

# The ABCs of OCT 1: AMD, DME, and retinal vascular disease

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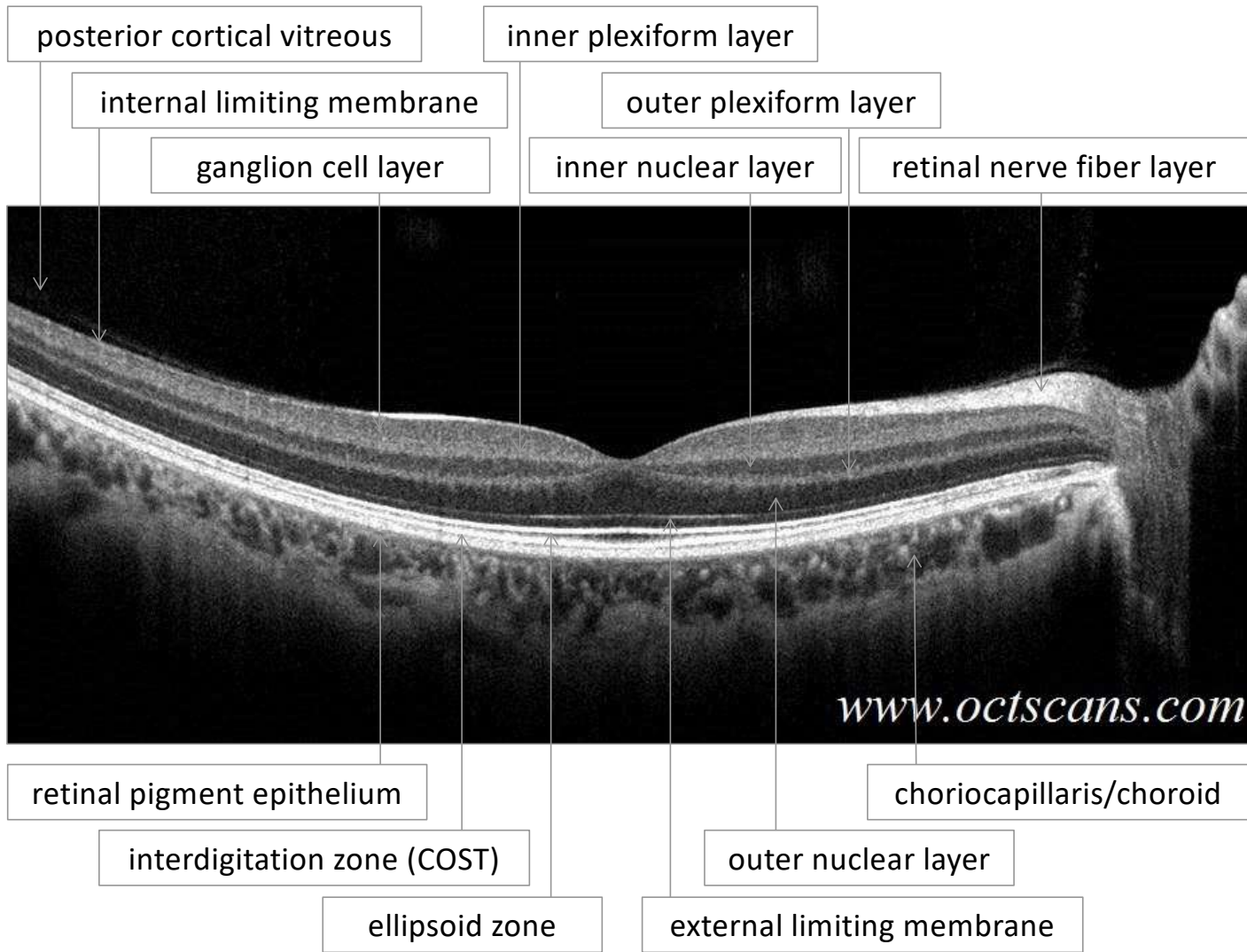


OCT has revolutionized the way we manage retinal disease  
**Pattern recognition and identification of disease biomarkers  
 is essential to image interpretation and prognosis<sup>1</sup>**

Qualitative evaluation of raster scans		
	Normal	Abnormal
Higher reflectivity	<ul style="list-style-type: none"> <li>retinal pigment epithelium</li> <li>ellipsoid zone and ELM</li> <li>outer and inner plexiform layers</li> <li>retinal nerve fiber layer</li> </ul>	<ul style="list-style-type: none"> <li>epiretinal membrane</li> <li>hemorrhage</li> <li>hard exudates and foci</li> <li>drusen</li> <li>neovascular membranes</li> <li>scar tissue</li> </ul>
Lower reflectivity	<ul style="list-style-type: none"> <li>outer and inner nuclear layers</li> <li>myoid zone (posterior to ellipsoid zone)</li> </ul>	<ul style="list-style-type: none"> <li>intraretinal fluid/cysts</li> <li>subretinal/sub-RPE fluid</li> <li><i>shadowing beneath hyper-reflective lesions</i></li> </ul>

1. Spaide RF, Curcio CA. Anatomical correlates to the bands seen in the outer retina by optical coherence tomography. *Retina* 2011;31:1609-19.

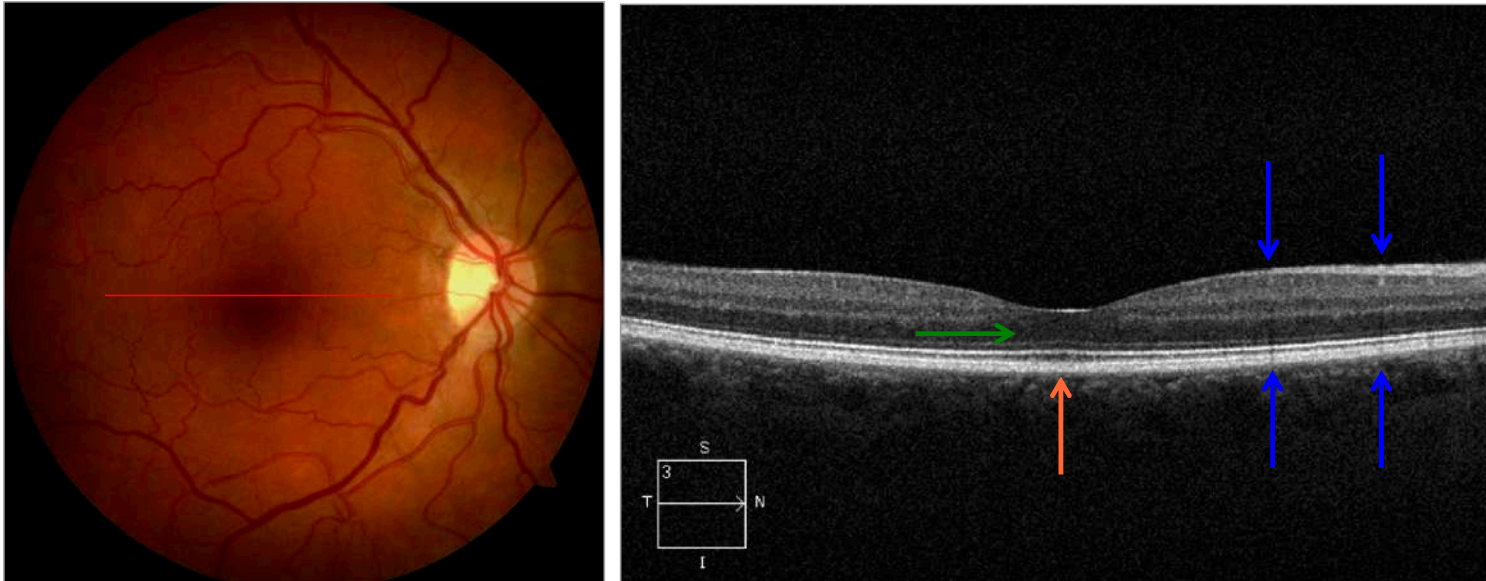






## Unremarkable macular raster scan

55 y/o WW; BCVA 6/6



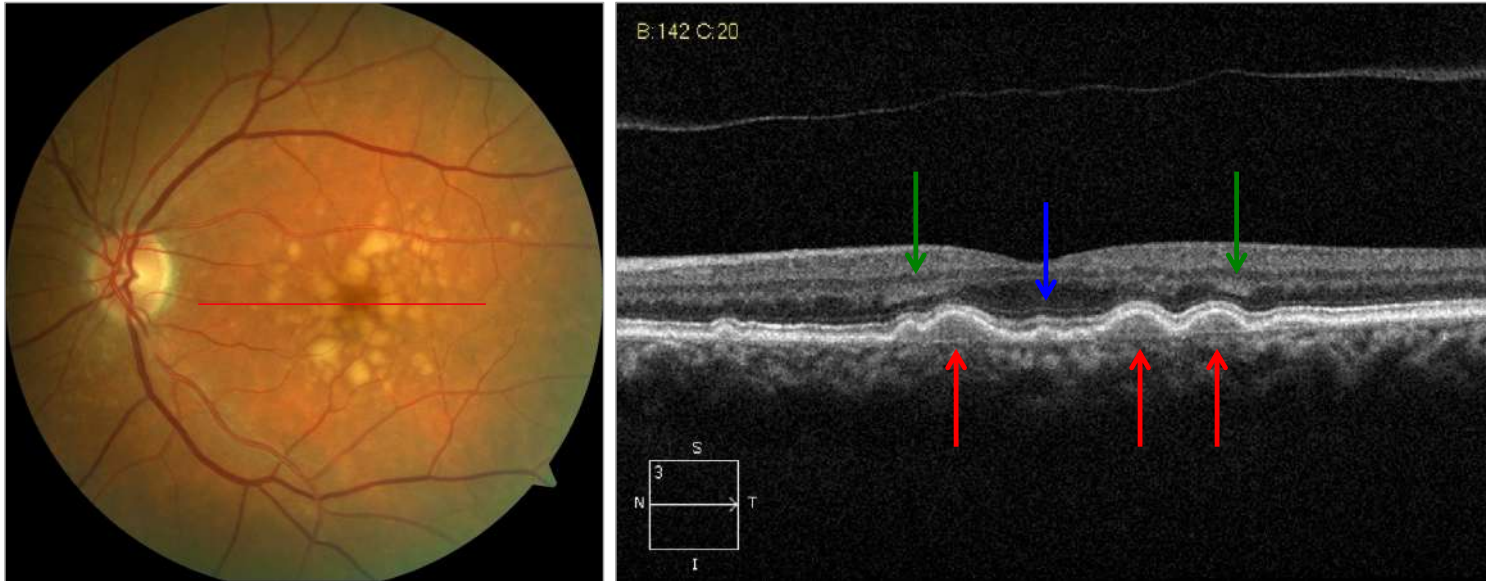
- **intact foveal RPE, faint IZ, EZ, and ELM: integral to good BCVA**
- **all layers anterior to ONL displaced, and EZ elongated at fovea**
- **deeper shadowing due to superficial blood vessels**

*No abnormalities: routine follow-up*



# Dry age-related macular degeneration

74 y/o WM; BCVA 6/7.5



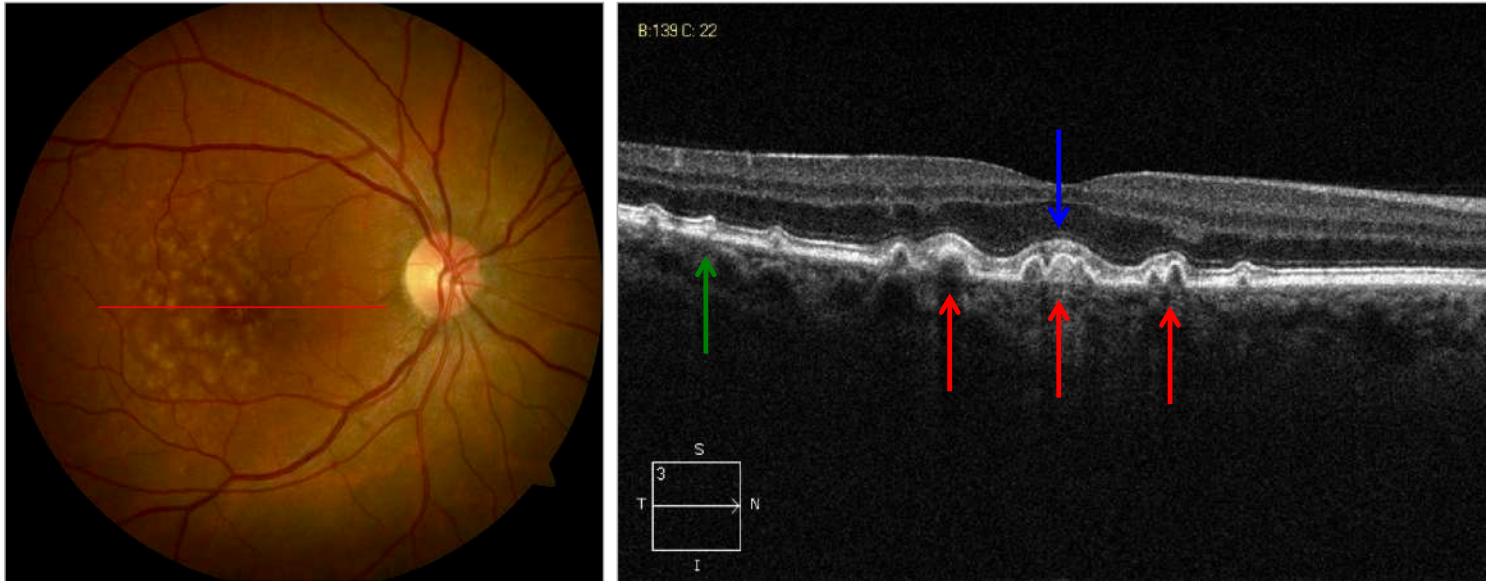
- **soft drusen/drusenoid PED: homogenous material and intact BM**
- **hyper-reflective Henle fiber layer (macular OPL): optical effect**
- **intact EZ and ELM: good BCVA**

*No active CNV, but high risk of progression: monitor every 6/12*



# Dry age-related macular degeneration

59 y/o WW; BCVA 6/12



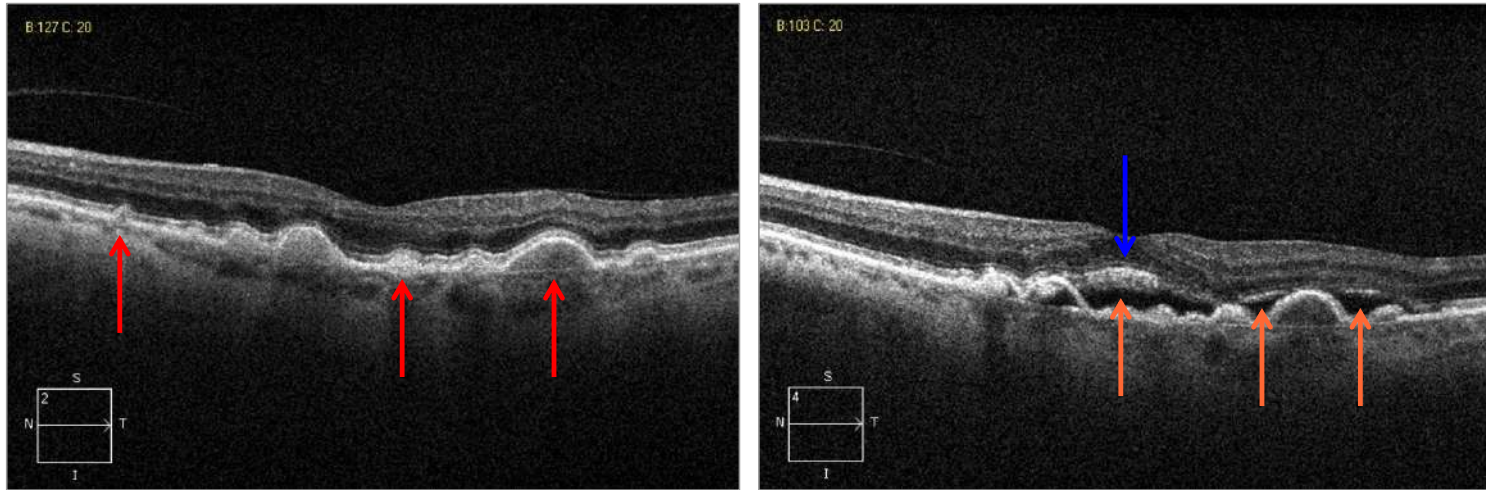
- **multiple smaller hard (cuticular) drusen and drusenoid PED**
- **possible subretinal drusenoid deposits/reticular pseudodrusen**
- **intact ELM but irregular/thickened EZ: reduced BCVA**

*No active CNV, but reduced BCVA: monitor every 6/12*



## Dry AMD converting to wet AMD

74 y/o WM; BCVA was 6/7.5, now 6/12

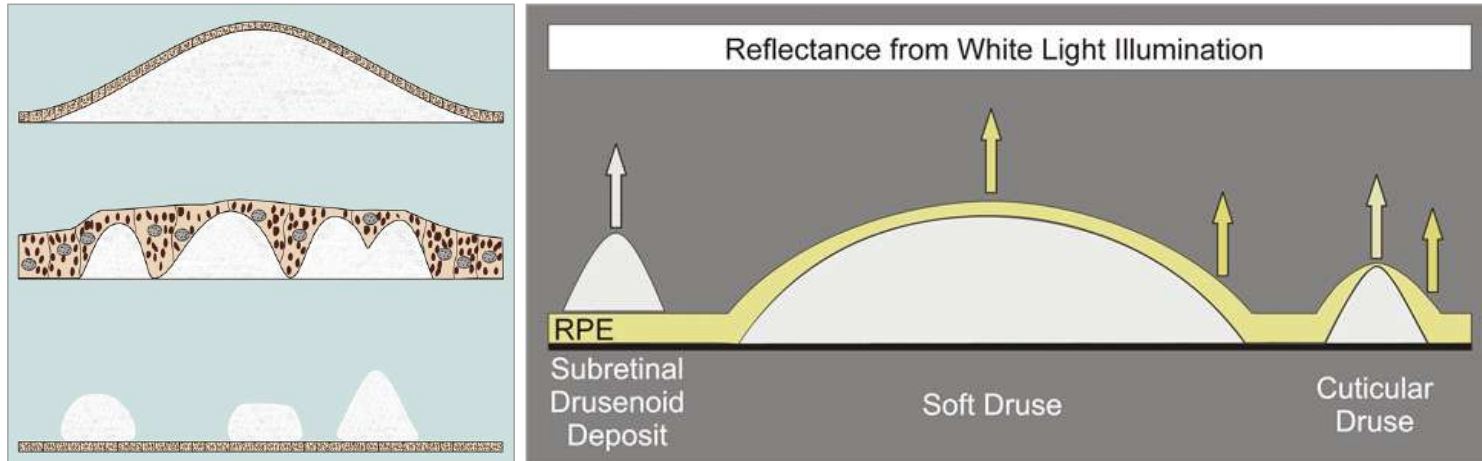


- **baseline (left): drusenoid PED, cuticular drusen, and stage 2 SDD**
- **follow-up (right): new onset subretinal fluid (SRF) suggests active choroidal neovascular membrane (at site of SDD?)**
- **serous RD and thickened foveal EZ: reduced BCVA (6/7.5 to 6/12)**  
*Likely CNV: refer to retina for anti-VEGF injection*





