Threshold Test
Fixation Monitor: Gaze/Blind Spot
Fixation Target: Central
Fixation Losses: 1/14
False POS Errors: 6%
False NEG Errors: 14%
Test Duration: 07:45
Fovea: Off
Stimulus: III, White
Background: 31.5 asb
Strategy: SITA Fast
Rx: +0.50 DS
Date:
Time:
Age: 6
Total Deviation
Pattern Deviation
GHT: Outside Normal Limits
VFI24-2: 87%
MD30-2: -8.95 dB P < 0.5%
PSD30-2: 4.91 dB P < 0.5%
P < 5%
P < 2%
P < 1%
P < 0.5%

90

PAPILLEDEMA – VF LOSS

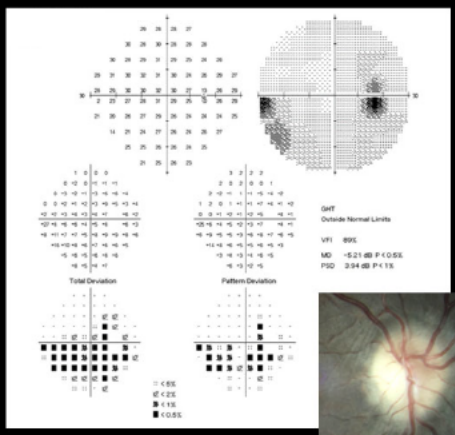
- **Poor correlation between VF loss/acuity and ON appearance**/degree of swelling

Early



Blind spot enlargement

Chronic



Localized NFL bundle defects & peripheral constriction

91

MRI/MRV brain and orbits with and without contrast

MRI Head

- **Mild concavity to the superior margin of the pituitary gland (AKA: partially empty sella)**...otherwise no intracranial space-occupying lesion and no evidence of acute hydrocephalus.

MRV Head

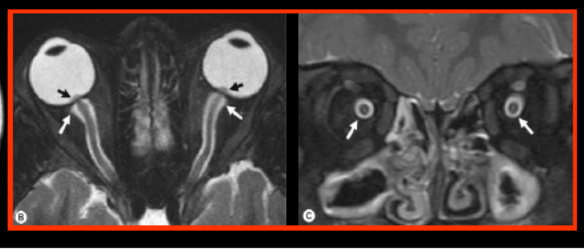
- The **left transverse sinus appears to be significantly hypoplastic**. Rt transverse sinus is dominant. No evidence of dural venous sinus thrombosis.

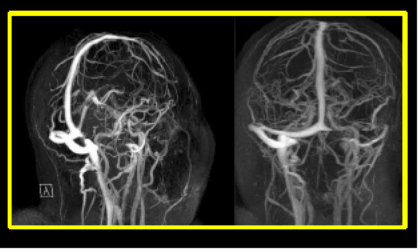
MRI Orbits

- Bilateral optic disc bulge and **increased nerve sheath CSF** (distended).

IMPRESSIONS

- <u>Findings suggestive of raised intracranial pressure</u>!





92

Lumbar Puncture

Opening Pressure
- Elevated at **288 mm of H2O**

CSF Analysis/cytology
- Glucose, lymphocytes, monocytes, protein and macrophages **all WNL**. No atypical or malignant cells identified. VDRL non-reactive and cryptococcus antigen negative.

ASSESSMENT
- **Pediatric IIH**

PLAN
- **Acetazolamide 9.4mL (235mg total) by mouth every 12 hours**

93

Papilledema Causes

Obstructed intracranial venous drainage	Increased intracranial volume	Infectious/inflammatory	Endocrine disorders
• Venous sinus thrombosis • Cerebral or spinal AV malformation/fistula • Chiari malformation • Hydrocephalus • Superior vena cava syndrome	• Intracranial or spinal cord mass lesions • Intracranial hemorrhage • Foster Kennedy syndrome	• Syphilis • Meningitis • Lupus • Ear infections	• Addison's disease • Hypoparathyroidism
Toxic/pharmacologic	**Medical conditions**	**Others**	
• Tetracycline, minocycline, doxycycline • Vitamin A hypervitaminosis • Accutane • Corticosteroid withdrawal	• Malignant hypertension • Hypercoagulable states • Anemia • Obstructive sleep apnea • Renal failure/transplantation • Pulmonary disease/respiratory • insufficiency • Guillain–Barré syndrome • Craniosynostosis	• Trauma • Idiopathic intracranial HTN	

