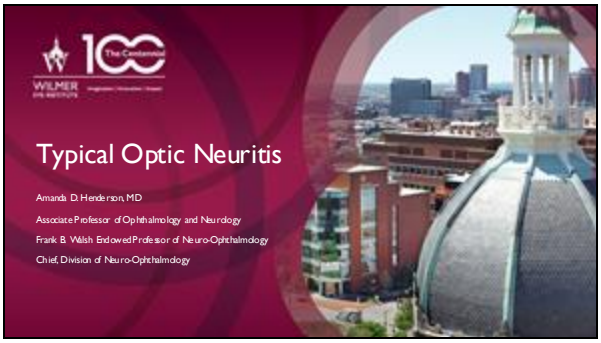

Disclosures

Wilmer Eye Institute
Johns Hopkins Medicine

Horizon Therapeutics/Amgen (Advisory Boards)

Argenx (Clinical Trial Site)

Objectives

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Johns Hopkins Medicine

By the end of this presentation, participants will be able to:

- Describe the clinical and neuroimaging characteristics of "typical" optic neuritis
- Develop a treatment plan for "typical" acute optic neuritis

Optic Neuritis

- Inflammatory optic neuropathy
- Most common acute optic neuropathy in young adults
- Incidence of unilateral optic neuritis ranges from 0·94 to 2·18 per 100 000 per year
- Female > male
- May be isolated or associated with an underlying systemic disease
- Underlying conditions may include:
  - Multiple sclerosis
  - Neuromyelitis optica spectrum disorder
  - Myelin oligodendrocyte glycoprotein (MOG) antibody associated disease
  - Other systemic disorders (connective tissue disease, granulomatous disease, infection)

Typical vs Atypical Optic Neuritis

- Multiple sclerosis
- Neuromyelitis optica spectrum disorder (NMOSD) (AQP4-IgG+)
- MOG-IgG-associated disease (MOGAD)

**It is important to identify the correct underlying cause, as prognosis and treatments are different

**Also, MS treatment can cause clinical worsening in NMO (and maybe MOGAD)

5

Case

A 35-year-old woman with no significant past medical history presents with blurry vision and pain with eye movements in the left eye x1 day.

Acuity is 20/20 in the right eye, 20/250 in the left

Left relative afferent pupillary defect

12/12 color plates OD, 1/12 OS

Anterior segment exam is unremarkable

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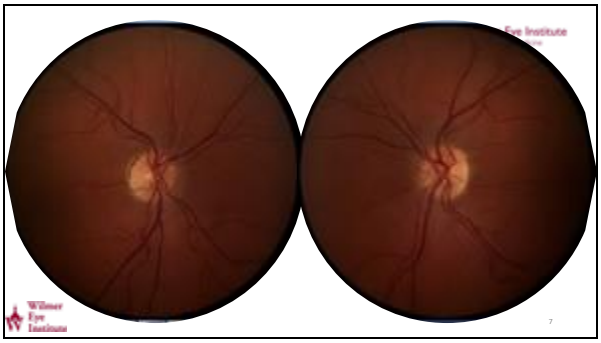
Eye Institute
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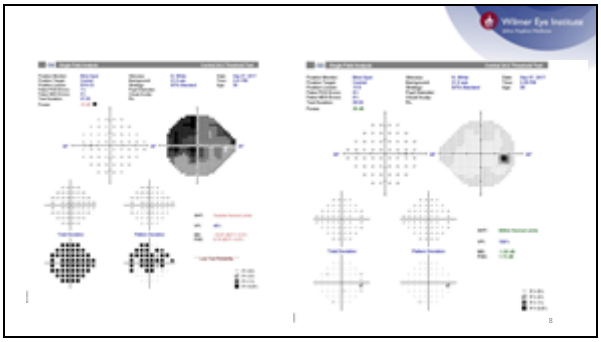
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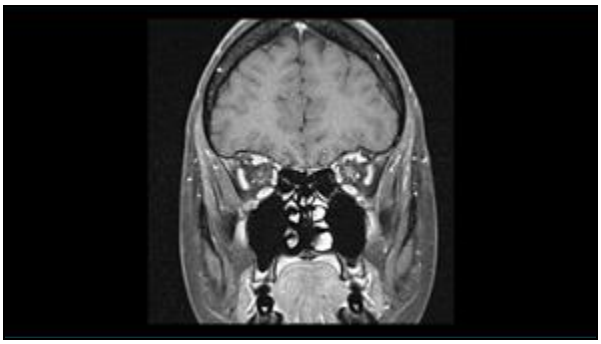

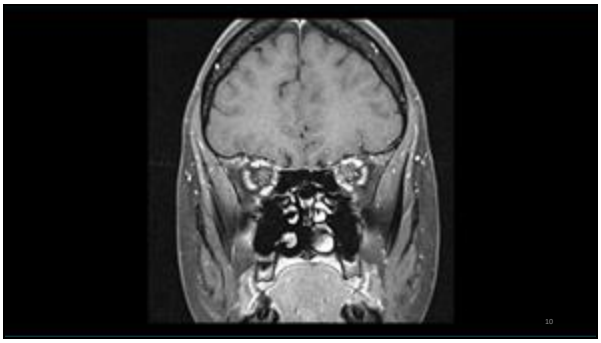
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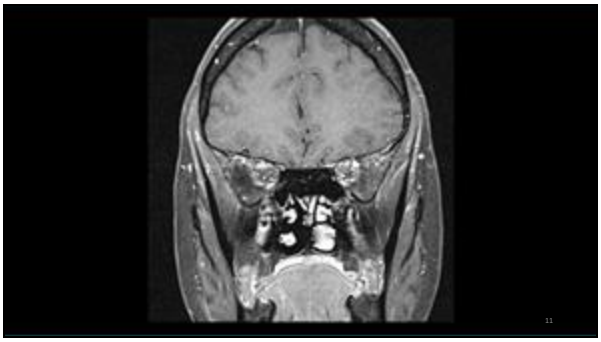
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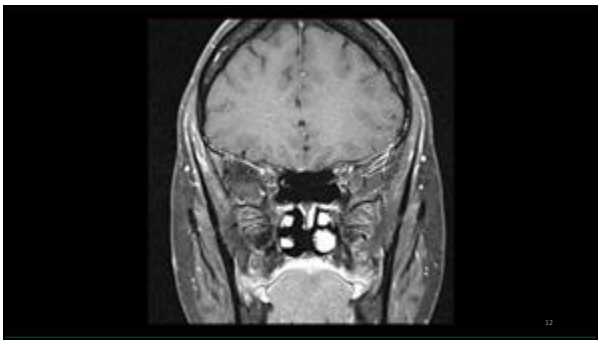
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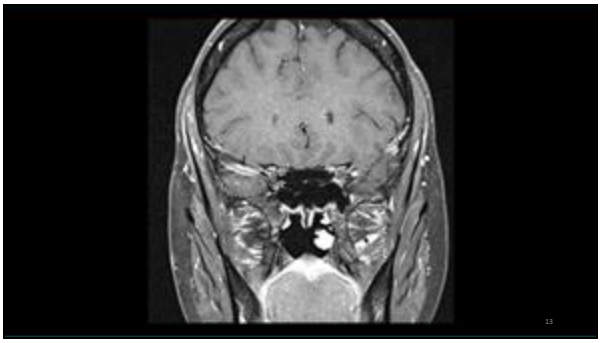
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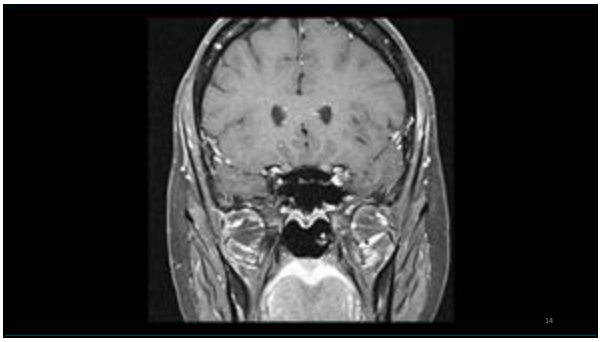
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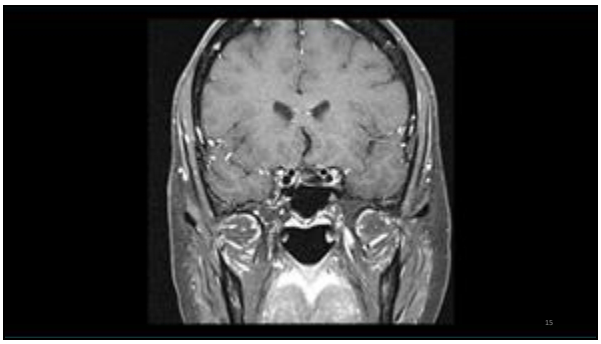
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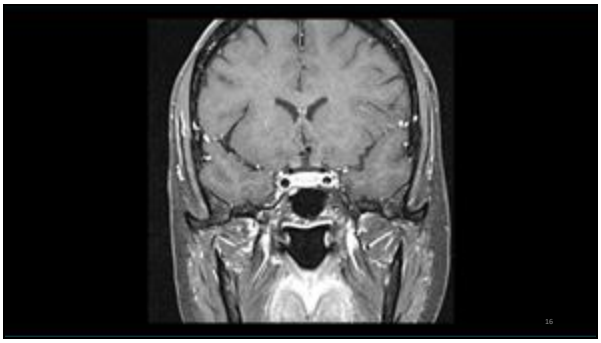
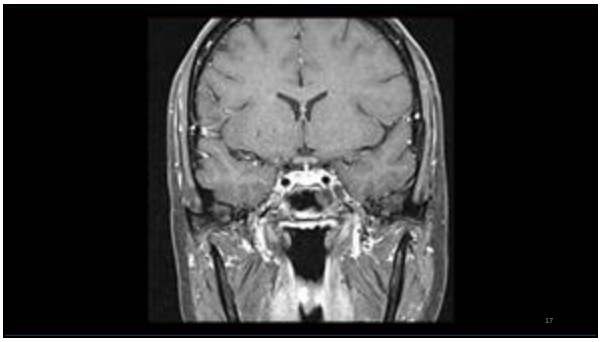
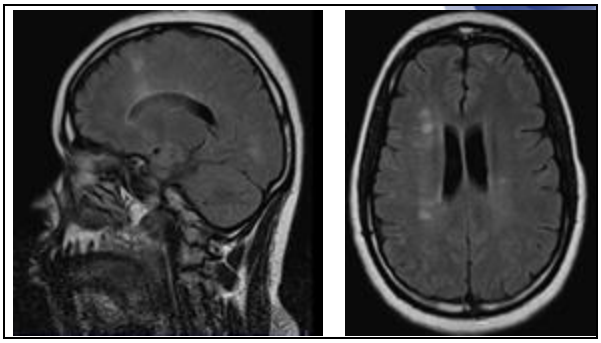




Clinical Presentation

- Acute / subacute, unilateral vision loss
- Pain with eye movements
- Decreased VA and color visio
- Central/cecocentral VF defect
- Normal appearing optic discs
- MRI: Short segment of unilateral optic nerve enhancement, no perineural involvement

How does this help narrow our differential diagnosis?
What other MRI findings would also help?



Revised McDonald Criteria 2017

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- Dissemination in space
  - One or more T2 lesions in at least two out of four CNS areas: periventricular, sub/juxtacortical, infratentorial, and spinal cord
- Dissemination in time
  - A new T2 and/or gadolinium-enhancing lesion with reference to a baseline scan OR
  - Simultaneous presence of asymptomatic gad-enhancing and non-enhancing lesions at any time OR
  - Clinical evidence of dissemination in time OR
  - Demonstration of CSF-specific oligoclonal bands

*2024 Update to Criteria, presented at ECTRIMS in 9/2024, publication pending

- Add optic nerve as a fifth potential CNS area to meet DIS criterion
- Add 6+ lesions with central vein sign, or kappa free light chains in CSF, as other options to meet DIT criterion

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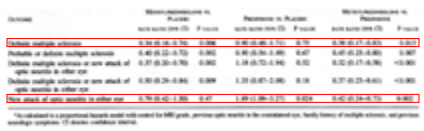
OPTIC NEURITIS TREATMENT TRIAL
ONTT

- 389 subjects with acute optic neuritis
- Enrolled between July 1, 1988, and June 30, 1991
- Followed prospectively for 15 years

Optic Neuritis TREATMENT Trial



Beck RW, Cleary PA, Trobe JD, et al. The effect of corticosteroids for acute optic neuritis on the subsequent development of multiple sclerosis. The Optic Neuritis Study Group. *N Engl J Med*. 1993; 329(24): 1764-1769.