## Soft drusen vs. cuticular drusen vs. SDD



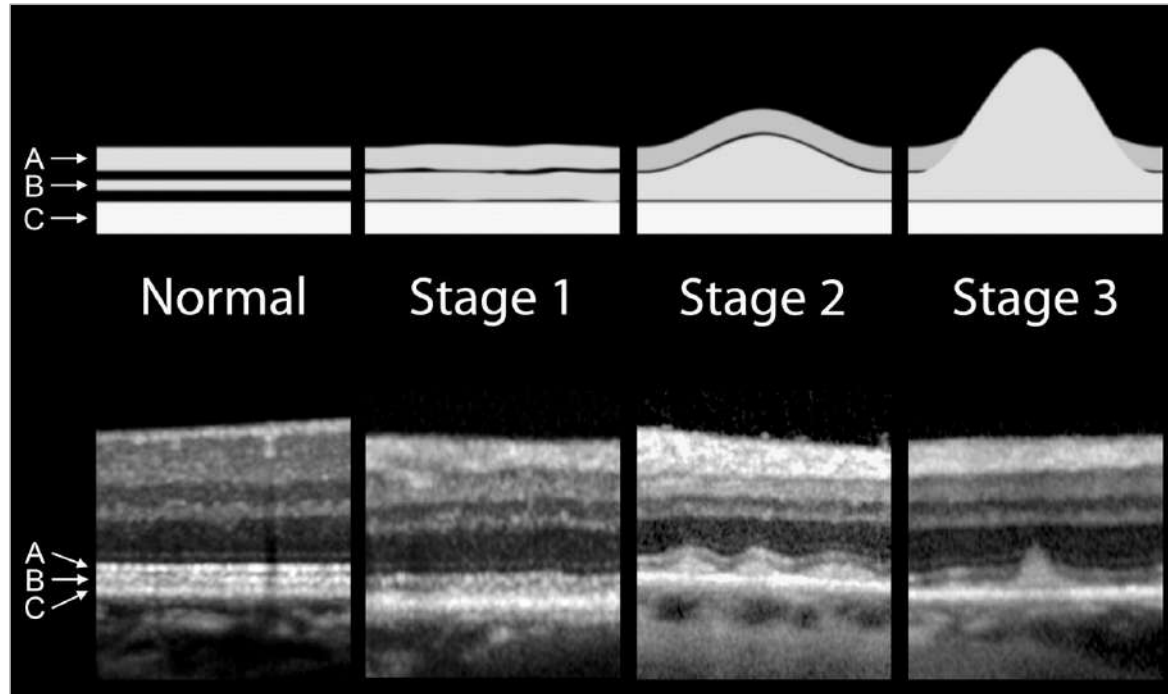
Larger soft drusen and smaller cuticular drusen are **beneath the RPE**, and are more visible with long wavelength red channel light (blue light is attenuated by passing through the RPE)<sup>1</sup>

SDD are **above the RPE**, of variable size, and can be confluent: they may resemble cuticular drusen, but are more visible with short wavelength blue channel light, and are **a biomarker of severe disease**

1. Spaide RF, Curcio CA. Drusen characterization with multimodal imaging. *Retina* 2010;30:1441-54.



## Staging subretinal drusenoid deposition



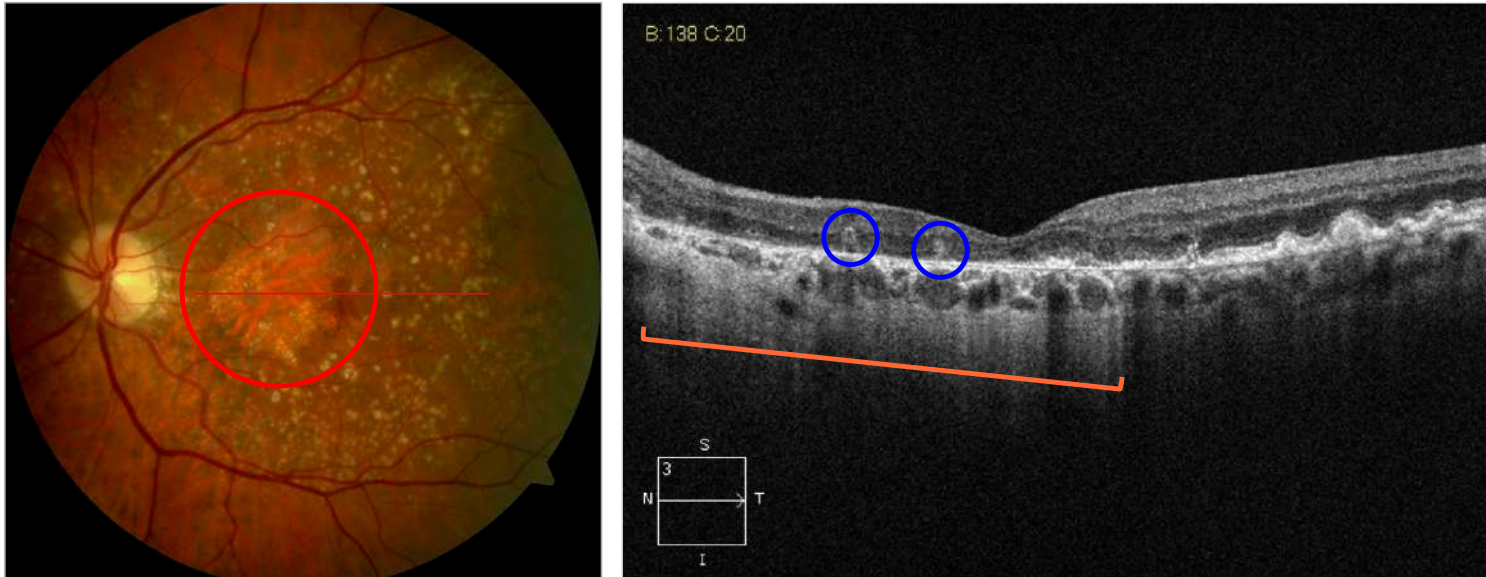
Stage 1: diffuse deposition of granular material between RPE and EZ  
Stage 2: mounding of material with change in contour of EZ  
Stage 3: conical-shaped deposits that break through EZ

1. Zweifel SA, et al. Reticular pseudodrusen are subretinal drusenoid deposits. *Ophthalmology* 2010;117:303-12.



# Dry AMD: geographic atrophy

66 y/o WM; BCVA 6/30

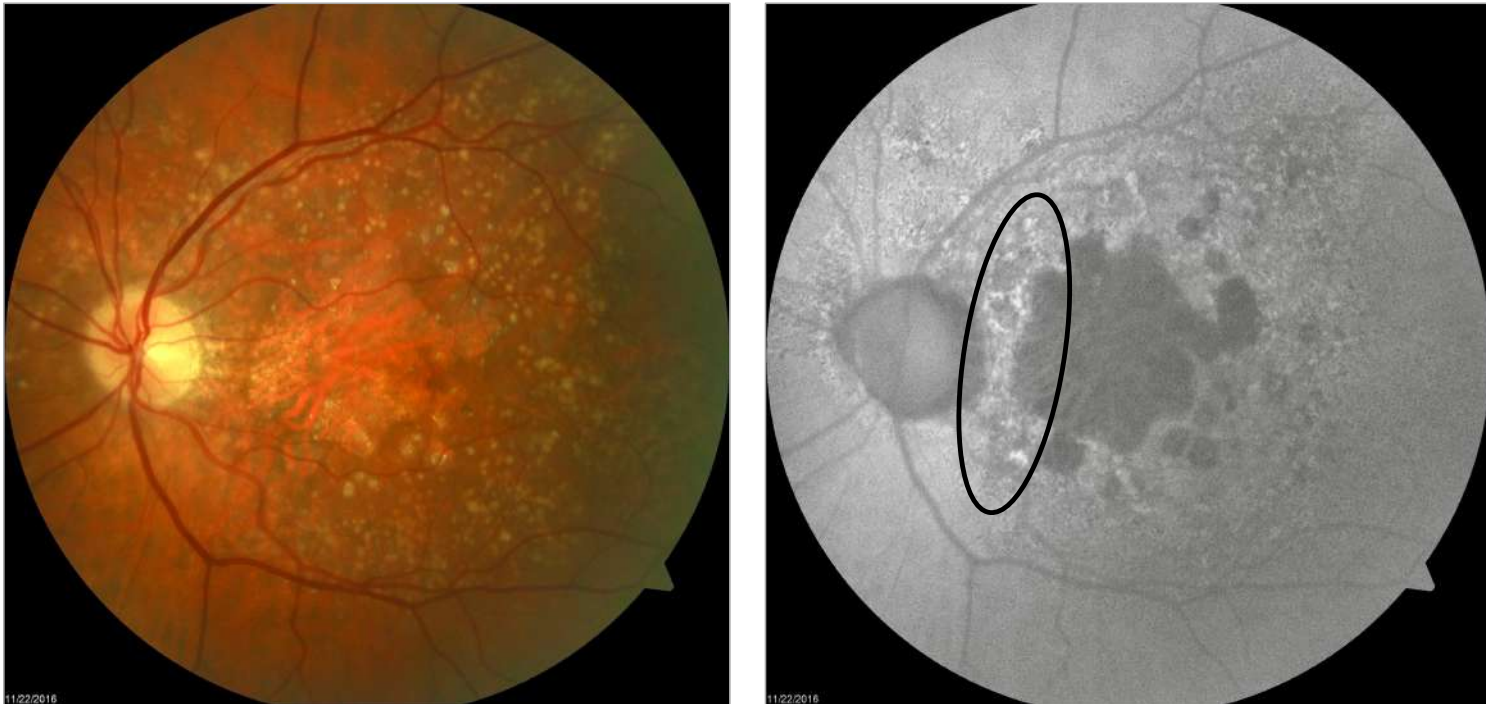


- **GA: atrophy of outer retina/RPE following drusen resorption**
  - **enhanced signal of choroid and sclera due to absence of RPE**
  - **outer retinal tubulation (ORT): degenerating photoreceptors**
- Inactive, but patient is functionally monocular: monitor every 6/12*



## GA: fundus autofluorescence

66 y/o WM; BCVA 6/30



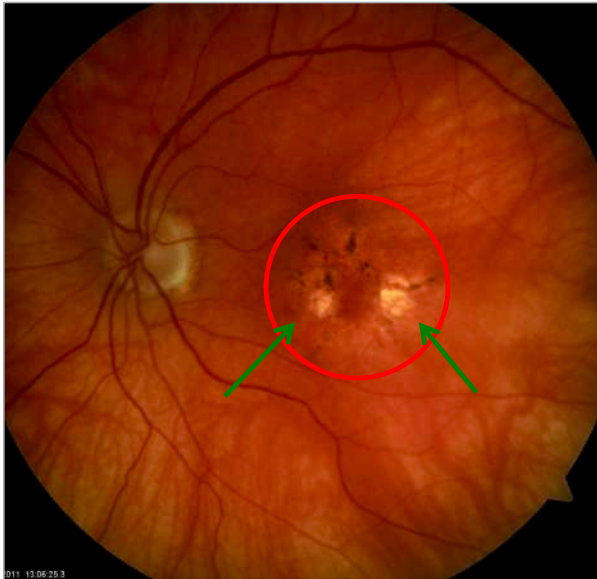
**Hyper-FAF identifies lipofuscin accumulation (RPE distress)  
and may help predict GA progression (in this case, away from fovea)**





# The evolution of geographic atrophy

65 y/o WM; BCVA 6/6



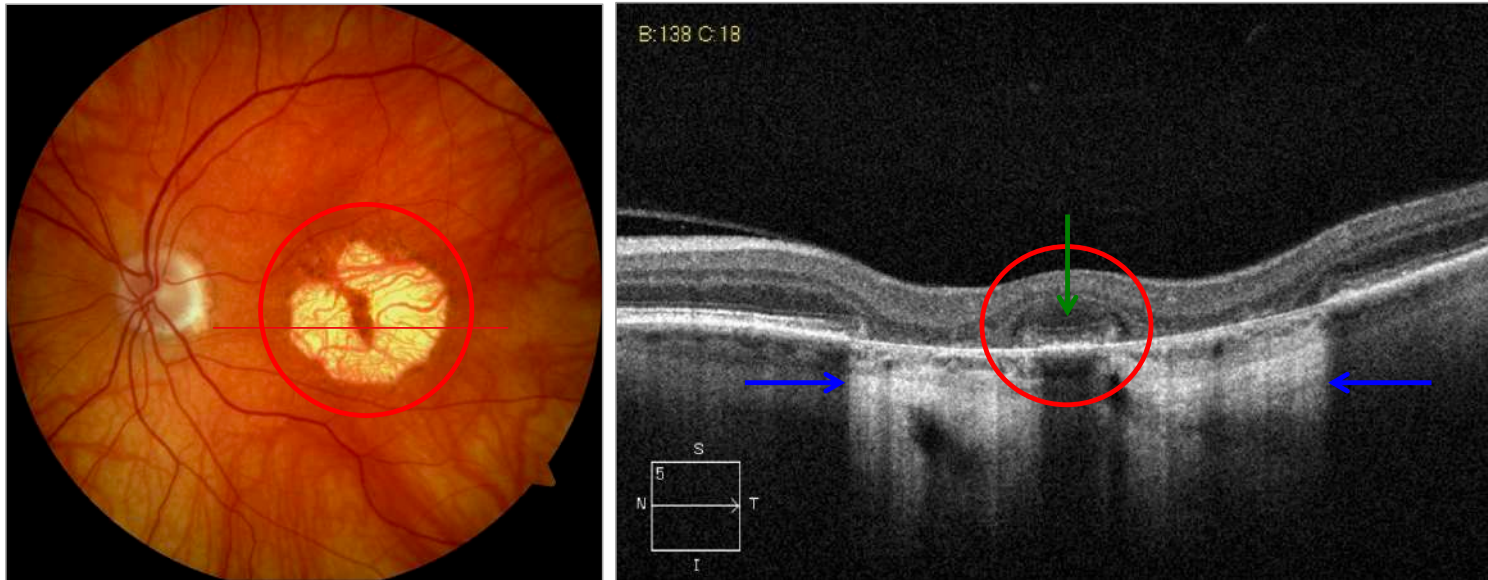
- **initial pigment irregularity surrounding largely intact fovea**
- **two areas of focal GA just nasal and temporal to fovea**
- **OCT not yet available on site: monitor with clinical examination**

*Given proximity of GA to fovea, monitor carefully (every 6/12)*



# The evolution of geographic atrophy

69 y/o WM; BCVA now 6/7.5



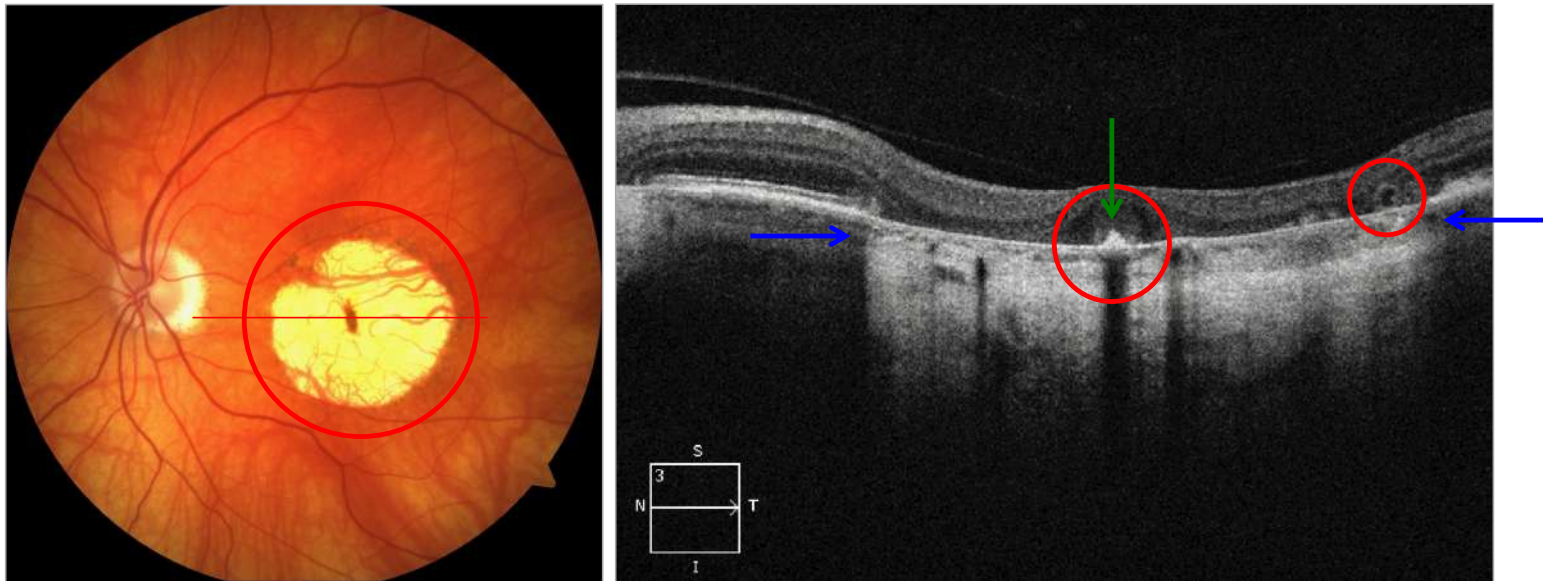
- **now horseshoe-shaped GA with (typical) relative foveal sparing**
- **intact ELM but irregular EZ: slightly decreased BCVA (6/7.5)**
- **extremely enhanced signaling adjacent to intact sub-foveal RPE**

