Experience EXPO With Us!

- Conference Happy Hour - *Friday, Sept 29, from 4:30 – 5:30 PM in Room 504-V*

  Kick off the weekend, join us for our Conference Happy Hour! Enjoy complimentary drinks and light snacks with your colleagues before your last course of the day or to simply end your day!

- Innovation Stage - *Exhibit Hall - Focus Neighborhood, Booth F1097*

  Our Innovation Stage sessions feature free, promotional content for all attendees.

- Vision Series - *Thursday, Sept 28 and Friday, Sept 29*

  Grab a bite to eat or drink and continue learning over breakfast or lunch!* Listen to industry leaders as they address the latest clinical innovations in a relaxed and collaborative environment.

  *Open to Optometrists only. Not for Credit. Meals offered on first-come, first-serve basis to pre-registered attendees.

- Exhibit Hall Hours

  Thursday, Sept 28 9:30am – 6:00pm
  Friday, Sept 29 9:30am – 6:00pm
  Saturday, Sept 30 9:30am – 3:00pm

Focus on Retina: Clinical Grand Rounds for Every OD

Mary Beth Yackey, OD

Jeffry Gerson, OD, FAAO

Disclosure

Dr. Yackey

- Iveric Bio
- Notal Vision
- Novartis
- Ocuterra
- Reliance

Dr Gerson

- Allergan
- Apellis
- Bausch Health
- Essilor
- EyePromise
- Iveric Bio
- Luneau
- Notal Vision
- Novartis
- Optos
- Oyster Point
- Regeneron

One of the most common “retina” calls every office gets

- New onset floaters x 5 days
  - No flashes and vision normal
- When will this patient be seen?

20/20 vision

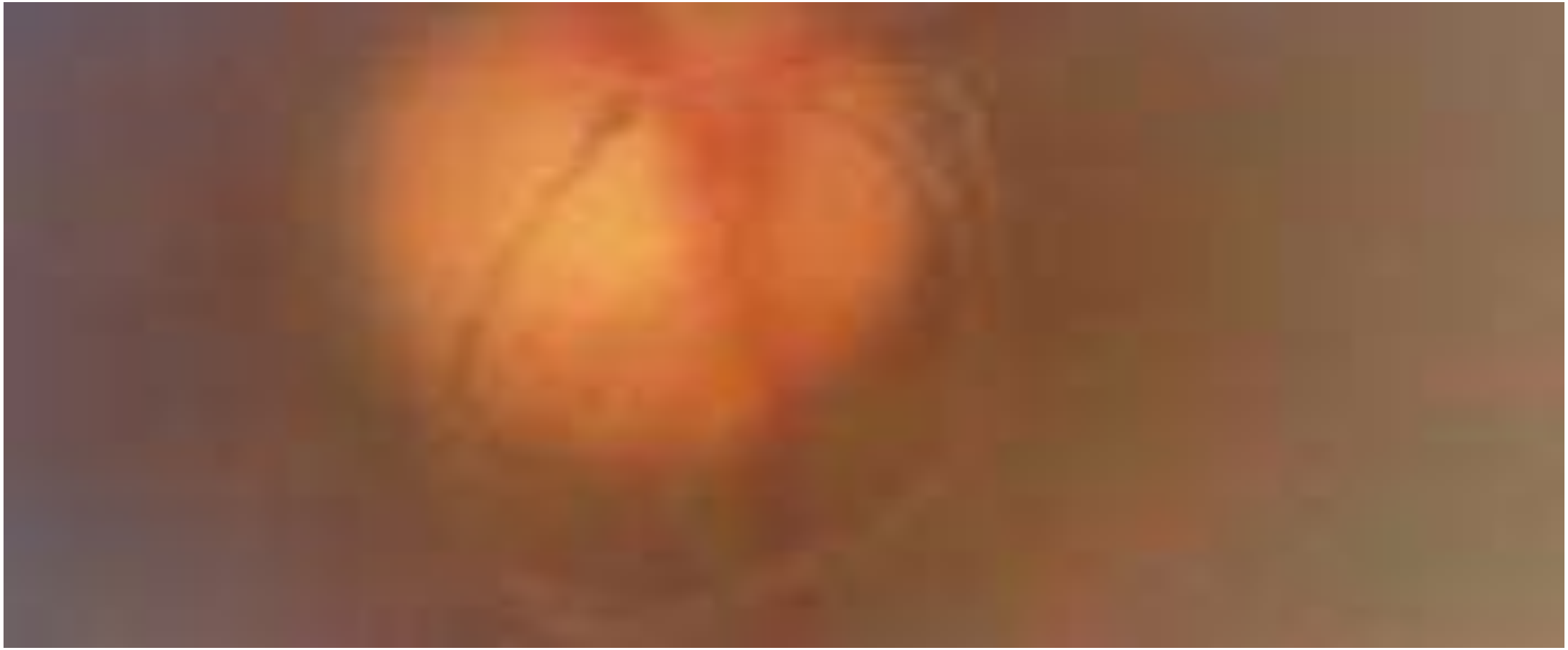
Normal pupils, CVF, motility

Normal anterior segment

Dilated exam performed

??What Drops?
What procedure??

Hello….Mr. Wiess



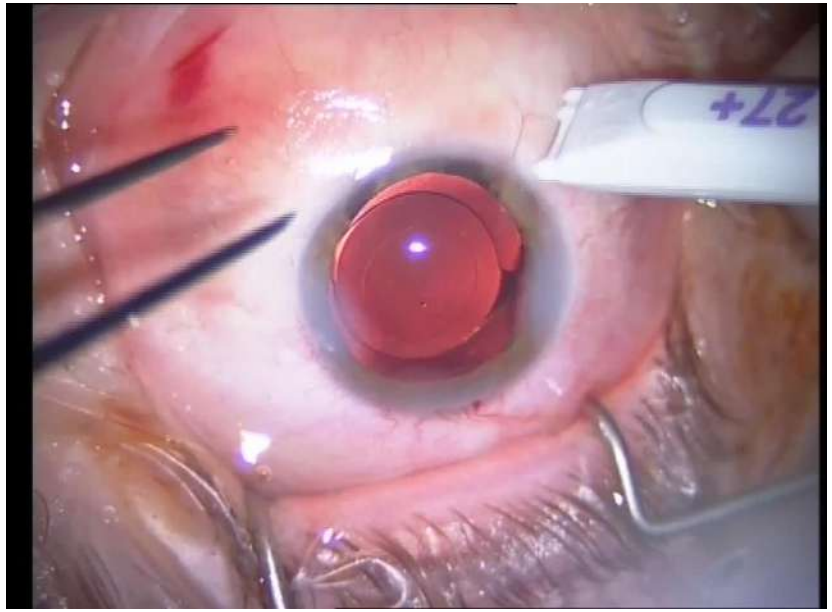
This image brings on nightmares for some…

So when would it be best to bring an acute PVD back?

- 8-26% acute PVDs have an associated RB/RD
  - **The chances of RB there after is <2-5%**
- AAO 2014 Guidelines: Depending on symptoms, risk factors, and clinical findings,
  - 1-8 weeks
- Rule of Thumb
  - Complicated PVD
    - MD in 2-4 weeks
  - Photopsia
    - 4-6 weeks
      - Double up visit: 2 w, 4w, 8w, 3 M…until done

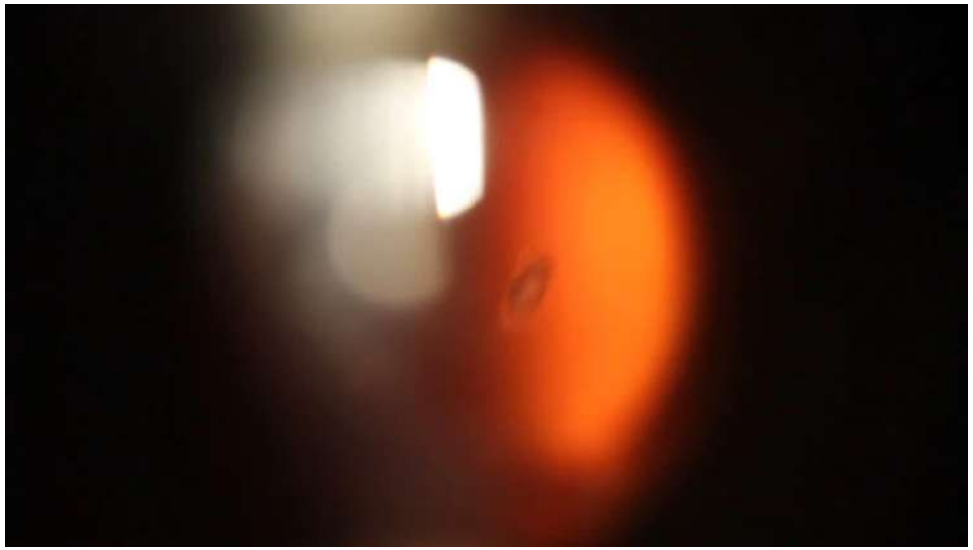
What is a floaterectomy?

- A vitrectomy to remove floaters
- Not a very common procedure
  - Same risk as any other vitrectomy
    - Likely higher risk for cataract since often young patients
  - Patient selection crucial



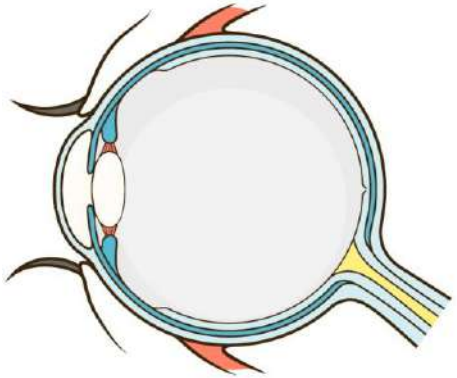
Laser floaterectomy aka: Yag vitreolysis

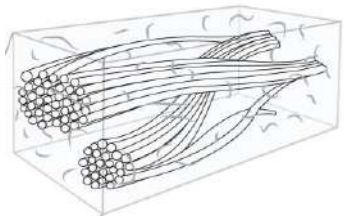
- Not a new concept
- Study of 52 pts w PVD and Weiss ring[1]
- 54 vs 9% Symptom improvement
- 53 vs 0% near total improvement of Sx
- Based on NEI VFQ improvements in:
  - Overall vision, peripheral vision and role difficulties



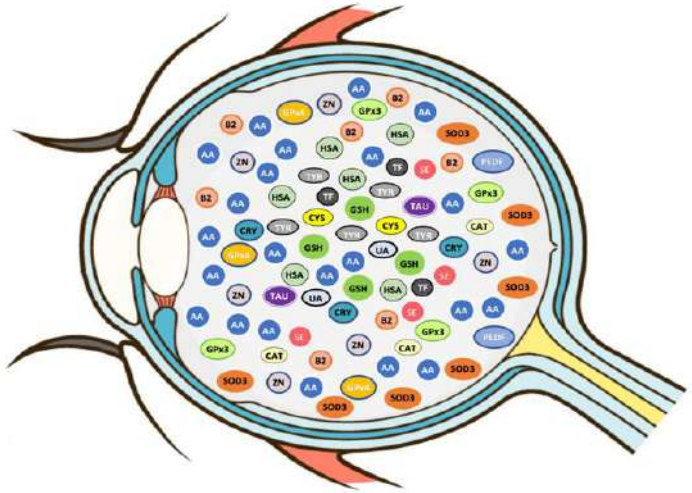
Shah et al. YAG Vitreolysis w Floaters.JAMA ophth. 9/17.

What about vitreous floaters?



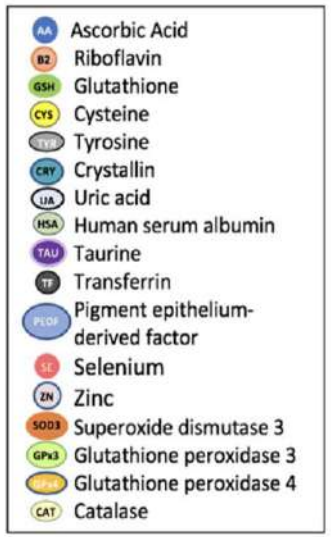


Healthy Vitreous



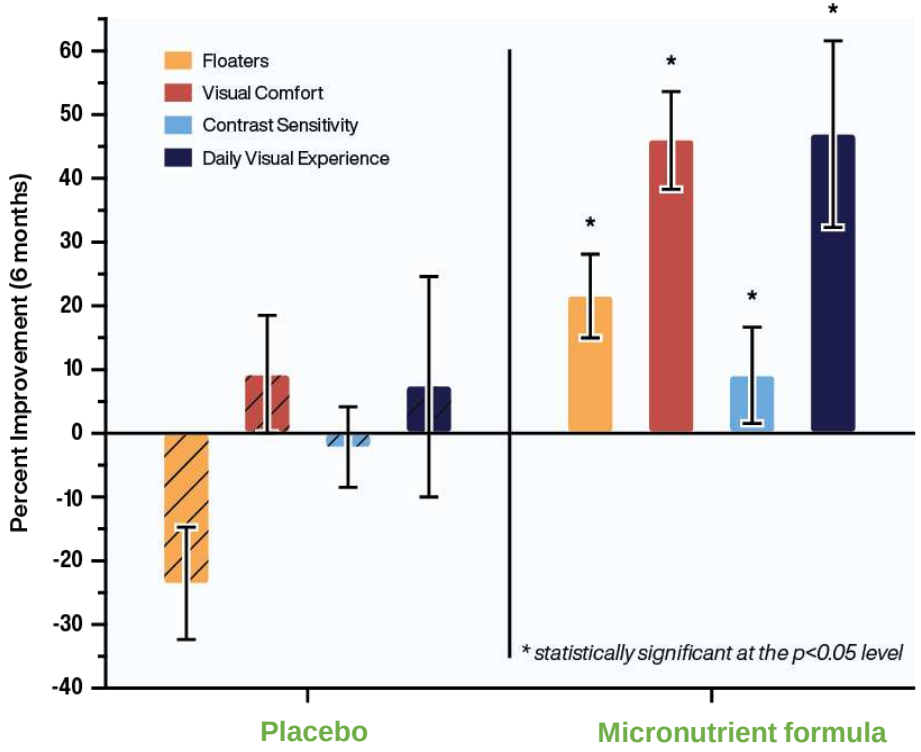
Vitreous Antioxidants

- Enzymatic & Non-enzymatic



Ankamah et al. Antioxidants. 2020:7

Results: Micronutrient Formula vs. Placebo



Those participants taking the micronutrient formula experienced:

- 21.5% decrease in vitreous opacity area
- 46% decrease in visual discomfort
- 9% improvement in contrast sensitivity
- 47% improvement in daily visual experience
- **67% of patients on active supplement experience an improvement in visual comfort**

Often related…

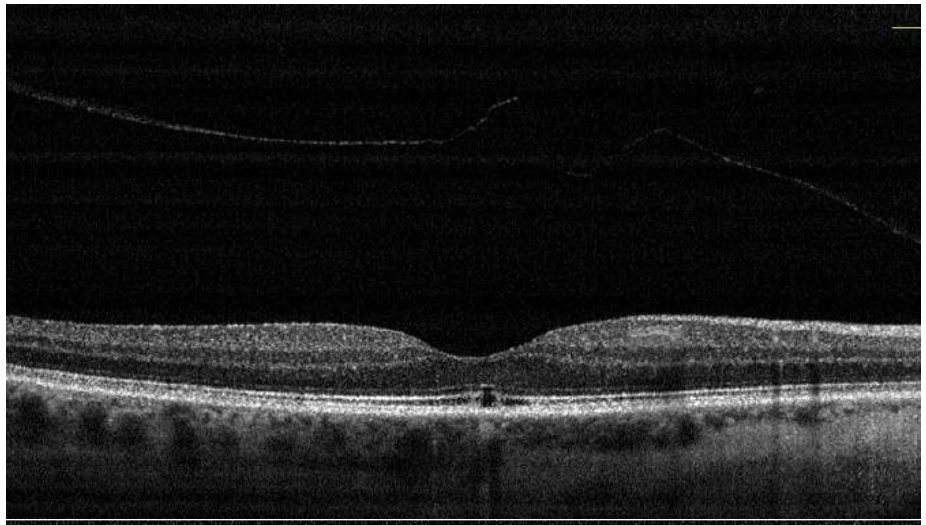
Vitreo-macular interface

What is VMA

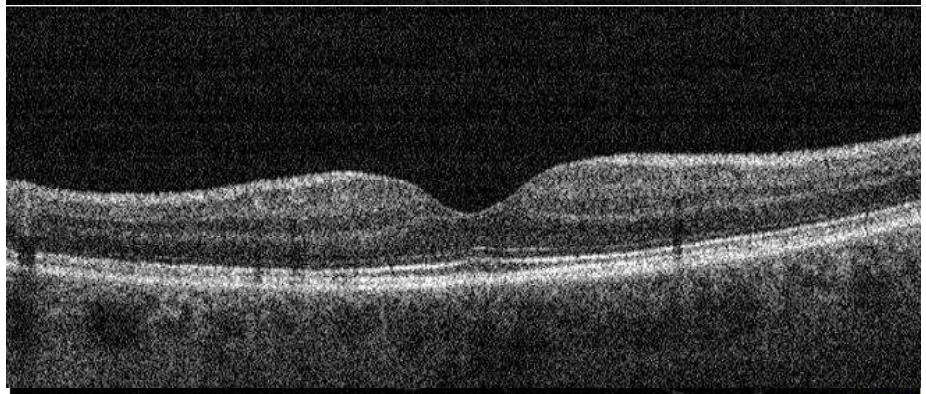
What is VMT

What is the treatment of either?

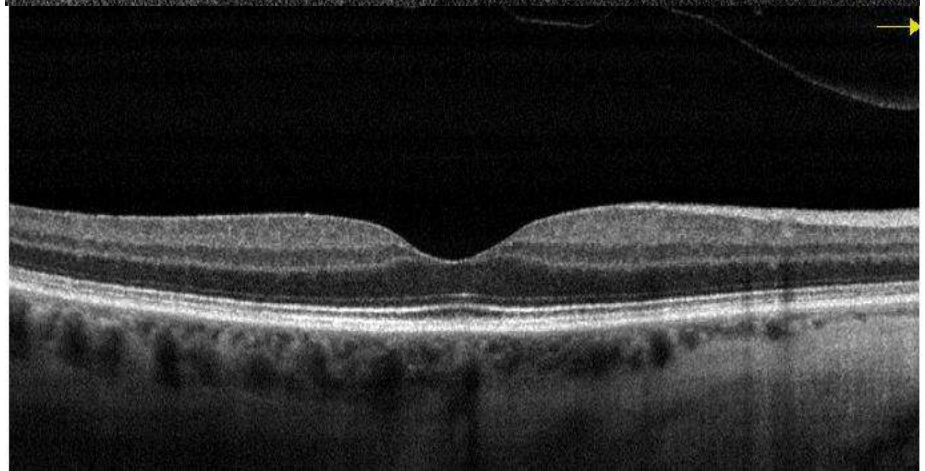
VMA/VMT case



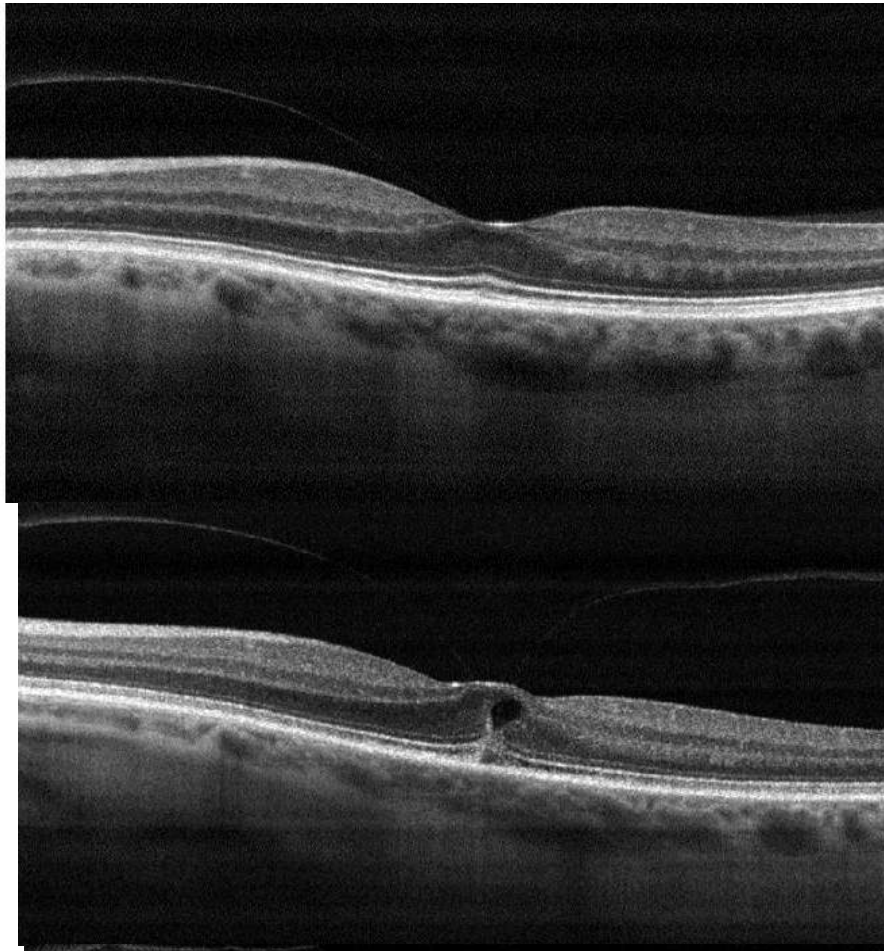
Initial visit: New floaters OD
20/20 OU

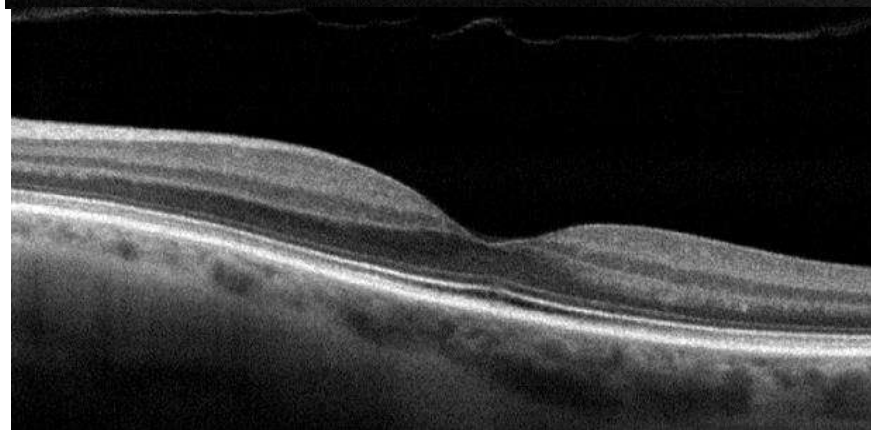


1 month later
No floaters, but distortion OS
20/20 OD and 20/20- OS



2 months after initial
20/20 OU
No floaters noted either eye





ORIGINAL STUDY

PREVALENCE OF VITREOMACULAR ADHESION IN PATIENTS WITHOUT MACULOPATHY OLDER THAN 40 YEARS

Rodman, Julie A. OD, MS*; Shechtman, Diana OD*; Sutton, Brad M. OD†; Pizzimenti, Joseph J. OD‡; Bittner, Ava K. OD, PhD*; VAST Study Group

Author Information

Retina: October 2018 - Volume 38 - Issue 10 - p 2056-2063
doi: 10.1097/IAE.0000000000001792

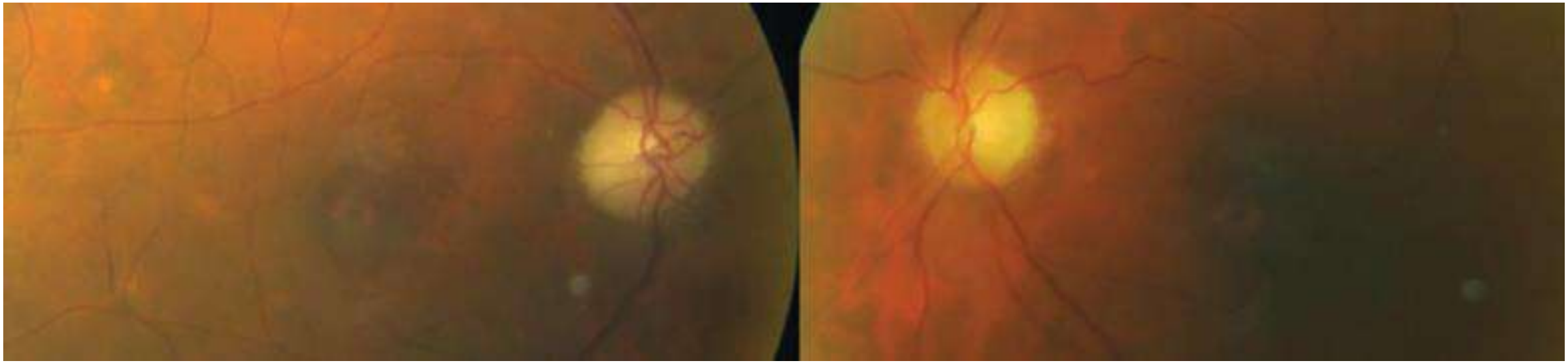
- **39/1% of eyes had VMA/VMT**
- **For every 1yr increase in age, there was a 7% decrease in incidence of VMA/VMT**
- **AA have 55% decreased odds vs Caucasians**
- **Larger area VMA more likely in younger adults and hyperopes (vs emmetropes)**
- **VMA more likely in primary care vs tertiary practices**
- **VMA more likely in patients without hyperlipidemia**