High-Dose Steroid Treatment: IV versus PO

- Oral steroids offer potential advantages over IV steroids:
  - Lower cost
  - Greater convenience
- Similar outcomes, tolerance, and relapse rates have been shown for equivalent doses of oral and intravenous steroid (Morrow 2018; Le Page 2015; Sharrack 2000)
- Based on these data, equivalent-dosage oral therapy is a suitable alterative to intravenous administration of steroids for acute optic neuritis
  - e.g., prednisone 1,250mg po daily x5 days = methylprednisolone 1gm iv daily x5 days
- When using oral steroid, give PPI or H2 blocker for GI prophylaxis

Summary: Evaluation

- MS-related optic neuritis typically presents with normal optic disc appearance, unilateral involvement
- MS-related optic neuritis less likely to show longitudinally-extensive optic nerve enhancement on MRI, compared with atypical causes (ie, short segment involvement common)

Beck R, N *Engl J Med* 1993

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Summary: Acute Management

- In MS-related optic neuritis, steroid treatment improves rate of recovery but not ultimate visual outcome
- Low dose steroid treatment may increase risk of optic neuritis recurrence compared with high dose and placebo
- Similar outcomes, tolerance, and relapse rates have been shown for equivalent doses of oral and intravenous steroid

Beck, *N Engl J Med* 1992; Morrow, *JAMA Neurol* 2018; Le Page, *Lancet* 2015

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Summary: Chronic Management

- Disease-Modifying Therapy for MS
  - Many options now available, choice may be tailored to individual cases
- Neuroimmunology consultation for drug selection and ongoing management

Disclosures

Wilmer Eye Institute

Horizon Therapeutics/Amgen (Advisory Boards)

Catalyst Pharmaceuticals (Advisory Board)

Argenx (Clinical Trial Site)

Objectives

Wilmer Eye Institute

By the end of this presentation, participants will be able to:

- Identify clinical and neuroimaging red flags for "atypical" optic neuritis
- Develop a treatment plan for "atypical" acute optic neuritis

Case 1

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49-year-old woman presents with 1 week of bilateral eye pain, worse with eye movements, and 2 days of blurry vision in both eyes.

Acuity 20/70 OD, 20/50 OS

No RAPD

Anterior segment exam unremarkable

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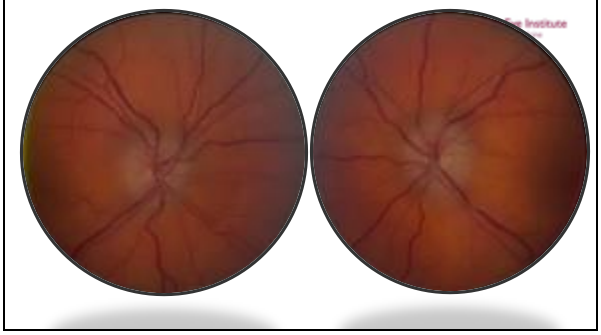

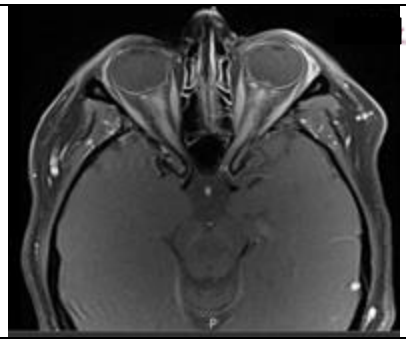

Clinical Presentation

- Acute / subacute, **bilateral** vision loss
- Pain with eye movements
- Decreased VA and color visio
- Central/cecocentral VF defect
- **Swollen** optic discs
- MRI: **Longitudinally-extensive bilateral** optic nerve enhancement, involving retrobulbar segment, **+perineural** involvement

How does this help narrow our differential diagnosis?

Laboratory Testing

Serum

- Unremarkable ESR, CRP, T-spot, ANA, ANCA, aquaporin-4-IgG, and serologies for syphilis, Lyme, bartonella, and HIV
- **MOG-IgG positive**

*live cell-based assay (Mayo clinic) for maximum sensitivity/specificity

CSF

- Opening pressure normal
- Unremarkable glucose, cell count, cultures
- CSF protein 62 (ULN 45)
- Identical oligoclonal bands in serum and CSF

Clinical and MRI Findings Characteristic of MOGAD-associated Optic Neuritis

- MOG-IgG+ optic neuritis typically presents with optic nerve swelling, commonly presents with bilateral involvement
- MOG-IgG+ commonly shows perineural enhancement on MRI
- MOG-IgG+ commonly shows longitudinally extensive optic nerve enhancement, with prominent involvement of the retrobulbar segment, on MRI

Akaishi Tet al. *J Neurol Neurosurg Psychiatry*. 2016;87(4):446-448. Akaishi Tet al. *Neurochem Int*. 2019;130:104 319. Chen J J et al. *Am J Ophthalmol*. 2018;195:8-15. Chen J J et al. *JAMA Ophthalmo l*. 2018;136(4):419-422. Chen Ji et al. *Mult Scler Relat Disord*. 2022;58:1035 25. Liu H et al. *Br J Opht halmol*. 2019;103(10):1423-1428. Lopez-Chir b ogaA S et al. *Neuroophth alm ology*. 2019;44(1):1-4. Mealy MA et al. *J Neurol Sci*. 2015;355(1-2):59-63. Peng Y et al. *Exp Ther Med*. 2018;16(2):950-958. Ramanat han Set al. *Mult Scler*. 2016;22(4):470-482. Zhao Y et al. *Br J Ophthalmol*. 2018 Oct ;102(10): 1372-1377.

MOG in the Optic Neuritis Treatment Trial

- 177 patients from the ONTT
- 3 with MOG-IgG
  - All had disc edema at presentation
  - 2 had recurrent optic neuritis
  - None developed MS

Chen JJ et al. *JAMA Ophthalmol*. 2018;136(4):419-422.

Management of MOGAD

- Early treatment with high-dose oral or IV steroids, typically followed by slow prednisone taper
- Treatment at onset of pain may even prevent vision loss
- MOG-IgG+ predicts higher risk of relapse than MS or seropositive NMOSD
- Prophylactic long-term immunosuppressive / immunomodulatory treatment may be considered in MOGAD, particularly if poor visual recovery or relapsing course

Chen JJ, Bhatti MT. *Curr Opin Neurol*. 2020;33(1):47-54. Chen JJ et al. *Neurology*. 2020;95(2):e111-e120. Jarius S et al. *J Neuroinflammation*. 2016;13:280. Jitprapaikulsan J et al. *Ophthalmology*. 2018;125(10):1628-1637. Pache F et al. *J Neuroinflammation*. 2016;13(1):282.

11

Case 2

27-year-old woman presents with 2 days of blurred vision in the left eye. No pain with eye movements.

Acuity 20/25 OD, counting fingers OS

+left RAPD

Anterior and posterior segment exams unremarkable

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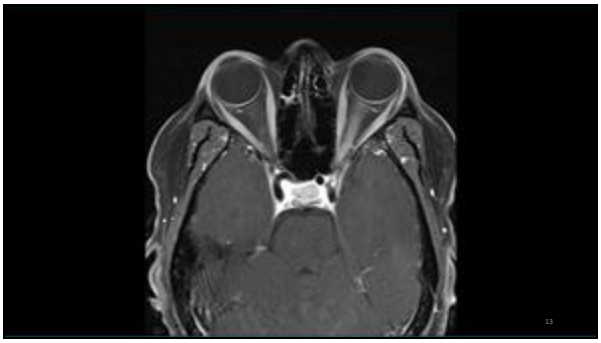
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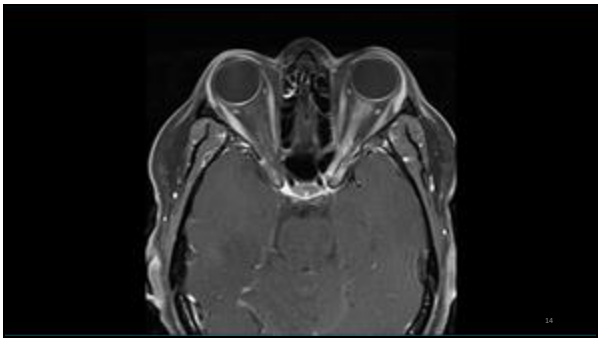
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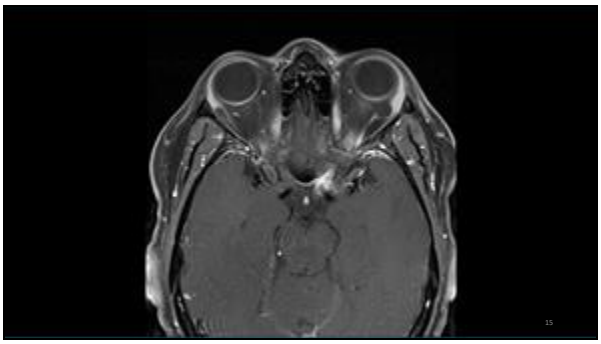
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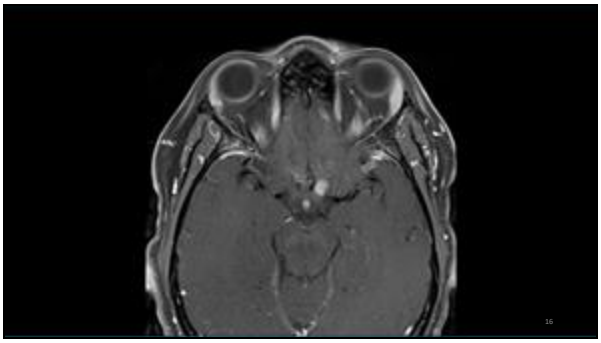
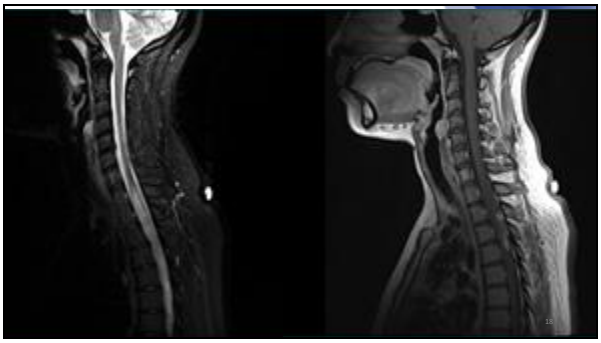


Clinical Presentation

- Acute / subacute, unilateral vision loss
- **No** pain with eye movements
- Decreased VA and color visio
- Central/cecocentral VF defect
- Normal appearing optic discs
- MRI: **Longitudinally-extensive** optic nerve enhancement, involving intracranial segment(s) of nerve

How does this help narrow our differential diagnosis? What other MRI findings would also help?



Neuromyelitis Optica (NMO)

- Previously considered a variant of MS, NMO now has been clearly shown to be a distinct disease
- Autoimmune astrocytopathy, in which CNS demyelination occurs due to a primary destruction of astrocytes
- Aquaporin-4 antibody has high sensitivity (68%-91%) and high specificity (85%-99%) for NMO

Papadopoulos et al, 2012

Diagnostic Criteria for NMOSD

1. At least 1 core clinical characteristic
    1. Optic neuritis
    2. Acute myelitis
    3. Area postrema syndrome
    4. Acute brainstem syndrome
    5. Symptomatic narcolepsy with correlating MRI lesions
    6. Symptomatic cerebral syndrome with correlating/typical brain lesions
2. AQP4-IgG positive
3. Exclusion of alternative diagnoses

Wingerchuk DM et al; International Panel for NMO Diagnosis. *Neurology*. 2015; 85(2): 177-89.

AQP4 in the ONTT

- 177 patients from the ONTT
- None with AQP4-IgG

Chen JJ. *JAMA Ophthalmol*. 2018;136(4): 419-422.

