Neuro for the Rest of Us

Cecelia Koetting, OD FAAO Virginia Eye Consultants Norfolk, VA

- Neuro vs glaucoma and long term care
- Neuro and DED

Disclosures

Alcon consultant

Neurology as an Optometrist

- Signs and Symptoms noted on routine exams
- First place some patients go
- Aid in diagnosis and treatment
 - Knowing when to order MRI, MRV, MRA
 - Lumbar puncture/Spinal Tap
 - Temporal artery biopsy
 - Prescribing treatment
 - Oral steroids
 - IV steroids

Neuro can be confusing, so lets break it down into a few categories

- Cranial Nerve Palsies
- Optic Nerve Changes

Cranial Nerve Palsies

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Cranial Nerve 3 Palsy

- Eye is turned "down and out"
 - Abducens and superior oblique still function
- Eyelid is shut
 - Levator palperae the lid retractor is paralyzed
- Possible pupil involvement

Pupil sparing (normal reaction)

 Non-Pupil sparing (affected pupil is not reactive to light shone in either eye)





http://www.nature.com/eye/journal/v18/n3/fig_tab/6700625f1.html



CN 3 Palsy Causes

- Pupil involvement implies likely tumor/aneurysm (most concerning)
 - Something pressing on the nerve from outside
- Non-Pupil involvement implies likely diabetes/HTN/ ischemia (most common)

Something affecting the nerve from inside

...sometimes

Whats the next step?

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Diagnosis

- Testing
 - o In office
 - Versions/Ductions/Prisms
 - Pupils
 - EOM, Ductions and versions
 - Forced ductions
- Outside office
 - No pupil involvement
 - BS/blood work for DM
 - BP
 - Monitor for future pupil involvement
 - Pupil involvement
 - Immediate MRI of head and orbit and MRV with and without contrast

Versions and Ductions















Forced Ductions



Prism Measurement of Deviations



Cranial Nerve 4 Palsy

CN 4 Palsy

- CN 4 Controls superior oblique extraocular muscle
- Upward deviation of affected eye
- Cyclotorsion of the eye
- Head tilt away from lesion
- Diplopia

CN Palsy 4 Causes

- Trochlear nerve is the longest and skinniest, susceptible to injury
- Most common causes
 - o Trauma
 - Old vs New
 - Congenital
 - o Ischemic
 - o Tumor







Diagnosis

- A good, detailed history
- Blood pressure, blood sugar
 - Possible blood work (cholesterol, A1C)
- Parks 3 step
- MRI of head and orbit with and without contrast

Parks 3 Step

- Using cover test localize the muscle effected
- 1. Which eye is higher in primary gaze?
- 2. Is it worse in right or left gaze?
- 3. Is the hypertropia worse with right or left head tilt?



Treatment

- Treatment based on findings
 - Congenital and trauma related- follow up with neurologist
 - Lesion/mass refer quickly to neuro surgeon
 - High BS/BP monitor and contact primary care doctor to help
- Continue to monitor the patient

Case #1

- 63 year old AA woman with chief complaint of new onset diplopia stable over the last 2 months
 - Images are side-by-side and occasionally triple
 - Resolves when right eye is covered
 - Increased headaches
 - Neurologist seen 2 months prior with normal MRI per patient

How we feel inside... DID SHE SAY DIPLOPIAP mgflip.com

History

- No prior ocular history of surgeries or strabismus
- Medical history includes lung cancer in 2005 with relapse in 2014
 - Former smoker, quit 12 years prior
- Hypertension and hyperlipidemia controlled with medication
- NOT diabetic or borderline DM

Exam Findings

- BCVA 20/20 OD and OS
- Red Cap and color vision normal
- No APD
- Version and duction test reveals a complete loss of abduction OD and full range of motion OS
 Primary gaze 40PD esotropia OD
- SLE and DFE show no other abnormalities







What do we do next?

 \bullet \bullet \bullet

In Office

- Forced duction test OD

 Negative
- Blood pressure
 0 140/80
- Dx: Cranial Nerve VI palsy

Further Testing Blood work including CBC, A1C, ESR, and CRP

o All negative

 Same day MRI and MRV of head and orbit with and without contrast


- Compared with her MRI two months prior, a sizable (2.1x1.6x2.4cm) perisphenoid lesion abutting the right cavernous sinus and involving right Meckel's cave was detected
- Two smaller enhancing brain nodules were found in the left parafalcine occipital lobe and in the left frontal lobe
- All were considered suspicious for lung cancer metastases.

