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Student Board Review Study Group:
Optic Nerve & Neuro-Ophthalmic Pathways

Bobby Saenz, OD, MS, FAAO

*Please Silence All Mobile Devices.*

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Disclosure statements:
*Oyster Point: Consulting Fees*

All relevant relationships have been mitigated.

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***BOARDS REVIEW:***
***NEURO***
***THE DISKY DIVAS***

Roberto (Bobby) Saenz, O.D., M.S., F.A.A.O.

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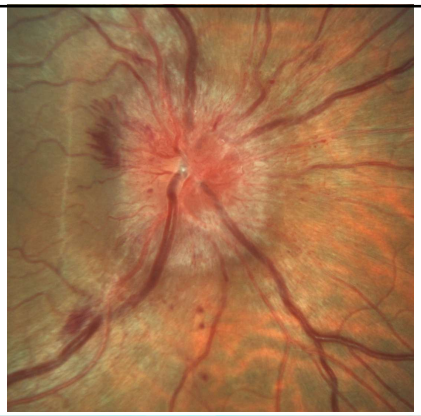

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**What can cause optic disc swelling or optic disc edema?**

The Disky Divas

…but who are the Disky Divas?

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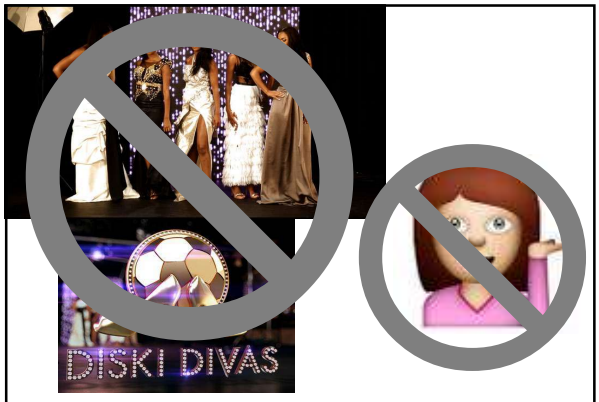


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## Meet The Divas

***O**cular Hypotony*
***C**ompressive*
***H**ereditary*
***I**nflammatory*
***I**nfiltrative*
***I**nfectious*
***I**schemic*
***T**rauma*
***T**oxic*

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## Meet The Divas

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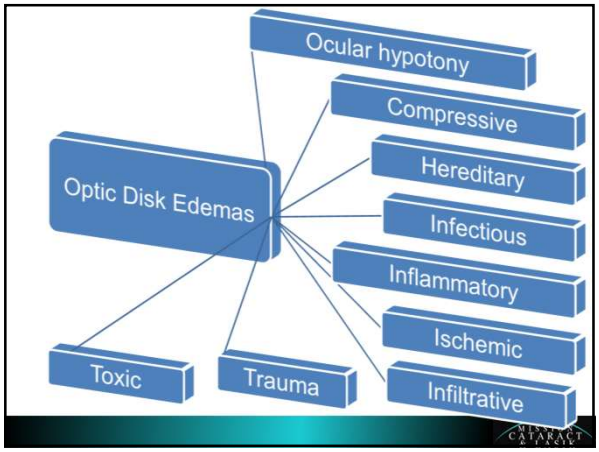


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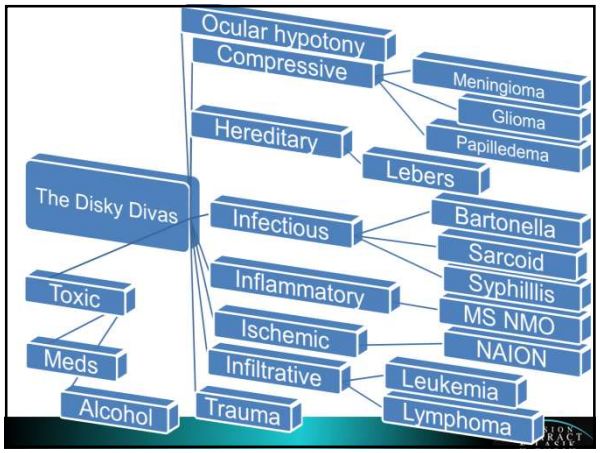


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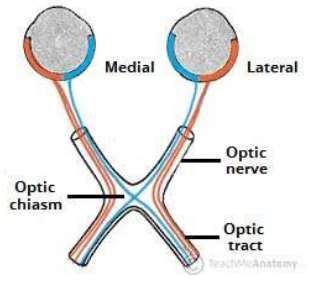
## Anatomy of the Nerve

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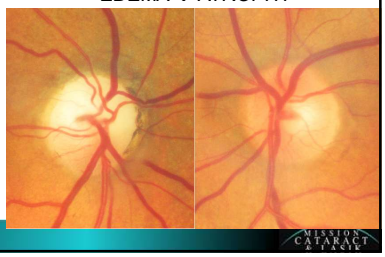
## Definitions

- Optic disc edema
- Optic neuritis
- Papilledema

EDEMA → ATROPHY



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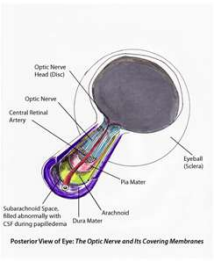
## Papilledema – bilateral optic disc edema secondary to elevated intracranial pressure



Posterior View of Eye: *The Optic Nerve and Its Covering Membranes*

*Intracranial Hypertension*

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## Important Information for finding the Divas

- Age of Patient
- **Side (Unilateral or Bilateral)**
- History
  - Symptoms (tingling of extremeties, poor bladder control)
- Medical History (meds, htn, dm)

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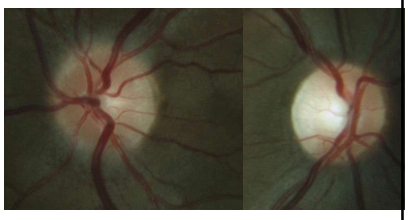
## Unilateral or Bilateral

- Optic Neuritis
  - Unilateral
- Papilledema
  - Bilateral
- NAION
  - Unilateral



***Can all three of these be bilateral?***

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## Important Clinic information

- Visual Acuity
- Color testing
- **Pupillary Testing**
- Ophthalmoscopy
- Ancillary Testing
  - **OCT**
  - **Visual Field**
  - **Neuroimaging**

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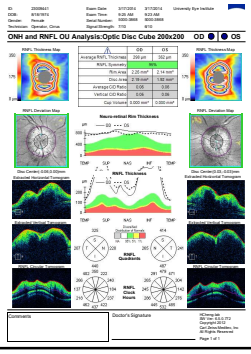
## OCT to determine disc edema

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## HVF

- Subjective measure of central and peripheral vision
- Useful in diagnosis and follow up
- Papilledema = Big blind spot
- Optic Neuritis = Diffuse and paracentral
- NAION = inferior altitudinal and inferior nasal

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## Neuroimaging

- Optic Neuritis
  - FOCAL enhancement
  - Brain: Periventricular, Infratentorial, Juxtacortical
- Papilledema
  - Empty sella
  - Flattening of the posterior aspect of the globe
  - Tortuous optic nerve
  - Transverse venous sinus stenosis
- NAION
  - What will MRI show?

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## Meet The Divas

***O**cular Hypotony*
***C**ompressive →→→→→→→→Papilledema*
***H**ereditary*
***I**nflammatory*
***I**nfiltrative*
***I**nfectious/Infiltrative*
***I**schemic*
***T**rauma*
***T**oxicity*

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## Compressive

- Glioma
- Meningioma
- Pituitary Adenoma
- **Papilledema**

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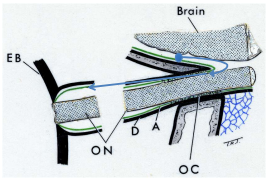
## Dr. Hayreh's study




- Papilledema occurs when increased perioptic CSF pressure

- 1st – Elevation/Blurred Disc margins
- 2nd – Vascular Changes

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## Pathophysiology – Mechanical Phenomenon

Increase in Cerebrospinal Fluid Pressure
↓
Axoplasmic Flow Stasis
↓
Swelling of Retinal Nerve Fibers
↓
Compression of Venules
↓
Venous Stasis and Fluid Leakage