Management of Seropositive NMOSD

- Treatment with high dose IV steroids, followed by prednisone taper
- Early use of plasma exchange (even concomitant with IV steroids) may improve visual outcomes in these cases
- Any patient with seropositive NMOSD should be considered at risk for relapse indefinitely, and morbidity and duration of NMO relapses are more severe than those of MS or MOGAD. Therefore, patients need to be maintained on chronic immunosuppression
  - Rituximab (anti-CD20, off label)
  - Eculizumab (C5 complement inhibitor)
  - Inebilizumab (anti-CD19)
  - Satralizumab (anti-IL6R)

Bonnan M et al. *J Neurol Neurosurg Psychiatry*. 2018;89(4):346-351.

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Summary: Evaluation

- MS-related optic neuritis typically presents with normal optic disc appearance, unilateral involvement

  (Beck R, *N Engl J Med* 1993)
- MOGAD-related optic neuritis (MOG-IgG+) typically presents with optic nerve swelling, commonly with bilateral involvement

  (Akaishi T, *J Neurol Neurosurg Psychiatry* 2016; Akaishi T, *Neurochem Int* 2019; Chen JJ, *Am J Ophthalmol* 2018; Chen JJ, *JAMA Ophthalmol* 2018; Peng Y, *Exp Ther Med* 2018; Ramanathan S, *Mult Scler* 2016; Zhao Y, *Br J Ophthalmol* 2018)
- Seropositive NMO-associated optic neuritis (AQP4-IgG+) commonly presents with bilateral involvement

  (Ramanathan, *Mult Scler* 2016)

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Summary: Evaluation

- MOGAD-ON commonly shows perineural enhancement on MRI

  (Akaishi T, *J Neurol Neurosurg Psychiatry* 2016; Chen JJ, *Am J Ophthalmol* 2018; Liu H, *B J Ophthalmol* 2019; Lopez-Chiriboga AS, *Neuro-Ophthalmology* 2019)
- MOGAD and NMO-ON more commonly show longitudinally-extensive optic nerve enhancement on MRI than MS (Akaishi T, *Neurochem Int* 2019; Chen JJ, *Am J Ophthalmol* 2018; Mealy A, *J Neurol Sci* 2015; Ramanathan S, *Mult Scler* 2016)
- NMO-ON more commonly shows intracranial involvement; MOGAD-ON more commonly shows retrobulbar involvement

  (Mealy A, *J Neurol Sci* 2015; Peng Y, *Exp Ther Med* 2018; Ramanathan S, *Mult Scler* 2016; Song H, *J Ophthalmol* 2019; Zhao Y, *Br J Ophthalmol* 2018)

24

Summary: Prognosis

- NMO predicts worse visual outcome than MOGAD or MS

(Akaishi T, *Neurochem Int* 2019; Fernandes DB, *J Neuroophthalmol* 2012; Ishikawa H, *Ophthalmology* 2019; Jitprapaikulsan J, *Ophthalmology* 2018; Martinez-Hernandez E, *JAMA Neurol* 2015; Matiello M, *Neurology* 2008; Peng Y, *Exp Ther Med* 2018; Ramanathan S, *Mult Scler* 2016; Song H, *J Ophthalmol* 2019; Sotirchos ES, *Mult Scler* 2019; Zhao Y, Br J Ophthalmol 2018)

- MOGAD predicts higher risk of relapse than NMO or MS

(Pache, *Journal of Neuroinflammation* 2016; Jitprapaikulsan, *Ophthalmology* 2018)

25

Summary: Acute Management

- In MS-related optic neuritis, steroid treatment improves rate of recovery but not ultimate visual outcome; however, early high-dose steroid treatment for seropositive NMOSD- and MOGAD-related optic neuritis may improve visual outcomes (Trebst, *J Neurol* 2014; Handzic, *Ophthalmology* 2023)
- Early use of PLEX may be beneficial in optic neuritis (Chen, *Am J Ophthalmol* 2023), particularly in seropositive NMO (Bonnan, *J Neurol, Neurosurg, Psychiatry* 2018)

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Summary: Chronic Management

- Immunosuppressive/Immunomodulatory Therapy for NMOSD, MOGAD
  - Seropositive NMOSD
    - Long-term immunosuppressive treatment required
    - MS disease-modifying therapies may cause worsening in NMOSD
  - MOGAD
    - Long-term immunosuppressive/immunomodulatory treatment may be considered in MOGAD, in cases of recurrence and/or poor visual recovery. Generally, not required for single episode with good visual recovery.
    - Optimum choice of treatment for MOGAD is not clear, although IVIg may be more effective at preventing relapse than other agents (Chen, *Neurology* 2020)

Summary

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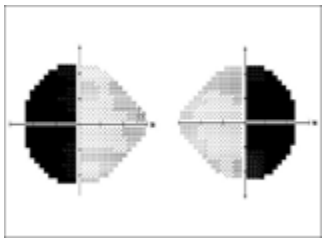
- Identification of optic neuritis presentations likely to represent NMOSD or MOGAD, and treatment accordingly, may improve outcomes in these groups
- High-dose PO steroids are ok in the treatment of acute optic neuritis
- Accurate diagnosis of the underlying condition (if any) is key for long-term management

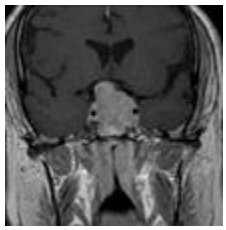
Five imaging studies every ophthalmologist needs to know

- Andrew G. Lee, MD
- Chair Ophthalmology, Houston Methodist Hospital, Professor of Ophthalmology, Neurology, & Neurosurgery, Weill Cornell Medical College; Adjunct Professor: Baylor College of Medicine, U. Iowa & Clinical Professor, UTMB Galveston, UT MD Anderson Cancer Center, U. Buffalo, SUNY

David refusing the armor of King Saul

No wonder I couldn't see that rock coming

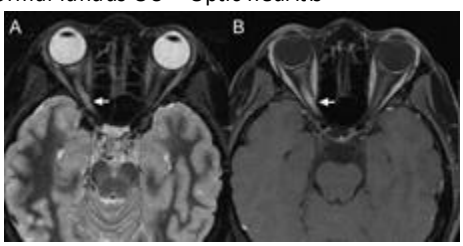




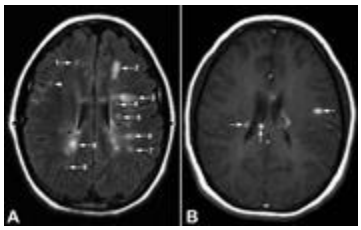
Five neuroimaging studies every ophthalmologist needs to know: Overview

- 1. T1 post contrast MRI of orbit with fat suppression: Optic neuritis
- 2. Sella sequence pre-contrast CT or MRI: Pituitary apoplexy
- 3. MRA or CTA: Posterior communicating artery aneurysm
- 4. CT/CTA or MRI/MRA head/neck/T2 in chest: Horner syndrome
- 5. CT of orbit/sinus: Rhinocerebral mucormycosis

20 y.o. WF: acute unilateral visual loss OD, central scotoma, pain with eye movement, RAPD OD, and normal fundus OU = Optic neuritis



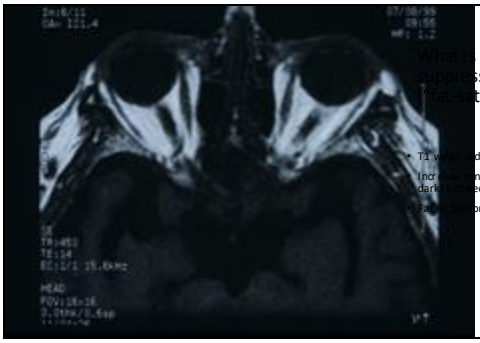
Optic neuritis: yes, MS but beware NMO if MRI negative for MS or bilateral ON



What happens if you don't give contrast?....

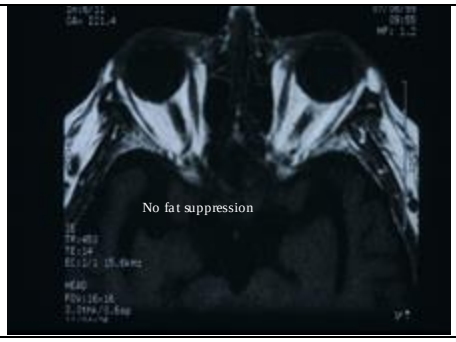
My house at NIGHT!!!

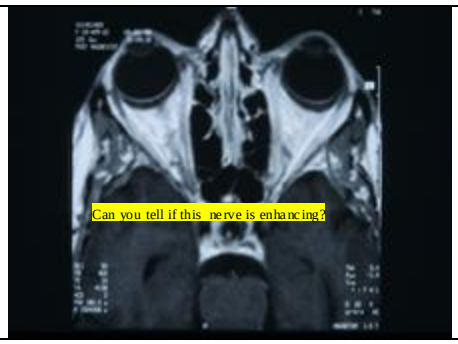
North Korea at night



What is Fat suppression ("fat-sat")? technique

- T1 weighted signal
- Increases contrast (light and dark) between structures
- Fat is "too bright" on T1





And the MRI of head was normal…..

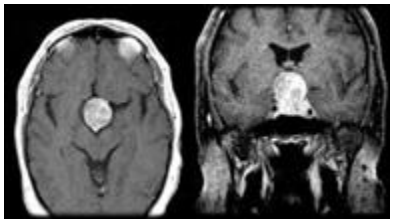
- WHY?

Polar bear in a snowstorm

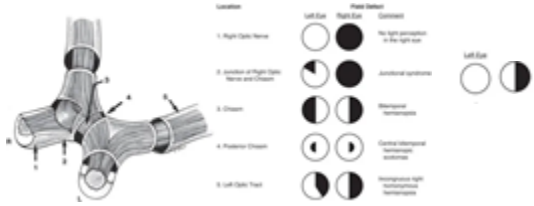
Always think about suprasellar lesions in visual field loss of any type

- Suprasellar masses produce bitemporal hemianopsia
- But also optic neuropathy, junctional visual field loss, and homonymous hemianopsia
- Big 5 in adults
  - Pituitary adenoma
  - Craniopharyngioma
  - Suprasellar aneurysm
  - Meningioma
  - Dysgerminoma
- Acute bitemporal hemianopsia can be life threatening pituitary apoplexy or ICA aneurysm

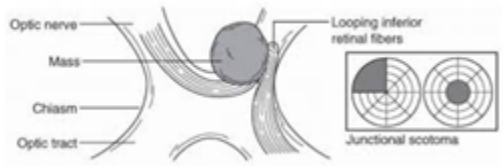
30 y.o. pregnant WF with bitemporal hemianopsia & worst headache of her life = pituitary apoplexy

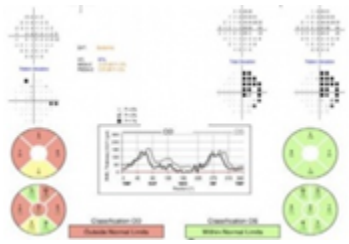


It can always be Chi (X)

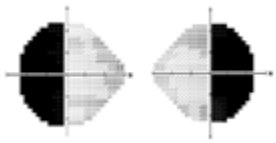


Junctional scotoma of Traquair





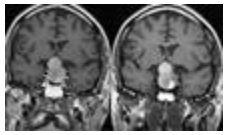
22 yo WF with worst HA of her life and bilateral visual loss. 20/20 OU. No RAPD. Normal fundus OU.