94

Papilledema

Work up

Papilledema is a medical emergency

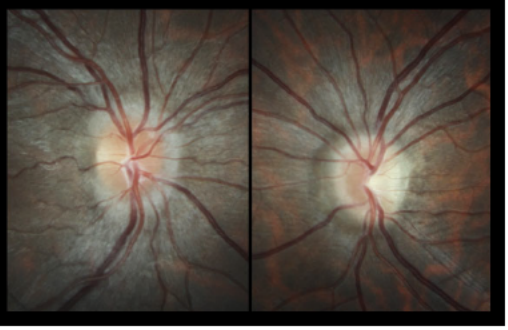
- **Full neurological examination, BP measurement**
- **MRI/MRV of brain (and orbits) with and w/o contrast**
  - Include spinal code if paraparesis or quadriparesis, lumbar pain, a sensory level, hyperreflexia, extensor plantar responses, or bladder dysfunction are present
  - Include MRA if AV malformation suspected
  - CT if hemorrhage suspected
- If other etiologies suspected:
  - ACE, tick antibody panel, ESR, CBC, RPR, FTA-abs, ANA, acid-fast staining, cytology for malignant cells
- **If imaging reveals no mass, no chiari, and ventricles not dilated proceed with LP**
  - **LP with opening pressure measurement & CSF analysis**

95

IDIOPATHIC INTRACRANIAL HYPERTENSION (IIH)

IIH Diagnostic criteria

- Awake and alert
- Signs and symptoms of increased ICP (HA, N & V, TVO, papilledema, diplopia)
- **Normal neurologic examination** (absence of localized findings aside from those expected from increased ICP such as CN 6 palsy)
- **Normal MRI/MRV neuroimaging** (normal brain parenchyma, ventricular system, and cerebral venous sinuses w/o signs of hydrocephalus, mass or structural defect, and without meningeal enhancement)
- **Elevated LP opening pressure (typically ≥250 mm H_2O in adults** lateral decub position)
- **Normal CSF composition**
- No other cause of increased ICP present (meds, etc)



96

IDIOPATHIC INTRACRANIAL HYPERTENSION (IIH)

Management

- Diagnosis and **co-management by neurologist or neuro-ophthalmologist**
- **Serial ocular exams (color, VFs, OCT, DFE)**
  - weekly or biweekly initially until vision stabilizes or improves
- **Weight loss (10%)**- nonpregnant overweight patients
  - Topiramate, bariatric surgeries, nutrition counseling
- **Acetazolamide**
  - **First line, decreases CSF production**
  - 500mg PO bid to tid initially
- **Topiramate- CAI, HA control, appetite suppression**
- HA management-analgesics
- Severe progressive cases, medical failure:
  - Optic nerve sheath decompression
  - Lumboperitoneal /ventriculoperitoneal shunt

97

Pediatric Idiopathic Intracranial Hypertension (IIH)

Pediatric IIH

- May be a different underlying mechanism
- 50% males (prepubertal pediatric patients)
- Affected adolescents tend to be overweight, but obesity and weight gain are not associated risk factors in patients younger than 11 years
- Presentation S/S similar to adults except CN VI palsy more common (33%)
- Most cases improve with medical treatment

Liu G, et al. Pediatric IIH. Surv Ophthalmol. Nov-Dec 2007;52(6):597-617.

98

UNDER PRESSURE

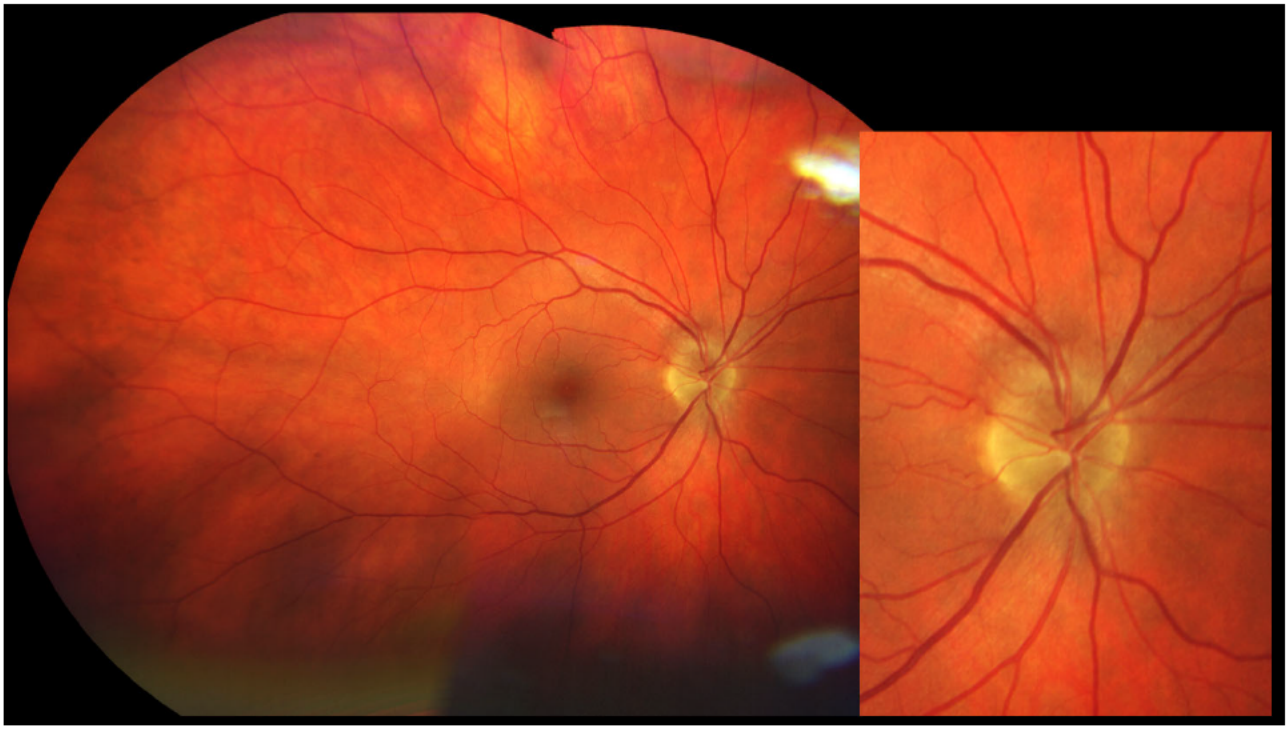
- 58 yo M – sent from PC to optometry for abnormal findings upon DM CFP screening
- CC: occasional halos around objects x last few days, pressure sensation around eyes OU, slight HA 2-3 days ago, new onset; wears hearing aids, new noise/swooshing in ears worse left ear x 2-3 weeks; denies transient visual obscurations w/w/out postural changes
- Medical history: HTN, DM type 2, hypercholesterolemia, hearing loss
- Medications: alogliptin, amlodipine, aspirin, atorvastatin, canagliflozin, lisinopril, metformin, pioglitazone, tamsulosin

