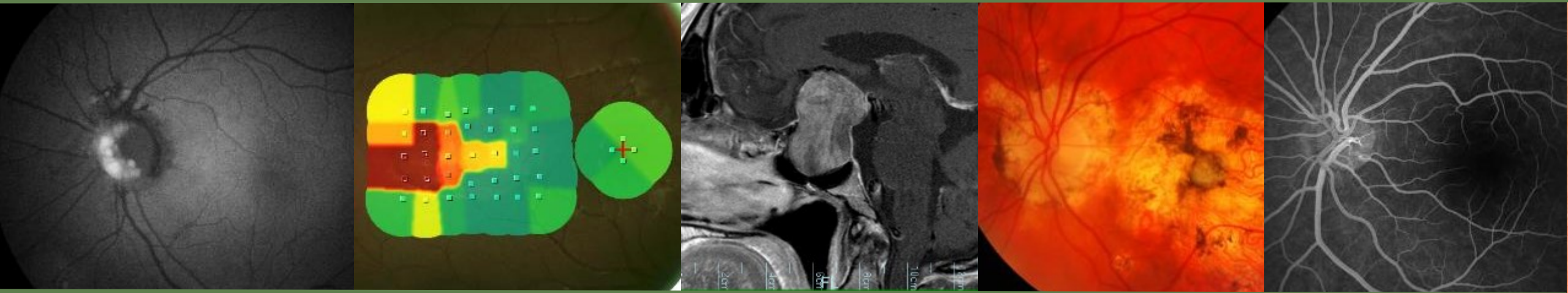


# *Grand Rounds*

## **MULTIMODAL DIAGNOSTICS**

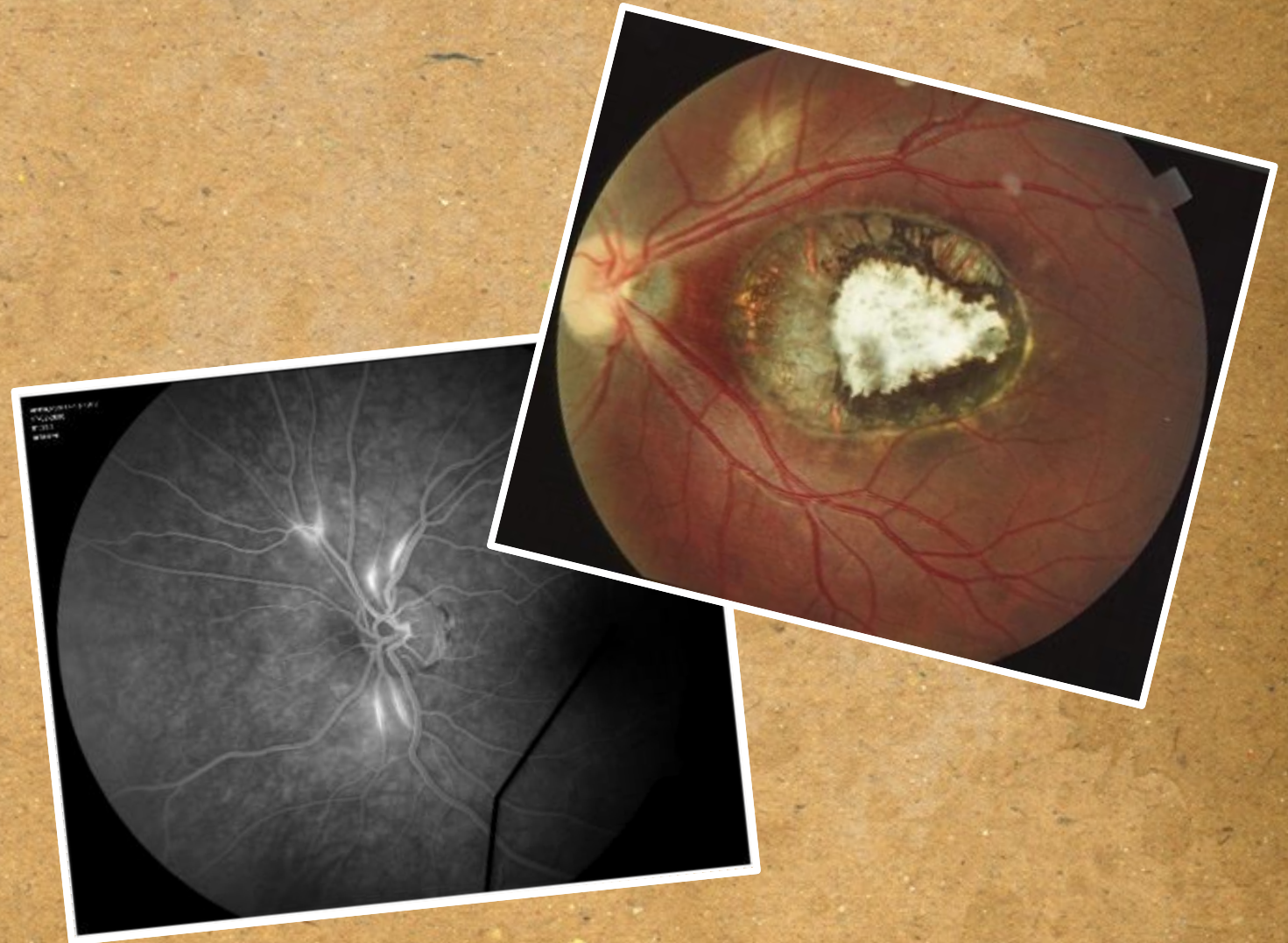


**Richard Trevino, OD, FAAO**

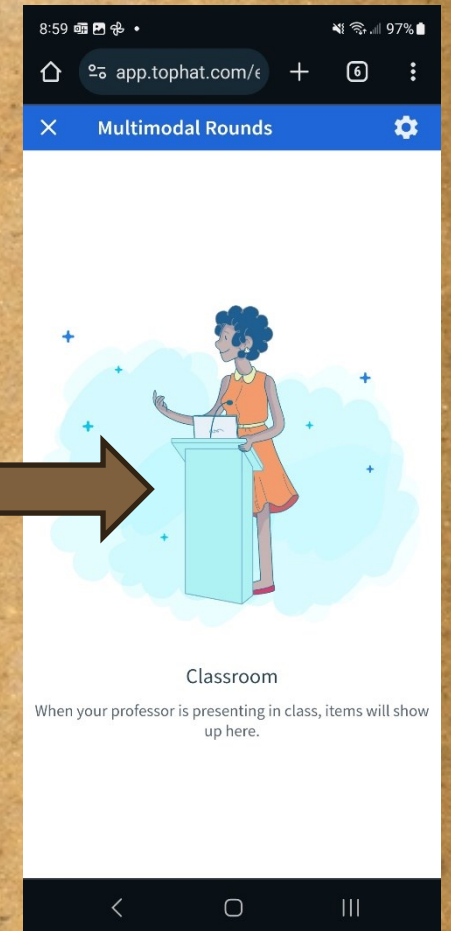
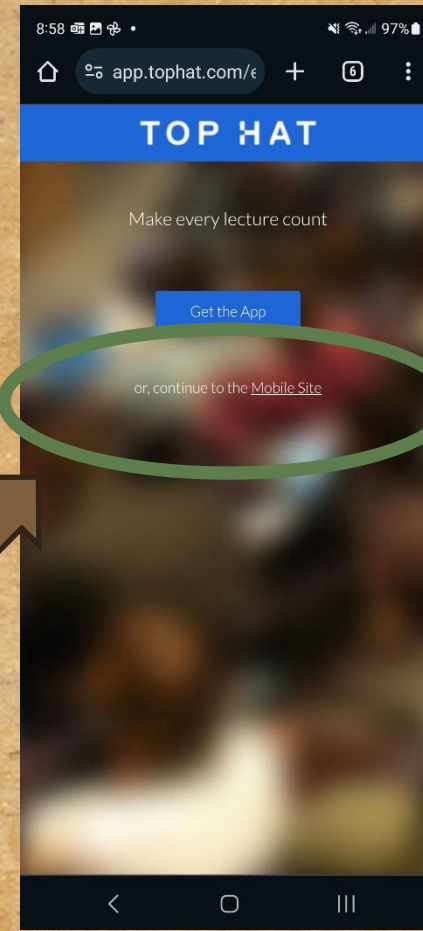
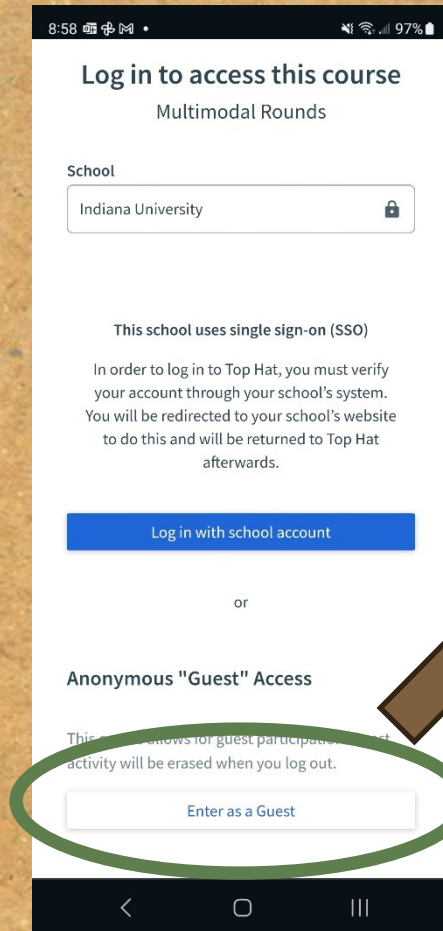
Indiana University School of Optometry

# Multimodal Grand Rounds

- **Online notes**
  - [richardtrevino.net](http://richardtrevino.net)
- **Email us**
  - [rctrevin@iu.edu](mailto:rctrevin@iu.edu)
- **Disclosures**
  - None



# Interactive Presentation



# Battle of the Superheros!



<https://app.tophat.com/e/777538>



Superman

**A**

Batman

**B**

Captain America

**C**

Wonder Woman

**D**

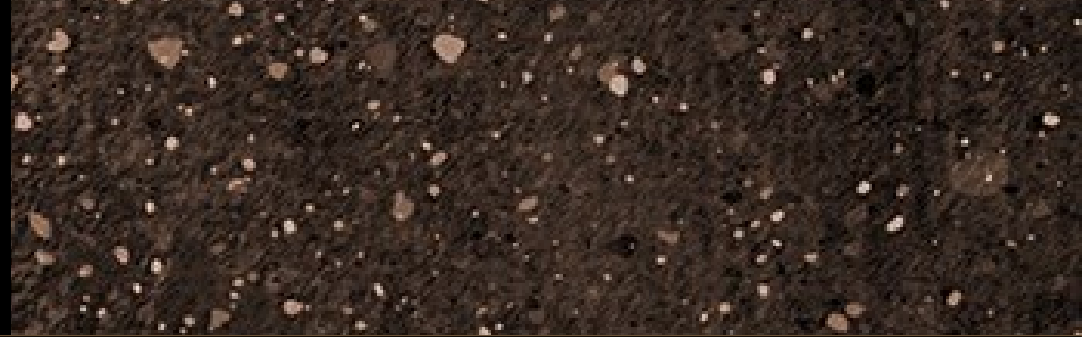
# CASE #1

*Don't sink my battleship!*

# CASE #1

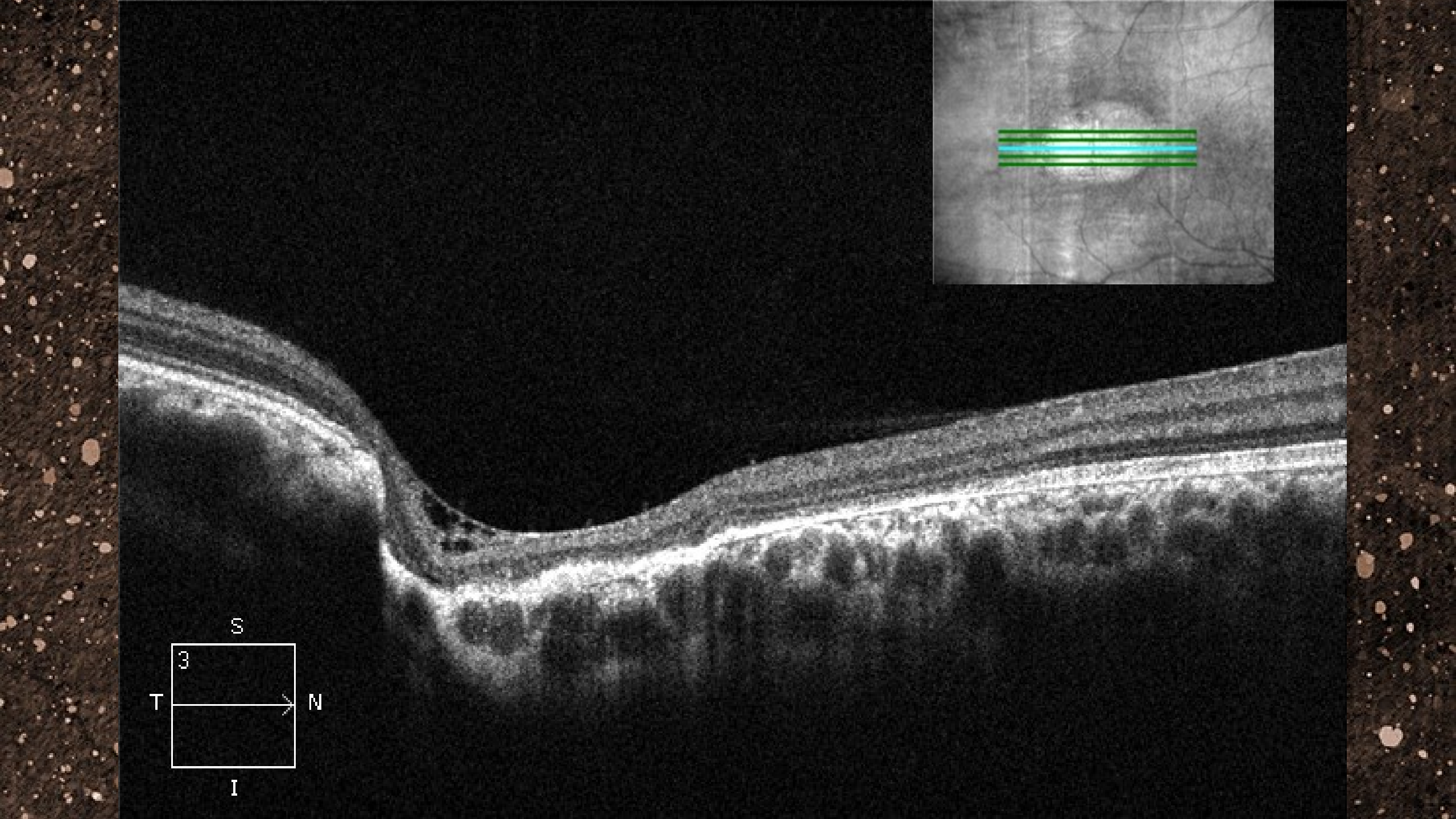
---

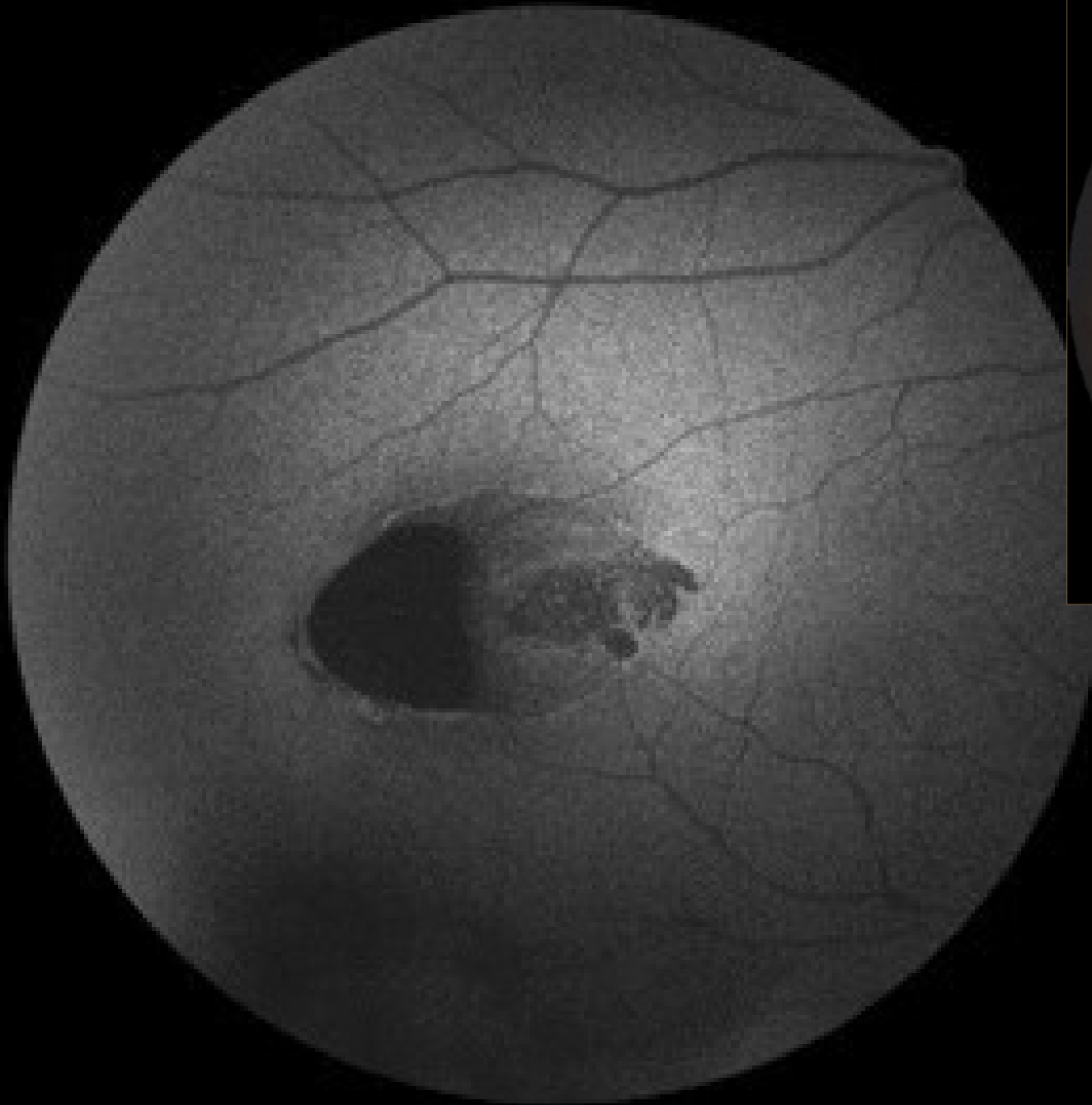
- 25yo East Indian woman presents without complaints for routine exam
- POH: Unremarkable. LEE: 1yr.
- MH: Good health. No medications
- Vision: 20/15 each eye without correction
- Entrance testing: Normal
- External exam: Normal OU
- Tonometry: 14/13 @11:00AM







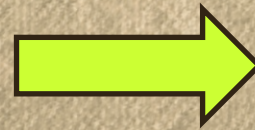
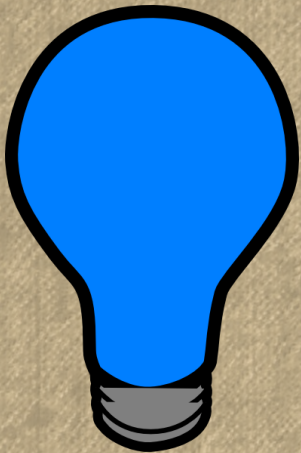


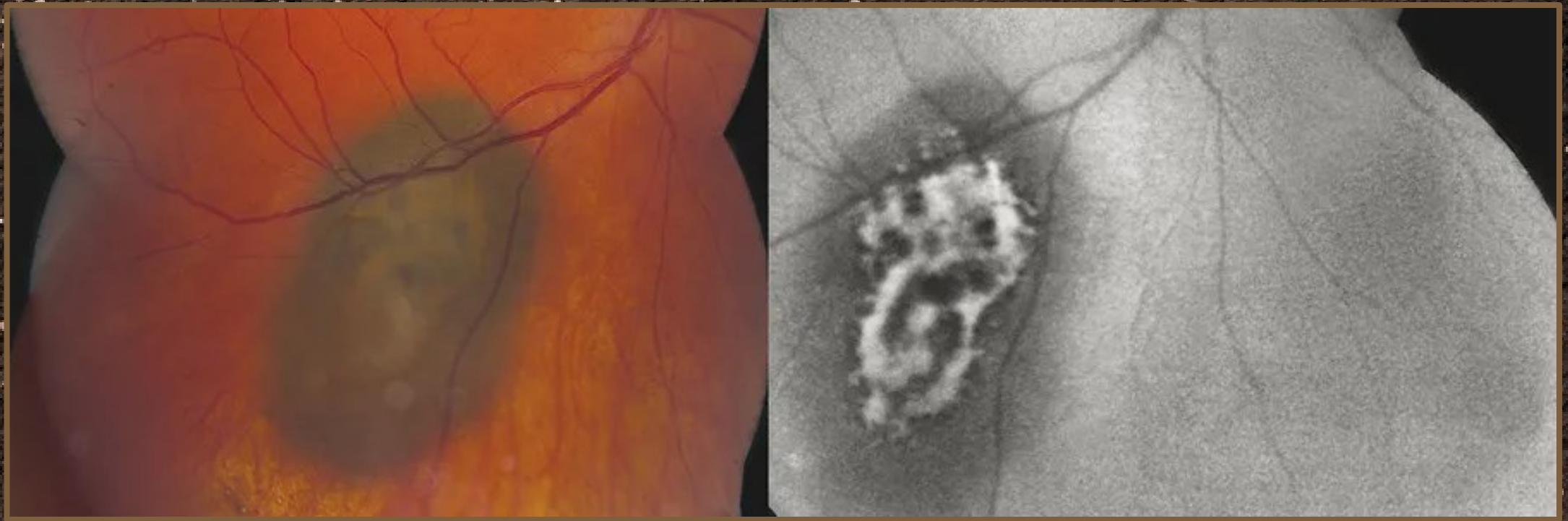


**Fundus autofluorescence (FAF)** uses blue light to stimulate lipofuscin in RPE cells to glow. Regions without viable RPE will appear dark.

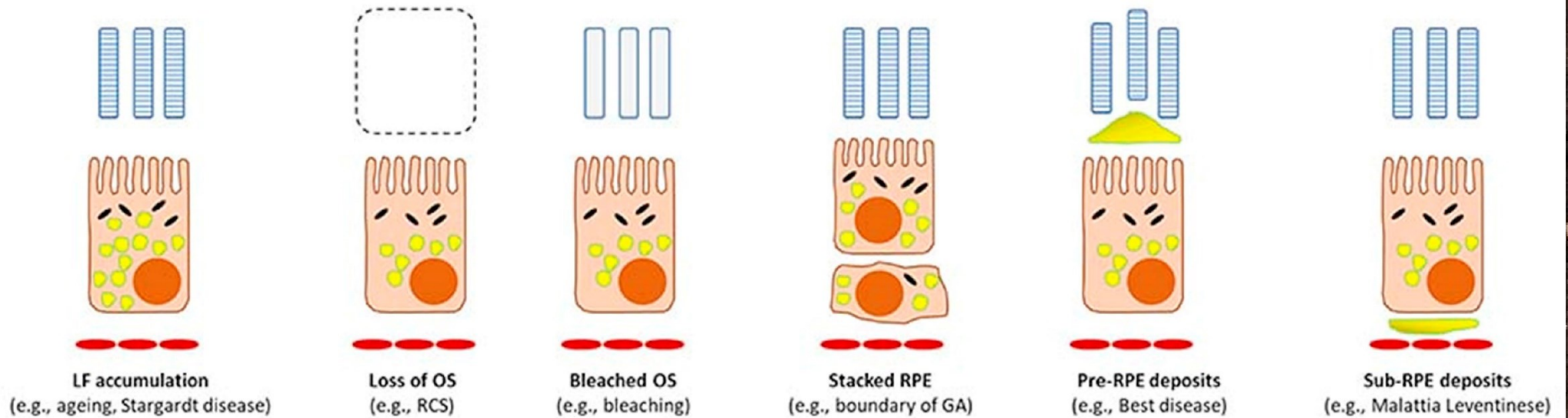
# About Fundus Autofluorescence

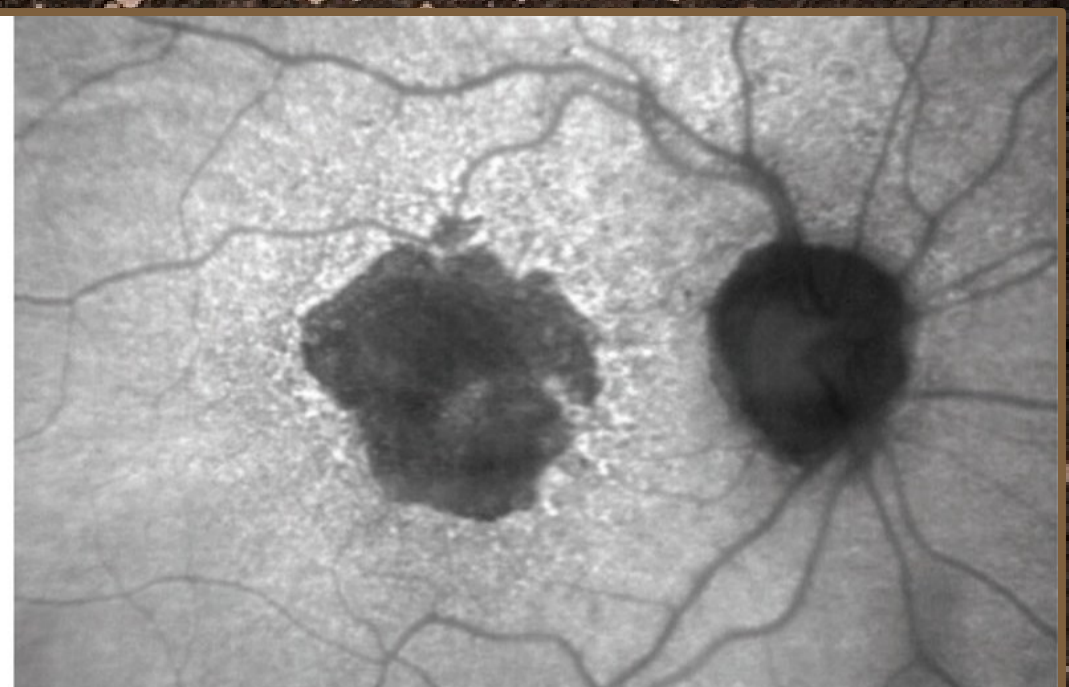
- Lipofuscin is a waste product of normal cell metabolism found throughout the body
- The RPE accumulates lipofuscin as it phagocytizes photoreceptor outer segments
- When stimulated by blue light, lipofuscin will glow





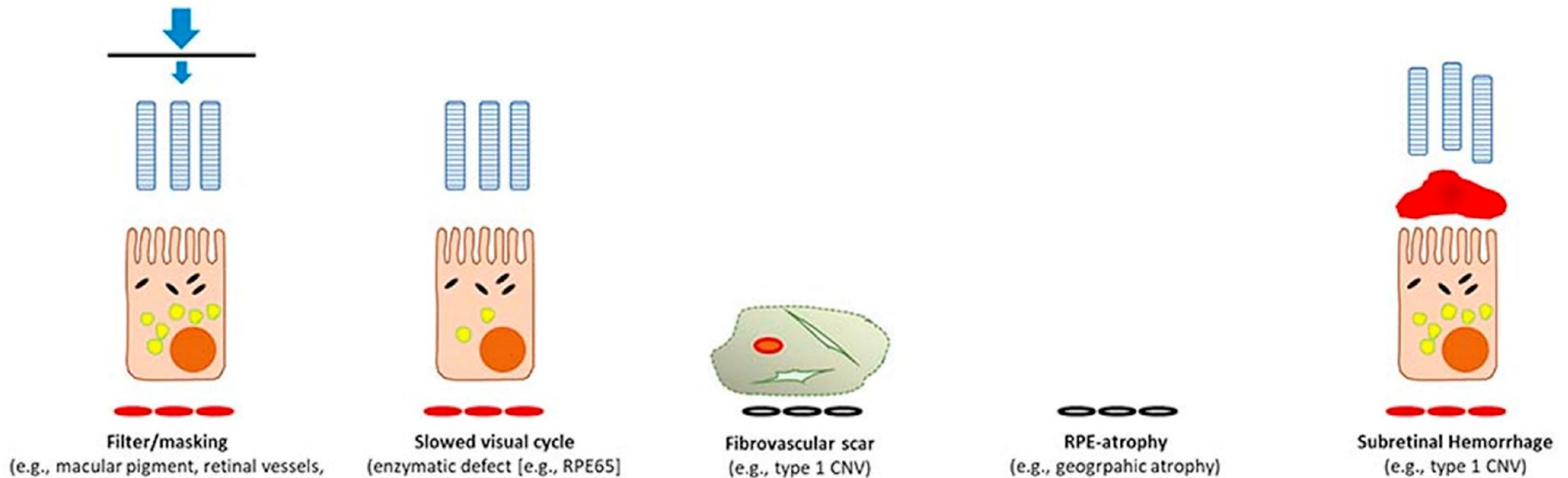
**Increased autofluorescence signal**

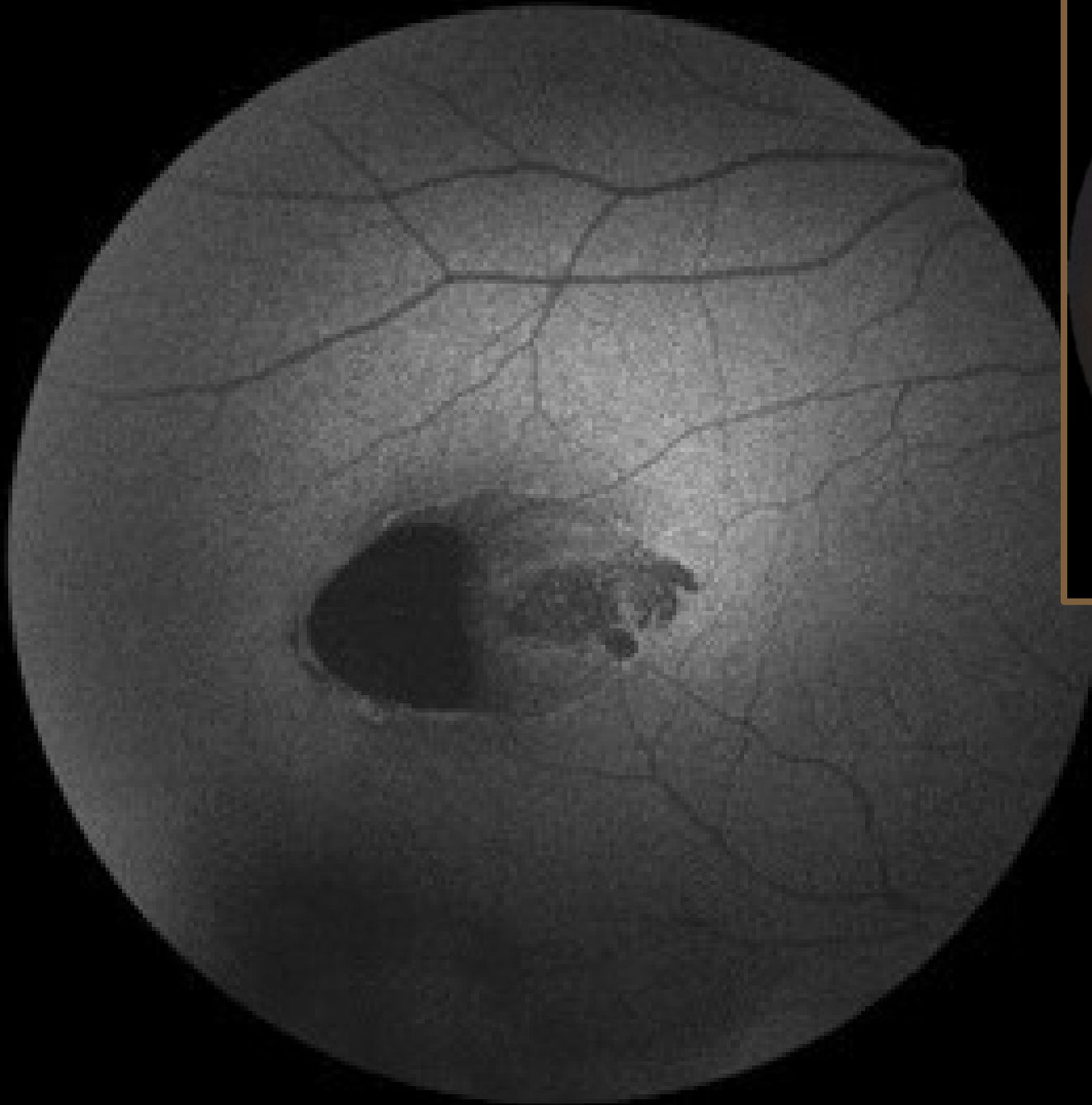




Decreased autofluorescence signal

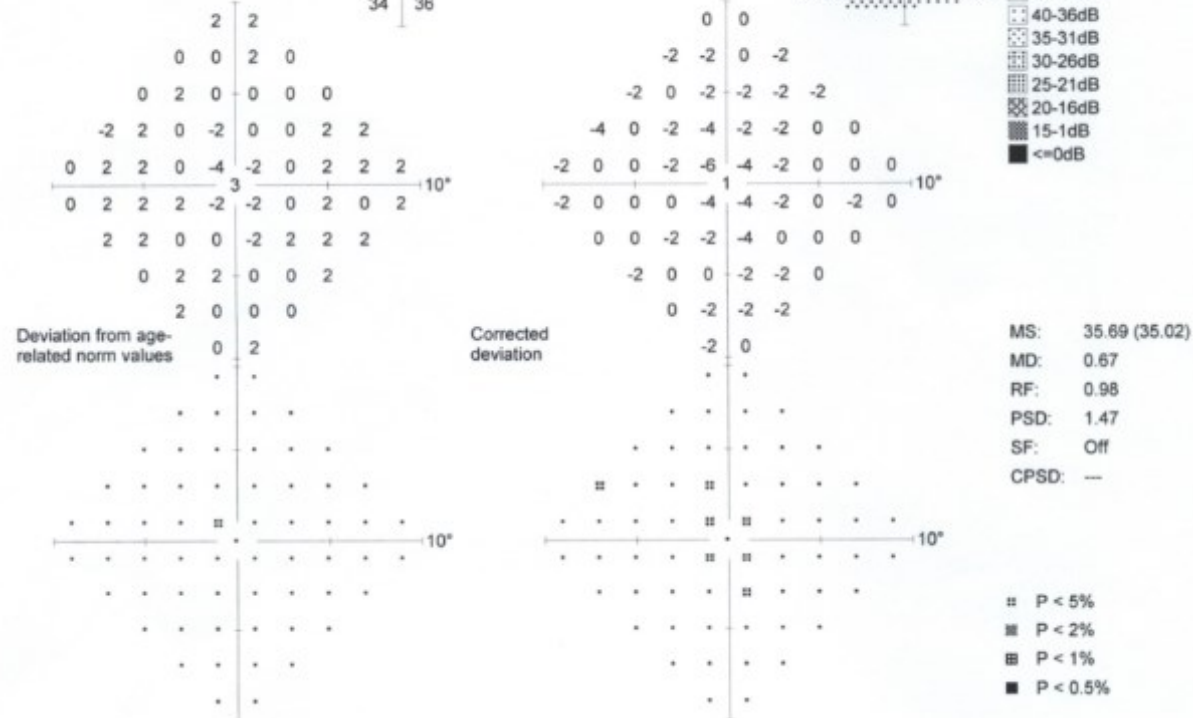
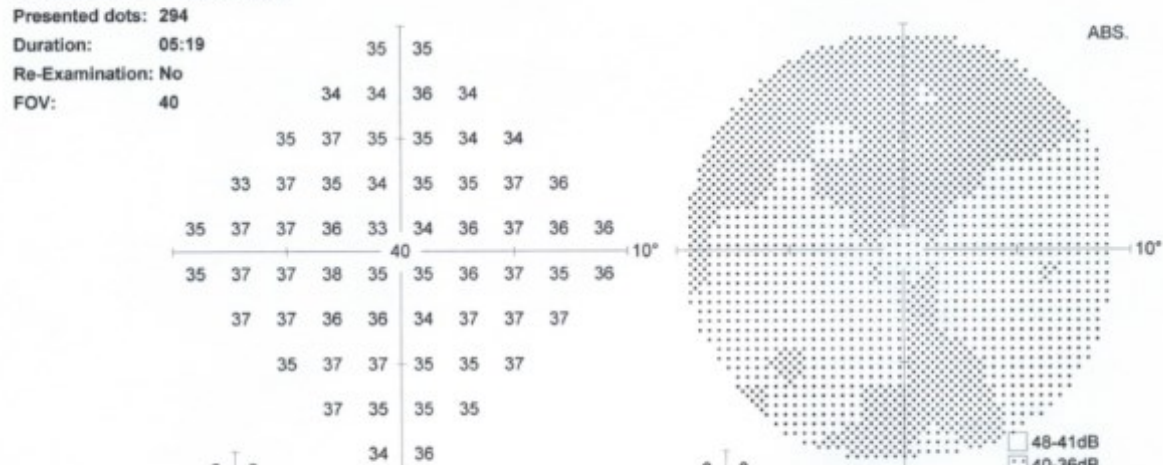
PMID: 32758681



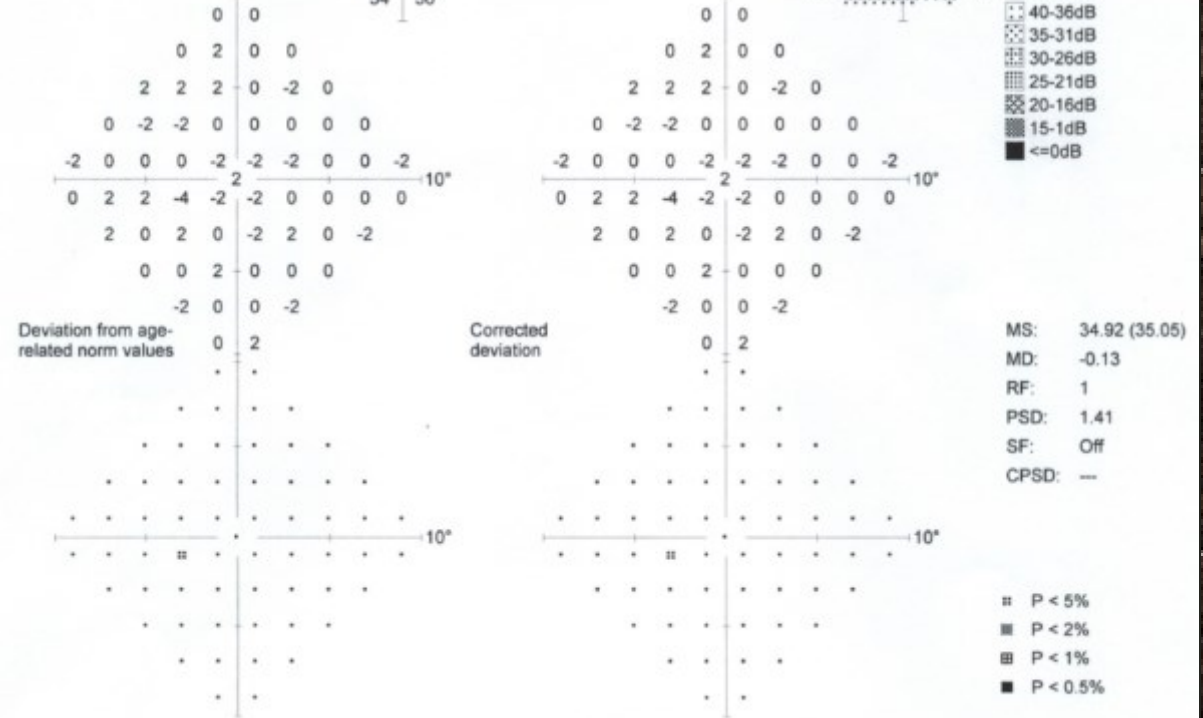
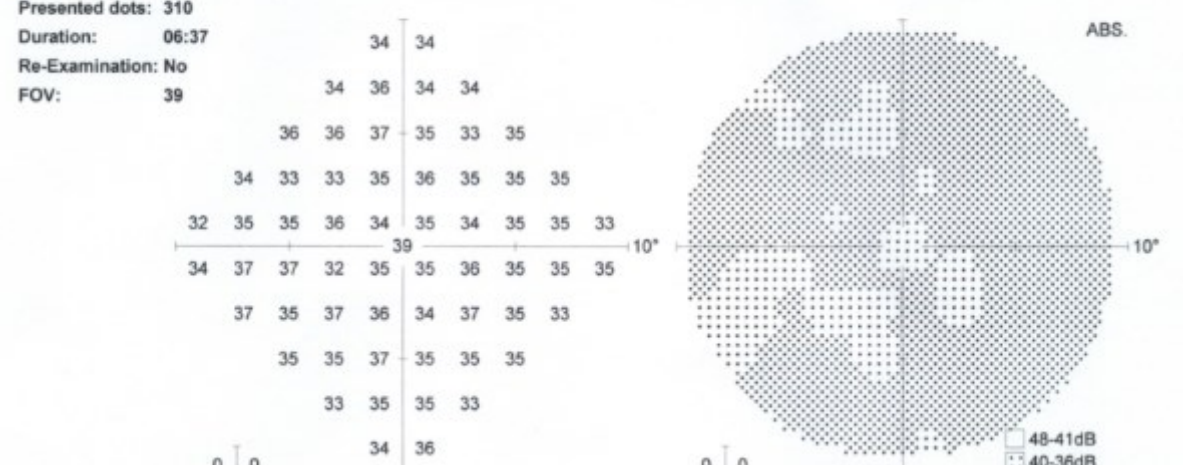


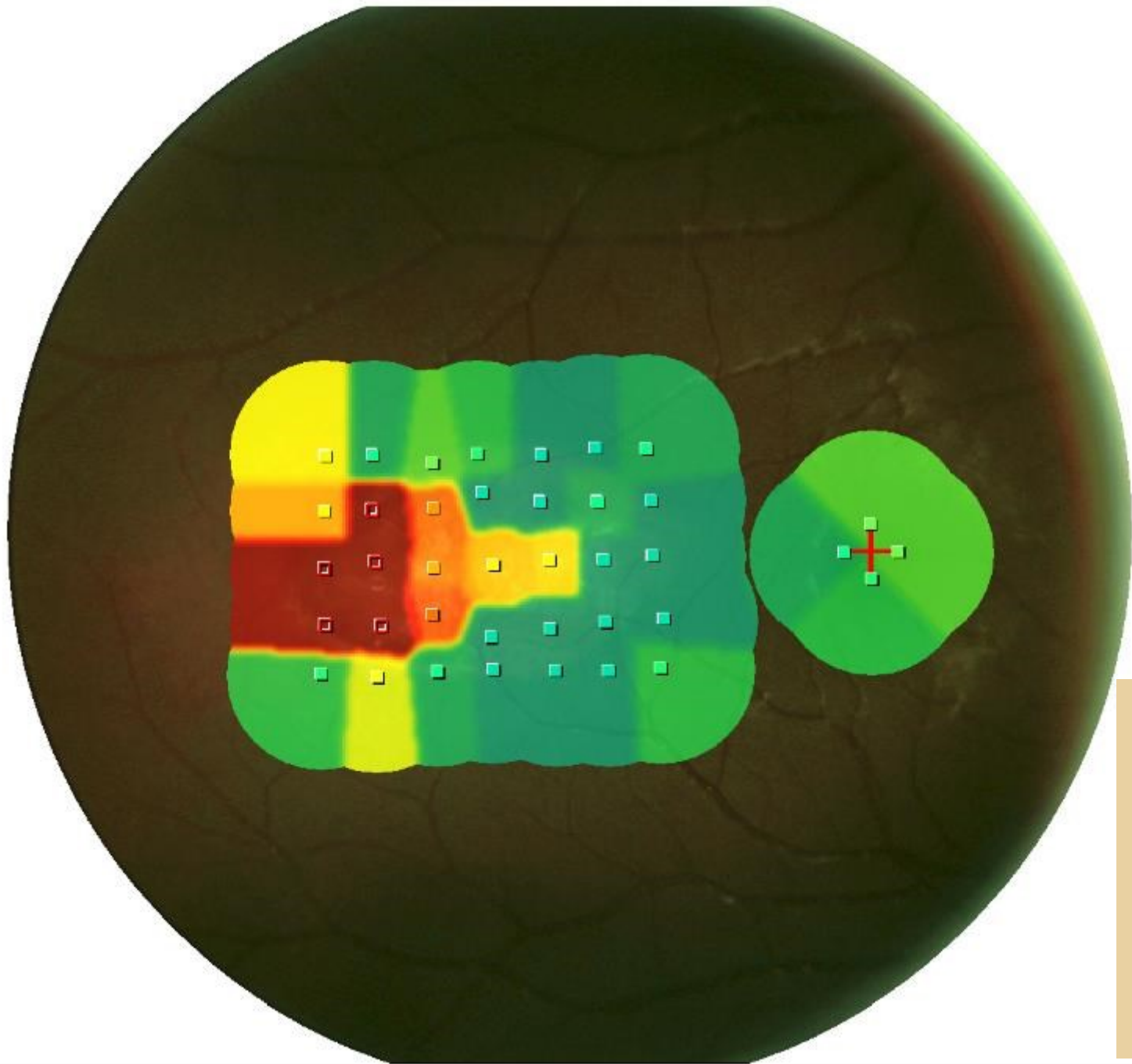
**Fundus autofluorescence (FAF)** uses blue light to stimulate lipofuscin in RPE cells to glow. Regions without viable RPE will appear dark.

Program: Macula screening Stimulus: Ill, white Pupil: --- Date of exam.: 07/23/2012  
 Area: 10-2 Background: 10 cd/m<sup>2</sup> (31.8 asb) Presentation time: 0.2 sec Time: 11:31:07  
 Strategy: Threshold Correction: No Interval time: 0.6 sec Age: 25  
 Fixation: Central 0 dB: 3180 cd/m<sup>2</sup> (simulated) Abs.loss: 0  
 Fixationcheck: 0/10 (0% Losses) Rel.loss: 0  
 False positive: 1/26 (4% Error)



Program: Macula screening Stimulus: Ill, white Pupil: --- Date of exam.: 07/23/2012  
 Area: 10-2 Background: 10 cd/m<sup>2</sup> (31.8 asb) Presentation time: 0.2 sec Time: 11:40:27  
 Strategy: Threshold Correction: No Interval time: 0.6 sec Age: 25  
 Fixation: Central 0 dB: 3180 cd/m<sup>2</sup> (simulated) Abs.loss: 0  
 Fixationcheck: 0/11 (0% Losses) Rel.loss: 0  
 False positive: 0/28 (0% Error)



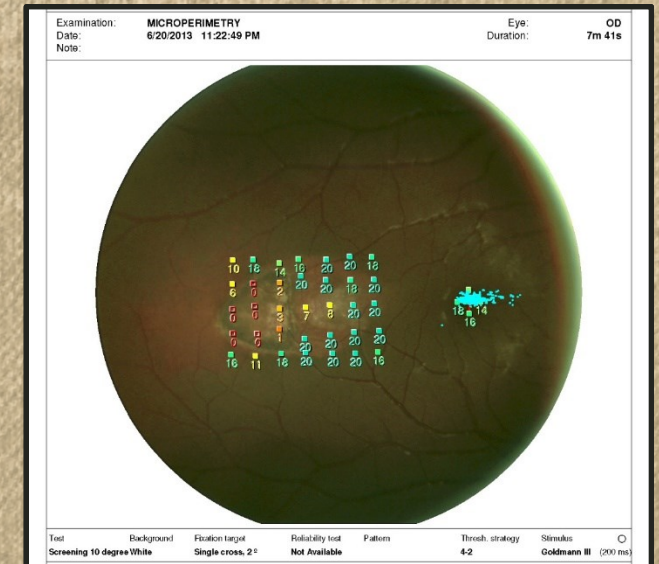


**Microperimetry** allows a retinal sensitivity map to be overlaid on a real-time fundus photograph



# About Microperimetry

- A visual field test that simultaneously performs perimetry and retinal imaging
- Stimuli can be placed at specific points on the retina
- Why use it?
  - Enables correlation of visual function (perimetry) with anatomy (retinal imaging)
  - Train eccentric viewing in low vision pts with central scotoma



# What is going on here?

<https://app.tophat.com/e/777538>



Macular coloboma **A**

Macular dystrophy **B**

Congenital toxoplasmosis **C**

Amelanotic choroidal nevus **D**

Torpedo maculopathy **E**

**Macular coloboma  
(macular dysplasia)**

A heterogenous group of developmental abnormalities. Often familial, frequently **bilateral**, systemic abnormalities not uncommon.

**Macular dystrophy**

Widespread retinal dysfunction, **bilateral** macular lesions, inheritance pattern, may be associated with features of Leber's amaurosis or RP. Electrophysiologic abnormalities common

**Macular scars**

Intrauterine infection with *Toxoplasma gondii* resulting in congenital toxoplasmic chorioretinitis.