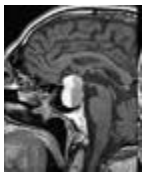


Panhypopituitarism after pituitary apoplexy

Endocrine defect	Percentage	References
Hypopituitarism	45-80	12,14,25,51
Adrenal insufficiency	60-75	11,25,51
Hypothyroidism	50-80	11,25,51
Hypogonadism	40-80	12,14,25,51
Growth hormone deficiency	90	69
Diabetes insipidus	5-20	14,54

Precipitating factors
Hypertension/hypotension
Major surgery
Coronary artery bypass grafting/stenting
Anticoagulation
Clotting disorder
Dynamic endocrine stimulation testing
Estrogen therapy
Dopamine agonist therapy
Head trauma
Radiotherapy
Pregnancy





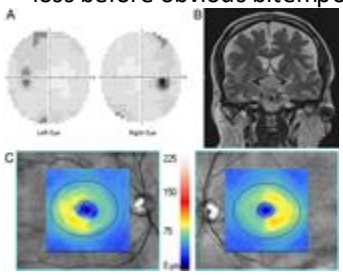
Acute & painful….Bitemporal hemianopsia

Never ignore "worst headache of my life"

Visual Outcome after Transsphenoidal Surgery in Patients with Pituitary Apoplexy

Bitemporal hemianopsia: Even orthopedic surgery knows this is chiasmal

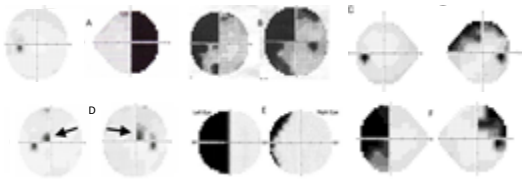
Macular ganglion cell loss can show binasal loss before obvious bitemporal hemianopsia



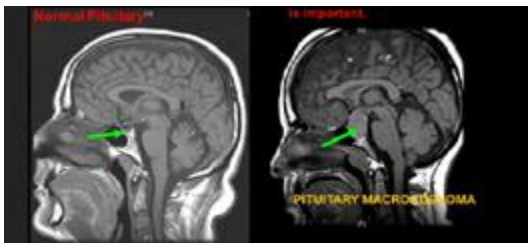
JNS JOURNAL OF NEUROSURGERY

Chiasmal damage shown by optical coherence tomography: case illustration

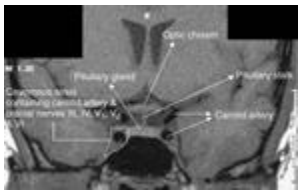
But all of these could be chiasmal syndromes

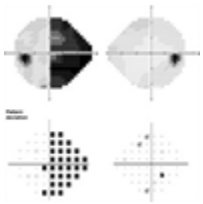


Normal pituitary anatomy

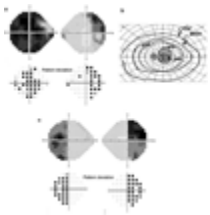


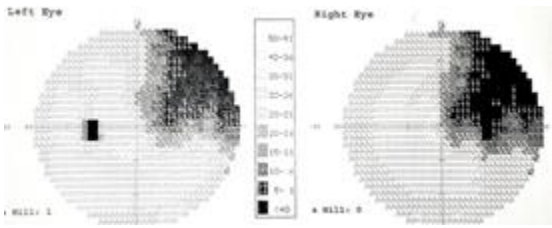
Normal coronal MRI anatomy

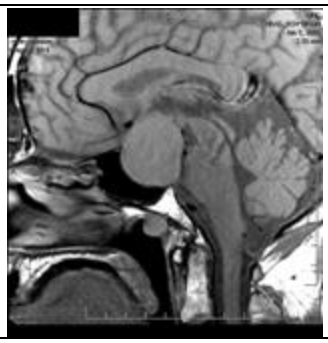




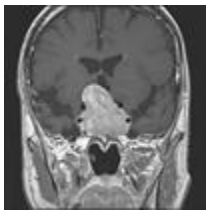
Junctional scotoma or bitemporal hemianopsia

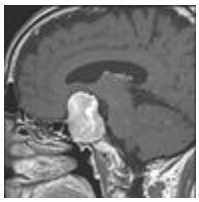


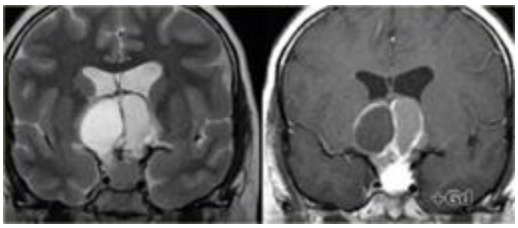


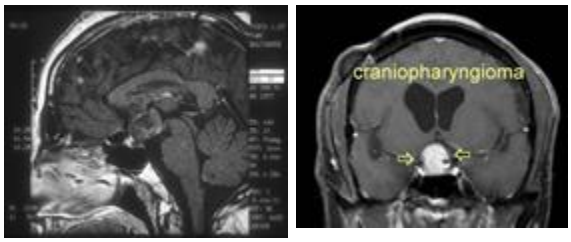


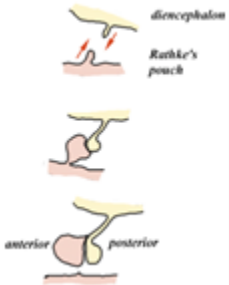
Pituitary adenoma: (“snowman”)

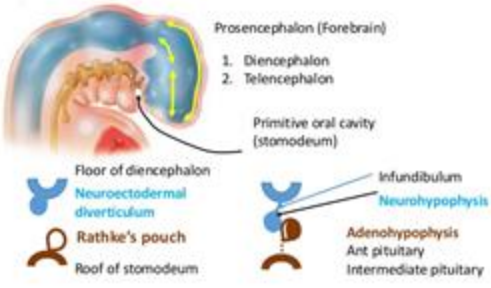


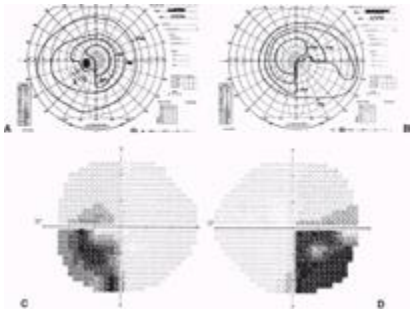




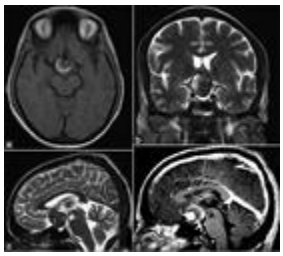

craniopharyngioma


diencephalon
Rathke's pouch
anterior
posterior

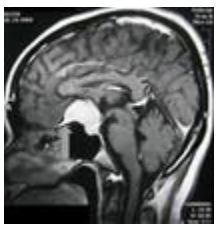

Prosencephalon (Forebrain)
1. Diencephalon
2. Telencephalon
Primitive oral cavity (stomodeum)
Floor of diencephalon
Neuroectodermal diverticulum
Rathke's pouch
Roof of stomodeum
Infundibulum
Neurohypophysis
Adenohypophysis
Ant pituitary
Intermediate pituitary

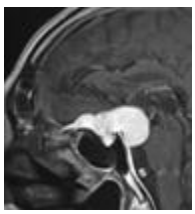


Suprasellar aneurysm ("black and white ball"): Flow void and thrombosis

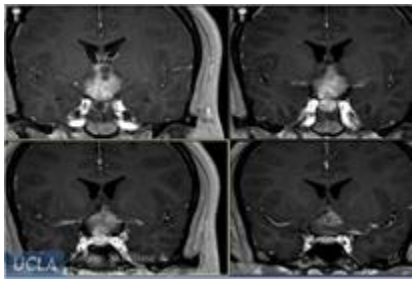


Suprasellar meningioma ("snail or bird")

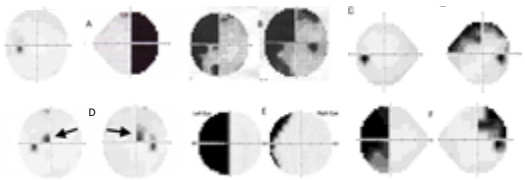




Suprasellar dysgerminoma



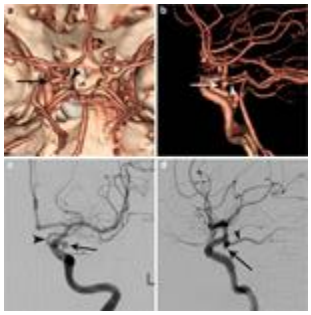
Bottom line all of these could be chi (X)



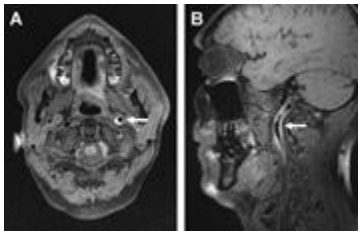
Summary: Chiasmal syndromes

- Yes, suprasellar masses produce bitemporal hemianopsia
- But also optic neuropathy, junctional visual field loss (JS, JST), and homonymous hemianopsia
- Top five in adults
  - Pituitary adenoma ("snowman")
  - Craniopharyngioma ("dirty snowball")
  - Suprasellar aneurysm ("black and white ball": flow void)
  - Meningioma ("snail tail")
  - Dysgerminoma (intrinsic intra-axial, young male)

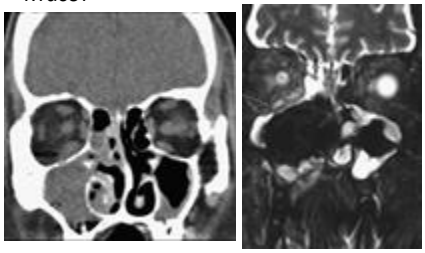
40 y.o. WF with painful, acute, pupil involved third nerve palsy OD = posterior communicating artery aneurysm



50 y.o. WF with acute, painful, anisocoria worse in the dark, 1 mm ptosis, dilation lag of pupil OS after MVA (whiplash) = Horner syndrome



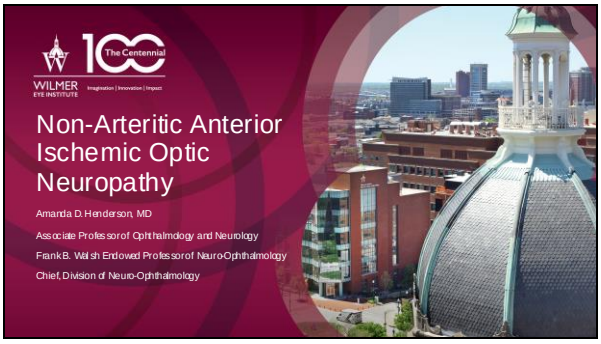
60 y.o. WF in DKA with acute, painful ophthalmoplegia, proptosis, & (RAPD) OD = Mucor



Medical Mycology Case Reports. 6: 2014, 51–54

Five neuroimaging studies every ophthalmologist needs to know: Summary

- 1. T1 post contrast MRI of orbit with fat suppression: Optic neuritis
- 2. Sella sequence pre-contrast CT or MRI: Pituitary apoplexy
- 3. MRA or CTA: Posterior communicating artery aneurysm
- 4. CT/CTA or MRI/MRA head/neck/T2 in chest: Horner syndrome
- 5. CT of orbit/sinus: Rhinocerebral mucormycosis



Disclosures

Wilmer Eye Institute
Johns Hopkins Medicine

Horizon Therapeutics/Amgen (Advisory Boards)

Catalyst Pharmaceuticals (Advisory Board)

Argenx (Clinical Trial Site)

Objectives

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By the end of this presentation, participants will be able to:

- Identify clinical and ancillary testing characteristics consistent with NAION
- Perform an evaluation to rule out mimickers of NAION
- Counsel patients with NAION

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65 yo man with acute vision loss OD, noticed upon awakening 3 days prior

Visual acuity OD 20/30, OS 20/20

Right relative afferent pupillary defect

Color vision full in both eyes

Anterior segment examination unremarkable

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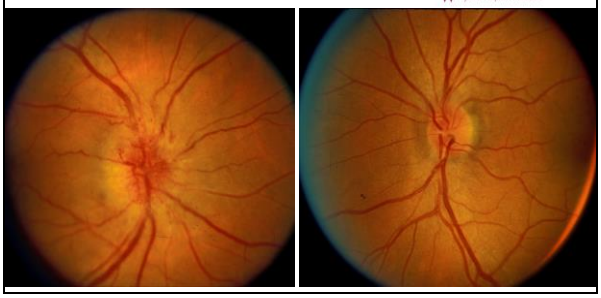

HVF

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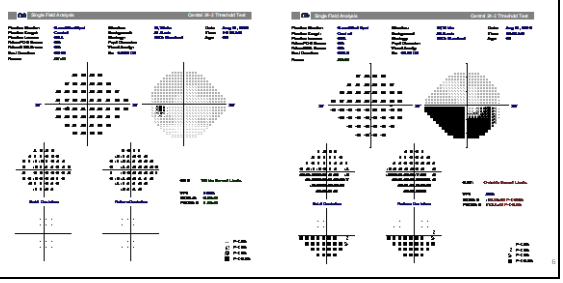

Differential Diagnosis

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- Anterior ischemic optic neuropathy
  - NAION
  - AAION (GCA)
- Optic neuritis

What additional information may be helpful?