Case 1: My Friend's Mom

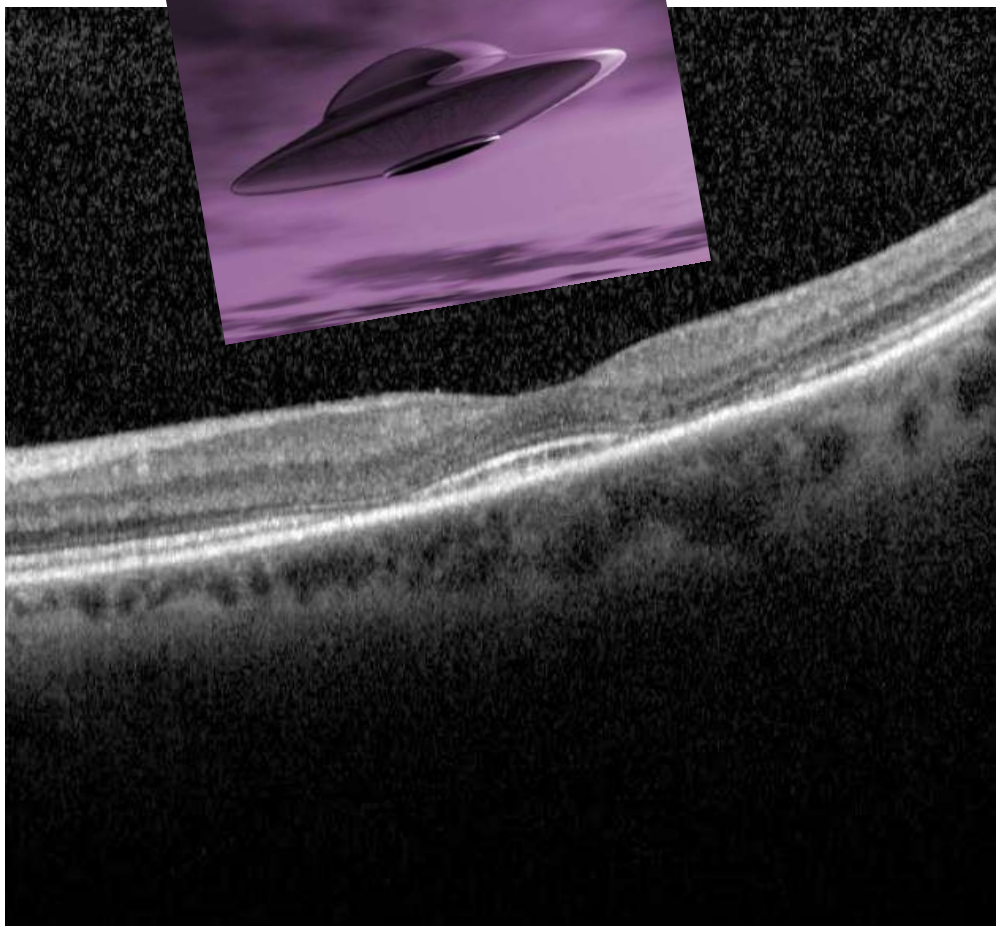
- 62 year-old white female
- CC: Patient presents for an eye exam because she has been frustrated with her vision. The patient states that she feels like she is "looking through a steamy shower curtain". The patient has been told she is developing cataracts in both eyes and she wants to know if it is time to have cataract surgery. The patient complains of difficulty seeing at night.
- Med Hx: Systemic HTN, RA, High Cholesterol
- Best corrected VA
  - DVA: OD 20/20-3 OS 20/100-1
  - NVA: OD J1+ OS J10
- IOP OD 18 mmHg OS 20 mmHg
- Pupils, EOMS and Adnexa were WNL OU
- Color Vision: OD 4/12 OS 0/12
- SLE: Unremarkable, except 1-2+ NSC OU

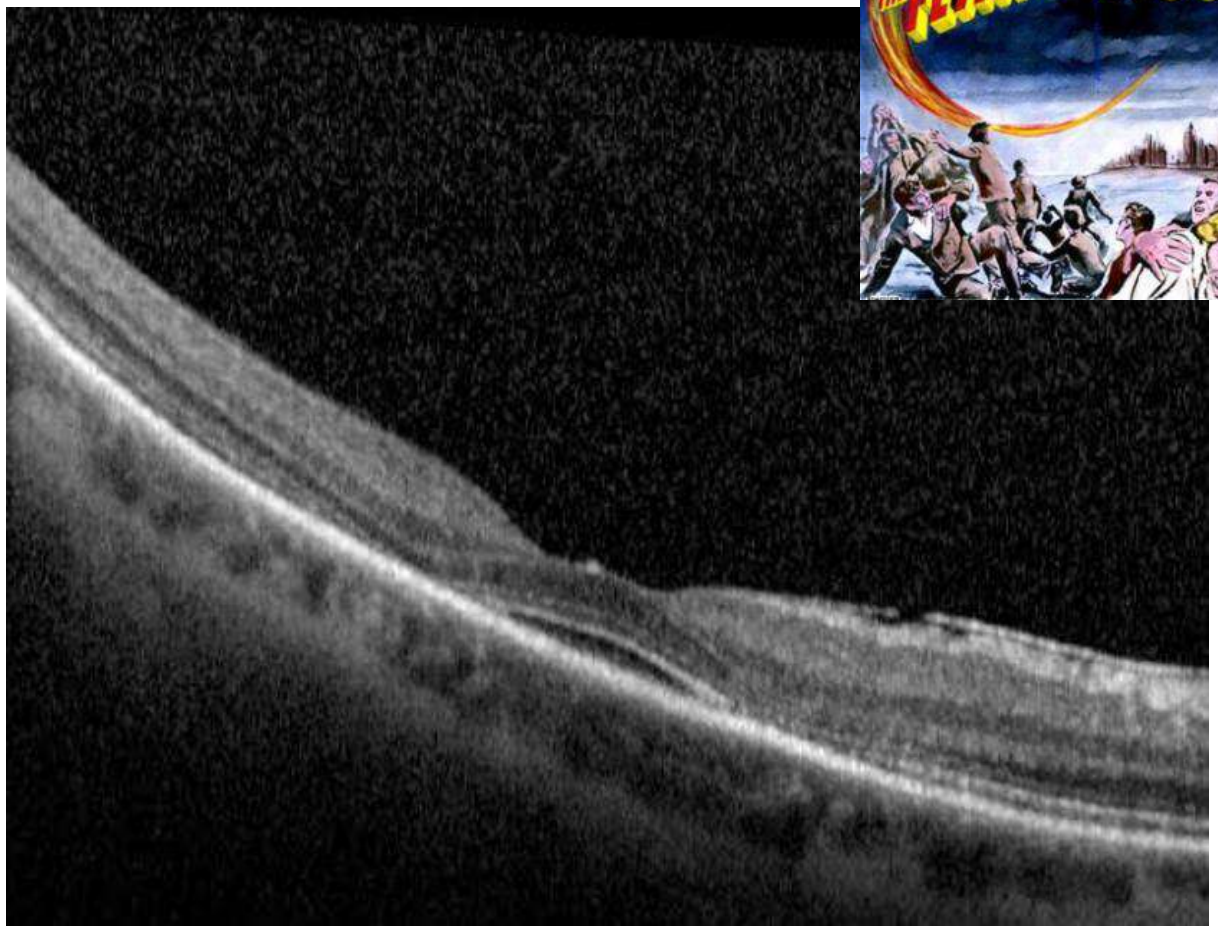
Dilated Fundus Exam

Need to look closer at the macula...

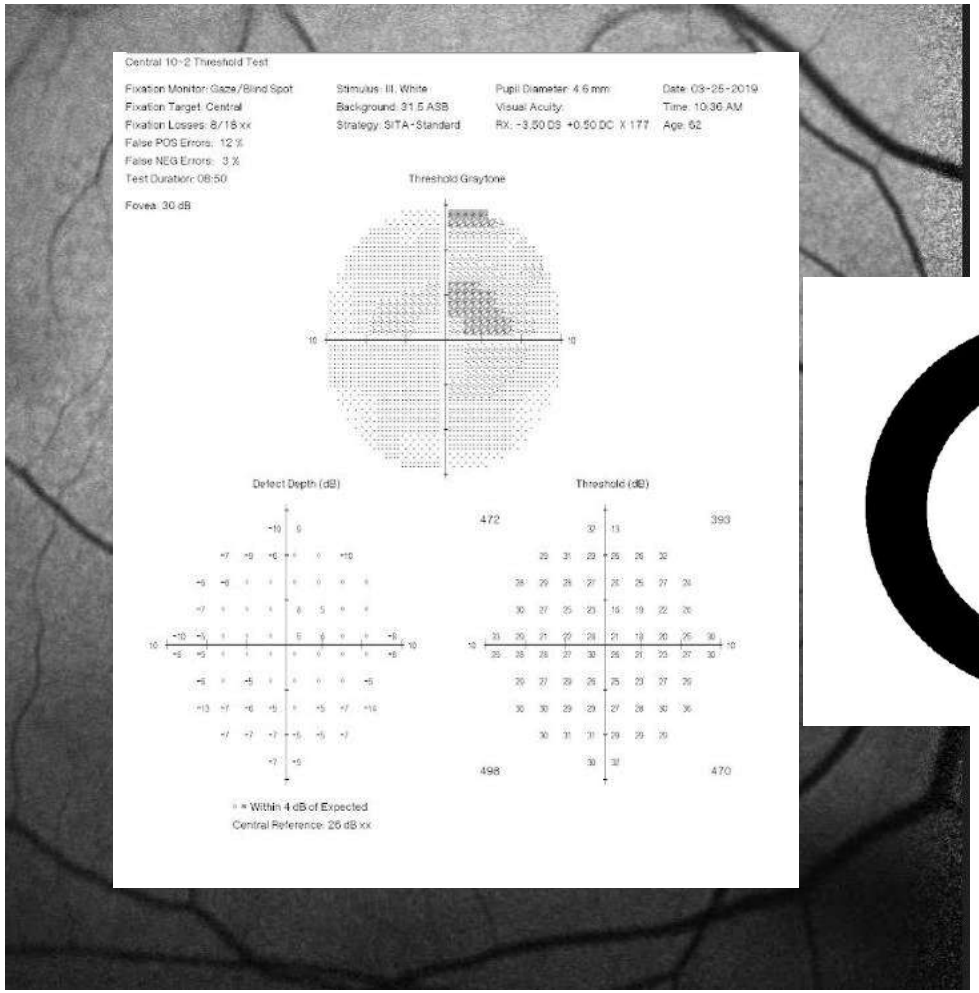


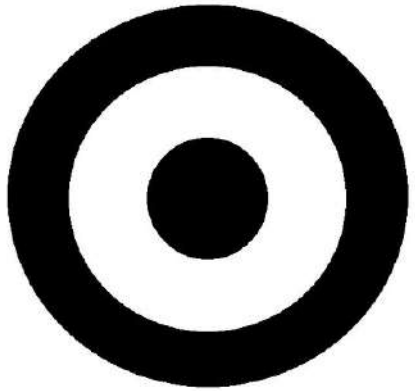
Another case

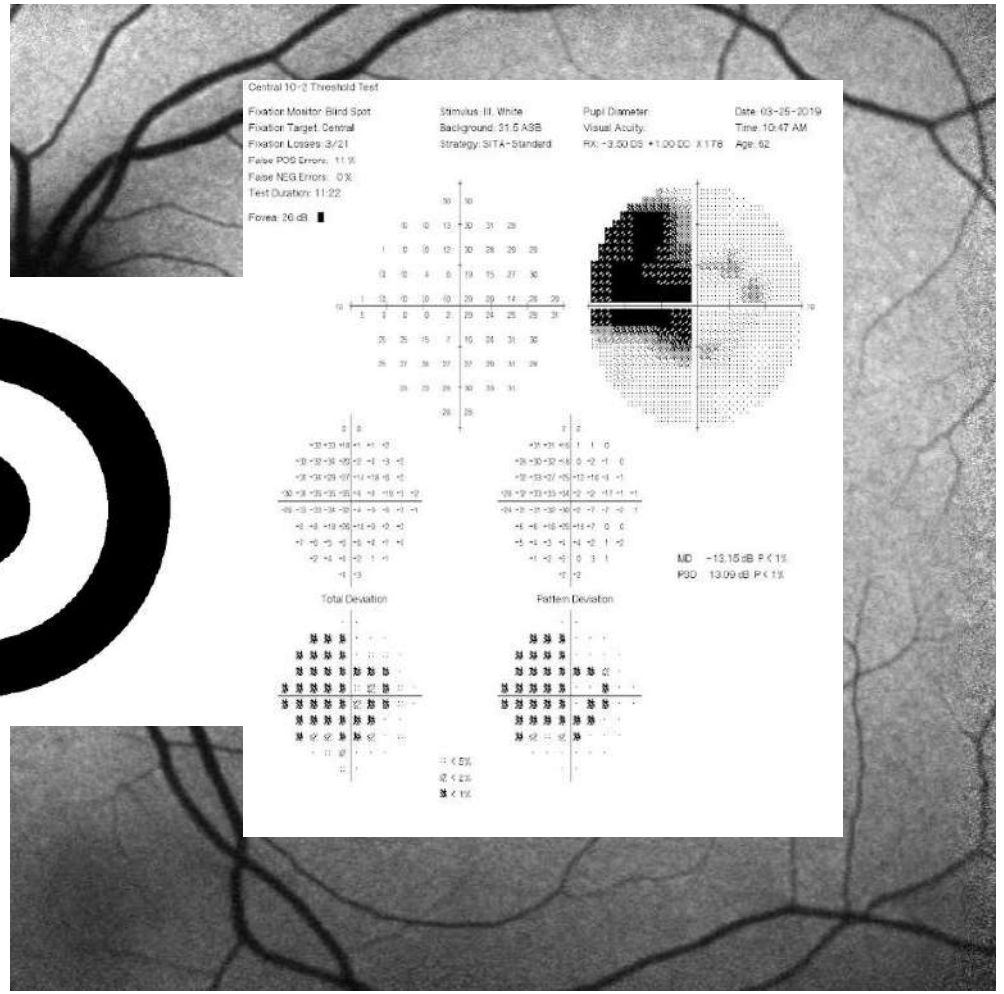




Case 1: FAF and HVF 10-2







Let’s take a look at our patient’s medication list...

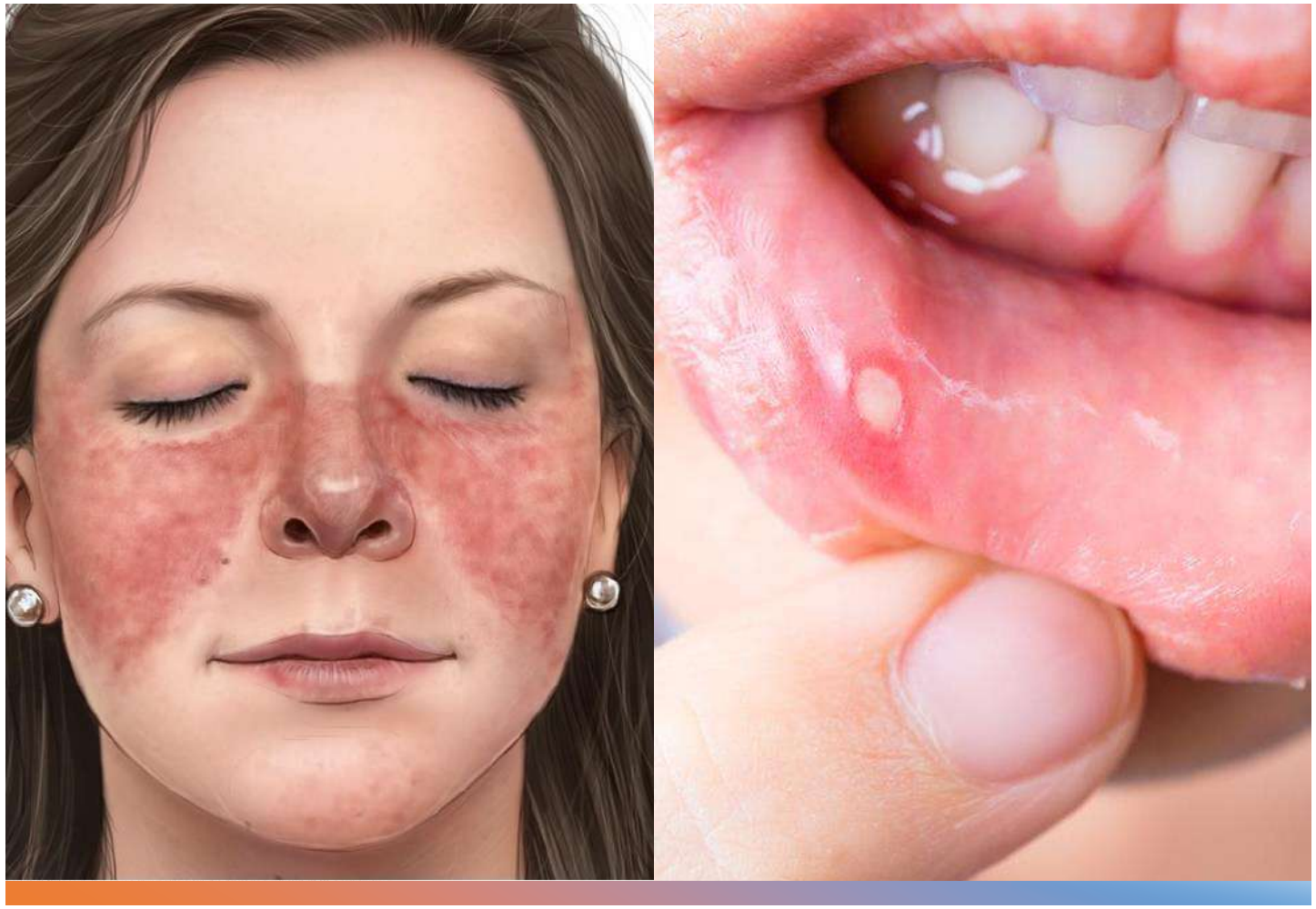
- Centrum Complete
- Vitamin D3
- Sertraline HCl
- Lisinopril
- Levothyroxine Sodium
- Diclofenac Sodium
- Hydroxychloroquine (Plaquenil) 200mg BID

What is Plaquenil (Hydroxychloroquine)

- A medication originally used to treat Malaria.
- Now used to treat a variety of autoimmune disorders and their symptoms.
  - Lupus
  - Rheumatoid Arthritis
  - Sjögren's Syndrome
- Plaquenil is used to treat skin disorders
  - Sarcoid, Eczema, Skin disorders where there is photosensitivity, Lichen Planus and Urticarial Vasculitis

How Plaquenil works

- Lowering the immune system’s ability to cause inflammation.
- Reduces inflammation
- Can help control symptoms:
  - Rash
  - Sores
  - Joint pain



Possible Ocular Effects of Plaquenil (Hydroxychloroquine)

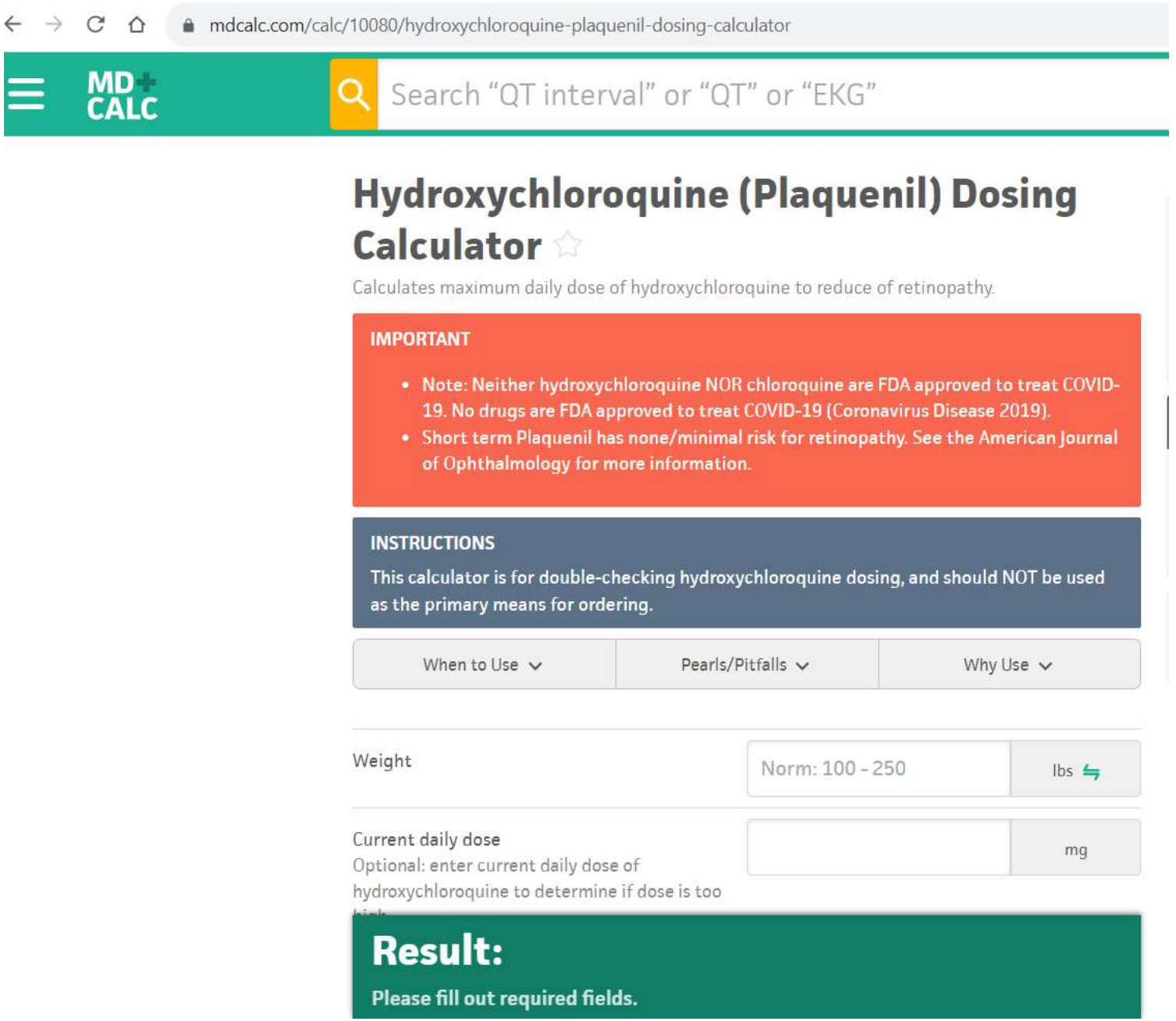
- A rare side effect of extended or over-dose Plaquenil use can be damaging, or toxic to the Retina.
- It is believed that Plaquenil binds itself to the Retinal Pigment Epithelium (RPE) and can cause damage to the photoreceptors.
- Typically asymptomatic in its early stages, but can lead to severe retinal damage, and permanent vision loss.
- Early signs include blurry central vision, losing the ability to read a digital clock, loss of color vision, and trouble seeing at night.

Higher Risk Factors for Toxicity:

- Taking Plaquenil for 5+ years
- Taking a higher than recommended dose
- Pre-existing Kidney or Liver Disease
- Pre-existing Retinal Disease
- Age 60 or older
- Losing a significant amount of weight while taking Plaquenil without adjusting your dose

- The current American Academy of Ophthalmology (AAO) guidelines, published in 2016, recommend a maximum daily hydroxychloroquine dose of ≤5.0 mg/kg of real weight. These guidelines were established to minimize the likelihood of permanent vision loss related to hydroxychloroquine retinopathy.
- The 2016 revision was prompted by a study by Melles and Marmor in 2014 which suggested that hydroxychloroquine retinopathy is more common than previously thought. They demonstrated that a daily consumption of 5.0 mg/kg real body weight or less is associated with a low risk for up to 10 years. However, there is significant variability in individuals that develop hydroxychloroquine retinopathy.
- This study was performed only in adult patients.
- The American College of Rheumatology updated their guidelines in August 2016 to acknowledge the American Academy of Ophthalmology's position, but does not specify a preferred dosing regimen.
- Doses must be adjusted for renal insufficiency.
- Patients with underlying retinal or macular disease may be at a higher risk for toxicity.
- Patients who are undergoing tamoxifen therapy for breast cancer have a higher risk for toxicity.

This website is very helpful in calculating safe doses.



Calculated Risk Factors

Weight: 180 lbs

Current daily dose
Optional: enter current daily dose of hydroxychloroquine to determine if dose is too high: 400 mg

408 mg daily dose
Maximum daily dose of hydroxychloroquine

4.9 mg/kg
Dose is within recommended daily dose of ≤5 mg/kg.

2 %
10-year risk of retinopathy (20% risk at 20 years)

Copy Results | Next Steps

Weight: 155 lbs

Current daily dose
Optional: enter current daily dose of hydroxychloroquine to determine if dose is too high: 400 mg

352 mg daily dose
Maximum daily dose of hydroxychloroquine

5.7 mg/kg
Warning: this is greater than the recommended daily dose of ≤5 mg/kg. Consider lowering dose.