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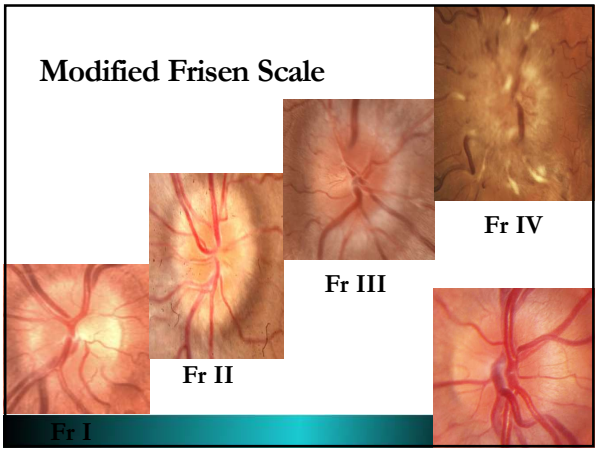
## Modified Frisen Scale

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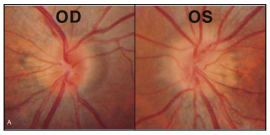
## Pseudopapilledema (PPE)

- Small, crowded discs
- Tilted/Oblique Insertion
- Drusen





Tarver – Carr et al (2006) and Karam

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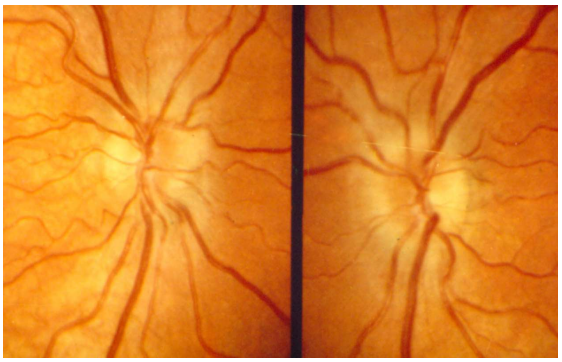

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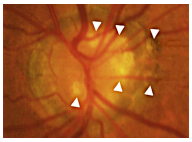
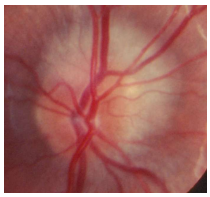
## Optic Nerve Head Drusen

- Incidence: 3.4 – 24 per 1,000 adults
- Calcific acellular bodies anterior to lamina cribrosa
- Alteration in axoplasmic flow➔ axonal degeneration ➔ deposition of calcium
- Other theories: vasculature & scleral canal





Auw - Haedrich (2000)

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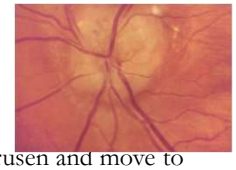
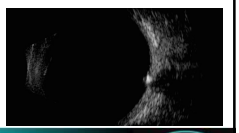
## Optic Nerve Head Drusen

- Most often bilateral
- Small or Large
- Surface or **Buried**
- Begin in younger as buried drusen and move to surface as they age.
- Symptomatic
  - TVOs or Visual Field defects





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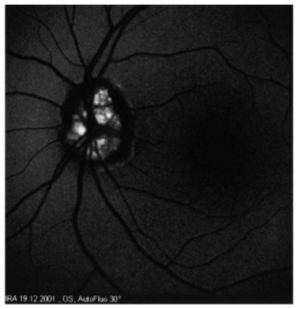
## Optic disc drusen gets mistaken for:

- Papilledema
- Glaucoma. Why?
- Any treatment?



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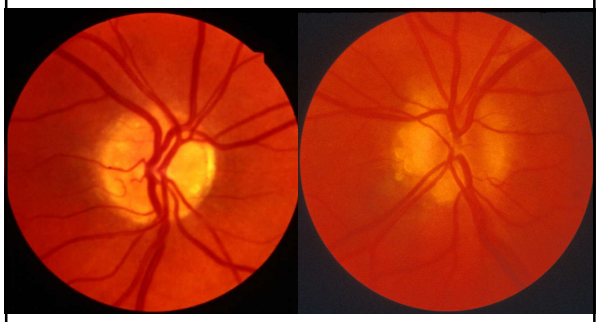

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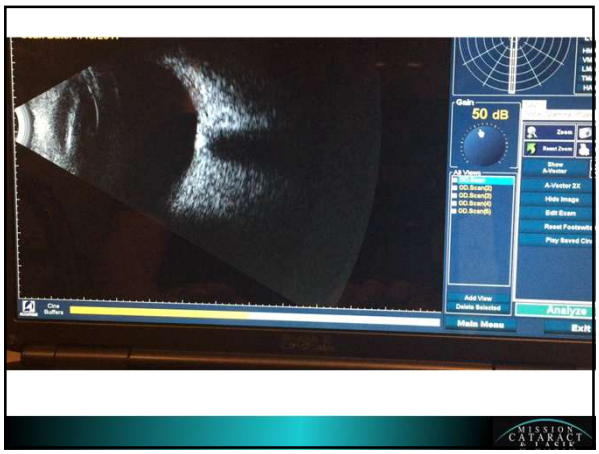


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## Differentiate PE from PPE


History - Symptoms


Fundus Examination


Diagnostic Testing

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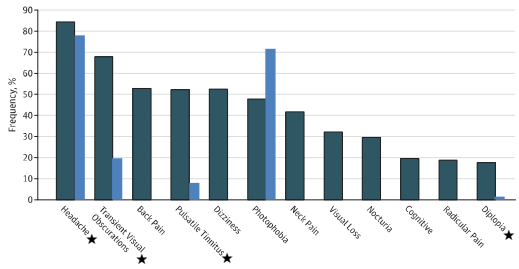
## Symptoms in PE and PPE




*Modified from Wall IIHTT (2014)*

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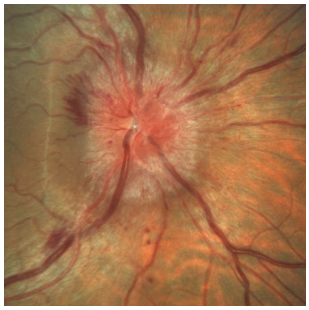
## Fundus Examination



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## Diagnostic Testing

- Orbital Ultrasound
  - A scan Optic Nerve Sheath Diameter (ONSD)
  - A scan 30° test
  - B scan Detection of Drusen
- Optical Coherence Tomography (OCT)
  - Quantitative measurements
  - Qualitative measurements

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## OCT

- Quantitative
  - **Retinal Nerve Fiber Layer Thickness (RNFLT)**
  - Peripapillary Elevation
  - Total Retinal Thickness
- Qualitative
  - Angulation of Bruch's membrane
  - Internal contour irregularities
  - Presence of hyper/hypo reflective mass

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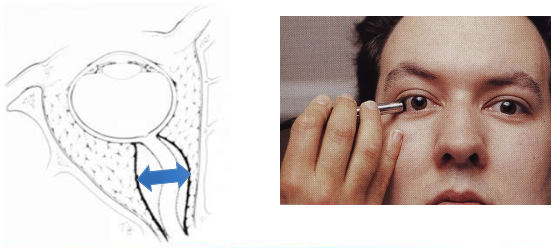
## Orbital Ultrasound

1) A scan for **ONSD**



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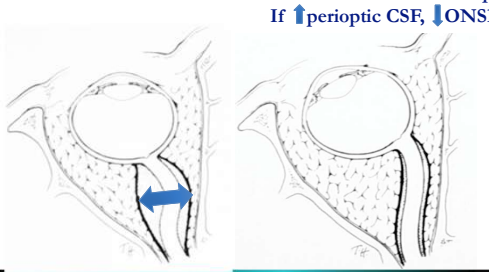
## Orbital Ultrasound

1) A scan for **ONSD**
2) A scan for **30° test**

**ONSD measured 30° temporally**
**If ⬆perioptic CSF, ⬇ONSD**



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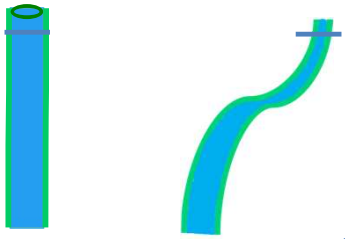
⬆ICP ⬆perioptic CSF



Primary Gaze Temporal Gaze: ⬇ ONSD

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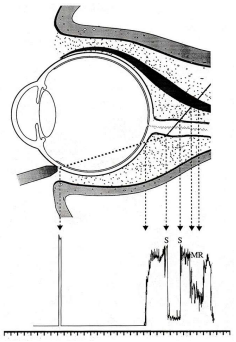
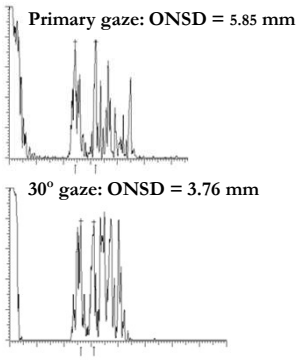


**Primary gaze: ONSD = 5.85 mm**

**30° gaze: ONSD = 3.76 mm**



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Optom Vis Sci. 2017 Dec;94(12):1081-1089. doi: 10.1097/OPX.0000000000001148.

