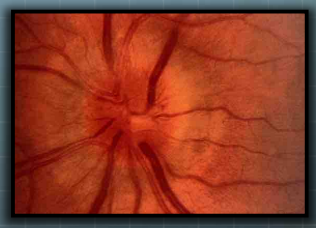


# Optic Neuropathies
## House Calls for Sick Discs

Mark Sawamura, OD, FAAO
Southern California College of Optometry
Monterey Symposium, 2018
msawamura@ketchum.edu

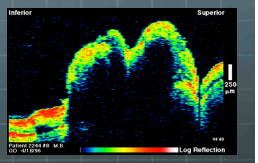
*I have no financial disclosures

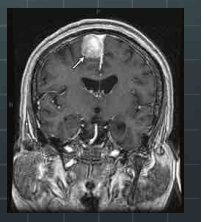
# Objectives

- Briefly discuss the general evaluation of optic disc
  - Dysfunction
  - Diagnostic testing
- Differential Diagnoses
- Optic Neuropathies
  - 3 to focus on
    - Optic Neuritis
    - Papilledema
    - AION
      - New thoughts and how OCT may be helpful

# Evaluation of the Swollen Disc

- VAs
- Pupils
- EOMs
- Color Vision
- Blood pressure
- Visual Fields
- Dilated Fundus Exam
- Fluorescein Angiography?
- OCT of the RNFL and Optic discs
- Neuroimaging of visual pathway, orbit, and brain

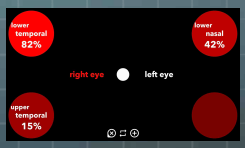




# How to assess the Optic Nerves

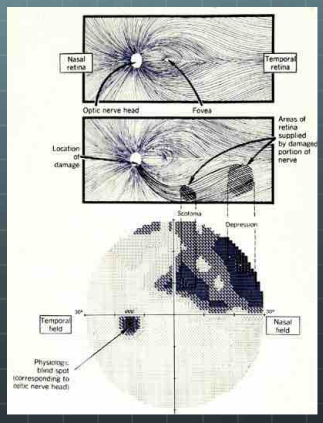
- Visual acuity: Variable dysfunction
  - Quantitative vs. Qualitative
- Pupil function: Look for an afferent defect
- Color vision: Red green color vision loss /desaturation
  - Red cap testing/Brightness sense
- Visual fields:
  - Kinetic vs. Static
  - Threshold perimetry is the preferred method
  - Test both eyes!
  - Does it respect the horizontal or vertical midlines???
- Contrast sensitivity
- VEP
- OCT: RNFL and Optic Discs




Smart Optometry App

# Visual Field characteristics

- Patterns
  - Paracentral scotoma
  - Nasal Step
  - Enlarged blindspot
  - Central-cecal scotoma
  - Bitemporal
  - Altitudinal defects
- Midlines
  - Vertical:
    - Chiasm and beyond
  - Horizontal:
    - Glaucoma, Disc Drusen, Papilledema



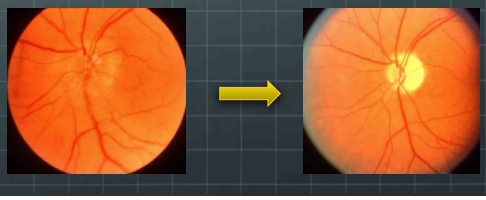



# OCT and Optic Neuropathy

- RNFL loss has been documented in all optic neuropathies.
  - May see thickening or thinning of RNFL based on stage of presentation
  - Can be used to track resolution
  - May be predictive of visual outcomes
  - Can be useful in differential diagnoses
- Ganglion cell/ Macular scans
  - Some optic neuropathies can involve the papillomacular bundle
  - Some present with sub-retinal fluid

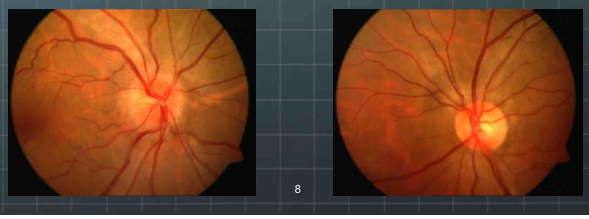
# Differential Diagnoses of Optic Neuropathies

- Disc Edema vs Optic Atrophy
  - Atrophy can be a sign of chronic compression by neoplastic process
  - Atrophy follows injury to the optic disc
    - AION, Traumatic optic neuropathy, Optic neuritis
    - Nutritional optic neuropathy
    - Hereditary optic neuropathy



| Unilateral * | Bilateral |
|---|---|
| Papillitis | Hypertension |
| AION | Papilledema |
| Diabetic Papillopathy | Pseudotumor |
| Compressive/ Sarcoid | Infections |
| CRVO | Leber's (separated in time) |
| Fistulas | Toxic |
| Neuroretinitis | Infilltrative |

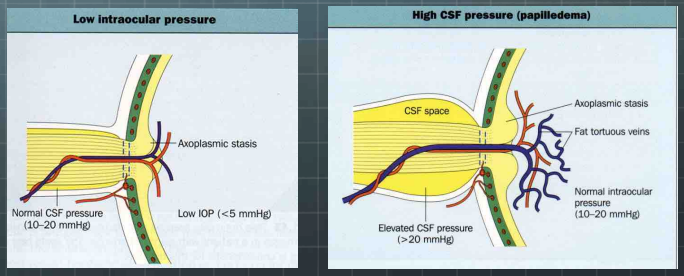
* More likely to be unilateral than bilateral





# Papilledema

- Increased intracranial pressure leads to swelling of the optic nerve head
- Disc swelling can occur due to translaminar gradient (CSF pressure > IOP)

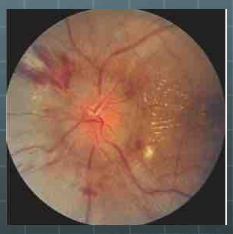



# Disc Edema by Age

Young

- Neuroretinis
- Optic Neuritis
- Pseudotumor Cerebri
- Malignant hypertension
- Anterior Ischemic Optic Neuropathy
- Giant Cell Arteritis

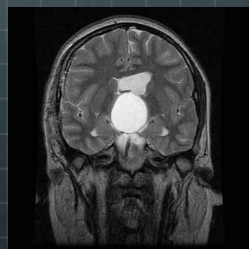
Older





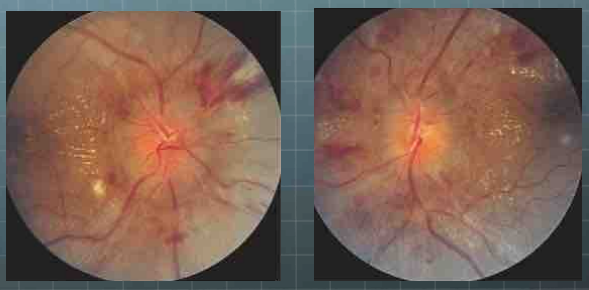
# Causes of Papilledema

- Hydrocephalus
- Intracranial Mass
- Increased Venous pressure
- Cerebral Venous Thrombosis
- Meningeal Disorder
- Idiopathic
- Increased ICP production
- Malignant Hypertension





# Malignant Hypertension



## Idiopathic Intracranial Hypertension (IIH)

- Often used interchangeably with pseudotumor cerebri
  - First reported in 1897
- Incidence: .9- 2/100,000 persons
  - 3.5/100,000 in women 15-44 yrs old
  - Mean age of diagnosis is 30
  - Obesity: 10 fold risk factor
    - NORDIC study: Ave BMI of 39
  - Associated with rapid weight gain
  - Men: Consider sleep apnea or malignant HTN

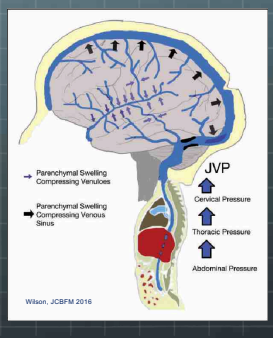
Wall,2014

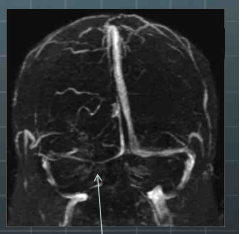
## Modified Dandy Criteria for IIH

- Signs and symptoms of increased intracranial pressure
- No neurological deficits except abducens palsy
- The patient is awake and alert
- Normal MRI and MRV (no venous sinus thrombosis)
- Increased ICP but normal CSF composition
  - Greater than 250 mm H20
- No secondary causes