10 %
10-year risk of retinopathy (40% risk at 20 years)

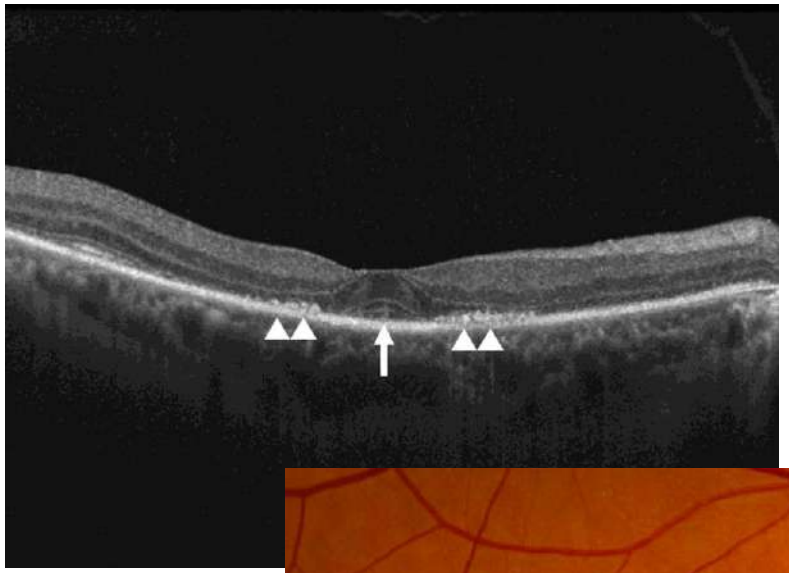
Copy Results | Next Steps

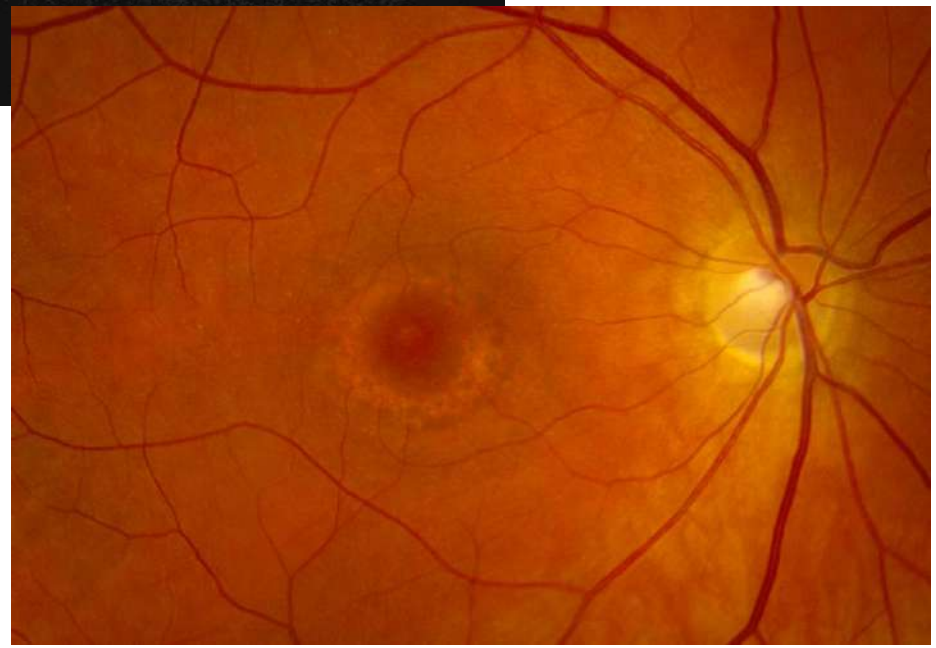
Important Exam Testing:

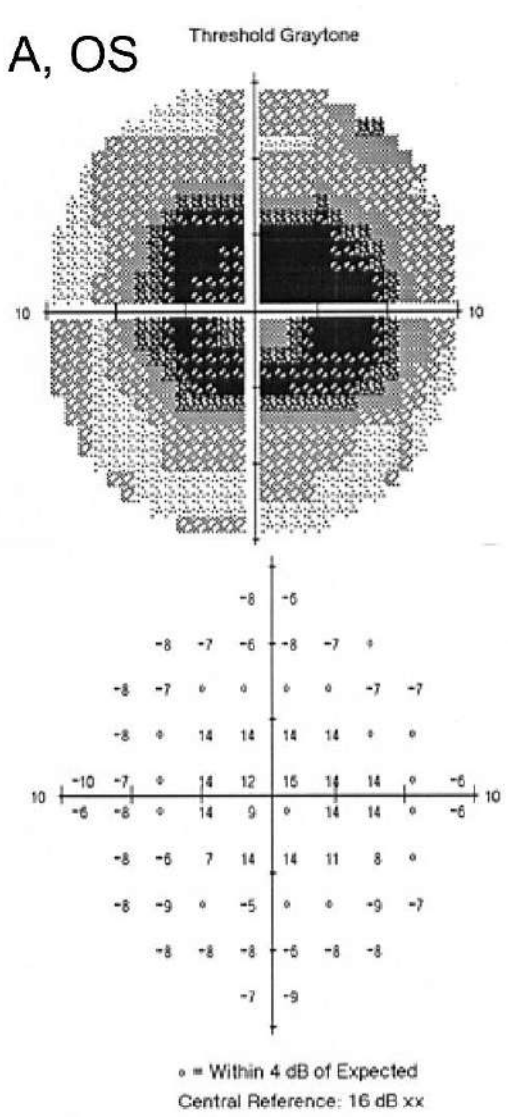
- HPI should include:
  - How long have you been taking Plaquenil?
  - What is your weight?
  - What is your dose?
  - Do you have a history of Kidney disease?
- Have you noticed any changes in your vision?
  - Baseline testing should be completed before, or within the first year of, beginning Plaquenil.

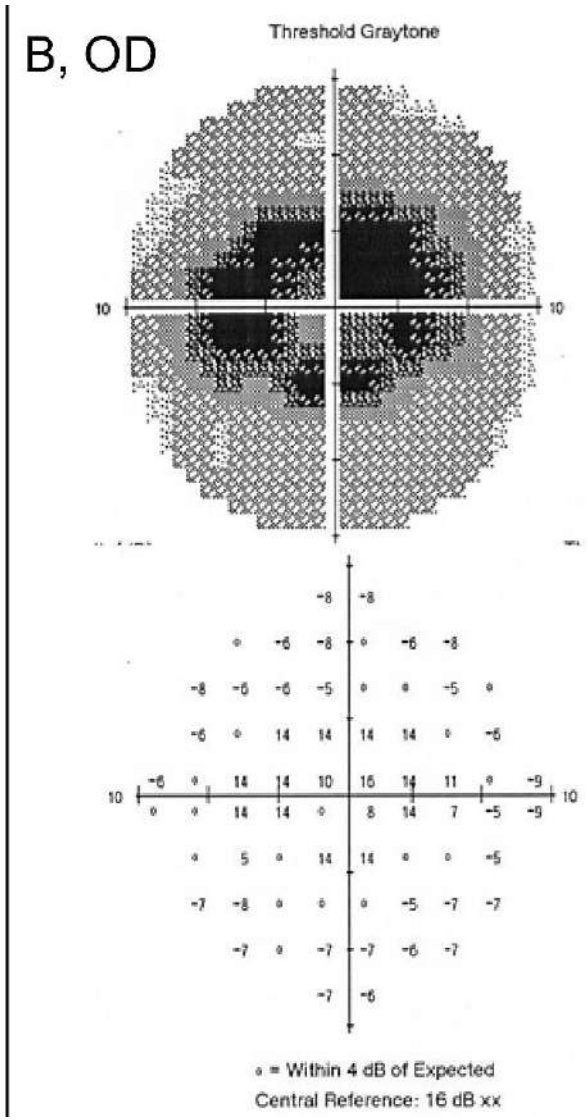
  - Testing:
    - 10-2 Humphrey Visual Field (HVF)
    - OCT Macula
    - Fundus Autofluorescence (AF/FAF)
    - Color vison

Plaquenil
Hydroxychloroquine Retinopathy





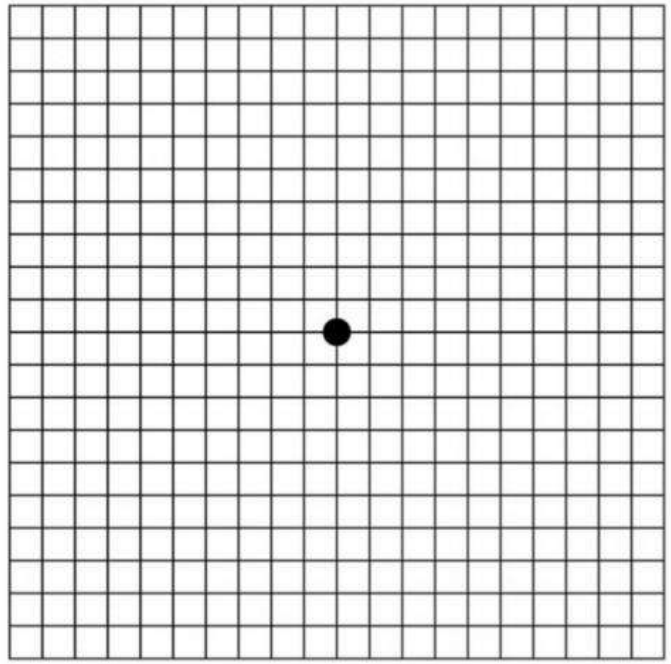




At home monitoring

- In between visits, patients should be encouraged to self-screen at home with an Amsler Grid to detect early defects in the vision.

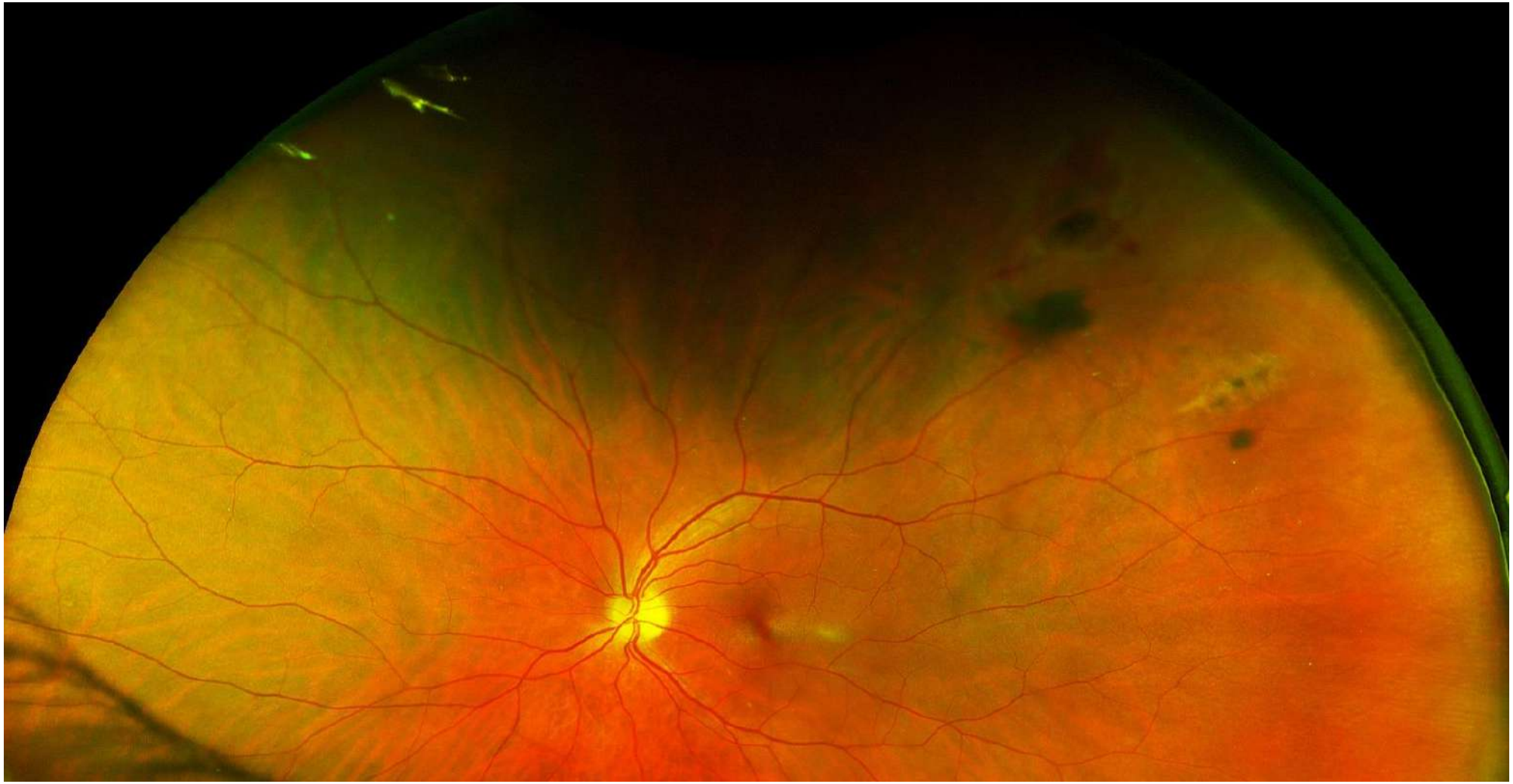
- If a change is detected in their vision, the patient should contact their Optometrist or Ophthalmologist for additional screening in the office.



New Onset floaters and change to vision OS

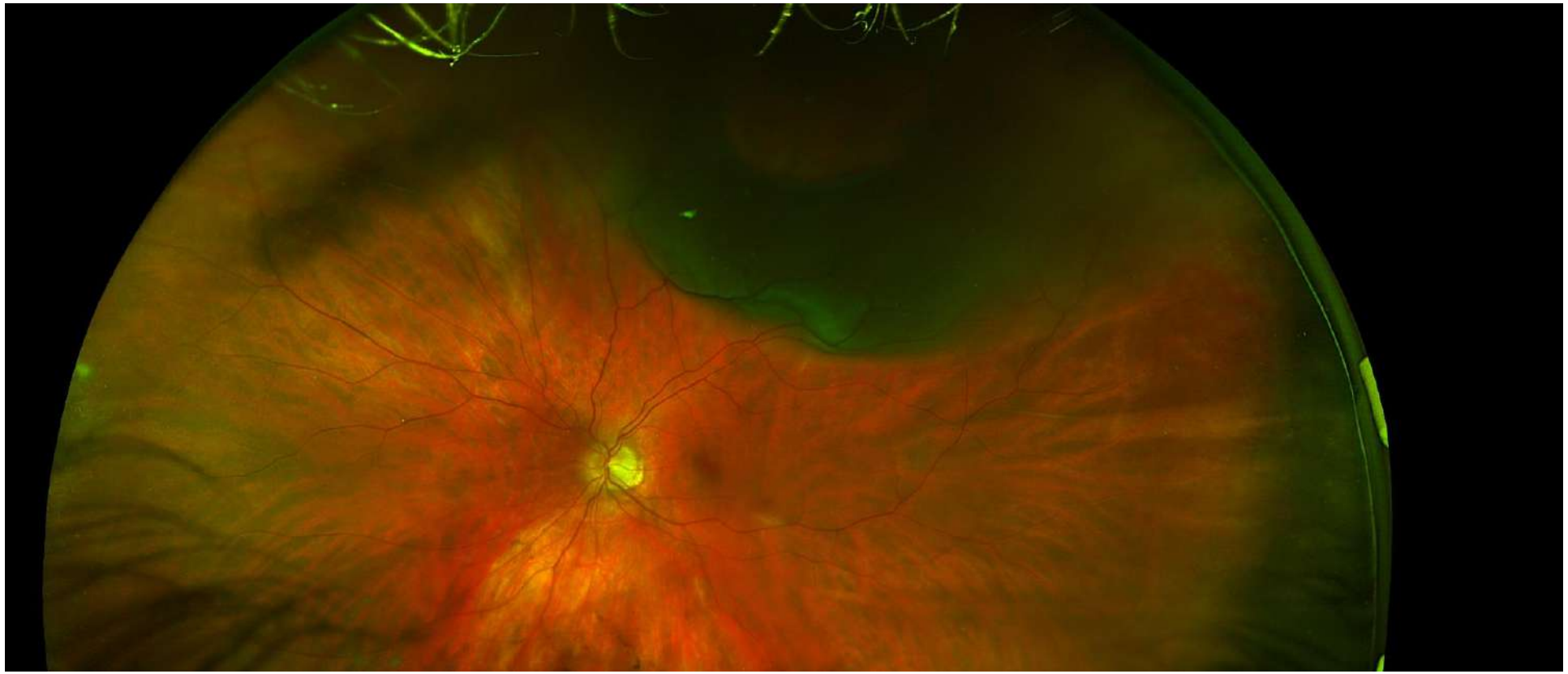
- Symptoms: New floaters OS
- Vision 20/20 OU
- Pupils, motility and CVF normal
- Anterior segment normal

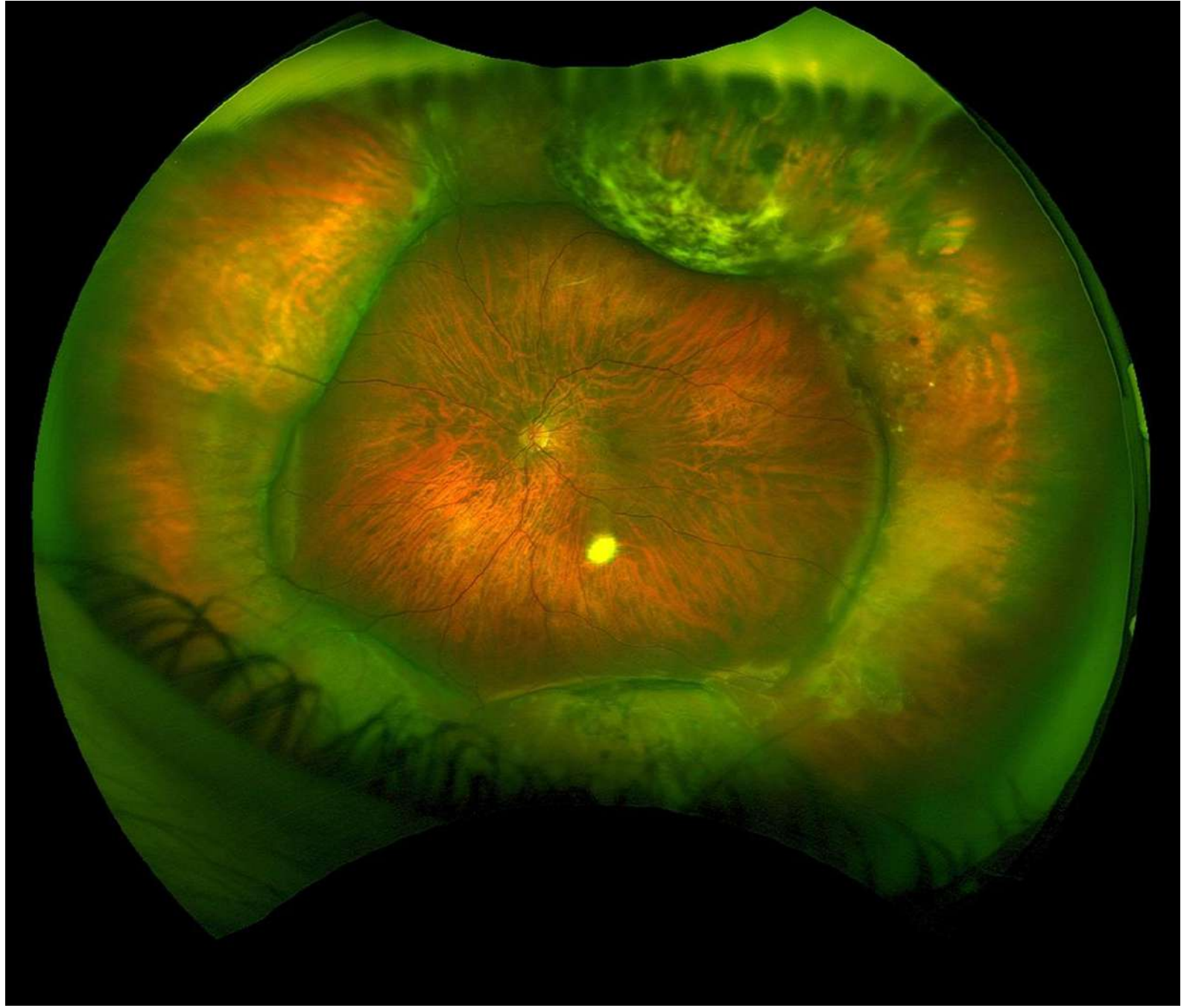
Initial OS



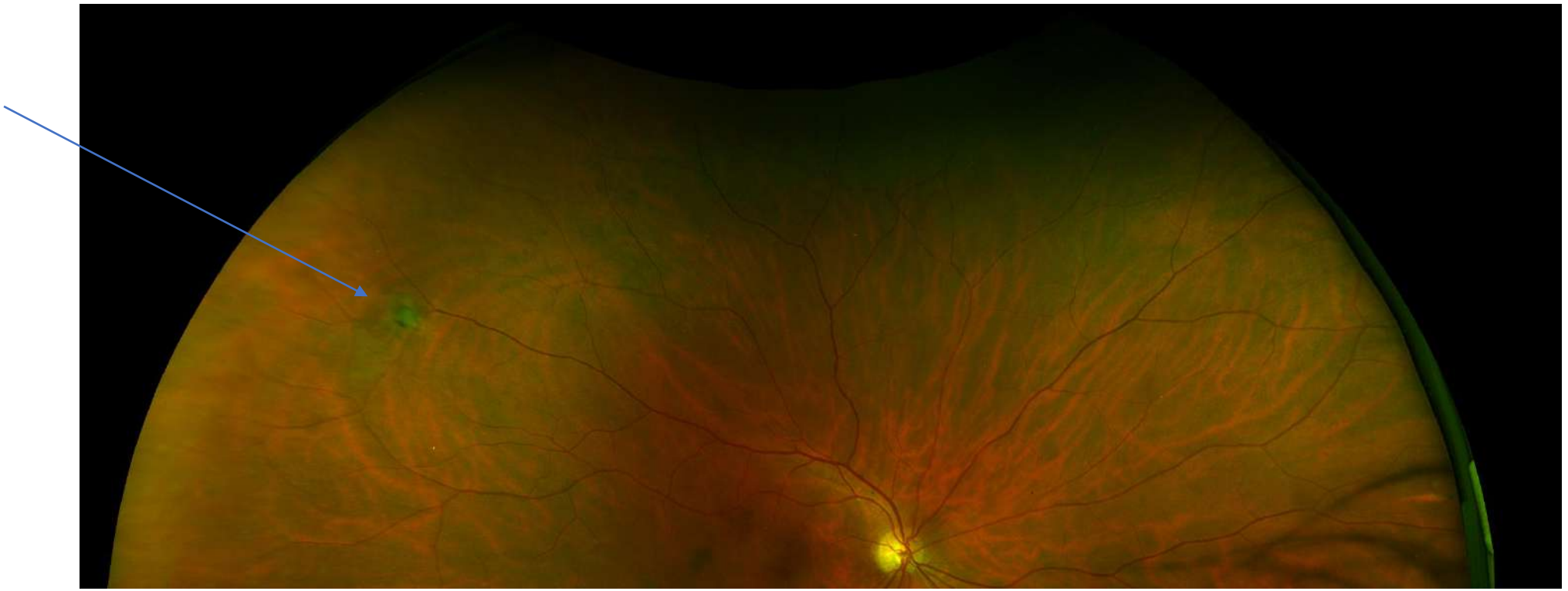
WWYD?

f/u OS: Did not go to RS for laser





Initial OD: look ST



Not much to see??

OD at f/u after OS fixed

An interesting RD

3/19:

67 yo CC of “Noticed vision on bottom half becoming dimmer over last 2 days and coming up toward the center”

Healthy general health

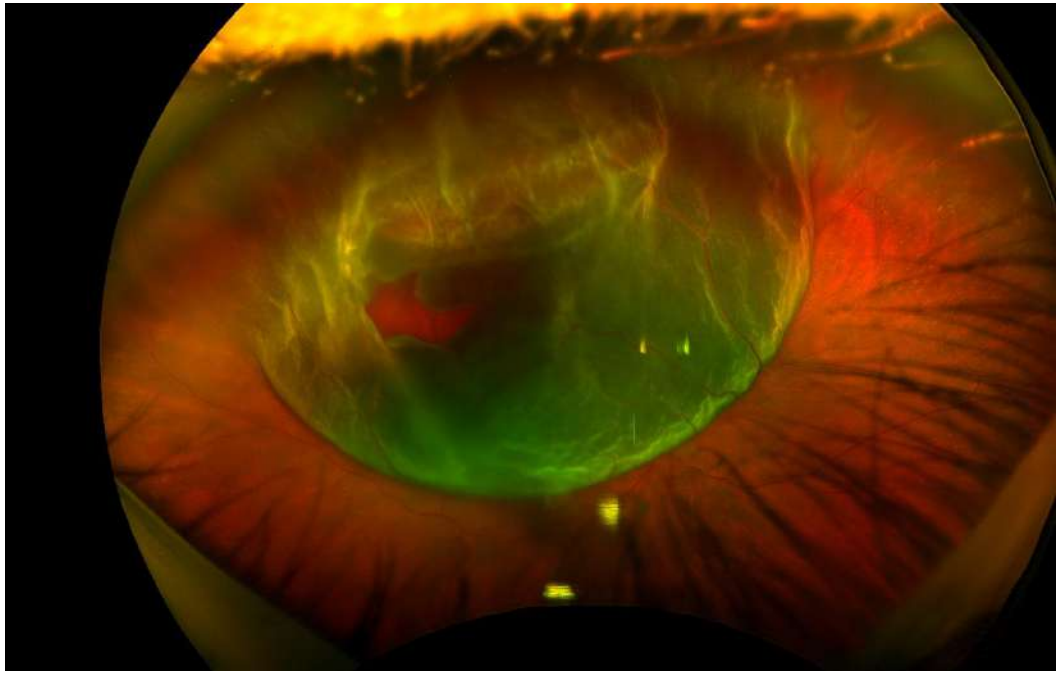
OcHx:

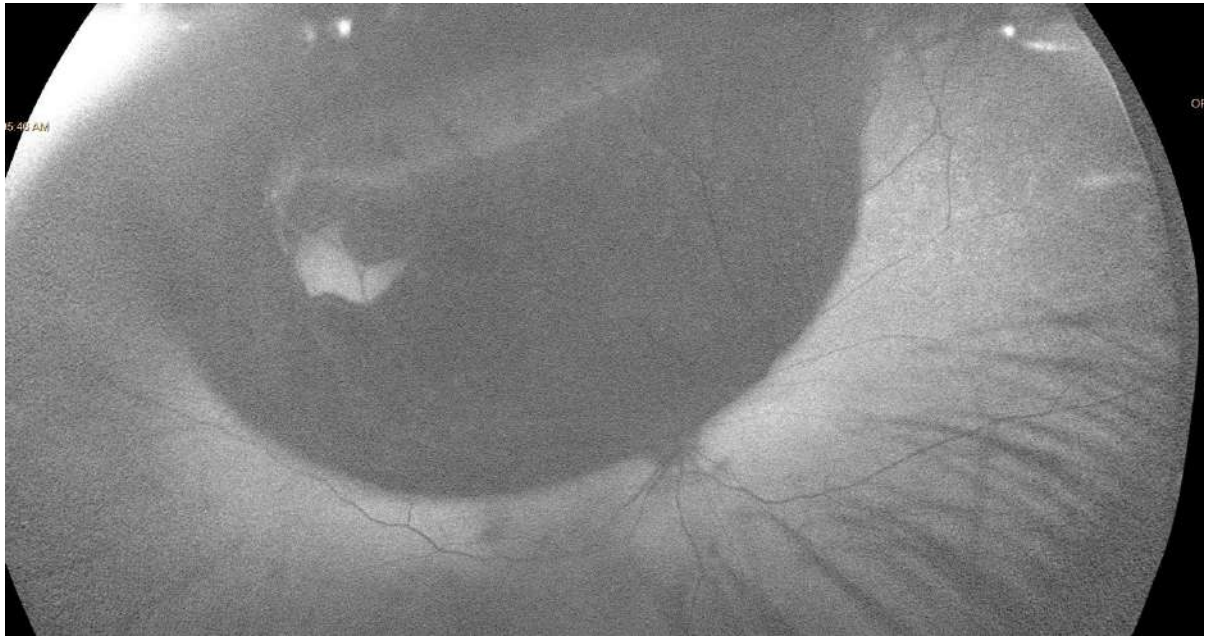
CE OD 1/16

Yag OD 2/19

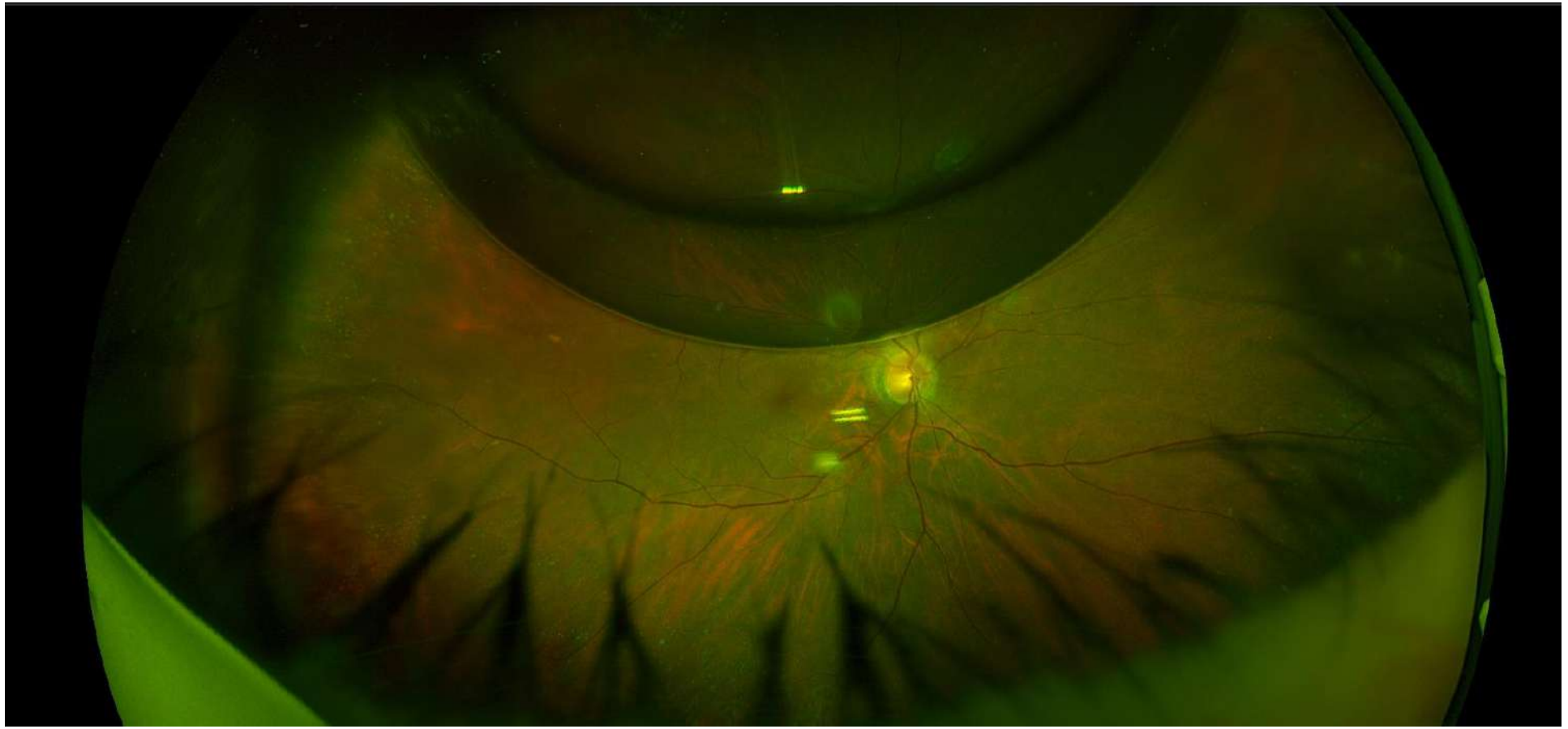
VA: OD: CF

OS: 20/20





What is this??