99

Examination Findings

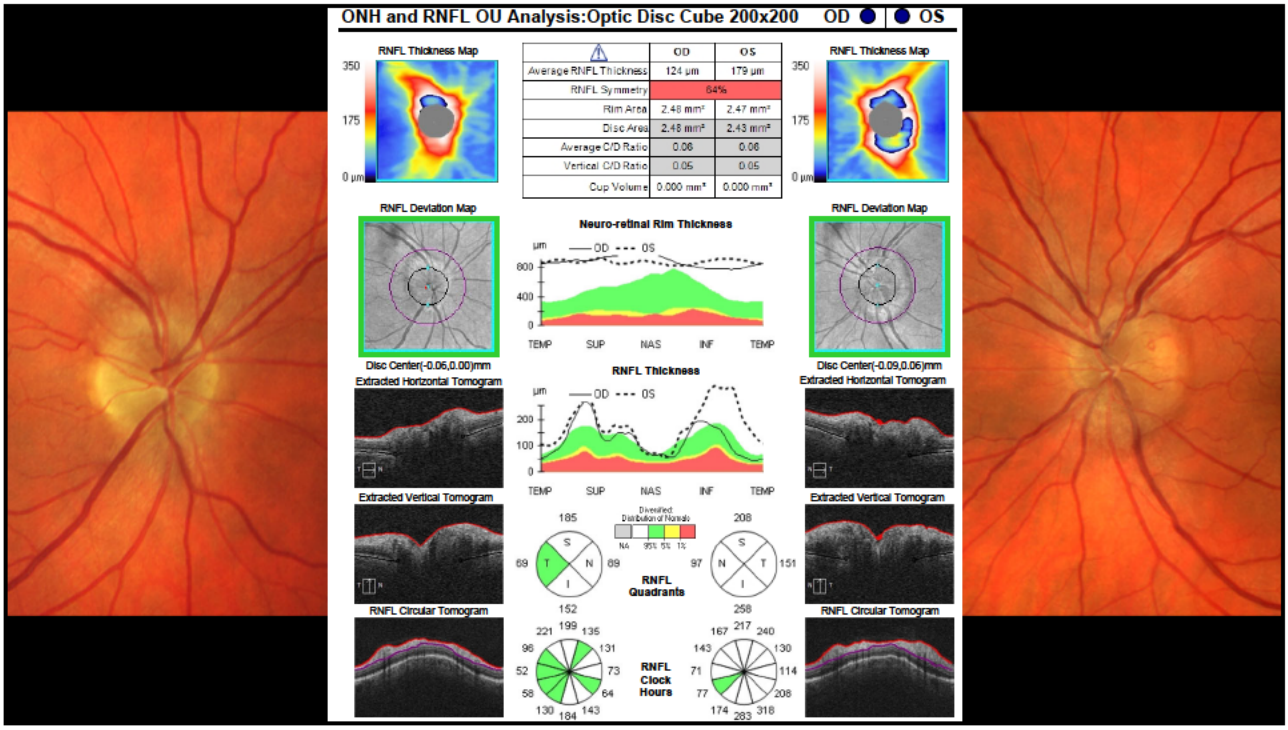
Assessment	OD	OS
Unaided VA	20/20	20/20
EOM	Normal	Normal
Pupils	PERRL OU, (-) APD	
IOP (GAT)	21 mmHg	22 mmHg
Anterior segment	Normal	Normal
BP	144/88	
A1c	9.0%	