## Pathophysiology of IIH

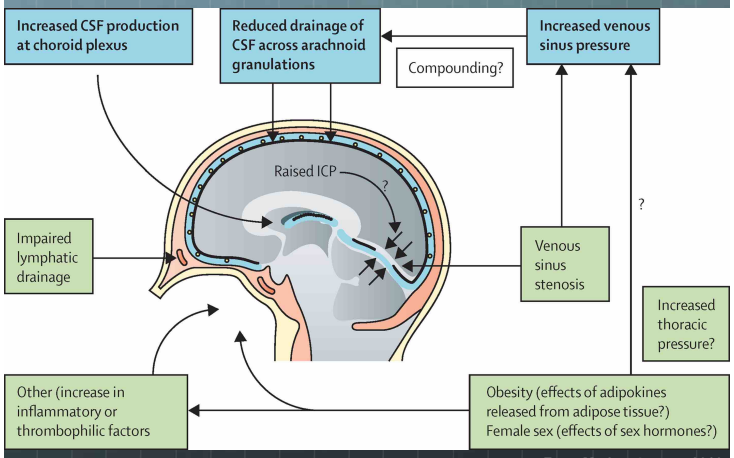
- Adipose tissue in the intrathoracic cavity reduces venous outflow from the brain
- Dural Venous sinus stenosis: 90% of cases



Narrowed Transverse Sinus on MRV

## Mechanisms of IIH




From Markey, Lancet, 2016

## Secondary Causes of Papilledema

- Drugs: Tetracycline, lithium, retinoids, growth hormone, steroids
- Vitamin A
- Iron deficiency
- Uremia
- Endocrine disorders
- Sleep apnea
- Oral contraceptives

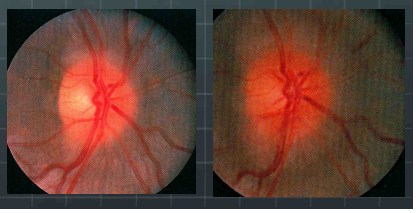


## Signs and Symptoms of Papilledema

- Headaches (94%)
  - Continuous, pulsatile headache
  - May awaken patient
  - Increases with Valsalva
  - Neck and shoulder pain
- Transient Visual obscuration (68%)
  - Seconds up to 1 minute
  - "Rheostat"
- Pulse synchronous Tinnitus (58%)
- Retrobulbar pain (44%) on eye movement
- Abducens paresis (38%)
- Nausea and Vomiting

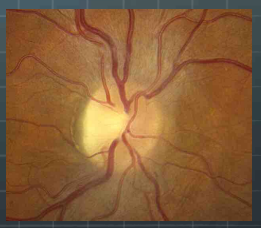
## How quickly does Papilledema develop?

- 1-7 days following rise in CSF pressure
  - Speed of ICP rise is important
    - Sub-arachnoid heme vs.Concussion
    - Brain tumor
- Look at the "poles"



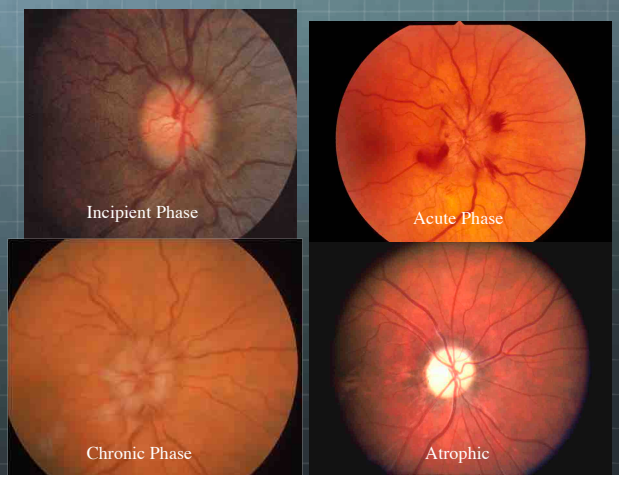
## The Stages of Papilledema

- Incipient (early):
  - Mild hyperemia, loss of SVP, blurred margins at 12 and 6 oclock
- Acute:
  - Blurred, elevated disc margins, NFL
  - Disc hyperemia, loss of SVP
  - Hemorrhages
  - Paton's lines
- Chronic:
  - Less hyperemia, pseudodrusen
- Atrophic:
  - Pale optic disc

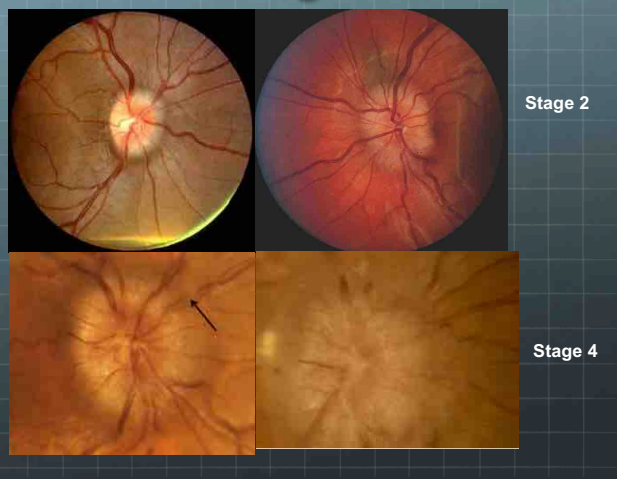




## Stages of Papilledema




## Frisen Grading criteria

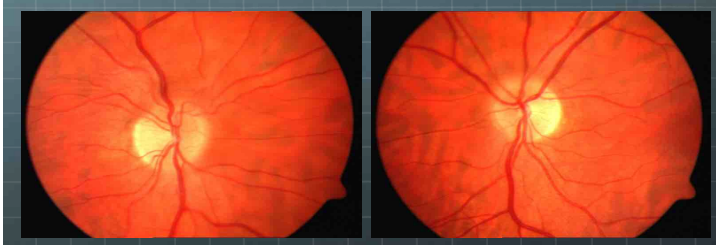



## "I need to sing in the Xmas pageant"

- 39 year old WF with headaches x 4 months
  - Tinnitus, transient loss of vision
  - VA's: 20/20, 20/20

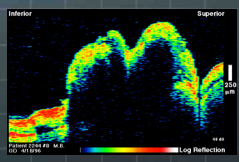
## Sang in the show, got an MRI!

- Dx: Sagittal sinus meningioma
- Photos: 4 months following surgery



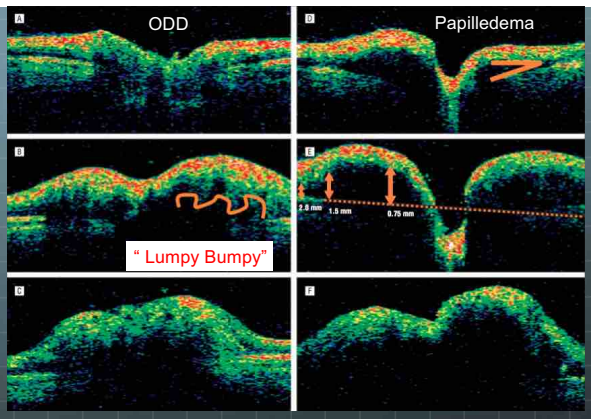
# OCT for disc edema

- Does not replace MRI
- Asssessment of Peripapillary NFL and disc parameters
  - Peripapillary NFL is thicker
    - Ave: 122 microns vs 91 for controls
  - Cross sectional imaging
    - Subretinal Fluid accumulation
    - Tissue thickening




  - Johnson et al, Arch Ophthalmol, 2009
  - Menke et al, IOVS, 2005

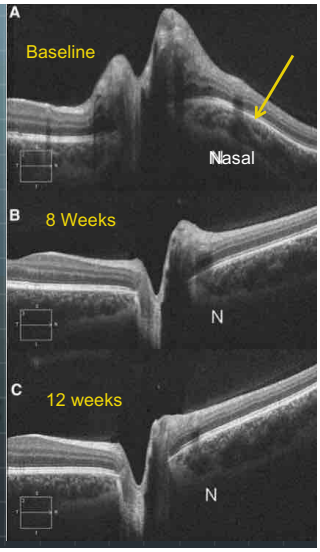





- Differentiation of Optic Disc Edema from Optic Nerve Drusen
  - Internal contour and separation of sub-retinal hyporeflective space can differentiate the two
    - Johnson et al, Arch Ophthal, 2009



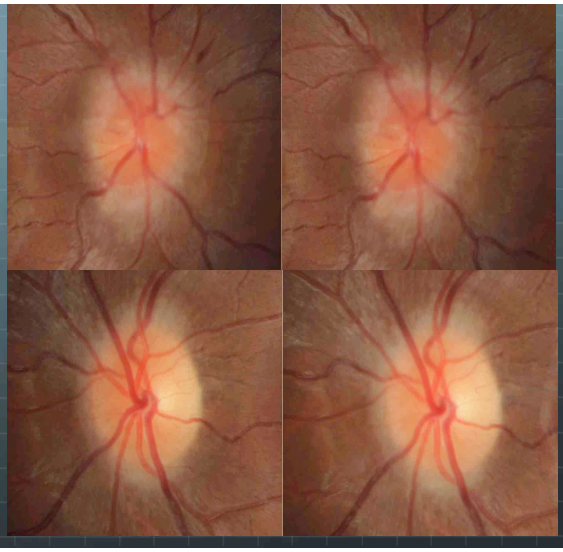
- Deformation of the RPE/ Basement Membrane in Swollen Optic Discs
  - Cirrus OCT – optic disc cube
  - 67% had Positive slope in early phases
    - Observed in about 10% of AION or Optic Neuritis cases
  - Angulation did not correlate with amount of disc swelling
  - Resolves over time
    - Kupersmith et al, IOVS, 2011