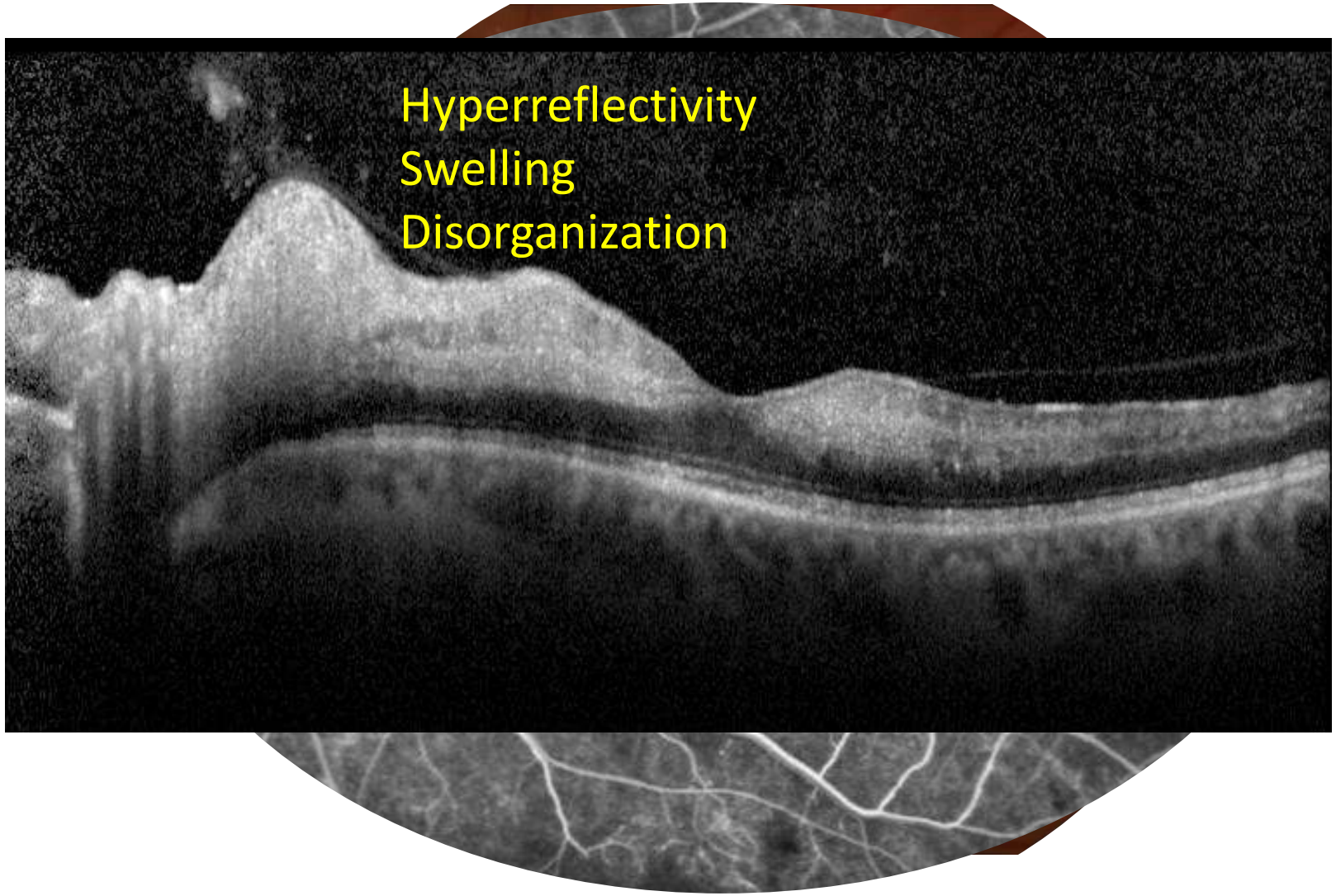
How to handle an RD

- What do you tell your patients
  - Options of procedures?
- Timeliness of referral?
- Expectations for outcome?

Next case: Referral from Optometrist

- 64 year-old white male
- CC: Patient referred from his optometrist. The patient woke up yesterday morning with acute, painless vision loss in the left eye. The patient says that he “is now blind in the left eye”. The right eye is doing well and has not noticed any changes in it. He also denies any new flashes of light or floaters in either eye.
- Med Hx: Systemic HTN, High Cholesterol, Heart Disease
- Best corrected VA
  - DVA: OD 20/20 OS LP
  - NVA: OD J1+ OS LP
- IOP OD 15 mmHg OS 17 mmHg
- Pupils: +APD OS
- EOMS and Adnexa were WNL OU
- SLE: Unremarkable, except 2+ NSC OU

Central Retinal Artery Occlusion



Retinal Artery Occlusion

- Acute obstruction of a retinal artery results in ischemia (dysfunction) and ultimately infarction (death)
- Acutely, inner retinal edema seen clinically as **retinal whitening**
- The normal underlying normal choroidal circulation yield the appearance of a **Cherry RED spot**
- Chief complaint:
  - scotoma, blindness
- +APD is always present regardless of VA
- Retinal embolus may be visible in up to 40% of patients.
  - The embolic material can be a shiny cholesterol plaque, gray-white platelet plaque, or white calcium plaque.
- Treatment controversial: paracentesis, globe compression, YAG laser embolysis, aspirin
- Workup: R/O GCA, Carotids, Echo, Hypercoagulability workup, Vasculitis/Uveitis workup

CEI

CINCINNATI EYE INSTITUTE

Please give this form to your Emergency Room Physician

Diagnosis (circle):

Transient monocular vision loss

Branch Retinal Artery Occlusion

Central Retinal Artery Occlusion

You have been diagnosed with Acute Retinal Ischemia. This diagnosis increases your risk of having a cerebral stroke. Based on the *American Academy of Ophthalmology Preferred Practice Pattern Guidelines* (Management of Acute Retinal Ischemia. *Ophthalmology*. October 2018. Volume 125, Issue 10, Pages 1597–1607) we recommend you go immediately to the nearest Emergency Room with stroke capabilities (see-attached list).

Workup recommended in setting of acute retinal ischemia (stroke equivalent)

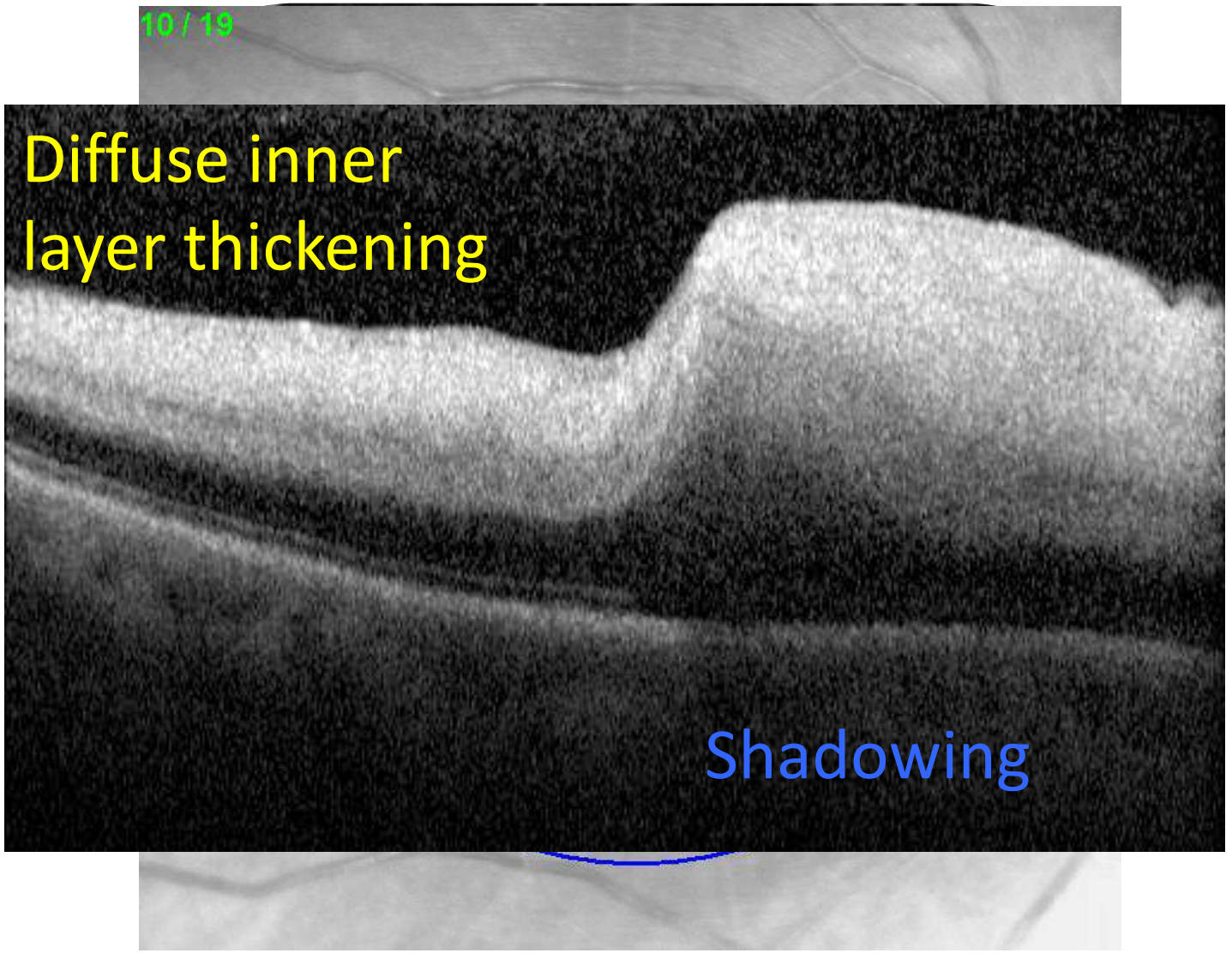
- Head MRI without contrast (DWI)
- Head/Neck MRA or CTA; if unable, then carotid Doppler
- Transthoracic Echocardiogram
- Stroke team consult
- If concern for Giant Cell Arteritis, obtain CBC, ESR, CRP and consult rheumatology if indicated

Who is at risk for CRAO?

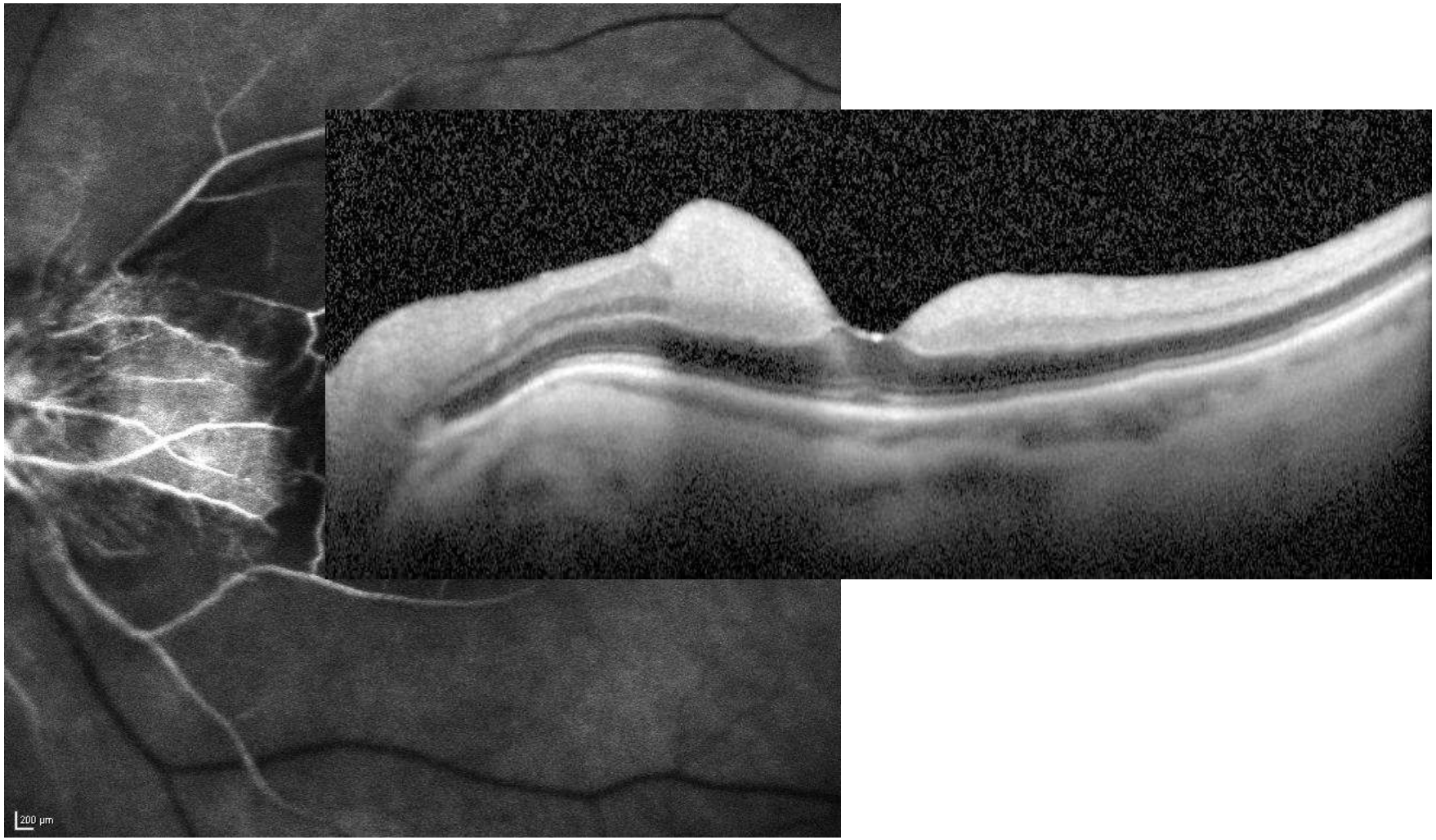
- The major risk factors for CRAO can be divided into nonarteritic and arteritic.
- **Nonarteritic.**
  - More than 90% of CRAOs are nonarteritic in origin.
  - Ipsilateral carotid artery atherosclerosis is the most common cause of retinal artery occlusion.
    - As high as 70% reported among patients with CRAO or branch retinal artery occlusion.
  - Other causes of nonarteritic retinal artery occlusion include
    - cardiogenic embolism, hematological conditions (sickle cell disease, hypercoagulable states, leukemia, lymphoma, etc.), and other vascular diseases, such as carotid artery dissection, moyamoya disease, and Fabry disease
- **Arteritic.**
  - CRAO of arteritic etiology is mostly caused by giant cell arteritis.
    - Other vasculitic disorders such as Susac syndrome, systemic lupus erythematosus, polyarteritis nodosa, and granulomatosis with polyangiitis have also been associated with retinal artery occlusion.

IV* acetazolamide	Reduce intraocular pressure
IV mannitol	Reduce intraocular pressure
Topical antiglaucoma medications	Reduce intraocular pressure
Pentoxifylline	Vasodilation to increase blood oxygen content
Inhalation of carbogen	Vasodilation to increase blood oxygen content
Sublingual isosorbide dinitrate	Vasodilation to increase blood oxygen content
IV methylprednisolone	Reduce retinal edema, only given in arteritic CRAO
IV or intra-arterial recombinant tissue plasminogen activator (rt-PA)	Thrombolytic therapy to dissolve clot
Hyperbaric oxygen therapy	Increase blood oxygen tension
Surgery/Procedures	
Anterior chamber paracentesis	Reduce intraocular pressure
Ocular massage	Fluctuation in intraocular pressure to mechanically dislodge clot
Nd:YAG laser embolectomy	Lyse or dislodge the clot
Pars plana vitrectomy	Surgical removal of the clot
Lifestyle Modification	
Optimization of atherosclerotic diseases	Secondary prevention