**Amelanotic  
choroidal nevus**

Shares all the features of a typical choroidal nevus minus the melanin. Choroidal nevi are flat or slightly elevated – never excavated

**Torpedo  
maculopathy**

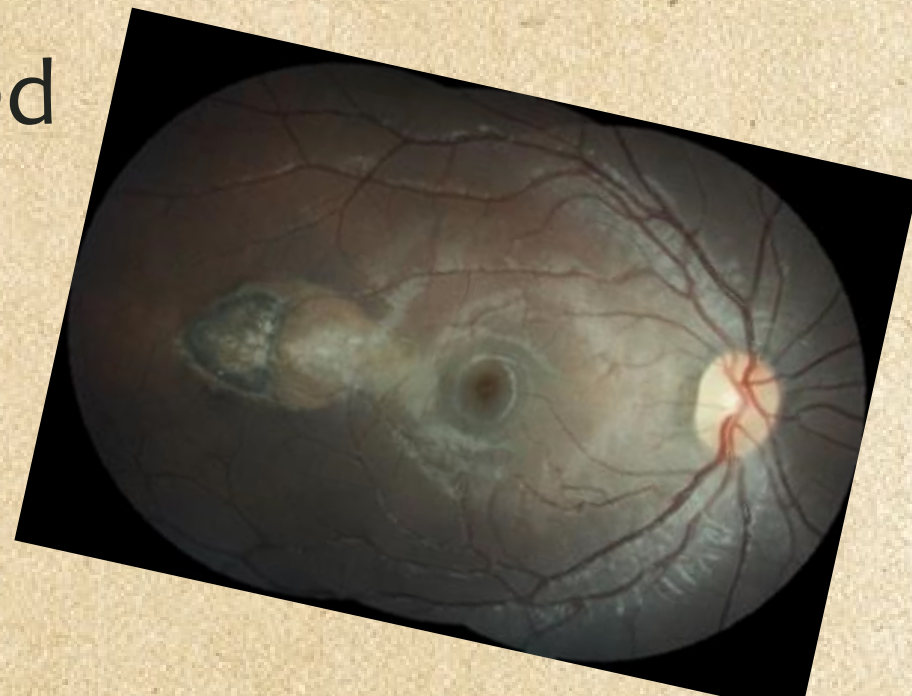
**Unilateral** congenital abnormality, characteristic “torpedo” shape, always located temporal to the fovea

# Assessment

- Torpedo maculopathy OD

# Management

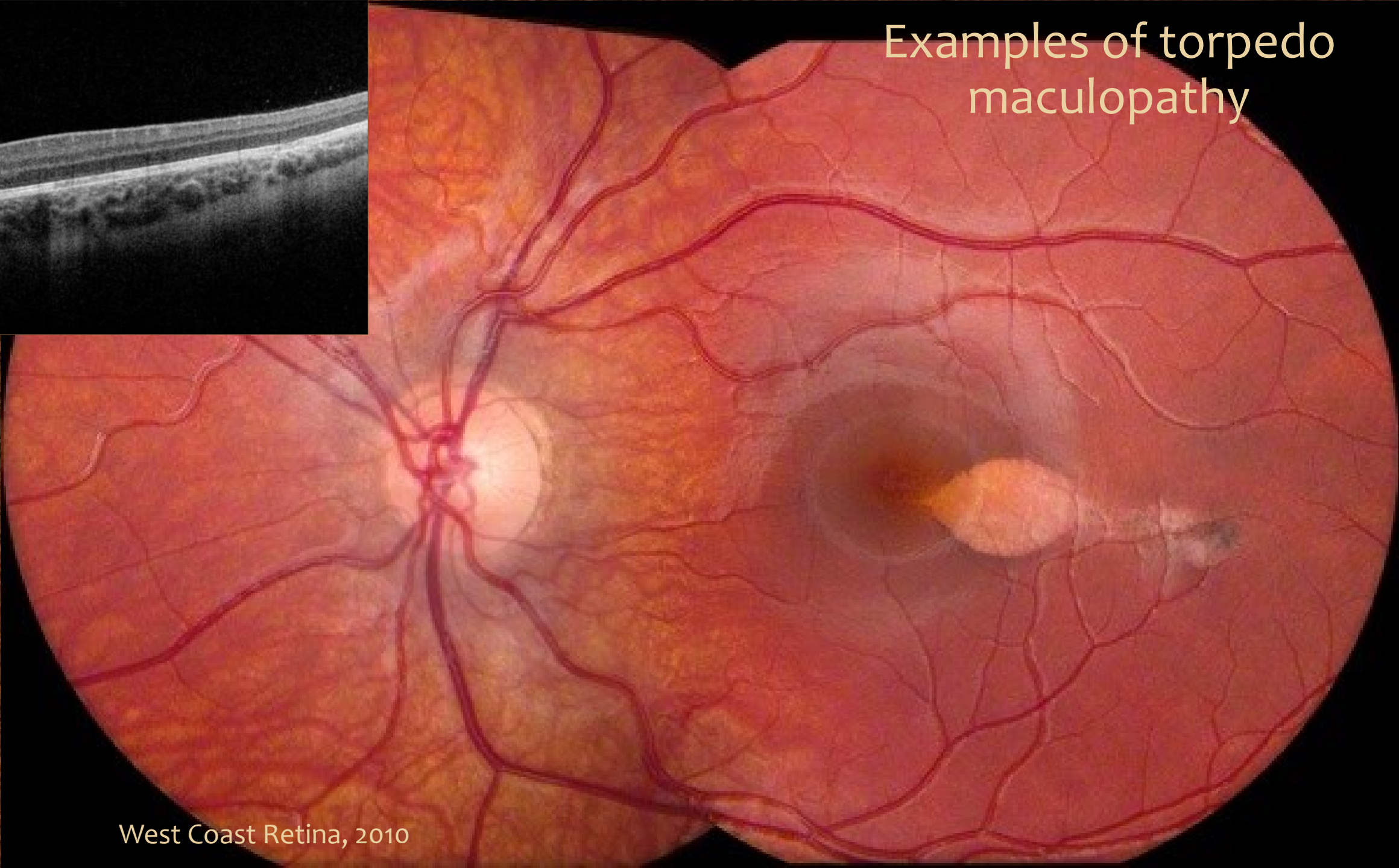
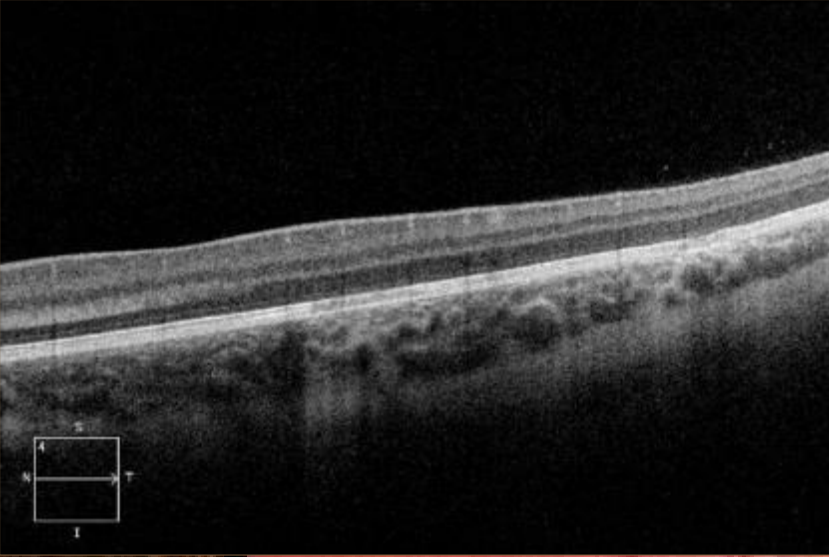
- No specific treatment indicated
- Patient education/Amsler
- Routine eye care



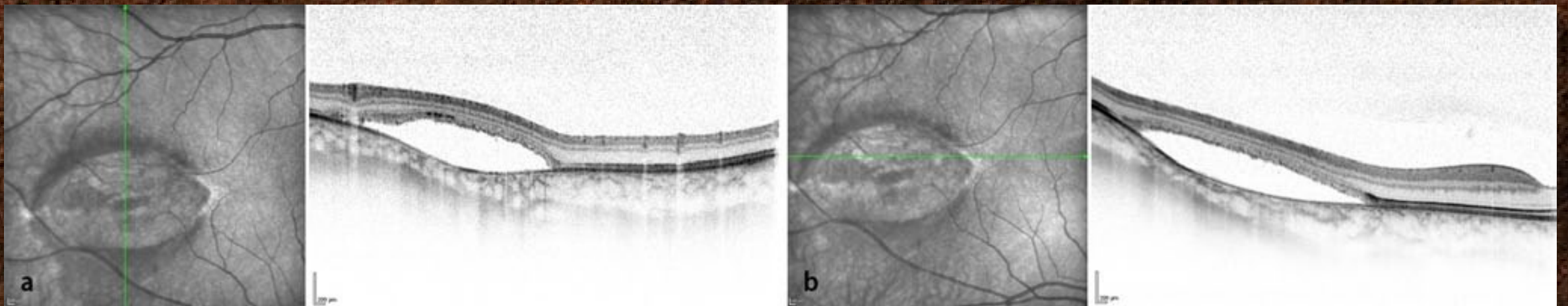
# Torpedo Maculopathy

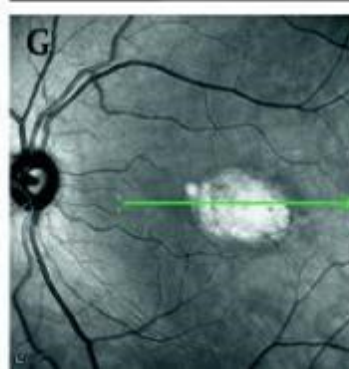
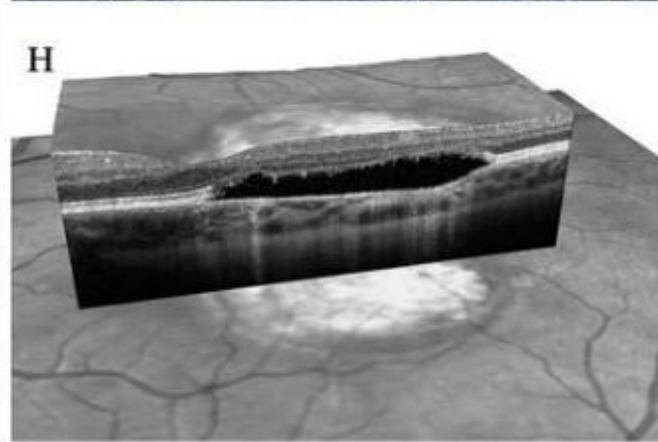
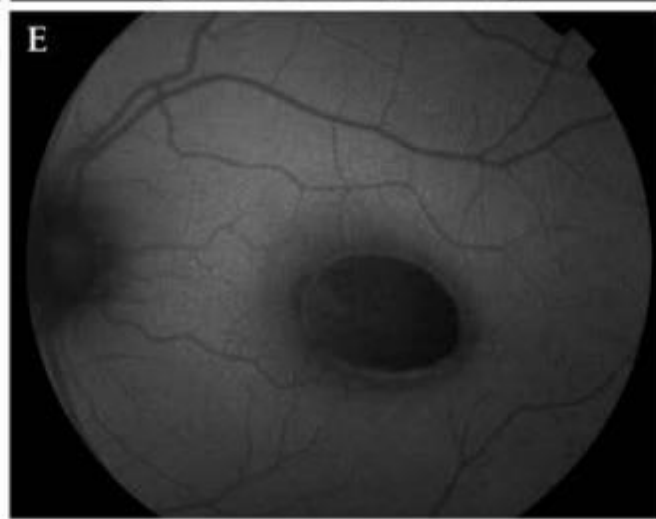
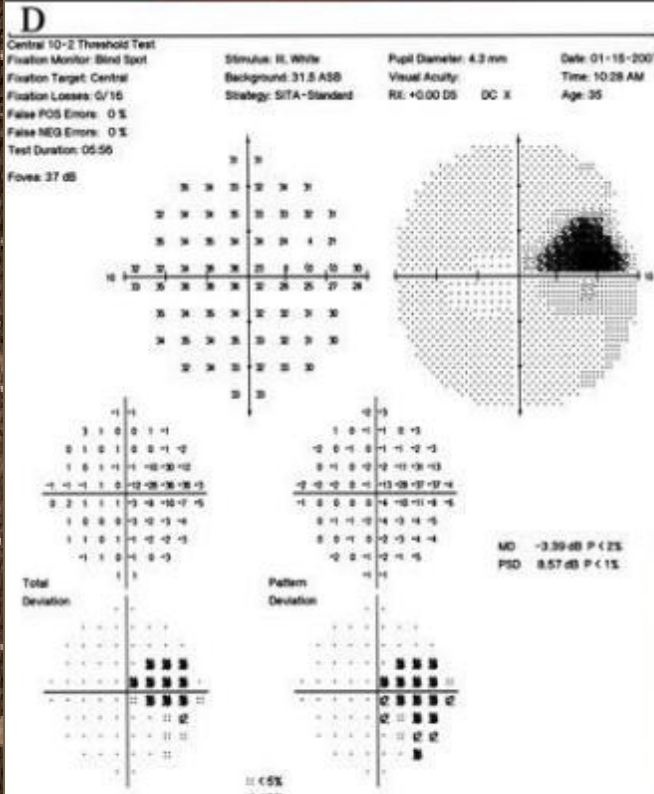
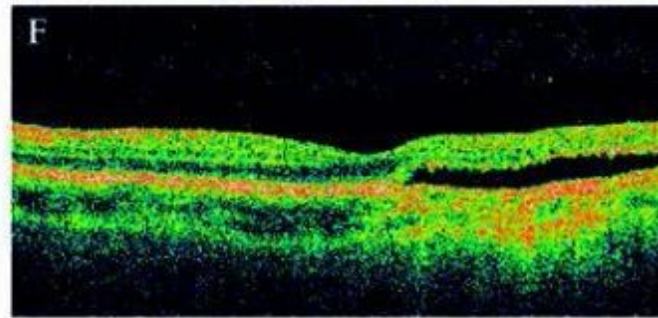
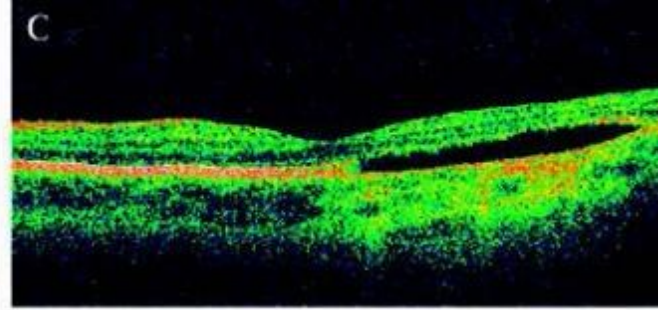
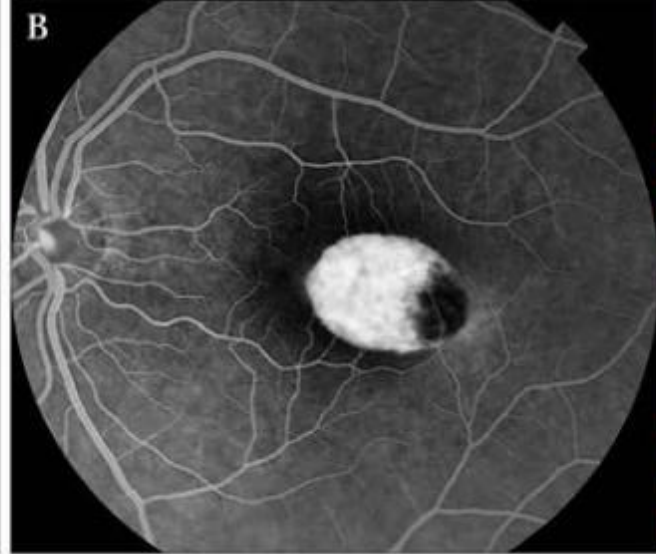
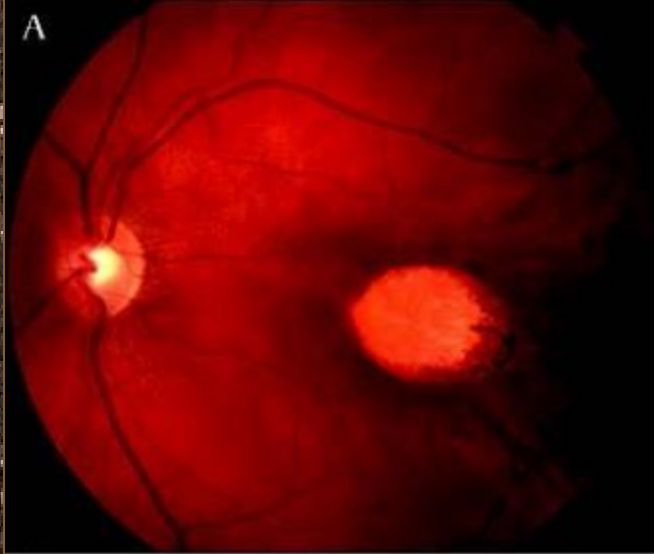
- Congenital, hypopigmented torpedo-shaped lesion in the temporal macula along the horizontal raphe
- Developmental defect of unknown etiology
- May encroach upon fovea, but rarely causes significant loss of vision
- Diagnosis based upon characteristic appearance and nonrandom location
- No treatment is required for this stable congenital lesion

# Examples of torpedo maculopathy



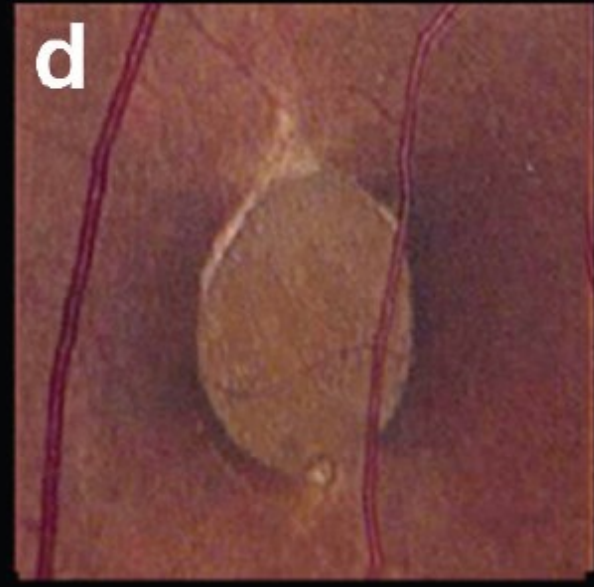
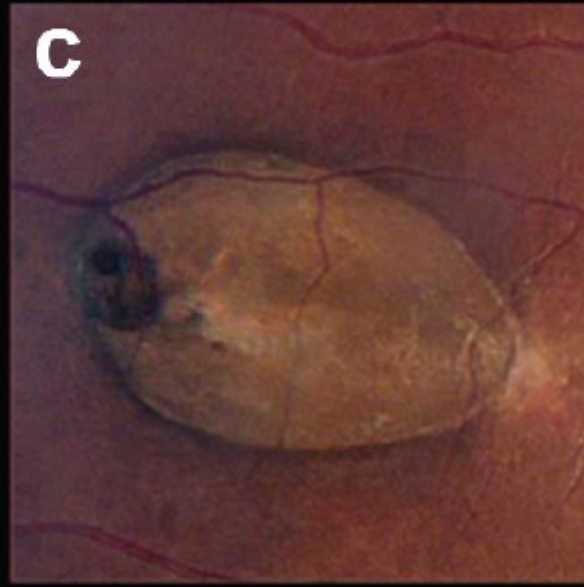
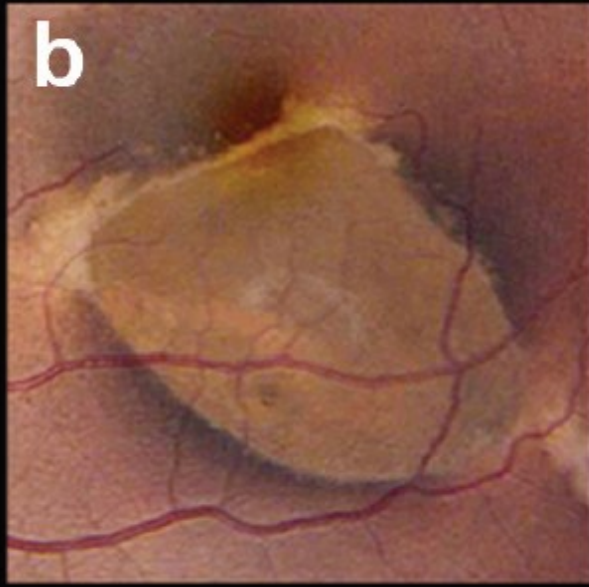
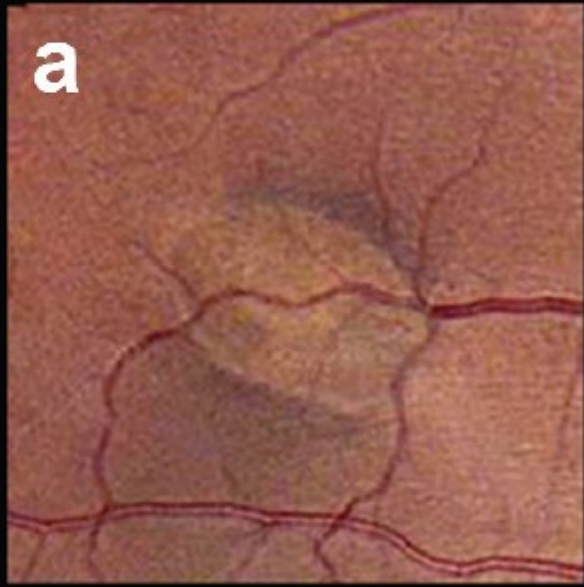
# Examples of torpedo maculopathy





PMID: 19822914





## Is torpedo maculopathy a subclinical form of Gardner Syndrome?

- (1) Genetic testing for Familial Adenomatous Polyposis
- (2) Colonoscopy for intestinal polyps
- (3) Eye exam of relatives for signs of Gardner syndrome.

# Take Home Message

- Torpedo maculopathy
  - Congenital perimacular lesion
  - Distinctive appearance
  - Usually asymptomatic
  - Rule out Gardner Syndrome



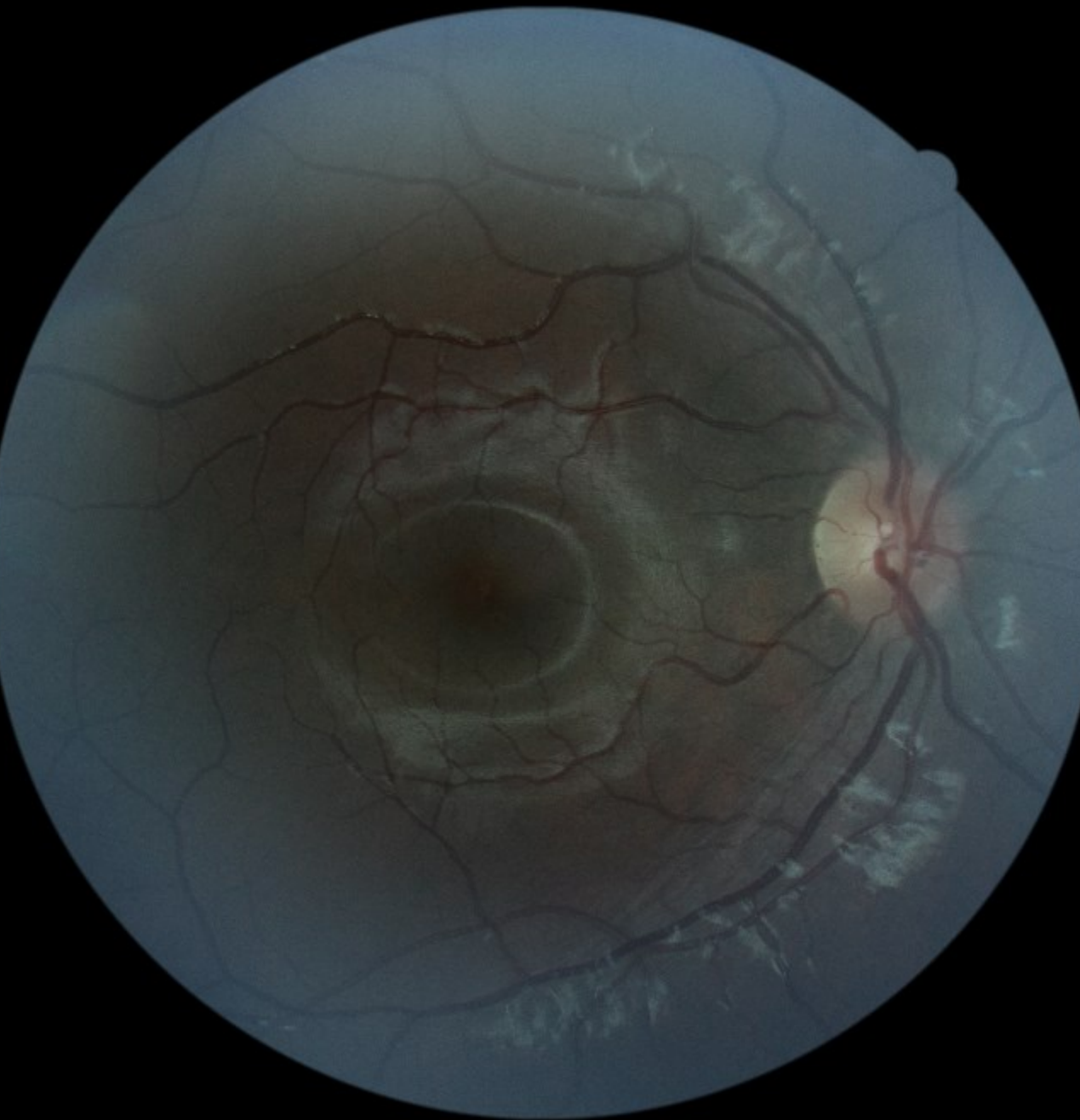
# CASE #2

 *Here comes the sun...* 

# CASE #2A

---

- 13yo Asian male referred for abnormal appearance of right macula on OCT
- POH: Refractive amblyopia OS
- PMH: Good health. No meds
  
- BCVA: 20/20 OD, 20/40 OS
- Ta: 12/13 @ 4PM; PERRL, No APD
- SLE: W&Q OU



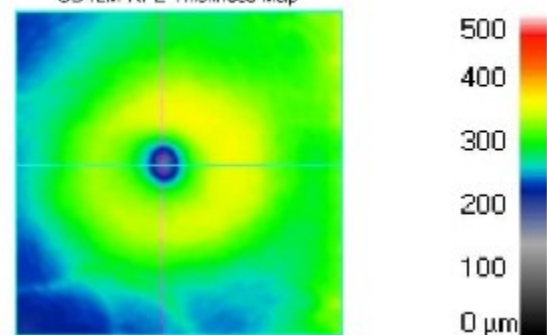


**Fundus  
Autofluorescence**

# Macula Thickness OU: Macular Cube 512x128

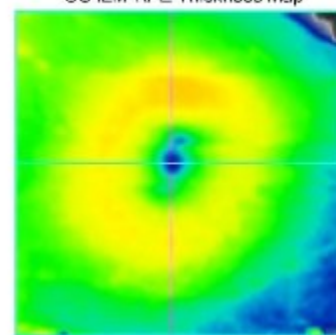
OD ● ● OS

OD ILM-RPE Thickness Map



Fovea: 232, 61

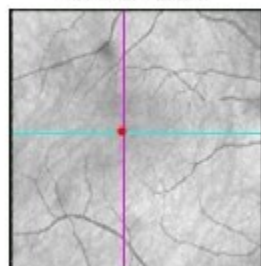
OS ILM-RPE Thickness Map



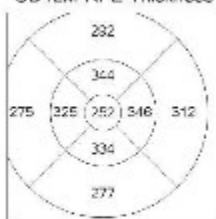
Fovea: 246, 60

Normative data is not available. Patient age < 18.

OD OCT Fundus



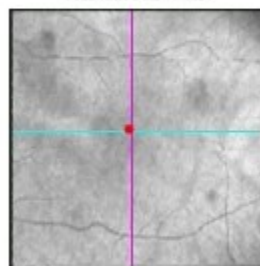
OD ILM-RPE Thickness



OS ILM-RPE Thickness



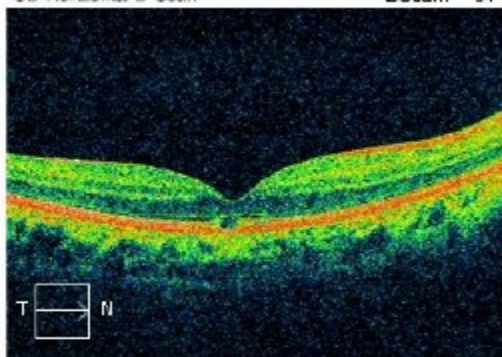
OS OCT Fundus



ILM - RPE	OD	OS
Thickness Central Subfield (µm)	252	274
Volume Cube (mm <sup>3</sup> )	10.6	11.1
Thickness Avg Cube (µm)	294	309

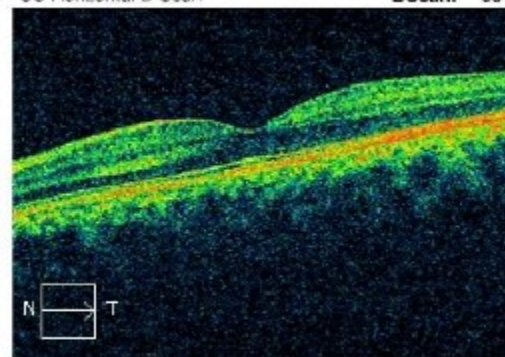
OD Horizontal B-Scan

BScan: 61



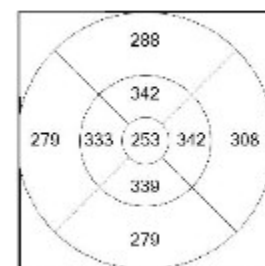
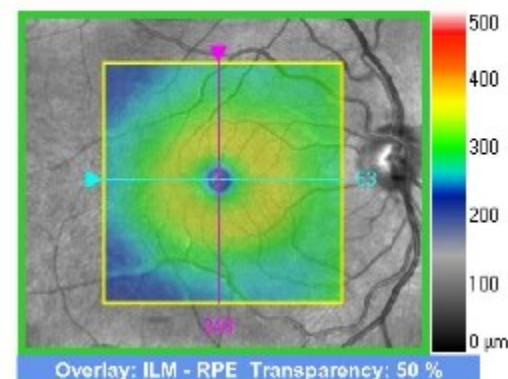
OS Horizontal B-Scan

BScan: 60

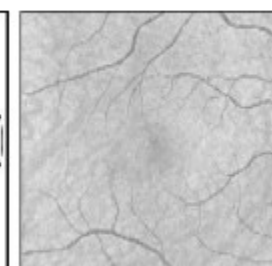


# Macula Thickness : Macular Cube 512x128

OD ● ○ OS

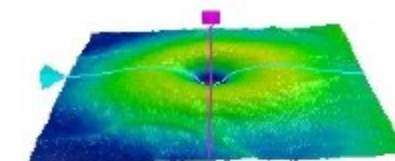
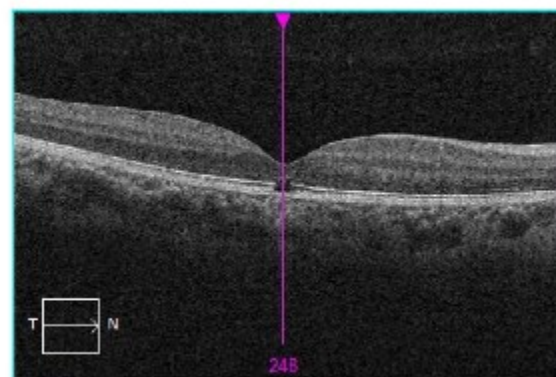


ILM-RPE Thickness (µm)

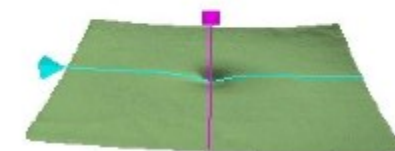
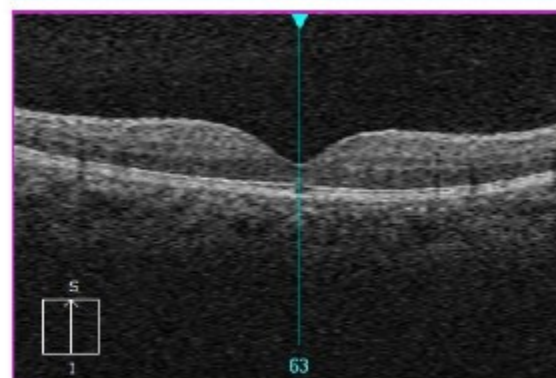


Fovea: 248, 64

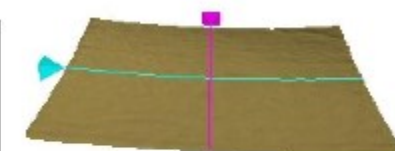
Overlay: ILM - RPE Transparency: 50 %



ILM - RPE



ILM



RPE

Normative data is not available. Patient age < 18.

	Central Subfield Thickness (µm)	Cube Volume (mm <sup>3</sup> )	Cube Average Thickness (µm)
ILM - RPE	253	10.5	291

## CASE #2B

---

- 12yo Asian F (sibling of Case 2A) C/O “color difference” between eyes, with vision of right eye appearing yellowish x 1 month
- POH: LEE 2yrs
- PMH: Good health, no meds
  
- BCVA: 20/25 OD, 20/20 OS
- Ta 15/15 @11AM; PERRL No APD
- SLE: W&Q OU



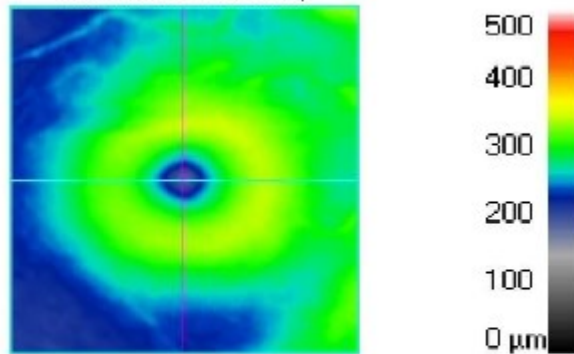
# Macula Thickness OU: Macular Cube 512x128

OD ● ● OS

# Macula Thickness : Macular Cube 512x128

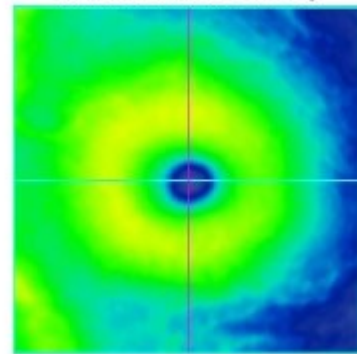
OD ● ○ OS

OD ILM-RPE Thickness Map



Fovea: 254, 65

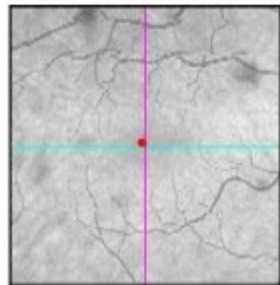
OS ILM-RPE Thickness Map



Fovea: 259, 65

Normative data is not available. Patient age < 18.

OD OCT Fundus



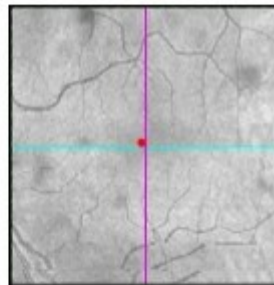
OD ILM-RPE Thickness



OS ILM-RPE Thickness



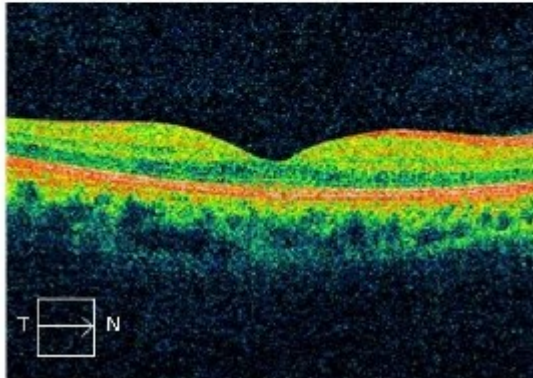
OS OCT Fundus



ILM - RPE	OD	OS
Thickness Central Subfield (µm)	237	238
Volume Cube (mm³)	9.9	10.1
Thickness Avg Cube (µm)	274	282

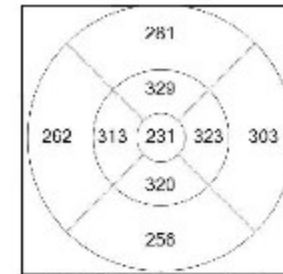
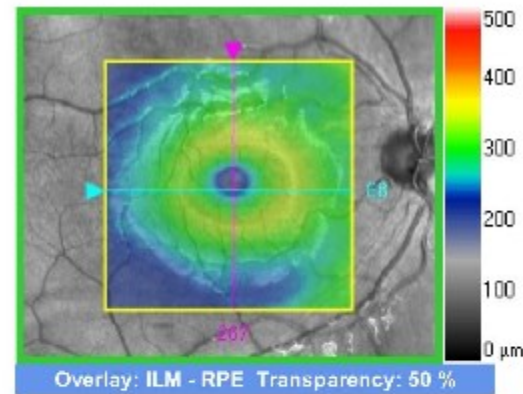
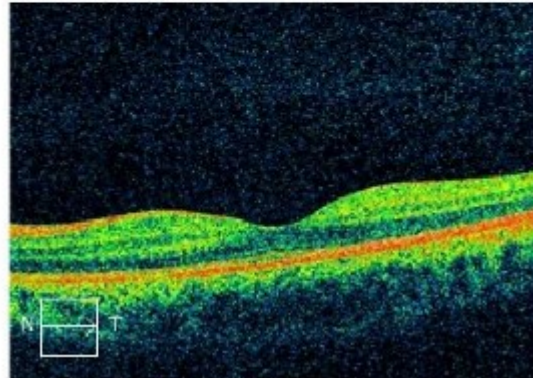
OD Horizontal B-Scan

BScan: 65

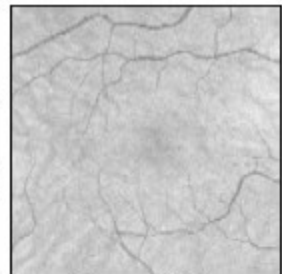


OS Horizontal B-Scan

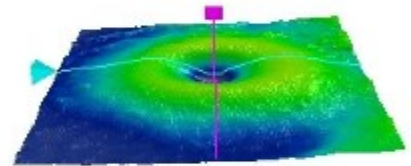
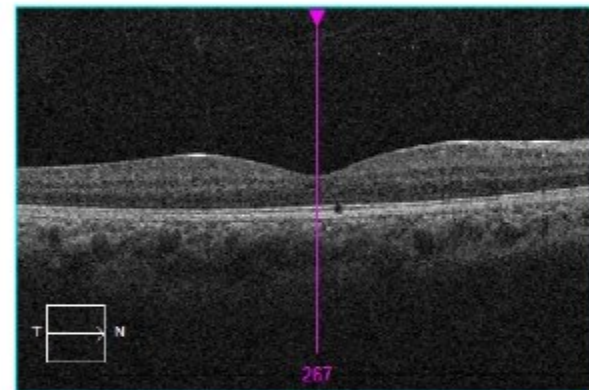
BScan: 65



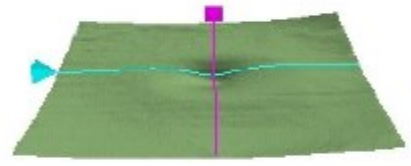
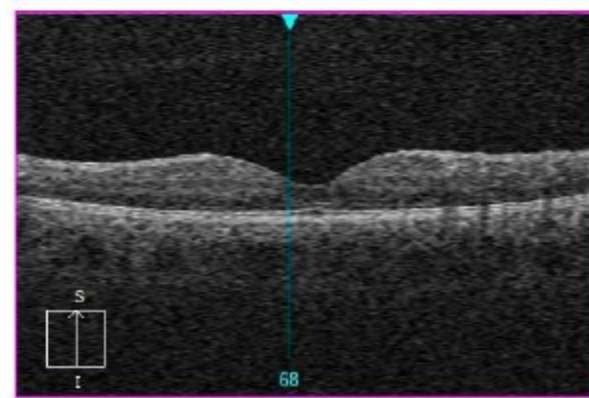
ILM-RPE Thickness (µm)



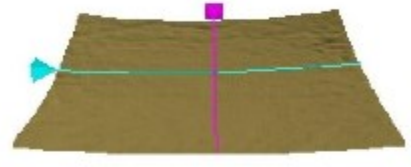
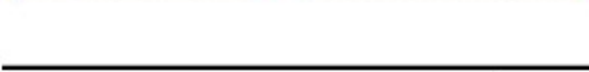
Fovea: 265, 63



ILM - RPE



ILM



RPE

Normative data is not available. Patient age < 18.

	Central Subfield Thickness (µm)	Cube Volume (mm³)	Cube Average Thickness (µm)
ILM - RPE	231	10.0	277

Comments

Doctor's Signature

# What is going on here?

<https://app.tophat.com/e/777538>



Macular Coloboma

Macular Dystrophy

Macular Hole

Solar Maculopathy

Macular Edema

**Macular coloboma  
(macular dysplasia)**

A heterogenous group of developmental abnormalities. Often **familial**, frequently **bilateral**, systemic abnormalities not uncommon.

**Macular dystrophy**

Widespread retinal dysfunction, **bilateral** macular lesions, **inheritance pattern**, may be associated with features of Leber's amaurosis or RP. Electrophysiologic abnormalities common

**Macular hole**

**Full-thickness** retinal defect associated with major loss of visual acuity. Early stage or lamellar lesions associated with **inner retinal defects**

**Solar maculopathy**

Bilateral or unilateral (dominant eye) **outer retinal OCT defect** with history of sun gazing

**Macular edema**

Intraretinal fluid accumulation with a corresponding **increase in macular thickness** on OCT

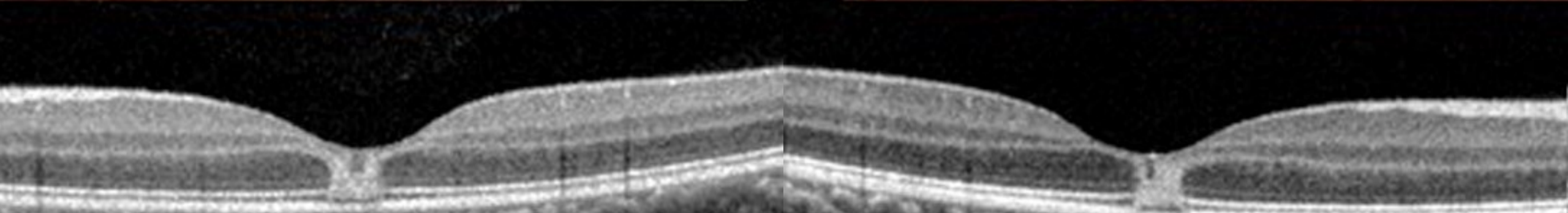
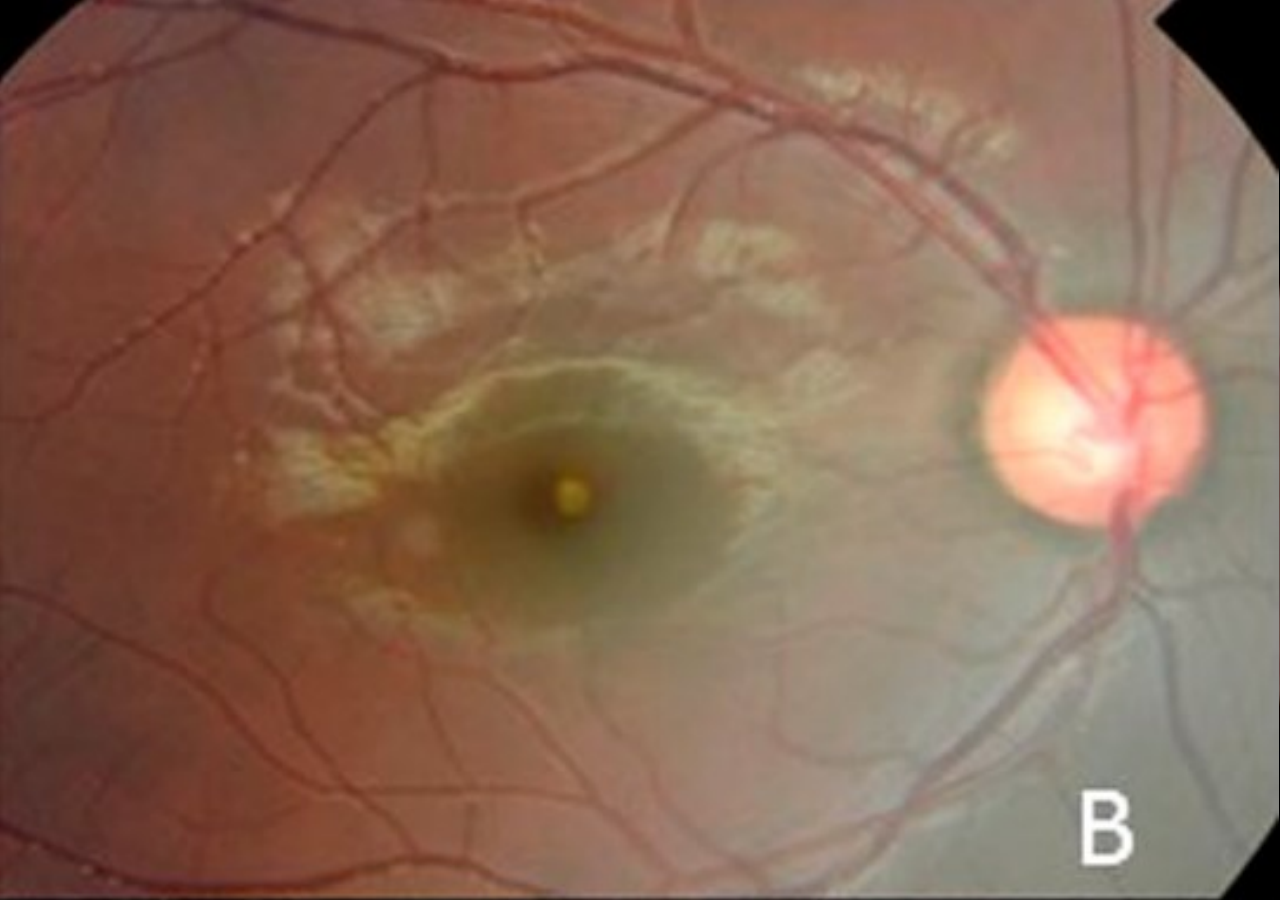
# Solar Maculopathy

- Thermal and photochemical damage to the RPE and photoreceptors
- **Solar maculopathy**: Prolonged direct sun gazing. Usually associated with eclipse viewing
- **Photic retinopathy**: Umbrella term that encompasses all retinal damage from light (laser, arc welding, solar)



# Solar Maculopathy

- Clinical manifestations vary with severity of exposure
- Rapid VA decrease (20/30-20/100) following exposure
- Little or no ophthalmoscopic changes in mild cases (indistinct FLR, greyish macula) or a yellowish RPE lesion in more severe cases
- Vision gradually (months) returns to normal or near normal. There is less recovery in severe cases.
- An outer retinal cyst-like lesion develops and remains permanently (Ddx: MacTel, Tamoxifen)



**Early severe solar maculopathy  
“Eclipse burn”**

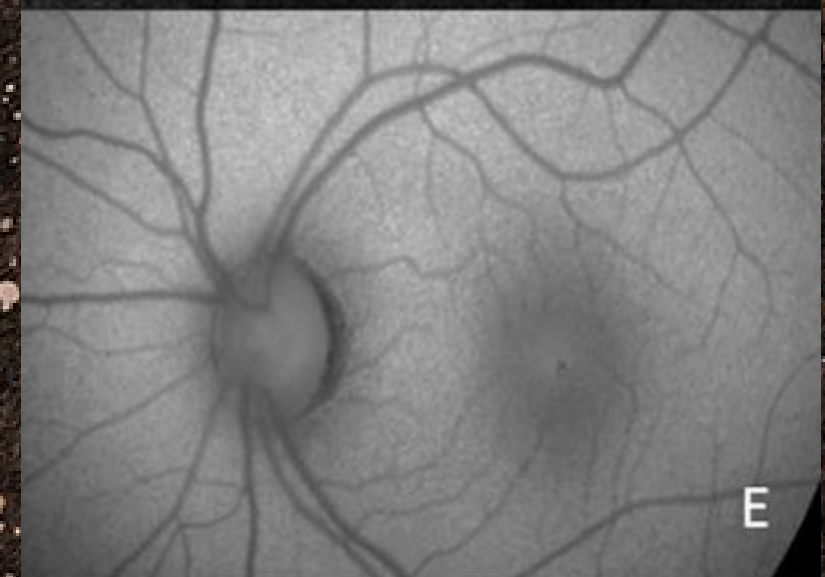
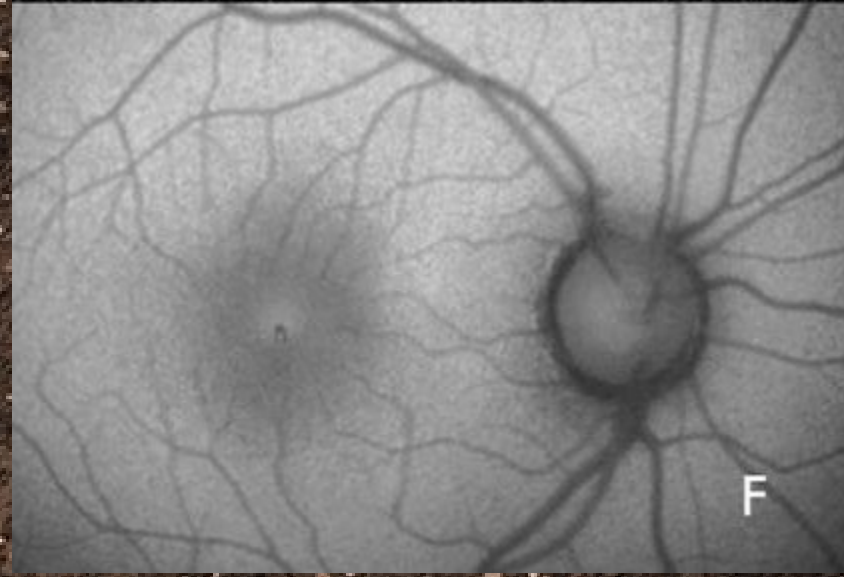
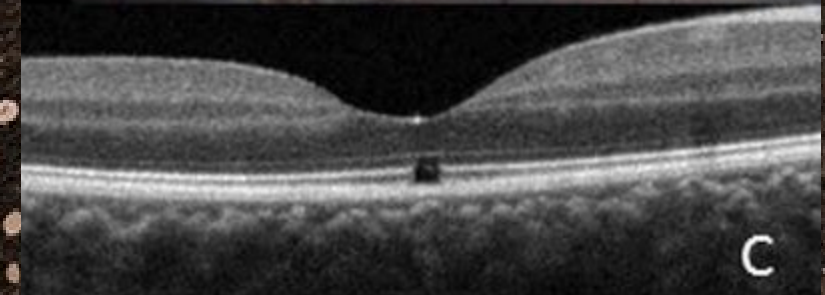
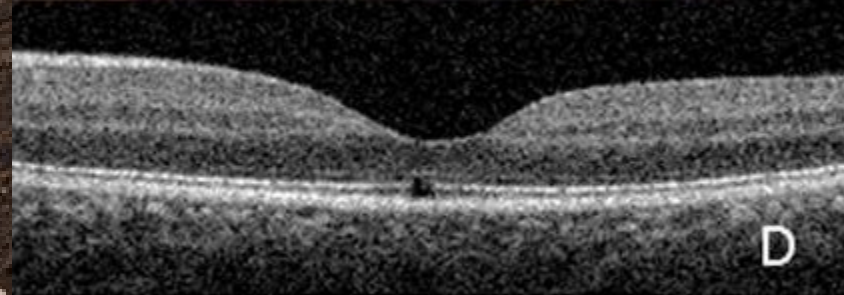
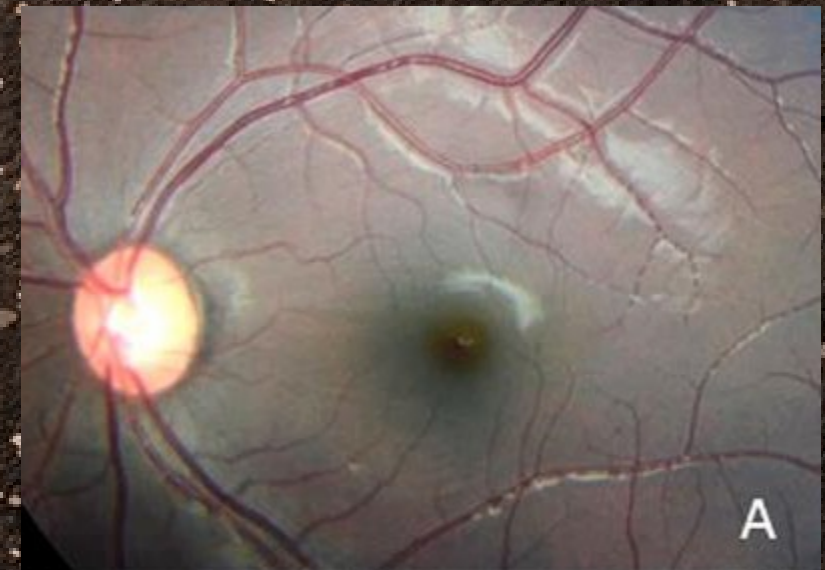
C