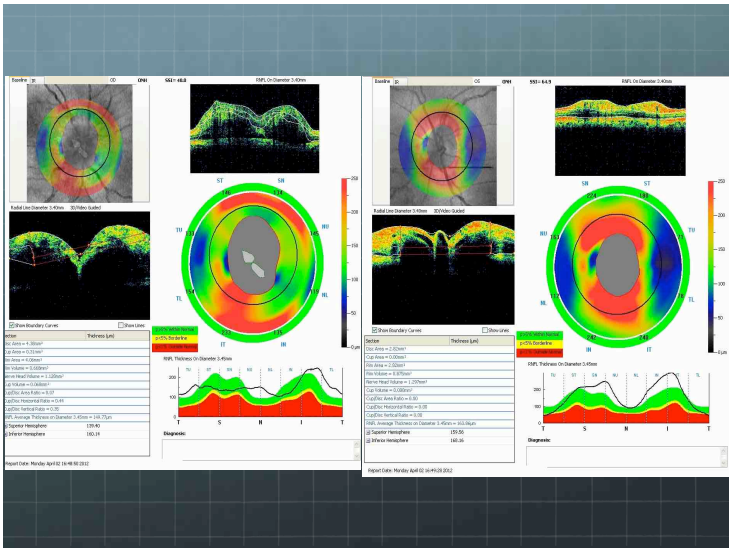





# 28 year old East Indian Female

- Headaches x 2 months
- Transient Visual Obscurations – 2 secs
- No medical problems or medications
- VA's: 20/20, 20/20
- Pupils: No APD
- BP: 118/78
- Anterior seg: unremarkable



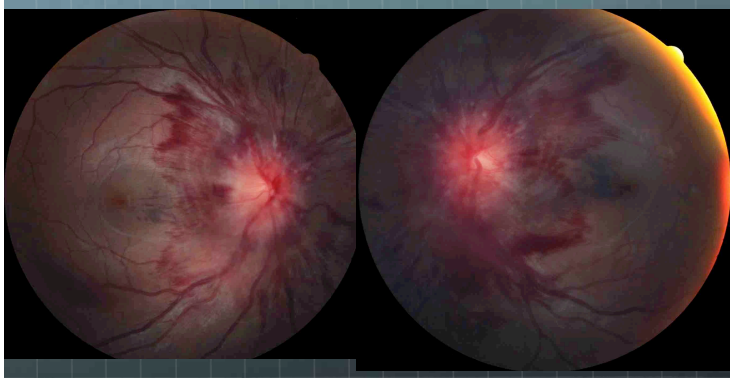


# 32 year old female

- CC: Intermittent flashes of light, blurred vision x 2 days.
- Recently diagnosed with viral meningitis. Polycystic ovarian syndrome, elevated cholesterol.
- Meds: Metformin, Amoxicillin, Lyrica, Hydrocortisone, Clarythromycin, Flexiril, Flonase
- BVA: OD: 20/60-, OS: 20/200
- BP: 117/76
- Pupils: 3+ reaction to light OU.

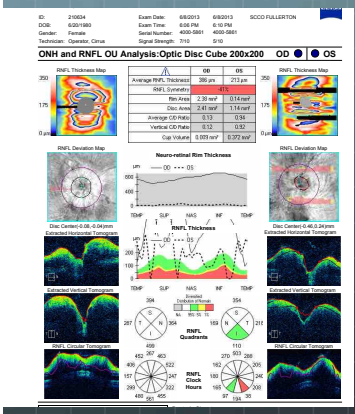


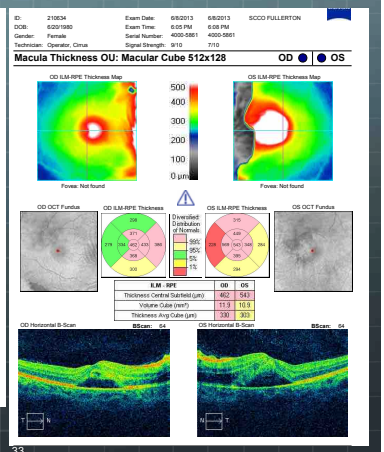
# Baseline photos





# OCT of ONH/RNFL and Macula

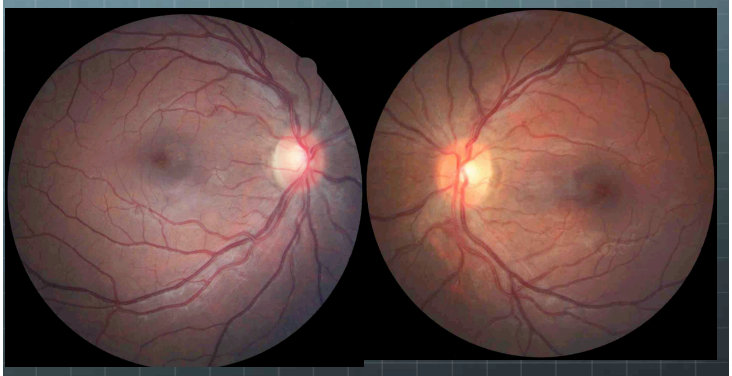



Tx: Started the patient on Diamox

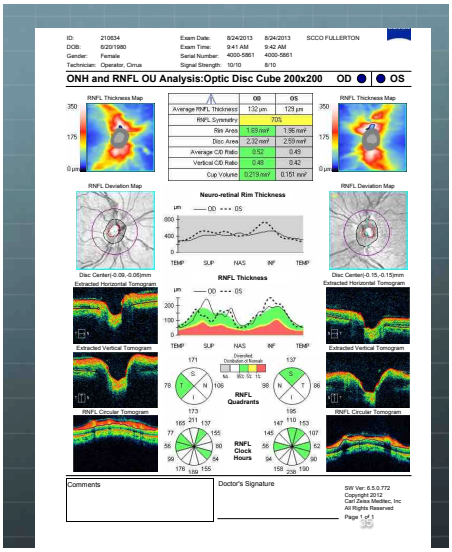


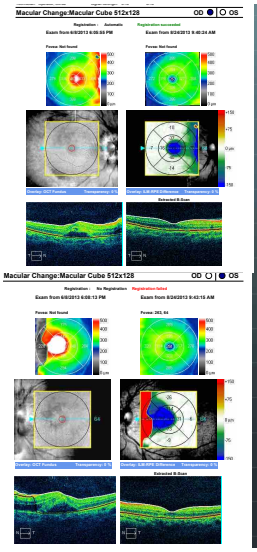
# 2 months later



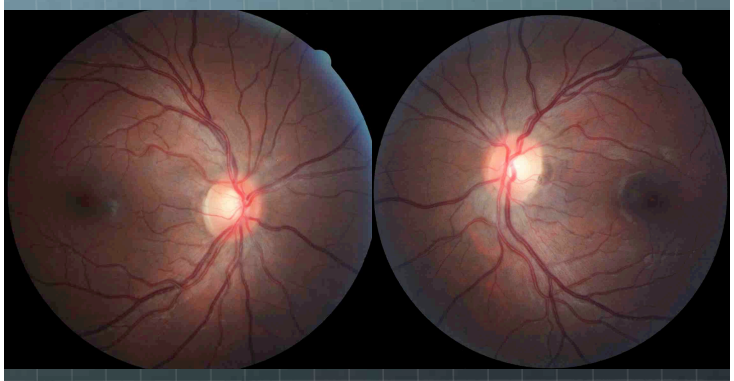
VA: OD: 20/20, OS: 20/40





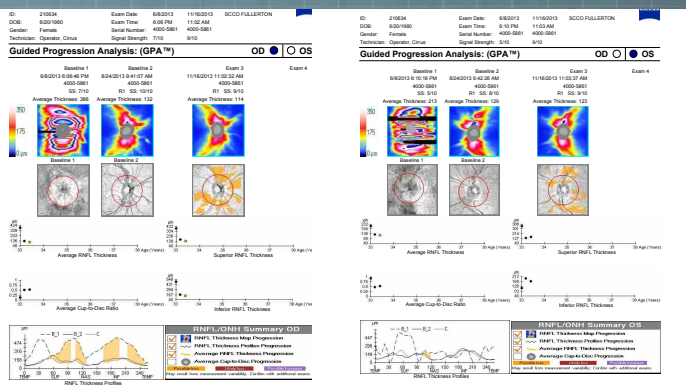




# 5 months later



BVA: OD: 20/20, OS: 20/25

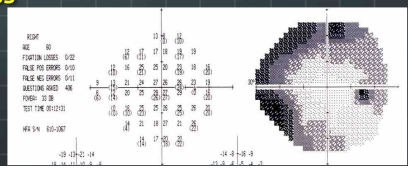
# Progression analysis

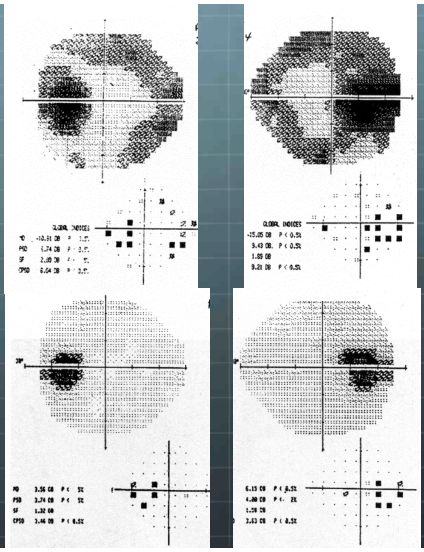





# Vision loss in Papilledema

- VF loss from compression of visual pathways
- Enlarged blind spot
- Glaucomatous VF defects
  - Arcuate bundles
  - Blindness
  - Reversible upon treatment
    - Unless axonal loss