*Given proximity of GA to fovea, monitor carefully (every 6/12)*



# The evolution of geographic atrophy

72 y/o WM; BCVA now 6/9-

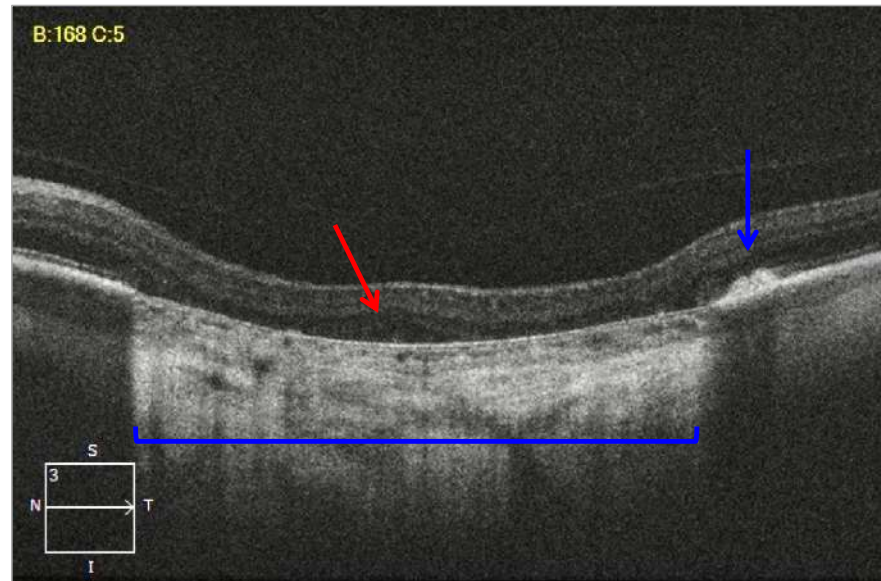


- **progression of GA: much smaller area of foveal sparing and ORT**
  - **ELM may still be intact, but EZ not detectable: BCVA still 6/9**
  - **enhanced signaling has expanded centrifugally and centripetally**
- Given imminent threat to fovea, continue frequent monitoring*



# The evolution of geographic atrophy

76 y/o WM; BCVA now <6/60



- **GA has progressed to obliterate outer retina: subtle deflection of OPL (some preservation?) but loss of EZ (BCVA <6/60)**
  - **enhanced signaling across entire lesion, with temporal pigment**
- Fellow eye still 6/6 despite pigment irregularity: monitor every 6/12*





## Concurrent GA and suspect CNV

82 y/o WM; BCVA 6/30



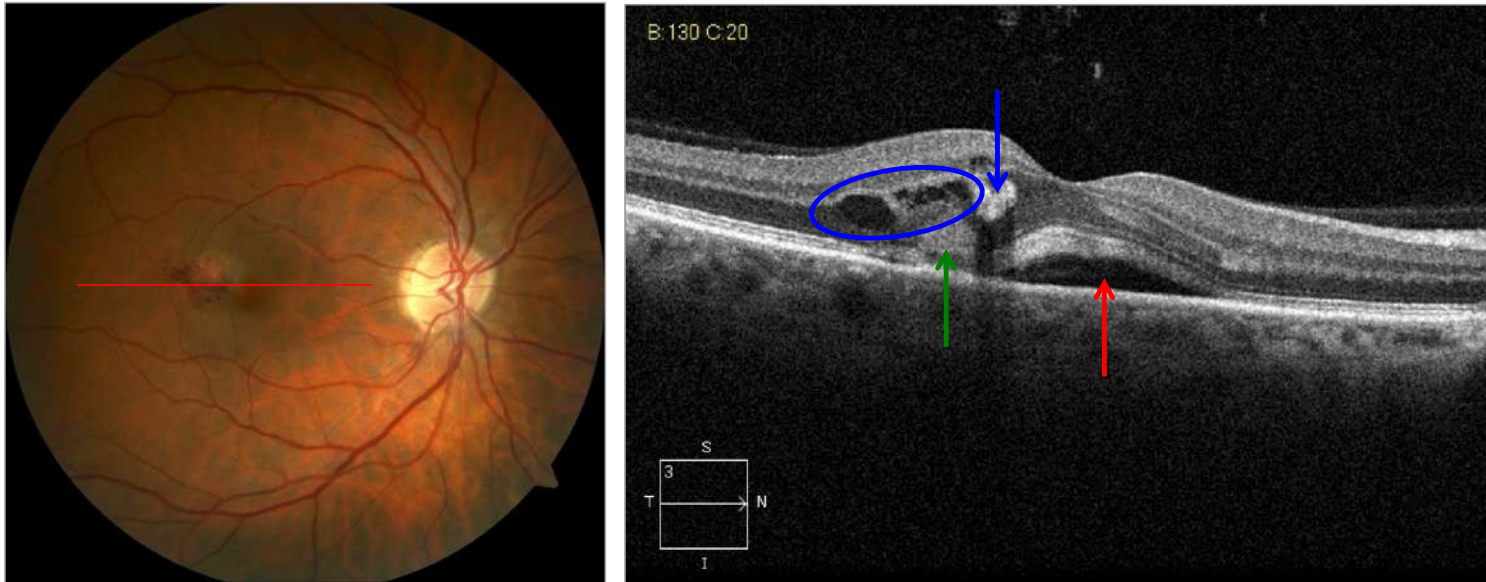
- **central GA: atrophy of outer retina/RPE and enhanced signal**
- **amorphous/consolidating subretinal hyper-reflective material**
- **PED and adjacent SRF suggests possibility of active CNV**

*Potential CNV: refer to retina for anti-VEGF consult*



# Wet age-related macular degeneration

56 y/o WM; BCVA 6/9



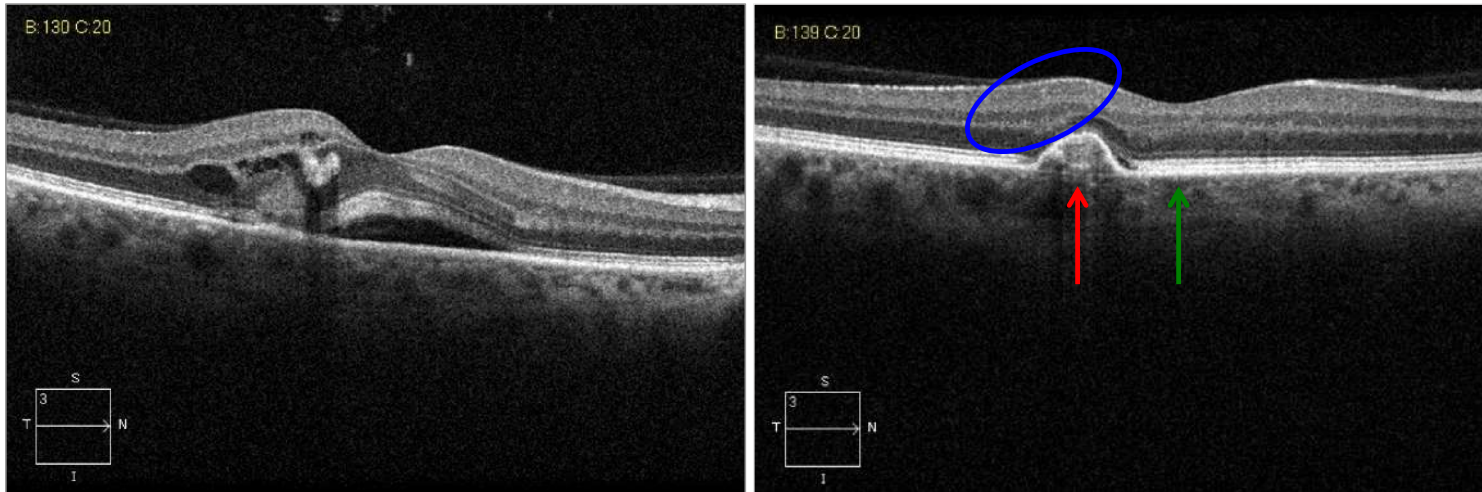
- **SRF and thickened overlying EZ: slightly reduced BCVA (6/9)**
- **amorphous subretinal hyper-reflective material (SHM): activity**
- **intraretinal cystoid lesions and hyper-reflective hemorrhage**

*Active CNV: immediate referral for anti-VEGF injection*



## Wet AMD: post-anti-VEGF injection (x2)

56 y/o WM; BCVA improved from 6/9 to 6/6



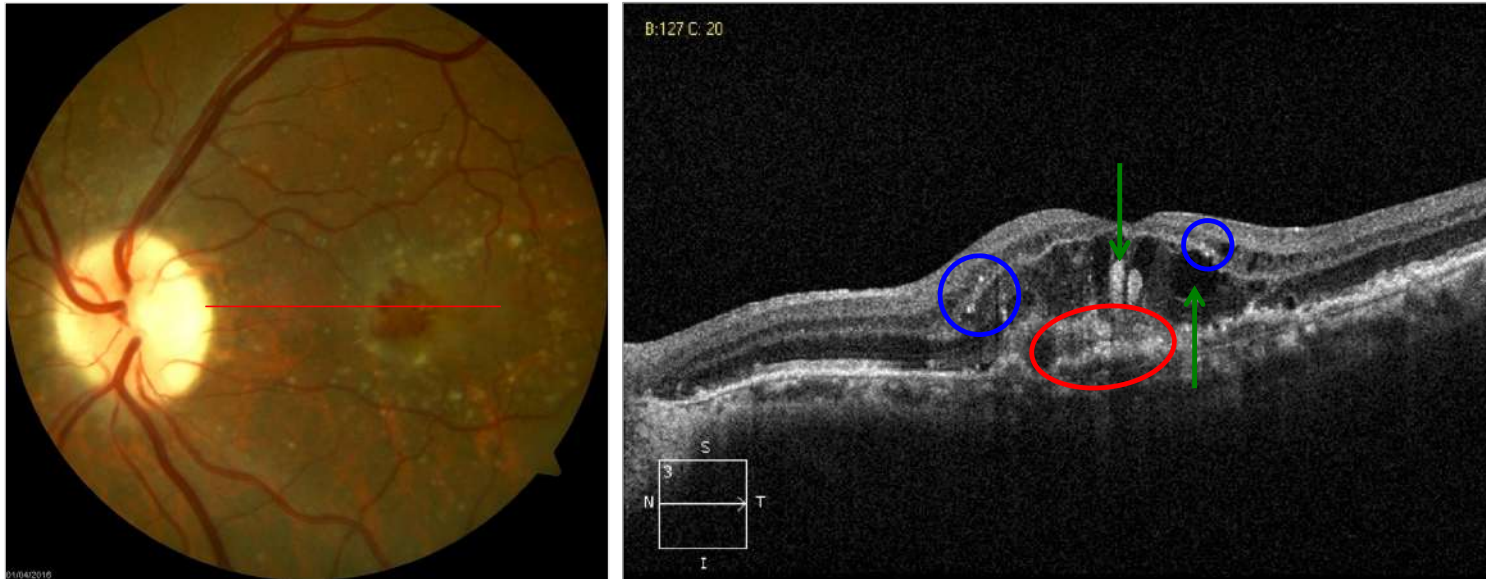
- pre-treatment on left
- **post-treatment on right: SHM consolidating to form solid PED**
- **trace residual SRF adjacent to PED but intact EZ: BCVA back to 6/6**
- **near-complete resolution of intraretinal cystoid lesions/IRH**

*Inactive post-anti-VEGF injection: monitor 6/12 for recurrence*



# Wet age-related macular degeneration

63 y/o WM; BCVA 6/18



- **amorphous SHM with irregular RPE and EZ: reduced BCVA (6/18)**
- **intraretinal cystoid lesions and hyper-reflective hemorrhage**
- **hyper-reflective foci (HF): activated microglia vs. pre-HE lipid?**

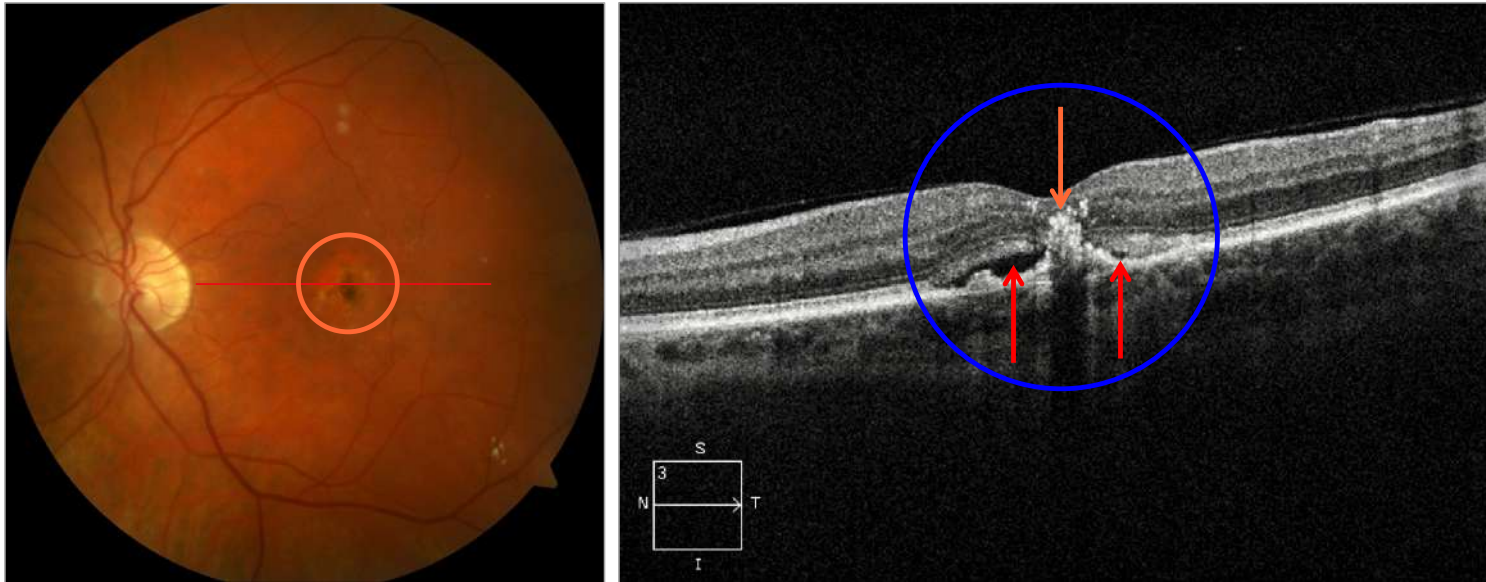
*Active CNV: immediate referral for anti-VEGF injection*





# Wet age-related macular degeneration

79 y/o WM; BCVA 6/30



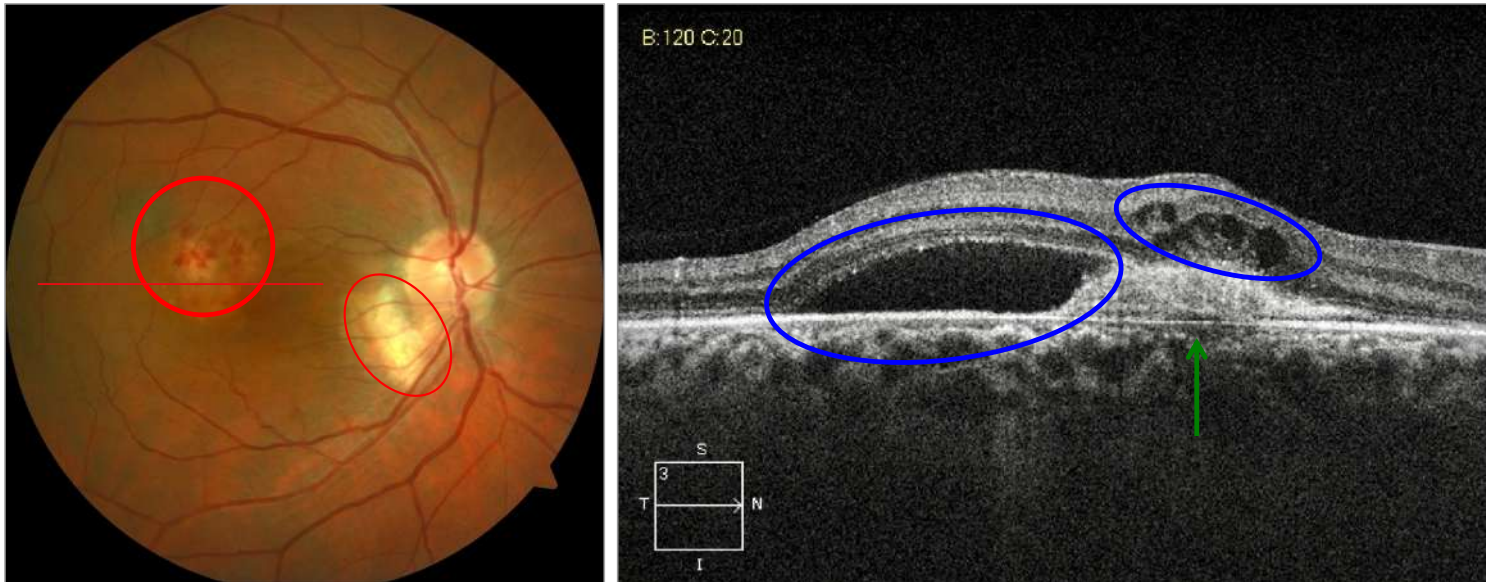
- **irregular central PED with adjacent SRF: likely active CNV**
- **focal pigment figure: anterior migration of RPE (higher risk)**
- **significant disruption of foveal EZ: very reduced BCVA (6/30)**

*Active CNV: immediate referral for anti-VEGF injection*



# Choroidal neovascularization

60 y/o Indian man; BCVA 6/15 (in his better eye: amblyopic O.S.)