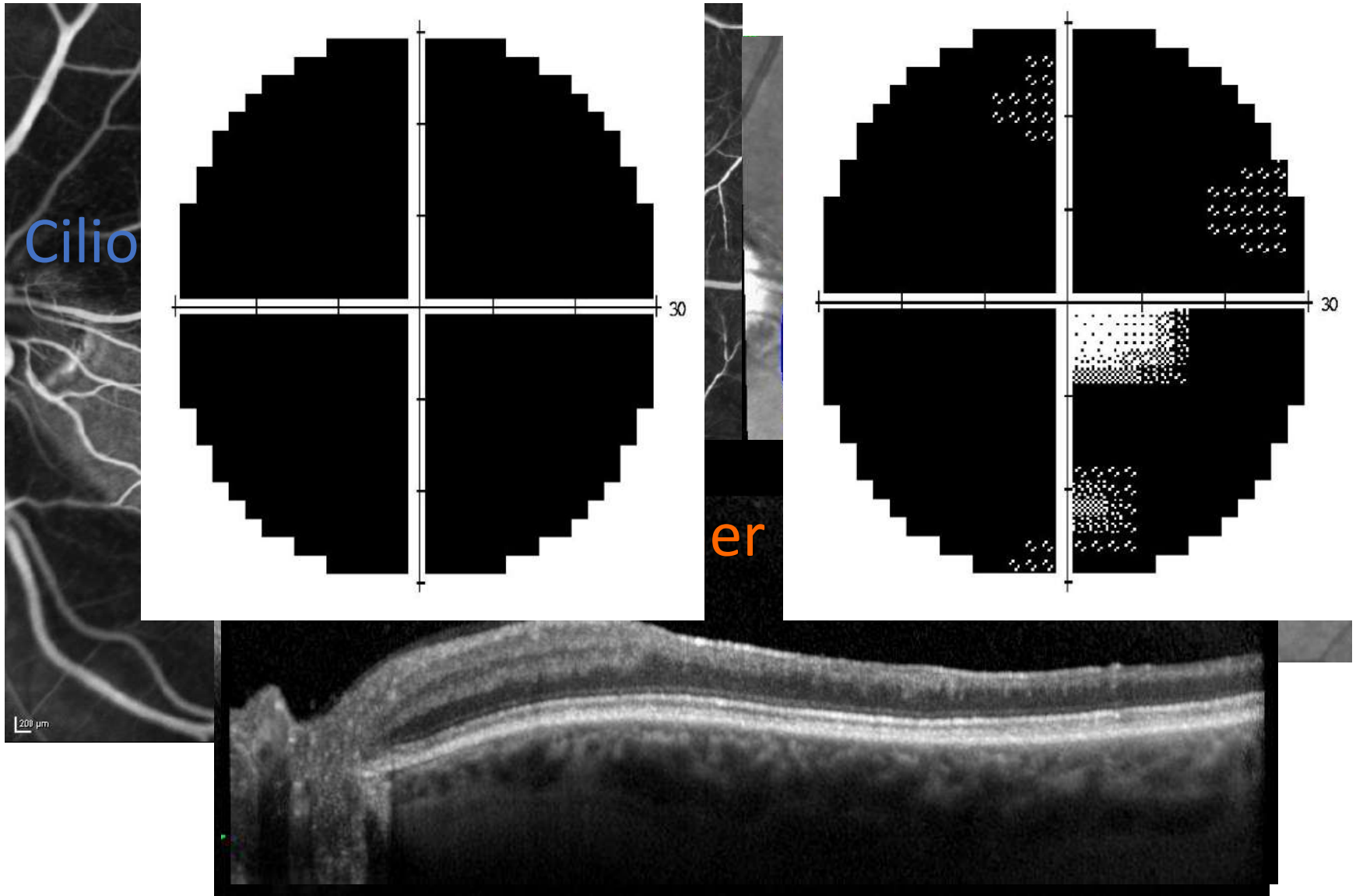
CRAO



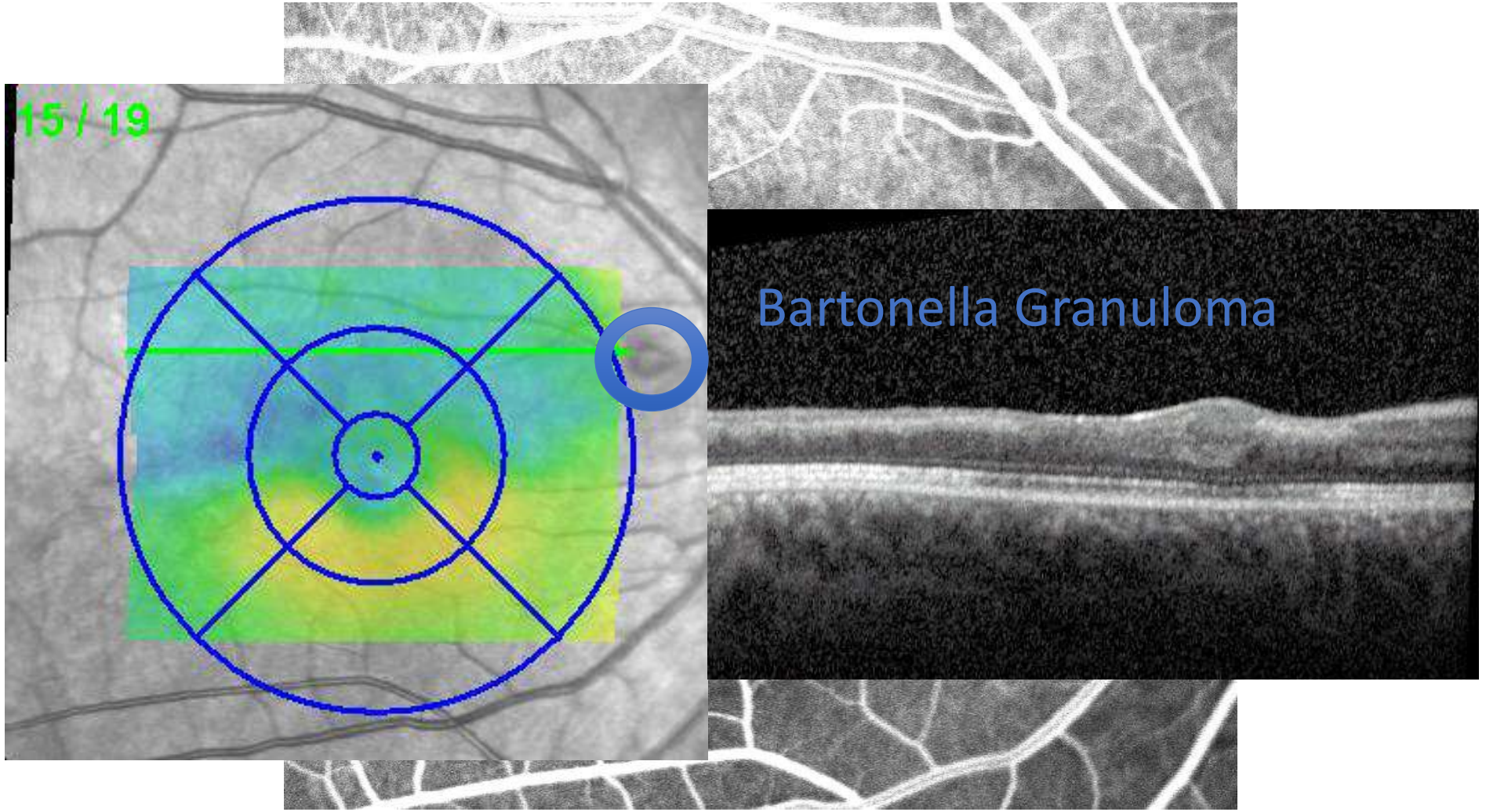
CRAO



Bilateral CRAO



Branch Retinal Artery Occlusion

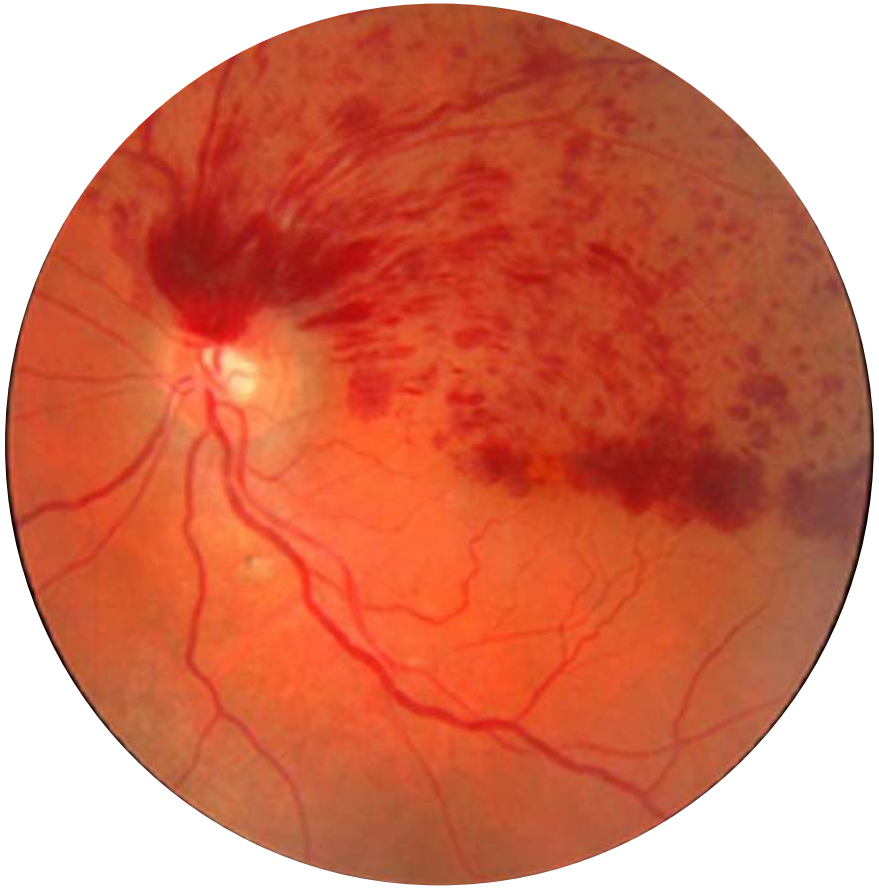


Follow up for Artery Occlusions

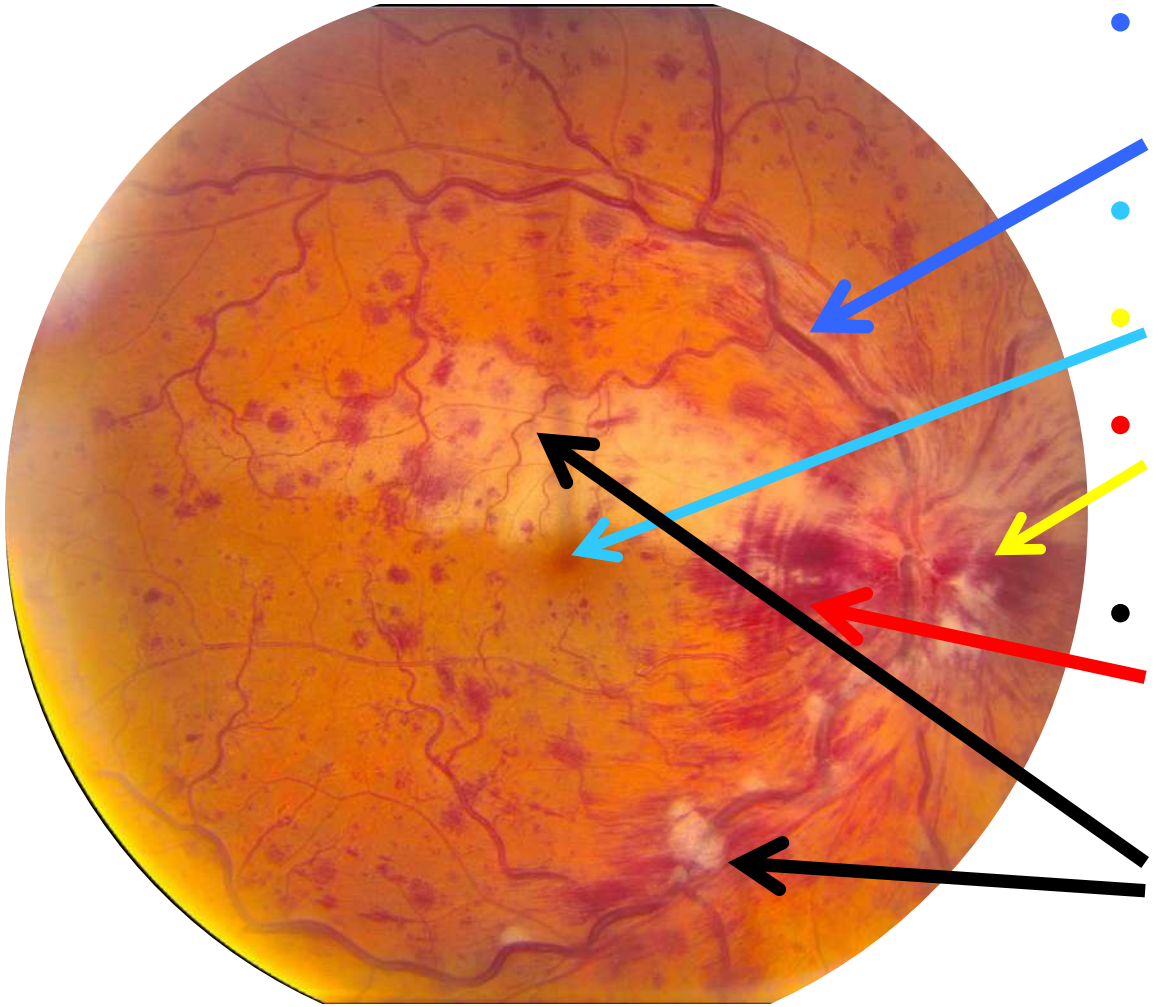
- Neovascularization may occur in patients with CRAO.
  - It may involve the retina, iris, or iridocorneal angle.
- The development of neovascularization necessitates prompt panretinal laser photocoagulation and/or IVAnti-VEGF to decrease retinal oxygen demand.

Retinal Vein Occlusion

- Venous obstruction releases contents of the bloodstream (water, blood cells, and cholesterol) into the retina
- Plumbing problem
- Chief Complaints: blurred vision, scotoma, metamorphopsia
- Treatment: manage underlying vascular disease (HTN, DM, OSA) and/or glaucomas, retinal lasers, intravitreal anti-VEGF and/or corticosteroids

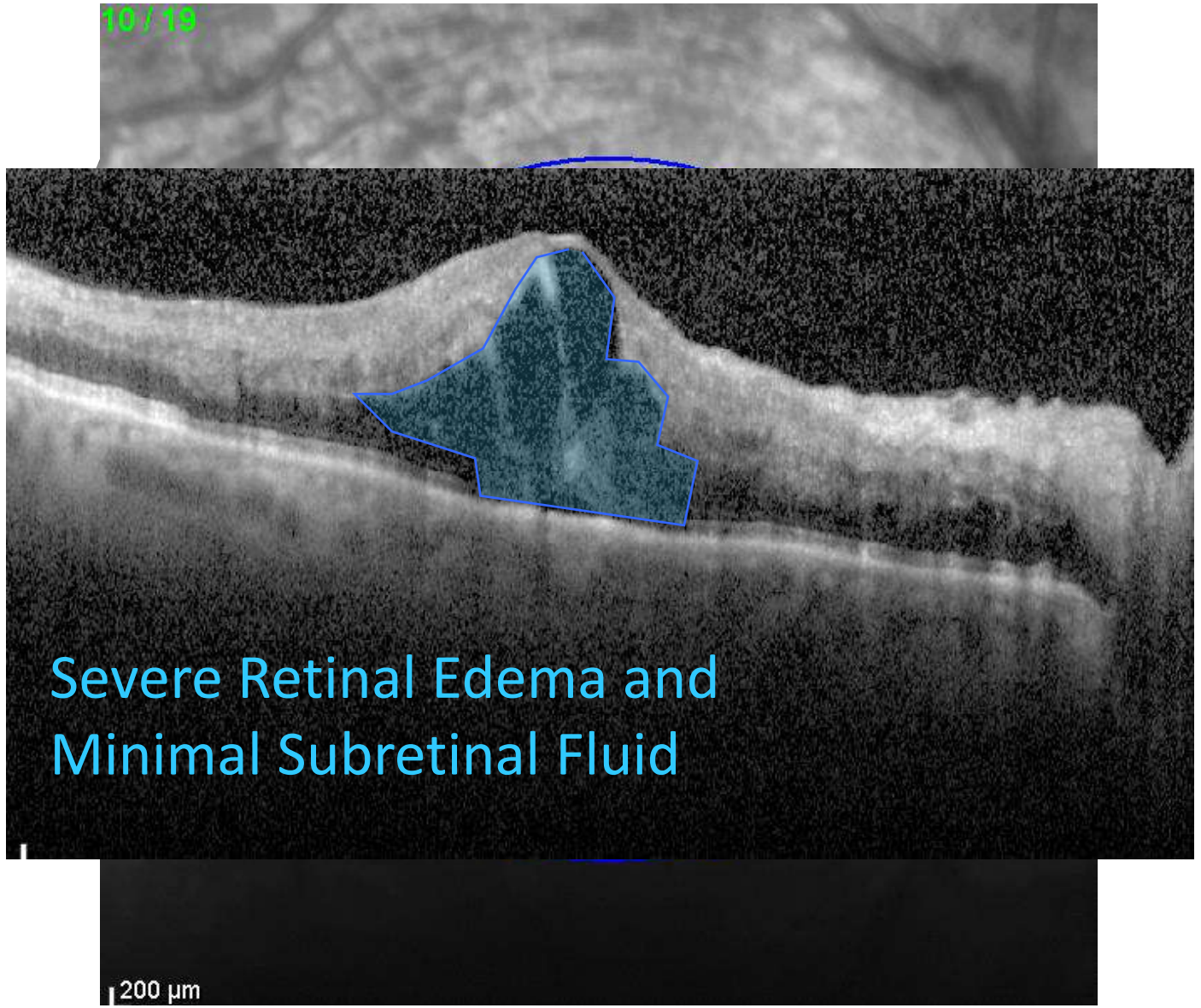


Central Retinal Vein Occlusion

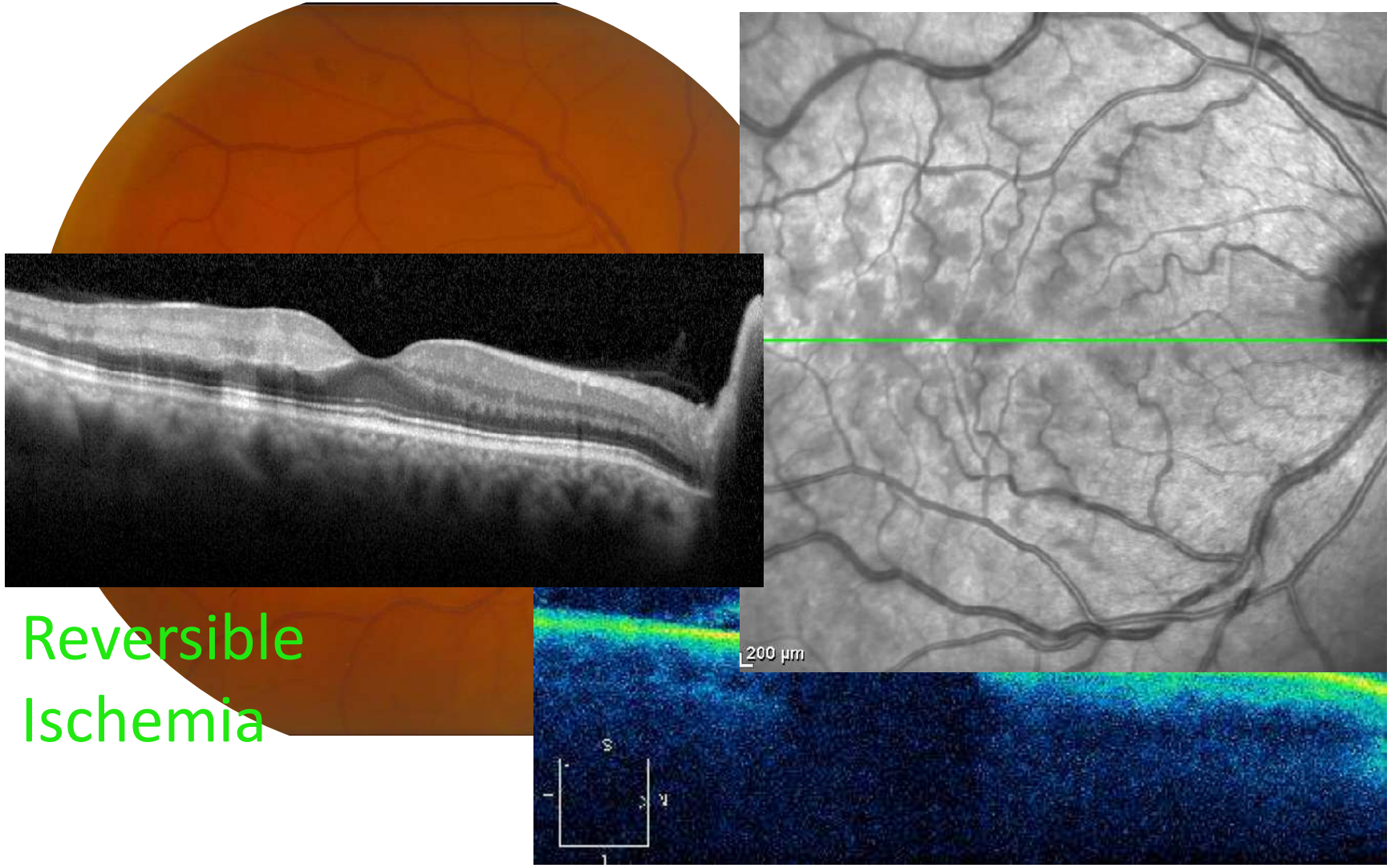


- **Venous Dilation and Tortuosity**
- **Retinal Edema**
- **Optic Disc Edema**
- **Intraretinal Hemorrhages**
- Ischemia: CWS, BRAO

CRVO

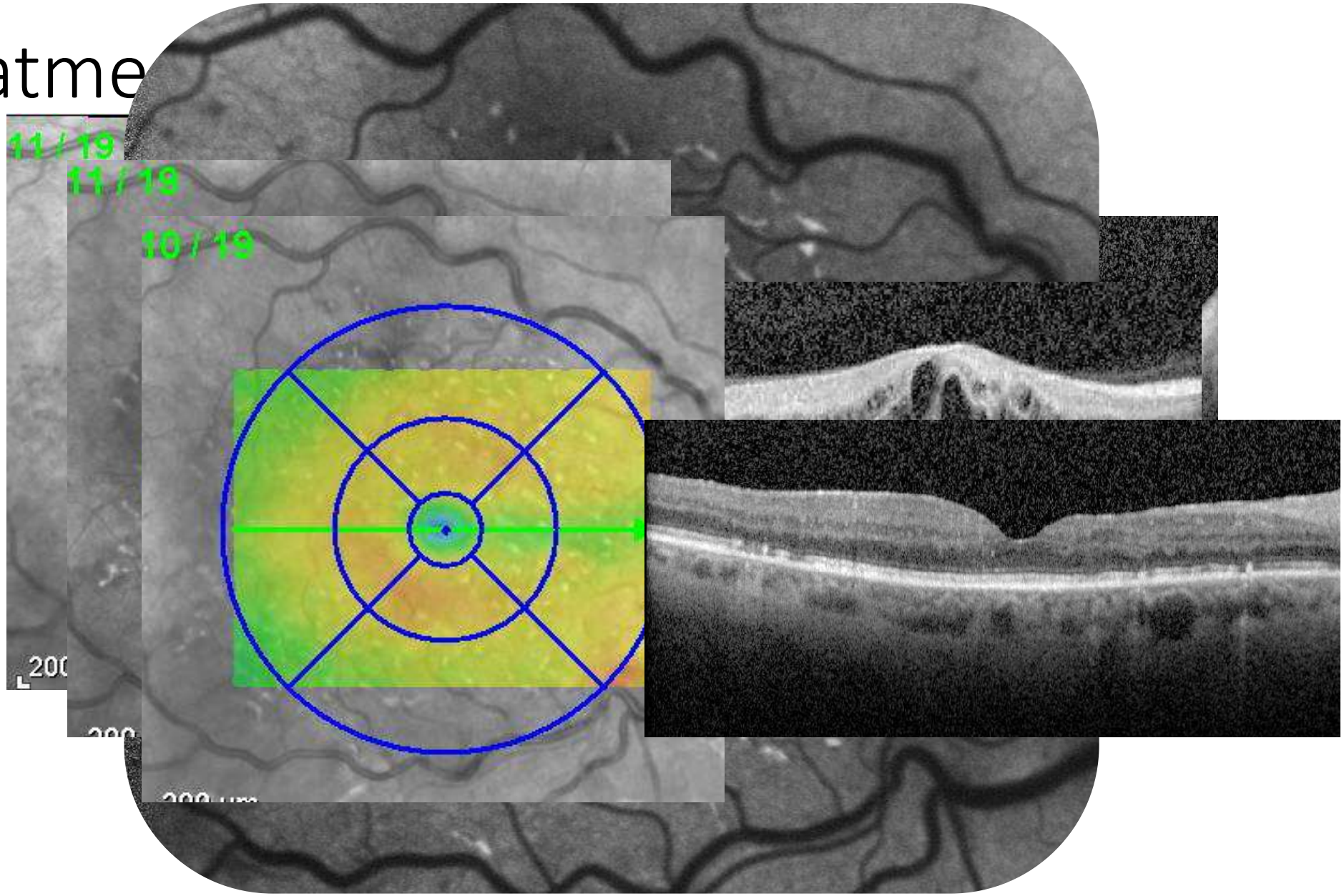

10 / 19
Severe Retinal Edema and
Minimal Subretinal Fluid
200 µm

Unusual Manifestations



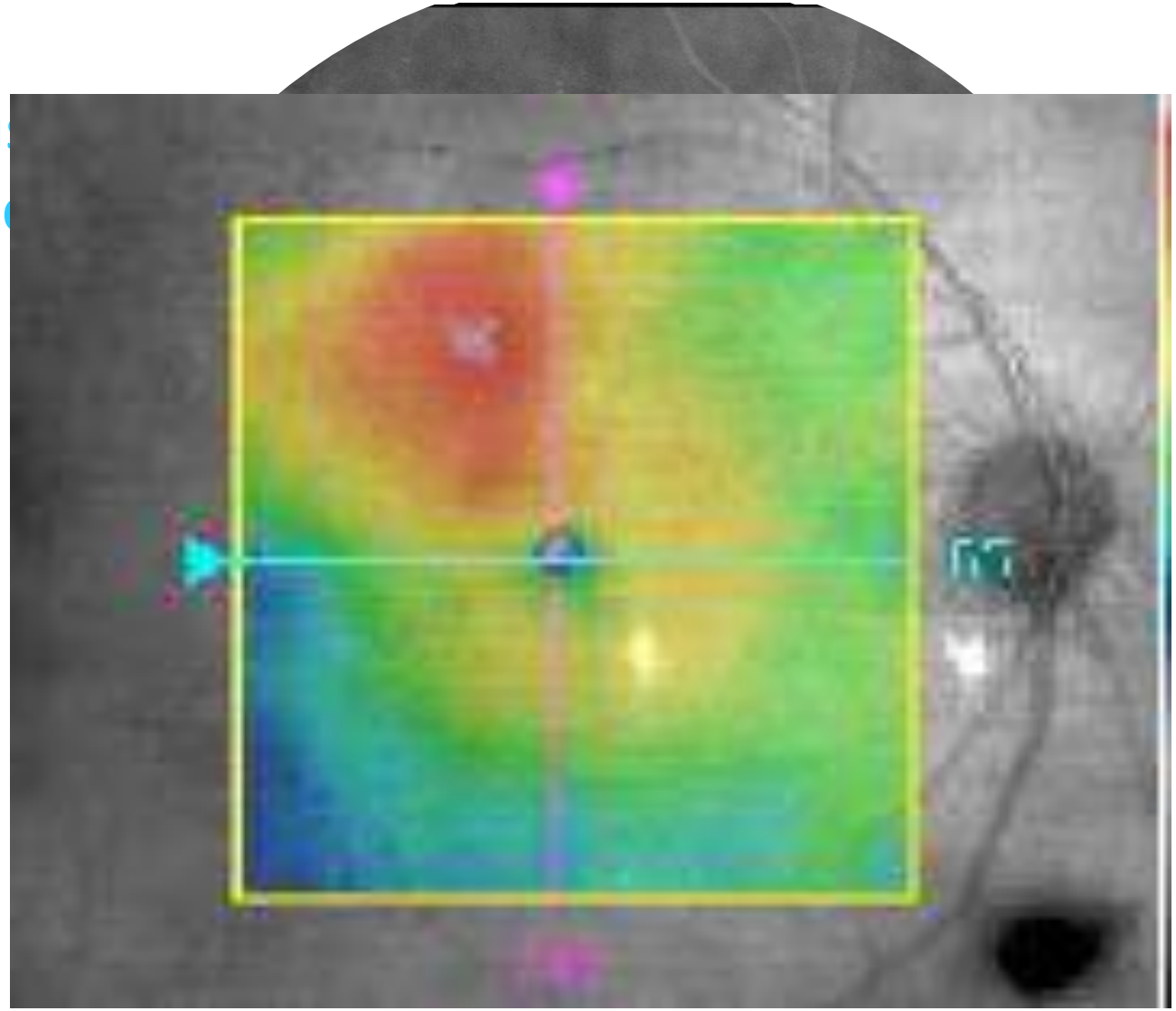
Reversible Ischemia

Treatme

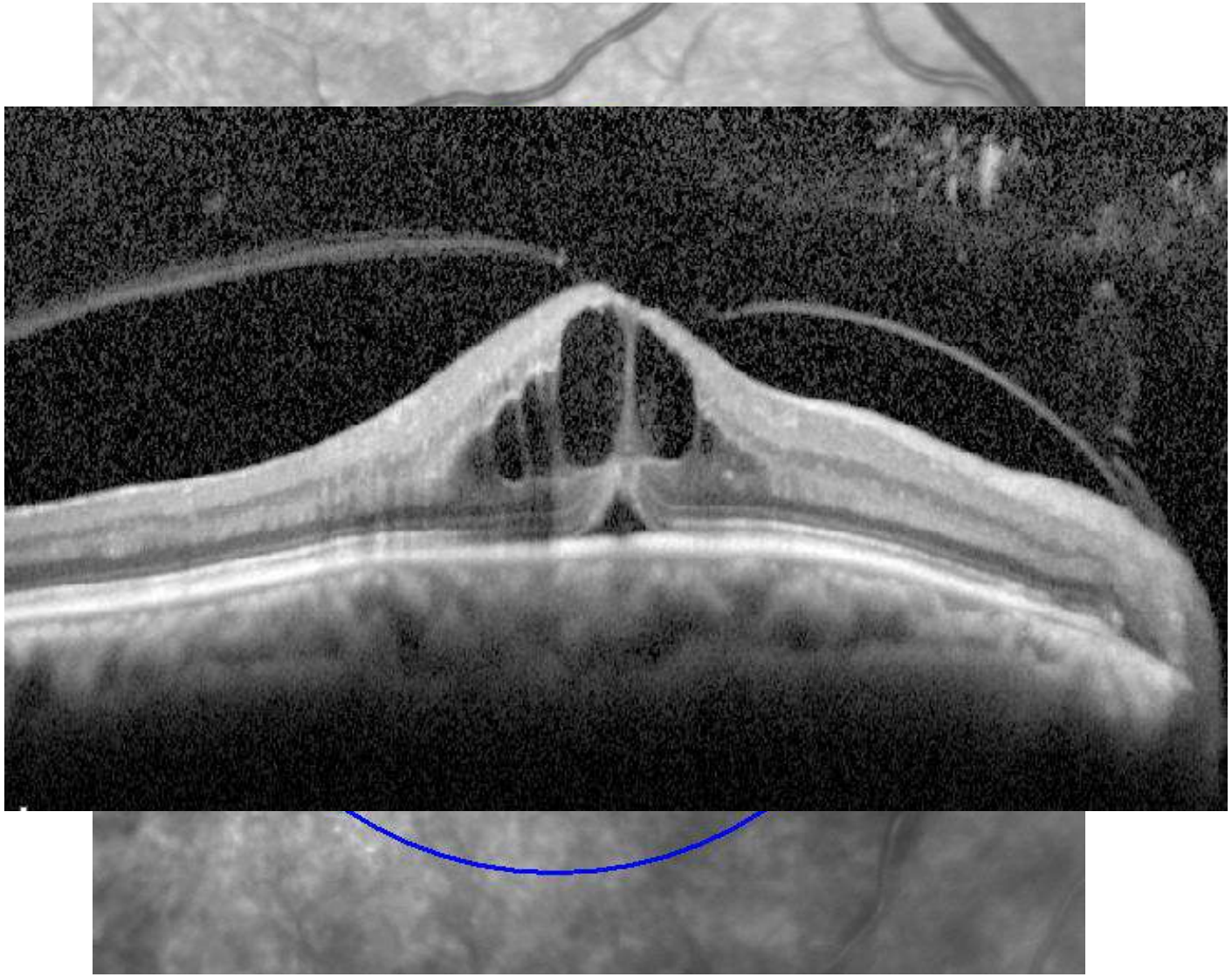


Branch Retinal Vein Occlusion

Veinou
Obstru



BRVO

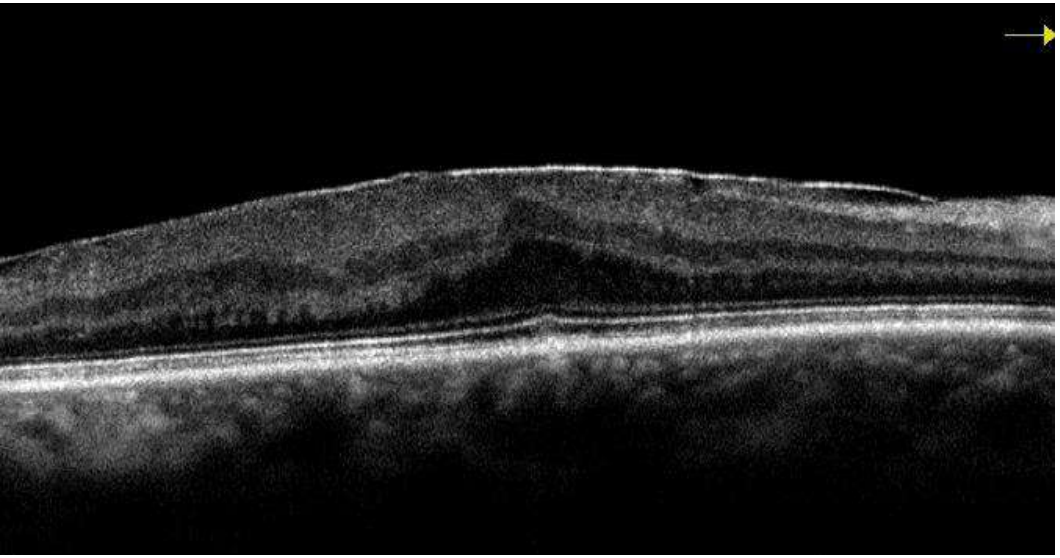


New patient with ”Blurry vision”