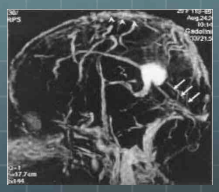


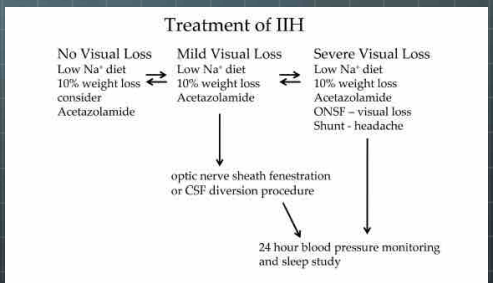
Before

1 month later

# Management of IIH

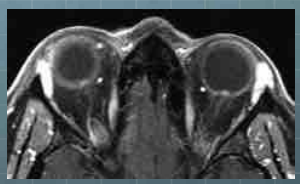
- Neuroimaging: MRI/MRV
- Lumbar puncture with opening pressure
- Lab tests to rule out coagulopathies
  - Cerebral Venous Sinus Thrombosis
- Serial VF and OCT

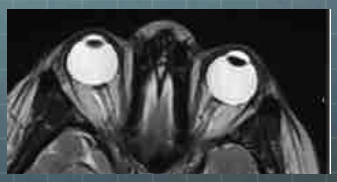





Wall, 2014

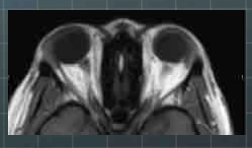
# Orbital findings on MRI





Elevated ONH

Flattened Posterior Sclera



Tortuosity of the Optic Nerve

Passi, Degnan, Levy, 2013

# Follow-up OCT

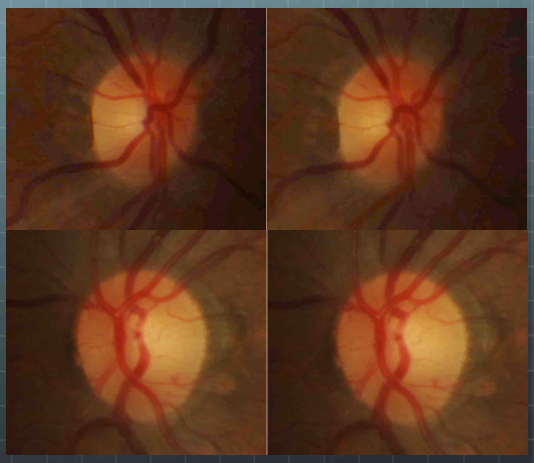
- Subsequent macular thinning:
  De-swelling vs. optic atrophy?
  - GCC is important for this distinction
- Rebolleda and Munoz-Negrete found that higher grades of papilledema at baseline had a worse visual outcome
  - Every 10 microns of increased RNFL thickening = a lowering of 0.6 dB on a VF at 1 year.

# Optic Neuritis

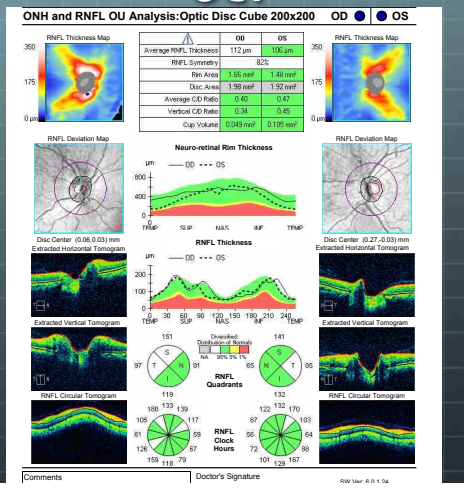
- **Patient profiles**
  - Young to middle age adults (16-55 yrs of age)
  - Female to male: 2:1
  - Annual incidence: 1-5/100,000
  - 20% of MS patients – ON is the initial symptom
  - 50% of MS patients have evidence of having ON
- **Symptoms:**
  - 90% have loss of vision, pain on eye movement, orbital pain, loss of peripheral vision, loss of color and contrast

# 43 year old Female

- **Chief Complaint:**
  - Decreased acuity OD x 2-3 weeks
  - Slight pain on eye movement
- **Occupation: Pet Groomer**
- **Medical history: DM, HTN, Elev. Cholesterol**
- **Vas: 20/80, 20/20 with current Rx**
- **Pupils: 2+ APD OD, color desaturation**
- **No restrictions of motility**
- **Anterior seg unremarkable**

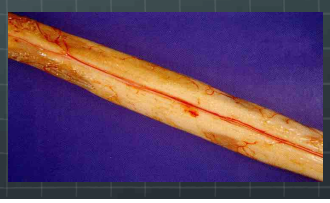


# OCT

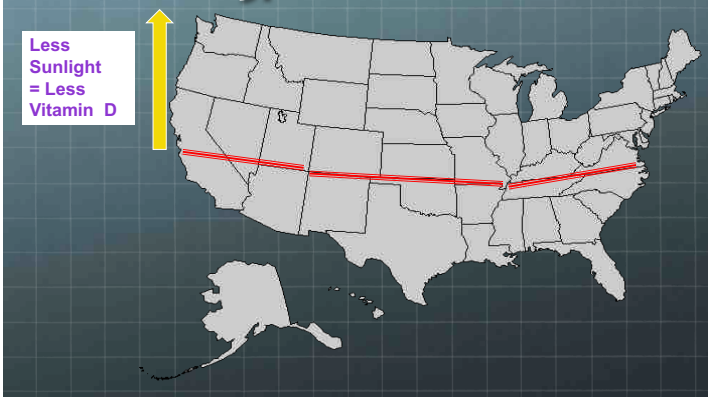



# Pathophysiology

- **Demyelination**
  - CNS white matter, optic nerve
  - Acute phase: Perivascular cuffing of T and B cells on the myelin sheath
  - Macrophages engulf products and glial cells proliferate resulting in permanent conduction deficits

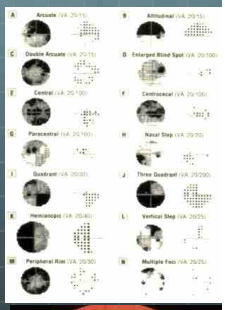


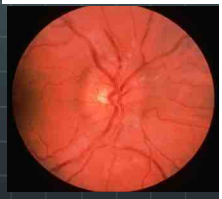
# 37th Parallel



# Signs of Optic Neuritis

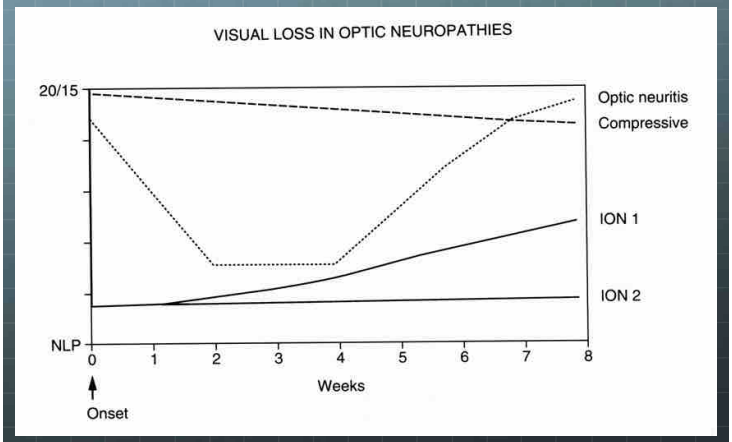
- Visual Acuity: 20/20 to LP
- Color vision: B-Y initially
- Contrast sensitivity
- Pupils: (+)APD
- Visual fields (Keltner, Arch Ophth, 1994)
  - Diffuse loss - 48%
  - Localized - 20%
  - Central - 8%
- Fundus appearance
  - 3:1: Retrobulbar optic neuritis: Papillitis