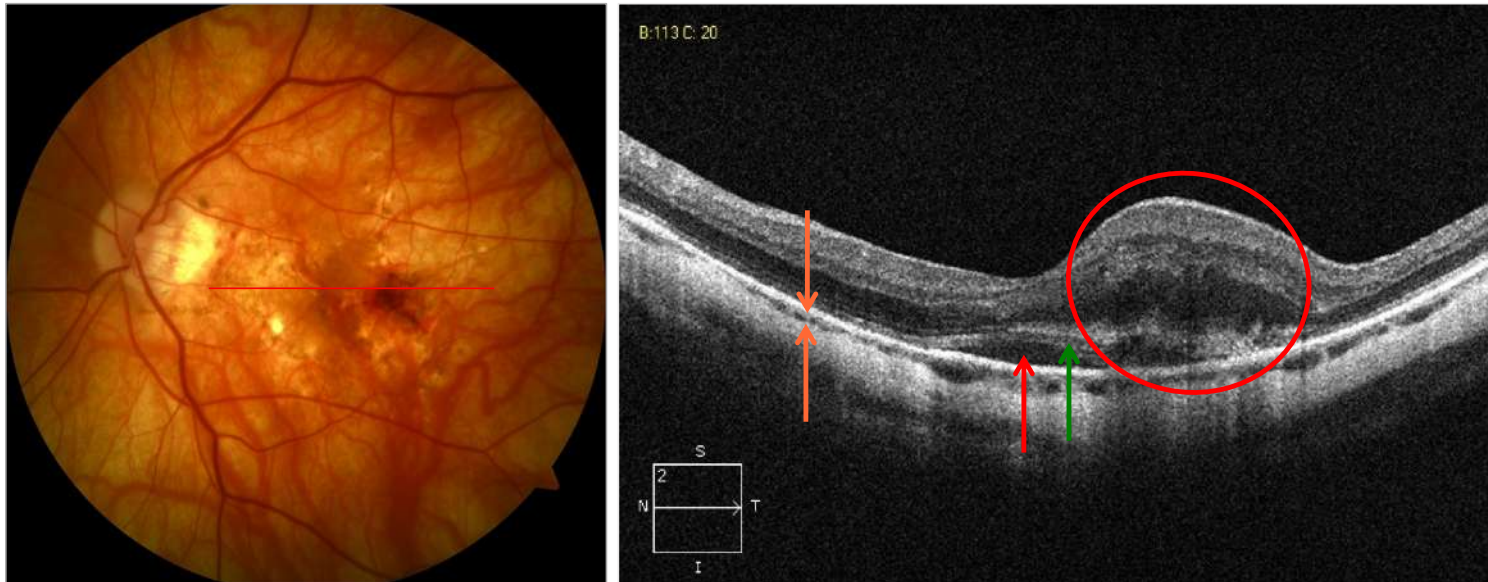
- **new hemorrhage at site of previous lesion (note PP CNV scarring)**
- **consolidated SHM and layered fibrotic PED: suggest chronicity**
- **chronic SRF (note photoreceptor granularity) and IRF: active CNV**

*Reactivation of CNV: immediate referral for anti-VEGF injection*



# Myopic maculopathy

65 y/o WW; BCVA 6/15



- **amorphous SHM/diffuse outer thickening and SRF suggest activity**
- **irregular EZ and overlying edema: reduced BCVA (6/15)**
- **concave sclera and extremely thin choroid typical of high myopia**

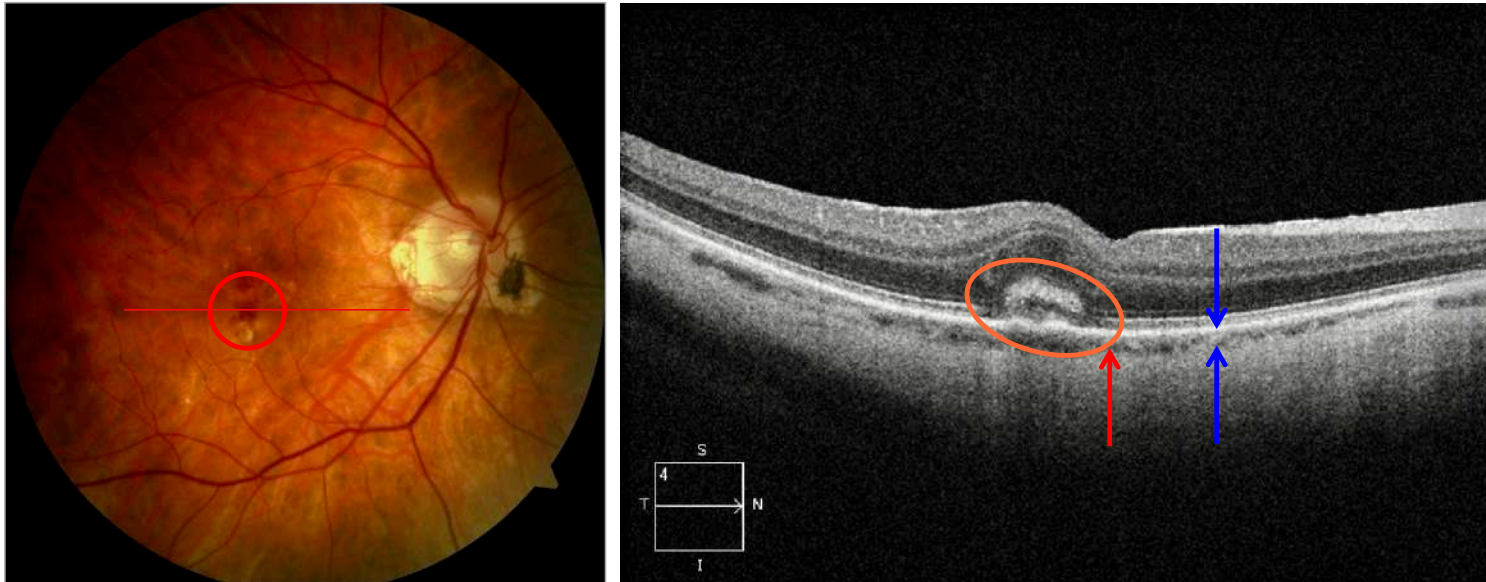
*Myopic CNV: refer for anti-VEGF injection*





# Myopic maculopathy

57 y/o WM; BCVA 6/9



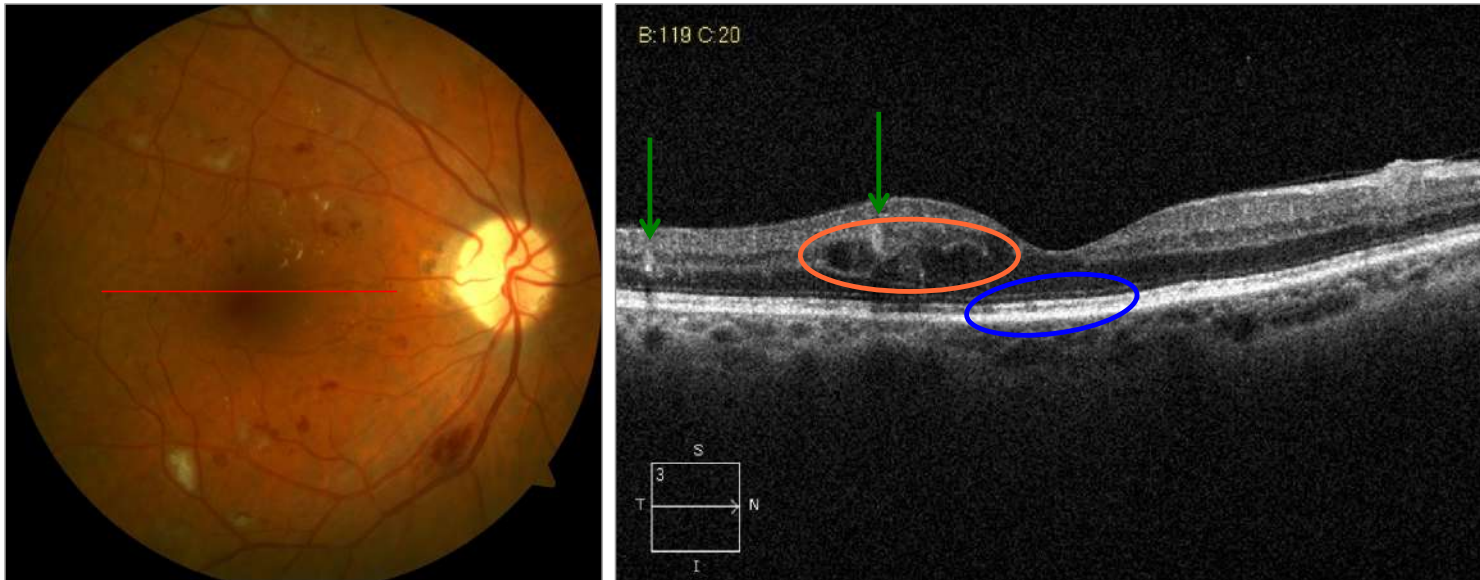
- **small hemorrhage inferior to fovea, but intact EZ: BCVA 6/9**
- **myopic CNV: SHM alone may indicate activity (SRF not necessary)**
- **less scleral concavity, but still extremely thin choroid**

*Suspect myopic CNV: refer for anti-VEGF injection*



# Diabetic macular edema

75 y/o WM; BCVA 6/6



- **speckled intraretinal fluid (IRF) typical of DME**
- **hyper-reflective intraretinal hemorrhages and microaneurysms**
- **center-involved DME (CI-DME), but intact EZ/ELM: good BCVA**

*CI-DME/good BCVA: monitor as per DRCR.net Protocol V*



## DME: 18 months later

76 y/o WM; BCVA 6/6



- **less NPDR, but larger pockets of speckled IRF temporal to fovea**
- **diffuse outer retinal thickening and HF (in outer retina: high risk)**
- **foveal EZ/ELM intact despite edema/hemorrhage: good BCVA**

*Retinal opinion sought despite BCVA: continue to monitor every 6/12*



## DME: another 6 months later

77 y/o WM; BCVA now 6/30



- **increased IRF with bridging (Muller/bipolar) tissue: positive sign**
- **increasing diffuse outer retinal thickening and outer retinal HF**
- **reduced BCVA due to overlying edema and disruption of EZ/ELM**

*Referral for anti-VEGF treatment as per RISE and RIDE Trials*





# The evolution of diabetic retinopathy

35 y/o WW with T1DM; BCVA 6/6 and 6/6



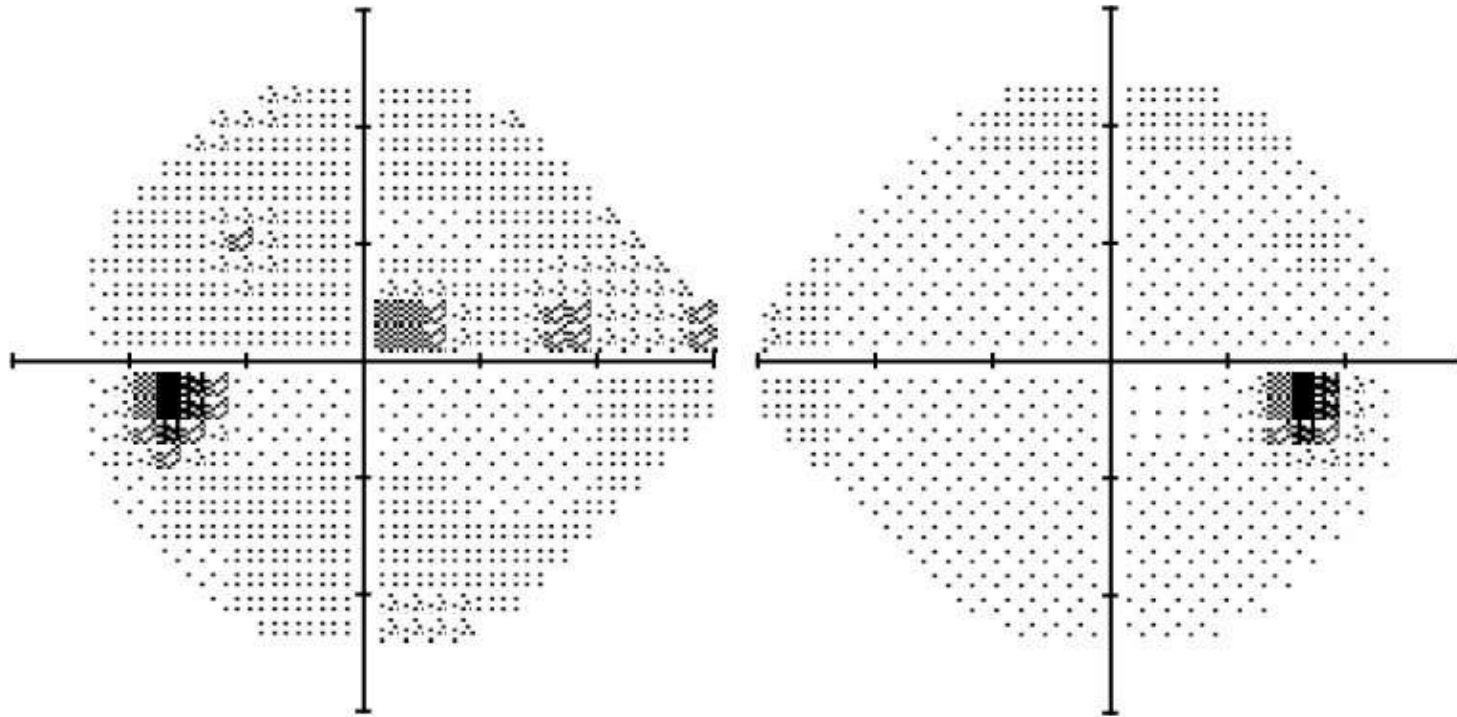
No detectable DR, but **broad RNFL defects** and **ONH pallor O.S.**





# The evolution of diabetic retinopathy

*35 y/o WW with T1DM; BCVA 6/6 and 6/6*

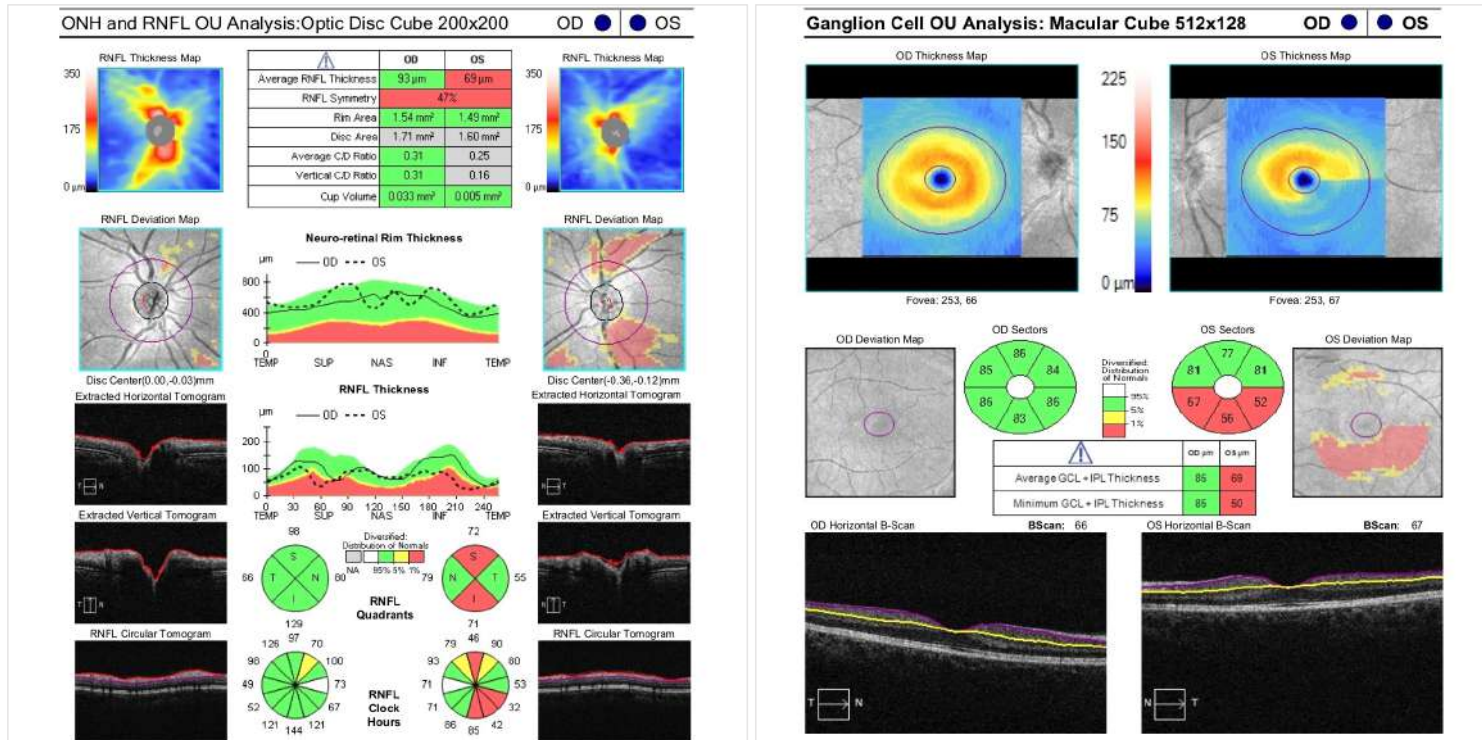


Unremarkable 24-2 AVF O.D., but **superior paracentral defect O.S.**



# The evolution of diabetic retinopathy

35 y/o WW with T1DM; BCVA 6/6 and 6/6



Unremarkable RNFL/GCIPL O.D., but **inferior > superior thinning O.S.**



## The evolution of diabetic retinopathy

37 y/o WW with T1DM; BCVA 6/6 and 6/6

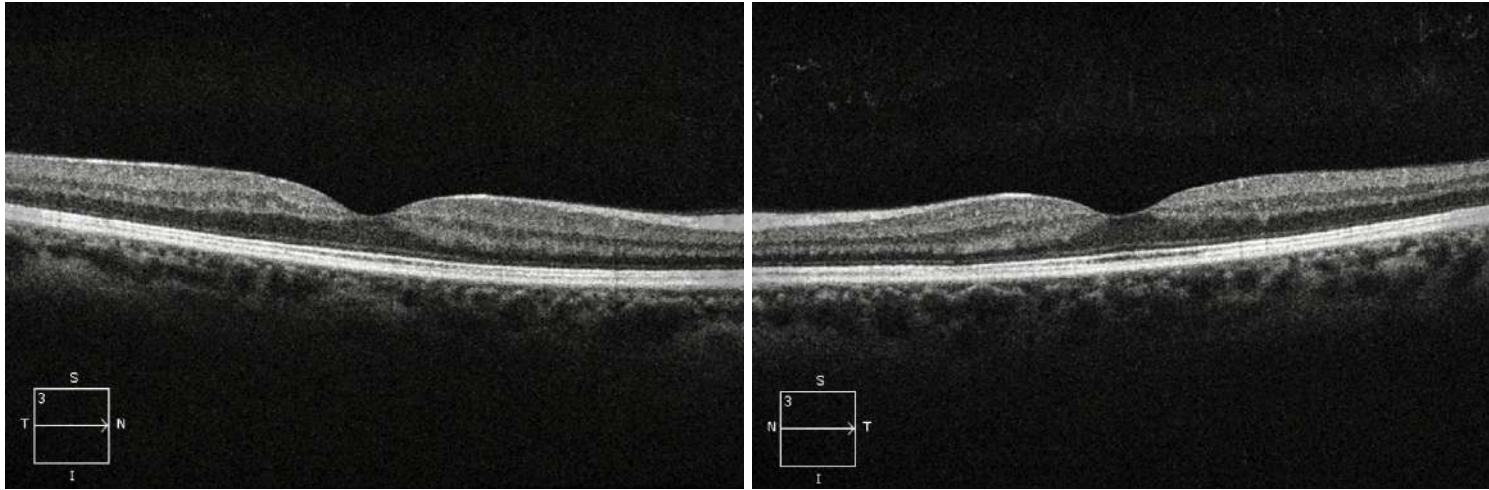


No detectable DR O.D., but **isolated superior-temporal HE O.S.**



# The evolution of diabetic retinopathy

*37 y/o WW with T1DM; BCVA 6/6 and 6/6*



Largely unremarkable macular raster bilaterally, despite HE O.S.