**Use of A-scan Ultrasound and Optical Coherence Tomography to Differentiate Papilledema From Pseudopapilledema.**

Saenz R[1], Cheng H, Prager TC[2], Frishman LJ[1], Tang RA[1].

Author information

**Abstract**

**SIGNIFICANCE:** Differentiating papilledema from pseudopapilledema reflecting tilted/crowded optic discs or disc drusen is critical but can be challenging. Our study suggests that spectral-domain optical coherence tomography (OCT) peripapillary retinal nerve fiber layer thickness and retrobulbar optic nerve sheath diameter (ONSD) measured by A-scan ultrasound provide useful information when differentiating the two conditions.

**PURPOSE:** To evaluate the use of A-scan ultrasound and spectral-domain OCT retinal nerve fiber layer thickness (RNFLT) in differentiating papilledema associated with idiopathic intracranial hypertension from pseudopapilledema.

**METHODS:** Retrospective cross-sectional analysis included 23 papilledema and 28 pseudopapilledema patients. Ultrasound-measured ONSD at primary gaze, percent change in ONSD at lateral gaze (30° test), and peripapillary RNFLT were analyzed. Receiver operating characteristic curves were constructed using one eye from each subject.

**RESULTS:** Compared with pseudopapilledema, papilledema eyes showed larger mean ONSD (5.4 ± 0.6 vs. 4.0 ± 0.3 mm, P < .0001), greater change of ONSD at lateral gaze (22.4 ± 8.4% vs. 2.8 ± 4.8%, P < .0001), and thicker retinal nerve fiber layer (219.1 ± 104.6 vs. 102.4 ± 20.1 μm, P < .0001). Optic nerve sheath diameter and 30° test had the greatest area under the receiver operating characteristic curve, 0.98 and 0.97, respectively; followed by inferior quadrant (0.90) and average RNFLT (0.87). All papilledema eyes with Frisén scale greater than grade II were accurately diagnosed by ONSD, 30° test, or OCT. In mild papilledema (Frisén scale grades I and II, n = 15), area under the receiver operating characteristic curve remained high for ONSD (0.95) and 30° test (0.93) but decreased to 0.61 to 0.71 for RNFLT. At 95% specificity, sensitivities for ONSD, 30° test, and RNFLT were 91.3%, 91.3%, and 56.5%, respectively, for the entire papilledema group and 80.0%, 86.7%, and 13.3% for the mild papilledema subgroup.

**CONCLUSIONS:** Retinal nerve fiber layer thickness can potentially be used to detect moderate to severe papilledema. A-scan may further assist differentiation of mild papilledema from pseudopapilledema.

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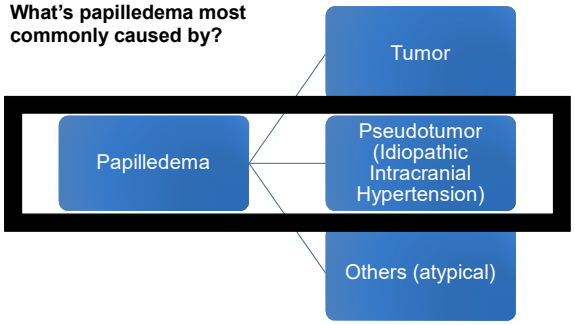
**What's papilledema most commonly caused by?**

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## OPTOMETRISTS WILL BE RELIED UPON TO MANAGE IIH. WHY?

-WE CAN ORDER MRI AND LP!

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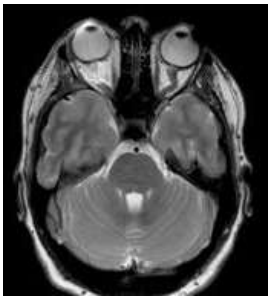
### Idiopathic Intracranial Hypertension aka Pseudotumor Cerebri

- 20 – 40 year old
- Obese female
- What's their VA (initially)?
- CN VI palsy



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### Papilledema Work Up

| | | |
|---|---|---|
| MRI | Brain Mass | Subarachnoid hemorrhage |
| MRV | Venous Thrombosis | Venous Stenosis |
| LP | Meningitis | **Idiopathic Intracranial Hypertension** ★ |

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*What are the "others"?*

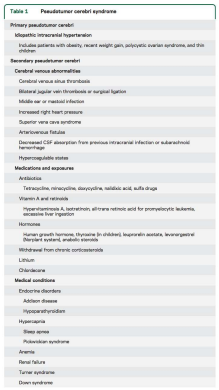

Table 1. Pseudotumor cerebri syndrome

| |
|---|
| Primary pseudotumor cerebri |
| Idiopathic intracranial hypertension |
| Includes patients with obesity, recent weight gain, polycystic ovarian syndrome, and thin children |
| Secondary pseudotumor cerebri |
| Cerebral venous abnormalities |
| Cerebral venous sinus thrombosis |
| Bilateral jugular vein thrombosis or surgical ligation |
| Middle-ear or mastoid infection |
| Increased right heart pressure |
| Superior vena cava syndrome |
| Arteriovenous fistulas |
| Decreased CSF absorption from previous intracranial infection or subarachnoid hemorrhage |
| Hypercoagulable states |
| Medications and exposures |
| Antibiotics |
| Tetracycline, minocycline, doxycycline, nalidixic acid, sulfa drugs |
| Vitamin A and retinoids |
| Hypervitaminosis A, tetratinoin, all-trans retinoic acid for promyelocytic leukemia, excessive liver ingestion |
| Hormones |
| Human growth hormone, thyroxine (in children), leuprorelin acetate, levonorgestrel (Norplant system), anabolic steroids |
| Withdrawal from chronic corticosteroids |
| Lithium |
| Chlordecone |
| Medical conditions |
| Endocrine disorders |
| Addison disease |
| Hypoparathyroidism |
| Hypercapnia |
| Sleep apnea |
| Pickwickian syndrome |
| Anemia |
| Renal failure |
| Turner syndrome |
| Down syndrome |

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Case 1

**18 HF – obese – (-) HA, TVO, tinnitus, diplopia**

MEDS: none MH: unremarkable BP: WNL

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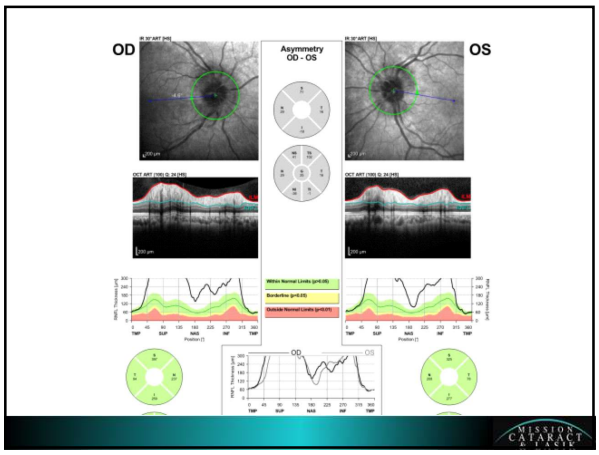


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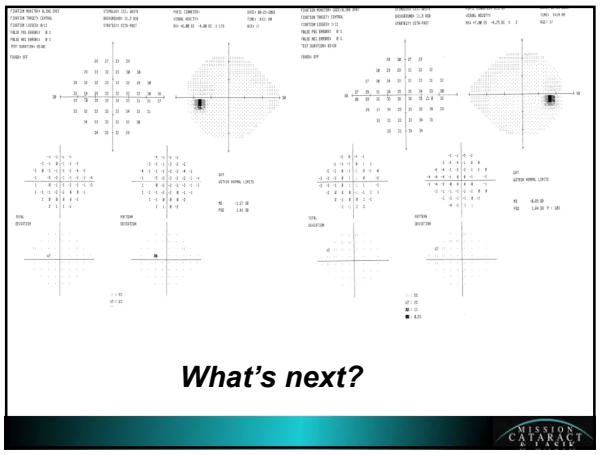