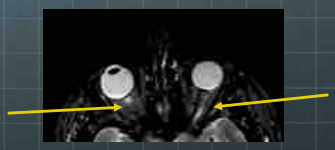
# DDx of Optic Neuritis

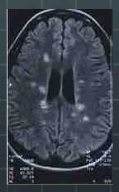
- Differential Diagnosis from Demyelination:
  - Infectious
  - Post-vaccination
  - Inflammatory disease
  - Medications
- Neuromyelitis Optica (Devic's Diseease)
  - NMO is a rare disorder
  - Characterized by Optic neuritis and acute myelitis
    - Negative brain MRI
    - Abnormal spinal cord MRI
    - Seropositive NMO- IgG
      - NMO criteria, 2006



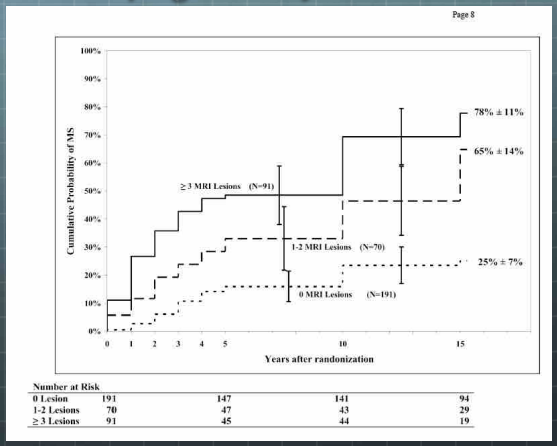

# Contrast enhanced MRI

- MRI is not routinely utilized to diagnose optic neuritis
  - Contrast enhanced MRI of the orbit with fat suppression.
- Value of MRI is to identify the presence of clinically silent demyelinating lesions
  - Periventricular white matter lesions on T2 scans





# Baseline MRI findings vs. risk of developing clinically definitite MS




ONTT – 15 year, Arch, Neurology, 2008

# Utility of OCT in Optic Neuritis

- OCT of the RNFL
  - Allows quantification of unmyelinated axons in eyes with and without a history of Optic Neuritis in MS patients
  - It can be used to follow recovery of optic neuritis over 12 months

## RNFL and acute optic neuritis

- Pro et al (2006) - HRT2 and OCT3
  - RNFL was slightly thicker in RON pts (no ophthalmoscopically evident swelling) at baseline.
  - HRT2 showed smaller mean cup size vs fellow eye and did not correlate to the MRI –demonstrated lesion
  - RNFL thinned temporally (46.8 microns vs. 57.8 – fellow eye)
  - Cup normalized at the follow-up (1 and 3 mos)

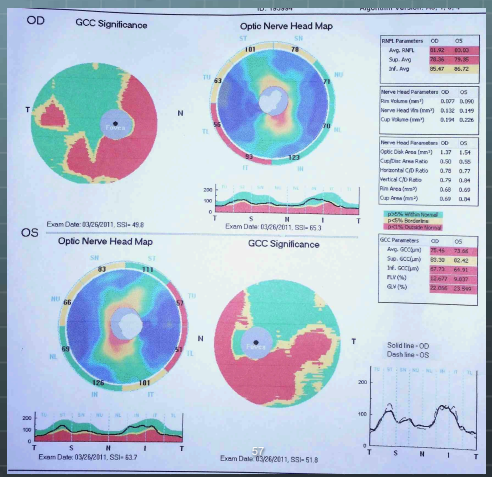


## Optic Neuritis

- Evolution of RNFL loss
  - RNFL thinning most often occurs between 3 and 6 months in 85% of patients
    - Ave RNFL of 78 microns vs. 100 microns
  - First inter-eye difference is seen at 2 mos
  - Stabilized between 7-12 months
  - No change between 1 and 2 years
    - Costello et al, 2006, 2008, 2009
  - Also noted that below 75 microns was when persistent visual dysfunction was predicted.



## 31 yr old pt with recurrent ON






## OCT and MS

- Multiple studies have shown statistically significant thinning of the unaffected eye in pts with ON.
- Patients with MS without a diagnosis of ON show thinning of the RNFL
- Evidence that MS is associated with progressive axonal loss in the afferent visual pathway
  - First noted by Charcot in the 1880's
  - Red-free photography (1970's)
  - Post mortem studies exhibit ON and RNFL changes



## OCT and MS

- Cohorts of MS patients across the US display similar findings of ave RNFL thickness vs. controls (90-93 microns vs. 103-105 microns) with OCT3
  - RNFL Thinning:
    - MS with ON> MS pts > Controls
- OCT findings differ among MS subtypes
  - Secondary progressive MS vs. Relapsing MS or primary progressive types
    - Secondary progressive shows more thinning

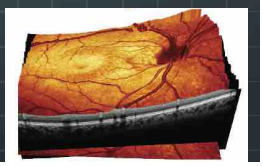


## OCT and MS

- Annals of Neurology, 2016
- 107 MS patients followed for 4 years
- Aim: to determine if OCT changes mirror changes on MRI in MS patients
- Conclusions:
  - Rate of tissue thinning in the eye ( ganglion cell and IPL) mirrored that of MRI degeneration in specific brain regions (whole brain, white matter, gray matter, thalamus)

## Predictors based on OCT?

- Progressive disability in MS is associated with axonal loss, not demyelination.
- For every 10 microns of RNFL loss, the odds of being ambulatory are decreased 2.5 fold
  - Costello, NANOS, 2009
  - But conflicting data is also reported
- Future risk of developing MS or progression??
  - Linear vs. Non-linear forms





Active MS is associated with accelerated retinal ganglion cell/inner plexiform layer thinning

- Ratchford, et al. Neurology, 2013.
- 164 pts w MS, 59 controls
  - Clinically isolated syndrome (CIS)
  - Relapsing, remitting MS
  - Primary progressive MS
  - Secondary progressive MS
- Underwent Cirrus SD-OCT scans every 6 months
  - Annual MRI
  - Mean follow-up time – 21.1 months
  - Development of ON during f/u - excluded



## Findings:

- Patients with new T2 lesions or gadolinium enhancing lesions and less than 5 yrs duration exhibited fastest rate of thinning.
- Conclusions: MS patients with clinical and/or radiologic non-ocular disease activity, particularly early in the disease course, exhibit accelerated GCIP(ganglion cell/Inner plex) thinning.
  Our findings suggest that retinal changes in MS reflect global CNS processes, and that OCT-derived GCIP thickness measures may have utility as an outcome measure for assessing neuroprotective agents, particularly in early, active MS.



## To treat or not to treat ON

- Optic Neuritis Treatment Trial
  - Oral vs. IV steroids
  - Showed no of Baseline MRI lesions was a predictor to development of CDMS
  - Clinical outcomes are the same.
- Early treatment delays conversion to CDMS but does not show any benefit in improving neurological disability.
- Neurological evaluation for management of global demyelinating disease
  - Treatment of the MS

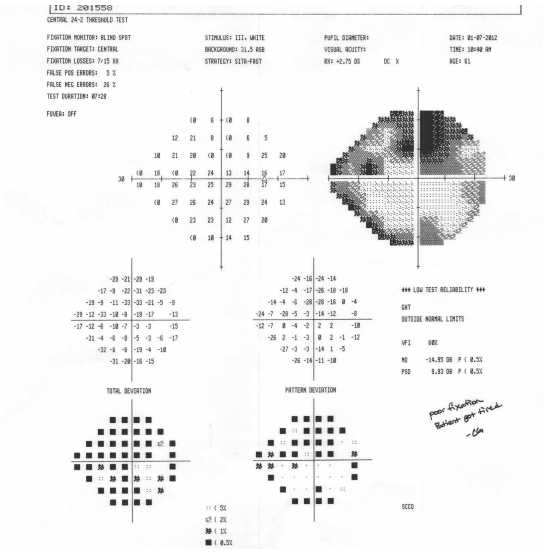
## Case : Dimming of Vision

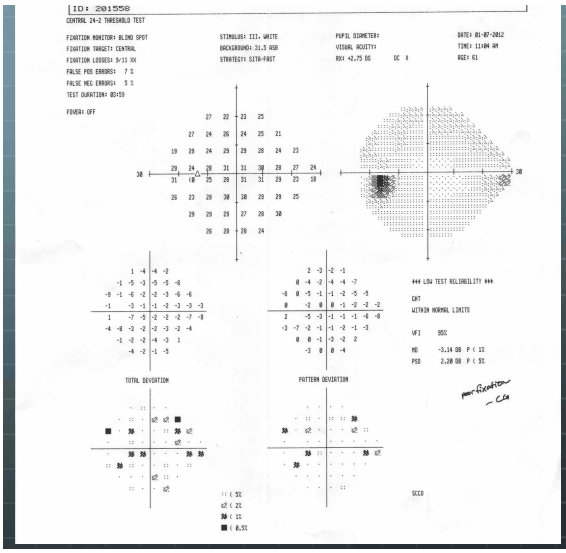
- 61 year old East Indian Male
- Blurred vision x 1 month.
  - Dimming of vision, thinks it may be related to a red eye he had
- Ocular Hx: Cataract Sx 2 yrs prior OU
- Med Hx: Type II DM x 5 yrs, HTN, hypercholesterolemia
- Meds: Tricor, Metformin, Lisinopril