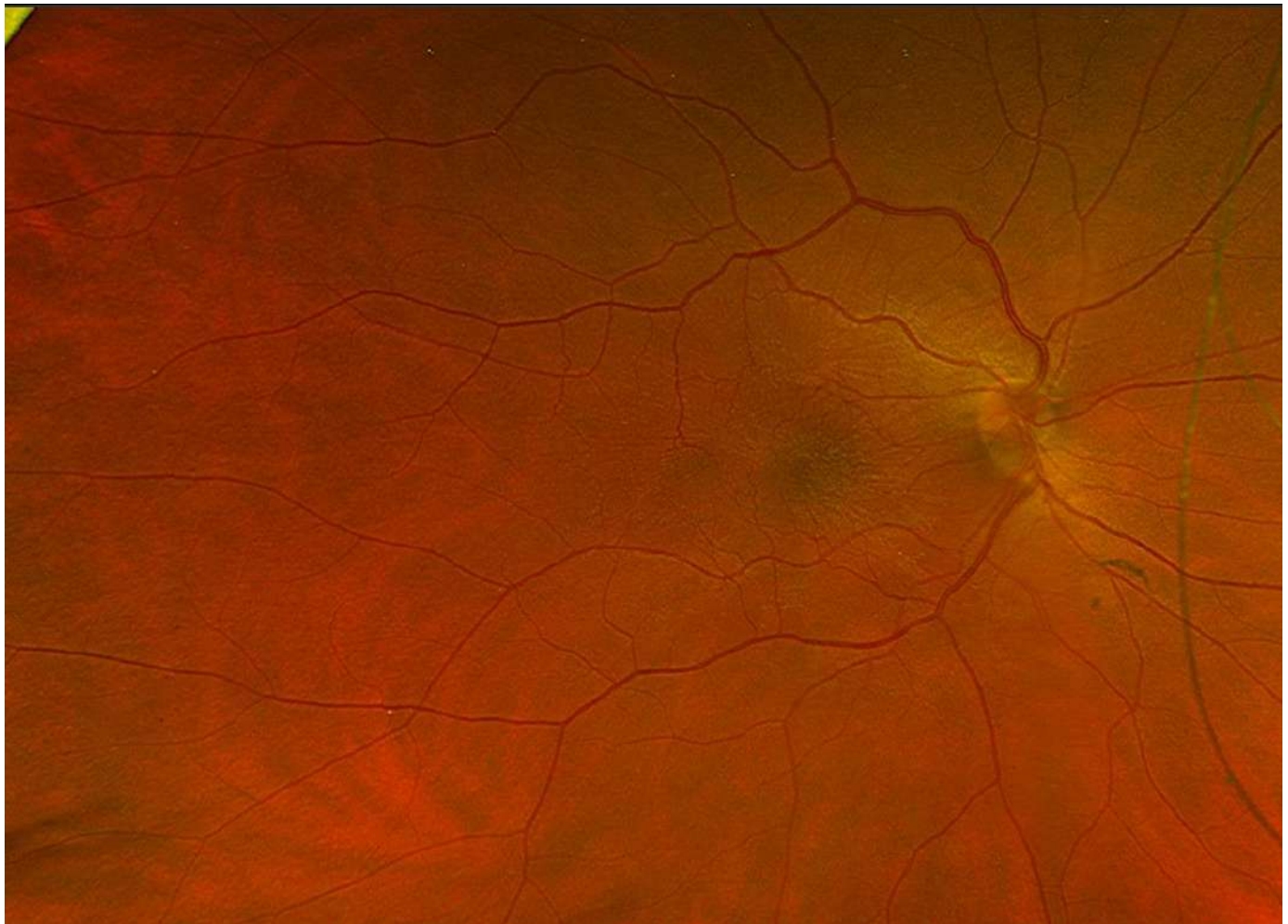
- 64-yo female w ccc: “Numbers are getting harder to see”
- Experiences starbursts when driving at night
- Eyes are dry on and off, tried expensive drops, but the cheap tears work just as well
- Health history: Asthma, HTN, high cholesterol, allergies
- Refraction of +3.50 OU 20/20
- Anterior seg normal, no SPK OD and mild SPK OS
- IOP: 13 OU
- ONH: .2 OU
- Any guesses yet?

Does this look like a 20/20 eye?

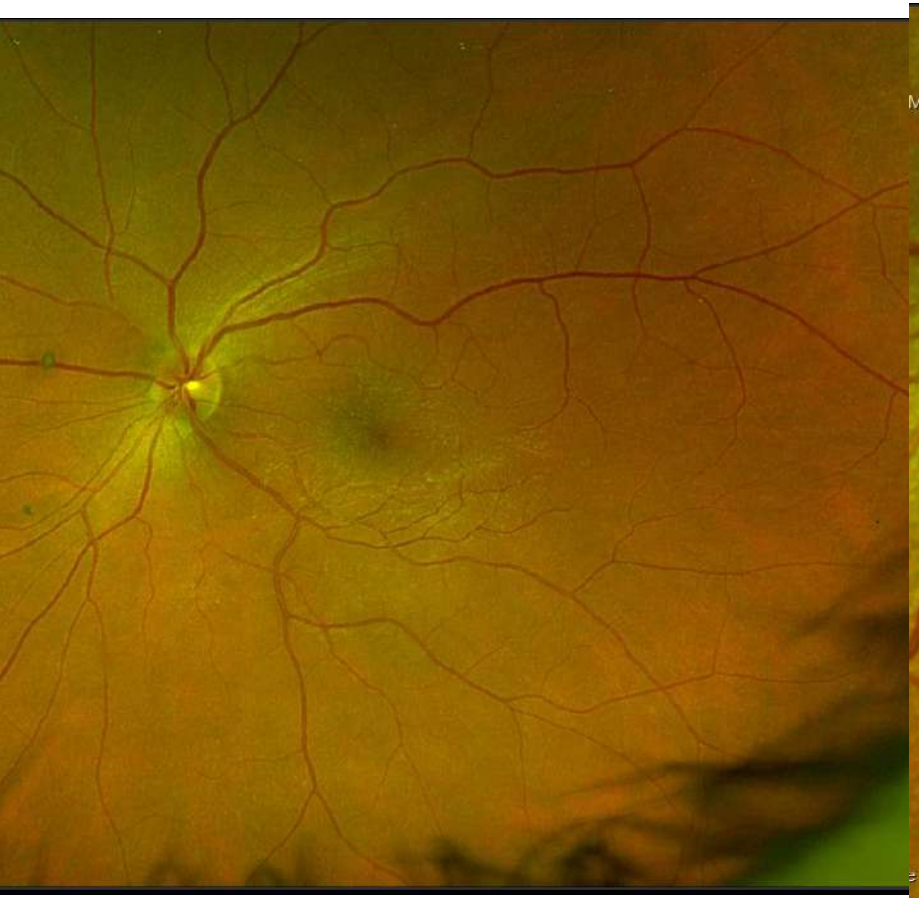
OD OCT

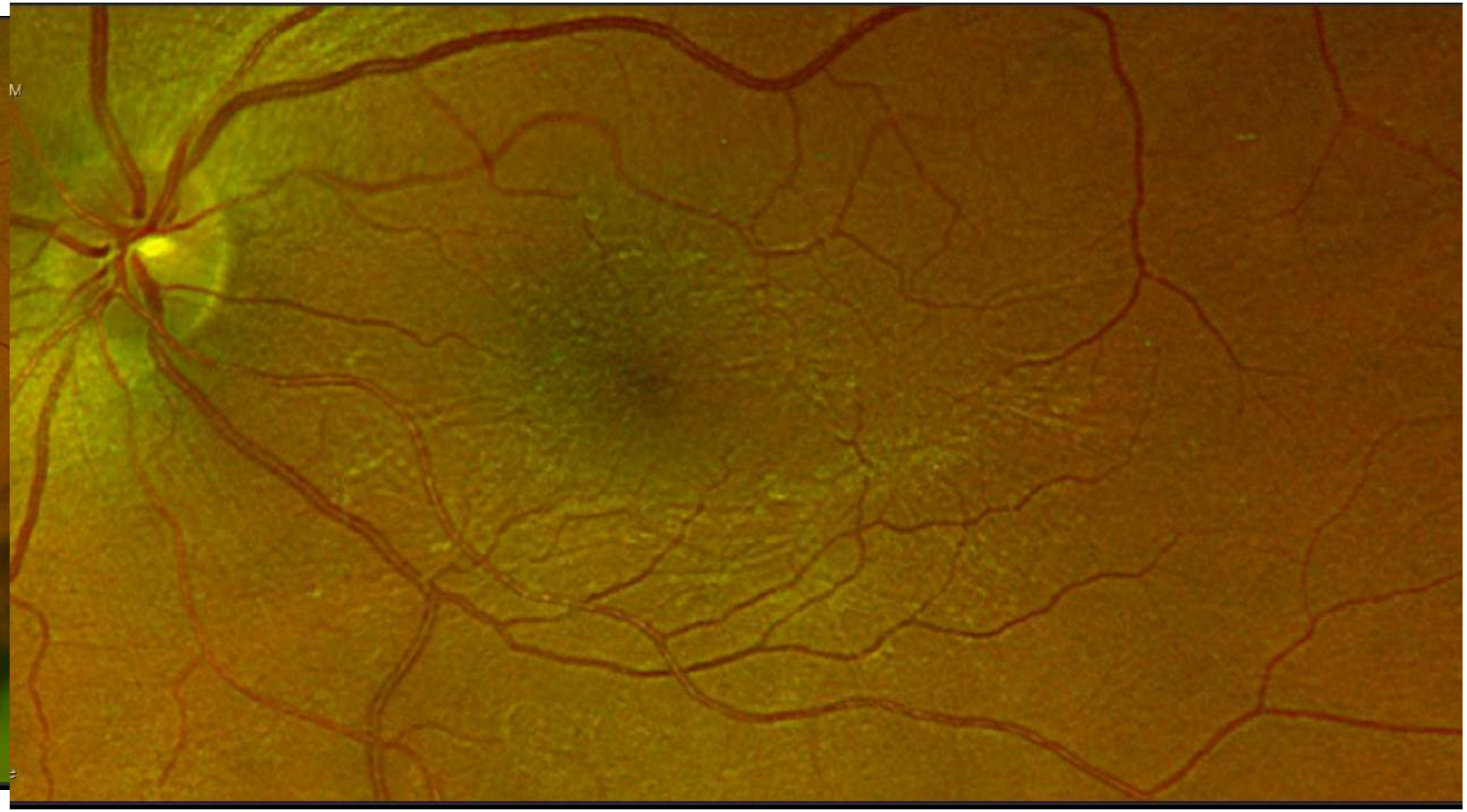


OD Fundus image

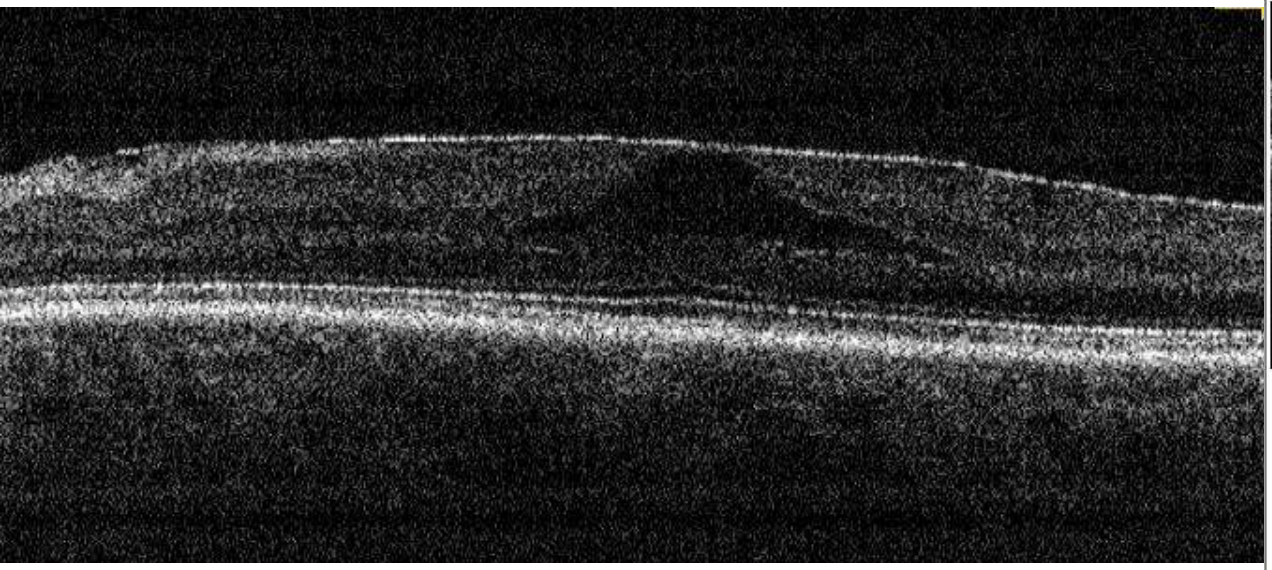


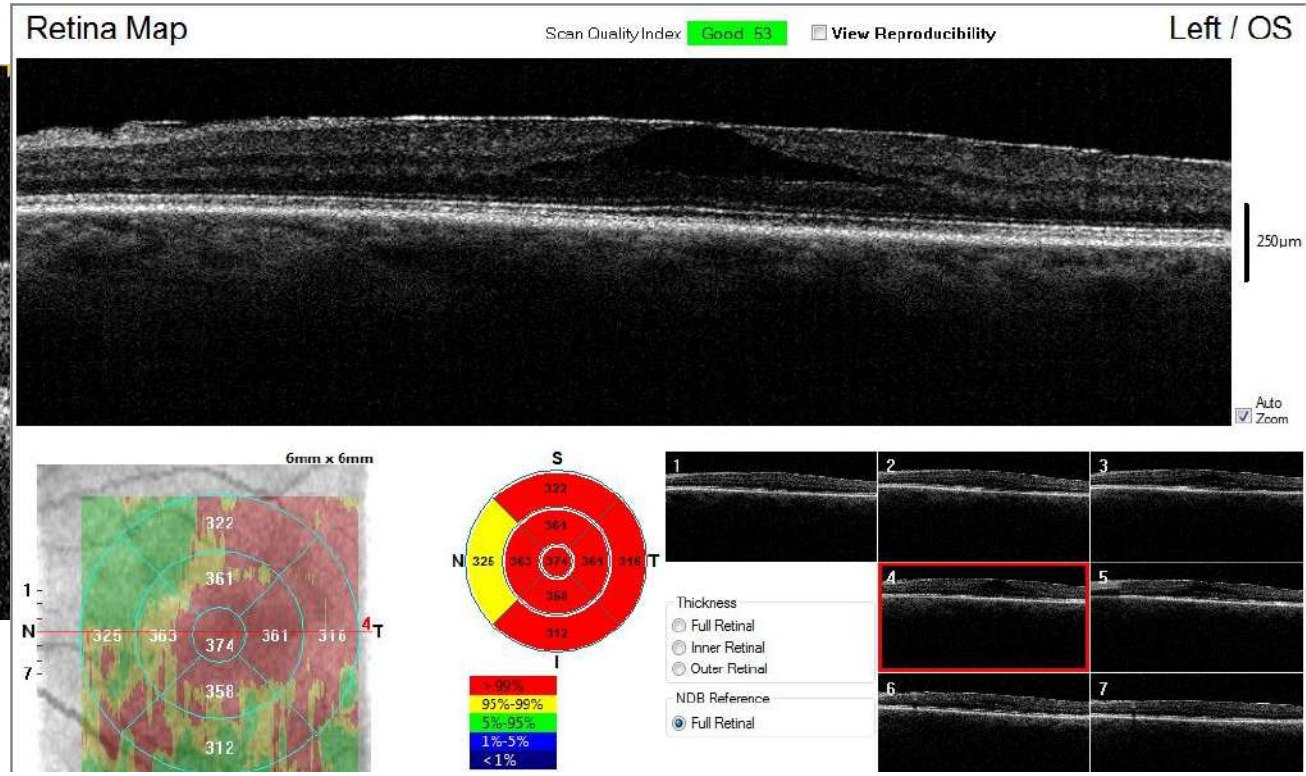
What about this one?





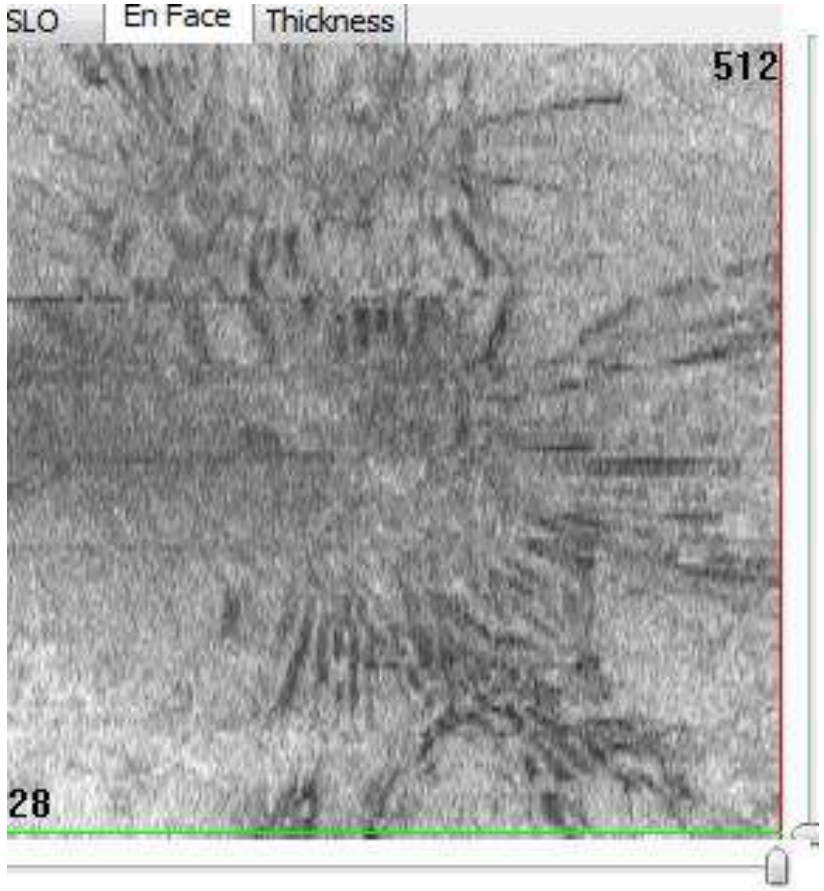
Now what do you think?



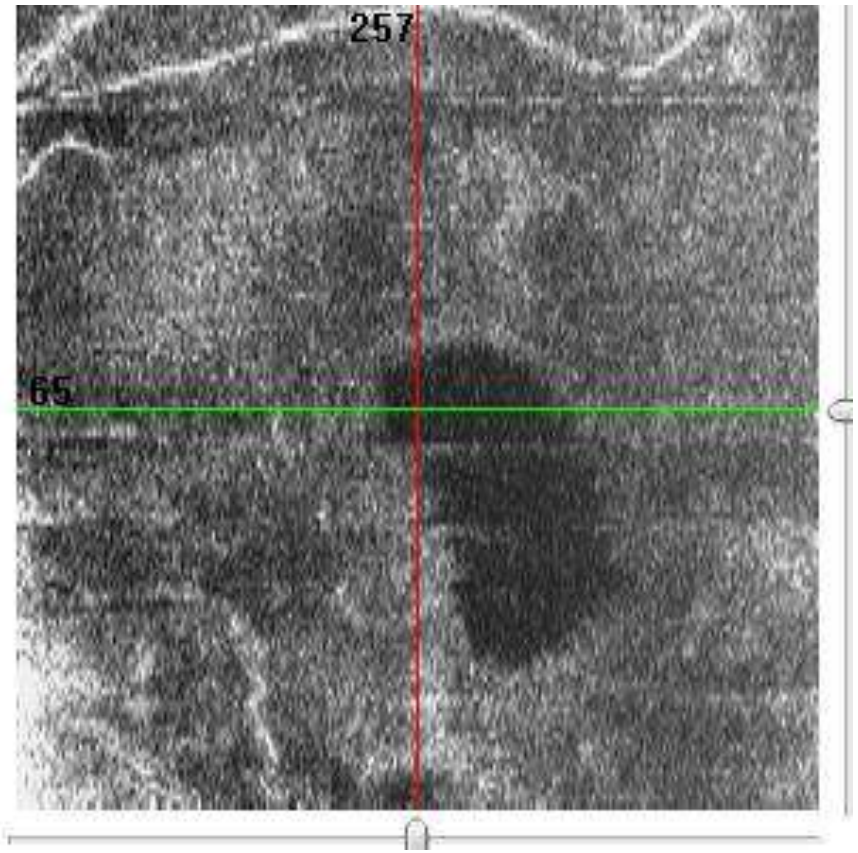


No OCT-A…no problem (kind of..)

En-Face OD



En-Face OS showing “space”



WWYD?

- Keep in mind patient is 20/20
- Presented with option of referral to retina for evaluation of surgery
- Patient wants to know “Is this macular degeneration” and “doesn’t want any new doctors, I’ll come back to you in 6 months and you can tell me how I’m doing”

- Anything else to be done????

We should be >½ way through

Now we're supposed to talk about AMD and DM/DR…

Which could be 2 hours each!!!

So...lets have some fun and do a few images, touch on a few topics and some Q&A

Stats

- AMD is the third leading cause of legal blindness worldwide
- The LEADING cause of legal blindness for age 65 and older in the USA
- Unfortunately, the number of those affected is growing in the USA
  - Currently 11 million have AMD and 22 million are projected to form AMD by 2050
  - 1.7 million adults were registered as Advanced AMD (with GA) in 2010 and is projected to grow to 3.8 million with Advanced AMD by 2050

Why is Age Related Macular Degeneration such a blinding condition?

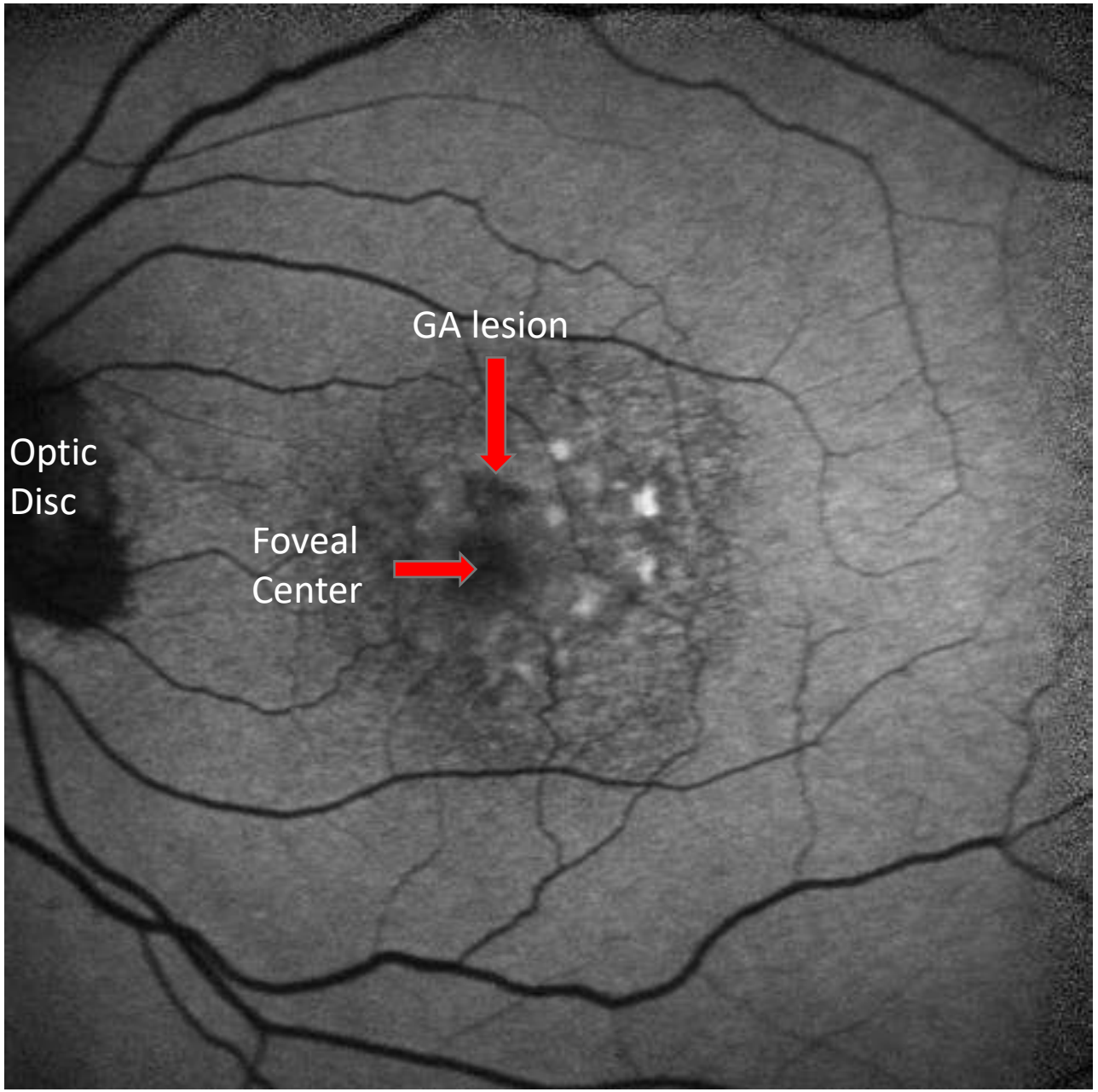
- It is a relentless anatomical progression of dying retina leading to geographic atrophy
  - DRY AMD: 80-90 percent
  - WET AMD: 10-20 percent

- There are things that we just cannot control that cause AMD:
  - Genetics
  - Aging
  - Environmental Stressors

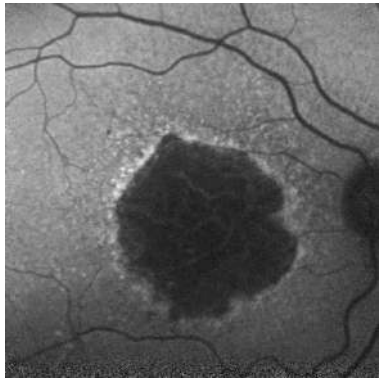
- To date, we have no cure for GA

Why is AMD such a blinding condition?