100



101

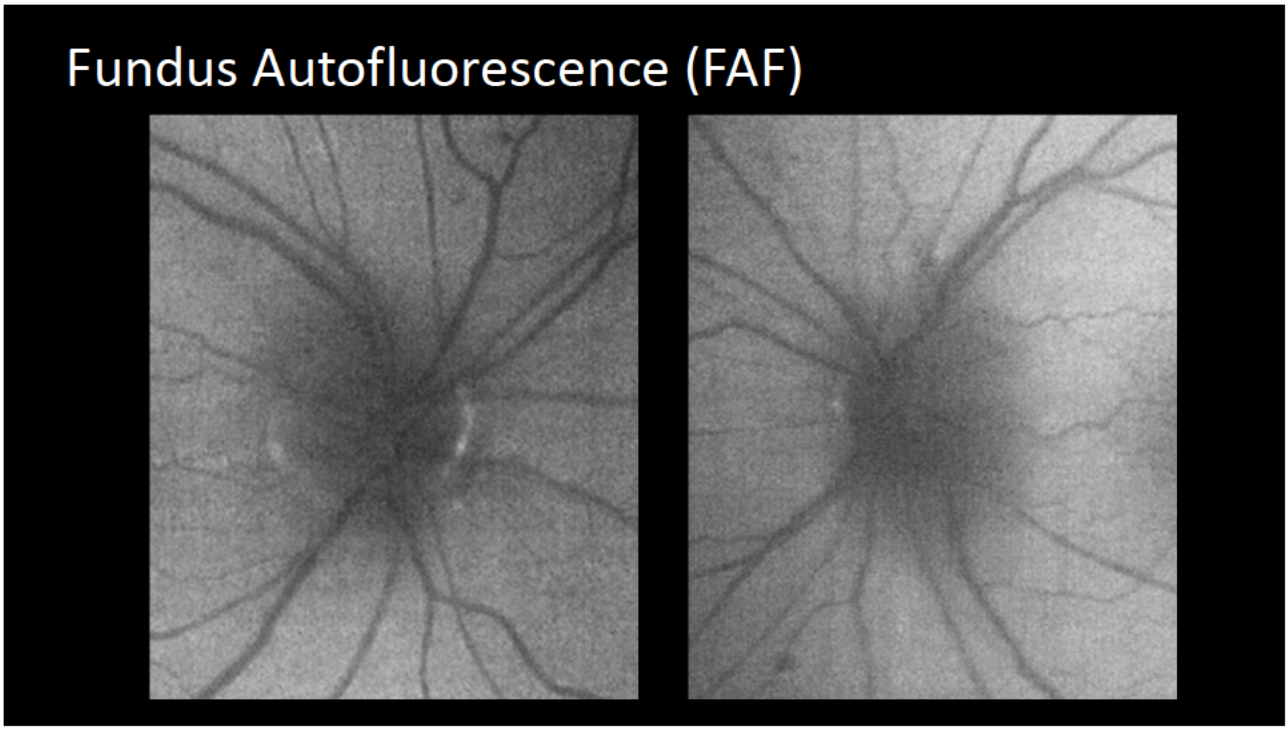
102



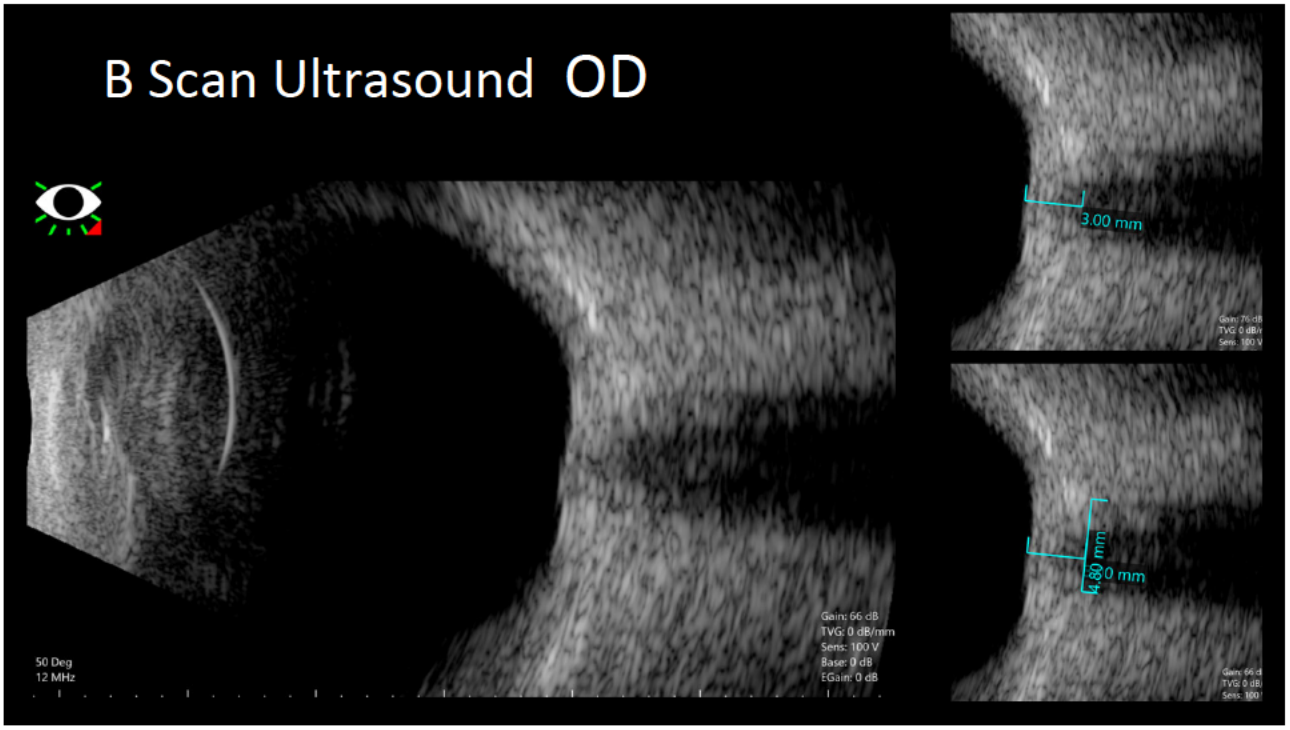
	OD	OS
Average RNFL Thickness	124 µm	179 µm
RNFL Symmetry	64%	
Rim Area	2.48 mm²	2.47 mm²