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- History of Present Illness
  - Pain
    - Headaches
    - Scalp tenderness
    - Jaw pain with chewing
    - Polymyalgia rheumatica
    - Pain with eye movements
  - Constitutional symptoms
    - Fever
    - Weight loss
  - Other visual symptoms
    - Preceding episodes of transient vision loss, diplopia
- Past Medical History
  - Hypertension
    - If yes, when BP meds taken?
  - Diabetes
  - Obstructive sleep apnea (or symptoms to suggest, eg, STOP-BANG)
  - Medication history (PDE5 inhibitors, semaglutide??)
  - Cancer history

Patient History

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- History of Present Illness
  - ~~Pain~~
    - ~~Headaches~~
    - ~~Scalp tenderness~~
    - ~~Jaw pain with chewing~~
    - ~~Pain with eye movements~~
    - ~~Polymyalgia rheumatica~~
  - ~~Constitutional symptoms~~
    - ~~Fever~~
    - ~~Weight loss~~
  - ~~Other visual symptoms~~
    - ~~Preceding episodes of transient vision loss, diplopia~~
- Past Medical History
  - Hypertension
    - If yes, when BP meds taken?
  - Diabetes
  - ~~Obstructive sleep apnea~~ (or symptoms to suggest, eg, STOP-BANG)
  - Medication history (PDE5 inhibitors, semaglutide??)
  - ~~Cancer history~~

NAION

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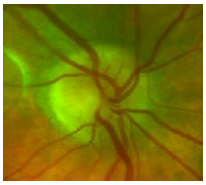
- Men and women equally affected
- Most have underlying risk factors (but may be undiagnosed at time of onset)
  - Systemic disease risk factors
    - Hypertension
    - Diabetes
    - Obstructive sleep apnea
  - Anatomic risk factors
    - Disc-at-risk
    - Disc drusen
- Occurs in 3-10 per 100,000



Johnson LN, Arnold AC. Incidence of nonarteritic and arteritic anterior ischemic optic neuropathy. Population-based study in the state of Missouri and Los Angeles County, California. J Neuroophthalmol. 1994 Mar;14(1):38-44.
Hattenhauer MG, Leavitt JA, Hodge DO, Grill R, Gray DT. Incidence of nonarteritic anterior ischemic optic neuropathy. Am J Ophthalmol. 1997 Jan;123(1):103-7.

NAION

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- Men and women equally affected
- Most have underlying risk factors (but may be undiagnosed at time of onset)
  - Systemic disease risk factors
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  - Anatomic risk factors
    - Disc-at-risk
    - Disc drusen
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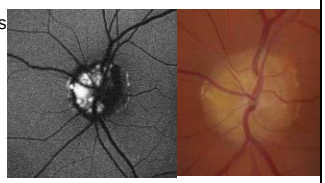

Management Questions

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- What work up is indicated?
- Is there treatment?

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What work up is indicated?

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Evaluation

- **Rule out giant cell arteritis (history, ESR, CRP, platelet count)**
- Primary care work up and management underlying vascular risk factors: hypertension, hypercholesterolemia, diabetes
- Sleep study to evaluate for obstructive sleep apnea, if not yet diagnosed

Chang MY, Keltner JL. Risk Factors for Fellow Eye Involvement in Nonarteritic Anterior Ischemic Optic Neuropathy. J Neuroophthalmol. 2019 Jun;39(2):147-152.
Sun MH, Lee CY, Liao YJ, Sun CC. Nonarteritic anterior ischaemic optic neuropathy and its association with obstructive sleep apnoea: a health insurance database study. Acta Ophthalmol. 2019 Feb;97(1):e64-e70.

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STOP BANG Questionnaire for Obstructive Sleep Apnea

** Snoring? (Loud)
** Tired? (Daytime fatigue, falling asleep while driving or talking)
** Observed? (Observed choking/gasping or stopped breathing during sleep)
** Pressure? (Hypertension)
** BMI >35?
** Age >50?
** Neck size large? (17" if male, 16" if female)
** Gender = male

** Total points

[Low OSA risk </=2 points
Intermediate OSA risk 3-4 points
High OSA risk 5+ points
or
2+ STOP points + male gender
or
2+ STOP points + BMI >35
or
2+ STOP points + large neck]

Evaluation

- **Rule out giant cell arteritis (history, ESR, CRP, platelet count)**
- Primary care work up and management underlying vascular risk factors: hypertension, hyperchdesterolemia, diabetes
- Sleep study to evaluate for obstructive sleep apnea, if not yet diagnosed
- Hypercoagulable work up in select sub-groups??
  - Young patients, + personal or family history of thromboembolism, negative history of cardiovascular risk factors
- Neuroimaging with MRI brain and orbits w and wo contrast
  - Reasonable, to exclude mimickers (specifically, optic neuritis)
- Additional lab work up
  - Reasonable to consider serum testing for syphilis, Lyme

Kuhli-Hattenbach C, Scharer I, Lüchtenberg M, Hattenbach LO. Selective thrombophilia screening of patients withnonarteritic anterior ischemic optic neuropathy. Graefes Arch Clin Exp Ophthalmol. 2009 Apr;247(4):485-90

16

Is there treatment?

17

Summary of Evidence- Treatment

- **None clearly beneficial**
- **ASA**: No benefit for visual outcome (Botelho, 1995); Benefit for prevention of second eye involvement controversial (Beck, 1997; Kupersmith, 1997; Newman, 2002)
- **Steroids**: Oral steroids controversial (Hayreh, 2008; Rebolleda, 2013; Pakravan, 2017; Saxena 2018; Chen 2019)
- **ONSF**: Not beneficial and may even be harmful (Ischemic Optic Neuropathy Decompression Trial Research Group, 1995)
- **Brimonidine**: Not beneficial (Fazzone, 2003; Wilhelm 2006)
- **Anti-VEGF agents**: Some individual reports of benefit, but no benefit in nonrandomized controlled trial (Bennett, 2007; Rootman, 2013)
- **Phenytoin**: Not beneficial, but randomized study begun 3 months after onset (Ellenberger, 1974)
- **Erythropoietin**: Controversial (Modarres, 2011; Pakravan, 2017; Nikkhah, 2020)
- **Hyperbaric oxygen**: Not beneficial (Arnold, 1996)
- **QPI-1007**: caspase 2 inhibitor; prospective, masked, randomized trial- did not meet primary end point; possible benefit for patients with worse vision at presentation in post hoc analysis (Levin, AAO, 2024)
- **RPh201**: gum mastic; prospective, masked, randomized trial, stopped early due to apparent lack of efficacy

Counseling

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- Prognosis
  - Recurrence in the same eye is rare
  - Risk of fellow eye involvement ~15-20%
- Vascular risk factor management, evaluation
- Medications
  - Aspirin?- controversial
  - BP meds in the morning (or before 5pm)- if ok with cardiologist/pcp
  - Avoid PDE5 inhibitors
  - Avoid semaglutide/GLP1 agonists?

Hayreh SS, Podhajsky PA, Zimmerman B. Ipsilateral recurrence of nonarteritic anterior ischemic opticneuropathy. Am J Ophthalmol. 2001 Nov;132(5):734-42.
Newman NJ, Scherer R, Langenberg P, Kelman S, Feldon S, Kaufman D, Dickens M, Ischemic Optic NeuropathyDecompressionTrial Research Group. The fellow eye inNAION: report from the ischemic opticneuropathydecompression trial follow-up study. Am J Ophthalmol. 2002Sep;134(3):317-28.
Pomeranz HD. The Relationship Between Phosphodiesterase-5 Inhibitors and Nonarteritic Anterior Ischemic Optic Neuropathy. J Neuroophthalmol 2016; 36 (2):193-196.
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Hathaway JT, Shah MP, Hathaway DB, Zekeri SM, Krasniqi D, Gittinger JW Jr, Costan D, Malleryk, Abboud B, Bouffard M, Chwalisz BK, Estrela T, Rizzo J 3rd. Risk of Nonarteritic Anterior IschemicOptic Neuropathyin Patients Prescribed Semaglutide. JAMA Ophthalmol. 2024Jul 3:e242196.

NAION- Take Home Points

Wilmer Eye Institute
Johns Hopkins Medicine

- Key diagnostic features include history (typically painless, no associated systemic symptoms), examination findings (optic disc swelling, contralateral disc-at-risk, altitudinal field defects most common though field defects can vary), exclusion of mimickers (GCA, optic neuritis)
- Risk factor management recommended, to potentially reduce risk of second eye involvement
- No proven treatment to improve the vision

Unexplained visual loss in seven easy steps

- Andrew G. Lee, MD
- Chair Ophthalmology, Houston Methodist Hospital, Professor, Weill Cornell MC; Adjunct Professor, Baylor COM, U Iowa, UTMB Galveston, UT MD Anderson Cancer Center, U. Buffalo (SUNY)

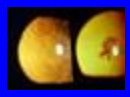
Step 2: Complete eye exam

- By complete I mean….complete (don't use short cuts in your neuro-op patients!)
- Check relative afferent pupillary defect yourself
- Check color vision & visual field
- Ophthalmoscopy
  - High magnification & high clinical suspicion

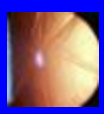
Don't take the shortcut

#3: Complete eye exam

- Slit lamp biomicroscopy
  - Look after dilation
  - Beware oil droplet cataract
  - Look for posterior subcapsular cataract
  - Match lens opacity to visual acuity
  - Retroillumination



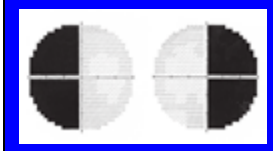
Look at lens & grade opacities ("NSC/PSC = 20/30" or ≠ "20/30")

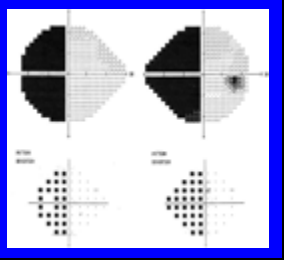


Step 4: Formal visual field

- "Unreliable" visual field is the same information as NO visual field performed
- Confrontation visual field = minimum
- Media & refractive etiologies rarely produce field defects
- Any respect of vertical meridian significant

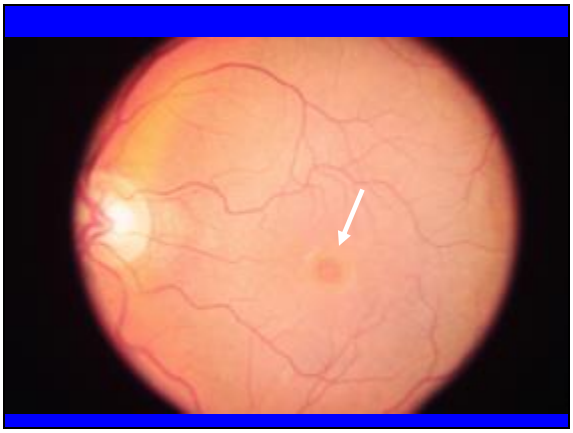
What will be the visual acuity, pupil, SLE, Motility, Ext, Fundus and OCT exam in these cases?

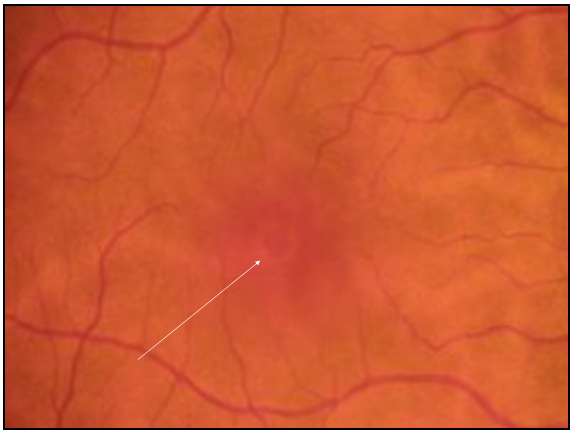


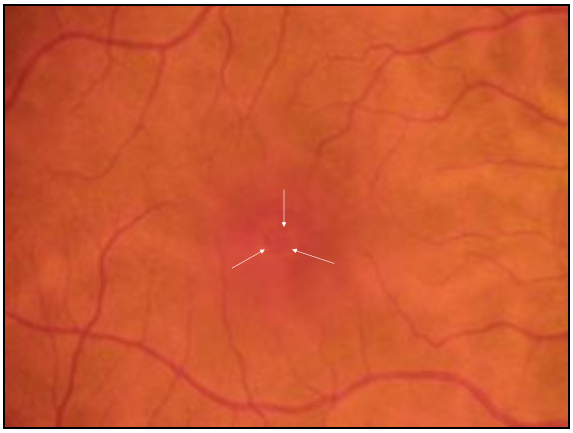


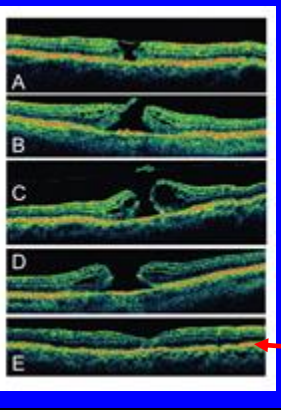
Look At The Macula

- Subtle macular lesions can be missed without high magnification and high suspicion (e.g. macular hole, cystoid macular edema)
- "WNL" should mean "*within normal limits*" *NOT "WE NEVER LOOKED'*







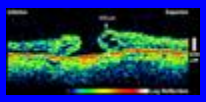


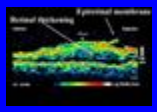
Don Gass MD saw all of this without OCT!