## Patient Data

- Corrected VA's: OD: 20/40-, OS: 20/25
- Pupils: (+) +2 APD OD
- EOM's: Unrestricted
- Anterior Segment: Unremarkable OD, 1+ PCO OS
- GAT: 15, 18
- BP – 120/70
- Visual fields: See slides
- Post Segment: See slides

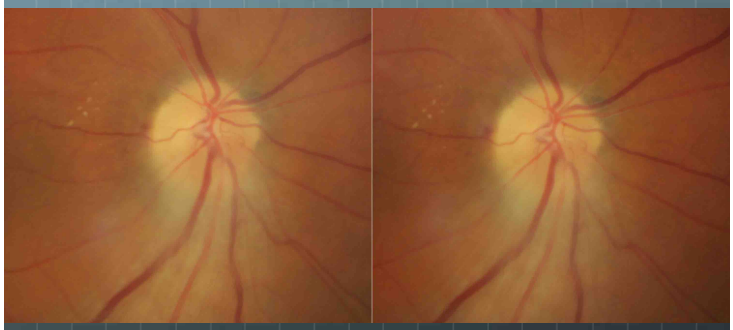
VF
OD



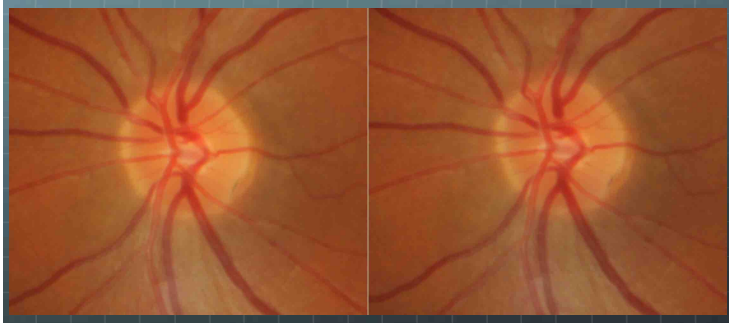


VF
OS

## Fundus Appearance OD



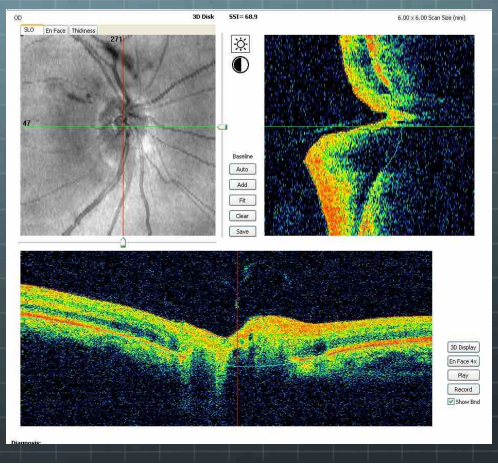
## Fundus OS



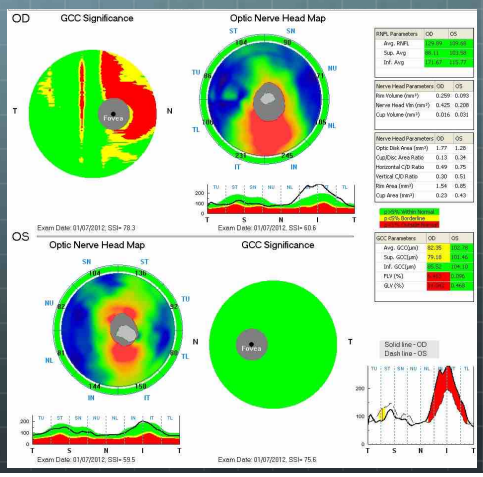
## Considerations?

- Swollen Optic Disc – Sectoral - OD
- Afferent pupillary defect OD
- Dimming of Vision
- History of Vascular Disease

## OCT of the OD ONH

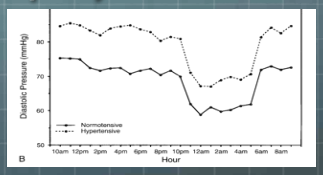


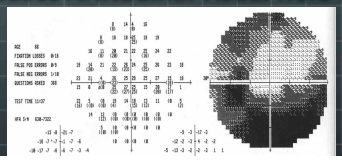
# OCT OU

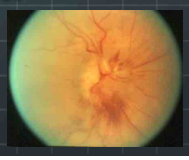



# What about Anterior Ischemic Optic Neuropathy?

- **What we remember:**
  - Disk at risk
  - Unilateral ON swelling
  - Poor disc perfusion
  - Two types:
    - **Arteritic – AION: Giant Cell or Temporal Arteritis**
    - **NA-AION: everything else**
  - Causes Altitudinal visual field defect





# Is it Arteritic or Non-Arteritic AION in my chair?

| | Arteritic-AION | Non-Arteritic AION |
|---|---|---|
| % of cases of AION | 12.5% | 87.5% |
| Age | 73 is the mean age | 50-65 |
| VA Loss | 75% are < 20/200 | Less Profound |
| Systemic Symptoms | 75% of patients | None |
| ESR | 75 or greater | 30 - 40 |
| Amaurosis Fugax | 75% -1-2 wks prior | 25% of patients |
| Disk Appearance | 50% edema, 50% pale | Sectoral or full edema |
| Bilateral Involvement | 75% in 1wk, if no tx | 11-48%in 2 years |
| Treatment | Corticosteroids | |
| Improvement | Rarely | 16-43% |

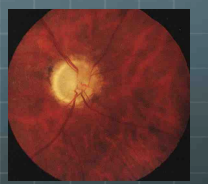
# Risk factors of Non-arteritic AION

- Small optic nerve
- Diabetes (Heyreh, 1990, Feldon, 1999)
- Hypertension/ Hypotension
  - Aggressive management/ QHS Dosing (Hayreh)
- Sleep Apnea (Arch Ophthal, May, 2002)
- Viagra, Cialis (J Neuroophth, Ophthalmol, Arch Ophthal)
- Carotid artery disease, Ischemic heart disease
- Hyperlipidemia (Ophthalmology, 2003)
- Smoking
- Migraine(Heyreh, 1997)
- Sticky Platelet syndrome (BJO, 2008)
- High altitude(Ind J Ophthal, 2002)
- Ocular surgery(AJO, 2003) , Spinal/Cardiac surgery(Surv Ophthal, 1998)
- Disc Drusen (NANOS, 2002)
- Shock induced or blood loss (Brown, 1994, Chun, 1997)

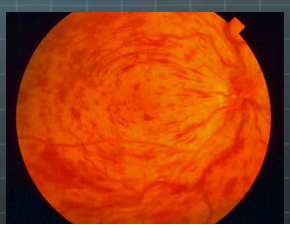


# But… are A-AION and NA-AION even more Different?

- **A-AION: Arterial disease**
  - VA/ Visual loss is more profound
  - Complete excavation of the disc
  - Less hemorrhages
- **NA-AION: Is it a Venous disorder?**
  - Can accompany CRVO
  - Hemorrhages are more common
  - Visual loss/ structural changes are more similar to venous occlusion in CRVO vs. arterial infarction
  - Assoc w/ low rate of large vessel occlusive dz and CVA
  - FANG shows mildly delayed arterial filling, normal choroidal circulation



| CRVO | CRAO |
|---|---|
| Hypertension | Hypertension |
| Diabetes | Diabetes |
| Bleeding /Clotting Disorders | Giant Cell Arteritis |
| Vasculitis | Embolism |
| Cardiovascular Disorders | Patent Foramen Ovale |
| ED drugs | Cardiac valve disease |
| Oral contraceptives | Atherosclerosis |
| Sleep apnea? | Hypercoagulable state |
| Hypotension | Collagen Vascular Dz |

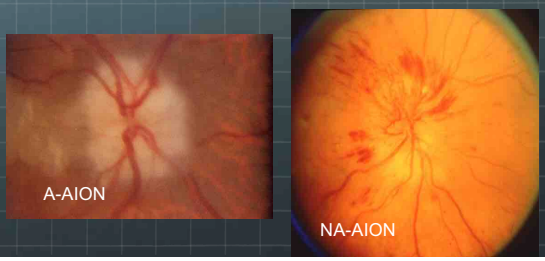


# New Pathophysiology?

- Dilation of the CRA causes compression upon the CRV in the pre-laminar disc
  - Results in venous congestion
  - Think CRAO (A-AION) vs. CRVO (NA-AION)
  - Causes of venous dilation
    - Sleep apnea
    - Prolonged hypotension
    - Erectile dysfunction drugs
  - Levin LA, Meyer-Danesh HV, Arch Ophthalmology, Nov, 2008

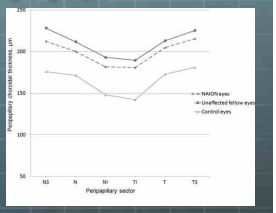
# Is this anterior venous ischemic optic neuropathy?