**Same case at 30-days**

**TOP:** Resolving yellow lesions

**MIDDLE:** Square-shaped foveal photoreceptor defect

**BOTTOM:** FAF hypofluorescent spot surrounded by hyperfluorescence

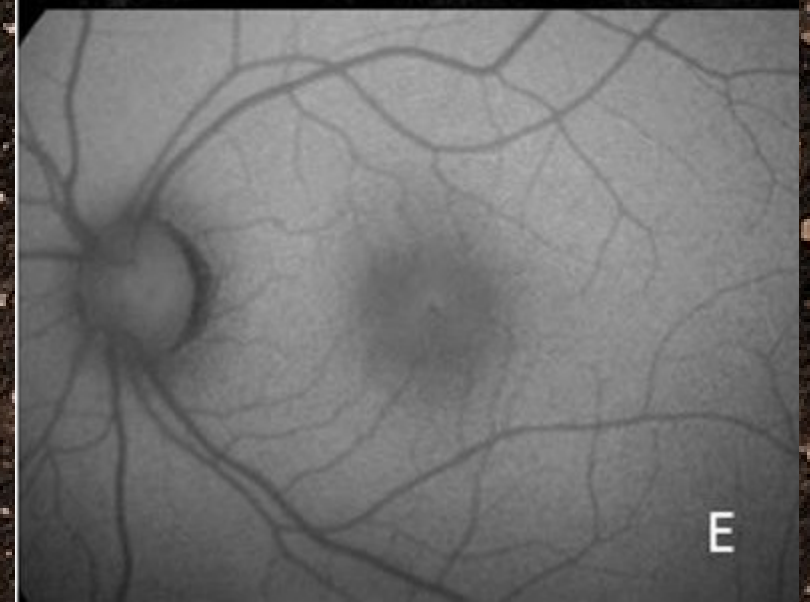
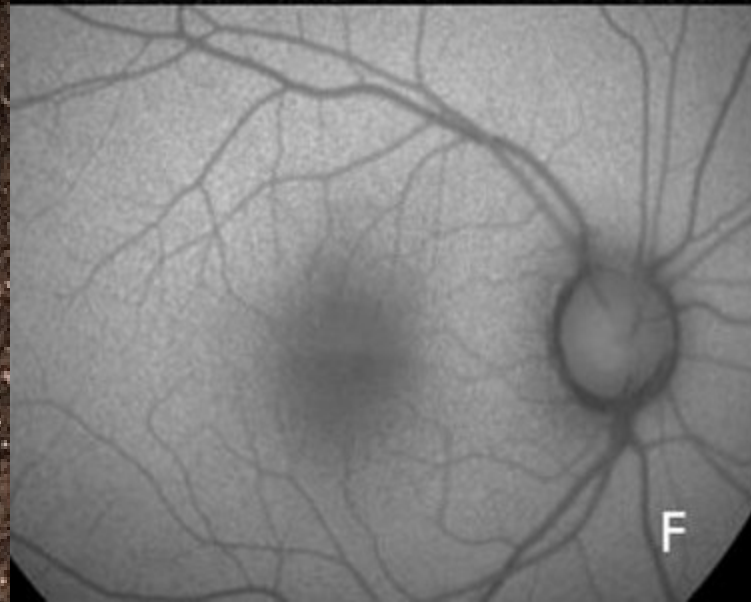
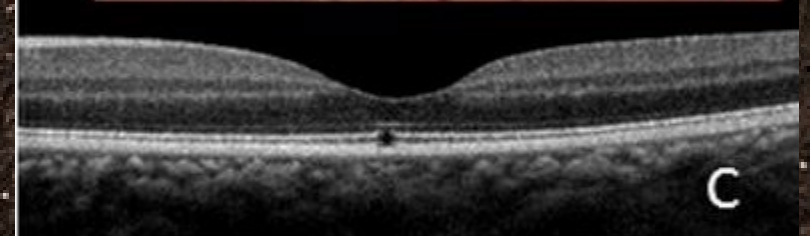
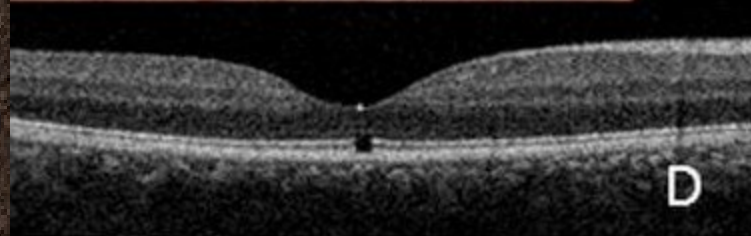
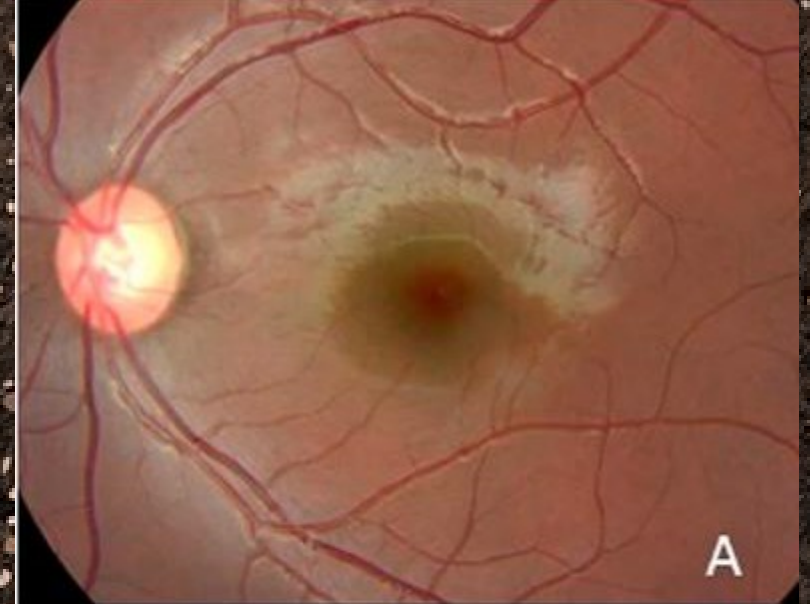


**Same case at 1 year:  
late-stage**

**TOP: Essentially normal  
ophthalmoscopic  
appearance**

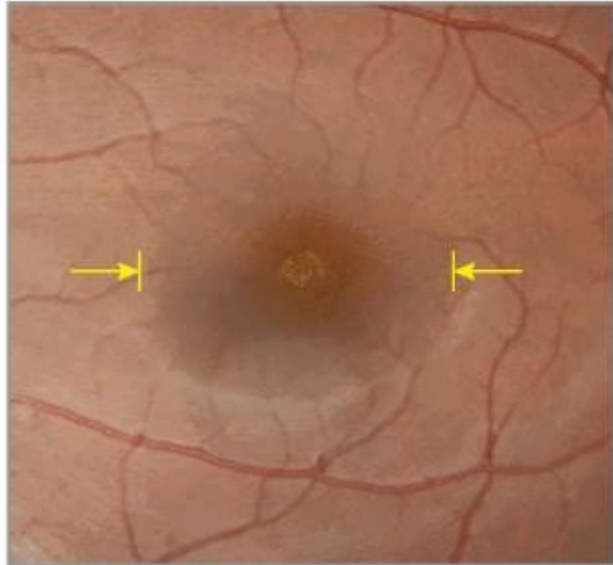
**MIDDLE: Rectangular  
photoreceptor defect  
in fovea on OCT**

**BOTTOM: FAF  
hypofluorescent spot  
corresponding to OCT  
photoreceptor defect**

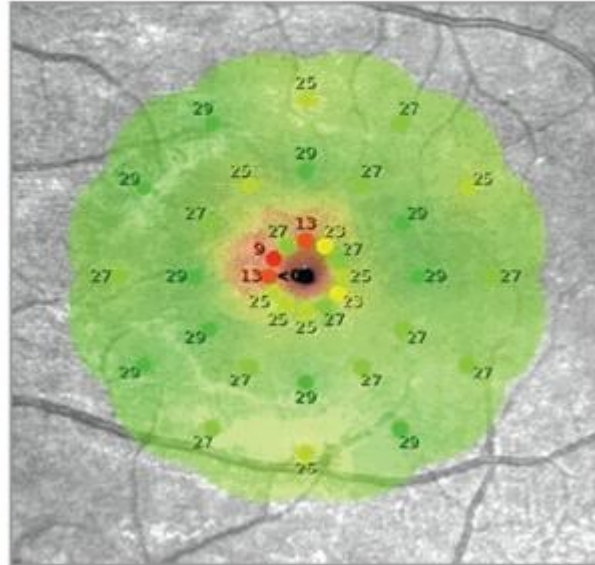




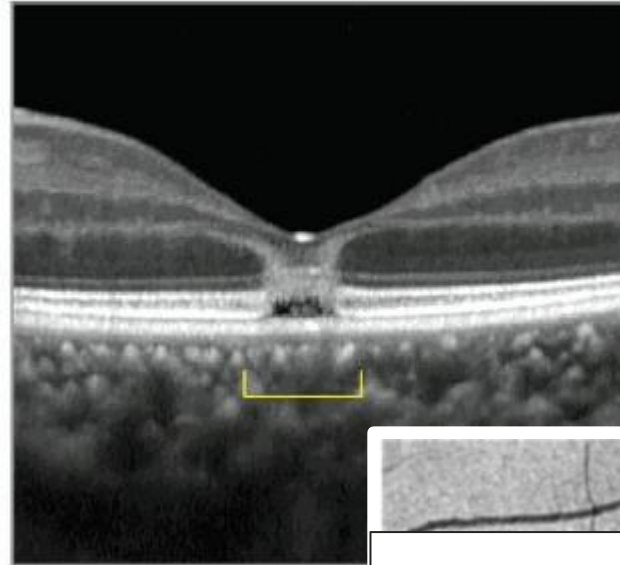
Fundus



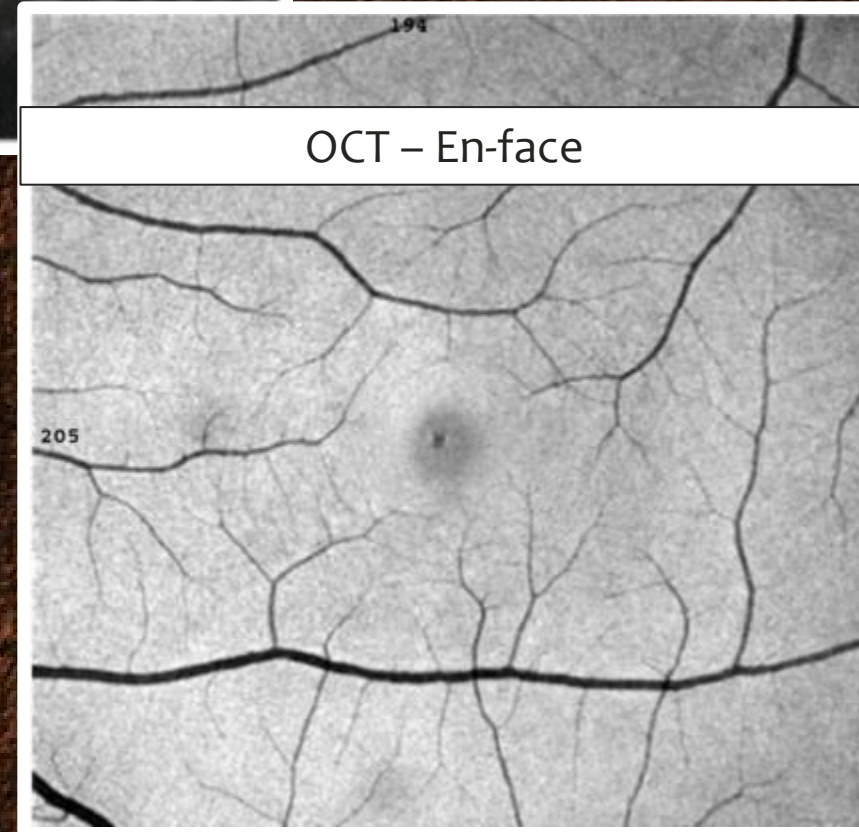
Microperimetry



OCT – B-scan



OCT – En-face



**Microperimetry documents scotoma corresponding to OCT defect**

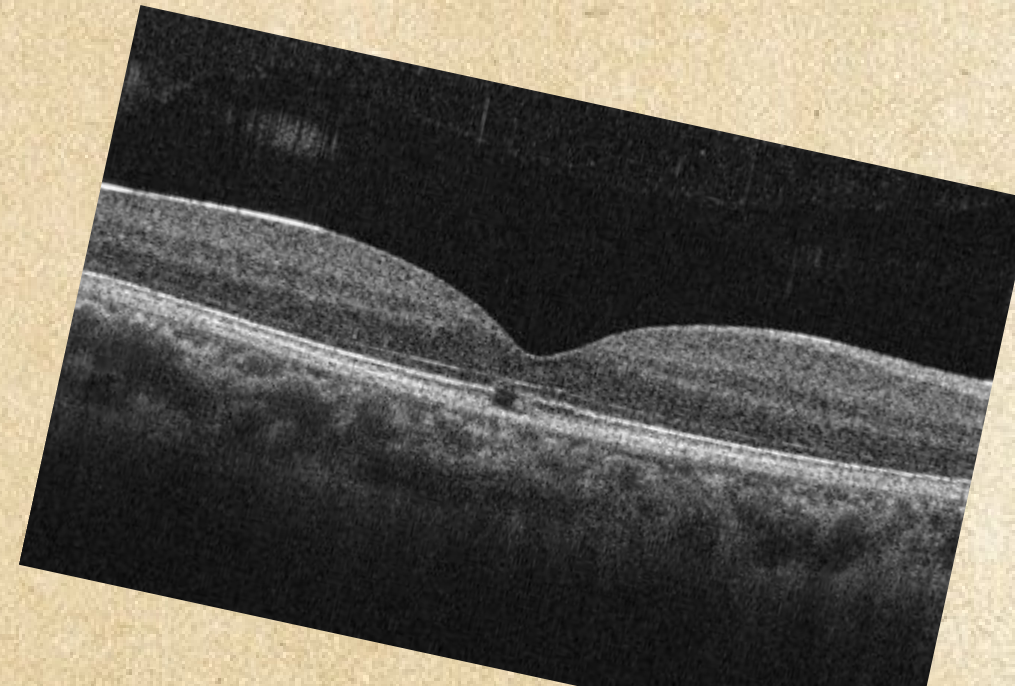
- **Visual acuity was 20/25**
- **En-face OCT –perifoveal photoreceptor punched-out lesion**

# Assessment

- Solar maculopathy secondary to sun gazing

# Management

- No specific treatment indicated
- Patient education
- Routine eye care



# Sun...Moon...You!

ANNULAR SOLAR ECLIPSE  
OCTOBER 14, 2023

APRIL 8, 2024  
TOTAL SOLAR ECLIPSE

<https://eclipse.aas.org>



# Eclipse viewing safety



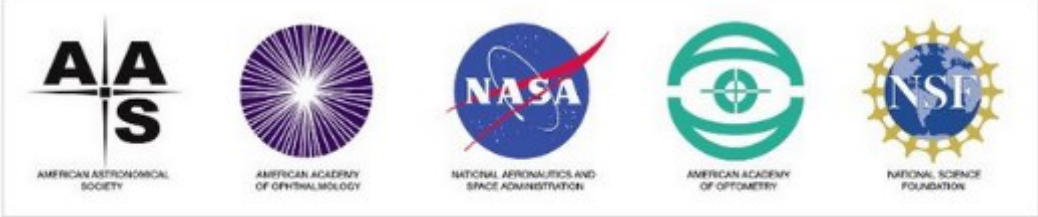
SUN-210



Home / Eclipse 101 / Safety

## Safety

Please feel free to download maps, posters, fact sheet, safety bulletin and other materials for use in your communities and events. We appreciate it if you credit NASA.



## How to View the 2017 Solar Eclipse Safely



Looking directly at the sun is unsafe except during the brief total phase of a solar eclipse ("totality"), when the moon entirely blocks the sun's bright face, which will happen only within the narrow path of totality.

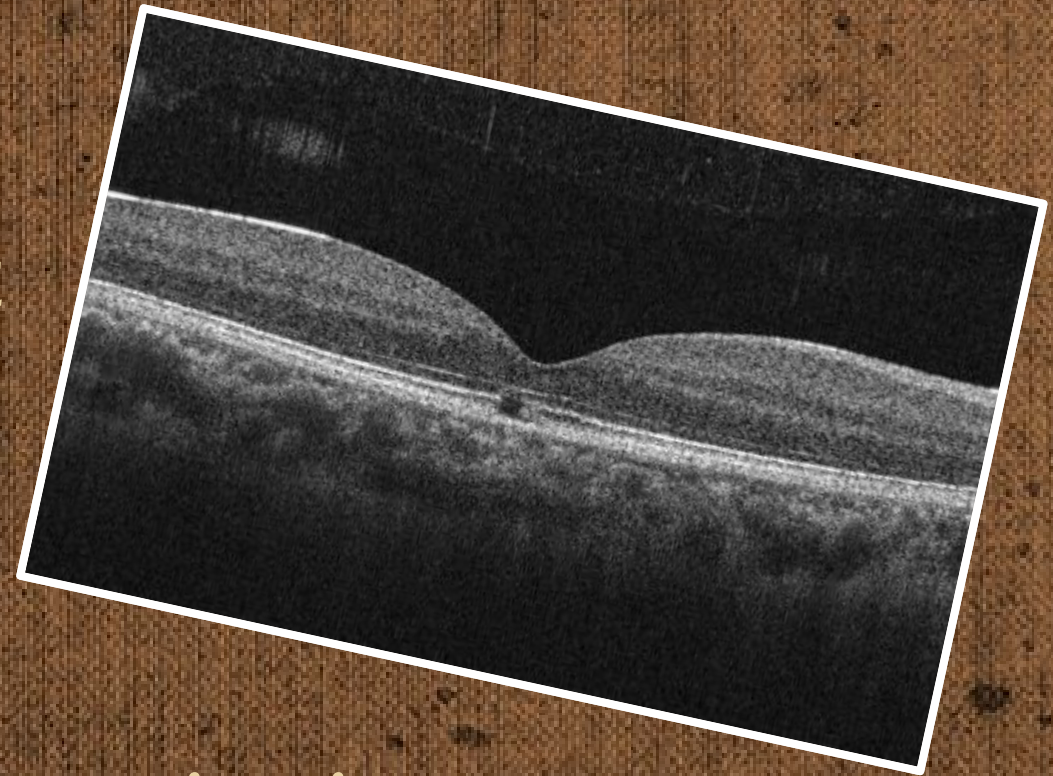


The only safe way to look directly at the uneclipsed or partially eclipsed sun is through special-purpose solar filters, such as "eclipse glasses" (example shown at left) or hand-held solar viewers. Homemade filters or ordinary sunglasses, even very dark ones, are not safe for looking at the sun; they transmit thousands of times too much sunlight. Refer to the American Astronomical Society (AAS) [Reputable](#)



# Take Home Message

- **Solar Maculopathy**
  - Mild visual acuity loss
  - Outer retinal OCT defect
  - History of sun gazing
  - No specific treatment
  - Patient education regarding safe solar eclipse viewing



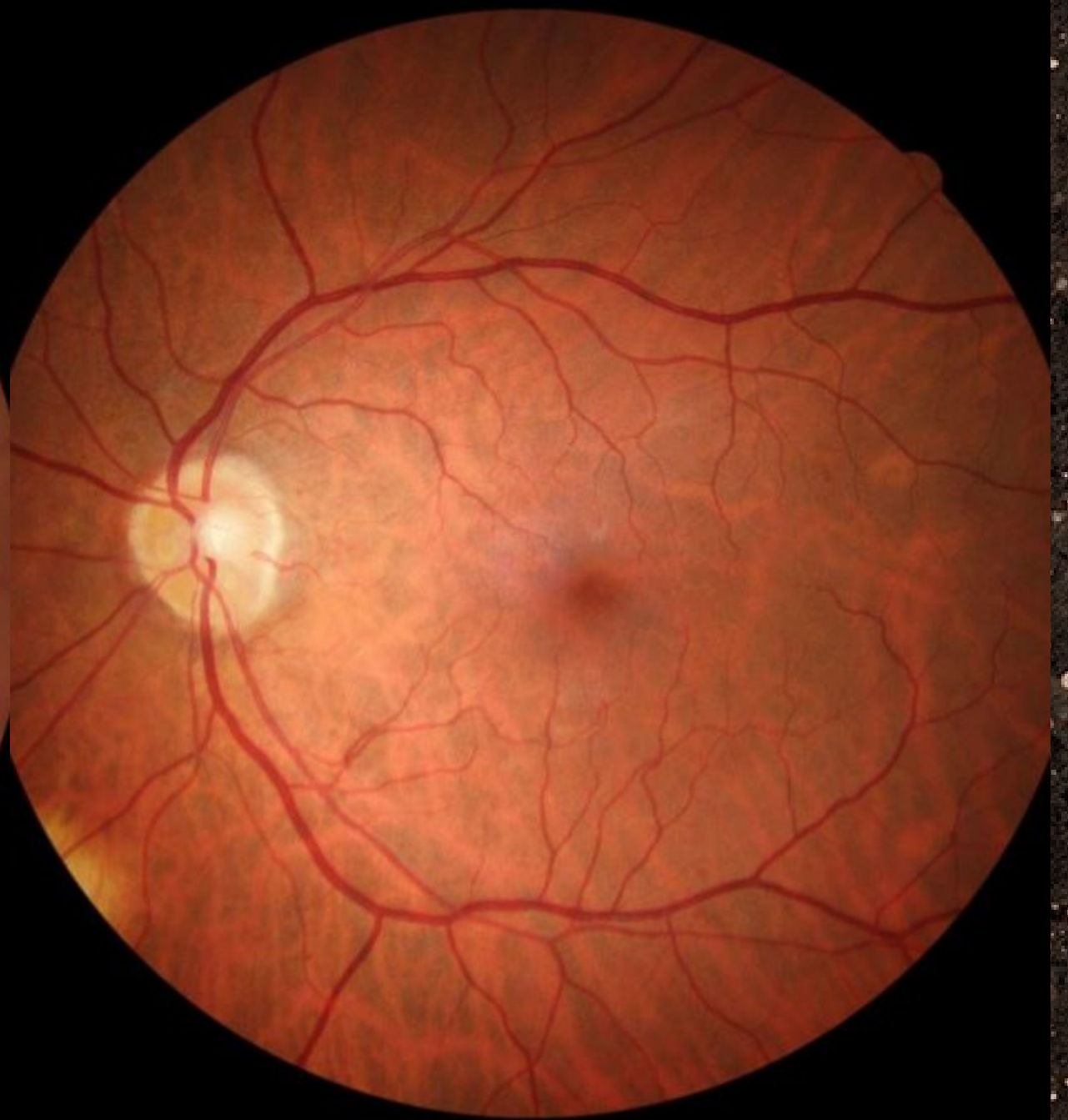
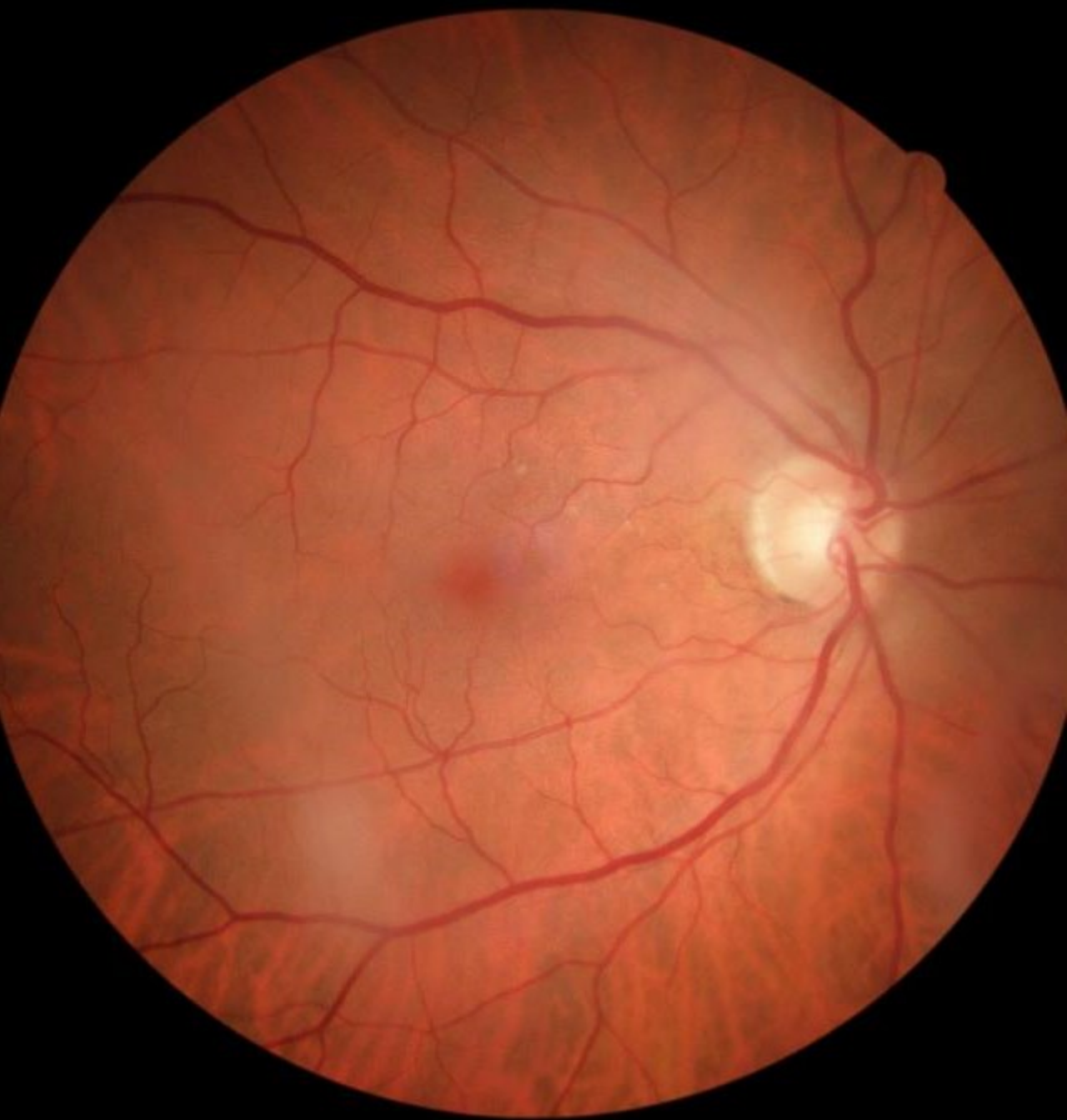
# CASE #3

*Bumps in the Night*

# CASE #3

---

- 66yo WM presents for routine exam
- POH: S/P localized RD OS (3yrs). S/P ECCE OU (5yrs)  
LEE: 3yr.
- MH: T2DM x 6yrs (HbA1c: 9.4), OSA, HTN
  
- Vision: 20/20 OD, 20/25 OS
- Ta: 14/22 @10:00AM; PERRL, No APD
- SLE: W&Q OU

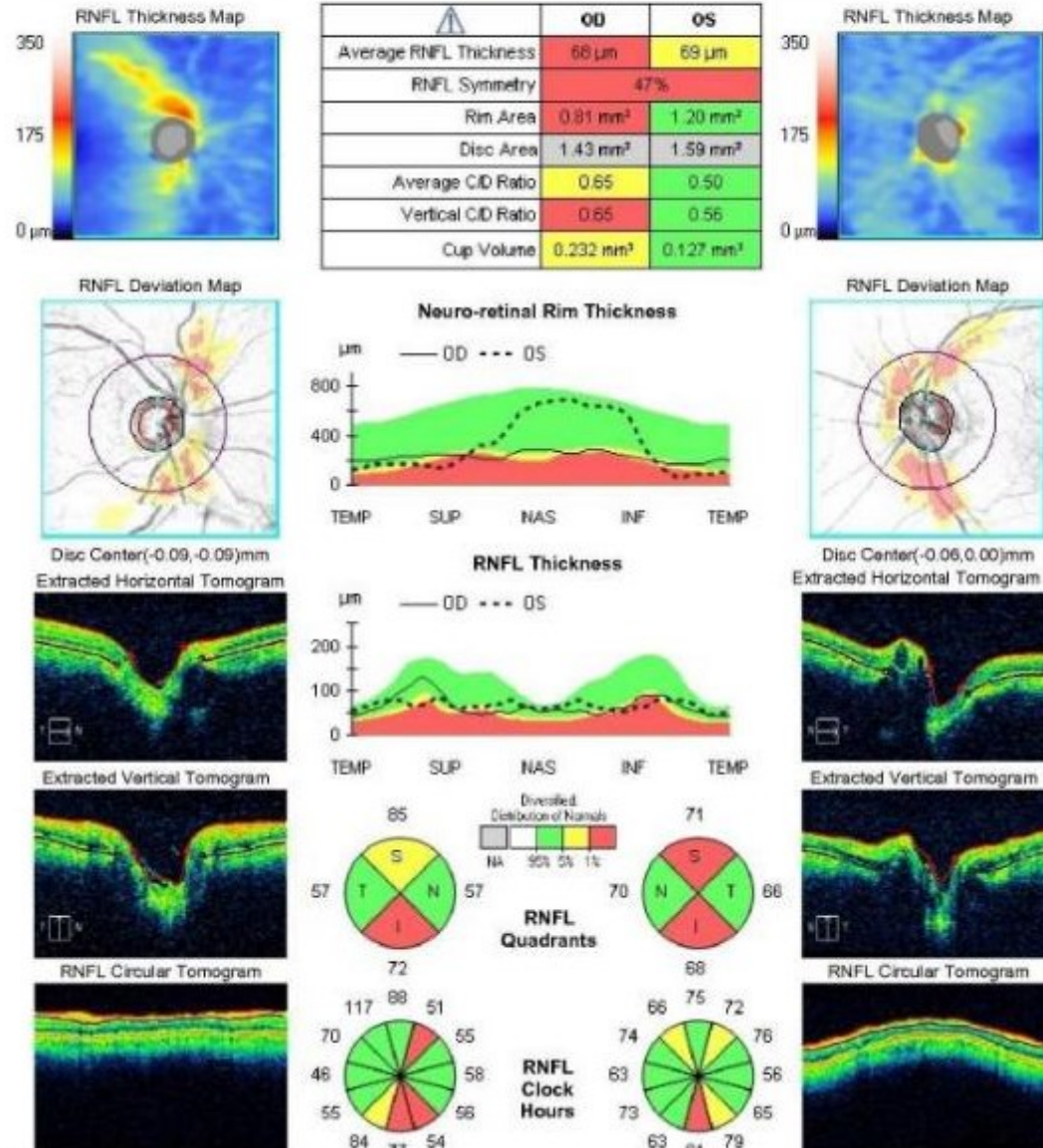




Name: OD OS  
 ID: Exam Date: 7/6/2018 7/6/2018  
 DOB: 2/18/1952 Exam Time: 11:32 AM 11:33 AM  
 Gender: Unknown Serial Number: 4000-6813 4000-6813  
 Technician: Operator, Cirrus Signal Strength: 10/10 10/10



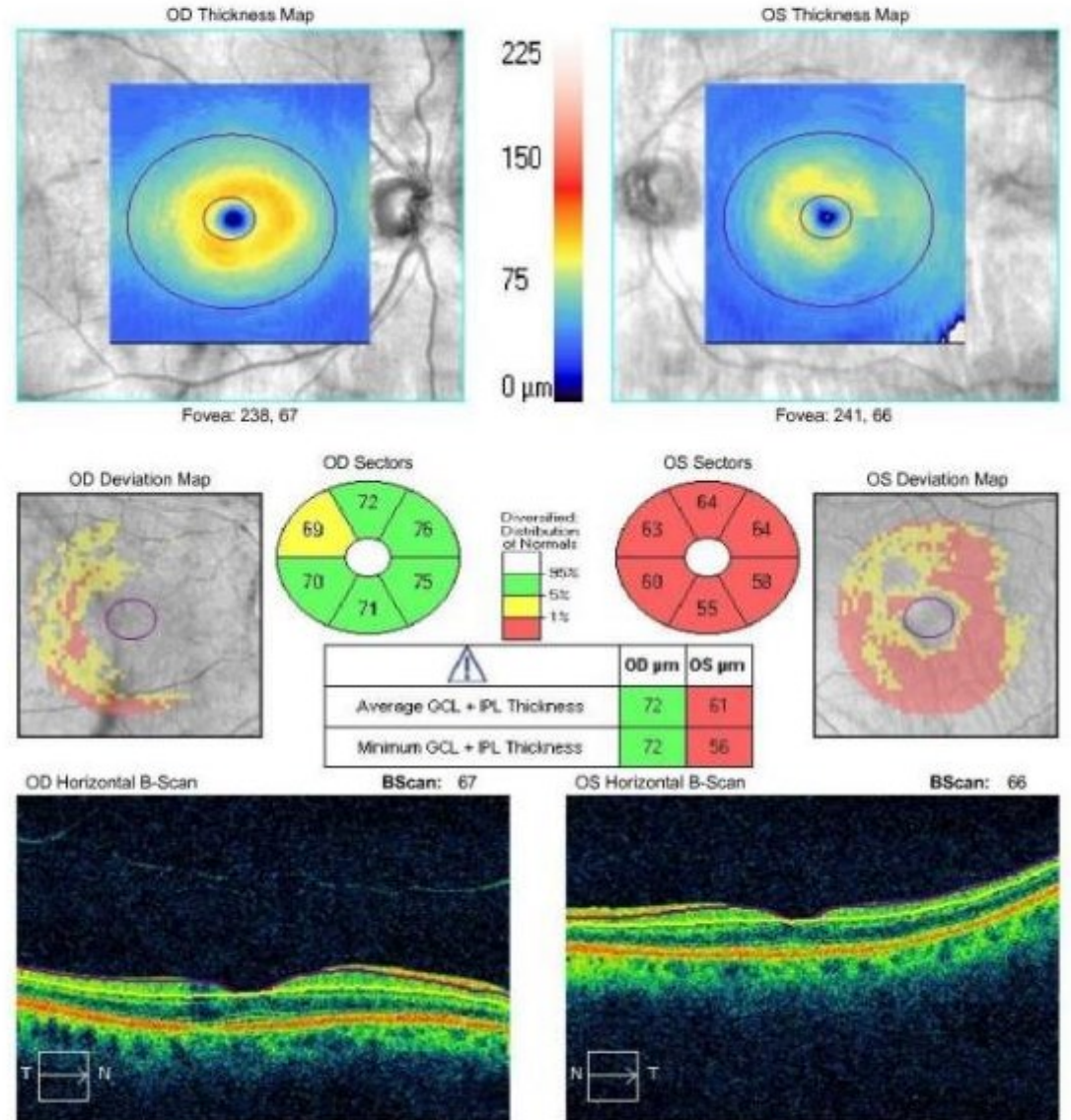
**ONH and RNFL OU Analysis: Optic Disc Cube 200x200** OD ● ● OS



Name: OD OS  
 ID: Exam Date: 4/25/2018 4/25/2018  
 DOB: 2/18/1952 Exam Time: 5:15 PM 5:15 PM  
 Gender: Unknown Serial Number: 4000-6813 4000-6813  
 Technician: Operator, Cirrus Signal Strength: 8/10 8/10



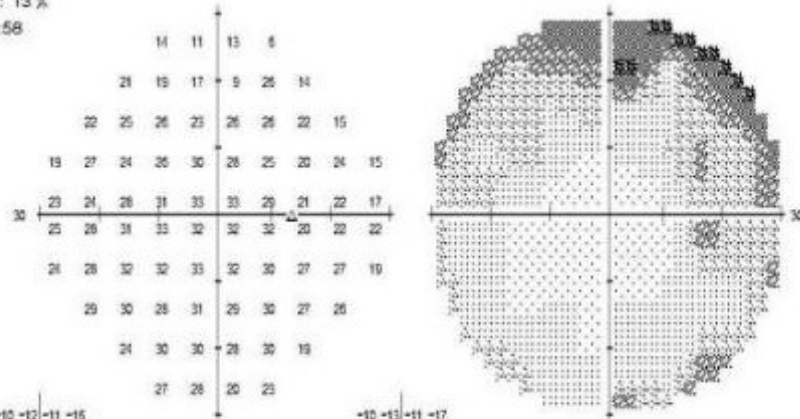
**Ganglion Cell OU Analysis: Macular Cube 512x128** OD ● ● OS



Central 30-2 Threshold Test

Fixation Monitor: Gaze/Blind Spot      Stimulus: III, White      Pupil Diameter:      Date: 07-06-2018  
 Fixation Target: Central      Background: 31.5 ASB      Visual Acuity:      Time: 9:21 AM  
 Fixation Losses: 4/19 xx      Strategy: SITA-Standard      RX: +3.00 DS      DC X      Age: 66  
 False POS Errors: 14 %  
 False NEG Errors: 13 %  
 Test Duration: 09:58

Fovea: OFF

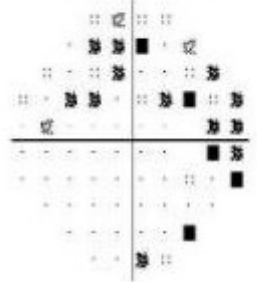
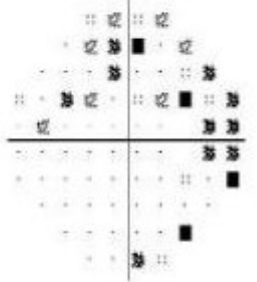


\*\*\* Low Test Reliability \*\*\*

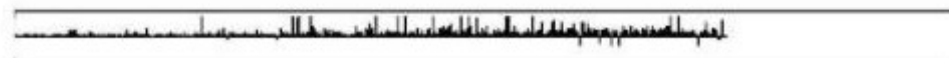
GHT  
 Outside Normal Limits  
 VFI 95%  
 MD -2.50 dB P < 5%  
 PSD 4.31 dB P < 0.5%

Total Deviation

Pattern Deviation



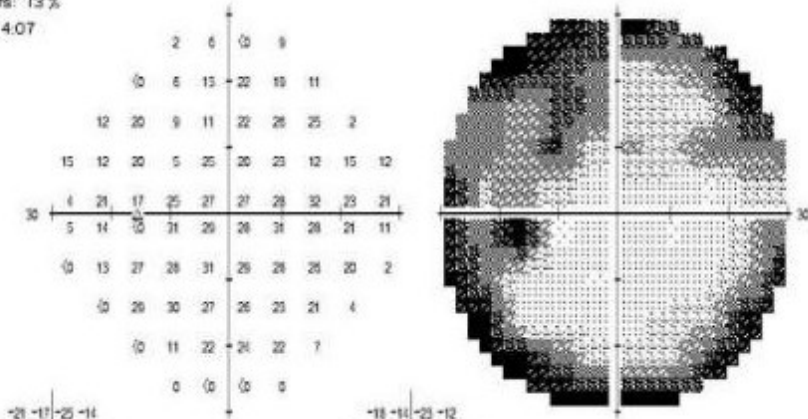
BOWDEN EYE CARE



Central 30-2 Threshold Test

Fixation Monitor: Gaze/Blind Spot      Stimulus: III, White      Pupil Diameter:      Date: 07-06-2018  
 Fixation Target: Central      Background: 31.5 ASB      Visual Acuity:      Time: 9:34 AM  
 Fixation Losses: 1/23      Strategy: SITA-Standard      RX: +1.50 DS      DC X      Age: 66  
 False POS Errors: 14 %  
 False NEG Errors: 13 %  
 Test Duration: 14:07

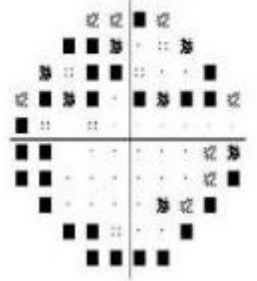
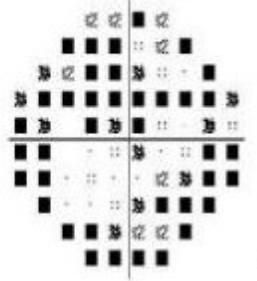
Fovea: OFF



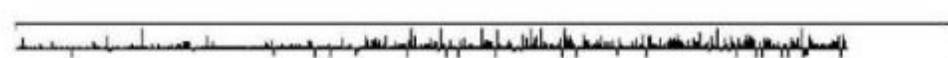
GHT  
 Outside Normal Limits  
 VFI 85%  
 MD -9.80 dB P < 0.5%  
 PSD 10.15 dB P < 0.5%

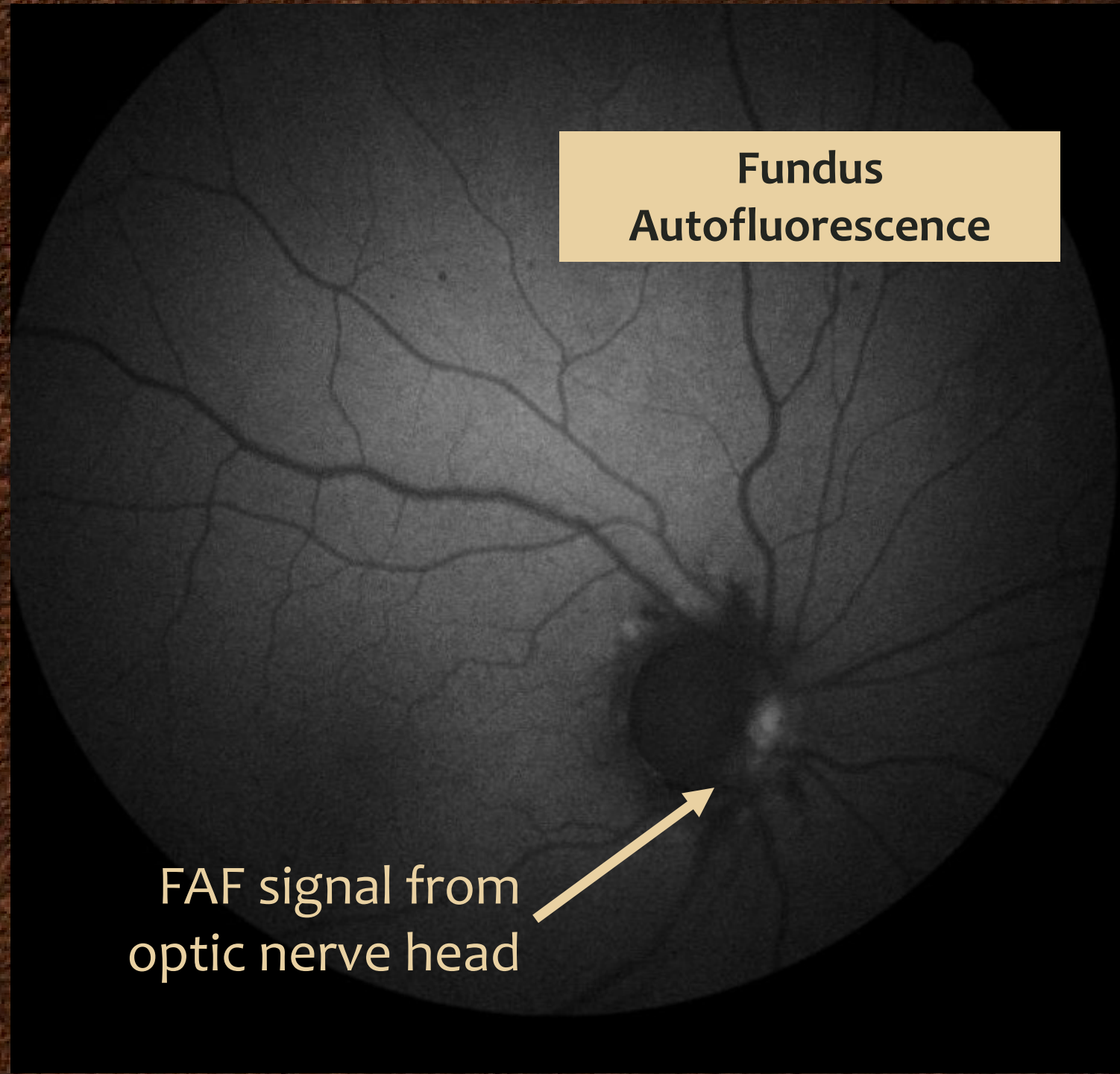
Total Deviation

Pattern Deviation



BOWDEN EYE CARE

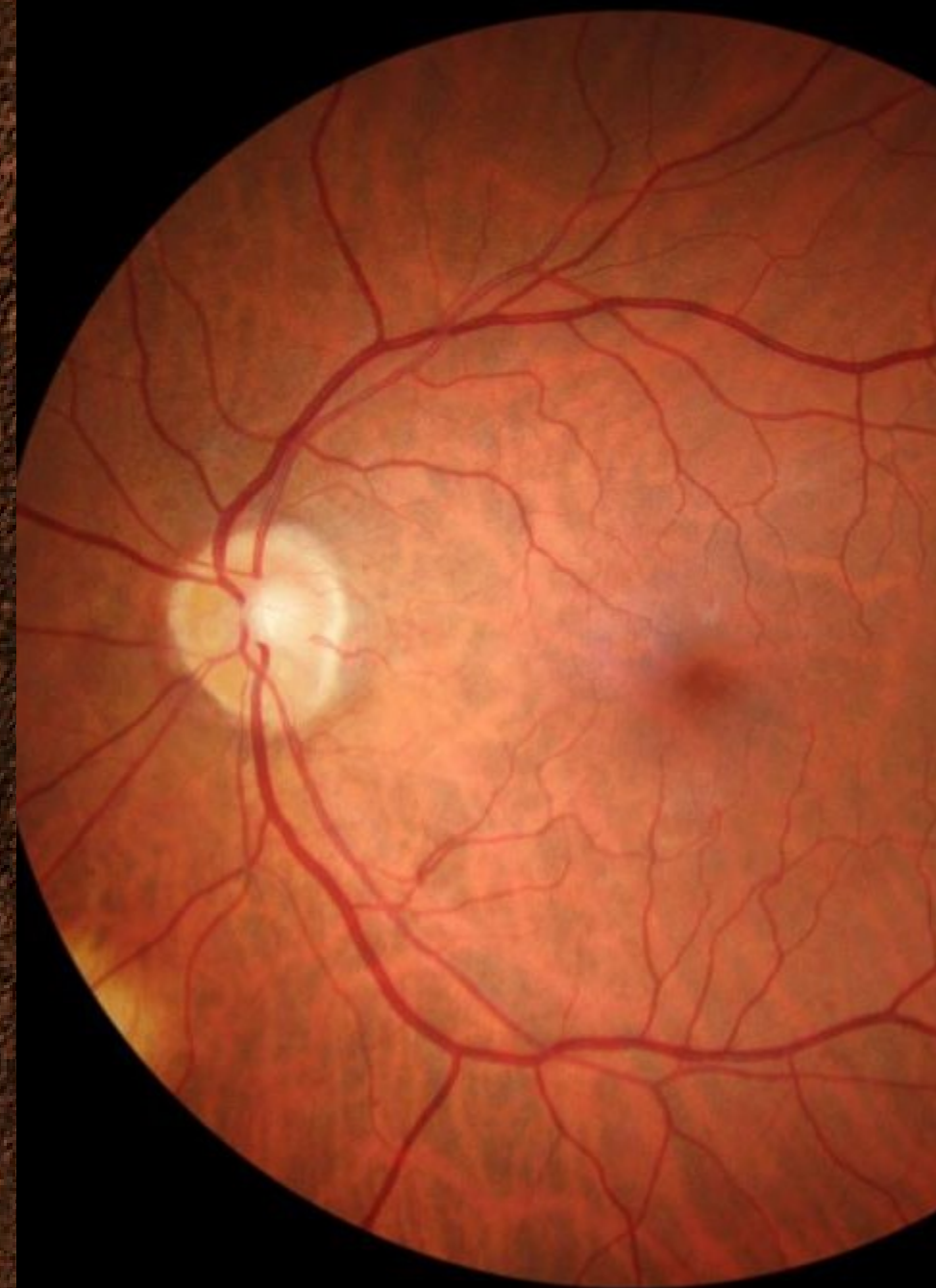




Fundus  
Autofluorescence

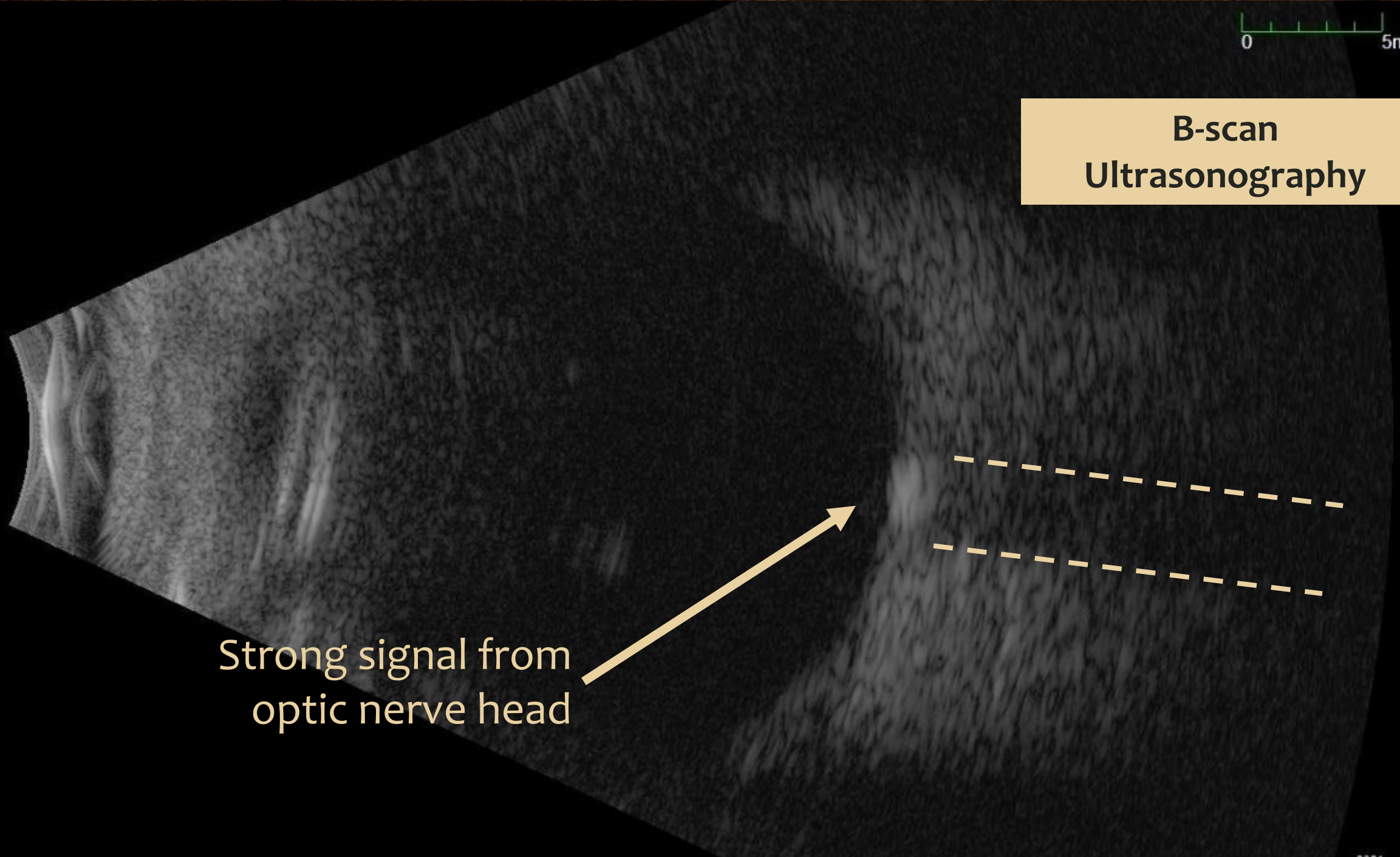
FAF signal from  
optic nerve head





0 5mm

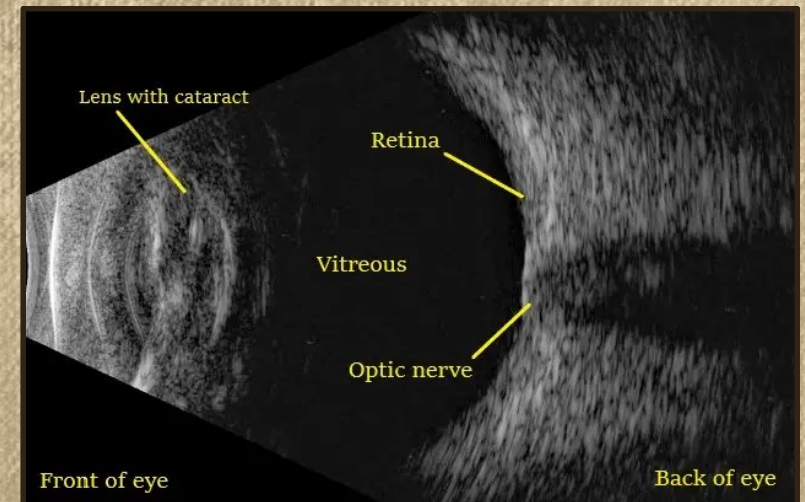
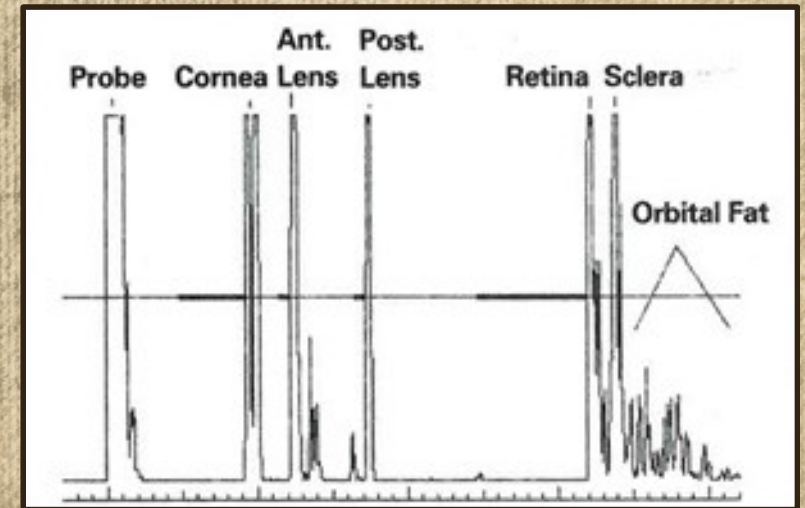
**B-scan  
Ultrasonography**