Post op!
www.revophth.com

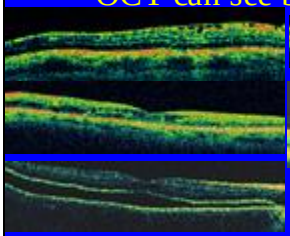
Step 5: OCT in Unexplained visual loss? Is it retina or optic nerve?

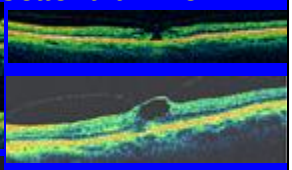
- Macular edema or macular hole
- Epiretinal membrane
- Cystoid macular edema or subretinal fluid
- Vitreous traction on macula or optic nerve





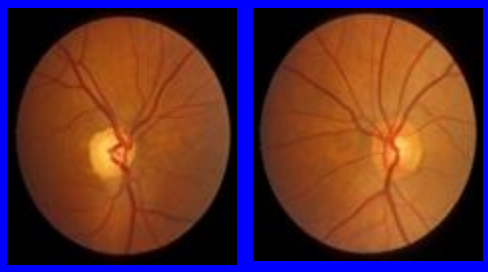
OCT can see better than me



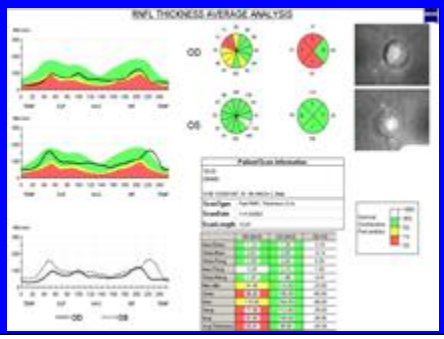


Epiretinal membrane
Macular hole
Vitreomacular traction
Serous fluid under retina
Cystoid macular edema

Determination of Pallor vs No Pallor



OCT can see better than me

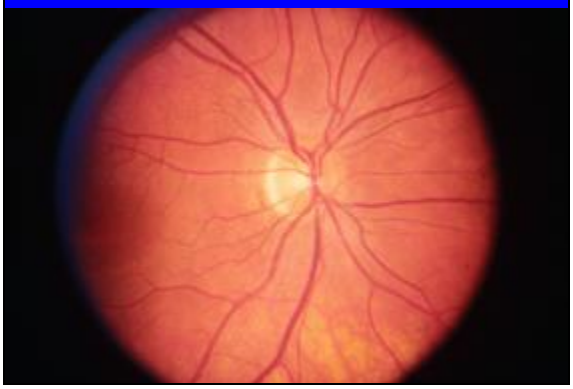


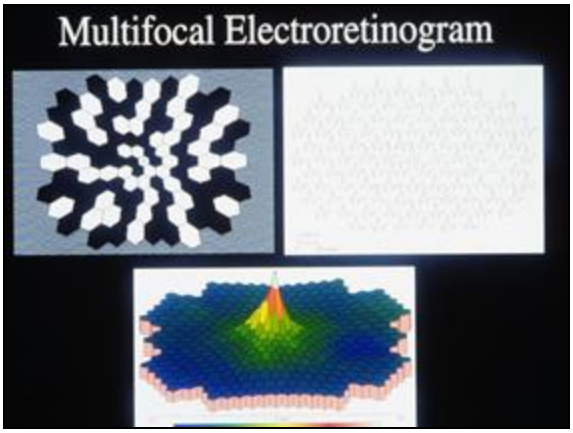
Am I pale?

Consider Ancillary Testing

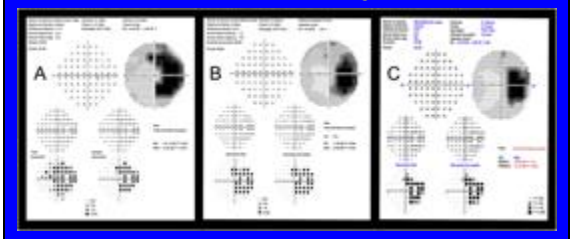
- Fluorescein angiography/OCT
  - If I see something funny in the macula
- Electrophysiology if it "smells like retina"
  - Big blind spot with normal peripapillary retina
  - Ring scotomas
  - Photopsias
  - Diffuse retinal arteriolar narrowing

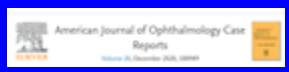
20 y/o WF with acute loss of visual field RE & photopsias



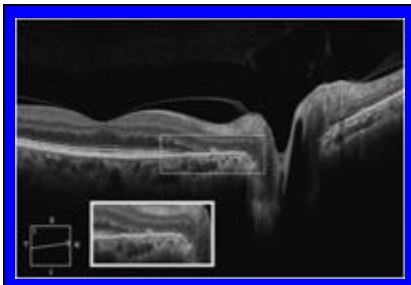


Enlarged blind spot OD: Acute, one month, and one year later

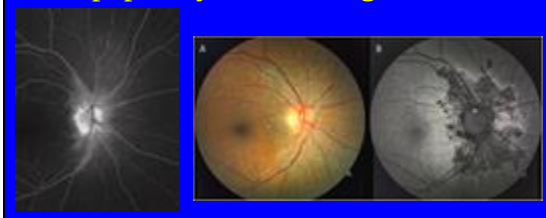




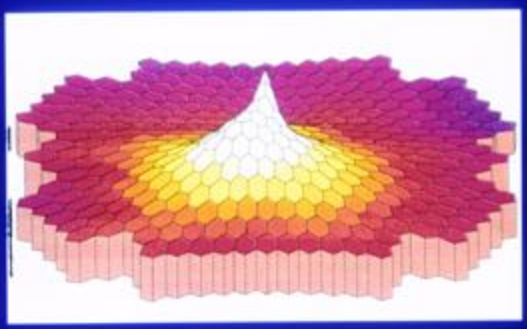
MERG shows depression corresponding with HVF



Acute: FFA hyperfluorescence Peripapillary RPE change over time

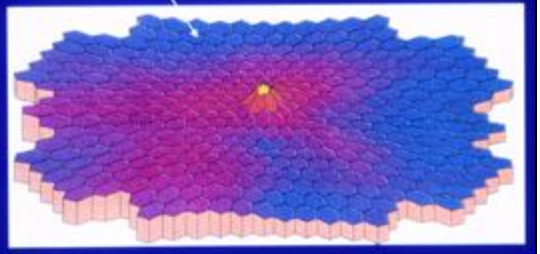


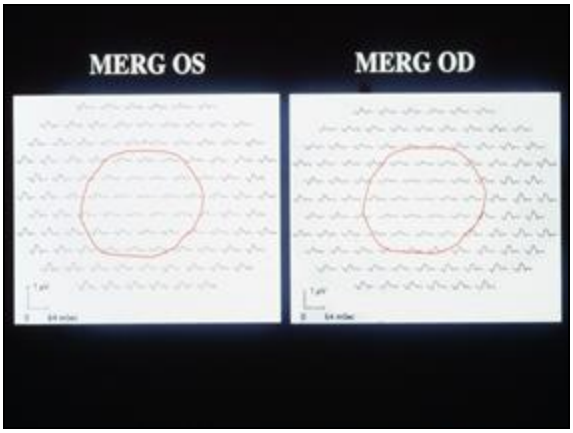
MERG: Normal



MERG

Markedly reduced amplitudes





Step 6: Rule out optic neuropathy

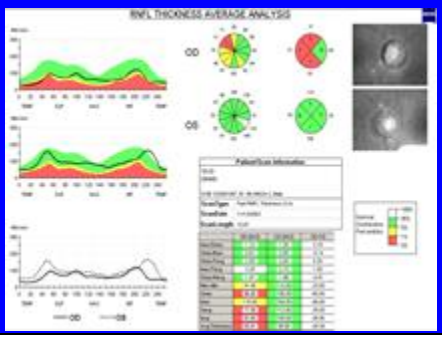
- Look for subtle signs of optic neuropathy
  - Decreased color vision
  - Relative afferent pupillary defect
  - OCT abnormal
  - Mild disc pallor or disc edema
  - Abnormal visual field
- If you miss a non-optic nerve cause for visual loss (PSC, ERM, refractive) it is no big deal
- If you miss an optic neuropathy it could be a big deal (compressive optic neuropathy)

Optic Atrophy Not Augenblick!

Is this nerve pale? Mild pallor? Temporal pallor? Optic atrophy?

Look for clinical signs of optic neuropathy (RAPD, visual field, fellow eye, OCT)

OCT can see better than me



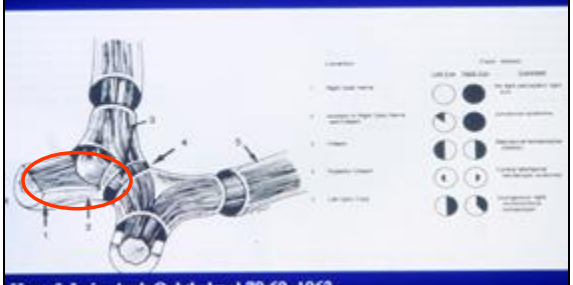
Why optic atrophy is dangerous?

- 50 patient clinic day
- Patients #1-49
  - Dx: Cataract; Plan: CE/IOL OD
  - Dx: ARMD (dry); Plan: AREDS vitamins
  - Dx: NPDR; Plan: Glucose control
  - Dx: RD; Plan; SB
- Patient #50: Dx = optic atrophy
- THIS IS NOT A DIAGNOSIS!

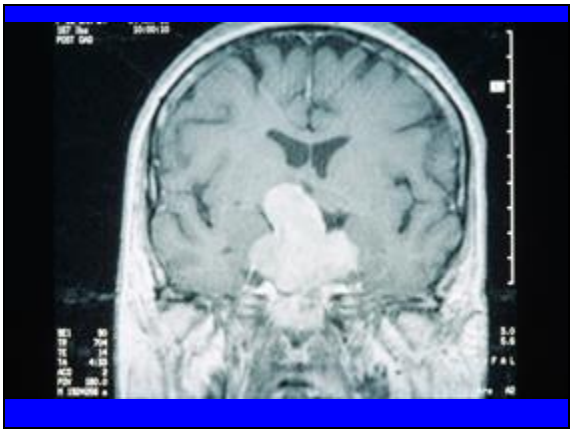
Always do a formal visual field in unexplained optic atrophy




CHIASMAL SYNDROMES



Hoyt & Luis. Arch Ophthalmol 70:69, 1963.



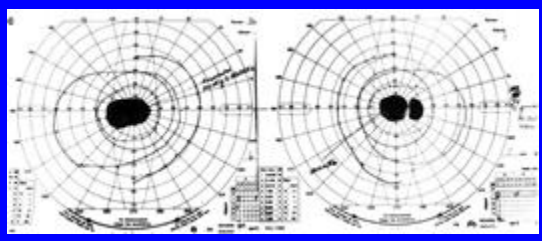
The usual suspects (labs)

- Syphilis (TPPA, RPR)
- Lyme disease (if travel to endemic area like USA)
- Tuberculosis (if travel to or from endemic area)
- Sarcoidosis (granulomatous findings)
- Infectious etiologies (if vitreous cells or anterior segment inflammation

When do I order B12/folate

- Central or cecocentral scotoma OU
- I don't order for unilateral optic atrophy
- History of nutritional deficiency risk factor (gastric surgery, ETOH)
- Consider Leber testing as well

Cecocentral or central scotoma



http://www.nature.com/eye/journal/v18/n11/images/6701591f1.jpg

I only have one glass of alcohol per day

Round up the usual suspects but…..