# The evolution of diabetic retinopathy

39 y/o WW with T1DM; BCVA 6/6 and 6/6



Early NPDR O.D.>O.S., with **scattered IRH/Ma temporal to fovea**



# The evolution of diabetic retinopathy

*41 y/o WW with T1DM; BCVA 6/6 and 6/6*

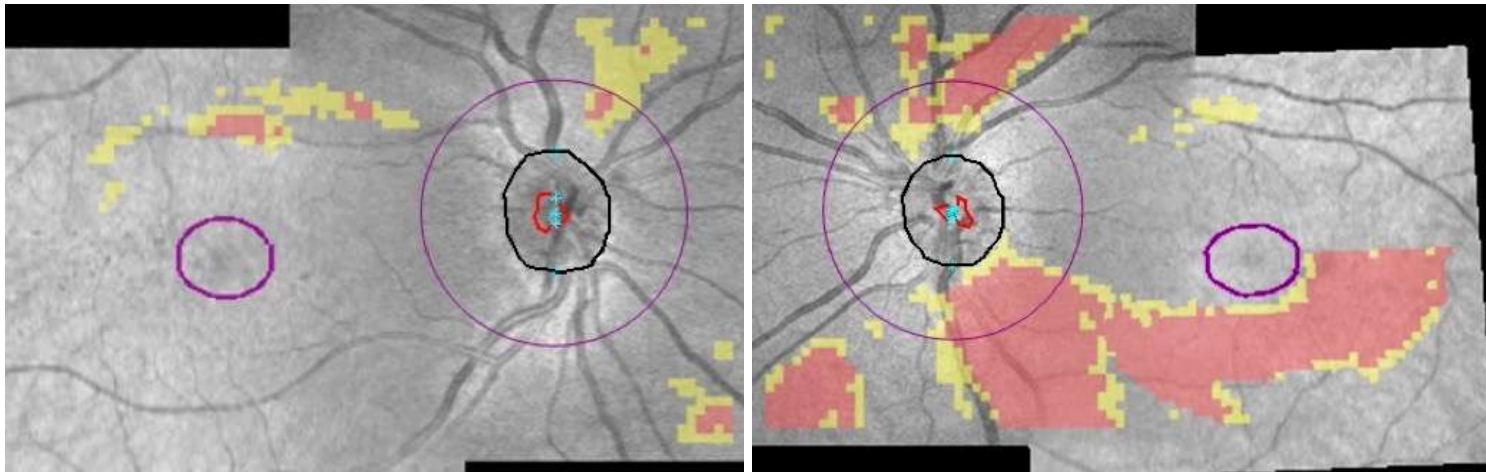


Advancing NPDR with **ischemic superior-temporal RNFL defect O.D.**



# The evolution of diabetic retinopathy

32 y/o WW with T1DM; BCVA 6/6 and 6/6



PanoMap integrating RNFL and GCIPL deviation maps showing  
**narrow band of superior RNFL and GCIPL thinning O.D.** and  
**broad bands of inferior > superior RNFL and GCIPL thinning O.S.**



# The evolution of diabetic retinopathy

*42 y/o WW with T1DM; BCVA now 6/9 and 6/6*



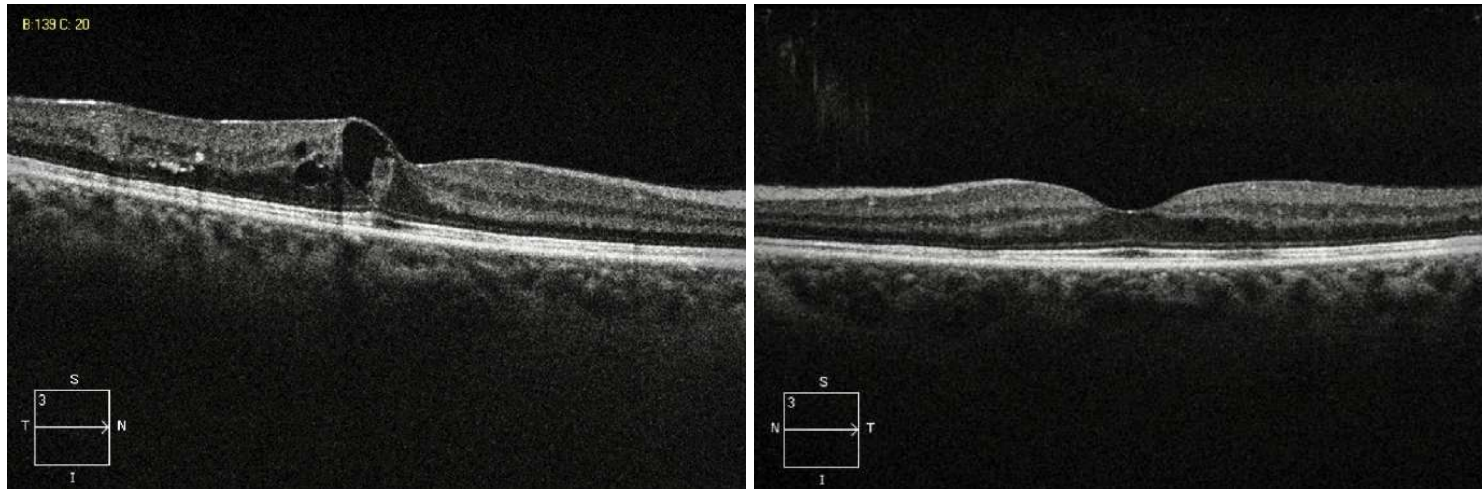
Now slightly reduced BCVA and **temporal parafoveal thickening O.D.**





# The evolution of diabetic retinopathy

*42 y/o WW with T1DM; BCVA now 6/9 and 6/6*

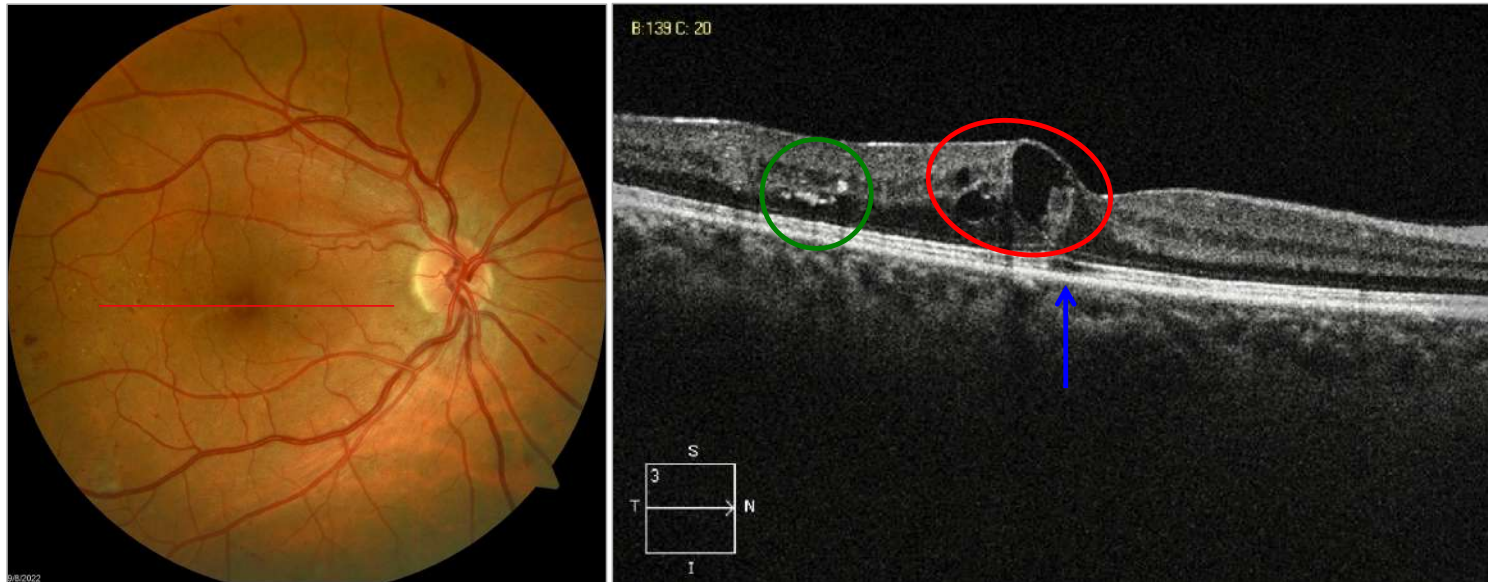


**Center-involved DME mainly temporal to fovea O.D.,**  
and largely unremarkable macular architecture O.S.



# The evolution of diabetic retinopathy

42 y/o WW with T1DM; BCVA now 6/9 and 6/6



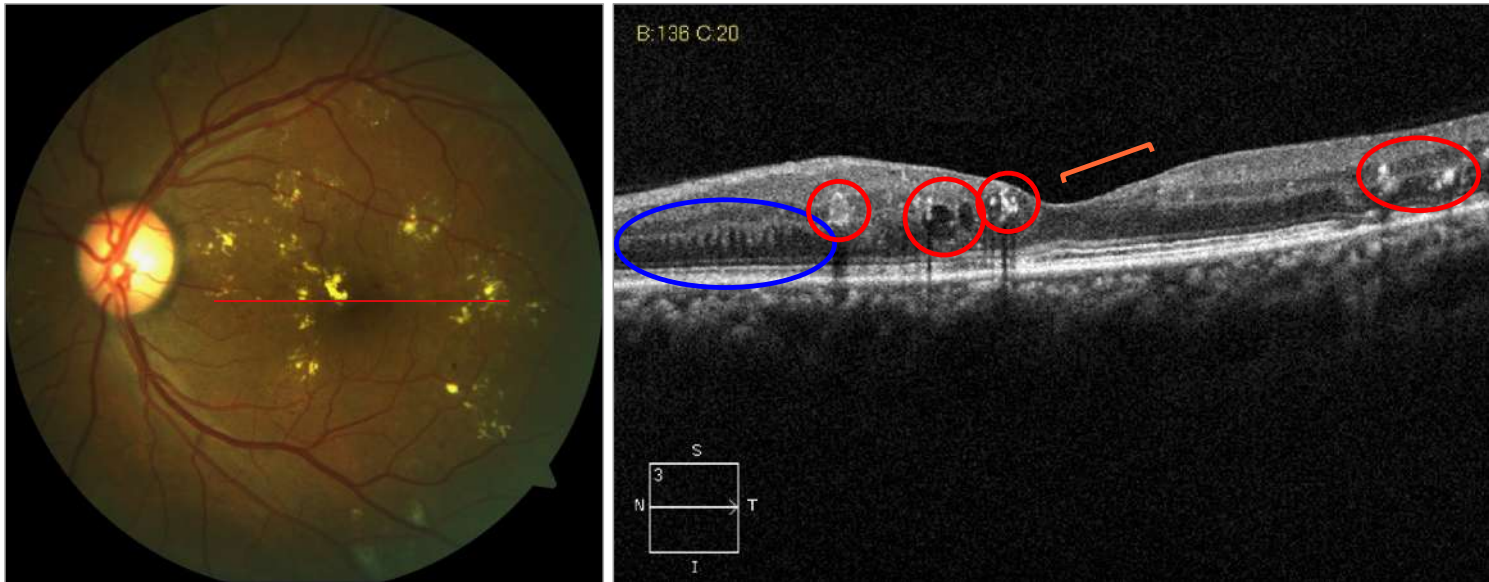
- **speckled IRF with larger outer cyst bounded anteriorly by ILM**
- **hyper-reflective HE in and adjacent to OPL**
- **CI-DME with ↓ BCVA (intact EZ, but edema and interrupted IZ)**

*CI-DME/reduced BCVA: referral as per RISE and RIDE Trials*



# Diabetic macular edema

56 y/o Indian man; BCVA 6/6-

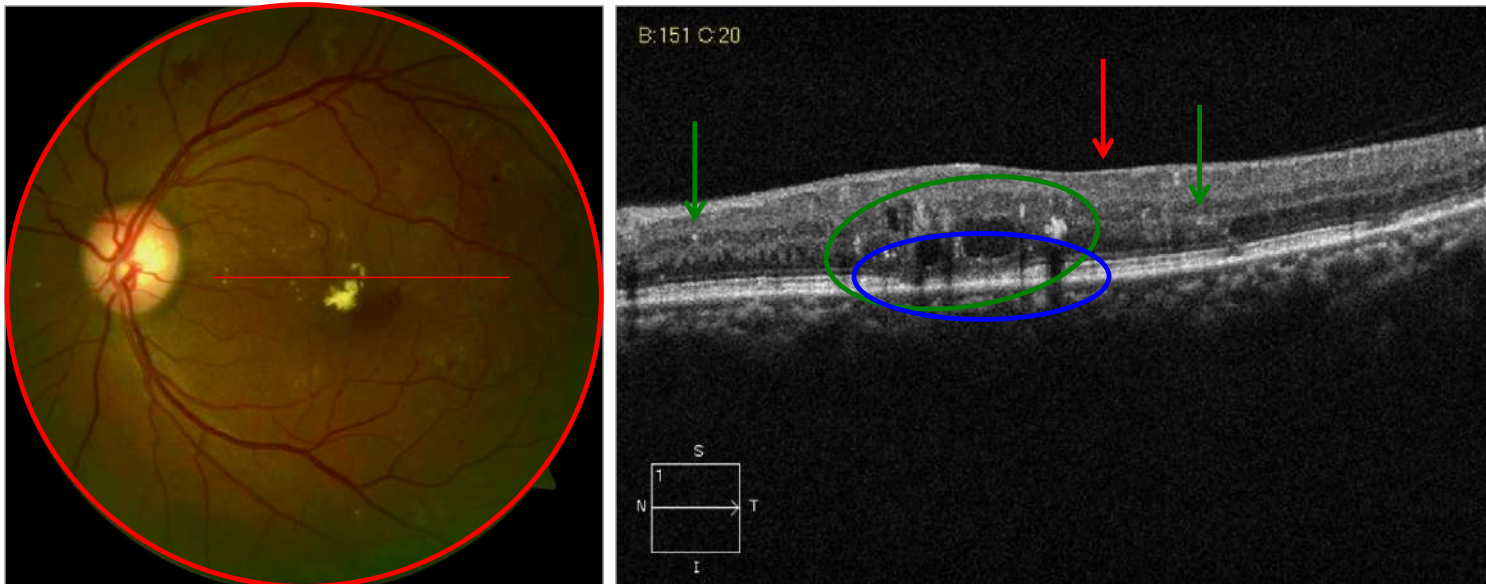


- **speckled IRF, and hyper-reflective HE, HF, and IRH/Ma**
  - **disorganization of retinal inner layers (DRIL): GCIPL/INL/OPL  $\Delta$**
  - **diffuse outer retinal thickening ("saw tooth" OPL/ONL junction)**
- CI-DME/slightly decreased BCVA: referral (Protocol V vs. RISE/RIDE?)*



## DME: 30 months post-anti-VEGF treatment

58 y/o Indian man; BCVA 6/7.5



- **note improved clinical retinal appearance and less DRIL ...**
- **... but diffuse thickening and persistent speckled IRF, HE, and HF**
- **EZ disturbed/shadowed nasally but intact centrally: good BCVA**