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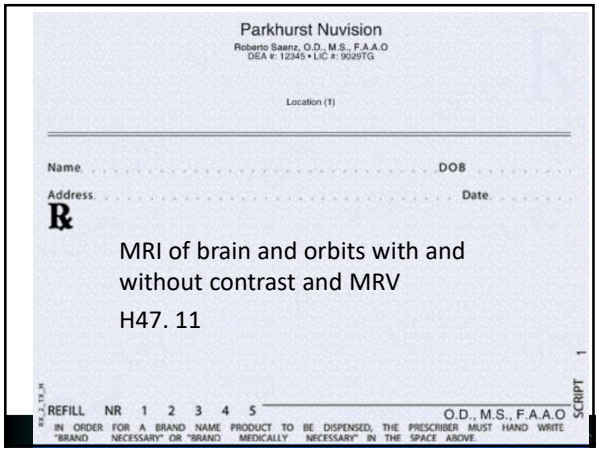


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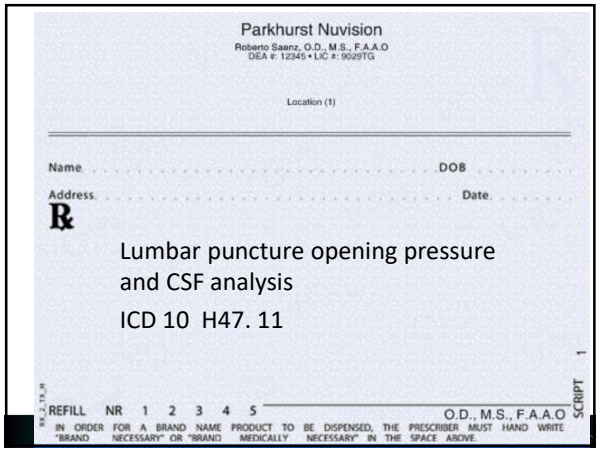


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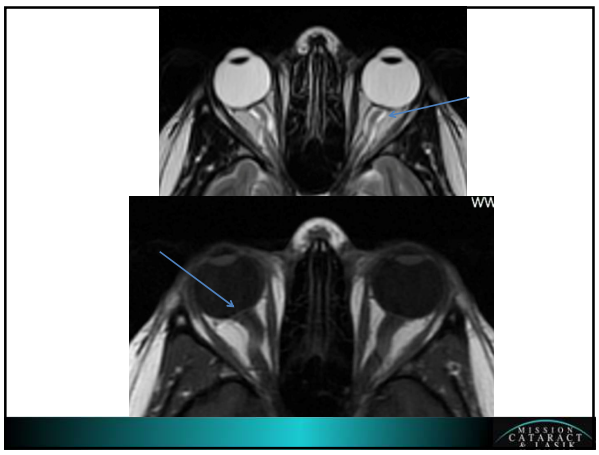

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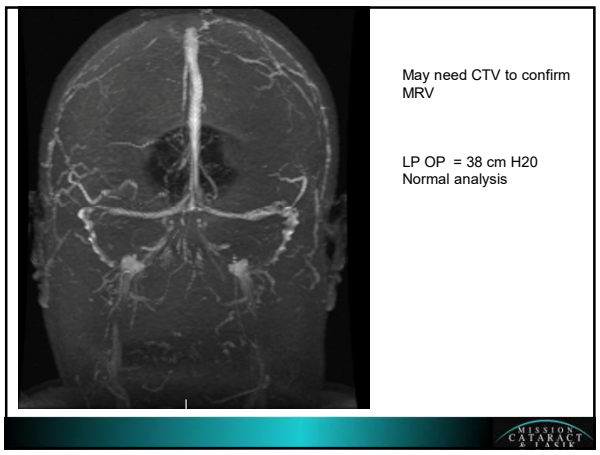


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## Treatment

- Therapeutic LP
- Oral
  - Diamox
    - + low Na diet
  - Topomax
- Surgical
  - ONH fenestrations?
  - LP or VP shunts

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## Case 1: Management

- Co-management: Call/Letter to Neurologist
- See back 3-4 weeks after LP OP
- Will see back q3 weeks until compliant/stable
  - then q3mo
  - then q6mo

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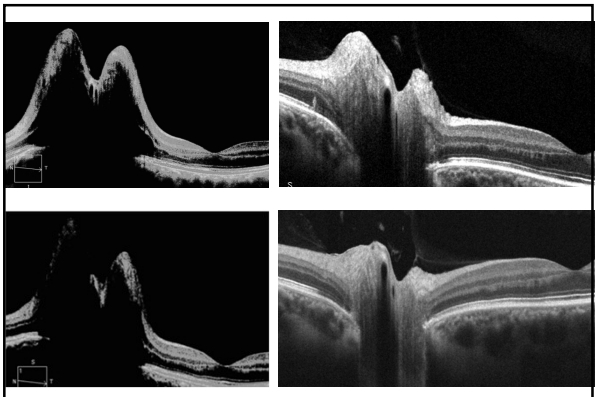

*Not published: 16/18 patients with IIH lost to follow up*

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## Case 2: 12 WF – new onset diplopia

PMH: **Crohn's**

MEDS:

- Imuran
- Iron
- Lialda
- Remicade
- Vitamin D3

**h/o oral steroids for 8 months last year**.

**(+) osteopenia**

Has **gained 50lbs** over the past 8 months.

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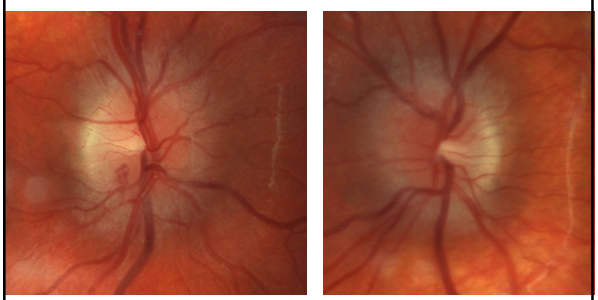
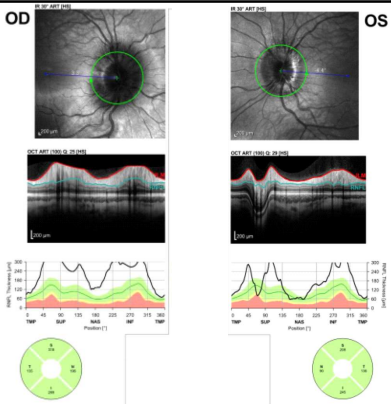
Case 2

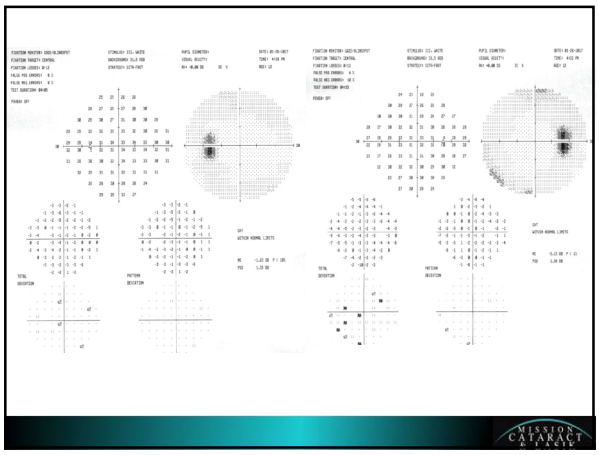
## 6th nerve palsy – then we dilate



59

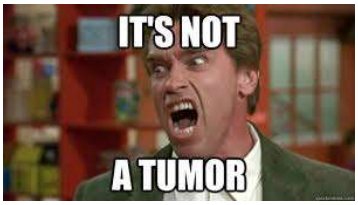



60



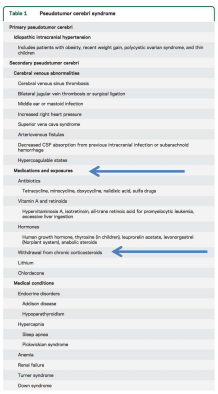
61

## What's the MRI likely going to say?




***LP OP = 42 cm $H_20$***

62



***THERE ARE OTHER THINGS THAT CAN CAUSE ELEVATED ICP!***

63

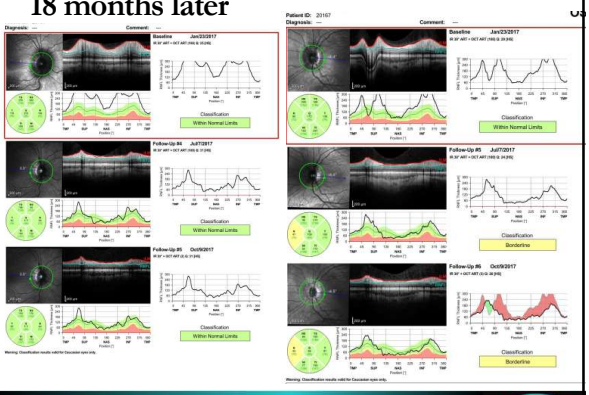
## How do you treat papilledema 2' steroid withdrawl?