Rule out optic neuropathy

- Step 1: Make sure that it is real optic atrophy (vs. physiologic pallor)
- Step 2: Directed history and exam
- Step 3: Think common etiologies first
- Step 4: Consider imaging vs. observation
- Step 5: Direct laboratory evaluation based upon your pretest likelihood of disease (i.e. your clinical suspicion)

Step 7: Prove non-organic before labeling patient non-organic

- Non-organic = preferred sign
  - Outdated terms or terms which imply psychologic motivation (hysterical, malingerer)
- Functional visual loss = preferred DSMV dx
- Do you really know they are faking?
- Do you know their motivation?
- They might be organic with overlay!

Seven steps in unexplained visual loss

1. Insure visual loss = actual chief complaint
2. Complete eye exam every time (no shortcuts)
3. Special effort to detect subtle causes of visual loss
4. Formal visual field if unexplained symptoms
5. Special tests (e.g., MERG, OCT, fluorescein angiography, neuroimaging if indicated)
6. Rule out optic neuropathy or hemianopsia
7. Rule out ORGANIC and prove non-organic BEFORE labeling someone as such

Chief complaint: NONE

- 73-year-old WF
- Chief complaint: NONE now (2010)
- PMH: Paraneoplastic optic neuropathy, recovered
- CXR: Small cell carcinoma of lung
- Resected, chemotherapy, radiation in 1997

- Published: Luiz JE, Lee AG, Keltner JL, Thirkill CE, Lai EC. Paraneoplastic optic neuropathy and autoantibody production in small-cell carcinoma of the lung. J Neuroophthalmol. 1998;18:178–181.

Follow up 2010

- Pt: "You don't remember me do you Dr. Lee?"
- Me: "Well,…I um….sure…maybe"
- Pt: "I had lung cancer & you found it thru my eye"
- Me: "Really"
- Pt: "Yeah, you wrote it up in a journal"
- Me: "Oh, yeah, sure, now I remember. How are you, why are you coming today?"
- Pt: "I just wanted to tell you that I was still alive and it is been 14 years, so thanks."

Two publications from one patient!

Case Reports > J Neuroophthalmol. 1998 Sep;18(3):178-81.

Paraneoplastic optic neuropathy and autoantibody production in small-cell carcinoma of the lung

J E Luiz [1], A G Lee, J L Keltner, C E Thirkill, E C Lai

Case Reports > J Neuroophthalmol. 2010 Dec;30(4):387. doi: 10.1097/WNO.0b013e3181fd9435.

Long-term survivor of paraneoplastic optic neuropathy

Derrick Pau, Sushma Yalamanchili, Andrew G Lee

Longest known survivor

Long-Term Survivor of Paraneoplastic Optic Neuropathy

Small cell lung cancer carries a very poor long-term prognosis. In a survey performed at the Mayo Clinic from 1997 to 2003, the 5-year survival rate was only 9% (1). In addition, to our knowledge, the longest published survival duration for paraneoplastic optic neuropathy secondary to small cell lung cancer has been 8 years (2). We wish to provide an update on a patient previously reported by one of us (A.G.L.) in this Journal in 1998 (3) who remained 14 years later without evidence of tumor recurrence and believed to be in clinical remission. The earlier detection of the tumor from her neuro-ophthalmologic examination followed by timely systemic treatment may have contributed to her favorable outcome. To the best of our knowledge, she is the longest survivor of paraneoplastic optic neuropathy secondary to small cell lung cancer. At the time of her diagnosis, she underwent surgery, chemotherapy, and radiation therapy and was believed to be in remission at the last follow-up.

The patient, a 75-year-old white woman, was last seen in the neuro-ophthalmology clinic on July 20, 2010. She was complaining of blurred vision in the left eye that had worsened since sustaining a fall on March 1, 2010. She was seen by her neurologist who obtained a brain MRI that showed no focal lesions.

in March 2010 showed no evidence of recurrent or metastatic disease. The patient returned to The Methodist Hospital after 10 years of follow-up to specifically report on her progress and survival from small cell carcinoma of the lung.

Derrick Pau, MD
Sushma Yalamanchili, MD
Department of Ophthalmology, The Methodist Hospital Houston, Texas

Andrew G. Lee, MD
Department of Ophthalmology, The Methodist Hospital Houston, Texas
Departments of Ophthalmology, Neurology, and Neurosurgery, Weill Cornell Medical College New York, New York
Department of Ophthalmology, University of Iowa Hospitals and Clinics Iowa City, Iowa
Department of Ophthalmology, UTMB Galveston Galveston, Texas

Thanks for your time and attention

- Andrew G. Lee, MD
- Chair Ophthalmology, Houston Methodist Hospital, Professor of Ophthalmology, Neurology, & Neurosurgery, Weill Cornell Medical College; Clinical Professor, UTMB Galveston; UT MD Anderson Cancer Center; Adjunct Professor, Baylor COM, U. Iowa and U. Buffalo, SUNY

Optic neuritis: The good, the bad, and the ugly (ON, MOG, NMO)

Andrew G. Lee MD
Houston, Texas, USA

Weill Cornell Medical College

Methodist

Idiopathic ("the good"); MS ("the bad"): NMO/MOG ("the ugly")

1978: I wanted to be a doctor… 2nd choice Jedi knight

I wanted to help people …but I also wanted a superpower and also to be a spaceman

- It turns out superpowers are real
- The power to detect disease and death by looking in people's eyes
- A real Jedi superpower

The Jedi superpower: The force

I have no financial interest but I have a definite interest (in you)

Your great responsibility

- Doctor first
- Ophthalmologist second
- Vitreoretinal surgeon third

Overview: You need to test for MOG and NMO because….

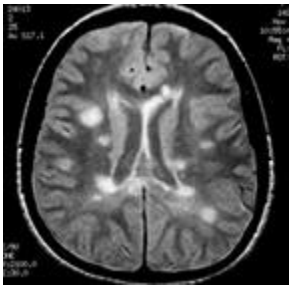
- It could be good (optic neuritis gets better)
- It could be bad (MOG needs IV steroids and may need immunosuppression)
- It could be ugly (NMO can blind you and without treatment can paralyze you)

What is typical ON?...Dad's rule of ducks

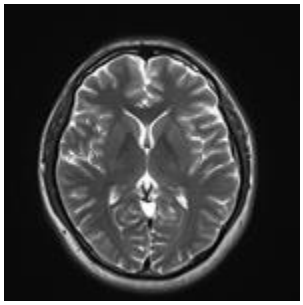
Typical duck (optic neuritis): "The Good"

- 20 yo white woman
- Acute unilateral loss of vision
- RAPD
- Pain with eye movement
- Normal fundus
- Recovers with or without steroids

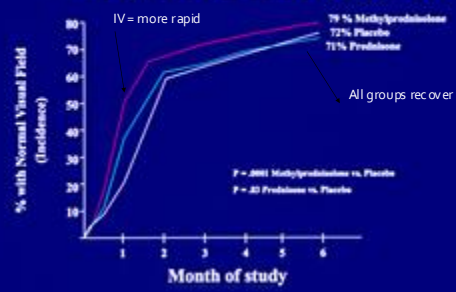
Typical MS optic neuritis (the bad)



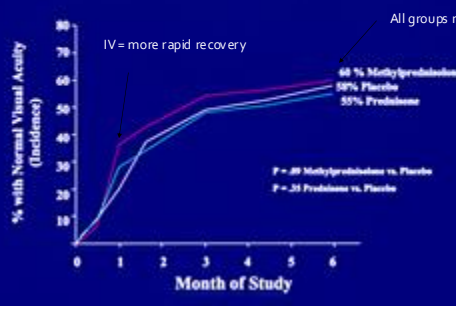
Good news: Your MRI was normal
Bad news: Your MRI was normal

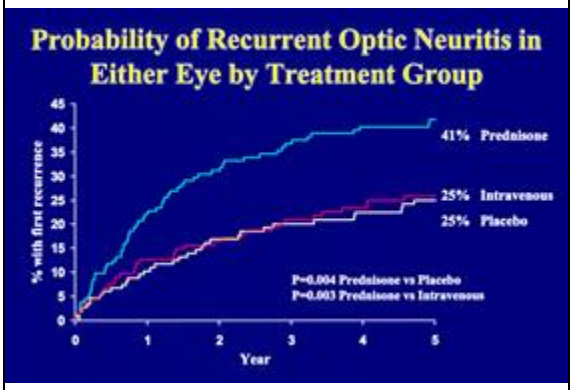


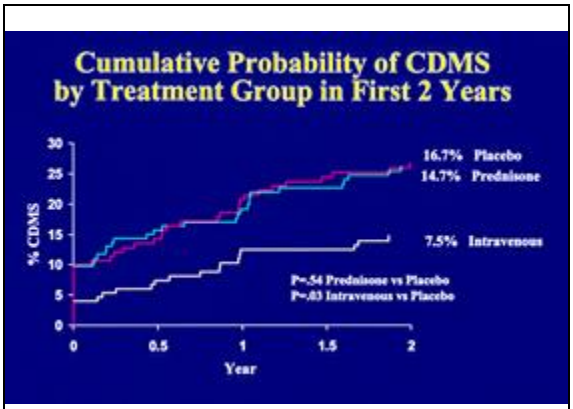
Cumulative Rates of Recovery of Normal Visual Field in First 6 Months



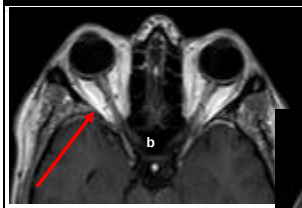
Cumulative Rates of Recovery of Normal Visual Acuity in First 6 Months



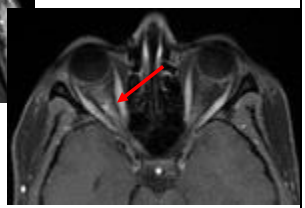




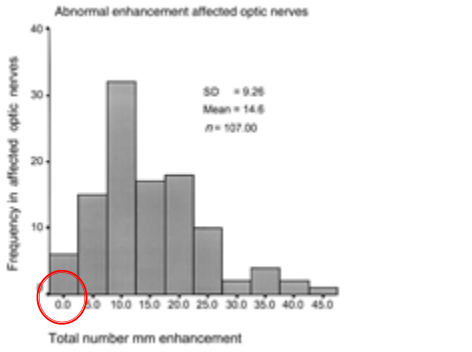
FAT SUPPRESSION POST GADOLINIUM



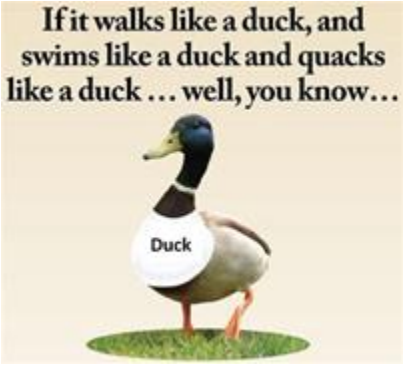
With fat suppression: Fat NOT bright on T1 MRI. Gad enhancing ON



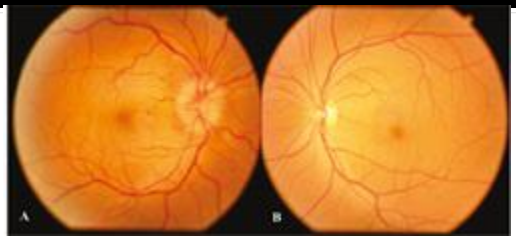
No fat suppression: Fat bright on T1 MRI



 Kupersmith M J et al. Brain 2002;125:812-822 BRAIN



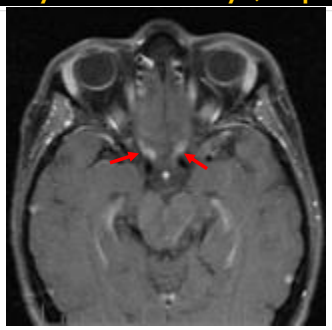
Brazil: Chikungunya, HIV, FTA, RPR, Dengue



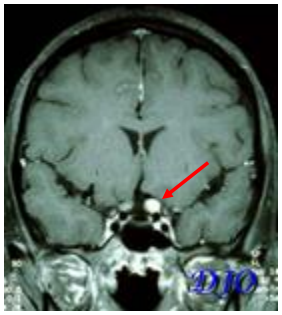
Revista Brasileira de Oftalmologia

CASE REPORT

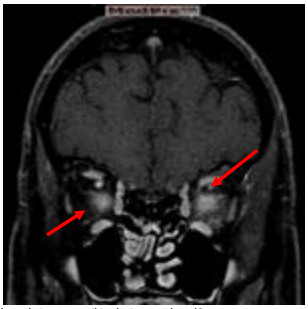
Beware bilateral ON enhancement especially intracranially (no pain)



Beware enhancing & enlarged ON



Beware enhancement outside of nerve itself….



http://rad.usuhs.edu/synapse/kiosk_image.html?