How to order an MRI

- If it is emergent (in the case of possible Optic Neuritis or AION)
 - Refer to local ER within 24-48 hours for MRI
 - Can send with a written script for MRI of head and orbits with and without contrast
 - Include why you are ordering it
 - Sudden decrease in vision OD with pain, possible optic neuritis
 - Include a phone number to reach the doctor at and be ready for a call
 - They will likely ask for treatment suggestion if confirmed diagnosis
 - Can send with standing order for how to treat if positive diagnosis

How to order an MRI

- In a non-emergent situation (papilledema likely IIH)
 - Order an MRI of the head and orbits with and without contrast within a few weeks
 - Can be scheduled with out patient clinics or at MRI centers
 - Your front desk staff can help the patient with this.

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Imaging Requisition Form (PLEASE FAX RESULTS TO: 1-757-793-4691)	
Patient's Name:	
Patient's DOB: / / Todays Date: / /	
G45.3 Amaurosis Fugax G70.00 Myasthenia Gravis H49.21 CN6 Palsy, Right D31.60 Neoplasm of Orbit, Benign H49.22 CN6 Palsy, Left H55.00 Nystagmus G51.0 CN7 Palsy (Bell's) H46.8 Optic Neuritis H05.241 Exophthalmos, Right H47.10 Papilledema H05.242 Exophthalmos, Left D86.89 Sarcoidosis H53.461 Homonymous Defect, Right M31.6 Temporal Arteritis H53.462 Homonymous Defect, Left H53.40 Visual Field Defect Other ICD 10: Description:	l
Carotid Doppler CT scan of the orbits with contrast CT scan of the orbits without contrast MRI of the head, with and without contrast, per radiology MRI of the orbits, with and without contrast, per radiology MRI / MRV of the head with and without contrast, per radiology X-ray of the chest PA and Lateral	
Other	
Physician Signature: Date:/	
The information below is to be filled out by the front desk staff, and then scanned into the patient's chart:	
Location/Address:	
Arrival Time: Date://	
Other Instructions:	
VEC Apt. Scheduler:	

PLEASE FAX RESULTS TO: 1-757-793-4691

Treatment

- Spoke with Neurologist that day, referred to neurosurgeon
- Patient underwent five rounds of radiation and came in every four weeks for versions/duction testing and visual fields

Cranial Nerve 6 Palsy

CN 6 Palsy

- CN 6 controls the lateral rectus muscle
 - Loss of abduction
 - May have head tilt
 - Double vision



CN 6 Palsy Causes

- Congenital
- Increased intracranial pressure
 - Idiopathic intracranial hypertension
 - o Meningitis
- Head injury
- Tumor
- Ischemic (HTN, DM)





- EOM, Ductions and versions
 o Forced duction
- Good history, BP and BS
 - Possible blood work, A1C cholesterol
- MRI of head and orbit
 - With and without contrast

Treatment

- Treatment based on findings
 - Lesion/mass refer immediately to neuro surgeon
 - High BS/BP monitor closely
 - If no improvement within 1 month obtain MRI if have not
 - Work closely with PCP to treat underlying cause

Case #2

- 28 YOA AA Female
- Presenting for LASIK evaluation
- On exam it is noted that she has a slight droop to the left side of her face with asymmetry of forehead wrinkling and smile.
- Pt lid closure OS is not tight when compared with OD
- Pt states she has never noted this before or when it may have begun

• Corneal findings:

Lipiscan

Bells palsy

Bell's Palsy

- Facial palsy caused by compression or inflammation and swelling of the facial nerve
- Usually only one side of the face
 Rarely both sides
- Can occur at any age
- Rapid onset of mild weakness to total paralysis on one side of the face
 - Within hours to days
- Facial droop
- Drooling
- Pain around jaw or behind ear on affected side
- Decreased taste
- Changes in amount of tears and saliva produced

Risk Factors

- Are pregnant, especially during the third trimester, or who are in the first week after giving birth
- Have an upper respiratory infection, such as the flu or a cold
- Have diabetes