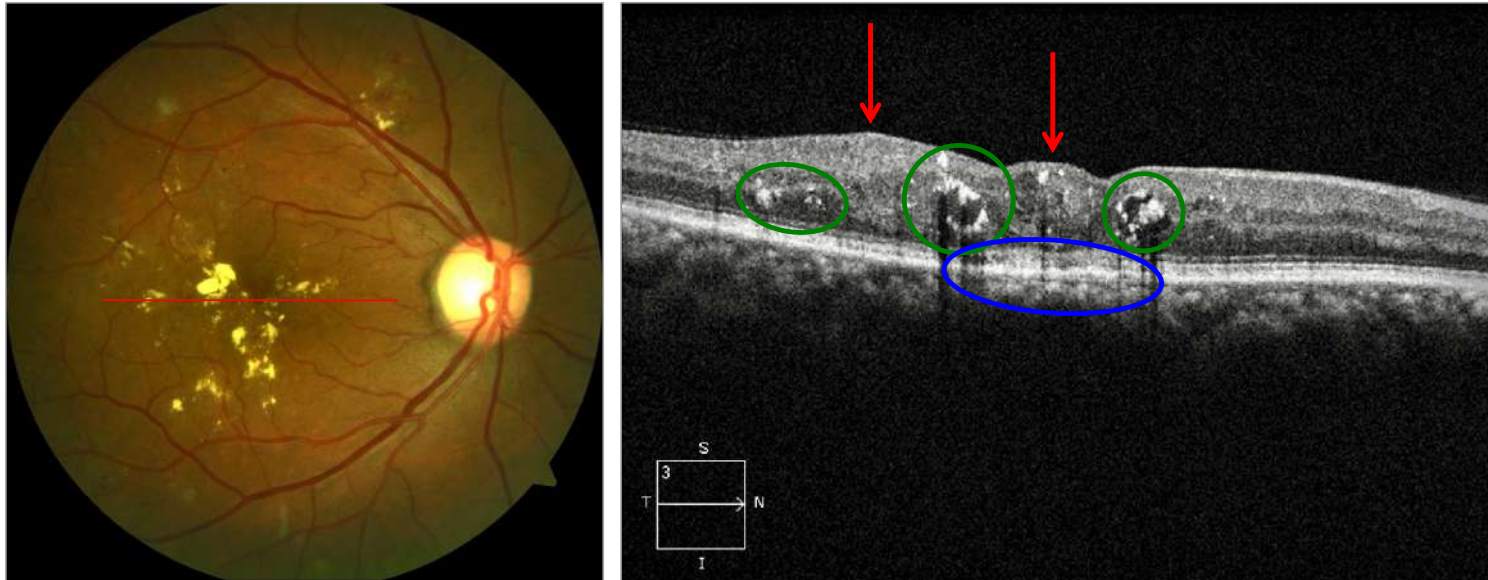
*Patient has deferred further treatment: monitor every 6/12*





# Diabetic macular edema

56 y/o Indian man; BCVA 6/9



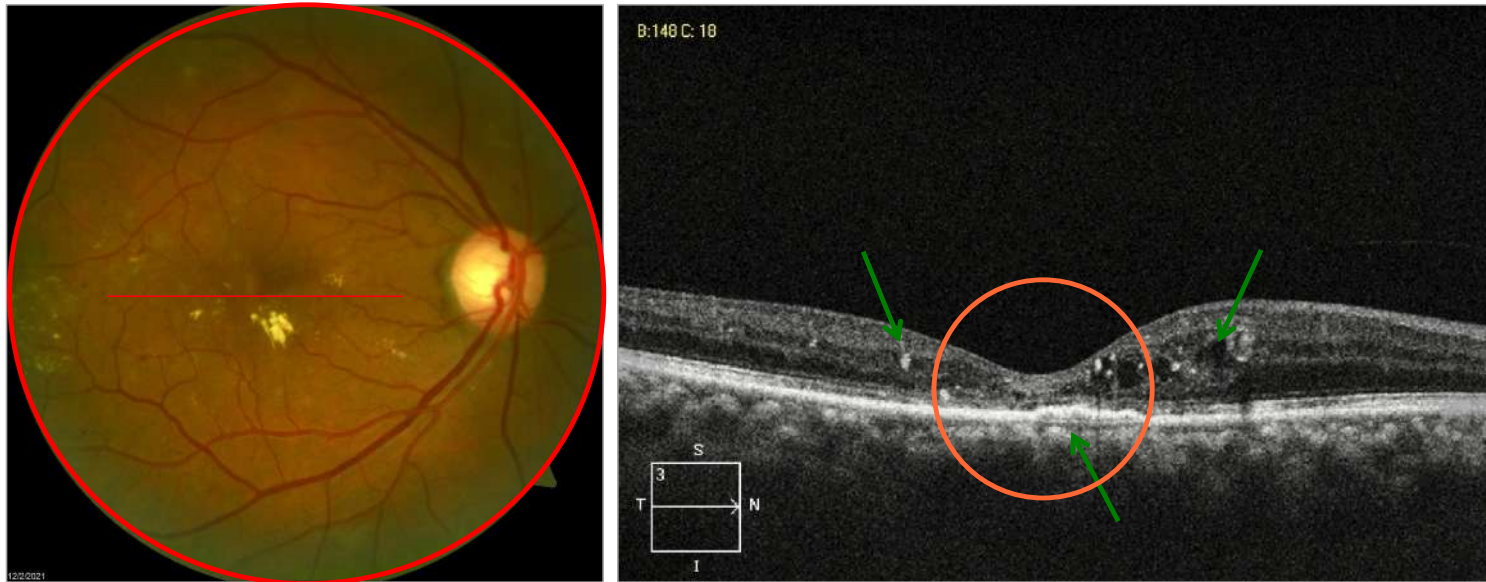
- **central speckled IRF, and central-temporal foveal DRIL**
- **“pearl necklace” sign and hyper-reflective HE (chronic OPL DME)**
- **disruption of foveal EZ/ELM: reduced BCVA (6/9)**

*CI-DME/reduced BCVA: referral as per RISE and RIDE Trials*



# Diabetic ischemic maculopathy

61 y/o Indian man; BCVA now 6/15



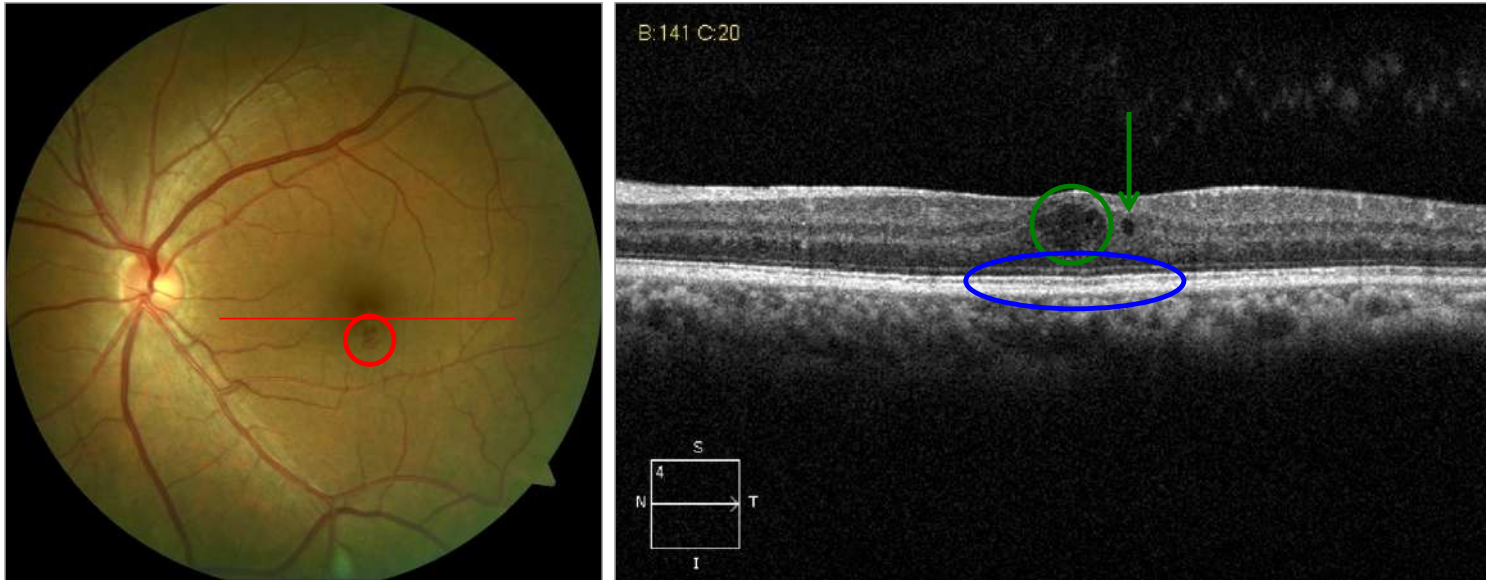
- **clinical exam: far fewer HE and IRH suggests “improved” NPDR**
- **fewer HE, scattered HF, and less IRF, but outer retinal irregularity**
- **extreme outer retinal thinning and DRIL due to chronic ischemia**

*Ultimate VH: vitrectomy and CE O.D., and bilateral PRP*



# Diabetic macular edema

56 y/o Guyanese man; BCVA 6/6



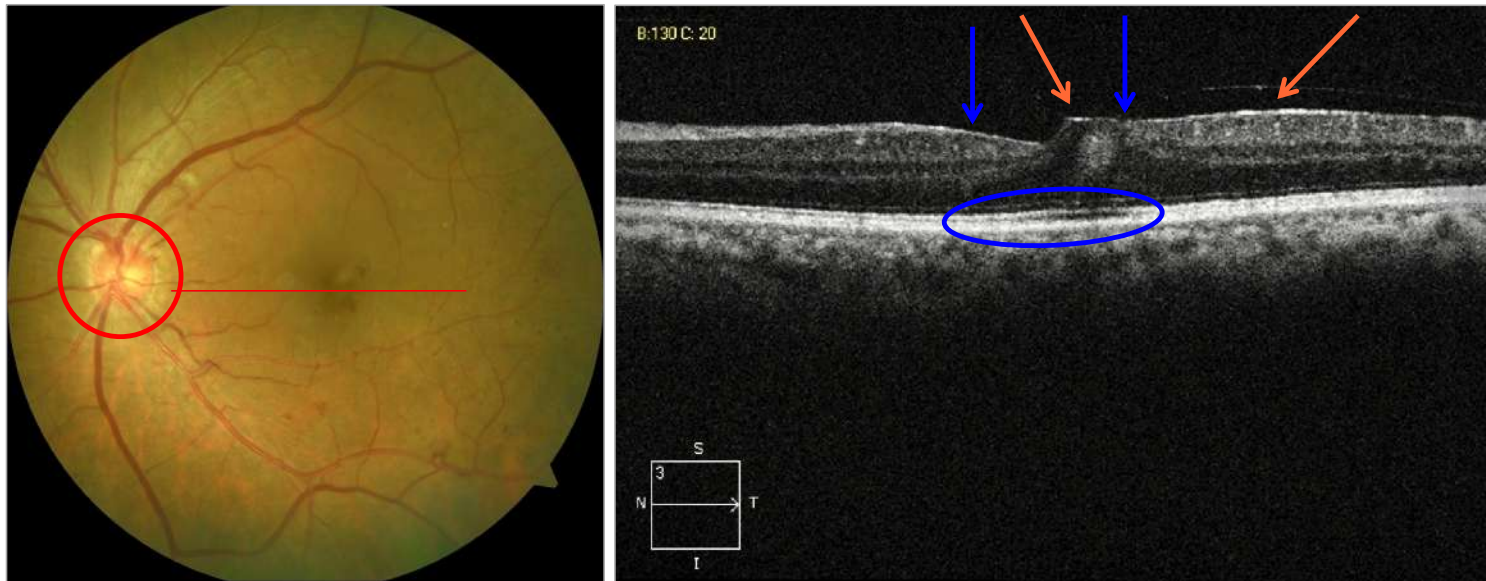
- **IRH/Ma just inferior to fovea (not visible on OCT raster line)**
- **central speckled IRF and adjacent clear cyst, but no obvious DRIL**
- **intact foveal outer retina (EZ and ELM): good BCVA (6/6)**

*CI-DME/good BCVA: monitor as per DRCR.net Protocol V*



# Proliferative diabetic retinopathy

*now 63 y/o Guyanese male; BCVA 6/7.5*



- **advancing DR with early NVD, but relatively stable DME and BCVA**
- **strong VMT/early ERM: poor prognostic signs (may prolong DME)**
- **intact outer retina (EZ/ELM) and minimal DRIL: good BCVA**

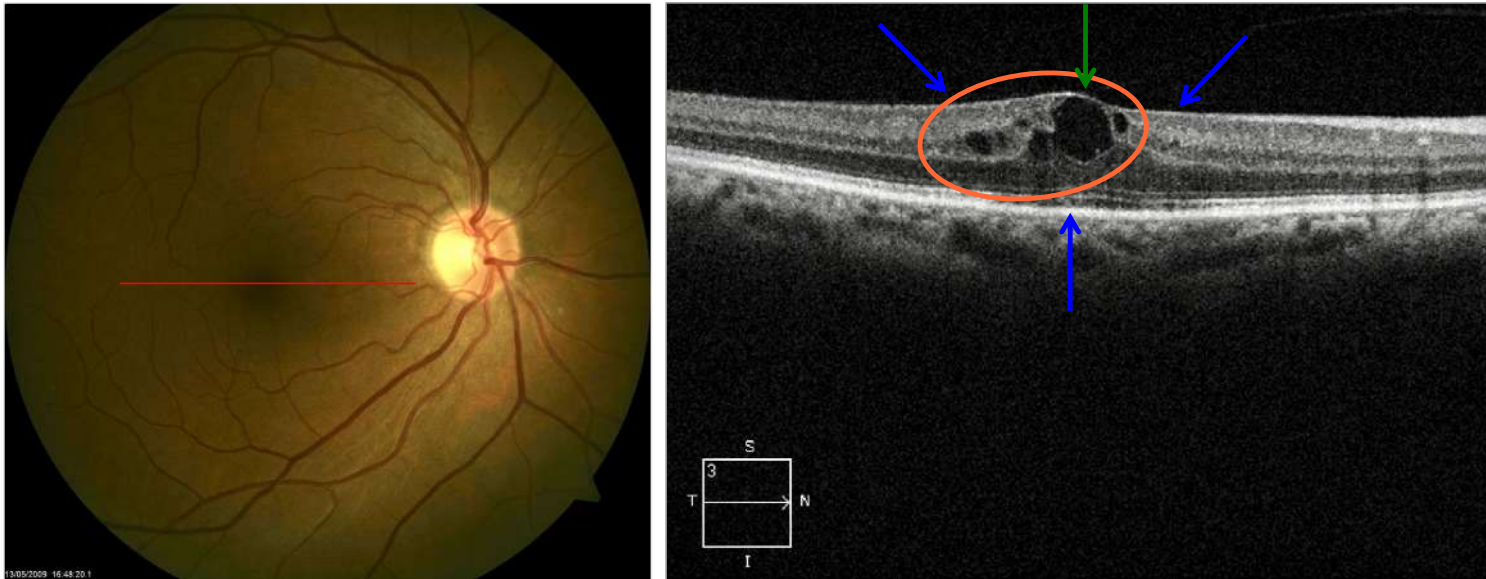
*Early PDR: anti-VEGF consult (but ultimate VH/vitreotomy/PRP)*





# Post-cataract extraction cystoid macular edema

72 y/o WW; BCVA 6/9 and “blotchy”



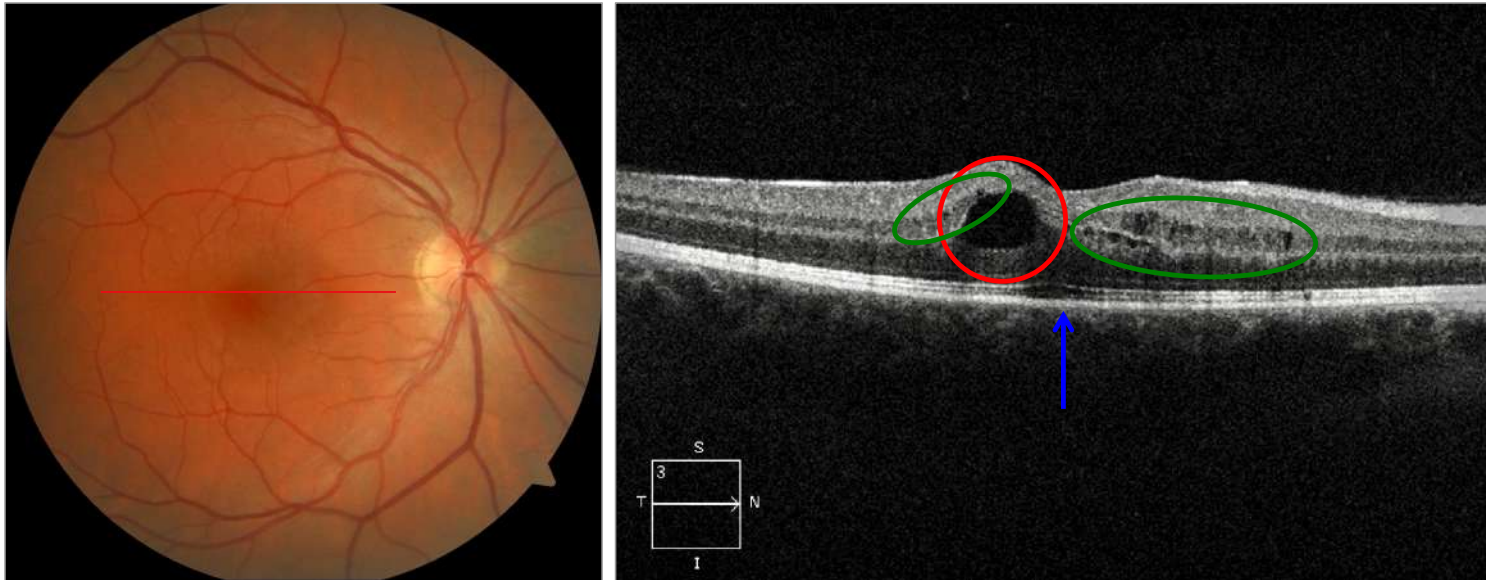
- **CME: clear cystic spaces involving both inner and outer retina**
- **anterior border of larger central cyst formed by ILM**
- **minimal outer retinal involvement and DRIL: BCVA remains 6/9**