- Patients have premonitory asymptomatic disc edema
- High incidence of diabetes: venous-capillary disease

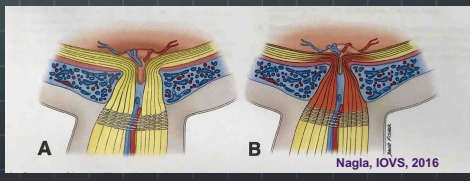



# Impact of the choroid?

- Peripapillary choroid is thicker in NA-AION eyes and fellow eyes vs. Control eyes. (121-143%)
  - Perez – Sarregui, 2018
  - Fard, IOVS, 2015



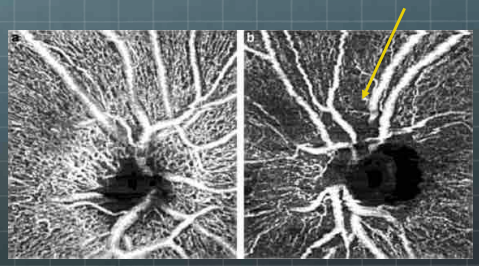

- Creates the "Disc at risk"
  - Bruch's membrane opening is not smaller in NA-AION eyes
- Peripapillary choroid thickens with PDE use in normal young subjects,
  - Moschos, JCEO, 2016



Nagla, IOVS, 2016

# OCT - Angiography

- OCT-A shows diffuse loss of peripapillary microvascular cuff
  - Also Sectoral loss that correlates w VF defect

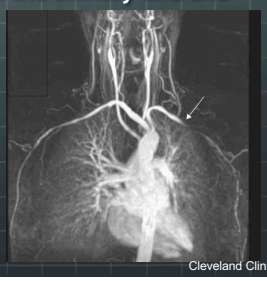


Normal AION

Rougier, 2017

# Giant Cell Arteritis

- Chronic vasculitis of the Medium and large extracranial arteries
- What is new?
  - Color Doppler/Duplex ultrasound of temporal arteries
  - Higher incidence of occlusions in the axillary and and subclavian arteries
  - Marked increase in developing aortic aneurysms/aortic dissection
    - MRA of these large vessels


Cleveland Clinic

# ACR Revised Criteria for GCA 2016

Table A: 2016 ACR revised criteria for early diagnosis of Giant Cell (Temporal) Arteritis [a]

| | |
|---|---|
| ❖ **Entry Criteria**: | |
| • Age at onset ≥ 50 years old | |
| • Absence of exclusion criteria [b] | |
| ❖ **Domain I criteria** | |
| • New onset localized headache [c] | 1.p |
| • Sudden onset of visual disturbances [c] | 1.p |
| • Polymyalgia Rheumatica (PMR) | 2.p |
| • Jaw Claudication [c] | 1.p |
| • Abnormal temporal artery [d] | Up to 2.p |
| ❖ **Domain II criteria** | |
| • Unexplained fever and/or anemia | 1.p |
| • ESR ≥ 50 mm/hour [e] | 1.p |
| • Compatible pathology [f] | Up to 2.p |

a. In the presence of 3 points or more out of 11 with at least one point belonging to domain I along with all entry criteria, the diagnosis of Giant cell arteritis can be established.

b. Exclusion criteria are including : ENT and eye inflammation, kidney, skin and peripheral nervous system involvement, lung infiltration, lymphadenopathies, stiff neck and digital gangrene or ulceration

c. No other etiologies can better explain any one of the criteria

d. Enlarged and/or pulseless temporal artery : 1.p. / tender temporal artery: 1.p

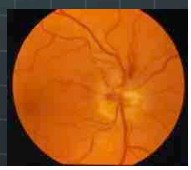
e. It must be ignored in the presence of PMR

f. Vascular and/or perivascular fibrinoid necrosis along with leukocyte infiltration: 1.p. /and granuloma: 1.p

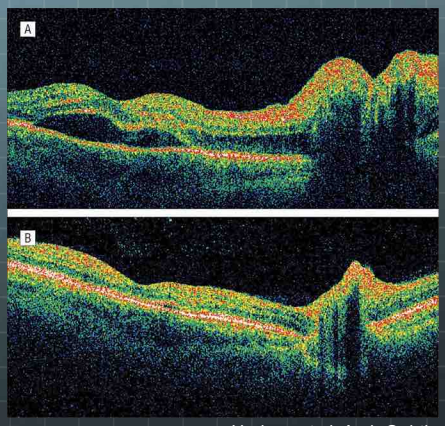
Salehi- Abari, 2016

## OCT and ION

- Contreras (Ophthal, 2007) demonstrated in eyes with NA-AION that the mean RNFL would increase 96% in the affected eye in the acute phase.
  - Then at 6 months, the RNFL was thinnest superiorly, inferiorly, temporally, then nasally
- Hedges et al, noted the presence of sub-retinal fluid in 8 patients.
  - Not from the choroid or retinal vessels based on FANG of one patient
  - Acuity improved as fluid resolved



## Baseline and 7 weeks later




Hedges et al, Arch Ophthalmol, 2008



## Management of NA-AION

- No proven treatments
  - Aspirin, high dose steroids, Avastin
- Rule out GCA in older patients
- Manage systemic disorders
- Sleep Apnea?
- Avoid hypotension – night time
  - Diastolic BP – IOP: Less than 30

## Management of A-AION

- Arteritic: Giant Cell Arteritis
  - Based on the presumptive diagnosis of GCA-AION, initiate immediate treatment of corticosteroids
    - Goal is to reduce profound VA loss in fellow eye
    - 75% of untreated cases can go bilateral in one wk
  - Then STAT ESR and C-Reactive Protein
  - Temporal artery biopsy
  - Doppler of temporal arteries
  - MRA of the aorta to rule out dissection/aneurysm
  - Prognosis of patients: Fair to poor
    - Recovery of vision
    - Cupping of the optic disc


## Management of AION

- Non-Arteritic
  - Control of underlying vascular disorder
  - Visual Prognosis: Can see visual improvement up to 43% of patients

Quark

- Phase III Quark Study (Now recruiting)
  - Intravenous injection of Neuroprotection agent
  - Vision loss in the last 14 days

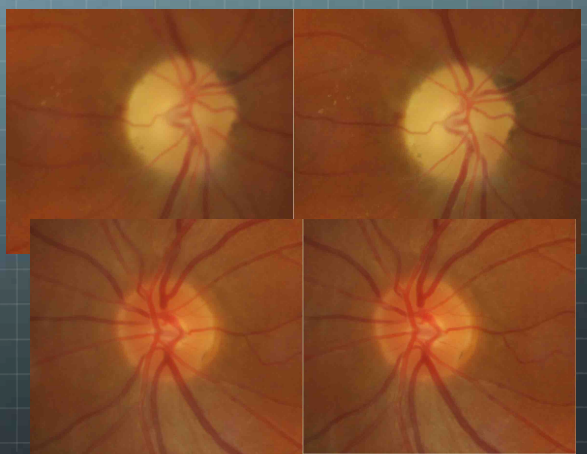
## Management of this Patient

- One month out - less efficacy with any Tx
- Sent to PCP for lab testing and better control of DM and HTN
- Take BP meds in the Morning, not the PM
- Monthly follow-ups

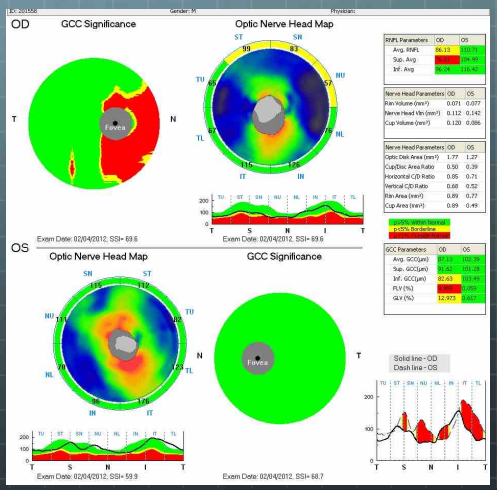
# Follow –up of patient

- One month follow-up
- CC: Vision seems to be about 10-20% better
- VAs:  OD: 20/40+,  OS: 20/25
- See Imaging and VF