Causes of Bell's Palsy

- Often related to viral infection
 - Herpes Simplex
 - Chickenpox and shingles (herpes zoster)
 - Infectious mononucleosis (Epstein-Barr)
 - Cytomegalovirus infections
 - Respiratory illnesses (adenovirus)
 - German measles (rubella)
 - Mumps (mumps virus)
 - Flu (influenza B)
 - Hand-foot-and-mouth disease (coxsackievirus)

Causes of Bell's Palsy

- Less often
 - o Tumor
 - o Skull fracture
- Ordering an MRI or CT to help rule out these causes

Treatment

- Most people will recover with or without treatment
- Will start to improve within a few weeks with complete recovery within about 6 months
 Occasionally permanent symptoms for life
 Can reoccur
- Oral corticosteroids
 - Helps decrease swelling of facial nerve
- Antiviral drugs
 - Although studies have shown no benefit compared with placebo

Optic Nerve Changes

Optic Nerve Head Edema

Unilateral ONH Edema

- Retinal vein occlusion
- Optic neuritis
- Arteritic ischemic optic neuropathy
- Non-Artertic ischemic optic neuropathy
- Diabetic papilitis

Case #3

- 30 year old white female
- CC: unilateral sudden decrease in vision in her right eye noted yesterday morning. No improvement today. States she feels some pain when she moves her eyes.
- DVA OD 20/100, PH NI; OS 20/20
- EOM: OD FROM (+)pain on movement in all gazes,; OS FROM (-)pain/diplopia

- Red cap test: OD 20%; OS 100%
- Color Vision Ishihara: OD 5/17 plates; OS 17/17 plates





Now what?

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MRI





- Inflammation of the optic nerve that damages the optic nerve tissue
- 6.4 per 100,000 in US
- Unilateral in 70% of cases
- 3:2 female:male
- Most often 30's, range 20-60 YOA
- Triad
 - o 1. loss of vision
 - Decrease varied, over hours to days
 - o 2. dyschromatopsia
 - 3. eye pain (worse with movement)
- Optic disc swelling
- (+)APD
- Orbital MRI will show inflammation of ON

- Visual field loss will usually occur
 Unilateral cecocentral defect most common
- Red cap and color vision changes
 Red cap desaturation and color vision deficiencies in the affected eye
- Pupil APD in affected eye may occur

- Treatment
 - IV steroids followed by oral steroids
- After treatment initiated, 30-2, color vision, and OCT-G should be performed to monitor improvement
- Patient should be referred to an Neurologist for risk of ON association with MS

Optic Nerve Treatment Trial

- o ONTT
 - 448 Patients with optic neuritis seen within 8 days of symptom onset
 - Improved visual prognosis with IV steroids vs oral vs placebo
 - Primary outcome showed those treated with IV methylprednisolone followed by oral steroids led to faster visual recovery
- Patients treated with vision 20/50 or worse at presentation had the best recovery
- Increased risk in re-occurrence with use of oral steroids alone
- Decreased rate of MS development with 2 years in patients who used IV steroids
 - o 16% IV vs 30% oral or placebo



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Affiliated Services

Virginia Surgery Center

Joanne Campbell, RN, CNOR Nurse Administrator

STANDING ORDERS

Methyl prednisolone IV infusion Optic Neuritis

Patient:

Solumedrol 1000 mg (1 gram) in 250 mL NS infused IV over 1 hour. Check serum potassium level and blood pressure prior to treatment. Hold treatment and contact ordering doctor for elevated potassium or blood pressure. Recheck blood pressure midway through infusion and after infusion. Inform patient that treatment side effects can include stomach upset, mood changes, and transient blood sugar elevation. Diabetic patients should consult their primary <u>physician</u> regarding blood sugar control.