Strong signal from  
optic nerve head

# About B-scan Ultrasonography

- High frequency sound is passed through the tissue and echos are recorded
- A-scans: 1-dimensional data
- B-scans: 2-dimensional images
- Why use it?
  - Ability to penetrate opaque tissue



# About B-scan Ultrasonography

Kinetic exam: Eye movement utilized in evaluating the patient's condition

- Retina: Tethered membrane
- Vitreous: Clothes dryer tumbling



# What is going on here?

<https://app.tophat.com/e/777538>



Glaucoma

Papilledema

Foster-Kennedy Syndrome

Optic Nerve Head Drusen

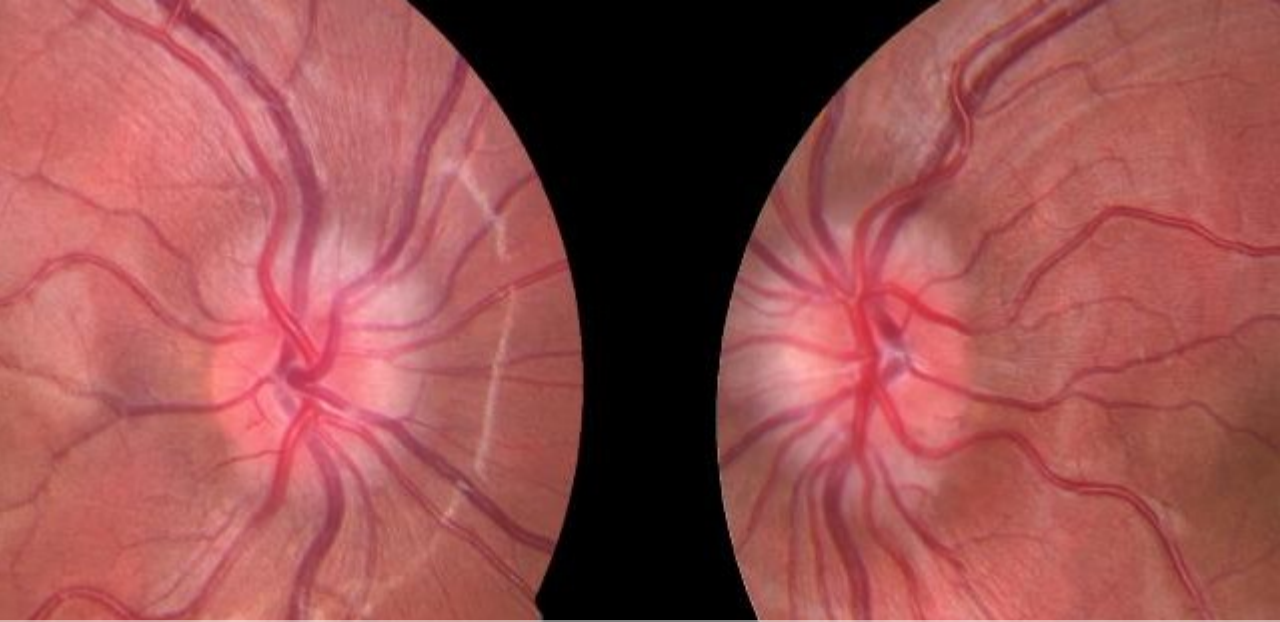
Ischemic Optic Neuropathy



<b>Glaucoma</b>	Abnormal <b>IOP asymmetry</b> (14/22). Large optic cups with <b>rim area</b> < 1 mm <sup>2</sup> . Characteristic <b>VF defects</b>
<b>Papilledema</b>	Bilateral <b>disc edema</b> secondary to increased intracranial pressure.
<b>Foster-Kennedy Syndrome</b>	<b>Unilateral papilledema</b> with optic atrophy in fellow eye. Associated with sphenoidal ridge meningiomas.
<b>Optic Nerve Head Drusen</b>	<b>Calcific deposits</b> in the prelaminar optic nerve head. Causes scalloping and obscuration of the disc margin without edema. Will <b>hyperfluoresce</b> on FAF
<b>Ischemic Optic Neuropathy</b>	Acute onset <b>pallor and edema</b> of ONH associated with APD, color vision loss, and nerve fiber bundle VF defects. <b>Disc-at-risk</b> . Systemic risk factors: <b>DM, OSA</b>

# Optic Nerve Head Drusen

- Acellular calcific deposits of the ONH; 66%-80% bilateral
- Idiopathic; Familial clustering suggests genetic factors
- Early childhood onset of lesions deep in ONH (“buried drusen”)
- Lesions usually increase in visibility and size over time
- 25%-75% of adults have VF defect (enlarged BS, arcuate)
- Visual acuity rarely affected



Buried drusen (10yo child)



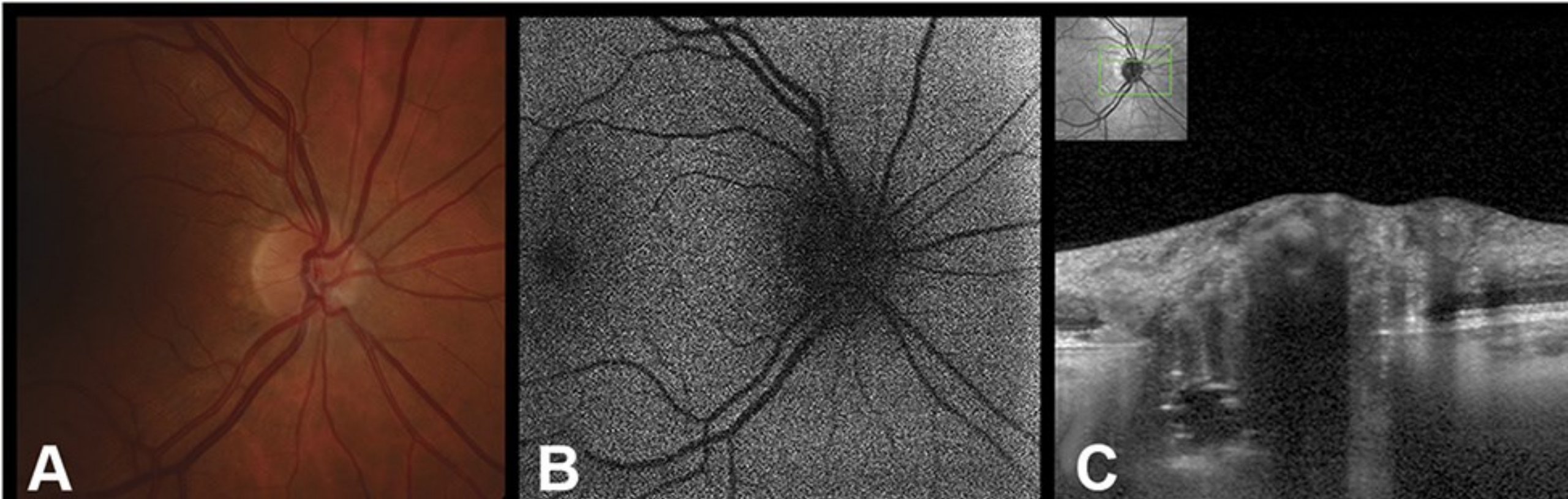
Partially exposed drusen (12yo child)



Superficial ONH drusen

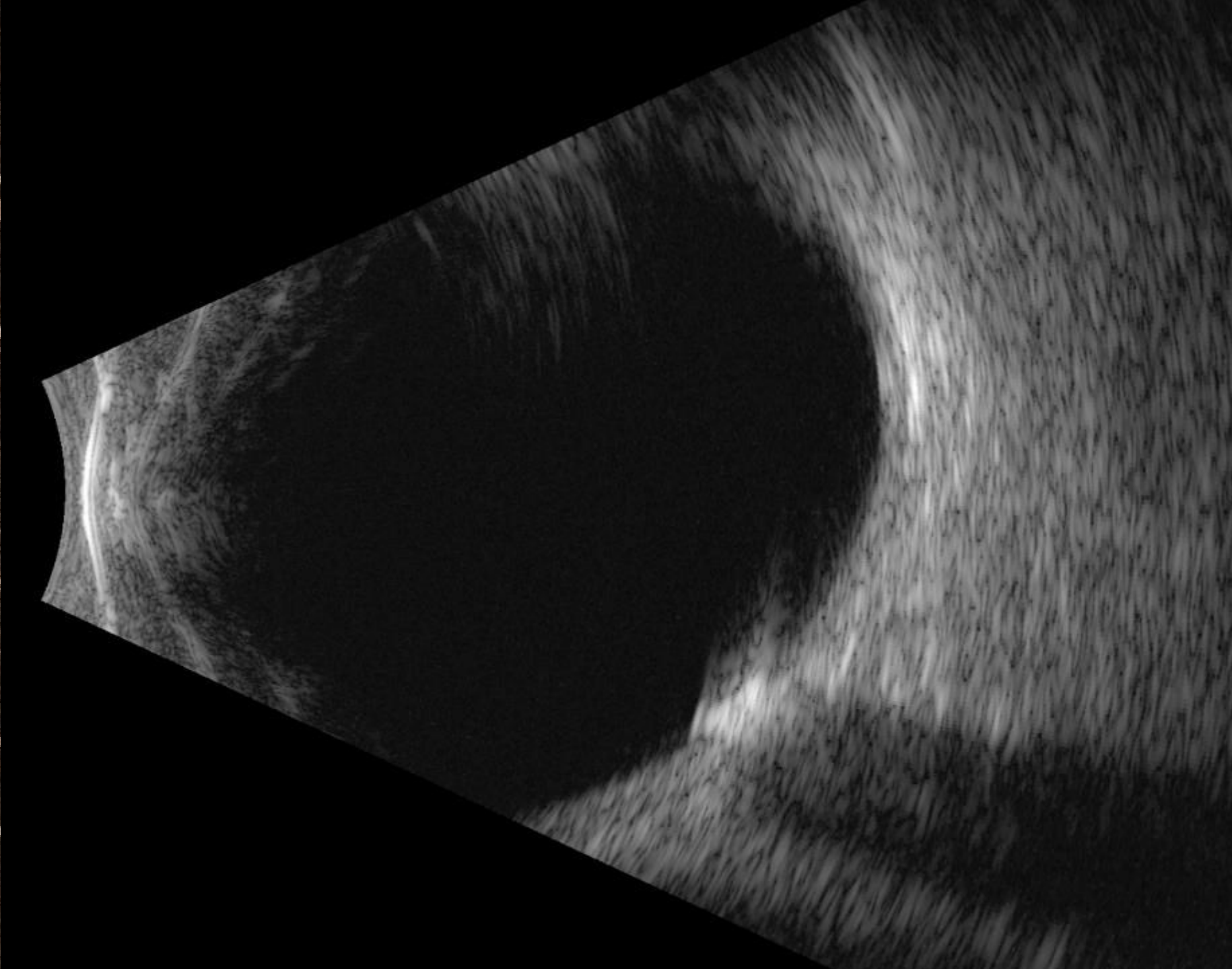


Superficial ONH drusen



**Absence of FAF signal in a pt with deep buried ONH drusen**  
**FAF is more likely to detect ONH drusen that are**  
***larger and more superficial***

PMID: 29649006



Ultrasonography has high sensitivity (90%) and specificity (80%) in diagnosis of ONH drusen

**BUT...**

The “gold standard” for diagnosing disc drusen is:

**Enhanced Depth Imaging OCT**

- ODD are **always** located above lamina cribrosa
- ODD **always** have a signal-poor core
- ODD are **often** seen with a hyperreflective margin, most prominent superiorly
- ODD are **sometimes** seen as conglomerates of smaller ODD with internal reflectivity within the signal-poor core
- Hyperreflective horizontal lines **might** represent early ODD but should **not** be diagnosed as ODD
- Peripapillary hyperreflective ovoid mass-like structures (PHOMS) should **not** be diagnosed as ODD

## ODD on EDI-OCT

ODD are “hollow” structures with a hyperreflective margin (“capsule”)

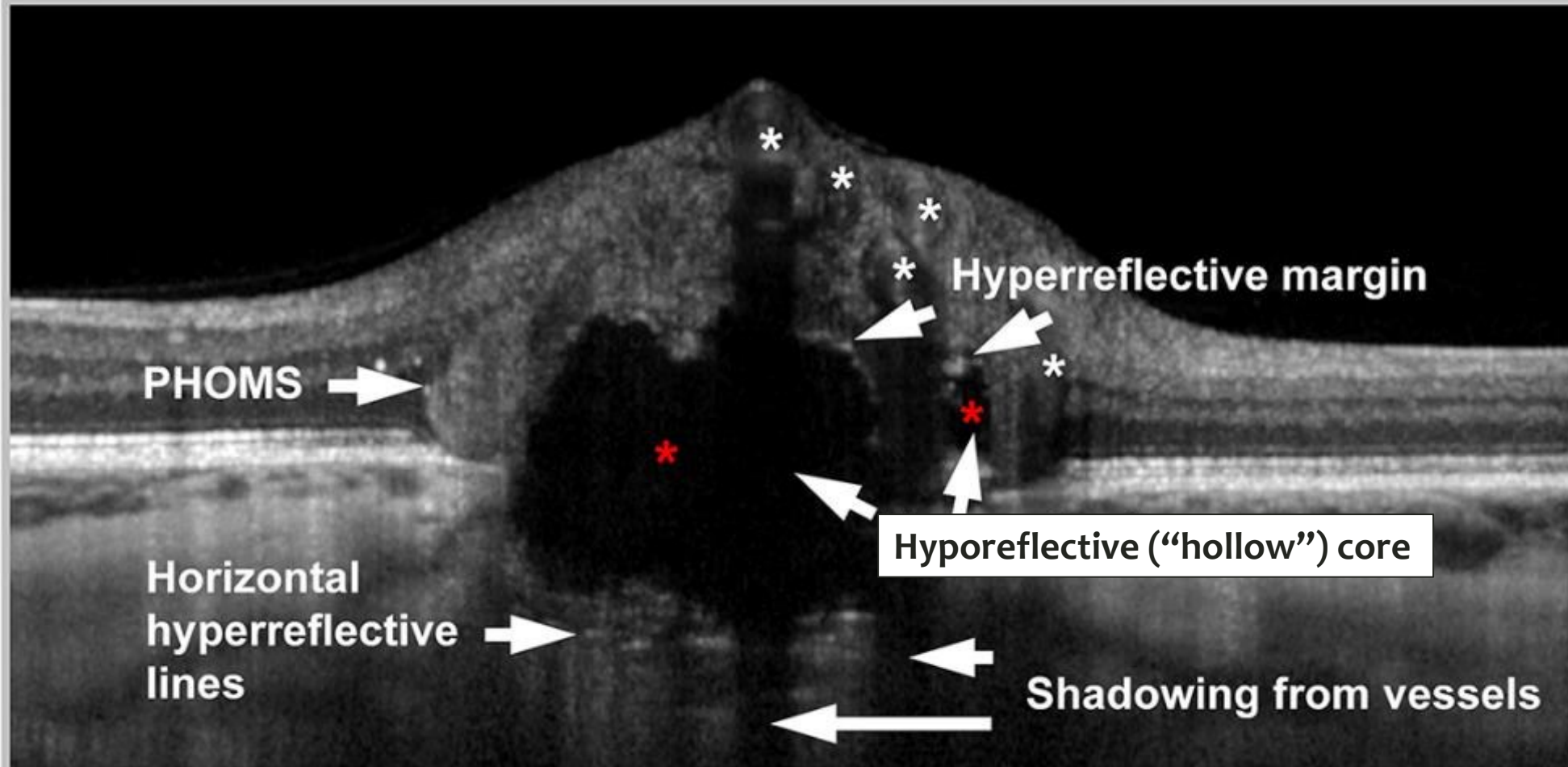
Hyperreflective horizontal lines might be early ODD

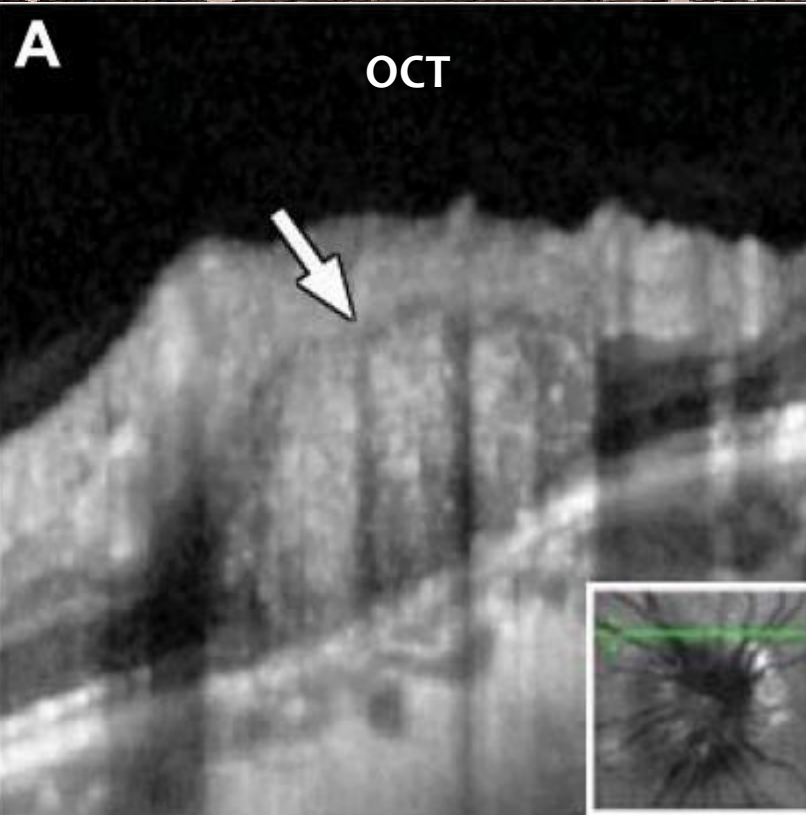
PHOMS circumscribe the disc and are not ODD.

**White asterisks:** vessels.  
**Red asterisks:** ODD.

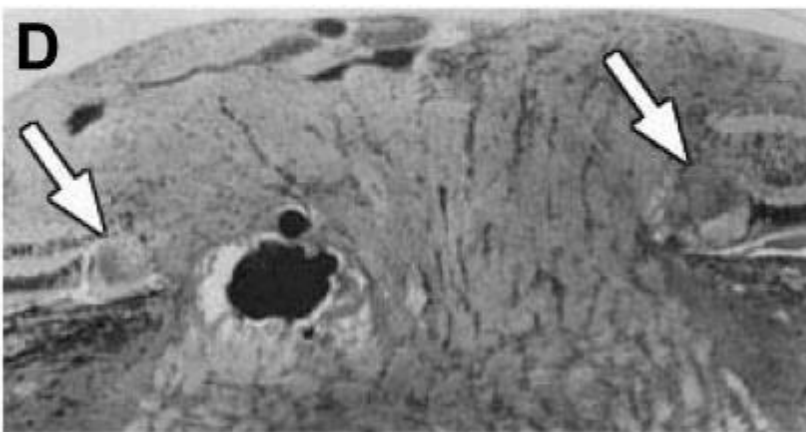
**ODD:** optic disc drusen  
**PHOMS:** peripapillary hyperreflective ovoid mass-like structures.

Malmqvist, 2017

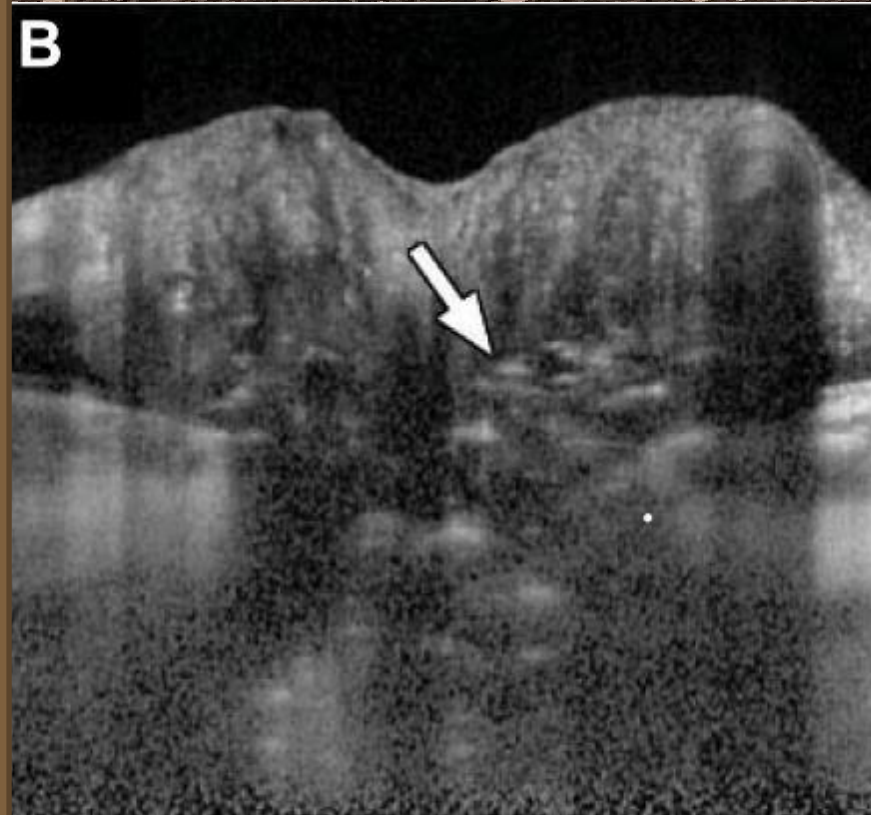




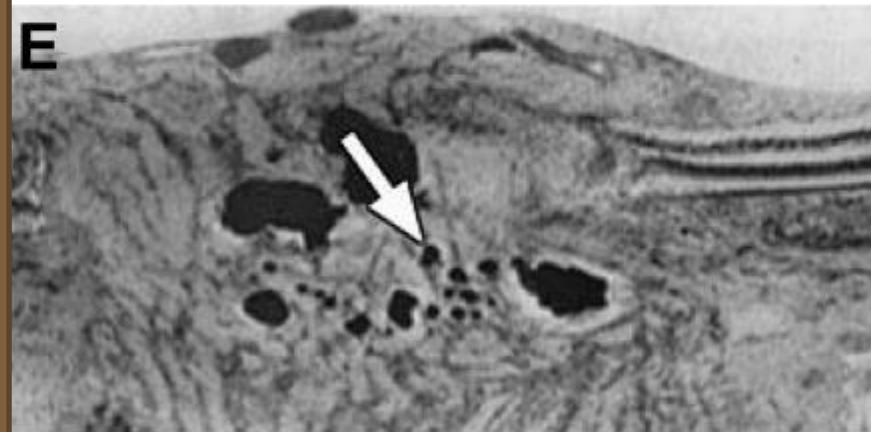
HISTOLOGY



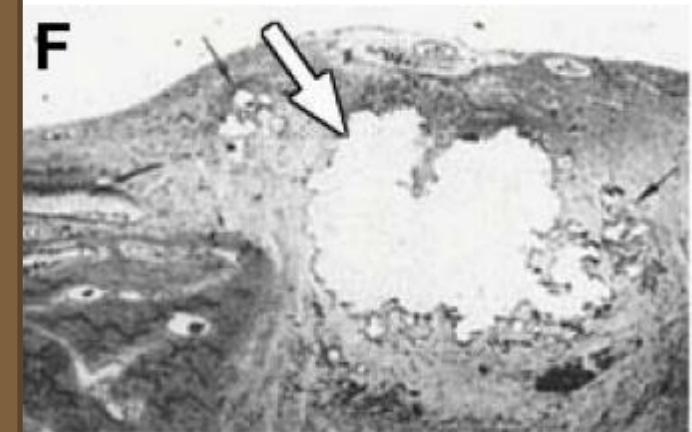
Peripapillary waste (not hollow)



PMID: 27817914



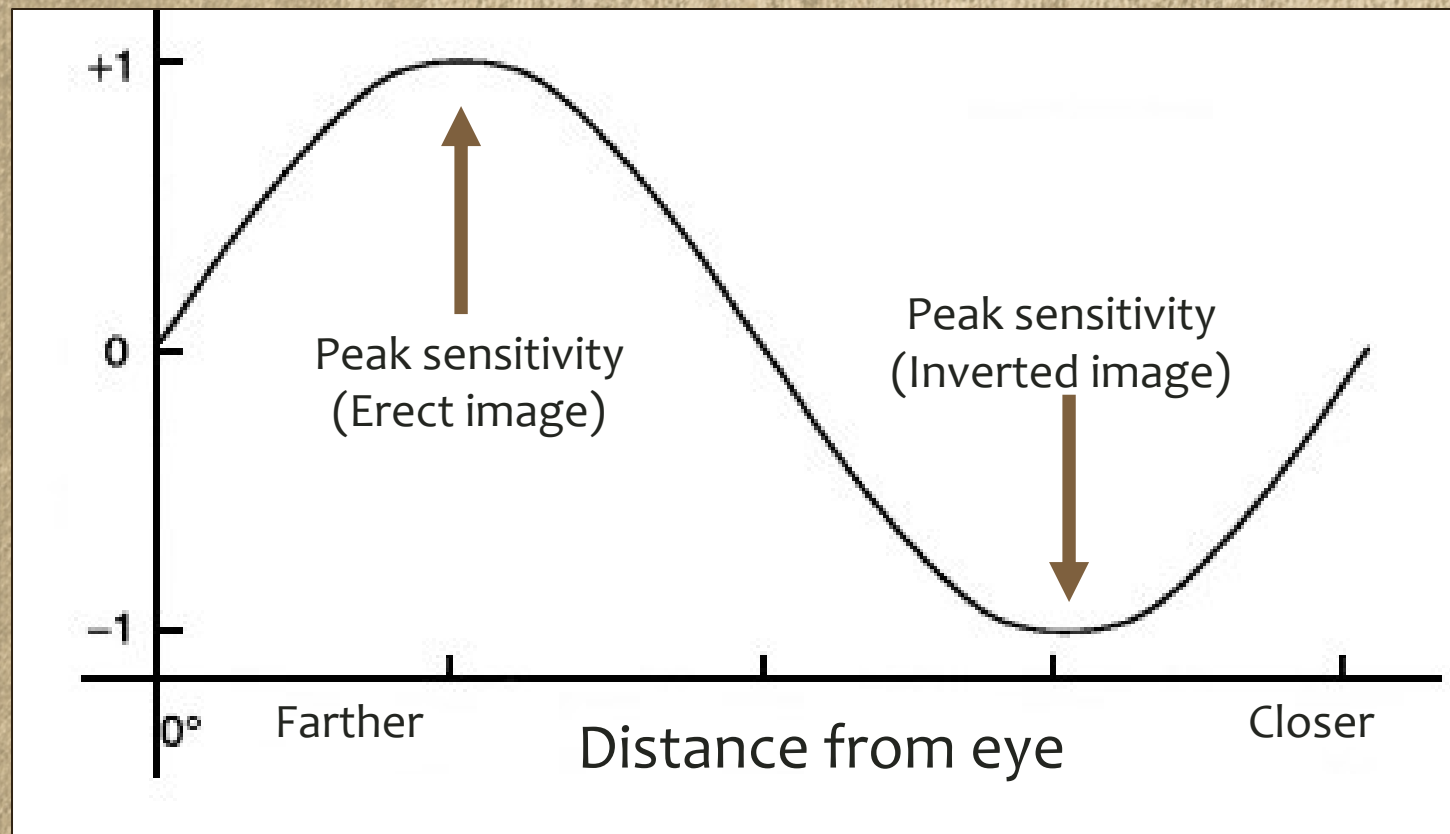
Small drusen (horizontal lines)



Confluent drusen (septa)

# About Enhanced Depth Imaging OCT

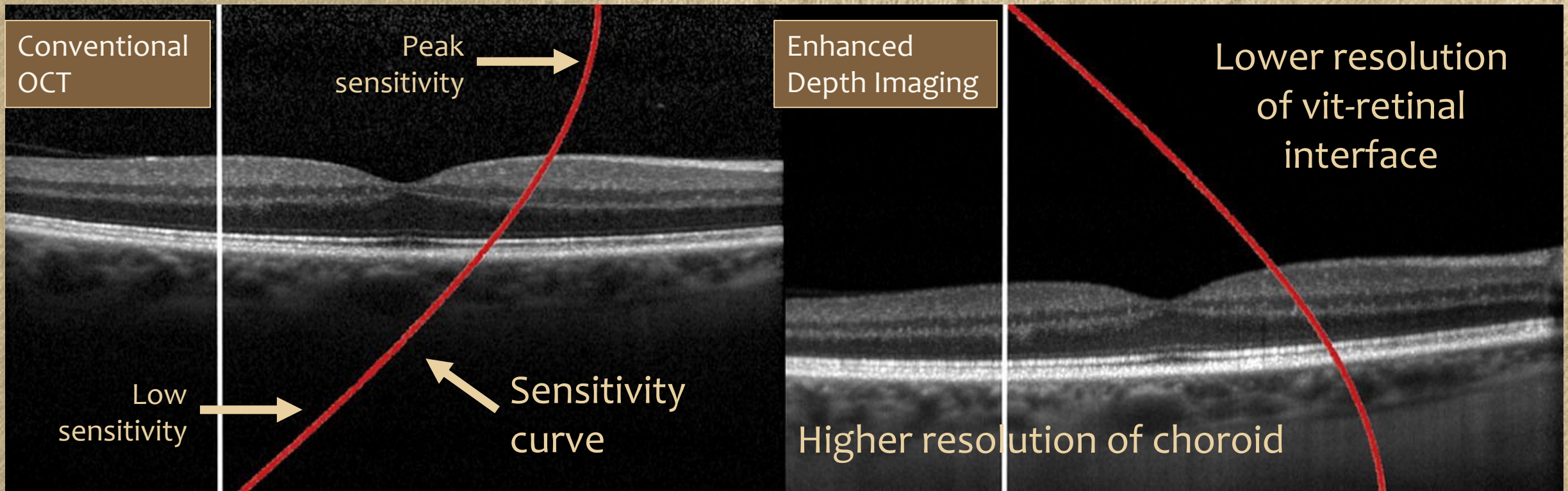
## OCT Sensitivity Curve





# About Enhanced Depth Imaging OCT

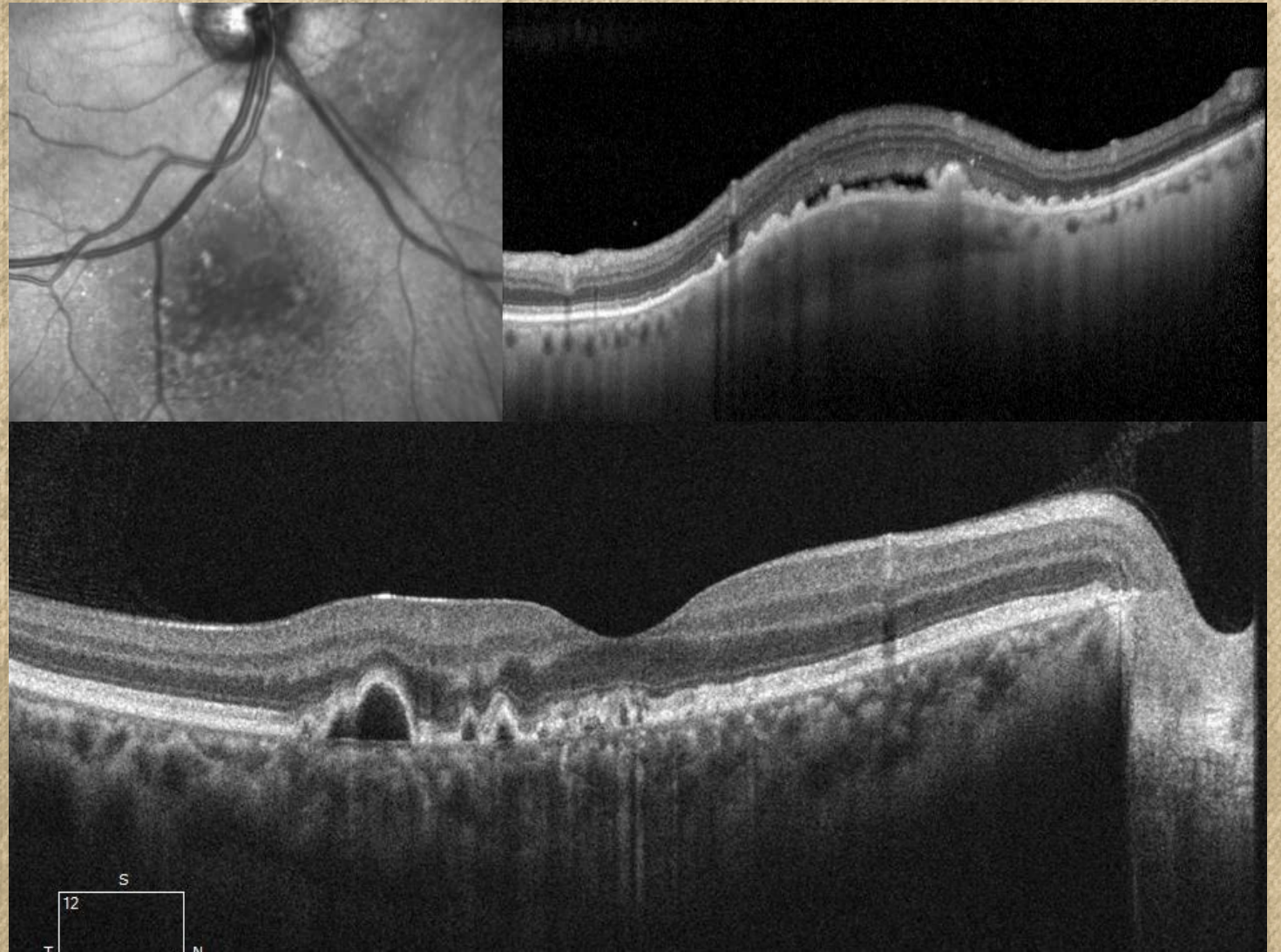
The OCT sensitivity curve is shifted deeper in the eye to more clearly image deeper structures at the cost of lower resolution of inner structures



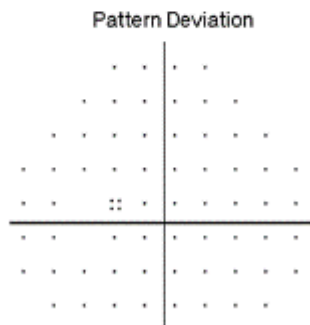
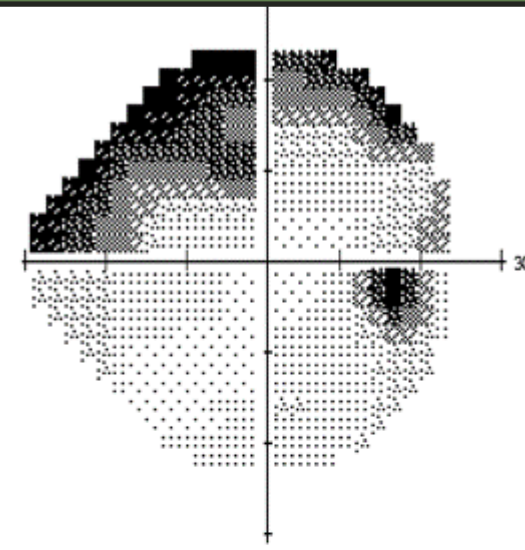
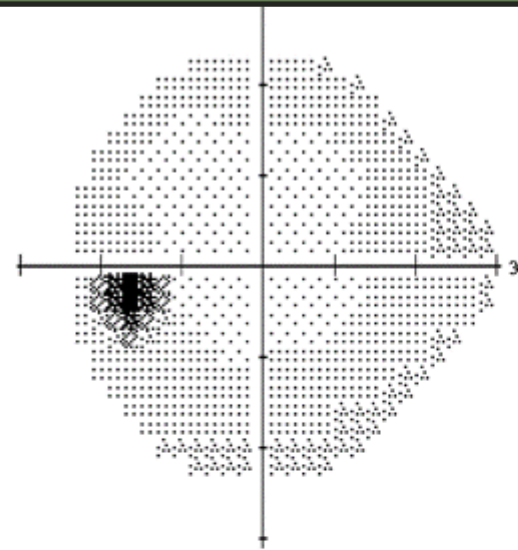
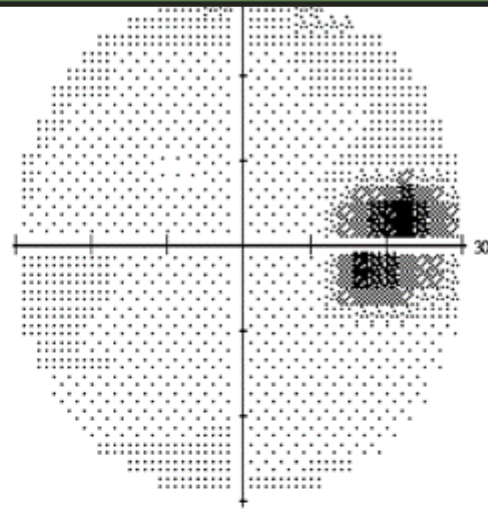
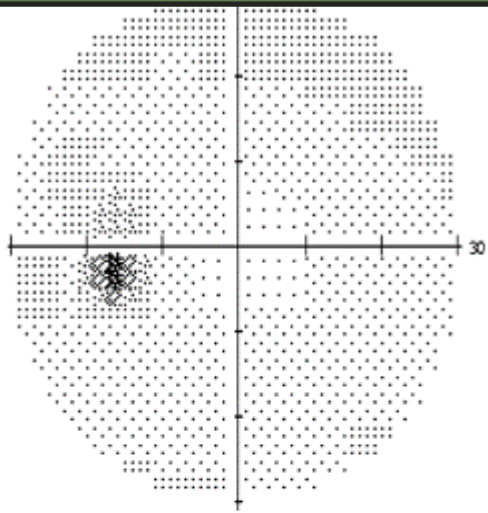
# About Enhanced Depth Imaging OCT

Why use it?

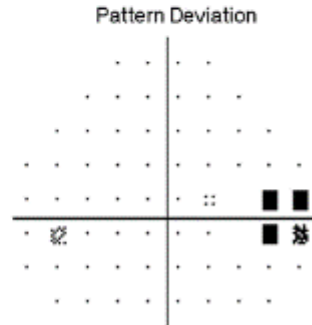
- Choroidal nevus
- Disc drusen
- Pachychoroid (Central serous)



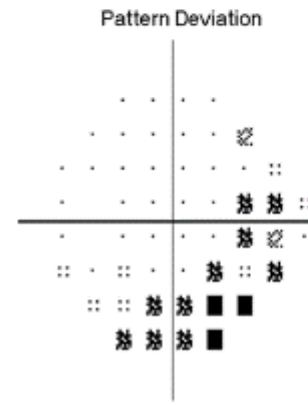
# VF Defects with ONH Drusen



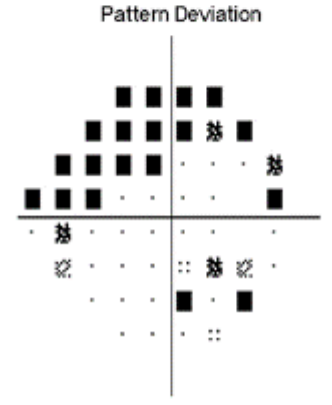
20% cases



50% cases



30% cases



Normal

Enlarged Blind Spot

Early Arcuate

Adv. Arcuate

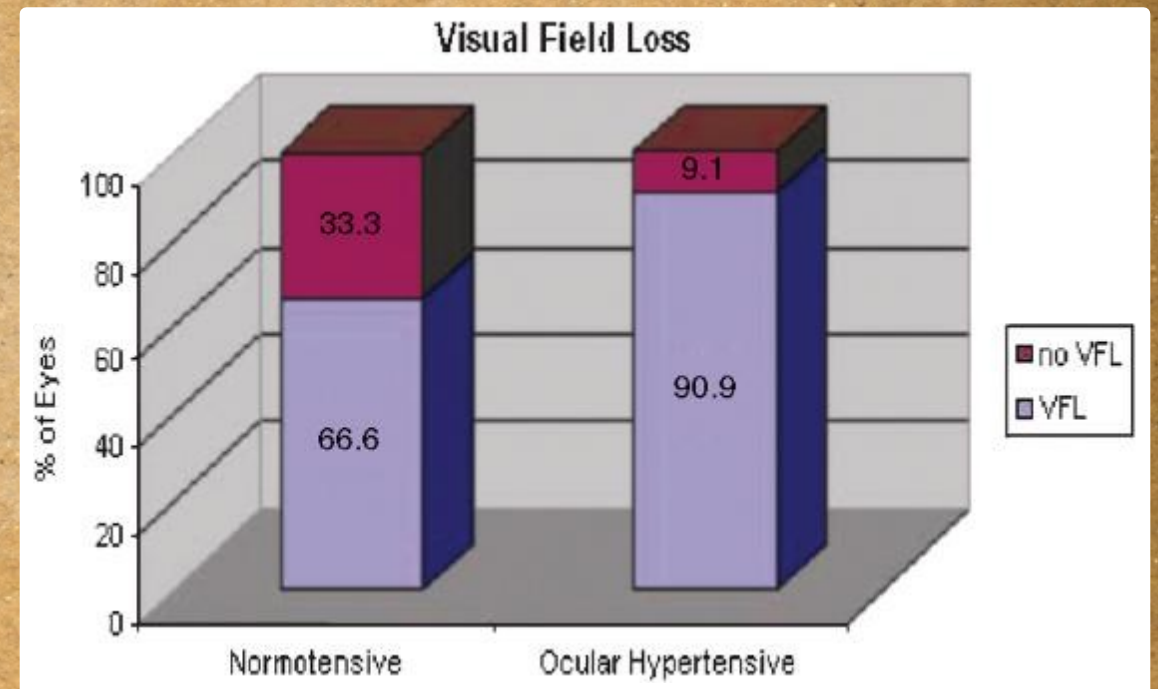
# VF Defects with ONH Drusen

- Over 80% of adult cases will have VF defects
  - Most common: Enlarged BS, arcuate defects, constricted VF
- Greater damage with larger and more superficial drusen
- VF loss tends to progress slowly over time
- Arcuate VF defects have associated RNFL thinning
- Visual acuity usually remains normal
- No effective treatment for ONH drusen

# ONH Drusen & Glaucoma

- Challenge of managing glaucoma in patients with concurrent ONH drusen
- Eyes with ONH drusen are more susceptible to glaucomatous VF loss
- IOP-lowering treatment should be considered in all patients with ONH drusen and elevated IOP

PMID: 18344754



# Take Home Message

## Glaucoma with ONH Drusen

- Perform OCT and FAF to confirm presence of ONH drusen
- Obtain ultrasound in difficult cases
- VF loss is common and can mimic glaucomatous loss
- Patients with ONH drusen and elevated IOP are more susceptible to VF loss and may benefit from IOP lowering therapy



# CASE #4

*The birdman cometh*

# Case #4

---

- 56yo WM presents for routine eye exam
- Occupation: Maintenance man and farmer
- POH: H/O vision loss OS due to “bleeding” 5 years ago. Treated with laser
- MH: NIDDM x 4yrs (HbA1c: 6.5)
- BVA: 20/20 OD, FC @ 10ft OS
- Pupils and motility: Normal
- IOP: 14/16 mmHg @ 2pm
- External: Normal OU



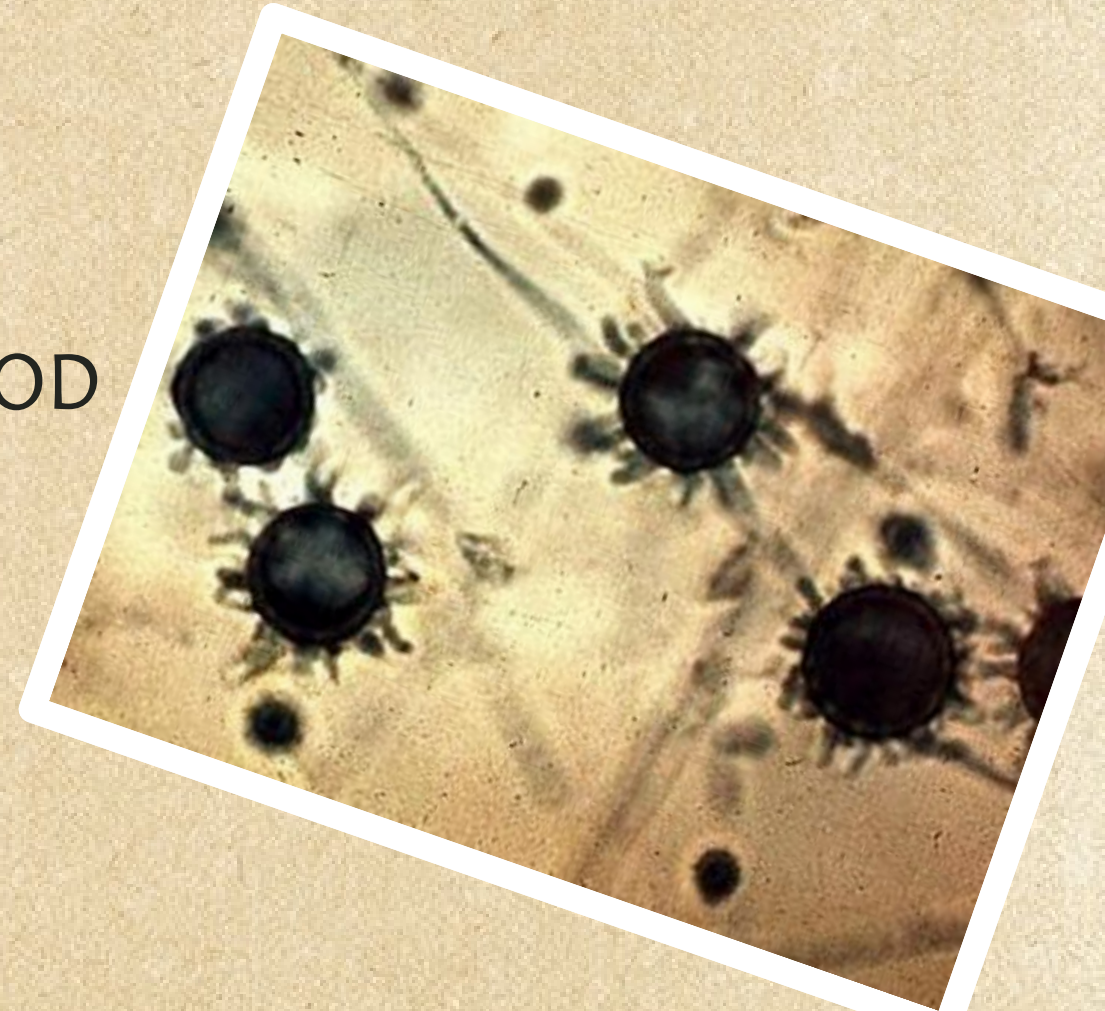
No subretinal fluid or blood, OU

20/20

Finger counting

# Assessment

- POHS OU – currently inactive
- Histoplasmic maculopathy OS with severe vision loss
- Evidence of prior reactivation OD
- No diabetic eye disease



# What is the plan?

<https://app.tophat.com/e/777538>



Referral for laser photocoagulation

Referral for Avastin injection

Mask or ventilator use at work

Referral for low vision care

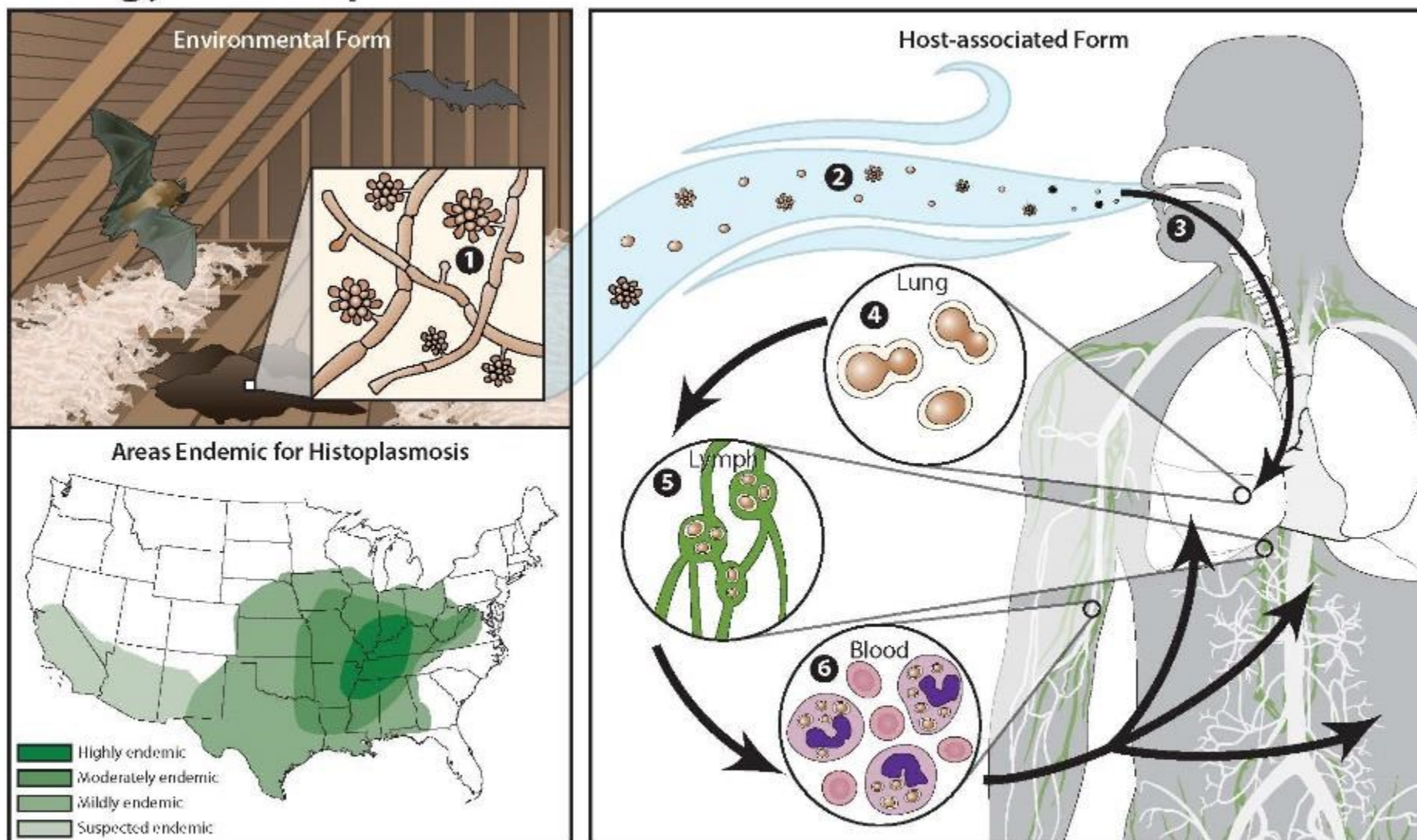
Routine annual eye exams

# Management

---

- Recommend use of protective devices because pt is frequently exposed to soil and bird droppings as a farmer and maintenance man.
- Daily Amsler grid and environmental Amsler
- Safety issues for monocular patients
- Patient education diabetic eye disease.
- RTC 6 months, sooner PRN

# Biology of Histoplasmosis

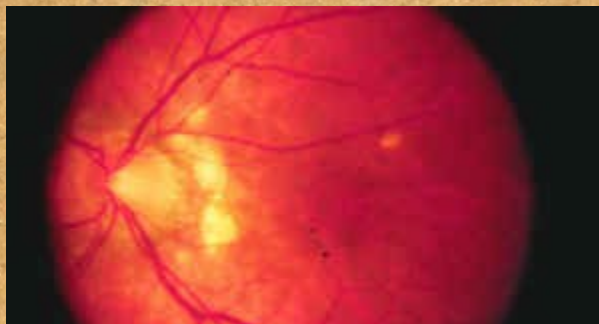


In the environment, *Histoplasma capsulatum* exists as a mold (1) with aerial hyphae. The hyphae produce macroconidia and microconidia (2) spores that are aerosolized and dispersed. Microconidia are inhaled into the lungs by a susceptible host (3). The warmer temperature inside the host signals a transformation to an oval, budding yeast (4). The yeasts are phagocytized by immune cells and transported to regional lymph nodes (5). From there they travel in the blood to other parts of the body (6).