- Restart steroids
- BUT she has osteopenia***
- So we started Diamox…
  - Also, how do you tell a kid to start low sodium diet?
- And that didn't happen
- Started on topomax …

18 months later….

64

## 18 months later



65

Case 4

## 26yo WF – sudden vision loss OD

- 5 days prior
- Pain on eye movements
- Medical history unremarkable
- VA OD CF OS 20/20

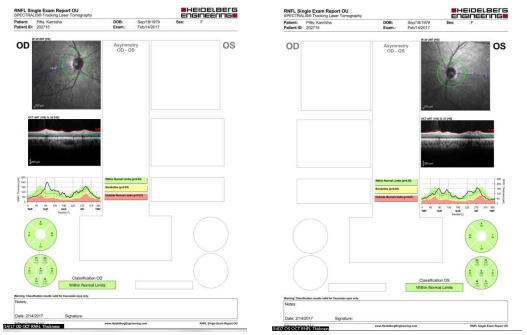
(+) APD OD

66

## You look in…..

Everything is normal….

67



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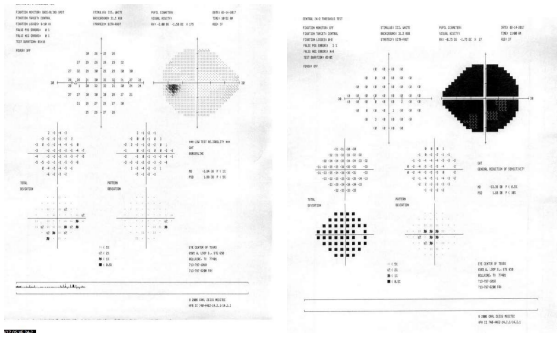


69

## What tests do we order?

70

## So now what….

- 1) Do you have to order the MRI?
- 2) Do we have to do any treatment?
- 3) Do we have to send to the ER?

71

## Assessment and Plan

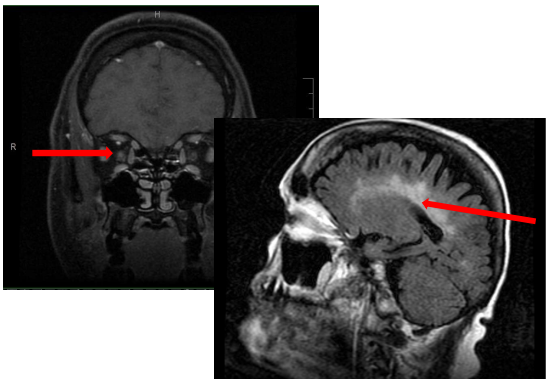
- Optic Disc Edema OD (likely optic neuritis)
- Order MRI of the brain and orbits with and without contrast with FLAIR sequencing

72

## MRI

- Brain: White matter lesions involving the periventricular white matter
- Orbits: Abnormal focal enhancement of the right optic nerve

73



74

## Meet The Divas

***O**cular Hypotony*
***C**ompressive →→→→→→→→Papilledema*
~~***H**ereditary*~~
***I**nflammatory→→→→→→→→MS/NMO*
~~***I**nfiltrative*~~
***I**nfectious/Infiltrative*
***I**schemic*
***T**rauma*
***T**oxicity*

75

## Optic Neuritis

- Inflammation of the optic nerve
- Anterior OR Retrobulbar
- Typical v Atypical
- Atypical
  - Hemes
  - Hard exudates
  - Vit cells
  - VA not improving over 30 days or NLP

76

## Typical optic neuritis

- Caucasian female
- 20-45 years of age
- No signs of inflammation in the eye
- 5 per 100,000 individuals
- Women 2x more likely to be affected

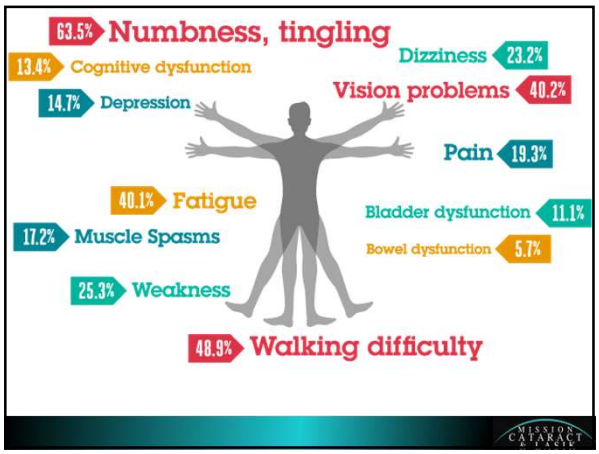
77

## Symptoms

- Unilateral, painful vision loss
- Pain on eye movements
- Altered colored vision
- Uthoff's phenomenon
  - Hot tub test
- Lhermitte's sign

In these patients we are worried about the relation between ON and ?

78




79

## Optic Neuritis Treatment Trial… what we thought

- Visual recovery was quicker with group treated with high-dose IV steroids
  - Notice only vision recovery not long term vision, which is why some docs don't treat ON with anything
- Oral steroid group had higher incidence of recurrence than IV group or placebo group
  - Which is why some doctors will NEVER use oral steroids in ON

80

## Why do we do MRI?

- Normal scan
  - 16% incidence of MS in 5 years
  - 22% incidence of MS in 10 years
  - 25% incidence of MS in 15 years
- Abnormal scan
  - 51% incidence of MS in 5 years (3 or > lesions)
  - 56% incidence of MS in 10 years (any lesions)
  - 72% incidence of MS in 15 years (any lesions)

81

## Blood Work for Atypical Optic Disc Edemas

| *Bobby's Helpful Hints* | Blood work |
|---|---|
| Sarcoid | ACE and CXR |
| Syphillis | RPR, FTA-ABS |
| TB | CXR, TB gold |
| Bartonella | Bartonella IgG and IgM |
| Toxoplasmosis | Toxoplasmosis IgG and IgM |
| Lupus | ANA & dsDNA |
| Neuromyelitis Optica | AQP4 NMO IgG antibody test |
| GCA | ESR, CRP |
| Lyme | Lyme Disease Ab with Reflex to Blot |
| HIV | HIV ½ Antigen and Antibodies, Fourth Generation, with Reflexes |

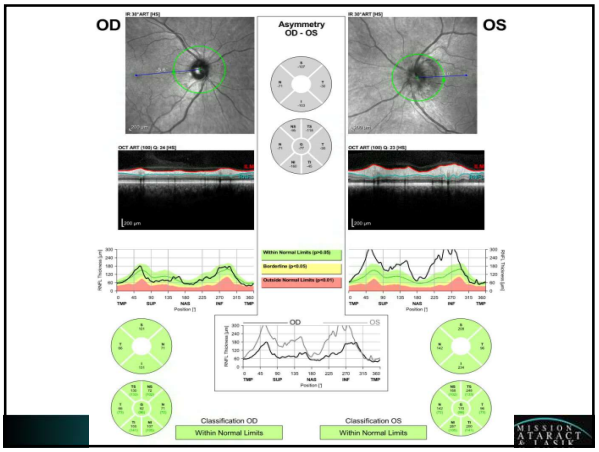
82

Case 7

## 50yo white female – sudden vision loss

- "optic disc edema eval"
- No pain on eye movements
- (+) HTN – Lisinopril
- VA 20/20 OS CF @ 1m
- APD OS

83




84

## Meet The Divas: Unilateral 50 WF

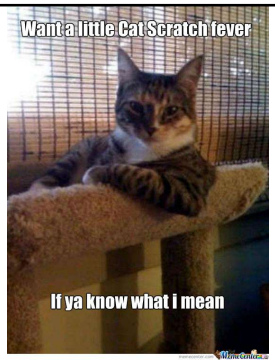
~~Ocular Hypotony~~
~~Compressive~~
~~Hereditary~~
*Inflammatory → MS/NMO [NO PAIN]*
~~Infiltrative~~
*Infectious → Syphillis/Bartonella/Toxo [MAYBE HERE?]*
*Ischemic → NAION [well controlled HTN?]*
~~Trauma~~
~~Toxic~~

85

## NAION?

- (+) HTN
- Order MRI of brain and orbits with and without contrast with flair sequencing
- Order blood work
- The last question we asked……..