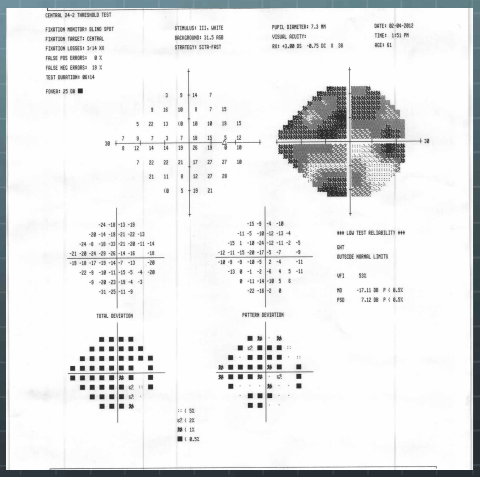
# Follow-up 1 month



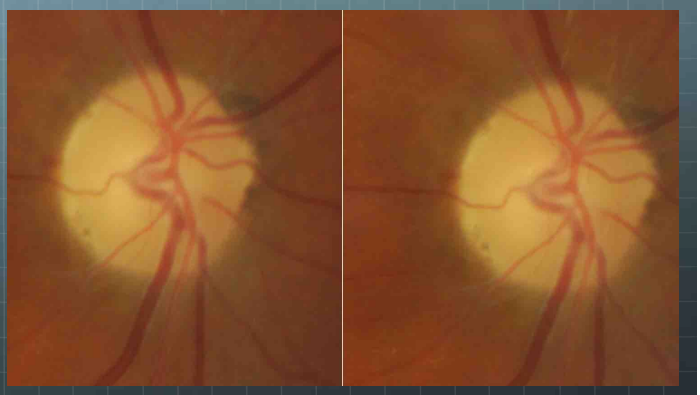
# OCT – 1 month

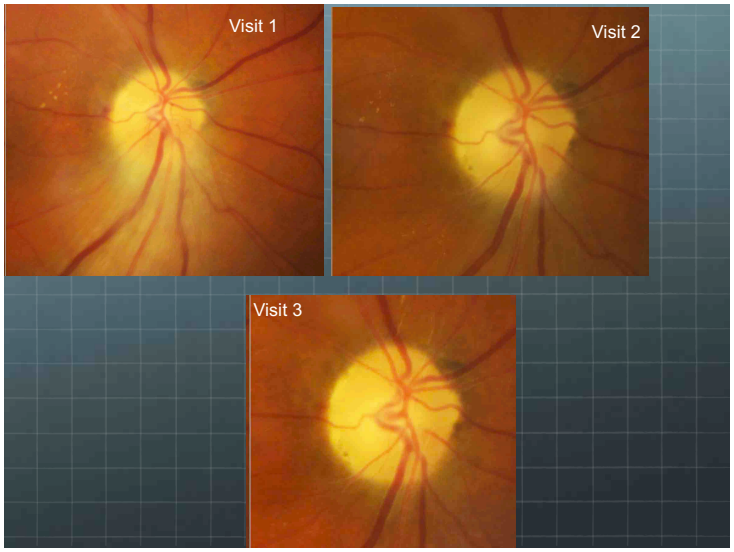


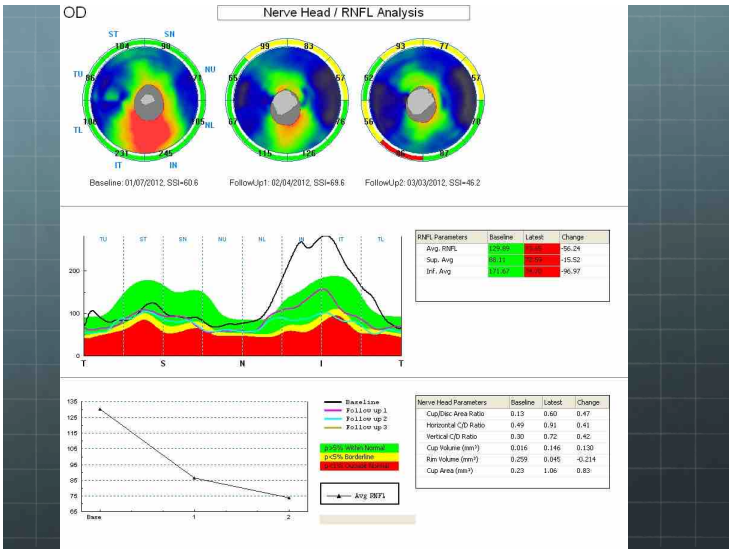

# VF - OD

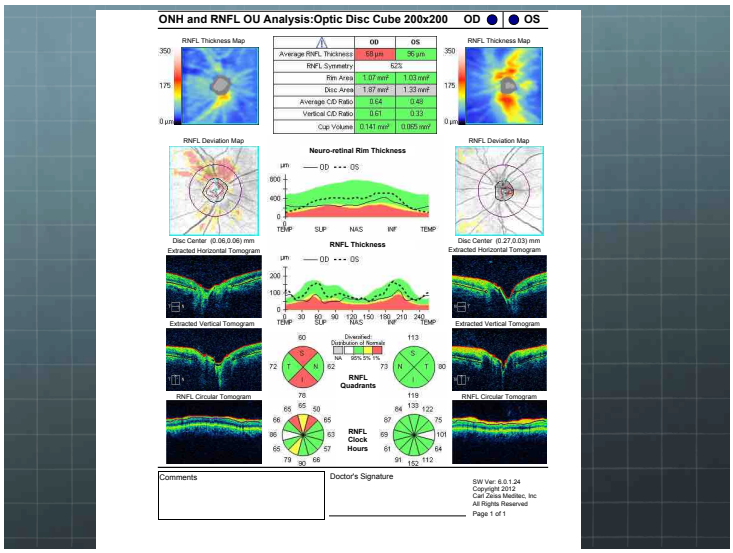


# Follow –up 2mos






OD
Nerve Head / RNFL Analysis
Baseline: 01/07/2012, SSI=60.6
FollowUp1: 02/04/2012, SSI=69.6
FollowUp2: 03/03/2012, SSI=46.2
RNFL Parameters | Baseline | Latest | Change
Avg. RNFL | 129.69 | 73.13 | -56.24
Sup. Avg | 161.11 | 125.59 | -35.52
Inf. Avg | 171.67 | 74.70 | -96.97
Nerve Head Parameters | Baseline | Latest | Change
Cup/Disc Area Ratio | 0.13 | 0.60 | 0.47
Horizontal C/D Ratio | 0.49 | 0.91 | 0.41
Vertical C/D Ratio | 0.30 | 0.72 | 0.42
Cup Volume (mm³) | 0.016 | 0.146 | 0.130
Rim Volume (mm³) | 0.259 | 0.045 | -0.214
Cup Area (mm²) | 0.23 | 1.06 | 0.83
Baseline
Follow up 1
Follow up 2
Follow up 3
p>5% Within Normal
p<5% Borderline
p<1% Outside Normal
Avg. RNFL


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
OD | OS
Average RNFL Thickness | 68 µm | 96 µm
RNFL Symmetry | 62%
Rim Area | 1.07 mm² | 1.33 mm²
Disc Area | 1.87 mm² | 1.33 mm²
Average C/D Ratio | 0.64 | 0.49
Vertical C/D Ratio | 0.61 | 0.33
Cup Volume | 0.141 mm³ | 0.085 mm³
Comments
Doctor's Signature
SW Ver: 6.0.1.24
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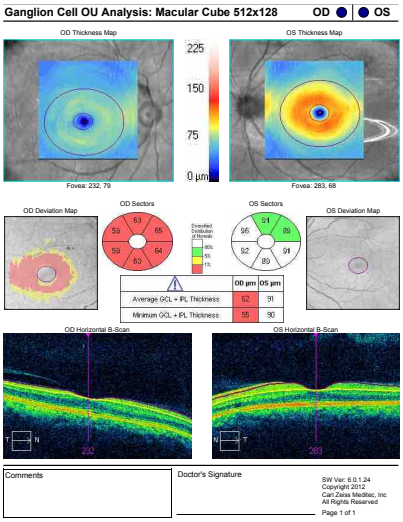
Ganglion Cell OU Analysis: Macular Cube 512x128 OD OS
OD Thickness Map
OS Thickness Map
Fovea: 232, 79
Fovea: 283, 68
OD Deviation Map
OS Deviation Map
OD Sectors
OS Sectors
OD µm | OS µm
Average GCL + IPL Thickness | 62 | 91
Minimum GCL + IPL Thickness | 55 | 90
OD Horizontal B-Scan
OS Horizontal B-Scan
Comments
Doctor's Signature
SW Ver: 6.0.1.24
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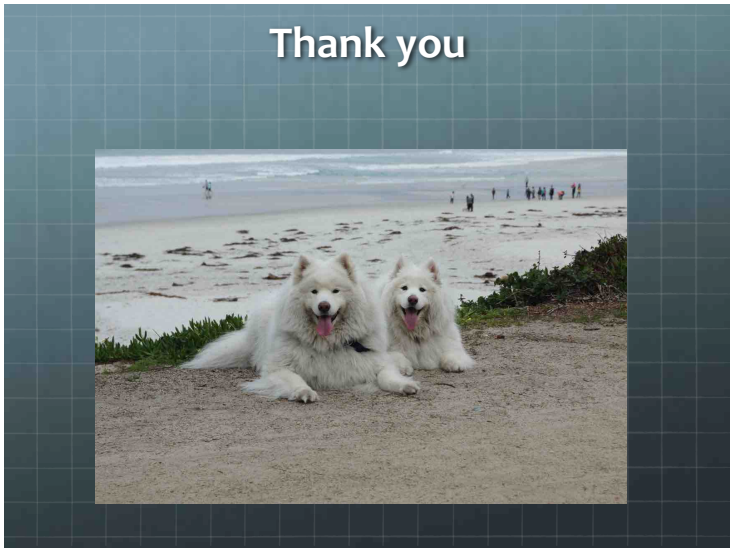
Thank you