Disc Area	2.48 mm²	2.43 mm²
Average C/D Ratio	0.06	0.06
Vertical C/D Ratio	0.05	0.05
Cup Volume	0.000 mm³	0.000 mm³

103

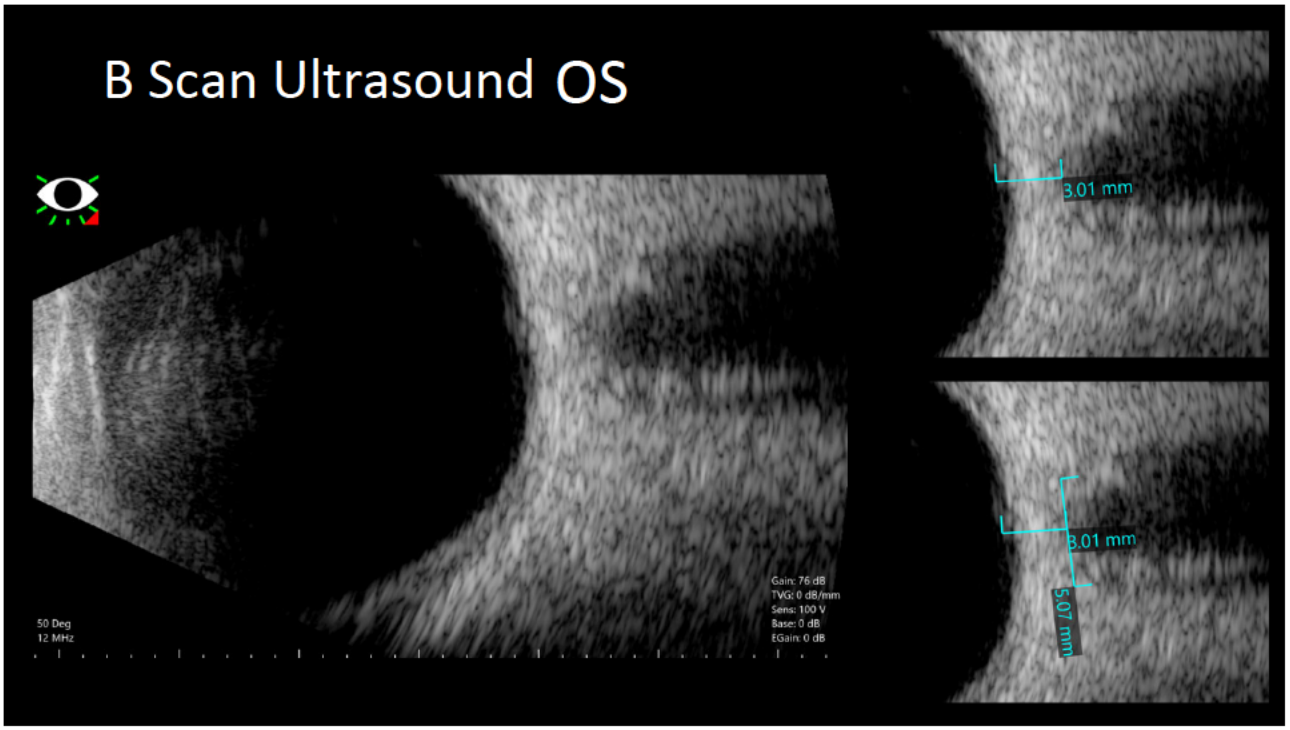
Fundus Autofluorescence (FAF)