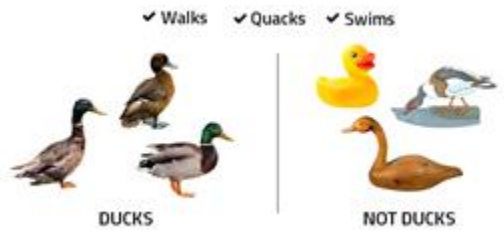
But what if the brain MRI is normal?

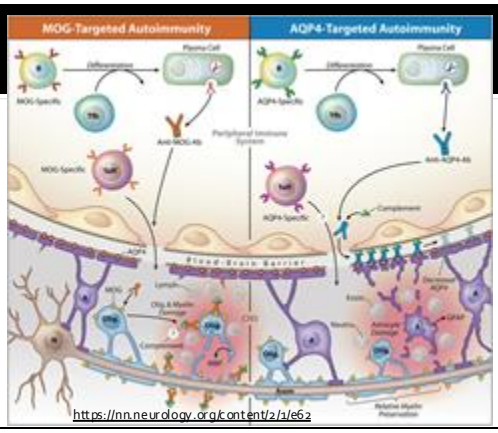
- The good: Optic neuritis, gets better, no MS
- The bad: MS (white matter lesions)
- The ugly: No MS lesions = might get ugly (NMO/MOG)

Atypical duck (optic neuritis):

- 60 (rather than 20) yo non-white woman
- Acute bilateral (rather than U/L) loss of vision
- No RAPD (because bilateral)
- Severe pain with eye movement
- Swollen disc(s) rather than normal fundus
- Fails to recover with or without steroids

Not a duck





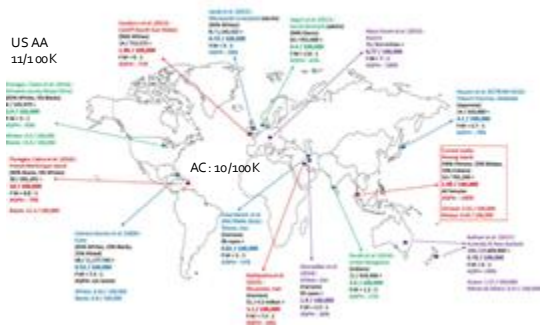
https://nn.neurology.org/content/2/1/e62

NMO

- Affects women more
- Disproportionately affects African-Americans, Afro-Caribbeeans, & Asians
- Asian form of MS more like NMO
- Most NMO patients are misdiagnosed initially as MS

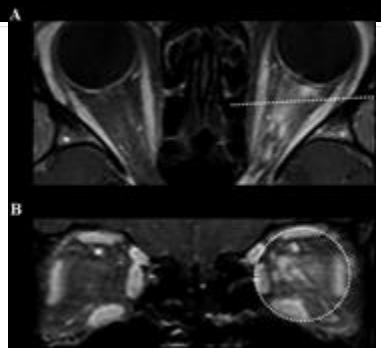
Wine countries

World Map Showing Population-Based Prevalence Studies of NMOSD



MS and Related Disorders. https://doi.org/10.1016/j.msard.2017.10.015

MOG MRI sheath enhancment



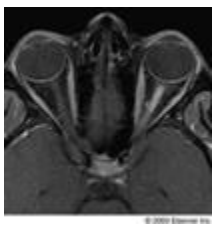
MOG

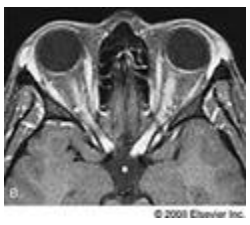
- Myelin oligodendrocytic glycoprotein (MOG)
- A more aggressive antibody mediated inflammatory optic neuropathy
- Not as bad generally as NMO but can be

Neuromyelitis optica (NMO)

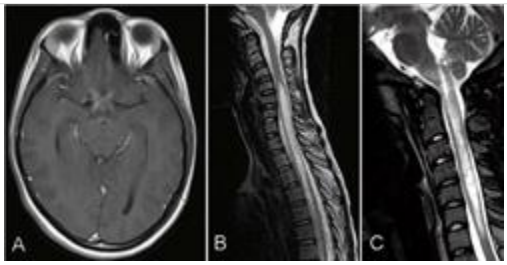
- NMO
  - Optic neuritis (bilateral, sequential or simultaneous)
  - Transverse myelitis (longitudinal > 3 segments)
  - NMO IgG
- Optic neuritis (but not typical)
  - Tends not to recover
  - Bilateral
- Brain MRI either normal ornot typical white matter lesions for MS
- LP pleocytosis (> 50 WBC)

Unilateral vs. Bilateral (adults) ON



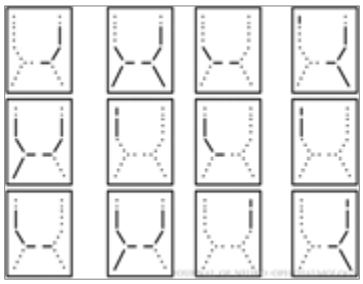


Longitudinally extensive lesions



https://www.researchgate.net/figure/Typical-imaging-findings-of-optic-neuritis-and-longitudinally-extensive-transverse_fig2_230616585

Pattern of visual pathway involvement in NMO on MRI

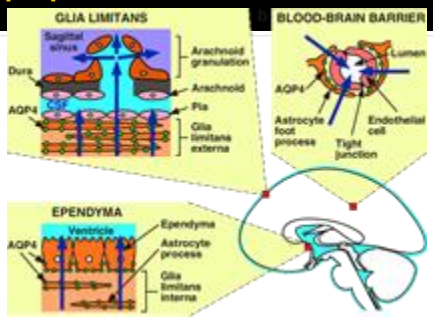


Distinguishing Optic Neuritis in Neuromyelitis Optica Spectrum Disease from Multiple Sclerosis: A Novel Magnetic Resonance Imaging Scoring System

Storoni, Mithu ;Davagnanam, Indran; Radon, Mark;Siddiqui, Aks; Plant ,Gordon T.
Journal of Neuro-Ophthalmology31(2): 123-123 June 2011.
doi: 10.1097/WNO.0b013e318213ed

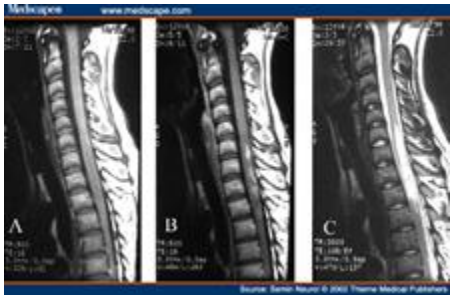
Wolters Kluwer

Aquaporin 4 rich areas: NMO

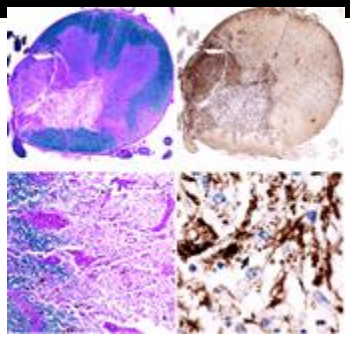


https://www.researchgate.net/figure/Routes-of-water-exit-from-the-brain-in-brain-edema-In-both-cytotoxic-and-vasogenic-types_fig1_6458778

Longitudinally extensive TM (> 3 vertebral segments) vs. rare in MS



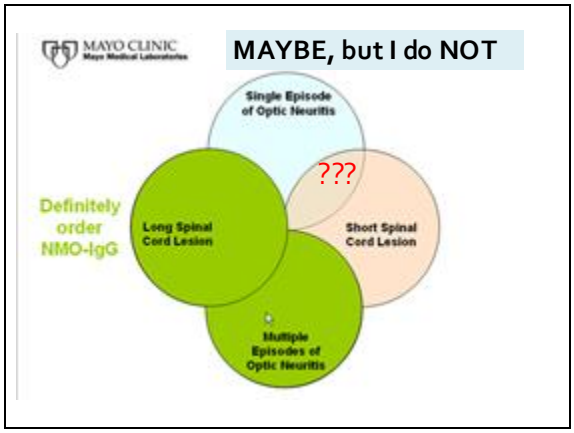
Demyelination (upper left), marked macrophage infiltration (upper right), sharply delineated plaque borders (lower left), and active demyelination with macrophages & debris (lower right).

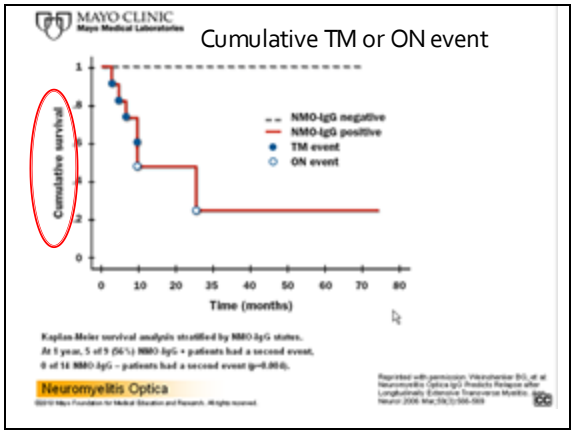


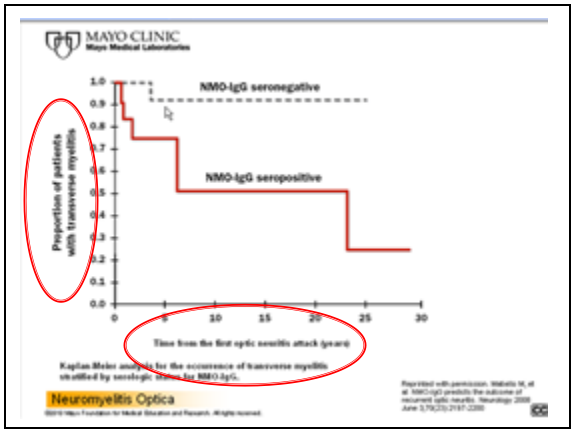
C. F. Lucchinetti *et al*, pp. 1450 - 61.

Five situations when I order NMO antibody

1. Non-recovering optic neuritis (<20/200)
2. Bilateral simultaneous or sequential ON
3. Recurrent ON & MRI brain not typical for MS
4. Atypical MS: MR negative & LP > 50 WBC CSF cells
5. Transverse myelitis (kids/adults)


MAYO CLINIC
Mayo Medical Laboratories
MAYBE, but I do NOT
Single Episode of Optic Neuritis
???
Definitely order NMO-IgG
Long Spinal Cord Lesion
Short Spinal Cord Lesion
Multiple Episodes of Optic Neuritis


MAYO CLINIC
Mayo Medical Laboratories
Cumulative TM or ON event
Cumulative survival
NMO-IgG negative
NMO-IgG positive
TM event
ON event
Time (months)
Neuromyelitis Optica


MAYO CLINIC
Mayo Medical Laboratories
NMO-IgG seronegative
NMO-IgG seropositive
Proportion of patients with transverse myelitis
Neuromyelitis Optica

Summary: You need to test for MOG and NMO because….

- It could be <u>good</u> (optic neuritis gets better)
- It could be <u>bad</u> (MOG needs IV steroids and may need immunosuppression)
- It could be <u>ugly</u> (NMO can blind you and without treatment can paralyze you)

1978: I wanted to be a doctor. 2nd choice Jedi knight… It turns out I get to do both.



Thank you for your time & attention

Thanks for your attention

Methodist