Repeat treatment for a total of 3 treatments over the course of 3 consecutive days

Signature:

Cecelia Koetting, OD FAAO (757) 622-2200 Date

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Multiple sclerosis

- Immune-mediated process directed against the CNS
 - Attacks the myelin and the nerve fibers
- Visual Field defects
 - Result of demyelination along visual pathway
- Bilateral internuclear ophthalmoplegia (INO)
 - o diplopia
- Brain stem and cerebellum lesions
 - Dysmetria (undershoot/overshoot saccades)
 - o Nystagmus
 - Cranial nerve palsies

Optic Neuritis

• 75% occurrence, initial symptom in 14-25%

MS Risk After Optic Neuritis

- High risk of developing MS (50%)
 - 15-year risk of MS was 50% overall
 - o 25% risk of MS when MRI is normal
 - 75% risk of MS when MRI shows one or more lesions
Neurolyelitis Optica (NMO)

- Previously thought of as variant of MS
- Demyelination of optic nerve and spinal cord
- Associated with aquaporin-4 (a water channel present in glial cells) antibodies.
- Testing for NMO-IgG should be considered in those patients with bilateral ON or ON coupled with longitudinally extensive transverse myelitis (LETM), recurrent ON, or brain MRIs atypical for MS
- No cure, but similar treatment to MS
- Poor prognosis, loss of muscle function, often death occurs 2/2 respiratory complications

Bilateral ONH Edema

- Papilledema
- Idiopathic intracranial hypertension
- Optic nerve pseudoedema

Case #4

- 25 year old African American female
- CC: Increase in headache frequency and intensity over the last few months.
- (+)weight gain over the last 6 months
- (-) tinnitus or birth control use
- DVA sc OD 20/20; OS 20/20





يستستا معاساته ساليها البالل لللبات التركية السنيس المستعم

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Central 30-2 Threshold Test

Fixation Monitor: Gaze	Stimulus: III, White						Pupil Diameter:	Date: 04-01-2016				
Fixation Target: Centr	Background: 31.5 ASB						Visual Acuity:	Time: 12:56 PM				
Fixation Losses: 5/21	Strategy: SITA-Standard						RX: +0.50 DS	Age: 33				
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Now what?

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- MRI of the head and orbit with and without contrast
 - MRI of head important for masses, lesions and will check the posterior portion of the brain for Chiari malformation
- May come back normal with no findings
- Indications of elevated CSF pressure
 - Posterior globe flattening
 - o Empty sella
 - Increased tortuosity of optic nerve
 - Enlarged optic nerve sheath

Neuro consult

- With a normal MRI or with findings that indicate likely IIH the next step is a neurology consult
- They will usually order a lumbar puncture to confirm the exiting pressure of CSF and if any infection present
- May order an MRV

Papilledema • •

Papilledema

- Bilateral swollen optic nerves secondary to increased intracranial pressure
- OCT-G and 30-2 HVF
- Most common VF defect
 - Enlarged blind spot
 - Peri-cecal scotomoa
 - Often no visual field defect
- Quickly accompanied by and MRI of head and orbit to rule out space occupying lesion
- Must be confirmed with a lumbar puncture to check the ICP

Idiopathic Intracranial Hypertension

IIH

- AKA Benign intracranial hypertension and pseudotumor cerebri
- Increased intracranial pressure with unknown cause

• Diagnosis of exclusion

- Signs and symptoms
 - Headaches, tinitis, tingling in fingers and toes
- Diagnosis
 - EOM, OCT-G, 30-2, color vision, red cap
 - o MRI
 - Within 1-2 weeks
 - Lumbar puncture
 - Increased exiting pressure with normal fluid
 - Pregnant patients
 - Usually not treated

IIH

- Causes
 - o Weight
 - Birth control
 - o PCOS
 - Minocycline, doxycycline, etc
- Long term concerns and treatment
 - Glaucoma/ONH damage
 - Monitor with OCT-G
 - Diamox (acetazolamide)
 - o Topamax
 - o Shunt
 - Optic nerve fenestration
 - Weight loss
 - Approx. 10% body weight loss has been show to reverse

Co-managing

- Monitor the patient closely along with neurology
- Patient sees neurology within a month for remaining testing, diagnosis, and treatment
 - Can't start Diamox prior to this or LP will be inaccurate
- Should see the patient back within 1-2 months of neurology for repeat OCT-G and 30-2 to monitor
- Follow patient every 3-6 months for repeat testing to aid neurologist in determining if medication is working adequately.