*Continue aggressive topical steroid/NSAID (resolved in 1/12)*



# CME secondary to recurrent anterior uveitis

41 y/o WW; BCVA 6/24

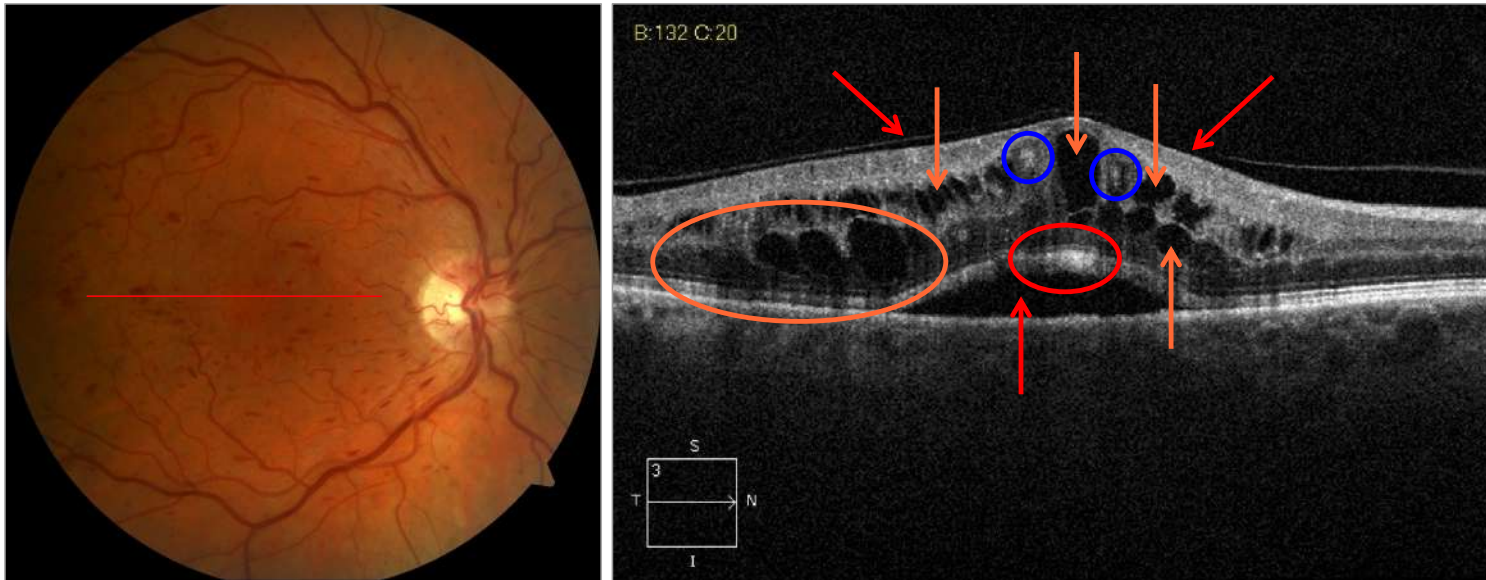


- **CME: larger clear cystic space (in anterior ONL and OPL) ...**
  - **... and multiple smaller pockets of IRF (in INL and OPL)**
  - **intact outer retina: BCVA reduced due to edema (and DRIL?)**
- AC quiet: continue topical NSAID (steroid responder; ultimately 6/6)*



# Central retinal vein occlusion

73 y/o WM; BCVA 6/30



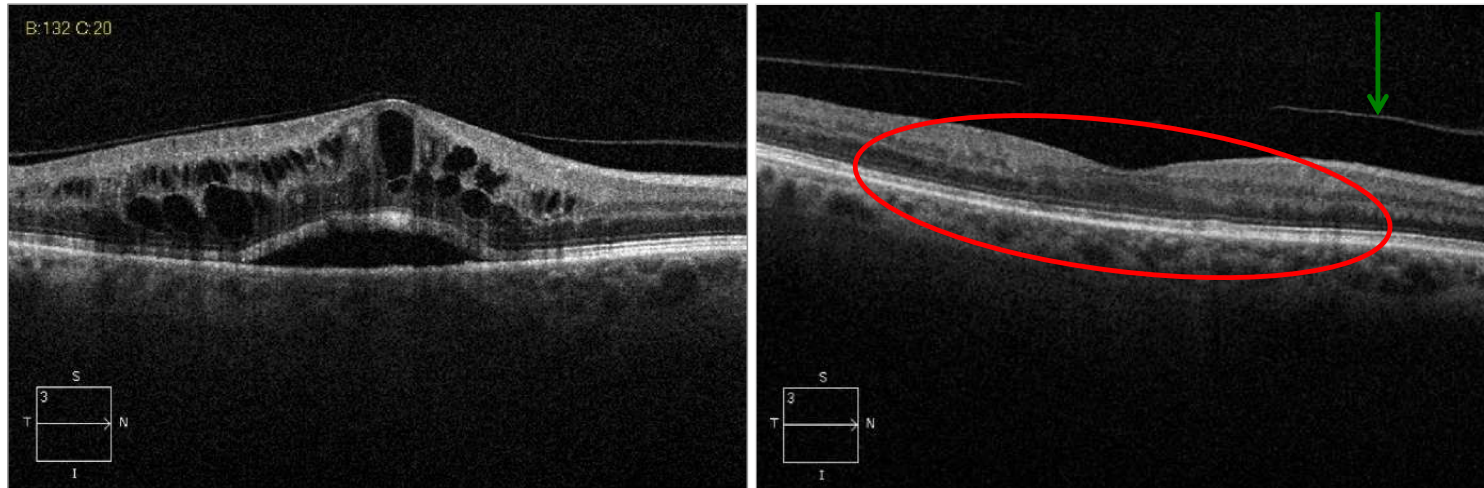
- **SRF/thickened EZ and DRIL: reduced BCVA (6/30)**
- **CME: multiple cysts (INL/ONL) with bridging tissue (positive sign)**
- **superficial hyper-reflective IRH with posterior shadowing**

*CME and SRF: referral for anti-VEGF consult*



## CRVO: post-anti-VEGF injection x4

73 y/o WM; BCVA now 6/6



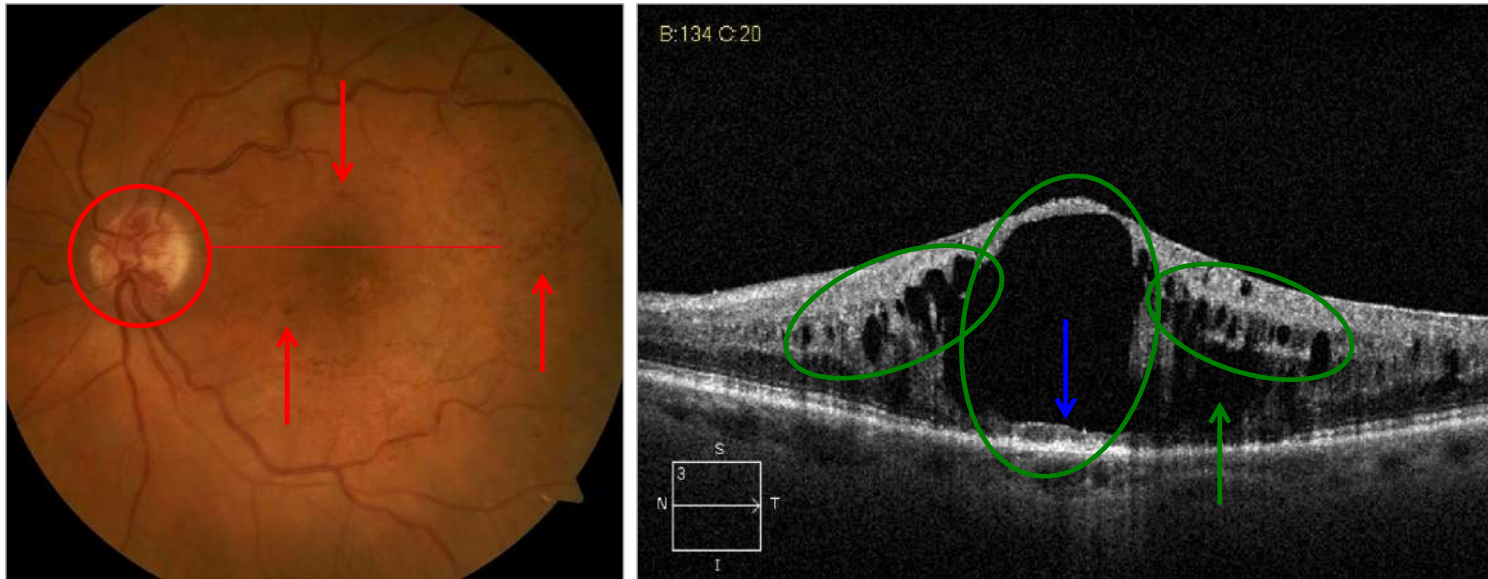
- pre-treatment (left)
  - **post-treatment (right): resolved CME/SRF; intact EZ; trace (?) DRIL**
  - **note separation of posterior cortical vitreous (complete PVD), now more obvious due to thinner post-treatment retina**
- Anti-VEGF injections ongoing: monitoring ~6/52*





## CRVO: not responsive to treatment

89 y/o WW; BCVA 6/30



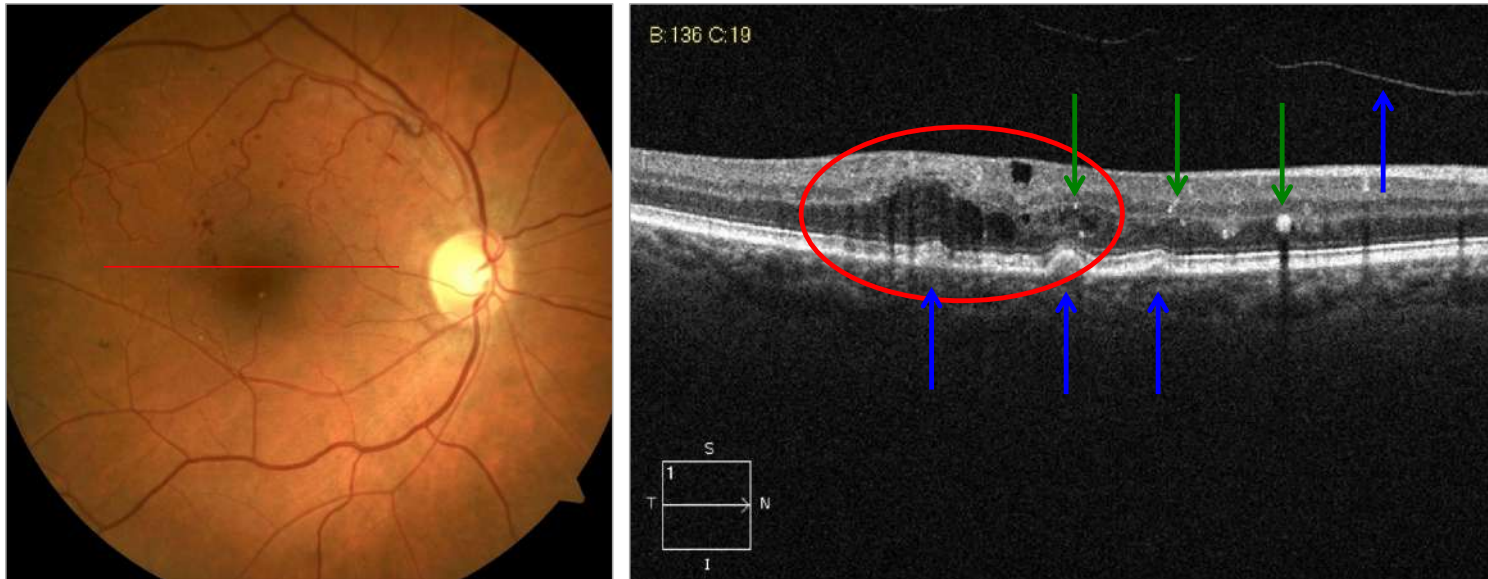
- **extensive collateral vessel formation and persistent hemorrhage**
- **central coalesced/multiple smaller cysts; diffuse outer thickening**
- **significant disruption of EZ and no bridging tissue: reduced BCVA**

*Monitor every 6/12 for (anterior segment) neovascularization*



# Branch retinal vein occlusion

71 y/o WM; BCVA 6/12



- **multiple inner and outer retinal cysts (GCIPL and ONL) and DRIL**
- **multiple HF and single larger IRH (posterior shadowing)**
- **coincidental drusenoid PED (query SDD?) and complete PVD**

*Referral declined: monitor carefully for increasing ME or NV*



# Central retinal artery occlusion

69 y/o WW; BCVA LP (in temporal periphery)



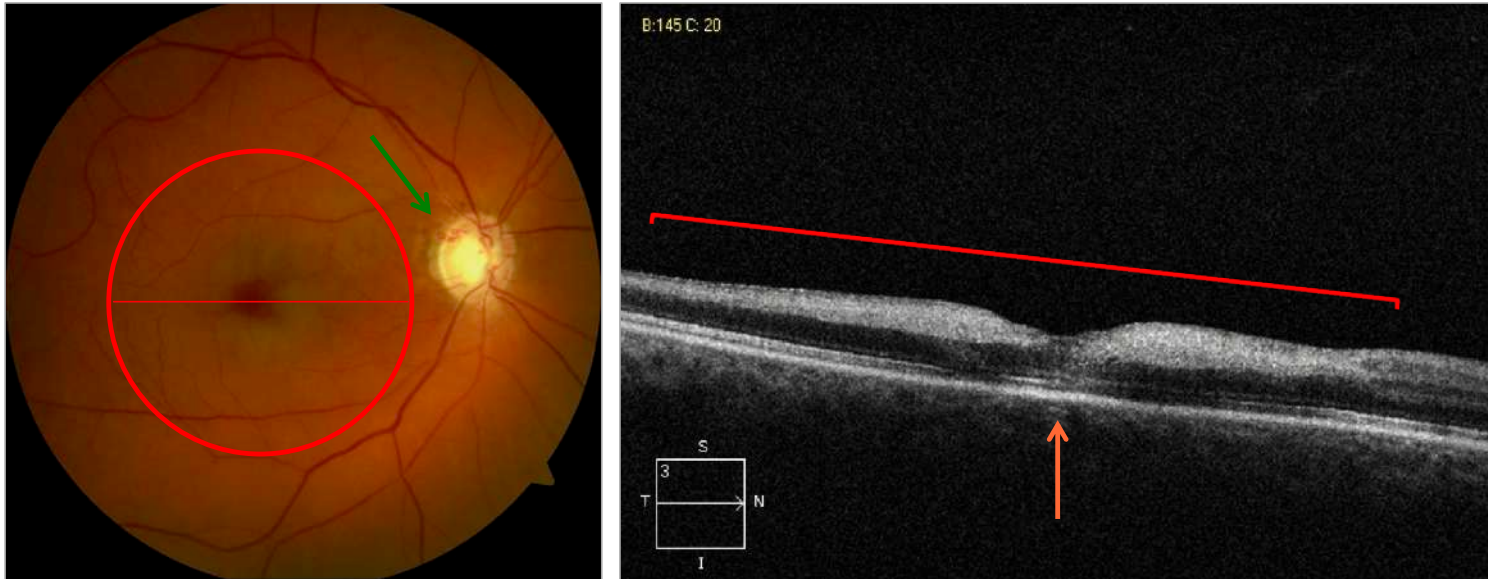
- **diffuse ischemia, cherry-red spot, and segmented blood column**
- **inner opacification (acute ischemia)/intact cilioretinal perfusion**
- **“middle limiting membrane”: OPL bipolar/photoreceptor junction**

*Immediate referral to retina and stroke clinic*



## CRAO: 7 weeks post-presentation

69 y/o WW; BCVA LP (in temporal periphery)



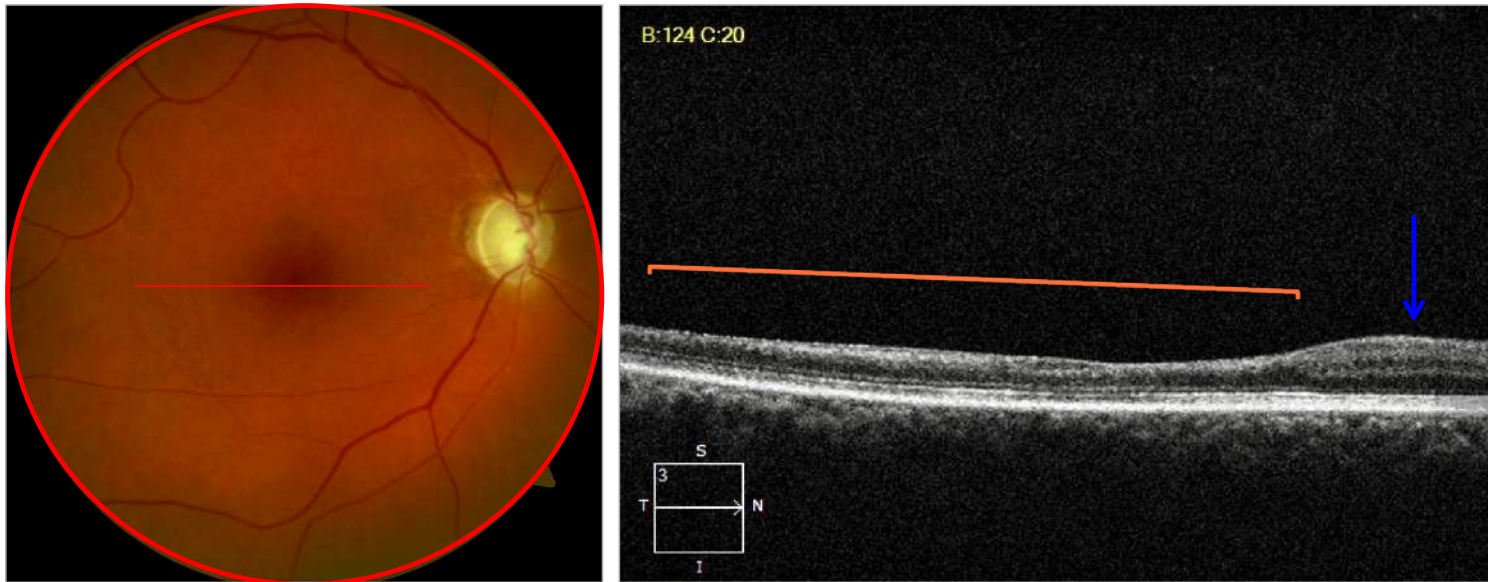
- **resolving inner retinal edema on both clinical exam and OCT**
  - **ONH pallor and superior-temporal collateral vessel formation**
  - **outer retina shadowed but intact: ↓ BCVA due to inner damage**
- No sign of cerebral stroke or giant cell arteritis: monitor for NVG*