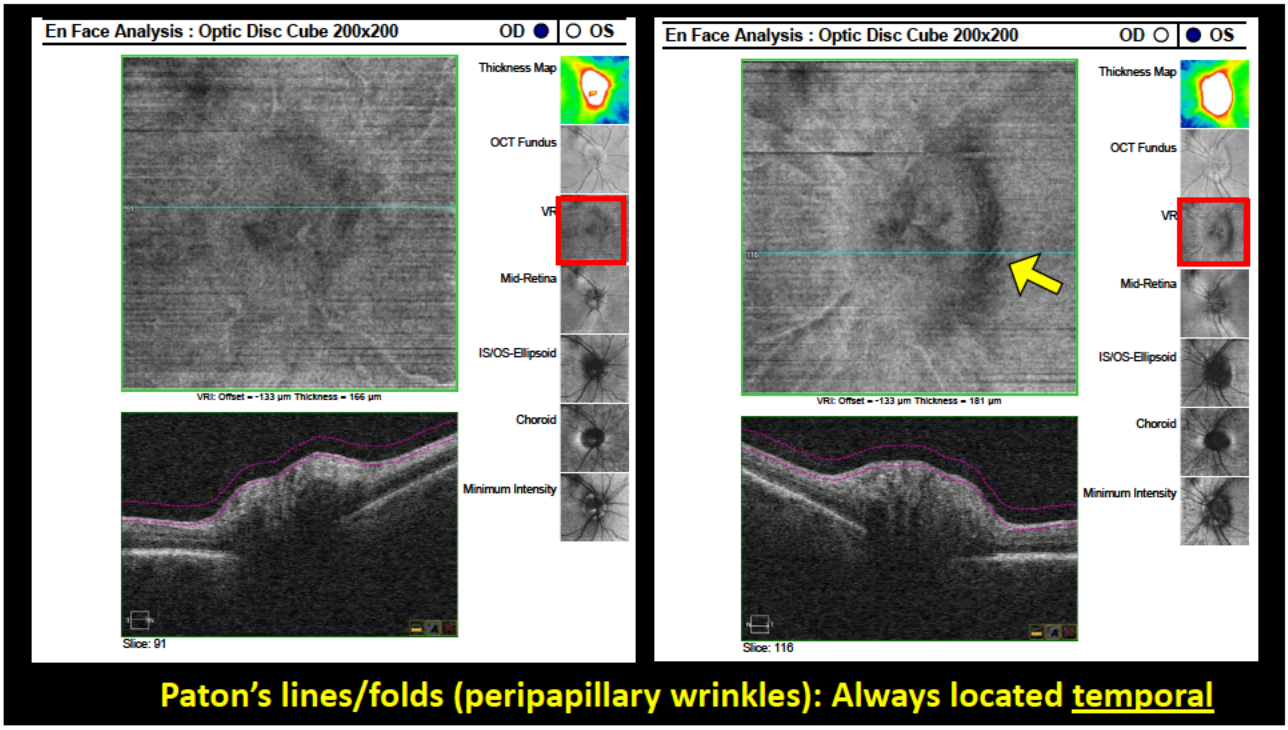
104


B Scan Ultrasound OD
3.00 mm
4.80 mm
50 Deg
12 MHz
Gain: 66 dB
TVG: 0 dB/mm
Sens: 100 V
Base: 0 dB
EGain: 0 dB