Pseudoedema

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Optic Nerve Head Buried Drusen

- Hyaline bodies that become calcified and are located within the optic nerve head.
- Approximately 3.4/1000 people
- As drusen become larger over time the can cause a progressive visual field defect due to the secondary thinning of the RNFL
- Confirmation gold standard is obtained with Bscan or CT
 - Can also perform a fluorescein angiography to confirm



Figure 1: Fundus photo OD, arrows pointing to area of elevation within optic nerve head.



ONH Drusen

- 24-87% of ONHD have a visual field defect
- Most common visual field defect
 - o Inferior nasal step
 - Sectoral arcuate scotoma
 - Enlarged blind spot
 - Concentric peripheral constriction



Treatment

- Monitor with visual fields and OCTG
- If vision becomes compromised can treat with topical IOP lowering medications
 - Secondary glaucoma

Case #5

- 65 year old white male
- CC: Noted a sudden decrease in vision in his left eye this morning when he woke up. Pt denies any pain, but has noted a slight throbbing in his temple.
- BC DVA OD 20/20; OS CF @5
- 3+ APD OS
- DFE: OD WNL; OS 3+ edema, hyperemia

MRI and Blood Work

- MRI of head and orbit with and without contrast within 24 hours
 - Normal no findings
- Blood work
 - CRP and ESR
 - Both elevated

Now What?

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Temporal Artery Biopsy

Arteritic Ischemic Optic Neuropathy

- Arteritic (Giant Cell Arteritis/ Temporal arteritis)
 - Vasculitis within the medium and small sized arteries around the head, pt usually over 60 YOA
 - No reports of GCA in any patient under the age of 50 YOA
 - Signs and symptoms
 - Sudden painless vision loss
 - Scalp tenderness/headache
 - Jaw claudication, especially while eating
 - Polymyalgia's of arm and shoulders
 - o Fever, night sweats, weight loss

Blood work

- Elevated Sedimentation Rate (ESR)
 - Elevates in response to acute and chronic inflammation
- C-Reactive Protein (CRP)
 - An acute phase protein that increases quickly with inflammation and decreases faster than ESR with resolution
- Do not start oral steroids until after blood work



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Laboratory Requisition Form

(PLEASE FAX RESULTS TO: 1-757-793-4691)

Patient's Name: Patient's DOB: Todays Date: Diagnosis: G45.3 Amaurosis Fugax G70.00 Myasthenia Gravis H16.203 Severe Atopic Disease H46.8 Optic Neuritis M31.6 Temporal Arteritis M05.69 Arthritis H10.45 Chronic Conjunctivitis D86.89 Sarcoidosis H20.13 Uveitis (OU) E05.00 Graves Disease H15.89 Scleritis Other: Laboratory Testing: ACE (Angiotensin-Converting Enzyme) ImmunoCAP Specific IgE Lyme Chronic: HNK1(CD57) Profile (labcorps AChR (Acetylcholine Receptor Antibody Panel -Binding, Blocking & Modulating) 505026) Lyme Disease Antibodies - (total & IgM) ANA (Antinuclear Antibody panel) with reflex to western blot on positive (Labcorps 258004) ANCA (Anti-Neutrophil Cytoplasmic Antibody) Lysozyme Bartonella Antibodies QuantiFERON Gold Brucella Antibodies RAST Zone II (Southeast) Cardiolipin Antibody, profile Rheumatoid Factor CBC/Diff (Complete Blood Count with RPR (Rapid Plasma Reagin) Differential) Sjö (LabCorp Only) (816387 Early Sjögren CH 50 (Complement total blood test) Panel, 012708 Anti-SS-A/SS-B, 808900 ANA (Hep-2), 806376 RF, IgG/M/A) CMP (comprehensive Metobolic Panel 80053) Total Serum IgE CRP (C-Reactive Protein) Toxoplasmosis IgG ESR (Erythrocyte Sedimentation Rate) TSI (Thyroid stimulating immunoglobulin) FTA-ABS (Fluorescent Treponemal Antibody Urinalysis Absorption) HLA B27 (Human Leukocyte Antigen B27) Other: Physician Signature: Date:

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Temporal Artery Biopsy

- Used to help confirm diagnosis of AION
- Skip lesions can occur decreasing the tests accuracy and specificity
 - Sometimes initiate treatment even without a positive TAB
- Do not start oral steroids until after blood work
 - May initiate treatment of oral steroids up to 72 hours prior to biopsy if highly suspect after blood work results
- These need to be managed by the patients PCP
 o Long term treatment of up to a year with oral steroids