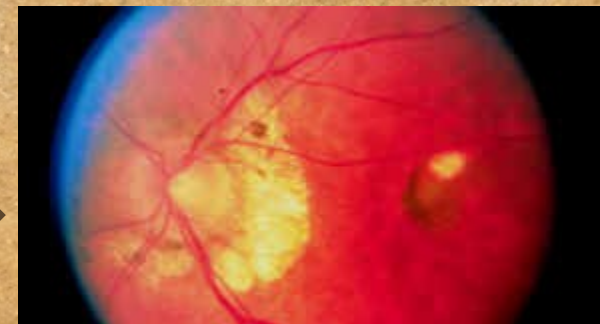


# POHS and Vision Loss

- Vision loss occurs secondary to exudative maculopathy caused by CNV &/or inflammation
- Reactivation of histo spots in the macular region is believed to play a role in triggering maculopathy
- Patients with perimacular histo spots, especially near the fovea, are at risk for vision loss



Perimacular histo spot



Active maculopathy

Inflammatory maculopathy pathway

Focal choroiditis



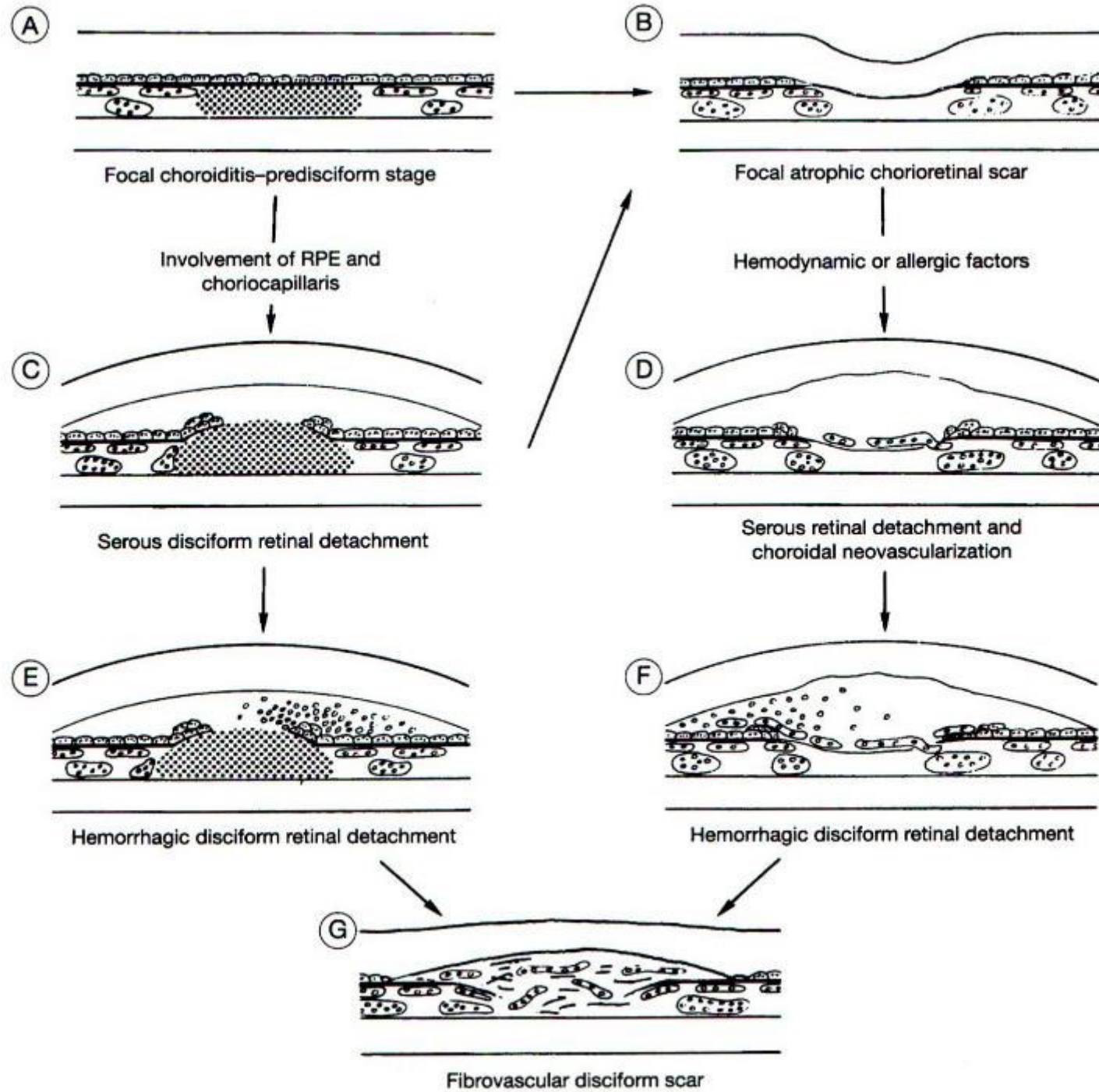
Serous detachment



Subretinal hemorrhage



Scar



Neovascular maculopathy pathway

Histo spot



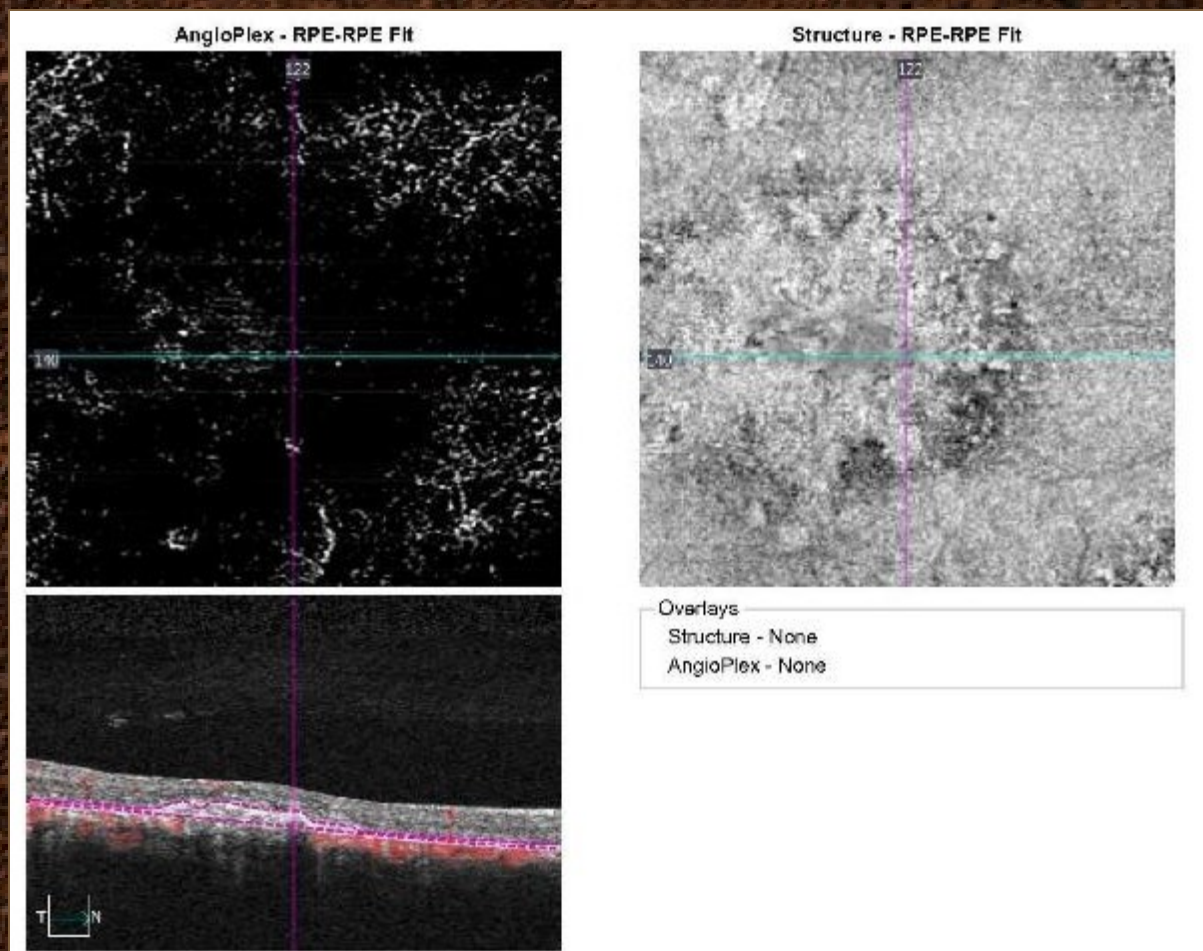
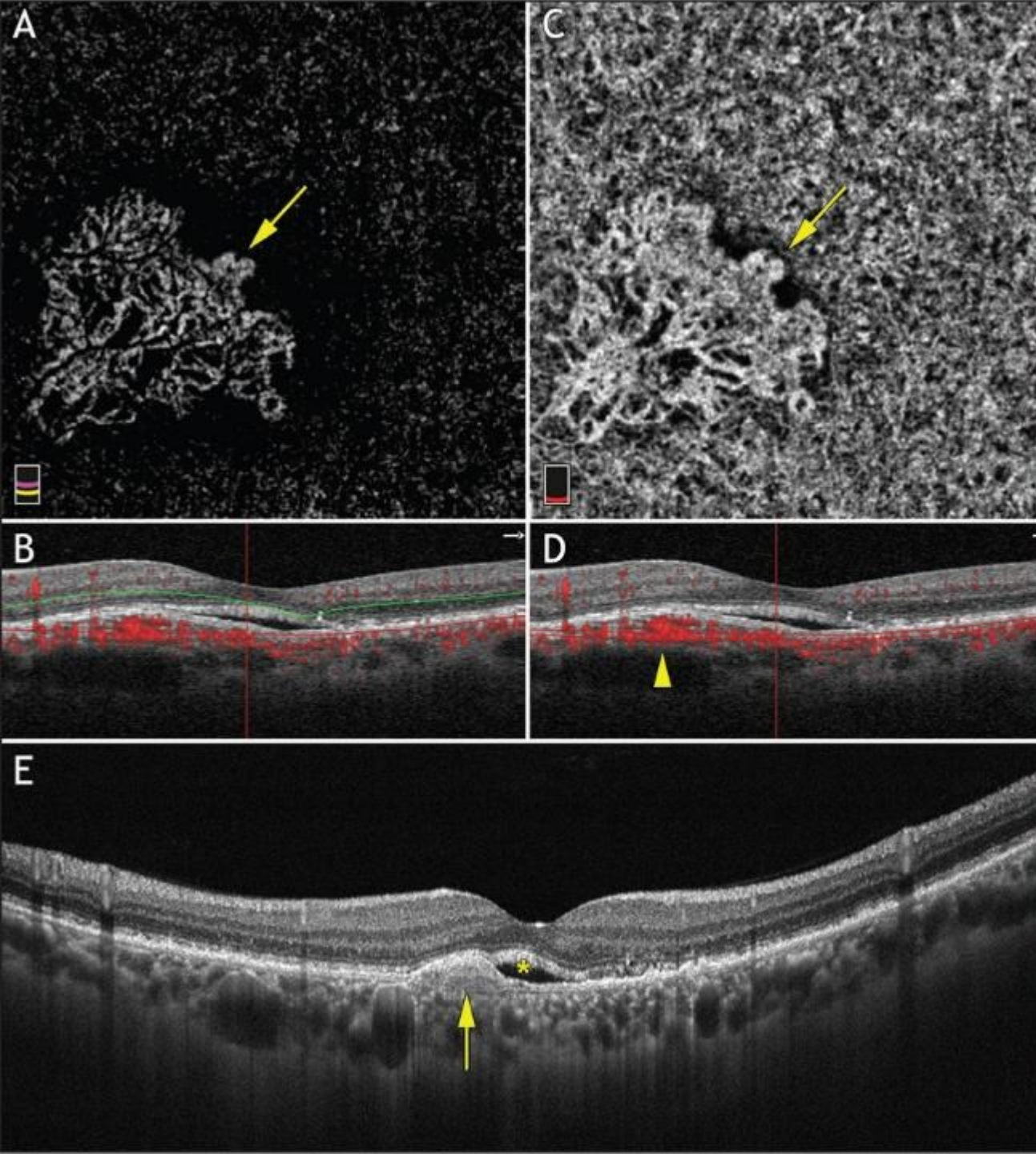
CNV



Subretinal hemorrhage



Scar



OCT angiography can differentiate neovascular from inflammatory maculopathy in patients with POHS



# About OCT Angiography

- Within milliseconds, multiple sequential OCT scans are obtained and analyzed for change
  - All change is assumed to represent blood flow
- All the benefits of conventional OCT plus blood flow data
  - 3D volumetric data, high resolution, quantitative analysis
- Key disadvantages: Slow, leakage not visualized, artifacts
- Why use it?
  - Detect and monitor neovascularization (CNV, Diabetic, etc)

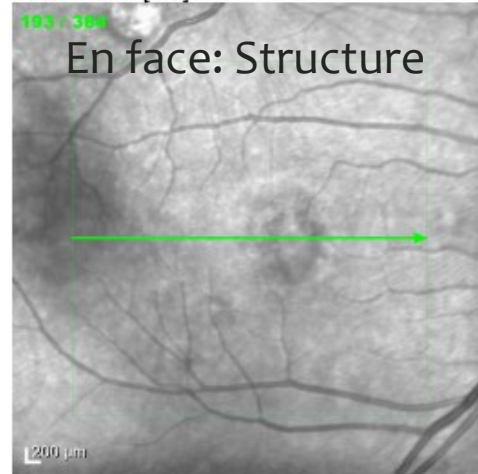
Patient:  
Patient ID:  
Diagnosis: ---

DOB: Apr/21/1962  
Exam.: Feb/19/2024  
Comment: ---

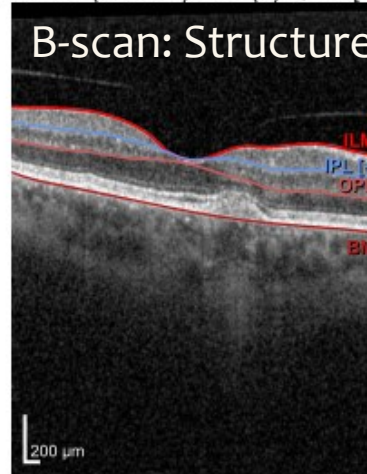
Sex: F

OD

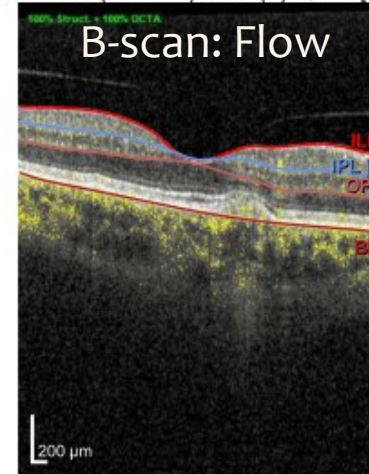
IR 20° ART [HS]



OCT 15° (4.3 mm) ART (4) Q: 33 [HS]



OCT 15° (4.3 mm) ART (4) Q: 33 [HS]



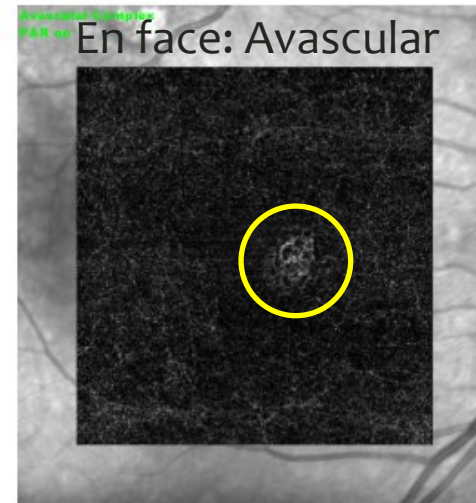
Superficial Vascular Complex  
PAR on



Deep Vascular Complex  
PAR on



Avascular Complex  
PAR on



Notes:

Date: 2/19/2024

Signature:

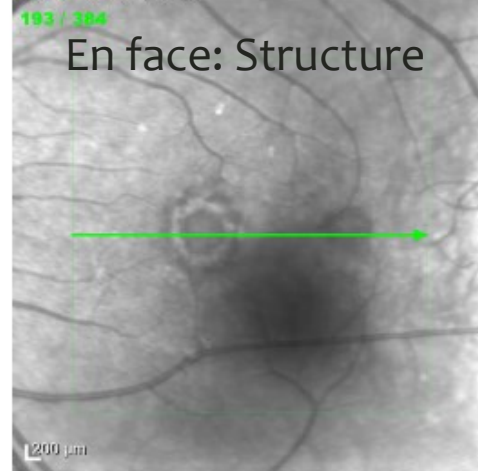
Patient:  
Patient ID:  
Diagnosis: ---

DOB: Apr/21/1962  
Exam.: Feb/19/2024  
Comment: ---

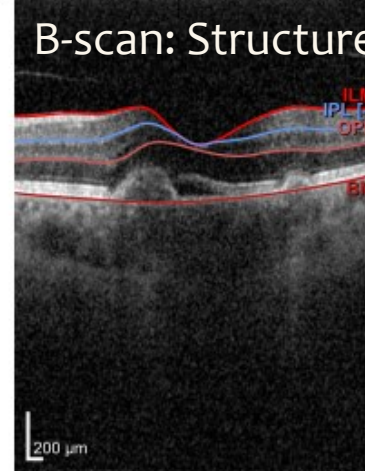
Sex: F

OS

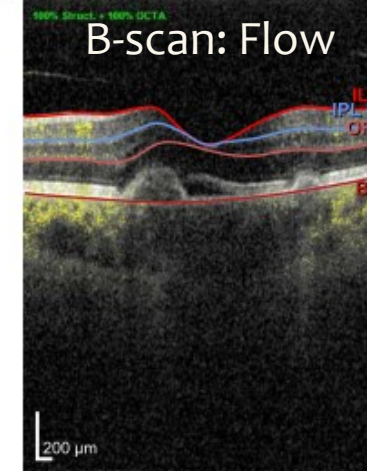
IR 20° ART [HS]



OCT 15° (4.3 mm) ART (4) Q: 31 [HS]



OCT 15° (4.3 mm) ART (4) Q: 31 [HS]



Superficial Vascular Tomogram



Deep Vascular Tomogram



Avascular Tomogram



Notes:

Date: 2/19/2024

Signature:

# Preventing Reactivation of POHS

- Re-exposure to histoplasmosis may play a role in reactivating retinal lesions and promoting development of maculopathy
- Avoid high risk areas where histoplasmosis levels tend to be highest
  - Caves, chicken coops, dusty old buildings
- Protect yourself or avoid high risk activities
  - Construction and demolition, working with poultry, HVAC installation or service, farming, gardening

# Preventing Reactivation of POHS

- Personal protective equipment
  - Masks and respirators
- Dust control
  - High efficiency air filters
  - Vacuum cleaning
  - Wetting contaminated soil
- Endogenous factors
  - Chronic fungal infections
  - LASIK



# Take Home Message

- Patients with histo spots near the central macula are at risk for vision loss due to maculopathy
- OCTA can differentiate neovascular from inflammatory maculopathy
- Take steps to decrease the risk of histo spot reactivation in at-risk patients



# CASE #5

*See no evil, hear no evil!*

# Visit 1 (1993)

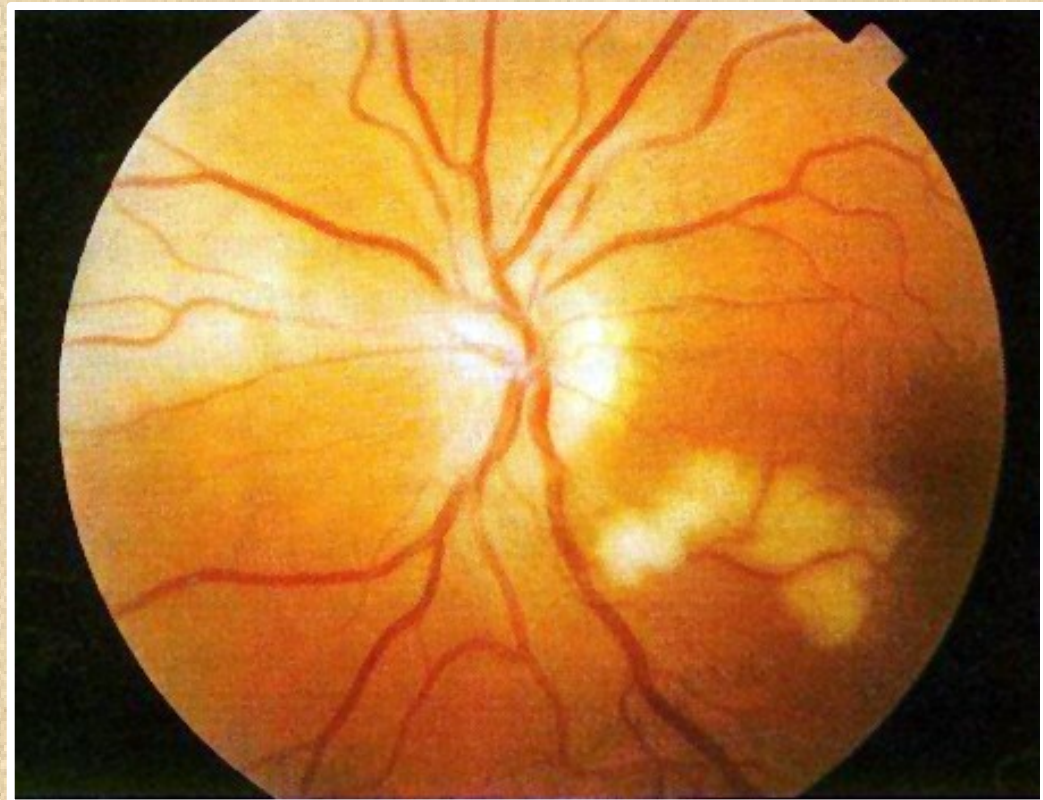
---

- 36yo WF presents with c/o “gray haze” OS x 2 weeks
  - 1 week prior she experienced left-sided HA and photopsias OS that sent her to the ER. Exam was normal and Fiorinal with codeine was prescribed
- POH: Normal.
- MH: Head and neck aches and parestheias in her arms following MVA x 2yrs
- FOH: Father with glaucoma



# Visit 1

- Vision 20/20 in each eye
- Pupils: Normal
- Motility: Normal
- IOP: 13 mmHg OU
- External: Normal



# Assessment

---

- Acute BRAO OS
- Evidence of older resolving BRAO OS

# Management

---

- Carotid duplex scan - Normal
- Echocardiography - Normal
- Labs: CBC, rheumatoid factor, RPR, fasting glucose, ANA – all normal
- Start daily low-dose ASA
- Retinal lesions resolved @ 1 month F/U visit

## Visit 2 – 3yrs Later

---

- C/O constant “flash bulb glare” OS x 1 day
- Vision: 20/20 OD, 20/25 OS
- Pupils normal, Motility normal, IOP 14/12 mmHg
- External: Normal OU
- DFE: Normal OD, BRAO of superior-temporal artery OS
  - No visible embolus
  - Occlusion does not appear to occur at bifurcation

# Assessment

---

- BRAO OS

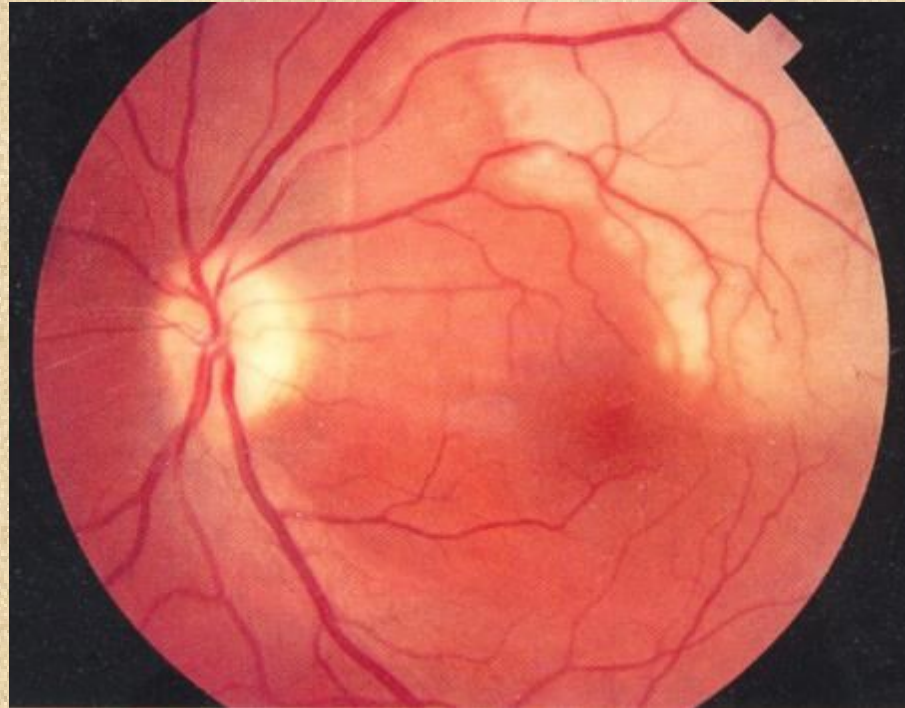
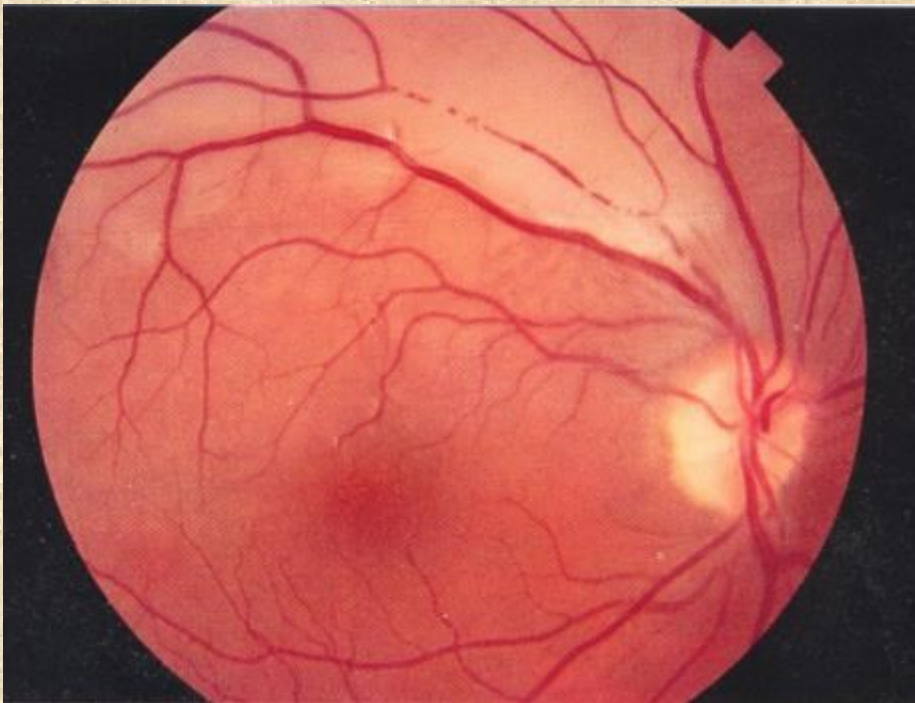
# Management

---

- Retinal consult – Diagnosis of “**idiopathic recurrent BRAO.**” No known cause or tx for condition
- IM consult – Obese (238lbs) but otherwise in good health. No BCPs, no vasculitis, synovitis, diabetes, or HTN
- Continue ASA, start low fat diet

## Visit 3

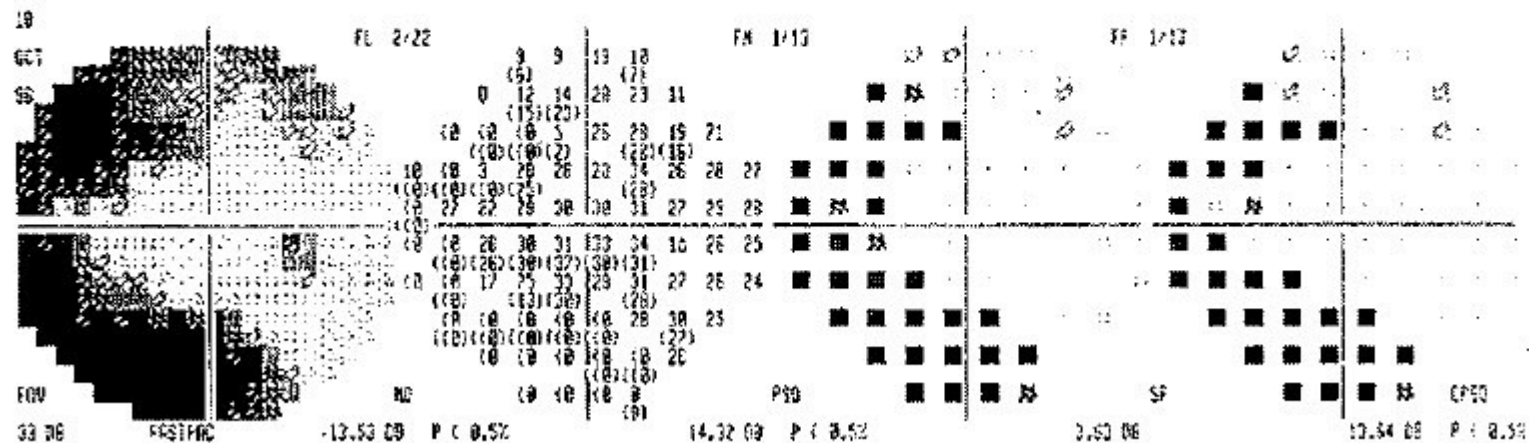
- Presented 2 weeks later with photopsia OD
- Examination was remarkable for the presence of new BRAO OD



RIGHT

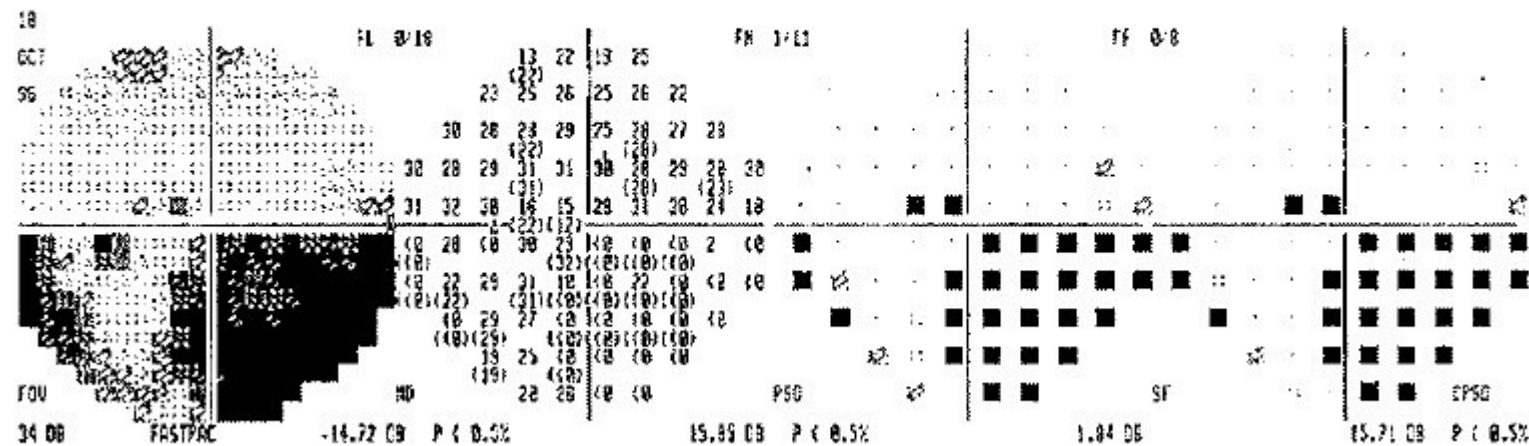
C30-2

AGE 40 STRATEGY FULL THRESHOLD



LEFT

AGE 40 STRATEGY FULL THRESHOLD



# Assessment

---

- Idiopathic recurrent BRAO, OU

# Management

---

- IM consult: Negative evaluation except mildly elevated ESR (27 mm/h, normal: 0-20)
- Rheumatology consult: Negative evaluation. Normal temporal artery biopsy
- Audiometry and otolaryngology consult: moderately severe **sensorineural hearing loss**. Referred for hearing aid fitting

# What is going on here?



<https://app.tophat.com/e/777538>

Multiple sclerosis

Systemic lupus erythematosus

Sarcoid

Susac's syndrome

Lyme disease





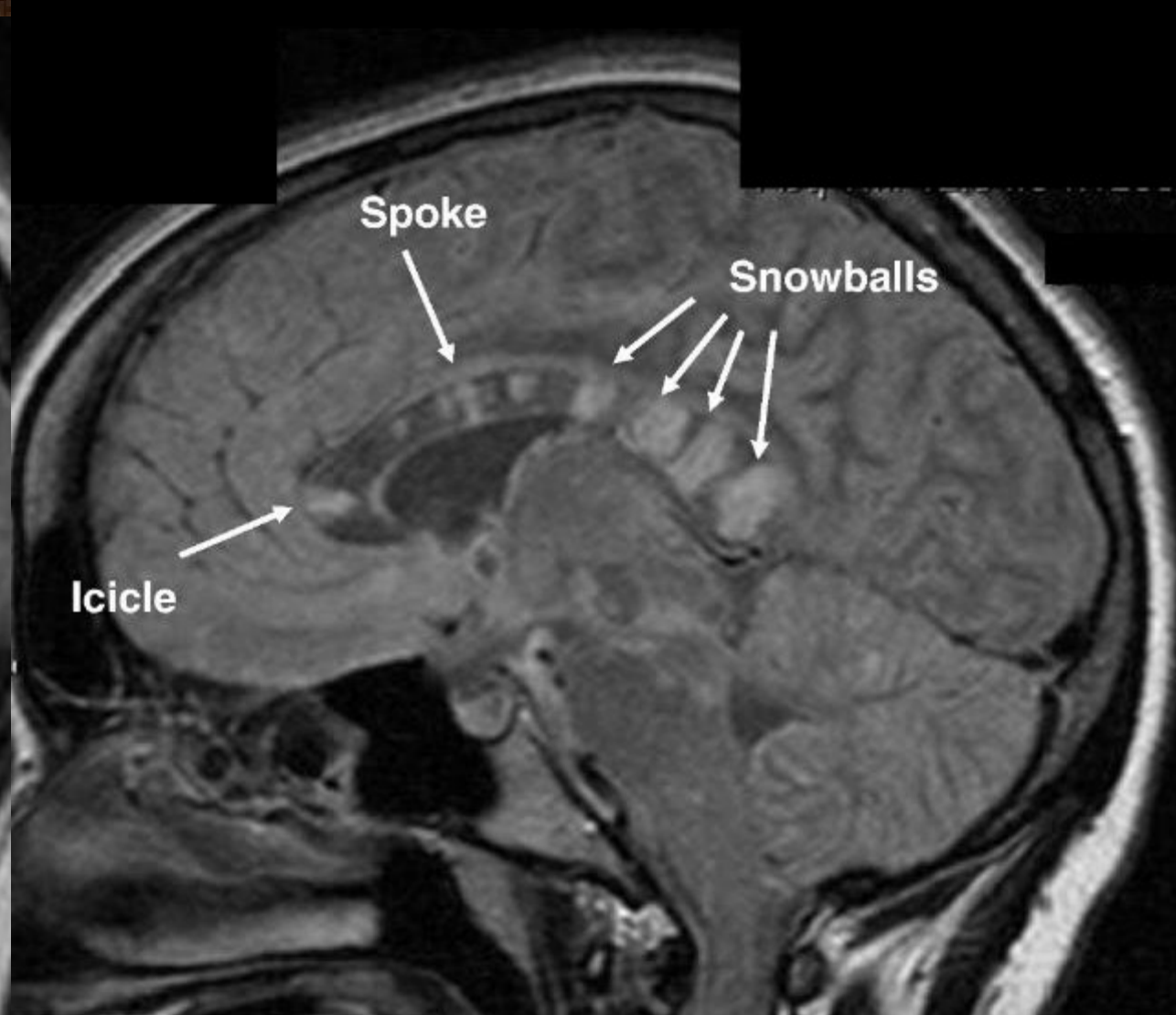
<b>Multiple sclerosis</b>	Uveitis and retinal phlebitis are common posterior segment manifestations of MS. <b>BRAO is not associated with MS</b>
<b>Systemic lupus erythematosus</b>	Retinopathy may include CWS, <b>BRAO</b> , neovascularization and VH. Diagnosis made on clinical and lab grounds.
<b>Sarcoid</b>	Antiphospholipid antibody syndrome may be associated with sarcoidosis and can lead to <b>retinal artery occlusion</b>
<b>Susac's syndrome</b>	Clinical triad of <b>encephalopathy, BRAO and hearing loss</b>
<b>Lyme disease</b>	<b>BRAO</b> has been reported as an uncommon complication of ocular Lyme borreliosis. Negative RPR test

# BRAO in the Young

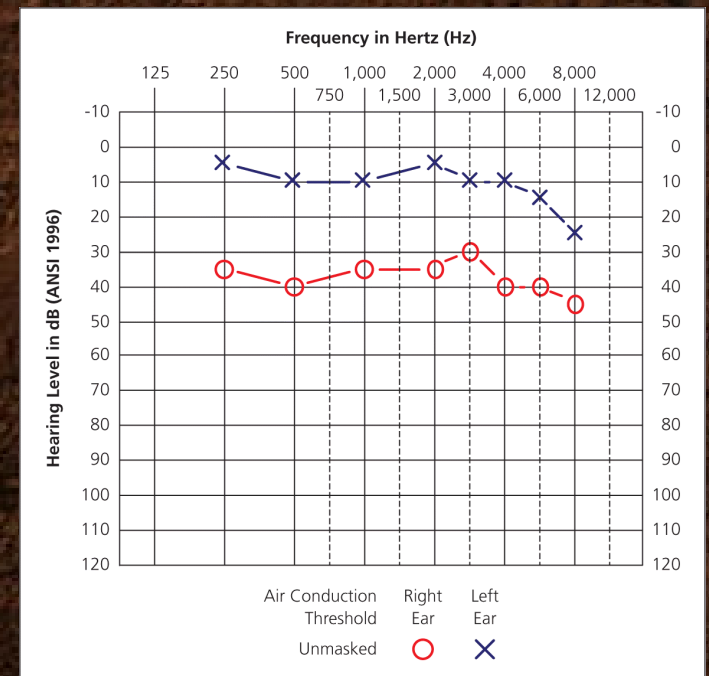
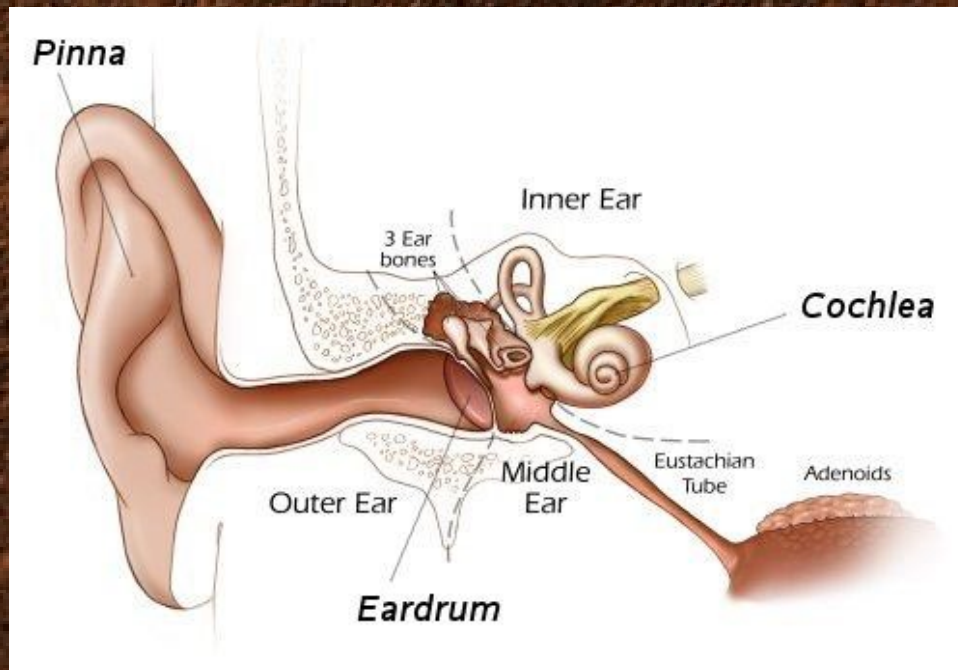
<b>Emboli</b>	Cardiac disease, IV drug abuse
<b>Thrombosis</b>	Pregnancy, BCP use, Coagulopathy
<b>Arteritis</b>	Lupus, Lyme <b><i>Susac's Syndrome</i></b>
<b>Arterial spasm</b>	Migraine, Drug abuse (cocaine, meth)
<b>Vascular compromise</b>	Orbital, optic nerve, retinal disease; Trauma

# Susac's Syndrome

- Clinical triad of (1) encephalopathy, (2) BRAO, and (3) hearing loss that typically occurs in young adult women
- First described by Susac in 1994
- Immune mediated microangiopathy affecting blood vessels in the retina, cochlea and brain
- MRI findings of lesions in the corpus callosum
- Treatment with steroids and immune suppressants can slow progression of the disease



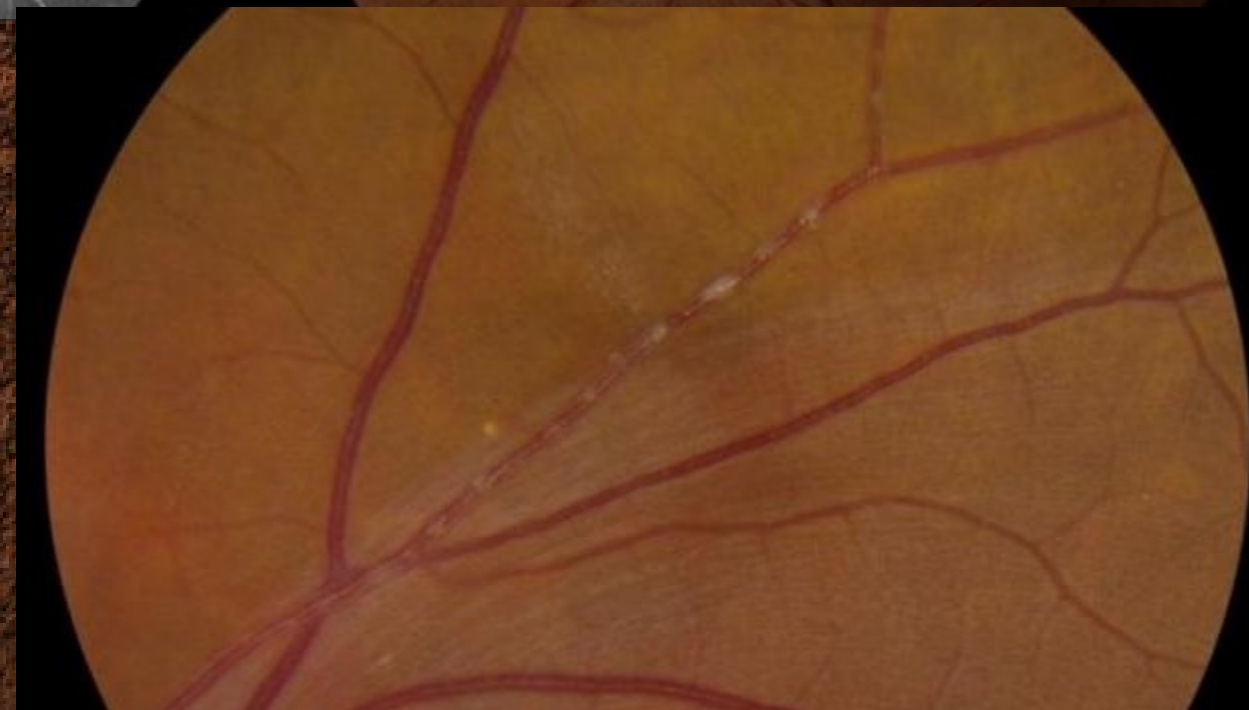
MRI findings in the corpus callosum  
of patients with Susac's syndrome



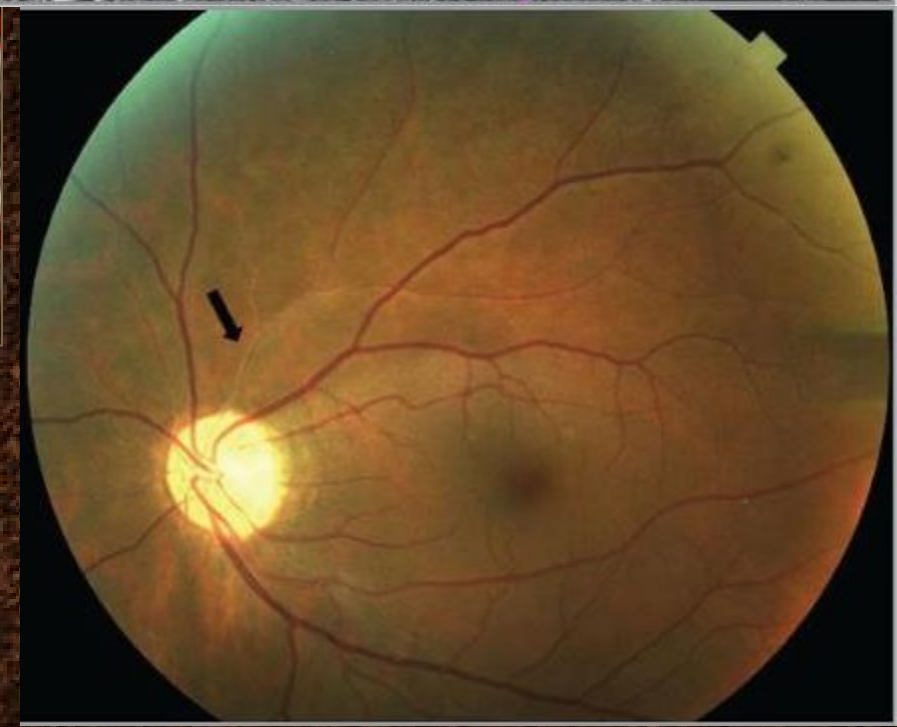
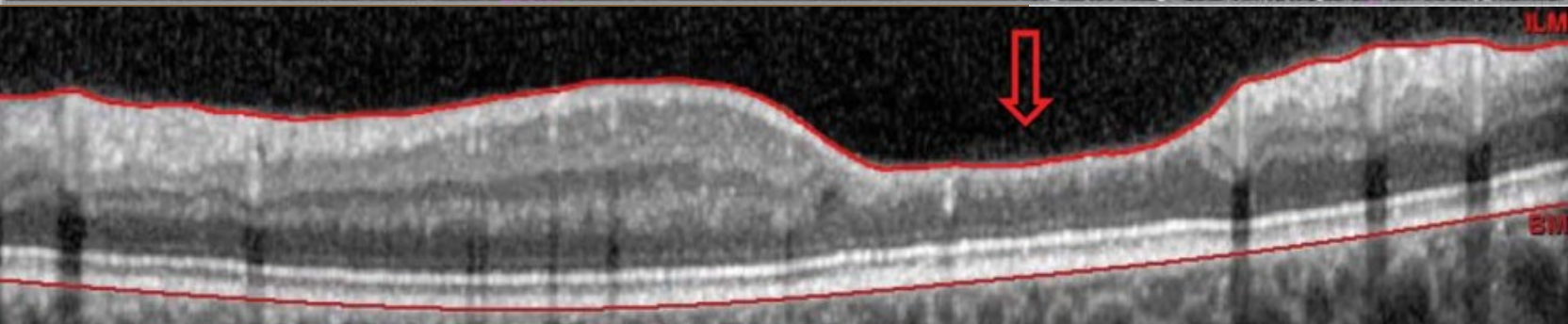
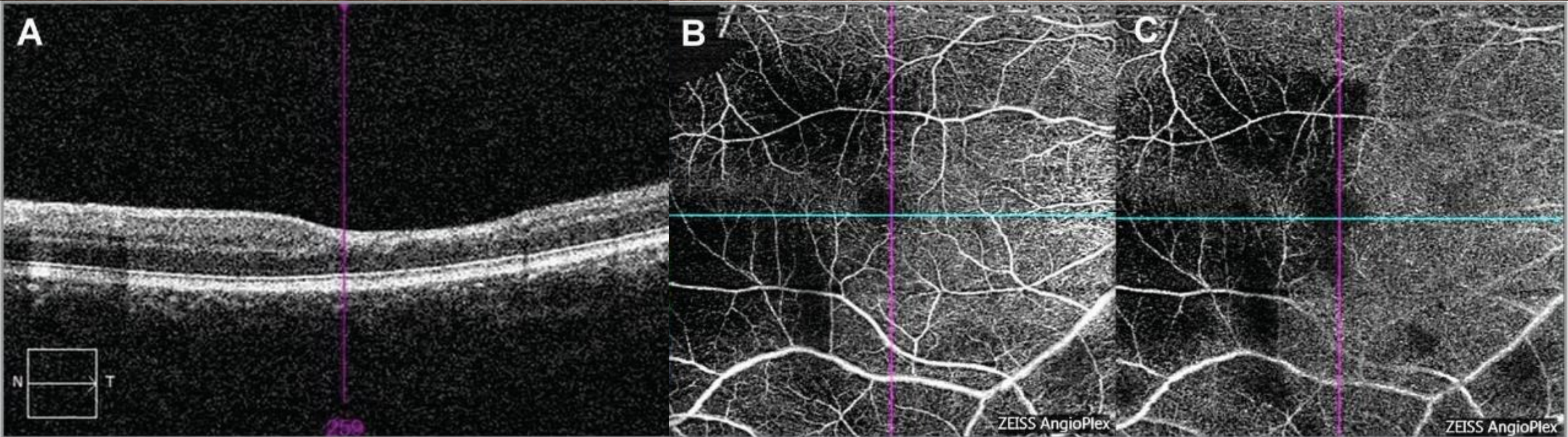
Damage to the cochlea (inner ear) results in sensorineural hearing loss, and is a key diagnostic finding in Susac's syndrome

PMID: 29933288





Acute retinal findings include arterial wall hyperfluorescence on FA and “Gass plaques” – yellow deposits believed to represent focal damage to the arterial wall



Late retinal findings include arterial attenuation, inner retinal atrophy and regions of nonperfusion

# Take Home Message

- BRAO in the young is less likely to be embolic
  - Look for coagulopathies and inflammatory disease
- Susac's syndrome is one cause of recurrent BRAO in the young
  - Check for hearing loss and MRI lesions





# CASE #6

*Glaucoma Plus!*

# CASE #6

---

- 57yo HM presents c/o distance blur x 2yrs
- POH: Diagnosed with glaucoma at age 20.
  - LEE: 10yrs ago
  - S/P unspecified glaucoma laser procedure 15yrs ago.
  - Glaucoma is not currently treated
- MH: HTN, Depression, Anxiety, OCD

# CASE #6

---

Vcc

- OD: 20/40
- OS: 20/150

Ta 28/27 @ 2:00PM

PERRL, (-) APD

FCCF: Constricted OU

Color: 2/0 (HRR)

SLE

- W&Q OU
- Patent LPI OD
- Closed LPI OS

Gonio: D4or OU

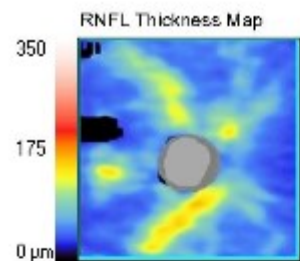
Pachs: 616/611



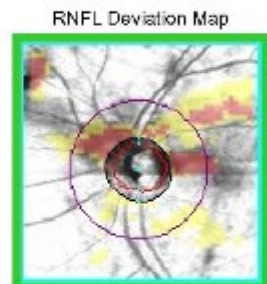
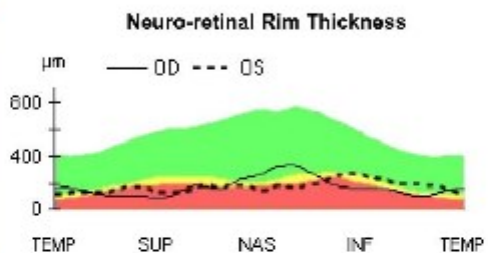
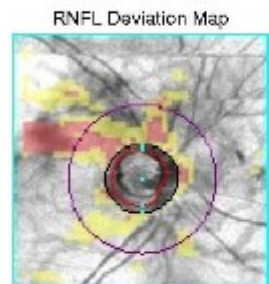
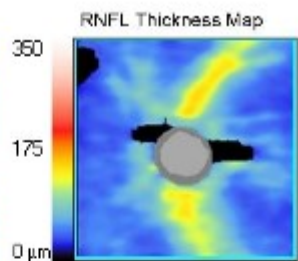
10/20/2017