## CRAO: two years post-presentation

71 y/o WW; BCVA LP (in temporal periphery)



- **“featureless” retina, ONH pallor, and regressed collateral vessels**
  - **extreme inner retinal atrophy, but preservation of outer retina**
  - **preservation of retina (and vision) perfused by cilioretinal artery**
- Patient wearing opaque CL due to peripheral vision being bothersome*



# Superior-temporal branch retinal artery occlusion

69 y/o WM; BCVA 6/15



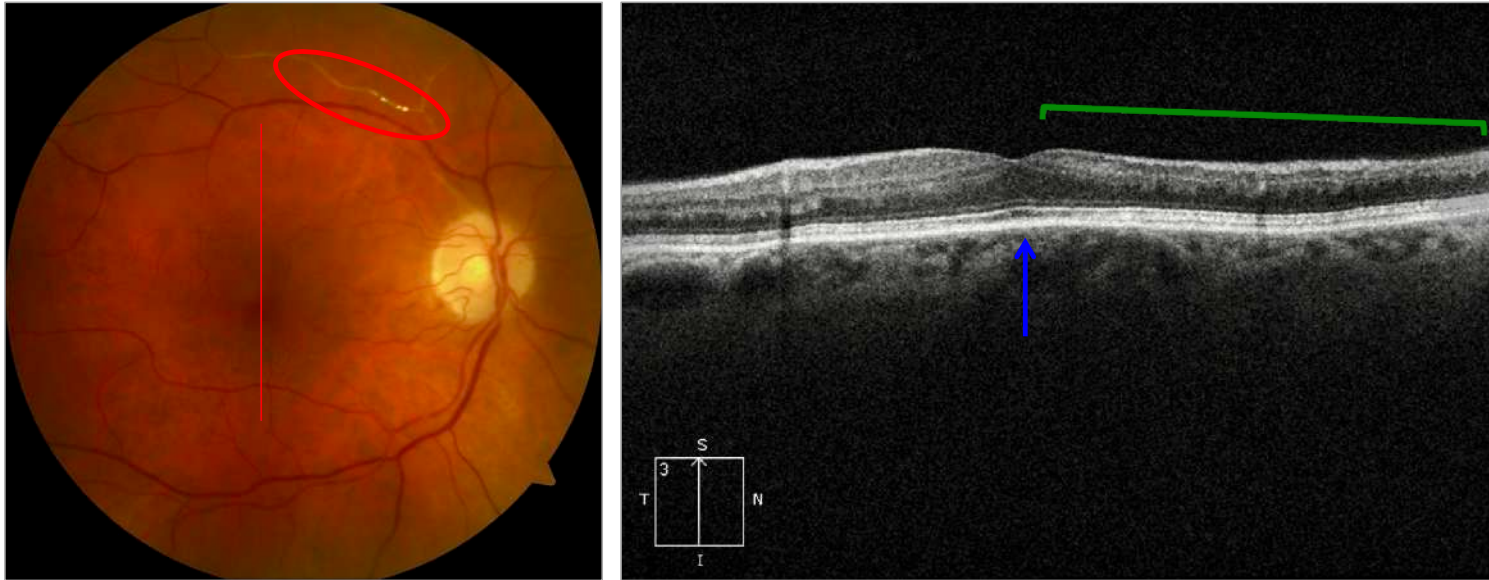
- **segmented superior-temporal arterial blood column (vs. emboli?)**
- **inner opacification: hyper-reflectivity due to acute ischemia**
- **“middle limiting membrane”: pathognomonic for RAO**

*Initially referred to rule out arteritis and cerebral ischemia*



# Superior-temporal BRAO: 6 months post-presentation

69 y/o WM; BCVA has improved to 6/7.5



- **multiple emboli in superior-temporal ghost vessel**
- **initially ischemic (thickened) inner retina now atrophic (thinned)**
- **foveal EZ/ELM intact: ↑ BCVA but inferior altitudinal VF loss**

*No arteritis or cerebral ischemia: ongoing 6/12 monitoring*



## Management of acute retinal ischemia

A stroke has been redefined as “... brain, spinal cord, or **retinal cell death** attributable to ischemia”<sup>1</sup>

A transient ischemic attack is now defined as “a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or **retinal ischemia** without acute infarction”

“... most health professionals ... consider retinal TIAs benign with a low risk of subsequent stroke.

**This is incorrect.**”<sup>2</sup>

“... acute retinal arterial ischemia [whether transient or permanent] is a stroke equivalent and represents an ophthalmologic and medical emergency ...”<sup>3</sup>

1. Sacco RL, et al. An updated definition of stroke for the 21<sup>st</sup> century. *Stroke* 2013;44:2064-89.
2. Brousse V. Acute retinal arterial ischemia: an emergency often ignored. *Am J Ophthalmol* 2014;157:1119-21.
3. Brousse V, et al. Management of acute retinal ischemia: follow the guidelines! *Am J Ophthalmol* 2018;125:1597-1607.





# Management of acute retinal ischemia

In patients presenting with acute RAO:

- one in three experience a coincident acute cerebral stroke
- one in three have severe carotid artery disease
- one in three present with a hypertensive emergency<sup>1</sup>

Patients with acute retinal ischemia (even asymptomatic) should be managed the same way as those with cerebral ischemia:

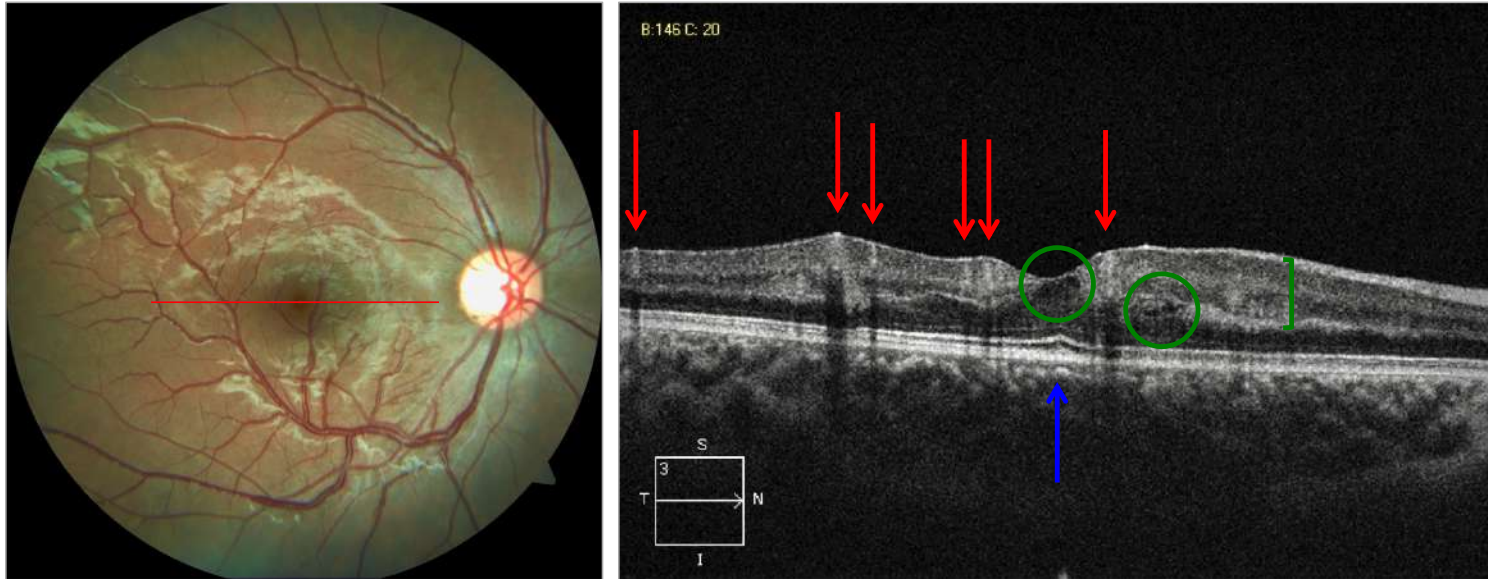
- immediate referral to the ER or a stroke neurologist
  - ESR, CRP, +/- temporal artery biopsy to rule out GCA
  - brain MRI to rule out cerebral ischemia/infarction
  - vascular imaging to rule out carotid stenosis
  - echocardiogram to rule out cardiac valve or aortic arch disease<sup>2</sup>

1. Lavin P, et al. Stroke risk and risk factors in patients with central retinal artery occlusion. *Am J Ophthalmol* 2018;196:96-100.  
2. Biousse V, et al. Management of acute retinal ischemia: follow the guidelines! *Am J Ophthalmol* 2018;125:1597-1607.



# Congenital retinal macrovessel (CRM)

17 y/o WW; BCVA 6/12

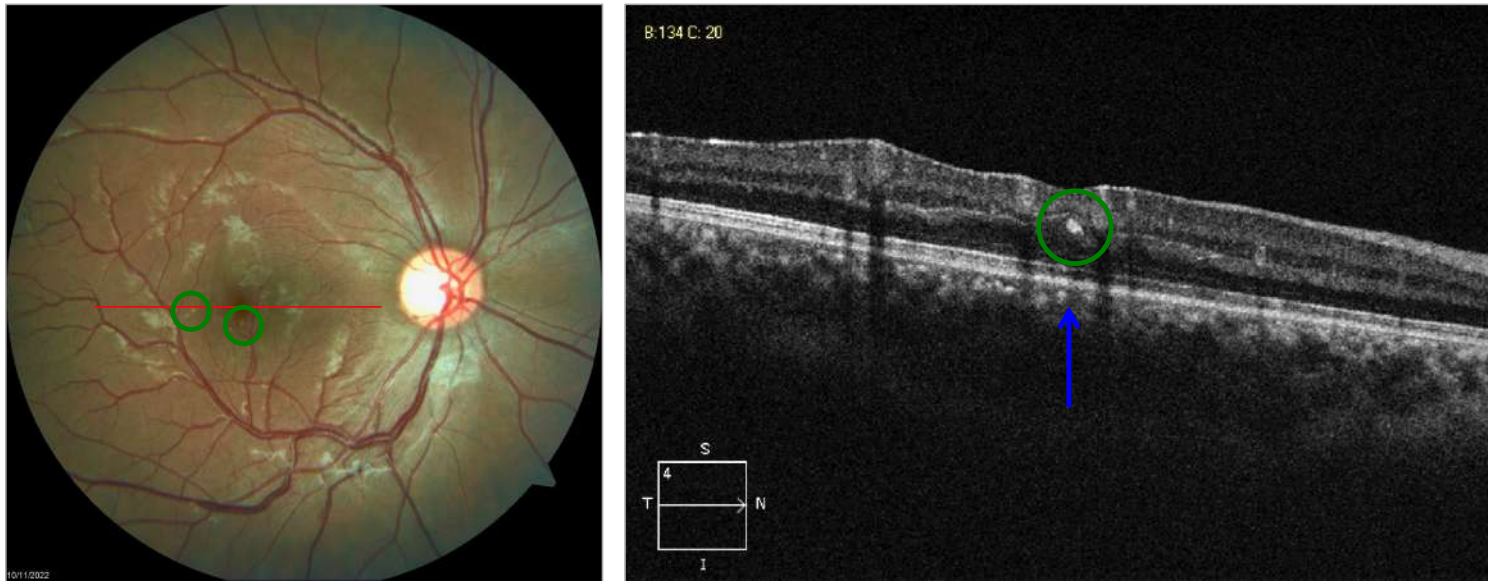


- **CRM crossing the horizontal raphe: six branches visible on OCT**
  - **diffuse inner thickening and cysts ( $\uparrow$  hydrostatic pressure)**
  - **intact outer retina, but decreased BCVA due to overlying edema**
- Monitored, as edema often resolves spontaneously (days to weeks)*



## CRM: one week post-presentation

17 y/o WW; BCVA stable at 6/12



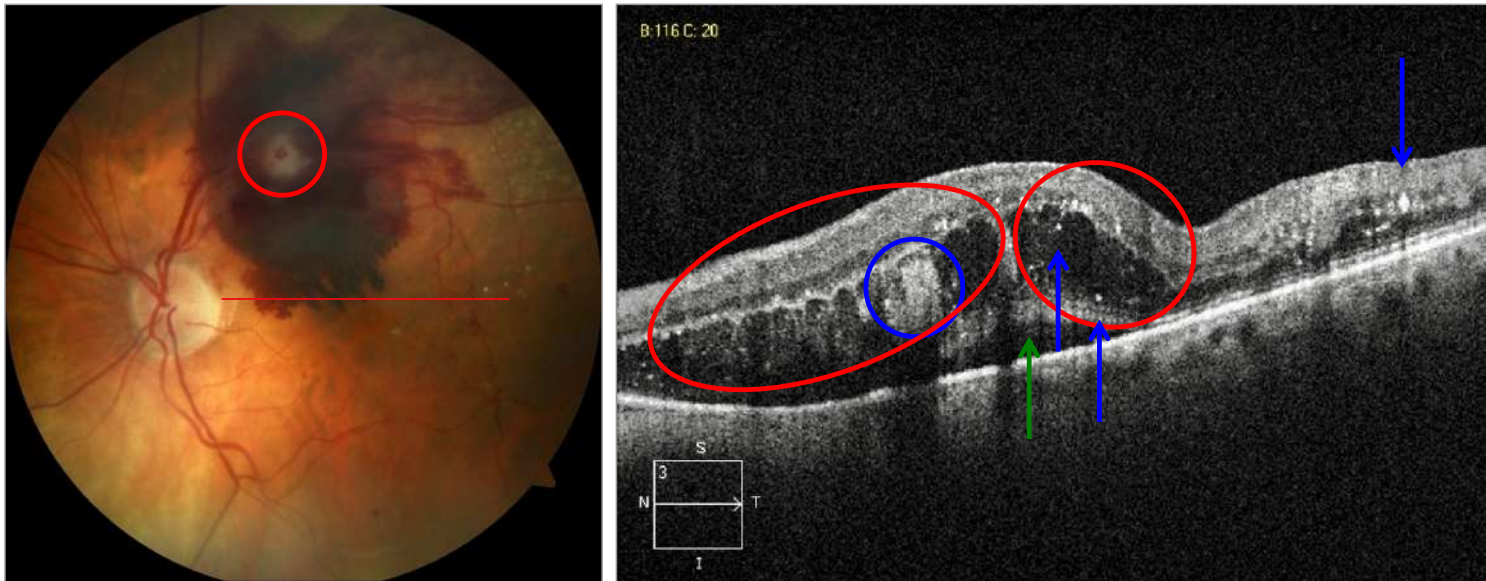
- **short-term stability: ↓ vessel caliber near fovea vs. 1 year ago?**
- **stable thickening/cysts and no DRIL, but several parafoveal HE**
- **outer retina remains intact and no change in BCVA**

*Continue to monitor for resolution, but refer to retina if worsens*



# Retinal arterial macroaneurysm (RAM)

92 y/o WW; BCVA 6/12

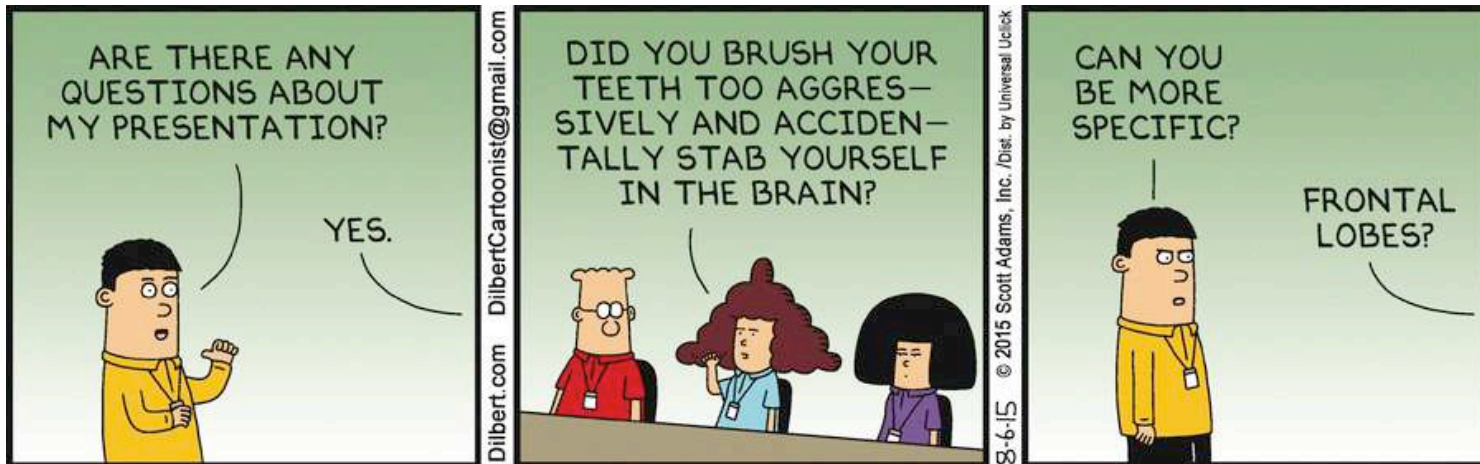


- **leaking aneurysm: diffuse outer retinal thickening; minimal DRIL**
- **SRF, thickened EZ, and overlying edema: reduced BCVA**
- **intraretinal hyper-reflective material: hemorrhage, exudate, foci**

*Referred, although RAM often resolve spontaneously*







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