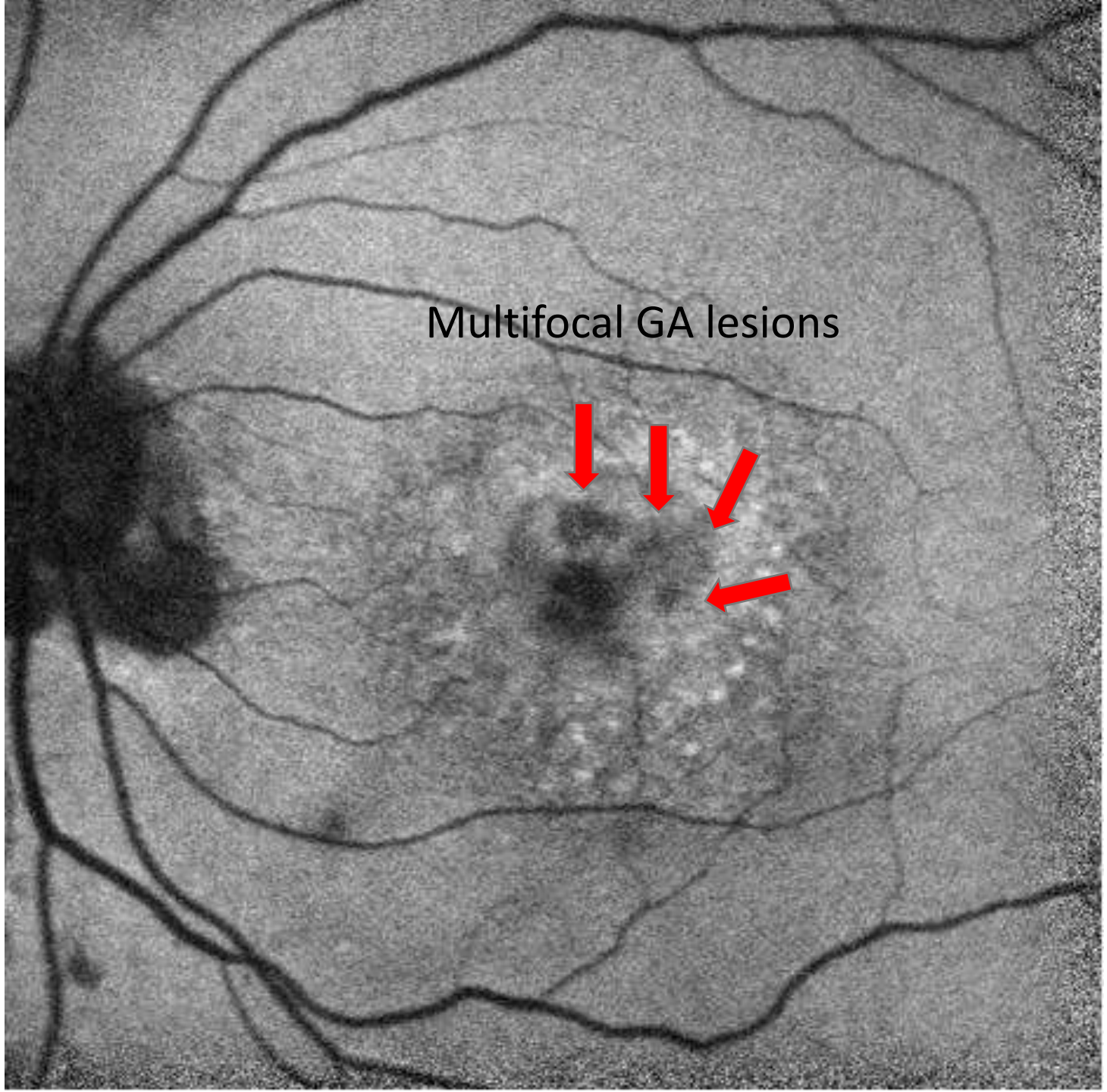
- While the disease state of AMD may appear to grow slowly, it is not reversable and is continuously growing.



- Geographic lesions may be unifocal

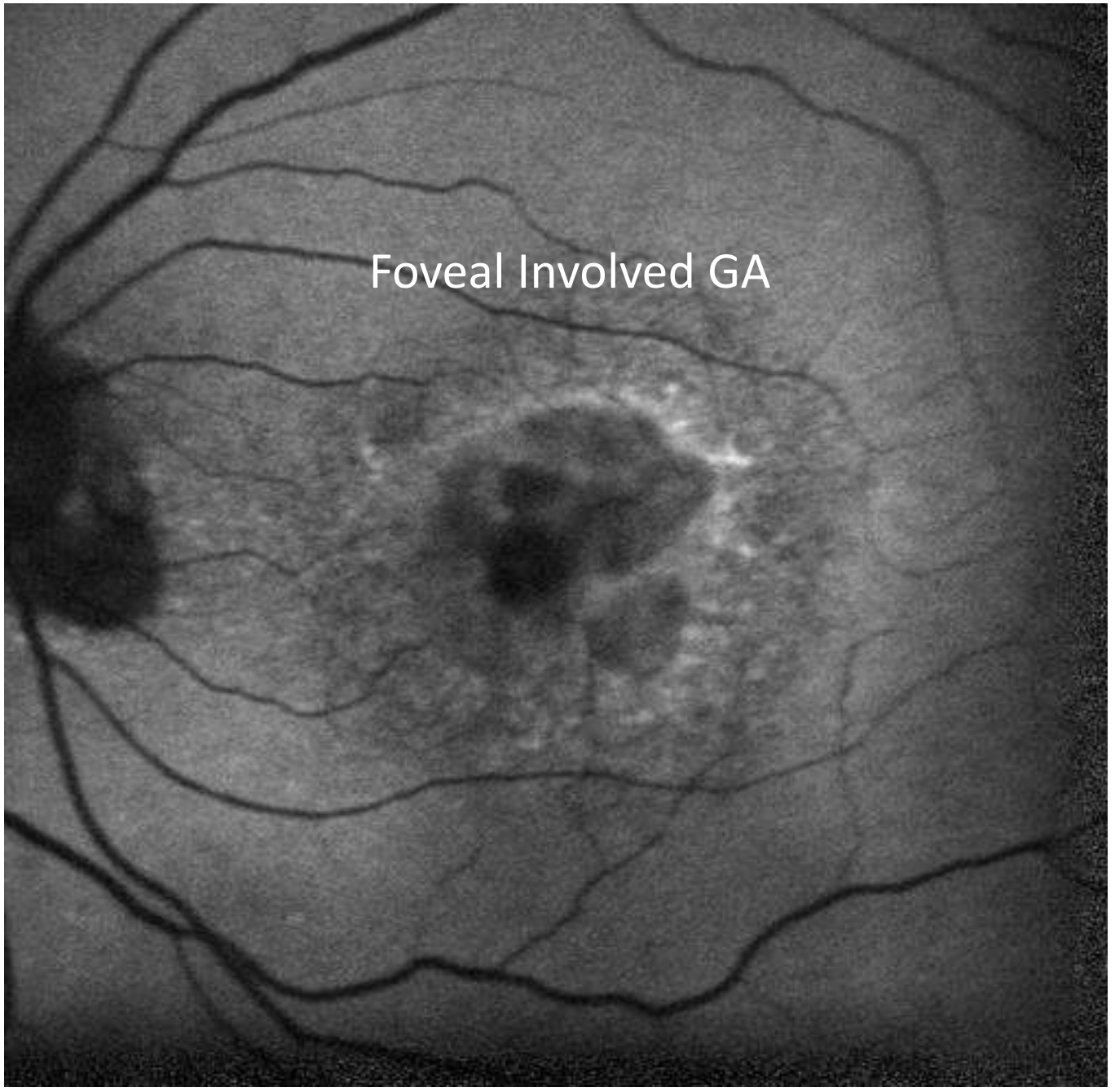


- GA may be multifocal which expand more quickly towards the fovea.
- From the AREDS study:
  - Of the 397 patients who developed central GA, the median time to central encroachment of fovea was 2.5 years from diagnosis.(N=3640)

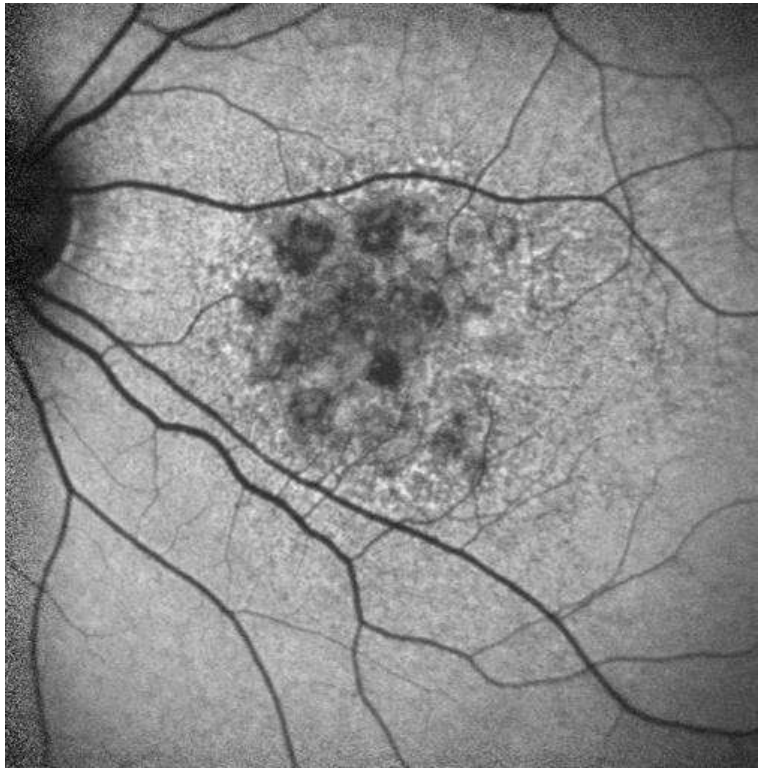


The larger the geographic lesion, the quicker the growth.

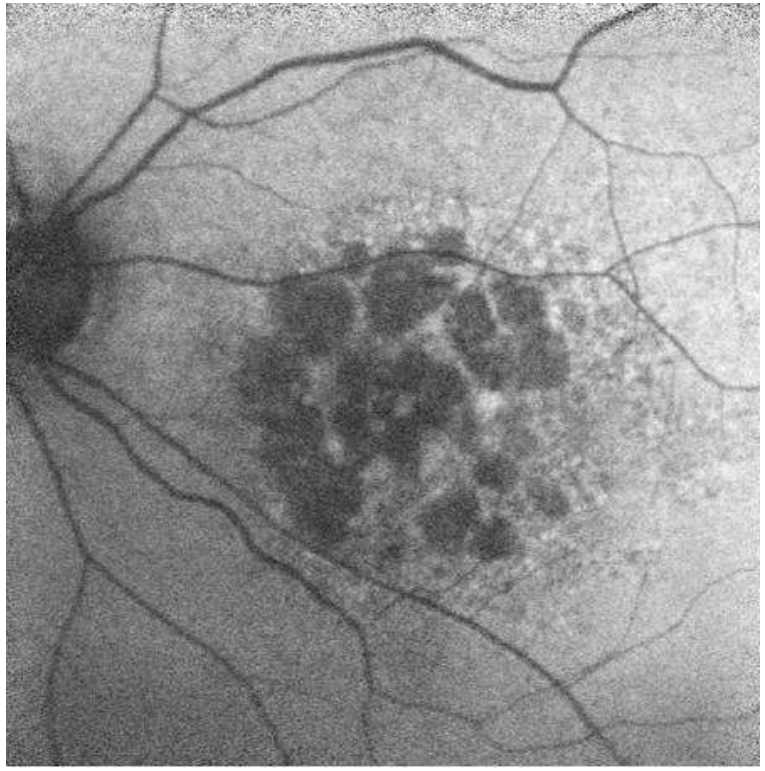
Lesion growth may affect the vision, even before the fovea is affected by GA.



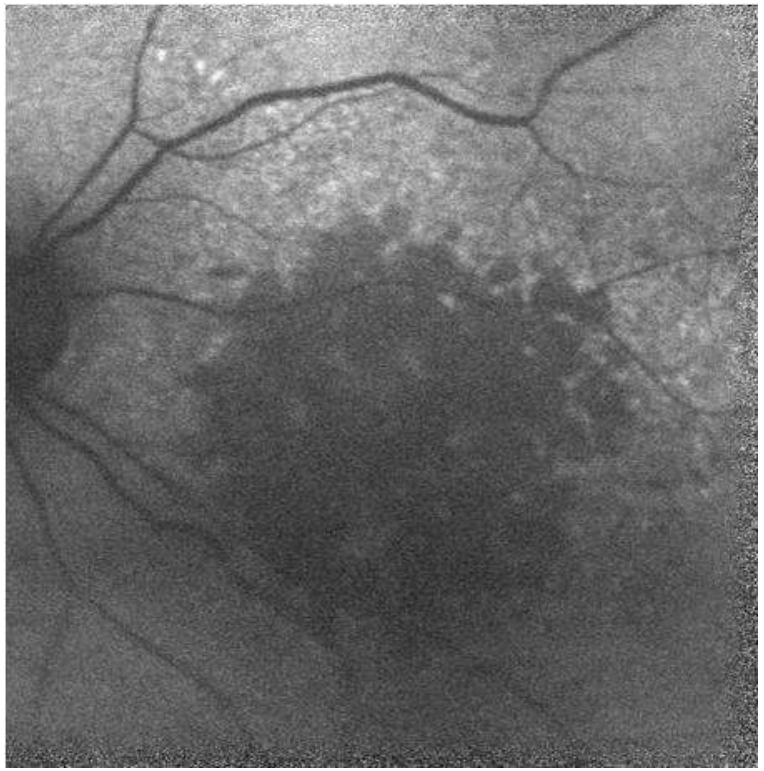
This is why AMD is such a blinding condition…



Year 1 BCVA 20/30



Year 2 VA 20/50

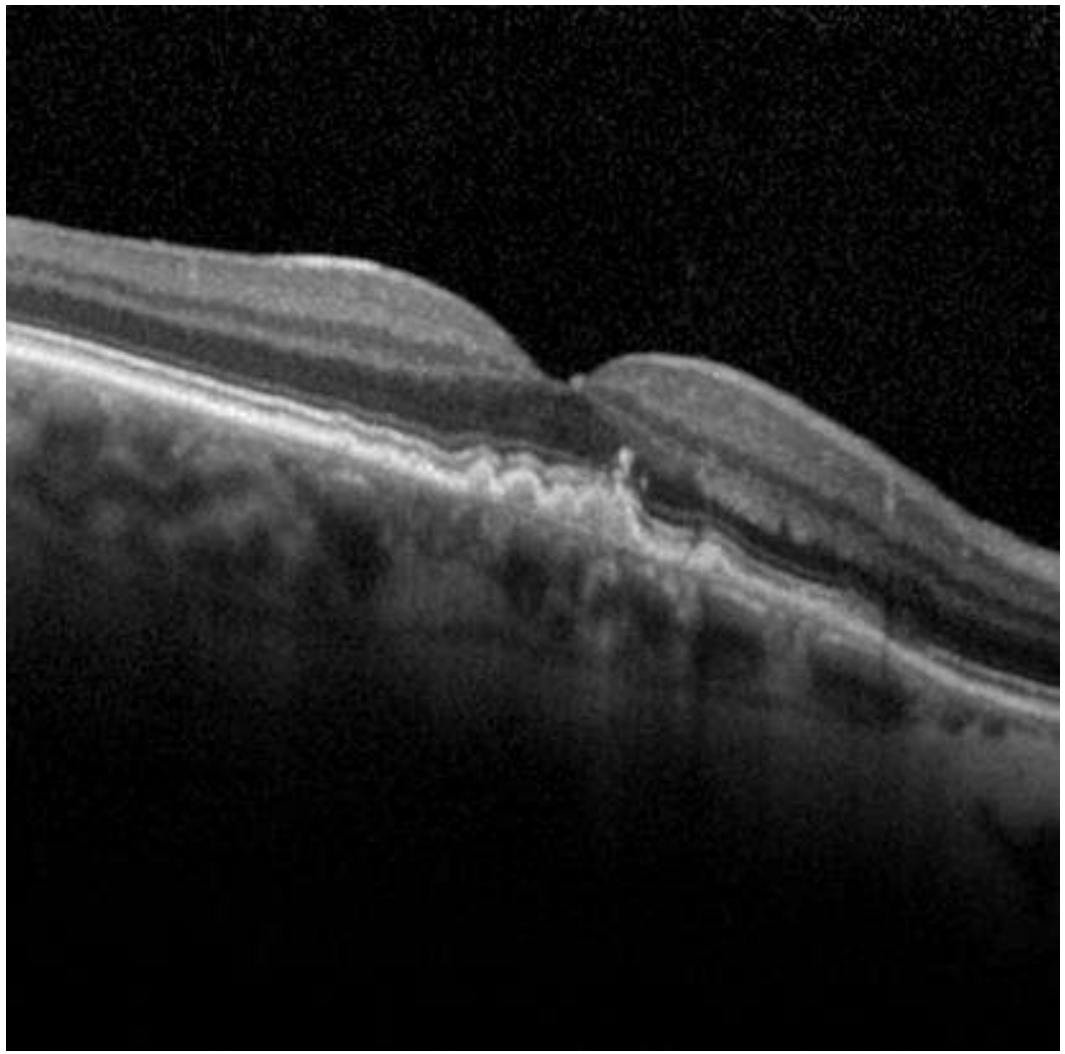


Year 5 VA 20/400

CAM: Classification of Atrophy Meeting

- In addition to drusen and pigment changes in the fundus, we have learned of many other risk factors for developing GA in AMD.
- An international group of experts surveyed the existing literature, performed a masked analysis of longitudinal multimodal imaging for a series of eyes with AMD, and reviewed the results of this analysis to define areas of agreement and disagreement.

- Defined biomarkers and nomenclature in geographic atrophy
    - SD-OCT helps to identify and differentiate the atrophy as it progresses

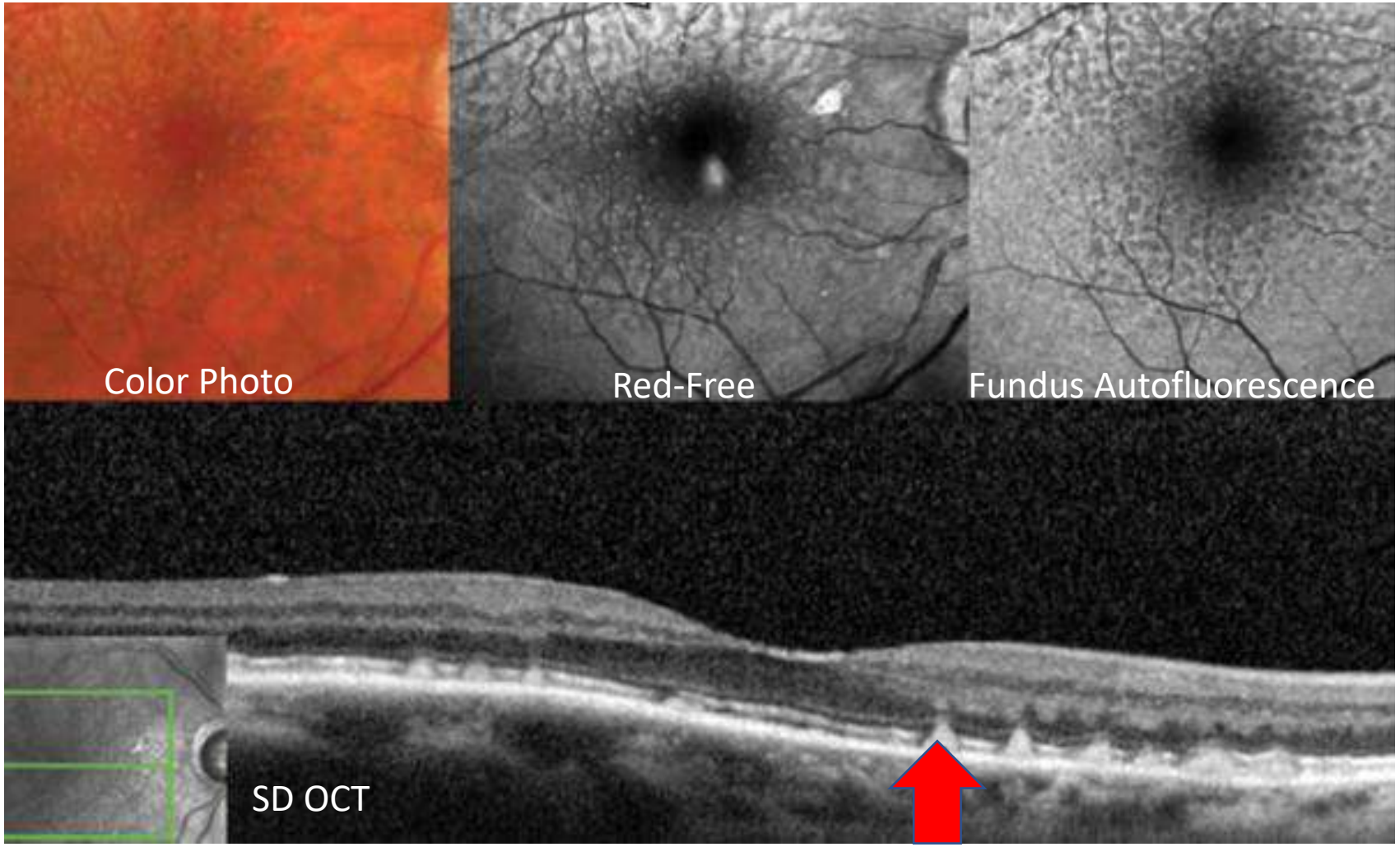


Hyperreflective Foci

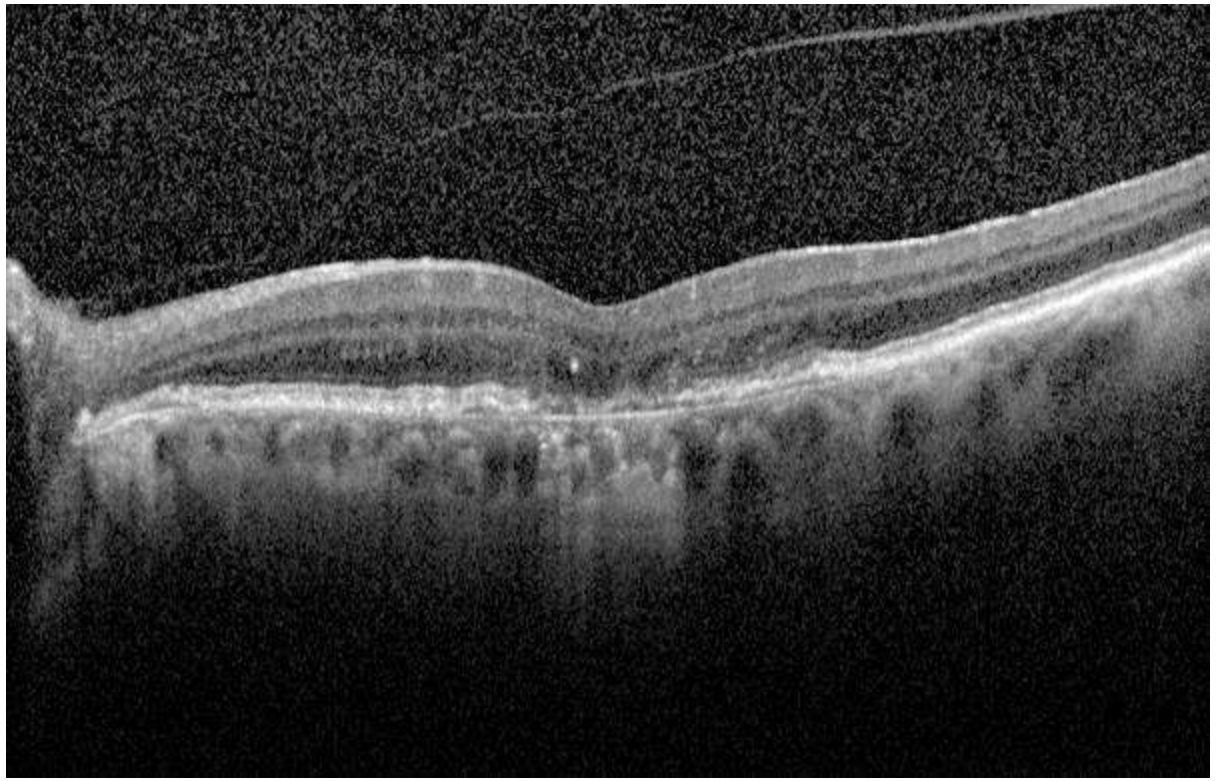
- Punctate intraretinal lesions
- Often at drusen apex
- Likely represent pigment granules
- Originate in the outer retina and migrate inward with time
- 5 x more likely to form GA within two years

Subretinal Drusen Deposits (SDD)

- AKA reticular pseudodrusen
- Difficult to distinguish from true drusen on color photography
- SD-OCT allows us to see the location as deposits in the subretinal space above RPE
- Early stage: granular hyperreflective deposit below EZ
- Progression is noted when material accumulates into small mounds that break the EZ.
- FAF: target shape with hypo- or isoautofluorescent surround. Collectively forms reticular pattern



SDDs = 2-6 x higher risk for GA



Hypertransmission defects signify a high risk towards nascent GA.

Hypertransmission defects

- Increased hyperreflectivity in the choroid as a result of RPE disruption.
- The overlying RPE may “appear” intact and unaltered, however hypertransmission defects indicate loss of integrity of the RPE.

VOCAB
per the CAM group

Nascent GA/iRORA

- **Nascent GA**: “subsidence” or collapse of the outer plexiform layer (OPL) and inner nuclear layer (INL) and a hyporeflective wedge shaped band within the OPL.
- **iRORA**: the same definition as Nascent GA, but includes choroidal hypertransmission defect, signs of photoreceptor degeneration, and RPE attenuation/disruption.