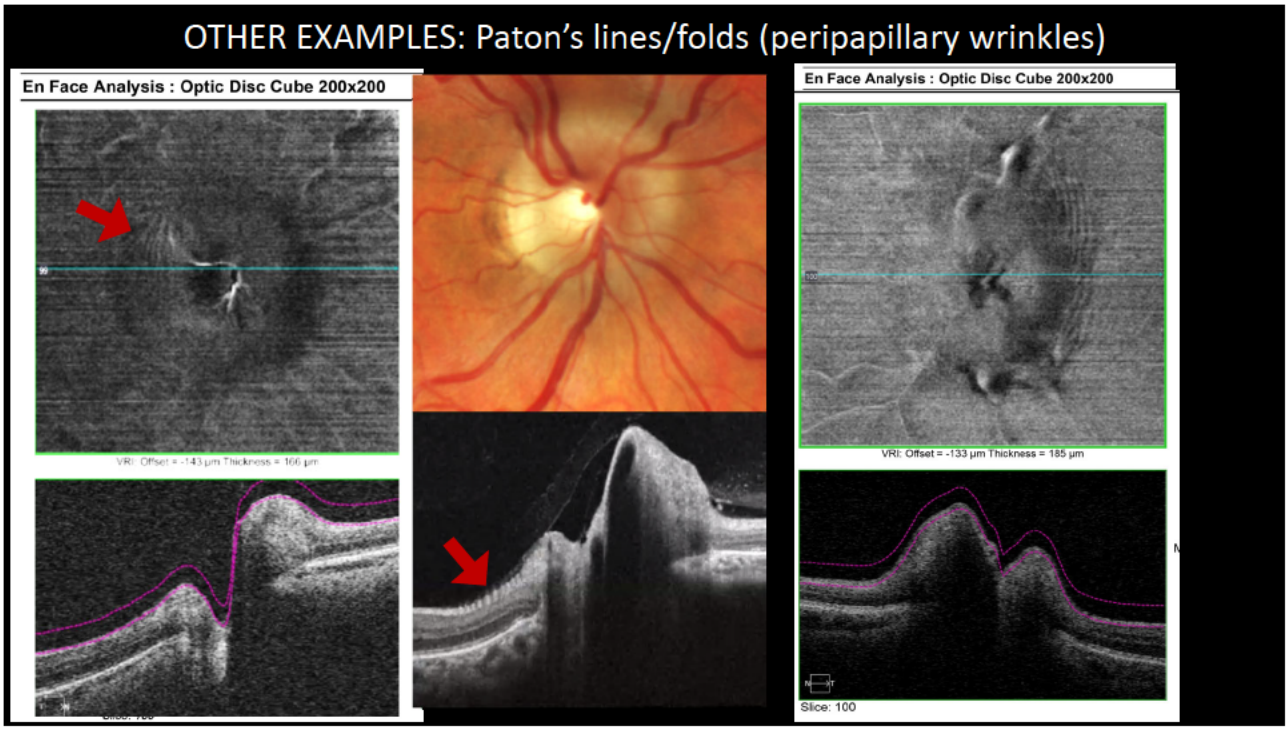
105


B Scan Ultrasound OS
3.01 mm
3.01 mm
5.07 mm
50 Deg
12 MHz
Gain: 76 dB
TVG: 0 dB/mm
Sens: 100 V
Base: 0 dB
EGain: 0 dB

106


En Face Analysis : Optic Disc Cube 200x200
OD
OS
Thickness Map
OCT Fundus
VR
Mid-Retina
IS/OS-Ellipsoid
Choroid
Minimum Intensity
VRI: Offset = -133 µm Thickness = 166 µm
Slice: 91
En Face Analysis : Optic Disc Cube 200x200
OD
OS
Thickness Map
OCT Fundus
VR
Mid-Retina
IS/OS-Ellipsoid
Choroid
Minimum Intensity
VRI: Offset = -133 µm Thickness = 181 µm
Slice: 116
Paton's lines/folds (peripapillary wrinkles): Always located temporal

107


OTHER EXAMPLES: Paton's lines/folds (peripapillary wrinkles)
En Face Analysis : Optic Disc Cube 200x200
En Face Analysis : Optic Disc Cube 200x200
VRI: Offset = -133 µm Thickness = 185 µm
Slice: 100