Non-arteritic Ischemic Optic Neuropathy

- Localized ischemic event at junction of optic nerve
- May be younger in age than AION (40-60 YOA)
- Signs and symptoms
 - Sudden painless vision loss
 - 30-2 severe defect
 - VA decreased
 - Less severe than AION
 - o APD
 - Pale disc swelling
 - Flame shaped heme

NAION

- Diagnosis of exclusion
 - o Normal MRI
 - May find chronic microvascular changes on MRV
 - Normal ESR/CRP
- 40% show some improvement in vision over the next 6 months
 - Monitor with visual fields
- Optic nerve edema will resolve within 8 weeks
 Can monitor with OCTG
- Risk of contralateral eye involvement

NAION Treatment

- It has been suggested in a study by Foulds in the 1970's that the patients may benefit long term visual recovery from the use of 40-60mg of oral prednisone for 1 month.
 - 85% of patients treated with 60mg oral prednisone showed visual acuity improvement compared to those untreated

NAION Treatment

- More recent study, 2008, Hayreh and Zimmerman 696 eyes
 - Treated within 2 weeks of onset with 70mg oral prednisone tapered
 - o 69.8% of eyes treated had an improvement in visual acuity
 - Only 40.5% of eyes untreated had an improvement in visual acuity

Levodopa

Graefes Arch Clin Exp Ophthalmol. 2016 Apr;254(4):757-64. doi: 10.1007/s00417-015-3191-z. Epub 2015 Oct 20.

Levodopa as a possible treatment of visual loss in nonarteritic anterior ischemic optic neuropathy.

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Author information

Abstract

PURPOSE: To determine the clinical effectiveness and potential neuroprotection of levodopa in improving visual acuity, visual field, and retinal nerve fiber layer (RNFL) thickness in eyes affected by NAION.

METHOD: Retrospective cohort study involving 59 eyes of 59 participants with NAION who were evaluated within 15 days of NAION onset. Participants received 25 mg carbidopa/100 mg levodopa three times daily with meals for 12 weeks (levodopa group) or were untreated (control group). Best-corrected visual acuity converted to logMAR, mean deviation (MD) threshold sensitivity on automated perimetry, and mean RNFL thickness on optical coherence tomography (OCT) were assessed. The primary outcome was the categorization of eyes into improved visual acuity (by 0.3 logMAR difference), worsened visual acuity (by 0.3 logMAR difference), or no change in visual acuity. The proportions in each category were compared between the levodopa and control groups.

RESULTS: Among participants with 20/60 or worse initial visual acuity, levodopa-treated participants had significant improvement (P < 0.0001) in the mean change from initial to final logMAR visual acuity of -0.74 ± 0.56 (95 % CI, -0.98 to -0.50), while the mean change for the control group at -0.37 ± 1.09 (95 % confidence interval estimate, -1.00 to +0.26) was not significant (P = 0.23). A significant difference between groups was observed (P = 0.0086) such that 19/23 (83 %) in the levodopa group improved and none got worse, as compared with 6/14 (43 %) in the control group improving while four (29 %) worsened. The change in visual field MD and RNFL thickness on OCT showed no significant difference at P = 0.23 and P = 0.75 respectively. No levodopa-treated participant had any adverse event from the levodopa.

CONCLUSIONS: Treatment within 15 days of onset of NAION with levodopa improved central visual acuity by an average of 6 lines on Snellen acuity chart. Levodopa may promote neuroprotection of the maculopapular retinal ganglion cell fibers in NAION.

KEYWORDS: Dopamine; Levodopa; NAION; Neuroprotection; Nonarteritic anterior ischemic optic neuropathy; Optic nerve

PMID: 26483145 DOI: 10.1007/s00417-015-3191-z

Levodopa for NAION

- 59 patients within 15 days of onset NAION
 - Either untreated or given 25mg carbidopa/ 100mg levodopa PO TID
 - 19/23 in the levodopa group BCVA improved and none got worse
 - 6/14 in control group BCVA improved and 4/14 got worse
Summary

- It is important to find the underlying cause of double vision and/or swollen optic nerve and to start testing early
- Optometrists can work alongside neurology to manage these patients



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Thank you!