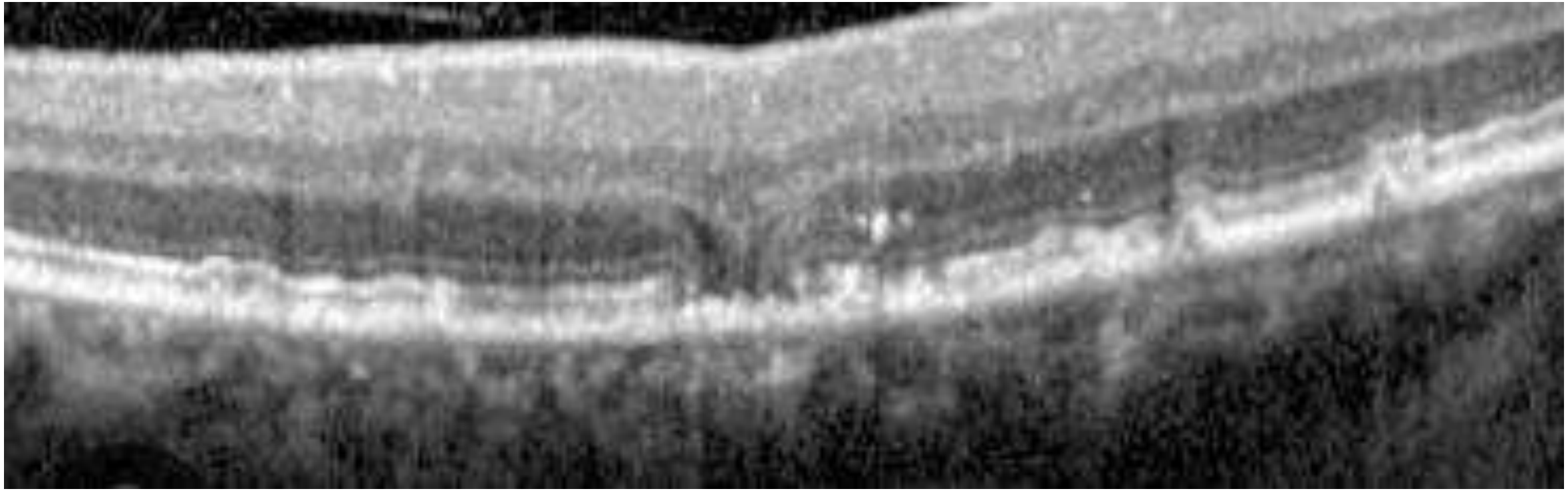
cRORA

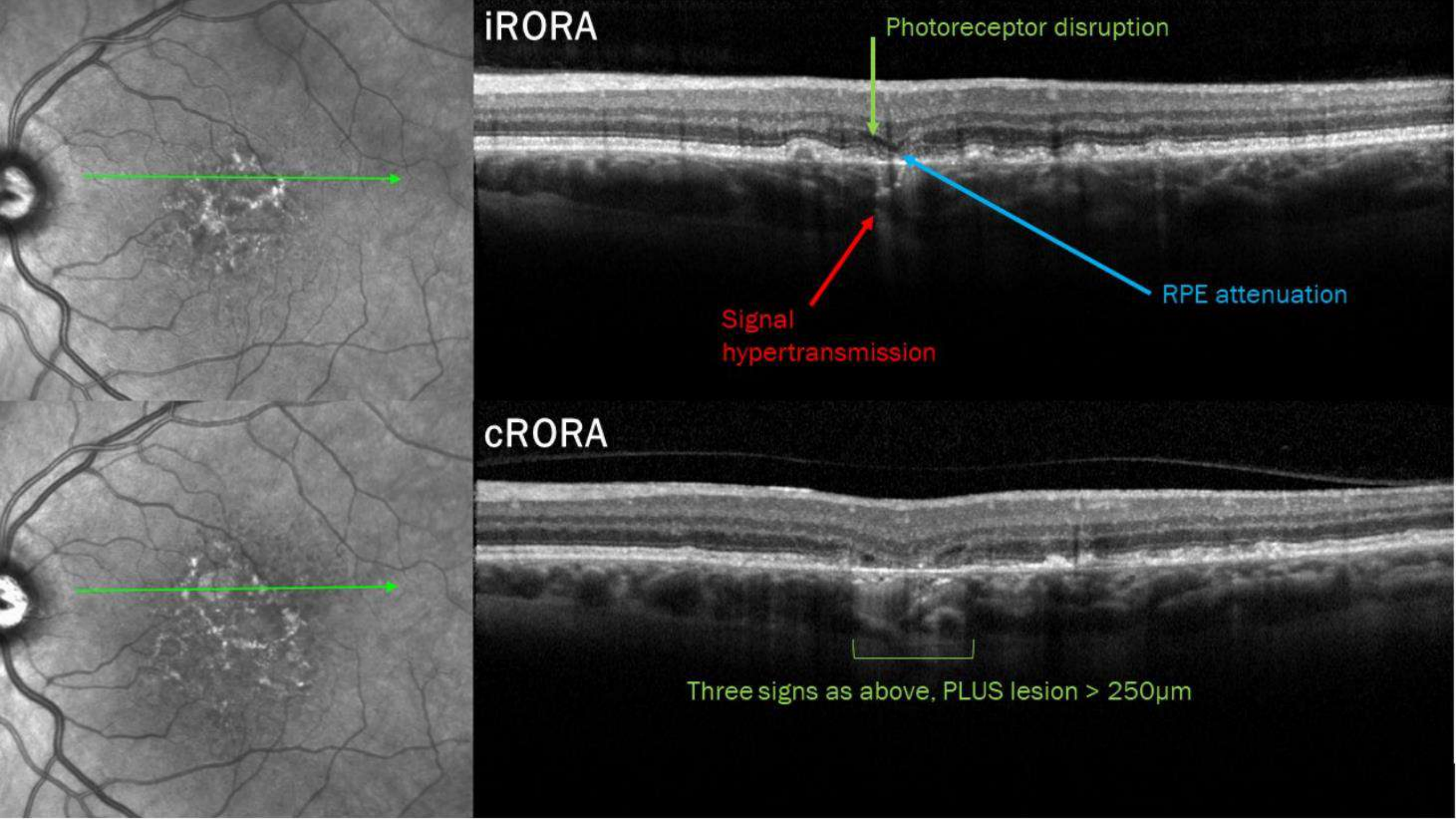
- Choroidal hypertransmission of 250 microns in diameter or greater
- A zone of attenuation or disruption of the RPE of at least 250 microns in diameter
- Evidence of overlying photoreceptor degeneration
  - ONL thinning, ELM loss, EZ or IZ loss
- All occur in the absence of signs of a RPE tear

*RORA=RPE and Outer Retinal Atrophy i=incomplete c=complete

Nascent GA

- 7% of eyes with AMD
- Subsidence of OPL and INL and a hypo-reflective wedge
- Typically in the central 1500 microns of macula




iRORA
Photoreceptor disruption
Signal hypertransmission
RPE attenuation
cRORA
Three signs as above, PLUS lesion > 250µm

Apellis

IVERIC BIO
An Astellas Company

astellas

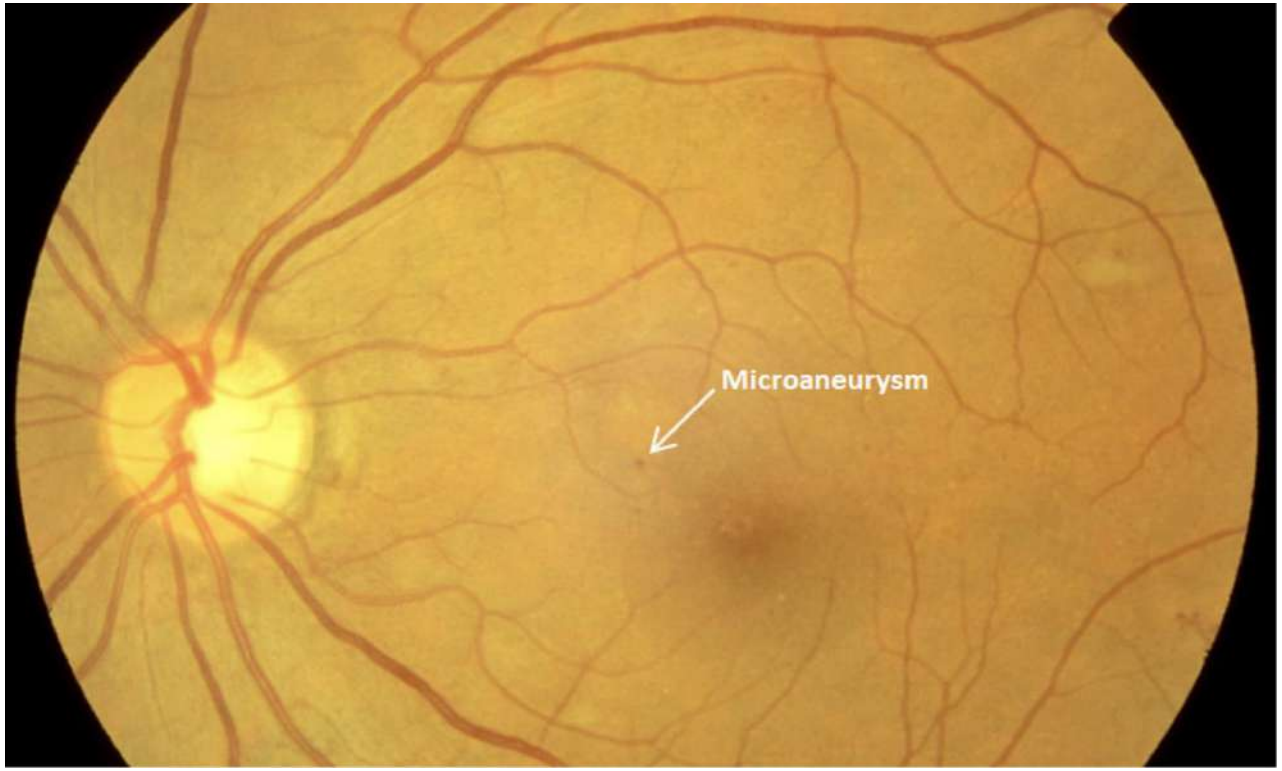
FDA APPROVED

Next topic: Diabetic retinopathy

This could be a 2 hour course easy, so we will make this very casual and open for questions

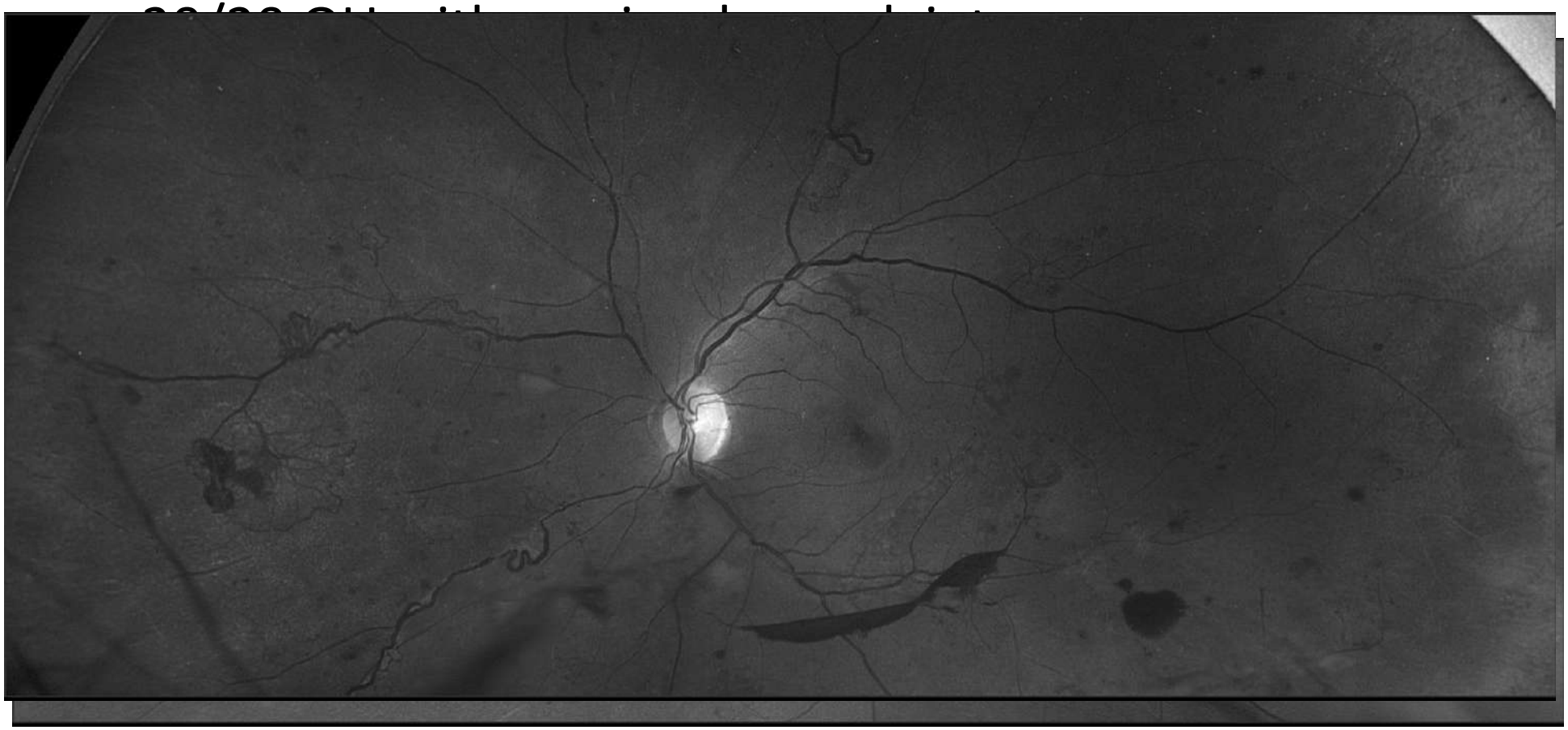
Lets start with a practical discussion

- If you have a patient with mild NPDR, when do you have them return to clinic?
  - Do you consider A1c (historic and current)
  - Do you do ERG
  - Do you consider HTN status
  - What else?



48yo T2DM, A1c 9.0

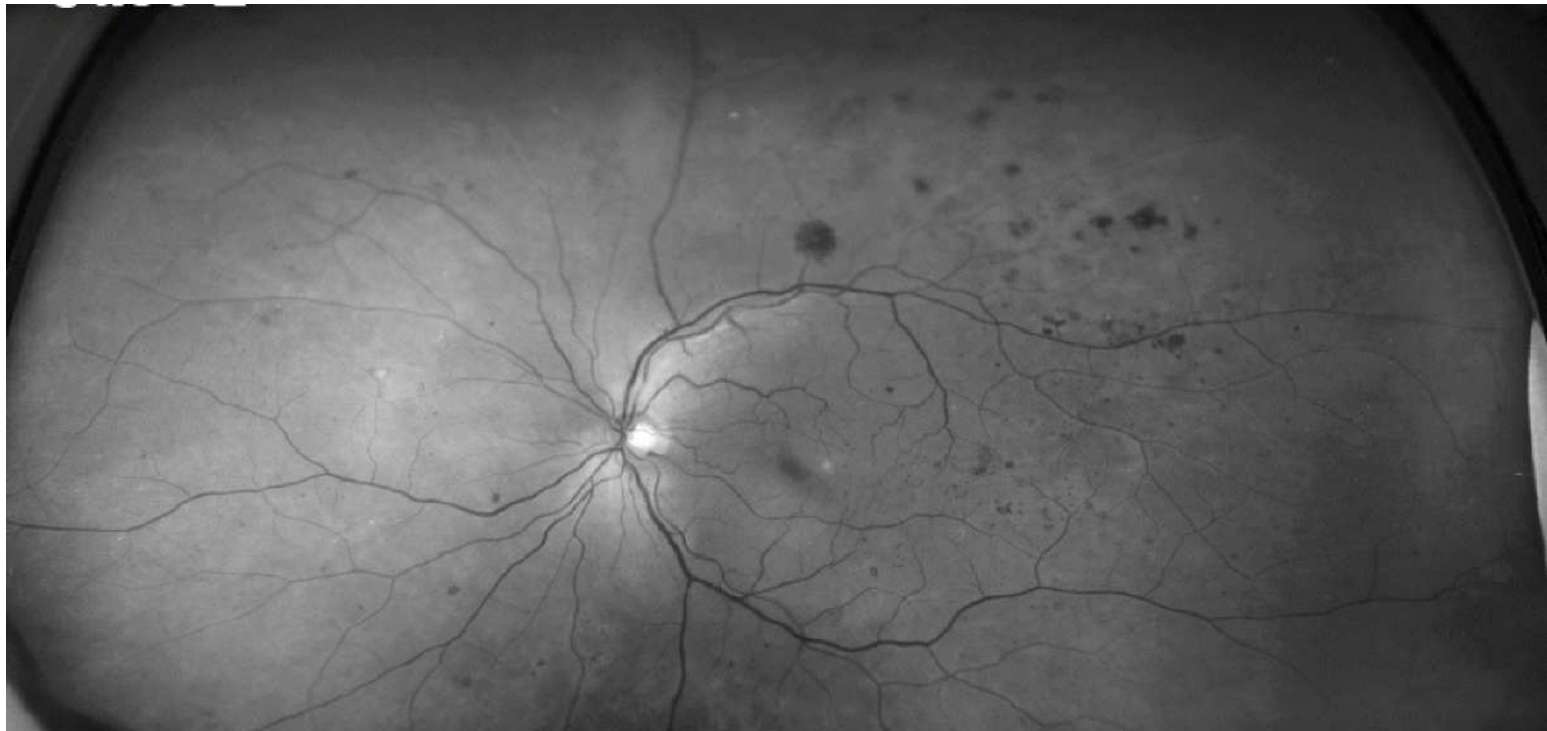
- Doc, be nice to her, she has been “shamed” due to her DM control



What about in between

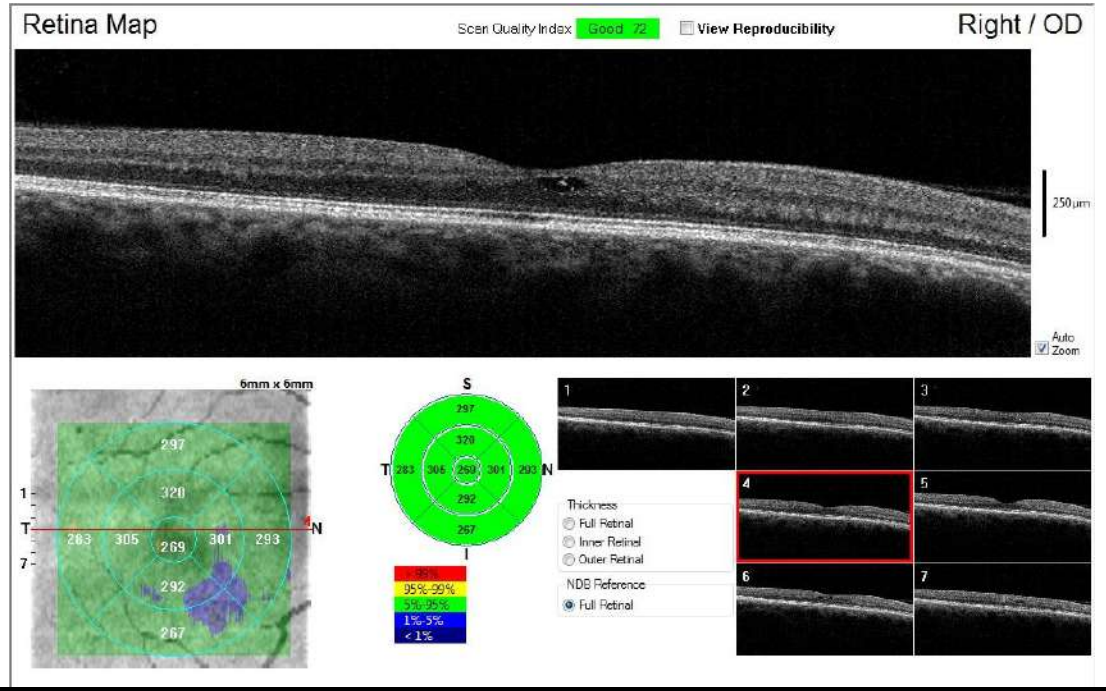
- Mild: No need for referral
- PDR: Obvious need for referral
- What about in between?

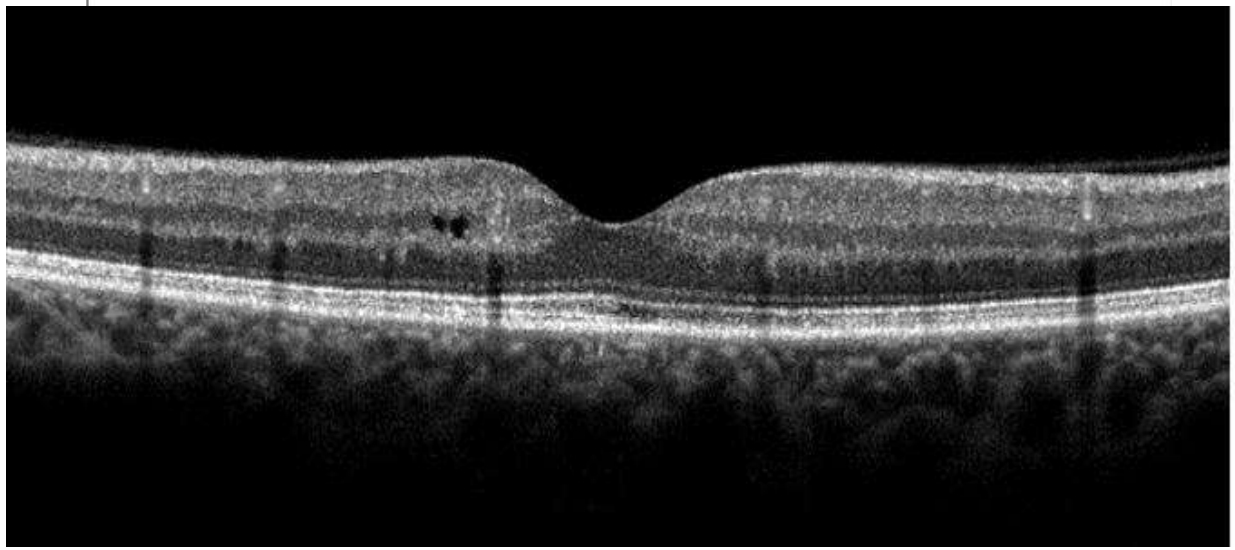
- What is level 47?



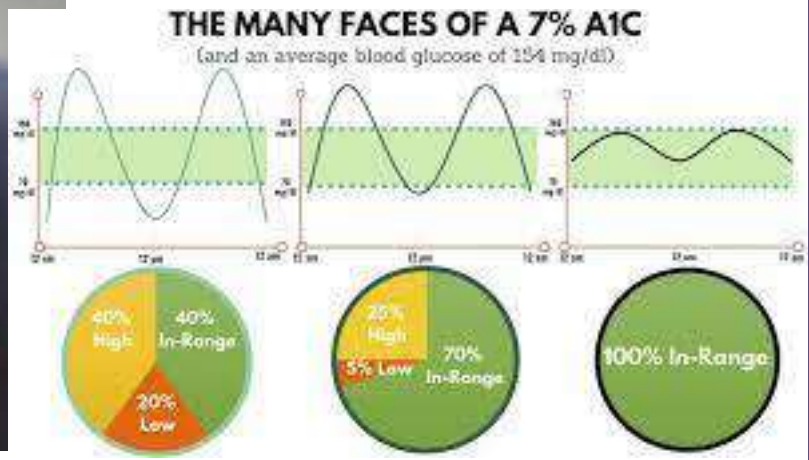
DME

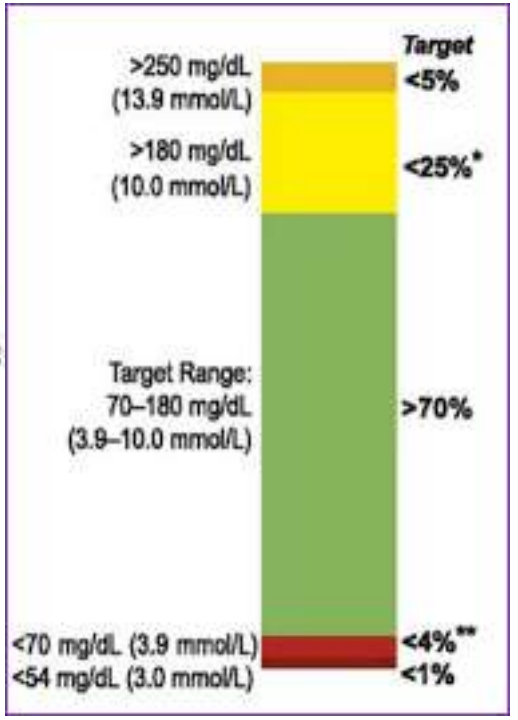
- CSME vs DME
  - CI DME vs Non-CI DME
- Is DME/CI-DME always a referral?

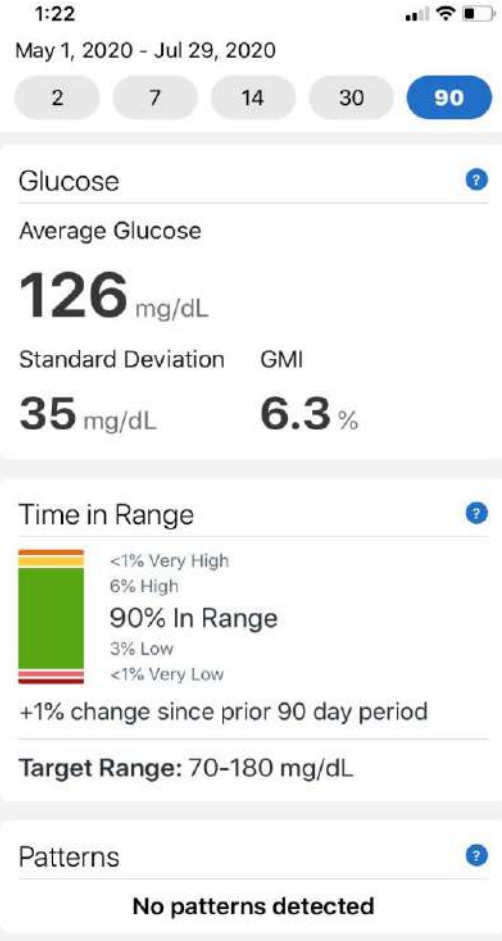




Systemic care







Questions???

Thank you!

Questions or concerns: dr.marybethyackey@gmail.com

Compliments: jgerson@Hotmail.com