10/20/2017

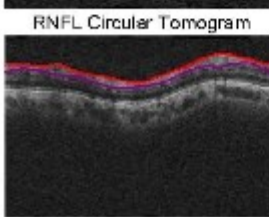
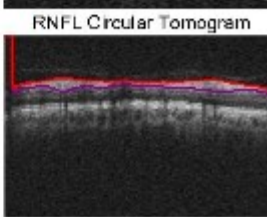
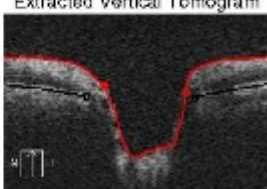
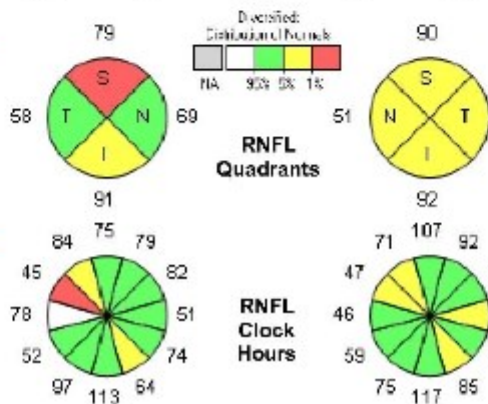
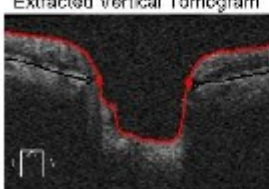
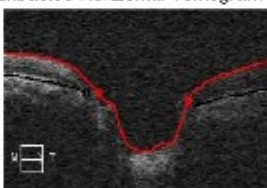
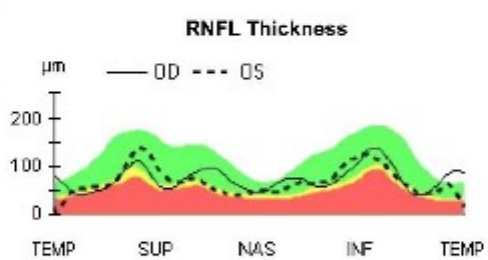
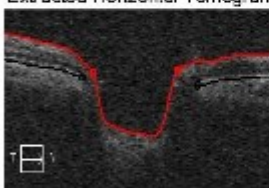


	OD	OS
Average RNFL Thickness	75 $\mu\text{m}$	70 $\mu\text{m}$
RNFL Symmetry	66%	
Rim Area	0.81 mm <sup>2</sup>	0.79 mm <sup>2</sup>
Disc Area	2.12 mm <sup>2</sup>	2.02 mm <sup>2</sup>
Average C/D Ratio	0.73	0.77
Vertical C/D Ratio	0.85	0.76
Cup Volume	0.774 mm <sup>3</sup>	0.623 mm <sup>3</sup>

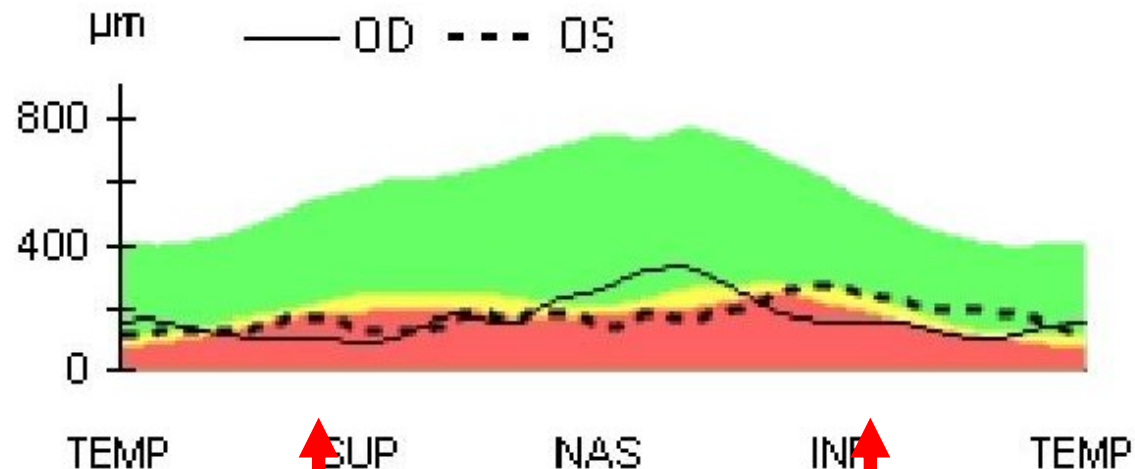


Disc Center(-0.09,-0.15)mm  
Extracted Horizontal Tomogram

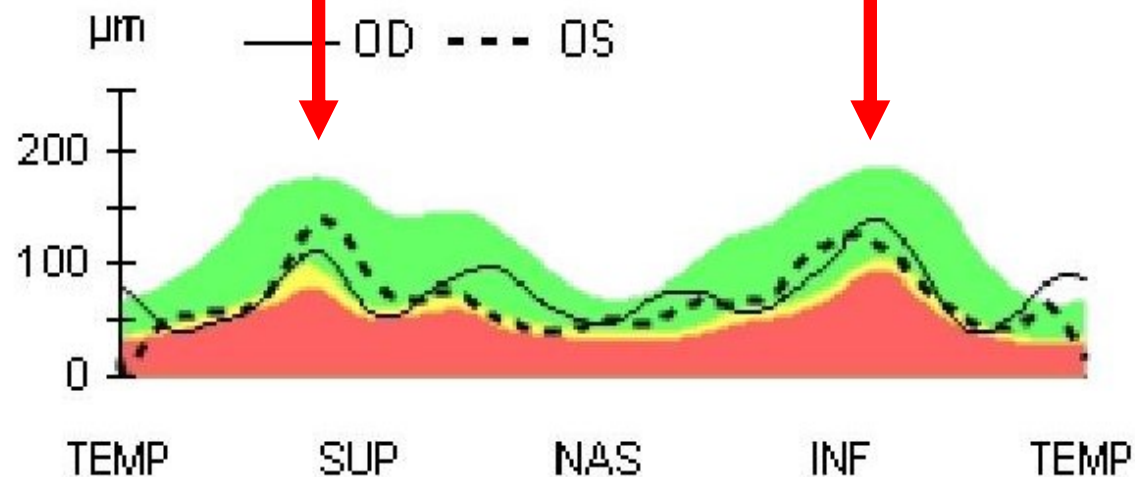
Disc Center(0.00,-0.36)mm  
Extracted Horizontal Tomogram



## Neuro-retinal Rim Thickness

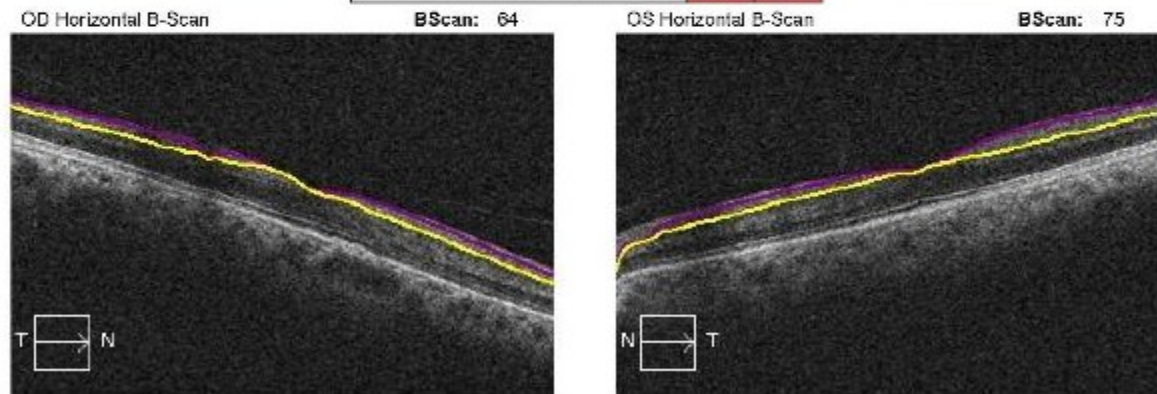
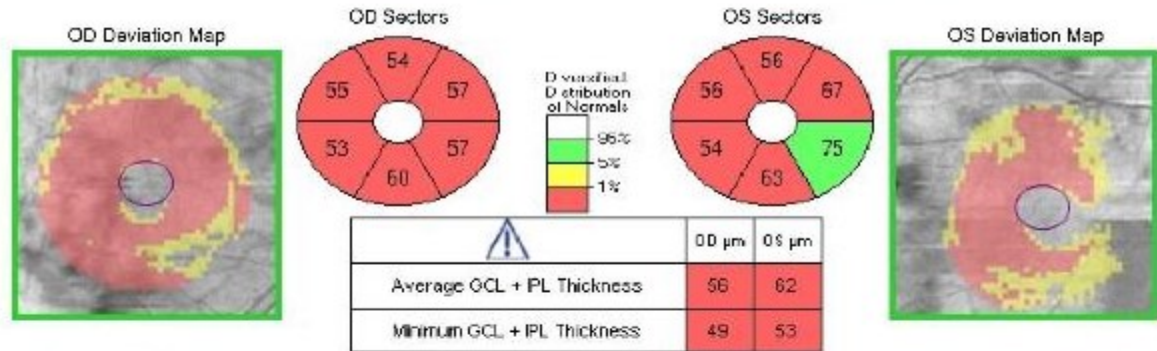
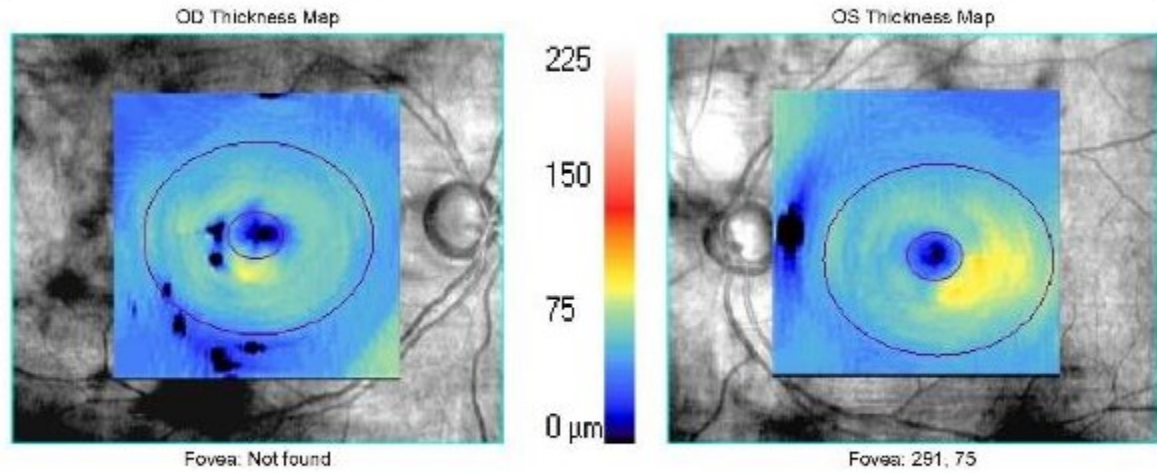


## RNFL Thickness

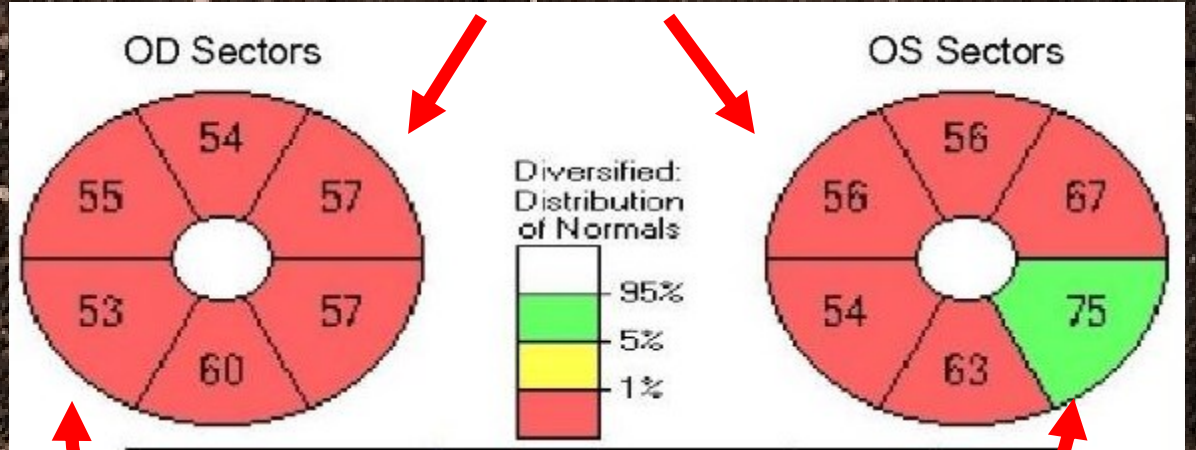


# Ganglion Cell OU Analysis: Macular Cube 512x128

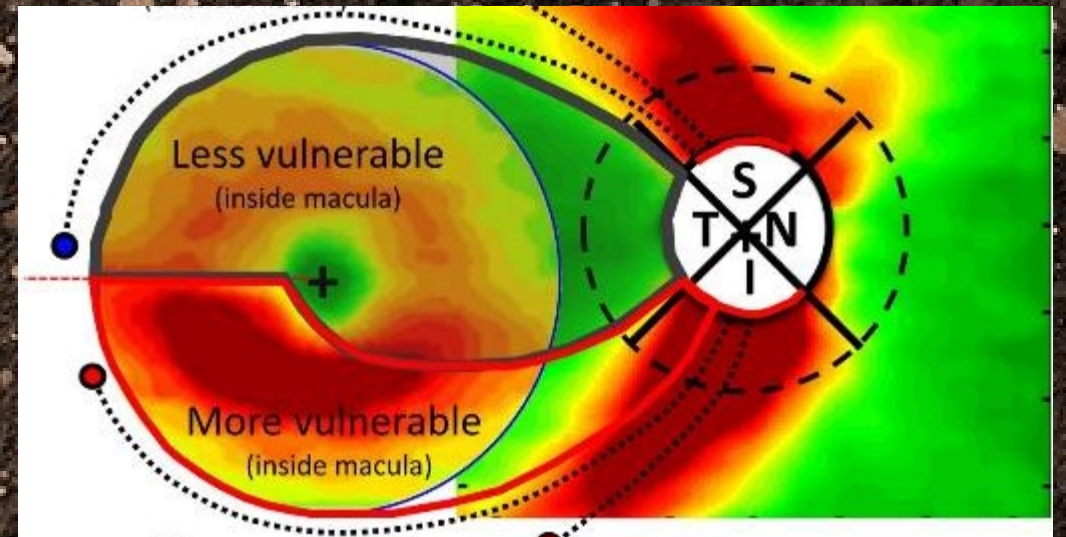
OD ● ● OS



NASAL



TEMPORAL



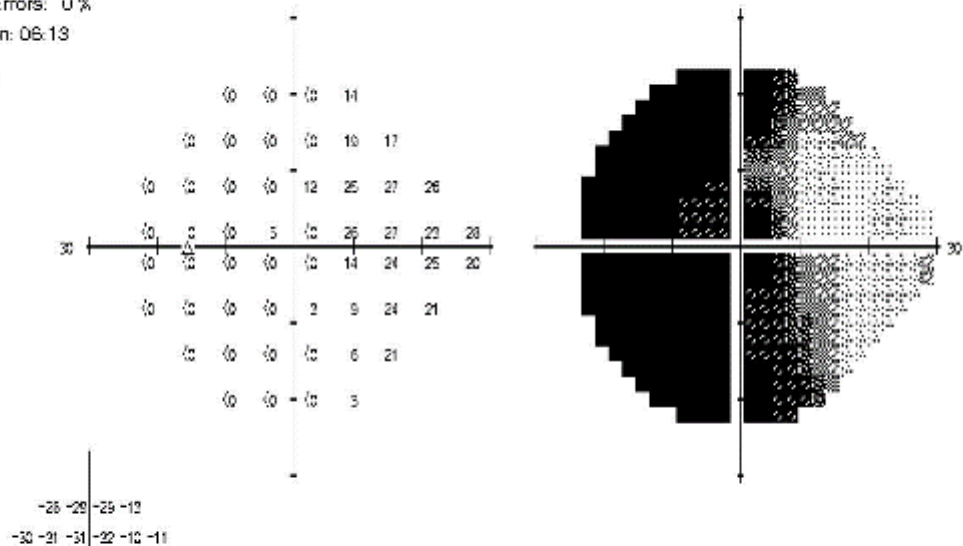
Fixation Monitor: Blind Spot  
 Fixation Target: Central  
 Fixation Losses: 1/14  
 False POS Errors: 3 %  
 False NEG Errors: 0 %  
 Test Duration: 08:13

Stimulus: Ill, White  
 Background: 31.5 ASB  
 Strategy: SITA-Standard

Pupil Diameter:  
 Visual Acuity:  
 RX: +1.75 DS DC X

Date: 10-20-2017  
 Time: 1:04 PM  
 Age: 57

Fovea: OFF



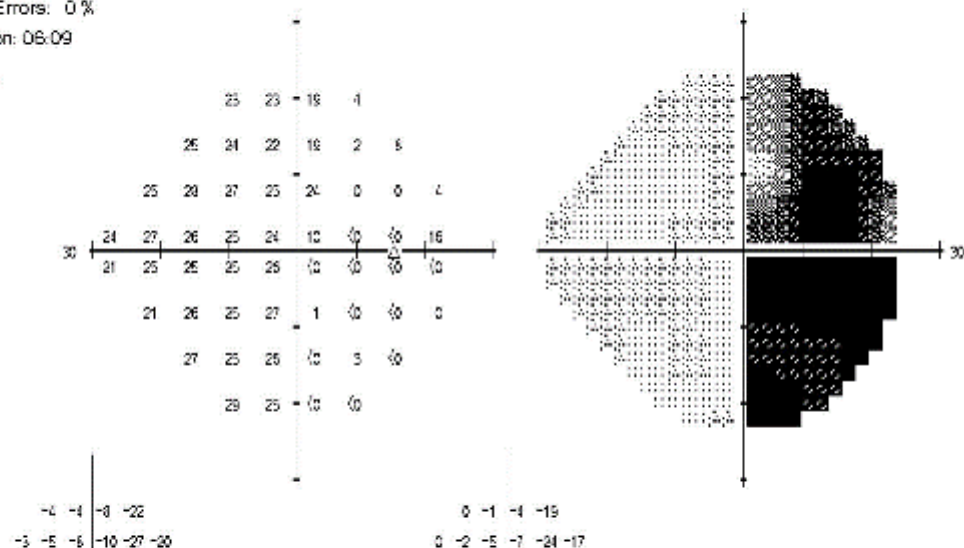
Fixation Monitor: Gaze/Blind Spot  
 Fixation Target: Central  
 Fixation Losses: 2/15  
 False POS Errors: 0 %  
 False NEG Errors: 0 %  
 Test Duration: 08:09

Stimulus: Ill, White  
 Background: 31.5 ASB  
 Strategy: SITA-Standard

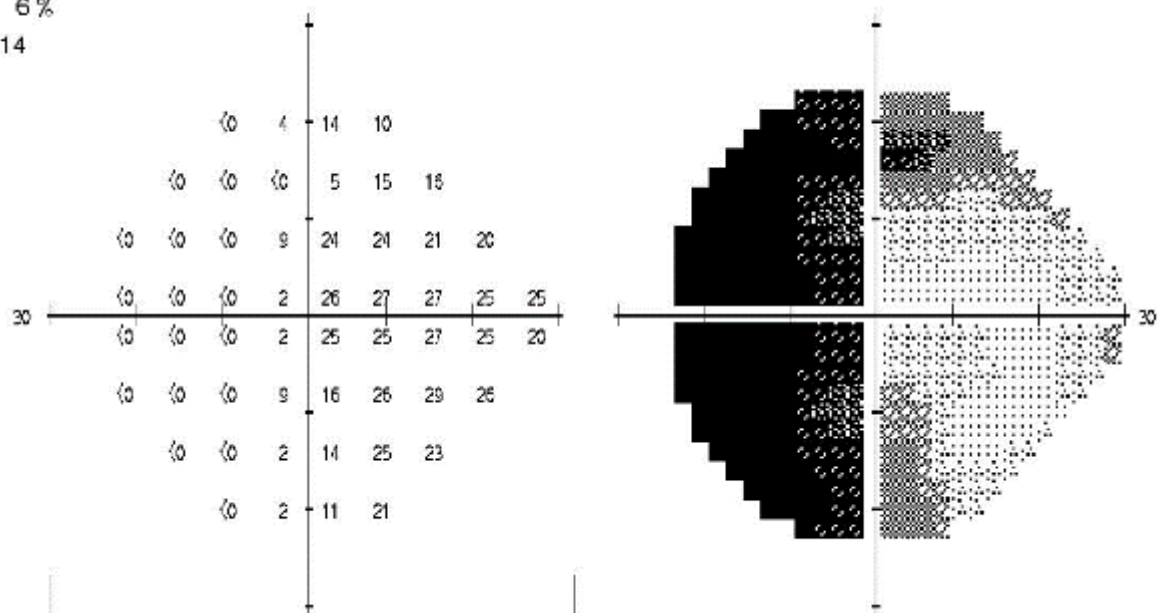
Pupil Diameter:  
 Visual Acuity:  
 RX: +1.00 DS -1.25 DC X 50

Date: 10-20-2017  
 Time: 12:56 PM  
 Age: 57

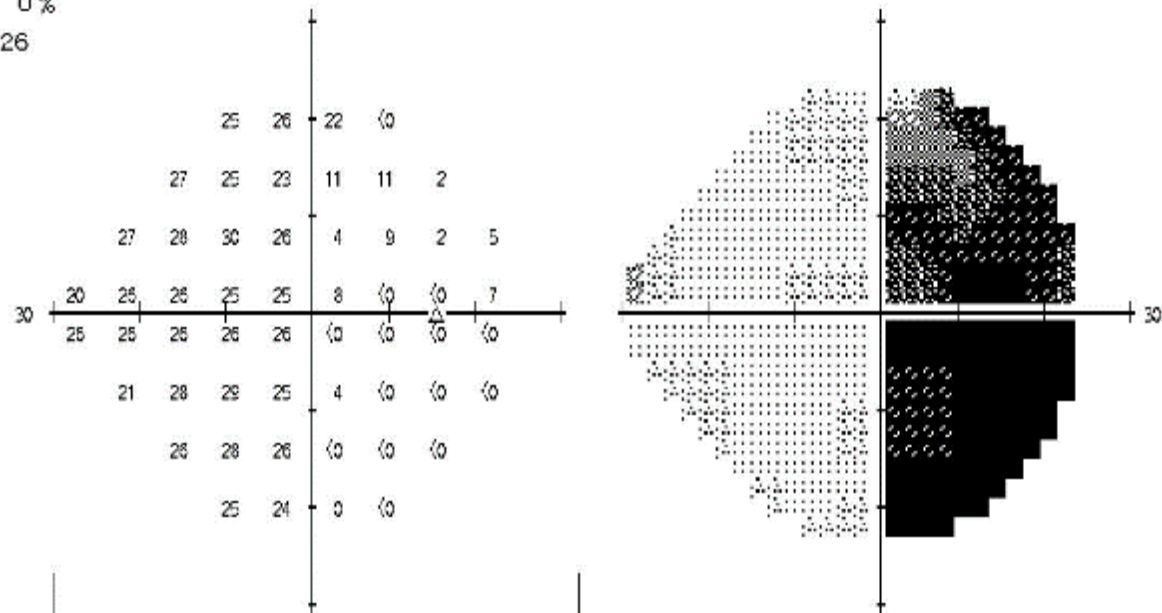
Fovea: OFF



ors: 6 %  
 7:14



ors: 0 %  
 07:26



# Now what?

<https://app.tophat.com/e/777538>



Send patient to ER stat

**A**

Refer patient to ophthalmology

**B**

Order MRI of head

**C**

Call patient's PCP

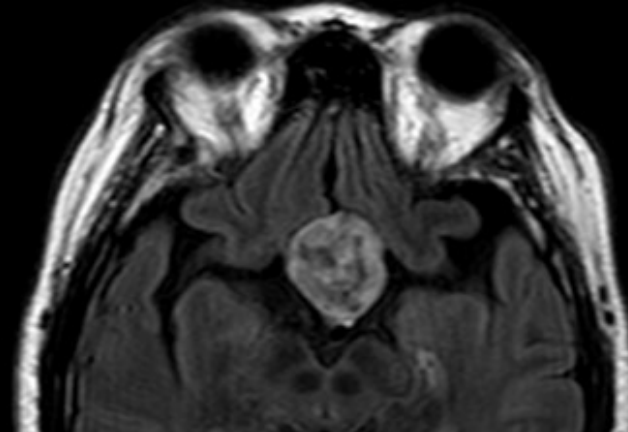
**D**

Prescribe PGA and RTC in 4 weeks

**E**



W: 1805 - L: 1039  
A# 33880217  
SE: 501 IM: 15  
3042.81064224243



Acq Tm: 9:11 PM  
Pat Pos: HFS

5cm

10cm

15cm

TE: 1  
TR: 1  
EC: 1  
4 Thk  
SP

Sorna

### IMPRESSION:

A 2.6 x 3 x 4.4 cm T1 isointense, T2 iso/hyperintense, heterogeneously enhancing sellar lesion with suprasellar extension, mass effect on the optic chiasm and less than 50% encasement of bilateral cavernous ICAs, compatible with macroadenoma.

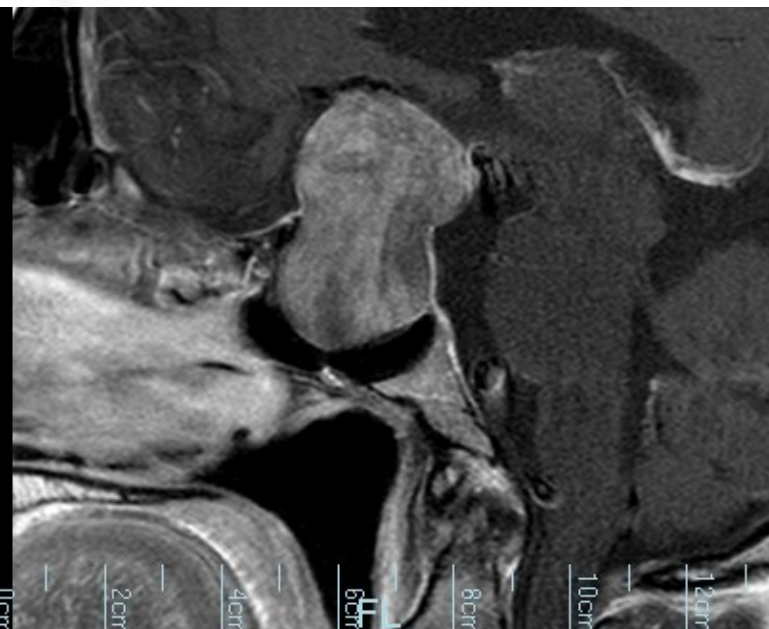
A# 3

SE: 401 IM: 7  
-3053.6687332479

AF

TE: 8.2 ET: 3  
TR: 460.651092529296  
EC: 1  
3 Thk 3.5 SP  
FC

Sorna Corporation



4cm

6cm

8cm

10cm

12cm

14cm

PH

NEX: 1  
T1 SAG 3MM PG CLEAR  
CONT:  
ZOOM: 55.47%  
768x768

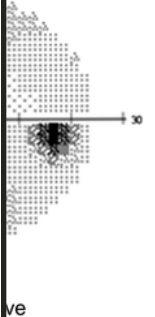
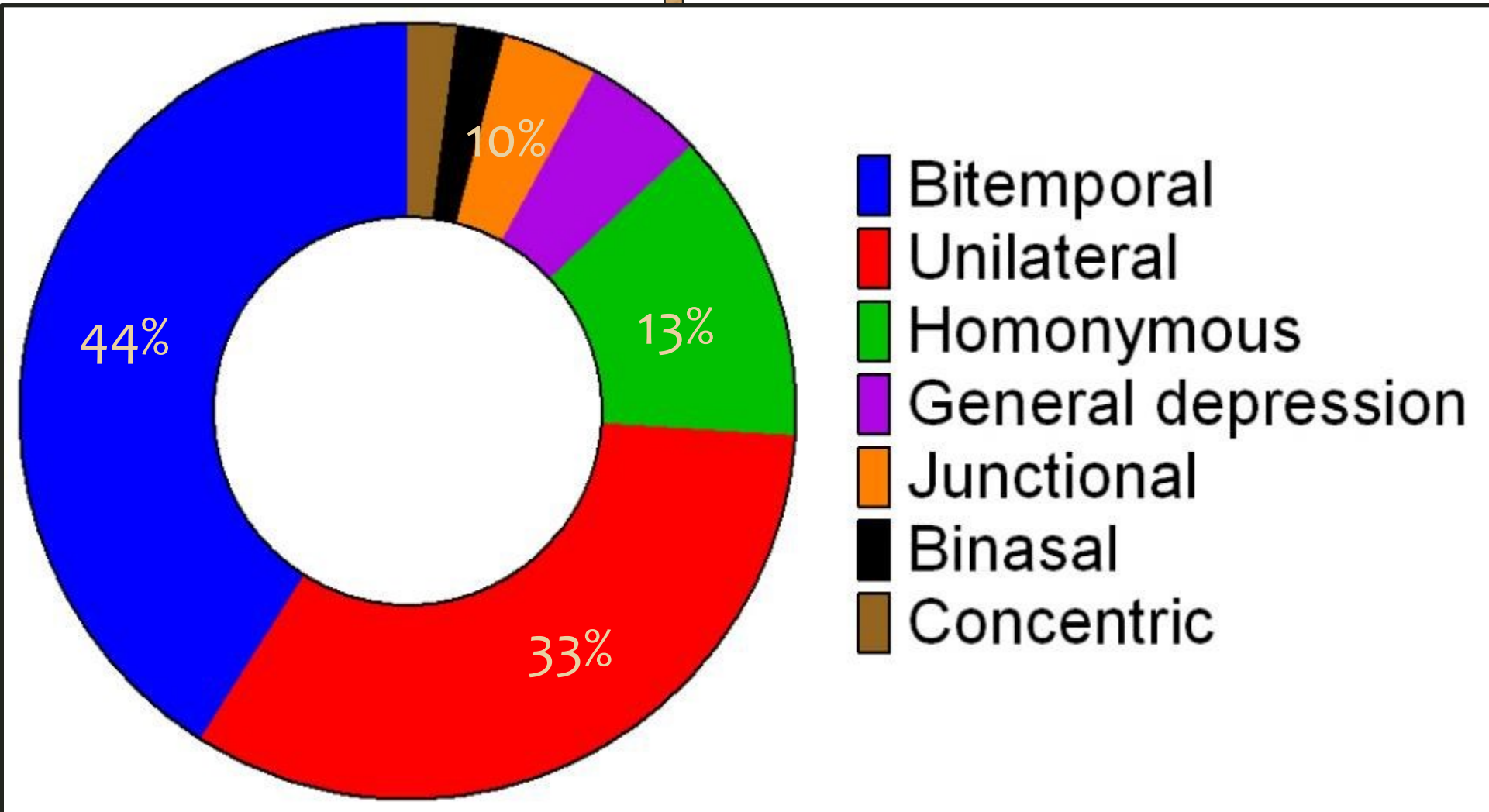
# Pituitary Adenoma Visual Field Defects

## Visual Defects in Pituitary Adenoma: Bitemporal

**OBJECTIVE.** The purpose of this study was to determine the prevalence of bitemporal hemianopia (BHA) in patients with pituitary adenoma and to assess the associated visual defects.

**MATERIALS AND METHODS.** A total of 119 patients with pituitary adenoma were included in the study. We then evaluated the visual field defects in these patients, as observed in the clinical examination. The defects included no contact, bitemporal depression ( $\geq 30^\circ$ ), and lateral displacement ( $\geq 30^\circ$ ) that were monocular or nonspecific.

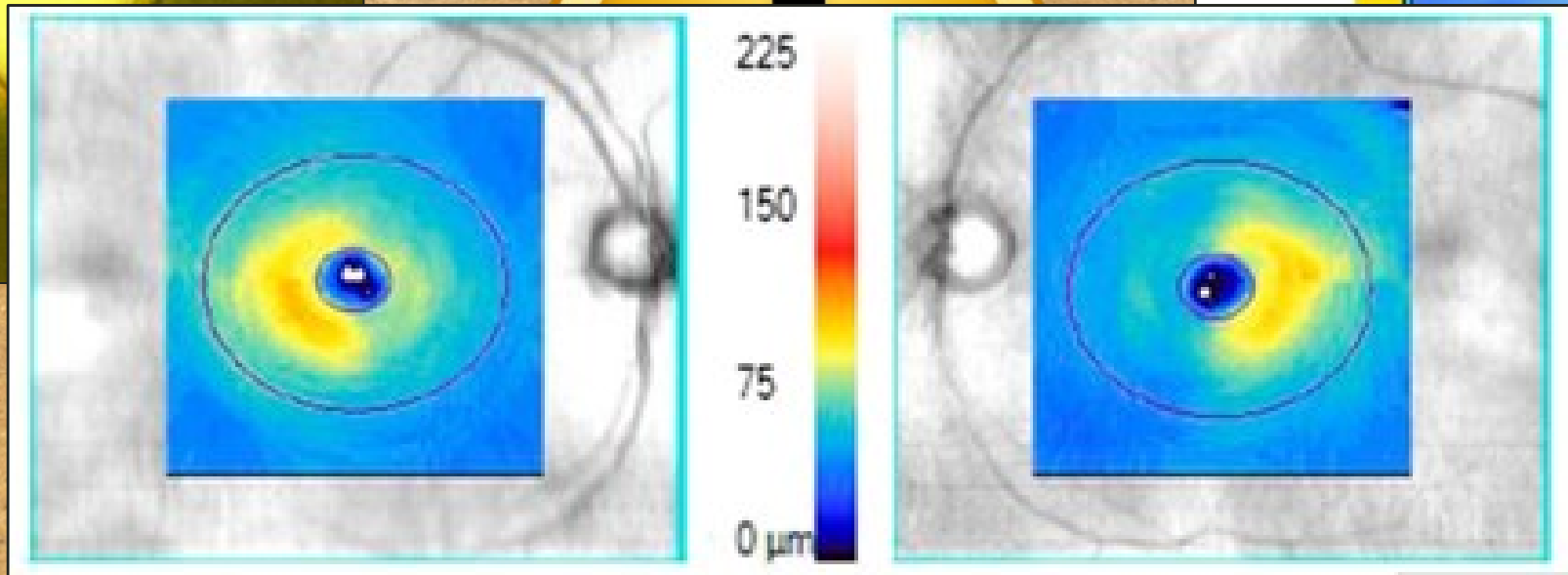
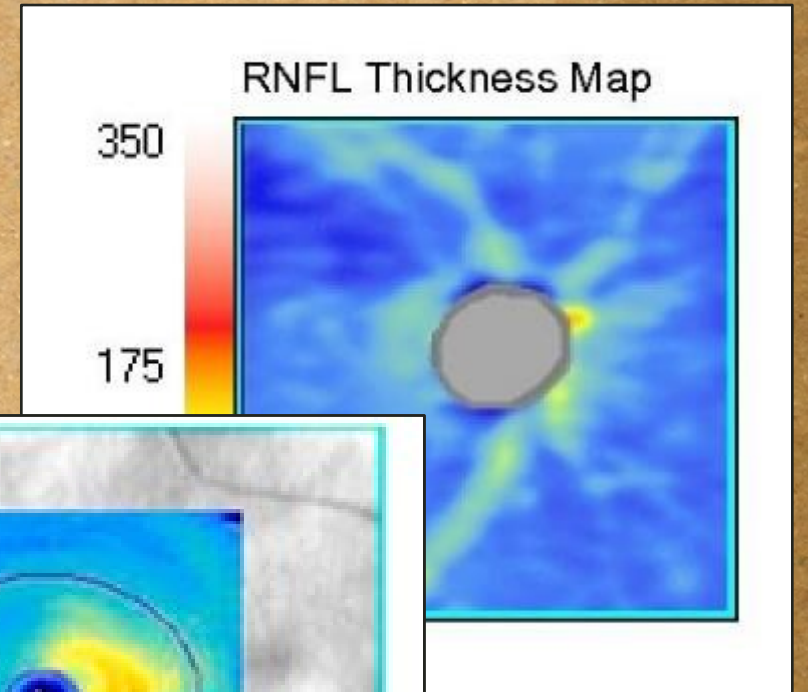
**RESULTS.** A total of 119 patients with BHA were included in the study. The most common



E

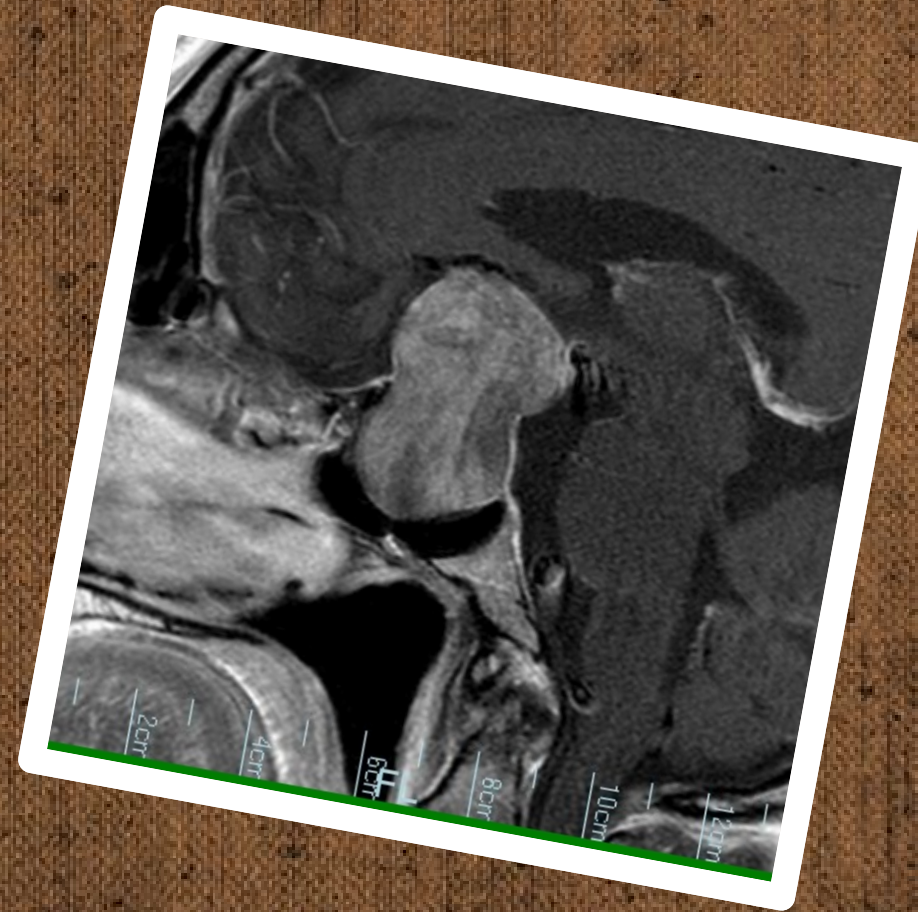
S

# Pituitary Adenomas + Glaucoma



# Take Home Message

- Glaucoma + Pituitary Adenoma
  - Chiasmal compression is an important cause of non-glaucomatous cupping
  - Binasal ganglion cell loss is a sensitive early indicator of chiasmal compression
  - Beware of glaucoma suspects with atypical findings
  - Patients can have more than one active disease process



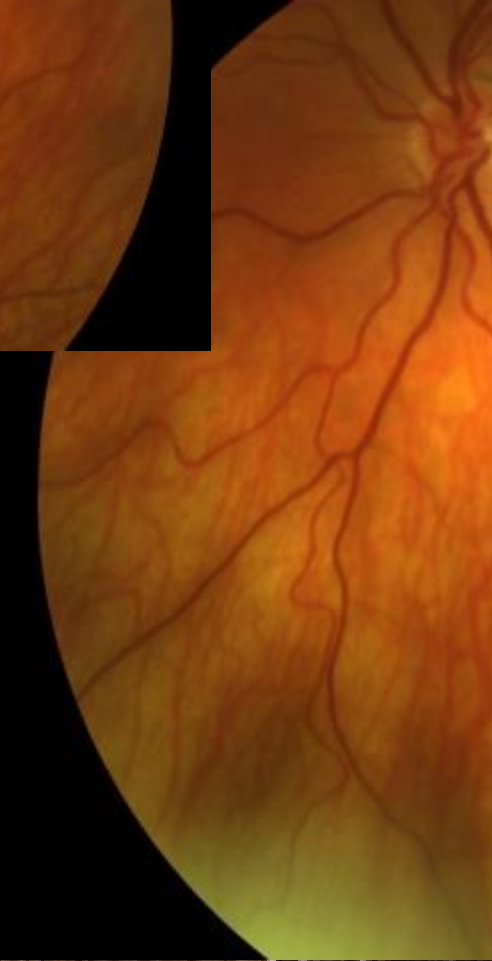
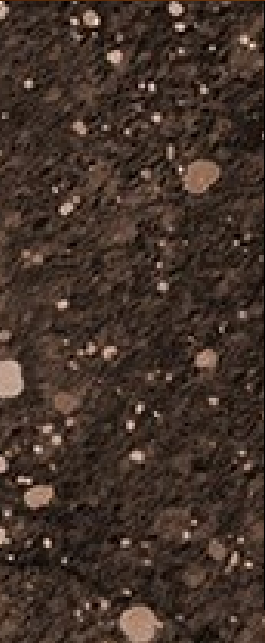
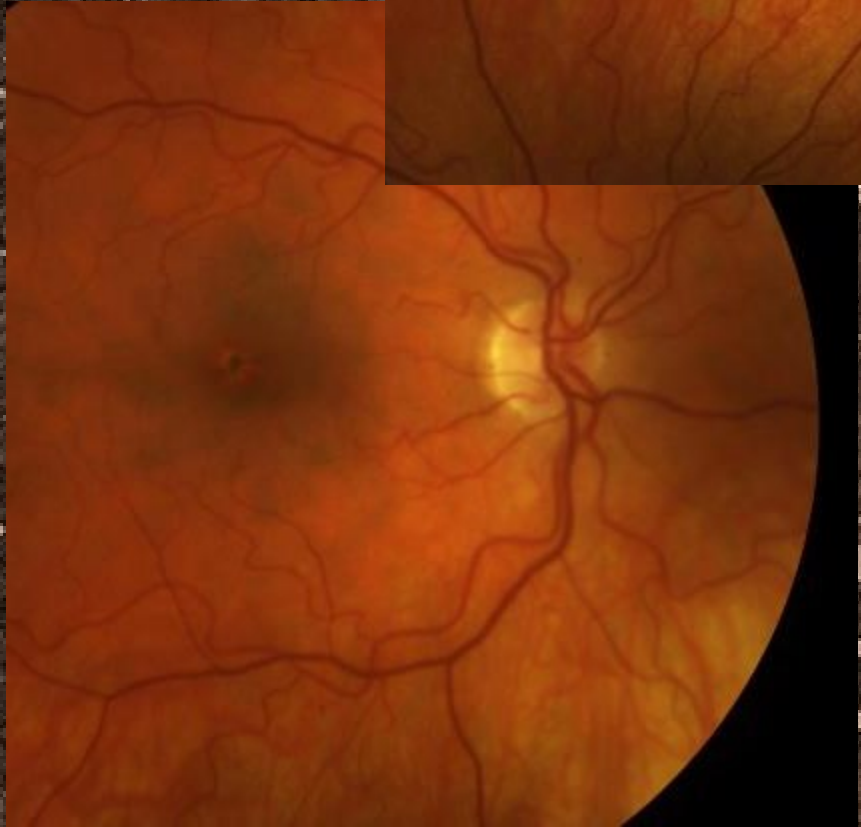
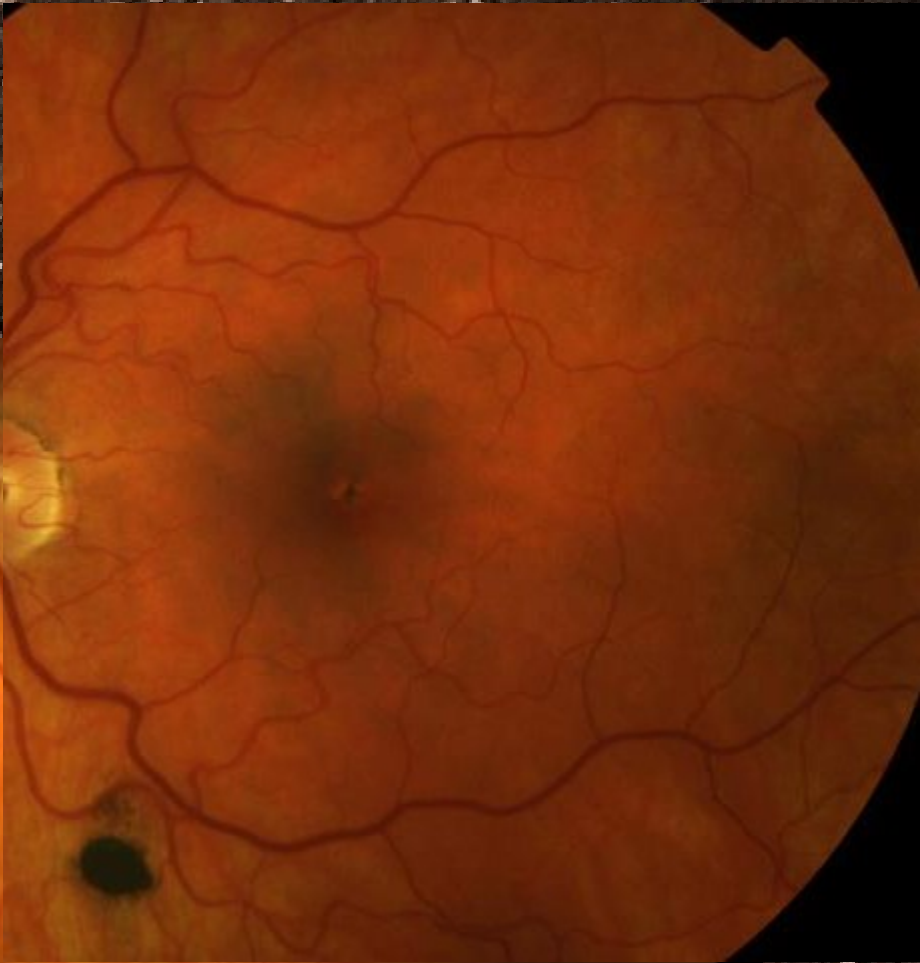
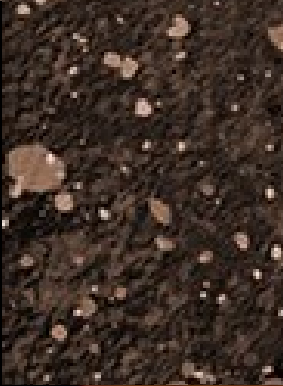
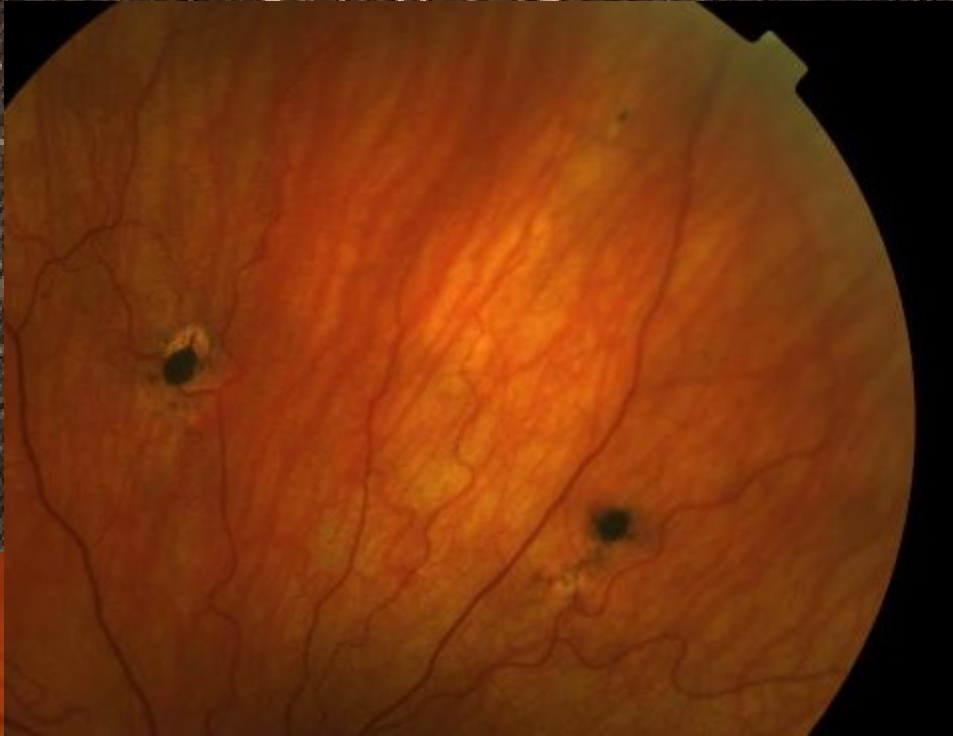
# CASE #7

*The spots are revealing!*

# Case #7

---

- Parents bring 7yo child in for his first eye exam. No complaints
- POH: No h/o any eye problems
- MH: Good health. No meds
- FOH: Unremarkable
- Vision: 20/20 each eye without correction
- Pupils and motility: Normal.
- IOP: 12/13 mmHg @ 9am
- External: Normal



Images courtesy Steven Cohen, MD

# What is going on here?

<https://app.tophat.com/e/777538>



Choroidal nevi

Congenital hypertrophy of RPE

Chorioretinal scars

Gardner's Syndrome

Retinoblastoma



<b>Choroidal nevi</b>	Slate gray mass with indistinct margins
<b>Congenital hypertrophy of RPE</b>	Flat jet-black retinal lesion with sharp margins. Multifocal CHRPE (“bear tracks”) are typically <u>unilateral</u> and clustered in a single quadrant
<b>Chorioretinal scars</b>	Composed of RPE hyperplasia (black) and fibrosis (white), sharp margins, often irregular in shape
<b>Gardner’s syndrome</b>	Familial adenomatous polyposis with CHRPE-like lesions. Retinal lesions are bilateral & may appear in >1 quadrant
<b>Retinoblastoma</b>	Yellow-white retinal mass frequently associated with subretinal and vitreous seeding

# Assessment

---

- Multiple, bilateral CHRPE-like lesions
- Suspect familial adenomatous polyposis