86




***Do you have any CATS***

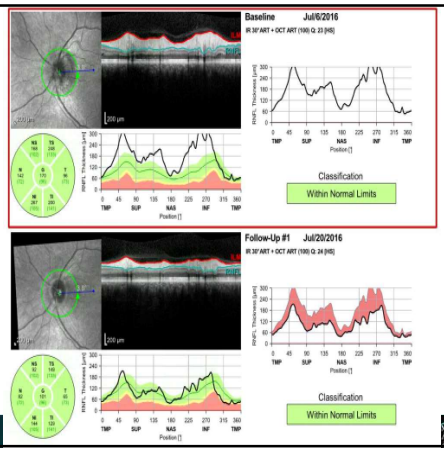
87

## Visit 2 – 2 wks later

- VA OD 20/25 OS 20/150
- Blood work ordered (+) B. Henselae
- Start Doxy 100mg BID x 28 days

DOES THE VISION COME BACK?

88



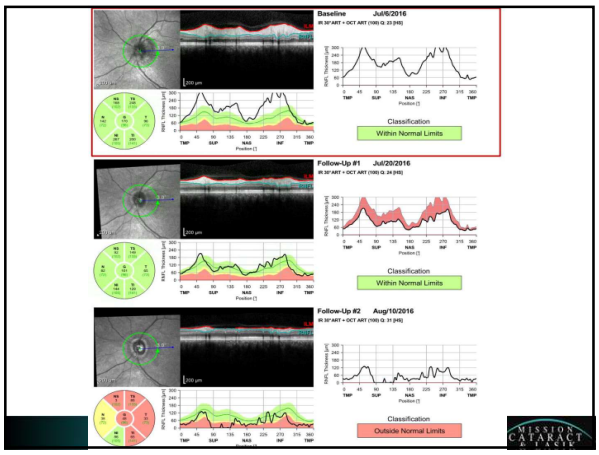

89

## Visit 3 – 5 weeks later

- VA OD 20/25 OS 20/150

NOT HAPPY VISION ISN'T IMPROVING
WANTS SECOND OPINION


90

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## When patients ask for a second opinon

**ENCOURAGE**
**AND**
**DOCUMENT**

92

## Second opinion:

- Order MRI of brain and orbits with and without contrast to rule out optic nerve enhancement or compressive ICM.
- "My concern is that despite her risk factor for NAION of HTN and age, **she does not have a small C/D ratio** in the left eye. In addition, patients with neuroretinitis usually have **spontaneous improvement of vision over time which this patient did not have**. I will have the patient follow up earlier if the MRI findings are abnormal. The different diagnosis in this patient with optic neuropathy consists of NAION, infectious/inflammatory, demyelinating disease, compressive lesion, glaucoma, etc. "

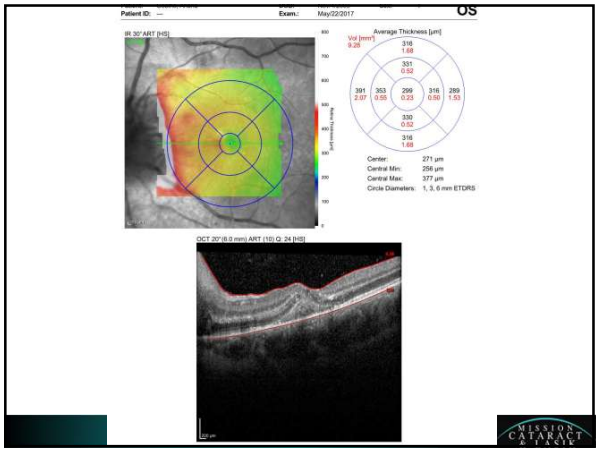
93

Case 8

## 12yo female – sudden vison loss x 2wks

- h/o fever 4 weeks ago & weakness
- (-) headaches, No eye pain
- VA OD 20/20 OS 20/200

94

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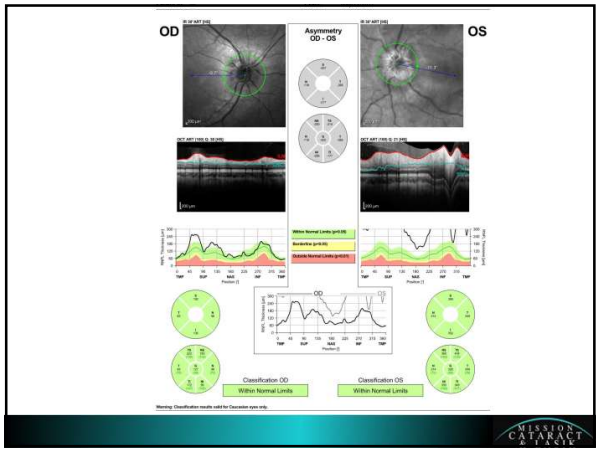


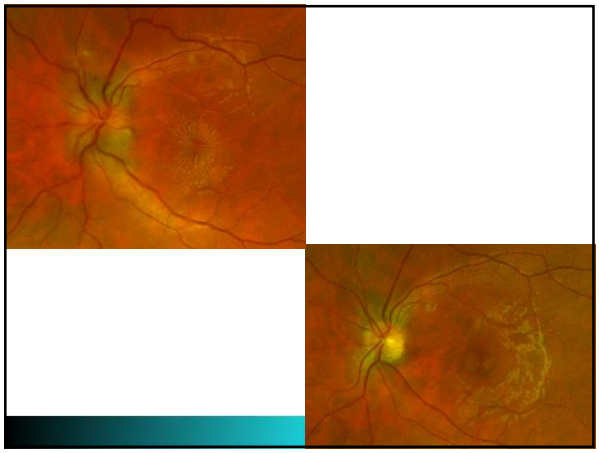

96

97

## Visit 2 – 2 weeks later

- Week 2: VA OD 20/20 OS 20/100
- Week 6: VA OD 20/20 OS 20/80
- 4mo later: VA OD 20/20 OS 20/30

98



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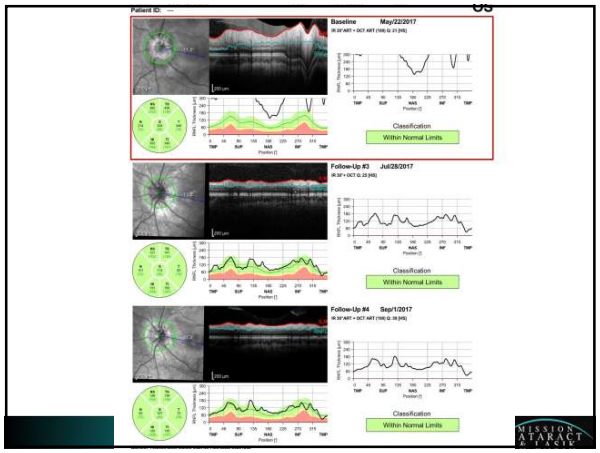



100

## Bartonella

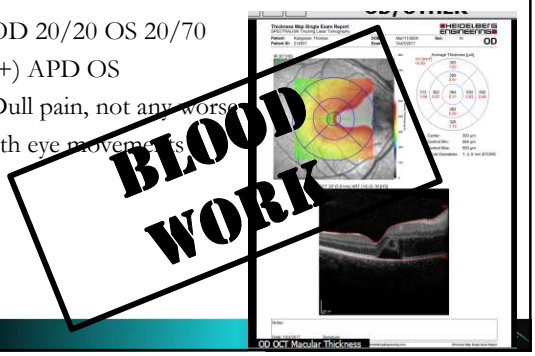
- Gram negative rod *B. Henselae*
- FLU LIKE symptoms prior to optic neuropathy
- Usually self-limiting with good visual prognosis
- Cat scratch optic neuropathy v neuroretinitis
  - Don't always need macular star
- Usually asymmetric and bilateral

101

Case 9

## 14yo HF - blurred VA centrally x 1 week

- OD 20/20 OS 20/70
- (+) APD OS
- Dull pain, not any worse with eye movements

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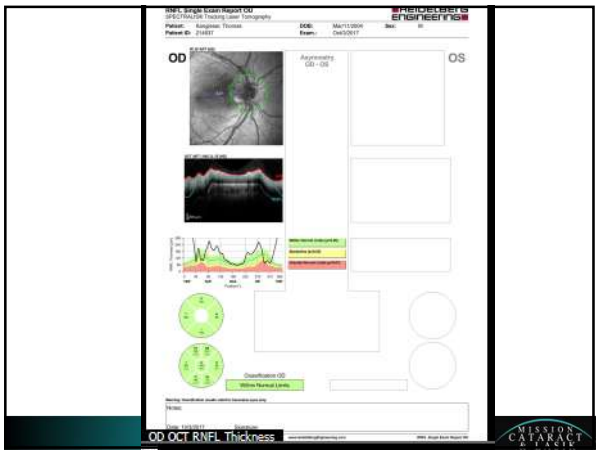