108

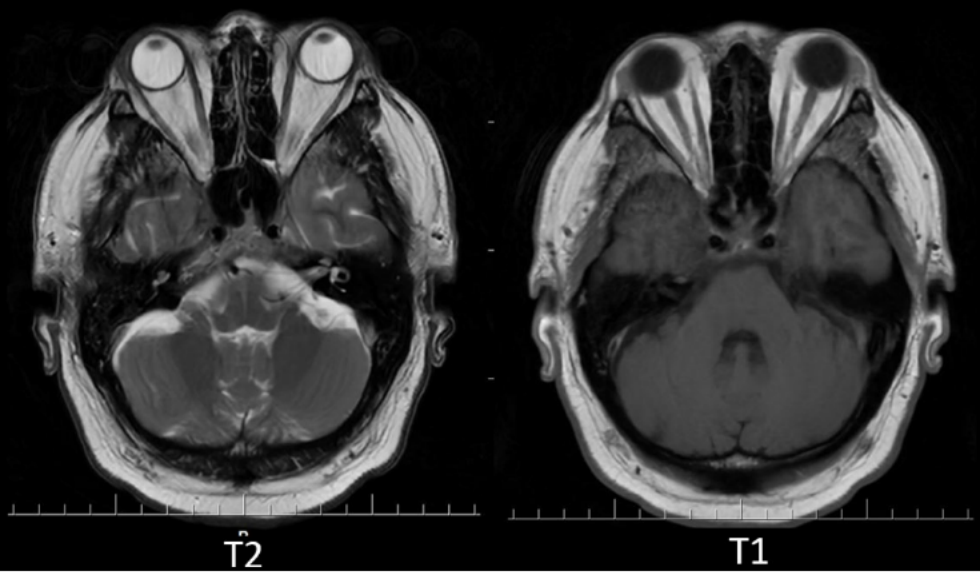
Management

Dx: papilledema OU

1. Referred to ER for emergency **MRI and MRV** of brain/orbits with and without contrast

109

MRI Results

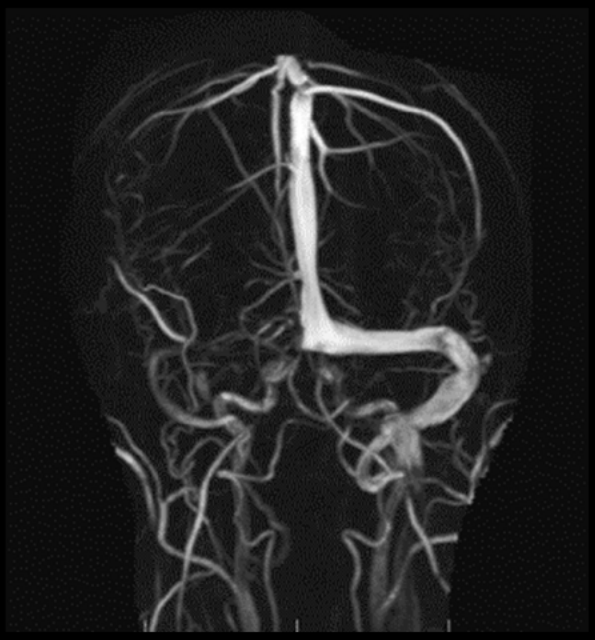


Normal MRI of brain and orbits!

110

MRV Results

Thrombosis of the right transverse sinus, sigmoid sinus, and internal jugular vein



111

Management

Dx: papilledema secondary to dural venous sinus thrombosis

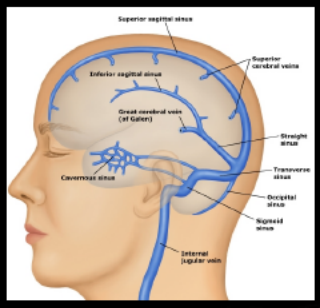
1. US venous doppler of lower extremities
2. Blood work (hypercoagulable panel)
3. Echocardiogram
4. Electrocardiogram
5. Started on Lovenox
6. **Rx oral apixaban 5 mg**

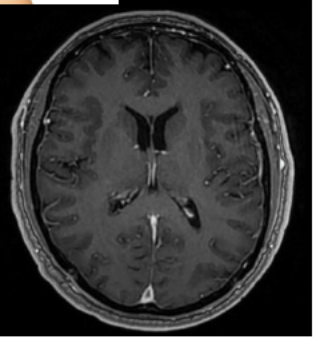
112

DURAL VENOUS SINUS THROMBOSIS (DVST)

- Thrombosis or obstruction of the cerebral dural venous sinuses
  - Venous hypertension and decreased CSF absorption
- Causes of clot formation- **Hypercoagulable states,** Contiguous infection (lateral sinus thrombosis secondary to mastoiditis), Contiguous neoplasm (may compress the venous sinuses, impeding venous drainage and leading to clot formation)
- Presentation – Similar to IIH but usually develop more rapidly
- EMERGENCY due to risk of stroke

RECOMMEND MRV ALONG WITH MRI IN PAPILLEDEMA WORK-UP!!





113

Relevance

Stroke
Volume 35, Issue 3, 1 March 2004; Pages 664-670
https://doi.org/10.1161/01.STR.0000117571.76197.26
American Heart Association.

ORIGINAL CONTRIBUTIONS

Prognosis of Cerebral Vein and Dural Sinus Thrombosis

Results of the International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT)

José M. Ferro, MD, PhD, Patrícia Canhão, MD, Jan Stam, MD, Marie-Germaine Bousser, MD, Fernando Barinagarrementeria, MD, and for the ISCVT Investigators

- Cerebral venous thrombosis represents 0.5-1% of all strokes
- Affects ~5 people per 1 million annually
- International Study on Cerebral Venous and Dural Sinuses Thrombosis (ISCVT)
  - 78% of cases occurred in patients <50 years of age
  - 8% death rate
  - Risk factors: male, >37 years of age, coma, mental status disorder, intracranial hemorrhage, thrombosis of the deep cerebral venous system, CNS infection, cancer
  - >80% of patients were anticoagulated with heparin

114


Thank you!
Carolyn Majcher, OD, FAAO, FORS
• majcher@nsuok.edu

115


Thank you!
Carolyn Majcher, OD, FAAO, FORS
• majcher@nsuok.edu

116