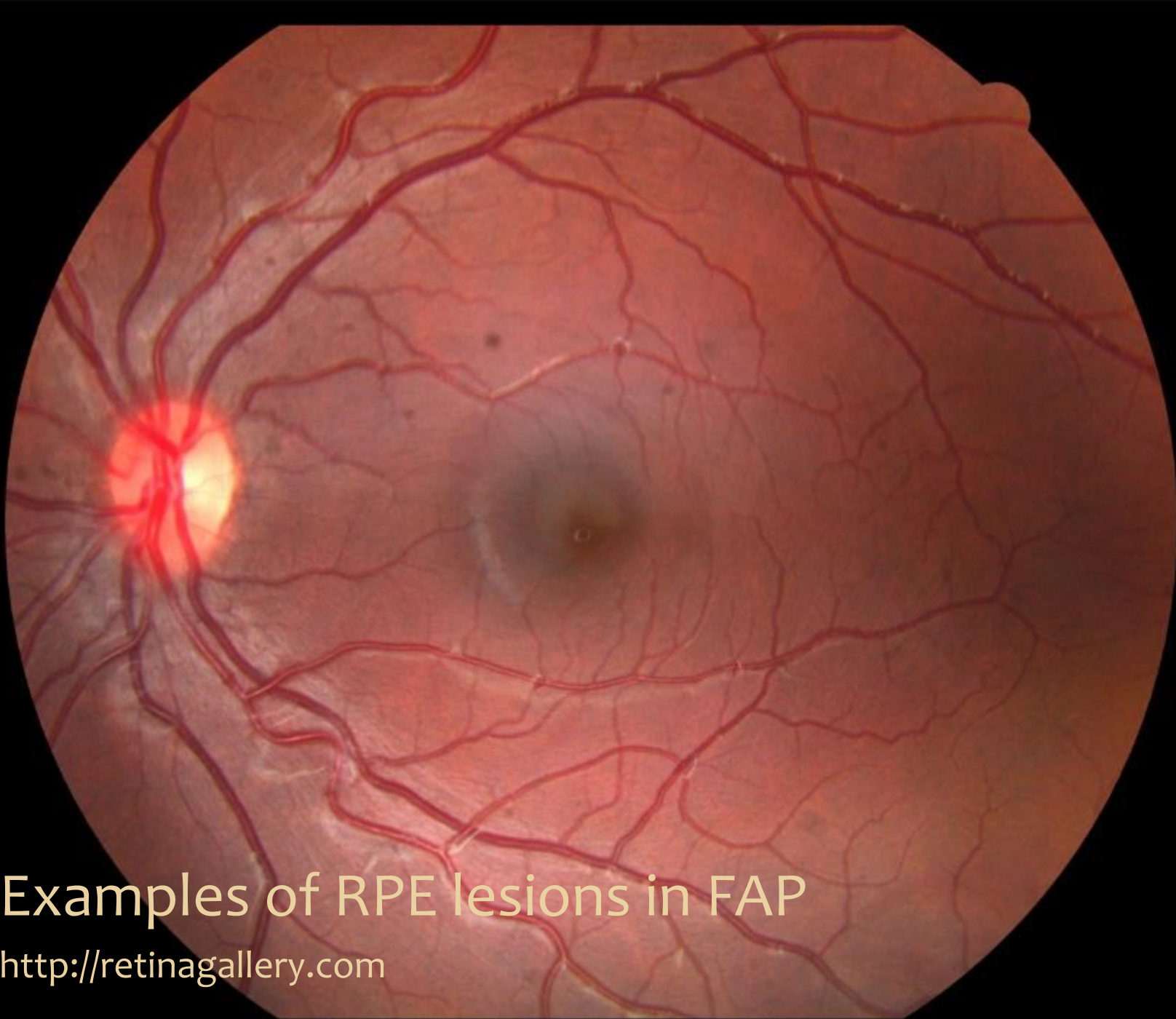
# Management

---

- Gastroenterology consult – negative colonoscopy of child and parents
- Genetic testing offered – declined by parents
- Medical surveillance for onset of polyposis

# Familial Adenomatous Polyposis

- An uncommon hereditary form of colon cancer (autosomal dominant)
  - About 1% of all colon cancer in US annually
- 20% of cases have no FH of FAP, suggesting a spontaneous mutation
- Some FAP patients have congenital CHRPE-like retinal lesions (hamartomas = benign RPE tumors)
  - Retinal lesions are a reliable clinical marker for FAP in these patients

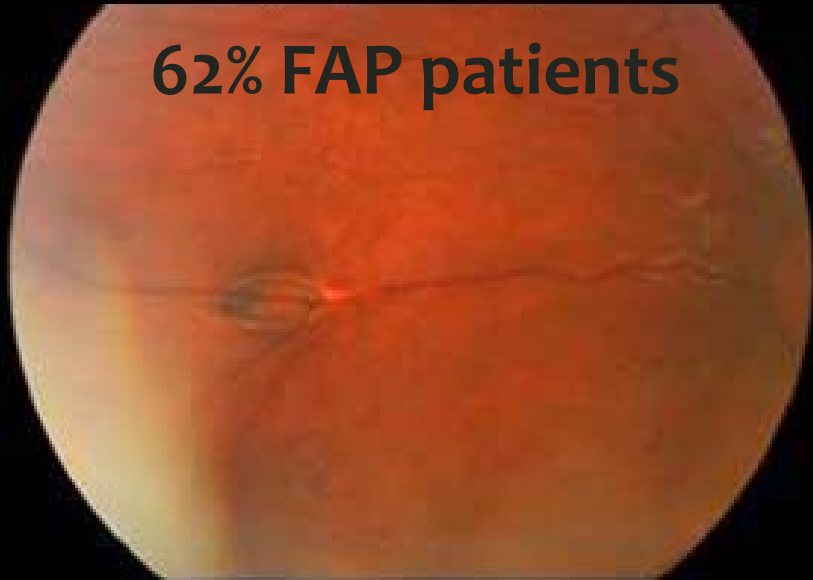


Examples of RPE lesions in FAP

<http://retinagallery.com>

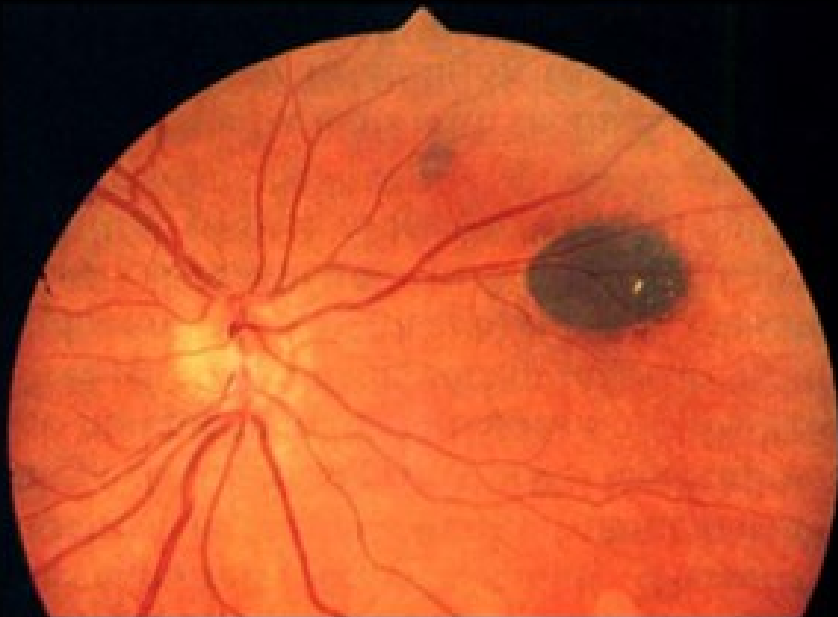
A: Oval, pigmented, with halo

62% FAP patients

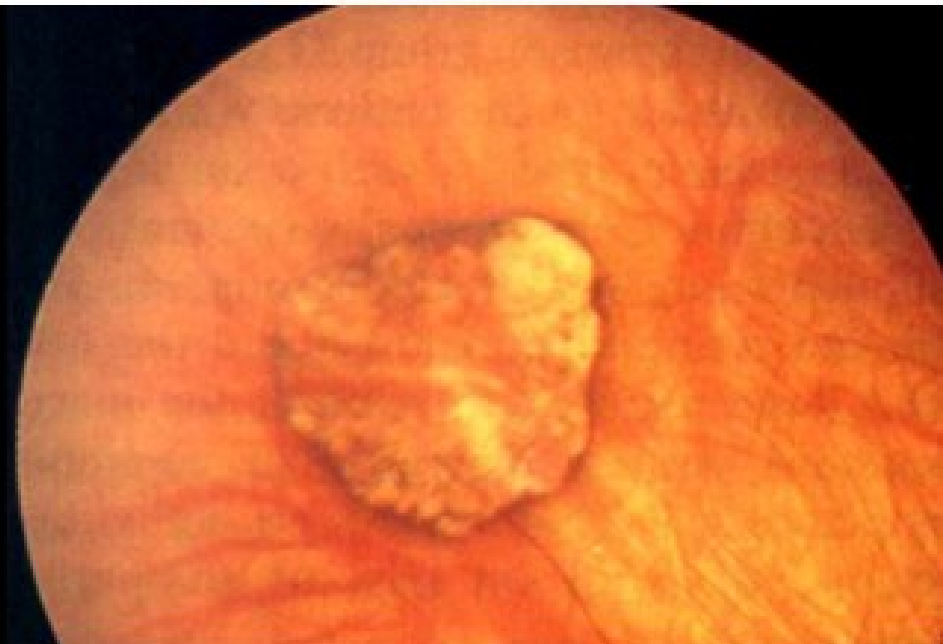


B: Round, small, pigmented, no halo

Most common lesion



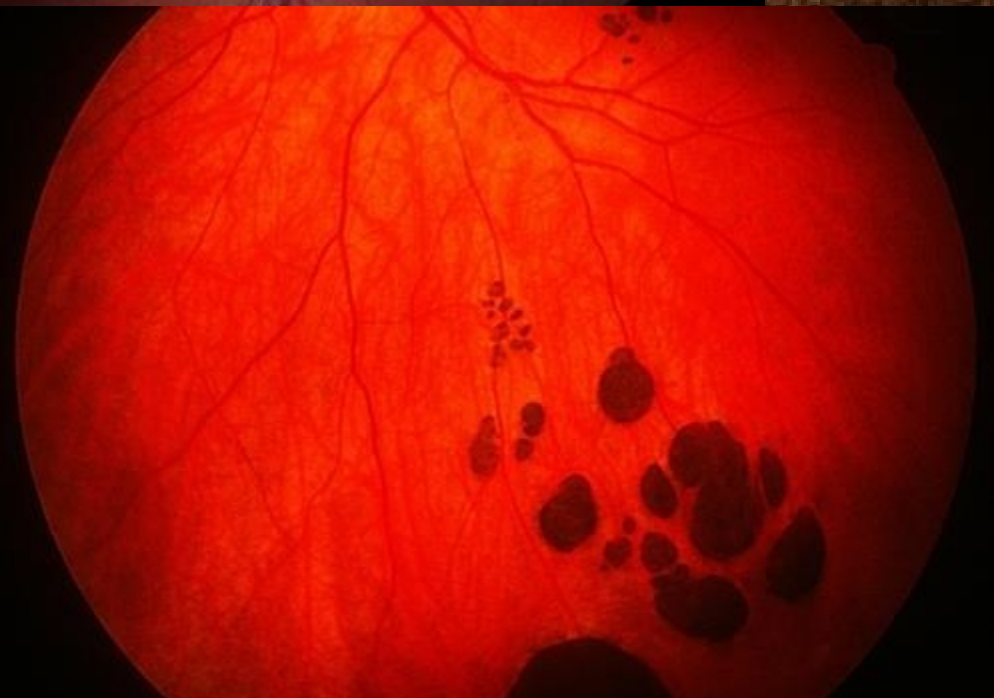
C: Round, large, pigmented



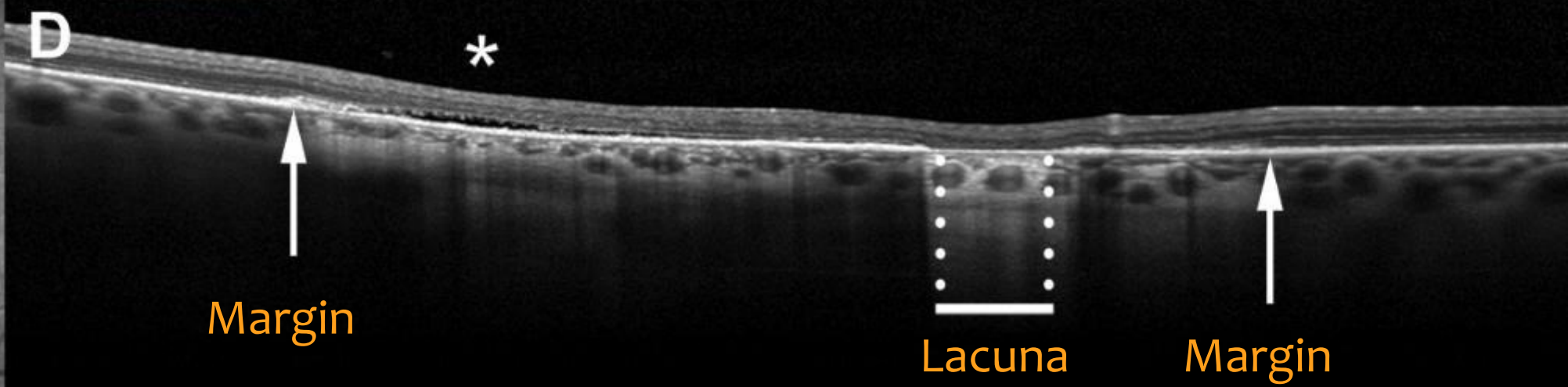
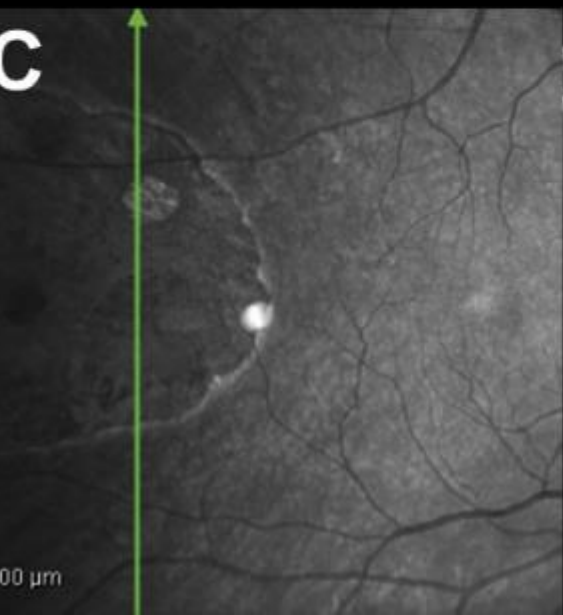
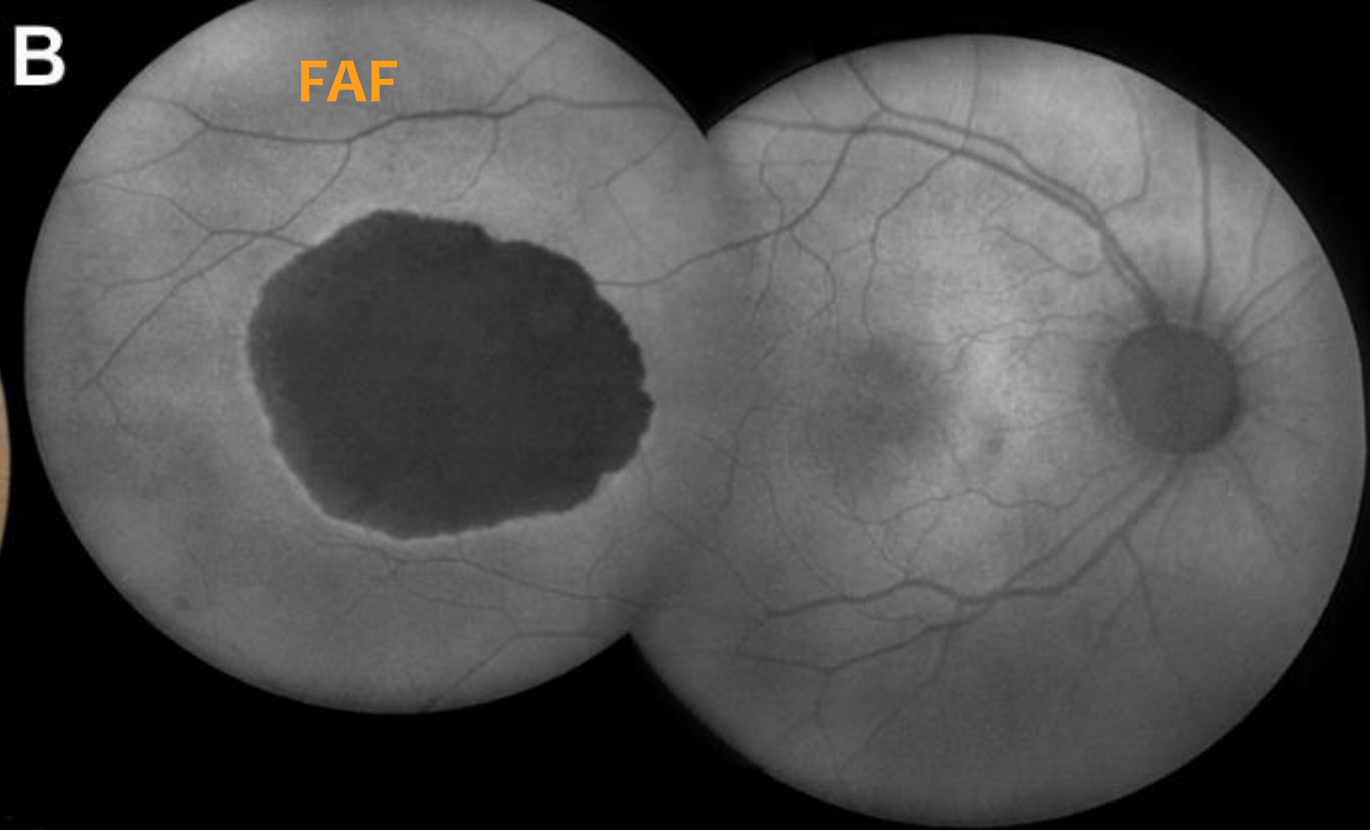
D: Round, large, depigmented

# CHRPE-like Lesions in FAP

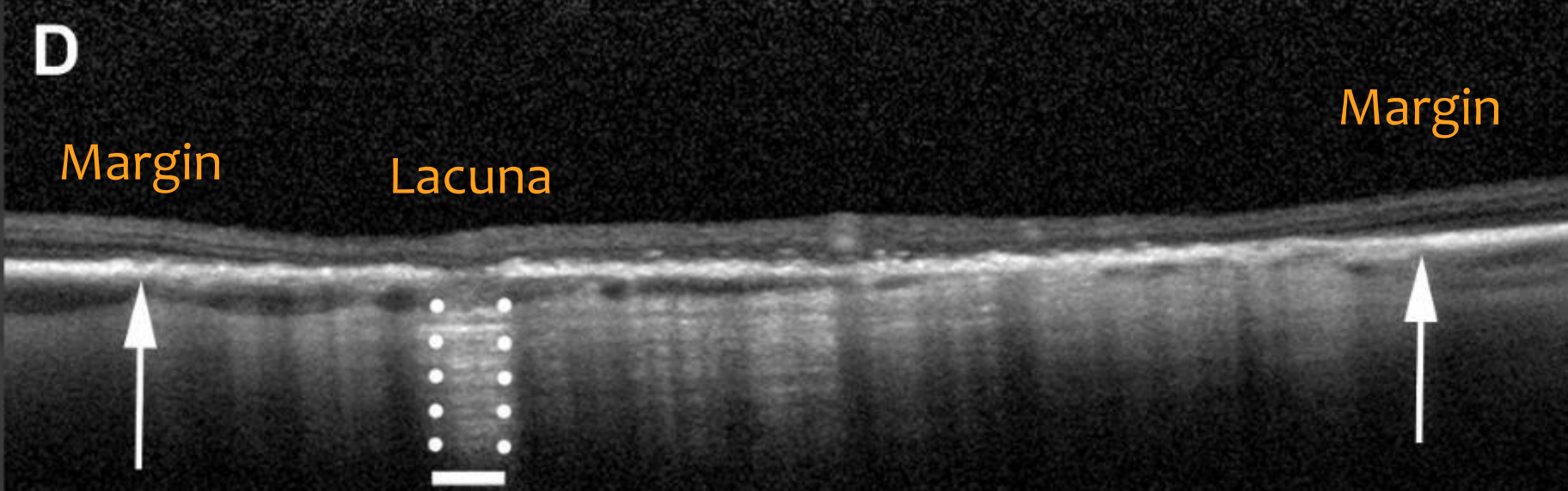
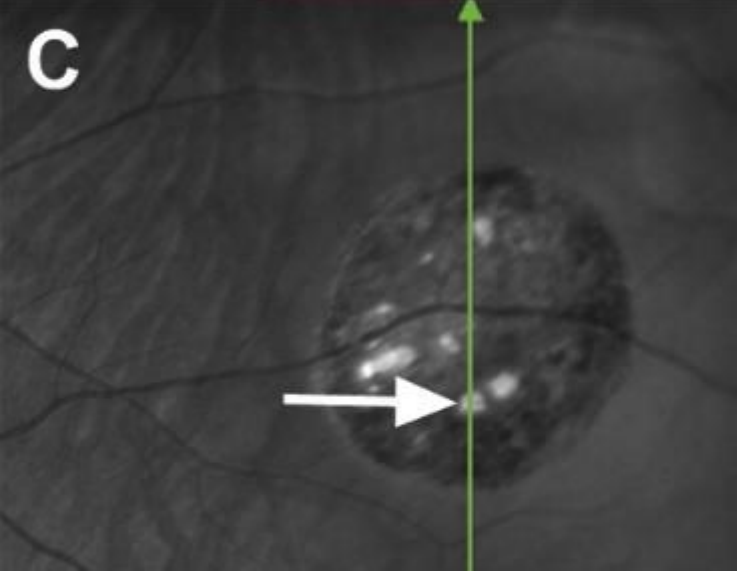
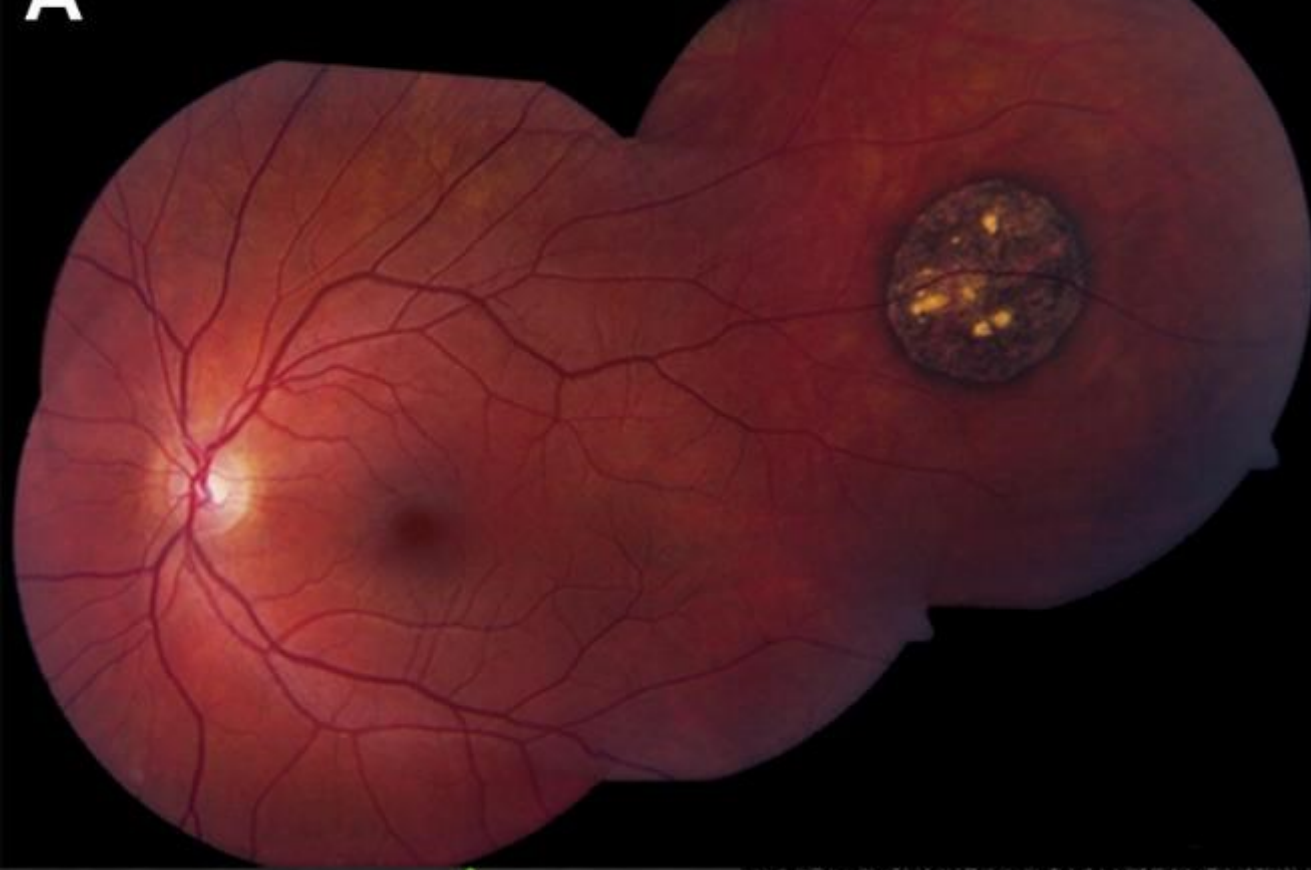
	Gardner's Syndrome	CHRPE
<b>Appearance</b>	Small: Identical to CHRPE Large: Oval with tail	Flat, round-oval, jet black
<b>Bilaterality</b>	Common (86% cases)	Very rare (5% cases)
<b>Multiple quadrants</b>	Common	Rare



Examples of  
"Bear Tracks"







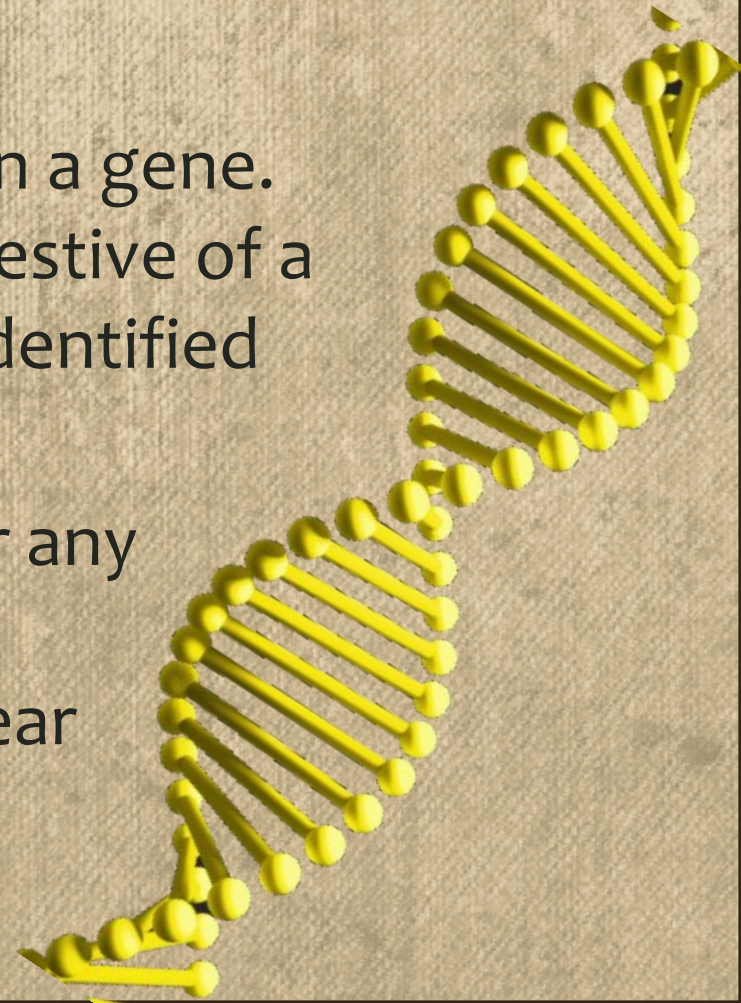
# Retinal Lesions as a Genetic Marker

- Retinal lesions are a sensitive and specific marker for FAP mutation carrier status
- Congenital retinal lesions may serve as an early marker for those patients destined to develop polyposis later in life
  - Onset of retinal lesions: Birth
  - Onset of polyposis: Age 25 yrs



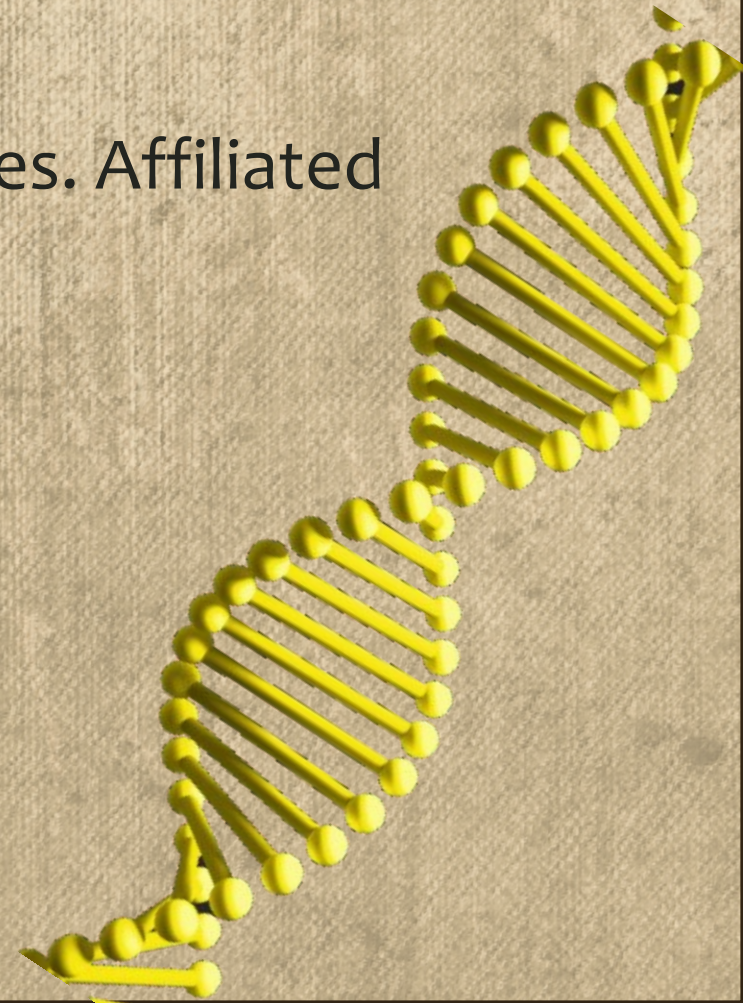
# About Genetic Testing

- Two basic modes of testing
  - Targeted testing: Look for a specific variant in a gene. Helpful in patients with clinical findings suggestive of a disorder whose causative genes have been identified (e.g. dystrophies)
  - Full gene sequencing: Genes are analyzed for any variation from normal. Useful when the suspected condition or genetic cause is unclear



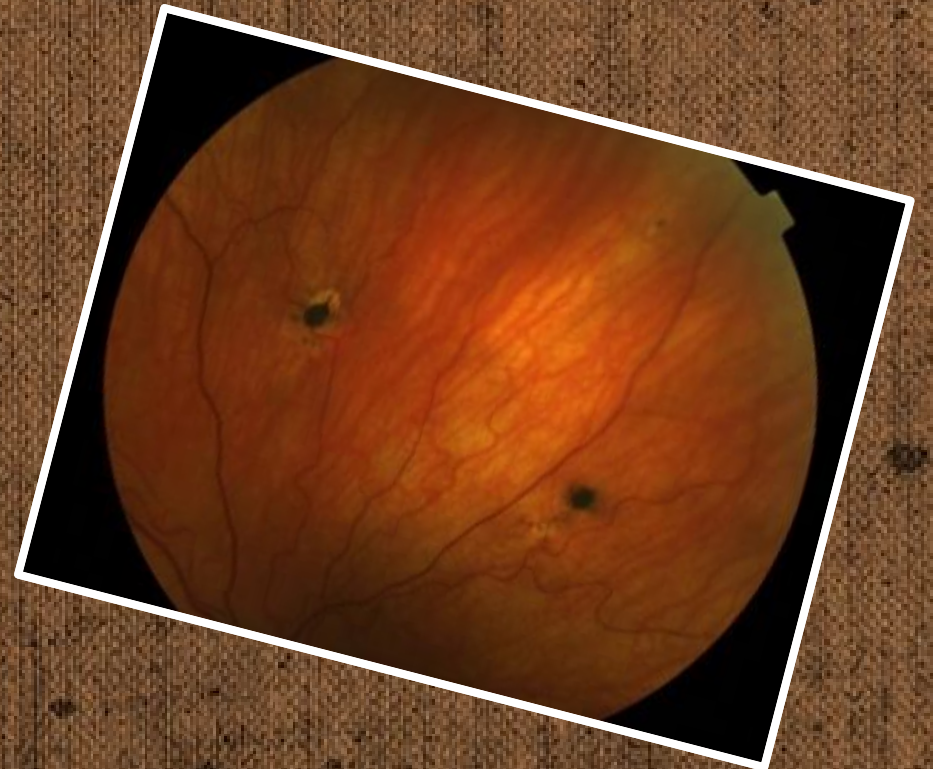
# About Genetic Testing

- Free targeted testing
  - [carverlab.org](http://carverlab.org): Retinal and optic nerve diseases. Affiliated with University of Iowa
  - [fightingblindness.org](http://fightingblindness.org): Retinal diseases only
  - [invitae.com](http://invitae.com): Retinal diseases only
- Paid full gene sequencing
  - NIH Genetic Testing Registry: Search for available conditions, tests and labs



# Take Home Message

- CHRPE-like lesions may signal risk of colon cancer
- How to spot suspicious CHRPE-like lesions
  1. **Bilaterality!**
  2. > 1 quadrant per eye
  3. Lesions associated with a depigmented streak
- Consider genetic testing in patients suspected of having an inherited disorder



# CASE #8

*Jagged little pill*

## Case #8

---

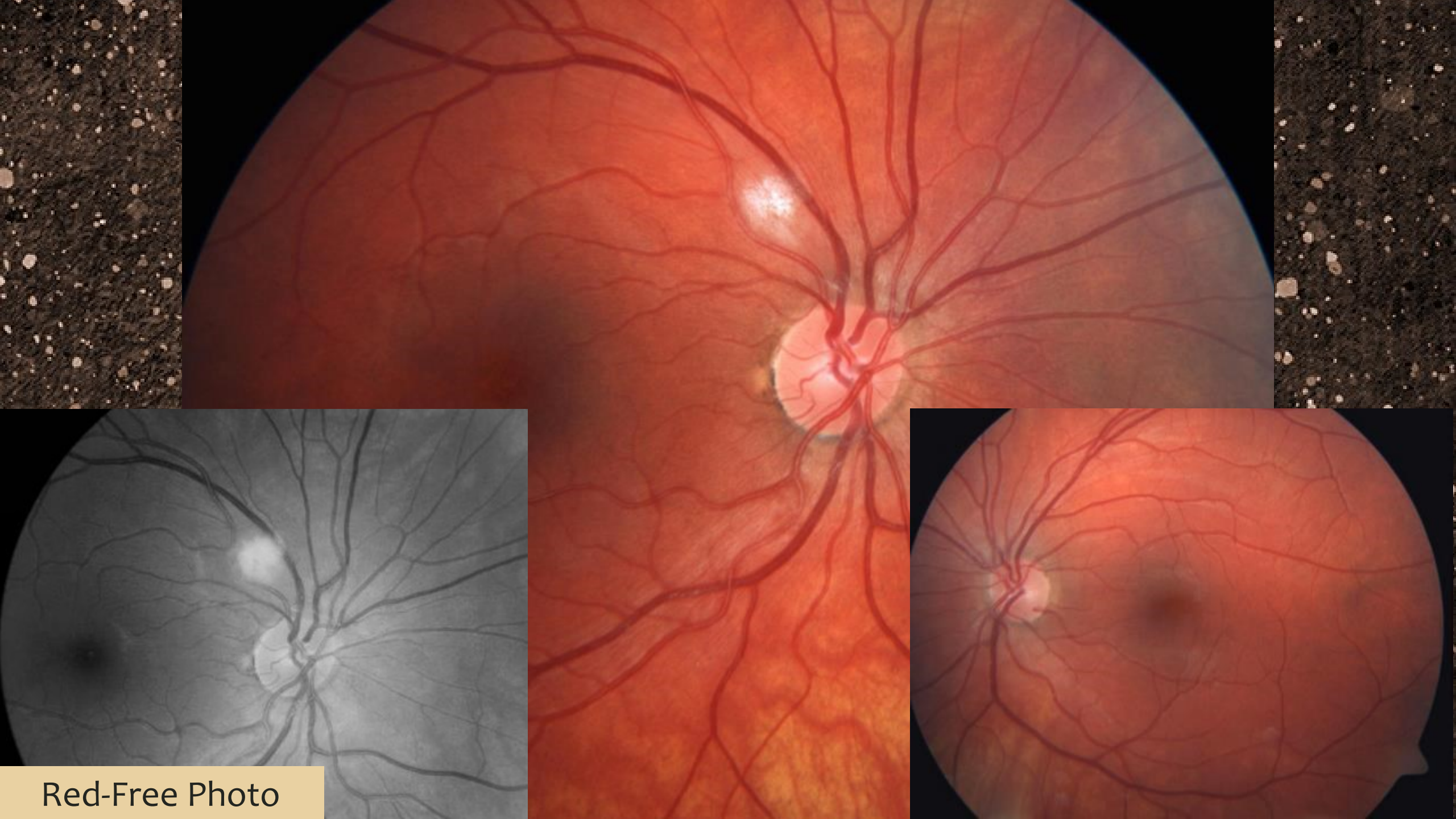
- 24yo WF presents with c/o “blurry light spot” in the *inferior-nasal* paracentral vision of the right eye x 1 week. Symptoms are made worse while exercising.
- POH: LEE 2yrs. Negative for any prior eye dx
- MH: Good health. Nonsmoker. Meds: BCP
- FH: MGM with diabetes

# Case #8

---

- BVA: 20/20 in each eye
- Pupils and motility: Normal
- BP: 113/71 RAS, Pulse: 81 bpm
- IOP: 13/12 mmHg @ 4pm
- Amsler: Blurred region *inferior-temporal* to fixation OD, Normal OS
- Color: Normal OU (HRR)
- External: Normal OU

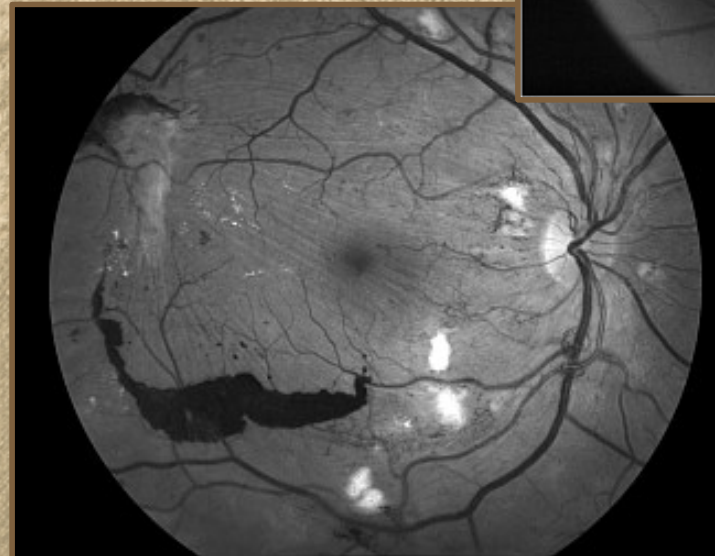
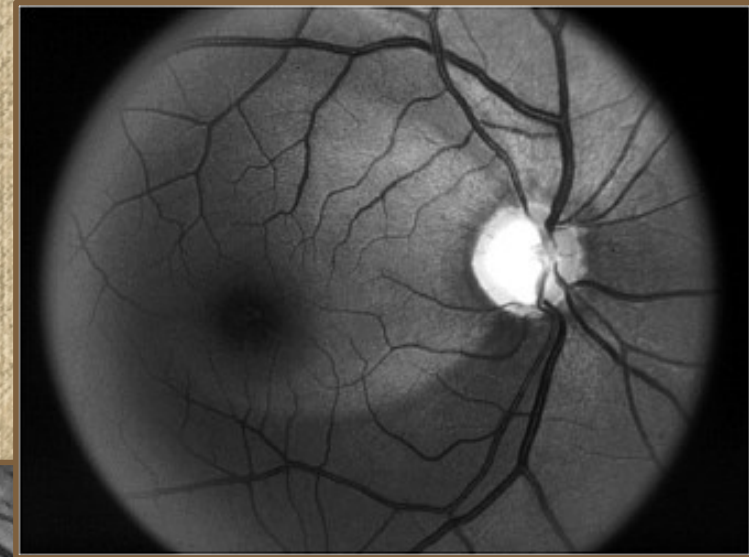


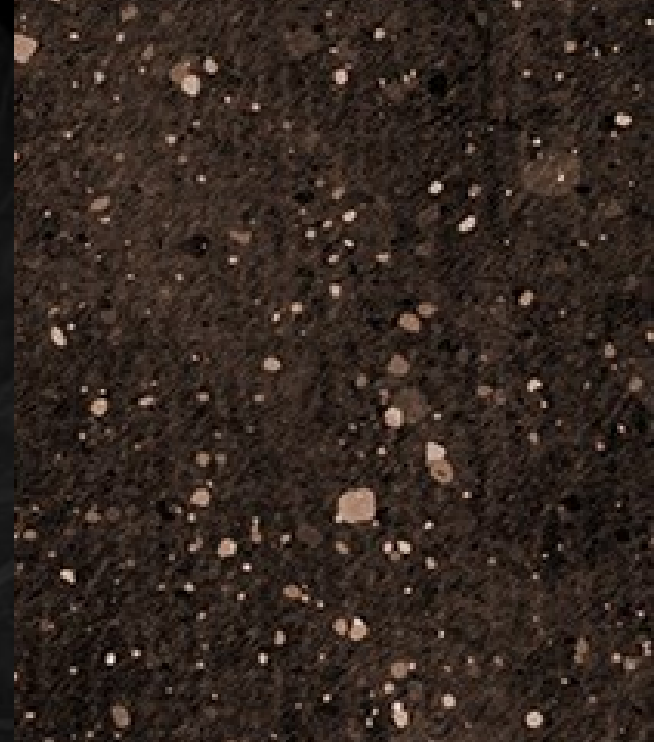
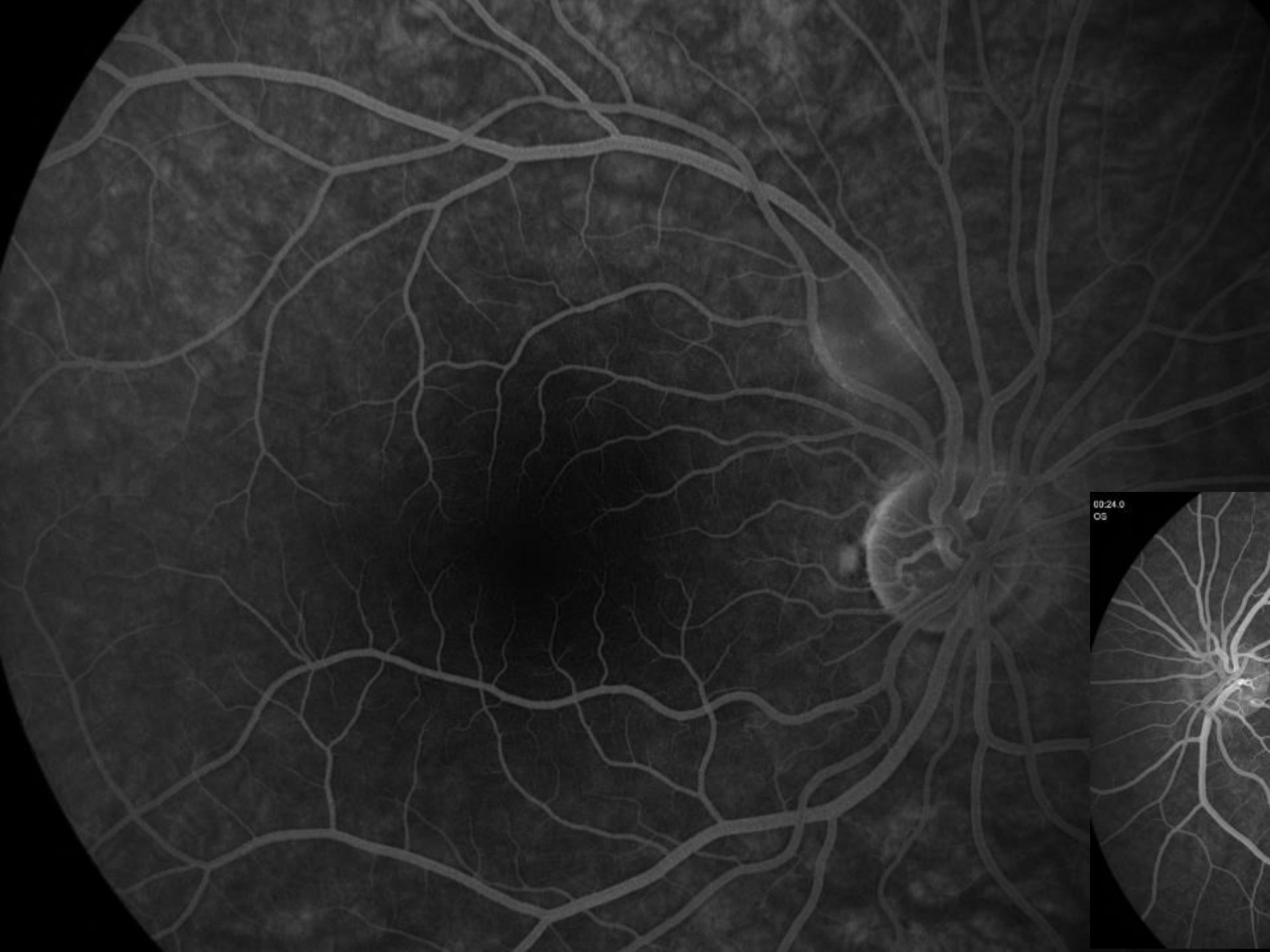


Red-Free Photo

# About Red-free Fundus Photography

- Uses green filter (540nm)
- Green light is absorbed by blood and does not penetrate RPE
- Why use it?
  - Visualization of blood and RNFL
  - **Doctors with abnormal color vision**





00:24.0  
C5

SINGLE FIELD ANALYSIS

EYE: RIGHT

NAME:

DOB: 03-18-1987

ID:

CENTRAL 24-2 THRESHOLD TEST

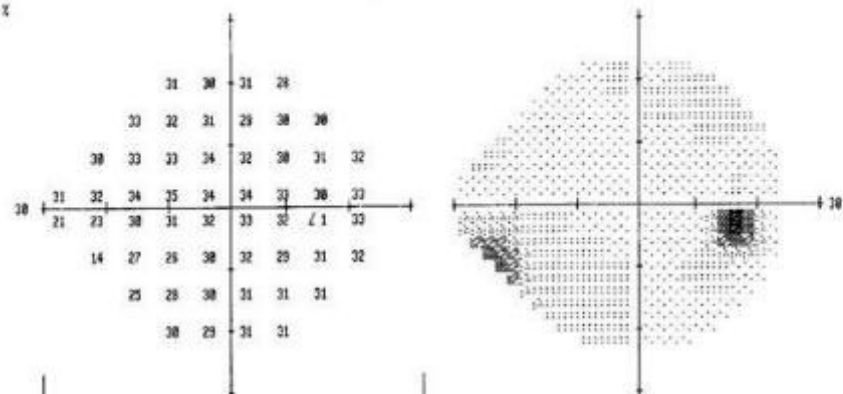
FIXATION MONITOR: GAZE/BLEND SPOT  
 FIXATION TARGET: CENTRAL  
 FIXATION LOSSES: 1/10  
 FALSE POS ERRORS: 0 X  
 FALSE NEG ERRORS: 0 X  
 TEST DURATION: 00:11

STIMULUS: III, WHITE  
 BACKGROUND: 31.5 ASB  
 STRATEGY: S10-F4ST

PUPIL DIAMETER: 7.7 MM  
 VISUAL ACUITY:  
 RX: -0.50 DS DC X

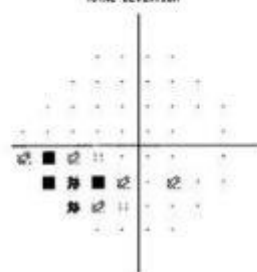
DATE: 07-22-2011  
 TIME: 2:31 PM  
 AGE: 24

FOVER: OFF



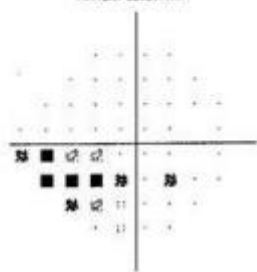
1	1	1	-1				
1	0	-1	-3	-2	-1		
-1	0	0	0	-1	-3	-1	0
1	0	1	0	-1	-1	-1	1
-3	-5	-4	-3	-2	-1	-2	1
-17	-6	-8	-4	-2	-5	-2	0
-5	-4	-3	-2	-2	-1		
-1	-3	-1	-1				

TOTAL DEVIATION



0	0	1	-2				
1	-1	-2	-4	-2	-2		
-2	0	0	0	-2	-3	-2	-1
1	-1	0	0	-2	-1	-2	0
-5	-3	-4	-4	-3	-2	-2	0
-18	-6	-8	-4	-3	-5	-2	-1
-7	-5	-4	-3	-2	-2		
-2	-3	-2	-1				

PATTERN DEVIATION



11 < 50  
 12 < 25  
 18 < 15  
 24 < 0.5%

GHT  
 OUTSIDE NORMAL LIMITS  
 WFI 57%  
 MD -2.11 DB P < 5%  
 PSD 3.38 DB P < 1%

ODU SCHOOL OF OPTOMETRY  
 5725 DATAPoint DRIVE  
 SAN ANTONIO, TX  
 78229

SINGLE FIELD ANALYSIS

EYE: LEFT

NAME:

DOB: 03-18-1987

ID:

CENTRAL 24-2 THRESHOLD TEST

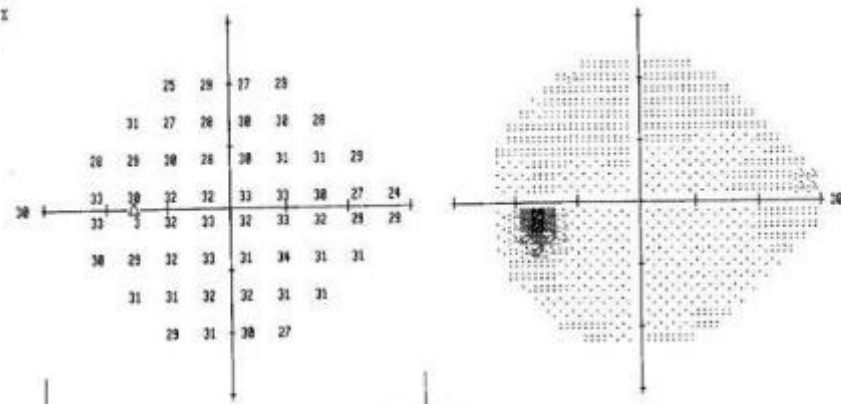
FIXATION MONITOR: GAZE/BLEND SPOT  
 FIXATION TARGET: CENTRAL  
 FIXATION LOSSES: 2/11  
 FALSE POS ERRORS: 4 X  
 FALSE NEG ERRORS: 0 X  
 TEST DURATION: 00:13

STIMULUS: III, WHITE  
 BACKGROUND: 31.5 ASB  
 STRATEGY: S10-F4ST

PUPIL DIAMETER: 7.4 MM  
 VISUAL ACUITY:  
 RX: 00 DC X

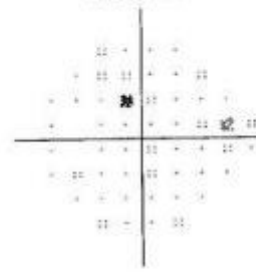
DATE: 07-22-2011  
 TIME: 2:38 PM  
 AGE: 24

FOVER: OFF



-5	-1	-3	-1				
0	-4	-4	-2	-2	-4		
-3	-3	-3	-5	-4	-2	-2	
1	-2	-2	-2	-2	-3	-5	-6
1	-2	-1	-3	-1	-1	-3	-1
-2	-4	-1	-1	-3	0	-2	-1
-1	-1	-1	-1	-2	-1		
-3	-1	-1	-3				

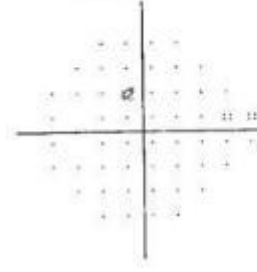
TOTAL DEVIATION



11 < 50  
 12 < 25  
 18 < 15  
 24 < 0.5%

-4	0	-2	0				
1	-3	-3	-1	-1	-3		
-2	-2	-2	-4	-3	-1	-1	-2
2	-1	-1	-1	-1	-2	-4	-5
2	-1	0	-2	0	0	-2	0
-1	-3	-1	0	-2	1	-1	0
0	-1	0	0	-1	0		
-2	0	-1	-3				

PATTERN DEVIATION



GHT  
 WITHIN NORMAL LIMITS  
 WFI 90%  
 MD -2.04 DB P < 5%  
 PSD 1.45 DB

ODU SCHOOL OF OPTOMETRY  
 5725 DATAPoint DRIVE  
 SAN ANTONIO, TX  
 78229

# Assessment

---

- Isolated cotton wool spot in an apparently healthy young woman

# Management

---

- Medical evaluation to identify cause for CWS
- Recommend D/C BCP and start ASA
- Follow-up in 2 weeks