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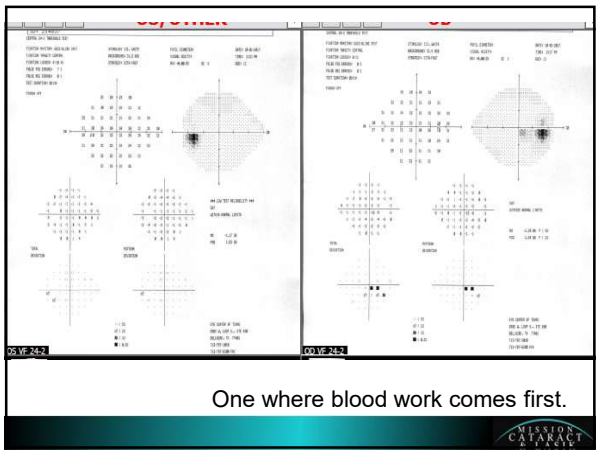


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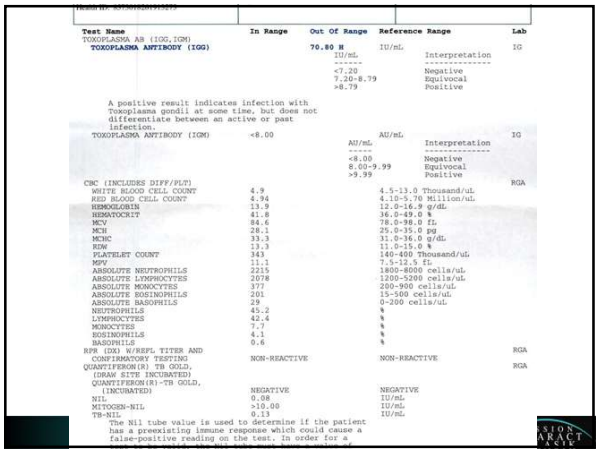


| Test Name | In Range | Out Of Range | Reference Range | Lab |
|---|---|---|---|---|
| TOXOPLASMA AB (IGG,IGM) | | | | |
| TOXOPLASMA ANTIBODY (IGG) | | 70.80 H | IU/mL | IG |

IU/mL — Interpretation
<7.20 — Negative
7.20-8.79 — Equivocal
>8.79 — Positive

A positive result indicates infection with Toxoplasma gondii at some time, but does not differentiate between an active or past infection.

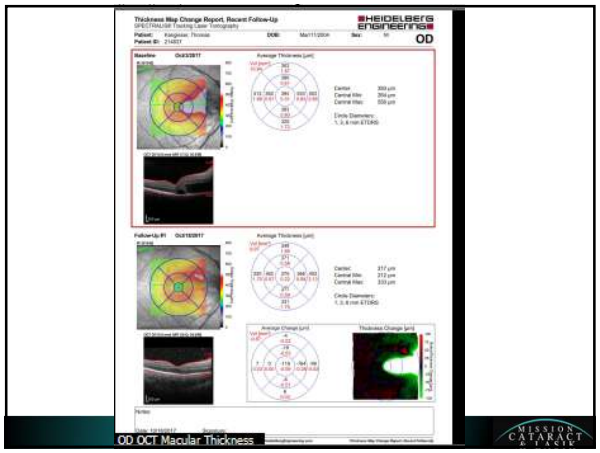
| | | | | |
|---|---|---|---|---|
| TOXOPLASMA ANTIBODY (IGM) | <8.00 | | AU/mL | IG |

AU/mL — Interpretation
<8.00 — Negative
8.00-9.99 — Equivocal
>9.99 — Positive

| | | | |
|---|---|---|---|
| CBC (INCLUDES DIFF/PLT) | | | RGA |
| WHITE BLOOD CELL COUNT | 4.9 | 4.5-13.0 | Thousand/uL |
| RED BLOOD CELL COUNT | 4.94 | 4.10-5.70 | Million/uL |
| HEMOGLOBIN | 13.9 | 12.0-16.9 | g/dL |
| HEMATOCRIT | 41.8 | 36.0-49.0 | % |
| MCV | 84.6 | 78.0-98.0 | fL |
| MCH | 28.1 | 25.0-35.0 | pg |
| MCHC | 33.3 | 31.0-36.0 | g/dL |
| RDW | 13.3 | 11.0-15.0 | % |
| PLATELET COUNT | 343 | 140-400 | Thousand/uL |
| MPV | 11.1 | 7.5-12.5 | fL |
| ABSOLUTE NEUTROPHILS | 2215 | 1800-8000 | cells/uL |
| ABSOLUTE LYMPHOCYTES | 2078 | 1200-5200 | cells/uL |
| ABSOLUTE MONOCYTES | 377 | 200-900 | cells/uL |
| ABSOLUTE EOSINOPHILS | 201 | 15-500 | cells/uL |
| ABSOLUTE BASOPHILS | 29 | 0-200 | cells/uL |
| NEUTROPHILS | 45.2 | | % |
| LYMPHOCYTES | 42.4 | | % |
| MONOCYTES | 7.7 | | % |
| EOSINOPHILS | 4.1 | | % |
| BASOPHILS | 0.6 | | % |
| RPR (DX) W/REFL TITER AND CONFIRMATORY TESTING | NON-REACTIVE | NON-REACTIVE | RGA |
| QUANTIFERON(R) TB GOLD, (DRAW SITE INCUBATED) | | | RGA |
| QUANTIFERON(R)-TB GOLD, (INCUBATED) | NEGATIVE | NEGATIVE | |
| NIL | 0.08 | | IU/mL |
| MITOGEN-NIL | >10.00 | | IU/mL |
| TB-NIL | 0.13 | | IU/mL |

The Nil tube value is used to determine if the patient has a preexisting immune response which could cause a false-positive reading on the test. In order for a

105




106

## Meet The Divas

***O**cular Hypotony*
***C**ompressive →→→→→→→→Papilledema*
***H**ereditary*
***I**nflammatory→→→→→→→→MS/NMO*
***I**nfiltrative*
***I**nfectious/Infiltrative→→→→→Syphillis/Bartonella/Toxo*
***I**schemic* **KIDS**
***T**rauma*
***T**oxicity*

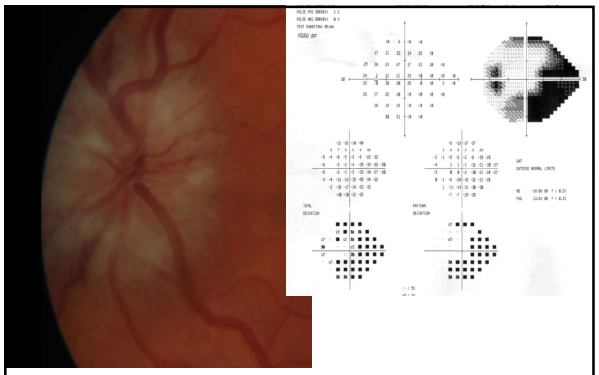
107

Case 11

**52yo male – (+) "decently" controlled HTN**

- VA OD 20/20 OS 20/20
- Film over left side of vision
- Optometrists said it's probably optic neuritis and I may have MS

108



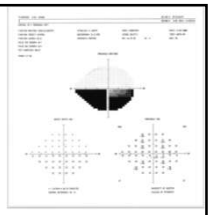
109

## NAION Treatment?

- Do we need to order MRI?

110

## NAION



- Most common disc edema >50
- Avg age 61-65
- Segmental swelling
- Tx: Optic nerve sheat decompression Aspirin, hyperbaric chambers, vasodilators, and steroids? have proved to ineffective.
- Avg VA 20/20 to NLP, 25-50% improve by 3 lines
- VF defect present at 6mo likely present

111

## Risk Factors

- HTN
- DM
- Sleep apnea
- Nightime dosing of blood pressure medications?
- ED meds?

112

## NAION

So if I have NAION can I manage this?

Yes….. BUT! Make sure you know what you are doing.

You can miss Bartonella.

Don't miss GCA.

Don't miss neurosyphillis.

113

## Meet The Divas

***O**cular Hypotony*
***C**ompressive →→→→→→→→→Papilledema*
***H**ereditary*
***I**nflammatory→→→→→→→→→MS/NMO*
***I**nfiltrative*
*Infectious/Infiltrative→→→→→→Syphillis/Bartonella*
***I**schemic→→→→→→→→→→→NAION*
*Trauma*
***T**oxicity*

*Intracranial Hypertension*

114

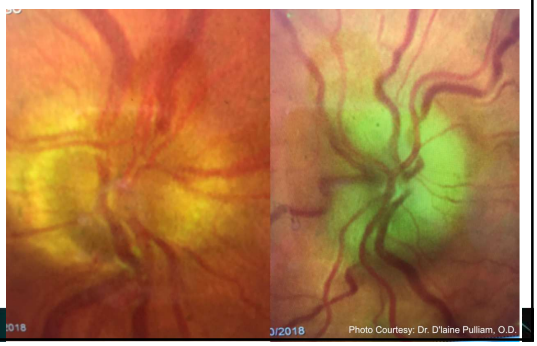
## AION (GCA)

- Avg age of onset = 70 years old
- Medium to large sized vessels
- Chalky white nerve
- Most common symptoms
  - Anorexia
  - Neck pain
  - Jaw claudication
- ESR and CRP
- Will my vision return?

115

Case 12

## 75WM – painful sudden vision loss OS

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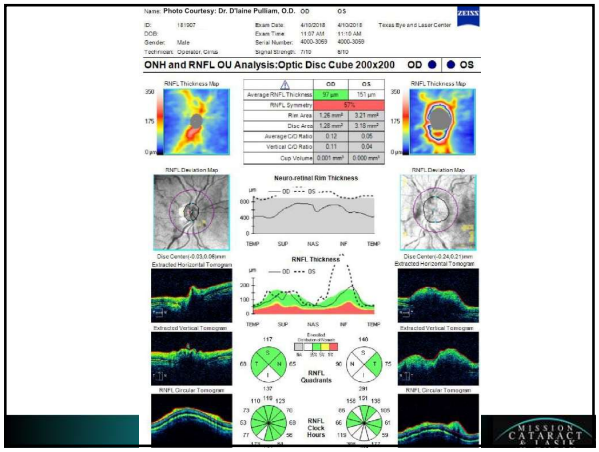


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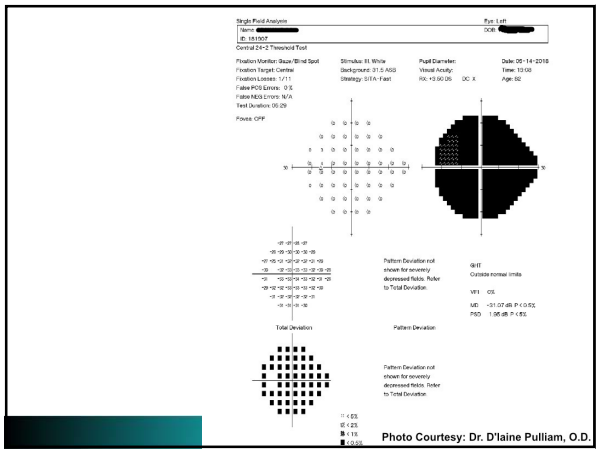


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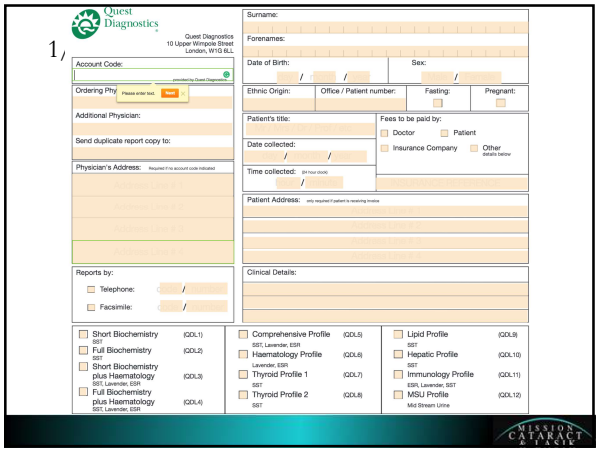


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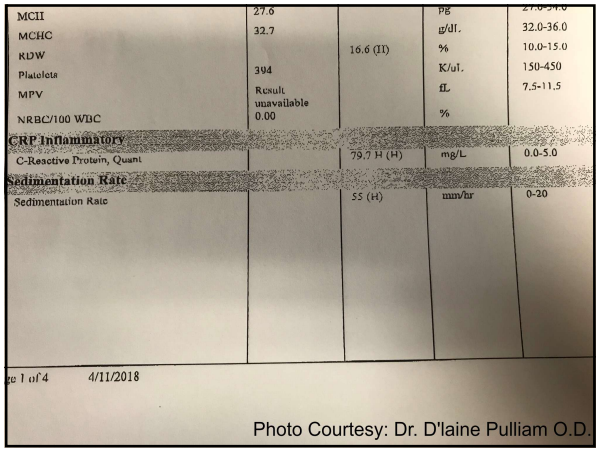


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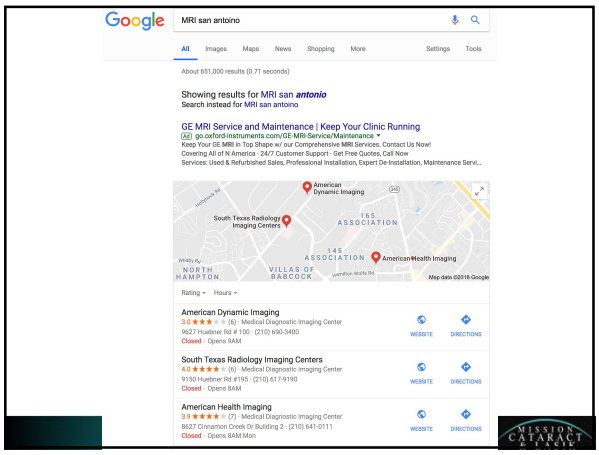


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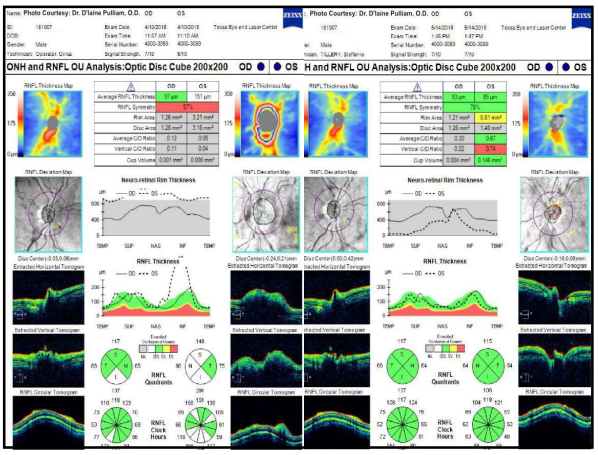


122

## Meet The Divas

***O**cular Hypotony*
***C**ompressive →→→→→→→→Papilledema*
***H**ereditary*
***I**nflammatory→→→→→→→→MS/NMO*
***I**nfiltrative*
***I**nfectious/Infiltrative→→→→→Syphillis/Bartonella*
***I**schemic→→→→→→→→→→NAION*
***T**rauma*
***T**oxicity→→→→→→→→→→Ethambutol*

123

## Ethambutol

- THE ONE DRUG YOU CAN'T MISS

J Ocul Pharmacol Ther. 1997 Oct;13(5):473-7.

**Reversibility of ethambutol optic neuropathy.**

Tsai RK[1], Lee YH.

Author information

**Abstract**

Ethambutol hydrochloride is one of the routinely used drugs as the first line of antitubercular agents. The delayed onset of ocular toxicity is usually thought to be reversible following rapid withdrawal of the drug. We collected ten consecutive patients with severe visual defects due to ethambutol toxicity, and these patients had received presumably safe ethambutol dosages. Although ethambutol was stopped immediately in all cases, only five patients (50%) experienced visual improvement after a period of 12 months to 3 years follow-up. The other five patients (50%) had permanent visual impairment without recovery. There were no predisposing or risk factors to contribute to poor visual outcome. In the group over 60 years old, only 20% (1/5) experienced visual improvement; in the group less than 60 years old, 80% (4/5) had some visual recovery, the difference between these two age groups being statistically significant. We need more patient collections to answer whether the older patients with ethambutol optic neuropathy have poor prognoses. Ethambutol optic neuropathy, in our follow-up study, is not always reversible, especially in the older population. It may cause permanent visual disability. There is no so-called "safe-dosage". We suggest reconsideration regarding the use of ethambutol as one of the first-line antitubercular drugs, especially in older patients.

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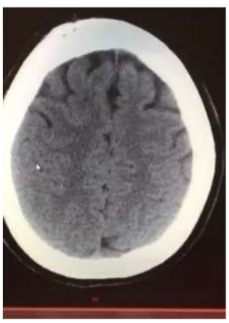
## ¿Questions?



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