# What is going on here?



<https://app.tophat.com/e/777538>

Idiopathic cotton wool spot

Undiagnosed diabetes

Undiagnosed HIV

Undiagnosed NTG

BRAO



## Idiopathic CWS

It has been reported that an underlying disorder can be found in 95% of patients with isolated CWS

## Diabetes

Undiagnosed diabetes is the **most common** cause of isolated CWS in an apparently normal patient

## HIV

CWS are a prominent feature of HIV noninfectious retinopathy, the prevalence of which is inversely related to the patient's CD4+ count

## NTG

Cotton wool spots are not associated with NTG

## BRAO

CWS are a universal feature of BRAO

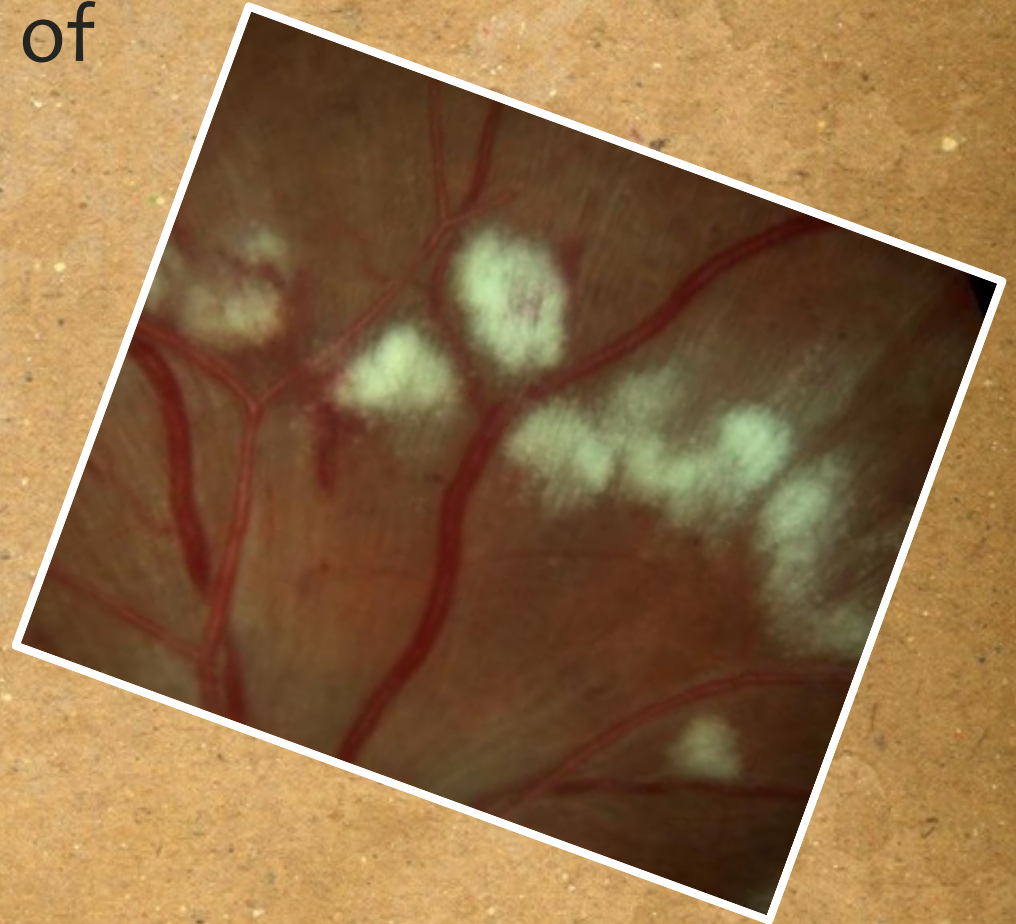
# Medical Evaluation

- Physical exam by PCP was normal
- Normal laboratory testing: Fasting glucose, CBC, ANA, Rheumatoid factor, C-reactive protein, HIV screen
- Normal carotid Doppler and echocardiogram
- FTA-ABS was minimally reactive
  - Serologic ELISA testing for Lyme disease recommended
- Follow-up: Photopsia persisted x 4-6 wks before abating. VF defect remained unchanged

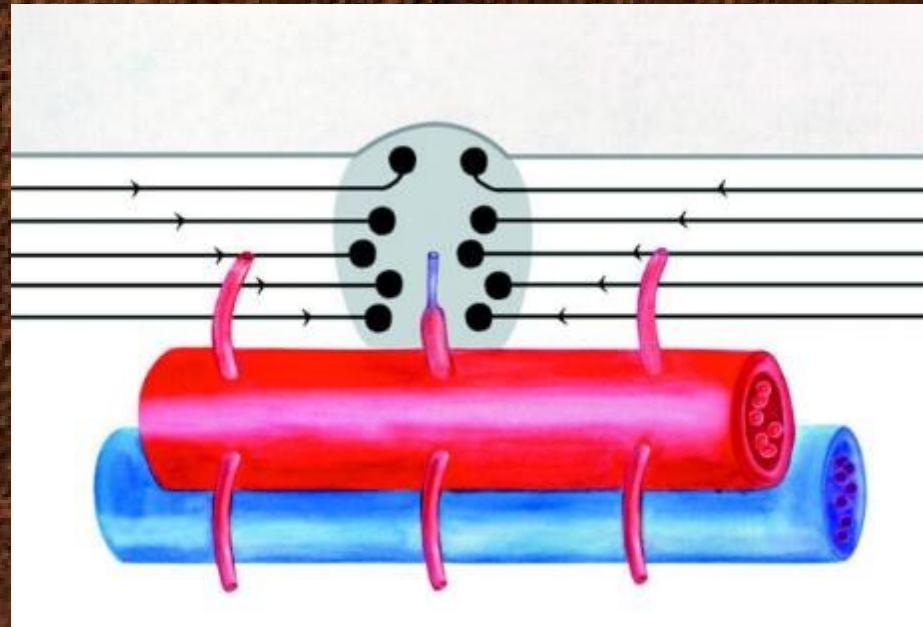


# What is a CWS?

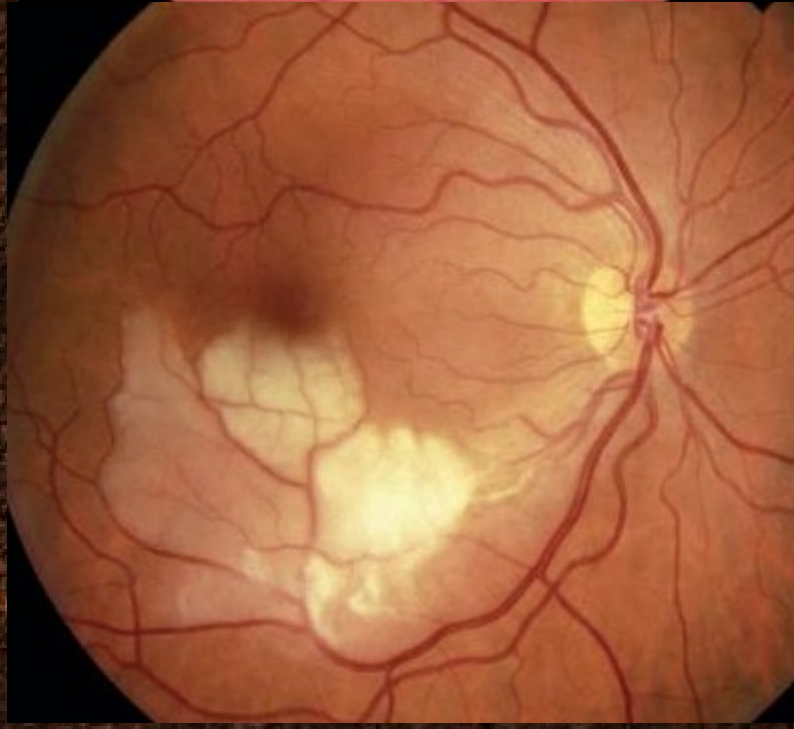
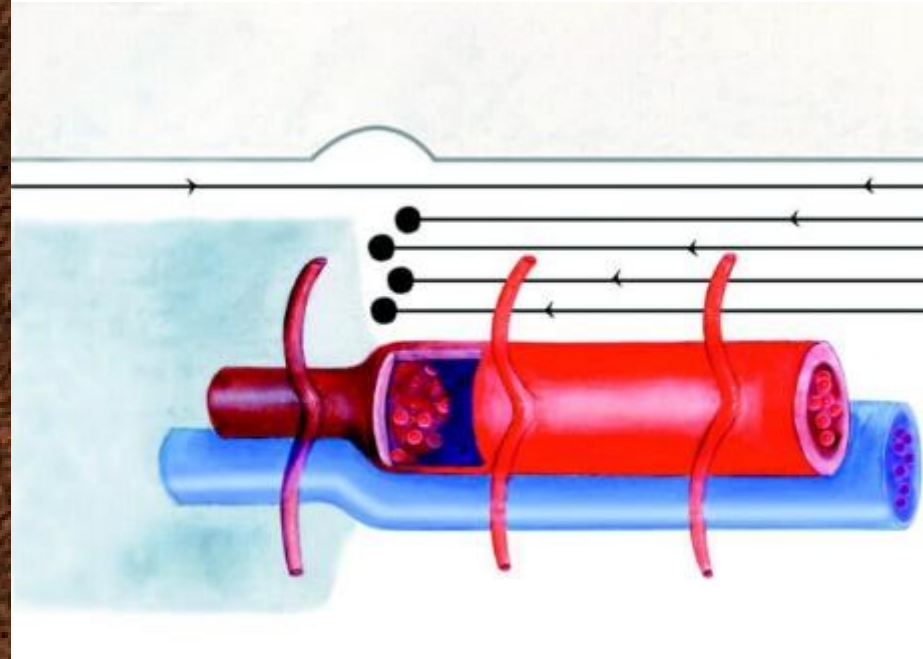
- CWS is a localized accumulations of axoplasmic material within adjacent bundles of ganglion cell axons.
- Two clinical presentations:
  1. Focal ischemia from terminal arteriolar occlusion.
  2. Appearance at the boundary of an ischemic region of the retina



Focal ischemia: Occlusion of a terminal branch of a retinal arteriole results in a small area of infarction (grey) in the RNFL where axoplasmic transport is obstructed.



Sentinal lesion: Occlusion of a retinal arteriole results in retinal infarction



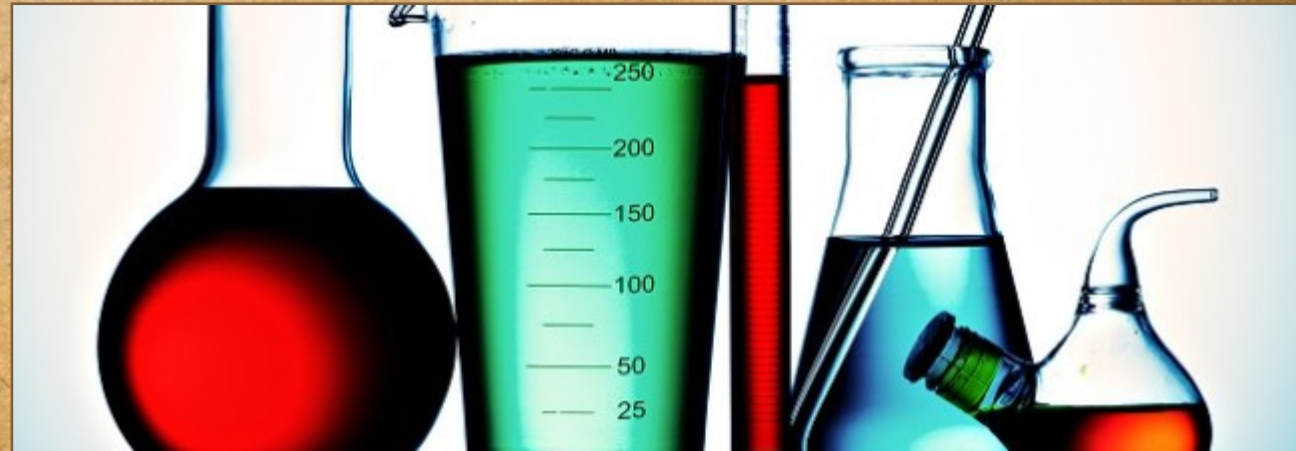
# Vision Loss Associated with CWS

- CWS are almost always asymptomatic
- Localized or arcuate scotomas common
- OCT studies reveal permanent loss of the inner retinal layers at the site of resolved CWS lesions
- Some visual recovery may occur following resolution of CWS lesions



# Evaluation of Idiopathic CWS

- Search for conditions that predispose the patient toward embolism and thrombosis
- Common: **Diabetes**, hypertension, and collagen vascular disease
- Less common: HIV and other infections, hematologic disease and coagulopathies, pancreatitis, embolic disease, trauma, and pregnancy



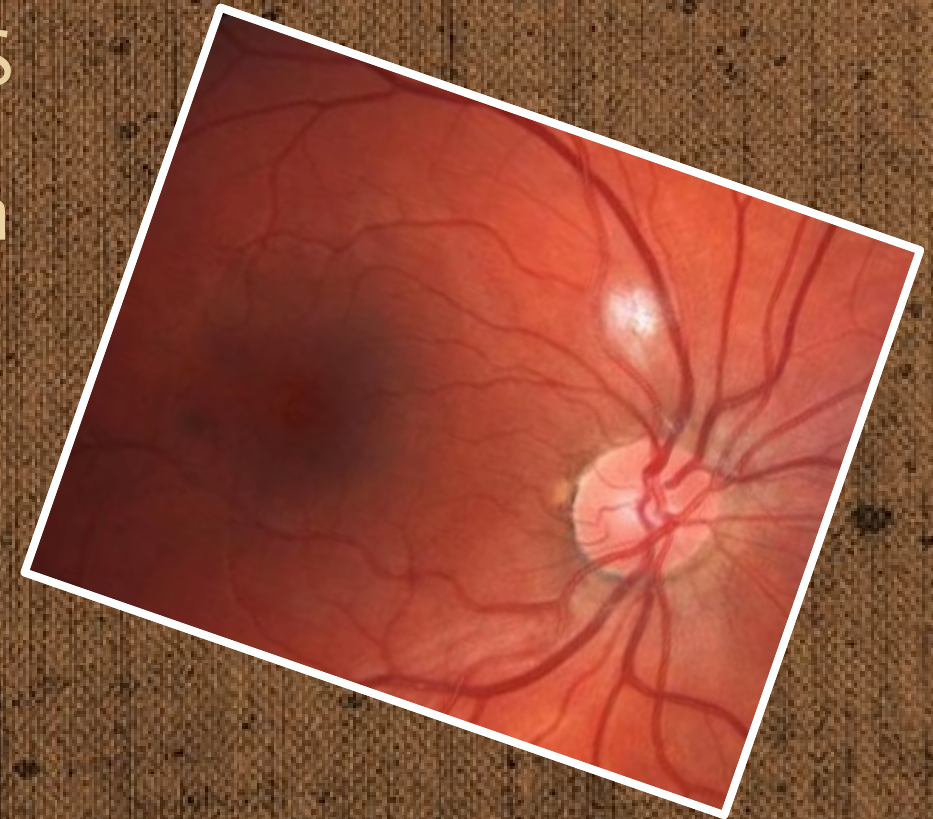
# OCP and Thromboembolism

- Birth control pills increase the risk of venous thromboembolism (VTE) by about 5x
  - Additional risk factors: **smoking**, obesity, HTN, coagulopathies, and a FH of thromboembolic disease
- Third & fourth generation OCPs
  - Lower risk of many side effects (weight gain, acne, headaches and unwanted hair growth)
  - **Risk of thrombosis higher than second generation pills**



# Take Home Message

- Isolated CWS are not truly isolated
- Diabetes, hypertension, and collagen vascular disease are common causes of CWS
- CWS are frequently associated with permanent VF defects
- Birth control pills are a significant risk factor for vascular occlusions in young healthy women



# CASE #9

*Hair of the Dog*

## Case #9

- 69yo WF presents for routine eye exam.
  - POH: LEE 1yr. Negative for any prior eye dx
  - MH: Good health
  
  - VA: 20/25+ OD, 20/20 OS
  - PERRL, (-)APD
  - GAT: 27/28 @ 12pm
  - SLE: White & Quiet
- ← ↑10 mmHg  
from 1yr ago



# Case #9

---

- C/D: 0.6 OD, 0.5 OS
- Mild ERM OD
- Gonio: D4of OU
- CCT: 572 OD, 576 OS
- IMP: Glaucoma suspect
- PLAN: Schedule OCT & VF

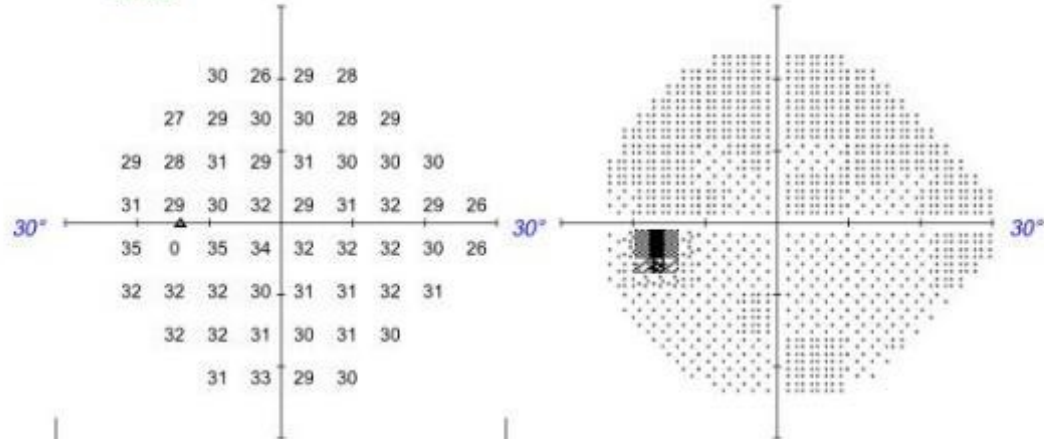
Fundus photos taken with OPTOS camera



Fixation Monitor: Gaze/Blind Spot  
 Fixation Target: Central  
 Fixation Losses: 7/15 XX  
 False POS Errors: 3%  
 False NEG Errors: 0%  
 Test Duration: 05:16  
 Fovea: 34 dB

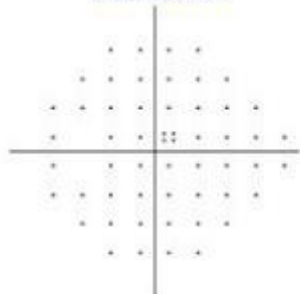
Stimulus: III, White  
 Background: 31.5 asb  
 Strategy: SITA Standard  
 Pupil Diameter: 4.6 mm \*  
 Visual Acuity:  
 Rx: +1.50 DS

Date: Dec 07, 2021  
 Time: 10:43 AM  
 Age: 69



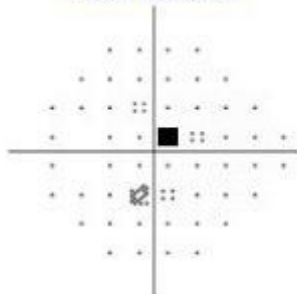
4	0	2	2				
-1	1	1	-1	1			
1	-1	1	-1	0	-1	1	2
2	-1	1	-3	-1	1	0	0
6	3	2	0	0	2	1	0
3	2	1	-2	-1	0	1	3
2	2	1	-1	1	1		
2	3	0	2				

Total Deviation



2	-2	0	-1				
-3	-1	-1	-1	-3	-1		
-1	-3	-2	-4	-2	-3	-2	0
0	-4	-2	-5	-3	-1	-2	-2
3	1	-1	-2	-2	-1	-1	-2
0	-1	-1	-4	-4	-2	-1	0
0	0	-2	-3	-2	-1		
0	1	-2	-1				

Pattern Deviation



GHT: **Borderline**

VFI24-2: **99%**  
 MD24-2: **0.78 dB**  
 PSD24-2: **1.65 dB**

\*\*\* Low Test Reliability \*\*\*

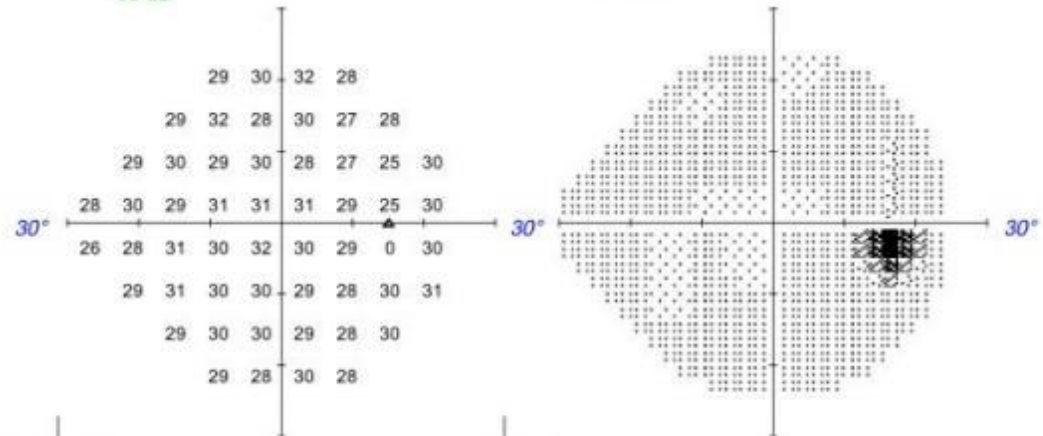
- :: P < 5%
- ⊗ P < 2%
- ⊗ P < 1%
- P < 0.5%



Fixation Monitor: Gaze/Blind Spot  
 Fixation Target: Central  
 Fixation Losses: 1/14  
 False POS Errors: 2%  
 False NEG Errors: 0%  
 Test Duration: 04:53  
 Fovea: 33 dB

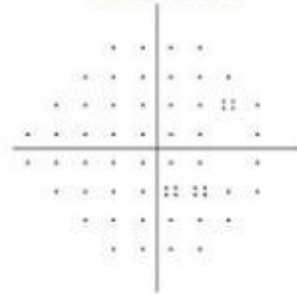
Stimulus: III, White  
 Background: 31.5 asb  
 Strategy: SITA Standard  
 Pupil Diameter: 5.3 mm \*  
 Visual Acuity:  
 Rx: +2.75 DS -1.25 DC X 70

Date: Dec 07, 2021  
 Time: 10:31 AM  
 Age: 69



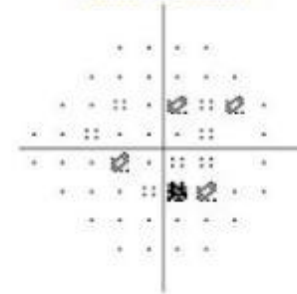
3	4	6	3				
1	3	-1	1	-1	1		
1	1	-1	-1	-2	-3	-4	2
2	1	-2	-1	-1	0	-2	1
0	-1	0	-2	0	-2	-2	1
1	1	-1	-1	-3	-3	0	2
0	0	-1	-1	-2	0		
1	-1	1	-1				

Total Deviation



1	2	4	1				
-1	1	-3	-1	-3	-1		
-1	-1	-3	-3	-4	-5	-6	0
0	-1	-4	-3	-3	-2	-4	-1
-2	-3	-2	-4	-2	-4	-4	-1
-1	-1	-3	-3	-5	-5	-2	0
-2	-2	-3	-3	-4	-2		
-1	-3	-1	-3				

Pattern Deviation



GHT: **Borderline**

VFI24-2: **98%**  
 MD24-2: **-0.44 dB**  
 PSD24-2: **1.74 dB**

- :: P < 5%
- ⊗ P < 2%
- ⊗ P < 1%
- P < 0.5%

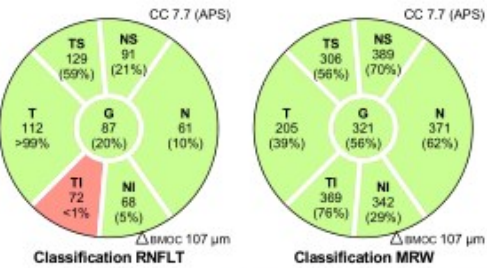
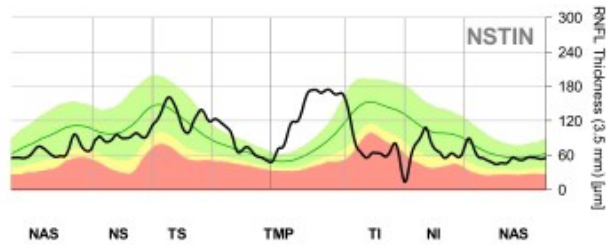
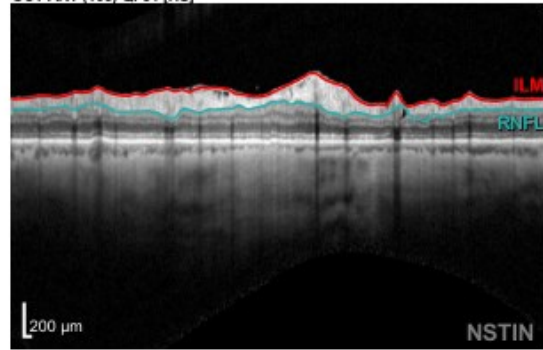


# Hood report reveals effect of ERM OD

OD

OS

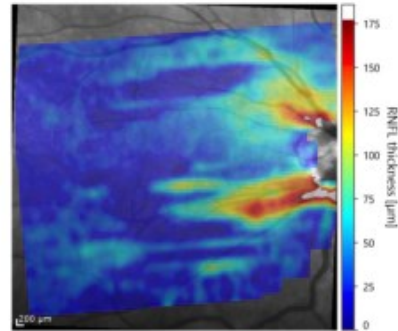
OCT ART (103) Q: 31 [HS]



Within Normal Limits (>5%)  
 Borderline (<5%)  
 Outside Normal Limits (<1%)

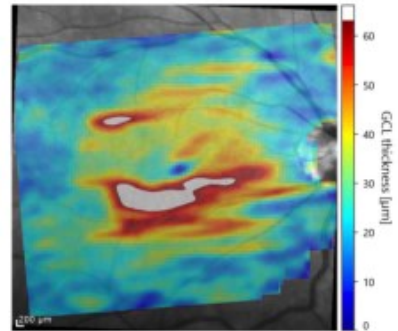
Retina view

Superior retina



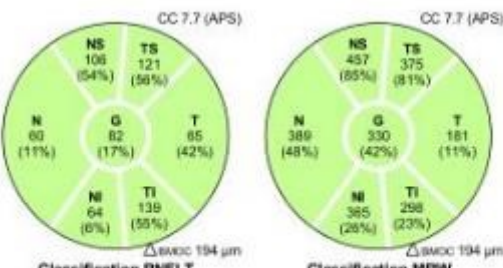
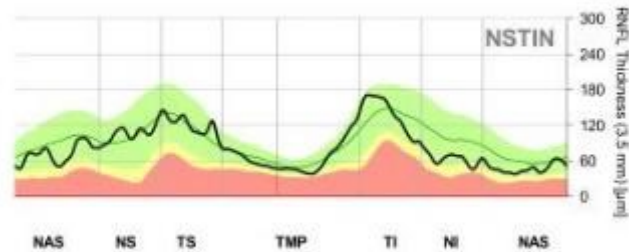
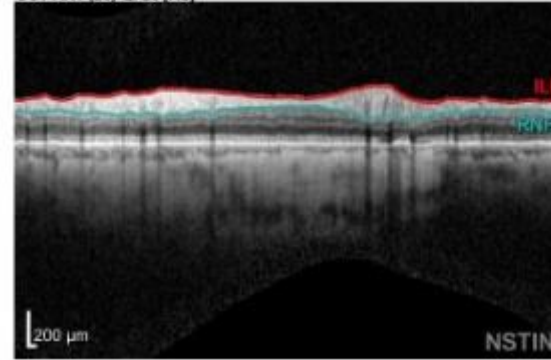
Inferior retina

Superior retina



Inferior retina

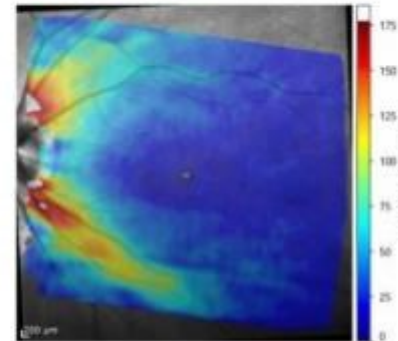
OCT ART (26) Q: 26 [HS]



Within Normal Limits (>5%)  
 Borderline (<5%)  
 Outside Normal Limits (<1%)

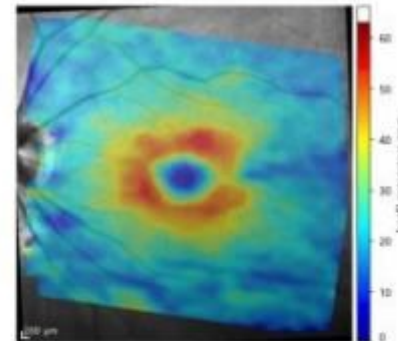
Retina view

Superior retina



Inferior retina

Superior retina



Inferior retina

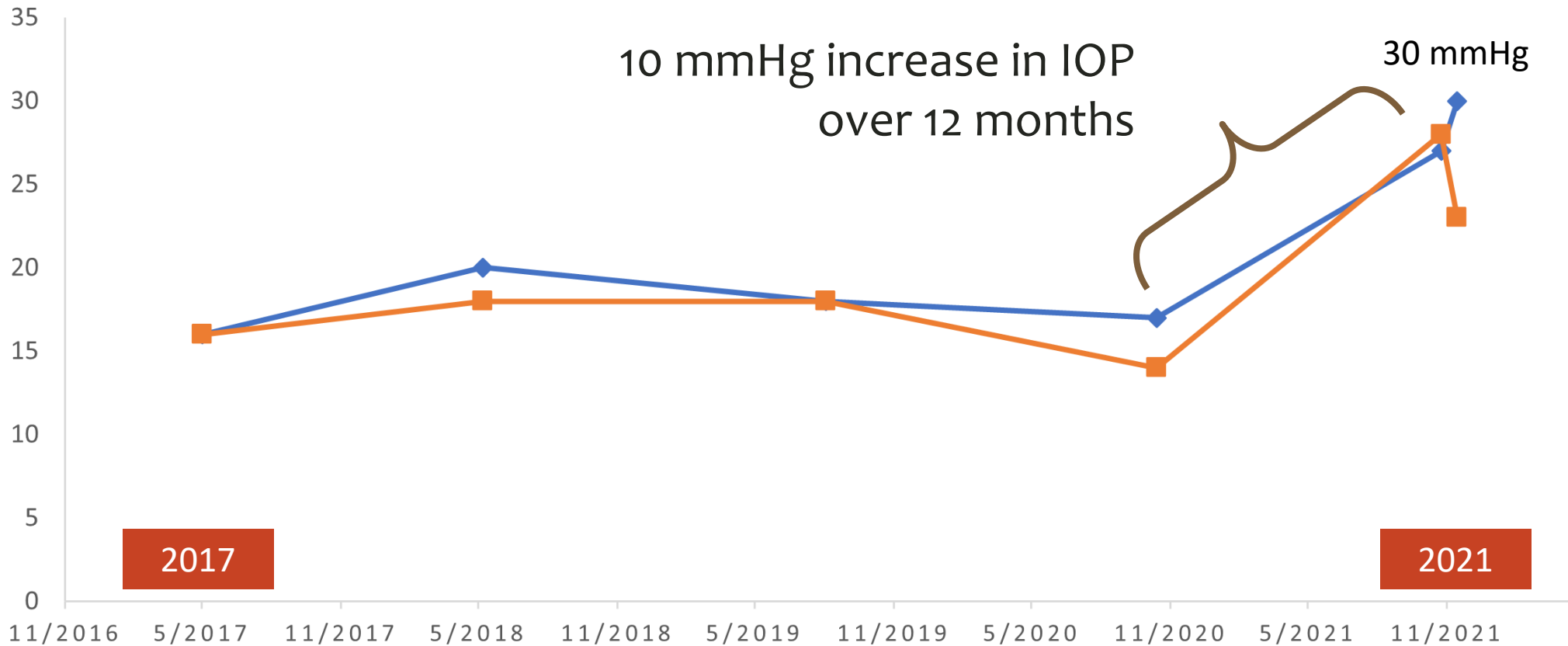
# IOP PROFILE

—◆— OD —■— OS

10 mmHg increase in IOP  
over 12 months

30 mmHg

IOP (mmHg)



	5/24/2017	5/30/2018	8/26/2019	11/5/2020	11/16/2021	12/7/2021
OD	16	20	18	17	27	30
OS	16	18	18	14	28	23

# What is going on here?



<https://app.tophat.com/e/777538>

Angle Closure

Start on steroid medication

Previously undetected large diurnal variation

Discontinuation of systemic beta blocker

Influence of other drugs or activities



<b>Angle closure</b>	Always suspect angle closure in patients with sudden changes in IOP
<b>Steroid</b>	Any steroid by any route of administration can elevate IOP
<b>Diurnal variation</b>	Normal: 2-6 mmHg. Checking IOP on another day, Water drinking test or iCare HOME to investigate
<b>Beta blocker</b>	Systemic beta blockers can affect IOP same as topical
<b>Other factors</b>	Caffeine, ethanol, marijuana, exercise

BEFORE



AFTER 16 WEEKS



Source: [lattise.com](http://lattise.com)

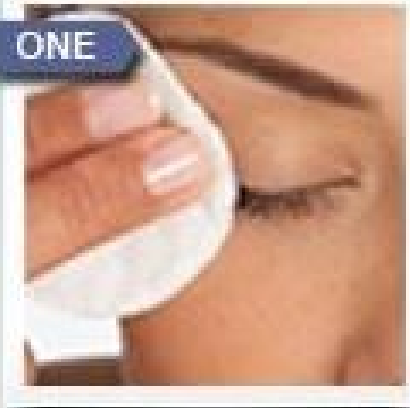


# Latisse

- Latisse == Generic Lumigan == bimatoprost 0.03%
- Latisse has all the same clinical effects as Lumigan
- Adverse effects with Latisse:  
conjunctival hyperemia and irritation,  
increase in iris pigmentation,  
periocular skin pigmentation,  
and periorbital fat atrophy



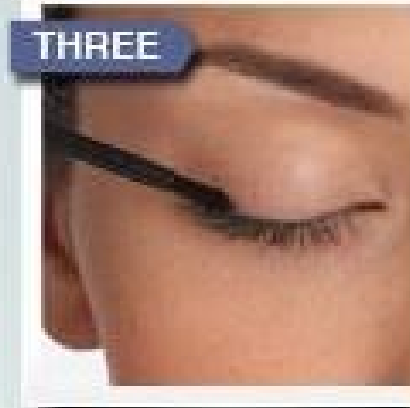
Latisse is applied to the upper lid only.  
Excess solution is immediately removed  
to minimize risk of periorbitopathy



Once nightly, start by ensuring your face is clean, makeup and contact lenses are removed.



Remove an applicator from its tray. Then, holding the sterile applicator horizontally, place one drop of LATISSE<sup>®</sup> on the area of the applicator closest to the tip but not on the tip.



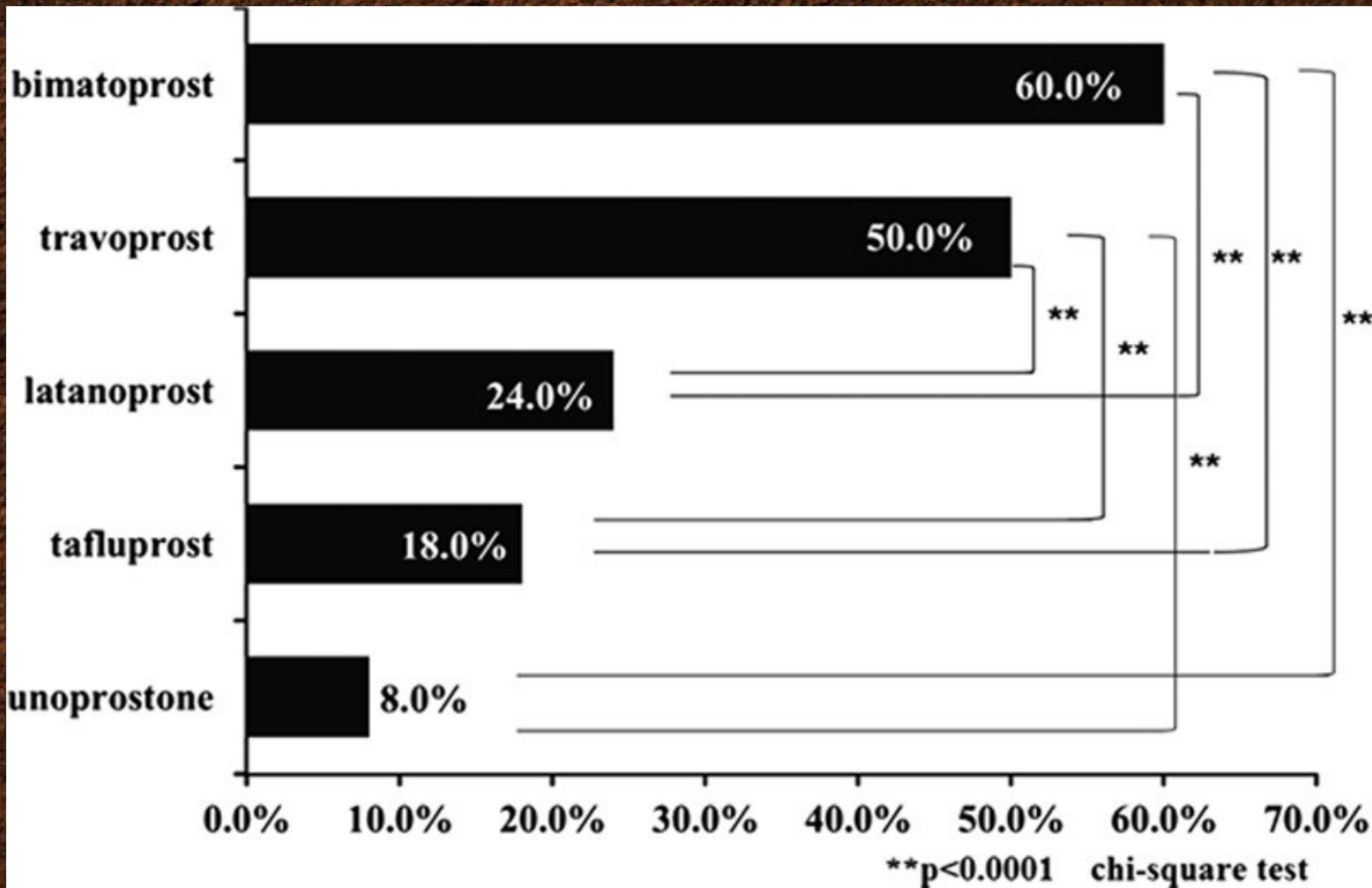
Then immediately draw the applicator carefully across the skin of the upper eyelid margin at the base of the eyelashes (where the eyelashes meet the skin) going from the inner part of your lash line to the outer part.



Blot any excess solution beyond the eyelid margin. If the solution gets into the eye, it is not expected to cause harm. The eye should not be rinsed.



Dispose of the applicator after one use. Repeat for the opposite upper eyelid margin using a new sterile applicator. This helps minimize any potential for contamination from one eyelid to another.

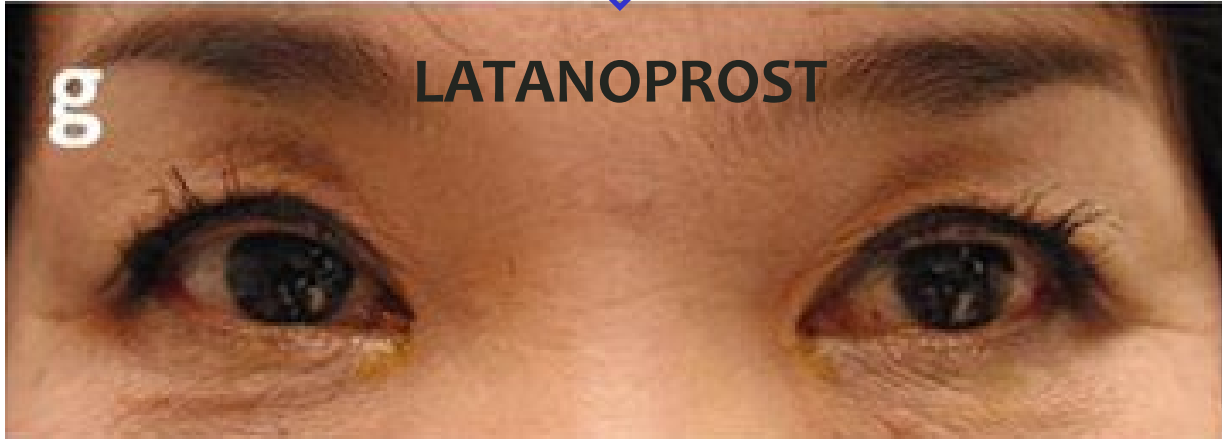


Frequency of upper lid sulcus deepening

There are significant differences in the incidence of PAP among various PGAs

Source: Kenji (2013)

Source: Sakata (2012)



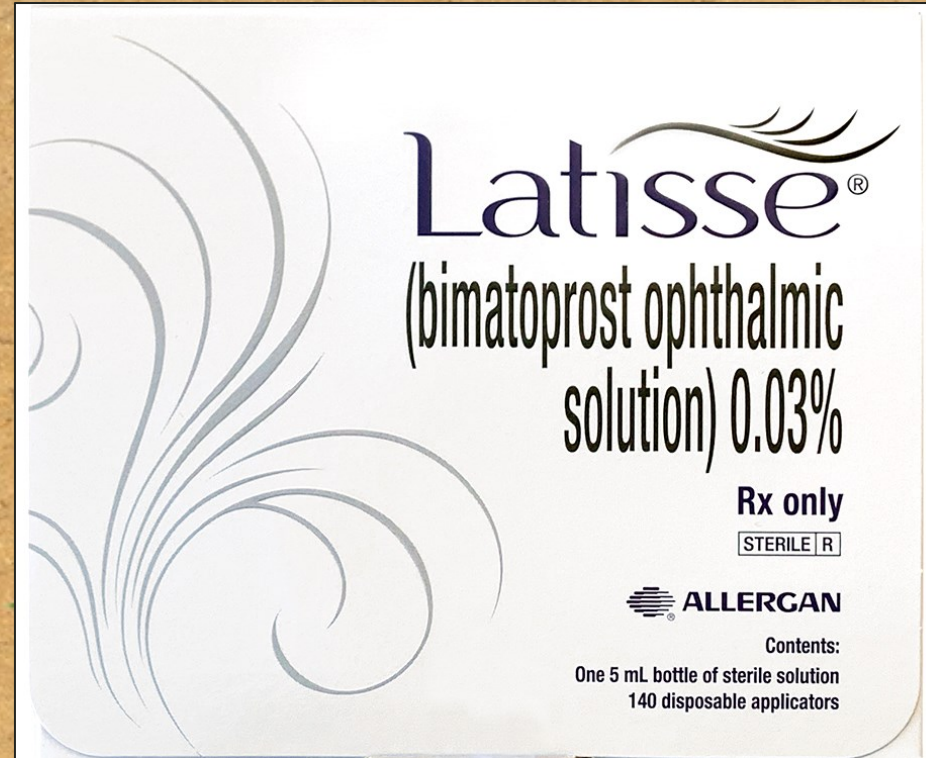
# Generic



Greater IOP lowering  
More side effects  
Less BAK (0.005%)



# Name-Brand



Less IOP lowering  
Fewer side effects  
More BAK (0.05%)



## Periocular discoloration after using a prostaglandin analog for eyelash enhancement: evaluation with reflectance confocal microscopy

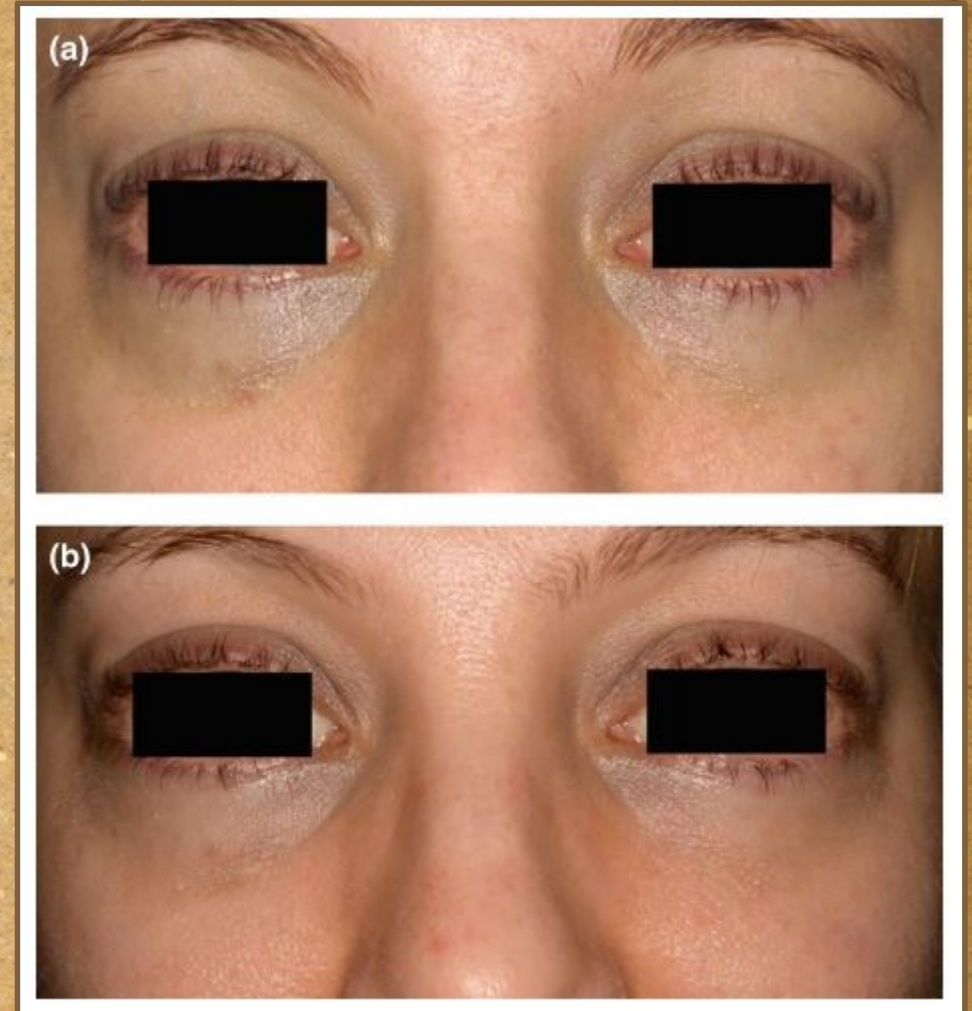
Orsolya N Horváth, MD, Valerie Letulé, MD, Thomas Ruzicka, MD, Thomas Herzinger, MD, Ilana Goldscheider, MD, & Tanja von Braunmühl, MD

*Department of Dermatology and Allergology, Ludwig Maximilian University, Munich, Germany*

**OTC eyelash serums may contain isopropyl cloprostenate, a PGA that can trigger the same periorbital changes seen with bimatoprost.**

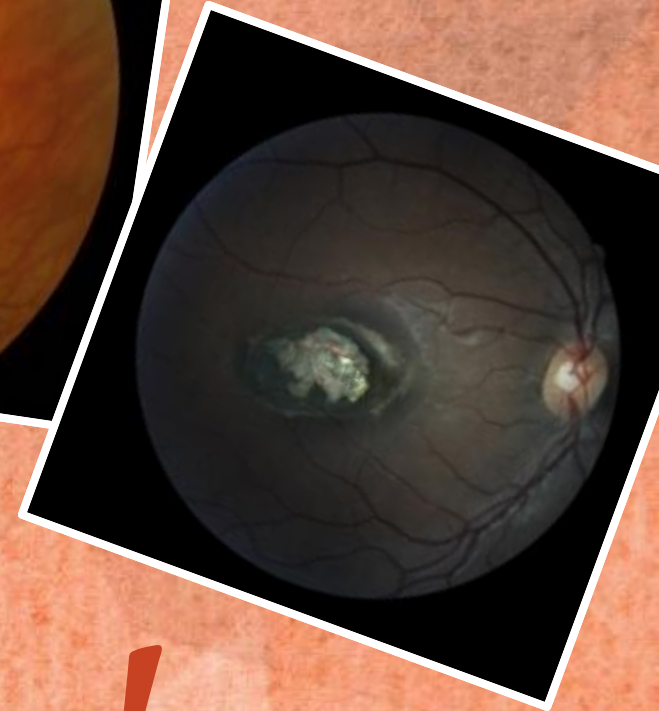
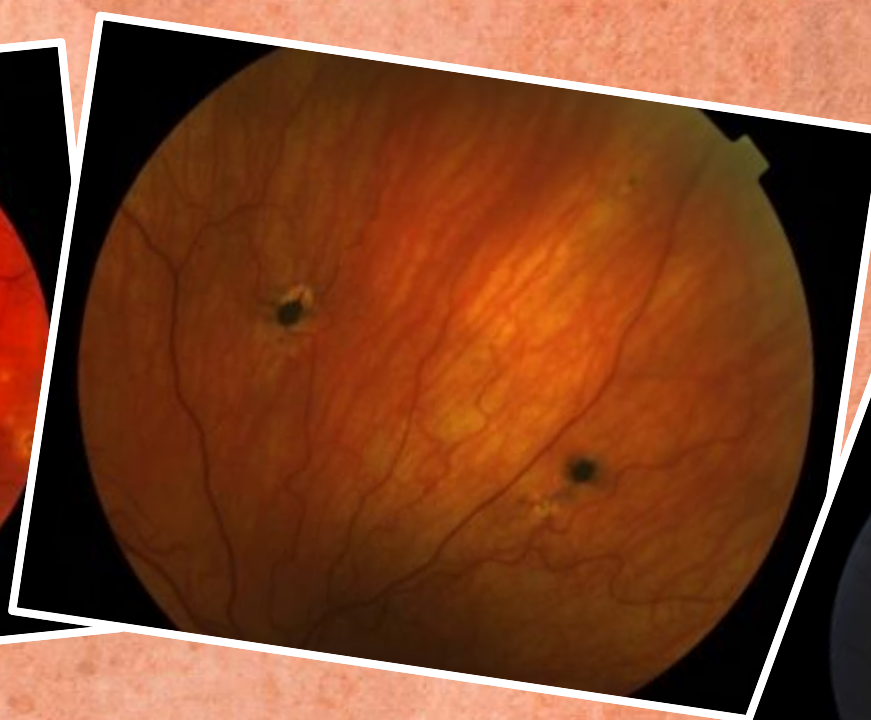
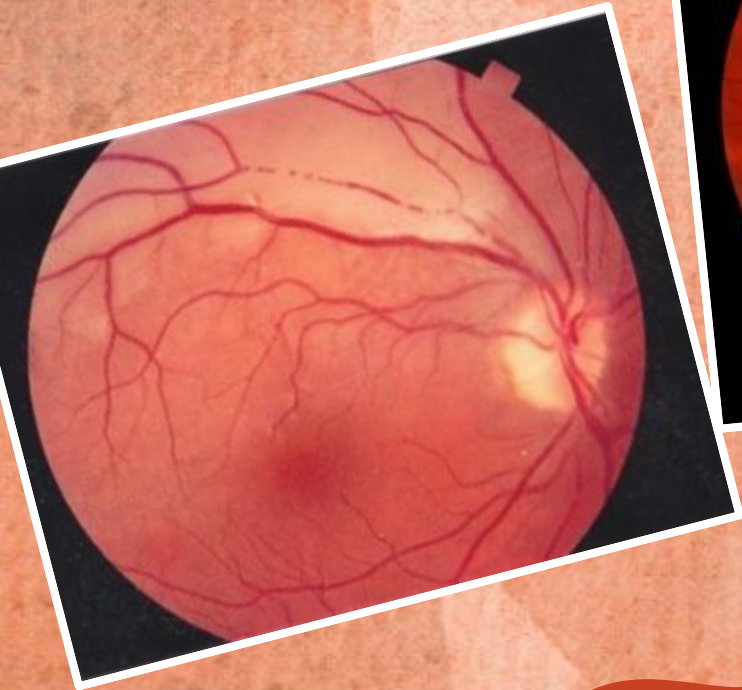
All have been withdrawn by FDA(?)

PMID: 27595988



# Take Home Message

- Latisse can significantly affect IOP
- Bimatoprost is more frequently associated with periorbitopathy than latanoprost
- Periorbitopathy improves following D/C of PGA, but may not fully resolve
- OTC products containing isopropyl cloprostenate can induce periorbitopathy



*Thank You!*